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THE PHYSIOLOGICAL TESTING OF

SENIOR AND VETERAN CYCLISTS

BY

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ABSTRACT

In order to gain a clearer understanding of factors which influence the physiological testing of senior and veteran cyclists this thesis:- assessed the validity and reproducibility of the Kingcycle ergometer; the reproducibility of laboratory based assessments of cycling performance; investigated cross sectional age-related declines in selected metabolic, cardio-respiratory and performance related variables; examined the effect of age and testing protocol on the determination of threshold exercise intensity; studied the effects of age on laboratory and field based cycling performance; and identified correlates to endurance cycling performance.

One hundred and fourteen well trained competitive male endurance cyclists participated in these studies and selected groups of subjects performed laboratory based and field based tests. These consisted of:- Kingcycle peak power (PP) and peak aerobic capacity (PAC) tests; lactate minimum (Lac_{min}); ramped lactate threshold (LT_{ramp}) continuous lactate threshold (LT_{inc}); discontinuous lactate threshold (LT_{dis}), laboratory based 16.1-km time trial (LTT) and field based 16.1-km time trial (FTT).

Power output recorded using Kingcycle was significantly higher than SRM (P<0.001). Ratio limits of agreement between SRM and Kingcycle showed a bias of 0.90 (95%CI = 0.89-0.91) with a random error of ×/+ 1.07. Mean coefficient of variation for peak power (W_{peak}) and average power recorded using SRM during PP and LTT trials were 1.3% (95%CI = 1.0-2.0%) and 2.8% (1.6-4.9%) respectively. Age was a modest predictor of peak physiological variables assessed during PAC. The decline in W_{peak} with age was between 25 and 30 W, (~ 6 to 9%) per decade. Regression analysis provided a prediction of the decline in peak heart rate (HR_{peak} b·min^{-1}) [210-(0.66·age, yr), SEE 5%, P<0.01]. Relative exercise intensity (%W_{peak}) maintained during LTT and at lactate threshold tended (P = 0.08 and 0.07, respectively) to be higher in the veterans and %W_{peak} at lactate threshold was affected by age (P<0.05). Mean values for power, heart rate and oxygen uptake ($\dot{V}O_2$) at designated blood lactate derived thresholds were affected by testing protocol (P<0.05) with no interactive effect (P>0.05) of age and testing protocol. Seniors and veterans maintained a similar (P>0.05) relative exercise intensity (%W_{peak}, %HR_{peak} and

%$\dot{V}O_{2peak}$) and blood lactate concentration during LTT. Economy for LTT was higher (P<0.05) in the seniors but was dependent on method of calculation.

Veterans maintained a higher (P<0.05) mean %W_{peak} during FTT when compared with the seniors and a higher (P<0.05) mean pedal cadence during FTT when compared with LTT. During LTT mean pedal cadence and %W_{peak} were similar between groups (P>0.05). Blood lactate concentration was higher for the seniors on completion of LTT (P<0.05) but not FTT (P>0.05). Peak heart rate was strongly correlated (P<0.001) to mean heart rate during FTT and $\dot{V}O_{2peak}$ was strongly related (P<0.001) to mean $\dot{V}O_2$ during LTT (P<0.001). A highly significant relationship was found between W_{peak} and mean power during FTT (r = 0.99, P<0.001) but not W_{peak} and FTT performance time (r = 0.46, P>0.05).

The Kingcycle ergometer did not provide a valid measure of power when compared with a SRM power meter. However testing methods and equipment used in the present study did provide reproducible assessments of selected peak physiological variables and cycling endurance performance. Methodological issues regarding the effects of age, laboratory based testing and subject selection were highlighted when using physiological tests to assess endurance capacity and investigate the effects of ageing on cycling performance. Peak power (W_{peak}) was identified as a key determinant of outdoor 16.1-km time trial mean power but not performance time in senior and veteran cyclists.

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ACKNOWLEDGEMENT

AUTHOR'S DECLARATION

The rational and procedures for each part of the study were conceived by the author. The author was responsible for the construction and piloting of all the testing procedures used in the study. All recruitment, physiological tests, data input and statistical analyses were carried out by the author.

PUBLICATIONS WHICH HAVE ORIGINATED FROM THIS WORK

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INTRODUCTION

Within a sport science support programme athletes are regularly tested in order to monitor the effectiveness of interventions and treatments and assess subsequent changes in exercise performance capacity (Bird and Davison, 1997). The application of sport science support has been particularly appropriate to the development of cycle-sport and consequently numerous physiological tests have been developed to gain a clearer understanding of the underlying mechanisms which influence bicycling performance.

When investigating factors which influence physiological responses and more importantly performance related outcomes it is essential to use methods of assessment which are both reproducible and valid (Hopkins *et al.*, 1999). Therefore in order to control for confounding effects of extraneous variables it is common practice to assess cycling performance within the controlled environment of the sport science laboratory.

The early development of bicycle ergometry by A.V. Hill and co-workers in the 1920's was completed in order to gain a clearer understanding of physiological responses to exercise, however it was the work of Åstrand *et al.* in the 1950's which established bicycle ergometry as a laboratory based method for testing athletes. Notably, further adaptation in the 1980's using 'windload simulators' enabled investigators to develop more valid methods of testing which identified functional abilities and morphological components strongly related to successful cycling performance (see Coyle, 1995).

The introduction of the Kingcyle air-braked ergometer in the late 1980's was a major breakthrough in bicycle ergometry as it allowed cyclists to use their own bicycles during laboratory based tests and provided instantaneous data concerning power output, heart rate, pedal cadence and cycling speed. Consequently a large number of Kingcycle studies have been completed which have assessed the exercise performance capacity of racing cyclists (El-Sayed *et al.*, 1997; Palmer *et al.*, 1998; Hawley *et al.*, 1997) and investigated factors which influence indoor cycling time trial performance (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Lindsay *et al.*, 1996; Stepto *et al.*, 1999; Westgarth-Taylor *et al.*, 1997). Yet surprisingly there is very little information available concerning the validity and reproducibility of data recorded using the Kingcycle ergometry system.

In recent years several Kingcycle tests have been used to assess cycling performance ability for the purposes of team selection and talent identification. Although these Kingcycle based protocols are included within the guidelines for the physiological assessment of competitive cyclists provided by the British Cycling Federation and British Association of Sport and Exercise Sciences, very few studies have considered the efficacy of these procedures to assess field based cycling performance ability.

There is very little data available concerning the physiological responses of well trained cyclists during actual field based cycling races. However, with the introduction of a new portable bicycle ergometry system (SRM power-meter) it is now possible to collect data during laboratory and field based settings (Jeukendrup and van Diemen, 1998) and directly compare data collected during laboratory based assessments with information obtained in the field. Furthermore the validity and reproducibility of data recorded using different bicycle ergometry systems can also be evaluated.

Although laboratory based physiological tests provide invaluable sport science support for senior aged cyclists it is surprising that very few studies have investigated the applicability of these methods to provide support for veteran aged competitors. This is unfortunate considering the increasing number of veterans who regularly train and compete in cycle-sport (Jarvis, 1991; Tulloh, 1990).

Age related changes in exercise performance capacity have been extensively studied (Babcock *et al.*, 1994a; 1994b; Cartee, 1994; Kenney and Johnson, 1992; Meltzer, 1994; Nieman *et al.*, 1993; Seals *et al.*, 1994: Tate *et al.*, 1994; Therminarias *et al.*, 1991) and reviewed (Mazzeo, 1994; Stamford; 1988). However, the majority of research which has considered the effects of age on exercise performance has typically examined sedentary or moderately active subjects. Very little attention has been paid to the age related responses of 'veteran' athletes. New information in this area would provide novel and informative data regarding the physiological testing of both senior and veteran age groups.

Therefore the general aims of this thesis are to:-

- gain new knowledge concerning the validity and reproducibility of laboratory based physiological tests and testing equipment commonly used to assess cycling performance ability;

- provide new information regarding relationships between age and selected physiological/performance related responses recorded during laboratory and field based trials;

- gain a clearer understanding of methodological issues which should be considered when comparisons are made between age groups and when using laboratory based tests to assess senior and veteran competitors.

The objectives of this thesis are to:-

- assess the validity and reproducibility of the Kingcycle air-braked cycle ergometer to record power output during maximal and sub-maximal cycling tests;

- assess the reproducibility of physiological and performance related responses recorded during selected laboratory based methods of testing;

- study cross sectional age-related changes in physiological variables recorded during laboratory based tests;

- investigate the interactive effect of age and testing method on the assessment of cycling endurance capacity;

- compare performance related responses recorded during indoor and outdoor time trial races between senior and veteran cyclists;

- assess the validity of laboratory based assessments to predict field based cycling time trial performance.

LIST OF ABBREVIATIONS

MEASURES

W - Watt

V - velocity

kg - kilogram

H - height

m - metre

cm - centimetre

km - kilometre

s - second

min - minute

h - hour

% - percentage

kcal - kilocalorie

FFM - fat free mass

LBM - lean body mass

BM - body mass

%BF - percentage body fat

METHODS OF TESTING

PAC - Kingcycle ramp test to volitional exhaustion to determine peak cardio-respiratory variables and W_{peak}

PP - Kingcycle test to volitional exhaustion to determine W_{peak}

LTT - laboratory based 16.1-km cycling time trial

FTT - field based 16.1-km cycling time trial

PPs - Kingcycle PP test using a stabilising kit and calibration checked against SRM

PP_{NS} - Kingcycle PP without stabilising kit and calibration checked against SRM

LT_{ramp} - blood lactate threshold ramp test

LT_{inc} - blood lactate threshold incremental test

LT_{dis} - blood lactate threshold discontinuous incremental test

PERFORMANCE DESCRIPTORS

W_{max} - maximal power output recorded during a progressive ramped or incremental cycle ergometer test completed to volitional exhaustion

W_{peak} - highest average power recorded during any 60-s period of a Kingcycle PP/PAC test

$King_{Wpeak}$ - highest average power recorded during any 60-s period of a Kingcycle PP/PAC test using the Kingcycle ergometry system

SRM_{Wpeak} - highest average power recorded during any 60-s period of a Kingcycle PP/PAC test using the SRM ergometry system

V_{max} - highest average running speed recorded during a treadmill $\dot{V}O_{2max}$ test

$V@\dot{V}O_{2max}$ - running velocity at maximal oxygen consumption

W_{LTT} - average power recorded during a laboratory based 16.1-km cycling time trial

$King_{WLTT}$ - average Kingcycle power recorded during a laboratory based 16.1-km cycling time trial

W_{FTT} - average power recorded during a field based 16.1-km cycling time trial

T_{LTT} - time recorded during a laboratory based 16.1-km cycling time trial

T_{FTT} - time recorded during a field based 16.1-km cycling time trial

rev·min^{-1} - pedal cadence

rev·s^{-1} - revolutions per second

THRESHOLD DESCRIPTORS

LT - lactate threshold: ($\dot{V}O_2$, power output and HR recorded during an incremental exercise test at the point when blood lactate concentration is 1 mmol·L^{-1} above the baseline blood lactate level)

TLac - lactate threshold: ($\dot{V}O_2$, power output and HR at the point when an abrupt increase in lactate concentration is recorded above baseline blood lactate level)

D_{max} - lactate threshold: ($\dot{V}O_2$, power output and HR at the point on a blood lactate response curve which is the maximum distance from a straight line formed between the two end points of the curve)

IAT - individual anaerobic threshold

LTP - lactate turning point

OBLA - onset of blood lactate accumulation

OPLA - onset of plasma lactate accumulation

LMP - lactate minimum point

MLSS - maximal lactate steady-state

CARDIO-RESPIRATORY PARAMETERS

HR - heart rate

HRmax - maximal heart rate recorded during a progressive ramped or incremental cycle ergometer test completed to volitional exhaustion

HR_{peak} - highest heart rate value recorded during a Kingcycle PP/PAC test.

$\dot{V}O_{2peak}$ - highest $\dot{V}O_2$ recorded during any 60-s period of a Kingcycle PAC test

$\dot{V}O_{2max}$ - highest rate of oxygen consumption during maximal whole body exercise

$\dot{V}CO_2$ - carbon dioxide production

$\dot{V}CO_{2peak}$ - highest $\dot{V}CO_2$ recorded during any 60-s period of a Kingcycle PAC test

$\dot{V}CO_{2max}$ - highest rate of carbon dioxide production during maximal whole body exercise

$\dot{V}_E$ - minute ventilation

$\dot{V}_{Emax}$ - highest rate of ventilation during maximal whole body exercise

$\dot{V}_{Epeak}$ - highest $\dot{V}_E$ recorded during any 60-s period of a Kingcycle PAC test

TV - tidal volume

RER - respiratory exchange ratio

LVEDV - left ventricular end diastolic volume

CO - cardiac output

CaO_2 - arterial oxygen content

SaO_2 - arterial oxygen saturation

a-$\bar{v}O_2$ - arterio-venous difference

MVV - maximum voluntary ventilation

VC - vital capacity

FVC - forced vital capacity

$FEV_{1.0}$ - forced expiratory volume recorded in 1-s

$FEV_{1.0}$-to-FVC - ratio of $FEV_{1.0}$-to-FVC

f - breathing frequency

IHD - ischemic heart disease

Hb - haemoglobin

Hct - haematocrit

METABOLIC PARAMETERS

BLa - blood lactate concentration

BLa_5 - blood lactate concentration recorded at 5 min post PP/PAC test

SDH - succinate dehydrogenase

LDH - lactate dehydrogenase

CS - citrate synthase

EQUIPMENT

DEXA - dual-energy X-ray absorptiometry

SRM - Schoberer Rad Meptechnik powermeter

STATISTICS

MAS - multivariate allometric scaling

CV - coefficient of variation

SD - standard deviation

ICC - intraclass correlation coefficient

LoA - limits of agreement

CI - confidence intervals

ln - natural logarithm

SEE - standard error of estimate

JOURNALS, BOOKS AND ORGANISATIONS

MSSE - Medicine and Science in Sports and Exercise

RTTC - Road Time Trials Council

BASES - British Association of Sport and Exercise Sciences

ECSS - European College of Sport Sciences

IOC - International Olympic Committee

ACSM - American College of Sports Medicine

CHAPTER 1

1   DETERMINANTS OF CYCLING PERFORMANCE

1.1   INTRODUCTION

Cycling is a multi-disciplinary sport which incorporates a wide variety of competitive activities such as road racing, track racing and off-road 'mountain-biking'. Not surprisingly, each discipline of the sport imposes its own set of demands which are, in part, dependent on individual tactics, the terrain of the course and the duration of the event (Padilla *et al.*, 1999). Success in competitive cycling is determined by physiological (Coyle, 1995), biomechanical (Gregor *et al.*, 1991), environmental (Swain *et al.*, 1997) and psychological factors (Davis, 1995; Davis and Bailey, 1997). A comprehensive review of all these 'components of success' is beyond the scope of this thesis, however these factors have been considered in the reviews of Kyle (1991) and Ryschon (1994).

Physiological variables associated with successful endurance performance include:- maximal oxygen consumption (Burke, 1980; Saltin and Åstrand, 1967; Ramsbottom *et al.*, 1987; 1992); maximal aerobic muscle power (Hawley and Noakes, 1992; Scott and Houmard, 1994); oxygen consumption at lactate threshold (Coyle *et al.*, 1988); power at lactate threshold (Bishop *et al.*, 1998); and economy and efficiency of movement (Horowitz *et al.*, 1994). Coyle (1995) discussed several interrelationships between these variables and put forward an integrated model which identified key 'functional abilities' associated with successful endurance performance (see Figure 1). The purpose of this chapter of the thesis is to review available literature which has investigated these 'abilities' and considered factors which are related to cycling endurance performance.

1.2   FACTORS WHICH LIMIT EXERCISE PERFORMANCE

Physiological mechanisms associated with the onset of fatigue have been extensively studied and reviewed (see Davis *et al.*, 1995; Fitts and Metzger, 1988; McLester, Jr., 1997). For the purposes of this chapter mechanisms associated with the curtailment of maximal performance and delimitations of endurance performance are discussed.

PERFORMANCE VELOCITY

Resistance to Movement

Performance Power

Performance $\dot{V}O_2$

PERFORMANCE ABILITIES

Lactate Threshold Power or Velocity

FUNCTIONAL ABILITIES

Lactate Threshold $\dot{V}O_2$

Economy of Movement

Gross Mechanical Efficiency

Maximal Oxygen Consumption

Muscle Capillary Density

Stroke Volume

Aerobic Enzyme Activity

Distribution of Power Output and Technique

Muscle Fiber Type Composition or % Type 1

MORPHOLOGICAL COMPONENTS

Figure 1. Hypothetical model of the interrelationships of the physiological factors determining endurance performance ability (Coyle, 1995)

1.2.1 MAXIMAL PERFORMANCE

Several paradigms have been developed to explain the onset of fatigue and curtailment of exercise during maximal work. Although the physiological mechanism(s) responsible for the cessation of maximal 'aerobic' exercise is/are unclear, several authors (Bassett and

Howley, 1997; Noakes, 1998) have completed extensive reviews of the literature in this area and put forward two theoretical models to explain this phenomenon; the 'cardiovascular / anaerobic' model based on the work of Hill and Lupton (1923) and the 'regulated skeletal muscle contraction' model of Noakes (1998).

The cardiovascular / anaerobic model of exercise performance is based on the theory that:
> "during maximal exercise oxygen uptake attains a maximum and constant value which can not go any higher due to the limitations of the circulatory and respiratory systems", consequently "progressive muscle hypoxia, anaerobiosis and the production of lactic acid limit maximal exercise performance" (Noakes, 1998 p. 1382).

Although this model suggests that muscles become hypoxic during exercise (which ultimately affects muscle contractility leading to a reduction in power output) there appears to be no information available to show the occurrence of muscle anaerobiosis (hypoxia) during exercise performance. Furthermore although a change in muscle pH, related to the accumulation of lactate and hydrogen ion concentration, has been linked with the onset of muscle fatigue (Fitts and Metzger, 1988), investigators have suggested that the onset of blood lactate accumulation during exercise performance is also not associated with muscle hypoxia (Brooks, 1991; Spurway, 1992).

It is widely acknowledged that fatigue during maximal exercise performance (~10 min duration) is linked to a continuous increase in blood lactate concentration and the attainment of $\dot{V}O_{2max}$. Billat (1998) recently found that when triathletes were required to complete a cycle ride and treadmill run at a fixed relative exercise intensity to the point of exhaustion intensity (identified for each mode of exercise), there was no relationship between changes in blood lactate concentration and $\dot{V}O_2$ when compared with time to exhaustion. Unfortunately the underlying mechanisms responsible for the onset of fatigue during each mode of exercise were not elucidated, however analysis of the data revealed that $\dot{V}O_{2max}$ was recorded during the cycling test but not the running test. Although relative exercise intensity may have been different for cycling versus running, subjects reached

volitional exhaustion within the same amount of time (637 vs 654 s for cycling and running respectively).

Noakes (1998) argued that:

> "if the ultimate limits for maximal exercise are set by the cardiovascular system, some mechanism (other than skeletal muscle anaerobiosis) must be present to terminate exercise before the heart is itself damaged by the very plateau in CO that is theoretically necessary to explain the plateau phenomenon... during maximal exercise, progressive myocardial ischemia preceding muscle anaerobiosis must be thwarted so that neither the heart nor the skeletal muscle develops irreversible rigor and necrosis with fatal consequences" (p. 1395).

Proponents of the cardiovascular/anaerobic model (Bassett and Howley, 1997) have argued that "regardless of whether a particular individual achieves a plateau or not, there is an upper 'ceiling' on the body's ability to take up and utilize oxygen" (p. 594). Noakes (1998) has postulated that performance is limited by O_2 supply, however curtailment of exercise is not due to the effects of hypoxia on muscle contractility but the effects of a control mechanism which 'cuts in' to prevent damage to the heart.

The regulated skeletal muscle contraction model of endurance performance suggests that:

> "skeletal muscle contractile activity is regulated by a series of central, predominantly neural and peripheral predominantly chemical regulators that act to prevent the development of organ damage or even death during exercise in both health and disease and under demanding environmental conditions" (Noakes, 1998, p. 571).

There is very little direct evidence to support the postulate of Noakes (1998) however a similar controlling mechanism has been proposed by Dempsey and co-workers (1996). These authors hypothesised that the excessive requirements of ventilatory work during maximal exercise may cause reflex vasoconstriction of locomotor muscle leading to cessation of exercise performance. The authors postulated that due to an increased vasodilation in the working respiratory muscles there is a reduction in blood pressure and baroreceptors elicit sympathetically mediated vasoconstriction of limb locomotor muscles

in order to preserve blood pressure. Harms *et al.* (1997) found that assisted breathing reduced the workload on respiratory muscles during maximal exercise and this resulted in an increase in blood flow to locomotor muscles. Alternatively the workload of normal breathing during maximal effort invoked vasoconstriction in locomotor muscles and compromised locomotor muscle perfusion and oxygen uptake.

Further support for the postulate that a 'regulator' controls skeletal muscle contraction was provided by Koskolou *et al.* (1997) and Richardson *et al.* (1995) who found a reduction in blood flow to the locomotor muscles when subjects breathed hypoxic gas during maximal single leg exercise. It was suggested that blood flow was reduced to compensate for reduced arterial O_2 content (CaO_2) and O_2 delivery. The mechanism responsible for this apparent coupling between work rate, $\dot{V}O_2$ and blood flow during maximal exercise was not elucidated.

Both models suggest that O_2 supply affects exercise performance, however Noakes (1998) has argued that skeletal muscle function determines endurance performance ability. For instance, studies which have assessed physiological and morphological characteristics of elite endurance runners have found that the best predictors of performance are factors related to muscle fatigue resistance (Coetzer *et al.*, 1993; Weston *et al.*, 1999;) not maximal oxygen consumption. Similarly, Padilla *et al.* (1999) found that the best predictors of performance in a group of professional cyclists was absolute maximal sustained power output (W_{max}).

1.2.1.1 OXYGEN UPTAKE RECORDED DURING MAXIMAL EXERCISE

Maximal oxygen consumption ($\dot{V}O_{2max}$) denotes the highest rate of O_2 consumption during maximal whole body exercise (Coyle, 1995) and the achievement of $\dot{V}O_{2max}$ is based on the postulate that during intense/maximal exercise, oxygen uptake attains a maximum and constant value which is not increased by an increase in external workload (Taylor *et al.*, 1955). By definition, $\dot{V}O_{2max}$ equals the product of maximal cardiac output and maximal arterio-venous difference (Fick Principle). Wagner (1992) explained that the maximal rate of oxygen consumption depends on the exchange of oxygen from the air into the blood stream via the respiratory system; the delivery of oxygen in the blood to the musculature

via the cardiovascular system; and the extraction and utilisation of oxygen by the working musculature. It is now widely accepted that during whole body maximal exercise the primary determinants of $\dot{V}O_{2max}$ are central factors which influence the delivery of O_2 to the working musculature (Richardson *et al.*, 1999).

1.2.1.1.1 *Central limitations to $\dot{V}O_{2max}$*

Several studies (Anderson *et al.*, 1985; Richardson *et al.*, 1999) have provided strong evidence to suggest that $\dot{V}O_{2max}$ is not limited by mitochondrial metabolic rate but rather O_2 supply. In the study by Richardson *et al.* (1999) $\dot{V}O_{2max}$ was recorded during single leg and whole body exercise. Notably even though O_2 delivery per unit of muscle mass was very high during the single leg exercise, an increase in O_2 delivery (afforded by breathing 100% O_2) resulted in an increase in $\dot{V}O_{2max}$. Anderson *et al.* (1985) found that muscle perfusion during maximal whole body exercise (involving a large muscle mass), was less than during single leg work. Bassett and Howley, (1997) explained that when whole body exercise is performed, the capacity for blood flow of the large muscle groups exceeds maximal cardiac output, therefore the heart becomes the limiting factor that determines how much blood and oxygen are supplied to the muscles.

Evidence to support the theory that $\dot{V}O_{2max}$ is limited by O_2 supply has been provided by studies which have recorded an improvement in $\dot{V}O_{2max}$ when subjects have breathed hyperoxic gas (Inbar *et al.*, 1993; Peltonen *et al.*, 1995; Powers *et al.*, 1989) increased maximal cardiac output due to the effects of training (Rowell, 1986) and increased total blood volume due to blood re-infusion (Kanstrup and Ekblom, 1984). Supply of O_2 to the working musculature is dependent on the pulmonary system and cardiovascular system (Bassett and Howley, 1997) and for the purposes of this review factors which influence pulmonary function, cardiac output and blood volume are considered.

1.2.1.1.1.1 *Pulmonary function*

Inbar *et al.* (1993) explained that when healthy individuals exercise at sea level, arterial O_2 content is not a limiting factor of $\dot{V}O_{2max}$. This postulate is based on the knowledge that during maximal exercise the pulmonary system does not reach its maximum capacity and arterial O_2 saturation ($\%SaO_2$) remains high. However, when investigators have assessed

the maximal pulmonary function of highly trained endurance athletes, exercise induced arterial hypoxemia has been recorded (Johnson and Dempsey, 1991; Prefaut et al., 1994). McClaren et al. (1995) defined arterial hypoxemia as an arterial O_2 saturation of $\leq 92\%$. Johnson and Dempsey (1991) suggested that decreases in SaO_2 observed in highly trained individuals may reduce $\dot{V}O_{2max}$ by up to 14% and Prefaut et al. (1994) argued that endurance athletes who exhibit the highest $\dot{V}O_{2max}$ show the greatest decrease in SaO_2. In conclusion, Dempsey (1986) argued that the pulmonary system may become a limiting factor in O_2 transport during maximal exercise in the highly trained and suggested that in elite endurance athletes ventilation and pulmonary gas exchange may not develop sufficiently to accommodate training adaptations in central and peripheral processes such as cardiac output and a-$\dot{V}O_2$ difference.

Incomplete pulmonary gas exchange has been attributed to limitations in alveolar end-capillary diffusion (Prefaut et al., 1994) combined with an interstitial pulmonary oedema (Younes and Burke, 1985) and a 'disequilibration' between the alveolar gas and the capillary system of the lung due to the speed of transit of capillary blood during very high cardiac outputs (Dempsey, 1986). Consequently, Inbar et al. (1993) concluded that "incomplete pulmonary gas exchange secondary to a relative hypoventilatory response to maximal aerobic exercise contributed significantly to limiting $\dot{V}O_{2max}$" (p. 83).

It is worth noting that Inbar et al. (1993) recorded an increase in W_{max} when subjects were allowed to breathe a hyperoxic gas during a cycling test. It was suggested that the increase in power output during maximal exercise was related to a decrease in hyperventilatory drive and subsequent drop in respiratory discomfort. The authors estimated that an increase in ventilation of 10 $L\cdot min^{-1}$ would correspond with an increase in $\dot{V}O_{2max}$ of 2 $mL\cdot kg^{-1}\cdot min^{-1}$ and ~ 5 W in maximal power output. In contrast to this, Harms et al. (1997) observed a significant decrease in $\dot{V}O_{2max}$ when subjects attained a higher maximal minute ventilation using a 'proportional assist ventilator'. Analysis of the data revealed that an increase in maximal $\dot{V}_E$ of 10 $L\cdot min^{-1}$ from 146 to 157 $L\cdot min^{-1}$ was associated with a decrease in $\dot{V}O_2$ of 0.26 $L\cdot min^{-1}$. Unfortunately times to exhaustion recorded for normal and assisted conditions were not reported. The O_2 cost of maximal exercise hyperpnea has been estimated to be about 8-12% (Harms et al., 1997) and 13% (Johnson et al., 1992) of

$\dot{V}O_{2max}$, and therefore a significant share of maximal cardiac output is required for respiratory muscle work during maximal exercise.

Dempsey *et al.* (1996) postulated that "even if respiratory muscles did steal a significant share of cardiac output from locomotor muscles, $\dot{V}O_{2max}$ would not be compromised" (p. 1127) but maximal work rate required to elicit $\dot{V}O_{2max}$ would be reduced. This could be explained by a redistribution of blood flow to respiratory muscles which would lead to a shortened performance time during heavy endurance exercise and a lower maximum power output at $\dot{V}O_{2max}$ during short-term progressive exercise tests. Harms *et al.* (1997) found that $\dot{V}O_{2max}$ was unchanged during inspiratory muscle loading, however a significant reduction in $\dot{V}O_{2max}$ was recorded when respiratory muscles were unloaded. The authors could not explain this finding and a further investigation to assess changes in cardiac output during loaded and unloaded breathing conditions was proposed. Harms *et al.* (1997) did not assess maximum workload achieved during loaded and unloaded $\dot{V}O_{2max}$ tests therefore the postulate that unloaded breathing would result in an increase in maximum work rate could not be verified. There is very little information available concerning the possible effects of unloaded breathing on power output maintained during prolonged high intensity endurance exercise.

Hagberg *et al.* (1988) found no difference in static and dynamic lung function between endurance athletes and sedentary individuals of similar body size. However, Inbar *et al.* (1993) found that trained endurance athletes had a lower ventilatory reserve (MVV-$\dot{V}_{Emax}$) and lower maximal exercise ventilatory equivalent ($\dot{V}_{Emax}/\dot{V}O_{2max}$) when compared with untrained individuals. In a recent study by Mota *et al.* (1999) well trained endurance cyclists did not reach expiratory flow limitation during a maximal exercise test. The authors concluded that mechanical constraints did not limit aerobic exercise capacity. Studies which have investigated pulmonary function during maximal exercise (for review see Dempsey, 1986) found that endurance training did not affect maximum voluntary ventilation (MVV) but enabled athletes to utilise a greater percentage of their ventilatory capacity.

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1.2.1.1.1.2 Cardiac output

Maximal cardiac output (maximal heart rate × maximal stroke volume) has been described as 'the principal determinant of systemic oxygen transport' (Pate and Kriska, 1984; Spina, 1999) and the key determinant of high $\dot{V}O_{2max}$ values recorded in highly trained endurance athletes (Joyner, 1993). Strong evidence in support of this was provided by Anderson *et al.* (1985) who developed the 'single leg' exercise model to show that during maximal exercise $\dot{V}O_{2max}$ is limited by the maximal rate of muscle perfusion. The authors explained that during whole body exercise maximal blood flow to the locomotor muscles did not exceed the maximal cardiac output of the heart. Saltin and Strange (1992) explained that during maximal exercise, large quantities of blood are required to perfuse a fully dilated muscle capillary bed, therefore there is a close relationship between maximal cardiac output and exercise capacity. Consequently the heart is the most significant contributor to $\dot{V}O_{2max}$ and the essential component in achieving high levels of oxygen consumption during maximal exercise. Not surprisingly, there is a plethora of literature available describing cardiovascular adaptations to exercise training and Spina (1999) has recently considered this topic in depth.

1.2.1.1.1.3 Blood volume and haemoglobin

Haemoglobin concentration (Hb) is an important determinant of $\dot{V}O_{2max}$ in trained endurance athletes (McArdle *et al.*, 1991). Low levels of Hb are associated with a reduced oxygen-carrying capacity and a lower level of systemic oxygen transport (Pate and Kriska, 1984). Research by Kanstrup and Ekblom (1984) has revealed that an increase in total blood volume (Hb × blood volume) significantly improved $\dot{V}O_{2max}$. It was postulated that increases in $\dot{V}O_{2max}$ were due to an increased stroke volume and cardiac output and that the augmented stroke volume was the result of a higher pre-load after total blood volume expansion, coupled with the Frank-Starling mechanism. Kanstrup and Ekblom (1984) concluded that both the size of the blood volume and the Hb concentration were important determinants of $\dot{V}O_{2max}$ and endurance performance.

Interestingly, Kanstrup and Ekblom (1984) also found that a reduced Hb concentration with an elevated plasma volume resulted in an unchanged $\dot{V}O_{2max}$. It was argued that the elevated plasma volume increased cardiac output and compensated for the reduction in

arterial O_2 content. The authors postulated that plasma volume expansion alone could not increase $\dot{V}O_{2max}$. It was suggested that when Hb concentration was decreased and plasma volume did not change, cardiac output could not be increased to offset the drop in arterial O_2 content, consequently $\dot{V}O_{2max}$ was reduced.

In contrast to the work of Kanstrup and Ekblom (1984), Krip et al. (1997) found that an increase in plasma volume without a change in Hb content resulted in an increase in $\dot{V}O_{2max}$. It was postulated that an increase in plasma volume did not affect the $\dot{V}O_{2max}$ of trained participants, but did affect the $\dot{V}O_{2max}$ of sedentary subjects. Sedentary individuals may have had a sub-optimal blood volume when compared with trained subjects, consequently, an increase in blood volume was more beneficial in the sedentary group. Krip et al. (1997) argued that in trained individuals an improvement in $\dot{V}O_{2max}$ (due to an increase in blood volume) was dependent on an increase in Hb content. This theory was confirmed by the work of Warburton et al. (1999) who tested highly trained endurance cyclists and found that an increase in plasma volume did not affect $\dot{V}O_{2max}$.

Miller (1990) commented that changes in Hb in response to endurance training were the result of an increase in blood volume (hemodilution) and not a decrease in total Hb. In support of this finding, Keen et al. (1995) showed that changes in Hb level recorded in endurance cyclists during a stage race were relative to changes in blood plasma volume and not variation in Hb. The observed 'dilution effect' in response to exercise training was due to an increase in plasma volume more than the red blood cell volume as shown by a decrease in haematocrit (Hct) i.e. the ratio of cell volume to total blood volume.

Several investigations have shown that hypohydration and hypovolemia due to the affects of heat stress, reduction in fluid intake and administration of diuretics can reduce $\dot{V}O_{2max}$ and W_{max} (for review see Sawka, 1992). The precise mechanism(s) responsible for the observed decline in performance is/are unclear, however during maximal efforts it is reasonable to assume that cardiac output is compromised by the redirection of blood flow to the cutaneous vasculature in response to thermoregulatory stress (Shaffrah and Adams, 1984) therefore competition arises between blood flow to improve heat dissipation and blood flow to increase the supply of O_2 to the working muscles. Increased total muscle

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blood flow during maximal exercise is mainly due to an increase in cardiac output and distribution of blood away from 'non-working' areas of the body (McArdle *et al.*, 1991) however when total blood volume is reduced due to the effects of hypohydration and hypovolemia the maximal delivery of O_2 to the locomotor muscles is diminished.

1.2.1.1.2 Peripheral limitations to $\dot{V}O_{2max}$

Research has shown that $\dot{V}O_{2max}$ can be significantly influenced by metabolic processes occurring within the active muscle (Green and Patla, 1992; Honig *et al.*, 1992). Notably improvements in $\dot{V}O_{2max}$ have been attributed to increases in muscle oxidative capacity and mitochondrial protein (Hoppeler *et al.* 1985). Alternatively, marked declines in $\dot{V}O_{2max}$ have been associated with a decrease in muscle glycogen concentration (Asmussen *et al.* 1974; Åstrand and Saltin, 1961; Heigenhauser *et al.* 1983).

1.2.1.1.2.1 Effect of muscle fibre type and muscle recruitment patterns on $\dot{V}O_{2max}$

During maximal exercise, whole body $\dot{V}O_2$ is an assessment of the energy expenditure of the active musculature (Ryschon, 1994). Therefore, muscle recruitment patterns which activate a large muscle mass can be a key determinant of $\dot{V}O_{2max}$ (Green and Patla, 1992). In a review by Coyle (1995) it was suggested that elite endurance cyclists are able to recruit a greater muscle mass during maximal exercise with a concomitant increase in power output. Several studies (Coyle *et al.* 1988; Horowitz *et al.* 1994) have shown that highly trained endurance athletes have a relatively high percentage of slow twitch (Type I) muscle fibres. Furthermore, Coyle *et al.* (1988) found a correlation between years of endurance training and %Type I fibres in the vastus lateralis muscle of highly trained cyclists. Ryschon (1994) found that $\dot{V}O_{2max}$ was affected by the relative percentage Type I fibres and suggested that "endurance specialisation may be the product of training-induced adaptation at the muscle level" (p. 19). It is important to note that the affect of endurance training on muscle fibre composition is unclear (see Gollnick, 1989). Notably, a recent review by McComas (1996) considered the longitudinal effect of endurance training on alterations in fibre-Type composition and identified several studies which found that a decrease in Type IIb and relative increase in Type IIa following a period of high intensity endurance training. However the effect of endurance training on Type I muscle fibres was equivocal.

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Endurance training increases muscle-fibre capillary ratio (Coggan *et al.*, 1992) mitochondrial density (Coggan *et al.*, 1990) and enzymes involved in aerobic energy transfer (Coggan *et al.* 1992; 1993), however Gollnick (1989) argued that increases in mitochondrial content and function were important for enhancing work capacity but not $\dot{V}O_{2max}$.

1.2.1.1.3 Factors which affect $\dot{V}O_{2max}$

Various investigations have shown that $\dot{V}O_{2max}$ can be affected by a multiple of factors such as:- body size (Nevill *et al.*, 1992); gender (Pate and Kriska, 1984); age (Spina, 1999); and mode of exercise testing (Stromme *et al.*, 1977). Declines in $\dot{V}O_{2max}$ with age and methodological issues concerning the assessment of $\dot{V}O_{2max}$ are considered in chapters two and three of this thesis. The effect of gender on $\dot{V}O_{2max}$ is beyond the scope of this thesis.

Studies which have assessed the effects of endurance exercise training on $\dot{V}O_{2max}$ have found significant improvements/reductions following periods of training and detraining (for review see Wenger and Bell, 1986) and have suggested that improvements in $\dot{V}O_{2max}$ are dependent on genotype (Bouchard *et al.*, 1992), the initial fitness of the individual (McArdle *et al.*, 1991), and the intensity, frequency and duration of training (Wenger and Bell, 1986).

It is well established that $\dot{V}O_{2max}$ expressed as $L \cdot min^{-1}$ is highly correlated with the mass of skeletal muscle tissue that is active during exercise (Pate and Kriska, 1984; Sawka, 1986). Therefore high values of $\dot{V}O_{2max}$ attained by racing cyclists are representative of the large active muscle mass involved in cycling exercise (Ryschon, 1994). However, in order to allow comparison between individuals of different body size $\dot{V}O_{2max}$ values are commonly scaled relative to the body mass of the athlete ($mL \cdot kg^{-1} \cdot min^{-1}$). The use of ratio scaling is widely used to assess the $\dot{V}O_{2max}$ of endurance athletes, however authors have advised the use of power function models to adjust $\dot{V}O_{2max}$ values to account for differences in body size. Notably Neville *et al.* (1992) and Heil (1997b) have suggested that dividing $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) by [body mass $(kg)^{-0.67}$] provides a more valid inter-individual assessment of $\dot{V}O_{2max}$.

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1.2.1.1.4 Values for $\dot{V}O_{2max}$

some of the highest values recorded for aerobic power when expressed as either $L \cdot min^{-1}$ and/or $mL \cdot kg^{-1} \cdot min^{-1}$ (Saltin and Åstrand, 1967). $\dot{V}O_{2max}$ values have ranged from 4-5 $L \cdot min^{-1}$ (60 $mL \cdot kg^{-1} \cdot min^{-1}$) for club standard cyclists (Hickey et al., 1992; Langenfeld et al., 1994; Pugh, 1974) to above 5 $L \cdot min^{-1}$ (70 $mL \cdot kg^{-1} \cdot min^{-1}$) for national/international standard (Coyle et al., 1991; Horowitz et al., 1994; Lucia et al., 1998) (see Table 1).

1.2.1.2 MAXIMAL/PEAK POWER.

Power output (W) refers to the measurable external (mechanical) power that a cyclist can generate over a specific period of time (de Groot et al. 1994) and peak aerobic power (W_{peak}) describes the highest average sustained power output recorded during any 60-s period of a progressive exercise test to volitional exhaustion (Keen et al., 1991). Notably, peak power has also been described as maximal sustained power output (Weston et al., 1997) maximal power (Hopkins et al., 1999; Stepto et al., 1999; Westgarth-Taylor et al., 1997) and maximal muscle power (Noakes, 1988) however this predominantly 'aerobic' measure of power output should not be confused with measures of peak and mean 'anaerobic' power recorded during a 30-s Wingate test (Vandewalle et al., 1987).

Table 1. Selected characteristics of elite and professional cyclists

Study	Group	BM (kg)	$\dot{V}O_{2max}$ $L \cdot min^{-1}$	$\dot{V}O_{2max}$ $mL \cdot kg^{-1} \cdot min^{-1}$	W_{max} (W)	W_{max}/BM ($W \cdot kg^{-1}$)
Lucia et al. (1998)	P	68.9	5.10	74.0	501	7.27
Wilber et al. (1997)	E	72.6	5.09	70.3	470	6.47
Lucia et al. (1998)	P	67.3	4.90	72.9	466	6.92
Vrijens et al. (1982)	E	71.4	4.81	67.6	448	6.27
Lucia et al. (1998)	E	69.2	5.10	73.9	429	6.20
Vrijens et al. (1982)	P	71.3	4.71	65.7	419	5.88
Mean ± SD		70.1 ± 2.0	4.95 ± 0.17	70.7 ± 3.5	456 ± 30	6.5 ± 0.5

P   = professional cyclists

E   = elite cyclists

BM   = body mass

1.2.1.2.1 Factors which affect maximal/peak power

Factors which influence maximal/peak power values achieved during maximal exercise include; body size (Winter and Nevill, 1996), age (Seiler *et al.*, 1998), training status (Lucia *et al.*, 1998), gender (Wilber *et al.*, 1997), exercise mode (Jones and Passfield, 1998), testing protocol (Davis *et al.*, 1982) and glycogen depletion (Heigenhauser *et al.*, 1983). Changes in W_{max} associated with ageing and methodological issues concerning the assessment of W_{max} are considered in chapters two and three of this thesis. Very few studies have assessed the relationship between W_{max} and body size, although authors have used standard ratio scales ($W \cdot kg^{-1}$) to evaluate differences in maximal/peak power between cyclists (Dobbins, 1996; Hawley and Noakes, 1992; Padilla *et al.*, 1999). Neville *et al.* (1992) investigated the relationship between peak power achieved during a Wingate test and body mass, and found that the best method of scaling power to account for differences in body size was to use the power function model [body mass $(kg)^{-0.67}$]. Unfortunately, there is very little information available concerning the relationship between body size and an assessment of W_{peak} and W_{max}.

A plethora of literature is available concerning biomechanical factors which have a direct influence on power output during cycling exercise. It is worth noting that Lucia *et al.* (1998) postulated that professional cyclists achieved a higher maximal power output at $\dot{V}O_{2max}$ due to the development of biomechanical adaptations in the pedal stroke. In support of this work Coyle *et al.* (1991) found that elite cyclists adopted a pedalling technique which applied large vertical forces to the pedal during the down stroke to maximise the amount of propulsive torque produced during this interval. The biomechanics of cycling are beyond the scope of this thesis, however, literature on this issue has been reviewed by Gregor *et al.* (1991) and Too (1990).

High intensity interval and endurance exercise training improves W_{max} (Lindsay *et al.*, 1996; Stepto *et al.*, 1999; Westgarth-Taylor *et al.*, 1997; Weston *et al.*, 1997) however the underlying mechanism(s) responsible for changes in maximal/peak power is/are unclear. Increases in power output in response to training have been attributed to peripheral adaptations in skeletal muscle fibre type, power output distribution among individual muscle fibres, muscle capillary density and oxidative enzyme activity as well as central

changes in maximal cardiac output and total blood volume (Coyle, 1995). Several authors (Hawley and Noakes, 1992; Keen *et al.*, 1991) have found that the relationship between W_{max} and $\dot{V}O_{2max}$ in endurance trained cyclists is highly significant ($r = 0.98$ and $r = 0.97$, respectively) consequently this relationship has been used to alternatively estimate $\dot{V}O_{2max}$ and W_{max} (Padilla *et al.*, 1999).

Very few studies have considered the relative contribution of anaerobic power to W_{max}. Craig *et al.* (1993) found a significant inverse relationship between anaerobic capacity and cycling performance time during a field based 4000m individual track pursuit ($r = -0.50$) and Black *et al.* (1998) found that time to complete a field based 16.1-km cycling time trial was significantly inversely related to peak power ($W \cdot kg^{-1}$, $r = -0.71$) and mean power ($W \cdot kg^{-1}$, $r = -0.58$) achieved during a Wingate test. Unfortunately no study has assessed the relationship between an assessment of W_{peak} or W_{max} and anaerobic power.

1.2.1.2.2 *Values for maximal/peak power*

Maximal/peak aerobic power values reported for competitive cyclists have ranged from ~380-420 W for trained cyclists (Dobbins, 1996; El-Sayed *et al.* 1997; Jeukendrup *et al.* 1996; Wagenmakers *et al.* 1996) to above 440 W for elite/highly trained cyclists (Wilber *et al.*, 1997) and above 480 W for professional cyclists (Lucia *et al.*, 1998). Notably, Padilla *et al.* (1999) calculated the W_{max}/body mass ratio of elite and professional cyclists and found that professional competitors had a W_{max}/body mass ratio above 6.5 $W \cdot kg^{-1}$ (see Table 1).

1.2.2 ENDURANCE PERFORMANCE

Bassett and Howley (1997) postulated that, "...performance in endurance events is limited by oxygen delivery ($mL \cdot kg^{-1} \cdot min^{-1}$) set by the subject's $\dot{V}O_{2max}$ and the $\%\dot{V}O_{2max}$ that can be maintained" (p. 597). In support of this supposition, studies have found that elite athletes are able to sustain a higher $\%\dot{V}O_{2max}$ during exercise performance (Coyle *et al.*, 1991), and that measures of performance associated with a threshold exercise intensity occur at a higher $\%\dot{V}O_{2max}$ in highly trained individuals (Allen *et al.*, 1985; Coyle *et al.*, 1988; Lopategui *et al.*, 1986).

Lopategui et al. (1986) found that the 'anaerobic' threshold of elite road racing cyclists was significantly higher than novice cyclists when expressed as $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and $\%\dot{V}O_{2max}$. Absolute $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) and W_{max} (W) for each group were not significantly different, however, $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and W_{max} ($W \cdot kg^{-1}$) were higher in the elite group. The authors suggested that differences in $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and $\%\dot{V}O_{2max}$ at the 'anaerobic' threshold could be used to identify novice and elite cyclists.

The threshold concept of endurance performance suggests that when exercising below threshold, variables such as BLa, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}_E$ remain relatively stable, however when exercising above threshold, these variables do not remain stable but continue to increase until $\dot{V}O_{2max}$ is reached and exercise performance is curtailed (Poole et al., 1994). An increased intensity above threshold leads to a more rapid increase in $\dot{V}O_2$ and a reduction in time to exhaustion (Whipp, 1994) and the exercise intensity an athlete can maintain for the duration of a race is dependent on their $\dot{V}O_{2max}$ and the absolute $\dot{V}O_2$ at race pace relative to $\dot{V}O_2$ at threshold (Coyle, 1995). For instance, a 1-h cycling time trial is performed at a relatively high exercise intensity with an average $\dot{V}O_2$ ($L \cdot min^{-1}$) about 14% above $\dot{V}O_2$ at lactate threshold, a relative exercise intensity of about 86-90% $\dot{V}O_{2max}$ (Coyle et al., 1991). For an event of 15 min duration such as a 5-km run athletes can maintain a about 93% $\dot{V}O_{2max}$ which is 8% above $\dot{V}O_2$ at OBLA (Ramsbottom et al., 1992) and endurance runners maintain about 75% $\dot{V}O_{2max}$ during a Marathon race which is 5% above $\dot{V}O_2$ at OPLA (Farrell et al., 1979). Evidently athletes can maintain an exercise intensity above designated threshold values whether stated as lactate threshold, OPLA or OBLA for different periods of time. In order to account for this anomaly authors have either established a threshold value which coincides with performance in a particular event (see Coyle, 1995) or have established a 'maximal steady state' workload which can be maintained for a fixed period of time (Beneke and von Duvillard, 1996; Jones and Doust, 1998). It is worth noting that different definitions and designations used to identify and quantify a threshold exercise intensity need to be considered when comparisons are made between studies concerning the relative performance related responses of endurance athletes.

Noakes (1998) has argued that studies which have found strong relationships between threshold values and endurance performance have typically assessed heterogeneous groups of competitors. Consequently the high correlation coefficients recorded between performance and threshold measures such as $\dot{V}O_2$ at the onset of blood lactate accumulation are an artefact of the statistical test. Furthermore the relationship between $\dot{V}O_{2max}$ and performance (expressed as $\%\dot{V}O_{2max}$) is dependent on the concept of a $\dot{V}O_2$-plateau. The regulated skeletal muscle function model of Noakes (1998) does not accommodate the plateau phenomenon therefore a measure of $\%\dot{V}O_{2max}$ in terms of performance and threshold is not applicable.

Endurance performance is measured as the time to complete a fixed distance, and each competitor completes the distance in their fastest time. Consequently, time to complete the race varies from the fastest time of the winner to the slowest time of the final competitor. Notably, Horowitz et al. (1994) and Coyle et al. (1991) assessed the performance related responses of groups of cyclists with similar $\dot{V}O_{2max}$ (L·min^{-1}). Horowitz et al, (1994) found that the $\%\dot{V}O_{2max}$ maintained during a laboratory based 40-km time trial was similar between groups, however Coyle et al. (1991) found that elite cyclists maintained a higher $\%\dot{V}O_{2max}$ during a laboratory based 1-h cycling time trial. There appears to be no information available concerning the effects of using a fixed time trial ride (Coyle et al., 1991) compared with fixed distance race (Horowitz et al., 1994) to assess performance related responses of cyclists during this type of test. Ramsbottom et al. (1992) argued that during a lab-based 5-km running time trial, 'oxygen consumption at race pace dictated running performance irrespective of the $\%\dot{V}O_{2max}$ it represented' (p. 10) and that time to complete a fixed distance race was dependent on the running speed/power output at $\dot{V}O_2$ maintained during the race. Coyle et al. (1983, 1988) argued that time to fatigue in highly trained competitors exercising above threshold was dependent on the difference between performance $\dot{V}O_2$ and $\dot{V}O_2$ at threshold and therefore was not dependent on $\dot{V}O_{2max}$. Furthermore, Lucia et al. (1998) postulated that endurance performance in professional cyclists was not related to $\dot{V}O_{2max}$ but the combination of a high W_{max} at $\dot{V}O_{2max}$ and a higher absolute and relative power output and $\dot{V}O_2$ at threshold.

It is generally believed that maximal performance is limited by the maximum rate at which the cardiovascular system (central factors) can supply active muscles with oxygen (Bassett and Howley *et al.*, 2000). However endurance events are not completed at 100% $\dot{V}O_{2max}$ therefore at sub-maximal exercise intensity performance is limited by the maximum rate at which the active muscles can use oxygen (peripheral factors). Wood (1998b) considered the key determinants of endurance running and explained that the ability to maintain a high running speed during field based races is dependent on an individual's oxidative capacity and not $\dot{V}O_{2max}$.

Wood (1998b) described oxidative capacity as "the ability of a muscle to use oxygen at a high rate" (Wood, 1998b, p. 51) and explained that inter-individual differences in oxidative capacity were dependent on concentrations of key aerobic enzymes (Coyle *et al.*, 1988) and relative proportion of type I muscle fibres within the active muscle (Coyle *et al.*, 1988; Ivy *et al.*, 1980). There appears to be no consensus available concerning the assessment of oxidative capacity, however Wood (1998b) argued that oxidative capacity determines the power output or running velocity which can be maintained during a race (usually expressed as a $\%W_{peak}$ or $\%\dot{V}O_{2max}$).

1.2.2.1 THRESHOLD CONCEPT OF ENDURANCE PERFORMANCE

The threshold concept has been extensively studied (Cabrera and Chizeck, 1996; Heck *et al.*, 1985) and reviewed (Antonutto and di Prampero, 1995; Hagberg, 1984; Spurway, 1992; Walsh and Banister, 1988) and there is a plethora of literature available concerning the identification and application of threshold phenomenon (Billat, 1996; Londeree, 1997). The development of the lactate threshold paradigm has been based on the assumption that the 'threshold' level of performance represents an 'optimum' training and competitive exercise intensity (Billat, 1996; Londeree, 1997) and is the single most important determinant of endurance performance ability (Coyle, 1995; Tanaka, 1990). For the purposes of this thesis, a threshold exercise intensity will be defined as the point of transition between a condition of relative homeostasis to non-homeostasis. In the homeostatic condition factors which contribute to fatigue remain relatively stable and in a non-homeostatic condition these factors are increasing.

1.2.2.1.1 *Lactate threshold*

Since the early work of Margaria *et al.* (1933) exercise physiologists have known that an increase in blood lactate concentration during a graded exercise test is indicative of metabolic stress, and that the accumulation of lactate within the working muscle is a contributor to the onset of fatigue. It was not until the late 1960's and 70's that the term threshold was popularised by Wasserman and co-workers (for review see Wasserman *et al.*, 1979) and the term threshold of anaerobic metabolism was introduced by Wasserman and McIlroy (1964) to describe the point of 'transition' from predominantly aerobic to predominantly anaerobic metabolism (see Antonutto and di Prampero, 1995). The term 'anaerobic threshold' has become a popular method of describing the sudden and continued increase in blood lactate concentration during progressive exercise, however Wasserman and colleagues (1983; 1990) described the anaerobic threshold as the point at which hyperventilation occurred during progressive exercise in response to a marked increase in blood lactate concentration.

Jones and Doust (1998) defined the maximal lactate steady state (MLSS) phenomenon as "the highest blood lactate concentration at which a balance exists between the rate of appearance of lactate in the blood and the rate of removal of lactate from the blood" (p. 1304). Several authors (Carter *et al.*, 1999; Jones and Doust, 1998) have suggested that at exercise intensities below MLSS, energy is provided mainly from the processes of aerobic metabolism, however at exercise intensities above MLSS, a greater contribution to overall energy production is provided by anaerobic metabolism. At exercise intensities above MLSS physiological variables such as blood lactate concentration ($mmol \cdot L^{-1}$), $\dot{V}O_2$ ($L \cdot min^{-1}$) and $\dot{V}_E$ ($L \cdot min^{-1}$) and do not attain a steady state condition but continue to increase to the point of fatigue. At exercise intensities below MLSS, $\dot{V}O_2$ ($L \cdot min^{-1}$) and $\dot{V}_E$ ($L \cdot min^{-1}$) remain relatively stable and blood lactate either remains at a relatively stable concentration or declines over time (Jones and Doust, 1998). Consequently, several authors (Jones and Doust, 1998; Carter *et al.*, 1999) have suggested that exercise intensity at MLSS is the key determinant of endurance performance.

Previously, the use of the term 'anaerobic' threshold suggested that the increase in blood lactate concentration observed during a progressive exercise test was due to muscle

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hypoxia and the supply of energy via anaerobic pathways (Katz and Sahlin, 1990). It is now widely accepted that the sudden increase in blood lactate concentration observed during progressive exercise is not due to hypoxia but is influenced by changes in substrate utilisation (Crowley *et al.*, 1996) patterns of motor unit recruitment (Davis *et al.*, 1983) hormone-mediated effects on glycogenolysis and glycolysis (Pokan *et al.*, 1995; Young *et al.*, 1991) and reduced lactate production to removal ratio (Donovan and Pagliassotti, 1990). The exact underlying mechanism(s) responsible for the lactate threshold phenomenon is/are unclear, possible mechanisms have been reviewed in detail by Walsh and Bannister (1988) and Stainsby and Brooks (1990) therefore these articles can be used as a reference point for the reader who wishes to investigate these issues further.

In order to avoid confusion when using the term 'anaerobic', Farrell *et al.* (1979) introduced the term 'onset of plasma lactate accumulation (OPLA) to describe the threshold phenomenon and lactate kinetics observed during a progressive incremental exercise test. The term onset of blood lactate accumulation (OBLA) was introduced by Jacobs *et al.* (1981) to define the exercise intensity at which blood lactate concentration reached 4 mmol·L^{-1}. Notably, exercise intensity at 4 mmol·L^{-1} was significantly higher than the intensity defined by OPLA (Farrell *et al.*, 1979). Therefore, in order to provide a clear distinction between each method of identification, Coyle and co-workers introduced the term 'lactate threshold' (LT) to define the exercise intensity at which blood lactate increased 1 mmol·L^{-1} above baseline blood lactate level. This point of identification was similar to the intensity designated by OPLA (Farrell *et al.*, 1979), however, Coyle (1995) explained that $\dot{V}O_2$ at LT intensity was about 5% higher than $\dot{V}O_2$ at OPLA, as this new definition of 'threshold' coincided closely with the self-selected pace of runners during a Marathon race.

Throughout the past two decades various authors (for review see Billat, 1996) have developed new methodologies and terms to identify and define a 'threshold' exercise intensity. Table 2 includes several terms that exist in the literature to define various threshold values recorded during progressive exercise tests. Assessments of a 'threshold' exercise intensity have typically involved invasive measures of blood lactate concentration however non-invasive measures of ventilation and heart rate have also been used to

designate a threshold level of performance. For the purposes of this thesis non-invasive measures of ventilation and heart rate threshold are not considered.

Table 2. Terms used to define threshold changes in blood lactate concentration.

Definition (abbreviation) and designation
Lactate threshold (LT): $\dot{V}O_2$, power output and HR recorded during an incremental exercise test at the point when blood lactate concentration is 1 mmol·L⁻¹ above the baseline blood lactate level (Coyle, 1995).
Lactate threshold (TLac), *also described as the 'aerobic' threshold and onset of plasma lactate accumulation*: $\dot{V}O_2$, power output and HR at the point when an abrupt increase in lactate concentration is recorded above baseline blood lactate level (Farrell *et al.*, 1979).
Lactate threshold (D$_{max}$): $\dot{V}O_2$, power output and HR at the point on a blood lactate response curve which is the maximum distance from a straight line formed between the two end points of the curve, (Cheng *et al.*, 1992).
Lactate turning point (LTP), *also described as the 'anaerobic' threshold*: $\dot{V}O_2$, power output and HR at the point when a second abrupt increase in blood lactate concentration is recorded above baseline blood lactate (Davis *et al.*, 1983).
Onset of blood lactate accumulation (OBLA), *also described as the 'anaerobic' threshold*: $\dot{V}O_2$, power output and HR at which blood lactate reaches a fixed concentration of 4 mmol·L⁻¹ (Jacobs *et al.*, 1981).
Individual anaerobic threshold (IAT): $\dot{V}O_2$, power output and HR at the point when the increase of blood lactate is peak and equal to the rate of diffusion of lactate from the exercising muscle, (Stegmann *et al.*, 1981).
Lactate minimum point (LMP): $\dot{V}O_2$, power output and HR corresponding to the minimum blood lactate concentration determined during an incremental test completed after a bout of intense exercise which induced lactic acidosis, (Tegtbur *et al.*, 1993).
Maximal steady-state of blood lactate level (MLSS): $\dot{V}O_2$, power output and HR which correspond to the highest exercise intensity at which blood lactate concentration does not change more than 1 mmol·L⁻¹ between 10 and 30 min of exercise (Beneke and von Duvillard, 1996; Jones and Doust, 1998).

A wide range of testing methods have been developed to identify and quantify a threshold exercise intensity. Widely accepted methods of determining blood lactate thresholds are described in the British Association of Sport and Exercise Sciences guidelines for the physiological testing of athletes (Bird and Davison, 1997). The lactate minimum test by Tegtbur et al. (1993) is a relatively new method used to determine a blood lactate threshold. The development of the Lac_{min} test originated from the earlier work of Davis and Gass (1979, 1981) and Davis et al. (1983) who investigated the effects of performing an incremental test during lactacidaemia on blood lactate and ventilatory response. Notably Davis et al. (1983) found that the occurrence of a ventilatory threshold during the incremental test performed during lactacidaemia coincided with a second abrupt increase in blood lactate concentration (LTP).

Tegtbur et al. (1993) referred to the work of Davis and co-workers (1979, 1981, 1983) in order to develop a new indirect method to predict maximum lactate steady state (MLSS) as well as an objective assessment of anaerobic threshold intensity during running. Tegtbur et al. (1993) described the equilibrium point between a decrease and increase in blood lactate concentration recorded during an incremental running test initiated during lactic acidosis as the lactate minimum speed (V@LMP) and the term lactate minimum test was used to describe the procedure. Although the basic concept of the testing protocol of Davis and colleagues (1979, 1981, 1983) was applied to the lactate minimum test, Tegtbur et al. (1993) adapted the test to accommodate the methodological restrictions imposed by treadmill testing and the need to use a discontinuous incremental protocol for the collection of blood samples during running. The testing protocol of Tegtbur et al. (1993) involved two sprint tests to induce lactacidaemia followed by a rest period of 8 min walking. Starting intensity for the incremental test was based on data collected during an habituation trial and was dependent on the fitness level of the athlete. Running speed was increased by 0.33 $m \cdot s^{-1}$ to the limit of tolerance and the duration for each incremental stage was 800 m distance and therefore the duration of each stage varied between subjects. Blood lactate samples were collected on completion of each increment during a 30-s rest period.

Brennan et al. (1996) adapted the lactate minimum test in order to determine LMP in cyclists and used a similar method to Davis and Gass (1981) and Davis et al. (1983).

Cyclists performed a Kingcycle peak aerobic power test (PP) immediately followed by a 5 min active recovery period. The second part of the test involved a progressive ramp protocol initiated at the same work rate of the PP test (either 200 or 250 W depending on the subject's body mass and ability) and ramp rate for the test was 6 $W \cdot min^{-1}$. Blood samples were collected at 2.5 min intervals throughout the second test for the determination of LMP.

1.2.2.1.1.1   Factors which affect lactate threshold

Numerous studies have investigated the various factors which directly affect lactate threshold (for review see Billat 1996). Coyle (1995) suggested that during high intensity endurance performance, $\dot{V}O_2$ at LT sets the limit of $\%\dot{V}O_{2max}$ which can be sustained for the duration of the event. In support of this, mean $\%\dot{V}O_{2max}$ maintained during a 1-h laboratory based performance ride by elite cyclists was 14% (0.55 $L \cdot min^{-1}$) above $\dot{V}O_2$ at LT (Coyle *et al.*, 1991). Endurance training increases TLac when expressed as $\%\dot{V}O_{2max}$ or absolute workload (Davis *et al.* 1979; Denis *et al.* 1984) and investigators have attributed changes in $\dot{V}O_2$ and power output at TLac to:- an increased rate of lactate clearance (Coyle, 1995; Mazzeo *et al.*, 1986); decreased catecholamine secretion during exercise (Brooks, 1991); an improvement in the distribution of power output and technique (Coyle, 1995); and increased O_2 delivery to the working muscles as well as a greater oxidative capacity through adaptations in mitochondrial function and content (see Gollnick *et al.*, 1986).

Coyle *et al.* (1988) assessed the relationships between $\dot{V}O_2$ at TLac and a wide range of selected physiological and morphological variables. Notably, percent type I muscle fibres, muscle capillary density and years of endurance training were the best predictors of $\dot{V}O_2$ at TLac in a group of well trained endurance cyclists. It was suggested that increases in TLac in response to training were strongly influenced by muscle capillary density, which in turn correlated to years of endurance training. It was hypothesised that a larger muscle capillary density increased $\dot{V}O_2$ at TLac due to an improved rate of diffusion of lactate from the active muscle. Aunolo and Rusko (1992) observed that the ratio of slow-twitch to fast-twitch muscle fibres in the vastus lateralis affected blood lactate response during progressive exercise. Subjects with a high percent slow-twitch fibres showed a reduced blood lactate response compared with subjects with a high percent fast-twitch fibres.

Consequently subjects with high percent Type I fibres attained a higher power output and %$\dot{V}O_{2max}$ at TLac. Coyle (1995) suggested that power output at TLac can be affected by mechanical efficiency. It was argued that endurance athletes can attain a high power output and $\dot{V}O_2$ at TLac through the recruitment of a relatively high number of muscle fibres during high intensity exercise. Furthermore, Coyle *et al.* (1988, 1991) and Horowitz *et al.* (1994) found that mechanical efficiency in endurance cyclists was highly correlated with a high percent Type I fibre composition.

Mayes *et al.* (1987) found that 6 weeks of endurance training significantly increased $\dot{V}O_{2max}$, but did not increase capacity to exercise at a high relative exercise intensity. Denis *et al.* (1984) suggested that %$\dot{V}O_{2max}$ at TLac only increases relative to $\dot{V}O_{2max}$ when $\dot{V}O_{2max}$ has stabilised. Billat (1996) suggested that changes in TLac are influenced by the genetic characteristics of the athlete and argued that the range in which the relative threshold can shift is controlled by the ratio of slow twitch to fast twitch muscle fibres in the active musculature. It was concluded that an individual's response to endurance training in terms of their %$\dot{V}O_{2max}$ and power output at TLac may be pre-determined by their fibre type composition.

1.2.2.1.1.2 Values for lactate threshold

Values for $\dot{V}O_2$ (expressed as %$\dot{V}O_{2max}$) at TLac and lactate turning point (LTP) in trained cyclists have usually ranged between 40-65% for TLac (Massé-Biron *et al.* 1992; Yoshida, 1984; Yoshida *et al.* 1987) and 72-86% for LTP (McClellan and Cheung, 1992; Quirion et al. 1988; Seburn *et al.* 1992; Tokmakidis and Leger, 1995; Yoshida, 1984). Values for power output at TLac and LTP have ranged from 63% W_{max} for TLac (Maassen and Busse, 1989) to 72-79% W_{max} for LTP (Hofmann *et al.* 1992; 1994; Massé-Biron *et al.* 1992; Maassen and Busse, 1989; Yoshida, 1984). Notably, Yoshida (1984) reported that HR at TLac was 58% maximum heart rate (%HR_{max}) and Hofmann *et al.* (1992; 1994) calculated that HR at LTP was about 90% HR_{max}.

1.2.2.2 ECONOMY OF MOVEMENT AND GROSS MECHANICAL EFFICIENCY

During cycling exercise, economy is a measure of power output (W) generated relative to energy expenditure measured from $\dot{V}O_2$ (Coyle, 1995). Economy is expressed as watts

produced per litre per min oxygen consumed [(W·$\dot{V}O_2$ (L·min^{-1})]. Gross mechanical efficiency is the percentage (%) of power produced relative to total energy expenditure when the total energy expenditure (kcal·min^{-1}) is calculated from steady-state $\dot{V}O_2$ and respiratory exchange ratio (RER), (Gaesser and Brooks, 1975).

Several authors (Coyle, 1995, Horowitz *et al.*, 1994; Joyner, 1993; Olds *et al.*, 1995) have suggested that economy and gross mechanical efficiency are key determinants of endurance performance ability. In agreement with this supposition, Anderson (1996) evaluated the effect of running economy on successful endurance performance and concluded that "at higher levels of competition, 'natural selection' tends to eliminate athletes who fail to either inherit or develop characteristics which favour economy" (p. 77). Horowitz *et al.* (1994) observed a significant difference in economy and gross mechanical efficiency between cyclists matched on i) performance $\dot{V}O_2$, ii) $\dot{V}O_{2max}$ iii) $\dot{V}O_2$ at lactate threshold and iv) years of endurance training. Each group had significantly different muscle fibre composition and higher values for power output, economy and gross mechanical efficiency were recorded in cyclists who exhibited a high percentage of Type I muscle fibres (344 W, 77 [(W·$\dot{V}O_2$ (L·min^{-1})], 22.1% for high %Type I and 314 W, 70 [(W·$\dot{V}O_2$ (L·min^{-1})], 20.3% for normal %Type I). It was concluded that cyclists with a relatively high percentage of Type I muscle fibres display at least a 9% greater performance ability than equally trained cyclists with normal fibre type composition.

