TRAINING TECHNIQUES TO IMPROVE CYCLING PERFORMANCE IN WELL-TRAINED CYCLISTS

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"Our deepest fear is not that we are inadequate, our deep fear is that we are powerful beyond measure. It is our light, not our darkness, that most frightens us. We ask ourselves, who am I to be brilliant, gorgeous, talented, and fabulous? Actually who are you not to be? You are a child of God. Your playing small doesn't serve the world. There's nothing enlightening about shrinking so that other people won't feel insecure around you. We were born to manifest the glory of God that is within us. It is not just in some of us, it's in everyone. And as we let our own light shine, we unconsciously give other people permission to be the same. As we are liberated from our fears, our presence automatically liberated others"

Dr. Martin Luther King Jnr.

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DECLARATION

I, Zuko Ntsele Kubukeli, hereby declare that the work contained in this thesis is my own in conception, design and execution except where otherwise acknowledged. Furthermore, this work is original and has not been presented for a higher degree at this institution or elsewhere.

Signed.....

Date.....

Place.....

List of Publications

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ABSTRACT

Training techniques to improve cycling performance in well-trained cyclists.

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While the physiological adaptations to endurance training have been extensively characterised in previously untrained subjects, far less is known about the responses to sustained high intensity interval training (HIT) in well-trained and elite athletes. Therefore, the aim of the thesis is to better understand the physiological responses of well-trained cyclists to selected laboratory performance tests and road racing during training and taper.

The physiological responses to a variable-intensity test, a conventional linear ramp test and their relation to road cycle racing performance in well-trained cyclists, training ~ 200 km/wk with W_{peak} values of ~ 400 W was examined. The effects of 6 HIT sessions, consisting of 3-4 x 15 min rides at 70% of W_{peak} on simulated, 100 km time-trial (TT_{100}) performances in addition to their endurance training was also examined. Previously, the reported training durations and intensities of 16 elite triathletes over the six weeks leading to the national championship were analysed. Following the HIT program, the effects of a 50% reduction in the frequency of high-intensity interval training (HIT) over 1, 2 and 3 weeks in two groups of 16 and 6 male competitive cyclists ($VO_{2peak} \geq 65$ mL.min^{-1}.kg^{-1} or 5-7 W.kg^{-1}) were examined. Heart rate data, peak power, muscle strength, electromyographic measurements, fuel substrate utilisation and lactate values at submaximal work loads, road cycle racing and laboratory 100-km time trial performances recorded during the various phases in their training and taper was compared to establish whether there are physiological differences in the responses of athletes similar in

cycling ability and secondly if these responses are altered by training and taper. After 6 weeks of habitual endurance training, the same laboratory measurements were made.

The correlation between the cyclists' initial declines in heart rate following both the linear ramp and the variable-intensity exercise tests, and their rankings after the road race was significant (both $r = -0.93$, $P \leq 0.01$) and more rapid in the better cyclists. Road cycling performances also tended to be related to the cyclists' W_{peak} and % of W_{peak} at 4 $mmol \cdot L^{-1}$ plasma lactate concentration (both $r = -0.75$, $P \leq 0.05$).

Training performed in the 2-week taper period before the competition was reduced to a mean of 48% (39 to 60%, $P = 0.0001$) of the training performed in the 6 weeks prior to the competition. Training time was reduced at all the intensities and surprisingly training at the hard intensity was significantly reduced during the 2-weeks prior to the competition. However, the correlation between the reduction in training time "taper" in the 2-week period and personal best performance in an Olympic-distance triathlon was not significant ($r = 0.40$) in this group of triathletes.

Six HIT sessions at 70% of W_{peak} had no immediate effect on 100-km time trial (TT_{100}) performance. Time trial performances only improved significantly from 38.1 ± 3.0 km/h pre-HIT to 40.2 ± 2.7 and 41.1 ± 2.6 km/h ($P \leq 0.05$) when HIT frequency was reduced by 50% over 1- 2- and 3-weeks. The improvements in TT_{100} were due to the cyclists being able to sustain significantly higher absolute (228 ± 32 to 258 ± 41 W) and relative work rates ($58 \pm 6\%$ to $66 \pm 7\%$ of pre-HIT W_{peak} values during the TT_{100} rides (both $P \leq 0.001$) after a 2-week taper. There was a significant linear relationship between W_{peak} and 2-week taper TT_{100} performance ($r = 0.81$; $P \leq 0.05$) but the relationship did not reach significance for the cycle race ($r = 0.74$; $P = 0.08$) and VO_{2peak} during taper ($r = 0.72$; $P = 0.11$).

The isokinetic peak force during a maximal voluntary contraction (MVC) was significantly reduced by the HIT program from 695 ± 148 to 572 ± 131 N and further reduced to 555 ± 107 N after 2 weeks of taper ($P \leq 0.03$). The electromyographic (EMG) activity and spectral shifts during the TAPER MVCs were not significantly affected by the intervention, although there was a tendency during the taper measurements for the spectral shift changes to be lower than during the PRE, POST and 6 WEEKS MVCs measurements. EMG activity, plasma lactate concentrations, rates of carbohydrate oxidation and heart rate responses to submaximal cycling workloads were not significantly altered by training and taper. After 6 weeks of habitual endurance training the TT_{100} performances, and the physiological responses to submaximal workloads and MVCs were reduced to pre-HIT training values.

In conclusion, it can be concluded from this series of studies is that performances in mass-start road races may not only be related to the cyclists' ability to sustain a given power output but also to recover rapidly from high work rates. The triathletes in the present study reduced their high-intensity training during the 2-weeks prior to the competition significantly while scientific recommendations suggest a maintenance or increase in high-intensity training during taper. Short term (4-6 weeks) HIT followed by a 2-week taper significantly improved cycling endurance performance in well-trained cyclists. These improvements in performance after training and taper were associated with higher sustained absolute and relative workrates, reduced isokinetic peak force, and faster recoveries after high-intensity workloads. Proposed mechanisms on how tapering optimises performance remain speculative, it is suspected that optimum taper depends on the intensity of the athletes' preceding training and their need to recover from exhaustive exercise in order to compete.

ABBREVIATIONS

3-HAD	3-hydroxyacyl CoA dehydrogenase	ADP	adenosine diphosphate
AK	adenylate kinase	ATP	adenosine triphosphate
βm	skeletal muscle buffering capacity	βm_{titr}	βm by titration
CK	creatine kinase	CRI	cycling repetitions interval
CS	citrate synthase	CV	coefficient of variation
F-6-P	fructose-6-phosphate	GAPDH	glyceraldehyde-3-P dehydrogenase
GP	glycogen phosphorylase	GPX	glutathione peroxidase
GR	glutathione reductase	GS	glycogen synthase
HI	high intensity	HIT	high-intensity interval training
HK	hexokinase	HR	heart rate
HR_{peak}	peak heart rate	LDH	lactate dehydrogenase
LI	low intensity	MDH	malate dehydrogenase
MVC	maximal voluntary contraction	Min	minutes
PCr	phosphocreatine	PFK	phosphofructokinase
PHOS	glycogen phosphorylase	POMS	Profile of Mood State
PPF	peak pedal frequency	PK	pyruvate kinase
RM	repeated maximum contractions	SDH	succinate dehydrogenase
Sec	seconds	SOD	superoxide dismutase
SR	sarcoplasmic reticulum	T_{SMF}	supra-maximal run to fatigue
TTF	time to fatigue	TT	time trial
UCT	University of Cape Town	VO_{2max}	maximal oxygen consumption
VO_{2peak}	peak oxygen consumption	W_{peak}	peak watts

CHAPTER 1

INTRODUCTION

There often is a "weak-link" between the research that is carried out in the laboratories and what athletes are practicing in the field. It is widely accepted that there is a need for laboratory-based exercise tests that simulate and mimic competition or field situations. When athletes are examined in a laboratory, they often perform progressive exercise tests to exhaustion. These conventional linear exercise ramp tests to exhaustion are designed to measure rises in plasma lactate concentration and peak rates of oxygen consumption. Differences in lactate concentrations at sub-maximal work rates and in VO_{2peak} values at exhaustion serve as indicators of whether athletes are "recreational", "competitive" or "elite". However, lactate concentrations and VO_{2peak} values are poor predictors of endurance performance among more homogenous groups of athletes.

Conversely, in the competition arena, especially at the highest level, the pace or power required is never increased or decreased linearly or exponentially as the time and distance elapse. Field and competition endurance performance is characterized by large, stochastic changes in power and intensity that are heavily influenced by tactics (Jeukendrup et al. 2000; Padilla et al. 2000), environmental conditions (Palmer et al. 1994; Padilla et al. 1999), competition standard and performance requirements during competition. The widely used laboratory tests are short, progressively fatiguing tests that tell us little about the competition-fitness of well-trained individuals.

Owing to the limited literature that is available on the physiological basis of endurance training techniques employed by professional cyclists, Hawley and Stepto (2001) have presented a theoretical model on the major training-induced adaptations in skeletal muscle that are likely to determine and improve performance capacity in elite cyclists. These authors concluded that the high volume and high intensity training performed by these athletes results in small measurable physiological adaptations while performances in competition puts these athletes in a select elite group.

In the increasingly competitive sports environment, the smallest edge can create the most telling advantage. To secure it demands tireless research and profound analysis. The major problem facing today's scientists, is recruiting and then convincing already-elite athletes to firstly, give of their time for scientific testing and secondly, to comply to a laboratory-derived training program. There are very few anecdotal accounts of athletes whose successes on the sports arena have been based on laboratory training and testing. On the contrary, the tests are often disruptive to the athlete's macro or micro training program and too invasive to be carried out with the necessary frequency to have any real value to the athlete and coach.

Training questionnaires and diaries provide the scientist with data that can be used to better design research that is both relevant and applicable and for the athlete and coach, and the data can be used to make training systematic with the primary aim of enhancing competition performance. The reliance on day to day subjective recording of training and questionnaire-based recall research is not ideal but often, is the only way of quantifying the training load of that athlete. Many different types of training diaries and questionnaires have been validated with an incorporation of the subjective interpretation of intensity. However, training diaries can pose problems if athletes are required to comply over a lengthy training period since the quality of the data generated can be questionable and be quite substantial. Poehlitz (1988) has

suggested that diary sheets should be designed to allow athletes to record their responses directly into boxes ready for entry into a computer.

O'Toole (1989) in her study of recreational and moderately- trained triathletes in preparation for the Hawaii Ironman Triathlon, attempted to find systematic differences in training, particularly in distance and pace, according to finish time. The time spent training and the intensity of the training by athletes, similar in ability, varied by as much as 50% of their training volume. Based on training data from the athletes in the study she was able to quantify the minimum amount of training required to complete the race in under 10.5 hours, while pointing out that adequate amounts of training are not a guarantee of successful completion of the Hawaii Ironman and cannot make up for inappropriate race strategy. Many of the athletes in her study were able to complete the race on very little training while others trained almost twice the recommended distances.

Many athletes are often in a predicament of wanting to reduce the negative fatigue effects of training while also wanting to maximize the positive effects of training with the primary aim of enhancing performance. Often as the competition date draws nearer, athletes cannot resist the urge to train harder and more frequently. To do the opposite takes confidence in one's training and ability.

There are numerous anecdotal accounts of athletes, especially endurance athletes, that manipulate the various components of training namely; volume, frequency and intensity, in order to achieve a peak in performance during competitions throughout a season. The training is usually altered in the period, typically days and/or weeks, prior to a major competition. Common to most athletes' training routine is a reduction in training volume and frequency

leading up to a competition. These reductions in training volume and frequency have been shown to be accompanied by slight increases (Costill et al. 1985; Johns et al. 1992; Shepley et al. 1992; Houmard et al. 1994; Mujika et al. 1996b), or decreases (Hickson et al. 1985; Neufer et al. 1987) or maintenance (Houmard et al. 1989, 1990; Wittig et al. 1989; Hooper et al. 1998, 1999; Mujika et al. 2000) in performance.

It is still under debate as to what length of taper is optimal, what should be reduced during taper and what combination of the training components, i.e. frequency, intensity, volume, should be manipulated. More recently Zarkadas et al. (1995) and Banister et al. (1999) have raised questions to whether the reduction in training should be in a single step, progressive or exponential fashion. For the most part, research in the area of tapering and the effects of training on performance has played a retrospective role, trying to explain why certain already accepted practices work.

Recently Jeukendrup et al. (2000) have suggested nomenclature and criteria as to what constitutes an 'untrained', 'trained', 'well-trained', 'elite' and 'world class' cyclists. These investigators propose classifying these athletes/subjects largely on the amount of training undertaken, racing status and physiological characteristics from selected laboratory tests. In line with these guidelines the cyclists in the current series of investigations are mainly from the 'well-trained' group with a few cyclists in the 'trained' and 'elite' groups.

Training research on the 'untrained' and 'trained' athlete population is extensive and well reviewed, while research on the 'well-trained', 'elite' and 'world class' athlete is limited. Further research will be of great benefit to both athlete and scientist if the athlete's training can be accurately quantified and for laboratory performance tests to be a closer simulation of field performances.

CHAPTER 2

REVIEW OF LITERATURE

INTRODUCTION TO THE REVIEW OF LITERATURE

The following chapters review the literature that has contributed significantly to our knowledge of the effects of training on physiological variables, athletic and maximal performance in previously untrained individuals and already trained athletes.

The first two sections (2.1 and 2.2) review the effects of endurance training and interval training in previously untrained individuals. These sections focus mainly on the physiological, biochemical and metabolic changes that are thought to be modified by training and then translate to improvements in performance. Already endurance-trained individuals exhibit different training adaptations to those reported in previously untrained individuals. The primary objective for training in this population is to improve athletic performance. A reduction in training or taper prior to a competition is considered by athletes, coaches and scientists to further enhance these improvements in performance. The literature on the effects of resistance training on aerobic performance is not conclusive. The literature on these two topics is reviewed in the latter sections (2.3 and 2.4).

CHAPTER 2.1

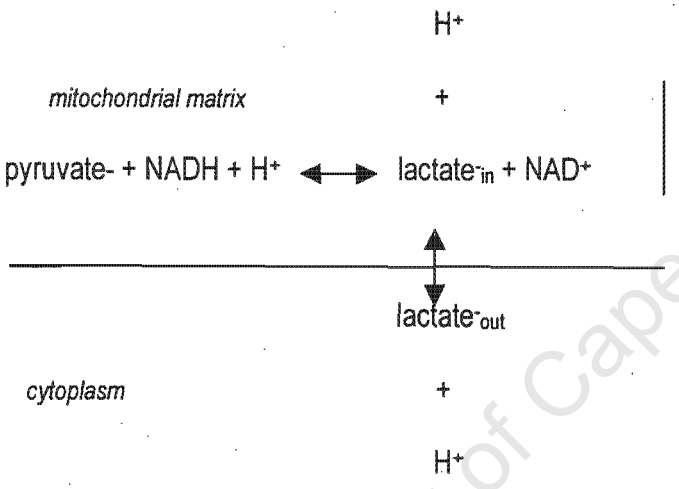
EFFECTS OF ENDURANCE TRAINING IN PREVIOUSLY LESS TRAINED INDIVIDUALS

Adaptations to endurance training in previously less trained individuals have been well characterised. Continuous low to moderate intensity exercise over several months primarily improves "aerobic" capacity. Improvements in peak oxygen uptake (VO_{2peak}) after endurance training are associated with changes in cardiovascular, muscular and metabolic responses to exercise (Foster et al. 1978; Hardman et al. 1986; Green et al. 1991). Cardiovascular changes with endurance training include increases in working muscle capillary density, rises in blood volume and resultant decreases in heart rate at similar absolute exercise intensities (Gollnick et al. 1973; Ingjer et al. 1979; Saltin and Gollnick 1983; Gollnick 1986). Muscular changes with endurance training include greater muscle glycogen storage (Greiwe et al. 1999), increases in Na^+-K^+ ATPase pump activity (McKenna et al. 1993; Madsen et al. 1994; Green et al. 1999), and rises in most mitochondrial enzymes, with little change in glycolytic enzymes (Holloszy 1975; Hamel et al. 1996).

Studies of the metabolic effects of increases in muscle mitochondrial content with training have been reviewed elsewhere (Henriksson 1977; Benzi 1981; Saltin and Gollnick 1983; Holloszy and Coyle 1984; Gollnick 1986). Increases in muscle mitochondrial content improve respiratory control sensitivity (Holloszy and Coyle 1984; Gollnick 1986). Lower cytosolic adenosine 5 diphosphate concentrations required for given rates of oxidative phosphorylation, more closely match the activation of glycogenolysis by displacements of the creatine kinase and adenylate kinase equilibria to the demands of the mitochondria for pyruvate$^-$ and H^+.

Similar arguments also apply to the mitochondrial re-oxidation of reduced cytosolic nicotinamide adenine nucleotide (NADH + H^+) via the malate-aspartate shuttle. An increased

mitochondrial content decreases the cytosolic NADH + H^+ concentrations required for given rates of reducing equivalent transport into mitochondria. Lower cytosolic pyruvate and NADH + H^+ concentrations limit the production and efflux of lactate by displacements of the lactate dehydrogenase and lactate translocase equilibria at high rates of carbohydrate oxidation as briefly described in the diagram below (Green et al. 1983, 1992b; Sahlin and Henriksson 1984; MacRae et al. 1995; Spengler et al. 1999).



Lower plasma lactate concentrations at similar relative work rates after endurance training are also due to a greater mitochondrial capacity to oxidise fat (Henriksson 1977). Higher rates of fat oxidation may potentially help to extend prolonged moderate endurance exercise by 'sparing' body carbohydrate stores.

CHAPTER 2.2

EFFECTS OF INTERVAL (SPRINT) TRAINING IN PREVIOUSLY UNTRAINED INDIVIDUALS

In contrast, high intensity interval (sprint) training may have less effect on the muscle mitochondrial content of previously untrained individuals and more effect on their muscle glyco(geno)lytic capacity than endurance training. While interval training does not always

increase muscle mitochondrial enzyme activities, it invariably raises the activity of one or more of the muscle glyco(geno)lytic enzymes (Table 2.1; Hickner et al. 1997). Comparisons of interval training with endurance training in previously untrained individuals have also suggested that interval training has a greater effect on enzymes associated with glyco(geno)lysis than on mitochondrial enzymes. All three studies in Table 2.2 showed that interval training increased phoshofructokinase or adenylate activities without raising mitochondrial, citrate synthase, succinate dehydrogenase, malate dehydrogenase or 3-hydroxyacyl CoA dehydrogenase activities. Rises in mitochondrial enzyme activities or reductions in plasma lactate concentrations and respiratory exchange ratios at the same relative exercise intensity were only observed in the corresponding endurance trained groups (Table 2.2).

In two of the studies in Table 2.2, only endurance training improved VO_{2peak} but, in the other study by Gorostiaga et al. (1991), interval training produced the greatest increase in VO_{2peak}. In the latter study, subjects either performed thirty 30-s rides at 100% of VO_{2peak} in 30 min or they cycled continuously at 50% of VO_{2peak} for 30 min. Unfortunately, comparisons between training regimes have to be interpreted with caution. Fifteen minutes of intermittent exercise at 100% of VO_{2peak} is a far greater training stimulus than 30 min of continuous exercise at 50% of VO_{2peak} (Hawley and Hopkins 1995; Hopkins et al. 1999; Stepto et al. 1999). Whereas trained cyclists can ride for hours at 50% of VO_{2peak}, they can cycle for only a few minutes at 100% of VO_{2peak}.

Table 2.1. Effect of interval training on muscle enzyme and performance changes in previously untrained individuals.

References	(n)	Training regimes	Enzyme changes	Performance changes
Thorstensson et al. 1975	(4)	~30x 5 s sprints @ ~22km/h with ~40s rest, 3-4x/wk for 8 wks	Mg^{2+} ATPase, AK & CK ▲	power output ▲
Saltin et al. 1976	(13)	30 – 40 s 1 leg sprints with 90s rests @150% of 1 leg VO_{2max} , 5x//wk for 4 wks	SDH (19%) ▲	$VO_{2\,peak}$ (11-15%) ▲
Costill et al. 1979	(5)	2x 30 s 1 leg max isokinetic exercise, 4x/wk for 7 wks	GP, PFK, SDH, MDH & CK ▲ ; LDH ▬	mean power output & peak torque ▲
Fournier et al. 1982	(12)	20 - 60 min sprint training @ 100% HR_{peak} 4x//wk for 12 wks	PFK▲ ; SDH ▬	$VO_{2\,peak}$ ▲
Roberts et al. 1982	(4)	8x 20-30 s sprints @ 90% max speed with 2 min rec., 3x/wk for 5 wks	GP, PFK , GAPDH , LDH & MDH ▲ ; SDH ▬	treadmill performance test ▲
Jacobs et al. 1987	(11)	2 – 6x 15 & 30s max. sprints, 2 –3x/wk for 6 wks	PFK & CS ▲	Wingate test ▬
Simoneau et al. 1987	(19)	25x 30min of endurance training @70% HR_{max} reserve and 35x 15 – 90s sprints @ 60 and 70% W_{peak} in 15 wks	HK , PFK, LDH, MDH, HADH & OGDH ▲ ; CK ▬	$VO_{2\,peak}$, 10s & 90s max. output ▲
Cadefau et al. 1990	(16)	30-80 m sprints & 100-500 m runs @100% of max, 3-4x/wk for 32 wks	GS, SDH, GP, PFK & PK ▲ ; LDH & CK ▬	athletic performance ▬
Linossier et al. 1993	(10)	2x series of 8x 5 s sprints @ 80% F_{max} with 55s rest and with 15min rest between series, 4/wk for 7 wks	PFK & LDH ▲ ; CS, 3-HAD & HK▬	F_{max} & W_{peak} Wingate test ▲ ; $VO_{2\,peak}$ ▬
Sharp et al. 1986	(8)	8x 30s cycling sprints @ HPR with 4min rec., 4x/wk for 8 wks	PFK ▲	$VO_{2\,peak}$, peak torque & total work ▲
Hellsten et al. 1996	(11)	15x10s sprints with 50s rec. @300-400% VO_{2peak}, 3x/wk for 6 wks then 2x/day for 1wk	PFK, LDH, CS, HAD, CK, GPX & GR ▲ ; SOD ▬	cycling performance ▬
Linossier et al 1997a	(8)	15x 5 s sprints with 35s recovery x2 sessions with 15min rec., 4/wk for 7 wks	GP (9%), PFK (17%), LDH (31%) & AK (18%) ▲ ; CS, HAD & CK ▬	$VO_{2\,peak}$ (3%) & W_{max} (28%) ▲
Dawson et al. 1998	(9)	20- 40x (30-80m) sprints @ ~95% max. speed with 2-4 min rec., 2-3x/wk for 6 wks	GP▲ ; PFK & AK▬; CS ▼	$VO_{2\,peak}$ & sprint performance ▲
McDougall et al. 1998	(12)	4 – 10x 30s sprints (Wingate) with ~3min rec., 3x/wk for 7 wks	HK, PFK, CS, SDH & MDH ▲	$VO_{2\,peak}$, peak & mean power output ▲

Initial VO_{2peak} values of the subjects in most of these studies were about 50 mL/min/kg. GP-glycogen phosphorylase, GS-glycogen synthase, HK-hexokinase, PFK-phosphofructokinase, GAPDH-glyceraldehyde-3-P dehydrogenase. PK-pyruvate kinase and LDH-lactate dehydrogenase are glycolytic enzymes. CS-citrate synthase, SDH-succinate dehydrogenase, MDH-malate dehydrogenase and HAD-3-hydroxyacyl CoA dehydrogenase are mitochondrial tricarboxylate cycle and β-oxidation enzymes. CK-creatine phosphokinase and AK-adenylate kinase are enzymes that assist in the provision of energy in the first few seconds of a sprint. GPX-glutathione peroxidase, GR-glutathione reductase and SOD-superoxide dismutase are enzymes involved in anti-oxidant defence. ▲ ,▼ , and ▬ indicate increased, decreased and not significantly changed, respectively. Recovery (rec.) is active rest while Rest is complete rest.

Table 2.2. Comparison of the effects of interval or endurance training on muscle enzyme and exercise performance changes in previously untrained individuals.

References	(n)	Training regimes	Enzyme changes	Performance changes
Fournier et al. 1982	(6)	20 – 60min sprint training, 4x/wk for 12 wks	PFK (21%) ▲ ; SDH —	$VO_{2\,peak}$ (10%) ▲
	(6)	2x ~20min runs with 5min rec. @ 60 – 90% of HR_{max}, 4x/wk for 12 wks	PFK & SDH (42%) ▲	$VO_{2\,peak}$ (12%) ▲
Gorostiaga et al. 1991	(6)	15 x 30s sprints at 100% VO_{2peak} in 30min, 3x/wk for 8 wks	AK (25%) ▲ ; CS & [Lac] & RER @ submax VO_{2peak} —	$VO_{2\,peak}$ (9-16%) ▲
	(6)	30 min rides at 50% of VO_{2peak}, 3x/wk for 8 wks	CS (25%) ▲ ; AK—; [Lac] & RER @ submax VO_{2peak} ▼	$VO_{2\,peak}$ (5-7%) ▲
Green et al. 1999	(9)	3 sets of 6-8 RM's, 3x/wk for 12 wks	PHOS ▲ ; PFK, HK, MDH & HAD —	VO_{2peak} —
	(7)	1x 2h ride/day @68% VO_{2peak}, 5-6x/wk for 11 wks	PFK, HK (28%), MDH (27%) & HAD ▲ ; PHOS & LDH—	VO_{2peak} (15%) ▲

Initial VO_{2peak} values of the subjects in most of these studies were again about 50 mL/min/kg. RM's are repeated maximum contractions. HK-hexokinase, and PFK- phosphofructokinase are glycolytic enzymes. CS-citrate synthase, SDH-succinate dehydrogenase, PHOS-glycogen phosphorylase, MDH-malate dehydrogenase and HAD-3-hydroxyacyl CoA dehydrogenase are mitochondrial tricarboxylate cycle and β-oxidation enzymes. AK-adenylate kinase is an enzyme that catalyses an amplification reaction to activate glyco(geno)lysis. ▲ , ▼ , and — indicate increased, decreased and not significantly changed, respectively.

Generally higher exercise intensities in interval training than in endurance training have led to a question of whether interval training inter-converts slow-twitch (type I), fast-twitch oxidative (type IIa) and fast-twitch glycolytic (type IIb) muscle fibres. While endurance training has little effect on type I, IIa and IIb fibre compositions (Saltin and Gollnick 1983), there are conflicting reports on changes in fibre types after interval training. Some groups have found either no change in fibre types or a transformation of type II to I fibres (Table 2.3). Other groups have observed a conversion of type I to IIa fibres with, in some cases, a transformation of type IIb to IIa fibres or an increase in IIa myosin heavy chain isoform expression within type IIb fibres (Table 2.3). It is also possible that with training there is an increased myosin light chain or ATPase expression designed to increase the cross-bridge cycle rate. The changes could be mechanical rather than structural. In addition, the inconsistent 6-10% changes in fibre types after interval training could have resulted from regional differences in the composition of fibre types within a muscle (Allemeier et al. 1994; Kernell 1998). Such changes are within the ~12% coefficient of variation of fibre type determinations in needle-biopsies from the same muscle (Blomstrand and Ekblom 1982) and may not be entirely due to alterations in fibre types.

It is also questionable whether endurance or interval training significantly affects muscle sarcoplasmic reticulum (SR) Ca^{2+} re-uptake capacity. Most studies have shown that muscle SR Ca^{2+} ATPase (pump) activity is unaffected by training (Table 2.4). The only exceptions were the studies by Green et al. (1998) and Hunter et al. (1999). Green et al. (1998) found that interval training helped to maintain SR Ca^{2+} re-uptake capacity during exercise through a mechanism that was independent of any change in SR Ca^{2+}-ATPase activity. Hunter et al. (1999) observed that interval training improved the low SR Ca^{2+} ATPase activities of nine elderly women but had no effect on the higher SR Ca^{2+} ATPase activities of ten younger women.

Table 2.3. Effect of interval training on muscle fibre-type transformations and exercise performance changes in previously untrained individuals.

References	(n)	Training regimes	Fibre-type transformations and changes	Performance changes
Thorstensson et al. 1975	(4)	~30x 5s sprints @ ~22km/h with ~40s rest, 3-4x/wk for 8 wks	No change	power output ▲
Saltin et al. 1976	(13)	30 – 40 s sprints with 90s rests @150% of 1 leg VO_{2max}, 5x/leg/wk for 4 wks	▲ IIa (16%) & ▼ IIb	$VO_{2\,peak}$ (11-15%) ▲
Costill et al. 1979	(5)	2x 30 s 1 leg maximum isokinetic exercise, 4x/wk for 7 wks	IIb ► IIa ◄ I	Not determined
Simoneau et al. 1985	(24)	25 endurance and 35 interval sessions (4 - 10x 15 – 90s) @ ~ 80% of max power for 15 wks	IIb ► I	Not determined
Jacobs et al. 1987	(11)	2 – 6x 15 & 30s max. sprints, 2 –3x/wk for 6 wks	IIa ◄ I	Wingate performance test ▬
Jansson et al. 1990	(15)	3x 30s (Wingate) sprints with 15min rec., 2-3x/wk for 4-6 wks	IIa ◄ I	Wingate performance test ▬
Cadefau et al. 1990	(16)	30-80 m sprints & 100-500 m runs @100% of max, 3-4x/wk for 8 mo	IIa, b, c ► I	athletic performance ▬
Esbjomsson et al. 1993	(11)	15x 10s sprints with 50s rec., 3x/wk for 6 wks then 14x/wk for 1 wk	IIb ► IIa ◄ I	peak power ▬
Linossier et al. 1993	(10)	8x 5 s sprints @ 80% F_{max} with 55s rec. 15min rest then repeat, 4/wk for 7 wks	IIb ► I	$VO_{2\,peak}$ ▬; W_{peak} & Wingate performance test ▲
Andersen et al. 1994	(6)	2 – 3hr/day sprint training @ , 6x/wk for 12 wks	Only IIa ◄ I; but IIb ► IIa ◄ I MHC expression	knee extensor strength & sprint performance ▲
Allemeier et al. 1994	(11)	3x 30s (Wingate) sprints, 2-3x/wk for 6 wks	No change; but IIb ► IIa MHC expression	anaerobic & aerobic performance ▬
Linossier et al.1997b	(7)	15x 5s sprints with 35s recovery x2 sessions with 15min rec., 4/wk for 9 wks	Some IIb ► IIa ► I. Others IIb ► IIa ◄ I	peak power (28%) ▲
Harridge et al. 1998	(7)	24x 3s sprints, 4x/wk for 5 wks, 6th wk 39x 3 s sprints	No change	isometric torque & PPK ▲
Dawson et al. 1998	(9)	20- 40x (30-80m) sprints @ ~95% max. speed with 2-4 min rec., 2-3x/wk for 6 wks	II ◄ I	$VO_{2\,peak}$ & sprint performance ▲
Pilegaard et al. 1999	(7)	2 – 5x 1min 1 leg extensor exercise with 2min rest repeated 3x, 3 - 5x/wk for 8 wks	No change	Peak, mean output (16% & 15%) & endurance (132%) ▲

I, IIa and IIb are slow-twitch oxidative, fast-twitch oxidatve and fast-twitch glycolytic muscle fibres, respectively. Histochemically identified type IIb muscle fibres contain both IIb and IIa myosin heavy chain (MHC) isoforms (Anderson et al. 1994), PPK- peak pedal frequency. ◄ and ► indicate direction of transformation and ▲ , ▬ indicates an improved and no change in exercise performance.

Table 2.4. Effects of training on muscle SR-Ca^{2+} re-uptake capacity and exercise performance changes in previously untrained or moderately-trained individuals.

References	(n)	Training regimes	Physiological changes	Performance changes
Madsen et al. 1994	(39)	25min training 3x/wk for 6 wks @ 93% of HR_{peak}	SR Ca^{2+} ATPase ▬	VO_{2max} & time to exhaustion (75%) ▲
McKenna et al. 1996 (review)		Endurance or strength training	SR Ca^{2+} ATPase ▬	Not reported
Green et al. 1998	(16)	3x 8RM's, 3x/wk for 12 wks	SR Ca^{2+} ATPase ▬, but maintenance of SR Ca^{2+} ATPase activity during exercise ▲ #	VO_{2peak} ▬
Hunter et al. 1999	(19)	9 young and 10 elderly trained 3x 6-8 RM's on quadriceps, 3/wk for 12 wks	SR Ca^{2+} ATPase ▲ *; SR Ca^{2+} uptake ▬	Quadricep strength ▲
	(22)	11 young and 11 elderly controls		

Sarcoplasmic reticulum (SR) Ca^{2+} ATPase's pumps are required for muscle relaxation. # Mechanism of better maintenance of SR Ca^{2+} ATPase activity during exercise after training not known. * SR Ca^{2+} ATPase activity was only increased in 9 elderly women but not in 10 young women. HR_{peak} is peak heart rate. RM's are repeated maximum contractions. ▲ and ▬ indicate increased and not significantly changed, respectively.

Interval training may also not reduce the intra-muscular acidosis that interferes with excitation-contraction coupling in exhaustive exercise (Fitts 1996). While trained sprinters have been shown to have a greater muscle physico-chemical H^+ buffering capacity (β_m) than endurance athletes or sedentary individuals (Sahlin and Henriksson 1984; Parkhouse et al. 1985), such findings have to be interpreted with caution. Cross-sectional studies carry a risk of biological selection. Several groups have found that sprint performances are related to percent of type II fibres (Inbar et al. 1981; Sadoyama et al. 1988; Mannion et al. 1995; Hautier et al. 1996) and that percent of type II fibres correlates with β_m in men (Boulay et al. 1985; Parkhouse et al. 1985; Bouchard et al. 1992; Mannion et al. 1995) and in rats (Weston et al. 1996). More reliable, longitudinal studies have shown that interval training either increased or had no effect on β_m in previously untrained individuals (Table 2.5) but improved power output. Interval training was also found to have no influence on two muscle metabolites that contribute to β_m (Table 2.5). One metabolite was the β-alanylhistidine dipeptide, carnosine. Carnosine and protein histidine residues buffer intra-cellular H^+ ions in the physiological pH range of 6 to 8. Another metabolite was creatine phosphate (CrP). As CrP is broken down to help maintain ATP levels in rapidly working muscles inorganic phosphate (P_i^{2-}) accumulation increases intracellular H^+ buffering capacity.

Unbuffered H^+ then leaves muscle cells via sarcolemmal Na^+/H^+ exchange and H^+-lactate$^-$ co-transport. Several groups have shown that training enhances sarcolemmal monocarboxylate transport (MCT) capacity in rats (McDermott and Bonen 1993; Pilegaard et al. 1993; Baker et al. 1998) and humans (Pilegaard et al. 1994; Bonen et al. 1998; Pilegaard et al. 1999). Pilegaard et al. (1999) found that 8 wk of interval training (Table 2.5) increased human skeletal muscle MCT1 and MCT4 content by about 70% and 30%, respectively.

Table 2.5. Effects of training on muscle fibre H^+ buffering capacity and exercise performance changes in previously untrained individuals.

References	(n)	Training regimes	Physiological changes	Performance changes
Thorstensson et al. 1975	(4)	~30x 5s sprints @ ~22km/h with ~40s rest, 3-4x/wk for 8 wks	CrP ▬	Power output ▲
Sharp et al. 1986	(8)	8 x 30s cycling sprints @ HPR with 4min rec., 4x/wk for 8 wks	β_m (12%) ▲ ; CrP ▬	Power output ▲
Bell & Wenger 1988	(9)	15 – 20x 20s 1 leg sprints with 60s rests @150% of 1 leg VO_{2max}, 4x/wk for 7 wks	β_m ▲ (16%)	Peak, mean power output (18%) & VO_{2peak} ▲
Nevill et al. 1989	(8)	8 wks training: 2x 30 s sprints with 10min rest - 2x/wk, 6 – 10x 6s sprints with 54s rest – 1x/wk, and 2 – 5x 2min runs @ 110% VO_{2max} with 5min rest – 1x/wk	β_m ▬	Peak & mean output (12% & 6%) ▲ ; 50- and 200-m time ▼
Linossier et al 1993	(10)	2x series of 8x 5 s sprints @ 80% F_{max} with 55s recovery with 15min rec. between series, 4/wk for 7 wks	CrP ▬	W_{peak} & Wingate performance ▲
Mannion et al. 1994	(13)	6x 25RM knee extensor @ 4.19 rad.s^{-1} with 30s rest, 3x/wk for 16 wk	(n = 23) β_m & [carnosine] ▬	(n = 23) Mean power output and work done ▲ ; 60% MVC
	(10)	5x 15 RM knee extensor @ 1.05 rad.s^{-1} with 40s rest, 3x/wk for 16 wk		endurance time ▬
Dawson et al. 1998	(9)	20- 40x (30-80m) sprints @ ~95% max. speed with 2-4 min rec., 2-3x/wk for 6 wks	CrP ▬	$VO_{2\,peak}$ & sprint performance ▲
Pilegaard et al. 1999	(7)	2 – 5x 1min 1 leg knee extensor exercise with 2min rest repeated 3x, 3 - 5x/wk for 8 wks	CrP ▬	Peak, mean output (16% & 15%) & endurance (132%) ▲

MVC's are maximum voluntary (isokinetic) contractions of the knee extensors and HPR is highest pedalling resistance. β_m is the physico-chemical H^+ buffering capacity of the muscle. The effects of training on muscle creatine phosphate (PCr) stores are included in this Table because net CrP hydrolysis buffers intracellular H^+ ions, as described in the text. Carnosine is a β-alanylhistidine dipeptide that also buffers H^+ ions. ▲ and ▬ indicate increased and not significantly changed, respectively.

Table 2.6. Effects of training on muscle fibre Na^+/K^+ATPase pump activity, K^+ re-uptake capacity and exercise performance changes in previously untrained or moderately-trained individuals.

