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OPTIMIZING PERFORMANCE IN TRIATHLETES

by

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B.Sc., McGill University, 1992

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SCIENCE

in the
School of Kinesiology

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OPTIMIZING PERFORMANCE IN TRIATHLETES

Author: PETER ZARKADAS

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SEPTEMBER 6, 1994  (date)
ABSTRACT

The purpose of this study was to determine the nature of taper required to optimize performance in Ironman triathletes and to monitor changes in physiological parameters that accompany this peaking phase. Eleven triathletes (26 ± 4 yrs, 77.0 ± 6.5 kg) took part in 3 months of training interspersed with two taper periods, one of 10 days (Taper 1) preceding a mid-season short course triathlon and another several weeks later for 13 days (Taper 2) preceding an Ironman competition. There was no change in intensity of each work-out between training and Taper 1 and Taper 2, although training frequency was reduced from 6 to 4 days per week in each case. Performance ability was measured serially from an all-out 5 km standard run, and from an incremental cycle ergometer test (30 W/min) to exhaustion respectively each week throughout the study. Pulmonary gas exchange and ventilation were computed breath-by-breath on three separate ergometer tests, two weeks prior to taper and during each week of Taper 2. Separate groups of the whole cohort peaked differently during each of the taper periods.

Reducing training volume by 50% in an exponential fashion (\( \tau \leq 5 \) days) in one group of triathletes during Taper 1 resulted in a 46 second (4%) improvement in their 5 km criterion run time and a 23 W (5%) increase in maximal ramp power output. A 30% step reduction in training volume in the second group did not result in any significant improvement in physical performance on the same measures. Training volume was reduced exponentially in both a high volume group (\( \tau \geq 8 \) days) and a low volume group (\( \tau \leq 4 \) days) during Taper 2. Criterion run time improved significantly by 74 seconds (6%) and 28 seconds (2%) in the high and low volume groups respectively, while maximal ramp power increased significantly by 34 W (8%) only in the low volume taper group. Maximal oxygen uptake increased
progressively from 62.9 ± 5.8 ml·kg⁻¹·min⁻¹ two weeks prior to taper, to a significantly higher level of 68.9 ± 4.2 ml·kg⁻¹·min⁻¹ during the final week of Taper 2 ( p ≤ 0.05 ). The anaerobic threshold determined by a non-invasive method (Whipp et al., 1989) was also observed to increase from 70.9 % to 74.9 % of a subject's maximal oxygen uptake during Taper 2.

These results demonstrate that proper placement of training volume during taper is a key factor in optimizing performance for a specific competition. A high volume of training in the immediate days preceding an event may be detrimental to physical performance and a well designed taper allows recovery from heavy training. This was evidenced in the present study by an improvement of physical performance in both a standard criterion run and an incremental cycle ergometer test to exhaustion, and physiologically by an increase in aerobic power and anaerobic threshold respectively.
DEDICATION

This thesis is dedicated to the triathletes who participated in this investigation who repeatedly volunteered their time, blood, and maximal physical effort to further advance our understanding of training and its effect on performance. The mental and physical demands of this study were enormous and often required a subject to push himself beyond his normal threshold and explore the upper limits of physical capability. The two triathlon competitions required for participation in this study are considered among the most grueling of endurance events, while the weekly performance measures and long hours of training were very demanding. The positive results observed in this thesis are a reflection of the commitment and dedication of these triathletes to whom I am very grateful.
ACKNOWLEDGMENTS

There are several individuals that deserve acknowledgment and thanks for their contribution towards the successful completion of this research. First of all I would like to thank Dr. Eric Banister for introducing me to the fascinating study of athletic training and performance and the benefit of modeling performance from training. His enthusiasm and expertise in the field of exercise physiology and human training research is quite obvious; an interest we definitely have in common. His expert guidance and impeccable editorial skills lead me to strive for greater quality and precision in both my research and writing. I would also like to acknowledge my fellow colleague and subject Jim Carter who provided me with valuable guidance in setting up this experiment and insight into the inherent practical and technical difficulties of field study. Dr. Mike Walsh is also recognized for his set up of the breath by breath analysis, and his expert technical assistance in teaching me how to perform a $\dot{V}O_2\text{max}$ test on a subject. I would also like to thank Tony Jordan for his generosity and recognition of this study by supplying free Cliff Bars and Fruit Source to the triathletes throughout its duration. This provided the triathletes with an excellent fuel supplement during training and competition and was a great incentive towards their compliance in the study. I would like to thank Ruth Westdal for her patience, understanding, and support during this degree. Finally I would also like to acknowledge my parents for their continued support of both my academic and athletic pursuits especially during my graduate work.
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INTRODUCTION

It is common practice among endurance athletes to focus upon optimizing their performance for a single competition. The complex interaction of several factors including training, diet, psychology and skill contribute to the realization of optimal athletic performance (Banister et al., 1986). Each factor's individual contribution to the final performance is often difficult, if not impossible, to isolate and acknowledge separately. It may be for this reason that an athlete often relies on previous training practice, or even day to day subjective feelings to prepare for competition. Inevitably this leads to sub-standard performance. A more precise and sophisticated approach to understanding the inherent complexity of training and its effect on performance was first introduced in a human systems model by Calvert et al. in 1976. The mathematical model related performance directly to training. Other intangible factors such as skill and psychology, whose effect on performance is often difficult to quantify were considered constant, with training the main stimulus for improving performance. By monitoring training quantitatively and modeling its effect on actual performance in a dose-response manner, insight into the training process may be gained such that an optimal training regime may be developed.

In the above mathematical model, the athlete is considered a black box controlled by two antagonistic systems associated with a two component first order transfer function designated as fitness and fatigue. Predicted performance in the system is the difference between fitness and fatigue at any point in time. The parameter vector of the model is established by computer iteration of the model parameters \((\tau_1, \tau_2, k_1, k_2)\) matching performance predicted from training against real performance to achieve a best fit. When good coincidence between actual and modeled performance occurs, the
transfer functions of fitness and fatigue may also be real. Application of this model has been successful in training swimmers (Calvert et al., 1976), runners (Morton et al., 1990), elite weight lifters (Busso et al., 1992), and most recently endurance triathletes (Banister, Abstract 1992, Zarkadas et al. Abstract, 1994).

A development of the model theory described as the Influence Curve (Fitz-Clarke et al., 1991) provides specific insight into the time course and intensity of preparation for a particular event. The Influence Curve dictates where the maximum density of training should fall and when training should cease, depending on the athlete's modeled parameters, in order to peak for a specific competition. The mathematics of the Influence Curve prescribe absolute rest of approximately 16 days prior to competition, as necessary to ensure optimal performance from accumulated training although in practice this is never done. The generally negative effect of detraining on performance, however, has been previously reported (Coyle et al., 1985, Houmard et al., 1992). If an individual athlete's modeled parameters undergo change during the course of a training season or in the final peaking period, this may preclude complete rest as an appropriate form of peaking and some training may be in fact beneficial during the taper phase.

One alternative to absolute rest immediately prior to competition is to reduce training volume in an exponential fashion (taper). This technique offers the athlete the opportunity to maintain the majority of the fitness accumulated through training while minimizing fatigue concomitantly accumulated so that performance may be optimized (Shepley et al., 1992). This method of preparation has received considerable attention recently especially among endurance athletes (Banister et al., 1992, Houmard and Johns, 1994, Houmard et al., 1994). In this system of athletic preparation, overtraining which is known to have a negative effect on performance
(Costill et al., 1991) may be avoided. Another similar method of preparation for competition involves a simple step reduction in training volume (McConnell et al., 1993), but the effectiveness of this method of peaking is unclear.

Training

Physical training may be defined as a repeated quantity or dose of exercise, which if sufficiently large, may stimulate an adaptive response in the human system proportional to the level of training stress imposed. Evidence suggests a period within which training is progressively reduced or tapered, is beneficial prior to a competitive event in order to allow recovery from preceding heavy training (Shepley et al., 1992, Houmard and Johns, 1994, Houmard et al., 1994). However, the precise balance among training volume, intensity, frequency and duration of taper remains elusive. Houmard and Johns (1994) suggested a taper protocol is advantageous compared with a standard step reduction in training volume. However, no study to date has attempted to compare these two peaking methods. If the quantity of training is reduced during the "off" phase of preparation so that it approaches complete training cessation, too much taper will result, and a decrement in physical performance below that expected from the extent of training will result (Coyle et al. 1984, 1986). In contrast, if the quantity of training is too large during the "on" phase of training, either because of excessively intense training or due to too frequently repeated training sessions at low intensity, overtraining may result. Overtraining is characterized by chronically depressed performance yet no single physiological or biochemical marker has been found to diagnose an overtrained athlete (Lehmann et al., 1992, 1993). The optimal combination of sufficiently intense exercise during training and appropriate recovery during taper requires further investigation.
Objective

The objective of this thesis was to train triathletes systematically so that they would achieve their optimal performance in the 1993 Canadian Ironman Triathlon. This was achieved by closely monitoring each subjects' training and performance during the course of a 3 month study, modeling the training as described above, to determine the nature and time course of cycles of training and tapering required to peak the athlete for the race. The interaction of several factors combine to produce optimal athletic performance and the following literature review summarizes current knowledge on the negative and positive contribution of training to performance. Also reviewed is the change in certain biochemical and hematological measures that result from heavy training. The current systems model of training will also be more extensively described.

I. TRAINING

TRIATHLON

Brief History

The triathlon is a sport that requires an athlete to perform three consecutive events in a race which includes swimming, biking, and running. The first triathlon competition dates from 1974 in the United States and the sport has greatly increased in popularity in the intervening period. Famous races such as the Hawaiian Ironman, the Nice Triathlon, and the proposed inclusion of a standardized distance short-course triathlon (1.5 km swim, 40 km bike, 10 km run) as an Olympic Sport in 1996 have contributed to the growth and success of this new sport. The extreme Ironman form of the competition has increased in number from the single Hawaii Ironman Triathlon in 1978 to the present total of six Ironman competitions worldwide. The Ironman triathlon comprises a 3.9 km swim, a 180.2 km bike, and a 42.2 km run. It is one of the most demanding endurance events. The level of competition has continuously increased as
may be judged from the improvement in finishing time of 11 hr 16 min in the 1978 Hawaii Ironman to the 8 hr 7 min 45 sec for the same race established in 1993. This increase in performance may be attributed in part to advanced technical equipment, but to a greater degree it has depended on advances made in training technique and nutrition of the athlete.

Training and Preparation

Preparation for an endurance event such as an Ironman triathlon requires a multi-faceted approach to training. An athlete must incorporate scientific principles of physiology, biomechanics, nutrition, and psychology in his/her training to achieve optimal results. Several books have been published on the topic of triathlon training, often authored by elite triathletes who prescribe their own subjective training regime with little or no knowledge of, or reference to, current scientific research on the topic (Sisson, 1983, Allen, 1988). Recent publications have described triathlon training in a more precise scientific manner (Cedaro, 1993, Town and Kearney, 1994). Precise exercise prescription, however, is highly individual and without serial measurement of performance as feed-back information, an athlete is unlikely ever to know whether a training program is optimal or demanding and whether he/she should train harder or reduce the training load. Training volume varies greatly in Ironman triathletes, varying anywhere from 14 to as much as 40 hrs/wk often at an unknown heart rate and metabolic intensity in the extreme case of preparation. Optimal performance at race time is difficult to achieve without better evaluation of the daily training dose and an objective training plan.
Review of the Literature

The topic of triathlon training and competition has received considerable attention in the literature with regard to the metabolic changes effected in a competitor during a competition (Harrison et al., 1991), the physiological characteristics needed by the athlete (O'toole et al., 1988, Laurenson et al., 1993) and the type of training necessary to develop these characteristics. Not until recently, however, has any scientific attempt been made to train a triathlete purposefully for such a race (Banister, Abstract 1992). A few studies have attempted to predict performance during a short-course triathlon by simple correlation of race results with selected physiological variables. Sleivert et al. (1993) found in one study of 25 triathletes that the only significant predictor of overall triathlon performance was running velocity at ventilatory threshold. Another study by Butts et al. (1991) was able to correlate either an absolute or relative attained $\dot{VO}_{2}\text{max}$ in a competitor with performance during each segment of a short-course triathlon. Although these studies provide a degree of prediction of performance during a short-course triathlon given a athlete's physiological profile, they do not necessarily reflect what may be the determining factor for success during an Ironman competition where such factors as climatic conditions, dehydration, and mental status often determine the final outcome. Furthermore, the above studies provide no insight into the training process or how to improve performance with training.

The application of systems theory to model the effects of physical training on performance in triathletes was first conducted by Banister (Abstract, 1992) in the study of endurance triathletes preparing for an Ironman competition. In this study, triathletes were systematically trained according to a vigorous protocol of repeated blocks of heavy training each followed by a regeneration or peaking period. Performance was serially measured throughout this investigation on both a cycle ergometer and an all-
out criterion run and modeled according to a modified version of the original systems model of training termed the Influence Curve. The results of this investigation were convincing in that all four triathletes performed a personal best time during the final Ironman competition and during an interim trial peaking period which consistently showed an improvement in criterion performances as well as during a final 2 week period leading to the actual Ironman. The present thesis used a similar training protocol to model the effect of training on performance especially during the critical peaking period where training duration, but not intensity, is reduced and performance improves.

DETRAINING

Definition

Detraining is defined as the loss of training adaptation as a result of training cessation.

Physiological Decrement

Physiological adaptation to exercise is unfortunately not permanent, and a significant decline in both cardiovascular and muscle metabolic potential occurs in the days or weeks after cessation of training. The consequences of detraining were first studied in athletes who underwent 20 days of best rest (Saltin et al., 1968). This protocol caused a large 28% decrease in $\dot{V}O_2max$ during the time period, primarily due to a 29% decrease in stroke volume. Enforced bed rest is an extreme case of immobility, and the observed decline in $\dot{V}O_2max$ may not be applicable to detraining in athletes, since bed rest is quite different even from the inactivity experienced in a normal sedentary life-style.

Decrement in cardiovascular function with detraining is manifested by a decrease in $\dot{V}O_2max$ (Coyle et al., 1986, Ehsani, 1992), cardiac output (Coyle et al., 1984),
plasma volume (Houmard et al., 1992), (a - v)DO₂ (Coyle et al., 1984), and cardiac hypertrophy (Ehsani et al., 1978). The time course of loss of adaptation accompanying removal of the exercise stimulus is very rapid. Cullinane et al. (1986) found a 5% decrease in plasma volume within 2 days after exercise cessation. Ehsani et al. (1978) reported an exponential decline in left ventricular wall thickness with a half life of 5 days, and a reduction of left ventricular diameter to a pre-exercise value complete after 7 days of inactivity. The latter may be due in part to the reduction in plasma volume, and reduced preload. Curiously, a decrease in physical performance does not always accompany loss of metabolic power (Hickson et al., 1982).