1.2.2.2.1 Factors which affect economy and gross mechanical efficiency

The effect(s) of endurance training on economy and gross mechanical efficiency of cycling is(are) unclear. Coyle (1995) explained that improvements in gross mechanical efficiency were influenced by the cyclists' ability to recruit a larger amount of muscle mass during the exercise movement. It was suggested that the recruitment of a larger volume of muscle resulted in an increase in the distribution of power output. Although the exact mechanism(s) responsible for the increase in economy and efficiency is(are) not known. Coyle (1995) postulated that a greater recruitment of muscle allows the active fibres to maintain a lower relative work rate and reduce the work rate per muscle fibre, consequently the individual energy requirement of each fibre is reduced. Coyle *et al.* (1988; 1991) found that years of endurance training was highly correlated with %$\dot{V}O_{2max}$ at TLac and

endurance performance during a 1-h time trial. It was proposed that an increase in muscle fibre recruitment and distribution of power output was a learned response over time. Therefore, cyclists with about 5 years cycling experience were able to produce a higher power output relative to O_2 consumed and had a high gross mechanical efficiency compared with less experienced cyclists.

Coyle *et al.* (1992) investigated various physiological factors which affect economy and gross mechanical efficiency during bicycling and showed that a relatively high percentage of slow-twitch muscle fibres is highly correlated with gross mechanical efficiency during cycling performance. Similarly, Horowitz *et al.* (1994) suggested that cyclists with a low level of performance $\dot{V}O_2$ and $\dot{V}O_2$ at LT but with a high percentage of type I muscle fibres maintain a high performance power due to their high level of economy and gross mechanical efficiency. Interestingly, Merrill and White (1984) found that gross mechanical efficiency during bicycling decreased from 25.6% to 21.2% with an increase in pedal speed from 60 to 90 rev·min^{-1}. Gaesser and Brooks (1975) suggested that increased recruitment of inefficient fast-twitch muscle fibres at higher pedal rates could explain the observed decrease in gross mechanical efficiency.

Very few studies have considered the effects of training-induced changes in respiratory muscles on improvements in cycling economy and efficiency. Although there is strong evidence to suggest that respiratory muscle training does not improve maximal exercise performance (Fairburn *et al.*, 1991; Morgan *et al.*, 1987) and that maximal ventilation is not limited by mechanical constraints (Mota *et al.*, 1999), Caine and McConnell (1998) recorded an increase in sub-maximal cycling performance following a resistive respiratory muscle training program.

1.2.2.2.2 *Values for economy and gross mechanical efficiency*

Several authors have measured economy and gross mechanical efficiency of endurance cyclists during cycling exercise. Coyle *et al.* (1991) and Horowitz *et al.* (1994) reported that the average economy of elite cyclists during a 1-h time trial performance ride was ~76 [(W·$\dot{V}O_2$ (L·min^{-1})]. Hickey *et al.* (1992) found that the economy of competitive cyclists during a 105 min performance ride averaged 70 [(W·$\dot{V}O_2$ (L·min^{-1})] and El-Sayed *et al.*

(1997) calculated that the economy of trained cyclists during a 1-h time trial averaged 68 [(W·$\dot{V}O_2$ (L·min^{-1})]. Nickleberry and Brooks (1996) reported that mean gross mechanical efficiency calculated for competitive cyclists (n = 6) was ~ 22-23% and Pugh (1974) found that gross mechanical efficiency for cycling was ~ 22% at high work loads (400 W) which decreased to ~ 17% for low levels of power (~100 W). Similarly, Nickleberry and Brooks (1996) showed gross mechanical efficiency was ~ 15-18% at low power outputs (50-100 W) when compared with ~ 22% at higher power outputs (250-300 W).

1.2.3 ROLE OF GENOTYPE

Various authors (for review see Bouchard *et al.*, 1992) have considered the effects of genotype on endurance performance. Although earlier studies by Hamel *et al.* (1986) and Prud'homme *et al.* (1984) suggested that changes in $\dot{V}O_{2max}$ in response to training were ~ 65-80% genotype-dependent, Bouchard *et al.* (1992) argued that the effect of genotype had been overestimated and suggested that endurance training can increase $\dot{V}O_{2max}$ by ~ 25% and that training indiced changes in $\dot{V}O_{2max}$ were ~ 40-60% genotype-dependent. The authors concluded that the magnitude of the increase in $\dot{V}O_{2max}$ was dependent on:- the initial level of fitness of the athlete and the training programme completed by the athlete. It is worth noting that Bouchard *et al.* (1986) argued that genetic factors have a stronger affect on oxidative capacity than $\dot{V}O_{2max}$ and Hamel *et al.* (1986) postulated that early adaptations to endurance training (over a period of ~ 6 to 12 months) were under less genetic control when compared with late improvements and that when an athlete approaches maximal trainability, determinants of successful endurance performance are more genotype-dependent.

Bouchard and Lortie (1984) explained that the performance ability of an elite endurance athlete is the integration of; prior endowment, a suitable and adequate endurance training program and genetic characteristics which make him/her responsive to high levels of endurance training.

1.3 SUMMARY

This chapter considered physiological factors which are associated with endurance performance ability. Although the precise mechanism(s) which limit endurance

performance is/are unclear, several models have been developed to discuss the importance of physiological variables such as $\dot{V}O_{2max}$, threshold exercise intensity and economy and efficiency. Coyle (1995) considered the interrelationships between these factors and put forward an integrated model to describe the determinants of performance (see Figure 1). Studies which have assessed interrelationships between these variables have found that when compared with more traditional assessments of endurance performance ability such as $\dot{V}O_{2max}$ and $\dot{V}O_2$ at threshold, maximal/peak aerobic power also described as maximal muscle power can also be used to predict endurance performance. In support of this, Noakes (1998) argued that exercise performance during maximal and sub-maximal exercise is determined by skeletal muscle function and not dependent on the attainment of high values for $\dot{V}O_{2max}$, $\dot{V}O_2$ at threshold or $\%\dot{V}O_{2max}$ maintained at race pace.

CHAPTER 2

2 EFFECT OF AGE ON DETERMINANTS OF CYCLING PERFORMANCE

2.1 INTRODUCTION

Ageing has been described as universal, decremental, progressive and intrinsic (Menard and Stanish, 1989) and the morphological and physiological conditions which constitute ageing are considerable and complex. In order to study the effects of ageing on physical performance investigators have assessed changes in muscular strength (Bemben, 1998; Frontera *et al.*, 1991), skill acquisition and motor control, (Etnier and Landers, 1998), anaerobic power (Chamari *et al.*, 1995; Ferretti *et al.*, 1994; Grassi *et al.*, 1991) and endurance performance (Joyner, 1993). For the purposes of this review, age-related declines in physiologic function which affect determinants of endurance performance are considered.

In order to examine the effects of ageing on physiologic function and exercise performance capacity studies have used cross-sectional (Hagberg *et al.*, 1985; Heath *et al.*, 1981; Millar, 1978) and longitudinal (Åstrand *et al.*, 1997; Kasch *et al.*, 1995; Pollock *et al.*, 1997; Trappe *et al.*, 1996) methods of investigation. Cross-sectional studies have the advantage of requiring less time and fewer resources, however choice of subjects may be heavily influenced by experimenter bias and not represent the subject population. To examine cross-sectional age-related declines in exercise performance authors have either studied large groups of athletes (Kavanagh and Shephard, 1990; Seiler *et al.*, 1998) matched subjects in terms of type of sport/training intensity, frequency and duration (Fuchi *et al.*, 1989; Hagberg *et al.*, 1985; Heath *et al.*, 1981; Rivera *et al.*, 1989) or compared older athletes with sedentary age-matched controls (Hagberg *et al.*, 1998; Stevenson *et al.*, 1994).

A recent paper by Seiler *et al.* (1998) has highlighted a significant problem associated with the analysis of cross sectional data. This was shown by examining the relationship between age and performance time for a group of 2487 male subjects during an annual indoor

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rowing competition. The age of the subjects ranged from 24-93 years. Analysis of the data revealed a weak relationship between age and performance time (r = 0.56). However when the top performers for each 2 year increment were identified and selected out, analysis of the data showed a strong relationship between age and performance time (r = 0.91). The authors postulated that age contributed more to the decline in endurance performance in elite rowers compared with mixed ability rowers. It was concluded that performance times obtained from large numbers of subjects across all ages can be significantly influenced by differences in physical stature, inherent endurance capacity, training habits, and competitive desire.

Longitudinal methods of analysis may provide a more valid assessment of structural, physiological and functional changes associated with the ageing process however, Stamford (1988), highlighted inherent methodological problems associated with the collection and interpretation of longitudinal data. These include:- isolation of the effects of ageing from other confounding factors; control and assessment of habitual activity during the period of the study; appreciation of the effects of chronic disease and illness on exercise performance capacity; subject mortality; accuracy of measurements of exercise performance capacity such as $\dot{V}O_{2max}$; reliability of equipment used to measure variables; and extrapolation of changes across intervening periods of time. Also exercise performance criteria such as field based personal best time may be influenced by external factors such as weather conditions, use of equipment and topography.

2.2 THEORIES OF AGEING

The exact underlying mechanism(s) responsible for age-related declines in physiologic function is/are not known and it is beyond the scope of this thesis to review the available literature concerning the various theories which have been developed to explain the ageing process. Briefly, theories to explain the ageing process have included the 'free radical', 'biologic clock' models and the 'genetic error' concept. The postulate that ageing is the result of error in the transcription of DNA has been discussed in detail elsewhere (Miquel, 1991) and the concept that ageing is under genetic control (biologic clock concept) has been considered by Robergs and Roberts (1996). Also, Merry (1999) recently discussed the theory that longevity is increased when calorie intake is restricted and hypothesised that an

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increase in lifespan was due to a decrease in free radical production. Consequently, damage to cellular components particularly mitochondrial protein and mtDNA was reduced. Robergs and Roberts (1996) concluded that ageing results from both internal and external factors and that death occurs due to the effects of external environmental forces such as infection and injury which can not be tolerated by the elderly individual.

2.3 ENDURANCE PERFORMANCE

There is very little information available concerning the decline in endurance performance associated with ageing. Numerous investigations (for review see Stamford, 1988; Spina, 1999) have assessed the endurance capacity of older athletes and sedentary individuals during progressive short term maximal exercise, however very few studies (Fuchi *et al.*, 1989; Grogan *et al.*, 1991; Seiler *et al.*, 1998) have considered the effects of ageing on actual competitive endurance performance.

An example of the decline in endurance performance ability with age is clearly shown in Figure 2. The Tour de France cycling race normally consists of 21 stages which take place over a three week period (~ 100-h of competition) during which cyclists are required to cover over 3500 km of level, uphill and downhill terrain (Lucia *et al.*, 1999; Padilla *et al.*, 1999). It is very likely that cyclists who are able to win this extreme test of physical performance possess morphological and physiological attributes which represent the limits of human potential therefore the finding that the highest number of Tour de France winners were aged 29, indicates that factors which affect performance are in decline beyond this age.

Various authors (Fuchi *et al.*, 1989; Grogan *et al.*, 1991; Joyner, 1993; McArdle *et al.*, 1991) have considered the effects of ageing on endurance performance time. Fuchi *et al.* (1989) estimated that the decline in running speed expressed as 10-km run velocity (m·min⁻¹) was 6% per decade for male runners (n = 49) aged between 30 to 80 yr. Grogan *et al.* (1991) used United States age records for 5-km, 10-km and Marathon running distances to calculate an age-related rate of decline in running velocity. A mathematical function was developed which predicted the highest achievable speed for each distance for runners aged between 30 and 90 yr. Analysis of the data revealed that in comparison to 10-km

performance time at 30 yr of age, declines in performance velocity would be 0.6% at age 40, 7.7% at age 50, 21.4% at age 60, 41.7% at age 70, 68.5% at age 80, and 101.8% at age 90. The authors also estimated that a faster rate of performance decline would occur during shorter running distances of 5-km and 10-km when compared with the Marathon.
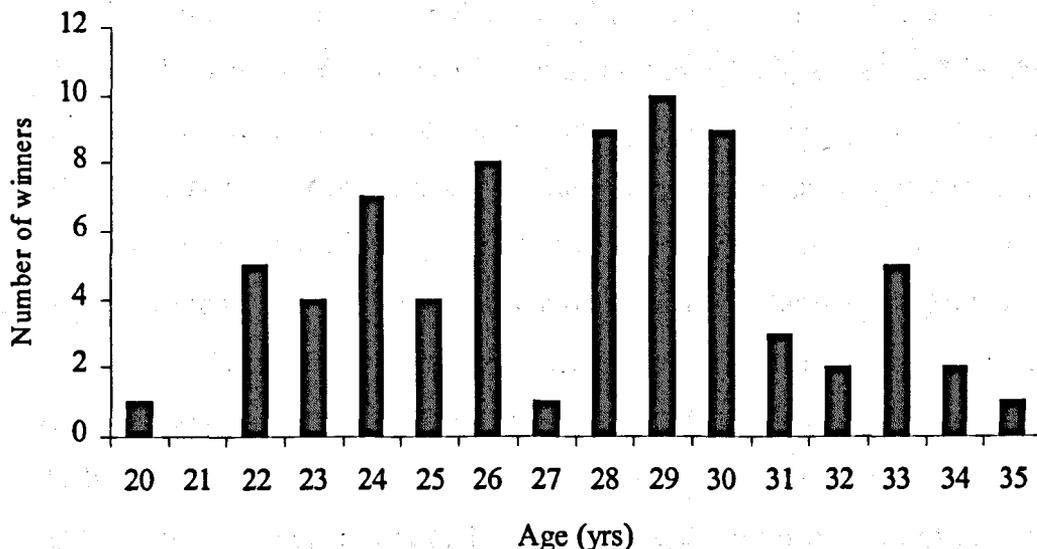
Figure 2. Relationship between age and number of cyclists who have won the 'Tour de France' cycling stage race (data from Cycling Plus, 1997)

Seiler *et al.* (1998) assessed the age-related decline in performance power (watts) during a 2500 m indoor rowing competition and calculated an average decline of 3.25 watts per year. Notably when the decline in performance of the top 2.5% of competitors was analysed data revealed a drop of performance power of 4 watts per year. The rate of decline in power output was slower from ages 30-55 and faster after the mid. 50s. The overall rate of performance decline equated to ~ 13% per decade with no difference in rate of decline for 800- and 10,000-m races.

For the purposes of this review the effect of age on cycling endurance performance was investigated. Road Time trial Council (1997) age records for 16.1-, 40.2-, 80.4-, and 160.9-km distances were compared and Table 3 shows the percentage change in cycling time trial performance was consistent across each distance at each age. The decline at age 40 was relatively small (~ 4%) and the decline in average speed between 50 and 60 years of age

was about 2%, however a dramatic decrease in speed occurred at the ages of 70 and 80. Figure 3 highlights the age related decline in cycling speed during individual cycling time trial races.

Measurement of changes in cycling speed may not provide a valid assessment of physiological declines in performance. Seiler *et al.* (1998) pointed out that power is a third-order polynomial function of velocity when moving through air, therefore "the change in muscular power output and the corresponding change in movement velocity of an object moving through air or water is not linear because of the exponential relationship between movement velocity and the resulting drag force acting on the object" (p. 126). At low power outputs a reduction in absolute power output due to the physiological effects of ageing will have a large affect on performance time, however at high power outputs a similar reduction in power will result in a relatively small change in performance (Seiler *et al.*, 1998). It is worth noting that the relationship between power and speed will be specific to each individual (di Prampero *et al.*, 1979) therefore the measurement of power output maintained during endurance performance races would provide a more valid assessment of age-related physiological decline. Unfortunately, there is very little information available concerning age-associated declines in laboratory or field based endurance performance power.

Table 3. Percentage change in average speed for RTTC competition records achieved during field based cycling time trials (data from RTTC, 1998)

Distance (km)	Senior record (km·h⁻¹)	Age (yr)				
		40	50	60	70	80
16.1	52.03	4.0*	10.6	12.4	23.2	36.2*
40.2	52.23	4.8	11.0*	12.3	24.5	41.6
80.4	49.31	4.1	7.8*	12.8	25.7	45.7
160.9	46.31	4.3*	9.5*	13.3*	20.5	47.5*

denotes estimation from interpolation of age record when value not provided by RTTC

Percentage changes for a given age were calculated by dividing the competition record by the age record and multiplying by 100

Average speed (km.hr^{-1}) vs Age (years)

...♦... 16.1 km TT

...■... 40.2 km TT

...●... 80.5 km TT
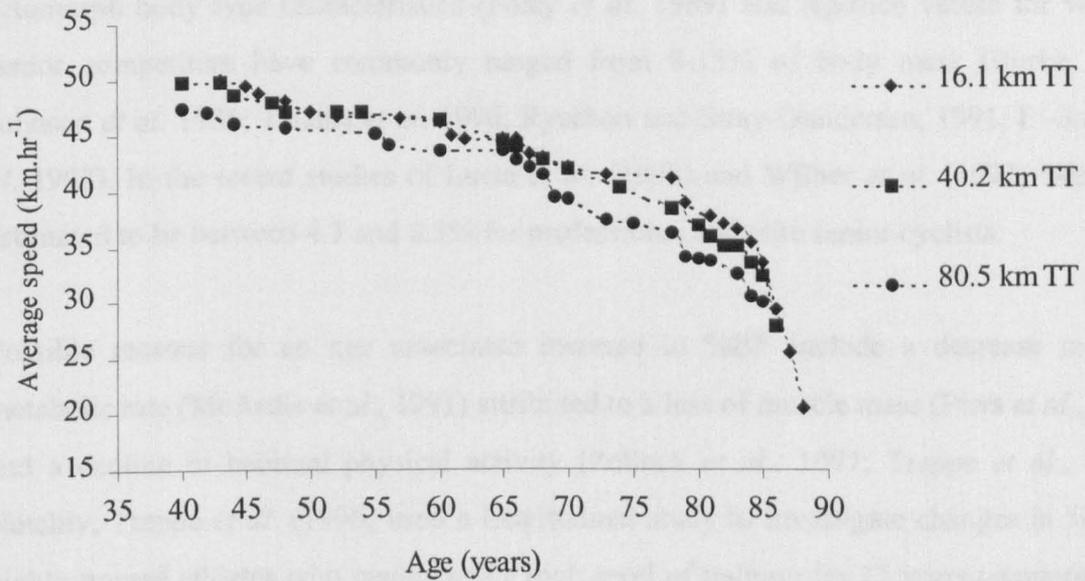
Figure 3. England and Wales road time trials council age records for field based cycling time trial performance (data from RTTC, 1998).

2.4 BODY COMPOSITION

Longitudinal studies (Marti and Howald, 1990; Pollock et al., 1997; Trappe et al., 1996) have assessed the percentage body fat (%BF) of senior and veteran athletes and found that %BF increased with age. Although a longitudinal study by Rogers et al. (1990) found no change in the %BF of veteran athletes who were assessed over a ten year period (mean age for test 1 was 60 yr) and a cross sectional study of Fuchi et al. (1989) reported that there was no change in %BF with an increase in age. Grassi et al. (1991) completed a cross-sectional comparison of %BF values recorded between senior and veteran competitors and found a progressive increase with age. Several cross-sectional studies have recorded higher values for %BF in veteran athletes when compared with senior competitors (Coggan et al., 1990; 1993; Proctor et al., 1995; Proctor and Joyner, 1997) whereas others have found no difference between seniors and veterans (Hagberg et al., 1985; 1988; Heath et al., 1981; Minson and Kenney, 1997) however values for %BF reported for veterans athletes have consistently been lower than age matched sedentary individuals (Hagberg et al., 1985; 1988; Heath et al., 1981; Rosen et al., 1998).

Anthropometric analysis of senior racing cyclists has shown that endurance cyclists exhibit ectomorph body type characteristics (Foley *et al.* 1989) and reported values for %BF in senior competitors have commonly ranged from 8-15% of body mass (Burke, 1980; Johnson *et al.* 1985; Tanaka *et al.* 1996; Ryschon and Stray-Gundersen, 1991; El-Sayed *et al.* 1997). In the recent studies of Lucia *et al.* (1998) and Wilber *et al.* (1997) %BF was estimated to be between 4.7 and 8.3% for professional and elite senior cyclists.

Possible reasons for an age associated increase in %BF include a decrease in basal metabolic rate (McArdle *et al.*, 1991) attributed to a loss of muscle mass (Piers *et al.*, 1998) and a decline in habitual physical activity (Pollock *et al.*, 1997; Trappe *et al.*, 1996). Notably, Trappe *et al.* (1996) used a longitudinal study to investigate changes in %BF of highly trained athletes who maintained a high level of training for 22 years compared with untrained individuals who maintained a low level of habitual physical activity for the same time period. Percentage BF increased from 8.7 to 21.6%, in the group with a low activity level, however it increased from 7.4 to 12.6% in the trained group. Further evidence to support the postulate that physical activity affects body composition was observed in a third group who maintained a moderate level of activity during the 22 year period; in this group %BF increased from 10.4 to 17.8%. Changes in physical activity appear to be the primary reason for the increased %BF reported in elderly populations. However different methods have been used to estimate %BF, therefore inter-study differences concerning equipment and techniques used to assess %BF need to be considered when comparisons are made between studies (Clarys *et al.*, 1987).

Several authors have suggested that increases in %BF relative to overall body mass are representative of a decrease in fat free mass (McArdle *et al.*, 1991; Stamford, 1988). Therefore, in order to account for age-related changes in body composition, research have expressed $\dot{V}O_{2max}$ in terms of $mL \cdot kg^{-1} \cdot min^{-1}$ or $mL \cdot kgFFM^{-1} \cdot min^{-1}$ (see Stamford, 1988). Various authors (Davies *et al.*, 1995; Heil *et al.*, 1997b; Neville *et al.*, 1992; Neville and Holder, 1995; Welsman *et al.*, 1996) have highlighted the methodological problems associated with ratio scaling and advised the use of allometric scaling when normalising $\dot{V}O_{2max}$ for differences in body size. Although this method has been applied to the study of

development, investigators have not applied similar scaling procedures to the study of age associated declines in maximal exercise capacity.

2.5 MAXIMUM OXYGEN UPTAKE

As previously discussed in chapter one of this thesis, maximal oxygen consumption is a key determinant of endurance performance, therefore declines in $\dot{V}O_{2max}$ recorded in elderly subjects are also associated with a reduction in exercise performance capacity. Numerous studies have measured $\dot{V}O_{2max}$ of sedentary and athletic populations across age groups and various authors have measured longitudinal age-related changes in $\dot{V}O_{2max}$.

Longitudinal studies which have measured the decrease in $\dot{V}O_{2max}$ with age have found an average absolute decline of 0.033 $L{\cdot}min^{-1}$ per year for an age range of 15-75 yr (Marti and Howald, 1990; Pollock *et al.*, 1997; Trappe *et al.*, 1996; Rogers *et al.*, 1990). Several authors have suggested that a sharper decrease in $\dot{V}O_{2max}$ occurs beyond the age of 60. For instance Pollock *et al.* (1997) found no difference in $\dot{V}O_{2max}$ of endurance runners who maintained a high level of training between the ages of about 50 and 60 yr (see Figure 4). However between 60 and 70 yr of age there was a large drop in $\dot{V}O_{2max}$ regardless of training. The authors argued that high levels of endurance training do not attenuate the age-related decline in $\dot{V}O_{2max}$. Trappe *et al.* (1996) used a longitudinal study to investigate the relationship between physical activity and age-related declines in $\dot{V}O_{2max}$. Analysis of the absolute rate of decline in $\dot{V}O_{2max}$ achieved by athletes who maintained high levels of training during the period of the study compared with athletes who maintained moderate levels of training showed there was no significant difference between groups (see Figure 4).

It is worth noting that the average absolute decline in $\dot{V}O_{2max}$ of 0.034 $L{\cdot}min^{-1}{\cdot}yr^{-1}$ calculated from cross-sectional studies of trained young and old individuals (Allen *et al.*, 1985; Aminoff *et al.*, 1996; Coggan *et al.*, 1990, 1993; Fuchi *et al.*, 1989; Grimby and Saltin, 1966; Hagberg *et al.*, 1985; 1988; 1998; Heath *et al.*, 1981; Massé-Biron *et al.*, 1992; Overend *et al.*, 1992; Posner *et al.*, 1987; Prefaut *et al.*, 1994; Proctor and Joyner, 1997; Proctor *et al.*, 1995; 1996; 1998; Rivera *et al.*, 1989) is very similar to the average absolute decline in $\dot{V}O_{2max}$ of 0.033 $L{\cdot}min^{-1}{\cdot}yr^{-1}$ calculated from longitudinal studies of athletes who maintained high levels of training for a long period of time (Marti and

Howald, 1990; Pollock *et al.*, 1997; Trappe *et al.*, 1996; Rogers *et al.*, 1990). The range of values for the rate of decline in VO_{2max} from cross sectional studies was calculated as 0.017 $L\cdot min^{-1}\cdot yr^{-1}$ (Aminoff *et al.*, 1996) to 0.069 $L\cdot min^{-1}\cdot yr^{-1}$ (Grimby and Saltin, 1966) and from the longitudinal studies was 0.018 $L\cdot min^{-1}\cdot yr^{-1}$ (Trappe *et al.*, 1996) to 0.047 $L\cdot min^{-1}\cdot yr^{-1}$ (Pollock *et al.*, 1997).
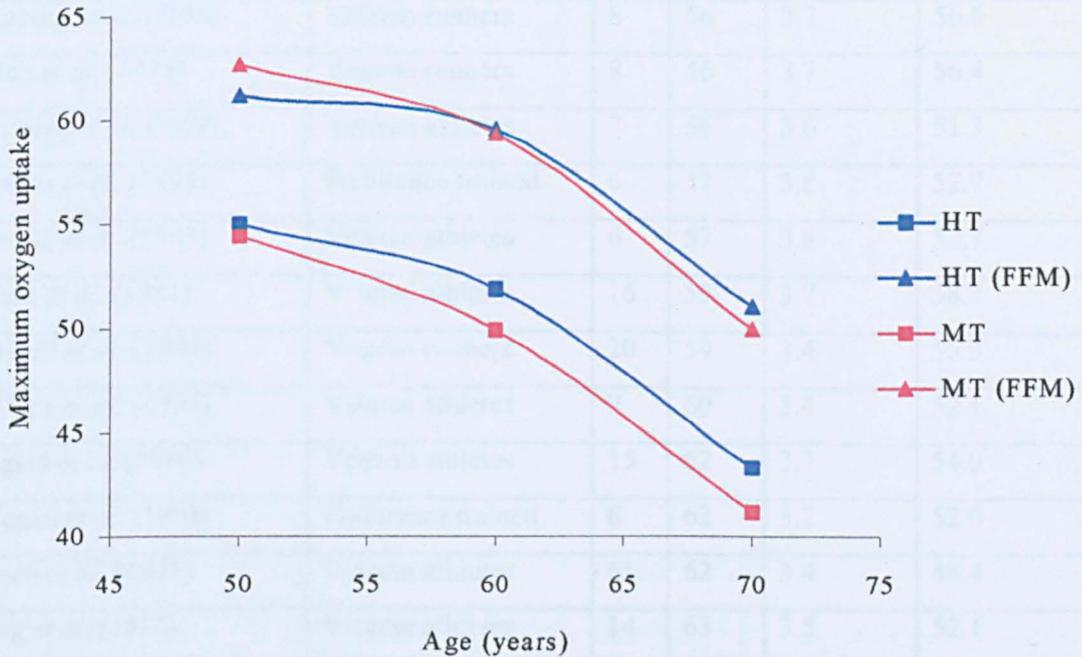
Figure 4. Longitudinal decline of VO_{2max}, $mL\cdot kg^{-1}\cdot min^{-1}$ and $mL\cdot kgFFM^{-1}\cdot min^{-1}$, assessed in athletes who maintained a high level of high (HT) and moderate (MT) level of training over a 20 year period (data from Trappe *et al.*, 1996)

When VO_{2max} was expressed relative to body mass, average absolute and relative (%) rate of decline calculated from the longitudinal studies was similar to rate of decline calculated from cross sectional work (0.52 vs 0.46 $mL\cdot kg^{-1}\cdot min^{-1}\cdot yr^{-1}$ and 0.90 vs 0.96 $\%\cdot yr^{-1}$, respectively). Tanaka *et al.* (1997) used a cross sectional study of healthy trained and untrained women to assess the rate of decline in VO_{2max} associated with ageing and found that the absolute rate of decline was higher in endurance trained women athletes when compared with sedentary controls (0.57 vs 0.32 $mL\cdot kg^{-1}\cdot min^{-1}\cdot yr^{-1}$), however the relative (%) rate of decline was similar (0.97 vs 0.91 $\%\cdot yr^{-1}$). The authors concluded that the higher absolute rate of decline (VO_{2max}) in highly physically active women was not related to age associated changes in maximal heart rate, body composition or training factors.

Table 4. Summary of data reported in the literature for $\dot{V}O_{2max}$ attained by veteran athletes

Study	Subjects	n	Age (yr)	$\dot{V}O_{2max}$ (L·min⁻¹)	$\dot{V}O_{2max}$ (mL·kg⁻¹·min⁻¹)
Pollock *et al.* (1997)	Veteran athletes	9	51	3.6	55.4
Barnard *et al.* (1979)	Veteran runners	13	55	3.5	54.0
Fuchi *et al.* (1989)	Veteran runners	15	55	2.8	51.5
Hagberg *et al.* (1985)	Veteran runners	8	56	3.7	56.6
Allen *et al.* (1985)	Veteran runners	8	56	3.7	56.4
Hagberg *et al.* (1998)	Veteran athletes	7	56	3.6	51.3
Proctor *et al.* (1995)	Endurance trained	6	57	3.8	52.7
Proctor *et al.* (1996)	Veteran athletes	6	57	3.8	52.7
Heath *et al.* (1981)	Veteran athletes	16	59	3.7	58.7
Maffuli *et al.* (1994)	Veteran runners	20	59	3.4	56.3
Pollock *et al.* (1997)	Veteran athletes	9	60	3.4	52.1
Rogers *et al.* (1990)	Veteran athletes	15	62	3.7	54.0
Coggan *et al.* (1993)	Endurance trained	6	62	3.2	52.0
Rosen *et al.* (1998)	Veteran athletes	61	62	3.4	48.4
Yerg *et al.* (1985)	Veteran athletes	14	63	3.5	52.1
Coggan *et al.* (1990)	Veteran runners	8	63	3.4	51.1
Prefaut *et al.* (1997)	Veteran cyclists	7	63	3.7	50.5
Fuchi *et al.* (1989)	Veteran runners	11	64	2.6	49.5
Proctor *et al.* (1998)	Endurance trained	8	64	3.5	45.9
Proctor and Joyner (1997)	Endurance trained	8	64	3.4	45.9
McLaren *et al.* (1995)	Veteran cyclists	10	65	3.9	54.0
Hagberg *et al.* (1988)	Trained runners	11	65	3.2	50.0
McLaren *et al.* (1995)	Veteran runners	11	65	3.5	48.0
Minson & Kenney (1997)	Veteran athletes	7	65	3.1	40.0
Prefaut *et al.* (1994)	Veteran cyclists	10	65	2.6	37.8
Massé-Biron *et al.* (1992)	Veteran cyclists	7	65	2.6	36.8
Rogers *et al.* (1990)	Veteran athletes	15	70	3.5	51.8
Pollock *et al.* (1997)	Veteran athletes	9	70	2.9	43.2
Fuchi *et al.* (1989)	Veteran runners	9	74	2.4	43.1

Cross sectional studies have reported higher values for $\dot{V}O_{2max}$ in veteran athletes when compared with age matched sedentary controls (Coggan *et al.*, 1993; Hagberg *et al.*, 1985; 1988; 1998; Heath *et al.*, 1981; Proctor *et al.*, 1996; Rosen *et al.*, 1998; Yerg *et al.*, 1985). However, $\dot{V}O_{2max}$ values recorded in veteran athletes have not exceeded the $\dot{V}O_{2max}$ of trained and physically active seniors (Allen *et al.*, 1985; Aminoff *et al.*, 1996; Coggan *et al.*, 1990; Hagberg *et al.*, 1985; 1988; Heath *et al.*, 1981; Massé-Biron *et al.*, 1992; Minson and Kenney, 1997; Prefaut *et al.*, 1994; Proctor and Joyner, 1997; Proctor *et al.*, 1995; 1998; Rivera *et al.*, 1989). In contrast to these studies, Proctor *et al.* (1996) and Coggan *et al.* (1993) reported lower values for $\dot{V}O_{2max}$ in senior aged subjects when compared with old competitors.

Using the data shown in Table 4, it is possible to predict the $\dot{V}O_{2max}$ of veteran athletes with reference to a particular age group. Regression equations for data presented in Table 4 based on a linear model are:- $\dot{V}O_{2max}$ (L·min^{-1}) = 5.81 - [0.04·age (yr)], r = 0.50, SEE = 0.37 (L·min^{-1}) and $\dot{V}O_{2max}$ (mL·kg^{1}·min^{-1}) = 91.57 - [0.67·age(yr)], r = 0.62, SEE = 4.46 (mL·kg^{1}·min^{-1}). Within these models, age accounted for 25 and 38% of the variance in $\dot{V}O_{2max}$ expressed as L·min^{-1} and mL·kg^{1}·min^{-1}, respectively. Therefore it is reasonable to assume that inter-individual differences concerning training status, previous training history, morphological and physiological ageing, as well as methodological considerations for the assessment of $\dot{V}O_{2max}$ affected maximal values recorded in these groups of athletes.

2.5.1 CENTRAL FACTORS

Studies which have investigated the physiological factors associated with age-related declines in $\dot{V}O_{2max}$ have found a strong relationship between a decrease in $\dot{V}O_{2max}$ and a reduction in maximal cardiac output (Fuchi *et al.*, 1989; Hagberg *et al.*, 1985; Heath *et al.*, 1981; Hunt *et al.*, 1998; Makrides *et al.*, 1990; Rivera *et al.*, 1989) and total blood volume (Hagberg *et al.*, 1998; Hunt *et al.*, 1998; Stevenson *et al.*, 1994). Hagberg *et al.* (1998) assessed the cardiovascular responses of trained and untrained elderly subjects during maximal exercise tests and found a higher stroke volume and total blood volume in veteran athletes when compared with age matched sedentary subjects.

However values for maximal cardiac output recorded in highly trained senior athletes consistently exceed values reported for veteran performers (Hagberg *et al.*, 1985; Rivera *et al.*, 1989). There is a plethora of literature available concerning the various central circulatory factors which affect exercise performance capacity. These issues are beyond the scope of this thesis, however an extensive review of the available literature concerning cardiovascular adaptations to endurance training in older men and women has recently been completed by Spina (1999).

Aminoff *et al.* (1996) provided strong evidence to suggest that during maximal exercise using large muscle groups, physical work capacity of elderly individuals is limited by central factors and that during maximal exercise using small muscle groups there is no difference in physical work capacity between young and old men. When subjects were required to complete a maximal cycling test, $\dot{V}O_{2max}$ was higher in the younger participants however when subjects completed one and two arm maximal cranking tests no difference was found between age groups. The authors concluded physical work capacity does not necessarily decline with age when exercise is more limited by peripheral rather than central factors.

2.5.1.1 HEART RATE

Maximal heart rate declines with age and age predicted heart rate is usually expressed as 220-age (Londeree and Moeschberger, 1982). Several cross-sectional and longitudinal investigations concerning age-related declines in performance have reported lower values for HR_{max} in elderly populations (for review see Spina, 1999). The underlying mechanism(s) responsible for the structural and functional changes in the older heart is/are unclear, however authors have suggested that the decline in HR_{max} is mediated by factors which control muscle contraction such as a down regulation of beta-adrenergic receptors which decreases the heart's sensitivity to catecholamine stimulation (Seals *et al.*, 1994) and changes in the volume of the sino atrial node and the number of pacemaker cells (Shiraishi *et al.*, 1992). Tate *et al.* (1994) noted that the underlying mechanism(s) responsible for the age associated decline in HR_{max} may not be intrinsic to cardiac muscle. For instance when elderly subjects experience ventricular arrhythmia, HR can exceed HR_{max} attained during a standard maximal exercise test.

Studies have shown that declines in HR_{max} with ageing are highly correlated with changes in $\dot{V}O_{2max}$ across all age groups (Hagberg *et al.*, 1985; Rivera *et al.*, 1989). However more recent work by Fitzgerald *et al.* (1997) has provided clear evidence to show that inter-individual differences in age-related declines in $\dot{V}O_{2max}$ do not necessarily correlate with declines in HR_{max}. Although significant relationships were found between HR_{max} and $\dot{V}O_{2max}$ in sedentary, active and endurance trained subjects (r = 0.85, 0.75 and 0.84, respectively), authors have postulated that 'other factors such as stroke volume and muscle oxidative capacity may have been responsible for differences in the absolute rates of decline in $\dot{V}O_{2max}$ observed in exercising and sedentary populations' (p. 164).

Using data available from longitudinal studies (see Table 5) it is possible to establish that the mean rate of decline in HR_{max} with age is about 0.73 b·min⁻¹·yr⁻¹ (Kasch *et al.*, 1995; Pollock *et al.*, 1997; Trappe *et al.*, 1996). Data from cross sectional studies suggest that across a broad range of subjects regardless of their activity status and previous history of training, heart rate declines at a very similar rate of about 0.60-0.70 b·min⁻¹·yr⁻¹ (Fuchi *et al.*, 1989; Proctor and Joyner, 1997). Tzankoff and Norris (1979) completed a large scale cross sectional assessment of age-related changes in HR_{max} recorded in untrained individuals and found that the average rate of decline was 0.91 b·min⁻¹·yr⁻¹. However, Fitzgerald *et al.* (1997) reviewed the available literature concerning cross sectional studies which assessed age-related declines in maximal aerobic capacity and HR_{max} in regularly exercising and sedentary women and found no difference (P>0.05) for the average rate of decline for HR_{max} (0.70 b·min⁻¹·yr⁻¹ for endurance trained athletes vs 0.79 b·min⁻¹·yr⁻¹ for sedentary subjects).

In a review of the affects of ageing on physiological factors which limit endurance running performance, Joyner (1993) cited the work of Rogers *et al.* (1990) to suggest that the decline in HR_{max} can be reduced with chronic exercise. When Rogers *et al.* (1990) assessed HR_{max} in veteran runners who maintained a high level of training over an eight year period (mean age of group was 62 years for test 1 and 70 years for test 2), analysis of the data revealed that there was no change in HR_{max} recorded for test 1 and 2 (171 vs 171 b·min⁻¹). However longitudinal (Kasch *et al.*, 1995; Pollock *et al.*, 1997; Trappe *et al.*, 1996) and cross-sectional (Fitzgerald *et al.*, 1997) studies have found no relationship between chronic

endurance exercise and an age-related decline in HR_{max} (see Table 5). Londeree and Moeschberger (1982) pointed out that mode of exercise, testing protocol and inter-individual differences in motivation can affect the highest HR recorded during maximal exercise and estimated that age can account for between 70-75% of the inter-individual variability in HR_{max}.

Table 5. Longitudinal age-related declines in HR_{max} recorded for groups of endurance trained runners and untrained controls

Study	n	Activity level	Age (yr)		HR_{max} ($b \cdot min^{-1}$)		Decline HR_{max} ($b \cdot min^{-1} \cdot yr^{-1}$)
			A	B	A	B	
Kasch et al. (1995)	12	Trained	42	71	180	158	0.63
	12	Untrained	48	69	175	143	0.58
Pollock et al. (1997)	9	Highly trained	51	70	173	160	0.74
	10	Trained	50	70	177	162	0.72
Trappe et al. (1996)	10	Highly trained	26	47	191	180	0.81
	18	Trained	27	49	186	174	0.79
	15	Untrained	23	45	187	178	0.81
Mean	12		38	60	181	165	0.73
± SD	3		12	12	7	13	0.09

A - trial 1, B - trial 2

2.5.1.2 STROKE VOLUME

Studies which have investigated the effects of exercise training on the aerobic capacity of elderly populations have consistently shown that improvements in $\dot{V}O_{2max}$ can be attributed to an increase in maximal stroke volume (Douglas and O'Toole, 1992; Ehsani et al., 1991; Stratton et al., 1994). Hagberg et al. (1998) found that during a maximal exercise test veteran athletes had a significantly higher stroke volume index ($mL \cdot m^2$) and cardiac index ($L \cdot min^{-1} \cdot m^2$) when compared with age matched sedentary individuals. These differences were attributed to a higher left ventricular end diastolic volume (LVEDV) index. The authors concluded that increased intravascular volumes, particularly plasma volume were

primary factors contributing to the higher $\dot{V}O_{2max}$, higher LVEDV, stroke volume and cardiac output during maximal exercise in endurance trained older men.

2.5.1.3 VENTILATION

Numerous investigations have found that static and dynamic lung function declines with age (see Åstrand and Rodahl, 1986). Although the exact physiological changes responsible for these reductions are unclear, it is generally acknowledged that an increase in residual volume as a percentage of total lung capacity due to a loss of lung elasticity (Johnson and Dempsey, 1991; Turner *et al.*, 1968) and static recoil pressure (Johnson *et al.*, 1991) are major contributors to an age-associated decrease in lung volume and maximum expiratory flow rate. An extensive discussion on this issue exceeds the scope of this thesis, however Johnson and Dempsey (1991) have provided a comprehensive review of the available literature and this reference can be used as a starting point for the reader who wishes to pursue this area further.

2.5.1.3.1 Static and dynamic lung volumes

Åstrand and Rodahl (1986) explained that static and dynamic lung volumes decrease with age however in the longitudinal study of Pollock *et al.* (1997) no difference in vital capacity (VC) was found in highly and moderately trained athletes when recorded over a period of ten years between the age of 50 to 60 years, although a marked decrease in VC was recorded in both groups at 70 years of age (see Table 6). Johnson and Dempsey (1991) noted that age-related changes in stature due to a decrease in invertebral spaces had a negative affect on lung volume. Several authors (Åstrand *et al.*, 1997; Millar, 1978; Pollock *et al.*, 1997; Prefaut *et al.*, 1994; Trappe *et al.*, 1996) have reported static and dynamic lung function in senior and veteran athletes, however very few studies (Hagberg *et al.*, 1988) have adjusted values to account for age-related changes in height.

Hagberg *et al.* (1988) found no difference in maximum voluntary ventilation (MVV) between veteran runners and age matched sedentary individuals (150 vs 153, L·min^{-1}, respectively). However, values for MVV recorded in older subjects were significantly lower than values recorded for senior runners and young sedentary subjects. In contrast to these findings Yerg *et al.* (1985) found that MVV recorded in veteran athletes was higher than age matched sedentary individuals (163 vs 144 L·min^{-1}, respectively). Millar (1978)

assessed the peak expiratory flow (PEF) of three groups of veteran athletes (mean age 44, 54 and 62 yr respectively) and found that (PEF) decreased with age (559, 549, 525 L·min⁻¹, respectively).

Table 6. Vital capacity (litres) recorded in veteran runners over a period of 20 years (Pollock *et al.*, 1997)

	Age (yr)		
	50	60	70
Highly trained runners (n = 9)	4.98	5.01	4.21
Moderately trained runners (n = 10)	5.45	5.45	4.53

Hagberg *et al.* (1988) commented that compared with values for $\dot{V}O_{2max}$ recorded in veteran athletes (which were about 38% above age matched sedentary individuals) values for lung volumes (vital capacity, total lung capacity) and pulmonary functions (maximum voluntary ventilation, $FEV_{1.0}$, $FEV_{1.0}$-to-vital capacity ratio) were about 8-18% higher than untrained subjects. It was postulated that the effects of prolonged training on pulmonary functions and lung volumes were not as strong as the effects of training on other physiological systems such as stroke volume.

2.5.1.3.2 *Maximal minute ventilation*

Several studies have recorded higher values for maximal ventilation ($\dot{V}_{Emax}$, L·min⁻¹) in veteran athletes when compared with age matched sedentary individuals (Hagberg *et al.*, 1985; 1988, Heath *et al.*, 1981; Yerg *et al.*, 1985) and have recorded similar $\dot{V}_{Emax}$ values in senior and veteran competitors (Heath *et al.*, 1981). Although values for $\dot{V}_{Emax}$ recorded in veteran athletes have exceeded those of sedentary individuals (Hagberg *et al.*, 1988) values for $\dot{V}_{Emax}$ recorded in young athletes have been significantly higher than $\dot{V}_{Emax}$ of veteran athletes (Aminoff *et al.*, 1996; Fuchi *et al.*, 1989; Grimby and Saltin, 1966; Hagberg *et al.*, 1985; Overend *et al.*, 1992; Proctor *et al.*, 1998; Rivera *et al.*, 1989). The rate of decline in $\dot{V}_{Emax}$ can be highly variable and unlike changes in HR_{max} appear to be strongly influenced by the habitual physical activity and training history of the individual (see Table 7).

Johnson *et al.* (1991) suggested that age-related changes in exercise performance may be related to declines in pulmonary function. Notably, McLaren *et al.* (1995) and Prefaut *et al.*

(1994; 1997) recorded hypoxemia in veteran athletes during maximal exercise and postulated that an increase in arterial desaturation could significantly affect the attainment of a high $\dot{V}O_{2max}$. However, there is no conclusive evidence to show that hypoxemia is more prevalent in highly trained veteran athletes than equally trained senior competitors (Joyner, 1993). Johnson et al. (1991) stated that 'although a sufficient hyperventilatory response was noted in the majority of subjects the cost of achieving this response due to mechanical limitations may have contributed to a reduced performance', (p. 975). The mean value for estimated cost of breathing was 12.7% $\dot{V}O_{2max}$ (range 6.7-23.2%). However the estimated cost of breathing was calculated from values recorded in young adults.

Table 7. The rate of decline in maximal minute ventilation ($L \cdot min^{-1} \cdot yr^{-1}$) calculated from longitudinal studies of trained and untrained senior and veteran subjects

Study	Activity level	n	Age (yr)		$\dot{V}_{Emax}$ ($L \cdot min^{-1}$)		Rate of decline ($L \cdot min^{-1} \cdot yr^{-1}$)
			T 1	T 2	T 1	T 2	
Kasch et al. (1995)	Trained	12	42	71	92	90	0.07
	Untrained	12	48	69	94	56	1.81
Pollock et al. (1997)	Highly trained	9	51	70	143	118	1.32
	Moderately trained	10	50	70	146	122	1.20
Trappe et al. (1996)	Highly trained	10	26	47	151	121	1.43
	Trained	18	27	49	130	109	0.95
	Untrained	15	23	45	136	111	1.14
Mean		12	38	60	127	104	1.13
± SD		3	12	12	24	24	0.54

$\dot{V}_{Emax}$ = maximal min ventilation ($L \cdot min^{-1}$)

Johnson and Dempsey (1991), suggested that lower values for $\dot{V}O_{2max}$ recorded in veteran athletes could be related to age associated changes in pulmonary function due to an increase in the oxygen cost of breathing during maximal work. The authors explained that the requirement for O_2 of the respiratory muscles may be higher in the veteran athlete due to the effects of an increase in physiological dead space and an increased stiffness of the chest wall, consequently during heavy work a redistribution of blood flow away from locomotor muscles would be required to fulfil the relatively higher demand for O_2 required

by the respiratory muscles. Dempsey *et al.* (1996) has argued that this 'steal effect' would not change $\dot{V}O_{2max}$ but would result in a change in maximum work rate. Therefore, a lower maximal power relative to $\dot{V}O_{2max}$ would be recorded in veteran athletes. There is very little information available to support this postulate.

Johnson *et al.* (1991) pointed out that the coincidence between ventilatory demand meeting $\dot{V}O_{2max}$ is remarkably similar between young and old, however due to the constraints imposed by the aged lung and chest wall, veteran athletes are unable to accommodate the high rates of ventilation needed to attain the $\dot{V}O_{2max}$ values recorded in highly trained seniors. Johnson *et al.* (1991) found that veteran athletes were able to achieve an appropriate alveolar hyperventilation for the metabolic demand of maximal exercise however Johnson and Dempsey (1991) speculated that 'the situation might be quite different during prolonged high intensity exercise during which the veteran athlete is required to sustain high ventilatory responses and pressure generation in the face of shortened inspiratory muscle length and a high metabolic cost of breathing'. There is very little information available concerning the affect of ventilatory work on high intensity endurance performance of veteran athletes.

2.5.1.3.3 *Ventilatory equivalent of O_2*

Ventilation (for a given amount of O_2) recorded during sub-maximal (Patrick *et al.*, 1983) and maximal exercise (Grimby and Saltin, 1966) is higher in the elderly. Similarly an increase in ventilatory equivalent for O_2 has been reported in longitudinal studies (Åstrand *et al.*, 1997; Kasch *et al.*, 1995; Pollock *et al.*, 1997) which have assessed age-related changes in ventilation over a long period of time. This finding is consistent with cross sectional studies which reported a significantly higher $\dot{V}_{Emax}/\dot{V}O_{2max}$ in veteran competitors (Fuchi *et al.*, 1989; Hagberg *et al.*, 1985; 1988; Heath *et al.*, 1981; Proctor *et al.*, 1998; Rivera *et al.*, 1989). In contrast to these studies, Trappe *et al.* (1996) completed a longitudinal assessment of elite endurance runners who had maintained a consistent level of high intensity endurance training over a period of 22 years and found that maximum ventilatory equivalent ($\dot{V}_{Emax}/\dot{V}O_{2max}$) decreased in this group. Inbar *et al.* (1993) reported relatively low values for $\dot{V}_{Emax}/\dot{V}O_{2max}$ in highly trained senior athletes and postulated that a low $\dot{V}_{Emax}/\dot{V}O_{2max}$ was indicative of a hypoventilatory response during maximal

performance. However the observation that $\dot{V}_{Emax}/\dot{V}O_{2max}$ increases with age suggests that veteran athletes exhibit a relatively higher hyperventilatory response.

A higher ventilatory equivalent suggests that the O_2 cost of breathing in older competitors could be relatively higher in veterans when compared with seniors. Stamford (1988) suggested that increases in ventilatory equivalent with age may be indicative of a greater response to CO_2 production in order to compensate for lower gas exchange efficiency, combined with an age-related reduced ability to regulate ventilation. Similarly, Brischetto et al. (1984) found that higher $\dot{V}_E/\dot{V}O_2$ recorded in elderly subjects was not produced by a higher anaerobiosis and/or arterial desaturation, but was influenced by the training status of the subjects. For instance Inbar et al. (1993) found that untrained subjects had a higher $\dot{V}_{Emax}/\dot{V}O_{2max}$ when compared with trained endurance athletes. The authors suggested that a low $\dot{V}_{Emax}/\dot{V}O_{2max}$ indicated efficient pulmonary function. Studies which have assessed the effects of training on pulmonary function in older individuals have consistently found a decrease in $\dot{V}_{Emax}/\dot{V}O_{2max}$ after training.

2.5.1.3.4 Respiratory exchange ratio

Studies which used a cross-sectional method of analysis to investigate the effects of ageing on cardio-respiratory responses during maximal exercise (Aminoff et al., 1996; Overend et al., 1992; Proctor et al., 1998; Tzankoff and Norris, 1979) consistently recorded a lower maximal respiratory exchange ratio (RER) for older groups of subjects compared with senior individuals. However although a similar trend was identified in the longitudinal study of Rogers et al. (1990) no difference in maximal RER was found by Pollock et al. (1997), who investigated the maximal exercise capacity of highly trained veteran athletes who maintained a high frequency and intensity of training between tests (about 19 yr).

2.5.1.4 BLOOD VOLUME AND HAEMOGLOBIN

Nakamura et al. (1989) found no relationship between age and red blood count, Hb and Hct, (r = -0.12, -0.26, -0.14, respectively). Similarly, Grimby and Saltin (1966) reported that there was no change in Hct and Hb with an increase in age and Proctor et al. (1998) found no difference in Hb concentration recorded for groups of young and old endurance trained men. Miller (1978) investigated the relationship between age and Hb concentration using a cross-sectional comparison of middle aged and veteran athletes and found no

difference across age groups. In the longitudinal study of Dill *et al.* (1967) Hb was assessed in 16 champion runners over an average period of 25 years and no change in Hb concentration was recorded during this period of time (r = 0.28). Hagberg *et al.* (1998) used lean body mass (kg) to match a group of veteran cyclists with sedentary individuals of similar age. Hct values recorded in the veteran cyclists were similar to values recorded for the age matched sedentary individuals, however plasma volume ($mL \cdot kg^{-1}$) red cell volume ($mL \cdot kg^{-1}$) and total blood volume ($mL \cdot kg^{-1}$) were all significantly higher in the veteran cyclists. Moderate relationships were observed between red cell volume, plasma volume and total blood volume and $\dot{V}O_{2max}$ (r = 0.68, 0.61, 0.70, respectively). A similar finding to Hagberg *et al.* (1998) was reported by Stevenson *et al.* (1994) who calculated that the correlation coefficient for total blood volume ($mL \cdot kg^{-1}$) and $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) was r = 0.79.

2.5.2 PERIPHERAL FACTORS

Stamford (1988) argued that declines in whole body $\dot{V}O_{2max}$ depend on changes in maximal cardiac output and maximal a-$\dot{V}O_2$ difference (Fick equation) and although there is little doubt that changes in maximal cardiac output have a significant affect on $\dot{V}O_{2max}$, a reduction in a-$\dot{V}O_2$ difference also occurs due to the age-related decline in muscle mass. Aminoff *et al.* (1996) recorded maximal $\dot{V}O_2$ and power output achieved during a maximal arm cranking exercise test and found no difference between values attained by veteran and senior performers. The authors concluded that age-related declines in physical capacity were more related to a decrease in cardiovascular performance and not a reduction in peripheral factors which influenced a-$\dot{V}O_2$ difference.

Muscle mass declines with age (Coggan *et al.*, 1993; Robergs and Roberts, 1996), however the exact mechanism(s) responsible for this reduction is/are not known. Age-related decreased muscle mass has been attributed to a decrease in habitual physical activity (Robergs and Roberts, 1996) and neuromuscular function, (Brooks and Faulkner, 1994) and possible causes for a reduction in the size of muscle fibres include deterioration of end-plate structures, impaired excitation-contraction coupling and a decrease in motor unit recruitment (Aoyagi and Shephard, 1992).

Brooks and Faulkner (1994) have suggested that age associated muscle atrophy and declines in muscle function are due to the effects of 'motor unit remodeling'. This process involves denervation of type II fibres and the re-innervation of type I fibres due to the turnover of synaptic connections which occur at the neuromuscular junction. Consequently, fibres which are not re-innervated undergo denervation atrophy. Sargeant (1994) postulated that this conversion may be due to a progressive loss of larger motorneurones, therefore muscle fibres supplied by larger motorneurones become denervated and re-innervation occurs from smaller motorneurones leading to a conversion to slow properties. It is difficult to assess whether these changes are a function of habitual physical activity and/or age-related changes in movement patterns which would require the recruitment of type II fibres.

Various investigations have found no difference between veteran and senior competitors for the percentage and cross sectional area of type I fibres (Coggan *et al.*, 1990; Proctor *et al.*, 1995) however, Proctor *et al.* (1995) found that type IIa and type IIb fibres were reduced in veteran athletes and age matched controls when compared with seniors. Also veteran athletes had a significantly higher %type I compared with senior sedentary subjects and age matched sedentary controls. No difference was found between senior and veteran athletes for %type I fibres. In the same study, %type IIb fibres were significantly lower in senior and veteran performers compared with their age matched controls, however there was no difference between %type IIa between trained and untrained subjects. Melichna *et al.* (1990) found that apparent gains in type I muscle fibre due to the effects of ageing were at the expense of type IIb and not type IIa fibres. Therefore age-related changes in muscle mass appear to be the result of atrophy of type IIb fibres. Coggan *et al.* (1992) found that an endurance training program increased the percentage of type IIa fibres with a concomitant decrease in type IIb fibres and there was no change in the percentage of type I fibres. However Melichna *et al.* (1990) found that endurance trained veteran athletes had a higher percentage type I fibre compared with sedentary age matched and senior controls. Similarly in untrained individuals, %type I muscle fibre gain with age was achieved at the expense of type IIb rather than type IIa fibre. In contrast to the work of Proctor *et al.* (1995), the authors found an age-related gain in %type I muscle fibre in endurance athletes. This was achieved at the expense of both type IIa and IIb fibres. Coggan *et al.* (1990)

found that an increase in %type I fibre in highly endurance trained senior athletes when compared with senior and veteran trained competitors was achieved at the expense of a significantly lower %type IIa fibres without a change in %type IIb fibres.

The relative type II fibre atrophy found in elderly trained and untrained subjects appears to be unaffected by prolonged endurance training (Proctor *et al.*, 1995). Notably there is strong evidence to suggest that endurance training in groups of senior and veteran athletes converts type IIb muscle fibres to type IIa and regardless of age, endurance training increases the oxidative capacity of type IIa fibres to levels found in type I fibres. Coggan *et al.* (1990) found that capillary density, number of capillaries and number of capillaries in contact with each muscle fibre were significantly less in veteran athletes compared with performance matched seniors. Similarly, Proctor *et al.* (1995) found that capillary density of type II muscle fibres were affected by age, although there was no difference between trained senior and veteran subjects when capillary data were expressed as capillary number per unit type II area. Proctor *et al.* (1995) concluded that prolonged endurance training did not prevent the age associated loss of muscle mass, or the relative decline in type II fibre area.

There is very little information available concerning the oxidative capacity of veteran cyclists, however Allen *et al.* (1985) found that the oxidative capacity of veteran runners was higher than younger runners who were matched on weekly training distance, pace and 10-km performance time. When maintaining 10-km race pace the relative exercise intensity ($\%\dot{V}O_{2max}$ and $\%HR_{max}$) of the veterans was significantly higher than the seniors.

The effect of age and training on oxidative capacity in groups of trained veteran and senior athletes and age matched controls was investigated by Proctor *et al.* (1995). These authors found that succinate dehydrogenase (SDH) and citrate synthase (CS) activity were similar between trained subjects and were significantly higher in trained groups compared with age matched controls. Similarly, Coggan *et al.* (1992) found that SDH, and β-hydroxyacyl-CoA dehydrogenase activity were higher in veteran athletes when compared with performance matched seniors and recorded a significant increase in SDH, CS, and β-

hydroxyacyl-CoA dehydrogenase activity in untrained older men after completion of an endurance training program.

It is worth noting that peripheral adaptations in veteran athletes are similar to those recorded in performance matched senior competitors, however they do not exceed values recorded in senior competitors with superior performance ability. Also, values for oxidative enzyme activity and capillary density are consistently lower in veteran athletes when compared with highly trained senior competitors (Coggan *et al.*, 1990).

2.6 MAXIMAL/PEAK POWER

Power output recorded during a maximal power output test declines with age (Hagberg *et al.*, 1998; Kavanagh and Shephard, 1990; Massé-Biron *et al.*, 1992; Overend *et al.*, 1992; Prefaut *et al.*, 1994; Seiler *et al.*, 1998) and the decrease in maximal power (W_{max}) recorded in veteran cyclists and untrained elderly subjects has been attributed to a decline in $\dot{V}O_{2max}$ (Aminoff *et al.*, 1996) and muscle function (Sargeant, 1994). Proctor *et al.* (1998) measured W_{max} in endurance trained veteran and senior cyclists and calculated that the rate of decline in W_{max} was about 3 watts per year. Very few studies have measured W_{max} in veteran cyclists, however a mean W_{max} of 250 W for a group of 7 veteran cyclists (mean age of 65 yr) was reported by Massé-Biron *et al.* (1992) and a mean value of 247 W for a group of 8 endurance trained cyclists (mean age of 64 yr) was reported by Proctor *et al.* (1998). Kavanagh and Shephard (1990) calculated that the absolute and relative age-related decline in W_{max} for 756 veteran competitors was about 3 $W \cdot yr^{-1}$ and 0.66 $\% \cdot yr^{-1}$.

Studies which have assessed the 'anaerobic' power of senior athletes during a 30-s Wingate sprint test have found moderate relationships between endurance performance and measures of both peak and mean power output recorded during the 30-s sprint (Craig *et al.*, 1993). However there is very little information available concerning the effects of ageing on anaerobic exercise performance (Chamari *et al.*, 1995; Grassi *et al.*, 1991). Notably Chamari *et al.* (1995) assessed the W_{max} and anaerobic power of senior and veteran cyclists and found that the age associated decline in W_{max} with age was much less pronounced than the reduction in 'anaerobic' peak and mean power achieved during brief intense intermittent sprints. Unfortunately the authors did not assess the interrelationship between these

variables. It was suggested that the decrease in anaerobic power may have been influenced by:- changes in muscle mass; a decrease in the proportion of type II muscle fibres; a loss of functioning motor units; and the predominantly aerobic endurance based training of the veteran group. Notably, Chamari *et al.* (1995) did not consider methodological problems associated with the measurement of 'anaerobic' peak power due to changes in flywheel inertia during acceleration and deceleration phases of sprint tests when using a friction braked cycle ergometer.

2.7 LACTATE THRESHOLD

Very few studies have investigated the effect of age on the use of blood lactate parameters to assess a threshold exercise intensity. Absolute values for power output, running speed and $\dot{V}O_2$ determined at lactate threshold, lactate turning point and OBLA have been shown to be much lower in veteran athletes however when expressed as a relative workload (%$\dot{V}O_{2max}$), values for TLac in untrained subjects (Iredale and Nimmo, 1997) trained cyclists (Massé-Biron *et al.*, 1992) and runners (Wiswell *et al.*, 2000) are higher in veterans. Similarly, values for %$\dot{V}O_{2max}$ at lactate turning point assessed in trained cyclists (Massé-Biron *et al.*, 1992) have been higher in veterans when compared with senior cyclists (85 vs 79% $\dot{V}O_{2max}$, respectively).

Allen *et al.* (1985) defined the lactate threshold as the steady state exercise intensity that elicited a 2.5 mmol·L^{-1} blood lactate concentration and found that %$\dot{V}O_{2max}$ at threshold was higher in a group of veteran runners when compared with a group of performance matched seniors (85 vs 79% $\dot{V}O_{2max}$, respectively). However no difference was found for %$\dot{V}O_{2max}$ at threshold between veteran runners and a group of highly trained senior competitors (85 vs 83% $\dot{V}O_{2max}$, respectively). The authors reported that when expressed relative to HR_{max}, HR at threshold was higher in the veteran group (91 vs 87% HR_{max}) when compared with the performance matched young runners.

Åstrand and Rodahl (1986) considered the effect of age on peak lactacidaemia and calculated that the age-related decline in peak blood lactate concentration was about 0.16 mmol·L^{-1}·yr^{-1}. However in the longitudinal study of Åstrand *et al.* (1997) there was no reduction in peak blood lactate concentration found between the mean age of 26 to 47 but

there was a significant decrease between the mean age of 47 and 59. Roecker *et al.* (2000) investigated an age-related change in peak blood lactate concentration recorded in a group of 7408 healthy participants on completion of an incremental running test. Analysis of the data revealed that the highest peak blood lactate concentration was recorded in the 20 to 25 yr age group and then declined slightly between 25 to 30 and 30 to 35 yr groups. No further decline was observed between 30 to 35 and 55 to 60 yr however, after 55 to 60 yr a pronounced decline was recorded in the 60 to 65 and 65 to 70 yr groups.

In the study by Massé-Biron *et al.* (1992) physically active elderly subjects (mean age 65) were unable to attain the lactate threshold defined as OBLA (fixed blood lactate concentration of 4 mmol·L^{-1}) and relative exercise intensity determined at a fixed blood lactate concentration of 2 mmol·L^{-1} was significantly higher in veteran cyclists when compared with trained senior cyclists (85 vs 55% $\dot{V}O_{2max}$, respectively). Similarly, Iredale and Nimmo (1997) found that relative exercise intensity at lactate threshold (TLac) was significantly higher in veteran untrained subjects when compared with senior sedentary individuals (83 vs 70% $\dot{V}O_{2max}$, respectively).

Although the exact mechanisms for higher TLac (%$\dot{V}O_{2max}$) are not known, possible reasons include:- a reduction in lactate production with age due to a decrease in β-adrenergic receptor sensitivity and changes in epinephrine sensitivity (Fleg *et al.*, 1985); a reduced lactate diffusion rate (Tzankoff and Norris, 1979); an age-related loss of muscle mass and reduction in the percentage of type II muscle fibres (Proctor *et al.*, 1995); and a reduced muscle glycogen content in the active muscle (Meredith *et al.*, 1989).

Coggan *et al.* (1990) postulated that lower levels of lactate dehydrogenase activity and higher respiratory enzyme activity found in veteran athletes would also favour a reduction in lactate production, and the well developed capillary supply and oxidative capacity of type I and type IIa fibres found in trained veteran competitors would also explain a higher lactate threshold when expressed as %$\dot{V}O_{2max}$. In support of this postulate, Seals *et al.* (1984) recorded a marked reduction in blood lactate concentration when elderly subjects were required to exercise at the same workload before and after an endurance training program. The authors hypothesised that the lower blood lactate concentration observed

after completion of the training was due to an increase in oxidative enzyme activity and an increased oxidation of fat, with a possible sparing of carbohydrate and muscle glycogen. Iredale and Nimmo (1997) suggested that the reduced blood lactate concentrations observed in older subjects during an incremental cycling exercise test may also have been due to a greater lactate clearance due to a higher sarcolemmal lactate transport capacity in type I muscle fibres.

Although there is very little information available concerning age-related changes in lactate kinetics (Tzankoff and Norris, 1979) and lactacidaemia (Massé-Biron *et al.*, 1992; Wiswell *et al.*, 2000) there is strong evidence to suggest that fixed blood lactate concentrations should not be used to identify and quantify a lactate threshold exercise intensity in elderly subjects. However further research is required to investigate the affect of age on blood lactate response during exercise performance. Very few studies have considered the affects of testing protocol and mode of exercise on blood lactate response in elderly populations.

2.8 ECONOMY AND GROSS MECHANICAL EFFICIENCY

Although various authors (Coyle, 1995; Bassett and Howley, 1997) have suggested that economy and gross mechanical efficiency are key determinants of successful endurance performance, there is very little information available concerning the effect of age on economy and gross mechanical efficiency in elderly athletes. In the study by Allen *et al.* (1985) no difference was found between senior and veteran athletes for economy calculated at a running speed of 188 $m \cdot min^{-1}$.

Data reported by Massé-Biron *et al.* (1992) showed that mean cycling economy (calculated from mean values for W_{max} and $\dot{V}O_{2max}$) was lower in a group of senior cyclists when compared with veterans (70 vs 78 [$W \cdot \dot{V}O_2 (L \cdot min^{-1})$], respectively). In contrast to this, data reported by Proctor *et al.* (1998) revealed that mean cycling economy calculated from values for W_{max} and $\dot{V}O_{2max}$ was higher in a group of senior athletes when compared with veterans (92 vs 82 [$W \cdot \dot{V}O_2 (L \cdot min^{-1})$], respectively).

