BENAJMIN LLOYD OWEN

ST05002650

SPORT AND EXERCISE SCIENCE

UNIVERSITY OF WALES INSTITUTE CARDIFF

A COMPARISON OF THE VO2 KINETIC RESPONSE BETWEEN 400- AND 1500-METRE TRACK ATHLTES TO SUPRAMAXIMAL TREADMILL RUNNING

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ABSTRACT

Purpose: The aim of the present study was to investigate if a difference existed in the time course of 400- and 1500m track athletes, in response to supramaximal treadmill running. **Methods:** Twelve collegiate level male athletes consisting of seven 400-metre sprinters (mean ± SD 400: age 20.7 ± 1.4 yr; $\dot{V}O_{2\,max}$ 62.8 ± 6.7 $mL \cdot kg^{-1} \cdot min^{-1}$; Personal best 50.08 ± 0.65 seconds) and five 1500-metre middle distance athletes (mean ± SD 1500: age 21 ± 0.9 yr; $\dot{V}O_{2\,max}$ 73.16 ± 4.6 $mL \cdot kg^{-1} \cdot min^{-1}$; Personal best 4.06 ± 0.2.5 minute, seconds) were used as subjects in the investigation. Each subject completed an initial incremental steady state $\dot{V}O_{2\,max}$ treadmill test to determine maximal treadmill velocity at $\dot{V}O_{2\,max}$. Subsequent exercise transitions of 3 minutes at 110% of peak $\dot{V}O_{2\,max}$ treadmill velocity. ($km \cdot h^{-1}$) to represent supramaximal intensity treadmill running were then used as a means of modelling the $\dot{V}O_2$ response to supramaximal intensity running. Pulmonary gas measurement was measured breath-by-breath and H.R was taken at 5-s intervals. **Results:** T-tests revealed that no significant difference (P= 0.1993) exists between the mean τ response exists between 400- and 1500-metre athletes in the supramaximal intensity domain. The homogenous populations exhibited significantly different $\dot{V}O_{2\,max}$ scores (P value =0.017) and final minute H.R (P-value 0.0075). **Conclusions:** From the responses exhibited by the two populations assumptions can be that the τ in response to supramaximal treadmill running is not a function of an athletes $\dot{V}O_{2\,max.}$ Any differences in $\dot{V}O_2$ kinetics observed between the two populations could be attributed to varying central and peripheral adaptations induced by the varying training methods for the two events although no significant difference existed.

CHAPTER I
INTRODUCTION

1.0 INTRODUCTION

The 400-m and the 1500-m track events require the unique ability to sustain high intensity work for prolonged periods of time. The body's ability to cope with the exercise challenges from these events involves significant energy contributions from both anaerobic and aerobic energy systems. The degree to which an athlete tolerates the demand of the exercise imposed will depend, in part, on the speed of their oxygen kinetics (Jones & Poole, 2005). Unresponsive VO_2 on-kinetics are believed to result in a greater depletion of intra-muscular high-energy phosphates, and greater accumulation of lactate and hydrogen ions (H^+) i.e. a greater anaerobic energy contribution (Jones & Koppo, 2005, in Jones & Poole 2005). An increase in [H^+] (acidosis) and inorganic phosphate (Pi) during exercise will lead to muscular fatigue and performance decrements (Wilkie, 1986). Ideally, it would be advantageous for athletes to offset the depletion of the muscles finite phosphocreatine (PCr) stores, and the accumulation of fatiguing anaerobic by-products. Faster VO_2 on-kinetics will facilitate this process, as the oxygen deficit is subsequently reduced, thereby indicating a sparing of intra-muscular [PCr] and attenuating the production of lactic acid (Jones & Poole, 2005, p.379).

The process of exercise training has consistently been shown to accelerate phase II VO_2 on-kinetics (Berger *et al*., 2006; Carter *et al*., 2000; Koppo *et al*., 2000). However, the optimal training strategy to accelerate VO_2 kinetics is, at present, unknown (Berger *et al*., 2005). The contrasting training undertaken by 400-m and 1500-m runners make them interesting populations to compare. Differences in VO_2 kinetics of sprint versus endurance athletes have been investigated for moderate intensity exercise but relatively untouched in the field of severe intensity exercise (Draper *et al*,. 2005). Specifically, 400-m athletes typically engage in repeated-

sprint training, while the 1500-m athlete counterparts undertake predominantly extended endurance bouts of exercise. The dynamic VO_2 response exhibited by these athletes, during an abrupt transition to a supramaximal work rate, will provide insights into the most suitable training interventions to accelerate VO_2 kinetics.

Initial energy pathways such as the ATP, adenosine di-phosphate (ADP) and creatine phosphate (CP), which provide the immediate source of energy, are exhausted within 30 seconds (Linderman & Gosselink, 1994). Thereafter, energy from both the aerobic and anaerobic pathways fuels the working muscles during events lasting over 40 seconds to 5 minutes i.e. 400-m and 1500-m (depending on ability). To execute supramaximal or severe intensity work, both sprint and endurance trained athletes will require cardio-respiratory and metabolic responses to exercise which will enable them to sustain the energy production necessary to optimise running speed over their chosen distances, while minimising the accumulation of fatiguing bi-products. This has recently received a great deal of research attention; primarily the dramatic increase in blood flow (O_2 delivery) and O_2 uptake observed at exercise onset, in order to meet this increased metabolic demand (Caputo & Denadai., 2004).

The nature of the 400-m event is that it involves heavy reliance upon all 3 of the energy pathways (Duffield *et al*., 2003), primarily being the ATP, ATP-PC and lactic acid systems. As the time to complete the 400-m exceeds 30s the predominating energy source for the event is anaerobic glycolysis (a non-oxidative metabolic process). At the onset of exercise the rapidity with which the rate of adenosine triphosphate (ATP) supply through oxidative phosphorylation can alter to meet the total ATP turnover rate in the transition to a higher metabolic rate is a

central determinant of exercise tolerance (Jones & Poole, 2005). Indeed, it has recently been found that the energy pathways supplying the ATP during the 400-m is more aerobic than previously assumed. Accordingly, 400m trials conducted by Spencer & Gastin (2000) found the event to have an aerobic contribution of 43% and as high as 59% (Duffield et al., 2003). Comparing this figure with that of the 1500-m discipline we see a substantial change in the contribution of aerobic metabolism, with studies (Hill. 1999; Spencer. et al., 1996; Duffield. et al., 2003) quantifying the aerobic contribution as high as 83%. Although the 400-m and 1500-m track events have significantly different energy system contributions, they both require a rapid response at the onset of exercise. With this in mind, it is in the interest of the researcher to investigate which of the two disciplines demonstrates a more immediate cardio-dynamic response to exercise.

The purpose of the current study is, therefore, to compare the VO_2 kinetics of trained 400-m and 1500-m runners to supramaximal exercise. From this it is then possible to infer the optimal training necessary to accelerate VO_2 kinetics. This would be of interest to both coaches and exercise physiologists and provides further information on the mechanisms behind the $\dot{V}O_2$ kinetic response.