References	(n)	Training regimes	Physiological changes
Green et al. 1993	(9)	2hr rides on 6 consecutive days @ 65% VO_{2max}	Na^+/K^+ATPase (14%) & K^+ homeostasis ▲
McKenna et al. 1993	(9)	4 – 10x 30s sprints with 4min rec., 3x/wk for 7 wks	Na^+/K^+ATPase & K^+ homeostasis 19% ▲
Madsen et al. 1994	(39)	25min training 3x/wk for 6 wks @ 93% of HR_{peak}	Na^+/K^+ATPase ▲ (15%)
McKenna et al. 1997	(6)	8x 30s cycling sprints @ HPR with 4min rec., 3x/wk for 7 wks	K^+ homeostasis ▲ ; plasma SID at exhaustion ▼
Evertsen et al. 1997	(10)	16 hr/wk for 20 wks @ 60 - 70% VO_2	Na^+/K^+ATPase ▲ (16%) both
	(10)	16hr/wk for 20 wks @ 80 – 90% VO_2	
Green et al. 1999	(9)	3 sets of 6-8 RM's, 3x/wk for 12 wk	Na^+/K^+ATPase (16%) by 7wk ▲
	(7)	1x 2 h ride/day @68% VO_{2peak}, 5-6x/wk for 11 wks	Na^+/K^+ATPase (22%) by 3wk ▲

HR_{peak} is peak heart rate. RM's are repeated maximum contractions. SID is the strong ion difference between $[Na^+] + [K^+]$ and $[Cl^-] + [lactate^-]$. Low SID values indicate acidosis. ▲ and ▬ indicate increased and not significantly changed, respectively.

The same group also found that training increased the number of sarcolemmal Na^+/K^+ ATPase pumps by about 13% (Pilegaard et al. 1999). Most studies have shown that endurance or interval training improves muscle Na^+/K^+ ATPase activity and K^+ re-uptake capacity by 15-20% in previously untrained individuals (Table 2.6).

CHAPTER 2.3

EFFECTS OF SUSTAINED HIGH INTENSITY INTERVAL TRAINING IN WELL-TRAINED ATHLETES

While the physiological adaptations to training have been studied extensively in previously untrained subjects, far less is known about the responses to 4-6 weeks of sustained high intensity interval training (HIT) in already well-trained athletes. Current ideas on the benefits of HIT prior to competition are derived mainly from the subjective observations and experiences of athletes in the field. There have been few objective studies on the effects of HIT on endurance performances of competitive athletes (Wells and Pate 1988).

Perhaps the first study of physiological adaptations to increases in training intensity was by Acevedo and Goldfarb in 1989 (Table 2.7). Their subjects were seven competitive long-distance runners who underwent 8-weeks of HIT at 90-95% of peak heart rate. HIT significantly improved their 10 km running performances by ~ 3.0% from 35:27 to 34:24 min and increased their "supra-maximal" running endurance by ~ 20% from 19:25 to 23:18 min, independently of any change in VO_{2peak}. Others have also shown that increases in training volume improve the performances of trained athletes without altering VO_{2peak} (Costill 1976; Daniels et al. 1978; Martin et al. 1986; Costill et al. 1988).

Table 2.7. Effects of sustained high-intensity interval training (HIT) on the athletic and maximum performances of already well-trained individuals.

References	(n)	HIT regimes	Athletic performances	Maximum performances
Acevedo & Goldfarb 1989	(7)	Interval workouts @ 90-95% of HR_{peak} with minimal rests, 3x/wk for 8 wks	TT_{10} (3.0%) ▲	T_{SMF} (20%) ▲ ; VO_{2peak} ═
Lindsay et al. 1996	(12)	6 – 8x 5min @ 80% of W_{peak} with 1min rest, 3x in 2 wks,	TT_{40} (0.5%) ═	W_{peak} (1.6%) ═; T_{F150} ▲ (12%)
		then 6x in 4wks	TT_{40} (3.5%) ▲	W_{peak} (4.3%) ▲ ; T_{F150} ▲ (22%)
Westgarth-Taylor et al. 1997	(8)	6-8x 5 min @ 80% of W_{peak} with 1min rests, 4 in ~2 wks,	Not determined	W_{peak} (3.7%) ▲
		then 8x in ~4 wks@,	TT_{40} (3.0%) ▲	W_{peak} (4.2%) ▲
		then 12 in ~6 wks	TT_{40} (3.5%) ▲	W_{peak} (4.9%) ▲
Stepto et al. 1999	(4)	12x 0.5min @ 175% of W_{peak} with 4.5min rest, 6x in 3 wks	TT_{40} (1.9%) ▲	W_{peak} (0.5%) ═
	(3)	12x 1min @ 100% of W_{peak} with 4min rest, 6x in 3 wks	TT_{40} (0.4%) ═	W_{peak} (1.6%) ═
	(4)	12x 2min @ 90% of Wpeak with 3min rest, 6x in 3 wks	TT_{40} (1.6%) ═	W_{peak} (1.8%) ▲
	(4)	8x 4min @ 85% of Wpeak with 1.5min rest, 6x in 3 wks	TT_{40} (3.3%) ▲	W_{peak} (3.8%) ▲
	(4)	4x 8min @ 80% of Wpeak with 1min rest, 6x in 3 wks	TT_{40} (-0.5%) ═	W_{peak} (1.1%) ═

All the subjects in these studies were highly trained individuals with VO_{2peak} values of ≥ 65 mL.min^{-1}kg^{-1} body mass and/or peak sustained power outputs (W_{peak}) of about 400W or around 5 W.kg-1 body mass. HR_{peak} was peak heart rate. TT_{10} was a 34-35 min, 10-km time-trial average running speed, TT_{40} was a 50-60 min, simulated 40-km time-trial average cycling speed. T_{SMF} was a 19-23 min "supra-maximal" run to fatigue, @ Unpublished observations of Westgarth-Taylor et al. reported by Hawley et al. (1997). ▲ indicates significant improvement and ═ indicates insignificant improvement, respectively.

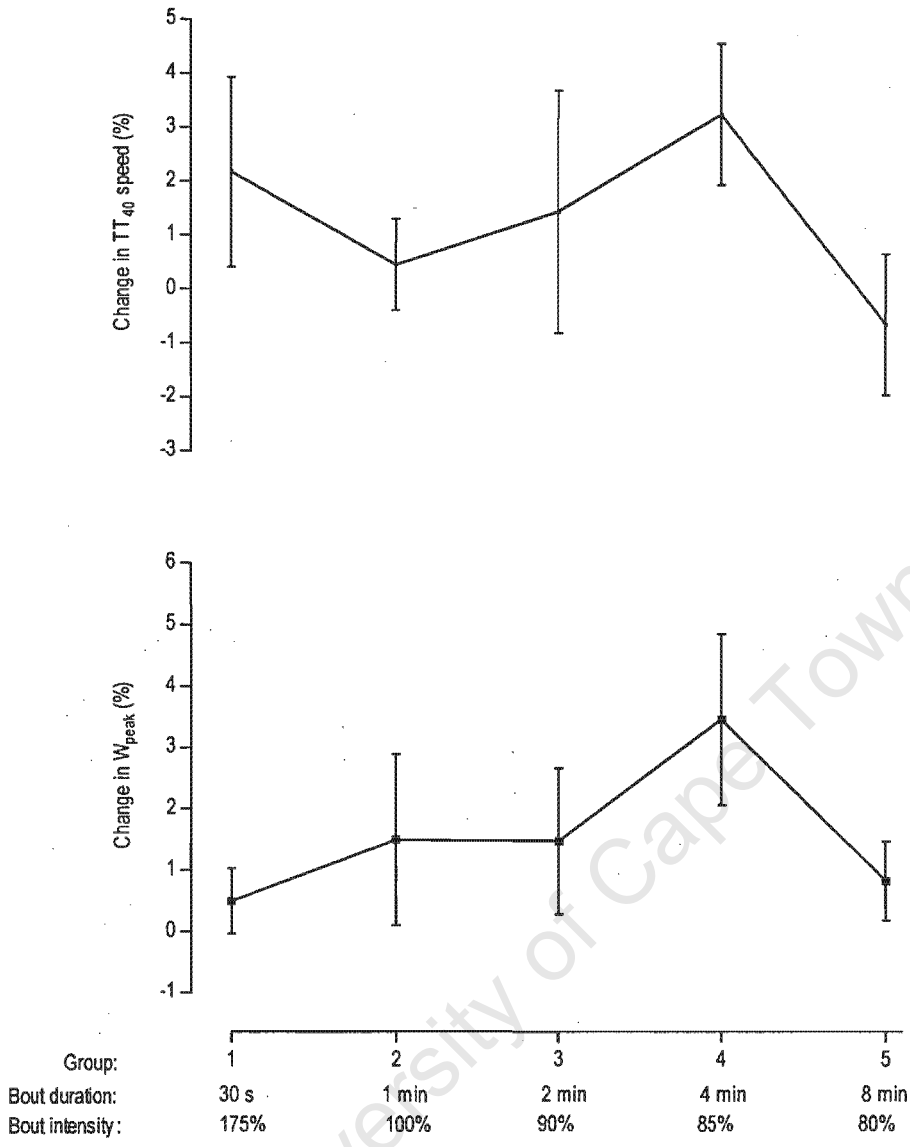


Figure 2.1. Percentage change in TT_{40} speed (*upper panel)* and W_{peak} (*lower panel)*after the five interval-training programs. Adapted from and courtesy of Stepto et al. 1999.

Subsequently, Lindsay et al. (1996), Weston et al. (1997) and Westgarth-Taylor et al. (1997) studied the effects of HIT in twenty male competitive cyclists (Table 2.7). The cyclists were all riding an average of 300 km/wk and had VO_{2peak} values of $\geq$ 65 $mL.min^{-1}.kg^{-1}$ and peak sustained power outputs (W_{peak}) of ~ 400 W, or ~ 5 $W.kg^{-1}$. Before any intervention, each cyclist performed several laboratory tests on separate occasions to ensure that his athletic performances were stable. One test was an ~ 1 min ride to fatigue at 150% of W_{peak} (T_{F150}). Another test was an ~ 10 min incremental ride to exhaustion for determinations of VO_{2peak} and W_{peak}, as described by Hawley and Noakes (1992). A third test was a 50-60 min simulated 40-km time-trial (TT_{40}) on the subject's own bicycle, as described by Palmer et al. (1996). The cyclists' mean baseline performances and their individual coefficients of variation (in parenthesis) in the T_{F150}, W_{peak} and TT_{40} tests were ~ 60.5 sec (2.1%), 411 W (1.3%) and 56.7 min (0.9%), respectively.

After baseline testing, the cyclists replaced 15 $\pm$ 2% (mean $\pm$ SD) of their ~ 300 km/wk endurance training with 6-12 sessions of HIT. HIT sessions took place once or twice a week for up to 6 weeks and consisted of six to nine 5-min cycle rides at 80% of W_{peak} (~ 86% VO_{2peak}) separated by 1 min rests. After every three or four HIT sessions over 2 weeks, the cyclists performed a further W_{peak} test and the exercise intensity of any subsequent HIT sessions was increased to 80% of the new (higher) W_{peak} value. T_{F150} and TT_{40} tests were also repeated at regular intervals to monitor the time-course of the effects of HIT on cycling performance.

In the study of Lindsay et al. (1996), 3 and 6 HIT sessions over 2 and 4 weeks increased the cyclists' times to fatigue at 150% of W_{peak} by ~ 12% and 22%. The same HIT programme also improved the cyclists' W_{peak} values by ~ 1.6% (not significant) and 4.3%. Similar rises in cyclists' W_{peak} values after HIT were also found in a follow-up study, from the same laboratory,

by Westgarth-Taylor et al. (1997). They showed that 4, 8 and 12 HIT sessions over 2, 4 and 6 weeks increased W_{peak} values by ~ 3.7%, 4.2% and 4.9%, respectively. In comparing these two studies, Hawley et al. (1997) concluded that the 4-5% increases in W_{peak} values after 4-6 HIT sessions were not further improved by > 6 HIT sessions.

Improvements in TT_{40} cycling performances with HIT also reached an asymptote after ~ 6 HIT sessions. Data from the studies of Lindsay et al. (1996) and Westgarth-Taylor et al. (reported by Hawley et al. 1997) showed that 6, 8 and 12 HIT sessions all decreased TT_{40} cycling times by 3.0- 3.5% (Table 2.7). Typical, 90-120 sec improvements in TT_{40} cycling times after HIT were due to the subjects being able to sustain significantly higher absolute and relative work rates during the time-trials. HIT increased the absolute work rates during the time-trials from ~ 300 to 330 W and the relative work rates from ~ 72% of pre-HIT W_{peak} to ~ 76% of post HIT W_{peak}. Although W_{peak} values were closely related to TT_{40} cycling speeds (r = 0.87), both before and after HIT, there was no significant correlation between the cyclists' 15-20 W increase in W_{peak} and their ~ 1.5 km/h faster TT_{40} performances after HIT.

Coyle et al. (1991) also noted that TT_{40} performances are determined by a combination of the cyclists' W_{peak} values and their ability to sustain a high percentage of that W_{peak} during exercise. They showed that US 'national class' cyclists could be distinguished from 'good state riders' with similar (~ 70 mL/min/kg) VO_{2peak} values by their ability to work at a higher (~ 90 vs. 86%) fraction of VO_{2peak}. The superior endurance performances of distance runners compared with equally fast runners up to 5 km was also found to be due to their ability to sustain higher percentages of VO_{2peak} with increasing race distance (Coetzer et al. 1993).

Although the greater 'fatigue resistance' of better distance athletes is not well-understood (Peronnet and Thibault 1989; Coetzer et al. 1993), superior endurance performances may be related to lower rates of lactate accumulation in working muscles (Costill et al. 1973, 1976; Farrell et al. 1979; LaFontaine et al. 1981; Sjodin and Jacobs 1981; Sjodin et al. 1982; Coetzer et al. 1993; Fukuba et al. 1999). In highly trained endurance athletes, there is little increase in plasma lactate concentration with increasing work rates until exercise intensity reaches 80-85% of VO_{2peak} (Costill et al. 1976; Farrell et al. 1979). Part of the increase in work rates in the TT_{40} rides after HIT may have resulted from a reduction in the rates of carbohydrate oxidation and lactate accumulation at the same absolute work rates after HIT. Westgarth-Taylor et al. (1997) showed that 12 HIT sessions decreased curvilinear rises in rates of carbohydrate oxidation and plasma lactate accumulation in successive 10 min rides at 50%, 60%, 70% and 80% of the cyclists' pre-HIT W_{peak}. However, rates of carbohydrate oxidation and plasma lactate accumulation were similar to the pre-HIT values when the cyclists repeated the rides at 50%, 60%, 70% and 80% of their new (higher) post-HIT values. Thus, the decreases in carbohydrate oxidation and lactate accumulation at the same absolute sub-maximal work rates after HIT were probably due to the cyclists riding at lower relative exercise intensities (Brooks and Mercier 1994).

Reductions in carbohydrate oxidation and lactate accumulation did not explain why the cyclists were able to sustain higher (76% vs. 72% of W_{peak}) relative exercise intensities during the TT_{40} performance rides after HIT. Improved TT_{40} performances after HIT accelerated estimated rates of carbohydrate oxidation from ~ 4.3 to 5.1 g/min and increased predicted plasma lactate concentrations from ~ 5.1 to 7.1 mmol.L-1 (Westgarth-Taylor et al. 1997).

The reduced reliance on carbohydrate metabolism at the same sub-maximal work rates after HIT was unlikely to have been a result of an increased muscle mitochondrial density, as occurs with endurance training in previously sedentary subjects (Henriksson 1977; Benzi 1981; Saltin and Gollnick 1983; Holloszy and Coyle 1984; Gollnick 1986; Hurley et al. 1986; MacRae et al. 1992, 1995; Green et al. 1992). Biopsies from the vastus lateralis of six of the eight subjects in the study of Lindsay et al. (1996) showed that HIT had no influence on muscle glycolytic or mitochondrial enzyme activities in trained cyclists (Weston et al. 1997). During HIT, muscle hexokinase, phosphofructokinase, citrate synthase and 3-hydroxyacyl CoA dehydrogenase activities all remained constant at 15-17, 300-350, 160-170 and 85-90 μmol/min/g protein, respectively. Others have also found little effect of increased training on muscle enzyme levels in well-trained athletes. Houston and Thomson (1977) showed that 6 weeks of intermittent hill sprints did not alter the muscle lactate dehydrogenase isoenzyme composition of endurance runners. In contrast, Sjodin et al. (1982) reported that 14 weeks of additional, intense training at 'the onset of blood lactate accumulation' increased the proportion of the heart forms of lactate dehydrogenase in the leg muscles of distance runners. However, the additional training did not increase muscle phosphofructokinase or citrate synthase activities. Costill et al. (1988) also found that a doubling of swim training from ~4 to 9 km/day for 10 days had no effect on muscle citrate synthase activities in college swimmers.

While HIT had no influence on certain muscle enzyme activities, it significantly improved muscle physiochemical H^+-buffering (β_m) capacity in trained athletes (Weston et al. 1997). After HIT, β_m was increased from ~ 200 to 240 μatom H^+/g dry wt/pH unit. Furthermore, β_m correlated with TT_{40} cycling speeds before HIT ($r = 0.82$, $P \leq 0.05$), but the relationship between increase in β_m and improvements in TT_{40} performances was not significant ($r = 0.74$). Increases in β_m found by Weston et al. (1997) probably did not measurably decrease

intracellular H^+ accumulation. Rises in venous plasma lactate concentrations with increases in carbohydrate oxidation were similar at the same relative exercise intensities before and after HIT (Westgarth-Taylor et al. 1997).

Following the demonstration that ≥ 6 sustained (5 min) high-intensity (80% of W_{peak}) interval training sessions maximally improved TT_{40} cycling speeds, the same group examined the effects of varying the intensity of 6 HIT sessions on endurance exercise performances. In an attempt to identify the best training stimulus, Stepto et al. (1999) randomly assigned 19 provincial-level, male endurance cyclists (VO_{2peak} ≥ 65 $mL.min^{-1}.kg^{-1}$) to one of five types of supervised HIT sessions in the laboratory. Details of the five HIT regimes are given in Table 2.7. Each HIT session lasted ~60 min and was designed to represent a training program that endurance cyclists would be prepared to undertake in preparation for a competition. Since the cyclists may have varied in their ability to tolerate the different degrees of 'effort' in each type of HIT, no attempt was made to control their endurance training volumes. The cyclists were only requested not to perform any HIT outside the laboratory.

Percent improvements in simulated TT_{40} performances after the different types of HIT were best fitted by a cubic polynomial function of the rank-ordered duration of the work bouts in each HIT protocol ($r = 0.70$, $P \leq 0.01$). A cubic polynomial function predicted the greatest improvements in TT_{40} performances after 6 HIT sessions consisting of either 8 x 4 min rides at 85% of W_{peak} or 12 x 30 s rides at 175% of W_{peak} (Table 2.7). Maximum ~3% improvements in TT_{40} performances after 4-min rides at 85% of W_{peak} were similar to those observed by Lindsay et al. (1996) and Westgarth-Taylor et al. (1997) after 5-min rides at 80% of W_{peak} (Table 2.7). These 3.0 - 3.5% faster TT_{40} cycle rides at 75 - 80% of W_{peak} after HIT at 80 - 85% of W_{peak} supported the idea that athletes should train for competition at exercise intensities specific to

their event (Hawley et al. 1997). In contrast, the ~2% improvements in TT_{40} performances after 30-s sprints at 175% of W_{peak} (Table 2.7) did not conform to the concept of training specificity. Sprint training was not expected to improve TT_{40} endurance performances in well-trained cyclists. An apparent nadir in the improvements in TT_{40} performances between the 4-min rides at 85% of W_{peak} and the 30-s sprints at 175% of W_{peak} suggested that the two HIT programs may have produced different adaptations. Whereas faster TT_{40} performances after HIT at 85% of W_{peak} were associated with ~4% increases in W_{peak}, faster TT_{40} performances after HIT at 175% of W_{peak} were independent any significant change in W_{peak} (Table 2.7). Again, improvements in TT_{40} performances after HIT were more closely related to the cyclists' ability to sustain higher percentages of W_{peak} during prolonged exercise, than to improvements in their W_{peak} values in progressive exercise ($r = 0.92$ vs. $r = 0.09$).

Abilities to sustain higher percentages of W_{peak} during exercise after HIT may have resulted from a greater motivation of the athletes to perform. Although Lindsay et al. (1996) found no effect of HIT on the mood states of their subjects, any alteration in training may influence human exercise performance. Athletes are very suggestive to new training regimes, especially if they believe they are supposed to improve performance. However, psychological factors could not explain the findings of Stepto et al. (1999). Their subjects would not have been able to predict which type of HIT should, or should not, improve TT_{40} cycling performances (Table 2.7).

Improved performances after HIT could also have been due to a strengthening of the working muscles. Tabata et al. (1990) and Martin et al. (1994) showed that cycling training at 70-90% of VO_{2peak} for 6 – 7 weeks increased quadriceps peak isokinetic torque in moderately trained male college students. Pedalling at knee extension velocities of ~210 °/sec predominantly increased

quadriceps peak torque at slow knee extension velocities of 30 - 120 °/sec. Cycling training had no effect on quadriceps peak torque at knee extension velocities of 180 - 300 °/sec.

CHAPTER 2.4

EFFECTS OF RESISTANCE (STRENGTH) TRAINING IN ENDURANCE TRAINED INDIVIDUALS

Increases in muscle strength are probably best achieved by resistance training (for review, see Kraemer et al. 1996). However, few studies have examined whether improvements in muscle strength gained from resistance training might enhance endurance performance in well-trained athletes. One of the first studies of the effects of resistance training on endurance exercise capacity was performed by Hickson et al. (1988). They supplemented the endurance training of two groups of eight moderately trained runners or cyclists with heavy resistance training on 3-days/wk for 10 weeks. Resistance training increased the subjects' leg muscle strength by 30% but gains in strength had no effect on VO_{2peak} values or on the runners' 10 km time-trial performances. In contrast, resistance training improved the long-term (70 - 85 min) endurance of the cyclists by ~20% and the short-term (4 – 8 min) endurance of both the runners and the cyclists by ~12%.

Marcinik et al. (1991) also found that resistance training on 3-days/wk for 12 weeks increased the leg strengths of 10 previously untrained males by 30 - 52%. Despite no change in the subjects' VO_{2peak} values after resistance training, their rates of plasma lactate accumulation during progressive exercise were decreased by ~12% and their cycling times to exhaustion at 75% of VO_{2peak} were increased by 35%. Large increases in the subjects' cycling endurance from ~26 to 35 min after resistance training may have resulted from a general improvement in

their fitness. Previously untrained individuals probably benefit from any improvement in either strength or endurance.

In contrast, further improvements in strength may not enhance the endurance performances of highly trained athletes who are already capable of sustaining high power outputs in their chosen sport. Rowing is a sport where competitors often perform some form of supplementary resistance training during their winter season. Bell et al. (1989) examined whether adding resistance training to rowing training improved performances in three groups of six varsity oarsmen. One group continued their normal rowing training. The other two groups supplemented their rowing with either 18 - 22 high-velocity, low-resistance repetitions or 6 - 8 low-velocity, high-resistance repetitions, on 4 days/wk for 5 weeks. Improvements in high and low velocity performances were specific to the resistance-training programs. Peak power outputs and peak plasma lactate concentrations in progressive rowing exercise tests to exhaustion were similar in the three groups.

Swimmers are another group of athletes who often practice some form of resistance training. Tanaka et al. (1993) investigated whether adding resistance training to pool training might improve swimming sprint performance in two groups of 12 experienced swimmers during their competitive season. One group continued their normal swimming training and the other group supplemented the same pool training sessions with resistance training on 3 days/wk for 8 weeks. The progressive resistance-training program increased the strength of the muscles employed in front crawl swimming by 25 - 35% but gains in strength did not improve stroke mechanics or swimming sprint performance.

The effects of progressive resistance training on 3-days/wk for 6 weeks on TT_{40} cycling performances were recently examined (James Home et al. unpublished observations). Resistance training was added to the 'normal' training of seven endurance cyclists who were riding ~200 km/wk. Each resistance training session consisted of three sets of 6-8 maximal, leg presses, quadriceps extensions and hamstring curls. Resistance training increased the strength of the muscles involved in cycling by ~25% but gains in strength did not improve TT_{40} cycling performances. On the contrary, resistance training slowed TT_{40} cycling times from ~59 to 62 min. Most cyclists complained of feeling 'tired and heavy' while riding and most were forced to reduce their weekly training distances by ~20%.

CHAPTER 2.5

EFFECT OF REDUCED TRAINING (TAPER) IN ENDURANCE TRAINED INDIVIDUALS

Before a competition, many endurance athletes reduce, or "taper", their training (for reviews, see Neufer 1989; Houmard 1991; Houmard and Johns 1994; Mujika 1998). Those athletes often face a taper with trepidation as they try to balance the recovery from the fatigue of intense training against a fear that reduced training will decrease their fitness. Although mathematical models predict that training should be drastically reduced in the last 12-14 days before a competition (Morton 1977; Banister et al. 1999), few athletes are brave enough to implement such a strategy. So far, there has been no systematic study of the optimum reductions in the frequency, duration and intensity of exercise to maximise performance.

Perhaps the first study of a reduced exercise frequency program on performance was conducted by Brynteson and Sinning in 1973. Their subjects cycled for 30 min/day at 80% of peak heart rate on 5 days/wk for 5 wk and then progressively decreased the frequency of their

training from 5 to 4, 3, 2 or 1 day(s)/wk over a further 5 weeks. Even the final 80% reduction in training frequency maintained previous ~10% improvements in cycling VO_{2peak} values.

Later, Hickson and colleagues [i.e., Hickson and Rosenkoetter (1981), Hickson et al. (1982) and Hickson et al. (1985)] sequentially studied the effects of detraining by decreasing either the frequency, duration or intensity of training in healthy subjects. First, the subjects ($n \geq 12$/group) alternately cycled or ran for 40 min/day on 6-days/wk for 10 weeks to improve their cycling VO_{2peak} values by ~10%. Then, they reduced their training frequency, duration or intensity by 33% or 66% over a subsequent 15 weeks. A decrease in training frequency from 6 to 4 or 2 days/wk or a reduction in training duration from 40 to 26 or 13 min/day maintained previous improvements in VO_{2peak} values. However, 33% and 66% decreases in training intensity reduced VO_{2peak} values by ~7.5% and 10%, respectively, within 10 weeks. A 66% reduction in training duration and 33% and 66% decreases in training intensity also shortened (~2 h) cycling endurance at 80% of pre-training VO_{2peak} by ~10%, 21% and 30%, respectively.

A comparison of two studies by Wittig et al. (1989, 1992) also suggests that exercise intensity must be maintained to prevent de-training during a taper (Costill et al. 1985; Coyle et al. 1984). They examined two groups of male distance runners (n = 10/study) who reduced their training volume by ~70% for 3 - 4 weeks, while either maintaining their training intensity or decreasing their running distances at > 70% of VO_{2peak} from 76% to 0%. While the reduced training volume had no effect on 5-km running performances, the decreases in training volume and intensity slowed 5 km race times by ~1% from ~16.6 to 16.8 min. Slower 5-km race times after the decrease in training intensity were associated with more negative mood states (Wittig et al. 1992) and increased carbohydrate metabolism during exercise, despite no changes in plasma volume or VO_{2peak} (McConell et al. 1993).

Conversely, Shepley et al. (1992) found that an increase in training intensity during a taper improved subsequent exercise performance. They assigned nine male competitive cross-country runners, to a random order of three different 1-week tapers separated by 4-week periods of continued training. During the 4-week periods, the subjects ran ~80 km/wk at 70-80% of VO_{2peak} and then either rested completely or performed low-intensity ($\leq$ 60% of VO_{2peak}), moderate-volume (30 km/wk) or high-intensity (115-120% of VO_{2peak}), low-volume (10 km/wk) tapers. In the first taper, the runners reduced their training intensity and volume by ~15 and 60% and, in the second taper, they replaced their habitual training with 3 - 5 x 500 m (~80 s) runs/day separated by 6-7 min recoveries. While ~60%, 90% and 100% reductions in training volume all increased quadriceps isometric contraction strength without changing VO_{2peak} values, only interval training at 50-60% higher exercise intensities increased blood volume, muscle citrate synthase activity and exercise performance. After high-intensity interval training (HIT), the subjects' treadmill running times to fatigue at their best 1500-m times were improved by ~20% from ~250 to ~320 sec.

Houmard et al. (1994) also examined the effects of 1 week of HIT and an ~85% reduction in training volume on running performance. Their runners (n = 8/group) replaced their habitual training with comparable intervals of high-intensity running or cycling exercise at ~90% of VO_{2peak}. The results showed that the benefits of HIT in a taper are (a) unrelated to the large reductions in training volume and (b) specific to the type of exercise. While cycling HIT had no effect on 5-km running performances, running HIT increased sub-maximal running economy by ~6% and improved 5-km race times by ~3% from ~17.3 to 16.8 min.

Effects of changes in training intensity during a taper on subsequent exercise performance are summarised in Table 2.8. Those studies suggest that training intensity should be maintained or

even slightly increased to preserve the fitness that might otherwise be lost with a marked reduction in training volume.

Certain sports may also require a minimum frequency of training. Neufer et al. (1987) studied male competitive swimmers (n=8/group) who reduced their pool training from 8.2 km/day on 6 days/wk to 2.7 km/day on 3 or 1 day(s)/wk for 4 weeks. Whereas the 50% reduction in training frequency maintained VO_{2peak} values and swimming stroke distances, the ~83% decrease in training frequency reduced VO_{2peak} values by ~3% and shortened stroke distances by ~8%. This finding may explain why competitive swimmers commonly decrease their training volume by 60 - 90% for 1 - 3 weeks before a competition but rarely reduce their training frequency by more than 20 - 30% (Houmard and Johns 1994). Swimmers in particular often complain that they lose the "feel" for the activity if they miss pool sessions for several days.

Alternatively, the decreased VO_{2peak} values and shortened stroke distances in the study by Neufer et al. (1987) could have resulted from the ~95% reduction in training volume from 49 to ≤ 3 km/wk over 4 weeks. As mentioned previously, Hickson et al. (1982) found that a 66% reduction in training volume over 15 weeks decreased ~2 h cycling endurance by ~10%.

In contrast, other studies have shown that more modest 20 - 80% single-step reductions in training volume over 1- 4 weeks had little effect on exercise performance (Table 2.9). Houmard et al. (1989, 1990) studied distance runners who decreased their 80-110 km/wk training volume by either ~30% for 10 days (n = 5) or ~75% for 3 weeks (n = 10). They found that neither taper had any effect on ratings of perceived exertion, heart rates, venous lactate concentrations, respiratory exchange ratios, VO_{2peak} values or indoor 5-km race times. Johns et al. (1992) studied 12 male intercollegiate swimmers who tapered for 10 and 14 days at the end of the

season. The taper increased the swimmers power in a tethered swim by 5% but had no effect on their stroke distance, VO_2 or venous lactate concentrations during a 183-m (200 yard) sub-maximal swim. Neary et al. (1992) studied subjects (6-8/group) who cycled for 1h/day at 75-85% VO_{2peak} on 5 days/wk for 8 weeks and then reduced their training volume by 50% for 4 or 8 days. Both tapers increased power output at the ventilation threshold by ~ 27 W (11%) but how that adaptation influenced cycling performance was not determined.

While single step reductions in training volume only maintain exercise performance, some progressive reductions in training volume may improve exercise performance. Costill et al. (1985) found that a progressive 65% reduction in the pool training of a group of 17 collegiate swimmers from ~ 8.8 to 3.1 km/day over 2 weeks increased swim bench strength by ~ 18%, tethered swim power by ~ 25% and 183-m (200 yard) swimming performance times by ~ 3%. .

Mujika et al. (1996b) found similar improvements in the performances of elite swimmers (n = 6/group) who progressively reduced their training by 30% over 3 weeks, 40% over 4 weeks and 43% over 6 weeks. After the 3-, 4- and 6-week tapers, individual swimming performances over a range of distances was improved by ~ 2.9%, 3.2% and 1.8%, respectively. In another study, Mujika et al. (1996a) also showed that a progressive 75% decrease in 8 swimmers' training volume over 4 weeks improved individual swim performance by 2.3%. They reported that changes in performance after the taper correlated with changes in testosterone/cortisol ratios ($r = 0.81$, $P \leq 0.05$).

Table 2.8. Effects of changes in training intensity during a taper or reduced training protocol (*) on subsequent athletic and maximum exercise performance in already trained individuals.

References	(n)	Changes in training intensity (%) during taper	Athletic performance changes	Maximum Performances
Hickson et al. 1985*	(6)	66% ▼ over 15 weeks	~2h endurance cycle (30%) ▼	VO_{2peak} (11.5%) ▼
	(7)	33% ▼ over 15 weeks	~2h endurance cycle (21%) ▼	VO_{2peak} (5.8%) ▼
Wittig et al. 1992	10)	▼ to > 70% $V0_{2max}$ over 4 weeks	TT_5 performance (1%) ▼	VO_{2peak} —
Shepley et al. 1992	(9)	20% ▲ in HIT over 5 days	TTF_{1500} (22%) ▲	VO_{2peak}—
	(9)	100% ▼ in HIT over 5 days	TTF_{1500} (6%) ▲	VO_{2peak}—
Houmard et al. 1994	(8)	85% ▼ in HIT over 7 days	TT_5 performance (3%) ▲	VO_{2peak}—

Subjects in these studies were already trained individuals with VO_{2peak} values of ± 55 $mL.min^{-1}.kg^{-1}$ body mass and/or peak sustained power outputs (W_{peak}) of about 350 W. TT_5, 5-km time trial run; TTF_{1500}, time to fatigue in a treadmill run at 1500-m best time. ▲, ▼, and — indicate increased or improved, decreased and not significantly changed, respectively.

Table 2.9. Effects of reduction in training volume and/or frequency during a taper or reduced training protocol (*) on subsequent athletic and maximum exercise performance in already trained individuals.

References	(n)	Reduction in training volume (TV) and/or frequency (TF) during taper	Athletic performances changes	Maximum Performances
		SINGLE STEP TAPERS		
Hickson et al. 1982*	(8)	33% ▼ in TV over 15 weeks	~2h endurance cycle ▬	VO_{2peak} (20%) ▲
	(7)	66% ▼ in TV over 15 weeks	~2h endurance cycle (10%) ▼	VO_{2peak} (10%) ▲
Neufer et al. 1987	(8)	66% ▼ in TV, 50% ▼ in TF over 4 weeks	Tethered swim (13.6%) ▼	VO_{2peak} ▬
	(8)	66% ▼ in TV, 83% ▼ in TF over 4 weeks	Tethered swim (13.6%) ▼	VO_{2peak} ▼
Wittig et al. 1989	(10)	70% ▼ in TV over 3 weeks	TT_5 performance ▬	Not determined
Houmard et al. 1989	(5)	27% ▼ in TV over 10 days	TT_5 performance ▬	VO_{2peak} ▬, TTE ▬
Houmard et al. 1990	(10)	70% ▼ in TV, 17% ▼ in TF over 3 weeks	TT_5 performance ▬	VO_{2peak} ▬, TTE (9.5%) ▲
Johns et al. 1992	(5)	76% ▼ in TV, 50% ▼ in TF over 10 days	Tethered swim (5%) ▲ *	VO_{2peak} ▬
	(7)	60% ▼ in TV, 50% ▼ in TF over 14 days	Tethered swim (5%) ▲	VO_{2peak} ▬
Zarkadas et al. 1995	(3)	30% ▼ in TV over 10 days	TT_5 performance ▬	W_{peak} ▬
Banister et al. 1999	(5)	22% ▼ in TV over 14 days	TT_5 performance ▬	W_{peak} (1.4%) ▲
		PROGRESSIVE TAPERS		
Costill et al. 1985	(17)	65% ▼ over 2 weeks	Individual swim performance (~3%) ▲	Swim power (24.6%) ▲
Mujika et al. 1996	(18)	30% ▼ in TV over 3 weeks	Individual swim performance (2.9%) ▲	Not determined
	(18)	40% ▼ in TV over 4 weeks	Individual swim performance (3.2%) ▲	
	(18)	43% ▼ in TV over 6 weeks	Individual swim performance (1.8%) ▬	
Mujika et al. 1996a	(8)	75% ▼ in TV over 4 weeks	Individual swim performance (2.3%) ▲	Not determined
Hooper et al. 1998	(9)	10% ▼ per day in TV for 10 days	100m and 400m swim performance ▬	Not determined
Hooper et al. 1999	(10)	35% ▼ in TV over 2 weeks	100m swim performance ▬	Not determined
Mujika et al. 2000	(4)	50% ▼ in TV over 6 days	800m running performance ▬	Not determined
	(4)	75% ▼ in TV over 6 days	800m running performance ▬	
		EXPONENTIAL TAPERS		
Zarkadas et al. 1995	(6)	50% ▼ in TV over 10 days ($\tau \leq 5$ days)	TT_5 performance (4%) ▲	W_{peak} (5%) ▲
Zarkadas et al. 1995	(6)	50% ▼ in TV over 13 days ($\tau \leq 4$ days)	TT_5 performance (2%) ▲	VO_{2peak} (8%), W_{peak} (8%) ▲
	(5)	50% ▼ in TV over 13 days ($\tau \leq 8$ days)	TT_5 performance (6%) ▲	W_{peak} ▬
Banister et al. 1999	(6)	31% ▼ in TV over 14 days ($\tau \leq 5$ days)	TT_5 performance (4%) ▲	W_{peak} (5%) ▲
	(5)	50% ▼ in TV over 14 days ($\tau \leq 8$ days)	TT_5 performance (3%) ▲	W_{peak} (4%) ▲
	(6)	65% ▼ in TV over 14 days ($\tau \leq 4$ days)	TT_5 performance (6%) ▲	W_{peak} (7%) ▲

All the subjects in these studies were highly trained individuals with VO_{2peak} values of ≥ 65 mL.min^{-1}kg^{-1} body mass and/or peak sustained power outputs (W_{peak}) of about 400W or around 5 W.kg^{-1} body mass. TT_5, 5-km time trial; TTE, time to exhaustion during maximal treadmill test; Tethered swim, swim power test. ▲, ▼, and ▬ indicate increased, decreased and not significantly changed, respectively. A time constant (τ) is the time taken for an exponential decay to decline to 37% of its starting value. * significant (5%) increase in mean distance per stroke during submaximal swim during taper after removal of exposed body hair (shaving). Not ruling out that intensity during these tapers could have taken place merely categorised according to the significant reductions in training volume or frequency. Some papers were not included in the table because too many variables were altered during taper. Banister et al. (1999) had the same subjects as Zarkadas et al. (1995).