2.9 SUMMARY

Exercise performance capacity declines with age due to changes in structural and functional abilities commonly associated with physical performance. Declines in exercise performance recorded in sedentary elderly populations can be attenuated by exercise training and values for $\dot{V}O_{2max}$ recorded in veteran athletes are significantly higher than values recorded in age matched untrained individuals. When compared with old 'non-exercisers', veteran athletes have lower values for %BF and higher values for stroke volume, pulmonary function, capillary density, oxidative enzyme capacity and %type I muscle fibre. However, structural and functional adaptations to training found in veteran athletes do not exceed improvements observed in senior competitors. Absolute exercise intensity determined at lactate thresholds declines with age, however relative exercise intensity at threshold expressed as $\%\dot{V}O_{2max}$ and $\%W_{max}$ is higher in untrained elderly when compared with young sedentary individuals. The exact mechanism(s) responsible for the age-related declines in exercise performance is/are not known, however a reduction in oxygen delivery to the exercising muscles and a loss of muscle function appear to be the main contributors to the reduction in maximal and endurance performance.

CHAPTER 3

3 CORRELATES TO CYCLING PERFORMANCE

3.1 INTRODUCTION

In order to identify key physiological determinants of endurance performance, investigators have assessed performance-related responses of competitive athletes during a wide variety of exercise tests. Consequently various physiological, anthropometric and morphological characteristics of athletic populations have been correlated with endurance performance and interrelationships between selected variables have been discussed (see Coyle, 1995).

3.2 VARIABLES ASSOCIATED WITH CYCLING PERFORMANCE

Several determinants of field based endurance cycling time trial performance are considered in chapter one of this thesis, these include maximal oxygen consumption ($\dot{V}O_{2max}$), maximal power (W_{max}), blood lactate threshold (TLac, OBLA, MLSS) and laboratory based cycling time trial performance. This chapter considers the validity of selected physiological variables to predict endurance performance.

3.3 MAXIMAL OXYGEN UPTAKE AND ENDURANCE PERFORMANCE

Since the early work of Hill and Lupton (1923) and Taylor *et al.* (1955) investigators have observed a strong relationship between $\dot{V}O_{2max}$ and endurance performance. For instance, Ramsbottom *et al.* (1987) found that $\dot{V}O_{2max}$ recorded in a heterogeneous group of trained runners was the best predictor of time to complete a field based 5-km run (r = -0.85). However in contrast to this work, Coyle *et al.* (1991) and Horowitz *et al.* (1994) assessed the relationship between $\dot{V}O_{2max}$ and cycling performance in homogenous groups of highly trained riders with similar values for $\dot{V}O_{2max}$ and found that $\dot{V}O_{2max}$ could not be used to explain differences in cycling time trial performance.

3.4 MAXIMAL POWER AND ENDURANCE PERFORMANCE

Noakes (1998) stated that "in athletes of similar abilities the best predictor of performance is usually a measure of the maximal workload achieved during exercise" (p. 1392-1393). In support of this postulate, several authors (Bishop *et al.*, 1998; Dobbins, 1996; Jones and

Doust, 1998; Noakes *et al.*, 1990; Scott and Houmard, 1994; Weston *et al.*, 1997) have found that a measure of maximal sustained power (described as W_{max} for cyclists and V_{max} for runners) during a progressive exercise test to volitional exhaustion can provide a more accurate prediction of endurance performance than $\dot{V}O_{2max}$. Furthermore, Wood *et al.* (1997) assessed the relationship between W_{peak} and average power sustained during a laboratory based simulated 16.1-km cycling time trial. Analysis of the data revealed that performance in the time trial was strongly related to W_{peak} ($r = 0.96$). Similarly, Davison *et al.* (1999) considered the relationship between W_{peak} and outdoor cycling time trial power for events ranging from 16.1-km to 12-h. Analysis of the data revealed an underlying relationship between W_{peak} and cycling performance. Padilla *et al.* (1999) concluded that differences in cycling performance between professional cyclists were due to differences in absolute power at W_{max} and Bishop *et al.* (1998) argued that the strong correlation between W_{max} and average power output maintained during a laboratory based cycling time trial was evidence to suggest that "the time-consuming and costly analysis of lactate was not necessary for the prediction of 1-h endurance performance" (p. 1274).

3.5 LACTATE THRESHOLD AND ENDURANCE PERFORMANCE

Several investigators (Coyle *et al.* 1988, 1991; Craig *et al.* 1993; Ramsbottom *et al.* 1992) have found that exercise intensity at lactate threshold provides a more valid assessment of endurance performance capacity than $\dot{V}O_{2max}$. In the study of Farrell *et al.* (1979) the relationship between race pace and running velocity determined at TLac (originally described as OPLA) was stronger than the relationship between race pace and $\dot{V}O_{2max}$ (see Table 8).

Evidence that the relationship between endurance performance and $\dot{V}O_{2max}$ is relatively weak was also provided by Coyle *et al.* (1983) who compared values for $\dot{V}O_{2max}$ and %$\dot{V}O_{2max}$ sustained during an 8-km run by normal trained subjects and patients with ischemic heart disease (IHD). Running speed was the same between groups, however subjects with IHD maintained a mean exercise intensity which corresponded with 100% $\dot{V}O_{2max}$. This value was significantly higher than the 84% $\dot{V}O_{2max}$ recorded in the normal subjects. It was suggested that IHD had attenuated maximal cardiac output which limited O_2 delivery and reduced $\dot{V}O_{2max}$. Therefore during the 8-km performance test both groups

of subjects maintained a running speed and $\dot{V}O_2$ which was relative to lactate threshold and not $\dot{V}O_{2max}$.

Table 8. Correlation coefficients (r) between race pace ($m \cdot min^{-1}$), maximal oxygen consumption ($mL \cdot kg^{-1} \cdot min^{-1}$) and running velocity at lactate threshold ($km \cdot h^{-1}$)

	42.2-km	19.3-km	15-km	9.7-km	3.2-km
$\dot{V}O_{2max}$	0.91	0.91	0.89	0.86	0.83
V@TLac	0.98	0.97	0.97	0.96	0.91

$\dot{V}O_{2max}$ - maximal oxygen consumption ($mL \cdot kg^{-1} \cdot min^{-1}$)

V@TLac - running speed ($km \cdot h^{-1}$) at lactate threshold

(data from Farrell *et al.*, 1979)

Coyle *et al.* (1988; 1991) found that a measurement of $\dot{V}O_2$ at LT was a stronger predictor of endurance performance than $\dot{V}O_{2max}$. Coyle *et al.* (1988) matched two groups of highly trained cyclists on $\dot{V}O_{2max}$, however %$\dot{V}O_{2max}$ at LT was significantly different. When subjects were required to exercise at a fixed workload (88% $\dot{V}O_{2max}$) to the point of volitional exhaustion, riders with a higher %$\dot{V}O_{2max}$ at LT exercised for a significantly longer period of time. In the study by Coyle *et al.* (1991) elite (n = 9) and highly trained (n = 6) cyclists with similar $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) completed a laboratory based 1-h performance ride and 40-km field based time trial. Analysis of the data for all of the subjects (n = 15) revealed the best predictor of average power maintained during the 1-h performance ride was $\dot{V}O_2$ ($L \cdot min^{-1}$) at LT (r = 0.93) and not $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) (r = 0.85). Unfortunately, Coyle and co workers were unable to record power output maintained by the riders during the field based time trial and values for maximal power and power output recorded at LT were not reported. Notably, correlation coefficients calculated between average $\dot{V}O_2$ ($L \cdot min^{-1}$) maintained during the 1-h performance ride and $\dot{V}O_2$ ($L \cdot min^{-1}$) at LT and $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) were relatively similar (r = 0.87 and 0.84, respectively).

Although Coyle *et al.* (1991) found that riders (n = 9) with a higher %$\dot{V}O_{2max}$ at LT were able to maintain a higher average absolute power during the 1-h performance ride and higher average speed during the field based 40-km time trial, the best predictor of average

speed for the 40-km time trial for this group was $\dot{V}O_{2max}$ (r = 0.94) and not $\dot{V}O_2$ at LT (r = 0.83) or average power achieved during the laboratory based ride (r = 0.87).

Wood *et al.* (1997) compared power output at OBLA and W_{peak} with average recorded power during a simulated 16.1-km cycling time trial and found the relationship between W_{peak} and power (r = 0.96) during the time trial was higher than for power at OBLA (r = 0.90). Furthermore when power at OBLA and average power during the time trial were expressed as $\%W_{peak}$ (to remove the influence of W_{peak}) there was no correlation found between OBLA and time trial power. It was concluded that the strong relationship observed between power at OBLA and performance power was a consequence of the relationship between OBLA and W_{peak}.

More recently, Bishop *et al.* (1998) used a group of trained female cyclists to assess the relationship between average power maintained during a 1-h laboratory based cycling performance ride and $\dot{V}O_{2max}$, W_{max} and power output recorded at lactate threshold. Notably several 'descriptors' were used to determine lactate threshold, these were LT, TLac, OBLA and D_{max} and the best predictors of 1-h performance power were power output at D_{max} (r = 0.84) and W_{max} (r = 0.81) but not $\dot{V}O_{2max}$ (r = 0.55). In a study by Nichols *et al.* (1997) the relationships between $\dot{V}O_{2max}$ (expressed as $mL \cdot kg^{-1} \cdot min^{-1}$ and $L \cdot min^{-1}$) and time to complete field based 13.5 and 20-km cycling time trials were relatively high (r = -0.85 for 13.5-km and r = -0.83 for 20-km) however the best predictor of time to complete the time trials was power output at LT (r = 0.91 and 0.88 for 13.5 and 20-km races respectively).

Jones and Doust (1998) completed a similar study to Bishop *et al.* (1998) but used a group of trained runners to assess the relationship between average running speed during an 8-km race and $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and running velocity ($km \cdot h^{-1}$) determined for MLSS, $\dot{V}O_{2max}$, TLac, OBLA, T_{vent} and LMS. The authors argued that the strongest relationship between a threshold 'descriptor' and running performance was running velocity at MLSS and described velocity at MLSS (V@MLSS) as the 'gold standard' criterion measure for the assessment of endurance capacity. However a reference for this conjecture was not provided. One possible explanation for this assertion involved the work of LaFontaine *et al.* (1981) who found that the relationship between V@MLSS and 8-km running pace in a

group of trained runners (n = 7) was r = 0.995. Jones and Doust (1998) assessed the performance of a homogenous group of trained runners and calculated that the correlation coefficients for V@MLSS and V@$\dot{V}O_{2max}$ when compared with 8-km pace in a homogenous group of trained runners were r = 0.92 and r = 0.94, respectively. However this finding did not support the premise that the V@MLSS represented the 'gold standard' measurement of endurance exercise capacity. It is worth noting that although Jones and Doust (1998) reported that the relationship between V@TLac and V@MLSS was very high (r = 0.94) the correlation between V@$\dot{V}O_{2max}$ and V@MLSS was also very high (r = 0.94) unfortunately this relationship was not reported.

The relationship between $\dot{V}O_{2max}$ and running performance can be affected by the fitness level of the subject and the duration of the test used to evaluate performance (Tanaka, 1990). For instance, Tokmakidis and Leger (1992) found that the correlation between average running speed and $\dot{V}O_{2max}$ improved from r = 0.22 to r = 0.66 and the relationship between average running speed and LTP increased from 0.55 to 0.91 for 10-km and Marathon distances, respectively. Padilla et al. (1999) recommended that W_{max} values recorded during an incremental test could be used to predict cycling performance in short distance time trials on level terrain, whereas exercise intensity at LT and OBLA were more appropriate predictors of performance in longer distance time trials and uphill cycling.

In order to investigate the validity of W@LMP to predict performance power during a laboratory based 16.1-km cycling time trial. Davison et al. (1997) assessed the relationship between W@LMP determined during the protocol of Brennan et al. (1996) with W@LMP assessed during an adapted Lac_{min} test. This test differed from the procedure of Brennan et al. (1996) with starting intensity for the second part of the test calculated as 60% of W_{peak} recorded during the PP test. A stronger relationship was found between time trial power and W@LMP determined during the protocol of Davison et al. (1997) when compared with the W@LMP derived from the method used by Brennan et al. (1996). Tegtbur et al. (1993) assessed the validity of V@LMP to predict V@MLSS and found that blood lactate concentration did not change during an 8 km running time trial completed at V@LMP. The authors concluded that the lactate minimum test could be used to prescribe training and competitive exercise intensity and provided an objective assessment of a threshold exercise

intensity which avoided methodological issues concerning the effects of pre-exercise diet and glycogen depletion on blood lactate concentration during progressive exercise tests. Jones and Doust (1998) used a similar testing procedure to Tegtbur *et al.* (1993) to investigate the validity of V@LMP to predict V@MLSS and found that V@LMP could be used to predict V@MLSS however the best predictor of V@MLSS was V@TLac.

Tanaka (1990) concluded that the most critical determinant of endurance exercise performance (such as time to complete a Marathon running race) was running velocity at which TLac occurred, however running velocity at OBLA provided the best predictor of performance in endurance events of 16-km or less.

3.6 LABORATORY AND FIELD BASED ASSESSMENTS OF CYCLING PERFORMANCE

Few investigations have assessed the relationship between indoor and outdoor cycling time trial performance. Coyle *et al.* (1991) found a strong relationship between average power recorded during an indoor 1-h performance ride and personal best 40-km cycling time trial time ($r = -0.88$) and Palmer *et al.* (1996) found that time to complete an indoor Kingcycle simulated 40-km cycling time trial was strongly related ($r = 0.98$) to mean time to complete two separate outdoor 40-km time trials performed on different 40-km time trial courses.

3.7 SUMMARY

Studies which have investigated the determinants of cycling endurance performance have consistently found a strong relationship between maximal power determined during a progressive test to volitional exhaustion and cycling time trial performance time assessed outdoors and cycling time trial performance power indoors. Oxygen uptake and/or power at a designated blood lactate threshold exercise intensity also provide strong predictors of laboratory and field based cycling performance. The relationship between $\dot{V}O_{2max}$ and endurance performance is unclear. However, relationships between endurance performance and selected physiological variables appear to be influenced by the mode of exercise, time to complete the performance task, heterogeneity of the sample and training status of the subject.

CHAPTER 4

4 PERFORMANCE RELATED RESPONSES DURING FIELD AND LABORATORY BASED CYCLING TESTS

4.1 INTRODUCTION

Descriptive data concerning performance related responses recorded during laboratory and field based cycling tests provides useful information when comparisons are made between studies and changes in performance are assessed over a period of time. Several studies have assessed endurance performance using laboratory based simulated race protocols in order to extrapolate data to outdoor trials, however it is important to note that laboratory based protocols may not always provide an ecologically valid assessment of field based performance. In support of this postulate Kenny *et al.* (1995) commented that,

> "while the conditions of rolling and wind resistance, thermal and humidity conditions, and the metabolic costs of maintaining balance or overcoming inertial characteristics of different apparatus are more difficult to control, they should be accounted for in performance comparisons between field and laboratory settings",
> (p. 410).

This chapter reviews the available literature concerning performance related responses recorded during cycling endurance performance tests.

4.2 PERFORMANCE $\dot{V}O_2$

Coyle (1995) described performance $\dot{V}O_2$ as 'the maximal rate of whole body aerobic energy expenditure as measured by oxygen consumption which can be sustained for the duration of an endurance event' (p. 27). Several studies have investigated performance $\dot{V}O_2$ during laboratory based cycling endurance performance tests however there is very little information concerning performance $\dot{V}O_2$ of endurance athletes during field based races. Coyle *et al.* (1991), El-Sayed *et al.* (1997) and Horowitz *et al.* (1994) reported performance $\dot{V}O_2$ during a laboratory based 1-h ride was 4.54, 3.98 and 4.48 $L \cdot min^{-1}$, 90, 87 and 88% $\dot{V}O_{2max}$ respectively and Hickey *et al.* (1992) found that cyclists were able to maintain 77% $\dot{V}O_{2max}$ (3.50 $L \cdot min^{-1}$) during a 105-min performance ride. In the study by Langenfeld *et al.*

(1994) mean performance $\dot{V}O_2$ was about 70% $\dot{V}O_{2max}$ for a simulated 129-km cycling time trial (mean time of completion for the 129-km was 247 min).

4.3 PERFORMANCE POWER

Performance power is defined as the rate of energy output or power production generated for a specific period of time expressed in watts (Coyle, 1995). Several investigators (El-Sayed *et al.* 1997; Robinson *et al.* 1995; Wagenmakers *et al.* 1996) have assessed the average power output maintained by trained cyclists during laboratory based simulated cycling time trials (see Table 9). Robinson *et al.* (1995) and Wagenmakers *et al.* (1996) found that during a 1-h ride trained cyclists maintained a mean performance power of 298 and 294 W, 68.8 and 75.6% of maximal power respectively. Lindsay *et al.* (1996) calculated that trained cyclists sustained an average power output of 301-326 W, 72.1-75.0% W_{max} during a lab-based 40-km time trial (average time to complete the 40-km distance was ~ 56 min) and Coyle *et al.* (1991) and Horowitz *et al.* (1994) found that elite cyclists were able to maintain an average performance power of ~ 340 W during a 1-h laboratory based cycling time trial. There is very little information available for performance power recorded outdoors.

Table 9. Cycling time trial performance during laboratory based tests

Study	Time (min:s)	Distance (km)	Speed (km·h⁻¹)	W_{max} (W)	W_{TT} (W)	%W_{max}
Wagenmakers *et al.* (1996)	60:09	-	-	390	298	76.4
Robinson *et al.* (1995)	60:00	43.10	43.10	433	303	70.0
Hawley *et al.* (1997)	60:00	41.16	41.16	402	266	66.2
El-Sayed *et al.* (1997)	60:00	40.41	40.41	392*	277	70.7
Palmer *et al.* (1996)	55:51	40.00	42.97	443	304	68.6
Palmer *et al.* (1996)	27:01	20.00	44.42	443	328	74.0
Palmer *et al.* (1997)	26:32	20.00	45.23	432	340	78.7
Palmer *et al.* (1998)	27:41	20.00	43.35	400	311	77.8
Wood *et al.* (1997)	23:18	16.09	41.43	359*	279	77.7
Davison *et al.* (1997)	22:55	16.09	42.13	355*	288	81.1

W_{max} - maximal power output

* - peak power assessed during a Kingcycle PP test

W_{TT} - average power output recorded during time trial.

4.4 PERFORMANCE HEART RATE

Several investigators have recorded heart rate response during cycling performance rides and calculated average heart rate (expressed as $\%HR_{max}$) for the duration of the race, (see Table 10). Selley *et al.* (1995) assessed the relative HR response of runners during competitive races and found that a similar $\%HR_{max}$ was maintained during the race regardless of running speed and performance ability.

Table 10. Mean heart rate response recorded during cycling time trial races

Study	TT Performance (km)	$\%HR_{max}$
Robinson *et al.* (1995)	43.1$_{IN}$	91.6
Neary *et al.* (1995)	40.0$_{IN}$	92.6
Lindsay *et al.* (1996)	40.0$_{IN}$	90.7
El-Sayed *et al.* (1997)	41.2$_{IN}$	91.1
Palmer *et al.* (1996)	40.0$_{IN}$	90.3
Palmer *et al.* (1996)	20.0$_{IN}$	92.0
Palmer *et al.* (1997)	20.0$_{IN}$	92.5
Palmer *et al.* (1998)	20.0$_{IN}$	90.5
Palmer *et al.* (1994)	16.1$_{OUT}$	94.1
Dobbins (1996)	16.1$_{OUT}$	93.7

IN - indoor laboratory based time trials

OUT - outdoor field based time trials

$\%HR_{max}$ - percentage of maximal heart rate

4.5 PERFORMANCE SPEED

Performance speed is the average speed a cyclist maintains over a set distance and is a function of performance power (propulsion) and the forces that present resistance to movement (drag), (Coyle, 1995). Due to the affects of variations in terrain, riding style and race tactics on the relationship between performance speed and power output, performance velocity does not necessarily provide an accurate measure of work rate maintained during cycling exercise (Jeukendrup and van Diemen, 1998). However, an individual cycling time trial (described as the 'race of truth,' by Borysewicz, 1985) requires each rider to cover a set distance in the fastest time and therefore has been used by several authors (Lindsay *et al.*,

1996; Westgarth-Taylor *et al.*, 1997) to assess endurance performance ability. Indeed, the distance covered during a 1-h time trial has been described as the 'blue riband' event of cycling performance (Fotheringham, 1994). Time trial performance is dependent on a multiple of variables and circumstances (Padilla *et al.*, 1999) however a reasonable illustration of the effect of race distance on performance velocity can be obtained from time trial competition records (see Figure 5).
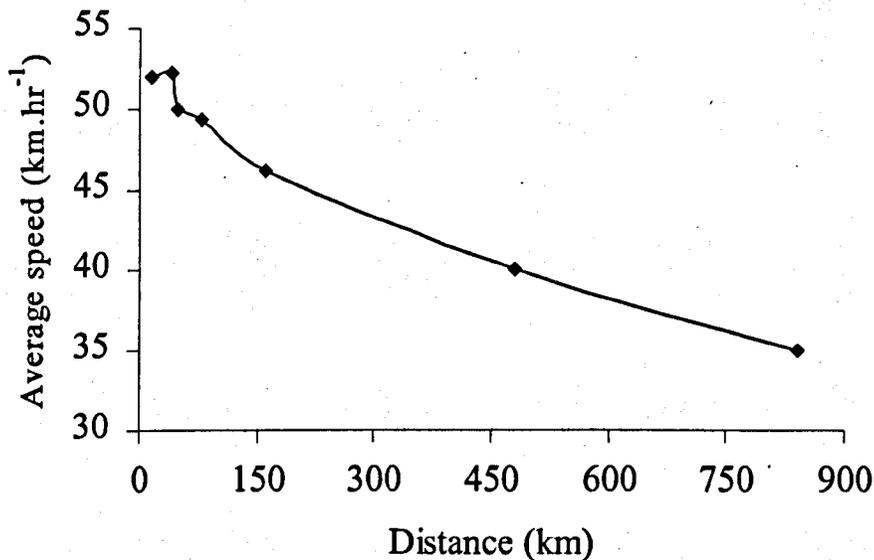
Figure 5. Depicts average speed (km·h[-1]) achieved for Road Time trials Council (RTTC) competition records during 16.1-, 40.2-, 48.3-, 80.5-, 160.9-km and 12- and 24-h time trial races (data from RTTC, 1998)

4.6 PERFORMANCE CADENCE

There is very little information available concerning spontaneously chosen pedal cadence (rev·min[-1]) during laboratory and field based cycling time trials. Sargeant (1994) included data for average cadence recorded during world record 1-h time trials performed on a cycling track. Pedal cadence ranged from 102 to 106 rev·min[-1], with cyclists using a track bicycle and single 'fixed wheel' gear ratio. This provided a continuous pedalling motion during the ride. and consequently track based data may not be transferable to 'road-based' time trials. Surprisingly there is no information available concerning pedal cadence maintained during field based cycling time trials.

4.7 Performance related responses of veteran athletes

There is very little information available concerning the performance related responses of veteran athletes during laboratory and field based endurance performance trials. Allen *et al.* (1985) found that when exercising at 10-km running pace veteran runners maintained a higher $\%\dot{V}O_{2max}$ and HR_{max} when compared with young runners with superior performance ability (92 vs 89% and 95 vs 92% respectively) however these differences did not reach the level of significance. Notably when the authors compared the veteran group with seniors who had a similar mean 10-km performance time, veterans maintained a significantly higher $\%\dot{V}O_{2max}$ when running at 10-km race pace (92 vs 81% $\dot{V}O_{2max}$, respectively).

Relative exercise intensity at lactate threshold (exercise intensity at a fixed blood lactate concentration of 2.5 $mmol\cdot L^{-1}$) was significantly higher in the veteran group, (85 vs 79 $\%\dot{V}O_{2max}$) when compared with the performance matched seniors and at race pace veterans maintained a higher relative exercise intensity (7 vs 2% above lactate threshold expressed as $\%\dot{V}O_{2max}$). No difference ($P>0.05$) was found between the veterans and superior performance senior group for $\%\dot{V}O_{2max}$ at LT when running at 10-km race pace.

Various studies have measured blood lactate concentration on completion of a laboratory based time trial ride (Coyle *et al.*, 1991; El-Sayed *et al.*, 1997; McNaughton *et al.*, 1999) however no study has recorded blood lactate immediately after a field based cycling time trial. Although post maximal exercise 'peak' blood lactate has been assessed in veteran athletes (Åstrand *et al.*, 1997) and older individuals (Nimmo *et al.*, 1994; Overend *et al.*, 1992; Tzankoff and Norris, 1979) there is no data available concerning the effect of age on blood lactate recorded after the cessation of laboratory and field based cycling time trial performance.

The effect of age on blood lactate concentration (BLa) recorded during indoor and outdoor cycling endurance performance has not been considered. However, one investigation (Overend *et al.*, 1992) has assessed blood lactate concentration in elderly subjects during high intensity cycling exercise. Subjects tested were physically active young (20-35 years) and old (>65 years) individuals. In this study, subjects were required to maintain a set power for 24 mins based on the individual assessment of critical power (for explanation

and review see Jenkins, 1995). Mean relative exercise intensity maintained during the test was higher in the older group (62 vs 67% W_{max}, for young and old respectively). Maximal power was determined during a progressive ramped test with ramp rate chosen to elicit volitional exhaustion in 8-12 min (a similar protocol to the Kingcycle peak power test). Mean value for peak whole BLa recorded during the test was higher in the younger group (8.1 vs 6.5 $mmol \cdot L^{-1}$) and BLa at 2 min intervals was significantly higher in the younger group from the 8th min onwards. The authors did not discuss the difference in lactacidaemia recorded between groups, however their finding did concur with other work which had reported an age-related decline in lactacidaemia in older populations (Iredale and Nimmo, 1997).

In contrast to this, Allen *et al.* (1985) found that when subjects were matched on mean 10-km race time, training distance and pace, blood lactate concentration recorded on completion of a 10 min bout of exercise at individual 10-km race pace was lower (P<0.05) in senior runners when compared with veterans (5.0 vs 3.1 $mmol \cdot L^{-1}$). Notably no difference (P>0.05) was found between the veteran runners and 'superior performance' seniors (5.0 vs 3.8 $mmol \cdot L^{-1}$).

Data concerning heart rate and oxygen uptake in veteran runners recorded when exercising at 10-km race pace were provided by Allen *et al.* (1985) however there appears to be no published data available for heart rate response and oxygen uptake of veterans during laboratory and field based cycling races. Overend *et al.* (1992) calculated that older subjects maintained a significantly higher relative exercise intensity (%$\dot{V}O_{2max}$) during a 24-min cycling test performed at critical power intensity. One explanation for this was the higher power (%W_{max}) maintained by the elderly group when compared with the young group. Notably, subjects in the study by Allen *et al.* (1985) maintained the same absolute exercise intensity (running velocity) but did not complete a 10-km run, therefore values for %$\dot{V}O_{2max}$ and %HR_{max} reported by the authors may not be a valid representation of age-related differences.

4.8 SUMMARY

This chapter considered performance related responses of senior and veteran cyclists recorded during indoor and outdoor cycling races. Although laboratory based assessments of cycling performance provide useful information for comparisons between subjects and studies there is very little information available concerning field based trials, therefore care should be taken when extrapolating indoor data to field based settings.

CHAPTER 5

5 METHODOLOGICAL ISSUES WHICH CAN AFFECT THE ASSESSMENT OF CYCLING PERFORMANCE

5.1 INTRODUCTION

During the past decade various laboratory-based physiological tests have been developed in order to assess the endurance capacity and endurance performance of competitive cyclists (for review see Hopkins *et al.*, 1999). Consequently new testing equipment has been introduced which has allowed investigators to study the physiological responses of elite performers using more ecologically valid methods of assessment (Seifert *et al.*, 1988; 1991). Various investigations have shown that laboratory based measures of endurance performance (Coyle *et al.*, 1991, Palmer *et al.*, 1996), maximal aerobic power (Hawley and Noakes, 1992; Lucia *et al.*, 1998) and maximal aerobic capacity (Saltin and Åstrand, 1967) are highly related to cycling performance in the field. However, in order to detect changes in cycling performance and study the effects of a selected intervention and/or treatment, it is critically important to ensure that the testing methods and equipment used to assess performance are reliable, reproducible and valid (Hopkins *et al.*, 1999).

The Kingcycle peak power/aerobic capacity tests (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Palmer *et al.*, 1996; 1997; 1998) have been used on numerous occasions to assess the endurance capacity of racing cyclists. However, there is very little information available concerning the reproducibility of selected physiological variables recorded during peak power tests and few studies have assessed the reproducibility of performance related responses recorded during simulated endurance performance tests. This chapter considers methodological issues which can affect the assessment these methods of testing.

5.2 ASSESSMENT OF POWER

A significant development in cycle ergometry was the introduction of 'windload simulators' in the 1970's which allowed cyclists to use their own bicycles in a laboratory setting. Previously cyclists were required to use an adapted friction based ergometer which incorporated pedals with toe clips, drop handlebars and a racing saddle (Coyle *et al.*, 1988;

1991). Consequently these laboratory specific ergometers prevented cyclists from cycling in their most biomechanically efficient body position (see Seifert *et al.*, 1988; 1991). This was unfortunate considering that cycling position can significantly affect power output recorded during cycling exercise (Gonzalez and Hull, 1989; Heil *et al.*, 1997a; Umberger *et al.*, 1998).

Very few studies have investigated the effect of cycle ergometer design on physiological responses recorded during cycling exercise. Seifert *et al.* (1991) compared physiological responses of recreational and competitive cyclists when exercising on a standard Monark cycle ergometer and an electromagnetically braked Velodyne™ cycle simulator. Different physiological responses were recorded when exercising at higher (~ 225 W) but not lower workloads (~ 100-175 W) and the authors concluded that calibration, changes in muscle fibre recruitment and ergonomics may have affected the mean values recorded. In conclusion Seifert *et al.* (1991) argued that cycle simulators could be used to provide a more ecologically valid method of assessing the physiological responses of racing cyclists during laboratory based tests.

Subsequently, several studies (Foster *et al.*, 1993; Snyder *et al.*, 1993) have used cycle simulators to investigate physiological responses of racing cyclists during cycling performance. Notably the Kingcycle air-braked ergometer was a major improvement in design. The Kingcycle enabled investigators to instantaneously record power output (monitored and displayed continuously by a microcomputer) heart rate, pedal cadence, speed and distance during laboratory based cycling performance rides and provided a reasonably accurate simulation of the inertial characteristics of actual cycling. The Kingcycle has been used on several occasions to assess the endurance capacity of highly trained cyclists (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Palmer *et al.*, 1998) evaluate the effects of ergogenic aids (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Palmer *et al.*, 1998; Robinson *et al.*, 1995) and investigate the affect of training interventions (Lindsay *et al.*, 1996; Palmer *et al.*, 1997; Stepto *et al.*, 1999; Westgarth-Taylor *et al.*, 1997; Weston *et al.*, 1997) on cycling time trial performance.

Several investigators (Keen *et al.*, 1991; Palmer *et al.*, 1996; and Schabort *et al.*, 1998a) have assessed the reproducibility of cycling performance tests performed using a Kingcycle ergometer and found that the within subject variations (CV%) for time to complete endurance performance rides (Palmer *et al.*, 1996; and Schabort *et al.*, 1998a) were reasonably low (<1.5%) and that test re-test correlation for maximal exercise performance (Keen *et al.*, 1991) was relatively high (r = 0.98).

5.3 ASSESSMENT OF EXPIRED AIR AND PULMONARY VENTILATION

The effects of exercise performance on pulmonary ventilation (Wasserman *et al.*, 1979; Whipp and Ward, 1980) and O_2 and CO_2 content in expired air (Poole and Richardson, 1997; Poole *et al.*, 1988) have been investigated in depth, consequently testing apparatus and protocols have continuously evolved to provide more reproducible and valid measures of pulmonary/respiratory responses during exercise (see Thoden, 1991). Although the collection of expired air using Douglas bags is considered the 'gold standard' method of assessing ventilation and respiratory gas (Hammond and Froelicher, 1984), it is now widely accepted that online gas analysers provide an immediate, practical and efficient method of measuring $\dot{V}_E$, $\dot{V}O_2$ and $\dot{V}CO_2$ during exercise (McArdle *et al.*, 1991).

The Covox™ online gas analysis system has recently been used to assess pulmonary and respiratory responses during cycling exercise performance (Balmer *et al.*, 1998). De Montfort University, Bedford, UK (unpublished data, 1994) have compared $\dot{V}_E$, $\dot{V}O_2$ and $\dot{V}CO_2$ recorded using Covox gas analysis system with a traditional Douglas bag method of assessment. Expired air collected using the Douglas bag method was analysed for O_2 and CO_2 content using a Servomex gas analyser (Crowborough, UK) and ventilation was assessed using a Harvard dry gas meter. Analysis of the De Montfort data revealed that the absolute differences between $\dot{V}O_2$ and $\dot{V}CO_2$ calculated using each analysis system were heteroscedastic. This was shown by the correlation between the absolute differences for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ against the mean value for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ (r = 0.79, 0.86 and 0.45). However when the data were logarithmically transformed correlations were reduced (r = 0.15, 0.21 and 0.37, respectively). Limits of agreement (Bland and Altman, 1986) between Douglas bag and Covox values were expressed as ratios based on clear evidence that absolute differences were greater at higher values. Therefore calculated ratio limits of

agreement for Douglas bag and Covox measures for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ were 1.10 ×/÷ 1.05 and 1.07 ×/÷ 1.03, 1.0 ×/÷ 1.02, respectively. The differences between Douglas bag and Covox values for $\dot{V}O_2$ and $\dot{V}CO_2$ were significant (P<0.05). However, estimates of within subject variation (CV%) for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ were similar for each gas analysis system 3.7, 5.4, 3.7 vs 4.1, 3.5, 4.8% for Covox compared with Douglas bag method. It is worth noting that calculations for CV% were based on data collected for one subject, who completed five 'steady-state' exercise trials performed at a relatively low exercise intensity during a period of eight days.

The De Montfort study showed that the Covox gas analysis system provided a valid and reliable assessment of pulmonary ventilation and a reliable but not valid measure of O_2 and CO_2 content in expired air when compared with a Servomex gas analyser.

5.4 ASSESSMENT OF BLOOD LACTATE

The relationship between blood lactate response and cycling exercise performance has been frequently used to assess the endurance capacity of racing cyclists and subsequently blood lactate analysers have become widely used for the purposes of research and sport science support. However, blood lactate concentration can be affected by several methodological factors such as:- sample site (El-Sayed et al., 1993a; Jorfeldt et al., 1978; Williams et al., 1992); method of assay (Williams et al., 1992); and analysis equipment (Davison et al., 2000a).

Bishop et al. (1992) found that when blood lactate concentration recorded using a spectrophotometer was compared with a YSI electrochemical lactate analyser (YSI. Model 2300 STAT analyser, Yellow Springs Instruments, Yellow Springs, USA) no difference was found between values recorded using each measuring device, therefore it was concluded that the YSI provided a valid and reliable measure of blood lactate. More recently, Davison et al. (2000a) compared a Biosen 5030 L blood lactate analyser (EKF Industrie - Electronik GmbH, Germany) with a YSI and calculated that the bias and random error for 95% limits of agreement (Bland and Altman, 1986) were 0.37 ± 1.22 mmol·L⁻¹. Notably, the test re-test reliability correlation coefficient of determination between repeated blood lactate samples assessed using the Biosen was very high ($r^2 = 0.99$,

P<0.05). Therefore the authors concluded that the Biosen provided a reliable and valid measure of blood lactate concentration when compared with a YSI.

Blood samples for lactate testing can be obtained from a number of sample sites. These include radial and brachial arteries (Yoshida *et al.*, 1982) antecubital or dorsal veins (Conconi *et al.*, 1982), and fingertip or ear lobe capillary samples (Foxdal *et al.*, 1990). Several studies (El-Sayed *et al.*, 1993a; Jorfeldt *et al.*, 1978; Williams *et al.*, 1992) have investigated the effects of sample site variation on blood lactate concentration and found that BLa in fingertip blood can be significantly higher when compared with venous blood. Williams *et al.* (1992) explained that values between finger tip blood and arterial blood are similar when investigators have made sure that the finger tip sample consists of arterialised blood. This can be achieved by an increase in the circulation of blood through the hand by pre-warming with moderately hot water and/or the completion of an adequate (about 5-10 min) warm-up prior to blood sampling.

The analysis of fingertip whole blood has a distinct methodological advantage over other sampling sites and methods of assay (Knowlton *et al.*, 1990). Therefore when working with athletes from various sports it is now common practice for blood samples to be taken either from ear lobe or fingertip with the BLa analysed in whole blood.

5.5 ASSESSMENT OF HEART RATE

Within the past ten years reliable lightweight telemetry systems have become increasingly popular with competitive cyclists and have been shown to provide both reliable and valid measures of heart rate response when compared with ECG monitors (Léger and Thivierge, 1988). Consequently, heart rate monitors have been extensively used to record heart rate data during training/racing (Jeukendrup and van Diemen, 1998) and laboratory based exercise tests (for review see Lambert *et al.*, 1998). Although several authors have questioned the validity and reproducibility of using heart rate response to improve training and racing performance (see Burke *et al.* 1994), heart rate monitoring during exercise has been used to effectively monitor endurance performance and detect the onset of over training.

It is important to note that the relationship between heart rate response and exercise intensity can be affected by several factors such as:- exposure to cold (Leweke *et al.*, 1995); heat (Galloway and Maughan, 1997; Sawka *et al.*, 1984); altitude (Sutton *et al.*, 1988); biorhythmicity (Reilly *et al.*, 1984); 'competition arousal' (Lambert *et al.*, 1998); and the phenomenon of 'cardiac drift' (Shaffrah and Adams, 1984). Also maximal heart rate can be subdued during periods of heavy training (Gaesser and Poole, 1986; Maassen and Busse, 1989) but return to normal levels following a period of rest (Lambert *et al.*, 1998).

Recently Jeukendrup and van Diemen, (1998) also found that changes in power output during training and racing were not accurately reflected in heart rate response during outdoor cycling performance and Kenny *et al.* (1995) showed that mechanical load experienced in the field was underestimated when heart rate (measured during laboratory based tests) was used to establish field based training intensity. In addition, there is strong evidence to suggest that body position can also affect heart rate recorded during laboratory and field based cycling tests response (Gnehm *et al.*, 1997; Heil *et al.*, 1997a; Price and Donne, 1997).

5.6 ASSESSMENT OF CYCLING PERFORMANCE

Although the assessment of maximal aerobic capacity has been traditionally used as a 'gold standard' measure of cardio-respiratory fitness (Åstrand and Saltin, 1961), recent studies have used measures of endurance capacity and endurance performance to assess the physiological responses of competitive cyclists (Bishop, 1997; Coyle *et al.*, 1991).

An important distinction has been made concerning the correct terminology to describe testing methods which measure exercise performance. For instance an exercise test designed to measure endurance capacity would involve a measure of time to exhaustion when maintaining a fixed workload and a test which measures endurance performance involves either; a distance or work bout covered in a minimum amount of time, or a maximal work bout achieved during a fixed amount of time (Williams *et al.*, 1990).

Traditionally, measures of endurance capacity have been used to assess cycling performance (Brouns *et al.* 1989; Coyle *et al.*, 1983a; 1986; Gleeson *et al.* 1986), however

more recent studies have used:- a measure of distance and/or power output recorded in a fixed time (Anantaraman *et al.*, 1995; Bishop *et al.*, 1998; Coyle *et al.*, 1991; El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Robinson *et al.*, 1995); a measure of time to complete a target workload (Luetkemeier and Thomas, 1994); time to complete a laboratory based fixed distance time trial (Lindsay *et al.*, 1996; Palmer *et al.*, 1998; Potteiger *et al.*, 1995; Westgarth-Taylor *et al.* 1997; Weston *et al.* 1997); and time to complete a field based fixed distance time trial (Coyle *et al.* 1991; Dobbins, 1996; Hawley and Noakes, 1992; Hoogeveen *et al.* 1999; Langenfeld, 1983; Langenfeld *et al.* 1994).

5.6.1 MAXIMAL/PEAK POWER TEST

Hawley and Noakes (1992) found that a measure of maximal power output (expressed as W_{max}) achieved during a progressive exercise test to volitional exhaustion was highly related to field based 20-km cycling performance (r = -0.91) and more recently Bishop *et al.* (1998) found that W_{max} was a stronger predictor of indoor 1-h cycling endurance performance (r = 0.81) when compared with maximal oxygen uptake (r = 0.55). Not surprisingly, a measure of maximal muscle power has been used by several investigators to assess; the exercise performance capacity of racing cyclists (Hawley and Noakes, 1992; Kuipers *et al.* 1985; Palmer *et al.* 1994; Westgarth-Taylor *et al.* 1997, Weston *et al.* 1997) and the effects of training on endurance performance (Lindsay *et al.* 1996; Weston *et al.* 1997) and several authors (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Palmer *et al.*, 1996; 1997; 1998) have used a measure of peak power (W_{peak}) recorded during a Kingcycle peak power test (PP) to assess the exercise performance capacity of endurance cyclists. It is worth noting that W_{max} is typically assessed using a continuous 25 W incremental test (the duration of each increment is 150-s) and W_{peak} is determined using a continuous Kingcycle (~ 20 W·min^{-1}) ramp test. Although both methods record aerobic power (also described as maximal muscle power by Hawley and Noakes, 1992). It is important to note that W_{max} is calculated as the last completed work rate in W plus the fraction of time spent in the final non completed work rate multiplied by 25 W (Stepto *et al.*, 1999) and W_{peak} is the highest average power output recorded during any 60-s period of the Kingcycle PP/PAC test. For the assessment of W_{max}; volitional exhaustion was defined as a drop in pedalling rate of >10 rev·min^{-1} (Stepto *et al.*, 1999) and for W_{peak}, volitional exhaustion was defined as the failure to maintain the required increase in power output.

5.6.1.1 Factors influencing the determination of maximal/peak power

Studies which have investigated factors which influence W_{max} have found that maximal power can be affected by:- the level of pre-exercise activity (Hughes *et al.*, 1982; Maassen and Busse, 1989); a combination of prior exercise and diet (Heigenhauser *et al.*, 1983), testing protocol (Davis *et al.*, 1982) and pedal cadence (Buchanan and Weltman, 1985). For the purposes of this review studies which have investigated the effects of testing protocol and pedal cadence will be considered.

5.6.1.1.1 Effect of test duration and rate of increase in workload

Several authors have found that the duration of the exercise test (Yoshida, 1984) and the rate at which the workload is increased during the test (Davis *et al.*, 1982; Hansen *et al.*, 1988) affects maximal power recorded during the test. However it is worth noting that studies which have reported a difference in W_{max} achieved during progressive exercise tests have consistently used radically different ramp rates (Davis *et al.*, 1982; Hansen *et al.*, 1988). In the studies by Davis *et al.* (1982) and Hansen *et al.* (1988) ramp rate ranged from 20 to 100 W·min^{-1} and 15 to 60 W·min^{-1}, respectively and in the study by Hansen *et al.* (1988) the average duration of a test which used a ramp rate of either 15 or 60 W·min^{-1} was about 16 and 5 min respectively. Similarly in the study by Yoshida (1984) a notable increase in maximal power was recorded when the duration of each stage of an incremental test to volitional exhaustion was reduced from 4 to 1 min. However average time to complete the test with an increment per 4 min was ~ 32 min compared with ~10 min when the workload increment was every 1 min. Interestingly Zhang *et al.* (1991) found that testing protocol did not affect maximal power when the duration of the test was kept relatively constant (~ 13 min). Kuipers *et al.* (1985) suggested that in order to compare work loads between, as well as within subjects, the rate at which the workload is increased should be normalised by converting absolute workloads to relative work loads. This can be achieved by increasing workload by a percentage of maximal/peak power achieved during a previous reference (habituation) test.

5.6.1.1.2 Pedal cadence

Buchanan and Weltman (1985) found that maximal workload achieved during a graded exercise test was significantly less when subjects were required to maintain a pedal cadence of 120 rev·min^{-1} compared with 90 and 60 rev·min^{-1} with no difference recorded

between 90 and 60 rev·min^{-1}. This finding was supported by the work of Hughes *et al.* (1982), who found no difference in maximal workload achieved during an incremental exercise test performed with a pedal cadence of 50 rev·min^{-1} when compared with 90 rev·min^{-1}. In the studies by Davis *et al.* (1982), Hansen *et al.* (1988) and Zhang *et al.* (1991) subjects maintained a pedal cadence of 60 rev·min^{-1} and in the investigation of Yoshida, (1984) pedal rate was kept constant at 50 rev·min^{-1}. However these pedal cadences (50 and 60 rev·min^{-1}) are surprisingly low when compared with the pedalling rates reported for racing cyclists during actual field based cycling (Jeukendrup and van Diemen, 1998). In the exercise protocols of Kuipers *et al.* (1985) and Keen *et al.* (1991) subjects were able to self-select pedal cadence during the test.

5.6.1.2 REPRODUCIBILITY OF MAXIMAL/PEAK POWER

In the study by Kuipers *et al.* (1985) ten subjects completed 32 ± 17 (mean $\pm$ SD) maximal tests over a period of ~12 months and CV calculated for W_{max} varied inter-individually by ~3.0% to 6.8% (mean 5.0%). However, in contrast to this Lindsay *et al.* (1996) and Westgarth-Taylor *et al.* (1997) calculated that the CV for W_{max} was 1.1 and 1.5%, respectively. One possible explanation for this discrepancy is that Kuipers *et al.* (1985) tested subjects over a longer period of time, therefore biological variability may have contributed more to the overall CV for W_{max}. Keen *et al.* (1991) calculated that the test re-test correlation for peak power (W_{peak}) assessed during Kingcycle peak tests was r = 0.98.

5.6.2 MAXIMAL/PEAK AEROBIC CAPACITY TEST

The measurement of maximal aerobic work rate (usually expressed as $\dot{V}O_{2max}$) is the most widely used method of assessing exercise performance capacity. Not surprisingly, the relationship between $\dot{V}O_{2max}$ and endurance performance has received considerable attention (see Noakes; 1988; 1997; 1998) and the measurement of maximal oxygen uptake during exercise performance has been extensively studied and reviewed (Hammond and Froelicher, 1984; Poole and Richardson, 1997; Shephard, 1984).

5.6.2.1 FACTORS INFLUENCING THE DETERMINATION OF MAXIMAL OXYGEN UPTAKE

Values for $\dot{V}O_{2max}$ recorded during aerobic capacity tests can be affected by the modality of exercise (Harrison *et al.*, 1980) and the specificity of the exercise (Hagberg *et al.*, 1978; Stromme *et al.*, 1977). Harrison *et al.* (1980) reported that $\dot{V}O_{2max}$ recorded in a group of

physically active subjects was 20% lower during a bicycle ergometer test when compared with a treadmill running test and Stromme et al. (1977) found that athletes recorded their highest $\dot{V}O_{2max}$ during a test which specifically replicated the physical demands of their sport. In support of this finding, Tanaka et al. (1996) reported no difference in $\dot{V}O_{2max}$ when trained cyclists completed bicycle ergometer tests in a seated and standing position and Heigenhauser et al. (1983) found that $\dot{V}O_{2max}$ recorded in five healthy subjects who completed a maximal cycle ergometer test was not affected by a reduction of muscle glycogen content by repeated maximum exercise and high fat-protein diet.

5.6.2.1.1 Effect of test duration and rate of increase in workload.

Buchfuhrer et al. (1983) suggested 10 min as an optimal length of time for the achievement and assessment of maximal cardio-pulmonary function and subsequently McLellan (1985) found that values for $\dot{V}O_{2max}$ were affected by the duration of the test. Studies which have investigated the affect of testing protocol on the assessment of $\dot{V}O_{2max}$ have consistently shown that $\dot{V}O_{2max}$ is not effected by a change in work increment (Davis et al., 1982; Hansen et al., 1988; Whipp et al., 1974) and/or the duration of the increment (Ribeiro et al. 1986; Yoshida, 1984; Zhang et al., 1991). For instance in the study by Whipp et al. (1974) there was no appreciable difference in $\dot{V}O_{2max}$ when ramp rate varied between 3.75 to 187 $W \cdot min^{-1}$. Similarly, Zhang et al. (1991) found no difference in $\dot{V}O_{2max}$ when the overall rate of increase in work rate was kept constant during ramp and incremental tests with 1-min, 2-min and 3-min stages. It is worth noting that Kuipers et al. (1985) used an incremental protocol to assess $\dot{V}O_{2max}$ and W_{max} and the increase in ramp rate for the test was standardised at 5% of W_{max} achieved during an habituation test. Therefore subjects attained $\dot{V}O_{2max}$ at a relatively similar time period during each test (usually between the 9th and 11th min).

5.6.2.1.2 Pedal cadence

Pivarnik et al. (1988) investigated the effect of pedal cadence on $\dot{V}O_{2max}$ recorded during incremental cycle ergometer tests. Analysis of the data revealed no difference in $\dot{V}O_{2max}$ when subjects cycled to volitional exhaustion with pedal speeds of 50 and 90 $rev \cdot min^{-1}$. In contrast to this, Buchanan and Weltman (1985) found that $\dot{V}O_{2max}$ was higher when racing cyclists maintained a pedal frequency of 60 compared with 90 and 120 $rev \cdot min^{-1}$, with no

difference in $\dot{V}O_{2max}$ with a pedal frequency of 90 and 120 rev·min⁻¹. Marsh and Martin, (1993) recorded the preferred pedal cadence of trained cyclists during a maximal aerobic capacity test performed using their own racing bicycle attached to a Velodyne™ electromagnetically braked cycle simulator. Analysis of the data revealed that preferred pedal cadence was 85 ± 9 rev·min⁻¹.

5.6.2.2 Reproducibility of $\dot{V}O_{2MAX}$

Several studies have assessed the reproducibility of $\dot{V}O_{2max}$ achieved during repeated maximal exercise tests. Recently Jensen and Johansen (1998) estimated that the test re-test reproducibility (CV) for $\dot{V}O_{2max}$ in a group of seven trained cyclists was a relatively low 1.9%. This value is less than CVs reported by Harrison et al. (1980), Jones and Kane, (1979) Katch et al. (1982) and Kuipers et al. (1985) of 4.7, 3.8, 5.6 and 7.9%, respectively. Furthermore Katch et al. (1982) estimated that ~ 90% of total measurement error was due to biological variability and 10% was due to technical error. Jones and Kane, (1979) estimated that the variability of $\dot{V}O_{2max}$ increased from 3.8 to 5.1% when measurements were taken over a longer period of time (5 days compared with 5 yr) and there was a further increase (3.8 to 10.6%) when each subject's $\dot{V}O_{2max}$ was measured in four different laboratories.

5.6.2.3 Criterion measures used to assess maximum oxygen uptake

Numerous testing methods have been developed in order to assess the $\dot{V}O_{2max}$ of endurance athletes (for review see Howley et al. 1995) and the following criteria have been frequently used to assess whether or not maximal oxygen uptake ($\dot{V}O_{2max}$) has been achieved during a maximal exercise test:- the attainment of a plateau in oxygen consumption with an increase in workload or an increase of $\dot{V}O_2$ less than 150 mL·min⁻¹ (Taylor et al., 1955); a respiratory exchange ratio in excess of 1.15 (Issekutz et al., 1962); a maximal heart rate within 5 b·min⁻¹ of age predicted maximum heart rate (Londeree and Moeschberger, 1982); and a peak blood lactate concentration greater than 8 mmol·L⁻¹ (Åstrand, 1952). Unfortunately, there is very little information available concerning the reproducibility and validity of $\dot{V}O_{2max}$ criteria and physiological variables recorded during a maximal aerobic capacity test (see Howley et al., 1995).

5.6.2.3.1 Assessment of a plateau in $\dot{V}O_2$

Although a plateau in oxygen uptake with an increasing workload is widely accepted as evidence for the achievement of $\dot{V}O_{2max}$, various authors have questioned whether a 'plateau' in $\dot{V}O_2$ is a physiological phenomenon or an artefact of the testing protocol (Bassett and Howley; 1997; Howley *et al.* 1995; Noakes; 1988; 1997; 1998). A recent study by Wood *et al.* (1998a) investigated the variability in oxygen uptake during a maximal test and its implications for the identification of a $\dot{V}O_2$ plateau. Analysis of the data revealed that the variability of $\dot{V}O_2$ decreased during high intensity exercise ($\sim 96\%$, $\dot{V}O_{2max}$) when compared with sub-maximal exercise ($\sim 70\% \ \dot{V}O_{2max}$). It was suggested that the $\dot{V}O_2$-plateau criterion of Taylor *et al.* (1955) i.e. an increase in $\dot{V}O_2$ of less than 150 $mL \cdot min^{-1}$ should not be adopted. The authors explained that the value of 150 $mL \cdot min^{-1}$ was derived from measurements of $\dot{V}O_2$ recorded during sub-maximal exercise and therefore did not provide a valid assessment of the variability in $\dot{V}O_2$ which occurs during high intensity/maximal work. Further investigation was suggested in order to quantify the variability in $\dot{V}O_2$ during maximal exercise and re-evaluate the criterion value proposed by Taylor *et al.* (1955).

The criteria for a $\dot{V}O_2$-plateau proposed by Taylor *et al.* (1955) was established using data collected during a discontinuous exercise protocol with tests repeated over a period of days until a plateau for $\dot{V}O_2$ had been recorded. Noakes (1998) argued that the $\dot{V}O_2$-plateau recorded by Taylor *et al.* (1955) may have been due to changes in running economy as a result of the previous tests. In order to assess the effect of testing protocol on the achievement of a $\dot{V}O_2$ plateau, Duncan *et al.* (1997) tested ten runners using continuous and discontinuous incremental treadmill runs. Analysis of the data showed that a definite plateau in $\dot{V}O_2$ was recorded in five runners during the continuous test and six runners during the discontinuous test. However when Hawley and Noakes (1992) used a continuous incremental exercise protocol to record $\dot{V}O_{2max}$ only 38% of subjects showed a definite plateau of $\dot{V}O_2$ with an increase in external work. Whipp *et al.* (1981) stated that when subjects completed a ramped cycling test a plateau in $\dot{V}O_2$ was frequently discerned, however the same subjects did not show a plateau during incremental tests with 1-min and 5-min stages. It is important to note that Whipp *et al.* (1981) measured respiratory gas and

ventilation using a 'breath by breath' gas analysis system and in the work of Duncan *et al.* (1997) and Hawley and Noakes (1992) expired air and pulmonary ventilation were measured using online mixing chamber systems with $\dot{V}O_2$ recorded at 1-min intervals.

5.6.2.3.2 Assessment of maximal RER

The attainment of a maximal RER value ≥ 1.15 (Issekutz *et al.*, 1962) is frequently cited as a criterion measure for the achievement of $\dot{V}O_{2max}$, (Howley *et al.*, 1995). However, Cunningham *et al.* (1977), Foster *et al.* (1986) and Sidney and Shephard (1977) have found that RER is affected by the age of the subject. In the studies of Cunningham *et al.* (1977) and Sidney and Shephard, (1977) the proportion of subjects who attained a maximal RER ≥ 1.15 was less than 40%.

Ribeiro *et al.* (1986), and Zhang *et al.* (1991) reported that RER values were not affected by testing protocol and Tanaka *et al.* (1996) found no difference in RER when subjects performed maximal bicycle ergometer tests in seated and standing positions. Also, Pivarnik *et al.* (1988) found that pedal cadence did not affect RER values recorded during cycle ergometer exercise. Howley *et al.* (1995) found that in 29 investigations which had assessed $\dot{V}O_{2max}$ about 51% of the studies used RER criterion values ranging from 1.00 to 1.13.

5.6.2.3.3 Assessment of peak blood lactate concentration

Few studies have used the criterion measure of peak blood lactate concentration for establishing $\dot{V}O_{2max}$ (see Howley *et al.*, 1995). As previously outlined in 5.4 of this section of the thesis differences in sample site, method of assay and testing equipment can affect blood lactate concentration recorded during testing trials. Notably Åstrand (1952) reported that peak blood lactate concentration recorded in young and elderly subjects did not exceed the criterion value of 8 mmol·L^{-1} and Maassen and Busse (1989) found that blood lactate recorded post maximal exercise was affected by pre-exercise diet.

Ribeiro *et al.* (1986) recorded no difference in peak blood lactate when subjects performed a fast (60 W·min^{-1}) compared with slow (15 W·min^{-1}) incremental maximal test. Similarly, Åstrand and Saltin (1961) and Yoshida (1984) found that test duration and maximal workload did not have a significant affect on peak blood lactate values recorded at the

cessation of exercise. Similarly, Ricci and Leger (1983) found no difference in peak blood lactate concentration when subjects performed three modes of maximal testing i) treadmill running ii) bicycle ergometer, and iii) velodrome cycling. Also Tanaka *et al.* (1987) found that peak lactate was similar when subjects completed a treadmill running test and bicycle ergometer test while standing and Tanaka *et al.* (1996) reported no difference in peak blood lactate when cyclists performed a maximal bicycle ergometer test in seated and standing positions. Unfortunately in the study by Åstrand and Saltin (1961) time at which the blood sample was taken was not reported however Åstrand and Rodahl (1986) and Billat (1996) suggested that peak blood lactate concentration could be determined between 5-9 minutes post maximal exercise. Notably, other studies have assessed peak lactate with a blood sample taken at 4 (Sitkowski *et al.*, 1993) and 5 mins (Ricci and Leger, 1983) post exercise. In order to determine peak blood lactate Tanaka and co-workers sampled blood between 4 to 5 (1987) and 3 to 6 (1996) minutes after the termination of maximal exercise and Ribeiro *et al.* (1986) assessed peak lactate using blood samples taken at 2, 4, 6 and 8 min during recovery, however the time at which peak lactate occurred during this time period was not reported. It is worth noting that several authors have assessed the reproducibility of peak blood lactate recorded post maximal cycling exercise and have calculated CVs between 7.2 to 11% (for review see Vandewalle *et al.* 1987). However, in a more recent study by Jensen and Johansen (1998) CV for peak blood lactate recorded following maximal cycling exercise was 14.9%.

5.6.2.3.4 *Assessment of maximal heart rate*

Maximum heart rate (HR_{max}) achieved during a maximal/peak aerobic capacity test has been frequently used as a criteria for $\dot{V}O_{2max}$ and several authors have assessed the affect of testing protocol on the attainment of HR_{max}. Notably the work of Åstrand and Saltin, (1961) and Yoshida, (1984) showed that values for heart rate were not affected by the duration of the test and Zhang *et al.* (1991) found no difference in heart rate values recorded during ramp and incremental exercise. In contrast to these findings Davis *et al.* (1982) found that maximal heart rate was significantly lower when subjects completed a 100 $W \cdot min^{-1}$ ramp test when compared with 20, 30, and 50 $W \cdot min^{-1}$ tests. It was noted that heart rate was successively higher as the duration of the ramp tests increased. This finding was confirmed by the work of Ribeiro *et al.* (1986) who reported that heart rate was higher during an

incremental test with a 15 W·min^{-1} (slow) increase in work rate when compared with an increase of 60 W·min^{-1}. Not surprisingly, there was a notable difference in test duration between the slow protocol (~ 23 min) compared with the fast protocol (~ 10 min). Ingjer (1991) showed that testing protocol can affect the assessment of maximal heart rate. Analysis of the data revealed that runners achieved the highest heart rate during a series of 'all-out' 3-4 min runs performed on a treadmill with a 3° uphill inclination. The effect of exercise modality on maximal heart rate was investigated by Tanaka *et al.* (1987). In this study subjects completed a treadmill running test and a bicycle ergometer tests with toe-stirrups while standing. Maximal heart rate was significantly higher during the treadmill test. When the effect of cycling position on maximal heart rate was assessed by Tanaka *et al.* (1996), no difference was recorded when subjects completed a maximal bicycle ergometer test in seated and standing positions. Pivarnik *et al.* (1988) investigated the effect of pedal cadence on heart rate recorded during a maximal bicycle ergometer test and found no difference when subjects were required to maintain a pedal cadence of 50 and 90 rev·min^{-1}.

Several authors (Åstrand and Saltin, 1961; Harrison *et al.*, 1980; Jensen and Johansen, 1998; Jones and Kane, 1979) have assessed the reproducibility of individual maximal heart rate recorded during exercise tests. Åstrand and Saltin (1961) reported that the standard deviation for maximal heart rate recorded during exercise to exhaustion performed on a cycle ergometer was ± 3 b·min^{-1} (1 SD) and Jones and Kane (1979) calculated CVs for maximal heart rate values recorded over a short (~ 1 month) and long (~ 5 yr) period of time were ± 3.0% and ± 4.7%, respectively. In the study by Harrison *et al.* (1980) CV for maximal heart rate during a six month period of testing was ~2.7% and more recently Jensen and Johansen (1998) reported a test re-test CV of 1.3%. Howley *et al.* (1995) discussed the validity of using a maximal heart rate value based on an age predicted values and argued that the error in the equation used to predict maximal heart rate was ± 11 b·min^{-1} (1 SD). Therefore investigators were advised not to use age predicted maximum heart rate as criterion for $\dot{V}O_{2max}$.

5.6.2.3.5 Assessment of $\dot{V}CO_{2max}$

Very few studies have assessed the reproducibility of maximal $\dot{V}CO_2$ recorded during a $\dot{V}O_{2max}$ test (Jensen and Johansen, 1998; Jones and Kane, 1979). Jones and Kane (1979) calculated that the CV% for $\dot{V}CO_{2max}$ was ~ 4.2% when subjects were tested on five consecutive days. Similarly, Jensen and Johansen (1998) estimated that the CV for $\dot{V}CO_2$ was within a range of about 4.75-5.25%. Notably, Jones and Kane (1979) found that the CV for $\dot{V}CO_{2max}$ increased to ~ 11.5% when assessed over a period of five years. There is no information valaible concerning the effect of testing protocol on the assessment of $\dot{V}CO_{2max}$.

5.6.2.3.6 Assessment of $\dot{V}_{Emax}$

Jensen and Johansen (1998) estimated that the test re-test reproducibility of $\dot{V}_{Emax}$ was 11.8% and in the study by Jones and Kane (1979), CV for $\dot{V}_{Emax}$ was calculated as 8%. Interestingly Jones and Kane (1979) reported that CV calculated for $\dot{V}_{Emax}$ was reduced from 8 to 5.0% when $\dot{V}_{Emax}$ was assessed over five years. One possible explanation for this was that subjects were not fully habituated to the demands of maximal exercise testing and that biological variability over a longer period of time was less for $\dot{V}_{Emax}$ when compared with $\dot{V}O_2$. It is reasonable to assume that training/detraining affected $\dot{V}O_2$ more than $\dot{V}_E$.

Several studies (Davis et al., 1982; Ribeiro et al., 1986; Yoshida, 1984; Zhang et al., 1991) have assessed the effect of testing procedure on $\dot{V}_{Emax}$. Yoshida (1984) reported no difference in $\dot{V}_{Emax}$ achieved during incremental exercise tests of ~10 and 32 min duration, and Ribeiro et al. (1986) found no change in $\dot{V}_{Emax}$ when subjects performed incremental tests with an increase in workload of either 15 or 60 $W \cdot min^{-1}$. Similarly Davis et al. (1982) and Zhang et al. (1991) found values for $\dot{V}_{Emax}$ were similar when tests were performed using different ramp rates.

5.6.3 THRESHOLD EXERCISE INTENSITY

Physiological tests which identify and quantify a threshold exercise intensity have been used to assess the effects of training (Londeree, 1997), to predict endurance performance (Farrell et al., 1979; Roecker et al., 1998) and to estimate MLSS (Jones and Doust, 1998). The threshold concept of exercise performance has evolved from the hypothesis that the

power, running speed and $\dot{V}O_2$ at threshold is a key determinant of endurance performance (Coyle, 1995). In order to provide meaningful information threshold parameters have to be both valid and reproducible. This section considers research which has investigated methodological issues which affect the assessment of threshold exercise intensity.

5.6.3.1 EFFECT OF TESTING PROTOCOL

5.6.3.1.1 Effect of ramp rate

Very few studies have assessed the affect of ramp rate on the determination of threshold exercise intensity. Ribeiro et al. (1986) found that ramp rate of 15 or 60 W·min^{-1} did not affect power at TLac but did affect power output at LTP. However in contrast to this, $\dot{V}O_2$ at TLac and LTP were not affected by the testing protocol. Campbell et al. (1989) found no difference in $\dot{V}O_2$ (L·min^{-1}) at TLac when ramp rate was 8 and 15 W·min^{-1} but $\dot{V}O_2$ at TLac was significantly higher when subjects completed a 50 W·min^{-1} ramp test (2.05, 2.04 and 2.24 L·min^{-1} for 8, 15 and 50 W·min^{-1} ramp rates, respectively). Unfortunately Campbell and co-authors did not investigate the effect of ramp rate on power output at TLac.

5.6.3.1.2 Effect of stage duration

Several studies have investigated the effect of stage duration on the assessment of threshold variables. Yoshida (1984) recorded running speed and $\dot{V}O_2$ at TLac and OBLA during incremental tests with 1 and 4 min stage duration and found that testing protocol did not affect $\dot{V}O_2$ at TLac or OBLA but did affect running speed at TLac and OBLA. This finding was confirmed by other investigators who reported that stage duration significantly affected power output at TLac (MacFarlane et al., 1983; Smith et al., 1997) but not $\dot{V}O_2$ at TLac (Smith et al., 1997)

McLellan (1985) reported that increment duration did not affect the determination of $\dot{V}O_2$ at OBLA during three continuous incremental cycling tests with either 1, 3 or 5 minutes stage duration. In contrast to this work, Watts et al. (1998) found that stage duration during an incremental test did not affect $\dot{V}O_2$ and HR at OBLA however there was a significant change in power at OBLA when subjects completed discontinuous incremental protocols with a stage duration of 2 and 4 mins, notably no difference was found in power at OBLA when stage duration was either 4 or 6 mins. Stockhausen et al. (1997) calculated that the

time required to reach a quasi-steady state blood lactate concentration exceeded two mins when stage increment was above 10 W. Unfortunately, Watts *et al.* (1998) did not report the increase in power for each stage of the test.

5.6.3.1.3 *Continuous versus discontinuous protocols*

Very few studies have assessed the effect of using continuous and discontinuous tests to identify and quantify threshold exercise intensity. Gullstrand *et al.* (1994) investigated the effect of a 30-s pause on the determination of TLac during an incremental test and found that there was no difference between continuous and discontinuous modes of exercise. This study has been cited on several occasions by investigators who have used an interrupted test protocol to assess threshold exercise intensity (Carter *et al.*, 1999; Jones and Doust, 1998). It is generally believed that during an incremental test the duration of each stage is an important methodological issue in order to allow sufficient time for blood lactate concentration to achieve a quasi-steady-state (Stockhausen *et al.*, 1997).

