

Adaptation of the Slow Component of VO₂ Following 6 wk of
High or Low Intensity Exercise Training

By

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(ABSTRACT)

Eighteen untrained males [age: 23 ± 0.6 yr. (SEM)] were randomized into high intensity (HIT: above lactate threshold, LT), moderate intensity (LIT: below the LT) or no training (NT) groups. Subjects trained on a cycle ergometer 4 days·wk⁻¹ for 6 wk with the power output held constant. Maximal cycle ergometry was performed before and after the training period to determine changes in power output and oxygen consumption (VO₂) at the LT and peak exertion. Before training and after 1, 2, 4, and 6 wk, subjects performed high constant-load (HCL) cycling bouts to quantify training adaptations in the SC. Training was designed to keep total work equivalent between the HIT and LIT groups. Increases in power output and VO₂ at LT and peak exercise after 6 wk were noted in the HIT and LIT groups in comparison to NT group ($p<0.05$). No differences were noted between HIT and LIT. Two-way repeated measures ANOVA revealed a significant trial*group interaction for adaptation in the SC ($p<0.001$). After 1wk of training, a significant reduction in the SC was noted for HIT [mean \pm SEM]: (pre-training (PT): 703 ± 61 ml·min⁻¹; 1 wk: 396 ± 60 ml·min⁻¹) (-44% from PT). Further adaptation for the HIT was also noted at 4 wk: 202 ± 45 ml·min⁻¹ (-71% from PT). For LIT, a significant reduction was noted at 2 wk (PT: 588 ± 76 ml·min⁻¹; 2 wk: 374 ± 50 ml·min⁻¹) (-36% from PT). Further adaptation for LIT group was noted at 6 wk (252 ± 38 ml·min⁻¹) (-57% from PT). Adaptation in SC was not noted at any interval for NT. Temporal changes in blood lactate ($r=0.40$) and ventilation ($r=0.72$) were significantly correlated with the changes for SC over the 6 wk training period ($p<0.05$). In conclusion, training at supra-LT and sub-LT intensities produces similar improvement in VO₂ and power output at peak exercise and in the LT, when total work output is held constant. However, training at supra-LT intensity promotes larger and faster adaptations in the SC than training at the sub-LT levels.

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“my greatest creations”

and Pam “I love you!”

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Chapter I

INTRODUCTION

There is a prevalent belief that during submaximal exercise pulmonary oxygen uptake (VO_2) increases as a linear function all power outputs to maximal levels of exertion (~9-11 ml O₂/Watt) (Whipp & Mahler, 1980; Whipp, 1987; Henson, Poole, & Whipp, 1989; Whipp, 1994). This belief has recently come under criticism, and has challenged many of the fundamental concepts in exercise physiology, such as the attainment of a “true” steady state during submaximal exercise and accurate quantitation of the oxygen deficit (Gaesser & Poole, 1996). There has been a reluctance in the physiologic literature, primarily in textbooks of exercise physiology (Powers & Howley, 1994; Wilmore & Costill, 1995), to acknowledge the belief that at work rates accompanied by a sustained increase in blood lactic acid concentration, i.e.> lactate threshold (LT), the linear relationship between VO_2 and work rate does not exist, with VO_2 exceeding the predicted estimation described above. Thus, with the recognition and documented existence of this phenomenon, fundamental concepts such as the steady state, oxygen deficit, and caloric equivalents for exercise have become indeterminate, or at least less definable at levels of exertion above the LT (Gaesser & Poole, 1996).

It is firmly established that the linearity function between work rate and VO_2 does indeed exist for work rates where an increase in [La] is not measured (Whipp, 1987; Paterson & Whipp, 1991; Gaesser & Poole, 1996). It was reported that following the onset of constant work rate exercise below which an increase in [La] is noted, VO_2 increases as a monoexponential process, achieving steady state by approximately 3 min (Whipp, 1987; Whipp, 1994; Wasserman, Hansen, Sue, Whipp, & Casaburi, 1994; Gaesser & Poole, 1996). However, for work rates accompanied

by a sustained increase in [La] (i.e. heavy exertion), VO_2 becomes a function of both work and time. At these power outputs, end exercise VO_2 is underestimated by the previously described linear relationship between power output and VO_2 . The characterization of the VO_2 response to power outputs accompanied by a sustained increase in [La] is more complex, and can no longer be described as a monoexponential process. Gaesser (1994) has described the onset of this domain of heavy exercise as "the lowest work rate at which blood lactate appearance exceeds its rate of removal", which physiologically can be defined as the maximal lactate steady state, or LT. In essence, following 80-110 sec of exercise (Barstow & Mole, 1991; Whipp, 1994), a second component, recently defined as "the slow component of VO_2 " becomes superimposed on the initial, abrupt rise noted following the onset of exercise (Whipp & Mahler, 1980; Barstow & Mole, 1991; Whipp, 1994, Gaesser & Poole, 1996). As exercise continued, steady state was delayed or not attained at all prior to exhaustion, and the linear relationship between VO_2 and work rate does not exist. If a steady state of VO_2 is eventually attained, the VO_2 response was often greater ($\geq 13 \text{ ml O}_2/\text{Watt}$) than that predicted for sub-LT work rates of moderate intensity work rates ($\sim 9-11 \text{ ml O}_2/\text{Watt}$) (Whipp & Mahler, 1980; Whipp, 1987; Henson *et al.*, 1989; Whipp, 1994). If a steady state was not eventually attained, subjects may have attained maximal VO_2 values at sub- $\text{VO}_{2\text{max}}$ work rates (Gaesser & Poole, 1996), at which the final VO_2 response was not defined as a steady state, but a limited state at which VO_2 no longer increased (Wasserman *et al.*, 1994).

In certain instances the measured slow component of VO_2 can be quite large. Several reports reported or described measurements of the slow component of VO_2 that amounted to an energy expenditure greater than 1.0 to 1.5 liters of oxygen above what would be predicted for

that work rate (Roston et al., 1987; Poole, Ward, Gardner, & Whipp, 1988; Barstow & Mole, 1991, Whipp, 1994, Gaesser & Poole, 1996).

Although Barstow and Mole (1991) demonstrated that this secondary slow component of VO_2 may be superimposed on the initial abrupt rise in VO_2 , it has typically been defined as the difference between the VO_2 at the end of exercise and at minute 3 of the exercise bout (Roston *et al.*, 1987; Casaburi, Storer, Ben-Dov, and Wasserman, 1995; Womack, Davis, Blumer, Barrett, Weltman & Gaesser, 1995).

To avoid comparison and confusion Gaesser and Poole (1996) suggested that this phenomenon, i.e. the slow component of VO_2 , not be confused with the increase in VO_2 that is noted during moderate intensity exercise (<LT) that occurs over an extended period of time. This phenomenon is commonly referred to as “ O_2 drift”. Typically, a much smaller increase in VO_2 was noted with the O_2 drift, and was not associated with any markable increase in [La] (Kalis, Freund, Joyner, Jilka, Nitolo, & Wilmore, 1988).

Despite overwhelming evidence concerning the effects of endurance exercise training on maximal or peak VO_2 , the same is not true regarding the reported effects of endurance exercise training on the submaximal VO_2 responses. During submaximal work, steady state VO_2 was either unchanged (Davis, Frank, Whipp, & Wasserman, 1979; Hagberg, Hickson, Ehsani, & Holloszy, 1980) or reduced (Belman & Gaesser, 1991; Casaburi, Storer, & Wasserman, 1987, Casaburi *et al.*, 1987, Casaburi *et al.*, 1995, Womack *et al.*, 1995) following exercise training. In a recent critique of the literature regarding this discrepancy, Gaesser (1994) contended that the conflicting reports regarding the submaximal VO_2 response following training can be explained, in part, by the intensity domain used for measurement. In essence, for work rates of moderate

intensity below the pre-training LT, steady state VO_2 was unaffected, and the linear $\text{VO}_2/\text{work rate}$ relationship was unchanged. However, it is believed that for work defined as heavy intensity, above the pre-training LT, there is a change in the $\text{VO}_2/\text{work rate}$ relationship. This results from either a decrease in the VO_2 at steady state, given the subject is able to attain a steady state at that level of exertion, or from a reduction in end exercise VO_2 .

In addition to conflicting evidence regarding the existence of change in submaximal VO_2 responses with endurance training, there is either a lack of evidence or there are conflicting reports regarding the time course of adaptation among those who have reported training-induced reductions in submaximal VO_2 following training. Reductions in the slow component of VO_2 were documented at 2 (Womack *et al.*, 1995), 7 (Poole, Ward & Whipp, 1990), and 8 wk of endurance training (Casaburi *et al.*, 1987; Belman & Gaesser, 1991; Casaburi *et al.*, 1995). However, only one study to date has specifically determined the temporal relationship between endurance exercise training and changes in the slow component of VO_2 . Womack *et al.*, (1995) recently demonstrated that following a high-intensity endurance training program, the slow component of VO_2 was rapidly attenuated, with greater than 50% reduction of the slow component of VO_2 within the first 2 wk of training. In addition, in the majority of these studies, the training was performed at intensities only above the LT, with little attention given to the effect of training intensity at levels below the LT on changes in the slow component of VO_2 . Thus, no study to date has investigated both the temporal adaptations in the slow component as a function of different training intensities, i.e. low vs. high intensity training.

Statement of the Problem

Thus, the purpose of this investigation was to determine the time course and magnitude of adaptation in the slow component of VO_2 resulting from 6 wk of high or low-intensity endurance exercise training in healthy, sedentary males. In addition, the associations of potential mediators were studied by assessing changes in ventilation and blood lactate concentrations over the 6 wk training study.

Significance of the Study

The reduction of the slow component of VO_2 following endurance training has significant practical implications that are related to the fatigue process (Poole, Barstow, Gaesser, Willis, & Whipp, 1994). By limiting the development the slow component of VO_2 , individuals could substantially increase the ability for sustaining physical activity, and increase work tolerance. In an apparently healthy subject, the steady state VO_2 increases linearly with work rate until the work rate surpasses the LT. At work rate above the LT, the VO_2 response becomes nonlinear, the VO_2 continues to increase, and the subject fatigues when VO_2 reaches a limited state. Following endurance exercise training, both the LT and $\text{VO}_{2\text{max}}$ have increased. Thus the point at which the VO_2 response and work rate becomes nonlinear is extended to a higher work rate, and in addition, the point at which the subject reaches the post-training $\text{VO}_{2\text{max}}$ is extended. In comparison, the response in subjects with cardiac and/or pulmonary limitations is quite different. To note, Gaesser, Cooper and Goodfellow (1991) reported a reduction in the slow component of VO_2 following low intensity exercise training in healthy subjects, without observing an increase in $\text{VO}_{2\text{max}}$. These findings could have significant clinical ramifications in patients who have a limited ability to increase $\text{VO}_{2\text{max}}$. Patients with cardiac and pulmonary limitations often have a

low $\text{VO}_{2\text{max}}$ and abnormally low LT. Thus, the point at which the VO_2 and work rate breaks from linearity, driving VO_2 toward a limited $\text{VO}_{2\text{max}}$, can occur at very low levels of physical exertion, i.e. activities of daily living. Although the physiologic and functional limitation may preclude any noticeable increase in maximum VO_2 , these patients may still benefit from exercise training by increasing the point at which VO_2 and work rate becomes nonlinear, thereby reducing the magnitude of the slow component of VO_2 . In effect, one would speculate that endurance exercise training in these individuals would increase their ability to perform activities of daily living.

Additionally, caloric expenditure is clearly greater during exercise that elicits the slow component of VO_2 . Individual variation in the threshold intensity that elicits a slow component limits the availability of developing "all encompassing" guidelines for all populations. However, it would seem beneficial for individuals in which caloric expenditure was vital, that, if tolerable, guidelines could be set to take advantage of this response.

Research Hypotheses

H_o (1): The change in the slow component of VO_2 during high-intensity, constant-load cycle ergometry is the same following 1, 2, 4, and 6 wk of endurance exercise training for the high-intensity (HIT) and low-intensity training groups (LIT), and the control group (NT).

H_o (2): No association exists between ΔVE and the slow component of VO_2 during a 6 wk period of endurance exercise training for HIT, LIT and NT groups.

H_0 (3): No association exists between $\Delta[La]$ or end exercise [La] and the slow component of VO_2 during 6 wk of endurance exercise training for HIT, LIT, and NT groups.

Assumptions

1. Subjects accurately answered the medical/health history questionnaire and, in particular, correctly reported their current physical activity regimen prior to the study.
2. Subjects exhibited a maximal effort during all submaximal and maximal exercise test protocols.
3. The work intensity and time that was assigned to each subject were monitored by a staff member and completed appropriately..
4. Subjects reported any additional cardiovascular training activity that was performed outside of this study.
5. Subjects did not alter their diet throughout the study.
6. Subjects complied with all pre-testing instructions.
7. The testing and training bicycle ergometer were accurately calibrated and maintained throughout the study period.
8. The MedGraphics CPX/D metabolic cart accurately measured all cardiopulmonary variables.
9. The YSI 2300 Lactate analyzer accurately measure the blood [La] during ramp and submaximal constant-load exercise testing.

Limitations

1. The sample make-up (gender, age, clinical status) limits the generalizability to gender, subjects of different age (e.g. elderly), and in individuals with cardiopulmonary and/or metabolic disorders
2. Due to technical difficulties in obtaining blood [La] samples during the high constant-load and/or low constant-load ergometry test, samples were not obtained for all individuals for all trials
3. Due to equipment malfunction/failure it was necessary to switch to similar alternative equipment that may have altered results due to possible variations in motor mechanics and mechanical efficiency.
4. Subjects were randomly assigned to either high intensity, low intensity, or control (no exercise training) groups following the completion of the initial testing with equal sample sizes for each group anticipated. Due to subject drop-out following testing, or during the training period, inequality of group membership resulted. For statistical purposes, members of the high intensity and low intensity group were randomly selected from each group.
5. The initial intensity set for the high-intensity and low-intensity constant work rate tests and initial exercise intensity prescribed to subject in the high and low exercise training groups were based on the initial ramp test. No attempts were made during the 6 wk study to adjust the intensity secondary to improvement in the subjects' conditioning level. Thus, by 6 wk of exercise training, the exercise training intensity performed by the subjects was lower, given any improvement in peak VO₂.

Delimitation's

1. Subjects were volunteers who attended Virginia Polytechnic Institute and State University between the ages of 18 and 30.
2. Subjects had not been involved in any cardiovascular training (defined as < 2 day/wk) for at least 6 months prior to the study.

Definitions of Symbols and Terms

1. Alveolar Ventilation (VE: the volume of inspired gas that reaches the alveoli per minute).
2. Arterial-venous oxygen difference (AVO_2 diff)- the difference in the oxygen content of the arterial and venous blood, usually expressed in milliliters of oxygen per deciliter or liter of blood.
3. Breath-by breath - a method for measurement of respiratory gas exchanges in which respiration gas volumes and simultaneously measured expired gas concentration are integrated and reported.
4. Carbon dioxide output (VCO_2) - the amount of carbon dioxide exhaled from the body, usually expressed per unit time in milliliters or liters per minute.
5. Cardiac output (Q) - the flow of blood from the heart in a particular period of time, usually expressed as liters per minute. It can be defined as the product of the stroke volume per beat and the heart rate per minute. In addition, by the Fick equation, it can be defined as oxygen uptake divided by arterial-venous oxygen difference.
6. Constant work rate test - an exercise test in which a constant power output is required of the subject.

7. Delta Lactate (ΔLa) - the difference between end-exercise [La] and [La] at 3 min of a high- or low-constant work rate test.
8. Delta Ventilation (ΔVE - the difference between end-exercise VE and VE at 3 min of a high- or low-constant work rate test.
9. Exponential - a process in which the rate of change of a variable (i.e. oxygen uptake) is proportional to the “distance from a steady-state level. Therefore, the rate of change of the function under consideration is rapid when it is far from the steady-state value and slows progressively as the function approaches its steady-state. If the process is known to be exponential, the time to reach 63% of the final value is termed the time constant of the response. In addition, if the process is exponential, the time constant is related to the half-time (the time to reach 50% of the final value) by the equation: half time = 0.693 time constant of the response.
10. Gas exchange ratio or respiratory exchange ratio (RER) - the ratio of carbon dioxide output to the oxygen uptake per unit of time.
11. Half time ($t^{1/2}$) - unlike the time constant, which requires evidence of exponentiality for its determination, the half time of the response is a simple description of the time to reach half of the change to its final value, regardless of the function. It is generally representative of the speed of approaching the steady-state.
12. High Constant Load Test (HCL) - subjects will cycle at [power output at LT + .75 (power output Peak - power output LT)]. All CL bouts will include collection of 3 min of seated baseline data, 15 min of exercise data, and 6 min of seated recovery data.

13. High-intensity training (HIT) - to determine the training intensity for subjects, total work was computed for 60 min of work at the low work intensity ([power output at LT * .90]. Following determination of the level to be assigned for the high intensity training subjects, the number of minutes for exercise was determined so that total work, regardless of the group assigned, was equivalent. An example of this calculation is included in the detailed methodology.
14. Incremental exercise test - an exercise test designed to provide gradational stress to the subject. The work rate is usually increased over uniform periods of time, for example, every 4 min, every min, every 15 sec, or even continuously (e.g. ramp pattern increment).
15. Lactate - the anion of lactic acid
16. Lactate threshold (LT) - the exercise oxygen uptake or work rate above which a net increase in lactate production results in a sustained increase in blood lactate concentration. In the present study, the LT was defined as the breakpoint in the relationship between [La] plotted as a function of VO₂ during the ramp protocol. Three separate individuals calculated the LT for each subject, with the final point being the average of the three.
17. Lactic acid - a three-carbon carboxylic acid that is one of the potential end-products of glucose oxidation.
18. Low Constant Load Test (LCL) - subjects will cycle at a power output equal to [power output at LT * .90]. All CL bouts will include collection of 3 min of seated baseline data, 15 min of exercise data, and 6 min of seated recovery data.

19. Low-intensity training (LIT) - to determine the training intensity for subjects, total work was computed for 60 min of work at the low work intensity ([power output at LT * .90]. An example of this calculation is included in the detailed methodology.
20. Maximal arterial-venous oxygen difference (AVO_{2max}) - the maximal difference in the oxygen content of the arterial and venous blood, which reflects the amount of oxygen extracted by the tissues. It is usually expressed in milliliters of oxygen per deciliter or liter of blood.
21. Maximal cardiac output (Q_{max}) - the maximal amount of flow of blood from the heart in a particular period of time, usually expressed as liters per minute. It can be defined as the product of the maximal stroke volume per beat and the maximal heart rate per minute. In addition, by the Fick equation, it can be defined as maximal oxygen uptake divided by maximal arterial-venous oxygen difference.
22. Maximal oxygen uptake (VO_{2max}) - the highest oxygen uptake attainable despite further work rate increases.
23. Oxygen deficit - the oxygen equivalent of the total energy utilized to perform the work that did not derive from reactions utilizing oxygen taken in from the body at the start of exercise.
24. Oxygen uptake (VO₂) - the common biological measure of total body work defined by the rate at which oxygen is consumed by the body, expressed in Liters per minute ($L \cdot min^{-1}$) or milliliter per kilogram per minute ($ml \cdot kg^{-1} \cdot min^{-1}$)
25. Peak oxygen uptake (VO_{2peak}) - whereas VO_{2max} represents the highest value attainable for the entire body, the expression VO_{2peak} is utilized when an exercise task involves the use

of a smaller percentage of total muscle mass, or when endpoints fail to satisfy physiologic criteria of $\text{VO}_{2\text{max}}$. Thus, the differentiation is made between endpoints that are brought on by symptoms such as local muscle fatigue, chest pain or shortness of breath (peak VO_2) and endpoints defined by functional limits of the cardiopulmonary system.

26. Phase I (oxygen kinetics) - the period of time following the onset of exercise that is required for the products of exercise metabolism to reach the lungs. Normally, this period is 15 to 20 sec.
27. Phase II (oxygen kinetics) - the period of time following the onset of exercise when the mixed venous blood gas concentrations continue to change because of changes in the exercising muscles. It, therefore, reflects the kinetic phase of the gas exchange that begins at the end of phase I and continues until steady state is obtained.
28. Phase III (oxygen kinetics) - the steady-state phase of gas exchange. For moderate exercise, this reflects the point where mixed venous gas concentrations have become constant.
29. Ramp exercise test - a type of protocol utilized during maximal exercise testing in which work increases constantly and continuously. This type of test allows for increases in work to be individualized, and a given maximal test duration targeted. The steps used to determine the increment utilized during the ramp for each subject was as per Wasserman *et al.* 1993:

Step 1: $\text{VO}_2 \text{ unloaded in ml/min} = 150 + (6 * \text{body weight in kilograms})$

Step 2: $\text{Maximum } \text{VO}_2 = (\text{height in cm} - \text{age in years}) * 20$

Step 3: Work rate per minute = maximum VO₂ - unloaded VO₂ / 100

30. Respiratory exchange ratio (R/RER) - the ratio of the rate of carbon dioxide production to oxygen consumption. This ratio reflects the metabolic exchange of the gases in the body's tissues and is dictated by substrate utilization.
31. Response time - a means of characterizing the rate at which a device or system responds to a given signal. For example, in response to sudden exercise, how long does it take the output (i.e. oxygen uptake) to become constant. This can be characterized by the time constant, half time, mean response time, or the time to reach 90% of its value.
32. Sedentary - subjects who had not been involved in a structured endurance activity (< 2 day/wk) for at least 6 months prior to the study. This definition was utilized in a prior study by Casaburi et al. (1987).
33. Slow component of oxygen uptake - although the mechanism(s) accounting for this phenomenon continue to be debated, it is currently described as the difference between end-exercise oxygen uptake and oxygen uptake at 3 min of constant work rate exercise test.
34. Steady state - this is a characteristic of a physiologic system in which its functional demands are being met such that its output per unit time becomes constant. If the system reaches the limits of its output, and, as a result, its output becomes constant, as is the case of oxygen uptake reaching its maximal value, this is then termed a limited state, and not a steady state.
35. Stroke volume - the amount of blood ejected from either ventricle of the heart in a single beat.

Summary

Despite the overwhelming evidence concerning the effects of endurance exercise training on $\text{VO}_{2\text{max}}$, the effects of endurance exercise training on the submaximal VO_2 responses are more conflicting. The need for further study and greater understanding, not only of the mechanism(s) responsible for the slow component of VO_2 , but of the magnitude of change, and work intensity required has both physiological and clinical significance.

Thus, this current study sought to determine the time course and magnitude of adaptation in the slow component of VO_2 resulting from 6 wk of high or low-intensity endurance exercise training in healthy, sedentary males. The contribution of potential mediators of the slow component were also studied.

Chapter II

REVIEW OF LITERATURE

Introduction

This study sought to determine the time course and magnitude of adaptation in the slow component of VO_2 resulting from 6 wk of high or low-intensity endurance exercise training in healthy, sedentary males. In addition, potential mediators of the slow component were studied. The purpose of this chapter is to provide the reader with a(n): a) review of the nature and extent to which physiologic changes occur with physical training, b) review the history of gas exchange kinetics and the slow component of oxygen uptake, c) examination and report findings of the literature that have examined the potential mediators of the slow component of VO_2 and d) definition of what is currently known about adaptations in oxygen kinetics following exercise training.