CHAPTER II
CRITICAL REVIEW OF LITERATURE

2.0 CRITICAL REVIEW OF LITERATURE

2.1 Historical overview of $\dot{V}O_2$ kinetics

Successful athletic performance is characterised by the coordinated function of many body systems and in most athletic events almost every major system of the body is engaged and displaced from the normal equilibrium (Gollnick *et al,* 1984). Movement or exercise in humans requires rather immediate transitions from one metabolic rate to another. It was the work of physicians Karl Wasserman and Brian J. Whipp (Whipp *et al*, 1971; Whipp *et al*, 1972 cited in Jones & Poole, 2005) who accelerated the research in the $\dot{V}O_2$ kinetics, facilitated by advances in technology allowing breath-to-breath measurements of ventilation and pulmonary gas exchange. The three phase response to exercise was characterised by the same two researchers (Whipp *et al*, 1971; Whipp *et al*, 1972; Beaver *et al*, 1986) who provided a mechanistic physiological basis of how the body responded to exercise. Scientists from varying fields have all contributed to the area of research known today as 'Oxygen Kinetics' in the broader field of physiology. As early as the early 18th and 19th century scientists Lavoisier, Zuntz and Geppert have used muscular exercise to enhance our knowledge of the functions of respiration and metabolism (Jones & Poole, 2005). It was the early work of Krogh and Lindhard (1913) that first recognised the dynamic phase of $\dot{V}O_2$ and its role in production of an oxygen deficit at the beginning of exercise.

2.2 Key controversies within the literature

A study by Whipp *et al*. (1990) was undertaken in the effort to clarify the mechanisms coupling mitochondrial energy production via the oxidative pathway to the ventilatory gas exchange and also to the connective circulatory gas transport. Breath-by-breath (BbB) measurement of the pulmonary oxygen uptake in humans provides a relatively simple, non invasive process of obtaining information upon integration of pulmonary, cardiovascular, neural and muscle energetic systems (Jones & Poole, 2005). With this advancement in automatic systems for computing on a breath-by-breath (BbB) basis analysis of $\dot{V}O_2$ has become increasingly popular. Although a vast amount of the literature (Xu et al 1999; Burnley et al, 2002; Barstow et al, 1996 and more recently Cleuziou et al, 2004) exists to report that the determinants of $\dot{V}O_2$ dynamics are difficult to localise, this keeps the subject of oxygen kinetics as the source of much debate.

2.2.1 Breath-by- breath analysis of pulmonary gas exchange

Attaining the rate of exchange of O_2 and CO_2 between alveolar gas and pulmonary capillary blood ($\dot{V}O_2$ and $\dot{V}CO_2$ respectively) via a breath-by-breath (BbB) basis is not accomplished solely by simply measuring change in the amount of O_2 and CO_2 inspired and expired at the mouth during just one particular breath. Other changes that occur are changes in the volume of O_2 and CO_2 stored in alveolar gas that do not get diffused from or to pulmonary capillary blood (Alvierti *et al*, 1996).

The remarkable progress achieved in modelling of the gas exchange kinetics in humans over the past two decades has also amounted with some criticism. Studies by

Cautero *et al*. (2002); Cautero *et al*. (2003) and Alvierti *et al*, 2004) have all criticised breath-by-breath measurement of alveolar gas stores and exchange, focusing their criticism primarily that commercially available devices for BbB gas measurement do not take the sources of error caused by storages in alveolar gas into account.

2.2.2 Pulmonary $\dot{V}O_2$ kinetics as a representation of muscle $\dot{V}O_2$ kinetics

Research by Grassi *et al*, (2006) demonstrated that careful measurements of pulmonary $\dot{V}O_2$ kinetics can provide an accurate representation (approx ± 10%) of the τ for skeletal muscle $\dot{V}O_2$ following the onset of exercise. Although during measurement pulmonary $\dot{V}O_2$ produces inherent breath-to-breath 'noise'. However this can be reduced using the correct optimal exercise modality; as a greater recruited muscle mass correlates with reduced breath breath-by-breath noise. With reference to the current study, the exercise modality used was treadmill running and the subjects were well-trained collegiate level athletes subsequently allowing BbB noise to be kept to a minimum. These techniques used in modelling a more accurate representation of pulmonary uptake kinetics are used in conjunction with data editing, averaging procedures of identical exercise transitions (Jones and Poole, 2005). Correct methodology and testing procedures will ensure that the most accurate representation of the $\dot{V}O_2$ response is obtained.

2.3 Sprint and endurance training

There have been reported differences in the $\dot{V}O_2$ between sprint- and endurance-trained runners but only at moderate intensity exercise (Edwards *et al*. 1999). This does have reference to the present in that it can justify an inquisition into whether the same reported differences occur in the severe/ extreme intensity domain. Although a recent study by Draper and Wood (2005) found no significant differences in τ between sprint-trained (11.2 ± 1.1-s) and endurance-trained athletes (9.3 ± 1.9-s) during ~ 2 min of maximal running exercise. This was the first study that compared the $\dot{V}O_2$ responses of sprint- and endurance trained athletes. A weakness did exist in this study as the total number of subjects was only twelve compared to that in the study by Berger *et al*., (2006), where eighty-four master athletes were tested. In this study there was conclusive evidence to support that endurance trained athletes elicited faster response times, but again only during moderate intensity exercise and using masters athletes (sedentary athletic population).

A continual effort is seemingly being made to address which type of training is more beneficial for each distance event in track running. Specificity within a training programme is vital for provoking the desired adaptations from training. Gollnick *et al*., (1984) posited that it is unrealistic to attribute the improvement of one physiological component to an individuals improved performance. But what athletes, coaches and physiologists are continually working towards is conclusive evidence to support certain types of training adapting various responses to exercise, Dudley *et al.* (1982) demonstrated that rats undertaking intense work bouts for shorter time induced similar increases in the maximal activities of several oxidative enzymes (i.e.

cytochrome *c*) to those observed after more prolonged submaximal exercise training although recent research has found low-intensity continuous training and high-intensity interval training to be similarly effective in accelerating $\dot{V}O_2$ on-kinetics (Berger *et al*., 2006).

Similar types of training specificity differences are witnessed in training programmes for the 400-m and 1500-m populations. Specifically, 400-m athletes typically engage in repeated-sprint training, weight training, low distance sessions amongst technique based activities; primarily increasing the peripheral physiological functions of the athlete e.g. muscle buffering capacity, fibre type composition, mitochondrial density amongst increasing glycolytic enzyme activity (Macdougall *et al,*1998). Whereas the 1500-m athlete undertake predominantly extended endurance bouts of exercise, which in turn will promote central adaptations e.g. increased cardiac output, pulmonary diffusing, capacity and O_2 carrying capacity often reflected in a greater $\dot{V}O_2$ max. Peripheral adaptation that may occur in the oxygen uptake response induced by 1500-metre endurance type training has been evidenced to cause minimum effect on glycolytic enzymes (Holloszy, 1975).

As the duration of a maximal performance increases, there is greater reliance on ATP production via oxidative phosphorylation as the $\dot{V}O_2$ in endurance performance is limited by central cardiovascular factors (Bassett *et al*, 1999).

Observing the dynamic $\dot{V}O_2$ response exhibited by these athletes, during an abrupt transition to a supramaximal work rate, will provide insights into the most suitable training interventions to accelerate $\dot{V}O_2$ kinetics.

Endurance training as previously stated typically involves either continuous training at submaximal work rates and interval exercise mixing high intensity work with low-intensity recovery periods, although both will stress the cardiovascular system (Berger *et al*. 2005), it has been suggested that interval training may elicit greater speeding of $\dot{V}O_2$ kinetics for work rates >LT (Berger *et al*, 2006) hence a study comparing populations who engage in both types of training could fuel a hypothesis that interval training may be more effective than continuous training for faster $\dot{V}O_2$ on-kinetics in athletes.