Weltman *et al.* (1990) investigated the effect of testing protocol on the running speed (km·h^{-1}), $\dot{V}O_2$ (mL·kg·min^{-1}) and HR (b·min^{-1}) at TLac and OBLA detected during continuous and discontinuous incremental tests. No difference was found between testing methods for variables determined at TLac and OBLA. However standard error of estimates (SEE) calculated for TLac and OBLA from the regression of continuous and discontinuous methods were higher for V@TLac (km·h^{-1}), $\dot{V}O_2$@TLac (mL·kg·min^{-1}) and HR@TLac (b·min^{-1}) than V@OBLA, $\dot{V}O_2$@OBLA and HR@OBLA (3.58, 13.1 and 6.5 vs 1.99, 6.5 and 3.7, respectively). There is appears to be no information available concerning the effects of continuous versus discontinuous exercise protocols on the assessment of threshold intensity during other modes of exercise. During a discontinuous incremental cycling protocol subjects are required to accelerate their power output in order to achieve the required power of the subsequent stage. The effects of this extra power on the determination of threshold exercise intensity has not been investigated.

5.6.3.1.4 *Effect of testing protocol on the determination of lactate minimum threshold*

The lactate minimum test (Lac$_{min}$) is a relatively new method of determining the anaerobic threshold and maximal lactate steady state (Tegtbur *et al.*, 1993). As previously described in chapter one of this thesis, the lactate minimum point (LMP) is designated as the $\dot{V}O_2$,

($\dot{V}O_2$@LMP) power output (W@LMP) and HR (HR@LMP) corresponding to the minimum blood lactate concentration determined during an incremental test completed after a bout of intense exercise which induced lactic acidosis (Tegtbur *et al.*, 1993).

Several authors have investigated the effects of testing protocol on the determination of V@LMP (Carter *et al.*, 1999) and W@LMP; (Caine *et al.*, 1997; Davison *et al.*, 1998; Morgan, 1997; Smith *et al.*, 1998). Investigators have evaluated the effect of lactate elevation mode on W@LMP (Smith *et al.*, 1999) and $\dot{V}O_2$@LMP (Caine *et al.*, 1997) and found no difference when trained cyclists performed repeated sprint tests and a Kingcycle PP test in order to induce lactacidaemia (Smith *et al.*, 1999). Similarly, no difference was found for $\dot{V}O_2$@LMP when physically active subjects completed either a Wingate sprint test or a progressive incremental $\dot{V}O_{2max}$ test to induce lactacidaemia (Caine *et al.*, 1997).

Carter *et al.* (1999), Davison *et al.* (1997) and Morgan (1998) investigated the effect of starting intensity for the second part of the Lac_{min} test on the determination of LMP. Notably Carter *et al.* (1999) found that starting intensity significantly affected V@LMP when using the Lac_{min} test described by Jones and Doust (1998). In agreement with this finding Davison *et al.* (1997) and Morgan (1998) found that starting intensity (calculated as a percentage of W_{peak} achieved during PP) had a significant affect on W@LMP. Davison *et al.* (1998) also showed that ramp rate during the second part of the test affected the power output at which the nadir in blood lactate concentration was recorded. Analysis of the blood lactate response recorded during the second part of the Lac_{min} test revealed that a faster ramp rate increased the power at LMP. Unfortunately Carter *et al.* (1999) and Davison *et al.* (1997) did not report data concerning the affect of Lac_{min} testing protocol on values for $\dot{V}O_2$@LMP. However, Morgan (1998) found that $\dot{V}O_2$@LMP and HR@LMP was higher when the second part of the test was initiated at 65% W_{peak} when compared with 60% W_{peak}, with no difference between $\dot{V}O_2$@LMP and HR@LMP when the starting intensity was 65 and 70% of W_{peak}.

5.6.3.2 EFFECT OF DIET

Blood and muscle lactate concentrations are altered by diet and exercise or the combination of diet and exercise (Gollnick *et al.*, 1986). Consequently studies have reported a decreased

blood lactate concentration following overtraining (Jeukendrup and Hesselink, 1994) and dietary modification (Hughes *et al.*, 1982). Maassen and Busse (1989) observed a significant increase in power output at OBLA when subjects ingested a low carbohydrate diet when compared with a high carbohydrate diet, however in the study by Hughes *et al.* (1982) glycogen depletion affected blood lactate concentration associated with the occurrence of TLac, but not power and $\dot{V}O_2$ at TLac. Similarly Quirion *et al.* (1988) studied the effects of dietary modifications on TLac and OBLA during progressive incremental exercise and found that the ingestion of either a high carbohydrate rich or fat rich diet did not significantly alter $\dot{V}O_2$ at TLac and OBLA and Tegtbur *et al.* (1993) found that diminished muscle glycogen stores did not affect running speed and blood lactate concentration at lactate minimum point.

5.6.3.3 EFFECT OF TESTING EQUIPMENT

There is very little information available concerning the affects of testing equipment on the determination of a threshold exercise intensity. However studies which have investigated the effect of using different breathing assemblies on TLac (Dooly *et al.*, 1996) and T_{vent} (Dooly *et al.*, 1996; Evans and Potteiger, 1995) have found that breathing assembly can affect respiratory rate and tidal volume recorded during progressive exercise but not $\dot{V}O_2$ at TLac.

5.6.3.4 PEDAL CADENCE

Buchanan and Weltman (1985) found that pedal cadence ($rev \cdot min^{-1}$) affected the determination of $\dot{V}O_2$ at OBLA but not TLac. An important finding of this study was that pedal rate had a significant affect on power at TLac and OBLA. Power at TLac and OBLA was lower when cadence was 120 $rev \cdot min^{-1}$ compared with 60 $rev \cdot min^{-1}$ but was similar when pedal cadence was 90 and 120 $rev \cdot min^{-1}$. Also, power at OBLA was lower at 120 $rev \cdot min^{-1}$ when compared with 60 and 90 $rev \cdot min^{-1}$ with no difference between 60 and 90 $rev \cdot min^{-1}$. Similarly, Hughes *et al.* (1982) assessed the effect of pedal cadence on power and $\dot{V}O_2$ at TLac. No difference was found for $\dot{V}O_2$ at TLac when subjects cycled at 50 and 90 $rev \cdot min^{-1}$ however power at TLac was significantly lower with a faster pedal rate.

5.6.3.5 METHODS USED TO DETECT THRESHOLD INTENSITY

Studies by Beaver *et al.* (1985; 1986), Campbell *et al.* (1989) and Sherrill *et al.* (1990) have compared the use of different statistical methods to detect a blood lactate threshold level of performance. In the study by Beaver *et al.* (1985) threshold exercise intensity was identified using a continuous incremental exercise protocol with an increment of 15 W·min⁻¹ and blood samples collected every 2 min. Log-log transformation was used to determine TLac as the point of intersection between two linear regression lines (an example of this method is shown in Figure 6).
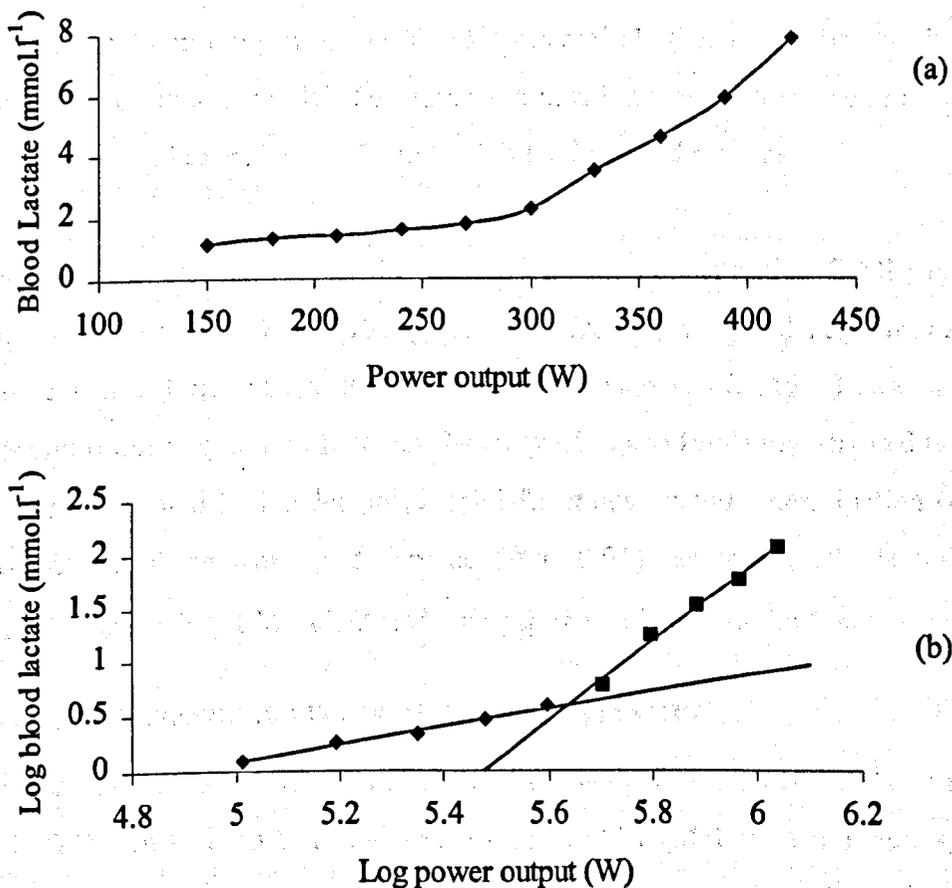
Figure 6 (a). A typical blood lactate response relative to power output during an incremental exercise test (b) shows the log-log plot of blood lactate vs power output. The two segments of data points are obtained using visual inspection of Figure 6 (a). Linear regression lines are fitted and the intersection point is identified as the blood lactate threshold (TLac)

An important feature of this method is that the division point between the two segments was selected visually to detect the point at which the steep portion of the curve occurred. Due to the time gap between blood samples it was necessary to estimate the point at threshold. It was assumed that the point of intersection of the linear regression lines would determine the point of 'transition' between below and above threshold conditions, regardless of the time between samples and the increase in workload between stages.

Campbell *et al.* (1989) compared the log-log threshold detection model of Beaver *et al.* (1985) with a continuous exponential model. Analysis of the data revealed that the mean square error for the residuals between actual and predicted values from the regression of blood lactate on oxygen uptake was significantly higher in the threshold model compared with the continuous model. The authors concluded that the increase in blood lactate during an incremental exercise test did not exhibit a threshold response.

In order to determine the nadir in blood lactate concentration (LMP) during the lactate minimum test Carter *et al.* (1999), Jones and Doust (1998) and Tegtbur *et al.* (1993) used a cubic spline function. In contrast to this Davison *et al.* (1997) assessed LMP using a second order polynomial function. Notably Morgan (1998) investigated the effect of using cubic spline and 2nd order polynomial functions on the determination of W@LMP and found that there was no difference (P = 0.95) between methods however limits of agreement for W@LMP identified using spline and polynomial functions were ± 13 W.

5.6.3.6 REPRODUCIBILITY OF THRESHOLD INTENSITY

Incremental tests provide a reproducible method of assessment of TLac (Dickhuth *et al.*, 1999) OBLA (Jensen and Johansen, 1998) and IAT (McLellan and Jacobs, 1993), however there is no information available concerning the reproducibility of lactate thresholds derived from ramped exercise protocols and lactate minimum tests.

Several studies have assessed the reproducibility of exercise intensity (power output or running speed, $\dot{V}O_2$ and HR) at threshold. For instance Pfitzinger and Freedson (1998) calculated that the biased co-efficient of variation (CV%) for running velocity ($km \cdot h^{-1}$), $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and HR ($b \cdot min^{-1}$) at OBLA were 1.4 , 2.9 and 1.2% respectively with

95%CI estimated as ± 1.14 (km·h⁻¹), ± 3.0 (mL·kg⁻¹·min⁻¹) and ± 3 (b·min⁻¹). Testing protocol consisted of a six stage progressive discontinuous incremental treadmill test. The duration of each stage was 5 min with 1 min rest between each increment. Level of activity and pre-trial preparation were controlled prior to each test and dietary analysis was used to evaluate pre-exercise dietary intake before each trial.

Jensen and Johansen (1998) completed a similar assessment of the reproducibility of exercise intensity detected at OBLA in a group of trained cyclists. Test re-test CV% calculated for power output (W), $\dot{V}O_2$ (L·min⁻¹) and HR (b·min⁻¹) were 5.9, 7.7 and 2.4% respectively with 95%CI of ± 18 (W), ± 0.30 (L·min⁻¹) and ± 4 (b·min⁻¹). Subjects completed a five stage progressive continuous incremental bicycle ergometer test. The duration of each stage was 5 min and each blood sample was collected during the last min of each stage. Pre-trial preparation was controlled, however dietary intake prior to each trial was not checked with dietary analysis. Both studies (Jensen and Johansen, 1998; Pfitzinger and Freedson, 1998) collected blood samples from a fingertip and used the same blood lactate analyser to measure blood lactate concentration.

The finding that the CVs for power and $\dot{V}O_2$ at OBLA reported by Jensen and Johansen (1998) were higher than values estimated by Pfitzinger and Freedson (1998) is difficult to explain. However, total measurement error (CV%) consists of technical error in the equipment and biological variability of the subject (Atkinson and Nevill, 1998). In the study by Heitkamp et al. (1991) the relative contribution of biological variability to overall measurement error was highlighted when investigators found that running tests performed on a treadmill were affected by the training status of the subject and the level of habituation to treadmill running prior to each test. Jensen and Johansen (1998) assessed CV using a test re-test comparison, therefore an improvement in CV across successive trials was not evaluated. Pfitzinger and Freedson (1998) did not calculate CV across trials (1 to 2 and 2 to 3) therefore the effect of test habituation on reproducibility was not considered. It is likely that methodological controls incorporated by Pfitzinger and Freedson (1998) improved the reproducibility of the variables assessed.

Armstrong and Costill (1985) estimated that the day-day variation in $\dot{V}O_2$ and $\dot{V}_E$ recorded during a progressive incremental exercise test would be about 4.0% and 3.6% respectively. These calculations were based on a 68% confidence interval, therefore for 95% confidence, it was suggested that values of 8 and 7% for $\dot{V}O_2$ and $\dot{V}_E$ were more appropriate. Notably, technical error and day to day variation in blood lactate concentration was assessed and total measurement error (CV%) was 13%. Consequently the authors questioned the validity of using a fixed blood lactate concentration to detect a fixed threshold exercise intensity.

5.6.4 ENDURANCE PERFORMANCE

Various factors can influence cycling endurance performance these include; diet, thermoregulation, altitude and training. It is beyond the scope of this thesis to consider the multiple effects of these factors however a recent textbook by Gregor and Conconi (2000) can be used as a reference for further investigation. Studies which have used laboratory based simulated cycling time trials to assess performance have allowed subjects to self-select gear ratio and pedal cadence (El-Sayed *et al.*, 1997; Palmer *et al.*, 1996, 1997, 1998) provided feedback on distance covered (Palmer *et al.*, 1996, 1997, 1998) and verbal encouragement during testing trials (El-Sayed *et al.*, 1997).

5.6.4.1 REPRODUCIBILITY OF POWER

Jeukendrup *et al.* (1996) assessed the reproducibility of endurance capacity and endurance performance tests and found that the coefficient of variation (CV%) for endurance capacity was significantly higher when compared with endurance performance (26% vs 3.5%, respectively). Table 11 shows mean CV calculated for endurance performance time trials.

Atkinson *et al.* (1999) reported that the test re-test 95% ratio limits of agreement for time to complete a 16.1-km time trial were ×/÷ 1.08 (~ 8%) which equates to a CV of ~ 2.9% (Bland, 1996). Hopkins *et al.* (1999) found that CV for power achieved during repeated time trial tests was also consistently higher than CV for completion time. This discrepancy was due to the non-linear relationship used to convert power to speed in order to imitate cycling performance when using a cycle simulator.

Table 11. Mean CV% for time trial performance time (min:s)

Study	Subjects (*n*)	Tests (*k*)	Time (min:s)	CV (%)
Hickey *et al.* (1992)	8	4	105:00	1.01
Jeukendrup *et al.* (1996)	10	5	62:13	3.40
Westgarth-Taylor *et al.* (1997)	8	2	57:12	1.00
Lindsay *et al.* (1996)	8	3	57:05	0.89
Hickey *et al.* (1992)	8	4	12:00	0.95
Hickey *et al.* (1992)	8	4	00:55	2.43

5.6.4.2 REPRODUCIBILITY OF HEART RATE

Very few studies have assessed the reproducibility (CV) of average heart rate recorded during endurance performance tests. However, Bishop (1997) calculated that the CV for average heart rate during a 1-h performance ride was 2.0%.

5.7 SUMMARY

Various testing methods have been developed to quantify selected physiological responses during endurance capacity tests. Numerous studies have highlighted methodological issues concerning different types of testing equipment and assessments. Investigators need to be aware of these issues when comparisons are made within and between studies.

CHAPTER 6

6 A COMPARISON OF POWER RECORDED USING A KINGCYCLE AND SRM POWER METER

6.1 INTRODUCTION

In order to produce a valid and reproducible assessment of performance capacity in racing cyclists it is essential to quantify the measurement error of equipment used to record exercise data (Atkinson and Nevill, 1998). The power output of elite cyclists is a key determinant of endurance performance (Coyle *et al.*, 1991) and therefore the validity of a power measuring device is extremely important; when comparing performance between cyclists, monitoring the effectiveness of training or investigating the use of ergogenic aids. This is particularly significant when a test is performed on different cyclists and used to establish a performance criterion for team selection and talent identification. The reproducibility of a cycling performance test has important implications on research design and the sample size required to detect the smallest worthwhile changes in performance (Hopkins *et al.*, 1999; Schabort *et al.*, 1998b). However few studies have assessed the relative contribution of technical error and biological variability to the reproducibility of cycling performance tests.

The Kingcycle air-braked cycle ergometer (which allows cyclists to ride their own bicycles in a laboratory setting) has been used in various studies to assess the reproducibility of laboratory based cycling performance (Palmer *et al.*, 1996; Schabort *et al.*, 1998a) investigate the effects of training on maximal power (Lindsay *et al.*, 1996; Westgarth-Taylor *et al.*, 1997) and study the effects of ergogenic aids (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Palmer *et al.*, 1998) on endurance performance. Surprisingly, there is no information available concerning the reproducibility and validity of power output recorded using the Kingcycle system.

Recently, Jones and Passfield (1998) and Martin *et al.* (1998) reported that the SRM power meter (an innovative cycle ergometry system which records power output during laboratory and field based trials) provides a valid (Jones and Passfield, 1998; Martin *et al.*, 1998) and

reliable (Jones and Passfield, 1998) measure of power when compared with a Monark cycle ergometer.

Martin *et al.* (1998) reported the difference between SRM power and Monark power to be 2.36 % ($r^2 = 0.997$). It was suggested that the difference in power output between SRM and Monark was due to a loss of power (~ 2%) in the ergometer chain drive system. This postulate is supported by the work of Kyle and Caiozzo (1986) who reported that losses in power transmission increased with power output and were between 1.9-3.9% for the range of power outputs used in the study of Martin *et al.* (1998). It is worth noting that transmission losses vary with pedal rate and power output which explains the difference between the values reported by Kyle and Caiozzo (1986) and Martin *et al.* (1998) (50 rev·min⁻¹ compared with 90 rev·min⁻¹, respectively).

Jones and Passfield (1998) showed that the random error calculated using ratio limits of agreement (Bland and Altman, 1986) for SRM compared with Monark were ± 1.8% with no significant (P>0.05) bias between the SRM system and Monark ergometer. Also, Jones and Passfield (1998) compared 4 and 20 strain gauge SRM and using 95% ratio limits of agreement found that the 4 strain gauge power meter provided a less reliable measure of power output compared with the 20 strain gauge SRM (± 1.8 and ± 0.3%, for 4 and 20 strain gauge SRM respectively). Van Praagh *et al.* (1995) stated that the error in power output recorded using a cycle ergometry system should be within ± 5%.

6.2   AIM OF STUDY 1

To evaluate the validity and reproducibility of a cycling ergometry system for the assessment of cycling performance.

6.3   OBJECTIVES

6.3.1   PART 1

To compare power recorded using the Kingcycle ergometer and SRM powermeter during a continuous incremental exercise test (LT$_{inc}$), a peak power test (PP) and a laboratory based 16.1-km cycling time trial (LTT).

6.3.2 PART 2

To compare power recorded using the Kingcycle and SRM powermeter during two continuous incremental exercise tests (LT_{inc}).

6.3.3 PART 3

To compare power recorded using the Kingcycle and SRM powermeter during three Kingcycle peak power (PP_S) tests using a stabilising kit with calibration checked against SRM.

6.3.4 PART 4

To compare power recorded using the Kingcycle and SRM powermeter during two peak power (PP_{NS}) tests completed without the use of a stabilising kit and calibration check.

6.4 METHODS

Before testing, each participant gave written informed consent when the nature and purpose of the tests had been fully explained. Before participating in the exercise trials subjects underwent habituation sessions in order to familiarise themselves with the testing procedures. Throughout each trial, laboratory conditions were maintained (ambient temperature 18-22°C, relative humidity 45-55%) and subjects were cooled using an electric fan.

Body mass was measured to the nearest 0.1 kg by means of beam balance scales (Seca, Germany). Pre-trial body mass was recorded on each visit to the laboratory with the subject barefooted, having removed all items of clothing except for cycling shorts. Standing body height measurements were recorded to the nearest 0.5 centimetre by means of a beam balance scales mounted stadiometer (Seca, Germany).

For each testing trial, subjects used a racing bicycle fitted with a SRM power meter (SRM, Jülich, Welldorf, Germany) with bicycle attached to a Kingcycle test rig (EDS Portaprompt Ltd, High Wycombe, UK). Power output (W) was recorded per 1 min using a Kingcycle rig with 5.5 computer software, sampling at a frequency of ~ 0.5 Hz, personal communication and by either a 4 or 20 strain gauge SRM with pedalling torque inductively transmitted at 500 kHz with power recorded at 1-s intervals (Jones and Passfield, 1998).

For all comparisons of power output reported in the study the average power recorded during a 60-s period was calculated. The calibration procedure and technical description of the Kingcycle test rig with 5.5 software has been previously described by Palmer *et al.* (1996). A full description of the technical aspects of the SRM has also been provided by Jones and Passfield (1998).

In order to calculate power output using the Kingcycle rig with version 5.5 computer software (EDS Portaprompt Ltd, High Wycombe, UK), bicycle rear tyre pressure was standardised using a tyre pump with pressure gauge (Silca, Italy). The subject's bicycle was secured to the ergometer by the front fork and supported by an adjustable pillar which rested beneath the bottom bracket. Through the vertical adjustment of the pillar the height of the bottom bracket was changed to standardise the rolling resistance placed on the rear tyre of the bicycle by an air-braked flywheel. This was achieved through a series of 'run down' calibrations during which the subject produced a power output of ~ 300 W immediately stopped cycling and maintained their normal racing position, during the deceleration of the flywheel the height of the pillar was adjusted so that a computer display confirmed the slowing of the flywheel was equal to the power decrease of a 65 kg cyclist. With the rolling resistance equal to that of a 65 kg cyclists riding on level terrain, an IBM compatible computer calculated the power output (W per minute) from the angular velocity of the flywheel using the following equation: Power output (W) $= 0.000136$ (rev·s^{-1})^2 + 1.09 rev·s^{-1}. Revolutions per second (rev·s^{-1}) were monitored using a photo-optic sensor (Kingcycle 5.5 software, sampling frequency ~ 0.5 Hz). The speed relative to power output (flywheel velocity) was calculated using the following equation: Speed (km·h^{-1}) = 3.0445·(power output (W) $^{0.4638}$).

In order to record power using the SRM ergometry system the chain-set of the subject's bicycle was removed and replaced by either a 4 or 20 strain gauge torque transducer 'power meter'. This transformed the force on the pedals (torque) and the pedal cadence (rev·min^{-1}) into a digital signal transmitted telemetrically (inductively transmitted at 500 kHz) to a sensor on the bicycle frame. Once fitted to the subject's bicycle the power meter was calibrated using the appropriate system software. Power output (W) recorded using SRM was calculated from the formula Power (W) = (F × D)/T with F; the torque (Nm) measured

by the strain gauges and D; the angular velocity of the crank set (cadence $\times 2\pi$; rad·s^{-1}) and T; the unit time (s) F and D are recorded.

6.4.1 PART 1

6.4.1.1 SUBJECTS

Twelve endurance trained male cyclists who regularly competed in local cycling time trial races participated in this part of the study. Subjects included highly trained senior (n = 6) and veteran cyclists (n = 6). Each subject completed three exercise tests i) a continuous incremental exercise test (LT$_{inc}$), ii) a peak power test (PP) and iii) a 16.1-km time trial (LTT). Tests were performed at the same time of day and separated by at least one week. Characteristics of the subjects are shown in Table 12.

Table 12. Characteristics of subjects who participated in study 1 (mean ± SD)

Part	n	Age (yr)	Height (m)	Body mass (kg)
1	12	43 ± 16	1.80 ± 0.06	76.1 ± 4.9
2	8	42 ± 15	1.83 ± 0.06	75.6 ± 5.5
3	9	32 ± 5	1.79 ± 0.04	71.1 ± 4.7
4	9	34 ± 13	1.84 ± 0.05	75.1 ± 6.0

6.4.1.2 KINGCYCLE CONTINUOUS INCREMENTAL TEST (LT$_{INC}$)

The duration of each increment was 4 min, with work rate increased by 24 W per stage with the required power output designated by the Kingcycle software. Starting power was 45% of W$_{peak}$ achieved during a habituation peak power test. Incremental stages were continued until subjects were unable to maintain the required power output. For comparison purposes the average one min power output recorded during the third min of each increment was calculated. The first two minutes and the fourth minute of each stage were not used for comparison due to the difference in power recorded using SRM when subjects were required to increase the workload between stages. This was due to the anomaly that the Kingcycle test rig recorded power based on the angular velocity of the flywheel and SRM recorded power output based on the angular torque within the crank set.

Therefore the extra power required to increase the angular velocity of the flywheel was recorded using SRM but not Kingcycle.

6.4.1.3 KINGCYCLE PEAK POWER (PP) TEST

On arrival at the laboratory, bicycle rear tyre pressure was standardised using a track pump with tyre pressure gauge (Silca, Italy) and calibration of the Kingcycle test rig was completed as previously outlined. For each PP test, subjects were instructed to warm-up at a self selected intensity. On completion of their warm-up each subject completed five minutes of continuous cycling, maintaining the starting power calculated for the PP test. During the PP test work rate increased per min by $5.0 \pm 0.2\%$ (mean $\pm$ SD) of peak power achieved during an habituation test. Ramp rate during the test ranged from 15 to 25 $W \cdot min^{-1}$, dependent on each individual cyclist's predicted peak power. Starting power output for the test was calculated for subjects to reach volitional exhaustion between 9-11 min. During tests gear ratio and pedal cadence ($rev \cdot min^{-1}$) were self selected. Peak power (W_{peak}) was calculated as the highest average power recorded during any 60-s period of the test.

6.4.1.4 LABORATORY BASED KINGCYCLE 16.1-KM TIME TRIAL (LTT)

Each subject completed a laboratory based 16.1-km time trial (LTT). Prior to the time trial each rider was instructed to follow their normal preparations for competing in a 16.1-km race. During the time trial subjects were informed of elapsed time (min:s), distance covered (km) and heart rate ($b \cdot min^{-1}$). To imitate racing conditions, each cyclist raced against a performance matched rider using another Kingcycle test rig. During each race verbal encouragement was provided by the same investigator.

6.4.2 PART 2

6.4.2.1 SUBJECTS

Eight subjects completed two continuous incremental exercise tests which were separated by one week. All subjects were competitive cyclists who had previous experience of the testing procedure. Characteristics of the subjects are shown in Table 12.

6.4.2.2 KINGCYCLE CONTINUOUS INCREMENTAL TEST (LT$_{INC}$)

Following a five min warm-up at a power output of about 45% of W_{peak}, subjects completed a continuous incremental exercise protocol. Starting power was ~ 45% W_{peak} and workload

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increased by 24 W at the end of each 4 min stage. Subjects selected their own pedal cadence (rev·min^{-1}) and were allowed to change gear *ad libitum*. The test was completed when subjects reached volitional exhaustion. For comparison purposes the average one min power output recorded during the third min of each increment was calculated.

6.4.3 PART 3

6.4.3.1 SUBJECTS

Nine highly trained competitive cyclists completed three Kingcycle peak power (PP$_S$) tests, performed at the same time of day and separated by at least one week. Characteristics of the subjects are shown in Table 12.

6.4.3.2 KINGCYCLE PEAK POWER TEST (PP$_S$)

Before each PP$_S$ test, a stabilising kit (supplied by the manufacturers of the Kingcycle) was fitted to the Kingcycle ergometer. This consisted of a strap fed through the rails of the subject's bicycle saddle and secured firmly to the ergometer frame. The stabilising kit was used to minimise changes in resistance between the tyre of the bicycle rear wheel and the roller of the air-braked flywheel. During the standard calibration procedure for the Kingcycle, the bias between SRM (W) and Kingcycle (W) was checked and the Kingcycle ergometer was adjusted until the difference in power recorded using the two systems was standardised at SRM (W) = ~ 0.90 of Kingcycle (W). The value of 0.90 was based on data collected from previous tests which had compared power recorded using each ergometry system (Balmer, unpublished observations). For each PP$_S$ test, subjects were instructed to warm-up at a self selected intensity. On completion of the warm-up each subject completed a peak power test as previously described. Ramp rate selected for the first PP$_S$ test was repeated for subsequent tests. Peak power (W$_{peak}$) achieved during the test was calculated as the highest average power during any 60-s period of the test using Kingcycle (King$_{Wpeak}$) and SRM (SRM$_{Wpeak}$). Subjects were not informed of their performance until all PP$_S$ tests had been completed.

6.4.4 PART 4

6.4.4.1 SUBJECTS

Nine highly trained competitive cyclists completed two Kingcycle peak power (PP_{NS}) tests which were performed on two separate occasions separated by a mean ± SD period of 54 ± 32 days. Characteristics of the subjects are shown in Table 12.

6.4.4.2 KINGCYCLE PEAK POWER TEST (PP_{NS})

Each subject completed two Kingcycle peak power tests (as previously described above) however, the stabilising kit and standardised calibration procedure used for the PP_S tests were not used in this part of the study.

6.4.5 STATISTICAL ANALYSES

For part 1 comparisons of power output recorded using the Kingcycle and SRM systems were performed using Bland and Altman's 95% limits of agreement (Bland and Altman, 1986). A full description of this method of reproducibility testing is also provided by Atkinson and Nevill (1998). If heteroscedasticity was present in the data, comparisons were logarithmically transformed to calculate the ratio limits of agreement. Correlation coefficients and 95% confidence interval (95%CI) were calculated using the methods outlined by Cohen and Holliday (1983) and Zhu (1997) respectively.

For part 2, the overall reproducibility of Kingcycle power was assessed using the difference score between ln transformed values for SRM and Kingcycle power of each individual during the repeated incremental tests with within-subject reproducibility (mean coefficient of variation, CV) derived by two-way analysis of variance (ANOVA) as described by Schabort *et al.* (1998b).

For parts 3 and 4 measures of within-subject reproducibility (mean coefficient of variation, CV) were derived by two-way analysis of variance (ANOVA) and 95% confidence intervals (95%CI) were calculated for CV using the methods of Tate and Klett (1959). Statistical significance ($P < 0.05$) for comparison between power recorded using SRM and Kingcycle was assessed with a paired Student's t-test and ANOVA. For comparison with the CV calculated in the present study, the ratio limits of agreement (Bland and Altman,

1986) of ± 1.8% reported by Jones and Passfield (1998) for SRM power compared with a Monark ergometer were divided by 2.8 (Bland, 1996). The value of 2.8 was calculated from the equation that limits of agreement are equal to 2·√2 multiplied by the within subject variation derived from ANOVA. Statistical analyses were completed using Microsoft Excel (Bellevue, WA) and Minitab (State College, PA). Values in the text are mean (± SD) unless otherwise stated.

6.5 RESULTS

6.5.1 PART 1

For part one of the study, a very strong correlation coefficient was calculated for power output recorded during the incremental test using SRM and Kingcycle (r = 0.99, 95% SEE 3.5%, n = 83). Data was not normally distributed and the correlation between the absolute differences between SRM and Kingcycle and the mean of SRM power and Kingcycle power was significant (r = 0.38). This showed that heteroscedasticity was present (see Figure 7) therefore the data was logarithmically transformed and the correlation was reduced (r = 0.21). Limits of agreement were expressed as ratios based on clear evidence that the absolute error was greater for higher power outputs. The ratio limits of agreement between SRM and Kingcycle were 0.90 (95%CI = 0.89-0.91) ×/÷ 1.07 (n = 83). The average bias of 0.90 was found to be highly significant using a paired t-test of the Kingcycle and SRM ln transformed values (t = 25.2, P<0.001).

Preliminary analysis of the data revealed that the difference between SRM and Kingcycle power for one subject was outside the estimated 95% confidence intervals. The data of this subject was not included in the subsequent analysis. Peak power (W_{peak}) and average power recorded for LTT were significantly higher (P<0.001) using Kingcycle compared with SRM (443 ± 65 vs 399 ± 54 W, 335 ± 51 vs 307 ± 47 W respectively). Correlation coefficients between SRM and Kingcycle were, 0.97 for W_{peak} (95%CI = 0.89-0.99, SEE 4% 95%CI = 3-5%) and 0.98 for LTT (95%CI, 0.92-0.99, SEE 4%, 95%CI = 3-6%). However further analysis of the data for SRM compared with Kingcycle showed ratio limits of agreement of 0.92 (95%CI = 0.90-0.94), ×/÷ 1.07 for W_{peak} and 0.90 (95%CI =

0.88-0.92) ×/÷ 1.07 for LTT. The average bias for the 2 tests (n = 22) for SRM compared with Kingcycle was 0.91 (95%CI = 0.90-0.92) and the random error was 1.07.
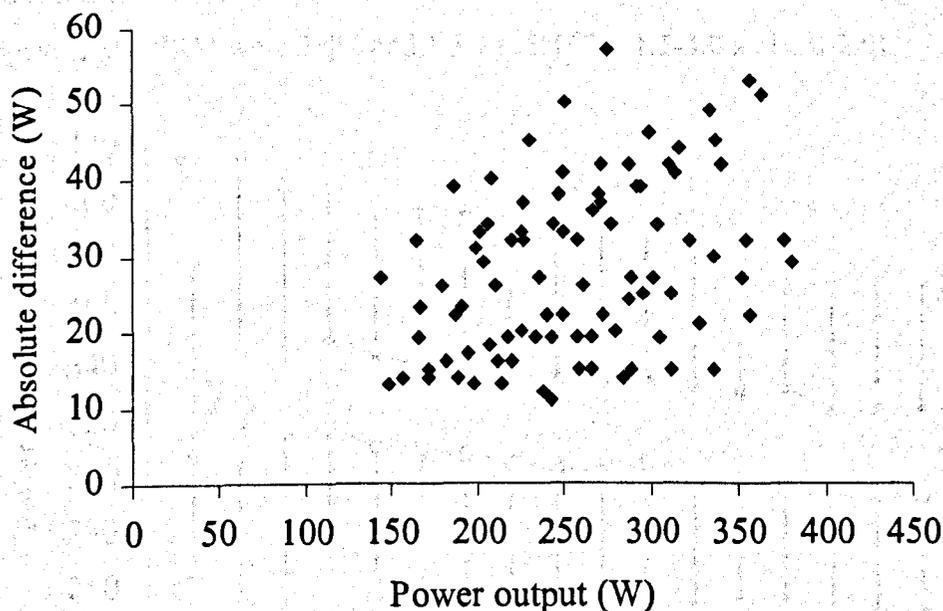
Figure 7. A plot of the absolute differences between power output recorded using SRM and Kingcycle and the individual means, for the examination of heteroscedasticity

Analysis of power recorded at 5% intervals during the LTT revealed that Kingcycle power was significantly higher (P<0.001) than SRM for the duration of the time trial except for the first 5% interval (see Figure 8). There was no change in the difference between power output recorded by Kingcycle and SRM at 5% intervals from 5-100% of the ride.

6.5.2   PART 2

Each subject (n = 8) completed seven stages for each incremental exercise test and the overall mean CV for the difference between Kingcycle and SRM power recorded during the stages of the repeated incremental test was ± 1% (at 68%CI).

6.5.3   PART 3

Table 13 shows the mean (± SD) $King_{Wpeak}$ and SRM_{Wpeak} recorded during the PP_S tests. $King_{Wpeak}$ was higher (P<0.001) in trial two compared with trials one and three. However there was no difference (P>0.05) across the three PP_S trials for SRM_{Wpeak}. The mean (± SD)

times for completion of the PP_s were 9:53 ± 0:40, 10:19 ± 0:34, and 9:57 ± 0:34 for tests one to three respectively. The mean improvement in $King_{Wpeak}$ of ~10 (W) for test two compared with tests one and three, represented an increase (P = 0.04) in mean time to exhaustion of about 24-s. The mean CV% for peak power achieved during the PP_s trials for Kingcycle and SRM were 2.0% and 1.3% (95%CI = 1.5-3.0 and 1.0-2.0).
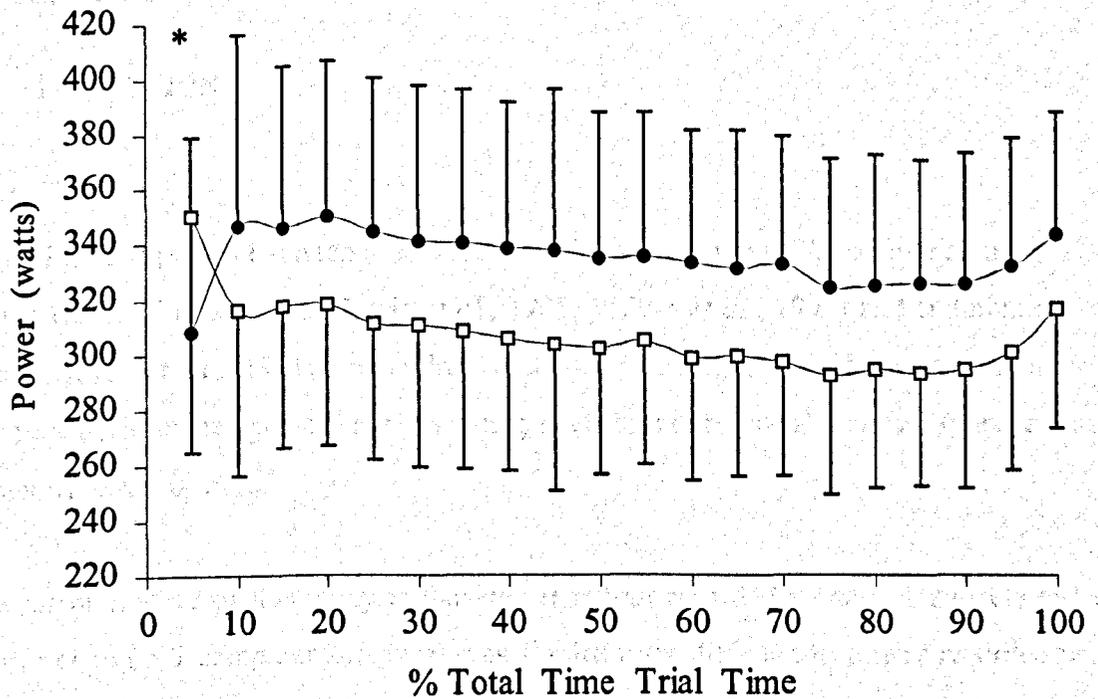
Figure 8. The relative values (means ± SD, n = 11) for power recorded during each 5% segment of a 16.1-km time trial (−•− for Kingcycle, −□− for SRM). * denotes significant (P<0.001) interactive difference

Table 13. Mean ± SD values for peak power recorded during 3 PP_s tests (n = 9)

	Trial 1	Trial 2	Trial 3	Mean
Kingcycle (W)	428 ± 50*	437 ± 48*$	426 ± 48*	430 ± 47*
SRM (W)	392 ± 45	394 ± 44	391 ± 43	392 ± 42

denotes significantly higher than SRM (P<0.05)

$ denotes significantly higher than trial 1 and 3 for Kingcycle (P<0.05)

6.5.4 PART 4

The mean ± SD of each individuals peak power recorded during the two PP_{NS} tests using Kingcycle and SRM were 457 ± 60 vs 453 ± 51 and 410 ± 51 vs 411 ± 44, for $King_{Wpeak}$ and SRM_{Wpeak} respectively. There was no difference (P>0.05) for $King_{Wpeak}$ and SRM_{Wpeak} recorded during the PP_{NS} trials. The mean CVs for $King_{Wpeak}$ and SRM_{Wpeak} were 4.6% (95%CI = 2.7-7.6) and 3.6% (95%CI = 2.1-6.0).

6.6 DISCUSSION

6.6.1 PART 1

Several investigations have reported values for the power output (W) of elite racing cyclists during maximal power tests (Lucia *et al.*, 1998; Wilber *et al.*, 1997) and endurance based races (Coyle *et al.*, 1991; Jeukendrup and van Diemen, 1998). However in order to compare performance power between racing cyclists it is essential that the equipment used to record power is valid.

The major finding of this study is that power output recorded using a Kingcycle test rig (with version 5.5 computer software) was significantly different to power recorded using an SRM ergometry system. For instance, using the ratio limits of agreement calculated from SRM and Kingcycle it was possible for a cyclist who averaged 335 W for a 16.1-km indoor time trial to record between 281-325 SRM (a range of 44 W). It is worth noting that the range of values for SRM power are below the mean value of 335 W recorded using the Kingcycle. This discrepancy was due to the 10% difference between SRM (W) and Kingcycle (W). Therefore, two cyclists using two SRM and two Kingcycle rigs could be riding a laboratory based time trial race at the same power recorded using the Kingcycle but with different (up to ± 7%) power recorded using SRM.

Although Martin *et al.* (1998) found that the difference between power delivered to the flywheel of a Monark ergometer versus power recorded with SRM was 2.4%, it was assumed that SRM power was valid due to a loss of efficiency when using a chain drive system. Unfortunately it is not possible to evaluate the variability in power measurement between SRM and Monark as Martin *et al.* (1998) did not report the limits of agreement in

this study. Jones and Passfield (1998) calculated the limits of agreement between power delivered to the flywheel of a Monark ergometer and SRM power to be less than ± 1.8%. Van Praagh *et al.* (1992) suggested that error in ergometry should be less than 5%, therefore the findings of this study for inter-individual differences in power revealed that across subjects the error in the Kingcycle ergometry system exceeded 5% when compared with SRM.

The calibration of the Kingcycle test rig is dependent on maintaining a constant resistance between the roller of the flywheel and the tyre of the rear wheel (Palmer *et al.*, 1996). Therefore changes in the resistance between tyre and roller will affect the power output required to rotate the flywheel. Similarly as the Kingcycle flywheel is air-braked, the air resistance imposed on the tyre is proportional to the speed of rotation. It is interesting to note that the subject who was excluded from the data analysis for part 1 of the present study had used a disc wheel during tests which may have reduced the force required to overcome the resistance imposed on the flywheel.

A change in weight distribution on the bicycle also affects the resistance between the roller and tyre. Therefore during each test, subjects are required to maintain their cycling position as much as possible. However during exercise trials subjects frequently move on the saddle (personal observation) and subsequently tend to pull forward or sit back, respectively reducing or increasing the resistance between rear wheel and Kingcycle. Mean data for the 16.1-km time trial showed that the difference in power between SRM and Kingcycle remained relatively consistent for the duration of the ride, however it is worth noting that power recorded using SRM was higher for the first 5% of the ride due to the extra power required to accelerate the Kingcycle flywheel to the self-selected velocity.

6.6.2   PARTS 2, 3 AND 4

In order to detect changes in cycling performance, it is essential to record power output using a reliable cycle ergometry system (see Hopkins *et al.*, 1999). Data from the present study showed that during the repeated PP_S tests, there was a significant change in performance when peak power was recorded using the Kingcycle, however there was no change in performance when peak power was recorded using SRM. Possible explanations

for the increase in King$_\text{Wpeak}$ recorded during the second PP$_\text{S}$ test include i) the effect of error in the calibration procedure which increased the difference (%) between SRM (W) and Kingcycle (W) and ii) in an effort to achieve a higher peak power in the second PP$_\text{S}$ test, subjects may have changed body position by moving forward on the saddle, this would have decreased the resistance between the tyre of the rear wheel of the subject's bicycle and the roller of the Kingcycle flywheel. Hopkins *et al.* (1999) suggested that the reproducibility of power output recorded using the Kingcycle air braked ergometer could be affected by changes in ambient temperature, humidity and the pressure of the bicycle tyres. However the increase in King$_\text{Wpeak}$ for the second PP$_\text{S}$ test in the present study could not be attributed to these factors, as each subject completed their PP$_\text{S}$ tests on different days within a three to four week period and the overall time period of the study was about 14 weeks.

During each PP$_\text{S}$ and PP$_\text{NS}$ test, peak power recorded using the Kingcycle test rig was significantly higher than power recorded using SRM. This discrepancy in power output between Kingcycle (W) and SRM (W) should be considered when peak power output values recorded using the Kingcycle test rig are compared with maximal values reported in other studies (Lucia *et al.*, 1998; Wilber *et al.*, 1997). Notably Jones and Passfield (1998) and Martin *et al.* (1998) have confirmed that the SRM power meter (W) can provide a valid measure of power when compared with a Monark ergometer, therefore it is reasonable to assume that the Kingcycle test rig may not always provide an accurate measure of power output during peak power tests.

When test re-test power output values recorded using the Kingcycle and SRM were compared for the 'constant load' conditions of an incremental exercise test, analysis of the data revealed that the variation (CV%) in Kingcycle when compared with SRM was reasonably low, therefore the reproducibility of Kingcycle power recorded during this method of testing was acceptable. This finding is important as it shows that variability in Kingcycle power when compared with SRM is influenced by the dynamics of the cycling exercise. For comparison purposes, power recorded using each measuring device was calculated as the average power during 60-s of exercise, however, this finding suggests that power recorded during ramp exercise protocols using SRM is affected by the inertial

characteristics of the Kingcycle flywheel and the ability of the cyclist to maintain a relatively 'smooth' increase in power during the test.

In order to evaluate the effect of training/detraining on peak power output it is important to have an estimation of the range of variation which is due to the technical error of the testing equipment and the biological variability of the individual subjects. Data from this study showed that for a change in power to be outside the 95% confidence intervals estimated from the coefficient of variation for $King_{Wpeak}$ and SRM_{Wpeak} a change in performance would need to exceed 3% and 2% respectively. Therefore a rider who achieved a $King_{Wpeak}$ of 460 W and SRM_{Wpeak} of 415 W would need to increase/decrease peak power output by about 14 W for $King_{Wpeak}$ and 9 W for SRM_{Wpeak}. This finding has important implications when estimating the sample size required to detect the smallest worthwhile (~ 1%) change in performance (Hopkins et al., 1999; Schabort et al., 1998b). For instance when estimating the sample size (Hopkins, 1997) using the data from the PP_S tests (for a repeated measures design without a control group, type I error rate of 5% with statistical power of 80%) power calculations using the CVs for $King_{Wpeak}$ and SRM_{Wpeak} estimate that 28 cyclists would be needed to detect a 1% change in performance using the CV for SRM_{Wpeak} however this number is increased to 61 cyclists based on the CV calculated for $King_{Wpeak}$. Obviously such a marked difference in sample size will have a major effect on resources.

Mean CVs estimated for $King_{Wpeak}$ and SRM_{Wpeak} recorded during the PP_{NS} tests, were higher than CVs recorded during part one of the investigation. Possible reasons for this difference include i) an increase in biological variability due to the longer period of time between PP_{NS} tests when compared with the PP_S tests and ii) technical error of the Kingcycle was higher during the PP_{NS} tests due to the exclusion of the calibration check and stabilising kit. It is difficult to assess the relative contributions of biological variability and technical error to the CV's estimated for PP_{NS} and PP_S trials. However using the calculation of Jones and Passfield (1998) that the ratio limits of agreement for SRM power output compared with a Monark ergometer were ± 1.8%, it is possible to estimate that the CV for the technical error of the SRM power meter was ~0.6%. Therefore biological variability during the PP_S tests contributed ~ 1.2% of the mean CV for both $King_{Wpeak}$ and

SRM_{Wpeak} and the technical error for $King_{Wpeak}$ was ~ 1.8% of the mean CV. Similarly for the PP_{NS} tests it is possible to estimate that ~ 3.5% of the change in $King_{Wpeak}$ and SRM_{Wpeak} was due to biological variability and that the technical error in $King_{Wpeak}$ was ~ 2.9% of the mean CV. It is reasonable to suggest that the calibration check and stabilising kit used in part one of the study reduced the technical error in $King_{Wpeak}$ by about 1%.

Very few investigators have assessed the relative contribution of technical error to total measurement error when investigating the reproducibility of cycling performance tests. Notably, van Praagh *et al.* (1995) suggested that the range of technical error for power output recorded using cycle ergometers should be within ± 5%. However this value appears to be relatively high considering the mean CVs for power recorded during repeated laboratory based cycling tests (Hopkins *et al.*, 1999). Data from this study showed that the reproducibility of a cycling test used to assess peak power can be improved with a more reliable and reproducible power measuring device. This finding has important implications on research design and the sample size required to detect changes in performance during repeated tests (Schabort *et al.*, 1998b).

6.7 SUMMARY

Data from the present study questions the validity of values reported for power output recorded using a Kingcycle test rig (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Lindsay *et al.*, 1996; Palmer *et al.*, 1997; 1998; Westgarth-Taylor *et al.*, 1997; Weston *et al.*, 1997) and shows that a Kingcycle test rig (with version 5.5 computer software) did not provide an accurate measure of power when compared with the SRM ergometry system. The mean bias between power recorded using the Kingcycle when compared with SRM was about +10% with a random error of 7%.

Investigators should be aware of the difference in peak power values recorded using SRM and Kingcycle and that a SRM power meter can be used to provide a more reproducible measure of peak power output during a Kingcycle PP test. However, during relatively constant load conditions the reproducibility of Kingcycle power is acceptable.

CHAPTER 7

7 REPRODUCIBILITY OF PEAK PHYSIOLOGICAL VARIABLES AND PERFORMANCE RELATED RESPONSES DURING CYCLING TESTS

7.1 INTRODUCTION

In order to detect a change in cycling endurance performance it is necessary to use a laboratory based assessment of endurance capacity and cycling performance which is reliable and valid (see Hopkins *et al.*, 1999). The Kingcycle peak power test has been used by several authors (El-Sayed *et al.*, 1997; Palmer *et al.*, 1998; Hawley *et al.*, 1997) to assess the exercise performance capacity of racing cyclists, and laboratory based Kingcycle time trials have been used on several occasions to investigate determinants of cycling endurance performance (El-Sayed *et al.*, 1997; Hawley *et al.*, 1997; Lindsay *et al.*, 1996; Stepto *et al.*, 1999; Westgarth-Taylor *et al.*, 1997).

7.2 AIM OF STUDY 2

To evaluate the range of measurement error associated with the assessment of key determinants of cycling endurance performance ability.

7.3 OBJECTIVES

7.3.1 PART 1

To assess the reproducibility of selected peak metabolic, cardio-respiratory and performance related responses recorded during a Kingcycle peak power test.

7.3.2 PART 2

To assess the reproducibility of selected performance related responses recorded during a Kingcycle peak power and 16.1-km time trial test.

7.4 METHODS

7.4.1 SUBJECTS

All of the subjects who participated in the study were well trained male endurance cyclists with extensive experience of competitive cycling. Before testing, each volunteer gave written informed consent when the nature and purpose of the tests had been explained. Subsequently each subject completed an habituation test in order to familiarise themselves with the laboratory environment and standard testing procedure. For each testing trial subjects were instructed to refrain from strenuous exercise for 24-h immediately prior to the test and to maintain their normal daily activity pattern and dietary intake as if preparing for a 16.1-km cycling time trial race. Subjects served as their own control and were requested to replicate the same level of activity and dietary intake for 24-h immediately prior to each test. Tests were conducted at the same time of day and laboratory conditions were maintained during testing (ambient temperature 18-22°C, relative humidity 45-55%).

Table 14 Characteristics of subjects who participated in study 2 (mean ± SD)

Part	n	Age (yr)	Height (m)	Body mass (kg)
1	9	32 ± 5	1.79 ± 0.04	71.1 ± 4.7
2	9	35 ± 14	1.82 ± 0.07	76.0 ± 3.7

n = number of subjects

7.4.2 PART 1

Nine subjects (characteristics shown in Table 14) completed three Kingcycle peak aerobic capacity tests (PAC) separated by 11 ± 6 days (mean ± SD). For the PAC test, subjects completed the Kingcycle peak power test previously outlined in 6.4.1.3, however in order to assess peak aerobic capacity, expired air was measured using a Covox online gas analysis system (Covox, Exeter, UK). Peak values for oxygen uptake ($\dot{V}O_2$), carbon dioxide ($\dot{V}CO_2$), ventilation ($\dot{V}_E$) respiratory exchange ratio (RER), ventilatory equivalents ($\dot{V}_E/\dot{V}O_2$, $\dot{V}_E/\dot{V}CO_2$), and breathing frequency (f) were determined as the highest average values recorded for 1 min during the test. All values for $\dot{V}O_2$ (L·min⁻¹), $\dot{V}CO_2$, (L·min⁻¹), and $\dot{V}_E$ (L·min⁻¹) reported in the text are STPD.

During tests, expired air was measured at 1 min intervals and pulmonary ventilation was calculated by measuring inhaled flow rates (contamination with the moisture found in normal expired air was avoided) on a breath-by-breath time scale. Airflow was measured using a pneumotachometer (PTM), a device based on the Fleish pneumotagraph laminar flow element, using a unique triple screen arrangement to convert the flow of air into a proportional differential pressure. The measurable range for peak flow was 0-800 litres per minute producing a maximum back pressure of 7cm H_2O. The mean back pressure during low level exercise was in the region of 1 cm H_2O (manufacturer's specifications). Prior to testing the PTM was calibrated by hand, against a precision 3 L gas syringe (Hans Rudolph Inc., Kansas, USA) calibration was achieved when air was passed across the PTM at a rate of 3 L per second to a volume of 30 L. During tests, expired air was analysed for the concentrations of O_2 and CO_2 by sampling through a paramagnetic transducer and an infrared analyser, with the O_2 and CO_2 analysers calibrated before each test using gases of known concentrations (British Oxygen Company, London, UK). Throughout each test subjects breathed through a face mask with a 2-way valve (Hans Rudolph Inc., Kansas, USA) connected to low-resistance wide bore tubing (Hans Rudolph Inc., Kansas, USA) attached to the gas analysis system. The following regression equations were calculated from the data collected during the study completed by De Montford University, Bedford, UK (1994). For the regression of Douglas bag $\dot{V}O_2$ (L·min^{-1}) on Covox $\dot{V}O_2$ (L·min^{-1}) and Douglas bag $\dot{V}CO_2$ (L·min^{-1}) on Covox $\dot{V}CO_2$ (L·min^{-1}) based on a linear model: Douglas bag $\dot{V}O_2$ (L·min^{-1}) = 0.91·(covox $\dot{V}O_2$ (L·min^{-1})) + 0.0057, (r^2 = 0.995, SEE = 0.09 at 68%CI). Douglas bag $\dot{V}CO_2$ (L·min^{-1}) = 0.9281· (covox $\dot{V}CO_2$ (L·min^{-1})) + 0.0152, (r^2 = 0.998, SEE = 0.07 at 68%CI).

During tests gear ratio and pedal cadence (rev·min^{-1}) were self selected. Peak pedal cadence (rev·min^{-1}) was calculated as the highest average cadence recorded during any 60-s period of the test. Peak heart rate (HR$_{peak}$) was recorded at 1-s intervals (Polar Kempele, Finland) and defined as the highest HR (b·min^{-1}) obtained during the test. At 5 min post completion of the tests a sample of fingertip capillary whole blood was taken for the determination of blood lactate concentration (Biosen 5030L, EKF Industrie, Electronik GmbH, Barleben, Germany). For blood sampling using the Biosen 5030L, (see Davison et al., 2000) the fingertip was wiped with a medi-swab and the first drop of blood was discarded. Free-flow

whole blood was collected in a 20 microlitre glass capillary tube and immediately mixed with lysing stabilising agent in a safe-lock vial. Before analysis, the analyser was calibrated with standard 12.0 mmol·L^{-1} solution. Each sample was collected in a 20 microlitre capillary tube and immediately mixed with a stabilising agent in a safe-lock vial. This involved shaking the sealed vial for approximately 15-s. Samples were analysed within 10 minutes from completion of the test. Each sample was analysed using an electrochemical methodology during this process some of the substrate diffused through a membrane; contacted an immobilised oxidase enzyme, and was rapidly oxidised producing hydrogen peroxide (H_2O_2). The hydrogen peroxide was oxidised at a platinum anode, producing electrons. A dynamic equilibrium was achieved when the rate of H_2O_2 production and the rate at which H_2O_2 left the immobilised enzyme layer were constant (as indicated by a steady state response). The electron flow was linearly proportional to the steady state H_2O_2 concentration and therefore the concentration of the lactate was recorded. Each blood sample was assayed (in duplicate) for the concentration of BLa (mmol·L^{-1}) and the average value of the duplicate samples was used in all subsequent analyses.

7.4.3 PART 2

Nine subjects (characteristics shown in Table 14) completed two indoor 16.1-km cycling time trials (LTT) as previously described separated by 49 ± 44 days (mean ± SD).

7.4.4 STATISTICAL ANALYSES

All data used in the subsequent analyses were ln transformed in order to remove heteroscedasticity. Measures of within-subject reproducibility (mean coefficient of variation, CV) were derived by two-way analysis of variance (ANOVA) as described by Schabort *et al.* (1998a). The 95% confidence intervals (95%CI) were calculated for CV using the methods of Tate and Klett (1959). Ratio limits of agreement and 95%CI were calculated using the methods of Atkinson and Nevill (1998) and Bland (1996) respectively. Relationships between variables were assessed using Pearson Product Moment Correlation Coefficients. Statistical significance ($P<0.05$) for comparison between repeated tests was assessed with a paired Student's t-test and ANOVA with post hoc analyses performed using Tukey HSD tests. Statistical analyses were completed using Microsoft Excel (Bellevue, WA) and Minitab (State College, PA). Values in the text are mean ± SD unless otherwise stated.

7.5 RESULTS

7.5.1 PART 1

Table 15 shows the mean ± SD values for peak cardio-respiratory variables recorded during the three PAC tests. The differences between mean values for peak $\dot{V}O_2$, $\dot{V}CO_2$, RER, f, HR, BLa and pedal cadence achieved during each trial were not statistically significant (P>0.05). The differences between mean $\dot{V}_E$ achieved during each trial were statistically significant (P<0.05). Further analysis of the data showed that mean $\dot{V}_E$ was higher (P<0.05) for trial 2 and 3 when compared with trial 1, but there was no difference between trial 2 and 3 (P = 0.4). A significant relationship was found between the change in ln $\dot{V}_{Epeak}$ and change in ln $\dot{V}CO_{2peak}$ between tests 1 and 2 (r = 0.78, 95%CI = 0.24 to 0.95, P < 0.05) but not for tests 2 and 3 (r = 0.40, 95%CI = -0.36 to 0.84, P = 0.29). However, no relationship was found between the change in ln $\dot{V}_{Epeak}$ and change in ln $\dot{V}O_{2peak}$ between tests 1 and 2 (r = 0.18, 95%CI = -0.55 to 0.75, P = 0.64) and 2 and 3 (r = 0.17, 95%CI = -0.56 to 0.75, P = 0.66).

Table 15. Mean ± SD values for peak metabolic, cardio-respiratory and performance related responses recorded as the highest average value during any 60-s period of the PAC test

	Trial 1	Trial 2	Trial 3
$\dot{V}O_2$ (L·min⁻¹)	4.99 ± 0.55	5.13 ± 0.56	5.23 ± 0.57
$\dot{V}O_2$ (mL·kg⁻¹·min⁻¹)	69.5 ± 6.0	71.8 ± 5.9	73.0 ± 6.8
$\dot{V}CO_2$ (L·min⁻¹)	5.93 ± 0.66	6.08 ± 0.61	6.13 ± 0.63
RER	1.18 ± 0.03	1.18 ± 0.03	1.21 ± 0.03
$\dot{V}_E$ (L·min⁻¹,STPD)	150 ± 17	157 ± 17 *	156 ± 15 *
f (breath·min⁻¹)	60 ± 6	60 ± 6	60 ± 6
Blood lactate (mmol·L⁻¹)	11.98 ± 1.53	12.38 ± 1.14	12.62 ± 1.29
HR (b·min⁻¹)	189 ± 6	190 ± 6	189 ± 6
Pedal cadence (rev·min⁻¹)	102 ± 5	102 ± 8	101 ± 8
Time of test (s)	593 ± 40	619 ± 34	597 ± 34

denotes significantly higher than trial 1 (P<0.05)

Table 16. Mean CV% for each individual's peak metabolic, cardio-respiratory and performance related responses recorded as the highest average value during any 60-s period during three PAC tests

	CV (95%CI)		
	Mean	*Trials 1-2*	*Trials 2-3*
$\dot{V}O_2$ (L·min^{-1})	3.8 (2.6-5.4)	4.5 (3.0-6.4)	1.6 (1.1-2.2)
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	4.2 (2.8-6.0)	4.7 (3.2-6.7)	2.1 (1.5-3.1)
$\dot{V}CO_2$ (L·min^{-1})	3.1 (2.1-4.4)	3.5 (2.4-5.0)	2.1 (1.4-3.0)
RER	1.3 (0.9-1.9)	1.4 (1.0-2.0)	1.3 (0.9-1.8)
$\dot{V}_E$ (L·min^{-1}, STPD)	3.5 (2.4-5.1)	3.8 (2.6-5.4)	2.2 (1.5-3.2)
f (breath·min^{-1})	4.8 (3.3-6.9)	4.7 (3.2-6.7)	2.7 (1.8-3.9)
Blood lactate (mmol·L^{-1})	6.7 (4.6-9.6)	5.4 (3.7-7.7)	5.9 (4.0-8.5)
HR (b·min^{-1})	1.0 (0.7-1.5)	0.9 (0.6-1.2)	1.1 (0.8-1.6)
Pedal cadence (rev·min^{-1})	4.5 (3.1-6.5)	4.5 (3.1-6.5)	2.5 (1.7-3.6)
Time of test (s)	3.5 (2.4-5.0)	2.5 (1.7-6.5)	3.5 (2.4-5.0)

Table 17. Ratio limits of agreement for peak metabolic, cardio-respiratory and performance related responses recorded as the highest average value during any 60-s period during three PAC tests

	Limits of agreement - bias (95%CI) ×/+ random error		
	Trials 1-2	*Trials 2-3*	*mean random error*
$\dot{V}O_2$ (L·min^{-1})	1.03 (0.98-1.08) ×/÷ 1.13	1.02 (1.00-1.04) ×/+ 1.04	×/+ 1.08
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	1.03 (0.98-1.09) ×/÷ 1.13	1.02 (0.99-1.04) ×/+ 1.06	×/+ 1.08
$\dot{V}CO_2$ (L·min^{-1})	1.03 (0.99-1.06) ×/÷ 1.10	1.01 (0.99-1.03) ×/+ 1.06	×/+ 1.06
RER	1.00 (0.99-1.02) ×/+ 1.04	1.02 (1.01-1.03) ×/+ 1.04	×/+ 1.03
$\dot{V}_E$ (L·min^{-1}, STPD)	1.04 (1.00-1.09) ×/÷ 1.11	0.99 (0.97-1.02) ×/+ 1.06	×/+ 1.07
f (breath·min^{-1})	1.00 (0.96-1.06) ×/+ 1.13	1.00 (0.97-1.03) ×/+ 1.08	×/+ 1.10
Blood lactate (mmol·L^{-1})	1.04 (0.98-1.10) ×/÷ 1.15	1.02 (0.96-1.08) ×/+ 1.17	×/+ 1.14
HR (b·min^{-1})	1.00 (0.99-1.01) ×/+ 1.02	1.00 (0.99-1.01) ×/+ 1.03	×+ 1.02
Pedal cadence (rev·min^{-1})	1.01 (0.96-1.06) ×/+ 1.13	0.99 (0.96-1.02) ×/+ 1.07	×/+ 1.09
Time of test (s)	1.04 (1.02-1.07) ×/+1.07	0.96 (0.93-1.00) ×/+ 1.14	×/+ 1.07

7.5.2 PART 2

The mean ± SD of each individual's average power (King$_{wLTT}$) and average heart rate (HR$_{LTT}$) recorded during the two LTT tests were 316 ± 37 vs 318 ± 37 (W) and 171 ± 14 vs 171 ± 13 b·min^{-1} for time trials 1 and 2, respectively. There was no difference (P>0.05) in average power and heart rate recorded across trials. Table 18 shows the calculated CV, and limits of agreement for average power and heart rate during the two LTT tests.

Table 18. Coefficient of variation (CV%) and ratio limits of agreement (LoA) for each individual's average power output (King$_{wLTT}$) and heart rate (HR$_{LTT}$) recorded during two LTT tests

	King$_{wLTT}$	HR$_{LTT}$
CV (95%CI)	2.8 (1.6-4.9)	1.3 (0.7-2.2)
LoA(95%CI)	1.01 (0.98-1.04) ×/÷ 1.08	1.00 (0.99-1.02) ×/÷ 1.04

7.6 DISCUSSION

Analysis of the data from the present study showed that the CV value calculated for average power achieved during an indoor 16.1-km time trial was similar to CV values calculated by Hopkins *et al.* (1999) for average power achieved during laboratory based simulated time trial rides. Ratio limits of agreement calculated for average power achieved during the time trial rides revealed that a cyclist who maintained a power output of about 350 W in a preliminary trial would need to achieve an average power of 322 or 378 W to be outside the range of biological and technical variability associated with this type of test. Ratio limits of agreement estimated for the 16.1-km time trial were similar to values reported by Atkinson *et al.* (1998) for an indoor 16.1-km cycling time trial performed on a Cybex™ cycle ergometer, however in the study by Atkinson *et al.* (1998) the time trial rides were separated by one week and in the present study the average time between tests was about 50 days, therefore it is reasonable to assume that performance power achieved during this type of test can remain relatively stable over a reasonable length of time. This finding has important implications when a study is designed to evaluate the effect of a training program on endurance performance. Measurement error for the 16.1-km time trial was higher than the error associated with the determination of SRM$_{Wpeak}$ reported in study one of the present thesis (2.8 vs 1.3%, respectively) and confirmed the findings of Jensen

and Johansen (1998) who found that peak power achieved during a cycling exercise test was a more reproducible measure of cycling performance when compared with average power recorded during repeated cycling time trial tests.

