Pulmonary Oxygen Uptake Kinetics in Middle- and Long-Distance Runners

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Abstract

The overall aim of this thesis was to evaluate the importance of pulmonary oxygen uptake ($\dot{V}O_2$) kinetics, in the moderate-domain, in the assessment of endurance-trained runners. Accordingly, there were five objectives: 1) to quantify the reproducibility of measures of $\dot{V}O_2$ kinetics; 2) to characterise and compare $\dot{V}O_2$ kinetics during the on- and off-transients in middle-distance (MD) and long-distance (LD) runners; 3) to assess the relationship between $\dot{V}O_2$ kinetics and maximal $\dot{V}O_2$ ($\dot{V}O_{2\,max}$), ventilatory threshold (V_T) and running economy (RE); 4) to determine the relationship between $\dot{V}O_2$ kinetics and running performance and 5) to assess whether $\dot{V}O_2$ kinetics is a determinant of running performance.

Twelve participants performed two assessments of $\dot{V}O_2$ kinetics on separate days to determine the reproducibility. Paired *t*-tests showed that parameters from test 1 and test 2 did not differ ($P > 0.05$). Furthermore, narrow 95% limits of agreement (LOA), low measurement and method error suggested that the on- and off-transient time-constants (τ_{on} and τ_{off}), mean response times (MRT_{on} and MRT_{off}) and amplitudes (A_{on} and A_{off}) were reproducible and could be used for the assessment of runners. Subsequently, $\dot{V}O_2$ kinetics were compared in 10 MD and 10 LD runners. There was a tendency for τ_{on} (12.5 ± 2.3 s vs. 14.2 ± 3.1 s, $P = 0.178$) and τ_{off} (24.1 ± 2.3 s vs. 27.1 ± 3.0 s, $P = 0.023$) to be shorter in LD than MD runners respectively, despite similar $\dot{V}O_{2\,max}$ (MD = 60.0 ± 4.9 ml·kg^{-1}·min^{-1}; LD = 59.0 ± 6.3 ml·kg^{-1}·min^{-1}, $P = 0.689$). Differences in $\dot{V}O_2$ kinetics between MD and LD runners were attributed to approaches to training since the volume of training was greater in LD (64.0 ± 15.7 km·wk^{-1}) than MD (47.5 ± 15.7 km·wk^{-1}) runners ($P = 0.047$). To detail the relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function ($\dot{V}O_{2\,max}$, V_T and RE), 16 MD and 16 LD runners were assessed. Relationships existed between τ_{on} and $\dot{V}O_{2\,max}$ ($r = -0.72$, $P = 0.002$), V_T ($r = -0.66$, $P = 0.006$) and RE ($r = -0.59$, $P = 0.016$) in LD runners, but not in MD runners ($P > 0.05$). In addition, τ_{on} was related to the volume of training in MD ($r = -0.63$, $P = 0.009$) and LD runners ($r = -0.65$, $P = 0.006$).

The importance of $\dot{V}O_2$ kinetics for 5 km running performance was investigated in 36 endurance trained runners. Runners were categorised as high (n=10), low (n=10) and combined [MD + LD (n=36)] performers according to running ability after performing a self-paced 5 km time-trial. Mean (±SD) speed for the 5 km time-trial was 5.2 ± 1.0 m·s^{-1} (high), 4.5 ± 0.2 m·s^{-1} (low) and 4.9 ± 0.3 m·s^{-1} (combined). Measures of on- and off-transient $\dot{V}O_2$ kinetics, $\dot{V}O_{2\,max}$, V_T and RE were also determined. Data were explored using bi-variate correlations, ANCOVA and multiple regression techniques. In high and low performers, $\dot{V}O_2$ kinetic parameters were not related to running performance. In combined runners, τ_{on}, τ_{off}, MRT_{on} and MRT_{off} were related ($r = -0.54$, $P = 0.001$; $r = -0.36$, $P = 0.030$; $r = -0.50$, $P = 0.002$; $r = -0.63$, $P = 0.003$) to running performance. Stepwise multiple regression models were used to identify the primary determinant(s) of 5 km running performance for each group. In high performers, $\dot{V}O_{2\,max}$ and RE were included in the model ($r = 0.92$, $R^2 = 0.85$, SEE = 0.08 m·s^{-1}; SEE% = 1.5). In low performers, $\dot{V}O_{2\,max}$ was included in the model ($r = 0.76$, $R^2 = 0.57$, SEE = 0.15 m·s^{-1}, SEE% = 3.3). In combined runners, $\dot{V}O_{2\,max}$, RE and MRT_{off} were included in the model ($r = 0.87$, $R^2 = 0.75$, SEE = 0.17 m·s^{-1}, SEE% = 3.5).

Collectively, the results suggest that: 1) $\dot{V}O_2$ kinetics can be reproducibly determined using a single visit protocol; 2) measures of $\dot{V}O_2$ kinetics are sensitive enough to differentiate MD and LD runners; 3) relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function exist in LD runners, but not in MD runners; 4) $\dot{V}O_2$ kinetics differ between high and low performers, but do not relate to running performance and 5) $\dot{V}O_2$ kinetics discriminate between high and low performers but only contribute minimally to the prediction of running performance in a multiple regression model for combined MD and LD runners.

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Presentations at Conferences

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Contents

List of Symbols and Abbreviations

ADP	adenosine diphopshate
A_{on}	amplitude of $\dot{V}O_2$: on-transient
A_{off}	amplitude of $\dot{V}O_2$: off-transient
ANCOVA	analysis of covariance
AT	anaerobic threshold
ATP	adenosine triphosphate
ATPase	adenosine triphosphatase
BASES	British Association of Sport and Exercise Sciences
BM	body mass
$C(a\text{-}v)O_2$	arterial-venous O_2 content difference
Cr	creatine
C_r	O_2 cost of running
CS	citrate synthase
CK	creatine kinase
CV	coefficient of variation
CO	cytochrome *c* oxidase
CO_2	carbon dioxide
δ_{on}	time delay: on-transient
δ_{off}	time delay: off-transient
DCA	dichloroacetate
ETC	electron transport chain
ELV	effective lung volume
FAD	flavin-adenine dinucleotide (oxidised form)
$FADH_2$	flavin-adenine dinucleotide (reduced form)
FIO_2	fractional concentration of O_2 in total inspired gas
FRC	functional residual capacity
H^+	hydrogen ion or proton
HCO_3^-	bicarbonate
HLa	blood lactate
HR	heart rate
HR_{max}	maximum heart rate
HR_{res}	heart rate reserve
kg	kilogram
l	litre
LD	long-distance
LDH	lactate dehydrogenase
LOA	limits of agreement
LT	lactate threshold
MD	middle-distance
ME	method error
min	minute: unit of time
ml	millilitre
MLa	muscle lactate
mmol	millimole
MRT_{on}	mean response time: on-transient
MRT_{off}	mean response time: off-transient
ms	millisecond: unit of time
NAD^+	nicotinamide-adenine dinucleotide (oxidised form)
NADH	nicotinamide-adenine dinucleotide (reduced form)

NLV	nominal lung volume
NMR	nuclear magnetic resonance spectroscopy
O_2	oxygen molecule
PCO2	partial pressure of CO_2
PCr	phosphocreatine or creatine phosphate
PDH	pyruvate dehydrogenase
$PETCO_2$	partial pressure end tidal CO_2
$PETO_2$	partial pressure end tidal O_2
PFK	phosphofructokinase
Pi	inorganic phosphate
^{31}P-MRS	phosphorous nuclear magnetic resonance spectroscopy
PO	power output
PO_2	partial pressure of oxygen
$\dot{Q}$	cardiac output
$\dot{Q}_{leg}$	leg blood flow
$\dot{Q}_{max}$	maximum cardiac output
$\dot{Q}O_2$	rate of muscle O_2 consumption
RE	running economy
RER	respiratory exchange ratio
s	second: unit of time
SD	standard deviation
SDH	succinate dehydrogenase
S_0	SD of the breath-by-breath noise
STPD	standard temperature and pressure, dry (0°C, 760 mmHg)
SV	stroke volume
t	time
TCA	tricarboxylic acid cycle
τ_{on}	time constant (tau): on-transient
τ_{off}	time constant (tau): off-transient
$\dot{V}CO_2$	rate of carbon dioxide production
$\dot{V}E$	minute ventilation
$\dot{V}O_{2\ t½}$	time to reach one half of the final oxygen uptake response
$\dot{V}O_2$	rate of oxygen uptake
$\dot{V}O_{2\ (b)}$	baseline oxygen uptake
$\dot{V}O_{2\ (m)}$	moderate-intensity (80%V_T) oxygen uptake
$\dot{V}O_{2\ max}$	maximal oxygen uptake
$\dot{V}O_{2\ peak}$	highest rate of oxygen uptake achieved during an incremental test
V_T	ventilatory threshold
Δ	the change in
[ ]	concentration

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CHAPTER 1

Introduction

1.1 Introduction

Developments in the physiology of exercise have made significant contributions to improvements in sporting performance, in particular to running performance. A major factor in this improvement has been advances in the physiological assessment of runners. Specifically, physiological assessments under controlled environmental laboratory conditions provide an opportunity to assess: 1) current physiological status; 2) adaptations to training and 3) performance capabilities. Importantly, physiological assessments also assist in the design and development of training methods that are specific to the physiological demands of the event (Kindermann *et al.*, 1979; Billat, 2001a, b). Indeed, the continuous improvement in performance in running events can be partially attributed to the scientific approach to training in order to maximise physiological adaptation.

Exercise testing with appropriate pulmonary gas-exchange measurements offers the possibility of simultaneous study of cellular, cardiovascular and ventilatory responses under conditions of precisely controlled metabolic stress (Wasserman *et al.*, 1994). According to Whipp *et al.* (1981), four gas-exchange measurements make up the 'aerobic' profile of a performer: 1) maximal oxygen uptake ($\dot{V}O_{2\,max}$); 2) anaerobic threshold (AT); 3) work efficiency (oxygen cost of exercise) and 4) oxygen uptake ($\dot{V}O_2$) kinetics. Any attempt to discriminate between performers or to predict performance capability should consider these four measures of aerobic function (Whipp *et al.*, 1981). However, physiological assessments of endurance-trained runners have primarily involved the measurement of only three of the four proposed aerobic variables, with minimal consideration of measures of $\dot{V}O_2$ kinetics.

Measures of pulmonary $\dot{V}O_2$ kinetics reflect the temporal profile of muscle O_2 consumption ($\dot{Q}O_2$) at the onset of exercise or to a sudden change in the intensity of exercise (Barstow *et al.*, 1987; Grassi *et al.*, 1996). Therefore, in healthy humans, measures of $\dot{V}O_2$ kinetics provide valuable information about the oxidative potential of muscle, rather than the integrated functioning of the cardiovascular, pulmonary and muscular systems (Poole and Richardson, 1997), that is reflected in the measurement of whole-body $\dot{V}O_{2\,max}$. Potentially, this could be a useful tool with which to assess the oxidative qualities of muscle in athletes. Evidence to support the use of measures of $\dot{V}O_2$ kinetics in athletes is provided in several studies that have shown that pulmonary $\dot{V}O_2$ kinetics during the on- (Hagberg *et al.*, 1980; Berry and Moritani, 1985) and off-transient (Hagberg *et al.*, 1980; Phillips *et al.*, 1995) are sensitive to training stimuli. Furthermore, measures of $\dot{V}O_2$ kinetics appear to be more sensitive to training than both $\dot{V}O_{2\,max}$ and the ventilatory threshold (V_T) (Fukuoka *et al.*, 1995; Phillips *et al.*, 1995). Therefore, it appears that $\dot{V}O_2$ kinetics might reflect physiological adaptation(s) in the muscle more precisely than other measures such as $\dot{V}O_{2\,max}$ and V_T (or lactate threshold, LT) and could be used to compare athletes with different training backgrounds.

The endurance training studies above considered measures of $\dot{V}O_2$ kinetics in the moderate-domain. Since the majority of studies, but not all (Carter *et al.*, 2002), have demonstrated that the time constant (τ) of the phase II $\dot{V}O_2$ response is invariant with increasing exercise intensity (Barstow *et al.*, 1993; Özyener *et al.*, 2001; Wells *et al.*, 2003), it would be appropriate to use moderate-intensity exercise to establish $\dot{V}O_2$ kinetics in endurance-trained runners. However, in acknowledgment of potential differences in τ above and below V_T, support for using moderate-intensity exercise is gained from information about the recruitment of muscle fibres during exercise below V_T. It is likely that only Type I fibres are recruited during moderate-intensity exercise (Vøllestad and Blom, 1985) and that these fibres predominate (>70%) in the muscles of endurance-trained athletes (Saltin and Gollnick, 1983). Therefore, measures of $\dot{V}O_2$

kinetics in the moderate-domain are likely to reflect, non-invasively, the oxidative potential of these important muscle fibres. Information about the oxidative function of exercising muscle is likely to be obscured during assessments of $\dot{V}O_2$ kinetics in the heavy-intensity domain since τ is influenced, at least in part, by muscle O_2 delivery (Tschakovsky and Hughson, 1999). This would make it difficult to exclusively attribute potential differences between athletes to muscle oxidative function. Rather, measures of $\dot{V}O_2$ kinetics in the heavy-intensity domain would provide an 'overall' reflection of the body's potential to both transport and utilise oxygen. Potentially, this might provide similar information already gained from measures of $\dot{V}O_{2\,max}$.

Because endurance-trained runners are likely to differ in their approach to training, depending on their preferred discipline, this might be reflected in their $\dot{V}O_2$ kinetic response during moderate-intensity exercise. It is well established that different frequencies, durations and intensities of training influence peripheral adaptations in muscle (Henriksson and Reitman, 1976; Harms and Hickson, 1983). Given that a greater volume of training is usually performed by long-distance (LD) runners (Costill *et al.*, 1976b), this might influence $\dot{V}O_2$ kinetics compared to middle-distance (MD) runners who perform high-intensity and low volumes of training. Accordingly, it could be hypothesised that a greater volume of aerobic training would improve the efficiency of oxidative metabolic regulation which would ultimately result in faster $\dot{V}O_2$ kinetics. This possibility warrants the separation of endurance-trained runners into MD and LD runners. If differences were apparent, this would support further work to explore relationships between training, physiological and biochemical adaptations and $\dot{V}O_2$ kinetics in more detail. If this revealed that physiological adaptation(s) in muscle caused by differing approaches to training (volume and intensity) could be quantified using measures of $\dot{V}O_2$ kinetics, this might offer an alternative approach to assess athletes.

The majority of $\dot{V}O_2$ kinetic related studies, including those involving runners, have used cycle ergometry (deVries *et al.*, 1982; Powers *et al.*, 1985). In consideration of the principle of specificity, it is more appropriate to measure $\dot{V}O_2$ kinetics in athletes according to their predominant mode of exercise. Acknowledgement of the effect(s) of different modes of exercise is important, especially because active and non-active muscle fibre compartments might display blood flow (Grassi *et al.*, 1996) and $\dot{Q}O_2$ heterogeneity (Whipp *et al.*, 2002), which might influence the underlying kinetic response measured at the mouth. Recently, however, treadmill ergometry has been successfully used to characterise both the on- (Williams *et al.*, 2001; Carter *et al.*, 2002a) and off-transient (Carter *et al.*, 2000a) $\dot{V}O_2$ kinetics in the moderate-domain in untrained individuals. This mode of exercise would be more appropriate for the assessment of $\dot{V}O_2$ kinetics in runners and would provide a more appropriate characterisation of $\dot{V}O_2$ kinetic responses than cycle ergometry.

The consideration of both on- and off-transient $\dot{V}O_2$ kinetics would permit an assessment of the symmetry and relationship between transients. The magnitude of symmetry between transitions has direct implications on whether mechanism(s) determining respiratory control are operating as a first-order linear system. Since most assessments of the symmetry between transients have been considered in un-trained individuals (e.g. Özyener *et al.*, 2001), it has yet to be established whether physiological adaptations in muscle (e.g. increased number of mitochondria, enhanced oxidative enzyme activity and increased capillarity; Saltin and Gollnick, 1983), caused by habitual training, distorts the symmetry between transients. If so, the proposed first-order linear model of respiratory control in skeletal muscle (Meyer, 1988; Meyer and Foley, 1994) would be refuted. Additionally, a lack of relationship between on- and off-transient $\dot{V}O_2$ kinetics would suggest that different physiological mechanisms are being reflected by each transient. Consequently, the on- and off-transient $\dot{V}O_2$ kinetics would need to be considered as two separate measures that provide different information about the physiological status of the muscle. This has not been considered

in any previous study of $\dot{V}O_2$ kinetics. Measurement of the off-transient $\dot{V}O_2$ kinetics might also provide useful information about how recovery mechanisms [primarily phosphocreatine (PCr) re-synthesis and the replenishment of O_2 and myoglobin stores; Gaesser and Brooks, 1984] operate in athletes who differ in their approach to training, and in particular whether off-transient $\dot{V}O_2$ kinetics are sensitive enough to differentiate between runners of different running disciplines.

There are three measures of aerobic function that are commonly used to physiologically assess (Londeree, 1986) and determine running performance (Joyner, 1991) in runners: 1) $\dot{V}O_{2\,max}$; 2) V_T/LT and 3) running economy (RE). The physiological determinants and underpinning mechanism(s) of each are well-documented (Brooks, 1991; Wagner, 1996). However, it is relatively unclear whether measures of $\dot{V}O_2$ kinetics provide similar, or additional, information about the muscle as these other measures. For example, whole-body $\dot{V}O_{2\,max}$ is determined by both central (O_2 delivery) and peripheral (O_2 utilisation) mechanisms. It is unclear whether fast $\dot{V}O_2$ kinetics (i.e. shorter τ_{on}) are synonymous with a high $\dot{V}O_{2\,max}$. A relationship between these measures might suggest that both $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics are influenced by similar determining factors. However, considering that $\dot{V}O_2$ kinetics has been shown to be more sensitive to training than $\dot{V}O_{2\,max}$ and V_T (Fukuoka *et al.*, 1995; Phillips *et al.*, 1995), then relationships might not be apparent. This also applies to RE which is frequently used to physiologically assess runners. Therefore, exploring potential relationships between $\dot{V}O_2$ kinetics and these measures, and considering the mechanism(s) underpinning them, could provide a useful insight into the independency of aerobic measures and adaptations, in particular those concerning $\dot{V}O_2$ kinetics.

Most importantly, however, is whether measures of $\dot{V}O_2$ kinetics are related to running performance. Several studies have investigated physiological measures which could potentially determine running performance. However, no study has considered measures of on- or off-transient $\dot{V}O_2$ kinetics as potential determinants of running

performance. This is surprising since $\dot{V}O_2$ kinetics in the moderate-domain reflects the ability of muscle to utilise oxygen and is sensitive to training. In support of the use of $\dot{V}O_2$ kinetics as a determinant of performance, a previous study has shown that changes in $\dot{V}O_2$ kinetics are more reflective of improvements in cycling performance than $\dot{V}O_{2\,max}$ and V_T (Norris and Peterson, 1998). However, no investigation has attempted to quantify relationships between $\dot{V}O_2$ kinetics and running performance. If significant relationships were identified, this would justify the inclusion of a measurement of $\dot{V}O_2$ kinetics to evaluate the physiological status of runners. This has the potential to replace other tests which might be invasive, less sensitive and/or disruptive to training, lack reproducibility and have a weaker correlation with running performance.

Measurement of $\dot{V}O_2$ kinetics is time-consuming because the attainment of accurate kinetic parameter estimations requires several visits to the laboratory. This is primarily due to inherent variability of breath-by-breath data that can significantly influence kinetic parameter estimations (Lamarra *et al.*, 1987). To attenuate this variability, participants have to complete several bouts of exercise usually over several days. However, this is impractical for endurance-trained runners and would be disruptive to training. Therefore the development of a protocol to measure $\dot{V}O_2$ kinetics in one visit to the laboratory would be beneficial. This would require an assessment of its reproducibility which would provide some indication of the day-to-day variability of $\dot{V}O_2$ kinetics. Reproducible measures of $\dot{V}O_2$ kinetics is essential if 1) athletes are to be accurately compared; 2) relationships between $\dot{V}O_2$ kinetics and other physiological measures are to be meaningfully explored and 3) $\dot{V}O_2$ kinetics is to be considered a determinant of running performance.

1.2 Aim and objectives

The overall aim of this work is to evaluate the importance and relevance of $\dot{V}O_2$ kinetics in the assessment of aerobic performance in endurance-trained runners. Accordingly, there are five specific objectives:

1. To establish and quantify the reproducibility of a protocol for the assessment of $\dot{V}O_2$ kinetics during treadmill running in MD and LD runners.
2. To characterise and compare $\dot{V}O_2$ kinetics during the on- and off-transients in MD and LD runners.
3. To assess the relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$, V_T and RE.
4. To assess the relationship between $\dot{V}O_2$ kinetics and 5 km running performance.
5. To determine the primary aerobic factors ($\dot{V}O_{2\,max}$, V_T, RE and $\dot{V}O_2$ kinetics) contributing to successful 5 km running performance.

CHAPTER 2

Review of literature

2.1 Historical background

Competitive MD and LD running has its origins in ancient times and is primarily associated with the ancient Olympic Games (Swaddling, 1999). These Games were first held in 776 BC as a religious, sporting and cultural festival in honour of Zeus, the father of the gods. Initially, the ancient Olympic Games involved only a short race (*stade*) that involved running the length of the stadium, which was approximately 192 m. However, the program of competitive events in the Games evolved over time and in 724 BC, the two-stade race (*diaulos*, 384 m) was introduced. By 720 BC, longer races [7 and 27 stades (*dolichos*)] were also held over distances between 1344 to 4608 m. There is also evidence of a four-stade race (768 m) which would be equivalent to the 800 m, although athletes were required to wear body armour. The ancient Olympic Games are known to have existed for 12 centuries until their demise in 394 A.D, which was attributable to the disintegration of the nationalistic and religious unity of the Greeks (Swaddling, 1999).

The symbolic power of the ancient Olympic Games was revived as the modern Olympic Games which first took place in Athens in 1896 and encompassed a greater range of distance races [100 m to the marathon (42.2 km)]. The standardisation of these distances from 1896 to present time, in addition to accurate measurements of performance times has permitted a longitudinal analyses of running performance via the progression of world-best times.

Parallel with the internationalisation of athletics has been the development of the physiology of exercise. Since the early part of the 20th century, scientists have developed growing interest in the body's responses and adaptations to exercise. Many concepts concerning these responses to exercise were first established by the pioneering

work of A.V. Hill, British Professor of Physiology, in a series of studies on exercise, lactic acid and the supply and utilisation of oxygen (Hill and Lupton, 1923). These early experiments clearly elucidated the concepts of maximal oxygen uptake ($\dot{V}O_{2\,max}$), steady-state exercise and oxygen debt. Other studies performed by Hill and his collaborators focused on the physiological responses during exercise, especially in MD and LD runners (Hill, 1965)

2.2 Characterisation of MD and LD running

Insights into the physiological requirements of MD and LD running can be obtained by considering: 1) the primary energy pathways that are utilized and 2) the intensity domains of exercise. The following will explain both of these considerations in more detail.

2.2.1 Energy production during running

The function of muscle is to exert force. The immediate source of energy for muscle during running and other forms of exercise is provided by the synthesis of the high-energy phosphate compound, adenosine tri-phosphate (ATP) in the reaction:

$$ATP \xrightarrow{ATPase} ADP + P_i + energy \qquad (1)$$

However, only a small quantity of ATP is stored in the cell; hence, it must be re-synthesised at the rate it is used to allow muscular activity to continue. This scenario provides a sensitive mechanism for regulating energy metabolism in the cell. There are three main energy producing pathways which interact to maintain the supply of ATP for muscular contraction: 1) ATP-PCr; 2) anaerobic glycolysis and 3) aerobic glycolysis (or oxidative phosphorylation) (Figure 2.1).

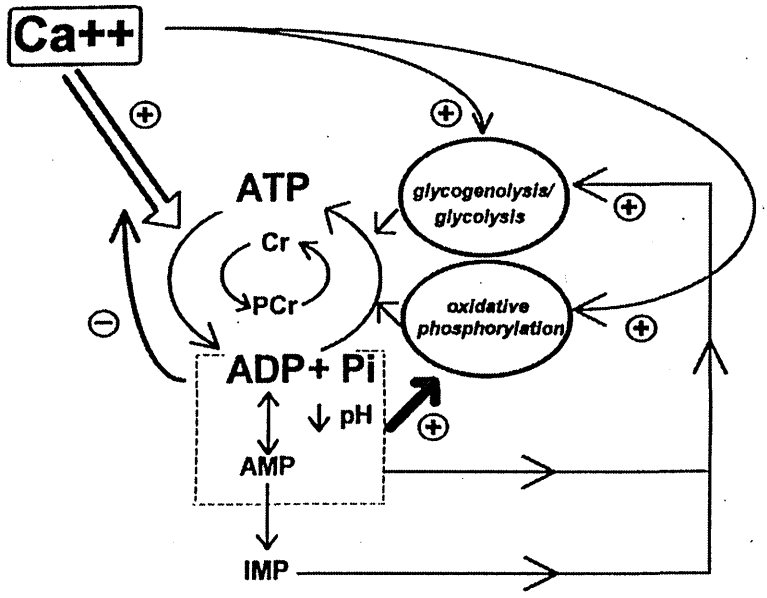


Figure 2.1 Schematic illustrating the three major pathways for ATP re-synthesis in skeletal muscle (Meyer and Foley, 1996).

The re-synthesis of ATP can be achieved by combining adenosine di-phosphate (ADP) and inorganic phosphate (P_i) via the creatine kinase (CK) reaction in the cytoplasm of the cell:

$$ADP + PCr + H^+ \xleftrightarrow{\text{creatine kinase}} ATP + Cr \qquad (2)$$

This pathway provides immediate energy for muscular contraction at the onset of exercise and during short-term, high-intensity exercise. However, because the storage of PCr in muscle is limited and can only be maintained in the short-term, a greater contribution from other simultaneously operating metabolic pathways (aerobic and anaerobic glycolysis) is required to regenerate ATP.

The activation of anaerobic glycolysis occurs almost instantaneously at the onset of exercise and involves the re-synthesis of ATP via the degradation of glucose (or glycogen) to pyruvate in the cytoplasm of the cell. However, when glycolytic flux

exceeds mitochondrial activity, as observed at the onset of exercise or during heavy-intensity exercise, pyruvate is subsequently converted to lactate, where:

$$\text{glucose} + 2\ \text{ADP} + 2\ \text{P}_\text{i} \rightarrow 2\ \text{lactate}^- + 2\ \text{H}^+ + 2\ \text{ATP} \quad (3)$$

Importantly, the net formation of lactate or pyruvate depends on relative glycolytic and mitochondrial activities and not on the presence of oxygen (Brooks *et al.*, 2000).

The third pathway for regenerating ATP is aerobic glycolysis which starts in a similar manner to that of anaerobic glycolysis. That is, glucose or glycogen are converted to pyruvate. However, because glycolytic flux does not exceed mitochondrial activity, lactate is not formed and allows oxidative phosphorylation to take place in the mitochondria. The final reaction of oxidative phosphorylation is:

$$\text{NADH} + \tfrac{1}{2}\text{O}_2 + \text{H}^+ + 3\ \text{ADP} + 3\ \text{Pi} \rightarrow 3\ \text{ATP} + \text{NAD}^+ + \text{H}_2\text{O} \quad (4)$$

There are two major metabolic pathways involved in oxidative phosphorylation: 1) the tricarboxylic acid cycle (TCA), which breaks down acetyl units derived from fuel molecules and generates the reduced coenzymes nicotinamide adenine dinucleotide (NADH) and flavin adenine dinucleotide ($FADH_2$) as well as CO_2 and 2) the electron transport chain (ETC) where free energy, released when electrons are transferred from NADH and $FADH_2$ to O_2, gets channelled into the phosphorylation of ADP to make ATP, that is, it drives the reaction:

$$\text{ADP} + \text{P}_\text{i} \rightarrow \text{ATP} + \text{H}_2\text{O} \quad (5)$$

During electron transfer from NADH and $FADH_2$ to O_2, the free energy released is employed to pump protons (H^+) from the matrix side of the inner membrane of the mitochondria to the outer side or cytosolic side thus creating an electrochemical

gradient. When protons return down the gradient, the free energy released is used to re-synthesise ATP from ADP and P_i.

It is important to emphasise the interaction of anaerobic and aerobic metabolic pathways in the re-synthesis of ATP during exercise. During running, the contribution of each pathway differs according to the intensity and duration of the event. For example, as the distance of the event increases, the contribution of aerobic energy producing pathways (i.e. oxidative phosphorylation) increases. Consequently, there is less reliance on anaerobic pathways (i.e. anaerobic glycolysis). With respect to running, the contribution of anaerobic pathways is greatest (~90%) in short-distance events such as the 100 m (Åstrand and Rodahl, 1986). Conversely, aerobic pathways predominate (~94%) in LD events such as the marathon (Wood, 1999). Perhaps surprisingly, aerobic pathways have also been found to contribute significantly to MD events such as the 800 m (67%) and 1500 m (83%) (Hill, 1999). Although the anaerobic contribution to the overall performance in LD events appears to be negligible, its contribution is still important at the start and end of competitive races.

2.2.2 Intensity domains of exercise

The terms sub-maximal and supra-maximal are often used to represent the intensity of exercise below and above $\dot{V}O_{2\,max}$ respectively. However, by considering pulmonary gas-exchange responses and blood acid-base status, it is more specific to classify the intensities of exercise as moderate (below V_T), heavy (between V_T and $\dot{V}O_{2\,max}$) and severe (above $\dot{V}O_{2\,max}$) (Whipp and Mahler, 1980; Whipp and Ward, 1982). Subsequently, running events can be categorised with respect to the intensity domain of exercise (Table 2.1).

Table 2.1 Categorisation of MD and LD events with respect to intensity of exercise in relation to $\dot{V}O_{2\,max}$.

Distance	Category	Intensity	Intensity Domain
800 - 1500 m	MD	Supra-maximal	Severe
3000 m	MD/LD	Sub-maximal	Heavy
5000 - 10000 m	LD		
Half-marathon			Heavy-moderate
Marathon			Moderate

Knowledge of the intensity, duration and energy requirements of an event can help identify the primary physiological mechanisms that are most likely to influence running performance. As a result, the physiological status of MD and LD runners has been assessed to determine such physiological characteristics.

2.3 Physiological assessments of MD and LD runners

Regardless of ability, MD and LD runners have the same aim: to improve their personal running performance through regular training. Their goal is underpinned by the challenge of maximising their own potential by developing specific physiological mechanisms that contribute to improved running performance. To identify physiological attributes that contribute to successful running performance, MD and LD runners have been subject to various laboratory-based assessments (Londeree, 1986). Primarily, these have involved the measurement of $\dot{V}O_{2\,max}$ (Åstrand, 1955; Saltin and Åstrand, 1967), blood lactate and ventilatory responses (Hollman, 1966; Farrell *et al.*, 1979; Powers *et al.*, 1983) and RE (Margaria *et al.*, 1963; Costill *et al.*, 1973; Daniels *et al.*, 1978). Collectively, these measures have been assessed with respect to their contribution towards the multiple regression modelling of optimal running performance and prediction of future world records in MD (Peronnet and Thibault, 1989) and LD events (Peronnet and Thibault, 1989; Joyner, 1991). However, in many instances running performance has not been fully accounted for by these measures. This suggests

that other physiological measures could have some contribution and which have yet to be considered with respect to running performance.

One such physiological characteristic that might be related to running performance is $\dot{V}O_2$ kinetics. This is the temporal profile of $\dot{V}O_2$ at the onset (or offset) of exercise. Compared to traditional measures, there have been few studies that have assessed $\dot{V}O_2$ kinetics in MD and LD runners and related them to running performance. According to Whipp *et al.* (1981), there are four gas-exchange measurements that make up the 'aerobic' profile of a performer: 1) $\dot{V}O_{2\,max}$; 2) AT; 3) work efficiency (or oxygen cost of exercise) and 4) $\dot{V}O_2$ kinetics. Any attempt to discriminate between performers or to predict performance capability should consider these four measures of aerobic function (Whipp *et al.*, 1981). In the following sections, each of these measures will be defined and examined with respect to underpinning mechanisms as well as methods of measurement. Importantly, the extent to which each relate to running performance will be considered.

2.4 Maximal oxygen uptake

Maximal oxygen uptake ($\dot{V}O_{2\,max}$) is defined as the maximum rate at which an individual can take up, transport and utilise oxygen at sea level (Åstrand and Rodahl, 1986). The $\dot{V}O_2$ is dependent on both cardiac output ($\dot{Q}$) and the arterial-venous O_2 content difference ($C(a\text{-}v)O_2$) and thus $\dot{V}O_{2\,max}$ represents maximum $\dot{Q}$ and $C(a\text{-}v)O_2$ as expressed in a rearrangement of the Fick equation (McArdle *et al.*, 2001):

$$\dot{V}O_{2\,max} = \dot{Q}_{max} \times C(a\text{-}v)O_{2\,max} \qquad (6)$$

Hill and Lupton (1923) first defined $\dot{V}O_{2\,max}$ and postulated that: 1) there is an upper limit to $\dot{V}O_2$; 2) there are inter-individual differences in $\dot{V}O_{2\,max}$ and 3) a high $\dot{V}O_{2\,max}$ is a pre-requisite for success in MD and LD running. With respect to the latter, it has been known for some time that $\dot{V}O_{2\,max}$ values in elite runners are exceptionally high

(Robinson *et al.*, 1937; Åstrand, 1955; Saltin and Åstrand, 1967; Billat *et al.*, 2001) with documented values of up to 82.0 $ml \cdot kg^{-1} \cdot min^{-1}$ (Saltin and Åstrand, 1967). Typical $\dot{V}O_{2\,max}$ values previously reported for elite male MD and LD runners are presented in Table 2.2. These data suggest that entry into the elite category for MD and LD performances is dependent upon having a $\dot{V}O_{2\,max}$ between 70 - 80 $ml \cdot kg^{-1} \cdot min^{-1}$ or, using allometric modelling, in excess of 300 $ml \cdot kg^{-0.67} \cdot min^{-1}$ (Nevill *et al.*, 2003).

Table 2.2 The $\dot{V}O_{2\,max}$ values for elite male MD and LD runners.

Reference	Distance	*n*	$\dot{V}O_{2\,max}$	
			($l \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}$)
Saltin and Åstrand (1967)	1500 m - 10 000 m	5	5.03	77.5
Boileau *et al.* (1982)	3000 m - 10 000 m	32	4.96	76.9
Svedenhag and Sjödin (1984)	800 m - 1500 m	5	4.87	71.9
	1500 m - 5000 m	6	4.94	75.3
	5000 m - 10 000 m	5	4.92	78.6
	10 000 m - marathon	5	4.86	73.9
Daniels and Daniels (1992)	800 m - 1500 m	13	5.00	72.5
	3000 m - 10 000 m	23	4.91	77.4
	Marathon	9	4.86	74.4
Morgan and Daniels (1994)	10 000 m	22	4.86	75.8
Billat *et al.* (2001)	Marathon	10	4.79	79.6
Nevill *et al.* (2003)	800 - 1500 m	11	5.10	75.8
	5000 m - marathon	10	4.82	77.4

2.4.1 Mechanisms determining $\dot{V}O_{2\,max}$

It is well accepted that there is a physiological limit to $\dot{V}O_{2\,max}$. However, the determining mechanism(s) involved are less well agreed (Rowell, 1986; Wagner, 1996; Bassett and Howley, 1997; 2000; Noakes, 1998). Potential limiting factors can be attributed to central or peripheral mechanisms (Figure 2.2). Central mechanisms

involve cardiac and pulmonary function and hence the transport and supply of O_2 to exercising muscle. Peripheral mechanisms relate to the utilisation of O_2 in the muscle and includes factors such as capillary density, fibre type composition, oxidative enzyme activity and mitochondrial content and function.

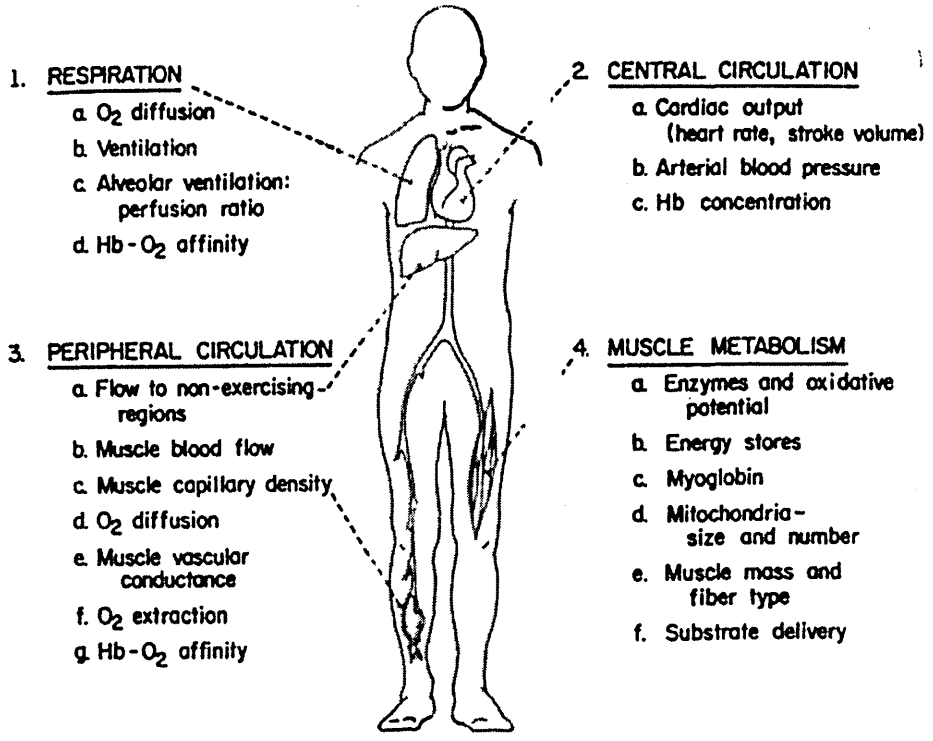


Figure 2.2 Potential physiological factors limiting $\dot{V}O_{2\,max}$ (Rowell, 1986).

Several approaches have been used to identify determining mechanism(s) of $\dot{V}O_{2\,max}$. Many studies have suggested that $\dot{V}O_{2\,max}$ is limited by the transport of O_2 to the exercising muscle (Reybrouck *et al.*, 1975; Knight *et al.*, 1993; Richardson *et al.*, 1995; 1999). Early evidence to support this was derived from studies combining arm and leg work (Reybrouck *et al.*, 1975; Secher *et al.*, 1977). Since arm exercise typically elicits only 65-75% of $\dot{V}O_{2\,max}$ determined by leg exercise alone, combined arm and leg exercise would be expected to result in a $\dot{V}O_{2\,max}$ higher than that seen in maximal leg exercise. However, $\dot{V}O_{2\,max}$ for combined arm and leg exercise has been shown to be

similar to leg exercise alone (Reybrouck *et al.*, 1975; Secher *et al.*, 1977). The imposition of arm work, whilst maintaining power output in the legs results in a decrease in leg blood flow and leg $\dot{V}O_2$ without a change in mean arterial pressure suggesting that blood flow to the exercising muscle is limited by vasoconstriction i.e. $\dot{Q}$ is unable to supply the demands of combined arm and leg exercise and still maintain blood pressure. The failure of combined arm and leg exercise to elicit further increases in $\dot{V}O_{2\,max}$ clearly suggests a limitation in the central cardiovascular system responsible for the delivery of O_2.

More detailed and direct evidence, obtained by blood gas and blood flow measurements during cycling (Knight *et al.*, 1993) and isolated knee extension exercise in cyclists (Richardson *et al.*, 1995; 1999), also suggests that O_2 supply is limiting $\dot{V}O_{2\,max}$. In these studies hyperoxia (1.0 inspired O_2 fraction [FIO_2]) has been shown to increase leg $\dot{V}O_{2\,max}$ (derived from measurements of femoral venous blood flow and arterial and femoral venous blood O_2 concentrations) whereas hypoxia decreased leg $\dot{V}O_{2\,max}$ without changing leg blood flow. That leg $\dot{V}O_{2\,max}$ is limited by O_2 supply is clear, since when provided with more O_2, skeletal muscle utilised more O_2.

Alternatively, limitations to $\dot{V}O_{2\,max}$ might reside within the muscle and involve mechanisms that are not related to the supply of O_2 to the exercising muscle. One potentially limiting factor is muscle mitochondria (Weibel, 1987; Taylor, 1987). This would suggest that the mitochondria have to respire maximally to elicit $\dot{V}O_{2\,max}$, if $\dot{V}O_{2\,max}$ is not limited by O_2 delivery. Experimental evidence to support this is provided in single limb training studies where by greater increases in $\dot{V}O_{2\,max}$ have been observed in the trained (23%) compared to the untrained limb (7%) (Saltin *et al.*, 1976). This difference (16%) was attributed to peripheral adaptations within the muscle, mainly oxidative enzyme activity [succinate dehydrogenase (SDH)], which is a pre-requisite for elevating $\dot{V}O_{2\,max}$.

The mitochondria provide the final step for O_2 and therefore an increase in mitochondrial content or mitochondrial enzyme activity should result in a concomitant increase in the number of sites available for O_2 utilisation in the muscle. This should also result in a concomitant increase in $\dot{V}O_{2\,max}$. In support, Saltin *et al.* (1977) found that a two-fold increase in mitochondrial enzymes was associated with a change in $\dot{V}O_{2\,max}$ ranging from 20-40%. This suggests that metabolic changes in skeletal muscle permits a greater extraction of O_2 from the blood by exercising muscle, thus contributing to an increased $\dot{V}O_{2\,max}$.

Peripheral limitations to $\dot{V}O_{2\,max}$ are not unequivocally supported. Some evidence is based on the disassociation between $\dot{V}O_{2\,max}$ and muscle related enzyme activity after training. Henriksson and Reitman (1977) showed that at the end of their 8 week endurance training study, $\dot{V}O_{2\,max}$ had increased by 19% and SDH and cytochrome *c* oxidase (CO) activity in the quadriceps had increased by 32 and 35% respectively. However, after 6 weeks of detraining, $\dot{V}O_{2\,max}$ remained unchanged but SDH and CO had returned to pre-training levels. This demonstrates that $\dot{V}O_{2\,max}$ can remain elevated without an accompanying elevation of oxidative enzymes. Furthermore, individuals with similar $\dot{V}O_{2\,max}$ values have been found to have a two-fold range in mitochondrial enzyme concentration (Holloszy, 1973; Holloszy and Coyle, 1984). This also suggests that mitochondrial enzyme activity does not limit $\dot{V}O_{2\,max}$.

Based on a variety of experimental approaches that have involved isolated muscle and whole-body exercise in humans, our understanding of the limitations to $\dot{V}O_{2\,max}$ has gradually evolved to appreciate the complex interplay of O_2 delivery, O_2 diffusion and muscle metabolic factors. It can be concluded that $\dot{V}O_{2\,max}$ is predominantly limited by O_2 delivery processes to exercising muscle, for it is obvious a muscle can use no more O_2 than it receives. It has been suggested that factors determining O_2 supply and O_2 diffusion from the blood to skeletal muscle play a key role in determining $\dot{V}O_{2\,max}$ (Richardson *et al.*, 1999). This implies that $\dot{V}O_{2\,max}$ is not limited by just one

component of the O_2 transport pathway, rather $\dot{V}O_{2\,max}$ is set by the quantitative interaction of mechanisms that are involved in the delivery of O_2 as a system (Honig, 1992; Wagner, 1996). However, peripheral mechanisms such as those influencing mitochondrial oxidative capacity and thus the utilisation of O_2, also appear to have an important role and might in some instances interact to determine $\dot{V}O_{2\,max}$. Consequently, until experimental models involving simultaneous manipulation of O_2 delivery, O_2 diffusion and O_2 utilisation are applied in exercising humans, the precise identification of the limiting mechanism(s) will continue to be debated.

2.4.2 Measurement of $\dot{V}O_{2\,max}$ in MD and LD runners

The measurement of $\dot{V}O_{2\,max}$ in runners involves incremental (step-wise or ramp) treadmill tests to volitional exhaustion, during which pulmonary gas-exchange is measured. The intensity of exercise is progressively increased either by increasing the speed (Noakes *et al.*, 1990; Scott and Houmard, 1994) or gradient of the treadmill (Costill *et al.*, 1973; Farrell *et al.*, 1979). However, $\dot{V}O_{2\,max}$ has been found to be ~4% lower at the end of a speed protocol compared to a gradient protocol (Draper *et al.*, 1998). This might be attributable to the increasing contribution of the upper extremity during uphill running and to the additional recruitment of less efficient Type II (fast twitch) fibres. Furthermore, the protocol (speed or gradient) used to establish $\dot{V}O_{2\,max}$ might have implications for predicting performances that are achieved on flat courses (Billat *et al.*, 2001). This is because higher $\dot{V}O_{2\,max}$ values attained during gradient protocols might not be achievable during flat running and this will influence the fraction of $\dot{V}O_{2\,max}$ ($\%\dot{V}O_{2\,max}$) that can be sustained. The advantage of using a speed protocol, however, is that peak treadmill speed (PTS) achieved during the test can be used as an additional measure with which to assess training status. The PTS has been shown to be highly related to running performance in LD events (Noakes *et al.* 1990; Scott and Houmard, 1994).

2.4.3 Relationship of $\dot{V}O_{2\,max}$ with running performance

The $\dot{V}O_{2\,max}$ is the most widely reported physiological measure of human athletic performance and has long been used as a determinant of performance in MD and LD events (Saltin and Åstrand, 1967). Relationships between $\dot{V}O_{2\,max}$ and running performance have been investigated over a range of MD and LD events including 800 m (Brandon and Boileau, 1987; Weyand *et al.*, 1994), 1500 m (Tanaka *et al.*, 1983; Abe *et al.*, 1998) 3000 m (Zacharogiannis and Farrally 1993; Grant et *al.*, 1997), 5000 m (Kumagai *et al.,* 1982; Scott and Houmard, 1994), 10 000 m (Powers *et al.*, 1983; Brandon and Boileau, 1987) and the marathon (Takeshima and Tanaka, 1995; Billat *et al.*, 2001). In addition, $\dot{V}O_{2\,max}$ has been shown to differentiate elite and good LD runners (Pollock *et al.*, 1980; Billat *et al.*, 2001).

The relationship between $\dot{V}O_{2\,max}$ and running performance has been shown to increase concomitantly with the distance of the event (Brandon and Boileau, 1987; Weyand *et al.*, 1994). For example, $\dot{V}O_{2\,max}$ correlated more strongly with 5 km (r = -0.93) than 1500 m (r = -0.79) and 800 m (r = -0.55) performance in runners who were heterogeneous with respect to $\dot{V}O_{2\,max}$ and performance (Weyand *et al.*, 1994). This finding is likely to reflect the increased aerobic contribution to energy production as the distance of the race increases. In LD events, the relationship between $\dot{V}O_{2\,max}$ and performance has been consistently high (Farrell *et al.*, 1979; Fay *et al.*, 1989) reflecting the high aerobic contribution to running distances beyond 3000 m (Wood, 1999).

However, many studies have demonstrated that $\dot{V}O_{2\,max}$ is not strongly associated with running performance (Costill *et al.*, 1976a; Conley and Krahenbuhl, 1980; Powers *et al.*, 1983; Housh *et al.*, 1988). The primary reason why $\dot{V}O_{2\,max}$ and performance are correlated only in some studies appears to be related to the range of $\dot{V}O_{2\,max}$ and ability of the athletes under investigation. For example, in groups of athletes who are heterogeneous with respect to running ability, $\dot{V}O_{2\,max}$ appears to be a good predictor of running performance (Foster, 1983; Grant *et al.*, 1997). Whereas in groups of athletes

who are homogeneous with respect to running ability, $\dot{V}O_{2\,max}$ is a poor predictor of performance (Conley and Krahenbuhl, 1980; Powers *et al.*, 1983; Morgan *et al.*, 1989).

Inconsistencies in the relationship between $\dot{V}O_{2\,max}$ and running performance might be attributable to the protocol used to determine $\dot{V}O_{2\,max}$. For instance, Billat *et al.* (2001) reported that in elite marathon runners (n=9), $\dot{V}O_{2\,max}$ measured on a flat surface was lower than that measured on a treadmill using a gradient protocol (71.7 ± 11.0 vs. 78.7 ± 7.0 $ml \cdot kg^{-1} \cdot min^{-1}$, P = 0.03). This 8.9% difference might have a direct effect on the relationship between $\dot{V}O_{2\,max}$ and running performances achieved on flat surface and $\dot{V}O_{2\,max}$ measured using a gradient protocol in the laboratory.

Several studies have demonstrated that an increase (Daniels *et al.*, 1978; Jones, 1998) and decrease (Houston *et al.*, 1979) in running performance can occur without reciprocal changes in $\dot{V}O_{2\,max}$. Thus, $\dot{V}O_{2\,max}$ is not a sensitive indicator of training adaptation and performance. It is possible that physiological adaptations within the muscle, such as mitochondrial function and oxidative enzyme activity, resulted in a reduced blood lactate concentration [HLa] for a given running speed and contributed towards improved performance without influencing $\dot{V}O_{2\,max}$. In support, reductions in the oxidative capacity (SDH activity) of the muscle of a magnitude similar to the reduction in performance has been observed (Houston *et al.*, 1979). Therefore, $\dot{V}O_{2\,max}$ might only be of limited interest for predicting running performance and monitoring physiological adaptations resulting from training or de-training in the competitive runner. Alternatively, non-physiological changes such as different pacing strategies and motivation amongst individuals might have contributed to changes in running performance and distorted the true physiological relationship.

In summary, although a high $\dot{V}O_{2\,max}$ is a pre-requisite for entry into the elite category of MD and LD runners, research to date appears equivocal in its findings with respect to the contribution and importance of $\dot{V}O_{2\,max}$ to successful endurance running

performance. The predictive qualities of $\dot{V}O_{2\,max}$ appear to be influenced by the distance of the event and the range of abilities of athletes investigated (homogeneous or heterogeneous). Furthermore, changes in performance can be achieved without changes in $\dot{V}O_{2\,max}$ signifying that this measure is not the limiting determinant of successful running performance. It is likely that other physiological adaptations that are related to the oxidative capacity of the muscle influence running performance.

2.5 The anaerobic threshold

The "anaerobic threshold" (AT) was first proposed by Wasserman and McIlroy (1964) who elaborated on the concept that measures of pulmonary gas-exchange at the mouth could be used to detect the onset of metabolic (lactic) acidosis in muscle. However, the term "anaerobic threshold" has resulted in much controversy as to its existence, definition and validity (Yeh *et al.*, 1983; Brooks, 1985; Hughson *et al.*, 1987). Subsequently, several alternative terms and approaches have been developed to provide a less mechanistic descriptor. The terms "lactate threshold" (LT, Coyle *et al.*, 1983), "ventilatory threshold" (V_T, Powers *et al.*, 1983), "onset of plasma lactate accumulation" (OPLA, Farrell *et al.*, 1979) and "aerobic threshold" (Skinner and McLellan, 1980; Aunola and Rusko, 1986) are used interchangeably in the literature. However, according to Wasserman *et al.* (1994), making the distinction only distinguishes the method of measurement and does not dispute the underlying mechanism.

2.5.1 Mechanisms of the anaerobic/ventilatory/lactate threshold

During moderate-intensity exercise, most of the hydrogen ions stripped from the substrate and carried by NADH are oxidised within the mitochondria and passed to oxygen via the ETC to form water. In these conditions, a biochemical steady state is achieved with minimal lactate accumulation since the rate of lactate appearance (R_a) is equal to the rate of disappearance (R_d). However, as the intensity of exercise increases beyond the moderate domain, the supplemented energy production from anaerobic

glycolysis and recruitment of less efficient Type II fibres causes an increase in the lactate-pyruvate ratio. Indeed, the close correlation between the percentage of Type I fibres in the muscle and the LT (Ivy *et al.*, 1980) suggests that the LT might coincide with an increased recruitment of Type II fibres as the intensity of exercise increase. Consequently, pyruvate reacts with $NADH + H^+$ and is reduced to lactate, via the enzyme lactate dehydrogenase (LDH), while regenerating NAD^+ and allowing anaerobic glycolysis to continue. This process is summarised as: -

$$\text{Pyruvate} + \text{NADH} + H^+ \xleftrightarrow{\text{LDH}} \text{Lactate} + NAD^+ \qquad (7)$$

The increased lactate is then immediately buffered intra-cellularly, predominantly by HCO_3^-, which generates additional CO_2. The HCO_3^- exchanges for lactate across the muscle cell membrane causing arterial blood HCO_3^- to decrease.

The traditional explanation proposed by Wasserman *et al.* (1994) was formulated around the insufficient availability of O_2, suggesting that lactate accumulation occurs because the "critical" capillary partial pressure of O_2 (PO_2) (defined as the lowest capillary PO_2 that allows mitochondria to receive and consume O_2 during exercise) is reached before the end of the capillary. The mitochondrial membrane proton shuttle then loses pace with the rate of $NADH + H^+$ production in the cytosol, resulting in a reduction in the cytosolic redox potential ($NADH/NAD^+$). However, several studies refute this and demonstrate that: 1) lactate is produced and removed under fully aerobic conditions in humans (Brooks, 1986 and 1991; Bergman *et al.*, 1999) and; 2) lactate is released at power outputs (PO) equivalent to 50% $\dot{V}O_{2\,max}$ despite no signs of O_2 lack (as evidenced by PO_2 remaining above the critical mitochondrial PO_2) (Richardson *et al.*, 1998). Clearly, this dissociates lactate production from anaerobiosis and lends support to the 'lactate-shuttle' hypothesis proposed by Brooks (1986). The existence of a lactate-shuttle permits the transportation of lactate between muscle fibres and allows oxidation and gluconeogenesis during rest and exercise so that although lactate is being

produced it is cleared at a commensurate rate. This opposes suggestions that lactate is a metabolic dead-end product that can only be removed during recovery and therefore, it is the ability to clear lactate upon production that determines lactate accumulation and thus the LT/V_T.

2.5.2 Measurement of anaerobic/ventilatory/lactate threshold

There are two approaches that can be used to identify the AT. The original, proposed by Wassermann and McIlroy (1964), involves measuring pulmonary gas-exchange responses during progressive increases in the intensity of exercise. Alternatively, [HLa] during an incremental exercise task can be measured.

2.5.2.1 Ventilatory threshold

The V_T is identified using non-invasive, pulmonary gas-exchange measures obtained during a progressive maximal exercise test (e.g. 15 $W \cdot min^{-1}$, Whipp *et al.*, 1981; Beaver *et al.*, 1986) that results in volitional exhaustion (Wasserman and McIlroy, 1964; Wasserman *et al.*, 1973; Whipp *et al.*, 1981). During the transition from low- to moderate-intensity exercise, $\dot{V}O_2$, the rate of carbon dioxide production ($\dot{V}CO_2$) and minute ventilation ($\dot{V}E$) increase linearly with increases in the intensity of exercise up to the V_T. Above the V_T, $\dot{V}CO_2$ increases more rapidly than $\dot{V}O_2$ because CO_2, generated by the bicarbonate buffering of lactic acid, is added to the metabolic CO_2 production (Wasserman *et al.*, 1994). Since $\dot{V}CO_2$ retains a constant relationship with $\dot{V}E$ during the period of isocapnic buffering, $\dot{V}E/\dot{V}CO_2$ remains constant (or may drop slightly) whilst at the same time $\dot{V}E/\dot{V}O_2$ systematically rises. Distinguishing the point at which $\dot{V}E/\dot{V}O_2$ rises but $\dot{V}E/\dot{V}CO_2$ remains constant also identifies the V_T (Caiozzo *et al.*, 1982). Alternative criteria have been proposed by Beaver *et al.* (1986) that are independent of respiratory chemoreceptor sensitivity and $\dot{V}E$. This method involves identifying the disproportionate increase in $\dot{V}CO_2$ with $\dot{V}O_2$ and is known as the *V*-slope method. This reflects additional CO_2 production as a result of the bicarbonate buffering of lactic acid.

2.5.2.2 Lactate threshold

The LT is identified during an incremental protocol that requires [HLa] measurements to be taken at the end of each incremental stage, which are typically 4 min long. The graphical representation of [HLa] to the gradually increasing intensity results in a two component response, one having a shallow, or zero slope, followed by a steeper component (Beaver *et al.*, 1985). The zero slope, below the LT, results from a successful balance between the production and removal of lactate, primarily through oxidation (Brooks, 1985). At this point [HLa] is usually below 2 $mmol \cdot l^{-1}$ or might increase above the resting level by less than 1 $mmol \cdot l^{-1}$. The intersection of the two lines is the LT. The LT can be identified and expressed as a $\dot{V}O_2$ and subsequently as a fraction of the individuals $\dot{V}O_{2\,max}$ (Farrell *et al.*, 1979). In highly-trained runners the LT occurs at intensities greater than 70% of $\dot{V}O_{2\,max}$ (Costill *et al.*, 1973; Tanaka and Matsuura, 1984). Some investigators have argued that increases in [HLa] follow a continuous exponential model (Hughson *et al.*, 1987). In runners, Farrell *et al.* (1979) observed both exponential and threshold responses suggesting that the lactate response does not conform to one specific model.

2.5.3 Relationship of V_T and LT with running performance

While the concept of the AT has been debated in the past (Brooks, 1985; Davis, 1985), support for this concept has come from studies showing strong correlations between running performance and the LT (Farrell *et al.*, 1979; Sjödin and Jacobs, 1981; Grant *et al.*, 1997; Roecker *et al.*, 1998) and V_T (Powers *et al.*, 1983; Rhodes and McKenzie, 1984; Peronnet *et al.*, 1987; Zacharogiannis and Farrally, 1993) over a range of distances, suggesting that direct measures of [HLa] and pulmonary gas-exchange are equally good methods to determine potential relationships with performance. In some studies, the $\dot{V}O_2$ at the LT and V_T have been shown to be more highly correlated with running performance than $\dot{V}O_{2\,max}$ (Farrell *et al.*, 1979; Kumagai *et al.*, 1982; Yoshida *et al.*, 1987).

The above findings are not representative of all studies that have assessed the relationship between LT/V_T and running performance. For instance, it has been reported that the LT/V_T is not related to 5000 and 10000 m (Iwaoka *et al.*, 1988) and marathon (Tanaka and Matsuura, 1984; Florence and Weir, 1997) running performance. A common characteristic of these studies is that athletes were homogeneous with respect to their performance. Similarly, in samples of trained runners who were heterogeneous with respect to $\dot{V}O_{2\,max}$ and performance, the correlation between running performance and the V_T was low (Brandon and Boileau, 1992). These findings could be partially explained by the shorter distances i.e. 800 and 1500 m used to characterise running performance, suggesting that the V_T has an increasing contribution to performance as the distance of the event increases. To account for such findings, it is probable that anaerobic pathways are contributing to performance during MD events and that this is not reflected in the measure of the LT or V_T. This would mean that two athletes with similar LT/V_T could be differentiated with respect to running performance on the basis of their anaerobic qualities.

In summary, the use of [HLa] and pulmonary gas-exchange criteria to determine the LT and V_T respectively, have both been shown to be closely related to running performance. In some instances the observed relationships have exceeded those found between $\dot{V}O_{2\,max}$ and performance suggesting that LT and V_T are closer determinants of performance. However, to a lesser extent than that found for $\dot{V}O_{2\,max}$, the LT and V_T can also be influenced by the characteristics of the sample (e.g. homogeneity) as well as the distance of the event. This suggests that although the V_T and LT are more closely related to performance than $\dot{V}O_{2\,max}$, they are not the exclusive determinants of running performance.

2.6 Running economy: oxygen cost of exercise

The term running economy (RE) is predominantly used to describe the O_2 cost of sub-maximal running. The RE is defined as the steady-state O_2 consumption for a given

running speed, expressed relative to body mass (BM) as a ratio standard, i.e. $ml \cdot kg^{-1} \cdot min^{-1}$ (Conley and Krahenbuhl, 1980; Cavanagh and Williams, 1982). Using this expression, the RE of a range of MD and LD runners has been established (Conley and Krahenbuhl, 1980; Svedenhag and Jacobs, 1984; Jones, 2002). Alternatively, the energy or O_2 cost of running (C_r) has been determined by dividing the $\dot{V}O_2$, minus resting $\dot{V}O_2$, corresponding to a sub-maximal speed by that speed (Margaria *et al.*, 1963; di Prampero *et al.*, 1986). The C_r is therefore expressed in ml $O_2 \cdot kg^{-1} \cdot km^{-1}$ and has been shown to be independent of running speed (Cavagna *et al.*, 1964; di Prampero *et al.*, 1986).

2.6.1 Measurement of RE

With respect to RE, the speeds over which MD and LD runners are assessed plays an important part in determining which runners are the most economical. It has been recommended that RE data are collected up to speeds that elicit an intensity equivalent to 90% $\dot{V}O_{2\,max}$ (Daniels and Daniels, 1992). This recommendation was derived from a study of elite runners where MD runners (800 and 1500 m) were found to be more economical than LD runners (marathon) at speeds faster than marathon race pace, but not at slower speeds (Daniels and Daniels, 1992). This suggests that RE is event specific. However, caution is required when measuring $\dot{V}O_2$ at running speeds when there are significant increases in [HLa]. This is because an additional slow component of $\dot{V}O_2$ is likely to develop during heavy-intensity exercise which is above the LT/V_T (Barstow, 1994; Whipp, 1994). This makes establishing the true O_2 cost of exercise difficult as a true steady-state is not achieved. This could result in a misleading representation of an individuals RE or C_r. For this reason, it would be preferable to determine measures of RE and C_r during bouts of moderate-intensity running that do not exceed the individuals LT/V_T.

2.6.2 Mechanisms determining or influencing RE

Mechanism(s) determining RE are attributed either to physiological or mechanical characteristics of the individual. Williams and Cavanagh (1987) have shown that physiological variables (e.g. percentage of Type I muscle fibres and $\dot{V}O_{2\,max}$) and biomechanical variables (e.g. ground reaction forces and leg kinematics) interact to influence RE. Therefore, it is possible that changing a specific aspect of a runner's technique could lead to an improvement in RE. However, changing one aspect could influence other variables and the effect on RE could be unpredictable.

To quantify the sensitivity of RE, several physiological aspects of RE have been manipulated by various experimental approaches. Ultimately, these have been conducted to assist in developing a better understanding of the mechanism(s) underlying or influencing RE. However, there is no consensus regarding the influence of variables such as body temperature (Gaesser and Brook, 1984), heart rate (HR) and $\dot{V}E$ (Pate *et al.*, 1992), muscle fibre type (Williams and Cavanagh, 1987), gender (Bransford and Howley, 1977; Daniels and Daniels, 1992) and environmental conditions (Leger and Mercier, 1984) on the RE of trained individuals. It appears that no single variable, or small subset of variables, can explain differences in RE between individuals. Thus, the likelihood that RE is related to a weighted sum of the influences of many physiological and biomechanical variables seems the most probable explanation for differences in RE amongst MD and LD runners.

2.6.3 The relationship between RE and performance

Hill and Lupton, as early as 1923, recognised RE as a factor that would affect running performance: "A man may fail to be a good runner by reason of a low oxygen uptake, a low maximum oxygen debt, or a *high oxygen requirement*; clumsy and uneconomical movements may lead to exhaustion just as well as may an imperfect supply of oxygen". Subsequently, the assessment of RE and C_r has become a widely accepted means of evaluating endurance running performance and has been considered as the physiological

criterion for efficient performance (Cavanagh and Kram, 1985). Several studies have identified a strong relationship between RE and running performance (Conley and Krahenbuhl, 1980; LaFontaine *et al.*, 1981). Conley and Krahenbuhl (1980) reported that RE measured at 3 sub-maximal running speeds (14.5, 16.1 and 17.7 $km \cdot h^{-1}$) correlated strongly with 10 km performance (r = 0.83, 0.82 and 0.79 respectively, P <0.01), whereas $\dot{V}O_{2\,max}$ did not (r = 0.12, P >0.05). Measures of RE explained 64.5% of the variability in 10 km performance. Thus the ability a runner has to minimise energy expenditure at a given speed is a performance-determining variable.

In MD and LD runners, RE can also be considered a poor predictor of running performance, since poor correlations between RE and performance for a range of distances including 3 km (Grant *et al.*, 1997), 10 km (Williams and Cavanagh, 1987), 10 mile (Costill *et al.*, 1973) and marathon (Noakes *et al.*, 1990; Billat *et al.*, 2001) races, have been reported. In the majority of these studies, runners displayed heterogeneous characteristics with respect both to $\dot{V}O_{2\,max}$ and performance, with $\dot{V}O_{2\,max}$ accounting for variations in performance. However, in the study of Powers *et al.* (1983) a poor correlation was observed despite the homogeneous characteristics of both $\dot{V}O_{2\,max}$ and 10 km performance in the participants studied. It is not completely clear why these data differ from the work of Conley and Krahenbuhl (1980); however, it can be speculated that the poor correlation between performance and RE resulted from homogeneity of RE in the athletes studied. This suggests that in some populations the individual differences in RE are not great, and that RE may be of limited value in differentiating distance running performance in homogeneous and heterogeneous groups.

The importance of RE is apparent when small numbers of athletes with similar $\dot{V}O_{2\,max}$ values are compared. For example, Costill and Winrow (1970) identified two athletes with similar $\dot{V}O_{2\,max}$ but with different running abilities and found that the superior RE accounted for the faster runner's better performance. This is further supported by

Morgan and Daniels (1994) who found a positive relationship between $\dot{V}O_{2\,max}$ and sub-maximal $\dot{V}O_2$ revealing that runners exhibiting higher aerobic demands of running (i.e. poorer RE) tended to have higher $\dot{V}O_{2\,max}$ values. These results suggest that trained runners who display similar metabolic and performance values show counter-balancing profiles of $\dot{V}O_{2\,max}$ and RE. Thus it appears that $\dot{V}O_{2\,max}$ and RE interact to determine the level of performance e.g. it is possible for an athlete with superior RE to compensate for an inferior $\dot{V}O_{2\,max}$ yet obtain a similar level of performance (Daniels, 1974). To account for this interaction, dividing the RE of a runner by their $\dot{V}O_{2\,max}$ results in the fractional utilisation of $\dot{V}O_{2\,max}$ ($\%\dot{V}O_{2\,max}$) for that running speed (Costill *et al.*, 1973). The $\%\dot{V}O_{2\,max}$ whilst running at a common sub-maximal speed (e.g. 16 $km \cdot h^{-1}$) might be a better way to express RE as it takes into consideration an individuals $\dot{V}O_{2\,max}$ and has been found to correlate highly with running performance in several studies that had previously found no relationship between RE when expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ (Costill *et al.*, 1973; Conley *et al.*, 1981; Noakes *et al.*, 1990; Grant *et al.*, 1997).

Runners with various running abilities have been shown to have similar C_r [range: 174 to 188 $ml \cdot kg^{-1} \cdot km^{-1}$ (di Prampero *et al.*, 1986; Brisswalter and Legros, 1994)] suggesting that C_r is independent of performance. In support, Billat *et al.* (2001) reported a poor correlation between C_r and marathon performance for elite marathon runners.

In summary, the relationship between running performance and O_2 cost of exercise during sub-maximal running, regardless of how it is expressed (RE or C_r), is not consistent and appears to be influenced by the homogeneity of the sample with respect to running ability and/or $\dot{V}O_{2\,max}$. The interaction between RE and $\dot{V}O_{2\,max}$ suggests that both of these measures should be considered when assessing or predicting running performance in MD and LD runners. Accordingly, it can be concluded that RE is not the exclusive determinant of MD and LD running performance, especially when considered separate to measures of $\dot{V}O_{2\,max}$.

2.6.4 Body size and $\dot{V}O_2$

Conventionally, $\dot{V}O_2$ during sub-maximal and maximal exercise is expressed in ratio with BM (i.e. $ml \cdot kg^{-1} \cdot min^{-1}$). This attempts to remove the influence of BM on the physiological variable in question. However, according to principles of allometry, $\dot{V}O_2$ does not increase linearly with BM (Schmidt-Nielson, 1984). It has been identified that $\dot{V}O_2$ does not increase proportionally to BM during sub-maximal (Bergh *et al.*, 1991) and maximal (Bergh *et al.*, 1991; Welsman *et al.*, 1996; Heil, 1997) running. This suggests that indiscriminate use of the ratio standard to account for differences in BM in runners could be misleading. Potentially, this could have implications for the evaluation of athletes and might be the reason why in many studies no or weak correlations between $\dot{V}O_2$ measures (sub-maximal and maximal) and running performance have been found (see previous sections). This raises the question of how differences in BM should be accounted for during running.

The most appropriate BM exponent to describe measures such as $\dot{V}O_{2\,max}$, RE (or C_r) has been the subject of debate. According to the theory of geometric similarities and surface laws, $\dot{V}O_{2\,max}$ should be proportional to $BM^{0.67}$ (Åstrand and Rodahl, 1986). Alternatively, a BM exponent of 0.75 has been proposed which is based on elastic similarity (Kleiber, 1947; McMahon, 1973). More recently, however, allometric cascade models have produced BM exponents of 0.86 and 0.75 for maximal exercise and rest respectively (Darveau *et al.*, 2002). This suggests that different exponents might be required for maximal and sub-maximal measures of $\dot{V}O_2$ during running.

Different BM exponents have been applied when expressing $\dot{V}O_2$ in runners (Helgurud, 1994; Svedenhag and Sjödin, 1994; Morgan *et al.*, 1995). In some instances this approach has had a significant effect on the results. For example, Svedenhag and Sjödin (1994) were able to differentiate between elite MD and LD runners when RE (at 18 $km \cdot h^{-1}$) was expressed in $ml \cdot kg^{-0.75} \cdot min^{-1}$ but not when expressed in $ml \cdot kg^{-1} \cdot min^{-1}$.

This emphasises the need to consider the most appropriate exponent which can effectively partition the effects of differences in BM.

To maximise potential relationships between physiological measures and running performance, a further step would be to identify the individual exponent for a given sample. Using this approach, Nevill *et al.* (1992) identified that the most appropriate exponent when expressing $\dot{V}O_{2\,max}$ and relating it to 5 km running performance was 1.0; identical to the ratio standard which is typically used to express measures of $\dot{V}O_2$. Furthermore, by using this exponent, Nevill *et al.* (1992) were more able to divide participants according to their performance than the 0.67 power function ratio when performance was expressed as mean speed ($m \cdot s^{-1}$). This supports the conventional expression of $\dot{V}O_2$ in $ml \cdot kg^{-1} \cdot min^{-1}$ for weight-bearing activities, which are highly dependent on BM. However, for a different sample the exponent might differ from 1.0 due to a different range of BM and $\dot{V}O_2$ measures and therefore, would require the sample-specific exponent to be identified.

2.7 Pulmonary $\dot{V}O_2$ kinetics

Pulmonary $\dot{V}O_2$ kinetics is a measure of the rate at which $\dot{V}O_2$ increases at the onset of exercise, or to an abrupt change in the intensity of exercise. In healthy individuals, measures of $\dot{V}O_2$ at the mouth provide an excellent representation of the rate of muscle O_2 consumption ($\dot{Q}O_2$) during exercise (Grassi *et al.*, 1996). Therefore, measures of $\dot{V}O_2$ kinetics provide a valuable insight into oxidative muscle metabolism during exercise.

Interpretation of pulmonary gas-exchange dynamics can be confusing and possibly misleading if they are not considered in the context of the intensity of exercise (Whipp and Ward, 1990). The dominant, first-order kinetic characteristics are changed to complex, multi-compartment behaviour as the intensity of exercise exceeds the V_T. The following description concerns primarily the dynamic $\dot{V}O_2$ response profile at the onset

of moderate-intensity exercise (i.e. below the V_T) although the dynamics of $\dot{V}O_2$ in response to heavy- and severe-intensity exercise will also be briefly discussed.

2.7.1 The $\dot{V}O_2$ response at the onset of moderate-intensity exercise

Since the pioneering work of Hill and Lupton (1923), it has been recognised that pulmonary $\dot{V}O_2$ rises exponentially at the onset of constant-load exercise. In the moderate-intensity domain, three separate and distinct time-related phases have been identified and quantified (Whipp *et al.*, 1982). These phases result from neural, mechanical and metabolic events in the body. The three phases are illustrated from experimental data in Figure 2.3.

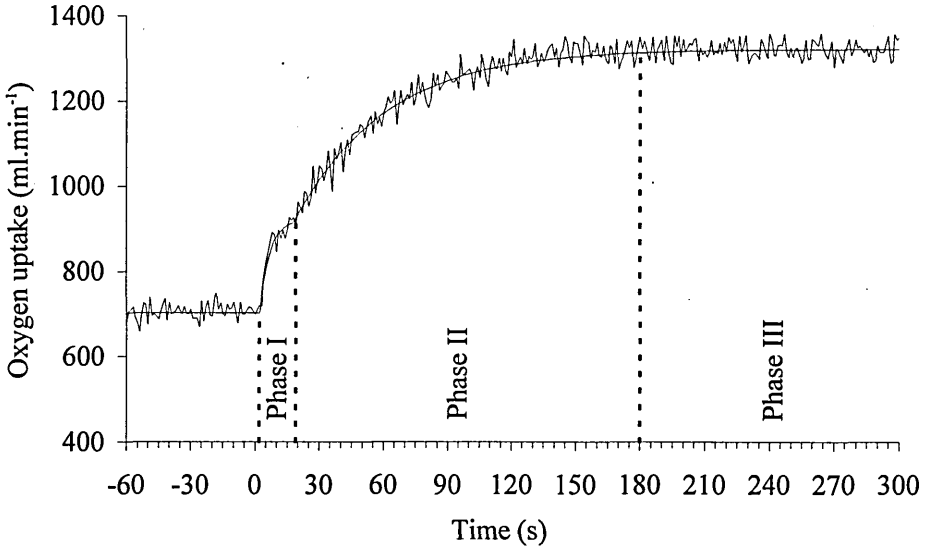


Figure 2.3 The $\dot{V}O_2$ response at the onset (time = 0) of moderate-intensity exercise. Phases (I - III) are highlighted and described in the text (adapted from Sietsema *et al.*, 1989).

The measurement of pulmonary $\dot{V}O_2$ by Krogh and Lindhard (1913) first identified the initial rapid increase in $\dot{V}O_2$ at the onset of exercise. This is caused predominantly by an increase in pulmonary blood flow and is attributed to an increased $\dot{Q}$ (Linnarsson, 1974; Whipp *et al.*, 1982; Hamar, 1991). This has been termed the "cardiodynamic" phase I (Whipp *et al.*, 1982) and is approximately 15 to 20 s in duration. Since it has

been shown that there is minimal extraction of O_2 at the muscle during this initial phase (Grassi *et al.*, 1996), it can be confirmed that phase I $\dot{V}O_2$ kinetics do not reflect metabolic events in the muscle.

Immediately following phase I, $\dot{V}O_2$ starts to increase mono-exponentially (Hill and Lupton, 1923; Henry and De Moor, 1956; Whipp, 1971). Traditionally, this increase has been termed phase II (Whipp *et al.*, 1982). During this phase, venous blood from the exercising muscle arrives at the lungs. This blood has a lower O_2 content than the blood arriving at the lungs during phase I and reflects the influence of muscle metabolic change on pulmonary $\dot{V}O_2$ measured at the mouth. The modelling of pulmonary $\dot{V}O_2$ and muscle $\dot{V}O_2$ kinetics during exercise transients (Barstow and Mole, 1987; Grassi *et al.*, 1996), as well as the observed equivalence between intra-muscular [PCr] and $\dot{V}O_2$ kinetics during phase II (Rossiter *et al.*, 1999), suggest that this second phase closely represents the behaviour of $\dot{Q}O_2$. Finally, phase III refers to the point at which a steady-state $\dot{V}O_2$ has been achieved. This demarcates the point at which $\dot{V}O_2$ is precisely coupled with cellular metabolism within the exercising muscle (Wasserman *et al.*, 1994), i.e. $\dot{V}O_2$ supply is equal to $\dot{V}O_2$ demand. The time taken for $\dot{V}O_2$ to reach phase III is usually 2-3 min, although this is dependent on both the health and physiological status of the individual.

During exercise above the V_T the $\dot{V}O_2$ response becomes more complex; displaying time and amplitude non-linearities (Barstow and Mole, 1991). In most circumstances, the phase III steady state $\dot{V}O_2$ is not achieved (Figure 2.4) and a further, but delayed, supplemental rise in $\dot{V}O_2$ termed the 'slow-component' (Whipp and Wasserman, 1972) occurs.

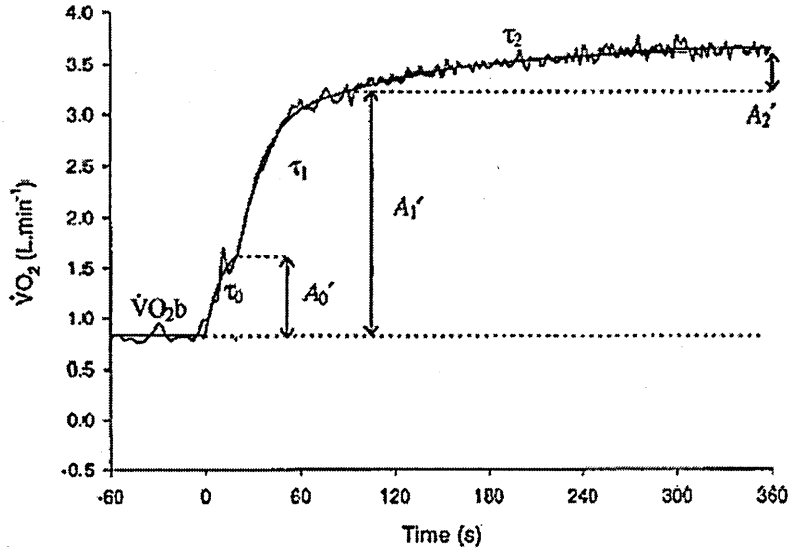


Figure 2.4 The $\dot{V}O_2$ response at the onset of heavy-intensity exercise illustrating the 'slow-component' (A_2') (adapted from Burnley *et al.*, 2000).

Uncertainty surrounds the precise physiological mechanism(s) responsible for the development of the $\dot{V}O_2$ slow-component, which is often associated with an increase in [HLa] (Casaburi *et al.*, 1987). However, the tight association between the temporal profile of [HLa] and the $\dot{V}O_2$ slow-component might be the consequence of several putative mediators including the progressive recruitment of less efficient Type II fibres (Barstow *et al.*, 1996), increased body temperature (Q_{10} effect), decreased muscle pH and/or increased respiratory and cardiac work (Aaron *et al.*, 1992; Stringer *et al.*, 1997).

2.7.2 Characterisation of $\dot{V}O_2$ kinetics as a function of the intensity of exercise

Within a given intensity domain, different temporal components of the $\dot{V}O_2$ response to constant-load exercise can be identified (Whipp *et al.*, 1982). One specific issue within studies of $\dot{V}O_2$ kinetics is whether the phase II τ is influenced by the intensity of exercise. Several studies to investigate this have clearly shown that τ is invariant for exercise intensities below and above V_T/LT during cycling in trained (Barstow and Mole, 1991; Barstow *et al.*, 1993) and un-trained individuals (Barstow *et al.*, 1993; Özyener *et al.*, 2001). For example, Barstow *et al.* (1993) demonstrated that for intensities between 30 and 100% $\dot{V}O_{2\,max}$, phase II τ was invariant (Barstow *et al.*,

1993). Similarly, Özyener *et al.* (2001) reported no differences between τ for $\dot{V}O_2$ at the onset of moderate (33 ± 16 s), heavy (32 ± 17 s), very heavy (34 ± 11 s) and severe (34 ± 7 s) intensity exercise. Collectively, these findings suggest that the primary $\dot{V}O_2$ response, even at higher power outputs, reflects a linear system. Consequently, transitions to moderate- and heavy-intensity exercise provide similar information about the oxidative potential of the muscle and supports the use of transitions to moderate-intensity exercise as a non-invasive estimate of $\dot{Q}O_2$ (Barstow and Mole, 1987).

There is, however, some evidence to suggest that τ might vary according to the intensity of exercise. Paterson and Whipp (1991) reported that the phase II τ was slowed (i.e. τ was longer) during heavy-intensity compared to moderate-intensity exercise (40.2 ± 2.7 s and 31.3 ± 3.3 s respectively). This conflicting finding could be attributable to inconsistent and/or inappropriate modelling techniques, as highlighted by Bell *et al.* (2001). For example, Paterson and Whipp (1991) modelled transitions to heavy-intensity exercise using a mono-exponential model which considered phase II $\dot{V}O_2$ and the $\dot{V}O_2$ slow component as one kinetic parameter. It is probable that inclusion of the slow-component of $\dot{V}O_2$ in the modelling of phase II artificially slowed the underlying τ during phase II for heavy-intensity exercise (Barstow, 1994). This would clearly influence the interpretation of the results. Therefore, a two-component model is more appropriate to partition the phase II and slow component $\dot{V}O_2$ responses during transitions to heavy-intensity exercise so that the influence of increasing exercise intensity on phase II τ can be explored more meaningfully.