The decrease in plasma volume observed with training cessation may account for some of the immediate change in cardiovascular function. The relative decrease in plasma volume calculated from hematocrit and hemoglobin concentration has been shown to range from 5% (Cullinane et al., 1986) to 9% (Coyle et al., 1984) when training ceases. It is well documented (Convertino, 1983) that endurance training produces a relative hypervolemia compared with the untrained state. Hypervolemia enhances both cardiovascular function by providing additional plasma volume for increased left ventricular filling and an increased stroke volume (Coyle et al., 1984), as well as an increased preload to stimulate cardiac hypertrophy (Ehsani, 1992). Hypervolemia may also enhance thermoregulatory adaptation by providing extra cellular water for sweating (Convertino, 1983).

Coyle and co-workers (1984) tested 7 endurance athletes 12, 21, 56, and 84 days after training cessation and found a 7% decline in VO₂max within 12 days of detraining, which persisted unchanged until day 21. VO₂max decreased a further 14% by day 56 and remained unchanged until day 84. The immediate decline in VO₂max preceding day 21 was attributed to a decrease in stroke volume and any further
decrease was attributed to a decline in the (a - v) DO$_2$. A decrease in plasma volume (although not measured) was thought responsible for an 8% decrease in cardiac output during the period up to 21 days. A decrease in plasma volume may be a mechanism responsible for a relative reduction in cardiovascular function. The decline in stroke volume with exercise cessation may be attenuated or partially reversed by infusing a 6% dextran solution in saline and expanding plasma volume (Coyle et al., 1986). Further reduction of VO$_2$max after day 21 may be due in part to a decrease in the number and size of mitochondria, evidenced by a large and rapid decrease in muscle mitochondria enzyme level (Coyle et al., 1986).

Exercise cessation has also been shown to cause a reduction in total myoglobin and hemoglobin content, skeletal muscle capillarization, and reduced vascular conductance (Neufer, 1989). In one study by Cullinane et al., (1986), 15 subjects trained by running 16 km/d for 2 weeks, followed by 10 days of rest. No change was observed in VO$_2$max or cardiac dimension for 10 days following exercise cessation. A rapid decline in plasma volume was accompanied by an increased maximal heart rate which might be an adaptation to preserve cardiac output. Maintenance of VO$_2$max and cardiac size may have been due in part to the rigorous training protocol preceding the rest period. The adaptation process may have been offset by the build up of fatigue and the time required for adaptation to occur. This study is limited because the effect of detraining on performance was not measured, and therefore no conclusion could be made regarding the loss of physiological adaptation on performance.

Performance Decrement

Reduced performance as determined from maximal and sub-maximal tests occurs within weeks of cessation of training (Neufer, 1989). Houston et al., (1979) observed
that 2 weeks of inactivity in a group of well trained runners reduced their submaximal run time (90% \( \dot{V}O_2 \text{max} \)) by 25%, accompanied only by a 4% decrease in \( \dot{V}O_2 \text{max} \). The observed decline in performance was therefore ascribed to a reduced capacity of the oxidative enzymes. Houmard et al., (1992) measured a 9% decrement in the time to exhaustion in a running test after 14 days of inactivity and a similar 4% decrease in \( \dot{V}O_2 \text{max} \). It is likely that other factors besides a decrease in \( \dot{V}O_2 \text{max} \) act to produce a decrease in physical performance such as an anticipated much larger lactate response to submaximal exercise (Coyle et al., 1985), or possibly a decrease in running economy (Noakes et al., 1988).

More recently, Madsen et al., (1993) detrained 9 well trained athletes for 4 weeks and found a large 21% decrease in aerobic power on a cycle ergometer without affecting \( \dot{V}O_2 \text{max} \). Endurance performance must be related to other factors other than \( \dot{V}O_2 \text{max} \), most likely to peripheral factors in the exercising muscles. Altered substrate utilization or change in electrolyte regulation was proposed as a mechanism for reduced aerobic power. There is, however, a lack of clear documentation on the time course of any performance increment or decrement in the immediate period following exercise cessation. Although the above studies found a decrement in performance after 2-4 weeks of detraining, it is very likely that performance (if measured) would have improved immediately following exercise cessation which may have implication for the type of taper necessary for improving performance.

Recent studies support the idea that even a short period of detraining may not alleviate the detriment to performance induced by heavy training and a longer period of additional training may required to recover (Neufer et al., 1987, Houmard, et al., 1992). It may be that some caution should be exercised in prescribing rest from training greater
than a few days necessary to recover from heavy training or injury. The majority of
detraining studies, however, have not systematically measured or accurately
documented the time course of change in physical performance with detraining.

TAPER

Definition

Taper is a highly specialized form of reduced training that decays exponentially
in a systematic non-linear fashion (Houmard, 1991). Reduced training and taper differ
in that reduced training refers to a set decrease in training volume.

Taper and Performance

Both coach and athlete fear a loss of physical conditioning and a decrement in
performance which might accompany a reduced training schedule for several days or
longer (Houmard, 1991). For this reason most athletes prefer to maintain training volume
elevated until the time of competition. This belief is largely unfounded (Banister et
al., 1991). Recent evidence has demonstrated that much of the performance decrement
and loss of training adaptation that occurs with detraining may be minimized, if training
is simply maintained at a reduced level (Hickson et al., 1982) or tapered (Houmard et
al., 1994). Tapering is a specialized training technique to decrease the fatigue of
heavy training, without a loss in training adaptation. This procedure has been studied
recently in swimmers (Johns et al., 1992), cyclists (Neary et al., 1992), and endurance
runners (Shepley et al., 1992, Houmard et al., 1994) with promising results.

There appears to be an advantage with the use of a taper protocol compared
with a standard step reduction in training volume. In one study, a standard 70% redunction
of normal training volume for 3 weeks did not significantly improve a 5 km
run performance or muscular power in distance runners (Houmard et al., 1990). In
contrast, a 7 day taper with an 85% reduction in weekly training volume improved a 5 km race time and increased muscular power (Shepley et al., 1992, Houmard et al., 1994). These results suggest that a taper protocol, where training volume is reduced progressively to an extremely low amount, may improve performance more than a standard single reduction in training volume, which only appears to maintain performance (Houmard et al., 1990). The different peaking protocols and often inappropriate and inadequate measures of improved performance used in each of these latter investigations, however, allows no firm conclusion about a preferred method of peaking.

Remarkably little is known about the physiological changes that result from tapering (Shepley et al., 1992). The majority of taper and reduced training studies have focused on performance events lasting from 1 to 20 minutes. Only recently did Banister (Abstract, 1992) apply a reduced training program in 4 triathletes preceding an Ironman triathlon, and demonstrated a large improvement in performance shown by a reduction in criterion 5 km run time and maximal power output on a cycle ergometer. However, no study has attempted to construct a taper program with an appropriate proportion of intensity and volume for an individual athlete to optimize his/her performance for an endurance race. Furthermore, no study has ever attempted to compare the effect of a reduced training versus taper protocol on performance.

Reduced training and tapering both may have the potential to reduce the obvious long term negative consequence of detraining and improve performance by arranging the volume and intensity of immediate pre-competition training following a period of intense training (Houmard and Johns, 1994). The intensity of exercise throughout a pre-competition tapering period plays an important role in providing an adequate stimulus so that training adaptation is not lost. A well designed taper has the...
potential to optimize performance during the critical time before competition and reduce the accumulated fatigue incurred from intense training. Scientific rationalization of the shape and intensity of training in the immediate pre-competition period in order to produce optimal performance has been attempted only theoretically by Fitz-Clarke et al., (1991) and empirically found still somewhat wanting.

**Components of Taper**

Variables which may be manipulated during a taper regime include training frequency, duration, and intensity of exercise, as well as the length of the taper itself. The response of an athlete to taper following heavy training is also highly individualistic and each athlete has their own inherent plasticity of response to removal of a training stimulus (Banister and Fitz-Clarke, 1993). The most precise method of prescribing a taper program requires an athlete to undergo a prior experimental taper, results of which may be used to manipulate each component of the taper to be tested at a future date. Obviously the number of parameters that can be manipulated in designing a taper are many and it is impossible to test each permutation and combination experimentally. However, scientific investigation of taper in endurance athletes has revealed several common characteristics which provides some insight into what comprises a successful taper (Houmard and Johns, 1994).

**Length of Taper**

One very important variable which can be altered is the time of onset of taper prior to competition. It is common practice among endurance athletes to taper between 7 and 21 days preceding a major competition (Houmard and Johns, 1994). One study by Yamamoto et al., (1988) compared the effect of a 45 and a 15 day taper on performance in swimmers and on hemoglobin concentration and hematocrit number. Peak performance was achieved after only 7 days of taper and the authors concluded
that 7 days was an optimal taper duration, prolongation beyond which might result in a less than optimal performance. Application of a swim taper schedule to a weight bearing exercise such as running may have limited applicability since recovery from muscle disruption due to heavy run training may require a longer regeneration period (Banister and Fitz-Clarke, 1993). Studies examining the effect of a 7 day taper on running performance have shown significant improvement of the order of 3 % during a 5 km race (Houmard et al., 1994) and as much as 22 % during a treadmill run to fatigue at a set speed (Shepley et al., 1992). It appears from these results that a taper less than 7 days is insufficiently long to optimize performance, whereas a taper lasting more than 21 days might result in a less than optimal performance continuing for a prolonged period since the taper now constitutes training at a lower level of intensity.

**Training Volume**

Training volume in this thesis is defined as the integral of a training impulse during a given period of time. The training impulse is the defined daily product of the duration and intensity of daily training given by the following equation:

\[ w(t) = D \cdot \Delta HR \text{ Ratio} \cdot Y \]

Training volume given by the following equation:

\[ V = \int_0^t w(t) \, dt \]

\[ = \int_0^t [ D \cdot \Delta HR \text{ ratio} \cdot Y ] \, dt \]

where

- \( V \) = volume of training
- \( w(t) \) = training impulse for one day
- \( D \) = duration of training
- \( \Delta HR \text{ ratio} \) = delta heart rate ratio
- \( Y \) = intensity multiplying factor
Training volume may be progressively reduced in a stepwise fashion (Houmard and Johns, 1994) or exponentially (Zarkadas et al., Abstract 1994) from 50% to 90% of a previous training volume. In the present thesis training volume was reduced by decreasing the duration of training alone while training intensity was not altered. This reduction is accompanied by a significant performance improvement. One very precise and simple way of describing an exponential reduction in training volume is by the characteristic value called the time constant \( \tau \) [Banister et al., 1986]. This value represents the time it takes for decay in a particular attribute (here training volume) to decline to 0.63 its initial value from the onset of peaking. This method of defining a particular prescribed taper will be used in this thesis.

The positive effect of taper on performance is thought to be primarily mediated by recovery from the accumulated fatigue of repeated days of intense training (Neufer 1989). Recuperation will only be observed, if training volume is substantially decreased. Shepley et al. (1992) tested 9 male distance runners after 8 weeks of intense training (80 km/wk, 2/3 continuous running and 1/3 intervals) and randomly assigned 3 subjects each to three 7 day tapers. The three taper protocols consisted of a high-intensity low-volume, low-intensity high-volume, and rest only taper. Run time to fatigue was measured before and after each taper on a treadmill at a velocity equivalent to each subject's best 1500-m time. Run time to fatigue increased by 22% when training volume was reduced by 90% and was not significantly different when training volume was reduced by 62% or rest only taper. This study is limited in that only three subjects were studied in each group, additionally run time to fatigue on a treadmill is not an accurate criterion performance measure. It would have been preferable, if the subjects had run all out on a 1500 m run. It is unclear in this study if reducing training duration or increasing training intensity was responsible for improving performance.
since both duration and intensity were manipulated without appropriate controls. These results, however, do suggest that a substantial reduction in weekly training duration is needed during taper to improve performance markedly. In this way the fatigue of heavy training is reduced and although fitness must also decline due to the relative non-training regimen of taper, but it decays much more slowly and is therefore substantially retained.

The frequency of exercise in this thesis is defined as the number of days an athlete trains each week and reduction of training duration with taper cannot be achieved solely at the expense of a drastic reduction in training frequency. One study by Neufer et al., (1987) examined the effect of reducing weekly training duration by 80% with a 50% reduction in training frequency in one group of swimmers, compared with a second group who reduced their weekly training duration by 95% with an 85% reduction in training frequency. Swim power decreased by the end of the first week of reduced training and continued to decrease after 4 weeks in both groups. Although training was not tapered in this study, the reduction in weekly training duration (80 to 95%) was similar to reduction taper studies in which performance measures were in fact improved (Shepley et al., 1992, Houmard et al., 1994). In the latter studies in which performance was improved, weekly training frequency was reduced by 20 to 50% compared with a 50 to 85% reduction in weekly training frequency where performance was impaired (Neufer et al., 1987). Houmard and Johns, (1994) concluded that weekly training frequency should not be reduced by more than 50% and even by as little as 20% in order not to lose any performance related adaptation.
Intensity of Exercise during Taper

A third common feature of a successful taper is the prevalence of intense exercise. Often exercise during taper is in the form of intense interval training (Neary et al., 1989), or a repeated criterion performance (Banister, 1992) with sufficient recovery between bouts of training to ensure an all-out effort. Reducing training by reducing the intensity of exercise (≤ 70 % \( \dot{V}O_2\max \)) has been shown either to maintain (Houmard et al., 1989) or actually impair running performance (McConnell et al., 1993). When sessional training intensity was increased during taper to ≥ 90 % \( \dot{V}O_2\max \) concomitantly with a sharp reduction in weekly training duration, running performance improved (Shepley et al., 1992, Houmard et al., 1994). Intense exercise may be required to maintain both the cardiorespiratory and muscular training induced adaptation as training is reduced during the taper period. Furthermore, training duration and intensity seem inversely related such that the degree by which training duration is reduced during taper will determine how much training intensity may be increased. Fine tuning of the interplay between intensity and volume requires further investigation.

Modeling Taper

Systems modeling of training provides valuable insight into the training process and can determine the time course and nature of taper required to optimize performance at some future point in time (Fitz-Clarke et al., 1991, Morton et al., 1990). Simulation results, using default parameters of a training model, suggest that current forms of taper may be too short in length and too physically demanding to achieve an optimal result from the training accumulated in a given period. The Influence Curve of Fitz-Clarke et al., (1991) dictates where the maximum density of training should fall and when training should cease, depending on the athlete's modeled parameters, in order to
peak for a specific competition. The modeled Influence Curve prescribes absolute rest of approximately 16 days given the default parameters $\tau_1 = 45$, $\tau_2 = 15$ days, $k_1 = 1$ and $k_2 = 2$. The time constant $\tau_1$ determines the rate at which modeled fitness decays and $\tau_2$ determines the rate at which fatigue decays. The multiplying factor $k_1$ determines the growth of fitness and the multiplying factor $k_2$ determines how much fatigue will grow. This duration of taper agrees with available scientific evidence discussed above, however, the rapid loss of training-associated adaptations with total training cessation is well documented (Neufer, 1989, Houmard, 1991).

