2006

Effects of hypoxia on exercise induced muscle damage

Trevor M. Farr
Edith Cowan University

2006

Effects of hypoxia on exercise induced muscle damage

Trevor M. Farr
Edith Cowan University

2006

Effects of hypoxia on exercise induced muscle damage

Trevor M. Farr
Edith Cowan University

Edith Cowan University

Copyright Warning

EFFECTS OF HYPOXIA ON EXERCISE INDUCED MUSCLE DAMAGE

by

Trevor M. Farr
Bachelor of Science with Honours

Master by Research

Faculty of Computing, Health and Science,
School of Exercise, Biomedical and Health Sciences

Date of Submission:

1st August, 2006

USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

ABSTRACT

FARR, T. Effects of hypoxia on exercise induced muscle damage.

Purpose: The present study investigated the hypothesis that maximal voluntary contractions (MVC) peak torque, VJ, muscle tenderness, and plasma creatine activity would be significantly less for the condition that subjects were exposed to hypoxic (H) condition for 4 hours after eccentric exercise compared with the normoxic (N) condition.

Methods: Methods: Sixteen male subjects performed 60 repetitions (6 sets x 10 reps) of maximal eccentric action of the knee extensors on their dominant leg (knee joint angle 0^{o} – 90^{o} at $9^{o\ s\text{-}1}$) to induce muscle damage. Subjects were then randomly assigned into hypoxic (H) (15.5% O_2) or normoxic (control, CON) conditions. Two hours after the eccentric EIMD protocol, subjects rested in H (4hr treatment of breathing a medical grade gas (15.5% O_2 and 84.63% N_2)) or N (4hr treatment of breathing ambient air) group. Maximal isometric strength (MVC), maximal isokinetic peak torque, muscle soreness, plasma creatine kinase (CK) activity and vertical jump (VJ) were measured 24, 48, 72, 96 & 120hr post-eccentric EIMD protocol. **Hypothesis:** The recovery of MVC, peak torque, VJ, muscle tenderness, and plasma creatine activity would be significantly less during the H versus CON conditions. **Results:** Each dependant variable was compared between the groups and times by a two way repeated measures ANOVA. Plasma CK and tenderness was not significantly different between the H and CON conditions. Although tenderness was greater in the H condition to that of the N condition at 48 & 72hr post-EIMD whilst soreness was significantly greater in the H condition compared to the N condition at 24hr post-EIMD ($p<0.05$). MVC at 35^{O} showed a significant difference between the H and N groups at 48hr ($p<0.05$). MVC at 55^{O} showed a significant difference ($p<0.05$) between H

and N groups percentage change at 72hr post-EIMD. MVC at 75^{O} showed no significant differences between the H and N group at any of the measurement times. Maximal isokinetic concentric strength at $60^{O}\cdot s^{-1}$, $120^{O}\cdot s^{-1}$ and $240^{O}\cdot s^{-1}$ was not significantly different between H and N groups although the N groups measured variables were consistently greater across time. Similarly VJ showed no significant difference between H and N groups. **Conclusion:** The result of the present investigation suggests that the effect of a 4hr H insult following eccentric EIMD did not retard muscle damage or affect recovery process. However, further investigation is required to provide conclusive evidence that muscle damage recovery is affected by H insult following exercise.

Key words: hypoxia, normoxia, normobaric, eccentric exercise, partial oxygen pressure (PO_2), creatine kinase (CK), muscle soreness, muscle tenderness.

DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

(i) *incorporate without acknowledgement any material previously submitted for a degree or diploma in any institution of higher education;*

(ii) *contain any material previously published or written by another person except where due reference is made in the text; or*

(iii) *contain any defamatory material.*

Candidate: Trevor M. Farr

Signed: ……

Date: ……

ACKNOWLEDGMENTS

I wish to acknowledge the contribution and assistance of Dr Fiona Naumann and Dr Robert Robergs in the preparation of my thesis. I extend my sincere appreciation to Mr Mike Newton, Dr Paul Sacco and Dr Ken Nosaka for their expert knowledge, advice, support and encouragement over a number of years. I would also like to acknowledge those subjects who participated in my research project and extend my sincere appreciation.

I wish to express my sincere thanks to my parents who gave me the impetuous to seek out my dreams. To my sisters I thank you for your support, encouragement and understanding, you have been my strength during difficult times.

TABLE OF CONTENTS

List of Tables

List of Figures

Appendices

CHAPTER ONE

INTRODUCTION

1.1 Background to study

Numerous studies (Ernsting, 1963; Ernsting, 1978; Ernsting & Sharp, 1978; Cable, 2003; Harding, & Mills, 1983; Robergs, Quintana, Parker & Frankel, 1998) have previously investigated hypoxia and the physiological effects experienced at high altitude. However, to date the effects of hypoxia on muscle damage recovery has not previously been investigated. Exercise, especially unaccustomed strenuous activity, most often, results in muscle damage and is characterized by soreness, tenderness and a reduction in normal levels of strength and power, on the day after the activity and may last for up to 48hr (Clarkson, Nosaka & Braun, 1992; Clarkson & Tremblay, 1988; Ebbling & Clarkson, 1989). Therefore, this study chose eccentric EIMD as the protocol to investigate the effect of H condition on muscle damage as an eccentric exercise bout consisting of 60 eccentric actions would produce muscle damage and characteristics similar to previous studies.

Elite athletes currently engage in post-game recovery methods, immediately following a game and possibly 24hr post-game. These methods include walking, jogging, stretching, massage, contrast immersion (hot/cold baths) and either/or pool activities such as swimming, walking/running, active stretching or a combination of methods (T. Farr, personal observation). However, the effects of a short bout of hypoxia, usually within 24hr post-game, whilst flying from one competition to the next competition has, to date, not been addressed by medical or fitness staff employed by athletes. Anecdotal evidence suggests exercise induced muscle damage (EIMD) recovery is delayed following short-term (4hr) air-travel. A group of elite athletes from Western Australia who travel by aircraft (2-

6hr) every second weekend during their competition period reported greater mental and physical fatigue and reduced training performances, following *4-48hr after high-intensity exercise. It is also hypothesised that those athletes who regularly travel by air for a period of not less than 4hr have a reduced competitive career compared to those who travel infrequently by air. (T. Farr, personal observation).

The recovery time following exercise induced muscle damage is possibly dependent on the oxygen (O_2) supply to the damaged tissues as the human body is extremely sensitive and vulnerable to the effects of hypoxia, which can result in the deterioration of bodily functions, organism necrosis and eventual death (Ernsting & Sharp, 1978, p. 45). Therefore, O_2 may be considered to be the most important component required for the maintenance of normal bodily functions and the repair of damaged tissue. Previous investigations by Robergs, Quintana, Parker & Frankel, 1998; Ernsting, 1963; Ernsting, 1978; Ernsting & Sharp, 1978; Cable, 2003, have presented information on the effects high altitude (hypoxia) has on the human physiology at high altitude however, these studies only assessed healthy individuals at various altitudes.

The potential benefits of post-game muscle damage recovery may be compromised by short term exposure to hypoxia, similar to that which is experienced during air-travel. Aircraft cabins are usually pressurized to a relative hypobaria of 560 mmHg and exposes travellers to a moderate hypoxia (15.4% O_2), equivalent to a reduction in atmospheric oxygen of approximately 5.5% (20.93% at sea level) (Harding & Mills, 1983). Reducing muscle damage recovery time increases the return to pre-game physical condition and a more rapid return to training and provides a more effective pre-game training session, which may result in improved performance. However, as stated, the effects of hypoxia on muscle damage recovery, has not been investigated previously.

1.2 Purpose of the study

The purpose of this investigation was to compare the effects of a 4hr normobaric hypoxia and normobaric ambient air insult on muscle damage recovery following a bout of eccentric exercise. The current study may clarify relationships between muscle strength, muscle function and normobaric hypoxia. The rationale for the 4hr intervention period was that anecdotal evidence suggests that hypoxic exposure, whilst traveling by aircraft, of less than 4hr has no physiological effect on muscle damage recovery or training performance.

1.3 Research Question

1.3.1 Does short term (4hr) exposure to normobaric hypoxia following eccentric exercise induced-muscle damage exacerbate recovery of muscle damage?

1.4 Hypotheses

1.4.1 Blood creatine kinase activity, muscle soreness and muscle tenderness would be greater in the hypoxic condition than the normoxic condition across time whilst the recovery of isometric muscular strength, peak torque, vertical jump height, would be significantly retarded after the hypoxic condition compared to the normoxic condition.

1.5 Abbreviations

CO_2 : *Carbon Dioxide*

EIMD: *Exercise Induced Muscle Damage*

FIo_2 : a *fractional concentration of oxygen in the inspired gas.*

ft : *feet; the measure of a distance*

H^+ : *Hydrogen ion*

H : *Hypobaric*

N : *Normobaric*

O_2 : *Molecular Oxygen*

Pco_2 : *Partial pressure of CO_2*

pH : *Potential Hydrogen; a scale representing the relative acidity or alkalinity of a solution. A value of 7.0 is neutral, below 7.0 is acid, and above 7.0 is alkaline. The numeric pH value indicates the relative concentration of hydrogen atoms in the solution compared to a standard solution (Mosby, 1990).*

PIo_2 : *inspired oxygen tension*

CHAPTER TWO

LITERATURE REVIEW

This literature review will focus on the body of published literature in the areas of hypoxia and the physiological effects of hypoxia. It then explores the oxygen therapies in association with comparison of the known association between treatment of severe trauma of skeletal muscle tissue and muscle damage recovery and the mechanisms that induce muscle damage.

2.1 Hypoxia

Hypoxia is the inadequate or reduced tension of O_2 in the arterial and capillary blood, whether in quantity or molecular concentration (Ernsting, 1963). Hypoxia is a phenomena characterized by cyanosis, tachycardia, hypertension, peripheral vasoconstriction, and mental confusion (Mosby, 1990, p. 598). Mild hypoxia may cause (hypoxic drive) low arterial oxygen pressure, which stimulates the peripheral chemoreceptors to increase heart and respiratory rates. If the amounts of oxygen are not adequate for aerobic cellular metabolism, energy is provided by less efficient anaerobic pathways that produce metabolites other than carbon dioxide (Mosby, 1990, p. 598). The human body is extremely sensitive and vulnerable to the effects of severe hypoxia, which can result in the deterioration of bodily functions, organism necrosis, and eventual death (Ernsting, & Sharp, 1988, p. 45). Hypobaric Hypoxia is a reduced molecular concentration of oxygen in the inspired air produced by a decrease in barrometric pressure (Ernsting & Sharp, 1988, p. 45).

Normobaric Hypoxia is the inspiration of 15.37% oxygen (medical grade gas) in an isolated chamber at sea level.

Living organisms obtain energy to maintain their biological functions by the oxidation of molecules of food substances, such as the conversion of glucose to water and carbon dioxide (Ernsting & Sharp, 1978, p. 45). The present investigation is concerned with hypoxic hypoxia and its physiological effect on muscle damage recovery, as a consequence of air-flight. Aircraft cabins are usually pressurized to a relative hypobaria (hyperbaric hypoxia) of 560 mmHg, which is equivalent to between 5,000 and 7,000 feet (ft). This hypobaria equates to a moderate hypoxia, equivalent to a reduction in atmospheric oxygen of 5.5% (20.93 to 15.4%).

2.2 Physiological Responses to Hypoxia

The energy required to maintain biological processes is obtained from the oxidation of complex chemical foods to simpler compounds whilst producing CO_2, water and other waste products. Oxygen is therefore possibly the most important component required for normal human function. The human physiological system is designed to function most efficiently and economically at sea level to maintain general requirements of the pulmonary and respiratory systems. In the event an individual is exposed to altitudes above sea level the physiological function of that individual may be altered.

Ernsting & Sharp (1978, p. 47) suggest that the increase in pulmonary ventilation that occurs as a result of exposure to altitudes above 8,000-10,000ft may be a result of two conflicting factors: the lowered arterial oxygen tension which stimulates ventilation and its effect on the chemoreceptors of the carotid and aortic bodies. However, the increase in ventilation is opposed by the respiratory depressant effect of the concomitant reduction in

carbon dioxide tension. The compromise between the two factors is the demand for an adequate oxygen supply versus the need to maintain a normal acid-base balance. During exposure to hypoxia, at rest, pulmonary ventilation increases at altitudes above sea level, induced by inspired air during air-travel, producing small insignificant increases in the total oxygen consumption, and in carbon dioxide production, of the tissues (Ernsting & Sharp, 1978, p. 48). This increase in metabolism is a consequence of the extra work associated with the rise in pulmonary ventilation and cardiac output (Ernsting & Sharp, 1978, p. 48).

In addition to the previous studies, Harding & Mills, (1983) investigated aviation pilot's performances of coordination tasks, and memory challenges following a number of tests. Features of hypobaric hypoxia, which occurs during ascent to altitude, include subtle personality change, euphoria, impaired judgement, short-term memory loss and mental in-coordination. Furthermore, Ren, Fatemian, & Robbins (2000) suggest the features associated with hypoxia vary between individuals, and the rate at which they develop depends on the severity of the hypoxic insult. These features reflect the ability of the cardiovascular and respiratory systems to respond to hypoxia. If the hypoxic environment were to vary in oxygen concentration between aircraft carriers an athlete may be at risk if they have a soft tissue injury as they may be more susceptible and sensitive to any change in oxygen quality or quantity as they are highly trained and exceptionally fit individuals. Similarly, it may be hypothesized that the athlete is more likely to be able to adjust accordingly to the environment as their cardiovascular system is highly trained to cope with any physical challenges with regard to oxygen availability and delivery to the physiological requirements. Therefore, it seems reasonable to investigate if there are any effects of hypoxia on muscle damage recovery in an attempt to answer unknown questions.

Hypoxia alters the hemoglobin affinity for oxygen. Increases in Pco_2 and H^+ decrease oxyhemoglobin saturation; a decrease in pH from 7.4 to 7.3 at 40 torr Po_2 decreases the oxyhemoglobin saturation by 6% and this is particularly significant in the capillaries of working muscles (Mosby, 1990, p.160). However, It is unsure if these effects will occur during a hypoxic insult of 5,000 to 8,000ft. If the exercise intensity was great enough to cause significant tissue damage, increases in Pco_2 and H^+ may cause hemoglobin affinity for oxygen to be further altered when exposed to a hypoxic condition.

Exposure to a hypoxic environment will cause a proportional increase in cardiac output, to that of heart rate (HR), although stroke volume remains constant (Ernsting & Sharp, 1978, p. 51). Although cardiac output increases, mean arterial pressure during moderate hypoxia is usually unchanged to that of breathing air at sea level. The systolic blood pressure usually increases with pulse pressure, although there is an overall reduction in peripheral resistance, a redistribution of blood flow by local and vasodilatation responses (Ernsting & Sharp, 1978, p. 52). An immediate increase in blood flow through the coronary and cerebral circulation, whilst the peripheral resistance in resting skeletal muscle is unchanged and renal blood flow is significantly reduced (Ernsting & Sharp, 1978, p. 52). The redistribution of cardiac output to essential organs, heart and brain, is increased at the expense of less acutely essential organs such as, viscera, skin and kidneys (Ernsting & Sharp, 1978, p. 52).

