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**THE EFFECT OF NORMOBARIC HYPOXIA ON SKELETAL
MUSCLE OXYGENATION IN 400M AND ENDURANCE
RUNNERS**

**(DISSERTATION SUBMITTED UNDER THE DISCIPLINE OF
PHYSIOLOGY AND HEALTH)**

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**The effect of normobaric hypoxia on
skeletal muscle oxygenation in 400m and
endurance runners**

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Abstract

Skeletal muscle adaptations are known to occur when chronic physiological stress such as training occurs. Type I to type II transitions have been verified by research going in both directions. With the high oxygen uptake of type I fibres it can be understood that this can cause and increase in systemic O₂ reduction during exercise, with it being exemplified in hypoxia. Leading to a hypothesis that a lower incidence of type I fibres, could allow for greater O₂ management in hypoxic conditions. EN and SPR represented type I and type II muscle fibres respectively, due to their self reported training modality.

During this study of 7 EN (mean age 29± 4.93 yr, height 180.86±6.09 cm, body mass 76.56±4.98 kg) and 5 SPR (mean age 21.8±2.86 yr, height 181.94±1.99 cm, body mass 74.86±2.72 kg), a supine cycling, sub-maximal protocol was followed.

NIRS was utilised to measure THC and S_mO₂ of the VL. It was observed that there was no main effect of training status ($p>0.05$) on THC in both hypoxia and normoxia. S_mO₂ in hypoxia presented no significant difference in all statistical testing ($p>0.05$). Normoxic conditions presented an inclination to a relationship between S_mO₂ and training status ($p=0.0688$), graphically identifying SPR attaining lower S_mO₂.

These findings support the hypothesis that in hypoxia, a high incidence of type I fibres causes a greater reduction in S_mO₂. And infers that due to a lower prevalence of type I in SPR, they can perform in hypoxia to a greater extent as a result of a lower rate of O₂ utilisation. As a result of these findings, sports performers could be informed about the possible effects of their training on their ability in hypoxia. Allowing for modifications prior to performance such as an increase in anaerobic exercise.

CHAPTER I
INTRODUCTION

1. Introduction

Muscular oxygenation plays a vital role in the ability to perform an exercise. To perform an action, skeletal muscle primarily requires oxygen. Therefore oxygen availability is often a determining factor of exercise performance. Endurance athletes rely on a suitable oxygen delivery for exercise performance for prolonged periods of time. Moreover, anaerobic athletes such as sprinters, rely on other energy systems to produce physical actions such as running, but due to inefficiency these bouts are often low duration. Both training styles of the athletes mentioned produce above average $\text{VO}_2 \text{ max}$ values, with this in mind other factors must affect the ability to perform in low oxygen (O_2) conditions.

1.1 Hypoxia

When a human body enters conditions of hypoxia, a reduced availability of oxygen to vital organs, skeletal muscle and blood is experienced. Multiple acute adaptations occur upon introduction to a hypoxic stimulus even at rest, in cardiovascular physiology an increase in heart rate (HR), increase in cardiac output and decrease in stroke volume (Thompson *et al.*, 2006). Alongside these cardiovascular adjustments, ventilatory responses occur. Ventilation rate adjustments occur 18 – 23 seconds after exposure (Weil *et al.*, 1970), when introduced to hypoxia this causes ventilation to increase, again a homeostatic response to maintain S_aO_2 .

Hypoxia has been employed as a training stimuli for decades, utilised by athletes for improvements in sea level performance and mountaineers for preparation for altitude (Millet *et al.*, 2010). Ever improving technology has now provided the opportunity to create normobaric hypoxic conditions at sea level, this opens up an athletes' options when it comes to the implementation of hypoxia during physical training. Intermittent hypoxic training (IHT) is often used in either Live High, Train Low (LHTL) or Live Low, Train High (LLTH) training regimes. A meta analysis (Bonetti & Hopkins, 2009) attempted to identify the most beneficial style of altitude training. The findings concluded that LHTL was the most effective in improving exercise performance for elite athletes. Although inconclusive, sub-elite athletes showed small improvements in most styles of altitude training.

It is well established that training in altitude can illicit significant improvements in maximal aerobic capacity, identified as VO_{2max} (Melissa *et al.* 1997). Furthermore, Ponsot *et al.* (2006) identified a significant VO_{2max} increase, in response to six weeks hypoxic training. With these adaptations becoming evident post training, it highlights the additional physiological stress that is hypoxia. More recent publications have begun to research the affect of hypoxic training on team sport athletes and anaerobic athletes alike. Similarly, to the findings of Ponsot *et al.* (2006), Hamilin *et al.* (2009) identified that with 10 days of IHT, significant increases in anaerobic power can be indicated. This presents the idea that both endurance trained (EN) and sprint trained (SPR) runners are affected by hypoxic stimuli.

1.2 Skeletal Muscle Adaptations

Morphologically, endurance trained and sprint trained runners are different when compared to both each other and untrained populations (Uth, 2005). The skeletal muscle adaptations that both training status' generate are the factors that could affect performance in hypoxia. Adaptations of the skeletal muscle and its oxidative capacity could determine an athlete's performance level when in a hypoxic environment. Current research suggests that an athlete who is EN will observe a high rate of muscular oxygen utilisation, due to muscle fibres being primarily oxidative type I. Research by Van Thienen and Hespel (2015) supported this by comparing endurance trained individuals and normal healthy individuals. SPR individuals may benefit from having a lower incidence of type I fibers (Saltin *et.al* 1977; Jansson *et al.* 1990) as they have a lower oxidative capacity.

1.3 Oxygen Consumption during Exercise

Through respiratory control, oxygen is delivered to the body via the cardio-respiratory system. To sustain normal functioning of the human body, oxygen consumption must meet the oxygen needs. A VO_{2max} test highlights the maximum to which a individual can physically work until fatigued. Maximal oxygen consumption is well known for being a key determining factor for exercise performance. The Fick equation being $VO_{2max} = Q \times (C_aO_2 - C_vO_2)$ highlights that a larger a- VO_2 difference would result in a larger VO_{2max} (McArdle *et al.*, 2015). It is well established that oxygen consumption increases with exercise, this is unavoidable due to the

metabolic demands of skeletal muscle for O₂. It has been widely established that muscle fibre types differ in their oxygen uptake kinetics, so fibre recruitment and proportions could affect a-VO₂ difference. Exercise intensity elicits different VO₂ responses, four categories can be identified; moderate where there is a linear increase until steady state VO₂, heavy exercise causes an initial increase in VO₂ until steady state exercise is achieved. The final two intensity groupings are severe and extreme, both of these achieve exhaustion yet only severe achieves VO_{2max} as it is deemed that extreme exercise causes fatigue prior to VO_{2max} (Espada *et al.*, 2015).

1.4 Arterial Blood Oxygenation and Exercise

Both skeletal muscle oxygenation and arterial blood oxygenation are indicators of the physical activity and its stress on the body. Exercise induced arterial hypoxemia (EIAH), defined as a low oxygen content in the arterial blood, has often been observed in elite EN athletes at sea level (Durand *et al.* 2000). Small decrements in arterial blood oxygen saturation (S_aO₂) have been identified as major causes of a reduction in VO_{2max} especially in athletes with already high VO_{2max} scores (Hopkins & McKenzie, 1989). A small decrement in S_aO₂ has the ability to increase the rate to which peripheral muscle fatigue, Romer *et al.* (2005) highlighted that minimal S_aO₂ reduction (<10%) lessens muscular fatigue rate.

