Effects of Intermittent Hypoxic Exposure on Physical Performance in Trained Basketball Players

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A thesis submitted to AUT University in partial fulfilment of the requirements for the degree of Master of Health Science (MHSc)

October 2009

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Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the qualification of any degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

Signed: _____

Date: _____/_____/_____

Acknowledgements

To my supervisor, Dr. Andrew E. Kilding, thank you for giving me the opportunity to work on such a cool project. And for having faith in my ability to get the job done (or at least you never showed it when you were losing faith), it was a real confidence booster. Your feedback was invaluable. Hopefully there will be a few more even cooler projects in the future. Cheers Andy.

To Andrew Chapman, from Airo NZ Ltd, I am very grateful for the opportunity to be able to play with some very cool technology. The study simply would not have been possible without Airo funding the necessary consumable costs and providing the necessary equipment. In addition, Airo NZ Ltd. was very generous in their support and contribution towards a Tertiary Education Commission Enterprise scholarship, for which I am very grateful. Thanks Andrew.

To Filomena Davies and Sara Metcalf, your support and guidance in getting my TEC scholarship application into some sort of respectable state was invaluable. AUT students are very lucky to have people such as yourselves on their side.

The Tertiary Education Commission, for the awarding of an Enterprise scholarship.

Darrell Bonetti, thanks for all your advice, tips, hints, and thoughts. Having someone in the office across the hall who knew the ins and outs of IHE was brilliant. Your input was always valued. Cheers.

To my flatmates over that past few years – Wendy, Brent, Jamie, Jan, and Pip. A normal man could have gone mad with the amount of time I've taken to finish this, so I thank you all for contributing towards my sanity with extracurricular activities. I owe you all a beer. Cheers.

The athletes, you guys were awesome. I realise being part of this project was a big commitment, so I am truly thankful for your dedication to it. Without you all, the study, quote obviously, would not have been possible.

Finally, to Mum and Dad, your support of my education over the many many years has been more than anyone could rightly expect. Whilst my educational journey will never be complete, I hope this goes some way to repaying some of faith you have shown in me over the years.

Ethics approval for this project was granted by the Auckland University of Technology Ethics Committee on November 22nd, 2007, Ethics Application Number 07/176.

Intellectual Property Rights

Abstract

Strong evidence exists to support the use of a continuous (>8hr/day) hypoxic stimulus (either geographical altitude or simulated hypoxia) for enhancing the physical performance of endurance athletes. However, evidence supporting the use of acutely intermittent hypoxia (<1hr/day) for enhancing performance is less clear. The purpose of this study was to determine the effect of acutely intermittent hypoxic exposure on physiological and physical performance measures in team sport athletes.

Using a single-blind controlled design, 14 trained basketball players (HYP = 7, CON = 7) were subjected to 15 days of intermittent hypoxia or normoxia. Each exposure was 37 minutes in duration (four cycles of 7min on, 3min off) and achieved using a nitrogen dilution device (Airo Ltd, Auckland, NZ). Prescribed peripheral oxygen saturation levels (SpO_2) were maintained using an automatic biofeedback system and were progressively decreased from 86-89% on Day 1 to 75-78% on Day 15. A range of physiological measures and performance tests were conducted seven and two days before, and ten days after the intervention. The tests were: an incremental treadmill test to establish peak oxygen consumption ($\dot{V}O_{2peak}$) and running economy (RE), Yo-Yo Intermittent Recovery Test (YYIRT), and the Repeated High-Intensity Endurance Test (RHIET). Whole-blood samples were taken to assess a range of haematological measures.

At 10 days post-intervention the HYP group, relative to the CON group, exhibited the following percent changes (±90% confidence limits, CL), and

effect sizes (ES; ±90% CL); YYIRT running $speed_{peak}$ (4.8; ± 1.6%, ES: 1.0 ± 0.4; benefit almost certain), RHIET total sprint time (-3.5; ± 1.6%; ES: -0.4 ± 0.2; benefit very likely), RHIET slowest sprint time (-5.0; ± 2.4%; ES: -0.5 ± 0.2; benefit very likely), soluble transferrin receptor (9.2; ± 10.1%; ES: 0.3 ± 0.3; benefit possible) running economy ($11km.hr^{-1}$) (-9.0; ± 9.7%; ES: -0.7 ± 0.7; benefit likely, probable), and running economy ($13km.hr^{-1}$) (-8.2; ± 6.9%; ES: -0.7 ± 0.5; benefit likely, probable). Changes to running economy ($9km.hr^{-1}$), $\dot{V}O_{2peak}$, maximum heart rate and lactate and all other blood measures were unclear.

In conclusion, acutely intermittent hypoxia resulted in worthwhile changes in physical performance of trained basketball players in tests relevant to competition. However, the lack of clear change in physiological and haematological measures makes it difficult to determine the underlying mechanism for such enhancement.

Chapter One: Introduction

Humans have habituated areas of high-altitude, which induce noticeable physiological responses, for thousands of years(Niermeyer, Zamudio, & Moore, 2001). Habitants of such areas have made, unknowingly, physiological adaptations (Beall, 2000; Beall et al., 1998; Beall & Reichsman, 1984) in response to their extreme environments to enhance their survivability. It is these adaptations that altitude training relies upon to alter individuals' physiological characteristics in a positive manner conducive to enhancing physical performance.

There is a substantial amount of research dedicated to human physiological responses and adaptations to acute (Easton, Slykerman, & Anthonisen, 1986; T. Klausen et al., 1996; Peltonen, Tikkanen, & Rusko, 2001; Powell, Milsom, & Mitchell, 1998; Vogel & Harris, 1967; Wagner et al., 1986) and chronic hypoxia (Alexander, Hartley, Modelski, & Grover, 1967; Huang et al., 1984; Katayama et al., 1999; Townsend et al., 2002). In contrast, however, there is substantially less research dedicated to the responses and adaptations to intermittent hypoxia. From the existing literature, it is clear that there are some fundamental differences, in terms of the human physiological response, to intermittent hypoxia in comparison to chronic hypoxia (Dwindell, Janssen, & Bisgard, 1997; Powell & Garcia, 2000; Powell et al., 1998). In addition, the limited existing literature tends to suggest some of the positive effects (in terms of haematological enhancement) of chronic hypoxic exposure are also achievable through short duration hypoxic exposure (Eckardt et al., 1989; Knaupp, Khilnani, Sherwood, Scharf, & Steinberg, 1992).

Deliberately exposing humans to hypoxia for the purposes of performance enhancement at sea level is a relatively recent phenomena. However, there is already a substantial body of research dedicated to examining the efficacy of hypoxic exposure on physical performance. Initial research examined the effects of the "Live High, Train High" (LHTH) technique, most of which was focused on endurance-based athletes (Gore et al., 1998; Ingjer & Myhre, 1992; Svedenhag & Saltinj, 1991). Subsequent modifications to the LHTH model have also been investigated. These have included, but not limited to; "Live High, Train Low" (LHTL), and aritificial "Live High, Train Low" (LHTL artificial), primarily using endurance-based athletes (Clark et al., 2004; Dehnert et al., 2002; Gore & Aughey, 2001; Hahn et al., 2001a; Hinckson et al., 2005; Wehrlin, Zuest, Hallen, & Marti, 2006). The efficacies of these techniques as a tool for enhancing performance have been reviewed in meta-analytic form recently (Bonetti & Hopkins, 2009). This analysis showed that there are likely performance benefits to be gained from LHTH, LHTL, and artificial LHTL approaches, but the extent of gains may be dependent on the training level of athlete (subelite or elite).

The disadvantages of continuous forms of hypoxic exposure (inconvenience, cost, time constraints) have driven the development of alternative methods to gain similar adaptations. This has formed the basis for short duration intermittent hypoxic exposure (IHE) devices and protocols. The development of a device and protocol that allows an athlete to enhance performance with as little inconvenience as possible represents a substantial move forward in altitude training. However, research suggests further development is required

before such devices/protocols are more widespread, despite their convenience (Bonetti & Hopkins, 2009). In their meta-analysis, Bonetti & Hopkins (2009) suggested that short duration IHE would be beneficial for subeltite athletes but not elite athletes, and highlight the need to further develop appropriate protocols (for all forms of hypoxic exposure) to enhance its efficacy.

The research to date using short duration IHE has produced contrasting findings with regards to its efficacy in improving sports performance (Babcock & Kirby, 2008; Bonetti, Hopkins, & Kilding, 2006; Bonetti, Hopkins, Lowe, & Kilding, 2009; Hamlin & Hellemans, 2007; Hinckson, Hamlin, Wood, & Hopkins, 2007; Hinckson, Hopkins, Downey, & Smith, 2006; Julian et al., 2004; Tadibi, Dehnert, Menold, & Bartsch, 2007; Wood, Dowson, & Hopkins, 2006). Most short duration IHE research has focussed on endurance related performance (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007) which is understandable given the primary mechanism for adaptation has largely been understood to be oxygen delivery related. In contrast, the effect of short duration IHE for team sport athletes remains relatively under-investigated, with a only a few studies conducted to date (Hinckson et al., 2007; Wood et al., 2006). Further work is warranted with team sports athletes to substantiate these findings.

In addition to the variation in athlete type and performance measure(s) used in short duration IHE studies to date, the method and technology used to deliver IHE is also of importance. Because the total quantity of hypoxia during IHE is inevitably smaller than continuous forms of hypoxic exposure(s), the

prescription of the intensity (i.e. FIO_2 or SpO_2%) in short duration IHE must be somewhat individualised to ensure a consistent stimulus is being received by each individual. Of the work using FIO_2 as the prescription tool (Hamlin & Hellemans, 2007; Julian et al., 2004; Tadibi et al., 2007), only one (Hamlin & Hellemans, 2007) study has found any enhancement of physical performance. By using SpO_2% as the prescription tool, the individuals physiological response provides feedback to the simulation device to adjust the intensity as and when required. This is known as a biofeedback mechanism. Because of a reasonably large variation in individual hypoxic ventilatory response (Hirshman, McCullough, & Weil, 1975) the biofeedback mechanism is used to maintain a relatively constant individual S_aO_2.

The aim of this study was to examine the efficacy of short duration IHE, delivered using a biofeedback controlled system, on the physical performance, physiological, and haematological characteristics of trained basketball players. The results of this thesis will serve to assist with recommendations for basketball players, and athletes from other that high-intensity intermittent team sports alike, in their possible use of short duration IHE as a training tool for enhancing performance.

Chapter Two: Review of the Literature

This review is presented as three main sections: 1) the physiological response to hypoxia and intermittent hypoxia; 2) an investigation into the technology for delivering short duration IHE, methods and protocols used during short duration IHE; and 3) a review of the intervention studies investigating the effect(s) of short duration IHE on physical and sport performance.

The methods used for identifying and selecting appropriate published research for section three of the review are detailed below. Searches of Pubmed and Google Scholar were performed for studies published in English up until, and including, May 2009. Keywords or phrases used for searching were: intermittent hypoxic exposure, intermittent hypoxic training. Subsequent reference lists, and those of review articles were examined for further applicable studies. Studies were only selected if using sea-level (or close to sea-level, <1000m) athletes, measuring sea-level performance or close to sea-level, <1000m). The review will also only examine studies using intermittent (alternating between hypoxic and normoxic inspired gas) hypoxic exposure up to a maximum of 90 minutes per day where physical performance measures are described.

Physiological responses and adaptations to hypoxic exposure

History

The concept of intentionally imposing hypobaric or hypoxic challenge upon an athlete for short or long durations, with or without the additional challenge of

exercise, in an attempt to induce physiological adaptation potentially leading to performance enhancement, is a relatively new concept in exercise physiology. However, the physiological mechanisms associated with adaptation to such stimuli are far from unique as humans have habituated geographical altitude for thousands of years. Linguistic and genetic research suggests that the Tibetan Plateau has been habitated for upward of 50,000 years, and the Andean antiplano for at least 5,000 years (Niermeyer et al., 2001, pp. 45-46). Undoubtedly, these early prehistoric migrants will have been the earliest to experience the acute altitude symptoms we now understand to be a consequence of hypoxia. Over time these migrants would also have, inadvertently, been the first humans to make chronic physiological adaptations to deal with this hypoxic stimulus. These physiological adaptations are the same being researched and taken advantage of by athletes today.

It was not until the 1968 Olympic Games hosted in Mexico City that the effects, both beneficial and harmful, of geographical altitude became a topic of interest amongst exercise physiologists and the wider sporting population. The track and field results in Mexico City, which is situated at 2,240m above sea level, provided very strong evidence for the effects of a hypoxic environment affecting physical performance. As a general rule, the results of the sprint and throwing events provided very good performances (in 1968 terms) including a number of world records (WR), whilst the middle and long-distance event results were relatively poor in comparison (the only exception being a WR equalling time in the Mens 800m). The prevailing rationale for these unique results is that the hypobaric environment provided less air resistance for the sprint and throwing events, which comprised only a very small aerobic component. Whereas the

middle and long distance results were impaired, as a result of the athletes compromised oxygen delivery. It was also noted that those middle and long-distance athletes that were native to areas of geographical altitude (most notably Kenyan and Ethiopian athletes) appeared to produce better results in comparison to those from sea level regions. Additionally, many sea level native athletes returned to compete at sea level destinations after the Olympic Games to produce surprisingly good performances. Whilst it is not possible to directly relate these phenomena entirely to the effects of altitude exposure, it did provide the foundation for a surge of interest in the physiological effects of altitude exposure.

Physiological response(s) to continuous hypoxic exposure

Regardless of the mechanism of the hypoxic exposure, the principle of adaptation to the stimulus remains essentially the same. Time dependent adaptations to continuous hypoxia, can be loosely grouped into either short-term (occur within the first 24 hours of hypoxic exposure) or long term (occur at least 24 hours after initial hypoxic exposure) changes. For the purposes of this specific review, I will not consider or discuss at length, the adaptations that occur as a result of altitude residency, where chronic hypoxic exposure exists beyond four weeks.

The most acute and influential physiological response to hypoxic exposure, which in effect initiates a cascade of physiological events, is the reduction in partial pressure of oxygen at the alveolar level (P_AO_2), which also leads to a reduction in arterial oxyhaemoglobin saturation (S_aO_2) (Vogel & Harris, 1967;

Wagner et al., 1986). This has been shown to occur within 1-2 minutes of hypoxic exposure, and is directly related to the intensity of the hypoxic exposure. There are a number of mechanisms that begin to take effect at this point to compensate for this reduction in oxygen availability. Probably the most recognisable response is the increase ventilation (V_E), which has been shown to occur within 1-2 minutes of hypoxic exposure (Easton et al., 1986). The increase in V_E is mediated by an increase in breathing frequency (B_f) and tidal volume (V_T) (Powell et al., 1998). This response is initiated as a result of peripheral chemoreceptors' detection of the hypoxic environment via lower than normal partial pressure of oxygen in arterial blood (P_aO_2) (Dempsey & Forster, 1982). The increase in $V_{E,}$ and resultant inspired oxygen quantity, ensures sufficient oxygen delivery to the necessary tissues and organs, at rest at least. After the initial rapid rise in $V_{E,}$ there is a reduction to approximately 20% (Easton et al., 1986) above normoxic conditions within 30 minutes of initial hypoxic exposure, a phenomenon which has been displayed in other mammals (Gershan et al., 1994). It has been shown that longer-term ventilatory acclimatisation is a result of an enhanced sensitivity of the peripheral chemoreceptors (Katayama et al., 1999; Townsend et al., 2002). From an athletic performance perspective, an enhanced hypoxic ventilatory response (HVR) is beneficial (i.e. ventilation responds quickly to a change in demands for oxygen, as occurs during exercise). The body responds to the hypoxic stimulus (at sea level, this may be exercise-induced hypoxia) more quickly by responding with an increased oxygen delivery to the working muscles. However, this enhancement of HVR is lost very quickly upon withdrawal of the hypoxic exposure (Sato, Severinghaus, & Bickler, 1994). In addition, it is worth noting that athletic populations have exhibited a different, less responsive, HVR than

non-athletic populations or mountaineers (Byrne-Quinn, Weil, Sodal, Filley, & Grover, 1971).

The cardiovascular system contributes to the compensatory responses by increasing heart rate (HR) and, resultant cardiac output (Q_c). Heart rate has been shown to increase above normoxic levels during rest in a hypoxic environment (Peltonen et al., 2001) and continue to increase for up to 30 hours (Vogel & Harris, 1967). Stroke Volume (SV), the other component of Q_c, appears to drop slightly during early hypoxic exposure (K. Klausen, 1966), an effect that is exacerbated by increasing hypoxic intensity (Nesterov, 2006) and after a period of time beyond 24 hours (Alexander et al., 1967). This can be partly attributed to the reduction in plasma volume as a result of increased urinary and respiratory water losses.

Whilst the aforementioned respiratory responses would appear to be the most responsive mechanisms to be able to maintain sufficient oxygen delivery in an acute sense, the haematological response mechanisms have also been shown to commence in very quick fashion after the onset of hypoxic exposure. Eckardt and colleagues (1989) and Knaupp and colleagues (1992) have shown increases in erythropoietin production within two hours of hypoxic exposure. Although this provides evidence that the erythropoietic cascade is initiated very quickly, there is no measurable change in red cell mass for 4-7 days (T. Klausen, Mohr, Ghisler, & Nielsen, 1991), due to the time required for RBC's to mature. Further discussion of the implication of this research will be discussed in later sections of the review. This evidence provides us with the time-course for hypoxic responses but does not indicate the threshold for which hypoxia is

initiated. In the classical study examining this phenomena, it has been shown that athletic populations have a enhanced HVR in comparison to the general population (Weil, 2003). Weil and colleagues (1968) investigated the necessary P_IO_2 necessary to initiate hypoxic adaptation. The group reported a threshold P_IO_2 of 67 mmHg as the minimum requirement for red cell mass to be altered, which represents a geographical altitude of approximately 2200 metres.

Physiological response(s) to intermittent hypoxic exposure

For the purposes of this review, intermittent hypoxia is differentiated from chronic hypoxia and acute hypoxia. For this purpose, intermittent hypoxia is defined as short duration hypoxia alternated with periods of normoxia or less-intense hypoxia at rest. This section will seek to identify the physiological response(s), adaptation(s) and acclimatisation mechanism(s) as a result of intermittent hypoxia, and discuss key differences between chronic and actute hypoxia.

The contradiction that is intermittent hypoxia has been well-described by Serebrovskaya and colleagues (2003) as:

"…a multi-headed Medusa in physiology and medicine." (p.1)

This excerpt is made in reference to the acknowledgment that intermittent hypoxia has some very contrasting physiological consequences. On one hand, it is considered a very debilitating health consequence of many medical conditions including, but not limited to, cerebrovascular (Schoene, 1999),

cardiopulmonary disease (Neubauer, 2001; Serebrovskaya, 2002), and sleep apnoea (Sieck, 2001). On the other hand, the adaptations and physiological responses as a result of chronic and intermittent hypoxia have been identified as beneficial for enhancing physical performance. The physical benefits of intermittent hypoxia have come from two clearly different avenues. Firstly, it has been used as a means of acclimatising to high altitude, particularly exercising at high altitude (Serebrovskaya, 2002). High-altitude mountaineers often use intermittent sojourns, and the associated intermittent hypoxia, as a means for acclimating to the hypoxia before making summit attempts to higher and more hypoxic altitudes (Powell & Garcia, 2000). Similarly, efforts have been made to utilise artificial intermittent hypoxia for enhancing performance of sporting events at altitude, albeit with limited success thus far (Hamlin, Hinckson, Wood, & Hopkins, 2008). Secondly, intermittent hypoxia is used as a means for enhancing physical performance at sea-level (Babcock & Kirby, 2008; Bonetti & Hopkins, 2009; Bonetti et al., 2006; Hamlin & Hellemans, 2007; Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007; Wood et al., 2006) with potentially promising consequences (Babcock & Kirby, 2008; Bonetti & Hopkins, 2009; Bonetti et al., 2006; Hamlin & Hellemans, 2007; Wood et al., 2006). This will be discussed in more depth in a later section.

In comparison to the extensive knowledge base that exists regarding the effects of acute and chronic continuous hypoxia, substantially less is known and understood about intermittent hypoxia. If we assume that there are in fact different responses to intermittent hypoxia than acute and/or chronic continuous hypoxia, we are therefore assuming that the periods of normoxia (or less intense hypoxia) where an element of homeostasis is resumed has some form

of re-setting benefit on the applicable system. Evidence for the difference between intermittent and other forms of hypoxia has been mentioned by previous authors (Powell & Garcia, 2000). The authors put forth the argument that the effects of intermittent hypoxia differ from acute hypoxia because of hysteresis of the physiologically responding systems. Hysteresis dictates that the responding system has some form of memory-like effect. The example Powell & Garcia (2000) use to explain this phenomena is the immediate response to sensing hypoxia may be switched "ON" substantially quicker than it takes it be switched "OFF" after the stimulus has been removed. The time-lag between sensing the hypoxic stimulus switching "OFF" and physiological response mechanisms may have some influence on the physiological adaptations to intermittent hypoxia.

