Effect of Hypoxia on the Oxygen Uptake Response to an Exhaustive Severe Intensity Run

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It has been shown that highly aerobically trained individuals are unable to achieve maximal oxygen uptake ($\dot{V}O_2$ max) during exhaustive running lasting ~2 min despite sufficient time for the response (Draper and Wood, 2005). However, hypoxia offers the opportunity to study the $\dot{V}O_2$ response to an exhaustive run relative to a reduced $\dot{V}O_2$ max. The purpose of the current study was to explore whether there is a difference in the percentage of $\dot{V}O_2$ max attained (during a 2 minute exhaustive run) in normoxia and hypoxia. Fourteen trained middle-distance runners (mean ± SD; age 21.4 ± 3.4 y, height 1.76 ± 0.05 m, mass 66.0 ± 7.0 kg, $\dot{V}O_2$ max 67.0 ± 5.2 $ml.kg^{-1}.min^{-1}$) volunteered for this study. Participants completed exhaustive treadmill ramp tests and square-wave tests (lasting 2 minutes), in normoxia and hypoxia (FiO_2 0.13). Oxygen uptake was determined on a breath-by-breath basis throughout each test. The $\dot{V}O_2$ data (excluding the first 15s) from the square-wave tests were modelled using a mono-exponential function. Repeated measures ANOVA (condition x test) was used to investigate the differences in $\dot{V}O_2$ peak and post-hoc related samples t-tests for each condition were performed to explore a significant interaction. There was a significant interaction effect for $\dot{V}O_2$ peak ($P < 0.001$). Post hoc tests revealed that the $\dot{V}O_2$ peak achieved during the square-wave test was lower than the ramp test in normoxia ($P < 0.001$) but not in hypoxia ($P = 0.49$). The mean ± SD percentage of the ramp $\dot{V}O_2$ peak achieved was; 86 ±0.06 vs. 102 ± 0.08%, for normoxia and hypoxia respectively. The phase II time constant was different between conditions ($P = 0.029$) demonstrating a slower oxygen uptake response to exercise in hypoxia (mean ± SD; 12.7 ± 2.8 vs. 10.4 ± 2.6 seconds, for hypoxia and normoxia respectively). The findings of the current study support the findings of Draper and Wood (2005) that suitably trained individuals do not achieve maximal oxygen uptake in running of this intensity. However, the present study has demonstrated that when maximal oxygen uptake is reduced through hypoxia it may then be achieved.

Declaration

I declare that the work in this thesis was carried out in accordance with the regulations of the University of Gloucestershire and is original except where indicated by specific reference in the text. No part of the thesis has been submitted as part of any other academic award. The thesis has not been presented to any other education institution in the United Kingdom or overseas. Any views expressed in the thesis are those of the author and in no way represent those of the University.

M. Block

Signed Date 13.03.12

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CHAPTER 1
INTRODUCTION

Sustained human performance is dependent on the supply and utilisation of oxygen (O_2) to the working muscle to enable the aerobic breakdown of the body's fuel sources. In the 1920s, Hill and Lupton (1923) recognised that a given activity had a given energy demand, and that the rate of metabolism at the muscle was reflected in the rate volume per unit ($\dot{V}O_2$) measured at the mouth. They proposed that there was an upper limit to $\dot{V}O_2$ that an individual can take in and utilise ($\dot{V}O_2$ max), and therefore concluded that human performance was determined by the $\dot{V}O_2$ max of an individual, and their capacity to work independent of oxygen (anaerobic metabolism) (Hill and Lupton, 1923). Although completed nearly a century ago, the work conducted by Hill and colleagues is integral to our understanding of performance, and form the foundations of current performance models with $\dot{V}O_2$ max being arguably the most commonly measured physiological parameter.

Hill and Lupton (1923) found that at the onset of exercise the O_2 uptake response ($\dot{V}O_2$ kinetics) followed an exponential curve which was driven to the $\dot{V}O_2$ required for a given activity. However, the response to increased workload is immediate, demanding an instantaneous increase in metabolism. The exponential nature of the $\dot{V}O_2$ kinetics created a shortfall in the $\dot{V}O_2$ supply and demand relationship, requiring the assistance of anaerobic metabolism to allow energy demand to be met.

The $\dot{V}O_2$ kinetics during the transition from rest to exercise follows a distinct profile composed of three phases (Barstow, 1994; Whipp, 1994), and has been shown to be dependent of several factors relating to both the supply and utilisation of O_2. At an intensity equal to or below the lactate threshold (LT), an intensity at which lactate production and removal is equal, the $\dot{V}O_2$ kinetics are similar to that described by Hill and Lupton (1923). Following an initial delay phase (largely representative of muscle to lung transit time) $\dot{V}O_2$ tends exponentially towards the $\dot{V}O_2$ required, reaching this asymptote then maintaining a steady state reflective of the energy utilisation of the working muscle (Hughson, 2009). However at intensities above the LT the third phase of the $\dot{V}O_2$ kinetics become distorted (Whipp, 1994). An additional rise in $\dot{V}O_2$ becomes manifest past the third minute of exercise (Burnley

and Jones, 2007) elevating the $\dot{V}O_2$ to either a greater steady state level than predicted from the extrapolation of the $\dot{V}O_2$-work rate (WR) relationship from speeds below the LT (Whipp and Ward, 1990), or elevating $\dot{V}O_2$ to rise until $\dot{V}O_2$ max or exhaustion (Xu and Rhodes, 1999; Gaesser and Poole, 1996; Whipp, 1994; Whipp and Ward, 1990), dependent on the exercise intensity. The delayed component has led to researchers referring to this phenomenon as the slow component of $\dot{V}O_2$ (Burnley and Jones, 2007).

The 800-m middle distance track running event requires an energy contribution in excess of that which can be provided solely by the aerobic pathway. Requiring a combined contribution from the energy systems of ~36 and ~64%, via the anaerobic and aerobic pathways respectively, to meet an overall energy demand that is ~110-120% of $\dot{V}O_2$ max (Duffield *et al.* 2005; Spencer and Gastin, 2001; Hill, 1999; Craig and Morgan, 1998). It is argued, that during exercise above the $\dot{V}O_2$ max, $\dot{V}O_2$ will tend exponentially towards the $\dot{V}O_2$ required, until $\dot{V}O_2$ max or exhaustion is achieved (Hill and Ferguson, 1999; Whipp, 1994). However, this assumption has recently been disputed. The assumptions were based on cycling exercise (Poole and Richardson, 1997), and were formed on participants of a lower aerobic fitness than that typically observed in individuals who regularly perform at an intensity above their $\dot{V}O_2$ max i.e. middle distance runners (Hill and Ferguson, 1999). Researchers (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; James *et al.* 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a) using more appropriate samples of highly trained middle-distance runners ($\dot{V}O_2$ max $\geq$ 60ml.kg^{-1}.min^{-1}) and using treadmill exercise have found that $\dot{V}O_2$ max is not achieved despite sufficient time for the response. Instead, it has been found that aerobically trained individuals completing exhaustive exercise of ~2 min duration achieve a steady-state value ~88% $\dot{V}O_2$ max. Furthermore, it has been demonstrated that individuals with a greater $\dot{V}O_2$ max achieved a lower percentage of that maximal value when performing a constant speed exhaustive treadmill test lasting ~2 min (Draper *et al.* 2008; Draper and Wood, 2005).

It has been found that individuals with a larger $\dot{V}O_2$ max have faster $\dot{V}O_2$ kinetics to exercise onset than lesser aerobically trained counterparts (Marwood *et al.* 2010; Ingham *et al.* 2007; Kilding *et al.* 2006; Draper and Wood, 2005); moreover, that

aerobic training is related to an improvement in the speed of the $\dot{V}O_2$ kinetics (Berger *et al.* 2006). Draper and Wood (2005) found that the $\dot{V}O_2$ kinetics to steady-state exercise reached a plateau within ~60s, despite exercise lasting ~2mins. Therefore, the response had sufficient time to develop, yet the individual was unable to achieve $\dot{V}O_2$ max. These findings question what $\dot{V}O_2$ is driven towards during exercise, and what is regulating $\dot{V}O_2$.

Several authors have reported reduced aerobic capability on acute exposure to altitude (Woorons *et al.* 2005; Gore *et al.* 1997), with Gore *et al.* (1997) reporting that reduced performance capabilities were present at an altitude of 580-m. Therefore, artificially lowering the O_2 concentration (hypoxia), simulating altitude, lowers the $\dot{V}O_2$ max that an individual can achieve. Researchers investigating the effect of the fraction of inspired oxygen (FiO_2) have found anaerobic performance is unaffected (Ogura *et al.* 2006; Calbet *et al.* 2003; McLellan *et al.* 1990), moreover exhaustive exercise lasting up to 180s is largely unaffected (Friedmann *et al.* 2007; Ogawa *et al.* 2005; Weyand *et al.* 1999). These findings suggest that exhaustive exercise lasting ~120s (approximately the time taken for a trained middle-distance runner to complete 800-m) could be maintained during conditions of reduced FiO_2. However, it is unclear whether performance would be maintained via an increased contribution of the anaerobic capacity, or an increased contribution in the relative $\dot{V}O_2$ max attained.

Weyand *et al.* (1999) tried to determine the energy system contribution to exercise of different durations in hypoxia and normoxia. However, the protocol employed was matched for absolute WR not duration and may have resulted in different relative exercise intensity. The treadmill was set to an inclination of 4.6° altering the muscle fibre recruitment pattern and type (Sloniger *et al.* 1997) and reducing the speed at which the exhaustive tests could be run. The recruitment of different muscle fibre types/numbers may have resulted in an increased energy cost, impacting the $\dot{V}O_2$ kinetics. These limitations may have affected the results with the possibility of different findings had the times to exhaustion been matched and the treadmill had been level.

The effect of hypoxia on the $\dot{V}O_2$ kinetics specific to exhaustive exercise lasting ~2 min has not yet been investigated. It is unclear whether highly aerobically trained individuals possessing fast $\dot{V}O_2$ kinetics will reach $\dot{V}O_2$ max in conditions where $\dot{V}O_2$ max is artificially lowered. Manipulation of the $\dot{V}O_2$ max, matching the relative exercise intensity between conditions and performing the exercise on a level treadmill may increase understanding of the mechanism(s) responsible for the regulation of $\dot{V}O_2$ and performance of exhaustive exercise of ~2min. The aim of the present study was to determine whether there was a difference in the percentage of $\dot{V}O_2$ max achieved (during an exhaustive ~ 2 min run) in normoxia and hypoxia.

CHAPTER 2

LITERATURE REVIEW

2.1 Middle distance running

Middle distance running is a term used to encompass a variety of track-running distances ranging from 800-3000 m. The middle-distance track events lie between the shorter sprint activities that rely predominately on anaerobic energy provision and the long- distance events that rely on a predominant aerobic contribution. Energy demand during middle-distance events is in excess of the $\dot{V}O_2$ max (James *et al.* 2008) and beyond the provision of energy that can be provided by aerobic metabolism (Spencer and Gastin, 2001). The middle-distance events rely on a combined energy contribution from both the anaerobic and aerobic pathways (Duffield *et al.* 2005; Spencer and Gastin, 2001). The interaction of the anaerobic and aerobic energy pathways presents the performer with a unique physiological challenge.

The 800-m is the shortest of the middle-distance running events, requiring a combined contribution from the energy systems of ~36 and ~64%, via the anaerobic and aerobic pathways respectively, to meet an overall energy demand that is ~110-120% of $\dot{V}O_2$ max (Duffield *et al.* 2005; Spencer and Gastin, 2001; Hill, 1999, Craig and Morgan, 1998). Trained middle distance runners typically have large aerobic capabilities with a $\dot{V}O_2$ max in excess of 60ml.kg^{-1}.min^{-1} (James *et al.* 2007). The 800-m is characterised by several transitional phases in speed. The world record time for the 800-m, 1:41.01 was achieved by running a positive split (a faster first lap than the second) equivalent to the first lap being run at ~105% of the average race pace, whilst the final lap was run at ~95%. This finding may suggest that the distribution of energy throughout exercise may have a significant impact on performance. The $\dot{V}O_2$ kinetics response to different pacing strategies will be discussed in section 2.7.

2.2 Domains of exercise intensity and $\dot{V}O_2$ kinetics

The $\dot{V}O_2$ kinetics have been shown to be exercise intensity dependent (Whipp *et al.* 1981). Although the current research is interested in the $\dot{V}O_2$ kinetics to exhaustive exercise of ~2 min, it is important to discuss how the $\dot{V}O_2$ kinetics vary at different exercise intensities. Therefore, it is important to define the exercise intensity domains and the physiological parameters that define their boundaries. Within the literature, authors have used different terminology and physiological measures to define the boundaries of the exercise intensity domains. However, it is not in the scope of the current research to investigate the conceptual differences in all exercise intensity domains, and will instead explore the discrepancies that exist, between authors, concerning exhaustive exercise of ~2 min.

Within this thesis the term moderate intensity exercise will be used to describe all WRs that occur below the LT thus WRs that do not result in an increased (above resting levels) metabolic academia (Xu and Rhodes, 1999; Gaesser and Poole, 1996; Whipp, 1994). The term heavy intensity exercise will be used to describe all intensities that are above the LT, the upper limit of this intensity being termed the critical power (CP) the point at which a maximal lactate steady state (MLSS) and a steady state VO_2 can be achieved (Wilkerson *et al.* 2004; Gaesser and Poole, 1996). In this thesis WRs above the heavy intensity domain are termed severe (Billat *et al.* 1998; Gaesser and Poole, 1996). The $\dot{V}O_2$ kinetics in this domain are described later in this section; it is however characterised by an increasing blood lactate (BLa) and $\dot{V}O_2$ with time, until $\dot{V}O_2$ max is attained or exercise is terminated (Jones *et al.* 2008; Burnley and Jones, 2007; Wilkerson *et al.* 2004; Billat *et al.* 1998; Gaesser and Poole, 1996).

Whilst the term severe intensity exercise has been used to describe all WRs above the MLSS, this exercise intensity domain has been further categorised with several researchers terming WRs above the $\dot{V}O_2$ max as supra maximal (Adami *et al.* 2011; Simmonds *et al.* 2010; Mortensen *et al.* 2008). Although the duration and intensity of exercise in the current thesis will demand a WR in excess of $\dot{V}O_2$ max, the term supra maximal will not be used given that it is conceptually impossible for exercise intensity to be greater than maximal. Due to the conceptual limitations regarding the

term supra maximal, several researchers have used the term peri maximal to describe WRs in excess of $\dot{V}O_2$ max (Wilkerson *et al.* 2004; Jones *et al.* 2003). However, there appears to be little consistency in terms throughout the literature, with these researchers having also used different terminology to describe exercise of this intensity. Jones and Poole (2005) termed all WRs above the heavy intensity domain as supra CP, within which there are two exercise intensity domains; very heavy and severe intensity. Very heavy intensity exercise includes all WRs between CP and the individuals $\dot{V}O_2$ max, whilst severe intensity exercise is intensities that occur above the $\dot{V}O_2$ max. Despite the lack of convention in terminology, the current researcher will use the term severe intensity to describe all WRs above the CP. The $\dot{V}O_2$ kinetics at WRs within the severe intensity domain are largely unaltered at WRs above or below the $\dot{V}O_2$ max (there are subtle changes in the magnitude of response) (Xu and Rhodes, 1999; Gaesser and Poole, 1996; Whipp, 1994; Whipp and Ward, 1990), therefore there is little point in further categorising WRs within the severe intensity domain. However, it is worth noting that the $\dot{V}O_2$ kinetics within the severe intensity domain are exercise modality and training status dependent (see section 2.5).

Although the current researcher has chosen to use the term severe intensity, it is recognised that conceptually this term has several limitations. By definition, provided sufficient duration exhaustive exercise within the severe intensity domain will result in the attainment of $\dot{V}O_2$ max (Whipp, 1994). Therefore, during exhaustive exercise of ~2 min a $\dot{V}O_2$ steady state should not be achieved. However, it has been found that during exhaustive treadmill exercise of ~2 min a steady state $\dot{V}O_2$ below the individuals $\dot{V}O_2$ max is achieved (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a). These research findings conceptually challenge the definition of the severe intensity domain and raise questions regarding the regulation of $\dot{V}O_2$ kinetics. Therefore, it is the belief of the current researcher that short duration exhaustive exercise which follows a mono-exponential response, with no discernible increase in $\dot{V}O_2$ during phase III should be considered a different intensity of exercise. However, no distinction currently exists.

There are three distinct phases to the $\dot{V}O_2$ kinetics during the transition from rest to exercise (Barstow, 1994; Whipp, 1994). Phase I is often treated as a delay phase representative of the transit time of O_2 unloading at the muscle and the arrival of the same blood to the pulmonary capillary network (Faisal *et al.* 2009; Hughson, 2009; Delp and O'Leary, 2004. This delay time is typically ~15s (Draper and Wood, 2005; Grassi *et al.* 1996). Due to the delay, the blood returning to the pulmonary capillary network for gaseous exchange has not been subjected to the increased O_2 extraction at the working muscle. During this period the O_2 demand at the muscle is greater than the O_2 supply, resulting in the energy demand being met through an increased contribution from anaerobic energy metabolism, the shortage of O_2 to meet the entire energy demand at the onset of exercise will be referred to in this thesis as the oxygen deficit. Due to the reliance on anaerobic energy metabolism at this stage, the pulmonary $\dot{V}O_2$ response does not represent the O_2 consumption at the working muscle, confirmed by largely unchanged values for the partial pressure of venous O_2 (PO_2) and carbon dioxide (PCO_2), and the respiratory exchange ratio (RER) (Burnley and Jones, 2005). However, there is a slight increase in $\dot{V}O_2$ during this phase reflective of a sudden increase in venous return, hence an increased stroke volume (Faisal *et al.* 2009). An abrupt change in PO_2, PCO_2, RER and VO_2 signals the start of the next phase (Burnley and Jones, 2005).

Phase II is manifest after ~15s (Draper and Wood, 2005; Grassi *et al.* 1996), and reflects the progressive desaturation of the venous blood and increase in cardiac output (Q), following the transit delay of venous return to the pulmonary capillaries from the working muscle. Therefore, measurement of this phase via collection of expirate from the mouth, directly reflects the changes in metabolism at the working muscle (Hughson, 2009; Burnley and Jones, 2005; Lucia *et al.* 2002), resulting in this phase being termed the primary phase of $\dot{V}O_2$. During phase II, $\dot{V}O_2$ is driven exponentially towards an asymptote to achieve a steady state that is capable of meeting the energy demand (Hughson, 2009).

Phase III of the $\dot{V}O_2$ kinetics, at least in the moderate intensity domain, represents the attainment of a steady state $\dot{V}O_2$. Above the LT, phase I and II are fundamentally unchanged, with subtle differences in onset and duration. However, instead of $\dot{V}O_2$ achieving a steady state during phase II that is maintained throughout phase III, a

third delayed slow component to $\dot{V}O_2$ emerges 90-180s after the onset of exercise (Burnley and Jones, 2007; Billat *et al.* 1998; Gaesser and Poole, 1996). During exercise performed in the heavy intensity domain, the slow component elevates $\dot{V}O_2$ to a greater steady state $\dot{V}O_2$ than that predicted from the extrapolation of $\dot{V}O_2$-WR relationship from speeds below LT (Whipp and Ward, 1990). In the severe intensity, the $\dot{V}O_2$ response is similar to that described during heavy intensity exercise. The key difference being that $\dot{V}O_2$ during exercise in the severe intensity domain will not achieve a steady state, instead continuing to rise towards $\dot{V}O_2$ max or exhaustion. However, it is worth noting that the presence of the slow component has been disputed during exhaustive exercise within the severe intensity domain (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a).