1.5 Muscle Oxygenation and Exercise

Skeletal muscle saturation is dependent on two factors, rate of oxygen delivery and the rate of oxygen consumption (Belardinelli *et al.*, 1995). Bhambhani (2004) presented a four phase model on skeletal muscle oxygenation (S_mO₂) during incremental exercise and recovery. Identifying an increase at the start of exercise, followed by exponential decline. Nearing maximal exercise, a plateau can be observed with rest causing S_mO₂ to rapidly increase often to above resting levels. When exposed to hypoxia, S_mO₂ declines even in dormant skeletal muscle. When exercise is introduced, de-oxygenation occurs to a greater degree in comparison to normoxic conditions (Dinunno, 2015).

1.6 Rationale

Recently outlined in research by Van Theinen and Hespel (2015), a distinctly higher number of type I muscle fibres will cause an increase in the O_2 uptake inside the muscle. Identified by multiple publications, an effect of continuous or long term endurance training is that the ratio of type I fibres increases (Coyle, 1995.; Kubukeli, Noakes, and Dennis, 2002). Findings of Van Theinen and Hespel (2015) were of a more clinical nature, in that they found the EN individuals were more likely to suffer from acute mountain sickness whilst at high altitude, due to the higher incidence of type I muscle fibres. In this field, the research on sprinters is currently minimal.

Similarly to EN, SPR have been observed experiencing greater reductions in both S_aO_2 and S_mO_2 (Oguri *et al.*, 2008). This infers that there is a negative training effect on exercise in acute hypoxia. The oxidative nature of type I fibres and anaerobic ability of type II, the fibre composition of athletes could be an influential factor when it comes to performance at altitude. This being due to evidence of both S_aO_2 and S_mO_2 decrements adversely effecting performance.

Furthermore, it has been identified that high intensity sprint training causes muscle fibre transitions opposite to what is observed in endurance training (Jansson *et al.* 1990). With sprinters having a lower incidence of type I fibres (Saltin *et al.*, 1977), it presents the theory that they could potentially perform to a higher degree than their endurance trained counterparts in hypoxia. Hypothetically if a muscle utilised a reduced amount of O_2 whilst performing, the athlete would be able to perform to a greater degree or continue for an extended period of time. Hence this study aims to identify if the training status of an athlete affects muscular oxygenation during exercise in hypoxia.

CHAPTER II
REVIEW OF LITERATURE

2. Literature Review

2.1 Hypoxia

Hypoxia is the term used when there is a reduction in the total oxygen availability (Nikinmaa, 2013). Prior to technical advancements, hypoxia was solely experienced when an ascent to altitude was made. With and ever increasing decrement of oxygen availability being observed as altitude increased. The reduction in overall barometric pressure (P_{bar}) drops, although oxygen fraction remains at 21%. Through technological advancements, reducing the overall oxygen fraction (F_{IO_2}) is now an option, thus the creation of two types of hypoxia. Hypobaric and normobaric hypoxia are experienced at altitude and sea level respectively. Normobaric hypoxia differentiates from hypobaric as the fraction of oxygen (F_{IO_2}) available is reduced, instead of an overall reduction of barometric pressure. A comparative study by Millet *et al.* (2012) attempted to identify if the responses observed in both hypoxic conditions differentiates. The research proposed that hypobaric hypoxia presents a more severe physiological stressor when compared to normobaric hypoxia. Findings identified inside the research, supported the proposition that hypobaric hypoxia was more severe, but the reasoning still lacked clarity, this was advocated by Van Theinen and Hespel, (2015).

At rest, hypoxia will cause acute physiological adaptations, this is the human body attempting to maintain homeostasis and counteract the adverse hypoxic stimuli. This occurs in both normobaric and hypobaric hypoxia (Millet *et al.*, 2012). An increase in HR and cardiac output has been identified by Thompson *et al.*, (2006), this is a result of a systemic reduction of available oxygen in the blood, represented by S_{aO_2} .

2.1.1 Exercise in Hypoxia

The physiological stressor that is hypoxia has long been identified to impinge aerobic performance. With the high O_2 requirements of aerobic exercise a reduction in O_2 availability will cause the exercise to become more demanding. When it comes to elite performance this can be problematic. Broadly two areas of interest occur, one being the physiological effect that hypoxia has on performance during exercise. The other being whether or not hypoxia can be utilised as a tool for performance enhancement. A key turning point in what is known about exercise at altitude was the 1968 summer Olympic Games, which was held in Mexico City. The altitude of

2,300m was attributed with affecting performance in most events. World records were set in sprint and jump events, whilst in aerobic performance many world class athletes performed distinctly slower (Wilber, 2003). With 2,300m offering roughly 16% effective oxygen (F_{IO_2}), it is a stark difference for athletes who train at sea level (21% F_{IO_2}) (Altitude-chart, 2015).

Hypoxia itself can cause immediate acute physiological adaptations. The respiratory system will immediately increase pulmonary ventilation to counteract the lower availability of O_2 . This compensatory mechanism occurs due to chemoreceptors in the carotid artery and aorta which detects a lower incidence of O_2 (Armstrong, 1999). Cardiovascular changes to acute hypoxia are preceded by an increase in heart rate and cardiac output, with a small reduction in stroke volume (Thompson et.al., 2006). This coincides with the increased ventilation in an attempt to ensure the muscular system's demand for O_2 is achieved, and removal of waste such as CO_2 is managed.

The Mexico City Olympics gave precedence for the use of altitude training and acclimatisation. Athletes who competed for countries located in higher altitude's had increased performance levels in middle and long distance events. The use of altitude to improve exercise performance has now been implicated across many endurance athletes training programmes and has become the basis of a large research area. Prolonged exposure to hypoxia or altitude, causes the physiological systems of the human body adapt to mitigate the difference in O_2 . Hoppler, Klossner and Vogt (2008) acknowledged that exercise in hypoxia caused a greater stress on skeletal muscle. If attempting to improve sea level performance or as apart of an acclimatisation procedure, this finding is very beneficial to an athlete. If preparing for performance at altitude, an increase in intensity experienced can be expected.

2.2 Blood Oxygen Saturation

It is firmly recognised in research that there is an effect of exercise on the oxygen saturation of the blood. This is caused by an increase in muscular O_2 uptake. At high intensities exercise performance is limited by the pulmonary systems ability to maintain O_2 saturation in the blood (S_aO_2). The amount of oxygen available in the blood has been shown to correlate with physical performance. Even small reductions in S_aO_2 have the ability to reduce the VO_{2max} attainable to an athlete with a typically

high VO_{2max} (Hopkins & McKenzie, 1989). Blood oxygen saturation (S_aO_2) has been known to consistently be affected by a hypoxic stimulus. Without exercise, hypoxia alone has the ability to reduce S_aO_2 . A publication by Kovtun *et al.* (2011) found that during rest, alternating hypoxic gas and normoxic gas for five minutes each helped identify the effect of hypoxia on S_aO_2 . This suggests that for aerobic events especially where exercise needs to remain below the lactate threshold for a sustained period of time, hypoxia would produce reductions in performance as seen by Hopkins and McKenzie (1989). Research has identified EN athlete's as being highly susceptible to hypoxemia, both at altitude (Van Theinen & Hespel, 2015) and at sea level (Durand *et al.* 2000). In the sea level experiment, the group that experienced EIAH the greatest were the EN athletes that had the highest training volume. Understanding this, it highlights possible training adaptations as being key factors when investigating performance in hypoxia. Oguri *et al.* (2008) investigated the effect of hypoxia on SPR males, it was discovered that along side EN, S_aO_2 of SPR athletes reduces to a greater degree in both hypoxia and normoxia when compared to untrained individuals. This further propels the theory that physical training causes adaptations to which could hinder performance of an un-acclimatised athlete in hypoxia, however the degree to which SPR compares to EN still lacks research.

