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UNIVERSITY OF ALBERTA

EXPERIMENTAL STUDIES ON THE PHYSIOLOGICAL ADAPTATIONS OF TAPERING IN ENDURANCE CYCLISTS

BY

J. PATRICK NEARY

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled, *"Experimental Studies on the Physiological Adaptations of Tapering in Endurance Cyclists"* submitted by J. Patrick Neary in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

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DEDICATION

This thesis is dedicated to my best friend and wife, Brenda, for her unselfish committment and continued support throughout our years. Without your understanding, and love this would not have been possible. And, to our first son Shamus John from whom I have taken time to complete my thesis. Thank you both for your patience.

ABSTRACT

This research examined the physiological and performance effects of reducing exercise intensity and/or duration prior to a cycling test following a taper period. "Tapering" is a specialised training technique which is used to prepare athletes in the days preceding competition by reducing the intensity and/or duration of exercise, i.e. manipulating the quantity of rest and exercise, while still maintaining the adaptations developed through training. The endurance training programme which preceded a taper in two separate studies elicited significant ($p<0.05$) increases in all oxidative enzymes (77-178%), glycogen (35%) and protein (34%) content, maximal oxygen consumption (6-12%) and endurance capacity (60 min ergometry cycle test in study 1, 35%; a simulated 40 km time trial ride (40TT) in study 2, 11%). Following the common training programme, cyclists were randomly assigned to one of four taper groups in study 1 (CON= no training; 4D= a group that maintained exercise intensity for 4 days; 8D= a group that maintained intensity for 8 days; NOTAPER= continued training) and one of three different 7 day groups in study 2 (MI= maintained intensity but reduced exercise duration; MD= maintained duration but reduced exercise intensity; CRT= regular training).

After tapering in study 1, power output (Watts; W) at ventilation threshold was significantly increased in the 4D (27.4 W) and 8D (27 W) groups but decreased 22 W in NOTAPER. ß-hydroxyacyl CoA dehydrogenase enzyme activity (HOAD) was

significantly decreased in the CON. Muscle glycogen was increased ($p<0.05$) in the 4D, 8D, and CON groups, and these values were significantly higher than NOTAPER.

In study 2, power output at ventilation threshold was significantly increased in the MI group. The average improvement in performance time for each group on the 40TT (MI= 2:33 min; MD= 1:13 min; CRT= 0:14 min) was not significant. In single vastus lateralis muscle fibres, analysed by quantitative histochemistry, HOAD and CYTOX activity values were significantly increased in the MI group and were higher than those from the CRT. Muscle glycogen content was significantly increased after tapering in MI and MD. These results demonstrated that metabolic muscular adaptations and improvements on the laboratory cycling tests can occur following specific tapering strategies. The MI protocol (maintained intensity/reduced duration) elicited the greatest effect as reflected by the increased oxidative enzyme activity levels and muscle glycogen concentration.

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CHAPTER 1

GENERAL INTRODUCTION

General Introduction

"Tapering", is a specialised exercise training technique which is used to prepare athletes for competition in the days preceding an athletic event by reducing the intensity and/or duration of exercise, and thus manipulating the quantity of rest and exercise, while still maintaining the peripheral muscular and central cardiovascular adaptations developed through training. This reduction in training which provides more rest will then allow athletes to compete at their highest level of fitness.

Tapering is a complex process that includes both a physiological and a psychological component. Undoubtedly, psychological factors (Pyke et al. 1988) and factors affecting the central nervous system and hormonal influence and modulation play a role in affecting performance following training (Barron et al. 1985). As well, performance can be affected by subcellular alterations in the relevant muscle. Muscle damage, oedema and elevated serum enzyme levels are well known cellular alterations caused by strenuous endurance training (for review see: Armstrong 1986; Milne 1988) and have been shown to be detrimental to performance (Ogilvie et al. 1985). While rest can reverse these adverse physiological effects, extended periods of no training may result in a reduction of the training effect and so a taper is designed to reduce fatigue but maintain the adaptations of training.

Many of the principles of endurance training are based on scientific research. The correct utilisation of these principles of training in a systematic and organised manner can assist the athlete to achieve a superior level of performance. One such method used to organise a training programme is termed periodization (Matveyev 1965). It is a process of dividing an annual training calender into smaller, organised periods of training to make training more manageable and systematic so that peaking for a major competition can be more easily facilitated (Bompa 1983). Although the principle of tapering is not well understood, it is an important component in the periodization model.

The three main periods of training as outlined by Matveyev (1965) are prepatory, competitive and transition. Each period is further divided into smaller phases. Tapering, sometimes referred to as "unloading", is a phase which is part of the competitive period that follows many months of training preparation.

It has been postulated that to perform at the highest possible level of fitness and achieve a successful performance, the endurance athlete must alter the volume of training prior to an event (Bompa 1983; Costill 1986). Anecdotal reports suggest that a reduction in the intensity and duration of training can elicit a superior performance during competition. For example, faster performance times have been reported (Gallagher 1977; Meisel 1977), increases

in white muscle fibre strength have been claimed (Meisel 1977), and an improvement in skill and technique are documented (Gallagher 1977). It appears that added rest periods interspersed between training and performance allow the body to recover from the fatigue of daily training (Morton et al. 1990). The type and duration of recovery from training is as important as the exercise stress itself in the resulting adaptation (McCafferty and Horvath 1977). Therefore, some combination of exercise training and rest which would maintain the training effect but reduce fatigue may lead to a superior performance. However, the optimal combination of these physiological factors is unknown.

Several investigations have examined the effects of a complete cessation of training, i.e. "detraining", on selected physiological variables (Houston et al. 1979; Chi et al. 1983; Coyle et al. 1984, 1985, 1986; Russell et al. 1987). These studies have shown that selected central circulatory and peripheral musculature adaptations are lost at different rates but in a relatively short period of time, especially when considering the quantity and quality of training needed to acquire such adaptations. A decrease in the central cardiovascular parameters such as maximal oxygen consumption, cardiac output, stroke volume, arterio-venous oxygen difference, and heart rate responses to an absolute amount of exercise are lost at a slower rate than are the labile enzymes of energy metabolism in peripheral muscle

which have short half lives (Henriksson and Reitman 1977; Clausen 1977; Saltin and Rowell 1980; Klausen et al. 1981; Hoppeler et al. 1985; Allen 1989). Hence, tapering may have its most significant impact on the maintenance of energy metabolism in skeletal muscle.

Some researchers have altered training volume by manipulating exercise duration and frequency of training and have shown that selected physiological variables can be maintained for extended periods of time (Hickson et al. 1982; Hickson and Rosenkoetter 1981; Houmard et al. 1990). Although detraining and reduced training studies have illustrated the responses of central and peripheral muscle properties, they have not provided an answer to the questions pertaining to the physiological changes which may occur during a taper, or to the optimal combination of training intensity, duration and length of taper programme that will elicit the best performance.

The independent variables of intensity, duration and length of the taper have been arbitrarily altered with little scientific basis to support their relative effectiveness (Daland 1977; Gallagher 1977; Bompa 1983; Crozier 1987). Anecdotal evidence support maintaining the training intensity while reducing the duration of exercise during the 3-21 days prior to competition (Hogg and Montpetit 1982; Gallagher 1977; Costill 1986). However, if high intensity training is reduced for prolonged periods,

training adaptations are lost and performance is compromised (Banister and Calvert 1980; Montpetit 1982).

The present studies examined a number of selected physiological variables (i.e. muscle enzymes, glycogen, oxygen consumption, heart rate, respiratory exchange ratio) which could optimise both the physiological changes and subsequent performance following both training and tapering. They are presented in a paper format in two separate chapters. **Chapter 2** (study 1) examined metabolic muscular enzyme and glycogen concentration adaptations and performance changes during a ventilation threshold test following both a 4 and an 8 day taper. The duration of exercise training was reduced in a systematic manner, while the intensity of training was maintained throughout the taper. The independent variable was exercise duration. **Chapter 3** (study 2) investigated the effects of altering duration or intensity of exercise during a 7 day taper. In this study, adaptations in selected metabolic properties of single muscle fibres and performance changes during both a ventilation threshold test and a simulated 40 km time trial cycle ride were examined. Therefore, exercise duration and intensity were the independent variables. **Chapter 4** is a general discussion which summarises the results and discusses the applications of this research and its implication for exercise training. Future research opportunities are also discussed.

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CHAPTER 2

THE EFFECTS OF A REDUCED EXERCISE DURATION TAPER PROGRAMME ON PERFORMANCE AND MUSCLE ENZYMES OF ENDURANCE CYCLISTS

A version of this chapter has been submitted for publication.

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INTRODUCTION

One goal of an endurance training programme is to elicit cellular and systemic adaptations to allow the body to function at a higher level of performance. The goal of tapering, which is a specialised exercise training technique, is to reduce the training volume for a short period of time in the days preceding competition to reverse fatigue resulting from training without a loss of the training adaptations. In essence, tapering is a fine tuning of the physiological mechanisms which generally occurs between 3-21 days prior to competition (Costill et al. 1985; Costill 1986). Banister and Calvert (1980) have suggested that during tapering the recovery from the residual fatigue of months of training occurs, while maintaining both central cardiovascular and peripheral muscular adaptations. Although tapering is widely practised by many athletes, there is limited research which has addressed the physiological mechanisms underlying the taper process (for review see Neufer 1989).

There are several studies of successful tapers reported in the literature, although each have inherent problems. For example, without a control group, Costill et al. (1985) demonstrated that swim performance was significantly improved from the swimmers previous best times during the year following a 2 wk taper consisting of interval training which was gradually reduced in total distance swum each day

from 7,500 (day 1) to 3,500 yd (day 13). Muscular arm power and swim performance also improved ($p<0.05$) but no changes were found in any of the blood parameters (pH, lactate, HCO_3^-) of the swimmers. Pyke et al. (1988) also had no control group but illustrated that improvements on a simulated pursuit cycling race can occur after a 7-10 day taper. While these two studies are helpful, the do not contribute to understanding the mechanisms involved in tapering. For example, muscle samples were not taken in either study and therefore it is not possible to determine if muscular cellular mechanisms contributed to the improved performance. In contrast, Montpetit (1982) in a limited study of muscle characteristics did find that citrate synthase activity was decreased significantly when exercise duration was reduced during a 2 wk taper in swimmers. Although the average performance times during the 1500 m swim were not significantly slower (14 s), he concluded that adaptations in enzyme activity can be lost if the training stimulus is not adequate. A study such as this starts to provide information which can lead to a understanding of the mechanisms by which tapering is controlled. Subsequently, better tapering methods can be established.

Currently, it is not possible to describe the physiological consequences of a taper or describe the possible mechanism(s) underlying a change in performance. However, it would seem reasonable to suggest that skeletal

muscle variables such as enzyme activity, glycogen and protein concentration changes may be important factors that are altered during tapering, as they have been shown to be altered significantly with short periods of detraining (Henriksson and Reitman 1977; Houston et al 1979). Therefore, the purpose of this study was to measure selected metabolic cellular changes in the vastus lateralis muscle of trained cyclists following a taper of either 4 days or 8 days in which exercise duration was progressively reduced with intensity kept constant. These physiological properties were studied with respect to their influence on ergometry cycling during a laboratory test, and to act as guidelines for future experimentation. A second, more practical purpose, was to determine whether a 4 day or an 8 day taper was more effective in enhancing performance.

METHODS

Subjects:

Twenty-five club level cyclists and triathletes (7 females, 18 males) signed informed consent forms (Appendix B), and volunteered to participate. This form was approved by the Department of Physical Education and Sport Studies Ethics Review Committee at the University of Alberta. The nature of the study, potential risks involved and the exercise training, testing and taper protocols were

described in detail. Subject physical characteristics for mean ($\bar{X}$ ± SD) age, height, weight and maximal oxygen consumption ($\dot{V}O_2max$) were 25 ± 6 yr, 178.5 ± 8.6 cm, 67.0 ± 8.8 kg, and 3.91 ± 0.48 $l \cdot min^{-1}$, respectively.

Testing:

The experimental design for the testing, training and taper programme is illustrated in Figure 2.1. $\dot{V}O_2max$ was determined on a Monark cycle ergometer using a continuous incremental protocol lasting between 10-14 min. Following a 2 min warm-up at 88 watts (W), power output (PO) was increased 44 W each min until the subject reached voluntary exhaustion as indicated by a plateau in $\dot{V}O_2$ with an increase in workload (Thoden 1991). Heart rate (HR) was monitored each minute using portable HR monitors (PE 3000 Sport Tester, Polar Electro, Finland).

A 60 min submaximal endurance test (ET_{60}) was used to establish changes in endurance capacity, as reflected by the ability of the cyclists to exercise at a higher power output after training. During the 60 min ride, PO was recorded (and adjusted if necessary) each minute to maintain the heart rate at 70% of maximum, which was established during the $\dot{V}O_2max$ test.

A ventilation threshold (VT) test was used to monitor the respiratory changes before and after tapering. The test was started by having the cyclists warm-up at 88 W, followed

by a 3 min continuous protocol of 44 W increments for the initial 12 min. Thereafter, workload was increased by 22 W each minute until threshold was detected by a disproportional increase in ventilation ($\dot{V}_E$) versus $\dot{V}O_2$ (Jones and Ehrsam 1982; Neary et al. 1985). A calibrated Beckman Metabolic Measurement Cart (Sensor Medics, California, USA) was used to collect and analyse expired gases at 30 s intervals.

Before training, the $\dot{V}O_2$max test was performed on Day 1; the muscle biopsy and ET_{60} were then performed on Day 2. During the last week of training, the ET_{60} was performed on the Wednesday. The $\dot{V}O_2$max test was performed on the Friday, followed by a muscle biopsy and VT test on Saturday. The taper started on Sunday (Fig. 2.1).

Training Programme:

The cyclists underwent a progressive overload training programme by exercising for 45 min·session^{-1} at an intensity below VT (80% HRmax) for 5 d·wk^{-1} during the initial 2 wk. Exercise intensity and duration were increased for the cyclists so that training was at or slightly above VT (90% HRmax) for 60 min·session^{-1} for the remaining 6 wk. The cyclists were instructed to try and maintain exercise training intensity at the highest possible intensity for each session. Training was performed by having all cyclists ride their own bicycle mounted on magnetic turbo trainers

(Munouri, Japan). HR was monitored every session using portable HR monitors, and maintained within a HR target zone which was established during the $\dot{V}O_2$max test.

Taper Programme:

Following training, all subjects were randomly assigned to 1 of 4 different taper groups: **CON** (no training, n=6), **4D** (a group that tapered for 4 days while maintaining training intensity but reducing exercise duration, n=7), **8D** (a group that tapered for 8 days while maintaining training intensity but reducing exercise duration, n=6), **NOTAPER** (continued training, n=6) (see Fig. 2.1 for details). The NOTAPER group served as an additional control group by continuing to train during the taper. The independent variable during tapering was exercise duration, with the intensity of training maintained at or above VT (90% HRmax). All training and taper sessions were supervised to ensure compliance with all aspects of the programme. During the taper the subjects performed only their prescribed exercise training as outlined in Fig. 2.1

Biochemical Analyses:

Muscle tissue samples (30-100 mg) from the lateral portion of the quadriceps femoris muscle (vastus lateralis) were obtained under local anaesthetic (1% xylocaine) using

the biopsy needle technique of Bergstom (1962) adapted for suction (Evans et al. 1982). Tissue samples were rapidly frozen (< 1 min) in liquid nitrogen and stored in individual polypropylene capsules at -80°C until analysed.

The tissue samples were homogenized (7x dilution) by hand (Schantz 1986) using glass tissue grinders in 50 mM Tris-HCl buffer containing 1 mM EDTA and 0.1% Triton X-100; pH 7.6 (Suarez et al. 1986). Homogenate samples were then sonicated (Biosonik, Bronwill Scientific, N.Y., USA) 3 times for 5 s at 30 s intervals at 25% of maximal probe intensity to ensure membrane disruption necessary for the enzyme analysis. Homogenate samples were then centrifuged (1000x g for 10 min; 4°C) and the supernatant collected for the analysis of the following enzymes:

1. Carnitine palmityltransferase (EC 2.3.1.23; CPT) (Suarez et al. 1986; CPT activity represents the transport of free fatty acids across the mitochondrial membrane). Ten µl of tissue supernatant was added to a stock solution of 1.1 mM EDTA, 0.1 mM 5,5'-dithio-bis (2-nitrobenzoic acid) (DTNB), and 116 mM Tris-HCl buffer (pH 8.0). Subsequently, palmityl CoA (0.032 mM) was added and the non-specific activity recorded for 20 s. L-carnitine (2.2 mM) was used to initiate the reaction, which was assayed spectrophotometrically at 412 nm. The difference in activity during the two conditions estimated the total enzymatic activity. All enzyme activity was calculated in

this manner.

2. Citrate synthase (EC 4.1.3.7; CS) (Lowry et al. 1978; CS is a Krebs cycle enzyme which was used to represent the state of cellular respiration). Five µl of tissue supernatant was added to a stock solution of 0.1 mM DTNB, 0.35 mM acetyl CoA, and 50 mM Tris-HCl buffer (pH 8.1) 20 s before starting the reaction with oxaloacetate (0.083 mM). Spectrophotometric analysis was performed at 412 nm.

3. Lactate dehydrogenase (EC 1.1.127; LDH) (Suarez et al. 1986; LDH is a glycolytic marker, and was used to examine the influence of endurance training on anaerobic metabolism). Five µl (10x dilution) of tissue supernatant was added to a stock solution of 0.15 mM NADH, 5 mM DL-dithiothreitol (DTT), and 50 mM Imidazole buffer (pH 7.6). Subsequently (20 s), Na-pyruvate (4 mM) was used to start the reaction. The assay was measured spectophotometrically at 340 nm.

4. ß-Hydroxyacyl CoA dehydrogenase (EC 1.1.1.35; HOAD) (Suarez et al. 1986; HOAD was used to represent the ß-oxidation energy production pathway). Five µl of tissue supernatant was added to a stock solution of 1 mM EDTA, 5 mM DTT, 0.15 mM NADH, and 50 mM Imidazole-HCl buffer (pH 7.4). Acetoacetyl CoA (0.1 mM) was used to initiate the reaction spectrophotometrically at 340 nm.

5. Cytochrome oxidase (EC 1.9.3.1; CYTOX) (Davies et al. 1981; CYTOX is an electron transport chain enzyme which was

used to reflect the respiratory capacity of the muscle). Oxygen consumption was determined polargraphically using a Clark oxygen electrode (Yellow Springs Instrument Model 53, Colorado, USA). The O_2 buffer medium (950 μl) was allowed to equilibrate (37°C) for 1 minute before 10 μl of each of Na-ascorbate (3.5 mM), N,N,N,N'-tetramethyl-p-phenylenediamine dihydrochloride salt (0.5 mM) and cytochrome c (2 μM) were added. Twenty μl of tissue supernatant containing the uncoupled mitochondria was then added to determine state 3 respiration (Chance and Williams 1955).

Muscle glycogen content was determined using the amyloglucosidase assay technique (Bergmeyer 1974). The protein concentration of the tissue homogenate was determined according to Lowry et al. (1951) using bovine serum albumin as a standard. All spectrophotometric assays were measured at 25°C on a final reaction volume of 600 μl using a Pye-Unicam PU8800 UV/Vis spectrophotometer with a linear chart recorder and a water jacketed cuvette holder.

Statistical Analysis:

A two-way analysis of variance (ANOVA) model with one factor (time) repeated, was utilized for analysis. A delta value (post- minus pre-taper) was also generated for comparison between groups and analysed using the ANOVA model (Milliken & Salmson 1984). A Least Significant Differences

Multiple Comparisons procedure was used to locate simple main effects if any main effect was found statistically significant at an alpha level of $p<0.05$. A Pearson Product-Moment Correlation Coefficient (r) was used to examine the relationship between selected variables. This statistical protocol was approved by the Department of Statistics at the University of Alberta.