Ventilatory response to intermittent hypoxia

The reflexive ventilatory responses to acute hypoxia are well understood and have been described in detail for a number of years (Smith et al., 1986). The primary means of sensing hypoxia is via the carotid and aortic bodies, which respond to changes in P_aO_2 (Lahiri, Rozanov, & Cherniack, 2004). The respective chemoreceptors respond to the stimulus by sending afferent impulses to the respiratory control centre within the medulla, which stimulates the increase in ventilation (Lahiri et al., 2004). This is followed by secondary physiological states, including hypocapnia and respiratory alkalosis, which in themselves lead to other physiological responses. Marshall (1994) has argued that the chemoreceptor activation also activates the cardiovascular responses, possibly as a result of hypocapnia and respiratory alkalosis. This results in

multiple components of the sympathetic nervous systems responding to a single stimulus, which are known to have different response time-frames. This lends evidence to the fact that it is likely that there are differences between chronic and intermittent hypoxic adaptations. Powell and colleagues (1998) have suggested two mechanisms in which the ventilatory response to intermittent hypoxia may differ to chronic hypoxic exposure; progressive augmentation and long-term facilitations. Progressive augmentation, Powell argues (1998), is the increase in magnitude of the hypoxic ventilatory response as response to successive episodes of hypoxia. This is shown in Figure 1. The phenomena has been witnessed in animal models, in particular cats (Fregosi & Mitchell, 1994) and goats (Turner & Mitchell, 1997), however doubt has arisen as to whether progressive augmentation is real when stimulated by higher-intensity hypoxia, which contributes to the difficulty in differentiating progressive augmentation from long-term facilitation. Long-term facilitation, also shown in Figure 1, is the progressive increase in ventilatory response which increases during the normoxic periods of IHE. Long-term facilitation has been reported to be unique to intermittent hypoxia (Dwindell et al., 1997). However, whilst this phenomena has been reported in animal models (Bach & Mitchell, 1996; Fregosi & Mitchell, 1994), it has not been well demonstrated or reported in awake human models (McEvoy, Popovic, Saunders, & White, 1996).

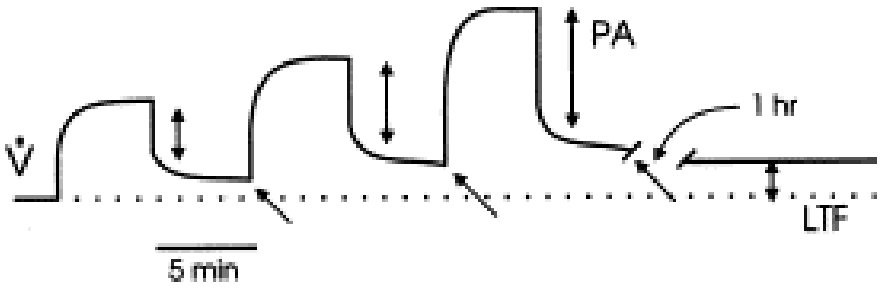

Figure 1. Schematic of the ventilatory responses during and after episodic hypoxic exposures, including progressive augmentation and long term facilitation.
PA = Progressive augmentation; LTF = Long-term facilitation (Powell et al., 1998).

It certainly appears there are ventilatory responses to intermittent hypoxia which are distinct from acute and chronic exposure, however some or all of these responses are not fully understood. It is possible that the differences in the responses of the different modes of hypoxic exposure are a result of the protocols being used. Therefore acknowledging the hypoxic intensity being experienced by the subjects is mandatory, as the majority of short duration IHE research has done to some extent at least. For the majority of simulated hypoxia research it is unpractical to measure S_aO_2, so monitoring of SpO_2% is essential for gauging this.

Haematological response to intermittent hypoxia

The underlying premise, or at least the primary premise, of hypoxic exposure for performance enhancement is via haematological mechanisms. During hypoxic exposure, cells throughout the body detect hypoxia, some of which are more sensitive to detection than others. This includes the hypoxic stimulus being detected by the kidney (Maxwell, Lappin, Johnston, Bridges, & McGeown, 1990; Richalet et al., 1994). It responds by stimulating the release or erythropoietin, which in time increases the development of red blood cell mass, thus enhancing

oxygen carrying capacity. The increase in serum EPO precedes the process of erythropoiesis within the red bone marrow, which produces more red blood cell mass. The time-course for these events lies at the heart of the differences between acute, chronic, and intermittent hypoxic exposure. Eckardt and colleagues (1989) were the first to describe the time-course of EPO production. The authors reported significantly elevated EPO levels after 114 and 84 minutes of hypobaric hypoxic exposure (simulating ~3,000m and 4,000m respectively). This was supported by Knaupp and colleagues (1992), whom showed changes (although not significant) in EPO production after only five minutes of hypoxic exposure (FIO_2 = 0.105) when measured after 30 minutes. However, significant changes were reported after 120 minutes of exposure (FIO_2 = 0.105) when measured at 240 minutes. It is worth noting that a large individual variation in erythropoietic response has been found. Jedlickova and colleagues (2003) examined the serum EPO after 24 hours of simulated altitude exposure in 48 athletes and found a -41 to 433% change from baseline. Similar large variations (serum EPO change of 10-185%) has been described in elite junior swimmers after four hours hypoxic exposure (Friedmann et al., 2005). The most likely reason for this variation is diurnal variation, of which Eckardt and Knaupp did not control for. However, since then, Klausen and colleagues (1996) measured EPO production throughout the day found variation of ~15%. Despite the relatively large diurnal variation during normoxia, these studies (Eckardt et al., 1989; T. Klausen et al., 1996; Knaupp et al., 1992) provide evidence that if the haematological changes seen as a result of such short durations of hypoxic exposure, short-duration hypoxic exposures may be a suitable substitute for longer duration hypoxic exposure methods (either natural or simulated) for performance enhancement, which traditionally require

significant time commitment in a hypoxic environment. However, it must be noted that because EPO has a relatively short half-life, a critical dose of hypoxia may be required to provide an adequate erythropoietic cascade.

Non-haematological mediation of performance enhancement

It is likely that there are additional non-haematological mechanisms at play when adapting to the effects of hypoxia and that these serve to mediate performance enhancement. These non-haematological mechanisms for performance enhancement have recently been reviewed extensively by Gore and colleagues (2007). One of the primary sources of evidence for the non-haematological mechanisms for adaptation comes from the variation in haematological and ventilatory adaptation between native highlanders in the Andes and Tibetan region (Beall, 2000; Beall et al., 1998; Beall & Reichsman, 1984). Two of the plausible non-haematological mechanisms for enhancing physical performance are cellular and molecular changes, and changes to movement economy. The cellular and molecular adaptations will be discussed in the next sub-section, whilst the changes to movement economy as a result of hypoxic exposure will be discussed in detail in a later section. The other possible non-haematological mechanism for performance enhancement is via muscle pH regulation and buffering capacity. A small number of studies have shown an increase in MCT1 and MCT4 monocarboxylate transporters after hypoxic exposure when combined with intense exercise. The MCT1 and MCT4 monocarboxylate transporters are essentially responsible for the transport of lactate across membranes, in erythrocytes (Clark et al., 2004; Juel, Lundby, Sander, Calbet, & van Hall, 2003) after hypoxic exposure and in skeletal muscle

(Zoll et al., 2006). Additionally, an enhanced ability to buffer any exercise-induced lactate increases is also plausible, and has been supported by past research (Gore & Aughey, 2001; Mizuno et al., 1990; Saltin et al., 1995). In the acute stage of hypoxic adaptation, hyperventilation causes a reduction PCO_2, and increase in pH. This causes an increase in the excretion of renal bicarbonate which serves to buffer H^+ ions and act as a significant buffer of lactic acid. Such an increase (5-6%) in buffering capacity has been reported in research examining well-trained runners after two weeks living at 2,700 metres (Mizuno et al., 1990) and 2,000 metres (Saltin et al., 1995). Likewise, Gore and colleagues (2001) demonstrated that 23 simulated (altitude house) nights of LHTL at 3000m was sufficient to increase buffering capacity by 18%, but this groups findings were not replicated in a further study (Clark et al., 2004) using a slightly lower (~13%) simulated altitude (approximately equivalent to 4000m). The issue of lactic acid changes, in response to hypoxic exposure, certainly remains unanswered and should be considered and examined in future pieces of work in conjunction with physical performance tests where possible.

Cellular and molecular response to intermittent hypoxia

The field of cellular and molecular adaptation to hypoxia has only recently become more possible with advances in necessary analytical technology. As a result, advancement in understanding what is occurring at the cellular level during and after hypoxia is becoming possible. One of the early studies in examining hypoxic responses at a cellular level was Wang and colleagues (1995), whom investigated the hypoxia-inducible factor 1 (HIF-1). Subsequent work and reviews (Zhu & Bunn, 1999) have highlighted the importance of HIF-1

to the cascade of the hypoxic response. HIF-1 is found in the majority of tissue types around the body, and acts as a first-responder to hypoxia. In this manner it strongly regulates oxygen homeostasis, via the responding cardiovascular and respiratory mechanisms that are initiated during hypoxic exposure. During these periods of hypoxia, HIF-1 causes the increased transcription of specific genes, from which they activate proteins for specific responsive roles i.e. haematological, cardiovascular responses. At present, the understanding of how chronic and intermittent hypoxia differ in their respective influence of HIF-1 and other cellular mechanisms is unclear. However, there is evidence that clusters of genes are activated by intermittent hypoxia independent of chronic hypoxia (Prabhakar, 2001). Whilst this is a new area for exercise physiologists, there is likely to be further understanding in the field via the relevance of hypoxia to various medical conditions. The importance of further understanding the cellular and molecular basis of hypoxic adaptation is likely to substantially drive the field forward in the near future.

In summary, our understanding of the physiological response to chronic hypoxic exposure is well understood. However in contrast, our understanding of the physiological response(s) to intermittent hypoxia and how these may differ from chronic hypoxia is still developing. There is growing evidence to suggest that different mechanisms respond to the different stressors and it is likely that further advancement in cellular and molecular physiology will enhance the understanding of these differences. Whether these differences have any impact on the ergogenic effect of intermittent hypoxia remains unclear. Regardless, there is enough evidence to warrant further use and investigation of intermittent hypoxia as a tool for physical performance enhancement.

Altitude simulation technologies

A variety of devices have been developed to provide altitude simulation, including devices targeted for intermittent use. The fundamental purpose of these devices is to reduce physiological alveolar oxygen levels. This can be delivered by two means. The first, which most closely replicates environmental altitude, subjects are enclosed in a low-pressure environment (hypobaric chamber), thus reducing the oxygen uptake by way of the lower pressure gradient. Because of the need for a hypobaric chamber for this method, this is rarely used for physical performance research or for athletic performance. A more common approach is to replicate the lower-oxygen availability in a normobaric environment, by way of delivering a low-oxygen gas mixture. There are a variety of technologies that have been developed to achieve this. As a rule, these systems are either room-based or mask-based. The advantages and disadvantages of the various devices for applying intermittent hypoxia will be examined in more detail. Table 1 summarises the key aspects of each category of altitude simulation technology.

Table 1. Summary of the characteristics of intermittent altitude-simulation devices.

	Hypobaric chambers	N_2 apartment/room	Altitude-simulation tents	Rebreathing devices	N_2 dilution/O_2 filtration mask-based simulation
Cost	+	+	++	+++++	+++
Consumables and maintenance	O_2	N_2 for diluting air, plus costs of maintaining an apartment/room	N_2 required	CO_2 scrubbers, plus mixers for altering the hypoxic intensity	N_2 devices – requires constant supply of N_2. O_2-filtration: no consumables, semi-regular maintenance
Safety mechanisms	Limited; incidents have occurred at high hypoxic intensities	Limited; simulated altitude usually low enough to not allow incidents	Limited; simulated altitude usually low enough to not allow incidents	Limited; subjects holds mask to face to prevent incidents	Continual monitoring of SpO_2% and HR, allows instant cessation at low levels
Comfort and convenience	+	++++	++	+++	++++
Biofeedback	No	No	No	Manual adjustment, based on SpO_2%	Yes, some systems do have automatic biofeedback
Hypoxic intensity responsiveness and resolution	Hypoxic intensity can be very high, and relatively quick to adjust	Slow to alter hypoxic intensity due to large volume	Can adjust altitude, dependent on make/model	Manual adjustment of hypoxic intensity, low resolution of change	Pre-set for each session, resolution good
Notes	Most closely replicates geographical altitude	High cost, not practical for personal use	Can be noisy, not always practical	Most transportable form of IHE	With auto biofeedback provides the most individual form of IHE as it responds the most quickly to individual S_pO_2 changes

Responsiveness: refers to the time required to alter the hypoxic intensity delivered; **Biofeedback:** refers to the capability of the unit to adjust the absolute hypoxic intensity via monitoring of the subjects response; **Resolution:** refers to the range of hypoxic intensity that can be delivered by the system; + = poor; ++ = fair; +++ = good; ++++ = very good; +++++ = excellent

Hypobaric chambers

Hypobaric chambers are an effective altitude simulation device which work by reducing the ambient air pressure within the enclosed airspace of the chamber to reduce oxygen availability. The majority of hypobaric chambers are built for aerospace training. Hypoxic training is often incorporated as part of a pilots' initial training, and regular ongoing training is common (Cable, 2003). A number of air accidents have been attributed to pilot error in situations where the cabin and/or cockpit has depressurised, reducing oxygen availability very quickly and reducing the pilots cognitive function due to the lower cerebral oxygen delivery (Cable, 2003; Files, Webb, & Pilmanis, 2005). It is used as a means for providing first-hand physical experience of the effects of hypoxia without being in any danger. Pilots are taught to identify their own personal physiological effects during hypoxia so they can quickly identify the symptoms if/when depressurisation occurs during flights. Thus, the intention is that pilots will have the ability to identify symptoms and act quickly, especially in the early stages of hypoxia, and seek oxygen before remedying the situation. The chambers have also been used for altitude training research. The chambers replicate the environmental conditions of geographical altitude by reducing the air pressure. The oxygen content remains constant with sea-level, around 20.93% vol/vol. However, as a result of the lower air pressure the pressure gradient from the atmosphere to the alveolar point of gas exchange is reduced which leads to a lower oxygen availability. The primary advantage, from a research perspective, is the fact that these chambers replicate the pressure and resultant physiological consequences of geographical altitude more than other forms of artificial altitude training methods. The chambers are typically large

enough for a person to habituate for reasonably long periods of time. Similarly, because of their size, it is possible to exercise on an ergometer within some hypobaric chambers. However, the obvious challenge with using hypobaric chambers is that they are relatively scarce in nature, mostly due to the cost involved of building and operating such a system. In addition, using a hypobaric chamber for intermittent hypoxic exposure in the sense referred to in this review (less than three hours with alternating periods of hypoxia and normoxia) is somewhat challenging logistically. It would require the subject to move between self-contained rooms of hypobaric and normobaric environments. Obviously this is challenging for the subject, but also challenging for the chamber to maintain a hypobaric environment as the chamber depressurises when the doors are open. For these reasons, hypobaric chambers are seldom used for IHE and more often used for continuous hypoxic exposure (short- , and long-duration).

Figure 2. Example of the inside of hypobaric chamber being used for pilot hypoxic training (http://www.defence.gov.au)

Nitrogen apartments/rooms

The high costs of travelling to geographical altitude and the impracticality of using hypobaric chambers has driven the exploration for alternative simulation devices. One such method uses increased nitrogen levels as a means of diluting the quantity of oxygen in the inspired air. The air pressure remains unchanged whilst the oxygen content is reduced in direct inverse proportion to the increase in nitrogen content. Various mediums have been used to facilitate the inhalation of the altered air content. Nitorogen rooms or apartments are the most extravagant and costly of these options. The apartments are purpose-built, enclosed areas enabling a group of athletes to live comfortably within for relatively long periods of time. Nitrogen apartments were developed by Finnish sport scientists in the 1990's as a means for providing altitude-simulation to their athletes without travelling out of Finland, which is largely low-altitude (Wilber, 2007). The oxygen content can be manipulated by altering the quantity of nitrogen entering the room, thus providing the capability to replicate the oxygen availability experienced at various levels of geographical altitude. The obvious disadvantage to the apartment system is the cost involved in investing in such a purpose-built room or building. Despite the costs, a number of national sporting bodies have purpose-built such apartments for use by their athletes. Nitrogen apartments are mostly used for exposure durations (typically >8 hours/day) (Wilber, 2001) longer than that required by IHE.

Altitude simulation tents

Altitude simulation tents typically reduce the oxygen availability by oxygen filtration. The system draws in ambient air, and using semi-permeable membranes, or pressure-swing absorption technology, filters out a portion of the oxygen as it enters the tent (Wilber, 2007). The physiological concept remains the same as nitrogen apartments/rooms, with far lower costs involved. Altitude-simulation tents are typically designed to be big enough for an athlete to comfortably sleep within. The disadvantage to altitude-simulation tents is that their relatively small size makes spending extended periods of time inside them uncomfortable and impractical. The athletes typically sleep and spend as much time (outside of physical training) as possible within the apartment/room/tent, whilst training outdoors in a normobaric normoxic environment. Because of their relatively low cost, altitude-simulation tents have been used extensively by a large number of national sports bodies (Wilber, 2007). The nitrogen tents and oxygen-filtration tents both fit under the same umbrella of altitude simulation. That is, they utilise the typical, albeit artificial, “Live High, Train Low” (LHTL) method, which requires as much time a possible spent within the hypoxic environment (apartment/room/tent), whilst training in a normoxic, sea-level environment.

Figure 3. Example of a hypoxic tent using oxygen-filtration (http://www.hypoxico.com)

Mask-based systems

One of the main disadvantages of the altitude-simulation devices mentioned above (chambers, apartments/rooms, tents) is their limited portability and availability. Thus, subsequent development has been directed towards creating simulation devices that are far more convenient and accessible for regular use. This has resulted in the creation of masked-based systems that provide the necessary hypoxia. Whilst these systems are far more portable and convenient for use in a variety of locations, the fact they require the subject to be fitted with a mask for the duration of the session, and the associated movement restriction means that spending long durations wearing a mask is not practical. Perhaps as a result of this restriction, shorter duration protocols have been developed. Thus, the majority of short duration IHE protocols, that use mask-based systems are less than 90 minutes per session (Bärtsch, Dehnert, Friedmann-

Bette, & Tadibi, 2008). Because the duration of the dose is significantly shorter, the intensity of the hypoxia is typically higher than that used for longer duration hypoxic methodologies (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007; Wood et al., 2006). The advantage to the user being provided the hypoxic gas mixture via a face-mask is that the users' physiological variables can be more closely monitored simultaneously.

Re-breathing devices

As the name suggests, mask-based rebreathing devices use the subjects expired air (which has a lower oxygen content ~ 16%/vol at rest) as the hypoxic stimulus when the subject re-breathes it. The unit has a CO_2 scrubber, which serves to extract the CO_2 before being rebreathed. Each inspired breath contains a portion of expired air, mixed inside the unit with a portion of ambient air. The intensity of the hypoxia depends on the ratio of fresh ambient air to rebreathed expired air i.e. more ambient air introduced to the mixture reduces the hypoxic intensity, and vice versa. The unit(s) adjust the level of hypoxia delivered by modifying the volume in which the two gas mixtures are mixed. Subjects or supervisors of the subjects are required to monitor the SpO_2% closely in order to reach the target hypoxic intensity, and adjust the number of mixers accordingly until the target is reached. The number of mixers required may change during the course of a session as the SpO_2% drifts or the mixers deteriorate with use. Therefore, the primary disadvantage of this system is the user requirement to constantly monitor SpO_2%, and adjust mixers as and when required. In reality this means there is a relatively constant level of hypoxia for

long periods, as adjusting the mixing volume is somewhat labour-intensive. Rebreathing devices rely on CO_2 scrubbers to reduce the quantity of CO_2 being rebreathed. These scrubbers need to be replaced on a semi-regular basis depending on their usage to ensure inspired CO_2 levels do not rise. There has been concerns raised by reviewers (Bärtsch et al., 2008) regarding the large dead space volume of rebreathing devices (estimated at 0.5-1.0 litres). The issue is that the dead space volume, caused by the connecting tubing, may cause subjects to hyperventilate during the course of a session, which may interfere with the expected physiological responses and adaptations. Another issue with using rebreathing devices, which are entirely mechanical devices as opposed to electronic devices, is the associated safety concerns with not having any mechanism for stopping a session if the levels of oxygen saturation become dangerously low. Subjects, and/or supervisors, are solely reliant on monitoring the SpO_2% manually. To overcome these concerns, the mask is not fitted to the subjects face with a harness; rather subjects are required to hold the mask to their face for the session duration. If the subject was to become syncopic or lose consciousness, the mask would presumably fall off the subjects face and allow normoxia to resume. Despite the issues with using rebreathing devices, they are typically the cheapest form of IHE devices available as they use no electrical components, with the exception of the pulse oximeter. They are also the most portable and convenient form of short duration IHE device available which is particularly useful for athletes travelling.

Figure 4. Example of a rebreathing device (http://www.altipower.com)

Nitrogen dilution devices

Similar to the nitrogen-dilution method used by apartments/rooms, the masked-based system uses ambient air, and dilutes it with nitrogen before being delivered to the subject via the mask. The intensity of the hypoxia can be quickly adjusted by adjusting the flow of the nitrogen (more nitrogen leads to a greater hypoxic stimulus as the dilution of available oxygen increases, and vice versa). This can not be achieved as easily using a nitrogen apartment, as the volume of the room dictates the time required to provide sufficient additional nitrogen to dilute the environment, which depending on the speed at which nitrogen can be delivered, is certainly not rapid. Additionally, the requirement for nitrogen is significantly less using mask-based systems. The nitrogen-dilution devices available commercially are electronic devices, allowing for a very precise delivery of the additional nitrogen and consequently the level of hypoxia. Nitrogen-dilution devices require a constant source of nitrogen for their use, therefore the cost of this needs to be taken into account. The

prohibitive size and weight of transporting a nitrogen bottle means that the portability of such devices makes them unsuitable for some situations.

Figure 5. Example of a nitrogen-dilution device (http://www.airo.co.nz)

Oxygen-filtration devices

Mask-based oxygen-filtration systems use the same technology used for altitude-simulation tents; semipermeable membrane or pressure-swing absorption technology. Both systems serve to filter some of the oxygen content from the ambient air before being delivered to the subject. As with nitrogen-dilution devices, commercially available mask-based oxygen filtration devices are electronic, providing the capability to alter the hypoxic delivery very quickly. In comparison to rebreathing devices and nitrogen-dilution devices there are far less consumable costs required to maintain the system. The downside is that this is outweighed by the significantly larger cost price of a unit. Some such systems have the ability to have multiple users receiving IHE simultaneously; however the cost of such a “team” system makes them almost prohibitively expensive for commercial users only. Oxygen filtration units are typically larger and less-portable than rebreathers, making them far less practical for travelling and transporting.