In contrast, Hooper et al. (1998) found no effect of a 2-week taper on the performances of three groups of 9 swimmers. One group reduced their training frequency according to each athlete's daily ratings of well-being. Another group tapered their total training volume by 10%/day over 10 days. A third group progressively reduced the intensity and volume of their interval training by 10%/day over 10 days. All three tapering regimens improved mood states and peak tethered swimming forces to similar extents (~5%) but none of the tapering regimens enhanced 100-m or 400-m swimming times.

Subsequently, Hooper et al. (1999) studied 10 elite swimmers who gradually decreased their pool training volume and intensity by 35% and 20% and their gym work by 90% for 2 weeks before a national swimming championship. Tapering reduced plasma norepinephrine concentrations after 100-m maximal swims but had no significant effect on 100-m swimming times.

Mujika et al. (2000) also found no change in the performances of well-trained male middle-distance runners (n = 4/group) who progressively reduced their low and high intensity training volume by either 50% or 75% over 6 days. Both tapers decreased erythrocyte count, mean corpuscular volume, haemoglobin concentration and mean erythrocyte haemoglobin content, but neither taper had any significant effect on ≥ 2 min 800-m running times.

Why the progressive tapers in the studies by Hooper et al. (1998, 1999) and Mujika et al. (2000) failed to improve exercise performance is not clear. Zarkadas et al. (1995) showed that similar exponential tapers significantly improved 5 km running times (Table 2.9). They studied 11 triathletes who performed 10 and 13-day tapers separated by 6 weeks. In the first 10-day

taper, one group (n = 3) reduced their training by 30% in a single step and the other group (n = 6) reduced their training exponentially by 50% with a time constant (τ) of $\leq$ 5 days. While the 30% single step reduction in training had no effect on performance, the 50% exponential reduction in training improved 5-km running time by 4% and maximal cycling power output by 5%. In the second 13-day taper, the triathletes varied the time constant of their 50% exponential reduction in training volume from $\tau \leq 4$ to $\tau \leq 8$ days. The rapid reduction in training ($\tau \leq 4$ days) improved 5-km running time by 2% and the more gradual reduction in training ($\tau \leq 8$ days) improved 5-km running time by 6%. Only the rapid reduction in training increased maximal cycling power output by 8%. During both exponential tapers, VO_{2peak} increased progressively from ~ 63 to 69 $mL.min^{-1}.kg^{-1}$ body mass and the ventilation 'threshold' rose from ~ 71% to 75% of VO_{2peak}.

Later, the same group studied another 11 triathletes who performed two 2-week tapers separated by 4-5 weeks (Banister et al. 1999). In the first taper, one group (n = 5) reduced their training by 22% in a single step and the other group (n = 6) reduced their training exponentially by 31% ($\tau \leq 5$ days) to produce a similar reduction in mean training volume. As in their previous study, the single step reduction in training had no effect on performance and the exponential reduction in training improved 5-km running time by ~ 4% and maximal cycling power output by 5%. In the second taper, the triathletes exponentially reduced their training volume either rapidly by 65% ($\tau \leq 4$ days) or more gradually by 50% ($\tau \leq 8$ days). In contrast to their previous findings, the rapid reduction in training improved 5-km running times and maximal cycling power outputs more than the gradual reduction in training (~ 6% and 8% vs. 2.5% and 4%, respectively). Again, VO_{2peak} increased progressively during both exponential tapers, from ~ 63 to 69 $mL.min^{-1}.kg^{-1}$ body mass and the ventilation 'threshold' rose from ~ 71% to 75% of VO_{2peak}.

In summary, this review suggests that ≥ 6 HIT sessions consisting of 8 x 4-5 min rides at 80-85% of W_{peak} are sufficient to maximally improve endurance performances in events where the athletes compete at 75-80% of W_{peak}. Six HIT sessions consisting of 12 x 30 s sprints at 175% of W_{peak} may also help to improve endurance performance. Apparently different adaptations to HIT at 85% and 175% of W_{peak} raise an interesting question of whether athlete's ability to resist fatigue at high work rates might be further improved by both types of training.

Another question is whether an optimum duration of a taper is influenced by preceding training intensity and percent reduction in training volume. Athletes training at higher than 70% of W_{peak}, intensities may require longer than 2 weeks to fully recover from exhaustive exercise before a competition. Conversely, competitors who reduce their HIT by > 50% may need to shorten their taper to prevent a loss of fitness. Until these questions have been answered, it would be premature for exercise physiologists to advise coaches on how to best prepare their athletes for competition.

CHAPTER 2.6

SUMMARY OF LITERATURE AND STATEMENT OF AIM OF THESIS

Adaptations to training have been studied extensively in previously untrained subjects. In such subjects, endurance training improves peak oxygen uptake (VO_{2peak}), increases capillary density of working muscle, raises blood volume and decreases heart rate during exercise. Endurance training also promotes greater muscle glycogen storage, increases sarcolemmal Na^+-K^+ ATPase activity and raises muscle mitochondrial content. More mitochondria improve respiratory control sensitivity and decrease reliance on carbohydrate utilisation during exercise. In contrast, sprint training has a greater effect on muscle glyco(geno)lytic capacity than on muscle mitochondrial content. Sprint training invariably raises the activity of one or more of the muscle glyco(geno)lytic or related enzymes and enhances sarcolemmal lactate transport capacity. Some groups have also reported that sprint training transforms muscle fibre types but those data are conflicting and not supported by any consistent alteration in sarcoplasmic reticulum Ca^{2+} ATPase activity or muscle physico-chemical H^+ buffering capacity.

While the adaptations to training have been studied extensively in previously sedentary individuals, far less is known about the responses to high-intensity interval training (HIT) in already highly trained athletes. The reported benefits of HIT prior to competition have been systematically studied (Lindsay et al. 1996; Westgarth-Taylor et al. 1997; Weston et al. 1997 and Stepto et al. 1999). These studies have shown that ≥ 6 HIT sessions, consisting of 6-9 x 5-min cycle rides at 80% of peak work rate (W_{peak}), 1-2 times a week, was sufficient to maximally increase W_{peak} values and simulated, 40-km time trial (TT_{40}) speeds of competitive cyclists by 4-5% and 3.0-3.5%, respectively. Later, Stepto et al. (1999) showed the greatest improvements in TT_{40} performances were after 6 HIT sessions consisting of either 8 x 4 min

rides at 85% of W_{peak} or 12 x 30 s rides at 175% of W_{peak}. Maximum 3.0-3.5% improvements in TT_{40} cycle rides at 75-80% of W_{peak} after HIT consisting of 4-5 min rides at 80-85% of W_{peak} supported the idea that athletes should train for competition at exercise intensities specific to their event. In contrast, the ~ 2% irnprovements in TT_{40} endurance performances after 30 s sprints at 175% of W_{peak} did not conform to the concept of training specificity and appeared to involve a different type of adaptation. Whereas faster TT_{40} performances after HIT at 85% of W_{peak} were associated with ~ 4% increases in W_{peak} values, faster TT_{40} performances after HIT at 175% of W_{peak} were independent any significant change in W_{peak}. Irrespective of the type of HIT, up to 1.5 km/h faster TT_{40} performances after HIT were more closely related to improvements in relative work rates from 72% to 76% of W_{peak} than to 15-20 W increases in W_{peak} values.

Abilities of athletes to sustain higher percentages of W_{peak} during exercise after HIT were not due to a reduction in carbohydrate utilisation at the same relative exercise intensity or to any increase in muscle glycolytic or oxidative enzyme activities. HIT slightly improved muscle H^+-buffering capacity but had no influence on muscle hexokinase, phosphofructokinase, citrate synthase and 3-hydroxyacyl CoA dehydrogenase activities. Faster TT_{40} cycling times after HIT probably also did not result from the athletes' belief that HIT should improve their performances. The TT_{40} cycling times of cyclists who completed HIT programs at different exercise intensities were not consistently decreased. It is also doubtful whether the improved performances after HIT were due to gains in strength. Improvements in muscle strength after resistance training do not enhance the endurance performances of well-trained athletes who are already capable of sustaining high power outputs in their chosen sport.

Equally poorly understood is the optimum reduction or 'taper' in intense training to recover from exhaustive exercise before a performance or competition. Only one group (Hickson and Rosenkoetter 1981; Hickson et al. 1982 and Hickson et al. 1985) has compared the effects of reductions in the frequency, duration and intensity of exercise on performance. Although these studies were reduced-exercise training or 'detraining' protocols, and not taper studies per se, the results provide an insight into the effects of reducing training components on performance. They found that it was more important to maintain training intensity than training volume. Whereas 33% or 66% decreases in training frequency or duration over 15 weeks maintained VO_{2peak} values, 33% and 66% decreases in training intensity reduced VO_{2peak} values by ~ 7.5% and 10%. A 66% reduction in training duration and 33% and 66% decreases in training intensity also shortened (~ 2 h) cycling endurance at 80% of pre-training VO_{2peak} by ~ 10%, 21% and 30%, respectively. Another group (Costill et al. 1985) showed that an ~ 95% reduction in pool training volume over 4 weeks shortened the stroke distances of competitive swimmers by ~ 8%. Alternatively, the shortened stroke distances might also have resulted from an ~ 83% decrease in training frequency. Swimmers often complain that they lose the 'feel' for the activity if they miss pool sessions for several days. Most studies have shown that 20-80% single-step reductions in training volume over 1-4 weeks have little effect on exercise performance.

In contrast, progressive 30-75% reductions in pool training volume over 2-4 weeks have been shown to improve swimming performances by 2-3%. However, more rapid ≤ 2 weeks, but otherwise, similar tapers had no effect on 100-m swimming and 800-m running times. Why such tapers did not improve exercise performances is not clear. Equally rapid exponential tapers improved 5-km running times by up to 6%. One possible explanation for these discrepancies may be that the optimum taper depends on the intensity of the athletes' preceding training and their need to recover from exhaustive exercise in order to compete. A

question that now warrants attention is how the optimum duration of a taper is influenced by preceding training intensity and percent reduction in training volume?

It is quite clear from the literature summarised above that "aerobic" training, endurance or interval training, has a marked effect on the athletic and maximal performances of previously untrained individuals. Many studies have suggested that these improvements in performance are made possible by more effective muscle enzymes of the glycolytic and oxidative pathways, increased activity of the muscle fibre Na^+/K^+ATPase pump, improved K^+ re-uptake capacity, and increased glycogen storage coupled with an ability to utilise fat as an energy source. Although the literature is unanimous that SR-Ca^{2+} re-uptake capacity is not affected by aerobic training in previously untrained young subjects, more research is warranted to fully investigate the possibility that aerobic training, endurance or interval, might result in muscle fibre –type transformations and/or improve muscle fibre H^+ buffering capacity.

While the body of scientific literature on previously untrained individuals is extensive, far less is known about the effects of training on already-trained individuals. It is well established that these already-trained individuals' responses to training differ from those of their untrained counterparts, and the improvements in performance following high-intensity training are independent of changes in muscle enzyme activities and peak oxygen consumption. The improvements have been associated with an enhanced ability to sustain a higher fraction of VO_{2peak} in runners and W_{peak} in cyclists, a greater resistance to fatigue and less collectively improved muscle fibre H^+ buffering capacity. However, these conclusions have been based exclusively from interval sessions and performance tests of short duration, which have had little relation to what athletes are doing in the field.

A reduction in training load or taper prior to a competition is speculated to further improve performance after a period of intensive training. The guidelines from the literature on how to reduce the training are set out as follows: a gradual reduction in training load (60 - 90%) and a maintenance or increase in training intensity in the 7- 21 days prior to a competition. Despite these guidelines many tapering studies have not significantly enhanced performance, but have suggested that the benefits of taper lie in improving the haematological, metabolic, psychological and physiological status of these athletes, variables which are supposedly hampered by repeated intensive training.

Therefore, the aim of the series of investigations is to better understand the physiological responses of already trained cyclists to selected laboratory performance tests and road racing during training and taper. The hypothesis for each chapter of this thesis is highlighted in the respective chapter introductions.

CHAPTER 3

PHYSIOLOGICAL RESPONSES TO A VARIABLE-INTENSITY TEST, LINEAR RAMP TEST AND ROAD CYCLING PERFORMANCE IN WELL-TRAINED CYCLISTS

RATIONALE AND AIM

When athletes are examined in a laboratory, they often perform progressive exercise tests to exhaustion. Measurements of plasma lactate concentrations at sub-maximal work rates and determinations of peak oxygen uptake ($VO_{2\,peak}$) at fatigue indicate whether athletes are recreational, competitive or elite. Faster athletes usually have lower plasma lactate concentrations at sub-maximal work rates (Coyle et al. 1988, 1991; Farrell et al. 1979; Heck et al. 1985; Jacobs 1986; Tanaka and Matsura 1984) and higher $VO_{2\,peak}$ values at fatigue (Costill et al. 1970; Foster 1983; Hawley and Noakes 1992). However, rises in lactate concentration with increasing VO_2 may be less reliable predictors of athletic performance in groups of athletes with similar abilities. Among comparable athletes, $VO_{2\,peak}$ values are poorly correlated with performance (Conley and Krahenbuhl 1980; Daniels et al. 1978; Noakes 1988; Pollock 1977; Scrimgeour et al. 1986) and maximum steady state plasma lactate concentrations vary from 2 - 8 mmol·L-1 (Stegmann and Kindermann 1982).

One possible explanation for the poor predictive value of $VO_{2\,peak}$ and lactate measurements among more homogeneous groups of athletes is the duration of time they are able to exercise at or above their "*anaerobic threshold*". As Stegmann and Kindermann (1982) showed, certain athletes are able to maintain exercise even when their plasma lactate concentrations exceed the previously defined *critical* value of 4 mmol·L^{-1} (Heck et al. 1985; Kindermann et al. 1979).

Another possible reason for the limited usefulness of lactate and $VO_{2\,peak}$ measurements in distinguishing between a homogenous group of athletes is that they are obtained from linear exercise ramp models involving fixed work rates. Many athletes do not exercise using linear ramp protocols nor do they race in competition at constant work rate. Palmer et al. (1994) showed that elite cyclists racing in a pack randomly vary their work rates from around 50% to almost 100% of peak sustained power output, independently of the course terrain. Coyle et al. (1991) also noted that TT_{40} performances are determined by a combination of the cyclists' W_{peak} values and their ability to sustain a high percentage of that W_{peak} during exercise.

Performances in exercise involving rapid changes in the cardiorespiratory and metabolic responses to transient increases and decreases in work rates may be related to not only an athlete's lactate threshold and $VO_{2\,peak}$ but also their ability to recover from high-intensity exercise. The hypothesis therefore was to examine whether heart rate recoveries from laboratory exercise tests would better predict cycling ability and performance than the conventional measures of cycling performance in well-trained cyclists. Accordingly, a variable-intensity cycling exercise test was compared with a conventional linear ramp cycling exercise test to exhaustion and the relationship between these findings and cycling performances in an 80-km, mass-start road race was examined.

Methods

Seven male endurance-trained cyclists who competed in various, local, mass-start road races during the previous cycle racing season were recruited for this investigation. The subjects' characteristics and their cycling performances in laboratory tests are presented in Tables 3.1 and 3.2. All of the subjects had competed for at least three preceding seasons prior to this study and ranged in ability from good club level cyclists to provincial and national level cyclists.

Subject 1 was a national level cyclist, subjects 2 and 3 were provincial level cyclists and subjects 4, 5, 6 and 7 were good club level cyclists

Laboratory exercise testing procedures were approved by the Research and Ethics Committee of the University of Cape Town Medical School. All of the subjects had been tested in the laboratory previously and were fully acquainted with the nature of the investigation before they signed consent forms.

Laboratory exercise tests

A conventional linear ramp exercise test to exhaustion and a variable-intensity exercise test were conducted in a random order separated by two weeks. On the day before each test, the subjects were asked to refrain from strenuous exercise and consume the same diet as before the previous test. After arriving at the laboratory, subjects urinated and were weighed to the nearest 0.1 kg in their shorts on a Seca precision balance (Model 770, Bonn, Germany). The subjects' body masses recorded on their first visit to the laboratory were used to establish the work rates to be employed in the subsequent exercise tests in $W \cdot kg^{-1}$ body mass. At this stage, the subjects' biceps, triceps, subscapular, and supra-iliac skinfolds were also measured in order to estimate their percent body fats, as described by Durnin and Womersley (1974).

Following the skinfold measurements, an 18 gauge Teflon cannula (Jelco, Johnson and Johnson, Halfway house, South Africa) was inserted into an antecubital vein and connected to a 3-way stopcock (Uniflex, Mallinckrodt, Hannef-Seig, Germany). This cannula was used for the collection of venous blood samples (5 mL) and was flushed periodically with 2-3 mL of sterile saline containing heparin ($5\ IU \cdot mL^{-1}$) to prevent blood clotting.

After the withdrawal of a resting blood sample, each subject performed one of the two exercise tests. Both exercise tests were conducted on an electronically braked cycle ergometer (Lode, Gronigen, Netherlands), modified with clip-in pedals, a racing saddle and a low profile handlebar. Power output on this ergometer is constant and independent of pedalling frequency between 80 and 120 revolutions·min^{-1}. Before each exercise test, the saddle height and handle bar position was adjusted to the subject's requirements and they warmed-up at a work rate of 50 W less than the starting work rate of 3.5 W·kg^{-1} in the exercise tests. The duration of the warm-up was self-selected and the same before each exercise test.

In the conventional linear ramp exercise tests to exhaustion, the starting work rate of 3.5 W·kg^{-1} was maintained for 150 s and then increased by 1 W·kg^{-1} every 150 s until the subject's pedalling frequency dropped from 90-110 to ≤ 75 revolutions·min^{-1} or their respiratory exchange ratio (RER) was > 1.1. Peak power output (W_{peak}) at fatigue was defined as the last completed work rate in W·kg^{-1} plus the fraction of time spent in the final non-completed work rate multiplied by 1 W·kg^{-1}.

The variable-intensity exercise test consisted of four 90 s increases in work rate from 3.5 W·kg^{-1} to 4.5, 5.5, 4.5 and 5.5 W·kg^{-1} separated by 60 s rides at the initial work rate of 3.5 W·kg^{-1}. Well-trained cyclists tested in our laboratories often exhibit power to weight ratios between 5.5 and 7.0 W.kg^{-1} (Hawley and Noakes 1992; Lindsay et al. 1996; Westgarth-Taylor et al. 1997). The workloads were designed to mimic road cycle racing in a non-invasive test of short duration. These linear ramp and variable-intensity exercise protocols are shown in the lower panels of Fig. 3.2.

Measurements of gas exchange

During the exercise tests and for 5 min after stopping pedalling, the subjects wore a nose-clip and breathed through a mouthpiece connected to an Oxycon Alpha automated gas analyser (Mijnhardt, The Netherlands). Before each test, the analyser was calibrated with a Hans Rudolph 3 l syringe, room air and a 5% CO_2: 95% N_2 gas mixture. Analyser outputs were processed by a computer, which calculated $l \cdot min^{-1}$ ventilation (V_E), oxygen consumption (VO_2) and CO_2 expiration (VCO_2) values for each breath. Peak VO_2 values were the average of the highest VO_2 values measured over 30 s in the final work rate(s).

Measurements of heart rates

On each visit to the laboratory, the subjects were provided with a Polar Sports Tester heart-rate monitor (Polar Electro OY, Kempele, Finland). This monitor recorded momentary heart rates at 15 s intervals throughout the exercise tests and the 5-min recovery periods. Heart rates recorded during these periods were "down-loaded" to a Microsoft EXCEL spreadsheet (Microsoft Corp. Redmond, WA, USA) in a laptop computer for subsequent analyses.

Declines in VO_2 and heart rates (Y) over the 5-min recovery period were treated as exponential functions of time (t) and $Y = A \cdot e^{[-B \cdot t]} + C$ equations were fitted to the data by non-linear, least squares analyses (Graphpad Software, San Diego, CA). Curves were compared by calculating the fall on VO_2 or heart rate in the first minute of the exponential decline.

Measurements of venous plasma lactate concentrations

Before, during and after the rides, venous blood samples (5 mL) were drawn at the end of each work rate and at 1-min intervals during a 5-min recovery. Blood samples were placed into tubes containing potassium oxalate and sodium fluoride and stored on ice until the end of the test.

The tubes were then centrifuged at 2,500 x g for 10 min at 4 °C and the supernatants were stored at - 20 °C for later determinations of plasma lactate concentrations. Plasma lactate concentrations were measured with a spectrophotometric (Beckman Model 35, Beckman Instruments Inc., Fullerton, Ca, USA) enzymatic assay (Lactate PAP, BioMerieux, Lyon, France).

Road cycle racing performances

On the weekend between the laboratory linear ramp and variable-intensity exercise tests, the subjects competed in an 80-km mass-start road race. This race consisted of four laps of a 20-km hilly course and took place on a sunny, warm day (~23° C) with a slight breeze, relative humidity of ~50%, with the elevation ranging between 50 and 250 m above sea level. The subjects cycled with more than 100 other cyclists while wearing a heart rate monitor. At the end of the event, the subjects' performance times were noted and their momentary heart rates at 1-min intervals were "down-loaded" into a laptop computer. Although the values provided by these heart rate monitors have been shown to be reliable and valid (Leger and Thiviere 1988), there were some problems with the electronic signal when the subjects rode in a bunch. Occasionally, the wrist-mounted receiver of one subject appeared to transiently interfere with signal transmissions from another cyclist's chest-electrode belt. Apparent heart rates of > 220 or ≤ 50 beats·min^{-1} were therefore excluded from subsequent analyses.

Statistical analyses

Results are presented as means ± standard deviations (SD) from n = 7 subjects. Rises in VO_2, heart rate and plasma lactate concentration with increasing work rates in the two laboratory exercise tests were compared with two-way analyses of variance for repeated measures followed by Scheffes post hoc-tests to locate where differences might have been significant.

Spearman's correlation coefficients (r) between the data from the laboratory exercise tests and the performances in the field were assessed with non-parametric Wilcoxon signed rank tests. Two-tailed values of $P \leq 0.05$ were regarded as significant.

Results

Figure 3.1 shows the subjects' heart rates during the 80-km mass-start road race. Subjects 1, 2 and 3 finished the race in that order amongst the leading pack in 146 min. Subjects 4 and 5 completed the race in 154 min and 165 min, respectively, while subjects 6 and 7 abandoned the race after 92 min and 85 min when they were unable to keep up with the leading bunch and lost the advantage of 'drafting'.

Racing performances were not related to the subjects' heart rates during the rides (Fig. 3.1). All of the cyclists rode the first 85 min of the race with heart rates varying randomly from ~ 70% to ~100% of peak and averaging ~ 90% of peak. These heart rates corresponded to a range of exercise intensities from ~ 55% to ~ 100% of VO_{2peak} and a mean exercise intensity of ~ 80% of $VO_{2\,peak}$ estimated from the laboratory exercise tests.

Table 3.1. Subjects' characteristics.

Subject No	Age Y	Body mass kg	Body fat %	Cycle racing Y
1	17	59	8	4
2	17	67	10	6
3	21	71	12	7
4	20	78	15	6
5	22	60	7	10
6	18	72	12	5
7	20	74	15	4
Mean	19	69	11	6
± SD	2	7	3	2

Subjects were numbered from 1 to 7 according to their performances in an 80-km mass-start road cycle race (Fig. 3.1). Subject 1 was a national level cyclist, subjects 2 and 3 were provincial level cyclists and subjects 4, 5, 6 and 7 were good club level cyclists. No is the subject number; Y is age in years and year's cycling competitively.

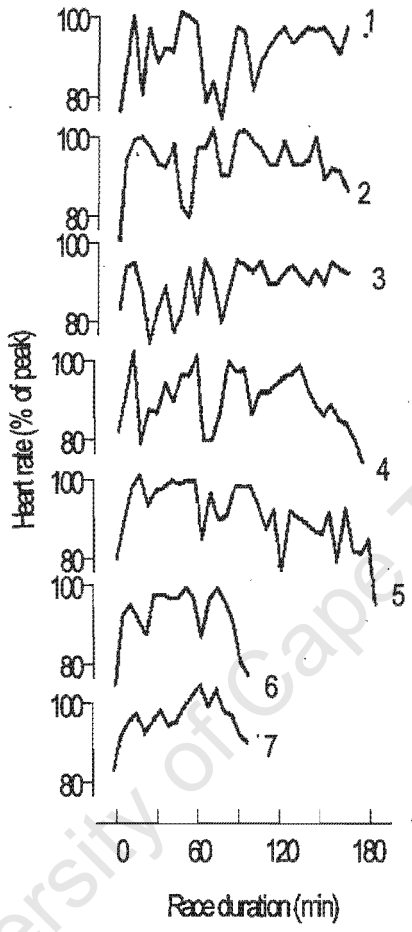


Figure 3.1. Heart rates during the 80-km mass-start road cycle race.

Subjects' heart rates are expressed as a percent of their highest heart rates in either the race or in the laboratory exercise tests.

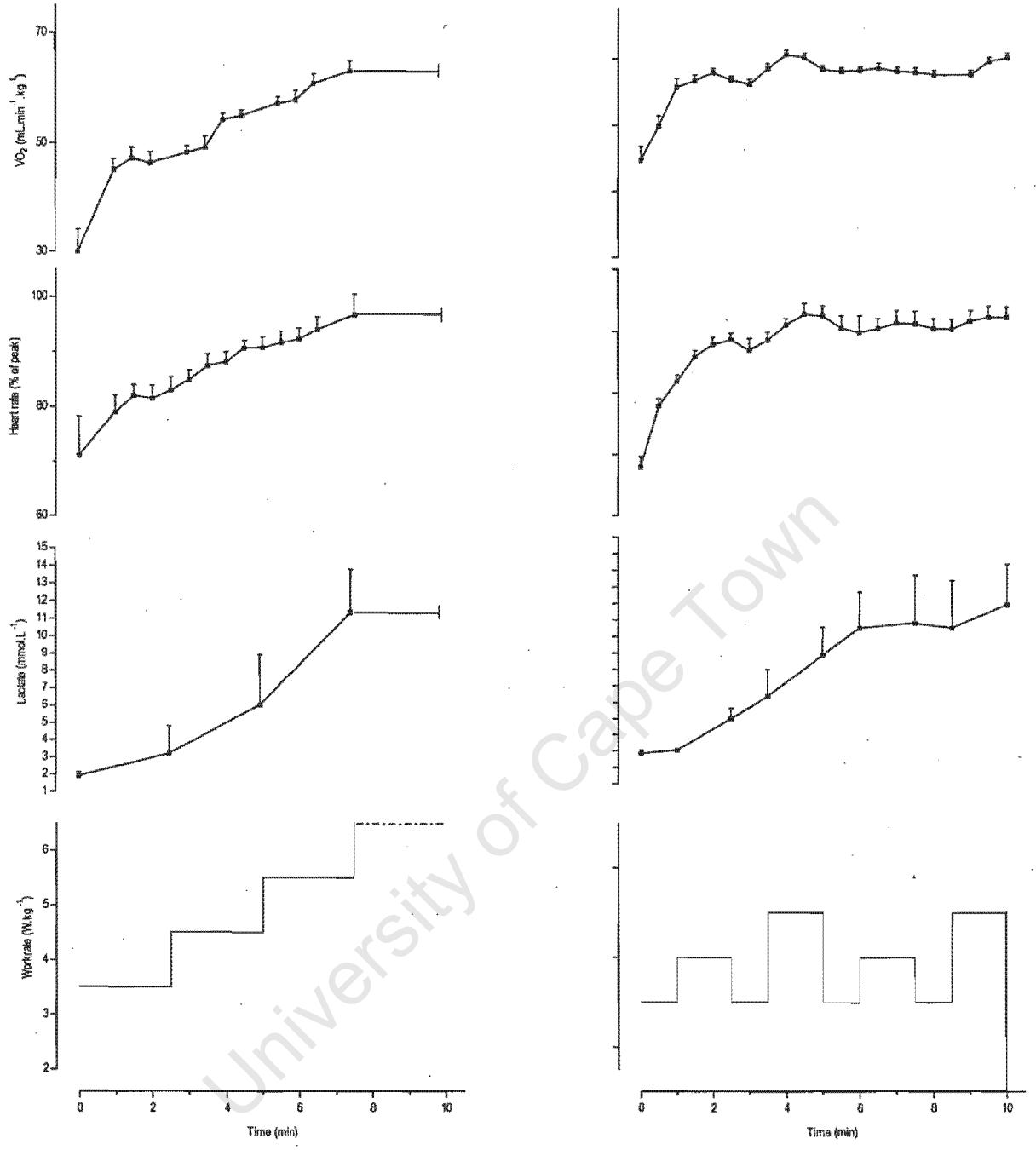


Figure 3.2. Rises in VO_2, heart rate and plasma lactate concentration during the linear ramp and variable-intensity exercise tests. Results from the linear ramp (*left panels*) and variable-intensity (*right panels*) exercise tests are means ± SD (n = 7). Peak oxygen consumptions, heart rates and power outputs in the two exercise tests are given in Table 3.2.

Rises in VO_2 and heart rates with increasing work rates were similar in the two laboratory exercise tests (Fig. 3.2). In the linear ramp and variable-intensity exercise protocols, VO_2 and heart rates rose to 69 ± 3 and 66 ± 4 mL·min^{-1}·kg^{-1} and 191 ± 10 and 187 ± 5 beats·min^{-1}, respectively (Table 3.2). The highest heart rates of 194, 192 and 188 beats·min^{-1} in subjects 1, 2 and 7 were recorded during the race (Fig. 3.1). The highest heart rates in the other subjects were recorded during the laboratory exercise tests (Table 3.2).

Although the peak VO_2 and heart rate values were comparable in the two exercise tests, the final work rates in the linear ramp exercise protocol were generally higher than in the variable-intensity exercise protocol (Table 3.2, Fig. 3.2). In the linear ramp exercise tests, subjects 1 - 6 all fatigued after ~7.5 min at work rates of 6.5 W·kg^{-1}, while subject 7 fatigued at 6 min during the 150 s of exercise at 5.5 W·kg^{-1}. In contrast, the subjects only had to perform two 90 s maximum work bouts at 5.5 W·kg^{-1} in the 10 min variable-intensity exercise tests and even subject 7 managed to complete those work bouts when they were preceded by 60 s rides at the initial work rate of 3.5 W·kg^{-1}.

The presence and/or absence of fatigue in the two exercise tests had no effect on the declines in VO_2 and heart rates during a 5-min recovery period. Within 30-60 s of the end of either exercise test, VO_2 and heart rates fell exponentially from > 60 to ≤ 20 mL·min^{-1}·kg^{-1} and from > 90% to ≤ 60% of peak (Fig. 3.3). Only plasma lactate concentrations were different in the two 5-min recovery periods (Fig. 3.3). After the linear ramp exercise tests to exhaustion, plasma lactate concentrations continued to rise from ~ 11 to 15-16 mmol·L^{-1} and, after the variable-intensity exercise tests, plasma lactate concentrations remained at 10-12 mmol·L^{-1} ($P \leq 0.01$).

In the 5-min following the variable-intensity exercise tests, plasma lactate concentrations were lower in the better cyclists than in the poorer cyclists (Fig. 3.4). Mean recovery plasma lactate concentrations were 7 - 8 mmol·L^{-1} in subjects 1 and 2, 11 - 12 mmol·L^{-1} in subjects 3, 4, 5 and 6.

Table 3.2. Subjects' peak power outputs, oxygen consumptions and heart rates in the laboratory exercise protocols.

Subject No	Linear ramp exercise			Variable-intensity exercise		
	W_{peak} W·kg^{-1}	$VO_{2\,peak}$ mL·min^{-1} kg^{-1}	HR_{peak} beats·min^{-1}	W_{peak} W·kg^{-1}	$VO_{2\,peak}$ mL·min^{-1} kg^{-1}	HR_{peak} beats·min^{-1}
1	6.5	72	193	5.5	71	185
2	5.9	65	189	5.5	67	182
3	5.9	69	201	5.5	63	190
4	5.6	72	195	5.5	70	192
5	6.3	72	198	5.5	62	183
6	5.6	64	187	5.5	66	192
7	4.9	67	174	5.5	61	182
Mean	5.8	69	191	5.5	66	187
± SD	1.0	3	10	0	4	5

Peak power output (W_{peak}), oxygen consumption ($VO_{2\,peak}$) and heart rate (HR_{peak}) were measured as described in the Methods. Peak heart rates in the laboratory exercise tests were similar to the 190 ± 2 beats·min^{-1} peak heart rates in the 80 km mass-start road cycle race.

and 16 mmol·L^{-1} in subject 7. After the linear ramp exercise tests to exhaustion, there was no significant relationship between the subjects' recovery plasma lactate concentrations and their rankings from 1st to 7th in the 80-km race (data not shown).

Exponential declines in VO_2 values 30-60 s after the variable-intensity exercise tests were also unrelated to cycling ability (Fig. 3.5). In the first min of the exponential declines, VO_2 values fell by 16 - 40 mL·min^{-1}·kg^{-1} and varied randomly in the subjects ranked from 1 to 7 on their racing performances.

In contrast, the corresponding exponential decreases in heart rate, 30-60 s following the variable-intensity exercise tests, were more rapid in the better cyclists than in the poorer cyclists (Fig. 3.6). In the subjects ranked from 1 to 7 on their racing performances, heart rates declined by 31%, 34%, 20%, 21%, 16%, 11% and 9% of peak·min^{-1}, respectively. After both laboratory exercise tests, the Spearman's correlation coefficient (r) between the subjects' initial falls in heart rate and their ranking in the 80-km race was greater (- 0.93 vs. – 0.75) than that between W_{peak} and race position (Fig. 3.7). Although W_{peak} and percentages of W_{peak} at a plasma lactate concentration of 4.0 mmol·L^{-1} tended to be higher in the better cyclists, the correlation between these values and the subject's race positions was - 0.75 (Fig. 3.7).

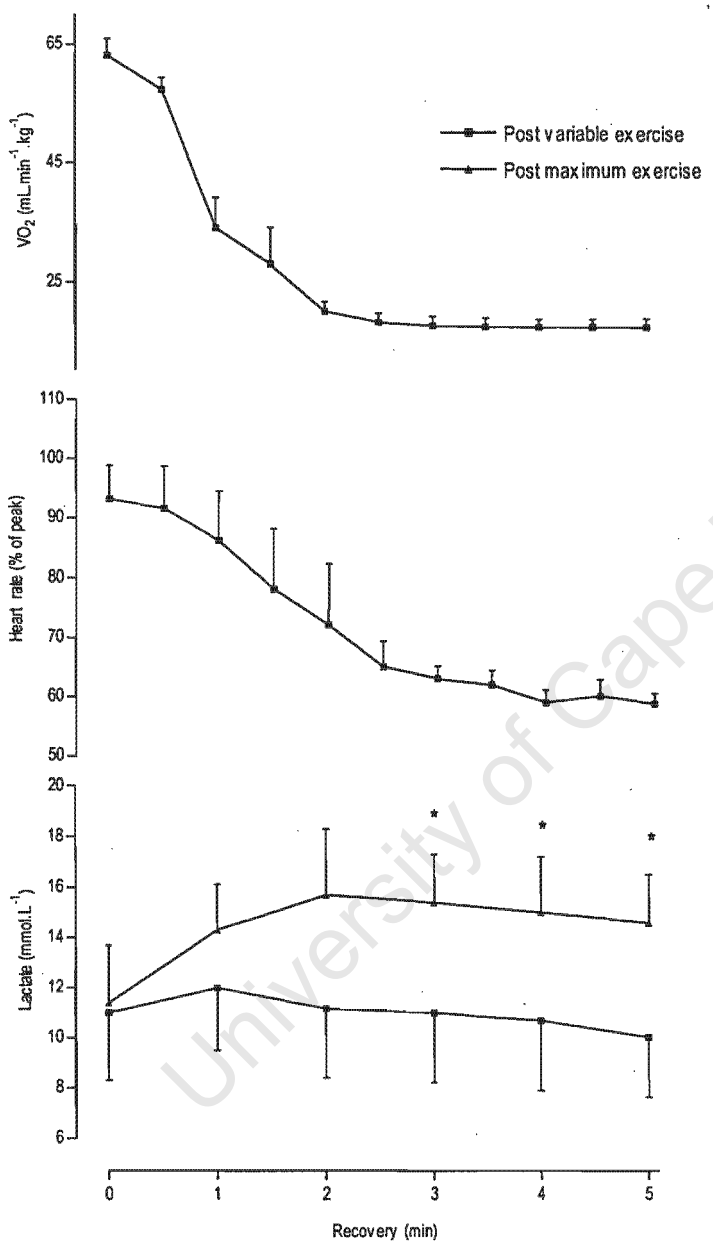


Figure 3.3. Changes in VO_2, heart rate and plasma lactate concentration following the exercise tests.

Means and SD's (n = 7) of the declines in VO_2 and heart rates following the linear ramp (maximum) exercise tests were similar to those shown after the variable-intensity (variable) exercise tests. In contrast, plasma lactate concentrations were higher after the linear ramp (maximum) exercise tests to exhaustion than after the variable-intensity (variable) exercise tests in the final 3 min of recovery ($P \leq 0.01$). *Indicates significant differences $P \leq 0.001$.