Physiologic changes following endurance exercise training

The changes in the pulmonary oxygen uptake (VO_2) response that result from endurance exercise training have been examined by both incremental exercise testing and submaximal, constant work rate exercise testing procedures (Gaesser, 1994). Historically, when utilizing an incremental protocol, in conjunction with cardiopulmonary gas exchange technologies, the parameter most often reported in ascertaining the effectiveness of a training program is the maximal pulmonary oxygen uptake ($\text{VO}_{2\text{max}}$). The $\text{VO}_{2\text{max}}$ describes the upper limit of the integrated cardiopulmonary and circulatory functions that involves transportation of oxygenated blood to the working muscles, utilization of the oxygen at the tissue level, and transportation and removal of carbon dioxide from the system. In theory, the $\text{VO}_{2\text{max}}$ is quantitatively related to

maximal cardiac output (Q_{\max}) and maximal arterio-venous oxygen difference ($A-V O_{2\max}$) and can be defined as:

$$VO_{2\max} = Q_{\max} \times A-V O_{2\max} \quad (\text{Rowell, 1993})$$

The Q_{\max} is a reflection of the peak pumping capacity of the heart. The $A-V O_{2\max}$ is indicative of the amount of oxygen being extracted from the blood by the tissues. At rest, approximately 25% of the oxygen is extracted by the tissues, or approximately 4 to 5 milliliters of oxygen per 100 milliliters of blood. At maximal exertion, the difference between the arterial and venous oxygen content dramatically widens with the exercising tissues extracting as much as 16 to 18 milliliters of oxygen per 100 milliliters of blood at peak exertion (Frolicher, Myers, Follansbee, and Lobovitz, 1993).

Improvements in $VO_{2\max}$ following an endurance exercise training program have widely varied, with improvements ranging anywhere from 4 to 50% (Pollock, 1973; Davis *et al.*, 1979; Casaburi *et al.*, 1987; Belman & Gaesser, 1991; Wilmore & Costill, 1995). Evidence has indicated that training-induced improvements in $VO_{2\max}$ are directly related to frequency, intensity, and duration of training. However when exercise is performed above a minimal threshold intensity, total work accomplished becomes a critical factor in the development of cardiorespiratory fitness (American College of Sports Medicine, 1995). Of all the factors listed above, none has gained as much attention the exercise training intensity. It is generally believed that there is a minimum training intensity, or threshold under which no cardiopulmonary improvements in training, regardless of the duration of training, will be obtained by the subject. The American College of Sports Medicine (1995) reports a minimal training intensity corresponding to approximately 40% of maximal VO_2 . However the authors recognize that in

certain clinical populations, such as individuals with cardiopulmonary disease or the elderly, measurable improvements can occur at intensities lower than reported previously.

In addition to the training parameters described above, the ability to detect training-induced changes in $\text{VO}_{2\text{max}}$ is also a function of the subjects' initial level of physical conditioning. Rowell (1993) suggests the farther away the pre-training $\text{VO}_{2\text{max}}$ value is below 45 $\text{ml O}_2 \cdot \text{kg}^{-1} \cdot \text{min}$, the greater the anticipated increase in $\text{VO}_{2\text{max}}$ with physical training (Rowell, 1993). In contrast, elite endurance athletes may exhibit very small increases in $\text{VO}_{2\text{max}}$ over the same period of training.

Maximal aerobic power, or $\text{VO}_{2\text{max}}$, has been accepted as the best measure of the functional limits of the cardiovascular system. A critical aspect in the measurement of $\text{VO}_{2\text{max}}$ is that "attainment of a true physiologic $\text{VO}_{2\text{max}}$ " requires that a certain proportion of total muscle mass be used during activity (Rowell, 1993). Although this fraction of total muscle mass needing to be physically activated has yet to be established, it is believed to be approximately 50% of the total muscle mass (Rowell, 1974).

Whereas $\text{VO}_{2\text{max}}$ represents the highest value attainable for the entire body, the expression maximum or peak VO_2 is used when an exercise task involves the use of a smaller percentage of total muscle mass, or when endpoints fail to satisfy objective physiologic criteria of a maximal level for VO_2 (i.e. no increase in VO_2 despite further increases in work rate). Thus, the differentiation is made between endpoints that are brought on by symptoms such as local muscle fatigue, chest pain or shortness of breath, or lack of motivation and endpoints defined by functional limits of the cardiopulmonary system (Q_{max} and A-V $\text{O}_{2\text{max}}$) (Rowell, 1993). Although the concept of a plateau in VO_2 despite an increase in work has long been a primary objective

criteria for having truly achieved $\text{VO}_{2\text{max}}$, it is not uncommon for subjects to complete a maximal exercise test and not obtain a plateau. Thus, secondary end-points have been used and include: 1) achievement of some percentage of age-predicted estimate of maximal heart rate, 2) exceeding a respiratory exchange ratio greater than unity (> 1.0), 3) achieving a high level of blood lactic acid at peak exertion or in the minutes following exercise, and 4) surpassing an rating of perceived exertion of > 17 . The use of any of these criteria, including the use of a plateau in VO_2 is suspect because these variables can have considerable measurement error and subject variability (Myers, Walsch, Sullivan, & Frolicher, 1990, Myers, 1996).

Changes in submaximal VO_2 following endurance exercise training

Despite overwhelming evidence concerning the effects of exercise training on $\text{VO}_{2\text{max}}$, the same is not true regarding the effects of endurance exercise training on submaximal VO_2 responses. During submaximal work, VO_2 is either unchanged (Davis *et al.*, 1979; Hagberg *et al.*, 1980) or reduced (Belman & Gaesser, 1991; Casaburi *et al.*, 1987; Poole *et al.*, 1990; Casaburi *et al.*, 1995; Womack *et al.*, 1995) following exercise training. Gaesser (1994) contends that the conflicting evidence regarding the submaximal VO_2 response following training can be explained, in part, by the intensity domain used for measurement (i.e. with or without lactic acidosis). To illustrate what is presently understood regarding the effects of training on the submaximal VO_2 response to exercise, and to clarify how the intensity domain can influence the observance of training-induced changes in the submaximal VO_2 response, a brief description of the dynamic VO_2 response to an exercise bout is warranted.

VO₂ kinetics

During the transition from rest to a constant work rate, steady state levels of VO₂ are not achieved instantly, rather the VO₂ response follows a temporal pattern than can be reasonably modeled as a single-exponential process for work of moderate intensity (Whipp, 1987). The profile of this response can be partitioned into 3 phases: phase I, phase II, and phase III (see figure 1; Whipp, Ward, Lamarra, Davis, & Wasserman, 1982; Whipp, 1994).

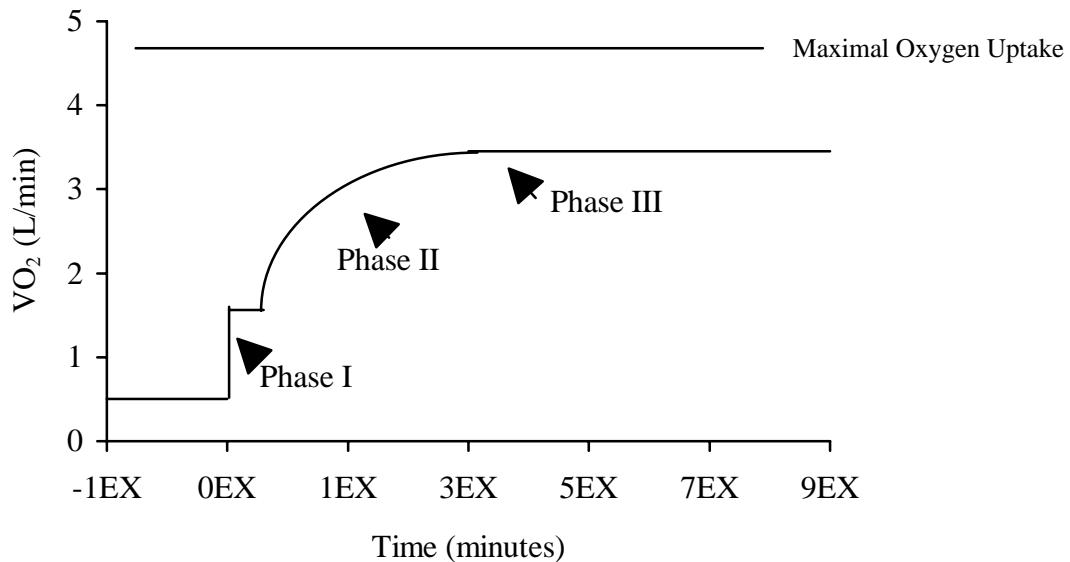


Figure 1. Schematic representing the 3 phases of oxygen kinetics described above. The diagram represents the typical kinetic response for exercise (EX) not accompanied by a sustained increase in [La] or below the LT (EX).

Phase I. Phase I is characterized by an early rapid increase in VO₂ at the start of exercise and is brief in duration, lasting approximately 15 to 20 sec in healthy subjects (Whipp *et al.* 1982; Weissman, Jones, Oren, Lamarra, Whipp, & Wasserman 1982; Sietsema, Daly, & Wasserman, 1989). It is during the initial period of exercise, before mixed venous blood entering

the pulmonary circulation begins to change as a result of increased gas exchange at the site of the active muscles, that the increase in VO_2 is determined by the increase in pulmonary blood flow (Whipp *et al.* 1987). Thus, if the arterial-A VO_2 difference has not increased during this period, it can be inferred that the change in VO_2 provides an indirect estimate of the increase in cardiac output (Whipp *et al.*, 1982; Whipp, 1987; Sietsema *et al.*, 1989). Although it is sometimes difficult to discern the phase I-phase II separation, the magnitude of phase I can be reported as the increase in VO_2 at 15 to 20 sec following the onset of exercise (Sietsema *et al.*, 1989). It can also be quantified by identifying the increase in VO_2 at the time in which a systematic increase in end-tidal PCO_2 and decrease in PO_2 , in association with a decrease in the respiratory exchange ratio (RER), are observed following the onset of exercise (Sietsema, 1992). The above pulmonary gas exchange changes would be indicative of the arrival of mixed venous blood from active muscles; the beginning of phase II.

Phase II. If the adjustment in VO_2 needed to match the energy requirement of the given work rate during phase I, VO_2 continues to increase as a single-exponential process toward steady state for work rates of moderate intensity; this is termed phase II (Whipp, 1994). During this time period, the mixed venous gas concentration of oxygen and carbon dioxide, which continues to change, is reflective of the increase in cellular respiration at the active muscles (Wasserman *et al.*, 1994). The time required for the completion of the phase II response has received a great deal of investigation being described and reported in the literature as: 1) the half-times of a response ($t^{1/2}$) (Diamond, Casaburi, Wasserman, & Whipp, 1977), by time constants, i.e. time it takes for VO_2 to reach 63 percent of the final steady state value (Whipp & Wasserman, 1970; Casaburi, Barstow, Robinson, & Wasserman, 1989; Whipp, 1994), or by

determining the relative size of the O_2 deficit in comparison to the total amount of oxygen required if energy would have been supplied solely by aerobic processes which has been termed the mean response time (Sietsema, Ben-Dov, Zhang, Sullivan, & Sietsema *et al.*, 1989). Whipp and Mahler (1980) have suggested that the increase in VO_2 during phase II is reflective of the rate of oxygen utilization within the muscle (QO_2). In support of their assertion, Barstow (1994) created computer simulated models that elicited time constants that were very similar for pulmonary VO_2 and muscle oxygen uptake (QO_2).

Phase III. Phase III of oxygen kinetics is defined as the steady state of VO_2 (Whipp, 1987, Whipp, 1994). Wasserman *et al.*, (1994) has defined a steady state as a "characteristic of a physiologic system in which its functional demands are being met such that its output per unit time becomes constant. A constant value attained by the system is not sufficient to determine that a system is in a steady state". Thus, according to the definition, it is important to distinguish between VO_2 at steady state and VO_2 in a "limited state" (i.e. $\text{VO}_{2\text{max}}$). When, and if a steady state for VO_2 is attained, the increase in VO_2 from baseline is said to be equal to the increase in the mean rate of muscle O_2 utilization at the muscle (Barstow, 1994).

Intensity Domain and VO_2 Kinetics

The submaximal VO_2 response is distinctive for work rates that are separated by work associated with and without an accumulation of blood lactate (i.e. above and below the lactate threshold [LT]) (Whipp, 1987; Whipp, 1994). For work below the LT (light to moderate exercise intensities), pulmonary VO_2 responds as a single-exponential process (Whipp, 1987). Considering that the time constant of response is invariant with work within this intensity domain (Barstow, 1994), a steady state level of VO_2 can generally be achieved by 3 min (see

figure 2) (Whipp & Mahler, 1980; Wasserman *et al.* 1994), and there is little, or no blood lactate accumulation (Diamond *et al.*, 1977; Whipp, 1994; Wasserman *et al.*, 1994).

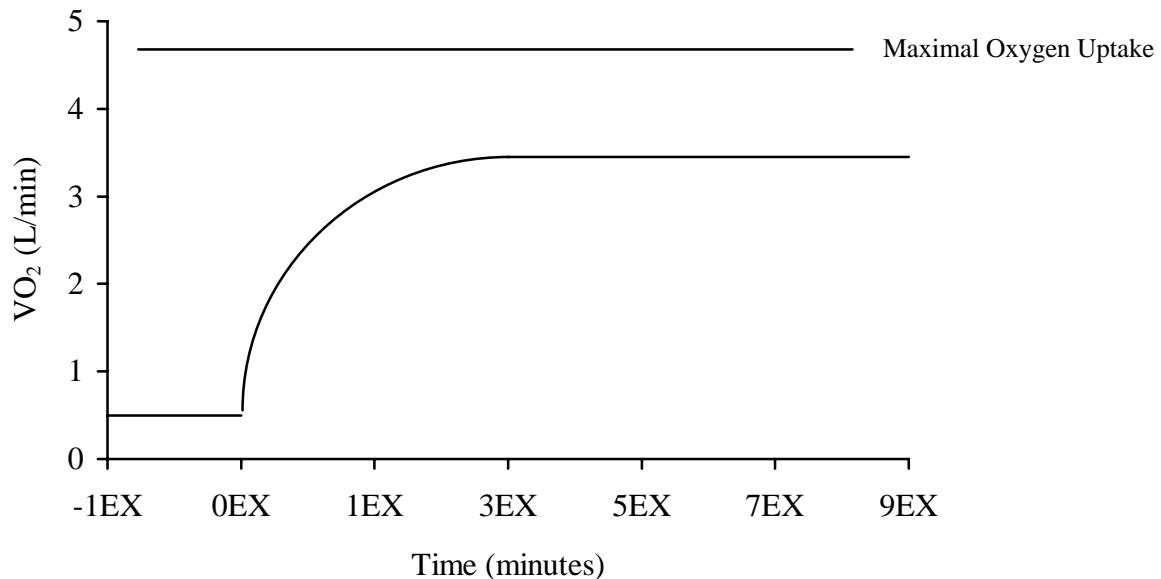


Figure 2. Schematic representing the typical oxygen uptake response for work rates not accompanied by a sustained increase in [La] or below the LT. Note the appearance of a steady state approximately 3 min of exercise (EX).

For work above the LT (heavy exercise), the VO₂ response is more complex, and can no longer be modeled as a single-exponential process (Barstow & Mole, 1991; Whipp, 1987; Whipp, 1994). At least two components, one rapid, and a secondary slower component are needed to describe the VO₂ response (see figure 3) (Whipp & Mahler, 1980; Barstow & Mole, 1991; Whipp, 1994). As the secondary slow component of VO₂ develops during heavy exercise, steady state may be delayed, or not attained at all prior to exhaustion (Whipp, 1994). In certain instances, if the subject can continue to exercise, VO_{2max} or supra-VO_{2peak} levels may be obtained for sub-VO_{2max} work rates (Davis, Ocel, & Craft, *in press*). In cases where a steady state is

eventually attained, the VO_2 response is often greater ($\cong 13 \text{ ml O}_2 \cdot \text{min}^{-1} \cdot \text{Watt}$) than that predicted for sub-LT work rates of moderate intensity work rates ($\cong 10 \text{ ml O}_2 \cdot \text{min}^{-1} \cdot \text{Watt}$) (Whipp & Mahler, 1980; Whipp, 1987; Henson *et al.*, 1989, Whipp, 1994). It was reported that the magnitude of the slow component of VO_2 can reportedly exceed $1 \text{ L O}_2 \cdot \text{min}^{-1}$ (Roston *et al.*, 1987; Poole *et al.*, 1988; Barstow & Mole, 1991, Whipp, 1994, Gaesser & Poole, 1996).

Although Barstow and Mole (1991) have demonstrated that this secondary slow component of VO_2 occurs at approximately 80 to 105 seconds into heavy exercise, being superimposed on the earlier VO_2 response (i.e. phase II), it has typically been defined as the difference between the VO_2 at the end of exercise and at min 3 of the exercise bout (Roston *et al.*, 1987; Casaburi *et al.*, 1995; Womack *et al.*, 1995; Davis *et al.*, in press).

Submaximal VO_2 Responses and Adaptations to Endurance Exercise Training

For work of light-to-moderate intensity (<LT), where no slow component of VO_2 is observed, it is generally reported that, although the speed at which VO_2 approaches steady state may increase following endurance exercise training (Hickson, Bomze, & Holloszy, 1978; Hagberg *et al.*, 1980; Berry & Moritani, 1985; Babcock, Patterson, & Cunningham, 1994), the VO_2 at steady state remains unchanged following endurance exercise training (Davis *et al.*, 1979; Gaesser, 1994).

For work of heavy intensity, in which a slow component of VO_2 is observed, a reduction in submaximal VO_2 has been reported (Gaesser *et al.*, 1991; Belman & Gaesser, 1991; Casaburi *et al.*, 1987, Casaburi *et al.*, 1995; Womack *et al.*, 1995).

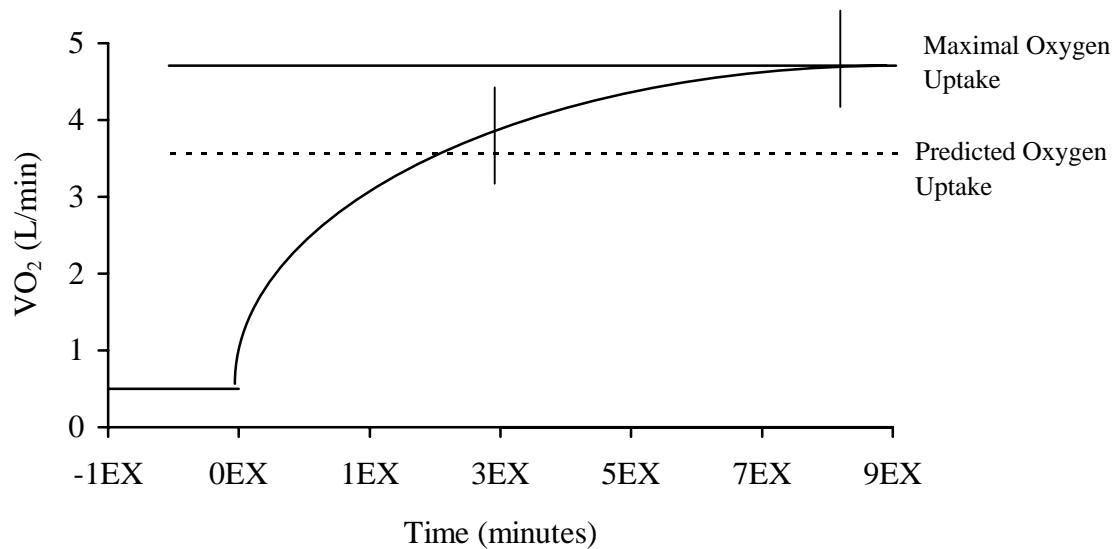


Figure 3. Schematic representing the typical oxygen uptake response for work rates accompanied by a sustained increase in [La] or above LT. Note the actual VO_2 exceeds the predicted VO_2 for that level of exercise (EX)

Prior to the 1990s, Casaburi et al. (1987) were the only group who have published results concerning the effects of endurance training on the "slow component of VO_2 " as defined. This group examined the effect of an 8 wk high-intensity endurance training program on the VO_2 response during heavy exercise on 10 healthy volunteers (six women and four men). Before and after the 8 wk training program ($5 \text{ days} \cdot \text{wk}^{-1}$; $45 \text{ min} \cdot \text{session}^{-1}$), subjects completed a series of 4 constant-load (CL) cycle ergometry bouts of 15 min at the following exercise intensities: 90% of the ventilatory threshold, and 25, 50, and 75 % of the difference between the ventilatory threshold and $\text{VO}_{2\max}$. Following the 8 wk training program, $\text{VO}_{2\max}$ was increased by 15% and a reduction in the slow component of VO_2 was observed only for intensities above the ventilatory

threshold (average reduction 150-200 ml·min⁻¹). The submaximal VO₂ was unchanged for the work rate corresponding to 90 % of the ventilatory threshold.

Belman and Gaesser (1991) reported a trend in a reduction of end-exercise VO₂ during a 6-minute, high-intensity exercise bout on a treadmill following a 8 wk training program (4 days · wk⁻¹; 30 min/session⁻¹). However, unlike Casaburi *et al.*, they posed the question of whether training intensity influenced the VO₂ responses during the high-intensity exercise bout. This study was also unique in that only elderly subjects (age range 65 to 75 yr) were utilized as subjects. Results indicated that regardless of the training intensity (<LT or >LT exercise training), VO_{2max} ($p < 0.02$) and LT ($p < 0.01$) were increased following training when compared to a control group. No differences were observed between the low and high-intensity training groups. The end-exercise VO₂ during high-intensity cycling was reduced to the same degree in both training groups, but failed to reach statistical significance ($p \geq .067$) when compared to the control group. Therefore there was a trend for endurance training lower than the LT to be of sufficient stimulus in reducing end-exercise VO₂ during high-intensity exercise.

Whether adaptations in submaximal VO₂ responses are dependent upon training intensity in a younger population are unknown. To note, in the study by Belman and Gaesser (1991), each subject in the high and low-intensity training groups exercised for a duration of 30 min. In completing the training protocol, high-intensity training subjects performed a greater amount of work than subjects within the low-intensity training group. It was not established whether the adaptations observed in the slow component of VO₂ were dependent upon the training intensity (high or low) or the total amount of work performed.

Temporal changes in Submaximal VO₂ response following Endurance Training

Training-induced reductions in submaximal VO₂ responses to heavy exercise occurred after 2 (Womack *et al.*, 1995), 6 (Casaburi *et al.*, 1995), 7 (Poole *et al.*, 1990) and 8 wk of endurance exercise training (Casaburi et al. 1987; Belman & Gaesser, 1991; Casaburi *et al.*, 1995). The disagreement regarding training-induced changes in the slow component may be explained by the (pre-training) exercise intensity chosen to elicit the submaximal VO₂ response. In other words, it would appear to be easier to observe reductions in the slow component of VO₂ when the magnitude of the pre-training slow component is large (Gaesser, 1994). Additionally, the magnitude of change across the period of training may be different dependent on whether the training intensity was kept constant (Womack *et al.*, 1995) or adjusted as cardiopulmonary adaptations were observed. Definitive characterization of the time course of adaptation to endurance training, and whether training intensity and/or training volume influences this time course and magnitude of adaptation remain to be established.