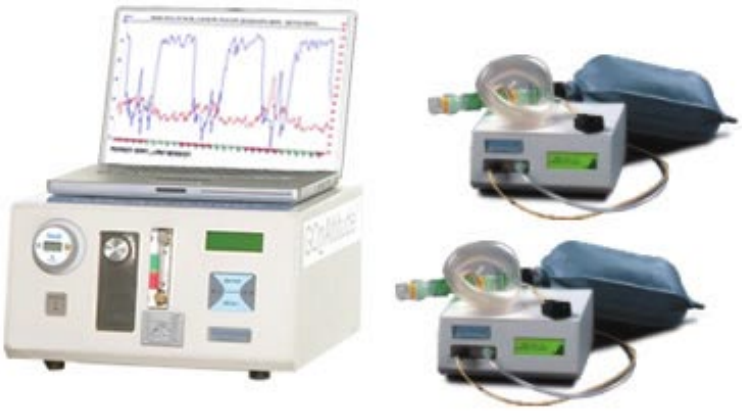

Figure 6. Example of an oxygen-filtration system (http://www.go2altitude.com)

Biofeedback mechanisms

The biggest advantage to the electronically monitored and delivered hypoxic systems is that they can utilise biofeedback to adjust the stimulus instantaneously. By measuring the SpO_2% via finger or ear oximeter, the individual response (at least in terms of SpO_2%) to hypoxic stimulus can be monitored. Two commercially available systems, the Hypoxicator (Biomedtech, Melbourne, Australia) and the Airo High Tech Mixing Head (Airo NZ Ltd, Auckland, New Zealand) systems have the ability to utilise the physiological feedback (biofeedback) from pulse oximeters to alter the hypoxic stimulus as and when appropriate. The majority of altitude-simulation systems (chambers, apartments/rooms, tents, rebreathing devices) use a constant level of hypoxic stimulus, typically described as either barometric pressure (mmHg) or fraction of inspired oxygen content (FIO_2), during the course of a session or day. Whilst this stimulus is often altered over the course of multiple sessions, to replicate ascension to altitude, it is not altered within a session. However, the biofeedback systems alter the hypoxic stimulus in concordance with the

individual users' physiological response (SpO_2%) to a given level of hypoxia. This is achieved by prescribing a target range for SpO_2% instead of a barometric pressure or FIO_2. Within a session, the FIO_2 fluctuates to meet the prescribed SpO_2% target range. Human physiology already provides us with built-in feedback mechanisms to deal with external stressors, to monitor and keep as constant as possible many physiological variables, such as temperature, blood pressure, and blood glucose. The bioefeedback system used by mask-based IHE systems uses a sensor outside of the body (pulse oximetry) to monitor then adjust the environmental conditions (quantity of oxygen being delivered to the subject) to maintain SpO_2% within the desired range. The proposed benefit of such biofeedback systems is that they will inevitably provide a stimulus that is more individualised, in comparison to the FIO_2-based prescription systems used in other altitude-simulation systems. Because the individual response to hypoxia varies, in terms of physical performance (Chapman, Stray-Gundersen, & Levine, 1998) and haematological responses (Friedmann et al., 2005; Ge et al., 2002), the biofeedback seeks to eliminate some of that variation so the stimulus received by the subject is more constant between subjects. Potentially, this may optimise the physiological adaptations and influence potential performance gains.

Intermittent hypoxic exposure protocols

There is a large body of research highlighting the beneficial effect of long duration hypoxia (geographical and simulated) on physical performance in various athletic populations (Wilber, 2001). However, there is less consensus that performance enhancements can be ascertained from short-duration IHE.

Since research to date has produced somewhat contrasting findings (Babcock & Kirby, 2008; Bonetti & Hopkins, 2009; Bonetti et al., 2006; Hamlin & Hellemans, 2007; Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007; Wood et al., 2006). Of the research meeting the review criteria, five studies (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Wood et al., 2006) reported some form of physical performance enhancement, whilst four studies did not (Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007). It is likely that a contributing factor to the conflicting results is the fact that there has been considerable variation in the protocols used to administer short duration IHE research. Specifically, these include:

i. Total number of sessions
ii. Total number of rest days
iii. Individual session duration
iv. Hypoxic intensity (whether FIO_2 or SpO_2%) and rate of ascension
v. ON-OFF durations/ratio
vi. Use of biofeedback mechanism

There are of course other sources of variation on the effects between studies, which are common to most sport performance research; selected population (age, sport, gender), performance measures and protocols utilised, training cycle (within the annual plan, and within the developmental training plan), and motivation and adherence.

By examining the short duration IHE protocols utilised in the nine studies mentioned in Tables 2-5, we can begin to interpret the findings with a little more clarity. The body of research, whilst still very small in comparison to other forms of altitude training, provides some insights as to what protocols might best mediate performance enhancement.

IHE duration

In terms of the total duration of short duration IHE-intervention, protocols from past research have ranged from 15 (Babcock & Kirby, 2008; Tadibi et al., 2007; Wood et al., 2006) to 26 days in total (Julian et al., 2004). This has included a range of 15-20 sessions of IHE. Benefits of short duration IHE appear to be mediated with only 15 sessions, as all of the studies showing benefit have used 15 sessions, spread over 15-20 days (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Wood et al., 2006). Whether or not these sessions are consecutive appears to be of little consequence to performance enhancement, although because of the small amount of research published, this relationship requires further investigation. The duration effect must also be matched with the mechanistic effect of performance enhancement mediation.

The duration of individual IHE sessions also appears to have little relevance on performance benefits. Studies eliciting a statistically or clinically significant improvement in some physical performance measure have used IHE sessions lasting between 60-90 minutes (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Wood et al., 2006), whilst studies finding no benefit have also used a duration range of 60-90 minutes

(Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007). It is likely that a number of factors have contributed towards the discrepancies in these findings, some of which may be related to the subject characteristics and study design. However, the conflicting findings to date provide some evidence that the duration of short duration IHE sessions, within a range of 60-90 min range, might not be critical. The intensity of the short duration IHE, however, may be more important.

IHE intensity

The intensity of short duration IHE may be influential in determining the physiological and performance enhancing effects. To date, two methods have been used to prescribe the intensity of the hypoxia being delivered. The first is the FIO_2, which is convenient as it allows a direct comparison to sea-level content, and also to an equivalent geographical altitude. All studies using this method of prescription have used a constant intensity for each individual session i.e. the same level of hypoxia for the duration of the "ON" phases of the session, with no concern for fluctuating SpO_2% (Hamlin & Hellemans, 2007; Julian et al., 2004; Tadibi et al., 2007). The alternative method adopted by some investigators (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Hinckson et al., 2007; Hinckson et al., 2006; Wood et al., 2006), uses the actual individuals SpO_2% for prescription purposes, usually within a ~3% range for each session. Of the studies mentioned above, that have utilised SpO_2% as the prescription tool the majority have reported performance improvements, in measures of aerobic performance, ranging from 1.3 to 8.2 % (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Wood et al., 2006). However two studies (Hinckson et al., 2007; Hinckson et al., 2006) using SpO_2% reported no

clear changes in this regard. Studies using the fixed FIO_2 method of hypoxic intensity prescription only one study (Hamlin & Hellemans, 2007) reported significant of substantial change in their respective physical performance measures (Tables 2 to 4). The method for prescribing the hypoxic intensity is typically related to the technology being used to prescribe the hypoxic stimulus, and also the use of a biofeedback system. As mentioned previously, the use of biofeedback appears an important element for short duration IHE. Studies prescribing intensity using SpO_2%, require a biofeedback mechanism to be able to maintain the appropriate stimulus. For some of the research (Bonetti et al., 2006; Bonetti et al., 2009; Hinckson et al., 2007; Hinckson et al., 2006; Wood et al., 2006), especially those using rebreathing devices, this was done manually by research supervisors observing the subjects SpO_2% and making adjustments to the hypoxic intensity manually by adjusting the effect of the respective device.

IHE Protocol

Within a typical IHE session (duration = 60-90 minutes), periods of hypoxia are alternated with periods of normoxia. The majority of studies to date have adopted very similar IHE protocols involving either five minutes “ON” (hypoxia), followed by five minutes “OFF” (normoxia) (Bonetti et al., 2006; Hamlin & Hellemans, 2007; Julian et al., 2004), or six minutes “ON”, followed by four minutes “OFF”, both contributing to 10-minute cycles (Hinckson et al., 2007; Hinckson et al., 2006; Tadibi et al., 2007; Wood et al., 2006). Whilst there are significant differences in the reported outcome measures (haematological, physiological and performance, Tables 2-5) of the studies mentioned above, it is

likely that these differences are caused by other factors such as the use of biofeedback mechanism and the characteristics of the subjects. In an attempt to determine if indeed the chosen short duration IHE protocol is influential, Bonetti et al. (2009) specifically examined the difference between 5:5min vs 3:3min "ON-OFF" protocols in well-trained cyclists. Whilst both groups exhibited improvements in physical performance, the differences between the two groups was unclear for a range of measures including: peak power (2.2; ± 3.9% (±90% CL)), lactate-profile power (-1.3; ± 3.0% (±90% CL)) and heart-rate profile power (-1.4; ± 3.9% (±90% CL)) at three days post-IHE. Thus, it appears that the "ON-OFF" ratio has little impact on physical performance, however further research could be aimed at examining ratios higher than 60% hypoxia (i.e. seven minutes "ON", followed by three minutes "OFF").

Table 2. Intermittent hypoxic exposure; effects on explosive/sprint performance

Author (Year)	Subject characteristics	Group size and gender breakdown	Sessions/ days	Session duration (min)	Hypoxic protocol	Performance measure(s)	Outcome
Bonetti et al. (2006)	10 sub-elite kayakers	HYP = 10M, CON = 10M (X-over design)	15/19	60	5min on, 5min OFF; SpO_2 90 - 76%	500 metre TT	↔ 0-10 second power
Bonetti et al. (2009)	18 competitive cyclists and triathletes	HYP_{3min} = 9M, HYP_{5min} = 9M; CON = 9M	15/19	60	HYP_{3min} : 3min ON, 3min OFF; HYP_{5min}: 5min ON, 5min OFF; SpO_2 90 - 76%	Repeated sprint test	↔ HYP_{3min}/HYP_{5min} first sprint power
Hinckson et al. (2007)	10 rugby players	HYP = 5M; CON = 5M	14/14	60	6min ON; 4min OFF; SpO_2 100-76%	Rugby simulation task	↓ scrummaging peak power, offensive and defensive sprint performance.
Tadibi et al. (2007)	20 endurance-trained athletes	HYP = 10M; CON = 10M	15/15	60	6min ON, 4min OFF; 11-10% FIO_2	Wingate	↔ peak power in Wingate test
Wood et al. (2006)	29 hockey and soccer players; National, regional, club level	HYP = 15M; CON = 14M	15/15	60	6min ON, 4min OFF; SpO_2 90 - 77%	RHIET	↑ first sprint time performance in RHIET

Table 3. Intermittent hypoxic exposure; effects on repeated sprint ability/anaerobic ability

Author (Year)	Subject characteristics	Group size and gender breakdown	Sessions/ days	Session duration (min)	Hypoxic protocol	Performance measure(s)	Outcome
Bonetti et al. (2006)	10 sub-elite kayakers	HYP = 10M, CON = 10M (X-over design)	15/19	60	5min on, 5min OFF; SpO_2 90 - 76%	Repeat kayak sprint test	↑ mean repeat power
Bonetti et al. (2009)	18 competitive cyclists and triathletes	HYP_{3min} = 9M, HYP_{5min} = 9M; CON = 9M	15/19	60	HYP_{3min} : 3min ON, 3min OFF; HYP_{5min}: 5min ON, 5min OFF; SpO_2 90 - 76%	Repeated cycling sprint test	↔HYP_{3min}/HYP_{5min} mean sprint power
Tadibi et al. (2007)	20 endurance-trained athletes	HYP = 10M; CON = 10M	15/15	60	6min ON, 4min OFF; 11-10% FIO_2	Wingte test	↔ Wingate performance measures
Wood et al. (2006)	29 hockey and soccer players; National, regional, club level	HYP = 15M; CON = 14M	15/15	60	6min ON, 4min OFF; SpO_2 90 - 77%	Incremental ergometer test, RHIET	↑ RHIET performance (last sprint, total time)

Table 4. Intermittent hypoxic exposure; effects on endurance performance, and associated physiological measures

Author (Year)	Subject characteristics	Group size and gender breakdown	Sessions/ days	Session duration (min)	Hypoxic protocol	Performance measure(s)	Outcome
Babcock et al. (2008)	18 well-trained cyclists	HYP = 9M, CON = 9M	15/15	?	SpO_2 90 -77%	15km and 3km cycling TT	↑ 15km TT performance in HYP group only, ↔ 3km TT
Bonetti et al. (2006)	10 sub-elite kayakers	HYP = 10M, CON = 10M (X-over design)	15/19	60	5min on, 5min OFF; SpO_2 90 - 76%	Incremental ergometer test	↑ peak power, ↔ $VO_{2\ peak}$, lactate threshold, economy.
Bonetti et al. (2009)	18 competitive cyclists and triathletes	HYP_{3min} = 9M, HYP_{5min} = 9M; CON = 9M	15/19	60	HYP_{3min} : 3min ON, 3min OFF; HYP_{5min}: 5min ON, 5min OFF; SpO_2 90 - 76%	Incremental ergometer test	Combined HYP_{3min} andHYP_{5min} ↑peak power, lactate-profile power, HR-profile power compared with CON group
Hamlin & Hellemans (2007)	22 Non-elite multisport athletes	13M, 9F; HYP = 12; CON = 10	15/19	90	5min on, 5min OFF; 13-10% FIO_2	3km running TT	↑ 3km TT performance at 2d and 17d post-exposure
Hinckson et al. (2006)	11 elite-level rowers	HYP = 5F, 2M; CON = 4F, 1M	15-20/20	90	6min ON, 4min OFF; SpO_2 92 - 80%	5km rowing TT	↔ 5km TT performance
Hinckson et al. (2007)	10 rugby players	HYP = 5M; CON = 5M	14/14	60	6min ON; 4min OFF; SpO_2 100-76%	MSFT	↔ MSFT performance
Julian et al. (2004)	14 elite-level runners	HYP = 7M, CON = 7M	20/26	70	5min ON, 5min OFF; 12-10% FIO_2	3km running TT	↔ $VO_{2\ max}$, 3km TT performance

Author (Year)	Subject characteristics	Group size and gender breakdown	Sessions/ days	Session duration (min)	Hypoxic protocol	Performance measure(s)	Outcome
Tadibi et al. (2007)	20 endurance-trained athletes	HYP = 10M; CON = 10M	15/15	60	6min ON, 4min OFF; 11-10% FIO_2	Incremental cycle ergometer test	↔ VO_{2max}, physical performance measures.
Wood et al. (2006)	29 hockey and soccer players; National, regional, club level	HYP = 15M; CON = 14M	15/15	60	6min ON, 4min OFF; SpO_2 90 - 77%	Incremental ergometer test	↑maximum running speed, HR- and B[Lac]-profile speeds

Table 5. Intermittent hypoxic exposure; effects on haematological measures

Author (Year)	Subject characteristics	Group size and gender breakdown	Sessions/days	Session duration (min)	Hypoxic protocol	Outcome
Babcock et al. (2008)	18 well-trained cyclists	HYP = 9M, CON = 9M	15/15	?	SaO_2 90 -77%	↔ Hct, reticulocytes, ferritin
Bonetti et al. (2006)	10 sub-elite kayakers	HYP = 10M, CON = 10M (X-over design)	15/19	60	5min on, 5min OFF; SpO_2 90 -76%	↑ Hb mid-intervention and 3-days post, ↑ Hct mid-intervention,↓ ferritin at 3-days post exposure
Bonetti et al. (2009)	18 competitive cyclists and triathletes	HYP_{3min} = 9M, HYP_{5min} = 9M; CON = 9M	15/19	60	HYP_{3min} : 3min ON, 3min OFF; HYP_{5min}: 5min ON, 5min OFF; SpO_2 90 -76%	↑ reticulocytes at 0, 14 -days, post exposure ↓ ferritin at 0 days, ↔ 2,3 DPG, WBC
Hamlin & Hellemans (2007)	22 Non-elite multisport athletes	13M, 9F; HYP = 12; CON = 10	15/19	90	5min on, 5min OFF; 13-10% FIO_2	Possible ↓ ferritin, ↔ Hb, possible ↑ reticulocytes, Hct
Hinckson et al. (2007)	10 rugby players	HYP = 5M; CON = 5M	14/14	60	6min ON; 4min OFF; SpO_2 100-76%	↑ ferritin, ↔ Hb, Hct, reticulocytes at 2-days post exposure
Julian et al. (2004)	14 elite-level runners	HYP = 7M, CON = 7M	20/26	70	5min ON, 5min OFF; 12-10% FIO_2	↔ Hct, Hb, EPO, sTfR
Tadibi et al. (2007)	20 endurance-trained athletes	HYP = 10M; CON = 10M	15/15	60	6min ON, 4min OFF; 11-10% FIO_2	↔ EPO, RBC, Hct,
Wood et al. (2006)	29 hockey and soccer players; National, regional, club level	HYP = 15M; CON = 14M	15/15	60	6min ON, 4min OFF; SpO_2 90 -77%	↑ WBC, possible ↑ Hct, Hb at 1-day

Hb = Haemoglobin, Hct = Haematocrit, EPO = Erythropoietin, sTfR = Soluble Transferrin Receptor, WBC = White blood cells, RBC = Red blood cells, 2,3 DPG = 2,3-diphosphoglycerate

Effects of IHE on physical performance and related physiological parameters

Whilst sport scientists are inevitably interested in the underlying mechanisms for any changes in performances, these become of secondary importance when dealing with an athletic population as performance enhancement is the driving factor. This section of the review of literature will examine the effect of short duration IHE on physical and sport performance.

The effect of short duration IHE on physical performance

The review of the effect of short duration IHE on physical performance is divided into three elements of physical performance: 1) explosive/sprint performance; 2) repeated sprint ability (RSA); and 3) endurance performance.

Explosive/sprint performance

There are relatively few papers that have investigated the effect of short duration IHE on explosive muscular power or single sprint performance. This is not surprising, considering the physiological rationale for IHE is not analogous with maximal power and sprint performance. All of the five studies (Babcock & Kirby, 2008; Bonetti et al., 2006; Bonetti et al., 2009; Tadibi et al., 2007; Wood et al., 2006) from which a measure of power or sprint performance has been measured and described has been the first (or one of

the first) repetition of a repeated maximal effort test (i.e. Wingate, RHIET). However, only one study (Wood et al., 2006) has shown an enhancement of such a measure (first sprint time in the RHIET; - 1.5; ± 1.7 (±90% CL); benefit likely), at 3-days post-IHE. All other studies have either shown no substantial change (Bonetti et al., 2006; Bonetti et al., 2009; Tadibi et al., 2007), or an impairment, in power and/or maximal sprint performance (6.8; ± 7.8% (±90% CL), and 11.0; ± 9.0% (±90% CL) impairment in offensive and defensive sprint times respectively) (Hinckson et al., 2007). Hinckson and colleagues (2007) are the only group to assess sport-specific measures of power. Specifically, they measured peak power generated by academy rugby players during simulated scrummaging within a simulated rugby performance test. Peak power during these two simulated scrimmaging sessions was impaired (-14.0; ± 8.0% (±90% CL) and -9.0; ± 7.0% (±90% CL)) compared to control subjects. The sample size of the group (5=HYP, 5=CON), the uniqueness of the task, and the commitment of the subjects, which the authors questioned, may however have contributed to the impairment. Whilst there is no clear physiological rationale for improvements in such short duration performances as a result of short duration IHE, it is also unlikely impairment could come from IHE in such tasks, as the majority of research suggests. Whilst the authors acknowledge the possibility that insufficient time was allowed for recovery after the intervention prior to performance measures being assessed (1- or 4-days post-IHE), other studies (Bonetti et al., 2006; Bonetti et al., 2009) have not reported impairments in explosive performance at a similar time post-IHE (three days).

Repeated sprint ability (RSA)

The ability to repeatedly sprint at a high intensity has been shown to be strongly correlated to aerobic capacity (Bishop, Edge, & Goodman, 2004; Tomlin & Wenger, 2001). It is therefore possible that adaptations associated with short duration IHE could have beneficial effects on RSA. Several studies have measured RSA after IHE interventions involving athletes, of which some (Bonetti et al., 2006; Bonetti et al., 2009; Wood et al., 2006) have reported clinically significant enhancements in their respective RSA measures. For example, Wood and colleagues (2006), using intermittent team-sport athletes, reported substantial improvements, compared to a control group, at three (-5.1; ± 2.2% (±90% CL)) and 12 days (-3.8; ± 1.7% (±90% CL)) post-IHE exposure in total sprint time from the RHIET. Similarly, but in endurance kayakers, Bonetti and colleagues (2006) reported a very likely benefit (8.3; ± 6.7% (±90% CL)) in a repeated-kayaking ergometer test at three days post-IHE but this was unclear (3.0; ± 7.2% (±90% CL)) by 10 days post-IHE. In contrast, during another study by the same researchers (Bonetti et al., 2009), an impairment of mean sprint power (-2.0; ± 2.6% (±90% CL)) was reported in a repeated cycling sprint test at 3 days post-IHE. It is possible that some of the discrepancies in findings are due to the difference in protocols between RSA assessment in different sports. Also, the small number of studies in the area confounds the lack of clarity. Further research is required to establish the effect short duration IHE has on RSA.