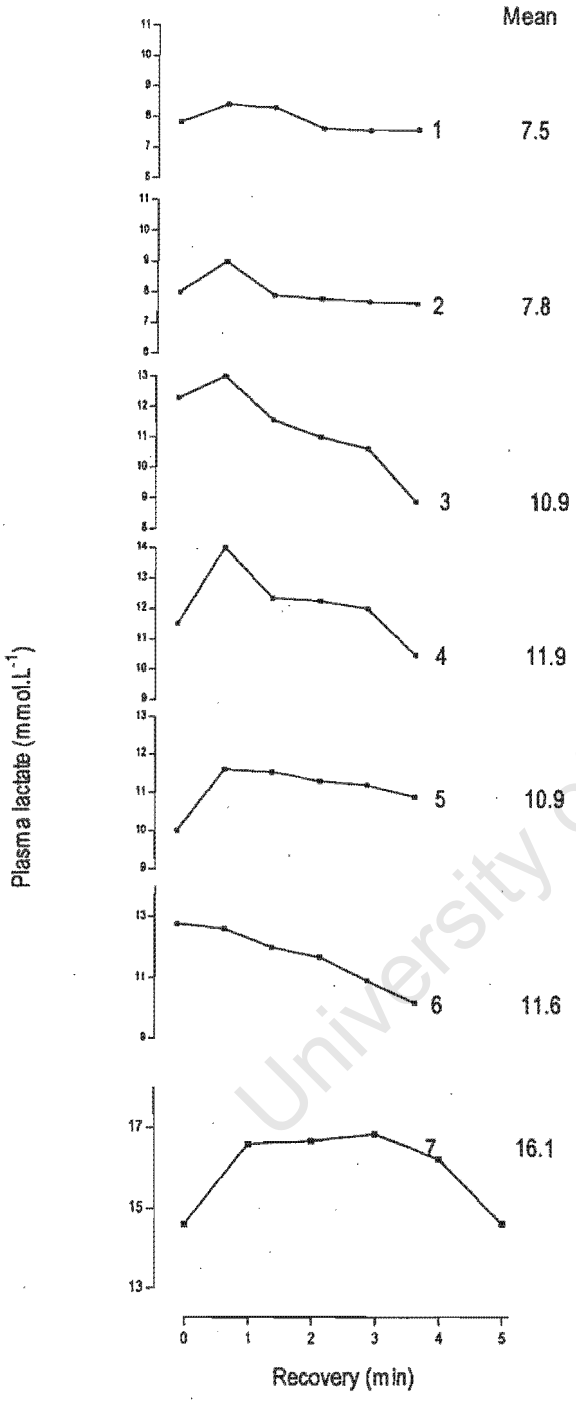


Figure 3.4. Plasma lactate concentrations of each subject after variable-intensity exercise tests. Subjects were ranked from 1 to 7 on their performances in the 80-km mass-start road cycle race (Fig. 3.1). Mean values are the average plasma lactate concentrations at 1-min intervals over the 5-min recovery period.

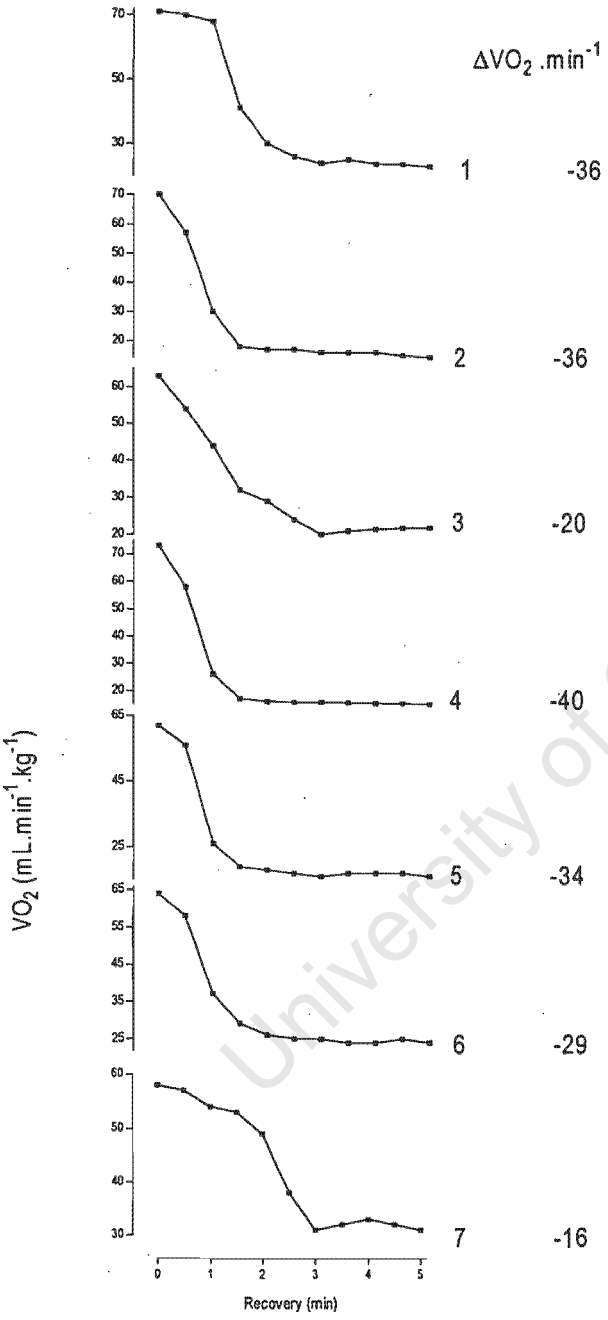


Figure 3.5. Declines in individual VO_2 values after the variable-intensity exercise tests. $\Delta VO_2 \cdot min^{-1}$ values are the $mL \cdot min^{-1} \cdot kg^{-1}$ falls in VO_2 in the first min of the exponential decline in VO_2 after 30-60 sec of recovery. Calculations of $\Delta VO_2 \cdot min^{-1}$ values are described in the Methods.

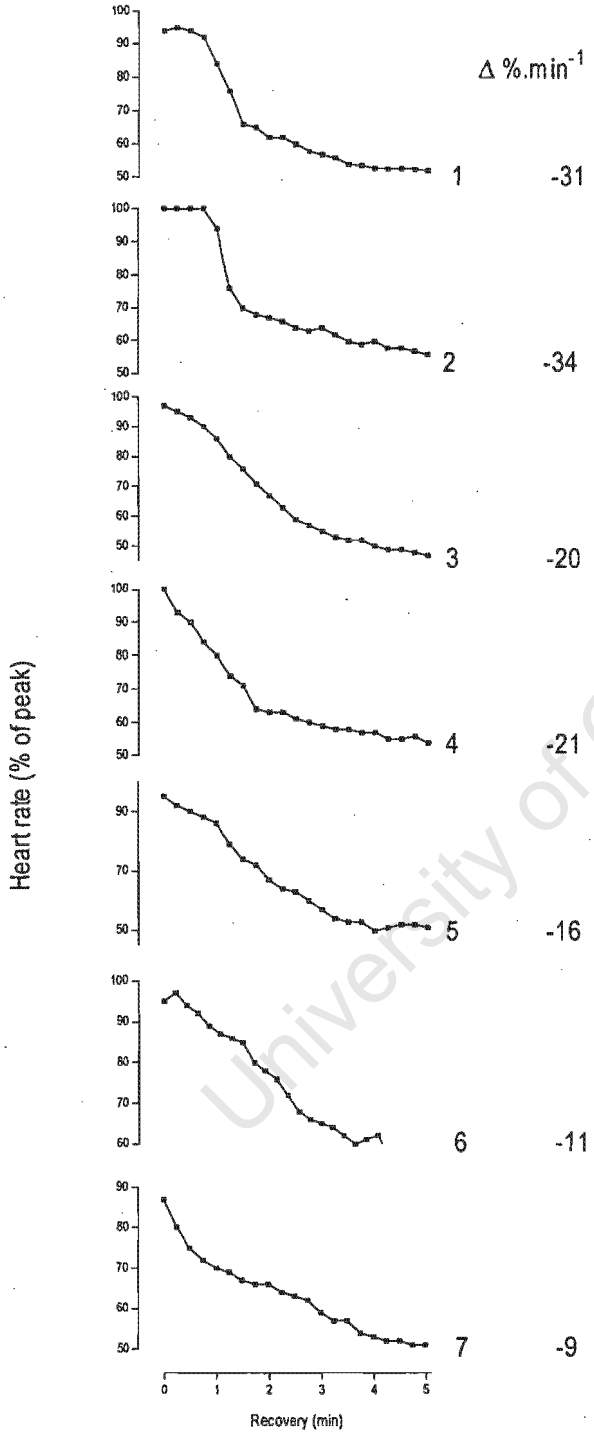


Figure 3.6. Declines in each subject's heart rates after the variable-intensity exercise tests.

The Δ% min^{-1} peak heart rate values of subjects 1 to 7 were calculated in the same way as the $\Delta VO_2 \cdot min^{-1}$ values in Fig. 3.5.

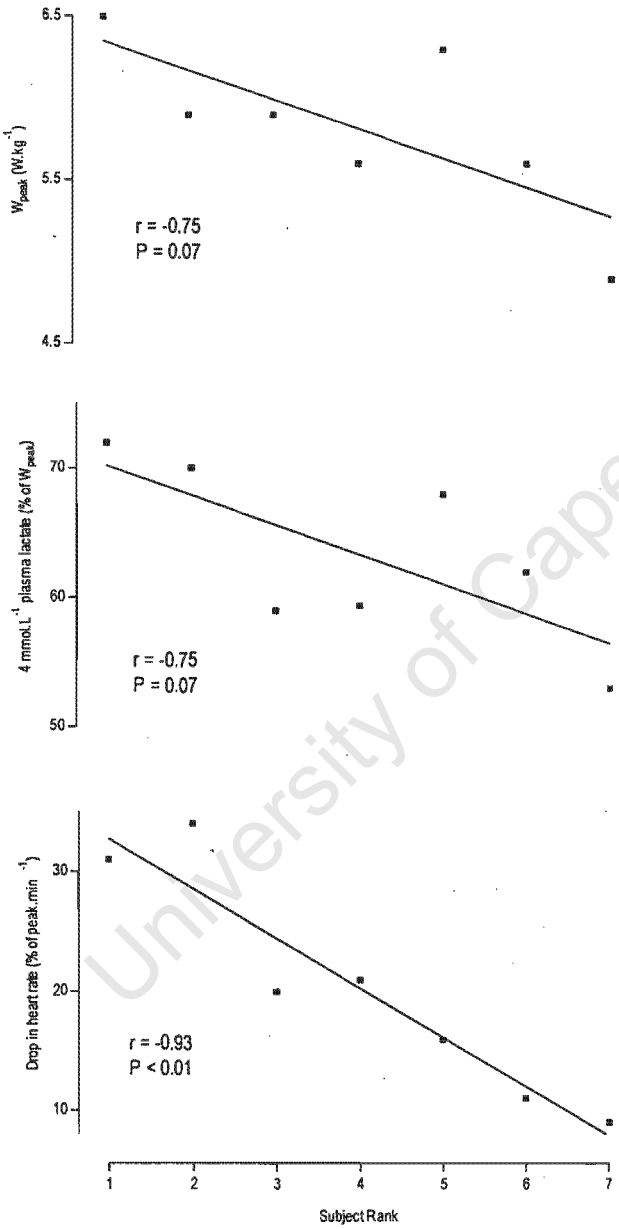


Figure 3.7. Peak power outputs (W_{peak}), % of W_{peak} at a plasma lactate concentration of 4 mmol·L^{-1} and recovery heart rates in the subjects ranked from 1 to 7 on their 80 km racing performances. W_{peak} values at exhaustion are from Table 3.2 and recovery heart rates are from Fig. 3.6. Spearman's correlation coefficients (r) were not improved by step-wise regressions.

Discussion

As would be expected, the subjects' performances in the 80-km mass-start road race (Fig. 3.1) reflected their rankings as national, provincial and good club level cyclists (Table 3.1). However, racing performances were not related to the subjects' heart rates during the road race. The significance of cardiovascular drift during the road race was probably negligible, since professional cyclists are known to maintain an adequate hydration status during stages (Saris et al. 1989). All of the cyclists rode the race with heart rates corresponding to a range of exercise intensities from ~ 55% to ~ 100% of VO_{2peak} and a mean exercise intensity of ~80% of VO_{2peak}.

Palmer et al. (1994) also found that cyclists racing in a pack randomly varied their work rates from around 50% to almost 100% of VO_{2peak}, independently of the course terrain. They suggested that the variable work rates of the cyclists were largely due to their periodically "drafting" behind other competitors. Kyle (1979) and McCole et al. (1990) showed that the reduction in the wind resistance of a cyclist riding at ~ 40-km/h behind a pack of eight riders decreased VO_2 by almost 40%. This energy saving has been calculated to allow a pack of cyclists to ride up to 5-km/h faster than a single cyclist (Kyle 1988). Such large advantages of riding within a group of cyclists probably explain why subjects 6 and 7 abandoned the race after 92 min and 85 min, when they were unable to keep up with the pack. Once a rider loses contact with the main bunch, it takes a supra-maximal effort to regain their position.

Variable work rates during the race did not lead to greater peak heart rates than when the work rates were dictated by the laboratory exercise tests, as has been found in simulated competition (Foster et al. 1993). In both the linear ramp and variable-intensity exercise protocols, peak heart rates were similar to those in the field (Table 3.2). Rises in VO_2 with

increasing work rates were also comparable in the two laboratory exercise protocols (Fig. 3.2), as were the exponential falls in heart rate and VO_2 during a subsequent 5-min recovery period (Fig. 3.3). The generally higher final work rates in the linear ramp exercise tests than in the variable-intensity exercise tests (Table 3.2) increased the plasma lactate concentrations following exercise (Fig. 3.3). Based on results from conventional laboratory tests of VO_{2peak} and W_{peak} alone, when corrected for body mass (Table 3.2), all the subjects were of a similar competitive nature, but their lactate, recovery heart rates responses and their road-racing performance was indicative of the cyclists' athletic abilities.

In the 5-min following the variable-intensity exercise tests, mean plasma lactate concentrations were lower in the better cyclists than in the poorer cyclists (Fig. 3.4). The reduced accumulation of lactate in the better cyclists may have resulted largely from them working at lower relative intensities in the variable-intensity exercise tests. Stepto et al. (2001) compared metabolic and muscular data from intense interval training performances by competitive endurance cyclists with data from a professional cyclist undertaking the same intervals, the same relative intensity and laboratory conditions. They observed smaller disturbances in acid-base status in the professional cyclist at higher absolute work rates with blood and muscle lactate levels of 20 and 34% lower than in the endurance trained cyclists. Similarly peak power outputs tended to be higher in the better cyclists than in the poorer cyclists in this study (Table 3.2). After the linear ramp exercise tests to exhaustion, there was no significant relationship between the subjects' recovery plasma lactate concentrations and their rankings from 1st to 7th in the 80-km race (data not shown). Therefore, variable intensity tests may better predict cycle racing ability.

Exponential declines in VO_2 after the variable-intensity exercise tests were also unrelated to cycling ability (Fig. 3.5). In the first min of the exponential declines, VO_2 values fell to varying extents in the subjects ranked from 1 to 7 on their racing performances.

In contrast, the corresponding exponential decreases in heart rate following the variable-intensity exercise tests were more rapid in the better cyclists than in the poorer cyclists (Fig. 3.6). After both laboratory exercise tests, the correlation (r) between the subjects' initial falls in heart rate and their race position in the 80 km race was -0.93 and greater than that between W_{peak} and race position (Fig. 3.7). Although peak power outputs tended to be higher in the better cyclists, the correlation between the subjects' W_{peak} values and their 80-km race positions was only - 0.75. Previously, studies have found much closer (> - 0.9) correlation's between cyclists' W_{peak} and their performances in laboratory simulated 20-km and 40-km cycling time-trials (Hawley and Noakes 1992; Lindsay et al. 1996). However, those W_{peak} values were not corrected for body mass and the time-trials were conducted on a flat surface, with no wind resistance and at a constant rolling resistance. Under these conditions, the advantages of the greater power outputs of the heavier cyclists would not have been reduced by their having to work harder to climb hills and overcome greater wind and rolling resistances, as would occur in a road race (Swain et al. 1987; Swain 1998).

The poorer correlation between peak power output and performance in this study could also have been due to the subjects having to be ranked on their race position rather than on their race time. When cyclists ride in a pack, there is little difference in their race times and their position in the pack is very dependent on their ability to "draft" behind the other cyclists and maintain the required work rate during the course of the race (Palmer et al. 1994). However,

what will determine their final race position in a pack, will largely be their ability to generate high power outputs (sprint) leading up to the finish line.

It is also possible that W_{peak} and VO_{2peak} values at a constant work rate and fixed pedalling frequency in a linear ramp exercise test to exhaustion is a better measure of a cyclist's ability to sustain the more steady-state exercise intensities observed in time-trials than to perform the variable exercise intensities found in mass-start road races (Palmer et al. 1994). While W_{peak} and VO_{2peak} values have been shown to indicate an athlete's competitiveness in cycling time-trial or distance running events (Costill et al. 1970; Foster 1983; Hawley and Noakes 1992; Lindsay et al. 1996) it remains to be determined whether heart rate recoveries after exercise could also be used to predict performances in more steady-state endurance events. More rapid heart rate recoveries in better cyclists than in poorer cyclists were presumably due to a faster recovery from fatigue and a decreased promotion of sympathetic drive by muscle afferent nerve activity. Whether there is any relationship between the rate of recovery from fatigue and resistance to fatigue is not known. However, we can conclude that performances in mass-start road races may not only be related to the cyclists' ability to sustain a given power output but also to recover rapidly from high work rates.

CHAPTER 4

THE TRAINING AND TAPERING PRACTISES OF ELITE MALE AND FEMALE TRIATHLETES

RATIONALE AND AIM

It has been suggested that a high volume of training in the days immediately preceding an event is detrimental to physical performance (Zarkadas et al. 1995). In accordance with this suggestion, many endurance athletes progressively "taper" (reduce) their training volume before a competition to enhance their performance (Houmard et al. 1989, 1994; Neary et al. 1992; Mujika et al. 1996a, 1998). Previous research on tapering suggests that a graded reduction in training volume of 60-90% within the last 7-21 days before a competition, coupled with a maintenance or increase in interval training, improves athletic performance (Shepley et al. 1992; Gibala et al. 1994; Houmard and Johns 1994).

Despite these scientific accounts of laboratory-based tapering regimes, to the best of our knowledge, no studies have documented the tapering practices of competitive athletes in the field leading up to a major competition or provided reports that athletes follow these recommendations. The hypothesis of this study was to investigate if elite triathletes follow the tapering guidelines currently available in the scientific literature. The aim of this study therefore was to examine the training and tapering practices of elite Olympic-distance triathletes in the 6 weeks preceding a national championship competition.

Methods

Subjects

Sixteen well-trained male and female, junior and senior triathletes participated in this study. Their characteristics are reported in Table 4.1. The triathletes were all of international standard in their respective age groups.

Training diaries

Two weeks prior to the South African national triathlon championship the triathletes were given training diaries and asked to record their training, on a daily basis, leading up to the event. The triathletes were requested to maintain their normal pre-race training routines.

The diary consisted of four sections: swimming, cycling, running, and any other training. An example of how to record the training in the diary was attached to each section. The training performed in the exercise modes was recorded as repetitions. Each set of repetitions was recorded individually as an interval session. The intensity of each exercise session was recorded as easy, moderate, hard, or very hard. Easy referred to warm-up and warm-down activity, moderate was 50-60% of perceived maximum effort, hard was 60-80% of perceived maximum effort, and very hard was race pace and/or 80-100% of perceived maximum effort. The recording of the interval training sessions required the number of repetitions, the distance covered in the repetition, the time to complete the repetition and the rest time between repetitions. The continuous training recall required a record of the time to complete the swim, ride, or run, the distance covered in that time and/or the average speed of the session. This type of training diary and retrospective questionnaire has been shown to be a valid and reliable method of assessing the training practises of competitive athletes (Hewson and Hopkins, 1995; Llow and Hopkins, 1996).

Retrospective Questionnaire

Within three days of the competition the triathletes came to the laboratory to complete a retrospective questionnaire on the training in the 4 weeks before they received the diary. With the help of the same researcher, the athletes recorded their hours of training in each exercise mode per week and the intensity of the training. The athletes were asked to record in more detail their training in the first week and in the hardest training week during this 4-week period. The data from their hardest training week was used for comparisons with the data from the diary.

Statistical analyses

The Statistical Analysis System (SAS Institute, Cary NC) was used for all statistical analyses. Proc mixed was used to perform linear modelling, with repeated measures and an appropriate covariance structure, when necessary. Correlation matrices were used to compare training in the different modes and training at the various intensities. Parametric tests were performed on all data except where the means were similar to the standard deviations and where the standard deviations were different between groups. These data were examined with a one-way non-parametric procedure for analysis of variance. Repeated-measures ANOVA were used to compare the training in the diary to that in the questionnaire. For comparisons of training performed in the first and last 5-d periods between seniors and juniors and between males and females, training times were log transformed.

Means and standard deviations were used to represent the average and typical spread of values of variables. We have shown the precision of our estimates of outcome statistics as 95% confidence limits. The P values shown represent the probabilities of an extreme absolute value than the observed value of the effect if the true value of the effect was zero or null.

Statistically significant effects are those for which the zero or null value of the effect lies outside the 95% confidence interval ($P \leq 0.05$).

Results

Table 4.1 summarises the characteristics and athletic abilities of the triathletes. The personal best 1.5-km swimming times were similar in all the groups. The personal best 10-km running times and total triathlon times of the senior male triathletes were faster than those of the other groups ($P = 0.001$). The mean training per day performed during the second week (hardest training week) of the month over which the questionnaire was completed was 109 ± 39 (range 29 – 180) min (Fig. 4.1). 27% (29 ± 24 min) of this time was spent at an easy intensity, 31% at a moderate intensity (34 ± 34 min), 28% at a hard intensity (30 ± 25 min) and 14% at a very hard intensity (15 ± 16 min).

Table 4.1. Characteristics of the triathletes

	Junior females	Junior males	Senior Females	Senior Males
Sample size	4	4	3	5
Age (y)	17 ± 2	17 ± 1	30 ± 6	26 ± 2
Sporting experience (y)	5.5 ± 1.0	8.0 ± 1.0	6.5 ± 4.0	13.0 ± 4.0
Triathlon experience (y)	1.5 ± 1.0	3.0 ± 1.0	3.0 ± 1.0	4.0 ± 1.0
Personal-best times[a]	140.5 ± 0.2*	128.4 ± 0.1*	131.4 ± 0.2*	119.4 ± 0.5
1.5-km swim (min)	20.3 ± 1.0	19.5 ± 4.0	19.0 ± 1.0	20.0 ± 1.0
40-km cycle (min)	73.2 ± 11.2	63.0 ± 3.3	60.1 ± 1.1	59.4 ± 1.0
10-km run (min)	40.5 ± 1.4*	37.5 ± 3.2*	38.3 ± 1.4*	32.3 ± 1.5

Data are mean ± SD.

[a]In competitive triathlons performed in the previous 12 months. *Indicates significantly faster 10-km running times and total triathlon times of the senior male triathletes than those of the other groups ($P \leq 0.001$).

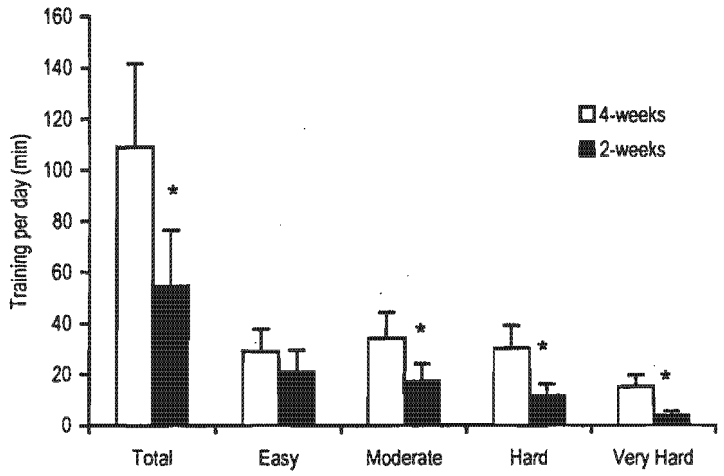


Figure 4.1. Total mean training per day 4 weeks (training reported in the questionnaire) and 2 weeks (training reported in the diary) before the competition. * Indicates significant differences between the training performed a month before the competition and two weeks before the competition. Values are means ± S.D.

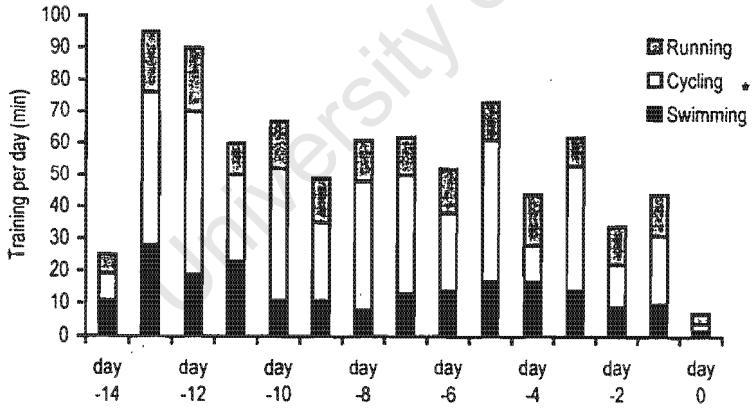


Figure 4.2. Total daily training in the 2-weeks before the competition. Day 0 is the day of the competition.

* Indicates significantly greater cycling training than swimming and running training during the 2-week period.

During the 2-week period during which diaries were completed, the total mean training per day was 55 ± 24 (range 25 – 110) min (Fig. 4.1). Thirty-nine percent (21 ± 21 min) of the training time was spent at an easy intensity, 32% (17 ± 12 min) at a moderate intensity, 21% (11 ± 10 min) at a hard intensity and 7% (4 ± 3 min) at a very hard intensity. Training at the very hard intensity was significantly less than training performed at the other intensities (P = 0.01). The mean swimming training per day performed during the two weeks before the competition, was 14 ± 9 (range 5 – 43) min, 29 ± 18 (range 6 – 73) min cycling and 13 ± 5 (range 6 – 23) min running (Fig. 4.2). Cycling training was significantly greater than both swimming (86%, 95% CI = 78% to 94%) and running training (85%, 79% to 92%), P=0.002. The triathletes that cycled more than the others also ran more than the others (r = 0.73; 95% CI = 0.37 to 0.90) but there was little or no relationship between swimming and either cycling (r = 0.21; -0.32 to 0.64) or running (r = 0.03; -0.47 to 0.52).

Training performed in the 2-week period before the competition was reduced to a mean of 48% (39 to 60%, P = 0.0001) of the training reported in the questionnaire. Total training was reduced at all intensities. At the easy and moderate intensities training was reduced to a mean of 60% (38 to 97%, P= 0.04) and 42% (28 to 62%, P = 0.0003), while training at the hard and very hard intensities was reduced to a mean of 38% (23 to 61%, P= 0.0006) and 26% (17 to 42%, P= 0.0001) of the training in the questionnaire, respectively (Fig. 4.1).

Figure 4.3 (*upper panel*) compares the total mean training per day (min) over the two weeks before the competition of the junior and senior triathletes. The junior triathletes swam more than the seniors (16 ± 12 min vs. 12 ± 7 min) in training but cycled less (18 ± 9 vs. 37 ± 19 min, P= 0.028) and ran less (9 ± 3 min vs. 15 ± 5 min, P= 0.048).

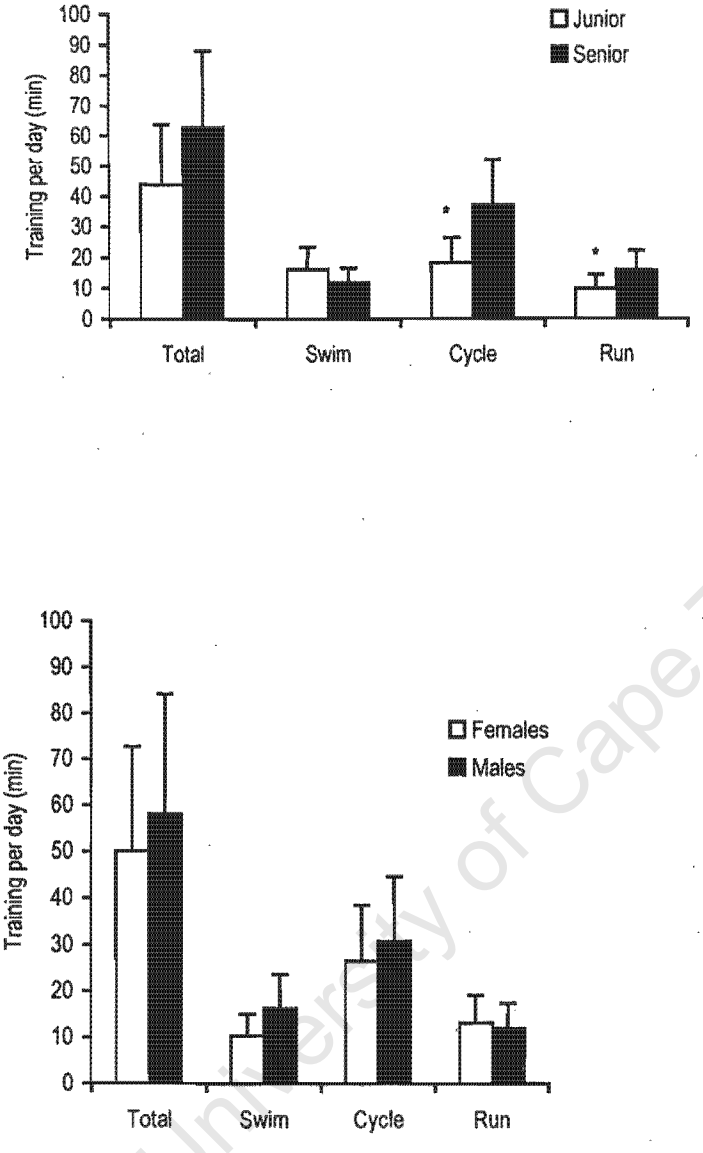


Figure 4.3. Mean training durations per day (min) of the junior and senior triathletes (*upper panel*) and of the male and female triathletes (*lower panel*) 2-weeks before the competition. * Indicates significantly different cycling and running training between the junior and senior triathletes.

In contrast, there were no significant differences between the female and male triathletes' mean swimming, cycling and running training per day (min) (Fig. 4.3, *lower panel*). Both the female and the male triathletes spent roughly twice the amount of time cycling than running or swimming during training (Fig. 4.3, *lower panel*).

The total training performed during the first and last five-day periods of the two weeks before the competition is shown in Fig. 4.4 (*upper panel*). Training performed during the last five-day period was reduced at all the intensities to a mean of 63% (45 to 87%, P=0.009) of the first five-day period. In the last five days the juniors reduced their training to a mean of 72% (45 to 116%) of the first five-day period, the seniors to 56% (36 to 87%, P =0.01), the females to 59% (36 to 95%, P= 0.03) and the males to 67% (43 to 105%, P= 0.07). The seniors' training at the easy and moderate intensities did not change significantly, but training at the hard and very hard intensities decreased to a mean of 31% (10 to 91%, P= 0.03) and 37% (10 to 137%, P=0.11), respectively (Fig. 4.4, *lower panel*).

The correlation between the mean total training time per day over the two-week period to the competition and personal best performance in an Olympic-distance triathlon was not significant (r =0.18; Fig. 4.5, *upper panel*). The correlation between the reduction in training time in the two-week period and personal best performance in an Olympic-distance triathlon was slightly higher but not significant (r =0.40) in this group of triathletes (Fig. 4.5, *lower panel*).

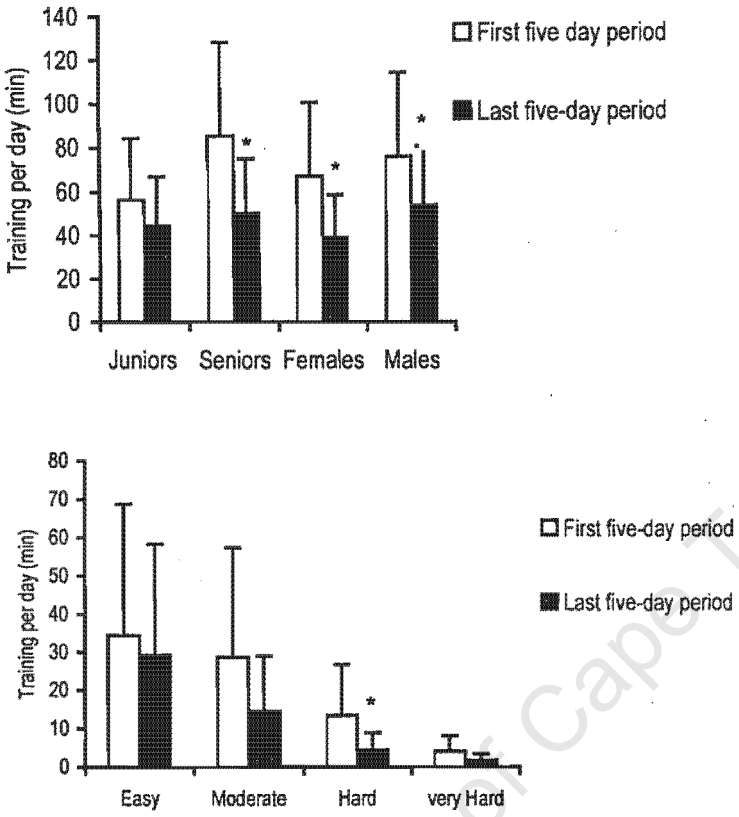


Figure 4.4. Mean training durations per day (min) in the first and last 5-d periods for all the sub-groups (*upper panel*) and a breakdown of the senior's training in the same period (*lower panel*). * Indicates a significant difference in the senior's and females' total training durations in the first and last 5-d period and the senior's training at the hard intensity between the time periods.

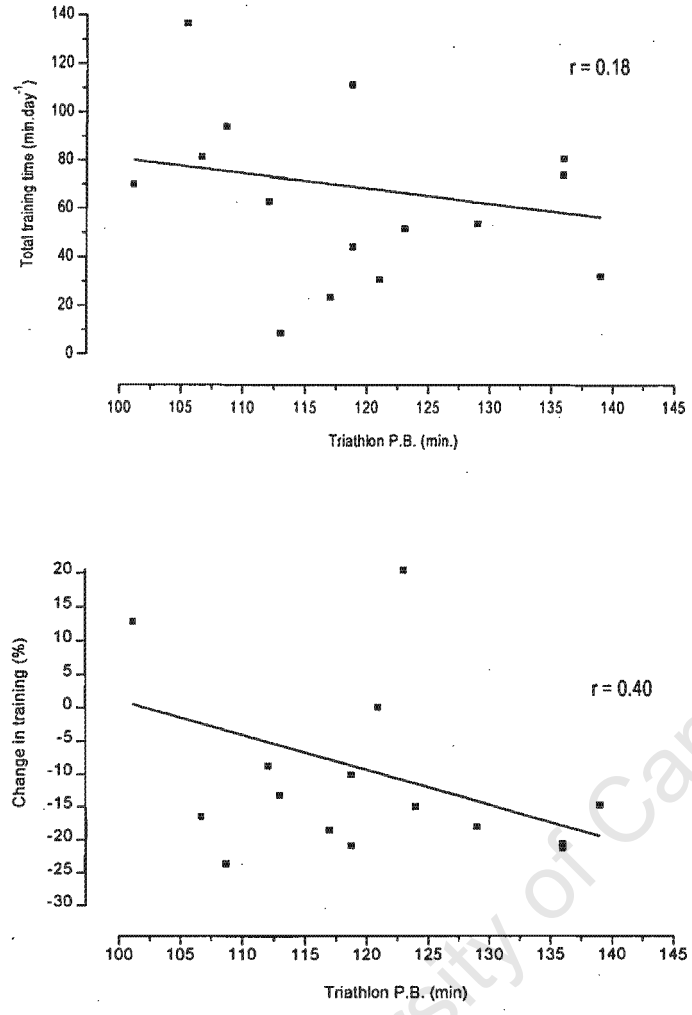


Figure 4.5. Relationship between the mean total training time per day (min) two weeks prior to the competition (*upper panel*), reduction in training time in the two-week period (*lower panel*) and personal best performance in an Olympic-distance triathlon.

Discussion

The main finding of this study was that all the triathletes regardless of gender and racing experience tapered their training before the competition. Furthermore, there was a 52% difference between the training reported in the questionnaire and that recorded in the diary. Most of the triathletes kept a training log from which they extracted the information for the questionnaire. The 52% difference in training time is therefore a valid training reduction or taper in this group of triathletes. The senior triathletes had been competing and training for triathlons longer than their junior counterparts, and tapered their training in the weeks leading up to the competition more than the other triathletes. There was however, no significant relationship between the reduction in training during this period and their best performance in an Olympic-distance triathlon (r =0.40). Cycling training, in particular, was "tapered" (reduced) more than the swimming and running training.

Gilman and Wells 1993, reported that women runners performed most of their training at an easy or moderate intensity, rather than at a hard intensity. Robinson et al. (1991) also found that male runners who competed in marathons had more training at an easy intensity than those who competed in shorter distance races. The reported training intensities of these single-sport athletes are similar to those reported by our triathletes.

In an Olympic-distance triathlon roughly 50% of the event is spent on the bike, 30% running and 20% swimming (McNaughton 1989). The triathletes in this study appear to train in proportion to the relative durations of the three type of exercise in their event.

A comparison of the training durations of the seniors and the juniors showed significant differences (Fig. 4.3 and 4.4, *upper panels*). The senior triathletes spent more time cycling and

running than the juniors. However, the juniors spent more time swimming and were as fast as the seniors in the swimming part of the triathlon (Fig. 4.3, *upper panel*).

Although there were no gender differences in the training durations of the triathletes in the three exercise modes (Fig. 4.3, *lower panel*), the males trained for longer than their female counterparts in swimming and cycling, but the females spent more time running. A possible explanation is that the female triathletes trained at a lower intensity and therefore took longer to complete their training runs and the running segment of the triathlon. Moreover, the female triathletes took longer to complete the triathlon compared to the male triathletes (Table 4.1). The mean training time per day performed in the three exercise modes for the female triathletes in this study were similar to those reported by Laurenson et al. (1993) and Leake et al. (1991) for elite and club level female triathletes. The female triathletes in the present study also had similar performance times for the Olympic-distance triathlon compared to the highly trained female triathletes studied by Schneider and Pollack (1991) and O'Toole et al. (1987). These researchers concluded that the time spent training per day is not a good determinant of success in triathlon but rather the quality of training undertaken. Therefore, the variations in triathlon performance times among the sub-groups (males vs. females and juniors vs. seniors) might be partially accounted for by the variations in training pace among these sub-groups. O'Toole (1989) speculated that adequate amounts of training are not a guarantee of success in triathlon but rather swimming skill, cycling efficiency and running economy.