Jones, Sutton, Taylor, and Toews (1977) investigated the effects of pH on cardiorespiratory and metabolic responses to exercise and showed that sodium bicarbonate supplemented subjects maintained a power equivalent to 95% of maximal rate of oxygen consumption for twice as long as the control group. The ammonium chloride subjects showed half the endurance time at the same power output. However, a further study showed

that the effects were caused, in part, by changes in muscle glycolytic flux (Sutton, Jones & Toews, 1981). McCartney, Heigenhauser, and Jones (1983) employed an identical experimental approach to manipulate blood acid-base status to investigate the effects on maximal short-term (anaerobic) cycling. In contrast to the previous study, McCartney, Heigenhauser & Jones (1983) found that the reductions in blood pH were associated with only small reductions in the total work performed in 30s of maximal exercise. The authors concluded that the blood acid-base alterations have an insignificant effect on short-term maximal performance mediated by changes in glycolytic flux. However, any variation between the two investigations methodologies may reflect the subject(s) ability to maintain the oxidation of lactate within the muscle (or the muscle of the resting limb), or an increase in gluconeogenesis during exercise may indicate fatigue, or insufficient stores of CP within the muscle would reduce the ability to maintain maximum total work output over time during anaerobic exercise.

Ahlborg, Hagenfeldt, & Wahren, (1975), Sutton, Jones, & Toews, (1981) have shown that lactate uptake by muscle, liver and other tissues increases during prolonged exercise and following a period of recovery. However, Richardson, Noyszewski, Leigh, & Wagner, (1998) suggest it is controversial whether lactate formation during progressive dynamic exercise from sub-maximal to maximal effort is due to muscle hypoxia. The authors investigated muscle hypoxia using a direct measure of arterial and femoral venous lactate concentration, a thermodilution blood flow technique, phosphorus magnetic resonance spectroscopy (MRS), and myoglobin (Mb) saturation measured by ^{1}H nuclear MRS in six trained subjects performing single-leg quadriceps exercise. They calculated net lactate efflux from the muscle and intracellular P_{O_2} with subjects breathing ambient O_2 and 12% O_2. Mb saturation was significantly lower in H compared to N throughout incremental

exercise to maximal work rate. The authors found the net blood lactate efflux was unrelated to intracellular P_{O_2} across the range of incremental exercise to maximum but linearly related to O_2 consumption with a greater slope in 12% O_2. The net lactate efflux was also linearly related to intracellular pH. The authors concluded the increasing work rate, at a given fraction of inspired O_2, lactate efflux is unrelated to muscle cytoplasmic P_{O_2}, yet the efflux is higher in hypoxia and that catecholamine values from similar studies indicate that lactate efflux in hypoxia may be due to systemic rather than intracellular hypoxia.

Diminished oxygen delivery to the kidney as a result of hypoxia exposure, diminished red cell mass, or reduced renal blood flow results in increased erythroproietin (EPO) production (Fried & Morley, 1985). To determine the duration of continuous exposure to normobaric hypoxia required to increase EPO production, Knaupp et al (1992) measured plasma EPO levels in subjects exposed to acute normobaric hypoxia for 5, 60 and 120min and examined the EPO response to intermittent hypoxic exposure for 240min. The investigation showed no increase in EPO after 5 and 60min exposures. However, a 50% increase was seen 240min after the initiation of the 120min hypoxic exposure. Intermittent exposure resulted in an increase of EPO by 52% 360min after the onset of exposure. They concluded that exposing humans continuously to a hypoxic condition for 120min or intermittently for 240min provides a sufficient stimulus to increase EPO production. Consideration of subject plasma volume should be considered when evaluating these results, as individual sensitivity to decreases in plasma volume and exposure to hypoxia conditions and fitness levels may explain any disparity between the observed values.

However, it is possible that hypoxia exposure may result in a decrease in the oxygen content of arterial blood (P_aO_2). This decrease in P_aO_2 and the subsequent decrease in O_2 diffusion gradient between blood and tissue may be the primary effect of hypoxic insult. A

secondary effect of the hypoxic insult that may inhibit the treatment of soft tissue injury is the increased inflammatory response possibly due to a PO_2-mediated vasoconstriction. After damaged tissue fragments have been cleared neutrophils and their state of function may be reduced, macrophages are then recruited by chemotactic agents to mediate the processes of growth and repair which may be reduced due to the hypoxic insult.

2.3 Oxygen Therapy

Hyperbaric oxygen therapy (HBO) is an accepted treatment of crush, burn and trauma injuries and skeletal muscle compartment syndromes (Bouachour, Cronier, Gouello, Toulemonde, Talha, & Alquier, 1996; Cianci & Sato, 1994; Strauss, Hargens, Gershuni, Greenberg, Crenshaw, Hart, & Akeson, 1983; Strauss, Hargens, Gershuni, Hart, & Akeson, 1986). Garcia-Covarrubias, McSwain, Van Meter, & Bell (2005) suggest HBO therapy has been recommended as an adjunct treatment in acute traumatic ischemia and crush injury and concluded that adjunctive HBO is not likely to be harmful and could be beneficial if administered early. HBO therapy has also been advocated for the treatment of severe trauma of the limbs in association with surgery because of its effects on peripheral oxygen transport, muscular ischemic necrosis, compartment syndrome, and infection prevention (Bouachour, Cronier, Gouello, Toulemonde, Talha, & Alquier, 1996) .

Staples, Clement, Taunton, McKenzie (1999), Ishii, Deie, Adachi, Yasunaga, Sharman, Miyanaga, & Ochi (2005) suggested HBO has become a prescribed treatment for athletes suffering soft tissue injuries because of the oxygen transport effect. Specifically, Staples et al., (1999) suggested that intermittent exposures to HBO may provide the opportunity to reduce recovery from delayed-onset muscle soreness (DOMS). In support of the use of HBO as a treatment for muscle injury recovery, Ishii et al, (2005) suggested that

HBO treatments have been reported to reduce post-injury swelling and provides a positive result for tissue remodeling after injury.

Several investigations indicate a potential for HBO treatment for reducing recovery time. In HBO studies, greater increases in eccentric torque and decreases in fatigue and soft tissue injury recovery time have been demonstrated in HBO treatment groups compared to other treatments groups (Bouachour et al, 1996; Staples, Clement, Taunton, McKenzie, 1999; Ishii et al, 2005). HBO therapy was administered over a 5-day period in two phases; the first phase four groups (control, hyperbaric oxygen treatment, delayed treatment, and sham treatment) showed a significant difference between the treatment group and the other groups for eccentric torque recovery, in phase two three groups (3 days of treatment, 5 days of treatment, and sham treatment) showed eccentric torque recovery for the 5-day treatment group was significantly greater compared to the sham group from immediately after exercise to 96hr after exercise (Staples et al, 1999).

In support of HBO treatment, Ishii et al, (2005) suggest that HBO treatment assists muscle injury recovery as oxygen is crucial during recovery from injury and physiological fatigue. By performing HBO treatment, more oxygen is dissolved in the plasma of the pulmonary vein via the alveolar, increasing the oxygen reaching the peripheral tissues. Therefore, it is expected that HBO treatment may reduce recovery time from injury and fatigue. Similarly, Sueblinvong, Egtasaeng, & Sanguangrangsirikul, (2004) found that HBO treatment of 30 minute exposure to 2.5 ATA with 100% O_2-inhalation on lactate concentration following muscular fatigue from incremental exercise on a cycle ergometer resulted in a significant decrease of blood lactate concentration at 15, 20 and 25 minutes post exercise intervals in the hyperbaric recovery group (HR) compared to rest recovery group (RR), oxygen recovery group (OR), and the hyperbaric oxygenation (HBO2).

Interestingly these results may initiate HBO therapy as a relatively quick treatment for recovery following fatigue in sports requiring repeated high intensity efforts. Although there has been reports of reduced post-injury swelling and tissue remodeling after injury, with injuries involving bones, muscles and ligaments showing improved recovery. HBO therapy may be ethical and moral as a treatment for trauma injuries and other injuries sustained however, the question of HBO therapy relevance, ethical and moral use during a sporting competition should be investigated and trialed before promoting it's use.

Despite its potential, HBO treatment does have its risks. Increasing oxygen levels in tissues poses a risk to DNA through oxidative damage, which can lead to pathological changes in the CNS and the lungs. Groger, Speit, Radermacher, & Muth (2005) investigated HBO, pure oxygen breathing at supra-atmospheric pressures, and suggested it represents a well-suited model for investigating oxidative stress-induced DNA damage as well as protective mechanisms. They concluded that both heme oxygenase-1 blockade and excess nitric oxide release promote DNA damage during HBO exposure in vivo. Regarding the operating of HBO systems, safer administration should be advised. Further research into HBO treatment is required if this therapy is to become more widespread. However, Ishii et al, (2005) suggest "it should become possible to tailor treatment to an individual's condition in order to use HBO treatment efficiently".

In contrast to the previous investigations, Webster, Syrotuik, Bell, Jones, & Hanstock, (2002) suggest HBO therapy is not an effective method of treatment for accelerating recovery following exercise-induced muscle damage. Strenuous eccentric exercise elicited muscle damage within the right gastrocnemius muscle. Subjects subsequently received either HBO (100% oxygen at 253 kPa [2.5 ATA] for 60 min; n = 6)

or sham (atmospheric air at 132 kPa [1.3 ATA] for 60 min; n = 6) treatment conditions. The first treatment was administered 3-4hr after damage, with a second and third at 24 and 48hr after the first, respectively. The results indicate there was little evidence of a difference in recovery rate between the HBO and sham groups although a faster recovery was observed in the HBO group for isometric peak torque and pain sensation and unpleasantness compared to the sham groups. Similarly Mekjavic, Exner, Tesch, & Eiken (2000) and Bennett, Best, Babul, Taunton, Lepawsky, & Bennett (2005) investigated the effects of HBO therapy on recovery after exercise-induced muscle injury. Over a 10 day recovery period, there was no difference in the rate of recovery of muscle strength between the two groups. Perceived soreness peaked at about 48hr after exercise with no difference between groups (Mekjavic et al, 2000). Bennett et al, (2005) showed there was some evidence that HBO therapy may increase interim pain in DOMS. However there was insufficient evidence from comparisons tested within randomised controlled trials to establish the effects of HBO therapy on soft tissue injury, or on experimentally induced DOMS. These results indicate that HBO therapy was not an effective therapy for the treatment of DOMS. Interestingly, the phagocytic and regenerative steps associated with muscle injury recovery may be dependant on the oxygen % and time of exposure to a HBO therapy. Similar to the previous study, Harrison, Robinson, Davison, Foley, Seda & Byrnes (2001) investigated the role of hyperbaric oxygen therapy in the treatment of exercise-induced muscle injury. The authors also concluded that the hyperbaric therapy was not effective in the treatment of exercise-induced muscle injury as indicated by the markers of isometric strength, rating of perceived soreness of the forearm flexors and creatine kinase. It is possible that a specific treatment may reduce muscle injury recovery and provide insight into the recovery process following EIMD after unaccustomed exercise.

There have been two mechanisms proposed whereby HBO may assist in the treatment of muscle injuries. The primary effect of HBO exposure is an increase in the oxygen content of arterial blood (P_aO_2) and a subsequent increase in the O_2 diffusion gradient between the blood and tissue. A secondary effect may be a reduction in the inflammatory response due to a PO_2-mediated vasoconstriction (Staples, Clement, Taunton, McKenzie, 1999). The timing of the HBO exposure may be of importance as the immediate post-injury administration would increase the potential oxygen diffusion gradient and reduce the inflammatory response (Harrison et al, 2001). However, it is still unclear whether or not there is significant soft tissue damage, and whether HBO therapy does reduce inflammation or just slow the process. It is possible that if the inflammatory process was over a longer time period it would cause necrosis and the subsequent tissue repair to be delayed. The current research on the effect of HBO therapy on muscle damage recovery is inconclusive. However, research employing similar methods and design to the HBO therapy have not previously been used too investigate the effects of hypoxia on skeletal muscle regeneration.

2.4 Air Travel

Many aspects of the effects of mild hypoxia on human performance have been investigated since 1935, making significant contributions to flight safety (Armstrong, 1935; McFarland & Evans, 1939; Bryan & Leach, 1960; McFarland, 1971; Harding & Mills, 1983). Armstrong's (1984) investigation of high-altitude flight included factors affecting tolerance of acute hypoxia, and oxygen toxicity, specifying the physiological requirements of sealed high-altitude aircraft compartments. The Royal Australian Air Force has recognized hypoxia as a significant physiological threat at altitude and have traditionally

trained aircrew to recognize the symptoms of hypoxia using hypobaric chamber training at simulated altitudes of 25,000 ft or more. Cable (2003) analyzed incidents of hypoxia reported to the Directorate of Flying Safety of the Australian Defence Force (DFS-ADF) for the period 1990-2001. The author found that twenty-seven reports of hypoxia involving twenty-nine aircrew were reported. In only two cases was consciousness lost, and one of these resulted in a fatality. Most incidents (85.1%) occurred in fighter or training aircraft with oxygen equipment routinely used with the majority of symptoms occurring between 10,000 and 19,000 ft. The most common cause of hypoxia (63%) in these aircraft was the failure of the mask or regulator, or a mask leak. The symptoms were subtle and often involved cognitive impairment or light-headedness with the vast majority (75.8%) of these episodes recognized by the aircrew themselves. Therefore, useful planning maybe used to develop training strategies which reinforce the importance and benefit of hypoxia training in aviation physiology. Cable (2003) establishes the importance and effectiveness of the implementation of a hypoxic training program. Hypoxia incidents are most common at altitudes less than 19,000 ft therefore it should be emphasized that hypoxia is not only a problem of high altitude. Hypoxia in flight still remains a serious threat to all air travelers and can result in fatalities.

This illustrates that aviation medicine was involved in arriving at the practical compromise with the often conflicting requirements of aircraft design (Ernsting, 1978). The cabins of virtually, all modern passenger aircraft are pressurized with air in order to prevent the occupants being exposed to low partial pressures of the environment at high altitude (Ernsting, 1978). Cabins are commonly pressurized to below 10,000ft and usually between 5,000 and 7,000ft (Harding & Mills, 1983). The acceptance of mild hypoxia induced by breathing air at altitudes of up to 8,000ft (alveolar Pco_2 of 40 mm Hg) is the correct

compromise for the allowable cabin altitudes of passenger aircraft (Ernsting, 1978). Respiration increases under the hypoxic drive, the symptoms and signs of hyperventilation develop with those of hypoxia. Hyperventilation is a normal response to a fall in alveolar PO_2 to below 7.3-8.0 kPa (55-60 mm Hg) (Harding & Mills, 1983). Depending on the relationship between cabin and aircraft altitudes, it may be necessary to increase the concentrations of oxygen in the inspired gas above that which produces an alveolar PO_2 of 103 mm Hg (ambient air at sea level), in an attempt to prevent hypobaric hypoxia (Ernsting, 1978). The correct compromise is to provide a level of oxygen concentration in the inspired gas which maintains a sea level equivalent of inspired PO_2 of 149 mm Hg, to inspired air at an altitude of 5,000ft, PO_2 of 122 mm Hg. There appears to be advantages in maintaining the absolute pressure within the cabin at 1 atm during flight (Ernsting, 1978). However, the requirements to maintain absolute cabin pressure would reduce aircraft performance and its structure. The degree of pressurization increases linearly with, but at a slower rate than actual altitude, from ground level to a high maximum differential pressure (Harding & Mills, 1983). The greater the differential pressure of the environment at high altitude, the stronger and heavier the structure of the aircraft cabin needs to be (Ernsting, 1978). Although medical authorities have accepted mild hypoxia as being a safe compromise for healthy individuals, the question of whether hypoxia exposure is detrimental to an injured athlete has not been answered.