Iterative modeling has demonstrated that model constants need to be reset every 60 to 90 days (Banister, 1991). Preliminary results of a pilot study re-evaluating data of triathletes (Banister, 1992), has demonstrated that the process of resetting model constants may be repeated more frequently during heavy training and taper to account for ongoing adaptation of the system. Results have shown that with heavy training $\tau_1$ may increase from 45 to 47 days and $\tau_2$ may decrease from 15 to 13 days within a period of only 30 days of training to achieve a better fit between modeled and criterion performance. This finding has significant implication for the length and type of taper which should be implemented before competition to achieve optimal performance. Physiologically, the change in time constants implies that with intense training, the athlete may become resistant to losing accumulated fitness and become able to shed accumulated fatigue faster. This would be beneficial for the design of a taper program so that modeled fitness may be maintained with a moderate but intense training stimulus, while allowing modeled fatigue to decay maximally and allow for optimal performance at the time of competition.
Physiological Effects of Taper

Maximal Oxygen Uptake

Maximal oxygen uptake, the physiological variable most often chosen by the sport scientist as the characteristic of a good athlete, is the maximum amount of oxygen per minute which an individual can use from the air in the surrounding atmosphere (Banister, et al., 1986). Although it is well known \( \text{VO}_2\text{max} \) increases with training (Neufer et al., 1987) current studies of taper have demonstrated an improvement in physical performance without an increase in \( \text{VO}_2\text{max} \) (Sheply et al., 1992, Houmard et al., 1994). Improvement in performance is attributed to adaptation at the muscle level where it is recovery from the fatigue of heavy training rather than improvement in \( \text{VO}_2\text{max} \) which the determining factor. However, heavy training imposes a fatiguing stress on both the cardiorespiratory and peripheral muscular systems. If training is sufficiently intense and enduring enough to stimulate adaptation it is logically possible, in fact mandatory to observe a recovery of both muscular and aerobic power with taper. This latter effect has yet to been experimentally demonstrated, however.

Blood Measures

Plasma creatine kinase (CK) activity is hypothesized to be positively related to the degree of muscle disruption, resulting in "leaky" cell membranes causing a loss of muscle enzyme to the vascular space (Banister and Fitz-Clarke, 1993). Plasma CK activity is normally < 100 iU·L\(^{-1}\) in sedentary individuals and may increase during seasonal training to between 160 and 448 iU·L\(^{-1}\) and decrease during relative non-training or taper to between 80 and 191 iU·L\(^{-1}\) (Yamamoto et al., 1988). The implication of this reduction in plasma CK activity on athletic performance remains to be
determined. Morton et al. (1990) observed a decline in elevated serum enzyme activity (ESEA) by the 11th day of a standard training program even though training continued for a further 17 days. This decline continued and later stabilized at a constant elevated plateau which then declined quickly through the early days of taper to a base level.

**Reduced Training Studies**

Hickson et al. (1982), studied 12 runners who trained consistently for 6 weeks, and reduced their training for 15 weeks to 1/3 or 2/3 the intensity, maintaining the same weekly volume of training. The intense workout protocol consisted of interval training performed on a cycle ergometer at a work rate which approached a subject's VO\(_2\)max. VO\(_2\)max was not maintained during the 15 week period on the 1/3 reduction in intensity protocol, yet it remained elevated above a pre-trained level. A 2/3 reduction in intensity caused an even greater decline in VO\(_2\)max in the detraining period compared with the 1/3 reduced intensity group. The results from this experiment led the authors to conclude training intensity is an essential requirement for maintaining increased aerobic power throughout a period of relative detraining. It was also suggested that intensity must be maintained at a level no less than 70% VO\(_2\)max or even by as much as 90-100% VO\(_2\)max to allow a training induced increase in cardiovascular function to persist. However, no concomitant study of a criterion physical performance was made in the above study so that a primary correlate determining actual physical performance was not measured.

Numerous other studies have demonstrated that reduced training is able to maintain a training adaptation in variables such as VO\(_2\)max (Neufer et al., 1987), cardiac hypertrophy (Hickson et al., 1982), as well as physical performance during maximal
power and endurance tests (Houmard et al., 1990). These same studies, however, demonstrated that the training stimulus necessary for maintenance of some of the acquired physiological adaptation accompanying increased physical performance was insufficient. This was evidenced by a decrease in plasma volume, and muscular power decrement (Neufer et al., 1987) observed during reduced training. Houmard et al. (1990) trained 10 endurance athletes for 4 weeks. These subjects ran 81 km/week, 75% of the time at a moderate pace, and 25% of the time using intense interval training. This training regime was followed by 3 weeks of reduced training, when training duration was decreased by 70% and frequency by 17% while maintaining the same intensity. Time to exhaustion during a run performance increased by 9.5% by week 3 of detraining, and a criterion 5 km race time improved by 4-5 seconds after 2 weeks of reduced training. This suggests that endurance runners were not negatively affected and performance was enhanced by a three week reduction in training. Thus the statement that no significant difference in physical performance results from detraining seems incorrect measured by the absolute criterion of actual run time data. Serum testosterone and cortisol concentration, and serum CK activity were also analyzed in this same group of runners to determine, if reduced training caused blood variables to be re-established to a normal pre-trained level (Houmard et al., 1990). Serum CK activity was significantly reduced from its elevated level at the cessation of training, while both the serum testosterone and cortisol concentration remained unchanged from the training level after three weeks of reduced training. However, the serial time course of change in these blood parameters was not measured. The time course of serum CK activity throughout heavy training and peaking may be a good indicator of the relative change in training stress (Morton et al., 1990), and to the direct trauma to muscle produced by heavy training (Armstrong et al., 1990).
Skeletal muscle is composed of a highly ordered, structurally specialized cell type, capable of converting metabolic energy to mechanical movement and capable of extensive regeneration after injury (Hurme et al., 1991). Although very plastic in nature, intense physical exercise such as eccentric contraction or weight bearing exercise such as running may produce dramatic pathological change to cellular muscle morphology and function. This so called "muscle damage" is manifested by disruption of normal myofilament structure (Byrd, 1992, Carlson and Faulkner, 1983) and ultrastructure (Waterman-Storer, 1991), loss of intramuscular proteins (Mair et al., 1992), and alteration in the structure and function of the sarcolemma, sarcoplasmic reticulum and mitochondria (Byrd, 1992, Carlson and Faulkner, 1983). Recent evidence has demonstrated that in order to enhance muscular performance, the adaptive process must be stressed sufficiently such that there is a high degree of cellular catabolism (Waterman-Storer, 1991). Banister et al. (Abstract, 1990) suggested that the degree of so-called muscular injury required to maximize adaptation might even resemble traumatized muscle tissue from a direct blow. It is unlikely, however, that physical exercise ever results in such a degree of muscle trauma, except by accidental injury or during an intense or debilitating competitive event, where muscular fatigue and pain are ignored. Normally, during a bout of heavy training the protective pain mechanism of muscle will forewarn an athlete before muscular trauma occurs (Armstrong, 1984). Optimal training must therefore produce a sufficient degree of cellular catabolism such that adaptation is maximized without producing any long term muscular impairment.

**Regeneration of Damaged Muscle**

The sequence of events of muscle regeneration is well established and begins with acute disruption of muscle fiber, followed immediately by an intrinsic degeneration within
the fiber itself (Carlson and Faulkner, 1983, Hurme et al., 1991, 1992). The disruptive phase involves disintegration of the muscle cytoskeleton, and damage to neighboring mitochondria, sarcoplasmic reticulum and sarcolemma. With more severe disruption the basal lamina and plasma membrane can also be disturbed. The subsequent degeneration phase includes an autogenic period of proteolytic and lipolytic digestion of cell structure due to a loss of calcium homeostasis (Armstrong, 1990), followed by macrophagic digestion and removal of cell debris (Fisher et al., 1990). Phagocytic cells are transported by the local circulation and therefore depend on the integrity of the local capillary network. It is often necessary for the capillary network to grow into the damaged area before cell-mediated degeneration can take place, thus delaying the healing process. Removal of dead muscle debris by a macrophage from the site of trauma begins as soon as possible as it is a prerequisite for the activation of satellite cells. Satellite cells lying beneath the basal lamina are activated, begin to proliferate and give rise to myoblasts which later fuse inside basal lamina cylinders into a multinucleated myotubes and produce muscle specific proteins which develop into a mature muscle fiber.

Morphology of Disruption

Injury to muscle is common when the exercise is relatively intense, enduring, or includes eccentric contraction such as running. Damage is characterized by alteration of the A-band and Z-line where streaming, smearing and severe swelling are commonly observed (Byrd, 1992). Waterman-Storer et al. (1991) presented evidence supporting cytoskeletal disturbance as a major contributing factor to the observed change in muscle ultrastructure. The myofibrillar cytoskeleton is made up of two sets of filaments, the exosarcomeric cytoskeleton and the endosarcomeric cytoskeleton. The exosarcomeric cytoskeleton is composed of three proteins desmin, vimentin, and synemin collectively called intermediate
filaments. The intermediate filaments are positioned longitudinally from Z-disc to Z-disc and serve as attachment sites for mitochondria and the sarcolemma or are found transversely attaching adjacent myofibrils. The endosarcomeric cytoskeleton is made up of the molecular size proteins titan and nebulin which lie adjacent to actin and myosin within the sarcomere. During eccentric exercise the Z-disc to Z-disc connection made by the intermediate filaments are stretched or broken leading to Z-disc smearing. Disruption of the endoskeleton is likely due to the disruption or breaking of titan filaments leading to Z-disc streaming.

**Muscle Disruption and Physical Performance**

The degree of muscle catabolism necessary to achieve optimal adaptation without a long lasting detrimental effect on physical performance remains elusive. The time course of regeneration of muscle falls within the same 1-2 week peaking period associated with recovery from adaptive training (Armstrong, 1990). More severe muscle disruption, or possibly repetitive muscle disruption that accompanies very intense and enduring daily training may overload the adaptive process and regeneration may require a much longer period leading to an overtrained state. However, it would be incorrect to characterize muscle disruption as the primary protagonist of overtraining in the endurance athlete since many other factors are known to lead to chronic fatigue (Lehmann et al., 1993). However, since muscle disruption is consistently observed and a possible cause of reduced performance both during training and during a competitive event (Galun et al., 1988, Harrison et al., 1991), this suggests that a marker of muscle disruption such as ESEA may be in fact a valuable measurement for the diagnosis of incipient overtraining if it is measured serially throughout a training period.
III. ELEVATED SERUM ENZYME ACTIVITY

Prediction of Performance

Elevated serum enzyme activity (ESEA) has been used to predict physical performance (Galun et al., 1988), assess the degree of muscle disruption (Apple and Rhodes, 1988), and as a possible marker of overtraining (Lehmann et al., 1993). Muscle enzyme leakage which occurs as a consequence of intense training, has been related to a temporary decline in muscular function and aerobic power (Galun et al., 1988). Therefore, ESEA might be a practical measure of performance ability during prolonged endurance training. Athletic performance deteriorates during chronic high intensity daily exercise. This deterioration is accompanied by a decline in oxygen uptake as the work rate achieved in any test of power output decreases. Serum enzyme activity also increases as the degree of muscle disruption produced by training increases before it declines once more as some degree of muscle adaptation to the current level of exertion is achieved (Morton et al., 1990).

Galun et al. (1988) measured peak oxygen uptake, serum creatine kinase (CK) and serum aspartate transaminase (AST) activity in 41 trained men during a 24 hour march as a means of assessing performance. Serum CK activity peaked at 16 hours during the march, while serum AST activity reached a maximum at 24 hours. Both enzyme activity levels returned to pre-exercise activity 40 hours after the activity ceased. Peak oxygen uptake, which was maximally reduced 16 hours into the march and returned to baseline after 64 hours was 90° out of phase with the rise and fall in serum enzyme activity. It was suggested that during prolonged exercise, muscle ATP is depleted, the cell membrane becomes "leaky" responding to exercise-induced inflammation, and enzymes are released to the vascular space. The focal micro-trauma caused to muscle during strenuous enduring
exercise may also limit the ability of the peripheral tissue to utilize oxygen efficiently. As oxygen consumption decreases with increasing focal disruption evidenced by high enzyme activity, the work rate is reduced and performance decreases.

A similar increase of serum enzyme activity was observed during an Ironman triathlon where serum CK, lactate dehydrogenase (LDH) and AST activity were significantly elevated from the beginning to the end of each segment of the race (Harrison et al., 1991). Serum CK activity for example increased from 150 iU·L\(^{-1}\) initially to 200 iU·L\(^{-1}\) following the swim segment, to 300 iU·L\(^{-1}\) after the bike stage, to 900 iU·L\(^{-1}\) immediately following the run segment, and peaked at 5000 iU·L\(^{-1}\) one day following the event. Activity in all three enzymes increased most dramatically during the weight bearing run segment suggesting that this segment produced the greatest muscle disruption, although results may be confounded by the muscle disruption incurred in the previous swim and bike stage and magnified by dehydration and reduced blood volume during the race itself. The change in serum CK, LDH and serum AST activity in the blood may provide excellent prediction of the relative reduction in physiological function caused by intense enduring exercise.

**Delayed Onset Muscular Soreness**

Delayed onset muscular soreness (DOMS) is the sensation of pain and stiffness that is felt 1 to 5 days following unaccustomed exercise and can lower performance by lowering the capacity of the muscle to generate force (Armstrong, 1984). Pain is usually most intense in the muscle-tendon region where receptors are most concentrated. The soreness can be reduced in part by previous eccentric training (Schwane et al., 1987), or by exercise itself due to an increased afferent input from large sensory units in the muscle (Armstrong, 1984). The time course of DOMS following a single bout of exercise is similar to the rise and fall of ESEA evidenced by a good correlation between muscle soreness measured subjectively
and the activity of plasma enzymes (Schwane et al., 1987). Muscle soreness following extended daily training peaks several days after onset and declines to an elevated plateau, very similar to the pattern of ESEA with heavy training (Banister et al., 1992). This decrease in the amount of perceived muscle soreness with continued exertion represents an important training adaptation and may account at least in part to improved criterion performance after only a few weeks of heavy training even as heavy training is continuing.

Schwane et al. (1987) investigated the effect of short term training on muscle soreness and ESEA in response to downhill running. A subject who ran on a treadmill at a -10 % incline for one week prior to testing experienced less muscle soreness measured on a subjective scale compared with a control. In contrast one week of uphill running at a 10 % incline was not effective in reducing exercise-induced soreness. Curiously no training regimen was effective in reducing serum CK activity significantly during a 45 minute downhill run test. The large inter-subject variability in ESEA response may be partly responsible for this negative result which was also due in part to the difference in fitness between subjects as well as to the uncontrolled nature of their training regime. No attempt was made in the above study to quantify training which might reduce muscle soreness. The actual duration of run training in this study was very short consisting of only 1 hour per week of either uphill or downhill running which is unlikely to produce a sufficient training stimulus to resist muscle disruption during a 45 minute downhill run test. Future experiments of this nature may benefit from a more intensive training regime where both the volume and intensity of exercise is controlled, not only the nature of the exercise.

ESEA and Muscle Disruption

Exercise may well be considered a catabolic process where focal cellular disruption to muscle results in ESEA, and produces protein specific fragments (PSF) from damaged
muscle cell constituents (Mader, 1992), or cell membrane digestion (Armstrong, 1990). Mader (1992) proposed a mathematical model where transcription/translation activity and protein expression are regulated by the concentration of PSF released during muscle catabolism. Nuclear translation and transcription activity are thus stimulated during heavy training through a negative feedback control circuit to increase protein synthesis and allow the adaptive processes of the cell to occur in order to maintain the level of stimulus currently induced. During recovery from the physical stimulus causing the initial catabolism the enhanced anabolism is not immediately switched off, and subsequently produces an enhanced physical ability when fatigue decreases.