Recently, Womack *et al.* (1995) reported the temporal effects of high-intensity cycle ergometry training in seven untrained males. The subjects trained on a cycle ergometer $4 \text{ d} \cdot \text{wk}^{-1}$ for 6 wk, with the absolute training workload held constant for the duration of the study. Before training, and following each week of training, the subjects performed a 20 min constant work rate exercise test designed to elicit a pronounced slow component of VO₂. They reported the slow component was adaptations following 2 wk of training (pre-training: $0.42 \pm 0.06 \text{ L} \cdot \text{min}^{-1}$; 2 wk: $0.20 \pm 0.04 \text{ L} \cdot \text{min}^{-1}$). The slow component of VO₂ remained significantly below the pre-training values throughout the study in each week with the exception of 4 wk where the mean value of the slow component of VO₂ was lower. However, unexplained increases in the variability resulted in

no significant difference at 4 wk from pre-training. There were no significant changes in the slow component of VO_2 following 2 wk of training. They found the VO_2 at 3 min to be unchanged. Reporting no change in VO_2 at 3 min of exercise is vital in that it suggests that the reduction in VO_2 can primarily be attributed to the changes in end-exercise VO_2 . It also has relevance in that phase II oxygen kinetics are reported quicker following exercise training. Therefore, if there were a difference, it would be speculative to report whether changes were due to a reduction in the slow component of VO_2 or due to an increase in phase II oxygen kinetics.

Despite these findings, it can be argued that the slow component of VO_2 was not correctly determined in the study by Womack *et al.* (1995). As described earlier, the slow component of VO_2 is defined as the difference between end exercise VO_2 and the VO_2 at 3 min of exercise. However, the 3 min VO_2 by Womack et al. was taken from data at [2:40, 3:00, and 3:20]. At first glance, one may improperly mistake the average of the three measurement for the 3 min VO_2 . However, the average of these three values would give the VO_2 at 2 min and 50 sec, a lower 3 min value for VO_2 , and possibly a larger slow component of VO_2 that could also intrude on phase II kinetics.

Mediators of the Slow Component of VO_2

Although the contribution explained by each the physiologic mechanisms discussed in the proceeding section is currently unknown, it was reported that the contribution arising from within the exercising limbs may account for as much as 86% of the excess VO_2 noted in the region defined as the slow component of VO_2 (Poole, Schaffartzik, Knight, Derion, Kennedy, Guy, Prediletto, & Wagner, 1991). This investigation suggests that the variables such as the additional VO_2 requirement associated with increased ventilatory, cardiac and the stimulatory effect at sites

not associated with the exercising limb (e.g. lactate, catecholamines, or Q_{10} effect via increasing core temperature), cannot be primary mediators of the slow component of VO_2 . Nevertheless, due to the large number of reports demonstrating a relationship between these peripheral mechanisms, a review of all of the potential mediators of this phenomenon is warranted.

Although the physiological mechanisms which have been speculated to account for the slow component of VO_2 , and, in addition mechanisms responsible for training-induced reductions in the slow component of VO_2 are poorly understood (Whipp, 1994; Gaesser, 1994), factors that are believed to contribute to the slow component of VO_2 include: 1) excess VO_2 associated with increased core and exercising muscle temperature during exercise (Q_{10} effect); 2) the increase in VO_2 involved with respiratory muscle work; 3) metabolic stimulatory effect of circulating catecholamines; 4) reduced efficiency caused by progressive recruitment of fast-twitch muscle fibers; 5) increased energy requirement of lactate metabolism in its conversion to glycogen in the liver and muscle; 6) accumulation and role of the lactic acidosis in maintaining PO_2 which facilitates oxygen unloading from hemoglobin as a result of the Bohr effect; and 7) the release and elevation of blood potassium concentration.

Increased core and muscle temperature (Q_{10} effect). The rate of enzymatic reactions are extremely sensitive to changes in temperature. In the body, the effect of temperature on enzymatic reactions is studied by changing the temperature in multiples of $10^\circ C$. The resulting change in the rate of reaction is termed a “ Q_{10} effect”. For example, if increasing the temperature $10^\circ C$ doubles the rate of a particular enzymatic reaction, the Q_{10} would equal two (Brooks and Fahey, 1984).

It is undisputed that both core and muscle temperatures increase during exercise (Hagberg, Mullin & Nagle, 1978; Casaburi *et al.*, 1987; Poole *et al.*, 1988). However, debate exists regarding the relative contribution, if any, that an increase in core or muscle temperature may have in the increase in VO_2 via a Q_{10} effect noted in exercise. In an early study, Hagberg *et al.* (1978), reported that increases in core temperature may account for 31% in the increase in VO_2 noted between minute 5 and 20 of exercise. However, Poole *et al.* (1991) suggest that, although small contributions may exist that are associated with peripheral mechanism(s), the primarily mechanism for the slow component of VO_2 ($> 86\%$) is located within the exercising muscle itself. Thus, the contribution of increasing core temperature to the slow component of VO_2 is believed to be relatively small.

Contrary to recent evidence that core temperature has little, or is not a contributing factor to the slow component of VO_2 , it was reported that increasing muscle temperature within the exercising limb may significantly contribute to the slow component of VO_2 . In the same experiment by Poole *et al.* (1991), the authors determined that at the end of exercise, a 1°C rise in femoral venous blood temperature could account for approximately 39% of the slow component of VO_2 , assuming a Q_{10} of 2.5.

In addition, Willis and Jackman (1994) studied the effect of increased temperature on mitochondria function. They contend that the slowly increasing VO_2 observed in the active limb during exercise above the LT implies that “either the efficiency of ATP utilization is decreasing, or that mitochondrial ADP/O is falling, or both.” The ADP/O ratio can be defined as the proportional breakdown and production of mitochondrial ATP, which requires a proportional rate of oxygen consumption. They found that elevations in muscle temperature decrease the

efficiency of the coupling between oxygen consumption to ATP production, and could account for 300-400 milliliters of oxygen per minute. Thus, according to these authors, increasing muscle temperatures may be a large contributor to the slow component of VO_2 .

Conversely, increased core and muscle temperatures were reported without a change in VO_2 (Poole *et al.*, 1988; Gaesser & Poole, 1996). However, it could be argued that as levels of work where an increase in VO_2 is not observed suggest that coupling efficiency is not decreased. Thus, no additional oxygen is required.

Increased VO_2 involved with respiratory muscle work. Although, as mentioned previously, the data from Poole *et al.* (1991) suggest that the primary mechanism for the slow component of VO_2 ($> 86\%$) is located within the exercising muscle itself, many report that the energy cost associated with respiration may contribute to the added oxygen requirement. As the ventilatory rate increases, so must the work of breathing and work of the associated muscles.

However, a problem exists in that it is difficult to quantify the estimated energy cost of breathing, which depends on the ventilatory rate, and is highly variable in healthy subjects (Aaron, Seow, Johnson & Dempsey, 1992). The cost of ventilation was approximately 1.79 ml O_2/L for ventilatory rates between 63-79 $\text{L}\cdot\text{min}^{-1}$, and 2.85 ml O_2/L for ventilatory rates between 117-147 ($\text{L}\cdot\text{min}^{-1}$).

In the study by Poole *et al.* (1991), the difference in the mean ventilation between end exercise and 3 min was $56.8 \text{ L}\cdot\text{min}^{-1}$. Assuming a value of 2.85 ml O_2/L , the increase in ventilation required an additional oxygen requirement of 162 ml O_2/L , which represented 23% of the slow component of VO_2 . Womack *et al.* (1995) recently reported that the contribution of increased ventilation was approximately 20%, and consequently reported that it was in

agreement with data reported by Poole *et al.* (1991). However, in this study the authors did not utilize the cost of ventilation to be 1.79 ml O₂/L (ventilatory rates between 63-79 L·min⁻¹) or the 2.85 ml O₂/L (ventilatory rates between 117-147 L·min⁻¹). These authors chose ≈2 ml O₂/L, thus lower than that used by Poole *et al.* (1991). In an earlier report by Hagberg *et al.* (1978) it was reported that the increased cost of ventilation accounted for 30% and 81% of the increased VO₂, at 65 and 85% of VO_{2max}.

Stimulatory effect of circulating catecholamines. Catecholamines, and namely epinephrine, have been suggested as possible contributors to the slow component of VO₂ (Casaburi *et al.*, 1987; Gaesser & Poole, 1988; Poole *et al.*, 1990; Patterson and Whipp, 1991; Gaesser, Ward, Baum, & Whipp, 1994, Gaesser, 1994). The epinephrine role is secondary to the calorogenic effects which are responsible in stimulating lipolysis and glycogenolysis. Plasma concentrations of epinephrine increase during exercise, with a substantial increase during exercise at work rates that elicit a pronounced slow component of VO₂ (e.g. heavy and severe exercise intensities) (Poole *et al.*, 1988; Poole *et al.*, 1991).

However, recent reports have failed to observe an effect on exercise VO₂ following epinephrine infusion (Gaesser, 1994). It was suggested that studies failing to observe an increase in VO₂ following epinephrine infusion may be flawed in that the exercise intensities utilized were too low (< 50% VO_{2max}) and that the infusion of epinephrine did not raise epinephrine levels to those noted during maximal exercise.

To resolve this issue, Gaesser *et al.* (1992) exercised subjects at a work rate > LT. After epinephrine infusion an increase in exercise VO₂ was not observed following 10 to 20 min of

cycle ergometry. This was noted despite raising epinephrine levels approximately 4 times higher than at rest.

In addition, Womack *et al.* (1995) performed epinephrine infusion in subjects following 6 wk of cycle ergometry training. Although significant reductions in plasma epinephrine occurred following 2 wk of training, the results of the epinephrine infusion suggest that the drop in epinephrine levels was not responsible for the reduction in the slow component of VO_2 . Infusion of epinephrine following 6 wk of training on plasma epinephrine levels that exceeded the pre-training plasma epinephrine levels by six fold, and the 6 wk end-exercise level by 16-fold. Despite the increase in plasma epinephrine, exercise VO_2 was unaffected.

Further evidence which seems to refute increases in plasma epinephrine as a contributor to the slow component was reported by Davis *et al.* (1994). Propranolol, a nonselective beta antagonist was administered to the subjects prior to exercise. The authors concluded that the magnitude of the slow component was unaffected following beta blockade.

Reduced efficiency caused by progressive recruitment of fast-twitch muscle fibers. The metabolic and bioenergetic differences between slow- and fast-twitch motor fibers are well documented (Crow and Kushmerick, 1982; Kushmerick, Meyer, and Brown, 1992) and may support the concept that the slow component of VO_2 may arise from these physiologic processes. Evidence that may support this hypothesis include reports by Coyle *et al.* (1992) and Gaesser *et al.* (1992). Both demonstrated that the greater the oxygen cost of cycle ergometry exercise, the greater the recruitment of fast-twitch motor fibers. In the study by Gaesser (1992) it was determined that the slow component was greater cycling at 100 versus 50 revolutions per minute, despite equivalent work rates. To specifically address the relationship between muscle motor

fiber recruitment and the slow component of VO_2 , Shinohara and Moritani (1992) concluded that motor fiber recruitment and firing, reflected by integrated electromyogram, was positively correlated with the rise noted in VO_2 . The slow component of VO_2 may have been due to the recruitment of more muscle units, and in particular fast-twitch, during the exercise bout".

In addition, Willis *et al.* (1994) recently conducted experiments in which the investigators studied the composition and function of isolated mitochondria of one particular skeletal muscle fiber type. They concluded that activation of type IIb muscle recruits mitochondria with slower phosphate kinetics and lower economy of oxygen utilization ($\approx 18\%$) due to a greater potential to utilize α -glycerophosphate type I fibers. These conclusions are based on reports that type IIb muscle possesses over 10-fold higher α -glycerophosphate concentrations than type I muscle, and because the α -glycerophosphate pathway is FAD-linked, the ADP/O ratio is 18% lower in the mitochondria of type IIb muscle.

Increased in lactate metabolism. Of all the potential mediators to be investigated, [La] has received the greatest attention. The accumulation of lactate during exercise may stimulate gluconeogenesis or glycogenolysis which would be an oxygen requiring process, this elevating VO_2 .

As mentioned previously, the slow component of VO_2 is only noted at exercise intensities in which an increase in [La] is noted. In addition, several authors have documented a high correlation between the slow component of VO_2 , or end exercise VO_2 , and the rise in [La] at a number of work rates (Roston *et al.*, 1987; Poole *et al.*, 1988; Wasserman *et al.*, 1994). These reported relationships appear to increase the greater the distance the work rate is above the LT.

Roston et al. (1987) reported a high correlation ($r = 0.88$) between the slow component of VO_2 and the change in blood [La]. In addition, it was reported that infusion of lactate increases VO_2 during exercise (Ryan, Sutton, Toews and Jones, 1979). Infusion of lactate increased blood [La] from 3.9 mM to 5.3 mM, resulting in an increase in exercising VO_2 of 129 $\text{ml} \cdot \text{min}^{-1}$.

Despite the high correlation demonstrated between the changes in [La] and the slow component of VO_2 , many have suggested that, at best, it may play an insignificant role in the elevation of VO_2 . The energy cost, or additional VO_2 requirement of glycogen resynthesis from lactate in the liver was estimated to be minimal (Whipp, 1987; Whipp, 1994).

Lactic acidosis in maintaining PO_2 which facilitates oxygen unloading for hemoglobin as a result of the Bohr effect. It has been argued by Wasserman *et al.* (1994) that perhaps the most important role of blood [La], is the role it plays during exercise in facilitating the dissociation between oxygen and hemoglobin. The reduction in pH due to the increase in [La] would shift the oxyhemoglobin curve to the right. In doing so, the AVO_2 difference would increase and greater oxygen would be supplied to the tissues. As a result of the Bohr effect, the rightward shift in the oxyhemoglobin dissociation curve is observed. This would facilitate the unloading of oxygen at the tissue level. Thus, Wasserman *et al.* (1994) contend that it may not be lactate per se, and the energetic cost of resynthesizing glucose, but rather the accompanying lactic acidosis which creates the shift in the oxyhemoglobin dissociation curve, and further unloading of oxygen.

Release and elevation of blood potassium concentration. It remains unclear whether changes in blood potassium concentrations and the slow component of VO_2 are related (Poole & Gaesser, 1996). During exercise, the plasma concentration of potassium increases (Yasuda, Ishida, and Miyamura, 1992). When frog muscle is incubated in a solution containing a high

potassium concentration, the metabolic rate is notably increased (Barnes, 1988). There are a limited number of studies reporting the relationship between of potassium concentration and the slow component of VO_2 in exercise. Although Yasuda *et al.* (1992) reported an extremely high relationship (0.93-0.98) between exercise VO_2 and blood potassium concentration is 6 subjects, by no means did this show that potassium was a causative factor. These authors failed to examine the potassium concentration at specified times period throughout exercise. Poole *et al.* (1991) reported that potassium concentrations increased abruptly at the onset of exercise. However, after 3 min the concentration of potassium remained relatively stable, in the same period where a slow component of VO_2 was $700 \text{ ml} \cdot \text{min}^{-1}$.

Summary

Despite accumulating evidence demonstrating the existence of the slow component of VO_2 , it has received little attention in the physiologic literature. The purpose of this review was to provide the reader with a) background on the nature and extent to which physiologic changes occur with physical training, b) a review of the history of gas exchange kinetics and the slow component of oxygen uptake, c) a discussion of potential mediators of the slow component of VO_2 , and d) a definition of what is currently known about adaptations in oxygen kinetics, namely the slow component of VO_2 , following exercise training.

The causes of the slow component and the physiological mechanism responsible for the adaptation of the slow component of VO_2 with endurance exercise training have yet to be completely understood. Current evidence by Poole *et al.* (1991) suggest that the primarily source is located within the exercising muscle itself. However, it is unclear what single mechanism is

the primary contributor to the slow component of VO_2 . In addition, debate continues on whether peripheral mechanisms, such as pulmonary ventilation, are potential primary contributors to the slow component of VO_2 .

Chapter III

Adaptation of the Slow Component of VO₂ Following High or Low Intensity Exercise Training

Running Head: Slow Component and Endurance Training

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ABSTRACT

Eighteen untrained males [age: 23 ± 0.6 yr (SEM)] were randomized into high intensity (HIT: above lactate threshold, LT), moderate intensity (LIT: below the LT) or no training (NT) groups. Subjects trained on a cycle ergometer 4 days \cdot wk $^{-1}$ for 6 wk with the power output held constant. Maximal cycle ergometry was performed before and after the training period to determine changes in power output and oxygen consumption (VO_2) at the LT and peak exertion. Before training and after 1, 2, 4, and 6 wk, subjects performed high constant-load (HCL) cycling bouts to quantify training adaptations in the SC. Training was designed to keep total work equivalent between the HIT and LIT groups. Increases in power output and VO_2 at LT and peak exercise after 6 wk were noted in the HIT and LIT groups in comparison to NT group ($p<0.05$). No differences were noted between HIT and LIT. Two-way repeated measures ANOVA revealed a significant trial*group interaction for adaptation in the SC ($p<0.001$). After 1wk of training, a significant reduction in the SC was noted for HIT [mean \pm SEM]: (pre-training (PT): 703 ± 61 ml \cdot min $^{-1}$; 1 wk: 396 ± 60 ml \cdot min $^{-1}$) (- 44% from PT). Further adaptation for the HIT was also noted at 4 wk: 202 ± 45 ml \cdot min $^{-1}$ (-71% from PT). For LIT, a significant reduction was noted at 2 wk (PT: 588 ± 76 ml \cdot min $^{-1}$; 2 wk: 374 ± 50 ml \cdot min $^{-1}$) (-36% from PT). Further adaptation for LIT group was noted at 6 wk (252 ± 38 ml \cdot min $^{-1}$) (- 57% from PT). Adaptation in SC was not noted at any interval for NT. Temporal changes in blood lactate ($r = 0.40$) and ventilation ($r = 0.72$) were significantly correlated with the changes for SC over the 6 wk training period ($p<0.05$). In conclusion, it was demonstrated that training at supra-LT and sub-LT intensities produces similar improvement in VO_2 and power output at peak exercise and in the LT, when total work output is controlled. However, training at supra-LT intensity promotes larger and faster adaptations in the SC than training at the sub-LT levels.

INTRODUCTION

For power outputs below the point where an accumulation in blood lactate is noted, i.e. the lactate threshold (LT), the dynamic pulmonary oxygen uptake (VO_2) response is described as a monoexponential process (14, 26, 28, 29). Considering the time constant of response is uniform for all power outputs within this domain of exercise, the VO_2 response increases as a linear function of work rate and the steady state level for VO_2 is attained by approximately 3 min (12, 15, 26, 27). At power outputs $>\text{LT}$, the VO_2 response becomes more complex and no longer can be described as a linear function of work rate (17, 28, 29). At least two components, one rapid, and a secondary slower component are needed to describe the VO_2 response to work rates $>\text{LT}$ defined as “heavy” or “severe” exercise (14, 28, 29, 32). A steady state for VO_2 may be delayed, or not attained at all prior to subject fatigue (9, 14, 24, 29). If a steady state for VO_2 is attained during exercise, the VO_2 response is greater ($\cong 13 \text{ ml O}_2 \cdot \text{min}^{-1} \cdot \text{Watt}$) (12, 26, 27) than the linear relationship described with sub-LT power outputs ($\text{VO}_2 \cong 10 \text{ ml O}_2 \cdot \text{min}^{-1} \cdot \text{Watt}$) (12, 28, 29). The magnitude of this additional energy requirement, recently titled “the slow component of VO_2 ” (SC), is not trivial. It is reported that, in extreme instances, the magnitude of the slow SC can exceed 1 to $1.5 \text{ L O}_2 \cdot \text{min}^{-1}$ (4, 14, 21, 24, 28, 29).

For power outputs $>\text{LT}$, adaptation in the SC is reported following endurance exercise training (5, 6, 7, 20, 32). These studies have primarily focused on the effects of high-intensity training on adaptations submaximal VO_2 response [either end-exercise VO_2 or the SC]. Adaptation in the SC was reported after exposure to endurance training periods of 2 (32), 5 (7), 7 (20), and 8 wk (5, 6). The magnitude of the pre-training SC in these studies, although not trivial, are considerably less than the SC, i.e. 1 to $1.5 \text{ L O}_2 \cdot \text{min}^{-1}$, that have been reported in the

literature (4, 14, 21, 24, 28, 29). The effects of training on the SC which approach these larger magnitudes have yet to be described.

Unlike previous studies, Womack *et al.* (32) were first to specifically report a pattern of temporal changes in the SC resulting from 6 weeks of high-intensity cycle ergometry training. Their primary focus was to test several hypothesized mediators of the SC over the course of training. Significant adaptations in the SC were noted at 2 wk of training with no additional adaptation thereafter. It was concluded that adaptations in the SC occurred rapidly, and was essentially complete after 2 wk of training.

Despite efforts to explain the physiological attributes of the SC (14, 22), limited evidence exists to demonstrate the effect of exercise training intensity on adaptations of the SC. Casaburi *et al.* (7) reported that moderate intensity exercise, i.e. 80% of the ventilatory threshold, produced similar adaptations for subjects tested at both submaximal and maximal loads when compared to a group performing high-intensity exercise (>LT) after 5 wk of training. No attempt was made to describe the temporal adaptations over the 5 weeks. Gaesser *et al.* (12) reported improved capacity for high-intensity exercise after what was termed “very-low” intensity exercise training. The clinical relevance to fully understand the magnitude and temporal adaptation which occur in the SC with alternative training regimens, namely exercise intensity, needs to be addressed. Clinical populations, such as those with cardiac or pulmonary limitations, may have a limited ability to increase peak VO₂ secondary to lower or sub-threshold training intensities needed to stimulate cardiopulmonary and metabolic adaptations to exercise training. However, these patients may still attain beneficial effects from exercise training by reducing the magnitude of the SC for a given work rate, thus extending the time to fatigue. This has relevance if one considers

that many of these patients may exhibit large SC performing simple activities of daily living. However, it remains unknown whether adaptations in the SC are realistic for lower intensities, how quickly they occur, or if a “threshold level” is needed to facilitate adaptations.

Thus, the purpose of this current study was to determine both the time course and magnitude of adaptation in the SC after 6 wk of high (<LT) or moderate-intensity (>LT) endurance exercise training. In addition, the association of potential mediators of the SC were examined.

METHODS

Subjects

Eighteen healthy adults males volunteered as subjects. After an explanation of the procedures, subjects signed an informed consent document approved by the Institutional Review Board for research involving human subjects at the University. Only subjects who had not participated in regular physical activity ($<2 \text{ day} \cdot \text{wk}^{-1}$ and $< 40 \text{ min} \cdot \text{day}^{-1}$) for at least 3 months prior to the study were eligible to participate in this study.