In agreement with Barstow *et al.* (1993) and Özyener *et al.* (2001), most other studies using alternative modes of ergometry, including treadmill running, have reported no significant differences in phase II τ at the onset of moderate- and heavy-intensity exercise (Carter *et al.*, 2000a, b; Williams *et al.*, 2001). Recently, however, a study examining $\dot{V}O_2$ kinetics during treadmill running across moderate, heavy and severe intensity domains [(i.e. speeds equivalent to 80% and 100% of the $\dot{V}O_2$ at LT and 20%,

40%, 60%, 80% and 100% of the difference between (Δ) the $\dot{V}O_2$ at LT and $\dot{V}O_{2\,max}$] was conducted by Carter *et al.* (2002). This study revealed that the phase II τ was shorter for moderate- (80%LT) than heavy-intensity (40%Δ) exercise (12.7 ± 1.4 s and 19.1 ± 0.8 s respectively; P = 0.035), but that τ was invariant at intensities above 100%LT. The difference in τ between moderate- and heavy-intensity exercise was primarily attributed to changing patterns of muscle fibre recruitment that occur as the intensity of exercise increases. Specifically, the increased recruitment of glycolytic Type II fibres, with presumably slower O_2 utilisation kinetics as the intensity of exercise increased, was suggested as the reason why τ was longer during heavy-intensity exercise. However, it remains unclear as to why similar findings were not observed in the study of Barstow *et al.* (1993), especially since similar modelling procedures were used to establish τ. Potentially, the inconsistent findings could be caused by differences in the mode of exercise (cycling vs. running). However, recent work from our laboratory (Wells *et al.*, 2003) using treadmill ergometry has revealed no difference in τ for participants with relatively similar $\dot{V}O_{2\,max}$ (54.6 ± 3.2 $ml \cdot kg^{-1} \cdot min^{-1}$) exercising at moderate (80%V_T; τ = 23.2 ± 5.5 s) and heavy intensities (50%Δ; τ = 23.7 ± 4.0 s), thus refuting the findings of Carter *et al.* (2002) and discounting the effect of exercise modality on τ.

In conclusion, uncertainty remains regarding the influence of exercise intensity on the time course of $\dot{V}O_2$ adjustments during phase II at the onset of exercise. This is clearly an area for future research to clarify, especially in regards to different modes of exercise. Perhaps most importantly is whether the physiological mechanism(s) determining phase II τ during moderate- and heavy-intensity exercise are similar. For example, if future studies continue to confirm that phase II τ above and below V_T is invariant then it would be acceptable to use moderate-intensity exercise to characterise phase II $\dot{V}O_2$ kinetics. However, if the phase II τ for heavy versus moderate-intensity exercise is found to be slowed, and the mechanistic basis for this slowing is attributed to the progressive recruitment of Type II muscle fibres (as suggested by Carter *et al.*,

2002), then this information should be considered when determining protocols for studies involving $\dot{V}O_2$ kinetics. For example, if information about peripheral adaptation(s) relating to muscle oxidative function in a proportion of muscle fibres (predominantly Type I fibres) is required, then the use of moderate-intensity exercise to characterise $\dot{V}O_2$ kinetics would be warranted. A useful application of this measure in the moderate-domain would be in endurance-trained runners, where the composition of Type I fibres in muscle can be greater than 70% (Saltin and Gollnick, 1983) and are likely to reflect training adaptation and/or determine performance in predominantly aerobic events. Clearly, further information clarifying the uncertainties of τ above and below V_T would be beneficial to future studies involving $\dot{V}O_2$ kinetics.

2.7.3 The $\dot{V}O_2$ response during recovery from moderate-intensity exercise

At the cessation of moderate-intensity exercise, the dynamics of $\dot{V}O_2$ during the off-transient exhibit a characteristic three-phase response equivalent to that observed during the on-transient (Figure 2.5), provided there was no significant increase in [HLa]. In summary, phase I involves a sudden decrease in $\dot{V}O_2$ resulting from a decrease in blood flow as a consequence of a reduced $\dot{Q}$; Phase II involves an exponential decrease in $\dot{V}O_2$ reflecting [PCr] and O_2 store replenishment. This phase has been termed the fast component of recovery (Margaria *et al.*, 1933; Henry, 1951; Gaesser and Brooks, 1984). Phase III involves the return of $\dot{V}O_2$ to pre-exercise levels.

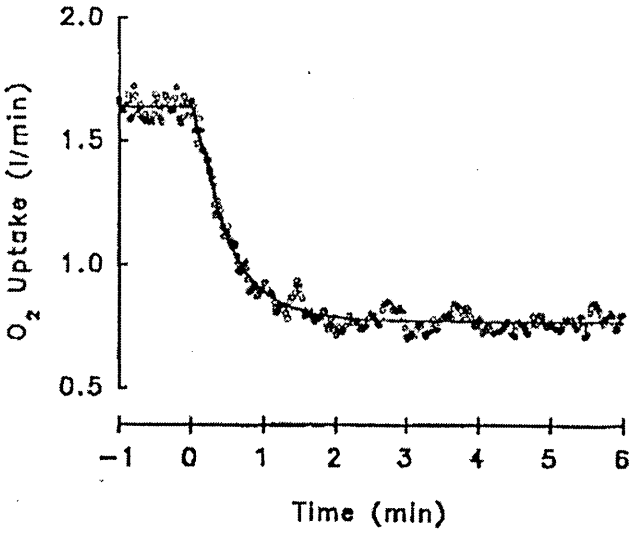


Figure 2.5 The $\dot{V}O_2$ response at the cessation (time = 0) of moderate-intensity exercise (from Scheuermann *et al.*, 1998).

2.7.4 Measurement of $\dot{V}O_2$ kinetics

Assessments of $\dot{V}O_2$ kinetics have predominantly used a square-wave (or step) transition(s) in the intensity of exercise which involves an abrupt change from rest (or low-intensity exercise) to moderate-intensity exercise. To ensure the intensity of exercise is moderate for all participants, the PO or speed is prescribed based on a percentage of the participant's V_T (Paterson and Whipp, 1991; Rossiter *et al.*, 1999) or LT (Carter *et al.*, 2000a; Williams *et al.*, 2001).

Breath-by-breath variability or 'noise' is an inherent characteristic of pulmonary gas-exchange measurements (Lamarra *et al.*, 1987). To minimise its effect, participants complete several square-wave transitions (Linnarsson, 1974; Whipp *et al.*, 1982; Lamarra *et al.*, 1987) either consecutively with appropriate rest periods, or on separate occasions. The data from each transition are interpolated and ensemble averaged to produce a single data set which is representative of the typical $\dot{V}O_2$ response. The kinetic response parameters are then obtained using exponential modelling techniques.

2.7.5 Modelling $\dot{V}O_2$ kinetics at the onset and cessation of exercise

Exponential modelling techniques, using least-squares non-linear regression, are used to determine the kinetic parameters that describe the temporal profile of $\dot{V}O_2$ at the onset and cessation of moderate-intensity exercise. It is generally acknowledged that the on- and off-transient $\dot{V}O_2$ response to a moderate-intensity, square-wave transition can be characterised adequately by a first-order exponential model incorporating a time-delay, constrained to start at the beginning of the phase II response (~20 s post onset of exercise) (Whipp *et al.*, 1982). This is to minimise distortion by early cardio-dynamic influences and therefore provides an accurate temporal representation of the exponential response during phase II. This model is widely used to characterise phase II $\dot{V}O_2$ kinetics during both the on- (Chilibeck *et al.*, 1998; Rossiter *et al.*, 1999) and off-transient (Paterson and Whipp, 1991; Özyener *et al.*, 2001). The output from this analysis consists of three kinetic parameters, described in terms of the on-transient kinetics these are: 1) time delay (δ), referring to the point after the onset of moderate-intensity exercise when $\dot{V}O_2$ starts to increase mono-exponentially (i.e. it is representative of the start of phase II); 2) τ, reflecting the rate at which $\dot{V}O_2$ is increasing and represents the time taken to reach 63% of the increase in $\dot{V}O_2$ from low-intensity (baseline) to moderate-intensity exercise and 3) amplitude term (A), representing the magnitude of the $\dot{V}O_2$ change from baseline conditions to the asymptotic steady-state $\dot{V}O_2$ achieved during moderate-intensity exercise. The τ is the most commonly referred to kinetic parameter, although earlier studies used the rate constant [k (Whipp and Casaburi, 1982; Weltman *et al.*, 1978)] or $\dot{V}O_2$ half-time [$\dot{V}O_2\, t_{½}$ (Diamond *et al.*, 1977; DeVries *et al.*, 1982; Powers *et al.*, 1985)] to describe $\dot{V}O_2$ kinetic responses.

Alternatively, some investigators have chosen to model their data using higher-order, double-exponential models (Barstow and Mole, 1991; Carter *et al.*, 2000a; Williams *et al.*, 2001). This approach models the phase I and phase II responses as separate components, each with independent δ, τ and A terms respectively. However, concerns

regarding the use of an exponential for phase I have been raised (Bell *et al.*, 2001), since experimental evidence supporting a physiological exponential increase during phase I has yet to be established.

Several additional physiological measures can be obtained from the kinetic parameter estimations obtained from mono-exponential modelling of the $\dot{V}O_2$ response. The mean response time (MRT) describes the overall rate of adjustment of $\dot{V}O_2$ and can be calculated from the sum of δ and τ (Linnarsson, 1974). This kinetic parameter has been previously used to describe the overall rate of response (Linnarsson, 1974). Most importantly, MRT has been shown to be more reproducible than τ (Kilding *et al.*, 2001). Furthermore, the MRT for the on- and off transient can be used to calculate the O_2 deficit and O_2 debt respectively (Linnarsson, 1974; Whipp *et al.*, 1982).

2.7.6 Symmetries between on- and off-transient kinetics

Berg (1947), Henry (1951) and Henry and De Moor (1956) were among the first to provide quantitative descriptions of the $\dot{V}O_2$ response and observed that the time course of change was generally similar between the adaptation to and the recovery from exercise. However, recent studies have demonstrated conflicting findings with respect to such symmetry between transients. In some studies, the $\dot{V}O_2$ kinetic response during the on- and off-transients have been found to be symmetrical (Paterson and Whipp, 1991; Yoshida and Whipp, 1994; Özyener *et al.*, 2001). Conversely, others have clearly demonstrated an asymmetry between the on- and off-transient responses (Hughson *et al.*, 1988; Carter *et al.*, 2000a).

Most investigations that have explored possible symmetry between the on- and off-transients at the onset and recovery from moderate-intensity exercise have involved cycle ergometry (Hughson *et al.*, 1988; Paterson and Whipp, 1991; Özyener *et al.*, 2001). However, fewer studies have assessed $\dot{V}O_2$ transient symmetries during treadmill exercise (Carter *et al.*, 2000a). This particular study measured $\dot{V}O_2$ kinetics

during cycle and treadmill exercise and demonstrated the on-transient kinetics (treadmill: 15.0 ± 2.0 s; cycle: 18.0 ± 4.0s) were shorter than the off-transient (treadmill: 39.3 ± 3.0 s; cycle: 35.9 ± 4.2 s, P <0.05) for both modes of exercise, suggesting that symmetry is independent of the mode of exercise. Disparity between this and previous findings could be attributable to different modelling techniques as well as the fitness of the participants.

2.7.7 Oxidative phosphorylation at exercise onset

At the onset of exercise, or when there is a sudden change in the intensity of exercise the rate of oxidative phosphorylation increases. This results from a decrease in the phosphorylation potential that stimulates the entry of ADP and P_i into the mitochondrion. The magnitude of the decrease in phosphorylation potential will be inversely related to the increase in the intensity of exercise. Simultaneously, the mitochondrial redox potential increases allowing electron transport from NADH to oxygen. The increase in redox potential will parallel increases in the intensity of exercise and occur when the rate of electron transfer from NADH to oxygen is not matched by the rate of formation of NADH through dehydrogenase enzymes. As NADH is oxidised to NAD^+, the inhibitory effect of NADH on the three irreversible TCA enzymes (pyruvate, isocitrate and α-ketoglutarate dehydrogenases) is reduced and the TCA cycle will speed up. Because of the initial mismatch between O_2 utilisation by the CO complex and O_2 delivery from the air to the fibre, the O_2 tension within the fibre will decline.

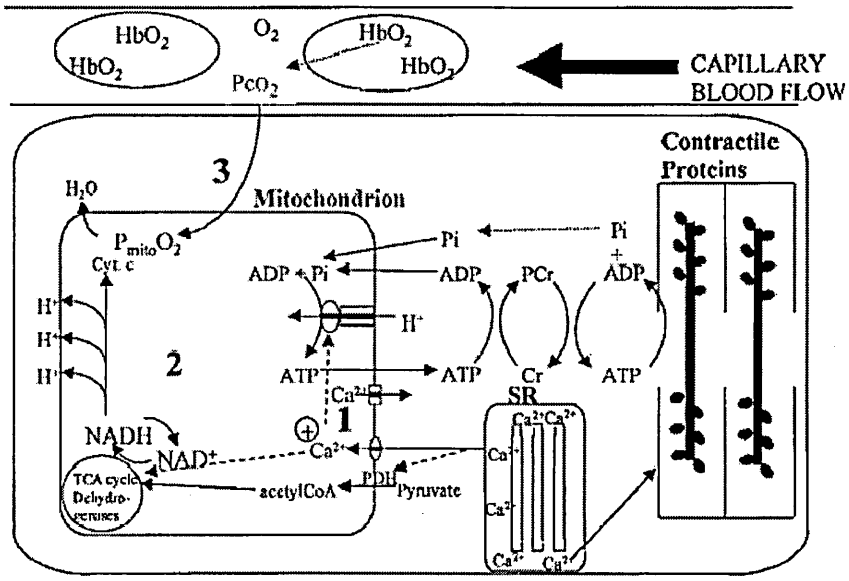


Figure 2.6 Schematic illustrating general mechanisms of oxidative phosphorylation. and local factors that might interact to determine muscle $\dot{V}O_2$ kinetics: 1) calcium levels in the mitochondrial matrix which activate dehydrogenases [i.e. lactate dehydrogenase (LDH)] and ATP synthase; 2) mitochondrial phosphorylation and redox potentials and 3) mitochondrial PO_2 ($P_{mito}O_2$) (Tschakovsky and Hughson, 1999).

The rate of oxidative phosphorylation can be increased by a combination of a decrease in phosphorylation potential, an increase in mitochondrial redox potential and a gradual increase in O_2 transport to the muscle mitochondria. Essentially, adjustments in phosphorylation potential and mitochondrial redox potential maintain oxidative phosphorylation in the face of declining O_2 availability to the respiratory chain (i.e. $P_{mito}O_2$). Any mismatch between ATP demand and ATP supplied at the onset of exercise, or to a change in the intensity of exercise, must be provided by PCr and anaerobic glycolysis. During transitions from low to moderate intensities, the former will predominate.

2.7.8 Mechanisms controlling $\dot{V}O_2$ at the onset of exercise

During the last 50 years, several models have been proposed to explain potential mechanism(s) of control. Primarily, these have involved kinetic and thermodynamic models of respiratory control.

2.7.8.1 Classic enzyme kinetic models of respiratory control

The classical kinetic (or acceptor) control model, proposed by Chance and Williams (1956), suggests that oxidative phosphorylation is controlled by ADP availability and thus there is a Michaelis-Menton type dependence of respiratory rate on cytoplasmic [ADP]. Inorganic phosphate (P_i) is generally assumed to play no role in kinetic control models because the Michaelis constant (K_m) of the mitochondrial P_i transport mechanism is several fold lower than the lowest [P_i] in resting muscle and thus must always be saturated (Meyer and Foley, 1994). However, the kinetic model has been questioned because increases in [ADP] at the mitochondria can be caused by changes in creatine ([Cr]) and [PCr] via mitochondrial isoenzymes of creatine phospho-kinase (CPK) (Whipp and Mahler, 1980).

Alternatively, the creatine-shuttle (Saks *et al.*, 1978; Bessman and Fonyo, 1981) hypothesis has been proposed as the most likely regulator of the dynamic response of $\dot{V}O_2$ (Whipp and Mahler, 1980). Conceptually, this model is similar to the classic ADP kinetic model but with ADP supply limited at the mitochondrial inner membrane by diffusion of Cr to the mitochondrial isozyme of creatine kinase (CK), rather than by cytoplasmic [ADP] *per se* (Meyer and Foley, 1996) (Figure 2.7). The creatine-shuttle hypothesis is plausible given the observed similarity between kinetics of $\dot{V}O_2$ and intra-muscular [PCr] in exercising humans (McCreary *et al.*, 1996; Rossiter *et al.*, 1999; 2002).

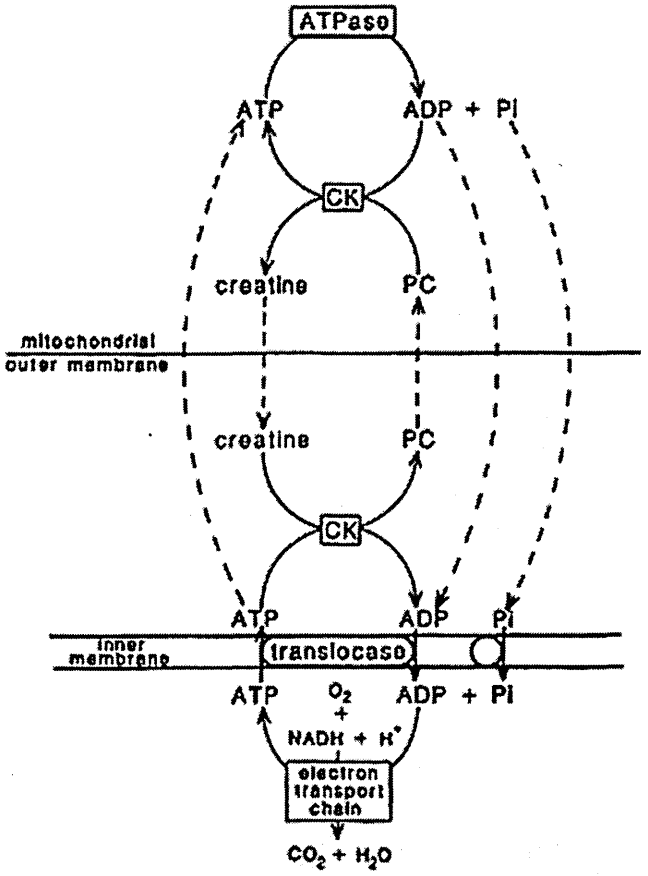


Figure 2.7 Simplified schematic diagram of the reaction by which oxygen consumption is coupled to extra-mitochondrial ATP hydrolysis in muscle via the creatine-shuttle [dashed lines indicate diffusion (Mahler, 1985)].

2.7.8.2 Thermodynamic models of respiratory control

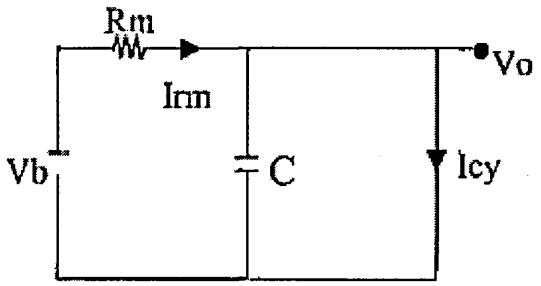
Classic enzyme models based on Michaelis-Menton kinetics have not always been applicable *in vivo*. Tschakovsky and Hughson (1999) highlighted the limitation of the Michaelis-Menten kinetic control model of oxidative phosphorylation in their review of factors determining $\dot{V}O_2$ at the onset of exercise. Primarily, it has been identified that kinetic models are unable to account for the large changes in ATP synthesis at the onset of exercise. As an alternative to kinetic control models, they proposed that cellular metabolic state (phosphorylation and redox potential), latent mitochondrial enzyme activation and $P_{mito}O_2$ are likely to be the most important determinants of the rate of mitochondrial respiration (Figure 2.6). However, given the strong evidence

demonstrating that O_2 availability is not limiting $\dot{V}O_2$ kinetics in normal conditions, then mechanism(s) of respiratory control within the muscle are likely to be the predominant determinants.

Thermodynamic models of respiratory control predict that mitochondrial redox potential ($NADH/NAD^+$), reflecting the degree of cytochrome *c* reduction, and phosphorylation potential ($[ATP]/[ADP]\times[P_i]$) determine respiratory rate such that it is not the absolute concentrations of substrates but rather the ratio of substrate to product which is the determining factor (Tschakovsky and Hughson, 1999). Compared with kinetic control models, thermodynamic models lead to two clear, testable predictions: 1) respiration rate should depend on cytoplasmic free energy of ATP hydrolysis, or phosphorylation potential, and hence should be sensitive to changes in cytoplasmic $[P_i]$ as well as to nucleotide levels and 2) the relationship between cytoplasmic phosphates and O_2 consumption should not be fixed by kinetic proprieties of mitochondrial enzymes but, in particular, should depend on the intra-mitochondrial $NADH/NAD^+$ ratio (Meyer and Foley, 1996).

To clarify such predictions, a series of experiments were conducted to investigate whether the relationship between respiration rate and phosphate metabolites (PCr, ATP and P_i) in intact muscle correspond to the predictions of kinetic or thermodynamic control models (Meyer *et al.*, 1985; Meyer, 1988). Collectively, these studies showed that the relationship between [PCr] and respiration rate is linear which favours thermodynamic regulation of O_2 consumption by cytoplasmic phosphorylation potential, rather than kinetic regulation by ADP (Chance and Williams, 1956).

In keeping with the thermodynamic control model of oxidative phosphorylation, Meyer (1988) proposed a simple electrical analog for first-order linear respiratory control (Figure 2.8).



Icy - cytosolic ATPase rate
Vo - cytosolic free energy of ATP hydrolysis
Vb - free energy potential available in the mitochondria which depends on the mitochondrial redox potential
C - PCr is the stored charge in the capacitor
Rm - mitochondrial resistance due to the number and properties of the mitochondria
Irm - rate of oxidative phosphorylation

Figure 2.8 Electrical analog model for linear first-order respiratory control of Meyer (1988) (cf. Tschakovsky and Hughson, 1999).

This model predicts that: 1) the response of $\dot{Q}O_2$ and PCr to a step-change in ATP demand is mono-exponential; 2) τ is a product of mitochondrial "resistance" (a function of the number and properties of the mitochondria) and the capacitance of the phosphate energy pool (total Cr); 3) the steady-state [PCr] is linearly related to $\dot{Q}O_2$ and the slope of this relationship depends on mitochondria resistance and 4) if mitochondrial redox state is relatively more reduced, the $\dot{Q}O_2$ vs. PCr slope will be unchanged, but the *y*-intercept will increase.

2.7.9 Mechanisms controlling $\dot{V}O_2$ at the onset of exercise

Investigations into mechanism(s) that control $\dot{V}O_2$ kinetics, and hence $\dot{Q}O_2$, at the onset of exercise have provided two opposing explanations. Namely, the rate of increase in oxidative phosphorylation is limited by the adaptation of O_2 utilisation (Whipp and Mahler, 1980; Barstow *et al.*, 1994) or O_2 transport (Hughson, 1990) mechanisms. An O_2 utilisation limitation reflects a metabolic inertia, meaning that the rate of oxidative phosphorylation at the onset of exercise is determined by levels of cellular metabolic controllers (Barstow *et al.*, 1994) and/or mitochondrial enzyme activation (Timmons *et al.*, 1996). An O_2 transport limitation reflects the inertia of O_2 delivery to the mitochondria. This implies that some of the oxidative metabolic

machinery is capable of increasing its rate of O_2 utilisation if more O_2 is made available (Hughson, 1990).

According to Walsh (1992), three methods can be used to identify the mechanism(s) controlling $\dot{V}O_2$ kinetics at the onset of exercise: 1) identifying the physiological compartment(s) with the same response characteristics as $\dot{V}O_2$; 2) modifying a process involved in the body's regulation of O_2 utilisation and/or O_2 transport and 3) implementing chronic interventions (i.e. exercise) and observing their effect on $\dot{V}O_2$ kinetics.

2.7.9.1 Similar compartment characteristics

One way to determine mechanism(s) that control $\dot{V}O_2$ is to characterise the kinetics of other physiological measures and assess whether or not they are temporally similar to those of $\dot{V}O_2$ kinetics. To date, this has included determining the kinetics of HR (Davies *et al.* 1972; Linnarsson, 1974), $\dot{Q}$ (Cerretelli *et al.*, 1966), blood flow (Grassi *et al.*, 1996) and [PCr] (Rossiter *et al.*, 1999; 2002).

If the rate of O_2 transport to the exercising muscle is a limiting factor in determining $\dot{V}O_2$ kinetics, as suggested by Hughson and Morrissey (1982), then the kinetics of $\dot{V}O_2$, HR, $\dot{Q}$ (and presumably blood flow) at the onset of exercise should be similar. However, several studies have demonstrated that HR (Davies *et al.* 1972; Linnarsson, 1974), $\dot{Q}$ (Cerretelli *et al.*, 1966) and blood flow kinetics (Grassi *et al.*, 1996) are faster than $\dot{V}O_2$ kinetics at the onset of exercise. This suggests that O_2 transport is not limiting $\dot{V}O_2$ kinetics. However, there is some evidence that shows similarities between $\dot{V}O_2$ and $\dot{Q}$ kinetics (Yoshida *et al.*, 1993) and, $\dot{V}O_2$ and HR kinetics (Hughson and Morrissey, 1983). These inconsistent findings could be attributed to contrasting modes of ergometry and/or different protocols used to measure $\dot{V}O_2$ kinetics.

The kinetics of [PCr] degradation at the onset of exercise might play a role in the control of mitochondrial respiration and thus $\dot{V}O_2$ kinetics. PCr kinetics are thought to reflect the kinetics of $\dot{Q}O_2$ (Whipp and Mahler, 1980) that are expressed at the lungs, following a transport delay, as pulmonary $\dot{V}O_2$ kinetics (Barstow *et al.*, 1990; Grassi *et al.*, 1996; Rossiter *et al.*, 1999). Both nuclear magnetic resonance (NMR) spectroscopy and 31-phosphorus magnetic resonance spectroscopy (^{31}P-MRS) have been used in several studies investigating the temporal behaviour of [PCr] at the onset (Barstow *et al.*, 1994; McCreary *et al.*, 1996; Rossiter *et al.*, 1999; 2002) and during recovery (McCreary *et al.*, 1996; Rossiter *et al.*, 2002) from moderate-intensity exercise. These studies have demonstrated a direct proportionality between [PCr] kinetics and pulmonary $\dot{V}O_2$ kinetics in exercising humans (Figure 2.9) suggesting that the phase II τ for $\dot{V}O_2$, measured at the mouth, provides a good estimate of the τ for [PCr] and by implication the τ of $\dot{Q}O_2$.

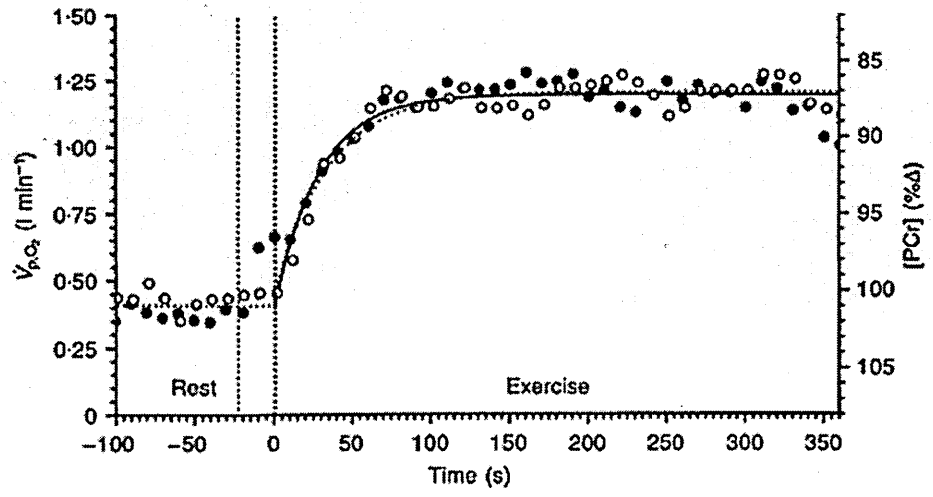


Figure 2.9 Temporal response of $\dot{V}O_2$ (●) and [PCr] (○) to a step change in exercise intensity of moderate-intensity for a representative participant (Rossiter *et al.*, 1999).

2.7.9.2 Manipulations of O_2 transport

Evidence to support the hypothesis that $\dot{V}O_2$ kinetics is limited by an O_2 transport mechanism has been derived from experimental models that have reduced O_2

availability. For example, in conditions of hypoxia, obtained by reducing arterial partial O_2 pressure, a slowing of $\dot{V}O_2$ kinetics has been observed (Linnarsson, 1974; Murphy *et al.*, 1989). Alternatively, but with similar physiological consequences (reduced O_2 delivery), the prescription of β-adrenergic receptor blockade drugs to reduce $\dot{Q}$ in healthy participants has also been shown to slow $\dot{V}O_2$ kinetics (Hughson and Kowalchuk, 1991).

Body position, through its effect on blood flow, has also been shown to influence $\dot{V}O_2$ kinetics (MacDonald *et al.*, 1998). In the supine position, MRT for knee-extension exercise was slower than when similar exercise was completed in the upright position. The slower $\dot{V}O_2$ kinetics could be due to a reduced supply of O_2 since leg blood flow ($\dot{Q}_{leg}$) kinetics were also slowed in the supine position (27.6 ± 3.9 vs. 17.3 ± 4.0 s, P <0.05). This demonstrates that when O_2 transport is temporarily impaired, a concomitant slower rate of oxidative phosphorylation occurs.

To clarify the role of blood flow on $\dot{V}O_2$ kinetics, Williamson *et al.* (1996) used lower body positive pressure to reduce skeletal muscle blood flow during leg exercise and observed its effect on $\dot{V}O_2$ kinetics. However, in contrast to the slowed $\dot{V}O_2$ and $\dot{Q}_{leg}$ kinetics observed by MacDonald *et al.* (1998), impaired blood flow (and presumably O_2 transport) did not slow $\dot{V}O_2$ kinetics during moderate-intensity exercise. This finding suggests that $\dot{V}O_2$ kinetics are independent of blood flow and that there is normally an excess of blood flow, and thus O_2 availability, to the exercising muscle. These contrasting findings could be attributable to different modes of exercise (knee extension vs. cycling) and subtle differences in body position (upright vs. semi-upright), both of which could influence muscle recruitment patterns and cause the distribution of oxygenated blood to differ.

If O_2 transport mechanisms limit $\dot{V}O_2$ kinetics at the onset of exercise, then in addition to slowing $\dot{V}O_2$ kinetics in conditions of O_2 transport impairment, enhancing the rate of

O_2 transport to exercising muscle should result in a speeding of $\dot{V}O_2$ kinetics. Experimental approaches to demonstrate this have increased O_2 transport to exercising muscles via circulatory occlusion of the legs prior to the onset of supine arm exercise (Hughson and Inman, 1986b), performing rhythmic hand-grip exercise below (vs. above) heart level (Hughson *et al.*, 1996) and exercising in conditions of hyperoxia (Linnarsson, 1974). All of which have been found to speed $\dot{V}O_2$ kinetics.

With respect to the speeding of $\dot{V}O_2$ kinetics in conditions of hyperoxia, MacDonald *et al.* (1997) demonstrated partially conflicting findings. Hyperoxia did not speed $\dot{V}O_2$ kinetics for exercise below the V_T compared with normoxia, but accelerated the on- and off-transient MRT as well as reducing the $\dot{V}O_2$ slow component for exercise above the V_T. This suggests that different muscle fibre types are affected by manipulations in O_2 delivery in different ways. It would appear that Type II fibres benefit from additional O_2 delivery during heavy-intensity exercise. Other investigations have also found that hyperoxia speeds $\dot{V}O_2$ kinetics for heavy but not moderate-intensity exercise (Hughson and Kowalchuk, 1995). This suggests that O_2 transport is not the rate limiting mechanism determining the rate of $\dot{V}O_2$ with respect to moderate-intensity exercise below the V_T, but might be more important for exercise above the V_T.

More direct evidence to support an O_2 utilisation mechanism is provided by unchanged $\dot{V}O_2$ kinetics in the presence of faster $\dot{Q}$ kinetics in heart-transplant recipients (Grassi *et al.*, 1997). Faster $\dot{Q}$ kinetics was obtained by a "priming" exercise, the purpose of which was to establish more favourable conditions with regard to the adjustment of O_2 delivery to the increased metabolic demand. While effective in speeding up the adjustment of convective O_2 flow to muscle fibres, increased O_2 delivery did not affect $\dot{V}O_2$ on-transient kinetics suggesting that slow $\dot{V}O_2$ kinetics were attributable to peripheral (muscular) factors and not deficiencies in O_2 transport.

The disagreement between findings in most studies could be attributed to differences in the characteristics and number of participants, number of transitions performed and the mode of exercise. Some studies have used small muscle groups (Hughson *et al.*, 1996) to permit the non-invasive measurement of blood flow during exercise. However, the reduced amplitude of $\dot{V}O_2$ which inevitably results during small muscle group exercise would be more sensitive to the effects of breath-by-breath noise and therefore could affect kinetic parameter estimations. Extrapolation of the above findings to larger muscle groups should be done with caution as the physiological stress associated with the recruitment of a larger muscle mass will differ. Clarity with respect to the intensity of exercise is important in studies measuring $\dot{V}O_2$ kinetics since exercise above and below the V_T results in differing physiological responses that can influence $\dot{V}O_2$ kinetics (MacDonald *et al.*, 1997).

Clear evidence suggesting that O_2 supply does not limit O_2 utilisation during moderate-intensity exercise has been demonstrated by Grassi *et al.* (1998a). Using isolated *in situ* canine gastrocnemius muscle, it was demonstrated that increased O_2 delivery, by pump perfusing the muscle with elevated blood flow from the last seconds of rest thus eliminating any delay in O_2 delivery, did not accelerate muscle $\dot{V}O_2$ kinetics in the initial phase of electrically induced muscle activity. In response to this finding, a further study by the same research group investigated the effects of enhanced peripheral O_2 diffusion on $\dot{V}O_2$ kinetics (Grassi *et al.*, 1998b). It was hypothesised that if peripheral O_2 diffusion were indeed limiting $\dot{V}O_2$ kinetics, then the increase in the driving pressure for O_2 from capillaries to mitochondria would determine, in the presence of constant convective O_2 transport, a faster $\dot{V}O_2$ kinetic response. Although peripheral O_2 diffusion was enhanced by increasing the driving pressure for O_2 from muscle capillaries to the mitochondria, muscle $\dot{V}O_2$ kinetics were unchanged. Collectively, the results of these two studies suggest that convective and diffusive O_2 delivery to exercising muscle does not influence $\dot{V}O_2$ kinetics at the onset of exercise. Unfortunately, the findings of Grassi *et al.* (1996; 1998a) do not permit a definitive

discrimination between the possible intra-muscular mal-distribution of blood flow and the inertia of the intra-cellular oxidative machinery as limiting mechanisms. This is due to the spatial and temporal heterogeneity of blood flow within active muscle (Piiper *et al.*, 1985), a factor that Grassi *et al.* (1996; 1998a) recognise.

Extrapolation of the above findings to exercising humans should be made with caution since there are several technical limitations to consider. First, the above studies were performed using isolated *in situ* canine gastrocnemius muscle. The fibre type composition of this muscle (predominantly Type I fibres) is considered to be different to that of human muscle, but less so when endurance athletes are considered (Grassi *et al.*, 1998a). Second, muscle activity was obtained by electrical stimulation that produced synchronous tetanic actions of all fibres within the muscle. Simultaneous activity of all fibres could be considered un-physiological compared to asynchronous, heterogeneous fibre activation in voluntary contracting muscle during cycling and running exercise in humans. This type of muscle stimulation might have produced intra-muscular blood pooling (Grassi *et al.*, 1998a). However, this pooling was expected to be minimal since the action of the muscle was every 2 s thus allowing extrusion of the blood from the muscle between active phases. Third, the transient was from rest to an intensity of 60-70% of peak $\dot{V}O_2$ ($\dot{V}O_{2\,peak}$) and therefore might only to apply to exercise performed under these conditions. Square-wave transitions from a low-intensity to moderate- or heavy-intensity might result in a different muscle $\dot{V}O_2$ kinetic response.

Although there is some evidence to suggest that enhancing and impairing O_2 transport mechanisms influences the $\dot{V}O_2$ kinetic response under some experimental conditions, confidence in some early finings might be reduced because of weaknesses in experimental design (e.g. sample size and number of transitions). This is probably the primary reason for the conflicting results. However, recent *in situ* and *in vivo* experiments (Grassi *et al.*, 1998a and 1998b) have provided strong evidence to suggest that neither O_2 delivery or O_2 diffusion mechanisms determine the rate of oxidative

phosphorylation at the onset of moderate-intensity exercise. Further work to replicate these findings in humans is necessary to confirm this conclusion.

2.7.9.3 O_2 utilisation

Evidence to suggest that the rate-limiting factor for $\dot{V}O_2$ kinetics resides in the utilisation of O_2 within the exercising muscle initially comes from studies that manipulated O_2 delivery mechanisms and found that $\dot{V}O_2$ kinetics were not affected (e.g. Grassi *et al.*, 1997). This suggests that factors within muscle are determining $\dot{V}O_2$ kinetics. However, the pathways of oxidative phosphorylation are complex and the exact location of the limiting step(s) is difficult to ascertain. To date, several O_2 utilisation-related limiting steps have been hypothesised.

Recent studies have proposed the activity of pyruvate dehydrogenase (PDH) as the limiting factor for $\dot{Q}O_2$ (Timmons *et al.*, 1998a and 1998b; Howlett *et al.*, 1999). The enzyme PDH catalyses the de-carboxylation of pyruvate, forming acetyl-CoA, which can be subsequently used in the TCA cycle. To enhance the activity of PDH, these studies have employed the pharmacological agent dichloroacetate (DCA), which activates the PDH enzyme complex, resulting in an increased oxidation of glucose and lactate and an amelioration of lactic acidosis (Stacpoole *et al.*, 1998). The administration of DCA has been found to increase the active fraction of PDH in skeletal muscle at rest (Timmons *et al.*, 1998a; 1998b; Gibala and Saltin, 1999) and attenuate substrate level phosphorylation during rest-to-work transitions in animals (Timmons *et al.*, 1996; 1997) and humans (Timmons *et al.*, 1998a; 1998b). Specifically in humans, pre-treatment with DCA resulted in a reduction in PCr utilisation and muscle lactate concentration [MLa] during sub-maximal knee-extension (Timmons *et al.*, 1998a) and cycle exercise (Howlett *et al.*, 1999). Collectively, these findings suggest that the provision of oxidative substrate is one factor limits oxidative phosphorylation early in exercise and that increasing the availability for substrate early in exercise allows for increased oxidative phosphorylation and decreased reliance on substrate

phosphorylation. The attenuation of anaerobic energy provision could be attributed to a faster adjustment of oxidative phosphorylation as a consequence of reducing the proposed inertia in substrate supply to the TCA cycle (Timmons *et al.*, 1996, 1997, 1998a and 1998b; Howlett *et al.*, 1999).

Until recently, there was no experimental evidence to support an acceleration in $\dot{V}O_2$ kinetics after DCA administration. However, Grassi *et al.* (2002) simultaneously measured muscle $\dot{V}O_2$ on-kinetics, [PCr] degradation and [MLa] in dog gastrocnemius *in situ* following activation of PDH by DCA administration. The DCA administration resulted in a significant activation of PDH as evidenced by a marked stockpiling of acetyl-carnitine at rest. However, conversely to the hypothesised speeding of muscle $\dot{V}O_2$ on-kinetics after DCA administration, $\dot{V}O_2$ kinetics were slower (control τ = 15.6 ± 0.7 s; DCA τ = 19.5 ± 1.7 s, P <0.05). Furthermore, the amplitude of $\dot{V}O_2$, [PCr] degradation and [MLa] between control and DCA conditions did not differ (P >0.05) suggesting that the stockpiling of acetyl groups at rest did not affect 'anaerobic' energy provision. This is contradictory to previous studies whereby DCA resulted in a significant reduction in [PCr] degradation (Timmons *et al.*, 1996, 1997, 1998a, b; Howlett *et al.*, 1999a). The findings of Grassi *et al.* (2002) suggest that PDH activation status is not responsible for the metabolic inertia in the adjustment of oxidative phosphorylation to sudden increases in the demand for ATP re-synthesis. These conflicting findings might be due to the experimental model used, especially with respect to the muscle preparation (*in situ* canine gastrocnemius) and the method of contraction (electrical isometric tetanic stimulation).

2.7.10 Application of measures of $\dot{V}O_2$ kinetics

Owing to the non-invasive and sub-maximal nature of measuring $\dot{V}O_2$ kinetics, there has been a wide and varied application of this approach. This has included medical applications involving patients with heart failure (Hepple *et al.*, 1999), heart transplants (Grassi *et al.*, 1997), peripheral arterial disease (Bauer *et al.*, 1999) and diabetes

(Brandenburg *et al.*, 1999). Whereas other investigations have focused on the effects of chronic interventions on $\dot{V}O_2$ kinetics such as de-conditioning bed rest (Eßfeld *et al.*, 1984) and exercise (Hagberg *et al.*, 1980; Phillips *et al.*, 1995). Collectively, these have been conducted to identify the physiological process(s) or mechanism(s) that regulates $\dot{V}O_2$ kinetics by observing and comparing possible couplings with other related physiological processes.

2.7.10.1 The effects of endurance training on $\dot{V}O_2$ kinetics

Several investigations have quantified the effects of endurance training on $\dot{V}O_2$ kinetics during the on- (Cerretelli *et al.*, 1979; Hagberg *et al.*, 1980; Phillips *et al.*, 1995; Norris and Peterson, 1998; Carter *et al.*, 2000b) and off-transient (Hagberg *et al.*, 1980; Phillips *et al.*, 1995). Generally, physiological adaptations induced by endurance training interact to accelerate $\dot{V}O_2$ at the onset (Cerretelli *et al.*, 1979; Berry and Moritani, 1985; Babcock *et al.,* 1994) and offset (Hagberg *et al.*, 1980; Phillips *et al.*, 1995) of moderate-intensity exercise. Several factors including an improved capacity for mitochondrial respiration in muscle, increased availability of blood and/or muscle O_2 stores, elevation of HR and $\dot{Q}$ thus causing increased muscle blood flow, could contribute to the speeding of $\dot{V}O_2$ kinetics after training.

Comparisons of the time course and magnitude of change in $\dot{V}O_2$ kinetics due to training should be viewed in consideration of the intensity and duration of time spent training. Changes in $\dot{V}O_2$ kinetics appear relatively consistent and demonstrate changes after approximately 8 hours of endurance training (Yoshida *et al.*, 1992; Phillips *et al.*, 1995). Faster $\dot{V}O_2$ kinetics during the on-transient have been observed with as little as 4 days of endurance training with further changes being significantly correlated with training days i.e. $\dot{V}O_2$ kinetics become progressively faster as training progressed (Yoshida *et al.*, 1992; Phillips *et al.*, 1995).

Different intensities of exercise have been shown to influence changes in $\dot{V}O_2$ kinetics. (Berry and Moritani, 1985). In their study, heavy-intensity interval training consisting of short distance sprints at 85-95% of maximum heart rate reserve (HR_{res}) with jogging recovery had a greater influence on the speeding of $\dot{V}O_2$ kinetics than continuous steady-state training (60-70% of HR_{res}), although the total distance covered by each group was equal. Exercising at 85-95% of HR_{res} can be considered a heavy-intensity aerobic session, whereas 60-70% is a moderate-intensity session.

Despite increasing $\dot{V}O_{2\,max}$, it has been reported that supra-maximal training (sprinting) does not change the amplitude or phase shift responses of $\dot{V}O_2$ to a sinusoidal WR forcing function (Fukuoka *et al.*, 1997). This might suggest that the physiological mechanism(s) determining $\dot{V}O_2$ kinetics are different to those determining $\dot{V}O_{2\,max}$. In support, Carter *et al.* (2000b) reported no change in the $\dot{V}O_2$ kinetics after 6 weeks of endurance training, despite significant increases in $\dot{V}O_{2\,max}$ and LT. The lack of change in this study however, could be attributed to the moderate-high fitness of participants prior to training and also to the number of square-wave transitions that were performed (n=3) by participants, which might not have been sufficient to identify any small but significant changes in $\dot{V}O_2$ kinetics.

The apparent sensitivity of $\dot{V}O_2$ kinetics to different training strategies warrants further consideration of the exact nature of training with respect to the intensity and duration as this might influence changes in $\dot{V}O_2$ kinetics. Also, the disassociation between changes in $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ in some studies (Fukuoka *et al.*, 1997; Carter *et al.*, 2000b) as well as the effects on other aerobic measures such as V_T/LT requires further attention.

2.7.10.2 Mechanisms of training-induced adaptations in $\dot{V}O_2$ kinetics

There are several mechanisms that could explain the accelerated $\dot{V}O_2$ kinetics after endurance training. The fact that $\dot{V}O_2$ kinetics are considerably faster in only the

trained muscles, suggests that mechanisms within the muscle are primarily involved in the training response (Cerretelli *et al.*, 1979). After prolonged endurance training, an increase in the oxidative potential of the active muscles is likely (Holloszy and Coyle, 1984). Aerobic training is known to increase the mitochondrial content of skeletal muscle (Saltin and Gollnick, 1983) which might contribute to the speeding of $\dot{V}O_2$ kinetics. Several studies have reported that an increased rate of O_2 utilisation is brought about by improved mitochondrial function that attenuates [HLa] (Hagberg *et al.*, 1980; Yoshida *et al.*, 1992). This would be in accordance with Meyer's (1988) first-order model of respiratory control (Figure 2.8) which suggests that τ is a product of mitochondrial "resistance" (a function of the *number* and *properties* of the mitochondria). A high percentage of Type I muscle fibres that are better equipped to use O_2 than Type II fibres has also been suggested to result in the speeding of $\dot{V}O_2$ kinetics (Weltman and Katch, 1976; Powers *et al.*, 1985).

Exercise-induced adaptations of $\dot{V}O_2$ kinetics are not always attributed to enhanced O_2 utilisation mechanisms. Phillips *et al.* (1995) reported that although $\dot{V}O_2$ kinetics were speeded after 4 days of endurance training, no concomitant increases in muscle oxidative potential [citrate synthase (CS) and SDH activity] or $\dot{V}O_{2\,max}$ were observed. This suggests that the early adaptation of $\dot{V}O_2$ kinetics to endurance training is not caused by changes in muscle. Several other characteristic training adaptations including lower [HLa], reduced [PCr] and glycogen depletion were also observed, but occurred before increases in mitochondrial potential. After 30 days training, further adaptations in muscle metabolism and muscle phosphorylation potential occurred as well as an increase in muscle mitochondrial capacity. The initial speeding of $\dot{V}O_2$ kinetics in the early stages of training was attributed to an increase in femoral artery blood flow leading to accelerated O_2 transport to the exercising muscle. Other work, demonstrating that short-term endurance training results in an increase in $\dot{Q}$, femoral artery mean blood and vascular conductance support this (Shoemaker *et al.*, 1996), assuming that the increased blood flow was directed to the active muscle fibres. Although, changes in

oxidative enzymes were not observed in the early stages of training, this can not exclude an O_2 utilisation mechanism as the reason for the speeding of $\dot{V}O_2$ kinetics. It is possible that other enzymes involved in oxidative phosphorylation, such as PDH, which were not measured could contribute to the speeding of the $\dot{V}O_2$ kinetic response. Future studies should consider and measure the responses of several enzymes involved in different metabolic pathways to ensure that important adaptations are not overlooked.

It is possible that endurance training could result in a reduction in the diffusion distance of O_2. In support, it has been shown that the size of the capillary-to-fibre interface is matched to mitochondrial volume/fibre length with adaptation to training (Poole and Mathieu-Costello, 1996). This would provide favourable conditions for O_2 to diffuse from the circulating blood and into the mitochondria at a faster rate and contribute to a speeding of $\dot{V}O_2$ kinetics.

Adaptations in the delivery and utilisation of O_2, induced by endurance training appear to interact to accelerate oxidative phosphorylation at the onset of moderate-intensity exercise. However, after the early stages of exercise, it appears that O_2 utilisation mechanisms predominate in the training responses. Identifying the time course, magnitude and relative contribution of O_2 delivery and O_2 utilisation related mechanisms to the speeding of $\dot{V}O_2$ kinetics would be useful to identify and could be focus for future work.

2.7.11 The characterisation of $\dot{V}O_2$ kinetics in MD and LD runners

Despite the potential relevance of $\dot{V}O_2$ kinetics as a physiological measure in athletes, as demonstrated by its sensitivity to training stimuli, only a few studies have characterised the on- (Cerretelli *et al.*, 1979; deVries *et al.*, 1982; Powers *et al.*, 1985; Lake *et al.*, 1986) and off-transient (Cerretelli *et al.*, 1979) $\dot{V}O_2$ kinetics in MD and LD runners during moderate-intensity exercise. The majority of these studies have been conducted using cycle ergometry which involves a non-sport specific exercise. Other

studies involving runners and measures of $\dot{V}O_2$ kinetics during running have been conducted using heavy-intensity exercise and involved few transitions (Demarle *et al.*, 2001; Billat *et al.* 2001). To date, no study has simultaneously reported on- and off-transient $\dot{V}O_2$ kinetics during treadmill running in MD and LD runners.

2.7.12 The application of $\dot{V}O_2$ kinetics to performance

Few studies have considered the effects of training on $\dot{V}O_2$ kinetics and performance. In previously trained competitive cyclists, Norris and Peterson (1998) reported that τ and 40 km cycling performance time significantly decreased during 8 weeks of endurance training. In the early stages of training (week 4), τ was reduced with concomitant increases in $\dot{V}O_{2\,max}$, $\dot{V}O_2$ at the V_T and the PO at the V_T. However, at the post-training assessment (week 8), further reductions in τ and 40 km performance time were observed, but with no further increases in $\dot{V}O_{2\,max}$ or V_T. This suggests that $\dot{V}O_2$ kinetics is more sensitive to changes in physiological status than $\dot{V}O_{2\,max}$ and V_T and that changes in $\dot{V}O_2$ kinetics are more closely associated with improvements in cycling performance. Despite the findings of Norris and Peterson (1998), the association between $\dot{V}O_2$ kinetics and performance in other sports, i.e. running, has not been investigated. The only evidence of $\dot{V}O_2$ kinetics being considered with respect to running performance was speculation that: 1) the rate at which $\dot{V}O_2$ increases at the start of the race is more important in determining performance in MD events than it is in LD events and 2) there seems little purpose in training to increase $\dot{V}O_2$ kinetics in the MD-LD events, as the 1500 and 3000 m are already run at approximately 100% $\dot{V}O_{2\,max}$ (Wood, 1999). It would appear that these authors are suggesting that a relationship exists between $\dot{V}O_2$ kinetics and running performance in MD events, presumably on the basis of a reduced oxygen deficit in the faster performers. Indeed, one advantage of faster $\dot{V}O_2$ kinetics is that the O_2 deficit will be smaller, which inevitably reduces the intra-cellular perturbation at a given intensity (Poole and Richardson, 1997). A reduced O_2 deficit and faster $\dot{V}O_2$ kinetics will enhance intra-cellular energetics, reduce anaerobic glycolysis and promote fat utilisation, which will help conserve intra-

muscular glycogen reserves (Poole and Richardson, 1997). Therefore, the relevance of $\dot{V}O_2$ kinetics to running events of moderate- to heavy-intensity for prolonged periods, i.e. 5000 m - marathon, appears justified.

It is likely that fluctuations in running speed occur during a competitive race. Potentially, an individual with fast $\dot{V}O_2$ kinetics would be able 'physiologically' to respond rapidly to changes in the intensity of exercise during a race which would help attenuate PCr degradation, [HLa] and require less assistance from substrate phosphorylation. During LD events, especially the marathon, this might serve to maintain performance levels as a result of a glycogen sparing effect. It has been proposed that faster $\dot{V}O_2$ kinetics would result in an increase in endurance at all sub-maximal intensities (Poole and Richardson, 1997). Such possibilities warrant the investigation of the importance of $\dot{V}O_2$ kinetics in MD and LD runners and its contribution to predicting running performance.

CHAPTER 3

Methods

The following methods are applicable to the individual studies completed as part of this thesis. This chapter includes a detailed description of: 1) the equipment used and its calibration; 2) exercise tests and protocols; 3) data preparation and analysis techniques and 4) methods of statistical analysis.

3.1 Equipment and calibration

The equipment used throughout each investigation is described in four separate sections. These are: 1) treadmill ergometry; 2) mass spectrometry (including calculations of alveolar gas exchange); 3) HR monitoring and 4) [HLa] analysis.

3.1.1 Treadmill ergometry

For the assessment of sub-maximal and maximal physiological responses, a treadmill (Saturn 250-75R, HP Cosmos, Germany) was used with a running surface length of 250 cm and width of 75 cm. Speed ranged between 0 and 40 $km \cdot h^{-1}$ (0 - 11.11 $m \cdot s^{-1}$) with 7 acceleration/deceleration possibilities (from 0 to 40.0 $km \cdot h^{-1}$ in 3 to 131 s). Elevation of the treadmill belt operated between 0 and 25% (0 - 14°) with adjustable electronic resolution (0.1%). Verification data for treadmill speed and gradient are provided in Appendix 2.

3.1.2 Mass spectrometry

Measurements of inspired and expired respiratory gas concentrations were made using a respiratory mass spectrometer (MGA-1100, Marquette Electronics Inc., Milwaukee, WI, USA). This particular mass spectrometer has been found to measure $\dot{V}O_2$ values that were not significantly different from Douglas bag methods (Babineau *et al.*, 1999). Breath-by-breath inspiratory and expiratory gas flows were measured using a bi-directional flow turbine (VMM 110, Interface Associates, Laguna Niguel, CA, USA). The ventilatory volume is determined indirectly as the integral of flow against time.

Ventilation and gas concentration values were digitally sampled at a frequency of 200 Hz. Four of the primary analogue signals generated by the mass spectrometer and ventilation system in response to several breathing cycles are shown in Figure 3.1.

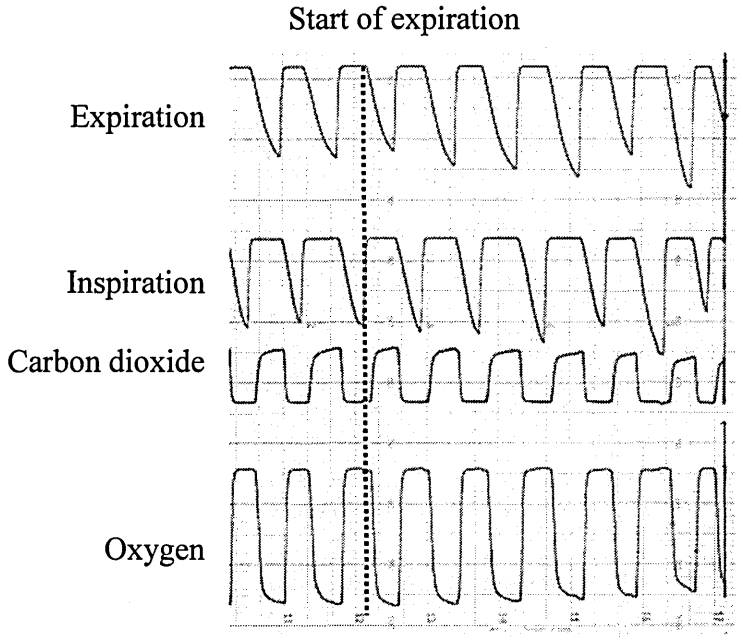


Figure 3.1 Analogue signals (CO_2 and O_2) generated by the mass spectrometer in relation to the ventilation signal generated by the flow turbine.

The signals from the mass spectrometer and flow measurement system were interfaced with a PC-compatible desktop computer (Ti'ko PS 325C, Ti'ko Computer Corporation, Broxburn, UK) via an analogue-to-digital converter. The signals were integrated on-line using custom-built software (First Breath Software *v*2.0, First Breath Inc., St Agatha, Ontario, Canada, 1992). This software provides estimates of alveolar gas-exchange based on the algorithm of Beaver *et al.* (1981).

3.1.2.1 Correction of gas volumes

When making comparisons between tests carried out under different atmospheric conditions it was necessary to apply a correction factor to account for the effects of differences in ambient temperature, pressure and water vapour on measured volumes. Standard temperature and pressure dry (STPD) was used for all metabolic calculations and is calculated as a dry gas at a temperature of 273 K and a pressure of 760 mmHg. When referring to a physiological measure such as $\dot{V}E$, body temperature and pressure

saturated (BTPS) was used [temperature of 310 K, ambient pressure and saturated with water vapour (Anderson *et al.*, 1971; Fox *et al.*, 1993)].

3.1.2.2 Calibration of the mass spectrometer

The mass spectrometer (MGA 1100, Marquette Electronics Inc., Milwaukee, USA) was calibrated immediately before and then verified after each exercise test. The calibration process involved three systematic stages: 1) gas calibration; 2) volume calibration and 3) analyser lag-time calibration.

3.1.2.2.1 Gas calibration

Calibration of the mass spectrometer was undertaken with two high tolerance (± 0.03%) calibration and reference gases (Medgraphics, US) of known composition (12% O_2, 5% CO_2, Bal N_2 and 21% O_2, 0% CO_2, Bal N_2). A two-point calibration was performed, 12% and 21% for O_2 and 0 and 5% for CO_2. A continuous sample of calibration gas was delivered down the sample line of the mass spectrometer at a rate representative of a normal physiologic breath. A successful calibration resulted in measurement of this reference gas ± 0.03%. To verify the calibration procedure, a 'pre-test' check was completed using the reference gases.

3.1.2.2.2 Volume calibration

A 3-litre syringe (Hans Rudolf Inc., Kansas City, MO, USA) was used to calibrate the low-dead-space (90 ml), low resistance (<1.5 cmH_2O at 3 $l \cdot s^{-1}$) volume turbine sensor (VMM 110, Alpha Technologies, Laguna Niguel, CA, USA) before each exercise test. Inspiration and expiration flow rate was similar to that of human ventilation during exercise (2 $l \cdot s^{-1}$). Three practice inspirations and expirations were performed prior to five recorded inspirations and expirations that were used for the calibration. Following this, the volume turbine sensor was re-checked by repeatedly pumping the syringe through the volume turbine at the desired flow rate with a required tolerance of ± 1% (Murphy *et al.*, 1989).

3.1.2.2.3 Analyser lag time

According to MacFarlane (2001), one of the greatest problems in modern gas-analysis systems is the temporal alignment of gas volumes and gas compositions. Calibration of the lag time was performed to ensure correct alignment between the ventilation signal and the measurement of gas compositions prior to performing the integrations necessary to determine $\dot{V}O_2$ and $\dot{V}CO_2$. The volume turbine sensor measures ventilation and produces a signal almost instantaneously. However, the signal from the mass spectrometer is delayed by the time required to transport the gas to the analysers and the response time of the individual analysers. The time-delay between the two signals is known as the 'lag-time'. The lag time measured during the lag time calibration is used to align gas volume and composition on a breath-by-breath basis. Incorrect alignment of the signals can result in significant errors (30%) in the determination of $\dot{V}O_2$ (Proctor and Beck, 1996).

An algorithm (First Breath software *v*2.0, First Breath Inc., St Agatha, Ontario, Canada, 1992) to determine τ for the exponential rise in measured CO_2 concentration, was used to calculate: 1) the lag-time between the signal change at the start of inspiration, detected by the turbine flow meter and 2) the rise time in the concentration in the CO_2 signal measured by the mass spectrometer. The systems lag-time was determined by exhaling through the assembled mouthpiece at a constant rate and then inhaling maximally. The actual lag-time is system specific i.e. it is dependent on the length and bore of the sample line and time taken to process the electronic signal (Arieli and Van Liew, 1981). Throughout the present investigations the lag time was stable at ~300 ms.

3.1.2.3 Estimation of alveolar gas exchange

The mass spectrometer, volume turbine and the First Breath *v*2.0 software were used to derive a breath-by-breath estimation of alveolar gas-exchange based on the algorithm of Beaver *et al.* (1981). This method requires an estimate to be made of effective lung volume (ELV). The initial estimate for the ELV was made using a volume equal to half the estimated functional residual capacity (FRC) obtained from normal tables which

take into account the stature, gender and age of the individual. The algorithm of Beaver *et al.* (1981) accounts for changes in pulmonary gas stores on a breath-by-breath basis and has been shown to have a flow meter error sensitivity of less than one (Swanson *et al.*, 1981). The sensitivity to expiratory error can be large in traditional methods that do not correct for changes in pulmonary gas stores. Therefore this approach is the method of choice for studies of transient gas-exchange analysis during exercise.

3.1.2.4 Reduction of breath-by-breath variability

An inherent characteristic of breath-by-breath measures of $\dot{V}O_2$ is random variability. This is commonly referred to as breath-by-breath 'noise' (Lamarra *et al.*, 1987). To minimise the effects of noise, breaths that are not considered to be reflective of the underlying response are discounted. Therefore, unusual, non-physiologic breaths caused by swallowing, coughing and sighing etc., were identified and omitted prior to subsequent data analysis and mathematical modelling. The First Breath *v*2.0 software filter was used to identify anomalous breaths from the data set.

3.1.3 Heart rate

The HR of participants was measured and recorded at 5 s intervals throughout all exercise tests using short-range telemetry (Accurex Plus, Polar Electro Oy, Kempele, Finland). Data were downloaded via an interface unit, which was linked to a PC computer with appropriate software (Polar Electro Oy, Kempele, Finland). Short-range telemetric HR monitors have been shown to be valid and reliable measures of HR (Leger and Thivierge, 1988).

3.1.4 Blood lactate analysis

Measures of [HLa] were performed using an automated lactate analyser (YSI 1500 Sport, YSI Inc., Yellow Springs, OH, USA) which uses immobilised enzyme electrode technology. A thin film of lactate enzyme is immobilised within a membrane. Hydrogen peroxide is produced when the lactate in the injected blood sample diffuses through the membrane. The hydrogen peroxide, measured at a platinum anode, is

proportional to the lactate in the sample. According to the manufacturers, the measurement range of the YSI 1500 Sport is 0 to 30 mmol·l^{-1} with a precision of ± 2% of the reading or 0.1 mmol·l^{-1}, whichever is larger. The recommended calibration point by the manufacturer is 5.0 mmol·l^{-1}.

3.1.4.1 Calibration of the lactate analyser

Prior to each exercise test, the lactate analyser was calibrated with a lactate standard (5 mmol·l^{-1}) supplied by the manufacturer (YSI 2327). Furthermore, the analyser was regularly checked for system linearity with a 2.5 mmol·l^{-1} self-made lactate standard (5 mmol·l^{-1} diluted with an equal amount of purified water) at regular intervals throughout the duration of the test. If an acceptable level of precision was not observed at any stage (lactate reading greater than ± 5%) then re-calibration of the analyser with the 5 mmol·l^{-1} lactate standard was performed. Reproducibility data for the lactate analyser is provided in Appendix 3.

3.1.5 Anthropometry

3.1.5.1 Stature

The stature of participants was measured and recorded to the nearest 0.1 cm using a stadiometer (Holtain Ltd, Crymych, Dyfed). Participants stood in bare feet with feet together against the backboard of the stadiometer. The measuring board was lowered onto the participants head and with the Frankfort plane in a horizontal position the participant took a deep inspiration whilst light traction was applied under the participant's mandible.

3.1.5.2 Body mass

The BM of each participant was measured in kg to the nearest 0.05 kg using a beam scale (Weylux, England). Participants were without footwear and wore shorts.

3.2 Exercise testing protocols

3.2.1 Ethics approval

Prior to each study, ethics approval was sought and granted by the School of Sport and Leisure Management Research Ethics Committee, Sheffield Hallam University, in accordance with the Declaration of Helsinki.

3.2.2 Informed consent

Prior to each individual investigation, participants were given clear and precise written documentation explaining the purpose, procedures and requirements of the study. This information was also communicated verbally to each participant on an individual basis. Information for participants and informed consent forms are included in Appendices 4 and 5 respectively.

3.2.3 Pre-exercise screening

All participants completed a pre-exercise medical questionnaire (Appendix 6) to screen for previous and current medical illnesses or conditions as well as any musculo-skeletal injuries.

3.2.4 Pre-test instructions/requirements

Prior to the completion of any exercise test, participants were requested to: 1) attend the laboratory in a 3-4 hour post-absorptive state; 2) maintain their normal dietary intake; 3) abstain from strenuous exercise in the 48 hours preceding an exercise test and 4) wear suitable clothing for physical activity.

3.2.5 Incremental treadmill protocol

Each participant performed a continuous incremental treadmill protocol to maximum volitional exhaustion to assess, via pulmonary gas-exchange, two parameters of aerobic function - $\dot{V}O_{2\,max}$ and V_T. Prior to the test, each participant was fully accustomed to running on a motorised treadmill ergometer, the pulmonary gas-exchange and HR apparatus. The aim of the protocol was to elicit $\dot{V}O_{2\,max}$ in approximately 8-15 min.

For all treadmill running tests, a gradient of 1% was used to simulate the energy cost of outdoor running (Jones and Doust, 1996). The incremental treadmill protocol for the determination of the V_T and $\dot{V}O_{2\,max}$ began with 3 min of running at 10 $km \cdot h^{-1}$ after which running speed was increased by 1 $km \cdot h^{-1}$ every minute until volitional exhaustion (Figure 3.2). Protocols with a similar size and duration of increments have been previously used to determine $\dot{V}O_{2\,max}$ in endurance trained runners (Noakes *et al.*, 1990; Scott and Houmard, 1994). Strong verbal encouragement was used to motivate the participants during the latter stages of the test. The test ended when the participant was unable to maintain the running speed. After completion of the test each participant completed at least 5 min of low-intensity exercise to cool down.

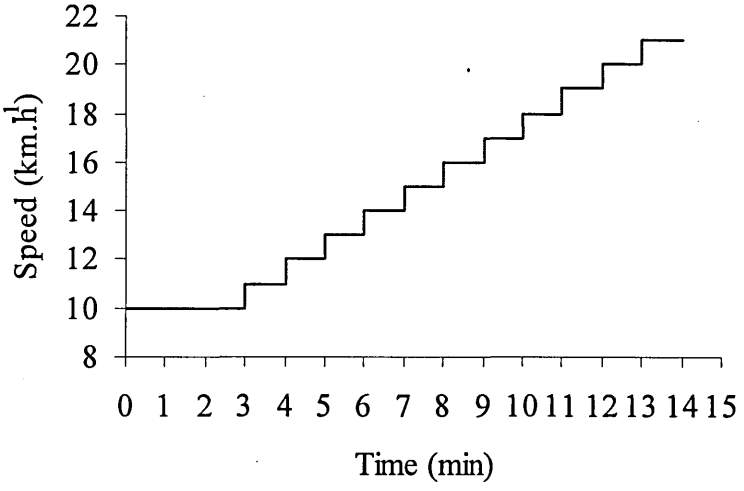


Figure 3.2 Schematic of the incremental protocol to volitional exhaustion for determination of V_T and $\dot{V}O_{2\,max}$.

3.2.5.1 Determination of $\dot{V}O_{2\,max}$

Breath-by-breath data measured throughout the incremental test was averaged on a 30 s basis. The highest $\dot{V}O_2$ attained at the end of the incremental exercise was accepted as $\dot{V}O_{2\,max}$ if a plateau in the $\dot{V}O_2$ - exercise intensity relationship was observed or, in accordance with the British Association of Sport and Exercise Sciences (BASES, 1997), an increase in $\dot{V}O_2$ <2 $ml \cdot kg^{-1} \cdot min^{-1}$ (3%) with a further increase in exercise intensity

was observed. If participants did not reach a plateau in $\dot{V}O_2$ then secondary criteria (BASES, 1997) were used. These were:

- the attainment of volitional exhaustion within 8-15 min
- at the end of the test, the participant must show and report intense effort
- a final respiratory exchange ratio (RER) of 1.15 or above
- a final HR within 10 $b \cdot min^{-1}$ of the maximum heart rate (HR_{max}) predicted from age (where age predicted HR_{max} is calculated as 220 $b \cdot min^{-1}$ minus age in years)
- a post-exercise [HLa] of 8 $mmol \cdot l^{-1}$ or greater.

If participants did not meet three of the secondary criteria the test was repeated.

3.2.5.2 Determination of V_T

The V_T was determined from the breath-by-breath gas exchange data collected during the incremental exercise test to exhaustion. Before any interpretation, breath-by-breath data were minimally smoothed using a three breath moving average to reduce the breath-by-breath fluctuations whilst at the same time retaining the underlying response to incremental increases in exercise intensity. The V_T was identified using the *V*-slope method (Beaver *et al.*, 1986) which involves an analysis of the behaviour of $\dot{V}CO_2$ as a function of $\dot{V}O_2$. Incremental exercise tests gradually exceed the LT and this is accompanied by the buffering of lactic acid by [HCO_3^-] with a consequent increase in CO_2. By constructing a graph of the relationship between $\dot{V}CO_2$ and $\dot{V}O_2$ during incremental exercise (Figure 3.3), the transition in the relationship or 'breakpoint' between $\dot{V}CO_2$ and $\dot{V}O_2$ can be visually and mathematically identified. When the breakpoint was difficult to discern, additional gas exchange variables (ventilatory equivalent method, Whipp *et al.*, 1981) were considered to aid in the identification of the V_T. This involved the construction of individual graphs $\dot{V}E/\dot{V}O_2$, $\dot{V}E/\dot{V}CO_2$, end-tidal PO_2 ($PETO_2$) and end-tidal PCO_2 ($PETCO_2$) against $\dot{V}O_2$ from which the nadir of $\dot{V}E/\dot{V}O_2$ and $PETO_2$ was identified on $\dot{V}E/\dot{V}CO_2$ and $PETCO_2$ relationships. Using these measures, the V_T represented the point at which $\dot{V}E$ increased out of proportion to

the increases in $\dot{V}O_2$ (hyperventilation with respect to O_2). Hyperventilation with respect to O_2, without concomitant hyperventilation for CO_2, only occurs during buffering of a metabolic acid by HCO_3^-. Other forms of hyperventilation should cause $PETO_2$ to increase and $PETCO_2$ to decrease while $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ increase together.

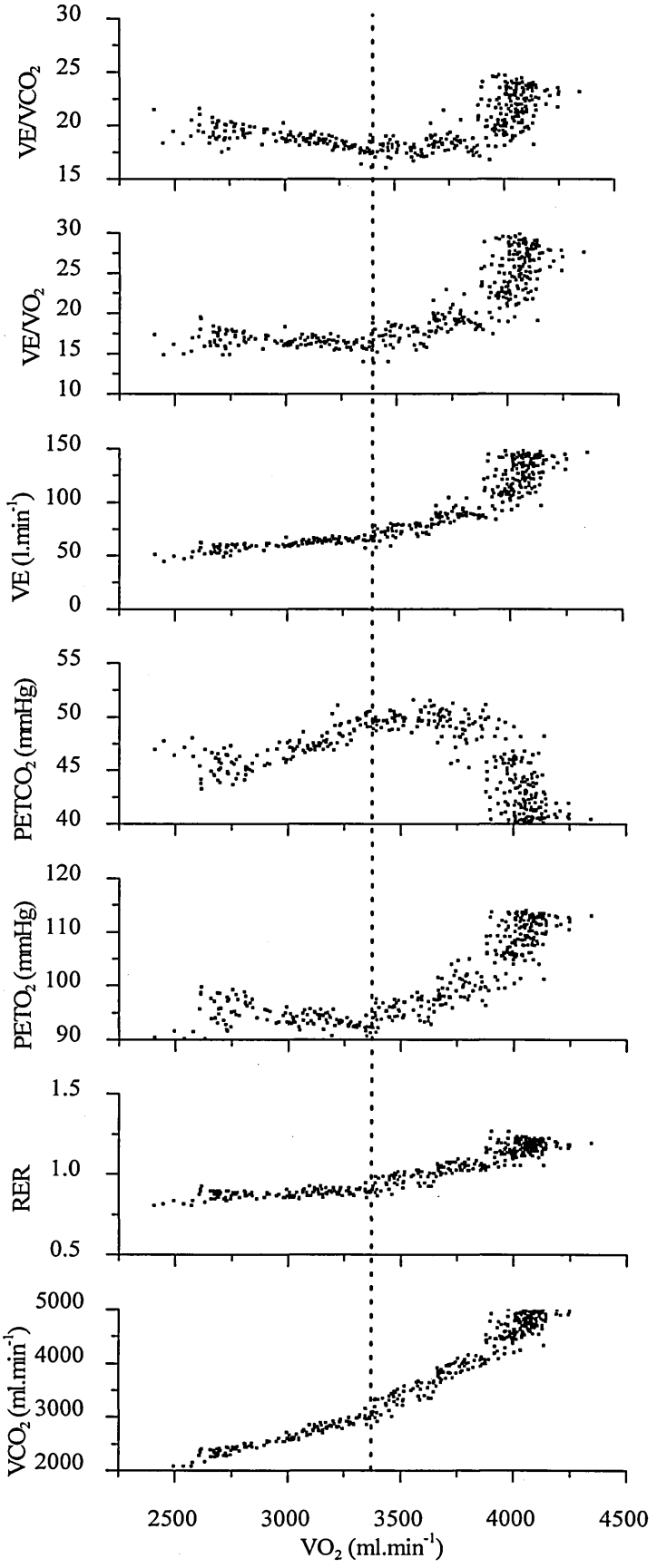


Figure 3.3 The *V*-slope ($\dot{V}O_2$ vs. $\dot{V}CO_2$) and additional gas-exchange measures used to identify the V_T from a typical participants $\dot{V}O_2$ response during incremental exercise to exhaustion. Dashed line represents V_T (3375 ml·min^{-1}).

3.2.6 Protocol for the determination of running economy

Participants completed 5-7, four-min bouts of running at speeds between 10 and 20 $km \cdot h^{-1}$. The most appropriate starting speed for a given individual was chosen according to their running ability and current level of fitness. Each stage was faster than the previously completed stage by 1 $km \cdot h^{-1}$. The treadmill was set at a 1% gradient to simulate the energetic requirement of outdoor running (Jones and Doust, 1996). Throughout the duration of each 4 min stage, $\dot{V}O_2$ was measured on a breath-by-breath basis. At the end of each stage, the participant supported their weight and stood astride of the moving treadmill belt whilst a small blood sample (~25µl) was taken for the immediate analysis of [HLa]. The time taken for the blood sampling procedure was approximately 15 s after which the participant resumed running at the next increased running speed. When [HLa] reached or exceeded 4 $mmol \cdot l^{-1}$ at the end of a stage, the test was ended. The mean HR over the last 30 s of each stage was recorded.

Given the expected disparity in performance capabilities of the athletes under study, attainable running speeds to assess RE varied considerably. Therefore, based on previous research, $\dot{V}O_2$ at 16 $km \cdot h^{-1}$ (Conley and Krahenbuhl, 1980; Daniels and Daniels, 1992; Morgan and Daniels, 1994; Jones, 2002) was used to quantify RE.

3.2.6.1 Allometric scaling for differences in body mass.

In consideration of evidence suggesting that $\dot{V}O_2$ does not increase in proportion to BM during running (Bergh *et al.*, 1991), $\dot{V}O_2$ was also expressed using several power-function ratios that have been suggested in the literature (Schmidt-Nielson, 1984). The sample-specific exponent was also calculated using log-log transformations and analysis of covariance (ANCOVA).

3.2.7 Protocol for the measurement of on- and off-transient $\dot{V}O_2$ kinetics

For assessment of the on- and off-transient $\dot{V}O_2$ kinetics, participants completed a multiple square-wave transition protocol (adapted from Wasserman and Whipp, 1993). This began with 2 min of standing (feet astride the treadmill belt) for the measurement

of resting $\dot{V}O_2$ after which the participants walked for a further four min at 4 km·h^{-1}. At the end of this first 6 min, the treadmill speed was abruptly increased to a speed which would elicit 80% of the individual's $\dot{V}O_2$ at their V_T (80% V_T), and was held for a further 6 min. The speed at 80% V_T was calculated from the linear relationship between running speed and $\dot{V}O_2$ below V_T during the assessment of RE (see Chapter 3, Section 3.2.6). For safety purposes, participants were instructed to be prepared for speed increases and balance themselves using the handrails of the treadmill until their stride rate matched the treadmill belt speed. Once this was achieved participants let go of the handrails and began un-aided running. All individuals were able to respond to the abrupt change in treadmill speed without or with minimal assistance from the handrails. The time taken for the abrupt transition was ~2 s. Similar procedures were adopted by participants at the end of the 6 min running interval when treadmill speed abruptly returned to 4 km·h^{-1} for a further 6 min. This walk-run transition was performed consecutively three times after which participants were given 15 min seated rest before repeating the protocol (Figure 3.4). In total, all participants completed six square-wave transitions of which the total time spent running at 80%V_T was 36 min.

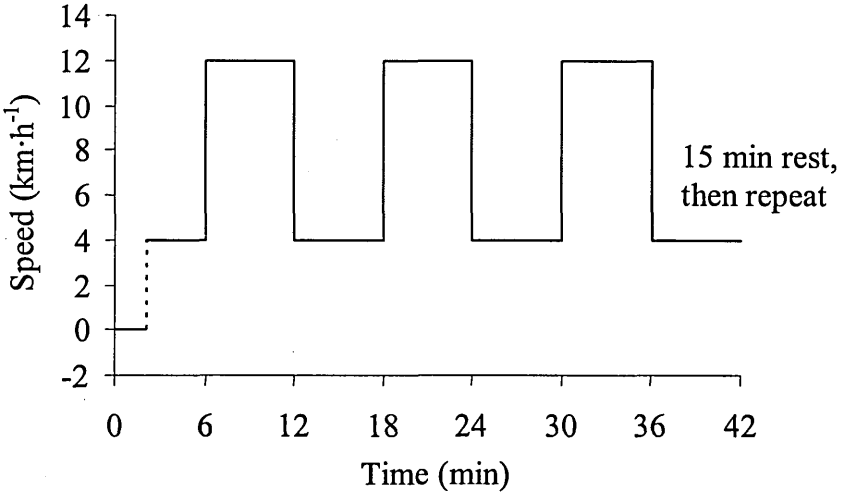


Figure 3.4 Schematic representation of the intermittent square-wave transition protocol for determination of on- and off-transient $\dot{V}O_2$ kinetics.

3.2.7.1 Data analysis of $\dot{V}O_2$ kinetics

Breath-by-breath $\dot{V}O_2$ data were linearly interpolated to yield $\dot{V}O_2$ values for every second during the test. The data from each of the six transitions were then split, time aligned and ensemble averaged to produce a single data set that was representative of the participant's underlying $\dot{V}O_2$ response. To characterise the on-transient $\dot{V}O_2$ kinetics, data were modelled from 20 s post-onset of exercise, thereby excluding the cardiodynamic phase, until end-exercise by non-linear least squares regression to a mono-exponential model incorporating a time delay. The on-transient exponential model was of the form:

$$\dot{V}O_2(t) = \dot{V}O_{2\,(b)} + A_{on}\,(1 - \exp^{-(t-\delta/\tau)}) \quad (8)$$

where t is time, $\dot{V}O_{2\,(b)}$ is baseline $\dot{V}O_2$, A_{on} is the amplitude of $\dot{V}O_2$ above the baseline value, δ_{on} is the on-transient time delay and τ_{on} is the on-transient time constant. The mean response time (MRT_{on}) was calculated as:

$$MRT_{on} = \delta_{on} + \tau_{on} \quad (9)$$

To characterise the off-transient $\dot{V}O_2$ kinetics, the following mono-exponential model (Özyener *et al.*, 2001) was used:

$$\dot{V}O_2(t) = \dot{V}O_{2\,(m)} + A_{off}\,(\exp^{-(t-\delta/\tau)}) \quad (10)$$

where $\dot{V}O_{2\,(m)}$ is moderate-intensity (80%V_T) exercise $\dot{V}O_2$. Other parameters have been previously described in Equation 8. The off-transient mean response time (MRT_{off}) was calculated as:

$$MRT_{off} = \delta_{off} + \tau_{off} \quad (11)$$

3.2.7.2 Calculation of 95% confidence intervals

Estimations of kinetic parameters using exponential modelling techniques can be affected by inherent breath-by-breath variability. For the estimation of τ_{on}, Lamarra *et al.* (1987) proposed two equations to determine the 95% confidence intervals (95%CI) for an individual's $\dot{V}O_2$ kinetic response. The accuracy of the non-linear least squares estimation of τ_{on} is directly proportional to the SD of the noise (S_0). This allows an *a-priori* determination to be made of the number of transitions required to achieve a desired 95%CI in the estimated parameter for a given participant. The 95%CI for τ_{on} was calculated as follows:

$$K_1 = \hat{L}\ \frac{S_0}{\Delta Y_{ss}} \tag{12}$$

where K_1 is the CI, S_0 is the SD of the noise, ΔY_{ss} is the amplitude of $\dot{V}O_2$ above baseline and $\hat{L}$ is a constant, as described in Lamarra *et al.* (1987). For the superposition of n independent transitions, the effective noise variance is reduced by a factor of n if the noise is assumed to be Gaussian and un-correlated between transitions. Hence, the confidence interval (K_n) is reduced by the factor of $\sqrt{n}$. The number of transitions (n) required for a desired 95%CI (K_n) is given by:

$$n = \left[\frac{\hat{L} \cdot S_0}{K_n \cdot \Delta Y_{ss}}\right]^2 \tag{13}$$

3.2.8 Running performance time-trial

To establish a single measure of running performance a 5 km time-trial was performed on a treadmill under controlled laboratory conditions (~20 °C). A self-selected warm-up preceded each time-trial. Following this warm-up, treadmill speed was increased to the participant's requested starting speed and timing initiated. Thereafter, the participant was permitted to verbally request an increase or decrease in treadmill speed throughout the remainder of the trial (Ramsbottom *et al.*, 1992; Scott and Houmard, 1994). Throughout the trial participants received feedback on distance run, running speed and

time elapsed from the treadmill control panel. In addition, split times for each individual km completed and the total distance remaining were verbally communicated to the participant.