2.3 Skeletal Muscle

Skeletal muscle is the functioning force behind almost all physiological movements of the human body. Constructed with a combination of muscle fibre types, a muscle can vary in its physiological ability. Skeletal muscle itself has shown to be highly adaptable to the modus of stimuli it is exposed to (MacIntosh, McComas, and Gardiner, 2006). In broad terms, skeletal muscles contain a mixture of Type I and Type II muscle fibres, these also being referred to as slow twitch and fast twitch respectively. The reason for the classification is due to their inherent physiological features. Originally the categories were created by measuring the shortening speed of the muscle, but morphological differences such as colour were observed. A white muscle fibre would be classified as a Type II fibre, due to its reduced content of myoglobin and capillaries. Type I fibres are a red in colour due to a high myoglobin count and an increased capillary density (Scott *et al.*, 2001). Type I fibres are primarily oxidative, whilst Type IIb being the most anaerobic (Gueguen *et al.*, 2005).

Both broad classifications of fibre types serve as beneficial to different specialisms in sport, these biological differentiations allow for alternate performance potentials. For example, EN athletes would benefit greatly from having a higher proportion of oxidative type I fibres as the majority of their exercise occurs under the ventilatory threshold where oxygen demand is satisfied.

A more recent publication found that human Type II skeletal muscle could be divided into three sub-categories. These being Type IIa, Type IIx and Type IIb. With Type I preceding them all, Type II fibres can be ranked a, x, b in reference to muscle shortening capabilities, with Type IIb being the fastest (Pellegrino *et al.*, 2003). There are studies which have observed physiologically adaptive differences between sporting populations. Training status' which have been identified for their potential adaptive differences are EN and SPR, who in terms of running have polar opposite aims in terms of running style and performance. An early piece of literature by Saltin *et al.* (1977) detected that sprinters often have a lower incidence of slow-twitch fibres when compared to other power orientated athletes. In support of this finding, Jansson *et al.* (1990) found that with high intensity training, adaptations inside their thigh musculature occurred. Post training, the results exhibited changes in both Type I and Type II muscle fibres, with a decrement of 9% and an increase of 6% respectively. As seen in the paper by Pellegrino *et al.* (2003), Type II fibres have a larger potential for explosive movements due to their speed of muscle shortening, this would immensely benefit an athlete such as a sprinter. Findings by Cristea *et al.* (2008) have shown evidence to limitations of muscle fibre type ratio adaptations. In this publication, older elite sprint athletes formed the subject group who underwent a 20-week training program. The exercise group exhibited no changes in the muscle fibre proportions, thus inferring that there is a limit to the adaptive potential of the skeletal muscle, and that a higher training status can cause an intolerance to adaptation.

Endurance trained athletes have been a main focal point of existing literature across all aspects of sports science. Their ability to maintain exercise for prolonged periods of time has shown to produce adaptations such as higher maximal oxygen consumption (VO_2), increase in percentage (%) of Type I muscle fibre type and muscle capillary density (Coyle, 1995.; Kubukeli, Noakes, and Dennis, 2002).

Although relatively dated, a publication by Fink *et al.* (1977) proved invaluable in the findings it presented, and has multiple citing's in present research. The subject groups used were world class, good and untrained and there were significant observations made in terms of slow twitch muscle fiber proportions. The world class and good endurance runners exhibited higher values of slow twitch fibres, which are stated as being of a higher oxidative capacity. Although beneficial in normoxia, this high precedence of oxidative fibres has been identified as a key detrimental factor to exercise performance in hypoxia (Van Theinen & Hespel, 2016). Results presenting prompter acute mountain sickness symptoms (AMS). Having a lower oxidative muscle fibre type such as Type II could be beneficial when performing at altitude. Evidence to support this comes from a study looking at muscle fibre structure of untrained high altitude dwellers. Hoppeler and Vogt (2001) highlighted that untrained residents of high altitude had a lower oxidative capacity in the muscle, compared to a lowland population. With this, it can be inferred that having a low oxidative skeletal muscle structure is beneficial at altitude as it could assist with O₂ balance inside the body.

2.3.1 Skeletal Muscle Oxygen Saturation and Exercise

It has been well established in research that during exercise a decrement in skeletal muscle oxygenation can be detected in the initial stages (Kawaguchi *et al.*, 2006). Supporting this, Bhambhani (2004) identifies four key stages of skeletal muscle oxygenation during incremental exercise. An initial increase in S_mO₂ is experienced, it has been suggested that this is caused by a redistribution of blood flow to the skin (Chuang *et al.*, 2002). Following this, an exponential decrement of S_mO₂ occurs. Prior to exhaustion, S_mO₂ is seen to plateau, often until VO₂ max is achieved. Post exercise a rapid increase in S_mO₂ is observed, in this publication S_mO₂ is seen to rise above resting baseline values. The relationship between muscle de-oxygenation and percentage of VO₂ max is evident in a study by Hiroyuki *et al.* (2002) who observed significant decrements in muscle oxygenation of both the vastus lateralis (VL) and gastrocnemius (GL). Observations were also made in terms of exercise intensity, in this case running speed and muscle oxygenation. As the speed increased a decline of S_mO₂ occurred in both VL and GL, the decrement seen in GL was larger than that of VL. This potentially infers that the more peripheral a muscle, the greater the degree of muscular de-oxygenation. Furthermore, the relationship of intensity and

ventilatory threshold have been found to produce alternate results whether above or below the threshold. Steady state exercise below the ventilatory threshold has prompted an initial reduction in S_mO_2 , followed by a gradual linear increase until the end of exercise (Chuang *et al.*, 2002). The initial reduction can be affected by the athletes VO_2 kinetics, for example their speed of recruitment of type II fibres to cope with a physical stimulus (Poole *et al.*, 2008). If an athlete has been exposed to large amounts of endurance training, the higher oxidative capacity of the skeletal muscle could result in a reduction or delay in the recruitment of type II fibre's, therefor maintaining a high oxygen uptake.

Haemoglobin's role in the blood has long been identified as a determining factor for survival due to its O_2 transportation properties (McArdle *et al.*, 2015), with its primary function of delivering O_2 around the body. Male's have an elevation of haemoglobin of roughly 10%, this has been attributed to the hormone testosterone. Both increases and decreases of THC have been attributed to increase and decreases in VO_{2max} respectively (Otto *et al.*, 2013). Although in this publication both these alterations were created by either the supplementation of erythropoietin (EPO) or the extraction of blood. EPO being the precursor to haemoglobin production. Studies have revealed a relationship between low S_aO_2 and the kidney organ's ability to produce EPO (Mairbäurl, 2013). With hypoxia inducing a lower S_aO_2 , the reasoning behind altitude training is transparent. During exercise it has been noted that THC remains relatively consistent throughout. Inside skeletal muscle, total haemoglobin concentration and oxygenated proportions are of interest, although current research has indicated that S_mO_2 may be of more significance. In continuous cycling, an increase in oxygenated haemoglobin can be observed (Kawaguchi *et al.*, 2006), this contradicts the literature that identifies skeletal muscle de-oxygenation during exercise. This increase has been attributed to an increase in skeletal muscle blood flow, similar to that seen in Bhambini's (2004) S_mO_2 increase at the initiation of exercise. It was also postulated that an overall rise in THC can be an effect of higher intramuscular pressure during exercise, where contracting skeletal muscle negatively effects capillary blood flow (Hiroyuki *et al.*, 2002).