Maximal Cycle Ergometer Testing

Before and after the 6 wk endurance training program subjects performed a maximal cycle ergometry test to exhaustion. Both tests were performed on a calibrated MedGraphics CardiO₂ stationary cycle ergometer (Medical Graphics Corporation, St. Paul, MN). All subjects were tested at the same time of day before and after training testing, and in a 4-hr post-absorptive state. The pre- and post-training tests were conducted 2 days prior to administration of the initial constant-load (CL) exercise bouts and 2 days following the final CL exercise bout, respectively. Testing consisted of a 5 min rest period followed by exercise with a continuous rate

of increase in power output starting at 0 and set to increase at $20 \text{ W} \cdot \text{min}^{-1}$. Testing was terminated if the subjects were unable to continue pedaling at a rate above 40 rpm. Peak $\text{VO}_2 (\text{L} \cdot \text{min}^{-1})$ was determined as the highest VO_2 achieved during the final minute of exercise. A blood sample (0.5-1.0 ml) was collected at rest and at the end of each minute through an indwelling venous catheter located in the forearm for determination of blood lactate concentration [La]. One 1.0 ml waste aliquot of blood was drawn and discarded prior to each of these samples to clear saline from the line and eliminate contaminants from the prior sample. All blood samples were collected in vacutainers that contained potassium oxalate to prevent further glycolysis and sodium fluoride to prevent coagulation. The samples were placed in an ice slurry and analyzed immediately. The LT was determined by three independent investigators who examined the relationship between [La] and power output. The LT was defined as the breakpoint in the linear relationship of blood [La] to VO_2 during the continuous ramping protocol (15, 27, 32). An increase in [La] of at least $0.2 \text{ mmol} \cdot \text{L}^{-1}$ was required for the determination of the LT. In the instances where the three investigators did not agree, the average value was utilized.

Constant-load Exercise Testing

Prior to the randomization to groups, subjects performed a high intensity constant-load (HCL) and a low intensity constant-load (LCL) cycle ergometry exercise bout at two designated power outputs selected on the basis of the pre-training maximal exercise tests. Subjects completed the HCL exercise bout initially and then, following a 30 min recovery period, completed the LCL bout. The power output for the HCL test was designed to correspond to a heavy/severe exercise intensity. The power output was set to equal [power output at LT + .75 (power output at Peak - power output at LT)](LT+75%). This intensity was reported to elicit a

pronounced SC (7). The power output for the LCL bout was designed to correspond to a moderate exercise intensity (< LT). The power output was set to equal [power output at LT * .90] (90%LT). During the 6 wk training period, only the HCL tests were repeated (wk 1, 2, 4, and wk 6) and power outputs for these tests were held constant at these intervals. All HCL testing was conducted at the same time of day. Subjects were not allowed to train on the day prior to their scheduled HCL test. For all constant-load testing , 5 min of baseline cardiopulmonary data was collected. At 5 min, cycling began immediately at the designated power for a duration up to 15 min of exercise. When subjects were unable to complete the full 15 min, the time was noted, and that time endpoint was used for all subsequent HCL testing.

For all testing, pulmonary gas exchange variables were measured continuously using the Medical Graphics CPX/D system (Medical Graphics Corporation, St. Paul, MN) for determination of VE, VO₂, and VCO₂. Blood [La] utilizing whole blood was measured utilizing the YSI Model 1500 Sport Lactate Analyzer (Yellow Springs, OH).

Exercise Training

Following the initial testing, subjects were randomly assigned to either: 1) high-intensity exercise training (HIT), 2) moderate intensity training (LIT), or no training (NT). The endurance training program consisted of supervised exercise on a stationary cycle ergometer (Monarch E 818 cycle ergometer). Ergometers were calibrated weekly. In addition to completing the HCL test on the first day of each week, subjects trained an additional 4 daywk⁻¹. Subjects in the LIT and HIT groups trained at 90% LT and LT+75%, respectively. The power output was kept constant throughout the 6 wk of training for each subject, and duration of the exercise sessions was specified to keep total work constant between the two training groups. All subjects in the

LIT group trained for 60 minute · session⁻¹. The average duration (mean \pm SEM) for subjects in the HIT group was 25.3 min \pm 1.7. All subjects were discouraged from engaging in physical activity outside of the program. Attendance for the training sessions averaged 92% for both the HIT and LIT training groups.

Statistical analyses

A two-way analysis of variance with repeated measure was used to determine the overall effect of training on the SC. The SC was defined and calculated by subtracting the end-exercise VO₂ from the VO₂ at the third minute of exercise (6, 7, 9, 14, 32). The reliability of defining the SC in this manner was reported to be high ($r = 0.91$) (9). When significant group, trial, or group \times trial interactions were detected, simple effects testing was performed. Tukey's HSD test was used post-hoc to identify specific group differences. Statistical significance was accepted if $p \leq 0.05$. Dispersion around the mean values are expressed as \pm SEM. Linear regression was used to assess the relationship between changes in the SC and training induced changes in V_E and [La]. The magnitude of change for both V_E, defined as the end exercise value for V_E less the value at 3 min of exercise. For La, the end-exercise value was taken from the pre- and post-training HCL tests and used in the analysis as the index of training induced change in this measure. Improvements in VO₂ and power output at peak and at the LT were assessed by using change scores based on the pre-training and post-training test results, i.e. $\Delta\text{VO}_2 = \text{Post-training VO}_2 - \text{Pre-training VO}_2$. One-way analysis of variance on the change scores was used to determine the effects of training on these measures. When significant group differences were detected ($p < 0.05$), Tukey's HSD was used to test for differences among the groups.

RESULTS

Physical characteristics of the subjects are presented in Table 1. As reported, the groups were well matched. The effects of the 6 wk endurance training program on VO₂ and power output at the LT and maximal exercise are reported in Table 2. Exercise training resulted in an increase in the peak VO₂ and peak power output as well as in the VO₂ and power output at the exercise intensity where LT occurred in both HIT and LIT groups in comparison to the NT group. No significant difference was noted between the HIT and LIT groups. In addition, no significant group differences noted for peak exercise responses which are often cited to support that peak effort was elicited in establishing a maximal value for pulmonary VO₂ uptake, i.e. no differences in peak [La], peak RER, peak heart rate, or peak RPE ($p>0.05$).

Table 3 shows the differences in the training program among the groups, including features of power output, intensity, and duration, as well as the responses for heart rate and oxygen uptake based on the initial HCL and LCL bouts. To note, the exercise training stimulus for the LIT group appears to exceed what has been previously reported (2, 3) as the “minimal threshold intensity” at which a training adaptations occur. Although, this concept continues to be a subject of great debate.

Table 4 shows the effect of training for each group from the pre-training HCL to the testing performed after wk 6. For each variable, namely end-exercise VO₂, VE, heart rate and lactate, the training groups experienced a significant reduction from the pre-training HCL test. No change was noted in the NT group. To note, the mean exercise VO₂ for the HCL test met, or surpassed, the peak VO₂ value from the baseline ramp test for all three groups. This event is not exclusive but has been noted by other investigators (7, 9)

The means values \pm SEM for the SC following each week of training are shown in Figure

1. For the HIT group, a significant reduction in the SC was noted following 1 wk of training (pre-training (PT) = $703 \pm 60 \text{ ml} \cdot \text{min}^{-1}$, 2 wk = $396 \pm 60 \text{ ml} \cdot \text{min}^{-1}$). Further adaptation in the SC was noted between at 4 wk ($202 \pm 45 \text{ ml} \cdot \text{min}^{-1}$). No additional adaptation in the SC was noted thereafter. Adaptation in the SC was noted in the LIT group, however significance was not apparent until 2 wk of training (PT = $588 \pm 80 \text{ ml} \cdot \text{min}^{-1}$, 2 wk = $374 \pm 50 \text{ ml} \cdot \text{min}^{-1}$). Further adaptation in the SC for the LIT group was noted at 6 wk of training ($252 \pm 38 \text{ ml} \cdot \text{min}^{-1}$). No changes in the SC were noted in the NT group. The VO_2 at 3 min was not significantly different over the 6 wk training period for all three groups suggesting that the reduction in the SC was primarily attributable to a reduction in end-exercise VO_2 .

The relationships between the SC and end-exercise [La] and V_E over the course of the 6 wk training program are presented in Figures 2 and Figures 3, respectively. Reduction in both end-exercise [La] ($r = 0.40$) and ΔV_E ($r = 0.72$) were significantly correlated with adaptations in the SC over the course of training.

DISCUSSION

The results of this study demonstrate that adaptations in the SC occur following both high ($>\text{LT}$) and moderate intensity training ($<\text{LT}$). The results in the HIT group demonstrate a rapid adaptation in response to training, with a an average reduction of $307 \text{ ml} \cdot \text{min}^{-1}$ achieved after 1 wk. This represented a 44% reduction in the pre-training SC. Although adaptations occurred earlier in training (1 wk), it is in agreement to that demonstrated previously by Womack *et al.* (32) who reported a rapid reduction (2 wk) in the SC following high-intensity exercise training.

Thus, the corroboration of these two studies would suggest that adaptations in the SC following high intensity training occur earlier than previously reported (5, 6, 7, 20).

A number of differences were noted between the current investigation and the recent study by Womack *et al.* (32). The magnitude of the pre-training SC were much larger in the present study ($703 \text{ ml} \cdot \text{min}^{-1}$ vs. Womack *et al.*, $420 \text{ ml} \cdot \text{min}^{-1}$) (32). The differences in the magnitude of the SC between the two studies may possibly be explained by the intensities utilized for the HCL testing. Our group chose to use an intensity corresponding to LT+75%, whereas LT+60% was used by Womack *et al.* (32). In addition, although both studies report a rapid adaptation in the SC, the current study reported a significant reduction 1 wk earlier, and further adaptation in the SC occurring after 4 wk of training. The results of the present study also differed from those who reported changes at 5, 7, or 8 wk (5, 6, 7, 18). These authors may not have expected to observe an adaptation in the SC or end-exercise VO_2 so rapidly, or the temporal adaptations in the SC over the course of training were not a major focus of the study.

The magnitude of our pre-training SC was much larger ($\approx 280 \text{ ml} \cdot \text{min}^{-1}$) than that reported by Womack *et al.* (32). A reduction in the magnitude of the SC to a “hypothetical threshold level” ($\approx 200 \text{ ml} \cdot \text{min}^{-1}$) was not noted until after 4 wk of training for the HIT group in the present study. No further reduction was noted despite continued training at an intensity >LT. A level of $\approx 200 \text{ ml}$ was noted after 2 wk of training in the report by Womack *et al.* (32). Subjects in HIT group trained at a higher fraction of their peak VO_2 than was the case for subjects in previous studies (32) who trained subjects at an intensity of 70% of their initial peak VO_2 for 40 min. Subjects also performed six 5 min intervals twice a week at the workload

utilized for the HCL testing. Casaburi *et al.* (7) trained at an intensity equivalent to 50% of the difference between the ventilatory threshold and peak, and like our study, controlled for total work. For this study, similar reductions in the SC were noted. Thus, exercise training at a higher intensity could have resulted in a more rapid adaptation, and further reduction in the SC later in training.

Subjects training in the LIT group experienced a slower adaptation (2 wk of training) in the SC when compared to HIT, however they continued to show a reduction through 6 wk of training. To note, 6 wk SC (252 ml) was significantly lower than the SC after 1 wk of training (472 ml). This, even in the LIT group we were able to detect adaptations somewhat early in training, and continued to see a reduction despite the lower intensities (<LT). The magnitude of reduction after 2 wk of training was smaller than that found for our HIT group at the 2 wk interval ($214 \text{ ml} \cdot \text{min}^{-1}$) and was comparable to that demonstrated after 5 wk of training at lower (80% LT), but similar training intensity (7). Casaburi *et al.* (7) made no attempt was made to investigate the temporal changes in the SC during 5 weeks of endurance exercise training. The final SC at 6 wk (252 ml) represented a 57% reduction in the pre-training SC for the LIT group. The magnitude and speed at which adaptations occurred in the SC were larger and faster in the HIT group. Following 2 wk of training, the HIT training group showed a reduction of $410 \text{ ml} \cdot \text{min}^{-1}$ in the SC, whereas the LIT group showed a reduction of only half as much ($210 \text{ ml} \cdot \text{min}^{-1}$). Thus, despite the fact that the pre-training SC of VO_2 in the HIT group of $\approx 120 \text{ ml} \cdot \text{min}^{-1}$ greater than the LIT group, after 1 wk of physical training the mean SC for the HIT group was lower than the LIT group.

Casaburi *et al.* (7) demonstrated improvements in peak VO_2 , however no change in VO_2 at the ventilatory threshold, VT for subjects training 5 wk at 80% VT. In the present study, both HIT and LIT groups experienced similar improvements in VO_2 and power output at peak exercise and at the LT when compared to the NT group. In addition, no differences were noted between the magnitude of adaptation in the training groups. Similar results have been reported by others regarding the effects of high versus low intensity training on peak VO_2 at LT (5, 6, 7, 27). Thus, when controlling total work output, exercise at levels < LT produce similar training adaptations in peak exercise parameters such as LT and peak VO_2 vs. exercise levels > LT. Although adaptation in SC appears to occur at both levels of training, training at levels > LT result in quicker and larger adaptations as a result of training even when total work is controlled.

The changes in end-exercise $[\text{La}]$ ($r = 0.40$) and ΔV_E ($r = 0.72$) were significantly correlated with the reduction in the SC over the course of training. Although Poole *et al.* (19) has suggested that the primary mechanism ($\approx 85\%$) for the SC is found within the exercising limb, other reports suggest that additional contributions could be attributed to central variables such as ventilation (6, 16, 32). However, despite this moderately-high correlation, it is unlikely that a change in V_E after training is a predominant mediator that account for the corresponding adaptation of the SC in either group. The total pre-training oxygen cost of exercise ventilation may be as high as $\sim 2.5 \text{ L} \cdot \text{min}^{-1}$ for the ventilatory rates observed in our subjects (1). The reduction in V_E at 6 wk of training in the HIT group might account only for 110 ml reduction in VO_2 (21%) of the 520 ml decrease observed in the SC. For subjects in the LIT group, the difference in ΔV_E from pre-training to wk six in the LIT group was $28.5 \text{ L} \cdot \text{min}^{-1}$. This would

only account for 71 ml reduction (21%) out of the total 336 ml decrease in the SC seen in this group.

Of the mechanisms postulated as primary mediators responsible for generation of the SC, [La] has attracted the most attention. The SC is primarily confined to power outputs exceeding the LT. Thus it would appear to reason that the SC is either caused or at least linked to blood lactate accumulation, i.e. LT). In addition, since the buffering of lactate stimulates VE (26) an association between lactate and the SC could explain the relation between VE and the SC. Roston *et al.* (24) demonstrated a correlation between the SC and changes in [La] at a number of power outputs ($r = 0.88$). In addition, it was reported that decreases in the slow component of VO_2 following training were correlated with changes in end-exercise [La] ($r = 0.64$) (6). An additional physiologic mechanism involving a possible link between [La] and the SC includes an increased [La] during exercise which may stimulate glycogen resynthesis; an oxygen requiring process. However, the energy cost associated with the resynthesis of lactate in the liver is reported to be small ($< 60 \text{ ml} \cdot \text{min}^{-1}$) (14, 28, 29). Stringer *et al.* (25) report that a reduction in pH due to the increase in [La] would shift the oxyhemoglobin curve to the right. In doing so, the arterial-venous oxygen difference would increase and a greater amount of oxygen would be unloaded to the tissues. Thus, the energetic cost in the resynthesis of lactate may contribute little to the SC. However the accompanying lactic acidosis which creates the shift in the oxyhemoglobin dissociation curve. Although we report a significant correlation ($r = 0.40$) between end exercise [La] and the SC, the correlation is lower than previous studies have noted (6, 24). There have been reports where [La] is progressively increasing while a steady state for VO_2 is noted, and just the opposite where a significant slow component is noted despite

unchanged levels of [La] (14). Increased [La] and the SC during high constant-load exercise may be due to the progressive recruitment of lower efficient fast-twitch motor units, which would result in a progressive elevation in the VO₂. It is possible that the activation of fewer fast-twitch motor units could account for the reduction in the SC following training. Both Coyle *et al.* (8) and Gaesser *et al.* (13) have reported evidence giving credence to this hypothesis.

In conclusion, the results of the present study demonstrate that following an endurance exercise training program, subjects training at moderate (<LT) and heavy exercise (>LT) exercise intensities will exhibit similar improvement in VO₂ and power output at peak exercise and the lactate threshold when total work is controlled. To our knowledge this is the first, and only study that has examined the temporal and magnitude of adaptation in the SC as a function of training intensity. The present study concludes that training at high exercise intensities will result in larger and faster adaptations of the SC when compared to training at moderate exercise intensities, even when controlling for total work between training groups. In addition, in comparison to previous reports on temporal adaptations in the SC following HIT, the present study reported a more rapid and larger reduction in the SC. In addition, we observed continued adaptations in the SC until 4 wk of HIT and 6 wk for LIT, which is contrary to other reported (32).

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Table 1. Physical characteristics of the subjects.

Training Group	N	Age (yr)	Height (kg)	Pre-weight (kg)	Post-Weight	Pre-training VO ₂ @ LT	Pre-training VO ₂ @ Peak
						(L · min ⁻¹)	(L · min ⁻¹)
HIT	6	23.5 ± 2.4	182.8 ± 3.4	87.2 ± 3.1	87.2 ± 3.3	1.7 ± 0.1	3.4 ± 0.1
LIT	6	22.8 ± 3.9	180.3 ± 4.3	91.3 ± 3.5	91.0 ± 3.5	1.4 ± 0.1	3.1 ± 0.1
NT	6	23.0 ± 2.3	177.8 ± 4.0	80.6 ± 7.6	80.6 ± 7.9	1.6 ± 0.1	3.0 ± 0.3

N = number of subjects in each group; each value is ± SEM

HIT: High-intensity training; LIT: Low-intensity training; NT: No training

* no differences among the three groups with respect to age, weight, height, pre-training VO₂ at LT or peak exertion (p>0.05)

Table 2. Effects of 6 wk training on VO_2 and power output at LT and peak exercise.

Group	N	$\Delta\text{VO}_2 (\text{L} \cdot \text{min}^{-1})$				$\Delta\text{Power Output (Watts)}$			
		@LT	%	@Peak	%	@LT	%	@Peak	%
HIT	6	$0.58 \pm 0.06^*$	33	$0.64 \pm 0.11^*$	18	$53.6 \pm 13.8^*$	42	$53.2 \pm 6.0^*$	17
LIT	6	$0.47 \pm 0.05^*$	35	$0.45 \pm 0.07^*$	15	$49.6 \pm 5.6^*$	50	$41.0 \pm 4.9^*$	16
NT	6	0.07 ± 0.04	4	0.05 ± 0.05	2	10.7 ± 7.0	6	10.0 ± 7.1	4

N = number of subjects in each group; each value is \pm SEM

HIT: High-intensity training; LIT: Low-intensity training; NT: No training

 ΔVO_2 = [post-training VO_2 - pre-training VO_2]; $\Delta\text{Power Output}$ = [post-training Power Output - pre-training Power Output]

% = % increase from pre-training

* significantly different from NT group; $p < 0.05$

Table 3. Characteristics of training sessions and pre-training responses to the training power output.

Training Group	N	Training Power Output (Watts)	Session Duration (min)	HR (b · min ⁻¹)	% HR _{max}	VO ₂ (L · min ⁻¹)	% peak VO ₂
HIT (LT+75%)	6	235 ± 10	25.3 ± 1.7	163 ± 2.6	88	3.0 ± 0.1	81
LIT (90% LT)	6	96 ± 7	60	126 ± 6.4	70	1.5 ± 0.1	48

N = number of subjects in each group; each value is \pm SEM

HIT: High-intensity training; LIT: Low-intensity training

% peak VO₂ = 3 minute VO₂

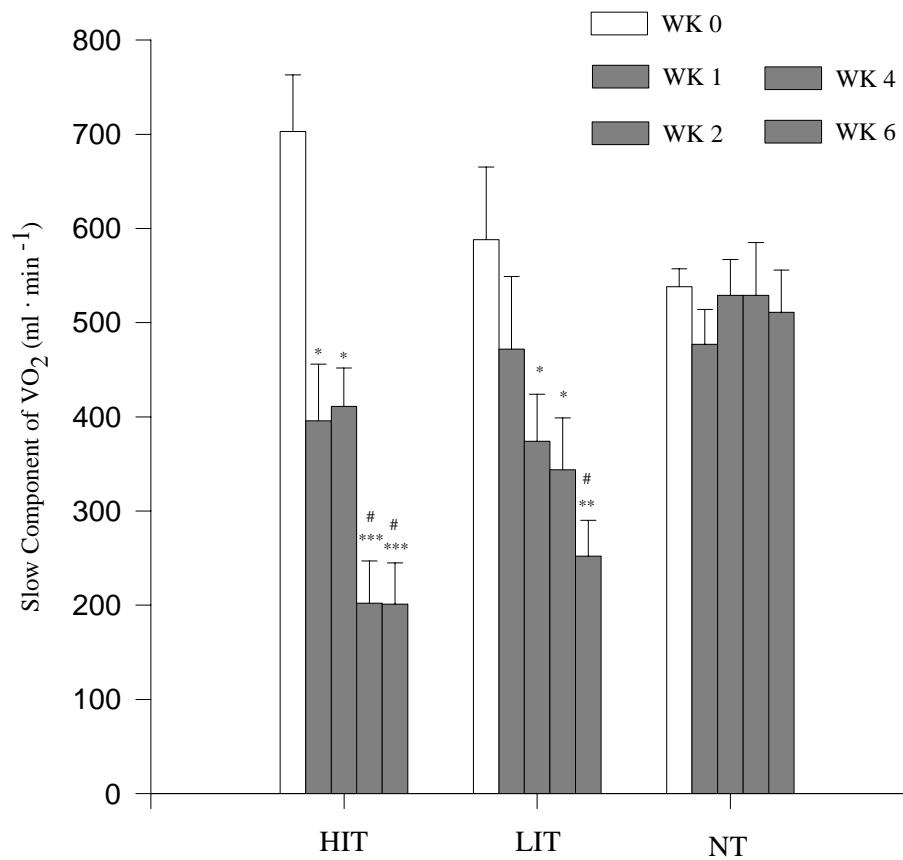
Table 4. Effects of training on end-exercise responses to HCL testing