Throughout the 5 km time-trial, HR was measured continuously. The HR at the end of each km, mean HR and HR_{max} were recorded. With knowledge of the relationship between $\dot{V}O_2$ and running speed, which was previously obtained for each participant during the RE assessment, it was possible to estimate the $\%\dot{V}O_{2\,max}$ sustained during the 5 km time-trial.

3.3 Statistical analyses

Various statistical tests have been employed within this thesis to determine the reproducibility of measures, identify differences and relationships between physiological measures and performances and predict running performance. These are outlined in detail below. All statistical analyses were performed using commercially available statistical software (SPSS for Windows *v*11.0; SPSS Inc., Chicago, IL, USA).

3.3.1 Limits of agreement

For an appropriate assessment of test-retest reproducibility, Atkinson and Nevill (1998) have provided evidence for and against a variety of statistical tests commonly used to quantify measurement error (reliability). As a result, it has become apparent that the 95% limits of agreement (LOA) first described by Bland and Altman (1986) are a useful method in which to assess reproducibility. Bland and Altman (1986) proposed an approach using simple calculations and graphical techniques using the differences between two measurements (measurement errors). The assumption that underpins the correct use of LOA is that the differences (error) are homoscedastic. That is, the differences are of the same magnitude regardless of the magnitude of the measure. To check for homoscedasticity, Bland and Altman (1986) recommend a scatter diagram (Bland and Altman plot) of the differences between two tests against the grand mean of two tests. If a relationship is visually detected it can be confirmed by calculating the

correlation coefficient between the absolute differences between the two tests and the grand mean. If a significant relationship exists i.e. the errors are heteroscedastic (larger error associated with larger measurement means), it is recommended that the logarithms of each measurement be taken and then the LOA calculations can be performed (Bland and Altman, 1986; Atkinson and Nevill, 1998). There has been strong evidence demonstrating that heteroscedastic errors are the norm in measurements recorded on a ratio scale, such as those typically seen in sports medicine and sport science (Nevill and Atkinson, 1997). However, provided that the previously stated assumption has been checked and the differences are homoscedatisc, the LOA can be calculated, without the need for logarithmic transformations, as:

$$\pm 95\%\ \text{LOA} = 1.96 \times SD_{diff} \tag{14}$$

where SD_{diff} is the SD of the differences between test 1 and test 2. In addition, the LOA in proportion to the grand mean of test 1 and 2 (measurement error) is calculated as:

$$\text{Measurement error (\%)} = \left(\frac{1.96 \times SD_{diff}}{\text{grand mean}}\right) \times 100 \tag{15}$$

where grand mean is (mean of test 1 + mean of test 2)/2. The systematic bias between measures taken on two separate occasions is calculated as: -

$$\text{Systematic bias (\%)} = \left(\frac{\overline{X}_{diff}}{\text{grand mean}}\right) \times 100 \tag{16}$$

where $\overline{X}_{diff}$ is the mean of the differences between test 1 and test 2.

Provided that it was not necessary to take logarithms of the measurements, the calculated values are in the original units of measurement. Therefore, interpretation of the agreement between two repeated measures relies upon a subjective assessment of

the LOA. The LOA can also be expressed in proportion to the grand mean of test 1 and 2. This is termed the measurement error (%).

3.3.2 Coefficient of variation

A more traditional method of establishing reproducibility between repeated measured, commonly adopted in biochemistry measures, was also undertaken to permit a direct comparison with previous studies. The CV (%) was calculated as follows:

$$CV\% = \frac{SD}{\overline{x}} \times 100 \quad (17)$$

where SD is the standard deviation and $\overline{X}$ is the mean.

3.3.3 Method error

The calculation of the CV requires the measurement of many repeated trials within the same day, or over several days. This might not be appropriate for some physiological tests that require maximal effort. Such measures could result in an order effect that might result in an improvement or decrement in physiological performance brought about by a training effect or the effect of fatigue respectively. In such instances, the use of the CV to assess reproducibility is not suitable. However, the use of the 'method error' (ME) to express the reproducibility of two repeated measurements can be used. Dahlberg (1940) demonstrated that the differences between two series of measures would have a mean of zero and that the SD_{diff} would be equal to the SD of an individual (SD_{indiv}) divided by the square root of two (Gore, 2000). In acknowledgement that infinite measures or infinite subjects is impossible, Dahlberg (1940) proposed the use of duplicate measures on a group of subjects to approximate the standard error of a single determination (cf. Gore 2000). This has been termed 'method error' (Thorstensson, 1976) and is calculated as:

$$\text{Method error (ME)} = \frac{SD_{diff}}{\sqrt{2}} \quad (18)$$

Similarly to LOA, ME can be expressed in the units of measurement, or as a test-retest CV as calculated below.

$$\text{Test-retest CV (\%)} = \frac{\text{ME}}{\text{grand mean}} \times 100 \quad (19)$$

3.3.4 *t*-tests

To determine whether two samples means differ from each other *t*-tests were used. More specifically, when there were two experimental conditions and different participants an independent samples *t*-test was used. When there were two experimental conditions and the same participant took part in both conditions a paired samples *t*-test was used. Prior to the calculation of each *t*-test the appropriate assumptions underpinning their use were checked. According to Vincent, (1995), the *t*-test is based on the following assumptions: 1) participants are randomly sampled; 2) data are normally distributed and parametric and 3) there is homogeneity of variance (i.e. variances between groups are equal). The latter can be assessed using Levene's Test for Equality of Variances. Providing the test is non-significant ($P > 0.05$), the variances can be assumed to be homogeneous.

3.3.5 Bi-variate correlations

Correlation is defined as a numerical coefficient that indicates the extent to which two variables are related or associated (Vincent, 1995). This can range from a non (zero) to perfect (1.0) relationship in either a positive or negative direction. To assess the possible relationships and associations between measures in the studies as part of this thesis, the Pearson's product moment correlation coefficient (r) was used. Prior to running this statistical test, a qualitative analysis was undertaken, via construction of a scatter plot of the data, to assess whether there appears to be a relationship between the variables. Furthermore, the assumptions underpinning this test (parametric and normally distributed data) were checked.

3.3.6 Analysis of co-variance

Analysis of co-variance (ANCOVA) is used in sport and exercise science as a method of statistically equating groups on other factors (covariates) that might influence the dependent variable. ANCOVA is based on the assumption that the covariate is related to the dependent variable, and thus they co-vary together (Vincent, 1994). In this study, ANCOVA was used to explore whether runners of different disciplines (MD and LD runners) could be considered as separate groups. Where necessary new groups were devised and adjusted means, based on the slopes and elevations of the linear relationships, were calculated. The assumptions underpinning ANCOVA are: 1) normality of data; 2) a linear relationship between the covariate and the dependent variable and 3) homogeneity of the regression coefficients (the slopes of the linear correlations) between the covariate and the dependent variable.

3.3.7 Multiple regression

Multiple regression is often used in the sport and exercise sciences to demonstrate associations and relationships between variables believed to be related. For example, recognising that success in distance running is most likely multi-factorial, multiple regression analysis has been successfully used to identify physiological measures that are closely related to distance running performance (Powers *et al.*, 1983; Bulbulian *et al.*, 1986).

Multiple regression is underpinned by assumptions that should be met if a valid interpretation of the results is required. These assumptions include 1) a normal distribution of data; 2) linearity; 3) multi-collinearity and singularity and 4) homoscedasticity of errors (Vincent, 1995; Tabachnik and Fidell, 1996). Failure to meet these assumptions could result in a regression model that is not representative of the data.

The multiple regression analyses were carried out using the 'stepwise' method as advocated by Tabachnik and Fidell (1996) after the assumptions underpinning its use were verified. Stepwise multiple regression was performed using the equation of the general form:

$$Y_p = b_1X_1 + b_2X_2 + \ldots\ldots + b_kX_k + C \pm (SE) \quad (20)$$

where b_1, b_2, ..., b_k are coefficients that give weight to the independent variables (X_1, X_2 ..., X_k) according to their relevant contributions to the prediction of *Y* (running performance). The number of independent variables is represented by *k*, *C* is the constant (intercept) and SE is the standard error.

3.3.7.1 Outliers and influential cases

The main purpose of examining residuals in linear or multiple regression analyses is to 1) isolate points for which the regression model fits poorly and 2) isolate points that exert an undue influence on the regression model. To assess the former, the studentized residual, standard residual and deviance statistics are used. To assess the influence of individual cases, statistics such as Cook's and Mahalanobis distances were calculated and considered.

CHAPTER 4

The reproducibility of pulmonary oxygen uptake kinetics in middle- and long-distance runners

4.1 Introduction

Traditionally, physiological assessments of MD and LD runners have involved the measurement of three gas-exchange indices: 1) $\dot{V}O_{2\,max}$; 2) V_T and 3) O_2 cost of running (i.e. RE). The $\dot{V}O_2$ kinetic response at the onset of moderate-intensity exercise (i.e. below the V_T) however, is also an important index of aerobic function (Whipp *et al.*, 1981) and reflects the integrative performance of the systems involved in O_2 transport from the atmosphere to the cell (Wasserman *et al.*, 1994).

The reproducibility of $\dot{V}O_{2\,max}$ (Weltman *et al.*, 1990), LT and V_T (Aunola and Rusko, 1984; Weltman *et al.*, 1990; McLellan and Jacobs, 1993) and RE (Armstrong and Costill, 1985, Morgan *et al.*, 1991; Pereira and Freedson, 1997) in endurance runners has previously been established. The reproducibility of $\dot{V}O_2$ kinetic parameters is less well documented in the literature and has not been established in endurance-trained runners. Establishing and quantifying the level of reproducibility for any physiological measure used to assess endurance runners is of paramount importance, since reproducibility will influence the interpretation of the results and worthiness of the findings.

4.1.1 Reproducibility of $\dot{V}O_2$ kinetic parameters

Considering the extensive research into $\dot{V}O_2$ kinetics in athletic and non-athletic populations, it is surprising that relatively few studies have quantified the reproducibility of $\dot{V}O_2$ kinetic parameter estimations at the onset (Berry and Moritani, 1985; Kilding *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) and recovery (Berg, 1947; Özyener *et al.*, 2001) from moderate-intensity exercise. Furthermore, few studies have approached the assessment of reproducibility using a test-retest research design. This would involve measuring $\dot{V}O_2$ kinetics twice on

separate days. This approach would permit the determination of the test-retest reproducibility and might also establish biological day-to-day variability.

With respect to on-transient $\dot{V}O_2$ kinetics, Berry and Moritani (1985) reported the test-retest reproducibility of their measures of the time course of $\dot{V}O_2$ during a single square-wave transition from rest to exercise (cycle ergometry) at 150 W. These measures of $\dot{V}O_2$ kinetics were made two weeks apart, prior to an investigation into the effects of different training intensities on $\dot{V}O_2$ kinetics. A correlation coefficient of 0.87 (P <0.01) and a mean difference of 0.73 s between repeated tests suggested that the level of reproducibility was satisfactory. More recently, the test-retest reproducibility of measures of $\dot{V}O_2$ kinetics was also investigated using cycle ergometry (Kilding *et al.*, 2001). Repeat tests were completed seven days apart and at the same time of day to minimise circadian and other similar influences. Each test consisted of three transitions to 80%V_T to ensure that exercise was below the V_T. Although paired t-tests revealed no significant differences for each kinetic parameter (δ, τ, MRT and A) between tests 1 and 2, wide 95% LOA and large measurement errors suggested substantial intra-participant variability between repeated tests. These findings would suggest that either three transitions are inadequate to reduce the effect of noise and so allow a reproducible determination of $\dot{V}O_2$ kinetic parameter estimations, or that biological day-to-day variability in $\dot{V}O_2$ kinetics is large. Of the time-related kinetic parameters, the MRT was the most reproducible and might be more appropriate to use when the amplitude of $\dot{V}O_2$ is low or there is a substantial amount of breath-by-breath noise.

The reproducibility of on-transient $\dot{V}O_2$ kinetic responses has also been established in individuals diagnosed with chronic obstructive pulmonary disease (COPD, Puente-Maestu *et al.*, 2001). In this study, patients completed two square-wave transitions, separated by 2 hours, from rest to 80% of the estimated LT or 50% of $\dot{V}O_{2\,peak}$ if LT was insufficiently differentiable. There was no systematic variation between repeated measures as identified by paired t-tests (P >0.05). Agreement between tests was further evaluated by intra-class correlation coefficients. The correlation coefficients were

consistently higher than 0.97, reflecting that individual differences were small and that reproducible estimates of τ and amplitude of $\dot{V}O_2$ (*A*) can be obtained in COPD patients providing that the data set provides a sufficiently large *A* and there is low breath-by-breath variability.

Using a single-subject design, Demarle *et al.* (2001) determined the reproducibility of repeated kinetic parameter estimations, established from three single transitions at the speed of the LT. The CV for δ, τ and amplitude were 6.8, 5.5, and 0.8% respectively, indicating very good reproducibility. However, these findings are difficult to interpret, since no indication of time between repeated tests was provided in their manuscript.

An early investigation by Berg (1947) was the first to report the reproducibility of $\dot{V}O_2$ kinetic parameter estimations. However, as opposed to the aforementioned studies, these were focussed exclusively on the off-transient recovery $\dot{V}O_2$ kinetics after moderate -intensity exercise. Repeated tests were carried out at the same time of day on consecutive days. The test-retest correlation *r* was 0.55 and the standard error of the measurement was ± 4.5 s (± 15% of the mean).

More recently, Özyener *et al.* (2001) investigated the reproducibility of on- and off-transient $\dot{V}O_2$ kinetics. The approach used by these authors assessed the variability between three estimations of τ established from single transitions. The findings suggested that the degree of reproducibility of τ during the on- and off-transient was not influenced by the intensity of exercise since there were no significant differences between the variations in τ over a range of exercise intensities (moderate, heavy, very heavy and severe). Subsequently, values for τ from each individual transition at each exercise intensity were grouped together and treated as one large population (n = 72). The individual values for τ for each of the three different determinations typically varied by up to 10%. The SD of the amplitude of $\dot{V}O_2$ and τ was 90 ml·min^{-1} and 6.2 s for the on-transient and 60 ml·min^{-1} and 4.0 s for the off-transient respectively. It was acknowledged that 'noise' associated within a single transition could readily account for

the 10% variation in the individual estimates of τ, although the possibility of 'real' variation on different occasions could not be excluded.

4.1.2 Effects of noise on kinetic parameter estimation and reproducibility

The accuracy with which $\dot{V}O_2$ kinetic parameters can be reproducibly determined is important, since kinetic parameter estimations are highly influenced by the effects of inherent breath-by-breath variability (Lamarra *et al.*, 1987). Methods, such as repeated transitions, to attenuate the effects of noise have been used to improve estimations of kinetic parameters (Linnarsson, 1974; Lamarra *et al.*, 1987). According to Lamarra *et al.* (1987), the effects of noise on kinetic parameter estimations can be reduced by the square root of the number of transitions performed. The effects of noise are highly dependent on the amplitude of $\dot{V}O_2$ above the baseline value and the SD of the breath-by-breath variability (SD_{noise}), both of which vary amongst individuals. Therefore, the number of transitions (n) required for an accurate determination of τ is specific to an individual and cannot be generalised to the entire population. Lamarra *et al.* (1987) proposed two equations that could be used to 1) calculate the number of transitions required for a given individual and 2) determine the 95%CI when n transitions have been performed by all participants in a study. The latter *post-hoc* approach is most commonly used (Rossiter *et al.*, 1999; Bearden and Moffatt, 2001) and gives an indication of the accuracy with which τ has been determined.

4.1.3 Participant characteristics and reproducibility

The majority of studies that have considered the reproducibility of $\dot{V}O_2$ kinetic parameters have been conducted on healthy untrained individuals (Whipp *et al.*, 1981; Berry and Moritani, 1985; Hughson and Inman, 1986; Edwards *et al.*, 2001; Kilding *et al.*, 2001; Özyener *et al.*, 2001). Recently, the reproducibility of $\dot{V}O_2$ kinetic parameters obtained from patients with COPD (Puente-Maestu *et al.*, 2001) and a single endurance trained runner (Demarle *et al.*, 2001) has also been documented. However, no study has reported the reproducibility of $\dot{V}O_2$ kinetic measures in a group of

endurance trained individuals. Furthermore, it is not known whether endurance training affects the day-to-day variability of $\dot{V}O_2$ kinetics.

4.1.4 Mode of ergometry and reproducibility

The use of cycle ergometry has been the primary method to assess $\dot{V}O_2$ kinetics during the on- (Berry and Moritani, 1985; Kilding *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) and off-transients (Özyener *et al.*, 2001) using square-wave or step transitions in exercise intensity. To date no study has determined the reproducibility of kinetic parameters using treadmill ergometry, although the reproducibility of kinetic parameters for a single endurance runner whilst track running has been quantified (Demarle *et al.*, 2001). Ideally, the mode of ergometry used to assess the physiological status of athletes should closely simulate the sport in which they regular participate. This is commonly referred to as the 'principle of specificity'. Despite that previous research has found no significant differences in τ between moderate-intensity cycle and treadmill exercise (Chilibeck *et al.*, 1996; Carter *et al.*, 2000a), it might be considered inappropriate to assess $\dot{V}O_2$ kinetics in athletes using a mode of ergometry to which they are unaccustomed.

4.1.5 Statistical approaches to assess the reproducibility of $\dot{V}O_2$ kinetics

There are many ways in which reproducibility can be expressed in sport and exercise science and attempts to standardise its reporting remains controversial (Atkinson and Nevill, 1998; Hopkins, 2000). Specifically, various methods have been used to assess the reproducibility of kinetic parameter estimations. This has included paired *t*-tests (Kilding *et al.*, 2001), correlation coefficients (Berg, 1947; Whipp *et al.*, 1981; Berry and Moritani, 1985; Puente-Maestu *et al.*, 2001), CV (Hughson and Inman, 1986; Demarle *et al.*, 2001) and 95% LOA (Edwards *et al.*, 2001; Kilding *et al.*, 2001). Each of these methods have been examined with respect to their appropriateness for the assessment of test-retest reliability or measurement error (Atkinson and Nevill, 1998). As a result, it has been recommended that researchers should cite and interpret a number

of statistical methods for assessing reliability and furthermore, the inclusion of the 95% LOA method is particularly encouraged (Atkinson and Nevill, 1998).

4.1.6 Aim of study

The aim of this study was to establish and quantify the reproducibility of $\dot{V}O_2$ kinetics during the on- and off-transient, at the onset and recovery from moderate-intensity treadmill exercise.

4.2 Participants and methods

4.2.1 Participants

Twelve male MD and LD runners provided informed consent and took part in this study. Participants' anthropometric and physiological characteristics are presented in Table 4.1. Ethics approval was obtained from the School of Sport and Leisure Management Research Ethics Committee, Sheffield Hallam University. Prior to participation in the study, each athlete completed a medical screening questionnaire (Appendix 6).

Table 4.1 Participants anthropometric and physiological characteristics (n=12). Values are mean ± SD.

Age (years)	Stature (cm)	BM (kg)	$\dot{V}O_{2\,max}$ ($ml \cdot min^{-1}$)	$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	$\dot{V}O_2$ at V_T ($ml \cdot min^{-1}$)
25.2	179.5	70.1	4138	59.2	240	3429
± 4.7	± 7.5	± 9.7	± 625	± 5.5	± 23	± 389

4.2.2 Experimental design

Participants visited the laboratory for physiological testing on three occasions within a seven-day period. Each test was separated by at least 48 hours and was performed at approximately the same time of day. The first visit to the laboratory involved an incremental exercise test to volitional exhaustion for the determination of V_T and $\dot{V}O_{2\,max}$. The second and third visits (tests 2 and 3) involved a square-wave protocol to determine $\dot{V}O_2$ kinetics at the onset and recovery from moderate-intensity exercise. Throughout the duration of the testing period, participants were requested to maintain their usual dietary intake, abstain from participating in heavy training between each test and consuming alcohol and caffeine in the 24 hours preceding each test.

4.2.3 Experimental protocols

All tests were performed on a motor driven treadmill (Saturn 250-75R, HP Cosmos, Germany). Each participant completed an incremental exercise test to exhaustion for the determination of the V_T and $\dot{V}O_{2\,max}$ (see Chapter 3, Section 3.2.5). To assess the reproducibility of $\dot{V}O_2$ kinetics, participants completed a square-wave protocol (see Chapter 3, Section 3.2.7) on two occasions. Briefly, this consisted of six transitions from walking at 4 $km \cdot h^{-1}$ to running at speed requiring 80% of the $\dot{V}O_2$ at V_T (80%V_T). Pulmonary gas-exchange was measured breath-by-breath and HR was measured continuously. The [HLa] measures were taken before the start of the square-wave protocol and immediately after the final bout of running at 80%V_T.

4.2.4 Data analysis

Breath-by-breath data obtained during the incremental exercise test to exhaustion and the square-wave protocol were analysed in accordance with procedures outlined in Chapter 3, Sections 3.2.5.1 and 3.2.7.1 respectively.

4.2.5 Statistical analyses

Descriptive statistics (mean ± SD) were calculated for each measure during tests 1 and 2. To establish differences between repeated measures, a paired *t*-test was used. To assess the test-retest reproducibility, the 95% LOA and method error techniques were used (see Chapter 3, Section 3.3.1 and 3.3.3). Prior to these analyses, appropriate checks were made to ensure that the assumptions underpinning each test were met. To allow comparisons with previous research, the traditional CV was calculated for each repeated kinetic parameter.

4.3 Results

The mean (± SD) $\dot{V}O_{2\,max}$ was 59.2 ± 5.5 ml·kg^{-1}·min^{-1}, or expressed as 0.67 power-function ratio of BM was 240 ± 23 ml·kg$^{-0.67}$·min^{-1}. The occurrence of the V_T as a percentage of $\dot{V}O_{2\,max}$ (%$\dot{V}O_{2\,max}$) was 83.3 ± 4.7%. The appropriate speed to elicit 80%V_T during tests 1 and 2 was determined *a-priori* from the $\dot{V}O_2$- running speed relationship below the V_T. This speed was 11.7 ± 0.8 km·h^{-1}. As determined from the mono-exponential model fit, this running speed resulted in a $\dot{V}O_2$ of 2741 ± 318 and 2722 ± 260 ml·min^{-1} during tests 1 and 2 respectively. This was equivalent to 80.1 ± 3.1% V_T (66.8 ± 4.7%$\dot{V}O_{2\,max}$) during test 1 and 79.6 ± 3.7%V_T (66.3 ± 5.1%$\dot{V}O_{2\,max}$) during test 2. This confirms the accuracy of the methods used. This moderate-intensity exercise incurred no significant increase in [HLa] above the resting level (Table 4.2). The physiological measures and the $\dot{V}O_2$ kinetic parameters obtained during the on-transient from tests 1 and 2 are presented in Table 4.2.

Table 4.2 On-transient $\dot{V}O_2$ kinetic parameters during moderate-intensity exercise for tests 1 and 2. Values are mean ± SD.

Measure	Test 1	Test 2
Running speed (km·h^{-1})	11.7 ± 0.8	11.7 ± 0.8
$\dot{V}O_{2\,(b)}$ (ml·min^{-1})	925 ± 98	928 ± 86
A_{on} (ml·min^{-1})	1822 ± 228	1794 ± 186
$\dot{V}O_{2\,(m)}$ (ml·min^{-1})	2747 ± 318	2722 ± 260
δ_{on} (s)	14.7 ± 1.4	14.7 ± 1.6
τ_{on} (s)	12.4 ± 1.9	12.3 ± 2.3
MRT_{on} (s)	27.1 ± 1.8	26.9 ± 1.9
Gain (ml·kg^{-1}·km^{-1})	204 ± 9	201 ± 14
$HR_{(b)}$ (b·min^{-1})	74 ± 9	73 ± 10
$HR_{(m)}$ (b·min^{-1})	137 ± 9	136 ± 9
Δ[HLa] (mmol·l^{-1})	0.0 ± 0.2	0.0 ± 0.1

The calculated 95% CI, using the equations of Lamarra *et al.* (1987), for the estimation of τ_{on} were 0.9 ± 0.2 s (test 1) and 1.0 ± 0.3 s (test 2). In proportion to the mean τ for tests 1 and 2, this equated to 7.3 and 8.1% respectively. This suggests that the magnitude of the effects of noise on kinetic parameter estimations were similar during both tests. Furthermore, the SD_{noise} as a proportion of A_{on} was similar during tests 1 (6.2 ± 1.4%) and 2 (6.7 ± 1.8%).

4.3.1 On-transient kinetics

A typical on-transient $\dot{V}O_2$ response for a representative participant (subject 5) during tests 1 and 2 can be seen in Figure 4.1. All participants displayed the expected characteristic three-phase response during the transition to moderate-intensity exercise. The $\dot{V}O_2$ during the on-transient was consistently found to be well-modelled with a first-order mono-exponential model with time-delay.

The assumption of normality was met for each parameter as identified by a non-significant (P >0.05) Shapiro-Wilks value (Appendix 7). Mean (±SD) values for the estimates of on-transient $\dot{V}O_2$ kinetic parameters obtained from the mono-exponential modelling are presented in Table 4.2. Mean values for A_{on}, δ_{on}, τ_{on}, and MRT_{on} obtained during test 1 did not differ from those obtained in test 2 (Appendix 7).

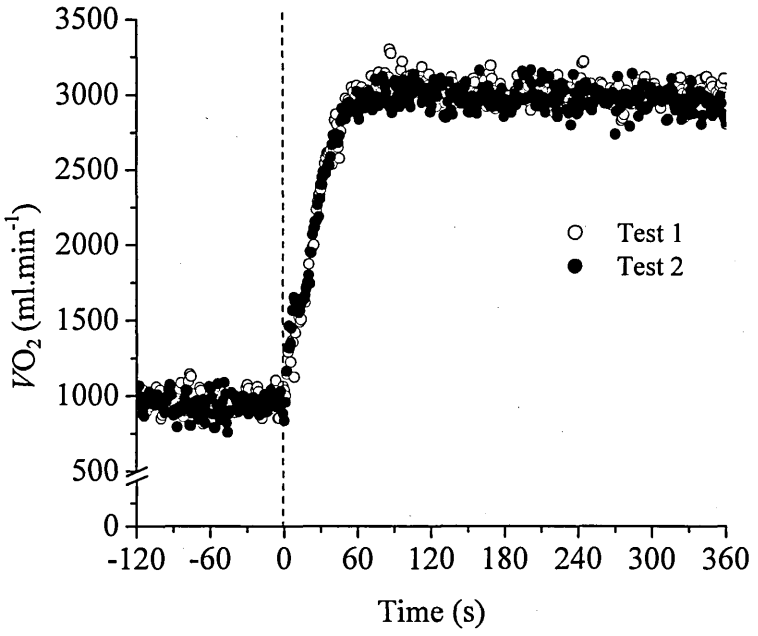


Figure 4.1 A representative participants (subject 11) on-transient $\dot{V}O_2$ response during test 1 (A_{on} = 2055 ml·min^{-1}; δ_{on} = 14.1 s; τ_{on} = 13.2 s; MRT_{on} = 27.3 s) and test 2 (A_{on} = 2036 ml·min^{-1}; δ_{on} = 13.5 s; τ_{on} = 13.3 s; MRT_{on} = 26.8 s).

The assumptions underpinning the correct use of the 95% LOA (see Chapter 3, Section 3.3.1) were met. More specifically, the observed differences between tests 1 and 2 for both the on- and off-transients were normally distributed (Appendix 7). A qualitative assessment of the graphical representations (Bland-Altman plots) of the test 1-test 2 differences vs. the grand mean of test 1 and 2 (Figures 4.2 to 4.5) was made to assess whether the dispersion of the test 1-test 2 differences was influenced by the magnitude of each kinetic parameter (A_{on}, δ_{on}, τ_{on} and MRT_{on}) during the on-transient. These plots suggest that the differences between test 1 and 2 are homoscedastic (i.e. there is no relationship between the differences between test 1 and 2 and the grand mean for test 1 and 2). This was quantitatively confirmed by a non-significant correlation between the absolute test 1-test 2 differences and the grand mean of test 1 and 2 for each on-transient kinetic parameter (Appendix 7).

The SD_{diff} in proportion to the grand mean of test 1 and 2 resulted in a measurement error of 15.1% for τ_{on}. The measurement error for MRT_{on} (4.3%) was considerably lower than that of the other kinetic parameters (Table 4.2). In addition, there was a

small but non-significant negative systematic bias for A_{on}, τ_{on} and MRT_{on} (Table 4.3) indicating that kinetic parameter estimations were less in test 2 than those for test 1. Specifically, the δ, τ and MRT all displayed low method errors, ranging from 0.4 to 0.7 s. Expressed as a test-retest CV, the calculated method error ranged from 1.6 to 5.5%. Of all parameters, the MRT_{on} had the lowest method error. Similar findings were also observed for the CV of repeated measures (Table 4.3).

Table 4.3 The 95% LOA, method error and CV for on-transient $\dot{V}O_2$ kinetic parameters obtained form the mono-exponential modelling for tests 1 and 2.

Measure	Mean of test 1 and 2	Mean ± SD difference	95% LOA	Measurement error (%)	Systematic bias (%)	Method error	Method error (%)	CV (%)
$\dot{V}O_{2\,(b)}$ ($ml \cdot min^{-1}$)	927	4 ± 29	-54 to 61	6.2	0.4	20.7	2.2	1.8
A_{on} ($ml \cdot min^{-1}$)	1808	-29 ± 104	-233 to 175	11.3	-1.6	73.7	4.1	3.4
$\dot{V}O_{2\,(m)}$ ($ml \cdot min^{-1}$)	2735	-25 ± 121	-263 to 213	8.7	-0.9	85.8	3.1	2.7
δ_{on} (s)	14.7	0.0 ± 1.0	-1.9 to 1.9	13.0	0.0	0.7	4.7	4.3
τ_{on} (s)	12.3	-0.1 ± 1.0	-2.0 to 1.8	15.1	-0.9	0.7	5.5	4.8
MRT_{on} (s)	27.0	-0.1 ± 0.6	-1.3 to 1.1	4.3	-0.4	0.4	1.6	1.3

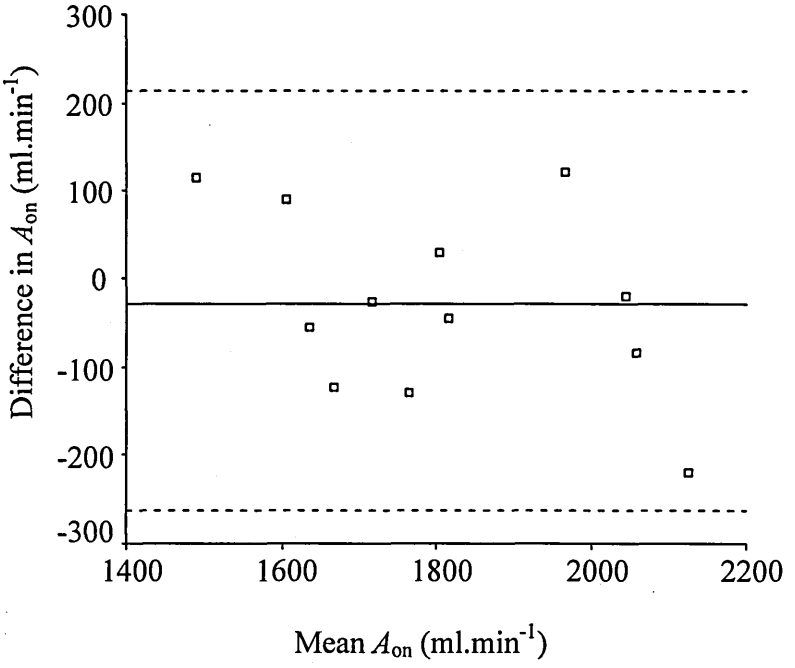


Figure 4.2 The differences (errors) between the estimated A_{on} from tests 1 and 2, plotted against the participants' mean A_{on} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

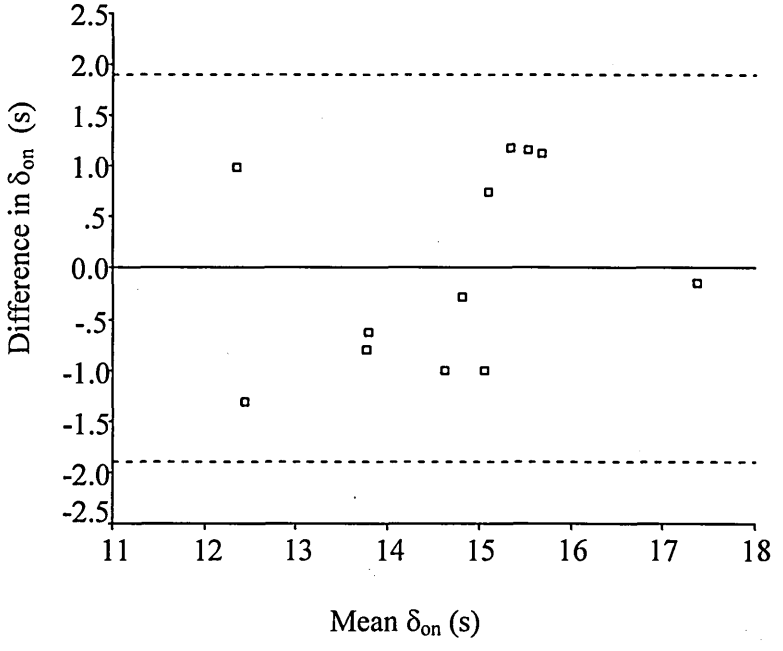


Figure 4.3 The differences (errors) between the estimated δ_{on} from tests 1 and 2, plotted against the participants' mean δ_{on} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

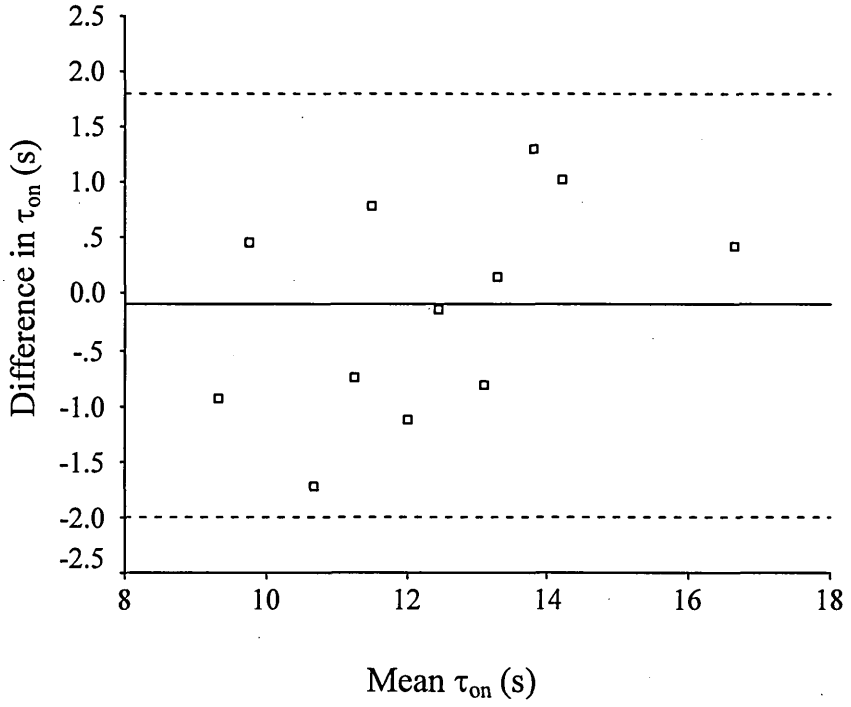


Figure 4.4 The differences (errors) between the estimated τ_{on} from tests 1 and 2, plotted against the participants' mean τ_{on} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

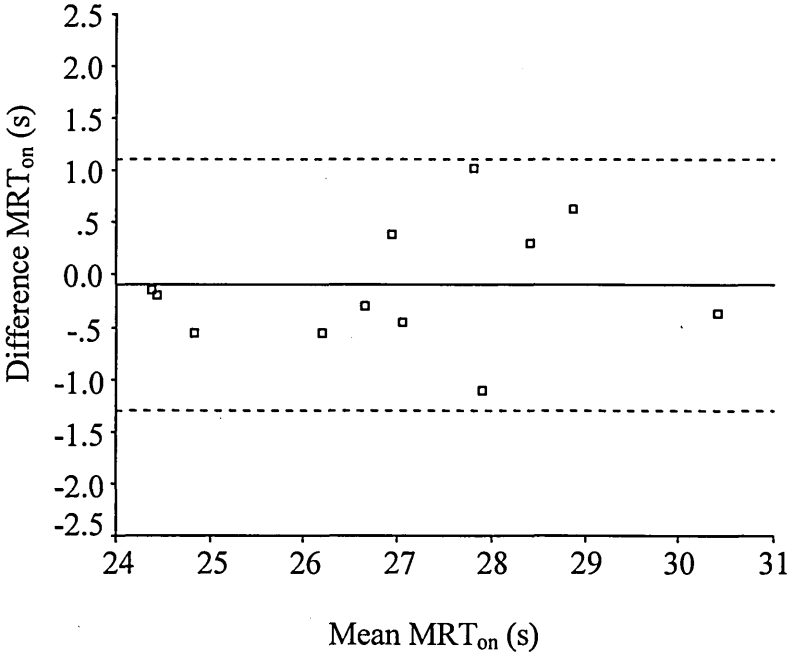


Figure 4.5 The differences (errors) between the estimated MRT_{on} from tests 1 and 2, plotted against the participants' mean MRT_{on} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

4.3.2 Off-transient kinetics

A typical participants off-transient $\dot{V}O_2$ kinetic response during tests 1 and 2 is illustrated in Figure 4.6. Similarly to the observed $\dot{V}O_2$ response during the on-transient, data was equally well-modelled during the off-transient using a first-order mono-exponential model with time-delay. It was consistently observed that participants' $\dot{V}O_2$ returned to its baseline during recovery. To check that this actually occurred, transitions 1 - 3 and 4 - 6 were split and analysed individually. Specifically, this analysis consisted of determining the actual mean $\dot{V}O_2$ in the 2 min preceding each bout of moderate-intensity exercise. The CV for this $\dot{V}O_2$ was 0.85%. A one-way, repeated measures ANOVA (Appendix 7.10) showed that there was no difference between $\dot{V}O_2$ before transition one (pre-exercise) and any other transition ($P = 0.454$). This suggests that $\dot{V}O_2$ had returned to its pre-exercise baseline condition after each bout of moderate-intensity exercise. This would also suggest that the asymptotic value reflected the steady-state $\dot{V}O_2$ and was attained within 6 min. Furthermore, the mean A_{on} and A_{off} were not significantly different ($P = 0.605$). Collectively, this suggests that 6 min was sufficient to permit full recovery between each bout of moderate-intensity exercise and that each transition was initiated from similar baseline conditions.

The mean (± SD) values for tests 1 and 2 are presented in Table 4.4. Paired *t*-tests revealed no differences between test 1 and 2 for any off-transient kinetic parameters ($P > 0.05$, see Appendix 7).

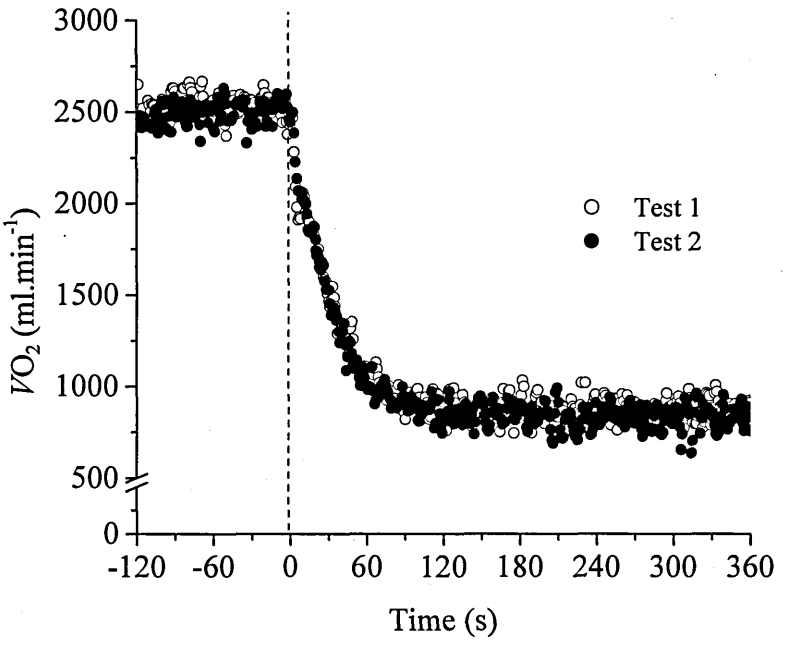


Figure 4.6 A representative participants (subject 10) off-transient $\dot{V}O_2$ response during test 1 (A_{off} = 1661 ml·min^{-1}; δ_{off} = 5.2 s; τ_{off} = 26.0 s; MRT_{off} = 31.2 s) and test 2 (A_{off} = 1664 ml·min^{-1}; δ_{off} = 5.8 s; τ_{off} = 25.9 s; MRT_{off} = 31.7 s).

Table 4.4 Off-transient $\dot{V}O_2$ kinetic parameters at the cessation of moderate-intensity exercise during tests 1 and 2. Values are means ± SD.

Measure	Test 1	Test 2
$\dot{V}O_{2\,(m)}$ (ml·min^{-1})	2742 ± 316	2720 ± 262
A_{off} (ml·min^{-1})	1816 ± 222	1794 ± 181
$\dot{V}O_{2\,(b)}$ (ml·min^{-1})	926 ± 102	926 ± 93
δ_{off} (s)	8.9 ± 3.0	9.2 ± 2.4
τ_{off} (s)	24.5 ± 2.3	24.1 ± 2.4
MRT_{off} (s)	33.4 ± 2.2	33.4 ± 2.6

In proportion to the grand mean of test 1 and 2, the measurement error for τ_{off} was 9.6% and was identical to that of MRT_{off}. A small negative systematic bias for A_{off} and τ_{off} (Table 4.5) was evident indicating that kinetic parameter estimations had a tendency to be lower in test 2. This difference was not significant.

Table 4.5 The 95% LOA, method error and CV for off-transient $\dot{V}O_2$ kinetic parameters obtained from the mono-exponential modelling for tests 1 and 2.

Measure	Mean of test 1 and 2	Mean ± SD difference	95% LOA	Measurement error (%)	Systematic bias (%)	Method error	Method error (%)	CV (%)
$\dot{V}O_{2\,(m)}$ ($ml \cdot min^{-1}$)	2731	-219 ± 122	-260 to 216	8.7	-0.8	85.9	3.1	2.7
A_{off} ($ml \cdot min^{-1}$)	1805	-22 ± 101	-220 to 176	11.0	-1.2	71.4	4.0	3.1
$\dot{V}O_{2\,(b)}$ ($ml \cdot min^{-1}$)	926	0 ± 33	-65 to 65	7.0	0.0	23.3	2.5	2.2
δ_{off} (s)	9.1	0.3 ± 1.5	-2.7 to 3.3	33.0	3.5	1.1	11.9	13.4
τ_{off} (s)	24.3	-0.3 ± 1.2	-2.7 to 2.0	9.6	-1.4	0.8	3.5	2.9
MRT_{off} (s)	33.4	-0.0 ± 1.6	-3.2 to 3.2	9.6	-0.1	1.2	3.5	2.7

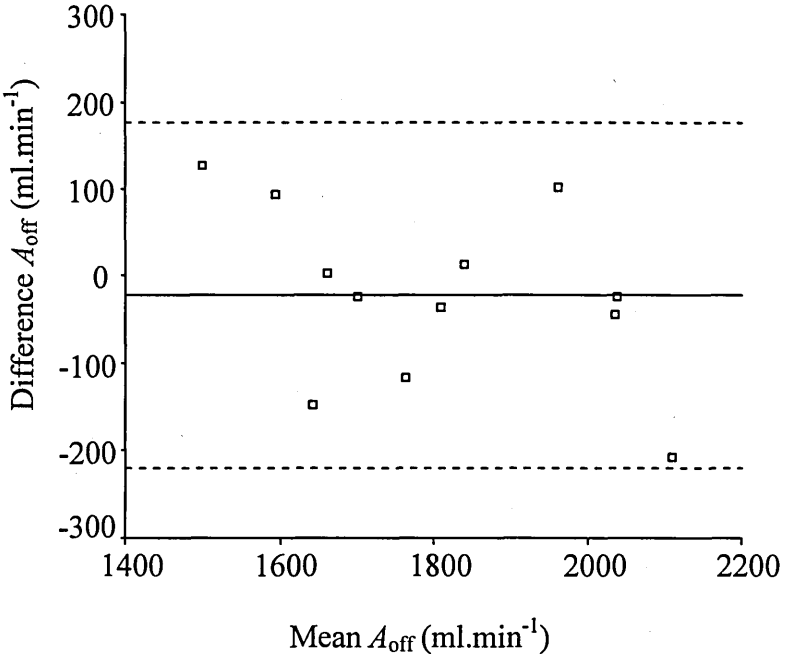


Figure 4.7 The differences (errors) between the estimated A_{off} from tests 1 and 2, plotted against the participants' mean A_{off} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

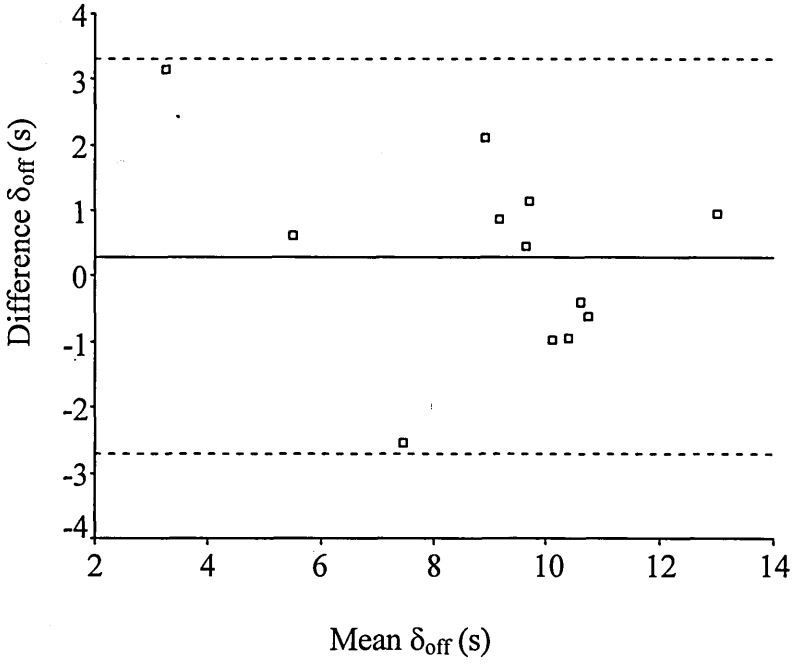


Figure 4.8 The differences (errors) between the estimated δ_{off} from tests 1 and 2, plotted against the participants' mean δ_{off} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

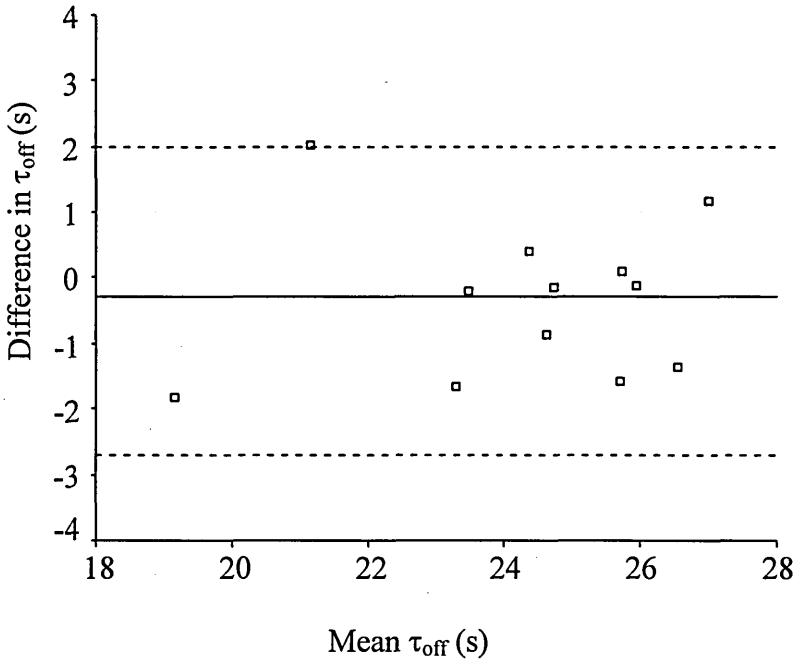


Figure 4.9 The differences (errors) between the estimated τ_{off} from tests 1 and 2, plotted against the participants' mean τ_{off} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

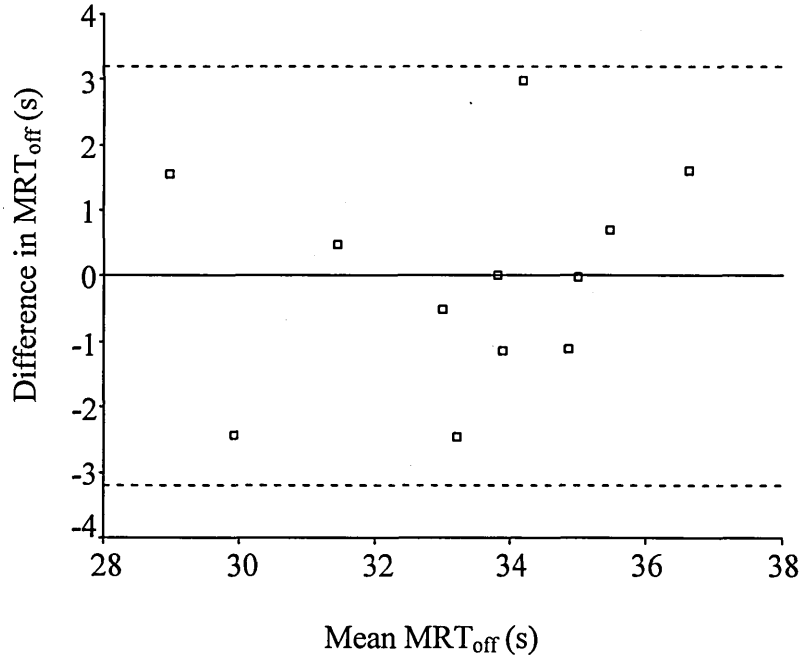


Figure 4.10 The differences (errors) between the estimated MRT_{off} from tests 1 and 2, plotted against the participants' mean MRT_{off} (combined mean of test 1 and 2 for each participant). The bias line (solid) and 95% LOA (dashed) are also presented.

The method error was lower for τ_{off} than the τ_{on}. However, both the δ_{off} and MRT_{off} displayed lower reproducibility (Table 4.5). The least reproducible kinetic parameter was δ_{off} which displayed a method error of 1.1 s, which when expressed as test-retest percentage was 11.1%. A CV of 13.4% confirmed this poor reproducibility. Conversely, $\dot{V}O_{2\,(m)}$, A_{off} and $\dot{V}O_{2\,(b)}$ all displayed similar reproducibility as suggested by a lower method error % (range 2.5 to 4.0%, Table 4.5).

4.4 Discussion

The aim of this study was to assess the reproducibility of measures of $\dot{V}O_2$ kinetic parameters during the on- and off-transients using treadmill ergometry.

4.4.1 Comparisons with previous literature

Since there is a paucity of research reporting the $\dot{V}O_2$ kinetic responses of MD and LD runners during treadmill running, comparisons between studies is difficult. However, a limited number of studies have reported the $\dot{V}O_2$ kinetics during treadmill running in young healthy untrained (Chilibeck *et al.*, 1996) and recreationally active individuals (Carter *et al.*, 2000a, b; Williams *et al.*, 2001). The $\dot{V}O_2$ kinetics during the on-transient from these studies compare relatively well with those of the present study values for treadmill running. The mean (±SD) τ_{on} for endurance trained runners in the present study was 12.4 ± 1.9 and 12.3 ± 2.3 s for test 1 and 2 respectively, compared to 15.0 ± 2.0 (Carter *et al.*, 2000a) and 14.7 ± 2.8 s (Williams *et al.*, 2001) for recreationally active participants. Similarly, Carter *et al.* (2000b) reported a slightly faster τ_{on} of 13.9 ± 1.4 s in recreationally active participants after six weeks of endurance training.

With respect to the off-transient $\dot{V}O_2$ kinetics during recovery from moderate-intensity treadmill running, the τ_{off} in the present study was 24.5 ± 2.3 and 24.1 ± 2.4 s for tests 1 and 2 respectively. These values are much shorter than those reported by Carter *et al.* (2000a) where the mean τ_{off} was 39.3 ± 3.0 s. Overall, the values for both τ_{on} and τ_{off} were lower than those previously reported and are indicative of faster kinetics at the onset of and recovery from moderate-intensity exercise. This was expected and is primarily due to the higher aerobic fitness levels of participants in the present study.

4.4.2 Reproducibility of on-transient $\dot{V}O_2$ kinetics

In the present study, the reproducibly of on-transient $\dot{V}O_2$ kinetic parameters have been established using treadmill ergometry. These can be compared to previous

investigations that have assessed reproducibility between repeated measures of $\dot{V}O_2$ kinetics using alternative modes of exercise including cycle ergometry (Berry and Moritani, 1985; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) and track running (Demarle *et al.*, 2001).

In this study, the intra-participant variability for kinetic parameters in terms of the 95% LOA (Table 4.2) were relatively narrow. These 95% LOA were an improvement compared to the 95% LOA previously reported (Kilding *et al.*, 2001). Puente-Maestu *et al.* (2001) adopted a similar approach to the present study with respect to the way they quantitatively assessed the reproducibility of kinetic parameters. However, despite presenting the $\overline{X}_{diff}$ and SD_{diff}, the authors chose not to calculate the 95% LOA for kinetic parameter estimations. To permit a comparison of the level of reproducibility attained, it was possible to calculate the 95% LOA using their data (Table 3, pp 438). The 95% LOA for τ were ± 10.4 s (pre-training) and 8.0 s (post-training) and are considerably wider than those of the present study (± 1.9 s). Using the 95% LOA of the present study (Table 4.3), interpretation of the 95% LOA suggests that if a participants τ_{on} was 12.3 s in test 1 (grand mean of test 1 and 2 in this study), it is possible (worst case scenario) that the same participant could obtain an estimate for τ_{on} as low as 10.3 s, or as high as 14.1 s in test 2. To demonstrate the magnitude of reproducibility obtained in the study of Puente-Maestu *et al.* (2001), the 95% LOA suggest that a repeated estimation of τ (using their pre-training mean τ of 85 s), could be as low as 75.3 s and as high as 96.1 s. Similarly (using their post-training mean τ of 78 s), a repeated estimation of τ could be as low as 71.3 s and as high as 87.3 s. Obviously, these 95% LOA for τ are considered to be very wide and are substantially wider than those of the present study, but narrower than others (Kilding *et al.*, 2001). However, with consideration of the magnitude of the underlying mean τ of the populations studied the 95% LOA can be interpreted as being equivocal between studies. Finally, based on a high intra-class correlation between repeated kinetic parameter estimations, the authors

interpreted their results as being satisfactorily reproducible for the determination of τ in patients with COPD.

The direct comparison of results between the present study and that of Puente-Maestu *et al.* (2001) should be done with caution as the experimental approach differed with respect to the time interval between repeated tests and the participants used. The present study assessed day-to-day reproducibility in MD and LD runners whereas Puente-Maestu *et al.* (2001) assessed within-day reproducibility in patients with COPD. These participants represent two distinct ends of the fitness continuum.

In agreement with Puente-Maestu *et al.* (2001), the findings of the present study also show that the dispersion of the test 1-test 2 differences is not influenced by the magnitude of the value for either δ_{on}, τ_{on}, MRT_{on} or A_{on} within the population studied (Figure 4.2 - 4.5). This suggests that there was no relationship between the magnitude of the value and its reproducibility. This was confirmed by the non-significant correlation between the absolute test 1-test 2 differences and the mean values for tests 1 and 2.

With respect to the measurement error in the present study, the most reproducible kinetic parameter during the on-transient was MRT_{on} (4.3%). The MRT was also found to be the most reproducible parameter in a previous study (Kilding *et al.*, 2001). Conversely, the least reproducible parameter for the on-transient was τ_{on} which displayed 15.1% measurement error. This is greater (Puente-Maestu *et al.*, 2001) and less than (Kilding *et al.*, 2001) previously reported values. In the present study, the measurement error for δ_{on} (13.0%) and τ_{on} (15.1%) were similar.

Although Berry and Moritani, (1985) reported good reproducibility, their chosen method of analysing test-retest data (correlation) might have resulted in an incorrect conclusion about their intra-participant variability. This is because the use of test-retest

correlation's is not appropriate for the assessment of agreement between two repeated measures because *r* measures the strength of a relationship between variables, not the agreement between them (Bland and Altman, 1986; Atkinson and Nevill, 1998). Berry and Moritani (1985) reported a correlation coefficient of 0.87 and mean difference of 0.73 s between repeated tests. At first, this would appear to indicate an acceptable level of reproducibility. In comparison, the mean difference in the present study was -0.1 s for τ_{on} and 0.2 s for MRT_{on}. However, positive and negative differences between individuals test values can interact and potentially balance each other out. In such circumstances, the mean difference between tests would appear acceptable. However, it gives limited information about the intra-participant variability between tests. The SD_{diff} would more appropriately quantify the intra-participant variability between two repeated measures. The 95% LOA and method error techniques are determined from the SD_{diff} and are interpreted with respect to the mean of tests 1 and 2. The low method error for on-transient kinetic parameters also suggests that the measures were reproducible.

In the study of Özyener *et al.* (2001), the values for τ_{on} and A_{on} that were estimated from a single transition were found to differ from the values obtained from the ensemble-average of three transitions. These differences were well characterised by a normal distribution. To gain an approximate quantification of their reproducibility for τ_{on}, the results from their Table 3 (pp 896) were used to calculate the CV between individual transitions. Based on a mean and SD of 33 and 6.2 s respectively for τ_{on}, the CV between the single transitions was 19%. This is substantially greater than the CV for τ_{on} in the present study (4.8%). The CV is likely to be lower in the present study since repeated transitions were used to reduce the effects of noise, prior to exponential modelling of the data. This also reduced the variability between transitions. This was supported when considering the magnitude of the calculated method error for each kinetic parameter.

4.4.3 Reproducibility of off-transient $\dot{V}O_2$ kinetics

Despite the marginally wider 95% LOA for τ_{off} than those for τ_{on}, the parameter estimates of $\dot{V}O_2$ kinetics during the off-transient were equally reproducible. The 95% LOA suggest that the τ_{off} can be determined with an accuracy of ± 2.3 s (Table 4.5). For example, if a participants τ_{off} was 24.3 s (grand mean of test 1 and 2 in this study) in test 1, it is possible (worst case scenario) that the same participant could obtain an estimate for τ_{off} as low as 22.0 s, or as high as 26.6 s in test 2.

The magnitude of the measurement error for τ_{off}, in contrast, is much lower than that of τ_{on}. This is because the mean τ_{off} is longer than that of τ_{on}. This is supported by a lower method error. In support, Özyener *et al.* (2001) reported improved reproducibility for off-transient kinetic parameters compared to the on-transient. Based on the mean and the SD of τ_{off} (35 s and 4.0 s, respectively), their CV was 11%, which is considerably lower than the CV for τ_{on} (19%). This level of variability is not surprising since parameters were estimated from single transitions. In contrast, the CV for τ_{off} in the present study was considerably lower (2.9%) and is primarily attributable to the increased number of transitions used in this study.

In contrast to the smaller measurement error observed for τ_{off} than τ_{on}, measurement error and method error for MRT_{off} was greater than that observed for MRT_{on} (Tables 4.3 and 4.5 respectively). Since MRT_{off} is the sum of δ_{off} and τ_{off}, the measurement error and method error for MRT_{off} are influenced by the measurement error and/or method error observed for δ_{off}. Results confirmed this and showed that measurement error for δ_{off} (33%) was much larger than that for δ_{on} (13%).

With respect to the reproducibility of the off-transient amplitudes, measurement error for A_{off} (11%) was similar to that for A_{on} (11.3%). This would suggest that the differences in reproducibility for τ_{on} and τ_{off} is independent of the reproducibility

observed for A_{on} and A_{off}. Similarities between the magnitude of error for A_{on} and A_{off} have also been reported by Özyener *et al.* (2001).

4.4.4 Effects of noise on parameter estimation and reproducibility

The mean (± SD) 95% CI for kinetic parameter estimations of τ_{on} for tests 1 (0.9 ± 0.2 s) and 2 (1.0 ± 0.3 s) suggest that τ_{on} can be estimated accurately. This indicates that the ensemble average of six transitions minimises breath-by-breath variability, therefore permitting an accurate and reproducible estimation of τ during the on- and off-transient.

To improve the confidence in kinetic parameter estimations, alternative methods to repeated transitions have been used to reduce the SD_{noise}. Primarily, these have involved applying a data averaging technique to the interpolated breath-by-breath data from a single transition prior to exponential modelling (Berry and Moritani, 1985; Demarle *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001). Based on the level of reproducibility reported from studies using this approach, it appears that data averaging techniques are not as effective in reducing the SD_{noise} as repeated transitions.

The SD_{noise} was not appreciably different between this study and that of Kilding *et al.* (2001). However, in this study, MD and LD runners displayed larger amplitudes of $\dot{V}O_2$ as a result of their increased range of exercise intensities within the moderate-intensity domain. Therefore, the SD_{noise} - amplitude ratio (%) was appreciably lower. This allowed a clearer distinction between the underlying physiological response and the noise (Lamarra *et al.*, 1987). This was reflected by a much improved 95% CI which contributed to the reproducible determination of on- and off-transient $\dot{V}O_2$ kinetic parameter estimations in the present study. It has been acknowledged that 'noise' associated with a single transition could readily account for the variation seen in estimates of τ, although the possibility of real variation cannot be excluded (Özyener *et al.*, 2001). The identification of real variation, i.e. biological variability, can only be distinguished if the SD_{noise} is sufficiently minimised and discounted as a possible source

of variability. With consideration of the results of the present study, this can only be achieved using several repeated transitions, probably six.

Despite the potentially confounding effects of a reduced amplitude of $\dot{V}O_2$ resulting from a restricted tolerance of exercise intensities within the moderate domain, parameter estimations of $\dot{V}O_2$ kinetics were found to be satisfactorily reproducible (Puente-Maestu *et al.*, 2001). However, the participants of this study were COPD patients and displayed a mean τ of ~80 s which is more than six times longer than τ_{on} in the present study. This increased amount of data which was available for exponential modelling throughout the transient has been shown to influence the 95% CI associated with kinetic parameter estimations (Lamarra *et al.*, 1987). Therefore, the reduced amplitude of $\dot{V}O_2$ in COPD patients was compensated for by a substantially longer τ_{on}.

4.4.5 Methodological influences on reproducibility

It is possible that differences in results between studies assessing reproducibility of kinetic parameter estimations can be attributable to differences in experimental design such as the time between repeated tests, number of transitions performed, mode of ergometry and the physiological characteristics (i.e. level of aerobic fitness) of the participants involved. It has yet to be established to what extent different protocols influence the reproducibility of $\dot{V}O_2$ kinetic parameters and which is the optimal. This study differed from previous studies (Berry and Moritani, 1985; Demarle *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) in that the reproducibility between parameter estimations was obtained from two data sets, each comprising six transitions. Furthermore, because this approach measured the $\dot{V}O_2$ kinetics during the on- and off-transients in one visit to the laboratory, on two separate days, it also permitted the quantification of biological day-to-day variability in $\dot{V}O_2$ kinetics. Previous approaches used to assess the reproducibility of $\dot{V}O_2$ kinetics have involved single transitions that are repeatedly performed on separate days (Berry and Moritani, 1985; Özyener *et al.*, 2001) or within the same day (Puente-Maestu *et al.*, 2001). These

approaches prevent assessments of day-to-day variability. In addition, the poorer reproducibility observed (Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) would suggest that the single transition approach is unsuitable to allow reproducible determinations of $\dot{V}O_2$ kinetic parameters since the effects of noise have not been minimised.

The protocol used in the present study was continuous. As a result, the second and third transitions of each set were inevitably preceded by one and two bouts of moderate-intensity exercise respectively. This could be perceived as a 'warm-up'. It has been shown in most studies that prior moderate- or heavy-intensity exercise does not influence τ during the transition to moderate-intensity exercise (Gerbino *et al.*, 1996; Burnley *et al.*, 2000). It is assumed that the on-transient $\dot{V}O_2$ kinetic response, during the square-wave transitions that were preceded by moderate-intensity exercise, was not influenced by prior bouts of moderate-intensity exercise.

Every effort was made to minimise both the technical error and the effects of noise on $\dot{V}O_2$ kinetic parameter estimations. It is realistic to conclude that the minimal amount of test-retest variability observed was not due to the protocol used.

4.4.6 Application of reproducibility to changes in kinetic parameters

To put the magnitude of variability observed between repeated tests into perspective, it is important to consider the changes in τ (or MRT) that might result from the prescription of a specific exercise training intervention. Puente-Maestu *et al.* (2001) calculated the magnitude of the change in τ_{on} that would be required to reach statistical significance using a one- and two-tailed paired-comparison test. This calculation was based on the SD_{diff} between repeated tests (prior to a training intervention study in patients with COPD - Puente-Maestu *et al.*, 2000), the number of participants (n=35) and the alpha level (P = 0.05). The required change in τ_{on} was 9 and 10.8 s for a one- and two-tailed paired-comparison respectively. Changes in τ_{on} of this magnitude have

been reported when exercise programs have been prescribed for the previously untrained (Yoshida *et al.*, 1992; Babcock *et al.*, 1994; Phillips *et al.*, 1995). For example, a mean decrease (i.e. faster $\dot{V}O_2$ kinetics) of 21.4 s (τ_{on}), 10.4 s (MRT_{on}), 6.3 s (τ_{off}) and 7.4 s (MRT_{off}) has been reported (Phillips *et al.*, 1995). This equates to an improvement of 57.5, 27.3, 20.1 and 19.5% respectively, compared to pre-training values. Similarly, a mean increase (i.e. slowing of $\dot{V}O_2$ kinetics) of 16.6 s (τ_{on}) and 13.4 s (MRT_{on}) was caused by the effects of ageing in older humans (Bell *et al.*, 1999). This equates to a 56.3 and 43.9% change when compared to the initial determination τ_{on} and MRT_{on} respectively. Changes in $\dot{V}O_2$ kinetics of this magnitude are easily detectable and in most instances will exceed the test-retest variability in parameter estimations (Kilding *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001). The problem resides, however, in studies where smaller, but significant changes in τ and MRT are expected and are meaningful. It is unlikely that changes in τ (or MRT) of this magnitude would be evident whilst monitoring endurance trained athletes. Therefore, the reproducibility previously reported for untrained individuals (Kilding *et al.*, 2001) and patients with COPD (Puente-Maestu *et al.*, 2001) would be unacceptable. In competitive athletes, significantly smaller changes in τ_{on} [pre = 29.2 s; mid = 24.4 s (16.4% decrease); post = 21.9 s (25% decrease)] during and after 8 weeks of endurance training have been reported (Norris and Peterson, 1998). Given the improved level of reproducibility established in the present study, it would be possible to identify smaller changes in τ. Using the approach of Puente-Maestu *et al.* (2001), a change of 1.7 and 2.1 s in τ_{on} would be required to reach statistical significance ($P < 0.05$) using a one- and two-tailed paired-comparison respectively.

4.4.7 Reproducibility of other physiological measures

The reproducibility of $\dot{V}O_2$ kinetic parameters found in this study compare well with the reproducibility of other measures that have been used to asses and monitor the physiological status of endurance trained athletes. These include $\dot{V}O_{2\,max}$ (Weltman *et al.*, 1990), LT/V_T (Aunola and Rusko, 1984; Weltman *et al.*, 1990; McLellan and

Jacobs, 1993) and RE (Armstrong and Costill, 1985, Morgan *et al.*, 1991; Pereira and Freedson, 1997). The CV of $\dot{V}O_{2\,max}$ has been reported as being 3.7 to 7.3% during 8-20 successive treadmill tests in five subjects (Katch *et al.*, 1982). This degree of variability is similar to the CV for each kinetic parameter measured in the present study, which ranges from 1.3 to 4.8% and 2.2 to 13.4% for the on- and off-transients respectively.

Weltman *et al.* (1990) conducted an extensive study examining the reliability and validity of a treadmill protocol for the determination of physiological measures (LT, fixed blood lactate concentrations of 2.0, 2.5 and 4.0 $mmol \cdot l^{-1}$ and $\dot{V}O_{2\,max}$) pertinent to the assessment of distance runners. Strong correlation's between repeated tests (range r = 0.70 - 0.95) were identified for each physiological measure. Similarly, the variability observed for RE has been reported (Armstrong and Costill, 1985) and is comparable to that of kinetic parameters observed in the present study. Armstrong and Costill (1985) found that the CV for sub-maximal $\dot{V}O_2$ in trained runners was 3.8%. In comparison, the CV for $\dot{V}O_{2\,(m)}$ and A_{on} (equivalent measures of steady-state $\dot{V}O_2$) in the present study was 2.7 and 3.4% respectively.

4.5 Conclusion

The main finding of this study suggests that a six-transition protocol allows a reproducible estimation of $\dot{V}O_2$ kinetic parameters during both the on- and off-transients. The narrow 95% LOA, small measurement and method error demonstrate this. This also suggests that the day-to-day biological variability in $\dot{V}O_2$ kinetics is small. However, biological variability can only be accurately discerned if the effects of noise on kinetic parameter estimations are sufficiently minimised. This was achieved in the present study by having participants perform several repeated transitions. The reproducibility established for $\dot{V}O_2$ kinetic parameters is superior to that previously reported and compares well to the reproducibility of alternative physiological measures commonly used to assess and monitor MD and LD runners. Collectively, these findings

exemplify the importance of establishing and quantifying the reproducibility of $\dot{V}O_2$ kinetics, since what is considered acceptable reproducibility in some populations might not be acceptable for others. The reproducibility established in this study is acceptable for further assessments of MD and LD runners.

CHAPTER 5

On- and off-transient pulmonary oxygen uptake kinetics in middle- and long-distance runners

5.1 Introduction

It has been demonstrated in several studies that τ_{on} is reduced after short-term endurance training in previously untrained (Hickson *et al.*, 1978; Hagberg *et al.*, 1980; Yoshida *et al.*, 1992; Phillips *et al.*, 1995) and trained individuals (Norris and Peterson, 1998). In addition, several studies have characterised on-transient $\dot{V}O_2$ kinetics in long-term, habitually trained individuals including sprinters (Edwards *et al.*, 1999), American footballers (Fukuoka *et al.*, 1995), swimmers (Cerretelli *et al.*, 1979) and distance runners (deVries *et al.*, 1982; Powers *et al.*, 1985; Lake *et al.*, 1986; Taylor *et al.*, 1999). Collectively, these studies have been conducted in an attempt to improve our understanding of the physiological effects of different training regimes in athletes and to identify the mechanism(s) determining $\dot{V}O_2$ kinetics at the onset of exercise.

5.1.1 On-transient $\dot{V}O_2$ kinetics

The $\dot{V}O_2$ kinetics in endurance-trained runners have been investigated in several studies using square-wave transitions in the intensity of exercise (deVries *et al.*, 1982; Powers *et al.*, 1985; Lake *et al.*, 1986). Predominantly, the $\dot{V}O_2$ $t_{½}$ (defined as the time to reach one-half of the steady-state $\dot{V}O_2$) has been used to characterise the rate of response. deVries *et al.* (1982) reported that the $\dot{V}O_2$ kinetics of young and old endurance-trained runners were similar (27.4 and 30.0 s respectively) despite large differences in $\dot{V}O_{2\,max}$. This suggests that $\dot{V}O_2$ kinetics, unlike $\dot{V}O_{2\,max}$, is independent of ageing in endurance-trained runners. In LD runners (n=10) who had similar training regimes but differed with respect to $\dot{V}O_{2\,max}$, Powers *et al.* (1985) reported the $\dot{V}O_2$ $t_{½}$ to range from 21.6 to 36.0 s and were faster in runners who had a higher $\dot{V}O_{2\,max}$. Lake *et al.* (1986) reported a mean $\dot{V}O_2$ $t_{½}$ of 33.4 ± 5.2 s when $\dot{V}O_2$ was plotted at the endpoint (EP) of a 20 s time interval but 26.7 ± 5.2 s when $\dot{V}O_2$ was plotted at the midpoint (MP) of a 20 s time

interval. Clearly, this highlights the sensitivity of different modelling techniques on $\dot{V}O_2$ kinetics. However, with respect to $\dot{V}O_2$ $t_{½}$, there appears to be some consistency between the mean values reported for endurance trained runners, despite differences in $\dot{V}O_{2\,max}$. In spite of this observation, the use of $\dot{V}O_2$ $t_{½}$ to characterise $\dot{V}O_2$ kinetics is inappropriate because the initial response of $\dot{V}O_2$, i.e. the 'cardiodynamic' phase (Whipp *et al.*, 1982), is included in the modelling. Exponential models, incorporating a delay-term, that exclude the phase I response should preferably be used. Using this approach, Lake *et al.* (1986) reported the τ_{on} of runners (n=8) which was 25.3 ± 8.7 s and 30.8 ± 7.3 s for EP and MP respectively. Conversion of the $\dot{V}O_2$ $t_{½}$ reported by Powers *et al.* (1985) corresponds to a τ_{on} ranging between ~31.2 and 51.9 s; longer than those reported by Lake *et al.* (1986). This reflects the higher $\dot{V}O_{2\,max}$ of the runners in the study of Lake *et al.* (1986).

5.1.2 Off-transient $\dot{V}O_2$ kinetics

The majority of research to date has characterised the kinetics of $\dot{V}O_2$ and/or assessed the effects of training on $\dot{V}O_2$ kinetics during the on-transient. However, early studies on the time course of $\dot{V}O_2$ focussed primarily on the recovery period (Margaria *et al.*, 1933; Berg, 1947). Berg (1947) was the first to measure $\dot{V}O_2$ both during the onset of exercise and during recovery but chose only to analyse the recovery responses of $\dot{V}O_2$. Today, few studies have investigated the effects of training (short- or long-term) on $\dot{V}O_2$ kinetics during both on- and off-transients (Hagberg *et al.*, 1980; Phillips *et al.*, 1995). After 9 weeks training, Hagberg *et al.* (1980) reported a shortening in both the on-transient $\dot{V}O_2$ $t_{½}$ and the off-transient $\dot{V}O_2$ $t_{½}$. Similarly, Phillips *et al.* (1995) reported faster off-transient $\dot{V}O_2$ kinetics after 30 days of endurance training with reductions in τ_{off} and MRT_{off} of ~20 %. Both studies highlight the sensitivity of off-transient $\dot{V}O_2$ kinetics to endurance training

The study of Carter *et al.* (2000a) is the only study that has characterised on- and off-transient $\dot{V}O_2$ kinetics for both treadmill and cycle exercise over a range of exercise

intensities in recreationally active individuals. Specifically, during recovery from moderate-intensity treadmill and cycle exercise, τ_{off} was 39.3 ± 3.0 s and 35.9 ± 4.2 s respectively. However, these values for τ_{off} are longer than the pre-training τ_{off} (31.3 ± 1.3 s) reported by Phillips *et al.* (1995), despite participants in both studies having a similar $\dot{V}O_{2\,max}$. This difference could be caused by the different methods of recovery between the studies. The study of Carter *et al.* (2000a) prescribed complete rest, whereas Phillips *et al.* (1995) used active recovery involving pedalling at 25 watts (W).

5.1.3 Symmetry between on- and off-transient kinetics

Partly as a consequence of the limited research into off-transient $\dot{V}O_2$ kinetics before and after training in runners, there are few comparisons concerning the symmetry between the on- and off-transients in this population, especially during treadmill running. There is disagreement about whether or not there are symmetries between on- and off-transients for exercise below the V_T in untrained individuals. For example, several studies have demonstrated symmetry between transients (Paterson and Whipp, 1991; Scheuermann *et al.*, 1998; Brittain *et al.*, 2001; Özyener *et al.*, 2001). Symmetry has also been observed before and after endurance training (Hagberg *et al.*, 1980). However, such symmetry is not always the case. Distinct asymmetries between τ_{on} and τ_{off} have been observed in untrained (Linnarsson, 1974; Hughson *et al.*, 1988; Carter *et al.*, 2000a) and trained individuals (Cerretelli *et al.*, 1979; Phillips *et al.*, 1995). These studies show that on-transient $\dot{V}O_2$ kinetics are faster than off-transient $\dot{V}O_2$ kinetics for cycle (Linnarsson, 1974; Hughson *et al.*, 1988; Carter *et al.*, 2000a) and treadmill (Carter *et al.*, 2000a) exercise. It has yet to be established whether there are symmetries (or asymmetries) between on- and off-transient $\dot{V}O_2$ kinetic responses in MD and LD runners using treadmill ergometry.

5.1.4 Methodological limitations to previous studies

There are limitations common to several of the aforementioned studies. First, most have used cycle ergometry in their determinations of phase II $\dot{V}O_2$ kinetics in MD and

LD runners (deVries *et al.*, 1982; Powers *et al.*, 1985). For an appropriate and specific determination of $\dot{V}O_2$ kinetics in runners, a mode of ergometry to which the athlete is accustomed would be preferable. Recently, the use of treadmill ergometry to characterise the on- (Carter *et al.*, 2000a, b; Williams *et al.*, 2001) and off-transient (Carter *et al.*, 2000a) $\dot{V}O_2$ kinetics in untrained individuals has been reported. However, no study has specifically assessed $\dot{V}O_2$ kinetics during both the on- and off-transients in MD and LD runners using treadmill ergometry. Second, the sampling intervals of $\dot{V}O_2$ in some studies have been unacceptably large. For example, $\dot{V}O_2$ kinetics have been measured using 10 (deVries *et al.*, 1982), 15 (Powers *et al.*, 1985) and 20 s (Lake *et al.*, 1986) time intervals respectively. Potentially, this might result in the inclusion of only two or three data points during the transient which could influence kinetic parameter estimations. Presently, most studies measure $\dot{V}O_2$ on a breath-by-breath basis, which improves the time resolution of data and most importantly, allows different phases of the $\dot{V}O_2$ response (Whipp *et al.*, 1982) to be quantified during both transients, providing there are enough transitions.

5.1.5 Aim of study

The aim of this study was two-fold. First, to characterise and compare $\dot{V}O_2$ kinetics during the on- and off-transients in MD and LD runners and second, to assess the symmetries (or asymmetries) and relationships between on- and off-transient $\dot{V}O_2$ kinetics at the onset and during recovery from moderate-intensity treadmill running.

5.2 Participants and methods

5.2.1 Participants

Ten male MD (800/1500 m) and 10 male LD (5000/10000 m) runners provided written informed consent and participated. Participants were recruited from athletic clubs in the North of England. Participants' age, anthropometric and physiological characteristics are presented in Table 5.1. Ethics approval was obtained from the Research Ethics Committee, Sheffield Hallam University. Prior to participation in the study each athlete completed a medical screening questionnaire (Appendix 6).

Table 5.1 Participants age, anthropometric and physiological characteristics. Values are mean ± SD.

Measure	MD (n=10)	LD (n=10)	Combined (n=20)
Age (years)	22.0 ± 6.8	25.8 ± 5.0	23.9 ± 6.1
Stature (cm)	176.6 ± 5.8	180.0 ± 8.1	178.3 ± 7.0
BM (kg)	65.3 ± 5.0	71.4 ± 9.8	68.4 ± 8.2
Volume of training ($km \cdot wk^{-1}$)	47.5 ± 18.8	64.0 ± 15.7*	55.7 ± 18.8
$\dot{V}O_{2\,max}$ ($ml \cdot min^{-1}$)	3912 ± 341	4202 ± 702	4057 ± 557
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	60.0 ± 4.9	59.0 ± 6.3	59.5 ± 5.5
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.79} \cdot min^{-1}$)$^{\$}$	144 ± 11	144 ± 16	144 ± 13
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	238 ± 18	241 ± 27	239 ± 22
$\dot{V}O_2$ at V_T ($ml \cdot min^{-1}$)	3248 ± 286	3437 ± 447	3343 ± 378
$\dot{V}O_2$ at V_T ($ml \cdot kg^{-1} \cdot min^{-1}$)	49.9 ± 4.8	48.4 ± 4.4	49.1 ± 4.6
$\dot{V}O_2$ at V_T ($ml \cdot kg^{-0.64} \cdot min^{-1}$)$^{\$}$	224 ± 19	224 ± 19	224 ± 19
$\dot{V}O_2$ at V_T ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	198 ± 17	197 ± 16	198 ± 16

$^{\$}$Empirically derived BM exponent from present data; *Significantly different from MD runners, P <0.05;

5.2.2 Experimental design

Participants visited the laboratory for physiological testing on two occasions within a seven-day period. Each test was separated by at least 48 hours and was performed at approximately the same time of day. In the first visit, participants performed an incremental exercise test to volitional exhaustion to allow the determination of V_T and $\dot{V}O_{2\,max}$. In the second visit, participants performed a square-wave exercise protocol to determine on- and off-transient $\dot{V}O_2$ kinetics during the onset and recovery from moderate-intensity exercise respectively. Throughout the testing period, participants were requested to maintain their usual dietary intake and to abstain from heavy training and consumption of alcohol and/or caffeine in the 48 hours preceding each test.