One of the major findings of the present study is that peak heart rate recorded during a Kingcycle PAC test and average heart rate recorded during repeated time trial tests is highly reproducible. Based on ratio limits of agreement it is possible to calculate that HR_{peak} and average heart rate recorded during the time trials varied by about 4% which would represent a change of 8 b·min^{-1} for an individual with a HR_{peak} of 190 b·min^{-1} and 7 b·min^{-1} for an individual with a HR_{LTT} of 170 b·min^{-1}. The mean CV calculated for HR_{peak} during PAC and average heart rate during the 16.1-km time trial was 1.0 and 1.3%, respectively. These values are similar to the values reported by Jensen and Johansen (1998) however, the CV for peak heart rate calculated in this study was lower than values reported by Jones and Kane (1991) and Harrison et al. (1980). During each of the three PAC tests peak heart rate achieved by each subject was within the $\dot{V}O_{2max}$ criterion measure of 5 b·min^{-1} of their age predicted maximum heart rate (Londeree and Moeschberger, 1982).

In part 1 of the study there was an increase in reproducibility for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ recorded between trials 2 and 3 when compared with trial one and the CV calculated for $\dot{V}O_2$ for the three trials (~ 4%) was similar to the value reported by Howley et al. (1995). Furthermore, based on the findings of Katch et al. (1982) it is reasonable to suggest that 10% of this variation could be attributed to the technical error of the Covox gas analyser. The CV calculated for $\dot{V}O_2$ recorded for trials 2 and 3 was considerably lower than values reported by Jones and Kane (1979), and Katch et al. (1982). It is reasonable to assume that the Covox gas analyser used in the present study provided a reliable measurement of pulmonary ventilation and O_2 and CO_2 content in the expired air, however, values for $\dot{V}O_2$ and $\dot{V}CO_2$ were relatively high for the fitness level of the cyclists tested. This finding is in agreement with the work completed at De Montfort University (1994) which reported that values for $\dot{V}O_2$ and $\dot{V}CO_2$ recorded using the Covox system were consistently higher than values recorded using a Servomex gas analyser. It is worth noting that during the three repeated PAC tests, the testing protocol used in the first test was repeated for subsequent tests and for each test the Covox gas analysis system measured expired air and pulmonary

ventilation at 1 min intervals. Unfortunately, values for $\dot{V}O_{2peak}$ could not be matched with peak power unless the minute of peak power coincided with the final 1 min gas sample.

During the three PAC tests of the present study a plateau in oxygen consumption (Taylor *et al.*, 1955) was recorded during five of the 27 tests completed. Therefore the criterion measure of a plateau in oxygen consumption for the achievement of $\dot{V}O_{2max}$ could not be applied to this data. Several studies have also reported a very low incidence of a $\dot{V}O_2$-plateau during maximal exercise tests (see Howley *et al.*, 1995). One possible explanation for the finding of the present study is that $\dot{V}O_2$ was recorded at 1 min intervals during the test (as explained above) and W_{peak} achieved during each trial did not coincide with $\dot{V}O_{2peak}$. Consequently $\dot{V}O_{2peak}$ was not always recorded during the final/peak minute of the test. Although Noakes (1998) has suggested that a $\dot{V}O_2$-plateau is an artefact of the testing protocol, it is reasonable to postulate that a plateau may have been recorded if a 'breath by breath' gas analysis system had been used to measure pulmonary/respiratory responses. Further investigation is warranted concerning the effect of ramp rate on $\dot{V}O_2$ kinetics and the attainment of a $\dot{V}O_2$-plateau. The findings of the present study suggest that maximum oxygen consumption ($\dot{V}O_{2max}$) was not achieved during the Kingcycle PAC test, therefore the highest $\dot{V}O_2$ recorded during this type of test should be described as a $\dot{V}O_{2peak}$. Data from the present study showed that values for $\dot{V}CO_2$ recorded during the three PAC tests were highly reproducible and therefore calculations of RER were also consistent. Mean values for RER achieved during each PAC test were notably higher than the 1.15 used as a criterion measure for the achievement of maximum oxygen uptake (Howley *et al.*, 1995), however on two occasions, one subject did not achieve an RER in excess of 1.15.

Mean value for CV calculated for pulmonary ventilation was less than the CV values reported by Jensen and Johansen (1998) and Jones and Kane (1979). Data from the present study provided strong evidence to suggest that the reproducibility of $\dot{V}_{Epeak}$ is acceptable when compared with other measures of peak aerobic capacity such as $\dot{V}O_{2peak}$ and that the Covox gas analyser (which measures ventilation on inhalation) provides a reliable method of recording peak minute ventilation during a Kingcycle PAC test. It is important to note that for the three PAC tests, $\dot{V}_{Epeak}$ was significantly higher (P<0.05) during test 2 and 3

when compared with test 1, however there was no difference in $\dot{V}O_{2peak}$ recorded across tests. The increase in $\dot{V}_{Epeak}$ for trials 2 and 3 occurred without a concomitant change in breathing frequency, therefore subjects reached a higher state of exercise hyperpnea by increasing tidal volume. Notably a plateau in $\dot{V}_E$ was not recorded during the PAC tests and therefore subjects did not attain a maximum ventilation. This is in agreement with the recent work of Lucia *et al.* (1998) who assessed the ventilation of professional cyclists and found that tidal volume and breathing frequency did not plateau during maximal exercise. In contrast to this Lucia and co-workers (1998) recorded a plateau in tidal volume but not breathing frequency. The authors noted that a *breathing reserve* existed in both groups as values for maximal minute ventilation were 86.9% and 90% of maximum voluntary ventilation (MVV) in elite and professional cyclists, respectively. In the present study peak values for $\dot{V}CO_2$ and blood lactate were higher during test 2 and 3 when compared with test 1, however, these differences were not significant ($P>0.05$). It is worth noting that a significant relationship was found between the change in $\dot{V}_{Epeak}$ and the change in $\dot{V}CO_{2peak}$ recorded between test 1 and 2. However there was no relationship found between these parameters for tests 2 and 3. Notably regression analysis revealed that there was no relationship between $\dot{V}_{Epeak}$ and $\dot{V}O_{2peak}$ between tests 1 and 2 or 2 and 3.

During the three PAC tests, blood lactate concentration recorded post exercise for each subject was considerably higher than the criterion value of 8 $mmol\cdot L^{-1}$ used to establish the attainment of $\dot{V}O_{2max}$ (Howley *et al.*, 1995). Few studies have assessed the reproducibility of blood lactate recorded post maximal cycling exercise and data from the present study suggested that blood lactate recorded post PAC did not provide a reproducible assessment of exercise performance capacity. It is important to note that Lucia *et al.* (1998) found that peak blood lactate concentration was significantly lower in professional cyclists who achieved significantly higher values for maximal aerobic power when compared with elite amateur cyclists. This data indicated that the relationship between blood lactate and W_{max} did not follow a positive trend. Investigators should be aware of this discrepancy when using blood lactate response to assess endurance performance ability. For part two of the present study, ratio limits of agreement estimated for blood lactate were ×/+ 14%. For instance, an initial post PAC blood lactate concentration of 14.0 $mmol\cdot L^{-1}$ would be between 12.3 and 16.0 $mmol\cdot L^{-1}$ when recorded during a one week re-test. This range of

variability is unacceptable and relatively high when compared with other peak values recorded during the PAC tests.

During the three PAC tests pedal cadence was self selected and analysis of the data revealed that peak pedal cadence recorded for each subject during the peak tests was relatively consistent. It is worth noting that the self selected pedal cadence recorded in the present study was notably higher than pedal cadences pre-selected by Davis *et al.* (1982), Hansen *et al.* (1988), Yoshida (1984) and Zhang *et al.* (1991) and the preferred peak pedal cadence recorded for a maximal aerobic capacity test by Marsh and Martin (1993). In the study by Marsh and Martin (1993) tests were performed using a different cycle simulator, therefore preferred pedal cadence may have been influenced by the inertial characteristics of the electromagnetic braked flywheel of the Velodyne™ simulator when compared with the air-braked flywheel of the Kingcycle. In contrast to the ramped protocol of the Kingcycle PAC test, Marsh and Martin (1993) used a continuous incremental exercise test to assess maximal aerobic capacity. There is very little information available concerning the effect of cycle ergometer design and testing protocol on peak pedal cadence recorded during PP/PAC tests. In the present study subjects were highly trained cyclists with extensive experience of testing procedures and it is reasonable to suggest that each subject self-selected their own 'optimum' pedal cadence during each PAC test.

7.7 SUMMARY

Within subject variation for peak $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ recorded during the Kingcycle PAC test were relatively low when compared with previous studies. The coefficient of variation calculated for peak blood lactate concentration recorded five minutes after completion of the PAC tests was relatively high and unacceptable for the assessment of cycling endurance capacity. Reproducibility of average power and heart rate recorded during an indoor 16.1-km cycling time trial was acceptable and similar to values reported for equivalent assessments of cycling endurance performance. The physiological tests and testing equipment used in the present study provided a reliable assessment of selected peak physiological variables and cycling endurance performance.

CHAPTER 8

8 AGE-RELATED CHANGES IN PEAK PHYSIOLOGICAL VARIABLES AND PERFORMANCE RELATED RESPONSES RECORDED DURING CYCLING TESTS

8.1 INTRODUCTION

Laboratory based physiological tests are frequently used to assess the exercise performance ability of senior cyclists, however very few studies have used these tests to assess veteran athletes. New information concerning the responses of senior and veteran competitors would be particularly useful when data is used to evaluate, monitor and prescribe training and competitive exercise intensity within a sport science support programme.

8.2 AIM OF STUDY 3

To gain a clearer understanding of age-related changes in physiological variables recorded during laboratory based cycling tests.

8.3 OBJECTIVES

8.3.1 PART 1

To investigate the relationships between $King_{Wpeak}$, HR_{peak} and age during a Kingcycle peak power test.

8.3.2 PART 2

To investigate the relationships between age and selected physiological variables assessed during a Kingcycle PAC test.

8.3.3 PART 3

To investigate the effect of age on threshold exercise intensity determined during a ramped exercise test (LT_{ramp}).

8.3.4 PART 4

To investigate age-related changes in performance related responses recorded during a laboratory based 16.1-km cycling time trial (LTT).

8.4 METHODS

8.4.1 SUBJECTS

All of the subjects who participated in the study were endurance trained male cyclists with extensive experience of competitive cycling. During all tests power was recorded using the Kingcycle ergometer with version 5.5 computer software. Tests were conducted at the same time of day and laboratory conditions were maintained during testing (ambient temperature 18-22°C relative humidity 45-55%).

Table 19. Mean ± SD values for physical characteristics and peak performance related responses recorded during a Kingcycle PP test (n = 114)

	Mean (± SD)	Range
Age (yr)	38 ± 15	15-73
Height (m)	1.78 ± 0.07	1.57-1.93
Body mass (kg)	73.5 ± 7.2	55.8-90.0
King$_{Wpeak}$ (W)	382 ± 64	226-550
HR$_{peak}$ (b·min^{-1})	185 ± 13	150-214

8.4.2 PART 1

One hundred and fourteen subjects (subject characteristics are shown in Table 19) completed a Kingcycle PP test as previously outlined in 6.4.1.3.

8.4.3 PART 2

Twenty nine subjects (characteristics shown in Table 21) completed a Kingcycle PAC test as previously outlined in 7.4.2. However before the PAC test, skin fold thickness at four sites - biceps, triceps, sub-scapular and supra-iliac were measured to the nearest 1 mm using a pair of Harpenden callipers (John Bull, England). All measurements were taken from the non-dominant side of the body while the subject stood relaxed in the reference

position. Each site was measured three times and the average value was calculated. The sum of the average value obtained for each site was used to predict body density when applied to the age-appropriate regression equation of Durnin and Womersley (1974). The Siri (1956) equation was then used to give a predicted percentage body fat.

8.4.4 PART 3

Thirty eight subjects (characteristics shown in Table 26) completed a ramped threshold test (LT_{ramp}). Following a five min warm-up maintaining a power output equivalent to 50% of W_{peak} (which had been previously determined from an habituation PP test) subjects completed a continuous ramped exercise protocol (LT_{ramp}, as described by Davison et al., 1997). Starting power was 50% W_{peak} and ramp rate was set at 6 $W \cdot min^{-1}$. For each test subjects selected their own pedal cadence ($rev \cdot min^{-1}$), changed gear ad libitum and the test was completed at volitional exhaustion. Fingertip blood samples (YSI, Yellow Springs, USA) were taken at 2.5 min intervals and power was recorded continuously. Heart rate was determined every 5-s, (Polar, Kempele, Finland) however average values for heart rate and power were calculated for each minute of the test. Threshold values for power and heart rate were determined at TLac (Farrell et al., 1979) and OBLA (Jacobs et al., 1981). For the determination of TLac (the power preceding a sudden and sustained increase in BLa) data for blood lactate concentration and power output were plotted, coded and presented blindly to two investigators for independent visual inspection. The mean selection of the two reviewers was subsequently used for analysis and reliability of inter-investigator TLac assessment was low (CV% = 1.8%; 95%CI = 1.5-2.4%). The power at OBLA was determined by interpolation.

8.4.5 PART 4

Forty five subjects (characteristics shown in Table 28) completed a laboratory based 16.1-km cycling time trial (LTT) as previously outlined.

8.4.6 STATISTICAL ANALYSES

Linear and quadratic regression analyses were performed to determine the effects of age on:- $King_{Wpeak}$ and HR_{peak} recorded during PP; selected metabolic and cardio-respiratory variables recorded during PAC; $King_W@TLac$, $King_W@OBLA$, HR@TLac, HR@OBLA during the LT_{ramp} test and average power and heart rate recorded during LTT. In order to

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control for heteroscedasticity selected values were ln transformed and the relationship between variables was assessed using a ln-linear model. In basic terms, the use of logarithmic transformation controls for heteroscedasticity in the data and provides a normally distributed error term. Consequently the standard error of estimate (SEE) is calculated as a percentage of the predicted value. In order to assess the age-related decline in $\dot{V}O_{2peak}$ independent of changes in body size and body composition, recorded values for body mass were normalised using the theoretical single-exponent parameter $k = 0.67$ (Neville and Holder, 1995). Statistical significance was set at P<0.05. Statistical analyses were completed using Microsoft Excel (Bellevue, WA) and Minitab (State College, PA). Values in the text are mean ± SD unless otherwise stated.

8.5 RESULTS

8.5.1 PART 1

8.5.1.1 PEAK POWER (KING$_{WPEAK}$)

The mean value for King$_{Wpeak}$ recorded in a heterogeneous group of male competitive cyclists aged 15-73 yr was 382 (W) with a range from 226 to 550 (W) (see Table 19). Individual King$_{Wpeak}$ versus age for the entire subject group (n = 114) is depicted in Figure 9. A moderate correlation was found between age and King$_{Wpeak}$ for both linear (r = 0.55, 95%CI = 0.41-0.67, P<0.001) and 4th order polynomial models, (r = 0.60, 95%CI = 0.47-0.71, P<0.001). The absolute and percentage (relative) decline in King$_{Wpeak}$ with age from the linear model was 2.43 W·yr^{-1} (~ 0.64 %·yr^{-1}) respectively and the regression of King$_{Wpeak}$ on age for n = 114 in the linear model was; King$_{Wpeak}$ (W) = 474 -2.43·age(yr), (r^2 = 0.30, SEE; 54 W 95%CI = 48-62 W). The correlation of King$_{Wpeak}$ on age using a ln-linear model was r = 0.52, (95%CI = 0.35-0.63, P<0.01). The equation for the regression of King$_{Wpeak}$ on age for the ln-linear model was; King$_{Wpeak}$ (W) = 824·[age(yr)$^{-0.22}$], (r^2 = 0.27, SEE; 16%, 95%CI = 14-18%).

In order to remove the confounding effects of i) an increase in King$_{Wpeak}$ with age in subjects aged between 15 and 25 yr due to maturation and ii) the relatively low number of subjects aged above 65 yr who participated in the study, further analysis of data for subjects aged between 25 and 65 yr was completed. The correlation between age and

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King$_{Wpeak}$ for a linear model was r = 0.56, (95%CI = 0.40-0.69, P<0.01) and the absolute and percentage decline in King$_{Wpeak}$ with age was calculated as 3.05 W·yr^{-1} (~ 0.81 %·yr^{-1}). The regression of King$_{Wpeak}$ on age for n = 87 in the linear model was; King$_{Wpeak}$ (W) = 504 - 3.05·age (yr), (r^2 = 0.31, SEE; 54 W, 95%CI = 48-62 W). The absolute and percentage decline in King$_{Wpeak}$ when expressed as W·kg^{-1} and W·kg$^{-0.67}$ was 0.033 W·kg^{-1}·yr^{-1} (~ 0.84 %·yr^{-1}) and 0.14 W·kg$^{-0.67}$·yr^{-1} (~ 0.87 %·yr^{-1}) respectively.
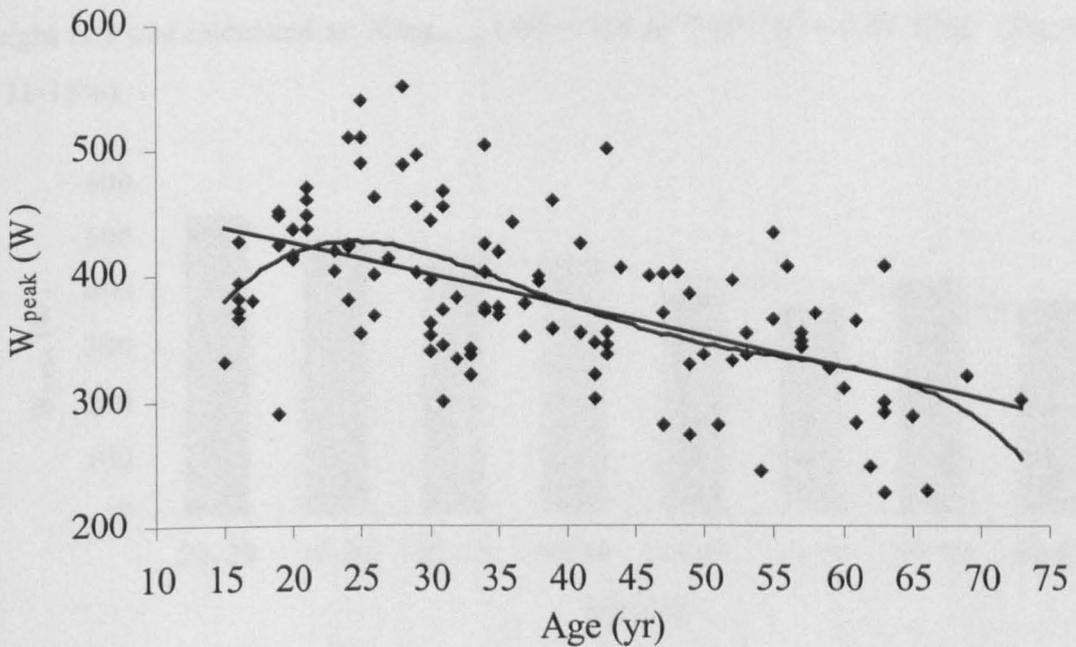
Figure 9. The regression of King$_{Wpeak}$ (W) on age for 114 men aged 15-73. Data fitted with a linear (r = 0.55, P<0.001) and 4th order polynomial model (r = 0.60, P<0.001)

The percentage rate of decline in King$_{Wpeak}$ (W·yr^{-1}) was similar (0.81 vs 0.84 %·yr^{-1}) to the percentage rate of decline in King$_{Wpeak}$ when expressed relative to body mass (W·kg^{-1}·yr^{-1}). However the relative rate of decline in King$_{Wpeak}$ when expressed relative to the mass exponent of k = 0.67 (W·kg$^{-0.67}$·yr^{-1}) was higher (0.87 vs 0.81 %·yr^{-1}) than the decline in King$_{Wpeak}$ (W·yr^{-1}).

Multiple regression of age (yr), height (m) and body mass (kg) on King$_{Wpeak}$ (W) showed that body mass did not contribute (P = 0.97) to the prediction of King$_{Wpeak}$ (W). Standard multiple regression was applied to King$_{Wpeak}$, age and height values of the 87 subjects aged 25-65. The correlation of age and height for the linear model was r = 0.69 (95%CI = 0.56-

0.79, P<0.01). The regression of age and height was calculated as; $King_{Wpeak}$ (W) = [418·height (m) - 2.29·age (yr)] - 270, (r^2 = 0.48 SEE; 47 W, 95%CI = 42-54 W).

Multivariate scaling (MAS) was used in order to predict $King_{Wpeak}$ from age and height. Multiple regression analysis was applied to ln $King_{Wpeak}$, ln height and ln age values of 87 subjects aged 25-65. The correlation of age and height on $King_{Wpeak}$ for the ln-linear model was r = 0.70, (95%CI = 0.57-0.79, P<0.01). The equation for the regression of age (A) and height (H) was calculated as; $King_{Wpeak}$ (W) = $314·A^{-0.26}·H^{1.96}$ (r^2 = 0.49, SEE; 13%, 95%CI = 11-15%).

Figure 10. The regression of $King_{Wpeak}$ (W) on age for mean of top 2 performers in each age category (n = 16). Data fitted with a linear model

The data set was then split into 5-yr age increments beginning at age 25-29 and ending with age 60-64. The regression of $King_{Wpeak}$ (W) on age for the mean $King_{Wpeak}$ of the top 2 performers in each age category was completed. A strong correlation was found between $King_{Wpeak}$ and age (r = 0.88, 95%CI = 0.68-0.96, P<0.01) The absolute and percentage decline in $King_{Wpeak}$ from the linear model was 4.10 $W·yr^{-1}$ (~ 0.94 %·yr^{-1}) and the regression equation of $King_{Wpeak}$ on age (n = 16) was calculated as; $King_{Wpeak}$ (W) = 532 - 4.10·age (yr), (r^2 = 0.78, SEE; 34 W, 95%CI = 26-51 W). The correlation of $King_{Wpeak}$ on age using a ln-linear model was r = 0.77, (95%CI = 0.67-0.84, P<0.01). The equation for the regression of $King_{Wpeak}$ on age for the ln-linear model was: $King_{Wpeak}$ (W) = 1186·[age(yr)$^{-0.28}$], (r^2 = 0.60, SEE; 11%, 95%CI = 8-16%). The absolute and percentage

decline in $King_{Wpeak}$ when expressed as $W \cdot kg^{-1}$ and $W \cdot kg^{-0.67}$ was 0.055 $W \cdot kg^{-1} \cdot yr^{-1}$ (~ 0.95 $\% \cdot yr^{-1}$) and 0.22 $W \cdot kg^{-0.67} \cdot yr^{-1}$ (~ 0.92 $\% \cdot yr^{-1}$) respectively.

Table 20. Absolute and relative rates of decline for $King_{Wpeak}$ with age

Number of subjects	Absolute rate of decline ($W \cdot yr^{-1}$)	Percentage (relative) rate of decline ($\% \cdot yr^{-1}$)
114 (group aged 15-73 yr)	2.43	0.64
87 (group aged 25-65 yr)	3.05	0.81
16 (best performers for 5-yr increments)	4.10	0.93

8.5.1.2 PEAK HEART RATE (HR_{PEAK})

The mean value for HR_{peak} recorded in a heterogeneous group of male competitive cyclists aged 15-73 yr was 185 ($b \cdot min^{-1}$) with a range from 150 to 214 ($b \cdot min^{-1}$) (see Table 19). Figure 11 depicts the individual HR_{peak} versus age for the subject group (n = 114). Strong correlates were found between age and HR_{peak} for both linear (r = 0.75, 95%CI = 0.66-0.82, P<0.01) and 2nd order polynomial models (r = 0.76, 95%CI = 0.67-0.83, P<0.01).

220
210
200
190
180
170
160
150
140
HR (b.min-1)
10 15 20 25 30 35 40 45 50 55 60 65 70 75 80
Age (years)
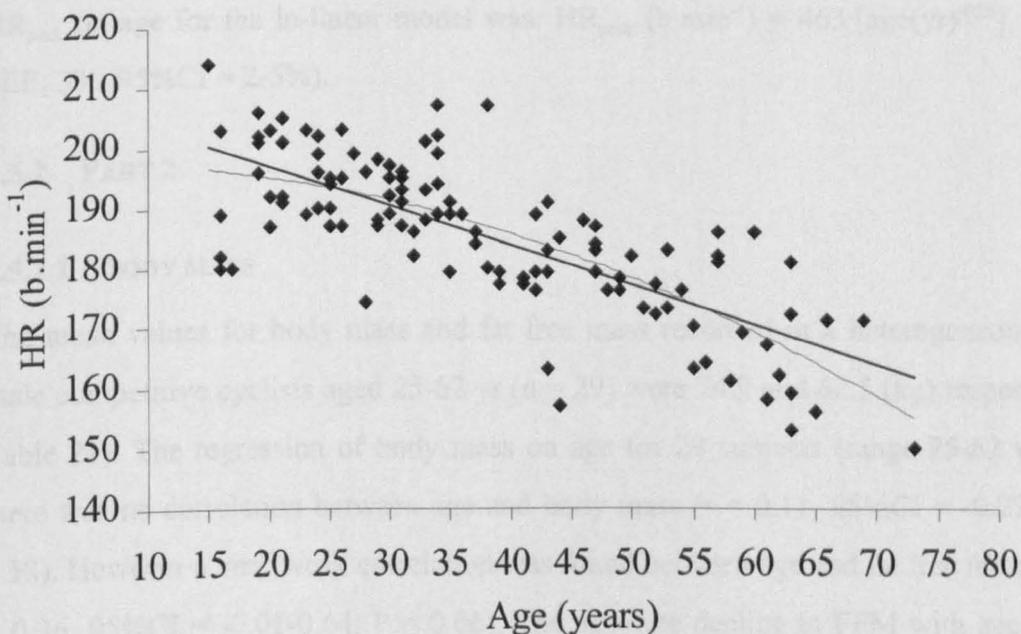
Figure 11. The regression of HR_{peak} ($b \cdot min^{-1}$) on age for 114 men aged 15-73. Data fitted with a linear and 2nd order polynomial model

No significant difference was found between linear and quadratic models (F = 1.04). The absolute and percentage decline in HR_{peak} with age was calculated as 0.66 b·min^{-1}·yr^{-1} (~ 0.36 %·yr^{-1}). The regression of HR_{peak} on age for n = 114 in the linear model was; HR_{peak} (b·min^{-1}) = 210-0.66·age (yr), (r^2 = 0.56, SEE; 9 b·min^{-1}, 95%CI = 8-10 b·min^{-1}). The correlation of HR_{peak} on age using a ln-linear model was r = 0.69, (95%CI = 0.58-0.78, P<0.01). The equation for the regression of HR_{peak} on age for the ln-linear model was: HR_{peak} (b·min^{-1}) = 282·[age (yr)$^{-0.12}$], (r^2 = 0.47, SEE; 5%, 95%CI = 4-6 %).

The data set was split into 5 yr age increments beginning at age 25-29 and ending with age 60-64. The regression of HR_{peak} (b·min^{-1}) on age for the mean HR_{peak} of the top 2 performers in each age category was completed. A strong correlation was found between HR_{peak} and age (r = 0.94, 95%CI = 0.83-0.98, P<0.001) The absolute and percentage decline in HR_{peak} from the linear model was 1.06 b·min^{-1}·yr^{-1} (~ 0.60 %·yr^{-1}) and the regression equation of HR_{peak} on age (n = 16) was calculated as; HR_{peak} (b·min^{-1}) = 225 -1.06·age (yr), (r^2 = 0.88, SEE; 5 b·min^{-1}, 95%CI = 4-8 b·min^{-1}). The correlation of HR_{peak} on age using a ln-linear model was r = 0.92, (95%CI = 0.78-0.97, P<0.001). The equation for the regression of HR_{peak} on age for the ln-linear model was: HR_{peak} (b·min^{-1}) = 463·[age(yr)$^{-0.26}$], (r^2 = 0.85, SEE; 3%, 95%CI = 2-5%).

8.5.2 PART 2

8.5.2.1 BODY MASS

The mean values for body mass and fat free mass recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) were 74.9 and 62.5 (kg) respectively (see Table 21). The regression of body mass on age for 29 subjects (range 25-62 yr) showed there was no correlation between age and body mass (r = 0.11, 95%CI = -0.27-0.46, P = 0.58). However a very weak correlation was found between age and fat free mass (FFM), (r = 0.36, 95%CI = -0.01-0.64, P = 0.06). The absolute decline in FFM with age calculated from a linear model was 0.15 kgFFM·yr^{-1} or 1.5 kgFFM·decade^{-1}. The regression of FFM on age for n = 29 using a linear model was; FFM (kg) = 68.8-0.15·age (yr), (r^2 = 0.13, SEE; 4.8 kg, 95%CI = 3.8-6.5 kg). The correlation of FFM on age using a ln-linear model was r = 0.39, (95%CI = 0.03-0.66, P = 0.035). The equation for the regression of FFM on age for

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the ln-linear model was: FFM (kg) = $92.1 \cdot [\text{age(yr)}^{-0.11}]$, ($r^2 = 0.15$, SEE; 8%, 95%CI = 6-11%).

Table 21. Mean $\pm$ SD values for subjects' physical characteristics and peak physiological variables recorded during a Kingcycle PAC test (n = 29)

	Mean $\pm$ SD	Range
Age (yr)	41 $\pm$ 12	25-62
Body mass (kg)	74.9 $\pm$ 5.9	63.5-88.7
Fat free mass (kg)	62.5 $\pm$ 5.0	51.3-73.4
Height (m)	1.79 $\pm$ 0.06	1.69-1.91
HR_{peak} (b·min^{-1})	181 $\pm$ 14	154-202
$King_{Wpeak}$ (W)	404 $\pm$ 65	295-550
$\dot{V}O_{2peak}$ (L·min^{-1})	4.61 $\pm$ 0.65	3.49-5.84
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	61.9 $\pm$ 9.3	43.9-77.1
$\dot{V}O_{2peak}$ (mL·kg$^{-0.67}$·min^{-1})	256.5 $\pm$ 36.8	187.1-307.6
$\dot{V}O_{2peak}$ (mL·kgFFM^{-1}·min^{-1})	73.8 $\pm$ 8.5	56.3-88.8
$\dot{V}O_{2peak}$ (mL·kgFFM$^{-0.67}$·min^{-1})	288.6 $\pm$ 34.2	125.9-345.8
$\dot{V}CO_{2peak}$ (L·min^{-1})	5.52 $\pm$ 0.80	4.02-7.02
V_{Epeak} (L·min^{-1}, STPD)	143 $\pm$ 18	114-173
$V_{Epeak}/\dot{V}O_{2peak}$	31.5 $\pm$ 3.2	24.8-38.3
$V_{Epeak}/\dot{V}CO_{2peak}$	26.3 $\pm$ 2.4	20.6-30.3
Peak RER	1.20 $\pm$ 0.05	1.06-1.27
Peak economy [W·$\dot{V}O_2$ (L·min^{-1})]	87 $\pm$ 6	76-98
Peak breathing frequency (breath·min^{-1})	58 $\pm$ 7	47-78
Lactate at 5 min post MAX (mmol·L^{-1})	11.9 $\pm$ 2.3	5.0-15.5

8.5.2.2 HEIGHT

The mean value for height recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 1.79 (m) with a range from 1.69 to 1.91 (m) (see Table 21). A weak correlation was found between age and height (r = 0.45, 95%CI = 0.10-0.70, P<0.05).

The absolute decline in height with age was 0.22 cm·yr^{-1} or 2.2 cm·decade^{-1}. The equation for the regression of height on age for n = 29 using a linear model was: Height (cm) = 1.88-0.22·age (yr), (r^2 = 0.20, SEE; 5.3 cm, 95%CI = 4.2-7.2 cm). The equation for the regression of height on age for the ln-linear model was: Height (cm) = 216·[age(yr)$^{-0.05}$], (r^2 = 0.22, SEE; 3%, 95%CI = 2-4%).

8.5.2.3 PERCENTAGE BODY FAT (%BF)

Mean value for estimated % body fat in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 16.4% with a range from 9.7 to 24.9% (see Table 22).

Table 22. Mean ± SD values for skinfolds and estimation of % body fat (n = 29)

	Mean ± SD	Range
Biceps	4.5 ± 1.1	2.4-8.0
Triceps	8.3 ± 2.1	4.8-12.9
Subscapular	11.2 ± 2.9	6.9-20.1
Suprailiac	6.6 ± 2.3	3.7-11.6
Sum of skinfolds	30.0 ± 7.5	18.3-50.8
% body fat	16.4 ± 4.3	9.7-24.9
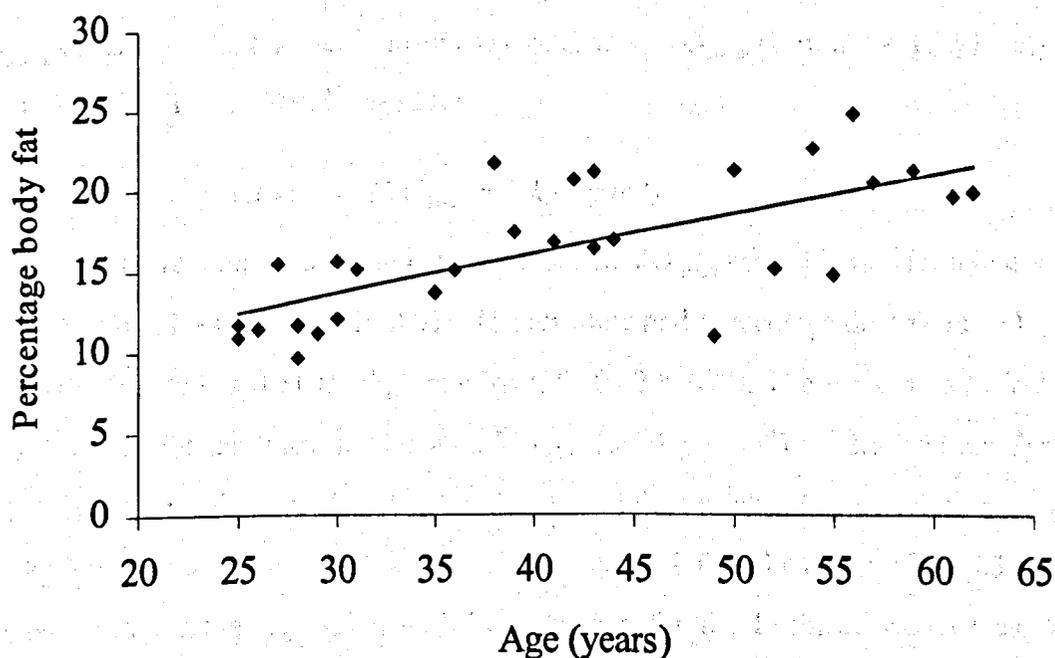
Figure 12. The regression of %BF on age for 29 men aged 25-62. Data fitted with a linear model

Individual percentage body fat (%BF) versus age is depicted in Figure 12. A moderate correlation was found between age and %BF for a linear model (r = 0.71, 95%CI = 0.46-0.85, P<0.01). The increase in %BF with age was calculated as 0.25 %·yr^{-1}. The regression of %BF on age for n = 29 using a linear model was; BF (%) = 6.2+0.25·age (yr), (r^2 = 0.50, SEE; 3%, 95%CI = 2-4%).

8.5.2.4 PEAK OXYGEN UPTAKE ($\dot{V}O_{2PEAK}$)

Mean values for $\dot{V}O_{2peak}$ (L·min^{-1}, mL·kg^{-1}·min^{-1}, mL·kgFFM^{-1}·min^{-1}, mL·kg$^{-0.67}$·min^{-1}, mL·kgFFM$^{-0.67}$·min^{-1}) recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr are shown in Table 21.

8.5.2.4.1 Peak oxygen uptake ($\dot{V}O_{2peak}$ L·min^{-1})

A moderate correlation was found between age and $\dot{V}O_{2peak}$ for a linear model (r = 0.67, 95%CI = 0.40-0.83, P<0.01). The absolute and percentage decline in $\dot{V}O_{2peak}$ with age was calculated as 0.036 L·min^{-1}·yr^{-1}, (~ 0.79 %·yr^{-1}). The regression of $\dot{V}O_{2peak}$ on age for n = 29 in a linear model was; $\dot{V}O_{2peak}$ (L·min^{-1}) = 6.12 -0.036·age (yr), (r^2 = 0.45, SEE; 0.44 L·min^{-1}, 95%CI = 0.35-0.60 L·min^{-1}). The correlation of $\dot{V}O_{2peak}$ on age using an allometric ln-linear model was r = 0.69, (95%CI = 0.43-0.84, P<0.01). The equation for the regression of $\dot{V}O_{2peak}$ on age for the allometric ln-linear model was: $\dot{V}O_{2peak}$ (L·min^{-1}) = 15.3·[age(yr)$^{-0.33}$], (r^2 = 0.48, SEE; 11%, 95%CI = 9-15%).

8.5.2.4.2 Peak oxygen uptake ($\dot{V}O_{2peak}$ mL·kg^{-1}·min^{-1})

A moderate correlation was found between age and $\dot{V}O_{2peak}$ mL·kg^{-1}·min^{-1} for a linear model (r = 0.66, 95%CI = 0.39-0.83, P<0.01). The absolute and percentage decline in $\dot{V}O_{2peak}$ with age was calculated as 0.51 mL·kg^{-1}·min^{-1}·yr^{-1}, (~ 0.82 %·yr^{-1}). The regression of $\dot{V}O_{2peak}$ on age for n = 29 for the linear model was; $\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1}) = 82.8-0.51·age (yr), (r^2 = 0.44, SEE; 7.1 mL·kg^{-1}·min^{-1}, 95%CI = 5.6-9.6 mL·kg^{-1}·min^{-1}). The correlation of $\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1}) on age using a ln-linear model was r = 0.66, (95%CI = 0.39-0.83, P<0.01). The equation for the regression of $\dot{V}O_{2peak}$ on age for the ln-linear model was: $\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1}) = 218·[age(yr)$^{-0.35}$], (r^2 = 0.43, SEE; 13%, 95%CI = 10-18%).

8.5.2.4.3 Peak oxygen uptake ($\dot{V}O_{2peak}$ $mL \cdot kg^{-0.67} \cdot min^{-1}$)

A moderate correlation was found between age and $\dot{V}O_{2peak}$ mL·kg$^{-0.67}$·min^{-1} for a linear model (r = 0.68, 95%CI = 0.42-0.84, P<0.01). The absolute and percentage decline in $\dot{V}O_{2peak}$ with age was calculated as 2.08 mL·kg$^{-0.67}$·min^{-1}, (~ 0.81 %·yr^{-1}). The regression of $\dot{V}O_{2peak}$ on age for n = 29 for the linear model was; $\dot{V}O_{2peak}$ (mL·kg$^{-0.67}$·min^{-1}) = 342.2-2.08·age (yr), (r^2 = 0.47, SEE; 27.4 mL·kg$^{-0.67}$·min^{-1}, 95%CI = 21.7-37.1 mL·kg$^{-0.67}$·min^{-1}). The correlation of $\dot{V}O_{2peak}$ mL·kg$^{-0.67}$·min^{-1}) on age using a ln-linear model was r = 0.69, (95%CI = 0.43-0.84, P<0.01). The equation for the regression of $\dot{V}O_{2peak}$ on age for the ln-linear model was: $\dot{V}O_{2peak}$ (mL·kg$^{-0.67}$·min^{-1}) = 887·[age(yr)$^{-0.34}$], (r^2 = 0.48, SEE; 12%, 95%CI = 10-16%).

8.5.2.4.4 Peak oxygen uptake ($\dot{V}O_{2peak}$ $mL \cdot kgFFM^{-1} \cdot min^{-1}$)

A moderate correlation was found between age and $\dot{V}O_{2peak}$ mL·kgFFM^{-1}·min^{-1} for a linear model (r = 0.56, 95%CI = 0.24-0.77, P<0.01). The absolute and percentage decline in $\dot{V}O_{2peak}$ with age was calculated as 0.39 mL·kgFFM^{-1}·min^{-1} (~ 0.53 %·yr^{-1}). The regression of $\dot{V}O_{2peak}$ on age for n = 29 for the linear model was; $\dot{V}O_{2peak}$ (mL·kgFFM^{-1}·min^{-1}) = 90.0-0.39·age (yr), (r^2 = 0.31, SEE; 7.2 mL·kgFFM^{-1}·min^{-1}, 95%CI = 5.7-9.7 mL·kgFFM^{-1}·min^{-1}). The correlation of $\dot{V}O_{2peak}$ (mL·kgFFM^{-1}·min^{-1}) on age using a ln-linear model was r = 0.56, (95%CI = 0.24-0.77, P<0.01). The equation for the regression of $\dot{V}O_{2peak}$ on age for the ln-linear model was: $\dot{V}O_{2peak}$ (mL·kgFFM^{-1}·min^{-1}) = 166·[age(yr)$^{-0.22}$], (r^2 = 0.31, SEE; 11%, 95%CI = 9-15%).

8.5.2.4.5 Peak oxygen uptake ($\dot{V}O_{2peak}$ $mL \cdot kgFFM^{-0.67} \cdot min^{-1}$)

A moderate correlation was found between age and $\dot{V}O_{2peak}$ mL·kgFFM$^{-0.67}$·min^{-1} for a linear model (r = 0.63, 95%CI = 0.34-0.81, P<0.01). The absolute and percentage decline in $\dot{V}O_{2peak}$ with age was calculated as 1.78 mL·kgFFM$^{-0.67}$·min^{-1}, (0.62 %·yr^{-1}). The regression of $\dot{V}O_{2peak}$ on age for n = 29 for the linear model was; $\dot{V}O_{2peak}$ (mL·kgFFM$^{-0.67}$·min^{-1}) = 362-1.8·age (yr), (r^2 = 0.40, SEE; 27.1 mL·kgFFM$^{-0.67}$·min^{-1}, 95%CI = 21.5-36.7 mL·kgFFM$^{-0.67}$·min^{-1}). The correlation of $\dot{V}O_{2peak}$ (mL·kgFFM$^{-0.67}$·min^{-1}) on age using a ln-linear model was r = 0.64, (95%CI = 0.36-0.82, P<0.01). The equation for the regression of $\dot{V}O_{2peak}$ on age for the ln-linear model was: $\dot{V}O_{2peak}$ (mL·kgFFM$^{-0.67}$·min^{-1}) = 739·[age(yr)$^{-0.26}$], (r^2 = 0.41, SEE; 10%, 95%CI = 8-14%).

Table 23. Absolute and % rate of decline for $\dot{V}O_{2peak}$ recorded in 29 men aged 25-62

$\dot{V}O_{2peak}$	Absolute rate of decline per year	Relative (%) rate of decline per year
$mL \cdot min^{-1}$	36.5	0.79
$mL \cdot kg^{-1} \cdot min^{-1}$	0.51	0.82
$mL \cdot kg^{-0.67} \cdot min^{-1}$	2.08	0.81
$mL \cdot kgFFM^{-1} \cdot min^{-1}$	0.39	0.53
$mL \cdot kgFFM^{-0.67} \cdot min^{-1}$	1.78	0.62

Table 24. Age-related variance (r^2) in $\dot{V}O_{2peak}$

$\dot{V}O_{2peak}$	linear model	Ln-linear model
$mL \cdot min^{-1}$	0.45	0.48
$mL \cdot kg^{-1} \cdot min^{-1}$	0.44	0.43
$mL \cdot kg^{-0.67} \cdot min^{-1}$	0.47	0.48
$mL \cdot kgFFM^{-1} \cdot min^{-1}$	0.31	0.31
$mL \cdot kgFFM^{-0.67} \cdot min^{-1}$	0.40	0.41

8.5.2.5 PEAK CARBON DIOXIDE PRODUCTION ($\dot{V}CO_2$)

The mean value for $\dot{V}O_{2peak}$ recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 5.52 ($L \cdot min^{-1}$) with a range from 4.02 to 7.02 ($L \cdot min^{-1}$) (see Table 21). A moderate correlation was found between individual peak carbon dioxide production $\dot{V}CO_2$ $L \cdot min^{-1}$ ($\dot{V}CO_{2peak}$) versus age using a linear model (r = 0.70, 95%CI = 0.45-0.85, P<0.01). The absolute and percentage decline in $\dot{V}CO_{2peak}$ with age was calculated as 0.046 $L \cdot min^{-1} \cdot yr^{-1}$, (~ 0.83 %·$yr^{-1}$). The regression of $\dot{V}CO_{2peak}$ on age for n = 29 using a linear model was; $\dot{V}CO_{2peak}$ ($L \cdot min^{-1}$) = 7.42-0.046·age (yr), (r^2 = 0.49, SEE; 0.54 $L \cdot min^{-1}$, 95%CI = 0.43-0.73 $L \cdot min^{-1}$). The correlation of $\dot{V}CO_{2peak}$ on age using a ln-linear model was r = 0.70, (95%CI = 0.45-0.85, P<0.01). The equation for the regression of $\dot{V}CO_{2peak}$ on age for the ln-linear model was: $\dot{V}CO_{2peak}$ ($L \cdot min^{-1}$) = 19.7·[age(yr)$^{-0.35}$], (r^2 = 0.50, SEE; 11%, 95%CI = 9-15%).

8.5.2.6 PEAK RESPIRATORY EXCHANGE RATIO (RER)

The mean value for peak RER recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 1.20 with a range from 1.06 to 1.27 (see Table 21). No relationship was found between individual peak RER and age using a linear model (r = 0.20, 95%CI = -0.18-0.53, P = 0.28).

8.5.2.7 PEAK CYCLING ECONOMY

The mean value for peak cycling economy ($King_{Wpeak}/\dot{V}O_{2peak}$) recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 87 [$W \cdot \dot{V}O_2 (L \cdot min^{-1})$] with a range from 76 to 98 [$W \cdot \dot{V}O_2 (L \cdot min^{-1})$] (see Table 25). Table 25 shows the Pearson product moment correlation coefficients calculated for individual peak economy and age using a linear model when economy was expressed relative to body size and scaled to body mass using the mass exponent 0.67. No relationship (P>0.05) was found between peak economy and age.

Table 25. Relationships between peak economy and age based on a linear model (n = 29)

Economy	r	95%CI	P
$W \cdot \dot{V}O_2 (L \cdot min^{-1})$	0.10	-0.28 to 0.45	0.60
$W \cdot \dot{V}O_2 (mL \cdot kg^{-1} \cdot min^{-1})$	0.01	-0.36 to 0.38	0.94
$W \cdot \dot{V}O_2 (mL \cdot kgFFM^{-1} \cdot min^{-1})$	0.32	-0.05 to 0.61	0.10
$W \cdot \dot{V}O_2 (mL \cdot kg^{-0.67} \cdot min^{-1})$	0.01	-0.36 to 0.38	0.94
$W \cdot \dot{V}O_2 (mL \cdot kgFFM^{-0.67} \cdot min^{-1})$	0.28	-0.10 to 0.59	0.14

8.5.2.8 PEAK VENTILATION ($\dot{V}_{EPEAK}$)

The mean value for $\dot{V}_{Epeak}$ recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 144 ($L \cdot min^{-1}$, STPD) with a range from 114 to 173 ($L \cdot min^{-1}$) (see Table 21). A moderate correlation was found for age and $\dot{V}_{Epeak}$ using a linear model (r = 0.51, 95%CI = 0.18-0.74, P< 0.01). The absolute and percentage decline in $\dot{V}_{Epeak}$ with age was calculated as 0.75 $L \cdot min^{-1} \cdot yr^{-1}$, (~ 0.52 %$\cdot yr^{-1}$). The regression of $\dot{V}_{Epeak}$ on age for n = 29 using a linear model was; $\dot{V}_{Epeak}$ ($L \cdot min^{-1}$) = 175-0.75$\cdot$age (yr), (r^2 = 0.26, SEE; 15.5 $L \cdot min^{-1}$, 95%CI = 12.3-21.0 $L \cdot min^{-1}$). The correlation of $\dot{V}_{Epeak}$ on age using a ln-linear model was r = 0.51, (95%CI = 0.18-0.74, P<0.01). The equation for the regression of $\dot{V}_{Epeak}$

on age for the ln-linear model was: $\dot{V}_{Epeak}$ (L·min⁻¹) = 307·[age(yr)⁻⁰·²¹], (r² = 0.26, SEE; 12%, 95%CI = 10-16%).

8.5.2.9 PEAK BREATHING FREQUENCY

The mean value for peak breathing frequency recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 58 (breath·min⁻¹) with a range from 47 to 78 (breath·min⁻¹) (see Table 21). No significant correlation was found between individual peak breathing frequency on age for 29 subjects (range 25-62 yr) using a linear model (r = 0.13, 95%CI = -0.25-0.47, P = 0.52).

8.5.2.10 PEAK VENTILATORY EQUIVALENTS $\dot{V}O_{2PEAK}$ AND $\dot{V}CO_{2PEAK}$)

The mean values for peak ventilatory equivalents for $\dot{V}O_2$ and $\dot{V}CO_2$ recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) were 31.5 and 26.3 respectively (see Table 21). No significant correlation was found between individual peak $\dot{V}_E/\dot{V}O_2$ on age using a linear model (r = 0.28, 95%CI = -0.10-0.59, P = 0.14). A weak positive correlation was found between individual peak $\dot{V}_E/\dot{V}CO_2$ on age using a linear model (r = 0.44, 95%CI = 0.09-0.69, P<0.05).

BLa₅ (mmol·l⁻¹)
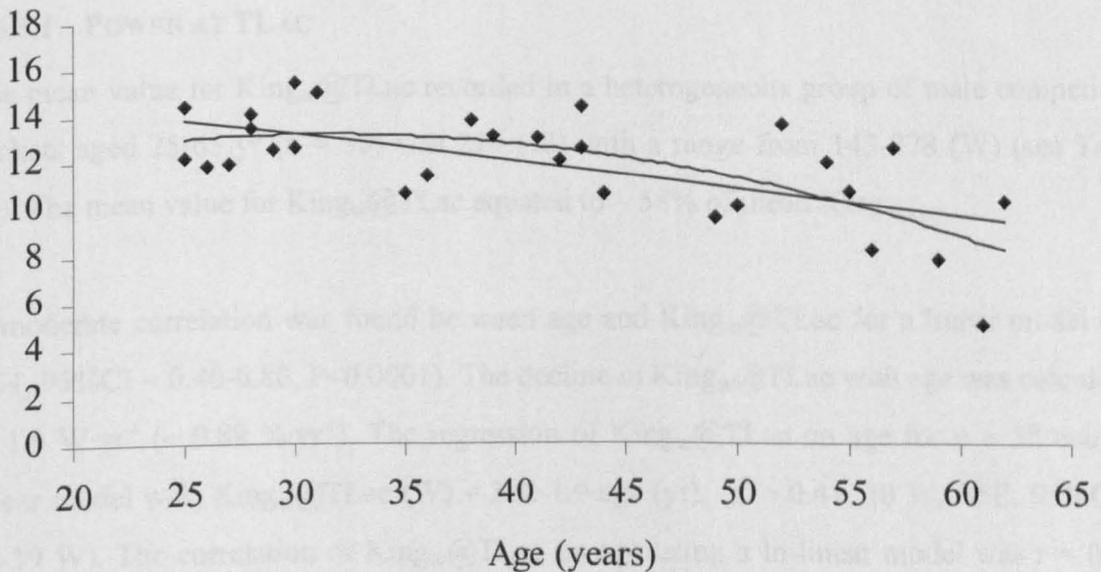
Figure 13. The regression of BLa₅ (mmol·L⁻¹) on age for 29 men aged 25-62. Data fitted with a linear and 2ⁿᵈ order polynomial model

8.5.2.11 BLOOD LACTATE AT 5 MIN POST PAC (BLa$_s$)

The mean value for BLa$_s$ recorded in a heterogeneous group of male competitive cyclists aged 25-62 yr (n = 29) was 11.90 (mmol·L^{-1}) with a range from 5.04 to 15.45 (mmol·L^{-1}) (see Table 21). Individual blood lactate concentration at 5 min post PAC (BLa$_s$) versus age is depicted in Figure 13. A moderate correlation was found between age and BLa$_s$ for a quadratic (2nd order polynomial) model (r = 0.69, 95%CI = 0.43-0.84, P<0.01) and linear model (r = 0.63, 95%CI = 0.34-0.81, P<0.01). There was no significant difference between linear and quadratic models (F = 1.16).

The regression of BLa$_s$ on age for n = 29 using a quadratic model was; BLa$_s$ (mmol·L^{-1}) = [-0.0052·(age(yr)^2]+0.3236·age (yr) + 8.149, (r^2 = 0.47, SEE; 1.50 mmol·L^{-1}, 95%CI = 1.19-2.03 mmol·L^{-1}). The regression of BLa$_s$ on age for n = 29 using a linear model was; BLa$_s$ (mmol·L^{-1}) = 16.89-0.12·age (yr), (r^2 = 0.40, SEE; 1.8 mmol·L^{-1}, 95%CI = 1.43-2.43 mmol·L^{-1}). The correlation of BLa$_s$ on age using a ln-linear model was r = 0.57, (95%CI = 0.26-0.77, P<0.01). The equation for the regression of BLa$_s$ on age for the ln-linear model was: BLa$_s$ (mmol·L^{-1}) = 39·[age(yr)$^{-0.32}$], (r^2 = 0.32, SEE; 8%, 95%CI = 6-11%).

8.5.3 PART 3

8.5.3.1 POWER AT TLAC

The mean value for King$_w$@TLac recorded in a heterogeneous group of male competitive cyclists aged 25-65 yr (n = 38) was 214 (W) with a range from 143-278 (W) (see Table 26). The mean value for King$_w$@TLac equated to ~ 58% of mean King$_{Wpeak}$.

A moderate correlation was found between age and King$_w$@TLac for a linear model (r = 0.64, 95%CI = 0.40-0.80, P<0.0001). The decline in King$_w$@TLac with age was calculated as 1.9 W·yr^{-1} (~ 0.89 %·yr^{-1}). The regression of King$_w$@TLac on age for n = 38 using a linear model was; King$_w$@TLac (W) = 302-1.9·age (yr), (r^2 = 0.41, 30 W, SEE; 95%CI = 25-39 W). The correlation of King$_w$@TLac on age using a ln-linear model was r = 0.61, (95%CI = 0.36-0.78, P<0.0001). The equation for the regression of King$_w$@TLac on age for the ln-linear model was: King$_w$@TLac (W) = 903·[age(yr)$^{-0.39}$], (r^2 = 0.38, SEE; 16%, 95%CI = 13-21%). The absolute (W) rate of decline in King$_w$@TLac was less than the

decline in $King_{Wpeak}$ (1.9 vs 3.1 $W \cdot yr^{-1}$, respectively) however this difference did not reach the level of significance (P = 0.07).

Table 26. Mean ± SD values for subjects' physical characteristics and threshold values determined during LT_{ramp} (n = 38)

	Mean (± SD)	Range
Age (yr)	45 ± 12	25-65
Height (m)	1.79 ± 0.06	1.69-1.91
Body mass (kg)	76.1 ± 5.8	62.8-92.4
$King_{Wpeak}$ (W)	370 ± 56	282-483
$King_W$@TLac (W)	214 ± 38	143-278
$King_W$@OBLA (W)	279 ± 44	189-383
HR_{peak} ($b \cdot min^{-1}$)	183 ± 13	155-207
HR@TLac ($b \cdot min^{-1}$)	139 ± 14	115-167
HR@OBLA ($b \cdot min^{-1}$)	161 ± 13	135-180
BLa@TLac ($mmol \cdot L^{-1}$)	1.52 ± 0.49	0.75-2.60

8.5.3.2 POWER AT OBLA

The mean value for $King_W$@OBLA recorded in a heterogeneous group of male competitive cyclists aged 25-65 yr (n = 38) was 279 (W) with a range from 189-383 (W) (see Table 26). The mean value for $King_W$@OBLA equated to ~ 75% of mean W_{peak}

A moderate correlation was found between age and $King_W$@OBLA for a linear model (r = 0.55, 95%CI = 0.27-0.74, P<0.0001). The decline in $King_W$@OBLA with age was calculated as 2.0 $W \cdot yr^{-1}$ (~ 0.72 $\% \cdot yr^{-1}$). The regression of $King_W$@OBLA on age for n = 38 using a linear model was; $King_W$@OBLA (W) = 368-2.0·age (yr), (r^2 = 0.31, SEE; 38 W, 95%CI = 30-48 W). The correlation of $King_W$@OBLA on age using a ln-linear model was r = 0.54, (95%CI = 0.26-0.74, P<0.0001). The equation for the regression of $King_W$@OBLA on age for the ln-linear model was: $King_W$@OBLA (W) = 824·[age(yr)$^{-0.29}$], (r^2 = 0.29, SEE 15%, 95%CI = 12-19%). The absolute rate of decline in $King_W$@OBLA was less than the

decline in $King_{Wpeak}$ (2.0 vs 3.1 $W \cdot yr^{-1}$, respectively) however this difference did not reach the level of significance (P = 0.11).

8.5.3.3 HEART RATE AT TLAC

The mean value for HR@TLac recorded in a heterogeneous group of male competitive cyclists aged 25-65 yr (n = 38) was 139 ($b \cdot min^{-1}$) with a range from 115-167 ($b \cdot min^{-1}$) (see Table 26). The mean value for HR@TLac equated to ~ 76% of mean HR_{peak}

A moderate correlation was found between age and HR@TLac for a linear model (r = 0.62, 95%CI = 0.37-0.79, P<0.0001). The decline in HR@TLac with age was calculated as 0.7 $b \cdot min^{-1} \cdot yr^{-1}$ (~ 0.50 %·yr^{-1}). The regression of HR@TLac on age for n = 38 using a linear model was; HR@TLac ($b \cdot min^{-1}$) = 171-0.7·age (yr), (r^2 = 0.39, SEE; 11 $b \cdot min^{-1}$, 95%CI = 9-14 $b \cdot min^{-1}$). The correlation of HR@TLac on age using a ln-linear model was r = 0.63, (95%CI = 0.38-0.79, P<0.0001). The equation for the regression of HR@TLac on age for the ln-linear model was: HR@TLac ($b \cdot min^{-1}$) = 317·[age(yr)$^{-0.22}$], (r^2 = 0.39, SEE 9%, 95%CI = 7-12%). There was no difference (P = 0.59) in the absolute rate of decline calculated for HR@TLac and HR_{peak} (0.7 vs 0.8 $b \cdot min^{-1} \cdot yr^{-1}$, respectively).

8.5.3.4 HEART RATE AT OBLA

The mean value for HR@OBLA recorded in a heterogeneous group of male competitive cyclists aged 25-65 yr (n = 38) was 161 ($b \cdot min^{-1}$) with a range from 135-180 ($b \cdot min^{-1}$) (see Table 26). The mean value for HR@OBLA equated to ~ 88% of mean HR_{peak}.

A moderate correlation was found between age and HR@OBLA for a linear model (r = 0.61, 95%CI = 0.36-0.78, P<0.0001). The decline in HR@OBLA with age was calculated as 0.6 $b \cdot min^{-1} \cdot yr^{-1}$ (~ 0.37 %·yr^{-1}). The regression of HR@OBLA on age for n = 38 using a linear model was; HR@OBLA ($b \cdot min^{-1}$) = 189-0.6·age (yr), (r^2 = 0.38, SEE; 10 $b \cdot min^{-1}$, 95%CI = 8-13 $b \cdot min^{-1}$). The correlation of HR@OBLA on age using a ln-linear model was r = 0.61, (95%CI = 0.36-0.78, P<0.0001). The equation for the regression of HR@OBLA on age for the ln-linear model was: HR@OBLA ($b \cdot min^{-1}$) = 301·[age(yr)$^{-0.17}$], (r^2 = 0.38, SEE 7%, 95%CI = 6-9%). There was no difference (P = 0.26) in the absolute rate of decline calculated for HR@OBLA and HR_{peak} (0.6 vs 0.8 $b \cdot min^{-1} \cdot yr^{-1}$, respectively).

Table 27. Absolute and relative decline for King$_{Wpeak}$ and HR$_{peak}$ and power and heart rate determined at TLac and OBLA thresholds

	Absolute decline per year	Percentage decline per year
King$_{Wpeak}$ (W)	3.1	0.84
King$_W$@TLac (W)	1.9	0.89
King$_W$@OBLA (W)	2.0	0.72
HR$_{peak}$ (b·min^{-1})	0.8	0.44
HR@TLac (b·min^{-1})	0.7	0.50
HR@OBLA (b·min^{-1})	0.6	0.37

8.5.3.5 BLOOD LACTATE AT TLAC

The mean value for BLa@TLac recorded in a heterogeneous group of male competitive cyclists aged 25-65 yr (n = 38) was 1.52 (mmol·L^{-1}) with a range from 0.75-2.60 (mmol·L^{-1}) (see Table 26). No correlation was found between individual BLa@TLac on age using a linear model (r = 0.07, 95%CI = -0.26-0.38, P = 0.64).

8.5.4 PART 4

8.5.4.1 AVERAGE POWER DURING A LABORATORY BASED 16.1-KM TIME TRIAL (KING$_{WLTT}$)

The mean value for King$_{WLTT}$ recorded in a heterogeneous group of male competitive cyclists aged 25-63 yr (n = 45) was 298 (W) with a range from 199 to 408 (W) (see Table 28). Individual average power sustained during a 16.1-km time trial (King$_{WLTT}$) and King$_{Wpeak}$ versus age are depicted in Figure 14.

A moderate correlation was found between age and King$_{WLTT}$ for a linear model (r = 0.66, 95%CI = 0.44-0.80, P<0.01). The decline in King$_{WLTT}$ with age was calculated as 2.6 W·yr^{-1} (~ 0.87 %·yr^{-1}). The regression of King$_{WLTT}$ on age for n = 45 using a linear model was; King$_{WLTT}$ (W) = 412-2.6·age (yr), (r^2 = 0.44, SEE; 38 W, 95%CI = 31-48 W). The correlation of King$_{WLTT}$ on age using a ln-linear model was r = 0.66, (95%CI = 0.45-0.80, P<0.01). The equation for the regression of King$_{WLTT}$ on age for the ln-linear model was: King$_{WLTT}$ (W) = 1147·[age(yr)$^{-0.36}$], (r^2 = 0.44, SEE; 13%, 95%CI = 11-16%). The absolute

and percentage decline in King$_{WLTT}$ when expressed as W·kg^{-1} and W·kg$^{-0.67}$ was calculated as 0.03 W·kg^{-1}·yr^{-1} (~ 0.83 %·yr^{-1}) and 0.14 W·kg$^{-0.67}$·yr^{-1} (~ 0.84 %·yr^{-1}) respectively.

Table 28. Mean ± SD values for subjects' physical characteristics and performance related responses during an indoor 16.1-km cycling time trial (n=45)

	Mean (± SD)	Range
Age (yr)	44 ± 13	25-63
Height (m)	1.79 ± 0.06	1.62-1.91
Body mass (kg)	75.5 ± 6.6	56.5-92.4
King$_{Wpeak}$ (W)	376 ± 65	226-538
King$_{WLTT}$ (W)	298 ± 50	199-408
T$_{LTT}$ (min:s)	22:45 ± 1:46	19:37-27:16
HR$_{peak}$ (b·min^{-1})	183 ± 13	155-207
HR$_{LTT}$ (b·min^{-1})	171 ± 12	142-192

T$_{LTT}$ = performance time for 16.1-km time trial
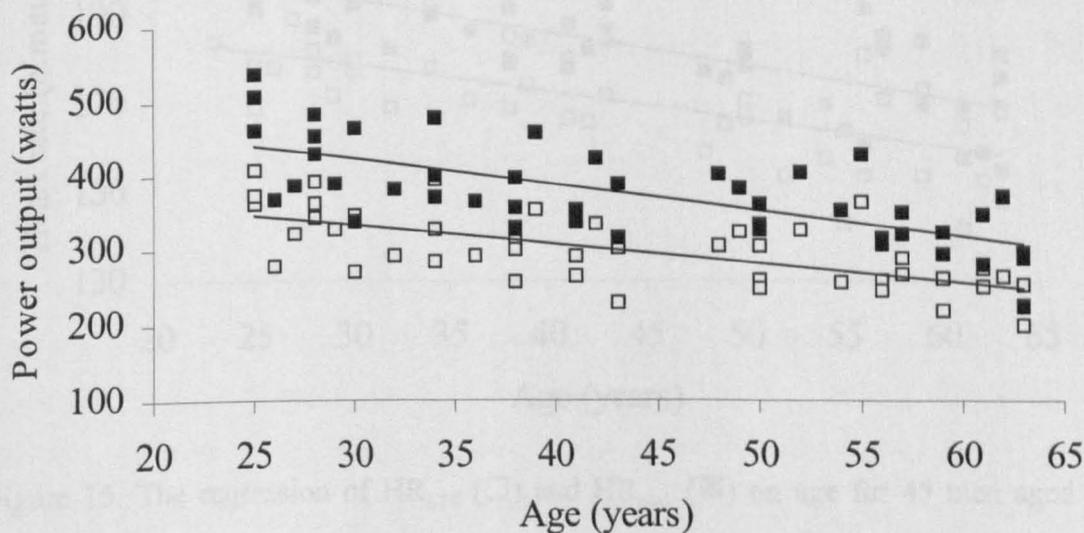
Figure 14. The regression of King$_{WLTT}$ (□) and King$_{Wpeak}$ (■) on age for 45 men aged 25-62 yr. Data fitted with linear models. The absolute (W) rate of decline in King$_{WLTT}$ was less than the decline in King$_{Wpeak}$ (2.6 vs 3.6 W·yr^{-1}, respectively), however this difference did not reach the level of significance (P = 0.18)

8.5.4.2 AVERAGE HEART RATE DURING A LABORATORY BASED 16.1-KM TIME TRIAL
(HR$_{LTT}$)

The mean value for HR$_{LTT}$ (b·min^{-1}) recorded in a heterogeneous group of male competitive cyclists aged 25-63 yr was 171 (b·min^{-1}) with a range from 142 to 192 (b·min^{-1}) (see Table 28). Individual average heart rate sustained during a 16.1-km time trial (HR$_{LTT}$) and HR$_{peak}$ versus age are depicted in Figure 15.