The physiological mechanism(s) responsible for the $\dot{V}O_2$ slow component are poorly understood, with several central and peripheral mechanisms having been proposed. However, the emergence of the slow component when performing severe intensity treadmill running has been found to be negligible (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a; Draper *et al.* 2003; Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). Therefore, readers are referred to the review papers by Grassi (2001) and Xu and Rhodes (1999) for a discussion of the mechanisms proposed to be responsible for the emergence of the slow component.

2.3 Gas analysis systems

The composition of expired gas can be analysed by two different approaches; off-line or on-line systems. An off-line system requires the collection of gas before determination of composition and volume (which is performed on a different piece of equipment). The Douglas bag method used by Hill and colleagues is an example of an off-line system. Although the Douglas bag method is considered to be the criterion method in gas collection, there are several limitations. A major limitation is that the gas analysis can only be determined for the entire sample. Due to improved gas analysis technology, on-line methods whereby gas is analysed on a breath-by-breath basis have been produced, enabling a more detailed profile of the $\dot{V}O_2$

kinetics to be observed. Although this method allows subtle differences in $\dot{V}O_2$ to be observed, it too has several limitations. The data is more likely to suffer from greater variability and the data needs to be extrapolated to reduce noise in the data i.e. through the participant coughing. Further limitations between on-line systems were identified in a review by Macfarlane (2001). This review investigated the validity in comparing measures of $\dot{V}O_2$ between different on-line systems, highlighting differences in researchers perceived levels of acceptable precision and accuracy of measures of $\dot{V}O_2$, and the differences recorded in gas concentrations and volumes of the different measurement systems.

The majority of research regarding the $\dot{V}O_2$ kinetics to 800-m running is in agreement that $\dot{V}O_2$ max is not achieved in trained participants (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a). However, findings by Thomas *et al.* (2005), using an equally trained sample, are in conflict with the majority of the research. A key difference between the studies are that Thomas *et al.* (2005) used a portable telemetric device (Cosmed K4, Roma, Italy) worn by the participant during exercise for the determination of gas volume and concentrations. The majority of researchers used laboratory based quadruple mass spectrometers (MSX 671; Ferraris Respiratory Europe Ltd, Hertford, UK or QP9000; Morgan Medical, Rainham, UK) (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Draper and Wood, 2005; 2005a) to analyse the gas concentrations and a turbine volume transducer (Interface Associates, Alifovieja, US) to determine gas volumes.

The review of on-line systems within the laboratory versus on-line portable systems, specifically research papers investigating the differences between systems similar to the devices used in the conflicting $\dot{V}O_2$ kinetics research were further explored. Further investigation of research surrounding measurement systems would indicate whether the conclusions of Macfarlane (2001) were warranted, and whether these differences could account for the conflict amongst the research.

Pinnington *et al.* (2001) and Duffield *et al.* (2004) reported significant differences between two measurement systems similar to those used in the research concerned with the $\dot{V}O_2$ kinetics to 800-m running. Pinnington *et al.* (2001) found that the

Cosmed K4b^2 system reported significantly lower values in the fraction of expired O_2 (FeO_2) and the fraction of expired carbon dioxide ($FeCO_2$) ($P < 0.001$) compared to the measures made by a laboratory based on-line system. Similarly, Duffield *et al.* (2004) found that FeO_2 was underestimated and $FeCO_2$ was overestimated by the K4b^2 system. Both researchers concluded that although the measurement systems showed acceptable limits of agreement, the measures obtained by the Cosmed $K4b_2$ system could cause physiologically significant differences in $\dot{V}O_2$ and $\dot{V}CO_2$.

As suggested by Macfarlane (2001), the literature regarding the accuracy of measurement systems proved to be equivocal with research supporting (Duffield *et al.* 2004; Pinnington *et al.* 2001) and refuting (Doyon *et al.* 2001; McLaughlin *et al.* 2001) the claim that measurement systems could cause physiologically significant differences in $\dot{V}O_2$ and $\dot{V}CO_2$. The research papers reviewed compared the findings across a range of intensities and ventilation rates. Therefore, the different findings observed in the literature concerning $\dot{V}O_2$ kinetics to 800-m running could not be attributed to exercise intensity, but instead to the manufacturer of the on-line system. Based on the evidence, comparison of findings between measurement systems should be interpreted with caution. It is possible that the findings reported by Thomas *et al.* (2005) are in conflict with the majority of the literature due to differences in measurement systems; this conclusion is in agreement with Macfarlane (2001). However, differences in the methods employed and the environmental conditions of the testing may also account for the differences in findings, discussed further in section 2.7.

2.4 Modelling the breath-by-breath data

Researchers have developed methods to model the breath-by-breath data obtained by on-line automated measurement systems to describe the $\dot{V}O_2$ kinetics, allowing a detailed description of the $\dot{V}O_2$ kinetics to exercise. There are a number of mathematical models that can be used (Bell *et al.* 2001); mono-, 2- component, and 3- component exponential models. The 3- component exponential model is fit to all phase of the $\dot{V}O_2$ kinetics, the 2- component exponential model is fit to phases II and III, and the mono- exponential model is fit to phase II. However, disagreement exists within the literature regarding the efficacy of the modelling procedure for the $\dot{V}O_2$ data.

Bell *et al.* (2001) explored the efficacy of each model for describing the $\dot{V}O_2$ kinetics, concluding that during heavy-exercise a 3- component exponential model provided the best statistical fit. However, Bell *et al.* (2001) did concede that the use of a 3- component exponential model is based on assumptions regarding the $\dot{V}O_2$ kinetics. A key assumption to this model is that the phase I data describing the cardio-dynamic adjustment to exercise is exponential. Although several researchers (Bell *et al.* 2001; Yoshida *et al.* 1993) have found close agreement between Q and the $\dot{V}O_2$ kinetics during this phase, no physiological evidence exists to support the use of an exponential term. However, despite the lack of physiological evidence to support its use, exponential models are commonly applied to phase I data.

Lamarra *et al.* (1987) found that the level of noise in the measured $\dot{V}O_2$ response was unchanged by intensity. Therefore, the magnitude of the response is an important factor to consider in the efficacy of modelling a given parameter, such that the magnitude of the phase I data is small yet the level of noise is as great during severe intensity exercise. The SD/GAIN relationship, where SD is standard deviation of the noise, and GAIN is the magnitude of the response, provides a measure of the fit of the model to the $\dot{V}O_2$ data, such that a large SD/GAIN relationship would indicate a poor model fit. The small magnitude of the phase I response and the level of noise imposed on the response, would result in a much larger SD/GAIN relationship for phase I data compared to phase II. Lamarra *et al.* (1987) found that to achieve 95% confidence limits of ± 2s for the model fit τ (the time taken to achieve 63% of the response) of phase II during moderate intensity exercise, eight transitions were required. Therefore, given the larger SD/GAIN relationship during phase I, a very large number of transitions would be needed for confidence to be achieved.

As previously mentioned, during severe intensity exhaustive treadmill exercise, the emergence of the slow component is negligible (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; Draper *et al.* 2003; Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). The emergence of the slow component during exercise of this modality and intensity was explored by Draper and Wood (2004). They investigated the pattern of

the residuals to the phase III $\dot{V}O_2$ kinetics, finding that the breath-by-breath noise was uncorrelated and normally distributed throughout, supporting the absence of the slow component. These findings question the need to model the phase III data.

2.5 Factors shown to influence the $\dot{V}O_2$ kinetics and $\dot{V}O_2$ peak

2.5.1 The effect of exercise modality

The majority of research concerning $\dot{V}O_2$ kinetics has been conducted on a cycle ergometer (Burnley *et al.* 2002; Burnley *et al.* 2000; MacDonald *et al.* 1997; Gerbino *et al.* 1996). The findings of these researchers have generated ideas and models that have been applied to other exercise modalities including running. However, the mechanics of running differ to that of cycling, with the exercise modes utilising different types of muscular contraction. Running exercise involves a greater amount of eccentric activity allowing the storage of elastic energy enhancing the subsequent concentric force production for a given neural input, reducing the O_2 cost of exercise (Van Inghen Schenau *et al.* 1997).

Several researchers have examined the $\dot{V}O_2$ kinetics of cycling and running in the same group of participants (Draper *et al.* 2003; Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). Carter *et al.* (2000) found that there was no difference in the $\dot{V}O_2$ kinetics between modalities in recreationally active participants. Conversely, Billat *et al.* (1998) found that in well trained triathletes the phase II τ was faster for running than cycling (15.9 ± 2.2 vs. 22.6 ± 5.4, respectively), however, this difference did not reach statistical significance. The findings of Hill *et al.* (2003) add increased weight to the observation of Billat *et al.* (1998), that the phase II response is faster in running than cycling (14 ± 5 vs. 25 ± 4; $P < 0.01$). It is unclear why the findings differed in the $\dot{V}O_2$ kinetics between modalities, but it is likely due to the differences in the length and intensity of their exercise conditions.

The emergence of the slow component during heavy and severe intensity exercise has been shown to differ between exercise modality. In a study involving participants of an equally trained status for running and cycling (triathletes), Billat *et al.* (1998) demonstrated that the $\dot{V}O_2$ slow component during running was virtually non-existent compared to cycling (20.9 ± 2 vs. 268.8 ± 24ml.min^{-1}; $P = 0.02$). The

findings of Draper *et al.* (2003) support the findings of Billat *et al.* (1998), observing no difference in the final two collection points during two minutes of exhaustive square-wave exercise during running (4.33 ± 0.46 and 4.33 ± 0.45ml.min^{-1}; P = 0.983) compared to cycling (3.99 ± 0.63 and 4.17 ± 0.65ml.min^{-1}; P = 0.007). However, no differences were observed in the emergence of the slow component during a 5- or 8- minutes exhaustive square-wave exercise (P = 0.18 and P = 0.13, respectively). Hill *et al.* (2003) found that the $\dot{V}O_2$ slow component was present during severe intensity running, but confirmed that the amplitude of the slow component is larger in cycling (177 ± 92 vs. 299 ± 153ml.min^{-1}; P = 0.03). These findings may suggest that $\dot{V}O_2$ kinetics are dependent on activated muscle fibre type.

2.5.2 The effect of muscle fibre type

Skeletal muscle is composed of two predominant muscle fibre types; namely slow (type I) and fast twitch (type II) fibres. Type I fibres generate energy for the resynthesis of adenosine triphosphate (ATP), a high energy compound broken down by the body to supply energy, through the aerobic system of energy transfer. Conversely, type II fibres generate energy for the resynthesis of ATP via anaerobic energy pathways (McArdle *et al.* 2007). It has been shown that individuals with a larger composition of type I muscle fibres possess a larger O_2 delivery to O_2 consumption ratio, compared to individuals with a larger type II fibre distribution (Behnke *et al.* 2003). Furthermore, mechanical efficiency has been correlated with the percentage of type I fibres in endurance athletes (r = 0.75, P < 0.001) (Horowitz *et al.* 1994), demonstrating a greater potential for aerobic activity in athletes with a larger type I composition.

Barstow *et al.* (1996) and Pringle *et al.* (2003) have shown that during exercise above the LT, the gain of the phase II VO_2 kinetics is muscle fibre type dependent. Barstow *et al.* (1996) found that the less O_2 efficient fibres i.e. type II fibres, demonstrated a decreased GAIN in phase II (i.e. a smaller increase in VO_2 per increase in power output). Similarly to this, Pringle *et al.* (2003) found that the GAIN of phase II was significantly correlated with the percentage of type I muscle fibres i.e. for an equivalent increase in power, VO_2 increased more in participants with a greater percentage of type I fibres.

Muscle fibre type has also been shown to influence the magnitude of the phase III kinetics at WRs above the LT (Pringle *et al.* 2003; Barstow *et al.* 1996), with individuals with a larger proportion of type II fibres demonstrating a greater magnitude response. However, the magnitude of the slow component has been shown to be negligible during severe intensity treadmill running (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a; Carter *et al.* 2000; Billat *et al.* 1998). Therefore, further discussion of these findings was not warranted.

2.5.3 The effect of prior exercise

The start of exercise causes an increase in O_2 delivery to the working muscles to support the required increase in muscle O_2 uptake. The profile of the response depends on several factors i.e. exercise intensity and modality (and others outlined in this chapter). Several authors (Bucheit *et al.* 2009; Jones *et al.* 2008; Draper *et al.* 2006) have investigated the effect of prior exercise on the $\dot{V}O_2$ kinetics during running. Jones *et al.* (2008) and Draper *et al.* (2006) found that prior heavy intensity exercise did not alter the $\dot{V}O_2$ kinetics to subsequent heavy or severe intensity exercise, respectively. A possible reason for having found no difference in the $\dot{V}O_2$ kinetics following prior exercise is the already fast $\dot{V}O_2$ kinetics inherent to the exercise mode (Jones *et al.* 2008). The findings of Bucheit *et al.* (2009) and Gurd *et al.* (2006; 2005) support this conclusion, reporting that individuals with slow initial $\dot{V}O_2$ kinetics to the onset of exercise experienced faster $\dot{V}O_2$ kinetics following prior exercise.

2.5.4 The effect of training status

The majority of research investigating the effect of training status on $\dot{V}O_2$ kinetics has been performed via cross-sectional based studies, whereby two groups (i.e. trained vs. untrained) are compared (Marwood *et al.* 2010; Ingham *et al.* 2007; Kilding *et al.* 2006; Cleuziou *et al.* 2005; Koppo *et al.* 2004). This study design has proved popular due to the ease of comparison between two groups of a different training status, compared to the difficulty in recruiting untrained, sedentary participants willing to undergo a training programme, as would be required in a

longitudinal study. Cross-sectional based studies have demonstrated that trained individuals display faster phase II kinetics to exercise onset when compared to lesser trained counterparts in transitions to moderate (Marwood *et al.* 2010; Ingham *et al.* 2007; Kilding *et al.* 2006) and heavy (Ingham *et al.* 2007) intensity domains. A slower response to the phase II kinetics is thought to be due to a larger recruitment of less efficient type II muscle fibres (Koppo *et al.* 2004). The research by Ingham *et al.* (2007) supports that training status alters the muscle fibre type recruitment pattern, finding that elite rowers had a reduced O_2 cost per watt of energy output for both moderate and heavy intensity exercise ($P = 0.02$ and $P = 0.005$, respectively), when compared to lesser trained club rowers. These findings suggest a greater reliance on type I muscle fibres in the elite group. The findings suggest that the phase II kinetics are influenced by training status, thus infer that the phase II kinetics are sensitive to training stimuli.

Carter *et al.* (2000a) found that the phase II kinetics were unchanged due to training, with no change being displayed in the phase II τ before and after a training intervention ($P = 0.63$). However, when the data for the 6 participants with the lowest fitness at recruitment was separately analysed, the phase II τ was reduced (31.5 ± 1.0 to 19.5 ± 1.5s; $P = 0.033$), clearly demonstrating that training can improve the $\dot{V}O_2$ kinetics. Berger *et al.* (2006) further supports that training improves the phase II τ to exercise onset. Additionally, Berger *et al.* (2006) found that the reduction in the phase II τ was significantly correlated to the initial speed of the $\dot{V}O_2$ kinetics, with participants demonstrating the slowest initial phase II τ showing the greatest improvement following training.

Collectively, the research findings have demonstrated that training status influences the phase II τ. Moreover, that more highly trained individuals have a faster phase II τ to exercise onset. The physiological mechanism(s) regulating the $\dot{V}O_2$ kinetics are unclear and continue to be debated. Exercise training, thus the training status of an individual has the potential to improve both O_2 delivery and utilisation; therefore either or both mechanisms could be responsible for an improved phase II τ (Poole *et al.* 2008). Despite the uncertainty regarding the control mechanism, it has been clearly established that the phase II τ is influenced by training.

2.5.5 The effect of the fraction of inspired oxygen

Linnarson *et al.* (1974) found that the manipulation of the fraction of inspired oxygen (FiO_2) altered the level of O_2 deficit incurred. The authors found that hypoxia ($FiO_2 < 0.21$) resulted in a larger O_2 deficit, and hyperoxia ($FiO_2 > 0.21$) resulted in the smallest. Although not directly measured, these findings suggest a slower rate of adjustment of $\dot{V}O_2$ with decreasing levels of FiO_2. These findings have been supported by several researchers (Wilkerson *et al.* 2006; Engelen *et al.* 1996). Engelen *et al.* (1996) investigated whether reduced FiO_2 (0.13) was associated with a longer phase II τ during moderate and heavy exercise. Finding that hypoxia was associated with a significantly slower τ in both moderate and severe intensity exercise. These findings were further supported by Wilkerson *et al.* (2006) who investigated the effect of hyperoxia on $\dot{V}O_2$ kinetics in a similar sample. These studies demonstrate that metabolic capacity in untrained individuals is perfectly matched to ambient O_2 availability as alterations in FiO_2 had a significant impact on the maximal metabolic rate.

However, the work by Wilkerson *et al.* (2006) used a mono- exponential model to describe the $\dot{V}O_2$ kinetics, which has been criticised for its inability to distinguish between the phase II and phase III $\dot{V}O_2$ kinetics. It should be noted that the exercise intensity and modality in the study by Wilkerson *et al.* (2006) should have resulted in a discernible phase III response. Research using a similar sample but a 2-component exponential model, disagree with the findings of Wilkerson *et al.* (2006), reporting that the phase II kinetics were not improved (Hughson and Kowalchuk, 1995), thus suggesting that the O_2 availability is not perfectly matched to ambient O_2, an opinion shared by Haseler *et al.* (2007). The conflicting results both support and disagree with O_2 supply limiting performance. Although similar samples were used by the researchers, different exercise intensities and models were applied to describe the $\dot{V}O_2$ kinetics. Hughson and Kowalchuk (1995) and Haseler *et al.* (2007) investigated moderate intensity exercise, whilst Wilkerson *et al.* (2006) investigated heavy exercise. Collectively, the findings of this research may suggest that regulation of $\dot{V}O_2$ kinetics is exercise intensity dependent.

It has been suggested that the effect of FiO_2 is intensity dependent, such that performance levels to exercise requiring a large aerobic contribution at WRs above the LT; are reduced in conditions of reduced FiO_2, and increased in conditions of increased FiO_2 (Haseler *et al.* 2007; Wilkerson *et al.* 2006; Engelen *et al.* 1996; Hughson and Kowalchuk, 1995). Therefore, it may be assumed that exercise requiring a large anaerobic contribution to the overall energy supply may be unaffected by changes in FiO_2. Researchers have found that reduced FiO_2 had no effect during the performance of the Wingate maximal anaerobic capacity test (WT) (Ogura *et al.* 2006; Calbet *et al.* 2003; McLellan *et al.* 1990). These findings have prompted researchers to investigate to what extent the anaerobic capacity can compensate for limitations in the aerobic contribution and at what point performance becomes limited.

Weyand *et al.* (1999) investigated the influence of reduced FiO_2 (0.13) on middle-distance performance lasting ~2 min. Aerobic capacity, as determined by a ramp test in both conditions, was reduced ~30% in hypoxia compared to normoxic values ($46.2 \pm 7.2 ml.kg^{-1}.min^{-1}$ vs. $60.0 \pm 9.0 ml.kg^{-1}.min^{-1}$, respectively). Running speed in the hypoxic condition was significantly slower than during the normoxic test ($P < 0.05$). However, the authors concluded that although significantly slower running speeds were maintained during the hypoxic testing, performance was largely unaffected. The current researcher believes that the term largely unaffected was used to describe that the severe hypoxic intervention employed in their study, reduced mean running speeds by $0.2 m.s^{-1}$ for sprints of 75s, whereas sprints of 180s were $0.7 m.s^{-1}$ slower in the hypoxic condition. The current thesis is interested in performance lasting ~2 min, therefore it is expected that mean speed will be reduced between $0.2 – 0.7 m.s^{-1}$ by the hypoxic intervention. The authors concluded that despite a ~30% reduction in aerobic power, performance was largely maintained due to an ~18% increase in the contribution from the anaerobic capacity.