Mair et al. (1992) produced evidence for the protein specific fragment hypothesis showing the presence of increased myosin heavy chain protein (MHC) in the blood using magnetic resonance imaging. Elevation of serum MHC is also indicative of cellular disruption and contractile apparatus degradation. In the above experiments the quadriceps muscle in six male subjects was eccentrically contracted against a load equivalent to 110% of maximum torque. Serum CK activity was elevated above baseline within two hours of the eccentric contraction and remained elevated for 13 days when the activity returned to baseline. Myoglobin showed a bi-phasic release into the blood, first peaking 1 day after the eccentric exercise bout and peaking for a second time between day 6 and day 9. It is interesting to note that the release of myoglobin from eccentrically damaged muscle peaks initially following mechanical disruption and secondly following the cellular proteolytic and macrophage digestion discussed previously. MHC fragments in the blood began to increase in the second day after the eccentric exercise bout and showed a similar peak between day 6 and day 9. MHC in plasma may require enzymatic degradation of myosin, or if the exercise is sufficiently intense such as with eccentric contraction, the myosin head may even be stripped from the contractile apparatus. Serum CK activity, MHC fragment
concentration, and myoglobin concentration all returned to baseline after 13 days of recovery. The presence of MHC, myoglobin, and CK activity in the plasma is an indication of focal cellular disruption, temporary fiber necrosis and a leaky plasma membrane. The time course of ESEA was observed to follow the rise and decline of MHC fragment concentration and myoglobin concentration in the serum closely. It has been suggested that the presence of MHC fragments in the serum indicate long term muscle damage of slow twitch fibers in response to eccentric exercise (Mair et al., 1992). This statement, however, seems improbable since after 9 days of recovery maximal torque generation was close to the pre-exercise level in all subjects, and although not measured, it is very probable that the maximal torque may have been restored or improved at day 13 when regeneration was complete.

Manfredi et al. (1991) measured serum CK activity as a marker for exercise induced skeletal muscle disruption in both young and older men following eccentric resistance exercise on a backward motion cycle ergometer. Immediately following three 15-min bouts of eccentric resistive pedaling against a motor driven pedal system at 90, 80, and 70% of maximal concentric power, a muscle biopsy was taken from the vastus lateralis muscle. The degree of muscle disruption evidenced by Z-band streaming and smearing, was more extensive following eccentric exercise in older men. This may be due in part to a lower level of fitness and smaller muscle mass in older men, and that the older men may have been unaccustomed to eccentric exercise. Serum CK activity peaked 5 days following eccentric exercise and despite the more extensive damage observed in older men, the activity of serum CK was similar in both groups. The authors therefore suggest that ESEA is not a sensitive index of exercise-induced muscle disruption. Serum CK activity, however, was consistently if not statistically higher in older men with each serial measure. It is likely that the significant difference
among groups was masked by a large inter-subject variability in serum CK response, which has also been observed in other studies (Schwane et al., 1987).

Nuviala et al. (1992) studied the pattern of ESEA following a marathon and found a maximum activity of serum LDH after the race. Serum CK activity was observed to increase from a base level to 126 % of this value at the finish and to 415% 24 hours later. Apple et al. (1988) assessed skeletal muscle disruption following a marathon in 35 women and 34 men from the cumulative amount of serum CK activity serially released by 24, 36, 48, 60, 72 and 96 hours respectively after the race. Using a mathematical model developed to predict infarct size from ESEA, they found that the male marathoners incurred greater skeletal damage than women marathoners possibly due to the fact that their running intensity was much greater or due to their larger muscle mass. Serum CK activity was observed to peak 24 hours after the marathon and remain elevated for up to 72 hours. Following an Ironman triathlon serum CK and serum LDH activity have been shown to be elevated ranging between 200 % and 3400 % of baseline activity, and to remain elevated for at least 6 days (Harrison et al., 1991). It is evident from the above results that muscle disruption does occur during an endurance event, and therefore training for the event must be titrated in such a fashion that various specific adaptive processes of the body have time to equilibrate to a new higher level of training and competition so that any undue disruption may be minimized during competition itself.

**Theoretical Models of ESEA**

A theoretical model of exercise-induced ESEA was described by Banister et al. (1992) based on serial measurement of ultrastructural change following both direct mechanical trauma and secondary inflammation. Five male subjects underwent 70 eccentric contractions of the quadriceps muscle and serum CK activity was measured for 10 days until serum CK activity returned to a pre-exercise level. In this model an impulse of training
(17 eccentric contractions of the quadriceps) is the stimulus for loss of enzyme either via cell membrane or via lymphatic flow. The time course of loss of CK activity from the cytosol is dependent greatly on the degree of muscle damage as well as the time course of the inflammatory response which introduces delay in the appearance of enzyme in the blood. A 2nd order two component model was proposed to simulate the loss of cellular enzyme to the vascular space and may provide valuable insight into the underlying mechanism of muscle adaptation.

Although the pattern of ESEA after a single bout of exercise has been studied extensively (Apple et al., 1988, Armstrong, 1990) and modeled (Banister et al., 1994), the mechanism and changing pattern of ESEA after repetitive training represents an adaptation that remains unclear. One possible theoretical explanation is that accompanying exercise adaptation a pool of so-called "weak" or easily damaged fibers becomes diminished resulting in a reduced ESEA response. Exercise repeated before adequate repair of disrupted muscle will lead to chronic ESEA. A complex mathematical model of ESEA is described by Morton and Carter et al. (1992) which unfortunately offers no easily understood explanation or immediate insight into the pattern of ESEA with exercise training.

Morton et al. (1990) serially measured the change in ESEA during run and cycle training in two subjects and found a temporal relationship with time delay between ESEA of three enzymes (LDH, CK and AST) and modeled fatigue. Intense training for 28 days, followed by 32 days of detraining caused a rapid rise in ESEA that peaked prior to day 28 and then declined to an elevated plateau during a continued period of training, possibly reflecting muscular adaptation, and then decreased rapidly to baseline when training decreased. Modeled fatigue paralleled the time course of ESEA although slightly out of phase with it. The plateau effect of ESEA with heavy training is one adaptation that may
have important diagnostic value for overtraining. The pattern of ESEA with training and taper may indicate the strength and acquisition of the adaptive process in this way.

Little is known about the precise detail of the physiological and biochemical change in the body during the transition between training adaptation and overtraining. ESEA may be a good indicator of this transition and marker for overtraining since prolonged muscle disruption which accompanies very intense training will result in ESEA that does not recover even following a regeneration or taper period. Since ESEA is variable between subjects, group comparisons often obscure the true time course of ESEA in the individual. Serial measurement of enzyme activity during a period of heavy training to assess for the degree of muscle disruption, may provide invaluable feedback to the endurance athlete. The athlete may in turn increase or reduce training dependent on whether serial ESEA measures remain at a chronically elevated plateau, suggesting possible overtraining (decrease training) or if the current value remains low indicating training is insufficient to stimulate optimal adaptation (increase training).

IV. OVERTRAINING

Classification

Several studies in the literature classify overtraining as both a short term and a long term phenomenon (Lehmann et al., 1992, 1993). Short term overtraining is characterized by training fatigue, muscle soreness, and a transient decrement in physical performance (Lehmann et al., 1993). A short term period of heavy training is more appropriately termed "adaptive training" since sufficient recovery, and improved performance may take only a few days to a maximum of 2 weeks (Lehmann et al., 1991). Adaptive training should therefore not be mistaken for overtraining since an athlete is able to recover relatively quickly and does not display the chronic symptoms
associated with the overtraining syndrome. Long term overtraining is characterized by an accumulation of fatigue, sore and stiff muscles, disturbed mood state, as well as reduced performance in competition for an extended period of time (Lehmann et al., 1993).

**Markers of Overtraining**

Although it is generally agreed that physical training stimulates improvement in both strength and endurance, a sudden increment in either training volume or intensity, may produce trauma rather than continuing adaptation (Costill et al., 1988). This overload may lead to staleness or a decrement in performance, and absolute or relative rest may be the only means by which performance may be restored (Costill et al., 1991; Koutekakis et al., 1990). An athlete showing symptoms of overtraining often complains of a feeling of heaviness and an inability to perform well during training and competition. Possible physiological and hematological markers of overtraining at rest have been identified, and these include an elevated oxygen consumption above that previously required for submaximal work, an elevated heart rate, increased serum CK activity, and elevated serum free fatty acid, above that previously attained at a similar submaximal effort. Increased serum cortisol, and uric acid concentration, as well as decreased serum ferritin, iron, hemoglobin, and testosterone concentration have also been associated with overtraining (Fry et al., 1992). During submaximal exercise, increased oxygen consumption, heart rate and ventilation above a previously attained level for the same effort may indicate an overtrained state.

Although it would be attractive to use these biological markers to maximize competitive preparation while avoiding overtraining, there is still no good agreement on the validity of their use. However taking cognizance of such empirical findings an effective training program should provide adequate rest in a trainee, and avoid an
extended period of incremental training stress. Periodization of training provides a system for including heavy training, which may then be followed by a regeneration or taper period which will optimize performance (Banister, *Abstract* 1992). Such a periodization plan accompanied by serial measurement of training for feedback on the effect of training will allow a better estimate of any incipient overtraining when performance will be optimal and fatigue absent. Serial measurement of physical performance throughout all training, in order to assess the effect of training on physical performance, is an integral measure in preventing overtraining during any extended physical preparation for competition and has only rarely been previously reported (Morton *et al.*,1990, Banister, *Abstract* 1992).

One common symptom of both adaptive training or long term overtraining is the well known phenomenon of muscle disruption commonly observed following intense or enduring exercise (Armstrong, 1990). Although it is generally agreed that physical training stimulates improvement in both strength and endurance, a sufficient increment in either training duration or intensity may produce muscle disruption rather than continued adaptation. The catabolism of the muscle ultrastructure (Waterman-Storer, 1991) and disruption of myofibrillar order also known as "micro-injury" (Armstrong, 1990), may be considered a necessary precursor for subsequent muscle anabolism (Banister *et al.*,1992). The time course of degeneration and regeneration of muscle is thought to parallel the morphological sequence of events of more severely traumatized muscle (Banister *et al.*,1994), and may even follow the impairment and subsequent improvement in a criterion performance time, measured serially throughout training and taper (Banister, 1992).

The most important factor in the diagnosis of overtraining is a decrease in the level of physical performance even following a regeneration period (Fry *et al.*,1992).
Attempts to identify a consistent and reliable physiological and/or biochemical profile of overtraining in an athlete have been unsuccessful due in part to the inherent limitations in overtraining studies to date, and the inability to establish or diagnose whether overtraining has taken place. Possible physiological and hematological markers of the overtraining syndrome at rest and during exercise have been identified yet no single parameter alone will determine whether overtraining has taken place. During submaximal exercise for example, increased oxygen consumption, heart rate and ventilation above a previously attained level for the same level of effort may be symptoms of the overtraining syndrome but not necessarily since causality is extremely difficult to prove in the case of so many potential activators. Although it would be attractive to use biological markers or better still a single marker to maximize competitive preparation while avoiding overtraining, there is still no good agreement on the validity of their use (Lehmann et al., 1992, 1993).

Avoidance of Overtraining

Optimal training lies somewhere along a continuum between too little training which does not provide an adequate stimulus for adaptation, and excessive training which can lead to a prolonged performance decrement or injury (Zarkadas et al., Abstract 1994). The challenge to both trainer and athlete is to maximize training adaptation without harmfully overloading the adaptive process. Pressure to perform, especially in the elite endurance athlete, may cause a competitor to train harder and harder to the extent of developing chronic fatigue. This overload in training volume may lead to impaired physical performance or "staleness" to a point where absolute or relative rest may be the only means by which performance may be restored (Koutekakis et al., 1990). An optimal training program must therefore provide adequate rest between both individual workouts, and
extended periods of heavy training so that accumulated fatigue and degradation of tissue, necessarily as a stimulus to adaptation has time to dissipate or decay (Banister, 1992).

**Overtraining Studies**

Verde *et al.* (37) attempted to identify potential markers of overtraining in ten runners who undertook a 35% increment in training distance during a three week period. All subjects were highly trained runners who underwent 3 weeks of training with an average weekly distance of 98.8 km/wk, and then increased their training volume to 130.3 km/wk for a further 3 weeks. Measurements made included serum cortisol concentration, serum CK activity, and several immunological blood parameters, since overtraining is also thought to lead to a depressed immune response (Fitzgerald, 1991). These investigators found no significant elevation in any of blood measure during the increased training period. It is likely that this period of increased training was insufficiently long or intense enough to induce any symptom of overtraining except for fatigue rated on a subjective psychological scale. Results from this study are of little value because the training was not measured quantitatively and no subject faced the stress of major competition and could taper if they experienced any fatigue. Furthermore, no serial blood measurement was taken during heavy training and no criterion performance was measured. The fact that there was no significant increase in serum CK activity suggests that these highly trained runners were not stressing their system sufficiently and therefore any attempt to identify a marker for overtraining in this experiment would be futile.

In a similar study, Lehmann *et al.* (1991) attempted to overtrain 8 competitive runners by increasing their weekly training distance from 86 to 175 km/wk during a 4 week period. The several findings in this study include a significant elevation in serial measurement of serum CK activity and serum cortisol concentration, and a decrease in
serum testosterone concentration at the end of the 4 week increased training period. Following the period of study not one of the runners improved nor even approximated their previous best personal record during the subsequent competitive season. It is possible that the large increase in training distance had a lasting detrimental effect on performance. Again a major limitation in this study was that the investigators were not certain if a state of overtraining had been reached since there exists no objective physiological or biochemical criterion associated with overtraining. There was not enough serial measures of physical performance with enough physiological/biochemical measures to determine any correlation between the latter's time course and that of permanent debilitation of physical performance.

The main criterion of the overtraining syndrome is a state of chronically depressed physical performance. It remains to be defined, however, how much physical performance must decrease throughout training or how long performance should be kept below a pre-established standard. Fry et al. (1992) attempted to assess chronically depressed performance in 5 subjects who doubled their current interval training sessions on a run test to exhaustion. An average 29% decrement of performance time was observed in all subjects following 10 days of intense training. Physical performance in each subject returned to a pre-trained value after only 5 days rest, and no serial test was performed following this rest period. A blood sample was taken from each subject and analyzed for a range of hematological, biochemical, and immunological parameters. Again the researchers were uncertain whether overtraining had taken place since only serum CK activity remained elevated after 5 days of rest and physical performance had returned to a pre-trained level. The testosterone: cortisol ratio which may be an indicator of the degree of anabolism and catabolism of muscle remained unchanged. Since performance returned to normal baseline after only 5 days of recovery this example of adaptive training discussed previously should not be classified as overtraining.
Costill et al. (1988) doubled the training volume in swimmers for ten days while maintaining training intensity to examine the physiological and performance change that may accompany a sudden increase in training stress. Results showed no change in swim power, sprint swim time or criterion swim time suggesting that the swimmers were able to absorb the extra training volume without any symptom of overtraining. All swimmers experienced local muscular fatigue and had difficulty in finishing workouts and were divided into either a fatigue or tolerant group based on a subjective rating of their own training effort and evaluation by the coach. Glycogen depletion was observed in the fatigue group after 10 days of heavy training compared with a pre-trained measure. Again such results are questionable since the fatigue group began heavy training with a significantly lower muscle glycogen content compared with the tolerant group suggesting that this group was fatigued before this experiment began. Secondly, reduced muscle glycogen may have been the result of an uncontrolled low carbohydrate diet and not due to the training itself. Furthermore, swimming may not be an appropriate exercise to study overtraining since it is not weight-bearing and muscle disruption, although present, may not be sufficient to cause significant impairment of performance. ESEA was also not measured in this study which may have given more insight into the nature of fatigue experienced by a swimmer.