Endurance performance

It is perhaps not surprising that all studies presented in Tables 2-5 have reported the effect of short duration IHE on some measure of endurance performance. Several of those studies have reported an improvement (1.7-8.2% performance improvement) in their respective measure of endurance performance including 15 km cycling time-trial (TT) (Babcock & Kirby, 2008), 3km running TT (Hamlin & Hellemans, 2007), peak power within an incremental step test in cycling (Bonetti et al., 2009) and kayaking (Bonetti et al., 2006), and maximum aerobic speed in the multi-stage fitness test (MSFT) (Wood et al., 2006). In some studies, endurance performance changes were found at three days post-exposure (Bonetti et al., 2006; Bonetti et al., 2009), however benefits had become unclear by 10 (Bonetti et al., 2006) and 14 days (Bonetti et al., 2009) post-IHE. The subject characteristics of most studies have been "trained", with the exception of subjects in Hamlin and Hellemans (2007), whom came from a range of training ages and were described as "mixed ability". These authors, in addition to reporting significant improvements in a 3km running TT, also highlighted the fact that the largest enhancements in performance from within the HYP group were found in those subjects with the lowest baseline performance in the 3km TT. This suggests that lower-level athletes who have a greater room for improvement are therefore more susceptible to performance improvements using similar training stimuli. This provides evidence that this variation in response to training stimuli applies to short duration IHE as well.

Other studies, however, have reported no significant change in endurance performance after short duration IHE. In particular, two studies (Julian et al., 2004; Tadibi et al., 2007) using highly-trained endurance athletes both found no significant change in endurance performance as measured by an incremental cycle test (Tadibi et al., 2007) and a 3km TT (Julian et al., 2004). Both of these studies used a protocol of short duration IHE whereby they were exposed intermittently to a fixed FIO_2 for one week at a time. More specifically, Tadibi et al. (2007) used a Douglas bag with an oxygen concentration of 11% for days 1-7, then reduced to 10% for days 8-15 of the intervention (60min/session, 6 minutes “ON’, 4 minutes “OFF”) . This is slightly different to studies that have reported at least some degree of benefit from short duration IHE for two reasons; a) the number of increments to the hypoxic intensity was significantly lower than other studies. Tadibi et al. (2007) and Julian et al. (2004) both used only two increments compared with six (Wood et al., 2006) to eight (Bonetti et al., 2006; Bonetti et al., 2009) increments used in other studies; and b) the absolute level of hypoxia remained constant within a single session, whereas previously mentioned work uses manual biofeedback mechanisms to ensure the subjects receives a constantly adjusting level of hypoxia within each session. It is also possible that by using already highly-trained subjects, that there is less room to further enhance performance. Two other studies (Hinckson et al., 2007; Hinckson et al., 2006) also found no clinically significant endurance performance benefit from using short duration IHE in a 500 m or 5 km rowing TT (Hinckson et al., 2006), and a modified MSFT (Hinckson et al., 2007).

The effect of IHE on performance-related physiological measures

Peak aerobic capacity ($\dot{V}O_{2peak}$)

Peak aerobic capacity ($\dot{V}O_{2peak}$), used almost synonymously with $\dot{V}O_{2max}$, is defined as maximum capacity of an individual to transport and utilize oxygen during incremental exercise. It is considered the single best measure of cardiorespiratory fitness (Wilmore & Costill, 2004).

Surprisingly, there have been only four studies (Bonetti et al., 2006; Bonetti et al., 2009; Julian et al., 2004; Tadibi et al., 2007) that have measured and reported $\dot{V}O_{2peak}$ after varying periods of IHE interventions. None of these studies reported an improvement in $\dot{V}O_{2peak}$. This evidence is not consistent with the prevailing theory that hypoxia mediates performance enhancement by improving relevant haematological variables, thus enhancing oxygen-carrying capacity and $\dot{V}O_{2peak}$ (Levine & Stray-Gundersen, 2005; Levine, Stray-Gundersen, Gore, & Hopkins, 1997; Stray-Gundersen, Chapman, & Levine, 2001). Conversely, given that these studies reported performance enhancement in other trials, suggests that there are other mechanisms at play mediate performance enhancements from short duration IHE.

Lactate threshold

The definition of the lactate threshold varies considerably in the literature (Faude, Kindermann, & Meyer, 2009). However, to most, it generally

demarcates the point at which there is change in the ratio between lactate production and lactate removal (Svedahl & MacIntosh, 2003). It is a sensitive to training stimuli (Acevedo & Goldfarb, 1989) and consequently is often used for assessing adaptation to training (Bosquet, Leger, & Legros, 2002), predicting endurance performance capability (Bosquet et al., 2002) and prescribing training intensities for endurance athletes (Kindermann, Simon, & Keul, 1979). The threshold is assessed in two broad formats; the intensity at which B[Lac] first rises above baseline levels and the highest intensity at which the production and removal of B[Lac] are equal (Faude et al., 2009). Many practical methods exist to measure this actual exercise intensity, and they differ slightly in their definition and terminology. Several short duration IHE studies have reported some form of lactate-threshold measure such as lactate-profile power (Bonetti et al., 2009), lactate-profile speed (Wood et al., 2006), P_{4mmol} (Hinckson et al., 2006; Tadibi et al., 2007), and lactate threshold (Bonetti et al., 2006). Of these, only two studies (Bonetti et al., 2009; Wood et al., 2006) reported an enhancement in their reported measure of lactate threshold after short duration IHE. Wood and colleagues (2006) reported HYP group subjects exhibited an almost certain benefit (3.7; ± 1.6% (±90% CL)) in lactate profile speed, as measured during a modified MSFT (one minute rest was taken every three minutes to take finger-prick blood samples). This enhancement was maintained until 12 days post-exposure (3.7; ± 1.4% (±90% CL)). Similarly, Bonetti et al. (2009) found that a group of cyclists exhibited a very likely benefit in lactate-profile power (6.5; ± 5.3% (±90% CL) after short duration IHE, however this change became unclear after 14 days. In contrast, other studies (Bonetti et al., 2006; Hinckson et al.,

2006; Tadibi et al., 2007) found no change for measures of lactate threshold. Whilst Bonetti and colleagues (2006) reported no substantial change in lactate threshold in a group of well-trained kayakers after short duration IHE (3.5; ± 7.0% (±90% CL)), the authors did report a notable rightward shirt in their lactate profile curve which suggest some underlying adaptation did occur.

Exercise economy

Recently, a plausible theory has been developed regarding the adaptations that occur as a result of real or simulated continuous hypoxic interventions. Specifically, several studies, using a variety of hypoxic methodologies (Green et al., 2000; Katayama, Matsuo, Ishida, Mori, & Miyamura, 2003; Katayama et al., 2004; Saunders et al., 2003), not including short duration IHE, have reported improvements (2-10%) in exercise economy in a variety of athletes. Exercise economy is defined as the energy demand for a given power output of submaximal work (Saunders, Pyne, Telford, & Hawley, 2004). However, to date, no short duration IHE studies have shown any such improvements in economy after short duration IHE. Babcock et al. (2008) did show a trend towards an improvement in economy, but it was not statistically significant using traditional statistical analysis ($p = 0.075$). More specifically, the study used $\dot{V}O_2$index (calculated as $\dot{V}O_2$ per $watt_{avg}$) to assess economy. Several other studies (Bonetti et al., 2006; Bonetti et al., 2009; Tadibi et al., 2007) have measured exercise economy, and reported unclear changes in economy after short duration IHE interventions. There is substantially more research

examining economy using other methods of hypoxic exposure, therefore with such little evidence using short duration IHE, further investigation of IHE is warranted.

The effect of short duration IHE on haematological measures

The most commonly theorised mechanism for mediating performance as a result of hypoxic exposure in general, is the enhancement in oxygen-carrying capacity via a change in haematological measures, in particularly EPO, Hb, and RBC (Levine & Stray-Gundersen, 2005; Levine et al., 1997; Stray-Gundersen et al., 2001). Therefore, it is no surprise that the majority of known short duration IHE studies have documented at least some representative haematological measurements. It is difficult to accurately interpret the findings from some studies because of lack of details provided. For example, Babcock & Kirby (2008) reported no changes to haematological measures in their study but did not detail the specifics of the results. Likewise, Tadibi and colleagues (2007) stated that each of the variables (haemoglobin, red blood cells, erythropoietin, haematocrit) did not change significantly between pre- and post-test results and between-groups. The group reported pre and post-IHE haematological results (means ± SD) but the statistical significance, in terms of actual p-values or effect sizes, were not reported. Fortunately, Hinckson and colleagues (2007) clearly reported their haematological results after short duration IHE, showing trivial but unclear changes in haemoglobin (Hb) (2.0; ± 4.7% (±90% CL)), haematocrit (Hct) (0.9; ± 5.2% (±90% CL)) and reticulocytes (0.7; ± 36.0% (±90% CL)), whilst

serum ferritin (10.5; ± 21.6% (±90% CL)) showed a trivial but clear change in the HYP group in comparison to the CON group. This increase in ferritin is unique within short duration IHE studies, as all other previous work has produced, either a clinically significant decrease (Bonetti et al., 2006; Bonetti et al., 2009) or trivial, but clinically insignificant, decrease (Hamlin & Hellemans, 2007) in ferritin in at least one of the post-intervention sampling points. This includes Hamlin & Hellemans (2007), which reported a possible ferritin reduction, whilst not being clinically significant (-8.9; ± 11.8% (±95 % CL)) at two days post-exposure, and after 12 days post-exposure (-4.8; ± 12.2% (±95 % CL)). Similarly, all haematological variables (Reticulocytes, Hct, Hb) reported by Hamlin and Hellemans were clinically insignificant despite the subjects producing significant performance benefits in a 3km running TT. However, possible beneficial changes were observed in reticulocytes (5.2; ± 8.7% (±95% CL)) and Hct (1.4; ± 1.7% (±95% CL)) at two days post-exposure, and Hb (3.6; ± 6.2% (±95% CL)) at 12 days post-exposure. Conversely, Julian (2004), using a group of elite distance runners, reported no significant differences between HYP and CON groups for any haematological variables. However, significant differences were seen in various measures within groups across the seven sampling points. Specifically, erythropoietin (EPO) was significantly lower than baseline in both groups post-IHE ($p<0.01$). Also, Hb was significantly higher ($p<0.05$) in the HYP group after 26 days compared with immediately post-IHE, but not compared with baseline. Conversely, Hb in the CON group was significantly less ($p<0.05$) 10 days post-IHE than baseline. Reticulocytes were significantly less ($p<0.05$) during the exposure than 10 days after exposure in

the HYP group, and although this was similar in the CON group, significance was lost. This is in accordance with reticulocytes changes of other research (Bonetti et al., 2009). Bonetti and colleagues reported a clinically significant increase in reticulocytes immediately (14.0; ± 17.0% (±90% CL)) after short duration IHE intervention, and this significance was enhanced at 14 days post-exposure (40.0; ± 32.0% (±90% CL)) in the combined HYP groups compared with the CON group. This was accompanied by a substantial increase in Hb at 14 days post-exposure (2.9; ± 3.4% (±90% CL)), and a reduction in ferritin immediately post-exposure (-15.0; ± 16.0% (±90% CL)). The reduction in ferritin has been reported by the same group in previously published work (Bonetti et al., 2006) at three days post-exposure (19.0; ± 15.0% (±90% CL)), at which point haemoglobin was elevated (3.6; ± 3.2% (±90% CL)) also above baseline in comparison to CON group subjects. However, Hb levels had peaked during mid-intervention sampling (10 days into IHE intervention) (4.0; ± 2.1% (±90% CL)). These changes are partly supported by changes in Hb and Hct measures seen in intermittent team sport athletes (Wood et al., 2006), whom exhibited possible changes in the respective measures at three days post-exposure.

Identifying trends between studies in changes to haematological variable is challenging when sampling points are not universal between studies, and the measures taken differ slightly (presumably due to the costs associated with laboratory sampling and analysis). In addition, the cause of change in some the relevant haematological variables are not isolated to the influence from hypoxic exposure. Dietary and physical activity levels/modalities can affect

these measures in isolation and in partnership with hypoxic exposure. For example, the influence of dietary iron has a significant effect of Hb and Hct levels. This is manifested further in the relevant short duration IHE studies due to the inconsistencies in the use of iron supplementation. Some studies provided iron tablets for all subjects (Julian et al., 2004; Wood et al., 2006), some provided iron tablets to subjects with "low iron" levels (Bonetti et al., 2006; Bonetti et al., 2009; Hamlin & Hellemans, 2007; Hinckson et al., 2006), of which each study used a slightly different cut-off for their definition of "low iron", and some provided no iron supplementation (Babcock & Kirby, 2008; Hinckson et al., 2007; Tadibi et al., 2007). Similarly, physical activity has been shown to effect such variables as reticulocytes, independent of any hypoxic exposure (Schmidt, Eckardt, Hilgendorf, Strauch, & Bauer, 1991). To confuse the issue regarding the triangular relationship between physical performance, haematological changes and hypoxic exposure further, and as mentioned in section one, it is possible that changes to performance are not necessarily mediated by changes in haematological measures, as reviewed by Gore and colleagues (Gore et al., 2007).

IHE: Application to continuous vs intermittent sports

The majority of research published investigating the effects of hypoxia (geographical altitude, long- and short-duration artificial methods) on physical performance has used cyclical endurance-based sports (i.e. running, cycling, swimming). It seems logical that athletes from endurance-based sports are most likely to benefit given the theorised adaptations achieved after exposure

to hypoxia. Whilst there may be some conjecture, it is relatively agreeable, based on a meta-analysis by Bonetti and Hopkins (2009), that for endurance-based sports, the majority of hypoxic exposure types can increase performance in sub-elite, and possible elite, athletic populations. However, there is less published research, and far less research and agreement regarding the effects of all types of hypoxic exposure on the physical performance of athletes from intermittent sports. For the purposes of this review, intermittent team-sports are of most interest. It would seem logical, given that the aerobic contribution to performance in intermittent team-sports is less than the aerobic contribution to cyclical endurance-based sports, that there would be less benefit to be had from hypoxic exposure. Anaerobic performance is also important for most team-sport athletes, including basketball, of which a body of evidence has developed examining the physiology and movements patterns of the sport. These highlighted the large numbers of high-speed movements (jumping and sprints) and high lactate levels during competition (Fox, 1984 ; Gillam, 1985; McInnes, Carlson, Jones, & McKenna, 1995; Rodriguez-Alonso, Fernandez-Garcia, Perez-Landaluce, & Terrados, 2003). This body of evidence places a high value of importance on the anaerobic pathways. This would suggest that improving aerobic capacity, and therefore hypoxic exposure for performance-enhancing purposes, would be of lower value and/or priority. Similar principles can be applied across other team sports. There is a misconception that aerobic capacity is unimportant for intermittent team-sport athletes, and assumed that hypoxic exposure will not be beneficial for such athletes.

Early research suggested that the ATP-CP energy system was the dominant energy system in basketball account for ~85% of total energy supply (Fox, 1984). However, more recent research in basketball (Abdelkrim, El Fazaa, & El Ati, 2007; McInnes et al., 1995; Taylor, 2003), rugby union (Duthie, Pyne, & Hooper, 2005), hockey (Spencer et al., 2004), and soccer (Ali & Farrally, 1991; Krustrup, Mohr, Ellingsgaard, & Bangsbo, 2005) have shown there to be a far higher contribution from the aerobic energy system. The ability to recover between high-intensity efforts during a game is strongly related to aerobic capacity (Tomlin & Wenger, 2001), therefore an enhanced aerobic capacity will enhance the ability to perform repeated bouts of short-duration high-intensity activities (sprints, jumps etc.) which are typical of intermittent team sports. Therefore it would appear plausible that team sport athletes may also benefit from short duration IHE. In addition, it is possible that team sport athletes have more to gain from such exposure, by virtue of the fact they are further from their genetic ceiling in term of their respective aerobic capacity than athletes from cyclical endurance-based sports. This hypothesis is based on the assumption that team sport athletes have a lower maximum oxygen uptakes than endurance athletes (Wisloff, Helgerud, & Hoff, 1998).

Summary

A considerable amount of research has been dedicated to examining the physiological response, and adaptations and performance consequences that occur as a result of continuous hypoxic exposure. Such effects are relatively well understood. However, substantially less is known regarding the effects of short term IHE on physical responses and performance. From studies to date,

there is contrasting evidence regarding the efficacy of short duration IHE on responses and physical sports performance. This could be due to methodological (subjects, design and hypoxic dose prescription) and technological (administration method of hypoxia) differences between studies. A limited amount of research has been performed using team sports athletes. The shortage of research in the area, and the lack of agreement between existing studies for individual endurance sports suggest that further investigation of the potential benefits of short duration IHE is required.

Chapter Three: Methodology

Experimental approach to the problem

This study adopted a single-blind experimental design. In matched pairs (for performance in YYIRT running $speed_{peak}$), subjects were assigned to one of two groups: an experimental (HYP) or control (CON) group. Subjects were required to participate in a 15-day intervention during which they passively inhaled (during seated rest) either hypoxic or normoxic gas once per day, intermittently, for 37-minutes. Before and after the intervention, subjects participated in a range of physical tests which measured their physical performance, and related physiological variables. In addition, several haematological measures were obtained at three time-points throughout the study.

Subjects

With institutional ethics approval (AUT University Ethics Committee, Appendix 1), 24 male basketball players volunteered to take part in the study. The mean (± SD) age, height, and body mass of the subjects that completed the intervention were 21.7 ± 4.4 years, 186.7 ± 10.8 cm, 87.2 ± 15.6 kg respectively. Subjects were recruited from National Basketball League (NBL) and Conference Basketball League (CBL) teams in the Auckland region. The CBL is the developmental league for New Zealand elite men's competitions. Thus, the subjects were considered well-trained basketball players. All

participants were informed of the procedures, risks, and benefits of the study. All subjects were provided with a study information sheet (Appendix 2) and given the opportunity to ask questions and make sure they understood the requirements of the study before commencing. Subsequently, all subjects provided written informed voluntary consent to participate in the study (Appendix 3), and completed an additional medical questionnaire (Appendix 4).

Experimental procedures

Several performance and physiological tests were performed over two days before and after the intervention, in addition to a familiarisation test conducted one week earlier. A schematic of the study design is illustrated in Figure 7. In total, this study involved six separate testing sessions with adequate recovery provided between the various physical testing sessions. Sessions 1 and 2 (PRE7) were used as familiarisation sessions which were conducted 6-7 days prior to commencing the breathing intervention. Sessions 3 and 4 (PRE) were conducted 1-2 days prior to the breathing intervention commencing. Sessions 5 and 6 (POST) were conducted 10 days after the breathing intervention had concluded. During test sessions 1, 3 and 5 subjects participated in a laboratory-based test of peak aerobic capacity ($\dot{V}O_{2peak}$). During test session 2, 4 and 6 subjects participated in two court-based tests of basketball-specific fitness: 1) the Repeated high-intensity endurance test (RHIET); and 2) the Yo-Yo intermittent recovery test (YYIRT). Prior to attending any of the physical performance tests, subjects were requested to refrain from participating in

vigorous physical activity within the preceding 24-hours or consuming caffeine within the preceding six hours. In addition, subjects completed a 24-hour diet recall form (Appendix 4) during session 1. Subjects were asked to replicate this diet as close as practicable prior to each of the subsequent physical performance testing sessions.

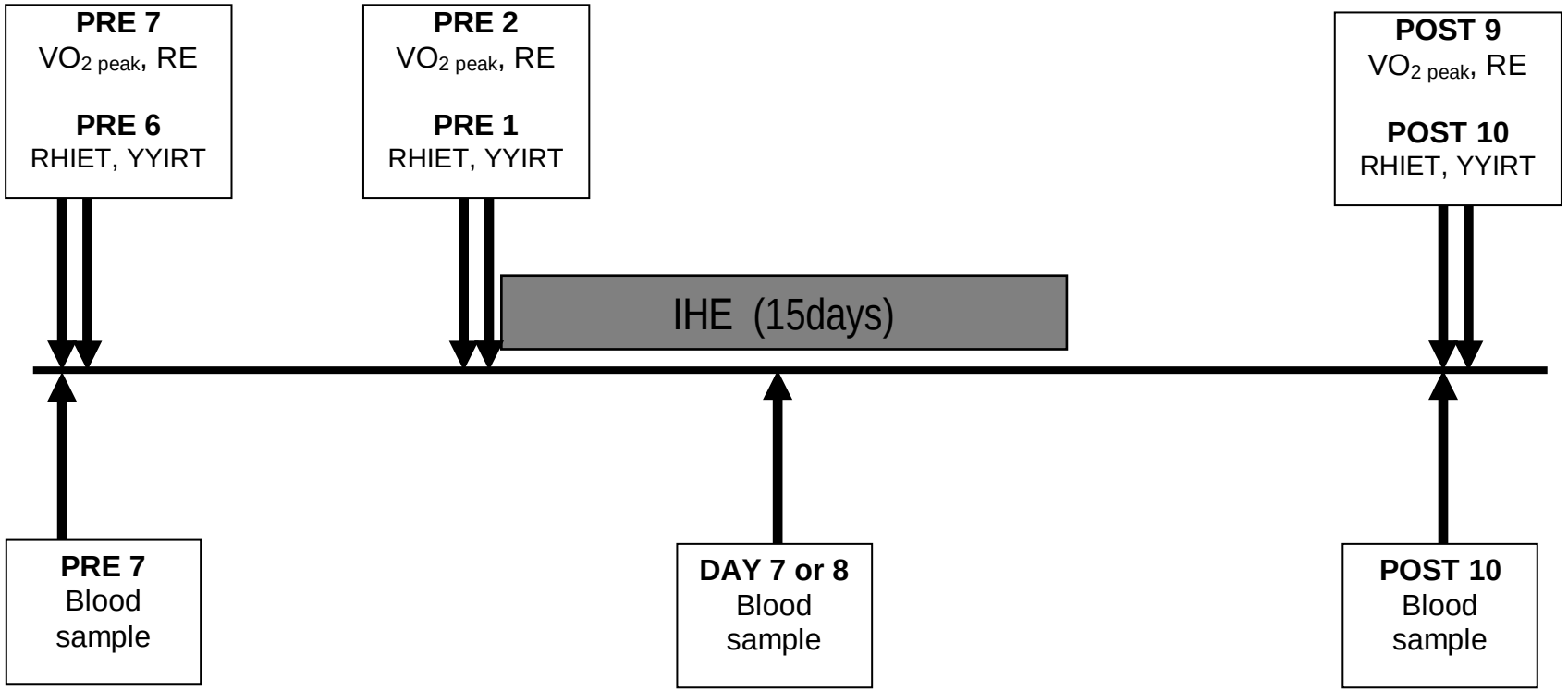

Figure 7. Schematic of study intervention, including performance, physiological and haematological testing times.