2.3.2 Skeletal Muscle and Hypoxia

Hypoxia can elicit both positive and negative adaptations to skeletal muscle. Chronic exposure to hypoxia has been attributed to a decline in skeletal muscle mass, where it was previously thought during ascent to altitude this atrophy occurred due to lower caloric intake and increased energy expenditure (Deldicque & Francaux, 2013). Additionally, this research proposes that acute hypoxia can cause physiological adaptations such as increase's in protein synthesis, growth hormone and type II fibre recruitment. The latter potentially correlating to the increases in performance for sprint and jump events at the 1985 Mexico City Olympic Games. Maximal exercise in hypoxic conditions are relatively well researched, with a major study by Martin *et al*, (2009) conducting a maximal ramp exercise protocol at various altitudes. It was shown that increasing altitude caused a greater decrement in S_{mO_2} . Further identified by Martin *et al*, (2009), a reduced resting S_{aO_2} caused S_{mO_2} to decrease at a much faster rate. This was attributed to two factors; a dampened hypoxic ventilatory response where ventilation is not sufficient to attain sea level values of S_{aO_2} . Further assisted by an increase in arterial-alveolar O_2 partial pressure difference.

2.4 Aims and Hypothesis

As outlined by both Van Theinen and Hespel (2015) and Oguri *et al*. (2008), both EN and SPR athletes are physiologically affected by a hypoxic stimulus to a greater degree than untrained individuals. Attributing this to a higher oxygen uptake during exercise causing larger decrements of S_{mO_2} and S_{aO_2} .

With the research identifying SPR as having lower incidences of type I fibres (Saltin *et al*., 1977; Jansson *et al*.,1990), it could be postulated that SPR would perform closer to typical sea level performance in hypoxia than EN athletes. The hypothesis being that at sea level EN would deoxygenate less than SPR during exercise due to high oxidative capacity of skeletal muscle. Whereas in hypoxia both groups would perform to a similar degree.

CHAPTER III
METHODOLOGY

3. Methodology

3.1 Participants

12 healthy, active males volunteered to participate in this study. Of the 14 volunteers, 7 were endurance trained (EN) individuals (mean age 29 ± 4.93 yr, height 180.86 ± 6.09 cm, body mass 76.56 ± 4.98 kg) who ran a minimum of 40km per week, the remaining 5 were self identified 400m sprinters (SPR) (mean age 21.8 ± 2.86 yr, height 181.94 ± 1.99 cm, body mass 74.86 ± 2.72 kg) of a good standard (< 50 s /400m). Information about the opportunity to participate in the study was circulated around the University student population, local running clubs and individual coaches via a participant information sheet. Word of mouth inside the athletics community was also used to distribute the study.

Prior to each test, anthropometric data consisting of body mass (kg) and height (cm) was collected. Weight was measured on digital scales (Vogel and Halke, Seca Model 770, Hamburg, Germany) and height determined via a fixed stadiometer (Holtain, Holtain Fixed Stadiometer, Pembrokeshire, UK). Each participant ensured accurate measurements by removing shoes and socks and any superfluous clothing bar shorts and t-shirt.

Table 1. Anthropometric characteristics of participants.

Descriptive	Age (yr)	Height (cm)	Mass (kg)	VO_{2peak} (ml/min/kg)
Endurance	29.00	180.86	76.56	43.29
En St Dev	4.93	6.09	4.98	5.61
Sprinter	21.8	181.94	74.86	44.16
Spr St Dev	2.86	1.99	2.72	3.63

Each participant was fully informed about the testing protocol and associated risks they were potentially volunteering for. The information provided, originated with the participant information sheet and was offered alongside the opportunity to ask any questions related to the study over email or in person. Health questionnaires which followed the guidelines set by American College of Sports Medicine Pre-Participation Screening Questionnaire (2006) were completed prior to both testing

sessions, providing information on the volunteers past and present general health status and level of physical activity. Enrolment onto the study would begin once the ACSM forms are completed with no more than one risk factor stated; if a risk factor was disclosed, the participation of the volunteer would be at the lead investigators discretion. If at any stage prior to or during testing should the participants' health raise any concerns, the test protocol will be halted and the participant be made aware of the issues that have arisen. The participant will then be recommended to visit a medical practitioner to ensure their wellbeing is sustained. The participant was also made fully aware that they reserved the right to remove themselves from the study at any stage without consequence.

3.2 Exercise Protocols

For the purpose of this study, the participant would be required to attend two sessions, firstly a VO_{2peak} , the second being two sub maximal incremental tests in normoxia and hypoxia. Both sessions were scheduled with at least two days' rest in between, but no more than two weeks. Each session followed the protocols as described below.

3.2.1 Visit One

The first visit consisted of a VO_{2peak} test to determine the maximal aerobic fitness of the participant, as expressed by their maximal oxygen consumption (VO_{2max}) value. This visit was required so the fitness level of the participant could be determined, allowing visit **two** to be relative to the participant's physical fitness. Immediately before the participant mounts the ergometer, a respiratory mask was fitted ensuring no air escaped. Furthermore, a wireless heart rate monitor chest strap (Polar Electro, FT1, Kempe, Finland) was fitted to the participant, and calibrated with the watch. The test was carried out on a LODE ergometer fixed to a supine bed which whilst cycling would be tilted to 45° (Cardiac stress table, Lode Medical Technology, Groningen, The Netherlands). Due to the study forming the basis of multiple research objectives, one being cardiac orientated, the 45° tilt was required for echocardiography. Ergonomic modifications were made to the tilt bed to ensure the participant's comfort. Pillows were used to support the head and left shoulder. The LODE ergometer was adjusted to ensure the participants knee was not extending maximally when cycling. A breath-by-breath analysis system (Oxycon Pro, Jaegar at

Viasys Healthcare, Warwick, UK) used to continuously record VO_2 data was affixed to the participant's respiratory mask. Calibration of this equipment was carried out prior to testing, with current ambient conditions being considered.

Once the participant was content with the supine cycling position and deemed ready to proceed, the VO_{2peak} test would begin at 40 watts (W) for three minutes. Once the warm up was completed, the resistance would increase immediately by 40W and continue to incrementally increase by 40W at 3 minute intervals. The participant would be verbally motivated to continue exercising, until volitional exhaustion. This being classified as the point to which the participant cannot maintain a cadence of between 60 and 70 RPM.

On cessation of the test, resistance was dropped to 40W and the end time and power output logged. The breath-by-breath analysis was stopped and a final heart rate was noted. Whilst the participant begins to cool down, the bed is tilted back to parallel and the respiratory mask removed. Warm down time was participant specific, the research investigators provided ample time for the participant to recover. A period of monitoring occurred to ensure the well being of the volunteer. Rehydration was recommended before leaving the laboratory.