2.5 Eccentric Exercise-Induced Muscle Damage

It is well-established that eccentric muscle actions induce muscle tissue damage, when the muscles are unaccustomed to the exercise (Armstrong, Warren, & Warren, 1990; Clarkson et al., 1992; Warren, Hayes, Lowe, & Armstrong, 1993; Hunter, & Faulkner,

1997; Ingalls, Warren, Williams, Ward, & Armstrong, 1998). Morphological changes are direct evidence of muscle damage (Friden, Sjostrom, & Ekblom 1983; Jones, Newham, Round, & Tolfree 1986). Indirect evidence such as the development of muscle soreness and swelling, decreases in force generation and range of motion, and elevated muscle protein levels (creatine kinase, myoglobin) in the blood demonstrate damage (Clarkson et al., 1992; Nosaka & Clarkson, 1996).

Mechanical and metabolic mechanisms have been proposed for the initial events of EIMD (Armstrong et al., 1991). EIMD is assessed by measuring the efflux of specific cytosolic enzymes into the circulation, combined with histological techniques or ultrastructural examination. Muscle damage to small focal areas of the fibres has been observed immediately after exercise with the damage often becoming more extensive 48-72hr post-exercise. Histological examination of the affected muscle shows myofibrillar disruption, increases in mitochondrial density and the content of cytoskeletal and myofibrillar proteins (Kuipers, Jannsen, Bosman, Frederik & Geurten, 1991). The increased concentration levels of cytosolic proteins in the circulation after exercise reflects muscle damage (Jones, Newham, Round & Tolfree, 1986). The most commonly studied protein is creatine kinase (CK), a protein normally detected in blood when muscle is damaged and caused by the leakiness of that muscle. Other muscle proteins such as lactate dehydrogenase, asparate aminotransferase, alanine aminotransferase, and myoglobin are also released into the blood and follow the same time course as CK (Nosaka, Clarkson, & Apple, 1992). The presence of these proteins and amino acids in the circulation reflects a significant alteration in the structure and permeability of the myofibrillar membrane (Jones et al., 1986). CK is found exclusively in muscle tissue therefore most studies of EIMD have included its measurement.

The time course of the CK response has been studied following downhill running, marathon running and isometric exercise and usually peaks between 24-72hr post-exercise (Nuttal, Jones, 1968; Nosaka, Clarkson, & Apple, 1992; Clarkson et al., 1985). However, the amount of change and the time course of CK following these exercises is markedly different too that found following eccentric exercise (Clarkson, Nosaka, & Braun 1992). Byrnes et al. (1985) found the increase in CK activity in the blood to be substantially lower following downhill running compared with eccentric exercise of the elbow flexors. The changes in CK activity following downhill running are similar to the changes observed for isometric exercises (Clarkson, Apple, Byrnes, McCormick, & Triffletti, 1987; Clarkson, Litchfield, Graves, Kirwan, & Byrnes, 1985). Furthermore, the magnitude of muscle damage is also influenced by many factors, and responses to eccentric exercise are different between arm and leg muscle eccentric exercise. Tiidus & Ianuzzo (1983) reported that the intensity and duration of exercise affected serum enzyme activity and muscular soreness independently, with intensity having a greater effect.

Why the various exercise models produce different results is not known. A possible explanation may be the extent of injury induced by the various regimes of exercise. Therefore, the larger increase in CK after high-force eccentric exercise compared with downhill running may be explained. However, Clarkson et al. (1992) suggest there is no explanation for the long delay before the rise in these proteins in the blood after high-force eccentric exercise. The blood levels of other muscle proteins (lactate dehydrogenase, asparate aminotransferase, alanine aminotransferase, and myoglobin) follow the same time course (Nosaka, Clarkson, & Apple, 1992). It is possible these proteins are not released by muscle fibers for at least 24hr, as Clarkson et al. (1992) suggested that the CK release from muscle following eccentric exercise may reflect necrosis of focal areas on the muscle

fibers. The eccentric exercise may initiate a series of events over a 48hr post-exercise period leading to focal necrosis. This series of events may involve an accumulation of calcium inside the cell, arising from damage to the membrane or to the sarcoplasmic reticulum (SR), activating cell degradation.

An elevation in intracellular calcium ion (Ca^{2+}) concentrations at the site of muscle damage indicates cellular necrosis and a loss of Ca^{2+} homeostasis. This elevation results in a decrement of muscle fibre performance (Armstrong et al., 1991). Ingalls et al., (1998) concluded that 'the primary site for the excitation-contraction (E-C) coupling failure is the interface of the t-tubule and the SR Ca^{2+} release channel'. The reduced Ca^{2+} release and Ca^{2+} sensitivity account for the reduction in muscle force generating capacity (Balnave & Allen, 1995). Despite numerous studies having demonstrated eccentric EIMD, there is a conflicting consensus as to the actual mechanism. In general, the most convincing evidence supports mechanical factors rather than metabolic factors responsible for the initiation of muscle damage (Warren et al., 1993; Armstrong, et al., 1991). Mechanical factors such as sacromere length (Hunter, & Faulkner, 1997; Jones, Newham, & Torgan, 1989) strain, (Brooks, Zerba, & Faulkner, 1995; McCully & Faulkner, 1985, 1986) velocity (Morgan, 1990) total work (Lieber, Woodburn, & Friden, 1991) and force (Brooks, Zerba, & Faulkner, 1995) during eccentric contractions have been proposed as factors determining the level of EIMD. The initial exposure and magnitude of an eccentric contraction, including the individual reaction, may influence the amount of muscle tissue damage and any further damage as a result of multiple trials.

Eccentric EIMD causes delayed-onset muscle soreness (DOMS) as a result of muscle fibre damage. DOMS usually occurs from 1–5 days following unaccustomed exercise, reducing muscular performance by a voluntary reduction of effort and/or from the

muscle's inability to produce force(s). The reduction in performance is temporary and without any permanent impairment. Clarkson, Nosaka, & Braun (1992) measured variables, maximal strength and the ability to flex the arm, following eccentric exercise and showed the greatest decrements immediately following the exercise, to three days after exercise with a linear restoration of these functions over the next ten days. However, the decrements may be relative to the muscle type damaged. The quadriceps muscles of the leg may not damage similarly to the biceps muscles of the arm as there is less mass and possibly more or less fast/slow twitch fibres in that particular muscle investigated. Therefore, the information presented must be met with caution when analyzing data.

2.5.1 Creatine Kinase Activity

Increased levels of muscle proteins in the blood have routinely been used to diagnose muscle damage (Byrnes et al., 1985; Clarkson, & Tremblay, 1988; Ebbling, & Clarkson, 1990). The most commonly studied protein is creatine kinase (CK), a protein normally detected in blood when muscle is damaged and caused by the leakiness of that muscle. The time course of the CK response has been studied following downhill running, marathon running and isometric exercise. However, the amount of change and the time course of CK following these exercises is markedly different to that found following eccentric exercise (Clarkson, Nosaka, & Braun 1992). Byrnes et al., (1985) found the increase in CK activity in the blood to be substantially lower following downhill running compared with eccentric exercise of the elbow flexors. The changes in CK activity following downhill running are similar to the changes observed for isometric exercises (Clarkson, Apple, Byrnes, McCormick, & Triffletti, 1987; Clarkson, Litchfield, Graves, Kirwan, & Byrnes, 1985).

Why the various exercise models produce different results is not known. A possible explanation may be the extent of injury induced by the various regimes of exercise. Therefore, the larger increase in CK after high-force eccentric exercise compared with downhill running may be explained. However, Clarkson, Nosaka, & Braun (1992) suggest there is no explanation for the long delay before the rise in these proteins in the blood after high-force eccentric exercise. The blood levels of other muscle proteins (lactate dehydrogenase, asparate aminotransferase, alanine aminotransferase, and myoglobin) follow the same time course (Nosaka, Clarkson, & Apple, 1992). It is possible these proteins are not released by muscle fibers for at least 24hr, as Clarkson, Nosaka, & Braun (1992) suggested that CK release from muscle following eccentric exercise may reflect necrosis of focal areas on the muscle fibers. The eccentric exercise may initiate a series of events over a 48hr post-exercise period leading to focal necrosis. This series of events may involve an accumulation of calcium inside the cell, arising from damage to the membrane or to the sarcoplasmic reticulum (SR), activating cell degradation.

2.6 Exercise-Induced Muscle Damage Recovery

Previous studies have shown that various forms of eccentric EIMD, although the damage mechanism and repair are unclear. Hurley (1983) suggested chemical, thermal or mechanical stimuli may be a mechanism responsible for muscle damage. The two mechanisms proposed in an effort to explain muscle damage following exercise are mechanical stress and metabolic stress. Mechanical stress has been proposed as being responsible for muscle damage as a direct result of the mechanical shear forces produced during exercise, whilst metabolic stress is characterized by disturbances in normal cellular metabolism caused by unaccustomed endurance exercise (Ebbeling, & Clarkson, 1989;

Armstrong, Warren, & Warren, 1991). It is possible that both mechanisms may be involved in initiating a sequence of inflammatory and immunological events leading to reversible damage to muscle fibres (Appell, Soares, & Duarte, 1992), removal of damaged tissue, promotion of growth, repair and restoration of normal physiological function (Armstrong, Warren, & Warren, 1991).

The process of inflammation also involves a vascular and cellular response. The vascular response causes an initial vasoconstriction for approximately 5-10min, followed several hours later by a period of vasodilation and increased vascular permeability (Ryan, & Majno, 1977). The cellular phase of inflammation involves two types of white blood cells, neutrophils and monocytes. Within a few hours following muscle damage there is an increase in the number of circulating neutrophils and monocytes, where the cells begin to migrate at the site of disruption. Monocyte migration begins several hours after neutrophil migration with the concentration at the site of injury rising steadily where it is maintained for 48 hours. When the monocytes leave the blood and enter the tissue compartment, they mature into adult macrophages. Their primary purpose is to dispose of necrotic tissue and remove foreign bodies (Peacock, 1984. pp. 1-14).

Several studies have investigated the use of therapies or treatments to restore injured athletes to full recovery (Harrison, Robinson, Davison, Foley, Seda, & Byrnes, 2001; Strauss, Hargens, Gershuni, Hart, & Akeson, 1986; Tiidus, 1997; Rodenburg, Bar, & De Boer, 1993). However, there are conflicting results regarding treatment efficacy for these related injuries. These studies have been limited by subject availability and study designs characterized by the precise control of variables; degree of injury, type of injury and timing of initial treatment.

At the elite level of competition the treatment of soft tissue injuries are frequently treated with the use of hypobaric oxygen. Hyperbaric oxygen therapy is the breathing of 100% oxygen in a treatment chamber at 2-3 atmospheres absolute pressure (Harrison, Robinson, Davison, Foley, Seda, & Byrnes, 2001). Although, the hemoglobin is 97% saturated by ambient air, breathing 100% oxygen increases the hemoglobin-oxygen saturation by 3% whilst the remaining oxygen is dissolved in the plasma. However, there is always a waste of approximately 5% oxygen when inspiration occurs, so the question of whether breathing 100% oxygen is of benefit has not been answered to date. Therefore, is breathing 100% oxygen an expensive luxury for those who can afford it and is it ethical if not everyone is able to afford it? It is suggested more research including ethical behaviour is required into HBO therapy for muscle damage recovery.

Following a high impact injury, Nylander, Lewis, Nordstrom, et al., (1985) and Nylander, Nordstrom, Lewis et al., (1987) established that ATP in postischemic muscle was preserved with a 50% reduction of edema following a hyperbaric treatment. Hyperbaric oxygen supposedly improves environment conditions for damaged muscles, tendons and ligaments. Presently, the benefit(s) associated with hyperbaric oxygen treatment, as a very expensive therapy modality of soft tissue injuries, remains unproven whilst the risks; such as tympanic membrane perforation are significant. Currently, the scientific evidence suggesting the use of hyperbaric oxygen in the treatment of soft tissue injury remains inconclusive.

2.8 Summary

The lack of literature on the effect of hypoxia on muscle damage suggest that recovery procedures which incorporate strategies for continued recovery post game and

training regimes need to be investigated so that training recovery procedures during travel will assist in providing adequate recovery of damaged muscle tissue in time for the next training session and for the following competition.

The investigators of the present study hypothesized hypoxia experienced during short-term air travel may delay exercise-induced muscle damage recovery. Muscle damage recovery following short-term (4hr) exposure to normobaric hypoxia after eccentric EIMD has not been previously investigated, therefore, the investigators of the present study believe research into the effects of short-term (4hr) normobaric hypoxia following muscle damage is valuable.

CHAPTER THREE

METHODOLOGY

Previous studies investigating the phenomena of exercise-induced muscle damage have shown measures of increased plasma CK, soreness, tenderness and prolonged loss of muscle function to be common indicators of muscle damage (Clarkson, & Tremblay, 1988; Byrnes et al., 1985; Sargeant, & Dolan 1987, Edwards, Mills & Newham, 1981; Eston, Finney, Baker, & Baltzopoulos, 1996). Previous studies (Nosaka, & Sakamoto, 2001; Schwane, Johnson, Vandenakker, & Armstrong, 1983a, Clarkson, Byrnes, Gillisson, & Harper, 1987) have shown eccentric muscle actions induce muscle damage. The present study's knee extensor exercise model was designed using the previous studies protocols as the knee extensor muscles are involved in most exercise activities.

3.1 Ethical Considerations

Ethical approval was granted by Edith Cowan University Ethical Committee and guidelines outlined by the University dated February 1998 were adhered to. Subjects gave their informed consent (Appendix A), and were free to withdraw their participation at any time of the investigation. Data handling was carried out by the primary investigator and secured in a filling cabinet and personal computer.

3.2 Subjects

The subject cohort for this investigation included sixteen male subjects aged 18-34 (mean $\pm$ SD) 21.9 $\pm$ 5.2 years, height 176.2 $\pm$ 8.8 cm and body mass 73.7 $\pm$ 8.2 kg (see

Table 1). Although the sample size in the present study is small, the number of subjects is similar to previously mentioned studies therefore allows for comparisons. Each subject signed a informed consent form (Appendix A) and completed an extensive medical history questionnaire (Appendix B), after being advised of the purposes of the investigation and methodology of tests. Subjects were randomly assigned to either a control (CON or normoxia) and hypoxic (H) groups (see Table 1). Although there is a significant difference between H and N group's ages there was no significant difference between the H and N group's pre-EIMD measured variables. Subject identification numbers were allocated and used to ensure confidentiality. Subjects had not been involved in eccentrically based conditioning or strength training of the lower limb in the 6 months prior to the investigation and reported not having any previous history of lower limb injury and were free from muscle soreness/tenderness. Subjects were informed they were able to participate in their normal daily activity, but were requested to refrain from taking anti-inflammatory medicines and/or similar medications.