2.4 VO_2 Kinetic response during the varying intensity domains

Three distinct $\dot{V}O_2$ phases have been evidenced to exist following the onset of moderate intensity exercise: Phase I is the sudden increase in $\dot{V}O_2$ and lasts for the initial 15-20 seconds, consequent of a sudden elevated blood flow through the pulmonary capillaries, a product of the rapid return to the lungs of blood due to the action of the muscle pump (Jones & Poole, 2005); Phase II is the "fundamental" exponential rise in $\dot{V}O_2$, the cardio-vascular reaction trying to obtain a new $\dot{V}O_2$ steady state: Phase III exists where the VO_2 has reached its steady state for the work rate (Berger et al., 2006). All three responses are shown in figure 1.

The $\dot{V}O_2$ kinetic response at the onset of exercise is a function of how the body responds to exercise intensity. Beginning with 'moderate-intensity' exercise (below the lactate threshold) increases exponentially to a steady-state level (Whipp, 1971). The 'heavy-intensity' domain requires work rates higher than the lactate threshold,

but below the maximum lactate steady-state. Intensity then increases to 'heavy' exercise, where the slow component causes $\dot{V}O_2$ to increase progressively, towards an upper limit, delaying the attainment of a steady-state level (Poole *et al*., 1988). The severe-intensity domain represents work rates between MLSS and $\dot{V}O_{2\,max}$, where $\dot{V}O_{2\,max}$ can be achieved, lactate is increased, and where fatigue is reached in ~ 10 min (Jones & Poole, 2005). Finally, for the intensities that exist above severe domain are categorised under the 'extreme' domain, where there is insufficient time for $\dot{V}O_2$ to attain its maximum, and fails to stabilise and continues to rise up to fatigue (Hill *et al*., 2002b). The slow component developed during severe exercise is much greater than that observed in lower intensity domains (Xu *et al*, 1999). Figure 1 is a representation of the oxygen uptake response to three exercise intensities. (The Phase I response has been deleted).

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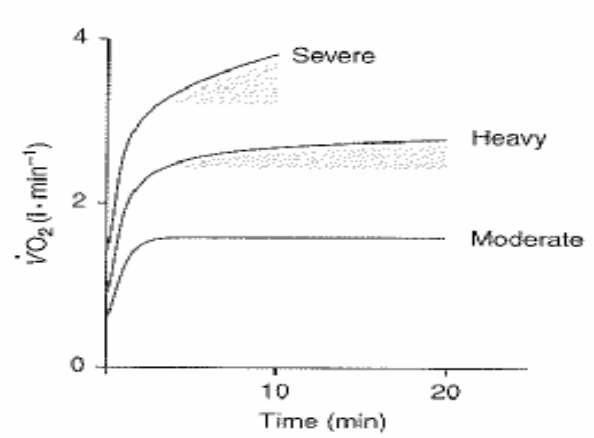

Figure 1, A Schematic representation of the $p\dot{V}O_2$ response to constant work-rate exercise in moderate, heavy and severe exercise intensities (Jones and Poole, 2005. p18)

The Aerobic energy pathway has been evidenced to respond quickly to the work demands from events ranging from 200 to 1500-metres, with response times ranging from 8-seconds upwards (Spencer *et al*., 2000; Jones & Poole, 2005, p.378). A 400-metre event will last between 44-50secs in elite and sub-elite athletes, therefore a 400-metre sprint athlete may spend 30-40 seconds utilising either the aerobic or anaerobic energy pathways although. However, no research to the present day has investigated the $\dot{V}O_2$ kinetic response in 400-m runners to supra-maximal treadmill running exercise. Duffield *et al*. (2005) recently found the aerobic/anaerobic energy system contribution to the 400-m to be 41/59% in males, far greater than traditionally assumed (e.g., 28/72%; Lacour *et al*., 1990)

As a sprint event requires a more rapid response from the body it is likely that because the $\dot{V}O_2$ slow component produced during severe exercise will be much greater than that during heavy exercise and could lead $\dot{V}O_2$ to increase up to exhaustion during severe intensity exercise. We may see the same occurring between sprint and endurance trained athletes response to a supra-maximal transition. The principal mechanism(s) to explain the $\dot{V}O_2$ slow component are still to be recognized and are more complex at higher intensities, but have been suggested to be relative to the proximity of the exercise intensity to the critical power (CP) (Cleuziou *et al*., 2004; Carter *et al*., 2001).

As exercise within the severe domain increases in intensity, the anaerobic contribution increases also (Whipp *et al*., 1998). It has been found that the favoured

quick aerobic response or acceleration of $\dot{V}O_2$ kinetics can be a product of endurance training (Demarle et al., 2001; Jones et al., 2003), which in turn reduces initial oxygen deficit and lactate production, possibly leading to prolonged maximal exertion during exercise (Caputo et al., 2004). An explanation to this higher anaerobic contribution as severe exercise intensifies or is prolonged may reside in the fibre recruitment patterns (Barstow *et al.*, 2000).

2.5 Energy Metabolism during Severe – Intensity Exercise

Energy metabolism or resynthesis of ATP is predominantly anaerobic for severe/extreme intensity exercise in excess of 30 seconds (mostly anaerobic glycolysis and PCr degradation) (Gaitanos *et al*., 1993; Medbo & Tabata, 1993). ATP turnover rate in the muscle increases during exercise and how well the body is able to cope with this higher ATP is subject to the speed of both the aerobic and anaerobic processes. The two major anaerobic processes are linked with lactate production and the breakdown of phosphocreatine (Medbo & Tabata, 1993). ATP stores will be broken down in order to facilitate muscle contraction. This occurs via the process of hydrolysis, the resultant action of the enzyme myosin ATPase and the subsequent formation of ADP and Pi ($H_2PO_4^-$) ;Robergs, 2001), subsequently the phosphagen system will be utilised to maintain ATP production for continued muscle contraction. Although the initial energy pathways previously mentioned can generate the high energy ATP, ADP and PC these are depleted in 30 seconds during high intensity exercise (Linderman & Gosselink, 1994) hence a smaller depletion of the high energy stores at the onset of exercise will result in more efficient use of these stores. Consequently an event that is high intensity, lasting over 30 seconds such as the 400-metre sprint will rely upon anaerobic glycolysis being the

predominant energy pathway (Duffield *et al*., 2003; McNaughton, 1992b). Any methods in which training induced adaptations can be acquired be athletes to make the initial stages of a race less taxing upon the fatiguing energy pathways in events such as 400-metre or 1500-metres may result in greater end of race performance.