A more recent study by Costill et al. (1991), monitored training of 24 collegiate swimmers during a 25 week season. After 4 weeks, one group of swimmers doubled their training for 6 weeks followed by a tapering period preceding a mid-season competition. A resting blood sample was taken from each swimmer and serum CK activity was not found to be significantly different between groups. On the average, the group that doubled their training experienced a 1% - 2% decrease in swim sprint performance (46 and 91m sprints) and a 1.4% - 2.9% increase during longer events (183 and 2743 m) measured at the end of heavy training and a preceding taper. The major finding in this experiment was that the
additional training volume did not produce a greater improvement in swim performance compared with the lesser trained group. This conclusion may be somewhat misleading since one would expect that immediately following heavy training, physical performance would still be impaired. A large improvement in sprint velocity was observed during the taper period which was most pronounced in the heavily trained group. It is not unlikely that if the taper were extended for even a further short period, the more heavily trained group of swimmers would have further improved their sprint velocity and eventually surpassed the performance of the lesser trained group. The value of an increased volume in a swimmer's training is questionable since the majority of the races are less than 3 minutes in duration. Doubling training volume in swimming may not tax the body sufficiently to result in chronic fatigue associated with overtraining, evidenced by quick recovery of ESEA and physical performance with taper in this experiment. Finally, since almost all swim training is performed at very high intensity, a result from a study such as this one may have little if any application to other events such as running where training may be less intense but more demanding on the musculo-skeletal system.

In one recent study, Hooper et al. (1993) measured stress hormones in 14 elite swimmers at five points during the course of 6 months during training, taper, and post competition. A positive correlation was found between training distance and plasma norepinephrine and epinephrine concentration respectively. Decrement in performance, a high level of fatigue rated subjectively, and a high serum norepinephrine concentration was found in 3 of the swimmers suggesting overtrained had occurred. A clear pattern of hormonal change with overtraining, however, has not yet been established. Several limitations of this study were that not all the subjects were male (9 females, 4 males), training was not quantified, fatigue was measured only subjectively on a point scale of 1 to 7, and criterion performance was not the same for sprint (100 m) and long distance swimmers (400 m). The method of determining overtrained swimmers is questionable since it was dependent
on only one criterion performance and a subjective rating of fatigue was used with no biochemical correlate. Furthermore, the 3 swimmers who were identified as being overtrained quickly rebounded with a taper of greater volume than the lesser fatigued group. It is very possible that a proper taper of substantially less volume would have increased their performance even further. The 3 "stale" athletes also consistently performed more dry-land training (the nature of which was not identified) than the other swimmers, especially during the late training season taper period which may have been a major cause of the increased catecholamine concentration during this period. It is unlikely that these swimmers were overtrained but more simply were responding to heavy training with low criterion scores which is to be expected. It is possible that the elevated blood norepinephrine concentration was a sympathetic response to the large training load and may be associated with the emotional stress previous to an important competition. It remains questionable whether elevated blood nor-epinephrine concentration is a reliable marker of overtraining.

The Immune Response

Although moderate exercise may have a positive effect on the immune system, an athlete subjected to a large volume of training may increase his/her susceptibility to infection (Fitzgerald, 1991, Parry Billings et al., 1992, Verde et al., 1992, Shikai et al., 1993). Overtraining is associated with colds, sore throats, and flu-like illnesses and there have been many reports of immuno-suppression in the overtrained athlete although the cause of this impairment of immune function is unknown. A short bout of intense exercise can temporarily impair the immune response for up to several hours, with a marked change in the number and functional capability of lymphocytes. Glutamine which is utilized extensively in the immune system and is essential for lymphocyte production has been shown to decrease after enduring exercise such as a marathon (Parry-Billings et al., 1992). The major source of glutamine in the body is skeletal muscle which synthesizes, stores and releases...
glutamine even at rest. Overtraining causes a decrease in glutamine release to the plasma which can lead to immuno-suppression. Maintaining muscle glycogen by carbohydrate supplements is protein sparing and may preserve the ability of the muscles to release adequate glutamine.

In one study by Parry-Billings et al. (1992) overtraining was diagnosed in 40 athletes who had chronically poor performance, depression, sleep disturbance and in whom the symptoms persisted for at least 3 weeks. The concentration of glutamine was decreased in plasma from overtrained athletes and from control runners following a marathon. Acute exercise as well as chronic stress associated with a period of heavy training may suppress immune function by reducing lymphocyte count. Verde et al. (1992) monitored ten elite runners who increased their training by 35%, and showed an 18% suppression lymphocyte proliferation following acute exercise. Results of this study demonstrate that a well trained individual may undertake a 30 minute bout of acute exercise with minimal and short lived suppression of immunity but any increase in training volume may amplify this suppression.

The lack of consistent or reliable data in overtraining studies does not allow any firm conclusion to be made about a marker of overtraining. It may be deduced from the above review that ESEA measured serially throughout a period of heavy training is potentially the best physiological marker of an overtrained state, since muscle disruption with heavy training is a consistent finding in almost every overtraining study. A training regime should also incorporate serial criterion measurement to assess any chronic decrement in physical performance so that training can be titrated against accumulating fatigue accordingly. Further experimentation is necessary to determine how much and how long physical performance may remain impaired and what optimal level of ESEA is necessary in order not to disrupt the delicate balance between training adaptation and overtraining.
V. MODELING

A systems model of training proposed by Calvert et al. (1976), described by Morton et al. (1990), and elaborated to explain the structural features of a training program by Fitz - Clarke et al. (1991) will be used to quantify and describe the pattern of training and peaking for competition in this thesis. Briefly, this model is based on the stimulus - response behavior of the body to exercise. The stimulus \( w(t) \) is provided by physical training which in turn modulates performance \( p(t) \) relative to a baseline level. The quantity of daily training may be calculated and presented as a training impulse (TRIMP), expressed in arbitrary training units (ATU). The stimulus of training is short compared with the body's adaptive response, and therefore can be considered as an impulse whose magnitude must be related to the intensity and duration of the training session. A single training bout can be described by the duration of the session (D) in minutes multiplied by the ratio of exercise to maximum heart rate, both above a resting value. This ratio defined as the delta heart rate ratio (\( \Delta \text{ HR Ratio} \)) ranges between a lower limit of 0 at rest, to an upper limit of 1 at maximal exercise. Maximal heart rate (HR\(_{\text{max}}\)) can be estimated as (220 - age) or may be measured directly from a treadmill test or an all out 400-800 m run. Basal heart rate (HR\(_{\text{rest}}\)) may be taken before rising in the morning, while exercise heart rate (HR\(_{\text{ex}}\)) is measured as an average during exercise. Thus training undertaken at any time \( t \) may be expressed as an area under the curve represented by the pseudointegral:

\[
\text{w(t)} = \text{Duration of Training} \times \frac{\text{HR}_{\text{ex}} - \text{HR}_{\text{rest}}}{\text{HR}_{\text{max}} - \text{HR}_{\text{rest}}}
\]

\[
= D \times \Delta \text{ HR Ratio}
\]
An intensity weighting factor $Y$ is used to correct the bias introduced into $w(t)$ from long non-strenuous training and to give more credit to short intense sessions which provide a greater training stimulus the weighting factor is given by:

$$Y = e^{bx}$$

where $x = \Delta HR\, Ratio$, $b$ is a constant, different for men (1.92) and women (1.67) respectively and $Y$ reflects the exponential rise of blood lactate as the fractional elevation of exercise heart rate above rest. Thus:

$$w(t) = D \times \Delta HR\, Ratio \times Y$$

A training impulse has two antagonistic effects: 1) to increase fitness $g(t)$ which causes an increase in performance, and 2) to increase fatigue $h(t)$ which subtracts from performance. The equations defining these components of the training stimulus effect are given by:

$$g(t) = g(t-i) e^{-i/\tau_1} + w(t)$$

$$h(t) = h(t-i) e^{-i/\tau_2} + w(t)$$

where $\tau_1$ and $\tau_2$ define decay constants of fitness and fatigue in days, and $i$ is the intervening period in days between training. The difference between fitness and fatigue at any time $t$ gives a net performance $p(t)$ given by:

$$p(t) = k_1 g(t) - k_2 h(t)$$

where $k_1$ and $k_2$ are arbitrary multiplying constants. The time constants for fitness $\tau_1$ and fatigue $\tau_2$, and their respective weighting factors $k_1$ and $k_2$ can be determined by iterative computer modeling of predictive performance $p(t)$ against real criterion performance $C_p$ measured experimentally. When the least squares fit of predicted to real performance is achieved these parameters may be used for a period to prescribe both the dose and pattern of training necessary to produce a desired future performance.

In a further elaboration of this model of training Fitz-Clark et al. (1991), proposed an Influence Curve that defined the structure of the training stimulus effect throughout a period of training and peaking which dictates an optimal strategy of physical
preparation to do well in a single future competitive event. The influence curve is given by:

\[ L(\mu) = k_1 e^{-\mu/\tau_1} - k_2 e^{-\mu/\tau_2} \]

where \( \mu \) is the time in days before performance. The dimensionless impulse response \( L(\mu) \) illustrates both the positive and negative contribution of each days training from the onset of training to competition. Using the default parameters 45, 15, 1 and 2 for \( \tau_1 \), \( \tau_2 \), \( k_1 \) and \( k_2 \) respectively, the greatest benefit of training is derived from training performed at a time when the Influence Curve is maximally positive calculated to be day 40 prior to competition. Using the same parameter vector, training within 16 days prior to competition theoretically contributes a negative component to the Influence Curve more and should be avoided.
METHODOLOGY

HYPOTHESES

Null Hypothesis

1) There is no difference between a step reduction in training volume protocol and an exponential decline in training volume taper protocol.

Positive Hypotheses

2) System parameters of a systems model of training for an individual induced by endurance training do change over time. The growth or decay of each individual parameter may be characterized by a specific time constant (see Figure 1) and such change will be consistent among subjects.

\[ \tau_1, \tau_2, \kappa_1, \kappa_2 \]

Figure 1 Hypothesized change in system model parameters. Each parameter will grow or decay with its own characteristic time constant.

3) If the previous hypothesis is true, then subjects can be characterized as plastic or non-plastic depending on the extent to which their system parameters change and there exists a different pattern of optimal taper for these individuals.
SUBJECTS

Eleven male triathletes volunteered to take part in this study. They gave their written consent after being medically approved for participation by a physician and after having been fully informed of the nature, risks and benefits of their participation. All tests performed on these subjects received approval of Simon Fraser University's Human Subjects Ethics Approval Committee. The physical characteristics of each subject is given in Table 1.

Table 1 Physical characteristics of the subjects.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (year)</th>
<th>Height (m)</th>
<th>Weight (kg)</th>
<th>Body Fat (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.P.</td>
<td>20</td>
<td>1.80</td>
<td>71.0</td>
<td>6.6</td>
</tr>
<tr>
<td>R.C.</td>
<td>24</td>
<td>1.84</td>
<td>74.0</td>
<td>7.5</td>
</tr>
<tr>
<td>G.A.</td>
<td>25</td>
<td>1.84</td>
<td>76.0</td>
<td>8.1</td>
</tr>
<tr>
<td>P.Z.</td>
<td>25</td>
<td>1.83</td>
<td>90.0</td>
<td>11.3</td>
</tr>
<tr>
<td>A.C.</td>
<td>26</td>
<td>1.73</td>
<td>81.0</td>
<td>9.2</td>
</tr>
<tr>
<td>D.C.</td>
<td>27</td>
<td>1.67</td>
<td>66.0</td>
<td>7.1</td>
</tr>
<tr>
<td>B.G.</td>
<td>27</td>
<td>1.71</td>
<td>68.0</td>
<td>8.6</td>
</tr>
<tr>
<td>J.B.</td>
<td>27</td>
<td>1.72</td>
<td>73.5</td>
<td>9.7</td>
</tr>
<tr>
<td>J.W.</td>
<td>28</td>
<td>1.75</td>
<td>73.5</td>
<td>7.1</td>
</tr>
<tr>
<td>J.C.</td>
<td>31</td>
<td>1.80</td>
<td>80.0</td>
<td>10.1</td>
</tr>
<tr>
<td>C.G.</td>
<td>32</td>
<td>1.86</td>
<td>83.0</td>
<td>9.1</td>
</tr>
<tr>
<td>X ± S.D.</td>
<td>26 ± 4</td>
<td>1.79 ± 0.06</td>
<td>77.0 ± 6.5</td>
<td>8.7 ± 1.4</td>
</tr>
</tbody>
</table>
Subjects had a diversity of training background and competitive triathlon experience. Eight of the triathletes had competed in at least one Ironman triathlon previous to the present experiment while three were novices. One subject joined halfway through the study and therefore data is presented for him only for the latter part of the experiment. Two subjects sustained an injury during training that prevented them from completing all the required criterion run test procedures.

**STUDY DESIGN**

The entire experiment lasted 98 days (from May 24 to August 29, 1993) and required each athlete to follow two separate periods of heavy training each followed by a peaking or taper period prior to a designated real competition (Figure 1). During the first block of training which lasted 4 weeks, subjects were required to maintain a training average of 1 to 2 hours per day which is equivalent to approximately 170 TRIMPS with a proportional amount of running, cycle, and swimming activity undertaken each week. Prior to Taper 1 subjects were randomly assigned to one of two taper groups, matched for age, weight, and initial criterion 5 km run time. Group 1 (n = 6) tapered their training volume by 50% in an exponential decay fashion while Group 2 (n = 3) reduced their training volume by 30% in a stepwise manner. One subject was excluded in Group 2 due to non-compliance to the training protocol and another subject reduced their training exponentially instead of in a stepwise fashion and was therefore included in Group 1.

Seven subjects competed in a mid season short course triathlon (2 km swim, 28 km bike, 13 km run, 18 km bike) immediately following this 10 day peaking phase.
(Taper 1). During the whole period each subject completed serial cycle ergometry tests to exhaustion one day per week and completed at least one criterion best effort 5 km run over a standard course each week.

![Diagram showing study design with tapers and training volumes.](image)

**Figure 1** Study design showing diagrammatically the step reduction taper compared with the exponential decay taper format during Taper 1 and the two time constant decay tapers during Taper 2.