Aerobic capacity and running economy

Subjects performed an incremental running test in order to determine their running economy (RE) and $\dot{V}O_{2peak}$. The test was completed on a motorised treadmill (Powerjog, Birmingham, UK) in a temperature controlled (19-21°C) laboratory. Prior to the test, the subjects' height (Seca, Hamburg, Germany), body mass (Seca, Hamburg, Germany) and resting heart rate (RS400sd, Polar Electro, Kempele, Finland) was recorded. The protocol began with subjects straddling the treadmill for one minute during which resting physiological values (HR and respiratory exchange) were determined. Thereafter, subjects ran 3 x 3-minute stages at sub-maximal speeds (9, 11 and 13 $km.hr^{-1}$) at 1% gradient. After the third stage, the gradient was repeatedly increased at one minute intervals until the subject terminated the test due to volitional exhaustion. When the subject voluntary concluded the test, final performance (running speed and gradient) and physiological variables (HR_{peak}) were noted. Similarly, at the conclusion of the test, a small finger-prick blood sample was taken for analysis of blood lactate concentration (B[Lac]) using a portable lactate analyser (Lactate Pro, Arkray, Kyoto, Japan) that has been shown to be reliable and valid (Pyne, Boston, Martin, & Logan, 2000). Prior to collecting and analysing blood samples, the Lactate Pro was calibrated using a calibration strip provided by the manufacturer.

During the test, oxygen uptake ($\dot{V}O_2$) was measured on a breath-by-breath basis using a gas analysis system (MetaMax 3B, Cortex, Leipzig, Germany). Prior to each test, the gas analysis system was calibrated for volume with a 3L syringe

(Series 5530, Hans Rudolph, Inc., Kansas City, USA) and for gas concentration, via a two-point gas calibration using gases of known O_2 and CO_2 concentrations. The average (of all data points) $\dot{V}O_2$during the last 30 seconds of each 3 min stage of the incremental treadmill test was used to determine running economy at 9, 11, and 13km.hr^{-1}. Similarly, $\dot{V}O_{2peak}$ was measured as the highest measured oxygen consumption during any 30-second period. The $\dot{V}O_2$was presented in both absolute (L.min^{-1}) and relative (ml.kg^{-1}.min^{-1}) terms, and all presented in STPD.

Repeated sprint ability (RSA)

The repeated sprint ability (RSA) of the subjects was assessed using the RHIET. The RHIET was conducted at least 24 hours after the incremental treadmill-based test. The court-based tests were conducted on an indoor, inner-sprung basketball court. The RHIET is primarily used as an assessment tool of anaerobic fitness, whilst incorporating an agility component due to the multiple changes of direction. Prior to commencing the test, subjects conducted a 10-minute warm-up consisting of jogging, lower-body dynamic movement patterns (lunges, high-knees, butt kicks, grapevine) followed by some dynamic stretching and finally a short period of passive recovery before beginning the test. Subjects begin with their foot 0.5m behind the start line (A), as shown in Figure 8. Subjects were instructed that their first movement and step must be forward of the body. The test consists of a 60m sprint which is broken into a 5 metre shuttle (B), 10 metre shuttle (C), and a 15 metre shuttle (D) run consecutively at maximum speed. These distances were clearly marked out with cones. Subjects were required to complete six such

repetitions at 30-second intervals, all at maximum effort. At the completion of each repetition, subjects were given the remainder of the 30-second period as rest before commencing the next repetition. The rest period was always upright, and typically the period was taken by the time required to walk back to the start line to prepare for the next repetition. The time to complete each sprint was measured using electronic single beam timing lights (KMS, Fitness Technology, SA, Australia). The timing of each successive sprint was under the verbal instruction of the researcher, using a digital stopwatch (HS70W-1D, Casio, New Zealand). Two variables were calculated to determine RSA. Firstly, the total sprint time was calculated as the sum of the six sprint repetition durations. Secondly, the fatigue index was calculated to represent the extent to which the subject fatigued over the course of the six repetitions:

Fatigue Index (%) = ($T_{slowest} - T_{fastest}$)/$T_{fastest}$ x 100

Where $T_{slowest}$ = slowest time measured of the six repetitions, $T_{fastest}$ = fastest time measured of the six repetitions. A finger-prick blood sample was taken and analysed immediately after the test to measure B[Lac]. The HR_{peak} for the test was also recorded.

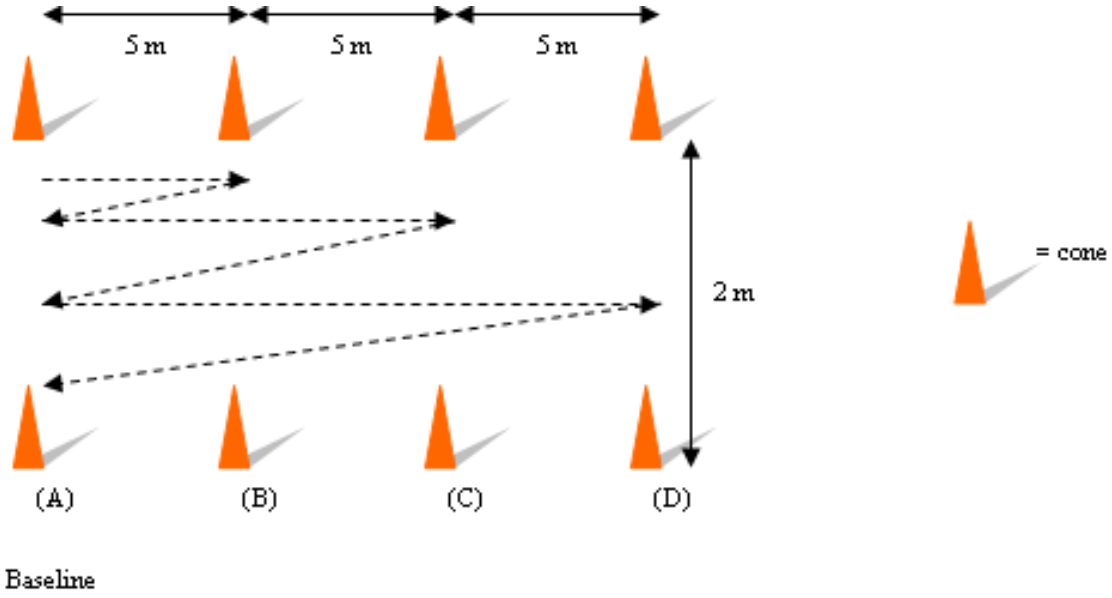

Figure 8. Layout of the repeated high-intensity endurance Test (RHIET)

Sport-specific aerobic fitness

The YYIRT, Level 1 (Krustrup et al., 2003) was used as a sport-specific measure of aerobic performance. Performance in the YYIRT has been shown to be correlated to basketball-match related performance decrements, thus providing a valid tool for measuring basketball-specific aerobic fitness (r = 0.52) (Castagna, Impellizzeri, Rampinini, D'Ottavio, & Manzi, 2008). The RHIET and YYIRT were conducted within the same testing session. To enable adequate energy system recovery, the RHIET was always performed first and a ~45 minute recovery period was prescribed prior to beginning the YYIRT. Because the subjects had already completed the RHIET and the fact the YYIRT provides a gradually increasing speed only an abbreviated warm-up was undertaken involving light jogging and dynamic stretching for 5 minutes. The YYIRT, as seen in Figure 9 consists of a shuttle run to and from a set of cones 20 metres away in time with fixed audible

cues from a stereo system. Interspersed between each 20 metre shuttle, is a 10-second active recovery period where subjects are required to jog/walk a distance of 2 x 5 metres, before waiting to commence the next shuttle. Subjects continue to run in time with the audible cues until volitional exhaustion. The test begins with 4 running bouts at 10–13 $km.h^{-1}$ (0–160 m) and another 7 runs at 13.5–14 $km.h^{-1}$ (160–440 m), where after it continues with stepwise 0.5 $km.h^{-1}$ speed increments after every 8 running bouts (i.e. after 760, 1080, 1400, 1720 m, etc.) until exhaustion (Krustrup et al., 2003). As with the RHIET, B[Lac] and HR_{peak} measures were sampled and recorded immediately after the test. Running $speed_{peak}$ was calculated using the following formula:

Running $speed_{peak} = V_{complete} + ((SS_{complete}/SS_{total}) * V_{step.})$

Where $V_{complete}$ represents the velocity of the last fully completed stage; V_{step} represents the increase in velocity between stages; $SS_{complete}$ represents the number of shuttles completed in final stage; and SS_{total} = total number of shuttles in final stage.

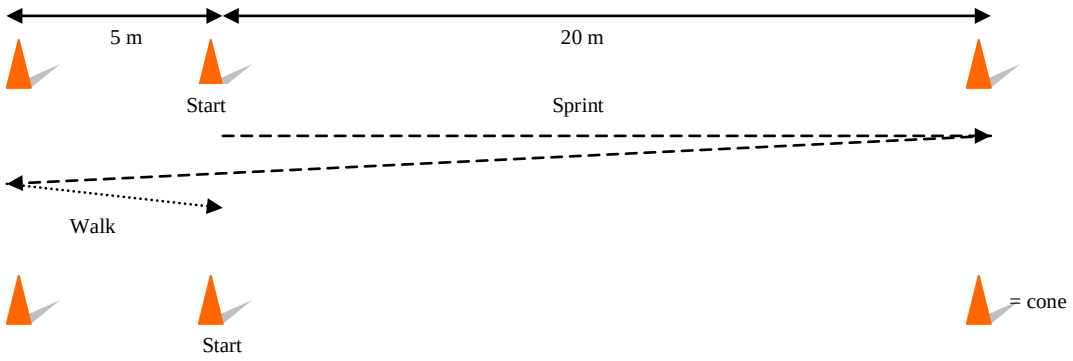

Figure 9. Layout of the Yo-Yo Intermittent Recovery Test (YYIRT)

Haematological measures

Subject were required to present themselves to a medical testing centre (Diagnostic MedLab, Auckland, NZ) on three occasions during the course of the study where a blood sample was taken via venepuncture from the forearm vein. The timing of these samples was: 6-7 days prior to the intervention (PRE), 7-8 days into the intervention (MID), and 10 days post-intervention (POST). The PRE and POST measures were taken to align with the timing of the physical performance tests (See Figure 7 for study overview). The haematological measures of interest were: haematocrit (Hct), haemoglobin concentration (Hb), ferritin, reticulocytes, white cell count (WBC), and soluble transferrin receptor (sTfR). Blood was drawn by a fully trained and qualified phlebotomist. Hct, HB, WBC, and reticulocytes were analysed using a Sysmex XE-2100 analyser (Sysmex America, Inc., Mundelein, United States). Ferritin was analysed using a Tina-quant Ferritin reagent kit. The sTfR samples were subsequently sent to a secondary laboratory (LabPlus, Auckland, NZ) for analysis, where samples were analysed on a Roche Hitachi Modular using immunoturbidometric techniques.

Hypoxic exposure

Hypoxia was generated by a High Tech Mixing Head (HTMH) which uses nitrogen dilution to provide a hypoxic gas (Airo Ltd, Auckland, NZ). The hypoxic unit is fully automated and provides a specified hypoxic stimulus (within a 3% range of SpO_2%) during the hypoxic period with no manipulation required from the subject or supervisor/researcher. Subjects were each provided a unit and relevant

consumables for use at their homes to facilitate compliance of the intervention. Subjects were requested to conduct the 37-minute session (HYP or CON) daily for 15 days consecutively. Subjects were omitted from the study if the 15 breathing sessions took more than 17 days i.e. if subjects missed more than two breathing sessions during the course of the intervention. Subjects were recommended to not conduct the session within 60 minutes of starting exercise or within 120 minutes of concluding exercise. Additionally, the unit was programmed to ensure a minimum of a 12-hour delay between breathing sessions. This provides an assurance that multiple sessions can not completed in a short period of time to catch up with the planned schedule. From a technical perspective, the subjects were required to fit the mask to cover their nose and mouth, and attach the pulse oximeter to their ear-lobe before pressing "start" to begin a single breathing session. Each 37-minute session consisted of seven minutes of hypoxia, with three minutes of normoxia, a process which was repeated four times. Each session has a target SpO_2 (within a 3% range) to meet during the seven minutes hypoxic stimulus, which was measured by the pulse oximeter on the subjects' ear-lobe. This measurement provided immediate biofeedback for the unit to alter the hypoxic stimulus as necessary. The target SpO_2% was progressively decreased over the course of the 15-day intervention, as shown in Table 6. During the 3-minute normoxic recovery phase, subjects still kept the mask on.

Table 6. Oxygen saturation upper and lower limits for each session of the breathing intervention

Breathing session	Upper limit (SpO_2%)	Lower limit (SpO_2%)
1	89	86
2	88	85
3	87	84
4	86	83
5	85	82
6	84	81
7	83	80
8	82	79
9	81	78
10	80	77
11	79	76
12	78	75
13	78	75
14	78	75
15	78	75

The data (HR and SpO_2%) from each individual hypoxic session was logged (at two-second intervals) within the unit for later downloading and analysis in MS Excel (Microsoft, Seattle, US). The actual hypoxic 'dose' was measured by calculating the amount of time subjects spent under 90% SpO_2. Any time spent lower than 90% SpO_2 was considered a hypoxic stimulus. It is unlikely that under normal physiological resting conditions that a subject would provide a SpO_2% below 90%. The formula for calculating the hypoxic dose is as follows:

Hypoxic dose = $\Sigma[90\text{-}SpO_2(2)]$

The formula provides the sum of the magnitude of data points (taken every two seconds) below an SpO_2 of 90%.

Training and diet

During the intervention subjects were provided with an iron supplement (18mg carbonyl iron, Douglas Pharmaceuticals Ltd, Auckland, New Zealand), which was taken to ensure each subject had sufficient physiological capability to produce red cell mass. Subjects took one tablet per day for the 15 days of the intervention. Each subject was also required to fill in a one-page questionnaire (Appendix 6) on a daily basis, in time with the daily hypoxic (or normoxic) session, which provided information on training, diet, energy levels, and sleep for the previous 24 hours. The training diary component of the questionnaire asked subjects to record the mode, duration (min), and intensity (using a rating of perceived exertion (RPE) scale of 1 to 10) of each bout of physical activity participated in. The "training load" – a product of the duration and intensity of training was calculated for each individual bout of exercise performed by the subjects. It is represented in arbitrary units (AU), and calculated as:

Training load = Duration (hours) x Intensity (1-10 scale)

As an example, if a subject listed a training session as a 90-minute run at an intensity of 6 out of 10, the training load would be calculated as:

Training load = 1.5 (hours) x 6 (RPE)

Training load = 9 A.U.'s

The training load for each subject was summed from each of the 15 days of the intervention to provide a total training load. This enabled a comparison to be made

between the HYP and CON groups. Previous research has shown this method to be well correlated to HR systems for measuring training load (Impellizzeri, Rampinini, Coutts, Sassi, & Marcora, 2004).

Training adherence

The adherence rate was calculated as the completed sessions divided by the number of actual days to complete.

Statistical analysis

Individual data points were entered manually into a MS Excel (Microsoft, Seattle, United States) spreadsheet previously set up by Hopkins (2006) for the purposes of comparing pre-and post-test variables. Means and standard deviations were calculated for each of the variables. A paired unequal-variances t-test was performed on the independent raw data variables. The level of statistical significance was set an alpha level of ≤ 0.05. Log-transformed data was analysed to deal with any systematic effect of an individual pre-test value on the change due to the intervention. The magnitude of log-transformed effects are expressed in percentages and Cohen units - the difference in the changes in the mean as a fraction or multiple of the pre-test between-subject standard deviation. The magnitude of the Cohen effect sizes was determined using the following scale: <0.2 is trivial, 0.2-0.5 is small, 0.6 - 1.1 is moderate, 1.2 - 1.9 is large, ≥ 2.0 is considered very large (Hopkins, 2002). Estimates of uncertainty were set at 90%

confidence limits for all effects. To determine if the changes were of clinical significance, 0.2 of the baseline between-subject standard deviation was selected as substantial. Correlation coefficients were determined using the following scale: <0.1 is trivial, 0.1-0.3 is small, 0.3 - 0.5 is moderate, 0.5 - 0.7 is large, 0.7 – 0.9 is very large, ≥ 0.9 is considered nearly perfect (Hopkins, 2002). The value of each Cohen effect size was described qualitatively, which represents the effect size as a probability of the effect being worthwhile.

Chapter Four: Results

Twenty-four subjects began the study, however, 10 subjects did not complete the study. Three subjects were injured and three subjects suffered from illness preventing them from continuing with their usual physical (and/or hypoxic) training schedule. Additionally, four subjects were omitted due to poor adherence (<88%, > 17 days completion) in completing the hypoxic sessions. Results from subjects that did not satisfactorily complete the study were omitted from analysis. Consequently, the results section reports the data for the 14 subjects (HYP = 7, CON = 7) that satisfactorily completed the study. Because of the high drop-out rate, it took longer than anticipated to collect sufficient data for analysis. Despite this, all subjects performed the intervention during a competitive season, albeit not the same competition season.

Subject characteristics

Table 7 shows the anthropometric characteristics, playing level, baseline physical performance (and associated physiological measures), and baseline haematological measures of subjects (n=14).

Table 7. Subject characteristics and baseline measurements

	HYP (Mean ± SD) n=7	CON (Mean ± SD) n=7
Characteristics		
Age (yrs)	23.3 ± 5.6	21.2 ± 3.3
Height (m)	1.87 ± 0.1	1.82 ± 0.13
Weight (kg)	91.4 ± 17.3	80.1 ± 15.0
Playing Level		
National Basketball League	3	1
Conference Basketball League	4	6
Incremental Treadmill Test		
VO_{2peak} ($ml.kg^{-1}.min^{-1}$)	54.3 ± 4.4	49.7 ± 4.8
Running Economy, 9$km.hr^{-1}$ ($ml.kg^{-1}.min^{-1}$)	36.2 ± 4.8	33.2 ± 6.2
Running Economy, 11 $km.hr^{-1}$ ($ml.kg^{-1}.min^{-1}$)	42.2 ± 5.4	39.1 ± 5.1
Running Economy, 13 $km.hr^{-1}$ ($ml.kg^{-1}.min^{-1}$)	47.5 ± 4.9	47.8 ± 4.1
RHIET		
Total Sprint Time (s)	93.1 ± 7.7	95.6 ± 7.9
Fastest Sprint (s)	14.3 ± 1.0	14.20 ± 1.5
Slowest Sprint (s)	16.1 ± 1.1	17.13 ± 1.9
Fatigue Index (%)	12.6 ± 4.4	22.5 ± 14.3
Peak Lactate (mM)	9.9 ± 1.8	11.64 ± 3.0
YYIRT		
Distance run (m)	1225 ± 470	1114 ± 370
Running $speed_{peak}$ ($km.hr^{-1}$))	15.2 ± 0.7	15.1 ± 0.6
Peak Lactate (mM)	10.7 ± 1.3	10.7 ± 1.8
Haematological characteristics		
Ferrtin ($\mu g.L^{-1}$)	147.3 ± 67.5	103.0 ± 34.0
Haemoglobin ($g.L^{-1}$)	143.0 ± 5.5	144.6 ± 10.6
Reticulocytes (units. nL^{-1})	39.2 ± 11.6	38.9 ± 13.4
Soluble Transferrin Receptor ($mg.L^{-1}$)	2.74 ± 0.75	3.06 ± 0.67

Hypoxic (and normoxic) exposure

Figure 10 shows a typical HR and SpO_2% trace of a single hypoxic session. The shaded areas, which represent the “ON” stages, clearly show a reduction in SpO_2% from resting levels, followed by a sharp recovery during the “OFF” stage

(unshaded areas). Additionally, the relationship between HR and SpO_2% should be noted. The trace shows HR increasing as SpO_2% decreases during the “ON” stages. Figure 11 shows the typical HR and SpO_2% trace from a CON group subject, where subjects were receiving normoxic gas for the duration of the session. The trace shows very little change in the SpO_2% during the 37-minute session, and does not go below ~98% at any time. The trace also shows the HR is relatively constant throughout the session. Whilst the HR range is 10-15 beats.min^{-1} over the course of the session, the general trend is relatively flat and constant.