2.6 Oxygen deficit in sprint and endurance events

The oxygen deficit (O_2 deficit) is the difference between the total O_2 demand created by exercise and the actual accumulated uptake (Hill *et al*., 2002). During high intensity exercise there is evidence to support the theory that there is a high level recruitment of low efficiency type IIb fibres, which in turn have been associated with an increase on the oxygen cost of exercise (Xu *et al*., 1999). Facilitating a smaller oxygen deficit would entail various physiological adaptations, elicited through either sprint or endurance training. A study by Hagberg *et al*. (1980) had 8 subjects performing a high intensity training programme for 9 weeks, which resulted in the confirmation that this sprint or endurance training can result in the speeding of $\dot{V}O_2$ kinetics and significantly lower magnitudes of O_2 deficit. In a subsequent paper, Hagberg *et al*. (1980) found there was a significant relationship between 7 days of continuous "head down bed rest" and an increase in the O_2 deficit during upright cycling. This then strengthens the notion that training has positive implications upon the oxygen deficit (consistent with slowed VO_2 kinetics) (Jones & Koppo, 2005). Therefore lowering the muscles O_2 requirement at the varying exercise intensities via more efficient blood flow kinetics or increased capillary-to-fibre ratio, could in turn reduce the body's net consumption of O_2 during at the varying intensities. Phillips *et*

al. (1995) attributed the speed at which 7 untrained subjects managed to rapidly change the time constant from 37.2s (pre-training) to 28.8s (4 days post training) and then eventually 15.8s (after 30 days/ completion of training programme) to 2 hours daily cycling on a stationary ergometer at 60% of VO_2max. Although this study demonstrated that the change/ adaptation can occur there is still no conclusive evidence to explain why it occurs.

2.6.1 Oxygen deficit and fatigue in extreme intensity treadmill running

Aerobic power may be quantified in exercise via the use of $\dot{V}O_2$ max testing whereas the anaerobic contribution to various cycle and sprint exercise has been found to be more difficult to ascertain (Medbo 1996). (Medbo *et al*., 1988) earlier found that for the O_2 deficit to be used as a measure of anaerobic capacity, a severe intensity exercise test must be used, where the subject fatigues in ~2 to 4 minutes.

2.7 $\dot{V}O_2$ slow component and fatigue during high intensity exercise

It is the slow component which is the complex underpinning of $\dot{V}O_2$ kinetics at high intensities due to the non conclusive evidence that exists attributing its existence to different physiological processes and components.

2.7.1 Blood lactate

A plethora of research exists to support the notion that the slow component of $\dot{V}O_2$ is closely linked with blood lactate concentration, derived from studies (Poole at al, 1988; Casaburi et al, 1989; Barstow, 1994) where the magnitude of the slow component of VO_2 correlates with increases in blood lactate. An excellent correlation between the decrease and blood lactate was reported by Casaburi et al, (1987) which was found to be the product of a training programme suggesting that the physiological mechanisms that adapt to training could be responsible for the $\dot{V}O_2$ slow components existence. In contrast though evidence from Barstow's 1994 paper suggests that the slow component $\dot{V}O_2$ was initiated 80-110 seconds into exercise and at the time, femoral blood lactate levels had already been elevated (Xu, 1999). Although more than 70% of the lactate which is formed during exercise is oxidised within the active muscles, the remainder is removed by hepatic gluconogensis (Brooks 1986) and it is this biochemical process which may be the cause of the slow increase in O_2 consumption as lactate oxidation occurs (Xu et al., 1999). Studies contradicting this explanation for slowed kinetics due to blood lactate do so using subject population of canine origin or in hypoxic conditions (Engelen et al, 1996; Poole et al, 1991; Poole et al, 1994) and this has little in common with the subjects and conditions in the present study.

Evidence supporting blood lactate playing a role in the generation of the $\dot{V}O_2$ slow component does so in attributing promotion of oxyhaemoglobin dissociation, which in turn, increases transportation of O_2 to the working muscle, maintaining PaO_2 above critical levels (Xu et al, 1999). Although some studies have shown a raltionship between blood lactate and the slow component, which may be explained via higher utilisation of the glycolytic pathways utilised by different athletes.

Much of the literature reviewed is that from studies focusing in the heavy and moderate intensities. Whereas the literature looking at blood lactate concentration and the $\dot{V}O_2$ slow component in the supra-maximal intensities sees little research attention even though this is the intensity at which the elite level performances occur in track events such as 1500-m.

2.7.2 Epinephrine –Adrenaline

Increases in plasma adrenaline share very similar characteristics to that of blood lactate (Xu *et al*, 1999) as the thresholds of lactate and epinephrine exist at very similar work rates (Turner *et al*, 1995). From the research in to the $\dot{V}O_2$ Epinephrine seems only to be able to explain kinetic responses due to its affect upon circulatory, respiratory and metabolic systems (Gaesser *et al*, 1994; Gaesser *et al*, 1995; Womack *et al*, 1996), whereas its effect upon the slow component remains inconclusive.

2.7.3 Ventilation

Hyperventilation accompanies any high intensity exercise, serving the purpose of effective gas exchange within the lungs. But it has been queried whether the elevated ventilation rate could be a mechanism for the $\dot{V}O_2$ slow component. Although, there is variance in the reported costs of ventilation ranging from 4.3ml when ventilation was above 89L/min (Shepherd, 1966) to 3ml O_2/L for ventilation in the range from 117 to 147 L/min (Aaron *et al*, 1992). With these observed differences in O_2 cost for

ventilatory increase it then becomes difficult to obtain its true energy cost during exercise. Three conclusive studies (Aaron *et al*,1992; Gaesser *et al*,1996); Womack *et al*, 1995) suggested that O_2 cost due to increased ventilation could account for as high as 23% of the total $\dot{V}O_2$ slow component.

2.7.4 Motor unit recruitment

As the need for muscle contraction is needed immediately during participation in the current study, a clearly observed slow component will exist and a well-established hypothesis states may be the product of additional recruitment of motor units (Whipp, 1994). This theory was reinforced when the discovery that 'priming exercise' could be used to reduce the $\dot{V}O_2$ slow component (Saunders et al., 2000; Perrey *et al*, 2001) when prior heavy exercise suggests that the has been an alteration of motor recruitment. But the findings attributed the reduction in the slow component primarily to this factor although prior exercise will cause other physiological responses (increased ventilation, increased body temperature, epinephrine secretion) also believed to be responsible for the slow component.

2.8 Fibre type and subsequent VO_2 response to exercise

Human skeletal muscle is composed of two main fibre types, type I (slow twitch) and type II (fast twitch) (Xu *et al*, 1999; Barstow *et al*, 1996) The kinetics of $\dot{V}O_2$ has been characterised as a function of the type I (slow twitch) muscle fibres present in the contracting muscles (Barstow *et al*, 2000). Studies looking at the energetics of these fibres found the high energy phosphate per oxygen molecule consumed (P/O) is less in type I fibres than in type II (Kushmerick *et al*, 1992). Barstow *et al*.,(1996) used the measurement of kinetics as a indirect approach of assessing whether the $\dot{V}O_2$ slow component was coupled with recruitment of type II muscle fibres. The

findings were that fibre type distribution was found to significantly affect the $\dot{V}O_2$ response during exercise. Some small limitations investigating $\dot{V}O_2$ kinetics and fibre type that exist are that only small biopsies are taken from muscles assuming them as a representation for the whole working muscle (Jones & Pringle, 2005,p.261). Although there is a case for flaws in the evidence obtained from studies examining the relationship between fibre type and performance, it is crucial that this data be obtained because if fibre type is known relative to response to exercise, then the desired response will become easier to train.

2.9 Summary from the literature

The literature reviewed in the area of oxygen uptake kinetics strongly suggests that faster τ is a product of training status, exercise intensity and physiological characteristics. A cross sectional study observing both the 400-metre and 1500-metre responses to supramaximal exercise may provide an interesting insight into the types of training or athlete physiology is needed to demonstrate the fastest oxygen uptake response to exercise.