Following the mid season competition heavy training was resumed for 6 weeks and each subject increased their training volume to 2 to 3 hours per day which is approximately equivalent to 220 TRIMPS. Subsequent to this period of heavy training all subjects were randomly assigned to 2 different exponential taper groups with either a low volume taper with a time constant of 4 days ($\tau \leq 4$) in Group B ($n = 4$) or a high volume taper with a time constant of 8 days ($\tau \geq 8$) in Group A ($n = 7$). During Taper 2 both groups performed a similar proportion of swim, bike and run training during the period. All subjects competed in the 1993 Canadian Ironman Championship in Penticton, B.C. (3.9 km swim, 180.2 km bike, 42.2 km run). All triathletes except one finished the race. One athlete did not finish due to an aggravated Achilles tendon injury.
SERIAL DATA COLLECTION

Heart Rate

Average training heart rate for each workout was recorded at 1 minute intervals via telemetry (Polar Vantage XL) in serial daily files for weekly downloading to a Macintosh IIfx computer. It was not possible to monitor heart rate during swim training and a 10 second count was used to give an estimate of the average heart rate during this activity.

Physical Characteristics

Height and weight were measured and percent body fat estimated from caliper skin fold measurements at six sites (triceps, scapula, abdominal, supra iliac, front thigh, and medial calf) using the Yuhasz equation. Both the initial weight and skin fold measurements were repeated at the middle and at the end of the experiment to account for any change in body composition.

Training Log

A training log book was given to each subject at the beginning of the study so that each could keep an accurate record of their training prior to each week's data entry into a computer data base (Appendix B).

Criterion Physical Performance

A real 'best effort' physical performance was measured serially during both training and taper to assess the cumulative response of a subject to the training stimulus undertaken each day.
**Criterion Run Field Test**

At least one and sometimes two 5 km criterion runs were performed each week to the best of the subject's current ability to monitor any change in this performance. Subjects ran all out on their own 5 km course which was chosen to be as flat as possible, and each subject ran this course at a time when there would be a minimal possible interruption to their effort (traffic, obstacles etc.).

**Scoring Performance**

Performance was scored on an exponential curve where 1000 points was assigned to a subject's personal best performance during the season and any measure less than that was assigned fewer points (see Appendix C). This method of scoring was used to equate performance data on a common scale for all subjects since each subject ran a personalized 5 km course, introducing some variation in result time. This scoring method was also used to give more emphasis to a performance closer to a subject's personal best where mere seconds of time separate a winning result that may not be significant statistically.

**Laboratory Tests**

A ramp test to exhaustion was also performed weekly by each subject on a cycle ergometer in a laboratory under ambient 21 °C and 40% relative humidity conditions to assess the serial change in maximal power output and in respiratory gas exchange, both of which were measured on selected dates during training and taper.

**Ergometry**

The cycle ergometer (Lode: Groningen, Holland) was electromagnetically braked and controlled by an external PC compatible Megacom computer incorporated with an
A/D, D/A interface (KEM Industries). A turbo C program was written to control the ramp at a variety of work rate protocols. The ramp test protocol consisted of a 4 minute warm-up period at a load of 30 watts followed by a 30 watts/min ramp until exhaustion or until a subject could not maintain their revolutions above 80 rpm.

**Ventilatory Data Collection**

On three ramp tests chosen two weeks prior to taper and during each week of Taper 2 a real time breath-by-breath $\dot{V}O_2$ acquisition was made using a Macintosh IIfx computer equipped with a National Instruments A/D board (NB-MIO-16) and a National Instruments software package (LabView II). Expiratory timing (sail switch), expiratory flow rate (modified from an Alpha Technologies Ventilation Module, model VMM110), fraction of expired oxygen $FEO_2$ (Applied Electrochemistry, model S-3A), and fraction of expired CO$_2$ $FECO_2$ (Applied Electrochemistry, model CD-3A), were recorded at 112 Hz (each channel) for each breath. Total time (TT) and expired time (TE) were recorded from the binary signal of the expiration sail switch. In addition, inspired volume (Alpha Technologies turbine), heart rate (Physio-Control monitor), work rate, and rpm were recorded each breath.

The data saved each breath included: run time (min), oxygen uptake $\dot{V}O_2$ (l·min$^{-1}$: STPD), CO$_2$ production $\dot{V}CO_2$ (l·min$^{-1}$: STPD), minute ventilation $VE$ (l·min$^{-1}$: BTPS), functional residual capacity $FRC$ (l: BTPS), end tidal oxygen fraction $FETO_2$(%) and end tidal CO$_2$ fraction $FETCO_2$(%), TE (ms), TT (ms), alveolar ventilation $VA$ (l:BTPS), inspired ventilation $VI$ (l:BTPS), work rate (Watts), and pedaling frequency (rpm). Extraneous data, due to swallowing, coughing, etc. were removed prior to analysis.
**Blood Sampling and Analysis**

A resting blood sample was drawn into vacutainer tubes from each subject just prior to the ramp test each week by venipuncture from the antecubital vein. Samples were allowed to clot for approximately 15 minutes, centrifuged for 10 minutes to separate the serum and then refrigerated until analysis. Total serum CK activity (iU·L\(^{-1}\)) was measured on a Roche Cobas Bio analyzer at 30 °C using a BMC (Boehringer- Mannheim CmdH, Mannheim, West Germany) reagent as outlined by Szasz *et. al.* (1976). The coefficient of variation in repeated serial analyses of standard samples of this enzyme ranged between 3-5% during the period of study. Individual unidentified duplicate samples sent through the period were also analyzed to within ± 5 iU·L\(^{-1}\).

**Diet**

All subjects were advised to incorporate as much complex carbohydrate and as little fat as possible into their diet. Diet, however, was not controlled for in this experiment except prior to blood sampling where a subject was required to fast for 12 hours. A weekly supply of high energy Cliff Bars\(^{®}\) and a liquid complex carbohydrate Fruit Source\(^{®}\) in a jar and plastic sachet form were given out each week to all subjects for supplement during training and competition. Food or liquid supplement was not permitted during either of the serial run or cycle performance measures.
DATA ANALYSIS

Statistics

The improvement in physical performance with taper within a group of subjects was statistically analyzed using a paired t-test for significance. The same paired t-test was used to assess any significant change in $\overline{\text{VO}_2\text{max}}$, anaerobic threshold, and serum CK activity. The difference between the two groups during training and taper periods, for all variables which include performance on a 5 km criterion run, and a 30 watt ramp maximal ergometer test was evaluated using a Generalized Linear Model (GLM) to account for the unbalanced number of subjects in each group. A t-test was also used to compare both groups on actual percent improvement on physical performance measures. Iteration of predicted against actual performance was correlated and expressed by the coefficient of determination, $r^2$. 
RESULTS

The effect of a 10 day taper (Taper 1) and a 13 day taper (Taper 2) on physical performance and relevant physiological and hematological parameters are summarized in the following tables and figures. The effectiveness of each taper protocol in this study was assessed by serial measurement taken during the actual taper period itself and compared with a single measure taken in the week prior to commencing taper. Actual physical performance on a 5 km criterion run and during a maximal ramp test on a cycle ergometer was also modeled by computer iteration to achieve a best fit with predicted performance of a training model (Morton et al., 1990). The modeled results of each individual subject are presented graphically as well as the mean change in system model parameters.

TAPER 1

An exponential decay in training volume (Group 1) was compared with a step reduction in training volume protocol (Group 2) during a 10 day taper (Taper 1). The average training for each group shown by a profile of individual training impulses (TRIMPS) during the first 42 days of this study is depicted in Figure 3. Average training volume preceding taper was similar in both groups, however, there was a significant interaction of training impulses between groups (GLM) during the taper period. Group 1 reduced their training exponentially with a time constant of approximately $\tau = 5$ days. This reduction was equivalent to a 50% reduction in training volume while Group 2 reduced their training by 30% in a step fashion. There was no significant change in intensity of training during the taper compared with the training average in both groups. Training frequency decreased significantly in the last week of taper from a training average of $5.5 \pm 1.2$ d/wk to $4.4 \pm 1.8$ d/wk ($X \pm S.D.$) in both groups.
Figure 3  Comparison of the profile of training and taper for both Group 1 (n = 6) and Group 2 (n = 3) taper groups. Training quantity is shown as individual training impulses repeated over the first 42 days of this study. A significant interaction was observed in training quantity during Taper 1 between the exponential decay taper group (τ = 5) and the step reduction in training volume group. Group 1 reduced their weekly training volume by 50% while Group 2 reduced their training by 30%.
Effect on Performance

The effect of a 10 day taper on physical performance was assessed for both a 5 km criterion run and a maximal ramp cycle ergometer test to exhaustion. These performance measures were used to compare the effectiveness of an exponential decay taper (Group 1) and a step reduction in training volume (Group 2). Results (tabulated in Table 2 and graphed in Figure 4) show significant improvement in both criterion run score and time and ramp score and maximal power output during the last five days of Taper 1 compared with pre-taper values in the exponential decay group. This improvement is equivalent to an average 46 second decrease (4 %) in 5km time and a mean 23 W (5 %) increase in maximal power output. The step reduction group improved their ramp score significantly in the last five days of Taper 1 compared with a pre-taper average score. 5 km run time decreased by a mean 15 seconds (1 %) and an average 4 W (1 %) increase in maximal ramp power although neither was significant. Group comparison revealed that an exponential decay taper (Group 1) improved both criterion run score and maximal ramp power significantly more than the step reduction in training volume taper (Group 2).

Table 2 Performance measures for both a criterion 5 km run and a maximal ramp test on a cycle ergometer immediately preceding and at one or two times during Taper 1. Values are means ± S.D.

<table>
<thead>
<tr>
<th></th>
<th>Exponential Decay Taper</th>
<th>Step Reduction in Training Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre Taper Days</td>
<td>First 5 Days of Taper</td>
</tr>
<tr>
<td>RUN Time (sec)</td>
<td>1149±109</td>
<td>1136 ± 86</td>
</tr>
<tr>
<td>Score</td>
<td>815 ± 98</td>
<td>883 ± 111</td>
</tr>
<tr>
<td>CYCLE Power (W)</td>
<td>423 ± 25</td>
<td>-</td>
</tr>
<tr>
<td>Score</td>
<td>633 ± 98</td>
<td>-</td>
</tr>
</tbody>
</table>

* Significant improvement compared with Pre Taper. † Significant group difference.
Figure 4 Comparison of performance improvement both between a step reduction in training volume group (n=3) and an exponential decline in training volume group (n=6) and within groups during Taper 1. Performance was compared in the week immediately preceding taper and one or two times during the taper itself by raw scores (B and D) and by a scoring method (A and C). Values are means ± S.D. % change compared with Pre-Taper 1.

* Significantly greater than Pre-Taper1 within a group.
† Significant group difference with a t-test for % improvement at p ≤ 0.05.
TAPER 2

A profile of training is presented in Figure 5 showing the pattern of training impulses for both Group A and Group B from day 42 of training to the end of Taper 2. This 13 day taper compared the effect of two exponential decay tapers, a high volume taper (τ ≥ 8 days) and a low volume taper (τ ≤ 4 days), on performance. The difference in training volume between groups during the final taper was not significant although the high volume taper reduced training volume by 50% and the low volume taper by 65%. Training intensity was maintained in both groups and training frequency decreased significantly from a training average of 5.6 ± 1.0 / wk to 4.4 ± 1.8 d / wk during the last week of Taper 2.

Effect on Performance

The effect of a 13 day taper on physical performance was assessed for both a 5 km criterion run and a maximal ramp cycle ergometer test to exhaustion. These performance measures were used to compare the effectiveness of a high volume taper (Group A) and a low volume taper (Group B). Results are tabulated in Table 3 and graphed in Figure 6 and show significant improvement in both groups. The high volume taper significantly improved performance on both the criterion run time and score and the ramp power and score during the first week of taper compared with corresponding pre-taper values. Performance remained significantly elevated in this group during the second week on all measures except for maximal ramp power output. The low volume taper group improved performance significantly on all measures during the first week of taper except for maximal power output. During the second week in the same group maximal power output improved significantly compared with a pre-taper value while all other performance measures remained elevated. A group comparison revealed that the low volume taper group improved maximal power on the cycle ergometer significantly more during the second week of taper compared with the high volume taper group.
Figure 5  Comparison of the profile of training and taper from day 42 onwards for both Group A (n = 4) and Group B (n = 7) exponential decay taper groups. The high volume taper (τ ≥ 8 days) reduced training during taper by 50% while the low volume taper (τ ≤ 4 days) reduced training by 65% compared with the training average.
Table 3 Performance measures for both criterion 5 km run and maximal cycle ergometry immediately preceding and at two times during Taper 2. Values are means ± S.D.

<table>
<thead>
<tr>
<th></th>
<th>Low Volume Taper</th>
<th></th>
<th>High Volume Taper</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre Taper Days</td>
<td>First Week of Taper</td>
<td>Last Week of Taper</td>
<td>Days</td>
</tr>
<tr>
<td>RUN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time (sec)</td>
<td>1167±80</td>
<td>1126±90 *</td>
<td>1093±90 *</td>
<td>1159±60</td>
</tr>
<tr>
<td>Score</td>
<td>790±67</td>
<td>886±20 *</td>
<td>982±40 *</td>
<td>876±16</td>
</tr>
<tr>
<td>CYCLE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Power (W)</td>
<td>433±36</td>
<td>440±26</td>
<td>467±28 *†</td>
<td>394±45</td>
</tr>
<tr>
<td>Score</td>
<td>721±184</td>
<td>759±87</td>
<td>986±31 *</td>
<td>783±165</td>
</tr>
</tbody>
</table>

* Significant improvement compared with Pre Taper.
† Significant group difference.

Effect on Physiological Variables

A 13 day taper significantly increased relative VO2max measured on a maximal ramp test to exhaustion on a cycle ergometer at three separate times: two weeks preceding taper (Pre-Taper) and each week during Taper 2 (see Table 4 and Figure 7). The anaerobic threshold (θan) determined from the same VO2 data mentioned above also increased significantly in the last week of Taper 2 (see Table 4 and Figure 8).

Table 4 Relative VO2max and anaerobic threshold (θan) measured two weeks prior to taper and at two times during Taper 2 (n = 8). Values are means ± S.D.

<table>
<thead>
<tr>
<th></th>
<th>Pre - Taper</th>
<th>First Week of Taper</th>
<th>Last Week of Taper</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2max (ml·kg⁻¹·min⁻¹)</td>
<td>62.9 ± 5.8</td>
<td>67.4 ± 5.6 *</td>
<td>68.6 ± 4.2 *</td>
</tr>
<tr>
<td>θan (% of VO2max)</td>
<td>71 ± 8</td>
<td>73 ± 6</td>
<td>75 ± 6 *</td>
</tr>
</tbody>
</table>

* Significantly larger than Pre -Taper. Paired t-test.
Figure 6  Comparison of performance improvement between both a high volume taper group (n=4) and a low volume group (n=5) and within groups during Taper 2. Performance was compared in the week immediately preceding taper and during the first and second week of taper by actual measures (B and D) and by a scoring method (A and C). Values are means ± S.D. % Increase compared with Pre-Taper 2.