RESULTS

Following the endurance training programme there was a significant increase in the 60 min submaximal endurance cycle test (35%) and $\dot{V}O_2max$ (6%), and in the activities of CPT, HOAD, CS, CYTOX (77-178%), and glycogen (35%) and protein (34%) concentrations (Table 2.1). Post-training CYTOX activity also correlated significantly with power output measured during the 60 min endurance cycle test ($r=0.695$, $p<0.05$; Appendix C.1).

The cycling test results after tapering illustrated that power output at the ventilation threshold was significantly increased in the 4D (27.4 W) and 8D (27 W) groups, but decreased ($p<0.05$) in the NOTAPER (22 W). No change was found in the CON (Table 2.2). The respiratory exchange ratio (RER) at an absolute PO of approximately 175 W was significantly increased in the CON group and decreased in the 4D, 8D and NOTAPER groups (Table 2.2).

A post hoc analysis was also performed to examine the

relationship between the post-training 60 min endurance cycle test and the power output at ventilation threshold and showed a significant positive correlation ($r=0.80$, $p<0.05$; Appendix C.2).

The response of the muscle variables during the taper for each group are illustrated in Table 2.3 and Figs. 2.2 to 2.6 and in Appendix C.3. Muscle glycogen levels were increased ($p<0.05$) after the taper in the 4D, 8D and CON groups which were significantly higher than NOTAPER at both the 4 and 8 day testing periods (Fig. 2.2). In the CON group, HOAD activity was significantly decreased (13.8 ± 0.6 to 11.0 ± 0.8 $\mu mol \cdot g^{-1} \cdot min^{-1}$, wet weight; w.w.). No other enzyme activities were significantly altered during the taper within their respective group. However, inter-group differences in enzyme activity did occur. CPT activity was significantly higher in the 8D (0.38 $\mu mol \cdot g^{-1} \cdot min^{-1}$; w.w) versus CON (0.24 $\mu mol \cdot g^{-1} \cdot min^{-1}$; w.w), and CYTOX activity was significantly higher in the 4D and 8D versus CON, and 8D versus 4D (Table 2.3 and Appendix C.3). Despite these inter-group differences, no clear pattern is apparent with respect to the type of activity performed during the taper on the muscle properties.

DISCUSSION

The present study examined the effects of varying the taper length (i.e. 4 or 8 days) while reducing exercise duration but maintaining intensity in a taper programme, on the power output generated during a ventilatory threshold cycling test and on selected muscle properties. The cycling test results revealed that the taper significantly increased power output at the ventilation threshold in the 4D and 8D groups, but was decreased in the NOTAPER with no change in the CON. No differences were found between the 4D and 8D groups.

Other studies that have examined tapering at the performance level only, (i.e. did not analyse muscle tissue), have also illustrated that improvements can occur following a taper programme. Pyke et al. (1988) found significant gains in power output during a simulated pursuit race in a group of national team cyclists following a 7-10 day taper when exercise intensity was maintained but duration was reduced. Costill et al. (1985) also found significant increases in muscle power measured in (24.6%) and out (17.7%) of the water in a group of collegiate swimmers. In addition, swim performance times were faster by 3.1% ($p<0.05$). Therefore, a significant increase in PO at a given exercise intensity, as demonstrated by the taper groups in this study, would suggest that endurance performance would also have improved after tapering for

either 4 or 8 days when intensity is maintained while exercise duration is progressively reduced.

It is well documented that endurance performance is correlated to muscle glycogen levels (for review see Conlee 1987). Many studies (Hultman 1967; Bergstrom et al. 1967; Hultman et al. 1971; Hermansen et al. 1967) have shown that glycogen levels have a determining effect on exercise duration during a high intensity effort. This too was consistent with the cycling test results during the taper in the current study. For example, the NOTAPER group showed a significant decrease in muscle glycogen after 4 days of daily cycling during the taper period, and continued to remain reduced when tested after 8 days (Fig 2.2). A reduced muscle glycogen concentration and power output at VT could limit endurance performance. Although a specific cycling performance test was not performed before and after the taper to substantiate this, the increase in power output at VT and muscle glycogen in the 4D and 8D groups suggest that cycling performance would also be increased. This assumption is based on the finding that a significant correlation ($r=0.80$, $p<0.05$) was found between the PO maintained during the post-training 60 min endurance test and the PO generated at VT during the pre-taper testing (Appendix C.2). Also, it is well documented that the ventilation threshold is a good predictor of running (Peronnet et al. 1987) and cycling (McLellan and Skinner

1985) performance. These data also substantiate that a continued high volume (duration and intensity) of training prior to competition (i.e. NOTAPER protocol) should not be used where aerobic capacity is the predominant energy production system.

No differences were observed between the 4D and 8D taper protocols, as reflected by the increase in PO at VT, suggesting that either the 4 or 8 day protocol could be used to elicit improvements in performance after tapering. Also, the lower RER values in both groups after tapering suggests that more fats contributed to the energy production. However, in the CON group, RER was significantly increased. This would be in agreement with the muscle enzyme results which showed that when exercise was not performed for 4 days (i.e. CON group), ß-hydroxyacyl CoA dehydrogenase activity was significantly decreased. This reduction in HOAD activity could result in a reduced free fatty acid oxidation in the mitochondria and lower energy production via ß-oxidation. This may infer that glycogenolysis will occur earlier during submaximal exercise if a selected power output is to be maintained (Holloszy 1976; Holloszy and Coyle 1984). Again, this inference is supported by the finding that the CON group exhibited an increase ($p<0.05$) in RER during submaximal exercise at the same absolute power output. However, in the 4D, 8D and NOTAPER groups, CPT and HOAD activity were maintained, suggesting that some level of

exercise training is necessary during the taper.

It has been suggested that CYTOX and cytochrome c may be the single best markers used to detect alterations in mitochondrial enzyme activity (Davies et al. 1981; Soussi et al. 1989). Davies et al. (1981) found a high positive correlation ($r=0.92$) between endurance performance time and CYTOX activity in rodents. The present results are also consistent with those of Davies et al. (1981) and demonstrate a significant positive relationship between CYTOX activity and the power output generated during the 60 min submaximal endurance cycle test ($r=0.695$, $p<0.05$) following training (Appendix C.1). The post-taper CYTOX activity in the 4D and 8D groups were significantly higher than the CON. This difference could alter energy production (Newsholme and Start 1973; Gollnick and Saltin 1982) and potentially limit endurance performance. Also, the significant delta values (i.e. post- minus pre-taper) in CYTOX activity (Appendix C.3) between the taper and CON groups suggest that these protocols were effective in maintaining enzyme activity. The reduction in oxidative enzyme activity (HOAD) in the CON is consistent with the detraining literature (Chi et al. 1983; Coyle et al. 1984).

Several studies have shown that enzyme adaptations are related to the intensity and duration of training. Dudley et al. (1982) and Schantz (1986) showed that to achieve an adaptive response, less daily activity was required if

intensity was increased. An exercise intensity of 80-90% $\dot{V}O_2max$ was optimal for the development of oxidative enzyme activity, and when the influence of exercise duration was considered, 30 minutes of training was adequate to elicit significant cellular changes. On average, exercise training during the taper period (i.e. 4D and 8D) was approximately 30 $min \cdot d^{-1}$ with intensity maintained at 80-85% $\dot{V}O_2max$.

The lack of statistically significant changes in enzyme activity before the taper compared with after may be related to several limitations. These include: small group sizes and a mixture of sexes. A small sample size would have reduced the statistical power (Cohen 1977; Sharp and Gahlinger 1988), while the mixture of sexes in each group (Hickson and Rosenkoetter 1981) may increase the variance, making it more difficult for group means to reach statistical significance (Hinkle et al. 1979). This has also been confirmed by Costill et al. (1979) who showed that muscle CPT activity of females adapt to a lesser degree than that of males under similar endurance training programmes.

The cellular mechanism(s) underlying the cycling test improvements and muscle glycogen increase during the taper are still unknown. In the study by Costill et al. (1985), it was hypothesised that an increase in maximal tension development through changes in the contractile mechanisms and/or neural control of fibre recruitment may be a possibility. This cannot be refuted as muscular strength

and power were not measured in the present study. However, the present results showed that oxidative enzyme activity can be decreased during short periods of inactivity. Booth and Holloszy (1977) have suggested that protein synthesis appears to be responsible for the alteration in enzymatic activity following training, and that the stimulus for cytochrome c production ceases abruptly at the beginning of detraining. Furthermore, Holloszy and Booth (1976) stated that mitochondrial enzyme activity will continue to rise as long as the intensity of the exercise stimulus is increased.

In summary, this research illustrated three key points regarding the physiological effects of tapering when exercise duration and length of the taper were altered. First, when the cycling test results and muscle glycogen are considered, the 4D, 8D and CON protocols appeared to be better than the continued training (NOTAPER) group. However, if only the enzyme data were evaluated, then the 4D, 8D, and NOTAPER groups would have been better than the CON which performed no exercise (i.e. decrease in HOAD and decreasing trend of CPT activity). Second, the 4D and 8D programmes were better than the NOTAPER protocol because of the reduction in power output at VT and muscle glycogen in the NOTAPER group. Third, no differences were found between the 4D and 8D protocols. Therefore, with these results in mind, and due to the fact that the underlying mechanisms are still unresolved, further investigation on tapering is warranted.

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Table 2.1. Skeletal muscle adaptations and changes in performance following the 8 wk endurance training programme.

	Muscle Indices							Performance Indices	
	CPT	CS	HOAD	CYTOX	LDH	GLY	Protein	ET_{60}	$\dot{V}O_2max$
Before	0.16	14.26	6.81	1.86	225.0	94.0	7.57	143.6	3.69
	0.02	0.89	0.42	0.21	9.6	4.1	0.43	6.8	0.12
After*	0.34	25.22	12.07	5.16	221.1	126.2	10.21	189.2	3.91
	0.12	0.76	0.44	0.15	6.6	2.9	0.28	7.6	0.22

Values are Mean ± SE

Enzyme activity= μmol g^{-1} min^{-1}, w.w.; CYTOX= natoms $\dot{O}_2$ min^{-1}, w.w.; glycogen concentration (GLY)= μmol g^{-1}, w.w.; protein= mg ml^{-1}; 60 min endurance cycle test (ET_{60})= Watts; $\dot{V}O_2max$= l min^{-1}.

* All values were significantly different ($p<0.05$) after training except LDH enzyme activity.

Table 2.2. Power output (PO; Watts) at ventilation threshold (VT) and respiratory exchange ratio at 175 W (RER_{175}) before and after the taper for each group (4D= 4 day; 8D= 8 day; CON= control; NOTAPER= continued training).

	4D	8D	CON	NOTAPER
Before				
PO at VT	228.0[a]	226.6[c]	240.6	254.5[f]
	5.2	7.7	13.3	12.1
RER_{175}	0.99[b]	0.99[d]	0.92[e]	1.00[g]
	0.01	0.01	0.01	0.01
After				
PO at VT	255.4[a]	253.6[c]	237.8	232.5[f]
	9.5	11.2	11.1	10.3
RER_{175}	0.96[b]	0.96[d]	0.96[e]	0.97[g]
	0.01	0.01	0.01	0.01

Values are Mean ± SE

Paired letters (a,a; b,b;.. g,g) indicate significant difference at $p<0.05$.

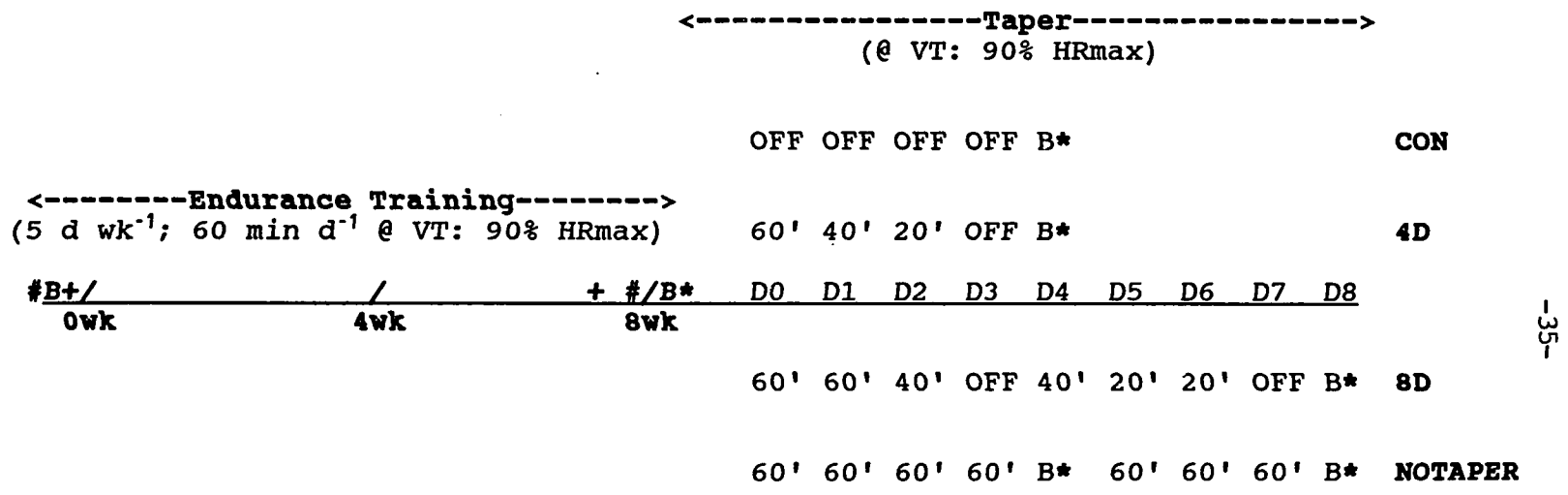
Table 2.3. Muscle enzyme activity ($\mu mol \cdot g^{-1} \cdot min^{-1}$; w.w.), glycogen ($\mu mol \cdot g^{-1}$; w.w.) and protein ($mg \cdot ml^{-1}$) concentration before and after tapering for each group. CYTOX is expressed in natoms $O_2 \cdot min^{-1}$; w.w. (supernatant).

Group/ Variable	Taper Period Before		After		Percent change
4D					
CPT	0.36	(0.02)	0.37	(0.01)	+2.8
CS	26.60	(0.36)[a]	27.97	(0.76)	+5.2
HOAD	10.03	(0.50)[bc]	10.83	(0.74)	+8.0
CYTOX	5.26	(0.18)	5.57	(0.11)[kl]	+5.9
LDH	230.65	(7.62)	218.81	(7.54)	-5.4
PROTEIN	10.71	(0.20)	10.61	(0.15)	-0.6
GLYCOGEN	127.77	(3.10)[d]	150.21	(6.60)[dm]	+17.6
8D					
CPT	0.35	(0.01)	0.38	(0.02)[n]	+7.9
CS	26.02	(0.48)	26.18	(0.58)	+0.6
HOAD	12.86	(0.45)[c]	12.86	(0.68)	-
CYTOX	6.09	(0.16)[ef]	6.66	(0.23)[kop]	+9.4
LDH	231.41	(5.71)	232.88	(11.84)	+0.6
PROTEIN	10.56	(0.46)	10.77	(0.47)	+2.0
GLYCOGEN	127.04	(2.00)[g]	159.67	(4.20)[gq]	+25.7
CON					
CPT	0.30	(0.03)	0.24	(0.03)[n]	-26.0
CS	25.27	(1.07)	24.08	(1.38)	-5.0
HOAD	13.77	(0.63)[bh]	11.01	(0.85)[h]	-25.0
CYTOX	4.72	(0.16)[e]	4.53	(0.08)[lo]	-4.2
LDH	200.10	(7.71)	207.64	(9.71)	+3.8
PROTEIN	9.84	(0.09)	9.92	(0.11)	+0.8
GLYCOGEN	128.20	(2.40)[i]	143.96	(4.30)[ir]	+12.3
NOTAPER					
CPT	0.34	(0.01)	0.34	(0.01)	-
CS	21.13	(0.55)[a]	23.38	(1.12)	+10.6
HOAD	11.95	(0.19)	12.50	(0.17)	+4.6
CYTOX	4.55	(0.09)[f]	4.90	(0.06)[p]	+7.7
LDH	222.25	(5.51)	212.28	(3.76)	-4.7
PROTEIN	9.66	(0.38)	10.65	(0.61)	+10.2
GLYCOGEN	121.51	(3.90)[j]	103.47	(2.50)[jmqr]	-17.4

Values are Mean ± (SE)

Paired letters (a,a; b,b;.. r,r) indicate significant differences at $p<0.05$.

Figure 2.1. Experimental design for the exercise testing, training and tapering programme.



#= $\dot{V}O_2$max test

*= Ventilation threshold (VT) test

B= Muscle biopsy sample

+= 60 min endurance cycle test (ET_{60})

Figure 2.2: Muscle glycogen concentration ($\mu mol \cdot g^{-1}$; w.w.) for each group before (DAY 0) and after (DAY 4, 8) the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.

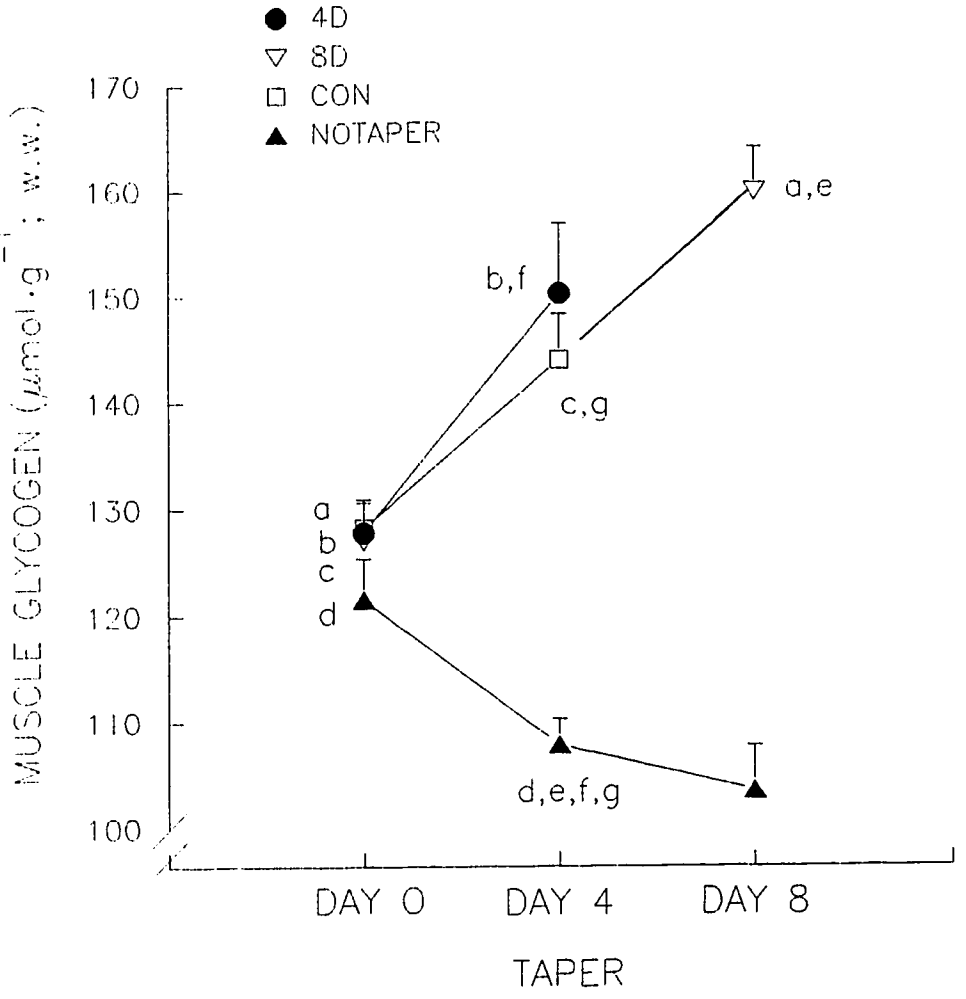


Figure 2.3: β-Hydroxyacyl CoA dehydrogenase activity (HOAD, $\mu mol \cdot g^{-1} \cdot min^{-1}$; w.w.) before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.