However, the study by Weyand *et al.* (1999) had several limitations. The authors selected treadmill speeds so that exhaustion could be achieved within a given time. However, the time to exhaustion that most closely matched the time taken for an elite athlete to run the 800-m (100 – 180s) allowed for a large variation in the time in which exhaustion could be achieved. Furthermore, the researchers did not report the

time to exhaustion between conditions, and no statistical analyses were performed to determine the effect of condition on time to exhaustion. Unmatched times to exhaustion between conditions could have resulted in a different relative energy contribution to the exercise. In addition, the square-wave tests were performed on a treadmill set to an inclination of 4.6°. The efficacy of employing an incline to account for air resistance is questionable, altering the mechanics of running (Sloniger *et al.* 1997; 1997a) thus the $\dot{V}O_2$ kinetics, and reducing the speed achieved during the exhaustive treadmill run compared to that typically observed in 800-m races. Therefore, application of these findings to 800-m track running could be misleading.

2.6 O_2 delivery/utilisation debate

$\dot{V}O_2$ kinetics have been demonstrated to be influenced by a variety of factors, relating to both central and peripheral mechanisms. Understanding whether the $\dot{V}O_2$ kinetics are dependent on supply/central or utilisation/peripheral mechanisms is key in optimising training practices to improve performance (Grassi, 2001). However, there is much debate as to the regulatory mechanism(s); moreover, whether O_2 supply is perfectly matched or is in excess of the metabolic capacity in normoxia; and whether exercise intensity, modality and/or other factors may influence the regulatory mechanism(s). Research findings are in conflict with it having been shown that O_2 supply regulates the $\dot{V}O_2$ kinetics whereby a change in O_2 supply corresponds to a speeding or slowing of the τ, and conversely that $\dot{V}O_2$ kinetics are regulated by O_2 utilisation within the muscle (Haseler *et al.* 2007; Hughson and Kowalchuk, 1995). The current viewpoint regarding the O_2 delivery/utilisation debate is that the arbitrary division of the mechanisms may be overly simplistic, and instead of one mechanism regulating the $\dot{V}O_2$ kinetics, there is a complex interaction of cardiovascular and metabolic factors that regulate the $\dot{V}O_2$ kinetics (Poole *et al.* 2008; Jones and Poole, 2005). For a more detailed discussion into the regulatory mechanisms of $\dot{V}O_2$ kinetics, readers are referred to the review article by Jones and Poole (2005).

2.7 Ecological validity

The research environment can have an effect on the results obtained; with the researcher employing methods to control for extraneous variables, and using measurement equipment not typically used in the sport. The environmental conditions in which tests are conducted affect the air resistance, the force that opposes movement in a given direction. Air resistance is dependent on internal and external conditions. In regard to the external conditions, resistance is dependent on the density of the fluid, in this case air (McArdle *et al.* 2007). Air density is influenced by temperature and pressure, being most dense when temperature is low and pressure is high, and least dense when temperature is high and pressure is low (Wood, 1999). Internal conditions refer to characteristics internal to the object trying to move through the fluid, or factors that can be manipulated by the object to influence the degree of resistance encountered. An example of this would be the surface area of a runner, and the speed the individual is running. In this example, the surface area cannot be readily changed, but the manipulation of speed will affect the degree of resistance encountered, increasing exponentially with speed (Krieg *et al.* 2006). A more readily available and beneficial method that the runner could use to reduce the level of resistance encountered at a given running speed would be to run closely behind a fellow competitor (draft). The characteristics of the competitor being drafted as well as those of the drafter will affect the level of air resistance, with the reduction in resistance being greatest when a small runner runs behind a larger runner (McArdle *et al.* 2007; Wood, 1999).

Within a laboratory environment, there is no air resistance encountered when running on a treadmill. The lack of resistance results in a lower energy cost experienced during treadmill running within a laboratory environment, compared to outdoor running at the same velocity (Jones and Doust, 1996). The different energetic costs experienced during indoor treadmill running has encouraged researchers to attempt to increase the validity of the results obtained on a treadmill, to that which could be expected in the field, by manipulation of speed (Draper and Wood, 2005) or inclination (Jones and Doust, 1996). Jones and Doust (1996) compared the results obtained on a treadmill at different speeds and inclinations, to results obtained in the same group running at different speeds on an outdoor track.

The authors concluded that a 1% treadmill gradient most accurately reflected the energetic demands required to run at the same speed outdoors. Draper and Wood (2005) attempted to account for air resistance when treadmill testing in the laboratory, by increasing the speed of the treadmill by 1km.h^{-1}. However, no research supported their chosen adjustment in speed. The decision by both authors to choose a single value to represent an entire sample is problematic. The manipulation of speed and/or gradient will influence the recruitment patterns of muscle fibre (Sloniger *et al.* 1997; 1997a) influencing the $\dot{V}O_2$ kinetics. In addition, the amount of air resistance experienced whilst running outdoors is unique to the individual dependent on surface area, and the environment.

The majority of research investigating the $\dot{V}O_2$ kinetics in 800-m running has been conducted in a laboratory environment without the influence of air resistance and race tactics i.e. drafting. Furthermore, despite noticeable transitional phases in speed during the 800-m event, with recent World Record performances being run with a positive split and at speeds corresponding to a faster start pacing strategy, the majority of research has utilised a constant speed (square-wave) protocol (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Draper and Wood, 2005; Draper *et al.* 2003). These authors found that during the square-wave exercise test $\dot{V}O_2$ max was not achieved.

Sandals *et al.* (2006) investigated the influence of pacing strategy on the $\dot{V}O_2$ kinetics to severe intensity running. They compared the O_2 uptake between the square-wave test, a race simulation run designed to reflect the optimal race strategy as determined by the transitional phases in speed of the World Record time, and an acceleration run which required the participant to achieve 100% of the average velocity attained in the 800-m race, over the initial 25-m. The findings demonstrated that the O_2 uptake response differed, with a greater $\dot{V}O_2$ peak achieved in the race simulation run (92.5 ± 3.1%) and the acceleration run (90.8 ± 2.8%) than the square-wave test (89.3 ± 2.4%). It has been suggested (Jones *et al.* 2008) that the fast start race strategy increases the speed of the $\dot{V}O_2$ kinetics, sparing the anaerobic capacity at exercise onset and allowing it to be utilised at a different stage. The findings of Jones *et al.* (2008) suggest that fast start pacing strategies may be optimal to performance, allowing the anaerobic capacity to increase power output throughout

exercise and therefore improve performance times (Billat *et al.* 2009; Abbiss and Laursen, 2008). The findings of Sandals *et al.* (2006) suggest that the square-wave test would underestimate the actual $\dot{V}O_2$ peak achieved during an 800-m race by 3.2%. However, despite $\dot{V}O_2$ peak being underestimated by the square-wave test, based on these findings had a race-pace strategy been used in other studies the $\dot{V}O_2$ achieved would still be below $\dot{V}O_2$ peak (~91%).

Research conducted on the $\dot{V}O_2$ kinetics to outdoor 800-m running has reported results contrary to the majority of the literature, instead finding that $\dot{V}O_2$ max was achieved (Thomas *et al.* 2005). All participants in the study by Thomas *et al.* (2005) achieved $\dot{V}O_2$ peak 316 ± 75m into the 800-m run. $\dot{V}O_2$ peak was maintained for the next 219 ± 41m, but then decreased ~24.1 ± 7.0% ($P < 0.05$) during the final 265 ± 104m representative of a decreased velocity. Participants were instructed to run at their optimal race pace and were allowed to alter their speed. Although, it would be difficult to accurately repeat trials, the experience level of the participants suggests that they would have a good idea of their optimal strategy. The measurement system used by Thomas *et al.* (2005) was a telemetric device (Cosmed K4, Roma, Italy) whereas the other studies all used a lab based measurement system. As discussed in chapter 2.3 researchers should approach the comparison of results obtained via different measurement systems with caution. Although it is unlikely that the differences observed between the studies were due solely to this, the measurement system may explain for some of the difference. The majority of the literature determined $\dot{V}O_2$ peak as the highest 15s moving average achieved throughout the test (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Draper and Wood, 2005; Draper *et al.* 2003). However, Thomas *et al.* (2005) used data obtained every 5s throughout the race, and averaged this data over each 25-m interval to normalise the data by distance. This approach to averaging the data is unconventional; averaging data by distance would create an inconsistent time averaging period such that at the start of the race, when the pace would be faster, less data could be used for the average, than at the end. It is likely that the averaging period used by Thomas *et al.* (2005) is responsible for some of the reported differences between the studies.

2.8 Summary

Middle-distance running performance provides a unique opportunity to investigate the interaction of the energy system contribution to exercise in excess of $\dot{V}O_2$ max. Middle-distance running is placed within the severe intensity domain of exercise. However, the $\dot{V}O_2$ kinetics to severe intensity exercise is meant to tend exponentially towards $\dot{V}O_2$ required. However, recent research involving highly aerobically trained individuals has demonstrated that despite sufficient time for the response to develop, during exhaustive exercise of ~2 min $\dot{V}O_2$ does not tend towards the $\dot{V}O_2$ required, and $\dot{V}O_2$ max is not achieved (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a). Furthermore, it has been shown that individuals with a larger aerobic ability achieve a lower percentage of their $\dot{V}O_2$ max than individuals with a smaller aerobic ability (Draper *et al.* 2008; Draper and Wood, 2005a). The $\dot{V}O_2$ kinetics to severe intensity exhaustive exercise is dependent on exercise modality, intensity and the level of fitness of the participants (Draper *et al.* 2003; Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). The magnitude of the $\dot{V}O_2$ kinetics during severe intensity exercise is large, resulting in a small SD/GAIN relationship. This relationship results in a more favourable model fit to fewer exercise transitions than necessary for other intensity domains. It has also been found that during severe intensity treadmill exercise the $\dot{V}O_2$ slow component does not emerge, with no observed pattern to the residuals (Draper and Wood, 2004).

It is therefore proposed that artificially lowering the aerobic capability of trained athletes, who have been shown to have fast phase II $\dot{V}O_2$ kinetics, may result in the attainment of $\dot{V}O_2$ max. However, the manipulation of aerobic capability in a group of highly aerobically trained athletes, whilst performing exhaustive treadmill exercise of ~2 min to investigate $\dot{V}O_2$ peak has not been conducted. Whether $\dot{V}O_2$ max is achieved during an exhaustive severe intensity run of ~2 min has important practical and theoretical implications, for the improvement in training practices and an increased understanding of the regulatory mechanism(s).

CHAPTER 3

METHODS

3.1 Participants

Thirteen males and one female (mean ± SD: age 21.4 ± 3.4 y, height 1.76 ± 0.06 m, mass 66.0 ± 7.0 kg) volunteered to participate in this study. All were competitive middle- and long- distance runners, recruited from local athletics clubs, with a $\dot{V}O_2$ peak (mean ± SD) 67.0 ± 5.2ml.kg^{-1}.min^{-1}. All participants had a seasonal best for the 800-m track event of < 130s. All participants completed a familiarisation session to allow them to experience treadmill running at high speeds and the laboratory procedures involved with the test. Throughout the testing procedure participants were asked to follow their normal training routine. They were required to report to the laboratory in a similar state for all testing having suspended their normal training 24h prior to testing, and to follow their usual competition preparation strategy.

The Ethics Committee of University of Gloucestershire approved the protocols used in the current research. Participants were provided with a written and verbal description of the task explaining the intent of the study, the testing procedure, and the risks of participation. Participants were informed of their right to withdraw from the study at any point without penalty. Prior to testing participants provided written informed consent (Appendix 1) and completion of a health history questionnaire (Appendix 2).

3.2 Study design

Firstly, participants completed a laboratory familiarisation session. All exercise tests were performed on a motorised treadmill (ELG 55, Woodway Gmbh, Weil am Rhein, Germany). The familiarisation session required the participant to perform a square-wave test at 800-m race pace allowing the participant to experience treadmill running at high speeds and also experience exercising whilst wearing a mouthpiece and nose-clip. The speed of the square-wave test was calculated from the participant's seasonal best and aimed to elicit exhaustion in 105-135s. The familiarisation session allowed the speed of the square-wave test to be adjusted to

match the relative intensity between conditions ensuring exhaustion was achieved in both conditions in the allowed time i.e. 105-135s. Although, time to exhaustion was used to match/control intensity between conditions, it is recognised that this is a contested concept.

Following familiarisation, the participants reported to the laboratory on four separate occasions at the same time of day, on four separate days to avoid circadian timing effects that may affect metabolic responses (Aldemir *et al.* 2000). Participants were required to complete a ramp test to exhaustion in hypoxia and normoxia (H_ramp and N_ramp, respectively), and a square-wave test at 800-m race pace in hypoxia and normoxia (H_square and N_square, respectively). The order of testing was counterbalanced to minimise any order effects.

Participants followed a predetermined warm-up prior to the square-wave exercise tests. To ensure the same relative intensity of warm-up between conditions, the warm-up was conducted in normoxic conditions. The participant was required to run for 5 minutes at 12km.h^{-1}, the speed then increased to 15km.h^{-1} for a further 2 minutes, participants were then required to complete three 10s transitions at the speed of the subsequent square-wave tests. Each transition was separated by 30s of rest. Participants were then encouraged to perform any necessary stretching for 2 minutes.

Following the warm-up participants were allowed a 5 minute rest period in which they straddled the treadmill allowing the belt to move at the required speed for the test. Heart rate was recorded for the final 2 minutes of the recovery, and breath-by-breath data was recorded for the final minute. Data from this period was used to determine baseline values.

3.3 Test protocols

All testing took place in an environmental chamber (Sanyo Gallenkamp PLC, Loughborough) with the FiO_2 being manipulated to reflect either sea level values or hypoxia (FiO_2 ~0.21 and ~0.13, respectively), consistent with Weyand *et al.* (1999).

Air temperature and humidity were controlled at ~16°C and ~40% respectively. Participants were not informed of the environmental conditions of the specific test.

The speed of the ramp test was increased by 0.1km.h^{-1} every 5s (a ramp rate of 1.2km.h^{-1}.min^{-1}). The initial speed of the ramp test was estimated depending on the fitness of the participant and aimed to elicit exhaustion between 8 – 12 minutes (Buchfuhrer *et al.* 1983). If exhaustion was reached outside of this time period they were required to return to the laboratory on another day and perform another ramp test in that condition with an adjusted initial speed.

The speed of the square-wave tests were based on the results of the familiarisation session, and aimed to elicit exhaustion within 105 – 130s. If exhaustion was not achieved within this time, the treadmill speed was adjusted accordingly and participants were required to return to the laboratory on another day and perform another square-wave test in that condition. The treadmill was level for all exercise tests.

3.4 Data Acquisition

Throughout testing participants were required to wear a chest strap to allow heart rate to be recorded every 5s using short-range telemetry (810i; Polar Electro Oy, Kempele, Finland), and to breath through a low dead-space (90ml), low resistance (5.5cmH^{2}0 at 510 l.min^{-1}) mouthpiece and turbine assembly. Gases were drawn continuously from the mouthpiece through a 2-m sampling line (0.5mm internal diameter) to a quadrupole mass spectrometer (MSX 671; Ferraris Respiratory Europe Ltd, Hertford, UK) where they were analysed for O_2, CO_2 and N_2. Expired volumes were determined using a turbine volume transducer (Interface Associates, Alifovieja, US). Prior to each test the mass spectrometer and the turbine were calibrated using gas mixtures (Linde Gas, London, UK) of known compositions, and a 3-l syringe (Hans Rudolf, Kansas, US), respectively. Oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$) were calculated for each breath.

3.5 Calibration

The environmental chamber had a hypoxic unit that allowed the gas concentration to be manipulated. To simulate hypoxic conditions, O_2 molecules were filtered through a membrane in the external O_2 unit allowing a greater concentration of nitrogen (N_2) to flow through into the environmental chamber to simulate hypoxia. Atmospheric conditions within the chamber were constantly monitored, and the external O_2 unit would automatically adjust the flow of gases to maintain the required O_2 percentage. To ensure the accuracy of the external O_2 unit thus the validity of the environmental conditions, it was necessary to also use a mass spectrometer, positioned outside of the environmental chamber, to monitor the conditions inside. Therefore, when testing in hypoxia, two mass spectrometers were used simultaneously, one outside of the environmental chamber to monitor the internal environment, and one within the chamber to record the breath-by-breath data during testing.

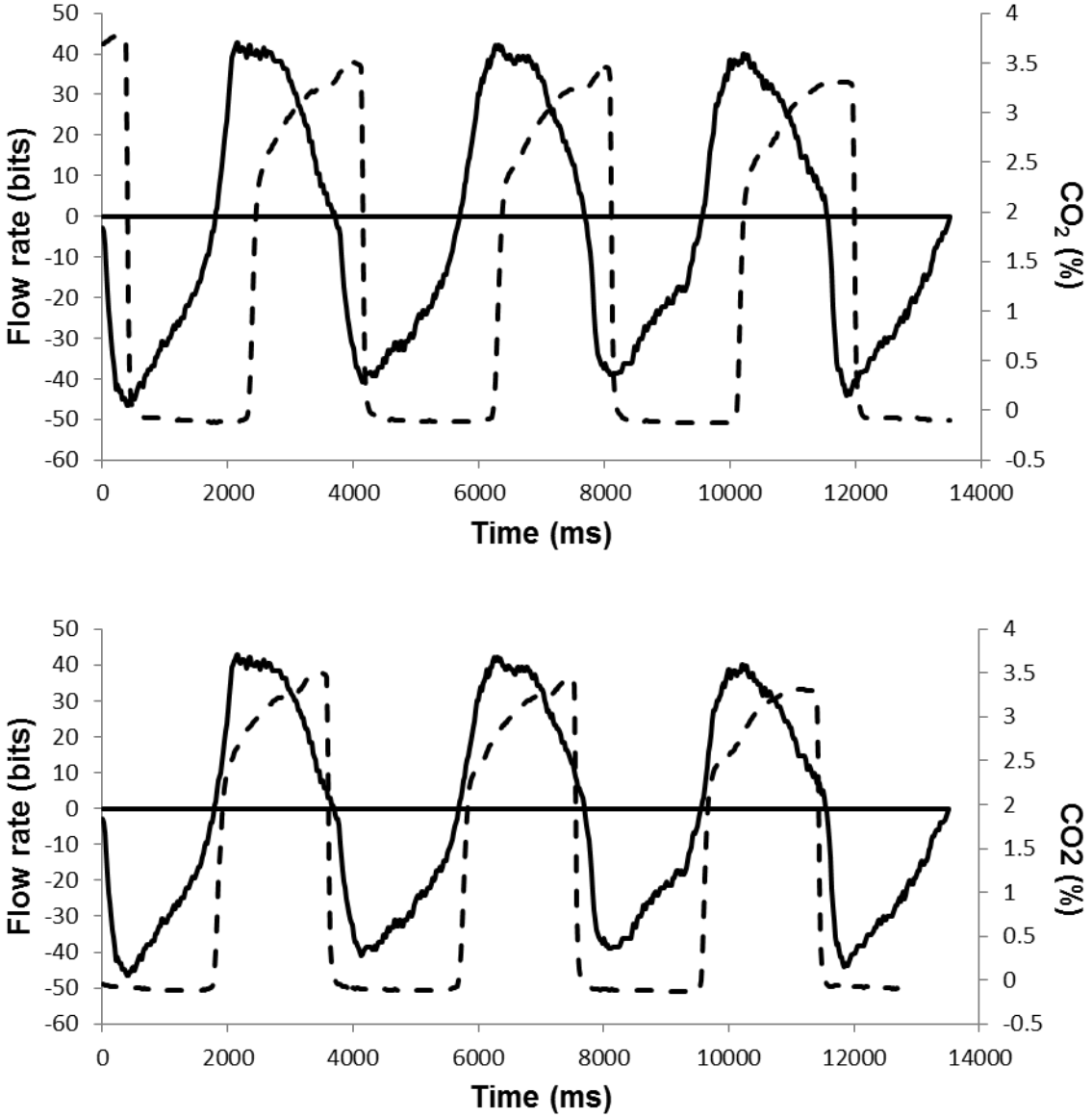
To accurately calibrate the mass spectrometers, gases of known concentrations had to be used. Due to limitations of the environmental chamber, hypoxic gas was leaked into the surrounding area, creating uncertainty to the air composition. To overcome this, the mass spectrometer outside of the environmental chamber had a sample line extracting outside atmospheric air of known composition. Calibration of this mass spectrometer could then occur against the known outside atmospheric air and a gas bottle of known concentration (14.99% O_2, 5.01% CO_2, 5.02% Argon, and 74.98% N_2). Following successful calibration, this mass spectrometer sampled the air inside the environmental chamber. When the values measured matched the desired environmental conditions, calibration of the mass spectrometer inside the environmental chamber could occur in line with these values. The mass spectrometer inside the environmental chamber was then calibrated using either a normoxic or hypoxic gas bottle, dependent on the conditions of the test. In hypoxic conditions, calibration was performed with a gas bottle composed of 5% O_2, 5.01% CO_2, 5.02% Argon, and 84.97% N_2; which allowed the detection of low levels of O_2 in the expirate gas. When testing in normoxia only the mass spectrometer inside the chamber was used. The gas bottle used to calibrate in normoxia was composed of 14.99% O_2, 5.01% CO_2, 5.02% Argon, and 74.98% N_2. When agreement was achieved between the two mass spectrometers for the composition of gas within the

environmental chamber, gas volume could then be calibrated and testing could commence.