CHAPTER III
METHODS

3.0 METHODS

3.1 Subjects and experimental design:

Twelve male subjects in total were selected from the track and field team at the University of Wales Institute Cardiff (UWIC) and agreed to participate. They comprised of seven 400-metre track athletes and five 1500-m/ cross country athletes. The study was granted ethical approval from the University of Wales Ethics board. The participants' anthropometric and physiological and performance characteristics are presented in Table 1.

Table 1, Descriptive statistics:

SUBJECTS:	AGE: (YEARS)	HEIGHT: (CM)	WEIGHT: (KG)	PERSONAL BEST: 400- (SECS) 1500- (MINS. SECS)
400-metre athlete	20.4±1.4	176.1±8.1	74.4±6.9	50.08±0.65
1500- metre athletes	18.8±1.3	179.8±5.2	66.2±5.6	4.06±0.26 s
Complete sample	19.8±1.5	177.7±7.0	71.0±7.5	N/A

Prior to the measurements being taken, each subject was fully informed of the risks, commitment required and the testing procedures. After verbal and written consent to participate in the study, a physical activity readiness questionnaire (PARQ) was completed by each subject (Appendix B). Each subject's personal best time for their event was also noted. The personal best times are representative of well trained collegiate level athletes.

Each subject was then familiarised with the equipment being used during the test to allow the subjects to get used to the testing procedure e.g. allowing the athletes to

become accustomed to initiating running on the treadmill when it is already at high speeds, although many of the subjects were already highly accustomed to high intensity treadmill running. The habituation also allowed the participants to become accustomed to running with heart rate monitor and face mask on. The subjects were also instructed to arrive at the laboratory in as rested and fully hydrated state as possible. Subjects were requested to avoid drinking alcohol and caffeine for a minimum of 12 hours prior to participating in each test and all transitions were conducted at the same time of day (± two hours), to reduce the effects of biological variation on the $\dot{V}O_2$ kinetic response (Carter *et al*., 2002).

3.2 Steady state $\dot{V}O_{2\,max}$ test

Following their recruitment to the study the subjects performed a steady state incremental $\dot{V}O_{2\,max}$ treadmill test to exhaustion in order to determine $\dot{V}O_{2\,max}$ and peak treadmill velocity.

Heart rate was collected at 5-s intervals during all exercise sessions using non-invasive telemetry (Polar S610, Polar Electro, Oy, Kempele, Finland) and the tests were all performed on a motorised treadmill (Woodway Ergo Rona ES70, Germany). During the test the treadmill remained at a constant gradient of 1% in order to represent the energy cost experienced during outdoor running (Jones & Doust, 1996). Each subject started the test at a speed where projected volitional exhaustion would be elicited in approximately 15 minutes or at least 5 stages of incremental exercise had been undertaken. Starting speed was thus determined by training status and perceived velocity at exhaustion. The treadmill speed was increased by 1 $km.h^{-1}$

every 3 minutes, whilst pulmonary gas exchange and ventilation was determined using a breath-by-breath through low resistance mouth-piece and impeller turbine assembly attached to a face mask. Gas concentrations were determined through O_2 and CO_2 analyzers (Erich Jaeger, Oxycon Delta, LA Bunnick, The Netherlands) via a capillary line connected to the mouthpiece attached to a face mask. Gas exchange variables were calculated and displayed once volume and concentration signals had been accounted for. Calibration took place immediately before each test.

The treadmill used had two handrails, which were used by the participants to lower themselves onto the treadmill belt and gather the required leg speed upon starting the test (typically taking 2-4 seconds). Upon exhaustion the treadmill was stopped and the face mask immediately removed. Subjects were given the option of both fluid intake and a slow walk at approximately 2.5km.h^{-1} to aid recovery.

The attainment of $\dot{V}O_{2\,max}$ was confirmed by the incidence of a plateau phenomenon in $\dot{V}O_2$, RER values above 1.10, and heart rate within 5 b.min^{-1} of age-predicted maximum heart rate (Carter *et al*., 2002).

3.3 Determination of VO_2 kinetics

For the assessment of on-transient VO_2 kinetics, participants completed a 3 minute severe intensity exercise transition on the treadmill, thus the speed of the treadmill was equivalent to 110% of peak treadmill velocity attained during the $\dot{V}O_{2\,max}$ test. Transitions were repeated on separate days until a 95% confidence limit of ± 5s was

achieved and it could be regarded as an accurate representation of the kinetic response.

Subjects were fitted with a heart rate monitor on arrival to the laboratory and given 10 minutes to perform any static stretching they believed necessary, while trying to keep their heart rate as close to resting as possible as any dynamic, pulse raising exercise could be regarded as prior exercise which has shown to promote a faster VO_2 kinetic response to exercise (Burnley *et al*., 2002).

The test began with 2 minutes of motionless standing (feet astride the treadmill belt) for the measurement of resting VO_2. Subjects were warned of the proximity of the test 1 min, 30 sec and 10 sec before a final 5 second count where the subjects were allowed to use the handrails to support body weight whilst gathering the required leg speed that matched the treadmill belt velocity. A safety harness was fitted to any athlete who requested it. After the 3 minute bout of exercise, the treadmill was stopped and subjects were then also requested to remain as static as possible for recovery kinetics to be obtained over 5 minutes.

3.4 Pulmonary $\dot{V}O_2$ kinetic analysis

Due to its nature $\dot{V}O_2$ data emerges as 'noisy' when recorded as a low signal-to-noise ratio (S/N) exists which in turn has significant influence on the confidence of the kinetic parameters and their interpretation (Jones & Poole, 2005). So participants breath by breath responses were averaged every 5s, time aligned and then averaged

to enhance the signal to noise ratio (Lamarra *et al*., 1987). A single exponential that included a time delay was used to analyse the averaged response using least squares non-linear regression analysis (Graph Pad Prism, Graph Pad Software, San Diego, CA). Also Here any errant breaths caused by coughing, swallowing, talking could be excluded. Doing this would also enhance the underlying response characteristics present in the data. The primary response known as the 'Phase I response' exists during the first 20s of VO_2 data and characterisation of the phase II pulmonary $\dot{V}O_2$ exercise onset responses was achieved by fitting a single-exponential model including a delay term following the initial 20 s (to exclude phase I) of the exercise.

$$p\,\dot{V}O_2\,(t) = p\,\dot{V}O_{2\,ss}\,W\,(1 - e\text{-}(t\text{-}^{DE})/^{\tau})$$

where $p\,\dot{V}O_2\,(t)$, $\dot{V}O_2$ ss, *DE* and τ represent the value of $p\,\dot{V}O_2$ at a given time (*t*), the amplitude change in $p\,\dot{V}O_2$ from baseline to a new steady-state amplitude, time delay and the time constant of the response, respectively.

3.5 Statistical Analysis

The data was analysed using a T-Test in a statistical analysis software package to compare the two means of the time constant to obtain if any significance existed between the two populations.(Graph Pad Prism, Graph Pad Software, San Diego, CA). The level of significance was set at $P < 0{\cdot}05$.