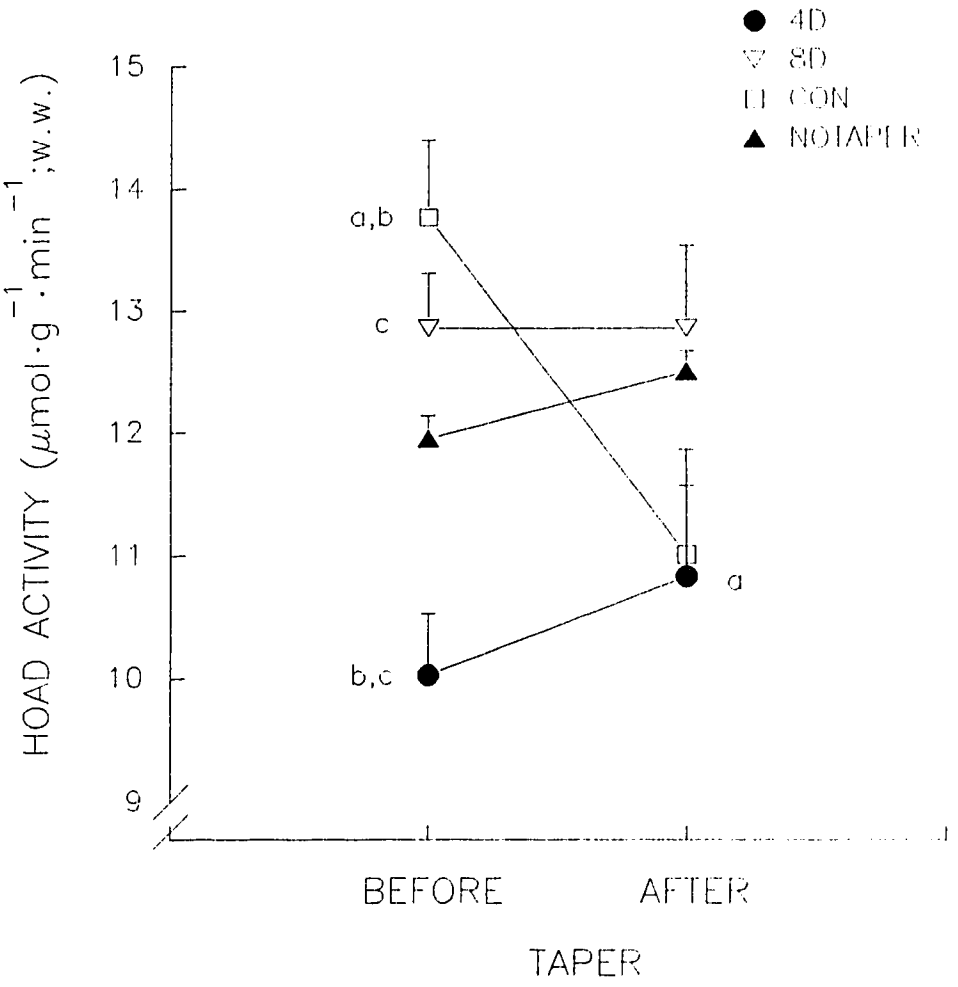


Figure 2.4: Carnitine palmityltransferase activity (CPT, μmol·g^{-1}·min^{-1}; w.w.) before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.

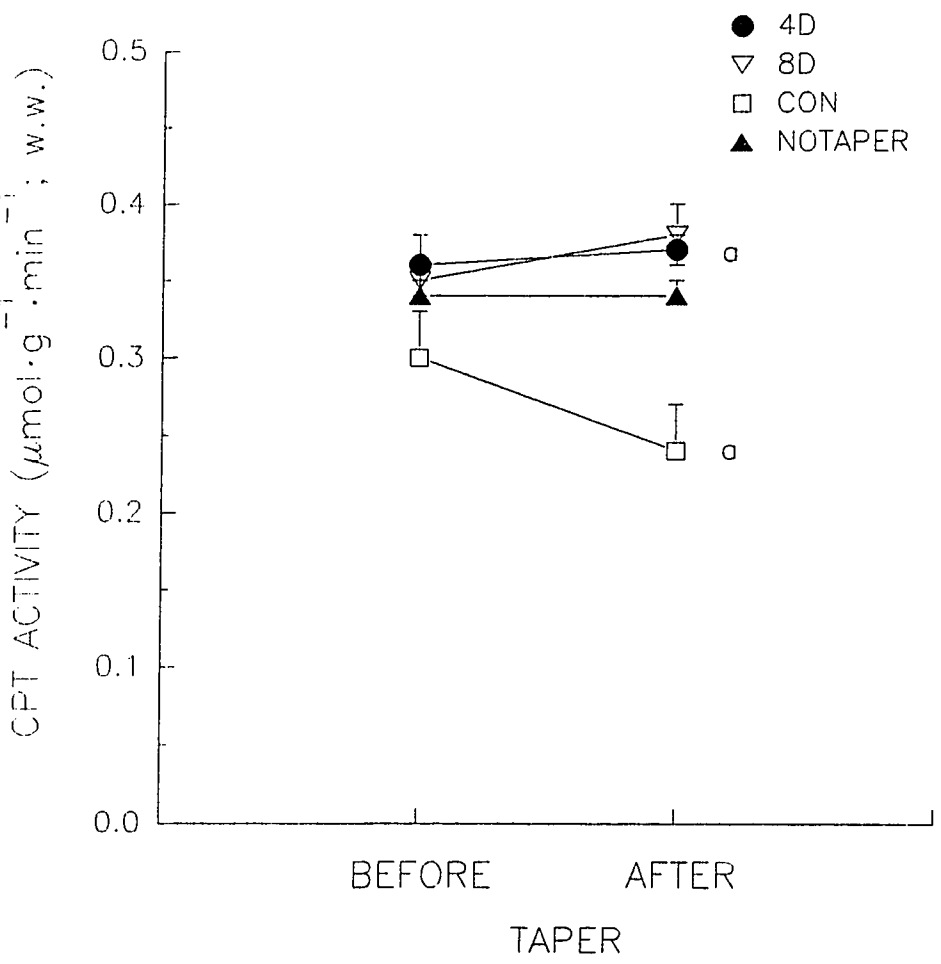


Figure 2.5: Cytochrome oxidase activity (CYTOX, natoms $O_2 \cdot min^{-1}$; w.w.) before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.

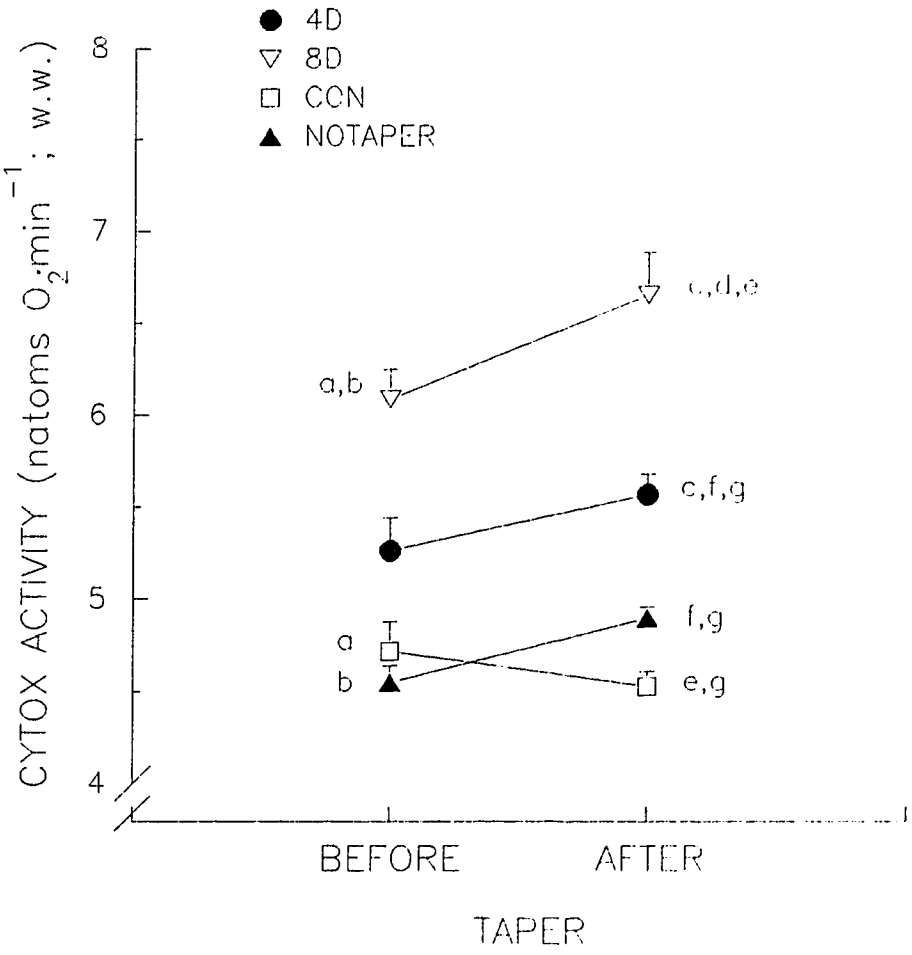
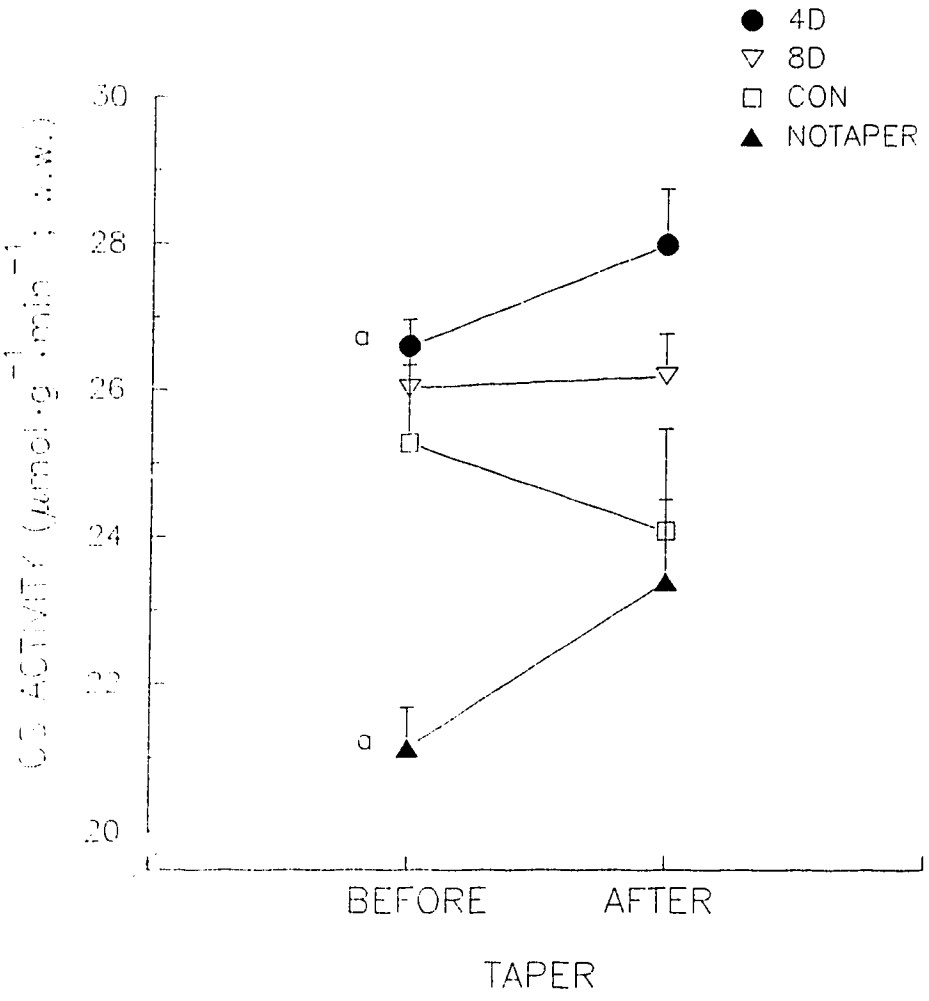


Figure 2.6: Citrate synthase activity (CS, $\mu mol \cdot g^{-1} \cdot min^{-1}$; w.w.) before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.



CHAPTER 3

THE EFFECTS OF REDUCED EXERCISE DURATION AND INTENSITY DURING TAPERING ON PERFORMANCE AND MUSCLE PROPERTIES OF ENDURANCE CYCLISTS

A version of this chapter was presented at the Canadian Association of Sport Sciences conference in Kingston, Ontario, in October 1991, and was also awarded the Canadian Association of Sport Sciences Young Investigator Award for 1991.

INTRODUCTION

A variety of taper protocols have been used to elicit improvements in performance but no study has defined what may be an optimal taper protocol required for maximal performance in any sport. For example, Costill et al. (1985) reduced the weekly training duration by 62% during interval swim training, and in a cycling study by Pyke et al. (1988), continuous and interval cycle training were reduced individually for each cyclist. Study 1 of this thesis showed that no differences were found between the length of tapering (i.e. 4D and 8D protocols) for any of the cycling variables tested when exercise intensity was maintained while exercise duration was reduced. Therefore, different combinations in the reduction of training volume (i.e. intensity and duration) have been used and all have elicited improvements in performance but more details are required to determine the optimal taper protocol. Hence, further studies are needed to ascertain the effects of altering exercise intensity and duration on performance and on selected physiological variables during tapering.

To quantify adequately the physiological changes in the muscle properties occurring during a taper, it is important that the instruments for measuring are sufficiently sensitive to detect the metabolic adaptations, and that the testing protocols are specific to the exercise task. The analysis of muscle tissue at the single fibre level may be a

sufficiently sensitive technique to detect the metabolic adaptations during tapering. Skeletal muscle has been analysed at the single fibre level using a computerised image analysis system, and this technique has been shown to be reliable and valid in determining the metabolic enzymatic profile of rodents and cats (Martin et al. 1985; Martin et al. 1988). Thus, skeletal muscle in the present study was analysed using this technique. In addition, a reliable, simulated 40 km time trial bicycle ride (Neary et al. 1990), was used to monitor changes in performance in endurance cyclists. Therefore, to examine the effects of alternate taper protocols on specific cycling performance and on selected muscle properties, training intensity and duration were manipulated during a 7 day taper. A 7 day taper was selected because this is a common training period (micro-cycle) used by athletes.

METHODS

Subjects:

Thirty-three male club-level cyclists and triathletes volunteered to participate in the training and taper programme. Signed informed consent (Appendix B) was obtained after describing, in detail, the nature of the study, the potential risks involved and the exercise testing and training procedures. Five subjects discontinued the

project due to injury or personal circumstances. The cyclists' (n=28) physical characteristics for mean ($\bar{X}$ ± SD) age, height, weight and maximal oxygen consumption were 27 ± 4 yr, 181.2 ± 5.5 cm, 75.3 ± 7.7 kg, and 3.98 ± 0.39 $l \cdot min^{-1}$, respectively, before training.

Testing:

The experimental design for the testing, training and taper programme is illustrated in Figure 3.1. A combined ventilation threshold (VT) and maximal oxygen consumption ($\dot{V}O_2max$) test was performed on a Monark cycle ergometer to measure aerobic fitness before and after training and tapering. The VT/$\dot{V}O_2max$ test protocol included a 2 min warm-up at 88 Watts (W), followed by 2 min incremental steps of 44 W for the initial 8 min. Thereafter, workload was increased by 22 W in order to detect VT ($\dot{V}_E$ versus $\dot{V}O_2$; Jones and Ehrsam 1982; Neary et al. 1985) and $\dot{V}O_2max$ (a plateau in $\dot{V}O_2$; Thoden 1991). This test lasted between 14-18 min. Heart rate (HR) was monitored every minute using portable HR monitors (PE 3000 Sport Tester, Polar Electro, Finland).

A calibrated Beckman Metabolic Measurement Cart (Sensor Medics, California, USA) was used to collect and analyse expired gases for 30 s intervals during the initial assessment at the beginning of training. Before and after tapering, a Horizon or 2900 gas analysis system (Sensor

Medics, California, USA) was used to detect respiratory changes every 15 s. Each cyclist was tested on the same analysis system before and after tapering. The reliability between the Horizon and 2900 was r=0.99 (Appendix D.1).

A simulated 40 km time trial performance ride (40TT) was used to monitor cycling performance (Neary et al. 1990). The 40TT was performed on a set of wind-loaded cycling rollers, fitted with a stabilizing bar attached to the handle bars of the cyclists' bicycle. This provided a safety feature during the high intensity maximal effort. The rollers were mechanically linked via a pulley and cable mechanism to an oedometer which recorded the distance cycled. This device was calibrated for distance by measuring the circumference of the rollers and pulley system. A computerised bicycle cateye oedometer was also used to ensure that the distance cycled was consistent before and after the taper. During the 40TT test ride, which was performed in a temperature regulated chamber at 16-17°C, time (min), $\dot{V}_E$ ($l \cdot min^{-1}$), $\dot{V}O_2$ ($l \cdot min^{-1}$), HR (bpm) and respiratory exchange ratio (RER) were recorded for 3 min continuously at each 10 km interval.

Training Programme:

The cyclists underwent a progressive overload training programme by exercising for 45 $min \cdot session^{-1}$ just below VT (80% HRmax) 4 $d \cdot wk^{-1}$ (Monday, Tuesday, Thursday, Friday)

during the initial 2 wk. Intensity and duration of exercise were increased so that training was at or above VT (90% HRmax) for 60 min·session^{-1} for the remaining 5 wk (Appendix D.2). Training was performed by having all cyclists ride their own bicycles mounted on magnetic turbo trainers or cycling rollers. HR was monitored every session using portable HR monitors.

Taper Programme:

Following training, all cyclists were randomly assigned to 1 of 3 different taper groups: **CRT** (control group, regular weekly training; n=8), **MD** (a group that maintained exercise duration by cycling for 60 min·session^{-1} but reduced intensity; n=9), **MI** (a group that maintained exercise intensity at 90% HRmax but reduced duration; n=11). During the 7 day taper the subjects performed only the prescribed exercise training (see Fig. 3.1 for details). All training and taper sessions were supervised to ensure compliance with all aspects of the programme.

All cyclists consented to muscle biopsies being taken from the vastus lateralis muscle under local anaesthesia (1% xylocaine). The biopsy needle technique of Bergstrom (1962), adapted for suction (Evans et al. 1982), was performed before and after the taper. Tissue samples were quickly oriented to provide a cross-section of the muscle fibres when sectioned with a cryostat (see below), mounted

on cork and rapidly frozen in isopentane cooled in liquid nitrogen. This procedure took about 1-1½ min. The tissue was then stored at -80°C until analysed.

Quantitative Histochemical Analyses:

A computerised image analysis system was used to determine the activities of myofibrillar adenosine triphosphatase (mATP), ß-hydroxyacyl CoA dehydrogenase (HOAD), cytochrome oxidase (CYTOX), succinate dehydrogenase (SDH), and α-glycerolphosphate dehydrogenase (GPD) in individual fibres. In using this system for the measurement of enzyme activity, the rate of product formation in the tissue cross-sections is directly proportional to changes in enzyme concentration at non-limiting substrate levels. A detailed description of the hardware and software components and its operation have been described elsewhere (Castlemen et al. 1984; Martin et al. 1985; Martin et al. 1988).

Frozen tissue cross-sections (6μm thick) were cut at -20°C in a cryostat. For each enzyme assay, 5 cross-sections were cut in succession of which 3 sections were incubated in a medium containing substrate, and 2 sections incubated in a medium that did not contain substrate and served as blanks. In each of the 3 tissue cross-sections containing the reaction product, the same individual fibres were outlined ($\bar{X}$=47 fibres) and their optical density (O.D.) was measured on each fibre. The enzymatic rate for each assay was

determined by dividing the measured O.D. of the reaction product by the time of the reaction ($O.D.\cdot min^{-1}$). The O.D. of the blank tissue was performed in the same manner.