A training variable that is increasingly being manipulated is the taper format. Zarkadas et al. (1995) and more recently, Banister et al. (1999) in their simulated tapering study on triathlon athletes reported that an exponential taper (γ =5 days) group made a significantly greater improvement above a pre-taper standard ($P \leq 0.05$) than the step reduction taper group in

cycle ergometry. Additionally, a fast exponential taper group (γ = 5 days) performed significantly better ($P \leq 0.05$) in maximal, cycle ergometry above a pre-taper training standard than a slow exponential taper group (γ = 8 days). In our study, the triathletes followed a progressive reduction in their training (Fig. 4.2) and on interviewing the triathletes there were no indications of an exponential or stepwise ramp method in the reduction of their training. Whether there is a purpose for these triathletes and other competitive athletes to reduce their training in this fashion remains to be investigated.

Tapering is designed to reduce the possible physiological and psychological stresses of daily training but the parameters defining the benefits of tapering from the negative effects of detraining are unclear (Houmard et al. 1989; Johns et al. 1992; Neary et al. 1992). The present study showed that these triathletes reduced their training by ~50% over the two weeks before the competition. While this is consistent with the available literature on reductions in training volume during taper (Johns et al. 1992; Zarkadas et al. 1995; Banister et al. 1999) as a means of improving performance, it seems reasonable to assume that the more training one performs the more one should taper. Proposed mechanisms on how tapering optimises performance remain speculative and further work in this field is required (Houmard et al. 1989, 1994; Kohrt et al. 1987,1989; Schneider et al. 1990; Martin et al. 1994; O'Toole 1995; Mujika et al. 1996a; Slievert and Rowlands 1996). In conclusion, the guidelines and recommendations from the findings of the studies and reviews on tapering (Neufer 1989; Costill et al.1991; Houmard 1991; Neary et al. 1992; Houmard and Johns 1994; Mujika 1998), suggest that training at the high intensities should be maintained or increased during the training period prior to a competition, surprisingly, the triathletes in our study reduced the training time spent at the hard and very hard intensities in the training period immediately preceding the competition. It remains to be established whether their taper regime was optimum for improving performance.

CHAPTER 5

THE EFFECTS OF INTERVAL TRAINING AND 2-WEEK TAPER ON 100-KM TIME TRIAL PERFORMANCE IN WELL-TRAINED CYCLISTS

RATIONALE AND AIM

While the adaptations to training have been studied extensively in previously sedentary individuals, far less is known about the responses to high-intensity interval training (HIT) and reductions in HIT prior to competition in highly trained athletes. A number of investigators have systematically studied the reported benefits of HIT on endurance exercise performance (Lindsay et al. 1996; Westgarth-Taylor et al. 1997; Hawley et al. 1997; Stepto et al. 1999) while more recently, Stepto et al. (2001) have investigated the metabolic demands of intense aerobic interval training in competitive cyclists. These studies have found that ≥ 6 HIT sessions, consisting of 6-9 x 4-5 min cycle rides at 80-85% of peak sustained work rate (W_{peak}), maximally improved simulated, 40 km time-trial (TT_{40}) performances. Improvements in the ability of athletes to sustain higher (76% vs. 72% of W_{peak}) relative work rates in TT_{40} rides after HIT at 80-85% of W_{peak} supported the idea that athletes should train for competition at exercise intensities slightly above those in their event (Hawley et al. 1997). The hypothesis of the present study, was to test whether 6 HIT sessions performed at a slightly higher (70% vs. ~63%) exercise intensity than previous 100 km time-trial (TT_{100}) performances of well-trained cyclists in our laboratories (Schabort et al. 1998), would significantly improve TT_{100} performance in line with the specificity of training exhibited in earlier studies (Lindsay et al. 1996; Westgarth-Taylor et al. 1997; Stepto et al. 1999). A second hypothesis was to examine whether heart rate recoveries between the high-intensity intervals would be faster in the better cyclists.

Following the HIT program, the effects of a 50% reduction or 'taper' in the frequency and/or volume of HIT over 1 and 2 weeks was investigated to see how a recovery from exhaustive training might influence TT_{100} performances. Previously, it was found that elite triathletes 'tapered' their training by 50% in the 2 weeks before a national competition (Chapter 4). This component of the study was designed to examine whether a 50% reduction in HIT volume over 2 weeks achieved the optimum theoretical balance between recovery from fatigue and loss of fitness (Zakardas et al. 1994, 1995; Morton et al. 1997; Banister et al. 1999). While many endurance athletes 'taper' or reduce their training in the week(s) before a competition (Neufer 1989; Houmard 1991; Houmard and Johns 1994; Mujika 1998), only Hickson et al. (1982, 1985) have studied the effects of reduced training on endurance performances. They showed that a 66% reduction in training duration and 33% and 66% decreases in training intensity over 15 weeks decreased ~ 2 h cycling endurance by ~ 10%, 20% and 30%, respectively (Tables 2.8 and 2.9). Other studies of the potential benefits of more realistic (< 4 weeks) reductions in training volume have all focused on performances in short events such as < 200 m swims and 800-5000 m runs and the results have been inconsistent (Table 2.9). A second aim of the present study therefore was to examine the time-course of the effects of tapering (≤ 2 weeks) on endurance performances in an event lasting longer than 30 min.

Methods

Sixteen, male, competitive, endurance cyclists participated in this study (Table 5.1). All the cyclists were training between 200 and 400 $km.wk^{-1}$ and competing in races on most weekends. The study was approved by the Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town and each cyclist completed a written informed consent form to participate.

Simulated 100 km time-trial (TT_{100}) cycling performances

All the cyclists performed three simulated TT_{100} rides, with the first TT_{100} ride being treated as a familiarisation ride, before any training intervention took place. The cyclists were then randomly assigned to an experimental group (n = 8) which then performed six sessions of supervised, sustained (3 x 15 min) high intensity (70% of W_{peak}) interval training (HIT) in three weeks. In the following week and two weeks the HIT training frequency and/or volume was reduced or tapered by 50% while the intensity of the sessions was maintained and performed a further three TT_{100} rides. Cyclists in the control group (n = 8) continued with their own unsupervised endurance training. In the following two weeks, the control group did not taper and also performed three TT_{100} rides.

Simulated TT_{100} rides were performed on a Kingcycle™ ergometry system (Kingcycle™ Ltd, High Wycombe, U.K.) as described by Palmer et al. (1996). Before each TT_{100} ride, the subject's racing bicycle was attached to the ergometer by the front fork and supported by an adjustable pillar under the bottom bracket. The pillar under the bottom bracket was then adjusted to set the rolling resistance of the rear bicycle wheel on an air-braked flywheel to match that of a 65-kg cyclist riding on level terrain. Rolling resistance was set by asking the cyclists to accelerate to a workload of ~200 W and immediately stop pedaling while remaining seated on the saddle. During these run-down calibrations, the bottom bracket support was adjusted until an IBM-compatible computer showed that the slowing of the flywheel matched a reference power decay curve (Palmer et al. 1996). Instantaneous power outputs (W) were computed from photo-optic measurements of the speed of the flywheel in revolutions per second (RPS) using the following equation:

$$W = 0.000136\ RPS^3 + 1.09\ RPS$$

After the calibration, the cyclists were provided with a Sport tester heart rate monitor (Polar Electro, Kempele, Finland) and began a standardized 5-min 'warm up' before the TT_{100} ride. To simulate the variable power demands of road cycle racing (Palmer et al. 1994), TT_{100} rides included 1-km sprints after 10, 32, 52, 72 and 99 km and 4-km sprints after 20, 40, 60 and 80 km (Palmer et al. 1996). Cyclists were informed when to sprint by computer display of their location on the 'course profile' (Fig. 5.6) and by the investigator's encouragement to prepare to cycle as 'fast' as possible. The principal investigator was present at all the testing sessions and for the duration of each ride to ensure that the subjects did not lose focus and reduce power outputs during the rides. Only elapsed distance and heart rate were given to cyclists during the TT_{100} rides. They were not informed of their continuously recorded power outputs, their TT_{100} performances or their 1 and 4 km sprint speeds until the end of the study.

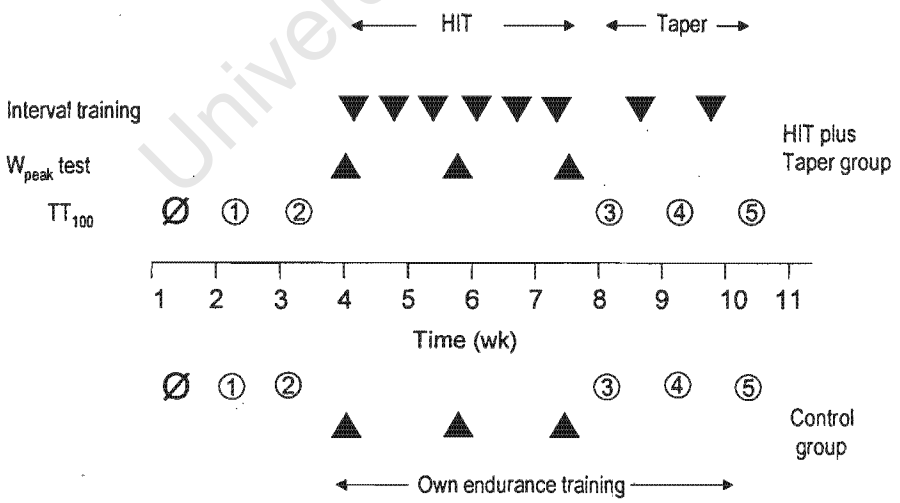


Figure 5.1. Schedule of testing during the study. Interval training was undertaken only by the HIT plus taper group. Ø Indicates the familiarization TT_{100} ride.

Previously, Schabort et al. (1998) showed that individual cyclist's (n = 8) coefficients of variation in three TT_{100} performances were 1.7% with a 95% confidence interval of 1.1% to 2.5%. Corresponding coefficients of variation in mean 1 and 4 km sprint speeds were 1.9% and 2.0%, respectively.

In the ≥ 24-h before the first, familiarization TT_{100} and during that ride, the cyclists were allowed to eat and drink ad libitum. Records of their intake of food and fluid were then given to the cyclists with a request that they consume the same diet before and during all subsequent TT_{100} performance rides. At that time, the cyclists were also given a training diary and asked to continue with their habitual training but to refrain from strenuous exercise on the day before a TT_{100} ride. All laboratory TT_{100} rides were performed at the same time of day in a constant ambient temperature of ~20°C and a relative humidity of ~55%.

Within a week of the first, familiarization TT_{100} ride (n = 16), each cyclist completed two more TT_{100} rides, separated by 4-7 days (Figure 5.1). These rides were designed to ensure that the cyclists' athletic performances were consistent (Neary et al. 1999) before any intervention.

Maximal incremental exercise test

Following the three baseline TT_{100} rides, the cyclists (n = 16) performed a progressive incremental test to exhaustion on an electronically braked cycle ergometer (Lode, Groningen, The Netherlands), as described in Chapter 3, for a determination of their peak power output (W_{peak}) and heart rate (HR_{peak}). This information was used to set the work rates in the HIT sessions to 70% of the cyclist's W_{peak}.

High-intensity interval training (HIT)

HIT took place after a self-paced standardized warm-up on 6 occasions over 3-4 wk. Each HIT session consisted of 3-4 x 15-min rides at 70% of W_{peak}, or 88-92% of HR_{peak}, separated by 5 min of active recovery (Fig. 6.11). Since the cyclists (n = 8) may have varied in their ability to tolerate HIT, no attempt was made to control their ~ 20% replacement of endurance training with HIT. The cyclists were only asked not to perform any HIT outside the laboratory. After three HIT sessions in ~12 days, the cyclists performed a further W_{peak} test and the intensity of the subsequent three HIT sessions was adjusted to increases in their W_{peak} values.

Post-HIT Taper

Within 4-days of the 6th HIT session, the cyclists from the experimental group (n = 8) performed a further W_{peak} test and a TT_{100} ride on separate days. They then reduced the frequency of their HIT by 50% from twice to once a week for two weeks and repeated the TT_{100} rides, 3 days after each HIT session. The 50% reduction in training frequency resulted in a 50% reduction in training volume during the week. Cyclists in the control group also performed a W_{peak} test and three TT_{100} rides after 3-4 weeks of continued endurance training.

Statistical analyses

All results are presented as means ± standard deviations (SD). Between and within group differences in performances over time were assessed with two- and one-way analyses of variance for repeated measures, followed by Scheffes post hoc tests (Statistica, StatSoft Incorporated, Tulsa, U.S.A.). Single, within group, differences were examined with a paired Student's t-test. A Pearson product moment correlation coefficient (r) was used to examine relationships between selected variables. A value of $P \leq 0.05$ was regarded as statistically significant.

Results

The physiological characteristics and base-line performances of the cyclists are shown in Table 5.1. All the cyclists had W_{peak} values of ~ 350-450 W and were training ~ 200 km/wk.

Table 5.1. Physiological characteristics and base-line performances of the cyclists.

	Control group	HIT plus taper group
(n)	(8)	(8)
Age (y)	23.1 ± 5.5	21.1 ± 3.9
Weight (kg)	65.8 ± 4.3	66.9 ± 6.3
Fat (%)	13.2 ± 2.9	12.2 ± 4.3
HR_{peak} (beats.min^{-1})	193 ± 6	194 ± 6
W_{peak} (W)	368 ± 44	390 ± 29
Training distance (km/week)	277 ± 95	202 ± 60

Peak heart rates (HR_{peak}) and sustained power outputs (W_{peak}) were measured during a maximal, incremental exercise test. All values are means ± SD.

Figure 5.2 (*upper panel*) shows the declines in heart rate (HR) during the 1st min of the 5-min recovery between the 2nd and 3rd 15-min intervals of the 8 HIT sessions. During the six HIT sessions before the taper, the average decline in HR was 30± 7 b.min^{-1} (~175 to 145 b.min^{-1}). Although not significant, 1- and 2-week after a 50% reduction in the frequency of HIT, the same HR recoveries improved progressively from 30±7 to 35 ± 8 and 40 ± 8 b.min^{-1}. In addition, the improved heart rate recoveries during the taper HIT sessions were associated with the large improvements in TT_{100} cycling speed after training and taper (r = -0.54, Fig. 5.2 *middle panel*). Similarly, the improved ability to recover from the 15-min training intervals during the Taper 2 HIT session was insignificantly related to the cyclists' performances during TT_{taper2} (r = 0.27, Fig. 5.2 *lower panel*).

Six HIT sessions at 70% of W_{peak} also had no immediate effect on the HIT plus taper group's TT_{100} performances (Fig. 5.3). TT_{100} performances only improved significantly when the frequency of HIT was subsequently reduced to 50% for 1 and 2 weeks (Fig. 5.3). One and two weeks after a 50% reduction in the frequency of HIT, TT_{100} average cycling speeds were increased from 38.1 ± 3.0 km/h pre-HIT to 40.2 ± 2.7 (Taper 1) and 41.1 ± 2.6 km/h (Taper 2) ($P \leq 0.05$). The HIT plus taper group took a mean of 158:22 ± 11:17 min:s to complete TT_{pre1}, 149:43 ± 10:07 min:s for TT_{taper1} and 146:37 ± 9:17 min:s for TT_{taper2} ($P \leq 0.05$). In contrast, there were no significant changes in the performances of the eight control cyclists, who continued their endurance training and repeated six TT_{100} rides at equivalent intervals (Fig. 5.3). The control cyclists' constant performances and their individual coefficients of variation (in parenthesis) in mean 1-km and 4-km sprint speeds and in overall TT_{100} cycling speeds (Fig. 5.3) were 42.9 ± 2.7 km/h (7.4 ± 1.3%), 39.6 ± 2.8 km/h (5.8 ± 2.8%) and 37.4 ± 2.5 km/h (3.1 ± 1.3%).

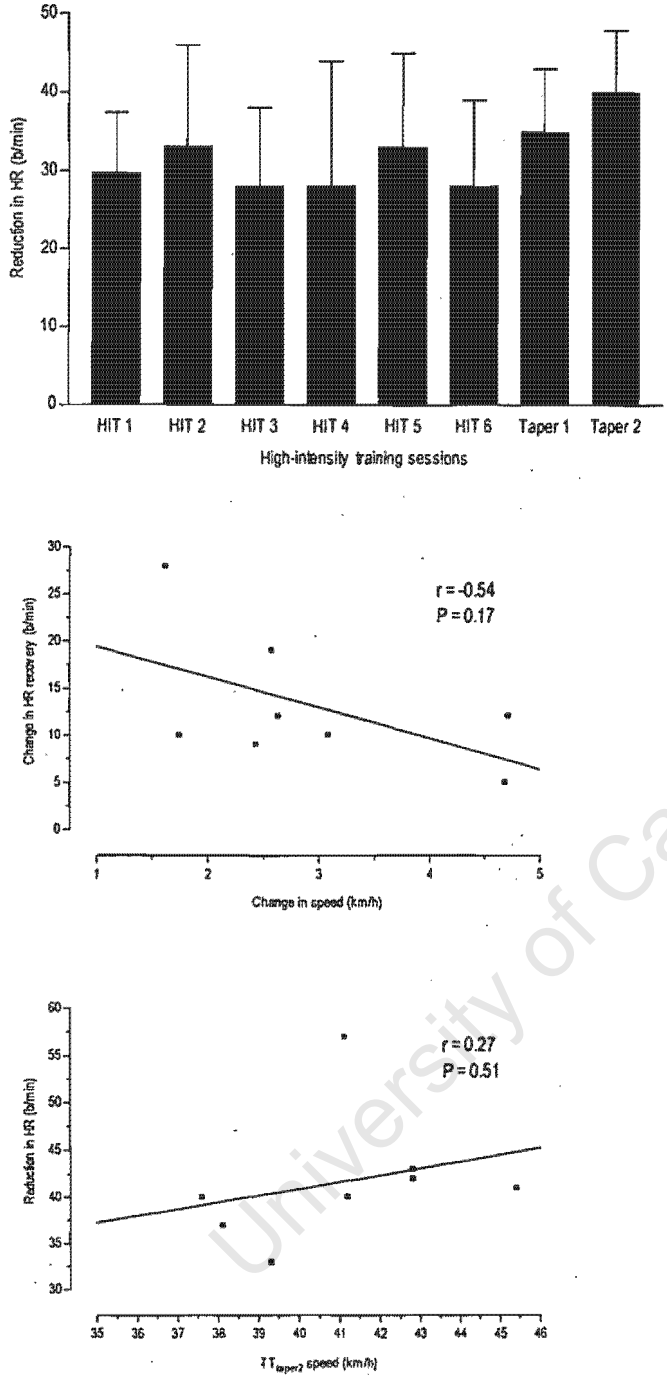


Figure 5.2. Mean decline in heart rate ($b.min^{-1}$) in the first minute of the 5-min recovery between the 2nd and 3rd 15-min interval cycling repetition during the 8 HIT sessions (*upper panel*). The middle panel shows the relationship between the reductions in heart rate recovery (HIT Taper 2 – HIT 1) and the improvements in TT_{100} speed (TT_{taper2} – TT_{pre1}). The lower panel shows the relationship between the reductions in heart rate during HIT Taper 2 and the cycling performances during TT_{taper2}.

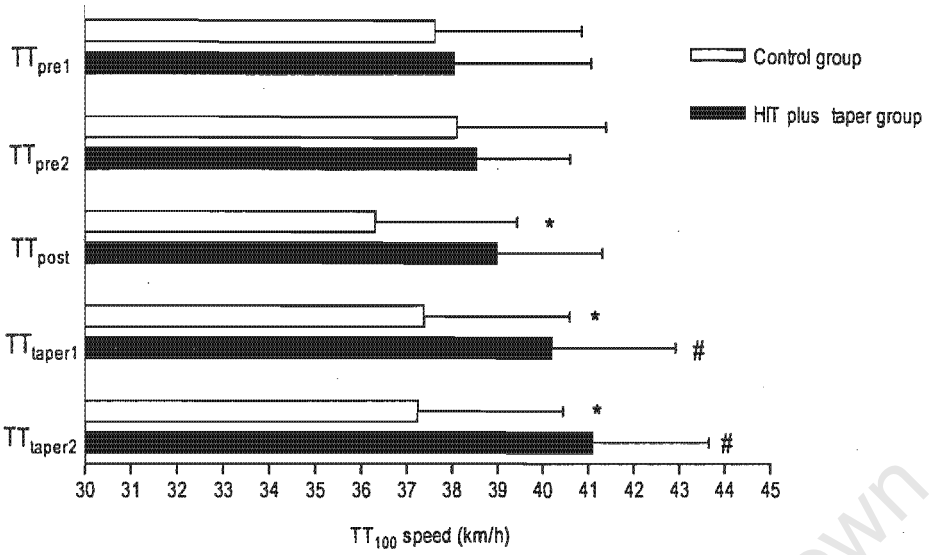


Figure 5.3. TT_{100} performances of the control and HIT plus taper groups. * Indicates significant differences between the control and HIT plus taper group of $P \leq 0.001$ and # indicates $P \leq 0.05$ difference compared to the HIT plus taper group's TT_{pre1}.

Improvements in TT_{100} performances after 1 and 2 weeks of a 50% reduction in HIT frequency were not due to any significant increases in the mean 5 x 1-km and 4 x 4-km sprint speeds in the TT_{100} rides (Table 5.2, Fig. 5.4). During the 2-wk taper, mean 1-km and 4-km sprint speeds rose only from 43.5 ± 2.5 to 45.6 ± 2.1 km/h and from 40.2 ± 2.3 to 41.6 ± 3.1 km/h, respectively (Table 5.2). Greater (~ 2.1 vs. 1.4 km/h) improvements in mean 1-km sprint speeds than in mean 4-km sprint speeds were largely due to significant improvements in the speeds of the most rapid, 1st and 5th 1-km sprints (Table 5.2). Between those 1-km sprints, the cyclists' 'pacing' strategy was similar to that before HIT plus a 2-week taper (Fig. 5.4).

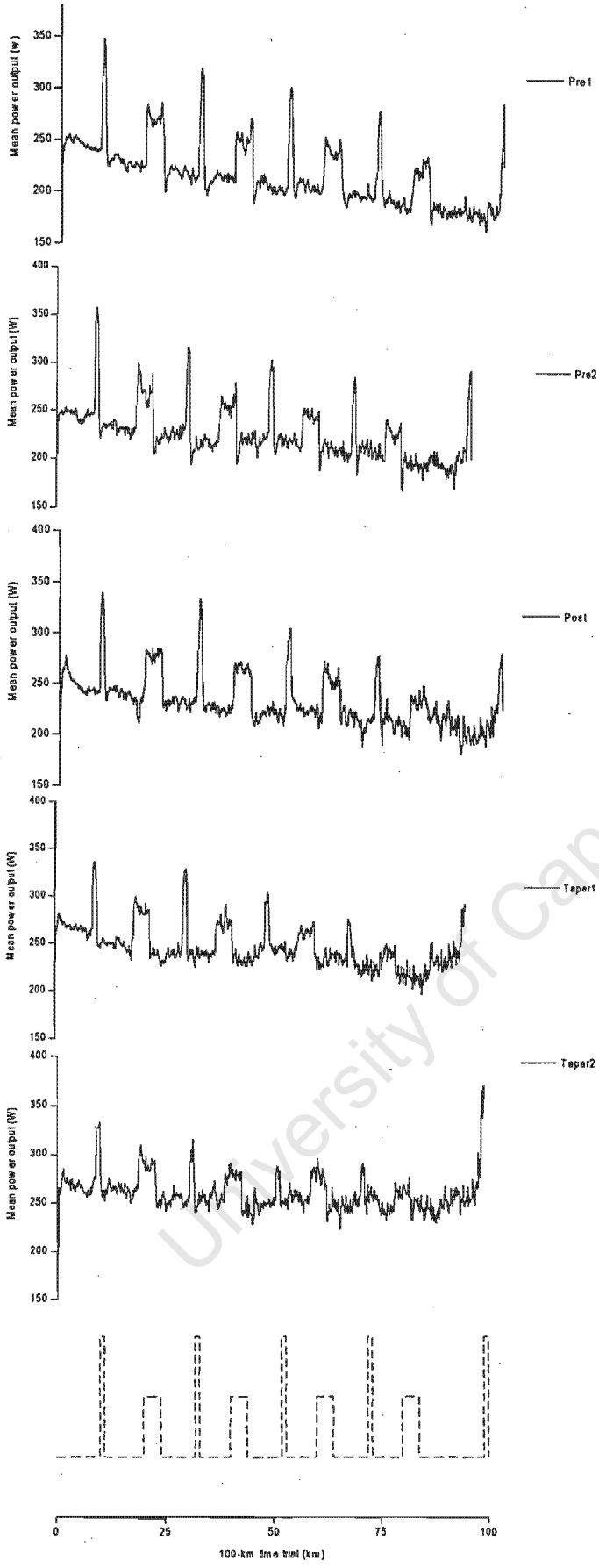


Figure 5.4. Mean power output profile of the five TT_{100} rides before HIT (TT_{pre1}, TT_{pre2}), 3 days after HIT (TT_{post}) and after a 1- and 2-wk taper (TT_{taper1}, TT_{taper2}) and a "course profile" of the 100-km time trial.

Table 5.2. Speed and mean power output of the HIT plus taper (n = 8) group's 1-km and 4-km sprints during TT_{pre1} and TT_{taper2} and the percent improvements during the sprints (TT_{pre1} vs. TT_{taper2}).

Trial	First sprint		Second sprint		Third sprint		Fourth sprint		Fifth sprint	
	Speed (km/h)	Power (W)	Speed (km/h)	Power (W)	Speed (km/h)	Power (W)	Speed (km/h)	Power (W)	Speed (km/h)	Power (W)
TT_{pre1}										
1 km	44.4 ± 6.0	317 ± 43	45.0 ± 2.8	325 ± 71	42.4 ± 3.0	284 ± 20	41.9 ± 3.4	276 ± 23	43.9 ± 3.7	308 ± 26
4 km	41.1 ± 3.4	265 ± 22	40.9 ± 2.9	279± 23	39.4 ± 3.0	236± 19	39.5 ± 2.4	238± 14		
TT_{taper2}										
1 km	48.7 ± 2.6*	383 ± 21	45.6 ± 4.0	334 ± 30	42.9± 5.1	292± 35	43.4± 3.1	300 ± 72	47.4± 1.9	363 ± 14
4 km	43.0 ± 3.2	294 ± 22	42.4 ± 3.7	284 ± 25	40.5 ± 5.3	254 ± 34	40.6 ± 3.9	256 ± 25		
~ % change pre vs. taper										
1 km	8.8*		1.3		1.2		3.5		7.4*	
4 km	4.4		3.5		2.7		2.7			

Values are mean ± SD for TT_{pre1} and TT_{taper2} (n = 8). * Indicates significantly faster 1st 1-km sprint vs. the 3rd and 4th 1-km sprint speed during the TT_{taper2} and significant improvements in sprint speeds during the 1st and 5th sprints (TT_{pre1} vs. TT_{taper2}), $P \leq 0.05$.

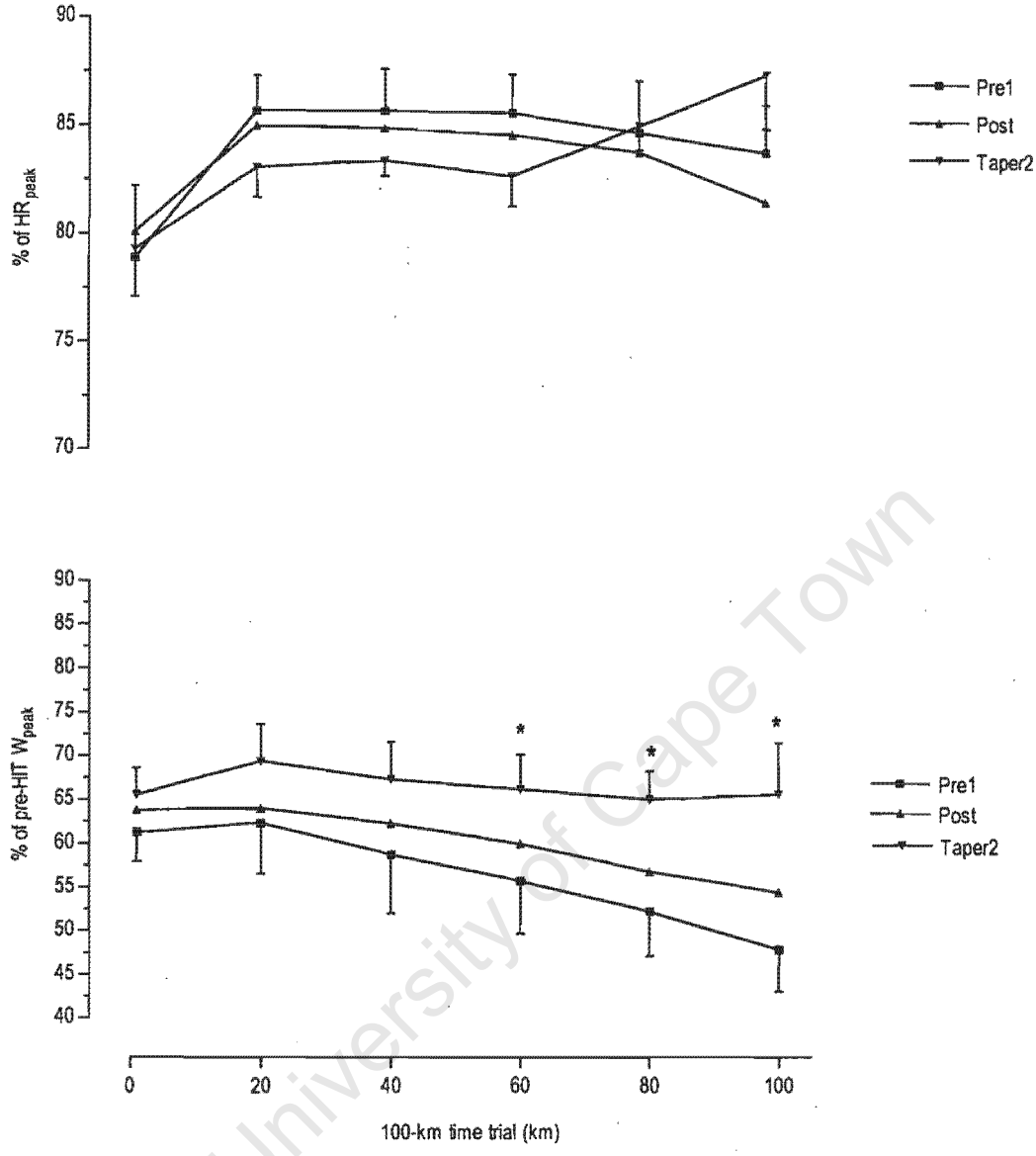


Figure 5.5. Mean percent of peak heart rate and mean power output of the TT_{pre1}, TT_{post} and TT_{taper2} rides during the 1st 1-km and then at 20-km intervals. *indicates significant differences in power output between the last 60-km of the TT_{pre1} and TT_{taper2} time trials, $P \leq 0.05$.

Faster TT_{100} cycling speeds after HIT plus a 2-week taper were also not associated with any significant differences in heart rates (Fig. 5.5, upper panel). Interestingly, the 1st 60 km of TT_{taper2} was performed at a slightly lower percent of HR_{peak} than before training and taper while over the last 40-km the cyclists were able to increase their heart rate by ~5% as opposed to the ~3% decline before training and taper.

In contrast, improvements in TT_{100} performances after 2 weeks of a 50% reduction in HIT frequency were due to the cyclists being able to sustain significantly higher absolute and relative work rates during the last 40-60 km of the TT_{100} rides (Fig. 5.5, lower panel). HIT plus a 2-week taper increased the mean absolute power outputs during the TT_{100} rides from 228 ± 32 to 258 ± 41 W and the relative sustained power outputs from 58 ± 6% to 66 ± 7% of pre-HIT W_{peak} values (both $P \leq 0.001$). Although the pre-trial W_{peak} values of the 16 cyclists in this study were related to their TT_{100} cycling speeds ($r = 0.75$, $P \leq 0.001$, Fig. 5.6), there was no correlation between the cyclists' modest (395 ± 32 vs. 390 ± 29 W) improvements in W_{peak} values and their ~2.3 km/h faster TT_{100} cycling speeds after HIT plus a 2-week taper ($r = -0.102$, data not shown). Instead, faster TT_{100} cycling speeds after HIT plus a 2-week taper were more closely related to the cyclists' ability to sustain higher % of W_{peak} ,%HR_{peak} and changes in relative work rates($r = 0.94$; 0.74 and 0.50, Fig.5.7 upper, middle and lower panels).

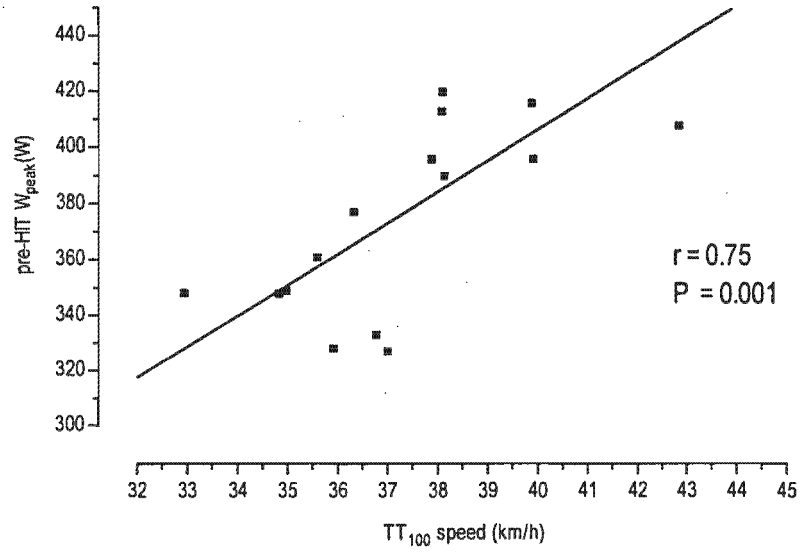


Figure 5.6. The relationship between the pre-HIT W_{peak} values and TT_{100} performances of all 16 cyclists.

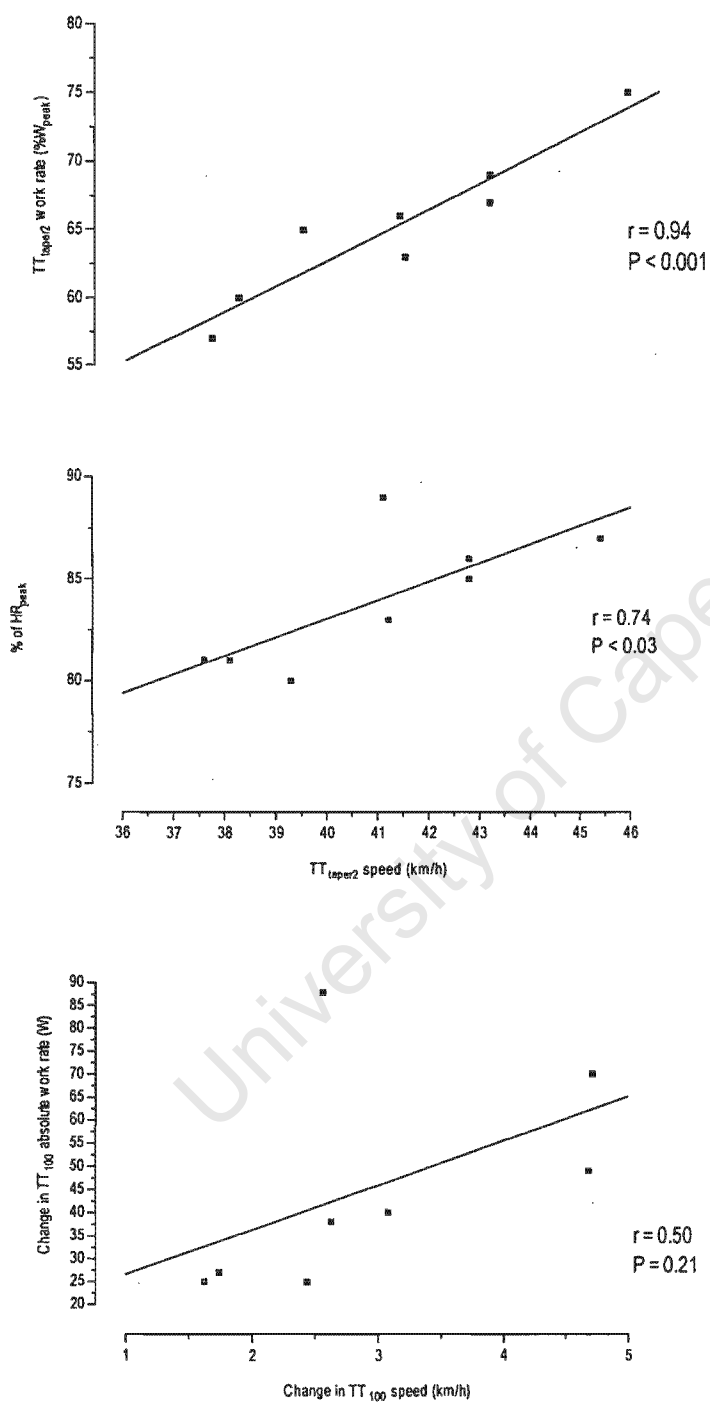


Figure 5.7. The relationship between $\%W_{peak}$ and $\%HR_{peak}$ and the performances during the TT_{taper2} rides (upper and middle panels) and the changes in relative work rate in relation to the change in speed during the TT_{taper2} vs. TT_{pre1} (lower panel).

Discussion

The main finding of the current study is that interval training followed by a taper significantly improves 100-km time trial performance in the laboratory. Six interval-training sessions of 15-minute cycling intervals, performed at 70% of W_{peak} provide a performance-enhancing stimulus to already trained cyclists only when followed by a taper lasting longer than a week whereby the HIT training volume and frequency are reduced by 50%. To our knowledge, this is the first study to document the effects of interval training and taper on athletic performance lasting longer than an hour.

Recently, Schabort et al. (1998) showed the TT_{100} performance test, to be both reliable and reproducible with a C.V. of 1.7% (95% confidence interval = 1.1 to 2.5). This was further confirmed by the low C.V. (2.4 ± 2.1%) values for the baseline 100-km time trial of the 16 cyclists. Similar to the present findings, Schabort et al. (1998) also found lower individual coefficients of variation in TT_{100} cycling performances, including 1- and 4-km sprints, than in measurements of W_{peak} values in progressive exercise tests to exhaustion further validating the use of testing protocols which allow cyclists to choose their own work rates during a simulated competitive effort, than tests in which a fixed workload is imposed on them over distances, which they are not accustomed to.