Table 1

Subject characteristics

Hypoxic Group				Normoxic Group			
Subject	Age (yr)	Height (cm)	Body Mass (kg)	Subject	Age (yr)	Height (cm)	Body Mass (kg)
H1	25	168.20	67.20	N1	19	176.50	76.80
H2	31	180.60	79.20	N2	18	180.00	79.50
H3	21	176.00	69.00	N3	19	187.00	81.90
H4	30	164.00	73.00	N4	22	179.00	71.00
H5	34	167.50	74.00	N5	21	175.00	75.70
H6	18	174.00	72.00	N6	18	160.50	59.30
H7	18	190.50	79.00	N7	18	168.00	61.00
H8	19	183.50	68.00	N8	20	188.00	92.50
Mean	25.29	174.40	73.34		19.29	175.14	72.17
SD	6.44	9.00	4.63		1.51	9.19	10.93
SE_M	2.28	3.18	1.64		0.53	3.25	3.86
SE_D	2.34	4.55	4.19				
t test	0.04	0.80	0.69				

3.3 Testing Procedures

3.3.1 Testing Schedule

Each subject was tested over a 10-day period (Table 2) with testing protocols identical for the H and N groups. Subjects were randomly assigned into a H or N group. As the test-retest results showed no significant difference between groups, testing immediately after the eccentric EIMD protocol was not included as previous studies have established 24-48hr post exercise is the period of maximal soreness and tenderness. Furthermore, the number of variables measured, and the H and N 4hr treatment following EIMD made it impractical to measure variables immediately after EIMD. However, it is noted that testing immediately following eccentric EIMD may be important to establish no significant differences between conditions at this time point.

Table 2.

Outline of the testing protocol over the 10-day period.

Day	-4	-3	-2	-1	0	1	2	3	4	5
Stage	Pre-test				EIMD	Post-test				
Test Number	1		2		3	4	5	6	7	8
Variables	1. Anthropometry Height (cm), Body mass (kg) 2. Creatine kinase activity 3. Soreness 4. Tenderness 5. Isometric max strength 6. Isokinetic peak torque 7. Vertical jump height 8. Eccentric leg strength				1. Warm up & stretch 2. Eccentric EIMD protocol 3. Cool down & stretch 4. Normobaric hypoxia exposure (test group) 5. Normobaric normoxia (control group)	Test 1 variables 2 – 8 Post – test time 24, 48, 72, 96, 120hr				

EIMD = Eccentric exercise induced muscle damage

The results for Tests 1 & 2, isometric strength, isokinetic peak torque and vertical jump for all subjects were used for determining the reliability of the testing protocols. Both

H and N groups performed the same series of tests. The N group was exposed to four-hours of normobaric hypoxia, while subjects in the H group experienced a four-hour normobaric normoxia condition following the eccentric EIMD protocol on the third testing session (day five). Baseline measures used for comparisons across time were taken from the subject's highest peak values recorded during either Test 1 or Test 2. Measurements taken during the various test days are shown in Table 3.

Table 3.

Testing sessions and variables measured

Testing Variables		
Test 1 & 2	Test 3	Tests 4 – 8
1. Anthropometry Height (cm), Body mass (kg) 2. Creatine kinase activity 3. Soreness 4. Tenderness 5. Isometric max strength 6. Isokinetic peak torque 7. Vertical jump height 8. Eccentric leg strength	1. Warm up & stretch 2. Eccentric EIMD protocol 3. Cool down & stretch 4. Normobaric hypoxia exposure (test group) 5. Normobaric normoxia (control group)	Test 1 variables 2 – 8 Post – test time 24, 48, 72, 96, 120hr

3.4 Testing Protocols

3.4.1 Warm-up

Subjects completed a six-minute intermittent warm up on a Repco cycle ergometer (RE7000, Australia) at intensities comfortable to the individual subject followed by stretching of the quadriceps, hamstrings and calf muscles of both lower limbs. Subjects were permitted a 2-minute preparation period to re-hydrate, adjust their clothing or visit the bathroom before performing the EIMD (knee extensor) protocol.

3.4.2 Eccentric Exercise Induced Muscle Damage Protocol

An intermittent eccentric contraction of the knee extensor muscle group (described by Tiidus & Ianuzzo, 1983) was used to induce muscle damage of the quadricep muscles for the H and control groups using a Cybex 6000 dynamometer (Cybex division of Lumex, Ronkonkoma, New York).

Subject's performed 60 (10 reps x 6 sets) eccentric contractions of the knee extensors of the dominant (preferred) leg at the angle from 0^{O} (fully extended position) to 90^{O} (fully flexed position). Muscle contractions were performed at a rate of $9^{O} \cdot s^{-1}$ to maximize the damage effect, controlled by Cybex 6000 isokinetic dynamometer (Cybex division of Lumex, Ronkonkoma, New York), with a 20s inter-trial rest. Each of the six sets was separated by a 2 minute rest period for maximal repeat efforts. The exercising limb was returned to the fully extended position at a rate of $9^{o} \cdot s^{-1}$ by the Cybex 6000 isokinetic dynamometer. The subject begins the exercise with the leg fully flexed (90^{o}). The investigator initiates the exercise using the computer system attached to the apparatus. The mechanical lever system then extends the limb to the fully extended position (0^{o}). The subject then starts the apparatus by contracting the quadriceps muscles. The mechanical advantage of the computer lever system is that it provides maximal resistance to the subject throughout the entire range of motion. Each of the six sets was separated by a 2 minute rest period. This allowed the subject to perform a series of maximal effort eccentric actions. Subjects were seated upright and stabilized using a lap sash seat belt around the trunk and pelvis, including a thigh strap securing the exercised limb. Subjects were required to fold their arms across their chest minimizing extraneous body motion and the effect of accessory muscle groups. Subjects received verbal encouragement throughout each trial.

3.4.3 Normobaric Hypoxia Protocol

The normobaric hypoxia protocol was performed exactly two hours after the eccentric EIMD and consisted of a four-hour treatment of breathing a medical grade gas, consisting of 15.37% O_2 and 84.63% N_2, in an isolated room with equipment (chairs set up in rows with small tables, a television and video, music and magazines) similar to that present within a passenger aircraft. Each subject wore a mask attached to an oxygen cylinder (tissot tank) with a flow rate of 5ml per minute. Humidity, barometric pressure and temperature were maintained and recorded.

3.4.4 Normobaric Normoxia Protocol

The normobaric normoxia protocol was performed exactly two hours after the eccentric EIMD and consisted of a four-hour (similar time frame for normobaric hypoxia protocol) breathing ambient air, in an isolated room with equipment (chairs set up in rows with small tables, a television and video, music and magazines) similar to that within a passenger aircraft. Subjects wore a mask attached to an oxygen cylinder (tissot tank) with a flow rate of 5ml per minute. Each subject was required to follow exactly the same protocols as the normobaric hypoxia condition, controlled by the investigator. Humidity, barometric pressure and temperature were maintained and recorded.

3.5 Testing Measures

3.5.1 Height

Subject's height was measured using height scales - Seca 220 whilst standing in the anatomical position with bare feet and heels together.

3.5.2 Body Mass

Subject's body mass was measured to the nearest 0.05kg using electronic scales – Mettler ID 1 MultiRange wearing underwear and bare feet.

3.6 Creatine Kinase (CK) Activity

A single blood sample, collection of 30 μL blood, from a finger prick sample from each subject was analyzed pre EIMD, immediately following the eccentric EIMD and 24, 48, 72, 96 and 120hr post eccentric EIMD using a Reflotron analyzer (Boehringer-Manheim, Pode, Czch Republic) to determine plasma creatine kinase (CK) activity. The normal reference range for CK activity using this method is 50-220 $IU \cdot L^{-1}$ according to the information provided by the spectrophotometer company.

3.7 Muscle Soreness

Measurements of quadriceps (Rectus femoris, Sartorius, Vastus & Vastus lateralis) muscular soreness was assessed using a scale of 1-10 (see Appendix C), where 1 = no soreness and 10 = very sore (Clarkson & Tremblay, 1988) at baseline, 24, 48, 72, 96, and 120hr post eccentric EIMD protocol. Subjects were required to walk 10 meters, in a straight line and assign a score of 1-10 when asked "how would you rate the level of pain you experienced in the quadriceps of the exercised leg normal (1), somewhat sore (3), moderately sore (5), very sore (8) or very, very sore (10)"?. The mean values for each subject's total soreness were used for statistical analysis.

3.8 Muscle Tenderness

Muscle tenderness evaluations were conducted on the quadriceps muscle group using a dobros myometer, with a maximum value of 100kp, at baseline and at 24, 48, 72,

96, and 120hr post eccentric EIMD. The myometer was placed on the muscle (Rectus femoris: located the anterior-inferior spine of the ilium and the top of the patella and patellar ligament to the tibial tuberosity, the point of measure was at the mid point on a straight line between the origin and insertion. Sartorius: located the notch between the anterior-superior and anterior-inferior spines of the ilium and the anterior medial condyle of the tibia, the point of measure was at the mid point on a straight line between the origin and insertion. Vastus lateralis: located the outer surface of the femur below the greater trochanter and upper half of the linea aspera and the outer half of the upper border of the patella and the patellar ligament to the tibial tuberosity, the point of measure was at the mid point on a straight line between the origin and insertion. Vastus: located the inner half of the upper boarder of the patella and patellar ligament to the tibial tuberosity, the point of measure was 10cm superiorly from the insertion along the linea aspera and internal condyloid ridge.), applying downward pressure until the subject indicated discomfort; a measure was then obtained from the myometer pressure gauge and recorded.

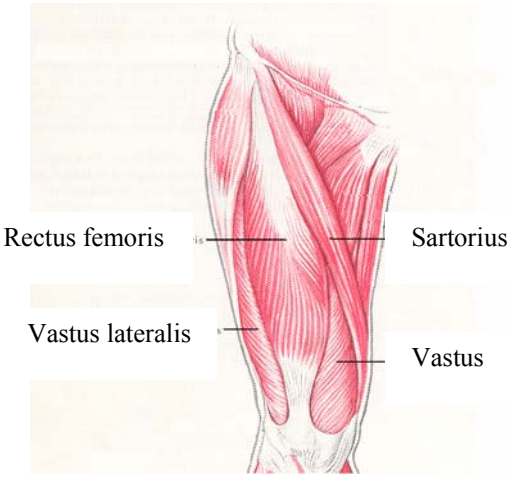


Figure 1 Quadriceps muscle group.
Markers indicate sites used for assessing tenderness.

Adapted from: Thompsom, C.W., (1985). Manual of structural kinesiology. (10th ed.). St.Louis: Times Mirror/Mosby.

3.9 Maximal Isometric Strength

Maximal isometric strength was determined using a Cybex 6000 Isokinetic Dynamometer (Cybex division of Lumex, Ronkonkoma, New York). Subjects were seated with the backrest at an angle of 125^0 and stabilized using a lap sash seat belt around the trunk and pelvis and a velcro strap around the thigh of the exercised limb. Subjects were required to fold their arms across their chest to minimize extraneous body motion and the effect of accessory muscle groups.

Subjects performed 3 maximal isometric voluntary contractions (MVC) involving the quadriceps muscle group (knee extensors) of the test limb. The axis of the dynamometer was aligned with the transverse axis of the knee joint with a shin pad placed 2.5 cm proximal to the medial malleoli. Gravitational effect on torque (GET) was determined for each subject and added to the knee extensor measure. The test protocol consisted of 2 x 10s maximal voluntary contractions (MVC) at 75^O, 55^O & 35^O of the knee extensors to assess recovery from one test to the second test. Tiidus & Ianuzzo (1983) reported the intensity and duration of exercise affected serum enzyme activity and muscular soreness independently, with intensity having the more pronounced effect therefore, 10's isometric model was used to increase the intensity and likelihood of muscle damage markers. Sixty seconds rest was permitted between the 2 test repetitions with 2 minutes rest between each test angle. The highest value measured for each set of 3 MVC was recorded as maximal strength (Nm). Subjects received verbal encouragement throughout the test.

3.10 Maximal Isokinetic Concentric Strength

Isokinetic peak torque was measured using a Cybex 6000 Isokinetic Dynamometer. Subjects performed 3 maximal concentric contractions involving the quadricep, hamstring

and gluteal muscle groups of the test limb, range of motion (ROM) from 90^{o} to 0^{o} (neutral) of knee extension/flexion at a velocity of 60, 120 and $240^{o}\cdot s^{-1}$. The axis of the dynamometer was aligned with the transverse axis of the knee joint with a shin pad placed 2.5 cm proximal to the medial malleoli. GET was determined for each subject and added to the knee extensor measure. The test protocol consisted of one sub-maximal practice repetition, followed by 3 MVCs of the knee extensors. Twenty seconds rest was permitted between each contraction. Each subject received verbal encouragement with the highest value of the 3 MVCs recorded as maximal isokinetic strength (Nm).

3.11 Squat Jump

Subjects muscle function followed protocols used by MacDougall, Wenger, and Green (1991, p64) using a TMFsports vertical jump tester. Subject's squat (or static) jump (SJ) height was measured as the difference between standing height and maximal height attained in a SJ. Subjects performed 3 single leg vertical jumps on their dominant limb with 1min rest between each repetition with the greatest height of the three measures recorded. The subject began the test in a crouched position with the hip and knee joints at a 90^{O} angle the subject was instructed to place their elbows to the side of their body, arms in a vertical position, hands in a position so that the fingers were at eye level. Following take off the subject's dominant arm extended to touch the vertical height indicator straps.

3.12 Statistical Analysis

Test-retest data for the coefficient of variation of method error and correlation of the coefficient for isometric strength, isometric torque and vertical jump height were determined from the two baseline tests (see Table 4). Each dependant variable was

analyzed using a 2 x 6 (group x time) repeated measure of analysis of variance (ANOVA), with significance set at $p<.05$. Significant differences found in the ANOVA were further analyzed using a SPSS statistical analysis program, Bonferroni adjustment. All results are reported as the mean ($\pm$ SEM) with raw and normalized data presented in Appendix D.

CHAPTER FOUR

RESULTS

4.1 Reliability

Data for the coefficient of variation of method error (CV) and correlation of the coefficient (*r*) for test-retest variables; isometric strength (35^0, 55^0 & 75^0 of knee flexion), isometric torque (60, 120 & 240 deg/s) and vertical jump height are presented in Table 4. Reliability for the testing variables was good as all test-retest variables have a coefficient of variation below 5%, indicating no systematic change between the two tests. The correlation coefficient (R) for test-retest measures of isometric strength, isokinetic torque and vertical jump were 0.96, 0.94 and 0.88 respectively.

Table 4.