Training Group	VO_2 ($\text{L} \cdot \text{min}^{-1}$)	% peak	V_E ($\text{L} \cdot \text{min}^{-1}$)	HR (bt $\cdot \text{min}^{-1}$)	% peak	Lactate (mmol $\cdot \text{L}^{-1}$)
HIT						
Pre-training	3.69 ± 0.13	106	142 ± 7.1	186 ± 3.0	101	12.1 ± 0.9
Post-training	$3.26 \pm 0.10^*$	79	$94 \pm 3.9^*$	$159 \pm 3.7^*$	87	$6.2 \pm 0.9^*$
LIT						
Pre-training	3.06 ± 0.10	100	119 ± 6.2	184 ± 5.8	101	10.8 ± 0.7
Post-training	$2.84 \pm 0.03^*$	81	91 ± 3.3	$168 \pm 5.7^*$	91	$6.9 \pm 0.8^*$
NT						
Pre-training	3.04 ± 0.32	101	116 ± 15.0	175 ± 2.1	97	8.2 ± 0.8
Post-training	3.02 ± 0.31	98	109 ± 13.6	174 ± 4.0	96	7.0 ± 0.9

* post-training significantly different from pre-training ($P<0.05$); each value is mean \pm SEM

HIT: High-intensity training; LIT: Low-intensity training; NT: No training

Figure 1. Time Course and Magnitude of Adaptation for the Slow Component of VO_2



Data are Means \pm SEM; n = 6 subject per group

* significant reduction from pre-training ($p < 0.05$)

** significantly reduction from wk 1 ($p < 0.05$)

*** significantly reduction from wk 2 ($p < 0.05$)

significantly lower than NT group ($p < 0.05$)

Figure 2. Relationship between the Slow Component of VO_2 and delta VE

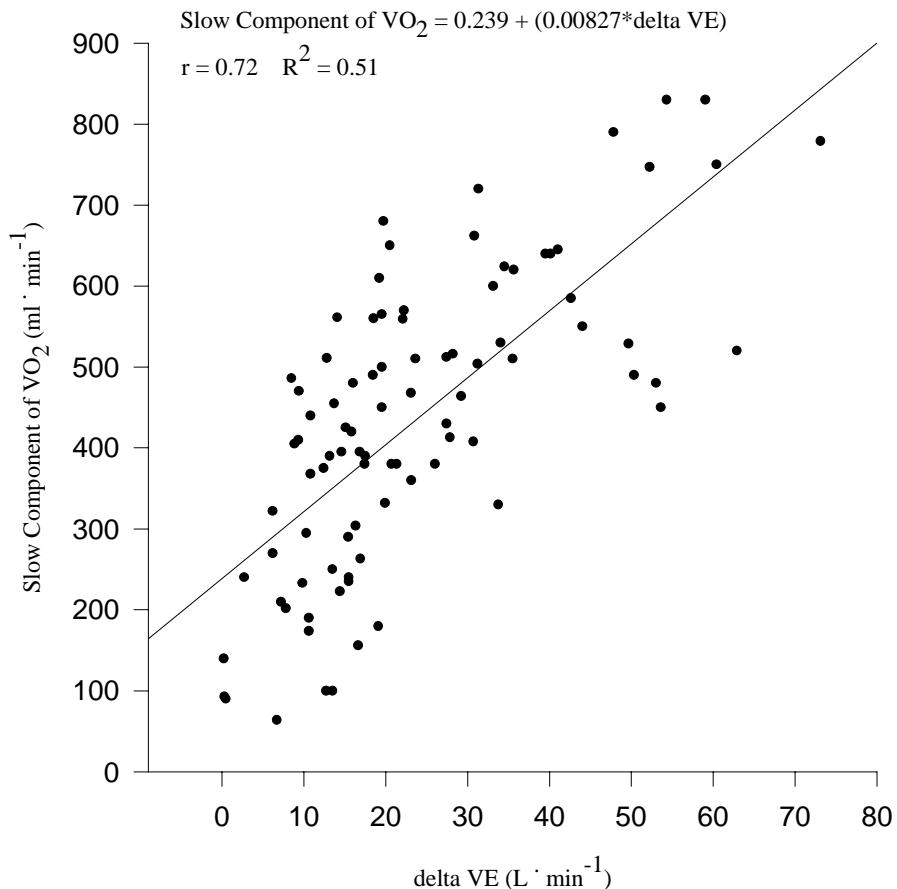
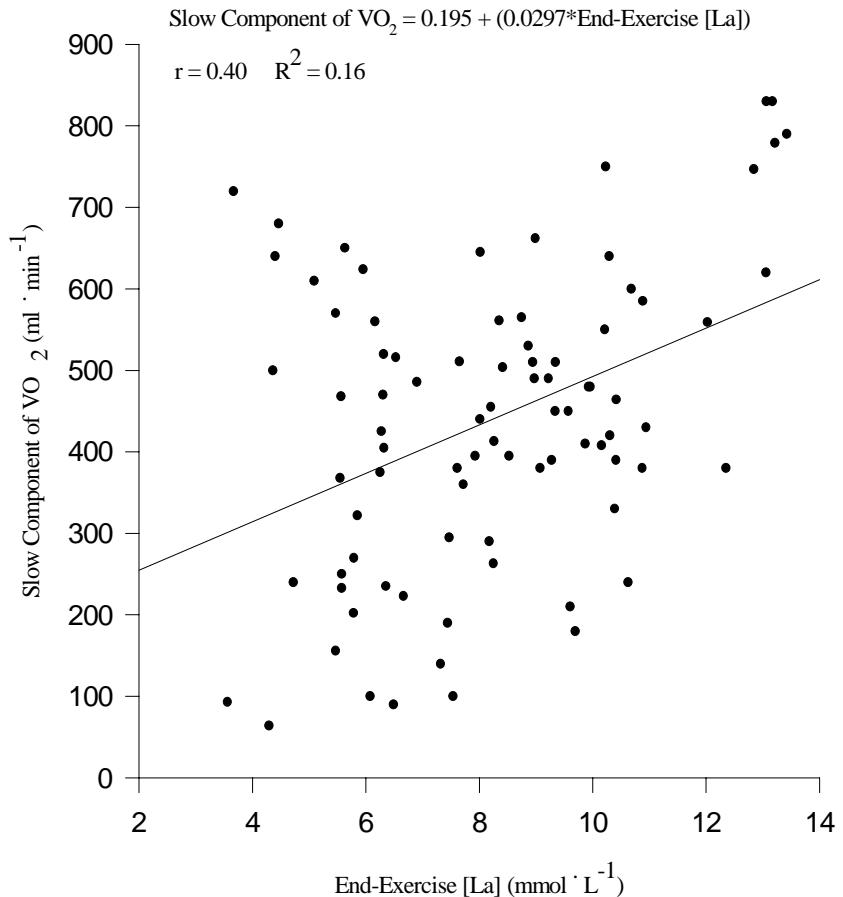


Figure 3. Relationship between the Slow Component of VO_2 and End-exercise [La]



Chapter IV

SUMMARY, IMPLICATIONS FOR CLINICAL PRACTICE AND RESEARCH

The SC was reduced following both high ($>$ LT) and moderate intensity training ($<$ LT). The HIT group demonstrated a more rapid and pronounced adaptation in response to training when compared to subject training at the lower training intensity although total work was controlled for training groups. The speed at which an adaptation in the slow component was noted occurred earlier than previous reports (Womack *et al.*, 1995). However, the previously reported pre-training magnitude of the slow component was much larger in the present study, which may be explained by the a higher work intensity utilized for HCL testing. In addition, a previous report that adaptation of the slow component of VO_2 is completed early in training (by wk 2) was not apparent in the present study with additional adaptation measured up to 4 wk of training in the HIT group. As Gaesser reported (1994) the pre-training slow components in previous studies were not trivial (mean values: ≈ 200 to $400 \text{ ml} \cdot \text{min}^{-1}$), he had noted that much larger slow components ($1.0 - 1.5 \text{ L} \cdot \text{min}^{-1}$) had been reported in the literature. Perhaps our study, because of the large pre-training slow component, gives a more clear understanding of the effects of endurance exercise training intensity and the magnitude and temporal effects associated with the SC.

Adaptation in the slow component of VO_2 was demonstrated in the LIT group. However, significance was not noted until after 2 wk of endurance training. The mean value of the SC did continued to decline with training with differences noted between wk 1 and 6 of training. The magnitude of reduction in the LIT group is comparable to that demonstrated after 5 wk of

training at a similar intensity by Casaburi *et al.* (1995), but no attempt was made to investigate the temporal changes that occurred across the 6 wk training period.

Improvement in both VO_2 and power output at the LT and peak exercise were noted for both HIT and LIT groups. No statistical difference was noted between the two training groups. It would be interesting to perform a similar training study on patients with cardiac and/or ventilatory limitations. In these clinical populations, the disease process may limit their ability to increase peak VO_2 or maximal voluntary ventilation (MVV). Hence, one would discern that any adaptation in the slow component of VO_2 would result from increasing the LT to a higher percentage of peak VO_2 .

Both the end-exercise [La] ($r = 0.40$) and ΔVE ($r = 0.72$) were significantly correlated with changes in the slow component over the course of training. Although Poole *et al.* (1991) have suggested that the primary mediator(s) for the slow component of VO_2 are found within the exercising limb, others have suggested that additional contributions could be attributed to central variables such as ventilation (Hagberg *et al.*, 1978, Casaburi *et al.*, 1987). In the current study, V_E was significantly correlated to the slow component of VO_2 over the course of the 6 wk study ($r = 0.72$). Despite the correlation, it is unlikely that V_E represents a primary mechanism involved in the slow component of VO_2 . We estimated, from data by Aaron *et al.* (1992), that the delta V_E would account for approximately 21% of the adaptation of the slow component of VO_2 for both HIT and LIT groups. It is unclear, and has not been reported, whether the same percentage would be noted in a clinical population limited primarily by V_E . In addition, we report a significant correlation ($r = 0.40$) between end exercise [La] and the slow component of

VO₂, the correlation we report is lower than previous studies have noted (6, 22). This discrepancy may be explained by the utilization of various work intensities compared to the one high-intensity constant-load bout in the present study.

The results of this study demonstrate that following endurance training, subjects training at a moderate and heavy exercise intensity will exhibit similar improvement in VO₂ and power output at peak exercise and the LT. However, subjects training at the high exercise intensity demonstrated larger and faster adaptations in VO₂ response “slow component of VO₂” to a high-intensity (<LT) constant-load exercise test when compared to the moderate exercise intensity training group. Despite training at moderate intensities (<LT), these individuals demonstrated improvements (i.e. an adaptation of the slow component of VO₂) with training. In comparison to previous reports, changes in the slow component occurred more rapidly after training, and were not complete until after wk 4 of training. In addition, it was demonstrated that V_E may be a small (20%) contributor to the slow component of VO₂.

Recommendations for Further Research

Based on the findings of the present study and relevant literature, the following recommendations seem warranted:

1. The present investigation and the majority of exercise training reports, are performed with young, college-aged adults or in healthy populations. Studies on clinical populations need to be performed to determine if: 1) this variable (e.g. slow component of VO₂, and the study of VO₂ kinetics) is useful in the clinical setting and, 2) if training adaptations to maximal and

- submaximal work are equivalent to what was found in this study, and 3) the influence of not only moderate and high-intensity exercise could be described as very low-intensity exercise.
2. Greater safety of constant-load test versus maximal testing seems intuitive. I believe the use of oxygen kinetic parameters (e.g. time constant of response, $t^{1/2}$, and the slow component of VO_2) is underutilized and could be of significant value to optimize care and therapy for clinical population, as well as providing a means of quantifying adaptations to exercise training. Methodology and easier means of obtaining this information need to be developed and studies need to be performed with this equipment and software.
 3. Due to unavailability of certain resources, we were unable to study all of the mediator of the SC. Studies often use temporal changes in the relationship between a physiological variable and various potential mediators as a study design. Clearly, the protocol, i.e. intensity for the high-constant-load bout, we chose demonstrated a large pre-training slow component, and would be more ideally suited for this type of study.

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APPENDIX A

Informed Consent and Medical History

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY**Informed Consent for Participants****of Investigational Projects****Subject #:** _____

Title of Project: Time course and magnitude of adaptation in the slow component of VO₂ following 6 wk of high or low intensity endurance exercise training in sedentary males

Investigator: Jeffrey V. Ocel, MS

I. The Purpose of the Research/Project

I am being asked to participate in a research study about the type of exercise training and amount of time required to change my pattern of oxygen consumption to high-intensity and low-intensity exercise. This research study will involve periodic exercise testing for the purpose of assessing the changes in oxygen consumption to exercise over 6 wk of endurance training on a bicycle ergometer. Although most experts agree that, following an endurance training program, I will be able to exercise at a higher maximal workload and my maximal oxygen consumption will increase, it remains unclear whether changes in exercise performance and oxygen consumption occur at submaximal exercise work levels. Forty-five healthy, physically inactive, males are being asked to participate in this research study.

II. Procedures

Prior to being included in the research study, I will complete a health history that will help to determine if there may be reasons why I should not participate in this study. If the results of the health history indicate that I am an appropriate subject for this study, than I will be

informed of when I should report to the Laboratory for Health and Exercise Sciences at 230 War Memorial Hall for the initial exercise testing.

Maximal exercise testing. As a subject, I understand that I will perform 2 maximal exercise tests on a cycle ergometer to determine the maximal amount of exercise that I can perform. The two tests will be performed at the start of the research study and immediately following the 6 wk training period. I will need to report to the laboratory 30 min before each test is to begin, and at least 4 hours after consuming any food or caffeinated beverage.

Prior to each test, I will be connected by electrodes and cables to an electrocardiograph recorder which will enable the technician to monitor my heart rate. I will also be fitted with a breathing apparatus (mouth piece and nose clip) which will measure my exhaled gases to properly measure oxygen consumption. A registered nurse/licensed medical technician will insert a special tube called an intravenous catheter into a vein in my arm to collect blood samples for blood lactate analysis. It will remain in place for the entire testing period: approximately 45 to 60 min. Through the course of the each maximal exercise test, a total of approximately 12.5 mls (amount is approximately equal to 2.5-3.0 teaspoons) of blood will be taken for laboratory analysis. These samples will be taken at rest and every minute during exercise and recovery.

The maximal exercise test which I will undergo will be performed on a stationary cycle ergometer with the amount of effort gradually increased. As I understand it, the increase in effort will continue until I report to the technician that I am unable to continue or the pedal rate falls below 40 revolutions per minute. I will decide when I am unable to continue and the technician will stop the test when I so request.

Submaximal testing. Approximately two days after completing the first maximal test, and periodically during the 6 wk of exercise training, I will be asked to perform a high-intensity and a low-intensity submaximal bicycle ergometer test. Unlike during the maximal exercise test where the amount of effort is gradually increasing, I will pedal at the same work rate for the entire duration of the test. The period during the study where I will perform the high-intensity test will include: the beginning of the research study, and after weeks 1, 2, 4, and 6. The low-intensity test will be performed only at the beginning of the research study and at the end of the 6 wk period. Therefore, upon completion of the study I will have completed 5 high-intensity exercise tests and 2 low-intensity submaximal bicycle tests over the 6 wk period.

I understand that I will need to report to the laboratory 30 min before these tests are to begin, and at least 4 hours after consuming any food or caffeinated beverage. I will again be connected with electrodes and cables to an electrocardiograph recorder which will enable the technician to monitor my heart rate. I will also be fitted with the breathing apparatus (mouth piece and nose clip) to measure my exhaled gases. Prior to each test, at 3 min of exercise, and at the end of the test, a registered nurse/licensed medical technician will insert a butterfly catheter into a vein in my arm to collect blood samples for blood lactate analysis. A total of approximately 3 mls (a little less than 0.5 teaspoons) of blood will be taken for laboratory analysis.

I understand that the workload used for the low and high-intensity exercise tests will be determined from the first maximal exercise test. This workload will not be changed throughout the duration of the study. For each constant-load test I will be asked to exercise for a period not greater than 15 min.

Exercise Training. Following the first maximal and submaximal tests, it is my understanding that I will be assigned to one of three groups: 1) high-intensity training group, 2) low-intensity training group, or 3) no training (control group). If I am assigned to one of the training groups, I will report to the Laboratory for Health and Exercise Science for 4 additional exercise sessions per wk, with each session lasting approximately 20 to 60 min. I understand that this training period will last 6 wk. If I am assigned to the no training (control group), I understand that I will still need to report at the times specified above for testing, however, I will not report for exercise training. I also understand that, regardless of what group I am assigned to, it is important that I do not perform additional aerobic training outside of the study (running, biking, swimming, stair stepping, etc.).

If I am assigned to the training group, it is my understanding that each training will include a brief warm-up period, followed by the bicycle training session, and a brief cool-down period. My training workload will be applied for a time that will depend on what training group I was assigned. For example, if I am in the low-intensity training group, I will be cycling for 45 to 60 min at a lower workload. However, if I am in the high-intensity training group, I will be training for approximately 30 min, but at a higher workload.

II. Risks

It is my understanding and I have been informed that there exists the possibility during exercise of adverse physiologic responses during the tests. I have been informed that these changes could include abnormal blood pressure, fainting, disorders of the heart beat, and in rare instances, heart attack, stroke, or death. Every effort will be made to minimize these risks by evaluation of the preliminary information relating to your health and by observations during

testing. Other possible discomforts I may experience in this study include leg fatigue, muscle soreness, a dry mouth (from the mouthpiece), pain, bleeding and local bruising at the site the blood was taken. I understand that the registered nurse or licensed medical technician collecting the blood samples, and that the technicians who may be handling the samples, will be wearing gloves at all times. I also understand that a registered nurse or licensed medical technician, certified exercise specialist and other support personnel will be present during all exercise testing to minimize the risks during exercise. I also understand that there is also a working telephone in the exercise testing area that can be used to alert the emergency rescue squad on the campus of Virginia Tech. Their average response time in getting to the Laboratory for Health and Exercise Science is approximately 4 to 5 min.

III. Benefits of the Project

My participation in this project will provide valuable information that will help clarify the type of training and time needed to change my ability to exercise at low and high intensity levels. In addition, I will receive information regarding my physical conditioning, exercise tolerance, and body composition. I will also be provided, at no cost, the use of exercise training equipment and facilities.

IV. Extent of Anonymity and Confidentiality

The results of this study will be kept strictly confidential. At no time will the researchers release my results of this study to anyone other than the individuals working on the project without your written consent. The information I provide will have my name removed and only a subject number (excluding social security numbers) will identify me during analyses and written reports of this research.

V. Compensation

I understand that there is no monetary or course credit compensation available for participants in this project.

VI. Freedom to Withdraw

I understand that if I refuse to participate in this research study or choose to discontinue my participation at anytime, no penalties or loss of benefits to which I am otherwise entitled to will occur.

VII. Approval of Research

This research project has been approved, as required, by the Institutional Review Board for projects involving human subjects at Virginia Polytechnic and State University and the Department of Human Nutrition and Foods.

VIII. Subject's Responsibilities

I know of no reason I cannot participate in this study. I have the following responsibilities:

1. Accurately report my medical history.
2. Arrive at the laboratory 30 min prior to all maximal and submaximal exercise testing.
3. Arrive at the testing laboratory 4 hours post-absorptive for all maximal and submaximal exercise testing.
4. Report and complete all training sessions (4 sessions/wk; 6 wk)
5. Refrain from other forms of physical training (i.e. jogging, biking, hiking, weight training, etc.) for the duration of the study
6. Report any unusual signs/symptoms during the study

IX. Subject's Permission

I have read and understand the informed consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project.

If I participate, I may withdraw at any time without penalty. I agree to abide by all the rules of this project.

Signature

Date

Should I have any questions about this research or its conduct, I will contact:

Jeffrey V. Ocel
Investigator

951-7586
Phone (Home)

231-5006
Phone (Laboratory)

231-8209
Phone (Office)

Marilyn Prehm
Nutrition, Foods, Exercise Science

231-5549
Phone (Office)

Ernest Stout
Chair, IRB
Research Division

231-5549
Phone (Office)

Appendix B
Medical Health History Questionnaire

VIRGINIA POLYTECHNIC AND STATE UNIVERSITY**Health History Questionnaire**

Subject #: _____

Title of Project: Time course of adaptation in the slow component of VO₂ after high and low intensity endurance exercise training in sedentary males

Investigator: Jeffrey V. Ocel, MS; William G. Herbert, Ph.D.

Please complete the following questionnaire as accurately as possible.

Name: _____ Age: _____

Date of Birth: _____ 19 _____

Address:

Phone: (540) _____ (work)

Phone: (540) _____ (home)

1. Do you have any known allergies? Y N

If yes, please explain:

2. Do you currently take any medications? Y N

If yes, please explain:

3. Do you currently exercise regularly? Y N

If yes, please explain:

4. Please circle all that apply to you?

High Blood Pressure	Asthma	Smoking/Tobacco		
Skipped Heart Beats	Chest Discomfort	Fast Heart Rate	Diabetes	Heart
Murmur	Short of Breath	Dizziness/Fainting	Musc/skeletal	
Joint Soreness				

If circled, please explain:

5. Is there any reason not mentioned above that would limit your ability to perform high-intensity exercise?

Y N

If yes, please explain:

6. Has a physician ever told you to refrain from activity or exercise for an extended period of time?

Y

N

If yes, please explain:

7. Do you have any difficulty with getting your blood taken?

Y

N

If yes, please explain:

Signature _____

Date _____

APPENDIX C

Detailed Methodology

METHODOLOGY

Subject Selection

Eighteen sedentary males were recruited from the Virginia Tech campus and surrounding community to participate in this study. The study coordinator met with each subject to discuss participation in the study. If the potential subject expressed interest in participating, the study was explained in detail, a copy of the informed consent document was provided, and any questions the subject had was answered at that time, or after the subject had time to review the entire protocol. In addition, because the attempt was to enroll subjects who were currently untrained, only subjects who had not participated in endurance exercise activities (<2 day/wk) for at least 3 months prior to study were eligible to participate in this study. The definition used defining subjects' as physically inactive was also used in a comparative study performed by Casaburi et al. (1994).

Protection of Human Subjects

The research protocol and informed consent document (Appendix A) were obtained from each subject and the research protocol was approved by the Institutional Review Board (IRB) for research involving human subjects at Virginia Polytechnic and State University. The IRB was informed in a timely manner of any adverse events which occur during the course of exercise testing or training. Prior to inclusion, subjects completed a medical history (Appendix B) to assure patient safety and identify any contraindication to vigorous exercise as described by the American College of Sports Medicine (1995). All exercise testing was conducted in the Laboratory for Health and Exercise Science with a staff of specialists with extensive experience in clinical exercise testing.

Orientation Procedures

An orientation session was completed by all subjects prior to the initial maximal exercise testing bout. The session consisted of familiarization with the Human Performance Laboratory, and an explanation of all procedures which included an introduction to both the testing and training cycle ergometers, and an explanation of how the cardiopulmonary and lactate data would be acquired. In addition, an explanation of how the testing and training power outputs would be set was also explained.

During the orientation, each subject sat on both the Monarch E 818 cycle ergometer and the MedGraphics CardiO₂ cycle, and the appropriate seat height was determined at that point. The subjects were allowed to free pedal briefly to become familiar with each cycle. As the subjects were cycling on the MedGraphics CardiO₂ cycle, each were instructed to place a mouthpiece and nose clip on to simulate collection of gas exchange data. At that time, subjects were instructed on how to communicate with the investigators and had ample time to become adjusted to the mouthpiece and testing apparatus.