3.2.2 Visit Two

Before the participant proceeds with the protocol, they were asked if their physical wellbeing had altered since visit one. The participant will be required to fill in the same ACSM questionnaire. Prior to testing the anthropometric data was recorded, using the same equipment as visit one. The participant would then lie on the tilt bed and establish a comfortable position, from this position all equipment was put in place. As experienced in visit one, during exercise the supine bed will be tilted at 45°.

3.2.2.1 Respiratory Data

In both exercise conditions the same breathing apparatus was used to allow for a single blind exercise protocol. This was to ensure the volunteer's physical performance was not influenced by the awareness of a hypoxic or normoxic condition. The participant would be required to wear a mouth piece connected to a two-way respiratory valve (Salford Valve, Two-way breathing valve, Birmingham,

UK) and breath-by-breath gas analysis system as used in visit one. A large bore breathing tube was connected to a Douglas bag (Cranlea & Company, Bournville, Birmingham, UK.) filled with a hypoxic gas during the hypoxic trial and left open to allow normal air to pass through during the normoxic trial. Vacuumed Douglas bags were filled prior to testing with a ~12% oxygen gas (Higher Peak performance, McKinley altitude simulator, Staffordshire, UK). An acclimatisation period of ten minutes was carried out before undertaking the exercise so that the participant was normalised in the breathing condition. This occurred in both conditions to ensure a single blind protocol.

3.2.2.2 Heart Rate

The ultrasound (Vivid E9, GE Vingmed Ultrasound, Horten, Norway) instrument used for cardiac imagery allowed accurate heart rate data to be recorded throughout the entire protocol with a three lead electrocardiogram (ECG). In support of the ECG, pulse oximetry also provided a HR measurement.

3.2.2.3 Arterial Blood Oxygen Saturation

A pulse-oximeter (Autocorr 3304, Smiths Medical PM inc, Waukesha, USA) was placed on the participants' middle phalanx on the right hand. Arterial blood oxygen levels were monitored throughout the entirety of the volunteer's participation. If S_aO_2 fell below 60% for a significant period of time, the protocol would be stopped and the participant would be allowed to breathe normoxic air.

3.2.2.4 Skeletal Muscle Oxygen Saturation

Near Infrared Spectroscopy (NIRS) was used to monitor and record the total haemoglobin concentration (THC), oxygenated haemoglobin (HbO) and deoxygenated haemoglobin (Hb). The ratio of THC and HbO can be presented as a percentage (%) to clearly display overall affect of exercise and condition on the participant's muscle oxygen saturation (S_mO_2). The participants leg hair proximal to the VL, was shaved to reduce the interference with the NIRS. The NIRS receiver was placed on the VL and then wrapped with medical bandaging to ensure no light pollution affected the probe.

3.2.2.5 Visit Two Exercise Protocol

Following the acclimatisation period, the participant would commence cycling. A cadence between 60-65 RPM was instructed. A 3-minute warm-up at 40W was followed by three 5-minute exercise bouts at 30%, 40% and 50% of their peak power output's (PPO) observed in the Visit 1 VO_{2max} . Once the first exercise session was complete, the participant was allowed off the supine bike and given thirty minutes to rehydrate and rest. The second session was then repeated identical to the first, the difference being the air condition they would breathe. After the second exercise session was complete, all equipment was removed and the participant was allowed to cool down on a low intensity. Furthermore, each participant was monitored for their own wellbeing and it was suggested that they rehydrate.

3.3 Data Analysis

Microsoft Excel (Microsoft Excel 2010, Microsoft, Seattle, WA) was used to collate and analyse the data collected from each participant. Means and standard deviations were calculated for the participants' anthropometrics, HR, VO_2 , SpO_2 and S_mO_2 and THC. For all parameters bar S_mO_2 , the data collected comprised of the last three minutes of each five-minute stage. For reasons of accuracy a ten second buffer either side was applied to the S_mO_2 data, causing the analysed time to be reduced to two minutes forty seconds.

3.4 Statistical Analysis

Statistical analysis was carried out on all analysed data using GraphPadPrism software (GraphPad Prism 6.00 for Mac; GraphPad Software Inc., La Jolla, CA, USA). Statistical significance being set to the value of below 0.05 ($p < 0.05$). Anthropometric and VO_{2max} data was tested for significant differences between the two groups using a paired t-test. Heart rate, VO_2 , SpO_2 and S_mO_2 and Total haemoglobin concentration were all tested using a repeated measures two-way ANOVA to highlight significance of the group, exercise and interaction effect. An independent t-test was conducted on SPR and EN for heights, mass, age and VO_{2peak} to identify a significant difference.

CHAPTER IV

RESULTS

4. Results

4.1 Mean HR and VO₂ Response to Exercise

Figure 1 (below) displays mean HR responses to normoxia and hypoxia in both EN and SPR. In both conditions a main effect of exercise intensity was observed ($p < 0.0001$), with increasing intensity eliciting higher HR. There was no main effect of training status or significant interaction (exercise*intensity; $p > 0.05$). This suggests that EN and SPR HR responds similarly to different intensities of exercise in both normoxia and hypoxia.

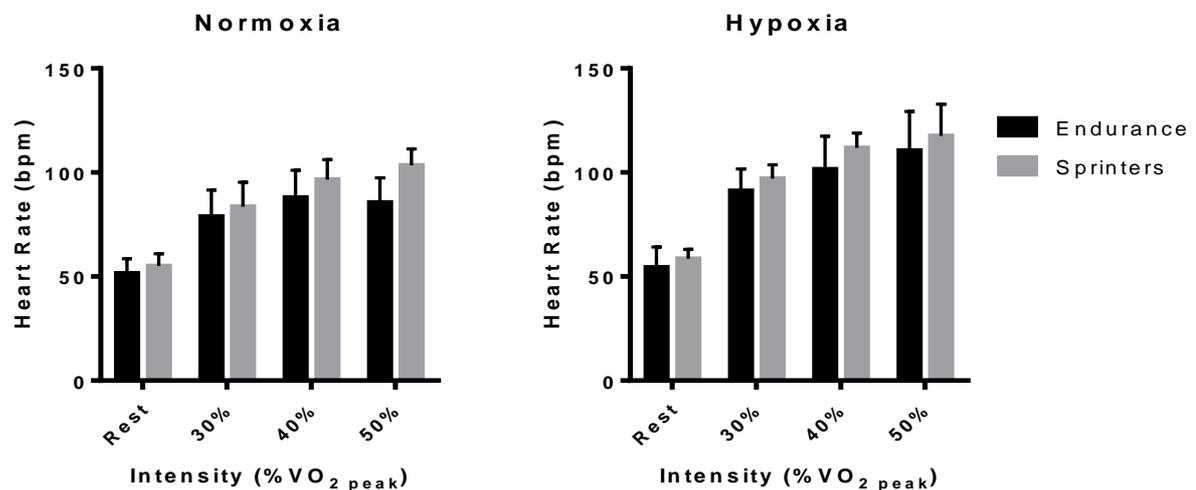


Figure 1. Mean HR response to exercise.

Figure 2 (below) presents EN and SPR Mean VO₂ responses to multiple exercise intensities in both normoxia and hypoxia. Exercise intensity being a main effect ($p < 0.0001$), causes Mean VO₂ to increase with each increase in intensity. No main effect of training status or interaction was observed (exercise*intensity; $p > 0.05$). This implies that the Mean VO₂ responses for both EN and SPR were similar in both normoxia and hypoxia.