Serial sections were prepared for the determination of enzymatic activity in the following manner:

1. Myofibrillar adenosine triphosphatase (mATP) (Bell et al., in press). Reaction medium: 9.7 ml of 100 mM 2-amino-2-methyl-1-propanol, 10 μM sodium azide (NaN_3) and 80 mg $CaCl_2 \cdot H_2O$; 100 μl each of Calmidazolium (sarcoplasmic reticulum ATPase inhibitor; 6.9 mg/ml dimethyl sulfoxide), Ouabain (Na^+/K^+ ATPase inhibitor; 1.5 mg/ml dH_2O), and Oligomycin (mitochondrial ATPase inhibitor; 1 mg/ml 100% ethanol) were added to the reaction medium to eliminate non-specific ATPase activity. Enzyme activity was measured in the presence of 12 mg ATP. Tissue blanks (non-specific staining) were incubated in an identical reaction medium simultaneously but without ATP substrate. The final reaction medium was adjusted to pH 8.6. Following incubation for 3 min at 37°C, tissue sections were reacted in 2% $CoCl_2$ (10 min), washed, and reacted in 1% $(NH_4)_2S$ solution (1 min) at room temperature. The tissue sections were left to air dry and then mounted on glass slides with aqua-mountant (Gurr, BDH Chemical).

2. ß-Hydroxyacyl CoA dehydrogenase (EC 1.1.1.35; HOAD) (Modified from Bergmeyer 1974). Reaction medium: 1.5 mM nitro blue tetrazolium (NBT), 1.5 mM nicotinamide adenine

dinucleotide (NAD^+), 5 mM EDTA (disodium salt), 10 μM NaN_3, and 0.23 mM ß-hydroxybutyryl CoA (ß-HBC) in 80 mM Tris-HCl buffer (pH 8.1). The tissue sections were incubated for 14 min at 37°C. Tissue blanks were assayed without ß-HBC.

3. Cytochrome Oxidase (EC 1.9.3.1; CYTOX) (Modified from Old and Johnson 1989). Reaction medium: 5 mM 3,3'-diamino-benzidine (DAB) and 80 μM cytochrome c in 100 mM phosphate buffer (pH 7.3). The reaction was assayed at 37°C for 13 min. Tissue blanks were assayed without the presence of cytochrome c.

4. Succinate dehydrogenase (EC 1.3.99.1; SDH) (Blanco et al. 1988). Reaction medium: 1.5 mM NBT, 5 mM EDTA (disodium salt), 10 μM NaN_3, 20 μM 1-methoxyphenazine methosulfate (mPMS), 48 mM succinate (disodium salt) in 100 mM phosphate buffer (pH 7.6). The reaction was assayed in the dark at room temperature for 6 min. Tissue blanks for non-specific staining were incubated without the substrate succinate.

5. α-Glycerolphosphate dehydrogenase (EC 1.1.1.8; GPD) (Martin et al. 1985). Reaction medium: 1.2 mM NBT, 10 μM NaN_3, 20 μM mPMS, 9.3 mM α-glycerol phosphate (disodium salt) in 100 mM phosphate buffer (pH 7.4). The blank tissue sections were incubated without α-glycerol phosphate. Both reactions were incubated at 37°C for 19 min.

All enzyme assays were stopped by rinsing with distilled water 4-5 times. Tissue sections were allowed to air dry in the dark before mounting on glass slides with

aqua-mountant. The histochemical enzyme quantification was performed immediately following the tissue cutting, staining drying and mounting procedures. The linearity for each enzyme assay (Appendix E) was first determined, and then each assay was run at a steady state reaction time under the conditions described above. These procedures, for the histochemical quantification of enzymatic activity in prepared muscle cross-section tissue, as outlined by Martin et al. (1985), meet the standards of reliability and validity detailed by Stoward (1980). Steady state enzyme activity ($O.D.\cdot min^{-1}\cdot 10^{3}$) was calculated for each enzyme from the mean of 3 serial tissue sections with reaction product, minus the mean of 2 serial sections from the blank tissue. Appendix D.3 documents the reliability of the measurement technique used for the repeated analysis on the same single muscle fibres in each of the 3 serial tissue sections containing the reaction product ($r=0.73-0.99$; $p<0.05$).

Muscle glycogen concentration was determined biochemically using the amyloglucosidase assay technique described by Bergmeyer (1974). The tissue samples were homogenized (7x dilution) by hand (Schantz 1986) using glass tissue grinders in 50 mM Tris-HCl buffer containing 1 mM EDTA and 0.1% Triton X-100; pH 7.6 (Suarez et al. 1986). The reaction product was measured at 340 nm and 25°C using a Pye-Unicam PU8800 UV/Vis spectrophotometer with a temperature controlled water-jacketed cuvette holder.

Statistical Analysis:

A two-way analysis of variance (ANOVA) model with one factor (time) repeated, was utilized for analysis. A Least Significant Differences Multiple Comparisons procedure was used to locate simple main effects if any main eff ct was found statistically significant at $p<0.05$. Values are means ($\bar{X}$) ± standard error (SE) unless otherwise stated. A Pearson Product-Moment Correlation Coefficient (r) was used to examine the relationship between selected variables. No statistical comparisons were made between the 2 taper protocols (MD, MI), as the intent of this study was to examine the effects of different taper protocols on selected muscle properties and cycling performance.

RESULTS

Following the 7 wk endurance training programme there was a significant improvement in maximal oxygen consumption and performance time during the 40TT for all groups. Power output at ventilation threshold was also significantly increased (13%) after training (Table 3.1).

After the taper, the power output generated at VT during the laboratory cycling test was significantly increased from 235.02 ± 5.32 to 264.13 ± 4.50 W in the MI group (Table 3.1). During the 40TT ride, $\dot{V}O_2$, $\dot{V}_E$, HR, and RER were not different at any 10 km interval for any group

(Appendices D.4.1 to D.4.3). Performance time on the 40TT was not significantly different for any group after tapering. Figures 3.5 to 3.7 illustrate each individual cyclist's performance time within his respective taper group. A closer examination of these results showed that the performance times ranged from a 2:26 min decrement to a 9:02 min improvement for the MI group ($\bar{X}$= +2:33 min, $p>0.05$; Fig. 3.7); a 5:00 min decrement to a 7:37 min improvement for the MD group ($\bar{X}$= +1:13 min, $p>0.05$; Fig. 3.6), and 5 of the 8 cyclists in the CRT group had slower times ($\bar{X}$= +0:14 min, $p>0.05$; Fig. 3.5). However, one cyclist improved by 9:03 min, and if analysed without him, the average time for the CRT would be slower ($\bar{X}$= -1:02 min; $p>0.05$). A post-hoc analysis revealed that the power output generated at ventilation threshold on the post-taper cycle test was significantly related to performance time on the 40 km time trial ($r=-0.74$, $p<0.05$; Appendix D.5).

The muscle properties analysed during the taper are illustrated in Table 3.2. CYTOX and HOAD activity were significantly increased in the MI group. Inter-group enzyme differences, post-taper, illustrated that CYTOX (Fig. 3.2) and HOAD activity (Fig. 3.3) were significantly higher in the MI group relative to the CRT and MD groups. A post hoc analysis also found that CYTOX activity was significantly related to cycling time on the 40TT ($r=-0.50$, $p<0.05$; Appendix D.6). Muscle glycogen concentration was increased

($p<0.05$) in both the MI and MD groups, which were significantly higher than the CRT (Table 3.2; Fig. 3.4).

DISCUSSION

The purpose of this study was to examine the effects of reducing training volume using two different taper programmes on selected metabolic properties of single muscle fibres and on performance changes during both a simulated 40 km time trial ride and a ventilation threshold test. The most significant finding was that the laboratory cycling test results as reflected by an increase in power output at ventilation threshold, and the oxidative enzymatic activity of ß-hydroxyacyl CoA dehydrogenase and cytochrome oxidase were significantly increased in the MI group, which maintained exercise intensity while exercise duration was progressively reduced. The ventilation threshold test has been shown to be a reliable and valid test and a good predictor of endurance performance during running (Peronnet et al. 1987; Kumagai et al. 1982; Lehmann et al. 1983) and cycling (McLellan and Skinner 1985). The significant increase in PO at VT in the MI group is consistent with the results found in study 1, illustrating that tapering prior to a test can elicit an improvement at the performance level. Hence, tapering can be advantageous from a physiological perspective and to the author's knowledge, this is the first scientific study to demonstrate that both

performance and physiological muscular adaptations can occur following a specific tapering strategy.

When power output at ventilation threshold was correlated with actual cycling time on the simulated 40TT, which has been shown to be a reliable indicator of endurance performance in cyclists (Neary et al. 1990), a significant relationship was found ($r=-0.74$; $p<0.05$). This suggests that performance time on the 40TT would also improve after tapering, especially in the MI group which demonstrated the significant increase in PO at VT. However, the improvement in performance time may be small and not related to statistical tests whereas in practical terms a small improvement may be very significant (Millard-Stafford et al. 1990). Certainly a 2:33 min increase over a 60 min performance event is important and would justify a taper programme. As a group, the performance times did improve in accordance with the correlational findings but unfortunately, the improvement was not statistically significant. There are several potential reasons for this, including: 1. a mixture of cyclists and triathletes were used and this may have varied cycling experience. A homogenous group of cyclists in terms of age and cycling experience may have provided a more accurate assessment of the performance changes that can occur during a taper. Coyle et al. (1991) have illustrated that cycling experience is an important factor which can influence endurance

performance during a simulated 40 km time trial; 2. a larger sample size (n) would have provided a greater statistical power (Sharp and Gahlinger 1988) making it easier to reach significance statistically; 3. although the psychological factors were kept to a minimum, they may still have played a role during the time trial performance ride. Motivation and psychological factors are important elements for a successful competition (Carron 1984), and Pyke et al. (1988) found that the mood state changes during a taper mirrored the physiological state of training. Therefore, these factors may have contributed to the results found during the 40TT and so they must be considered when implementing a taper in preparation for endurance competition.

Muscle glycogen levels after the taper were significantly increased for both taper groups. This is in agreement with research by Sherman et al. (1981) and Roedde et al. (1986) who showed that glycogen concentration was elevated with diet and a 6-7 day taper protocol that maintained exercise intensity but reduced exercise duration. The increased glycogen levels following the taper may also, in part, be a result of a glycogen sparing effect because of the enhanced oxidative capacity through ß-oxidation (i.e. increased HOAD activity in the MI group). Reduced glycogenolysis is a major adaptation to endurance training, and based on these results, also occurred during tapering.

The significant increase in HOAD and CYTOX activity in

the MI group may be related to the fact that this group had more rest during the taper and/or possibly because of the maintenance of exercise intensity. This supports other training data which showed that the maintenance of exercise intensity is a key factor necessary for enzymatic adaptations to occur in skeletal muscle. Fitts et al. (1975) showed that steady state levels of cytochrome c were directly related exercise intensity, and Booth and Holloszy (1977) also found that intensity played a major role in the time course of the increase in cytochrome c. These studies demonstrated that training intensity is an important component required to elicit metabolic and biochemical changes in skeletal muscle as well as improvements at the performance level.

Gollnick and Saltin (1982) have also provided evidence that regulatory factors such as an increased oxidative enzyme activity, as shown by the significant increase in HOAD and CYTOX activity in the MI group, can assist to control the overall metabolic flux through the energy production pathways. Essen (1978) has suggested that these enzymatic regulatory factors are responsible for increased free fatty acid utilization and are influenced by exercise intensity. Increases in the oxidative capacity of muscle have been shown to have a direct influence on the sensitivity of cytosolic respiratory control which controls cellular energy balance (Dudley et al. 1987). Cellular

energy balance (i.e. recovery from fatigue) is important during the taper because rest is necessary to perform at an optimal level during competition (Morton et al. 1990).

Some studies have used reduced training as a model and have shown that training adaptations can be sustained if exercise intensity is maintained at a high level (Hickson et al. 1982; Houmard et al. 1989, 1990). Dudley et al. (1982) also showed that the adaptive response of cytochrome c in skeletal muscle of rodents was accelerated when intensity was increased. If the intensity of activity is important for the training response (Dudley et al. 1982; Hickson et al. 1982; Hoppeler et al. 1985), it would seem logical that the intensity of exercise would also be important during the taper. This would be consistent with the significant increase in HOAD and CYTOX activity in the MI group.

The physiological mechanism(s) responsible for the enzyme adaptations during a taper programme are presently unknown. However, the increase in HOAD and CYTOX activity was manifested when the training stimulus was maintained at a high intensity and when added rest (due to a reduction in exercise duration) was provided. Recently, Morton et al. (1990) proposed a theoretical model that could be used to predict athletic performance based on training intensity. In their model the balance between fatigue and fitness, which is directly related to the amount of rest, determines athletic performance. It now remains to be seen whether

this model is consistent with the cellular adaptations such as protein turnover rates.

In a review on protein turnover, Booth and Watson (1985) have suggested that enzymatic protein synthesis increases after the first 2-3 hr post-exercise and remained elevated for an undefined period of time. Meerson (1975) has also suggested that an elevated metabolic demand activates the synthesis of both structural and enzymatic mitochondrial proteins leading to the biogenesis of mitochondria. Conversely, enzyme activity may begin to decrease relatively abruptly upon detraining (Booth 1977), as was found in the control group from study 1. Much of this work on enzymatic protein turnover has been performed using cytochrome c and/or cytochrome oxidase. It has been proposed that the cytochromes may be the most sensitive mitochondrial constituents responding to training, and therefore probably the best predictors of endurance capacity (Fitts et al. 1975; Davies et al. 1981; Soussi et al. 1989). This supports the significant increase found in CYTOX activity in the MI protocol, and based on these results, may also include HOAD activity as a predictor of endurance capacity during training and tapering.

In summary, these results demonstrated that physiological adaptations can occur at the peripheral muscular and systemic levels after a taper. A significant increase in power output at the ventilation threshold was

found in the MI group, as was the enzymatic activity from ß-oxidation (i.e. HOAD) and the electron transport chain (i.e. CYTOX) pathways. No significant changes were found during the 40TT after tapering, suggesting that other factors also played a role during an actual performance ride. The physiological mechanism(s) responsible for the adaptations during tapering remain to be elucidated.

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Table 3.1. Maximal oxygen consumption ($\dot{V}O_2max$; $l \cdot min^{-1}$), power output at ventilation threshold (PO @ VT; Watts), oxygen consumption at 40 km during the 40TT ($\dot{V}O_2$ @ 40K; $l \cdot min^{-1}$) and Time (min) at 40 km during the 40TT before and after training and tapering for each group.

Group/ Variable	Endurance Training Before		Endurance Training After		Taper After	
CRT						
$\dot{V}O_2max$	3.97	(0.11)[a]	4.38	(0.10)[a]	4.33	(0.12)
PO @ VT	209.61	(15.61)[b]	237.82	(11.42)[b]	236.10	(8.80)
$\dot{V}O_2$ @ 40K	3.36	(0.20)	3.56	(0.14)	3.67	(0.16)
Time @ 40K	75:43	(4:08)[c]	66:32	(2:14)[c]	66:18	(1:28)
MD						
$\dot{V}O_2max$	4.04	(0.17)[d]	4.47	(0.19)[d]	4.51	(0.22)
PO @ VT	207.63	(13.40)[e]	238.72	(11.41)[e]	258.64	(12.70)
$\dot{V}O_2$ @ 40K	3.52	(0.25)	3.56	(0.18)	3.71	(0.19)
Time @ 40K	76:12	(3:20)[f]	66:45	(1:55)[f]	65:22	(2:32)
MI						
$\dot{V}O_2max$	3.95	(0.08)[g]	4.41	(0.06)[g]	4.51	(0.07)
PO @ VT	202.11	(13.74)[h]	235.02	(5.32)[hi]	264.13	(4.5)[i]
$\dot{V}O_2$ @ 40K	3.44	(0.14)	3.47	(0.12)	3.58	(0.08)
Time @ 40K	76:39	(2:23)[j]	66:42	(2:16)[j]	64:08	(1:49)

Values are Mean ± (SE)

Paired letters (a,a; b,b;.. j,j) indicate significant differences at $p<0.05$.

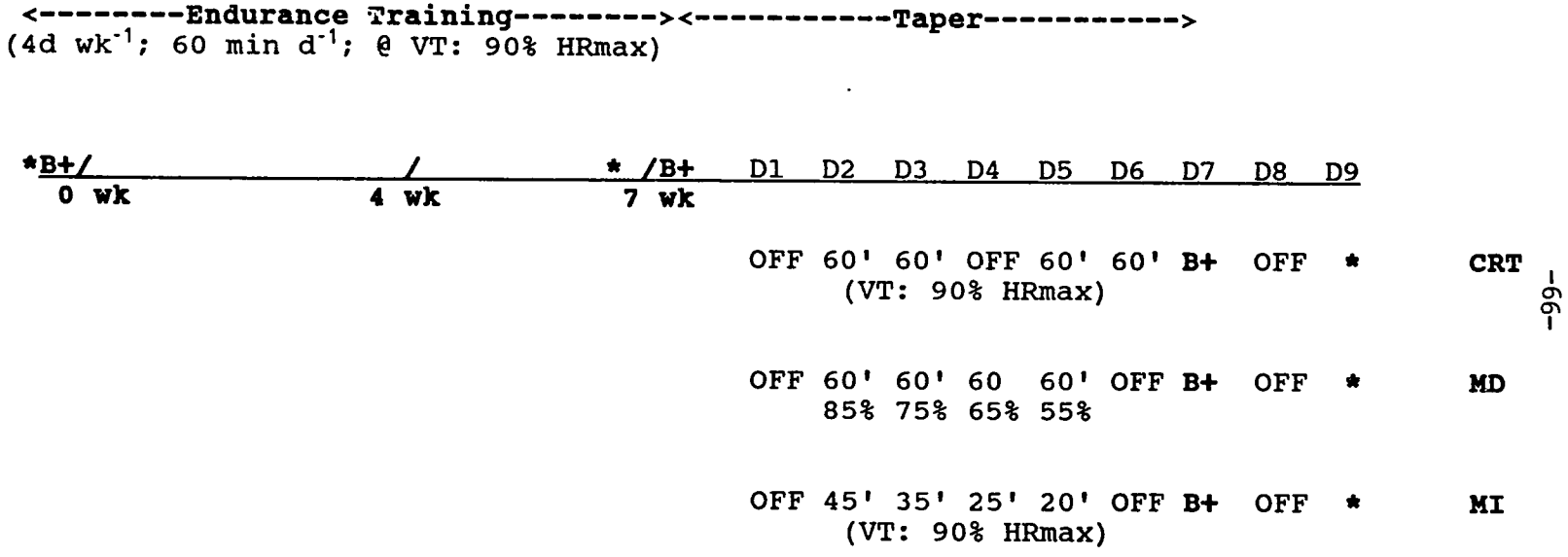
Table 3.2. Quantitative histochemical enzyme activity ($O.D._{\cdot} \cdot min^{-1} \cdot 10^{3}$) and muscle glycogen concentration ($\mu mol \cdot g^{-1}$; w.w.) before and after tapering for each group.

Group/ Variable	Taper Before		After		Percent change
CRT					
HOAD	45.49	(1.67)	49.28	(2.37)[a]	+8.3
CYTOX	69.05	(4.88)	73.09	(3.97)[b]	+5.8
SDH	189.46	(13.82)	209.46	(10.65)	+5.4
mATP	667.20	(24.24)	605.24	(44.83)	-10.2
GPD	15.43	(1.81)	19.54	(2.29)	+26.6
GLYCOGEN	118.29	(3.25)	120.18	(5.10)[c,d]	+1.6
MD					
HOAD	48.00	(3.13)	56.78	(4.03)	+18.3
CYTOX	66.38	(3.24)	75.76	(5.16)	+14.1
SDH	211.02	(20.27)	233.88	(24.18)	+10.8
mATP	675.37	(40.24)	614.81	(52.92)	-9.8
GPD	17.68	(1.23)	17.31	(1.95)	+2.1
GLYCOGEN	126.06	(5.53)[e]	162.37	(12.62)[c,e]	+28.8
MI					
HOAD	49.69	(3.87)[f]	60.95	(3.08)[a,f]	+22.7
CYTOX	71.95	(1.65)[g]	85.27	(3.99)[b,g]	+18.5
SDH	234.00	(21.81)	255.86	(23.58)	+9.3
mATP	715.51	(26.12)	672.32	(24.46)	-6.4
GPD	17.22	(1.45)	17.33	(1.59)	+0.6
GLYCOGEN	117.50	(6.37)[h]	158.44	(12.07)[d,h]	+34.8

Values are Mean ± (SE)

Paired letters (a,a; b,b;..h,h) indicate significant differences at $p<0.05$.