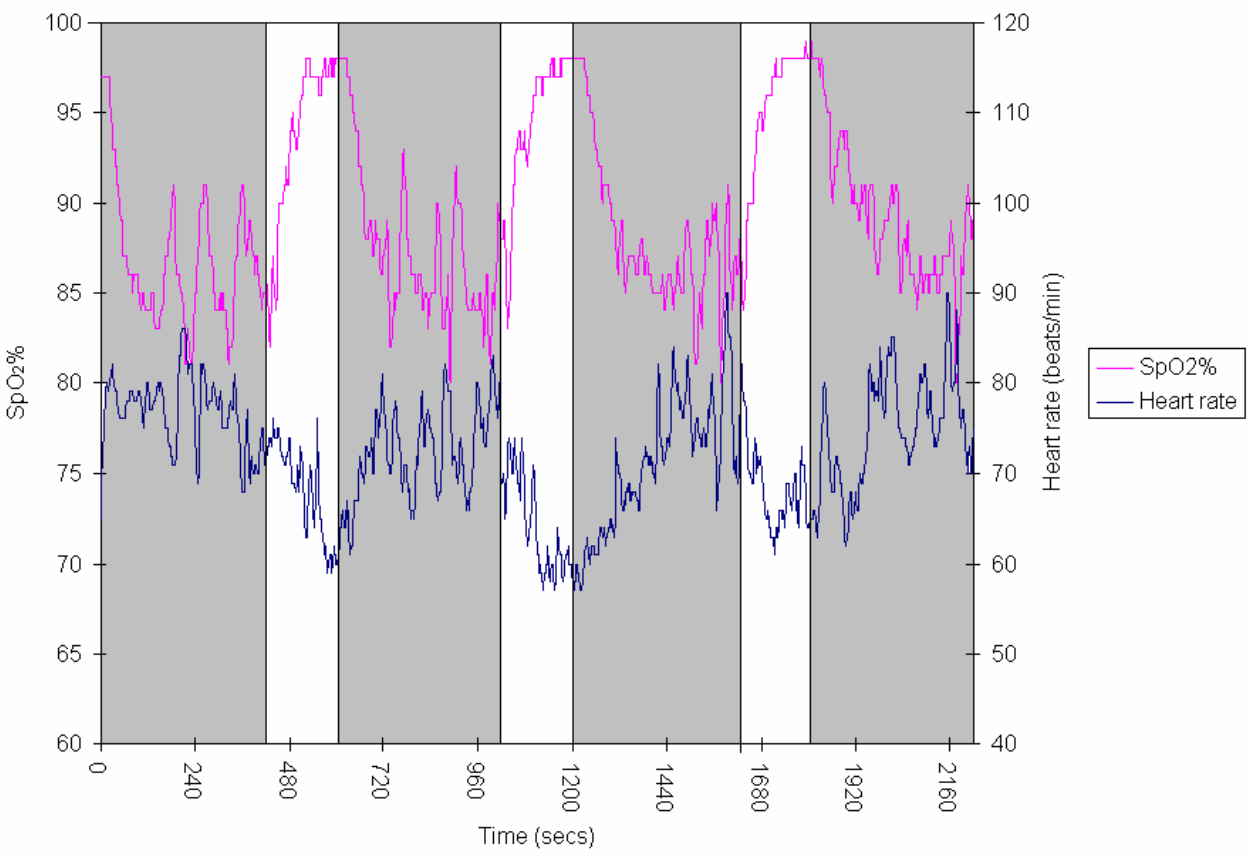

Figure 10. A typical trace showing the heart rate and SpO_2% during a hypoxic session. The grey shaded areas represent the seven-minute "ON" stages, and the un-shaded area represents the three-minute "OFF" stages

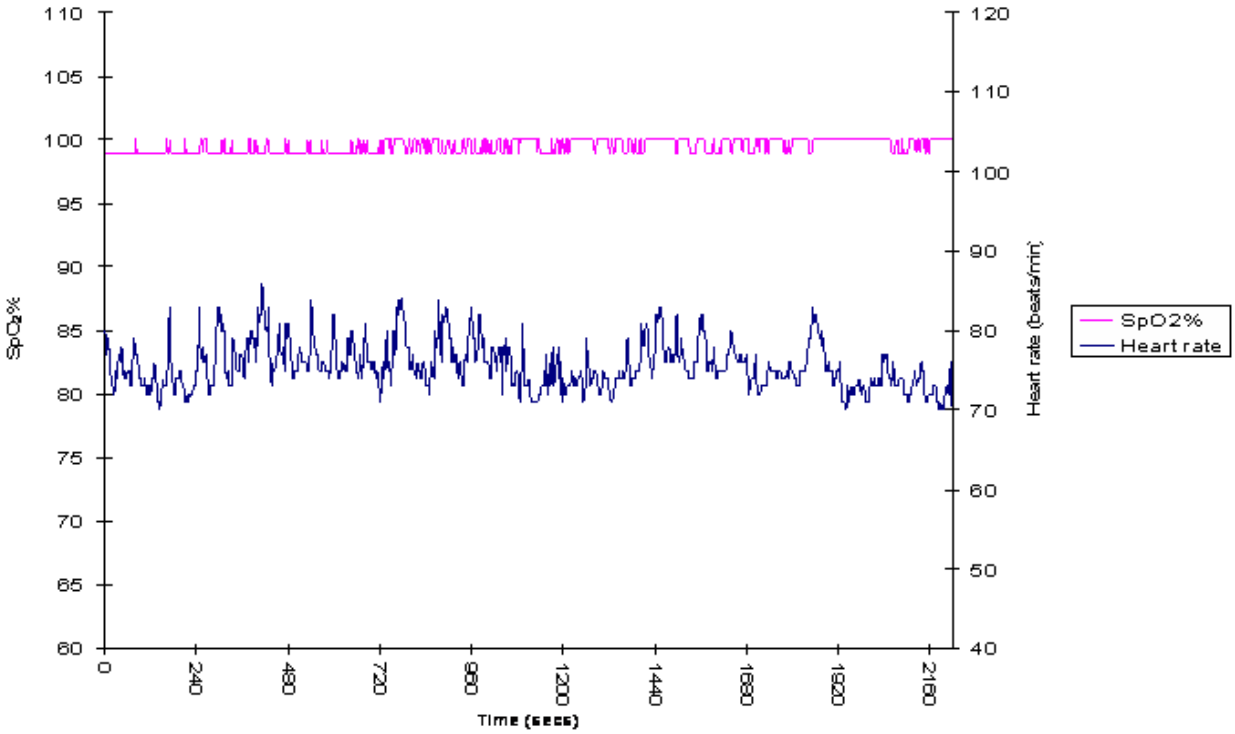

Figure 11. A typical trace showing the heart rate and SpO_2% during a normoxic session (from the CON group)

Hypoxic dose

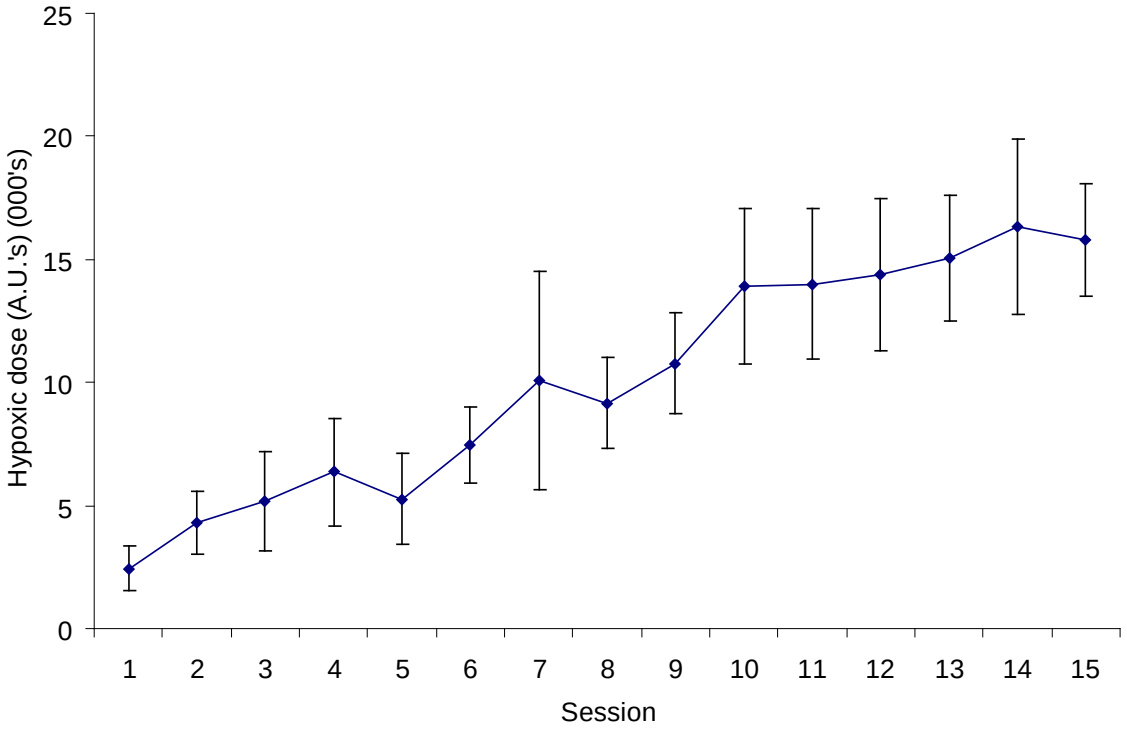

Figure 12. Hypoxic dose received by the HYP group subjects over the 15 sessions
The stimulus is calculated as the product of the intensity (SpO_2%) and duration (sec) the subject spent below a SpO_2 of 90%.

Table 6 details the prescribed hypoxic stimulus each HYP (mean ± SD) subject was exposed to. During session 1, the target SpO_2 was 86-89% during the "ON" stages, and by session 12 (and the following three sessions) the prescribed target was 75-78%. Figure 12 shows the actual mean hypoxic dose (for the HYP group), as measured by pulse oximetery, that subjects received. It shows the increasing nature of the dose being received over the course of the 15 sessions. The total mean hypoxic dose received by the HYP group was 150,500 ± 24,577 A.U.'s. As anticipated, all subjects in the CON group produced a hypoxic dose of zero for all sessions. In Figures 13 and 14, the sum of the shaded areas encompassed by the inserted line and the subjects' individual trace represents the hypoxic dose. Figure

13 represents a typical session 1, and Figure 14 represents a typical session 15. The change to the size of the shaded areas in the two figures shows the increases to the hypoxic intensity and resultant hypoxic dose over the duration of the 15-session intervention.

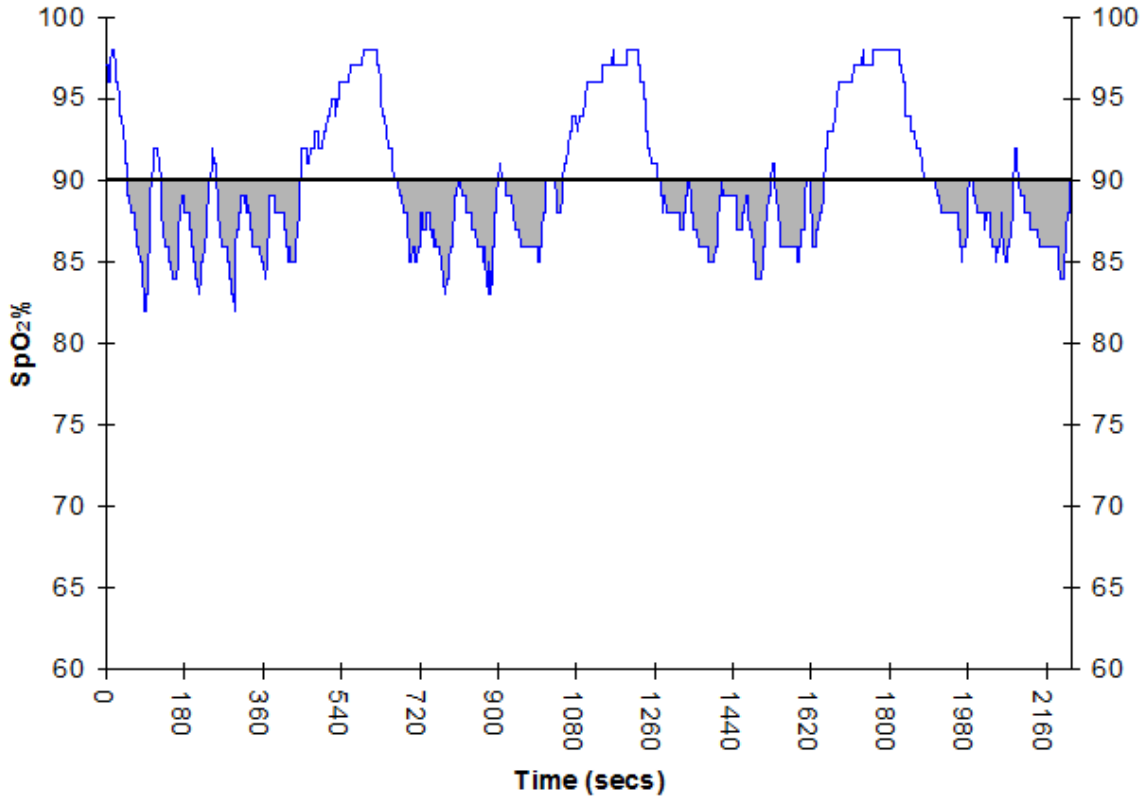

Figure 13. Typical trace of SpO_2% from session 1 (the first session of the 15-day intervention), and the graphical representation of the hypoxic dose.
The shaded area under the inserted 90% line represents the hypoxic dose of the stimulus. Hypoxic dose = 4,198 A.U.'s

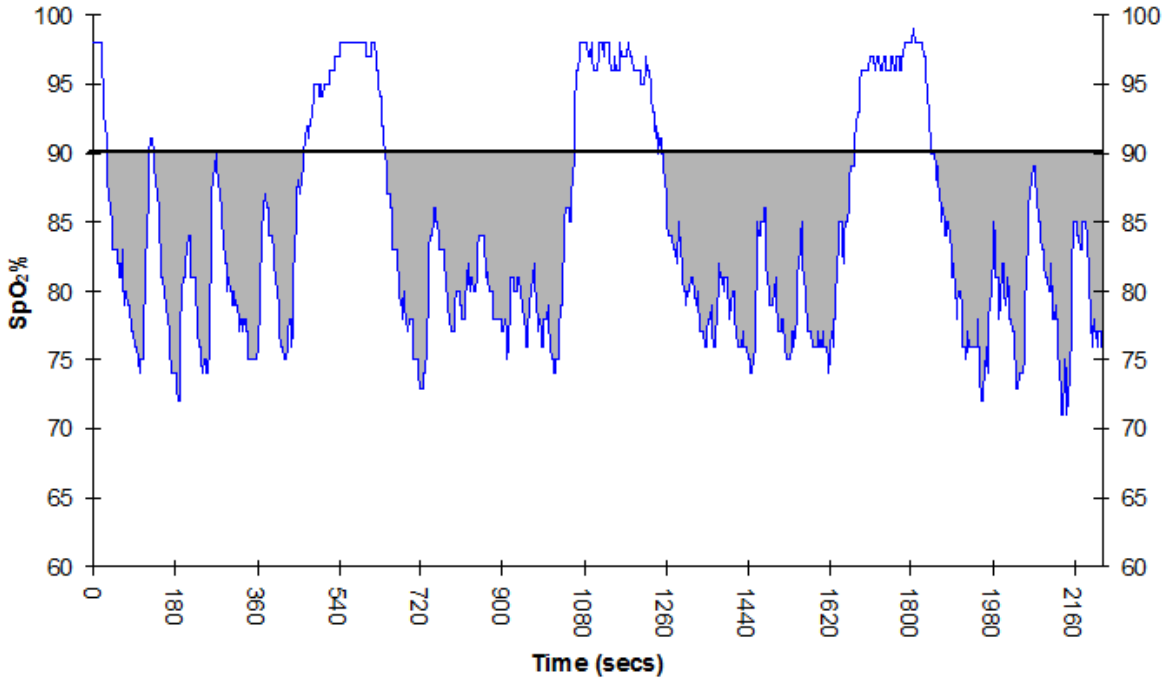

Figure 14. Typical trace of SpO_2% from session 15 (the final session of the 15-day intervention), and the graphical representation of the hypoxic dose.
The shaded area under the inserted 90% line represents the hypoxic dose of the stimulus. Hypoxic dose = 16,464 A.U.'s

Physical performance tests and associated physiological measures

Table 8 shows the mean (± SD) changes in physical performance of the relevant performance variables in YYIRT and RHIET. At 10 days post-exposure, the HYP group showed substantial performance enhancement in both tests, in comparison to the CON group. Performance in the RHIET, showed substantial improvements for the fastest sprint (-1.2; ± 2.3% (±90% CL); benefit possibly -ve), slowest sprint (-5.0; ± 2.4% (±90% CL); benefit very likely –ve; ES: -0.5 ± 0.2), total sprint time (-3.5; ± 1.6% (±90% CL); benefit very likely –ve; ES: -0.4 ± 0.2), but not fatigue index (-24.4; ± 47.2% (±90% CL); unclear). Performance in the YYIRT, as quantified by running $speed_{peak}$, was substantially improved (4.8; ± 1.6% (±90% CL); benefit almost certainly +ve; ES: 1.0 ± 0.4).

The post-RHIET $B[Lac]_{peak}$ showed a substantial increase (13.0; ± 13.8% (±90% CL); likely, probably +ve; ES: 0.5 ± 0.3), however, the post-YYIRT $B[Lac]_{peak}$ (19.9; ± 31.3% (±90% CL); unclear; ES: 1.2 ± 1.7) was relatively unchanged.

Table 8. Changes in physical performance measures between baseline and post-intervention tests

	Change in measure*				
	HYP (Mean ± SD)	CON (Mean ± SD)	Difference; ±90% CL	Effect Size†	Practical Inference‡
RHIET					
Fastest sprint	-2.1 ± 2.6	-1.0 ± 2.1	-1.2 ± 2.3	-0.1 ± 0.3	Benefit possibly –ve
Slowest sprint	-3.6 ± 2.2	1.4 ± 2.3	-5.0 ± 2.4	-0.5 ± 0.2	Benefit very likely –ve
Total sprint time	-3.4 ± 1.9	0.1 ± 1.2	-3.5 ± 1.6	-0.4 ± 0.2	Benefit very likely –ve
Fatigue Index	-12.3 ± 45.4	16.0 ± 24.9	-24.4 ± 47.2	-0.5 ± 0.7	Unclear
B[Lac]	4.8 ± 13.4	-7.2 ± 12.8	13.0 ± 13.8	0.5 ± 0.3	Likely, probably +ve
HR_{max}	1.9 ± 4.5	2.5 ± 3.5	-0.5 ± 4.1	-0.1 ± 0.7	Unclear
YYIRT					
Running $speed_{peak}$	3.9 ± 1.8	-0.8 ± 1.5	4.8 ± 1.6	1.0 ± 0.4	Benefit almost certainly +ve
B[Lac]	2.0 ± 26.8	-14.9 ± 36.3	19.9 ± 31.3	1.2 ± 1.7	Unclear
HR_{max}	-0.1 ± 3.1	0.7 ± 2.1	-0.8 ± 2.7	-0.1 ± 0.4	Unclear

*Units of change are percentage means ± SD for all measures

†Cohen ES±90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

‡Based on a smallest beneficial or harmful change in performance of 0.2 of the baseline between-subject standard deviation for each measure.

Physiological measures

Tables 8 and 9 show the mean percentage changes (± SD) in the physiological variables from the three physical performance tests. At 10 days post-exposure physiological changes in the HYP group were somewhat unremarkable. There was a probable enhancement in running economy at 11 (-9.0; ± 9.7% (±90% CL); likely, probably +ve; ES: -0.7 ± 0.7) and 13 km.hr^{-1} (-8.2; ± 6.9% (±90% CL); likely, probably +ve; ES: -0.7 ± 0.5), whilst economy at 9 km.hr^{-1} was unclear (-4.6; ± 12.4% (±90% CL)). This was not accompanied by any substantial changes to $\dot{V}O_{2peak}$ (-3.6; ± 6.3% (±90% CL); unclear).

Table 9. Changes in the incremental treadmill running test to exhaustion between the pre- and post-tests

	Change in measure*				
	HYP (Mean ± SD)	CON (Mean ± SD)	Difference; ±90% CL	Effect size†	Practical Inference‡
Incremental Treadmill Test					
VO_{2peak}	-1.3 ± 5.4	2.4 ± 5.5	-3.6 ± 6.3	-0.4 ± 0.6	Unclear
Oxygen cost, 9km/hr	0.4 ± 11.4	5.3 ± 11.1	-4.6 ± 12.4	-0.3 ± 0.7	Unclear
Oxygen cost, 11km/hr	-3.3 ± 8.9	6.3 ± 9.9	-9.0 ± 9.7	-0.7 ± 0.7	Likely, probably +ve
Oxygen cost, 13km/hr	-3.8± 7.7	4.8 ± 4.1	-8.2 ± 6.9	-0.7 ± 0.5	Likely, probably +ve

*Units of change are percentage means ± SD for all measures
†Cohen ES±90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
‡Based on a smallest beneficial or harmful change in performance of 0.2 of the baseline between-subject standard deviation for each measure.

Haematological variables

Table 10 shows all haematological measures. All changes to the haematological measures were unclear at the mid- and post-intervention samples, when comparing them to the pre-intervention sample, with the exception of the sTfR, post-intervention which increased (9.2; ± 10.1% (±90% CL); benefit possible; ES: 0.3 ± 0.3).