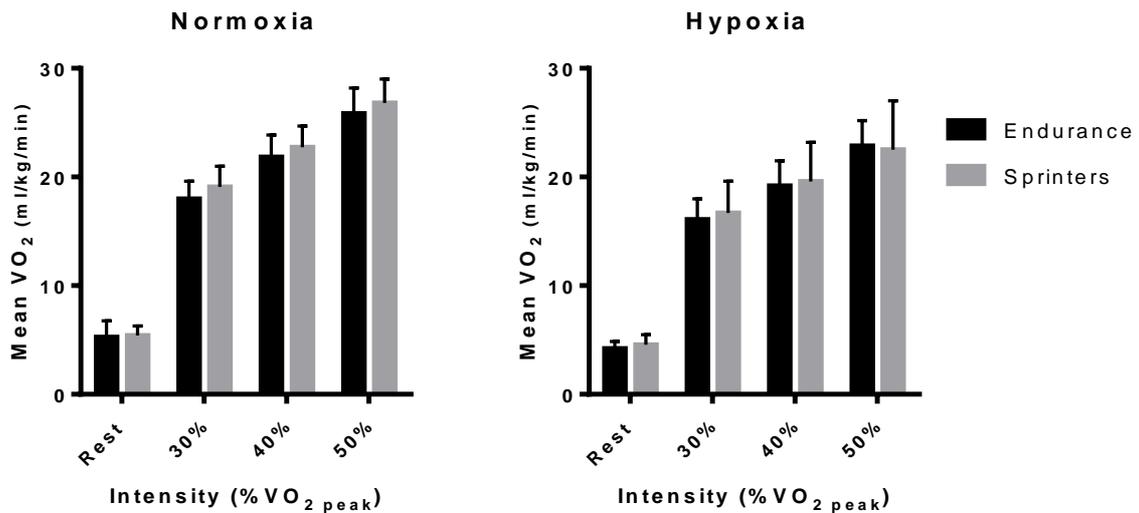


Figure 2. Mean VO₂ response to exercise.

4.2 Arterial Blood Oxygen Saturation Response to Exercise

The exercise response of S_aO₂ in normoxia and hypoxia can be observed in Figure 3 (below). For both conditions a main effect of exercise intensity is indicated (p<0.0001). There was also another main effect of training status in normoxic condition (p=0.0315), identifying a difference between the two groups, in this case implying that SPR desaturated to a greater degree than EN. There was no significant interaction effect (p>0.05).

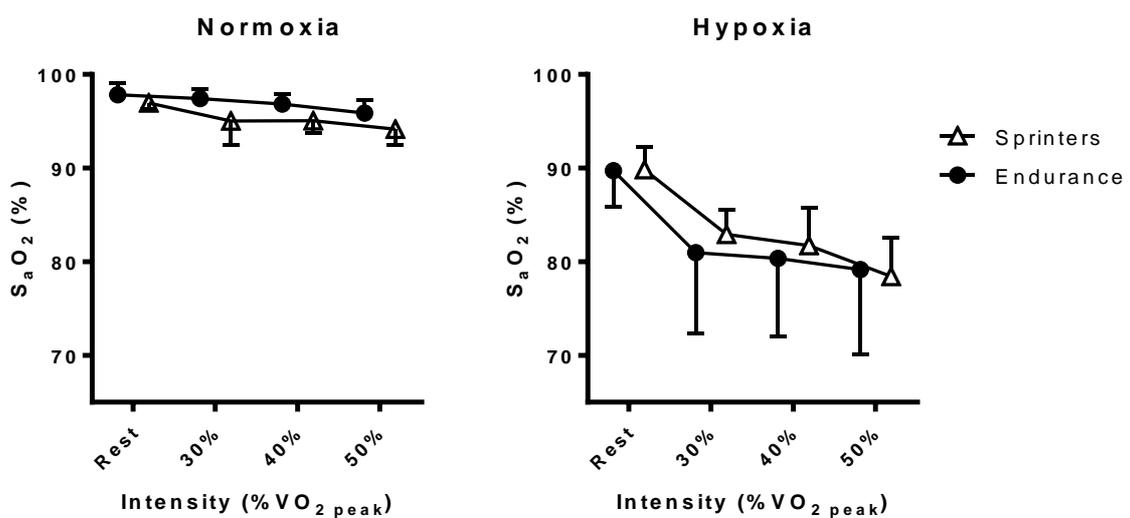


Figure 3. Arterial blood oxygen saturation response to exercise.

4.3 Skeletal Muscle Oxygenation Response to Exercise

Figure 4 (below) displays the effect of exercise intensity on THC for both training groups. In both hypoxia and normoxia exercise intensity was seen as a main effect ($p < 0.0001$). Training status had no significant effect on THC in both normoxia and hypoxia ($p > 0.05$). This infers that the training status of the volunteer has little effect on THC during exercise.

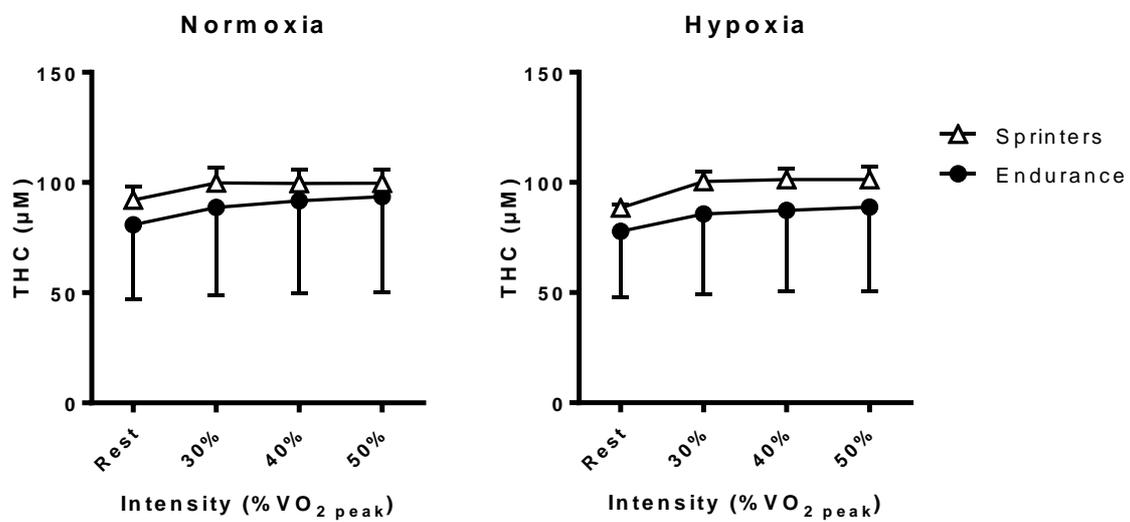


Figure 4. Total haemoglobin concentration response to exercise.

Figure 5 (below) presents the S_{mO_2} of both EN and SPR in normoxia and hypoxia. Inline with all previous results, a main effect of exercise intensity can be identified in both conditions ($p < 0.0001$). There was no main effect of training status in both normoxia and hypoxia ($p > 0.05$). Although there was a trend suggesting that in normoxia SPR desaturated to a greater extent ($p = 0.0688$). There was an interaction effect between the two factors ($p = 0.0244$).