Figure 3.1. Experimental design for the exercise testing, training and tapering programme.



*= $VT/\dot{V}O_2max$ test (Note: $VT/\dot{V}O_2max$ test was performed on the last training day and then following the taper on D9

B= Muscle biopsy sample

+= 40 km time trial performance ride (40TT)

Figure 3.2: Cytochrome oxidase activity ($O.D. \cdot min^{-1} \cdot 10^{3}$) in single muscle fibres of endurance cyclists before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean ± SE.

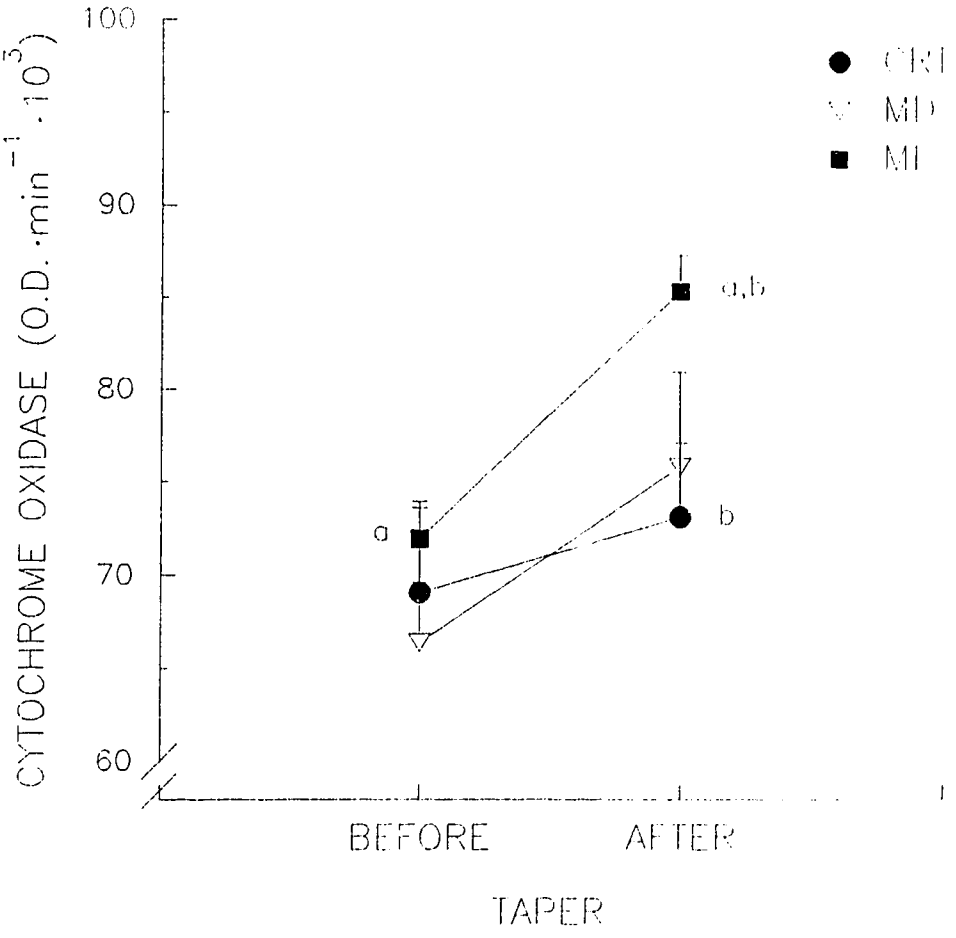


Figure 5.5: β-hydroxyacyl CoA dehydrogenase activity (O.D.$\cdot$min$^{-1}\cdot 10^{-3}$) in single muscle fibres of endurance cyclists before and after the taper. Paired letters indicate significance at $p<0.05$. Values are Mean $\pm$ SE.

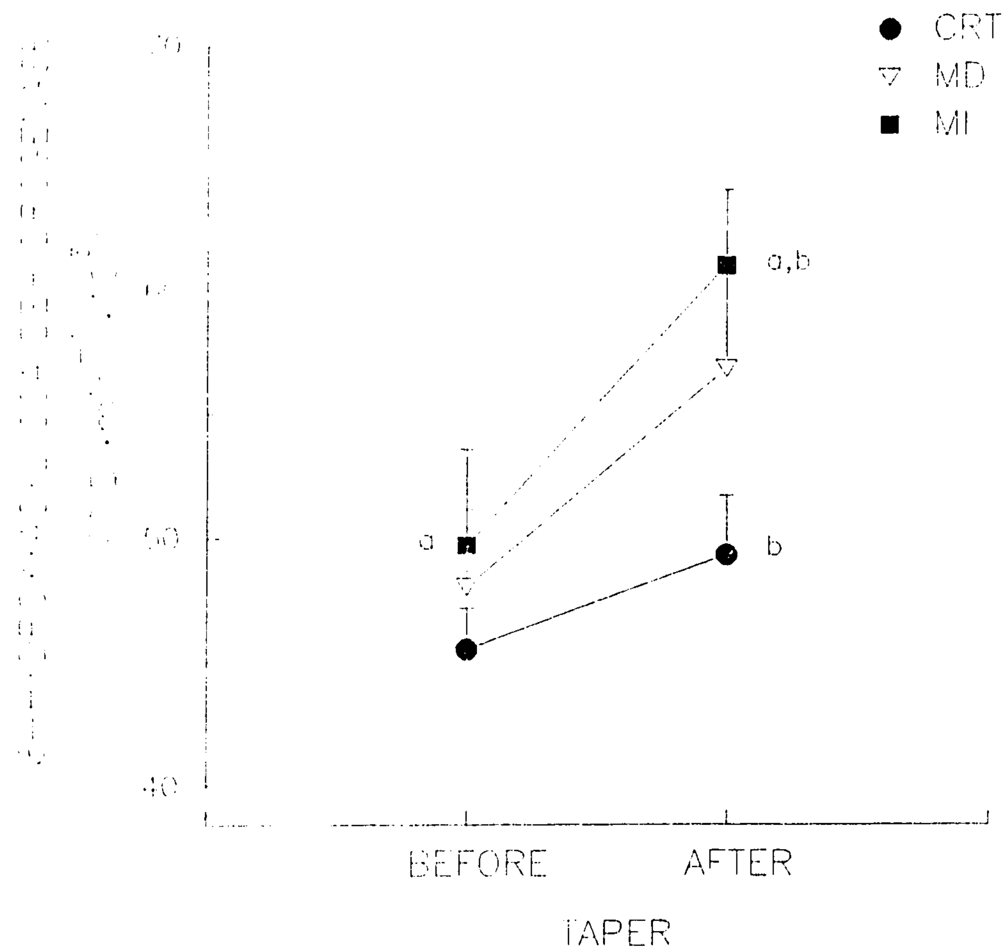


Figure 3.4: Muscle glycogen concentration [illegible] w.r.t. [illegible] endurance [illegible] before and [illegible] the taper. Different letters indicate significance at p<0.05. Values are Mean ± S.E.

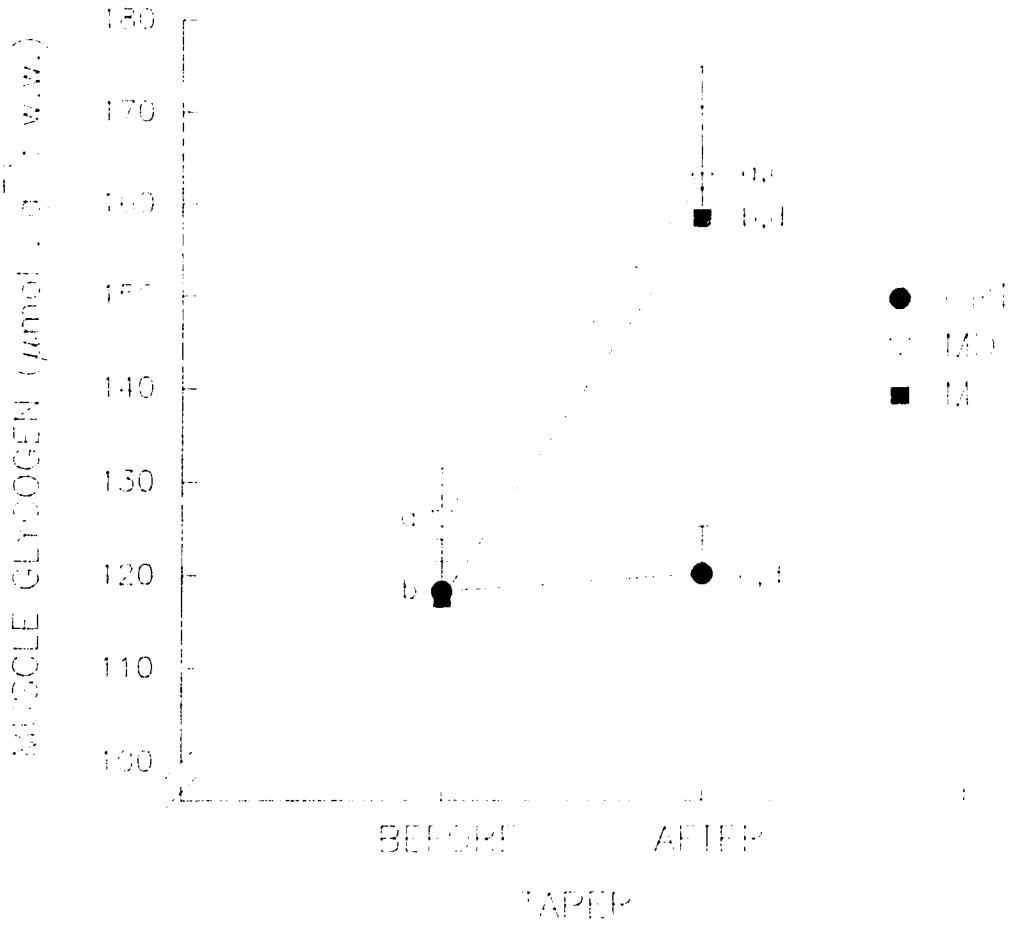


Figure 4.5: Performance time (min) for each cyclist during the simulated 40 km time trial before and after the taper in the control (C.H.) group.

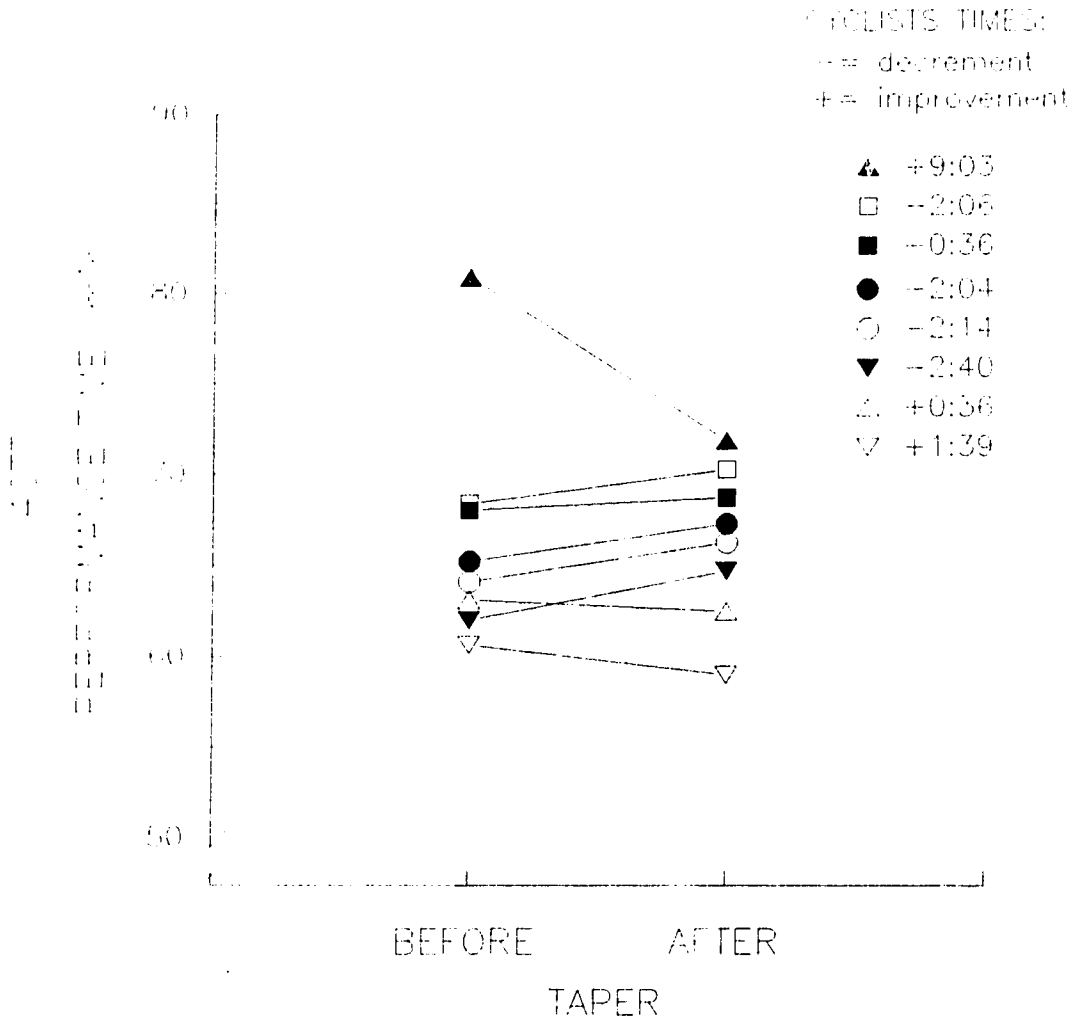


Figure [illegible]: Performance time (min) for each cyclist during the simulated [illegible] time trial before and after the taper in the group that maintained exercise [illegible]

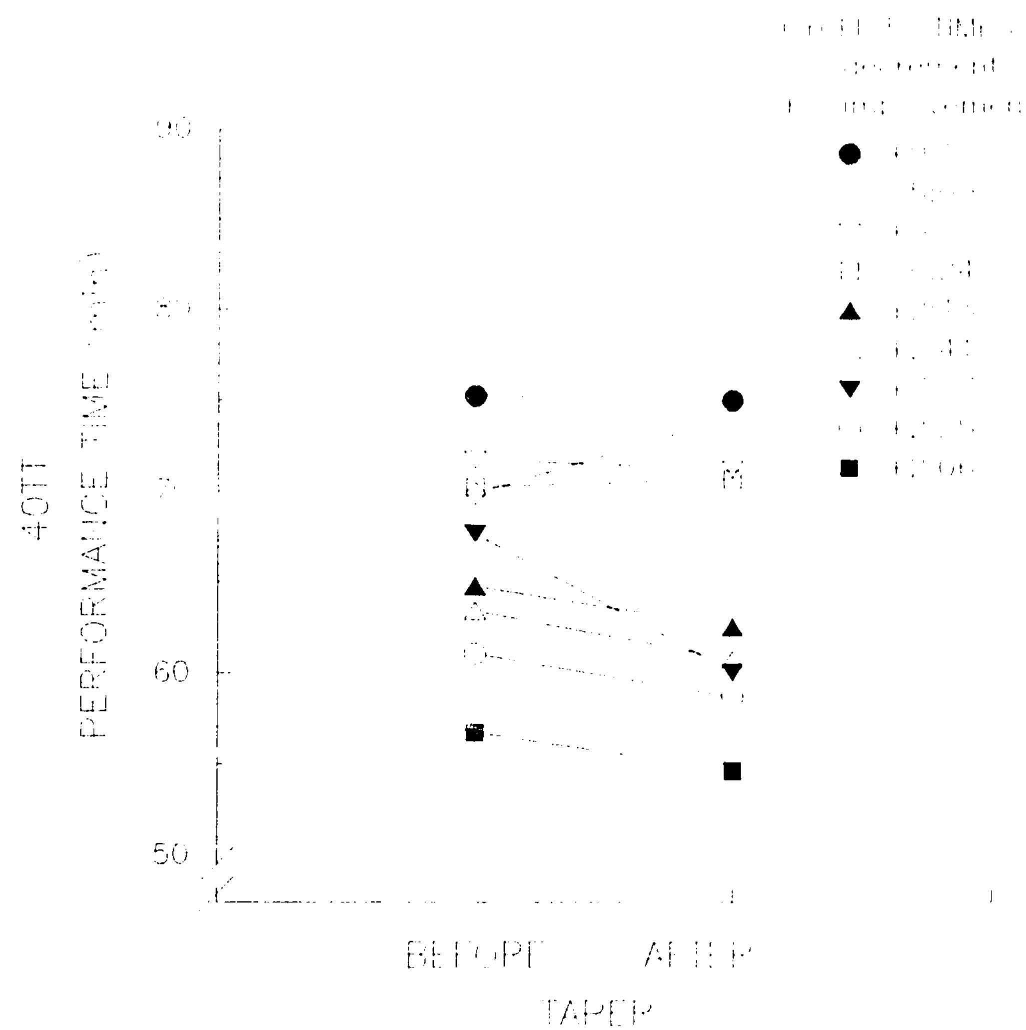
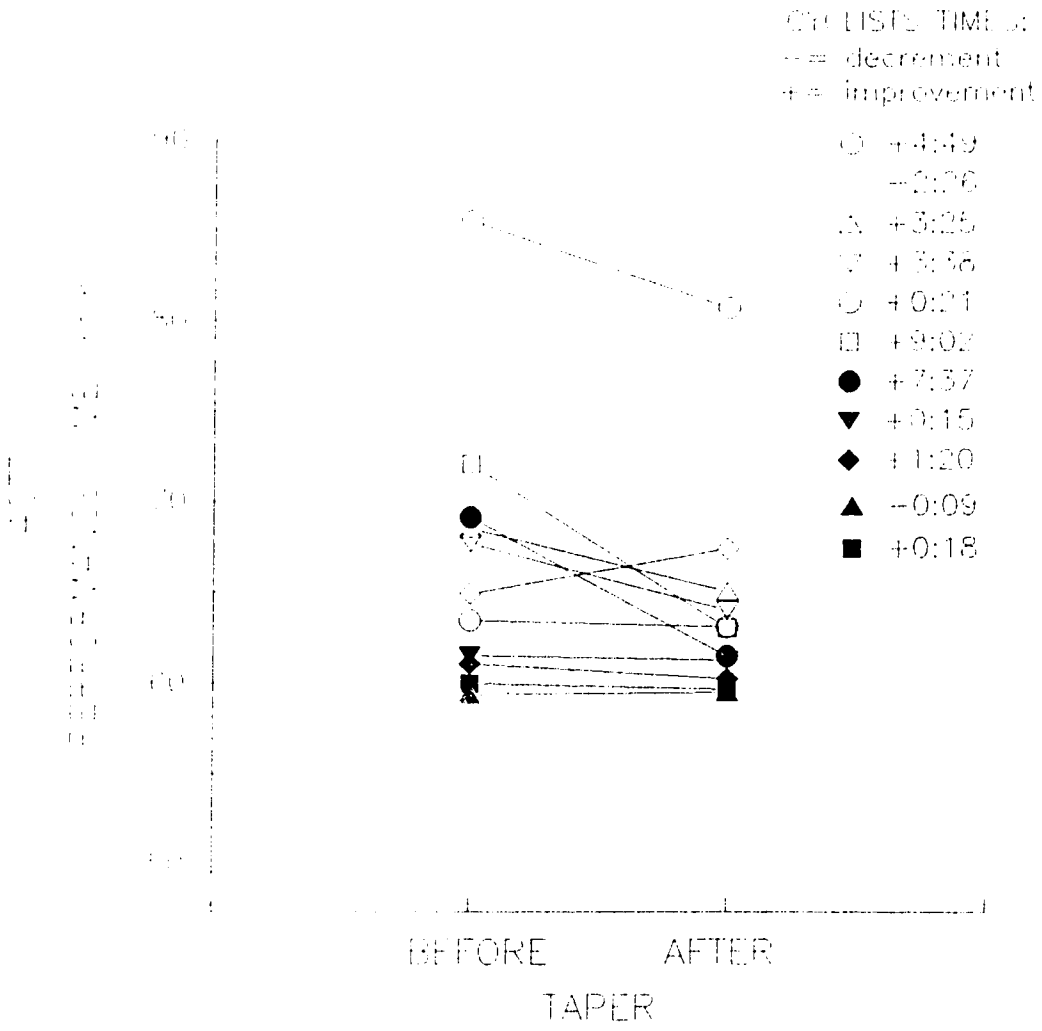


Figure 4.7: Performance time (min) for each cyclist during the simulated 40 km time trial before and after the taper in the group that maintained exercise intensity (EMI).