Delay time is also an important factor to consider when using an on-line gas analysis system. The online-gas analysis system calculates both flow and gas concentration. However, the measurement of flow is almost instantaneous whereas the measurement of gas concentration takes slightly longer. The online-gas analysis system accounts for the difference observed in the timings of determination of flow and gas concentration, termed delay time. The computer software can automatically calculate the delay time, being programmed to detect changes in the O_2 and CO_2 concentrations and flow rate differences indicative of inspiration and expiration. However, it is programmed to detect values of normoxic O_2 and CO_2, and therefore may be insensitive to the determination of breaths during the hypoxic conditions. Therefore, the automatic determination of delay-time by the computer software may result in the misalignment of flow rate and gas concentration data. The effect of the misalignment of gas volume and concentration data is illustrated in Figure 3.1.

The misalignment of the flow and CO_2 expiration data i.e. an in-accurate delay time, can affect the values calculated for $\dot{V}O_2$. As highlighted in Figure 3.1, an inaccurate delay time results in the incorrect alignment of the breath being analysed, resulting in an inaccurate representation of the breath data. Therefore, manual determination of delay time was deemed necessary prior to each test. The analysis software produces a raw data file that reports the gas concentrations and flow values every 20 ms, allowing the start and end of each breath to be determined. The flow data is provided in the raw data file by bit rate values, with inspiration and expiration being represented by a negative and positive bit rate respectively. Therefore, the point at which the bit rate switches from a positive to a negative value marks the end of expiration and the beginning of inspiration, thus the end of inspiration and the beginning of expiration is the point at which bit rate switches from a negative to a positive value. Due to O_2 consumption at the muscle and the resultant production of CO_2, inspiration and expiration can be determined by gas concentration. The point at which O_2 concentration begins to increase and CO_2 concentration decrease represents the start of inspiration and end of expiration. The point at which O_2 concentration decreases and CO_2 concentration increases represents the start of

expiration and end of inspiration. Therefore, an accurate delay time can be determined through the difference between the start and end point of each breath as defined by flow and gas concentrations.



Key:

—— Flow rate

----- CO_2 Output

Figure 3.1 The alignment of breath data when the delay time is set to automatic (top panel) and 540 ms (bottom panel).

3.6 Data Analysis

Breath-by-breath data were converted to second-to-second data using linear interpolation between breaths; data were then time aligned to the start of the test. Moving 15s averages were used to calculate $\dot{V}O_2$ for every complete 15s period throughout the test conditions; both the ramp and the square-wave tests. The highest $\dot{V}O_2$ reported by the 15s moving average was considered to be the participants $\dot{V}O_2$ peak for the respective test. The $\dot{V}O_2$ peak and HR peak data were tested for normality according to the criteria described by Duffy and Jacobsen (2001). These tests confirmed that the data was normally distributed as the coefficient did not exceed 1.96 times its own standard error. The phase I data was removed from the $\dot{V}O_2$ data. The phase II data was then described from the interpolated $\dot{V}O_2$ data from the square-wave tests using equation 1.

$$\dot{V}O_2\,(t) = \text{Baseline} + A*(1 - e^{-(t-\delta)/\tau})$$

eq.1

Where t is time, Baseline is the average of the $\dot{V}O_2$ attained during the final minute of gas collection during the 5 minute rest period, 1 minute prior to the start of the test, A is the amplitude (above Baseline) of the phase II response, δ is the delay between the start of the square-wave test and the onset of phase II, and τ is the time constant of the exponential response of phase II. To derive estimates for the parameters A, τ and δ, unconstrained non-linear regression (least sum of squares by iteration; SPSS for Windows version 16.0) was used to fit equation 1 to each participants $\dot{V}O_2$ data, having first excluded the phase I data (Draper and Wood, 2005).

The interaction of test type (ramp vs. square-wave) and condition (normoxic vs. hypoxic) was evaluated for $\dot{V}O_2$ peak (highest 15s moving average) and HRpeak (highest 5s value), using 2 x 2 (test x condition) repeated measures ANOVA. Separate related samples t-tests were performed to explore any significant effect. Related sample t-tests were used to interrogate the difference between the percentage of $\dot{V}O_2$ peak achieved during the square-wave test in that respective condition; the

difference between $\dot{V}O_2$ peak achieved during the square-wave tests as determined by the N_ramp test; and the time to exhaustion between conditions for the square-wave tests. The modelled $\dot{V}O_2$ data to the square-wave tests (parameter estimates) were compared in terms of Baseline, A, τ, δ, mean response time ($\tau + \delta$), and the asymptotic $\dot{V}O_2$ (Baseline + A) using related samples t-tests. The Bootstrap standard error of the estimate method was used to determine the accuracy of the parameter estimates. Pearson's Product Moment correlation was used to investigate the relationship between N_ramp $\dot{V}O_2$ peak and the percentage of this peak achieved during the N_square tests. Statistical significance of all tests was set at $P \leq 0.05$.

CHAPTER 4

RESULTS

Table 4.1 displays the $\dot{V}O_2$ peak, HR peak and duration (mean ± SD) from the four exercise tests. The $\dot{V}O_2$ kinetics for the square-wave tests in both conditions for a representative participant, and the group mean are shown in Figure 4.4. Key elements of SPSS outputs for the data analyses can be found in appendix 5.

The ANOVA revealed a significant interaction effect for $\dot{V}O_2$ peak ($P < 0.001$). A main effect for test and condition was revealed ($P < 0.001$ and $P < 0.001$, respectively). Figure 4.1 illustrates the interaction effect for $\dot{V}O_2$ peak. Post-hoc related samples t-test revealed a significant difference in the percentage of $\dot{V}O_2$ peak achieved in the square-wave tests (as determined by the ramp test in the respective condition) ($P < 0.001$); and that $\dot{V}O_2$ peak during the hypoxic ramp and square-wave exercise tests were not significantly different ($P = 0.48$). The percentage of $\dot{V}O_2$ peak achieved was greater during the H_square (mean ± SD: 102 ± 0.08 vs. 86 ± 0.06%, respectively). The related samples t-test for the determination of differences between the percentage N_ramp $\dot{V}O_2$ peak achieved during the H_square and N-square tests, revealed a significant difference ($P < 0.001$). The percentage of N_ramp $\dot{V}O_2$ peak achieved was greater in the N_square test (mean ± SD: 86 ± 0.06 vs. 69 ± 0.06%). The relationship between $\dot{V}O_2$ peak from the N_ramp test and the percentage achieved during the N_square test was significant and negatively correlated ($r = -.637$, $P = 0.014$), this relationship is shown in Figure 4.3.

The ANOVA revealed no significant interaction for HR peak ($P = 0.26$) (Figure 4.2). No difference was found in the main effect for test. The main effect for condition was found to be significant ($P < 0.001$).

The related samples t-test for the determination of differences in test duration between the square-wave tests, found no differences existed ($P = 0.873$).

The parameter estimates from the modelled square-wave tests in normoxia and hypoxia are displayed in Table 4.2. The range of values for the bootstrap standard error of estimates are displayed in Table 4.3.

Table 4.1 $\dot{V}O_2$ peak, HR peak and duration from the exercise tests (mean ± SD). $\dot{V}O_2$ is expressed in ml.min^{-1}, HR is expressed in beats per.min^{-1}, duration is presented in seconds, and speed is presented in km.h^{-1}.

Test	$\dot{V}O_2$peak	HRpeak	Duration	Speed
N_square	3786 ± 471	185 ± 7	114 ± 5	21.9 ± 1.0
H_square	3015 ± 299	181 ± 6	114 ± 11	20.6 ± 1.1
N_ramp	4404 ± 423	189 ± 7	564 ± 46	-
H_ramp	2970 ± 272	181 ± 7	571 ± 118	-

*The speed data for the square-wave tests is displayed for each participant in appendix 4.

Table 4.2 Data from the parameter estimates (mean ± SD)

	Normoxia	Hypoxia	P - value
Baseline $\dot{V}O_2$ (ml.min^{-1})	600 ± 109	669 ± 153	0.181
GAIN (ml.min^{-1})	2447 ± 496	1611 ± 266	0.001
Asymptote (ml.min^{-1})	3047 ± 505	2280 ± 206	0.001
% $\dot{V}O_2$ peak	84 ± 0.06	102 ± 0.07	0.001
τ (s)	10.4 ± 2.6	12.7 ± 2.8	0.029
δ (s)	7.6 ± 2.6	7.4 ± 3.3	0.851
MRT (s)	18.0 ± 2.9	20.1 ± 3.3	0.067

Table 4.3 The range of the data for the bootstrap standard error of estimates

	Normoxia	Hypoxia	P - value
Asymptote (ml.min^{-1})	2.402 - 12.690	3.149 - 10.196	0.782
τ (s)	0.290 - 0.775	0.182 - 0.629	0.942
δ (s)	0.201 - 0.928	0.124 - 0.948	0.197

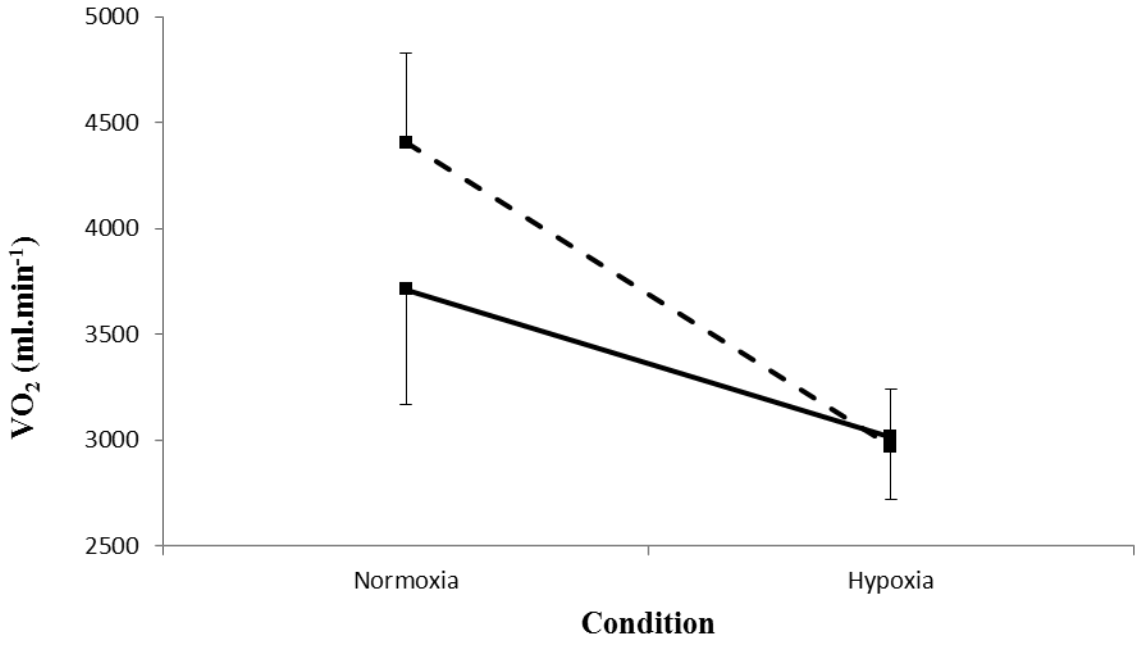


Figure 4.1 The interaction effect for the mean $\dot{V}O_2$ peak for the ramp tests (broken line) and the square-wave tests (solid line).

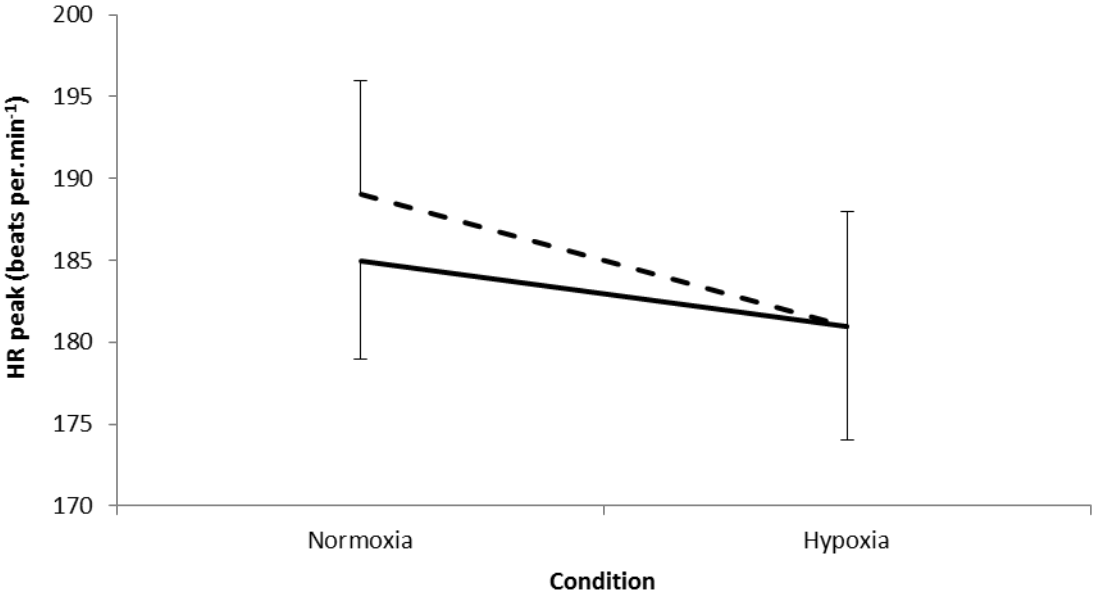


Figure 4.2 The interaction effect for the mean HR peak for the ramp tests (broken line) and the square-wave tests (solid line).

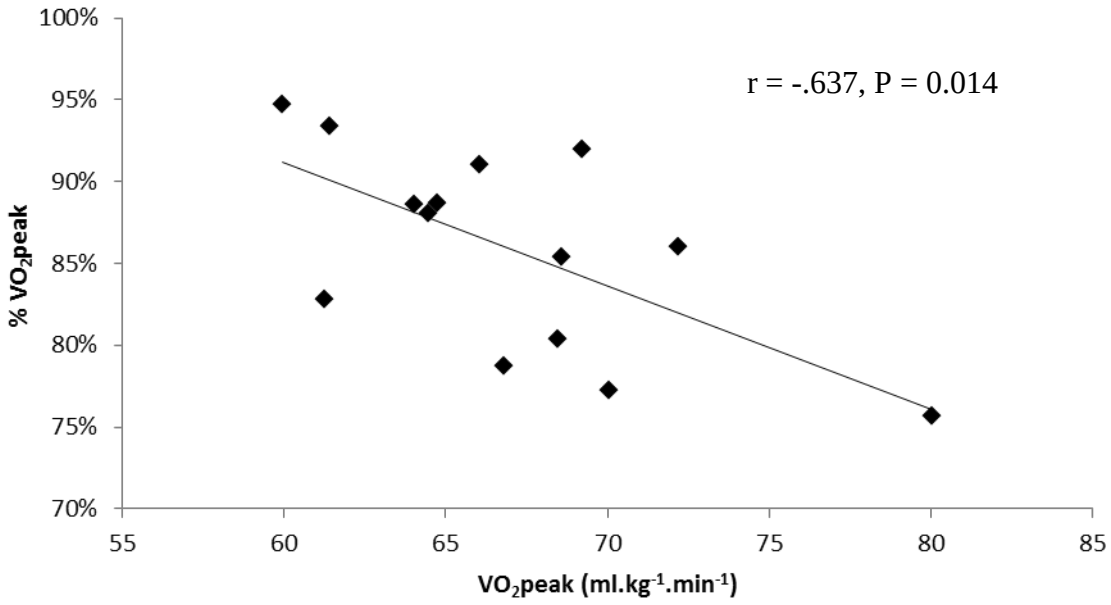


Figure 4.3 Relationship between the N_ramp $\dot{V}O_2$ peak and the percentage achieved during the N_square test.