A moderate correlation was found between age and average heart rate maintained during an indoor 16.1-km TT (HR$_{LTT}$) for a linear model (r = 0.66, 95%CI = 0.44-0.80, P<0.01). The decline in HR$_{LTT}$ with age was calculated as 0.61 b·min^{-1}·yr^{-1} (0.36 %·yr^{-1}). The regression of HR$_{LTT}$ on age for n = 45 using a linear model was; HR$_{LTT}$ (b·min^{-1}) = 197-0.62·age (yr), (r^2 = 0.44, SEE; 9 b·min^{-1}, 95%CI = 7-11 b·min^{-1}). The correlation of HR$_{LTT}$ on age using a ln-linear model was r = 0.65, (95%CI = 0.44-0.79, P<0.01). The equation for the regression of HR$_{LTT}$ on age for the ln-linear model was: HR$_{LTT}$ (b·min^{-1}) = 295·[age(yr)$^{-0.15}$], (r^2 = 0.42, SEE; 6%, 95%CI = 5-8 %).
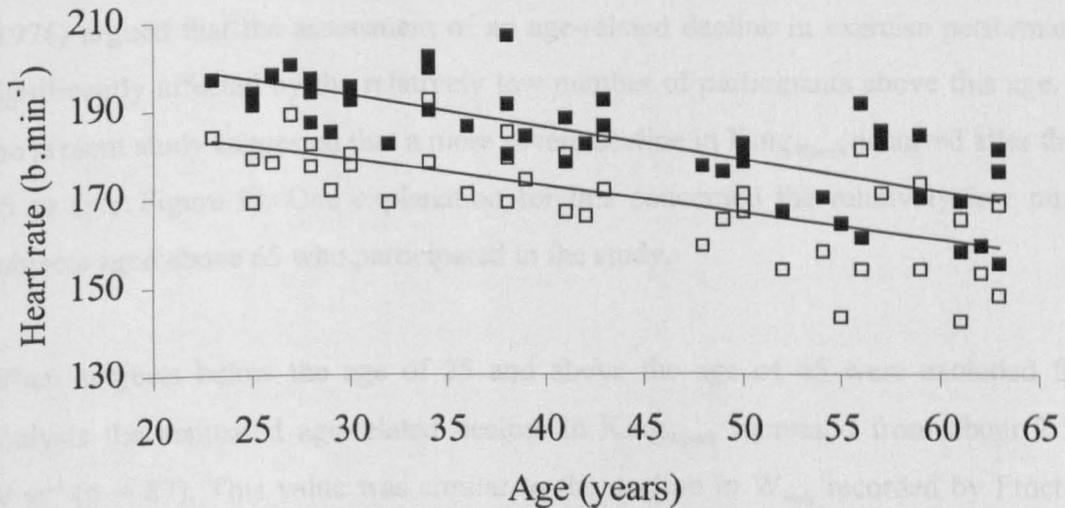
Figure 15. The regression of HR$_{LTT}$ (□) and HR$_{peak}$ (■) on age for 45 men aged 25-62. Data fitted with linear models. There was no difference (P = 0.35) in the absolute rate of decline calculated for HR$_{LTT}$ and HR$_{peak}$ (0.62 vs 0.75 b·min^{-1}·yr^{-1} respectively)

8.6 DISCUSSION

Hawley and Noakes (1992) found that a measure of maximal power whether expressed as $\dot{V}O_{2max}$ or W$_{max}$ was highly related to endurance performance ability in trained cyclists.

Surprisingly, there is very little information available concerning the age-related decline in metabolic, cardio-respiratory and performance related responses recorded during a cycling maximal exercise test and there is no information available concerning the age-related decline in cycling endurance performance of trained competitive cyclists.

8.6.1 AGE-RELATED CHANGES IN KING$_{Wpeak}$

Very few studies have investigated the decline in maximal power of endurance cyclists (Kavanagh and Shephard, 1990; Proctor *et al.*, 1998). Data from the present study revealed that the estimated age-related decline in W$_{peak}$ was similar to the value reported by Kavanagh and Shephard (1990) for a group of 756 mixed ability competitive athletes. In the present study, peak power (King$_{Wpeak}$) was highest at about 30 yr of age (see Figure 9), this value concurred with data concerning the age of cyclists who have won the Tour de France cycling race (see Figure 2) and with studies which have assessed the cross sectional age-related decline in endurance performance (Grogan *et al.*, 1991; Joyner, 1993; Moore, 1975; Salthouse, 1976). Several authors have observed a sharper decline in maximal aerobic power after the age of 60 (Joyner, 1993; Pollock *et al.*, 1997). However, Salthouse (1976) argued that the assessment of an age-related decline in exercise performance was significantly affected by the relatively low number of participants above this age. Data in the present study suggested that a more severe decline in King$_{Wpeak}$ occurred after the age of 65 yr (see Figure 9). One explanation for this concerned the relatively low number of subjects aged above 65 who participated in the study.

When subjects below the age of 25 and above the age of 65 were excluded from the analysis the estimated age-related decline in King$_{Wpeak}$ increased from about 2.5 to 3.0 W·yr^{-1} (n = 87). This value was similar to the decline in W$_{max}$ recorded by Proctor *et al.* (1998) who assessed the endurance capacity of 30 veteran athletes. In the present study, when the decline in King$_{Wpeak}$ was calculated for the top two performers in age categories of five year from the age of 25 to 29 and 60 to 64 the absolute decline in King$_{Wpeak}$ was higher when compared with a large mixed ability group (4.10 vs 3.05 W·yr^{-1}). This finding was similar to the work of Seiler *et al.* (1998) who found that the absolute age-related rate of decline in power output maintained during an indoor rowing ergometer performance test

was higher for best performers (4.14 vs 3.25 $W \cdot yr^{-1}$). It is important to note that all of these calculations were based on cross-sectional methods of analysis.

Tanaka *et al.* (1997) found that the absolute rate of decline calculated for $\dot{V}O_{2max}$ per year was higher in highly trained athletes when compared with relatively untrained age matched individuals. However the relative (%) rate of decline was similar for each group. Data from the present study revealed that the absolute and relative rate of decline in $King_{Wpeak}$ was higher in the top performers (n = 16) when compared with the mixed ability group (n = 87). It is difficult to determine whether this finding was due to the use of cross sectional analysis and the selection of subjects who participated in the study or was a true reflection of the age-related decline in peak power. In contrast to this, Seiler *et al.* (1998) found that the relative rate of decline (based on cross sectional analysis) for power output maintained during a rowing test was lower in the elite competitors (~ 0.90 vs 1.25 $\% \cdot yr^{-1}$), however the number of subjects in this study was relatively high (n = 2487). Unfortunately there is no other information available concerning age-related declines in W_{peak} recorded during a Kingcycle PP test, therefore the findings of the present study provide new information on this issue.

No information is available concerning the relative contribution of anaerobic power to W_{peak}, however age-related declines in peak and mean anaerobic power can be more pronounced than declines in peak 'aerobic' power (Chamari *et al.*, 1995), this would explain the higher rate of decline observed in the group of best performers in that anaerobic power contributed more to the values of W_{peak} achieved by senior highly trained cyclists and a more pronounced age-related decline in anaerobic power affected the W_{peak} of the cyclists who achieved the highest values. Data concerning the effect of endurance training on muscle fibre type (for review see McComas, 1996) suggests that the combined effect of age and endurance training would result in a marked change in fibre type composition in the veteran athlete. The alteration of Type IIb to Type IIa linked to endurance training and ageing could lead to a reduction in anaerobic power (Sargeant, 1994), therefore this supposition may explain the observed cross sectional age-related decline in W_{peak}.

In the present study age accounted for about 31% of the variance in $King_{Wpeak}$ in a heterogeneous group of cyclists (n = 87). The decline in W_{peak} can be affected by changes in body composition and environmental factors such as habitual physical activity, nutrition and chronic diseases (Menard and Stanish, 1989) and it is reasonable to assume that levels of physical activity and training were more consistent among the performers with the highest values for $King_{Wpeak}$ relative to age, in support of this postulate age accounted for about 78% of the variance in $King_{Wpeak}$ in the more homogenous (best performers) group.

The combination of age and physical stature accounted for 49% of the variance in $King_{Wpeak}$ (n = 87). Therefore inter-individual differences in body size had a significant affect on $King_{Wpeak}$. Further evidence to support this postulate was found when the relative rate of decline in $King_{Wpeak}$ was increased when values were normalised for differences in body size ($W \cdot kg^{-0.67} \cdot yr^{-1}$), however this increase was not observed in the more homogenous group of best performers (n = 16). These findings highlighted problems associated with subject selection and cross sectional assessment of absolute and relative declines in W_{peak} and suggests that issues of using appropriate methods of scaling may be more important when evaluating large heterogeneous groups of subjects. Unfortunately there is no information available concerning the longitudinal decline in W_{peak} with ageing.

8.6.2 AGE-RELATED CHANGES IN HR_{PEAK}

Several longitudinal studies have assessed the age-related rate of decline in HR_{max} (Kasch et al., 1995; Pollock et al., 1997; Trappe et al., 1996). Data from the present study revealed a cross sectional decline in HR_{peak} with age of about 0.66 ($b \cdot min^{-1} \cdot yr^{-1}$) this value was similar to the decline in HR_{max} reported by Kasch et al. (1995). In the present study, age accounted for 56% of the variance in HR_{peak} and the standard error of estimate for the prediction of HR_{peak} with 95% confidence was 4 to 6%, therefore an accurate estimation of HR_{peak} would not be obtained using a prediction equation such as 220-age as proposed by Londeree and Moeschberger (1982). For instance, 95% ratio limits of agreement calculated for HR_{peak} recorded during PP (n = 114) and predicted HR_{peak} based on the equation 220 - age were similar ($\times/\div$ 1.11). Therefore as an example, an athlete with an age predicted HR_{peak} of 190 $b \cdot min^{-1}$ could have a maximum value anywhere between 171 and 211 $b \cdot min^{-1}$. This questions the efficacy of using the Londeree and Moeschberger (1982) equation to

predict HR$_{peak}$. Data collected in the present study suggests that the equation 210-(0.66·age) could be used as effectively to predict HR$_{peak}$ in cyclists. As can be seen in Figure 16 predicted values for HR$_{peak}$ based on each equation are notably different, particularly in the older age range. Therefore the equation based on data collected in the present study may provide a more effective method of predicting HR$_{peak}$ in veteran cyclists. However, it is important to note that 18 cyclists (16%) of the group (n = 114) did not attain the HR$_{peak}$ value used as a criterion measure to assess the achievement of VO$_{2max}$ (Howley et al., 1995).
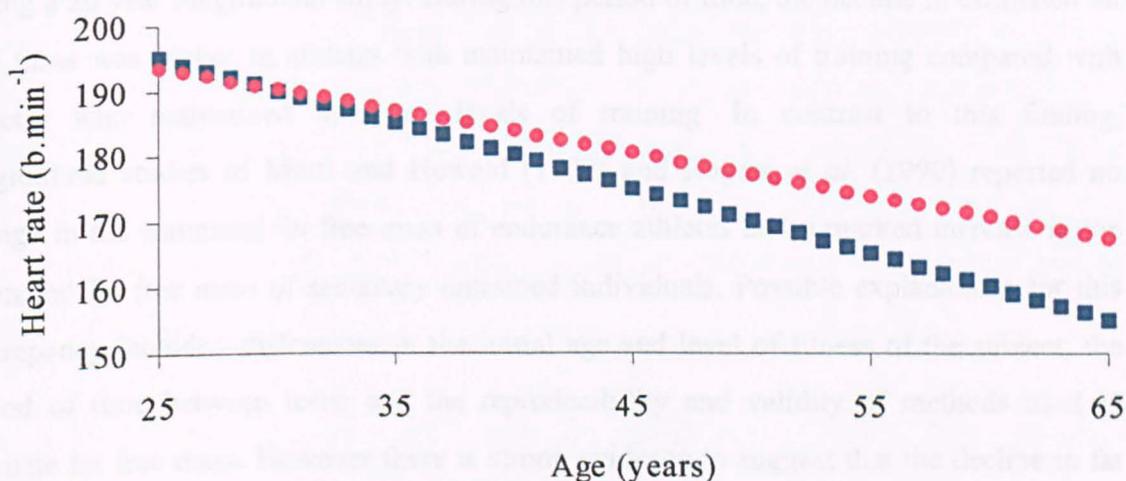
Figure 16. Predicted values for HR$_{peak}$ based on the regression equation 220-age (■) from Londeree and Moeschberger (1982) and 210-(0.66·age) (●) from the present study

Joyner (1993) postulated that the rate of decline in HR$_{max}$ can be reduced with chronic exercise, however there is very little information available to support this supposition. In the present study, a higher rate of decline was recorded in the group of best performers (n =16) when compared with the heterogeneous group of competitors and age accounted for 88% of the variance in HR$_{peak}$ in the group of best performers. It is worth noting that longitudinal and cross sectional studies have estimated the rate of decline in HR$_{max}$ with age using peak heart rate achieved by endurance runners during a running test (Pollock et al., 1997; Trappe et al., 1996) and sedentary and trained individuals during a cycle ergometer test (Babcock et al., 1992; Overend et al., 1992; Proctor et al., 1998). To my knowledge there is no information available concerning the age-related decline of HR$_{peak}$ in competitive cyclists.

8.6.3 AGE-RELATED CHANGES IN BODY MASS AND FAT FREE MASS

Various authors have reported an increase in body mass with age (McArdle *et al.*, 1991; Robergs and Roberts, 1996) however in the present study no age-related increase in body mass was found in a heterogeneous group of 29 competitive cyclists. Notably a significant decline in fat free mass (FFM) with age was found in this group of cyclists. Age accounted for about 15% of the variance in fat free mass therefore other factors such as inter-individual differences in physical stature, inherent endurance capacity and habitual physical activity may have influenced estimated fat free mass values. Pollock *et al.* (1997) investigated age-related changes in estimated fat free mass of endurance trained athletes during a 20 year longitudinal study. During this period of time, the decline in estimated fat free mass was higher in athletes who maintained high levels of training compared with athletes who maintained moderate levels of training. In contrast to this finding, longitudinal studies of Marti and Howald (1990) and Rogers *et al.* (1990) reported no change in the estimated fat free mass of endurance athletes and a marked increase in the estimated fat free mass of sedentary untrained individuals. Possible explanations for this discrepancy include:- differences in the initial age and level of fitness of the subject; the period of time between tests; and the reproducibility and validity of methods used to estimate fat free mass. However there is strong evidence to suggest that the decline in fat free mass with age is more pronounced in endurance athletes. This postulate is supported by cross sectional studies (Coggan *et al.*, 1993; Hagberg *et al.*, 1988) which have reported a faster rate of decline in FFM estimated in veteran athletes when compared with sedentary controls.

8.6.4 AGE-RELATED CHANGES IN HEIGHT

A reduction in height due to the effects of ageing has been frequently reported in the literature (Robergs and Roberts, 1996). In the present study a significant relationship was found between age and height in a heterogeneous group of 29 competitive cyclists and age accounted for about 22% of the variation in height recorded in this group. When multiple In linear regression analysis was used to assess the interrelationships between age, height and body mass on $King_{Wpeak}$ in a heterogeneous group of 114 cyclists, analysis of the data revealed that body mass did not contribute to the variance in $King_{Wpeak}$ and the combination of age and height accounted for 49% of the variance in $King_{Wpeak}$. Interestingly the

exponent for height (H) calculated using multivariate allometric scaling of age and height (H) was $H^{1.96}$ which was similar to the value of H^2 used in the estimation of body surface area (McArdle *et al.*, 1991).

8.6.5 AGE-RELATED CHANGES IN PERCENTAGE BODY FAT

Several cross sectional and longitudinal studies have reported an age-related increase in percentage body fat (%BF) (Coggan *et al.*, 1990; 1993; Hagberg *et al.*, 1985; 1988; Heath *et al.*, 1981; Proctor and Joyner, 1997; Proctor *et al.*, 1995; Rosen *et al.*, 1998). In the present study there was a significant increase in %BF relative to age in a heterogeneous group of trained cyclists. Notably, age accounted for 50% of the variance in %BF assessed in this group of competitors. Trappe *et al.* (1996) found that %BF assessed in veteran runners was affected by inter-individual differences in training, therefore it is reasonable to assume that the wide range of values for %BF reported in the present study were influenced by the training history of each subject. The increase in %BF with age has also been attributed to a reduction in basal metabolic rate due to the age-related loss of muscle mass (Piers *et al.*, 1998).

Methodological problems associated with the estimation of %BF using skinfold measurements need to be considered when evaluating the relationship between age and changes in %BF (Clarys *et al.* 1987). For instance, estimations of body density and %BF are based on equations developed from the assessment of untrained individuals. Notably, Lillystone *et al.* (1999) assessed the validity of several methods of measuring %BF against dual-energy X-ray absorptiometry (DEXA) and found that skinfolds were a more valid method of assessment of %BF in male marathon runners when compared with underwater weighing and bioelectrical impedance. However the validity of skinfold measures for the estimation of %BF of endurance cyclists has not been investigated.

8.6.6 AGE-RELATED CHANGES IN $\dot{V}O_{2PEAK}$

The effect of age on maximal aerobic power ($\dot{V}O_{2max}$) has been extensively studied and reviewed (Joyner, 1993; Menard and Stanish, 1989; Stamford, 1988). Longitudinal studies which have assessed the rate of decline in $\dot{V}O_{2max}$ relative to age have reported a wide range of values and have suggested that the absolute decline in $\dot{V}O_{2max}$ is higher in trained athletes when compared with untrained individuals (Tanaka *et al.*, 1997). However the

relative rate of decline is similar regardless of training habits and endurance capacity (Tanaka et al., 1997). In the present study a $\dot{V}O_2$-plateau (an increase in $\dot{V}O_2$ of less than 150 $mL \cdot min^{-1}$ with an increase in external workload, Taylor et al., 1955) was recorded in 17 of the subjects (59% of n = 29). Absolute values expressed as $L \cdot min^{-1}$ and $mL \cdot kg^{-1} \cdot min^{-1}$ and percentage values calculated in terms of $mL \cdot kg^{-1} \cdot min^{-1}$ for the rate of decline in $\dot{V}O_{2peak}$ with age were similar to average values calculated from longitudinal work (Marti and Howald, 1990; Pollock et al., 1997; Trappe et al., 1996; Rogers et al., 1990). However, the percentage rate of decline in $\dot{V}O_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) was lower than average values reported in longitudinal (Marti and Howald, 1990; Pollock et al., 1997; Trappe et al., 1996; Rogers et al., 1990) and cross sectional studies (Allen et al., 1985; Aminoff et al., 1996; Coggan et al., 1990, 1993; Fuchi et al., 1989; Grimby and Saltin, 1966; Hagberg et al., 1985; 1988; 1998; Heath et al., 1981; Massé-Biron et al., 1992; Overend et al., 1992; Posner et al., 1987; Prefaut et al., 1994; Proctor and Joyner, 1997; Proctor et al., 1995; 1996; 1998; Rivera et al., 1989).

Unfortunately there is very little information available concerning the effect of changes in body composition on the relative rate of decline in $\dot{V}O_{2peak}$ in older athletes. However data reported in the present study showed that changes in FFM can have a significant affect on normalised $\dot{V}O_{2peak}$ values. Age accounted for 41% of the variance in $\dot{V}O_{2peak}$ when expressed as $mL \cdot kg^{-1} \cdot min^{-1}$. Therefore about 26% of the variance in $\dot{V}O_{2peak}$ could be attributed to changes in FFM. Numerous studies have investigated the effects of training on $\dot{V}O_{2max}$ values recorded in trained and untrained young and old individuals (see Spina, 1999) however there is very little information available concerning the rate of decline attributed to the process of ageing per se. Interestingly, Batterham et al. (1999) concluded that over 50% of the cross sectional decline in $\dot{V}O_{2max}$ with ageing recorded in a large heterogeneous group of subjects (n = 1314) was due to variance in body composition and physical activity.

8.6.7 AGE-RELATED CHANGES IN $\dot{V}CO_{2PEAK}$

Very few studies have investigated the effect of age on peak values of $\dot{V}CO_2$ recorded during maximal exercise. Davis et al. (1979) observed a strong relationship between absolute values for $\dot{V}CO_{2max}$ and $\dot{V}_{Emax}$ and also the relative change in $\dot{V}_{Emax}$ and $\dot{V}CO_{2max}$

after completion of an endurance training program. Data from the present study showed that subjects who attained the highest values for $\dot{V}CO_2$ also achieved the highest values for $\dot{V}O_{2peak}$ and $\dot{V}_{Epeak}$.

8.6.8   AGE-RELATED CHANGES IN PEAK RER

Several cross sectional studies have assessed the effect of age on maximal values for RER recorded during maximal exercise and have recorded lower values of RER in older subjects (Aminoff *et al.*, 1996; Overend *et al.*, 1992; Proctor *et al.*, 1998; Tzankoff and Norris, 1979). Similarly Rogers *et al.* (1990) found a reduction in RER with age during a longitudinal assessment of the effects of ageing on maximal exercise performance capacity. In contrast to these findings Pollock *et al.* (1997) found that maximal RER recorded in highly trained athletes who maintained high levels of training during the intervening years between tests did not decline with age. In the present study there was no relationship found between age and peak RER recorded in a cross sectional heterogeneous group of trained competitive cyclists. However it is worth noting that one senior and three veteran cyclists (14% of n = 29) did not attain the value of RER (≥ 1.15) used as a criterion measure to assess the achievement of $\dot{V}O_{2max}$ (Howley *et al.*, 1995).

8.6.9   AGE-RELATED CHANGES IN CYCLING ECONOMY

No relationship between economy and age was found in the heterogeneous group of cyclists who participated in the present study. Similarly Allen *et al.* (1985) found no relationship between running economy and age. There is very little information available concerning the effect of age on maximal and sub-maximal cycling economy, this is unfortunate considering the postulate that economy is a key determinant of successful endurance performance (Anderson, 1996; Coyle *et al.*, 1991, Coyle, 1995, Horowitz *et al.*, 1994; Joyner, 1993; Olds *et al.*, 1995). Studies which have recorded W_{max} and $\dot{V}O_{2max}$ in senior and veteran athletes have found higher values for economy in senior (Proctor *et al.*, 1998) and veteran performers (Massé-Biron *et al.*, 1992).

Coyle (1995) suggested that cycling economy was strongly related to the percentage of type I muscle fibres observed in highly trained and elite cyclists and suggested that muscle fibre recruitment patterns could also explain the high values for economy recorded in these

competitors. Several studies have reported a decline in muscle mass (Coggan *et al.*, 1993; Lexell *et al.*, 1988) and a reduction in the percentage of type II fibres (Coggan *et al.*, 1990; Proctor *et al.*, 1995) with ageing. However the effect of these changes on cycling economy has not been investigated. Unfortunately age-related changes in muscle structure and function were not assessed in the present study. However the postulate that age-related changes in muscle structure and function do not affect peak cycling economy of veteran cyclists needs further consideration.

8.6.10 AGE-RELATED CHANGES IN $\dot{V}_{Epeak}$

The decline in $\dot{V}_{Emax}$ with age has been investigated on several occasions (for review see Johnson and Dempsey, 1991) and various studies have recorded a longitudinal decline in $\dot{V}_{Emax}$ with age in both highly and moderately trained athletes (Pollock *et al.*, 1997; Trappe *et al.*, 1996). Data to support the postulate that exercise training can attenuate the age-related decline in $\dot{V}_{Emax}$ are equivocal (Kasch *et al.*, 1995; Pollock *et al.*, 1997; Trappe *et al.*, 1996). In the present study the cross sectional decline in $\dot{V}_{Epeak}$ with age in competitive cyclists was relatively low compared with values determined from longitudinal work. However, it is reasonable to suggest that values reported in this study may not represent an age-related effect but indicate methodological problems associated with subject selection and the use of cross sectional analysis. However, data from the present study may provide new evidence to suggest that the age-related decline in $\dot{V}_{Epeak}$ observed in competitive cyclists is less when compared with endurance runners and untrained individuals. Notably longitudinal work which has provided inconclusive evidence regarding the effect of training on $\dot{V}_{Emax}$ have consistently investigated physiological changes in trained runners (Pollock *et al.*, 1997; Trappe *et al.*, 1996). There is no information available concerning the longitudinal decline in $\dot{V}_{Epeak}$ of endurance trained cyclists. In this study, age accounted for less than 30% of the change in $\dot{V}_{Epeak}$, therefore further research is warranted concerning the effects of other factors such as endurance training and habitual physical activity.

8.6.11 AGE-RELATED CHANGES IN PEAK BREATHING FREQUENCY

Very few studies have investigated the effect of age on breathing frequency attained during maximal exercise. Åstrand *et al.* (1997) reported a longitudinal decrease in maximal breathing frequency of about 5 breath·min^{-1} over a period of 33 years. However data in the

present study showed that there was no relationship between age and breathing frequency. The decline in $\dot{V}_{Epeak}$ with age found in the present group of cyclists appears to have been influenced by a reduction in tidal volume. There is a large volume of longitudinal (Pollock *et al.*, 1997) and cross sectional work (Morris *et al.*, 1971) to show that static and dynamic lung volumes decline with age and data presented here provides strong evidence to suggest that the decline in $\dot{V}_{Epeak}$ with age can be attenuated by the maintenance of a relatively high breathing frequency.

8.6.12 AGE-RELATED CHANGES IN VENTILATORY EQUIVALENTS FOR $\dot{V}O_{2PEAK}$ AND $\dot{V}CO_{2PEAK}$

Several authors have reported a longitudinal age-related increase in ventilatory equivalent for $\dot{V}O_{2max}$ (Åstrand *et al.*, 1997; Kasch *et al.*, 1995; Pollock *et al.*, 1997). These findings have been supported by cross sectional work which has also reported a similar age-related increase in ventilatory equivalent for $\dot{V}O_{2max}$ (Fuchi *et al.*, 1989; Heath *et al.*, 1981; Proctor *et al.*, 1998). In the present study, a very weak relationship between age and ventilatory equivalent for $\dot{V}CO_2$ was found and the relationship between age and $\dot{V}E_{peak}/\dot{V}O_{2peak}$ was not significant. Very few studies have investigated the effect of age on the ventilatory equivalent for $\dot{V}CO_2$, therefore it is difficult to compare the findings of the present study with previous work. Stamford, (1988) suggested that an increased ventilatory equivalent for $\dot{V}O_2$ with age was related to a greater response to $\dot{V}CO_2$.

8.6.13 AGE-RELATED CHANGES IN BLOOD LACTATE ASSESSED AT 5 MIN POST PAC

There is surprisingly very little information available concerning the effect of age on blood lactate concentration (BLa) recorded after the completion of maximal exercise (Åstrand and Rodahl, 1986; Roecker *et al.*, 2000). Data from the present study revealed an age-related decline in blood lactate concentration recorded five minutes after completion of a Kingcycle PAC test. This decline of 0.12 mmol·L^{-1}·yr^{-1} was less than the 0.16 mmol·L^{-1}·yr^{-1} calculated from the model of Åstrand and Rodahl (1986). Massé-Biron *et al.* (1992) argued that level of fitness can have a significant affect on peak lactatacidaemia, and methodological problems associated with the measurement of blood lactate concentration have been previously highlighted (Williams *et al.*, 1992). Study two of this thesis highlights the range of measurement error associated with the assessment of peak blood

lactate concentration which needs to be considered when comparisons are made between studies.

Although various authors have used a measure of peak BLa to assess changes in anaerobic capacity it is now generally accepted that peak BLa does not provide a valid indirect assessment of anaerobic capacity (see Vandewalle *et al.*, 1987). It is reasonable to assume that the decline in peak BLa reported in this study may be more related to a reduction in lean body mass (Proctor *et al.*, 1995). It is worth noting that values for BLa_5 recorded for three veteran cyclists (about 10% of n = 29) did not reach the value for post maximal exercise BLa of ≥ 8 $mmol \cdot L^{-1}$ used as a criterion measure to assess the achievement of $\dot{V}O_{2max}$ (Howley *et al.*, 1995).

8.6.14 AGE-RELATED CHANGES IN POWER AT TLAC AND OBLA

Very few studies (Massé-Biron *et al.*, 1992) have investigated age-related changes in threshold power in senior and veteran cyclists. Therefore data presented in this study provides new information on this issue. One important finding is that power at TLac and OBLA when expressed as a percentage of $King_{Wpeak}$ tended to increase with age. However this age associated increase did not reach the level of significance (p = 0.11). The 38 subjects who participated in the present study were of mixed ability and represented a heterogeneous population. Large inter-individual differences in performance ability, level of fitness and training history may have concealed the underlying relationship between age and exercise intensity at threshold. Several authors (Allen *et al.*, 1985; Massé-Biron *et al.*, 1992) have reported that relative exercise intensity ($\%\dot{V}O_{2max}$) at threshold was higher in veteran athletes when compared with seniors and Iredale and Nimmo (1997) found that TLac (expressed as $\%\dot{V}O_{2max}$) was higher in elderly subjects when compared with young sedentary individuals. The exact mechanism(s) responsible for this age-related change is/are unclear, however Coggan *et al.* (1990) hypothesised that lower levels of lactate dehydrogenase activity, combined with higher respiratory enzyme activity and capillary supply found in veteran athletes favoured a reduction in lactate production and thereby explained the higher lactate threshold ($\%\dot{V}O_{2max}$) recorded in elderly populations and veteran performers. Unfortunately oxygen consumption at TLac and OBLA was not

assessed in the present study, however it is likely that higher values for $\%\text{King}_{\text{Wpeak}}$ at TLac and OBLA with age would have coincided with a higher $\%\dot{V}O_{2\text{peak}}$.

Massé-Biron *et al.* (1992) found that veteran cyclists (mean age 65 yr) were unable to attain a fixed blood lactate concentration of 4 mmol·L^{-1} (OBLA) during an incremental cycling test. In the present study all of the subjects were able to attain a fixed blood lactate of 4 mmol·L^{-1}. This discrepancy may have been due to lower age of the veteran cyclists who participated in the present study, the different testing protocol used to establish OBLA exercise intensity and the fact that Massé-Biron *et al.* (1992) measured lactate concentration in venous whole blood. Several authors have suggested that peak lactacidaemia declines with age (Åstrand and Rodahl, 1986) however comparisons between senior and veteran competitors can be problematic due to the effects of diet, pre-test preparation and level of fitness on peak blood lactate concentration (see Howley *et al.* 1995). Also several methodological issues concerning the assessment of BLa such as sample site, method of assay and testing equipment need to be considered when comparisons are made between studies.

There is no information available concerning longitudinal age-related changes in power at TLac and OBLA and in order to investigate the effect of age on these variables cross sectional research has typically matched groups of senior and veteran athletes on training status (Massé-Biron *et al.* 1992) and performance (Allen *et al.* 1985). The use of a between subject factor to compare across groups is ultimately dependent on the homogeneity of variance of each group and the control of experimenter bias, therefore, comparisons between age groups may be more indicative of subject selection and not the ageing process *per se*.

8.6.15 AGE-RELATED CHANGES IN HEART RATE AT TLAC AND OBLA

Allen *et al.* (1985) assessed the effect of age on heart rate associated with a fixed blood lactate concentration of 2.5 mmol·L^{-1} and found that heart rate (expressed as $\%\text{HR}_{\text{max}}$) was higher in a group of performance matched veteran runners. Data from the present study revealed that although the absolute value for heart rate at TLac and OBLA declined with age there was no age associated decline when values were expressed as $\%\text{HR}_{\text{peak}}$. Although

relative exercise intensity (%King$_{Wpeak}$) at threshold tended to increase with age there was no concomitant increase in heart rate (%HR$_{peak}$) this indicated that the relationship between exercise intensity and heart rate varied with age and that workload at TLac and OBLA tended to be higher in veterans when expressed as %King$_{Wpeak}$ but not HR$_{peak}$. Investigators should be aware of this discrepancy when using heart rate to prescribe a relative training and competitive exercise intensity based on the relationship between power and heart rate at threshold.

8.6.16 AGE-RELATED CHANGES IN AVERAGE POWER DURING AN INDOOR 16.1-KM TIME TRIAL

There is no information available concerning the effect of age on cycling time trial performance. However investigators have assessed the relative age-related cross sectional decline in running performance time (Grogan *et al.*, 1991; Fuchi *et al.*, 1989; Salthouse, 1976) and average power output maintained during a 2500 m rowing ergometer race (Seiler *et al.*, 1998). Data collected in the present study showed that the percentage rate of decline for average power maintained during a 16.1-km time trial was less than values reported by Selier *et al.* (1998) (8.0 vs 12.5% per decade, respectively), however performance time in the rowing race was about 10 minutes compared with an average time for the 16.1-km time trial of about 23 minutes. Grogan *et al.* (1991) found that a faster rate of performance decline associated with ageing occurred during shorter running distances of 5- and 10-km when compared with longer races such as the Marathon. The authors postulated that performance declined more quickly with age during 5- and 10-km races due to an age-related change in running economy and a decline in lean body mass and strength. In shorter distance events the relative contribution of energy from anaerobic sources is higher than long distance events (McArdle *et al.*, 1991), therefore it is reasonable to assume that an age-related decline in anaerobic capacity may have influenced these findings.

In the present study, age accounted for about 44% of the variance in average power output maintained by a heterogeneous group of cyclists during a 16.1-km time trial, therefore differences in performance can be attributed to inter individual variation in training habits and endurance capacity. There was no change in the relationship between age and time trial power output when performance power was expressed relative to body mass and age-

related changes in percentage body fat were not assessed in this part of the study. It is reasonable to assume that changes in body composition and a decline in fat free mass with age would have contributed to the decline in average power output.

Data collected in the present study revealed that the age-related cross sectional decline in average power output maintained during a laboratory based 16.1-km time trial was less than the age-related rate of decline for $King_{Wpeak}$. Although this difference did not reach the level of significance, data revealed a tendency for older cyclists to maintain a higher relative exercise intensity (expressed as $\%King_{Wpeak}$) compared with seniors. Allen *et al.* (1985) found that veteran runners were able to maintain a higher $\%\dot{V}O_{2max}$ at 10-km race pace when compared with performance matched senior competitors, however no study has assessed $\%W_{peak}$ maintained by veteran cyclists during time trial races.

Explanations for the finding that veterans can maintain a higher relative exercise intensity during endurance performance tests include the effects of an age-related reduction in anaerobic capacity (Chamari *et al.*, 1985) and a faster rate of decline in W_{max} due to age-related changes in maximal cardio-respiratory function (Aminoff *et al.*, 1996). It can be argued that veterans exercise at a higher $\%W_{max}$ due to a decline in W_{max} and not an improvement in physiological and morphological factors which influence sub-maximal race pace. The absolute rate of decline in rowing performance reported by Seiler *et al.* (1998) was higher than the rate of decline in 16.1-km time trial power assessed in the present study (3.25 vs 2.60 W per year, respectively). However, methodological issues concerning the validity and reproducibility of power recorded during cycling ergometery may have influenced the difference found between studies.

Methodological problems concerning the validity of power recorded using the Kingcycle have been discussed elsewhere (see chapter seven) and due to the random nature of the error in the Kingcycle identified previously there is no evidence to suggest that the age-related declines calculated for peak power and average performance power during the time trial were affected by this discrepancy. However the difference in Kingcycle power when compared with SRM power should be considered when comparisons are made within and between studies.

8.6.17 AGE-RELATED CHANGES IN AVERAGE HEART RATE DURING AN INDOOR 16.1-KM TIME TRIAL

There is no information available concerning the effect of age on average heart rate maintained during cycling endurance performance tests. Data collected in the present study revealed that the age-related decline in average heart rate maintained during a laboratory based 16.1-km time trial tended to be less than the age-related rate of decline calculated for peak heart rate, this difference was not significant. Investigators should be aware that veteran cyclists tend to maintain a higher relative workload (expressed as $\%W_{peak}$) and that during a 16.1-km time trial this age-related increase is not matched by a similar increase in relative heart rate response.

8.7 SUMMARY

Moderate negative correlations were found for age and $King_{Wpeak}$, HR_{peak} recorded during a Kingcycle peak power test. However the age related absolute decline in $King_{Wpeak}$ was influenced by the endurance performance ability of the subject group and the relative decline in $King_{Wpeak}$ was also influenced by body composition. The age related decline in $King_{Wpeak}$ and HR_{peak} was about 35-40 W and 7 $b \cdot min^{-1}$ per decade. Moderate negative correlations were also found for $\dot{V}O_{2peak}$, $\dot{V}CO_{2peak}$, $\dot{V}E_{peak}$ and BLa_5. Age related declines in these variables were estimated as 0.36, 0.46 and 7.5 $L \cdot min^{-1}$ and 1.2 $mmol \cdot L^{-1}$ per decade, respectively. A positive correlation was found for age and %BF which equated to an increase of 2.5% per decade. No correlation was found for age and peak values for breathing frequency, cycling economy and RER.

Moderate negative correlations were found for age and power at TLac and OBLA and age and HR at TLac and OBLA. Age related declines in these variables were calculated as 20 W and 6-7 $b \cdot min^{-1}$ per decade for both TLac and OBLA respectively. Moderate negative correlations were also found for age; and average power and heart rate recorded during a laboratory based 16.1-km cycling time trial. The age related decline in average power and heart rate during this event was estimated as 26 W and 6 $b \cdot min^{-1}$ per decade. Age related changes in relative exercise intensity determined at TLac and OBLA and exercise intensity maintained during an indoor time trial did not reach the level of significance however there was evidence to suggest that veteran athletes maintained a relatively higher workload

during cycling performance. Investigators need to be aware of age related declines in physiological variables associated with successful endurance performance when comparisons are made between age groups and when using normative values to assess relative exercise performance capacity.

CHAPTER 9

9 THE EFFECT OF AGE AND TESTING PROTOCOL ON THE DETERMINATION OF THRESHOLD EXERCISE INTENSITY

9.1 INTRODUCTION

Research has found that the assessment of a threshold level of exercise intensity can be affected by a multiple of factors such as diet, pedal cadence and testing protocol (see chapter four of this thesis) and the effect of age on blood lactate derived thresholds has been investigated in competitive cyclists (Massé-Biron *et al.*, 1992) however there is no information available concerning the interactive effect of age and testing method on the determination of blood lactate thresholds.

9.2 AIM OF STUDY 4

To gain new knowledge concerning methodological issues which affect the determination of threshold exercise intensity.

9.3 OBJECTIVES

9.3.1 PART 1

To compare the frequency and intensity of training between a group of seniors and veterans in order to select cyclists to participate in part 2 of the study.

9.3.2 PART 2

To investigate the interactive effect of age and testing protocol on the determination of threshold exercise intensity.

9.4 METHODS

9.4.1 PART 1

A questionnaire was compiled to compare the frequency and intensity of training between seniors and veterans in order to select cyclists to participate in part 2 of this study. Frequency of racing and training and the intensity of training/racing was assessed during a

9 month period. Twenty senior (35 ± 6 yr) and 20 veterans (57 ± 6 yr) who regularly competed in cycling time trial races completed a questionnaire (see appendices). This ascertained the number of races completed in one season and the type, frequency and intensity of cycle training/racing performed during the first week of June, September, December and March. During this period, cyclists maintained an activity diary and used a heart rate monitor (Polar, Kempele, Finland) during training and racing. Each subject performed a Kingcycle peak power test (PP) for the determination of maximum heart rate (HR_{peak}). Intensity of training/racing was based on 4 levels of heart rate response i) level 1, below 75% maximum heart rate (HR_{peak}) ii) low level 2, 75-80% HR_{peak} iii) high level 2, 80-85% HR_{peak} and iv) level 3, above 85% HR_{peak} (Westell, 1990).

9.4.2 PART 2

Subjects who participated in the study were all well trained cyclists who regularly competed in local cycling races. Six senior and six veteran cyclists were matched on level of training (frequency and relative intensity) using the information obtained from the training questionnaire. Subject characteristics are shown in Table 29.

Table 29. Mean ± SD and range of values for subject characteristics

	Seniors (n = 6)	Range	Veterans (n = 6)	Range
Age (yr)	28 ± 3	25-32	58 ± 4	52-63
Height (m)	1.86 ± 0.05	1.79-1.91	1.77 ± 0.04	1.73-1.84
Body mass (kg)	75.3 ± 4.0	71.0-82.5	76.8 ± 6.0	71.5-86.0
SRM_{Wpeak} (W)	444 ± 27	403-484	338 ± 26	304-368
HR_{peak} (b·min^{-1})	194 ± 4	191-202	167 ± 8	157-179
$\dot{V}O_{2peak}$ (L·min^{-1})	5.28 ± 0.17	5.06-5.46	4.18 ± 0.23	3.89-4.51

Each subject performed :- i) a peak aerobic capacity test (PAC) to determine SRM_{Wpeak}; ii) a blood lactate threshold test using a continuous ramped exercise protocol (LT_{ramp}); iii) a blood lactate threshold test using a continuous incremental protocol (LT_{inc}) and iv) a blood lactate threshold test using a discontinuous incremental protocol (LT_{dis}). Each test was randomly assigned and separated by at least one week. For each test, subjects used their

own bicycle fitted with SRM attached to a Kingcycle air braked ergometer. During all tests power was recorded using a SRM power meter.

9.4.2.1 PEAK AEROBIC CAPACITY TEST (PAC)

Each subject completed a Kingcycle peak aerobic capacity test as previously described in 7.4.2.

9.4.2.2 THRESHOLD TEST USING A RAMPED PROTOCOL (LT_{RAMP})

Following a five min warm-up at a power output equivalent to about 45% of $King_{Wpeak}$, subjects completed a continuous ramped exercise protocol as described by Davison *et al.* (1996). Starting power was ~ 45% $King_{Wpeak}$ and ramp rate was 6 $W \cdot min^{-1}$. During the test subjects selected their own pedal cadence ($rev \cdot min^{-1}$) and were allowed to change gear *ad libitum*. The test was completed when subjects reached volitional exhaustion.
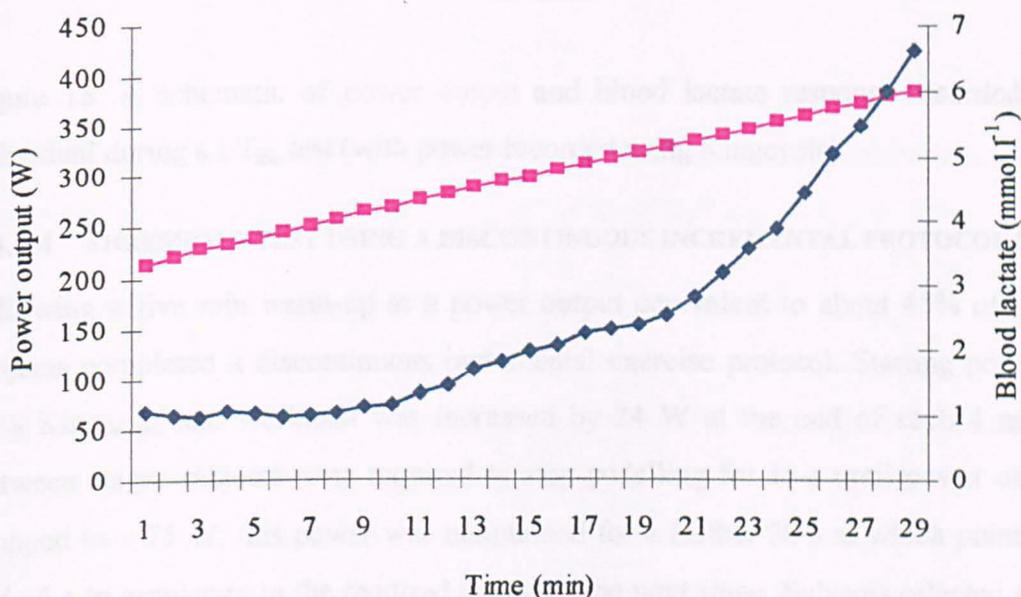
Figure 17. A schematic of power output and blood lactate response recorded for one individual during a LT_{ramp} test (with power recorded using Kingcycle)

9.4.2.3 THRESHOLD TEST USING A CONTINUOUS INCREMENTAL PROTOCOL (LT_{INC})

Following a 5 min warm-up at a power output of about 45% of $King_{Wpeak}$ subjects completed a continuous incremental exercise test. Starting power was ~45% $King_{Wpeak}$ and workload increased by 24 W at the end of each 4 minute stage. Subjects self-selected

pedal cadence (rev·min^{-1}) and changed gear ratio *ad libitum*. The test was completed when subjects reached volitional exhaustion.
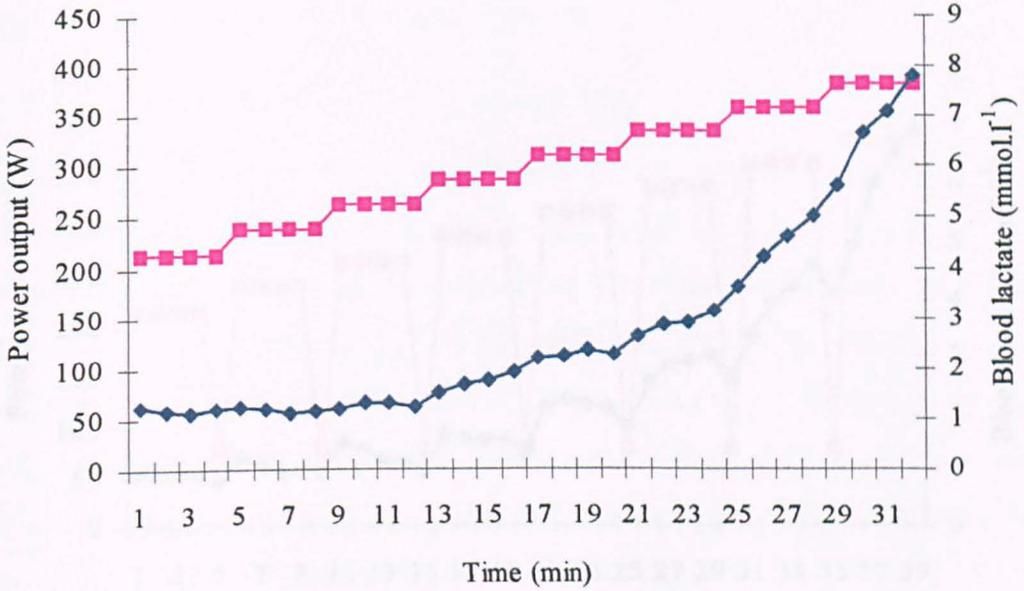
Figure 18. A schematic of power output and blood lactate response recorded for one individual during a LT_{inc} test (with power recorded using Kingcycle)

9.4.2.4 THRESHOLD TEST USING A DISCONTINUOUS INCREMENTAL PROTOCOL (LT_{DIS})

Following a five min warm-up at a power output equivalent to about 45% of $King_{Wpeak}$ subjects completed a discontinuous incremental exercise protocol. Starting power was ~ 45% $King_{Wpeak}$ and workload was increased by 24 W at the end of each 4 min stage. Between stages subjects were required to stop pedalling for 15-s until power output had dropped to ~ 75 W, this power was maintained for a further 30-s at which point subjects had 15-s to accelerate to the required power of the next stage. Subjects selected their own pedal cadence (rev·min^{-1}) and were allowed to change gear *ad libitum*. The test was completed when subjects reached volitional exhaustion.

9.4.2.5 BLOOD LACTATE DURING LT_{RAMP}, LT_{INC} AND LT_{DIS}

For the LT_{ramp}, LT_{inc} and LT_{dis}, tests fingertip capillary blood samples were collected at 1-min intervals from the start of the warm-up to completion of the test. Each sample of fingertip capillary whole blood was assayed for BLa (Biosen 5030L, EKF Industrie, Electronik GmbH, Barleben, Germany). Each blood sample was assayed (in duplicate) for

the concentration of BLa (mmol·L^{-1}) and the average value of the duplicate samples was used in all subsequent analyses.
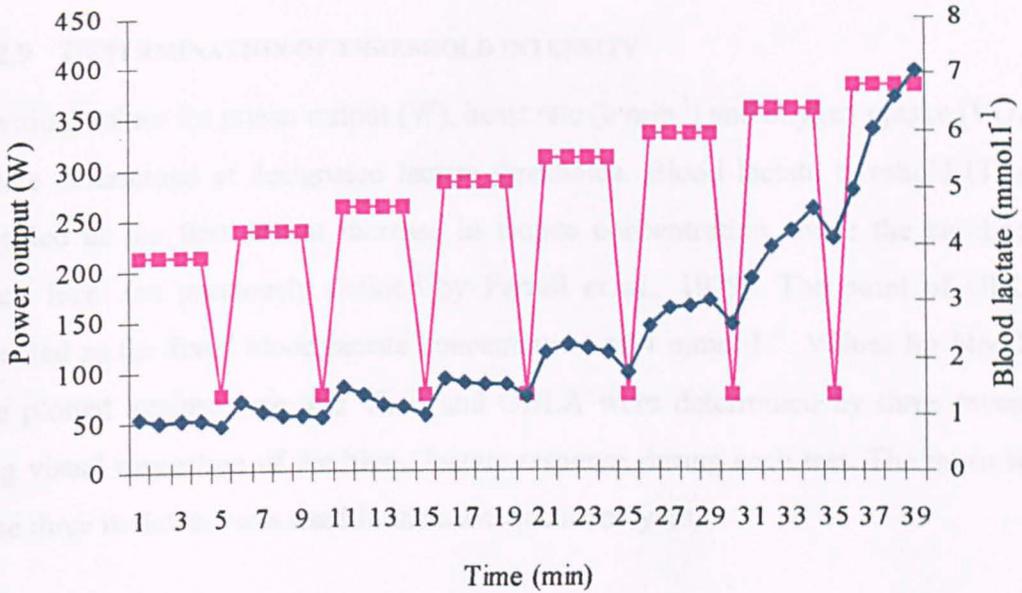
Figure 19. A schematic of power output and blood lactate response recorded for one individual during a LT_{dis} test (with power recorded using Kingcycle)

9.4.2.6 POWER AND CADENCE DURING LT_{RAMP}, LT_{INC} AND LT_{DIS}

Before testing, each subject reported to the laboratory with their racing bicycle. The chain-set of the bike was removed and replaced by either a 4 or 20 strain gauge torque transducer 'power meter' (SRM, Julich, Germany). The power meter was calibrated using the appropriate system software and calibration was checked before and after each test was completed. During the tests power output (W) and pedal cadence (rev·min^{-1}) were recorded at 1-s intervals and mean values were calculated for each min of the test. Each test was performed using a Kingcycle test rig and subjects were required to maintain the power output displayed on a computer screen using version 5.5 Kingcycle computer software.

9.4.2.7 HEART RATE DURING LT_{RAMP}, LT_{INC} AND LT_{DIS}

Heart rate (b·min^{-1}) was recorded at 5-s intervals (Polar, Kempele, Finland) throughout each test and mean values were calculated for each min.

9.4.2.8 EXPIRED GAS VARIABLES DURING LT$_{RAMP}$, LT$_{INC}$ AND LT$_{DIS}$

During LT$_{ramp}$, LT$_{inc}$ and LT$_{dis,}$ tests expired air was measured over one min intervals using the Covox online gas analysis system. Values for $\dot{V}O_2$, were recorded at one min intervals during the warm-up period and throughout the test.

9.4.2.9 DETERMINATION OF THRESHOLD INTENSITY

Individual values for power output (W), heart rate (b·min^{-1}) and oxygen uptake ($\dot{V}O_2$ L·min^{-1}) were determined at designated lactate thresholds. Blood lactate threshold (TLac) was identified as the first abrupt increase in lactate concentration above the baseline blood lactate level (as previously defined by Farrell *et al.*, 1979). The point of OBLA was identified as the fixed blood lactate concentration of 4 mmol·L^{-1}. Values for blood lactate were plotted against time and TLac and OBLA were determined by three investigators using visual inspection of the blood lactate response during each test. The mean selection of the three reviewers was used in the subsequent analyses.

9.4.3 STATISTICAL ANALYSES

Results are expressed as mean ± SD. Power output (W), heart rate (HR), oxygen uptake ($\dot{V}O_2$), blood lactate concentration (BLa) and pedal cadence (Rev) for each threshold were expressed as absolute values (W, b·min^{-1}, L·min^{-1}, mmol·L^{-1} and rev·min^{-1} respectively) and relative values (%SRM$_{Wpeak}$, %HR$_{peak}$ and %$\dot{V}O_{2peak}$ respectively). Data were checked using SPSS for violations of the assumptions for using parametric statistics. Analysis of variance (ANOVA) with one between subject factor (age) and one repeated measures factor (tests) were used to compare absolute values for blood lactate concentration at TLac and pedal cadence at TLac and OBLA and absolute and relative values for power, HR and $\dot{V}O_2$ at TLac and OBLA determined during LT$_{ramp}$, LT$_{inc}$ and LT$_{disc}$ tests. Level of significance was P<0.05 and post hoc comparisons were completed using Tukey HSD tests.

9.5 RESULTS

9.5.1 PART 1

Data from the questionnaire revealed no difference (P>0.05) between groups for the number of races completed, overall time spent cycling and the amount of training during the first week of June, Sept., Dec. and March. Training increased (P<0.05) in the veteran

group between the first week of December and March and although training intensity was similar between groups (P>0.05) veterans did tend (P = 0.16) to train more at a low intensity (i.e. below 75% HR_{peak}) when compared with seniors.

Table 30 Amount of time (mean ± S.D) veteran (n = 20) and senior (n = 20) cyclists trained during first week of June, September, December and March

Week	1st wk June (h:min)	1st wk Sept. (h:min)	1st wk Dec. (h:min	1st wk March (h:min)
seniors	6:56 ± 3:21	7:24 ± 2:44	7:47 ± 3:40	9:09 ± 3:19
veterans	7:32 ± 3:57	7:23 ± 3:28	6:35 ± 3:17	9:05 ± 3:46*

significantly higher (P<0.05) than 1st week of December in veteran cyclists
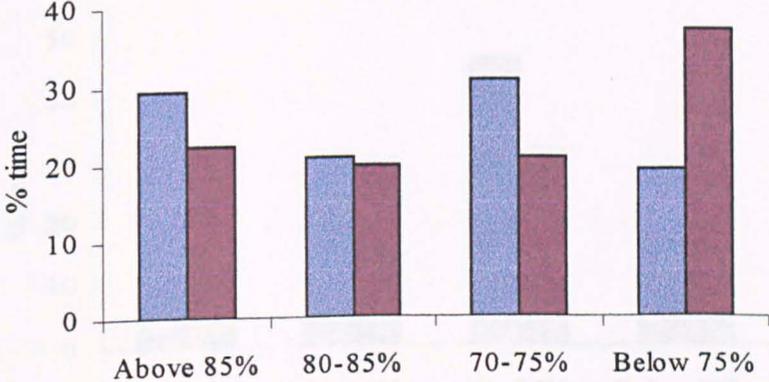
Figure 20. Percentage of mean overall time senior (n = 20) and veteran (n = 20) cyclists trained at each exercise intensity (%HR_{peak}) during the 1st week of June
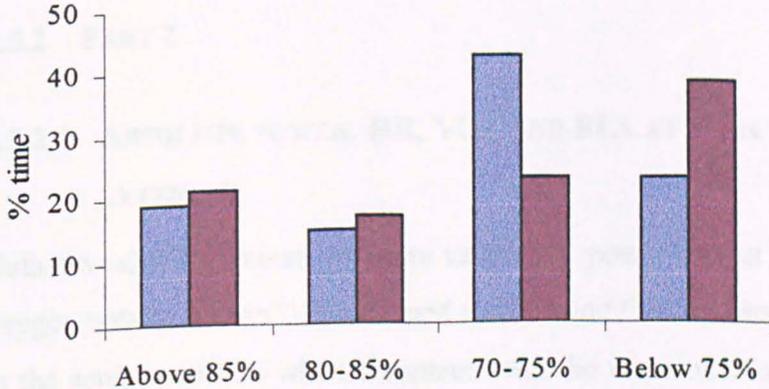
Figure 21. Percentage of mean overall time senior (n = 20) and veteran (n = 20) cyclists trained at each exercise intensity (%HR_{peak}) during the 1st week of September

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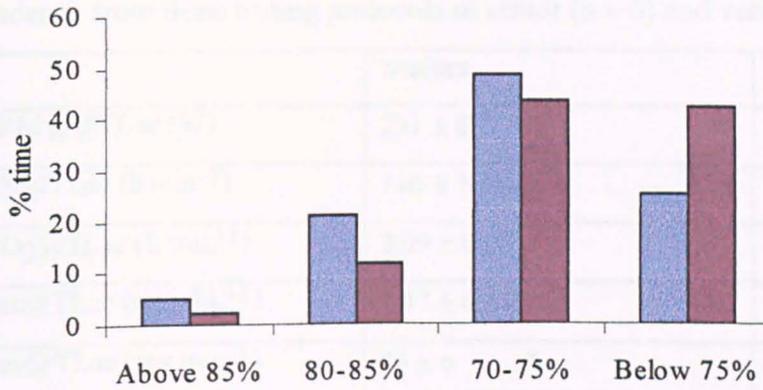
Figure 22. Percentage of mean overall time senior (n = 20) and veteran (n = 20) cyclists trained at each exercise intensity (%HR$_{peak}$) during the 1st week of December
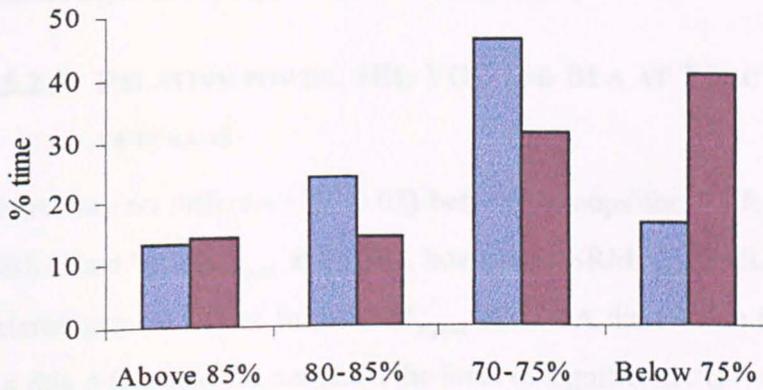
Figure 23. Percentage of mean overall time senior (n = 20) and veteran (n = 20) cyclists trained at each exercise intensity (%HR$_{peak}$) during the 1st week of March

9.5.2 PART 2

9.5.2.1 ABSOLUTE POWER, HR, VO$_2$, AND BLA AT TLAC AND OBLA IN SENIORS AND VETERANS

Data revealed that mean absolute values for power output (W), heart rate (b·min^{-1}) and oxygen uptake (L·min^{-1}) determined at TLac and OBLA were significantly higher (P<0.05) in the seniors cyclists when compared with the veterans. However no difference (P>0.05) was found between age groups for blood lactate concentration at TLac and cadence was higher (P<0.05) in the senior group at TLac but not OBLA (P = 0.15).

Table 31. Absolute values (mean ± SD) for power, heart rate, $\dot{V}O_2$ blood lactate and pedal cadence from three testing protocols in senior (n = 6) and veteran (n = 6) cyclists

	Seniors	Veterans
SRM$_W$@TLac (W)	231 ± 8 *	193 ± 22
HR@TLac (b·min^{-1})	140 ± 7 *	120 ± 10
$\dot{V}O_2$@TLac (L·min^{-1})	3.09 ± 0.35 *	2.60 ± 0.31
Lac@TLac (mmol·L^{-1})	1.47 ± 0.37	1.71 ± 0.49
Rev@TLac (rev·min^{-1})	96 ± 6 *	88 ± 5
SRM$_W$@OBLA (W)	309 ± 32 *	253 ± 21
HR@OBLA (b·min^{-1})	164 ± 7 *	142 ± 8
$\dot{V}O_2$@OBLA (L·min^{-1})	4.20 ± 0.48 *	3.47 ± 0.37
Rev@OBLA (rev·min^{-1})	97 ± 6	92 ± 6

denotes significantly higher than veterans (P<0.05)

9.5.2.2 RELATIVE POWER, HR, $\dot{V}O_2$, AND BLA AT TLAC AND OBLA IN SENIORS AND VETERANS

There was no difference (P>0.05) between groups for %HR$_{peak}$ and %$\dot{V}O_{2peak}$ at TLac and OBLA and %SRM$_{Wpeak}$ at OBLA, however %SRM$_{Wpeak}$ at TLac was higher (P<0.05) in the veteran group. Values for %SRM$_{Wpeak}$ at OBLA did tend to be higher in the veteran group but this difference did not reach the level of significance (P = 0.14).

Table 32. Relative values (mean ± SD) for power, heart rate and $\dot{V}O_2$ determined from the three testing protocols in senior (n = 6) and veteran (n = 6) cyclists

	Seniors	Veterans	P values	95%CI
%SRM$_{Wpeak}$@TLac	52.4 ± 3.3	56.7 ± 5.6	0.04 $	0.09-8.5
%HR$_{peak}$@TLac	71.9 ± 3.3	72.2 ± 5.9	0.92	
%$\dot{V}O_{2peak}$@TLac	58.5 ± 6.0	62.1 ± 6.7	0.27	
%SRM$_{Wpeak}$@OBLA	69.7 ± 4.5	74.4 ± 5.9	0.14	-1.8-11.2
%HR$_{peak}$@OBLA	84.2 ± 2.2	85.4 ± 5.6	0.62	
%$\dot{V}O_{2peak}$@OBLA	79.6 ± 8.1	83.1 ± 9.2	0.51	

$ denotes significant difference between seniors and veterans (P<0.05)

95%CI - 95% confidence intervals for difference in the mean of senior and veteran groups

Table 33. P values for post hoc comparisons across LT_{ramp}, LT_{inc}, LT_{dis} tests

	LT_{ramp} - LT_{inc}	LT_{ramp} - LT_{dis}	LT_{inc} - LT_{dis}
%SRM$_{Wpeak}$@TLac	0.02 *	0.01 *	0.99
%HR$_{peak}$@TLac	0.02 *	0.01 *	0.94
%VO$_{2peak}$@TLac	0.04 *	0.13	0.82
%SRM$_{Wpeak}$@OBLA	0.08	0.89	0.03 *
%HR$_{peak}$@OBLA	0.04 *	0.01 *	0.29
%VO$_{2peak}$@OBLA	0.01 *	0.04 *	0.001 *

denotes significant difference (P<0.05)

Table 34. Mean ± SD values for power output (W), heart rate (b·min^{-1}), $\dot{V}O_2$ (L·min^{-1}), blood lactate concentration (mmol·L^{-1}) and pedal cadence (rev·min^{-1}) at TLac (n = 12)

	LT_{ramp}		LT_{inc}		LT_{dis}	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
SRM$_W$@TLac	224 ± 29 *#	174-260	207 ± 30	168-252	206 ± 21	174-235
HR@TLac	135 ± 13 *#	109-152	128 ± 14	106-151	127 ± 11	110-146
$\dot{V}O_2$@TLac	2.97 ± 0.46 *	2.11- 3.91	2.76 ± 0.46	1.94-3.54	2.81 ± 0.30	2.31-3.31
BLa@TLac	1.66 ± 0.55	1.02-2.85	1.53 ± 0.38	0.90-2.3	1.58 ± 0.41	1.01-2.42
Rev@TLac	94 ± 8	79-105	91 ± 7	79-105	91 ± 6	81-105

denotes significantly higher than LT_{inc} (P<0.05)

# denotes significantly higher than LT_{dis} (P<0.05)

9.5.2.3 ABSOLUTE VALUES AT TLAC FOR COMBINED GROUP

Mean values (n = 12) for power output (W), heart rate (b·min^{-1}), oxygen uptake (L·min^{-1}) and blood lactate concentration (mmol·L^{-1}) at TLac determined from the three LT tests are shown in Table 34. There was no difference (P>0.05) for oxygen uptake and blood lactate concentration recorded for each test. Power output at TLac (SRM$_W$@TLac) and heart rate at TLac (HR@TLac) for LT_{ramp} was higher (P<0.05) when compared with LT_{inc} and LT_{dis}. There was no difference between tests (P = 0.13) for pedal cadence (Rev@TLac).

9.5.2.4 ABSOLUTE VALUES AT OBLA FOR COMBINED GROUP

Mean values (n = 12) for power output (W), HR (b·min^{-1}) and oxygen uptake (L·min^{-1}) determined at OBLA for the three LT tests are shown in Table 35. Values for power at OBLA (SRM$_W$@OBLA) were higher (P<0.05) for the LT_{ramp} and LT_{dis} tests when

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compared with LT_{inc} and heart rate at OBLA (HR@OBLA) was higher (P<0.05) for LT_{ramp} than LT_{inc} and LT_{dis}. Oxygen uptake ($\dot{V}O_2$@OBLA) was higher (P<0.05) for LT_{dis} than LT_{ramp} and LT_{inc} and $\dot{V}O_2$@OBLA for LT_{ramp} was higher than LT_{inc}. There was no difference between tests (P = 0.13) for pedal cadence (Rev@OBLA).

Table 35. Mean ± SD values for power output (W), heart rate (b·min⁻¹), oxygen uptake (L·min⁻¹) and pedal cadence (rev·min⁻¹) at OBLA (n = 12)

	LT_{ramp}		LT_{inc}		LT_{dis}	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
SRM_W@OBLA	283 ± 40 *	225-354	274 ± 37	219-348	285 ± 42 *	229-351
HR@OBLA	156 ± 13 * #	135-179	152 ± 13	134-172	151 ± 14	128-171
$\dot{V}O_2$@OBLA	3.85 ± 0.62 *	2.80-4.80	3.73 ± 0.55	2.80-4.63	3.94 ± 0.55 * $	3.20-4.90
Rev@OBLA	95 ± 6	83-105	96 ± 7	86-111	93 ± 8	81-104

denotes significantly higher than LT_{inc} (P<0.05)

# denotes significantly higher than LT_{dis} (P<0.05)

$ denotes significantly higher than LT_{ramp} (P<0.05)

9.5.2.5 RELATIVE VALUES AT TLAC FOR COMBINED GROUP

Mean values (n = 12) for power output (%SRM_{Wpeak}), heart rate (%HR_{peak}) and oxygen uptake (%$\dot{V}O_{2peak}$) at TLac for the three LT tests are shown in Table 36. There was no difference (P>0.05) in %$\dot{V}O_{2peak}$ calculated for each test. Power at TLac (%SRM_{Wpeak}) and heart rate at TLac (%HR_{peak}) were higher (P<0.05) for LT_{ramp} when compared with LT_{inc} and LT_{dis} was higher (P<0.05) for LT_{ramp} when compared with LT_{inc} but not LT_{dis}.

Table 36. Mean ± SD values for power output (%SRM_{Wpeak}), heart rate (%HR_{peak}), oxygen uptake (%$\dot{V}O_{2peak}$) at TLac (n = 12)

	LT_{ramp}		LT_{inc}		LT_{dis}	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
%SRM_{Wpeak}@TLac	57.5 ± 4.3 *#	50.0-63.4	53.1 ± 5.2	48.4-66.4	53.2 ± 4.5	46.6-61.8
%HR_{peak}@TLac	74.9 ± 5.0 *#	64.1-83.0	70.8 ± 4.5	62.4-77.8	70.5 ± 3.3	66.5-79.0
%$\dot{V}O_{2peak}$@TLac	62.9 ± 7.6 *	51.6-73.3	58.3 ± 5.8	47.4-65.8	59.7 ± 5.8	47.8-69.1

denotes significantly higher than LT_{inc} (P<0.05)

# denotes significantly higher than LT_{dis} (P<0.05)

9.5.2.6 RELATIVE VALUES AT OBLA FOR COMBINED GROUP

Mean values (n = 12) for power output ($\%SRM_{Wpeak}$), heart rate ($\%HR_{peak}$) and oxygen uptake ($\%\dot{V}O_{2peak}$) at OBLA for the three LT tests are shown in Table 37. Power ($\%SRM_{Wpeak}$) was higher (P<0.05) for LT_{ramp} and LT_{dis} when compared with LT_{inc} however $\%SRM_{Wpeak}$ for LT_{ramp} and LT_{dis} were similar (P>0.05). Heart rate ($\%HR_{peak}$) was higher (P<0.05) for LT_{ramp} when compared with LT_{inc} and LT_{dis}. Oxygen uptake ($\%\dot{V}O_{2peak}$) was higher (P<0.05) for LT_{ramp} and LT_{dis} when compared with LT_{inc} and $\%HR_{peak}$ for LT_{dis} was higher (P<0.05) than LT_{ramp}

Table 37. Mean ± SD values for power output ($\%W_{peak}$), heart rate ($\%HR_{peak}$), oxygen uptake ($\%\dot{V}O_{2peak}$) at OBLA (n = 12)

	LT_{ramp}		LT_{inc}		LT_{dis}	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
$\%SRM_{Wpeak}$@OBLA	72.7 ± 6.1 *	65.3-82.9	70.4 ± 6.0	59.8-81.9	73.1 ± 4.9 *	64.3-80.3
$\%HR_{peak}$ @OBLA	86.3 ± 4.6 * #	79.4-98.1	84.6 ± 3.9	78.8-93.6	83.5 ± 4.0	75.3-92.4
$\%\dot{V}O_{2peak}$ @OBLA	81.5 ± 9.4 *	66.0-99.0	79.0 ± 8.5	65.8-96.7	83.5 ± 8.6 * $	65.2-100.0

* denotes significantly higher than LT_{inc} (P<0.05)

# denotes significantly higher than LT_{dis} (P<0.05)

$ denotes significantly higher than LT_{ramp} (P<0.05)

Table 38. P values calculated for relative comparisons between age groups (seniors vs veterans), within the three tests (LT_{ramp}, LT_{inc}, LT_{dis}) and the interaction between age and tests

	Test	Interaction
$\%SRM_{Wpeak}$@TLac	0.009 *	0.89
$\%HR_{peak}$@TLac	0.005 *	0.59
$\%\dot{V}O_{2peak}$@TLac	0.03 *	0.11
$\%SRM_{Wpeak}$@OBLA	0.03 *	0.17
$\%HR_{peak}$@OBLA	0.002 *	0.65
$\%\dot{V}O_{2peak}$@OBLA	0.0001 *	0.21

* denotes significant difference within tests (P<0.05)

Mean values of senior (n = 6) and veterans (n = 6) for power output (W), HR (b·min^{-1}), oxygen uptake (L·min^{-1}) and blood lactate concentration (mmol·L^{-1}) at TLac for the three LT tests are shown in Table 39. There was no interaction between age and tests for power, heart rate, oxygen uptake, blood lactate concentration and pedal cadence (see Table 38).