Coefficient of variation of method error for testing variables

		Coefficient of Variation (C*V*) and Correlation of Coefficient (*r*)			
		Hypoxic (H) Group		Normoxic (CON) Group	
Test Variables					
Isometric Strength	angle	*CV* (%)	*r*	*CV* (%)	*r*
	35	2.36	0.98	3.55	0.94
	55	3.52	0.95	5.42	0.87
	75	4.07	0.97	4.32	0.95
Isokinetic Torque	deg/s				
	60	2.44	0.98	2.51	0.98
	120	4.61	0.93	3.68	0.97
	240	4.31	0.96	4.54	0.98
Vertical Jump		7.93	0.81	3.01	0.95

4.2 Extensor Isometric Strength

Isometric strength measures at 35^{O}, 55^{O} and 75^{O} are presented in Figures 2-5. A significantly greater strength increment at the angle 35^{O} was recorded from baseline to 120hr for the N group ($p<0.05$; Figure 2) compared to no significant difference between baseline to 120hr for H group. Figure 5 shows, extensor isometric strength at 35^{O}, a significant difference between the H and N groups at 48hr ($p<0.05$). H group isometric strength at 35^{O} decreased from baseline measures by 6% at 24hr then increased by 14% at 120hr compared to the N group isometric strength increase from baseline by 7% and 20% respectively. The testing bouts over time and the treatment by time interaction were statistically significant for both groups ($p<0.05$).

At 55^{O} the H and N group showed no significant difference across time although strength was elevated at 120hr compared to baseline. The H group showed a decline in strength at 24hr post-EIMD compared to the N group small increase (Figure 3). Isometric strength at 55^{O} showed a significant difference ($p<0.05$) between the H and N groups percentage change at 72hr post-EIMD. No significant differences were recorded for isometric strength between the H and the N group at the angle 55^{O} at any of the measurement times (Figure 3). However, mean isometric strength at 55^{O} for the H group decreased by 7% from baseline to 24hr post-EIMD compared to the N group, which increased by 5% from baseline to 24hr.

Following EIMD both the H and N group failed to show any decrement in strength (Figure 5). Isometric strength at 75^{O} showed no significant differences between the H and N groups at any of the measurement times (Figure 5). N group's isometric strength increased by 15% at 72hr, 20% at 96hr and 30% at 120hr post-EIMD compared to baseline measure. Whilst H group's isometric strength increased by 14% at 72hr, 7% at 96hr and 25% at

120hr compared to baseline. Although not significant, the overall pattern of increase in the isometric strength over time was greater for the N group compared to the H group. However, the H group shows a linear increase in strength across time whereas the N group shows incremental increases in strength from 24-48hr and 96-120hr.

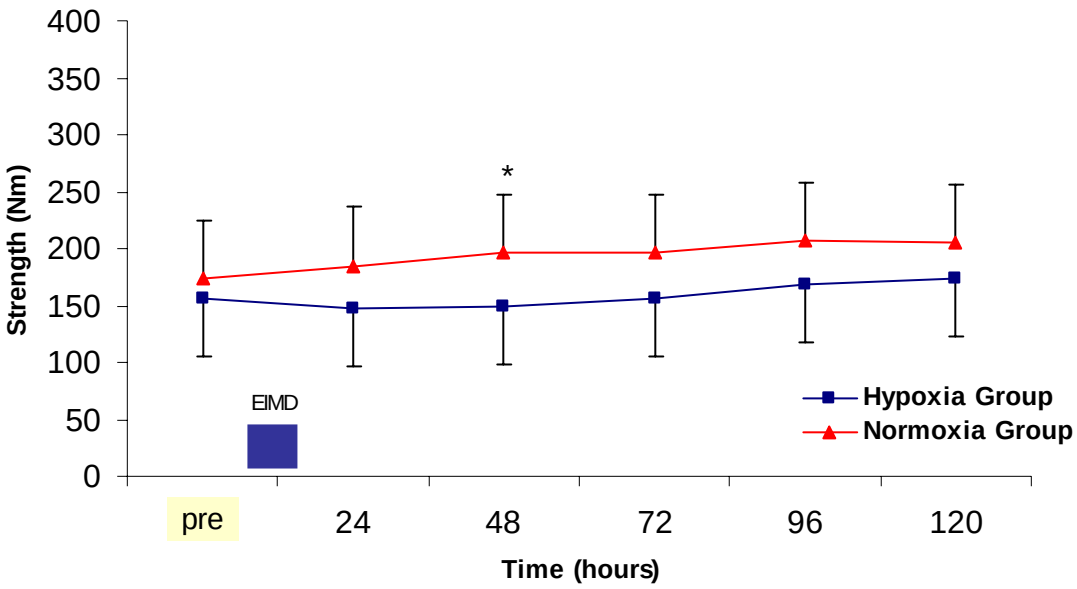


Figure 2. Extensor isometric strength (35 deg) across time for Hypoxia group (n=8) and Normoxia group (n=8). *Significant difference between H and N groups at 48hr ($p < 0.05$).

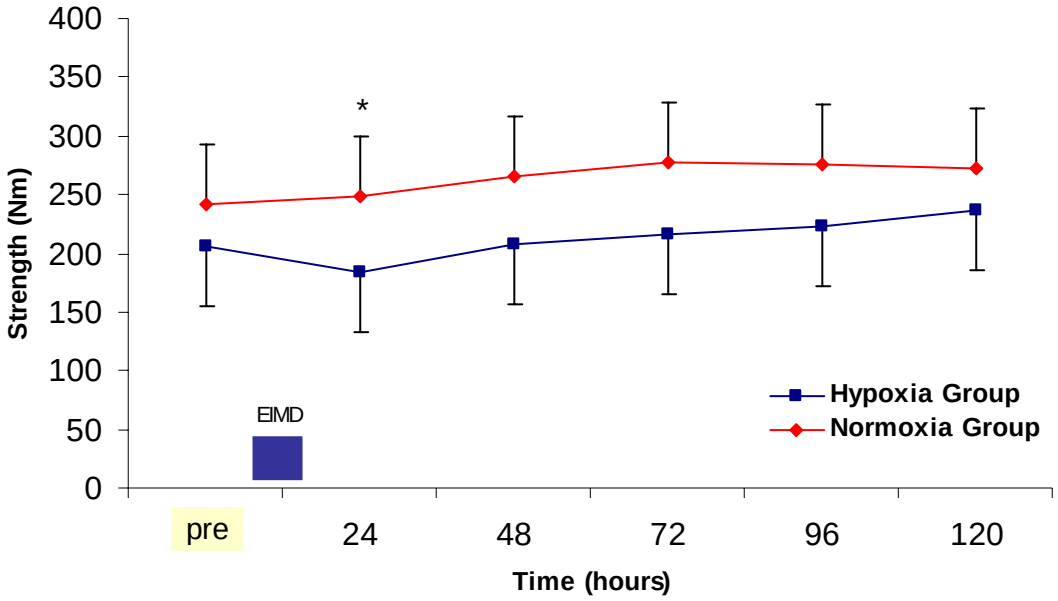


Figure 3. Extensor isometric strength (55 deg) across time for Hypoxia group (n=8) and Normoxia group (n=8).

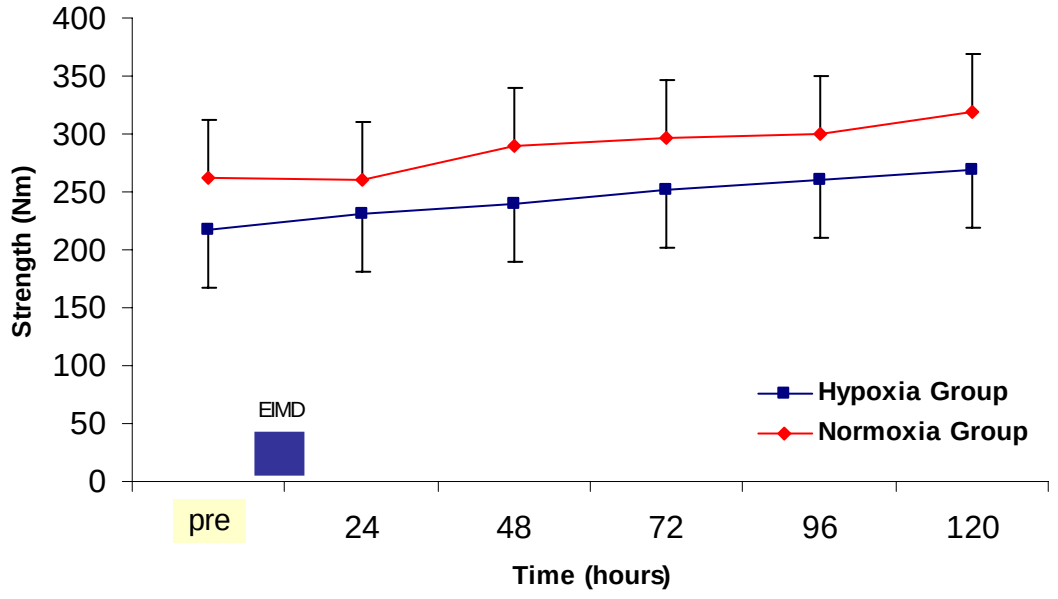


Figure 4. Extensor isometric strength (75 deg) across time for Hypoxia group (n=8) and Normoxia group (n=8).

4.3 Extensor Isokinetic Torque

Isokinetic torque at $60^{O\text{-}sec}$ was not significantly different between the H and N groups (Figure 5). Post-EIMD Isokinetic $60^{0\text{-}sec}$ torque for the N group increased progressively, peaking (13.3% greater than baseline) at 120hr. In comparison, H group $60^{0\text{-}sec}$ torque decreased progressively between baseline and 96hr post-EIMD, then peaked (7% greater than baseline) at 120hr. Figure 5 shows there was no significant group by time interaction effect for the N or H groups.

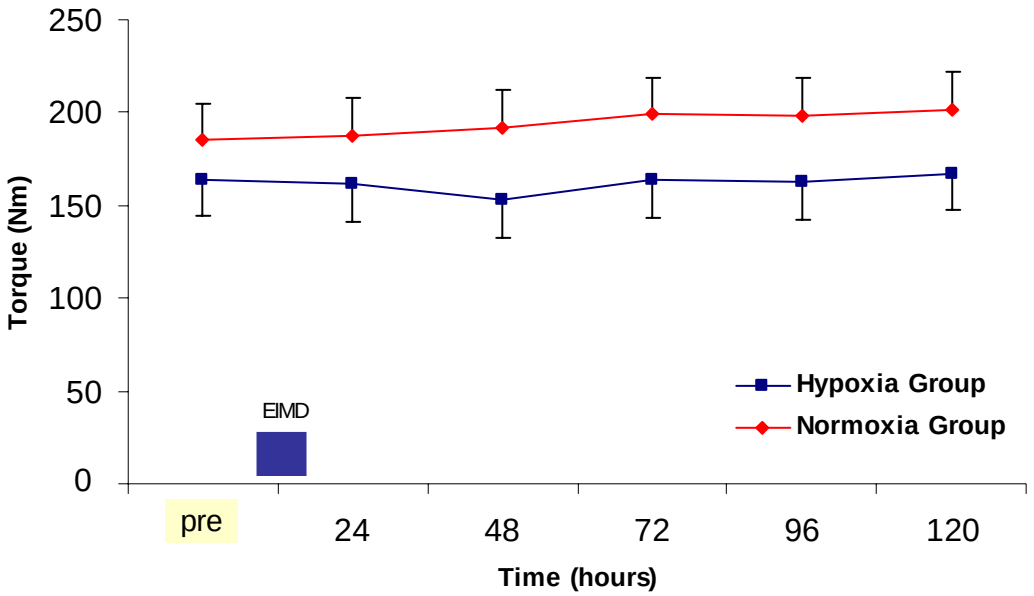


Figure 5. Extensor isokinetic torque (Nm) at 60 deg/sec across time for Hypoxia group (n=8) and Normoxia group (n=8).

Isokinetic torque at $120^{O\text{-}sec}$ was not significantly different between the H and N groups (Figure 6). Isokinetic torque for the N group was shown to increase by 3% (24hr) and 10% (120hr). However, a decrease of 7% (24hr) and increases of 16% (48hr) and 6% (120hr) were observed for the H group. There was no significant group by time interaction effect for the N or H groups.

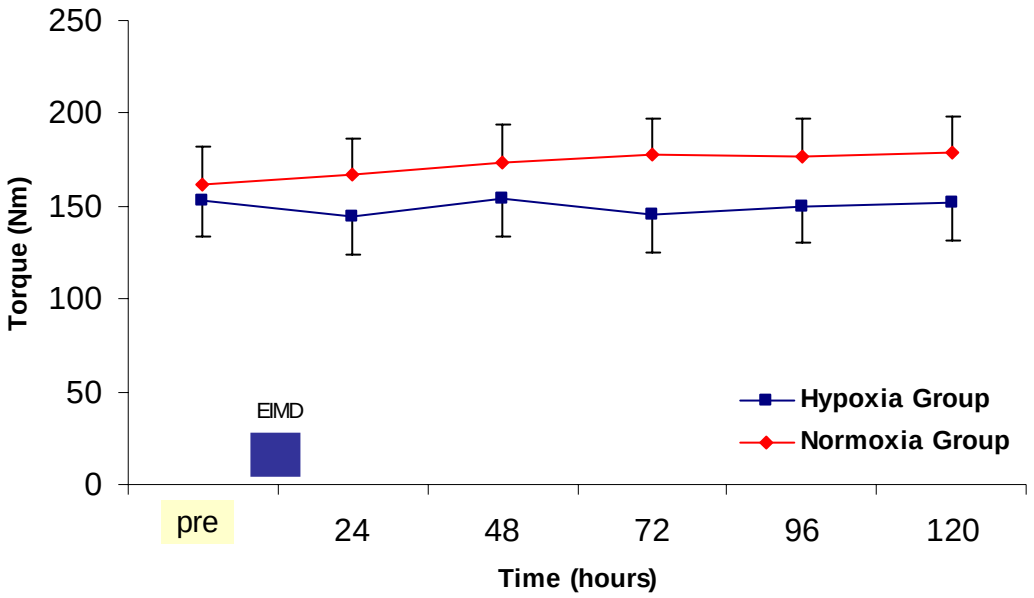


Figure 6. Extensor isokinetic torque (Nm) at 120 deg/sec across time for Hypoxia group (n=8) and Normoxia group (n=8).

Isokinetic torque at $240^{O\text{-}sec}$ was not significantly different between the N and H groups (Figure 7). Isokinetic torque at $240^{O\text{-}sec}$ increased by 16% (48hr) and 14% (120hr) for the N group, while the H group increased by 4% (48hr) and 5% (120hr). The results show there was no overall significant group by time interaction effect for both N and H groups.

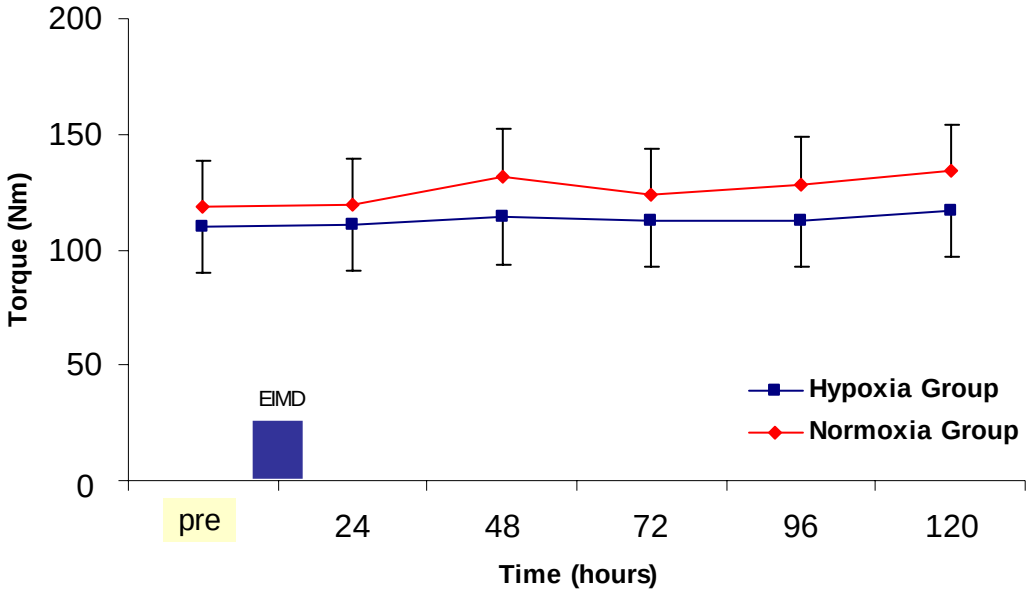


Figure 7. Extensor isokinetic torque (Nm) at 240 deg/sec across time for Hypoxia group (n=8) and Normoxia group (n=8).