5.2.3 Experimental protocols

All running tests were performed on a motor-driven treadmill (Saturn 250-75R, HP Cosmos, Germany). Each participant completed an incremental exercise test to volitional exhaustion for the determination of V_T and $\dot{V}O_{2\,max}$ (see Chapter 3, Section 3.2.5). To assess $\dot{V}O_2$ kinetics, participants completed a square-wave protocol (see Chapter 3, Section 3.2.7) consisting of alternating 6 min bouts of walking (4 $km \cdot h^{-1}$) and running (speed requiring 80%V_T). Pulmonary gas-exchange (breath-by-breath) and HR were measured during all exercise tests. The [HLa] was measured before and after the square-wave protocol.

5.2.4 Data analysis

Breath-by-breath data obtained during the incremental exercise test and the square-wave protocol were analysed in accordance with procedures outlined in Chapter 3, Section 3.2.5.1 and 3.2.7.1 respectively.

5.2.5 Statistical analyses

Descriptive statistics (mean ± SD) were calculated for each physiological measure during each test. For the most appropriate expression of $\dot{V}O_{2\,max}$ and V_T with respect to

BM, log-log transformations and ANCOVA were used to identify the common b exponents for MD and LD runners respectively. Independent t-tests were used to compare $\dot{V}O_2$ kinetics of MD and LD runners. Dependent t-tests were used to compare on- and off-transient $\dot{V}O_2$ kinetics. Relationships between $\dot{V}O_2$ kinetic parameters were determined using Pearson's product moment correlation coefficient. Statistical significance for all tests was set at P <0.05. Prior to conducting these analyses, appropriate checks were made to ensure that the assumptions underpinning these statistical approaches were met (see Chapter 3, Section 3.3).

5.3 Results

5.3.1 Incremental test

The $\dot{V}O_{2\,max}$ and V_T in MD and LD runners are presented in Table 5.1. Measures are presented in absolute terms ($ml \cdot min^{-1}$), proportional to BM ($ml \cdot kg^{-1} \cdot min^{-1}$) and as a 0.67 power-function ratio of BM. Log-log transformations and ANCOVA showed that a common *b* exponent could be used for MD and LD runners since the slope and estimated marginal means were not significantly different. The *b* exponents for $\dot{V}O_{2\,max}$ and V_T were 0.79 ± 0.20 and 0.64 ± 0.19 respectively; mean ± SE. Consideration of the leverage statistics (Mahalanobis and Cooks' distances) suggested that no outlying data were influencing the regression models. There were no differences between MD and LD runners for either $\dot{V}O_{2\,max}$ or V_T regardless of how they were expressed ($P > 0.05$). The actual $\dot{V}O_2$ at the pre-determined speeds ($\dot{V}O_{2\,(m)}$, Table 5.2) was equivalent to 81.4 ± 3.4 and 80.5 ± 4.0%V_T for MD and LD runners respectively. This demonstrates that the protocol used to identify the appropriate running speed for a given intensity of exercise ensured that each runner exercised at the same relative intensity (i.e. ~80%V_T). The volume of training ($km \cdot wk^{-1}$) completed by LD runners was greater than that of MD runners ($P = 0.047$).

5.3.2 On-transient $\dot{V}O_2$ kinetics

Typical breath-by-breath $\dot{V}O_2$ responses for the transition from walking at 4 $km \cdot h^{-1}$ to running at a moderate-intensity (80%V_T) are presented in Figure 5.1a and b for both a MD and LD runner respectively. The characteristic three-phase response of $\dot{V}O_2$ at the onset of exercise was clearly apparent and a steady-state $\dot{V}O_2$ was attained within 2-3 min. Qualitatively, modelling the on-transient response with a mono-exponential model, with time-delay, resulted in a good fit of the data whereby residuals throughout the fitting window were seen to fluctuate randomly around zero error (Figure 5.1a, b).

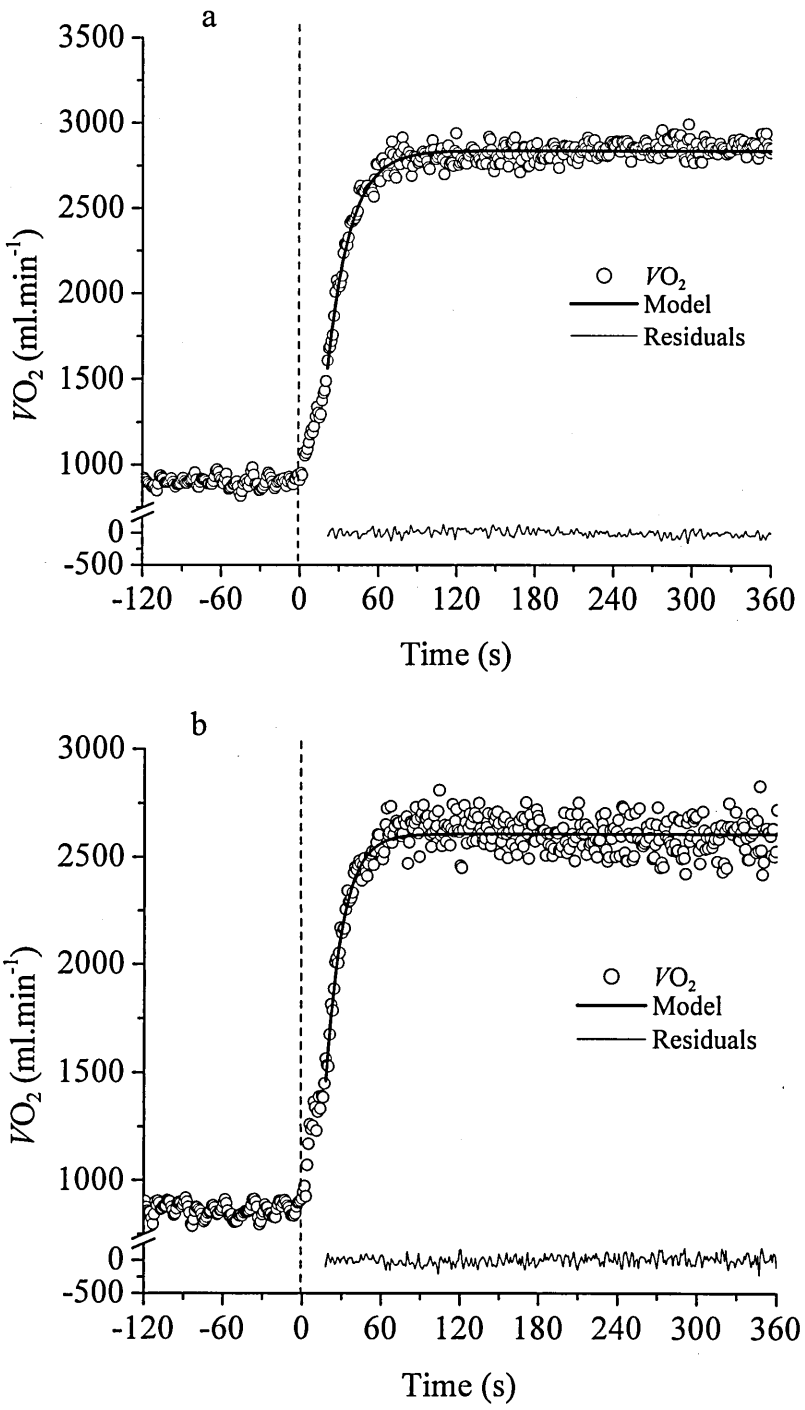


Figure 5.1 a, b The typical $\dot{V}O_2$ response for a square-wave transition from walking to moderate-intensity running for a MD runner (a) (where A_{on} = 1937 ml·min^{-1}; δ_{on} = 13.8 s; τ_{on} = 17.1 s; MRT_{on} = 30.9 s) and (b) LD runner (where A_{on} = 1729 ml·min^{-1}; δ_{on} = 11.9 s; τ_{on} = 12.6 s; MRT_{on} = 27.1 s). The vertical dashed line denotes the onset of moderate-intensity exercise.

All kinetic parameters from the modelled $\dot{V}O_2$ responses at the onset of moderate-intensity exercise are presented in Table 5.2. The HR and [HLa] responses are also presented. There were no differences (P >0.05) between MD and LD runners for any kinetic parameter. Using the equations of Lamarra *et al.* (1987), the 95% CI associated with kinetic parameter estimations during the on-transient were 1.1 ± 0.3 and 0.9 ± 0.3 s for MD and LD runners respectively. Pre- and post-test [HLa] did not differ (P = 0.532) and are presented in Table 5.2 as delta (Δ) values (the difference between resting and end-exercise values). Both $HR_{(b)}$ (73 ± 9 vs. 88 ± 13 b·min^{-1}; P = 0.004) and $HR_{(m)}$ (135 ± 9 vs. 147 ± 9 b·min^{-1}; P = 0.005) were lower in LD runners than in MD runners respectively.

Table 5.2 Measures associated with the $\dot{V}O_2$, HR and [HLa] responses during moderate-intensity exercise in runners. Values are mean ± SD.

Measure	MD (n=10)	LD (n=10)	Combined (n=20)
Running speed ($km \cdot h^{-1}$)	11.7 ± 0.8	11.6 ± 0.7	11.7 ± 0.8
Resting $\dot{V}O_2$ ($ml \cdot min^{-1}$)	397 ± 25	396 ± 46	397 ± 36
$\dot{V}O_{2\,(b)}$ ($ml \cdot min^{-1}$)	901 ± 39	908 ± 112	905 ± 82
A_{on} ($ml \cdot min^{-1}$)	1739 ± 170	1855 ± 257	1797 ± 220
$\dot{V}O_{2\,(m)}$ ($ml \cdot min^{-1}$)	2641 ± 196	2764 ± 348	2702 ± 282
δ_{on} (s)	14.4 ± 1.3	14.6 ± 1.5	14.5 ± 1.3
τ_{on} (s)	14.2 ± 3.1	12.5 ± 2.3	13.3 ± 2.8
MRT_{on} (s)	28.6 ± 2.5	27.1 ± 2.2	27.8 ± 2.4
Gain ($ml \cdot kg^{-1} \cdot km^{-1}$)	177.1 ± 9.5	171.7 ± 9.4	174.4 ± 9.6
$HR_{(b)}$ ($b \cdot min^{-1}$)	88 ± 13	72 ± 9**	80 ± 14
$HR_{(m)}$ ($b \cdot min^{-1}$)	147 ± 9	135 ± 9**	141 ± 10
Δ[HLa] ($mmol \cdot l^{-1}$)	-0.03 ± 0.08	0.01 ± 0.11	-0.01 ± 0.10

Gain, $\dot{V}O_{2\,(m)}$ minus resting $\dot{V}O_2$, relative to BM and running speed at 80%V_T. *Lower in LD runners, P <0.05; **Lower in LD runners, P <0.01.

5.3.3 Off-transient $\dot{V}O_2$ kinetics

The $\dot{V}O_2$ response of a MD and LD runner at the cessation of moderate-intensity exercise is illustrated in Figure 5.2 a and b respectively. The residuals from the mono-exponential modelling are also presented and, as observed during the on-transient, demonstrate a random distribution around zero error.

In contrast with on-transient $\dot{V}O_2$ kinetic parameters, both τ_{off} (27.1 ± 3.0 s vs. 24.1 ± 2.3 s; P = 0.023) and MRT_{off} (36.0 ± 3.1 s vs. 32.4 ± 2.4 s; P = 0.011) were faster in LD runners than in MD runners. The MD and LD runners did not differ in their δ_{off} (P = 0.660) or A_{off} (P = 0.219).

Table 5.3 Measures of the $\dot{V}O_2$ kinetic response during recovery from moderate-intensity exercise in runners. Values are means ± SD.

Measure	MD (n=10)	LD (n=10)	Combined (n=20)
$\dot{V}O_{2\,(m)}$ (ml·min^{-1})	2463 ± 194	2761 ± 347	2702 ± 280
A_{off} (ml·min^{-1})	1731 ± 173	1857 ± 261	1794 ± 225
$\dot{V}O_{2\,(b)}$ (ml·min^{-1})	912 ± 38	904 ± 120	908 ± 86
δ_{off} (s)	8.9 ± 2.3$^{\#\#}$	8.3 ± 3.3$^{\#\#}$	8.6 ± 2.8$^{\#\#}$
τ_{off} (s)	27.1 ± 3.0$^{\#\#}$	24.1 ± 2.3*$^{\#\#}$	25.6 ± 3.0$^{\#\#}$
MRT_{off} (s)	36.0 ± 3.1$^{\#\#}$	32.4 ± 2.4*$^{\#\#}$	34.2 ± 3.3$^{\#\#}$

*Lower in LD runners, P <0.05; $^{\#\#}$Different from on-transient, P <0.01.

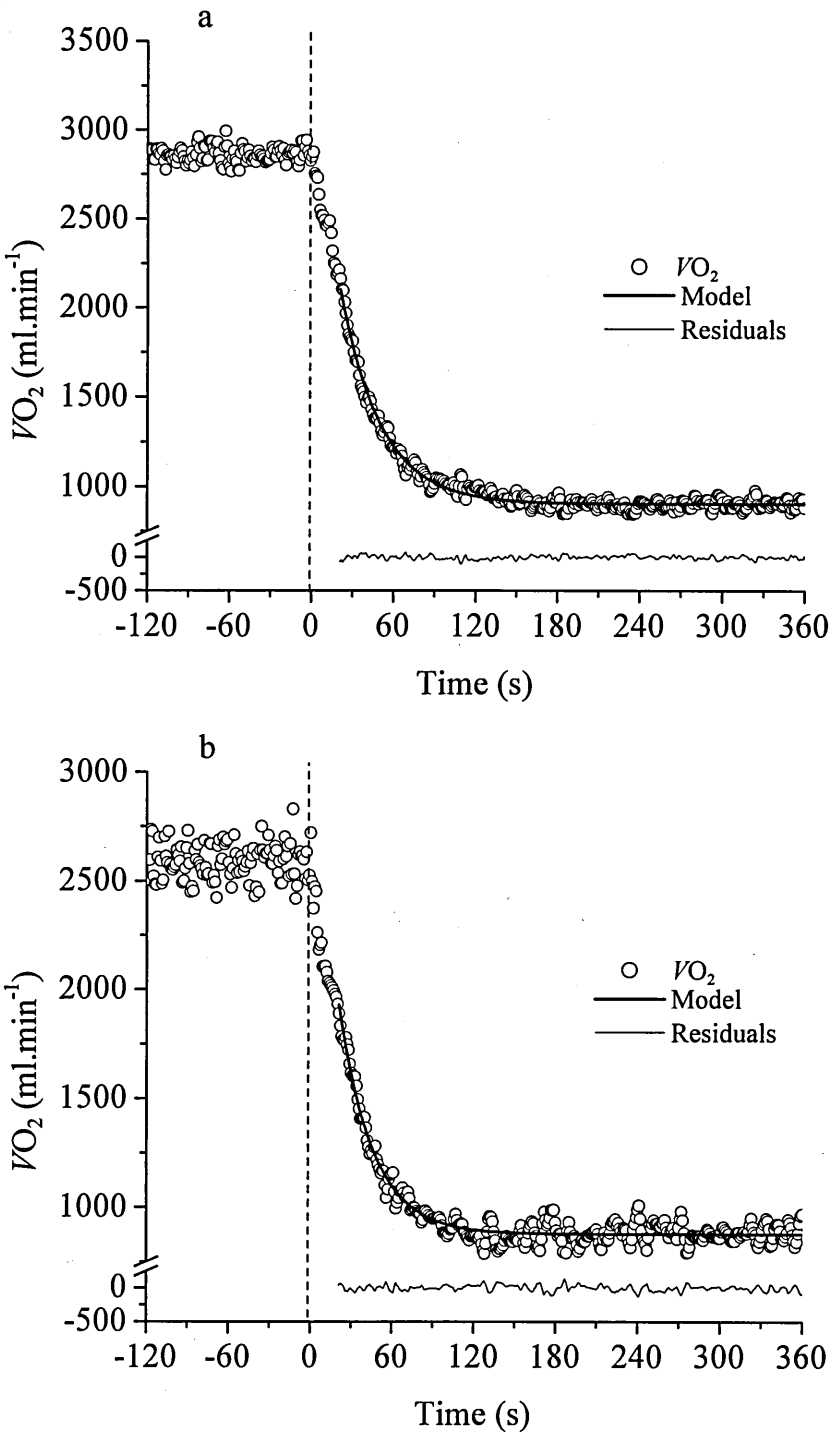


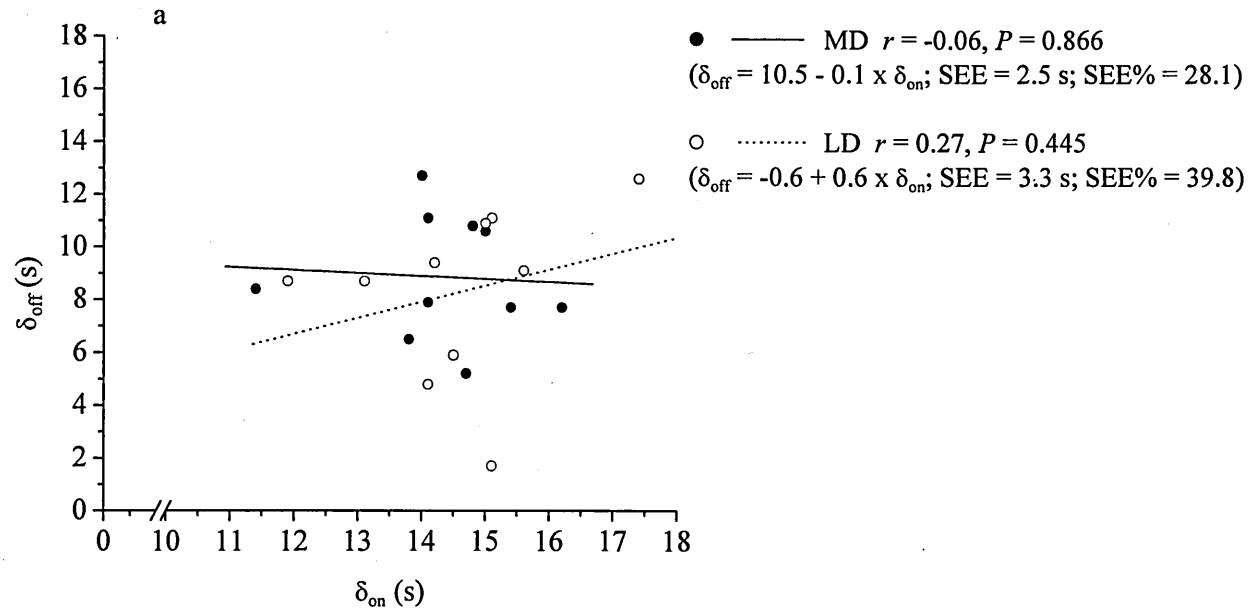
Figure 5.2 a, b. The typical $\dot{V}O_2$ response for a square-wave transition from moderate-intensity running to walking for a MD runner (a) (where A_{off} = 1729 ml·min^{-1}; δ_{off} = 7.7 s; τ_{off} = 31.1 s; MRT_{off} = 38.9 s) and (b) LD runner (where A_{off} = 1717 ml·min^{-1}; δ_{off} = 8.7 s; τ_{off} = 25.1 s; MRT_{off} = 33.8 s). The vertical dashed line denotes the end of moderate-intensity exercise.

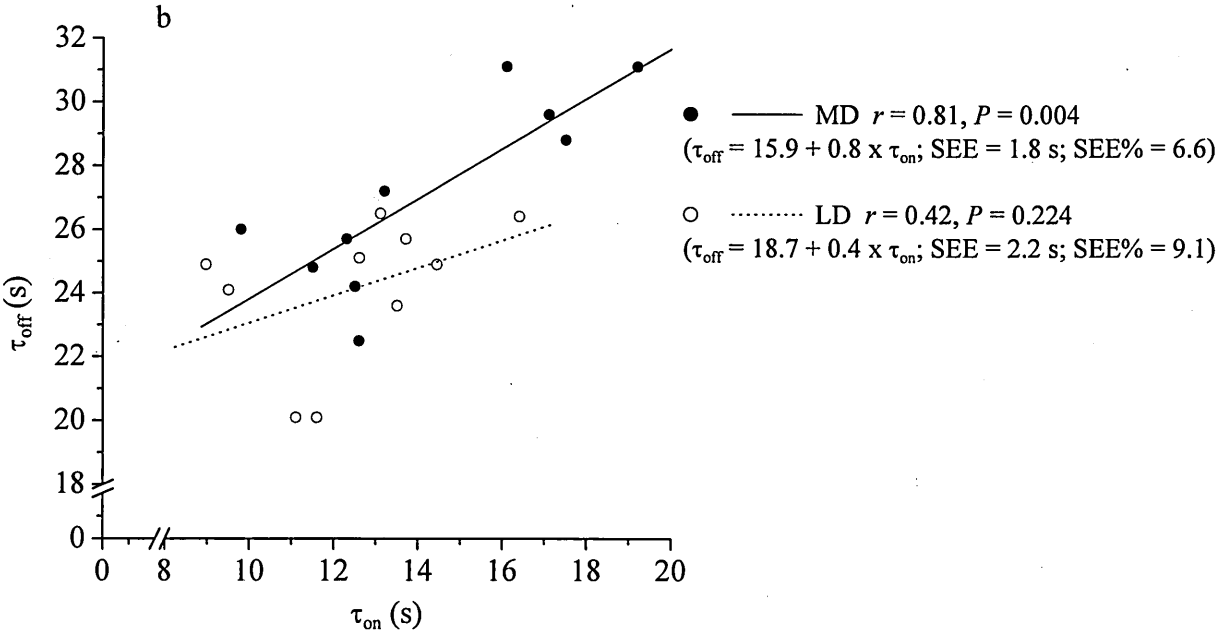
5.3.4 On- vs. Off-transient $\dot{V}O_2$ kinetics

Comparisons between on- and off-transients revealed some differences (Tables 5.2 and 5.3). For example, δ_{on} (14.4 ± 1.3 s and 14.6 ± 1.5 s) was longer than δ_{off} (8.9 ± 2.3 s and 8.3 ± 3.3 s) both in MD and LD runners respectively (P <0.001). Conversely, τ_{on} (14.2 ± 3.1 s and 12.5 ± 2.3 s) and MRT_{on} (28.6 ± 2.5 s and 27.1 ± 2.2 s) were shorter than τ_{off} (27.1 ± 3.0 s and 24.1 ± 2.3 s) and MRT_{off} (36.0 ± 3.1 s and 32.4 ± 2.4 s) in both MD and LD runners respectively (P <0.001). However, there were no differences between A_{on} (MD = 1739 ± 170 ml·min^{-1}; LD = 1855 ± 257 ml·min^{-1}) and A_{off} (MD = 1731 ± 173 ml·min^{-1}; LD = 1857 ± 261 ml·min^{-1}) in both MD and LD runners respectively (P = 0.605).

5.3.5 Correlations between $\dot{V}O_2$ kinetic parameters

Correlation coefficients between the on- and off-transient $\dot{V}O_2$ kinetic parameters are illustrated in Figure 5.3 (a-d). In MD and LD runners, δ_{on} and δ_{off} were not related (r = 0.13; P = 0.575). Conversely, there was a relationship between τ_{on} and τ_{off} for MD (r = 0.81, P = 0.004) runners but not for LD runners (r = 0.42, P = 0.224). Similarly, MRT_{on} and MRT_{off} were highly related in MD (r = 0.86, P = 0.001), but not for LD runners (r = 0.14, P = 0.706). These findings suggest that a shorter τ_{on} (or MRT_{on}) is associated with a short τ_{off} (or MRT_{off}) only in MD runners. In MD and LD runners, there was a relationship between A_{on} and A_{off} (r = 0.98 and 0.99 respectively; P <0.001).


a
● —— MD r = -0.06, P = 0.866
(δ_{off} = 10.5 - 0.1 x δ_{on}; SEE = 2.5 s; SEE% = 28.1)
○ ····· LD r = 0.27, P = 0.445
(δ_{off} = -0.6 + 0.6 x δ_{on}; SEE = 3.3 s; SEE% = 39.8)
δ_{off} (s)
δ_{on} (s)


b
● —— MD r = 0.81, P = 0.004
(τ_{off} = 15.9 + 0.8 x τ_{on}; SEE = 1.8 s; SEE% = 6.6)
○ ····· LD r = 0.42, P = 0.224
(τ_{off} = 18.7 + 0.4 x τ_{on}; SEE = 2.2 s; SEE% = 9.1)
τ_{off} (s)
τ_{on} (s)

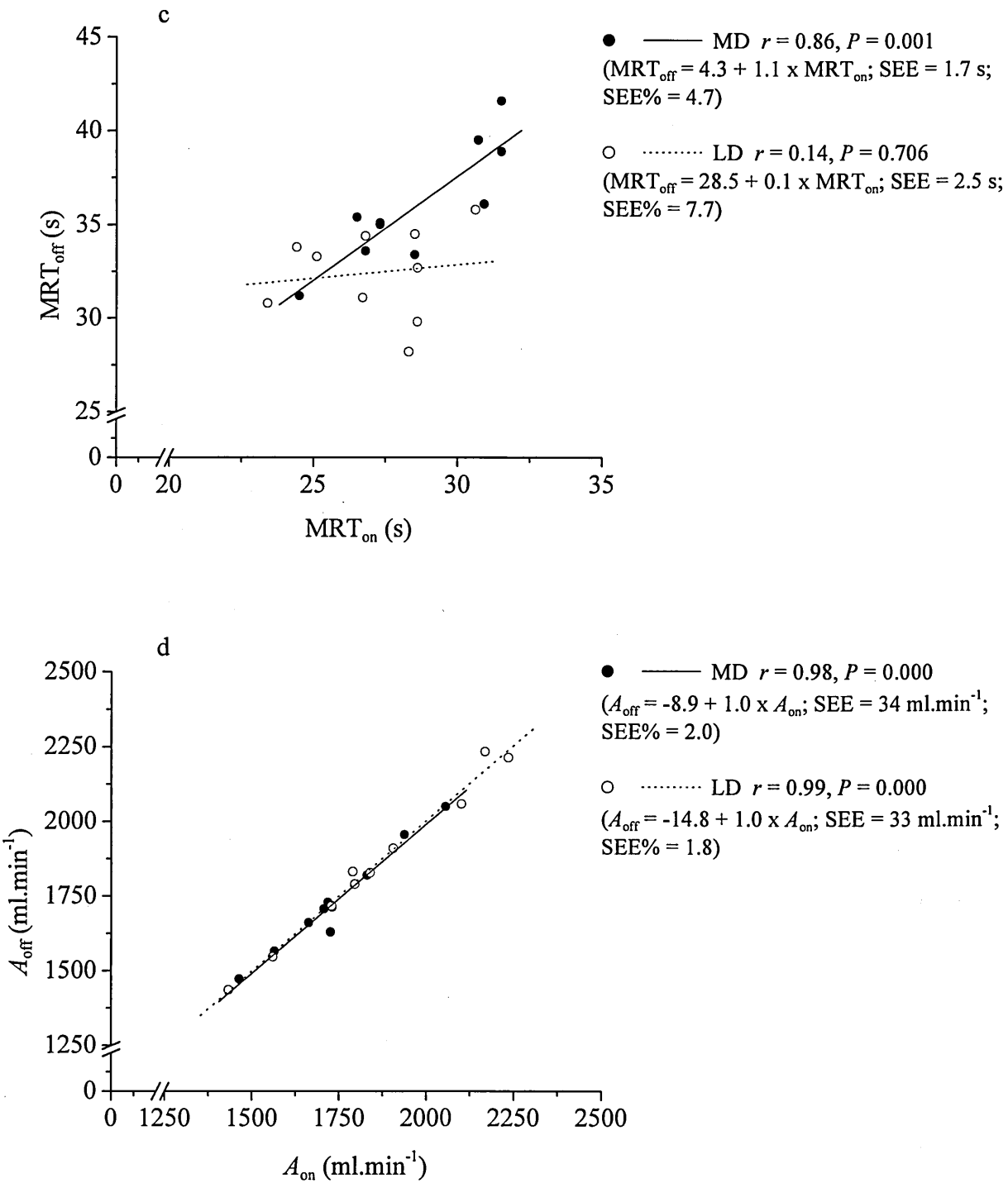


Figure 5.3 (a-d) Correlation's between on- and off-transient $\dot{V}O_2$ kinetic parameters for (a) δ, (b) τ, (c) MRT and (d) A for MD (n=10) and LD (n=10) runners.

5.4 Discussion

5.4.1 On-transient $\dot{V}O_2$ kinetics

The on-transient $\dot{V}O_2$ kinetics for MD, LD and combined runners are presented in Table 5.2. Specifically, τ_{on} is similar to previously reported values for moderate-intensity treadmill running (15.0 ± 2.0 s, Carter *et al.*, 2000a; 14.7 ± 2.8 s, Williams *et al.*, 2001). This was unexpected given the training status of the athletes used in this study. In an attempt to understand why there was such a similarity for on-transient $\dot{V}O_2$ kinetics, further investigations into other physiological characteristics of the participants were undertaken. This revealed that participants had a $\dot{V}O_{2\,max}$ of 50.7 ± 13.0 $ml \cdot kg^{-1} \cdot min^{-1}$ (Carter *et al.*, 2000a) and 56.6 ± 3.0 $ml \cdot kg^{-1} \cdot min^{-1}$ (Williams *et al.*, 2001) which are lower than the $\dot{V}O_{2\,max}$ of MD and LD runners reported here (Table 5.1). This suggests that phase II $\dot{V}O_2$ kinetics during moderate-intensity treadmill running is independent of $\dot{V}O_{2\,max}$. Collectively, these findings oppose the high correlation between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ found in previous studies involving untrained individuals (Chilibeck *et al.*, 1996; Fawkner *et al.*, 2002).

In support of the values obtained for MD and LD runners in this study, Carter *et al.* (2002) reported a mean τ_{on} of 12.7 ± 1.4 s for recreational active men and women with a $\dot{V}O_{2\,max}$ of 59.3 ± 10.0 $ml \cdot kg^{-1} \cdot min^{-1}$. This is almost identical to τ_{on} (12.5 ± 2.3 s) in this study for LD runners with similar $\dot{V}O_{2\,max}$ (Table 5.2). Clearly, further work to establish the relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ is warranted on the basis of these inconsistent findings.

Differences between the methods used here and in previous studies might account for the seemingly fast $\dot{V}O_2$ kinetics observed for recreationally active (Carter *et al.*, 2000a) and untrained individuals (Williams *et al.*, 2001). First, the transition to moderate-intensity exercise reported here was initiated from walking, whereas in other studies transitions were initiated from resting conditions (Carter *et al.*, 2000a; Williams *et al.*, 2001). Second, in this study, a mono-exponential model with time delay was used

(Whipp *et al.*, 1982), whereas Carter *et al.* (2000a) and Williams *et al.* (2001) used a higher order, two-component exponential model. Third, the daily use of the muscle groups might be a physiological explanation as to why $\dot{V}O_2$ kinetics are similar in recreationally active individuals and MD and LD runners. Chilibeck *et al.* (1997) reported that differences between $\dot{V}O_2$ kinetics in old and young individuals were not as pronounced when measured during plantar flexion exercise compared to treadmill exercise. This was attributed to the fact that both age groups use the plantar flexors in every day activities (i.e. walking). To some extent, a similar explanation might also apply to treadmill-based assessments of $\dot{V}O_2$ kinetics in younger, more active individuals since the muscles of the lower extremity are recruited during walking, jogging and running and play a significant part in most young individual's recreationally active lifestyles. Therefore, it might not be surprising that τ_{on} (and MRT_{on}) do not differ substantially between recreationally active individuals and MD and LD runners for treadmill exercise. Finally, a physiologic or genetic 'upper limit' (or ceiling) to the $\dot{V}O_2$ kinetic response at the onset of exercise cannot be ruled out. This would imply that no further speeding in τ_{on} is possible regardless of further training stimuli. Further research is required to investigate this possibility. Alternatively, the results of this study might differ from previous studies because of different protocols used during recovery from moderate-intensity exercise i.e. walking or standing. Clearly, further work is necessary to clarify the effect of different protocols on off-transient $\dot{V}O_2$ kinetics.

The results here differ from previous studies involving runners (deVries *et al.*, 1982; Powers *et al.*, 1985; Lake *et al.*, 1986). The most relevant study is that of Lake *et al.* (1986) who reported τ_{on} for runners during treadmill running. Most other studies involving runners have used cycle ergometry (deVries *et al.*, 1982; Powers *et al.*, 1985). However, Lake *et al.* (1986) reported a much longer τ_{on} [25.3 ± 8.7 s (EP) and 30.8 ± 7.3 s (MP)] than the present study. This was apparent despite their runners having a higher $\dot{V}O_{2\,max}$ (67.1 ± 5.6 $ml \cdot kg^{-1} \cdot min^{-1}$). In the study of Powers *et al.* (1985), runners had a similar $\dot{V}O_{2\,max}$ to the MD and LD runners in the present study (~59

$ml \cdot kg^{-1} \cdot min^{-1}$). However, Powers *et al.* (1985) reported a $\dot{V}O_2$ $t_{½}$ that ranged from 21.6 to 36.0 s. This is equivalent to a τ_{on} of ≅ 31.2 to 52 s which is considerably slower than those of the present study. Similarly, deVries *et al.* (1982) reported a mean $\dot{V}O_2$ $t_{½}$ of 27.4 s corresponding to a τ_{on} of ≅ 39.5 s for young endurance runners with similar $\dot{V}O_{2\,max}$. Collectively, these findings suggest that τ_{on} is independent of $\dot{V}O_{2\,max}$ in trained runners. This counters previous findings that $\dot{V}O_2$ kinetics ($\dot{V}O_2$ $t_{½}$) is proportional to $\dot{V}O_{2\,max}$ in runners (Powers *et al.*, 1985).

In early studies (deVries *et al.*, 1982; Powers *et al.*, 1985; Lake *et al.*, 1986) the time resolution of $\dot{V}O_2$ measures (every 10, 15 and 20 s respectively) might have prevented the true measurement of $\dot{V}O_2$ kinetics. Furthermore, kinetic parameters were estimated from a single transition. Potentially, the distorting effects of breath-by-breath noise might not have been sufficiently reduced and could have influenced parameter estimations (Lamarra *et al.*, 1987). To avoid this possibility and attenuate the effects of noise in the present study, a protocol was used that provided enough transitions (n=6) to allow reproducible estimations of the underlying $\dot{V}O_2$ response characteristics using exponential modelling techniques. Subsequently, the 95%CI for estimations of τ_{on} was 1.1 ± 0.3 s (MD) and 0.9 ± 0.3 s (LD) which is more sensitive than previously reported in other studies (Chilibeck *et al.*, 1998; Rossiter *et al.*, 1999). Such a high degree of confidence in kinetic parameter estimations is essential if a true and meaningful insight into the effects of training or comparisons between populations is to occur.

In this study, there was a tendency for τ_{on} and MRT_{on} to be shorter in LD runners than in MD runners, although these differences were not significant. At the outset, given that both MD and LD runners demonstrated similar $\dot{V}O_{2\,max}$ and V_T, this finding is acceptable. However, the lack of difference between MD and LD runners could potentially be attributed to the inter-subject variability which resulted in an inflated SD of the mean response (i.e. τ_{on} range for MD = 9.8 to 19.2 s; LD = 9.0 to 16.4 s). Closer inspection of the data revealed that one MD runner (subject 4) had a τ_{on} of 9.8 s which

was considerably shorter than that of all other MD runners and most LD runners. Given the narrow 95%CI for kinetic parameter estimations, this particular τ_{on} is likely to be truly representative of this individual underlying $\dot{V}O_2$ response. To ensure that this τ_{on} did not adversely influence the results, a re-analysis of the data was performed with the participants data removed. However, re-analysis confirmed the similarity between MD and LD runners with respect to τ_{on} ($P > 0.05$).

There are several possible explanations for the faster $\dot{V}O_2$ kinetics in endurance-trained runners compared to recreationally active individuals (Carter *et al.*, 2000a) and LD runners compared to MD runners. One relates to differences in muscle fibre type. Type I fibres (slow twitch) are known to be rich in mitochondria (Saltin *et al.*, 1977) which might result in faster $\dot{V}O_2$ kinetics. This supports the linear first-order model of respiratory control which suggests that mitochondrial resistance - a function of the number and properties of mitochondria - has an important role in determining the rate of oxidative phosphorylation (Meyer, 1988). Therefore, if endurance training causes an increase in the number and function of the mitochondria, then τ_{on} should be shorter.

Adjustments in muscle [PCr] and $\dot{Q}O_2$ are faster for isolated muscles with predominantly Type I fibres, compared to muscles with Type II fibres (Crow and Kushmerick, 1982; Kushmerick *et al.*, 1992). In athletes with a predominance of Type I fibres, presumably LD runners (Costill *et al.*, 1976b; Saltin and Gollnick, 1983), there could be a tendency for τ_{on} to be shorter than that observed in MD runners. Opposing this hypothesis, it has been demonstrated that there is no relationship between the percentage of Type I fibres and $\dot{V}O_2$ kinetics at the onset of heavy-intensity exercise (Barstow *et al.*, 1996). However, correlations between the fibre type percentage of an individual muscle (e.g. gastrocnemius) and measures of $\dot{V}O_2$ kinetics during cycling could be misleading because cycling requires the recruitment of several different muscles. These might display heterogeneity with respect to their individual $\dot{V}O_2$ kinetics which could prevent the relationship between muscle fibre type and $\dot{V}O_2$

kinetics from being identified. Potentially, a relationship could exist between τ_{on} and the percentage of Type I fibres for moderate-intensity exercise in an individual muscle, although research to date has not been conducted to investigate this possibility.

It has been proposed that $\dot{V}O_2$ kinetics are determined by intrinsic metabolic factors governing the utilisation of O_2 in exercising muscle (Mahler, 1980; Whipp and Mahler, 1980; Grassi *et al.*, 1996). In consideration of possible determining mechanisms, $\dot{V}O_2$ kinetics at the onset of moderate-intensity exercise in MD and LD runners could be limited by mitochondrial enzyme activation and/or substrate provision within the muscle. In such circumstances, there would be a tendency for biochemical reactions that govern mitochondrial O_2 utilisation to be speeded in the trained compared to the untrained. Evidence to support a peripheral mechanism within the muscle has been demonstrated in a study of specific muscle training where $\dot{V}O_2$ kinetics in trained muscles were significantly faster than the $\dot{V}O_2$ kinetics in untrained muscle (Cerretelli *et al.*, 1979). However, an enhanced and more uniform blood flow in trained muscles, with decreased diffusion distance for O_2, could also result in a faster delivery of O_2 and might be responsible for faster $\dot{V}O_2$ kinetics in runners compared to the untrained. In young subjects, skeletal muscle capillarity, capillary-to-fibre ratio and capillary density have all been shown to increase after endurance training (Ingjer, 1979). Similarly, faster $\dot{V}O_2$ kinetics in the early stages of endurance training (after 4 days) have been attributed to increased blood flow and not mitochondrial potential, since oxidative enzyme activity was unchanged (Phillips *et al.*, 1995). However, further acceleration of $\dot{V}O_2$ kinetics after 30 days of training was accompanied by an increase CS activity. This suggests that in the early stages of endurance training increased blood flow, and hence increased O_2 delivery, might be the primary mechanism that results in faster $\dot{V}O_2$ kinetics. However, once this early adaptation takes place, mechanism(s) within the exercising muscle predominate and determine $\dot{V}O_2$ kinetics.

The gain term ($ml \cdot kg^{-1} \cdot km^{-1}$) used in this and previous $\dot{V}O_2$ kinetic studies (Williams *et al.*, 2001; Carter *et al.*, 2002) is analogous to C_r as described by Margaria *et al.* (1963). The C_r has been found to be ~180 ml $O_2 \cdot kg^{-1} \cdot km^{-1}$ in MD (Lacour *et al.*, 1990) and LD runners (di Prampero *et al.*, 1986). In the present study, the mean gain for MD runners (177.1 ± 9.5 $ml \cdot kg^{-1} \cdot min^{-1}$) was consistent with this. The gain for LD runners (171.7 ± 9.4 $ml \cdot kg^{-1} \cdot min^{-1}$) was lower and is indicative of superior RE. In addition, HR responses during walking at 4 $km \cdot h^{-1}$ ($HR_{(b)}$, $P = 0.004$) and running at 80%V_T ($HR_{(m)}$, $P = 0.005$) were also lower in LD runners than in MD runners. This finding can not be attributed to differences in running speeds between the groups because the mean speed during walking (4 $km \cdot h^{-1}$) and running (80%V_T) was almost identical (Table 5.2). Furthermore, even when expressed as a percentage of HR_{max} achieved during the incremental test to volitional exhaustion, HR was still lower in the LD runners ($HR_{(b)}$ = 45 ± 7 vs. 38 ± 4%; $HR_{(m)}$ = 75 ± 4 vs. 71 ± 4%, Table 5.2).

5.4.2 Off-transient $\dot{V}O_2$ kinetics

Although τ_{on} for MD and LD runners was only marginally faster than that previously reported (Carter *et al.*, 2000a, b; Williams *et al.*, 2001), in contrast, the off-transient $\dot{V}O_2$ kinetics (τ_{off} and MRT_{off}) were considerably faster. The mean (± SD) τ_{off} in this study was 27.1 ± 3.0 s (MD) and 24.1 ± 2.3 s (LD) which was substantially less than that previously reported for untrained individuals during treadmill running (39.9 ± 3.0 s; Carter *et al.*, 2000a) and cycling (36.8 ± 1.9 s; Paterson and Whipp, 1991). After endurance training, Phillips *et al.* (1995) reported a mean (±SD) τ_{off} and MRT_{off} of 25.0 ± 1.8 s and 30.6 ± 0.9 s respectively for cycle exercise, which are closer to the present values for treadmill running (Table 5.3). The larger difference between off-transient $\dot{V}O_2$ kinetics in MD and LD runners compared to untrained individuals reflects the difference in training status and hence, the physiological status of the muscle. These findings suggest that the recovery process, primarily involving the re-synthesis of [PCr], is faster in MD and LD runners, probably as a result of regular training.

In support for the use of off-transient $\dot{V}O_2$ kinetics as a measure of physiological status, Chilibeck *et al.* (1997) reported a much stronger relationship between capillarisation per-fibre-area and off-transient $\dot{V}O_2$ kinetics than that observed for on-transient $\dot{V}O_2$ kinetics. The suggested mechanism to explain this relationship was that O_2 delivery might have a greater influence on $\dot{V}O_2$ adjustment during recovery than $\dot{V}O_2$ adjustment at the onset of exercise as a result of a shorter diffusion distance. This is supported by Idstrom *et al.* (1985) who found that the rate of [PCr] re-synthesis after contractions of perfused rat hind-limb was related to O_2 supply through the perfusate, although the rate of [PCr] degradation at the start the onset of exercise was not. The findings of Idstrom *et al.* (1985) require the assumption that [PCr] kinetics reflect the kinetics of $\dot{Q}O_2$. Strong evidence supporting this assumption in humans has been reported (Barstow *et al.*, 1994; Rossiter *et al.*, 1999).

In the present study, MD and LD runners could be statistically differentiated on the basis of their off-transient $\dot{V}O_2$ kinetics. Both τ_{off} and MRT_{off} were shorter (i.e. faster $\dot{V}O_2$ kinetics) in LD runners (Table 5.3). This suggests that not only is the recovery process accelerated in trained individuals when compared to untrained individuals (Hagberg *et al.*, 1980; Phillips *et al.*, 1995), but that the recovery process is also accelerated within groups of trained runners (i.e. MD vs. LD). Differentiation between untrained and LD runners (Yoshida and Watari, 1993) and between MD and LD runners (McCully *et al.*, 1992) have been shown with respect to [PCr] recovery kinetics. The rate constant for [PCr] recovery has been directly interpreted as an index of muscle oxidative capacity (McCully *et al.*, 1992; Paganini *et al.*, 1997). This would suggest a greater muscle oxidative capacity in LD runners.

Differences between the $\dot{V}O_2$ and [PCr] kinetics in MD and LD runners might be attributable to different approaches to training with respect to volume, frequency and intensity. In support, the volume of training was greater for LD runners than MD

runners. Approaches to training in running are likely to be largely influenced by the aerobic and anaerobic energy contributions to the overall performance.

Similar to the on-transient, differences in off-transient $\dot{V}O_2$ kinetics might also reflect an increased percentage of Type I fibres within the muscle and/or enhanced mitochondrial enzyme activity in LD runners. In support of the latter, a linear relationship between [PCr] recovery and CS activity has been demonstrated (McCully *et al.*, 1993; Paganini *et al.*, 1997). Oxidative enzyme concentrations are likely to be heavily influenced by the volume and/or intensity of training. It is expected that MD runners complete more anaerobic and speed-orientated training than a typical LD runner and thus there is a greater reliance on less efficient fast twitch (Type IIa and IIx) muscle fibres. As a result of these differences, the percentage of slow twitch (Type I) fibres and oxidative enzymes is lower in MD runners when compared to LD runners (Costill *et al.*, 1976b; Saltin and Gollnick, 1983) and could be responsible for slower $\dot{V}O_2$ off-transient kinetics in MD runners. To date, no research has considered the relationship between off-transient $\dot{V}O_2$ kinetics and fibre type distribution in trained individuals.

5.4.3 Symmetry between on- and off-transients

Comparisons between the on- and off-transients revealed a distinct asymmetry showing that τ_{on} (MD = 14.2 ± 3.1 s; LD = 12.5 ± 2.3 s) was faster than τ_{off} (MD = 27.1 ± 3.0 s; LD = 24.1 ± 2.3 s). In most instances τ_{off} was two-fold greater than the value observed for τ_{on}. Likewise, MRT_{on} and MRT_{off} also differed in both MD and LD runners (P <0.001; Tables 5.2 and 5.3 respectively). Asymmetry between transients is a significant finding because it implies that $\dot{V}O_2$ during treadmill running below the V_T does not conform to a dynamically linear system. Dynamic linearity is a characteristic of a first-order system and as such, τ_{on} (or τ_{off}) should be independent of any prior conditions, the intensity of exercise and hence the amplitude of response (Lamarra, 1990). This finding of asymmetry is in agreement with previous studies conducted in the moderate-intensity domain using treadmill (Carter *et al.*, 2000a), cycle (Linnarsson, 1974; Hughson *et al.*,

1988; Scheuermann *et al.*, 1998; Carter *et al.*, 2000a; Rossiter *et al.*, 2002) and knee-extension (Rossiter *et al.*, 2002) exercise.

One explanation for the asymmetry between transients for $\dot{V}O_2$ kinetics can be implied from a quantitative analysis of the time course of [PCr] degradation and recovery kinetics. Kushmerick (1998) has shown, via computer modelling, that the full expression of creatine kinase (forward and reverse flux) and differing processes during the imposed increased ATPase activity in the breakdown and recovery phases, results in asymmetry between [PCr] on- and off-transients. Given the kinetic similarities between [PCr] and $\dot{V}O_2$ (Rossiter *et al.*, 1999; 2002), this is anticipated to cause an asymmetry between $\dot{V}O_2$ on- and off-transients as was observed in the present study. However, the suggestions of Kushmerick (1998) still need to be explored and demonstrated experimentally in humans.

Although symmetry between on- and off-transient $\dot{V}O_2$ responses was not observed in MD and LD runners, there was a relationship between kinetic parameters during the on- and off-transients, especially A_{on} and A_{off} (Figure 5.3d). With respect to both τ_{on} and τ_{off} and MRT_{on} and MRT_{off}, there was also a high correlation for MD runners (Figure 5.3b), but not for LD runners. However, when MD and LD runners were combined [to represent a larger sample (n=20) of endurance-trained runners], a relationship was observed between τ_{on} and τ_{off} (r = 0.71, P <0.001). A correlation was also found between MRT_{on} and MRT_{off} for combined runners (r = 0.62, P = 0.004) suggesting that overall, runners with fast on-transient $\dot{V}O_2$ kinetics also display fast off-transient kinetics. This finding supports previous studies reporting a high correlation between on- and off-transient $\dot{V}O_2$ kinetics (Hagberg *et al.*, 1980; Yoshida and Whipp, 1994). However, no correlation's between τ_{on} and τ_{off} or MRT_{on} and MRT_{off} were observed in LD runners in this study (Figures 5.3 b and c). This might be attributable to the homogeneity (narrower range) of values for LD runners compared to MD runners.

5.5 Conclusion

This study has characterised $\dot{V}O_2$ kinetics during the on- and off-transients in MD and LD runners using treadmill ergometry. The on-transient $\dot{V}O_2$ kinetics in MD and LD runners were faster than those previously reported for recreationally active individuals using this form of exercise. This was attributed to the higher fitness and endurance-trained state of the participants of the present study. However, despite the differences in the physiological characteristics of the participants (i.e. $\dot{V}O_{2\,max}$ and V_T) and especially their training histories, the magnitude of the differences with respect to $\dot{V}O_2$ on-transient kinetics was surprisingly small. Conversely, the magnitude of differences in the off-transient $\dot{V}O_2$ kinetics was larger. This suggests that off-transient $\dot{V}O_2$ kinetics give an improved indication of an individual's overall aerobic physiological condition and/or training status. The on- and off-transient $\dot{V}O_2$ kinetics are not symmetrical in MD and LD runners; the off-transient $\dot{V}O_2$ kinetics (τ_{off} and MRT_{off}) being consistently slower than the on-transient. However, the on- and off-transients are related.

CHAPTER 6

Inter-relationships among aerobic parameters in middle- and long-distance runners

6.1 Introduction

Endurance training results in several physiological adaptations at a cellular level such as an increased number of mitochondria and increased oxidative enzyme activity (Gollnick *et al.*, 1972; Costill *et al.*, 1976a; Saltin and Gollnick, 1983). Collectively, these are likely to speed $\dot{V}O_2$ kinetics at the onset of moderate-intensity exercise. However, the influence of cellular changes on $\dot{V}O_{2\,max}$, V_T and RE might be less pronounced which could potentially dissociate these measures. The temporal dissociation between changes in mitochondrial enzyme activity and $\dot{V}O_{2\,max}$ after endurance training (Henriksson and Reitman, 1977) suggests that there might be poor correlation between measures of peripheral (i.e. $\dot{V}O_2$ kinetics) and central (i.e. $\dot{V}O_{2\,max}$) physiological status.

Studies that have investigated the relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ in untrained individuals have yielded conflicting results (Chilibeck *et al.*, 1996; Bell *et al.*, 1999; Whipp *et al.*, 2001; Fawkner *et al.*, 2002). For example, it has been demonstrated that individuals with a high $\dot{V}O_{2\,max}$ display fast $\dot{V}O_2$ kinetics (Weltman and Katch, 1976; Weltman *et al.*, 1978; Chilibeck *et al.*, 1996). The highest correlation (r = -0.85, P <0.001) reported between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ was in young adults (Chilibeck *et al.*, 1996). Other studies, however, reported no correlation (Bell *et al.*, 1999; Barstow *et al.*, 2000) suggesting that mechanisms determining each measure are independent.

In endurance-trained runners, studies of the relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ are less well documented and have also produced inconsistent findings (Powers *et al.*, 1985; Lake *et al.*, 1986). Powers *et al.* (1985) reported a negative correlation between $\dot{V}O_2\,t_{½}$ and $\dot{V}O_{2\,max}$ (r = -0.80, P <0.05) in LD runners, whereas Lake *et al.* (1986) reported no correlation between τ_{on} and $\dot{V}O_{2\,max}$ (r = 0.19, P >0.05). These

contrasting findings could be attributable to differences in: 1) mode of exercise (cycle vs. treadmill); 2) methods of expressing $\dot{V}O_2$ kinetics ($\dot{V}O_2$ $t_{½}$ vs. τ_{on}) and 3) mean ± SD $\dot{V}O_{2\,max}$ [67.1 ± 5.6 ml·kg^{-1}·min^{-1}, Lake *et al.* (1986); 58.0 ± 2.6 ml·kg^{-1}·min^{-1}, Powers *et al.* (1985)]. With respect to the latter, a heterogeneous sample, such as that of Lake *et al.* (1986), might display a stronger correlation due to a greater spread of data. However, the stronger relationship was observed in the more homogeneous data (as indicated by a lower SD) of Powers *et al.* (1985). Unfortunately, the relationship between $\dot{V}O_{2\,max}$ and off-transient $\dot{V}O_2$ kinetics from moderate-intensity exercise was not considered in any of these studies and has received minimal consideration elsewhere. This is clearly an area for further investigation.

The $\dot{V}O_2$ at V_T has also been shown to be associated with on-transient $\dot{V}O_2$ kinetics (Weltman *et al.*, 1978; Chilibeck *et al*, 1996; Whipp *et al.*, 2001). Specifically, Chilibeck *et al.* (1996) identified a relationship between τ_{on} and V_T (r = -0.62, P <0.02) in untrained participants (n=16), although this relationship was less than that observed for $\dot{V}O_{2\,max}$ (r = -0.85, P <0.001). With regards to the relationship between off-transient $\dot{V}O_2$ kinetics and V_T, although no correlation analysis was performed, Weltman *et al.* (1978) reported that the off-transient $\dot{V}O_2$ $t_{½}$ (34.6 s vs. 40.3 s) was shorter in individuals with a high V_T. To date, no other study has assessed the relationship between off-transient $\dot{V}O_2$ kinetics and V_T.

Most studies that have assessed the relationship between $\dot{V}O_2$ kinetics, $\dot{V}O_{2\,max}$ and V_T have expressed $\dot{V}O_2$ relative to BM as a ratio standard, i.e. ml·kg^{-1}·min^{-1}. However, according to principles of allometry, $\dot{V}O_2$ does not increase linearly with BM (Schmidt-Nielson, 1984). Specifically, it has been shown that sub-maximal $\dot{V}O_2$ and $\dot{V}O_{2\,max}$ do not increase linearly with BM in runners (Bergh *et al.*, 1991), thus confirming the need to adjust for differences in BM in this population by ways other than the ratio standard. Therefore, if $\dot{V}O_2$ is inappropriately expressed in MD and LD runners for treadmill running, this might distort and prevent a meaningful assessment of the relationship

between pulmonary gas-exchange measures. The way in which $\dot{V}O_2$ is expressed could affect apparent relationships between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ and account for a lack of observed association in previous studies involving runners (e.g. Lake *et al.*, 1986).

Fawkner *et al.* (2002) recently assessed the relationship between $\dot{V}O_2$ kinetics and allometrically scaled $\dot{V}O_{2\,max}$ in men and women (n=25) during moderate-intensity cycling. It was found that there was a relationship between $\dot{V}O_{2\,peak}$ and τ_{on} (r = -0.62, -0.81 and -0.82, P <0.05) for the men when $\dot{V}O_{2\,max}$ was expressed in absolute terms ($l \cdot min^{-1}$), as a ratio standard ($ml \cdot kg^{-1} \cdot min^{-1}$) or as a power-function ratio ($ml \cdot kg^{-0.89} \cdot min^{-1}$) respectively. However, expressing $\dot{V}O_2$ as a power-function ratio of BM did not substantially improve the relationship between τ_{on} and $\dot{V}O_{2\,max}$ compared to the ratio standard. Thus, accounting for differences in BM did not affect the relationship between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics in this population, probably because the identified exponent (mean ± SE; 0.89 ± 0.17) was close to the ratio standard, especially when the SE was considered. Regardless of the method for expressing $\dot{V}O_2$, there was no relationship between τ_{on} and $\dot{V}O_{2\,max}$ for the women. Although an explanation for such gender differences is difficult to ascertain, this finding highlighted the need to investigate individual groups based on gender rather than on adults alone. However, these findings could be specific to the mode of exercise (cycle ergometry which is non weight-bearing) and the fitness of participants under investigation.

To date, the relationship between $\dot{V}O_2$ kinetics and RE has not been reported. The RE is a measure often used to assess MD and LD runners (Svedenhag and Sjödin, 1984; Pate *et al.*, 1992) and has been shown to be related to running performance (Conley and Krahenbuhl, 1980). However, if a relationship exists between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics in runners, as shown previously (Powers *et al.*, 1985), then it might be expected that there is an opposite relationship between RE and $\dot{V}O_2$ kinetics. This is because a negative relationship exists between $\dot{V}O_{2\,max}$ and RE (Pate *et al.*, 1992; Morgan and Daniels, 1994) which implies that a runner with a high $\dot{V}O_{2\,max}$ is likely to have poor

RE compared to a runner with a low $\dot{V}O_{2\,max}$ who has good RE. If these relationships hold true for the present data then a runner who has good RE (i.e. low $\dot{V}O_2$ for a given sub-maximal running speed) should have slower $\dot{V}O_2$ kinetics compared to a runner with poor RE (i.e. high $\dot{V}O_2$ for a given sub-maximal running speed).

6.1.1 Relationships between measures: Correlation analysis

Correlation analysis is widely used in sport and exercise science research to explore relationships between measures of physiological function (e.g. Fawkner *et al.*, 2002) or between physiologic function and sports performance (e.g. Grant *et al.*, 1997). This approach might facilitate the identification of a common factor(s) that influences or determines two physiological measures. However, it is important to understand what factors determine and/or influence correlation analyses to aid interpretation.

Specifically, correlation analysis is a statistical technique used to determine whether two variables are interdependent, or co-vary, that is vary together (Sokal and Rohlf, 1995). When the data are parametric, the Pearson product-moment correlation coefficient (r) is commonly used. This method assesses the extent to which the *direction* and *size* of the deviations from the mean in one variable are related to the direction and size of deviations from the mean in another variable (Vincent, 1995). With respect to the interpretation of r, if the value is positive (i.e. $0>r\leq1$), the variables are said to be positively correlated such that as one variable increases the other increases at a linearly proportional rate; if it is negative ($-1\geq r<0$), they are negatively correlated such that as one variable increases the other decreases at a linearly proportional rate. The interpretation of the magnitude of r has been considered by Cohen and Holliday (1982) to be: 1) very low (0.00 to 0.19); 2) low (0.20 to 0.39); 3) modest (0.40 to 0.69); 4) high (0.70 to 0.89) and 5) very high (>0.90). However, the magnitude of r is influenced by the ranges of the two variables under consideration. Large ranges (heterogeneity) in one or both measures can produce high r values, where as small ranges (homogeneity) can depress r. This scenario has been highlighted by Sale (1991) who demonstrated how

the precision of the relationship between measures can be misrepresented when the range of values is manipulated. In addition to the variability in the measures, the value and significance of r can be influenced by the size of the sample (n). The number of pairs of scores influences the degrees of freedom (df) which represents the number of values that are free to vary when the sum of the variables is set (Vincent, 1995). When n is small (e.g. 5 pairs of scores), it is possible that spuriously high r values can be obtained by chance (Vincent, 1995). Furthermore, when n is small r must be high to be significant (Vincent, 1995). A larger n provides the researcher with more confidence that r is real and does not occur by chance. However, a large n can result in a small r being significant.

The correlation coefficient r can also be influenced by anomalous data that is not truly representative of the sample. Such data can exert considerable leverage and yield less meaningful high and low r values. For this reason it is important to qualitatively inspect the data and consider excluding extreme outliers. Since an assumption of correlation analyses is that the variables are linearly proportional to each other, a pre-analysis scatter plot can be used to identify any possible outliers. These can then be removed, objectively, on the basis of statistical criteria using Mahalanobis and Cook's distance statistics (Tabachnik and Fidell, 1996).

Prior knowledge of the reproducibility and day-to-day variability of the measures in question is advantageous to rule out this variability, which could have a substantial influence on r and its significance. In any instance it is important to realise that a significant relationship between two measures does not prove causation; it only shows that a non-chance relationship exists. However, reasoned logic and findings from previous research may point to a possible reason(s) for the magnitude, direction and significance of relationships identified.

6.1.2 Aim of study

The purpose of this study was to investigate the relationship between measures of on- and off-transient $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$, V_T and RE in MD and LD runners.

6.2 Participants and methods

6.2.1 Participants

Sixteen male MD (800/1500 m) and 16 male LD (5000/10000 m) runners provided written informed consent and participated. Participants were recruited from athletic clubs in the North of England. Participants' age, anthropometric and training characteristics are presented in Table 6.1. Prior to participation in the study each athlete completed a medical screening questionnaire (Appendix 6).

Table 6.1 Age, anthropometric and training characteristics of participants. Values are mean ± SD.

Measure	MD (n=16)	LD (n=16)	Combined (n=32)
Age (years)	21.3 ± 5.5	25.0 ± 4.2	23.2 ± 5.1
Stature (cm)	176.8 ± 6.8	180.3 ± 7.0	178.6 ± 7.0
BM (kg)	66.6 ± 5.8	69.9 ± 8.4	68.2 ± 7.3
Volume of training ($km \cdot wk^{-1}$)	43.5 ± 15.6	66.6 ± 14.4	55.1 ± 18.9

6.2.2 Experimental Design

Participants visited the laboratory for physiological testing on two occasions within a seven-day period. Each test was separated by at least 48 hours and was performed at approximately the same time of day. Physiological testing during the first visit to the laboratory involved the measurement of RE, V_T and $\dot{V}O_{2\,max}$. Visit 2 involved a square-wave exercise protocol to determine on- and off-transient $\dot{V}O_2$ kinetics. Throughout the duration of the testing period, participants were requested to maintain their usual dietary intake and to abstain from participating in heavy training and consuming alcohol and/or caffeine in the 48 hours preceding each test.

6.2.3 Experimental protocols

Each participant completed: 1) a series of 4-6 four-min bouts of running with speed increasing by 1 $km \cdot h^{-1}$ every stage for the determination of RE (see Chapter 3, Section 3.2.6); 2) an incremental exercise test to volitional exhaustion for the determination of V_T and $\dot{V}O_{2\,max}$ (see Chapter 3, Section 3.2.5) and 3) a square-wave protocol consisting of alternating six-min bouts of walking (4 $km \cdot h^{-1}$) and running (speed requiring 80%V_T) to determine on- and off-transient $\dot{V}O_2$ kinetics (see Chapter 3, Section 3.2.7). Throughout each test, pulmonary gas-exchange was measured breath-by-breath.

6.2.4 Data analysis

Breath-by-breath measures of $\dot{V}O_2$ obtained during the assessment of RE, V_T, $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics were analysed in accordance with procedures outlined in Chapter 3, Sections 3.2.5.1 to 3.2.7.1.

6.2.5 Statistical analyses

Descriptive statistics (mean ± SD) were calculated for each physiological measure during each test. The $\dot{V}O_{2\,max}$, V_T and RE were expressed in absolute terms, relative to BM in standard ratio terms ($ml \cdot kg^{-1} \cdot min^{-1}$) and as three separate power-function ratios of BM ($ml \cdot kg^{-b} \cdot min^{-1}$; where b = 0.67, 0.75 and 0.86) derived from previous research. Furthermore, the individual b exponent for MD and LD runners representing the gradient of the log-log relationship between $\dot{V}O_{2\,max}$, V_T, RE and BM was determined empirically using log-linear ANCOVA. Relationships between measures were determined using Pearson's product moment correlation coefficient and were performed on MD (n=16), LD (n=16) and combined MD and LD runners (n=32). Assumptions about the normality of distributions of data were checked by means of the Shapiro-Wilk and Kolmogorov-Smirnov statistics. Statistical significance was set at P <0.05.

6.3 Results

The participants' $\dot{V}O_{2\,max}$, V_T and RE are presented in Table 6.2. Log-log transformations and ANCOVA showed that a common *b* exponent could be used for MD and LD runners since the slope and estimated marginal means were not different ($P > 0.05$). The *b* exponents for $\dot{V}O_{2\,max}$, V_T and RE were 0.68 ± 0.15, 0.54 ± 0.14 and 0.74 ± 0.10 respectively; mean ± SE. Consideration of the leverage statistics (Mahalanobis and Cooks' distances) suggested that no outlying data were influencing the regression models and that the *b* exponent for BM when expressing $\dot{V}O_{2\,max}$, V_T and RE were truly representative of the data. In Table 6.2, all measures of $\dot{V}O_2$ have been expressed using the four BM exponents. However, Figures illustrating the relationship between $\dot{V}O_{2\,max}$, V_T and RE with $\dot{V}O_2$ kinetics (Figures 6.1 - 6.3), express $\dot{V}O_2$ measures using the *b* exponent for BM determined empirically using data from the current sample.

Table 6.2 Physiological characteristics of MD, LD and combined runners.

Measure	MD (n=16)	LD (n=16)	Combined (n=32)
$\dot{V}O_{2\,max}$ ($ml \cdot min^{-1}$)	3968 ± 300	4193 ± 583	4069 ± 477
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	59.8 ± 4.7	60.2 ± 5.8	60.0 ± 5.2
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	239 ± 16	244 ± 6	241 ± 4
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.75} \cdot min^{-1}$)	171 ± 12	174 ± 4	172 ± 14
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.86} \cdot min^{-1}$)	107 ± 8	109 ± 10	108 ± 9
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-0.68} \cdot min^{-1}$)$^{\$}$	229 ± 15	234 ± 23	231 ± 19
V_T ($ml \cdot min^{-1}$)	3261 ± 241	3435 ± 387	3349 ± 329
V_T ($ml \cdot kg^{-1} \cdot min^{-1}$)	49.2 ± 4.6	49.4 ± 4.4	49.3 ± 4.4
V_T ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	196 ± 15	200 ± 16	198 ± 15
V_T ($ml \cdot kg^{-0.75} \cdot min^{-1}$)	140 ± 11	142 ± 11	141 ± 11
V_T ($ml \cdot kg^{-0.54} \cdot min^{-1}$)$^{\$}$	340 ± 25	349 ± 28	344 ± 27
V_T % $\dot{V}O_{2\,max}$	82.3 ± 3.5	82.3 ± 4.4	82.3 ± 3.9
RE at 16 $km \cdot h^{-1}$ ($ml \cdot min^{-1}$)	3568 ± 270	3605 ± 404	3587 ± 341
RE at 16 $km \cdot h^{-1}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	53.9 ± 3.8	51.7 ± 2.9	52.8 ± 3.5
RE at 16 $km \cdot h^{-1}$ ($ml \cdot kg^{-0.67} \cdot min^{-1}$)	215 ± 13	210 ± 12	212 ± 12
RE at 16 $km \cdot h^{-1}$ ($ml \cdot kg^{-0.75} \cdot min^{-1}$)	154 ± 9	149 ± 8	151 ± 9
RE at 16 $km \cdot h^{-1}$ ($ml \cdot kg^{-0.74} \cdot min^{-1}$)$^{\$}$	162 ± 10	158 ± 9	160 ± 9

Values are mean ± SD; $^{\$}$Empirically derived from present data.

Parameter estimations of the on- and off-transient $\dot{V}O_2$ kinetics are presented in Tables 6.3 and 6.4 respectively. The 95% CI associated with determinations of τ_{on} (Lamarra *et al.*, 1987) from six square-wave transitions was 1.0 ± 0.3 s.

Table 6.3 Measures of the $\dot{V}O_2$ kinetic response at the onset of moderate-intensity exercise in MD, LD and combined runners. Values are mean ± SD.

Measure	MD (n=16)	LD (n=16)	Combined (n=32)
Running speed ($km \cdot h^{-1}$)	11.4 ± 0.9	12.0 ± 0.8	11.7 ± 0.9
Resting $\dot{V}O_2$ ($ml \cdot min^{-1}$)	395 ± 33	389 ± 40	392 ± 36
$\dot{V}O_{2\,(b)}$ ($ml \cdot min^{-1}$)	903 ± 71	904 ± 98	904 ± 84
A_{on} ($ml \cdot min^{-1}$)	1746 ± 194	1832 ± 281	1789 ± 241
$\dot{V}O_{2\,(m)}$ ($ml \cdot min^{-1}$)	2649 ± 244	2736 ± 355	2692 ± 303
δ_{on} (s)	13.1 ± 2.5	15.4 ± 1.8	14.3 ± 2.4
τ_{on} (s)	16.4 ± 4.1	12.3 ± 2.2	14.4 ± 3.8
MRT_{on} (s)	29.5 ± 3.0	27.8 ± 2.4	28.6 ± 2.8

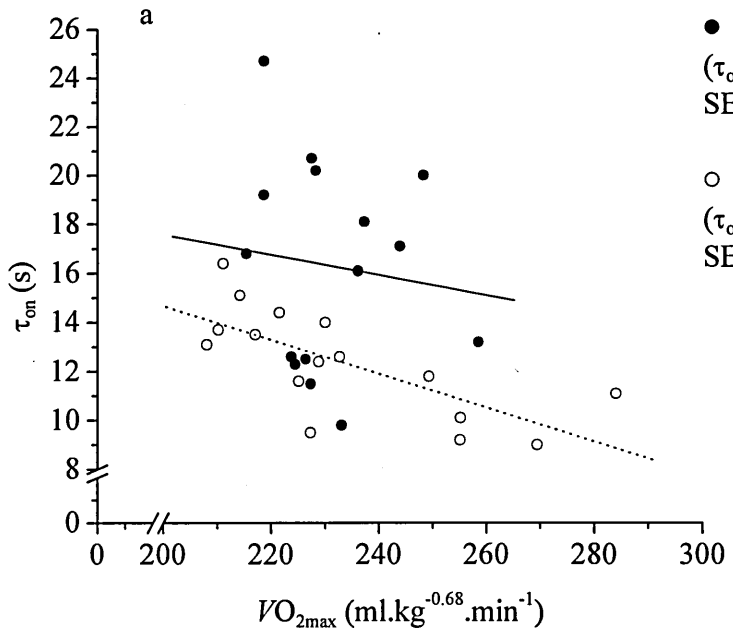
Table 6.4 Measures of the $\dot{V}O_2$ response during recovery from moderate-intensity exercise in MD, LD and combined runners. Values are mean ± SD.

Measure	MD (n=16)	LD (n=16)	Combined (n=32)
$\dot{V}O_{2\,(m)}$ ($ml \cdot min^{-1}$)	2658 ± 254	2732 ± 357	2695 ± 307
A_{off} ($ml \cdot min^{-1}$)	1697 ± 298	1831 ± 284	1788 ± 244
$\dot{V}O_{2\,(b)}$ ($ml \cdot min^{-1}$)	912 ± 72	901 ± 106	907 ± 90
δ_{off} (s)	9.1 ± 2.0	8.6 ± 2.7	8.9 ± 2.3
τ_{off} (s)	26.9 ± 3.2	24.3 ± 2.5	25.7 ± 3.0
MRT_{off} (s)	36.0 ± 3.5	33.1 ± 2.4	34.6 ± 3.3

6.3.2 Relationships with $\dot{V}O_{2\,max}$

When MD and LD runners were considered together (n=32), a relationship (r = -0.40, P = 0.05) was identified between τ_{on} and $\dot{V}O_{2\,max}$ regardless of the method of expressing $\dot{V}O_{2\,max}$. Similar relationships [r = -0.37 (P = 0.038) to r = -0.42 (P = 0.017)] existed between MRT_{on} and $\dot{V}O_{2\,max}$. There were no correlations between τ_{off} and $\dot{V}O_{2\,max}$ (P

>0.05). However, MRT_{off} correlated with $\dot{V}O_{2\,max}$ when expressed relative to BM (r = -0.35, P = 0.049). For MD runners (n=16), there were no relationships between $\dot{V}O_{2\,max}$ and on-transient kinetic parameters. However, for LD runners (n=16), relationships (r = -0.70 to -0.72, P <0.01) were consistently identified between τ_{on} and $\dot{V}O_{2\,max}$ irrespective of BM exponents. There was also a relationship (r = -0.55, P = 0.029) between τ_{off} and $\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) for LD runners, but not MD runners (r = 0.03; P = 0.900). There were no relationships between $\dot{V}O_{2\,max}$ and MRT_{on} or MRT_{off} (see Appendix 9.5 and 9.6 for MD and LD runners respectively).



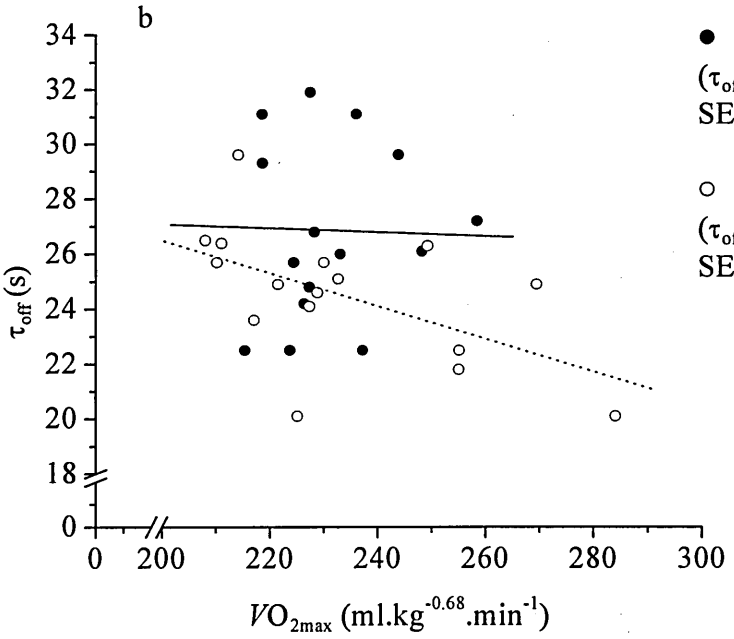


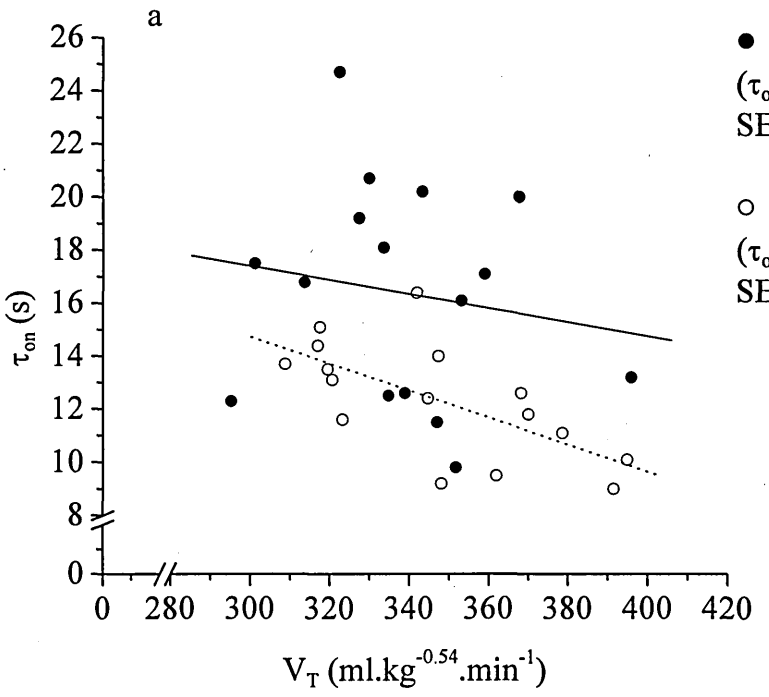
Figure 6.1 The relationship between $\dot{V}O_{2\,max}$ (ml·kg$^{-0.68}$·min^{-1}) and (a) τ_{on} and (b) τ_{off} in MD (n=16) and LD (n=16) runners.

6.3.3 Relationships with V_T

The relationships between on- and off-transient $\dot{V}O_2$ kinetics and V_T, expressed using the sample-specific BM exponent (-0.54), are illustrated in Figure 6.2. The

relationships between $\dot{V}O_2$ kinetics and V_T using alternative BM exponents are presented in Appendices 9.7 to 9.9.

For LD runners, τ_{on} was related to V_T (r = -0.65, P = 0.006; Figure 6.2a). However, there was no relationship between τ_{off} and V_T, regardless of the BM exponent used to express V_T. In MD runners, there were no relationships between any on- or off-transient $\dot{V}O_2$ kinetics parameter and V_T (Figure 6.2a, b). When MD and LD runners were considered collectively, τ_{on} and V_T were related when V_T was expressed as a 0.67 and 0.54 power-function ratio of BM (r = -0.35, P = 0.047 and r = -0.36, P = 0.044 respectively; see Appendix 9.7).



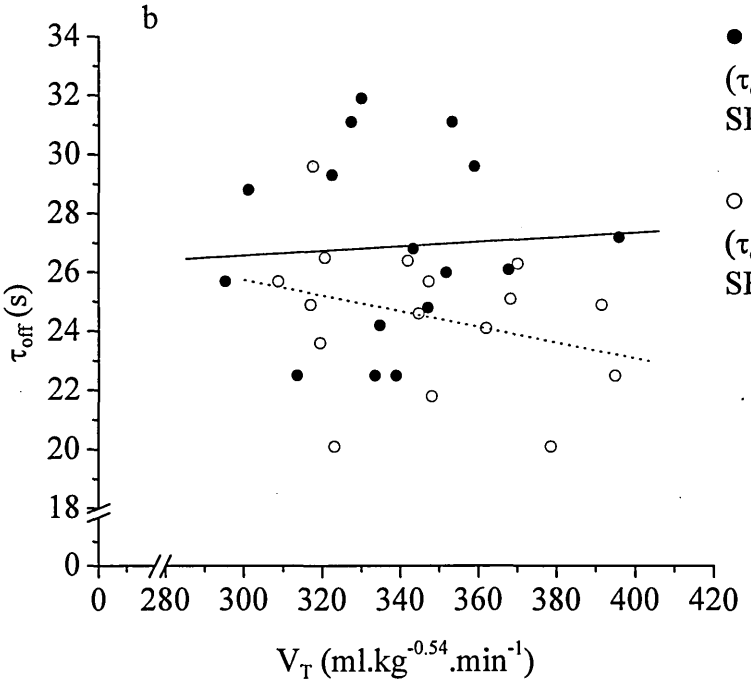


Figure 6.2 The relationship between V_T (ml·kg$^{-0.54}$·min^{-1} and (a) τ_{on} and (b) τ_{off} in MD (n=16) and LD (n=16) runners.

6.3.4 Relationships with RE

For the runners as a whole (n=32), there were no relationships between RE and any on- or off-transient $\dot{V}O_2$ kinetic parameter. For MD runners only, RE expressed in absolute terms (l·min^{-1}) was related to τ_{on} and MRT_{on} (r = 0.59, P = 0.020 and r = 0.58, P =

0.022 respectively; see Appendix 9.10), but not to τ_{off} or MRT_{off}. For LD runners, RE was related to τ_{on} (r = -0.55 to -0.59, P <0.05) and τ_{off} (r = -0.56 to -0.65, P <0.05) regardless of the BM exponent. The MRT_{on} was related with RE only when expressed as a ratio standard of BM (see Appendix 9.10). In any group, there were no relationships between MRT_{off} and RE (P >0.05).