Study Design

Upon admission to the study, and following completion of a pre-training maximal exercise test and two constant-load exercise testing bouts, each subject was randomly assigned to one of the following groups: 1) high-intensity endurance training [HIT]; 2) low-intensity endurance training [LIT]; or 3) control [NT]). The duration of the training period was 6 wk. Each subject was asked to refrain from performing additional exercise training outside of the study, and was asked to report additional activities to the principal investigator or one of the exercise leaders throughout the duration of the study period.

Protocol for Determination of Peak VO₂ and Lactate Threshold (LT). Prior to and after the 6 wk endurance training program subjects performed a maximal cycle ergometry test to exhaustion. Both tests were performed on a calibrated MedGraphics CardiO₂ stationary cycle ergometer (Medical Graphics Corporation, St. Paul, MN). All subjects were tested at the same time of day before and after training testing, and performed on subjects at least 4-hours post-absorptive. The pre-training and post-training tests were conducted 2 days prior to the initial constant-load exercise bouts and 2 days following the final CL exercise bouts, respectively. Testing consisted of a 5 min rest period followed by a continuous rate of increase in power output starting at 0 and set to increase at 20 W · min⁻¹. The protocol was chosen to elicit fatigue in 8-15 min in order to optimize attainment of peak VO₂ (3). The protocol was continued until exhaustion supervened, which was noted by the subjects inability to continues the pedaling rate above 40 rpm. Peak VO₂ (L · min⁻¹) was determined as the highest VO₂ achieved during the final minute of exercise. Two blood samples (0.5-1.0 cc) were drawn at rest, and at the end of each minute through an indwelling venous catheter located in the forearm for determination of [La]. One 1.0 cc waste sample was drawn prior to each these samples to eliminate contamination from the prior sample. All blood samples were collected into vacutainers that contained potassium oxalate to prevent further glycolysis and sodium flouride to prevent coagulation. Following collection of the sample it was placed in an ice slurry and analyzed immediately.

The LT was determined by three independent investigators who examined the relationship between [La] and power output. The LT was defined as the breakpoint in the relationship between blood [La] plotted as a function of the VO₂ during the continuous ramp

protocol (references). An increase in [La] of at least 0.2 mmol/L was required for the determination of the LT. In the instances where the 3 investigators did not agree, the average value was utilized.

Protocol for Constant Load Cycle Ergometry Exercise Tests. Prior to the randomization to training group, subjects performed a high-intensity constant-load (HCL) and low-intensity constant-load (LCL) cycle ergometry exercise test. Subjects completed the HCL exercise bout initially, and then, following a 30 minute recovery interval, completed the LCL bout. During the 6 wk training period, only the HCL bout was repeated by the subjects at wk 1, 2, 4, and wk 6. The power output was kept constant for each HCL test throughout the duration of the experimental period (i.e. power output was not readjusted based on any improvement in peak VO_2). Subjects were not allowed to train the day prior to a HCL test, thus all testing was performed approximately 48 hours following the previous exercise training session. All HCL exercise bouts were conducted at approximately the same time of day throughout the experimental period and all subjects performed the testing at least 4-hours post-absorptive. During the 4-hour period subjects were told to refrain from food, caffeinated beverages, and tobacco products.

The power outputs utilized for the LCL and HCL exercise bouts were derived from the baseline maximal test data. Subjects cycled at a power output equal to [power output at LT * .90] (**90% LT**) and [power output at LT + .75 (power output at Peak - power output at LT)] (**LT + 75%**) for the LCL and HCL exercise bouts, respectively. All constant-load bouts included collection of 3 min of seated baseline data, up to 15 min of exercise data, and 6 min of seated recovery data. If the subject was not able to complete the entire 15 minute period, the time was

noted, and all subsequent HCL testing were discontinued at that time. The termination endpoint was used to represent the end-exercise VO_2 .

Two blood samples (0.5-1.0 cc) for [La] determination were drawn at 3 min of exercise, and at the end of each CL exercise bout. Samples were obtained with a 23 gauge butterfly needle and collected into vacutainers that contained potassium oxalate to prevent further glycolysis and sodium fluoride to prevent coagulation. Following collection of the sample it was placed in an ice slurry, and analyzed immediately. Ratings of perceived exertion (overall and peripheral) was determined using the Borg category scale (Borg, 1970) at 5, 10, and 15 min of exercise.

Determination of the Slow Component of VO_2 . The slow component of VO_2 was calculated by subtracting the end-exercise VO_2 from the VO_2 at 3 min of exercise. The reliability of defining the slow component in this manner has been reported to be high ($r = 0.91$) (Davis, Ocel, and Craft, in press).

Determination of magnitude of increase in [La] ($\Delta[\text{La}]$). The $\Delta[\text{La}]$ was determined by subtracting the end-exercise [La] from the [La] at 3 min for the HCL exercise test.

Determination of the Pulmonary Ventilation drift (ΔVE). ΔVE will be calculated by subtracting the end-exercise VE from the VE at 3 min for the HCL exercise test.

Data Collection

Pulmonary gas exchange. During all maximal and CL exercise testing, ventilation and pulmonary gas exchange variables were measured continuously using the Medical Graphics CPX/D system (Medical Graphics Corporation, St. Paul, MN) for determination of VE , VO_2 , and VCO_2 . Prior to testing, gas analyzers will be calibrated with commercial gases certified to be within $\pm 0.01\%$ (room air reference: O_2 , 21%; CO_2 , 0% and calibration: O_2 , 12%; CO_2 , 5%). The

calibration routine includes a routine to determine the phase lag to synchronize the flow wave form with CO₂ and O₂ wave forms for each breath. Pulmonary ventilation will be determined by measuring expiratory gases through a disposable pitot pneumotach (dead space 20 ml). This device has been reported to be reliable (within 1% for piston pump experiments; 2% during human exercise studies) in the measurement of pulmonary ventilation as reported by Porszasz, Barstow and Wasserman (1994).

Heart rate. Heart rate was continuously monitored utilizing a Physio-Control Lifepack 9.

Blood [La]. Blood [La] was measured utilizing the YSI Model 1500 Sport Lactate Analyzer (Yellow Springs, OH). This system is a versatile instrument that allows the investigator to manipulate the way on collects the sample, injects the sample into the instrument, and analysis of the sample (i.e. serum, plasma, whole blood). The detectable range of the analyzer is 0 to 30 mmol/L (0 to 270 mg/dL) and offers a resolution to the nearest 0.01 mmol/L (0.2mg/dL). Calibration is performed at 5 mmol/L (45 mg/dL) and system linearity or upper limit can be checked at 15 mmol/L (135 mg/dL) and/or 30 mmol/L (270 mg/dL).

The sampling chamber contains a sensor which is comprised of a 3-layer membrane which contains immobilized lactase oxidase in the middle membrane layer. As the sample is injected, the substrate (lactate) diffuses through the membrane, comes into contact with the immobilized enzyme (lactate oxidase), and is rapidly oxidized, producing hydrogen peroxide (H₂O₂). The H₂O₂ is, in turn, oxidized at a platinum anode, producing electrons. The electron flow is proportionally linear to the steady state H₂O₂ concentration and, therefore, to the concentration of lactate in the sample.

Rating of Perceived Exertion (RPE). RPE will be obtained during the incremental and CL bouts using the Borg category scale (Borg, G., Hassmen, P., & Lagerstrom, M. (1987).

Training Program. Following the initial constant-load testing, subjects were randomly assigned to either: 1) high-intensity exercise training (HIT), 2) moderate intensity training (LIT), or no training (NT). The endurance training program consisted of supervised exercise on a stationary cycle ergometer (Monarch E 818 cycle ergometer). Ergometers were calibrated wkly. In addition to each subject completing the HCL test on the first day of each wk, subject trained an additional 4 day · wk⁻¹ on the Monarch ergometer. Based on the pre-training exercise studies, subjects in the LIT group were assigned to train at 90% LT which was designed to elicit a moderate exercise training intensity. Subjects in the HIT group were assigned to train at LT + 75% which was designed to elicit a high exercise training intensity. The training power output was kept constant throughout the 6 wk study. The duration of the exercise sessions for the two groups were designed so that the total work performed per session did not depend on group assignment. Specifically, all subjects in the LIT group trained for 60 minute · session⁻¹. For subjects in the HIT group, total work was calculated as if the subject had been assigned to the LIT group. The total work was then divided by the actual power output used during HIT to determine the time needed to train each session. All subjects were discouraged from engaging in physical activity outside of the program. Attendance for the training sessions averaged 90% for the HIT group and 93% for subjects in the LIT group.

Statistical analysis

A 2-way analysis of variance with repeated measure was used to determine the overall effect of training on the slow component of VO₂. The slow component of VO₂ during the HCL

exercise bout was defined and calculated by subtracting the end-exercise VO_2 from the VO_2 at the third minute of exercise (Davis et al, in press). When significant group, trial or group x trial differences were detected, simple effects testing was performed. The Tukeys HSD test was used post-hoc to identify specific group differences.

Linear regression was used to assess the relationship between changes in the slow component of VO_2 and training induced changes in VE and [La].

Improvements in peak VO_2 and LT were assessed by using change scores. A One-way analysis of variance was then used to determine the effects of training. When significant group differences were detected, simple effects testing was performed with Tukeys HSD used to test mean differences .

APPENDIX D

Raw Data

Raw Data: Demographics

Group	Age (yrs)	Height (cm)	Pre-weight (kg)	Post-weight (kg)	# Training sessions	% Training sessions
HIT	23	177	92	90.9	28	0.93
HIT	27	170.5	99.1	101	27	0.9
HIT	22	183	82.9	81.5	26	0.97
HIT	26	183	77.5	78.2	29	0.97
HIT	21	192.5	87.6	87.3	27	0.9
HIT	21	192	92.1	94.2	29	0.97
HIT	22	190.5	84.1	84.1	28	0.93
LIT	25	193.5	97	93	28	0.93
LIT	29	177	90.5	90.25	27	0.9
LIT	24	166	91	91.6	28	0.93
LIT	19	177	101.4	101.6	25	0.83
LIT	19	176	76	75.4	28	0.93
NT	23	181	83	80	0	0
NT	26	187	113.4	115	0	0
NT	19	166	58.2	57.5	0	0
NT	23	164.5	72.1	73.2	0	0
NT	24	184	72.5	73	0	0
NT	23	184	84.2	86.4	0	0

Maximal Cycle ergometry Testing: Raw Data

Group	Time	HR (bpm)	[La] (mM/L)	RPE (legs)	RPE (overall)	VO ₂ (L·min ⁻¹)	VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (L·min ⁻¹)	VE (L·min ⁻¹)	R	W
1	Pre	178	6.6	19	17	3.383	36.75	4.152	122.9	1.23	294
1	Post	170	4.62	19	16	3.93	43.25	4.496	136.8	1.15	327
1	Pre	182	6.805	15	16	3.303	33.35	3.74	105.9	1.14	254
1	Post	182	9.7	17	17	3.97	39.3	4.5	129	1.16	326
1	Pre	181	12.54	17	16	3.643	46.15	4.849	151	1.27	326
1	Post	178	12.32	17	17	4.706	57.75	5.589	189.1	1.19	390
1	Pre	188	9.765	17	16	2.991	38.6	3.546	103	1.19	268
1	Post	186	10.88	18	17	3.776	48.3	4.398	126.1	1.17	314
1	Pre	190	8.93	19	17	3.54	40.5	4.26	124.2	1.21	300
1	Post	195	9.97	19	18	3.94	45.1	4.56	149.6	1.16	343
1	Pre	184	7.425	18	18	4.01	48.5	4.661	151.4	1.16	344
1	Post	184	13.51	19	19	4.388	51.1	5.303	168.9	1.21	405
2	Pre	171	7.36	19	18	3.228	33.25	3.68	97.1	1.14	265
2	Post	164	7.4	19	17	3.701	39.8	3.701	113.9	1.13	318
2	Pre	174	7.74	18	17	2.854	31.55	3.495	96	1.23	239
2	Post	184	8.75	19	12	3.194	35.3	3.84	99.9	1.21	269
2	Pre	170	9.455	17	19	2.84	31.2	3.439	100.6	1.21	239
2	Post	175	10.27	18	17	3.38	36.8	3.911	135.8	1.16	270
2	Pre	192	8.55	17	16	2.95	28.6	3.346	98.8	1.16	247
2	Post	190	11.245	16	16	3.71	36.45	4.34	138.5	1.18	304
2	Pre	194	8.72	17	19	3.344	44	3.757	119.8	1.12	284
2	Post	201	9.315	20	20	3.679	48.8	4.478	160.9	1.22	328
2	Pre	194	9.55	17	15	3.162	34.3	3.866	121.1	1.22	269
2	Post	192	6.085	19	15	3.392	35.6	3.999	130.3	1.18	300
3	Pre	187	9.605	18	17	3.744	45.1	4.669	137	1.25	297

3	Post	183	9.6	17	16	3.94	49.5	4.73	151.7	1.19	330
3	Pre	188	10.62	19	19	3.87	34.13	4.635	161.4	1.2	333
3	Post	185	9.78	19	18	4.05	35.22	4.829	171.6	1.19	329
3	Pre	179	12.21	15	16	2.164	37.2	2.843	94.3	1.32	182
3	Post	182	10.58	15	13	2.215	38.85	2.712	93.7	1.22	212
3	Pre	181	7.36	16	14	2.2	30.6	3.05	114	1.35	170
3	Post	188	8.35	16	14	2.22	30.1	3.105	109	1.41	180
3	Pre	174	10.8	20	17	3.34	46.6	4.422	133.8	1.32	275
3	Post	169	8.82	20	18	3.191	43.7	3.828	136.2	1.2	270
3	Pre	173	6.18	20	14	2.8	33.4	3.34	93.6	1.19	230
3	Post	172	7.93	18	15	2.85	33	3.3	92	1.16	228
Group	Time	HR (bpm)	[La] (mM/L)	RPE (legs)	RPE (overall)	VO ₂ (L·min ⁻¹)	VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (L·min ⁻¹)	VE (L·min ⁻¹)	RER	W

*Group 1: HIT; Group 2: LIT; Group 3: NT

Maximal Cycle Ergometry Testing: Lactate Threshold Raw Data

Group	Time	Observer 1		Observer 2		Observer 3		Average Watts	Average VO ₂ (L·min ⁻¹)	% of peak VO ₂
		Watts	VO ₂ (L·min ⁻¹)	Watts	VO ₂ (L·min ⁻¹)	Watts	VO ₂ (L·min ⁻¹)			
1	pre	155	2.04	138	1.77	155	2.04	149.3	1.95	57.7
1	post	176	2.42	176	2.42	176	2.42	176.0	2.42	61.6
1	pre	116	1.77	116	1.77	116	1.77	116.0	1.77	53.6
1	post	197	2.53	197	2.53	197	2.53	197.0	2.53	63.7
1	pre	140	1.98	119	1.78	119	1.78	126.0	1.85	50.8
1	post	180	2.40	180	2.40	180	2.40	180.0	2.40	51
1	pre	121	1.47	96	1.33	96	1.33	104.3	1.38	45.3
1	post	99	1.54	120	1.84	120	1.84	113.0	1.74	46.1
1	pre	129	1.69	95	1.28	124	1.53	116.0	1.50	42.4
1	post	160	2.05	160	2.05	180	2.18	166.7	2.09	53.1
1	pre	133	2.00	133	2.00	133	2.00	133.0	2.00	50
1	post	240	2.82	240	2.82	221	2.60	233.7	2.75	62.6
2	pre	100	1.34	100	1.34	100	1.34	100.0	1.34	41.4
2	post	120	1.63	185	2.20	185	2.20	163.3	2.01	54.3
2	pre	73	1.21	73	1.21	73	1.21	73.0	1.21	43.3
2	post	119	1.59	119	1.59	119	1.59	119.0	1.59	49.8
2	pre	103	1.33	103	1.33	77	1.25	94.3	1.30	45.9
2	post	120	1.70	120	1.70	120	1.70	120.0	1.70	50
2	pre	115	1.52	115	1.52	98	1.39	109.3	1.48	51
2	post	156	1.98	156	1.98	156	1.98	156.0	1.98	53.4
2	pre	103	1.42	83	1.23	103	1.42	96.3	1.36	40.7
2	post	158	1.86	158	1.86	158	1.86	158.0	1.86	50.6
2	pre	111	1.47	111	1.47	111	1.47	111.0	1.47	46.4
2	post	165	1.84	165	1.84	165	1.84	165.0	1.84	54.2
3	pre	124	1.93	124	1.93	124	1.93	124.0	1.93	51.5

3	post	157	2.11	157	2.11	157	2.11	157.0	2.11	53.6
3	pre	142	1.91	142	1.91	142	1.91	142.0	1.91	49.4
3	post	155	2.10	155	2.10	133	1.84	147.7	2.01	49.6
3	pre	80	1.16	80	1.16	80	1.16	80.0	1.16	54.1
3	post	103	1.22	103	1.22	103	1.22	103.0	1.22	55.1
3	pre	92	1.23	92	1.23	92	1.23	92.0	1.23	56
3	post	80	1.22	80	1.22	80	1.22	80.0	1.22	55
3	pre	142	1.83	142	1.83	115	1.83	133.0	1.83	54.7
3	post	111	1.77	140	1.77	140	1.77	130.3	1.77	55.5
3	pre	103	1.44	103	1.44	103	1.44	1.0	1.44	51.4
3	post	120	1.60	120	1.60	120	1.60	120.0	1.60	56.1
Group	Time	Observer 1		Observer 2		Observer 3		Average	Average	% of peak
		Watts	VO ₂ (L·min ⁻¹)	Watts	VO ₂ (L·min ⁻¹)	Watts	VO ₂ (L·min ⁻¹)	Watts	VO ₂ (L·min ⁻¹)	VO ₂

Group 1: HIT; Group 2: LIT; Group 3: NT
 High Constant-load Testing: Raw Data

Group	Trial	Rest VO ₂ (L·min ⁻¹)	3min-VO ₂ (L·min ⁻¹)	End-VO ₂ (L·min ⁻¹)	ΔVO ₂ (L·min ⁻¹)	3min-[La] (mmol/L)	End-[La] (mmol/L)	Δ[La] (mmol/L)
1	1	0.405	3.108	3.637	0.529	2.3		
1	2	0.377	3.095	3.559	0.464	2.17	10.415	8.245
1	3	0.268	3.208	3.72	0.512			
1	4	0.333	3.377	3.6	0.223	1.79	6.665	4.875
1	5	0.266	3.211	3.385	0.174			
1	1	0.372	3.022	3.526	0.504	2.07	8.41	6.34
1	2	0.254	3.085	3.241	0.156	2.14	5.47	3.33
1	3	0.465	3.098	3.338	0.24	1.635	4.72	3.085
1	4	0.299	2.902	2.966	0.064	1.475	4.29	2.815
1	5	0.321	2.974	3.067	0.093	1.67	3.56	1.89
1	1	0.269	3.21	3.957	0.747	4.79	12.84	8.05
1	2	0.299	3.439	3.734	0.295	1.45	7.47	6.02
1	3	0.233	3.337	3.742	0.405	1.32	6.32	5
1	4	0.232	3.444	3.646	0.202	1.38	5.78	4.4
1	5	0.321	3.317	3.55	0.233	1.215	5.575	4.36
1	1	0.286	2.375	3.154	0.779	1.825	13.215	11.39
1	2	0.366	2.609	3.168	0.559	1.975	12.02	10.045
1	3	0.322	2.54	2.93	0.39	1.245	9.275	8.03
1	4	0.368	2.73	2.92	0.19	1.185	7.44	6.255
1	5	0.377	2.55	2.93	0.38	1.56	9.07	7.51
1	1	0.59	3.03	3.86	0.83	3.54	13.165	9.625
1	2	0.53	3	3.48	0.48	2.11	9.925	7.815
1	3	0.46	2.96	3.47	0.51	1.88	9.34	7.46
1	4	0.46	3.125	3.52	0.395	2.42	7.925	5.505
1	5	0.525	2.935	3.17	0.235	1.855	6.35	4.495
1	1	0.382	3.2	4.03	0.83	2.56	13.06	10.5

1	2	0.35	3.395	3.815	0.42		10.3	
1	3	0.405	3.4	3.81	0.41	2.205	9.865	7.66
1	4	0.3	3.49	3.63	0.14	1.84	7.32	5.48
1	5	0.365	3.4	3.49	0.09	3.325	6.49	3.165
2	1	0.43	2.433	3.095	0.662	1.11	8.99	7.88
2	2	0.442	2.87	3.295	0.425	1.12	6.27	5.15
2	3	0.359	3.152	3.62	0.468	1.62	5.56	3.94
2	4	0.3	2.743	3.111	0.368	1.16	5.545	4.385
2	5	0.303	2.66	2.93	0.27	1.18	5.79	4.61
2	1	0.32	2.43	3.015	0.585	2.595	10.88	8.285
2	2	0.328	2.52	3.16	0.64	2.44	10.29	7.85
2	3	0.329	2.447	2.86	0.413	2.18	8.26	6.08
2	4	0.316	2.474	2.99	0.516	1.6	6.53	4.93
2	5	0.345	2.39	2.77	0.38	1.41	7.61	6.2
2	1	0.396	2.41	2.818	0.408	2.39	10.155	7.765
2	2	0.448	2.597	2.86	0.263	2.3	8.25	5.95
2	3	0.269	2.59	2.88	0.29	3.09	8.175	5.085
2	4	0.477	2.648	2.98	0.332	2.68		
2	5	0.42	2.516	2.82	0.304			
2	1	0.304	2.62	3.37	0.75	2.4	10.225	7.825
2	2	0.265	2.705	3.35	0.645		8.015	
2	3	0.35	2.74	3.25	0.51		8.94	8.94
2	4	0.32	2.84	3.2	0.36	2.06	7.72	5.66
2	5	0.374	2.65	2.9	0.25	2.01	5.575	3.565
2	1	0.52	2.65	2.98	0.33	2.655	10.39	7.735
2	2	0.36	2.85	3.09	0.24		10.625	
2	3	0.395	2.68	2.86	0.18	2.415	9.69	7.275
2	4	0.55	2.83	2.93	0.1	1.645	7.535	5.89
2	5	0.27	2.64	2.74	0.1	2.52	6.075	3.555
2	1	0.32	2.31	3.1	0.79	4.81	13.42	8.61