4.4 Vertical Jump (VJ)

Squat jump (SJ), presented in Figure 10 showed that there was not a significant difference between the groups at any of the measurement times following EIMD, although SJ levels in the N group was greater than the H group. H group's SJ values decreased by 6% at 24hr post-EIMD in comparison to the N group's decrease of 3% respectively.

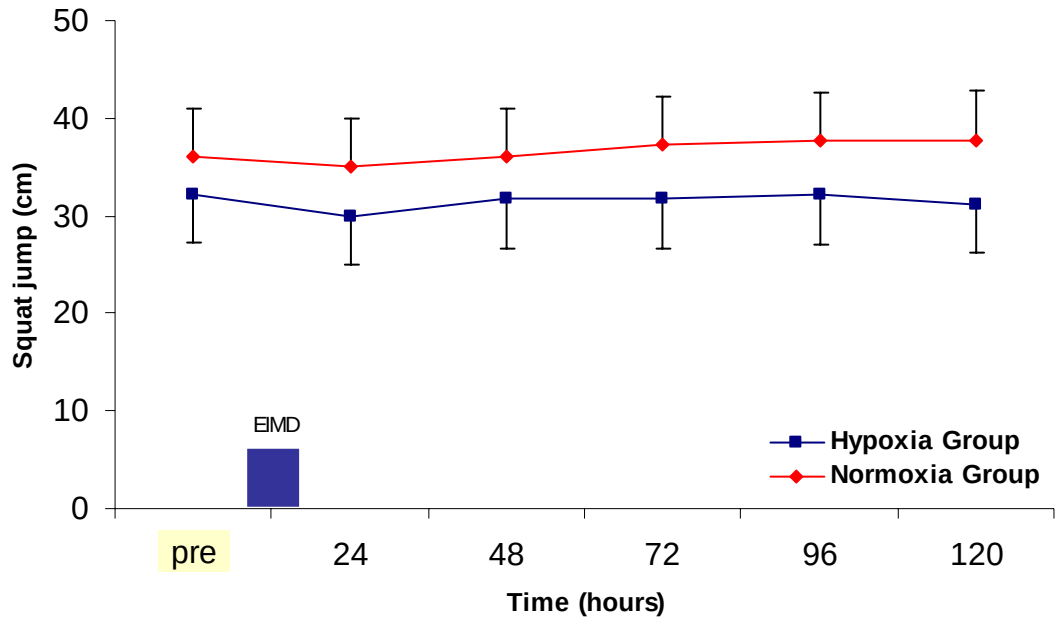


Figure 8. Vertical jump across time for the Hypoxia group (n=8) and Normoxia group (n=8).

4.5 Plasma Creatine Kinase Activity

Plasma creatine kinase (CK) activity is presented in Figure 9. Plasma CK was significantly greater (p<0.05) for the N and H groups at 24-96hr and 96-120hr respectively after EIMD compared to baseline measures, then gradually recovered. However, plasma CK was still elevated at 120hr after EIMD for both groups. Plasma CK activity was not significantly different between the H and N groups, although significantly larger increases in CK following EIMD were found between baseline measures and post-EIMD times for both groups (p<0.05). Plasma CK activity for both N and H groups peaked at 48hr and 96hr post-EIMD, respectively.

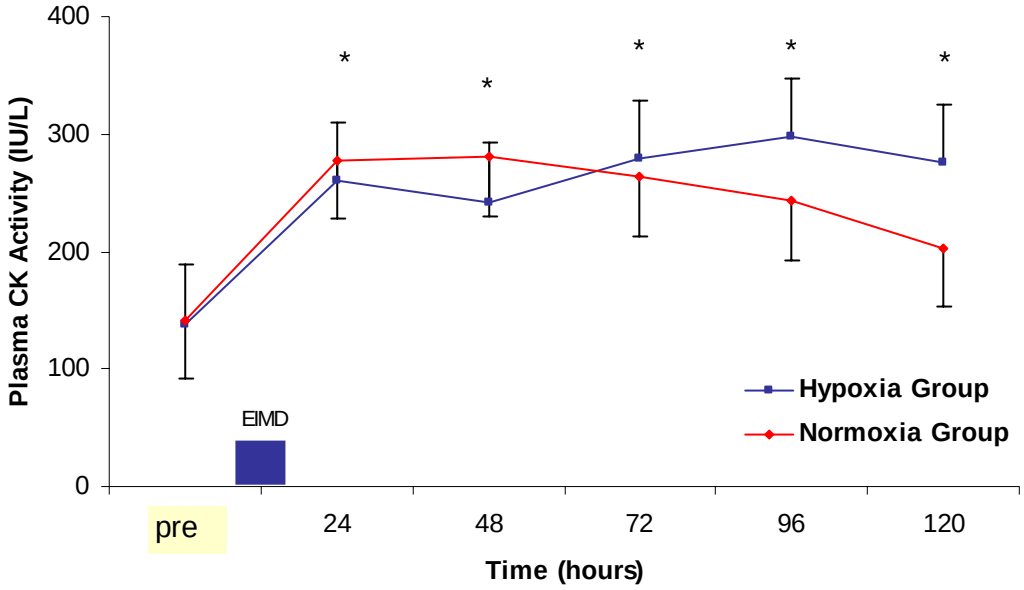


Figure 9. Plasma creatine kinase (CK) activity across time for the Hypoxia (n=8) and Normoxia groups (n=8). *Plasma CK activity levels were significantly ($p < 0.05$) elevated post- eccentric EIMD compared to baseline (0hr) at 24-96hr for N group and 96-120hr for H group.

4.6 Muscle Tenderness

Figure 10 shows H group tenderness declined from baseline to 48hr post-EIMD, then increased from 48hr to 72-96hr, where tenderness returned close to baseline levels at 120hr. N group tenderness also declined, similar to H group, from baseline to 24hr post-EIMD, however N group tenderness recovered from 24-96hr, returning close to baseline levels at 120hr. Muscle tenderness for both groups showed a similar recovery trend across time. Muscle tenderness during palpation was not significantly different between the H and N groups across time. Although not significantly different, muscle tenderness was greater for the H group compared to that of the N group at 48hr -120hr post-EIMD. Although not

significantly different, Figure 10 shows somewhat of a delay in recovery from muscle tenderness for the H group for all post-EIMD times compared to the N group.

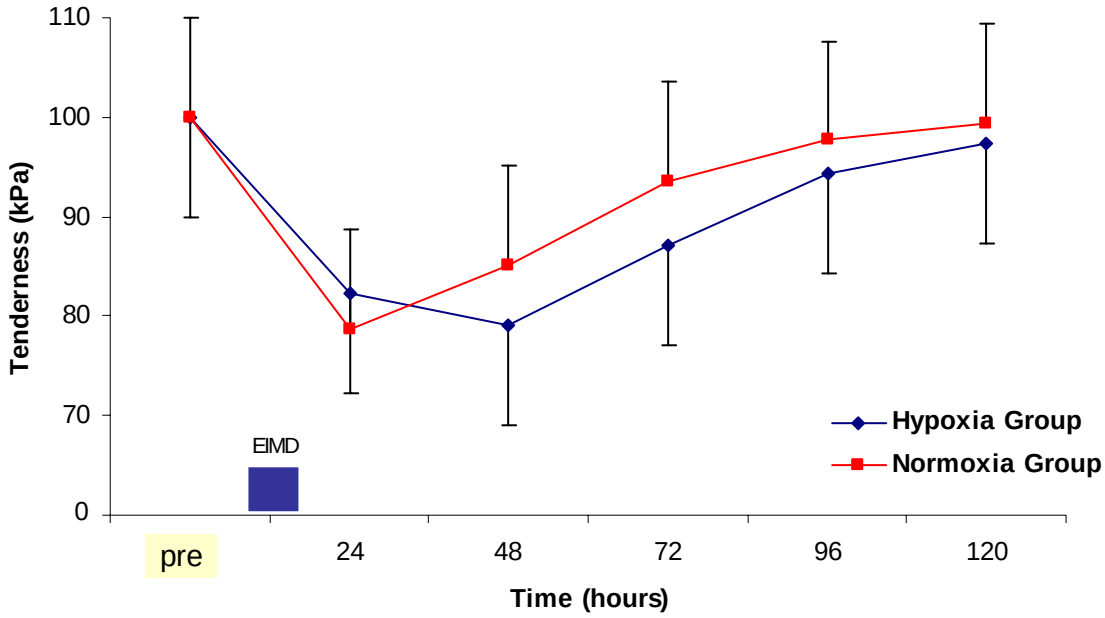


Figure 10. Mean tenderness across time for Hypoxia group (n=8) and Normoxia group (n=8).

4.7 Muscle Soreness

Figure 11 shows muscle soreness was significantly (p<0.05) different to baseline measures for the N and H groups at 24hr after EIMD. Muscle soreness peaked at 24hr post-EIMD in both the H and N groups, whilst both groups showed a similar trend across time. Muscle soreness recovery for the H group was delayed between 24-96hr compared to the N group delay between 24-72hr post-EIMD, whilst both groups returned to normal by 120hr post-EIMD. A significant difference was recorded between the H and N groups at 24hr post-EIMD (p<0.05) for muscle soreness. Although not significant, soreness was greater in

the H group compared with the N group at time point's 48-120hr post-EIMD. Perceived muscle soreness between the H and N groups showed an interaction between treatment and post-EIMD time ($p<0.05$).

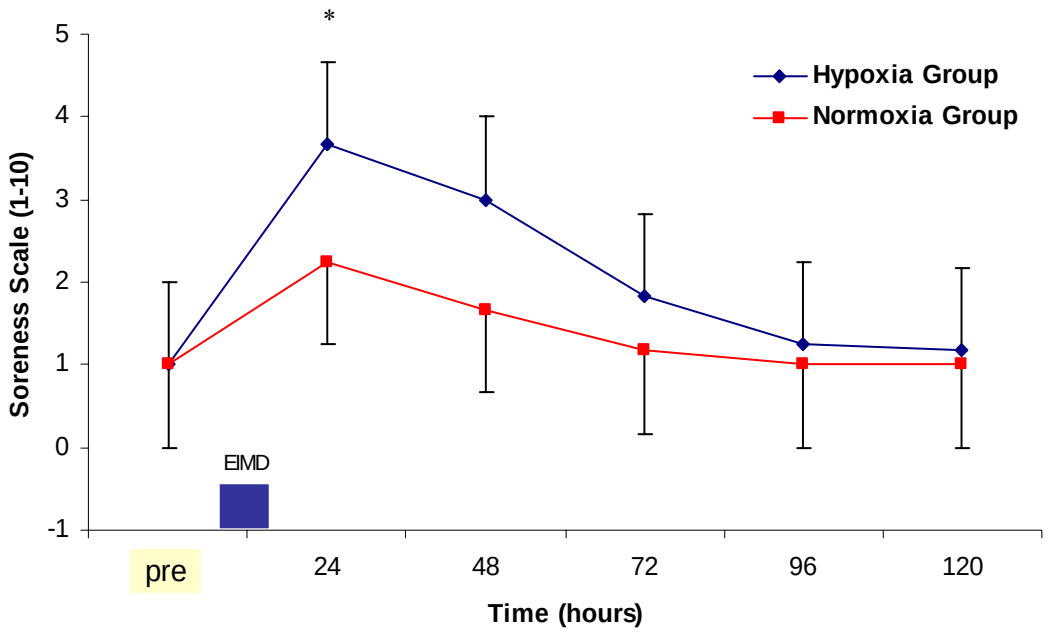


Figure 11. Mean soreness across time for Hypoxia group (n=8) and Normoxia group (n=8). * Significant ($p < 0.05$) difference between H and N groups at 24hr.

CHAPTER FIVE

DISCUSSION

The present study found that isometric strength at 35^{O} was significantly different between the H and N groups at 48hr post-EIMD ($p<0.05$). Isokinetic torque ($60^{O\text{-}sec}$, $120^{O\text{-}sec}$ and $240^{O\text{-}sec}$) results show there was no overall significant effect across time between the N and H groups. SJ showed that there was no significant difference between the groups at any of the measurement times, although SJ levels in the N group were greater compared to the H group. Plasma CK activity peaked at 48hr (N) and 96hr (H) post-EIMD. Plasma CK significantly increased ($p<0.05$) at 24hr after EIMD for both groups, this increase was sustained for the next 24-96hr, then started to gradually recover however, plasma CK was still elevated at 120hr after EIMD. Muscle tenderness, although not significantly different, was greater for the H group compared to that of the N group at 48hr -120hr post-EIMD, both groups showed a similar recovery trend across time. Muscle soreness peaked at 24hr post-EIMD in both the H and N groups, a significant difference was observed between the H and N groups at 24hr post-EIMD ($p<0.05$).

The purpose of the present study was to investigate the effects of a short-term (4hr) normobaric hypoxia insult on eccentric-EIMD recovery. Although previous studies (Ernsting & Sharp, 1978; Robergs, Quintana, Parker, & Frankel, 1998) have investigated hypoxia, to the author's knowledge, the effects of hypoxia on EIMD recovery has not been previously investigated. Anecdotal evidence suggests that exposure to a hypoxia insult would retard recovery from EIMD. A group of elite athletes who travel by air for a short time, west to east (3.5hr) for competition and return east to west (4hr) 2hr following

competition reported feeling lethargic, muscle soreness and tenderness and a loss of strength for a period of up to 72-96hr following the air-travel after competition compared to feelings of normality at 24-48hr when there was no air-travel after competition. The athletes wanted to know if the effects were a result of the physical, both internal and external, impact of competition the travel or a combination of a number of unknown factors. It was hypothesized that the recovery of isometric muscular strength, isokinetic peak torque, vertical jump, muscle soreness, muscle tenderness, and plasma CK activity would be significantly slower for the H group than the N group. However, the results showed there was not a significant difference between the H and N groups following EIMD. The results suggest that a 4hr hypoxia treatment after EIMD did not exacerbate muscle damage or slow the repair process.

5.1 Isokinetic Torque & Isometric Strength

The hypothesis that a hypoxic insult will delay muscle damage recovery was not supported by the isometric and isokinetic muscle tests. Although the N group was able to produce greater isometric strength and isokinetic torque measures and total work compared to the H group, this was to be expected seeing that the N group presented a marginally larger mass than the H group, and therefore likely possessed a greater lean muscle mass than the H group. Furthermore, H group was significantly older (yr) than the N group which suggests a potential for greater strength to be produced compared to the younger N group due to muscle maturity. However, the change in muscle strength was not significantly different between the H and N groups.