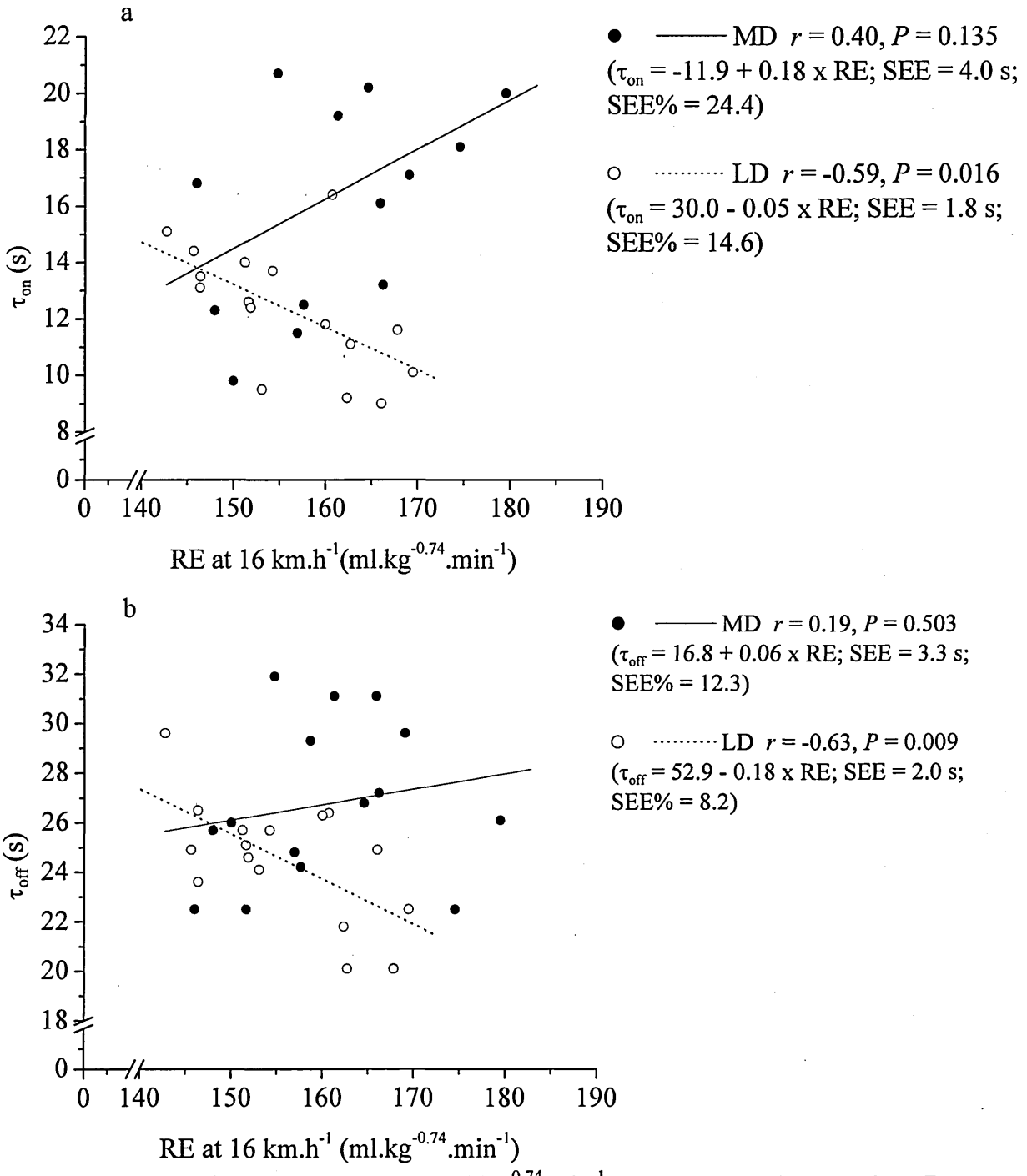


Figure 6.3 The relationship between RE ($ml \cdot kg^{-0.74} \cdot min^{-1}$) and (a) τ_{on} and (b) τ_{off} in MD (n=16) and LD (n=16) runners.

In acknowledgment that some MD and LD runners were exercising above V_T at 16 $km \cdot h^{-1}$, the $\dot{V}O_2$ ($ml \cdot kg^{-0.74} \cdot min^{-1}$) at 16 $km \cdot h^{-1}$ was adjusted to account for a potentially developing slow component of $\dot{V}O_2$. This could distort the relationship between $\dot{V}O_2$ kinetic parameters and RE. The magnitude of the slow component for each runner was determined by calculating how much the individual was exercising above V_T during the assessment of RE. This was done by calculating the difference in $\dot{V}O_2$ between V_T and RE and expressing this difference as a percentage of the difference between V_T and $\dot{V}O_{2\,max}$ ($\Delta\%$). Using the data (A, δ and τ for the slow component) of Carter *et al.* (2002), who's participants were of a similar level of fitness, the contracted slow component of $\dot{V}O_2$ was estimated for each runner. This additional $\dot{V}O_2$ was deducted from the measure of RE at 16 $km \cdot h^{-1}$ (i.e. the mean $\dot{V}O_2$ during the last 30 s of a 4 min bout of running at 16 $km \cdot h^{-1}$). Subsequently, correlation analyses were performed on the new 'adjusted' values. For combined, MD and LD runners, the minor adjustments of $\dot{V}O_2$ did not substantially effect the size and significance of the correlation coefficients for relationships between RE and $\dot{V}O_2$ kinetic parameters (Appendices 9.13 - 9.15) and therefore did not influence the interpretation of these relationships.

6.4 Discussion

Based on previous studies that have identified relationships between τ_{on} and $\dot{V}O_{2\,max}$ in only some groups (Fawkner *et al.*, 2002), it was considered appropriate to assess the relationships between measures in individual groups of MD and LD runners as well as a combined group in this study. However, it is acknowledged that a smaller sample size is more likely to be influenced by anomalous data which could significantly affect apparent relationships. However, assessment of leverage statistics when determining the *b* exponent for BM for each $\dot{V}O_2$ measure (Mahalanobis and Cook's distances) confirmed that no outliers or other adversely influential points were present. The actual *b* exponents for maximal ($\dot{V}O_{2\,max}$; 0.68 ± 0.15) and sub-maximal (RE; 0.74 ± 0.10) running derived from the current data were similar to those previously reported during treadmill running (Bergh *et al.*, 1991) and support the need for different BM exponents for different intensities of exercise (Darveau *et al.*, 2002). Furthermore, the SE for empirically derived exponents resulted in the *b* exponent encompassing alternative theoretical exponents which are based on surface-law (Åstrand and Rodahl, 1986), elasticity (Kleiber, 1947; McMahon, 1973) and allometric cascade models (Darveau *et al.*, 2002), but not the ratio standard. This demonstrates that there is a clear need for allometric adjustment of differences in BM in MD and LD runners.

6.4.1 Relationships between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$

When MD and LD runners were considered together, the relationship between τ_{on} and $\dot{V}O_{2\,max}$ was poor irrespective of how $\dot{V}O_{2\,max}$ was expressed. This was also apparent for MRT_{on} which reflects the entire $\dot{V}O_2$ response from the onset of exercise (i.e. including phase I). The poor relationship between these measures is consistent with a previous study involving endurance-trained runners (Lake *et al.*, 1986). However, when relationships were assessed in groups of runners according to their preferred distance, the relationship between $\dot{V}O_{2\,max}$ and τ_{on} for LD runners was considerably higher than that of MD runners (Figure 6.1). This relationship between τ_{on} and $\dot{V}O_{2\,max}$ is in agreement with several previous studies (Powers *et al.*, 1985; Chilibeck *et al.*, 1996;

Fawkner *et al.*, 2002) and runs counter to suggestions that $\dot{V}O_2$ kinetics do not provide a useful predictive index for maximum aerobic performance (Whipp *et al.*, 2001).

For a direct comparison, the study of Powers *et al.* (1985) is most relevant because they reported a strong relationship between $\dot{V}O_2\ t_{½}$ and $\dot{V}O_{2\ max}$ in LD runners who were similar to the runners in the present study with respect to $\dot{V}O_{2\ max}$ (mean ± SEM: 58 ± 2.6 $ml \cdot kg^{-1} \cdot min^{-1}$). Furthermore, runners were heterogeneous with respect to both $\dot{V}O_{2\ max}$ (range = ~50 to 70 $ml \cdot kg^{-1} \cdot min^{-1}$) and $\dot{V}O_2\ t_{½}$ (range = 21.6 to 36 s). The heterogeneity observed for the present sample for $\dot{V}O_{2\ max}$ ranged from 49.0 to 70.1 $ml \cdot kg^{-1} \cdot min^{-1}$ and τ_{on} ranged from 9.0 to 24.7 s, suggesting that the samples were similar. However, the studies differ with respect to the adopted mode of exercise. Powers *et al.* (1985) used cycle ergometry compared to treadmill ergometry in the present study. This suggests that the relationship between τ_{on} and $\dot{V}O_{2\ max}$ is independent of the mode of exercise in LD runners, although the use of treadmill ergometry for the assessment of $\dot{V}O_2$ kinetics in MD and LD runners is more specific than cycle ergometry.

Despite the above, no relationships were observed between τ_{on} and $\dot{V}O_{2\ max}$ in MD runners, suggesting that the magnitude of $\dot{V}O_{2\ max}$ does not dictate the dynamic response of $\dot{V}O_2$ at the onset of moderate-intensity exercise. This observation could be attributable to the greater variability observed for each measure. As illustrated in Figure 6.1a, there was considerable individual variability in τ_{on} for MD runners despite runners having a similar $\dot{V}O_{2\ max}$. For example, MD runners with a $\dot{V}O_{2\ max}$ of ~59 $ml \cdot kg^{-1} \cdot min^{-1}$ manifested a τ_{on} that ranged from ~12 to 20 s. Consequently, this 'overlap' resulted in a poor relationship between τ_{on} and $\dot{V}O_2$ kinetics in this group. A similar degree of variability has been reported elsewhere in untrained and trained individuals of varying fitness (Whipp *et al.*, 2001). This suggests a dissociation between τ_{on} and $\dot{V}O_{2\ max}$ in MD runners which might be caused by variations in training duration and/or intensity compared to LD runners. A greater volume of training ($km \cdot wk^{-1}$) was evident

in LD runners compared to MD runners (Table 6.1) - a characteristic of training regimes previously observed in elite MD and LD runners (Costill *et al.*, 1976b). Potentially, the volume of training could be a stimulus for faster $\dot{V}O_2$ kinetics which would account for the difference in $\dot{V}O_2$ kinetics between MD and LD runners. In support of this suggestion, training volume has been shown to influence the magnitude of response of mitochondrial oxidative enzymes (Fitts *et al.*, 1975; Hickson, 1981) which is also likely to influence $\dot{V}O_2$ kinetics. To investigate this possibility, the relationship between the volume of training and $\dot{V}O_2$ kinetics was assessed. This revealed a relationship between τ_{on} and $km \cdot wk^{-1}$ for MD (r = -0.63, P = 0.009), LD (r = -0.65, P <0.001) and combined runners (r = -0.72, P <0.001) suggesting that characteristics of training in MD and LD runners, which potentially influence adaptations at the cellular level, are reflected in a runner's $\dot{V}O_2$ kinetics.

Different training regimes between MD and LD runners could influence $\dot{V}O_2$ kinetics. Owing to the greater anaerobic energy contributions to performance in MD and LD events, the training for MD events usually consists of short-duration, high-intensity running. However, high-intensity training can enhance the oxidative capacity of Type IIa fibres (Saltin *et al.*, 1976; 1977) which would contribute to the attainment of $\dot{V}O_{2\,max}$. Therefore, in MD runners, this could result in a $\dot{V}O_{2\,max}$ that is similar to LD runners, despite inferior metabolic adaptations at the cellular level in Type I fibres. Consequently, this might result in a longer τ_{on} than in LD runners and would affect the relationship between $\dot{V}O_{2\,max}$ and τ_{on}.

It has been suggested (Gollnick *et al.*, 1972) that the oxidative capacity of muscle cells is unlikely to limit the capacity of the body to utilise oxygen during maximal exercise. Therefore, if measures of $\dot{V}O_2$ kinetics actually reflect the oxidative capacity or potential of the muscle, then a relationship with $\dot{V}O_{2\,max}$ might not be observed. In support, results from studies involving endurance training (Hurley *et al.*, 1984) and de-

conditioning (Henrikksson and Reitman, 1977) clearly show that an enhanced oxidative potential of skeletal muscle occurs independently of changes in $\dot{V}O_{2\,max}$.

It could be that measures of $\dot{V}O_2$ kinetics during moderate-intensity exercise are not strongly related to $\dot{V}O_{2\,max}$. This is because only Type I fibres are likely to be recruited during moderate-intensity exercise (Vøllestad and Blom, 1985) which was used to measure $\dot{V}O_2$ kinetics. During the incremental tests to determine $\dot{V}O_{2\,max}$, it is probable that both Type IIa and Type IIx fibres are sequentially recruited. Therefore, measures of $\dot{V}O_2$ at the onset of heavy-intensity exercise, which presumably reflect the $\dot{V}O_2$ kinetics of Type I and II fibres, could have stronger relationships with $\dot{V}O_{2\,max}$ than with $\dot{V}O_2$ kinetics measures obtained during moderate-intensity exercise. However, experimental evidence demonstrating that this might not be the case has been provided by Barstow *et al.* (1996) who showed that there was no relationship between $\dot{V}O_{2\,max}$ and phase II $\dot{V}O_2$ kinetics for heavy-intensity cycle exercise in individuals who varied in training status.

6.4.2 Relationship between $\dot{V}O_2$ kinetics and V_T

The respiratory capacity of the muscle is of primary importance in determining the intensity of exercise, i.e. speed or PO, at which lactate accumulates (Ivy *et al.*, 1980). The slow twitch Type I fibres have been shown to have a high mitochondrial density and mitochondrial enzyme activity (Howald *et al.*, 1985) which favour oxidative energy production. In this regard, τ_{on} might be more related to a measure other than $\dot{V}O_{2\,max}$ that is predominantly determined by peripheral mechanisms e.g. V_T. For LD runners, however, a moderate relationship was identified between τ_{on} and V_T (r = -0.65, P = 0.006; Figure 6.2a) suggesting that peripheral mechanisms determining a high V_T are concurrent with fast $\dot{V}O_2$ kinetics. This supports the findings of previous studies (Weltman *et al.*, 1978; Chilibeck *et al.*, 1996). However, the relationship between τ_{on} and V_T in LD runners was lower than that observed for $\dot{V}O_{2\,max}$ - a finding also observed by Chilibeck *et al.* (1996). The above findings are not apparent in MD or

combined runners since no relationship between V_T and on-transient $\dot{V}O_2$ kinetic parameters were observed. Similar to the lack of relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$, it is likely that this is attributable to the intra-participant variability. Despite a wide range of values with respect to the off-transient $\dot{V}O_2$ kinetics, a clear dissociation between τ_{off} and V_T was observed. This was evident regardless of whether combined or individual groups of MD and LD runners were assessed.

6.4.3 Relationship between $\dot{V}O_2$ kinetics and RE

Prior to this investigation, the relationship between $\dot{V}O_2$ kinetics and RE had not been established. When runners were considered as a combined group or as MD runners only, there was no relationship between any on- or off-transient $\dot{V}O_2$ kinetic parameter and RE. However, in LD runners, both τ_{on} (r = -0.55, P = 0.027 to r = -0.59, P = 0.016) and τ_{off} (r = -0.62, P = 0.010 to r = -0.65, P = 0.006) were negatively related with RE (Figure 6.3a, b), suggesting that runners with a low $\dot{V}O_2$ whilst running at 16 km·h^{-1} have slow on- and off-transient $\dot{V}O_2$ kinetics. This finding is understandable given that there was a positive relationship between RE and $\dot{V}O_{2\,max}$ (r = 0.65, P <0.001) which supports previous studies (Pate *et al.*, 1992; Morgan and Daniels, 1994).

Biomechanical factors, such as ground reaction forces and joint angles, also influence RE (Williams and Cavanagh, 1987) and could potentially prevent the true physiological relationship between RE, fibre type and $\dot{V}O_2$ kinetics from being identified. This could be why no relationship between $\dot{V}O_2$ kinetics and RE was observed in MD runners. However, a biomechanical assessment of running technique and its effect on RE and $\dot{V}O_2$ kinetics was beyond the scope of this study.

There are contra-indications of using RE at a given speed or absolute intensity, especially when assessing the relationship between RE and $\dot{V}O_2$ kinetics. The relative intensity of running at 16 km·h^{-1} is likely to differ between runners. Inevitably, this might result in a greater recruitment of Type II muscle fibres in runners who are less

well-trained. In the present study, the intensity of exercise for measuring $\dot{V}O_2$ kinetics was constrained to be 80%V_T and this intensity of exercise, based on previous work (Vøllestad and Blom, 1985), would be likely to require the recruitment of only Type I fibres. Therefore, correlations between τ_{on} (predominantly Type I fibres) and RE (potentially different proportions of Type I and II fibres according to fitness) might be distorted because of the different recruitment of muscle fibres.

In this study, any potential influences of BM on $\dot{V}O_{2\,max}$, V_T and RE were effectively partitioned out by allometric modelling. This permitted an appropriate assessment of the relationship between these aerobic measures. However, in most instances, expressing $\dot{V}O_2$ as a power-function ratio of BM (Table 6.2) did not result in a significant improvement in the relationships between these three measures and on- and off-transient $\dot{V}O_2$ kinetic parameters (see Appendices 9.4 - 9.15 for correlation matrices). This was also apparent in the study of Fawkner *et al.* (2002) who showed that $\dot{V}O_{2\,max}$ and τ_{on} were similarly correlated when $\dot{V}O_{2\,max}$ was expressed as a ratio of BM and as a 0.89 power-function ratio of BM (r = -0.81 and -0.82 respectively, P <0.05).

6.5 Conclusion

The findings of this study show that there are relationships between on-transient $\dot{V}O_2$ kinetics, $\dot{V}O_{2\,max}$, RE and V_T, primarily in LD runners. This suggests that on a functional level, the responses of the cardiovascular system and skeletal muscle to habitual endurance-training are closely linked. Relationships between $\dot{V}O_2$ kinetic and other measures of aerobic function in MD are less consistent which is probably due to the observed greater intra-participant variability of measures. With respect to off-transient $\dot{V}O_2$ kinetics, relationships were inconsistent and less well-defined compared to on-transient $\dot{V}O_2$ kinetics which suggests that the latter might be more appropriate for assessing physiological status. Furthermore, the volume of training also appears to influence on-transient $\dot{V}O_2$ kinetics in MD and LD runners. The importance of on- and

off-transient $\dot{V}O_2$ kinetics in MD and LD runners can not be ignored since it has not yet been established whether this measure accounts for variations in running performance.

CHAPTER 7

An investigation of factors contributing to successful 5 km running performance

7.1 Introduction

Laboratory tests have been used extensively to assess the physiological status of endurance-trained runners (Londeree, 1986). Accordingly, relationships between various physiological measures and running performance have been assessed for these types of runner (Costill, 1967; Farrell *et al.*, 1979; Brandon and Boileau, 1992; Abe *et al.*, 1998). Primarily, investigators have considered the contribution of three aerobic measures to predict running performance that can be determined using pulmonary gas-exchange responses during sub-maximal and maximal exercise: 1) $\dot{V}O_{2\,max}$; 2) V_T and 3) RE.

Traditionally, $\dot{V}O_{2\,max}$ has been the standard measure for the assessment of runners' ability or potential in MD or LD events with a high $\dot{V}O_{2\,max}$ being considered a pre-requisite for endurance running success (Saltin and Åstrand, 1967; Costill, 1967). This is because the O_2 cost of running is directly proportional to running speed (Margaria *et al.*, 1963). In several studies it has been shown that $\dot{V}O_{2\,max}$ is highly correlated with running performance (Costill *et al.*, 1972; Foster, 1983). However, the majority of these studies were conducted using samples of runners that were heterogeneous (i.e. displayed a wide range both of $\dot{V}O_{2\,max}$ and performance values). In homogeneous groups of runners, $\dot{V}O_{2\,max}$ appears to be a poor predictor of performance (Conley and Krahenbuhl, 1980; Morgan *et al.*, 1989) which suggests that other measures contribute to and/or determine running performance.

Other measures such as RE (Conley and Krahenbuhl, 1980) and V_T (Powers *et al.*, 1983) have been found to correlate with running performance, especially when groups of runners are homogeneous in terms of running ability. In some instances, the $\dot{V}O_2$ at the V_T/LT has

been shown to correlate more with running performance than $\dot{V}O_{2\,max}$ *per se* (Farrell *et al.*, 1979). In terms of RE, the lower the $\dot{V}O_2$ at a sub-maximal speed the better the RE. In elite-distance runners, with a narrow range of $\dot{V}O_{2\,max}$, RE at different speeds is highly related to 10 km running performance (Conley and Krahenbuhl, 1980). Collectively, these findings demonstrate the importance of considering several maximal and sub-maximal measures to predict running performance.

It has been suggested that $\dot{V}O_{2\,max}$ is largely determined by cardiovascular function (Saltin, 1990), whereas sub-maximal measures of $\dot{V}O_2$, i.e. V_T and RE, are associated with peripheral factors such as the fibre composition and respiratory capacity (i.e. intra-muscular concentration of oxidative enzymes) of muscle (Ivy *et al.*, 1980; Rusko *et al.*, 1980). Collectively, this suggests that peripheral mechanisms that determine the utilisation of O_2 in muscle have a greater influence on running performance than $\dot{V}O_{2\,max}$. In support, Costill *et al.* (1976a, b) has suggested that muscle oxidative enzyme activity and muscle fibre composition might have an improved relationship with distance running performance.

One other measure that could be related to running performance is that of $\dot{V}O_2$ kinetics. It is likely that such kinetics, especially in the moderate-domain, are determined by intrinsic mechanisms involved in the utilisation of O_2 in muscle (Whipp and Mahler, 1980). It has been shown that $\dot{V}O_2$ kinetics are sensitive to endurance training and become faster as training progresses both in previously untrained (Phillips *et al.*, 1995) and trained individuals (Norris and Peterson, 1998). Recognising the importance of $\dot{V}O_2$ kinetics, Whipp *et al.* (1981) suggested that they are one of four measures of aerobic function (including $\dot{V}O_{2\,max}$, O_2 cost of exercise and AT) that make up the 'aerobic' profile of a performer and any attempt to differentiate performers or to predict performance capability should consider all four. However, only a limited number of studies have considered the relationship between $\dot{V}O_2$ kinetics and performance. For example, Norris and Peterson (1998) showed that changes in τ_{on} were more closely related to changes in cycling

performance than $\dot{V}O_{2\,max}$. In runners, Demarle *et al.* (2001) showed that a reduction in the O_2 deficit after endurance training, primarily resulting from faster $\dot{V}O_2$ kinetics, was correlated with improvements in running performance. Collectively, both studies suggest that: 1) measures of $\dot{V}O_2$ kinetics reflect physiological training adaptations and 2) measures of $\dot{V}O_2$ kinetics might be useful when predicting performance in competitive athletes.

7.1.1 Quantifying running performance

Running performance in competitive runners is most often quantified using race times. However, there are disadvantages to using such times in the assessment of relationships between performance and physiological measures. For example, when the race is not in close proximity to the start of a study and the race conditions (weather, level of competition and tactics) are not identical for all athletes, race times might not reflect an athlete's true physiological performance capabilities. Consequently, relationships with physiological measures could be confounded. Similarly, outdoor time-trials can be influenced by inconsistent environmental conditions which might disadvantage some runners. Alternatively, indoor treadmill-based time-trials could be used which possess several advantages: 1) laboratory time-trials allow a valid measure of an athlete's running performance that can be obtained in close proximity to when physiological measures are collected; 2) time-trial performance tests allow an assessment of running performance under controlled laboratory conditions; 3) reproducibility has been previously reported as good (Ramsbottom *et al.*, 1992) and 4) treadmill 5 km time-trials are highly correlated with outdoor running performance (R^2 = 0.97, P <0.001) which suggests that laboratory performance is indicative of actual race performance (Scott and Houmard, 1994). In support of this, Scott and Houmard (1994) found that their physiological measures ($\dot{V}O_{2\,peak}$, time to exhaustion and peak running velocity) correlated more strongly with a treadmill time-trial than a recent best 5 km race time.

7.1.2 Predicting running performance

Success in MD and LD running is multi-factorial and therefore influenced by several physiological, mechanical and psychological factors that interact. Using multiple regression techniques, several studies have attempted to model MD (Powers *et al.*, 1983; Bulbulian *et al.*, 1986; Housh *et al.*, 1988; Brandon and Boileau, 1992; Grant *et al.*, 1997) and LD running performance (Kumagai *et al.*, 1982; Roecker *et al.*, 1998) to determine the most important physiological measure(s). In most studies, traditional aerobic measures such as $\dot{V}O_{2\,max}$, V_T/LT and RE have been considered and collectively, have been shown to contribute (Powers *et al.*, 1983; Weyand *et al.*, 1994; Roecker *et al.*, 1998). However, in most studies, some of the variability in performance remains unaccounted for which suggests that other physiological measures, not yet considered, might contribute to the prediction of running performance. To date, no study has considered measures of on- and off-transient $\dot{V}O_2$ kinetics as potential determinants of running performance in endurance-trained runners.

7.1.3 Aim of study

The original aim of this study was two-fold: 1) to assess the relationships between $\dot{V}O_2$ kinetic parameters and 5 km running performance in MD and LD runners and 2) to determine the primary aerobic factor(s) ($\dot{V}O_{2\,max}$, V_T, RE and $\dot{V}O_2$ kinetics) determining 5 km running performance.

7.2 Participants and methods

7.2.1 Participants

Eighteen male MD (800/1500m) and 18 male LD (5000/10000m) runners, accustomed to the procedures of physiological testing, provided written informed consent and participated. Participants were moderately well-trained competitive MD and LD runners recruited from athletic clubs in the North of England. The age, anthropometric and training characteristics of participants are presented in Table 7.1. Ethics approval was obtained from the Research Ethics Committee, Sheffield Hallam University. Prior to participation in the study each athlete completed a medical screening questionnaire (Appendix 6).

Table 7.1 Age, anthropometry and training volume of participants. Values are mean ± SD.

Measure	MD (n=18)	LD (n=18)	Combined (n=36)
Age (years)	21.7 ± 5.4	25.3 ± 4.3*	23.5 ± 5.1
Stature (cm)	177.8 ± 7.4	180.1 ± 6.6	179.0 ± 7.0
BM (kg)	67.2 ± 5.9	69.6 ± 8.2	68.4 ± 7.2
Volume of training ($km \cdot wk^{-1}$)	43.6 ± 15.2	68.2 ± 15.3**	55.9 ± 19.5

*Greater than MD runners, $P = 0.033$, **$P < 0.001$.

7.2.2 Experimental design

Participants visited the laboratory for physiological testing on three occasions within a seven-day period. Each test was separated by at least 48 hours and was performed at approximately the same time of day. Physiological testing during the first visit to the laboratory involved the measurement of RE at 16 $km \cdot h^{-1}$ and an incremental exercise test to volitional exhaustion to allow the determination of V_T and $\dot{V}O_{2\,max}$. Visit 2 involved a square-wave exercise protocol to determine on- and off-transient $\dot{V}O_2$ kinetics. Visit 3 involved a 5 km treadmill-based time-trial. Throughout the testing period, participants were requested to maintain their usual dietary intake and to abstain from participation in

heavy training and consumption of alcohol and/or caffeine in the 48 hours preceding each test.

7.2.3 Experimental protocols

Each participant completed: 1) a series of 4-6 four-min bouts of sub-maximal exercise with running speed increasing by 1 $km \cdot h^{-1}$ every stage for the determination of RE at 16 $km \cdot h^{-1}$; 2) an incremental exercise test to volitional exhaustion for the determination of V_T and $\dot{V}O_{2\,max}$; 3) a square-wave protocol consisting of alternating 6 min bouts of walking (4 $km \cdot h^{-1}$) and running (speed requiring 80%V_T) to determine $\dot{V}O_2$ kinetics and 4) a treadmill-based 5 km time-trial to determine running performance (see Chapter 3, Section 3.2.8).

Pulmonary gas-exchange was measured breath-by-breath during all exercise tests (excluding the 5 km time-trial). The HR and [HLa] were also measured during all tests (see Chapter 3, Section 3.1.3 and 3.1.4).

7.2.4 Data analysis

Breath-by-breath data obtained during the assessment of RE, V_T, $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics were analysed in accordance with procedures outlined in Chapter 3, Sections 3.2.5.1 to 3.2.7.1.

7.2.5 Statistical analyses

Prior to regression analysis, ANCOVA was performed to determine the appropriateness of treating MD and LD runners as separate groups. The ANCOVA highlighted that neither the variances about regression, slopes or elevation for MD and LD runners differed sufficiently for them to be considered as two groups for all physiological measures (for ANCOVA summary see Appendix 10.3a). Therefore, collapsing the groups into one combined sample (n=36) and selecting the ten fastest runners (high performers, rank 1-10)

and the slowest ten runners (low performers, rank 27-36) was justified as a way to discriminate between performers and identify primary determinant(s) of performance. This approach avoided the effects that influential 'overlapping' data might have on the relationships and regression models. The original combined sample of MD and LD runners (n=36) was also used in further analysis and was considered as a sample of 'endurance-trained' runners.

Descriptive statistics (mean ± SD) were calculated for each physiological and performance measure for each group. The physiological and running performance data was assessed in three ways: 1) relationships between physiological measures and 5 km running performance were explored for each group using Pearson's product moment correlation coefficient; 2) ANCOVA was performed to determine whether the variances, slopes and elevation of data were different between high and low performers (where P <0.05, adjusted means were calculated) and 3) multiple regression was used to formulate an equation to identify the primary physiological determinant(s) of 5 km running performance for each group. Specifically, stepwise regression was used to obtain: 1) the lowest SEE; 2) the highest r and 3) accomplish this with the fewest IV's. Prior to conducting any statistical analyses, appropriate checks were made to ensure that the assumptions underpinning bi-variate correlation, ANCOVA and multiple regression techniques were adequately met (see Chapter 3, Section 3.3.7).

7.3 Results

The mean ± SD for anthropometric/performance data and physiological data for each group of runner are displayed in Tables 7.2 and 7.3 respectively. As anticipated, the high performing group consisted predominantly of LD runners (MD, n=2; LD, n=8) and the low performing group consisted predominantly of MD runners (MD, n=7; LD, n=3).

Table 7.2 Age, anthropometric and performance data of participants. Values are mean ± SD.

Measure	High (n=10)	Low (n=10)	Combined (n=36)
Age (years)	23.0 ± 2.8	24.9 ± 7.3	23.5 ± 5.1
Stature (cm)	179.5 ± 4.8	174.3 ± 5.7	179.0 ± 7.0
BM (kg)	64.0 ± 3.6	68.3 ± 7.3	68.4 ± 7.2
Volume of training ($km \cdot wk^{-1}$)	76.9 ± 13.7	41.9 ± 15.9**	55.9 ± 19.5
5 km time (min:s)	15:56 ± 0:26	18:38 ± 0:58**	17:10 ± 1:12
5 km speed ($m \cdot s^{-1}$)	5.2 ± 0.2	4.5 ± 0.2**	4.9 ± 0.3
$\%\dot{V}O_{2\,max}$ sustained	94.5 ± 3.1	95.0 ± 3.0	94.8 ± 3.0

$\%\dot{V}O_{2\,max}$ sustained during the 5 km time-trial is estimated from the $\dot{V}O_2$ - running speed relationship and $\dot{V}O_{2\,max}$ for each runner. **Different from high performers, P <0.001.

Table 7.3 Physiological characteristics of participants. Values are mean ± SD.

Measure	High (n=10)	Low (n=10)	Combined (n=36)
$\dot{V}O_{2\,max}$ ($ml \cdot min^{-1}$)	4168 ± 368	3957 ± 317	4122 ± 471
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	65.1 ± 3.9	58.3 ± 5.6**	60.5 ± 5.2
V_T ($ml \cdot min^{-1}$)	3458 ± 284	3230 ± 300	3370 ± 327
V_T ($ml \cdot kg^{-1} \cdot min^{-1}$)	54.1 ± 3.8	47.5 ± 4.3**	49.5 ± 4.4
RE at 16 $km \cdot h^{-1}$ ($ml \cdot min^{-1}$)	3430 ± 290	3691 ± 319	3613 ± 355
RE at 16 $km \cdot h^{-1}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	53.6 ± 2.3	54.3 ± 4.3	53.0 ± 3.5
τ_{on} (s)	11.4 ± 1.8	16.7 ± 3.8**	14.5 ± 3.9
MRT_{on} (s)	26.3 ± 2.0	29.9 ± 2.6**	28.6 ± 3.0
τ_{off} (s)	24.7 ± 1.7	26.5 ± 3.4*	25.6 ± 3.0
MRT_{off} (s)	32.4 ± 2.0	35.7 ± 3.8*	34.5 ± 3.4

*Different than high performers, P <0.05, **P <0.01.

The participants' physiological characteristics for all measures are presented in Table 7.3. Measures of $\dot{V}O_2$ ($\dot{V}O_{2\,max}$, V_T and RE) are expressed in absolute terms ($ml \cdot min^{-1}$) and relative to BM ($ml \cdot kg^{-1} \cdot min^{-1}$). In addition, $\dot{V}O_2$ measures expressed as theoretically derived power-function ratios of BM were also considered (Appendix 10.1 and 10.2).

The results from the ANCOVA on high and low performers show that all physiological measures, including $\dot{V}O_2$ kinetics, could be used to discriminate between performers of different running ability (P <0.001). The adjusted mean for each physiological measure, based on the ANCOVA analysis, is presented in Table 7.4. A summary of the variance about regression, slopes and elevation is presented in Appendix 10.3b.

Table 7.4 Adjusted means for the high and low performers. Values are mean ± SEE.

Measure	High (n=10)	Low (n=10)	P
$\dot{V}O_{2\,max}$ (ml·kg^{-1}·min^{-1})	65.1 ± 0.1	58.3 ± 0.2	<0.001
V_T (ml·kg^{-1}·min^{-1})	54.1 ± 0.0	47.5 ± 0.2	<0.001
RE at 16 km·h^{-1} (ml·kg^{-1}·min^{-1})	53.5 ± 0.1	54.3 ± 0.2	<0.001
τ_{on} (s)	11.4 ± 0.1	16.7 ± 0.2	<0.001
MRT_{on} (s)	26.3 ± 0.1	29.8 ± 0.2	<0.001
τ_{off} (s)	24.7 ± 0.1	26.5 ± 0.2	<0.001
MRT_{off} (s)	32.4 ± 0.1	35.7 ± 0.2	<0.001

7.3.1 Physiological relationships with performance

Correlations between 5 km running performance and physiological measures expressed using several BM exponents were investigated in high and low performers and combined runners. Correlation matrices are presented in Appendices 10.6 to 10.17 which highlight the relationships between all physiological measures and 5 km running performance.

7.3.1.1 $\dot{V}O_2$ kinetics and 5 km running performance

The relationships between on-transient $\dot{V}O_2$ kinetic parameters (τ_{on} and MRT_{on}) and 5 km performance in high and low performers are illustrated in Figures 7.1 and 7.2 respectively. There were no relationships between τ_{on}, MRT_{on} and performance in separate high and low performing groups of runner (P >0.05). However, for combined runners, relationships between 5 km performance and τ_{on} (r = -0.54, P = 0.001) and MRT_{on} (r = -0.50, P = 0.002) were observed (see Appendix 10.17).

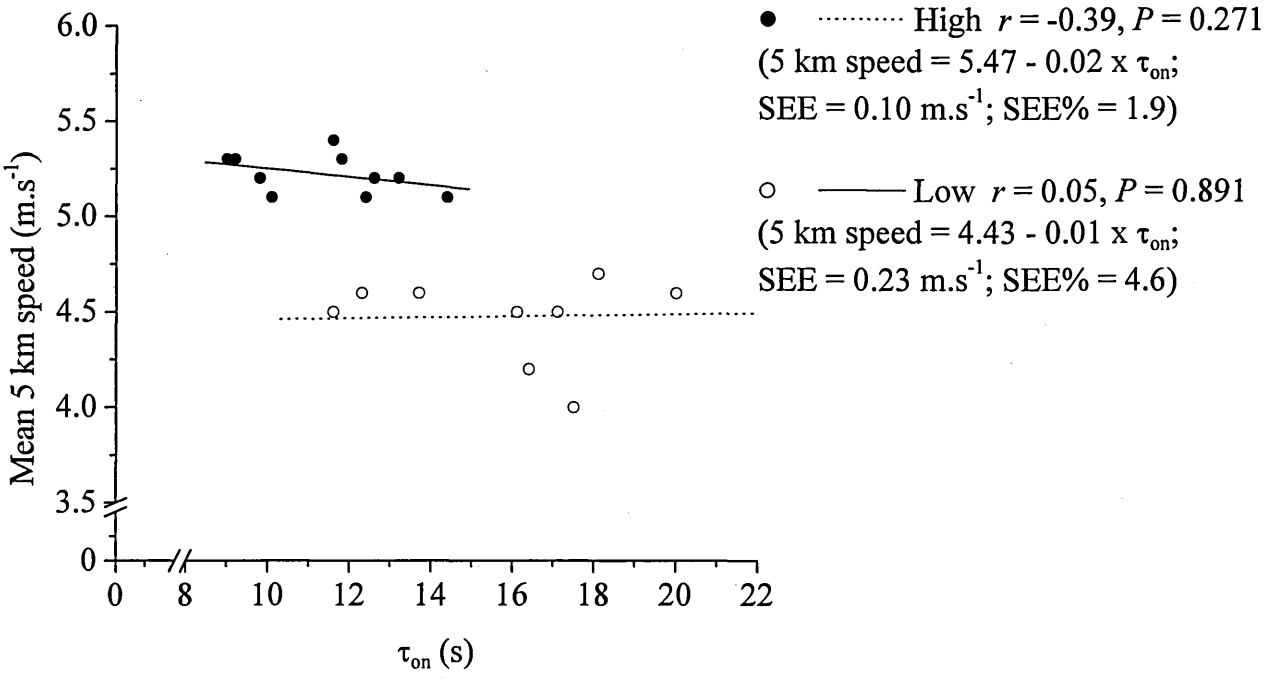


Figure 7.1 The relationship between τ_{on} and 5 km running performance in high (n=10) and low performers (n=10).

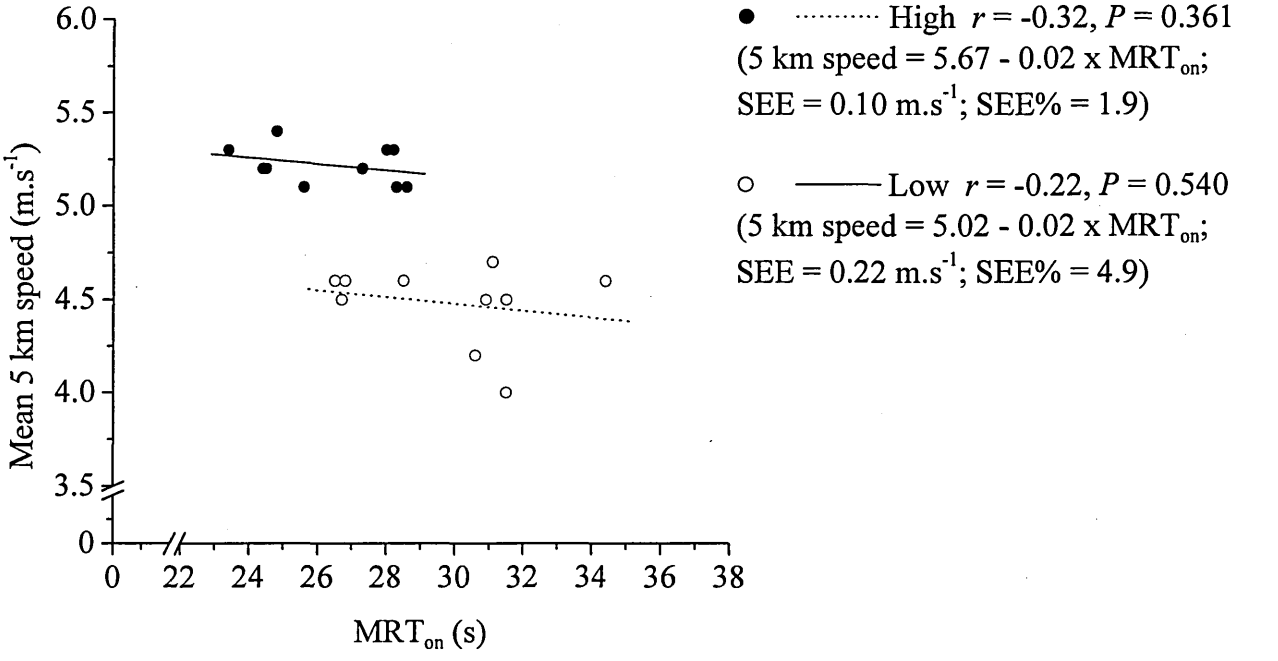


Figure 7.2 The relationship between MRT_{on} and 5 km running performance in high (n=10) and low performers (n=10).

Similar to the on-transient, no off-transient $\dot{V}O_2$ kinetic parameter (τ_{off} and MRT_{off}) was related to running performance in high or low performers (Figures 7.3 and 7.4). However, for combined runners, a relationship was observed for τ_{off} (r = -0.36, P = 0.030) and MRT_{off} and performance (r = -0.63, P = 0.003; see Appendix 10.17).

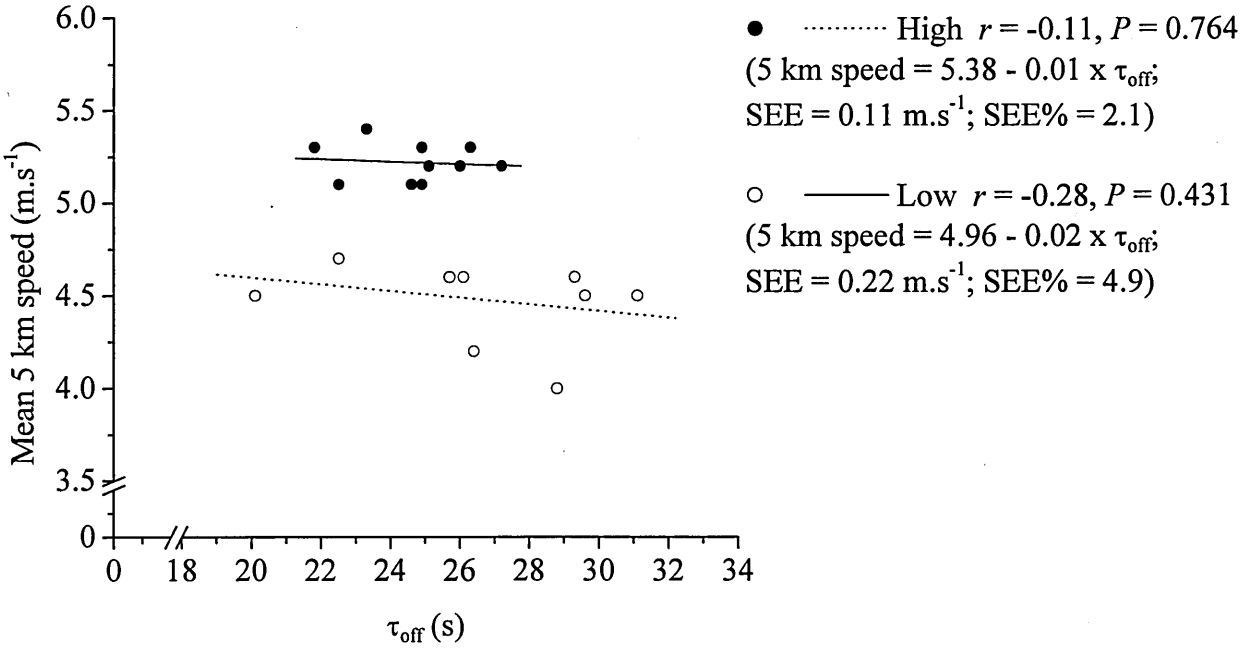


Figure 7.3 The relationship between τ_{off} and 5 km running performance in high (n=10) and low performers (n=10).

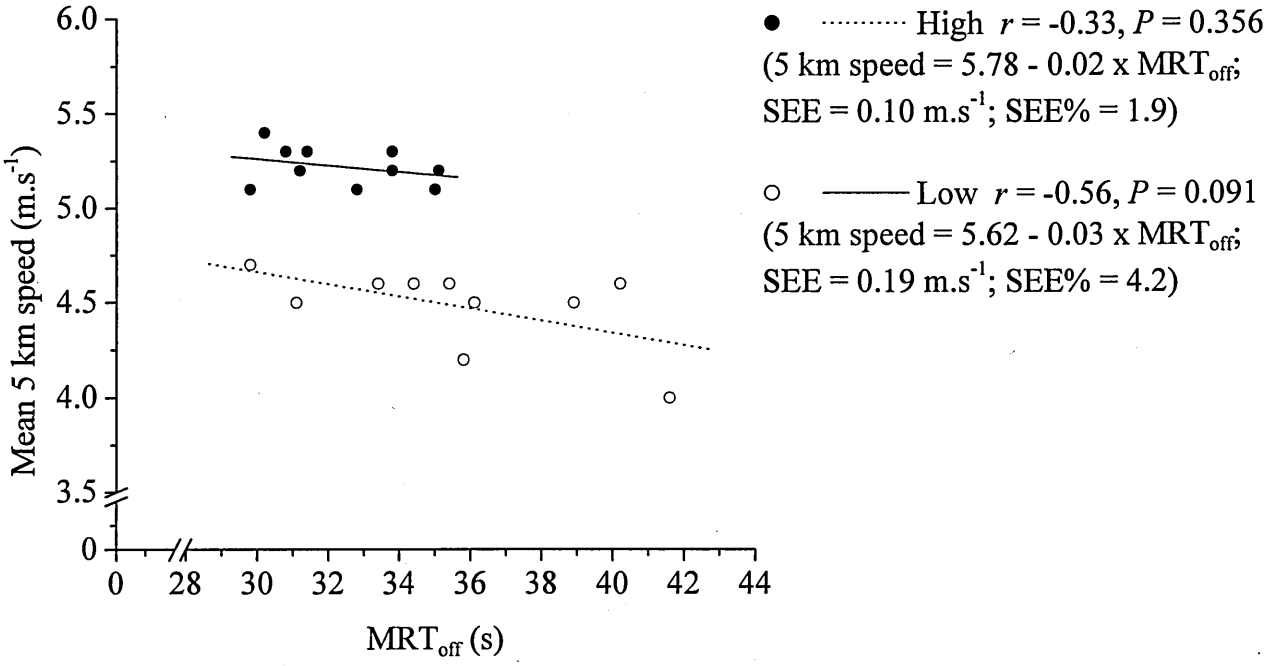


Figure 7.4 The relationship between MRT_{off} and 5 km running performance in high (n=10) and low performers (n=10).

7.3.1.2 $\dot{V}O_{2\,max}$, V_T and RE and 5 km running performance

The relationships between 5 km running performance and $\dot{V}O_{2\,max}$, V_T, RE are presented in Figures 7.5 - 7.7. For consistency, $\dot{V}O_2$ measures are expressed in $ml \cdot kg^{-1} \cdot min^{-1}$. This is because in most instances $\dot{V}O_2$ measures expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ were more highly related to performance than other BM exponents.

A relationship between $\dot{V}O_{2\,max}$ and performance was observed in both groups of runners, but was higher in high and low performers (r = 0.76, P = 0.010 and r = 0.76, P = 0.011 respectively) than in combined runners (r = 0.66, P <0.001; see Appendix 10.8). The method of expressing $\dot{V}O_{2\,max}$ with respect to the BM exponent had minimal influence on the magnitude of the relationships.

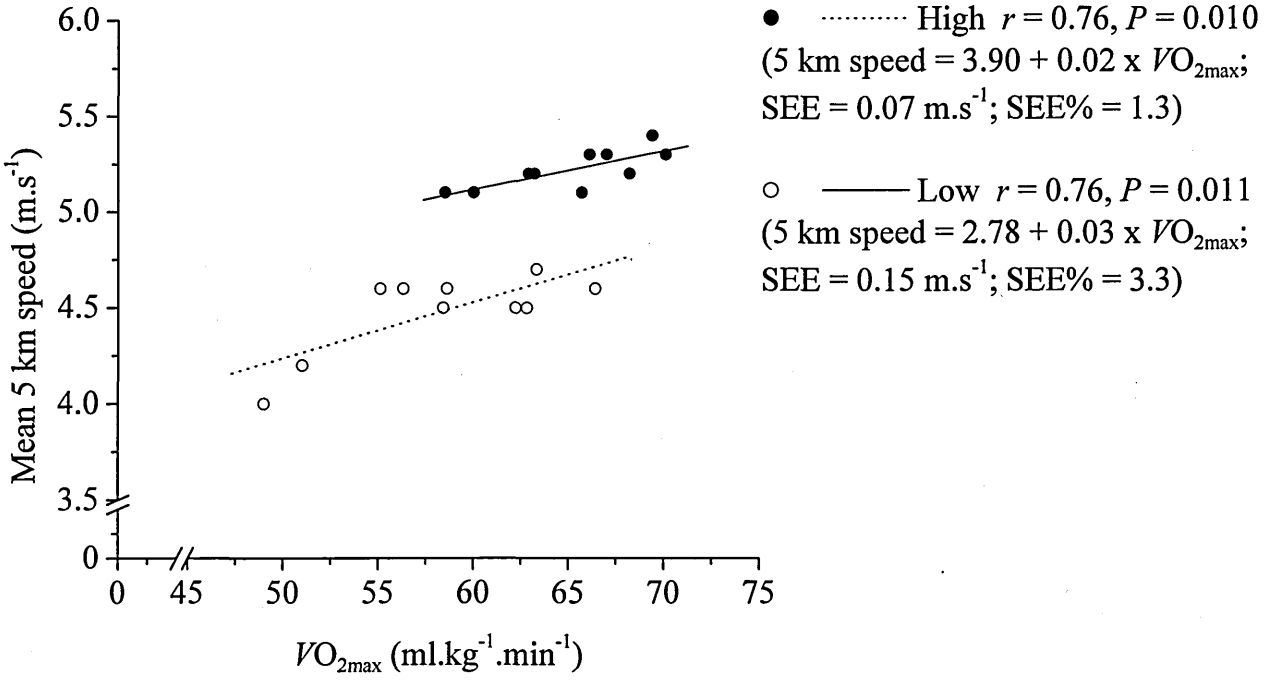


Figure 7.5 The relationship between $\dot{V}O_{2\ max}$ and 5 km running performance in high (n=10) and low performers (n=10).

The V_T was not related with running performance in either high or low performers (Figure 7.6). However, a relationship was observed in combined runners (r = 0.62, P <0.001; see Appendix 10.11). The BM exponent had no influence on the relationship between V_T and performance.

There were no relationships between RE and running performance in any group of runners regardless of the BM exponent used to express RE measures (Figure 7.7 and Appendix 10.14).

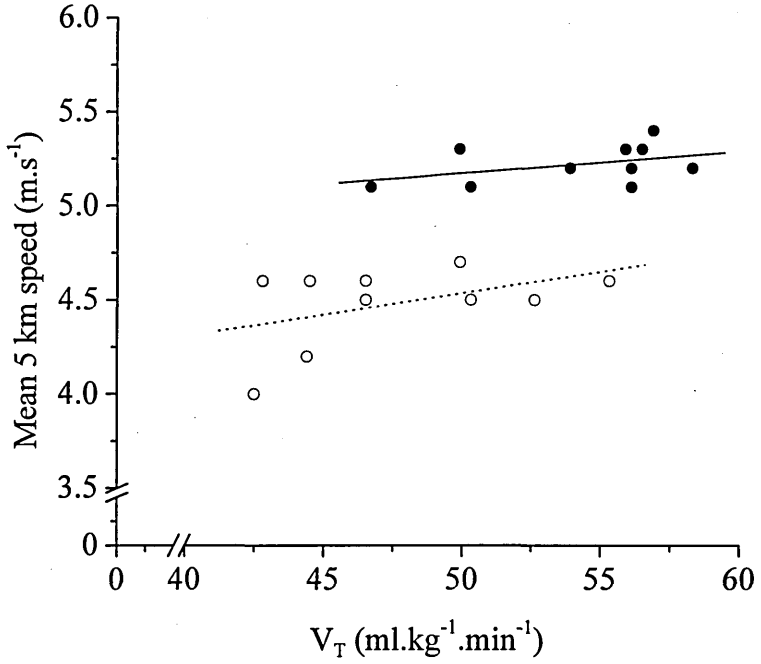


Figure 7.6 The relationship between V_T and 5 km running performance in high (n=10) and low performers (n=10).

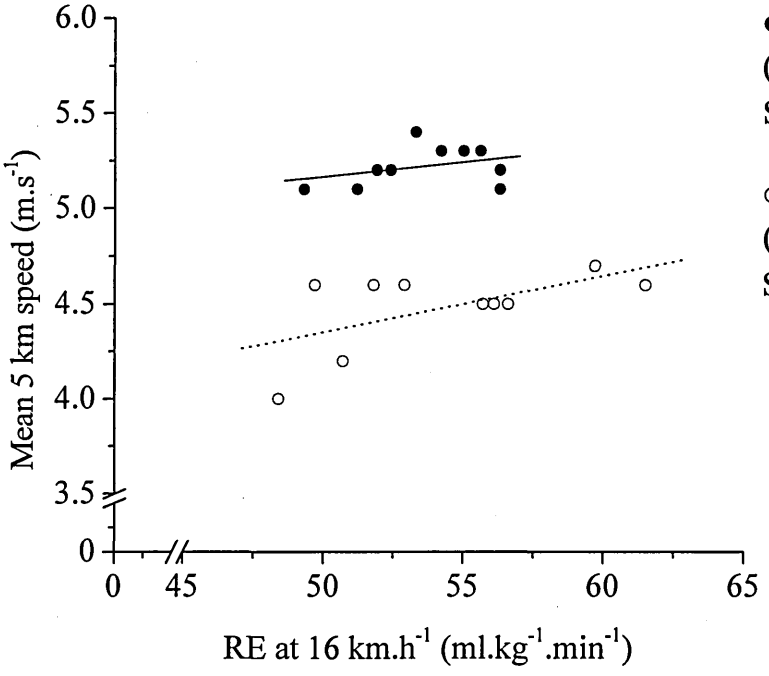


Figure 7.7 The relationship between RE at 16 km·h^{-1} and 5 km running performance in high (n=10) and low performers (n=10).

7.3.2 Predicting 5 km running performance

Several stepwise multiple regression analyses were performed to identify the most influential physiological measure(s) that contributed to the prediction of 5 km running performance in high, low and combined runners. This approach was used to investigate whether physiological determinants of running performance differed according to the standard of performer.

7.3.2.1 Regression models for high, low and combined runners

The relationships between 5 km running performance and $\dot{V}O_{2\,max}$, V_T and RE were explored using different BM exponents. However, in most instances, the different exponents had minimal influence over the magnitude of the correlation coefficients. Therefore, $\dot{V}O_2$ expressed as a ratio standard of BM was entered into the regression models. Consequently, six independent variables [$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$), V_T, ($ml \cdot kg^{-1} \cdot min^{-1}$), RE ($ml \cdot kg^{-1} \cdot min^{-1}$), τ_{on}, τ_{off}, MRT_{on} and MRT_{off}] were entered into each regression analysis. The regression models for predicting performance from physiological measures in high, low and combined performers are presented in Table 7.5. The model for high performers included $\dot{V}O_{2\,max}$ and RE which accounted for 86% of the variability in performance. The SEE and SEE% were 0.04 $m \cdot s^{-1}$ and 0.8% respectively. For low performers, only $\dot{V}O_{2\,max}$ was included in the final model which accounted for 57% of the variability in performance (SEE = 0.15 $m \cdot s^{-1}$; SEE% = 3.3%). In combined runners, the model included $\dot{V}O_{2\,max}$, RE and MRT_{off} which accounted for 75% of the variability in performance (SEE = 0.17 $m \cdot s^{-1}$; SEE% = 3.5%). The standardised β-coefficient, representing each measure's contribution to the regression equation, is also presented in Table 7.5.

Finally, the self-reported volume of training ($km \cdot wk^{-1}$) was entered into each regression analyses to explore whether this measure was influential. However, self-reported volume of training did not contribute to any regression model.

Table 7.5 Stepwise regression models to identify physiological determinants of running performance in high, low and combined performers .

Group	Regression model	R	R^2	Adj R^2	SEE	SEE%	*P*
High (n=10)	0.042 ($\dot{V}O_{2\,max}$, ml·kg^{-1}·min^{-1}) - 0.042 (RE, ml·kg^{-1}·min^{-1}) + 4.778 [β-coefficients: $\dot{V}O_{2\,max}$ = 1.562; RE = -0.958]	0.93	0.86	0.82	0.04	0.8	<0.001
Low (n=10)	0.0291 ($\dot{V}O_{2\,max}$, ml·kg^{-1}·min^{-1}) + 2.781 [β-coefficients: $\dot{V}O_{2\,max}$ = 0.756]	0.76	0.57	0.52	0.15	3.3	0.011
Combined (n=36)	0.0554 ($\dot{V}O_{2\,max}$, ml·kg^{-1}·min^{-1}) - 0.0144 (RE, ml·kg^{-1}·min^{-1}) - 0.0208 (MRT_{off}, s) + 5.291 [β-coefficients: $\dot{V}O_{2\,max}$ = 0.876; RE = -0.566; τ_{off} = -0.210]	0.87	0.75	0.73	0.17	3.5	<0.001

where high (rank 1-10): MD n=2, LD n=8; low (rank 27-36): MD n=7, LD n=3.

Table 7.5 Stepwise regression models to identify physiological determinants of running performance in high, low and combined performers .

Group	Regression model	R	R^2	Adj R^2	SEE	SEE%	*P*
High	0.042 ($\dot{V}O_{2\ max}$, $ml \cdot kg^{-1} \cdot min^{-1}$) - 0.042 (RE, $ml \cdot kg^{-1} \cdot min^{-1}$) + 4.778	0.93	0.86	0.82	0.04	0.8	<0.001
(*n*=10)	[*β*-coefficients: $\dot{V}O_{2\ max}$ = 1.562; RE = -0.958]						
Low	0.0291 ($\dot{V}O_{2\ max}$, $ml \cdot kg^{-1} \cdot min^{-1}$) + 2.781	0.76	0.57	0.52	0.15	3.3	0.011
(*n*=10)	[*β*-coefficients: $\dot{V}O_{2\ max}$ = 0.756]						
Combined	0.0554 ($\dot{V}O_{2\ max}$, $ml \cdot kg^{-1} \cdot min^{-1}$) - 0.0144 (RE, $ml \cdot kg^{-1} \cdot min^{-1}$) - 0.0208	0.87	0.75	0.73	0.17	3.5	<0.001
(*n*=36)	(MRT_{off}, s) + 5.291						
	[*β*-coefficients: $\dot{V}O_{2\ max}$ = 0.876; RE = -0.566; τ_{off} = -0.210]						

where high (rank 1-10): MD *n*=2, LD *n*=8; low (rank 27-36): MD *n*=7, LD *n*=3.

7.4 Discussion

The purpose of this study was: 1) to explore the relationships between on- and off-transient $\dot{V}O_2$ kinetics and 5 km running performance and 2) to assess whether $\dot{V}O_2$ kinetics is a determinant of 5 km running performance. The main finding of this study was that moderate relationships (bi-variate) exist between $\dot{V}O_2$ kinetics and running performance and that parameters of $\dot{V}O_2$ kinetics contributed minimally to the prediction of running performance in endurance-trained runners.

Since some 'overlap' in running performance was evident between MD and LD runners (as highlighted by ANCOVA), a distorted regression model might have been produced for these groups. Potentially, this could cause a misleading interpretation of the results. To avoid this possibility, two groups of runners positioned at opposite ends of the performance continuum (i.e. high and low performers) were selected. This improved identification of physiological measures that determine running performance and/or discriminate between performers of high and low ability. Further ANCOVA on high and low performers confirmed the differences between these new groups (Table 7.4). The following interpretations of the results were made accordingly.

7.4.1 $\dot{V}O_2$ kinetics and running performance

The relationships between on- and off-transient $\dot{V}O_2$ kinetics and running performance were inconsistent and differed in magnitude between high, low and combined runners. Several $\dot{V}O_2$ kinetic parameters were found to correlate with running performance when combined runners (n=36) were assessed which suggests that runners with faster $\dot{V}O_2$ kinetics performed better during the 5 km time-trial. However, despite high performers having a significantly shorter τ_{on}, τ_{off}, MRT_{on} and MRT_{off} (Table 7.3 and 7.4) than low performers, correlations between $\dot{V}O_2$ kinetic parameters and running performance were not apparent *within* these groups. This suggests that physiological measures other than $\dot{V}O_2$ kinetics might be more important for success in running. Such inconsistencies in

relationships between these three groups could be attributable to various influences on correlation coefficients, such as the spread of the data (greater heterogeneity in combined runners) and different sample sizes (n=10 vs. n=36).

Despite these observations, there is some evidence to suggest that faster $\dot{V}O_2$ kinetics are related to improved running performance. Physiologically, a faster adjustment of $\dot{V}O_2$ at the onset of exercise - or to a sudden change in the intensity of exercise - after endurance training is likely to result in a decreased transient lactate production (Casaburi *et al.*, 1989), attenuation of PCr degradation (Phillips *et al.*, 1995) and consequently, less reliance on substrate phosphorylation. Collectively, these adaptations should result in the potential for improved running performance. In agreement, Poole and Richardson, (1997) have suggested that a reduction in O_2 deficit might lead to an increase in the time to exhaustion. A reduction in O_2 deficit is achieved by having faster $\dot{V}O_2$ kinetics and/or a decreased A_{on} for a given intensity of exercise. In support, Demarle *et al.* (2001) recently demonstrated that a reduction in the O_2 deficit (~34%), resulting from a shorter τ_{on} (~45%) and reduced A_{on} (~8%), was correlated with an increased time to exhaustion after training in MD and LD runners. This shows that faster $\dot{V}O_2$ kinetics after endurance training is linked to an improvement in running performance. This suggests that an individual's $\dot{V}O_2$ kinetics might be closely indicative of their performance capabilities over time.

7.4.2 $\dot{V}O_{2\,max}$, V_T, RE and running performance

The actual $\dot{V}O_{2\,max}$, V_T and RE values of runners (Table 7.3) compare favourably with previous studies of trained MD (Deason *et al.*, 1991) and LD runners (Powers *et al.*, 1985; Brandon and Boileau, 1992). In combined runners, $\dot{V}O_{2\,max}$ ranged from 49.0 to 70.1 $ml \cdot kg^{-1} \cdot min^{-1}$ which indicates a heterogeneous sample similar to that of previous studies involving runners (Costill *et al.*, 1973; Farrell *et al.*, 1979; Tanaka *et al.*, 1984; Conley and Krahenbuhl, 1980). The high and low performers were less heterogeneous than combined runners since the $\dot{V}O_{2\,max}$ ranged from 58.5 to 70.1 $ml \cdot kg^{-1} \cdot min^{-1}$ and 49.0 to 66.4

$ml \cdot kg^{-1} \cdot min^{-1}$ respectively. The $\dot{V}O_{2\,max}$ and V_T were greater in high performers than low performers (P = 0.005 and 0.002 respectively), which clearly suggests the groups were physiologically different. Given that the group of high performers consisted predominantly of LD runners (n=8), it is perhaps not surprising that they had a greater $\dot{V}O_{2\,max}$ and V_T than the group of low performers which consisted primarily of MD (n=7) runners. Several previous studies have shown that LD runners (runners that compete at events of 5000 m or longer) typically have higher $\dot{V}O_{2\,max}$ values, use O_2 more effectively and have lower lactate accumulation than MD runners (runners that compete at distances of 800 to 3000 m) (Costill *et al.*, 1976a; Conley and Krahenbuhl, 1980; Boileau *et al.*, 1982).

There was a relationship between $\dot{V}O_{2\,max}$ and 5 km running performance both in high and low performers (Figure 7.5). The magnitude of these relationships were similar in high (r = 0.76, P = 0.010) and in low performers (r = 0.76, P = 0.011). Both were higher than for combined runners (r = 0.66, P <0.001). This is surprising since the combination of all runners (n=36) represents a more heterogeneous sample than when the groups are treated separately and the correlation coefficient might be expected to be higher. However, in Figure 7.5, it can be seen that both groups are heterogeneous. Therefore, these correlations support previous studies where $\dot{V}O_{2\,max}$ has been identified as an important variable in running performance in similarly heterogeneous groups of runners (Costill *et al.*, 1973; Farrell *et al.*, 1979; Scott and Houmard, 1994). The moderate-to-high correlation between 5 km running performance and $\dot{V}O_{2\,max}$ both in high and low performers consolidates the importance of a high $\dot{V}O_{2\,max}$, especially since it has been shown that a high $\dot{V}O_{2\,max}$ is a pre-requisite for entry into the 'elite' category both for MD and LD runners (Pollock *et al.*, 1980).

A relationship between V_T and running performance was not observed for high or low performers but was observed when combined runners were considered (r = 0.62, P <0.001). The explanation for the lower correlation with running performance in high and low

runners, compared to combined runners, could be attributed to less heterogeneity in individual groups. In support, previous studies reporting poor correlations between V_T and running performance have tended to involve homogeneous groups of runners (Conley and Krahenbuhl, 1980).

The RE at a given sub-maximal running speed has been found to vary among trained runners and correlations with running performance range from r = 0.08 (Bulbulian *et al.*, 1986) to 0.83 (Conley and Krahenbuhl, 1980). In this study, RE was poorly related to 5 km performance in high, low and combined performers, regardless of the method of expression. In previous studies, poor correlations between RE and running performance have been primarily attributed to the heterogeneity of $\dot{V}O_{2\,max}$, which predominantly accounted for the differences in performance (Powers *et al.*, 1983; Bulbulian *et al.*, 1986). Similarly, it can be seen in Figure 7.5 that the $\dot{V}O_{2\,max}$ data, especially for low performers, is moderately heterogeneous and therefore it is perhaps not surprising that no relationship between RE and performance was observed in these runners.

7.4.3 Predicting performance in high and low performers

Several previous studies have investigated physiological variables that contribute to running performance, in particular 5 km running performance (Kumagai *et al.*, 1982; Tanaka *et al.*, 1984; Ramsbottom *et al.*, 1992). To date, no study has considered on- and off-transient $\dot{V}O_2$ kinetics as potential determinants of running performance and produced separate regression models for high and low performers.

Prior to multiple regression analyses, the original data for MD (n=18) and LD (n=18) runners were explored using ANCOVA. This revealed that MD and LD were not consistently different for each physiological measures and could be collapsed into one combined group (n=36). To remove any distorting influences of 'overlapping' data, runners were ranked and re-classified as high (rank 1-10) or low (rank 27-36) performers. Further

ANCOVA on high and low performers revealed that groups could be differentiated between using all physiological measures, including on- and off-transient $\dot{V}O_2$ kinetic parameters (P <0.001, Table 7.4). Subsequently, physiological and performance measures were entered into the stepwise multiple regression analysis. This revealed that the final models for high, low and combined performers were different. Specifically, for high performers, the regression model showed that $\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) and RE ($ml \cdot kg^{-1} \cdot min^{-1}$) were the primary contributors to the prediction of 5 km running performance, accounting for 85% of the variability in performance (Table 7.4). The standardised β-coefficients suggested that $\dot{V}O_{2\,max}$ was the strongest contributor to the model. The inclusion of RE was surprising since the bi-variate relationship between RE and performance in high performers was poor (Figure 7.7). The inclusion both of $\dot{V}O_{2\,max}$ and RE in high performers suggests that once a high $\dot{V}O_{2\,max}$ is attained, another measure (RE) is required to differentiate performers. The non-inclusion of other variables, in particular $\dot{V}O_2$ kinetic parameters, and V_T supports the lack of correlation between these measures and running performance in high performers (Figures 7.1 - 7.4 and 7.6 respectively).

The model for low performers only incorporated $\dot{V}O_{2\,max}$ (Table 7.4) which accounted for a modest 57% of the variability in performance. On the basis of the SEE% (3.3), this is a less accurate model for predicting 5 km running performance than that for high performers (SEE% = 0.8). Owing to the moderate-to-high correlation between $\dot{V}O_{2\,max}$ and running performance in low performers, the inclusion of this measure only in the final regression models was anticipated. This model would seem acceptable as low performers would still have the potential to increase $\dot{V}O_{2\,max}$ which would probably result in an improvement in performance.

When the original sample of MD and LD runners (n=36) were considered as a sample of 'endurance-trained' runners, the regression model (Table 7.4) differed and included a

parameter of $\dot{V}O_2$ kinetics (MRT_{off}) in addition to both $\dot{V}O_{2\,max}$ and RE. This clearly suggests that V_T is not an important predictor of running performance in a heterogeneous group of trained runners in this study. That $\dot{V}O_{2\,max}$ and RE were the primary contributors in all models, based on the size of the standardised β-coefficients, predicting running performance supports previous studies (Fay *et al.*, 1989; Weyand *et al.*, 1994) but is in contrast to the findings of Powers *et al.* (1983) who reported that the V_T accounted for 88% of variability of performance, whilst $\dot{V}O_{2\,max}$ and RE did not contribute to the improvement in R^2. It should be acknowledged that differences between the relationships and regression models in this study could be attributable to different sample sizes when comparing individual (n=10) and combined groups (n=36).

It is difficult to compare the models produced in this study with previous studies because of different combinations (Fay *et al.*, 1989; Weyand *et al.*, 1994) and expressions (Paavolainen *et al.*, 1999) of the physiological variables measured. However, the SEE% can be used to compare the accuracy of different models. In the present study, the SEE% were 0.8, 3.3 and 3.5 for high, low and combined runners respectively. As such, the model for high performers can be considered the most accurate model with which to predict 5 km running performance. In comparison, all models produced in this study are more sensitive than previous studies predicting running performance (5.7%, Housh *et al.*, 1988; 6.2%, Brandon and Boileau, 1992; 5.1%, Weyand *et al.*, 1994).

A likely explanation as to why measures of on-transient $\dot{V}O_2$ kinetics did not contribute to the final regression model could be attributed to some 'shared variance' with other aerobic physiological measures. This is plausible given the relationship between $\dot{V}O_{2\,max}$ and τ_{on} in LD runners (see Chapter 6). It is probable that there is some shared variance between these measures and that on-transient $\dot{V}O_2$ kinetics did not offer any additional information to the regression model. It is perhaps not surprising that MRT_{off} was the only $\dot{V}O_2$ kinetic measure included in the model to predict performance in combined runners since the

relationship with $\dot{V}O_{2\,max}$ in combined runners was poor i.e. no shared variance with $\dot{V}O_{2\,max}$. However, the inclusion of MRT_{off} should be interpreted with some caution since the size of the standardised β-coefficient indicates that the contribution of MRT_{off} to the final regression model, although significant, was smaller than both $\dot{V}O_{2\,max}$ and RE. Despite this, however, both r and R^2 were improved from 0.85 to 0.87 and 0.71 to 0.75 respectively when MRT_{off} was added to the model. Thus, MRT_{off} appears to be a useful addition to the model which offers information relating to the oxidative potential of muscle.

Running performance in low performers (predominantly MD runners) might have been achieved with substantial energy contributions from non-oxidative pathways which might have resulted in measures of aerobic function, especially $\dot{V}O_2$ kinetics and V_T, having minimal contribution to the prediction of performance. The variability in performance not accounted for by these aerobic parameters in low performers runners (43% un-accounted), might therefore be attributable to physiological characteristics of the performer which were not measured. In support, anaerobic (Bulbulian *et al.*, 1986; Houmard *et al.*, 1991), neuromuscular (Paavolainen *et al.*, 1999) and anthropometric (Housh *et al.*, 1986) characteristics have been found to differentiate runners according to their performance. It is possible that the addition of these measures to the multiple regression model for low performers could have improved predictive power (i.e. increased r and R^2).

Several studies have demonstrated that the volume (Sjödin and Jacobs, 1981; Roecker *et al.*, 1998) and intensity of training (Hagan *et al.*, 1981; Foster, 1983) contribute to the prediction of running performance. To assess this possibility, the relationship between self-reported training volume ($km \cdot wk^{-1}$) and 5 km performance was investigated. In this study however, models did not differ from the original models for high, low and combined performers.

7.4.4 Application of results

In a practical application, the overall findings suggest that moderately-trained competitive runners should adopt a training approach to improve their $\dot{V}O_{2\,max}$ and RE if their aim is to be successful at 5 km running. The benefit of increasing $\dot{V}O_{2\,max}$ has been demonstrated by di Prampero *et al.* (2000) who showed that a 5% increase can potentially result in a 3.9% improvement in 5 km running performance time. Furthermore, $\dot{V}O_{2\,max}$ and RE interact to determine the speed at which $\dot{V}O_{2\,max}$ is attained, i.e. $v\dot{V}O_{2\,max}$ (Daniels, 1985) or a percentage of $\dot{V}O_{2\,max}$, and thus determine the performance time that can be achieved. As a consequence of this interaction, $v\dot{V}O_{2\,max}$ is highly related to running performance (Morgan *et al.*, 1989).

A brief analysis of the influence of different training regimes in high and low performers suggests that the volume of training is not a contributing variable to the prediction of running performance. This is probably because the overall training stimuli (interaction of volume and intensity of training) are sufficient to promote the development of $\dot{V}O_{2\,max}$. The greater volume of training might also be reflected in a better RE (i.e. more economical technique and/or more efficient Type I fibres resulting in a lower $\dot{V}O_2$ for a given running speed). However, it should be acknowledged that information about the quantification of training in runners in this study was obtained from self-reported training diaries. Therefore, interpretation of findings on training volume should be viewed with caution.

7.5 Conclusion

The main findings of this study were two-fold. First, on- and off-transient $\dot{V}O_2$ kinetic parameters are moderately related to running performance in a heterogeneous group (n=36) of competitive endurance-trained runners (combined MD and LD runners). However, in less heterogeneous groups of high and low performing runners, relationships between $\dot{V}O_2$ kinetic parameters and running performance were not apparent. Second, multiple regression analyses demonstrated that variability in 5 km running performance is largely

accounted for by measures of aerobic function. Despite the ability of measures of $\dot{V}O_2$ kinetics to differentiate *between* groups of high and low performers, $\dot{V}O_2$ kinetic measures were not included in the regression models for differentiating performance *within* groups of high and low performers. However, $\dot{V}O_2$ kinetics contributed minimally to the model of running performance for combined runners. Specifically, $\dot{V}O_{2\,max}$ (high, low and combined performers) and RE (high and combined performers) were considered the primary determinants of performance, based on their contributions to the regression models. This finding suggests that $\dot{V}O_2$ kinetics cannot be considered an important determinant of running performance since they do not differentiate between a cross-sectional assessment of high and low performers. However, measures of $\dot{V}O_2$ kinetics might still be useful for longitudinal assessments of physiological adaptation(s) to training, and consequent changes in running performance, in *individual* athletes over time.

CHAPTER 8

Overall discussion

8.1 Discussion

The on- and off-transient $\dot{V}O_2$ kinetics during moderate-intensity treadmill running in MD and LD runners has received minimal consideration in the literature, especially in relation to running performance. Therefore, the overall aim of this thesis was to establish the importance of on- and off-transient $\dot{V}O_2$ kinetics for running performance.

First, and most importantly, the reproducibility of measures of on- and off-transient $\dot{V}O_2$ kinetics in MD and LD runners was determined. The reproducibility of measures has implications for the interpretation and meaningfulness for studies that aim to compare and quantify relationships between physiological and performance measures. This is especially important for measures of $\dot{V}O_2$ kinetics as inherent breath-by-breath variability can influence parameter estimations (Lamarra *et al.*, 1987). The results of the reproducibility study revealed that measures of on- and off-transient $\dot{V}O_2$ kinetics were satisfactorily reproducible, which suggested that a multiple-transition (n=6) protocol was appropriate for the assessment of $\dot{V}O_2$ kinetics in MD and LD runners. In comparison, previous studies reported poor reproducibility of their measures of on- (Kilding *et al.*, 2001; Özyener *et al.*, 2001; Puente-Maestu *et al.*, 2001) and off-transient $\dot{V}O_2$ kinetics (Özyener *et al.*, 2001). The reproducible measures of $\dot{V}O_2$ kinetics in this study meant that comparisons between MD and LD runners and relationships between physiological and performance measures could be quantified accurately and meaningfully interpreted.

The second study of this thesis characterised and compared $\dot{V}O_2$ kinetics in MD and LD runners during treadmill running. This study demonstrated faster on- and off-transient $\dot{V}O_2$ kinetics in LD runners. This was apparent despite similar $\dot{V}O_{2\,max}$. The primary explanation for this difference is attributed to different approaches to training adopted by MD and LD runners. Because physiological and biochemical adaptations

to training are specific to the training load[1] (Fox *et al.*, 1973; Harms and Hickson, 1983), different training regimes are also likely to be reflected in measures of $\dot{V}O_2$ kinetics.

Information relating to the training of MD and LD runners was collected which revealed that the volume of training[2] ($km \cdot wk^{-1}$) was greater in LD runners. Continuous training regimes are predominantly used by LD runners where total distance covered per session or per week is an important aspect of training overload. It has been reported that LD runners train more frequently at moderate-intensities (<85% $\dot{V}O_{2\,max}$), close to LT (Lacour *et al.*, 1990; Daniels and Daniels, 1992) whilst MD runners train and compete at high running speeds (>95% $\dot{V}O_{2\,max}$). Differences in the volume and intensity of training will determine the magnitude of physiological and biochemical adaptations which are likely to influence measures of aerobic (and anaerobic) function and running performance.

8.1.1 Adaptations to endurance training

Endurance training results in central (Fox *et al.*, 1975; Giada *et al.*, 1998) and peripheral (Hickson *et al.*, 1976; Harms and Hickson, 1983) adaptations which improve the delivery and diffusion and utilisation of O_2 to the exercising muscle. Specifically, central adaptations result from an improvement in the heart's ability to pump blood, mainly by increasing the stroke volume which occurs because of an increase in end-diastolic volume and an increase in left ventricular mass (Brooks *et al.*, 2000). These changes are induced by the increased volume load placed on the heart during endurance exercise. Subsequently, these adaptations result in an increased $\dot{Q}_{max}$, which, according to the Fick equation, will increase $\dot{V}O_{2\,max}$. The $\dot{V}O_{2\,max}$ has been described as an important characteristic of endurance athletes (Saltin and Åstrand, 1967) and its increase with training is well documented (Fox *et al.*, 1973; Hickson *et al.*, 1978). However, it is apparent that appropriate peripheral adaptations are also necessary for

[1] Training load refers to the frequency, duration and intensity of training.
[2] Volume of training is a composite measure of the frequency and duration of training

improved performance (Saltin *et al.*, 1976). The importance of peripheral mechanisms for improving running performance is emphasised when considering the similarity of recently reported $\dot{V}O_{2\,max}$ values of elite MD and LD runners (Billat *et al.*, 2001) compared to those reported in earlier studies (Saltin and Åstrand, 1967), despite substantial performance differences. Furthermore, in already well-trained runners, $\dot{V}O_{2\,max}$ has been found to be a relatively stable feature of an athlete's physiological profile (Daniels, 1974), despite further improvements in running performance. This suggests that central adaptations to endurance training are not exclusively limiting running performance. Consistently, throughout this thesis, $\dot{V}O_{2\,max}$ was similar between MD and LD runners, despite different performance levels, suggesting that their peripheral adaptation to training differed. This is supported by faster on- and off-transient $\dot{V}O_2$ kinetics in LD runners. These findings suggest that: 1) O_2 delivery (or central) mechanisms do not determine the rate of $\dot{V}O_2$ kinetics at the onset of moderate-intensity exercise (Grassi *et al.*, 1998a, b) and 2) performance differences between MD and LD runners are due to peripheral mechanisms within the muscle. It is therefore necessary to consider specifically, the peripheral adaptations to training.

8.1.1.1 Peripheral adaptations to training

It is well established that endurance training results in major adaptations in skeletal muscle. Such changes include: 1) increased myoglobin (Hickson, 1981); 2) increased mitochondrial size and number (Kiessling *et al.*, 1971); 3) increased oxidative enzyme activity (Gollnick *et al.*, 1973); 4) altered muscle fibre composition (Henriksson and Reitman, 1977) and 5) preferential use of FFA as an energy substrate (Holloszy, 1973; Holloszy and Coyle, 1984). These peripheral adaptations may be of limited importance for whole-body $\dot{V}O_{2\,max}$, since maximum oxidative power (defined as the maximum rate of oxidative phosphorylation in muscle) is in excess of what is required during two-legged exercise (Anderson and Saltin, 1985). However, it is likely that an increase in muscle aerobic potential plays a major role in the increased endurance and the reduced metabolic perturbation observed after aerobic training (Saltin and Gollnick, 1983).

The adaptation of skeletal muscle metabolism in response to aerobic training results in tighter coupling between ATP supply and demand (Dudley *et al.*, 1987) and is characterised by a lesser increase in free ADP, AMP, IMP, Cr and P_i by a lesser decrease in PCr. Consequently, there is a smaller perturbation of the cytosolic phosphorylation potential in response to a change in the intensity of exercise. In addition, tighter integration of ATP supply and demand is associated with less stimulation of glycolysis, resulting in a decrease in lactate production and glucose utilisation, a lower cytosolic redox state, and an improved coupling between pyruvate oxidation and glycolytic flux (Holloszy and Coyle, 1984). One of the main mechanisms thought to be involved in the tighter coupling of ATP supply and demand is the improvement of muscle oxidative capacity which is brought about by an increase in mitochondrial volume density and in the activity of several enzymes of oxidative metabolism. Specifically, it is these improvements that are likely to influence $\dot{V}O_2$ kinetics and account for differences between MD and LD runners. Also, since the distribution of Type I fibres has been shown to be superior in LD runners, compared to MD runners (Saltin and Gollnick, 1983), this will influence the potential magnitude of the increase in mitochondrial density and oxidative enzyme activity in the muscle. As a consequence, it could be anticipated that faster $\dot{V}O_2$ kinetics would be observed in LD runners. However, this would only be apparent if the magnitude of the training load was sufficient.

8.1.2 Endurance training: frequency, duration and intensity

The magnitude of cardiovascular (Wenger and Bell, 1986) and biochemical (Hickson, 1981) adaptations to endurance training is influenced by the frequency (Hickson, 1981), duration (Fox *et al.*, 1975; Hickson *et al.*, 1976) and intensity of training (Hickson *et al.*, 1976; Harms and Hickson, 1983). Thus, differences between the $\dot{V}O_2$ kinetics of MD and LD runners might be a reflection of differential changes in the metabolic characteristics of the different fibre types induced by various approaches to training. Indeed, longitudinal studies have shown that the increased oxidative potential of a muscle, by augmentation of mitochondrial enzyme activity, capillary density and

enhancement of FFA oxidation, is localised in the fibres most active in the training programme and occurs in both Type I and Type II fibres (Henriksson and Reitman, 1977).