* Significantly greater than Pre-Taper 1 within a group.
† Significant group difference analyzed by Generalized Linear Model at p ≤ 0.05.
Figure 7  A Improvement in $\dot{V}O_2\text{max}$ with Taper 2 in 8 subjects. B Representative breath by breath $\dot{V}O_2$ response to an incremental cycle ergometer test to exhaustion in a single subject. This data was used to determine absolute $\dot{V}O_2\text{max}$.

* Significantly larger than the Pre-Taper 2 value at $p \leq 0.05$. Mean ± S.D.
Figure 8  A Increase in the anaerobic threshold for 8 subjects with Taper 2. B Breath by breath response of one variable ( $\dot{V}_E/\dot{V}O_2$ ) used to measure the anaerobic threshold ($\theta_{an}$) during an incremental exercise test to exhaustion on a cycle ergometer for one subject. *Significantly larger than Pre-Taper 2. Mean ± S.D. $p \leq 0.05$. 

Problem for Group 9
ELEVATED SERUM ENZYME ACTIVITY

Serum creatine kinase (CK) activity did not change significantly in either Taper 1 or Taper 2 although the mean activity decreased in the final blood sampling in both tapers. The average CK activity decreased from 198 ± 102 iU·L⁻¹ to 166 ± 68 iU·L⁻¹ during the last five days of Taper 1. Similarly mean CK activity decreased in a step-wise fashion from 265 ± 212 iU·L⁻¹ to 183 ± 38 iU·L⁻¹ during the final week of Taper 2.

Figure 9 Recovery of serum creatine kinase (CK) activity with both a 10 day Taper 1 (top graph) and 13 day Taper 2 (bottom graph). Mean ± S.D.
MEAN RESPONSE

Physical performance displayed a characteristic pattern of impairment and recovery during heavy training and taper respectively for all subjects (see Figure 10). The average improvement in physical performance during Taper 2 is superior to the improvement in physical performance during a shorter taper (Taper 1). Average training intensity remained constant during both tapers compared to the training average. The frequency of training or number of training sessions per week decreased significantly during both Taper 1 and Taper 2 compared with the training average.

COMPETITION RESULTS

Results of both the mid-season short course triathlon and the Ironman Triathlon competition are shown in the table below. It should be noted that these results were not used as a criterion measure of physical performance. Two triathletes (B.G. and R.C.) achieved a personal best performance during the Ironman Triathlon improving their time by 10 and 30 minutes respectively. Three triathletes (P.Z., G.A. and A.C) with little or no prior competitive triathlon experience performed very well during the Ironman Triathlon with respectable finishing times. Five triathletes did not improve their personal best time in the Ironman Triathlon and one triathlete (D.C.) did not complete the race due to an aggravated injury.

Table 5  Race results for both the mid-season triathlon (Sasview Triathlon) and Ironman Triathlon competition. - Did not compete. * Dropped out.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>Sasview (hr.min.sec)</th>
<th>Ironman (hr.min.sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.W.</td>
<td>3.01.13</td>
<td>10.16.51</td>
</tr>
<tr>
<td>C.G.</td>
<td>3.27.38</td>
<td>10.59.02</td>
</tr>
<tr>
<td>R.C.</td>
<td>3.15.40</td>
<td>10.59.02</td>
</tr>
<tr>
<td>J.P.</td>
<td>3.42.16</td>
<td>11.06.33</td>
</tr>
<tr>
<td>B.G.</td>
<td>3.37.17</td>
<td>11.34.10</td>
</tr>
<tr>
<td>P.Z.</td>
<td>3.37.12</td>
<td>11.57.20</td>
</tr>
<tr>
<td>A.C</td>
<td>3.23.50</td>
<td>12.18.12</td>
</tr>
<tr>
<td>G.A.</td>
<td></td>
<td>12.32.35</td>
</tr>
<tr>
<td>J.C.</td>
<td></td>
<td>12.51.55</td>
</tr>
<tr>
<td>J.B.</td>
<td></td>
<td>13.33.24</td>
</tr>
<tr>
<td>D.C.</td>
<td></td>
<td>*</td>
</tr>
</tbody>
</table>
Figure 10 Average measurement of selected variables for all subjects during training and Taper 1 and Taper 2. Error bars represent one standard deviation from the mean.
MODELING

Each subject's physical performance on both a 5 km criterion run and maximal ramp test to exhaustion were iteratively modeled to best fit predicted performance by changing the system parameters at four points during the study: 1. At the onset of training, 2. during Taper 1, 3. when training resumed, and 4. during Taper 2. The k₁ fitness multiplying factor did not change while k₂, τ₁ and τ₂ all increased during the course of the study the time course of each of which was characterized by a logarithmic regression curve (see Table 6 and Figure 11). The time constants of fitness and fatigue displayed a cyclic change between training and taper. τ₁ tended to increase during taper and τ₂ decreased during taper.

Table 6 Change in system model parameters over the course of 98 days of 2 blocks of training each followed by taper for all subjects. Values are means ± S.D.

<table>
<thead>
<tr>
<th></th>
<th>RUN</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Train</td>
<td>1</td>
<td>1.8 ± 0.2</td>
<td>45</td>
<td>16 ± 2</td>
</tr>
<tr>
<td></td>
<td>Taper 1</td>
<td>1</td>
<td>1.8 ± 0.2</td>
<td>51 ± 3</td>
<td>15 ± 4</td>
</tr>
<tr>
<td></td>
<td>Train</td>
<td>1</td>
<td>1.9 ± 0.2</td>
<td>46 ± 2</td>
<td>21 ± 4</td>
</tr>
<tr>
<td></td>
<td>Taper 2</td>
<td>1</td>
<td>1.9 ± 0.2</td>
<td>52 ± 3</td>
<td>17 ± 3</td>
</tr>
<tr>
<td>CYCLE</td>
<td>Train</td>
<td>1</td>
<td>1.8 ± 0.2</td>
<td>45</td>
<td>16 ± 3</td>
</tr>
<tr>
<td></td>
<td>Taper 1</td>
<td>1</td>
<td>1.8 ± 0.2</td>
<td>51 ± 2</td>
<td>16 ± 3</td>
</tr>
<tr>
<td></td>
<td>Train</td>
<td>1</td>
<td>1.8 ± 0.2</td>
<td>45 ± 2</td>
<td>19 ± 4</td>
</tr>
<tr>
<td></td>
<td>Taper 2</td>
<td>1</td>
<td>2.0 ± 0.4</td>
<td>52 ± 3</td>
<td>17 ± 4</td>
</tr>
</tbody>
</table>
Figure 11 (Following 6 pages) Individual results for each subject. Fitness and fatigue curves generated by matching the pattern of predicted performance from a training model to actual performance on both a 5 km criterion run and a maximal ramp test to exhaustion were generated by computer iteration to achieve a best fit. The pattern of ESEA is shown as well as the pattern of training depicted by individual training impulses for each subject.
Subject: J.P.
Figure 12 Change in system parameters ($k_2$, $\tau_1$ and $\tau_2$) with remodeling at four separate points during training and preceding each taper period for both run and cycle performances. $X \pm S.D.$
DISCUSSION

TAPER

Training Volume

An exponential decay in training volume taper was found to be a very effective form of taper in peaking triathletes for competition. The effect of a ten day taper (Taper 1) on two physical performance measures resulted in a mean 23 W (5%) increase in maximal ramp power on a cycle ergometer, and a mean 46 second (4%) decrease in performance time on a 5 km criterion run compared with performance in the week prior to taper. These results are similar to those reported by Houmard et al. (1994) in runners who improved their performance on a 5 km race by 3% with a 7 day decay taper.

A step reduction in training volume during the same peaking period resulted in a mean 6 Watt (2%) increase and mean 13 second (1%) decrease during the ramp and criterion run respectively, compared with the pre-taper values. These results were not significant and therefore suggest that an exponential decay in training volume taper is an optimal method of peaking an athlete for a specific competition. In a recent review, Houmard and Johns (1994) suggested that tapering is more effective than simply reducing training in the days and/or weeks preceding an event, however, no study has previously compared the two types, step vs. an exponential peaking protocol directly. Group comparison revealed a superior result in the exponential decay group for both the criterion run score and maximal ramp power output during the last five days of Taper 1 lending further support that this method of peaking is more effective.
There may be some inequality in directly comparing the exponential decay taper group with the step reduction in training volume group in Taper 1 due to the fact the actual training volume decrease during this time was not identical. The exponential decay taper group decreased training volume by 50% while the step reduction group decreased their volume by only 30%. A more conclusive comparison may have been made if both groups decreased their training by an identical amount during this ten day peaking period, however, due to non-compliance of some of the subjects this did not occur. It is important to note that taper groups in this study were designated on an individual basis with equal numbers of subjects in each group. In practice this distribution did not occur because one subject was injured and other subjects did not comply to the taper protocol exactly and were either discarded or regrouped post hoc according to the actual training they undertook.

Training intensity and frequency as well as the duration of the peaking period was not different between groups. Human training studies have investigated both the effect of step reduced training (Hickson et al., 1982, Neufer et al., 1987, Houmard et al., 1990) and taper training (Shepley et al., 1992, Houmard et al., 1994) on physical performance, however, no study has ever compared both protocols in a single study to determine which is more effective. Furthermore, all previous studies define training volume by duration of training alone. This is not appropriate since training volume also depends on training intensity which itself is a complex of heart rate intensity and a metabolic factor determined from heart rate. The present study, therefore, represents the first scientific attempt to quantify training volume in a precise and systematic manner and compare both types of peaking to determine an optimal taper protocol. The
results of Taper 1 demonstrate that an exponential decay taper is a superior peaking method, yet a firm conclusion cannot be reached due to the slight difference in training volume between the step and exponential taper groups.

The effect of a high volume ( \( \tau \geq 8 \) days) versus a low volume exponential decay taper ( \( \tau \leq 4 \) days) on physical performance was compared in Taper 2. Results of this 13 day taper produced a significant improvement in almost all criterion measures in both groups. Criterion run time decreased in the final week of Taper 2 compared with pre-taper values by 28 seconds (2%) and 74 seconds (6%) in the high and low volume taper groups respectively. Maximal ramp power was significantly higher in the final week of Taper 2 only in the low volume taper group by 34 Watts (8%). There was also a significant interaction on maximal ramp power between groups during the last week of Taper 2 suggesting that the low volume taper group was a more optimal taper. This conclusion is also supported by the large improvement in the low volume group which was on average 46 seconds faster on a 5 km criterion run compared with the higher volume group in the final week of Taper 2.

The results of both Taper 1 and Taper 2 suggest that additional training volume in the immediate days preceding competition whether it is derived from a reduced training protocol or by an exponential taper with a slower decay time constant may be detrimental to physical performance. It appears that training volume can be reduced by as much as 50% from a previous training average with optimal results. Frequency of training in both Taper 1 and Taper 2 decreased significantly by 1 day/week in the final half of each taper in both groups. Since no concomitant impairment in performance was observed, it may be beneficial for an athlete to take one or two days of complete rest in the week prior to
competition. This may allow maximal recovery of training fatigue to optimize preparation for competition.

A well designed taper protocol must allow for maximal recovery from training fatigue and this may be done by reducing training volume at a certain decay rate. The rate of decay of training volume may be expressed by the following exponential equation:

\[ Y = A \ e^{-i/\tau} \]

where

- \( Y \) = taper TRIMP value
- \( A \) = average TRIMP prior to taper
- \( i \) = day of taper
- \( \tau \) = time constant of decay

Application of the above equation in this study was an easy and effective way by which to plan a taper protocol given only a subject's final training volume preceding taper. The results of this study suggest that a time constant of \( \tau \leq 5 \) days is optimal where training volume is reduced to 63% of previous training volume within 5 days of taper.

**Taper Intensity**

Average intensity of training during both Taper 1 and Taper 2, measured quantitatively via telemetry and heart rate monitor did not change compared with the average intensity during training. Exercise intensity is a key factor in either maintaining (Hickson et al., 1982), or improving performance (Shepley et al., 1992, Houmard et al., 1994), and much of the performance improvement in this study may be attributed to the maintenance of intense training during both Taper 1 and Taper 2. Serial criterion performance repeated 2-3 times / week during taper
provided the necessary intensity of training during taper and a means by which to assess performance in this study.

Some studies have reported that an increase in intensity of training during taper to \( \geq 90\% \) \( \dot{V}O_2\text{max} \) concomitantly with a sharp reduction in training volume was necessary to improve running performance (Shepley et al., 1992, Houmard et al., 1994). Curiously training intensity in the present study did not increase during taper, yet physical performance improved significantly. It is likely, however, that the triathletes in the present study were already training at a very high intensity \( \geq 70\% \Delta \text{HR ratio} \) and that an increase in intensity of training during taper was not necessary. Previous studies also did not precisely quantify training dose, while training intensity was only estimated, and therefore it is uncertain exactly what type of training subjects underwent in these experiments. Results from this study suggest that an optimal taper would require sufficient decay in training volume (\( \leq 50\% \) reduction) while intensity of training is maintained or increased above approximately \( 70\% \Delta \text{HR ratio} \) to observe improvement in physical performance.

**Length of Taper**

The exponential decay 10 day taper group (Group 1, Taper 1) and the 13 day low volume taper group (Group B, Taper 2) with similar volume and intensity of taper training were compared to determine the optimal length of taper. The 13 day taper improved the criterion 5 km run by 74 seconds compared with a 46 second improvement with a 10 day taper. Similarly, maximal ramp power output was increased by 34 Watts in the low volume exponential decay taper group and 23 Watts in the exponential decay taper group. These results suggest that a longer taper may be advantageous in preparation for competition,
which is supported by a study by Costill et al. (1991) who tapered swimmers for 21 days and observed performance improvement right until the end of taper.

It is likely, however, that such a comparison in this study is not completely valid since the 10 day taper preceded the 13 day taper by 6 weeks of heavy training and these triathletes would have been better trained entering Taper 2. However, if the plasticity of these individuals was the same in Taper 1 and Taper 2, the rate at which each subject rebounds from heavy training would be the same and therefore it would take longer to reach a maximal performance. It should also be noted that physical performance in this study was not measured at the end of each taper protocol since each taper was immediately followed by a triathlon competition. The last criterion run and cycle ergometer test were measured 2-3 days before the end of each taper. Any physical performance measure in the day previous to competition that may have impaired race results was avoided. The improvement in physical performance reported in this study may be less than expected if performance was measured on the final day following taper.