It has been well documented that high intensity unaccustomed exercise or exercise involving significant eccentric actions will result in reduced muscle strength lasting up to 5-

10 days post exercise (Armstrong, Warren & Warren,1991; Kuipers, 1994). The greatest disruption to the muscles ability to generate force is evident immediately following eccentric exercise, with muscle force slowly returning to normal over several days (Clarkson, Nosaka, & Braun, 1992). Talag (1972) assumed that strength decrements following eccentric muscle actions were due to pain which prevented maximal muscle activation. Furthermore, the loss of function following eccentric EIMD is associated with acute inflammation and DOMS (Smith, 1991). A loss of function implies an inability of the affected area to generate force. This loss of force is not completely understood but it is possible that a mechanical interference to the muscle was caused by swelling or a muscles reflex inhibition of the muscles experiencing pain which demonstrates a critical period for healing and the formation of new capillaries that are likely to be extremely fragile or the synthesis of new connective tissue (Peacock, 1984).

Newham et al., 1983 and Talag, 1973 investigated the time courses for development of soreness and for loss of strength and concluded there was little or no relationship between the two parameters. In support of these findings the present study revealed no significant change between strength measures or between groups over time. There was also no significant change in strength measures over time compared to baseline measures. However, soreness and tenderness were greater at 24hr post EIMD and remained greater than baseline measures at 120hr post EIMD. Clarkson & Tremblay, 1988; Newham et al., 1987 showed there was an immediate decrease in maximal force after eccentric exercise followed by a slow recovery whilst strength may remain depressed for seven days or longer (Neham et al., 1987). Interestingly, the present study revealed no significant change in strength immediately or over time compared to baseline measures, suggesting that the repeated bout of eccentric actions was not detrimental to the muscle to produce

force post-EIMD. As a result it is likely the EIMD protocol employed in the present study induced muscle damage to peripheral alterations which explains plasma CK increases. However, the damage to muscle tissue after EIMD may not have caused myofibrillar disruption, streaming or irregular z-lines, z-line material distributed throughout the sacromere, sarcolemma disruption, widening of A- and I-bands, displacement of organelles, increases in mitochondrial density. An interesting finding is that the N and H group strength measures were not significantly different suggesting that a hypoxic insult had no effect on EIMD recovery. Clearly some adaptation occurs in response to the initial microtrauma and subsequent healing which acts to protect the damaged muscle. It is clear that adaptations may last for a considerable period of time but it is not known how long after the initial eccentric exercise this adaptation occurs. Mechanical stress produced during EIMD actions may cause structural damage accompanied by a decrease in neuromuscular performance. This may suggest that if injury occurred only to the peripheral area of the muscle then motor units initially recruited initiate recruitment of additional units to produce the required forces. From the strength measures purported in the present study for the H and N groups it is obvious that the H treatment had no effect on the nervous or neurological system as the measures were not significantly different between the H and N groups or showed any significant decrement between baseline and post-EIMD testing times.

5.2 Vertical Jump (Squat Jump)

The present study hypothesized that recovery of SJ height after EIMD would be significantly different between the H and CON groups. The results of this study showed that SJ following EIMD was not significantly different between H and CON groups. The results also indicated that SJ height decline was greatest at 24hr for both the H and N

groups. However, the H group's SJ height did not return to baseline level by 120hr. Interestingly, SJ height for the N group returned to baseline levels by 48hr and progressively increased to 120hr post-EIMD. Previous studies (Eston et al., 1994; Eston et al., 1996; MacIntyre et al., 1996) investigated eccentric exercise, soreness and tenderness and presented evidence of muscle damage using eccentric torque and isometric strength. The present study was interested in the functional aspects associated with EIMD, including a modality specific to a performance. Therefore, SJ as a performance indicator was included to try and establish another muscle damage indicator.

The results indicate that neither, the N or H, insult had an effect on muscle function when performing the SJ. Strength loss after EIMD seems to be independent of the muscle action being performed. However, the impairment of muscle function, SJ, was not attenuated when the stretch-shortening cycle was employed during the vertical jump exercise. It is possible that the stretch shortening cycle used in the SJ was unaffected as the eccentric exercised muscle is characterised by an inability to generate high force and power, however the elastic energy may have been able to maintain its ability to produce force and power. Such functional outcomes are consistent with the proposition that type II fibres are selectively recruited or damaged during eccentric exercise. The ability to maintain SJ height, following EIMD, suggests strength loss is mainly due to peripheral alterations and that alteration of the excitation-contraction coupling was not involved. Presently, there is no clear explanation for either the N condition SJ height increase in recovery or the magnitude of the H condition SJ height decline, but this finding suggests that 4hr hypoxia had no effect on SJ height, and likely muscle damage.

5.3 Plasma CK Activity

In the present study, plasma CK activity in response to a short-term normobaric hypoxia insult following EIMD presented no significant difference between the H and N conditions (Figure 1). However, both groups showed a significant increase in CK activity after EIMD. Clarkson and Tremblay (1988) suggest that the muscle sacromeres are disrupted, misaligned, or a minor muscle tear occurs during eccentric actions causing a CK influx into the blood stream. This suggests that severe damage was produced in the exercised muscles during EIMD protocol of the present study as indicated by the increased CK measures at 24hr (Figure 1). It was hypothesized that CK activity for the H group would be significantly greater than the N group. The observed CK activity in the N group following EIMD showed that a mean peak response between 24-48hr post-EIMD, and supports Clarkson et al.'s (1987) findings. In comparison, the H group showed a mean peak at 96hr post-EIMD, similar to the finding of Schwane et. al., (1983a). Although there was no significant difference between these groups as hypothesized, it is possible the post-EIMD isokinetic and isometric testing may have interfered with EIMD recovery. Maximal efforts during the post-EIMD testing protocols may have caused further minor muscle damage in the H group as indicated by increased plasma CK levels at 72, 96 and 120hr post-EIMD, whilst isometric and isokinetic exercise performances may have been negatively affected. The delayed appearance of increased plasma CK activity that may have been caused by the post-isometric and isokinetic testing, and represent changes in sarcolemma integrity, focal necrosis and delayed muscle fibre regeneration.

Clarkson et. al. (1987) has shown that repeating eccentric exercise with the same limb results in a reduced CK response following a second bout similar to that demonstrated by the N group. However, concentric and isometric muscle actions following an EIMD

bout may cause a greater CK response, as concentric contractions require a greater metabolic flux than eccentric contractions. Another likely explanation for the finding of no significant differences in CK levels between groups is that 4hr of hypoxia is not a long enough period to influence the recovery process.

Following the process of inflammation, it is possible that normoxia affected recovery by limiting tissue edema and any possible potential for maintaining localized tissue pressure by minimizing the outflow of protein rich fluid from the plasma and its accumulation in the interstitial tissue (Armstrong, Warren, & Warren, 1991). Normoxia may also have inhibited the activation of the immune complexes, or a phagocytic challenge, neutrophils and macrophages as they release oxygen-derived free radicals and lysomal enzymes, which may have the potential to be detrimental during the inflammatory process (Armstrong et al., 1991). While O_2 depravation to the stressed fragile muscle fibers could delay a noxious stimulus, which is responsible for the inflammatory response and the pathological reactions such as muscle tissue necrosis, repair and regeneration (Armstrong, Warren & Warren, 1991), it is possible that oxygen treatment after EIMD leads to the production of free radical scavengers that terminates lipid peroxidation and therefore regulates the recovering environment.

5.4 Muscle Soreness and Tenderness

Clinical symptoms associated with EIMD are soreness; perceived pain whilst performing movements such as walking, referred to as pain in the muscle during movement (Miles & Clarkson, 1994). The results show the N group's perceived soreness returned to baseline levels between 48hr to 72hr, whilst the H group's perceived soreness remained greater than that of baseline level. The high tensions placed on muscles and tendons

associated with eccentric contractions may cause a disruption to the connective tissue(s) inflicting painful sensations involving the activation of pain afferents, most likely type being III and IV nerve fibers (Mense & Stahnke, 1983). The sensation of pain, or soreness, in skeletal muscle is transmitted by myelinated group III (A-delta) and unmyelinated group IV (C) afferent fibers (Mense, 1977). Chemical substance(s) are required for generating painful sensations, the most likely being prostaglandins of the E series (Bomalaski, Williamson & Lurier, 1983), which do not directly cause pain, but sensitize pain receptors. These pain receptors are more likely to be activated by chemical, mechanical or thermal stimuli (Smith, 1991). However, it is still unclear which mechanisms cause the sensation of soreness. Consequently, on the basis of this study, and previous studies (Bomalaski, Williamson & Lurier, 1983 and Smith, 1991) one must be careful in concluding that the combination of modalities or one specific method is important in the development of soreness during recovery from EIMD. Furthermore, chemical substances such as serotonin, histamine and potassium evoke an action potential on muscle group IV fibers providing a stimulus sufficient to increase perceived pain (Fock & Mense, 1976.). This concept receives some support from the present study which found strength levels after EIMD was greater in the N group than the H group and possibly reflects the perceived soreness levels and a reduced action potential.

From the results of the present study it is not possible to suggest why the H condition may have resulted in a greater delay in soreness recovery to that of the N condition, other than to consider the importance of and the extent to which the controlled intervention of a hypoxic insult may have on the nervous system. It is suggested further research should emphasize investigating more variables in a prospective study design. It is acknowledged that the present study design involved two groups of not the same

individuals and that any future studies should use a repeated measures design whereby the same subjects are tested on two different occasions in a counterbalanced crossover fashion with an appropriate washout period.

The eccentric EIMD protocol used in the present study produced tenderness in the N group (peaked at 24hr) and the H group (peaked at 48hr). However, Edwards Mills & Newham (1981) and Eston, Critchley & Baltzopoulos (1994), reported subject tenderness peaked 48hr after EIMD. It is not immediately apparent how the N or H condition affects the recovery from EIMD. The significant tenderness difference between the H and N condition observed at 48 and 72hr in the present study may reflect changes in intravascular volume. It is possible the H group tender muscles were spared compared to the N group, due to diminished muscle pump activation (McArdle, Katch & Katch, 1986, p329). It is therefore possible that any rise in resting blood flow (venous pooling) within the H group may be responsible for the observed muscle tenderness. Another possibility is that any increase in metabolite (calcium) accumulation within damaged muscle tissue, to abnormal levels, would result in a greater level of cell damage (McArdle, Katch & Katch, 1986, p329) and delay recovery from EIMD.

The effect of hypoxia on EIMD recovery has not been previously investigated therefore, it is not known how the damaged muscle tissue reacts or why muscle-tenderness maybe affected by a mild hypoxic insult. One possible consideration is the biomechanical pathway responsible for cellular respiration. Respiratory physiology focuses on four integrated stages involved in external respiration, pulmonary ventilation, gas diffusion across the respiratory membrane, the storage and transport of oxygen and carbon dioxide and the exchange of dissolved gases. Abnormalities affecting anyone of these stages will affect the gas concentrations of the interstitial fluids and cellular activity (Martini, 1988,

p834). If the oxygen content were to decline the affected tissue would become oxygen-starved and place severe limits on the metabolic activities of the affected area. If the oxygen supply were to be cut off completely, the condition of anoxia results and necrosis of the damaged muscle tissue occurs very quickly (Martin, 1988, p834).

The ability to maintain cellular activity is an integral process during muscle tissue damage recovery. If the damaged muscle tissue is starved of oxygen, limiting the metabolic activity of that area, the muscle tenderness differences observed between groups in the present study maybe explained. The quantity of oxygen available to damaged muscle tissue seems to be a contentious issue and requires further research into a number of unanswered questions.

5.5 Inflammatory Response

Eccentric or unaccustomed exercise may result in temporary and repairable damage to skeletal muscle raging from focal disruption and ultrastructural damage to individual fibres of the muscle. When active muscle is stretched, damage can be caused to the myofibrils within the muscle, connective tissue linking adjacent myofribils, the basil lamina adjacent to plasma membranes, the plasma membrane of the myofibre, sarcomeres and sarcoplasmic reticulum, or a combination (Friden, Sjostrom & Ekblom, 1983; Stauber, 1989). The immune system protects the body from invasion by creating local barriers and inflammation. The inflammatory response is to protect the tissues of the body to irritation or injury. It may be acute or chronic with signs of redness, heat, swelling, pain and a loss of function. The process begins with a brief increase in vascular permeability. Then a prolonged period consisting of a sustained increase in vascular permeability, excudation of fluids from the vessels, clustering of leukocytes along the vessel walls, phagocytosis of

microorganisms, deposition of fibrin in the vessel, disposal of the accumulated debris by macrophages and finally the migration of fibroblasts to the area and the development of new cells. The severity, timing and local character of any inflammatory response depends on the cause, the area affected and the condition of the host (Mosby's 1990, p 620). It is generally considered that a group of cytokines, including interleukin-1, interferon, interleukin-2, interleukin-6 and tumour necrosis factor mediate the inflammatory process (Imura, Fukata & Mori, 1991). Although these cytokins were not measured in the present study there was no evidence of swelling, redness or associated loss of force which accompanies inflammatory response to injury. Based on the present study results, it is proposed that the eccentric exercise did not cause an inflammatory response. Although plasma CK measures increased they were not elevated as expected after EIMD. Damage to focal areas of muscle fibres with histological examination of the affected muscle shows myofibrillar disruption, streaming or irregular Z-lines, Z-line material distributed throughout the sacromere, sarcolemma disruption, widening of A and I-bands, displacement of organelles, increases in mitochondrial density and in the content of cyto-skeletal and myofibrillar proteins (Kuipers et al., 1991). Although the present study did not measure these variables it is proposed that damage to the focal area of the affected muscle may have presented one of the histological examination measures as there was evidence of soreness and tenderness. The increased plasma CK in the circulation after exercise reflects muscle damage however the associated decrement in force was not seen suggesting the slightly damaged fibres were able to recruit more fibres to perform exercises after EIMD. The mechanical stress mechanism has been proposed to account for the damage caused by the shear forces produced during eccentric exercise. However, the metabolic stress

mechanism may have disturbed normal metabolic metabolism provoked by exhaustive endurance exercise such as eccentric actions.

Following EIMD, a hypoxic insult was expected to reduce oxygen utilisation and overwhelm the capacity of the defence systems to scavenge reactive oxygen species and slow the oxidative damage to cellular components. Damage to the plasma membrane may lead to a loss in cell viability and possibly to cell necrosis over a longer period. Therefore, the immune response would involve a decrease in the rate of differentiation of monocytes and a decreased rate of transfer of mature monocytes from the bone marrow to the circulation. The possibility that hypoxic insult would be responsible for the delayed onset of neutrophils and macrophages invading skeletal muscle was the subject of debate. Neutrophils and macrophages which may extend to the inflammatory consequences of eccentric exercise and damaged muscle tissue by the release of reactive oxygen and nitrogen species, and cytokines is delayed. The pro-inflammatory cytokines interleukin (IL)-1β and tumor necrosis factor (TNF)-α would also be expressed within skeletal muscle over a longer period of time also delaying the inflammatory process and muscle damage recovery. IL-1β and TNF-α influence in initiating the breakdown of damaged muscle tissue would be less significant. Other cytokines such as IL-6 and transforming growth factor (TGF)-β1, and inflammatory antigens such as leukemia inhibitory factor (LIF) and hypoxia inducible factor (HIF)-1β also expressed in skeletal muscle in the days following eccentric exercise would appear less responsive.