Table 10. Change in haematological measures measure between baseline, mid-intervention, and post-intervention

		Change in measure*				
	Sampling point	HYP (Mean ± SD)	CON (Mean ± SD)	Difference; ±90% CL	Effect Size†	Practical Inference‡
Ferritin	Mid	1.8 ± 33.5	-1.5 ± 12.9	3.3 ± 27.5	0.1 ± 0.5	Unclear
	Post	-6.4 ± 32.3	7.1 ± 9.7	-12.6 ± 25.6	-0.3 ± 0.5	Unclear
Haemoglobin	Mid	-1.5 ± 4.9	0.2 ± 4.5	-1.7 ± 4.7	-0.4 ± 1.0	Unclear
	Post	2.4 ± 2.5	1.1 ± 5.0	1.3 ± 4.0	0.3 ± 0.8	Unclear
Reticulocytes	Mid	-5.5 ± 30.3	4.5 ± 17.5	-9.6 ± 27.3	-0.3 ± 0.8	Unclear
	Post	7.0 ±6.9	9.2 ± 18.8	-2.0 ± 14.7	-0.1 ± 0.4	Unclear
Soluble Transferrin Receptor	Mid	2.5 ± 7.2	5.6 ± 12.4	-3.0 ± 10.4	-0.1 ± 0.3	Unclear
	Post	12.1 ± 8.1	2.7 ± 11.6	9.2 ± 10.1	0.3 ± 0.3	Benefit possible

*Units of change are percentage means ± SD for all measures; †Cohen ES±90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference; ‡Based on a smallest beneficial or harmful change in performance of 1%; Mid = Day 8 of the 15-day intervention; Post = 10-days post-intervention.

Relationship between hypoxic dose and the change in physical performance, physiological and haematological variables

Tables 11 to 13 show the relationship between the hypoxic dose received for HYP and CON subjects and the percentage change in; physical performance (Table 11), physiological variables (Table 12), and haematological variables (Table 13).

Table 11. Relationships between hypoxic dose and the magnitude of change in physical performance tests

Variable	r	p
RHIET		
Fastest sprint	-0.11	0.808
Slowest sprint	-0.12	0.794
Total sprint time	-0.28	0.542
Fatigue Index	-0.03	0.955
YYIRT		
Running $speed_{peak}$	-0.04	0.924

Table 12. Relationships between hypoxic dose and the magnitude of change in physiological variables

Variable	r	p
Incremental Treadmill Test		
VO_{2peak}	-0.16	0.733
Running Economy, 9km/hr	-0.50	0.314
Running Economy, 11km/hr	0.36	0.430
Running Economy, 13km/hr	-0.11	0.822

Table 13. Relationships between hypoxic dose and the magnitude of change in haematological variables

Variable	r	p
Ferritin	0.56	0.193
Haemoglobin	0.24	0.607
Reticulocytes	-0.36	0.431
Soluble Transferrin Receptor	0.23	0.625

Subject adherence rate

The mean adherence rate for the HYP group was 92.1± 0.95%, whilst the mean adherence rate for the CON group was 92.3 ± 0.90%.

Training load

The daily training load and total training load of the HYP and CON groups is shown in Figure 15 The total summed training load (mean ± SD) for the HYP and CON groups was 97.0 ± 21.7 and 100.4 ± 12.6 A.U.'s respectively

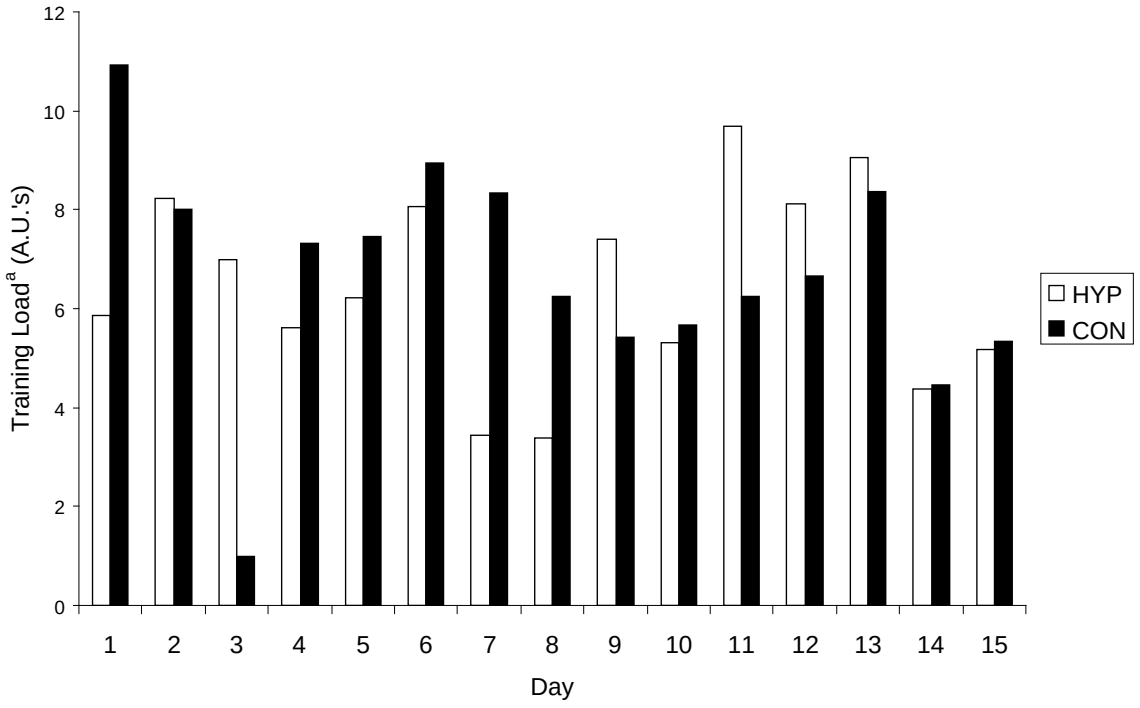

Figure 15. Daily (and total) training load of the HYP and CON group during the 15-day intervention

a = Training load = duration (hours) x intensity (1-10 RPE scale). Duration and intensity were taken from self-recorded training diaries; A.U.'s = Arbitrary units

Reliability of measures

Table 14. Reliability of all performance and physiological variables

	TE (% (90% CL))
Incremental Treadmill Test	
VO_{2peak}	5.7 (4.3-8.7)
Running Economy, 9km.hr^{-1}	6.5 (4.8-10.2)
Running Economy, 11 km.hr^{-1}	6.0 (4.5-9.3)
Running Economy, 13 km.hr^{-1}	3.6 (2.7-5.5)
RHIET	
Total Sprint Time	1.5 (1.2-2.3)
Fastest Sprint	1.2 (0.9-1.8)
Slowest Sprint	1.6 (1.2-2.4)
B[Lac]	12.3 (9.3-18.9)
YYIRT	
Running $speed_{peak}$	2.2 (1.7-3.3)
B[Lac]	8.8 (6.7-13.4)

Table 14 shows the reliability, typical error (TE) expressed as a percent coefficient of variation of the performance and physiological variables from the two baseline measures. The percent TE for parameters obtained from the incremental treadmill test show relatively poor reliability (3.6-5.7%), specifically VO_{2peak} and running economy. Similarly, the reliability of post-test B[Lac] measures were poor. The performance variables associated with the court-based tests (RHIET and YYIRT) displayed much better levels of reliability.

Chapter Five: Discussion

The aim of the current study was to investigate the effects of short duration IHE on physical performance in trained basketball players, and associated physiological and haematological variables. It was found that 15 days of short duration IHE provided sufficient stimulus to improve physical performance in some specific performance tests relevant to the demands of basketball. This enhancement in performance was accompanied by substantial changes in some physiological variables, but did not appear to be mediated by haematological changes.

Physical performance measures

Explosive/sprint performance

The current study did not dedicate a specific field test to measure explosive power or sprinting performance. However, the fastest sprint repetition time from the RHIET can be interpreted as maximal sprint performance as it is a measure typically lasting less than 16 seconds, and is performed in a non-fatigued state since it usually occurs in the first or second repetition of the test. The traditional physical performance mechanisms associated with altitude training (improvements in aerobic capacity via haematological adaptations) are not conducive to improving explosive/sprint performance. However, it is possible that IHE causes a shift towards a higher glycolytic contribution to ATP turnover, as has been indicted by previous research

(Katayama et al., 2004). Thus, events lasting longer than 2-3 seconds which are dependent on a contribution from glycolysis are likely to benefit from glycolytic-mediated enhancements. This is supported by the only other short duration IHE study (to the authors knowledge) that also used the fastest single trial from the RHIET as a sprint performance measure (Wood et al., 2006), also and reported a likely enhancement (-1.5; ± 1.7% (±90% CL)) at three days post-exposure, and possible enhancement (-0.9; ± 1.6% (±90% CL)) at 12 day post-exposure. In the current study, the HYP group subjects also produced a -1.2; ± 2.3% (±90% CL) change relative to the CON group comparing pre- vs post-IHE performance (this change represents an enhancement in performance as the unit of measurement is time). It is possible, based on the observed increase in $B[Lac]_{peak}$ (Table 8), that these improvements could be attributed to an increase in glycolytic turnover and/or a greater glycolytic contribution towards ATP production.

Repeated sprint ability (RSA)

In the present study, the RHIET was used to assess RSA because it is commonly used for basketball teams, and the subjects were familiar with it prior to the study. In addition, the reliability for RHIET parameters were all very good. The total sprint time, calculated as the sum of the sprint times of the six repetitions, improved in the HYP group (-3.5; ± 1.6% (±90% CL); ES: -0.4 ± 0.2), relative to the CON group. This represented a very likely improvement of performance using qualitative inferences. In agreement, Wood et al. (2006) reported similar findings in RHIET total sprint time at three

days post-IHE (-4.6; ± 1.3% (±90% CL)) and 12 days post-exposure: (-3.8; ± 1.1% (±90% CL)). Others, however, have reported no improvement in other forms of anaerobic performance trials after short duration IHE. For example, Tadibi and colleagues (2007) reported no change in the mean or peak power produced in the Wingate test in endurance-trained males after 15 IHE sessions in consecutive days. In addition, Bonetti and colleagues (2009) reported a possible negative effect on mean sprint power in cyclists and triathletes during repeated sprint performance (-2.0; ± 2.6% (±90% CL)) at three days post-exposure (15 IHE sessions over 19 days), although this became unclear at 14 days post-exposure.

There is evidence showing that the ability to reproduce bouts of high intensity exercise is correlated to aerobic capacity (Bishop et al., 2004). Thus an improvement in sprint ability, particularly for later sprints in a test involving multiple bouts such as the RHIET, maybe manifest themselves from improvements in aerobic fitness as a result of hypoxic exposure. Indeed, an improved RHIET performance in the current study supports this whereby HYP group subjects showed a -5.0; ± 2.4%, (±90% CL) change in slowest sprint performance relative to the CON group after IHE. This represented a 'very likely benefit' in performance (ES: -0.5 ± 0.2 (±90% CL). This finding is similar to Wood and colleagues (2006) who also reported positive RHIET performance enhancement in the HYP group (-6.1; ± 1.3% (±90% CL)), relative to CON group, at 12 days post-IHE in team sport athletes.

Sport-specific aerobic fitness

The YYIRT was used as the primary method of assessing the subjects sport-specific aerobic performance because in previous studies the YYIRT has been considered a valid basketball-specific test for the assessment of aerobic fitness (Castagna et al., 2008) and, importantly, changes in YYIRT performance have been related to basketball game performance (Castagna et al., 2008). In the present study, subjects produced baseline performances of 1225 ± 470 and 1114 ± 370 metres in the total distance run before exhaustion in the HYP and CON groups respectively. These values are lower than those previously reported (1678 ± 397m) in junior male basketball players (Castagna et al., 2008), but higher than those reported in male "recreational athletes" (1010 ± 419m) and male age-group representative cricket players (1049 ± 285m) (Thomas, Dawson, & Goodman, 2006) but expectedly less than those performances reported in elite female soccer players (1379m) (Krustrup et al., 2005), and semi-professional and elite rugby league players (1656 ± 403m and 1564 ± 415m respectively) (Atkins, 2006). This highlights that although the subjects were playing at a good level of competition (NBL and CBL basketball), their baseline level of sport-specific aerobic capacity was relatively low compared to other team sports. It is difficult to ascertain whether this sample was representative of the population of NBL and CBL players as no data exists to represent this population. Regardless, the relatively low level of aerobic capacity would lend support to the subjects having a larger range for improvement from short duration IHE, as suggested in a previous short duration IHE study (Hamlin & Hellemans, 2007). This is

supported by Bonetti & Hopkins (2009) recent meta-analysis that suggested use of short duration IHE for subelite athletic groups was worthwhile, whilst the benefits for elite athletes were less clear. Indeed, the HYP group in the present study displayed a substantially improved YYIRT performance (3.9; ± 1.8% (±90% CL), Table 8), compared to the CON group (-0.8; ± 1.5%, Table 8). This represents a 4.8 ± 1.6% (±90% CL) difference in performance change between groups and a very large ES (1.0 ± 0.4). This change represents the largest change in all of the physical performance measures in the current study. Unlike Castagna et al. (2008), we were unfortunately not in a position to associate our observed improvements in YYIRT performance with potential improvements in actual game performance.

Physiological measures

Aerobic capacity ($\dot{V}O_{2peak}$)

There is conflicting data surrounding the change in aerobic capacity after exposure to chronic and acute hypoxia. Likewise, for IHE, several studies have measured $\dot{V}O_{2peak}$ and reported no substantial change (Bonetti et al., 2006; Bonetti et al., 2009; Julian et al., 2004; Tadibi et al., 2007) after a short duration IHE intervention. Indeed a recent meta-analysis (Bonetti & Hopkins, 2009) showed the change to $\dot{V}O_{2peak}$ in athletes after short duration IHE was negligible (0.1; ± 2.8% (±90% CL)). In the present study, the pre- vs post-test changes in the HYP group were unclear (-3.6; ± 6.3% (±90% CL); ES: -0.4 ± 0.6), with a tendency for $\dot{V}O_{2peak}$ to be lower post-IHE intervention. In support, all previous short duration IHE studies that have reported $\dot{V}O_{2peak}$,

have not reported any improvements to $\dot{V}O_{2peak}$ as a result of the short duration IHE intervention (Bonetti et al., 2006; Bonetti et al., 2009; Julian et al., 2004; Tadibi et al., 2007), despite two of the studies (Bonetti et al., 2006; Bonetti et al., 2009) also reporting an enhancement in aerobic performance measures. This suggests that the improvements in performance were not mediated by improvements in $\dot{V}O_{2peak}$. There are studies using other methodologies of hypoxic exposure where improvements in performance have been reported without an accompanying change in $\dot{V}O_{2peak}$ (Ashenden, Gore, Dobson, & Hahn, 1999; Hahn et al., 2001b). The evidence regarding non-haematological mechanisms for enhancing aerobic performance have been well-reviewed (Gore et al., 2007). Gore and colleagues (2007) highlighted a number of plausible physiological mechanisms which may be involved with enhancing performance as a result of hypoxic exposure including molecular, economy and buffering capacity changes. It appears that the adaptations that result from hypoxic exposure, including short duration IHE, are not exclusively limited to the haematological cascade impacting on $\dot{V}O_{2peak.}$ It is more likely that multiple adaptations are responsible for the summed changes in physical performance seen in the current study. Further research is required to identify and isolate the mechanisms responsible.

Running economy

Improvements to movement economy at submaximal intensities has been one of the more recent theorised physiological changes after hypoxic exposure (Gore et al., 2007). Previous work (Green et al., 2000; Katayama et al., 2003;

Katayama et al., 2004; Saunders et al., 2003; Schmitt et al., 2006) has supported this change in economy, including research in short duration IHE (Babcock & Kirby, 2008). However, other short duration IHE research has not substantiated these findings, both in kayakers (Bonetti et al., 2006) and cyclists (Bonetti et al., 2009; Tadibi et al., 2007). In the present study, RE was substantially improved at 11km.hr^{-1} and 13km.hr^{-1}, but not at 9km.hr^{-1} (Table 9). The magnitude of changes observed (-4.6 to -9.0%, Table 9) are at the higher end of the range compared to previous research reporting improvement after short duration IHE (Bonetti & Hopkins, 2009). For comparison, Gore and colleagues (Gore et al., 2007) summarised the findings of seven different research groups, and nine separate studies (Gore & Aughey, 2001; Green et al., 2000; Hochachka et al., 1991; Katayama et al., 2003; Katayama et al., 2004; Marconi, Marzorati, Sciuto, Ferri, & Cerretelli, 2005; Neya, Enoki, Kumai, Sugoh, & Kawahara, 2007; Saunders, Telford et al., 2004; Schmitt et al., 2006) by detailing a reported change of 3-10% improvement in economy after a variety of hypoxic interventions. The only studies with reported economy changes greater than the current study were those from native high-altitude residents (Hochachka et al., 1991; Marconi et al., 2005). Identifying the mechanisms responsible for altering the possible changes in economy is beyond the scope and capability of the current performance-focussed study; however it is important to identify the mechanisms if athletic population wish to manipulate the mechanisms for further performance enhancement. Previous research has suggested such possible mechanisms. Logical mechanisms, as postulated by Gore and colleagues (Gore et al., 2007) include a decrease in work during ventilation

and improved substrate use. However no research exists to support these notions. In fact, research examining elite runners after 20 days of simulated moderate altitude (Saunders, Telford et al., 2004), has not supported the concept of changes in ventilation or substrate use being involved with changes in economy. The study found substantial improvements in RE (3.3% reduction in averaged $\dot{V}O_2$ across 14, 16, and 18 km.hr^{-1}) after 20 days of simulated LHTL, whilst ventilation, respiratory exchange ratio, and lactate concentration remained unchanged. A plausible alternative is a reduction in energy costs, in terms of ATP or oxygen, required for muscular contraction (Green et al., 2000). This is supported by one of two concepts that both have evidential support; a) an increase in the quantity of ATP produced per mole of oxygen used (Hochachka et al., 1991); and b) an absolute decrease in the quantity of ATP required for muscle contraction (Ponsot et al., 2006). However, further research is needed to determine if: 1) the improvements seen in movement economy seen after long duration hypoxic exposure, and in the current study, are able to be replicated in future short duration IHE studies; and 2) what are the mechanisms that mediate the changes? It is likely that the mechanisms for economy can be best identified by examining the cellular and molecular mechanisms mediating mitochondrial efficiency, which effect muscular efficiency. This area of research is likely to be fruitful for future hypoxia research.

The poor reliability associated with the aerobic capacity and running economy makes interpreting the true magnitude of change in physiological measures very difficult. It is possible that the true magnitude of the change is more

closely aligned with the substantial improvements seen in the YYIRT. This is supported by the fact that previous research using the same metabolic gas analysis system has also found unclear changes in aerobic capacity whilst subjects improved measures of physical performance (Bonetti et al., 2006; Bonetti et al., 2009).

Blood lactate measures

The current study did not measure the lactate threshold, however, post-test B[Lac] measures were taken, primarily as a means of identifying maximal effort. The $B[Lac]_{peak}$ measured after the RHIET was significantly higher post-intervention in the HYP group compared to CON group. However this was not replicated in $B[Lac]_{peak}$ measures from the YYIRT, where changes were unclear. The increase in $B[Lac]_{peak}$ measures post-RHIET was not consistent with previous short-duration IHE studies, which have shown either no change (Bonetti et al., 2006; Hinckson et al., 2006; Tadibi et al., 2007), or a decrease (Bonetti et al., 2009; Wood et al., 2006) in $B[Lac]_{peak}$ measures, which indicates a reduction in lactate production.

In contrast to Wood and colleagues (2006), the current study reported an increase in $B[Lac]_{peak}$ post-RHIET. This increase (13.0; ± 13.8% (±90% CL)), represented a practical inference of very likely benefit in the difference between HYP and CON group changes between pre- and post-tests. The performance enhancement seen in total RHIET sprint time, combined with the increase in $B[Lac]_{peak}$ suggests an increase muscle buffering capacity. Buffering capacity was not measured in the current study, or ever been

measured in previous short duration IHE research, however the mechanism has been postulated in past research using other means of hypoxic exposure; two weeks at 2,100 metres (Mizuno et al., 1990), and 23 nights in a live high (3,000 metres), train low (600 metres) environment (Gore et al., 2001) and reviewed previously (Gore et al., 2007). To strengthen the evidence, past RSA research has shown the positive relationship between an improved muscle buffering capacity and RSA (Bishop et al., 2004). In addition, increases in $B[Lac]_{peak}$ combined with RSA enhancement have previously been associated with an increase in muscle buffering capacity (Edge, Bishop, Hill-Haas, Dawson, & Goodman, 2006). However, the finding was not supported by the $B[Lac]_{peak}$ measures at the conclusion of the YYIRT. Whilst the difference between the HYP and CON group changes was large (19.9; ± 31.3% (±90% CL)), the variation between subjects makes a practical inference unclear. It is possible the variation surrounding $B[Lac]_{peak}$ readings, which are easily altered by diet, recent exercise, and effort, could be contributing to the lack of clarity in the results. It is possible that the increase in $B[Lac]_{peak}$ is a result of increased glycolysis. This would lend support to glycolysis-mediated improvements in fastest RHIET sprint time.

Haematological measures

Previous short duration IHE research has provided contrasting evidence with regards to haematological changes. The current study suggests that the performance changes were not mediated by haematological changes since there was no clear changes were observed in ferritin, reticulocytes, and Hb. There was a possible benefit (9.2; ± 10.1% (±90% CL)) in sTfR at 10 days

post-exposure but confidence limits were wide. Of the eight known short duration IHE published studies that have measured and reported haematological variables, three have reported no significant changes in any haematological measure (Babcock & Kirby, 2008; Julian et al., 2004; Tadibi et al., 2007), including HB, Hct, EPO, RBC, WBC, sTfR, ferritin, and reticulocytes. The current study mostly supports these findings. The studies which have found the most profound changes in physical performance have typically also found at least some degree of beneficial change in haematological changes including; an increase in Hb (1.1; ± 1.9% (±90%CL)), Hct (2.0; ± 2.9% (±90%CL)), and WBC (13.0; ± 9.0% (±90%CL)) (Wood et al., 2006), decreased ferritin (-15.0; ± 16.0 (±90%CL)), and increased reticulocytes immediately post-IHE (14.0; ± 17.0% (±90%CL)) and 14-days post-IHE (40.0; ± 32.0% (±90%CL)) (Bonetti et al., 2009), increased Hb mid-intervention (4.0; ± 2.1 (±90%CL)) and at 3-days (3.6; ± 3.2% (±90%CL)), Hct mid-intervention (4.1; ± 2.5% (±90%CL), and decreased ferritin at 3-days (-19.0; ± 15.0% (±90%CL) (Bonetti et al., 2006). Table 5 summarises these haematological changes. These studies lend support to the haematologically-mediated performance enhancement theory and mechanisms.