CHAPTER IV
RESULTS

4.0 RESULTS

4.1 $\dot{V}O_{2\,max}$ & Heart rate

The parameters of the $\dot{V}O_2$ kinetic response in the transition to the same absolute severe work rate (110% of peak treadmill velocity corresponding to $\dot{V}O_{2\,max}$) are displayed in tables 1 & 2. The trained subjects were all aerobically fit on recruitment to the study, as reflected within the $\dot{V}O_{2\,max}$ 400-m 62.8±6.7 $ml.kg^{-1}.min^{-1}$ and 1500-metre 73.2±4.6 $ml.kg^{-1}.min^{-1}$. T-tests revealed significant difference (P value =0.017) in the $\dot{V}O_{2\,max}$ of the populations, representing a significantly higher $\dot{V}O_{2\,max}$ amongst the 1500-m athletes. Figures for amplitude of the $\dot{V}O_2$ response (A1) for the 400-m 3430±162.4 ($mL.min^{-1}$) are considerably lower than that of the 1500-m athletes where A1 = 3919±293.4. Both delay time (s) and time constant (τ) were faster amongst the 1500-m athletes. Although as displayed in table 3 below no variable was significantly different between the two populations. Final minute HR was also recorded for 400-metre athletes at a mean value of 174.8±0.7 bpm and 1500-metre mean HR of 170.4 ±5.4 bpm, which revealed significant difference (P-value 0.0075) between the populations. Although a significant difference existed between both heart rate and $\dot{V}O_{2\,max,}$ no difference (P value =0.3986 between heights, P value=0.0574 between weights) existed between the height and weight characteristics of the two populations competing in the different event categories.

Table 2, The Mean ± SD for all responses to treadmill running

Variable	400m (mean ± sd)		1500m		P Value	Significant Difference
A1	3430	±162.4	Mean:3919	SD±293.4	0.1474	NO
DE	6.041	±1.0	Mean:4.978	SD±1.2	0.5165	NO
TAU	22.14	±2.6	Mean:17.67	SD±1.3	0.1993	NO
VO2 MAX	62.8	±6.7	Mean:73.2	SD±4.6	0.0174	YES
HR (Final Minute)	174.8	±0.7	Mean:170.4	SD±5.4	0.0075	YES

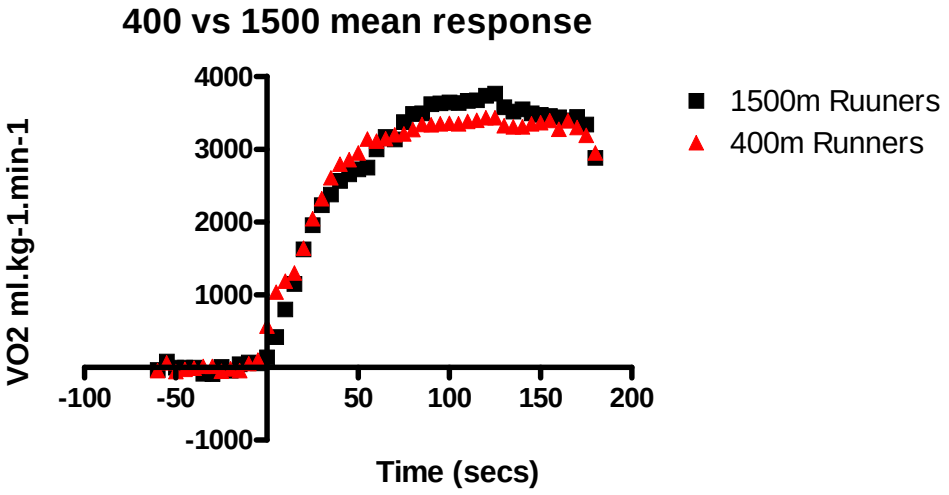

Figure 2. The two mean exponential for 400- and 1500-metre time constants in transition to obtain a steady state to the same absolute work rate (110% of VO_{2max}) during severe intensity running.

4.2 Comparison of the VO_2 on-transient response between 400-m and 1500-m athletes

When 400- and 1500-metre track athletes were compared it was clear that the 1500-m endurance trained athletes' elicited the lower τ (1500-m: 17.7±1.28 vs. 400-m: 22.1±2.56) The parameters of the $\dot{V}O_2$ on-kinetics in 400- and 1500-m athletes in severe intensity running transitions are displayed in table 3 and figure 1.

Although it is clearly visible from the mean τ exponentials in figure 2 and the mean values shown in table 3 that a clear difference exist between the two populations, T-test indicates no significant differences in the oxygen uptake response to severe intensity treadmill running between 400- and 1500-m athletes (P= 0.1993).

CHAPTER V
DISCUSSION

5.0 DISCUSSION

The rate of $\dot{V}O_2$ increase at the onset of supra-maximal treadmill running (Phase II, on kinetic response) did not differ significantly between the 400- and 1500 metre athletes , although a difference in mean TAU (**τ)** of 4.47 seconds did occur. Two significant differences that did emerge between the 400- sprint trained athletes and 1500-metre endurance athletes were in $\dot{V}O_{2\,max}$ and the H.R during the final minute of the exercise transitions. Similar results were found by Nummela *et al*, (1995) in their investigation of exhaustive running in anaerobic and aerobically trained athletes. This difference in $\dot{V}O_{2\,max}$ has previously been shown to have a significant effect on the time course of $\dot{V}O_2$ (Hickson *et al*, 1978).

5.1 Maximum VO_2 and oxygen uptake kinetics

Reasons for the 1500-metre athletes' possibly exhibiting faster oxygen uptake kinetics (although not significant) may be due to the elevated VO_{2max}. Studies have reported high correlation between $\dot{V}O_{2\,max}$ and $\dot{V}O_2$ kinetics (Chilibeck *et al*, 1996; Powers *et al*, 1985) therefore this could be an explanation in why an observed difference occurred in the time constant between 400- and 1500m athletes. The significant differences observed between the two populations' $\dot{V}O_{2\,max}$ could be attributed to training specificity causing differing cardiovascular and muscular physiological adaptations, subsequently increasing $\dot{V}O_{2\,max}$, thus possibly speeding the oxygen uptake kinetic response to exercise.

Athletes competing in middle distance events are known to have a high percentage of type I (slow twitch) muscle fibres (Bret *et al*, 2003). These fibres are known to be rich in mitochondria, which have shown to result in a more rapid adjustment of respiration to meet the energy demand of exercise (Saltin *et al*, 1977: cited in Powers *et al,* 1996). Although it does appear possible that individual differences may be caused a range of physiological factors. This was evident in that one subject in the 400-metre subject group who exhibited the fastest $\dot{V}O_2$ kinetic response (τ) in the entire study (JM $\boldsymbol{\tau}$=12.67 s, Appendix A) for reasons unknown to the researcher as the subject also displayed the lowest $\dot{V}O_{2\,max}$ value.

5.2 Physiological factors limiting oxygen uptake

Potential impediment for O_2 flux exists during several steps, ranging from O_2 in the atmosphere to the mitochondria. Physiological factors that could limit $\dot{V}O_{2\,max}$ in well trained track athletes are maximal cardiac output, oxygen carrying capacity of the blood and skeletal muscle characteristics. The initial two factors are classed as "central" whereas the fourth termed a "peripheral" factor (Bassett *et al*, 2000).