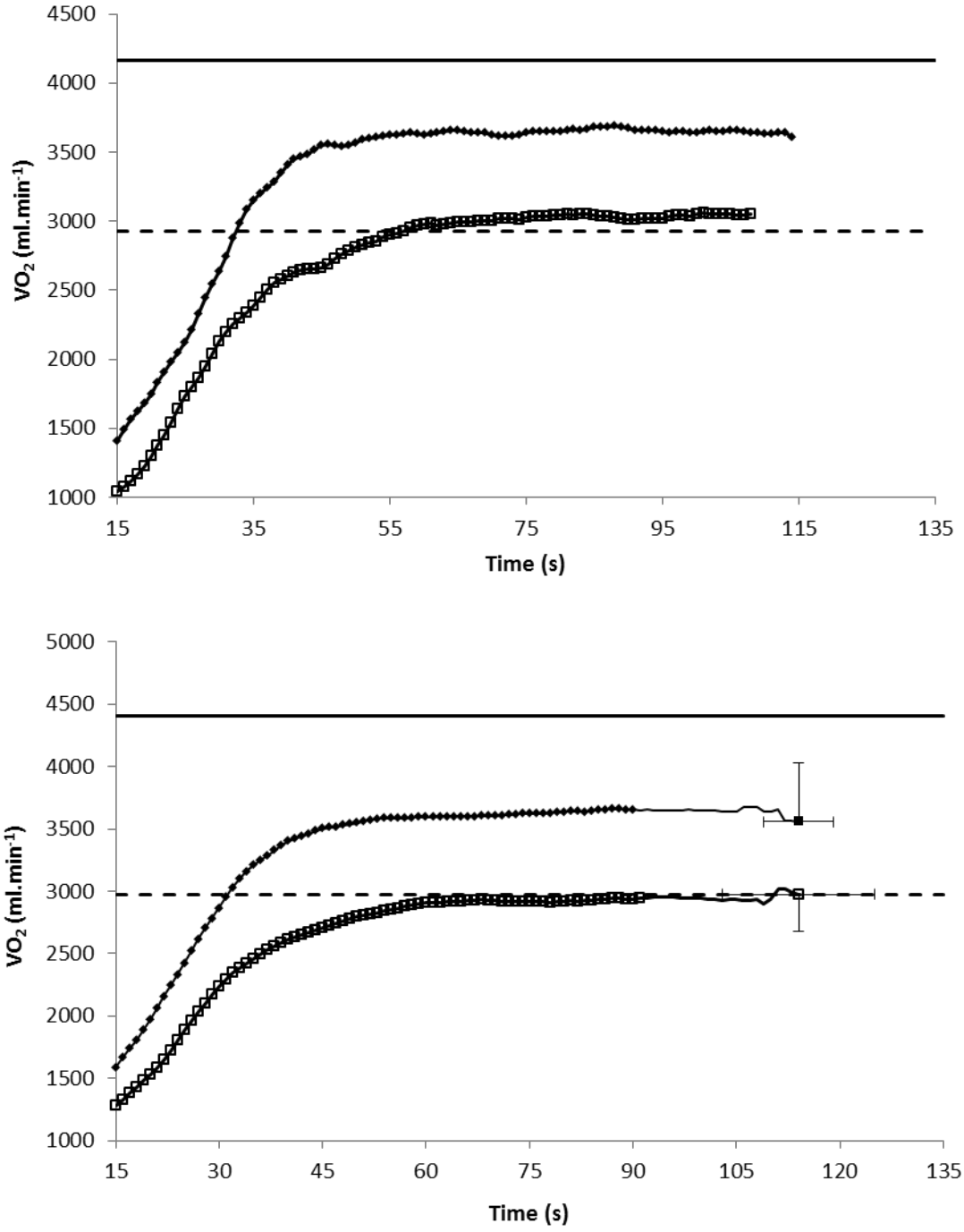


Figure 4.4 The $\dot{V}O_2$ kinetics of a representative participant (top panel) the group mean (bottom panel) to the N_square test (closed symbols) and H_square test (open symbols). The $\dot{V}O_2$ peak to the N_ramp (solid line) and H_ramp (broken line) is also provided.

Note: Error bars represent the standard error of measurement. For clarity the error bars are omitted for all but the final data point.

CHAPTER 5

DISCUSSION

5.1 Key Findings

It was found that during matched duration severe intensity treadmill exercise to exhaustion in normoxia and hypoxia, the percentage of peak aerobic power achieved (as determined by the ramp test in the respective condition) was greater in the hypoxic condition. Moreover, it was found that individuals unable to achieve $\dot{V}O_2$ peak during severe intensity exercise to exhaustion in normoxia, were able to achieve $\dot{V}O_2$ peak when performing the same type of exercise in hypoxia. Although, the severe intensity treadmill run in normoxia produced the greatest $\dot{V}O_2$ values when expressed as a percentage of the N_ramp $\dot{V}O_2$ peak. Furthermore, individuals with a greater normoxic peak aerobic power achieved a lower percentage of that peak during the severe intensity treadmill exercise in normoxia. The bootstrap standard error of estimate for the time based parameters of the modelled response demonstrated no difference between conditions. Moreover, the bootstrap standard error indicated a good model fit to the data thus a high degree of confidence in the parameter estimates.

5.2 $\dot{V}O_2$ peak and HR peak

The focus of the current study was to manipulate the aerobic capability of an individual to determine whether artificially lowering aerobic capability, would result in the attainment of peak aerobic power during severe intensity treadmill running ~2 min. As expected, reducing FiO_2 (0.13) reduced the $\dot{V}O_2$ peak achieved during the ramp test by ~33%. This reduction in peak aerobic power is similar to other values reported in the literature when FiO_2 had been reduced to a similar level and participants of an equally trained status had been used (Weyand *et al*. 1999).

It was found that during severe intensity treadmill exercise in normoxia, $\dot{V}O_2$ peak was not achieved. This finding is in agreement with other research investigating exercise of this mode and intensity, in highly aerobically trained individuals (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals

et al. 2006; Draper and Wood, 2005; 2005a). The current study also supports previous research that has found individuals with the greatest peak aerobic power achieve a lower percentage of this power during exercise of this kind (Draper *et al.* 2008; James *et al.* 2007a; Draper and Wood, 2005a). The literature reports a mean $\dot{V}O_2$ peak of ~88%. The participants recruited by the current thesis achieved ~86% of their peak aerobic power, a value typical to the exercise mode and intensity when using highly trained individuals. However, this response seems to be specific to the exercise mode, with highly aerobically trained individuals shown to achieve $\dot{V}O_2$ peak during severe intensity cycling exercise (Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998; Hill and Ferguson, 1999; Poole and Richardson, 1997). The different responses observed between severe intensity running and cycling exercise has been attributed to the mechanics of the exercise, with running allowing for a greater storage of elastic energy (Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998; Van Inghen Schenau *et al.* 1997).

A novel finding of the current thesis was that individuals unable to achieve $\dot{V}O_2$ peak during exhaustive severe intensity treadmill running ~2 min in normoxia, were able to achieve $\dot{V}O_2$ peak in hypoxia. This finding is supported by research that has found the percentage of $\dot{V}O_2$ peak achieved is related to aerobic capability, with individuals possessing a lower peak aerobic power, achieving a greater percentage (Draper *et al.* 2008; Draper and Wood, 2005). The inverse relationship between peak aerobic power and the percentage of that power achieved, clearly demonstrates that individuals with a larger $\dot{V}O_2$ peak are unable to utilise this aerobic advantage during an effort of this kind. In the current thesis peak aerobic power was reduced through the manipulation of FiO_2. This finding demonstrates that during severe intensity exhaustive treadmill exercise ~2 min, there is sufficient time for $\dot{V}O_2$ peak to be achieved. This finding presents questions regarding the mechanism(s) responsible for regulating O_2 uptake.

Having determined $\dot{V}O_2$ peak could be achieved during exhaustive severe intensity exercise ~2 min when peak aerobic power was reduced, it was important to investigate whether the $\dot{V}O_2$ values achieved differed between conditions. It was found that the percentage of the N_ramp $\dot{V}O_2$ peak achieved was different between the square-wave exercise tests, being greater in normoxia than hypoxia. Assuming a

similar overall energy demand to the square-wave exercise tests between the two conditions, this finding demonstrates that exercise in the hypoxic condition is not sustained by an increased aerobic contribution, a conclusion in agreement with Weyand *et al.* (1999). Although anaerobic contribution was not measured in the current study, the findings suggest that during exhaustive exercise of matched duration, there is an increased contribution from the anaerobic pathways of energy metabolism. Weyand *et al.* (1999) estimated a ~18% increase in anaerobic metabolism. These findings suggest that the anaerobic capacity is not fully exhausted during severe intensity running and/or question whether the anaerobic capacity is in fact finite.

Although an interaction effect was found for $\dot{V}O_2$ peak, no interaction was found for HR peak. Instead, HR peak was found to have a main effect for condition, with a greater HR peak being achieved in normoxia. This finding demonstrates that during the hypoxic tests maximal HR was not achieved. Assuming that HR peak is representative of cardiac output, such that an increase in HR peak results in an increased cardiac output, the findings suggest that cardiac output was reduced during the hypoxic tests. However, despite HR peak thus cardiac output not achieving peak values in the hypoxic condition, $\dot{V}O_2$ peak was still achieved. In addition, no difference was found in the HR peak between tests. Therefore, it is unlikely that HR peak thus cardiac output per se is the factor limiting the attainment of $\dot{V}O_2$ peak during normoxic severe intensity exercise.

5.3 Parameter Estimates

The $\dot{V}O_2$ kinetics to severe intensity exhaustive exercise lasting ~2 min was found to be different between conditions. No change was found in the phase II delta (δ). The phase II time constant (τ) was different between conditions, with a greater value for τ thus a slower response to exercise being evident in the hypoxic condition. However, no difference was found in the mean response time (MRT) (τ + δ) between conditions. The parameter estimates for GAIN, Asymptote and Asymptotic $\dot{V}O_2$ were different between conditions, with the normoxic condition producing greater values. These findings demonstrate that despite $\dot{V}O_2$ tending towards a greater

asymptote during the N_square tests, the $\dot{V}O_2$ kinetics were significantly faster in the normoxic condition.

Several authors have found that the $\dot{V}O_2$ kinetics at the onset of exercise are much slower than reported in the current thesis. Wilkerson *et al.* (2006) reported a MRT of 43 ± 5s, similarly Engelen *et al.* (1996) reported a MRT of ~48.5s. However, $\dot{V}O_2$ kinetics have been shown to be dependent on training status (Marwood *et al.* 2010; Ingham *et al.* 2007; Berger *et al.* 2006; Kilding *et al.* 2006; Draper and Wood, 2005a), exercise intensity, and mode (Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). Researchers investigating severe intensity treadmill running using similarly highly aerobically trained participants have reported similar parameter estimates as the current thesis (Draper *et al.* 2006; Draper and Wood, 2005; 2005a).

The current thesis found that a slower $\dot{V}O_2$ kinetic response was demonstrated during the hypoxic exercise. The δ time was not found to be different between conditions. Therefore, the increased MRT, evident in the hypoxic condition, was due to an increased τ. These findings support those of Engelen (1996) who reported that $\dot{V}O_2$ kinetics were increasingly slowed with increasing levels of hypoxia; such that the $\dot{V}O_2$ kinetics at an FiO_2 of 0.12 were significantly slowed compared to normoxia and FiO_2 0.15 ($P < 0.01$). However, Wilkerson *et al.* (2006) reported no differences in the $\dot{V}O_2$ kinetics to exercise in hyperoxia ($FiO_2 = 0.5$). These findings may be explained by the sigmoidal shape of the oxygen dissociation curve. During exercise in normoxia, a healthy individual remains within the flat region of the sigmoidal curve, such that a reduction in the partial pressure of oxygen in the blood (PaO_2) has little effect on blood saturation. By extension, due to the small effect observed on levels of blood saturation during normoxic exercise, increased PaO_2 through hyperoxia can have little effect, possibly explaining the findings of Wilkerson *et al.* (2006). However, during exercise in hypoxia, PaO_2 is reduced past the flat region of the sigmoidal curve, whereby small reductions in PaO_2 result in large reductions in blood saturation.

A reduction in the phase II τ may have potential performance implications. An improved MRT would theoretically reduce the reliance on anaerobic energy metabolism to supply energy at the onset of exercise (Billat *et al.* 2009; Jones *et al.*

2008). Assuming a finite anaerobic capacity, a change in the relative energy contribution provided by aerobic and anaerobic metabolism would theoretically impact performance. Jones *et al.* (2008) concluded that faster phase II kinetics reduced the anaerobic contribution at the onset of exercise allowing participants to prolong exercise utilising the spared anaerobic energy later in the exercise. It is therefore suggested, that during exercise of a fixed distance/workload, faster phase II kinetics may spare the anaerobic system and allow the anaerobic capacity to release energy evenly throughout the exercise to improve performance time/work performed. Although this assumption has yet to be confirmed, this may partly explain the differences in the speed of the square-wave exercise tests between the conditions. The faster phase II kinetics during the N_square tests reduced the anaerobic contribution at exercise onset, allowing the anaerobic capacity to improve the speed maintained, compared to the slower speeds achieved in hypoxia (table 4.1; appendix 4).

5.4 Bootstrap standard error of estimate for the time based parameters

The bootstrap standard error of estimate reported in the current thesis demonstrates a good model fit to the data, with no difference in the parameter estimates found between conditions. The standard error reported demonstrates that exercise of severe intensity can be modelled with a high degree of confidence from fewer transitions than exercise of a lower intensity. Moreover, the data of this thesis demonstrate greater levels of confidence in the modelled response to a single transition than previous research which has modelled eight transitions Lamarra *et al.* 1987). Lamarra *et al.* (1987) found that during moderate intensity exercise eight transitions were needed to report confidence limits of ± 2s of the model fit to the phase II response. The current thesis reported greater confidence due to a greater magnitude of response thus a smaller SD/GAIN ratio. The findings of the current thesis are similar to those reported by other researchers who also modelled the response of severe intensity treadmill exercise from a single transition (Draper *et al.* 2006). The bootstrap standard error reported by Draper *et al.* (2006) ranged from 9 – 41 $ml.min^{-1}$, 0.35 – 1.07 s, and 0.33 – 1.09 s for the asymptote, τ and δ, respectively. The confidence limits of the current thesis are better than those previously reported

(Draper *et al.* 2006), this difference may be due to the current thesis recruiting participants of greater homogeneity and peak aerobic power.

5.5 Methodological implications

5.5.1 Test Duration

The duration of test was an important methodological factor. Previous investigation into severe intensity treadmill exercise in normoxia and hypoxia (FiO_2 = 0.13) had not matched time to exhaustion between conditions, possibly resulting in a different relative intensity between conditions (Weyand *et al.* 1999). The current thesis aimed to control/match the relative intensity, through matched test duration between conditions; this would allow any observed difference to be attributed to the test condition and not due to differences in test duration.

A valid ramp test, for the determination of $\dot{V}O_2$ peak and HR peak, is dependent on the participant achieving central fatigue before peripheral fatigue (Buchfuhrer *et al.* 1983). In an investigation to determine the optimal ramp test duration, Buchfuhrer *et al.* (1983) found that the participant must reach exhaustion between 8- and 12-minutes to produce a valid $\dot{V}O_2$ peak. In the current study, the ramp test protocol elicited exhaustion in all participants between 8- and 12- minutes in both conditions. Therefore, the $\dot{V}O_2$ peak achieved during the ramp tests can be attributed to the change in FiO_2. Moreover, the reduction in $\dot{V}O_2$ peak is similar to that previously reported (Weyand *et al.* 1999). However, it should be noted that in order to elicit exhaustion in both conditions, in a similar duration, the starting speed of the ramp test in hypoxia was reduced.

The square wave tests were also controlled/matched for time to exhaustion to ensure the same relative intensity was achieved between the conditions. No significant difference was observed between the test duration of the square-wave exercise tests between conditions. Previous to this thesis, no research had been conducted that accurately matched the test duration between conditions. The findings of the current study are novel, demonstrating important differences between matched exercise intensities at different FiO2. However, it should be noted that to match the relative

intensity of the exercise between conditions, the speed of the H_square tests were reduced compared to the N_square tests (table 4.1; appendix 4).

5.5.2 Modelling of the breath-by-breath response

As previously discussed (chapter 2.4) there are a variety of approaches to model the $\dot{V}O_2$ kinetics. The exhaustive square-wave exercise tests described in the current thesis, were of an exercise mode and intensity not typically associated with an increase in $\dot{V}O_2$ during phase III (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; James *et al.* 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; Draper *et al.* 2003; Carter *et al.* 2000; Hill *et al.* 2003; Billat *et al.* 1998). Visual inspection of the $\dot{V}O_2$-time relationship confirmed that no phase III increase in $\dot{V}O_2$ was manifest in the current study. Having concluded no discernible phase III response was present in the data, the current researcher had to decide how to deal with the phase I data.

Bell *et al.* (2001) presented many arguments against the inclusion of phase I data, whilst presenting few and weak arguments to justify its inclusion in the modelled response. Modelling the phase I data is appealing as it would allow all data points to be included in the analysis. However, there are two major objections to the inclusion of phase I data; 1) the assumption that the response is in fact exponential, 2) the small confidence limits that phase I data can be modelled. Several researchers have found close agreement between cardiac output and the $\dot{V}O_2$ kinetics (Bell *et al.* 2001; Yoshida *et al.* 1993). However, despite close agreement, no physiological evidence was found to support the use of an exponential term to model this response.

With regard to the low confidence limits to which phase I data can be modelled, Lamarra *et al.* (1987) found that the level of noise in the measured $\dot{V}O_2$ response was unchanged by intensity. This finding demonstrates the importance of the magnitude of the response in the efficacy of modelling a given parameter. The magnitude of the phase I response is much smaller than the phase II response and that of the $\dot{V}O_2$ slow component (if manifest). Therefore, the SD/GAIN relationship of the phase I data would indicate a poor model fit. Additionally, Lamarra *et al.* (1987) found eight transitions were needed to achieve 95% confidence limits of ± 2 s

of the model fit τ to the phase II response, during moderate intensity exercise. Assuming the same level of noise throughout the response, an even greater number of transitions would be needed to achieve the same degree of confidence in the parameter estimates for the phase I data. Therefore, the phase I data was not modelled in the current thesis.

The accurate determination of the start of phase II, thus the exclusion of phase I data, is important when modelling the phase II response. The inclusion of data points belonging to phase I, and/or the removal of data points belonging to phase II would impact the parameter estimates of τ and δ, thus the MRT. The $\dot{V}$O2 kinetics are dependent on the training status of the participants (Marwood *et al.* 2010; Ingham *et al.* 2007; Berger *et al.* 2006; Kilding *et al.* 2006; Draper and Wood, 2005a), exercise intensity and mode (Draper *et al.* 2003; Hill *et al.* 2003; Carter *et al.* 2000; Billat *et al.* 1998). Therefore, the duration of phase I is varied within the research. The current thesis adopted the approach used by previous researchers that have recruited similarly trained participants, and investigating exercise of the same intensity and mode as the current thesis, removing the first 15 s of data from any modelled response (Draper *et al.* 2005).

5.6 Mechanism(s) proposed to regulate the $\dot{V}O_2$ response

The findings of the current thesis are in support of the literature (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; Draper *et al.* 2003), finding that $\dot{V}O_2$ peak was not achieved during exhaustive severe intensity treadmill exercise ~2 min, in trained individuals in normoxia. However, when the aerobic capability was reduced via the manipulation of FiO_2, the same individuals were able to achieve $\dot{V}O_2$ peak. This finding demonstrates that the exercise is of sufficient duration for $\dot{V}O_2$ peak to be achieved. The fundamental question remained; why did $\dot{V}O_2$ not tend to peak during exercise where the $\dot{V}O_2$ required was greater than $\dot{V}O_2$ peak?

Although $\dot{V}O_2$ peak was achieved in hypoxia, HR peak was not. Conversely, during the same type of exercise in normoxia, HR peak was achieved, but $\dot{V}O_2$ peak was not. As discussed previously (section 5.2), it is unlikely that HR peak thus cardiac

output per se is the limiting factor. It may then be deduced that the distribution of blood flow during the exercise between the conditions may vary; such that the blood flow to the working muscles is limiting the attainment of $\dot{V}O_2$ peak in normoxia, however further research is warranted to confirm this.

During severe intensity treadmill exercise lasting ~2 min, it has been shown that prior exercise had no effect on the subsequent $\dot{V}O_2$ kinetics (Jones *et al.* 2008; Draper *et al.* 2006). The only factor shown to affect the $\dot{V}O_2$ kinetics to severe intensity treadmill exercise is the aerobic capability of the individual (Draper *et al.* 2008; Draper and Wood, 2005a). Draper *et al.* (2008) and Draper and Wood (2005a) found that individuals with a greater $\dot{V}O_2$ peak achieved a lower percentage of that peak during severe intensity treadmill exercise lasting ~2 min. The findings of the current study further support this, demonstrating that when $\dot{V}O_2$ peak is reduced via the manipulation of FiO_2, individuals who were unable to achieve $\dot{V}O_2$ peak in normoxia were able to achieve $\dot{V}O_2$ peak in hypoxia. Aerobic capability is influenced by training status; however, training will result in central and peripheral adaptations. Therefore, it is unclear as to what mechanism(s) is responsible for the regulation of oxygen during severe intensity exercise, however, the HR peak findings of the current study suggests that the distribution of blood during exercise may play a role.