Table 39. Mean ± SD values for power output (W), heart rate (b·min^{-1}), oxygen uptake (L·min^{-1}), blood lactate concentration (mmol·L^{-1}) and pedal cadence (rev·min^{-1}) at TLac for seniors (n = 6) and veterans (n = 6)

		LT$_{ramp}$		LT$_{inc}$		LT$_{dis}$	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
SRM$_W$@TLac	sens	245 ± 17	217-260	226 ± 20	204-252	223 ± 11	209-235
	vets	203 ± 22	174-226	187 ± 26	168-239	189 ± 15	174-214
HR@TLac	sens	145 ± 7	136-152	139 ± 7	129-151	136 ± 6	130-146
	vets	126 ± 11	109-142	117 ± 9	106-130	118 ± 8	110-131
V̇O$_2$@TLac	sens	3.21 ± 0.44	2.75-3.91	3.09 ± 0.34	2.78-3.54	2.98 ± 0.30	2.57-3.31
	vets	2.72 ± 0.36	2.11-3.20	2.43 ± 0.31	1.94-2.90	2.63 ± 0.23	2.31-2.98
BLa@TLac	sens	1.56 ± 0.50	1.02-2.33	1.49 ± 0.32	1.00-1.79	1.35 ± 0.26	1.01-1.76
	vets	1.76 ± 0.63	1.21-2.85	1.56 ± 0.47	0.90-2.3	1.81 ± 0.42	1.11-2.42
Rev@TLac	sens	99 ± 6	92-105	94 ± 7	86-105	94 ± 7	88-105
	vets	89 ± 6	79-94	87 ± 6	79-96	88 ± 4	81-92

9.5.2.8 ABSOLUTE VALUES AT OBLA FOR SENIORS AND VETERANS

Mean values of senior (n = 6) and veterans (n = 6) for power output (W), HR (b·min^{-1}) and oxygen uptake (L·min^{-1}) at OBLA for the three LT tests are shown in Table 40. There was no interaction between age and tests for power, heart rate, oxygen uptake, blood lactate concentration and pedal cadence(see Table 38).

9.5.2.9 RELATIVE VALUES AT TLAC FOR SENIORS AND VETERANS

Mean values of senior (n = 6) and veterans (n = 6) for power output (%SRM$_{Wpeak}$), heart rate (%HR$_{peak}$) and oxygen uptake (%V̇O$_{2peak}$) at TLac for the three LT tests are shown in Table 41. There was no interaction between age and tests for %SRM$_{Wpeak}$, %HR$_{peak}$ and %V̇O$_{2peak}$ (see Table 38).

Table 40. Mean ± SD values for power output (W), heart rate (b·min⁻¹), oxygen uptake (L·min⁻¹) and pedal cadence (rev·min⁻¹) at OBLA for seniors (n = 6) and veterans (n = 6)

		LT_{ramp}		LT_{inc}		LT_{dis}	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
SRM_w@OBLA	sens	312 ± 30	275-354	297 ± 35	253-348	317 ± 32	272-351
	vets	254 ± 24	225-283	251 ± 21	219-282	253 ± 20	229-279
HR@OBLA	sens	166 ± 7	160-179	163 ± 7	155-172	162 ± 7	155-171
	vets	145 ± 6	135-154	142 ± 8	134-155	140 ± 6	128-151
$\dot{V}O_2$@OBLA	sens	4.25 ± 0.54	3.55-4.80	4.09 ± 0.44	3.54-4.63	4.28 ± 0.52	3.51-4.90
	vets	3.45 ± 0.42	2.80-3.85	3.37 ± 0.41	2.80-3.80	3.59 ± 0.31	3.20-3.89
Rev@OBLA	sens	98 ± 4	93-105	99 ± 7	93-111	95 ± 8	81-104
	vets	93 ± 8	83-102	92 ± 5	86-98	91 ± 7	81-100

9.5.2.10 RELATIVE VALUES AT OBLA FOR SENIORS AND VETERANS

Mean values of senior (n = 6) and veterans (n = 6) for power output (%SRM_{Wpeak}), heart rate (%HR_{peak}) and oxygen uptake (%$\dot{V}O_{2peak}$) at OBLA for the three LT tests are shown in Table 42. There was no interaction between age and tests for %SRM_{Wpeak}, %HR_{peak} and %$\dot{V}O_{2peak}$ (see Table 38).

Table 41. Mean ± SD values for power output (%SRM_{Wpeak}), heart rate (%HR_{peak}), oxygen uptake (%$\dot{V}O_{2peak}$) at TLac for seniors (n = 6) and veterans (n = 6)

		LT_{ramp}		LT_{inc}		LT_{dis}	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
%SRM_{Wpeak}@TLac	sens	55.4 ± 2.3	52.5-58.4	51.2 ± 2.4	48.4-55.6	50.6 ± 3.2	46.6-55.6
	vets	59.6 ± 4.9	50.0-63.4	55.0 ± 6.7	49.2-66.4	55.7 ± 4.2	50.0-61.8
%HR_{peak}@TLac	sens	74.4 ± 3.0	71.2-78.4	71.5 ± 3.5	67.5-77.8	69.9 ± 1.9	67.7-72.3
	vets	75.4 ± 6.8	64.1-83.0	70.2 ± 5.6	62.4-74.8	71.1 ± 4.5	56.5-69.2
%$\dot{V}O_{2peak}$@TLac	sens	60.7 ± 7.2	54.3-72.7	58.5 ± 5.4	52.4-65.8	56.4 ± 5.3	47.8-61.5
	vets	65.1 ± 7.8	51.6-73.3	58.1 ± 6.7	47.4-65.8	63.0 ± 4.6	66.5-79.0

Table 42. Mean ± SD values for power output (%SRM$_{Wpeak}$), heart rate (%HR$_{peak}$), oxygen uptake (%$\dot{V}O_{2peak}$) at OBLA for seniors (n = 6) and veterans (n = 6)

		LT$_{ramp}$		LT$_{inc}$		LT$_{dis}$	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
%SRM$_{Wpeak}$@OBLA	sens	70.4 ± 3.9	65.3-74.2	67.1 ± 4.5	59.8-71.9	71.7 ± 4.3	64.3-77.5
	vets	74.9 ± 7.5	66.4-82.9	73.7 ± 5.7	67.6-81.9	74.5 ± 5.4	67.8-80.3
%HR$_{peak}$@OBLA	sens	85.4 ± 1.8	83.8-88.6	84.0 ± 2.3	81.2-86.6	83.2 ± 2.2	80.7-86.1
	vets	87.0 ± 6.4	79.4-98.1	85.1 ± 5.2	78.8-93.6	83.8 ± 5.5	75.3-92.4
%$\dot{V}O_{2peak}$@OBLA	sens	80.4 ± 9.1	66.0-88.9	77.4 ± 7.2	65.8-84.8	81.0 ± 9.0	65.2-89.7
	vets	82.6 ± 10.3	68.5-99.0	80.7 ± 10.0	68.5-96.7	86.1 ± 8.1	78.2-100

9.6 DISCUSSION

9.6.1 PART 1

Although veterans raced and trained as often as younger cyclists, seniors did tend to maintain a higher relative exercise intensity during training, however differences between groups did not reach the level of significance due to the large inter-individual variation in training regimen. There is no published information available concerning age-related differences in training between senior and veteran racing cyclists.

9.6.2 PART 2

Exercise intensity associated with a blood lactate threshold has been used by numerous investigators to predict the performance ability of endurance cyclists (for review see Coyle, 1995). Notably only one study has compared blood lactate derived assessments in senior and veteran cyclists (Massé-Biron et al., 1992). In the present study %SRM$_{Wpeak}$ at TLac determined for the six seniors was lower than the value calculated by Maassen and Busse (1989) (53 vs 63% respectively) and %HR$_{peak}$ at TLac for the seniors was higher than the value reported by Yoshida (1984) for eight healthy male students (72 vs 58% respectively). Although Iredale and Nimmo (1997) calculated that TLac occurred at 70% $\dot{V}O_{2max}$ in untrained subjects, in the present investigation TLac occurred at about 59% $\dot{V}O_{2peak}$ in the seniors. This was markedly higher than values reported by Massé-Biron et al. (1992), Yoshida (1984) and Yoshida et al. (1987) for senior aged non cyclists (49, 36 and 40%, respectively).

Mean $\%VO_{2peak}$ at TLac in the six veteran cyclists was higher in the present study when compared with the mean value calculated by Massé-Biron *et al.* (1992) (62 vs 56%, respectively) and lower than the value reported by Iredale and Nimmo (1997) for untrained older men (62 vs 83%, respectively). In the present study, mean values for the seniors $\%SRM_{Wpeak}$, $\%HR_{peak}$ and $\%\dot{V}O_{2peak}$ at OBLA for the three methods of testing (see Table 32) were very similar to values reported by Yoshida (1984) for $\%W_{max}$, $\%HR_{max}$ and $\%\dot{V}O_{2max}$ (70 vs 72%, 84 vs 84% and 80 vs 78%, respectively). Interestingly the testing protocol used by Yoshida and co-authors was a 25 W, 1 min incremental test with arterial blood sampled at 1 min intervals. Therefore 1 min samples may have afforded a more accurate determination of absolute values at threshold. Unfortunately no other study has assessed absolute and relative values for power at TLac and power, HR and $\dot{V}O_2$ at OBLA in veteran cyclists.

Relative values calculated for power, HR and $\dot{V}O_2$ at threshold were dependent on the validity and reproducibility of the method used to assess peak and threshold values. Although methodological problems associated with subject selection are frequently encountered when using cross sectional methods of analysis, there appears to be no longitudinal data available concerning the effect of age on changes in blood lactate derived thresholds.

9.6.2.1 EFFECT OF AGE ON THRESHOLDS DURING LT_{RAMP}, LT_{INC} AND LT_{DIS} TESTS

Absolute values for power, HR and $\dot{V}O_2$ at TLac and OBLA were higher in the seniors. This finding is not surprising considering the age-related declines in power, HR and $\dot{V}O_2$ reported in study three of the present thesis. No difference was found between seniors and veterans for blood lactate concentration at TLac, this finding contradicts data reported by Massé-Biron *et al.* (1992) who found that lactate concentration at threshold was significantly lower in veteran cyclists. The finding that BLa at TLac was not influenced by the age of the subject concurs with data presented in study three of this thesis which revealed no age-related cross-sectional decline in blood lactate concentration at TLac in a heterogeneous group of 38 cyclists. Factors such as habitual physical activity/training status and absolute age (Iredale and Nimmo, 1997) appear to have a direct influence on blood lactate concentration recorded during progressive exercise tests, however few studies

have investigated these factors in veteran cyclists (Massé-Biron et al., 1992). All of the participants in the present study were competitive endurance cyclists and therefore levels of habitual physical activity were relatively high. It is difficult to assess whether age-related declines in lactacidaemia are due to external factors such as reduced physical activity or part of the ageing process per se. Unfortunately, no study has investigated the longitudinal relationship between age and blood lactate concentration at TLac in competitive cyclists.

When mean values for power, HR and $\dot{V}O_2$ at TLac and OBLA were expressed as relative values (%W_{peak}, %HR_{peak}, and %$\dot{V}O_{2peak}$) no differences were found between senior and veterans except for %W_{peak} at TLac. This finding agrees with Massé-Biron et al. (1992) who calculated that relative values for power at threshold were higher in veterans. Previous studies (Allen et al., 1985; Iredale and Nimmo, 1997) have also recorded higher values for %HR_{max} and %$\dot{V}O_{2max}$ at TLac in veteran runners and elderly subjects respectively, in the present study there was no difference between age groups for %HR_{peak} and %$\dot{V}O_{2peak}$ at TLac.

Relative values for power, HR and $\dot{V}O_2$ calculated for seniors and veterans are dependent on peak values achieved during the Kingcycle PAC test. Values for %W_{peak}, HR_{peak} and %$\dot{V}O_{2peak}$ at threshold in veteran groups may reflect the decline in peak values and not an age-related increase in relative exercise intensity at threshold. The validity of cross sectional research is strongly dependent on subject selection and therefore values reported in other investigations (Allen et al., 1985; Iredale and Nimmo, 1997; Massé-Biron et al., 1992) may be representative of the group specific decline in peak values and not age-related differences in threshold exercise intensity. This would explain the finding of Iredale and Nimmo (1997) that %$\dot{V}O_{2max}$ at TLac in a group of untrained elderly men was considerably higher than values reported for older trained athletes (Massé-Biron et al., 1997). Cross sectional studies by Coggan et al. (1992) and Proctor et al. (1995) have found that oxidative enzyme capacity was higher in veteran athletes when compared with untrained age matched individuals and Proctor et al. (1995) found that veteran athletes had a significantly higher percentage of type I muscle fibres when compared with age matched untrained subjects. Muscle respiratory capacity and fibre type are strongly related to

absolute and relative oxygen uptake at threshold (Ivy et al., 1980) therefore the relatively high $\%\dot{V}O_{2max}$ at TLac reported by Iredale and Nimmo (1997) may have been an artefact of an augmented decline in $\dot{V}O_{2max}$.

Ribeiro et al. (1986) commented that a faster loading rate during an incremental exercise test would result in a higher power at threshold due to the time dependent efflux of lactate from muscle to blood. In the present study there was no difference in ramp rate between age groups however relative loading rate (expressed as $\%W_{peak}$) was faster in the veteran group and a faster loading rate did correspond with a higher relative power at TLac in the veteran group. Further investigation is warranted concerning the interactive effect of loading rate and age on power at threshold.

Data presented in study three of this thesis revealed that cross sectional age-related declines in power and HR at TLac were not significantly different to the declines in SRM_{Wpeak} and HR_{peak}. Mean relative value for power at OBLA did tend to occur at a higher percentage of SRM_{Wpeak} in the veterans however this difference did not reach the level of significance. Data presented in study three revealed a large range of inter-individual variation in SRM_{Wpeak}, power at TLac and OBLA in a heterogeneous group of cyclists (n = 38). This factor should be considered when subjects are selected to participate in cross sectional research. In the present study six senior and six veteran cyclists were matched on frequency and intensity of training but not level of performance. In the cross sectional study of Massé-Biron et al. (1992) seven veteran cyclists and seven young physical education students were not matched on level of training and/or level of performance. In the study by Massé-Biron et al. (1992) $\%\dot{V}O_{2max}$ at TLac was significantly higher in the veterans group when compared with senior aged cyclists (56 vs 46%, respectively).

Massé-Biron et al. (1992) reported a cross sectional age-related decline in lactacidaemia and suggested that a decrease in lactate production could explain this finding. Age-related differences in blood lactate kinetics have been reported on several occasions (Allen et al., 1985; Belman and Gaesser, 1991; Iredale and Nimmo, 1997; Massé-Biron et al., 1992; Tzankoff and Norris, 1979). The present study found that power at TLac occurred at a higher $\%W_{peak}$ in veteran cyclists. Age-related declines in lactate production have been

attributed to a decline in ß-adrenergic receptor sensitivity and epinephrine sensitivity (Fleg *et al.*, 1985) and declines in these variables would relate to a decreased muscle glycogen breakdown and lower blood lactate concentration during exercise (Crowley *et al.*, 1996). Alterations in the size and number of type II relative to type I muscle fibres have also been recorded in elderly populations (Proctor *et al.*, 1995) and veteran performers (Coggan *et al.*, 1992). A proportional increase in type I oxidative fibres with a decrease in type II glycolytic fibres with age would be associated with lower levels of blood lactate during exercise and a higher relative TLac (Ivy *et al.*, 1980).

Lactate threshold is related to muscle oxidative capacity (Rusko *et al.* 1980) and the percentage of type I muscle fibres (Coyle *et al.*, 1988; Ivy *et al.*, 1980). These variables were not assessed in the present study, however higher values for oxidative enzyme activity and lower values for lactate dehydrogenase have been recorded in veteran athletes (Coggan *et al.*, 1990). The attainment of a high relative value for power at threshold can be attributed to training induced increases in oxidative capacity (Holloszy *et al.*, 1984). Veteran cyclists who participated in the present study were regularly competing in cycling time trial races and maintained high levels of training before and during the testing period therefore higher levels of oxidative enzyme activity and lower values for lactate dehydrogenase may have been present in this group.

Massé-Biron *et al.* (1992) found that low trained veteran performers were unable to attain an exercise intensity which corresponded with OBLA. In the present study veteran cyclists were able to attain OBLA during each method of testing. One explanation for this concerns the age of the subject and that veterans tested by Massé-Biron *et al.* (1992) were older than the cyclists in the present study (mean age 65 vs 58 year, respectively). Research has highlighted an accelerated reduction in muscle fibre number and area beyond the age of 60-65 yr (Lexell *et al.*, 1988) and several authors have found strong relationships between muscle structure and function and the occurrence of a blood lactate threshold (Coyle *et al.*, 1988; Ivy *et al.*, 1980). Therefore age-related changes in these factors could explain the difference between studies. Another explanation for the decline in lactacidaemia recorded by Massé-Biron *et al.* (1992) concerned the method used to assess blood lactate concentration. Massé-Biron and co-authors assessed blood lactate using venous whole

blood samples (antecubital vein) and in the present study fingertip arterialised whole blood samples were analysed. El-Sayed *et al.* (1993a; 1993b) and Flohr *et al.* (1996) found that venous whole blood lactate (antecubital vein) during progressive incremental exercise was significantly lower than capillary whole blood (fingertip) and the difference between values increased in proportion to work intensity. Furthermore Yeh *et al.* (1983) calculated that the rise in venous blood lactate lagged behind the rise in arterial lactate by 1.5 mins. It is likely that veteran cyclists in the study of Massé-Biron *et al.* (1992) were unable to attain OBLA workload due to the assessment of BLa using venous samples. Investigators need to be aware that fixed blood lactate thresholds (Allen *et al.*, 1985; Massé-Biron *et al.*, 1992) are dependent on both the testing protocol used to identify threshold and the method of analysis used to measure blood lactate concentration.

9.6.2.2 EFFECT OF PROTOCOL ON THRESHOLDS DURING LT_{RAMP}, LT_{INC} AND LT_{DIS} TESTS

Values for power, HR and $\dot{V}O_2$ at TLac and OBLA were affected by the method of testing. This finding is in agreement with several investigations (Hughson and Green, 1982; MacFarlane *et al.*, 1983; Ribeiro *et al.*, 1986; Smith *et al.*, 1997; Watts *et al.*, 1998; Whipp *et al.*, 1974; Yoshida, 1984) which found similar values determined at threshold were also test dependent. Absolute values for power, HR and $\dot{V}O_2$ at TLac were significantly higher during the ramp test, however no difference was found between incremental and discontinuous tests. This finding has important implications when selecting an appropriate test to determine TLac intensity.

The effect of 1 and 4 min blood sampling rates on the determination of threshold was not assessed in the present study, however precision of measurement is an important consideration when assessing performance variables (Hopkins *et al.*, 1999). Stockhausen *et al.* (1997) calculated that an increment of 30 W between stages required an exercise duration of at least 4 min for blood lactate concentration to achieve a quasi-steady state. In the present study, work load increased by 24 W per 4 min. During the ramp protocol, blood lactate concentration did not attain a quasi-steady state and therefore power associated with a sudden increase in blood lactate (TLac) or fixed concentration (OBLA) may have been overestimated during this method of testing. Weltman *et al.* (1990) found that values for $\dot{V}O_2$ at TLac during incremental and discontinuous tests were similar, this finding agrees

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with data in the present investigation. However Weltman *et al.* (1990) did not compare ramp, incremental and discontinuous protocols. In the present study there was no difference between ramp and discontinuous tests for $\dot{V}O_2$ at OBLA however there was a difference between incremental and discontinuous tests. It is not surprising that oxygen uptake recorded during the 'steady-state' incremental test was lower than ramp and discontinuous protocols due to the continuously increasing workload of the ramp and extra work required to attain the designated power of each increment following the rest period during the discontinuous test.

Endurance athletes use heart rate response to monitor racing/training intensity (Jeukendrup and van Diemen, 1998; Lambert *et al.*, 1998) and set levels of training (Westell, 1990). If lactate derived thresholds are used to prescribe training intensity in competitive cyclists investigators need to be aware that a ramp testing protocol can affect heart rate at TLac. Notably, $\%HR_{peak}$ at TLac and OBLA during the ramp test were higher than values for incremental and discontinuous tests but there was no difference between incremental and discontinuous tests.

Studies which have investigated the effect of testing protocol on blood lactate derived thresholds have typically used a fixed pedal cadence ($rev\cdot min^{-1}$) throughout the test (Ribeiro *et al.*, 1986; Smith *et al.*, 1997; Yoshida, 1984). In the present study subjects self-selected pedal cadence during each test and a significant difference was found between age groups for mean overall pedal cadence for the three methods of testing however there was no difference in pedal rate across testing protocols. Atkinson and Lloyd (1998) found that during laboratory based tests spontaneously chosen pedal cadence was lower in veteran cyclists when compared with seniors and suggested that the difference may have involved a 'selective age-dependent change in muscle fibre types' (p. 36) however further research is warranted to establish whether there is a physiological rationale for this finding.

There is no information available concerning the effect of age on spontaneously chosen pedal rates during exercise tests. In the present study, mean age-related difference in pedal cadence at TLac was 8 $rev\cdot min^{-1}$. Buchanan and Weltman (1985) found no difference for power at TLac and OBLA when subjects maintained pedal rates of 90 and 120 $rev\cdot min^{-1}$

but power at threshold was lower when 120 rev·min^{-1} was compared with 60 rev·min^{-1}. In the study by Hughes *et al.* (1982) power at TLac was significantly higher with a faster pedal rate however the difference in pedal rates was 40 rev·min^{-1}. In comparison to these studies mean difference in pedal rate recorded in the present study was considerably less (8 rev·min^{-1}) therefore the effect of this small difference in pedal cadence was probably minimal. No difference was found between groups for pedal cadence at OBLA, although higher values were recorded in the seniors (P = 0.15). Mean pedal rates across TLac and OBLA parameters were similar in the seniors, however an increase was observed from TLac to OBLA in the veterans. Therefore spontaneously chosen pedal rate appeared to be influenced by intensity of exercise in the older cyclists but not the seniors.

9.6.2.3 INTERACTION OF AGE & PROTOCOL ON THRESHOLDS DURING LT$_{RAMP}$, LT$_{INC}$ & LT$_{DIS}$ TESTS

No other study has investigated the interaction between age and test when using blood lactate derived assessments of TLac and OBLA. No interactive difference was found between age and test for power, HR, $\dot{V}O_2$ and pedal cadence at TLac and OBLA thresholds. This finding indicated that differences between testing protocols were independent of the age of the rider. Consequently comparisons across tests could be completed using the combined group of cyclists. However the finding that power output at TLac differed between age groups has important implications for the prescription of competitive and training exercise intensity using fixed percentages of W$_{peak}$.

9.7 SUMMARY

This study investigated the effect of age and testing protocol on blood lactate derived thresholds. Although absolute values for power, HR and $\dot{V}O_2$ at thresholds were higher in seniors there was no difference between age groups for blood lactate at TLac. Pedal cadence was similar between age groups at OBLA but not at TLac. Relative values for %SRM$_{Wpeak}$, %HR$_{peak}$ and %$\dot{V}O_{2peak}$ were similar between groups except for %SRM$_{Wpeak}$ at TLac. Consequently, age of the rider may need to be considered when evaluating variables associated with TLac. Testing protocol affected power, HR and oxygen uptake at threshold exercise intensity but not blood lactate concentration and pedal cadence. There was no interactive difference between age and test, therefore the affects of testing method were

independent of age. These findings have important implications when selecting an appropriate blood lactate derived threshold and testing method for the assessment of cycling performance in senior and veteran cyclists.

CHAPTER 10

10 THE EFFECT OF AGE ON LABORATORY AND FIELD BASED CYCLING PERFORMANCE

10.1 INTRODUCTION

There is very little information available concerning blood lactate, cardio-respiratory and performance related responses of veteran athletes during laboratory and field based endurance performance trials and no study has assessed performance related responses of veteran cyclists. Laboratory based assessments of power, oxygen uptake, heart rate and blood lactate response are frequently used to assess and predict cycling endurance performance capacity and ability. However due to technical difficulties inherent in collecting data in the field there is very little information available concerning physiological responses of cyclists during real competition.

With the development of new technology it is now possible to record power output and blood lactate data in the field. There is no information available concerning the blood lactate response of veterans during actual field based cycling trials, however several studies have assessed the blood lactate response of senior cyclists during laboratory (El-Sayed *et al.,* 1997; McNaughton *et al.,* 1999; Neary *et al.,* 1999; Potteiger *et al.,* 1995) and field based (Hoogeveen, and Schep, 1997; Nichols *et al.,* 1997) competitive performance. To my knowledge, only one study has considered the effect of age on blood lactate response during prolonged high intensity exercise (Overend *et al.* 1992) therefore more information is required concerning the effects of age on blood lactate response during actual or simulated cycling competition.

10.2 AIM OF STUDY 5

To provide a clearer understanding of the determinants of laboratory and field based cycling performance and the applicability of a laboratory based simulated time trial to a real world setting.

10.3 OBJECTIVE

To compare blood lactate and performance related responses of senior and veteran cyclists during a laboratory and field based 16.1-km time trial.

10.4 METHODS

For each test subjects used their own racing bicycle (fitted with SRM). During laboratory tests the bicycle was attached to a Kingcycle air braked ergometer (EDS Portaprompt Ltd, High Wycombe, UK). Laboratory tests were conducted at the same time of day and environmental conditions were maintained during testing (ambient temperature 18-22° C, relative humidity 45-55%). During tests gear ratio and pedal cadence (rev·min^{-1}) were self selected. Throughout each test, power output (W) was averaged at 1-s intervals by either a 4 or 20 strain gauge SRM (pedaling torque inductively transmitted at 500 kHz).

10.4.1 SUBJECTS

Subjects who participated in this part of the study were selected using information obtained from a pre-test training questionnaire as described in 9.5.1. Senior and veteran cyclists were matched on frequency and intensity of training, but not performance. Seven senior (28 ± 3 yr) and seven veteran (58 ± 4 yr) well trained male cyclists completed 3 tests; i) a maximum aerobic power ramp test (PAC), ii) a laboratory based 16.1-km time trial (LTT) and iii), a field based 16.1-km time trial (FTT).

Before testing, static and dynamic lung volumes and capacities (see Table 43) were assessed for each subject. Vital capacity (VC), forced vital capacity (FVC), and maximum voluntary ventilation (MVV) were measured using an electronic spirometer (Vitalograph compact, Buckingham, England) and peak expiratory flow (PEF) was recorded using a peak flow meter (Wright, UK). All measurements were taken with the subject standing in an upright position.

During the time trial, 2 subjects competed against each other and were required to complete the 16.1-km distance as fast as possible. During PAC and LTT tests, power was recorded at 1-s intervals using either a 4 or 20 strain gauge power meter (SRM), heart rate was determined every 5-s (Polar) and respiratory gases were measured every 1 min (Covox).

Fingertip capillary blood samples were taken at 2.5 min intervals during LTT to assess blood lactate concentration (Biosen 5030L, Magdeburg, Germany). During each test, power output and pedal cadence were recorded continuously. On completion of LTT and FTT, a fingertip capillary blood sample was collected for 5 min at 1 min intervals for the analysis of blood lactate concentration (Biosen 5030L, Magdeburg, Germany). Percentage body fat (%BF) and fat free mass (kg) were assessed using the methods previously outlined.

10.4.2 STATISTICAL ANALYSES

Level of significance was set at $P<0.05$ and post hoc comparisons were completed using Tukey HSD tests. Statistical analyses were completed using Microsoft Excel (Bellevue, WA), Minitab (State College, PA) and Statistica Mac 5.1 (Statsoft, Inc). Values in the text for standard error of estimate (SEE) are for the 95% confidence interval and the mean ($\pm$ SD) unless otherwise stated. Comparisons between age groups were completed using independent t-tests and analysis of variance (ANOVA) with one between subject factor for age and repeated measures factors for either test and/or time. Values for economy were calculated as average power divided by average $\dot{V}O_2$ recorded during the time trial.

10.5 RESULTS

Table 43. Mean $\pm$ SD values for static and dynamic lung volumes and capacities in senior (n = 6) and veteran (n = 6) cyclists

	Seniors		Veterans	
	Mean $\pm$ SD	Range	Mean $\pm$ SD	Range
Vital Capacity (L)	6.88 ± 0.68	6.23-7.83	5.40 ± 0.69 $	4.27-6.35
Forced vital capacity (L)	6.88 ± 0.69	6.31-7.78	5.37 ± 0.69 $	4.60-6.44
$FEV_{1.0}$ (L)	5.12 ± 0.56	4.51-5.73	3.97 ± 0.55 $	3.09-4.81
$FEV_{1.0}$-to-FVC ratio	75 ± 6	70-85	74 ± 7	65-84
PEF (L·min^{-1})	614 ± 24	575-640	603 ± 63	520-705
MVV (L·min^{-1})	197 ± 44	137-257	176 ± 26	138-204

PEF   - peak expiratory flow
MVV   - maximum voluntary ventilation
$ denotes significantly lower than seniors (P<0.05)

Mean values for power, $\dot{V}O_2$, $\dot{V}_E$ and HR recorded during the indoor 16.1-km time trial ride were higher (P<0.05) in the seniors (see Table 44) and mean time to complete the indoor time trial was lower (P<0.05) in the seniors (20:17 ± 0:29 vs 23:00 ± 0:29 min:s, for seniors and veterans respectively). Assessment of static and dynamic lung volumes revealed that mean values for VC, FVC and $FEV_{1.0}$ were lower (P<0.05) in the veterans. However there was no difference (P>0.05) between groups when dynamic lung capacities ($FEV_{1.0}$, $FEV_{1.0}$-to-FVC ratio, PEF, MVV) were compared.

Table 44. Mean ± SD for peak variables recorded during a Kingcycle PAC test and absolute and relative values for average variables measured during an indoor 16.1-km time trial in seniors and veteran cyclists

	Seniors (n = 6)	Veterans (n = 6)
W_{peak} (W)	444 ± 27 *	339 ± 26
W_{LTT} (W)	344 ± 20 *	256 ± 22
%W_{peak}	77.5 ± 1.4	75.8 ± 4.2
$\dot{V}O_{2peak}$ ($L\cdot min^{-1}$)	5.28 ± 0.17 *	4.11 ± 0.32
$\dot{V}O_{2LTT}$ ($L\cdot min^{-1}$)	4.67 ± 0.31 *	3.66 ± 0.34
%$\dot{V}O_{2peak}$	88.4 ± 3.5	89.2 ± 5.0
$\dot{V}_{Epeak}$ ($L\cdot min^{-1}$ STPD)	159.2 ± 6.7 *	133.0 ± 16.3
$\dot{V}_{ELTT}$ ($L\cdot min^{-1}$ STPD)	136.8 ± 8.6 *	112.0 ± 20.1
%$\dot{V}_{Epeak}$	85.7 ± 6.3	84.2 ± 5.1
HR_{peak} ($b\cdot min^{-1}$)	193 ± 6 *	162 ± 8
HR_{LTT} ($b\cdot min^{-1}$)	177 ± 6 *	149 ± 8
%HR_{peak}	91.6 ± 1.8	92.2 ± 3.6
Breathing frequency ($breath\cdot min^{-1}$)	56 ± 8 *	45 ± 4
RER	1.09 ± 0.03	1.07 ± 0.04

* denotes significantly higher than veterans (P<0.05)

Analysis of mean %W_{peak} at 2.5 min intervals during the 16.1-km ride showed there was no difference (P>0.05) between groups except for the first 2.5 min when the seniors maintained a significantly higher %W_{peak}. Absolute values for power, $\dot{V}O_2$, $\dot{V}_E$, HR and breathing frequency were higher in the seniors (P<0.05) however there was no difference between groups for RER (P = 0.32). Mean values for power, $\dot{V}O_2$, $\dot{V}_E$ and HR expressed as

percentage of W_{peak}, VO_{2peak}, V_{Epeak} and HR_{peak} were similar between groups (P = 0.39, 0.73, 0.54 and 0.77 respectively). There was no difference (P>0.05) between groups in mean BLa during the time trial race, however BLa did tend (P = 0.08) to be higher in the seniors. Although blood lactate increased (P<0.05) in both groups during the time trial, there was no further change (P>0.05) from 7.5 min of the ride (see Figure 24).
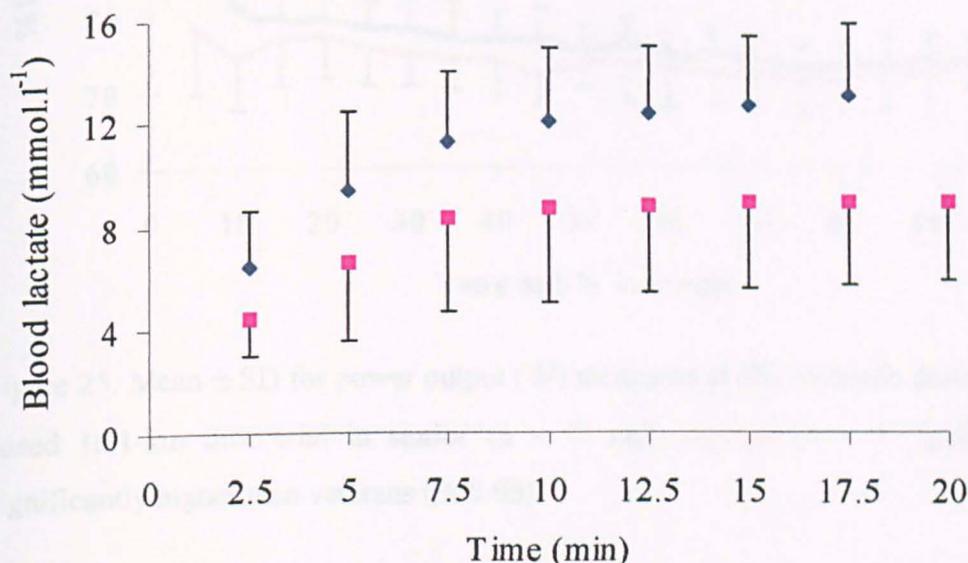
Figure 24. Mean ± SD for blood lactate (mmol·L^{-1}) measured at 2.5 min intervals during a laboratory based 16.1-km time trial in **senior** (n = 6) and **veteran** (n = 6) cyclists

Table 45. Mean ± SD for economy calculated from average power and VO_2 recorded during a laboratory based 16.1-km time trial in senior (n = 6) and veteran (n = 6) cyclists

Economy	seniors	veterans	P (95% CI)
[W·VO$_2$(L·min^{-1})]	73.19 ± 3.26	70.08 ± 2.76	0.02 (0.6-5.6)
[W·VO$_2$(mL·kg^{-1}·min^{-1}]	5.51 ± 0.20	5.39 ± 0.50	0.66 (-0.5-0.8)
[W·VO$_2$(mL·kg$^{-0.67}$·min^{-1})]	13.23 ± 0.40	12.85 ± 0.90	0.41 (-0.7-1.5)
[W·VO$_2$(mL·kgFFM^{-1}·min^{-1})]	4.86 ± 0.23	4.27 ± 0.30	0.02 (0.1-1.1)
[W·VO$_2$(mL·kgFFM$^{-0.67}$·min^{-1})	12.17 ± 0.46	10.99 ± 0.58	0.02 (0.3-2.0)

Mean value for economy expressed as [W·VO$_2$(L·min^{-1})] [W·VO$_2$(mL·kgFFM^{-1}·min^{-1})] and [W·VO$_2$(mL·kgFFM$^{-0.67}$·min^{-1}) was higher in the seniors (see Table 45) however there was

no difference between age groups when economy was expressed as $[W \cdot VO_2(mL \cdot kg^{-1} \cdot min^{-1})]$ and $[W \cdot VO_2(mL \cdot kg^{-0.67} \cdot min^{-1})]$.
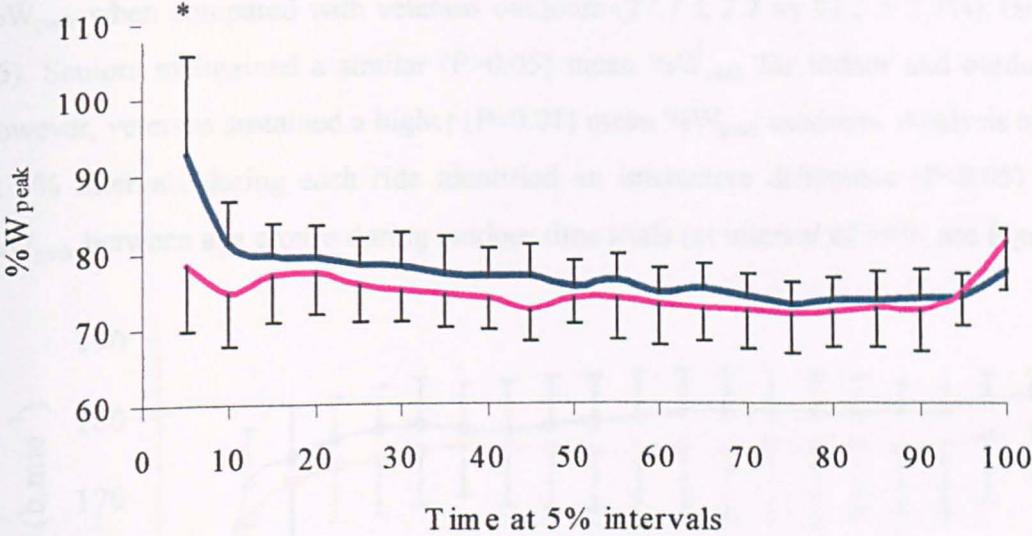
Figure 25. Mean ± SD for power output (W) measured at 5% intervals during a laboratory based 16.1-km time trial in **senior** (n = 7) and **veteran** (n = 7) cyclists * denotes significantly higher than veterans (P<0.05)
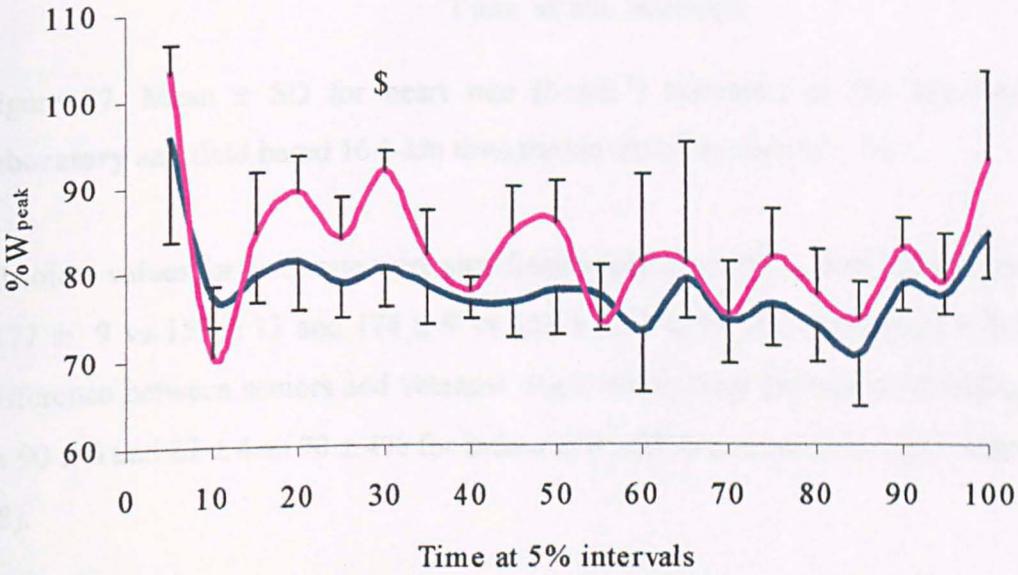
Figure 26. Mean ± SD for power output (W) measured at 5% intervals during a field based 16.1-km time trial in **senior** (n = 7) and **veteran** (n = 7) cyclists. $ denotes significantly higher than seniors (P<0.05)

There was no difference (P>0.05) in $\%W_{peak}$ between seniors and veterans during LTT (76.5 ± 2.3 vs $74.9 \pm 4.5\%$, see Figure 25). During FTT, seniors sustained a lower (P<0.01) $\%W_{peak}$ when compared with veterans outdoors (77.7 ± 2.7 vs $82.2 \pm 2.7\%$), (see Figure 26). Seniors maintained a similar (P>0.05) mean $\%W_{peak}$ for indoor and outdoor trials, however, veterans sustained a higher (P<0.01) mean $\%W_{peak}$ outdoors. Analysis of $\%W_{peak}$ at 5% intervals during each ride identified an interactive difference (P<0.05) in mean $\%W_{peak}$ between age groups during outdoor time trials (at interval of 30%, see Figure 26).
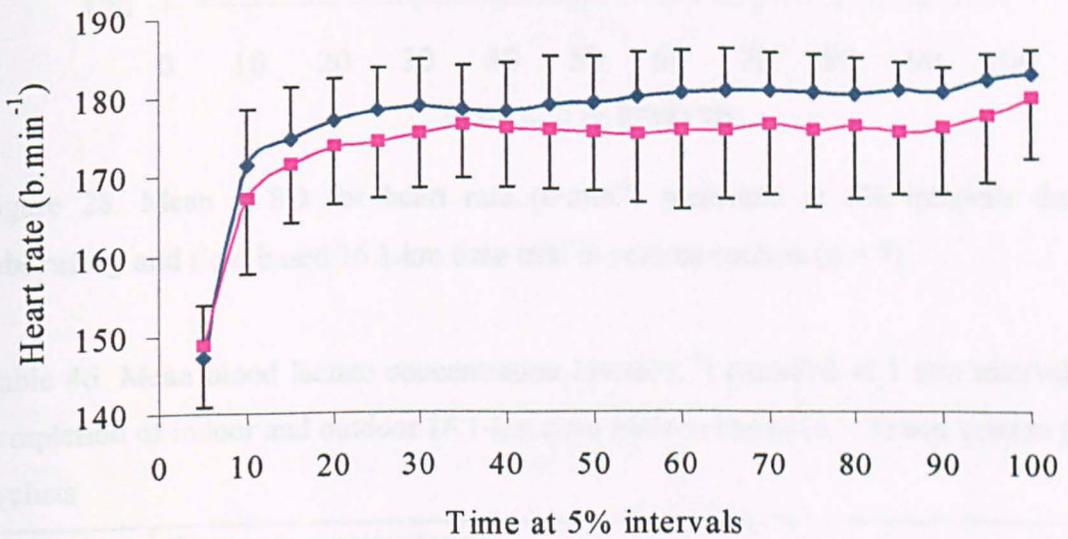
Figure 27. Mean $\pm$ SD for heart rate ($b \cdot min^{-1}$) measured at 5% intervals during a **laboratory** and **field** based 16.1-km time trial in senior cyclists (n = 7)

Absolute values for heart rate were significantly higher in the seniors indoors and outdoors (177 ± 9 vs 153 ± 13 and 174 ± 9 vs 152 ± 12 $b \cdot min^{-1}$ respectively), but there was no difference between seniors and veterans when values were expressed as $\%HR_{peak}$ (91 ± 4 vs 90 ± 6 and 89 ± 4 vs $90 \pm 4\%$ for indoor and outdoor, respectively) (see Figures 27 and 28).

Comparison between age groups for mean BLa of five blood samples taken at 1 min intervals (see Table 46) revealed no difference (P>0.05) between seniors and veterans on completion of the field based time trial ride (10.91 ± 1.98 vs 9.57 ± 2.53 $mmol \cdot L^{-1}$) however mean BLa was higher (P<0.01) in the senior group on completion of the laboratory based ride (12.73 ± 2.90 vs 8.34 ± 2.17 $mmol \cdot L^{-1}$).
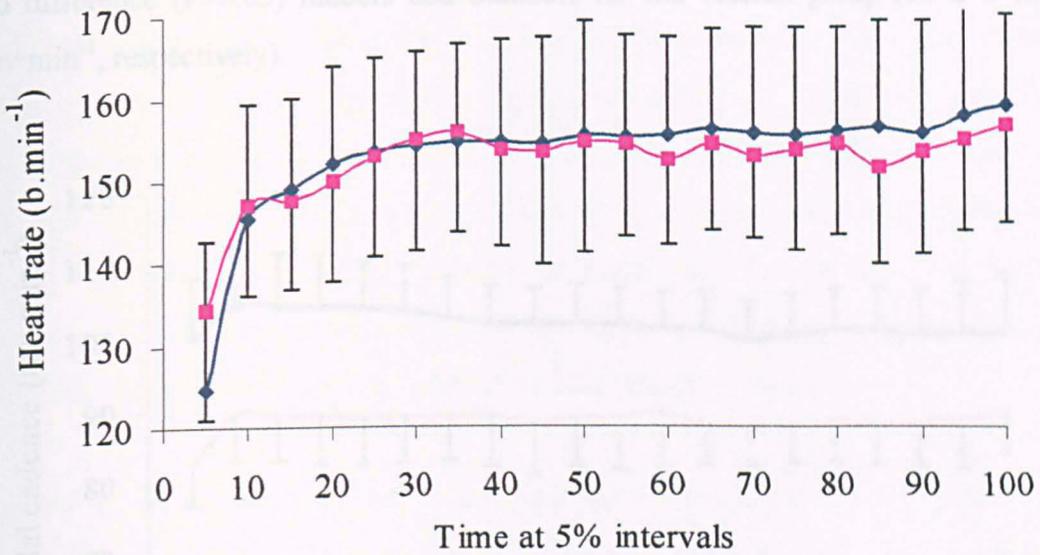
Figure 28. Mean ± SD for heart rate (b·min^{-1}) measured at 5% intervals during a **laboratory** and **field** based 16.1-km time trial in veteran cyclists (n = 7)

Table 46. Mean blood lactate concentration (mmol·L^{-1}) recorded at 1 min intervals post completion of indoor and outdoor 16.1-km time trials in senior (n = 7) and veteran (n = 7) cyclists

	Time post exercise (min)				
	1	2	3	4	5
sens (L)	13.52 ± 2.80	12.88 ± 3.13	12.68 ± 3.01	12.42 ± 3.03	12.14 ± 3.23
sens (F)	11.66 ± 1.86	11.21 ± 1.95	10.86 ± 2.15	10.56 ± 2.12	10.26 ± 2.08
vets (L)	8.99 ± 2.51	8.61 ± 2.35	8.31 ± 2.24	8.00 ± 2.12	7.81 ± 2.10
vets (F)	10.34 ± 2.76	9.88 ± 2.57	9.54 ± 2.63	9.27 ± 2.69	8.81 ± 2.50

L = laboratory based time trial

F = field based time trial

Mean pedal cadence was higher (P<0.001) in the seniors indoors (101 ± 6 vs 88 ± 6 rev·min^{-1}) however there was no difference (P>0.05) between groups for mean pedal cadence outdoors (86 ± 4 vs 84 ± 4 rev·min^{-1} for seniors and veterans respectively), (see Figures 29 and 30). Mean pedal cadence recorded for the seniors was higher (P<0.001) indoors compared with outdoors (101 ± 6 vs 86 ± 4 rev·min^{-1}, respectively) but there was

no difference (P>0.05) indoors and outdoors for the veteran group (88 ± 6 vs 84 ± 4 rev·min^{-1}, respectively).
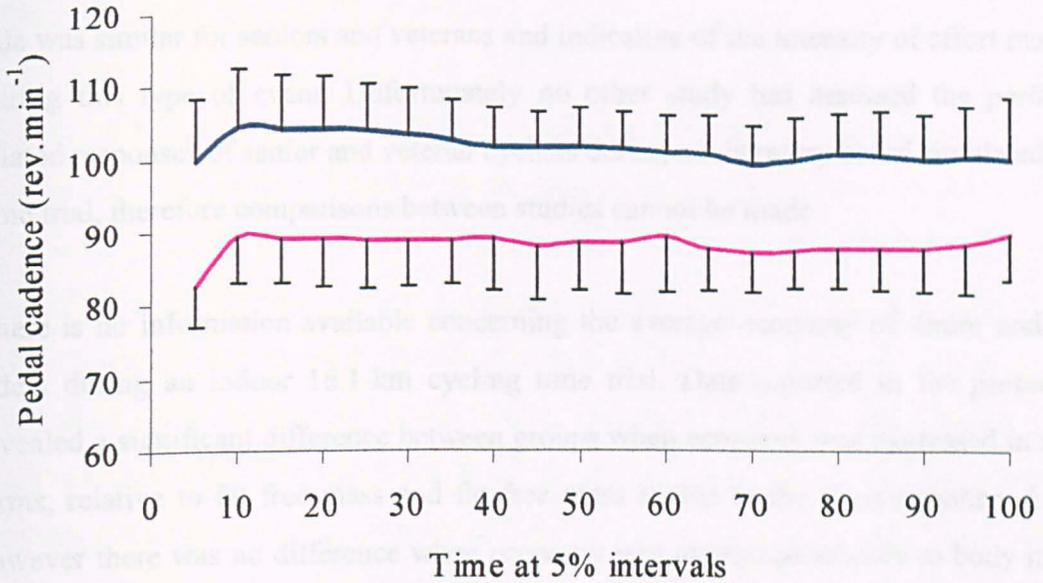
Figure 29. Mean ± SD for pedal cadence (rev·min^{-1}) measured at 5% intervals during a laboratory based 16.1-km time trial in **senior** (n = 7) and **veteran** (n = 7) cyclists
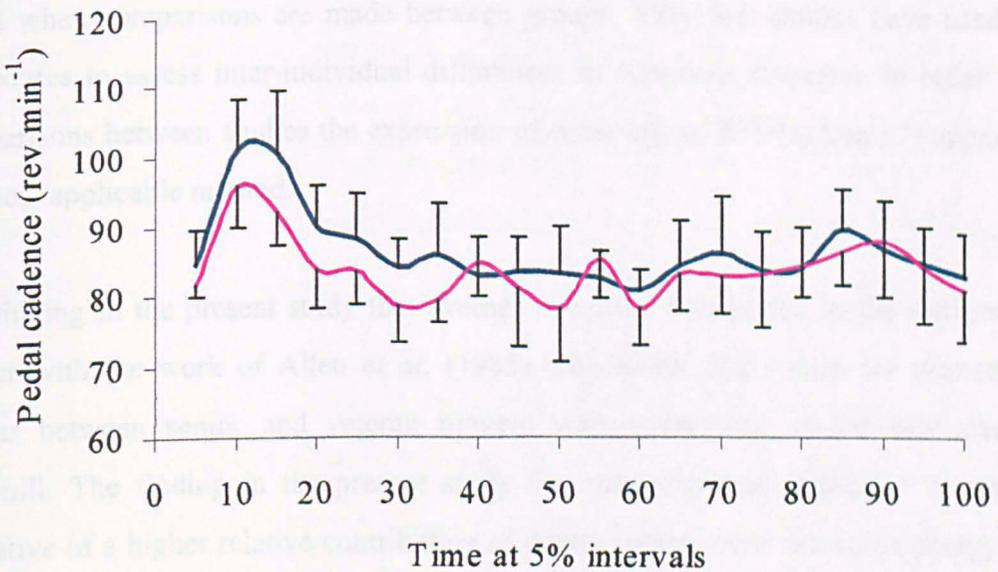
Figure 30. Mean ± SD for pedal cadence (rev·min^{-1}) measured at 5% intervals during a field based 16.1-km time trial in **senior** (n = 7) and **veteran** (n = 7) cyclists

10.6 DISCUSSION

Although absolute values for power, $\dot{V}O_2$, and HR were higher in the seniors there was no difference between groups for relative exercise intensity ($\%W_{peak}$, $\%\dot{V}O_{2peak}$, and $\%HR_{peak}$) during the indoor time trial. An important finding was that mean RER recorded during the ride was similar for seniors and veterans and indicative of the intensity of effort maintained during this type of event. Unfortunately no other study has assessed the performance related responses of senior and veteran cyclists during a laboratory based simulated cycling time trial, therefore comparisons between studies cannot be made.

There is no information available concerning the average economy of senior and veteran riders during an indoor 16.1-km cycling time trial. Data reported in the present study revealed a significant difference between groups when economy was expressed in absolute terms, relative to fat free mass and fat free mass scaled to the mass exponent $k = 0.67$, however there was no difference when economy was expressed relative to body mass and body mass scaled to the mass exponent of $k = 0.67$. This finding concurs with previous work which has used $W \cdot \dot{V}O_2(L \cdot min^{-1})$ to compare measures of economy between groups of cyclists (for review see Coyle, 1995). However this finding also highlights how scaling procedures can affect the interpretation of data and investigators need to be aware of this effect when comparisons are made between groups. Very few studies have used scaling procedures to assess inter-individual differences in economy, therefore in order to make comparisons between studies the expression of economy as $W \cdot \dot{V}O_2(L \cdot min^{-1})$ appears to be the most applicable method.

The finding in the present study that average economy was higher in the seniors did not concur with the work of Allen *et al.* (1985) who found that values for economy were similar between senior and veteran runners who maintained 10-km race pace on a treadmill. The finding in the present study that economy was higher in the seniors is indicative of a higher relative contribution of power output from anaerobic energy sources and/or the ability produce more power without a concomitant increase in oxygen consumption. Coyle (1995) proposed that trained cyclists were able to produce more power for an equivalent $\dot{V}O_2$ due to a greater recruitment of muscle fibres and found that elite competitors had a higher percentage of type I muscle fibres within their vastus lateralis

muscle. However, it is reasonable to assume that a higher percentage of type I muscle fibres was present within the veteran group due to the combined effects of ageing and endurance training on alterations in muscle fibre composition. Further research is required to investigate the relationship between muscle fibre content and endurance performance during a 16.1-km cycling time trial race.

Data for static lung volumes revealed a significant age-related reduction in vital capacity and forced vital capacity however there was no difference between senior and veteran cyclists for dynamic measures of lung function and capacity. Hagberg *et al.* (1988) found no difference in maximum voluntary ventilation (MVV) between veteran runners and age matched sedentary individuals. However, values for MVV recorded in older subjects were lower than values recorded for senior runners and young sedentary subjects. In the present study no difference was found for MVV in cyclists matched on training intensity and frequency. The effect of training on the age-related decline in dynamic lung capacity is unclear however Yerg *et al.* (1985) found that MVV was higher in veteran athletes when compared with age matched sedentary individuals. In the present study MVV and PEF recorded in the veteran group were indicative of their training status and provided data to support the postulate that habitual physical activity can attenuate age-related declines in dynamic lung function. Hagberg *et al.* (1988) argued that training attenuated the decline in VO_{2max} more than static and dynamic lung performance. However, in a recent longitudinal study by Pollock *et al.* (1997) the authors reported no change in vital capacity in veteran runners who maintained high and moderate training regimens for twenty years. Further investigation is warranted to study the relationship between training load (frequency and intensity) and age related changes in static volumes and dynamic lung function.

Relative ventilation ($\%\dot{V}_{Epeak}$) maintained during the indoor time trial was similar between groups. However breathing frequency and absolute values for ventilation were significantly higher in the seniors. During the indoor time trials expired air was analysed for O_2 and CO_2 content using the Covox analysis system, with ventilation measured on inspiration. Several veteran cyclists commented that inspiration through the Covox system affected their performance. Although it could be argued that during the indoor time trial both groups of subjects were required to breathe through the Covox system, Johnson and

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Dempsey (1991) speculated that during prolonged high intensity exercise (when the veteran athlete is required to sustain high ventilatory responses) the metabolic cost of breathing may be higher due to the effects of an increase in physiological dead space and an increased stiffness of the chest wall. This could lead to a redistribution of blood away from locomotor muscles to fulfil the relatively higher demand of O_2 required by the respiratory muscles during heavy work, this postulate could explain the lower economy recorded for the veteran group during the 16.1-km time trial of the present study. The authors also suggested that this 'steal effect' would not change $\dot{V}O_2$ but would result in a lower work rate. This could also explain the lower relative power of the veterans recorded during the indoor time trial of the present study. The hypothesis of Johnson and Dempsey (1991) is based on research concerning maximal exercise performance and the effects of ventilatory response on maximum oxygen uptake and power, unfortunately no study has applied this theory to prolonged high intensity exercise. Further investigation is warranted to establish whether inspiration through a gas analysis system would affect the performance of veterans more than seniors.

The exercise intensity achieved by both groups of cyclists appears to have been related to maintaining a 'steady state' blood lactate concentration. This finding concurs with the pattern of blood lactate response reported by McNaughton et al. (1999). In this study blood lactate concentration was assessed at 10 minute intervals during an indoor 1-h cycling performance ride and the investigators found that blood lactate increased during the first 20 min of the ride with no further change from 20 to 60 min. In the investigation by Nichols et al. (1997) no difference was found between blood lactate concentration recorded at 6.75- and 13.50-km during an outdoor 20-km time trial. The authors stated that this finding was indicative of maintaining a 'steady state' exercise intensity during the event. However Nichols et al. (1997) did not record power output during the field based time trial. In the present study there was no change in mean power recorded at 2.5 minute intervals after the first 2.5 minutes of the indoor time trial. Therefore after this time, senior and veterans maintained a relatively constant power output.

The effect of age on blood lactate concentration recorded during indoor cycling endurance performance has not been considered. However Overend et al. (1992) assessed blood

lactate concentration in elderly subjects during 24 minutes of high intensity cycling exercise. Mean relative exercise intensity ($\%W_{max}$) maintained during the test was higher in the older group with maximal power determined during a similar protocol to the Kingcycle PP test. Mean value for peak whole BLa recorded during the test was higher in the younger group (8.1 vs 6.5 $mmol \cdot L^{-1}$). The authors did not discuss this age-related difference however other investigators have reported an age-related decline in lactacidaemia in older populations (Massé-Biron et al., 1992; Iredale and Nimmo, 1997; Wiswell et al., 2000). Issues concerning the assessment of blood lactate concentration have been discussed previously in this thesis and these methodological problems need to be considered when inter-individual comparisons are made within and between studies. In the present study a large inter-individual variation was found for blood lactate concentration recorded during the time trial and no difference was found between age groups. Cross sectional age-related declines in lactacidaemia appear to be influenced by the criteria used for subject selection and the method used to measure blood lactate concentration.

The most significant finding of this study was that during a field based time trial veterans maintained a higher relative exercise intensity when expressed as a percentage of W_{peak} when compared with senior cyclists. No other study has recorded the power output of veteran cyclists during indoor and outdoor time trials and therefore this data provides new information concerning performance related responses of senior and veteran cyclists. One possible reason for the finding that veterans maintained a higher $\%W_{peak}$ outdoors concerns the effects of pacing strategy on power recorded during the time trial. During the indoor time trials both groups of cyclists maintained a relatively constant power output for the duration of the ride, however power maintained by the veterans outdoors varied considerably with a significant interaction between power and time when compared with the seniors. Analysis of mean power at 5% intervals throughout the outdoor ride revealed that veterans maintained a higher relative exercise intensity between the 25 and 35% of the ride which corresponded with an undulation on the time trial course. The veteran riders maintained a higher relative W_{peak} during the first 5% interval and first half of the race and uphill sections of the course, however these differences did not reach the level of significance. There is very little information available concerning an optimal pacing strategy for the completion of a 16.1-km time trial. data collected during indoor trials

suggest that riders adopt relatively even pace when riding on the Kingcycle ergometer however during outdoor trials variations in environmental conditions such as the topography of the course, wind direction and speed prevent the rider from maintaining a similar even paced power output. Investigators need to be aware of these effects on performance related responses when developing indoor tests to simulate outdoor performance within a controlled laboratory environment. Furthermore more information is required concerning the effect of pacing strategy on power maintained during indoor and outdoor cycling performance rides.

Data collected during the indoor 16.1-km time trial revealed there was no difference between groups for relative power, oxygen uptake, and heart rate. However data from the outdoor time trial indicated that the veteran cyclists maintained a higher relative power without a concomitant change in heart rate. It is reasonable to assume that the higher relative power of the veterans outdoors was indicative of a higher oxidative capacity (Allen et al., 1985) and/or a higher contribution of energy via anaerobic sources. With the development of a portable gas analysis system it is now possible to measure oxygen uptake and ventilation during field based cycling time trials. Future study could use this analysis system to compare seniors and veterans to establish differences in the metabolic cost of indoor and outdoor cycling performance and investigate the effects of outdoor performance on the relationship between power:oxygen uptake.

An interesting finding in the present study was that senior riders maintained a significantly higher relative mean power output during the first 5% interval of the time trial performed indoors but not outdoors. This finding suggests i) that the seniors were required to produce more power to overcome the inertial characteristics of the Kingcycle ergometer in order to attain the self selected power to complete the time trial or ii) the veterans made more effort outdoors than indoors particularly during the first 5% of the ride. No other study has reported the time trial power of seniors outdoors and the power output of veteran cyclists indoors and outdoors. However mean relative power output maintained for the duration of the indoor 16.1-km time trials in the senior group were similar to the value of 77.7% reported by Wood et al. (1997) and lower than the value of 81.1% calculated by Davison et al. (1997) for indoor Kingcycle 16.1-km time trials. One possible explanation for the

difference between studies concerns the training status of the subjects and the mean value for W_{peak} achieved during the Kingcyle PP test. Also confounding effects due to a lack of subject habituation may have influenced the outcomes of these studies.

No other study has reported heart rate values of veteran cyclists recorded during indoor and outdoor cycling time trials. Although absolute values for average heart rate recorded during indoor and outdoor time trials were significantly lower in the veteran cyclists, there was no difference between groups when heart rate values were expressed relative to peak heart rate recorded during a Kingcycle PAC test.

Selley *et al.* (1995) found that average heart rate recorded during field based running races of about 35 min duration was independent of performance time when expressed as a percentage of maximal heart rate. This finding concurs with data reported in the present study which showed that seniors and veterans maintained the same $\%HR_{peak}$ irrespective of performance time recorded for indoor and outdoor cycling time trials. Allen *et al.* (1985) found that mean absolute heart rate recorded at 10-km race pace was significantly higher in seniors when compared with veteran runners. However the authors did not assess whether there was a difference between groups when heart rate was expressed relative to maximal values.

When $\%HR_{peak}$ was compared across groups, analysis of the data revealed that veterans maintained a similar relative exercise intensity to seniors during indoor and outdoor time trials. However veterans maintained a higher $\%W_{peak}$ outdoors and a similar $\%W_{peak}$ indoors when compared with seniors. Investigators need to be aware of the discrepancy between seniors and veterans when using heart rate values determined during laboratory based time trials to prescribe competitive exercise intensity for outdoor time trials. Although the relationship between mean heart rate and power output in the seniors remained consistent for indoor and outdoor events, it is important to note that mean heart rate values for the veterans did not accurately reflect mean power output recorded during the outdoor time trial. This finding highlights problems associated with using heart rate to assess exercise intensity during cycling races (Jeukendrup and van Diemen, 1998). Furthermore the postulate that heart rate response does not accurately reflect exercise intensity in both

senior and veterans can be clearly seen when the HR:power relationship at 5% intervals for the indoor time trial is compared with the HR:power relationship for outdoor performance.

Mean relative values for heart rate recorded outdoors in the seniors were lower than values reported by Dobbins (1996) and Palmer *et al.* (1994) for outdoor 16.1-km time trials. One explanation for this difference concerns the assessment of maximal/peak heart rate and its effect on the calculation of a relative value. Cyclists who participated in the present study were experienced competitive cyclists who had completed several Kingcycle PAC tests, therefore it is likely that heart rate recorded in this group represented a more valid assessment of a peak value.

No other study has recorded blood lactate concentration of veteran cyclists on completion of an outdoor 16.1-km time trial therefore this data represents new and important information concerning age-related differences between seniors and veterans. There was no difference in mean blood lactate concentration found between groups for the outdoor time trial even though blood lactate concentration was higher in the senior group indoors. One explanation for this concerns the interaction between power maintained indoors by each group when compared with outdoors. Notably there was no significant difference between groups for power maintained outdoors during the final 5% period of the time trial, however average power recorded for the veteran group did vary indoor and outdoors.

Peak lactacidaemia is lower in elderly populations (Åstrand and Rodahl, 1986; Roecker et al., 2000), however data presented in the present study suggests that there is no marked age-related decline in lactacidaemia in endurance athletes matched on training intensity and frequency. Care should be taken when cross sectional comparisons are made between age groups in order to account for differences between subjects which may relate more to habitual physical activity and lifestyle pattern and not ageing *per se*. Further longitudinal study is required to assess whether peak blood lactate concentration declines with age in veteran cyclists who maintain high levels of physical activity. Unfortunately there is no information available concerning this issue.

Very few studies have recorded peak blood lactate concentration of senior and veteran cyclists immediately on completion of an indoor cycling time trial however Overend *et al.* (1992) recorded a peak venous whole blood lactate concentration of 6.49 mmol·L^{-1} on completion of 24 mins of high intensity exercise ($\sim$ 74% W_{max}) in a group of 13 active elderly subjects with an average age of 70 yr and Nichols *et al.* (1997) recorded a mean fingertip blood lactate concentration of 5.69 mmol·L^{-1} three minutes after completion of an outdoor 13.5-km time trial in a group of 13 trained female cyclists with an average age of 48 yr. These values are markedly lower than the blood lactate concentration recorded in the veteran cyclists in the present study (10 to 11 mmol·L^{-1}) with mean age of 58 yr, however issues regarding measurement of blood lactate need to be considered when comparisons are made across investigations.