One noticeable difference between the training of MD and LD runners was that the volume of training (self-reported) was greater in LD runners. In addition, the volume of training was related to τ_{on} in both MD and LD runners. This suggests that an increased volume of training is a stimulus for faster $\dot{V}O_2$ kinetics. To support this possibility, oxidative adaptations to training in muscle fibres have been shown to be proportional to the volume of training (Sjödin *et al.*, 1976; Terjung, 1976). Specifically, increased mitochondrial content has been shown to be related to the frequency and duration of endurance training (Fitts *et al.*, 1975; Hickson, 1981). It is therefore anticipated that the potentially greater mitochondrial content in LD runners as a result of a greater volume of training would influence $\dot{V}O_2$ kinetics. However, muscle biopsies would be necessary to confirm this possibility.

As an alternative to considering the physiological effects of manipulating the volume of training, Henriksson and Reitman (1976) found that high-intensity interval training carried out at maximal intensity resulted in a 20-30% increase in SDH activity. Analysis of single fibres, however, showed that high-intensity training increased SDH activity in Type II fibres by ~50% with no increase in SDH activity in Type I fibres. Conversely, sub-maximal continuous training resulted in a 30% increase in SDH activity of Type I fibres, with no change in SDH activity of Type II fibres. This confirms that training-induced adaptations in muscle fibres are intensity specific and that this could explain why $\dot{V}O_2$ kinetics of MD and LD runners differ. Only one study has considered the effects of different training intensities on $\dot{V}O_2$ kinetics during the on-transient (Berry and Moritani, 1985). Unfortunately, this study only investigated training intensities that would be representative of a LD runner's training and did not consider high-intensity training. However, the differentiation of $\dot{V}O_2$ kinetics between

elite distance runners and elite sprinters (Edwards *et al.*, 1999) suggests that the volume and intensity of training are important determinants of $\dot{V}O_2$ kinetics.

Whether adaptations induced by anaerobic training have a positive or negative effect on $\dot{V}O_2$ kinetics is not yet known. Several studies have reported that sprint training has a positive effect (Saltin *et al.*, 1976; MacDougall *et al.*, 1998) on mitochondrial enzyme activity, but less than that induced by endurance training (MacDougall *et al.*, 1998). Consequently, the time spent training at high-intensities by MD runners will reduce the opportunity for aerobic adaptations to take place. Ultimately, this will result in less speeding of $\dot{V}O_2$ kinetics. In addition, high-intensity training could result in adaptations that are antagonistic to those promoting oxidative phosphorylation in muscle. One such mechanism involves potential increases in [PCr] and [Cr] observed with high-intensity training (Parra *et al.*, 2000) and/or oral Cr supplementation (Greenhaff *et al.*, 1994). Experimental findings in rats demonstrated that CS activity increased after a reduction in [Cr] (Sweeney, 1994) and that PCr re-synthesis is speeded by [Cr] depletion (Fitch *et al.*, 1979). Sweeney (1994) suggested that a greater [Cr] would result in a slower mitochondrial turn on. In support, Meyer and Foley (1994) have demonstrated that the rate of oxidative phosphorylation is linearly dependent on total Cr ([PCr]+[Cr]). Collectively, these findings suggest that $\dot{V}O_2$ kinetics might be slowed in MD runners as a result of increases in [PCr] or [Cr], in response to regular high-intensity training and/or oral supplementation.

8.1.3 Endurance training: mitochondrial number and function

Endurance training results in an increase in the mitochondrial content of the cell (Holloszy and Coyle, 1984; Dudley *et al.*, 1987) and this alters the mitochondrial sensitivity to the regulators of respiration (Dudley *et al.*, 1987). The mitochondrial content itself has been shown to play a important role in controlling oxidative phosphorylation (Dudley *et al.*, 1987; Burelle and Hochachka, 2002). Accordingly, a greater increase in mitochondrial number and function in LD runners compared to MD runners, brought about by different training stimuli, could be responsible for the

differentiation of $\dot{V}O_2$ kinetics between MD and LD runners. Corroborating this suggestion, muscle mitochondrial content has been considered as an important determinant of $\dot{V}O_2$ kinetics (Whipp and Mahler, 1980) and also plays an important role in Meyer's (1988) linear model of respiratory control. Overall, the findings of faster $\dot{V}O_2$ kinetics in LD runners would support the importance of mitochondria for oxidative phosphorylation.

In this study, there was clear asymmetry between on- and off-transient $\dot{V}O_2$ kinetics in MD and LD runners. However, on- and off-transient $\dot{V}O_2$ kinetics were related suggesting that each transient was representative of training status. This is useful information because the rate of PCr re-synthesis during the immediate post-exercise period has been considered a valid *in vivo* measure of muscle oxidative power in MD (McCully *et al.*, 1992) and LD runners (McCully *et al.*, 1992; Yoshida and Watari, 1993). Because dynamic symmetry exists between PCr and $\dot{V}O_2$ kinetics during the off-transient (Rossiter *et al.*, 2002), measures of off-transient $\dot{V}O_2$ kinetics could provide a useful representation of the oxidative capacity of the exercising muscle(s). Evidence to suggest that faster off-transient $\dot{V}O_2$ kinetics in LD runners is attributable to greater muscle oxidative capacity is clearly provided by the findings of Paganini *et al.* (1997) who demonstrated a linear dependence of muscle PCr kinetics on oxidative capacity in rats. These authors also reported a strong relationship (r = 0.84, P <0.01) between PCr kinetics during recovery and CS activity after training. Furthermore, PCr kinetics were subsequently slowed by reducing the mitochondrial content by chemical thyroidectomy. These findings support the potential usefulness of off-transient $\dot{V}O_2$ kinetics as a measure of muscle oxidative capacity in humans, especially athletes. However, similar to the on-transient, a further study is necessary to clarify the influence of various training regimes on off-transient $\dot{V}O_2$ kinetics.

8.1.4 Muscle fibre type composition

The degree of mitochondrial adaptation to training together with the associated increase in oxidative enzyme activity, will be largely determined by the composition and

characteristics of the muscle fibres. In humans, skeletal muscle is not homogeneous; its fibre types differ according to their morphological and biochemical properties. According to their myosin heavy-chain gene expressions, three different fibres - Type I, Type IIa and Type IIx - have been identified in human skeletal muscle (Ennion *et al.*, 1995).

In previously untrained humans, the mitochondrial content of Type I fibres has been reported to be 50% greater than that in Type IIa fibres and three times greater than that in Type IIx fibres after endurance training (Howald *et al.*, 1985). Therefore, a runner with a greater percentage of trained Type I fibres and subsequently a higher mitochondrial content, would be anticipated to display faster $\dot{V}O_2$ kinetics. In support, Kushmerick *et al.* (1992) reported NMR data on working isolated Type I and Type II muscle fibres and demonstrated that the energy phosphate kinetics of Type I fibres are approximately twice as rapid as Type II fibres. Because there is a tendency for LD runners to have a greater percentage of Type I fibres (Costill *et al.*, 1976), the potential for faster $\dot{V}O_2$ kinetics, assuming appropriate physical training has been completed, is greater in runners with a genetic predisposition to a high percentage of Type I fibres. This supports the findings of Barstow *et al.* (1996) who clearly demonstrated the effect of muscle fibre Type I percentage on $\dot{V}O_2$ kinetics (Figure 8.1). However, runners with predisposition to a higher percentage of Type I fibres might not necessarily be those classified as LD runners in this study. This could explain the greater variability and short τ_{on} and τ_{off} in some MD runners as well as the inconsistent relationships between $\dot{V}O_2$ kinetics and other physiological measures in MD runners, compared to LD runners. Future studies should consider the actual differences between the athletes as an influencing factor and one that could affect the interpretation of results. To ensure that MD and LD were two very distinct groups, comparisons between MD and LD runners would probably be confined to those performing at national and international level.

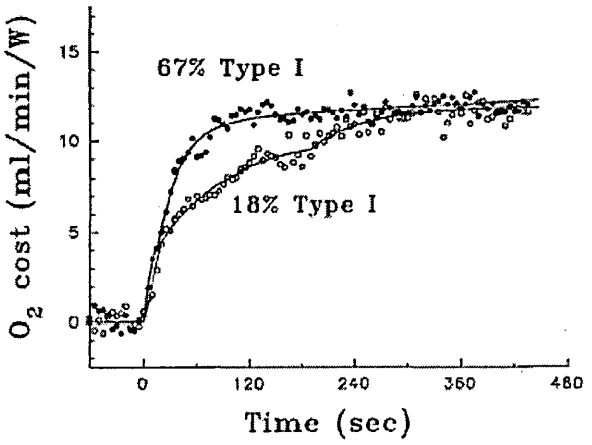


Figure 8.1 The influence of muscle fibre type percentage on $\dot{V}O_2$ kinetics during heavy-intensity exercise in two participants (from Barstow *et al.*, 1996).

Heavy-intensity endurance training increases the mitochondrial content in all fibres types, but is greatest in Type IIa fibres (Howald *et al.*, 1985). Potentially, a greater oxidative capacity of Type IIa fibres in MD compared to LD runners could account for the slower $\dot{V}O_2$ kinetics but similar $\dot{V}O_{2\,max}$ values between MD and LD runners. For example, a MD runner with a moderate percentage of Type I fibres but a high percentage of oxidative Type IIa fibres could have a similar $\dot{V}O_{2\,max}$ but slower $\dot{V}O_2$ kinetics compared to a LD runners with a higher percentage of Type I fibres but a lower percentage of Type IIa fibres. The enhanced oxidative capacity of Type IIa fibres might also explain the poor relationship between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics in this group. This is because the recruitment of Type IIa fibres would contribute substantially to measures of $\dot{V}O_{2\,max}$ but minimally to measures of $\dot{V}O_2$ kinetics in the moderate domain. It is also possible that the oxidative capacity of some Type IIa fibres is not too dissimilar to some of the Type I fibres.

Differences in muscle fibre type might explain the faster off-transient $\dot{V}O_2$ kinetics observed in LD runners. Tesch *et al.* (1989), described that [PCr] 60 s after exercise was greater in Type I fibres than Type II fibres which suggests that the rate of PCr re-synthesis (and presumably τ_{off}; Rossiter *et al.*, 2002) was faster in the Type I fibres. However, the importance of Type IIa fibres for endurance running performance should

not be ignored. Weston *et al.* (1999) suggested that rather than being detrimental to endurance performance, Type IIa fibres could serve as important power generators at high running speeds. Although Type IIa fibres are generally less efficient at using oxygen than Type I fibres (Kushmerick *et al.*, 1992), it is possible that Type IIa fibres exhibit considerable variability in their oxidative capacity and that a continuum of oxidative potential of fibres from Type I to Type IIx probably exists. To some extent, the composition of muscle fibres will be determined by characteristics of the training load.

One explanation as to why $\dot{V}O_2$ kinetics did not consistently correlate with $\dot{V}O_{2\,max}$, V_T or RE in MD runners might be attributed to the potential heterogeneity of muscle compartments. This possibility has led some authors to consider not what the apparent kinetic τ_{on} reveals, but what it might conceal (Whipp *et al.*, 2002). For example, two individuals could display the same overall τ_{on} despite having different compartment profiles. That is, one individual could display a homogeneous profile whereby τ_{on} differs very little among compartments, whereas the other could have a large distribution of τ_{on} values amongst compartments, but the overall τ_{on} could be similar. In the latter, metabolic stress would be greater in those compartments displaying slower kinetics and this would, therefore, lead to an increased demand for supplemental regional energy transfer from lactate yielding mechanisms, resulting in different regional V_T, despite the same average τ_{on} (Whipp *et al.*, 2002). If such heterogeneity does exist, then this could explain: 1) the "overlap" in τ_{on} (and MRT_{on}) in MD and LD runners and 2) the weak correlations between on- and off-transient $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$, V_T and RE in MD runners.

8.2 The importance of $\dot{V}O_2$ kinetics to running performance

In the present study, the relationship between $\dot{V}O_2$ kinetics and 5 km running performance and the contribution of $\dot{V}O_2$ kinetics to the prediction of running performance was small compared to other measures of aerobic function, particularly $\dot{V}O_{2\,max}$. However, if phase II $\dot{V}O_2$ kinetics are slower above V_T (Carter *et al.*, 2002), it

is possible that the contribution of τ to the regression model might vary depending on the intensity of exercise used to measure $\dot{V}O_2$ kinetics. This might have some implications for the findings of the present study and should be considered.

In the present study, measures of $\dot{V}O_2$ kinetics were obtained during moderate-intensity exercise only. In this intensity domain, it would appear that $\dot{V}O_2$ kinetics provide non-invasive, peripheral information reflecting the oxidative function of Type I muscle fibres which predominate in endurance-trained runners (Costill *et al.*, 1976b; Saltin and Gollnick, 1983). If however, as suggested by Carter *et al.* (2002), $\dot{V}O_2$ kinetics in the heavy-intensity domain involves the progressive recruitment of less O_2 efficient Type II fibres, resulting in a longer τ, then information about the oxidative function of a greater proportion of fibres (including Type II fibres) would be gained. In addition, because O_2 transport mechanisms have been suggested to influence τ during heavy-intensity exercise (Tschakovsky and Hughson, 1999), the body's ability to deliver O_2 to exercising muscle could also be reflected. Collectively, these factors might increase the contribution of τ to predict running performance in heavy-intensity exercise compared to moderate-intensity exercise. However, one clear disadvantage of using heavy-intensity exercise is that it would be impossible to partition peripheral and central factors to determine their contribution to the performance model. Therefore, if future studies clearly identify differences in τ above and below V_T, then further studies applying measures of $\dot{V}O_2$ kinetics to performance should consider measuring τ both above (reflecting Type I and II fibres and O_2 delivery) and below (reflecting Type I fibres) V_T to obtain a separate description of the oxidative function of muscle and the body's ability to deliver O_2 during exercise of different intensities. However, if τ is invariant across intensity-domains (Barstow *et al.*, 1993; Özyener *et al.*, 2001; Wells *et al.*, 2003), measures of τ in the moderate-domain are justified to reflect changes in overall physiological status since no additional information would be obtained from determining τ during heavy-intensity exercise.

Whilst acknowledging the comparatively small contribution of $\dot{V}O_2$ kinetics to the prediction of performance in combined MD and LD runners (n=36), faster $\dot{V}O_2$ kinetics in any runner, regardless of the intensity in which they are measured, will have specific advantageous functional and metabolic consequences. For example, faster $\dot{V}O_2$ kinetics resulting from increased mitochondrial content will improve respiratory control which will reduce non-oxidative ATP supply (substrate-level phosphorylation) and increase oxidative phosphorylation at the onset of exercise. Consequently, this will result in some muscle glycogen sparing. However, during the actual transient, the amount of energy conserved by faster $\dot{V}O_2$ kinetics would be small. For example, using physiological data from two athletes, one with fast (τ_{on} = 9.0 s; MRT_{on} = 23.4 s; A_{on} = 1869 ml·min^{-1}) and the other with slow (τ_{on} = 20.7 s; MRT_{on} = 34.7 s; A_{on} = 2167 ml·min^{-1}) $\dot{V}O_2$ kinetics [participants 5 (MD) and 2 (LD), pp254], the calculated O_2 deficit (MRT_{on} x A_{on}) was 845 and 1081 ml respectively. As an approximation (RER = 0.96), the energy equivalent of O_2 amounts to 20.9 kJ·l^{-1} (di Prampero, 1986). Therefore the amount of energy from anaerobic sources during the transient would be equal to 17.7 and 22.6 kJ. The difference amounting to 4.9 kJ. This conservation of energy would be of negligible benefit to running performance.

It would appear that it is not the conservation of energy (i.e. reducing the O_2 deficit) resulting from faster $\dot{V}O_2$ kinetics that is important for running performance. The status of several physiological factors, such as mitochondrial density and oxidative enzyme activity, which operate continuously throughout exercise of any intensity are likely to be the most important *peripheral* factors influencing running performance, and it is these factors which determine $\dot{V}O_2$ kinetics in the moderate-domain (Whipp and Mahler, 1980). The faster $\dot{V}O_2$ kinetics observed after endurance training, primarily resulting from an increased number and function of mitochondria and increased oxidative enzyme activity, occurs concomitantly with a reduction of intra-muscular lactate accumulation (Yoshida *et al.*, 1992; Phillips *et al.*, 1995). Collectively, these peripheral adaptations will allow higher intensities of exercise to be attained with lower

[HLa]. Ultimately, this will have positive influence on moving the V_T/LT to a higher running speed, which would be of great advantage in MD and LD running events.

The importance of measuring $\dot{V}O_2$ kinetics was more apparent in combined runners, than in high and low performers separately as there was consistently stronger relationships between τ_{on} and running performance. Furthermore, MRT_{off}, which can also be considered a measure of muscle oxidative capacity (McCully *et al.*, 1992), was a contributor to 5 km running performance. Based on these findings, schematics summarising the relationships between physiological adaptations to training and the principal measures determining 5 km running performance in high (Figure 8.2), low (Figure 8.2) and combined runners (Figure 8.3) were produced.

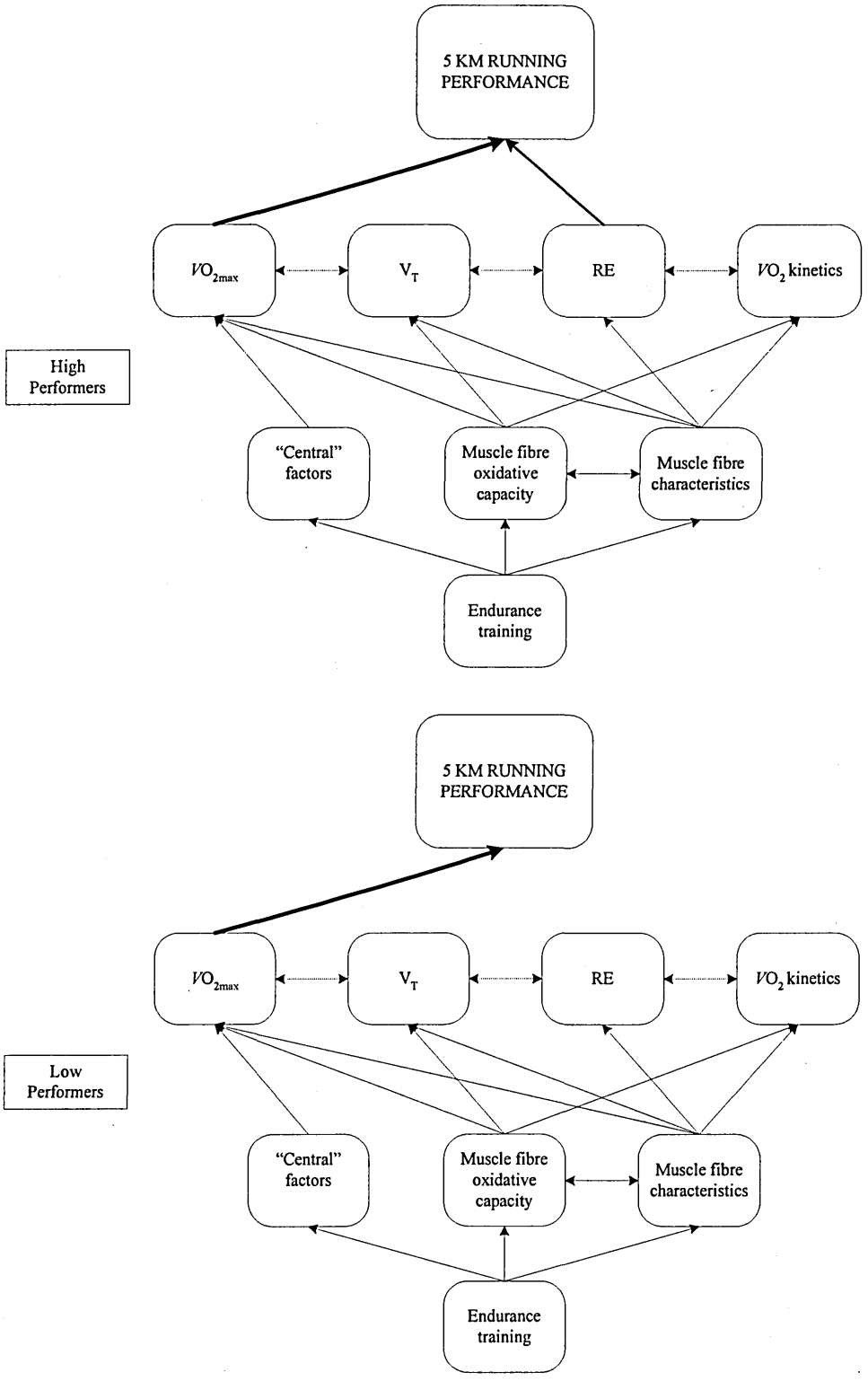


Figure 8.2 Models illustrating the physiological and biochemical factors influencing $\dot{V}O_2$ kinetics in the moderate-intensity domain and the contribution of measures of aerobic function to the prediction of 5 km running performance in high and low performing runners[3].

[3] At the upper level of each model, the thickness of the line denotes the contribution to the prediction of running performance (see Table 7.5 for actual values).

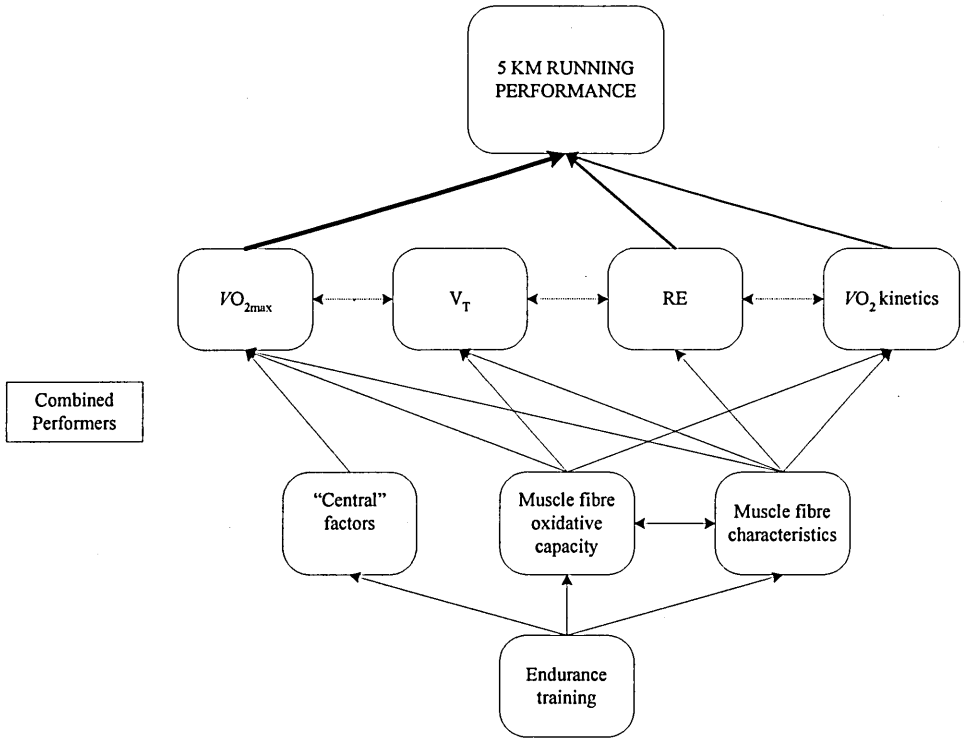


Figure 8.3 Model illustrating the physiological and biochemical factors influencing $\dot{V}O_2$ kinetics in the moderate-intensity domain and the contribution of measures of aerobic function to the prediction of 5 km running performance in combined MD and LD runners (n=36).

8.3 Limitations

The inherent difficulty in classifying runners as exclusively MD or LD specialists could confound comparisons between groups of runners. In the present study, runners were classified according to their self-selected discipline, although in some instances there was a tendency for this standard of runner to compete in a range of running events that were shorter and longer than their specialist event. For this reason, some runners might not have been physiologically too dissimilar and attempts to differentiate them on the basis of their physiological measures, especially $\dot{V}O_2$ kinetics, might have been confounded. To attenuate any potential effects when assessing performance measures, ANCOVA was used to compare groups and where appropriate MD and LD were collapsed and re-assigned as high and low performers (see Chapter 7 for details). Further ANCOVA was performed on the new groups to explore which measure(s) differentiated between high and low performers.

To account for the differences between MD and LD runners, characteristics of their training were considered. Unfortunately, only self-reported, quantitative information relating to the volume of training was collected. A more detailed and comprehensive analysis of the frequency, intensity and duration of training completed by MD and LD runners in this study (e.g. specific nature of intervals sessions and intensity of continuous runs) would have been beneficial to the interpretation of the findings. Specifically, this would allow further conclusions to be made about the physiological and biochemical adaptations to different intensities of training in MD and LD runners and its subsequent effect on $\dot{V}O_2$ kinetics and performance (see section 8.4 for future directions).

The differences between MD and LD runners and the inconsistent relationships between physiological measures have been attributed to mitochondrial number and function, oxidative enzyme activity and muscle fibre type compositions. However, no direct measures of these important characteristics were completed to consolidate and/or confirm the findings of the present studies. Furthermore, the addition of these measures might have been strongly related to running performance and contributed to the multiple regression analysis, thus resulting in a more accurate regression model.

8.4 Future directions

The findings and methodological limitations of this thesis have revealed some potentially useful areas for future research to explore. Further studies would be useful to gain a clearer understanding of the sensitivity of $\dot{V}O_2$ kinetics to different training regimes and the usefulness of $\dot{V}O_2$ kinetics in physiological assessments of runners and related performance.

The recent findings of Carter *et al.* (2002), showing that τ_{on} is slowed for heavy compared to moderate-intensity exercise, should be considered before further research involving measures of $\dot{V}O_2$ kinetics exclusively in the moderate-domain is conducted.

If τ_{on} is not invariant for treadmill exercise across intensity domains below and above V_T, then it might be necessary to measure $\dot{V}O_2$ kinetics over a range of moderate, heavy and severe exercise intensities, especially if an overall measure of O_2 *delivery* and *utilisation* is required. This 'profiling' of $\dot{V}O_2$ kinetics in athletes would then span the actual intensities experienced in different MD and LD running events. This wider ranging approach might also provide further information about the physiological and biochemical adaptation(s) occurring in Type II muscle fibres, as these would be progressively recruited, according to Carter *et al.* (2002), during exercise in the heavy- and severe-domains. This might increase the potential usefulness of $\dot{V}O_2$ kinetics as a measure of physiological status in MD and LD runners during training and elucidate whether meaningful relationships exist between $\dot{V}O_2$ kinetics and running performance. However, O_2 delivery mechanisms would also need to be considered for exercise above V_T.

The first proposed future study would be to assess directly the sensitivity of $\dot{V}O_2$ kinetics to training stimuli via a controlled training intervention study lasting approximately 6-8 weeks. Specifically, the aim of this study would be to test the hypothesis that $\dot{V}O_2$ kinetics are faster after volume-orientated training compared to high-intensity training. Initially, this study would involve non-runners as this might highlight more clearly the effects of different training loads (intensity and volume) on $\dot{V}O_2$ kinetics. This type of study would require three separate groups: 1) a high volume - low intensity group; 2) a high intensity - low volume group; 3) a control/sedentary/un-trained group. The actual number of individuals in each group could be determined based on a calculation to determine statistical power, usually 80% (Vincent, 1995). The calculation considers 1) the effect size (1.25 s, obtained from pre- and post-training differences from two previous studies; Norris and Peterson, 1998; Carter *et al.*, 2000b); 2) the reliability of the measure (r = 0.92, obtained from the test-retest correlation in study one of this thesis) and 3) the number of repeated measures (n=3) (Park and Shultz, 1999). Using the above data, each group would consist of 13 participants. Initially, it would be necessary to establish baseline (pre-training, week 0) data for each

physiological and performance measure. Using this approach, the volume and intensity of training can be accurately prescribed for each participant. The training for the high volume - low intensity group would consist of frequent, continuous bouts of sub-maximal running at a relative intensity according to pre-training measures of V_T and $\dot{V}O_{2\,max}$ - characteristic of training by LD runners. Conversely, the high intensity - low volume group would primarily perform shorter, supra-maximal bouts of exercise indicative of a MD runners training. To ensure an adequate and continuous overload, the training volume and intensity would be re-assessed and progressively increased, if appropriate, at a mid-training physiological and performance assessment (week 3-4). Finally, post-training measures would be taken to establish the overall effect of 6-8 weeks of volume or intensity orientated training. Statistical analysis for three repeated measures (pre-, mid- and post-training) in three different groups (volume, intensity and control) would require a mixed factorial ANOVA design. Any differences could be identified during a *post-hoc* analysis of the data.

A training intervention study of this design would consolidate findings of this thesis by determining whether 1) a greater volume of training speeds $\dot{V}O_2$ kinetics more than high-intensity training. A greater volume ($km \cdot wk^{-1}$) of training in LD runners compared to MD runners was consistently observed throughout this thesis. However, it is acknowledged that this information was obtained from self-reported training diaries; 2) high-intensity training disassociates $\dot{V}O_2$ kinetics from $\dot{V}O_{2\,max}$ and V_T. More specifically, does high-intensity training influence the relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function (especially since no relationships were observed in MD runners in this thesis)?; 3) differing physiological and biochemical adaptations in response to aerobic vs. anaerobic training have potential antagonistic effects on $\dot{V}O_2$ kinetics. This possibility might account for differences in $\dot{V}O_2$ kinetics between MD and LD runners observed throughout this thesis and 4) an inverse relationship exists between $\dot{V}O_2$ kinetics and anaerobic capacity in muscle.

In this thesis, measures of $\dot{V}O_2$ kinetics were only moderately-related to running performance in combined runners (τ_{on}: r = -0.54, P = 0.001; MRT_{on}: r = -0.50, P = 0.002; τ_{off}: r = -0.36, P = 0.030; MRT_{off}: r = -0.63, P = 0.003). However, the potential of $\dot{V}O_2$ kinetics to reflect the physiological status of an *individual* athlete over time might be more relevant and meaningful to explain variations in running performance. Similar conclusions about peripheral adaptation to training were drawn by Foster *et al.* (1978) who suggested that skeletal muscle metabolism apparently contributes little to cross-sectional differences in performance, but might be of greater importance to individual variations in performance. Indeed, small improvements in physiological measures over time might potentially translate into significant improvements in running performance. To test the hypothesis that on- and off-transient $\dot{V}O_2$ kinetics are sensitive to varying training loads and reflect performance in MD and LD runners, a longitudinal study of runners would be required. This would assess the sensitivity of $\dot{V}O_2$ kinetics, and other physiological and biochemical adaptations, to different training loads during a typical training and competitive year. For example, the preparatory phase of training, when the emphasis of training is to establish an aerobic base, is characterised by an increased volume of training (Martin and Coe, 1991). After this period of training a runner might be expected to display faster $\dot{V}O_2$ kinetics than those seen towards the end of the competitive season when the volume of training is lower but the intensity is higher. Thus, a longitudinal approach that monitors MD and LD runners several times throughout a typical training and competitive year (~44 weeks) might elucidate more clearly the precise effects of different training loads on $\dot{V}O_2$ kinetics. This study would require physiological measures before and after each training and competitive phase. To quantify the volume (frequency and duration) and intensity of training for each training phase, detailed training diaries from each athlete would be required. Since the magnitude of change in $\dot{V}O_2$ kinetics has not been established for already trained runners, determination of the sample size for this study is difficult to ascertain. However, since potential changes are likely to be smaller, the sample size required for optimal statistical power is likely to be greater than that observed for

previously untrained individuals. Some pilot work to estimate the expected change in τ_{on} with different types of training would be required to estimate an appropriate sample.

In this thesis, MD and LD runners could be differentiated on the basis of their on- and off-transient $\dot{V}O_2$ kinetics, despite some overlap. Primarily, differences were attributed to varying mitochondrial function, oxidative enzyme activities and muscle fibre characteristics induced by different approaches to training. However, these muscle fibre characteristics were not measured and therefore no direct evidence to support this suggestion was available. Clearly, there is a need to investigate these measures so that differences between MD and LD runners can be more accurately explained. A suitable hypothesis for this study would be that differences in $\dot{V}O_2$ kinetics between MD and LD runners are attributable to fibre type and biochemical differences in muscle. The additional biochemical measures might also aid interpretation and clarify the inconsistent inter-relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function ($\dot{V}O_{2\,max}$ and V_T) that were apparent in MD runners in study three of this thesis. This proposed study would involve the measurement of $\dot{V}O_2$ kinetics, $\dot{V}O_{2\,max}$, V_T and RE as well as a muscle biopsy in MD (n=8) and LD (n=8) runners [where n is calculated from an effect size of 1.3 (determined form the differences and SD between τ_{on} in MD and LD runners in study three of this thesis) and statistical power of 0.80 using the equation of Lenth, 2001)]. It would be important that these measures were taken in close proximity to each other. However, the duration of this study would be largely dependent on athlete availability. From the muscle biopsy, fibre type composition, mitochondrial content and oxidative and glycolytic enzyme activity could be established. This would permit the relationships between $\dot{V}O_2$ kinetics and physiological and biochemical characteristics of muscle to be explored more meaningfully. Particularly, this would clarify whether a relationship exists between muscle fibre composition and $\dot{V}O_2$ kinetics in MD and LD runners. More important, however, is whether the proportion and composition of Type IIa and IIx, as influenced by high-intensity training, can account for the distorted relationship between $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$ and V_T observed in MD runners (study three). Collectively, this

would ensure that the proposed physiological and biochemical explanations for the findings of the present studies are acceptable and would support the separation of endurance-trained runners into their preferred distance. This cross-sectional study could also be expanded into an intervention study as previously described for untrained individuals. This would involve the pre- and post-training measures of $\dot{V}O_2$ kinetics and muscle biopsies. However, the time course of adaptation in muscle fibre composition would require further consideration. Based on several previous studies, highlighted by Saltin and Gollnick (1983), 6 - 8 weeks of training would probably be sufficient to observe changes in muscle.

8.5 Conclusion

To summarise, the findings of this thesis have established that: -

1. Pulmonary on- and off-transient $\dot{V}O_2$ kinetics in the moderate-domain can be reproducibly determined using a multiple-transition protocol.
2. MD and LD runners differ in their on- and off-transient $\dot{V}O_2$ kinetic responses in the moderate-intensity domain.
3. On- and off-transient $\dot{V}O_2$ kinetics in the moderate-intensity domain are related to other aerobic measures ($\dot{V}O_{2\,max}$, V_T and RE), primarily in LD runners.
4. Pulmonary $\dot{V}O_2$ kinetics, in the moderate-intensity domain, are faster in high performers than low performers but are not related to 5 km running performance.
5. Off-transient pulmonary $\dot{V}O_2$ kinetics in the moderate-intensity domain contribute minimally to a multiple regression model predicting running performance in combined runners when $\dot{V}O_{2\,max}$, V_T and RE are also considered. However, pulmonary $\dot{V}O_2$ kinetics do not contribute to successful running performance *within* groups of high and low performers.

This thesis has revealed several novel findings which contribute to the body of knowledge with respect to 1) relationships between measures of aerobic performance and 2) methodological aspects of measuring $\dot{V}O_2$ kinetics. This is the first study to quantify the reproducibility and day-to-day variability of both on- and off-transient $\dot{V}O_2$ kinetic parameters during treadmill running, using a single visit protocol. The reproducibility and minimal day-to-day variability of $\dot{V}O_2$ kinetics parameters from a single visit protocol means that $\dot{V}O_2$ kinetics can be measured in a short period of time. This has advantages over other protocols that usually require multiple visits to the laboratory which are inappropriate for competitive athletes. Importantly, it identified that both on- and off-transient $\dot{V}O_2$ kinetic parameters could confidently be used to characterise and compare athletes.

This is the first study to characterise and differentiate the on and off-transient $\dot{V}O_2$ kinetics, in the moderate-domain, in MD and LD runners. This contributes to the limited database of values for treadmill based measures of on- and off-transient $\dot{V}O_2$ kinetics which could then be used to compare future studies involving trained subjects and/or intervention strategies. Specifically, it has been shown that $\dot{V}O_2$ kinetics are faster in LD runners than in MD runners. Primarily, this was attributable to a greater volume of training performed by LD runners and the relationship between training volume and τ_{on}. The greater volume of training performed by LD runners is likely to have increased mitochondrial density and oxidative enzyme activity. However, it should be acknowledged that information about training volume in MD and LD runners was obtained from self-reported diaries and that the effect(s) of different types of training (volume and intensity) on $\dot{V}O_2$ kinetics, and other physiological and biochemical measures, was not measured in this study. Further research to investigate the effect(s) of training with precisely controlled volume and intensity of training is required to consolidate the findings of the present study. In addition, it is also acknowledged that assessments of $\dot{V}O_2$ kinetics in the moderate-domain are not truly reflective of intensities experienced during competitive MD and LD running events. However, the use of moderate-domain $\dot{V}O_2$ kinetics to reflect muscle oxidative potential is still worthy of consideration regardless of the intensity of MD and LD events. The measurement of $\dot{V}O_2$ kinetics in the moderate-domain allows a partitioning of the peripheral adaptation(s), relating to O_2 utilisation in muscle, from central adaptations relating to O_2 transport and delivery. However, at present there is uncertainty about the invariance of τ above and below V_T. Clearly, further research above and below V_T would help establish the usefulness of $\dot{V}O_2$ kinetics in the moderate- and heavy-intensity domain to further assess endurance-trained runners.

This thesis clearly revealed relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function ($\dot{V}O_{2\,max}$, V_T and RE). That these relationships were only observed in LD runners is also a novel and valuable finding. Based on previous literature that highlighted underpinning mechanisms of these measures, the inconsistent relationships,

especially with respect to the independency of $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics in MD runners, suggests that factors determining these two measures differ. This also provides support for further investigation into the effect(s) of different training regimes on $\dot{V}O_2$ kinetics and whether adaptation(s) are concomitant with changes in other measures of aerobic function.

Physiological and biochemical adaptations to training would probably influence $\dot{V}O_2$ kinetics which might have functional implications for a runner. Primarily, this thesis explored whether $\dot{V}O_2$ kinetics could be considered a potential determinant of 5 km running performance, since this had not been previously considered. Prior to analysis, after appropriately categorising runners as high and low performers, it was clear that high performers had much faster $\dot{V}O_2$ kinetics than low performers. The bi-variate correlations between $\dot{V}O_2$ kinetic parameters and 5 km running performance revealed no relationships for high or low performers. However, when groups were combined, a moderate to high relationship was observed between τ_{on}, MRT_{on}, τ_{off} and MRT_{off} and 5 km running performance. It is probable that the increased size and greater heterogeneity of the sample when runners were combined influenced the relationship which collectively resulted in a increased *r*.

When other measures of aerobic function were considered in addition to $\dot{V}O_2$ kinetics, in a multiple regression model, it was revealed that $\dot{V}O_2$ kinetics did not contribute to the regression models when high and low performers were considered separately. In these models, other measures of aerobic function (predominantly $\dot{V}O_{2\,max}$ and RE) explained most of the variation in running performance. For these groups of runner, it is probable that measures of $\dot{V}O_2$ kinetics offered no 'additional' information to the models that was not already accounted for by $\dot{V}O_{2\,max}$. However, MRT_{off} did offer some additional information to the model when combined high and low performers were considered. This suggests some potential use of $\dot{V}O_2$ kinetics, in the moderate-domain, to determine running performance but perhaps only in a cross-sectional assessment of heterogeneous runners. As such, the usefulness of $\dot{V}O_2$ kinetics to

determine 5 km running performance in cross-sectional studies, compared to $\dot{V}O_{2\,max}$, is questionable. Instead, the application of $\dot{V}O_2$ kinetic measures for longitudinal assessments of runners might be more useful and is worthy of consideration for future research.

Overall, these findings demonstrate the potential usefulness of $\dot{V}O_2$ kinetics as a measure to reflect adaptation(s) in muscle in MD and LD runners. However, specific intervention studies are required to elucidate the metabolic consequences of different training regimes in MD and LD runners and whether these are reflected in 1) measures of on- and off-transient $\dot{V}O_2$ kinetics; 2) the relationship between $\dot{V}O_2$ kinetics and other measures of aerobic function and 3) running performance.

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Appendix 1 Conference communications

1.1 ECSS 2001 Abstract

TEST-RETEST REPRODUCIBILITY OF MEASURES OF OXYGEN UPTAKE KINETICS

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Oxygen uptake ($\dot{V}O_2$) kinetics during moderate intensity exercise are reflective of an individual's aerobic fitness. However, inherent breath-by-breath variability in $\dot{V}O_2$ means that several transitions are required to minimise the effect of noise on estimates of kinetic parameters. This results in a prolonged test that might detract from physiological assessments of athletes. As few as 3 transitions have been used in the assessment of $\dot{V}O_2$ kinetics in competitive cyclists (Norris & Peterson 1998). Since the reproducibility of physiological measurements should be established before they are applied to sports performance, the purpose of this study was to establish the test-retest reproducibility of $\dot{V}O_2$ kinetics using 3 repeat step transitions.

Nine men (mean ± SD: age 20.5 ± 2.1 years; body mass 71.0 ± 7.3 kg; stature 178.8 ± 8.5 cm) provided written informed consent and completed an incremental ramp cycle test to establish maximal oxygen uptake ($\dot{V}O_{2\,max}$) and ventilatory threshold (VT). Mean (± SD) $\dot{V}O_{2\,max}$ and VT were 3538 ± 403 $ml \cdot min^{-1}$ and 1968 ± 230 $ml \cdot min^{-1}$ respectively. Participants then completed two step tests 7 days apart. Each test consisted of 3 consecutive transitions of cycling at 30 W for 6 min, followed by 6 min at a moderate intensity (80% VT). Breath-by-breath $\dot{V}O_2$ data were interpolated at 1 s intervals, time aligned and ensemble averaged to produce a single data set. Time delay (δ), time constant (τ) and mean response time (MRT) were identified from a mono-exponential model of the $\dot{V}O_2$ - time relationship. Paired t-tests compared means of repeat tests. The 95% limits of agreement (LOA), measurement error and systematic bias for δ, τ and MRT were calculated to assess reproducibility. Statistical significance was set at $P < 0.05$.

Kinetic parameters did not differ between tests, but 95% LOA indicated test-retest variability (Table 1) with MRT the least variable kinetic parameter.

Table 1. Limits of agreement of kinetic parameters from tests 1 and 2 (n = 9)

	δ	τ	MRT
Mean of test 1 and test 2 (s)	11.5 ± 4.3	22.3 ± 4.9	33.8 ± 4.4
Mean difference (s)	1.0 ± 5.4	-0.3 ± 6.7	0.7 ± 3.8
95% LOA (s)	-9.5 to 11.5	-13.4 to 12.8	-6.7 to 8.1
Measurement error (%)	92	59	22
Systematic bias (%)	10	-1	2

(MRT = $\delta + \tau$)

These findings show that a test session of only 3 transitions does not provide reproducible measures of $\dot{V}O_2$ kinetics. The test-retest reproducibility is determined by a combination of the effect of biological variability and noise on kinetic parameter estimation. The finding of higher variability in δ and τ than in MRT could be attributable to the effect of noise on the model fitting procedure since for any individual, a test-retest difference in δ tended to be offset by a change in τ. Methods proposed by Lamarra et al. (1987) could be used to quantify the effect of noise and thereby determine the underlying biological variability. If this variability is large, the test would be unsuitable for the assessment of athletes.

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RUNNING ECONOMY IN MIDDLE- AND LONG-DISTANCE RUNNERS

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In the assessment of running economy (RE), oxygen uptake ($\dot{V}O_2$) for a given running speed (e.g. 16 km·h^{-1}) relative to body mass (BM) is commonly expressed as a ratio standard, i.e. ml·kg^{-1}·min^{-1}. However, according to principles of allometry, $\dot{V}O_2$ does not increase linearly with BM (Schmidt-Nielson, 1984). Using these principles, Svedenhag and Sjödin (1994) were able to differentiate between elite middle-distance (MD) and long-distance (LD) runners when $\dot{V}O_2$ was expressed in ml·kg$^{-0.75}$·min^{-1}. There are few studies that have compared the outcomes of different scaling techniques in runners. The purpose of this study was to make such comparisons on apparent RE in MD and LD runners.
With institutional ethics approval, ten male MD (M ± SD: age 20.8 ± 2.7 years; stature 180.5 ± 8.3 cm; BM 67.2 ± 7.3 kg) and ten LD (M ± SD: age 24.1 ± 5.6 years; stature 179.7 ± 7.8 cm; 70.0 ± 8.7 kg) runners provided written informed consent and participated. All runners were accustomed to treadmill running. Participants completed 6-8 four min bouts of exercise with running speed increases of 1 km·h^{-1} for each bout. After a 15 min recovery period, an incremental test to volitional exhaustion was performed to determine maximal oxygen uptake ($\dot{V}O_{2\,max}$). During all tests, pulmonary gas-exchange was measured breath-by-breath using a mass spectrometer (MGA 1100, Marquette Electronics, MW, USA) that was calibrated before and verified after each test. The $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ at 16 km·h^{-1} were expressed as ratio standards and power function ratios, with exponents for BM of 0.67 and 0.75. Groups were compared using independent *t*-tests. Significance was set at $P < 0.05$.
The results are illustrated in Table 1.

Table 1. The $\dot{V}O_{2\,max}$ and RE at 16 km·h^{-1} in MD (n=10) and LD (n=10) runners (Values are M ± SD).

	$\dot{V}O_{2\,max}$ ml·kg^{-1}·min^{-1}	$\dot{V}O_{2\,max}$ ml·kg$^{-0.67}$·min^{-1}	$\dot{V}O_{2\,max}$ ml·kg$^{-0.75}$·min^{-1}	RE ml·kg^{-1}·min^{-1}	RE ml·kg$^{-0.67}$·min^{-1}	RE ml·kg$^{-0.75}$·min^{-1}
MD	62.0 ± 4.0	248 ± 17	177 ± 12	53.7 ± 3.1	154 ± 10	215 ± 14
LD	60.1 ± 7.1	244 ± 31	174 ± 22	51.5 ± 3.1	149 ± 8	209 ± 12
P value	0.463	0.698	0.635	0.135	0.314	0.245

Irrespective of the way in which $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ at 16 km·h^{-1} were scaled for differences in BM, no differences were identified between MD and LD runners ($P > 0.05$, Table 1). These findings are in contrast to those of Svedenhag and Sjödin (1994) although in their study runners were elite. The standard of runner might influence the ability to differentiate between groups. The lack of difference between groups observed here could be attributable to similarities in training regimes. The results suggest that non-elite MD and LD runners do not differ in RE at 16 km·h^{-1} regardless of how $\dot{V}O_2$ is expressed.

Schmidt-Nielson, K. (1984). Scaling: Why is animal size so important? Cambridge University Press, Cambridge.

Svedenhag, J., and Sjödin, B. (1994). Body-mass-modified running economy and step length in elite male middle- and long-distance runners. International Journal of Sports Medicine, 15, 305-310.

PULMONARY OXYGEN UPTAKE KINETICS IN MIDDLE- AND LONG-DISTANCE RUNNERS

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Pulmonary oxygen uptake ($\dot{V}O_2$) kinetics at the onset of moderate-intensity exercise are sensitive to training stimuli and are faster in the trained individual (Hagberg et al 1980). Accordingly, the measurement of $\dot{V}O_2$ kinetics in the physiological assessment of athletes seems justified. Assessments of $\dot{V}O_2$ kinetics have tended to use cycle ergometry, which might not be appropriate for the assessment of middle-distance (MD) and long-distance (LD) runners. Therefore, the purpose of this study was to compare the $\dot{V}O_2$ kinetics of MD and LD runners obtained during moderate-intensity treadmill running.

With institutional ethics approval, 10 male MD (mean ± SD: age: 22.0 ± 6.8 years; stature: 176.6 ± 5.8 cm; BM: 65.3 ± 5.0 kg) and 10 LD (mean ± SD: age: 25.8 ± 5.0 years; stature: 180.0 ± 8.1 cm; BM: 71.4 ± 9.8 kg) runners participated. Each completed an incremental test to volitional exhaustion to determine ventilatory threshold (V_T, MD: 49.9 ± 4.8; LD: 48.4 ± 4.4 $ml.kg^{-1}.min^{-1}$) and maximal oxygen uptake (MD: 60.0 ± 4.9; LD: 59.0 ± 6.3 $ml.kg^{-1}.min^{-1}$). On a separate occasion participants completed six square-wave transitions from walking at 4 $km.h^{-1}$ for 6 min to running for 6 min at a moderate intensity (80% V_T), before returning to walking at 4 $km.h^{-1}$ for a further 6 min. Pulmonary gas-exchange was measured breath-by-breath using a mass spectrometer (MGA 1100, Marquette Electronics, MW, USA) that was calibrated before and verified after each test. Breath-by-breath $\dot{V}O_2$ data were interpolated at 1 s intervals, time aligned and ensemble averaged to produce a single data set. The kinetic parameters of amplitude (A), time delay (δ), time constant (τ) and mean response time (MRT) were identified from a mono exponential model of the $\dot{V}O_2$ - time relationship during both on- and off-transients. Independent t-tests compared means between MD and LD runners and paired t-tests compared means between on- and off-transients. Statistical significance was set at $P < 0.05$.

Table 1. On-transient kinetic parameter estimations for MD (n=10) and LD (n=10) runners.

	A ($ml.min^{-1}$)	δ (s)	τ (s)	MRT (s)
MD	1739 ± 170	14.4 ± 1.3#	14.2 ± 3.1#	28.6 ± 2.5#
LD	1855 ± 257	14.6 ± 1.5#	12.5 ± 2.3#	27.1 ± 2.2#

Table 2. Off-transient kinetic parameter estimations for MD (n=10) and LD (n=10) runners.

	A ($ml.min^{-1}$)	δ (s)	τ (s)	MRT (s)
MD	1653 ± 329	8.9 ± 2.3	27.1 ± 3.0	36.0 ± 3.1
LD	1857 ± 261	8.3 ± 3.3	24.1 ± 2.3*	32.4 ± 2.4*

*Significantly lower in LD runners, $P < 0.05$; #Significantly different from off-transient, $P < 0.01$.

The results are illustrated in Tables 1 and 2. Kinetic parameters during the on-transient did not differ between MD and LD runners ($P > 0.05$). During the off-transient, LD runners had a shorter τ and MRT ($P < 0.05$, Table 2). On- and off-transient responses were different in MD and LD runners (Tables 1 and 2).

The findings show that recovery from a bout of moderate-intensity exercise is quicker in LD runners. This could be due to several physiological adaptations that can be influenced by training. In MD and LD runners, there was asymmetry between on- and off-transient $\dot{V}O_2$ responses, with the off-transient being considerably longer. This is in contrast to the observed symmetry between transients for cycling (Paterson and Whipp 1991). The results suggest that mode of exercise influences the symmetry between on- and off-transients for moderate-intensity exercise.

REFERENCES

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Paterson and Whipp (1991). *J Physiol* 443: 575-586

Relationships between oxygen uptake kinetics and other measures of aerobic function in MD and LD runners.

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Introduction

Endurance training results in several physiological adaptations at a cellular level such as an increased number of mitochondria and increased oxidative enzyme activity (Saltin and Gollnick, 1983). Collectively, these are likely to speed oxygen uptake ($\dot{V}O_2$) kinetics at the onset of moderate-intensity exercise. However, the influence of endurance training on other measures of aerobic function, such as maximal $\dot{V}O_2$ ($\dot{V}O_{2\,max}$), ventilatory threshold (V_T) and O_2 cost of running (C_r), might be less pronounced which could dissociate these measures. The temporal dissociation between changes in mitochondrial enzyme activity and $\dot{V}O_{2\,max}$ after endurance training (Henriksson and Reitman, 1977) suggests that there might be poor correlation between measures of peripheral (i.e. $\dot{V}O_2$ kinetics) and central (i.e. $\dot{V}O_{2\,max}$) physiological status, as found by Bell et al (1999). The characteristics of endurance training (intensity and volume) might influence relationships between measures of aerobic function. Since middle-distance (MD) and long-distance (LD) runners differ in terms of the intensity and volume of their training, the purpose of this study was to explore the relationships between $\dot{V}O_2$ kinetics and other measures of aerobic function.

Methods

With ethics approval, 16 male MD (mean ± SD: age: 21.3 ± 5.5 years; stature: 176.8 ± 6.8 cm; BM: 66.6 ± 5.8 kg) and 16 male LD (mean ± SD: age: 25.0 ± 4.2 years; stature: 180.3 ± 7.0 cm; BM: 69.9 ± 8.4 kg) runners participated. Each completed an incremental test to volitional exhaustion to determine V_T (MD: 49.2 ± 4.6; LD: 49.4 ± 4.4 $ml.kg^{-1}.min^{-1}$) and $\dot{V}O_{2\,max}$ (MD: 59.8 ± 4.7; LD: 60.2 ± 5.8 $ml.kg^{-1}.min^{-1}$). On two separate occasions, participants completed 1) a series of 4 min bouts of sub-maximal running to determine C_r ($ml.kg^{-1}.km^{-1}$) and 2) six square-wave transitions to and from moderate-intensity exercise (80% V_T) to establish on- and off-transient $\dot{V}O_2$ kinetics (τ_{on} and τ_{off} respectively). Relationships between measures were explored using Pearson's product moment correlation coefficient. Significance was set at $P < 0.05$.

Results

The $\dot{V}O_{2\,max}$, V_T and C_r did not differ between MD and LD runners ($P > 0.05$). However, both τ_{on} (MD: 16.4 ± 4.1 s; LD: 12.3 ± 2.2 s; $P < 0.01$) and τ_{off} (MD: 26.9 ± 3.2 s; LD: 24.5 ± 2.5 s; $P < 0.05$) were significantly shorter in LD runners. The correlation coefficients between $\dot{V}O_2$ kinetics and other measures in MD and LD runners are presented in Table 1. Significant relationships were observed between τ_{on} and $\dot{V}O_{2\,max}$ and τ_{on} and V_T in LD runners. The C_r was not related to either τ_{on} or τ_{off} in LD runners. There were no relationships between $\dot{V}O_2$ kinetics and any other measure in MD runners. The τ_{off} was not related to any measure in either MD or LD runners.

Table 1: *Correlation coefficients (r) between measures of aerobic function in MD and LD runners.*

	MD					LD				
Measure	τ_{on}	τ_{off}	$\dot{V}O_{2\,max}$	V_T	C_r	τ_{on}	τ_{off}	$\dot{V}O_{2\,max}$	V_T	C_r
τ_{on}	1.00	0.47	-0.24	-0.26	0.35	1.00	0.59*	-0.70**	-0.57*	-0.42
τ_{off}		1.00	-0.09	-0.02	0.15		1.00	-0.40	-0.07	-0.35

**P <0.01; *P <0.05.

Discussion/Conclusion

The results of this study show that on-transient $\dot{V}O_2$ kinetics (τ_{on}) and other measures of aerobic function ($\dot{V}O_{2\,max}$ and V_T) are inter-related (Table 1). The significant relationship between τ_{on} and $\dot{V}O_{2\,max}$ in LD runners supports previous findings (Powers et al 1985) and suggests that these measures are reflective of each other. However, the above is not apparent in MD runners, despite similarities in $\dot{V}O_{2\,max}$ and V_T. This could be attributed to the greater emphasis on anaerobic training in MD runners which might dissociate the adaptations in $\dot{V}O_2$ kinetics and $\dot{V}O_{2\,max}$. Future research should explore the effects of different intensities and volumes of training on $\dot{V}O_2$ kinetics and identify whether adaptations in $\dot{V}O_2$ kinetics are concomitant with adaptations in other aerobic measures.

References

Bell C et al (1999). Exp Physiol 84, 747-759.
Henriksson J, Reitman J (1977). Acta Physiol Scand 99, 91-97.
Saltin B, Gollnick P (1983). Am Physiol Soc:Handbook of Physiology 10, 555-631.
Powers S et al (1985). Eur J Appl Physiol 54, 306-308.

Appendix 2 Verification of treadmill speed and gradient

2.1 Treadmill speed

Prior to each exercise testing session, the treadmill speed was verified over a range of speeds and gradients that were pertinent to those used in each investigation. Such speeds ranged between 4 and 20 $km \cdot h^{-1}$ (1.1 to 5.6 $m \cdot s^{-1}$). Verification procedures were performed in two conditions. First with an 'unloaded' treadmill belt and second with a 'loaded' treadmill belt. In the loaded condition, an individual of approximately 70 kg BM ran on the treadmill at each of the speeds that was to be verified.

The length of the treadmill belt was measured to the nearest 0.01 m. The distance travelled by the treadmill belt in 20 full revolutions at each of the pre-defined speeds was calculated: -

Treadmill belt length = 5.58 m

Distance travelled = 5.58×20

= 111.6 m

A marker was placed on the treadmill belt to allow identification of one full revolution of the treadmill belt. Time taken for 20 revolutions of the belt at each of the five pre-defined speeds was recorded with a stopwatch (C200sport, Casio, UK) recording to 0.1 s. This procedure was repeated twice and the mean time taken for each speed was calculated to within 0.1 s. The actual speed of the treadmill belt for the five displayed speeds in both unloaded and loaded conditions was then calculated using the formula:

$$\text{Speed } (m \cdot s^{-1}) = \frac{\text{Distance (m)}}{\text{Time (s)}}$$

Table A1. Verification of treadmill speed: displayed speed vs. actual speed.

Displayed speed		**Time for 20 revolutions (s)**		**Actual Speed ($m \cdot s^{-1}$)**	
$km \cdot h^{-1}$	$m \cdot s^{-1}$	Unloaded	Loaded	Unloaded	Loaded
0	0	0	0	0	0
4	1.1	101.1	100.7	1.1	1.1
8	2.2	50.5	50.1	2.2	2.2
12	3.3	33.8	33.7	3.3	3.3
16	4.4	25.2	25.2	4.4	4.4
20	5.6	20.2	20.1	5.5	5.6

The relationship between calculated 'actual' speeds and 'indicated' speeds displayed on the treadmill control panel are illustrated for unloaded and loaded conditions in Figures A1 and A2 respectively. The linear modelling technique used was least squares (x on y) regression.

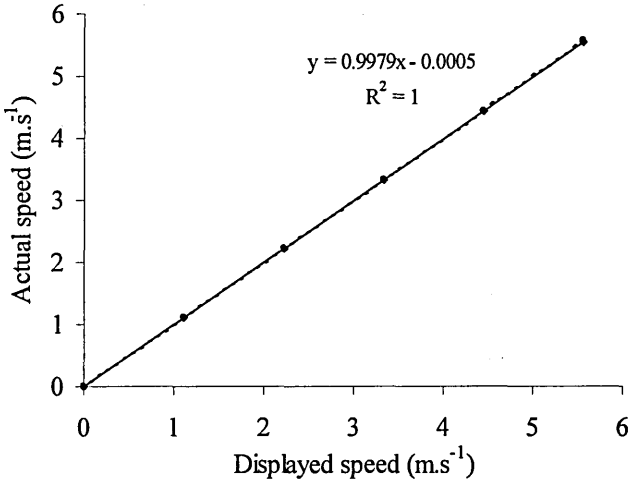


Figure A1 Verification of treadmill belt speed in the un-loaded condition. Dashed line represents line of identity.

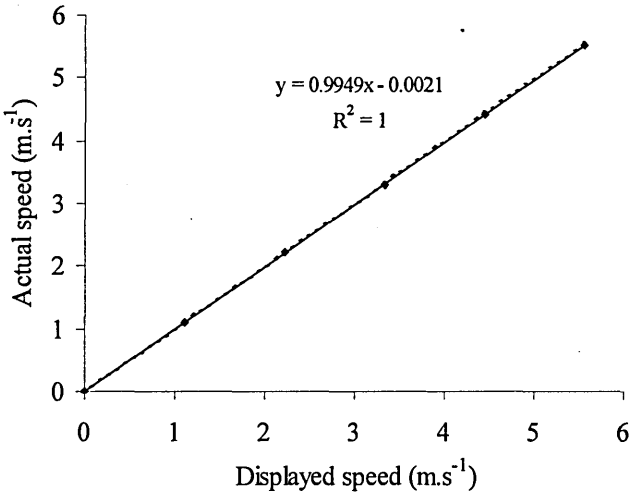


Figure A2 Verification of treadmill belt speed in the loaded (70 kg) condition. Dashed line represents line of identity.

2.2 Verification of Treadmill gradient

The gradient of the treadmill bed from 0% to 20% was verified. The method used expresses gradient as the sine of the angle, in which sine equals the vertical rise over the hypotenuse. Therefore: -

$$\text{Treadmill elevation (sine)} = \frac{rise}{hypotenuse}$$

For treadmills with both moveable rear and front axles, such as the one used throughout the investigations, the vertical rise was equal to the sum of the rise in the front axle and the drop of the rear axle. When divided by the axle-axle length, the grade is expressed as a fraction. The axle-to-axle length of the treadmill was 254.5 cm.

Table A2. Verification of treadmill gradient: displayed gradient vs. actual gradient.

Display	**Distance from ground (cm)**		**Height**	**Actual**	**Difference**
(%)	Front axle	Rear axle	**change (cm)**	**gradient (%)**	**(%)**
0.0	46.0	46.0	0.0	0.0	0.0
1.0	48.7	46.0	2.7	1.1	0.1
5.0	57.5	46.0	11.5	4.5	-0.5
10.0	68.8	45.7	22.8	9.0	-1.0
15.0	79.8	45.5	34.1	13.4	-1.6
20.0	90.5	45.2	45.0	17.7	-2.3

Linear regression equations were generated to determine the relationship between displayed gradient and actual gradient (Figure A3).

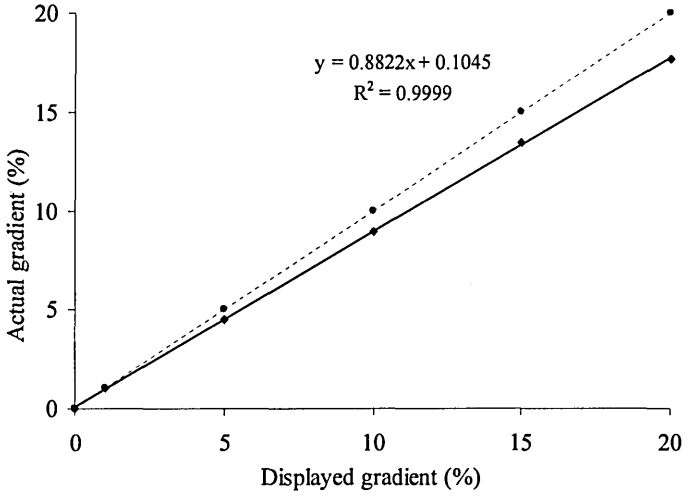


Figure A3 Verification of displayed treadmill gradient vs. actual treadmill gradient. Dashed line represents line of identity.

The calibration check for gradient over a range of elevations between 0 and 20% (displayed values) shows minimal differences between 0 and 5%. However, at elevations greater than 5% there is a tendency for the displayed gradient to under-read the actual gradient (range -0.5 to -2.3%). During the investigations undertaken as part of this thesis, gradients above 1% were not required and therefore no correction for differences between displayed and actual gradient was applied.

Appendix 3 Lactate analyser reproducibility

To assess the reproducibility of the lactate analyser, the CV was established. Five 25 µl samples of lactate standard (5 mmol·l^{-1}) were analysed. Between each injection of lactate standard, identical procedures (injection technique, timing and automatic wash-out) were followed to minimise possible sources of variability which might affect the reproducibility.

Table A3. Data from the calculation of the CV for [HLa] measures.

5 mmol·l^{-1} sample	**Reading**
1	5.01
2	5.10
3	5.02
4	4.98
5	4.96
Mean	5.06
SD	0.05
CV%	1.1

Appendix 4 Details of study and testing procedures for participants

Study Details and Informed Consent

Project Title: The assessment of $\dot{V}O_2$ kinetics and endurance running performance in
Investigator: Andrew Kilding

Purpose of study

The purpose of this study is to assess $\dot{V}O_2$ kinetics and endurance running performance in endurance trained runners. It will involve you completing 4 running tests during 3 visits to the laboratory. Each visit and the tests to be completed are explained below.

Description of exercise tests and procedures

Laboratory visit 1 - Assessment of Running Economy and $\dot{V}O_{2\ max}$
You will be required to run for a series (4-6) of 4 minute stages at gradually increasing speeds (e.g. 12, 13, 14 and 15 $km \cdot h^{-1}$). During each 4 minute run, your oxygen consumption and heart rate will be measured. At the end of each stage a small blood sample will be taken for lactate analysis. After 10 minutes rest you will be required to perform a maximal exercise test. You will start the test at a running speed of 10 $km \cdot h^{-1}$. The speed will be increased by 1 km/h every minute. You are required to run whilst the intensity gets progressively harder, until you can no longer maintain it. The duration of the test will be approximately 8 - 12 minutes, followed by a 5 minute cool down. You will feel discomfort towards the end of the test due to fatigue. These feelings will last for a few minutes and are similar to those experienced at the end of a hard training session or race. The risk of injury or cardiovascular complications during the test is very low. Throughout the test oxygen consumption and heart rate will be measured. After the test a small sample of blood will be taken for lactate analysis.

Laboratory visit 2 - Assessment of $\dot{V}O_2$ kinetics and 5Km time trial
Approximately 2-3 days after the maximal exercise test you will be required to complete a $\dot{V}O_2$ kinetics test. This involves walking at 4 km/h for 6 minutes followed by an increase to running, at a pre-determined faster speed, for a further 6 minutes. The intensity of this faster running speed is of moderate intensity and is prescribed in relation to your performance during the previous $\dot{V}O_{2\ max}$ test. This walk-run transition will be repeated 3 times, after which you will be given a 15 minute rest before the same is repeated again (total of 6 walk-run transitions). Oxygen consumption and heart rate will be measured and a small sample of blood will be taken for lactate analysis before and after the test.

Laboratory visit 3 - 5km performance trial
You are required to complete a 5km time trial. This involves running this distance in the quickest time possible on a treadmill. Prior to the test you will be advised to warm-up (jog, stretch etc) using your own pre-race routine. Throughout the test you will be able to increase or decrease the running speed as you require. During the time-trial, your heart rate will be monitored.

Appendix 5 Informed consent form

Sheffield Hallam University

School of Sport and Leisure Management

Research Ethics Committee

INFORMED CONSENT FORM	
TITLE OF PROJECT: The assessment of $\dot{V}O_2$ kinetics and endurance running performance in middle- and long-distance runners.	
The participant should complete the whole of this sheet himself/herself	
Have you read the Participant Information Sheet?	YES/NO
Have you had an opportunity to ask questions and discuss this study?	YES/NO
Have you received satisfactory answers to all of your questions?	YES/NO
Have you received enough information about the study?	YES/NO
Who have you spoken to? ..... Do you understand that you are free to withdraw from the study: • at any time • without having to give a reason for withdrawing • and without affecting your future medical care	YES/NO
Do you agree to take part in this study	YES/NO
Signed.....Date..... (NAME IN BLOCK LETTERS).....	
Signature of Parent / Guardian in the case of a minor	

Sheffield Hallam University

PRE-TEST MEDICAL QUESTIONNAIRE

Surname: _____ First name/s: _____
Date of Birth: _____ Gender : Male ☐ Female ☐

Please answer the following questions by ticking the appropriate box, or filling in the blank.

1. **How would you describe your present level of activity?**

   Sedentary ☐ Moderately active ☐ Active ☐ Highly active ☐

2. **How would you describe you present level of fitness?**

   Unfit ☐ Moderately fit ☐ Trained ☐ Highly trained ☐

3. **How would you consider your present body weight?**

   Underweight ☐ Ideal ☐ Slightly over ☐ Very overweight ☐

4. **Smoking Habits:**

Are you currently a smoker?	Yes ☐ No ☐
How many do you smoke	.....per day
Are you a previous smoker?	Yes ☐ No ☐
How long is it since you stopped?	.....years
Were you an occasional smoker?	Yes ☐ No ☐
	.....per day
Were you a regular smoker	Yes ☐ No ☐
	.....per day

5. **Do you drink alcohol?** Yes ☐ No ☐
   If you answered **Yes**, do you have?

   An occasional drink ☐ A drink every day ☐ More than one drink a day ☐

6. **Have you had to consult your doctor within the last six months?** Yes ☐ No ☐
   If you answered **Yes**, please give details.....
   .....

7. **Are you presently taking any form of medication?** Yes ☐ No ☐
   If you answered **Yes**, please give details.....
   .....

8. **As far as you are aware, do you suffer or have you ever suffered from:**

a Diabetes?	Yes □ No □	**b** Asthma?	Yes □ No □
c Epilepsy?	Yes □ No □	**d** Bronchitis?	Yes □ No □
e *Any form of heart complaint?	Yes □ No □	**f** Raynaud's Disease?	Yes □ No □
g *Marfan's Syndrome?	Yes □ No □	**h** *Aneurysm/embolism?	Yes □ No□
I Aneamia	Yes □ No □		

9. ***Is there a history of heart disease in your family?** Yes □ No □

10. ***Do you currently have any form of muscle or joint injury?** Yes □ No □
If you answered **Yes**, please give details.....
.....
.....

11. **Have you had to suspend you normal training in the last two weeks?**
Yes □ No □
If the answer is **Yes** please give details.....
.....
.....

12. **Please read the following questions:**

a)	Are you suffering from any known serious infection?	Yes □ No □
b)	Have you had jaundice within the previous year?	Yes □ No □
c)	Have you ever had any form of hepatitis?	Yes □ No □
d)	Are you HIV antibody positive	Yes □ No □
e)	Have you had unprotected sexual intercourse with any person from an HIV high-risk population?	Yes □ No □
f)	Have you ever been involved in intravenous drug use?	Yes □ No □
g)	Are you haemophiliac?	Yes □ No □

13. **As far as you are aware, is there anything that might prevent you from successfully completing the tests that have been outlined to you?**
Yes □ No □

If the answer to any of the above is yes then:

Discuss with the Centre for Sport and Exercise Science the nature of the problem. Questions indicated by (*) Allow your Doctor to fill out the 'Doctors Consent Form' provided.

Signed: _____ Date:_____

Signature of guardian / parent (if under 18) _____

Signature of tester: _____ Date:_____

Appendix 7 Chapter 4: Raw data and statistical analyses

7.1 Raw data: On-transient

Test 1: On-transient

Participant	S_o	S_o/A%	Rest VO_2	$VO_{2(b)}$	$VO_{2(m)}$	A_{on}	TD_{on}	Tau_{on}	MRT_{on}	O_2 def	O_2 stores	CI (s)	*n* reps
1	0.088	5.7	414	842	2402	1560	15.1	11.6	26.7	696	393	0.83	2
2	0.107	5.1	451	1036	3137	2100	15.6	9.5	25.1	879	545	0.72	1
3	0.112	7.8	325	740	2173	1433	13.1	13.7	26.8	640	313	1.19	3
4	0.112	6.5	419	938	2668	1730	14.8	12.5	27.3	787	426	0.97	2
5	0.093	5.1	367	824	2664	1839	15.1	13.1	28.3	866	464	0.76	1
6	0.163	9.1	448	955	2744	1789	15.0	13.5	28.5	849	446	1.38	3
7	0.082	4.3	421	1025	2930	1905	14.2	16.4	30.6	972	450	0.68	1
8	0.132	5.9	439	1086	3320	2234	17.4	11.1	28.6	1063	650	0.86	2
9	0.082	4.7	348	875	2604	1729	11.9	12.6	24.4	704	342	0.71	1
10	0.099	6.0	379	887	2550	1663	14.7	9.8	24.5	680	408	0.85	2
11	0.141	6.9	431	969	3024	2055	14.1	13.2	27.3	935	483	1.04	2
12	0.142	7.8	360	920	2749	1830	15.0	11.5	26.5	808	456	1.14	2
Mean	**0.113**	**6.2**	**400**	**925**	**2747**	**1822**	**14.7**	**12.4**	**27.0**	**823**	**448**	**0.9**	**2**
SD	**0.026**	**1.4**	**42**	**98**	**318**	**228**	**1.3**	**1.9**	**1.8**	**129**	**89**	**0.2**	**1**

Test 2: On-transient

Participant	S_o	S_o/A%	Rest VO_2	$VO_{2(b)}$	$VO_{2(m)}$	A_{on}	TD_{on}	Tau_{on}	MRT_{on}	O_2 def	O_2 stores	CI (s)	*n* reps
1	0.09	5.3	436	860	2511	1651	16.2	10.9	27.1	746	447	0.77	1
2	0.145	7.2	518	1022	3038	2016	14.6	10.0	24.6	825	489	1.03	2
3	0.076	4.9	402	797	2345	1548	11.8	14.7	26.5	684	304	0.76	1
4	0.124	7.3	434	895	2598	1703	15.9	12.4	28.3	804	452	1.08	2
5	0.158	8.8	351	823	2618	1795	14.1	14.4	28.6	855	423	1.36	3
6	0.178	9.8	478	975	2793	1818	14.7	12.7	27.4	829	444	1.47	4
7	0.078	3.9	498	1047	3073	2026	13.4	16.8	30.2	1020	451	0.62	1
8	0.096	4.8	455	1059	3075	2016	17.3	11.9	29.2	981	581	0.70	1
9	0.126	7.8	411	876	2481	1606	12.8	11.4	24.3	650	344	1.15	2
10	0.089	5.5	412	885	2493	1609	15.5	8.9	24.3	652	414	0.78	1
11	0.169	8.3	497	943	2979	2036	13.5	13.3	26.8	911	458	1.26	3
12	0.125	7.4	413	959	2660	1700	16.1	9.8	25.9	735	457	1.05	2
Mean	**0.121**	**6.7**	**442**	**928**	**2722**	**1794**	**14.7**	**12.3**	**26.9**	**808**	**439**	**1.0**	**2**
SD	**0.036**	**1.8**	**49**	**86**	**260**	**186**	**1.6**	**2.3**	**1.9**	**121**	**69**	**0.3**	**1**

where O_2 def (ml.min) = Amp*mrt/60; O_2 stores (ml.min) = Amp*(MRT-tau)/60

7.2 Raw data: Off-transient

Test 1: Off-transient

Participant	$VO2_{(m)}$	$VO2_{(b)}$	A_{off}	TD_{off}	Tau_{off}	MRT_{off}	O_2 debt
1	2387	841	1547	11.1	20.1	31.1	803
2	3107	1049	2059	9.1	24.1	33.3	1141
3	2181	744	1436	8.7	25.7	34.4	824
4	2662	950	1713	10.8	24.2	35.0	999
5	2662	834	1828	1.7	26.5	28.2	859
6	2751	919	1832	10.9	23.6	34.5	1053
7	2948	1038	1910	9.4	26.4	35.8	1140
8	3316	1102	2214	12.6	20.1	32.7	1207
9	2594	876	1717	8.7	25.1	33.8	967
10	2530	869	1661	5.2	26.0	31.2	864
11	3011	961	2050	7.9	27.2	35.1	1200
12	2753	932	1820	10.6	24.8	35.4	1075
Mean	**2742**	**926**	**1816**	**8.9**	**24.5**	**33.4**	**1011**
SD	**296**	**99**	**205**	**2.9**	**1.9**	**2.1**	**132**

Test 2: Off-transient

Participant	$VO2_{(m)}$	$VO2_{(b)}$	A_{off}	TD_{off}	Tau_{off}	MRT_{off}	O_2 debt
1	2516	876	1640	10.4	18.2	28.7	784
2	3045	1031	2015	10.3	22.5	32.8	1100
3	2357	794	1563	6.2	25.8	32.0	833
4	2590	901	1689	10.4	24.6	35.0	985
5	2608	817	1790	4.8	24.9	29.8	888
6	2806	961	1845	9.9	23.4	33.3	1024
7	3075	1063	2012	9.9	27.6	37.4	1256
8	3081	1074	2007	13.5	22.2	35.7	1194
9	2450	880	1570	9.6	24.2	33.8	885
10	2500	836	1664	5.8	25.9	31.7	879
11	2961	934	2026	9.9	25.9	35.8	1210
12	2652	949	1704	9.6	24.7	34.3	974
Mean	**2720**	**926**	**1794**	**9.2**	**24.1**	**33.4**	**1001**
SD	**262**	**93**	**181**	**2.4**	**2.4**	**2.6**	**158**

where O_2 debt (ml.min) = Amp*mrt/60

7.3 Tests of normality: On-transient

Tests of normality for all physiological (and anthropometric) measures during the on-transient response for tests 1 and 2.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
AGE	.177	12	.200*	.938	12	.467
STATURE	.131	12	.200*	.974	12	.911
B.MASS	.195	12	.200*	.870	12	.072
ABSVO2MX	.181	12	.200*	.843	12	.034
RELVO2MX	.140	12	.200*	.940	12	.483
VO2SCALE	.288	12	.007	.818	12	.016
ABSTVENT	.248	12	.041	.937	12	.461
RELTVENT	.256	12	.029	.891	12	.156
VO2BASE1	.095	12	.200*	.990	12	.990*
VO2MOD1	.164	12	.200*	.983	12	.974
AMP1	.137	12	.200*	.982	12	.967
TD1	.197	12	.200*	.918	12	.332
TAU1	.160	12	.200*	.949	12	.583
MRT1	.126	12	.200*	.954	12	.649
O2DEF1	.155	12	.200*	.967	12	.818
O2STORE1	.180	12	.200*	.937	12	.461
HRBASE1	.180	12	.200*	.909	12	.276
HRMOD1	.151	12	.200*	.938	12	.464
GAIN1	.150	12	.200*	.955	12	.665
VO2BASE2	.151	12	.200*	.959	12	.713
VO2MOD2	.178	12	.200*	.902	12	.223
AMP2	.217	12	.122	.875	12	.083
TD2	.120	12	.200*	.981	12	.964
TAU2	.094	12	.200*	.978	12	.945
MRT2	.140	12	.200*	.955	12	.655
O2DEF2	.110	12	.200*	.953	12	.629
O2STORE2	.225	12	.095	.897	12	.195
HRBASE2	.200	12	.198	.950	12	.600
HRMOD2	.170	12	.200*	.932	12	.428
GAIN2	.183	12	.200*	.949	12	.573

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

7.4 Tests of normality: Off-transient

The following output illustrates the tests of normality for all physiological variables considered during the off-transient response for tests 1 and 2.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
VO2MOFF1	.153	12	.200*	.987	12	.990*
VO2BOFF1	.119	12	.200*	.979	12	.949
AMP1OFF	.137	12	.200*	.981	12	.964
TD1OFF	.228	12	.086	.866	12	.066
TAU1OFF	.187	12	.200*	.868	12	.070
MRT1OFF	.180	12	.200*	.889	12	.139
O2DEB1OF	.174	12	.200*	.922	12	.363
VO2MOFF2	.185	12	.200*	.903	12	.230
VO2BOFF2	.119	12	.200*	.950	12	.597
AMP2OFF	.214	12	.136	.874	12	.080
TDOFF2	.314	12	.002	.856	12	.047
TAUOFF2	.178	12	.200*	.904	12	.237
MRTOFF2	.092	12	.200*	.980	12	.958
O2DEB2OF	.180	12	.200*	.934	12	.442

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

7.5 Normal distribution of the differences

Tests of normality of the absolute differences between tests 1 and 2 for kinetic parameters during the on- and off-transients.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
ABDIFTAU	.122	12	.200*	.965	12	.798
ABDIFAMP	.154	12	.200*	.914	12	.308
ABDIFMRT	.185	12	.200*	.887	12	.124
ABDIFTD	.219	12	.118	.912	12	.293
OFFAMDIF	.204	12	.179	.922	12	.361
OFFTDDIF	.290	12	.006	.813	12	.013
OFTAUDIF	.195	12	.200*	.888	12	.132
OFMRTDIF	.137	12	.200*	.940	12	.480

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

7.6 Checks for homoscedasticity: On-transient

Correlation's between the absolute differences between tests 1 and 2 and the combined means of tests 1 and 2 for variables related to the on transient.

Correlations

		ABDIFTAU	TAU1AND2	ABDIFTD	TD1AND2	ABDIFAMP	AMP1AND2	ABDIFMRT	MRT1AND2
ABDIFTAU	Pearson Correlation	1.000	-.220	.206	-.019	.339	-.395	-.341	-.257
	Sig. (2-tailed)	.	.491	.521	.953	.281	.203	.279	.420
	N	12	12	12	12	12	12	12	12
TAU1AND2	Pearson Correlation	-.220	1.000	-.010	-.474	-.057	.056	.014	.743**
	Sig. (2-tailed)	.491	.	.975	.119	.860	.862	.965	.006
	N	12	12	12	12	12	12	12	12
ABDIFTD	Pearson Correlation	.206	-.010	1.000	-.415	-.164	-.611*	-.331	-.325
	Sig. (2-tailed)	.521	.975	.	.180	.611	.035	.293	.303
	N	12	12	12	12	12	12	12	12
TD1AND2	Pearson Correlation	-.019	-.474	-.415	1.000	.246	.404	.439	.237
	Sig. (2-tailed)	.953	.119	.180	.	.441	.193	.153	.459
	N	12	12	12	12	12	12	12	12
ABDIFAMP	Pearson Correlation	.339	-.057	-.164	.246	1.000	.193	-.259	.124
	Sig. (2-tailed)	.281	.860	.611	.441	.	.547	.416	.701
	N	12	12	12	12	12	12	12	12
AMP1AND2	Pearson Correlation	-.395	.056	-.611*	.404	.193	1.000	.239	.368
	Sig. (2-tailed)	.203	.862	.035	.193	.547	.	.454	.239
	N	12	12	12	12	12	12	12	12
ABDIFMRT	Pearson Correlation	-.341	.014	-.331	.439	-.259	.239	1.000	.350
	Sig. (2-tailed)	.279	.965	.293	.153	.416	.454	.	.265
	N	12	12	12	12	12	12	12	12
MRT1AND2	Pearson Correlation	-.257	.743**	-.325	.237	.124	.368	.350	1.000
	Sig. (2-tailed)	.420	.006	.303	.459	.701	.239	.265	.
	N	12	12	12	12	12	12	12	12

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

7.7 Checks for homoscedasticity: Off-transient

Correlation's between the absolute differences between tests 1 and 2 and the combined means of tests 1 and 2 for variables related to the off -transient.