The linearity in the relationship between intensity (oxygen consumption) and heart rate still remains the best and most widely used model for prescribing training and monitoring the training response. Lambert et al. (1998) in their study of long distance runners reported reductions in heart rates at the same submaximal work rates after endurance training which were greater than the reported day-to-day variations (3 to 6 beats/min) in monitoring heart rate in controlled conditions. The recovery between the interval cycling repetitions during the taper

HIT sessions was greater than before taper by ~ 8 $b.min^{-1}$. The improved recovery between the interval work bouts was not significantly correlated to the significant improvement in cycling performances after 1 week and 2 weeks of taper (Fig. 5.2, middle and lower panels). A possible reason for this could be that the better cyclists in the HIT plus taper group were able to train at a slightly higher percent of W_{peak} (73 vs. 68%) during the HIT sessions than their counterparts making it difficult to assess how well they would have recovered had all the cyclists been working at the same absolute intensity.

An earlier finding in chapter 3, was that the exponential decreases in heart rate in the first minute of the recovery following a variable-intensity exercise test are more rapid in the better cyclists than in the poorer cyclists. The correlation between the heart rate reductions and their cycle race performance was greater than that reported in this study (r = 0.93 vs. 0.27) when correlated to the laboratory TT_{100} cycling performance. The significant difference between these two study could be due to the subjects in the current study having to perform constant interval training repetitions (~ 70% of W_{peak} or ~ 4.1 $W.kg^{-1}$) for 15 minutes as compared to the 90-second variable work rates (3.5 to 5.5 $W.kg^{-1}$) during the exercise test. In the former study the work loads during the exercise tests were calculated using watts per kilogram body mass ratios whereas in the current study the workloads were derived from a laboratory performance test to exhaustion (W_{peak} test). The two methods have contrasting biases in that a 5.5 $W.kg^{-1}$ workload to a well-trained cyclists with peak $W.kg^{-1}$ of > 6.0 does not present a difficult task whereas 70% of the laboratory-determined W_{peak} workload lasting 15 minutes does. The converse is advantageous for the poorer cyclists whose $W.kg^{-1}$ ratio is ~ 5.0 and the W_{peak} value is not a true representation of their maximum power output.

The six 100-km time trial rides performed by the control group provided a training stimulus that maintained their performances within 3.6 ± 1.3% throughout the study (Fig. 5.3). The high-intensity interval training was performed at a higher intensity than the pre-training TT_{100} rides (~70% vs. ~60% of W_{peak}) and had no immediate effect on the HIT plus taper group's subsequent time trial performance (TT_{post}). Only after 1 week and 2 weeks of taper in the present study, where training frequency and volume was reduced by 50%, did TT_{100} cycling performances improve by ~4% and 7% respectively.

Similar to the conclusions of Lindsay et al. (1996); Westgarth-Taylor et al. (1997) and Weston et al. (1997) the improvements in cycling performance were due to the subjects being able to sustain significantly higher absolute and relative work rates during final 40-60 kms of the time-trials (Fig. 5.4 and 5.5). The rationale behind the significant improvements in TT_{100} in the study after a week and after two weeks of taper is unclear. We suspect that the preceding training program and the intensity at which it was performed provided an overload stimulus to these cyclists from which a recovery period was required. Shepley et al. (1992) in their study of middle-distance runners, who either increased or decreased the training intensity during the 7-day taper while reducing the training volume, reported significant performance increases in a time to fatigue treadmill run in the group that increased the training intensity again underlining the importance of training intensity coupled with significant training volume reductions to subsequent athletic performance.

Alternatively, the intensity and duration of the subsequent performance exercise after the training protocol might also have demanded a recovery period during which the training was reduced significantly while the intensity is maintained.

After the high-intensity training there were no significant changes in the ability to produce peak power outputs, as measured by the W_{peak} test (390 ± 29 vs. 395± 32 W). The ability to sustain a higher percent of maximum power during the 1-km and 4-km sprints was significantly enhanced from 77% to 86% of pre-HIT W_{peak} for the 1-km sprints and 65% to 70% for the 4-km sprints (Table 5.2). The sprints only accounted for 20% of the total race distance (Fig. 5.4) and ~1% of the improvements in overall performance. The remaining ~5% improvement in performance must have occurred during the non-sprint sections of the trial (Fig. 5.4). This result provides some explanation for the poor association between improvements in W_{peak} and in time trial performance. Westgarth-Taylor et al. (1997) also reported a poor association between improvements in W_{peak} and improvements in 40-km time trial performance after high-intensity interval training. The improvements were not accounted for by the increases in absolute or relative heart rate during the trials but by the cyclists' ability to sustain a higher percent of work during the last 40 km of the trial (Fig. 5.5). This is however not surprising as the interval training program was designed to enhance power outputs at intensities around 70% of W_{peak} supporting the specificity of training at these intensities and performance tests of this duration.

In the present study the better cyclists were able to attain a higher W_{peak} value and train at a slightly higher percent of W_{peak} than their similarly trained counterparts. Consequently during the event they were able to sustain a higher percent of HR_{peak} and W_{peak} during the latter stages of the time trial (Figs. 5.5 and 5.7). The greater 'fatigue resistance' of better distance athletes (Peronnet and Thibault, 1989, Coetzer et al. 1993) and their ability to utilize a higher percentage of peak HR and W_{peak} is not well-understood (Hawley 1997). The superior endurance performances may in part be accounted for by the lower rates of lactate accumulation in working muscles (Costill et al. 1973, 1976; Farrell et al. 1979; LaFontaine et al. 1981; Sjodin and Jacobs 1981; Sjodin et al. 1982; Coetzer et al. 1993), a greater tolerance of

waste products of anaerobic metabolism (Martin et al. 1994), and the faster heart rate recovery after high intensity work bouts (chapter 3, Fig. 3.7).

A number of reviews (Neufer 1989; Houmard et al.1994; Mujika 1998) and studies (Table 2.8 and 2.9) have reported improvements in performance following a taper to be around 3%. To the best of our knowledge, only two studies (Shepley et al. 1992; Zarkadas et al.1995) have reported athletic performance changes of greater than 5% following taper. Shepley et al. (1992) increased the training intensity over the 7-day progressive taper period which could have accounted for the large improvements in running to exhaustion. Zarkadas et al. (1994, 1995) in their study exponentially reduced the training volume of well-trained triathletes over 8 days of a 13-day taper by 50% and reported significant performance improvements in a 5-km running time trial.

The cyclists in this study improved endurance performance by ~6% following a 50% progressive reduction in training volume and frequency over two weeks. Whether performance would have further improved after a third week of taper it remains to be investigated.

CHAPTER 6

PHYSIOLOGICAL RESPONSES TO INTERVAL TRAINING AND TAPER ON CYCLING PERFORMANCE IN WELL TRAINED CYCLISTS

RATIONALE AND AIM

The practice of tapering is still widely believed to reduce the physiological and psychological stress associated with intensive training. While many taper studies have reported performance improvements of ~3% (Costill et al. 1985; Johns et al. 1992; Houmard et al. 1994; Zarkadas et al. 1995; Mujika et al. 1996b; Banister et al. 1999), the athletes in the current studies improved their endurance performances after a 1-week taper by 5.4% and after a 2-week taper by 7.3% (chapter 5).

The physiological, metabolic and psychological adaptations that occur during taper have been reviewed in Chapter 2.4. Only a handful of studies (Neary et al. 1992; Shepley et al. 1992; Gibala et al. 1994; Trappe et al. 2001) have investigated the changes or adaptations occurring specifically in the muscle during training and taper. The hypothesis therefore, was would electromyographic spectral shifts within the muscle provide a direct measure of recovery or fatigue resistance.

A primary aim of this study was to investigate if there are changes in fuel utilisation, muscle recruitment or muscle strength during a high-intensity interval training phase, taper and during a habitual training phase. Secondly, whether these changes are significantly associated with changes in athletic endurance performance and thirdly to investigate whether a longer taper (3 vs. 2 weeks) would further improve performance in already-trained cyclists.

Methods

Six competitive, endurance-trained male cyclists participated in this study. At the time of the investigation, the cyclists were training between 200-500 km.wk^{-1} in preparation for an international annual 104-km Argus cycle road race. Participating in this race was a requirement of the study. The study was approved by the Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town and each cyclist completed a written informed consent form.

Simulated 100 km time-trial (TT_{100}) cycling performances

Simulated TT_{100} rides were performed on a Kingcycle™ ergometry system (Kingcycle™ Ltd, High Wycombe, U.K.). The system allows cyclists to ride their own racing bicycles in the laboratory. The testing procedure and the system has been described in detail in the previous chapter. It has a coefficient of variation of 1.1 ± 0.9% and 1.0 ± 0.5% for 20 and 40-km cycling time (Hickey et al. 1992; Palmer et al. 1996, respectively).

Each cyclist completed two TT_{100} rides prior to any intervention. Although the cyclists were used to cycling on a cycle ergometer in a laboratory setting, the first TT_{100} was regarded as a familiarisation ride. The second TT_{100} ride (TT_{pre}), was performed within four days of the familiarisation trial and was regarded as the first performance test of the study. All subsequent time trials (Post, Taper1, Taper2, Taper3 and 6weeks) were performed at the same time of day. Laboratory conditions remained constant (~20°C, 55% relative humidity) during all trials. The testing procedure is described in detail in the Methods section of Chapter 5.

Submaximal testing

Three days after TT_{pre}, the cyclists performed successive 10-min rides at 50 and 100% of the mean power output for TT_{pre}, and a further 5-min at 115% of the time trial's mean power output. These intensities corresponded to about 30, 60 and 75% of W_{peak}.

Before the submaximal rides, an 18-guage Teflon cannula (Jelco, Johnson and Johnson, Halfway house, South Africa) was positioned in an antecubital vein and connected to a two-way stopcock (Uniflex, Mallinckrodt, Hennef-Seig, Germany). This cannula was flushed periodically with 2-3 mL of sterile saline containing heparin (5 IU mL^{-1}) and was used for the collection of venous blood samples (10 mL) at rest and during exercise. Venous blood samples (10mL) were drawn at rest, 1, 5, 10, 11, 15, 20, 21 and 25 min during the submaximal test for measurements of plasma lactate concentrations as described in the Methods section of Chapter 3.

Prior to the submaximal exercise, the subject's height (cm), weight (kg), thigh length (cm) and mid-thigh circumference (cm) were measured. Tricep, bicep, suprailiac, subscapular, mid-thigh skinfold measurements (mm) were also recorded to calculate percent body fat using the formula of Durnin and Wolmersley (1974). Lean thigh volume was calculated by assuming the thigh to be a truncated cone and taking into account the thigh skinfold measurement (Katch and Katch, 1974). The subject's knee extensor isometric strength was determined on a Kin-Com isokinetic dynamometer (Chattanooga Group Inc, USA). The knee extensors were tested isometrically at an angle of 65°. Each subject performed 4 sub-maximal familiarisation trials on the isokinetic dynamometer. Following the warm-up, subjects performed 4 maximal extensions for each test. The subjects were verbally encouraged during the test to exert maximal efforts for all 4 trials. The highest value recorded of the 4 trials was used in subsequent analyses.

All the submaximal and W_{peak} tests were performed on an electrically braked cycle ergometer (Lode, Groningen, Netherlands). During the tests, the cyclists breathed through a facemask connected to an Oxycon Alpha automated gas analyser (Mijnhardt, Netherlands). Before each test, the analyser was calibrated with a Hans Rudolph 3L syringe (Vacuumed, Ventura, USA), room air and a 5% CO_2: 95% N_2 gas mixture. Analyser outputs were processed by a computer, which calculated L.min^{-1} ventilation (V_E), oxygen consumption (VO_2) and carbon dioxide production (VCO_2) values for each breath. VO_2 peak values were the average of the highest VO_2 values measured over 60 s in the final work rate.

Electromyographic activity testing and analyses

Prior to the isokinetic testing, the subject's skin overlying the "belly" of his rectus femoris, midway between the superior surface of the patella and the anterior superior iliac crest was shaved, abraded and swabbed with an alcohol pad. A triode electrode was then taped onto the site, covered with cotton swabs to minimise sweat-induced interference and connected, via a fibre-optic cable, to a Flexcomp/DSP EMG apparatus (Thought Technology USA) and a computer. To eliminate interference from electrical sources, EMG signals were filtered at 50 Hz. The raw EMG was automatically anti-aliased by the hardware (Thought Technology, U.S.A.). Each recording was sampled at a 1984 Hz capture rate for 5 s during the 4 maximal isometric trials and during the submaximal tests at 1, 5, 10, 11, 15, 20, 21 and 25 min. All the EMG recordings on the Lode bike were collected at constant pedalling cadence of 80 revolutions per min.

Motion artefact was removed from the raw EMG signals with a 2nd order high-pass Butterworth filter with a 3dB cut-off at 15Hz. A root mean square (rms) of the 5 s EMG recordings during

maximal voluntary contractions was taken as a measure of "total myoelectric activity" (Moritani et al. 1987 and Green and Patla 1992) and used to normalise the subsequent EMG recordings during sub-maximal exercise.

The spectral frequency of the raw EMG data was assessed with a fast Fourier transformation algorithm. Shifts in the percentile frequency were calculated from the mean shift in the 65th to 90th percentile range of the total cumulative amplitude spectra. This procedure was described by Lowery et al. (1998) and is a modification of the work of Lo Conte and Merletti (1996). These authors suggested that this method is a more accurate estimation of spectral compression than analysing a single (50th) percentile median frequency (Lo Conte et al. 1996; Merletti et al. 1996; Lowery et al. 1998).

Maximal incremental exercise test

On completion of the submaximal testing protocol, subjects were allowed a recovery period of their choice before performing a progressive incremental test to exhaustion for the determination of peak power output on an electronically braked cycle ergometer (Lode, Groningen, The Netherlands). The same testing procedure was followed as described in the Methods of Chapter 3. The highest heart rate attained during the maximal incremental test was referred to as the heart rate peak (HR_{peak}) while the VO_2 peak value was the average of the highest VO_2 values measured over 60 s in the final work rate.

High-intensity interval training (HIT)

On completion of the preliminary testing, the cyclists were given diaries to record their training and instructed to continue their normal training in between supervised HIT sessions as described in the Methods section of Chapter 5. Since the cyclists may have varied in their ability to tolerate the 'effort' in

HIT, no attempt was made to control their endurance training volumes. The cyclists were only requested not to perform any HIT outside the laboratory.

Post-HIT testing and Taper

Within seven days after the sixth HIT session, cyclists performed another TT_{100} (TT_{post}), a second submaximal test (SUBMAX POST) and a W_{peak} test. The frequency of their HIT was reduced by 50% over 3 weeks (resulting in a 50% training volume reduction over the same period) while each week TT_{100} rides (Taper1, 2 and 3) were also performed. Two weeks into the 3-week taper, the cyclists also performed another submaximal and Wpeak test (SUBMAX TAPER). Following the 10 weeks of testing and training, the cyclists returned to their normal training and racing on weekends for 6 weeks and then repeated the submaximal (SUBMAX 6 WEEKS) and W_{peak} tests and, within 3 days, performed another TT_{100} ride (TT_{6weeks}).

Ratings of Perceived Exertion

During each TT_{100} trial, subjective ratings of perceived exertion were measured using the Borg scale (Borg 1975) after 10, 50, 85 and 99-km.

Statistical analyses

Results are presented as means ± S.D. except those of the electromyographic activity which are expressed as means ± S.E.M. Statistical significance was assessed with a one-way analysis of variance (ANOVA) for repeated measures followed by Scheffes post hoc tests (Statistica, StatSoft Incorporated, Tulsa, U.S.A.). A Pearson product moment correlation (r) was used to examine relationships between selected variables. Results were considered significant when the P value was ≤ 0.05.

The test-to-retest reliability of the measurements of W_{peak} and simulated TT_{40} performance have previously been reported to be 2.0 ± 1.7% (Neary et al. 1999; Westgarth-Taylor et al. 1997). The reliability of the rms-EMG data across a 5-second recording of muscle activity at a constant workload and cadence was r = 0.81 while the measure of the spectral shift changes was r = 0.87.

Results

The physiological characteristics and performance data of the six subjects who completed the study are shown in Table 6.1.

Table 6.1. Physiological and performance characteristics of the subjects.

Subjects	Age (y)	HR_{peak} (b/min)	Fat (%)	LTV (cm^3)	P/W ratio ($W.kg^{-1}$)	Cycle training (km/wk)	TT_{100} (min:s)	Argus Race (min:s)
1	27	178	11.4	5201	5.86	250	132:57	166:42
2	19	192	11.9	4562	6.04	205	151:49	172:37
3	20	184	12.6	4617	4.99	182	149:27	177:02
4	22	188	13.2	5651	5.43	204	166:02	167:22
5	28	180	9.3	4365	5.27	188	151:44	171:45
6	22	190	8.6	5445	6.08	174	157:25	185:57
Mean	23	185	11.2	4974	5.61	200	151:34	173:34
S.D.	3	6	1.8	529	0.45	27	11:02	7:08

HR_{peak}, is the peak heart rate recorded during the road race or during the maximal, incremental test; LTV is the lean thigh volume; P/W is the power to weight ratio. Cycle training is the average cycle training per week before the start of the study. The best TT_{100} time in min were recorded during the baseline measurements. Race time is the time taken to complete an international 104-km Argus road cycle race.

There were no significant changes (only one group) in the subjects' HR_{peak}, estimated body fat, lean thigh volume or power to weight ratio during the course of the study. The simulated 100-km time trials were performed at a faster velocity (9.5 ± 5.7% faster) than the 104-km road cycle race (39.8 ± 3.0 vs. 36.0 ± 1.4 km/h; P = 0.01).

Figure 6.1 shows the effects of HIT, taper and six weeks of habitual training on peak power output, W_{peak} and peak oxygen consumption, VO_{2peak}. Although not statistically significant W_{peak} on the maximal, incremental test increased from 372 ± 37 to 387 ± 44 W (~ 4%) and VO_{2peak} declined from 69.2 ± 2.9 to 65.8 ± 4.8 $mL.min^{-1}.kg^{-1}$ (~ 5.2%) after the intervention. The isokinetic peak force during a maximal voluntary contraction (MVC) was significantly reduced by the HIT program from 695 ± 148 to 572 ± 131 N and further reduced to 555 ± 107 N after 2 weeks of taper (P = 0.03; Fig. 6.2, *upper panel*). The electromyographic activity and spectral shifts during the TAPER MVC (Fig. 6.3, *middle panel and lower panel*) were not significantly affected by the intervention, although there was a tendency during the taper measurements for the spectral shift changes to be lower than during the PRE, POST and 6 WEEKS MVCs.

The average speeds and power outputs during the TT_{100}s rides improved after the HIT program from 39.8 ± 3.0 to 40.2 ± 1.7 km/h and from 240 ± 46 to 246 ± 25 W, and further improved during taper, with peak performances observed two weeks into the taper, 41.8 ± 2.9 km/h and 269 ± 52 W (P ≤ 0.05, Fig. 6.3 (*upper and middle panels*). Six weeks after the taper, (TT_{6weeks}) speeds and power outputs were reduced to 39.4 ± 3.0 km/h and 233 ± 41 W and were similar to TT_{pre} values. The latter performances were significantly slower than those in TT_{taper2} (P ≤ 0.05).

Figure 6.4 (*upper, middle and lower panels)* show the cyclists' mean sustained power output profiles during the TT_{pre}, TT_{taper2} and TT_{taper3} and their individual power outputs during the TT_{pre} and TT_{taper2}.

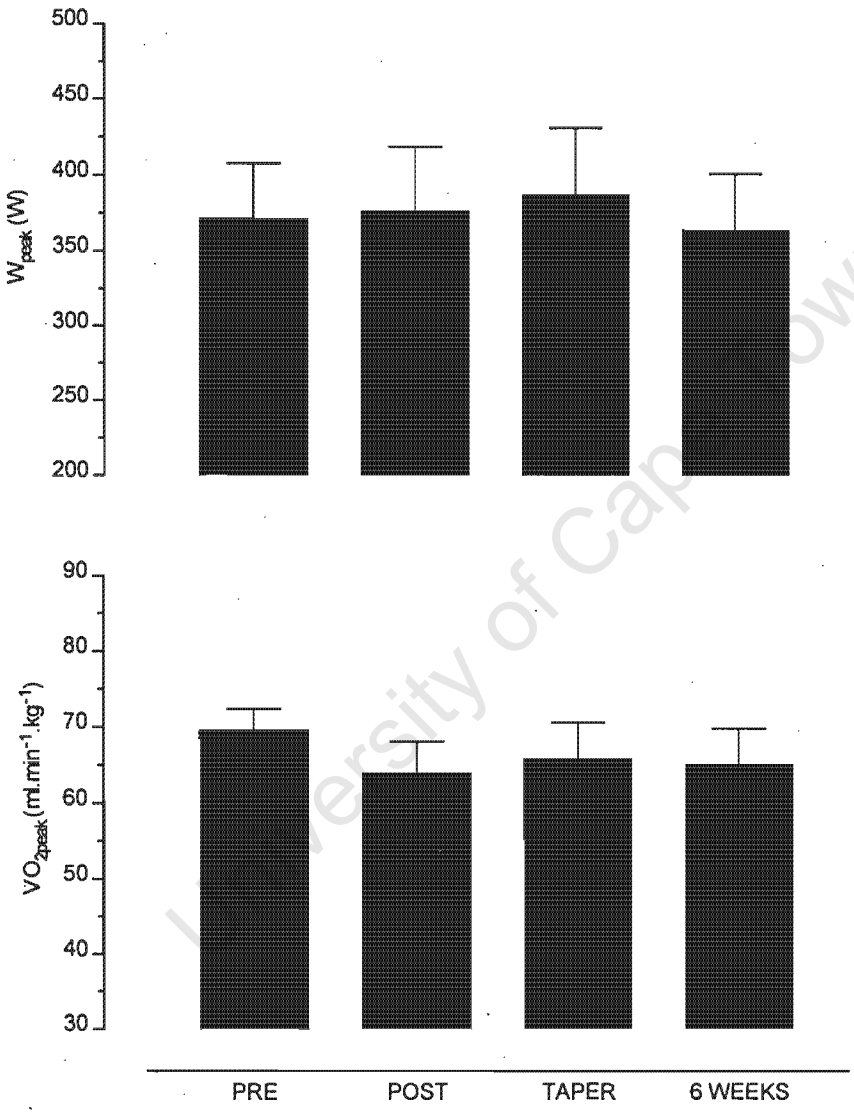


Figure 6.1. Effects of sustained high-intensity interval training (HIT) and taper on peak power outputs W_{peak} and peak oxygen consumption VO_{2peak}. Values are means (SD).

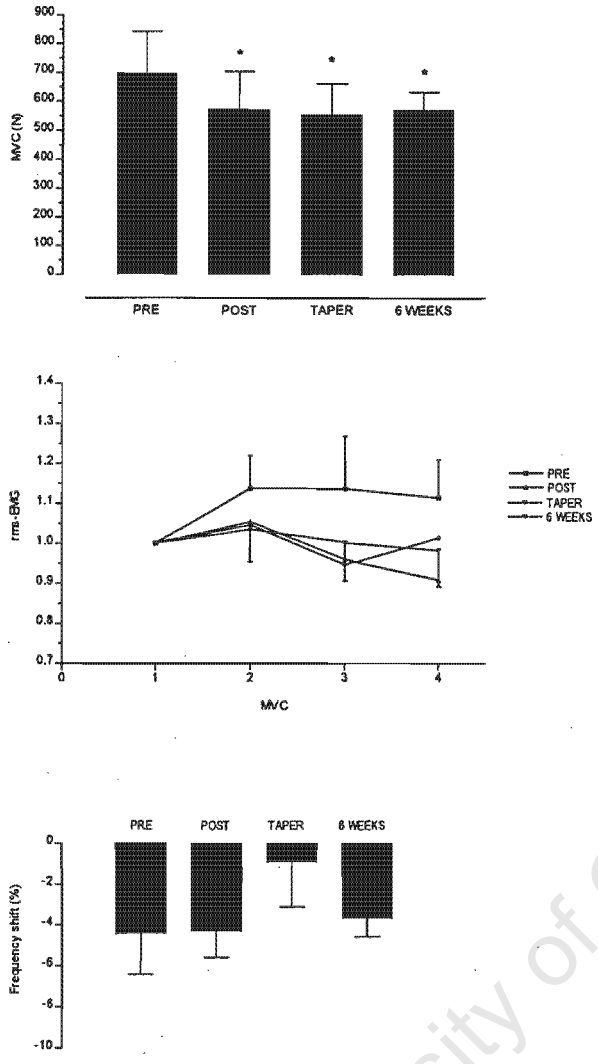


Figure 6.2. Effects of training and taper on peak isokinetic strength and electromyographic activity. Peak isokinetic force values during the 5-sec maximal voluntary contraction (MVC) (*upper panel*). rms-EMG activity during the 4x 5-sec MVCs normalised to the 1st MVC (*middle panel*). Frequency shift changes during the maximal contractions (*lower panel*). *Indicates significantly different from PRE, $P < 0.05$. EMG activity values are means (SE).

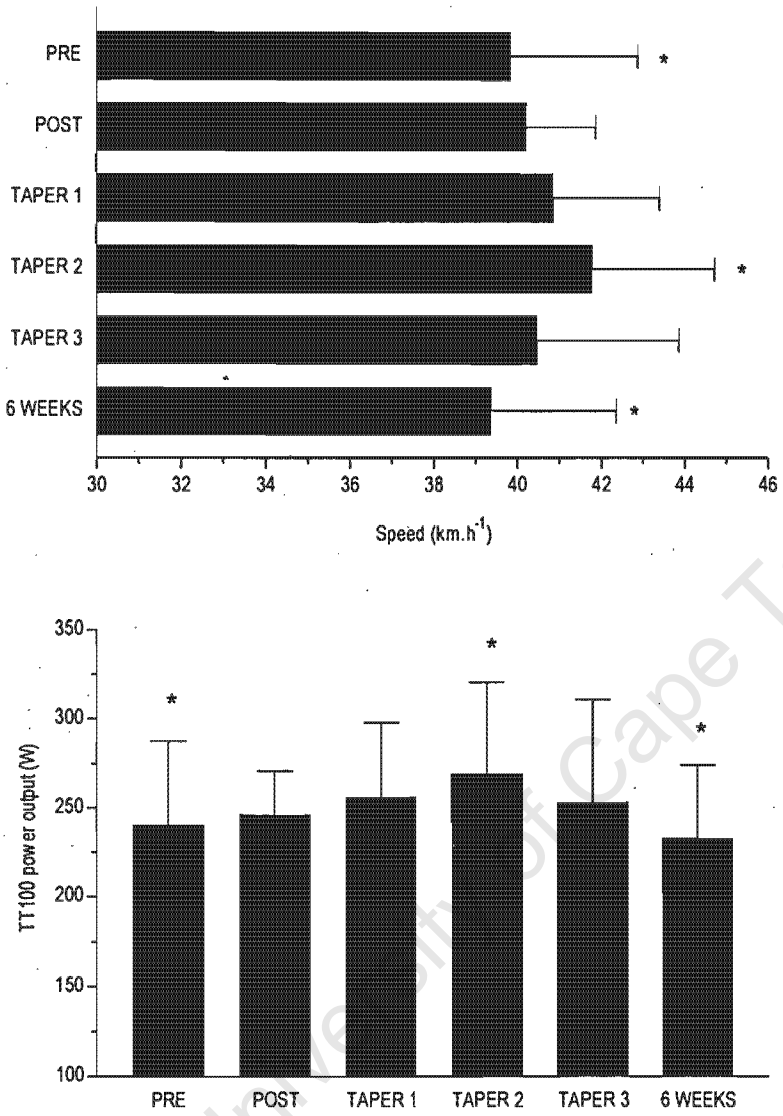


Figure 6.3. Effects of high-intensity interval training and taper on average cycling speeds (upper panel) and absolute power outputs during the simulated TT_{100} (lower panel) . *Indicates TAPER 2 was performed significantly faster and at higher power outputs than PRE and 6 WEEKS, $P < 0.05$.

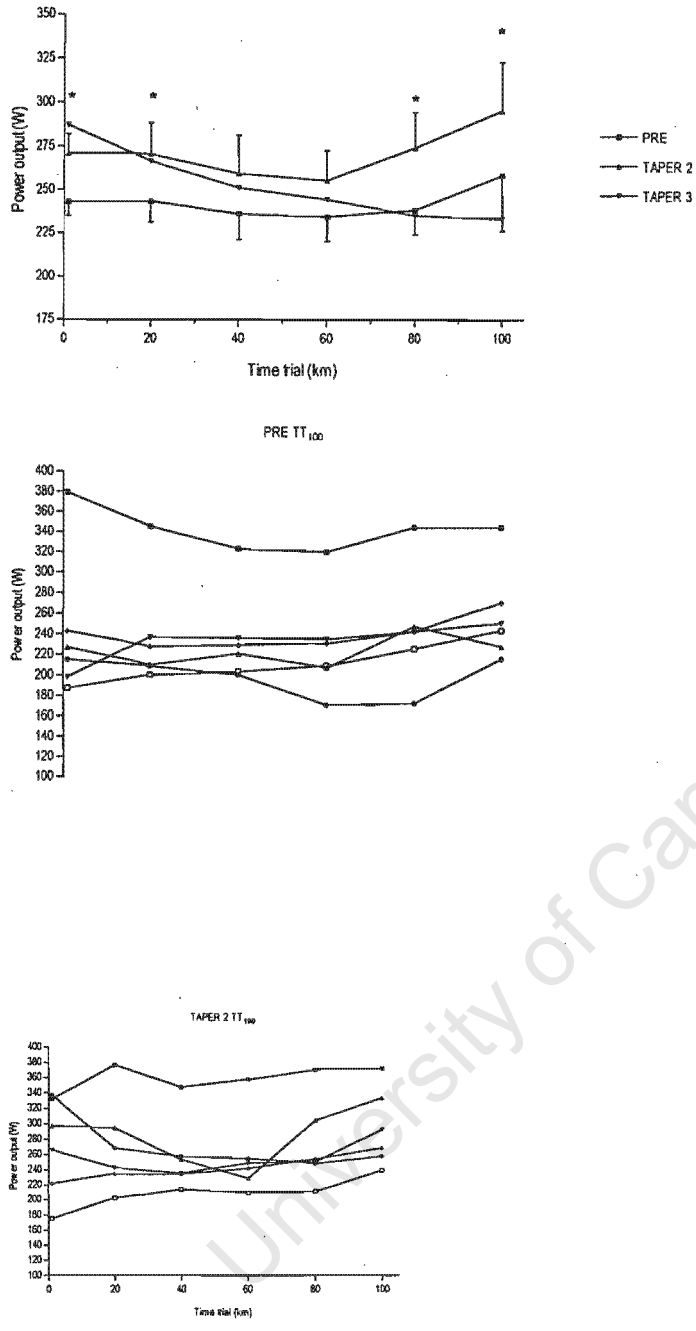


Figure 6.4. Sustained power output profiles for the PRE,TAPER 2 and TAPER 3 TT_{100}s. Mean power outputs during the PRE, TAPER 2 and TAPER 3 TT_{100}s(*upper panel*). Individual mean power outputs during the PRE TT_{100} (*middle panel*). Individual mean power outputs during the TAPER 2 TT_{100} (*lower panel*).
*Indicates significantly higher power outputs than PRE, $P < 0.05$.

HIT and a 2-week taper significantly increased the sustained power outputs during the first 1-km and 20-km, and during the last 40-km of the TT_{taper2} compared to the TT_{pre} ($P \leq 0.05$). The power output over the last 20-km of TT_{taper2} was a particularly good predictor of the overall performance in the trial ($r = -0.96$, $P = 0.01$). The subjective ratings of perceived exertion (RPE) on the Borg scale, after 10, 50, 85 and 99-km of the time trials were not altered by HIT and tapering. As expected, there was an increase in the rating of perceived exertion over time (data not shown, $P = 0.001$).

Figures 6.5 and 6.6 show the heart rate responses of the cyclists during the Taper2 100-km time trial and during the 104-km cycle race. As expected, there was a trend for the heart rates to increase in response to the 1-km and 4-km sprints, but during the cycle race the heart rates varied independently of the course terrain.

There was a significant linear relationship between W_{peak} and TT_{taper2} speed (km/h) and during taper ($r = 0.81$; $P \leq 0.05$) but the relationship did not reach significance for the cycle race ($r = 0.74$; $P = 0.08$) and VO_{2peak} during taper ($r = 0.72$; $P = 0.11$), Fig. 6.7 and 6.8.

Figure 6.9 shows the heart rate responses and rates of carbohydrate oxidation (*left panels*), and the EMG activity and plasma lactate concentrations (*right panels)* during the 10-min cycling bouts at work rates of 30 and 60% of pre-HIT W_{peak} and during 5-min rides at 75% pre-HIT W_{peak}. Fifteen minutes into the TAPER submaximal test, the EMG activity was significantly lower than in the PRE submaximal test ($P = 0.04$). Plasma lactate concentrations after the HIT and 2-week taper were reduced throughout the test and reached significance 15 and 25 minutes (PRE 4.51 ± 2.59 and 6.32 ± 4.67 vs. TAPER 2.56 ± 0.97 and 4.75 ± 2.38 $mmol.L^{-1}$) into the test ($P \leq 0.05$). Rates of carbohydrate oxidation and heart rate responses were

significantly higher during cycling at 60 and 75% of pre-HIT W_{peak} values when compared to 30% of pre-HIT W_{peak} values ($P \leq 0.05$) and the training and taper did not significantly alter these responses.

The increases in heart rate, the EMG spectral shifts and the EMG amplitudes during the 8th HIT session after a 2-week taper were less than in the 1st HIT session (Fig. 6.10). In particular, the EMG activity during the 8th HIT after a 2-week taper did not increase as a function of time as occurred during the 1st HIT session.

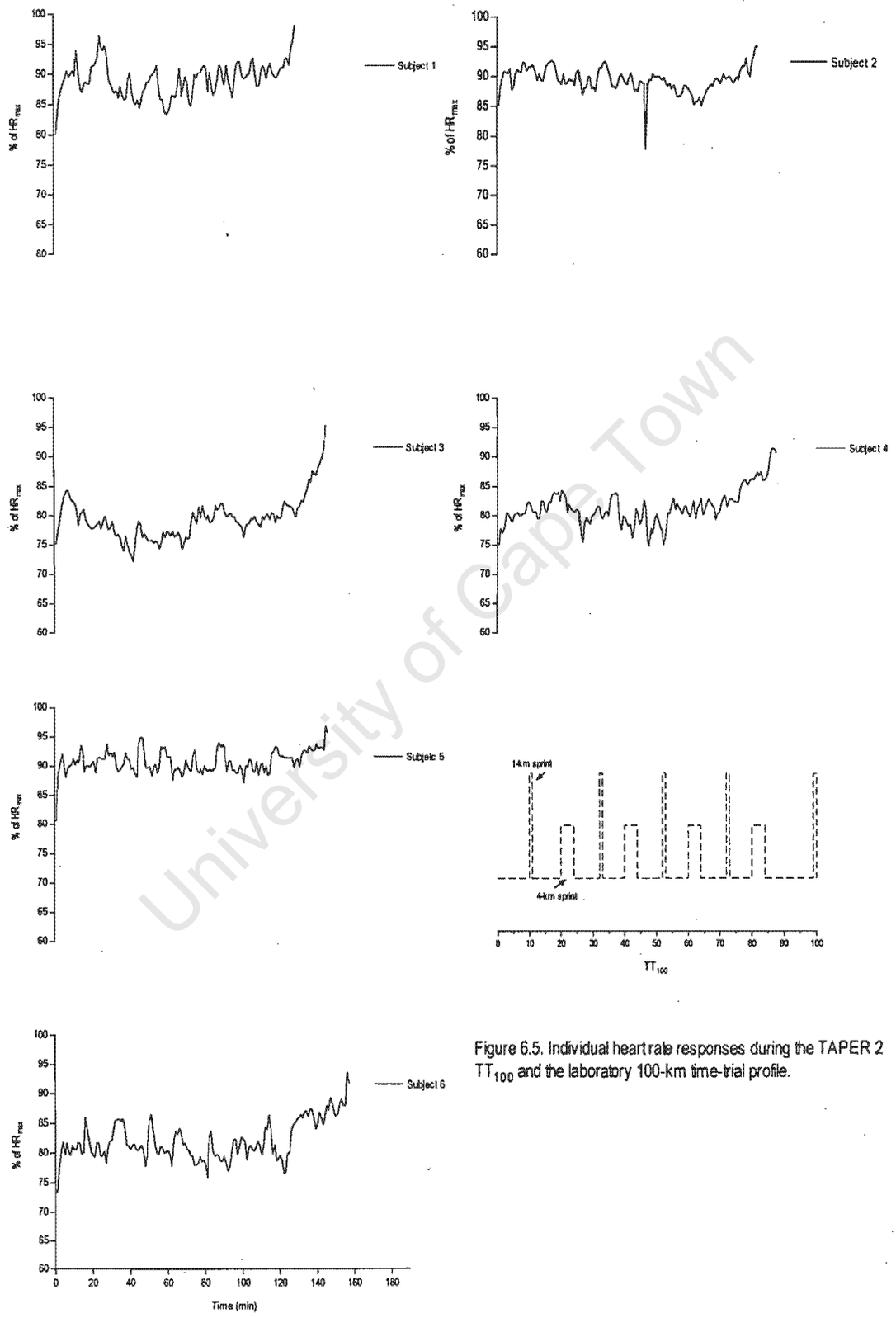
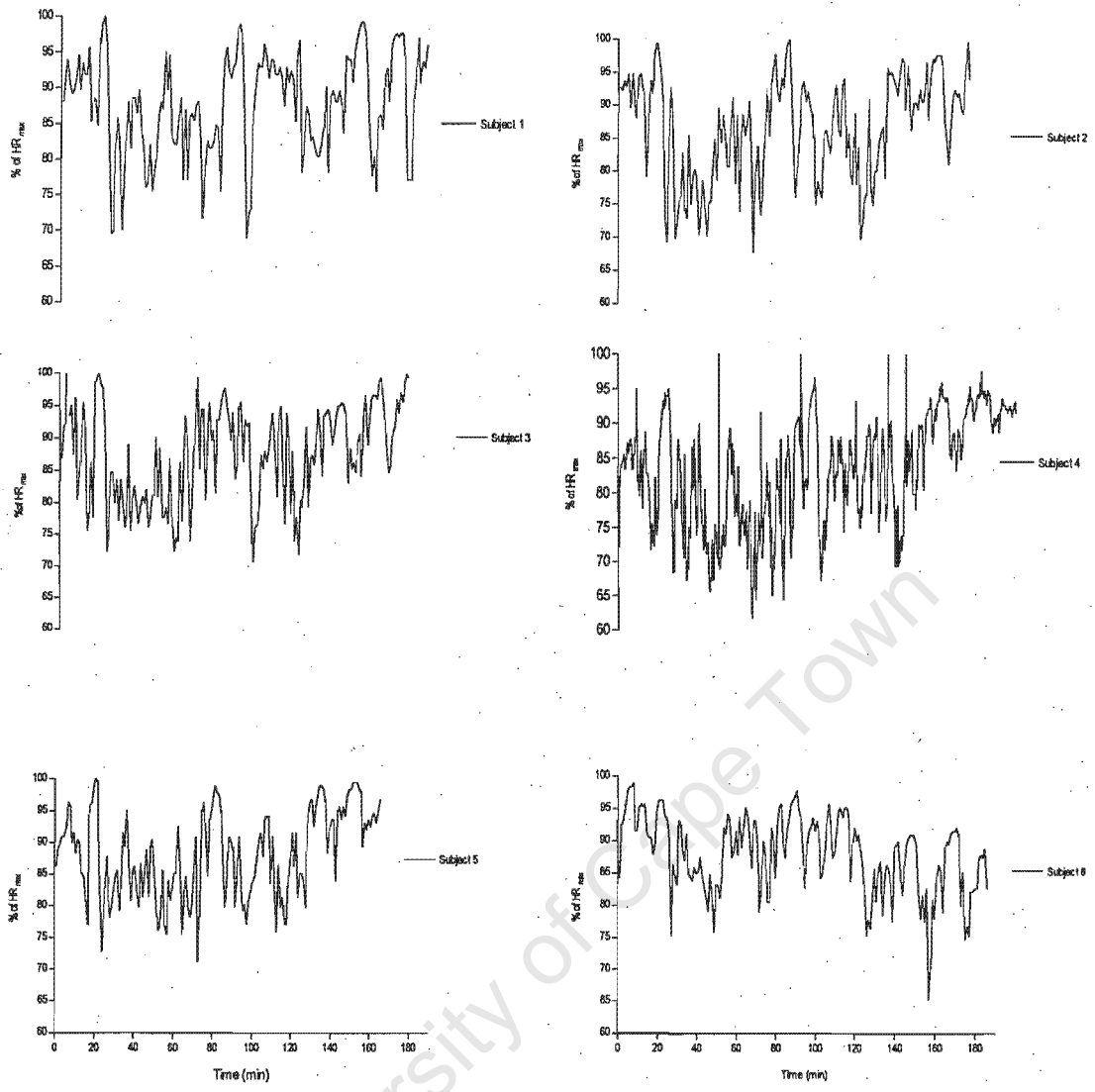


Figure 6.5. Individual heart rate responses during the TAPER 2 TT_{100} and the laboratory 100-km time-trial profile.