Despite observing some substantial changes in physical performance in the present study, the gains do not appear linked to any substantial haematological changes. This lends support to non-haematologically-mediated performance enhancement, of which potential mechanisms of which have been reviewed recently by Gore and colleagues (2007). It has been well documented (Beall, 2000; Beall et al., 1998) that native residents to high-

altitude regions of the world, primarily Andeans and Tibetans, have significantly different haematological characteristics in comparison to each other; Andeans exhibit high Hb levels, in comparison to sea-level residents (Beall, 2000), whilst Tibetan residents exhibit relatively normal Hb characteristics, when compared to sea-level residents (Beall & Reichsman, 1984). As Gore and colleagues (2007) highlighted, this body of evidence certainly suggests that physical gains are not solely mediated by haematological changes for chronic adaptation at least. There is currently no evidence to suggest that these discrepancies in physiological adaptation to hypoxia are exclusive to chronic adaptations.

Hypoxic dose

The current study used an electronic automatic biofeedback mechanism. The most significant advantage of electronic nitrogen-dilution and oxygen filtration systems is that they typically incorporate an automatic biofeedback system that enables real-time monitoring and adjustment of the hypoxic stimulus within a relatively tight range for the duration of a session. The subjects SpO_2% was used to provide automatic biofeedback to the Airo unit, which responded by adjusting the absolute level of hypoxia being delivered, to ensure SpO_2% remained within the target range. This is not the first study to use this method, however most of the previous work (Bonetti et al., 2006; Bonetti et al., 2009; Wood et al., 2006) have used manual methods of adjusting hypoxic delivery which might not be as accurate or responsive. There is a somewhat clear relationship between those studies that have used some method of biofeedback during short duration IHE sessions and those

that have not, in terms of the physical performance changes. The exception to this relationship is a study of “mixed-ability” multisport athletes (Hamlin & Hellemans, 2007). Whilst there are many other variables involved in a short duration IHE intervention, and efficacy is rarely influenced by a single measure, studies using biofeedback (Bonetti et al., 2006; Bonetti et al., 2009; Wood et al., 2006) appear to have reported more practically beneficial enhancements in performance than equivalent studies that have not used biofeedback (Hinckson et al., 2007; Hinckson et al., 2006; Julian et al., 2004; Tadibi et al., 2007). Our physical performance changes using an automated biofeedback system further support this trend.

It would be unwise to suggest that biofeedback is the sole predictor of efficacy in short duration IHE research, however it does appear to have some influence, and this appears logical if other physiological models are considered. For example, if we compare this technique to a pharmokinetic model, where the dose being received by the patients is measured by the quantity of the drug being delivered; this is an absolute quantity, regardless of the patients physiological response to the drug. However, the physiological effect of the drug is likely to vary between patients depending on various physiological determinants (i.e. body mass, previous exposure to the drug, blood flow etc.). This is similar to using FIO_2 as the prescription tool for hypoxic delivery. It does not take into account the individual response to the stimulus. Alternatively, if the patients level of the drug is measured in real-time as the drug is being delivered, and we know what level of the drug is optimal for efficacy and safety, we can adjust (provide more or less) the

delivery of the drug dependent on how the patient responds. The stimulus being provided is individualised to the patient's unique physiological response. This is equivalent to using SpO_2% as a means of providing biofeedback during an IHE session. Future research using short duration IHE should consider incorporating automatic biofeedback as the primary means for determining the level of hypoxic delivery.

In addition to the system constantly adjusting the hypoxic stimulus, by monitoring SpO_2%, the method also enables the comparison between hypoxic protocols regardless of the hypoxic prescription method. It is presently very difficult to accurately compare the hypoxic stimulus between one study that has used FIO_2 for its hypoxic stimulus prescription and another that has used a manual target SpO_2%. Furthermore, because of the somewhat large individual variation to a specific FIO_2 between subjects and sessions, it is possible the stimulus being received is vastly different between subjects. The measuring and reporting of the actual hypoxic dose enables this to be taken into account. This is a significant step forward in being able to compare and contrast the changes between studies. In the present study, the total time spent below 90% SpO_2 during each IHE session was determined. A cut-off of 90% SpO_2 was selected in the present study for two reasons; a) there is no doubt that all time spent below this value is providing some element of stimulus (i.e. it can not be incorporating resting values in healthy humans at rest); and b) an existing altitude-simulation device company (BiomedTech, Australia) already uses 90% as their criteria for beneficial hypoxic stimulus. In reality selecting the cut-off for the stimulus is not as important (assuming the

measured hypoxic dose including resting SpO_2 values) as ensuring that future studies and work use the same cut-off. There is no research to suggest what this value should be. Regardless, our approach provided the opportunity to examine the actual physiological stimulus that was being received by each subject. Figure 12 shows the progressive increase in the hypoxic dose received in each of the 15 sessions. This is an expected outcome, as the hypoxic delivery is preset to provide an increasing hypoxic challenge, as outlined in Table 6.

Correlates between hypoxic dose and the change in physical performance, physiological and haematological variables

To the authors' knowledge, this is the first study to report the individual hypoxic dose received by the subjects, as measured by SpO_2%, which represents a substantial move forward in the reporting of actual hypoxic dosage for IHE research. Past work (Hamlin & Hellemans, 2007) has signalled that a dose-response relationship is likely, and the concept of a dose-response has been well reviewed regarding natural altitude training (Levine & Stray-Gundersen, 2006) . For the present study, the relationship between total individual hypoxic dose and the magnitude of change in physical performance, physiological, and haematological variables was determined (Tables 11 to 13). However, only moderate, non-significant, correlations were observed between hypoxic dose and arrange of measures including: running economy (9 $km.hr^{-1}$) ($r = -0.54$; $p= 0.206$), running economy (11 $km.hr^{-1}$) ($r = -0.36$; $p= 0.430$), ferritin ($r = -0.56$; $p = 0.193$), reticulocytes ($r = 0.36$; $p = 0.431$). For some measures, although non-significant, this

provides a small amount of support to the concept that the quantity of hypoxia received by each individual somewhat influences the ergogenic effects of short duration IHE. However, it also suggests that there may be other factors that influence the ergogenic effect in other performance tests, such as the RHIET total sprint time. It is possible that with a larger sample size, some of the above correlations may become clearer, if indeed they exist.

Training load

The physical training load of participants was calculated as a function of the duration and self-reported intensity of the exercise on a daily basis, and summed to represent a total training load throughout the intervention, as used in a previous short duration IHE study (Hamlin & Hellemans, 2007). There appeared to be no substantial differences in the total training load between the HYP and CON groups. Whilst no specific exercise prescription was provided to subjects during the short duration IHE intervention, subjects were requested to maintain their current training schedule. Therefore, the results suggest that any observed differences between groups as a result of the IHE intervention (i.e. physical performance) are unlikely to be caused by differences in physical training load during the intervention.

Limitations and de-limitations

The non-completion rate from the current study was unfortunately relatively high. Specifically, ten subjects were excluded for a number of reasons (injured = 3, illness = 3, poor adherence = 4). A large sample size is desirable to maintain the power of the study, so the drop out rate was disappointing. A

drop-out rate due to poor adherence was not unexpected. The time-demanding nature of the protocol made it difficult for some subjects to complete as prescribed. Thus, whilst the target was 15 sessions in 15 consecutive days, an allowance of two additional days was allowed to take this into account. Four subjects were excluded from final testing and analysis due to them not competing the 15 required sessions within the 17 days allowed. Whilst there is little published research suggesting the appropriate number of sessions to be completed consecutively, it was deemed that because of the small stimulus being provided during short duration IHE, in comparison to LHTH and LHTL methods, that the stimulus must be regular enough to ensure an appropriate adaptation and prevent any detraining effect. Further research examining the frequency and length (number of sessions) of short duration IHE protocols and the effect of rest days within the IHE intervention, would help determine if this is an issue.

To determine training load, it was intended to use a heart rate monitoring system to monitor all training sessions of all subjects during the 15-day intervention. However, due to technical issues with the system, this was not possible. Therefore, the training diaries, of which all subjects completed on a daily basis, were used to compare training load(s) of the two groups. Whilst a more thorough means of monitoring training load was preferred, past short duration IHE research has used this method (Hamlin & Hellemans, 2007). The use of RPE-based training diaries has previously been shown to be an effective method for monitoring training load (Impellizzeri et al., 2004).

Although the allocation of subjects to the HYP or CON group was blinded, it is possible for subjects to predict which group they may have been allocated by recognising some of the effects of the hypoxic exposure, thus introducing a placebo effect. For this reason subjects were asked at the completion of the study whether they thought they were in the HYP or CON group. All subjects (100%) believed they were in the HYP group. Unfamiliarity with hypoxia is likely to have led to CON group subjects being deceived to thinking they were receiving the hypoxic exposure. Regardless of the reason, this does negate any possible placebo effect.

Future directions

The current study was performed during the playing season. To the authors' knowledge, no research has been published examining the effect of any form of hypoxic exposure at different stages of a competitive season. By implementing short duration IHE interventions during competition phases, when athletes are more likely to be at their peak level of physical fitness, it increases the chance that any changes to performance (or physiology) is due to the intervention and not simply day-to-day physical training. Whilst this is logical from a research robustness perspective, it does not necessarily mean that in-season is the most beneficial timing for hypoxic exposure. Future research into the efficacy of short duration IHE at various stages of an athletic season, and how the IHE affects, or is affected by, training load changes in different training phases is warranted. Similarly, there is no published research examining the long-term use of short duration IHE. In fact very little

research exists regarding long-term use of other methods of hypoxia. This is most likely to be a result of the time required to conduct such research.

Several companies offer short duration IHE technology and use protocols which have very little evidence behind them. To date, only a single study has looked at comparing different IHE protocols (Bonetti et al., 2009). Further work to establish optimal duration and doses is required if the benefits of this technology are to be fully realised.

The advantages of an automatic biofeedback mechanism have been discussed in previous sections. It seems logical that future research should use such systems where possible, or at least manually adjust hypoxic intensity where needed. Similarly, individuals' physiological hypoxic response (SpO_2%) should be monitored and recorded, regardless of the altitude simulation technique being used, as a means of comparing the hypoxic dose between studies. Few previous studies examining the effect of short duration IHE on physical performance at sea-level have used this method.

There is also a need for closer examination of the most suitable candidates for short duration IHE. For example, an investigation into the use of short duration IHE during periods of enforced or recommended low aerobic or anaerobic training volume, could act as a substitute for this missing training to reduce the inevitable detraining effect seen in these periods. Such periods of enforced reduction in aerobic and anaerobic training volume are seen commonly in injured athletes, and often recommended for specific athletes during muscular hypertrophy phases. From an injured athlete or coach's

perspective the maintenance of fitness would certainly be a positive outcome in such a situation.

Conclusion

In conclusion, 15 days of short duration IHE can improve the physical performance of trained male basketball players. These changes in performance did not appear mediated by haematological changes or in combination with changes in $\dot{V}O_{2peak}$. However, there was a substantial change in running economy, and some indirect evidence of an improvement in muscle buffering capacity and a shift towards an increased glycolytic turnover. Whilst the current study is limited to postulating mechanistic hypotheses based on indirect evidence, it certainly provides support for the use of IHE as an ergogenic aid for sub-elite level team sport athletes. There is a need for further research using short duration IHE for sport performance enhancement.

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Appendicies

Appendix 1. Ethics approval form

M E M O R A N D U M
Auckland University of Technology Ethics Committee (AUTEC)

To:	Andrew Kilding
From:	**Madeline Banda** Executive Secretary, AUTEC
Date:	22 November 2007
Subject:	Ethics Application Number 07/176 **The effects of intermittent simulated-altitude training on physical performance in trained basketball players.**

Dear Andrew

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC) at their meeting on 8 October 2007 and that as the Executive Secretary of AUTEC I have approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTEC's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement at AUTEC's meeting on 10 December 2007.

Your ethics application is approved for a period of three years until 22 November 2010.

I advise that as part of the ethics approval process, you are required to submit to AUTEC the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2, which is available online through *http://www.aut.ac.nz/about/ethics*, including when necessary a request for extension of the approval one month prior to its expiry on 22 November 2010;
- A brief report on the status of the project using form EA3, which is available online through *http://www.aut.ac.nz/about/ethics*. This report is to be submitted either when the approval expires on 22 November 2010 or on completion of the project, whichever comes sooner;

It is also a condition of approval that AUTEC is notified of any adverse events or if the research does not commence and that AUTEC approval is sought for any alteration to the research, including any alteration of or addition to the participant documents involved.

You are reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTEC for approval before that change is implemented.

Please note that AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at charles.grinter@aut.ac.nz or by telephone on 921 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Madeline Banda
Executive Secretary
Auckland University of Technology Ethics Committee

Appendix 2. Participant information sheet

Participant Information Sheet

Date Information Sheet Produced:
24th September 2007

Project Title

The effects of intermittent simulated-altitude training on physical performance in trained basketball players

An Invitation

You are invited to take part in a research study which seeks to assess the changes in physical performance, primarily aerobic capacity and repeated sprint ability, in trained basketball players as a result of intermittent simulated-altitude training. This study is being undertaken as a part of the researchers Masters of Health Science qualification. Participation is completely voluntary and you may withdraw at any time.

What is the purpose of this research?

The aim of the project is to determine how effective intermittent simulated-altitude training is in improving physical function in trained basketball players. Such data has benefits to not only basketball players seeking to improve physical capability for their sport, but to many other high-intensity intermittent team sport athletes (i.e. hockey, netball, rugby union etc.).

How was I chosen for this invitation?

We are seeking trained male basketball players (playing either NBL or CBL basketball) in the 2007 or 2008 season.

What will happen in this research?

The study is five weeks in duration. During week one, you will be required to conduct three fitness tests (one laboratory-based (30min), and two court-based (40min)), measuring aerobic capacity and repeated sprint ability. After each test, a small finger prick will be made to collect a blood sample to determine if true maximum effort was achieved. Over the next 15 days participants will be separated into two groups: the Hypoxic group (HG) or the Control group (CG). You will not be made aware of which group you are allocated until the conclusion of the study. Both groups will be required to

participate in your usual basketball training (or games) at least four sessions/week. In this period both groups will be required to attend simulated-altitude breathing sessions, which involve the breathing of air through a face mask. The HG will be breathing a gas that simulates the oxygen content at altitude, whereas the CG will be breathing normal atmospheric air. For both groups, this will take 37min/day, for 15 days. During the following 10days both groups will be required to continue to participate in basketball trainings (or games). At the conclusion of this period the same laboratory and court-based tests conducted in week one will be conducted again, for means of comparison. In addition, a blood and saliva sample will be required to be taken four times; at the beginning of the study, after 7 days of altitude exposure and 10 days after the intervention. These fluid samples will be tested for various markers known to be associated with altitude exposure and aerobic capacity.

What are the discomforts and risks?

The risks involved are minimal. You may experience mild muscular discomfort during the fitness testing protocols, however these are not likely to be any greater than participating in a game of competitive basketball. There may be some discomfort associated with providing a blood sample. In addition, there is the possibility of slight discomfort during the simulated-altitude exposure, including light-headedness, nausea, and headaches.

How will these discomforts and risks be alleviated?

The AIRO unit responsible for providing the simulated-altitude exposure constantly measures the level of oxygen in your blood, and will prevent it from getting to unsafe levels. In addition, you can remove the face-mask at any time in the event you feel uncomfortable. All researchers involved with fitness testing and monitoring altitude exposure all have current first-aid certificates. Similarly, professionals taking blood samples are appropriately qualified in this discipline.

What are the benefits?

There may be numerous benefits to both you and to other athletes in high-intensity intermittent sports. Firstly, you will gain information relating to your current level of aerobic capacity ($V0_{2max}$). Study participants will receive a 15-day course of simulated-altitude exposure, which has been associated with improvements in aerobic capacity. Therefore, there is the possibility you could improve this aspect of physical performance during the study.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details satisfy the requirements of the law and the Corporation's regulations. All

participants that complete the study will be compensated for their time and travel with $100 petrol vouchers.

How will my privacy be protected?

Your confidentiality will be maintained throughout this study. No material, which could personally identify you, will be used in any reports on this study. Data collected in this study will be kept in a secure cabinet in a locked office and will be shredded on completion of the study. No material which could personally identify you will be used in any reports on this study.

What are the costs of participating in this research?

There are no costs to your participation in this study except your time. You will be required to attend a local medical laboratory (e.g. Diagnostic Medlab) to provide a blood sample five times during the course of the study. You will also be required to attend fitness testing on six separate occasions. You will be required to attend breathing sessions once/day for 15 days during weeks 2-3 (37min/day). In addition, you will be required to participate in basketball activities (trainings/scrimmages/games) four times per week.

What opportunity do I have to consider this invitation?

You will have a period of one week to make a decision on whether you wish to participate in this study.

How do I agree to participate in this research?

You will need to complete the attached consent form if you wish to participate in this study. Your participation is entirely voluntary. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason.

Will I receive feedback on the results of this research?

If you wish, at the completion of the study you willl be sent a copy of your results and a short summary of the results as a whole. No individuals will be identified in the summary results. The results of this study will also be submitted for publication in an academic journal and for presentation at a national / international conference. It is usual for there to be a substantial delay between the end of the data collection and publication or presentation in these scientific forums.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, Andrew Kilding, andrew.kilding@aut.ac.nz, 09 921 9999 x7056.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, *madeline.banda@aut.ac.nz* , 921 9999 ext 8044.

Whom do I contact for further information about this research?

Researcher Contact details:

Mr Bryan Dobson
School of Sport and Recreation
AUT University
Private Bag 92006
Auckland
09 921 9999 x 9747
09 921 9747 (Fax)
bryan.dobson@aut.ac.nz

Project Supervisor contact details:

Dr Andrew Kilding (PhD)
School of Sport and Recreation
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09 921 9960 (Fax)
andrew.kilding@aut.ac.nz

Approved by the Auckland University of Technology Ethics Committee on: AUTEC Reference number: 07/176

Appendix 3. Participant consent form

Consent Form

Project title: **The effect of intermittent simulated-altitude training on physical performance in trained basketball players**

Project Supervisor: ***Dr. Andrew Kilding***

Researcher: ***Mr. Bryan Dobson***

- ○ I have read and understood the information provided about this research project in the Information Sheet dated 24th September 2007.
- ○ I have had an opportunity to ask questions and to have them answered.
- ○ I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- ○ I am not suffering from heart disease, high blood pressure, any respiratory condition (mild asthma excluded), any illness or injury that impairs my physical performance, infections, or current musculoskeletal injuries that would prevent me from participating in this study
- ○ I agree to provide blood and saliva samples
- ○ I agree to take part in this research.
- ○ I wish to receive a copy of the report from the research (please tick one):

  Yes○ No○

Participant'signature :.....
…
Participant's
name:…….....……
Participant's Contact Details (if appropriate):
……..
……..
……..
……..
Date:

Approved by the Auckland University of Technology Ethics Committee on AUTEC Reference number

Note: The Participant should retain a copy of this form.

Appendix 4. 24-hour diet recall form

24 Hour Diet Recall

Athlete Name:…….
Date:…….

Please record all food and drink you have consumed since…….(24 hours from time of test).

Time Consumed	***Food Item***	***Serving Size (g or ml)***

Appendix 5. Medical questionnaire

Medical Questionnaire

Name: _____

Address: _____

Date of Birth: _____ Ph. No. _____

Emergency Contact: _____ Telephone No. _____

Doctors Name: _____ Telephone No. _____

Please indicate if you suffer from any of the following?

- asthma
- bronchitis
- emphysema
- heart disease
- high blood pressure
- epilepsy
- current infection of any sort (including a cold)
- iron deficiency or any other blood related conditions
- any other illness not mentioned above

Have you had a hospital admission or operation in the past 6 months? Yes / No (circle)
If yes, please give details: _____

_____

Are you currently on any medications? Yes / No If yes, please list:

_____

_____

Appendix 6. Daily subject questionnaire

Questionnaire ***Date:*** ***Athlete:***

TRAINING TODAY

Session 1 Type: e.g. Game, team training, Weights, run, swim

Time: am / pm Duration: min Intensity: /10
(1= very weak, 10= extremely strong)

Session 2 Type: *(if applicable)* e.g. Game, team training, Weights, run, swim

Time: am / pm Duration: min RPE: /10
(1= very weak, 10= extremely strong)

BREATHING SESSION

Time session conducted: am / pm

When did you last eat?: am / pm

How did you feel during the breathing session?
(e.g. light-headed, OK, fine, great, normal, nauseous, no change, tired etc.)

How did you feel after the breathing session?
(e.g. light-headed, OK, fine, great, normal, nauseous, no change, tired etc.)

Please list the foods you have consumed in the last 24 hours
(Include time consumed, food type, and approximate quantity)

Approxiatmately how many glasses of fluid did you consume in the last 24 hours?
(Do not include coffee, tea, energy drinks or alcohol in this total)

Have you consumed any coffee, tea, energy drinks or alcohol in the last 24 hours?
(If so, how many and what type?)

GENERAL

Have you been feeling energetic and lively in the past 24 hours, as you usually feel?

Strongly Disagree Neutral Agree Strongly

Disagree				Agree

Have you been feeling mentally or physically tired in the last 24 hours?

Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree

Please provide possible reasons for the above answers, where possible

_____

_____

Hours of sleep last night: ☐ hrs

Is this a normal amount for you? (Y/N) ☐