2	2	0.45	2.58	3.2	0.62	4.965	13.055	8.09
2	3	0.45	2.59	2.97	0.38	3.85	12.35	8.5
2	4	0.41	2.66	3.05	0.39	4.35	10.41	6.06
2	5	0.46	2.71	2.92	0.21	2.51	9.6	7.09
3	1	0.369	2.85	3.426	0.561	2.14	8.35	6.21
3	2	0.731	2.919	3.43	0.511	1.865	7.65	5.785
3	3	0.383	2.91	3.396	0.486	1.565	6.9	5.335
3	4	0.388	3.045	3.367	0.322	2.675	5.85	3.175
3	5	0.352	2.83	3.3	0.47		6.3	
3	1	0.406	3.68	4.2	0.52		6.315	
3	2	0.501	3.53	4.154	0.624	1.51	5.95	4.44
3	3	0.505	3.3	3.98	0.68	1.26	4.46	3.2
3	4	0.539	3.4	4.12	0.72	1.335	3.665	2.33
3	5	0.65	3.46	4.1	0.64		4.4	
3	1	0.328	1.63	2.12	0.49	2.38	8.97	6.59
3	2	0.303	1.595	1.99	0.395	2.405	8.525	6.12
3	3	0.364	1.595	2.05	0.455	1.66	8.205	6.545
3	4	0.287	1.6	2.04	0.44	1.45	8.01	6.56
3	5	0.356	1.595	1.97	0.375	1.43	6.25	4.82
3	1	0.39	1.72	2.21	0.49	3.765	9.22	5.455
3	2	0.29	1.85	2.3	0.45	3.15	9.57	6.42
3	3	0.3	1.92	2.485	0.565	2.885	8.745	5.86
3	4	0.25	1.93	2.46	0.53	3.03	8.86	5.83
3	5	0.32	1.93	2.41	0.48	4.515	9.96	5.445
3	1	0.36	2.65	3.25	0.6	4.43	10.68	6.25
3	2	0.225	2.85	3.23	0.38	4.465	10.875	6.41
3	3	0.34	2.86	3.29	0.43	4.33	10.94	6.61
3	4	0.41	2.85	3.4	0.55		10.215	
3	5	0.375	2.85	3.3	0.45	3.99	9.335	5.345
3	1	0.45	2.48	3.05	0.57	2.01	5.47	3.46

3	2	0.34	2.65	3.15	0.5	1.85	4.36	2.51
3	3	0.46	2.47	3.03	0.56	2.06	6.16	4.1
3	4	0.58	2.43	3.04	0.61	2	5.09	3.09
3	5	0.49	2.4	3.05	0.65	4.32	5.63	1.31

Group	Trial	Rest VO ₂ (L·min ⁻¹)	3min-VO ₂ (L·min ⁻¹)	End-VO ₂ (L·min ⁻¹)	ΔVO ₂ (L·min ⁻¹)	3min-[La] (mmol/L)	End-[La] (mmol/L)	Δ[La] (mmol/L)
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Group	Trial	3min-VE (L·min ⁻¹)	End-VE (L·min ⁻¹)	ΔVE (L·min ⁻¹)	HR-rest (beats·min ⁻¹)	3min-HR (beats·min ⁻¹)	End-[HR] (beats·min ⁻¹)	RPE (leg)	RPE (overall)
1	1	90.7	140.3	49.65	79	158	182	17	15
1	2	88.1	117.3	29.2	67	147	167	16	13
1	3	93.6	121	27.4	65	147	163	14	12
1	4	85.3	99.7	14.4	59	141	157	13	11
1	5	84.6	95.2	10.6	62	136	147	13	11
1	1	77.8	109	31.2	95	166	180	17	18
1	2	81.3	97.9	16.6	71	158	167	14	14
1	3	78.4	81.1	2.7	88	159	166	13	12
1	4	69.5	76.2	6.7	86	155	160	11	11
1	5	79.2	79.5	0.3	71	151	155	13	13
1	1	105.5	157.7	52.2	65	152	180	19	19
1	2	97.5	107.8	10.3	60	142	153	14	14
1	3	93.4	102.2	8.85	50	132	152	15	15
1	4	89.5	97.3	7.8	65	138	150	12	11
1	5	92.2	102	9.8	60	143	154	12	12

1	1	74.6	147.7	73.1	80	167	197	17	17
1	2	77.7	99.8	22.1	84	159	175	15	15
1	3	70	87.5	17.5	80	151	166	13	13
1	4	73.1	83.7	10.6	76	150	171	13	12
1	5	70.3	87.7	17.4	70	149	171	13	12
1	1	98.7	153	54.3	86	181	194	17	16
1	2	96	112	16	72	168	185	15	13
1	3	94.4	118	23.6	65	162	178	15	13
1	4	93.7	108.3	14.6	64	164	173	15	14
1	5	81	96.5	15.5	76	158	163	12	11
1	1	83	142	59	77	166	185	19	19
1	2	106	121.8	15.8	84	165	180	15	14
1	3	100.7	110	9.3	80	163	175	13	12
1	4	103.6	103.8	0.2	77	156	169	13	12
1	5	105.5	106	0.5	76	150	167	13	12
2	1	69.7	100.5	30.8	69	145	162	19	18
2	2	76.9	92	15.1	64	141	154	18	15
2	3	98.05	121.1	23.05	60	144	162	19	15
2	4	74.4	85.2	10.8	55	128	145	17	14
2	5	70.2	76.4	6.2	59	132	144	17	15
2	1	73.7	116.3	42.6	54	150	184	17	15
2	2	71.9	112	40.1	60	145	188	16	13
2	3	72.8	100.6	27.8	75	145	183	15	13
2	4	68.2	96.4	28.2	62	135	170	15	10
2	5	67.8	88.5	20.7	56	138	170	13	11
2	1	78.3	109	30.7	59	150	174	17	19
2	2	79.4	96.3	16.9	68	143	167	17	17
2	3	85.1	100.5	15.4	60	136	164	17	17
2	4	88.1	108	19.9	77	130	155	18	15
2	5	79.1	95.4	16.3	86	142	166	18	16

2	1	84.6	145	60.4	76	176	200	17	17
2	2	80.5	121.5	41	70	163	190	17	16
2	3	83.7	119.2	35.5	76	162	188	17	16
2	4	84.7	107.8	23.1	60	152	172	14	13
2	5	77.5	91	13.5	94	162	176	14	13
2	1	86.25	120	33.75	90	170	196	18	18
2	2	94.5	110	15.5	60	166	189	15	19
2	3	87	106.1	19.1	76	172	190	15	17
2	4	90	103.5	13.5	70	170	188	15	16
2	5	86.9	99.6	12.7	72	168	186	16	14
2	1	77.1	124.9	47.8	77	154	190	19	16
2	2	92.5	128.1	35.6	72	153	185	19	15
2	3	90.2	111.5	21.3	71	160	178	18	13
2	4	90.35	103.5	13.15	80	158	170	16	10
2	5	87.15	94.35	7.2	77	151	170	15	12
3	1	73.8	87.9	14.1	68	144	174	14	15
3	2	66.8	79.6	12.8	64	144	168	14	14
3	3	73.9	82.4	8.5	60	141	165	14	13
3	4	69.7	75.9	6.2	61	146	166	13	12
3	5	65.4	74.8	9.4	68	140	163	15	14
3	1	111.4	174.3	62.9	74	160	173	18	18
3	2	109	143.5	34.5	78	157	166	18	19
3	3	107.5	127.2	19.7	78	146	170	17	17
3	4	108.7	140	31.3	62	148	168	14	14
3	5	109	148.5	39.5	72	156	170	15	15
3	1	55.1	73.5	18.4	75	134	173	12	12
3	2	58.57	73.8	16.8	90	148	177	11	11
3	3	60.8	74.5	13.7	70	145	168	12	12
3	4	58.8	69.6	10.8	76	136	170	14	14
3	5	56.6	69	12.4	76	140	172	15	15

Group	Trial	3min-VE (L·min ⁻¹)	End-VE (L·min ⁻¹)	ΔVE (L·min ⁻¹)	HR-rest (beats·min ⁻¹)	3min-HR (beats·min ⁻¹)	End-[HR] (beats·min ⁻¹)	RPE (leg)	RPE (overall)
3	1	69.7	120	50.3	75	165	185	15	14
3	2	76.4	130	53.6	63	160	187	14	15
3	3	73	92.5	19.5	62	163	185	12	11
3	4	74.5	108.5	34	70	168	190	12	11
3	5	67	120	53	58	168	191	12	13
3	1	106	139.1	33.1	60	140	170	19	14
3	2	113	139	26	60	151	174	18	14
3	3	111.6	139	27.4	79	156	174	18	14
3	4	111	155	44	65	152	174	19	15
3	5	122.5	142	19.5	65	158	180	19	15
3	1	76	98.2	22.2	77	156	175	17	15
3	2	86	105.5	19.5	72	160	173	13	11
3	3	83	101.5	18.5	72	161	178	12	11
3	4	78.7	97.9	19.2	90	164	179	10	8
3	5	82	102.5	20.5	69	163	171	9	9

*Group 1: HIT; Group 2: LIT; Group 3: NT

APPENDIX E

Summary ANOVA Tables

Descriptive Data

One Way Analysis of Variance for Age (yrs)

Source of Variation	DF	SS	MS	F	P
Between Treatments	2	1.444	0.722	0.0819	0.922
Residual	15	132.333	8.822		
Total	17	133.778			

Group	Mean	Std Dev	SEM
1	23.500	2.429	0.992
2	22.833	3.920	1.600
3	23.000	2.280	0.931

One Way Analysis of Variance for Height (cm)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	75.000	37.500	0.407	0.673
Residual	15	1382.125	92.142		
Total	17	1457.125			

Group	Mean	Std Dev	SEM
1	182.75	8.23	3.358
2	180.25	10.54	4.305
3	177.75	9.88	4.033

One Way Analysis of Variance for Post-training weight - Pre-training weight(kg)

Source of Variation	DF	SS	MS	F	P
Between Treatments	2	1.111	0.555	0.182	0.836
Residual	15	45.820	3.055		
Total	17	46.931			

Group	Mean	Std Dev	SEM
1	-0.0333	1.213	0.495
2	-0.325	2.030	0.829
3	0.283	1.890	0.772

HCL Testing

Two Way Repeated Measures ANOVA for Slow Component (SC) ($L \cdot min^{-1}$)

Source of Variation	DF	SS	MS	F	P
Group	2	0.308	0.154	2.966	0.082
Group(Subjects)	15	0.780	0.052		
Trial	4	0.891	0.223	32.169	<0.001
Group x Trial	8	0.534	0.067	9.644	<0.001
Residual	60	0.415	0.007		
Total	89	2.928	0.033		

One Way Repeated Measures ANOVA for SC : Trial at HIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	0.234	0.047		
Trial	4	1.016	0.25	33.486	<0.001
Residual	20	0.152	0.008		
Total	29	1.402			

Trial	Mean	Std Dev	SEM
1	0.703	0.148	0.0605
2	0.396	0.146	0.0596
3	0.411	0.0997	0.0407
4	0.202	0.110	0.0450
5	0.201	0.109	0.0443

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	0.502	5	14.130	Yes
1 vs. 4	0.501	5	14.088	Yes
1 vs. 2	0.307	5	8.650	Yes
1 vs. 3	0.292	5	8.214	Yes
3 vs. 5	0.210	5	5.916	Yes
3 vs. 4	0.209	5	5.874	Yes
3 vs. 2	0.0155	5	0.436	No
2 vs. 5	0.195	5	5.480	Yes
2 vs. 4	0.193	5	5.438	Yes
4 vs. 5	0.00150	5	0.0422	No

One Way Repeated Measures ANOVA for SC: Trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	0.400	0.080		
Trial	4	0.395	0.099	12.223	<0.001
Residual	20	0.161	0.008		
Total	29	0.956			

Trial	Mean	Std Dev	SEM
1	0.588	0.185	0.0756
2	0.472	0.190	0.0774
3	0.374	0.121	0.0495
4	0.344	0.136	0.0554
5	0.252	0.0941	0.0384

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	0.335		5	9.137 Yes
1 vs. 4	0.243		5	6.629 Yes
1 vs. 3	0.214		5	5.834 Yes
1 vs. 2	0.115		5	3.144 No
2 vs. 5	0.220		5	5.993 Yes
2 vs. 4	0.128		5	3.485 No
2 vs. 3	0.0987		5	2.690 No
3 vs. 5	0.121		5	3.303 No
3 vs. 4	0.0292		5	0.795 No
4 vs. 5	0.0920		5	2.508 No

One Way Repeated Measures ANOVA for SC: Trial at NT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	0.145	0.029		
Trial	4	0.0145	0.004	0.709	0.596
Residual	20	0.102	0.005		
Total	29	0.262			

Group	Mean	Std Dev	SEM
1	0.538	0.0454	0.0186
2	0.477	0.0896	0.0366
3	0.529	0.0919	0.0375
4	0.529	0.137	0.0561
5	0.511	0.110	0.0450

One Way ANOVA for SC: Group at Trial 1

Source of Variation	DF	SS	MS	F	P
Group	2	0.0858	0.043	2.68	>.05
Residual	75	1.195	0.016		
Total	77	0.377			

Group	Mean	Std Dev	SEM
1	0.703	0.148	0.0605
2	0.588	0.185	0.0756
3	0.538	0.0454	0.0186

One Way ANOVA for SC: Group at Trial 2

Source of Variation	DF	SS	MS	F	P
Group	2	0.0249	0.0124	0.775	>.05
Residual	75	1.195	0.016		
Total	77	0.351			

Group	Mean	Std Dev	SEM
1	0.396	0.146	0.0596
2	0.472	0.190	0.0774
3	0.477	0.0896	0.0366

One Way ANOVA for SC: Group at Trial 3

Source of Variation	DF	SS	MS	F	P
Group	2	0.0793	0.0397	2.48	>.05
Residual	75	1.195	0.016		
Total	77	0.245			

Group	Mean	Std Dev	SEM
1	0.411	0.0997	0.0407
2	0.374	0.121	0.0495
3	0.529	0.0919	0.0375

One Way ANOVA for SC: Group at Trial 4

Source of Variation	DF	SS	MS	F	P
Group	2	0.321	0.161	10.06	<0.01
Residual	75	1.195	0.016		
Total	77	0.568			

Group	Mean	Std Dev	SEM
1	0.202	0.110	0.0450
2	0.344	0.136	0.0554
3	0.529	0.137	0.0561

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
3 vs. 1	0.326	3	6.227	Yes
3 vs. 2	0.184	3	3.517	No
2 vs. 1	0.142	3	2.710	No

One Way ANOVA for SC: Group at Trial 5

Source of Variation	DF	SS	MS	F	P
Group	2	0.331	0.166	10.375	<0.01
Residual	75	1.195	0.016		
Total	17	0.495			

Group	Mean	Std Dev	SEM
1	0.201	0.109	0.0443
2	0.252	0.0941	0.0384
3	0.511	0.110	0.0450

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
3 vs. 1	0.310	3	7.261	Yes
3 vs. 2	0.259	3	6.055	Yes
2 vs. 1	0.0515	3	1.206	No

Two Way Repeated Measures ANOVA on 3 minute VO₂ (L · min⁻¹)

Source of Variation	DF	SS	MS	F	P
Group	2	5.300	2.650	2.698	0.100
Group(Subjects)	15	14.731	0.982		
Trial	4	0.254	0.064	6.367	<0.001
Group x Trial	8	0.101	0.013	1.271	0.276
Residual	60	0.598	0.099		
Total	89	20.983	0.236		

One Way ANOVA on 3 minute VO₂: Trials

Source of Variation	DF	SS	MS	F	P
Between Trials	4	0.254	0.0635	0.260	0.903
Residual	85	20.730	0.244		
Total	89	20.983			

Group	Mean	Std Dev	SEM
1	2.657	0.514	0.121
2	2.786	0.492	0.116
3	2.766	0.481	0.113
4	2.807	0.500	0.118
5	2.723	0.481	0.113

Two Way Repeated Measures ANOVA for End Exercise VO₂ (L · min⁻¹)

Source of Variation	DF	SS	MS	F	P
Group	2	3.664	1.832	1.657	0.224
Group(Subjects)	15	16.580	1.105		
Trial	4	0.531	0.133	12.055	<0.001
Group x Trial	8	0.407	0.051	4.625	<0.001
Residual	60	0.661	0.0110		
Total	89	21.843	0.245		

One Way Repeated Measures ANOVA on End Exercise VO₂: Trial at HIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	2.103	0.421		
Trial	4	0.612	0.153	14.497	<0.001
Residual	20	0.211	0.0106		
Total	29	2.926			

Trial	Mean	Std Dev	SEM
1	3.694	0.326	0.133
2	3.500	0.259	0.106
3	3.502	0.333	0.136
4	3.380	0.342	0.140
5	3.265	0.248	0.101

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	0.429		5	10.223 Yes

1 vs. 4	0.314	5	7.480	Yes
1 vs. 2	0.194	5	4.638	Yes
1 vs. 3	0.192	5	4.587	Yes
3 vs. 5	0.236	5	5.636	Yes
3 vs. 4	0.121	5	2.894	No
3 vs. 2	0.00217	5	0.052	No
2 vs. 5	0.234	5	5.584	Yes
2 vs. 4	0.119	5	2.842	No
4 vs. 5	0.115	5	2.742	No

One Way Repeated Measures ANOVA for End Exercise VO₂: Trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	0.543	0.109		
Between Trials	4	0.319	0.08	4.911	0.006
Residual	20	0.325	0.0162		
Total	29	1.187			

Group	Mean	Std Dev	SEM
1	3.063	0.182	0.0744
2	3.159	0.174	0.0709
3	3.073	0.306	0.125
4	3.044	0.0989	0.0404
5	2.847	0.0814	0.0332

Multiple Comparison Procedures (Tukey Test):

Group	Diff of Means	p	q	P<0.05
2 vs. 5	0.313	5	6.006	Yes
2 vs. 4	0.116	5	2.223	No
2 vs. 1	0.0962	5	1.848	No
2 vs. 3	0.0858	5	1.650	No
3 vs. 5	0.227	5	4.356	Yes
3 vs. 4	0.0298	5	0.573	No
3 vs. 1	0.0103	5	0.199	No
1 vs. 5	0.216	5	4.157	No
1 vs. 4	0.0195	5	0.375	No
4 vs. 5	0.197	5	3.783	No

One Way Repeated Measures ANOVA for End Exercise VO₂: Trial for NT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	13.934	2.787		
Trials	4	0.00761	0.002	0.305	0.871
Residual	20	0.125	0.0062		
Total	29	14.066			

Group	Mean	Std Dev	SEM
1	3.043	0.784	0.320
2	3.042	0.786	0.321
3	3.038	0.687	0.281
4	3.071	0.739	0.302
5	3.022	0.748	0.305

One Way Analysis of Variance for End Exercise VO₂: Group at Trial 1

Source of Variation	DF	SS	MS	F	P
Group	2	1.646	0.823	3.57	<.05
Residual	75	17.246	0.2299		
Total	77	18.892			

Group	Mean	Std Dev	SEM
1	3.694	0.326	0.133
2	3.063	0.182	0.0744
3	3.043	0.784	0.320

One Way Analysis of Variance for End Exercise VO₂: Group at Trial 2

Source of Variation	DF	SS	MS	F	P
Group	2	0.677	0.338	1.47	>.05
Residual	75	17.246	0.2299		
Total	77	17.923			

Group	Mean	Std Dev	SEM
1	3.500	0.259	0.106
2	3.159	0.174	0.0709
3	3.042	0.786	0.321

One Way Analysis of Variance for End Exercise VO₂: Group at Trial 3

Source of Variation	DF	SS	MS	F	P
Group	2	0.798	0.399	1.74	>.05
Residual	75	17.246	0.2299		
Total	77	18.044			

Group	Mean	Std Dev	SEM
1	3.502	0.333	0.136
2	3.073	0.306	0.125
3	3.038	0.687	0.281

One Way Analysis of Variance for End Exercise VO₂: Group at Trial 4

Source of Variation	DF	SS	MS	F	P
Group	2	0.420	0.210	0.913	>.05
Residual	75	17.246	0.2299		
Total	77	17.666			

Group	Mean	Std Dev	SEM
1	3.380	0.342	0.140
2	3.044	0.0989	0.0404
3	3.071	0.739	0.302

One Way Analysis of Variance on End Exercise VO₂: Group at Trial 5

Source of Variation	DF	SS	MS	F	P
Group	2	0.531	0.265	1.15	>.05
Residual	75	17.246	0.2299		
Total	77	17.777			

Group	Mean	Std Dev	SEM
1	3.265	0.248	0.101
2	2.847	0.0814	0.0332
3	3.022	0.748	0.305

Two Way Repeated Measures ANOVA for ΔVE

Source of Variation	DF	SS	MS	F	P
Group	2	378.934	189.5	0.436	0.654
Group(Subjects)	15	6516.020	434.4		
Trial	4	8742.036	2185.5	39.193	<0.001
Group x Trial	8	2996.763	374.6	6.718	<0.001
Residual	60	3345.799	55.76		
Total	89	21979.551	246.961		

One Way Repeated Measures ANOVA for ΔVE : Trial at HIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	1049.028	209.81		
Between Trial	4	8221.843	2055.5	42.921	<0.001
Residual	20	957.783	47.889		
Total	29	10228.654			
Group	Mean	Std Dev	SEM		
1	53.242	13.627	5.563		
2	18.333	6.507	2.656		
3	14.892	9.545	3.897		
4	9.050	5.427	2.215		
5	9.017	7.266	2.966		

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	44.225	5	15.654	Yes
1 vs. 4	44.192	5	15.642	Yes
1 vs. 3	38.350	5	13.574	Yes
1 vs. 2	34.908	5	12.356	Yes
2 vs. 5	9.317	5	3.298	No
2 vs. 4	9.283	5	3.286	No
2 vs. 3	3.442	5	1.218	No
3 vs. 5	5.875	5	2.080	No
3 vs. 4	5.842	5	2.068	No
4 vs. 5	0.0333	5	0.0118	No

One Way Repeated Measures ANOVA for Δ VE: Trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	1326.317	265.3		
Between Trials	4	2759.201	689.8	17.029	<0.001
Residual	20	810.139	40.51		
Total	29	4895.657			

Group	Mean	Std Dev	SEM
1	41.008	11.729	4.788
2	27.367	12.780	5.217
3	23.700	7.109	2.902
4	18.108	6.771	2.764
5	12.767	5.479	2.237

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	28.242		5	10.869 Yes
1 vs. 4	22.900		5	8.813 Yes
1 vs. 3	17.308		5	6.661 Yes
1 vs. 2	13.642		5	5.250 Yes
2 vs. 5	14.600		5	5.619 Yes
2 vs. 4	9.258		5	3.563 No
2 vs. 3	3.667		5	1.411 No
3 vs. 5	10.933		5	4.208 No
3 vs. 4	5.592		5	2.152 No
4 vs. 5	5.342		5	2.056 No

One Way Repeated Measures ANOVA for Δ VE: Trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	4140.675	828.1		
Between Trials	4	757.755	189.4	2.401	0.084
Residual	20	1577.877	78.89		
Total	29	6476.307			
Group	Mean	Std Dev	SEM		
1	33.500	19.385	7.914		
2	27.200	15.010	6.128		
3	17.883	6.363	2.597		
4	24.250	14.608	5.964		
5	25.717	16.993	6.937		

One Way ANOVA on ΔVE: Group at trial 1

Source of Variation	DF	SS	MS	F	P
Between Groups	2	1191.526	595.8	4.485	<0.05
Residual	75	9961.8	132.82		
Total	77	11153.33			

Group	Mean	Std Dev	SEM
1	53.242	13.627	5.563
2	41.008	11.729	4.788
3	33.500	19.385	7.914