Despite over a decade of research, no consensus yet exists on the regulation of $\dot{V}O_2$ kinetics, and the arguments presented by Grassi (2001) and Xu and Rhodes (1999) are still being debated. However, it has recently been proposed that the mechanism(s) responsible for the $\dot{V}O_2$ kinetics; whether it is limited by supply or utilisation may not be independent (Poole *et al.* 2008). Instead, there may be a complex interaction of cardiovascular and metabolic factors that regulate $\dot{V}O_2$ kinetics (Poole *et al.* 2008; Jones and Poole, 2005).

5.7 Practical implications

The findings of the current thesis, and that of the literature (Draper *et al.* 2008; James *et al.* 2008; James *et al.* 2007; 2007a; Draper *et al.* 2006; Sandals *et al.* 2006; Draper and Wood, 2005; 2005a) have found that when performing severe intensity

treadmill exercise to exhaustion (lasting ~2 min) $\dot{V}O_2$ peak is not achieved, instead achieving ~88%. Moreover, it has been found that participants with the greatest peak aerobic power achieve a lower percentage of this peak during an effort of this kind (Draper *et al.* 2008; James *et al.* 2007a; Draper and Wood, 2005a). The duration and intensity of the exercise tests used in these studies are similar to that of 800-m running performance. Therefore, the findings have important training implications for an 800-m athlete, highlighting that peak aerobic power is unattainable thus stressing the importance of the anaerobic contribution as a key performance determinant among athletes with a similar peak aerobic power.

CHAPTER 6

CONCLUSION

It was found that the $\dot{V}O_2$ peak achieved during the ramp tests was greater in normoxia than hypoxia. It was also found that when the time to exhaustion during severe intensity treadmill exercise was matched between conditions; 1) the percentage of $\dot{V}O_2$ peak achieved was greater in hypoxia than normoxia (when determined by the ramp test in the respective condition), 2) the percentage of normoxic ramp $\dot{V}O_2$ peak was greater during the N_square than the H_square tests. The linear regression analysis of normoxic $\dot{V}O_2$ peak and the percentage achieved during the N_square tests demonstrated that individuals with a greater $\dot{V}O_2$ peak achieved a lower percentage of that peak. The HR peak was different between conditions, but demonstrated no differences between tests. The parameter estimates for τ were greater during the H_square tests whilst no difference was observed in δ between conditions. This resulted in greater MRT in the hypoxic condition. The bootstrap standard error of estimate demonstrated a good fit to the modelled response to a single transition, moreover a better fit than has previously been reported.

The findings of the current thesis demonstrate that individuals unable to achieve $\dot{V}O_2$ peak in normoxia can achieve $\dot{V}O_2$ in hypoxia, demonstrating that there is sufficient time for the full response to become manifest, questioning the regulatory mechanism(s) of oxygen uptake kinetics during severe intensity treadmill running. Furthermore, it was found that although the proportional energy contribution from the aerobic system was increased during the H_square tests, in absolute terms the contribution was less. In addition, the $\dot{V}O_2$ kinetics were slower during the square-wave exercise tests in hypoxia, thus increasing the anaerobic contribution to exercise onset compared to exercise in normoxia. This finding suggests that performance of the H_square tests were maintained via an increased contribution from the anaerobic system, questioning whether anaerobic capacity is fully exhausted during the N_square tests and/or whether there is a finite anaerobic capacity.

Many researchers have investigated the effect of hypoxia on aerobic capability, and also the effect of hypoxia on sprint performance. However, the effect of hypoxia on

middle-distance performance has received little attention. The present study was the first to explore whether a group of highly aerobically trained individuals, that were unable to achieve $\dot{V}O_2$ peak during severe intensity treadmill exercise (lasting ~2 min) in normoxia, were able to achieve $\dot{V}O_2$ peak in hypoxia. This study was the first to match the square-wave test duration between conditions, and therefore was the first to determine the differences in aerobic energy contribution to exhaustive severe intensity treadmill exercise (lasting ~2 min) in normoxia and hypoxia. These findings have presented important questions regarding the physiological control mechanism(s) for $\dot{V}O_2$ kinetics, and can also provide key information to coaches/athletes regarding training practices for the middle-distance events.

In conclusion, the present study found the $\dot{V}O_2$ peak achieved during the ramp test was significantly reduced in hypoxia, compared to normoxia. The percentage of $\dot{V}O_2$ peak achieved (when determined by the ramp test in the respective condition) was greater during the H_square than the N_square tests. Moreover, when the relative exercise intensity was matched between the conditions, athletes unable to achieve their $\dot{V}O_2$ peak in normoxia, were able to achieve $\dot{V}O_2$ peak in hypoxia. HR peak was significantly different between condition, but not test, being greater in normoxia than hypoxia. The findings demonstrate that there is sufficient time for $\dot{V}O_2$ peak to be achieved, and suggest that cardiac output, as suggested from HR peak, is not constraining the $\dot{V}O_2$ response.

References

Abbiss, C., Laursen, P. (2008). Describing and Understanding Pacing Strategies During Athletic Competition. *Sports Medicine,* 38(3), 239-252.

Adami, A., Pogliaghi, S., De Roia, G., and Capelli, C. (2011). Oxygen Uptake, Cardiac Output and Muscle Deoxygenation at the Onset of Moderate and Supramaximal Exercise in Humans. *European Journal of Applied Physiology,* 111(7), 1517-1527.

Aldemir, H., Atkinson, G., Cable, T., Edwards, B., Waterhouse, J., and Reilly, T. (2000). A Comparison of the Immediate Effects of Moderate Exercise in the Early Morning and Late Afternoon on Core Temperature and Cutaneous Thermoregulatory Mechanisms. *Chronobiology International,* 17(2), 197-207.

Barstow, T. (1994). Characterisation of $\dot{V}O_2$ Kinetics During Heavy Exercise. *Medicine and Science in Sports and Exercise,* 26(11), 1327-1334.

Barstow, T., Jones, A., Nguyen, P., and Casaburi, R. (1996). Influence of Muscle Fibre Type and Pedal Frequency on Oxygen Uptake Kinetics of Heavy Exercise. *Journal of Applied Physiology,* 81, 1642-1650.

Behnke, B., McDonough, P., Padilla, D., Musch, T., and Poole, D. (2003). Oxygen Exchange Profile in Rat Muscles of Contrasting Fibre Types. *Journal of Physiology,* 549(2), 597-605.

Bell, C., Paterson, D., Kowalchuk, J., Padilla, J., and Cunningham, D. (2001). A Comparison of Modelling Techniques Used to Characterise Oxygen Uptake Kinetics During the On-Transient of Exercise. *Experimental Physiology,* 86(5), 667-676.

Berger, N., Tolfrey, K., Williams, A., and Jones, A. (2006). Influence of Continuous and Interval Training on Oxygen Uptake On-Kinetics. Medicine and Science in Sports and Exercise, 38(3), 504-512.

Billat, V., Hamard, L., Koralsztein, J-P., and Morton, H. (2009). Differential Modelling of Anaerobic and Aerobic Metabolism in the 800-m and 1500-m run. *Journal of Applied Physiology,* 107, 478-487.

Billat, V., Richard, R., Binsse, V., Koralsztein, J., and Haouzi, P. (1998). The $\dot{V}O_2$ Slow Component For Severe Exericse Depends on Type of Exercise and is not Correlated With Time to Fatigue. *Journal of Applied Physiology,* 85(6), 2118-2124.

Bucheit, M., Laursen, P., Ahmaidi, S. (2009). Effect of Prior Exercise on Pulmonary O_2 Uptake and Estimated Muscle Capillary Blood Flow Kinetics During Moderate-Intensity Field Running in Men. *Journal of Applied Physiology,* 107, 460-470.

Buchfuhrer, M., Hansen, J., Robinson, T., Sue, D., Wasserman, K., and Whipp, B. (1983). Optimising the Exercise Protocol for Cardiopulmonary assessment. *Journal of Applied Physiology*, 55, 1558-1564.

Burnley, M., and Jones, A. (2007). Oxygen uptake kinetics as a determinant of sports performance. *European Journal of Applied Physiology*, 7(2), 63-79.

Burnley, M., Doust, J., Ball, D., and Jones, A. (2002). Effects of Prior Heavy Exercise on $\dot{V}O_2$ Kinetics During Heavy Exercise are Related to Changes in Muscle Activity. *Journal of Applied Physiology,* 93, 167-174.

Burnley, M., Jones, A., Carter, H., and Doust, J. (2000). Effects of Prior Heavy Exercise on Phase II Pulmonary Oxygen Uptake Kinetics During Heavy Exercise. *Journal of Applied Physiology,* 89, 1387-1396.

Calbet, J., De Paz, J., Garatachea, N., Cabeza De Vaca, S., and Chavarren, J. (2003). Anaerobic Energy Provision Does Not Limit Wingate Exercise Performance in Endurance Trained Cyclists. *Journal of Applied Physiology*, 94, 668-676.

Carter, H., Jones, A., Barstow, T., Burnley, M., Williams, C., and Doust, J. (2000). Oxygen Uptake Kinetics in Treadmill Running and Cycle Ergometry: a Comparison. *Journal of Applied Physiology*, 89, 899-907.

Carter, H., Jones, A., Barstow, T., Burnley, M., Williams, C., and Doust, J. (2000a). Effect of Endurance Training on Oxygen Uptake Kinetics During Treadmill Running. *Journal of Applied Physiology,* 89, 1744-1752.

Cleuziou, C., Perrey, S., Borrani, F., Lecoq, A., Courteix, D., Germain, P., and Obert, P. (2004). $\dot{V}O_2$ and EMG Activity Kinetics During Moderate and Severe Constant Work Rate Exercise in Trained Cyclists. *Canadian Journal of Applied Physiology*, 29(6), 758-772.

Craig, I., and Morgan, D. (1998). Relationship Between 800-m Running Performance and Accumulated Oxygen Deficit in Middle-Distance Runners. *Medicine and Science in Sports and Exercise*, 30(11), 1631-1636.

Delp, M., and O'Leary, D. (2004). Integrative Control of the Skeletal Muscle Microcirculation in the Maintenance of Arterial Pressure During Exercise. *Journal of Applied Physiology,* 97, 1112-1118.

Doyon, K., Perrey, S., Ade, D., and Hughson, R. (2001). Field Testing of $\dot{V}O_2$peak in Cross-Country Skiers with Portable Breath-by-Breath System. *Canadian Journal of Applied Physiology*, 26(1), 1-11.

Draper, S., and Wood, D. (2004). Breath-by-Breath 'Noise' and Parameter Estimation for $\dot{V}O_2$ Kinetics in Severe Intensity Running. *Ninth Annual Congress of European College of Sport Science,* Clermont-Ferrand 3rd-6th July.

Draper, S., and Wood, D. (2005). The Oxygen Uptake Response of Sprint- vs. Endurance Trained Runners to Severe Intensity Running. *Journal of Science and Medicine in Sport,* 8(2), 233-243.

Draper, S., and Wood, D. (2005a). The $\dot{V}O_2$ Response for an Exhaustive Treadmill Run at 800-m Pace: a Breath-by-Breath Analysis. *European Journal of Applied Physiology,* 93(4), 381-389.

Draper, S., Hopkinson, Y., Potter, C., and James, D. (2008). Influence of $\dot{V}O_2$max on the %VO_2max Attained and $\dot{V}O_2$ Kinetics in Short Duration Exhaustive Running. *Thirteenth Annual Congress of European College of Sport Science.* Estoril 9th-12th July.

Draper, S., Wood, D., Corbett, J., James, D., and Potter, C. (2006). The Effect of Prior Moderate and Heavy Intensity Running on the $\dot{V}O_2$ Response to Severe Intensity Running. *International Journal of Sports Physiology and Performance, 1, 359-372.*

Duffield, R., Dawson, B., and Goodman, C. (2005). Energy System Contribution to 400-metre and 800-metre Track Running. *Journal of Sports Sciences,* 23(3), 299-307.

Duffield, R., Dawson, B., Pinnington, H., and Wong, P. (2004). Accuracy and Reliability of a Cosmed K4b2 Portable Gas Analysis System. *Journal of Science and Medicine in Sport,* 7(1), 11-22.

Duffy, M., and Jacobsen, B. (2001). Univariate Descriptive Statistics. In: Munro, B (ed.) *Statistical Methods for Health Care Research.* Philadelphia: Lippincott Williams & Wilkins, 29-62.

Engelen, M., Porszasz, J., Riley, M., Wasserman, K., Maehara, K., and Barstow, T. (1996). Effects of Hypoxic Hypoxia on O_2 Uptake and Heart Rate Kinetics During Heavy Exercise. *Journal of Applied Physiology,* 81(6), 2500-2508.

Faisal, A., Beavers, K., Robertson, A., and Hughson, R. (2009). Prior Moderate and Heavy Exercise Accelerate Oxygen Uptake and Cardiac Output Kinetics in Endurance Athletes. *Journal of Applied Physiology,* 106, 1553-1563.

Friedmann, B., Frese, F., Menold, E., and Bartsch, P. (2007). Effects of Acute Moderate Hypoxia on Anaerobic Capactiy in Endurance-Trained Runners. *European Journal of Applied Physiology,* 101, 67-73.

Gaesser, G., and Poole, D. (1996). The Slow Component of Oxygen Uptake Kinetics in Humans. *Exercise and Sport Sciences Reviews,* 24(1), 35-70.

Gerbino, A., Ward, S., and Whipp, B. (1996). Effects of Prior Exercise on Pulmonary Gas-Exchange Kinetics During High-Intensity Exercise in Humans. *Journal of Applied Physiology,* 80(1), 99-107.

Gore, C., Little, S., Hahn, A., Scroop, G., Norton, K., Bourdon, P., Woolford, S., Buckley, J., Stanef, T., Campbell, D., Watson, D., and Emonson, D. (1997). Reduced Performance of Male and Female Athletes at 580 m Altitude. *European Journal of Applied Physiology,* 75, 136-143.

Grassi, B. (2001). Regulation of Oxygen Consumption at Exercise Onset: Is It Really Controversial? *Exercise and Sport Science Review,* 29(3), 134-138.

Grassi, B., Poole, D., Richardson, R., Knight, D., Erickson, B., and Wagner, P. (1996). Muscle O_2 Uptake Kinetics in Humans: Implications for Metabolic Control. *Journal of Applied Physiology,* 80(3), 988-998.

Gurd, B., Peter, S., Heigenhauser, G., LeBlanc, P., Doherty, T., Paterson, D., and Kowalchuk, J. (2006). Prior Heavy Exercise Elevatees Pyruvate Dehydrogenase Activity and Speeds O_2 Uptake Kinetics During Subsequent Moderate-Intensity Exercise in Healthy Young Adults. *Journal of Applied Physiology,* 577(3), 985-996.

Gurd, B., Scheurmann, B., Paterson, D., Kowalchuk, J. (2005). Prior Heavy-Intensity Exercise Speeds $\dot{V}O_2$ Kinetics During Moderate-Intensity Exercise in Young Adults. *Journal of Applied Physiology,* 98, 1371-1378.

Haseler, L., Lin, A., Hoff, J., and Richardson, R. (2007). Oxygen Availability and PCr Recovery Rate in Untrained Human Calf Muscle: Evidence of Metabolic Limitation in Normoxia. *American Journal of Physiology - Regulative, Integrative and Comparative Physiology,* 293, 2046-2051.

Hill, A., and Lupton, H. (1923). Muscular Exercise, Lactic Acid and the Supply and Utilization of Oxygen. *The Quartely Journal of Medicine,* 16, 135-171.

Hill, D. (1999). Energy System Contributions in Middle-Distance Running Events. *Journal of Sports Sciences,* 17, 477-483.

Hill, D., and Ferguson, C. (1999). A Physiological Description of Critical Velocity. *European Journal of Applied Physiology,* 79(3), 290-293.

Hill, D., Halcomb, J., and Stevens, E. (2003). Oxygen Uptake Kinetics During Severe Intensity Running and Cycling. *European Journal of Applied Physiology,* 89, 612-618.

Horowitz, J., Sidossis, L., and Coyle, E. (1994). High Efficiency of Type I Muscle Fibres Improves Performance, *International Journal of Sports Medicine,* 15(3), 152-157.

Hughson, R. (2009). Oxygen Uptake Kinetics: Historical Perspective and Future Directions. *Applied Physiology, Nutrition, and Metabolism,* 34(5), 840-850.

Hughson, R., and Kowalchuk, J. (1995). Kinetics of Oxygen Uptake for Submaximal Exercise in Hyperoxia, Normoxia, and Hypoxia. *Canadian Journal of Applied Physiology,* 20(2), 198-210.

Ingham, S., Carter, H., Whyte, G., and Doust, J. (2007). Comparison of the Oxygen Uptake Kinetics of Club and Olympic Champion Rowers. *Medicine and Science in Sports and Exercise,* 39(5), 865-871.

James, D., Sandals, L., Draper, S., and Wood, D. (2007a). Relationship between Maximal Oxygen Uptake Attained During Treadmill Middle-Distance Running. *Journal of Sports Sciences,* 25(8), 851-858.

James, D., Sandals, L., Draper, S., Maldondo-Martin, S., and Wood, D. (2007). $\dot{V}O_2$ Attained During Treadmill Running: The Influence of a Specialist (400-m or 800-m) Event. *International Journal of Sports Physiology and Performance,* 2, 128-136.

James, D., Sandals, L., Draper, S., Maldondo-Martin, S., and Wood, D. (2008). Influence of Test Duration on Oxygen Uptake Attained During Treadmill Running. *European Journal of Sport Science,* 8(4), 225-230.

Jones, A., and Doust, J. (1996). A 1% Treadmill Grade Most Accurately Reflects the Energetic Cost of Outdoor Running. *Journal of Sports Sciences,* 14, 321-327.

Jones, A., and Poole, D. (2005). *Oxygen Uptake Kinetics in Sport, Exercise and Medicine,* Oxon: Routledge.

Jones, A., DiMenna, F., Lothian, F., Taylor, E., Garland, S., Hayes, P., and Thompson, K. (2008). 'Priming' Exercise and O_2 Uptake Kinetics During Treadmill Running. *Respiratory Physiology and Neurobiology,* 161, 182-188.

Jones, A., Wilkerson, D., Burnley, M., and Koppo, K. (2003). Prior Heavy Exercise Enhanves Performance During Subsequent Perimaximal Exercise. *Medicine and Science in Sports and Exercise,* 35(12), 2085-2092.

Jones, A., Wilkerson, D., Vanhatalo, A., Burnley, M. (2008). Influence of Pacing Strategy on O_2 Uptake and Exercise Tolerance. *Scandinavian Journal of Medicine and Science in Sports,* 18(5), 615-626.

Kilding, A., Winter, E., and Fysh, M. (2006). Comparison of Pulmonary Oxygen Uptake Kinetics in Middle- and Long-Distance Runners. *International Journal of Sports Medicine,* 27, 419-426.

Koppo, K., Bouckaert, J., and Jones, A. (2004). Effects of Training Status and Exercise Intensity on Phase II $\dot{V}O_2$ Kinetics. *Medicine and Science in Sports Exercise,* 36(2), 225-232.

Lamarra, N., Whipp, B., Ward, S., and Wasserman, K. (1987). Effect of Interbreath Fluctuations on Charactrising Exercise Gas Exchange Kinetics. *Journal of Applied Physiology,* 62, 2003-2012.