There is very little information available concerning mean pedal cadence during laboratory and field based cycling time trials. This is unfortunate considering the number of studies which have investigated the effects of pedal cadence on cycling economy and efficiency (Hagberg *et al.*, 1981; Marsh and Martin, 1993; 1997; Takaishi *et al.*, 1996). Notably although Hagberg *et al.*, 1981 stated that the self selected pedal cadence of cyclists during bunch start road racing was 90 rev·min^{-1} there appears to be no direct evidence to show that cyclists maintain this rate during field based time trial races. During an indoor 16.1-km time trial Loftin and Warren (1994) found that highly trained cyclists maintained an average pedal cadence of 118 rev·min^{-1} which was significantly higher than the mean value of 97 rev·min^{-1} maintained by trained cyclists. Subjects completed a simulated 16.1-km time trial using their own racing bicycle attached to a Velodyne™ electromagnetically braked cycle ergometer. Not surprisingly, mean power was significantly higher in the highly trained group (262 vs 205 W) therefore the higher pedal cadence may have been an artefact of the power output maintained and not differences in training status. Marsh and Martin (1997) found that training status influenced preferred pedal cadence and that experienced cyclists maintained higher average cadences (between 90 and 100 rev·min^{-1}) when compared with non-cyclists when riding at fixed power outputs on a Velodyne ergometer. There is very little data available concerning the effects of ergometer design on the relationship between power output and preferred pedal cadence and further study is warranted concerning the possible interaction of training status, power output, and

ergometer design on preferred pedal cadence during laboratory based cycling time trials. Preferred pedal cadence can be influenced by gradient (Davison *et al.*, 2000b) and in a review of the literature concerning the relationship between workload and cadence Too (1990) explained that; a significant interaction exists between workload and pedal cadence, and that the most efficient pedalling rate increases with power, and also a most efficient cadence exists for each power output. Similarly, Sargeant (1994) discussed the relationship between force and velocity and used an inverted 'U' to identify the optimum cadence/pedal velocity (rev·min^{-1}) for maximal and sub-maximal workloads. In this review Sargeant (1994) defined maximal work (W) as peak force recorded during a single all-out effort measured using an isokinetic cycle ergometer and therefore this definition of maximal power is in contrast to the term commonly used to describe a sustained maximal effort recorded during a progressive exercise test to volitional exhaustion (W_{max}). Sargeant (1994) illustrated the interaction between muscle fibre type and optimum cadence and explained that each type of muscle fibre has an optimum shortening for the production of maximal force. Notably optimum shortening velocity for type I muscle fibre is markedly lower than type II fibre. In the present study it is possible that the higher pedal cadence maintained by the senior group during the indoor time trial was indicative of muscle fibre composition and a higher relative type II fibre content. Age related changes in muscle fibre composition have been considered elsewhere in this thesis, however further investigation concerning the relationship between muscle fibre content and optimum cadence for senior and veteran cyclists during time trial performance is warranted.

Sargeant (1994) highlighted data concerning the average pedal cadence (~ 104 rev·min^{-1}) maintained by professional cyclists during a 1-h time trial performed on a smooth level terrain cycle track. Notably the bicycle used during this event was specifically designed for track cycling and involved a single gear-fixed wheel which did not allow the rider to stop pedalling. No study has investigated the effect of riding a track bicycle when compared with a conventional bicycle on preferred pedal cadence during this type of event, however it is reasonable to suggest that bicycle design can significantly affect preferred pedal cadence during time trial races. Personal observation of professional cyclists who completed a field based 16.5-km time trial in the Tour de France stage race (2000) found that cyclists riding conventional bicycles with self selected gear ratio maintained a pedal

cadence of between 96 to 102 rev·min^{-1}. Notably, the duration of this event was about 20 min and the course consisted of undulating roads. This pedal rate is markedly higher than the average pedal cadence recorded during the 16.1-km time trial completed by senior and veteran cyclists who participated in the present study. Explanations for this difference include the training status of the cyclist and the topography of the time trial course. Although it is highly likely that professional cyclists in the Tour de France maintained a significantly higher mean time trial power this does not explain why no difference in mean pedal cadence was found between seniors and veterans who maintained different power outputs for the outdoor time trial. Based on the assumption that the topography of the course was relatively similar between the 16.5-km time trial in the Tour de France and the 16.1-km completed in the present study, it is reasonable to suggest that the similar training status of the senior and veteran cyclists who completed the 16.1-km time trial influenced the finding that pedal cadence was similar between age groups.

Assuming that the effect of training status was controlled for between groups it is possible to argue that during the indoor 16.1-km time trial the main contributor to the difference in pedal cadence was the ergometer used to simulate field based cycling. Notably the Kingcycle ergometer used in the present study was designed to simulate the inertial characteristics experienced when riding on level terrain (similar to track cycling using a conventional gear ratio bicycle). However it is likely that the relationship between power and cadence is different when riding outdoors on uneven roads. Investigation is warranted to elucidate the relationship between power and cadence when using laboratory based ergometry and field based courses.

Atkinson and Lloyd (1998) studied the effects of age on preferred pedal cadence when cycling on a Kingcycle ergometer and found a cross sectional age-related decline in cadence when maintaining a relative work rate (percentage of predicted peak power). The authors postulated that a selective age-dependent change in muscle fibre types could explain the age-related difference. In the present study preferred pedal cadence during the indoor 16.1-km time trial was lower in the veterans and indicative of an age-related physiological change. However it is important to note that no difference was found between seniors and veterans for mean pedal cadence recorded outdoors. No study has

recorded the preferred pedal cadence of veteran cyclists during actual 'real world' outdoor time trials, therefore data reported in the present study provides novel and useful information concerning this issue and questions the assumption that laboratory based performance responses are transferable to the field. During the indoor time trials seniors and veterans maintained a relatively constant pedal cadence when mean pedal cadence was calculated at 5% intervals. However mean pedal cadence recorded outdoors varied considerably. Although the 16.1-km time trial course used for the outdoor time trial was relatively flat, cyclists were required to negotiate several undulations and turns in order to complete the course. In contrast to this, the Kingcycle ergometer provided a continuously smooth surface without interruption. No difference between average power indoors and outdoors was found in the senior group therefore the higher cadence indoors appeared to be an artefact of the testing apparatus.

10.7 SUMMARY

Senior and veteran cyclists maintained a similar relative exercise intensity during a laboratory based 16.1-km cycling time trial. There was no difference between groups for blood lactate response assessed during the time trial, however mean value for blood lactate concentration was higher (P = 0.08) in the seniors. Average economy for the duration of the time trial was significantly lower in the veterans but was dependent on the method of calculation. These findings do not concur with previous work which recorded lower values for blood lactate concentration in older athletes and a higher relative exercise intensity at race pace in veteran runners. During a field based 16.1-km time trial veteran cyclists maintained a higher mean relative exercise intensity when compared with; senior cyclists, and a laboratory based ride. There was no difference in mean relative heart rate response across indoor and outdoor rides or between groups for indoor and outdoor rides. Seniors maintained a higher mean pedal cadence indoors but not outdoors. However veterans maintained a similar mean pedal cadence indoors and outdoors. Blood lactate concentration on completion of the indoor ride was higher for the seniors but there was no difference between groups for the outdoor trial. These findings highlight important methodological problems associated with using indoor laboratory based tests to investigate the effect of age on cycling endurance performance and questioned the validity of indoor tests to evaluate cycling performance.

CHAPTER 11

11 PHYSIOLOGICAL CORRELATES TO FIELD BASED 16.1-KM CYCLING TIME TRIAL PERFORMANCE

11.1 INTRODUCTION

In order to identify key factors which are related to cycling endurance performance numerous studies have investigated the inter-relationships between selected physiological variables assessed during laboratory based tests with performance related responses recorded indoors and outdoors. There is no information available concerning the relationship between physiological variables recorded during laboratory based tests and field based performance in senior and veteran cyclists.

In order to investigate the relationship between physiological variables measured during a laboratory test with field based cycling performance, several studies have used average speed or time to complete field based time trials as the criterion measure (Coyle *et al.*, 1991; Hawley and Noakes, 1992; Miller and Manfredi, 1987; Palmer *et al.*, 1996), however, with the development of the SRM power meter, it is now possible to record power output during field based cycling performance (Jeukendrup and van Diemen, 1998).

11.2 AIM OF STUDY 6

To gain a clearer understanding of the physiological correlates of field based cycling performance.

11.3 OBJECTIVES

11.3.1 PART 1

To assess the validity of selected physiological variables to predict field based 16.1-km cycling time trial performance.

11.3.2 PART 2

To assess the validity of W_{peak} to predict field based 16.1-km time trial power and performance time.

11.4 METHODS

Subjects who participated in this part of the study were selected using information obtained from a pre-test training questionnaire (see appendices). Senior and veteran riders were matched on frequency and intensity of training, but not performance. During all tests power was recorded using either a 4 or 20 strain gauge SRM power meter.

11.4.1 PART 1

Six senior and six veteran riders participated in this part of the study. Each subject completed seven tests i) a Kingcycle PP test ii) a lactate minimum test iii) LT_{ramp} iv) LT_{inc} v) LT_{dis} vi) indoor 16.1-km time trial and vii) outdoor 16.1-km time trial. Subject characteristics are shown in Table 47

Table 47. Mean ± SD values for subject characteristics (n = 12)

Age (yr)	43 ± 16
Height (m)	1.82 ± 0.06
Body mass (kg)	76.1 ± 4.9
%BF	16.2 ± 5.3

11.4.1.1 PEAK POWER TEST

Subjects completed a Kingcycle PP test as described in 6.4.1.3.

11.4.1.2 LACTATE MINIMUM TEST

The lactate minimum test consisted of three parts (as described by Davison *et al.*, 2000b).

Subjects completed a Kingcycle PAC test as described in 7.4.2.

The PAC test was immediately followed by a five min active recovery period at an intensity of about 75 W.

The rest period was immediately followed by a progressive ramped test starting at 60% of W_{peak} achieved during PAC with intensity increasing by 6 W·min^{-1} until subjects reached volitional exhaustion.

11.4.1.3 THRESHOLD TEST USING A RAMPED PROTOCOL (LT_{RAMP})

Subjects completed a LT_{ramp} test as described in 9.4.2.2.

11.4.1.4 THRESHOLD TEST USING A CONTINUOUS INCREMENTAL PROTOCOL (LT$_{INC}$)

Subjects completed a LT$_{ramp}$ test as described in 9.4.2.3.

11.4.1.5 THRESHOLD TEST USING A DISCONTINUOUS INCREMENTAL PROTOCOL (LT$_{DIS}$)

Subjects completed a LT$_{ramp}$ test as described in 9.4.2.4.

11.4.1.6 INDOOR 16.1-KM TIME TRIAL

Subjects completed an indoor time trial race (LTT) as described in 6.4.1.4.

11.4.1.7 OUTDOOR 16.1-KM TIME TRIAL

Subjects completed an outdoor time trial race (FTT). Each rider had previous experience of racing on the outdoor 16.1-km time trial course used for the study and was regularly competing in a mid-week racing event. Each race was completed under Road Time Trials Council regulations (RTTC, 1998). During each time trial, each rider started the race at one min intervals and ~30 cyclists raced against each other to complete the 16.1-km distance in the fastest time. Subjects had feedback concerning heart rate and time but not power output. Pedal cadence and gear ratio were self-selected.

11.4.1.8 DETERMINATION OF THRESHOLD INTENSITY

Individual values for power output (W), heart rate (b·min^{-1}) and oxygen uptake ($\dot{V}O_2$ L·min^{-1}) were determined at designated lactate thresholds. Each threshold was identified using visual inspection of blood lactate values plotted against power output and time.

11.4.1.8.1 LMP

Lactate minimum point (LMP) was identified as the minimum blood lactate concentration determined during an incremental test completed after a bout of intense exercise which induced lactic acidosis (Tegtbur *et al.*, 1993).

11.4.1.8.2 TLac

Blood lactate threshold (TLac) was identified as the first abrupt increase in lactate concentration above the baseline blood lactate level (Farrell *et al.*, 1979).

11.4.1.8.3 OBLA

The point of OBLA was identified as the point at which blood lactate reached a fixed concentration of 4 mmol·L^{-1} (Jacobs et al., 1981).

11.4.2 PART 2

Sixteen well trained male endurance racing cyclists (seven seniors, nine veterans) completed a PP test and a field based 16.1-km time trial. During the field based time trials environmental conditions were variable due to changes in the weather. Wind speed varied between ~10-32 km·h^{-1} however wind direction was relatively consistent and resulted in a tail-wind for the outward bound section of the course. Ambient temperature and barometric pressure varied between 15-23°C and 745-775 mmHg, respectively.

Tests were separated by at least one week and each subject competed in a separate 16.1-km time trial race completed on the same course. Data for the whole group was collected over a period of about 4 months (June to September). All subjects had extensive experience of competitive cycling and during the period of the study were regularly participating in local time trial races. The physical characteristics of the subjects are shown in Table 52.

11.4.3 STATISTICAL ANALYSES

Level of significance was set at $P<0.05$ and post hoc comparisons were completed using Tukey HSD tests. Statistical analyses were completed using Microsoft Excel (Bellevue, WA), Minitab (State College, PA) and Statistica Mac 5.1 (Statsoft, Inc). Values in the text are mean (± SD) unless otherwise stated.

11.4.3.1 PART 1

The relationship between variables was assessed using linear models. Pearson product moment correlation coefficient (r) was used to assess the relationship between two variables (Atkinson and Nevill, 1998). However these relationships can be influenced by the heterogeneity of the sample (Hopkins, 1997). The strength of prediction was evaluated using the standard error of estimate (SEE). Few studies report this, however SEE with 95% confidence intervals allows the reader to evaluate the range of error associated with using a predictor. Standard error of estimate (SEE) was calculated as an absolute value and percentage of the predicted value.

11.4.3.2 PART 2

The relationship between W_{peak} and performance during the 16.1-km time trial was assessed using linear and ln-linear models. The ln-linear model described a curvilinear relationship between variables and assumed a multiplicative error term which controlled for spread in the data (heteroscedasticity). Prediction equations based on linear and power function models were calculated with a zero intercept, this allowed predicted values to be extrapolated beyond the actual data points. Confidence intervals for the standard error of estimate (SEE) were based on a chi-squared distribution (Hopkins, 1997).

11.5 RESULTS

11.5.1 PART 1

Mean values (absolute and relative) for power, HR and $\dot{V}O_2$ determined at TLac and OBLA thresholds during ramp, incremental and discontinuous tests are presented in study five of the present thesis. Mean values (absolute and relative) for power, HR and $\dot{V}O_2$ recorded during indoor and outdoor time trials and W_{peak}, HR_{peak} and $\dot{V}O_{2peak}$ assessed during the PP test are presented in part 1 and 2 of this study.

Table 48. Mean ± SD for power output (W), heart rate (b·min⁻¹) and $\dot{V}O_2$ (L·min⁻¹) recorded during selected physiological tests (n = 12)

	Power	Heart rate	Oxygen uptake
Outdoor 16.1-km TT	314 ± 37	161 ± 15	-
Indoor 16.1-km TT	300 ± 50	163 ± 16	4.17 ± 0.61
Peak value	391 ± 59	180 ± 16	4.73 ± 0.60
LMP	273 ± 38	154 ± 15	3.75 ± 0.53
TLac (ramp)	224 ± 29	135 ± 13	2.97 ± 0.46
TLac (incremental)	207 ± 30	128 ± 14	2.76 ± 0.46
TLac (discontinuous)	206 ± 21	127 ± 11	2.81 ± 0.30
OBLA (ramp)	283 ± 40	156 ± 13	3.85 ± 0.62
OBLA (incremental)	274 ± 37	152 ± 13	3.73 ± 0.55
OBLA (discontinuous)	285 ± 42	151 ± 14	3.94 ± 0.55

There was no difference (P>0.05) between average power recorded during indoor and outdoor time trials. Average power for outdoor time trials was higher (P<0.01) than power at LMP, TLac (ramp, incremental, discontinuous) and OBLA (ramp, incremental, discontinuous). Although average power for the indoor time trial was higher than power at LMP and TLac (ramp, incremental, discontinuous) and OBLA (incremental) it was similar to power at OBLA (ramp and discontinuous). There was no difference (P>0.05) between mean values for power at LMP and OBLA incremental.

There was no difference (P>0.05) between average heart rate recorded during indoor and outdoor time trials. Average heart rates for indoor and outdoor time trials were higher (P<0.01) than heart rate at LMP, TLac (ramp, incremental, discontinuous) and OBLA (ramp, incremental, discontinuous). Mean values for heart rate at LMP and OBLA (ramp, incremental, discontinuous) were similar (P>0.05).

Table 49. Relationships (Pearson product moment correlation coefficient) between average power (W) recorded during the 16.1-km time trial race and power output recorded during laboratory based assessments of endurance performance (n = 12)

	r (95%CI)	SEE (W) (95%CI)	SEE (%) (95%CI)
Indoor 16.1-km TT	0.97 (0.89-0.99) *	9 (6-15)	3 (2-5)
W_{peak}	0.99 (0.96-1.00) *	6 (4-10)	2 (1-3)
LMP	0.96 (0.86-0.99) *	11 (8-18)	4 (3-7)
TLac (ramp)	0.88 (0.62-0.97) *	18 (13-30)	7 (5-12)
TLac (incremental)	0.75 (0.31-0.93) $	27 (19-45)	9 (6-15)
TLac (discontinuous)	0.85 (0.54-0.96) $	21 (15-35)	7 (5-12)
OBLA (ramp)	0.84 (0.51-0.95) *	21 (15-35)	8 (6-14)
OBLA (incremental)	0.81 (0.44-0.94) $	23 (16-38)	8 (6-14)
OBLA (discontinuous)	0.89 (0.65-0.97) *	18 (13-30)	6 (4-10)

* denotes significant correlation (P<0.001)

$ denotes significant correlation (P<0.01)

There was no difference (P>0.05) between average oxygen uptake recorded during indoor time trials and mean $\dot{V}O_2$ at OBLA (discontinuous). Mean value for $\dot{V}O_2$ at LMP and

OBLA incremental were similar (P>0.05). Average $\dot{V}O_2$ for the indoor time trial was higher than $\dot{V}O_2$ at LMP, TLac (ramp, incremental, discontinuous) and OBLA (ramp, incremental, discontinuous).

Table 50. Relationships (Pearson product moment correlation coefficient) between average heart rate (b·min⁻¹) recorded during the 16.1-km time trial race and heart rate recorded during laboratory based assessments of endurance performance (n = 12)

	r (95%CI)	SEE (b·min⁻¹) (95%CI)	SEE (%) (95%CI)
Indoor 16.1-km TT	0.97 (0.89-0.99) *	4 (3-7)	3 (2-5)
HR$_{peak}$	0.98 (0.93-0.99) *	3 (2-5)	2 (1-3)
LMP	0.91 (0.70-0.97) *	6 (4-10)	4 (3-7)
TLac (ramp)	0.74 (0.29-0.92) $	10 (7-17)	7 (5-12)
TLac (incremental)	0.81 (0.44-0.94) $	9 (6-15)	6 (4-10)
TLac (discontinuous)	0.86 (0.56-0.96) *	8 (6-14)	5 (4-8)
OBLA (ramp)	0.84 (0.51-0.95) *	8 (6-14)	6 (4-10)
OBLA (incremental)	0.86 (0.56-0.96) *	8 (6-14)	5 (4-8)
OBLA (discontinuous)	0.87 (0.59-0.96) *	7 (5-12)	5 (4-8)

* denotes significant correlation (P<0.001), $ denotes significant correlation (P<0.01)

Strong relationships were found between power determined from each method of assessment and outdoor time trial power. However 95% confidence intervals for SEE revealed that W$_{peak}$, power at LMP and average power indoors provided the most accurate method of determining outdoor 16.1-km time trial power. A strong relationship was found between $\dot{V}O_{2peak}$ and outdoor time trial power in the group of 12 trained cyclists, r = 0.96 (95%CI 0.86-0.99).

A relatively weak relationship was found r = 0.76 (95%CI 0.33-0.93) when the relationship between $\dot{V}O_{2peak}$ and time trial power was assessed in a more homogenous group of the six cyclists who had achieved the highest value for $\dot{V}O_{2peak}$ Standard error of estimates for the prediction of time trial power using $\dot{V}O_{2peak}$ were similar between the heterogeneous and homogenous groups (4% (95%CI 3-7%) vs 3% (95%CI 2-7%) for n = 12 and 6, respectively). The relationship between W$_{peak}$ and time trial power and W$_{peak}$ and $\dot{V}O_{2peak}$

were not diminished in the group of six riders (r = 0.99; 95%CI 0.96-1.00 with SEE of 0.5% (95%CI 0.3-1.3%), and r = 0.97 (95%CI 0.89-0.99) with SEE of 3% (95%CI 2-6%) respectively).

Strong relationships were found between heart rate determined from each method of assessment and average heart rate recorded during the outdoor time trial. However 95% confidence intervals for SEE revealed that HR_{peak}, heart rate at LMP and average heart rate recorded during the indoor time trial provided the most accurate method of determining the average heart rate recorded during an outdoor 16.1-km time trial.

Table 51. Relationships (Pearson product moment correlation coefficient) between average oxygen uptake (L·min^{-1}) recorded during an indoor 16.1-km time trial and oxygen uptake recorded during laboratory based assessments of endurance performance (n = 12)

	r (95%CI)	SEE (L·min^{-1}) (95%CI)	SEE (%) (95%CI)
$\dot{V}O_{2peak}$	0.96 (0.86-0.99) *	0.17 (0.12-0.29)	5 (4-8)
LMP	0.86 (0.56-0.96) *	0.32 (0.23-0.54)	9 (6-15)
TLac (ramp)	0.65 (0.12-0.89) #	0.50 (0.35-0.85)	13 (9-22)
TLac (incremental)	0.85 (0.54-0.96) $	0.35 (0.25-0.59)	9 (6-15)
TLac (discontinuous)	0.63 (0.09-0.88) #	0.51 (0.36-0.87)	13 (9-22)
OBLA (ramp)	0.74 (0.29-0.92) $	0.43 (0.30-0.73)	12 (9-20)
OBLA (incremental)	0.77 (0.35-0.93) $	0.41 (0.29-0.70)	12 (9-20)
OBLA (discontinuous)	0.73 (0.27-0.92) $	0.44 (0.31-0.75)	12 (9-20)

denotes significant correlation (P<0.001)

$ denotes significant correlation (P<0.01)

# denotes significant correlation (P<0.05)

Peak oxygen uptake and $\dot{V}O_2$ at LMP and TLac (incremental) were strongly related to average $\dot{V}O_2$ recorded during the indoor 16.1-km time trial. However relatively weak relationships were found between average $\dot{V}O_2$ for the time trial and $\dot{V}O_2$ at TLac (ramp, discontinuous) and OBLA (ramp, incremental, discontinuous). Notably 95% confidence intervals for SEE revealed that $\dot{V}O_{2peak}$ provided the most accurate method of determining the average $\dot{V}O_2$ recorded during an indoor 16.1-km time trial.

11.5.2 PART 2

Table 52. Subject characteristics and individual performance during a field based 16.1-km time trial (n = 16)

Subject	Age (yr)	Height (m)	Mass (kg)	W_{peak} (W)	16.1-km TT (W)	Speed (km·h⁻¹)	Time (min:s)
1	25	1.82	76.5	484	368	44.00	21:49
2	25	1.79	71.0	432	341	46.01	20:52
3	25	1.84	82.5	457	353	39.72	24:10
4	29	1.87	73.0	453	348	45.46	21:07
5	30	1.91	73.5	403	325	42.11	22:48
6	30	1.78	66.5	425	338	45.35	21:10
7	31	1.91	75.5	423	337	40.85	23:30
8	42	1.75	73.6	378	296	44.31	21:40
9	46	1.81	76.5	454	357	43.15	22:15
10	52	1.78	71.5	368	307	43.70	21:48
11	55	1.76	71.5	348	283	41.62	23:04
12	56	1.76	72.5	360	292	43.37	22:08
13	58	1.84	86.0	339	292	39.40	24:22
14	60	1.80	79.5	280	224	38.30	25:07
15	61	1.77	79.0	304	251	41.50	23:08
16	63	1.73	80.5	324	270	43.80	21:55
Mean	43	1.81	75.6	390	311	42.67	22:34
SD	15	0.05	5.0	61	41	2.28	1:14

Table 52 shows the individual data for W_{peak}, average power output (W_{FTT}) and performance time (T_{FTT}) recorded during the 16.1-km time trial and Figure 31 shows the relationship between W_{FTT} and W_{peak}. A very strong relationship was found between W_{peak} and W_{FTT} using linear (r = 0.97; P<0.001, 95%CI 0.91-0.99; SEE 7 W, 95%CI 5 to 11 W) and ln-linear models (r = 0.99; P<0.001, 95%CI 0.97-1.0, SEE of 2.5%, 95%CI = 1.8 to 3.9%).

Figure 32 shows the relationship between W_{peak} and T_{FTT}, a weak correlation was found between W_{peak} and T_{FTT} using linear (r = 0.46, P = 0.07; 95%CI -0.05-0.78, SEE of 1:09 min:s, 95%CI 0:51 to 1:49 min:s) and ln-linear models (r = 0.48, P = 0.06; 95%CI -0.02-

0.79, SEE of 5.1%, 95%CI 3.7 to 8.0%). Similarly, a weak correlation was found between W_{FTT} and T_{FTT} using linear and ln-linear models (r = 0.46 and 0.48, respectively). However, correlation coefficients were significantly improved when the ratio of W_{peak}:body mass ($W \cdot kg^{-1}$) and W_{FTT}:body mass ($W \cdot kg^{-1}$) were regressed on T_{FTT} using a linear model (r = 0.64; P<0.01, 95%CI 0.21-0.86, SEE of 1:00 min:s, 95%CI 0:44 to 1:35 min:s and r = 0.66; P<0.01, 95%CI 0.24-0.87, SEE of 0:58 min:s, 95%CI 0:43 to 1:31 min:s) respectively.
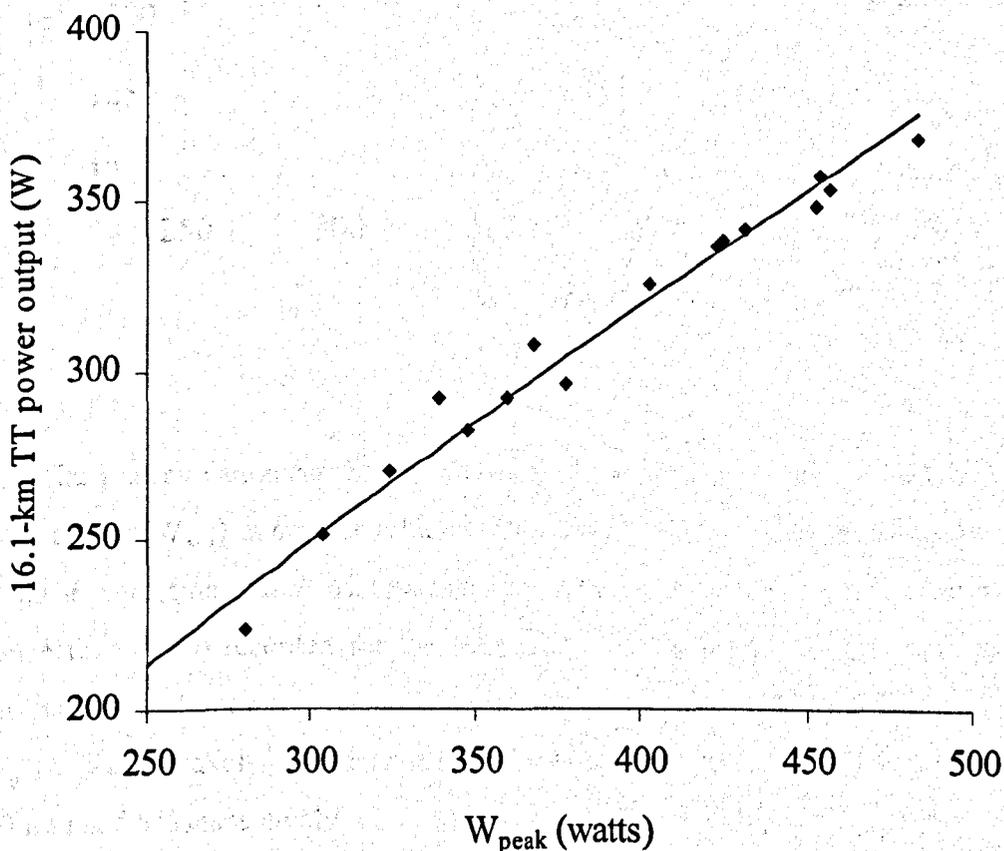
Figure 31. The relationship between time trial performance (W, mean power output for 16.1-km) and peak power output (W_{peak}) in sixteen subjects. Data fitted with a line of best fit calculated from the ln-linear regression of time trial performance power on peak power (r = 0.99, P<0.001). Power (watts) measured during the time trial was related to W_{peak} (watts) by the following linear and power function equations:- $W_{FTT} = 0.67*(W_{peak}) + 50$ and $1.92*(W_{peak}^{0.85})$ respectively. Standard error of estimate for linear and ln-linear models were 7 W and 2.5% respectively.
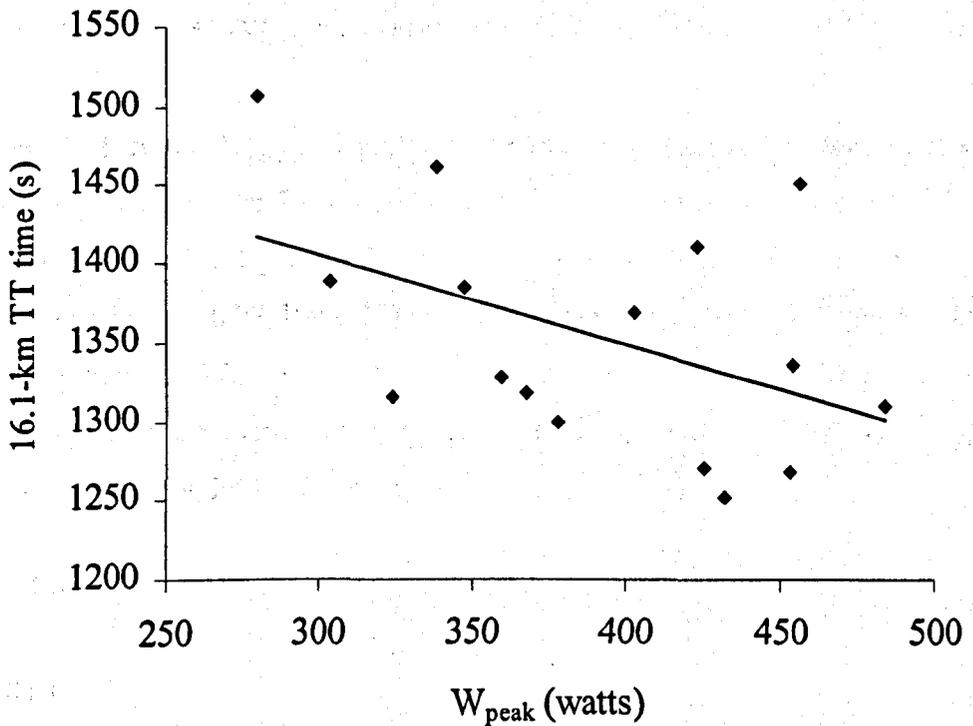
Figure 32. The linear relationship between time trial performance time (s, 16.1-km) and peak power output (W_{peak}) in sixteen subjects. Data fitted with a line of best fit calculated from the linear regression of time trial performance time on peak power ($r = 0.46$, $P>0.05$). Performance time (secs) recorded for the time trial was related to W_{peak} (watts) by the following linear and power function equations:- $T_{FTT} = 1575-0.57*(W_{peak})$; and $3585*(W_{peak}^{-0.16})$ respectively. Standard error of estimate for linear and ln-linear models were 1:09 min and 8% respectively.

When the relationship between W_{peak} and W_{FTT} was assessed in seven riders who achieved the highest values for W_{peak} (range 423 to 484 W) and W_{FTT} (range 337 to 368 W), further analysis of the data revealed a strong relationship between W_{peak} and W_{FTT} using linear ($r = 0.99$; $P<0.001$, 95%CI 0.91-0.99; SEE of 3 W, 95%CI 2 to 4 W) and ln-linear models ($r = 0.98$; $P<0.001$, 95%CI 0.97-1.0, SEE of 0.8%, 95%CI 0.6 to 1.3%). However, for this group no relationship was found between W_{peak} and T_{FTT} ($r = 0.06$ and 0.07, $P = 0.89$ and 0.87 for linear and ln-linear models respectively). Power (W) measured during the time

trial was related to W_{peak} by the following linear and power function equations:- $W_{FTT} = 0.51 \cdot (W_{peak}) + 121$ and $6.53 \cdot (W_{peak}^{0.65})$ respectively.

The relationship between W_{peak} and performance time was assessed in the seven senior aged cyclists (age range from 25-31) and nine older riders (age range from 42-63) who formed the complete subject group of sixteen. No relationship was found between W_{peak} and T_{FTT} in the senior ($r = 0.07$, $P = 0.87$) and veteran ($r = 0.59$, $P = 0.09$) groups and when the relationship between W_{peak} and performance power was assessed in the young ($n = 7$) and older ($n = 9$) riders, however a very strong relationship was found between W_{peak} and time trial power ($r = 0.99$, $P<0.0001$ and $r = 0.98$, $P<0.0001$) respectively.

11.6 DISCUSSION

11.6.1 PART 1

The main finding of this study was that W_{peak} recorded in senior and veteran cyclists was highly related to mean power recorded during a field based 16.1-km time trial and that W_{peak} provided a valid prediction of time trial power when compared with several blood lactate parameters. This finding concurs with the work of several investigators who reported that maximal power is strongly related to indoor (Bishop *et al.*, 1998; Hawley and Noakes, 1992; Wood *et al.*, 1997) and outdoor (Davison *et al.*, 1999; Dobbins, 1996) cycling time trial performance.

In the present study, correlation coefficients revealed the strongest blood lactate predictor of outdoor time trial power was power at lactate minimum point. This finding agreed with the work of Balmer *et al.* (1997) and Davison *et al.* (1997) who found that power at lactate minimum point provided a more accurate estimation of 16.1-km time trial power than power at TLac and OBLA. In the present study, a strong correlation was found for power at TLac and OBLA and field based time trial power, however 95% confidence intervals for SEE revealed that power at TLac and OBLA did not provide an accurate prediction of outdoor time trial power when compared with power at lactate minimum point, W_{peak} and average power recorded during the indoor time trial.

The most accurate predictor of time trial heart rate was peak heart rate recorded during the Kingcycle PAC test and the strongest predictor of average $\dot{V}O_2$ during an indoor 16.1-km time trial was $\dot{V}O_{2peak}$ recorded during the PAC test. Notably HR_{peak} provided the most accurate predictor of field based heart rate when compared with heart rate values determined using blood lactate parameters and average heart rate recorded during the indoor time trial. Similarly $\dot{V}O_{2peak}$ provided the most accurate predictor of average $\dot{V}O_2$ during an indoor 16.1-km time trial. Therefore data from a single Kingcycle PAC test provided values for W_{peak}, HR_{peak} and $\dot{V}O_{2peak}$ which could be used to predict average power and heart rate outdoors and average oxygen uptake indoors.

The assessment of a maximal exercise intensity in elderly individuals has serious implications for health and safety, however veteran cyclists frequently complete field based races at or near maximal intensity. The issue of maximal testing veteran cyclists is particularly problematic and therefore future study should also identify a sub-maximal assessment which could provide an equally valid predictor of field based performance. In the present study, analysis of the data revealed that power at TLac determined using a ramp protocol provided a valid predictor of 16.1-km time trial power. Notably threshold values for TLac occurred at a relatively low intensity when compared with OBLA. Massé-Biron *et al.* (1992) found that veteran riders could not attain an exercise intensity associated with OBLA, however in the present study OBLA intensity was determined in all of the veteran cyclists. Several studies have found that power at lactate threshold can predict outdoor 40-km cycling time trial time (Coyle *et al.*, 1991) and indoor 1-h time trial power (Bishop *et al.*, 1998). The present study revealed that power at TLac can provide a reasonable prediction of outdoor time trial power (SEE of 7%).

Investigators have found that maximal power can provide a more accurate assessment of laboratory-based cycling endurance performance than $\dot{V}O_{2max}$ in cyclists (Dobbins, 1996; Weston *et al.*, 1997) and runners (Jones and Doust, 1998; Noakes *et al.*, 1990; Scott and Houmard, 1994). In the present study, relationships found between W_{peak}, $\dot{V}O_{2peak}$ and outdoor time trial power were similar. One possible explanation for this finding concerns the method of testing used to establish W_{peak} and VO_{2peak}. Keen *et al.*, (1991) developed the

Kingcycle PP test in order to predict $\dot{V}O_{2peak}$ from W_{peak} and found that the relationship between W_{peak} and $\dot{V}O_{2peak}$ was very high (r = 0.96 with SEE of 0.15 L·min^{-1}).

The postulate that W_{peak} is highly dependent on $\dot{V}O_{2peak}$ is equivocal. For instance in the study by Bishop et al. (1998), 24 female cyclists completed an incremental cycling test with a 25 W increase every 3 min to volitional exhaustion and a weak relationship was found between W_{max} and $\dot{V}O_{2max}$ (r = 0.42). This was in marked contrast to the study of Hawley and Noakes (1992) who used an incremental cycling test with a 25 W increase every 2.5 min and found that W_{max} was highly related to $\dot{V}O_{2max}$ (r = 0.97). One explanation concerns the heterogeneity of the group tested by Hawley and Noakes (1992), however Bishop et al. (1998) also tested a heterogeneous group of female cyclists. In a recent study by Wiswell et al. (2000) the strongest predictor of performance time for 5-km, 10-km and marathon distances in a large heterogeneous group of master athletes was $\dot{V}O_{2max}$ (L·min^{-1} and mL·kg^{-1}·min^{-1}). Notably these authors reported relatively weak relationships between performance times and blood lactate parameters, however they did not assess running velocity at $\dot{V}O_{2max}$. One explanation for the strong relationship between W_{peak} and $\dot{V}O_{2peak}$ found in the present study concerns the effects of testing protocol and measuring apparatus on the determination of these variables. There is very little information available concerning the effect of ergometer design and method used to provide a workload resistance on the assessment of W_{peak} and $\dot{V}O_{2peak}$ and the relationship between W_{peak}:$\dot{V}O_{2peak}$.

When evaluating the effects of training on changes in exercise performance capacity, Jones and Doust (1998) argued that the relationship between a predictor and criterion measure may be of less importance than confidence in the reproducibility of the measure. An assessment of the relationship between two variables will depend on intra-individual biological variability and inherent technical error associated with the determination of each variable. For instance, studies have reported that the test re-test reproducibility (coefficient of variation) for $\dot{V}O_{2max}$ was 8% (Howley et al.,1995), LT was 1.5% (Pfitzinger and Freedson, 1998) and performance power for a 40-km field based cycling time trial was 2% (Smith et al., 2000). In study 2 assessment of the reproducibility of $\dot{V}O_{2peak}$ determined during the Kingcycle PAC test revealed a coefficient of variation of 4%. The strong

relationships between W_{peak} and $\dot{V}O_{2peak}$ and 16.1-km time trial power reported in the present study may have been influenced by the low range of variability associated with the determination of these variables.

Maximal power output at $\dot{V}O_{2max}$ is dependent on the method of testing (see review of the literature in chapter 5 of the present thesis), one possible explanation for the different relationship between W_{max} and $\dot{V}O_{2max}$ observed in elite and professional riders (Lucia *et al.*, 1998) concerns the effect of using a standardised testing protocol to assess these variables. In the present study relative ramp rate was determined from W_{peak} recorded during a previous PAC test and may have removed the influence of training status from the testing protocol. If the achievement of $\dot{V}O_{2peak}$ and W_{peak} is affected by the combination of training status and testing protocol, it is reasonable to assume that a relative load increment would provide a more valid method of assessing inter-individual differences in this relationship. Further investigation is warranted to study longitudinal intra-individual changes in $\dot{V}O_{2peak}$ and W_{peak} and to examine the effect of testing protocol on the W_{peak}:$\dot{V}O_{2peak}$ relationship.

The relationship between two variables assessed using Pearson product moment correlation coefficient is dependent on the heterogeneity of the sample (Hopkins, 1997) and studies which have found a correlation between $\dot{V}O_{2max}$ and performance have tended to include subjects with a wide range of ability (Hawley and Noakes, 1992; Saltin and Åstrand, 1967). Interestingly when mean relative $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) was used to match two groups of cyclists (elite vs highly trained), Coyle *et al.* (1991) found that cyclists with a higher absolute $\dot{V}O_2$ and relative $\%\dot{V}O_{2max}$ at LT were able to maintain a higher average power during a laboratory-based 1-h performance ride and average speed during a field based 40-km time trial. However Coyle and co workers argued that $\dot{V}O_{2max}$ could not be used to identify inter-individual differences in performance even though best predictor of average speed for the field based 40-km time trial in the elite group of cyclists was $\dot{V}O_{2max}$ and not $\dot{V}O_2$ at LT, or average power achieved during the laboratory based 1-h performance ride. This finding contradicts the argument that $\dot{V}O_{2max}$ does not provide a valid assessment of cycling performance ability in a homogenous group of elite competitors. However, it could be argued that the elite group assessed by Coyle *et al.* (1991) was heterogeneous

with a range of $\dot{V}O_{2max}$ of 4.56 to 5.64 (L·min⁻¹). In the present study a strong relationship was found between $\dot{V}O_{2peak}$ and outdoor 16.1-km time trial power in a heterogeneous group of 12 trained cyclists with a range of $\dot{V}O_{2peak}$ of 3.89 to 5.46 (L·min⁻¹). However a relatively weak relationship was found between $\dot{V}O_{2peak}$ and time trial power in the homogenous group of six trained cyclists with a range of $\dot{V}O_{2peak}$ of 5.06 to 5.46 (L·min⁻¹). This data highlighted problems concerning the effect of heterogeneity on the interpretation of relationships identified using Pearson product moment correlation coefficient.

In the recent study by Hoogeveen (2000) six elite and nine professional cyclists (n = 15) were tested off-season and in the middle of the racing season. A significant increase in both $\dot{V}O_{2max}$ and W_{max} was recorded over this period of time. Unfortunately Hoogeveen (2000) did not assess cycling performance however it was assumed that performance had increased from pre-season values. Lucia et al. (1998) found that W_{max} was higher in professional riders when compared with elite amateurs without a difference in $\dot{V}O_{2max}$. This finding suggests that a higher W_{max} is achieved due to a greater reliance on anaerobic sources of energy production. In contrast to this, Hoogeveen (2000) suggested that training induced improvements in $\dot{V}O_2$ kinetics increased the oxygen uptake recorded during an incremental exercise test. Cyclists were able to achieve a higher maximal power due to an increased $\dot{V}O_2$ at each stage of the test which was indicative of an increased aerobic contribution to the energy requirements for each load increment. Consequently training induced adaptations in $\dot{V}O_2$ kinetics allowed the cyclists to attain a higher $\dot{V}O_{2max}$ and W_{max} due to a reduction in the need to provide energy via anaerobic sources.

Endurance events involve the utilisation of both aerobic and anaerobic energy sources (McArdle et al., 1991) and the relative contribution of aerobic and anaerobic pathways depends on the duration of the event, the relative exercise intensity of the event and inter- and intra-individual differences in oxidative and anaerobic capacity (Wood, 1998b). It is difficult to assess an individual's anaerobic capacity (see Vandewalle et al., 1987) and measure the relative contribution of aerobic and anaerobic energy sources during high intensity exercise. Work completed by Craig et al. (1993) found that anaerobic power was significantly related to performance of a short distance (4-km) time trial completed on a cycling track. However, methodological issues concerning the quantification of anaerobic

capacity have to be considered when evaluating these findings. Further investigation is warranted concerning the effects of age, testing protocol and training status on the interaction of aerobic and anaerobic energy production during maximal and sub-maximal cycling performance.

Very little is known about the relative contributions of aerobic and anaerobic energy sources during the 60-s period of W_{peak}. The close relationship between W_{peak} and $\dot{V}O_{2peak}$ suggests that aerobic sources are the main contributors to improvements in W_{peak} with training, however data presented by Lucia *et al.* (1998) showed that higher values of W_{max} achieved by professional riders was due to an increase in economy (more power without a change in $\dot{V}O_2$). Differences in W_{max} between professional and elite amateur cyclists were attributed to biomechanical factors which influenced power output and a greater relative energy contribution from anaerobic sources. Wilber *et al.*, (1997) compared elite and well trained groups of cyclists and found no difference between groups for $\dot{V}O_{2max}$ but a significantly higher W_{max} for the elite group. The authors postulated that the higher W_{max} could be linked to differences in pedalling biomechanics and differences in neuromuscular parameters and patterning. This postulate was based on the work of Coyle *et al.* (1991) who found that peak torque recorded during each pedal stroke was higher in elite when compared with well trained cyclists maintaining the mean power output pre-determined from a 1-h time trial.

The relationship between $\dot{V}O_2$ and power output is dependent on:- the absolute power required to complete the task; the load increment (rate of loading) required to achieve the power; the duration of the task; the training status of the individual and the validity and reproducibility of the device used to record power output and oxygen uptake. Therefore investigators need to be aware of these effects when comparisons are made between studies which have investigated the relationship between power and oxygen uptake during cycling performance.

11.6.2 PART 2

In order to evaluate the performance ability of endurance athletes it is necessary to use laboratory based tests which are reliable, valid and sensitive to small changes in an athlete's

fitness level (Hopkins *et al.*, 1999). This is particularly important when performing repeated measurements over a period of time and when laboratory data is applied to the field to predict actual performance.

Data from the present study shows that individual performance power during a 16.1-km time trial can be accurately predicted from W_{peak} ($r = 0.99$). Therefore instead of using heart rate response and/or average speed, a cyclist using an SRM power meter could ride to a pre-determined power output based on W_{peak} recorded during a PP test. It is worth noting that Jeukendrup and van Diemen (1998) found that the relationship between heart rate and power can be uncoupled during prolonged exercise and can be affected by the rider's position and environmental conditions. The authors concluded that a measure of power output during training and racing could be used to provide a more reliable assessment of exercise intensity. Similarly, Maassen and Busse, (1989) showed that the relationship between heart rate and relative power output ($\%W_{max}$) was significantly different after completion of an endurance training program.

A recent study by Smith *et al.* (2000) assessed the reproducibility of average power recorded during indoor and outdoor 40-km cycling time trials and calculated that the coefficient of variation across trials 2 and 3 was 1.9 and 2.1% respectively. Several studies have assessed the variability of indoor time trial performance (Bishop, 1997; Palmer *et al.*, 1996) however this is the first study to investigate the reproducibility of outdoor time trial power. Notably, values for coefficient of variation reported by Smith *et al.* (2000) were similar to values reported for indoor time trials completed in a controlled laboratory environment (see Hopkins *et al.*, 1999).

The strong relationship between W_{peak} and time trial performance power reported in this study suggests that a change in W_{peak} could have a direct affect on 16.1-km time trial power. Notably in the study by Lindsay *et al.* (1996) maximal workload (W_{max}) achieved during a maximal test increased in highly trained cyclists who completed a four week high intensity interval training program. However, even though the relationship between W_{max} and time trial performance was high ($r = 0.84$) increases in W_{max} ($\sim 5\%$) were not significantly related to an improvement in Kingcycle 40-km time. Similarly, Westgarth-

Taylor *et al.* (1997) found that high intensity training increased W_{max}, but there was no significant relationship between the change in W_{max} and a decrease in 40-km time. It was postulated that cycling performance was a combination of the cyclists' absolute W_{max} and their ability to sustain a high percentage of W_{max} and that W_{max} could account for 70-90% (Hawley and Noakes, 1992; Lindsay *et al.*, 1996) of the variation in time trial performance time. Data from the present study suggests that W_{peak} can account for 98% of the variation in time trial performance power and 21% of the variation in time trial time.

It is worth noting that the assessment of W_{max} by Lindsay *et al.* (1996) and Westgarth-Taylor *et al.* (1997) involved a continuous 25 W incremental test (the duration of each increment was 150-s) performed on a Lode ergometer. In the present study W_{peak} was assessed using a continuous Kingcycle ($\sim$ 20 W·min^{-1}) ramp test. Although both of these tests assess maximal/peak power, the measurement of W_{max} in the study of Lindsay *et al.* (1996) and Westgarth-Taylor *et al.* (1997) was based on the calculation of completed work rate in W plus the fraction of time spent in the final non-completed stage multiplied by the final load increment in W. In the present study W_{peak} was calculated as the highest average power output recorded during any 60-s period of the ramp protocol. Therefore the determination of W_{max} in the other studies was based on an estimation of maximal minute power and not a direct measurement. It is surprising that this method of testing has been used by several investigators to assess the W_{max} of well trained (Bishop *et al.*, 1998; Hawley and Noakes, 1992; Jeukendrup *et al.*, 1996; Kuipers *et al.*, 1985; Lindsay *et al.*, 1996; Palmer *et al.*, 1994; Stepto *et al.*, 1999; Westgarth-Taylor *et al.* 1997; Weston *et al.*, 1997), elite (Wilber *et al.*, 1997) and professional cyclists (Hoogeveen, 2000; Padilla *et al.*, 1999; 2000).

Several studies (Davis *et al.*, 1982; Hansen *et al.*, 1988, Yoshida, 1984) have found that the assessment of maximal power can be affected by the method of testing, however further investigation is required to establish whether a change in W_{max} due to the effects of training/detraining would be matched by a change in time trial performance power. Furthermore there is very little information available concerning the effect of cycle ergometer design on the assessment of W_{peak} (with particular reference to the method used to provide resistance and the inertial characteristics of equipment used to mimic actual

cycling). Future study could investigate the effects of using different cycle ergometers to measure W_{peak} with power measured using a SRM power meter.

Peak power output was a strong predictor of 16.1-km time trial performance power across a wide range of ability (time trial power 224 to 368 W). Therefore the heterogeneity of the subjects may have influenced the relationship between W_{peak} and 16.1-km time trial power. However, Figure 31 shows that the relationship between W_{peak} and time trial power was also high in the best performers (time trial power 337 to 368 W) and therefore peak power could be used to predict the performance power of cyclists who maintain both high and low power outputs. Although a weak correlation was found between peak power and time trial time, it is important to note that no correlation was found between peak power and performance time in the group of best performers. This finding needs to be considered when studies investigate the effects of a treatment/intervention on cycling performance and use performance time to indirectly assess the power output of competitive cyclists.

The finding that peak power was not strongly related to time trial time is not surprising when the effects of individual aerodynamics and variable environmental conditions are considered. The relationship between power output and speed is dependent on factors such as wind speed and direction, ambient temperature and atmospheric pressure as well as body size, racing position and bicycle design. In the present study cyclists competed in separate time trial races and therefore environmental conditions were not standardised. Furthermore, each rider's performance power and time trial time was assessed during a single event and cyclists used their own self selected racing position and bicycle equipment. Studies which have reported strong relationships between field based performance time and a laboratory based measure of power output have typically recorded the time trial time of a group of riders during the same event completed on the same day (Hawley and Noakes, 1992), used the personal best performance time of each rider (Coyle et al., 1991) or the mean individual performance time achieved during a series of races (Palmer et al., 1996). Data reported in the present study showed that average power output provided a more valid assessment of endurance performance than time trial time when comparisons are made across different days and events.

Coyle *et al.* (1991) found that laboratory based 1-h performance power was highly related to personal best time to complete a 40-km time trial (r = -0.88) however, SEE for the prediction of performance time was 1:48 min:s (95%CI 1:18-2:54 min:s). Similarly, Hawley and Noakes (1992) found a strong relationship between 20-km cycle time and W_{max} (r = -0.91) with a SEE of 1:36 min:s (95%CI 1:12-2:22 min:s). In the present study a weak relationship was found between time trial power output and time trial time (SEE of 1:09 min:s, 95%CI 0:51-1:49 min:s). However, the SEE for the prediction of time trial power was 2.5% (95%CI 1.8-3.9%). Further research is required to determine the effect of ± 2.5% performance power on 16.1-km cycle time.

Hawley and Noakes (1992) reported that the correlation between W_{max} and outdoor 20-km cycling time was decreased when W_{max} was expressed relative to body mass, it was suggested that for a relatively low ratio between W_{max} and body mass, performance time was less for riders with a large body mass. It was explained that when riding on a flat course the larger rider experiences less wind resistance due to a relatively smaller body surface area. It is reasonable to assume that the topography of the course used in this study may have influenced the finding that the relationship between peak power output and performance time was improved when W_{peak} was expressed relative to body mass.

The weak correlation found between performance power and time trial time can be explained by inter-individual differences in aerodynamics and the effects of environmental factors such as wind speed and direction and the topography of the course. Unlike performance time and/or average speed, power output is not dependent on aerodynamics, environmental factors and topography. Data from this study showed that time trial time was not related to W_{peak} or performance power when riders participated in separate 16.1-km races performed on different days.

11.7 SUMMARY

11.7.1 PART 1

Data collected in the present study revealed the underlying relationship between W_{peak} and 16.1-km time trial power and the inter-relationship between W_{peak} and $\dot{V}O_{2peak}$. Although a

laboratory based simulated 16.1-km time trial and blood lactate response during progressive exercise provided valid methods to predict outdoor 16.1-km time trial power and heart rate no discernible difference was found between these methods of assessment and values obtained from a Kingcycle PP/PAC test. Peak values for power, heart rate and oxygen uptake were valid predictors of average power and heart rate recorded during a field based 16.1-km time trial and average oxygen uptake during an indoor 16.1-km time trial. Bishop *et al.* (1998) commented 'that the time-consuming and costly analysis of lactate was not necessary for the prediction of average power maintained during a laboratory based 1-h cycling time trial' (p. 1274). The findings of the present study confirm this statement and suggest that the determination of lactate threshold is not necessary for the prediction of field based 16.1-km time trial power in a group which consisted of senior and veteran cyclists.

11.7.2 PART 2

Peak power achieved during a Kingcycle PP/PAC test can be used effectively to predict power output but not performance time during a field based 16.1-km time trial. This method of testing affords a relatively inexpensive, non-invasive, reproducible and valid assessment of cycling endurance performance for senior and veteran cyclists. Further investigation is warranted concerning the relationship between W_{peak} and endurance cycling performance during a period of training/detraining as occurs within a normal training/racing season.

12 CONCLUSIONS

VALIDITY AND REPRODUCIBILITY OF THE KINGCYCLE

The Kingcycle test rig (with version 5.5 computer software) did not provide an accurate measure of power when compared with the SRM ergometry system. Investigators should be aware of this discrepancy (~ 10%) when using each measuring device and when inter-individual comparisons are made within and between studies. Within subject variation in Kingcycle power was acceptable and during relatively constant load conditions the reproducibility of Kingcycle power was high (CV ~ 1%). However SRM did provide a more reproducible measure of W_{peak} during a Kingcycle peak power test and this finding has implications concerning sample size and resources required for future intervention studies. Based on the findings of this study investigators are advised to use the SRM power meter to assess power output during the physiological testing of senior and veteran cyclists.

REPRODUCIBILITY OF PEAK PHYSIOLOGICAL VARIABLES AND ENDURANCE PERFORMANCE DURING CYCLING TESTS

Within subject variation (CV%) for SRM_{Wpeak} HR_{peak}, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ recorded during peak power and peak aerobic capacity tests were relatively low (1.3, 1.0, 3.8, 3.1, 3.5%, respectively). Coefficient of variation calculated for peak blood lactate concentration recorded five minutes after completion of the peak aerobic capacity test was relatively high (6.7%). Reproducibility (CV%) of average power and heart rate recorded during an indoor 16.1-km cycling time trial was acceptable (2.8, 1.3%, respectively). Therefore the testing methods and equipment used in the present study provided a reliable assessment of selected peak physiological variables and endurance performance during indoor cycling tests. Based on the findings of this study it is possible to conclude that these testing methods can be used effectively to assess age related changes in these key physiological variables and provide informative comparisons between senior and veteran cyclists.

AGE-RELATED CHANGES IN PEAK PHYSIOLOGICAL VARIABLES

Age was a modest predictor of peak physiological variables assessed during a Kingcycle PAC test in a heterogeneous population of competitive cyclists. The decline in W_{peak} with age was between 25 and 30 W, (~ 6 to 9%) per decade with a more pronounced in a more

homogenous group who achieved the highest values for W_{peak} decline (~ 40 W or 10% per decade). Regression analysis provided a prediction of the age related decline in HR_{peak} (210-0.66·age). The findings of the present study confirm data reported in previous cross sectional work that age related declines are dependent on the endurance performance ability of the subject group.

Absolute age-related declines in $\dot{V}O_{2peak}$ were similar to values reported in the literature calculated from both cross sectional and longitudinal work. Age accounted for about 30 to 45% of the variance in $\dot{V}O_{2peak}$ dependent on the method of scaling used to account for changes in body composition. There was no relationship between age and peak values for RER, breathing frequency and economy. However relationships were found between age and declines in peak $\dot{V}CO_2$, ventilation, pedal cadence and blood lactate concentration.

Although longitudinal studies appear to provide a more valid assessment of the effect of age on endurance performance capacity, data reported in the present study revealed new information concerning the relationship between age and selected physiological variables assessed during a Kingcycle PAC test. *Investigators need to be aware of age related declines in these physiological variables which are commonly associated with successful endurance performance particularly when comparisons are made between age groups and when using normative values to assess relative exercise performance capacity.*

AGE-RELATED CHANGES IN TLAC AND OBLA THRESHOLD EXERCISE INTENSITY

There was a significant age associated decline in absolute values for power and HR determined at TLac and OBLA thresholds. However new data concerning the age-related change in power and heart rate at threshold revealed that relative exercise intensity at TLac and OBLA when expressed as $\%W_{peak}$ and $\%HR_{peak}$ tended to increase with age however this change did not reach the level of significance (P = 0.11 and 0.26, respectively). This finding did not concur with previous work which found that relative exercise intensity at threshold increased with age. Methodological issues concerning subject selection and cross sectional analysis were highlighted, therefore investigators need to be aware of these issues when comparisons are made between age groups particularly when using laboratory based assessments of endurance capacity such as TLac and OBLA.

AGE-RELATED CHANGES IN PERFORMANCE RELATED RESPONSES

Absolute values for performance power and heart rate maintained during an indoor cycling time trial declined with age, however relative exercise intensity when expressed as $\%W_{peak}$ and $\%HR_{peak}$ was not influenced by age (P = 0.18, 0.35, respectively). Issues concerning subject selection and cross sectional methods of analysis were considered as possible explanations for this finding.

EFFECT OF AGE AND TESTING PROTOCOL ON THRESHOLD EXERCISE INTENSITY

Although absolute values for power, heart rate and $\dot{V}O_2$ at thresholds were higher in seniors cyclists there was no difference between age groups for blood lactate at TLac threshold. Pedal cadence was similar between age groups at OBLA but not TLac and relative values for $\%SRM_{Wpeak}$, $\%HR_{peak}$ and $\%\dot{V}O_{2peak}$ were similar between groups except for $\%SRM_{Wpeak}$ at TLac. Consequently the age of the rider needed to be considered when evaluating variables associated with TLac.

Testing protocol affected power, heart rate and oxygen uptake at threshold exercise intensity but not blood lactate concentration and pedal cadence. There was no interactive difference between age and test, therefore effects of testing method were independent of age. These findings had important implications for the selection of an appropriate blood lactate derived threshold and testing method to assess the endurance capacity of senior and veteran cyclists. Methodological issues regarding subject selection and cross sectional analysis were also highlighted with particular reference to comparisons between age groups and data interpretation.

EFFECT OF AGE ON LABORATORY BASED CYCLING PERFORMANCE

Senior and veteran cyclists maintained a similar relative exercise intensity during a laboratory based 16.1-km cycling time trial. There was no difference between groups for blood lactate response assessed during the time trial, however mean value for blood lactate concentration was higher (P = 0.08) in the seniors. Economy for the duration of the time trial was lower in the veterans. These findings did concur with previous work which recorded lower values for blood lactate concentration in older athletes and a higher relative exercise intensity at race pace in veteran runners.

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EFFECT OF AGE ON FIELD BASED CYCLING PERFORMANCE

During a field based 16.1-km time trial veteran cyclists maintained a higher relative exercise intensity when compared with; senior cyclists, and a laboratory based ride. There was no difference in relative heart rate response for indoor and outdoor rides or between groups, however seniors maintained a higher pedal cadence indoors but not outdoors. Blood lactate concentration on completion of the indoor ride was higher for the seniors but there was no difference between groups for the outdoor trial. These findings highlighted important methodological problems associated with using indoor laboratory based tests to investigate the effect of age on cycling endurance performance and questioned the validity of indoor tests to evaluate cycling performance.

CORRELATES OF PERFORMANCE RELATED RESPONSES DURING CYCLING TIME TRIALS IN SENIORS AND VETERANS

Data collected in the present study revealed the underlying relationship between W_{peak} and 16.1-km time trial power and the inter-relationship between W_{peak} and $\dot{V}O_{2peak}$. Although variables recorded during a laboratory based simulated 16.1-km time trial and blood lactate parameters assessed during progressive exercise protocols provided valid predictors of outdoor power and heart rate. No discernible difference was found between the different methods used in this study.

Peak values for power, heart rate and oxygen uptake recorded during a single peak aerobic capacity test were valid predictors of average power and heart rate recorded during a field based 16.1-km time trial and average oxygen uptake during an indoor 16.1-km time trial. This finding supported the postulate that the determination of threshold exercise intensity was not necessary for the prediction of performance related responses during cycling time trials and that peak power was a strong correlate to cycling performance.

VALIDITY OF W_{PEAK} TO PREDICT OUTDOOR PERFORMANCE POWER BUT NOT PERFORMANCE TIME IN SENIORS AND VETERANS

Peak power recorded during a Kingcycle peak power test predicted mean power but not performance time during a field based 16.1-km time trial. Individual differences in body size and the effects of aerodynamics could account for this finding. Investigators need to be

aware that mean performance power and peak power provide a valid assessment of endurance performance when cyclists participate in separate time trial races performed under different environmental conditions.

OVERALL SUMMARY

This thesis investigated the use of physiological tests to assess the cycling endurance performance ability of senior and veteran competitors. Key issues concerning the reproducibility and validity of equipment and methods were addressed in order to establish an appropriate valid and reproducible method to investigate age related changes in endurance capacity and performance and *compare senior and veteran cyclists*. Methodological problems associated with using cross sectional methods of analysis were highlighted on several occasions and illustrated the need for caution when inter-individual comparisons between senior and veteran cyclists are completed.

The major findings of this thesis were that W_{peak} assessed during a non-invasive ramped peak power test was highly reproducible and afforded a valid method to assess inter-individual differences in cycling performance ability regardless of age. The attainment of a high W_{peak} appears to be a key determinant of successful endurance performance and therefore should be considered a key factor when investigating age related declines in endurance capacity and endurance performance. Central factors such as age related declines in peak heart rate and ventilation and peripheral changes in muscle fibre structure and function could explain age related declines in peak oxygen consumption, W_{peak} and endurance performance. The factors which determine W_{peak} warrant further investigation in order to identify the underlying mechanisms responsible for variations in cycling endurance performance due to the ageing process and the effects of training/detraining.

13 FUTURE RESEARCH

- Investigate central and peripheral factors which determine W_{peak}.

- Assess whether W_{peak} is sensitive to changes in performance power.

- Study the effects of training interventions on W_{peak} and performance power.

- Investigate the relationship between muscle fibre composition and aerobic/anaerobic exercise performance in senior and veteran cyclists.

- Examine the effects of respiratory muscle work on exercise performance in veteran cyclists.

14 APPENDICES

14.1 INFORMED CONSENT FORM

Subject Number ....

Subject name: .... Sex: Male/Female

Supervisor: ....

Investigator: ....

Project Title:

....

....

Brief description of study:

....

....

....

....

....

....

....

....

....

I fully understand what is involved in taking part in this study. Any questions I have about the study, or my participation in it, have been answered to my satisfaction. If I decide to withdraw I understand that it will not have any undesirable consequences. It has been made clear to me that should I feel that my interests are ignored, neglected or denied, I should inform the head of the department of sport science who will undertake to investigate my complaint.

Signed .... Date ....

I certify that the details of this study have been explained and described in writing to .... and have been understood by him/her and that I consent to his/her participation in this study.

Signed .... Date ....
(parental consent for minors only)

TRAINING AND RACING QUESTIONNAIRE

ALL INFORMATION PROVIDED WILL BE TREATED WITH THE STRICTEST CONFIDENCE

NAME   _____

ADDRESS   .   _____

                 _____

                 _____   POST CODE   _____

TEL No   _____

CYCLING CLUB   _____   AGE (IN YEARS)   _____

1. How many years have you been involved in cycling (also please state number of years you have continuously raced/trained) ?

_____

2. How many races did you ride last season (please circle) ?

0-5 races   5-15 races   15-30 races   30-45 races   45+ races

3. What was your personal best time for 1995 in 10 and 25 mile time trials if applicable (please circle) ?

10 miles -	20-22 min	25 miles	50-55 min
	22-24 "		55-60 "
	24-27 "		60-65 "
	27+ "		67-70 "
			70+ "

4. What distance would you normally cover during your most preferred road race, if applicable (please circle) ?

20-40 miles   40-60 miles   60-80 miles   80+ miles

Do you keep a training diary (please circle) ?

YES OR NO

The table below is designed to identify the level of intensity you train at.

Each level in the table should represent your average heart rate response during your training ride(s).

level A	between 15-25 beats per minute below your maximum heart rate
level B	between 25-35 beats per minute below your maximum heart rate
level C	between 35-45 beats per minute below your maximum heart rate
level D	more than 45 beats per minute below your maximum heart rate

please state the level of intensity you train at, as either:-   A   B   C   or   D

If you keep a training diary please include as much information as possible for each week highlighted below.

If you do not keep a training diary please include as much information as you can rememeber for a typical week during that period of the year.

PREVIOUS AND CURRENT TRAINING and RACING SCHEDULE

please list your training and racing schedule during the **1st week of June 1995.**

If applicable also include additional exercise such as weight training, stretching etc

	TRAINING/RACING	DURATION (TIME)	INTENSITY
MONDAY			
TUESDAY			
WEDNESDAY			
THURSDAY			
FRIDAY			
SATURDAY			
SUNDAY			

PLEASE LIST your training and racing schedule during the **1st week of September 1995.**

	TRAINING/RACING	DURATION (TIME)	INTENSITY
MONDAY			
TUESDAY			
WEDNESDAY			
THURSDAY			
FRIDAY			
SATURDAY			
SUNDAY			

PLEASE LIST your training schedule during the **1st week of December 1995.**

	TRAINING/RACING	DURATION (TIME)	INTENSITY
MONDAY			
TUESDAY			
WEDNESDAY			
THURSDAY			
FRIDAY			
SATURDAY			
SUNDAY			

please list your **CURRENT** training and racing schedule. DATE = _____

	TRAINING/RACING	DURATION (TIME)	INTENSITY
MONDAY			
TUESDAY			
WEDNESDAY			
THURSDAY			
FRIDAY			
SATURDAY			
SUNDAY			

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