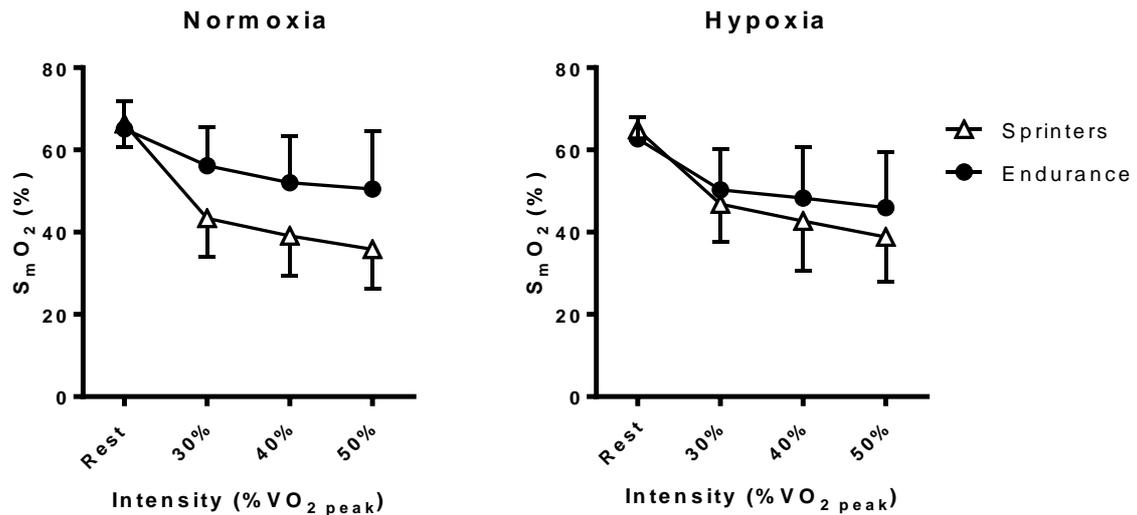


Figure 5. Skeletal muscle oxygen saturation response to exercise.

CHAPTER V
DISCUSSION

5. Discussion

The aim of this study was to investigate and identify the effect of normobaric hypoxia on muscle oxygenation, specifically the VL, in SPR and EN. The hypothesis primarily emanated from the findings of Van Theinen and Hespel (2015) who identified that EN are at a higher risk of developing hypoxemia than untrained individuals. This was attributed to a higher incidence of oxidative type I skeletal muscle fibres. With the theory that anaerobic interval training causes an increase in anaerobic type II fibres (Jansson *et al.*, 1990) and Saltin *et al.* (1977) finding sprinters had a higher incidence type II fibres, SPR could potentially perform in hypoxia similar to that in normoxia. The results of S_mO_2 portray that in normoxia, EN do not experience desaturation to the degree exhibited by SPR. In terms of skeletal muscle this is as expected due to sufficient available oxygen during the submaximal exercise and high oxidative fibres. Whereas in hypoxia both training groups respond comparably with no statistical difference ($p>0.05$).

5.1 Arterial Blood Oxygen Saturation

Normoxic conditions have minimal effect on S_aO_2 during submaximal exercise (Figure 3), as a result of satisfactory ventilatory rate adequate for oxygen consumption. As it is known that both SPR and EN experience higher rates of S_aO_2 desaturation when compared to untrained individuals during exercise (Oguri *et al.*, 2008; Van Theinen & Hespel, 2015), it is interesting to observe that in normoxia there was a significant training effect. SPR had consistently lower S_aO_2 over the four averaged intensities of exercise than EN. With all other variables being the same in the exercise protocol, it is evident that SPR have underlying physiological features causing a larger desaturation in normoxia. For both training groups, as expected, hypoxia presented an increased physiological stressor. There was a distinct effect of hypoxia on S_aO_2 even at rest. This is in agreement with the findings of Koytun *et al.* (2011), who's results suggested that hypoxia causes S_aO_2 to decline at rest. The effect of each intensity was very similar in both training groups. With no influence being found in the effect of training status on S_aO_2 in hypoxia, yet significance being found in normoxia. It gives foundation to the theory that there is a larger effect of hypoxia on EN. This mirrors the findings of Van Theinen and Hespel (2015). Understanding the physiological differences between EN and SPR has been paramount to this study, to help comprehend the reason why SPR athletes have

lower S_aO_2 during normoxic exercise. A major theory emanates from the skeletal muscle differences that have been observed between SPR and EN.

5.2 Total Haemoglobin Concentration

Increasing THC has been a target of many professional athletes for decades. Haemoglobins correspondence with the ability to transport O_2 to working muscles seems like an adaptation to improve performance (McArdle et al., 2015). This ability to improve performance, outlined by Otto *et al.* (2013), has even caused athletes to supplement with its precursor EPO to stimulate haemoglobin production. As presented in Figure 4 above, only the effect of intensity had a significant difference on THC during exercise, this previously being attributed to increased blood flow (Hiroyuki *et al.*, 2002) and intramuscular pressure (Kawaguchi *et al.*, 2006). One contradiction with previous publications is that there was no initial drop in THC before a gradual increase during exercise as found by (Kawaguchi *et al.*, 2005). This discrepancy could be a result of intensity differences, with higher intensities causing an increase in SBF and intramuscular pressure. With the THC of both SPR and EN adapting similarly from the onset of exercise in both normoxia and hypoxia, it mirrors the findings of Van Theinen and Hespel (2015) who also observed increases in THC during exercise in both hypoxia and normoxia. Furthering this, THC did not linearly change to increasing exercise demands, like muscle oxygen saturation as observed in figure 5. Although no statistical difference occurred in either condition or group, trends did occur. As graphically evident in Figure 4, the THC of EN athletes is consistently lower than SPR, this may be a statistical anomaly as the standard deviations for EN are much larger also. High inter-group variability of THC for EN could prove a limitation to the study as Mairbäurl (2013) established that different durations of exercise training, for example weeks compared to months, can cause varying THC in athletes.

Understanding that THC differs little between groups and conditions, it could identify that adaptations to THC occur due to chronic exposure to exercise or hypoxic stimuli. Supporting this, it has been observed that mountaineers returning from a sojourn at high altitude (8,848m) to moderate altitude (5,300m) have higher THC than others who did not sojourn (Martin *et al.*, 2009). Moreover, the EPO production of haemoglobin in the kidneys is amplified when S_aO_2 is low (Mairbäurl, 2013), further evidence to the benefit of chronic or intermittent hypoxia is provided.

5.3 Skeletal Muscle Oxygenation During Exercise

Using Figure 5 for graphical representation, it was identified that S_mO_2 in this study closely matched trends set out by previous research. A decrement was observed as intensity increased. In the normoxic condition the two groups, although not significant ($p>0.05$), physiologically responded differently to the exercise protocol. As displayed, SPR desaturated to a greater degree than EN, this follows the same pattern observed in S_aO_2 . Interpreting this result using previous findings, it can be assumed that both training groups in this study would have desaturated to a greater degree than untrained individuals (Van Theinen & Hespel, 2015; Oguri *et al.*, 2008). The ability of EN to maintain a higher S_mO_2 comes as no surprise, as EN are physiologically adapted to maintain exercise of extended periods of time. Hypoxic conditions provided more insight into the physiological adaptations of both SPR and EN. Unlike in normoxia, SPR and EN responded to hypoxia similarly. This potentially novel finding identifies that SPR trained athletes are less affected by hypoxia than EN. It could be inferred that SPR maintain a similar level of performance in both hypoxia and normoxia, whereas EN experience hypoxia as a greater physiological stressor. These findings support postulations that skeletal muscle composition is a main factor in performance in hypoxia.