Linnarsson, D., Karlsson, J., Fagraeus, L., and Saltin, B. (1974). Muscle Metabolites and Oxygen Deficit with Exercise in Hypoxia and Hyperoxia. *Journal of Applied Physiology,* 36(4), 399-402.

Lucia, A., Hoyos, J., Santalla, A., Perez, M., and Chicharro, J. (2002). Kinetics of $\dot{V}O_2$ in Professional Cyclists. *Medicine and Science in Sports and Exercise*, 34(2), 320-325.

MacDonald, M., Pedersen, P., and Hughson, R. (1997). Acceleration of $\dot{V}O_2$ Kinetics in Heavy Submaximal Exercise by Hyperoxia and Prior High-Intensity Exercise. *Journal of Applied Physiology,* 83(4), 1318-1325.

Macfarlane, D. (2001). Automated Metabolic Gas Analysis Systems: A Review. *Sports Medicine,* 31(12), 841-861.

Marwood, S., Roche, D., Rowland, T., Garrard, M., and Unnithan, V. (2010). Faster Pulmonary Oxygen Uptake Kinetics in Trained Versus Untrained Male Adolescents. *Medicine and Science in Sports and Exercise*, 42(1), 127-134.

McArdle, W., Katch, F., and Katch, V. (2007). *Exercise Physiology: Energy, Nutrition, and Human Performance.* Philadelphia: Lippincott Williams & Wilkins.

McLaughlin, J., King, G., Howley, E., Bassett, D., and Ainsworth, B. (2001). Validation of the Cosmed K4b2 Portable Metabolic System. *International Journal of Sports Medicine,* 22, 280-284.

McLellan, T., Kavanagh, M., and Jacobs, I. (1990). The Effect of Hypoxia on Performance During 30s or 45s of Supramaximal Exercise. *European Journal of Applied Physiology,* 60, 155-161.

Mortensen, S., Damsgaard, R., Dawson, E., Secher, N., and Gonzalez-Alonso, J. (2008). Restrictions in Systemic and Locomotor Skeletal Muscle Perfusion, Oxygen Supply and $\dot{V}O_2$ During High-Intensity Whole-Body Exercise in Humans. *Journal of Physiology*, 586(10), 2621-2635.

Ogawa, T., Ohba, K., Nabekura, Y., Nagai, J., Hayashi, K., Wada, H., and Nishiyasu, T. (2005). Intermittent Short-Term Graded Running Performance in Middle-Distance Runners in Hypobaric Hypoxia. *European Journal of Applied Physiology*, 94, 254-261.

Ogura, Y., Katamoto, S., Uchimaru, J., Takahashi, K., and Naito, H. (2006). Effects of Low and High Levels of Moderate Hypoxia on Anaerobic Energy Release During Supramaximal Cycle Exercise. *European Journal of Applied Physiology*, 98, 41-47.

Pinnington, H., Wong, P., Tay, J., Green, D., and Dawson, B. (2001). The Level of Accuracy and Agreement in Measures of FEO_2, $FECO_2$ and $\dot{V}E$ Between the Cosmed K4b2 Portable, Respiratory Gas Analysis System and a Metabolic Cart. *Journal of Science and Medicine in Sport*, 4(3), 324-335.

Poole, D., and Richardson, R. (1997). Determinants of Oxygen Uptake. Implications for Exercise Testing. *Sports Medicine,* 24(5), 308-320.

Poole, D., Barstow, T., McDonough, P., and Jones, A. (2008). Control of Oxygen Uptake During Exercise. *Medicine and Science in Sports and Exercise*, 40(3), 462-474.

Pringle, S., Doust, J., Carter, H., Tolfrey, K., Campbell, I., and Jones, A. (2003). Oxygen Uptake Kinetics During Moderate, Heavy and Severe Intensity 'Submaximal' Exercise in Humans: the Influence of Muscle Fibre Type and Capilliarisation. *European Journal of Applied Physiology*, 89, 289-300.

Sandals, L., Wood, D., Draper, S., and James, D. (2006). Influence of Pacing Strategy on Oxygen Uptake During Treadmill Middle-Distance Running. *International Journal of Sports Medicine,* 27, 37-42.

Simmonds, M., Minahan, C., and Sabapathy, S. (2010). Caffeine Improves Supramaximal Cycling but not the Rate of Anaerobic Energy Release. *European Journal of Applied Physiology,* 109(2), 287-295.

Sloniger, M., Cureton, K., Prior, B., and Evans, E. (1997). Lower Extremity Muscle Activation During Horizontal and Uphill Running. *Journal of Applied Physiology,* 83(6), 2073-2079.

Sloniger, M., Cureton, K., Prior, B., and Evans, E. (1997a). Anaerobic Capacity and Muscle Activation During Horizontal and Uphill Running. *Journal of Applied Physiology*, 83(1), 262-269.

Spencer, M., and Gastin, P. (2001). Energy System Contribution During 200- to 1500-m Running in Highly Trained Athletes. *Medicine and Science in Sports and Exercise*, 33(1), 157-162.

Thomas, C., Hanon, C., Perrey, S., Le Chevalier, J., Couturier, A., and Vandewalle, H. (2005). Oxygen Uptake Response to an 800-m Running Race. *International Journal of Sports Medicine*, 26, 268-273.

Van Ingen Schenau, G., Bobbert, M., and De Haan, A. (1997). Does Elastic Energy Enhance Work and Efficiency in the Stretch-Shortening Cycle? *Journal of Applied Biomechanics*, 13, 389-415.

Weyand, P., Lee, C., Martinez-Ruiz, R., Bundle, M., Bellizzi, M., and Wright, S. (1999). High-Speed Running Performance is Largely Unaffected by Hypoxic Reductions in Aerobic Power. *Journal of Applied Physiology*, 86(6), 2059-2064.

Whipp, B. (1994). The Slow Component of O_2 Uptake Kinetics During Heavy Exercise. *Medicine and Science in Sports and Exercise*, 26(11), 1319-1326.

Whipp, B. , and Ward, S. (1990). Physiological Determinants of Pulmonary Gas Exchange Kinetics During Exercise. *Medicine and Science in Sports and Exercise*, 22(1), 62-71.

Whipp, B., Davis, J., Torres, F., and Wasserman, K. (1981). A Test to Determine Parameters of Aerobic Function During Exercise. *Journal of Applied Physiology, 50(1), 217-221.*

Wilkerson, D., Berger, N., and Jones, A. (2006). Influence of Hyperoxia on Pulmonary O_2 Uptake Kinetics Following the Onset of Exercise in Humans. *Repiratory Physiology and Neurobiology*, 153, 92-106.

Wilkerson, D., Koppo, K., Barstow, T., Jones, A. (2004). Effect of Prior Multiple-Sprint Exercise on Pulmonary O_2 Uptake Kinetics Following the Onset of Perimaximal Exercise. *Journal of Applied Physiology*, 97, 1227-1236.

Wood, D. (1999). Physiological Demands of Middle-Distance Running. In J. Fallowfield and D.Wilkinson (Eds.) *Improving Sports Performance in Middle and Long-Distance Running: A Scientific Approach to Race Preperation* (pp. 14-39). Chichester: John Wiley & Sons.

Woorons, X., Mollard, P., Lamberto, C., Letournel, M., and Richalet, J-P. (2005). Effect of Acute Hypoxia on Maximal Exercise in Trained and Sedentary Women. *Medicine and Science in Sports and Exercise*, 37(1), 147-154.

Xu, F., and Rhodes, E. (1999). Oxygen Uptake Kinetics During Exercise. *Sports Medicine,* 27(5), 313-327.

Yoshida, T., Yamamoto, K., and Udo, M. (1993). Relationship Between Cardiac Output and Oxygen Uptake at the Onset of Exercise. *European Journal of Applied Physiology*, 66, 155-160.

Appendix 1

Statement of informed consent

UNIVERSITY OF GLOUCESTERSHIRE

SPORT & EXERCISE LABORATORIES

Informed Consent Form

Description of study:

I have had full details of the tests I am about to complete explained to me. I understand the risks and benefits involved, and that I am free to withdraw from the tests at any point. I confirm that I have completed a health questionnaire, and I am in a fit condition to undertake the required exercise.

Name: .....

Signed: .....… Date: .....……

Name of Guardian*: .....…….

Signed*: .....…… Date* .....…….

Tester: .....……

Signed: .....….. Date: .....……

*to be completed only if the participant is under 18 years of age

Appendix 2

Health questionnaire

UNIVERSITY OF GLOUCESTERSHIRE

SPORT & EXERCISE LABORATORIES

Health Questionnaire

About this questionnaire:
The purpose of this questionnaire is to gather information about your health and lifestyle. We will use this information to decide whether you are eligible to take part in the testing for which you have volunteered. It is important that you answer the questions truthfully. The information you give will be treated in confidence. Your completed form will be stored securely for 5 years and then destroyed.

Section 1, which has been completed by the tester, provides basic information about the testing for which you have volunteered. Sections 2 to 7 are for you to complete: please circle the appropriate response or write your answer in the space provided. Please also complete section 8. Sections 9 and 10 will be completed by the tester, after you have completed sections 2 to 8.

Section 1: The testing (completed by tester)

To complete the testing for which you have volunteered you will be required to undertake:

Moderate exercise (i.e., exercise that makes you breathe more heavily than you do at rest but not so heavily that you are unable to maintain a conversation) ☐

Vigorous exercise (i.e., exercise that makes you breath so heavily that you are unable to maintain a conversation) ☐

The testing involves:

Walking	☐	Generating or absorbing high forces through your arms	☐
Running	☐	Generating or absorbing high forces through your shoulders	☐
Cycling	☐	Generating or absorbing high forces through your trunk	☐
Rowing	☐	Generating or absorbing high forces through your hips	☐
Swimming	☐	Generating or absorbing high forces through your legs	☐
Jumping	☐		

Section 2: General information

Name: ..... Sex: M F Age:

Height (approx.): ..... Weight (approx.):

Section 3: Initial considerations

1. Do any of the following apply to you? No Yes

a) I have HIV, Hepatitis A, Hepatitis B or Hepatitis C
b) I am pregnant
c) I have a muscle or joint problem that could be aggravated by the testing described in section 1

d) I am feeling unwell today
e) I have had a fever in the last 7 days

(If you have answered "Yes" to question 1, go straight to section 8)

Section 4: Habitual physical activity

2a. Do you typically perform moderate exercise (as defined in section 1) for 20 minutes or longer at least twice a week? No Yes

2b. Have you performed this type of exercise within the last 10 days? No Yes

3a. Do you typically perform vigorous exercise (as defined in section 1) at least once a week? No Yes

3b. Have you performed this type of exercise within the last 10 days? No Yes

Section 5: Known medical conditions

4. Do **any** of the following apply to you? No Yes

   a) I have had insulin-dependent diabetes for more than 15 years
   b) I have insulin-dependent diabetes and am over 30 years old
   c) I have non-insulin-dependent diabetes and am over 35 years old

5. Have you ever had a stroke? No Yes

6. Has your doctor ever said you have heart trouble? No Yes

7. Do **both** of the following apply to you? No Yes

   a) I take asthma medication

   b) I have experienced shortness of breath or difficulty with breathing in the last 4 weeks?

8. Do you have any of the following: cancer, COPD, cystic fibrosis, other lung disease, liver disease, kidney disease, mental illness, osteoporosis, severe arthritis, a thyroid problem? No Yes

(If you have answered "Yes" to any questions in section 5, go straight to section 8.)

Section 6: Signs and symptoms

9. Do you often have pains in your heart, chest, or the surrounding areas? No Yes

10. Do you experience shortness of breath, either at rest or with mild exertion? No Yes

11. Do you often feel faint or have spells of severe dizziness? No Yes

12. Have you, in the last 12 months, experienced difficulty with breathing when lying down or been awakened at night by shortness of breath? No Yes

13. Do you experience swelling or a build up of fluid in or around your ankles? No Yes

14. Do you often get the feeling that your heart is racing or skipping beats, either at rest or during exercise? No Yes

15. Do you regularly get pains in your calves and lower legs during exercise that are not due to soreness or stiffness? No Yes

16. Has your doctor ever told you that you have a heart murmur? No Yes

17. Do you experience unusual fatigue or shortness of breath during everyday activities? No Yes

(If you have answered "Yes" to any questions in section 6, go straight to section 8.)

Section 7: Risk factors

18. Does **either** of the following apply to you? No Yes

a) I smoke cigarettes on a daily basis
b) I stopped smoking cigarettes on a daily basis less than 6 months ago

19. Has your doctor ever told you that you have high blood pressure? No Yes
20. Has your doctor ever told you that you have high cholesterol? No Yes
21. Has your father or any of your brothers had a heart attack, heart surgery, or a stroke before the age of 55? No Yes

22. Has your mother or any of your sisters had a heart attack, heart surgery, or a stroke before the age of 65? No Yes

23. Do **any** of the following apply to you? No Yes

a) I have had insulin-dependent diabetes for less than 15 years
b) I have insulin-dependent diabetes and am 30 or younger
c) I have non-insulin-dependent diabetes and am 35 or younger

Section 8: Signatures

Participant: …….. Date: .....…….....

Guardian*: .....……..... Date: .....…….....

(*Required only if the participant is under 18 years of age.)

Section 9: Additional risk factors (to be completed by the tester if relevant)

24. Is the participant's body mass index >30 kg/m^2? No Yes

25. Has the participant answered no to questions 2a **and** 3a? No Yes

Section 10: Eligibility (to be completed by the tester)

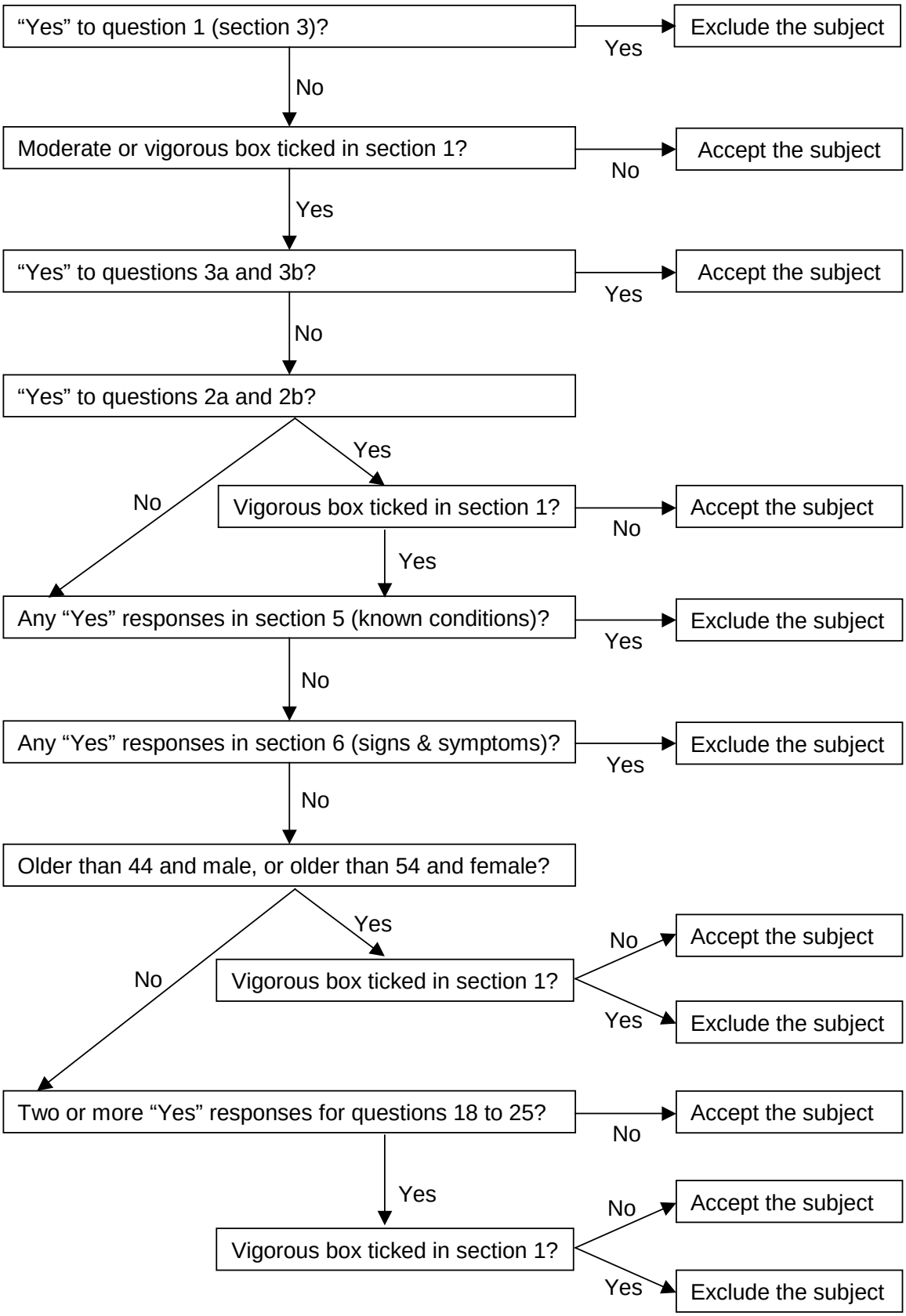
26. Is the participant eligible for the testing? No Yes

Name (of tester): .....……..……

Signature: ……. Date: ……...……..