**Cardiorespiratory Results**

A 13 day taper (Taper 2) significantly increased relative \( \dot{V}O_{2\max} \) measured on a maximal ramp test to exhaustion in 8 subjects. \( \dot{V}O_{2\max} \) increased from 62.9 ± 5.8 ml·kg\(^{-1}\)·min\(^{-1}\) measured two weeks prior to taper to a significantly higher value of 67.4 ± 5.6 ml·kg\(^{-1}\)·min\(^{-1}\) during the first week of taper to 68.6 ± 4.2 ml·kg\(^{-1}\)·min\(^{-1}\) during the second week of taper. An increase in \( \dot{V}O_{2\max} \) with taper has not been previously reported in the literature although \( \dot{V}O_{2\max} \) has been shown to increase with training (Neufer et al., 1987) and decrease with a prolonged reduction in training volume (Hickson et al., 1982).
Recent taper studies have reported improvement in physical performance with no increase in \( \text{\( \dot{V} \)}O_2\text{\( \text{\( \text{max} \)}} \) (Shepley \textit{et al.}, 1992, Houmard \textit{et al.}, 1994). The accuracy of determined \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) from a constrained run on a treadmill at a specific speed used in the above studies leaves room for doubt about the ability of an athlete to run maximally under such conditions compared with the ergometer incremental test. It also appears that training intensity is a key factor in maintenance of aerobic power (Hickson \textit{et al.}, 1982). The observed 9% increase in \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) observed in this study between the pre-taper and final week of taper is very similar to the 8% increase in maximal power output on the cycle ergometer in the low volume taper group. It is possible that this increase in \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) may have attributed at least in part to the improved performance on the ramp test, and may accompany an increase in physical performance observed in this study.

The underlying physiological mechanism behind the recovery or improvement in \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) during taper observed in this study is unknown. One clue may be related to recovery of ESEA, in particular serum CK activity, observed during Taper 2 as an indicator of the regeneration of disrupted muscle. Heavy training imposes a fatiguing stress on both the cardiorespiratory and peripheral muscular systems (Galun \textit{et al.}, 1988). Houmard and Johns (1994) suggest that alterations in performance that are independent of \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) are associated with adaptation at the muscular level. Muscle disruption that often accompanies heavy training may limit oxygen consumption at the tissue level, while another possible explanation of an increase in \( \dot{V}O_2\text{\( \text{\( \text{max} \)}} \) with taper is an increase in hemoglobin concentration or hematocrit number that may accompany taper (Yamamoto \textit{et al.}, 1988). This in turn may increase the oxygen delivery to the working tissues.
It is also possible that since physical performance was increasing during taper a larger \( \bar{VO}_2 \) would be required to achieve greater maximal power output on the cycle ergometer. Subjects recovering from the physiological and psychological fatigue induced by heavy training are likely to achieve a higher \( \bar{VO}_2 \) max since they can push themselves to a greater extent. Further investigation is required to identify the actual physiological rationale for an increase in \( \bar{VO}_2 \) max with taper since such an occurrence has not been previously reported.

**Elevated Serum Enzyme Activity**

There was no significant recovery of ESEA during either Taper 1 or Taper 2 compared with the pre-taper average for all subjects. The mean serum CK activity did decrease, however, from pre-taper to the last half of both Taper 1 and Taper 2. These results are consistent with studies examining swimmers who reported increased serum CK activity during seasonal training (160 to 448 iU/L) which reduced with taper (80 to 191 iU/L) [Costill *et al.*, 1988; Yamamoto *et al.*, 1988]. In the present study serum CK activity was highly variable between subjects (ranging from 67 to 9200 iU/L) and was mainly dependent on the intensity and nature of training prior to blood sampling. The large variability between subjects introduced a large standard deviation among the means resulting in a non-significant recovery of serum CK activity during either taper period.

Serum CK activity is hypothesized to be positively related to the degree of muscle disruption ([Apple and Rhodes, 1988]). The recovery of serum CK activity in this study may be an indication that muscle regeneration is taking place. No muscle biopsy was taken to assess the degree of muscle disruption and therefore
no firm conclusion may be reached with regards to the role of serum CK activity in assessing the degree of muscle disruption.

OVERTRAINING

Since there is no reliable or quantifiable marker of overtraining it was difficult to assess whether or not any one of the triathletes became overtraining during the course of this study. Subject’s often expressed discomfort and muscle soreness and an overall feeling of fatigue during periods of heavy training, which is a very common phenomenon among endurance athletes (Lehmann et al., 1993). Some athletes during the course of the study became very sick with a cold or flu which may be attributed to a suppressed immune system (Fitzgerald, 1991, Pary-Billings et al., 1992). Subjective feeling of the athlete alone, however, is not sufficient to diagnose a state of overtraining. Possibly the most reliable marker of overtraining is a prolonged decrease in physical performance even following a regeneration period (Fry et al., 1992, Koutekakis et al., 1990). Serial physical performance measurement of running and cycle ergometry provided valuable feedback throughout this study about the effect of a subject’s training on their physical performance. In this manner an athlete could monitor any sudden increment or decrement in physical performance and adjust the intensity of their training accordingly.

One subject in this experiment (D.C.) complained of inflamed and painful Achilles tendons early in the study which became progressively worse as evidenced by a rapid and prolonged decrement in criterion 5 km time even following the first 10 day taper period. This subject was unable to run for 6 weeks prior to the Ironman competition and could not complete the run segment during
the race and had to withdraw from competing further. Curiously, this athlete showed characteristic signs of overtraining evidenced by a decrement in 5 km performance time, yet physical performance on the cycle ergometer actually improved with both tapers (see Figure 11). Furthermore, the serum CK activity of this subject remained within a controlled range during training (maximum 390 iU/L) and recovered during taper. Data from this individual suggests that this athlete was not overtrained, since performance improved on the cycle ergometer, however, an overuse injury sustained as a result of previous overtraining led to a prolonged decrement in criterion run performance. Perhaps this athlete who trained everyday for 4 years prior to this study would have benefited from either absolute or relative rest as a means of restoring performance (Koutekakis et al., 1990). Physical performance in all other subjects showed a characteristic pattern of impairment during heavy training and subsequent recovery during taper suggesting that these athletes were not overtrained.

**DETRAINING**

One important finding was observed in one subject (B.G.) who did not train for a period of nine days due to a summer vacation during the first training period. Performance on the cycle ergometer and the all-out 5 km criterion run measured immediately before and after this period of detraining were both found be impaired. Maximal power output on the cycle ergometer decreased from 369 W prior to detraining to 351 W immediately following absolute rest with no change in \( \dot{V}O_2 \)max. Similarly, the 5 km criterion run performance time increased from 1174 seconds immediately previous to cessation of training to 1261 seconds right after this detraining period. This represents a 5% decrement in maximal cycling performance and a 7% impairment in running performance. These results are similar those reported by Cullinane et al. (1986) who did not observe any
decline in $\dot{V}O_2\text{max}$ after 10 days of rest and Madsen et al. (1993) who did not observe any decline in $\dot{V}O_2\text{max}$ even with a 21% decrease in endurance capacity on a cycle ergometer. Houmard et al., (1992) observed a 9% decrease in running performance after 14 days of detraining which is very similar to the 7% decrease in running performance reported in this study.

Although this subject did not comply with the prescribed training regime, these results provide valuable insight into the effect of detraining on physical performance. Currently the systems model of training prescribes approximately 16 days absolute rest prior to competition for optimal performance, although in practice this is never done. The negative effect of detraining on physical performance is well documented (Neufer et al., 1987, Houmard et al., 1992) and further supported by incidental evidence in one subject during this study. It may be concluded therefore that absolute rest is not an effective way in which to peak an athlete for competition.

RACE RESULTS

The environmental conditions during the 1993 Ironman Canada Triathlon were very hot and windy, and therefore the average finishing time was slower than previous races. This made it difficult to compare current performance with a previous personal best finishing time among the veteran triathletes, although two subjects did improve their Ironman time by 10 and 30 minutes respectively. Five triathletes in this study did not improve their previous personal best time on this course and this is most likely due to the harsh environmental conditions of the race and less likely as a result of inadequate training preparation. The three
novice triathletes completed the Ironman with respectable finishing times while one triathlete did not finish the race due to injury.

The multiplicity of confounding variables that lead to a final performance outcome during an endurance race include such things as psychology, physiology, nutrition, biomechanics, anthropometry, technique and technical considerations (Cedaro, 1993). The number of complicating factors makes it very difficult to predict performance outcome for any one individual athlete. Consider for example anthropometric considerations alone, since 4 of the athletes in the present study weighed above 80 kg which is considered "heavy" for a competitive triathlete. Race results may be due in part to the weight of these individuals demonstrated in a study by Deitrick (1991) who found significant differences in \( \dot{V}O_{2\text{max}} \) between heavy and light triathletes (not observed in the present study). Deitrick concluded that the heavy triathlete is at a physiological disadvantage when competing in endurance events. The excess weight increases the physiological strain at sub maximal speeds and decreases distance running performance as well as the ability to cycle up hills efficiently, independent of absolute cardiorespiratory capacity.

**MODELING**

Physical performance for both the criterion run and maximal ramp test to exhaustion were iteratively modeled to best fit predicted performance by changing the system parameters at four points during the study. It has been suggested that system parameters last for approximately 60 to 90 days before the
process of iterative modeling and resetting the model constants needs to be repeated (Banister, 1991). It was hypothesized in this thesis that the system parameters of the training model do change over time and the growth or decay of each individual parameter may be characterized by a specific time constant.

Remodeling at the onset of training and during both taper periods revealed consistent change in system parameters for both run and cycle performance in all subjects. The fitness multiplying factor remained constant at \( k_1 = 1 \) while the fatigue multiplying factor \( k_2 \) tended to increase during the course of the study from a mean of \( 1.8 \pm 0.2 \) to \( 1.9 \pm 0.2 \) for the criterion run, and up to \( 2.0 \pm 0.4 \) for maximal cycle ergometry for all subjects. This increase in \( k_2 \) may be interpreted to mean that training fatigue is becoming more and more dominant on physical performance as training progresses. The fatigue multiplying factor \( k_2 \) determines how much fatigue will be incurred from each daily training impulse. As fatigue builds with repeated days of intensified training, physical performance is now determined to a greater extent by fatigue. During the course of three months, the fatigue incurred from repeated workouts will be greater than at the onset of training and will require a longer regeneration period for the subject in order for him to effectively recover. This makes practical sense since the harder and longer an athlete trains, the longer the taper period required to fully recover from heavy training (Houmard and Johns, 1994, Zarkadas et al., Abstract, 1994).

The time constant of fitness \( (\tau_1) \) and fatigue \( (\tau_2) \) also displayed a tendency to increase during the course of the study (see Figure 11). The pattern of change in the fitness and fatigue time constants may be more appropriately expressed as a cyclic variation between training and taper. The fitness time constant \( (\tau_1) \) tended to increase from approximately 45 days during training to
51 days during taper. This suggests that with taper an athlete’s fitness will decay more slowly and fitness will remain elevated despite a relative reduction in training volume. The fatigue time constant (τ2) showed the opposite pattern in that it decreased from a range of approximately 16 to 21 days during training to 15 to 17 days during taper. The reduction in τ2 suggests that during taper fatigue decays more quickly leading to improvement in physical performance during taper. The plasticity of an individual athlete to training may be characterized by the rate at which the time constant of fatigue decays and the rate at which the time constant of fitness is maintained or increased during the taper period. The more rapid the separation between fitness and fatigue curves, determined by their respective time constants τ1 and τ2, the more quickly an athlete may rebound from heavy training and the more plastic the individual.

Although the change in system parameters is not very dramatic it may have important consequences in determining the nature of taper required to optimize performance in endurance athletes. For example, if the remodeled parameters for Taper 1 (τ1 = 51, τ2 = 15, k1 = 1 and k2 = 1.8) are used to determine the critical point when the increment to fatigue begins to exceed that of fitness then

\[ \mu = \frac{\tau_1 \tau_2}{\tau_1 - \tau_2} \ln \frac{k_2}{k_1} = 12 \text{ days} \]

where \( \mu \) is the number of days of taper required prior to competition.

In this example 12 days was determined to be the optimal length of taper which corresponds well with the 10 day peaking period conducted during Taper 1. Although a significant improvement was observed after 10 days of taper it is very probable that 12 days may have been an optimal period and may have produced an even better result.
CONCLUSION

The present study was the first scientific attempt to quantify and compare both an exponential decay taper with a step reduction in training volume protocol and to compare two different exponential peaking methods directly. The results of this study suggest that an exponential decay taper is a very effective way of peaking an athlete for competition. Although the findings in this study offer guidelines for designing an effective taper, it remains to be demonstrated whether an optimal taper schedule exists. This may be difficult to determine as there is a multiple number of variables to manipulate and the plasticity of each athlete is highly likely to be different suggesting that every athlete may benefit from an individualized taper program. Future experiments may be improved by a large well controlled cohort of subjects where athletes are participating in a single sport endurance event, unlike the present study where results were confounded by participation in three different activities. The potential of modeling performance from training may provide valuable insight into the training process and especially the nature of taper required to optimize performance.
REFERENCES


APPENDIX A

Simon Fraser University

Subject Questionnaire

NAME: ____________________________ AGE: _______

HEIGHT: ________ (cm) WEIGHT: ________ (kg)

Are you currently registered for the Canadian Ironman? ______(yes/no)

Have you completed an Ironman triathlon before? ______(yes/no)

If yes please state the how many Ironmen you have completed and give the time and year of your best Ironman performance as well as the time and year of the most recent Ironman performance if not the same.

________________________________________________________________________

________________________________________________________________________

What time do you expect to finish the Canadian 1993 Ironman? _______

Please list the short course triathlon(s) that you expect to compete/participate in from May 24 to Aug 28 and their respective dates.

________________________________________________________________________

________________________________________________________________________

Best 5 km run time ________
APPENDIX B
MAY 1993

Friday 28

Swim ______ km time ______ file #______ intensity____ av. HR______

Bike ______ km time ______ file #______ intensity____ av. HR______

Run ______ km time ______ file #______ intensity____ av. HR______

Criterion run time _________ min.
Ramp performed _________ (yes or no)
Morning heart rate _________
Comments: __________________________________________

Saturday 29

Swim ______ km time ______ file #______ intensity____ av. HR______

Bike ______ km time ______ file #______ intensity____ av. HR______

Run ______ km time ______ file #______ intensity____ av. HR______

Criterion run time _________ min.
Ramp performed _________ (yes or no)
Morning heart rate _________
Comments: __________________________________________

Sunday 30

Swim ______ km time ______ file #______ intensity____ av. HR______

Bike ______ km time ______ file #______ intensity____ av. HR______

Run ______ km time ______ file #______ intensity____ av. HR______

Criterion run time _________ min.
Ramp performed _________ (yes or no)
Morning heart rate _________
Comments: __________________________________________
Then the exponential:  \( y = Ae^{-bx} \)

where:  \( y(x) = Y - L \)  and:  \( Y = \) measured performance  

(in appropriate units)

\[ \begin{align*} 
A &= a - L \\
X &= CP \text{ points} \\
CPWR &= 1000 \text{ points} \\
EPP &= 0 \text{ points}
\end{align*} \]

Thus:

\[ Y = Ae^{bx} \]

\[ Y - L = (a - L)e^{b \cdot 1000} \]

\[ \frac{Y - L}{a - L} = e^{-b \cdot 1000} \]

\[ \ln \left( \frac{Y - L}{a - L} \right) = -b \cdot 1000 \]

\[ \ln \left( \frac{a - L}{Y - L} \right) = b \cdot 1000 \]

\[ b = \frac{1}{1000} \ln \left( \frac{a - L}{Y - L} \right) \]

Thus \( CP(x) \) for any Criterion Performance \( Y(x) \):

\[ x = \frac{-1}{b} \ln \left( \frac{a - L}{Y - L} \right) \]

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