5.2.1Central Limitations

With reference to a lower $\dot{V}O_{2\,max}$ observed amongst the 400-metre population, a limiting factor that may determine the maximum O_2 consumption observed especially in sprint trained 400-metre athletes, may reside in the maximal capacity for carrying oxygenated blood to the muscles. In supra-maximal treadmill running involving all the muscles of the body, or a substantial fraction, the blood supply to the working muscles may approximate very close to cardiac output. Therefore any observed differences in the populations with different $\dot{V}O_{2\,max}$ characteristics are

likely to stem from the limitations of cardiac output and its component factors, namely ventricular volume and heart frequency (Margaria *et al,* 1965). The 1500 metre athletes are likely to exhibit higher levels of $\dot{V}O_{2\,max}$ as a consequence of circulatory and/or metabolic adaptations induced by the specific training schedules often performed by endurance athletes (Spencer *et al*, 2001). Further evidence for cardiac output limiting the 400-m populations $\dot{V}O_{2\,max}$ and perhaps the subsequent oxygen uptake kinetic responses, is that in the final minute of the exercise transitions; where 1500-metre athletes exhibited far lower Heart rates (HR). This could be attributed to endurance training performed by 1500-m athletes resulting in improvements to the factors contributing to cardiac output.

5.2.2 Central adaptations from training

Central adaptations incurred from endurance training facilitate improved delivery of oxygen to working muscles. Maximal heart rate has been found to remain unchanged in response to endurance training (Zavorsky, 2000) and improvements in oxygen delivery to exercising muscles during high-intensity exercise can be attributed to an increase in stroke volume. Stroke volume can increase through a higher left-ventricular contractile force and/or through an increase in cardiac filling pressure, which raises end-diastolic volume and resultant stroke volume (Laursen *et al,* 2002).

5.3 Peripheral limitations

It seems likely that various peripheral limitations exist for both sprint and endurance athletes. Peripheral limitations that exist with regard to maximum oxygen consumption are peripheral diffusion gradients, mitochondrial enzyme levels and capillary density all of which are functions of skeletal muscle. Reasons why peripheral limitations may differ between sprint and endurance trained athletes may be due to differences in skeletal muscle characteristics namely differences in fibre type. Holloszy *et al.*,(1975) reported that endurance training unlike sprint interval training has little/ minimum effect on glycolytic enzymes, therefore athletes who engage in SIT are likely to have less constraint on τ attributed to increased muscle enzyme activity (MacDougall *et al*, 1998)

O_2 transport from the lungs via the blood and its subsequent utilisation in the exercising tissues, coupled with the adjustments involved in these processes are governed by the intensity of the energy process that take place in the muscles, and not by the actual oxygen uptake in the muscles (Margaria *et al,* 1965). Thus attributing $\dot{V}O_2$ kinetic differences to metabolic inertia, cohering with results found by Whipp & Mahler (1980) cited in Kidling *et al* (2007).

Results from the study indicate that on-transient time constant could be determined by oxidative enzyme inertia within the muscle. Peripheral adaptations caused by exercise training refer to an improved ability of working muscle to produce and utilise ATP. It has been well documented that endurance training results in the physiological adaptations at cellular level that have been linked with faster τ and these are increased mitochondrial density and increased oxidative enzyme activity

(Gollnick *et al*, 1972; Costill *et al,* 1976 in Kidling *et al,* 2007). Given that a likely determinant of the τ is due to reduction in metabolic inertia, perhaps training induced adaptations are likely to speed τ at the onset of exercise.

5.4 Muscle fibre type and Oxygen uptake kinetics

The apparent differences in fibre type between sprint and endurance athletes could be a means by which peripheral limitations to oxygen uptake kinetics could be investigated. As middle distance athletes are known to present a higher percentage of ST fibres than sprint runners (Bret *et al,* 2003) therefore any apparent differences in τ could be explained by the mechanisms linked with fibre muscle fibre type.

Endurance events such as the 1500-metres are performed at an almost constant pace, and for those performed at the intensities below or close to $\dot{V}O_2$ max, mean performance power or speed is the product of $\dot{V}O_2$ max, the fraction of $\dot{V}O_2$ max sustained, and aerobic energy economy (di Prampero, 1986). Barstow *et al*, (2000) reported that a greater proportion of type I fibres and/or a high $\dot{V}O_2$ peak are equally associated with a greater ΔVo_2 / ΔW in exercise intensity domains above LT and proposed that this could be due to recruitment of type I fibres in subjects who are aerobically conditioned. Aerobic conditioning performed by middle distance athletes may consist of Interval training, at intensities around $\dot{V}O_{2\,max}$ (intervals lasting 2-10 min) improving mainly submaximal endurance performance (by ~6%) through improvements of all three components of the aerobic system ($\dot{V}O_{2\,max}$, anaerobic threshold and economy) (Paton *et al,* 2004) which also have been linked to speeding of the $\dot{V}O_2$ on kinetic response.

From glycogen depletion patterns within working muscle it has been found that type I fibres appear to be recruited first during exercise; only when exercise intensity or duration increases that type II fibres will be recruited (Barstow *et al*, 2000). This could explain faster $\dot{V}O_2$ on kinetics exhibited in athletes with higher percentage of the aerobic/ higher efficiency type I fibres. Considering this a coach or an athlete looking to improve performance could look at oxygen uptake kinetics within a sprint or endurance performance. An increased usage of type I fibres could offset the depletion of the muscles finite phosphocreatine (PCr) stores, and the accumulation of fatiguing anaerobic by-products (Jones & Poole, 2005).

5.5 400 vs. 1500, peripheral or central adaptations?

1500-metre athletes have the benefit of experiencing both central adaptations of increased stroke volume and cardiac output whereas 400m trained athletes may only experience training that induces peripheral adaptations, muscle oxidative capacity, and buffering capacity are among such adaptations. Sprint trained athletes may not receive the added extra of having a greater $\dot{V}O_2$ max where the oxygen is not only delivered in higher quantities induced from their training regimens, The metabolic inertia hypothesis proposes that the levels of cellular metabolic controllers and/ or enzyme activation predominantly limit the rate of adjustment of oxidative phosphorylation during the transition from rest to exercise (Grassi 2005). Recently research undertaken by Wilkerson *et al* (2004, 2005, 2006) has disputed that $\dot{V}O_2$ kinetics are principally limited by an O_2 delivery in the severe intensity domain. Therefore the metabolic inertia appears to contribute significantly to the regulation of $\dot{V}O_2$ kinetics during exercise in the severe intensity domain

5.6 Treadmill velocity effects upon oxygen uptake

Although treadmill velocity during all exercise transitions is determined by peak treadmill velocity attained during the steady state $\dot{V}O_{2\ max}$ test and is thus relative to each subjects upper limit of aerobic functioning, this could have become a methodological constraint of the study in that the 400 metre runners were running slower than the 1500 metre athletes during the supramaximal exercise transitions. Therefore this may not be an accurate representation of the 'in-race' uptake kinetics. A more appropriate design may have been similar to that of Spencer *et al* (2001) where 'event simulation' was created. A possible area for future research using similar athletic populations may be to compare the responses of set intensities with actual performance specific durations and velocities.

5.7 Training effects on oxygen uptake kinetics:

A relationship that has been reported between oxygen uptake kinetics and $\dot{V}O_{2\ max}$ is that oxygen consumption will increase at the onset of exercise but as a product of the intensity and training state: The higher the intensity the longer the time course, the greater the level of training the shorter the time course (Powers *et al*, 1996; Hagberg *et al,* 1978; Hickson *et al*, 1978; Whipp *et al*, 1972). Training-induced-enhancements in muscle blood flow and/ muscle oxidative capacity have been evidenced to have occurred in a number of studies (Barstow *et al*,2005; Jones & Carter, 2000; Jones & Koppo, 2005) and the current study used a cross sectional design, similar to that of Nummela *et al* (2005) to investigate the effects of event specific training upon oxygen uptake kinetics. A limitation that arises in this type of study, is that training

specificity & status of the athlete is assumed by the researcher i.e. 400-metre sprinters perform intense sprint interval training and 1500m athletes perform endurance based training. The time of which the study was conducted (November to January) may have also made this a significant limitation on responses exhibited by the subjects. Firstly the 400-m athletes who were competing during the indoor season were performing sessions focused upon indoor track running speed and were also tapering for indoor competitions, whereas other athletes in the same sample were performing "winter aerobic conditioning, in preparation for the outdoor season", as they were not competing during the indoor athletic season, subsequently creating greater variation within the 400-metre sample. This type of study is often utilised to look at comparison between extreme groups (i.e. sedentary individuals and elite athletes) therefore observing two event specific responses may require a longitudinal study to be effectively analysed. Here a training intervention could be imposed and a cause and effect of specific training analysed.