CHAPTER 4

GENERAL DISCUSSION

GENERAL DISCUSSION

The principle of tapering is poorly understood but appears to be important in preparing athletes for competition. For endurance athletes to perform at their maximal level of fitness, a training programme may need to include a taper. Tapering has been defined in this thesis as a specialised exercise training technique which is used to prepare athletes for competition in the days preceding an athletic event by reducing the intensity and/or duration of exercise, and thus manipulating the quantity of rest and exercise, while still maintaining the peripheral muscular and central cardiovascular adaptations developed through training. A variety of taper protocols, most of which have come from anecdotal reports, have been proposed. Due to the limited scientific information on tapering and because of the variety of taper protocols used by athletes, there is a need for scientifically controlled studies to examine what type of taper protocol is most suitable for enhancing endurance performance. Also, to fully understand the taper process, as well as for the development of better training programmes to maximize performance, there is a need to understand what are the physiological mechanism(s) occurring at the cellular level. Therefore, these studies were undertaken to examine the physiological changes in selected skeletal muscle properties and performance benefits following a taper programme that altered the length of

training and exercise intensity or duration.

Following the taper, a significant increase was found in the power output at ventilation threshold in the 4D, 8D and MI groups, and in selected metabolic properties of skeletal muscle. Although oxidative enzyme activity was not statistically increased during the taper for either the 4D or 8D taper groups in study 1, the results still provided evidence that metabolic changes did occur when training was altered. For example, in the control group HOAD activity was decreased ($p<0.05$) following 4 days of inactivity. This is consistent with the reports on highly trained athletes which showed that oxidative enzyme activity is reduced quickly during the detraining period (Houston et al. 1979; Chi et al. 1983; Coyle et al. 1984). When the training stimulus is removed, enzymatic activity is decreased as demonstrated by the labile nature of cytochrome oxidase activity which has a short half-life in humans (4-5 days) and rodents (6-7 days) (Booth and Holloszy 1977; Booth 1977; Henriksson and Reitman 1977).

The enzymes of ß-oxidation (i.e. HOAD) have been suggested to be in "constant proportionality" with cytochrome c, and they too may have a short half-life (Molé et al. 1971). During endurance exercise, it is essential that the energy production pathways keep pace with the energy demands of the exercising muscle. If training is stopped for too long prior to competition, as shown in the

CON group, then the enzyme activity needed for energy production via oxidative metabolism of fatty acids is reduced which may compromise endurance performance. This was demonstrated by the increased respiratory exchange ratio at the same absolute power output in the CON group. Also, the absolute change in CYTOX (Appendix C.3) was significantly higher in the taper (4D and 8D) versus control (CON and NOTAPER) groups which suggests that oxidative enzyme activity was at least maintained during the 4D and 8D protocols. Dudley et al. (1987) have shown that changes in the oxidative capacity of muscle directly influenced the sensitivity of cytosolic respiratory control. Hence, a decrease in mitochondrial enzyme activity (i.e. HOAD activity in the CON group) may reduce the muscles' capacity to perform exercise of long duration.

A number of studies have documented that reduced muscle glycogen levels can increase the onset of fatigue and decrease endurance performance when exercise is performed at a high intensity (for review see Conlee 1987). This is consistent with the results of the NOTAPER group which showed that muscle glycogen concentration and power output at VT were significantly decreased even after Day 4 of the taper period (Fig 2.2). A decrease in these variables will lead to a quicker onset of fatigue. Therefore, muscle glycogen concentration is important when performing high intensity exercise for prolonged periods (Ahlborg et al.

1967; Bergstrom et al. 1967; Costill and Miller 1980).

In study 2, the effects of maintaining either exercise intensity or duration showed that HOAD and CYTOX enzyme activity in single skeletal muscle fibres were significantly increased in the MI group (maintenance of intensity but a decrease in exercise duration). It is well understood from the early training literature that a major adaptation of endurance training is an increase in free fatty acid oxidation (Molé et al. 1971; Holloszy et al. 1970; Holloszy 1976; Costill et al. 1979). Essen (1978) has also illustrated that a greater contribution of lipids, both intramuscular triglyceride stores and blood borne fatty acids, were utilized as an energy source during 60 min of continuous moderate (157W) exercise.

The increased free fatty acid oxidation may be a result of an increased concentration of ß-oxidation enzymes (Molé et al. 1971; Baldwin and Winder 1977; Holloszy and Coyle 1984). Therefore, it would seem logical to suggest that the activity of the ß-oxidation enzymes should also be maintained at a high level for the taper to be effective. ß-oxidation was a prominent energy production pathway utilized during the taper in the MI group, and the adaptations in HOAD and CYTOX activity are consistent with the significant increase in power output at ventilation threshold. Also evident from these results was the fact that tapering did not compromise changes in ß-oxidation as

seen in the CON group from study 1. This would be consistent with other studies of exercise training (Booth and Holloszy 1977; Hickson et al. 1982; Dudley et al. 1982), reduced training (Hickson et al. 1984) and detraining (Chi et al. 1983; Coyle et al. 1984). A positive outcome of this adaptation in ß-oxidation is a decreased glycogenolysis (i.e. glycogen sparing) during endurance performance which allows exercise to be maintained at the same absolute workload for a longer period of time before stopping (Hermansen et al. 1967; Fitts et al. 1975; Baldwin and Winder 1977; Gollnick and Saltin 1982).

The metabolic muscular adaptations from studies 1 and 2 appeared to be consistent with the cycling tests in the laboratory. First, there was a strong correlation between CYTOX activity and the maximum power output generated during the post-training 60 min endurance cycle test in study 1 ($r=0.695$, $p<0.05$) and between CYTOX activity and the post-taper performance time during the 40TT in study 2 ($r=-0.50$, $p<0.05$).

Second, power output at ventilation threshold was significantly increased after the taper in both the 4 and 8 day groups in study 1, and in the MI group from study 2. In addition, when PO at VT was correlated with the cycling tests (i.e. 60 min endurance cycle test and the 40TT), a significant correlation was found ($r=0.80$ and $r=-0.74$; respectively). This is in agreement with Coyle et al.

(1991) who showed that the absolute power output generated during a simulated 40TT on a cycle ergometer was significantly correlated with performance time during an actual 40 km time trial road race ($r=-0.88$; $p<0.001$). However, a primary objective of a training and taper programme is to transfer the acquired physiological adaptations directly to the athletic event to achieve an improvement in performance. This was not evident from the group results on the 40TT which was designed specifically for these endurance cyclists. If individual cycling performance times are considered, 16 of 20 cyclists (80%) improved after tapering (MD and MI groups combined). However, the group data showed that the average improvement in cycling time for the MI (2:33 min) and MD (1:13 min) groups were not statistically significant. From both a practical (Millard-Stafford et al. 1990) and a physiological (Roedde et al. 1986) point of view, a provincial or national level cyclist would consider a 2:33 and 1:13 min improvement over a 40 km distance to be a significant improvement in their performance.

Third, muscle glycogen concentration was significantly increased in all groups undertaking a taper protocol which either reduced the duration or intensity of training (4D, 8D, MI, MD). Whether the maintenance of intensity (MI) or duration (MD) had a more significant affect on cycling performance is equivocal. The individual cycling data

appears to suggest that both protocols were equally effective. In the MD group, 7 of 9 cyclists (78%) showed improvements, while 9 of the 11 cyclists (82%) improved in the MI group. Therefore, it would appear that the reduction in the total volume (intensity and duration) may be an important factor.

The cellular mechanism(s) responsible for the acquired adaptations during tapering are unknown. However, based on these results it appears that the critical factors in the unloading process may be related to the maintenance of oxidative enzyme activity (i.e. HOAD and CYTOX) and muscle glycogen concentration. Booth and Watson (1985) revealed that enzymatic protein turnover can begin relatively quickly after acute exercise. This is not unexpected given the short half-lives of mitochondrial enzymes and may be related to the adaptations in oxidative enzyme activity observed following the taper. The enzyme results reported here support the theoretical model of training and tapering proposed by Morton et al. (1990). Morton et al. suggest that periodic evaluation of the physiological and biochemical responses of the athletes' peak preparedness assist to determine what intrinsic changes are needed fo. optimal performance. The significant increase in HOAD and CYTOX activity suggests that the fatigue curve for oxidative enzyme activity can be reduced with a 7 day taper when exercise duration is progressively reduced while maintaining

exercise intensity. By knowing the characteristics of the fatigue curve performance can then be predicted.

Other factors not examined in this study may also play a critical role during the taper. A recent study has provided evidence that hormonal changes may be important in the response of enzymatic protein turnover. Urhausen et al. (1987) examined the anabolic-catabolic relationship between serum testosterone and cortisol during an intense rowing training programme and found that a 1 wk "regenerative period" of reduced exercise intensity increased the testosterone/cortisol ratio back to normal. The taper may provide a regenerative period whereby the hormonal balance is re-established. This too, warrants further investigation.

As with any research project a variety of limitations are not uncommon. Some of the limitations in these studies included: 1). a small number (n) of subjects were involved which can have an affect on the results by altering the statistical power (Sharp and Gahlinger 1988); 2). a mixture of cyclists and triathletes were used as subjects. Cycling experience has been shown to contribute as a major determinant of cycling performance (Coyle et al. 1988; 1991); 3). the subjects in these studies varied in age from 17-36 yr. Much of the written anecdotal information on tapering has suggested that subject age also plays an important role, with younger athletes requiring less time to

taper (Wilkie and Madsen 1986); 4). some of the cyclists may not have reached a steady state training condition because of the length of a training programme. The length of training can influence the physiological and biochemical adaptations (Holloszy 1976; Hickson et al. 1984); 5). the length of the taper programme can also influence how each individual performs during competition. Since individuals respond differently to training (Bouchard 1986; Dionne et al. 1991), it is logical to assume that each cyclist may have responded differently to the 7 day taper with some of the cyclists requiring more or less time to taper (Morton et al. 1990); and 6). although every attempt was made to eliminate the psychological factors during testing, a psychological profile was not recorded after tapering so it is not possible to determine the extent of its effect on performance. Psychological factors do play a role during athletic performance (Carron 1984; Pyke et al. 1988).

Designing a Taper Programme

The information gathered here has made a considerable contribution to our limited knowledge on the taper principle. It is the first study to demonstrate that both physiological and performance level changes can occur after a specific tapering strategy. Although the information from these studies can only be generalized to this group of club level cyclists and triathletes, this research may be of

importance to other competitive endurance athletes training to optimize performance. It is recommended that training intensity should be maintained at a high level (90% HRmax) throughout the taper, while simultaneously reducing the duration of exercise to provide more rest to maintain both oxidative enzyme activity and the improvements found during the laboratory cycling tests (i.e. PO at VT). The total reduction in exercise duration during the 7 day MI protocol in study 2 was approximately 52% of the weekly training duration. This reduction was slightly less than the 62% decrease which was used in the swimming study by Costill et al. (1985) and suggested for runners (Costill 1986).

Future Research

The exercise-rest combination used for the MI protocol in study 2 had a significant effect on selected muscle enzymes, glycogen concentration and on the power output generated at the ventilation threshold. However, there are a great number of training combinations and permutations that can be examined during a taper. Future research studies are needed to examine more closely the reduction in total workload to determine whether maintaining exercise intensity or exercise duration is the more important factor. The optimal exercise intensity and duration must also be explored. Research on the effects of the length of the taper (i.e. number of days), influence of diet, type of

athletic sporting events (eg. cycling versus running versus swimming), interval versus continuous exercise, aerobic versus anaerobic versus strength training, and individual characteristics (gender, age, cycling experience) are all potential factors for consideration. Collaborative studies to investigate both the physiological and psychological profile of athletes during tapering are also warranted.

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APPENDIX A

REVIEW OF RELATED LITERATURE

REVIEW OF RELATED LITERATURE

The biological adaptations of a system are a result of chronic stress placed upon the system (Selye 1937; Meerson 1975). Regular physical exercise is one such stress which can alter the structural and functional components of an organism. The nature of the exercise stimulus specifically determines the type of adaptation that will result within the particular system being stressed (Holloszy 1976; Essen 1978; Martin 1987). For example, endurance exercise (i.e. running, swimming, cycling, rowing) involving large muscle groups contracting at submaximal tension for a prolonged period enhances the functional capacity of both the cardiovascular system and the oxidative metabolic capacity within skeletal muscle (Holloszy 1976; Clausen 1977; Baldwin and Winder 1977; Holloszy et al. 1977; Hoppeler et al. 1985; Schantz 1986). The extreme plasticity of skeletal muscle is a result of its capability to maintain a high protein turnover rate (Green 1990), with some metabolic enzymes having a half-life as short as 1 day (Illg and Pette 1979). For these biological adaptations to occur, adequate rest is an important component of a training programme (Riley 1977; Banister and Calvert 1980; Morton et al. 1990). Therefore, maximizing the training effect may be dependent upon provision of sufficient physical stress combined with adequate periods of rest.

"Tapering", as defined in this thesis, is a specialised

exercise training technique which is used to prepare athletes for competition in the days preceding an athletic event by reducing the intensity and/or duration of exercise, and thus manipulating the quantity of rest and exercise, while still maintaining the peripheral muscular and central cardiovascular adaptations developed through training. Thus, the accumulation of fatigue from training can then disappear. The term "peaking" has also been used to describe the preparation stage immediately preceding competition. However, peaking has been defined as the level of performance (Daland 1977) or the state of perfect physical and psychological readiness (Hogg and Montpetit 1982) which is demonstrated by a superior biological state that quickly adapts to a training stimuli (Bompa 1983, 1984). Bompa (1983) further suggested that peaking is a special training state characterized by a high central nervous system adaptation, motor and biological harmony, high motivation, the ability to cope with frustration and a high level of self-confidence.

There have been many anecdotal articles written on tapering and peaking (Daland 1977; Gallagher 1977; Banister and Calvert 1980; Hogg and Montpetit 1982; Bompa 1983; Costill 1986; Wilkie and Madsen 1986). The majority of these articles describe swimming tapers. By far, swimmers are the strongest advocates of tapering. This may be related to their heavy training schedule, which generally

includes 2 (2-3 hr) workouts per day. Most of these anecdotal articles are a theoretical account of what possibly happens during tapering (Gallagher 1977; Banister and Calvert 1980; Bompa 1983, 1984).

It is proposed that a reduction in exercise quantity (duration) and not the quality (intensity) that is important when attempting to maximise the difference between fatigue and the trained state (Morton et al. 1990). A fundamental problem associated with the aforementioned anecdotal reports is the lack of a generalized approach to defining the actual attributes of the taper programme. For example, several descriptive terms have been used such as the "drop", "slide", "blitz", "extended", "sawtooth", "mixed" and "gradual" taper. The fact remains that all reflect a variation of reducing both the intensity and duration of training. Further, the type of taper used is usually dictated by other factors such as the training state of the individual, importance of the event (i.e. major versus minor), type of event (sprint versus endurance) and the characteristics of the athlete (Gallagher 1977; Daland 1977; Miller 1977; Meisel 1977; Hogg and Montpetit 1982).

If workload is greatly reduced during the taper, there will be a loss of the training adaptations (Chi et al. 1983; Coyle et al. 1984; Neufer 1989). Much of this information has come from studies which have examined the effects of "detraining" and "reduced" training on cardiovascular and

metabolic parameters (Henriksson and Reitman 1977; Houston et al. 1979; Saltin and Rowell 1980; Chi et al. 1983; Coyle et al. 1984, 1985, 1986; Costill et al. 1985a, 1985b; Russell et al. 1987; Hawley 1987; Allen 1989 Houmard et al. 1990). However, the majority of these studies have focused on metabolic and enzymatic changes occurring over extended periods of time (2-20 wk), and have shown that training adaptations (i.e. central cardiovascular versus peripheral muscular) are lost at different rates (Coyle et al. 1985; Allen 1989). Although, detraining is a useful model it does not provide an answer to what is the optimal exercise intensity, duration, or length of the training period needed preceding an athletic event. Manipulating the volume of exercise preceding a major competitive athletic event, i.e. "tapering", is important if the athlete is expected to perform at a high level. It is difficult to perform at a maximal level of competition if the athlete is in the middle of a heavy training phase because of the amount of fatigue which accompanies daily training (Banister and Calvert 1980; Morton et al. 1990). Therefore, reducing the total training volume will allow both general and local body fatigue to diminish, while at the same time maintaining both the cardiovascular and peripheral metabolic adaptations at an optimal state of physiological function. The goal of tapering is to reduce the workload for a short period of time to reverse fatigue without a lose of the training

adaptations.

Many articles have been written on tapering, but only a limited body of research literature is available. Costill et al. (1985b) used a group of collegiate swimmers and demonstrated that strength, power and performance time improved following a 2 wk taper where exercise duration was reduced, but intensity was maintained. In a case study on six Australian national cycling team members, Pyke et al. (1988) found a general improvement in the performance variables tested with significant gains in power output during a simulated cycling race after 7-10 days of tapering. Yamamoto et al. (1988) found that serum creatine kinase enzyme was a sensitive indicator of physiological stress during both training and tapering. During a 20 day taper using a small group (n=9) of college swimmers, Van Handel et al. (1988) found no changes in any of the performance or blood biochemical indices; whereas Montpetit (1982) found decreases in skeletal muscle citrate synthase enzyme activity, although swim performance times after 14 days of tapering were not significantly slower (14 s) statistically. Although there were no control groups, and muscle biopsy tissue was only taken in the Montpetit (1982) study, the general consensus was that performance can be enhanced after a period of tapering. However, because of these limitations it was not possible to describe the regulatory factors at the muscular cellular level that are responsible for changes

that occurred during the taper. Costill et al. (1985b) speculated that increases in muscle strength may be related to the changes in the contractile mechanisms and/or neural control of fibre recruitment.

Where might the taper help?

During the taper, many processes are occurring at the local muscle level which may influence athletic performance. For example, it has been shown that strenuous acute and chronic endurance training can result in significant injury to skeletal muscle fibres (for review see Armstrong 1986). This damage includes: elevated blood levels of muscle enzymes such as creatine kinase, lactate dehydrogenase and aspartate aminotransferase (Sanders and Bloor 1975), hyperkalaemia and phosphatemia (Knochel 1982), and haemoglobin, myoglobin and hydroxyproline in the urine (Abraham 1977), Z-band streaming (George et al. 1987), reduced intracellular ATP (Oberc and Engel 1977; Dudley et al. 1987) and oedema. The consequence of this damage is a reduction in both maximal voluntary and involuntary strength (Newham et al. 1983), which will ultimately reduce performance. Therefore, it is important that adequate periods of rest be interspersed during training to reverse these damages to promote an improved performance.

Another important site which may contribute to an improved function during the taper is in the central nervous

system. Bompa (1983) suggests that the nervous tissue becomes fatigued if over-stressed for a prolonged period of time without adequate rest, and this may contribute to a reduced performance during competition. Hence, psychological readiness may contribute to peak performance following a taper (Pyke et al. 1988). Hormonal changes (Barron et al. 1985; Urhausen et al. 1987) may also play an indirect role in the taper response. Some evidence has been provided which suggests that altered testosterone levels may reduce glycogenesis and influence energy metabolism in skeletal muscle (Gillespie and Edgerton 1970).