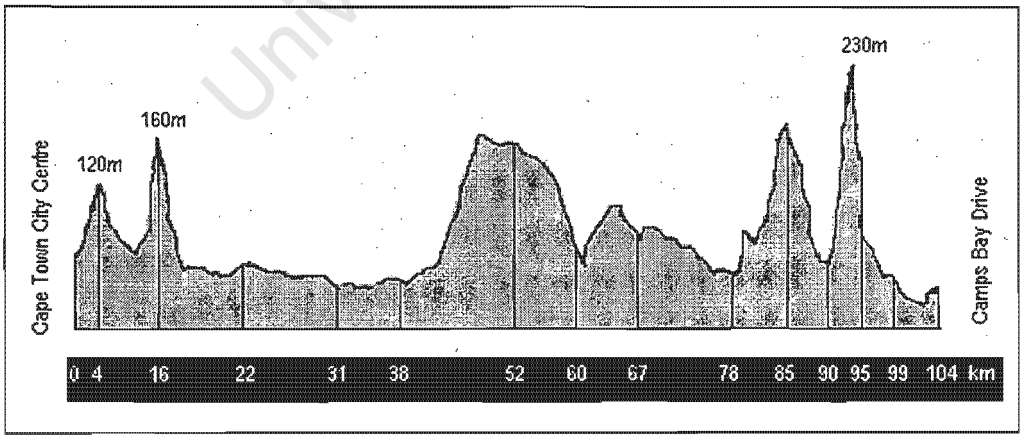


Figure 6.6. Individual heart rate responses during the Argus cycle race and the course profile.

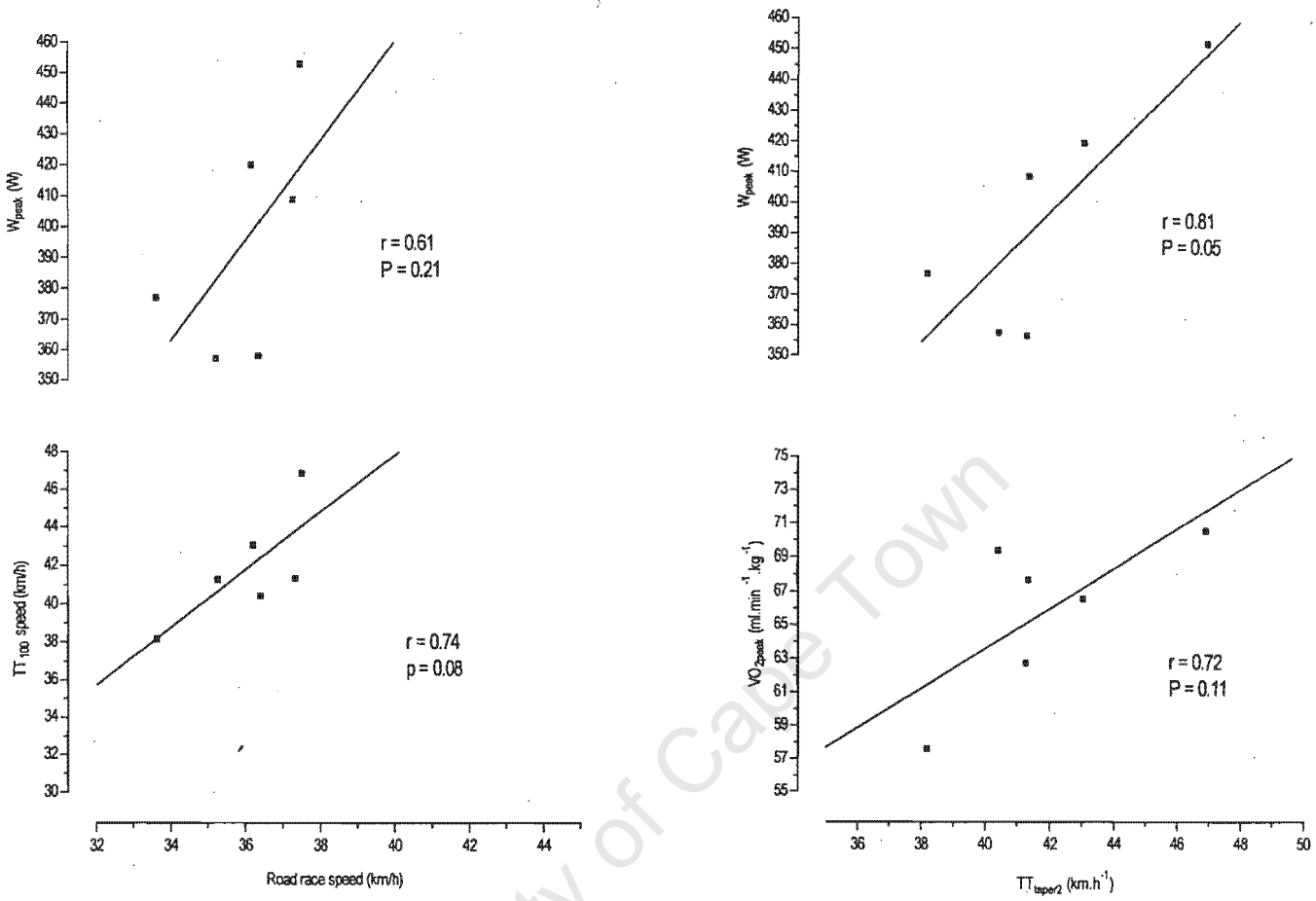


Figure 6.7 and 6.8. Peak power output (W_{peak}) and performance in the laboratory 100-km time trial and their relationship with performance in a mass-start road race (left panels); peak power output (W_{peak}) and VO_{2peak} during the maximal incremental test and their relationship with performance during the TT_{taper2} (n = 6) (right panels).

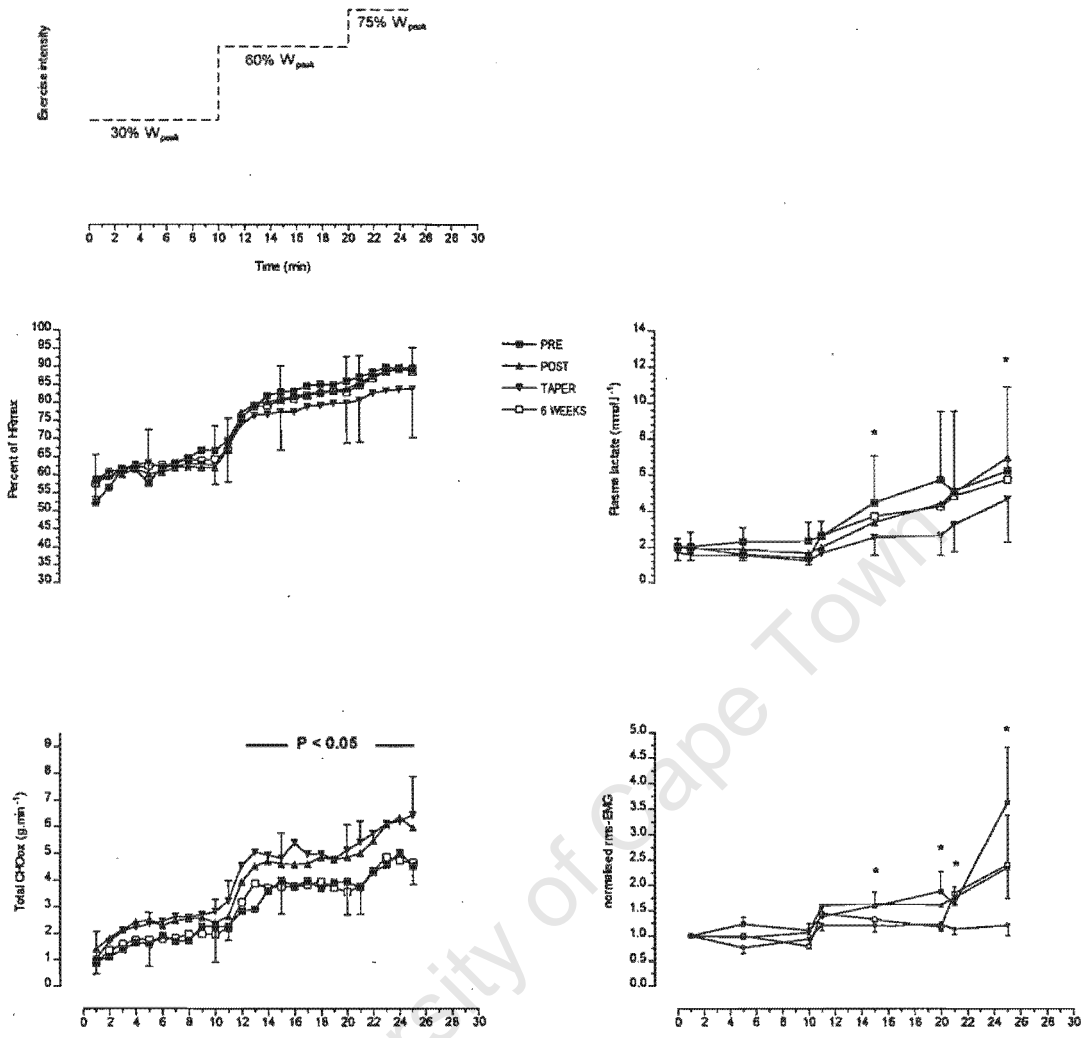


Figure 6.9. Increases in heart rate, carbohydrate oxidation (*left panels*) and EMG activity, plasma lactate concentrations (*right* panels) with rises in submaximal workloads. *Indicates significantly lower TAPER values than PRE (P≤ 0.05).

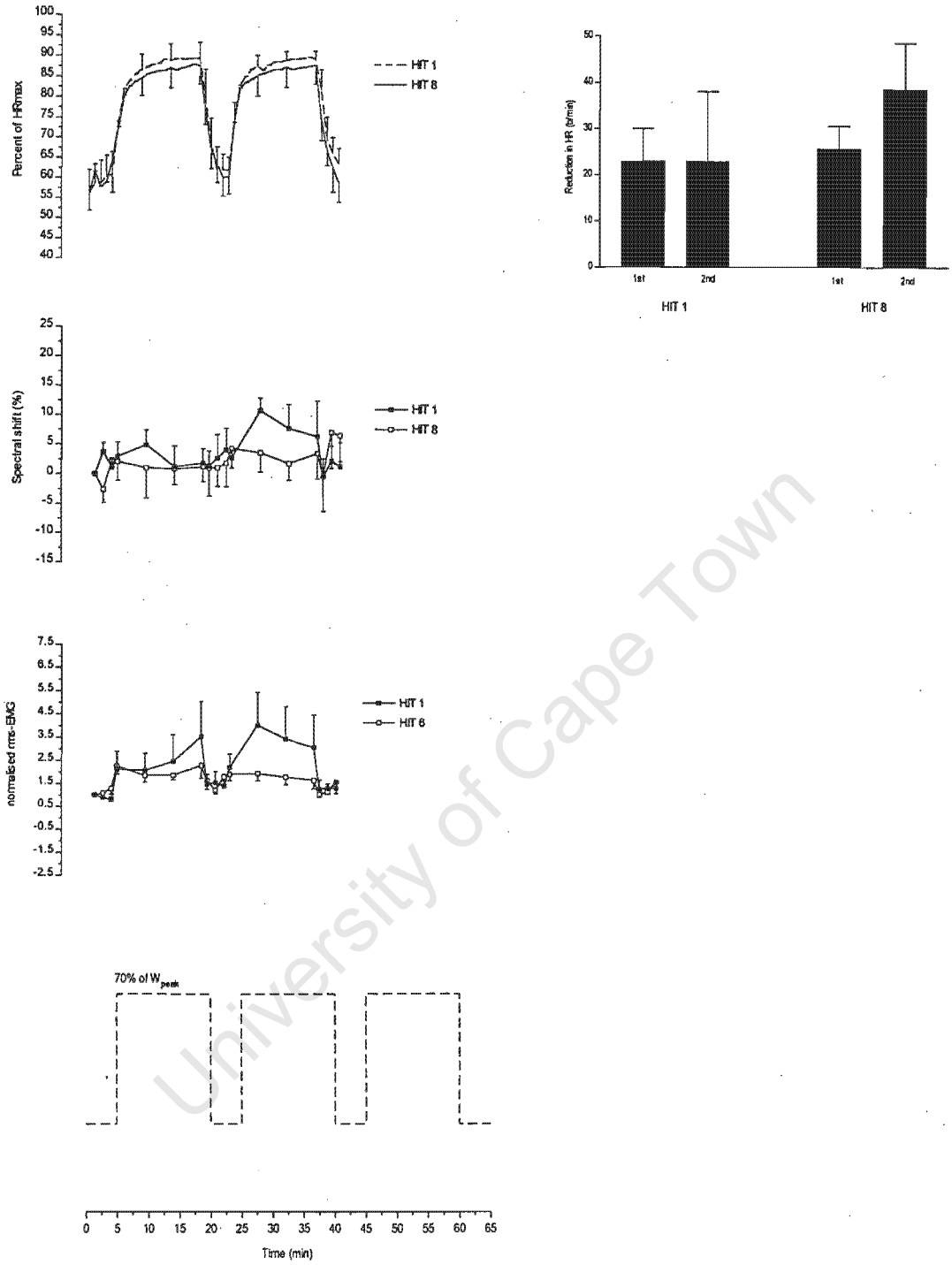


Figure 6.10. Mean heart rate responses and the electromyographic activity during the 1st and 8th high-intensity interval training sessions and the reductions in heart rate during the first minute of the 5-min recovery after the 1st and 2nd cycling repetitions. Electromyographic activity values are means (SEM).

Discussion

As expected, six high-intensity interval training sessions followed by taper significantly enhanced endurance performance in already trained cyclists. The conclusion of the previous chapter (chapter 5) that a 2-week taper was optimal for improving endurance performance in already trained cyclists was further confirmed by the decrement in TT_{100} performance after 3 weeks of taper.

A number of studies have investigated the reliability of surface electromyographic measurements during maximal voluntary contractions (Kollmitzer et al. 1999) of the knee extensor and during submaximal cycling activities. The reliability and reproducibility of the quadricep muscle groups (rectus femoris, vastus lateralis and vastus medialis) was determined. These studies reported the EMG to be better reproducible if submaximal measurements are performed during isometric knee extension tests (Kollmitzer et al. 1999). The rectus femoris muscle was also the most reliable of the three muscle groups when assessing EMG activity and frequency shifts (Kollmitzer et al. 1999). The results from our data are quite reliable ($r = 0.87$) and are representative of the electromyographic activity of the rectus femoris during the exercise tests.

In agreement with earlier findings (chapter 3) W_{peak} did not predict road-racing performance significantly ($r = 0.61$, $P = 0.21$). The ability to produce large power output is partly determined by body mass. This is an advantage for the cyclist when racing on courses that are flat with little or no wind resistance and at a near constant rolling resistance. A laboratory time trial simulates these conditions and therefore provides a bias to the heavier cyclists. The subjects in this study were of a similar weight (range 60 - 72kg) and therefore minimised the benefit of body mass on performance. This point is partly confirmed by the fact that the heaviest cyclist in

this study was not the fastest in the road race or laboratory time trial, nor was the cyclist with the highest power to weight ratio. These data indicate that road racing performance is probably determined by a combination of factors which might include; the ability to recover quickly after periods of high workload, racing experience, tactical ability and sprinting ability. Further research on this topic is warranted, although finding athletes, of this calibre, that are willing to have their training manipulated is an increasingly difficult task.

Contrary to the studies by Shepley et al. (1992) and Martin et al. (1994) who reported increases in muscle strength following taper in well trained middle-distance runners and cyclists, significant reductions in peak isokinetic strength during maximal voluntary contractions were observed in this study. In addition, rms-EMG activity during the contractions were reduced, while the frequency shifts were reduced during taper suggesting a better fatigue resistance during the maximal contractions possibly as a result of training and taper. The relevance of the muscle strength tests that test the muscles at different angles and speeds to those of the sport are questionable. It is questionable whether improvements in muscle strength gained from resistance training might enhance endurance performance in well-trained athletes (Martin et al. 1994). It is speculated that further strength gains may not enhance the endurance performances of highly trained athletes who are already capable of sustaining high power outputs in their chosen sport.

Peak power output however, has been shown to significantly predict time trial performance over a wide range of distances in the laboratory (chapter 5; Noakes and Hawley 1992; Lindsay et al. 1996; Westgarth-Taylor 1997). Similar to the studies of Lindsay et al. (1996) and Westgarth-Taylor et al. (1997), the improvements in time trial performance were not accounted for by the changes in W_{peak} and VO_{2peak} after training and during taper. The ~1.3% increase in

the cyclists' W_{peak} was not related to the ~4.6% improvements in TT_{100} cycling performance. The (significant) relationship reported by Lindsay et al. (1996) for pre-HIT (baseline) W_{peak} and 40-km cycling speed ($r = 0.83$; $P \leq 0.01$) was not found between pre-HIT (baseline) W_{peak} and TT_{pre} cycling speed ($r = 0.53$; $P = 0.2$) in this study. Similar to the findings in Chapter 3, the relationship between W_{peak} and performance in a mass-start road race was not significant ($r = 0.61$, $P = 0.21$) Fig. 6.9.

Similar to the studies by Coyle et al. (1991) and Lindsay et al. (1996), lean thigh volume did not change significantly or account for the changes in performance or the morphological differences between the cyclists in the present study. Coyle et al. (1991) in their study of cyclists with varying cycling abilities, concluded that the greater force generation by the elite cyclists could have been realised by years of cycling which may result in altered muscle contractility (Noakes 1988). Altered muscle contractility may strongly influence neuromuscular recruitment patterns and together through many seasons of training these adaptations may allow competitive cyclists to recruit a greater number of muscle fibers and spread the power production over a larger active muscle mass while pedalling compared with less well-trained cyclists (Coyle et al. 1991; Hawley and Stepto 2001). However more research using reliable electromyographic measurements in longitudinal studies of well-trained or elite cyclists is warranted.

Pacing strategy may have been an important consideration during the endurance performance test on these cyclists. After the training intervention they expected to complete the time trial faster and/or with less effort (personal communication). However, after 3 weeks of taper, their performances declined and were similar to the post-training time trial performance. A miscalculation of their pacing strategy, overtraining, psychological factors and possibly a

decline in the ability to resist fatigue could have resulted in their performance after 3 weeks of reduced training to not have improved further (Fig. 6.4). In addition, after six weeks, time trial performances were similar to the pre-training performances.

The heart rate responses during the laboratory TT_{100} were not independent of the course profile. Although the cyclists were verbally encouraged to produce supra-maximal efforts during the 1-km and 4-km sprints, they did not reach their heart rate peak. During the mass-start road race the cyclists had to respond to transient increases and decreases in work rate that were beyond their control and largely influenced by the course terrain and other cyclists. The mean heart rate, expressed as a percent of maximum heart rate, during the TT_{100}s and the road race were not significantly different (85.4 ± 4.9% vs. 86.9 ± 1.8%). The variability (20.0 ± 3.36% vs. 31.0 ± 0.72%) and peak heart rates (95.2 ± 2.1% vs. 99.2 ± 1.6%) attained during the road race were greater than during the TT_{100} ($P \leq 0.05$). Because of this large variability caused possibly by environmental influences and other cyclists, the road race was performed ~10% slower (i.e. velocity) than the laboratory time trials.

Although the laboratory TT_{100} predicted road racing performance better than VO_{2peak} and W_{peak}, the heart rate responses during the road race suggest that laboratory tests that increase the work rate to maximal outputs for short periods of time interspersed by work rates of submaximal work rate would probably simulate the stochastic nature of road racing better than the conventional ramp tests (chapter 3).

Training and taper in this group of cyclists increased the rates of CHO_{ox} and decreased the rates of FAT_{ox} during the submaximal tests (data not shown). This is in contrast with other researchers (Henriksson 1977; Hurley et al. 1986; MacRae et al. 1995; Westgarth-Taylor et al.

1997; van Loon et al. 1999) who found increased rates of FAT_{ox} and reduced CHO_{ox} rates in well trained and previously less fit subjects after training. The athletes in this study indicated that their food intake had increased during the intense training period, especially their intake of carbohydrate. This temporary alteration in feeding behaviour might have influenced the substrate selection during exercise and increased their rates of carbohydrate oxidation. More research is warranted on the feeding behaviour of athletes during intensive training phases since it probably has an effect on the metabolic rate and the maintenance of body weight.

Cafarelli et al. (1995) investigated the effects of 8 weeks of single-leg endurance cycling training at 60% of VO_{2peak} on muscle activation. Measurements were made during separate 20-min, single –leg rides at 70% of pre-training VO_{2peak}. After training, vastus lateralis EMG activity, plasma lactate, and heart rates were all significantly lower when cycling with the trained or untrained leg. These authors concluded that the endurance training had enhanced the intramuscular environment in the lower limbs by virtue of both central and peripheral mechanisms.

In the current study, the rises in heart rate, plasma lactate concentration and EMG activity during the sub-maximal rides at 30, 60 and 75% of pre-HIT W_{peak} were attenuated after training and taper. The sub-maximal work loads probably represented a slightly lower relative exercising intensity after training and attenuated the recruitment of additional motor units to maintain the same power output. Reduced motor outflow has been shown to reduce the fatigue in the muscle (Cafarelli et al. 1995). We suspect this might cause less metabolic disturbances allowing a maintenance of the muscle conduction velocity and continued recruitment of the same type of muscle fibres. This point is further evident during the high-intensity training sessions where the 8^{th} training session (taper session) performed at the same absolute

intensity as the first session exhibited a reduced heart rate response, EMG activity and reduced frequency shift. The rapid accumulation of metabolic by-products in the muscle during the high-intensity intervals and their contribution in the changes in pH of the sarcolemma of the muscle fibre have been thought to be responsible for slowing down fibre conduction velocity (Lindstrom et al. 1970).

Recently, Trappe et al. (2001) reported significant changes in the contractile properties of human single muscle fibres (increases in diameter, faster shortening velocity and greater force generation) of the type II muscle fibres following taper. Although it is difficult to extrapolate the results from a single cell environment in vitro, the possibility that these changes partly accounted for the changes to the whole muscle functioning exist. In summary, the attenuated metabolic and physiological response to sub-maximal exercise after training is suggestive of an improvement or a more efficient functioning of the physiological system. However the physiological changes of the athletes in this study do not readily explain their ability to sustain higher work rates after HIT. The significance of the reduced left shift and decreased electromyographic activity after HIT is interesting but how it improves performance in these athletes remains to be established.

CHAPTER 7

SUMMARY OF MAIN FINDINGS, LIMITATIONS AND FUTURE DIRECTIONS

The results of the current series of studies firstly, lend support and highlight the need for on-going research on laboratory tests that are more representative of competition performances. Secondly, an account of how elite athletes reduce their training load in preparation for a major competition is qualitatively measured using reliable training diaries and retrospective questionnaires. The subsequent studies on how endurance trained cyclists should taper for a time trial or a road race are based on what athletes are currently doing in the field. Thirdly, these studies have shown that a 14-day taper preceded by a period of high-intensity training "overload" will improve endurance performance in already trained cyclists. A more detailed discussion on the physiological effects of this high-intensity training and taper is provided in the relevant chapters. What follows is a summary of the main findings and then a discussion on the limitations of the current series of studies, as well as an indication of future studies which are warranted as a result of the current findings.

The first major finding was that in both the linear ramp and variable-intensity exercise protocols, the falls in heart rate during the 5 minutes immediately post exercise, better predicted road-racing performance in endurance-trained cyclists than conventional laboratory tests of VO_{2peak} and W_{peak} ($r = 0.93$ vs. 0.75) and lactate concentrations. More rapid heart rate recoveries in better cyclists than in poorer cyclists were presumably due to a faster recovery from fatigue made possible by a decreased promotion of sympathetic drive by muscle afferent nerve activity. This suggests that performances in mass-start road races may not only be related to the cyclists' ability to sustain a given power output but also to recover rapidly from high work rates.

The second major finding is that elite triathletes appear to train in proportion to the relative durations of the three types of exercise in their event. In an Olympic-distance triathlon roughly 50% of the event is spent on the bike, 30% running and 20% swimming (McNaughton 1989). It is not clear if this is an intuitive training strategy or empirically planned. The third major finding was the poor correlation (r = 0.40) between the reduction in training in the two weeks prior to a major competition and personal-best performance in a triathlon. However our investigation also showed that triathletes reduced their training by ~50% over the two weeks before a major competition with reductions in cycling training being the most significant. Interestingly and contrary to scientific guidelines these triathletes reduced training time spent at the hard and very hard intensity during this period.

The fourth major finding of the current study is that the addition of high intensity interval training sessions followed by a taper significantly improved 100-km time trial performance in the laboratory. Six cycling interval training sessions performed at 70% of W_{peak} in addition to endurance training, enhanced endurance cycling performance of these already trained cyclists only when followed by a 50% volume reduction in their HIT training. High-intensity training without taper only succeeded in maintaining pre-training time trial performances. A week taper improved 100-km time trial performance (speed) by ~3.3% while a 2-week taper resulted in the greatest improvements (4.7% - 8%). Furthermore, after a 3-week taper, time-trial performances were marginally better than pre-training values (~ 1.6%) and performances after six weeks of only endurance training were worse than after two weeks of taper.

The fifth major finding was therefore the significant correlation between laboratory 100-km time trial performance and W_{peak} (r = 0.83). The results from previous investigations have shown W_{peak} to be a significant predictor (r = 0.82) of 20-km and 40-km laboratory time-trial

performance (Hawley and Noakes 1992; Lindsay et al. 1996; Westgarth-Taylor et al. 1997; Weston et al. 1997). Surprisingly, W_{peak} did not predict road-racing performance significantly over a distance of 80-km and 104-km in well-trained cyclists.

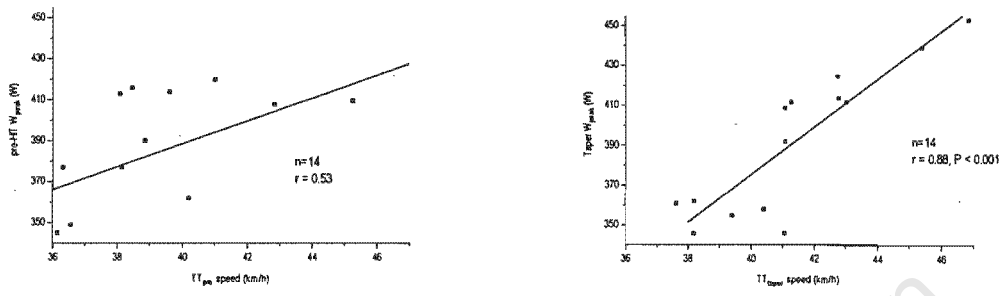


Figure 7.1. The relationship between peak power output (W_{peak}) and TT_{100} performance before HIT (*left panel*) and during taper (*right panel*) for the cyclists in chapters 5 and 6 (n= 14).

A major attribute of we-trained or elite cyclists is the ability to sustain high absolute work rates and maintain a high percentage of their peak power output for prolonged periods of time. The cyclists in the present studies were able to sustain power outputs of ~300W (70% of W_{peak}) during the high intensity training sessions which lasted 60 minutes and during the time trials were able to sustain ~280W (65% of W_{peak}) for over 2 hours. A sixth major finding therefore was the importance of training at a higher intensity than competition or race pace which could possibly have an impact on lactate turnover and metabolite build-up during these high intensity exercise sessions.

In conclusion, the physiological changes associated with the improvements in laboratory cycling performance during taper after 4-6 weeks of HIT were

1) reductions in peak isokinetic strength during maximal voluntary contractions
2) insignificant changes in W_{peak} after training and during taper
3) reduced plasma lactate concentrations, electromyographic activity and frequency shifts and increased carbohydrate oxidation rates during submaximal testing at the same absolute exercise intensities when compared to pre and post training results.

I suspect the physiological rationale for these improvements lies within the contractile property of the muscle fibre. The EMG results provide some insight and suggest an improved and more efficient functioning of the contractile machinery which translates to a resistance to fatigue and a greater power output.

Limitations:

Elite, by definition, is a small population. Recruitment of highly trained subjects for studies that involve invasive procedures or repeated visits to the laboratory is getting more and more difficult. Only in the last 7 years has laboratory and field data from elite (world class) cyclists become available (Palmer et al. 1994; Padilla et al. 1999, 2000; Brosnan et al. 2000). Therefore results based on studies performed on these small populations, with the lack of control groups that are equally well trained or elite, and extrapolation of these findings to the general sporting public should perhaps be interpreted with caution.

Secondly, highly trained athletes are reluctant to partake in studies during competition. Ideally, it would be best to have a larger subject number, and to measure more parameters in several events. Not only is this technically difficult, but has a sig+nificant effect on the performance.

Thirdly, performance tests in the laboratory for endurance events have largely been of the time trial format. Field performance is characterised by an interplay between course terrain, the influence of other competitors and environmental conditions. These variables are presently impossible to simulate in laboratories and thus may have been a limitation of the study.

Future directions:

Heart rate declines with reductions in work rate were far more reliable predictors of mass-start road racing performance than lactate concentrations or VO_{2peak} values. This superior ability of the better cyclists to recover from near exhaustive exercise should be even more evident in the recoveries of EMG power spectra with the reductions in work rates. Unlike heart rates, which are a complex function of the body's milieu and the venous return or stroke volume, EMG spectra are potentially a more direct measurement of muscle recovery. A possibility for future studies therefore would be to examine whether shifts in EMG spectra in intermittent pulse exercise tests might be better predictors of road cycle racing performances than heart rate recoveries.

Another possibility is that HIT increases muscle Na^+/K^+ ATPase capacity. An enhanced re-uptake of K^+ into the muscle after HIT would slow the rises in serum K^+ concentrations with increasing exercise intensity and delay the leftward shifts in the frequency of the power spectrum of electromyographic (EMG) activity towards exhaustion. Leftward shifts in EMG spectra are thought to result from K^+ accumulation in the muscle transverse tubules slowing action potential conduction and impairing the recruitment of additional motor units. Whether improvements after HIT alter EMG activity during exercise and enhances the athlete's ability to recruit additional motor units and delays fatigue, remains to be established.

Although the reliability of the 100-km time trial in the laboratory and the high correlation value to W_{peak} ($r = 0.83$) is encouraging for laboratory predictions of endurance performance, competitive and professional cyclists will not be amenable to testing that is invasive and exhaustive. Multiple linear ramp exercise tests to exhaustion interfere with an athlete's training, while these repeated intermittent pulse exercise tests are not only representative of what athletes do in training but could be incorporated in an athlete's training program. These intermittent pulse exercise tests would allow for more research to be performed on the effects of training on athletic performance. Heart rates have been shown to be a reliable tool for prescribing and monitoring training intensity (Lucia et al. 2000). This author believes non-invasive tests such as heart rate recoveries from submaximal variable intensity tests and electromyographic measurements can play a role in monitoring training status in athletes throughout a competitive season.

Training programmes for improving road cycling performance should evoke multiple adaptations that enable the cyclist to increase the energy production from both aerobic and oxygen-independent pathways and delay the onset of muscular fatigue (Hawley and Stepto 2001).

The literature on the reductions in training prior to a competition or "taper" is limited. To this author's knowledge this is the first study that has examined the effects of taper on endurance performance lasting longer than an hour. The current literature on tapering is also very limited with examples of tapers that have improved performance significantly. Athletic success for athletes at this level is finely balanced while the guidelines on how athletes should taper are still very broad. More research using well trained or elite athletes is warranted to further streamline the guidelines for athletes and coaches.

Conclusion:

These studies indicate that aerobic/endurance fitness may not be the only deciding factor for cycling endurance performance in the field but rather determined by a combination of factors that may include; the ability to recover quickly after periods of high workload, racing experience, tactical ability and sprinting ability. This author strongly suspects that improvements in performance after a short taper period (7- 14 days) which is preceded by a period where training is overloaded allows for adequate recovery time while the benefits of training are maximized.

CHAPTER 8

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CHAPTER 9

APPENDICES

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Appendix 9.1

TRIATHLON TRAINING PROGRAM

Sports Science Institute of South Africa

- The purpose of this diary is to get detailed information about how you are training. We will then be able to advise you about possible ways to improve your training, if need be.
- Fill in the diaries from Monday 17 through to Monday 31 March.
- Follow the example shown in the first page of each section.
- Prepare for the Nationals as you have already planned. Do not change your training simply because you are now being monitored.
- If you are not sure what to do, contact Zuko Kubukeli at 080 3456 789 or John Hawley at (021) 123 4567.
- Bring the COMPLETED diary with you when you visit the Sports Science Institute on Thursday 3 April. DON'T FORGET!

Name: _____ Phone: _____

Address: _____

APPENDIX 9.2 - SWIMMING DIARY

athlete: [| | S] 1 2 3

Use one row of boxes for each set of reps.
Include any warm up and warm down as separate sets.
Fill in the Brick box **only** to indicate session(s) done immediately before the current session. Otherwise leave blank
Put any comments in the box at the bottom of the page. Link the comments to the sessions.

- Show the date and time only for the first rep of the session.
- C = cycle before R = run before B = both before
- A = AM P = PM
- 1 = freestyle 2 = butterfly 3 = backstroke 4 = breastroke 5 = other (specify in Comments)
- V = Very hard H = Hard M = Moderate E = Easy
- Show heart rates only if you wore a monitor. Show the range for most of the set. Don't count heart rates during rest intervals.

day	month	year	time A or P	Brick session	stroke	no. of reps		distance for each rep (m)	average time for each rep (min:sec)	ON (min:sec)	effort VHME	heart-rate range		
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	

day	month	year	time A or P	Brick session	stroke	no. of reps		distance for each rep (m)	average time for each rep (min:sec)	ON (min:sec)	effort VHME	heart-rate range		
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	

day	month	year	time A or P	Brick session	stroke	no. of reps		distance for each rep (m)	average time for each rep (min:sec)	ON (min:sec)	effort VHME	heart-rate range		
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	

day	month	year	time A or P	Brick session	stroke	no. of reps		distance for each rep (m)	average time for each rep (min:sec)	ON (min:sec)	effort VHME	heart-rate range		
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	
1 2	3 4	5 6	7	8	9	10 11		12 13 14 15	16 17 18	19 20 21	22	23 24 25		26 27 28
							X		:	:			-	

Comments

APPENDIX 9.3 -CYCLING DIARY

athlete: C (1 2 3)

Use one row of boxes for each bout of continuous cycling or each set of reps.
Include any warm up and warm down as separate sets.
Fill in the Brick box **only** to indicate session(s) done immediately before the current session. Otherwise leave blank
Put any comments in the box at the bottom of the page. Link the comments to the sessions.

Show the date and time only for the first bout or rep of the session.