With regard to the time course of training-induced responses, it may be that high-intensity sprint training stimulates a more rapid up-regulation of selected physiological/ metabolic markers than previously believed, causing a smaller difference to exist in oxygen uptake kinetics between 400- and 1500-metre athletes. Burgomaster *et al*(2006) found that four to six 30 s sprints separated by 4–5 min of passive recovery undertaken 3 days per week results in comparable increases in markers of skeletal muscle carbohydrate metabolism. Similar training bouts are likely to occur in a 400 metre training programme, where 250-metre repetitions (lasting 30 seconds in collegiate level athletes) may be carried out, perhaps obtaining similar adaptations in a similar time course. The rapidity of these changes may have

also been an issue in regard to the current study; Athletes having to repeat transitions to achieve an accurate representation of their kinetic response did so over a two week period, training similar to that in the Burgomaster *et al* (2006) study could have caused significant changes within the athletes leading to insignificant results.

Reasons to why the interaction between training status and exercise intensity remained to be determined is that many of the cross sectional studies have looked at the differences between trained and untrained individuals. A strength the current study possessed is that it observed the oxygen uptake kinetic response between two event specific populations, using a cross sectional study design. This allows for training effects to be assessed & athletic population differences to be observed.

5.8 Study limitations

A longitudinal study design may have been more appropriate to investigate the differences in oxygen uptake kinetics in 400- and 1500-metre populations although highly impractical as many of the specialist athletes available for participation are already engaged in specific training programmes. The limitations of a cross sectional study design is that it does not account for the preceding sequence of events (namely training) to be accounted for, therefore no cause and effect relationship could have been evidenced. However to reduce this limitation subjects could have also been asked to keep training diaries for two weeks previous to participation to further understand any differences that emerged within the subject groups; as the recruited subjects came from two different training groups. Subjects JL, DC, and BO all came from a 400-metre training group that did a high amount of endurance based sessions (perhaps evidenced with greater $\dot{V}O_{2\,max}$ figures), whereas the remaining subjects in

the sample engaged in speed based repeated sprint training to improve 400-metre performance.

The study may have resulted in a significant difference between the mean τ between the two populations if more subjects had been recruited, this is evident in that Nummela & Rusko, (1995) and Draper & Wood, (2005) both reported non significant differences between SIT and END athletes when recruiting ≤16 subjects whereas Berger *et al*, (2005) reported significant differences in the same variable with a greater number of subjects.

Studies have already indicated that enhanced physical fitness (Caputo & Denadiai, 2004) or specific training interventions (Krustrup *et al*, 2004) leads to increased or altered $\dot{V}O_2$ uptake response to exercise. There is definitely scope for a study into the various training methods used for specific events, their subsequent effect on $\dot{V}O_2$ kinetics and eventually performance.

5.9 Conclusion

In conclusion, the observations made between 400- and 1500-metre track running populations' oxygen uptake kinetic response to supramaximal treadmill running, suggest that in the supramaximal intensity domain there is no significant difference that exists between the two populations (P= 0.1993) responses. Although an observed difference did occur between the populations, it could have been due to the significantly different factors that contribute to $\dot{V}O_2$ kinetic response, which are $\dot{V}O_{2\,max}$ and heart rate (H.R) in accordance with the results found by Chilibeck *et al*, (1996) & Powers *et al*, (1985).

Reasons why the τ did not exhibit significant difference could be due to variations in training undertaken by each individual athlete, perhaps evidenced by a difference of 16.7 ml·kg·min in the 400-metre $\dot{V}O_{2\,max}$ scores, arguably because a difference in the performance levels of the athletes, but this was not so as their event performance bests only ranged by just over half a second (50.08±0.65) respectively. But what these results more likely suggest is that even within a homogenous sample of athletes, different training methods used by their coaches will induce different physiological adaptations. Both types of training used in 400- and 1500-metre training programmes (SIT and END) have been evidenced to cause adaptations linked with faster $\dot{V}O_2$ uptake response to exercise (Gibala *et al*. 2006) and the extent to which each training programme can decrease both peripheral and central limitations may in turn effect the oxygen uptake response to severe intensity exercise.

The only way in which the responses could be viewed as significant is that the results indicate a degree of practical significance. Practical significance implies that the 4.47 sec difference observed in the τ between the two populations mean could be evident in race specific situations. A faster response time could elicit lower glycolysis rates within the athlete leading to lower levels of blood lactate. A faster oxygen uptake kinetic response then means lower intra muscular [PCr] degradation in the working muscle. An effect the previous two mechanisms will have is that it will facilitate a faster/ more powerful "kick-phase" in a 1500-m performance whilst allowing greater running form and economy to be observed in the final stages of a 400-metre race.

Future research directions that would add to the knowledge base in the field of oxygen uptake kinetics would be further research into specific track event responses to exercise and subsequent response times affect on performance.

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APPENDICIES

APPENDIX A

Table 4. All the mean kinetic responses to supramaximal intensity treadmill running.

Subject 400	**Amplitude (mL.min^{1})**	**Time Delay (s)**	**Time constant (s)**	**Subject 1500**	**Amplitude (mL.min^{1})**	**Time Delay (s)**	**Time constant (s)**
Subject 1-(AST)	3669.000	6.902	29.280	Subject 1-(AF)	4846.000	7.290	17.550
Subject 2-(BO)	3092.000	9.518	16.580	Subject 2-(HJ)	4094.000	1.679	22.300
Subject 3-(DC)	3504.000	8.447	20.400	Subject 3-(PB)	3574.000	7.364	15.860
Subject 4-(GF)	2763.000	3.934	28.170	Subject 4-(TM)	3079.000	6.151	17.800
Subject 5-(JH)	3796.000	3.403	29.280	Subject 5-(TP)	4004.000	2.408	14.860
Subject 6-(JL)	3980.000	2.712	18.570				
Subject 7-(JM)	3208.000	7.369	12.670				
Average - 400m	3430.3	6.0	22.1±2.56	Average - 1500m	3919.4	5.0	17.7±1.28

APPENDIX B

UWIC PARTICIPANT CONSENT FORM
UWIC Ethics Protocol Number:

Participant name or Study ID Number:

Title of Project:

Name of Researcher:

Participant to complete this section: Please initial each box.

1. I confirm that I have read and understand the information sheet dated ..... for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my relationship with UWIC, or my legal rights, being affected.

3. I understand that relevant sections of any of research notes and data collected during the study may be looked at by responsible individuals from UWIC for monitoring purposes, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.

4 I agree to take part in the above study.

_____ _____

Signature of Participant

Date

_____ _____

Name of person taking consent Date

_____

Signature of person taking consent

_____

APPENDIX D