Correlations

		OFFAMDIF	OFFAM1_2	OFFTDDIF	OFFTD1_2	OFTAUDIF	OFTAU1_2	OFMRTDIF	OFMRT1_2
OFFAMDIF	Pearson Correlation	1.000	.041	-.078	.462	.279	-.327	.565	.198
	Sig. (2-tailed)	.	.898	.809	.130	.380	.299	.055	.537
	N	12	12	12	12	12	12	12	12
OFFAM1_2	Pearson Correlation	.041	1.000	-.032	.331	.565	.014	.053	.394
	Sig. (2-tailed)	.898	.	.922	.293	.056	.966	.871	.206
	N	12	12	12	12	12	12	12	12
OFFTDDIF	Pearson Correlation	-.078	-.032	1.000	-.630*	.109	.339	.237	-.380
	Sig. (2-tailed)	.809	.922	.	.028	.737	.282	.458	.223
	N	12	12	12	12	12	12	12	12
OFFTD1_2	Pearson Correlation	.462	.331	-.630*	1.000	.196	-.577*	.193	.562
	Sig. (2-tailed)	.130	.293	.028	.	.542	.050	.548	.057
	N	12	12	12	12	12	12	12	12
OFTAUDIF	Pearson Correlation	.279	.565	.109	.196	1.000	-.465	.378	-.246
	Sig. (2-tailed)	.380	.056	.737	.542	.	.128	.225	.441
	N	12	12	12	12	12	12	12	12
OFTAU1_2	Pearson Correlation	-.327	.014	.339	-.577*	-.465	1.000	-.431	.351
	Sig. (2-tailed)	.299	.966	.282	.050	.128	.	.162	.263
	N	12	12	12	12	12	12	12	12
OFMRTDIF	Pearson Correlation	.565	.053	.237	.193	.378	-.431	1.000	-.215
	Sig. (2-tailed)	.055	.871	.458	.548	.225	.162	.	.502
	N	12	12	12	12	12	12	12	12
OFMRT1_2	Pearson Correlation	.198	.394	-.380	.562	-.246	.351	-.215	1.000
	Sig. (2-tailed)	.537	.206	.223	.057	.441	.263	.502	.
	N	12	12	12	12	12	12	12	12

*. Correlation is significant at the 0.05 level (2-tailed).

7.8 Paired *t*-tests: On-transient

Paired *t*-tests for a statistical comparisons of means between tests 1 and 2 for all physiological variables and $\dot{V}O_2$ kinetic parameters during the on-transient.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	VO2BASE1	924.83	12	98.44	28.42
	VO2BASE2	928.44	12	86.32	24.92
Pair 2	VO2MOD1	2747.16	12	317.87	91.76
	VO2MOD2	2722.01	12	260.41	75.17
Pair 3	AMP1	1822.33	12	228.04	65.83
	AMP2	1793.57	12	185.81	53.64
Pair 4	TD1	14.658	12	1.349	.390
	TD2	14.658	12	1.615	.466
Pair 5	TAU1	12.391	12	1.865	.538
	TAU2	12.278	12	2.306	.666
Pair 6	MRT1	27.049	12	1.805	.521
	MRT2	26.937	12	1.937	.559
Pair 7	O2DEF1	823.18	12	129.26	37.31
	O2DEF2	807.62	12	121.39	35.04
Pair 8	O2STORE1	447.90	12	88.51	25.55
	O2STORE2	438.72	12	68.69	19.83
Pair 9	HRBASE1	73.43	12	8.83	2.55
	HRBASE2	73.18	12	9.73	2.81
Pair 10	HRMOD1	136.92	12	9.13	2.63
	HRMOD2	136.03	12	8.60	2.48
Pair 11	GAIN1	203.648	12	9.255	2.672
	GAIN2	201.144	12	13.775	3.976
Pair 12	LAC_PRE1	1.033	12	.130	3.760E-02
	LAC_POS1	1.024	12	.160	4.620E-02
Pair 13	LAC_PRE2	.981	12	.121	3.487E-02
	LAC_POS2	1.013	12	.134	3.869E-02
Pair 14	DELTLAC1	-8.33E-03	12	.173	4.988E-02
	DELTLAC2	3.167E-02	12	.130	3.745E-02

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	VO2BASE1 & VO2BASE2	12	.958	.000
Pair 2	VO2MOD1 & VO2MOD2	12	.931	.000
Pair 3	AMP1 & AMP2	12	.893	.000
Pair 4	TD1 & TD2	12	.799	.002
Pair 5	TAU1 & TAU2	12	.917	.000
Pair 6	MRT1 & MRT2	12	.952	.000
Pair 7	O2DEF1 & O2DEF2	12	.932	.000
Pair 8	O2STORE1 & O2STORE2	12	.936	.000
Pair 9	HRBASE1 & HRBASE2	12	.952	.000
Pair 10	HRMOD1 & HRMOD2	12	.932	.000
Pair 11	GAIN1 & GAIN2	12	.549	.065
Pair 12	LAC_PRE1 & LAC_POS1	12	.305	.335
Pair 13	LAC_PRE2 & LAC_POS2	12	.486	.110
Pair 14	DELTLAC1 & DELTLAC2	12	.364	.245

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VO2BASE1 - VO2BASE2	-3.61	29.33	8.47	-22.24	15.02	-.426	11	.678
Pair 2	VO2MOD1 - VO2MOD2	25.15	121.28	35.01	-51.91	102.20	.718	11	.488
Pair 3	AMP1 - AMP2	28.76	104.17	30.07	-37.43	94.94	.956	11	.359
Pair 4	TD1 - TD2	.000	.973	.281	-.618	.618	.000	11	1.000
Pair 5	TAU1 - TAU2	.112	.951	.275	-.492	.717	.410	11	.690
Pair 6	MRT1 - MRT2	.113	.592	.171	-.264	.489	.658	11	.524
Pair 7	O2DEF1 - O2DEF2	15.56	46.70	13.48	-14.11	45.23	1.154	11	.273
Pair 8	O2STORE1 - O2STORE2	9.18	34.20	9.87	-12.55	30.91	.930	11	.372
Pair 9	HRBASE1 - HRBASE2	.25	3.00	.87	-1.65	2.15	.289	11	.778
Pair 10	HRMOD1 - HRMOD2	.88	3.32	.96	-1.22	2.99	.923	11	.376
Pair 11	GAIN1 - GAIN2	2.504	11.639	3.360	-4.891	9.899	.745	11	.472
Pair 12	LAC_PRE1 - LAC_POS1	8.333E-03	.173	4.988E-02	-.101	.118	.167	11	.870
Pair 13	LAC_PRE2 - LAC_POS2	-3.17E-02	.130	3.745E-02	-.114	5.077E-02	-.845	11	.416
Pair 14	DELTLAC1 - DELTLAC2	-4.00E-02	.174	5.030E-02	-.151	7.071E-02	-.795	11	.443

7.9 Paired *t*-tests: Off-transient

Paired *t*-tests for comparisons of means for tests 1 and 2 for all physiological variables and $\dot{V}O_2$ kinetic parameters during the off-transient.

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	VO2MOFF1	2741.90	12	316.04	91.23
	VO2MOFF2	2720.16	12	262.09	75.66
Pair 2	VO2BOFF1	926.33	12	102.21	29.51
	VO2BOFF2	926.37	12	93.25	26.92
Pair 3	AMP1OFF	1815.58	12	222.09	64.11
	AMP2OFF	1793.79	12	181.35	52.35
Pair 4	TD1OFF	8.895	12	2.951	.852
	TDOFF2	9.209	12	2.415	.697
Pair 5	TAU1OFF	24.488	12	2.315	.668
	TAUOFF2	24.143	12	2.406	.695
Pair 6	MRT1OFF	33.382	12	2.241	.647
	MRTOFF2	33.353	12	2.551	.736
Pair 7	O2DEB1OF	1011.02	12	147.27	42.51
	O2DEB2OF	1000.90	12	157.60	45.50

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	VO2MOFF1 & VO2MOFF2	12	.928	.000
Pair 2	VO2BOFF1 & VO2BOFF2	12	.947	.000
Pair 3	AMP1OFF & AMP2OFF	12	.894	.000
Pair 4	TD1OFF & TDOFF2	12	.857	.000
Pair 5	TAU1OFF & TAUOFF2	12	.875	.000
Pair 6	MRT1OFF & MRTOFF2	12	.775	.003
Pair 7	O2DEB1OF & O2DEB2OF	12	.937	.000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VO2MOFF1 - VO2MOFF2	21.75	121.53	35.08	-55.47	98.96	.620	11	.548
Pair 2	VO2BOFF1 - VO2BOFF2	-4.08E-02	32.97	9.52	-20.99	20.91	-.004	11	.997
Pair 3	AMP1OFF - AMP2OFF	21.79	100.91	29.13	-42.32	85.91	.748	11	.470
Pair 4	TD1OFF - TDOFF2	-.314	1.523	.440	-1.282	.654	-.714	11	.490
Pair 5	TAU1OFF - TAUOFF2	.344	1.186	.342	-.409	1.098	1.005	11	.336
Pair 6	MRT1OFF - MRTOFF2	2.917E-02	1.635	.472	-1.010	1.068	.062	11	.952
Pair 7	O2DEB1OF - O2DEB2OF	10.12	55.23	15.94	-24.97	45.22	.635	11	.538

7.10 Pre- and post-transition analysis of $\dot{V}O_2$

Within-Subjects Factors

Measure: MEASURE_1

TRANS	Dependent Variable
1	VO2B_1
2	VO2B_2
3	VO2B_3
4	VO2B_4
5	VO2B_5
6	VO2B_6
7	VO2B_ENS

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
TRANS	.002	52.984	20	.000	.412	.540	.167

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: TRANS

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TRANS	Sphericity Assumed	3478.286	6	579.714	.863	.527
	Greenhouse-Geisser	3478.286	2.469	1408.780	.863	.454
	Huynh-Feldt	3478.286	3.239	1074.025	.863	.476
	Lower-bound	3478.286	1.000	3478.286	.863	.373
Error(TRANS)	Sphericity Assumed	44324.286	66	671.580		
	Greenhouse-Geisser	44324.286	27.159	1632.026		
	Huynh-Feldt	44324.286	35.624	1244.223		
	Lower-bound	44324.286	11.000	4029.481		

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	71539885.7	1	71539885.71	1028.197	.000
Error	765357.714	11	69577.974		

Estimates

Measure: MEASURE_1

TRANS	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	919.000	29.804	853.401	984.599
2	916.167	30.072	849.978	982.355
3	921.750	29.761	856.245	987.255
4	931.917	29.264	867.508	996.325
5	914.750	30.604	847.391	982.109
6	931.667	29.250	867.288	996.046
7	924.750	28.409	862.223	987.277

Pairwise Comparisons

Measure: MEASURE_1

(I) TRANS	(J) TRANS	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	2.833	12.744	1.000	-47.162	52.829
	3	-2.750	10.054	1.000	-42.190	36.690
	4	-12.917	9.714	1.000	-51.022	25.189
	5	4.250	14.971	1.000	-54.481	62.981
	6	-12.667	15.942	1.000	-75.205	49.871
	7	-5.750	9.662	1.000	-43.652	32.152
2	1	-2.833	12.744	1.000	-52.829	47.162
	3	-5.583	6.457	1.000	-30.912	19.746
	4	-15.750	10.700	1.000	-57.724	26.224
	5	1.417	13.689	1.000	-52.283	55.117
	6	-15.500	12.313	1.000	-63.801	32.801
	7	-8.583	7.482	1.000	-37.933	20.767
3	1	2.750	10.054	1.000	-36.690	42.190
	2	5.583	6.457	1.000	-19.746	30.912
	4	-10.167	6.987	1.000	-37.575	17.242
	5	7.000	10.966	1.000	-36.019	50.019
	6	-9.917	11.074	1.000	-53.361	33.527
	7	-3.000	5.264	1.000	-23.651	17.651
4	1	12.917	9.714	1.000	-25.189	51.022
	2	15.750	10.700	1.000	-26.224	57.724
	3	10.167	6.987	1.000	-17.242	37.575
	5	17.167	12.736	1.000	-32.794	67.127
	6	.250	13.697	1.000	-53.482	53.982
	7	7.167	7.997	1.000	-24.205	38.538
5	1	-4.250	14.971	1.000	-62.981	54.481
	2	-1.417	13.689	1.000	-55.117	52.283
	3	-7.000	10.966	1.000	-50.019	36.019
	4	-17.167	12.736	1.000	-67.127	32.794
	6	-16.917	4.492	.066	-34.536	.703
	7	-10.000	7.626	1.000	-39.915	19.915
6	1	12.667	15.942	1.000	-49.871	75.205
	2	15.500	12.313	1.000	-32.801	63.801
	3	9.917	11.074	1.000	-33.527	53.361
	4	-.250	13.697	1.000	-53.982	53.482
	5	16.917	4.492	.066	-.703	34.536
	7	6.917	7.642	1.000	-23.062	36.896
7	1	5.750	9.662	1.000	-32.152	43.652
	2	8.583	7.482	1.000	-20.767	37.933
	3	3.000	5.264	1.000	-17.651	23.651
	4	-7.167	7.997	1.000	-38.538	24.205
	5	10.000	7.626	1.000	-19.915	39.915
	6	-6.917	7.642	1.000	-36.896	23.062

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Appendix 8 Chapter 5: Raw data and statistical analyses

8.1 Raw data: On- and off-transients

Dist	Name	Age	BM	Stature	Volume (Km.wk)	VO_{2max} (l.min)	VO_{2max} (ml.kg)	VO_{2max} (ml.67)	HR_{max} (b.min)	V_T (l.min)	V_T (ml.kg)	V_T (ml.67)	V_T% max	80%V_T as % max	80%V_T Speed	Gain (Rest-A_{on})
MD	dysa	19.0	65.8	176.7	56.3	3856	58.6	233	193	3250	49.4	197	84.3	67.4	12.3	161
MD	keig	19.0	71.2	182.8	48.3	4116	57.8	236	200	3350	47.1	192	81.4	65.1	11.6	163
MD	mara	40.0	71.5	175.1	40.2	4447	62.2	254	183	3600	50.3	206	81.0	64.8	11.3	182
MD	nore	23.0	59.1	175.6	88.5	3730	63.1	243	197	3183	53.9	207	85.3	68.3	12	184
MD	otor	19.0	56.8	162.2	32.2	3409	60.0	228	196	2900	51.1	194	85.1	68.1	11.3	179
MD	prya	18.0	70.6	176.0	40.2	3461	49.0	200	199	3000	42.5	173	86.7	69.3	10.4	170
MD	regd	18.0	66.6	177.0	24.1	3900	58.6	234	197	2850	42.8	171	73.1	58.5	11.0	178
MD	rees	26.0	64.3	182.0	48.3	4385	68.2	269	191	3750	58.3	230	85.5	68.4	13.5	179
MD	tayc	18.0	62.7	181.0	32.2	3937	62.8	246	204	3300	52.6	206	83.8	67.1	11.3	190
MD	wara	20.0	64.8	177.4	64.4	3877	59.8	237	207	3300	50.9	202	85.1	68.1	12.0	184
Mean		**22.0**	**65.3**	**176.6**	**47.5**	**3912**	**60.0**	**238**	**197**	**3248**	**49.9**	**198**	**83.1**	**66.5**	**11.7**	**177**
SD		**6.8**	**5.0**	**5.8**	**18.8**	**341**	**4.9**	**18**	**7**	**286**	**4.8**	**17**	**4.0**	**3.2**	**0.8**	**10**

Dist	Name	Age	BM	Stature	Volume (Km.Wk)	VO_{2max} (l.min)	VO_{2max} (ml.kg)	VO_{2max} (ml.67)	HR_{max} (b.min)	V_T (l.min)	V_T (ml.kg)	V_T (ml.67)	V_T% max	80%V_T as % max	80%V_T Speed	Gain (Rest-A_{on})
LD	bolm	18.0	67.3	180.4	77.2	4715	70.1	281.0	193.0	3800	56.5	226.5	80.6	64.5	12.9	183
LD	dunr	31.0	67.8	167.6	84.5	3959	58.4	234.8	184.0	3150	46.5	186.8	79.6	63.7	10.7	164
LD	giss	33.0	84.0	193.4	64.4	4625	55.1	237.6	188.0	3960	47.1	203.4	85.6	68.5	12.0	160
LD	jams	30.0	61.3	172.0	40.2	3452	56.3	219.0	207.0	2850	46.5	180.8	82.6	66.0	10.7	169
LD	kila	26.0	65.2	183.0	64.4	3563	54.6	216.9	185.0	3060	46.9	186.3	85.9	68.7	12.0	176
LD	Kilm	26.0	64.2	183.0	72.4	3754	58.5	230.9	181.0	3000	46.7	184.5	79.9	63.9	12.0	177
LD	manc	23.0	75.5	187.0	51.5	4107	54.4	226.6	196.0	3300	43.7	182.1	80.4	64.3	11.0	166
LD	meas	29.0	84.4	173.8	40.2	4308	51.0	220.6	187.0	3750	44.4	192.0	87.0	69.6	11.0	162
LD	nold	22.0	84.3	186.1	64.4	5793	68.7	296.9	189.0	4150	49.2	212.7	71.6	57.3	12.0	171
LD	Nora	20.0	59.7	173.3	80.5	3746	62.7	241.9	194.0	3350	56.1	216.3	89.4	71.5	12.0	189
Mean		**25.8**	**71.4**	**180.0**	**64.0**	**4202**	**59.0**	**241**	**190**	**3437**	**48.4**	**197**	**82.3**	**65.8**	**11.6**	**172**
SD		**5.0**	**9.8**	**8.1**	**15.7**	**702**	**6.3**	**27**	**7**	**447**	**4.4**	**16**	**5.1**	**4.1**	**0.7**	**9**
Combined mean		**23.9**	**68.4**	**178.3**	**55.7**	**4057**	**59.5**	**239**	**194**	**3343**	**49.1**	**197**	**82.7**	**66.2**	**11.7**	**174**
Combined SD		**6.1**	**8.2**	**7.0**	**18.8**	**557**	**5.5**	**22**	**8**	**378**	**4.6**	**16**	**4.4**	**3.6**	**0.8**	**10**

Confidence Intervals (Lamarra et al., 1987); * n reps is based on a desired CI of +/- 2 seconds

S_o	So/A%	Rest VO_2	$VO_{2(b)}$	$VO_{2(m)}$	A_{on}	TD_{on}	Tau_{on}	MRT_{on}	O_2 def (L.min)	O_2 def%	O_2 stores
0.075	4.4	423	884	2591	1707	14.1	12.6	26.8	762	44.6	402
0.112	6.5	419	938	2668	1730	14.8	12.5	27.3	787	45.5	426
0.082	4.2	392	901	2838	1937	13.8	17.1	30.9	999	51.6	446
0.099	6.0	379	887	2550	1663	14.7	9.8	24.5	680	40.9	408
0.149	10.2	394	849	2313	1464	11.4	19.2	30.7	748	51.1	279
0.150	9.6	416	927	2492	1565	14.0	17.5	31.5	821	52.5	365
0.152	8.8	394	845	2572	1726	16.2	12.3	28.5	819	47.5	466
0.141	6.9	431	969	3024	2055	14.1	13.2	27.3	935	45.5	483
0.114	6.6	363	893	2611	1718	15.4	16.1	31.5	903	52.6	442
0.142	7.8	360	920	2749	1830	15.0	11.5	26.5	808	44.1	456
0.1	**7.1**	**397**	**901**	**2641**	**1739**	**14.4**	**14.2**	**28.6**	**826**	**47.6**	**417**
0.0	**2.0**	**25**	**39**	**196**	**170**	**1.3**	**3.1**	**2.5**	**95**	**4.1**	**59**

S_o	So/A%	Rest VO_2	$VO_{2(b)}$	$VO_{2(m)}$	A_{on}	TD_{on}	Tau_{on}	MRT_{on}	O_2 def (L.min)	O_2 def%	O_2 stores
0.072	3.3	399	877	3043	2167	14.5	8.971	23.4	846	39.0	522
0.088	5.7	414	842	2402	1560	15.1	11.6	26.7	696	44.6	393
0.107	5.1	451	1036	3137	2100	15.6	9.5	25.1	879	41.8	545
0.112	7.8	325	740	2173	1433	13.1	13.7	26.8	640	44.7	313
0.093	5.1	367	824	2664	1839	15.1	13.1	28.3	866	47.1	464
0.158	8.8	350	823	2618	1795	14.1	14.44	28.6	855	47.6	423
0.163	9.1	448	955	2744	1789	15.0	13.5	28.5	849	47.4	446
0.082	4.3	421	1025	2930	1905	14.2	16.4	30.6	972	51.0	450
0.132	5.9	439	1086	3320	2234	17.4	11.1	28.6	1063	47.6	650
0.082	4.7	348	875	2604	1729	11.9	12.6	24.4	704	40.7	342
0.1	**6.0**	**396**	**908**	**2764**	**1855**	**14.6**	**12.5**	**27.1**	**837**	**45.2**	**455**
0.0	**2.0**	**46**	**112**	**348**	**257**	**1.5**	**2.3**	**2.2**	**129**	**3.7**	**99**

0.1	**6.5**	**397**	**905**	**2702**	**1797**	**14.5**	**13.3**	**27.8**	**832**	**46.4**	**436**
0.0	**2.0**	**36**	**82**	**282**	**220**	**1.3**	**2.8**	**2.4**	**110**	**4.0**	**82**

$HR_{(b)}$	$HR_{(b)}$ %HR_{max}	$HR_{(m)}$	$HR_{(m)}$ %HR_{max}	Pre [HLa]	Post [HLa]	Delta [HLa]	S_o	So/A%	A_{on}	Tau_{on}	CI (s)	*n* reps
96	49.7	151	78.2	0.98	0.91	-0.07	0.075	4.4	1707	12.6	0.7	1
84	42.0	137	68.5	1.18	1.08	-0.10	0.112	6.5	1730	12.5	1.0	2
82	44.8	140	76.5	1.85	1.85	0.01	0.082	4.2	1937	17.1	0.7	1
77	39.1	137	69.5	1.07	1.24	0.17	0.099	6.0	1663	9.8	0.9	2
108	55.1	151	77.0	1.02	0.93	-0.08	0.149	10.2	1464	19.2	1.7	5
86	43.2	148	74.4	0.95	0.89	-0.06	0.150	9.6	1565	17.5	1.6	4
100	50.8	145	73.6	0.85	0.78	-0.07	0.152	8.8	1726	12.3	1.3	3
80	41.9	149	78.0	1.00	0.93	-0.08	0.141	6.9	2055	13.2	1.0	2
104	51.0	166	81.4	1.10	1.07	-0.03	0.114	6.6	1718	16.1	1.1	2
67	32.4	150	72.5	0.97	0.95	-0.02	0.142	7.8	1830	11.5	1.1	2
88.4	**45.0**	**147.4**	**75.0**	**1.10**	**1.06**	**-0.03**	**0.1**	**7.1**	**1739**	**14.2**	**1.1**	**2**
13.1	**6.7**	**8.6**	**4.0**	**0.28**	**0.30**	**0.08**	**0.0**	**2.0**	**170**	**3.1**	**0.3**	**1**

$HR_{(b)}$	$HR_{(b)}$ %HR_{max}	$HR_{(m)}$	$HR_{(m)}$ %HR_{max}	Pre [HLa]	Post [HLa]	Delta [HLa]	S_o	So/A%	A_{on}	Tau_{on}	CI (s)	*n* reps
76	39.4	146	75.6	0.90	0.98	0.08	0.072	3	2167	9.0	0.5	1
56	30.4	122	66.3	1.00	0.93	-0.06	0.088	5.7	1560	11.6	0.8	2
79	42.0	133	70.7	0.98	1.00	0.03	0.107	5.1	2100	9.5	0.7	1
77	37.2	144	69.6	0.86	0.92	0.07	0.112	7.8	1433	13.7	1.2	3
65	35.1	136	73.5	1.01	0.93	-0.08	0.093	5.1	1839	13.1	0.8	1
67	37.0	135	74.6	0.85	0.66	-0.19	0.158	8.8	1795	14.4	1.4	3
84	42.9	144	73.5	1.14	1.03	-0.11	0.163	9.1	1789	13.5	1.4	3
81	43.3	133	71.1	1.10	1.28	0.18	0.082	4.3	1905	16.4	0.7	1
65	34.4	121	64.0	0.80	0.88	0.08	0.132	5.9	2234	11.1	0.9	2
68	35.1	138	71.1	1.25	1.33	0.08	0.082	4.7	1729	12.6	0.7	1
71.8	**37.7**	**135**	**71.0**	**0.99**	**0.99**	**0.01**	**0.1**	**6.0**	**1855**	**12.5**	**0.9**	**2**
8.9	**4.2**	**9**	**3.6**	**0.14**	**0.19**	**0.11**	**0.0**	**2.0**	**257**	**2.3**	**0.3**	**1**

80	**41**	**141**	**73**	**1.04**	**1.03**	**-0.01**	**0.1**	**6.5**	**1797**	**13.3**	**1.0**	**2**
14	**7**	**10**	**4**	**0.22**	**0.25**	**0.10**	**0.0**	**2.0**	**220**	**2.8**	**0.3**	**1**

Distance	Name	$VO_{2(m)}$	$VO_{2(b)}$	A_{off}	TD_{off}	Tau_{off}	MRT_{off}	O_2 debt	O_2 debt%
MD	dysa	2586	880	1707	11.1	22.5	33.6	955	56.0
MD	keig	2662	950	1713	10.8	24.2	35.0	999	58.3
MD	mara	2860	904	1956	6.5	29.6	36.1	1176	60.1
MD	Nore	2530	869	1661	5.2	26.0	31.2	864	52.0
MD	otor	2322	848	1473	8.4	31.1	39.5	969	65.8
MD	prya	2499	932	1566	12.7	28.8	41.6	1085	69.3
MD	regd	2576	946	1630	7.7	25.7	33.4	907	55.7
MD	rees	3011	961	2050	7.9	27.2	35.1	1200	58.5
MD	tayc	2627	898	1729	7.7	31.1	38.9	1120	64.8
MD	wara	2753	932	1820	10.6	24.8	35.4	1075	59.0
mean		**2643**	**912**	**1731**	**9**	**27**	**36**	**1035**	**60**
SD		**194**	**38**	**173**	**2**	**3**	**3**	**113**	**5**

Distance	Name	$VO_{2(m)}$	$VO_{2(b)}$	A_{off}	TD_{off}	Tau_{off}	MRT_{off}	O_2 debt	O_2 debt%
LD	bolm	3051	816	2234	5.9	24.9	30.8	1148	51.4
LD	dunr	2387	841	1547	11.1	20.1	31.1	803	51.9
LD	giss	3107	1049	2059	9.1	24.1	33.3	1141	55.4
LD	jams	2181	744	1436	8.7	25.7	34.4	824	57.4
LD	kila	2662	834	1828	1.7	26.5	28.2	859	47.0
LD	kilm	2608	817	1790	4.8	24.9	29.8	888	49.6
LD	manc	2751	919	1832	10.9	23.6	34.5	1053	57.5
LD	meas	2948	1038	1910	9.4	26.4	35.8	1140	59.7
LD	nold	3316	1102	2214	12.6	20.1	32.7	1207	54.5
LD	Nora	2594	876	1717	8.7	25.1	33.8	967	56.3
mean		**2761**	**904**	**1857**	**8.3**	**24.1**	**32.4**	**1003**	**54.1**
SD		**347**	**120**	**261**	**3.3**	**2.3**	**2.4**	**153**	**4.0**
Combined mean		**2702**	**908**	**1794**	**8.6**	**25.6**	**34.2**	**1019**	**57.0**
Combined SD		**280**	**86**	**225**	**2.8**	**3.0**	**3.3**	**132**	**5.4**

8.2 Tests of normality: Non-kinetic variables

Tests of Normality

		Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	DISTANCE	Statistic	df	Sig.	Statistic	df	Sig.
AGE	MD	.315	10	.006	.644	10	.000
	LD	.140	10	.200*	.963	10	.822
BM	MD	.155	10	.200*	.938	10	.532
	LD	.242	10	.101	.855	10	.066
STATURE	MD	.299	10	.012	.797	10	.013
	LD	.178	10	.200*	.961	10	.800
VO2MAX	MD	.171	10	.200*	.943	10	.583
	LD	.154	10	.200*	.888	10	.163
VO2ML.KG	MD	.227	10	.154	.908	10	.265
	LD	.230	10	.142	.893	10	.186
VO2ML.67	MD	.197	10	.200*	.936	10	.511
	LD	.281	10	.024	.798	10	.014
VTL.MIN	MD	.161	10	.200*	.953	10	.703
	LD	.177	10	.200*	.934	10	.489
VTML.KG	MD	.160	10	.200*	.952	10	.690
	LD	.312	10	.007	.791	10	.011

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

8.3 Tests of normality: On-transient

Tests of Normality

		Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	DISTANCE	Statistic	df	Sig.	Statistic	df	Sig.
RESTVO2	MD	.177	10	.200*	.931	10	.456
	LD	.154	10	.200*	.918	10	.337
A0	MD	.127	10	.200*	.969	10	.878
	LD	.210	10	.200*	.939	10	.545
A1	MD	.161	10	.200*	.967	10	.866
	LD	.123	10	.200*	.982	10	.977
AMP	MD	.222	10	.176	.960	10	.789
	LD	.130	10	.200*	.961	10	.799
TD	MD	.233	10	.134	.895	10	.190
	LD	.167	10	.200*	.960	10	.789
TAU	MD	.225	10	.162	.928	10	.429
	LD	.119	10	.200*	.975	10	.934
MRT	MD	.209	10	.200*	.903	10	.236
	LD	.203	10	.200*	.944	10	.594
O2DEF	MD	.219	10	.190	.963	10	.823
	LD	.225	10	.164	.938	10	.530
RELO2DEF	MD	.209	10	.200*	.903	10	.236
	LD	.203	10	.200*	.944	10	.594
O2STORES	MD	.194	10	.200*	.877	10	.120
	LD	.168	10	.200*	.970	10	.888
HRBASE	MD	.173	10	.200*	.959	10	.778
	LD	.182	10	.200*	.944	10	.594
HRMOD	MD	.237	10	.118	.894	10	.186
	LD	.199	10	.200*	.909	10	.272
PRELAC	MD	.294	10	.015	.675	10	.000
	LD	.139	10	.200*	.955	10	.731
POSTLAC	MD	.276	10	.029	.736	10	.002
	LD	.232	10	.136	.903	10	.238
DELTALAC	MD	.232	10	.134	.736	10	.002
	LD	.200	10	.200*	.942	10	.573

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

8.4 Tests of normality: Off-transient

Tests of Normality

		Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	DISTANCE	Statistic	df	Sig.	Statistic	df	Sig.
AO_OFF	MD	.160	10	.200*	.971	10	.896
	LD	.116	10	.200*	.981	10	.970
A1_OFF	MD	.200	10	.200*	.944	10	.601
	LD	.199	10	.200*	.903	10	.236
AMP_OFF	MD	.203	10	.200*	.958	10	.762
	LD	.138	10	.200*	.957	10	.754
TD_OFF	MD	.178	10	.200*	.953	10	.702
	LD	.250	10	.077	.932	10	.468
TAU_OFF	MD	.145	10	.200*	.947	10	.639
	LD	.229	10	.147	.829	10	.033
MRT_OFF	MD	.185	10	.200*	.955	10	.723
	LD	.143	10	.200*	.961	10	.801
O2DEBOFF	MD	.133	10	.200*	.960	10	.791
	LD	.214	10	.200*	.893	10	.183
RELDEBT	MD	.185	10	.200*	.955	10	.723
	LD	.143	10	.200*	.961	10	.801

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

8.5 Independent *t*-tests between MD and LD runners

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
AGE	Equal variances assumed	.105	.750	-1.421	18	.172	-3.8000	2.67416	-9.41819	1.81819
	Equal variances not assumed			-1.421	16.467	.174	-3.8000	2.67416	-9.45593	1.85593
BM	Equal variances assumed	9.313	.007	-1.731	18	.101	-6.0300	3.48311	-13.34774	1.28774
	Equal variances not assumed			-1.731	13.312	.107	-6.0300	3.48311	-13.53691	1.47691
STATURE	Equal variances assumed	2.754	.114	-1.079	18	.295	-3.3800	3.13358	-9.96341	3.20341
	Equal variances not assumed			-1.079	16.291	.296	-3.3800	3.13358	-10.01328	3.25328
VO2MAX	Equal variances assumed	3.343	.084	-1.177	18	.255	-290.4000	246.72034	-808.740	227.94019
	Equal variances not assumed			-1.177	13.025	.260	-290.4000	246.72034	-823.304	242.50401
VO2ML.KG	Equal variances assumed	1.074	.314	.406	18	.689	1.0300	2.53409	-4.29393	6.35393
	Equal variances not assumed			.406	17.012	.689	1.0300	2.53409	-4.31618	6.37618
VO2ML.67	Equal variances assumed	1.205	.287	-.251	18	.805	-2.5796	10.28475	-24.18705	19.02785
	Equal variances not assumed			-.251	15.743	.805	-2.5796	10.28475	-24.41123	19.25203
VTL.MIN	Equal variances assumed	4.192	.055	-1.124	18	.276	-188.7000	167.82829	-541.294	163.89415
	Equal variances not assumed			-1.124	15.317	.278	-188.7000	167.82829	-545.775	168.37463
VTML.KG	Equal variances assumed	.026	.874	.737	18	.470	1.5300	2.07464	-2.82866	5.88866
	Equal variances not assumed			.737	17.878	.470	1.5300	2.07464	-2.83080	5.89080

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
SPEED	Equal variances assumed	.000	1.000	.113	18	.911	.0400	.35374	-.70318	.78318
	Equal variances not assumed			.113	17.635	.911	.0400	.35374	-.70429	.78429
GAIN	Equal variances assumed	.000	.993	1.291	18	.213	5.4500	4.22149	-3.41901	14.31901
	Equal variances not assumed			1.291	17.993	.213	5.4500	4.22149	-3.41925	14.31925
RESTVO2	Equal variances assumed	6.104	.024	.038	18	.970	.6300	16.43607	-33.90089	35.16089
	Equal variances not assumed			.038	13.938	.970	.6300	16.43607	-34.63662	35.89662
A0	Equal variances assumed	12.484	.002	-.187	18	.854	-7.0000	37.46023	-85.70102	71.70102
	Equal variances not assumed			-.187	11.110	.855	-7.0000	37.46023	-89.34999	75.34999
A1	Equal variances assumed	3.317	.085	-.972	18	.344	-122.7000	126.28971	-388.025	142.62483
	Equal variances not assumed			-.972	14.163	.348	-122.7000	126.28971	-393.273	147.87306
AMP	Equal variances assumed	1.647	.216	-1.187	18	.251	-115.6000	97.37884	-320.185	88.98534
	Equal variances not assumed			-1.187	15.634	.253	-115.6000	97.37884	-322.427	91.22699
TD	Equal variances assumed	.167	.688	-.407	18	.689	-.2500	.61450	-1.54102	1.04102
	Equal variances not assumed			-.407	17.628	.689	-.2500	.61450	-1.54297	1.04297
TAU	Equal variances assumed	2.293	.147	1.402	18	.178	1.6889	1.20473	-.84213	4.21993
	Equal variances not assumed			1.402	16.554	.179	1.6889	1.20473	-.85808	4.23588
MRT	Equal variances assumed	.261	.616	1.377	18	.185	1.4500	1.05264	-.76152	3.66152
	Equal variances not assumed			1.377	17.856	.185	1.4500	1.05264	-.76280	3.66280

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
O2DEF	Equal variances assumed	.552	.467	-.214	18	.833	-10.8237	50.64679	-117.229	95.58129
	Equal variances not assumed			-.214	16.491	.833	-10.8237	50.64679	-117.931	96.28349
RELO2DEF	Equal variances assumed	.261	.616	1.377	18	.185	2.4167	1.75440	-1.26920	6.10253
	Equal variances not assumed			1.377	17.856	.185	2.4167	1.75440	-1.27133	6.10467
HRBASE	Equal variances assumed	1.965	.178	3.317	18	.004	16.6000	5.00444	6.08606	27.11394
	Equal variances not assumed			3.317	15.829	.004	16.6000	5.00444	5.98172	27.21828
HRMOD	Equal variances assumed	.013	.910	3.181	18	.005	12.2000	3.83551	4.14190	20.25810
	Equal variances not assumed			3.181	17.999	.005	12.2000	3.83551	4.14188	20.25812
PRELAC	Equal variances assumed	.612	.444	1.109	18	.282	.1095	.09875	-.09796	.31696
	Equal variances not assumed			1.109	13.397	.287	.1095	.09875	-.10319	.32219
POSTLAC	Equal variances assumed	.634	.436	.615	18	.546	.0700	.11374	-.16895	.30895
	Equal variances not assumed			.615	15.196	.547	.0700	.11374	-.17216	.31216
DELTALAC	Equal variances assumed	2.909	.105	-.910	18	.375	-.0395	.04338	-.13065	.05165
	Equal variances not assumed			-.910	16.059	.376	-.0395	.04338	-.13144	.05244

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
AO_OFF	Equal variances assumed	3.476	.079	-.937	18	.361	-117.9000	125.77736	-382.148	146.34842
	Equal variances not assumed			-.937	14.133	.364	-117.9000	125.77736	-387.428	151.62775
A1_OFF	Equal variances assumed	11.651	.003	.211	18	.835	8.4000	39.72187	-75.05254	91.85254
	Equal variances not assumed			.211	10.800	.836	8.4000	39.72187	-79.22505	96.02505
AMP_OFF	Equal variances assumed	1.385	.255	-1.275	18	.219	-126.2000	99.00531	-334.202	81.80243
	Equal variances not assumed			-1.275	15.648	.221	-126.2000	99.00531	-336.466	84.06584
TD_OFF	Equal variances assumed	.576	.458	.447	18	.660	.5700	1.27442	-2.10746	3.24746
	Equal variances not assumed			.447	16.282	.661	.5700	1.27442	-2.12785	3.26785
TAU_OFF	Equal variances assumed	1.297	.270	2.489	18	.023	2.9600	1.18932	.46133	5.45867
	Equal variances not assumed			2.489	16.989	.023	2.9600	1.18932	.45062	5.46938
MRT_OFF	Equal variances assumed	.451	.511	2.826	18	.011	3.5400	1.25264	.90830	6.17170
	Equal variances not assumed			2.826	16.767	.012	3.5400	1.25264	.89436	6.18564
O2DEBOFF	Equal variances assumed	2.584	.125	.536	18	.599	32.2820	60.23789	-94.27312	158.83712
	Equal variances not assumed			.536	16.607	.599	32.2820	60.23789	-95.03840	159.60240
RELDEBT	Equal variances assumed	.451	.511	2.826	18	.011	5.9000	2.08774	1.51383	10.28617
	Equal variances not assumed			2.826	16.767	.012	5.9000	2.08774	1.49060	10.30940

8.6 Paired *t*-tests between on- and off-transient $\dot{V}O_2$ kinetic parameters

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	AMP	1797.3000	20	220.07824	49.21099
	AMP_OFF	1793.6000	20	224.99319	50.31001
Pair 2	TD	14.4750	20	1.34355	.30043
	TD_OFF	8.5750	20	2.78905	.62365
Pair 3	TAU	13.3356	20	2.76143	.61747
	TAU_OFF	25.6200	20	3.00098	.67104
Pair 4	MRT	27.8250	20	2.40873	.53861
	MRT_OFF	34.2100	20	3.27573	.73248
Pair 5	O2DEF	831.6122	20	110.36888	24.67923
	O2DEBOFF	1019.1210	20	132.14525	29.54858
Pair 6	RELO2DEF	46.3750	20	4.01455	.89768
	RELDEBT	57.0167	20	5.45955	1.22079

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	AMP & AMP_OFF	20	.990	.000
Pair 2	TD & TD_OFF	20	.133	.575
Pair 3	TAU & TAU_OFF	20	.706	.000
Pair 4	MRT & MRT_OFF	20	.617	.004
Pair 5	O2DEF & O2DEBOFF	20	.788	.000
Pair 6	RELO2DEF & RELDEBT	20	.617	.004

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
				95% Confidence Interval of the Difference				
	Mean	Std. Deviation	Std. Error Mean	Lower	Upper			
Pair 1 AMP - AMP_OFF	3.7000	31.48617	7.04052	-11.0360	18.4360	.526	19	.605
Pair 2 TD - TD_OFF	5.9000	2.93006	.65518	4.5287	7.2713	9.005	19	.000
Pair 3 TAU - TAU_OFF	-12.2844	2.21906	.49620	-13.3230	-11.2459	-24.757	19	.000
Pair 4 MRT - MRT_OFF	-6.3850	2.60713	.58297	-7.6052	-5.1648	-10.952	19	.000
Pair 5 O2DEF - O2DEBO	-87.5088	81.63570	8.25430	-25.7155	-49.3021	-10.272	19	.000
Pair 6 RELO2DEF - RELD	-10.6417	4.34522	.97162	-12.6753	-8.6080	-10.952	19	.000

8.7 Correlations between on- and off-transient kinetic parameters in combined MD and LD runners

Correlations

		AMP	TD	TAU	MRT	O2DEF	RELO2DEF	AMP_OFF	TD_OFF	TAU_OFF	MRT_OFF	O2DEBOFF	RELDEBT
AMP	Pearson Correlation	1	.515*	-.446*	-.225	.764**	-.225	.990**	-.088	-.273	-.323	.721**	-.323
	Sig. (2-tailed)	.	.020	.049	.341	.000	.341	.000	.713	.244	.165	.000	.165
	N	20	20	20	20	20	20	20	20	20	20	20	20
TD	Pearson Correlation	.515*	1	-.503*	-.015	.478*	-.015	.456*	.133	-.478*	-.324	.222	-.324
	Sig. (2-tailed)	.020	.	.024	.950	.033	.950	.043	.575	.033	.164	.346	.164
	N	20	20	20	20	20	20	20	20	20	20	20	20
TAU	Pearson Correlation	-.446*	-.503*	1	.872**	.158	.872**	-.414	.055	.706**	.697**	.091	.697**
	Sig. (2-tailed)	.049	.024	.	.000	.506	.000	.070	.819	.000	.001	.704	.001
	N	20	20	20	20	20	20	20	20	20	20	20	20
MRT	Pearson Correlation	-.225	-.015	.872**	1	.452*	1.000**	-.222	.136	.542*	.617**	.225	.617**
	Sig. (2-tailed)	.341	.950	.000	.	.045	.	.348	.567	.014	.004	.341	.004
	N	20	20	20	20	20	20	20	20	20	20	20	20
O2DEF	Pearson Correlation	.764**	.478*	.158	.452*	1	.452*	.754**	.011	.078	.085	.788**	.085
	Sig. (2-tailed)	.000	.033	.506	.045	.	.045	.000	.964	.743	.722	.000	.722
	N	20	20	20	20	20	20	20	20	20	20	20	20
RELO2DEF	Pearson Correlation	-.225	-.015	.872**	1.000**	.452*	1	-.222	.136	.542*	.617**	.225	.617**
	Sig. (2-tailed)	.341	.950	.000	.	.045	.	.348	.567	.014	.004	.341	.004
	N	20	20	20	20	20	20	20	20	20	20	20	20
AMP_OFF	Pearson Correlation	.990**	.456*	-.414	-.222	.754**	-.222	1	-.097	-.242	-.302	.742**	-.302
	Sig. (2-tailed)	.000	.043	.070	.348	.000	.348	.	.685	.304	.195	.000	.195
	N	20	20	20	20	20	20	20	20	20	20	20	20
TD_OFF	Pearson Correlation	-.088	.133	.055	.136	.011	.136	-.097	1	-.368	.513*	.277	.513*
	Sig. (2-tailed)	.713	.575	.819	.567	.964	.567	.685	.	.111	.021	.236	.021
	N	20	20	20	20	20	20	20	20	20	20	20	20
TAU_OFF	Pearson Correlation	-.273	-.478*	.706**	.542*	.078	.542*	-.242	-.368	1	.609**	.185	.609**
	Sig. (2-tailed)	.244	.033	.000	.014	.743	.014	.304	.111	.	.004	.434	.004
	N	20	20	20	20	20	20	20	20	20	20	20	20
MRT_OFF	Pearson Correlation	-.323	-.324	.697**	.617**	.085	.617**	-.302	.513*	.609**	1	.411	1.000**
	Sig. (2-tailed)	.165	.164	.001	.004	.722	.004	.195	.021	.004	.	.072	.000
	N	20	20	20	20	20	20	20	20	20	20	20	20
O2DEBOFF	Pearson Correlation	.721**	.222	.091	.225	.788**	.225	.742**	.277	.185	.411	1	.411
	Sig. (2-tailed)	.000	.346	.704	.341	.000	.341	.000	.236	.434	.072	.	.072
	N	20	20	20	20	20	20	20	20	20	20	20	20
RELDEBT	Pearson Correlation	-.323	-.324	.697**	.617**	.085	.617**	-.302	.513*	.609**	1.000**	.411	1
	Sig. (2-tailed)	.165	.164	.001	.004	.722	.004	.195	.021	.004	.000	.072	.
	N	20	20	20	20	20	20	20	20	20	20	20	20

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

8.8 Correlations between on- and off-transient kinetic parameters in MD runners

Correlations

		AMP	TD	TAU	MRT	O2DEF	RELO2DEF	AMP_OFF	TD_OFF	TAU_OFF	MRT_OFF	O2DEBOFF	RELDEBT
AMP	Pearson Correlation	1	.347	-.330	-.241	.695*	-.241	.982**	-.258	-.195	-.379	.623	-.379
	Sig. (2-tailed)	.	.326	.351	.502	.026	.502	.000	.472	.589	.280	.054	.280
	N	10	10	10	10	10	10	10	10	10	10	10	10
TD	Pearson Correlation	.347	1	-.640*	-.293	.102	-.293	.231	-.062	-.419	-.438	-.093	-.438
	Sig. (2-tailed)	.326	.	.046	.412	.779	.412	.521	.866	.228	.206	.798	.206
	N	10	10	10	10	10	10	10	10	10	10	10	10
TAU	Pearson Correlation	-.330	-.640*	1	.922**	.386	.922**	-.246	.139	.811**	.871**	.425	.871**
	Sig. (2-tailed)	.351	.046	.	.000	.271	.000	.493	.701	.004	.001	.221	.001
	N	10	10	10	10	10	10	10	10	10	10	10	10
MRT	Pearson Correlation	-.241	-.293	.922**	1	.528	1.000**	-.195	.146	.794**	.862**	.476	.862**
	Sig. (2-tailed)	.502	.412	.000	.	.117	.	.589	.688	.006	.001	.165	.001
	N	10	10	10	10	10	10	10	10	10	10	10	10
O2DEF	Pearson Correlation	.695*	.102	.386	.528	1	.528	.714*	-.142	.414	.286	.882**	.286
	Sig. (2-tailed)	.026	.779	.271	.117	.	.117	.020	.696	.235	.423	.001	.423
	N	10	10	10	10	10	10	10	10	10	10	10	10
RELO2DEF	Pearson Correlation	-.241	-.293	.922**	1.000**	.528	1	-.195	.146	.794**	.862**	.476	.862**
	Sig. (2-tailed)	.502	.412	.000	.	.117	.	.589	.688	.006	.001	.165	.001
	N	10	10	10	10	10	10	10	10	10	10	10	10
AMP_OFF	Pearson Correlation	.982**	.231	-.246	-.195	.714*	-.195	1	-.249	-.119	-.300	.698*	-.300
	Sig. (2-tailed)	.000	.521	.493	.589	.020	.589	.	.487	.744	.400	.025	.400
	N	10	10	10	10	10	10	10	10	10	10	10	10
TD_OFF	Pearson Correlation	-.258	-.062	.139	.146	-.142	.146	-.249	1	-.324	.442	.096	.442
	Sig. (2-tailed)	.472	.866	.701	.688	.696	.688	.487	.	.362	.201	.793	.201
	N	10	10	10	10	10	10	10	10	10	10	10	10
TAU_OFF	Pearson Correlation	-.195	-.419	.811**	.794**	.414	.794**	-.119	-.324	1	.706*	.424	.706*
	Sig. (2-tailed)	.589	.228	.004	.006	.235	.006	.744	.362	.	.022	.222	.022
	N	10	10	10	10	10	10	10	10	10	10	10	10
MRT_OFF	Pearson Correlation	-.379	-.438	.871**	.862**	.286	.862**	-.300	.442	.706*	1	.473	1.000**
	Sig. (2-tailed)	.280	.206	.001	.001	.423	.001	.400	.201	.022	.	.167	.000
	N	10	10	10	10	10	10	10	10	10	10	10	10
O2DEBOFF	Pearson Correlation	.623	-.093	.425	.476	.882**	.476	.698*	.096	.424	.473	1	.473
	Sig. (2-tailed)	.054	.798	.221	.165	.001	.165	.025	.793	.222	.167	.	.167
	N	10	10	10	10	10	10	10	10	10	10	10	10
RELDEBT	Pearson Correlation	-.379	-.438	.871**	.862**	.286	.862**	-.300	.442	.706*	1.000**	.473	1
	Sig. (2-tailed)	.280	.206	.001	.001	.423	.001	.400	.201	.022	.000	.167	.
	N	10	10	10	10	10	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

8.9 Correlations between on- and off-transient kinetic parameters in LD runners

Correlations

		AMP	TD	TAU	MRT	O2DEF	RELO2DEF	AMP_OFF	TD_OFF	TAU_OFF	MRT_OFF	O2DEBOFF	RELDEBT
AMP	Pearson Correlation	1	.611	-.501	-.100	.822**	-.100	.993**	.030	-.155	-.099	.873**	-.099
	Sig. (2-tailed)	.	.060	.140	.784	.004	.784	.000	.933	.670	.785	.001	.785
	N	10	10	10	10	10	10	10	10	10	10	10	10
TD	Pearson Correlation	.611	1	-.365	.307	.714*	.307	.581	.273	-.611	-.216	.450	-.216
	Sig. (2-tailed)	.060	.	.300	.388	.020	.388	.078	.445	.061	.548	.192	.548
	N	10	10	10	10	10	10	10	10	10	10	10	10
TAU	Pearson Correlation	-.501	-.365	1	.774**	-.001	.774**	-.498	-.087	.422	.288	-.313	.288
	Sig. (2-tailed)	.140	.300	.	.009	.999	.009	.143	.811	.224	.419	.379	.419
	N	10	10	10	10	10	10	10	10	10	10	10	10
MRT	Pearson Correlation	-.100	.307	.774**	1	.482	1.000**	-.118	.086	.019	.137	-.023	.137
	Sig. (2-tailed)	.784	.388	.009	.	.158	.	.746	.812	.958	.706	.951	.706
	N	10	10	10	10	10	10	10	10	10	10	10	10
O2DEF	Pearson Correlation	.822**	.714*	-.001	.482	1	.482	.802**	.099	-.146	.002	.758*	.002
	Sig. (2-tailed)	.004	.020	.999	.158	.	.158	.005	.786	.688	.996	.011	.996
	N	10	10	10	10	10	10	10	10	10	10	10	10
RELO2DEF	Pearson Correlation	-.100	.307	.774**	1.000**	.482	1	-.118	.086	.019	.137	-.023	.137
	Sig. (2-tailed)	.784	.388	.009	.	.158	.	.746	.812	.958	.706	.951	.706
	N	10	10	10	10	10	10	10	10	10	10	10	10
AMP_OFF	Pearson Correlation	.993**	.581	-.498	-.118	.802**	-.118	1	.015	-.127	-.094	.880**	-.094
	Sig. (2-tailed)	.000	.078	.143	.746	.005	.746	.	.966	.727	.797	.001	.797
	N	10	10	10	10	10	10	10	10	10	10	10	10
TD_OFF	Pearson Correlation	.030	.273	-.087	.086	.099	.086	.015	1	-.684*	.703*	.359	.703*
	Sig. (2-tailed)	.933	.445	.811	.812	.786	.812	.966	.	.029	.023	.308	.023
	N	10	10	10	10	10	10	10	10	10	10	10	10
TAU_OFF	Pearson Correlation	-.155	-.611	.422	.019	-.146	.019	-.127	-.684*	1	.038	-.115	.038
	Sig. (2-tailed)	.670	.061	.224	.958	.688	.958	.727	.029	.	.916	.752	.916
	N	10	10	10	10	10	10	10	10	10	10	10	10
MRT_OFF	Pearson Correlation	-.099	-.216	.288	.137	.002	.137	-.094	.703*	.038	1	.389	1.000**
	Sig. (2-tailed)	.785	.548	.419	.706	.996	.706	.797	.023	.916	.	.267	.
	N	10	10	10	10	10	10	10	10	10	10	10	10
O2DEBOFF	Pearson Correlation	.873**	.450	-.313	-.023	.758*	-.023	.880**	.359	-.115	.389	1	.389
	Sig. (2-tailed)	.001	.192	.379	.951	.011	.951	.001	.308	.752	.267	.	.267
	N	10	10	10	10	10	10	10	10	10	10	10	10
RELDEBT	Pearson Correlation	-.099	-.216	.288	.137	.002	.137	-.094	.703*	.038	1.000**	.389	1
	Sig. (2-tailed)	.785	.548	.419	.706	.996	.706	.797	.023	.916	.	.267	.
	N	10	10	10	10	10	10	10	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Appendix 9 Chapter 6: Raw data and statistical analyses

9.1 Raw data

	Distance	Filename	Age (yrs)	Stature (cm)	BM (kg)	Km/Wk	$VO_{2\,max}$ (ml.min)	$VO_{2\,max}$ (ml.kg.min)	V_T (ml.min)	V_T (ml.kg.min)	V_T (%max)	V_T speed (km.h)	RE 16 (ml.min)	RE 16 (ml.kg.min)
1	MD	brod	19.0	177.2	69.6	40.2	3856	55.4	3100	44.5	80.4	15.1	3372	48.4
2	MD	davc	22.0	168.1	62.1	40.2	3931	63.3	3100	49.9	78.9	13.0	3705	59.7
3	MD	davg	22.0	168.1	61.5	40.2	4086	66.4	3400	55.3	83.2	13.6	3782	61.5
4	MD	dysa	19.0	176.7	65.8	56.3	3856	58.6	3250	49.4	84.3	15.4	3360	51.1
5	MD	ells	19.0	190.0	79.3	32.2	4452	56.1	3500	44.1	78.6	13.6	3936	49.6
6	MD	evaa	21.0	184.7	74.2	37.0	4089	55.1	3300	44.5	80.7	12.8	3842	51.8
7	MD	keig	19.0	182.8	71.2	48.3	4116	57.8	3350	47.1	81.4	14.5	3702	52.0
8	MD	mara	40.0	175.1	71.5	40.2	4447	62.2	3600	50.3	81.0	14.2	3983	55.7
9	MD	nore	23.0	175.6	59.1	88.5	3734	63.2	3183	53.9	85.2	16.7	3069	51.9
10	MD	otor	19.0	162.2	56.8	32.2	3409	60.0	2900	51.1	85.1	14.1	3205	56.4
11	MD	prya	18.0	176.0	70.6	40.2	3461	49.0	3000	42.5	86.7	13.0		
12	MD	ragd	18.0	177.0	66.6	24.1	3900	58.6	2850	42.8	73.1	13.4	3308	49.7
13	MD	rees	26.0	182.0	64.3	48.3	4385	68.2	3750	58.3	85.5	16.9	3620	56.3
14	MD	tayc	18.0	181.0	62.7	32.2	3937	62.8	3300	52.6	83.8	14.1	3547	56.6
15	MD	wara	20.0	177.4	64.8	64.4	3877	59.8	3300	50.9	85.1	15.0	3438	53.1
16	MD	wilp	18.0	175.7	66.1	32.2	3946	59.7	3300	49.9	83.6	13.8	3658	55.3
	Mean		21.3	176.9	66.6	43.5	3968	59.8	3261	49.2	82.3	14.3	3568	53.9
	SD		5.5	6.8	5.8	15.6	300	4.7	241	4.6	3.5	1.2	270	3.8

	Distance	Filename	Age (yrs)	Stature (cm)	BM (kg)	Km/Wk	VO_{2max} (ml.min)	VO_{2max} (ml.kg.min)	V_T (ml.min)	V_T (ml.kg.min)	V_T (%max)	V_T speed (km.h)	RE 16 (ml.min)	RE 16 (ml.kg.min)
1	LD	bait	20.0	185.9	68.1	88.5	4500	66.1	3400	49.9	75.6	15.0	3689	54.2
2	LD	bolm	18.0	180.4	67.3	77.2	4715	70.1	3800	56.5	80.6	16.2	3741	55.6
3	LD	dunr	31.0	167.6	67.8	84.5	3959	58.4	3150	46.5	79.6	13.4	3801	56.1
4	LD	giss	33.0	193.4	84.0	64.4	4625	55.1	3960	47.1	85.6	15.8	4063	48.4
5	LD	jams	30.0	172.0	61.3	40.2	3452	56.3	2850	46.5	82.6	14.0	3242	52.9
6	LD	kila	26.0	183.0	65.2	64.4	3563	54.6	3060	46.9	85.9	15.0	3220	49.4
7	LD	kilm	26.0	183.0	64.2	72.4	3754	58.5	3000	46.7	79.9	15.0	3168	49.3
8	LD	lee	24.0	184.0	69.5	80.5	4564	65.7	3900	56.1	85.5	16.1	3910	56.3
9	LD	manc	23.0	187.0	75.5	51.5	4107	54.4	3300	43.7	80.4	13.8	3590	47.5
10	LD	meas	29.0	173.8	84.4	40.2	4308	51.0	3750	44.4	87.0	13.7	4280	50.7
11	LD	nold	22.0	186.1	84.3	64.4	5793	68.7	4150	49.2	71.6	15.2	4330	51.4
12	LD	Nora	20.0	173.3	59.7	80.5	3753	62.9	3350	56.1	89.3	17.3	3126	52.4
13	LD	thaa	25.0	183.3	76.0	64.4	4372	57.5	3600	47.4	82.3	15.4	3727	49.0
14	LD	warj	23.0	181.0	64.0	56.3	3622	56.6	3000	46.9	82.8	15.6	3098	48.4
15	LD	warr	26.0	171.0	60.8	72.4	4071	67.0	3400	55.9	83.5	16.3	3343	55.0
16	LD	wris	24.0	180.0	65.6	64.4	3935	60.0	3300	50.3	83.9	15.7	3357	51.2
	Mean		25.0	180.3	69.9	66.6	4193	60.2	3436	49.4	82.3	15.2	3605	51.7
	SD		4.2	7.0	8.4	14.4	583	5.8	387	4.4	4.4	1.1	404	3.0

	Combined Mean		23.3	178.7	68.3	55.8	4085	60.0	3350	49.3	82.2	14.8	3585	52.7
	Combined SD		5.1	7.0	7.3	18.9	470	5.2	329	4.4	3.9	1.2	341	3.5

RE (%max)	VO_2 80%V_T	Speed 80%V_T	S_o (l.min)	So/A_{on} (%)	Rest VO_2 (ml.min)	$\dot{V}O_{2(b)on}$ (ml.min)	$VO_{2(m)on}$ (ml.min)	A_{on} (ml.min)	Gain (ml.kg.km)	Td_{on} (s)	Tau_{on} (s)	MRT_{on} (s)
87.4	2480	12.1	0.115	5.7	366	896	2921	2025	182.1	11.5	16.8	28.3
94.3	2480	10.9	0.081	6.2	378	698	2008	1309	144.4	13.0	18.1	31.1
92.6	2720	9.9	0.075	4.3	459	938	2680	1742	218.9	6.5	20.0	26.5
87.1	2600	12.3	0.075	4.4	423	884	2591	1707	160.7	14.1	12.6	26.8
88.4	2800	11.0	0.129	6.9	413	962	2831	1869	166.4	14.0	20.7	34.7
94.0	2640	10.2	0.104	6.1	328	1025	2740	1715	191.3	9.8	24.7	34.4
89.9	2680	11.6	0.112	6.5	419	938	2668	1730	163.4	14.8	12.5	27.3
89.6	2880	11.3	0.082	4.2	392	901	2838	1937	181.7	13.8	17.1	30.9
82.2	2546	12.0	0.099	6.0	379	887	2550	1663	183.7	14.7	9.8	24.5
94.0	2320	11.3	0.149	10.2	394	849	2313	1464	179.5	11.4	19.2	30.7
	2400	10.4	0.150	9.6	416	927	2492	1565	169.6	14.0	17.5	31.5
84.8	2280	11.0	0.152	8.8	394	845	2572	1726	178.4	16.2	12.3	28.5
82.6	3000	13.5	0.141	6.9	431	969	3024	2055	179.3	14.1	13.2	27.3
90.1	2640	11.3	0.114	6.6	363	893	2611	1718	190.3	15.4	16.1	31.5
88.7	2640	12.0	0.142	7.8	360	920	2749	1830	184.4	15.0	11.5	26.5
92.7	2640	11.1	0.096	5.1	411	913	2789	1877	194.5	10.9	20.2	31.2
89.2	**2609**	**11.4**	**0.1**	**6.6**	**395**	**903**	**2649**	**1746**	**179**	**13.1**	**16.4**	**29.5**
3.9	**193**	**0.9**	**0.0**	**1.8**	**33**	**71**	**244**	**194**	**17**	**2.5**	**4.1**	**3.0**

RE (%max)	VO_2 80%V_T	Speed 80%V_T	S_o (l.min)	S_o/A_{on} (%)	Rest VO_2 (ml.min)	$VO_{2(b)on}$ (ml.min)	$VO_{2(m)on}$ (ml.min)	A_{on} (ml.min)	Gain (ml.kg.km)	Td_{on} (s)	Tau_{on} (s)	MRT_{on} (s)
82.0	2720	12.0	0.104	7.3	379	841	2269	1427	138.7	19.1	9.2	28.2
79.3	3040	12.9	0.072	3.3	399	877	3043	2167	182.7	14.5	9.0	23.4
96.0	2520	10.7	0.088	5.7	414	842	2402	1560	164.4	15.1	11.6	26.7
87.8	3168	12.0	0.107	5.1	451	1036	3137	2100	159.9	15.6	9.5	25.1
93.9	2280	10.7	0.112	7.8	325	740	2173	1433	169.0	13.1	13.7	26.8
90.4	2448	12.0	0.093	5.1	367	824	2664	1839	176.1	15.1	13.1	28.3
84.4	2400	12.0	0.158	8.8	350	823	2618	1795	176.6	14.1	14.4	28.6
85.7	3120	12.9	0.102	5.0	340	1022	3042	2021	180.8	15.5	10.1	25.6
87.4	2640	11.0	0.163	9.1	448	955	2744	1789	165.9	15.0	13.5	28.5
99.4	3000	11.0	0.082	4.3	421	1025	2930	1905	162.1	14.2	16.4	30.6
74.7	3320	12.0	0.132	5.9	439	1086	3320	2234	170.9	17.4	11.1	28.6
83.3	2680	12.0	0.082	4.7	348	875	2604	1729	188.9	11.9	12.6	24.4
85.2	2880	12.3	0.071	3.4	394	953	3043	2090	170.0	16.1	14.0	30.1
85.5	2400	12.5	0.101	7.3	361	816	2191	1374	137.2	18.1	15.1	33.2
82.1	2720	13.0	0.070	3.3	370	861	3001	2140	199.7	16.2	11.8	28.0
85.3	2640	12.5	0.106	6.2	417	895	2598	1703	159.6	15.9	12.4	28.3
86.4	**2749**	**12.0**	**0.1**	**5.8**	**389**	**904**	**2736**	**1832**	**169**	**15.4**	**12.3**	**27.8**
6.2	**310**	**0.8**	**0.0**	**1.9**	**40**	**98**	**355**	**281**	**16**	**1.8**	**2.2**	**2.4**
87.6	**2680**	**11.7**	**0.1**	**6.2**	**392**	**903**	**2689**	**1786**	**173**	**14.4**	**14.2**	**28.5**
5.3	**264**	**0.9**	**0.0**	**1.8**	**36**	**84**	**303**	**241**	**17**	**2.4**	**3.8**	**2.8**

So (l.min)	So/A_{on} (%)	A_{on} (ml.min)	Tau_{on} (s)	Cl (s)	*n* reps	VO_2m_{off}	VO_2b_{off}	A_{off}	Td_{off}	Tau_{off}	MRT_{off}
0.115	5.7	2025	16.8	0.9	2	2945	950	1995	11.1	22.5	33.5
0.081	6.2	1309	18.1	1.0	2	1978	687	1291	7.4	22.5	29.8
0.075	4.3	1747	20.0	0.7	1	2765	928	1837	9.3	26.1	35.4
0.075	4.4	1707	12.6	0.7	1	2586	880	1707	11.1	22.5	33.6
0.129	6.9	1869	20.7	1.2	3	2843	972	1871	10.0	31.9	41.9
0.104	6.1	1715	24.7	1.1	2	2760	1005	1755	10.9	29.3	40.2
0.112	6.5	1730	12.5	1.0	2	2662	950	1713	10.8	24.2	35.0
0.082	4.2	1937	17.1	0.7	1	2860	904	1956	6.5	29.6	36.1
0.099	6.0	1663	9.8	0.9	2	2530	869	1661	5.2	26.0	31.2
0.149	10.2	1464	19.2	1.7	5	2322	848	1473	8.4	31.1	39.5
0.150	9.6	1565	17.5	1.6	4	2499	932	1566	12.7	28.8	41.6
0.152	8.8	1726	12.3	1.3	3	2576	946	850	7.7	25.7	33.4
0.141	6.9	2055	13.2	1.0	2	3011	961	2050	7.9	27.2	35.1
0.114	6.6	1718	16.1	1.1	2	2627	898	1729	7.7	31.1	38.9
0.142	7.8	1830	11.5	1.1	2	2753	932	1820	10.6	24.8	35.4
0.096	5.1	1877	20.2	0.9	2	2808	933	1875	8.8	26.8	35.6
0.1	**6.6**	**1746**	**16.4**	**1.0**	**2**	**2658**	**912**	**1697**	**9.1**	**26.9**	**36.0**
0.0	**1.8**	**194**	**4.1**	**0.3**	**1**	**254**	**72**	**298**	**2.0**	**3.2**	**3.5**

S_o (l.min)	So/A_{on} (%)	A_{on} (ml.min)	Tau_{on} (s)	Cl (s)	*n* reps	VO_2m_{off}	VO_2b_{off}	A_{off}	Td_{off}	Tau_{off}	MRT_{off}
0.104	7.3	1427	9.2	*1.0*	2	2233	838	1395	9.6	21.8	31.4
0.072	3.3	2167	9.0	0.5	1	3051	816	2234	5.9	22.1	30.8
0.088	5.7	1560	11.6	0.8	2	2387	841	1547	11.1	20.1	31.1
0.107	5.1	2100	9.5	0.7	1	3107	1049	2059	9.1	24.1	33.3
0.112	7.8	1433	13.7	1.2	3	2181	744	1436	8.7	25.7	34.4
0.093	5.1	1839	13.1	0.8	1	2662	834	1828	1.7	26.5	28.2
0.158	8.8	1795	14.4	1.4	3	2608	817	1790	4.8	24.9	29.8
0.102	5.0	2021	10.1	0.7	1	3045	1031	2015	10.3	22.5	32.8
0.163	9.1	1789	13.5	1.4	3	2751	919	1832	10.9	23.6	34.5
0.082	4.3	1905	16.4	0.7	1	2948	1038	1910	9.4	26.4	35.8
0.132	5.9	2234	11.1	0.9	2	3316	1102	2214	12.6	20.1	32.7
0.082	4.7	1729	12.6	0.7	1	2594	876	1717	8.7	25.1	33.8
0.071	3.4	2090	14.0	0.5	1	3053	976	2077	9.5	25.7	35.1
0.101	7.3	1374	15.1	1.1	2	2199	798	1401	7.7	29.6	37.3
0.070	3.3	2140	11.8	0.3	1	2985	841	2145	7.5	26.3	33.8
0.106	6.2	1703	12.4	0.9	2	2590	901	1689	10.4	24.6	35.0
0.1	**5.8**	**1832**	**12.3**	**0.9**	**2**	**2732**	**901**	**1831**	**8.6**	**24.3**	**33.1**
0.0	**1.9**	**281**	**2.2**	**0.3**	**1**	**357**	**106**	**284**	**2.7**	**2.5**	**2.4**

0.1	**6.2**	**1786**	**14.2**	**1.0**	**2.0**	**2691.2**	**905.9**	**1760.1**	**8.9**	**25.6**	**34.5**
0.0	**1.8**	**241**	**3.8**	**0.3**	**1.0**	**307.0**	**89.5**	**294.2**	**2.3**	**3.1**	**3.3**

9.2 Tests of normality for on- and off-transient $\dot{V}O_2$ kinetics

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
AMP_ON	.111	32	.200*	.973	32	.580
GAIN	.098	32	.200*	.960	32	.271
TDON	.177	32	.012	.933	32	.048
TAUON	.132	32	.167	.943	32	.094
MRTON	.160	32	.036	.970	32	.501
AMP_OFF	.061	32	.200*	.982	32	.852
TDOFF	.111	32	.200*	.944	32	.100
TAUOFF	.113	32	.200*	.967	32	.417
MRTOFF	.135	32	.143	.960	32	.272

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

9.3 Example statistical analyses illustrating the empirical identification of the most appropriate BM exponent for expressing $\dot{V}O_{2\,max}$ in MD and LD runners

Variables Entered/Removed[b]

Model	Variables Entered	Variables Removed	Method
1	DISTANCE, Ln BM, DIS_LNBM[a]	.	Enter

a. All requested variables entered.

b. Dependent Variable: Ln Vo2max

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.691[a]	.478	.422	.08276

a. Predictors: (Constant), DISTANCE, Ln BM, DIS_LNBM

b. Dependent Variable: Ln Vo2max

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	8.2113	8.4897	8.3080	.07525	32
Std. Predicted Value	-1.285	2.415	.000	1.000	32
Standard Error of Predicted Value	.02069	.04827	.02826	.00771	32
Adjusted Predicted Value	8.2083	8.5301	8.3084	.07578	32
Residual	-.1621	.1756	.0000	.07865	32
Std. Residual	-1.959	2.122	.000	.950	32
Stud. Residual	-2.060	2.446	-.002	1.027	32
Deleted Residual	-.1793	.2332	-.0003	.09229	32
Stud. Deleted Residual	-2.196	2.708	.005	1.068	32
Mahal. Distance	.969	9.576	2.906	2.270	32
Cook's Distance	.000	.490	.046	.094	32
Centered Leverage Value	.031	.309	.094	.073	32

a. Dependent Variable: Ln Vo2max

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.176	3	.059	8.545	.000[a]
	Residual	.192	28	.007		
	Total	.367	31			

a. Predictors: (Constant), DISTANCE, Ln BM, DIS_LNBM

b. Dependent Variable: Ln Vo2max

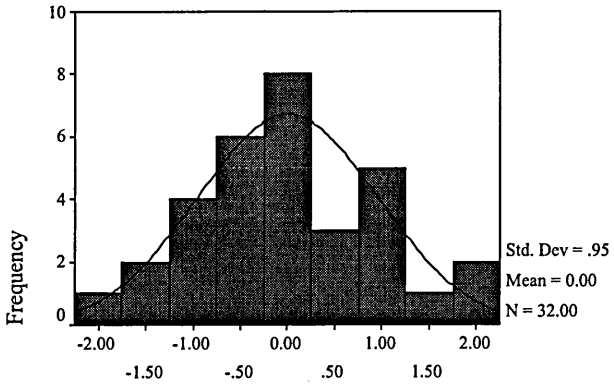
Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	6.353	1.031		6.163	.000
	DIS_LNBM	.342	.306	6.765	1.116	.274
	Ln BM	.460	.246	.439	1.873	.071
	DISTANCE	-1.419	1.290	-6.623	-1.100	.281

a. Dependent Variable: Ln Vo2max

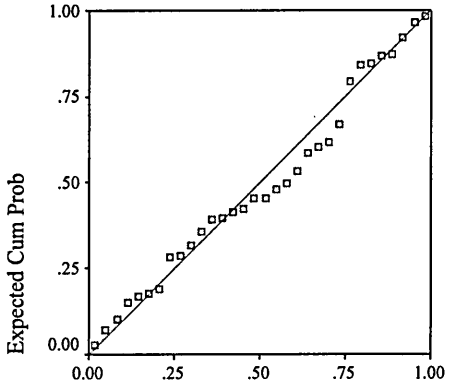
Histogram

Dependent Variable: Ln Vo2max



Normal P-P Plot of Regression Standa

Dependent Variable: Ln Vo2max



Variables Entered/Removed[b]

Model	Variables Entered	Variables Removed	Method
1	DISTANCE, Ln BM[a]	.	Enter

a. All requested variables entered.

b. Dependent Variable: Ln Vo2max

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.674[a]	.455	.417	.08310

a. Predictors: (Constant), DISTANCE, Ln BM

b. Dependent Variable: Ln Vo2max

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.167	2	.084	12.092	.000[a]
	Residual	.200	29	.007		
	Total	.367	31			

a. Predictors: (Constant), DISTANCE, Ln BM

b. Dependent Variable: Ln Vo2max

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	5.430	.618		8.784	.000
	Ln BM	.680	.147	.649	4.619	.000
	DISTANCE	1.957E-02	.030	.091	.650	.521

a. Dependent Variable: Ln Vo2max

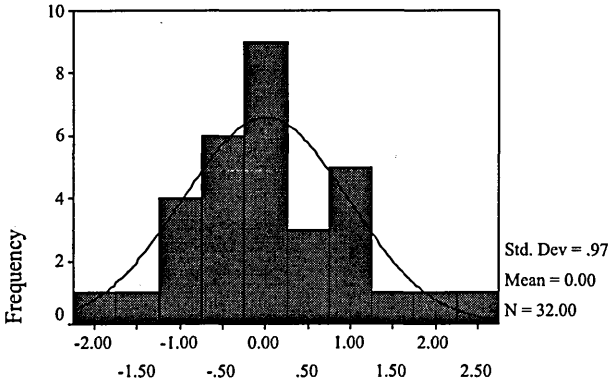
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	8.1770	8.4659	8.3080	.07340	32
Std. Predicted Value	-1.785	2.151	.000	1.000	32
Standard Error of Predicted Value	.02078	.03552	.02503	.00466	32
Adjusted Predicted Value	8.1839	8.4877	8.3080	.07334	32
Residual	-.1756	.1993	.0000	.08038	32
Std. Residual	-2.113	2.398	.000	.967	32
Stud. Residual	-2.196	2.650	.000	1.023	32
Deleted Residual	-.1897	.2434	.0000	.09002	32
Stud. Deleted Residual	-2.363	2.992	.009	1.072	32
Mahal. Distance	.969	4.695	1.937	1.159	32
Cook's Distance	.000	.518	.041	.093	32
Centered Leverage Value	.031	.151	.062	.037	32

a. Dependent Variable: Ln Vo2max

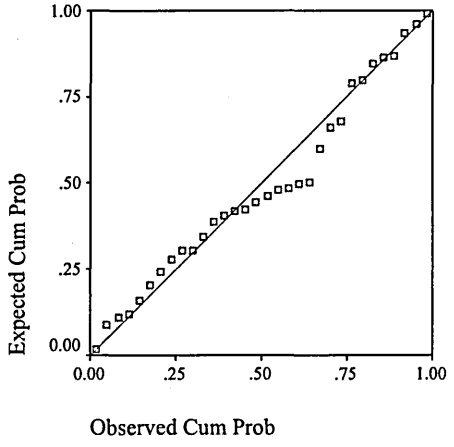
Histogram

Dependent Variable: Ln Vo2max



Normal P-P Plot of Regression Standa

Dependent Variable: Ln Vo2max



Univariate Analysis of Variance

Between-Subjects Factors

		Value Label	N
DISTANCE	.00	MD	16
	1.00	LD	16

Levene's Test of Equality of Error Variances[a]

Dependent Variable: Ln Vo2max

F	df1	df2	Sig.
2.568	1	30	.120

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept+LOGBMASS+DISTANCE

Tests of Between-Subjects Effects

Dependent Variable: Ln Vo2max

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.167[a]	2	8.351E-02	12.092	.000
Intercept	.530	1	.530	76.678	.000
LOGBMASS	.147	1	.147	21.332	.000
DISTANCE	2.922E-03	1	2.922E-03	.423	.521
Error	.200	29	6.906E-03		
Total	2209.111	32			
Corrected Total	.367	31			

a. R Squared = .455 (Adjusted R Squared = .417)

Estimated Marginal Means

DISTANCE

Dependent Variable: Ln Vo2max

DISTANCE	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
MD	8.298[a]	.021	8.255	8.341
LD	8.318[a]	.021	8.275	8.361

a. Evaluated at covariates appeared in the model: Ln BM = 4.2178.