Potential problems with the taper

As previously discussed, peripheral adaptations such as enzymatic activity can be lost quickly if the level of training stimulus is inappropriate. For example, Montpetit (1982) tapered a group of young (16-17 yr) swimmers and found a significant decrease in citrate synthase activity following a 2 wk taper but not during a 1 wk taper. This may suggest that the exercise load was inadequate to maintain the training effect. Because tapering can be considered a fine tuning of the physiological mechanism(s), it is probable that only small changes will be occurring during this period of time. Thus, the experimental design properties to be studied, and measurement sensitivity must be carefully controlled. Variables such as diet and

hydration, training environment, training state, travel arrangements and medical problems are important considerations. Other factors that can also influence the taper include individual characteristics such as age, gender, training and competition experience, skill and ability, and a variety of psychological factors from social relationships (i.e. family, friends, peers) to event distractions (i.e. type of competitive environment, sleeping and eating facilities) (Hogg and Montpetit 1982).

The data collected from these studies has added additional information on the physiological adaptations which can occur during tapering. When selected metabolic properties of muscle were investigated, the results demonstrated that significant increases occurred in the activities of ß-oxidation and electron transport pathway enzymes, and in muscle glycogen concentration. The cycling results from the laboratory tests as reflected by the PO generated at VT was also significantly increased. It appears that a reduction in exercise duration while maintaining exercise intensity is an important factor to consider during the taper.

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APPENDIX B

INFORMED CONSENT

SPORT PHYSIOLOGY LAB
DEPARTMENT OF PHYSICAL EDUCATION
AND SPORT STUDIES
UNIVERSITY OF ALBERTA

INFORMED CONSENT FOR PHYSIOLOGICAL ASSESSMENTS

The following physiological assessments (indicated by a check unless otherwise stated) will be conducted by qualified personnel as a part of a personal fitness appraisal and/or research project:

A). **Physiological Responses to a Submaximal Workload**

Ergometer:

a). heart rate[1] ( )

b). metabolic responses[2] ( )

c). other ( ) _____

B). **Maximal Oxygen Consumption ($\dot{V}O_2max$)**

Ergometer:

a). heart rate ( )

b). metabolic responses ( )

c). other ( ) _____

C). **Anaerobic Threshold (Ventilation and/or Lactate Threshold)**

Ergometer:

a). heart rate ( )

b). metabolic responses ( )

c). blood lactate ( )

d). other ( ) _____

D). **Isokinetic Strength Assessment at Various Angular Velocities**

Dynamometer:

a). peak torque ( )

b). work ( )

c). muscular endurance ( )

d). joint(s) ( )

e). number of contractions ( )

f). angular velocities ( )

E). **Anaerobic Power and/or Capacity**

Ergometer:

a). power output ( )

b). total rpms ( )

c). blood lactate ( )

d). other ( )

_____

F). **Body Composition**

Ergometer:

a). height/weight ( )

b). skinfolds ( )

c). body densitometry ( )

G). **Flexibility**

a). sit and reach ( )

b). other ( )

_____

H). **Muscular Endurance**

a). sit-ups ( )

b). push-ups ( )

c). other ( )

_____

I). **Blood/Urine Chemistry Analyses**[3]

a). haemoglobin ( )

b). haematocrit ( )

c). lactate ( )

d). glucose ( )

e). other ( )

_____

1. Heart rate will be determined with either an ECG ( ) or a sport tester heart rate monitor ( ).
2. Metabolic responses will be assessed using open circuit spirometry with analyses of expired air by a gas analysis system.
3. A blood sample will be taken from an antecubital vein, a finger prick, or from an indwelling catheter by a qualified laboratory technician or an IV nurse.

RESEARCH PROJECT

The purpose of this research project is to determine the effects of tapering, following strenuous endurance cycle training, on endurance performance and on the physiological adaptations resulting from a taper phase. The physiological assessment of each cyclist will include selected variables from those mentioned above as well as muscle tissue samples. Only two muscle samples, one before and one after the taper, will be taken from the vastus lateralis (quadriceps) muscle by a qualified physician at the Glen Sather Sport Medicine Clinic at the University of Alberta. A local anaesthetic will be used to freeze that portion of the leg where the tissue sample will be taken.

Training

All training will be done on your own bike mounted on magnetic turbo trainers or rollers. Endurance training will strenuous and continuous in nature, 4-5 days/wk for 7-8 wk.

Training intensity will be at 85-90% of HRmax for 60 min/session. Following the training, you will be randomly assigned to a taper group, during which time you will be required to performed only the specific exercises I give you. Training will be monitored using portable HR monitors.

Blood samples will be collected by a qualified laboratory technician using the venipuncture technique. One sample will be collected from a superficial forearm vein before and 5 min after each test. In addition, a weekly blood sample will be collected throughout the duration of the study. These samples will be analysed for lactic acid and creatine kinase enzyme. A 24 hour urine sample will be collected once a week and analysed for enzymes and hormones which are used to indicate changes in training status.

Risks

During blood sampling a small bruise may occur later at the site of blood taking. The amount of blood taken (4-5 ml) will not affect your physical performance during or after exercise. The $\dot{V}O_2$max test will be intense and therefore will require a maximal effort on your part. Some feelings of light headedness may occur during testing, blood taking and muscle sampling. The muscle biopsies may cause some discomfort and you may experience a mild "charlie-horse" sensation the day following; however, this should not affect your testing or training.

INFORMED CONSENT FOR SUBJECTS

I have read the above and agree to participate in this research project at my own risk. I realize that I may expect a thorough explanation and/or demonstration of any procedures now or at any time in the future, and that I may terminate participation at any time in any or all procedures of my own volition. I will also be assured anonymity of all results gathered in this research.

Having voluntarily assumed participation and the risks thereof, in this project, I hereby disclaim and release the University of Alberta, its agents, servants, or employees, including all personnel involved in this research project, from any and all liability that might otherwise arise as a result of my participation as a research subject in this project.

Name:_____ Date:_____

Address:_____

_____

Phone:_____

Signature:_____

I, _____(parent's name), am fully aware of the benefits and risks involved in having _____ _____(cyclist's name) participate in this scientific research study being conducted at the Sport Performance Laboratory, University of Alberta, Edmonton. I understand that all experimental procedures (blood and muscle tissue administration, and physiological fitness tests) will be conducted by qualified and medical personnel, and that emergency first-aid is readily available. I also understand that _____ (cyclist's name) can withdraw from the study at any time. I release the University of Alberta, its agents, employees and all personnel involved in this research study from any and all liability that might otherwise arise as a result of _____ (cyclist's name) participation as a research subject in this project.

Parent's Name:_____ Date:_____

Address:_____

_____

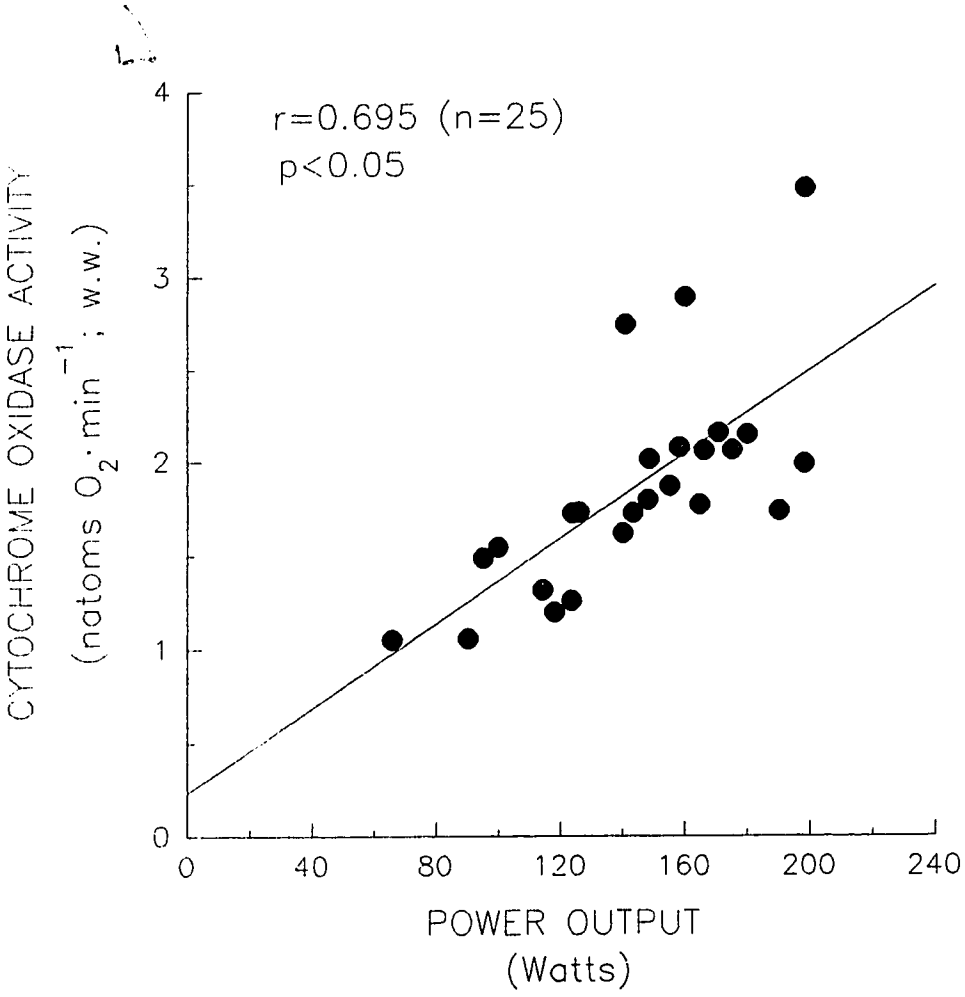
Phone:_____

Parent's Signature:_____

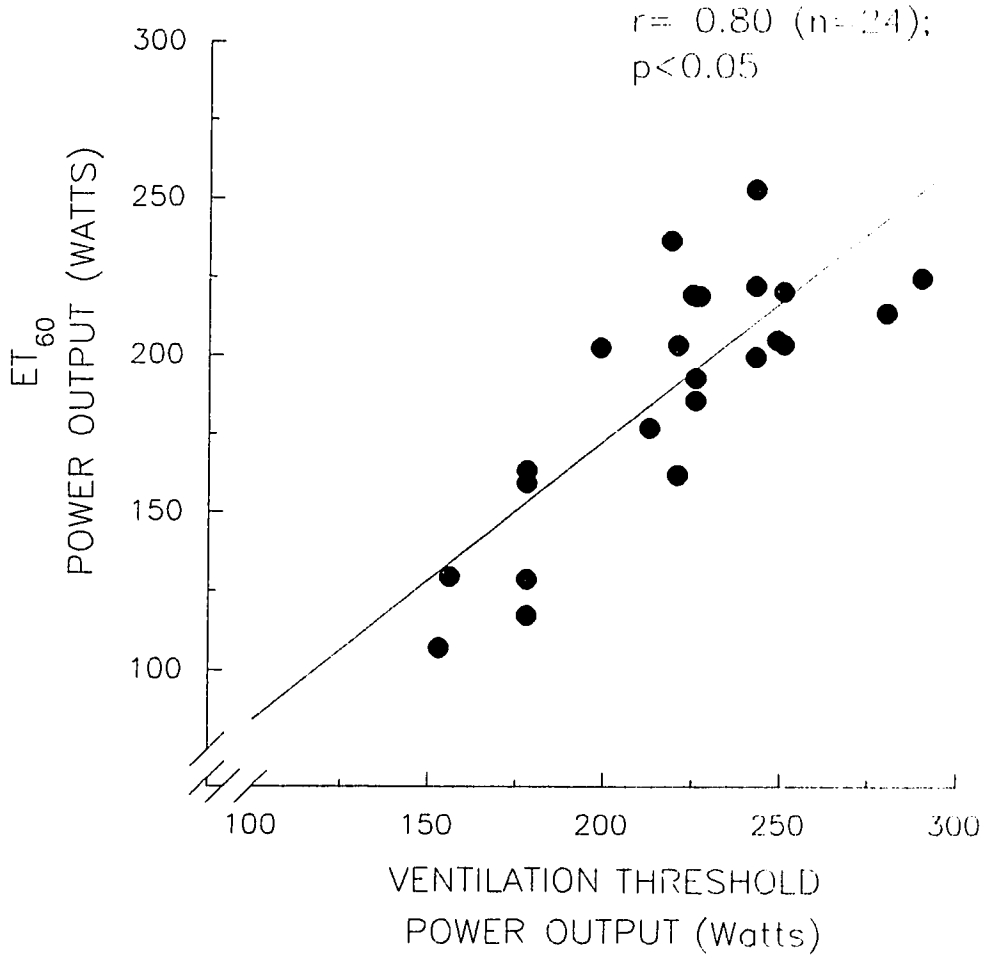
APPENDIX C

ADDITIONAL RESULTS (CHAPTER 2)

Appendix C.1: Relationship between the post-training cytochrome oxidase activity (natoms O_2 min^{-1}; w.w.) and power output (Watts) during the 60 min endurance cycling test.



Appendix C.2: Relationship between the power output (Watts) maintained during the post training 60 min endurance cycle test (ET_{60}) versus power output at the ventilation threshold.



Appendix C.3. Comparison of oxidative enzyme activity ($\mu mol \cdot g^{-1} \cdot min^{-1}$, w.w.; CYTOX= natoms $O_2 \cdot min^{-1}$; w.w) and muscle glycogen concentration ($\mu mol \cdot g^{-1}$; w.w.) between groups. Values are delta scores (i.e. post- minus pre-taper).

Variable	Group			
	4D	8D	CON	NOTAPER
CPT	0.01	0.03	-0.06	0.0
CS	1.37	0.16	-1.19	2.25
HOAD	0.80	-0.01	-2.75	0.57
CYTOX	0.50[a]	0.57[b]	-0.19[ab]	0.35
GLYCOGEN	22.44[c]	32.63[d]	15.76[e]	-18.04[cde]

Paired letters (a,a; b,b;..e,e) indicate significant differences at $p<0.05$.

Minus (-) sign indicates a decrease after the taper.

APPENDIX D

ADDITIONAL RESULTS (CHAPTER 3)

Appendix D.1: Pearson product-moment correlation coefficients (r) for the respiratory variables for ventilation ($\dot{V}_E$; $l \cdot min^{-1}$), oxygen consumption ($\dot{V}O_2$; $l \cdot min^{-1}$), carbon dioxide production ($\dot{V}CO_2$; $l \cdot min^{-1}$) and respiration exchange ratio (RER) between the 2900 and Horizon gas analyses systems at a submaximal power output (n=2) and at maximal oxygen consumption (n=2)

	Submaximal (125 W)		Maximal (2900 only)	
Variable	2900	Horizon	1st Trial	2nd Trial
$\dot{V}_E$	42.8 (11.9)	43.1 (11.1)	144.1 (18.3)	166.2 (11.6)
$\dot{V}O_2$	1.74 (0.44)	1.78 (0.40)	3.82 (0.56)	3.96 (0.53)
$\dot{V}CO_2$	1.51 (0.36)	1.59 (0.31)	4.20 (0.58)	4.49 (0.25)
RER	0.87 (0.03)	0.89 (0.03)	1.10 (0.01)	1.14 (0.01)
Correlation:		r=0.99 ($p<0.05$)		r=0.99 ($p<0.05$)

Values are Mean ± (SD)

Appendix D.2. Cyclists' maximal and submaximal heart rates (HR; bpm) during the maximal oxygen consumption ($\dot{V}O_2max$) test and during the endurance training programme for each group.

Group	$\dot{V}O_2max$ Test	Endurance Training	% HR Training Intensity
CRT (n=8)	195 (4.0)	174 (3.0)	89%
MD (n=9)	193 (3.0)	176 (3.0)	91%
MI (n=11)	192 (3.0)	174 (3.0)	90%

Values are Mean ± (SE)

Appendix D.3. Reliability of the quantitative histochemical enzyme activity ($O.D.\cdot min^{-1}\cdot 10^{3}$) analyses for myofibrillar ATP (mATP), cytochrome oxidase (CYTOX), α-glycerol phosphate dehydrogenase (GPD), ß-hydroxyacyl CoA dehydrogenase (HOAD) and succinate dehydrogenase (SDH) on the same single muscle fibres in serial tissue sections (n=2).

	Enzyme Assay				
	mATP	CYTOX	GPD	HOAD	SDH
r=	0.79-0.99	0.93-0.99	0.73-0.99	0.98-0.99	0.73-0.79
p=	(p<0.05)	(p<0.05)	(p<0.05)	(p<0.05)	(p<0.05)

r= Pearson Product-Moment Correlation

Appendix D.4.1 Time (min), oxygen consumption ($\dot{V}O_2$;l·min^{-1}), ventilation ($\dot{V}_E$; l·min^{-1}), heart rate (HR; bpm) and respiratory exchange ratio (RER) at each 10 km interval during the 40 km time trial before and after training and tapering in the CRT (regular weekly training) group (n=8).

Distance/ Variable	Endurance Training Before		After		Taper After	
10km						
Time	18:02	(0:42)[a]	15:53	(0:52)[a]	15:53	(0:38)*
$\dot{V}O_2$	3.44	(0.17)	3.48	(0.12)	3.63	(0.10)
$\dot{V}_E$	98.38	(6.33)	119.66	(7.12)	124.60	(7.10)*
HR	178.0	(3.0)	172.0	(4.0)	172.0	(4.0)
RER	1.01	(0.02)[b]	1.09	(0.02)[b]	1.06	(0.01)*
20km						
Time	37:05	(1:35)[c]	32:49	(0:51)[c]	32:30	(0:35)*
$\dot{V}O_2$	3.46	(0.18)	3.45	(0.12)	3.50	(0.15)
$\dot{V}_E$	94.25	(6.10)	112.07	(5.95)	122.20	(5.78)
HR	178.0	(4.0)	171.0	(4.0)	172.0	(4.0)
RER	1.01	(0.02)[d]	1.04	(0.01)[d]	1.05	(0.01)*
30km						
Time	57:19	(2:46)[e]	49:59	(1:15)[e]	49:28	(0:56)*
$\dot{V}O_2$	3.41	(0.20)	3.48	(0.12)	3.48	(0.12)
$\dot{V}_E$	91.93	(6.10)	112.47	(4.92)	124.47	(7.65)*
HR	177.0	(3.0)	172.0	(3.0)	173.0	(5.0)
RER	0.96	(0.01)[f]	1.04	(0.02)[f]	1.04	(0.01)*
40km						
Time	75:43	(4:08)[g]	66:32	(2:14)[g]	66:18	(1:28)*
$\dot{V}O_2$	3.36	(0.22)	3.56	(0.13)	3.64	(0.14)
$\dot{V}_E$	89.23	(5.79)[h]	124.24	(4.18)[h]	125.99	(4.19)*
HR	174.0	(3.0)	176.0	(4.0)	174.0	(4.0)
RER	0.97	(0.01)[i]	1.08	(0.01)[i]	1.05	(0.02)*

Values are Mean ± (SE)

Paired letters (a,a; b,b;.. i,i) indicate significant differences at $p<0.05$.

* significantly different from pre-training ($p<0.05$).

Appendix D.4.2 Time (min), oxygen consumption ($\dot{V}O_2$;l·min^{-1}), ventilation ($\dot{V}_E$; l·min^{-1}), heart rate (HR; bpm) and respiratory exchange ratio (RER) at each 10 km interval during the 40 km time trial before and after training and tapering in the MD (maintained duration, reduced intensity) group (n=9).