Appendix 3

Processing the completed questionnaire



Appendix 4

The speed data for the square-wave tests in both conditions

Participant	N_Square	H_Square
1	22	21
2	22	20.5
3	21	19.5
4	24	23
5	23	22
6	21.5	20
7	21	20
8	21	19.5
9	21	20
10	20	19
11	23	21.5
12	22.5	21.5
13	22	21
14	22	21
Mean	21.9	20.6
SD	1.0	1.1

Appendix 5

SPSS Output

SPSS Output – VO_2 ANOVA

Within-Subjects Factors

Measure:MEASURE_1

test	condition	Dependent Variable
1	1	NS_VO2
	2	HS_VO2
2	1	NRamp_VO2
	2	HRamp_VO2

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
test	Pillai's Trace	.752	39.393[a]	1.000	13.000	.000
	Wilks' Lambda	.248	39.393[a]	1.000	13.000	.000
	Hotelling's Trace	3.030	39.393[a]	1.000	13.000	.000
	Roy's Largest Root	3.030	39.393[a]	1.000	13.000	.000
condition	Pillai's Trace	.939	2.008E2[a]	1.000	13.000	.000
	Wilks' Lambda	.061	2.008E2[a]	1.000	13.000	.000
	Hotelling's Trace	15.443	2.008E2[a]	1.000	13.000	.000
	Roy's Largest Root	15.443	2.008E2[a]	1.000	13.000	.000
test * condition	Pillai's Trace	.761	41.298[a]	1.000	13.000	.000
	Wilks' Lambda	.239	41.298[a]	1.000	13.000	.000
	Hotelling's Trace	3.177	41.298[a]	1.000	13.000	.000
	Roy's Largest Root	3.177	41.298[a]	1.000	13.000	.000

a. Exact statistic

b. Design: Intercept

Within Subjects Design: test + condition + test * condition

Tests of Within-Subjects Effects

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
test	Sphericity Assumed	1147433.143	1	1147433.143	39.393	.000
	Greenhouse-Geisser	1147433.143	1.000	1147433.143	39.393	.000
	Huynh-Feldt	1147433.143	1.000	1147433.143	39.393	.000
	Lower-bound	1147433.143	1.000	1147433.143	39.393	.000
Error(test)	Sphericity Assumed	378666.357	13	29128.181		
	Greenhouse-Geisser	378666.357	13.000	29128.181		
	Huynh-Feldt	378666.357	13.000	29128.181		
	Lower-bound	378666.357	13.000	29128.181		
condition	Sphericity Assumed	1.703E7	1	1.703E7	200.764	.000
	Greenhouse-Geisser	1.703E7	1.000	1.703E7	200.764	.000
	Huynh-Feldt	1.703E7	1.000	1.703E7	200.764	.000
	Lower-bound	1.703E7	1.000	1.703E7	200.764	.000
Error(condition)	Sphericity Assumed	1102616.214	13	84816.632		
	Greenhouse-Geisser	1102616.214	13.000	84816.632		
	Huynh-Feldt	1102616.214	13.000	84816.632		
	Lower-bound	1102616.214	13.000	84816.632		
test * condition	Sphericity Assumed	1537165.786	1	1537165.786	41.298	.000
	Greenhouse-Geisser	1537165.786	1.000	1537165.786	41.298	.000
	Huynh-Feldt	1537165.786	1.000	1537165.786	41.298	.000
	Lower-bound	1537165.786	1.000	1537165.786	41.298	.000
Error(test*condition)	Sphericity Assumed	483878.714	13	37221.440		
	Greenhouse-Geisser	483878.714	13.000	37221.440		
	Huynh-Feldt	483878.714	13.000	37221.440		
	Lower-bound	483878.714	13.000	37221.440		

VO_2peak achieved in the square-wave exercise tests relative to the VO_2peak in the respective condition

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	.8592	14	.06126	.01637
	h	1.0173	14	.07622	.02037

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 n - h	-.15805	.10391	.02777	-.21805	-.09805	-5.691	13	.000

Percentage of N_ramp VO_2peak achieved during the square-wave exercise tests

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	.8592	14	.06126	.01637
	h	1.0173	14	.07622	.02037

Paired Samples Test

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1	n - h	-.15805	.10391	.02777	-.21805	-.09805	-5.691	13	.000

Relationship between N_ramp VO_2peak and the percentage achieved during N_square tests

Correlations

		ramp	square
ramp	Pearson Correlation	1.000	-.637*
	Sig. (2-tailed)		.014
	N	14	14
square	Pearson Correlation	-.637*	1.000
	Sig. (2-tailed)	.014	
	N	14	14

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

HRpeak ANOVA

Within-Subjects Factors

Measure:MEASURE_1

test	condition	Dependent Variable
1	1	NS_HR
	2	HS_HR
2	1	NRamp_HR
	2	HRamp_HR

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
test	Pillai's Trace	.087	1.245[a]	1.000	13.000	.285
	Wilks' Lambda	.913	1.245[a]	1.000	13.000	.285
	Hotelling's Trace	.096	1.245[a]	1.000	13.000	.285
	Roy's Largest Root	.096	1.245[a]	1.000	13.000	.285
condition	Pillai's Trace	.756	40.356[a]	1.000	13.000	.000
	Wilks' Lambda	.244	40.356[a]	1.000	13.000	.000
	Hotelling's Trace	3.104	40.356[a]	1.000	13.000	.000
	Roy's Largest Root	3.104	40.356[a]	1.000	13.000	.000
test * condition	Pillai's Trace	.318	6.062[a]	1.000	13.000	.029
	Wilks' Lambda	.682	6.062[a]	1.000	13.000	.029
	Hotelling's Trace	.466	6.062[a]	1.000	13.000	.029
	Roy's Largest Root	.466	6.062[a]	1.000	13.000	.029

a. Exact statistic

b. Design: Intercept

Within Subjects Design: test + condition + test * condition

Tests of Within-Subjects Effects

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
test	Sphericity Assumed	28.571	1	28.571	1.245	.285
	Greenhouse-Geisser	28.571	1.000	28.571	1.245	.285
	Huynh-Feldt	28.571	1.000	28.571	1.245	.285
	Lower-bound	28.571	1.000	28.571	1.245	.285
Error(test)	Sphericity Assumed	298.429	13	22.956		
	Greenhouse-Geisser	298.429	13.000	22.956		
	Huynh-Feldt	298.429	13.000	22.956		
	Lower-bound	298.429	13.000	22.956		
condition	Sphericity Assumed	480.286	1	480.286	40.356	.000
	Greenhouse-Geisser	480.286	1.000	480.286	40.356	.000
	Huynh-Feldt	480.286	1.000	480.286	40.356	.000
	Lower-bound	480.286	1.000	480.286	40.356	.000
Error(condition)	Sphericity Assumed	154.714	13	11.901		
	Greenhouse-Geisser	154.714	13.000	11.901		
	Huynh-Feldt	154.714	13.000	11.901		
	Lower-bound	154.714	13.000	11.901		
test * condition	Sphericity Assumed	73.143	1	73.143	6.062	.029
	Greenhouse-Geisser	73.143	1.000	73.143	6.062	.029
	Huynh-Feldt	73.143	1.000	73.143	6.062	.029
	Lower-bound	73.143	1.000	73.143	6.062	.029
Error(test*condition)	Sphericity Assumed	156.857	13	12.066		
	Greenhouse-Geisser	156.857	13.000	12.066		
	Huynh-Feldt	156.857	13.000	12.066		
	Lower-bound	156.857	13.000	12.066		

Test duration

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	h	114.0714	14	11.33724	3.03000
	n	113.7143	14	5.49725	1.46920

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	h - n	.35714	8.20513	2.19291	-4.38036	5.09464	.163	13	.873

Parameter estimates and Bootstrap standard error of estimates for the Square-wave exercise tests

Participant 1

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3289.531	19.967	3249.921	3329.141		
	tau	13.113	.689	11.747	14.480		
	delta	11.527	.524	10.487	12.568		
Bootstrap[a]	asymptote	2202.936	5.296	2192.339	2213.532	2193.759	2211.586
	tau	8.071	.567	6.935	9.206	6.588	8.592
	delta	11.254	.928	9.398	13.111	10.516	13.868

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2234.061	4.811	2224.512	2243.610		
	tau	12.162	.322	11.524	12.801		
	delta	7.701	.336	7.035	8.367		
Bootstrap[a]	asymptote	2234.061	5.162	2223.733	2244.390	2224.315	2242.365
	tau	12.162	.355	11.451	12.873	11.349	12.737
	delta	7.701	.403	6.895	8.507	7.175	8.544

a. Based on 60 samples.

Participant 2

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3772.587	5.686	3761.293	3783.881		
	tau	10.239	.193	9.857	10.622		
	delta	9.415	.194	9.029	9.801		
Bootstrap[a]	asymptote	3772.587	5.307	3761.967	3783.207	3761.242	3782.674
	tau	10.239	.319	9.601	10.878	9.544	10.712
	delta	9.415	.385	8.644	10.186	8.880	10.362

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2174.213	7.599	2159.114	2189.313		
	tau	10.373	.280	9.817	10.929		
	delta	14.099	.179	13.744	14.454		
Bootstrap[a]	asymptote	2174.213	5.675	2162.857	2185.570	2163.255	2185.534
	tau	10.373	.494	9.385	11.361	9.312	11.123
	delta	14.099	.335	13.428	14.770	13.736	14.810

a. Based on 60 samples.

Participant 3

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3421.849	3.069	3415.755	3427.943		
	tau	11.720	.141	11.439	12.001		
	delta	6.925	.159	6.610	7.240		
Bootstrap[a]	asymptote	3421.849	2.402	3417.042	3426.656	3417.686	3426.450
	tau	11.720	.223	11.273	12.168	11.043	12.000
	delta	6.925	.313	6.298	7.552	6.559	7.939

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2521.649	8.618	2504.574	2538.724		
	tau	19.847	.579	18.699	20.995		
	delta	5.853	.500	4.862	6.844		
Bootstrap[a]	asymptote	2521.649	10.196	2501.246	2542.052	2506.826	2543.952
	tau	19.847	.479	18.888	20.805	18.928	21.037
	delta	5.853	.435	4.983	6.723	4.754	6.667

a. Based on 60 samples.

Participant 4

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2989.369	3.409	2982.593	2996.145		
	tau	8.710	.274	8.165	9.255		
	delta	3.920	.461	3.003	4.836		
Bootstrap[a]	asymptote	2989.369	3.944	2981.477	2997.262	2982.371	2998.522
	tau	8.710	.380	7.950	9.471	7.950	9.699
	delta	3.920	.725	2.468	5.371	2.023	5.011

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	1989.827	6.881	1976.153	2003.501		
	tau	9.284	.414	8.461	10.108		
	delta	9.969	.419	9.137	10.800		
Bootstrap[a]	asymptote	1989.827	4.690	1980.444	1999.211	1978.872	1997.537
	tau	9.284	.629	8.025	10.544	7.498	9.883
	delta	9.969	.948	8.071	11.866	9.060	12.163

a. Based on 60 samples.

Participant 5

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3581.630	7.743	3566.258	3597.001		
	tau	12.776	.351	12.080	13.471		
	delta	6.426	.385	5.662	7.189		
Bootstrap[a]	asymptote	3581.630	9.633	3562.355	3600.905	3558.648	3598.912
	tau	12.776	.492	11.791	13.760	11.720	13.683
	delta	6.426	.557	5.311	7.541	5.620	7.512

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2581.178	7.479	2566.331	2596.025		
	tau	9.616	.442	8.738	10.493		
	delta	8.057	.520	7.026	9.089		
Bootstrap[a]	asymptote	2581.178	9.247	2562.675	2599.681	2564.264	2606.922
	tau	9.616	.302	9.012	10.219	9.071	10.244
	delta	8.057	.279	7.499	8.615	7.411	8.619

a. Based on 60 samples.

Participant 6

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2976.213	4.746	2966.776	2985.651		
	tau	10.441	.241	9.963	10.919		
	delta	7.133	.283	6.570	7.697		
Bootstrap[a]	asymptote	2976.213	5.068	2966.073	2986.354	2966.530	2987.096
	tau	10.441	.262	9.916	10.966	9.862	10.851
	delta	7.133	.365	6.403	7.864	6.613	7.964

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2223.153	6.397	2210.425	2235.882		
	tau	12.801	.381	12.043	13.559		
	delta	7.644	.373	6.901	8.386		
Bootstrap[a]	asymptote	2223.153	8.997	2205.151	2241.156	2199.878	2235.500
	tau	12.801	.431	11.939	13.663	11.325	13.468
	delta	7.644	.401	6.841	8.446	6.907	9.101

a. Based on 60 samples.

Participant 7

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2583.507	3.726	2576.098	2590.916		
	tau	11.086	.175	10.738	11.433		
	delta	9.340	.167	9.008	9.672		
Bootstrap[a]	asymptote	2583.507	3.105	2577.295	2589.719	2578.487	2589.984
	tau	11.086	.203	10.680	11.491	10.631	11.477
	delta	9.340	.201	8.938	9.741	9.057	9.943

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2051.795	5.307	2041.249	2062.341		
	tau	14.863	.368	14.132	15.594		
	delta	7.062	.344	6.378	7.746		
Bootstrap[a]	asymptote	2051.795	4.890	2042.010	2061.580	2039.886	2058.588
	tau	14.863	.602	13.658	16.067	13.403	15.673
	delta	7.062	.747	5.566	8.558	6.287	8.919

a. Based on 60 samples.

Participant 8

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3654.479	9.669	3635.251	3673.707		
	tau	16.591	.488	15.620	17.562		
	delta	1.632	.550	.537	2.726		
Bootstrap[a]	asymptote	3654.479	12.690	3629.086	3679.872	3631.631	3688.731
	tau	16.591	.775	15.041	18.141	15.459	18.135
	delta	1.632	.906	-.182	3.445	-.609	2.678

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2390.008	3.883	2382.279	2397.738		
	tau	12.553	.286	11.984	13.122		
	delta	3.922	.357	3.211	4.634		
Bootstrap[a]	asymptote	2390.008	4.774	2380.456	2399.561	2383.997	2402.273
	tau	12.553	.249	12.055	13.052	12.156	13.013
	delta	3.922	.261	3.400	4.445	3.438	4.390

a. Based on 60 samples.

Participant 9

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2991.091	3.891	2983.358	2998.824		
	tau	8.649	.157	8.336	8.962		
	delta	9.903	.166	9.573	10.234		
Bootstrap[a]	asymptote	2991.091	3.187	2984.714	2997.467	2983.391	2996.721
	tau	8.649	.364	7.921	9.377	7.846	9.090
	delta	9.903	.526	8.851	10.956	9.344	11.165

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2237.999	4.495	2229.042	2246.957		
	tau	14.591	.399	13.796	15.386		
	delta	.319	.521	-.719	1.356		
Bootstrap[a]	asymptote	2237.999	3.289	2231.418	2244.580	2230.953	2246.598
	tau	14.591	.421	13.749	15.432	13.721	15.402
	delta	.319	.500	-.682	1.320	-.635	1.377

a. Based on 60 samples.

Participant 10

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2236.276	4.805	2226.741	2245.811		
	tau	11.359	.383	10.599	12.119		
	delta	6.025	.469	5.095	6.956		
Bootstrap[a]	asymptote	2236.276	6.046	2224.178	2248.374	2226.587	2247.084
	tau	11.359	.589	10.180	12.538	10.758	12.812
	delta	6.025	.856	4.313	7.738	3.963	6.544

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2119.227	5.218	2108.880	2129.573		
	tau	14.228	.477	13.282	15.174		
	delta	4.403	.555	3.302	5.504		
Bootstrap[a]	asymptote	2119.227	5.481	2108.259	2130.194	2107.652	2131.348
	tau	14.228	.340	13.547	14.909	13.894	15.517
	delta	4.403	.497	3.408	5.398	2.507	4.737

a. Based on 60 samples.

Participant 11

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3466.569	8.724	3449.243	3483.895		
	tau	8.302	.324	7.659	8.944		
	delta	9.730	.357	9.020	10.440		
Bootstrap[a]	asymptote	3466.569	9.646	3447.268	3485.870	3449.839	3488.467
	tau	8.302	.500	7.302	9.301	7.253	9.041
	delta	9.730	.639	8.452	11.008	9.113	11.037

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2684.150	5.209	2673.822	2694.478		
	tau	12.207	.288	11.636	12.777		
	delta	8.548	.287	7.979	9.116		
Bootstrap[a]	asymptote	2684.150	5.051	2674.043	2694.257	2677.241	2694.493
	tau	12.207	.346	11.515	12.898	11.833	13.315
	delta	8.548	.435	7.677	9.418	7.041	8.998

a. Based on 60 samples.

Participant 12

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	3197.655	6.884	3183.982	3211.328		
	tau	7.981	.382	7.222	8.741		
	delta	7.215	.542	6.139	8.291		
Bootstrap[a]	asymptote	3197.655	7.087	3183.474	3211.836	3184.918	3208.619
	tau	7.981	.369	7.243	8.719	7.114	8.361
	delta	7.215	.712	5.790	8.640	6.669	8.776

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2383.191	5.116	2373.004	2393.379		
	tau	10.395	.328	9.742	11.049		
	delta	6.431	.403	5.629	7.233		
Bootstrap[a]	asymptote	2383.191	4.140	2374.907	2391.476	2376.713	2391.639
	tau	10.395	.418	9.559	11.232	9.832	11.238
	delta	6.431	.540	5.350	7.513	5.204	7.081

a. Based on 60 samples.

Participant 13

Normoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2991.091	3.891	2983.358	2998.824		
	tau	8.649	.157	8.336	8.962		
	delta	9.903	.166	9.573	10.234		
Bootstrap[a]	asymptote	2991.091	3.187	2984.714	2997.467	2983.391	2996.721
	tau	8.649	.364	7.921	9.377	7.846	9.090
	delta	9.903	.526	8.851	10.956	9.344	11.165

a. Based on 60 samples.

Hypoxia

Parameter Estimates

	Parameter	Estimate	Std. Error	95% Confidence Interval		95% Trimmed Range	
				Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2238.262	3.782	2230.741	2245.783		
	tau	14.567	.184	14.201	14.933		
	delta	10.418	.137	10.145	10.691		
Bootstrap[a]	asymptote	2238.262	3.149	2231.960	2244.563	2232.171	2245.584
	tau	14.567	.182	14.203	14.932	14.172	14.896
	delta	10.418	.124	10.169	10.667	10.249	10.710

a. Based on 60 samples.

Participant 14

Normoxia

Parameter Estimates

				95% Confidence Interval		95% Trimmed Range	
	Parameter	Estimate	Std. Error	Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2600.209	5.179	2589.886	2610.532		
	tau	6.428	.359	5.712	7.144		
	delta	7.134	.587	5.964	8.305		
Bootstrap[a]	asymptote	2600.209	5.803	2588.598	2611.820	2588.558	2613.241
	tau	6.428	.290	5.848	7.008	5.537	6.721
	delta	7.134	.549	6.036	8.233	6.680	9.096

a. Based on 60 samples.

Hypoxia

Parameter Estimates

				95% Confidence Interval		95% Trimmed Range	
	Parameter	Estimate	Std. Error	Lower Bound	Upper Bound	Lower Bound	Upper Bound
Asymptotic	asymptote	2096.155	5.428	2085.340	2106.971		
	tau	10.397	.299	9.801	10.993		
	delta	9.075	.297	8.483	9.668		
Bootstrap[a]	asymptote	2096.155	3.762	2088.627	2103.684	2086.495	2101.739
	tau	10.397	.521	9.354	11.441	8.850	11.185
	delta	9.075	.660	7.754	10.397	8.343	11.192

a. Based on 60 samples.

Related samples T-test to explore the differences between parameter estimates between the exercise conditions.

Baseline

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	normoxia	600.2857	14	109.43943	29.24892
	hypoxia	669.4286	14	153.03077	40.89919

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	normoxia - hypoxia	-69.14286	183.20516	48.96364	-174.92237	36.63665	-1.412	13	.181

GAIN

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	normoxia	2447.2472	14	495.73365	132.49039
	hypoxia	1610.9191	14	266.46777	71.21651

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	normoxia - hypoxia	836.32807	475.50605	127.08434	561.77905	1110.87709	6.581	13	.000

Asymptote

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	3047.5329	14	505.30482	135.04839
	h	2280.3477	14	205.90363	55.03006

Paired Samples Test

	Paired Differences							
				95% Confidence Interval of the Difference				
	Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1 n - h	767.18521	416.20978	111.23674	526.87284	1007.49758	6.897	13	.000

τ

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	10.4317	14	2.62383	.70125
	h	12.7046	14	2.81393	.75205

Paired Samples Test

	Paired Differences							
				95% Confidence Interval of the Difference				
	Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1 n - h	-2.27286	3.47138	.92777	-4.27717	-.26854	-2.450	13	.029

δ

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	7.5877	14	2.64492	.70689
	h	7.3929	14	3.28112	.87692

Paired Samples Test

	Paired Differences							
				95% Confidence Interval of the Difference				
	Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1 n - h	.19479	3.79949	1.01546	-1.99897	2.38855	.192	13	.851

MRT

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	18.0194	14	2.93759	.78510
	h	20.0968	14	3.26339	.87218

Paired Samples Test

	Paired Differences							
				95% Confidence Interval of the Difference				
	Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1 n - h	-2.07736	3.88731	1.03893	-4.32182	.16711	-2.000	13	.067

Bootstrap standard error of estimate between conditions

Asymptote

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	n	5.8858	14	2.98086	.79667
	h	5.6074	14	2.24205	.59921

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 n - h	.27843	3.68053	.98366	-1.84665	2.40350	.283	13	.782

τ

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	h	.4121	14	.12744	.03406
	n	.4069	14	.16025	.04283

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 h - n	.00514	.26034	.06958	-.14518	.15546	.074	13	.942

δ

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	h	.4689	14	.21079	.05634
	n	.5849	14	.22389	.05984

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	h - n	-.11593	.31889	.08523	-.30005	.06819	-1.360	13	.197