9.4 Correlations matrix between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetic parameters for combined runners

Correlations

		ABVO2MAX	RELVO2MA	Vo2max 0.67	Vo2max 0.75	Vo2max (Darveau =0.86)	Vo2max 0.68	TAUON	MRTON	TAUOFF	MRTOFF
ABVO2MAX	Pearson Correlation	1	.470**	.781**	.713**	.610**	.773**	-.267	-.108	-.344	-.129
	Sig. (2-tailed)	.	.007	.000	.000	.000	.000	.139	.557	.054	.480
	N	32	32	32	32	32	32	32	32	32	32
RELVO2MA	Pearson Correlation	.470**	1	.918**	.954**	.986**	.923**	-.351*	-.421*	-.235	-.351*
	Sig. (2-tailed)	.007	.	.000	.000	.000	.000	.049	.017	.196	.049
	N	32	32	32	32	32	32	32	32	32	32
Vo2max 0.67	Pearson Correlation	.781**	.918**	1	.995**	.971**	1.000**	-.368*	-.344	-.322	-.307
	Sig. (2-tailed)	.000	.000	.	.000	.000	.000	.038	.054	.072	.087
	N	32	32	32	32	32	32	32	32	32	32
Vo2max 0.75	Pearson Correlation	.713**	.954**	.995**	1	.990**	.996**	-.369*	-.369*	-.305	-.323
	Sig. (2-tailed)	.000	.000	.000	.	.000	.000	.038	.038	.090	.071
	N	32	32	32	32	32	32	32	32	32	32
Vo2max (Darveau=0.86)	Pearson Correlation	.610**	.986**	.971**	.990**	1	.974**	-.364*	-.396*	-.276	-.339
	Sig. (2-tailed)	.000	.000	.000	.000	.	.000	.040	.025	.126	.058
	N	32	32	32	32	32	32	32	32	32	32
Vo2max 0.68	Pearson Correlation	.773**	.923**	1.000**	.996**	.974**	1	-.368*	-.348	-.320	-.309
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.038	.051	.074	.085
	N	32	32	32	32	32	32	32	32	32	32
TAUON	Pearson Correlation	-.267	-.351*	-.368*	-.369*	-.364*	-.368*	1	.770**	.597**	.634**
	Sig. (2-tailed)	.139	.049	.038	.038	.040	.038	.	.000	.000	.000
	N	32	32	32	32	32	32	32	32	32	32
MRTON	Pearson Correlation	-.108	-.421*	-.344	-.369*	-.396*	-.348	.770**	1	.599**	.644**
	Sig. (2-tailed)	.557	.017	.054	.038	.025	.051	.000	.	.000	.000
	N	32	32	32	32	32	32	32	32	32	32
TAUOFF	Pearson Correlation	-.344	-.235	-.322	-.305	-.276	-.320	.597**	.599**	1	.731**
	Sig. (2-tailed)	.054	.196	.072	.090	.126	.074	.000	.000	.	.000
	N	32	32	32	32	32	32	32	32	32	32
MRTOFF	Pearson Correlation	-.129	-.351*	-.307	-.323	-.339	-.309	.634**	.644**	.731**	1
	Sig. (2-tailed)	.480	.049	.087	.071	.058	.085	.000	.000	.000	.
	N	32	32	32	32	32	32	32	32	32	32

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.5 Correlations matrix between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetic parameters for MD runners

Correlations

		ABVO2MAX	RELVO2MA	Vo2max 0.67	Vo2max 0.75	Vo2max (Darveau =0.86)	Vo2max 0.68	TAUON	MRTON	TAUOFF	MRTOFF
ABVO2MAX	Pearson Correlation	1	.370	.671**	.595*	.493	.661**	.126	.172	.128	.005
	Sig. (2-tailed)	.	.159	.004	.015	.053	.005	.642	.523	.636	.986
	N	16	16	16	16	16	16	16	16	16	16
RELVO2MA	Pearson Correlation	.370	1	.937**	.967**	.991**	.941**	-.242	-.433	-.090	-.493
	Sig. (2-tailed)	.159	.	.000	.000	.000	.000	.366	.094	.739	.052
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.67	Pearson Correlation	.671**	.937**	1	.995**	.976**	1.000**	-.150	-.286	-.032	-.397
	Sig. (2-tailed)	.004	.000	.	.000	.000	.000	.580	.283	.907	.128
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.75	Pearson Correlation	.595*	.967**	.995**	1	.992**	.996**	-.177	-.331	-.049	-.429
	Sig. (2-tailed)	.015	.000	.000	.	.000	.000	.511	.211	.857	.098
	N	16	16	16	16	16	16	16	16	16	16
Vo2max (Darveau=0.86)	Pearson Correlation	.493	.991**	.976**	.992**	1	.978**	-.209	-.383	-.069	-.463
	Sig. (2-tailed)	.053	.000	.000	.000	.	.000	.436	.143	.798	.071
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.68	Pearson Correlation	.661**	.941**	1.000**	.996**	.978**	1	-.153	-.292	-.034	-.401
	Sig. (2-tailed)	.005	.000	.000	.000	.000	.	.570	.273	.900	.124
	N	16	16	16	16	16	16	16	16	16	16
TAUON	Pearson Correlation	.126	-.242	-.150	-.177	-.209	-.153	1	.798**	.468	.559*
	Sig. (2-tailed)	.642	.366	.580	.511	.436	.570	.	.000	.068	.024
	N	16	16	16	16	16	16	16	16	16	16
MRTON	Pearson Correlation	.172	-.433	-.286	-.331	-.383	-.292	.798**	1	.633**	.677**
	Sig. (2-tailed)	.523	.094	.283	.211	.143	.273	.000	.	.008	.004
	N	16	16	16	16	16	16	16	16	16	16
TAUOFF	Pearson Correlation	.128	-.090	-.032	-.049	-.069	-.034	.468	.633**	1	.823**
	Sig. (2-tailed)	.636	.739	.907	.857	.798	.900	.068	.008	.	.000
	N	16	16	16	16	16	16	16	16	16	16
MRTOFF	Pearson Correlation	.005	-.493	-.397	-.429	-.463	-.401	.559*	.677**	.823**	1
	Sig. (2-tailed)	.986	.052	.128	.098	.071	.124	.024	.004	.000	.
	N	16	16	16	16	16	16	16	16	16	16

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.6 Correlations matrix between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetic parameters for LD runners

Correlations

		ABVO2MAX	RELVO2MA	Vo2max 0.67	Vo2max 0.75	Vo2max (Darveau =0.86)	Vo2max 0.68	TAUON	MRTON	TAUOFF	MRTOFF
ABVO2MAX	Pearson Correlation	1	.532*	.822**	.763**	.669**	.815**	-.521*	-.169	-.592*	-.052
	Sig. (2-tailed)	.	.034	.000	.001	.005	.000	.038	.533	.016	.848
	N	16	16	16	16	16	16	16	16	16	16
RELVO2MA	Pearson Correlation	.532*	1	.920**	.953**	.985**	.924**	-.700**	-.442	-.404	-.263
	Sig. (2-tailed)	.034	.	.000	.000	.000	.000	.003	.086	.120	.324
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.67	Pearson Correlation	.822**	.920**	1	.995**	.973**	1.000**	-.715**	-.374	-.549*	-.205
	Sig. (2-tailed)	.000	.000	.	.000	.000	.000	.002	.154	.028	.446
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.75	Pearson Correlation	.763**	.953**	.995**	1	.991**	.996**	-.722**	-.396	-.522*	-.223
	Sig. (2-tailed)	.001	.000	.000	.	.000	.000	.002	.129	.038	.407
	N	16	16	16	16	16	16	16	16	16	16
Vo2max (Darveau=0.86)	Pearson Correlation	.669**	.985**	.973**	.991**	1	.976**	-.720**	-.421	-.476	-.244
	Sig. (2-tailed)	.005	.000	.000	.000	.	.000	.002	.105	.063	.362
	N	16	16	16	16	16	16	16	16	16	16
Vo2max 0.68	Pearson Correlation	.815**	.924**	1.000**	.996**	.976**	1	-.716**	-.377	-.546*	-.207
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.002	.150	.029	.441
	N	16	16	16	16	16	16	16	16	16	16
TAUON	Pearson Correlation	-.521*	-.700**	-.715**	-.722**	-.720**	-.716**	1	.704**	.586*	.434
	Sig. (2-tailed)	.038	.003	.002	.002	.002	.002	.	.002	.017	.093
	N	16	16	16	16	16	16	16	16	16	16
MRTON	Pearson Correlation	-.169	-.442	-.374	-.396	-.421	-.377	.704**	1	.413	.454
	Sig. (2-tailed)	.533	.086	.154	.129	.105	.150	.002	.	.112	.077
	N	16	16	16	16	16	16	16	16	16	16
TAUOFF	Pearson Correlation	-.592*	-.404	-.549*	-.522*	-.476	-.546*	.586*	.413	1	.395
	Sig. (2-tailed)	.016	.120	.028	.038	.063	.029	.017	.112	.	.130
	N	16	16	16	16	16	16	16	16	16	16
MRTOFF	Pearson Correlation	-.052	-.263	-.205	-.223	-.244	-.207	.434	.454	.395	1
	Sig. (2-tailed)	.848	.324	.446	.407	.362	.441	.093	.077	.130	.
	N	16	16	16	16	16	16	16	16	16	16

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

9.7 Correlations matrix between V_T and $\dot{V}O_2$ kinetic parameters for combined runners

Correlations

		VTL.MIN	VTML.KG	VT scaled 0.67	VT scaled 0.75	VT (Darveau =0.75)	VT 0.539	TAUON	MRTON	TAUOFF	MRTOFF
VTL.MIN	Pearson Correlation	1	.365*	.690**	.611**	.611**	.807**	-.282	-.224	-.228	-.060
	Sig. (2-tailed)	.	.040	.000	.000	.000	.000	.118	.218	.210	.745
	N	32	32	32	32	32	32	32	32	32	32
VTML.KG	Pearson Correlation	.365*	1	.926**	.960**	.960**	.844**	-.307	-.512**	-.044	-.234
	Sig. (2-tailed)	.040	.	.000	.000	.000	.000	.087	.003	.811	.197
	N	32	32	32	32	32	32	32	32	32	32
VT scaled 0.67	Pearson Correlation	.690**	.926**	1	.995**	.995**	.984**	-.354*	-.489**	-.129	-.209
	Sig. (2-tailed)	.000	.000	.	.000	.000	.000	.047	.005	.481	.252
	N	32	32	32	32	32	32	32	32	32	32
VT scaled 0.75	Pearson Correlation	.611**	.960**	.995**	1	1.000**	.961**	-.347	-.502**	-.108	-.219
	Sig. (2-tailed)	.000	.000	.000	.	.	.000	.052	.003	.556	.228
	N	32	32	32	32	32	32	32	32	32	32
VT (Darveau=0.75)	Pearson Correlation	.611**	.960**	.995**	1.000**	1	.961**	-.347	-.502**	-.108	-.219
	Sig. (2-tailed)	.000	.000	.000	.	.	.000	.052	.003	.556	.228
	N	32	32	32	32	32	32	32	32	32	32
VT 0.539	Pearson Correlation	.807**	.844**	.984**	.961**	.961**	1	-.358*	-.453**	-.162	-.185
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.044	.009	.376	.310
	N	32	32	32	32	32	32	32	32	32	32
TAUON	Pearson Correlation	-.282	-.307	-.354*	-.347	-.347	-.358*	1	.770**	.597**	.634**
	Sig. (2-tailed)	.118	.087	.047	.052	.052	.044	.	.000	.000	.000
	N	32	32	32	32	32	32	32	32	32	32
MRTON	Pearson Correlation	-.224	-.512**	-.489**	-.502**	-.502**	-.453**	.770**	1	.599**	.644**
	Sig. (2-tailed)	.218	.003	.005	.003	.003	.009	.000	.	.000	.000
	N	32	32	32	32	32	32	32	32	32	32
TAUOFF	Pearson Correlation	-.228	-.044	-.129	-.108	-.108	-.162	.597**	.599**	1	.731**
	Sig. (2-tailed)	.210	.811	.481	.556	.556	.376	.000	.000	.	.000
	N	32	32	32	32	32	32	32	32	32	32
MRTOFF	Pearson Correlation	-.060	-.234	-.209	-.219	-.219	-.185	.634**	.644**	.731**	1
	Sig. (2-tailed)	.745	.197	.252	.228	.228	.310	.000	.000	.000	.
	N	32	32	32	32	32	32	32	32	32	32

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

9.8 Correlations matrix between V_T and $\dot{V}O_2$ kinetic parameters for MD runners

Correlations

		VTL.MIN	VTML.KG	VT scaled 0.67	VT scaled 0.75	VT (Darveau =0.75)	VT 0.539	TAUON	MRTON	TAUOFF	MRTOFF
VTL.MIN	Pearson Correlation	1	.494	.712**	.657**	.657**	.801**	.049	.005	.183	.095
	Sig. (2-tailed)	.	.052	.002	.006	.006	.000	.856	.984	.496	.726
	N	16	16	16	16	16	16	16	16	16	16
VTML.KG	Pearson Correlation	.494	1	.962**	.980**	.980**	.916**	-.260	-.492	-.019	-.328
	Sig. (2-tailed)	.052	.	.000	.000	.000	.000	.331	.053	.944	.214
	N	16	16	16	16	16	16	16	16	16	16
VT scaled 0.67	Pearson Correlation	.712**	.962**	1	.997**	.997**	.991**	-.199	-.399	.034	-.242
	Sig. (2-tailed)	.002	.000	.	.000	.000	.000	.461	.126	.900	.368
	N	16	16	16	16	16	16	16	16	16	16
VT scaled 0.75	Pearson Correlation	.657**	.980**	.997**	1	1.000**	.978**	-.218	-.428	.019	-.268
	Sig. (2-tailed)	.006	.000	.000	.	.	.000	.418	.098	.945	.315
	N	16	16	16	16	16	16	16	16	16	16
VT (Darveau=0.75)	Pearson Correlation	.657**	.980**	.997**	1.000**	1	.978**	-.218	-.428	.019	-.268
	Sig. (2-tailed)	.006	.000	.000	.	.	.000	.418	.098	.945	.315
	N	16	16	16	16	16	16	16	16	16	16
VT 0.539	Pearson Correlation	.801**	.916**	.991**	.978**	.978**	1	-.161	-.340	.062	-.189
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.551	.197	.819	.483
	N	16	16	16	16	16	16	16	16	16	16
TAUON	Pearson Correlation	.049	-.260	-.199	-.218	-.218	-.161	1	.798**	.468	.559*
	Sig. (2-tailed)	.856	.331	.461	.418	.418	.551	.	.000	.068	.024
	N	16	16	16	16	16	16	16	16	16	16
MRTON	Pearson Correlation	.005	-.492	-.399	-.428	-.428	-.340	.798**	1	.633**	.677**
	Sig. (2-tailed)	.984	.053	.126	.098	.098	.197	.000	.	.008	.004
	N	16	16	16	16	16	16	16	16	16	16
TAUOFF	Pearson Correlation	.183	-.019	.034	.019	.019	.062	.468	.633**	1	.823**
	Sig. (2-tailed)	.496	.944	.900	.945	.945	.819	.068	.008	.	.000
	N	16	16	16	16	16	16	16	16	16	16
MRTOFF	Pearson Correlation	.095	-.328	-.242	-.268	-.268	-.189	.559*	.677**	.823**	1
	Sig. (2-tailed)	.726	.214	.368	.315	.315	.483	.024	.004	.000	.
	N	16	16	16	16	16	16	16	16	16	16

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.9 Correlations matrix between V_T and $\dot{V}O_2$ kinetic parameters for LD runners

Correlations

		VTL.MIN	VTML.KG	VT scaled 0.67	VT scaled 0.75	VT (Darveau =0.75)	VT 0.539	TAUON	MRTON	TAUOFF	MRTOFF
VTL.MIN	Pearson Correlation	1	.315	.697**	.608*	.608*	.822**	-.482	-.288	-.407	.058
	Sig. (2-tailed)	.	.235	.003	.012	.012	.000	.059	.280	.117	.830
	N	16	16	16	16	16	16	16	16	16	16
VTML.KG	Pearson Correlation	.315	1	.900**	.945**	.945**	.799**	-.570*	-.586*	-.066	-.137
	Sig. (2-tailed)	.235	.	.000	.000	.000	.000	.021	.017	.809	.612
	N	16	16	16	16	16	16	16	16	16	16
VT scaled 0.67	Pearson Correlation	.697**	.900**	1	.993**	.993**	.981**	-.657**	-.575*	-.241	-.082
	Sig. (2-tailed)	.003	.000	.	.000	.000	.000	.006	.020	.369	.761
	N	16	16	16	16	16	16	16	16	16	16
VT scaled 0.75	Pearson Correlation	.608*	.945**	.993**	1	1.000**	.952**	-.647**	-.589*	-.199	-.100
	Sig. (2-tailed)	.012	.000	.000	.	.	.000	.007	.016	.461	.713
	N	16	16	16	16	16	16	16	16	16	16
VT (Darveau=0.75)	Pearson Correlation	.608*	.945**	.993**	1.000**	1	.952**	-.647**	-.589*	-.199	-.100
	Sig. (2-tailed)	.012	.000	.000	.	.	.000	.007	.016	.461	.713
	N	16	16	16	16	16	16	16	16	16	16
VT 0.539	Pearson Correlation	.822**	.799**	.981**	.952**	.952**	1	-.652**	-.534*	-.302	-.051
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.006	.033	.256	.851
	N	16	16	16	16	16	16	16	16	16	16
TAUON	Pearson Correlation	-.482	-.570*	-.657**	-.647**	-.647**	-.652**	1	.704**	.586*	.434
	Sig. (2-tailed)	.059	.021	.006	.007	.007	.006	.	.002	.017	.093
	N	16	16	16	16	16	16	16	16	16	16
MRTON	Pearson Correlation	-.288	-.586*	-.575*	-.589*	-.589*	-.534*	.704**	1	.413	.454
	Sig. (2-tailed)	.280	.017	.020	.016	.016	.033	.002	.	.112	.077
	N	16	16	16	16	16	16	16	16	16	16
TAUOFF	Pearson Correlation	-.407	-.066	-.241	-.199	-.199	-.302	.586*	.413	1	.395
	Sig. (2-tailed)	.117	.809	.369	.461	.461	.256	.017	.112	.	.130
	N	16	16	16	16	16	16	16	16	16	16
MRTOFF	Pearson Correlation	.058	-.137	-.082	-.100	-.100	-.051	.434	.454	.395	1
	Sig. (2-tailed)	.830	.612	.761	.713	.713	.851	.093	.077	.130	.
	N	16	16	16	16	16	16	16	16	16	16

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.10 Correlations matrix between RE and $\dot{V}O_2$ kinetic parameters for combined runners

Correlations

		RE16L.MI	RE16ML.K	RE scaled 0.67	RE scaled 0.75	RE scaled (Darveau/empirical)	REMAX	TAUON	MRTON	TAUOFF	MRTOFF
RE16L.MI	Pearson Correlation	1	.158	.645**	.528**	.543**	.122	.113	.166	-.170	.155
	Sig. (2-tailed)	.	.396	.000	.002	.002	.514	.543	.371	.360	.406
	N	31	31	31	31	31	31	31	31	31	31
RE16ML.K	Pearson Correlation	.158	1	.856**	.922**	.914**	.221	.173	-.117	.034	-.010
	Sig. (2-tailed)	.396	.	.000	.000	.000	.231	.352	.530	.857	.959
	N	31	31	31	31	31	31	31	31	31	31
RE scaled 0.67	Pearson Correlation	.645**	.856**	1	.990**	.992**	.235	.195	-.001	-.065	.072
	Sig. (2-tailed)	.000	.000	.	.000	.000	.203	.293	.997	.728	.702
	N	31	31	31	31	31	31	31	31	31	31
RE scaled 0.75	Pearson Correlation	.528**	.922**	.990**	1	1.000**	.239	.196	-.032	-.039	.051
	Sig. (2-tailed)	.002	.000	.000	.	.000	.196	.291	.863	.835	.784
	N	31	31	31	31	31	31	31	31	31	31
RE scaled (Darveau/empirical)	Pearson Correlation	.543**	.914**	.992**	1.000**	1	.238	.196	-.029	-.042	.054
	Sig. (2-tailed)	.002	.000	.000	.000	.	.196	.291	.879	.821	.774
	N	31	31	31	31	31	31	31	31	31	31
REMAX	Pearson Correlation	.122	.221	.235	.239	.238	1	.593**	.370*	.258	.291
	Sig. (2-tailed)	.514	.231	.203	.196	.196	.	.000	.040	.161	.112
	N	31	31	31	31	31	31	31	31	31	31
TAUON	Pearson Correlation	.113	.173	.195	.196	.196	.593**	1	.770**	.597**	.634**
	Sig. (2-tailed)	.543	.352	.293	.291	.291	.000	.	.000	.000	.000
	N	31	31	31	31	31	31	32	32	32	32
MRTON	Pearson Correlation	.166	-.117	-.001	-.032	-.029	.370*	.770**	1	.599**	.644**
	Sig. (2-tailed)	.371	.530	.997	.863	.879	.040	.000	.	.000	.000
	N	31	31	31	31	31	31	32	32	32	32
TAUOFF	Pearson Correlation	-.170	.034	-.065	-.039	-.042	.258	.597**	.599**	1	.731**
	Sig. (2-tailed)	.360	.857	.728	.835	.821	.161	.000	.000	.	.000
	N	31	31	31	31	31	31	32	32	32	32
MRTOFF	Pearson Correlation	.155	-.010	.072	.051	.054	.291	.634**	.644**	.731**	1
	Sig. (2-tailed)	.406	.959	.702	.784	.774	.112	.000	.000	.000	.
	N	31	31	31	31	31	31	32	32	32	32

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.11 Correlations matrix between RE and $\dot{V}O_2$ kinetic parameters for MD runners

Correlations

		RE16L.MI	RE16ML.K	RE scaled 0.67	RE scaled 0.75	RE scaled (Darveau/empirical)	REMAX	TAUON	MRTON	TAUOFF	MRTOFF
RE16L.MI	Pearson Correlation	1	.270	.636*	.544*	.556*	.429	.594*	.583*	.302	.396
	Sig. (2-tailed)	.	.330	.011	.036	.032	.110	.020	.022	.274	.144
	N	15	15	15	15	15	15	15	15	15	15
RE16ML.K	Pearson Correlation	.270	1	.915**	.955**	.951**	.477	.251	-.020	.113	-.097
	Sig. (2-tailed)	.330	.	.000	.000	.000	.072	.366	.945	.689	.732
	N	15	15	15	15	15	15	15	15	15	15
RE scaled 0.67	Pearson Correlation	.636*	.915**	1	.994**	.995**	.559*	.446	.223	.208	.080
	Sig. (2-tailed)	.011	.000	.	.000	.000	.030	.096	.425	.457	.776
	N	15	15	15	15	15	15	15	15	15	15
RE scaled 0.75	Pearson Correlation	.544*	.955**	.994**	1	1.000**	.545*	.399	.158	.185	.032
	Sig. (2-tailed)	.036	.000	.000	.	.000	.035	.141	.574	.510	.911
	N	15	15	15	15	15	15	15	15	15	15
RE scaled (Darveau/empirical)	Pearson Correlation	.556*	.951**	.995**	1.000**	1	.548*	.405	.166	.188	.037
	Sig. (2-tailed)	.032	.000	.000	.000	.	.035	.135	.555	.503	.895
	N	15	15	15	15	15	15	15	15	15	15
REMAX	Pearson Correlation	.429	.477	.559*	.545*	.548*	1	.755**	.562*	.185	.328
	Sig. (2-tailed)	.110	.072	.030	.035	.035	.	.001	.029	.509	.233
	N	15	15	15	15	15	15	15	15	15	15
TAUON	Pearson Correlation	.594*	.251	.446	.399	.405	.755**	1	.798**	.468	.559*
	Sig. (2-tailed)	.020	.366	.096	.141	.135	.001	.	.000	.068	.024
	N	15	15	15	15	15	15	16	16	16	16
MRTON	Pearson Correlation	.583*	-.020	.223	.158	.166	.562*	.798**	1	.633**	.677**
	Sig. (2-tailed)	.022	.945	.425	.574	.555	.029	.000	.	.008	.004
	N	15	15	15	15	15	15	16	16	16	16
TAUOFF	Pearson Correlation	.302	.113	.208	.185	.188	.185	.468	.633**	1	.823**
	Sig. (2-tailed)	.274	.689	.457	.510	.503	.509	.068	.008	.	.000
	N	15	15	15	15	15	15	16	16	16	16
MRTOFF	Pearson Correlation	.396	-.097	.080	.032	.037	.328	.559*	.677**	.823**	1
	Sig. (2-tailed)	.144	.732	.776	.911	.895	.233	.024	.004	.000	.
	N	15	15	15	15	15	15	16	16	16	16

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

9.12 Correlations matrix between RE and $\dot{V}O_2$ kinetic parameters for LD runners

Correlations

		RE16L.MI	RE16ML.K	RE scaled 0.67	RE scaled 0.75	RE scaled (Darveau/empirical)	REMAX	TAUON	MRTON	TAUOFF	MRTOFF
RE16L.MI	Pearson Correlation	1	.134	.731**	.609*	.626**	.028	-.295	-.095	-.556*	.045
	Sig. (2-tailed)	.	.620	.001	.012	.010	.918	.267	.726	.025	.869
	N	16	16	16	16	16	16	16	16	16	16
RE16ML.K	Pearson Correlation	.134	1	.774**	.867**	.857**	-.083	-.551*	-.532*	-.426	-.274
	Sig. (2-tailed)	.620	.	.000	.000	.000	.761	.027	.034	.100	.304
	N	16	16	16	16	16	16	16	16	16	16
RE scaled 0.67	Pearson Correlation	.731**	.774**	1	.987**	.990**	-.038	-.570*	-.424	-.652**	-.166
	Sig. (2-tailed)	.001	.000	.	.000	.000	.888	.021	.102	.006	.540
	N	16	16	16	16	16	16	16	16	16	16
RE scaled 0.75	Pearson Correlation	.609*	.867**	.987**	1	1.000**	-.051	-.590*	-.470	-.623**	-.202
	Sig. (2-tailed)	.012	.000	.000	.	.000	.850	.016	.066	.010	.454
	N	16	16	16	16	16	16	16	16	16	16
RE scaled (Darveau/empirical)	Pearson Correlation	.626**	.857**	.990**	1.000**	1	-.050	-.589*	-.465	-.628**	-.197
	Sig. (2-tailed)	.010	.000	.000	.000	.	.855	.016	.070	.009	.464
	N	16	16	16	16	16	16	16	16	16	16
REMAX	Pearson Correlation	.028	-.083	-.038	-.051	-.050	1	.504*	.170	.189	.134
	Sig. (2-tailed)	.918	.761	.888	.850	.855	.	.046	.529	.484	.622
	N	16	16	16	16	16	16	16	16	16	16
TAUON	Pearson Correlation	-.295	-.551*	-.570*	-.590*	-.589*	.504*	1	.704**	.586*	.434
	Sig. (2-tailed)	.267	.027	.021	.016	.016	.046	.	.002	.017	.093
	N	16	16	16	16	16	16	16	16	16	16
MRTON	Pearson Correlation	-.095	-.532*	-.424	-.470	-.465	.170	.704**	1	.413	.454
	Sig. (2-tailed)	.726	.034	.102	.066	.070	.529	.002	.	.112	.077
	N	16	16	16	16	16	16	16	16	16	16
TAUOFF	Pearson Correlation	-.556*	-.426	-.652**	-.623**	-.628**	.189	.586*	.413	1	.395
	Sig. (2-tailed)	.025	.100	.006	.010	.009	.484	.017	.112	.	.130
	N	16	16	16	16	16	16	16	16	16	16
MRTOFF	Pearson Correlation	.045	-.274	-.166	-.202	-.197	.134	.434	.454	.395	1
	Sig. (2-tailed)	.869	.304	.540	.454	.464	.622	.093	.077	.130	.
	N	16	16	16	16	16	16	16	16	16	16

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.13 Correlations matrix between adjusted RE values and $\dot{V}O_2$ kinetic parameters in combined, MD and LD runners

Correlations

		TAUON	MRTON	TAUOFF	MRTOFF	RE_ADJUS
TAUON	Pearson Correlation	1	.770**	.626**	.634**	-.048
	Sig. (2-tailed)	.	.000	.000	.000	.797
	N	32	32	32	32	31
MRTON	Pearson Correlation	.770**	1	.642**	.644**	-.205
	Sig. (2-tailed)	.000	.	.000	.000	.270
	N	32	32	32	32	31
TAUOFF	Pearson Correlation	.626**	.642**	1	.749**	-.154
	Sig. (2-tailed)	.000	.000	.	.000	.408
	N	32	32	32	32	31
MRTOFF	Pearson Correlation	.634**	.644**	.749**	1	-.029
	Sig. (2-tailed)	.000	.000	.000	.	.875
	N	32	32	32	32	31
RE_ADJUS	Pearson Correlation	-.048	-.205	-.154	-.029	1
	Sig. (2-tailed)	.797	.270	.408	.875	.
	N	31	31	31	31	31

**. Correlation is significant at the 0.01 level (2-tailed).

9.14 Correlations matrix between adjusted RE values and $\dot{V}O_2$ kinetic parameters in MD runners

Correlations

		TAUON	MRTON	TAUOFF	MRTOFF	RE_ADJUS
TAUON	Pearson Correlation	1	.798**	.468	.559*	.190
	Sig. (2-tailed)	.	.000	.068	.024	.498
	N	16	16	16	16	15
MRTON	Pearson Correlation	.798**	1	.633**	.677**	-.032
	Sig. (2-tailed)	.000	.	.008	.004	.909
	N	16	16	16	16	15
TAUOFF	Pearson Correlation	.468	.633**	1	.823**	.129
	Sig. (2-tailed)	.068	.008	.	.000	.647
	N	16	16	16	16	15
MRTOFF	Pearson Correlation	.559*	.677**	.823**	1	-.050
	Sig. (2-tailed)	.024	.004	.000	.	.860
	N	16	16	16	16	15
RE_ADJUS	Pearson Correlation	.190	-.032	.129	-.050	1
	Sig. (2-tailed)	.498	.909	.647	.860	.
	N	15	15	15	15	15

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.15 Correlations matrix between adjusted RE values and $\dot{V}O_2$ kinetic parameters in LD runners

Correlations

		TAUON	MRTON	TAUOFF	MRTOFF	RE_ADJUS
TAUON	Pearson Correlation	1	.704**	.683**	.434	-.738**
	Sig. (2-tailed)	.	.002	.004	.093	.001
	N	16	16	16	16	16
MRTON	Pearson Correlation	.704**	1	.533*	.454	-.506*
	Sig. (2-tailed)	.002	.	.033	.077	.046
	N	16	16	16	16	16
TAUOFF	Pearson Correlation	.683**	.533*	1	.456	-.644**
	Sig. (2-tailed)	.004	.033	.	.076	.007
	N	16	16	16	16	16
MRTOFF	Pearson Correlation	.434	.454	.456	1	-.148
	Sig. (2-tailed)	.093	.077	.076	.	.584
	N	16	16	16	16	16
RE_ADJUS	Pearson Correlation	-.738**	-.506*	-.644**	-.148	1
	Sig. (2-tailed)	.001	.046	.007	.584	.
	N	16	16	16	16	16

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

9.16 Relationships between adjusted RE and τ_{on} and τ_{off} in MD and LD runners

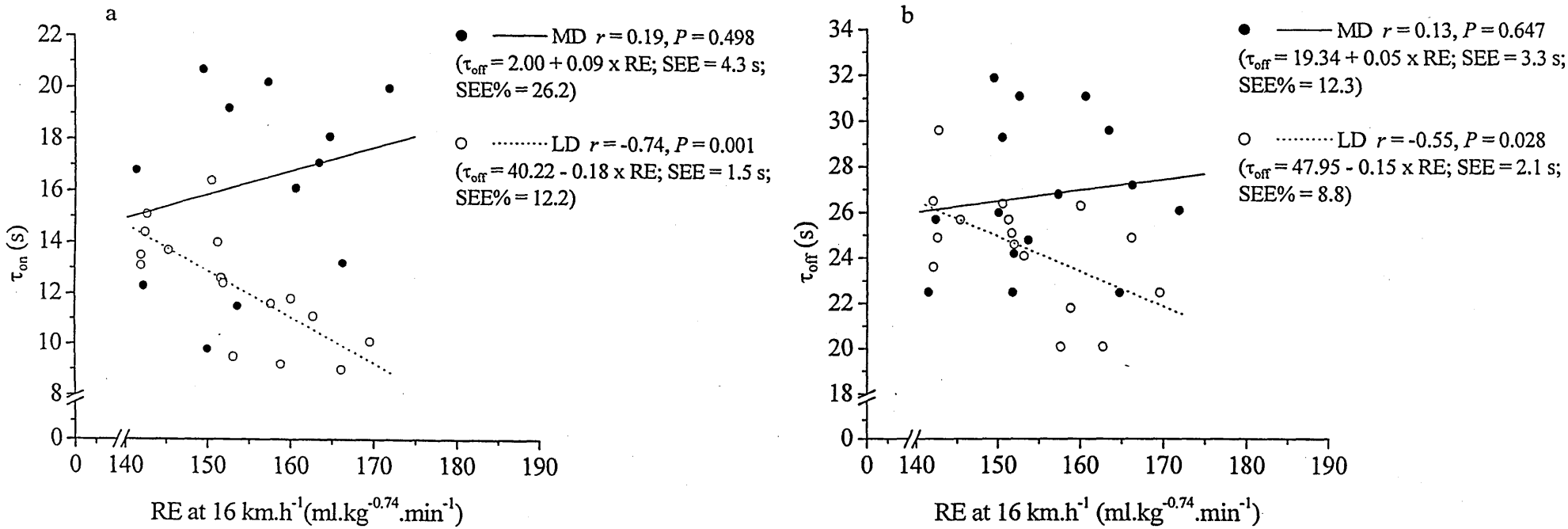


Figure A4 Relationship between RE (ml·kg$^{-0.74}$·min^{-1}) and (a) τ_{on} and (b) τ_{off} in MD (n=16) and LD (n=16) runners.

Appendix 10 Chapter 7: Raw data and statistical analyses

10.1 Raw data

Filename	Distance	Age (yr)	Stature (cm)	BM (kg)	VO_{2max} (ml.min)	VO_{2max} (ml.kg.min)	V_T (l.min)	V_T (ml.kg.min)	V_T $\%VO_{2max}$	V_T speed	RE16 (l.min)	RE16 (ml.kg.min)	RE $\%VO_{2max}$	80%V_T	80%V_T speed	S_o	S_o/A%
bait	LD	20	185.9	68.1	4500	66.1	3400	49.9	75.6	15.0	3689	54.2	82.0	2720	12.0	0.104	7.3
bolm	LD	18	180.4	67.3	4715	70.1	3800	56.5	80.6	16.2	3741	55.6	79.3	3040	12.9	0.072	3.3
brod	MD	19	177.2	69.6	3856	55.4	3100	44.5	80.4	15.1	3372	48.4	87.4	2480	12.1	0.115	5.7
davc	MD	22	168.0	62.1	3931	63.3	3100	49.9	78.9	13.0	3705	59.7	94.3	2480	9.9	0.081	6.2
davg	MD	22	168.1	61.5	4086	66.4	3400	55.3	83.2	13.6	3782	61.5	92.6	2720	10.9	0.075	4.3
dunr	LD	31	167.6	67.8	3959	58.4	3150	46.5	79.6	13.4	3801	56.1	96.0	2520	10.7	0.088	5.7
dysa	MD	19	176.7	65.8	3856	58.6	3250	49.4	84.3	15.4	3360	51.1	87.1	2600	12.3	0.075	4.4
ells	MD	19	190.0	79.3	4452	56.1	3500	44.1	78.6	13.6	3936	49.6	88.4	2800	11.0	0.129	6.9
evaa	MD	21	184.7	74.2	4089	55.1	3300	44.5	80.7	12.8	3842	51.8	94.0	2640	10.2	0.104	6.1
giss	LD	33	193.4	84.0	4625	55.1	3960	47.1	85.6	15.8	4063	48.4	87.8	3168	12.0	0.107	5.1
jams	LD	30	172.0	61.3	3452	56.3	2850	46.5	82.6	14.0	3242	52.9	93.9	2280	10.7	0.112	7.8
keig	MD	19	182.8	71.2	4116	57.8	3350	47.1	81.4	14.5	3702	52.0	89.9	2680	11.6	0.112	6.5
kila	LD	26	183.0	65.2	3563	54.6	3060	46.9	85.9	15.0	3220	49.4	90.4	2448	12.0	0.093	5.1
kilm	LD	26	183.0	64.2	3754	58.5	3000	46.7	79.9	15.0	3168	49.3	84.4	2400	12.0	0.158	8.8
leem	LD	24	184.0	69.5	4564	65.7	3900	56.1	85.5	16.1	3910	56.3	85.7	3120	12.9	0.102	5.0
macr	MD	28	192.7	75.8	4690	61.9	3700	48.8	78.9	13.2	4261	56.2	90.9	2960	10.6	0.110	5.5
manc	LD	23	187.0	75.5	4107	54.4	3300	43.7	80.4	13.8	3590	47.5	87.4	2640	11.0	0.163	9.1
mara	MD	40	175.1	71.5	4447	62.2	3600	50.3	81.0	14.2	3983	55.7	89.6	2880	11.3	0.082	4.2
meas	LD	29	173.8	84.4	4308	51.0	3750	44.4	87.0	13.7	4280	50.7	99.4	3000	11.0	0.082	4.3
metp	MD	21	179.0	67.1	4032	60.1	3150	46.9	78.1	13.4	3695	55.1	91.6	2520	10.8	0.101	4.3
newj	LD	32	178.5	73.0	4820	66.0	3800	52.1	78.8	14.6	4189	57.4	86.9	3040	11.7	0.110	5.6
nold	LD	22	186.1	84.3	5793	68.7	4150	49.2	71.6	15.2	4330	51.4	74.7	3320	12.1	0.132	5.9
nora	LD	20	173.3	59.7	3753	62.9	3350	56.1	89.3	17.3	3126	52.4	83.3	2680	12.0	0.082	4.7
nore	MD	23	175.6	59.1	3734	63.2	3183	53.9	85.2	16.7	3069	51.9	82.2	2546	12.0	0.099	6.0
otor	MD	19	162.2	56.8	3409	60.0	2900	51.1	85.1	14.1	3205	56.4	94.0	2320	11.3	0.149	10.2
prya	MD	18	176.0	70.6	3461	49.0	3000	42.5	86.7	13.0	3420	48.4	98.8	2400	10.4	0.150	9.6
ragd	MD	18	177.0	66.6	3900	58.6	2850	42.8	73.1	13.4	3308	49.7	84.8	2280	11.0	0.152	8.8
rees	MD	26	182.0	64.3	4385	68.2	3750	58.3	85.5	16.9	3620	56.3	82.6	3000	13.5	0.141	6.9
tayc	MD	18	181.0	62.7	3937	62.8	3300	52.6	83.8	14.1	3547	56.6	90.1	2640	11.3	0.114	6.6
thaa	LD	25	183.3	76.0	4372	57.5	3600	47.4	82.3	15.4	3727	49.0	85.2	2880	12.3	0.071	3.4
warj	LD	23	181.0	64.0	3622	56.6	3000	46.9	82.8	15.6	3098	48.4	85.5	2400	12.5	0.101	7.3
warr	LD	26	171.0	60.8	4071	67.0	3400	55.9	83.5	16.3	3343	55.0	82.1	2720	13.0	0.070	3.3
wara	MD	20	177.4	64.8	3877	59.8	3300	50.9	85.1	15.0	3438	53.1	88.7	2640	12.0	0.142	7.8
warm	LD	23	179.3	61.5	4266	69.4	3500	56.9	82.0	16.9	3279	53.3	76.9	2800	13.5	0.126	6.5
wilp	MD	18	175.7	66.1	3946	59.7	3300	49.9	83.6	13.8	3658	55.3	92.7	2640	11.1	0.096	5.1
Wris	LD	24	180.0	65.6	3935	60.0	3300	50.3	83.9	15.7	3357	51.2	85.3	2640	12.5	0.106	6.2
Mean		**23.5**	**179.0**	**68.4**	**4122**	**60.5**	**3370**	**49.5**	**82.0**	**14.7**	**3613**	**53.0**	**87.9**	**2696**	**11.7**	**0.108**	**6.1**
SD		**5.1**	**7.0**	**7.2**	**471**	**5.2**	**327**	**4.4**	**3.8**	**1.3**	**355**	**3.5**	**5.7**	**261**	**0.9**	**0.027**	**1.8**
CV%		**21.9**	**3.9**	**10.5**	**11.4**	**8.6**	**9.7**	**8.9**	**4.6**	**8.5**	**9.8**	**6.6**	**6.4**	**9.7**	**7.7**	**24.5**	**29.0**

Rest VO_2	$VO_{2(b)}$	$VO_{2(m)}$	A_{on}	Gain	TD_{on}	Tau_{on}	MRT_{on}	$VO2_{(m)off}$	$VO2_{(b)off}$	A_{off}	TD_{off}	Tau_{off}	MRT_{off}	$HR_{(b)}$	$HR_{(m)}$	Pre [HLa]	Post [HLa]	Delta [HLa]	Km Week	5k Time min:sec	5k Time sec	5k speed m.s	5kspeed /km	$\%VO_{2max}$
379	841	2269	1427	139	19.1	9.2	28.2	2233	838	1395	9.6	21.8	31.4	57	112	0.91	0.87	-0.04	88.5	15:45.2	945	5.3	03:09.0	96.4
399	877	3043	2167	183	14.5	9.0	23.4	3051	816	2234	5.9	24.9	30.8	76	146	0.90	0.98	0.08	77.2	15:42.0	942	5.3	03:08.4	93.2
366	896	2921	2025	182	11.5	16.8	28.3	2945	950	1995	11.1	22.5	33.5	64	139	1.04	0.92	-0.12	40.2	17:00.0	1020	4.9	03:24.0	92.0
378	698	2008	1309	159	13.0	18.1	31.1	1978	687	1291	7.4	22.5	29.8	78	131	1.08	1.13	0.05	40.2	17:53.0	1073	4.7	03:34.6	97.0
459	938	2680	1742	199	6.5	20.0	26.5	2765	928	1837	9.3	26.1	35.4	79	137	1.05	0.98	-0.07	40.2	18:06.0	1086	4.6	03:37.2	95.1
414	842	2402	1560	164	15.1	11.6	26.7	2387	841	1547	11.1	20.1	31.1	56	122	1.00	0.93	-0.06	84.5	18:35.0	1115	4.5	03:43.0	98.4
423	884	2591	1707	161	14.1	12.6	26.8	2586	880	1707	11.1	22.5	33.6	96	151	0.98	0.91	-0.07	56.3	17:02.0	1022	4.9	03:24.4	92.8
413	962	2831	1869	166	14.0	20.7	34.7	2843	972	1871	10.0	31.9	41.9	81	137	0.98	1.06	0.08	32.2	17:41.0	1061	4.7	03:32.2	92.8
328	1025	2740	1715	191	9.8	24.7	34.4	2760	1005	1755	10.9	29.3	40.2	79	132	1.05	0.92	-0.13	37.0	18:03.0	1083	4.6	03:36.6	97.1
451	1036	3137	2100	160	15.6	9.5	25.1	3107	1049	2059	9.1	24.1	33.3	79	133	0.98	1.00	0.03	64.4	16:53.0	1013	4.9	03:22.6	95.0
325	740	2173	1433	169	13.1	13.7	26.8	2181	744	1436	8.7	25.7	34.4	77	144	0.86	0.92	0.07	40.2	17:57.0	1077	4.6	03:35.4	96.6
419	938	2668	1730	163	14.8	12.5	27.3	2662	950	1713	10.8	24.2	35.0	84	137	1.18	1.08	-0.10	48.3	17:29.1	1049	4.8	03:29.8	94.7
367	824	2664	1839	176	15.1	13.1	28.3	2662	834	1828	1.7	26.5	28.2	65	136	1.01	0.93	-0.08	64.4	16:38.0	998	5.0	03:19.6	97.5
350	823	2618	1795	177	14.1	14.4	28.6	2608	817	1790	4.8	24.9	29.8	67	135	0.85	0.66	-0.19	72.4	16:11.2	971	5.1	03:14.2	94.3
340	1022	3042	2021	181	15.5	10.1	25.6	3045	1031	2015	10.3	22.5	32.8	71	150	0.95	1.01	0.06	80.5	16:12.8	973	5.1	03:14.6	95.0
509	1065	3060	1995	190	12.8	22.6	35.4	3074	1059	2014	7.9	29.2	37.1	94	143	1.13	0.96	-0.17	32.2	17:08.0	1028	4.9	03:25.6	97.9
448	955	2744	1789	166	15.0	13.5	28.5	2751	919	1832	10.9	23.6	34.5	84	144	1.14	1.03	-0.11	51.5	17:13.7	1034	4.8	03:26.7	91.8
392	901	2838	1937	182	13.8	17.1	30.9	2860	904	1956	6.5	29.6	36.1	82	140	1.85	1.85	0.01	40.2	18:20.9	1101	4.5	03:40.2	92.7
421	1025	2930	1905	162	14.2	16.4	30.6	2948	1038	1910	9.4	26.4	35.8	81	133	1.10	1.28	0.18	40.2	19:39.0	1179	4.2	03:55.8	96.1
391	879	2298	1419	158	9.5	15.0	24.5	2299	874	1425	13.7	24.2	37.9	79	137	0.91	1.10	0.19	56.3	17:08.5	1029	4.9	03:25.7	98.3
474	985	2946	1962	174	16.3	11.0	27.3	2938	966	1972	7.1	23.0	30.1	68	126	1.03	0.47	-0.56	64.4	16:34.0	994	5.0	03:18.8	97.0
439	1086	3320	2234	170	17.4	11.1	28.6	3316	1102	2214	12.6	20.1	32.7	65	121	0.80	0.88	0.08	64.4	16:32.2	992	5.0	03:18.4	85.2
348	875	2604	1729	189	11.9	12.6	24.4	2594	876	1717	8.7	25.1	33.8	68	138	1.25	1.33	0.08	80.5	16:08.0	968	5.2	03:13.6	94.1
379	887	2550	1663	184	14.7	9.8	24.5	2530	869	1661	5.2	26.0	31.2	77	137	1.07	1.24	0.17	88.5	16:04.0	964	5.2	03:12.8	93.4
394	849	2313	1464	179	11.4	19.2	30.7	2322	848	1473	8.4	31.1	39.5	108	151	1.02	0.93	-0.08	32.2	17:37.0	1057	4.7	03:31.4	98.3
416	927	2492	1565	170	14.0	17.5	31.5	2499	932	1566	12.7	28.8	41.6	86	148	0.95	0.89	-0.06	40.2	20:58.0	1258	4.0	04:11.6	93.7
394	845	2572	1726	178	16.2	12.3	28.5	2576	946	850	7.7	25.7	33.4	100	145	0.85	0.78	-0.07	24.1	18:11.0	1091	4.6	03:38.2	85.8
431	969	3024	2055	179	14.1	13.2	27.3	3011	961	2050	7.9	27.2	35.1	80	149	1.00	0.93	-0.08	48.3	16:03.2	963	5.2	03:12.6	93.2
363	893	2611	1718	190	15.4	16.1	31.5	2627	898	1729	7.7	31.1	38.9	104	166	1.10	1.07	-0.03	32.2	18:33.7	1114	4.5	03:42.7	92.2
394	953	3043	2090	170	16.1	14.0	30.1	3053	976	2077	9.5	25.7	35.1	77	131	1.20	1.08	-0.12	64.4	16:34.0	994	5.0	03:18.8	95.6
361	816	2191	1374	137	18.1	15.1	33.2	2199	798	1401	7.7	29.6	37.3	78	143	0.76	0.85	0.09	56.3	16:58.0	1018	4.9	03:23.6	94.7
370	861	3001	2140	200	16.2	11.8	28.0	2985	841	2145	7.5	26.3	33.8	79	144	0.81	0.69	-0.12	72.4	15:36.5	937	5.3	03:07.3	96.0
360	920	2749	1830	184	15.0	11.5	26.5	2753	932	1820	10.6	24.8	35.4	67	150	0.97	0.95	-0.02	64.4	16:54.0	1014	4.9	03:22.8	96.8
398	844	2781	1936	186	13.2	11.6	24.8	2769	838	1931	6.9	23.3	30.2	85	146	0.98	0.68	-0.30	96.5	15:20.0	920	5.4	03:04.0	96.6
411	913	2789	1877	195	10.9	20.2	31.2	2808	933	1875	8.8	26.8	35.6	72	125	1.15	0.86	-0.29	32.2	17:37.0	1057	4.7	03:31.4	97.3
417	895	2598	1703	160	15.9	12.4	28.3	2590	901	1689	10.4	24.6	35.0	69	132	0.94	0.91	-0.03	64.4	16:23.0	984	5.1	03:16.6	98.0
398	**908**	**2701**	**1793**	**174**	**14.1**	**14.5**	**28.6**	**2703**	**910**	**1772**	**8.9**	**25.6**	**34.5**	**78**	**139**	**1.02**	**0.97**	**-0.05**	**55.9**	**00:17:11**	**1031**	**4.9**	**00:03:26**	**94.8**
41	**85**	**303**	**241**	**15**	**2.5**	**3.9**	**3.0**	**306**	**90**	**289**	**2.4**	**3.0**	**3.4**	**12**	**10**	**0.18**	**0.23**	**0.15**	**19.5**	**00:01:10**	**70**	**0.3**	**00:00:14**	**3.0**
10.3	**9.4**	**11.2**	**13.5**	**8.5**	**17.5**	**27.2**	**10.6**	**11.3**	**9.8**	**16.3**	**27.0**	**11.7**	**9.7**	**15.2**	**7.5**	**17.8**	**23.1**	**-297.5**	**34.9**	**6.8**	**6.8**	**6.5**	**6.8**	**3.2**

10.2 Tests of normality for age, physical and physiological characteristics of MD and LD runners

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
AGE	.148	36	.045	.886	36	.001
STATURE	.066	36	.200*	.989	36	.969
B.MASS	.126	36	.159	.935	36	.037
TIME_MIN	.094	36	.200*	.957	36	.171
M_S_5K	.120	36	.200*	.975	36	.575
MAXSUST	.117	36	.200*	.860	36	.000

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
VO2MXABS	.144	36	.058	.919	36	.011
VO2MXRAT	.111	36	.200*	.976	36	.595
VO2MX.67	.148	36	.044	.965	36	.315
VO2MX.75	.134	36	.100	.971	36	.445
Darveau	.120	36	.200*	.973	36	.498
VO2MX.69	.135	36	.097	.967	36	.350
VT_ABS	.130	36	.133	.965	36	.302
VT_RATIO	.127	36	.149	.948	36	.090
VT_.67	.094	36	.200*	.973	36	.516
Also Darveau	.105	36	.200*	.969	36	.387
VT_.54	.075	36	.200*	.976	36	.626
VT_MAX	.078	36	.200*	.959	36	.207
RE_16ABS	.112	36	.200*	.956	36	.163
RE_16RAT	.106	36	.200*	.954	36	.137
RE_16.67	.078	36	.200*	.967	36	.358
Also Darveau	.091	36	.200*	.967	36	.352
RE_16.79	.095	36	.200*	.967	36	.352
RE_MAX	.064	36	.200*	.989	36	.977
TAU_ON	.131	36	.125	.937	36	.042
MRT_ON	.161	36	.019	.959	36	.193
TAU_OFF	.104	36	.200*	.967	36	.353
MRT_OFF	.097	36	.200*	.971	36	.461

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

10.3 ANCOVA summary for (a) MD and LD runners and (b) high and low performers

(a)*

Variances				
	df_B	df_E	*f*	*P*
VO_{2max}	16	16	1.07	0.447
V_T	16	16	1.17	0.379
RE16	16	16	1.45	0.233
τ_{on}	16	16	1.32	0.293
τ_{off}	16	16	2.17	0.066
MRT_{on}	16	16	1.59	0.182
MRT_{off}	16	16	1.76	0.134

Slopes				
	df_B	df_E	*f*	*P*
VO_{2max}	1	32	0.29	0.594
V_T	1	32	2.03	0.164
RE16	1	32	0.28	0.600
τ_{on}	1	32	2.53	0.122
τ_{off}	1	32	0.99	0.327
MRT_{on}	1	32	0.32	0.576
MRT_{off}	1	32	0.11	0.742

Elevations				
	df_B	df_E	*f*	*P*
VO_{2max}	1	33	7.91	0.008
V_T	1	33	7.47	0.010
RE16	1	33	7.22	0.011
τ_{on}	1	33	0.61	0.440
τ_{off}	1	33	0.45	0.507
MRT_{on}	1	33	3.03	0.091
MRT_{off}	1	33	0.89	0.352

(b)*

Variances				
	df_B	df_E	*f*	*P*
VO_{2max}	8	8	4.44	0.025
V_T	8	8	4.17	0.030
RE16	8	8	3.18	0.061
τ_{on}	8	8	5.08	0.017
τ_{off}	8	8	4.04	0.032
MRT_{on}	8	8	4.60	0.022
MRT_{off}	8	8	3.32	0.055

Slopes				
	df_B	df_E	*f*	*P*
VO_{2max}	1	16	0.53	0.477
V_T	1	16	0.36	0.557
RE16	1	16	0.36	0.557
τ_{on}	1	16	0.48	0.498
τ_{off}	1	16	0.09	0.768
MRT_{on}	1	16	0.00	1.000
MRT_{off}	1	16	0.26	0.617

Elevations				
	df_B	df_E	*f*	*P*
VO_{2max}	1	17	76.42	0.000
V_T	1	17	46.13	0.000
RE16	1	17	134.06	0.000
τ_{on}	1	17	47.22	0.000
τ_{off}	1	17	78.62	0.000
MRT_{on}	1	17	49.05	0.000
MRT_{off}	1	17	70.76	0.000

*It is acknowledged that a small number of measures did not meet the assumption of normality and homogeneity of variance for ANCOVA. However, because the majority of data did meet the assumptions, any advantages of single log-transformations were considered minimal and/or inappropriate. Furthermore, it is probable that ANCOVA would be robust to minimal violation of normality/homogeneity, as observed for this data, which consequently would have minimal effect on the present results and interpretation.

10.4 Descriptive statistics for high and low performers

Descriptive Statistics

	N	Minimum	Maximum	Mean		Std.
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
AGE	20	18.00	40.00	23.9500	1.2279	5.49138
STATURE	20	167.60	185.90	176.8900	1.2878	5.75910
B.MASS	20	59.10	84.40	66.1400	1.3456	6.01790
VO2MXRAT	20	49.00	70.10	61.7100	1.3053	5.83762
VT_RATIO	20	42.50	58.30	50.7950	1.1593	5.18444
RE_16RAT	20	48.40	61.50	53.9300	.7627	3.41099
TAU_ON	20	9.00	24.70	14.0800	.8955	4.00494
MRT_ON	20	23.40	34.40	28.0800	.6470	2.89366
TAU_OFF	20	20.10	31.10	25.5950	.6193	2.76947
MRT_OFF	20	29.80	41.60	34.0300	.7557	3.37968
KM_WK	20	24.14	96.54	59.4123	5.1514	23.03778
TIME_MIN	20	15:20:09	20:58:00	17:16:59	0:20:44	1:32:46
M_S_5K	20	4.00	5.40	4.8500	.0925	.41359
MAXSUST	20	85.80	98.40	94.7450	.6215	2.77934
Valid N (listwise)	20					

10.5 Independent t-tests between high and low performers

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
AGE	Equal variances assumed	9.902	.006	-.765	18	.454	-1.9000	2.48305	-7.11670	3.31670
	Equal variances not assumed			-.765	11.625	.459	-1.9000	2.48305	-7.32951	3.52951
STATURE	Equal variances assumed	.276	.606	2.174	18	.043	5.1200	2.35491	.17251	10.06749
	Equal variances not assumed			2.174	17.484	.044	5.1200	2.35491	.16202	10.07798
B.MASS	Equal variances assumed	2.852	.108	-1.654	18	.116	-4.2600	2.57628	-9.67256	1.15256
	Equal variances not assumed			-1.654	13.242	.122	-4.2600	2.57628	-9.81541	1.29541
VO2MXRAT	Equal variances assumed	1.036	.322	3.162	18	.005	6.8000	2.15065	2.28164	11.31836
	Equal variances not assumed			3.162	16.074	.006	6.8000	2.15065	2.24252	11.35748
VT_RATIO	Equal variances assumed	.319	.579	3.592	18	.002	6.5300	1.81807	2.71037	10.34963
	Equal variances not assumed			3.592	17.699	.002	6.5300	1.81807	2.70571	10.35429
RE_16RAT	Equal variances assumed	5.020	.038	-.488	18	.631	-.7600	1.55697	-4.03107	2.51107
	Equal variances not assumed			-.488	13.868	.633	-.7600	1.55697	-4.10235	2.58235
TAU_ON	Equal variances assumed	2.071	.167	-3.978	18	.001	-5.3400	1.34236	-8.16020	-2.51980
	Equal variances not assumed			-3.978	12.847	.002	-5.3400	1.34236	-8.24352	-2.43648
MRT_ON	Equal variances assumed	.861	.366	-3.420	18	.003	-3.5400	1.03513	-5.71472	-1.36528
	Equal variances not assumed			-3.420	16.704	.003	-3.5400	1.03513	-5.72688	-1.35312
TAU_OFF	Equal variances assumed	2.961	.102	-1.567	18	.135	-1.8700	1.19371	-4.37789	.63789
	Equal variances not assumed			-1.567	13.289	.141	-1.8700	1.19371	-4.44316	.70316
MRT_OFF	Equal variances assumed	2.217	.154	-2.436	18	.025	-3.2800	1.34672	-6.10936	-.45064
	Equal variances not assumed			-2.436	13.534	.029	-3.2800	1.34672	-6.17780	-.38220
KM_WK	Equal variances assumed	.094	.762	5.275	18	.000	34.9958	6.63370	21.05887	48.93263
	Equal variances not assumed			5.275	17.640	.000	34.9958	6.63370	21.03844	48.95306
TIME_MIN	Equal variances assumed	3.473	.079	-8.309	18	.000	-2:41:04	0:19:23	-3:21:48	-2:00:21
	Equal variances not assumed			-8.309	11.031	.000	-2:41:04	0:19:23	-3:23:44	-1:58:25
M_S_5K	Equal variances assumed	1.978	.177	9.811	18	.000	.7400	.07542	.58154	.89846
	Equal variances not assumed			9.811	12.944	.000	.7400	.07542	.57698	.90302
MAXSUST	Equal variances assumed	2.900	.106	.433	18	.670	.5500	1.27042	-2.11905	3.21905
	Equal variances not assumed			.433	12.564	.672	.5500	1.27042	-2.20429	3.30429

10.6 Correlations between $\dot{V}O_{2\,max}$ and running performance in high performers

Correlations

		VO2MXABS	VO2MXRAT	VO2MX.67	VO2MX.75	Darveau	VO2MX.69	KM_WK	M_S_5K
VO2MXABS	Pearson Correlation	1	.767**	.919**	.891**	.843**	.913**	-.003	.359
	Sig. (2-tailed)	.	.010	.000	.001	.002	.000	.993	.308
	N	10	10	10	10	10	10	10	10
VO2MXRAT	Pearson Correlation	.767**	1	.958**	.975**	.992**	.962**	.138	.763*
	Sig. (2-tailed)	.010	.	.000	.000	.000	.000	.704	.010
	N	10	10	10	10	10	10	10	10
VO2MX.67	Pearson Correlation	.919**	.958**	1	.998**	.987**	1.000**	.080	.630
	Sig. (2-tailed)	.000	.000	.	.000	.000	.000	.826	.051
	N	10	10	10	10	10	10	10	10
VO2MX.75	Pearson Correlation	.891**	.975**	.998**	1	.995**	.999**	.093	.665*
	Sig. (2-tailed)	.001	.000	.000	.	.000	.000	.797	.036
	N	10	10	10	10	10	10	10	10
Darveau	Pearson Correlation	.843**	.992**	.987**	.995**	1	.989**	.112	.711*
	Sig. (2-tailed)	.002	.000	.000	.000	.	.000	.757	.021
	N	10	10	10	10	10	10	10	10
VO2MX.69	Pearson Correlation	.913**	.962**	1.000**	.999**	.989**	1	.083	.639*
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.819	.047
	N	10	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	-.003	.138	.080	.093	.112	.083	1	.471
	Sig. (2-tailed)	.993	.704	.826	.797	.757	.819	.	.169
	N	10	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	.359	.763*	.630	.665*	.711*	.639*	.471	1
	Sig. (2-tailed)	.308	.010	.051	.036	.021	.047	.169	.
	N	10	10	10	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

10.7 Correlations between $\dot{V}O_{2\,max}$ and running performance in low performers

Correlations

		VO2MXABS	VO2MXRAT	VO2MX.67	VO2MX.75	Darveau	VO2MX.69	KM_WK	M_S_5K
VO2MXABS	Pearson Correlation	1	.337	.614	.540	.445	.595	.016	.175
	Sig. (2-tailed)	.	.341	.059	.107	.197	.069	.964	.628
	N	10	10	10	10	10	10	10	10
VO2MXRAT	Pearson Correlation	.337	1	.950**	.974**	.993**	.957**	-.033	.756*
	Sig. (2-tailed)	.341	.	.000	.000	.000	.000	.927	.011
	N	10	10	10	10	10	10	10	10
VO2MX.67	Pearson Correlation	.614	.950**	1	.996**	.980**	1.000**	-.019	.691*
	Sig. (2-tailed)	.059	.000	.	.000	.000	.000	.958	.027
	N	10	10	10	10	10	10	10	10
VO2MX.75	Pearson Correlation	.540	.974**	.996**	1	.994**	.998**	-.023	.717*
	Sig. (2-tailed)	.107	.000	.000	.	.000	.000	.949	.020
	N	10	10	10	10	10	10	10	10
Darveau	Pearson Correlation	.445	.993**	.980**	.994**	1	.984**	-.028	.740*
	Sig. (2-tailed)	.197	.000	.000	.000	.	.000	.939	.014
	N	10	10	10	10	10	10	10	10
VO2MX.69	Pearson Correlation	.595	.957**	1.000**	.998**	.984**	1	-.020	.698*
	Sig. (2-tailed)	.069	.000	.000	.000	.000	.	.956	.025
	N	10	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	.016	-.033	-.019	-.023	-.028	-.020	1	-.052
	Sig. (2-tailed)	.964	.927	.958	.949	.939	.956	.	.887
	N	10	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	.175	.756*	.691*	.717*	.740*	.698*	-.052	1
	Sig. (2-tailed)	.628	.011	.027	.020	.014	.025	.887	.
	N	10	10	10	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

10.8 Correlations between $\dot{V}O_{2\,max}$ and running performance in combined runners

Correlations

		VO2MXABS	VO2MXPFR	VO2MX.67	VO2MX.75	Darveau	VO2MX.69	M_S_5K
VO2MXABS	Pearson Correlation	1	.490**	.790**	.725**	.626**	.774**	.263
	Sig. (2-tailed)	.	.002	.000	.000	.000	.000	.122
	N	36	36	36	36	36	36	36
VO2MXPFR	Pearson Correlation	.490**	1	.922**	.956**	.987**	.931**	.659**
	Sig. (2-tailed)	.002	.	.000	.000	.000	.000	.000
	N	36	36	36	36	36	36	36
VO2MX.67	Pearson Correlation	.790**	.922**	1	.995**	.973**	1.000**	.580**
	Sig. (2-tailed)	.000	.000	.	.000	.000	.000	.000
	N	36	36	36	36	36	36	36
VO2MX.75	Pearson Correlation	.725**	.956**	.995**	1	.991**	.997**	.609**
	Sig. (2-tailed)	.000	.000	.000	.	.000	.000	.000
	N	36	36	36	36	36	36	36
Darveau	Pearson Correlation	.626**	.987**	.973**	.991**	1	.978**	.638**
	Sig. (2-tailed)	.000	.000	.000	.000	.	.000	.000
	N	36	36	36	36	36	36	36
VO2MX.69	Pearson Correlation	.774**	.931**	1.000**	.997**	.978**	1	.588**
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.	.000
	N	36	36	36	36	36	36	36
M_S_5K	Pearson Correlation	.263	.659**	.580**	.609**	.638**	.588**	1
	Sig. (2-tailed)	.122	.000	.000	.000	.000	.000	.
	N	36	36	36	36	36	36	36

**. Correlation is significant at the 0.01 level (2-tailed).

10.9 Correlations between V_T and running performance in high performers

Correlations

		VT_ABS	VT_RATIO	VT_.67	Also Darveau	VT_.54	KM_WK	M_S_5K
VT_ABS	Pearson Correlation	1	.723*	.883**	.850**	.928**	-.165	.183
	Sig. (2-tailed)	.	.018	.001	.002	.000	.649	.613
	N	10	10	10	10	10	10	10
VT_RATIO	Pearson Correlation	.723*	1	.962**	.978**	.928**	-.061	.422
	Sig. (2-tailed)	.018	.	.000	.000	.000	.868	.224
	N	10	10	10	10	10	10	10
VT_.67	Pearson Correlation	.883**	.962**	1	.998**	.994**	-.109	.359
	Sig. (2-tailed)	.001	.000	.	.000	.000	.764	.308
	N	10	10	10	10	10	10	10
Also Darveau	Pearson Correlation	.850**	.978**	.998**	1	.985**	-.099	.377
	Sig. (2-tailed)	.002	.000	.000	.	.000	.786	.283
	N	10	10	10	10	10	10	10
VT_.54	Pearson Correlation	.928**	.928**	.994**	.985**	1	-.125	.327
	Sig. (2-tailed)	.000	.000	.000	.000	.	.731	.357
	N	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	-.165	-.061	-.109	-.099	-.125	1	.471
	Sig. (2-tailed)	.649	.868	.764	.786	.731	.	.169
	N	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	.183	.422	.359	.377	.327	.471	1
	Sig. (2-tailed)	.613	.224	.308	.283	.357	.169	.
	N	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

10.10 Correlations between V_T and running performance in low performers

Correlations

		VT_ABS	VT_RATIO	VT_.67	Also Darveau	VT_.54	KM_WK	M_S_5K
VT_ABS	Pearson Correlation	1	.341	.659*	.579	.781**	.042	-.205
	Sig. (2-tailed)	.	.335	.038	.079	.008	.909	.570
	N	10	10	10	10	10	10	10
VT_RATIO	Pearson Correlation	.341	1	.932**	.964**	.853**	-.001	.457
	Sig. (2-tailed)	.335	.	.000	.000	.002	.998	.184
	N	10	10	10	10	10	10	10
VT_.67	Pearson Correlation	.659*	.932**	1	.995**	.984**	.016	.287
	Sig. (2-tailed)	.038	.000	.	.000	.000	.965	.422
	N	10	10	10	10	10	10	10
Also Darveau	Pearson Correlation	.579	.964**	.995**	1	.961**	.011	.338
	Sig. (2-tailed)	.079	.000	.000	.	.000	.976	.339
	N	10	10	10	10	10	10	10
VT_.54	Pearson Correlation	.781**	.853**	.984**	.961**	1	.024	.190
	Sig. (2-tailed)	.008	.002	.000	.000	.	.947	.599
	N	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	.042	-.001	.016	.011	.024	1	-.052
	Sig. (2-tailed)	.909	.998	.965	.976	.947	.	.887
	N	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	-.205	.457	.287	.338	.190	-.052	1
	Sig. (2-tailed)	.570	.184	.422	.339	.599	.887	.
	N	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

10.11 Correlations between V_T and running performance in combined runners

Correlations

		VT_ABS	VT_PFR	VT_.67	Also Darveau	VT_.54	VT_MAX	M_S_5K
VT_ABS	Pearson Correlation	1	.379*	.697**	.621**	.811**	-.068	.275
	Sig. (2-tailed)	.	.023	.000	.000	.000	.693	.104
	N	36	36	36	36	36	36	36
VT_PFR	Pearson Correlation	.379*	1	.927**	.961**	.849**	.301	.620**
	Sig. (2-tailed)	.023	.	.000	.000	.000	.074	.000
	N	36	36	36	36	36	36	36
VT_.67	Pearson Correlation	.697**	.927**	1	.995**	.985**	.203	.593**
	Sig. (2-tailed)	.000	.000	.	.000	.000	.236	.000
	N	36	36	36	36	36	36	36
Also Darveau	Pearson Correlation	.621**	.961**	.995**	1	.962**	.232	.609**
	Sig. (2-tailed)	.000	.000	.000	.	.000	.173	.000
	N	36	36	36	36	36	36	36
VT_.54	Pearson Correlation	.811**	.849**	.985**	.962**	1	.148	.551**
	Sig. (2-tailed)	.000	.000	.000	.000	.	.389	.000
	N	36	36	36	36	36	36	36
VT_MAX	Pearson Correlation	-.068	.301	.203	.232	.148	1	-.060
	Sig. (2-tailed)	.693	.074	.236	.173	.389	.	.729
	N	36	36	36	36	36	36	36
M_S_5K	Pearson Correlation	.275	.620**	.593**	.609**	.551**	-.060	1
	Sig. (2-tailed)	.104	.000	.000	.000	.000	.729	.
	N	36	36	36	36	36	36	36

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

10.12 Correlations between RE and running performance in high performers

Correlations

		RE_16ABS	RE_16RAT	RE_16.67	Also Darveau	RE_16.79	KM_WK	M_S_5K
RE_16ABS	Pearson Correlation	1	.782**	.941**	.916**	.901**	-.178	.013
	Sig. (2-tailed)	.	.008	.000	.000	.000	.623	.971
	N	10	10	10	10	10	10	10
RE_16RAT	Pearson Correlation	.782**	1	.947**	.966**	.975**	-.161	.344
	Sig. (2-tailed)	.008	.	.000	.000	.000	.656	.331
	N	10	10	10	10	10	10	10
RE_16.67	Pearson Correlation	.941**	.947**	1	.998**	.995**	-.183	.195
	Sig. (2-tailed)	.000	.000	.	.000	.000	.613	.589
	N	10	10	10	10	10	10	10
Also Darveau	Pearson Correlation	.916**	.966**	.998**	1	.999**	-.180	.228
	Sig. (2-tailed)	.000	.000	.000	.	.000	.618	.526
	N	10	10	10	10	10	10	10
RE_16.79	Pearson Correlation	.901**	.975**	.995**	.999**	1	-.179	.246
	Sig. (2-tailed)	.000	.000	.000	.000	.	.622	.493
	N	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	-.178	-.161	-.183	-.180	-.179	1	.471
	Sig. (2-tailed)	.623	.656	.613	.618	.622	.	.169
	N	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	.013	.344	.195	.228	.246	.471	1
	Sig. (2-tailed)	.971	.331	.589	.526	.493	.169	.
	N	10	10	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

10.13 Correlations between RE and running performance in low performers

Correlations

		RE_16ABS	RE_16RAT	RE_16.67	Also Darveau	RE_16.79	RE_MAX	KM_WK	M_S_5K
RE_16ABS	Pearson Correlation	1	.201	.589	.488	.438	.382	.257	-.158
	Sig. (2-tailed)	.	.578	.073	.152	.205	.276	.474	.664
	N	10	10	10	10	10	10	10	10
RE_16RAT	Pearson Correlation	.201	1	.910**	.953**	.968**	-.148	.232	.595
	Sig. (2-tailed)	.578	.	.000	.000	.000	.684	.520	.070
	N	10	10	10	10	10	10	10	10
RE_16.67	Pearson Correlation	.589	.910**	1	.993**	.984**	.037	.305	.422
	Sig. (2-tailed)	.073	.000	.	.000	.000	.919	.392	.224
	N	10	10	10	10	10	10	10	10
Also Darveau	Pearson Correlation	.488	.953**	.993**	1	.998**	-.015	.290	.479
	Sig. (2-tailed)	.152	.000	.000	.	.000	.966	.417	.161
	N	10	10	10	10	10	10	10	10
RE_16.79	Pearson Correlation	.438	.968**	.984**	.998**	1	-.040	.281	.504
	Sig. (2-tailed)	.205	.000	.000	.000	.	.913	.432	.138
	N	10	10	10	10	10	10	10	10
RE_MAX	Pearson Correlation	.382	-.148	.037	-.015	-.040	1	.445	-.608
	Sig. (2-tailed)	.276	.684	.919	.966	.913	.	.197	.062
	N	10	10	10	10	10	10	10	10
KM_WK	Pearson Correlation	.257	.232	.305	.290	.281	.445	1	-.052
	Sig. (2-tailed)	.474	.520	.392	.417	.432	.197	.	.887
	N	10	10	10	10	10	10	10	10
M_S_5K	Pearson Correlation	-.158	.595	.422	.479	.504	-.608	-.052	1
	Sig. (2-tailed)	.664	.070	.224	.161	.138	.062	.887	.
	N	10	10	10	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

10.14 Correlations between RE and running performance in combined runners

Correlations

		RE_16ABS	RE_16RAT	RE_16.67	Also Darveau	RE_16.79	RE_MAX	M_S_5K
RE_16ABS	Pearson Correlation	1	.262	.710**	.610**	.556**	.142	-.244
	Sig. (2-tailed)	.	.123	.000	.000	.000	.408	.151
	N	36	36	36	36	36	36	36
RE_16RAT	Pearson Correlation	.262	1	.865**	.924**	.948**	.124	.044
	Sig. (2-tailed)	.123	.	.000	.000	.000	.473	.801
	N	36	36	36	36	36	36	36
RE_16.67	Pearson Correlation	.710**	.865**	1	.991**	.980**	.166	-.096
	Sig. (2-tailed)	.000	.000	.	.000	.000	.333	.578
	N	36	36	36	36	36	36	36
Also Darveau	Pearson Correlation	.610**	.924**	.991**	1	.998**	.160	-.062
	Sig. (2-tailed)	.000	.000	.000	.	.000	.352	.721
	N	36	36	36	36	36	36	36
RE_16.79	Pearson Correlation	.556**	.948**	.980**	.998**	1	.155	-.044
	Sig. (2-tailed)	.000	.000	.000	.000	.	.366	.798
	N	36	36	36	36	36	36	36
RE_MAX	Pearson Correlation	.142	.124	.166	.160	.155	1	-.840**
	Sig. (2-tailed)	.408	.473	.333	.352	.366	.	.000
	N	36	36	36	36	36	36	36
M_S_5K	Pearson Correlation	-.244	.044	-.096	-.062	-.044	-.840**	1
	Sig. (2-tailed)	.151	.801	.578	.721	.798	.000	.
	N	36	36	36	36	36	36	36

**. Correlation is significant at the 0.01 level (2-tailed).

10.15 Correlations between $\dot{V}O_2$ kinetics and running performance in high performers

Correlations

		TAU_ON	MRT_ON	TAU_OFF	MRT_OFF	KM_WK	M_S_5K
TAU_ON	Pearson Correlation	1	.478	.451	.277	-.537	-.386
	Sig. (2-tailed)	.	.162	.191	.438	.109	.271
	N	10	10	10	10	10	10
MRT_ON	Pearson Correlation	.478	1	-.002	.275	-.453	-.324
	Sig. (2-tailed)	.162	.	.995	.442	.188	.361
	N	10	10	10	10	10	10
TAU_OFF	Pearson Correlation	.451	-.002	1	.399	-.623	-.109
	Sig. (2-tailed)	.191	.995	.	.253	.054	.764
	N	10	10	10	10	10	10
MRT_OFF	Pearson Correlation	.277	.275	.399	1	-.689*	-.328
	Sig. (2-tailed)	.438	.442	.253	.	.027	.356
	N	10	10	10	10	10	10
KM_WK	Pearson Correlation	-.537	-.453	-.623	-.689*	1	.471
	Sig. (2-tailed)	.109	.188	.054	.027	.	.169
	N	10	10	10	10	10	10
M_S_5K	Pearson Correlation	-.386	-.324	-.109	-.328	.471	1
	Sig. (2-tailed)	.271	.361	.764	.356	.169	.
	N	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

10.16 Correlations between $\dot{V}O_2$ kinetics and running performance in low performers

Correlations

		TAU_ON	MRT_ON	TAU_OFF	MRT_OFF	KM_WK	M_S_5K
TAU_ON	Pearson Correlation	1	.650*	.467	.515	-.323	.050
	Sig. (2-tailed)	.	.042	.173	.128	.363	.891
	N	10	10	10	10	10	10
MRT_ON	Pearson Correlation	.650*	1	.596	.575	-.390	-.221
	Sig. (2-tailed)	.042	.	.069	.082	.266	.539
	N	10	10	10	10	10	10
TAU_OFF	Pearson Correlation	.467	.596	1	.856**	-.658*	-.281
	Sig. (2-tailed)	.173	.069	.	.002	.038	.431
	N	10	10	10	10	10	10
MRT_OFF	Pearson Correlation	.515	.575	.856**	1	-.382	-.562
	Sig. (2-tailed)	.128	.082	.002	.	.275	.091
	N	10	10	10	10	10	10
KM_WK	Pearson Correlation	-.323	-.390	-.658*	-.382	1	-.052
	Sig. (2-tailed)	.363	.266	.038	.275	.	.887
	N	10	10	10	10	10	10
M_S_5K	Pearson Correlation	.050	-.221	-.281	-.562	-.052	1
	Sig. (2-tailed)	.891	.539	.431	.091	.887	.
	N	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

10.17 Correlations between $\dot{V}O_2$ kinetics and running performance in combined runners

Correlations

		GAIN	TAU_ON	MRT_ON	TAU_OFF	MRT_OFF	M_S_5K
GAIN	Pearson Correlation	1	.275	-.049	.261	.046	.075
	Sig. (2-tailed)	.	.105	.775	.125	.790	.663
	N	36	36	36	36	36	36
TAU_ON	Pearson Correlation	.275	1	.777**	.629**	.647**	-.537**
	Sig. (2-tailed)	.105	.	.000	.000	.000	.001
	N	36	36	36	36	36	36
MRT_ON	Pearson Correlation	-.049	.777**	1	.631**	.590**	-.497**
	Sig. (2-tailed)	.775	.000	.	.000	.000	.002
	N	36	36	36	36	36	36
TAU_OFF	Pearson Correlation	.261	.629**	.631**	1	.721**	-.362*
	Sig. (2-tailed)	.125	.000	.000	.	.000	.030
	N	36	36	36	36	36	36
MRT_OFF	Pearson Correlation	.046	.647**	.590**	.721**	1	-.555**
	Sig. (2-tailed)	.790	.000	.000	.000	.	.000
	N	36	36	36	36	36	36
M_S_5K	Pearson Correlation	.075	-.537**	-.497**	-.362*	-.555**	1
	Sig. (2-tailed)	.663	.001	.002	.030	.000	.
	N	36	36	36	36	36	36

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

10.18 Multiple regression output for predicting 5 km performance in high performers

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	VO2MXRAT	.	Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).
2	RE_16RAT	.	Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).

a. Dependent Variable: M_S_5K

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.763[a]	.582	.530	.07080
2	.929[b]	.862	.823	.04347

a. Predictors: (Constant), VO2MXRAT

b. Predictors: (Constant), VO2MXRAT, RE_16RAT

ANOVA[c]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.056	1	.056	11.149	.010[a]
	Residual	.040	8	.005		
	Total	.096	9			
2	Regression	.083	2	.041	21.904	.001[b]
	Residual	.013	7	.002		
	Total	.096	9			

a. Predictors: (Constant), VO2MXRAT

b. Predictors: (Constant), VO2MXRAT, RE_16RAT

c. Dependent Variable: M_S_5K

Excluded Variables[c]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics: Tolerance
1	VT_RATIO	-.394[a]	-1.126	.297	-.391	.413
	RE_16RAT	-.958[a]	-3.772	.007	-.819	.305
	TAU_ON	.002[a]	.006	.995	.002	.742
	MRT_ON	.024[a]	.087	.933	.033	.798
	TAU_OFF	-.076[a]	-.312	.764	-.117	.998
	MRT_OFF	-.283[a]	-1.286	.239	-.437	.996
2	VT_RATIO	-.164[b]	-.691	.515	-.272	.376
	TAU_ON	-.068[b]	-.389	.711	-.157	.733
	MRT_ON	.186[b]	1.175	.285	.432	.747
	TAU_OFF	-.073[b]	-.489	.642	-.196	.998
	MRT_OFF	.091[b]	.469	.656	.188	.594

a. Predictors in the Model: (Constant), VO2MXRAT

b. Predictors in the Model: (Constant), VO2MXRAT, RE_16RAT

c. Dependent Variable: M_S_5K

10.19 Multiple regression output for predicting 5 km performance in low performers

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	VO2MXRAT		Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).

a. Dependent Variable: M_S_5K

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.756[a]	.572	.518	.14926

a. Predictors: (Constant), VO2MXRAT

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.238	1	.238	10.672	.011[a]
	Residual	.178	8	.022		
	Total	.416	9			

a. Predictors: (Constant), VO2MXRAT

b. Dependent Variable: M_S_5K

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.781	.522		5.327	.001
	VO2MXRAT	2.913E-02	.009	.756	3.267	.011

a. Dependent Variable: M_S_5K

Excluded Variables[b]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics: Tolerance
1	VT_RATIO	-.801[a]	-2.030	.082	-.609	.248
	RE_16RAT	-.329[a]	-.642	.541	-.236	.221
	TAU_ON	.027[a]	.108	.917	.041	.999
	MRT_ON	.006[a]	.022	.983	.008	.910
	TAU_OFF	-.197[a]	-.830	.434	-.299	.987
	MRT_OFF	-.281[a]	-1.099	.308	-.384	.802

a. Predictors in the Model: (Constant), VO2MXRAT

b. Dependent Variable: M_S_5K

10.20 Multiple regression output for predicting 5 km performance in combined runners

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	VO2MXRAT	.	Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).
2	RE_16	.	Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).
3	MRT_OFF	.	Stepwise (Criteria: Probability-of-F-to-enter <= .050, Probability-of-F-to-remove >= .100).

a. Dependent Variable: M_S_5K

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.659[a]	.435	.418	.25258
2	.845[b]	.714	.697	.18237
3	.865[c]	.748	.725	.17371

a. Predictors: (Constant), VO2MXRAT

b. Predictors: (Constant), VO2MXRAT, RE_16

c. Predictors: (Constant), VO2MXRAT, RE_16, MRT_OFF

ANOVA[d]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	1.667	1	1.667	26.135	.000[a]
	Residual	2.169	34	.064		
	Total	3.836	35			
2	Regression	2.739	2	1.369	41.175	.000[b]
	Residual	1.098	33	.033		
	Total	3.836	35			
3	Regression	2.871	3	.957	31.714	.000[c]
	Residual	.966	32	.030		
	Total	3.836	35			

a. Predictors: (Constant), VO2MXRAT

b. Predictors: (Constant), VO2MXRAT, RE_16

c. Predictors: (Constant), VO2MXRAT, RE_16, MRT_OFF

d. Dependent Variable: M_S_5K

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.349	.495		4.747	.000
	VO2MXRAT	4.169E-02	.008	.659	5.112	.000
2	(Constant)	4.453	.515		8.650	.000
	VO2MXRAT	6.274E-02	.007	.992	9.016	.000
	RE_16	-1.58E-02	.003	-.625	-5.676	.000
3	(Constant)	5.291	.633		8.355	.000
	VO2MXRAT	5.541E-02	.007	.876	7.389	.000
	RE_16	-1.43E-02	.003	-.566	-5.214	.000
	MRT_OFF	-2.07E-02	.010	-.210	-2.091	.045

a. Dependent Variable: M_S_5K

Excluded Variables[d]

Model		Beta In	t	Sig.	Partial Correlation	Collinearity Statistics
						Tolerance
1	VT_RATIO	.210[a]	.848	.403	.146	.272
	TAU_ON	-.351[a]	-2.800	.008	-.438	.881
	MRT_ON	-.280[a]	-2.092	.044	-.342	.843
	TAU_OFF	-.202[a]	-1.537	.134	-.258	.929
	MRT_OFF	-.345[a]	-2.661	.012	-.420	.837
	RE_16	-.625[a]	-5.676	.000	-.703	.716
2	VT_RATIO	.100[b]	.552	.585	.097	.269
	TAU_ON	-.107[b]	-.945	.352	-.165	.681
	MRT_ON	-.102[b]	-.952	.348	-.166	.752
	TAU_OFF	-.159[b]	-1.684	.102	-.285	.924
	MRT_OFF	-.210[b]	-2.091	.045	-.347	.781
3	VT_RATIO	.139[c]	.802	.429	.143	.266
	TAU_ON	.024[c]	.187	.853	.034	.474
	MRT_ON	-.003[c]	-.024	.981	-.004	.587
	TAU_OFF	-.046[c]	-.352	.727	-.063	.469

a. Predictors in the Model: (Constant), VO2MXRAT

b. Predictors in the Model: (Constant), VO2MXRAT, RE_16

c. Predictors in the Model: (Constant), VO2MXRAT, RE_16, MRT_OFF

d. Dependent Variable: M_S_5K