R = run before S = swim before B = both before

A = AM
P = PM

1 = racing bike 2 = wind/turbo/erg
3 = mountain bike 4 = motor pacing

V = Very hard
H = Hard
M = Moderate
E = Easy

Show heart rates only if you wore a monitor. Show the range for most of the set. Don't count heart rates during rest intervals.

day		month		year		time A or P	Brick session	training type
1	2	3	4	5	6	7	8	9

Fill in these for continuous cycling:

time to complete ride (min)			distance covered (km)		
20	21	22	23	24	25

effort VHME	heart-rate range					
26	27	28	29 –	30	31	32

Fill in these for a set of intervals:

no. of reps		X	average speed (km/h) for each rep (if known)		average time for each rep (min:sec)			ON (min:sec)		
10	11	X	12	13	14	15	16	17	18	19

day		month		year		time A or P	Brick session	training type
1	2	3	4	5	6	7	8	9

Fill in these for continuous cycling:

time to complete ride (min)			distance covered (km)		
20	21	22	23	24	25

effort VHME	heart-rate range					
26	27	28	29 –	30	31	32

Fill in these for a set of intervals:

no. of reps		X	average speed (km/h) for each rep (if known)		average time for each rep (min:sec)			ON (min:sec)		
10	11	X	12	13	14	15	16	17	18	19

day		month		year		time A or P	Brick session	training type
1	2	3	4	5	6	7	8	9

Fill in these for continuous cycling:

time to complete ride (min)			distance covered (km)		
20	21	22	23	24	25

effort VHME	heart-rate range					
26	27	28	29 –	30	31	32

Fill in these for a set of intervals:

no. of reps		X	average speed (km/h) for each rep (if known)		average time for each rep (min:sec)			ON (min:sec)		
10	11	X	12	13	14	15	16	17	18	19

day		month		year		time A or P	Brick session	training type
1	2	3	4	5	6	7	8	9

Fill in these for continuous cycling:

time to complete ride (min)			distance covered (km)		
20	21	22	23	24	25

effort VHME	heart-rate range					
26	27	28	29 –	30	31	32

Fill in these for a set of intervals:

no. of reps		X	average speed (km/h) for each rep (if known)		average time for each rep (min:sec)			ON (min:sec)		
10	11	X	12	13	14	15	16	17	18	19

Comments

APPENDIX 9.4 -RUNNING DIARY

athlete R (1 2 3)

Use one row of boxes for each bout of continuous running or each set of reps.
Include warm up and warm down as separate sets.
Fill in the Brick box **only** to indicate session(s) done immediately before the current session. Otherwise leave blank
Put any comments in the box at the bottom of the page. Link the comments to the sessions.

Show the date and time only for the first bout or rep of the session.

C = cycle before S = swim before B = both before

A = AM P = PM

1 = road 2 = track 3 = treadmill 4 = hills 5 = other (specify in Comments)

whole numbers (no decimals)

V = Very hard H = Hard M = Moderate E = Easy

Show heart rates only if you wore a monitor. Show the range for most of the set. Don't count heart rates during rest intervals.

day (1 2) month (3 4) year (5 6) | time A or P (7) | Brick session (8) | training type (9)

Fill in these for continuous running: time to complete run (min) (22 23 24) | distance covered (km) (25 26) | effort VHME (27) | heart-rate range (28 29 30 - 31 32 33)

Fill in these for a set of intervals: no. of reps (10 11) X distance for each rep (m) (12 13 14 15) | average time for each rep (min:sec) (16 17 18) | rest between reps (min:sec) (19 20 21)

day (1 2) month (3 4) year (5 6) | time A or P (7) | Brick session (8) | training type (9)

Fill in these for continuous running: time to complete run (min) (22 23 24) | distance covered (km) (25 26) | effort VHME (27) | heart-rate range (28 29 30 - 31 32 33)

Fill in these for a set of intervals: no. of reps (10 11) X distance for each rep (m) (12 13 14 15) | average time for each rep (min:sec) (16 17 18) | rest between reps (min:sec) (19 20 21)

day (1 2) month (3 4) year (5 6) | time A or P (7) | Brick session (8) | training type (9)

Fill in these for continuous running: time to complete run (min) (22 23 24) | distance covered (km) (25 26) | effort VHME (27) | heart-rate range (28 29 30 - 31 32 33)

Fill in these for a set of intervals: no. of reps (10 11) X distance for each rep (m) (12 13 14 15) | average time for each rep (min:sec) (16 17 18) | rest between reps (min:sec) (19 20 21)

day (1 2) month (3 4) year (5 6) | time A or P (7) | Brick session (8) | training type (9)

Fill in these for continuous running: time to complete run (min) (22 23 24) | distance covered (km) (25 26) | effort VHME (27) | heart-rate range (28 29 30 - 31 32 33)

Fill in these for a set of intervals: no. of reps (10 11) X distance for each rep (m) (12 13 14 15) | average time for each rep (min:sec) (16 17 18) | rest between reps (min:sec) (19 20 21)

Comments

APPENDIX 9.5 - OTHER-TRAINING DIARY

athlete

1 2 3 T

Use one row of boxes for each type of workout.
Include warm up and warm down as separate types.
Put any comments in the box at the bottom of the page. Link the comments to the sessions.

Show the date and time only for the first rep of the session.

A = AM
P = PM

1 = weights 2 = swimbench 3 = cords 4 = deep-water running
5 = shallow-water running 6 = other (specify in Comments)

1 = legs 2 = arms 3 = abdominals
4 = back 5 = most muscles

V = Very hard
H = Hard
M = Moderate
E = Easy

Show heart rates only if you wore a monitor. Show the range for most of the set. Don't count heart rates during rest intervals.

day | month | year | time A or P | type of session | muscles | duration of workout (min) | effort VHME | heart-rate range

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

day | month | year | time A or P | type of session | muscles | duration of workout (min) | effort VHME | heart-rate range

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

day | month | year | time A or P | type of session | muscles | duration of workout (min) | effort VHME | heart-rate range

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

day | month | year | time A or P | type of session | muscles | duration of workout (min) | effort VHME | heart-rate range

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 - 17 18 19

Comments

APPENDIX 9.6 -YOUR RECENT TRIATHLON TRAINING

athlete (1 2)

There's been a round of major competitions lately (e.g. African and South African Championships). We want to find out about your training in the 4 weeks leading up to the first competition.

leave blank Q2 Q4 (5 6)

1. How many weeks before the the S.A. Championships was the first of these major competitions for you? (3 4)

Now, let's examine your training in the month before that first competition.

2. Did anything prevent you from training the way you would normally train (e.g. injury, flu)?

Tick: No ◯

Yes ◯ If YES, what was the problem: _____

Please tell us about the training you would *normally* have done if you didn't have this problem.

3. Write down the training hours per week you were doing in the first week of that month (that is, 28-21 days before the first competition). Count warm-ups and cool-downs as easy sessions. For interval training, keep the work periods separate from the recovery (easy/rest) periods. Convert minutes to hours as follows: 10 min = 0.2, 20 min = 0.3, 30 min = 0.5, 40 min = 0.7, 50 min = 0.8

	continuous training (hours per week)				interval training (hours per week)			
	very hard	hard	moderate	easy	very hard	hard	moderate	easy/rest
swimming	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)
cycling	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)
running	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)

4. This diagram shows the 4 weeks before the first competition. Circle the hardest training week. — 4 — 3 — 2 — 1 —

5. What training did you do in that week? (Skip this question if you circled Week 4.)

	continuous training (hours per week)				interval training (hours per week)			
	very hard	hard	moderate	easy	very hard	hard	moderate	easy/rest
swimming	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)
cycling	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)
running	(1 2 3)	(4 5 6)	(7 8 9)	(10 11 12)	(13 14 15)	(16 17 18)	(19 20 21)	(22 23 24)

Finally, some personal details:

age (1 2) weight (kg) (3 4 5) height (cm) (6 7 8) years as a competitive *tri*athlete (9 10) years as a competitive athlete (11 12)

Triathlon PB for 1500-m swim (min:sec) (13 14 15 16) Triathlon PB for 40-km ride (min:sec) (17 18 19 20) Triathlon PB for 10-km run (min:sec) (21 22 23 24)

Appendix 9.7

Electromyographic activity analysis

The language used by the programmers in writing the program is quite simply and easy to follow. The writing in bold are commands that the program could recognise. There are three parts that have to be followed in order to get the required answers and analyses.

PART 1

function [] = poo(file01, file02, file03, file04, file05, file06, file07, file08, file09, file10, file11, file12, file13, file14, file15, file16, file17, file18, file19, file20, file21)

usage: poo file.pdf ≤file2.pdf> ≤file3.pdf> ≤file4.pdf>...≤file21.pdf>

POO is great. It has, however, been written without any definite header information. Therefore it is only recommended to run this utility on data that you know to have been sampled at 1984Hz. (The program should nonetheless tell you if the EMG has not been sampled at this rate. First you will be prompted for the length of data in seconds that you wish to process.

POO then plots the EMG data contained in file.pdf. Next, you will be asked to select an area of interest on the graph for processing. The left hand side of the graph will be the start of the time period that you specified for processing. Keep an eye on the graph's title - it will give timing information. The button on the bottom left can be used to see the data not on screen that will be selected when 'return' is pressed.

Alternatively one can just enter the start time in seconds at the prompt.

POO will then plot the selected raw EMG, the filtered EMG and the EMG spectrum.

If a second filename is specified, the process will repeat for that file, and the frequency shift of the second EMG selection will be expressed as a percentage of the first. Note that the same sample length of data will be taken from the second file as from the first. If you want to compare data from different parts of the same file, enter the filename twice on the command line argument.

If more files are specified, the spectral shift of these files will be expressed in terms of the frequency spectra of the first file.

In other words,

THE FIRST EMG FILE SHOULD CONTAIN THE NORMALISATION EMG DATA

A table of ratios of pertinent data is then outputed to the screen and to an ascii file.

The default file name is results.txt and is in the root directory. i.e. c:\results.txt

Filtering Information (as by zegait.m):

The signal's mean is removed.

A 2nd 3dB @ 15Hz Butterworth high pass filter is used to remove the low frequency motion artifact. Note that this filter was used for gait cycle EMG. For isometric work a lower cutoff frequency is advisable but not necessary.

The signal is then rectified.

The signal's amplitude envelope is calculated using a 2nd 3dB @ 5Hz Butterworth low pass filter.

Finally the data is subsampled

names(1,:) = 'file01';

names(2,:) = 'file02';

names(3,:) = 'file03';

names(4,:) = 'file04';

names(5,:) = 'file05';

names(6,:) = 'file06';

names(7,:) = 'file07';

and so on...

SUBTLETY: The signals are filtered both ways through the digital filters. This means that all the signal's frequencies will travel through the filter at the same speed, leading to no distortion. This also doubles the order of the filters, which means that the 3dB cut-off points of the filtering have been moved to about 18 and 4 Hz for the high and low pass filters respectively.

Open the files - check that they are all real - and don't reopen any files

for i = 1:nargin

filename = deblank(filez(i,:));

```
if(i==1)
fid(i) = fopen(filename,'r');

elseif(strcmp(filez(i,:),filez(i-1,:)));
fid(i) = fid(i-1);
else
fid(i) = fopen(filename,'r');
end
end
```

There are a few commands which will prompt the user when errors or incorrect commands are entered. The program then points out the error by displaying messages.

disp(' ') means display to the user;

[ ] is where the user will type in their commands

disp('Remember: Flexcomp pdf files are stored in directories named the same as their reference number.')

disp('The first number is the recording session, the second number is the channel number.');

disp('Example: c:\flexpat\fred01\0003_02.pdf -> Patient fred01, session 3, channel 2 data.');

Jump in 29 bits into the file and read what I believe to be the sampling rate marker

```
fseek(fid(i),28,'bof');
temp = fread(fid(i),6,'int8') - [0 8 33 4 58 0]';
```

```
if(sum(abs(temp)))
disp error('This file does not appear to have been sampled at 1984Hz.  Exiting...')
end
```

Jump in 150 bits into the file from its start

```
fseek(fid(i),150,'bof');
```

Read in the data. That big long number converts from ADC values to volts. It was also determined empirically by comparing max/min values with that provided by the flexcomp program.

e.g. EMGdata = 0.20757511668611e-6 * fread(fid(i),'short');

Find out the total number of samples, along with other info and output to screen.

```
datalen(i) = length(EMGdata);
datamax(i) = max(EMGdata);
datamin(i) = min(EMGdata);

disp(['File no. ', num2str(i),' (', deblank(filez(i,:)),'), samples: ',num2str(datalen(i)),', EMG
Max: ',num2str(datamax(i)),'V, EMG Min: ',num2str(datamin(i)),'V'])
```

Create a time vector for plotting

```
time = (0:1/1984:(datalen(i)-1)/1984)';
```

timemax(i) = max(time);

Plot out the data, each one on a separate figure. The figures are plotted through the MATLAB 4 program.

figure

plot(time,EMGdata)

xlabel('time (secs)')

ylabel('amplitude (volts)')

title(['File no. ',num2str(i),': ',filez(i,:)])

set(gca,'XLim',[0 timemax(i)])

end

Here we clear all variables except the things we need. We do this out of memory

Important : clear EMGdata time temp ans global tselection i time EMGdata timemax

Loop through all the figures

for i = 1:nargin

Load back in the EMG from the current file

fseek(fid(i),150,'bof');

EMGdata = 0.20757511668611e-6 * fread(fid(i),'short');

time = (0:1/1984:(datalen(i)-1)/1984)';

The setting-up run, where the amount of data (in seconds) to be processed is decided.

Prompt for the user and all comments marking statements executed when i==1 only are marked N.

if(i==1)

N Blurb for user

disp('The file(s) have been displayed on separate figures. Specify the amount of data in seconds from the left hand side of the screen that you wish to process. Maximum time allowed is the shortest EMG data set.')

disp('Hitting 'return' will select just the displayed EMG for processing.')

tselection = input(['ENTER TIME IN SECONDS (max ',num2str(min(timemax)),']:

N Check the selection time is not too large here

while (tselection > min(timemax))

disp(' ')

```
tselection = input(['Time specified too large.  Enter number (max
',num2str(min(timemax)),')  (ctrl-c to quit): ']);
end

N  Decide which mode we are going into...fixed time or screen selection
if(isempty(tselection))
mode = 0;

disp('Only plotted data will be processed')
else
mode = 1;
disp(['Now processing file number ',num2str(i),' called ',filez(i,:)])
disp([num2str(tselection), ' second(s) of data will be processed starting from left hand side of
plot.'])
end

N Close down all the figures and start selecting the start (and if appropriate) the
data end-points
close all

N Plot the EMG channel
figure;
plot(time,EMGdata)
ylabel('amplitude (V)');
xlabel('time (s)');
```

```
set(gca, 'XLim', [0, timemax(i)]);
```

N Now a routine for data selection - the tricky stuff is just to give data on cursor position in the title of plot and the small buttons on the bottom

```
we_are_not_happy = 1;
while(we_are_not_happy)
zoom xon
zoom(1)
set(gcf,'units','pixels');
grid
```

disp('Click and drag so that the left hand side of the displayed data corresponds to the desired start of the EMG selection. Right mouse button zooms out.')

```
set(gcf,'WindowButtonMotionFcn', 'axislimits=get(gca,"XLim");mainscreen =
get(0,"PointerLocation");thefigure = get(gcf,"Position");jiggerypokery1 = [mainscreen(1)
- thefigure(1),mainscreen(2) -
thefigure(2)];jiggerypokery2=get(gca,"CurrentPoint");title([num2str(axislimits(1))," to
",num2str(axislimits(2))," secs displayed, now at ",num2str(jiggerypokery2(1))."
secs."])');
if(mode)
uicontrol('String','View Proposed Data','Position',[20, 5, 140,
20],'Callback','v=get(gca,"XLim");global tselection;set(gca,"XLim",[v(1) (v(1) +
tselection)]);');
```

```
uicontrol('String','Reset Plot','Position',[160, 5, 100, 20],'Callback','global time EMGdata
timemax i;plot(time,EMGdata);set(gca,"XLim",[0 timemax(abs(i))]);zoom xon;grid
on;ylabel("amplitude (V)");xlabel("time (s)");set(gcf,"WindowButtonMotionFcn",
"axislimits=get(gca,""XLim"");mainscreen = get(0,""PointerLocation"");thefigure =
get(gcf,""Position"");jiggerypokery1 = [mainscreen(1) - thefigure(1),mainscreen(2) -
thefigure(2)];jiggerypokery2=get(gca,""CurrentPoint"");title([num2str(axislimits(1)),"" to
"",num2str(axislimits(2)),"" secs displayed, now at "",num2str(jiggerypokery2(1)),""
secs.""])");');
end

if(mode)
choosetime = input('Press return when satisfied, or enter a time:  ');
else
disp(['Press 'return' when satisfied'])
pause
end

N  Now find out what data was selected
N  This just gets the start and stop of the x-axis on the plot
vv(:,i) = get(gca, 'XLim')';
if(~isempty(choosetime))
vv(1,i) = choosetime;
```

```
end

N  Change the limits if a specific amount of time was selected
if(mode)
vv(2,i) = vv(1,i) + tselection;
end

if (vv(1,i)>timemax(i))

disp('The specified start time is after the end of the data sequence')

N  Check that in the case of screen selection mode that the time selected is less
than the smallest data sequence
elseif(vv(2,i)-vv(1,i)>min(timemax))

disp(['The screen selected data is longer than an entire other EMG file of
',num2str(min(timemax)),' secs.'])
disp('Please select a shorter data sequence.')

N  Check that the calculated end point is within the data sequence
elseif (vv(2,i) > max(timemax(i)))

disp('The ending time of the selected data sequence is outside the range of the data.')
if(mode)
choosetime = [];
```

```
disp('Please select an earlier start to the data sequence.');
else
disp('Please select an earlier start to the data sequence.')
end
else
we_are_not_happy = 0;
end

end  end of while loop
```

If we are in plotted data only mode, determine the tselection time for further files

```
if((~mode)&(i==1))
tselection = vv(2,i)-vv(1,i);
end
```

Close the figure

```
close
```

That's the end of the set-up where the time of processed data is decided and the normalising data is obtained.

PART 2

We have a general setup now to process the files that contain the data to be normalised.

All comments in this section are marked by D. It is essentially the same process with different error checking and no time selection.

```
else
D  Blurb for user

disp(['Now processing file number ',num2str(i),' called ',filez(i,:)])
disp([num2str(vv(2,1)-vv(1,1)), ' second(s) of data will be processed.'])

D  Plot the EMG channel
figure;
plot(time,EMGdata)
title(['Recorded EMG data in file ', num2str(i),': ',filez(i,:),'.'])
ylabel('amplitude (V)');
xlabel('time (s)');
set(gca, 'XLim', [0, timemax(i)]);


D  Now a routine for data selection - the tricky stuff is just to give data on cursor
position in the title of plot and again, that small button
we_are_not_happy = 1;
while(we_are_not_happy)
zoom xon
zoom(1)
set(gcf,'units','pixels');
```

```
grid
disp(' ')
disp('Click and drag so that the left hand side of the displayed data corresponds to the desired start of the EMG selection.  Right mouse button zooms out.')
disp('Press return when satisfied.')
set(gcf,'WindowButtonMotionFcn', 'axislimits=get(gca,"XLim");mainscreen = get(0,"PointerLocation");thefigure = get(gcf,"Position");jiggerypokery1 = [mainscreen(1) - thefigure(1),mainscreen(2) - thefigure(2)];jiggerypokery2=get(gca,"CurrentPoint");title([num2str(axislimits(1))," to ",num2str(axislimits(2))," secs displayed, now at ",num2str(jiggerypokery2(1))," secs."])');
uicontrol('String','View Proposed Data','Position',[20, 5, 140, 20],'Callback','v=get(gca,"XLim");global tselection;set(gca,"XLim",[v(1) (v(1) + tselection)]);');
uicontrol('String','Reset Plot','Position',[160, 5, 100, 20],'Callback','global time EMGdata timemax i;plot(time,EMGdata);set(gca,"XLim",[0 timemax(abs(i))]);zoom xon;grid on;ylabel("amplitude (V)");xlabel("time (s)");set(gcf,"WindowButtonMotionFcn", "axislimits=get(gca,""XLim"");mainscreen = get(0,""PointerLocation"");thefigure = get(gcf,""Position"");jiggerypokery1 = [mainscreen(1) - thefigure(1),mainscreen(2) - thefigure(2)];jiggerypokery2=get(gca,""CurrentPoint"");title([num2str(axislimits(1)),"" to "",num2str(axislimits(2)),"" secs displayed, now at "",num2str(jiggerypokery2(1)),"" secs.""])");');
pause
```

```
Now find out what data was selected


This just gets the start and stop of the x-axis on the plot
vv(:,i) = get(gca, 'XLim')';


A time selection is at this stage always specified
vv(2,i) = vv(1,i) + tselection;


Check that the calculated end point is within the data sequence
if (vv(2,i) > max(timemax(i)))
disp(' ')
disp('The ending time of the selected data sequence is outside the range of the data. Please
select an earlier start to the data sequence.')
else
we_are_not_happy = 0;
end



end of while loop


Close the figure
close
```

PART 3

END OF FIRST FILE/OTHER FILE DIVISIONS

We then take the raw data - this business of adding two very small irrational numbers to both limits is to make the likelihood of selecting exactly a sampling time as the start time of the data selection, which will result in that time having one more sample and messing up the subsequent analysis. This can happen if one chooses zero time as the start of the EMG data selection.

```
index = find((time<=vv(2,i)+pi/3000000)&(time>=vv(1,i)+pi/3000000));
```

```
EMG = EMGdata(min(index):max(index));
```

So far so good, we have selected our data - let's process it, one at a time (we are still in the original filename loop at the very top.

Plot the raw EMG in the top of the figure

```
figure
subplot(3,1,1); plot(time(min(index):max(index)),EMG)
title(['Raw Selected EMG']);
ylabel('amplitude (V)');
set(gca,'XLim',vv(:,i));
```

Then get the linear envelope and find the area under the curve and RMS

We process using the zegait.m file algorithms. The following is a slight modification of that file

Start of zegait.m

Low pass filter characteristics

nfiltl=2;

wcl=5/(1984/2);

High pass filter characteristics

nfilth=2;

wch=15/(1984/2);

Beginning the algorithm

y=EMG-mean(EMG); removing the mean

Here it can be high pass filtered

[b,a]=butter(nfilth,wch,'high');

y = filtfilt(b,a,y);

RMS(i) = sqrt(mean(y.^2));

y=abs(y); rectifying the signal

Low pass filter

[b,a]=butter(nfiltl,wcl);

Be sure to filter in both directions to make sure the filtered data has zero phase.

Make a data vector properly pre- and ap- pended to filter forwards and back

so end effects can be obliterated.

y = filtfilt(b,a,y);

yout = y(1:10:length(y));

tout = time(min(index):10:max(index));

End of zegait.m

Plot the envelope

subplot(3,1,2); plot(tout,yout);

title('Filtered selected EMG');

ylabel('amplitude');

set(gca,'XLim', vv(:,i));

Calculate the area under the sampled curve

area = 0;

```
for nn = 1:size(tout)-1;
arrea = arrea + (tout(nn+1)-tout(nn)) * ( yout(nn) + (yout(nn+1)-yout(nn))/2);
end
area(i) = arrea;
```

Output this info to the screen

```
disp(['RMS is ', num2str(RMS(i)), '.'])
```

Finally plot the frequency spectrum and calculate the cumulative power spectrum

Now we calculate the frequency spectrum of our selected data

```
fEMG=fft(EMG);
```

We plot the fft data. All this code just makes sure that the correct frequency axis is displayed. See the Matlab demo to find out more.

```
fEMG(1) = 0;
n = length(fEMG);
if(rem(n,2)~=0)
n = n - 1;
end
```

Note that the amplitude variable stores the fft amplitude information for each file in the command line argument.

```
amplitude(:,i) = abs(fEMG(1:n/2));
```

```
nyquist  = 1984/2;
freq = (1:n/2)'/(n/2)*nyquist;
subplot(3,1,3); plot(freq, amplitude(:,i))
title('Selected EMG frequencies');
ylabel('amplitude');
set(gca,'XLim', [freq(1) freq(length(freq))]);
xlabel('frequency (Hz)');
```

Here we reclear all the data which may affect the next run through. We hold on to the frequency data if there are files to be compared later

```
clear global EMGdata
clear global time
global time EMGdata
```

End of all the file looping - NO LOOPS FROM HERE ON OUT

```
if (nargin>1)
```

Using the cumulative power spectra, find the spectral shift for each file in comparison with the first

This is the corrected version of the zig.m file.

Find the length of the data streams up to 500, 100 and 350 Hz respectively in

the amplitude spectra - this will be the same for all files

```
h500 = round((n/2)*500/(1984/2));

h100 = round((n/2)*100/(1984/2));

h350 = round((n/2)*350/(1984/2));
```

Calculate the cumulutative amplitude spectrum for all data samples

```
for count = 1:nargin

totalpowerin500Hz = sum(amplitude(1:h500,count));

for nn = 1:h500

q(count,nn) = sum(amplitude(1:nn,count))/totalpowerin500Hz;

end
```

Now for 100 Hz to 350 Hz in the first data file, find the corresponding frequencies in the other data files below which the same amount of amplitude is contained

```
for count = 1:nargin;

for nn = 1:h350-h100

[c,d] = min(abs(q(1,nn+h100-1)-q(count,:)));

k(nn,count) = d*(1984/2)/(n/2);

end
```

Finally divide these frequencies by the original frequencies to find by how much they have shifted (on average) in the range 100 to 350 Hz

```
f = (h100:h350-1)*(1984/2)/(n/2);
for count = 1:nargin
ratio(count) = mean(k(1:h350-h100,count)./f');
end
```

This is end the corrected version of the zig.m file.

Output all the data in tabular format to the workspace

```
disp('Basic Normalised Results')
disp(['Normalisation file (', deblank(filez(1,:)), '),  RMS:  ', num2str(RMS(1)/RMS(1)),',
frequency compression:  ', num2str(100-ratio(1)*100),'%.'])
for nn = 2:nargin
disp(['Data file no. ',num2str(nn-1),' (', deblank(filez(nn,:)), '),  RMS:  ',
num2str(RMS(nn)/RMS(1)),',  frequency compression:  ', num2str(100-ratio(nn)*100),'%.'])
end
```

Output all this data to an ascii file and more

```
outputn  = input('Specify a file name for the detailed summary
information (default = c:\results.txt): ','s');

time = clock;
hours = num2str(time(4));
mins = num2str(time(5));
```

```
if(isempty(outputn))
outputn = 'c:\results.txt';
end

fidd = fopen(outputn,'w');

fprintf(fidd,'POO Ver 2.1 Flexcomp EMG Summary Information.');
fprintf(fidd,['\nThis file was created on ', date, ' at ', hours,':',mins,'hrs.']);
fprintf(fidd,'\n\nNormalisation file: %s, data taken from %f to %f
secs.',deblank(filez(1,:)),vv(1,1),vv(2,1));
for nn = 2:nargin
fprintf(fidd,'\nComparison file no. %d: %s, data taken from %f to %f secs, normalised
RMS %f , frequency compression %f %%.',nn-
1,deblank(filez(1,:)),vv(1,nn),vv(2,nn),RMS(nn)/RMS(1),100-ratio(nn)*100);
end
```

That's the end of the comparisons and outputting in general

Close everything

```
clear all;
clear global;
fclose('all');
```

Appendix 9.8

Muscle enzyme and biopsy experiment

Introduction

The recruitment of highly trained or elite subjects for studies that involve invasive procedures is an increasingly difficult task. Of the 10 subjects we successfully recruited for the study, 6 complied with the strict requirements of the study and completed the 12-week program. Of those 6 only 2 agreed to have muscle biopsies taken during various stages of the study. The procedures and results of that data are presented below. All the muscle enzyme and biopsy data was obtained during the subjects' visits for the submaximal testing namely; PRE- and POST- high-intensity training, during TAPER and after 6-WEEKS of habitual training.

Methods

Muscle biopsy

A needle biopsy of the vastus lateralis muscle was obtained using the technique described by Bergstrom et al. (1962) following administration of a local anaesthetic, as described by Evans et al. (1982). A small incision was made prior to insertion of the biopsy needle. The biopsy sample was immediately divided into two portions. Half was immediately frozen in liquid nitrogen and stored at –70^{0}C for later enzyme assays. The remaining portion was orientated and embedded in Tissue-tek and frozen in n-Pentane cooled in liquid nitrogen and stored at -20^{0}C for later histological analyses.

Histology

Routine histology was carried out using myosin ATPase stain at pH 4.3, 4.6 and 9.4 to differentiate type I and type IIA and IIB fibres (Dubowitz, 1973). All the fibres viewed in one microscope field at a magnification of 10x were counted to calculate the relative proportions of fibre types.

Muscle enzyme activity

Muscle samples were homogenised on ice in a phosphate buffer [100 mmol.L-1 potassium phosphate, 2 mmol.L-1 ethylenediamine-tetra-acetic acid (EDTA), 5 mmol.L-1 $MgCl_2$ and 20 mmol.L-1 2-mercaptoethanol, pH 7.5]. Homogenates were then sonicated on ice for 3 x 10 s (Heat systems, Ultrasonics), divided into four aliquots and assayed for phosphofructokinase (PFK), hexokinase (HK), citrate synthase (CS) and 3-hydroxyacyl CoA dehydrogenase (3-HAD) activities as described in the methods of Ling et al. (1965), Bass et al. (1969) and Srere et al. (1969). All enzymes assays were performed in duplicate at 25^{0}C using a spectrophotometer (Beckman DU-62, Beckman Instruments, USA). Results are expressed relative to protein concentration. Protein concentrations were assayed in triplicate by the Biuret method (Kingsley 1942).

Muscle buffering capacity

Muscle buffering capacity (βm) was measured from the acid required to titrate the pH of muscle homogenates over a physiological range of pH 7.1 to 6,5 as described by Weston et al. (1997) and Marlin and Harris (1991). All muscle sample (6.5-13.8 mg wet wt.) homogenates were assayed on the same day and in random order that was unknown to the investigator. The coefficient of variation for βm_{titr} measurements in this laboratory is ± 3.0% (Weston et al. 1996) and is comparable to that reported elsewhere (Costill et al. 1982; Mannion et al. 1993). Results for the muscle buffering capacity are expressed per full pH unit for final expression as µmol H^+. g protein dw^{-1}. pH^{-1} and µmol H^+ (units). g muscle for the skeletal muscle enzyme activity.

No statistical analyses could be performed on this data because of the small sample size. Results are therefore presented for each subject and the results describe the trends that were evident from these two sample analyses.

Results

The physiological and performance characteristics of the subjects are detailed in Chapter 6. The effects of the training intervention and 6 weeks of habitual training on their time trial performance is also presented in Chapter 6, however, the effects of the training, taper and 6 weeks of habitual training on maximal glycolytic enzyme activity are presented in Figure 1A and B. During the taper the activity of HK more than doubled in both subjects when compared to pre- and post-HIT training values and after 6 weeks of habitual training. In contrast, the activity of PFK was at its lowest in both subjects during the taper and peak values were observed pre-HIT training and after 6-weeks of habitual training.

The oxidative enzyme activities are presented in Figure 1C and D. The training intervention and 6 weeks of habitual training did not have a notable effect on the activity of CS or 3-HAD. The activity of these enzymes did not vary by more than 38% throughout the study when it was measured in both subjects.

Skeletal muscle buffering capacity was marginally at its peak in both subjects during the taper (Fig. 2) and muscle fibre type distribution (%) was not changed by the training intervention in these two subjects (Table 1).

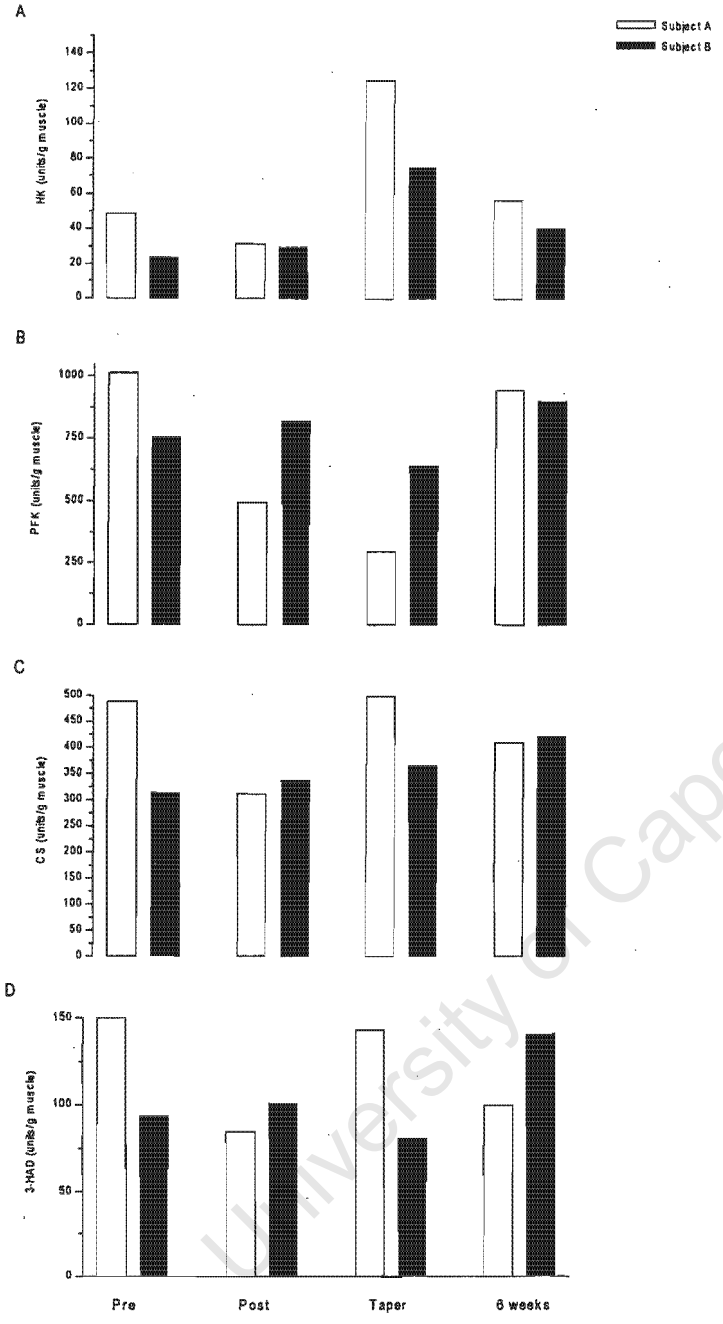

Figure 1. Maximal glycolytic (A- Hexokinase, B- Phosphofrictokinase) and oxidative (C- Citrate synthase, D- 3-Hydroxyacyl CoA dehydrogenase) enzyme activities pre- and post- HIT training, during taper and after 6 weeks of habitual training.

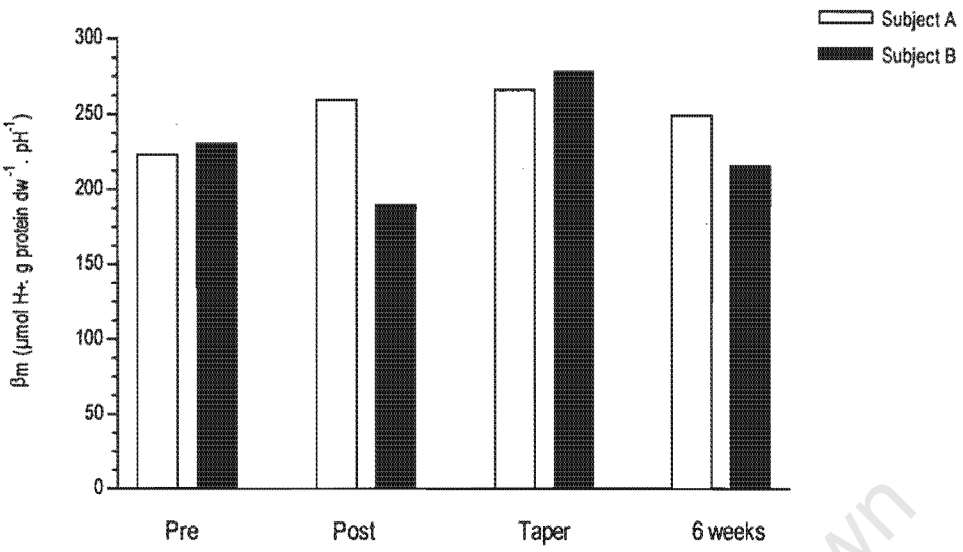

Figure 2. Skeletal muscle buffering capacity (βm) pre- and post- HIT training, during taper and after 6 weeks of habitual training.

Table 1. Muscle fibre-type distribution, taken from the vastus lateralis, pre-and post-training, during taper and after 6 weeks of habitual training.

		Pre-	Post-	Taper	6 weeks
% Type I -	Subject A	67	55	64	53
	Subject B	52	56	52	60
% Type IIA-	Subject A	29	41	33	46
	Subject B	46	39	44	39
% Type IIB-	Subject A	3	4	1	1
	Subject B	0	5	0	1
% Type IIC-	Subject A	0	0	2	0
	Subject B	1	0	4	0

Conclusion

Conclusions can not be made based on this sample size, however chapter 2.2 of the review of the literature and Hawley and Stepto (2001), provide some guidelines on these results. Interval training has been shown to increase the activity of one or more of the muscle glycolytic enzymes in previously untrained individuals (Table 2.1) while a number of studies of well-trained subjects (Sjodin et al. 1982; Costill et al. 1988 and Weston et al. 1997) have shown no increases in oxidative or glycolytic enzyme activities after interval training. In this study, albeit only two subjects, training seems to have had a perplexed effect on the glycoltyic enzyme capacity. The current research on the effects of interval training on muscle enzyme activity in well-trained subjects is not sufficient and further investigation is warranted with larger subject numbers.

Three studies to our knowledge have reported interval training to significantly increase muscle buffering capacity in previously untrained (Sharp et al. 1986; Bell and Wenger 1988) and well trained (Weston et al. 1997) subjects. A high muscle buffering capacity is traditionally associated with good sprinting performance and sprint-trained individuals have generally been shown to have a higher muscle buffering capacity than endurance-trained or sedentary subjects (Sahlin and Henriksson 1984; Parkhouse et al. 1985). The inconsistent changes in fibre type distribution after training from several studies are well within the ~12% coefficient of variation of fibre type determinants in needle biopsies from the same muscle as measured by Blomstrand and Ekblom (1982). The findings currently available in the literature have to be interpreted with caution and this point is further elaborated on in Chapter 2.2 of the review of the literature.