Distance/ Variable	Endurance Training Before		After		Taper After	
10km						
Time	18:13	(0:52)[a]	16:09	(0:34)[a]	15:36	(0:36)*
$\dot{V}O_2$	3.53	(0.20)	3.52	(0.20)	3.70	(0.18)
$\dot{V}_E$	102.81	(6.98)	123.18	(7.25)	133.93	(7.80)
HR	172.0	(4.0)	170.0	(2.0)	173.0	(2.0)
RER	0.97	(0.01)[b]	1.07	(0.01)[b]	1.10	(0.01)*
20km						
Time	36:50	(1:37)[c]	32:57	(1:00)[c]	32:06	(1:14)*
$\dot{V}O_2$	3.59	(0.18)	3.44	(0.21)	3.55	(0.18)
$\dot{V}_E$	103.12	(5.35)	118.62	(6.87)	130.74	(9.09)*
HR	172.0	(4.0)	171.0	(3.0)	173.0	(3.0)
RER	0.96	(0.01)[d]	1.04	(0.02)[d]	1.07	(0.02)*
30km						
Time	56:08	(2:30)[e]	50:04	(1:29)[e]	49:00	(1:54)*
$\dot{V}O_2$	3.47	(0.21)	3.44	(0.19)	3.45	(0.19)
$\dot{V}_E$	96.36	(6.78)[f]	120.61	(6.01)[f]	123.97	(8.81)*
HR	175.0	(3.0)	170.0	(3.0)	169.0	(3.0)
RER	0.94	(0.02)[g]	1.06	(0.01)[g]	1.06	(0.01)*
40km						
Time	76:12	(3:20)[h]	66:45	(1:55)[h]	65:22	(2:32)*
$\dot{V}O_2$	3.52	(0.25)	3.59	(0.18)	3.71	(0.19)
$\dot{V}_E$	101.08	(7.85)[i]	130.39	(5.17)[i]	136.62	(8.34)*
HR	172.0	(4.0)	174.0	(3.0)	174.0	(3.0)
RER	0.95	(0.02)[j]	1.07	(0.02)[j]	1.08	(0.01)*

Values are Mean ± (SE)

Paired letters (a,a; b,b;.. j,j) indicate significant differences at $p<0.05$.

* significantly different from pre-training ($p<0.05$).

Appendix D.4.3 Time (min), oxygen consumption ($\dot{V}O_2$;l·min^{-1}), ventilation ($\dot{V}_E$; l·min^{-1}), heart rate (HR; bpm) and respiratory exchange ratio (RER) at each 10 km interval during the 40 km time trial before and after training and tapering for the MI (maintained intensity, reduced duration) group (n=11).

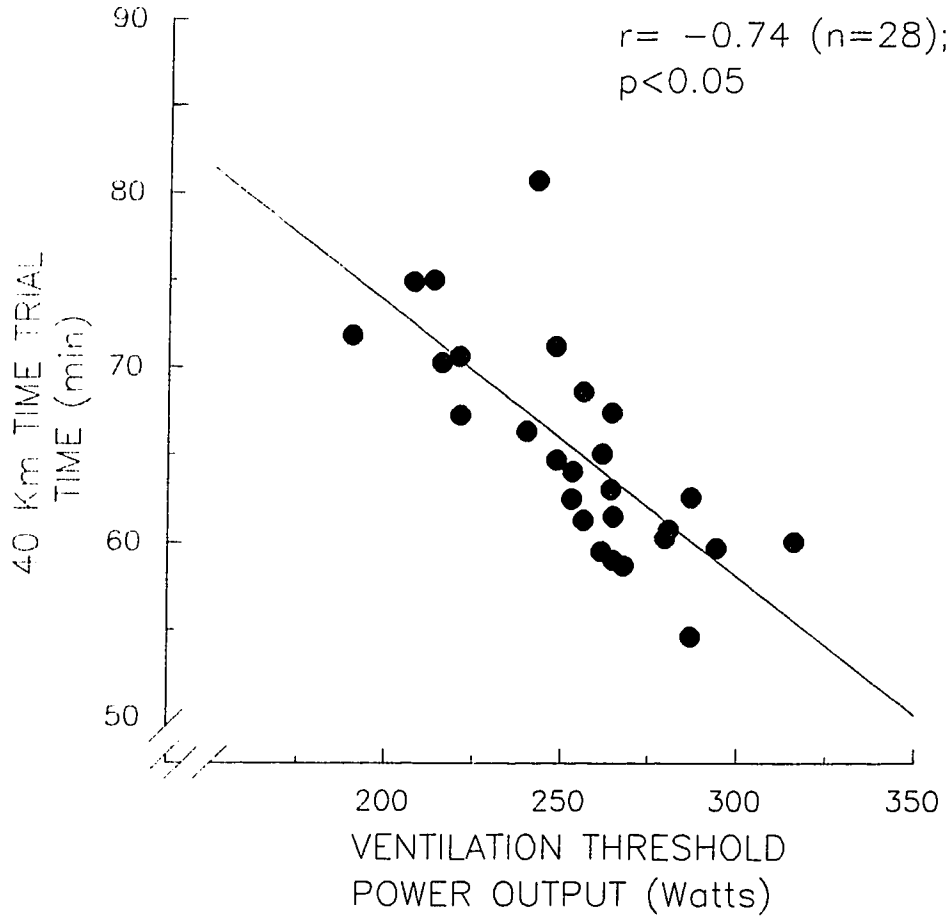
Distance/ Variable	Endurance Training Before		After		Taper After	
10km						
Time	16:55	(0:22)[a]	15:52	(0:30)[a]	15:18	(0:29)*
$\dot{V}O_2$	3.64	(0.08)	3.53	(0.06)	3.73	(0.07)
$\dot{V}_E$	111.28	(5.27)	128.22	(3.44)	134.30	(6.70)*
HR	176.0	(2.0)	176.0	(2.0)	177.0	(2.0)
RER	0.96	(0.01)[b]	1.09	(0.02)[b]	1.06	(0.01)*
20km						
Time	34:22	(0:45)[c]	32:29	(1:02)[c]	31:38	(0:56)*
$\dot{V}O_2$	3.54	(0.13)	3.50	(0.08)	3.59	(0.09)
$\dot{V}_E$	104.94	(5.40)	125.60	(5.32)	129.20	(5.62)*
HR	176.0	(2.0)	176.0	(2.0)	177.0	(2.0)
RER	0.95	(0.01)[d]	1.04	(0.01)[d]	1.05	(0.02)*
30km						
Time	53:14	(1:30)[e]	49:60	(1:39)[e]	48:05	(1:25)*
$\dot{V}O_2$	3.41	(0.15)	3.38	(0.10)	3.55	(0.07)
$\dot{V}_E$	98.95	(6.20)	118.28	(4.63)	128.30	(4.76)*
HR	172.0	(2.0)	173.0	(2.0)	176.0	(2.0)
RER	0.94	(0.01)[f]	1.05	(0.02)[f]	1.06	(0.02)*
40km						
Time	76:39	(2:23)[g]	66:42	(2:16)[g]	64:08	(1:49)*
$\dot{V}O_2$	3.44	(0.16)	3.47	(0.12)	3.58	(0.08)
$\dot{V}_E$	101.08	(7.13)[h]	124.80	(5.48)[h]	128.70	(3.72)*
HR	172.0	(3.0)	175.0	(3.0)	179.0	(2.0)
RER	0.94	(0.01)[i]	1.07	(0.01)[i]	1.05	(0.02)*

Values are Mean ± (SE)

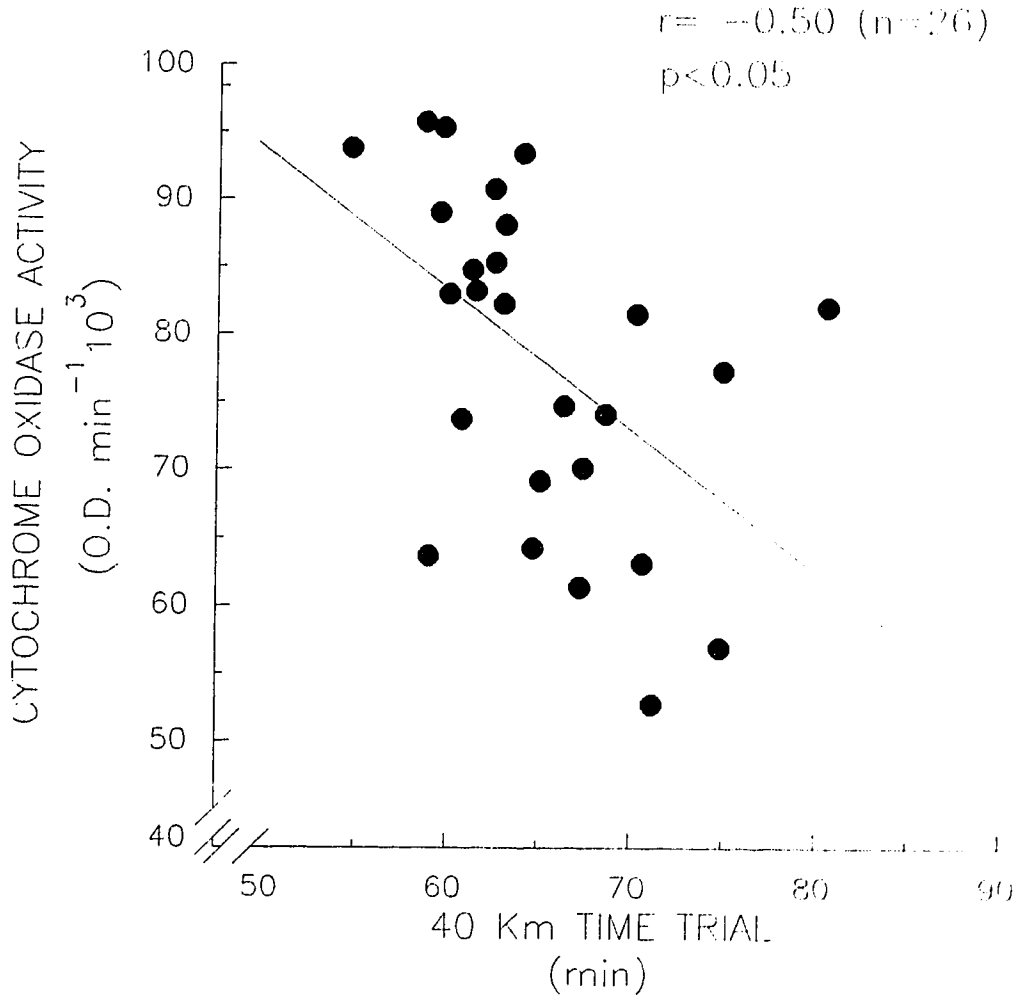
Paired letters (a,a; b,b;.. i,i) indicate significant differences at $p<0.05$.

* significantly different from pre-training ($p<0.05$).

Appendix D.5: Relationship between power output (Watts) at the ventilation threshold versus time (min) on the post-taper simulated 40 km time trial.



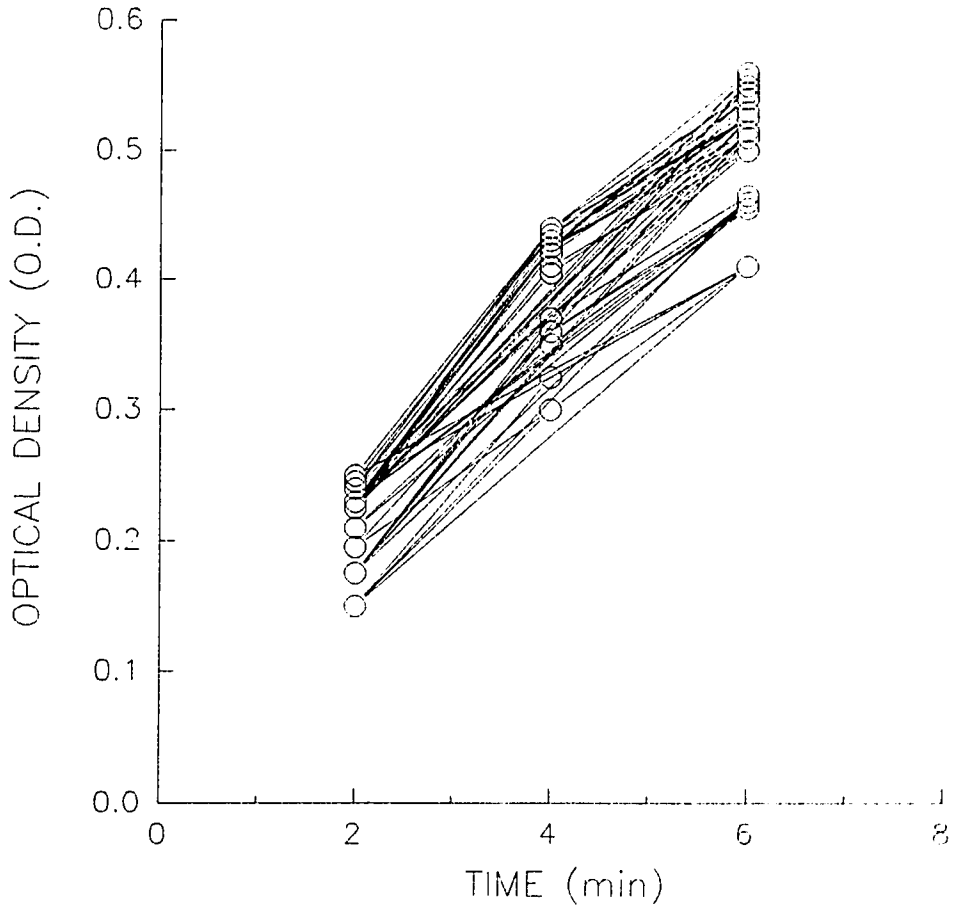
Appendix D.6: Relationship between the post training cytochrome oxidase activity (O.D. min^{-1} 10^{3}) and performance time during the post-taper simulated 40 km time trial.



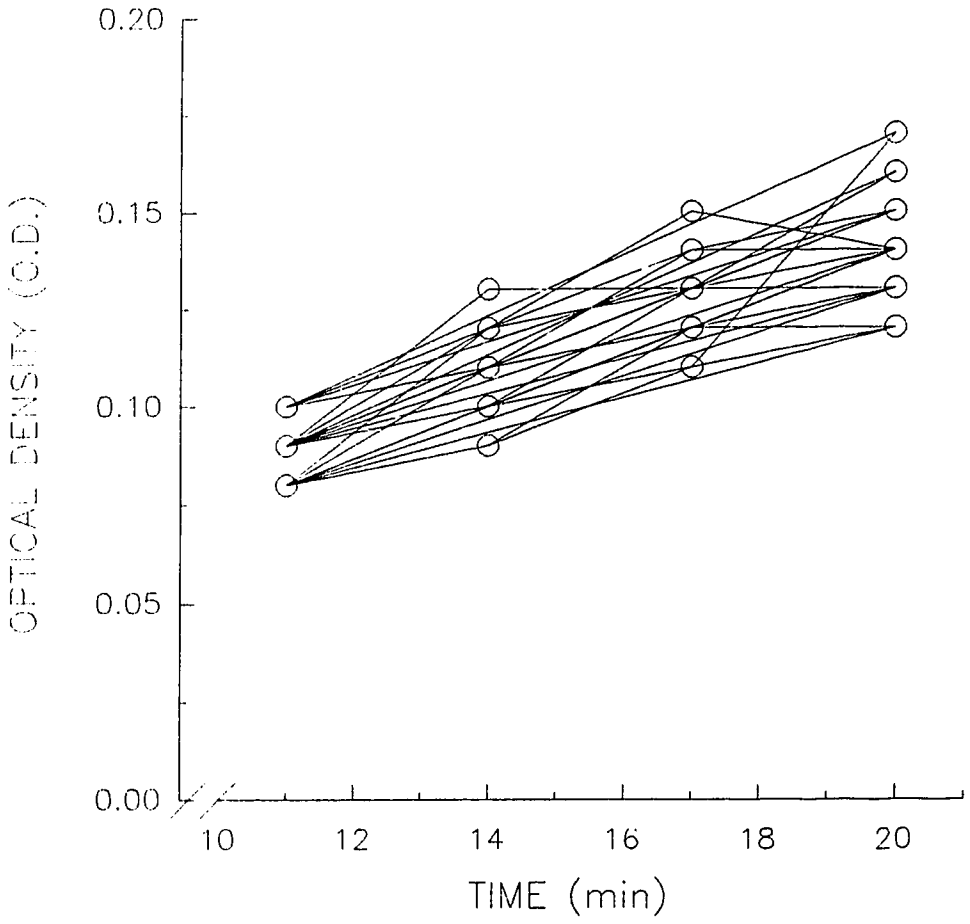
APPENDIX E

STEADY STATE ENZYME CONDITIONS

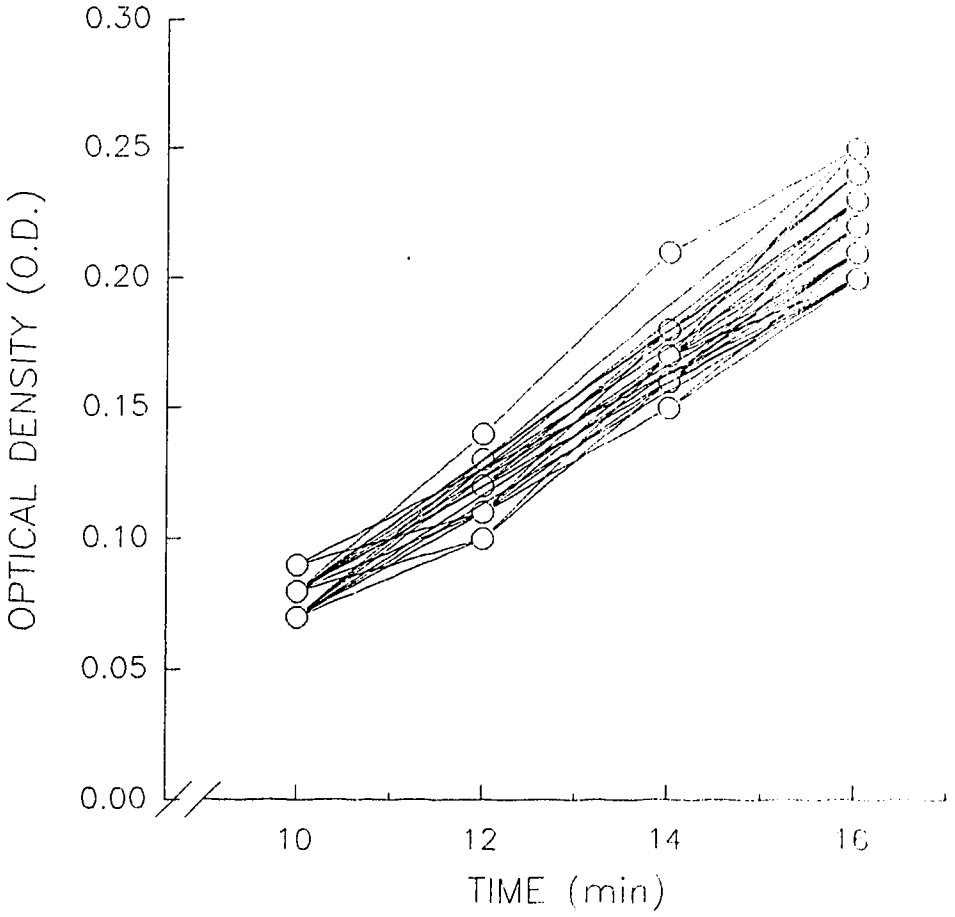
Appendix E.1: Steady state linearity for myofibrillar adenosine triphosphatase (mATP) activity in human single muscle fibres (n=25).



Appendix E.2: Steady state linearity for β-hydroxyacyl CoA dehydrogenase activity (HOAD) in human single muscle fibres (n=22).



Appendix E.3: Steady state linearity for cytochrome oxidase activity (CYTOX) in human single muscle fibres (n=25).



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British Columbia Forest Products Bursary Award, 1981

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1. Neary, J.P. **Cycling for pleasure and competition: training methods to improve your fitness.** Pulse, Publication of Sports Medicine Council of Alberta, 4(1):6-7, 1990.

2. Neary, J.P. **Taper Principle.** Alberta Bicycle Association, September 1989.

3. Neary, J.P. **Hydration and its importance during exercise.** Alberta Triathlon Association, March 1988.

4. Neary, J.P. **Putting the wind back into your sail with fitness: An off-season training programme to help you sail better.** Sailing Canada Magazine; Monthly Health & Fitness editorial, March - December 1987.