Naturally the human body will attempt to physiologically adapt to higher altitude or hypoxia, an example being that untrained high altitude natives have a higher proportions of type II muscle fibres (Hoppeler & Vogt, 2001). It supports the findings that a lower incidence of type I muscle fibres are beneficial at altitude. With the adaptability of skeletal muscle fibres being demonstrated by multiple research studies (Jansson *et al.*, 1990; Kubukeli *et al.*, 2002 MacIntosh *et al.*, 2006), it allows for inference that the participants in this study held adaptations relative to their sporting discipline. For EN trained athletes, the high oxidative capacity of type I muscle fibres could produce the systematic reduction of O_2 in both arterial blood and skeletal muscle blood, due to higher O_2 utilisation. SPR athletes having high prevalence of type II fibres has evidently allowed them to maintain performance when introduced to hypoxic stimuli. Through a lower oxygen uptake inside skeletal muscle, the SPR athletes have been able suspend the effect of hypoxia on S_mO_2 depletion.

5.4 Reliability of Methodology

Using relative intensities to each participants VO_{2peak} ensured that each participant was experiencing the same level of physical exertion. Using this method, between the two exercise bouts the only changing factor was the hypoxic or normoxic condition, allowing inference to that all changes in results occurred due to either the hypoxic or normoxic stimuli or the individuals training status. Figure 2 presents the findings that there was no difference in VO_2 attainment through the exercise, therefore relative intensity, in both SPR and EN. Inside each training group there was evident homogeneity as seen in Table 1. Unpaired t-tests were carried out to identify any statistical difference between SPR and EN. Height, Mass and VO_{2peak} were all identified as not significantly different ($p>0.05$). Age was highlighted as being statistically different ($p=0.0156$), although it has been shown that $\dot{V}O_2$ increase with age (Bianba *et al.*, 2006), in the current study there is no significant difference in $\dot{V}O_2$ (Figure 4).

NIRS reliability has been extensively researched, with multiple papers advocating NIRS as a suitable non-invasive method of monitoring and collecting muscle oxygen data (Boushel *et al.*, 2001). It has been highlighted that high adipose tissue thickness can affect NIRS measurements negatively (Ferrari *et al.*, 2004). Although skin-fold measurements were not taken in this experiment, all participants were young healthy and highly active males inferring that they had low sub-cutaneous adipose tissue. The possibility of capillary vasoconstriction affecting NIRS results (Martin *et al.*, 2009) was mitigated by carrying out the procedure in a neutral temperature and completing a warm up prior to data collection.

Research has been conducted on the effect of pedalling cadence on S_mO_2 . When contracting it has been hypothesised that intramuscular pressure in skeletal muscle restricts blood flow. After observing Lance Armstrong maintain unusually high cadence on an ascent, it was proposed that a higher cadence reduced the time blood flow to the quadriceps was restricted (Lucia, 2002). Considering this, a possible limitation of this study was the pedalling cadence of the protocol (60-65 RPM) being potentially too low and hypothetically accelerating hypoxemia in the muscle. Postural influence on exercise performance must be taken into account for this research. With the protocol being conducted on a supine cycle ergometer,

performance decrements are expected. It has been identified that supine cycling causes greater muscle-deoxygenation, lower O₂ uptake kinetics however time to muscle fatigue was unaffected (Denis and Perrey, 2006).

5.5 Practical Implications

In addition to the clinical findings of Van Theinen and Hespel (2015), it could be proposed that adjustments in training could alleviate the effects of AMS. When preparing for ascent to altitude, physically training for type I to type II muscle fibres could yield a better buffering capacity to hypoxia. With evidence for this being in Figure 5.

With the major finding emanating from this research being that for both EN and SPR, exercise in hypoxia produces similar acute physiological alterations. The inference that the negative effect of hypoxia on SPR is lesser than that on EN, allows for sporting adaptations to be recommended. Preparing for performance at altitude, in events such as the Mexico City Olympic Games, the results identify the greater requirement for EN to acclimatise. Although acclimation to the target altitude would be beneficial to SPR in terms of recovery ability, hypoxic performance over short term may not be affected to a great degree for SPR.

Implicating hypoxic training to endurance athletes training regime has long been accepted to improve performance (Ponsot *et al.*, 2006; Bonetti & Hopkins, 2009; Millet *et al.*, 2010). The results in this current study identified that SPR S_mO₂ in hypoxia did not follow trends set by EN in both normoxia and hypoxia, inferring that SPR had a higher capacity to buffer the effect of hypoxia. Understanding this SPR training in hypoxia may not be as beneficial as EN, contradicting evidence by Ponsot *et al.* (2006), it was proposed by Hamilin *et al.* (2009), anaerobic performance only improved slightly, potentially down to the lack of extra stimuli. The fiscal implications to sporting athletes may be that finance could be better allocated for improving performance for SPR than hypoxic training.

As VO₂ is still a larger factor of performance, it is evident in our study that even with different training modalities, similar VO_{2max} can be achieved. Both SPR and EN had VO_{2peak} values of 44.16ml/min/kg and 43.29ml/min/kg respectively. Further postural effects, than observed by Denis and Perrey (2006), have to be taken into account for this exercise protocol as it was conducted on a 45° supine cycle.

5.6 Future Research

Verifying the exact skeletal muscle fibre proportions inside SPR and EN would serve to greatly support the inferences made in this publication. Utilising skeletal muscle biopsies, although invasive, could prove invaluable in its insight into the exact cause of differential oxygen uptake inside skeletal muscle in athletes.

Utilising a larger population would allow for higher homogeneity and applicability to each athlete training modality. Furthermore, using alternate sporting groups, such as; rowers and team sport athletes, could assist in understanding the underlying adaptations causing the variations in S_mO_2 saturation. The physiology of skeletal muscle in elite rowers has been identified as being primarily type I fibres (Hagerman, 1984), but with high anaerobic demand at the start of a race it could signify potentially beneficial adaptations to performance in hypoxia.

CHAPTER VI
CONCLUSION

6. Conclusion

To conclude, the effect of normobaric hypoxia on S_mO_2 did not significantly differ between SPR and EN. Corroborating with what is known about the aerobic ability of EN it was evident that in normoxia they performed better as trends portrayed (Figure 5). With both groups portraying similar ability in hypoxia, it can be assumed that in hypoxia, SPR mitigated the reduction of O_2 due to their high incidence of type II muscle fibres, where EN continued to experience a high oxygen utilisation in the muscle. Conforming with evidence of ENs high oxygen extraction rate due to type I muscle fibres (Van Theinen & Hespel, 2015). Findings emanating from this research can assist athletes and coaches alike in understanding the inter-athlete variability, due to skeletal muscle, and how it affects performance. With further research, as detailed above, findings could help inform on the effect of muscle composition on sports performance in hypoxia or at altitude.

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APPENDIX A

Appendix A – Ethical Approval

This research study was ethically approved by the Cardiff Metropolitan School of Sport.

Ethics Code: 15/5/374U