One Way ANOVA on ΔVE: Group at trial 2

Source of Variation	DF	SS	MS	F	P
Between Groups	2	320.493	160.25	1.206	>0.05
Residual	75	9961.8	132.82		
Total	77	10282.3			
Group	Mean	Std Dev	SEM		
1	18.333	6.507	2.656		
2	27.367	12.780	5.217		
3	27.200	15.010	6.128		

One Way ANOVA on ΔVE: Group at trial 3

Source of Variation	DF	SS	MS	F	P
Between Groups	2	240.741	120.4	.906	>0.05
Residual	75	9961.8	132.82		
Total	77	10202.5			

Group	Mean	Std Dev	SEM
1	14.892	9.545	3.897
2	23.700	7.109	2.902
3	17.883	6.363	2.597

One Way ANOVA on ΔVE: Group at trial 4

Source of Variation	DF	SS	MS	F	P
Between Groups	2	701.627	350.8	2.64	>0.05
Residual	75	9961.8	132.82		
Total	77	2145.099			

Group	Mean	Std Dev	SEM
1	9.050	5.427	2.215
2	18.108	6.771	2.764
3	24.250	14.608	5.964

One Way ANOVA on ΔVE: Group at trial 5

Source of Variation	DF	SS	MS	F	P
Between Groups	2	921.310	460.7	3.46	0.049
Residual	75	9961.8	132.82		
Total	17	2779.120			

Group	Mean	Std Dev	SEM
1	9.017	7.266	2.966
2	12.767	5.479	2.237
3	25.717	16.993	6.937

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
3 vs. 1	16.700	3	3.676	Yes
3 vs. 2	12.950	3	2.850	No
2 vs. 1	3.750	3	0.825	No

Two Way Repeated Measures ANOVA for End Exercise [La] (mmol · L)

Source of Variation	DF	SS	MS	F	P
Group	2	23.686	11.843	0.575	0.577
Group(Subjects)	13	267.927	20.610		
Trial	4	138.442	34.610	51.657	<0.001
Group x Trial	8	41.228	5.153	7.692	<0.001
Residual	52	34.840	0.670		
Total	79	496.098	6.280		

One Way Repeated Measures ANOVA for End Exercise [La]: trial at HIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	4	77.328	19.332		
Between Trials	4	114.193	28.548	39.034	<0.001
Residual	16	11.702	0.731		
Total	24	203.223			

Group	Mean	Std Dev	SEM
1	12.138	2.089	0.934
2	9.037	2.572	1.150
3	7.904	2.260	1.011
4	6.551	1.498	0.670
5	6.209	1.981	0.886

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	5.929		5	15.502 Yes
1 vs. 4	5.587		5	14.608 Yes
1 vs. 3	4.234		5	11.071 Yes
1 vs. 2	3.101		5	8.108 Yes
2 vs. 5	2.828		5	7.394 Yes
2 vs. 4	2.486		5	6.500 Yes
2 vs. 3	1.133		5	2.962 No
3 vs. 5	1.695		5	4.432 Yes
3 vs. 4	1.353		5	3.538 No
4 vs. 5	0.342		5	0.894 No

One Way Repeated Measures ANOVA for End Exercise [La]: trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	4	74.601	18.650		
Between Trials	4	48.676	12.169	16.366	<0.001
Residual	16	11.897	0.744		
Total	24	135.174			

Group	Mean	Std Dev	SEM
1	10.781	1.631	0.730
2	9.651	2.601	1.163
3	8.960	2.453	1.097
4	7.548	1.821	0.815
5	6.930	1.692	0.757

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	3.851		5	9.986 Yes
1 vs. 4	3.233		5	8.384 Yes
1 vs. 3	1.821		5	4.722 Yes
1 vs. 2	1.130		5	2.930 No

2 vs. 5	2.721	5	7.056	Yes
2 vs. 4	2.103	5	5.453	Yes
2 vs. 3	0.691	5	1.792	No
3 vs. 5	2.030	5	5.264	Yes
3 vs. 4	1.412	5	3.662	No
4 vs. 5	0.618	5	1.603	No

One Way Repeated Measures ANOVA for End Exercise [La]: trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	115.998	23.200		
Between Trials	4	6.776	1.694	3.014	0.043
Residual	20	11.241	0.562		
Total	29	134.015			

Group	Mean	Std Dev	SEM
1	8.167	1.939	0.792
2	7.822	2.383	0.973
3	7.568	2.247	0.917
4	6.948	2.486	1.015
5	6.979	2.186	0.893

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 4	1.219	5	3.983	No
1 vs. 5	1.188	5	3.883	No
1 vs. 3	0.599	5	1.958	No
1 vs. 2	0.346	5	1.130	No
2 vs. 4	0.873	5	2.853	No
2 vs. 5	0.842	5	2.753	No
2 vs. 3	0.253	5	0.828	No
3 vs. 4	0.620	5	2.026	No
3 vs. 5	0.589	5	1.925	No
5 vs. 4	0.0308	5	0.101	No

One Way ANOVA for End Exercise [La]: group at trial 1

Source of Variation	DF	SS	MS	F	P
Between Groups	2	45.243	22.622	4.85	<0.05
Residual	65	302.8	4.66		
Total	67	348.043			

Group	Mean	Std Dev	SEM
1	12.138	2.089	0.934
2	10.781	1.631	0.730
3	8.167	1.939	0.792

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	3.971		3	4.882 Yes
1 vs. 2	1.357		3	1.597 No
2 vs. 3	2.614		3	3.213 No

One Way ANOVA for End Exercise [La]: group at trial 2

Source of Variation	DF	SS	MS	F	P
Between Groups	2	9.633	4.817	1.03	>0.05
Residual	65	302.8	4.66		
Total	67	312.433			

Group	Mean	Std Dev	SEM
1	9.037	2.572	1.150
2	9.651	2.601	1.163
3	7.822	2.383	0.973

One Way ANOVA for End Exercise [La]: group at trial 3

Source of Variation	DF	SS	MS	F	P
Between Treatments	2	5.585	2.793	0.59	>0.05
Residual	65	302.8	4.66		
Residual	67	308.385			
Total					

Group	Mean	Std Dev	SEM
1	7.904	2.260	1.011
2	8.960	2.453	1.097
3	7.568	2.247	0.917

One Way ANOVA for End Exercise [La]: group at trial 4

Source of Variation	DF	SS	MS	F	P
Between Treatments	2	2.523	1.262	0.27	>0.05
Residual	65	302.8	4.66		
Total	67	305.32			

Group	Mean	Std Dev	SEM
1	6.551	1.498	0.670
2	7.548	1.821	0.815
3	6.948	2.486	1.015

One Way ANOVA for End Exercise [La]: group at trial 5

Source of Variation	DF	SS	MS	F	P
Between Treatments	2	1.929	0.964	0.21	>0.05
Residual	65	302.8	4.66		
Total	67	304.73			

Group	Mean	Std Dev	SEM
1	6.209	1.981	0.886
2	6.930	1.692	0.757
3	6.979	2.186	0.893

Two Way Repeated Measures ANOVA on End Exercise HR (beats · min⁻¹)

Source of Variation	DF	SS	MS	F	P
Group	2	584.600	292.300	0.576	0.574
Group(Subjects)	15	7605.500	507.033		
Trial	4	2434.067	608.517	34.961	<0.001
Group x Trial	8	1472.400	184.050	10.574	<0.001
Residual	60	1044.333	17.406		
Total	89	13140.900	147.651		

One Way Repeated Measures ANOVA: trial at HIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	1746.400	349.280		
Between Trials	4	2592.867	648.217	30.581	<0.001
Residual	20	423.933	21.197		
Total	29	4763.200			

Group	Mean	Std Dev	SEM
1	186.333	7.394	3.018
2	171.167	11.392	4.651
3	166.667	9.245	3.774
4	163.333	9.092	3.712
5	159.500	9.028	3.686

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 5	26.833	5	14.276	Yes
1 vs. 4	23.000	5	12.237	Yes
1 vs. 3	19.667	5	10.463	Yes
1 vs. 2	15.167	5	8.069	Yes
2 vs. 5	11.667	5	6.207	Yes
2 vs. 4	7.833	5	4.168	No
2 vs. 3	4.500	5	2.394	No
3 vs. 5	7.167	5	3.813	No
3 vs. 4	3.333	5	1.773	No
4 vs. 5	3.833	5	2.039	No

One Way Repeated Measures ANOVA: trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	4572.000	914.400		
Between Trials	4	1304.467	326.117	18.202	<0.001
Residual	20	358.333	17.917		
Total	29	6234.800			

Group	Mean	Std Dev	SEM
1	184.333	14.278	5.829
2	178.833	14.878	6.074
3	177.500	11.996	4.897
4	166.667	14.909	6.086
5	168.667	13.952	5.696

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 4	17.667	5	10.224	Yes
1 vs. 5	15.667	5	9.066	Yes
1 vs. 3	6.833	5	3.954	No
1 vs. 2	5.500	5	3.183	No
2 vs. 4	12.167	5	7.041	Yes
2 vs. 5	10.167	5	5.883	Yes
2 vs. 3	1.333	5	0.772	No
3 vs. 4	10.833	5	6.269	Yes
3 vs. 5	8.833	5	5.112	Yes
5 vs. 4	2.000	5	1.157	No

One Way Repeated Measures ANOVA: trial at LIT

Source of Variation	DF	SS	MS	F	P
Between Subjects	5	1287.100	257.420		
Between Trials	4	9.133	2.283	0.174	0.949
Residual	20	262.067	13.103		
Total	29	1558.300			

Group	Mean	Std Dev	SEM
1	175.000	5.177	2.113
2	174.167	7.468	3.049
3	173.333	7.312	2.985
4	174.500	8.894	3.631
5	174.500	9.731	3.973

One Way ANOVA for HR: Group at trial 1

Source of Variation	DF	SS	MS	F	P
Between Groups	2	439.111	219.556	1.904	>0.05
Residual	75	8649.9	115.3		
Total	77	9089.111			

Group	Mean	Std Dev	SEM
1	186.333	7.394	3.018
2	184.333	14.278	5.829
3	175.000	5.177	2.113

One Way ANOVA for HR: Group at trial 2

Source of Variation	DF	SS	MS	F	P
Between Groups	2	179.111	89.556	0.78	>0.05
Residual	75	8649.9	115.3		
Total	77	8829.01			

Group	Mean	Std Dev	SEM
1	171.167	11.392	4.651
2	178.833	14.878	6.074
3	174.167	7.468	3.049

One Way Analysis of Variance for HR: group at trial 3

Source of Variation	DF	SS	MS	F	P
Between Groups	2	358.333	179.167	1.55	>0.05
Residual	75	8649.9	115.3		
Total	77	1772.500			

Group	Mean	Std Dev	SEM
1	166.667	9.245	3.774
2	177.500	11.996	4.897
3	173.333	7.312	2.985

One Way Analysis of Variance for HR: group at trial 4

Source of Variation	DF	SS	MS	F	P
Between Groups	2	394.333	197.167	1.71	>0.05
Residual	75	8649.9	115.3		
Total	77	9044.333			

Group	Mean	Std Dev	SEM
1	163.333	9.092	3.712
2	166.667	14.909	6.086
3	174.500	8.894	3.631

One Way Analysis of Variance for HR: group at trial 5

Source of Variation	DF	SS	MS	F	P
Between Groups	2	686.111	343.056	2.98	>0.05
Residual	75	8649.9	115.3		
Total	77	9366.011			

Group	Mean	Std Dev	SEM
1	159.500	9.028	3.686
2	168.667	13.952	5.696
3	174.500	9.731	3.973

Maximal Exercise Testing

One Way ANOVA for Maximal Heart Rate (Δ HR)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	32.333	16.167	0.592	0.566
Residual	15	409.667	27.311		
Total	17	442.000			

Group	Mean	Std Dev	SEM
1	-1.333	4.274	1.745
2	1.833	6.494	2.651
3	-0.500	4.637	1.893

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	183.833	4.491	1.833
Post	182.500	8.337	3.403

LIT	Mean	Std Dev	SEM
Pre	182.500	11.962	4.884
Post	184.333	13.186	5.383

NT	Mean	Std Dev	SEM
Pre	180.333	6.314	2.578
Post	179.833	7.574	3.092

One Way ANOVA for peak [La] in mmol · L (Δ [La])

Source of Variation	DF	SS	MS	F	P
Between Groups	2	9.861	4.931	1.056	0.372
Residual	15	70.065	4.671		
Total	17	79.926			

Group	Mean	Std Dev	SEM
1	1.489	2.771	1.131
2	0.282	2.041	0.833
3	-0.286	1.472	0.601

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	8.678	2.261	0.923
Post	10.167	3.077	1.256

LIT	Mean	Std Dev	SEM
Pre	8.563	0.885	0.361
Post	8.844	1.883	0.769

NT	Mean	Std Dev	SEM
Pre	9.463	2.276	0.929
Post	9.177	0.988	0.403

One Way ANOVA for RPE legs (Δ RPE-L)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	7.444	3.722	3.317	0.064
Residual	15	16.833	1.122		
Total	17	24.278			

Group	Mean	Std Dev	SEM
1	0.667	0.816	0.333
2	1.000	1.414	0.577
3	-0.500	0.837	0.342

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	17.500	1.517	0.619
Post	18.167	0.983	0.401

LIT	Mean	Std Dev	SEM
Pre	17.500	0.837	0.342
Post	18.500	1.378	0.563

NT	Mean	Std Dev	SEM
Pre	18.000	2.098	0.856
Post	17.500	1.871	0.764

One Way ANOVA for Overall RPE (Δ RPE-O)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	10.333	5.167	2.058	0.162
Residual	15	37.667	2.511		
Total	17	48.000			

Group	Mean	Std Dev	SEM
1	0.667	0.816	0.333
2	-1.167	2.137	0.872
3	-0.500	1.517	0.619

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	16.667	0.816	0.333
Post	17.333	1.033	0.422

LIT	Mean	Std Dev	SEM
Pre	17.333	1.633	0.667
Post	16.167	2.639	1.078

NT	Mean	Std Dev	SEM
Pre	16.167	1.941	0.792
Post	15.667	2.066	0.843

One Way ANOVA for peak VO₂ in L · min⁻¹ (Δ VO₂)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	1.054	0.527	13.35	<0.001
Residual	15	0.592	0.0395		
Total	17	1.646			

Group	Mean	Std Dev	SEM
1	0.640	0.259	0.106
2	0.446	0.189	0.0771
3	0.0580	0.125	0.0511

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	0.582	3	7.174	Yes
1 vs. 2	0.194	3	2.387	No
2 vs. 3	0.388	3	4.787	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	3.478	0.344	0.140
Post	4.118	0.353	0.144

LIT	Mean	Std Dev	SEM
Pre	3.063	0.211	0.0861
Post	3.509	0.217	0.0886

NT	Mean	Std Dev	SEM
Pre	3.020	0.749	0.306
Post	3.078	0.804	0.328

One Way ANOVA for peak VO₂ in ml · kg⁻¹min⁻¹ (ΔVO_2)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	124.526	62.263	8.404	0.004
Residual	15	111.126	7.408		
Total	17	235.653			

Group	Mean	Std Dev	SEM
1	6.827	3.308	1.351
2	4.975	2.288	0.934
3	0.557	2.459	1.004

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	6.270	3	5.643	Yes
1 vs. 2	1.852	3	1.666	No
2 vs. 3	4.418	3	3.976	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	40.642	5.738	2.342
Post	47.467	6.476	2.644

LIT	Mean	Std Dev	SEM
Pre	33.817	5.356	2.187
Post	38.792	5.158	2.106

NT	Mean	Std Dev	SEM
Pre	37.838	6.569	2.682
Post	38.395	7.206	2.942

One Way ANOVA for VCO₂ (Δ VCO₂)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	1.529	0.765	8.737	0.003
Residual	15	1.313	0.0875		
Total	17	2.842			

Group	Mean	Std Dev	SEM
1	0.606	0.231	0.0941
2	0.448	0.365	0.149
3	-0.0758	0.276	0.113

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	0.682	3	5.649	Yes
1 vs. 2	0.159	3	1.314	No
2 vs. 3	0.523	3	4.335	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	4.201	0.506	0.206
Post	4.808	0.505	0.206

LIT	Mean	Std Dev	SEM
Pre	3.597	0.202	0.0823
Post	4.045	0.302	0.123

	Mean	Std Dev	SEM
Pre	3.826	0.840	0.343
Post	3.751	0.875	0.357

One Way ANOVA for Peak Ventilation (ΔVE)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	1693.9	846.936	6.448	0.010
Residual	15	1970.4	131.357		
Total	17	3664.2			

Group	Mean	Std Dev	SEM
1	23.517	8.310	3.392
2	24.317	16.362	6.680
3	3.350	7.569	3.090

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
2 vs. 3	20.967	3	4.481	Yes
2 vs. 1	0.800	3	0.171	No
1 vs. 3	20.167	3	4.310	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	126.400	21.048	8.593
Post	149.917	24.789	10.120

LIT	Mean	Std Dev	SEM
Pre	105.567	11.641	4.752
Post	129.883	21.099	8.614

NT	Mean	Std Dev	SEM
Pre	122.350	26.660	10.884
Post	125.700	32.655	13.331

One Way ANOVA for peak RER (ΔRER)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	0.00573	0.00287	0.849	0.448
Residual	15	0.0507	0.00338		
Total	17	0.0564			

Group	Mean	Std Dev	SEM
1	-0.0267	0.0535	0.0219
2	-0.0001	0.0548	0.0224
3	-0.0433	0.0653	0.0267

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	1.200	0.0473	0.0193
Post	1.173	0.0225	0.00919

LIT	Mean	Std Dev	SEM
Pre	1.180	0.0460	0.0188
Post	1.180	0.0329	0.0134

NT	Mean	Std Dev	SEM
Pre	1.272	0.0679	0.0277
Post	1.228	0.0911	0.0372

One Way ANOVA for Peak Watts (Δ Watts)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	5846.3	2923.2	13.21	<0.001
Residual	15	3318.2	221.2		
Total	17	9164.5			
Group	Mean	Std Dev	SEM		
1	53.167	14.798	6.041		
2	41.000	12.083	4.933		
3	10.333	17.282	7.055		

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	42.833	3	7.054	Yes
1 vs. 2	12.167	3	2.004	No
2 vs. 3	30.667	3	5.051	Yes

HIT	Mean	Std Dev	SEM
Pre	297.667	33.927	13.851
Post	350.833	37.605	15.352

LIT	Mean	Std Dev	SEM
Pre	257.167	18.357	7.494
Post	298.167	24.351	9.941

NT	Mean	Std Dev	SEM
Pre	247.833	64.991	26.532
Post	258.167	62.400	25.475

Lactate Threshold

One Way ANOVA for Watts at LT (Δ Watts@LT)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	6751.76	3375.9	6.274	0.010
Residual	15	8071.5	538.1		
Total	17	14823.26			

Group	Mean	Std Dev	SEM
1	53.633	33.815	13.805
2	49.567	13.755	5.616
3	10.667	16.782	6.851

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	42.967	3	4.537	Yes
1 vs. 2	4.067	3	0.429	No
2 vs. 3	38.900	3	4.108	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre	124.100	15.755	6.432
Post	177.733	39.557	16.149

LIT	Mean	Std Dev	SEM
Pre	97.317	13.707	5.596
Post	146.883	21.469	8.765

NT	Mean	Std Dev	SEM
Pre	112.333	24.451	9.982
Post	123.000	28.557	11.658

One Way ANOVA for VO₂ at LT (Δ VO₂@LT)

Source of Variation	DF	SS	MS	F	P
Between Groups	2	0.858	0.429	27.738	<0.001
Residual	15	0.232	0.0155		
Total	17	1.090			

Group	Mean	Std Dev	SEM
1	0.580	0.157	0.0640
2	0.470	0.114	0.0465
3	0.0717	0.0943	0.0385

Multiple Comparison Procedures (Tukey Test):

Comparison	Diff of Means	p	q	P<0.05
1 vs. 3	0.508	3	10.010	Yes
1 vs. 2	0.110	3	2.166	No
2 vs. 3	0.398	3	7.844	Yes

Summary of actual means:

HIT	Mean	Std Dev	SEM
Pre1	1.742	0.250	0.102
Post	2.322	0.356	0.145

LIT	Mean	Std Dev	SEM
Pre	1.360	0.103	0.0420
Post	1.830	0.161	0.0659

NT	Mean	Std Dev	SEM
Pre	1.583	0.350	0.143
Post	1.655	0.382	0.156

Appendix E
Summary Regression Analyses

Linear Regression: ΔVE regressed on the Slow Component of VO₂

	DF	SS	MS	F	P
Regression	1	1.503	1.503	92.775	<0.001
Residual	88	1.425	0.016		
Total	89	2.928	0.033		

Slow Component of VO₂ = 0.239 + (0.00827 x ΔVE)

R = 0.72 R² = 0.513

SEE = 0.127

	Coefficient	Std. Error	t	P
Constant	0.239	0.0244	9.791	<0.001
Delta VE	0.00827	0.000858	9.632	<0.001

N = 90.000

Linear Regression: End Exercise [La] regressed on the Slow Component of VO₂

	DF	SS	MS	F	P
Regression	1	0.477	0.477	15.672	<0.001
Residual	83	2.369	0.0285		
Total	84	2.816	0.0335		

Slow Component of VO₂ = 0.195 + (0.0297 x End exercise [La])

R = 0.40 R² = 0.16

SEE = 0.169

	Coefficient	Std. Error	t	P
Constant	0.195	0.0642	3.044	0.003
End Exercise [La]	0.0297	0.00750	4.959	<0.001

N = 85.000

VITA

Jeffrey Vincent was raised by Samuel and Mary Ocel in the Midwestern town of Apple Valley, Minnesota where he spent his childhood and young adulthood:

playing games together and battling with,
competing against (both academically and athletically)
rooting for and probably sometimes against,

his seven brothers and sisters, who at the age of 31 years, he misses dearly.

Jeffrey received his B.S. degree in Corporate and Community Fitness from North Dakota State University in Fargo, North Dakota. At NDSU, Jeff was a 3X NCAA All-American and a member of the 1988 Division II National Championship wrestling squad.

Upon graduation from NDSU, Jeffrey immediately entered the Masters degree program at the University of Wisconsin at La Crosse under the mentorship of Drs. John Porcari and Phillip Wilson. While attending La Crosse, Jeff earned the Preston Clayton Award yearly to top student of that class. After completing his MS degree in Cardiac rehabilitation, Jeffrey remained at La Crosse as the Director of Cardiac Rehabilitation and as an Physiologist at St. Francis Medical Center.

With the desire, and a little push from Drs. Porcari and Wilson, to advance his knowledge and become more active in clinical research, Jeffrey returned to school at Virginia Polytechnic Institute and State University in Blacksburg, Virginia to receive his doctorate in Clinical Exercise Physiology. At Virginia Tech, Jeffrey was the Clinical Laboratory Coordinator for the Cardiac and Intervention Center and had an active role in both doctoral and the masters students research

endeavors. The week Jeffrey finished data collection for his dissertation, he uprooted his family and moved to Boston, MA.

Currently, Jeffrey is employed as a Clinical Physiologist at the Beth Israel Deaconess Medical Center in Boston, Massachusetts.