Inflammatory responses in muscle after eccentric exercise, the measurement of plasma CK allows for the comparison of responses to different types of post- EIMD measures. A large volume of data is available relating to changes in plasma CK and force production. Evidence suggests that changes were not significantly different. These non-

significant differences may reflect the influence of exercise intensities and muscle damage versus the effect of hypoxic insult. In addition to the muscle damage and functional activity alterations in damaged muscle tissue was minimal. There is no clear effect of a short term hypoxic insult after eccentric exercise on muscle damage recovery. This disparity might be due to differences in the type of eccentric exercise, exposure to previous eccentric exercise, as well as the methods used to measure soreness, tenderness and functional activity.

Oxidative phosphorylation is the most important source of energy, oxygen capture, convective and diffusive oxygen transport, as well as the final intracellular oxygen utilisation within the mitochondria represents highly refined mechanisms, maintained by a number of physiological control systems. It is possible that any inflammatory process interfering with the delivery of oxygen to tissue will ultimately lead to an impairment of cellular energy production. Generally, cellular hypoxia may result from either reduced oxygen uptake (hypoxic hypoxia), reduced convective and diffusive oxygen transport (circulatory and anemic hypoxia), impaired oxygen consumption (histotoxic hypoxia), or a combination of these states. At present there is no definitive explanation which links the sequence of physiological responses (soreness, tenderness and decrement in force production) after EIMD.

CHAPTER SIX

CONCLUSION

The results of the present study showed that a 4hr hypoxic treatment after EIMD did not exacerbate muscle damage (MVC, plasma CK, soreness, tenderness, SJ) or retard the recovery process. Therefore, these results suggest that EIMD recovery is not affected by a hypoxic treatment. The non-significant difference evident between the H and N groups in the present study suggest athletes suffering from EIMD are reasonably assured that a short, 4hr, air-flight (H insult) will not increase EIMD recovery time compared to N insult.

A weakness of this study was in the subject numbers used and the fact that two groups of different subjects were used. Also, the development and recovery of any injury is influenced by many intrinsic and extrinsic factors. The fact that only a hypoxic condition influence on EIMD recovery was examined could also be considered a limitation of this study.

It is recommended that further research into the effects of a hypoxia insult following exercise on muscle damage recovery include protocols that include two subject groups which participate in both a normoxic and hypoxic insult. Other possible reasons for delays in EIMD recovery following air travel include cramped and static positions, and perhaps further studies should also attempt to examine this.

CHAPTER SEVEN

REFERENCES

Ahlborg, G., Hagenfeldt, L., & Wahren, J. (1975). Substrate utilization by the inactive leg during one-leg or arm exercise. Journal of Applied Physiology, 39(5) 718-723.

Appell, H.J., Soares, J.M.C., & Duarte, J.A.R. (1992). Exercise, muscle damage and fatigue. Sports Medicine, 13, 108-115.

Apple, F.S., Hellsten, Y., & Clarkson, P.M. (1988). Early detection of skeletal muscle injury by assay of creatine kinase MM isoforms in serum after acute exercise. Clinical Chemistry, 34, 1102-1104.

Armstrong, R.B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Medicine and Science in Sports and Exercise, 16 , 529-538.

Armstrong, R.B., Warren, G.L., & Warren, G.L. (1991). Mechanisms of exercise-induced muscle fibre injury. Sports Medicine, 12(3), 184-207.

Balnave, C.D., & Allen, D.G. (1995). Intracellular calcium and force in single mouse muscle fibres following repeated contractions with stretch. Journal of Physiology, 488(1), 25-36.

Bennett, M., Best, T., Babul, S., Taunton, J., Lepawsky, M., & Bennett, M. (2005). Hyperbaric oxygen therapy for delayed onset muscle soreness and closed soft tissue injury. Cochrane Database Systems Review, 19(4), CD004713.

Berne, R.M., & Levy, M.N. (1964). Heart. Annual Review of Physiology, 26, 153-186.

Bomalaski, J.S., Williamson, P.K., & Zurier, R.B. (1983). Prostaglandins and the inflammatory response. Clinics in Laboratory Medicine, 64(4), 1-8.

Bouachour, G., Cronier, P., Gouello, J.P., Toulemonde, J.L, Talha, A., & Alquier, P. (1996). Hyperbaric oxygen therapy in the management of crush injuries: a randomized double-blind placebo-controlled clinical trial. Journal of Trauma, 41(2), 333-339.

Brooks, S.V., Zerba, E., & Faulkner, J.A. (1995). Injury to muscle firbres after single stretches of passive and maximally stimulated muscles in mice. Journal of Physiology, 488(2), 459-469.

Byrnes, W.C., Clarkson, P.M., White, J.S., Hseih, S.S., Frykman, P.N., & Maughan, R.J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. Journal of Applied Physiology, 59, 710-715.

Cianci, P., & Sato, R. (1994). Adjunctive hyperbaric oxygen therapy in the treatment of thermal burns: a review. Burns, 20(1), 5-14.

Clarkson, P.M., Apple, F.S., Byrnes, W.C., McCormick, K.M., & Triffletti, P. (1987). Creatine kinase isoforms following isometric exercise. Muscle and Nerve, 10, 41-44.

Clarkson, P.M., Byrnes, W.C., Gillisson, E., & Harper, E. (1987). Adaptation to exercise-induced muscle damage. Clinical Science, 73, 383-386.

Clarkson, P.M., Litchfield, P., Graves, J., Kirwan, A., & Byrnes, W.C. (1985). Serum creatine kinase activity following forearm flexion isometric exercise. European Journal of Applied Physiology, 53, 368-371.

Clarkson, P.M., Nosaka, K., & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. Medicine and Science in Sports and Exercise, 24, 512-520.

Clarkson, P.M., & Tremblay, I. (1988). Exercise-induced muscle damage, repair, and adaptation in humans. Journal of Applied Physiology, 65(1), 1-6.

Conrad, B., Benecke, R., & Goehmann, M. (1983). Premovement silent period in fast movement initiation. . Experimental Brain Research, 51(2), 310-313.

Ebbling, C.B., & Clarkson, P.M. (1989), Exercise-induced muscle damage and adaptation. Sports Medicine, 7, 207-304.

Ebbling, C.B., & Clarkson, P.M. (1990). Muscle adaptation prior to recovery following eccentric exercise. European Journal of Applied Physiology, 60, 26-31.

Edwards, R.H.T., Mills, K.R., & Newham, D.J. (1981). Measurement of severity and distribution of experimental muscle tenderness. Journal of Physiology, 317, 1-2P.

Ernsting, J. (1963). The effect of brief profound hypoxia upon the arterial and venous oxygen tension in man. Journal of Physiology, 169, 292-311.

Ernsting, J. (1978). Prevention of hypoxia-acceptable compromises. Aviation and Space and Environmental Medicine, 49(3), 495-502.

Ernsting, J., & Sharp, G.R. (1978). Hypoxia and hyperventilation. In G. Dhenin (Ed.), Aviation Medicine (Vol 1., pp. 45-59). London: Tri-Med Books.

Eston, R.G., Critchley, N., & Baltzopoulos, V. (1994). Delayed-onset muscle soreness, strength loss characteristics and creatine kinase activity follwing uphill and downhill running. Journal of Sports Science, 12, 135.

Eston, R., Finney, S., Baker, S., & Baltzopoulos, V. (1996). Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. Journal of Sports Science, 14, 291-299.

Ferretti, G., Moia, C., Thomet, J., & Kayser. (1997). The decrease of maximal oxygen consumption during hypoxia in man: a mirror image of the oxygen equilibrium curve. Journal of Physiology, 498(1), 231-237.

Fock, S., & Mense, S. (1976). Excitatory effects of 5-hydroxytryptamine, histamine and potassium ions on muscular group IV afferent units: a comparison with bradykinin. Brain Research, 105(3), 459-469.

Franklin, M., Currier, D & Franklin, R. (1991). The effect of one session of muscle soreness-inducing weight lifting exercise on WBC count, serum creatine kinase, and plasma volume. Journal of Orthopaedic and Sports Physical Therapy, 13(6), 316-322.

Friden, J., Sjostrom, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. International Journal of Sports Medicine, 4, 170-176.

Fried, W., & Morley, C. (1985). Effects of androgenic steroids on erythropoiesis. Steriods, 46(4-5), 799-826.

Cable, G.G., (2003). In-flight hypoxia incidents in military aircraft: causes and implications for training. Aviation and Space Environmental Medicine, 74(2), 169-172.

Garcia-Covarrubias, L., McSwain, N.E. Jr., Van Meter, K., Bell, R.M. (2005). Adjuvant hyperbaric oxygen therapy in the management of crush injury and traumatic ischemia: an evidence-based approach. American Surgery, 71(2), 144-151.

Groger, M., Speit, G., Radermacher, P., & Muth, C.M. (2005). Interaction of hyperbaric oxygen, nitric oxide, and heme oxygenase on DNA strand breaks in vivo. Mutate Research, 572(1-2),167-172.

Harding, R.M., & Mills, F.J. (1983). Problems of altitude: hypoxia and hyperventilation. British Medical Journal, 286, 1408-1410.

Harrison, B.C., Robinson, D., Davison, B.J., Foley, B., Seda, E., & Byrnes, W.C. (2001). Treatment of exercise-induced muscle injury via hyperbaric oxygen therapy. Medicine and Science in Sports and Exercise, 33(1), 36-42.

Hunter, K.D., & Faulkner, J.A. (1997). Pliometric contraction-induced injury of mouse skeletal muscle: effect of initial length. Journal of Applied Physiology, 82(1), 278-283.

Holloszy, J.O., & Coyle, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology, 56(4), 831-838.

Imura, H., Fukata, J., & Mori, T. (1991). Cytokines and endocrine function: an interaction between the immune and neuroendocrine systems. Clinical Endocrinology, 35, 107-115.

Ingalls, C.P., Warren, G.L., Williams, J.H., Ward, C.W., & Armstrong, R.B. (1998). E-C coupling failure in mouse EDL muscle after in vivo eccentric contractions. Journal of Applied Physiology, 85(1), 58-67.

Ishii, Y., Deie, M., Adachi, N., Yasunaga, Y., Sharman, P., Miyanaga, Y., & Ochi, M. (2005). Hyperbaric oxygen as an adjuvant for athletes. Sports Medicine, 35(9), 739-46.

Jones, D.A., Newham, D.J., & Torgan, C. (1989). Mechanical influences on long-lasting human muscle fatigue and delayed-onset pain. Journal of Physiology, 412, 415-427.

Jones, D. A., Newham, D. J., Round, J. M., & Tolfree, S. E. J. (1986). Experimental human muscle damage: morphorlogical changes in relation to other indices of damage. Journal of Physiology, 375, 435-448.

Jones, N.L., Sutton, J.R., Taylor, R., & Toews, C.J. (1977). Effect of pH on cardiorespiratory and metabolic responses to exercise. Journal of Applied Physiology, 43(6), 959-964.

Kerr, F.W., & Wilson, P.R. (1978). Pain. Annual Review of Neuroscience, 1, 83-102.

Knaupp, W., Khilnani, S., Sherwood, J., Scharf, S., & Steinberg, H. (1992). Erythropoietin response to acute normobaric hypoxia in humans. Journal of Applied Physiology, 73(3), 837-840.

Kuipers, H., Jannsen, G.M., Bosman, F., Frederik, P.M., & Geurten, P. (1991). Structural and ultrastructural changes in skeletal muscle associated with long-distance training and running. International Journal of Sports Medicine, 10(3), 156-159.

Kuipers, H. (1994). Exercise-induced muscle damage. International Journal of Sports Medicine, 15, 132-135.

Lieber, R.L., Woodburn, T.M., & Friden, J. (1991). Muscle damage induced by eccentric contractions of 25% strain. Journal of Applied Physiology, 70(6), 2498-2507.

Marieb, E.N. (1992). Human Anatomy and Physiology (2nd ed). California: The Benjamin/Cummings Publishing Company, Inc.

Martini, F.H. (1998). Fundamentals of Anatomy and Physiology (4th ed). New Jersey: Prentice Hall.

MacDougall, J.D., Wenger, H.A., & Green, H.D. (1991). Physiological testing of the high-performance athlete (2nd ed). Champaign, IL: Human Kinetics.

MacIntyre, D., Reid, W., Lyster, D., Szasz, I., & McKenzie, D. (1996). Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise. Journal of Applied Physiology, 80(3), 1006-1013.

Maughan, R.J., Donnelly, A.E., Gleeson, M., Whiting, P.H., Walker, K.A., & Clough, P.J. (1989). Delayed-onset muscle damage and lipid peroxidation in man after a downhill run. Muscle and Nerve, 12, 332-336.

Mekjavic, I.B., Exner, J.A., Tesch, P.A., & Eiken, O. (2000). Hyperbaric oxygen therapy does not affect recovery from delayed onset muscle soreness. Medicine and Science in Sports and Exercise, 32(3), 558-563

Mense, S. (1977). Nervous outflow from skeletal muscle following chemical noxious stimulation. Journal of Physiology, 267(1), 75-88.

Mense, S., & Stahnke, M. (1983). Responses in muscle afferent fibers of slow conduction velocity to contractions and ischemia in the cat. Journal of Physiology, 342, 383-397.

Mense, S., & Schmidt, R.F. (1974). Activation group IV afferent units from muscle by algesic agents. Brain Research, 72(2), 305-310.

McArdle, W.D., Katch, F.I., & Katch, V.L. (1996). Exercise Physiology: energy, nutrition, and human performance (4th ed). Baltimore, MD: Williams & Wilkins.

McCartney, N., Heigenhauser, G.J., & Jones, N.L. (1983). Effects of pH on maximal power output and fatigue during short-term dynamic exercise. Journal of Applied Physiology, 51(1:1), 225-229.

McCully, K.K., & Faulkner, J.A. (1985). Injury to skeletal muscle fibres of mice following lengthening contractions. Journal of Applied Physiology, 59(1), 119-126.

McCully, K.K., & Faulkner, J.A. (1985). Characteristics of lengthening contractions associated with injury to skeletal muscle fibres. Journal of Applied Physiology, 61(1), 293-299.

Miles, M.P., & Clarkson, P.M. (1994). Exercise-induced muscle pain, soreness, and cramps. Journal of Sports Medicine and Physical Fitness, 34(3), 203-216.

Moritani, T. (1993). Neuromuscular adaptations during the acquisition of muscle strength, power and motor tasks. Journal of Biomechanics, 26(1), 95-107.

Morgan, D.L. (1990). New insights into the behavior of muscle during active lengthening. Biophysiology Journal, 57, 209-221.

Mosby's Medical, Nursing, and Allied Health Dictionary. (1990). (3rd ed.). St Louis, Missouri: Mosby.

Newham, D.J., Mills, K.R., Quigley, B.M., & Edwards, H.T. (1983). Pain and fatigue after concentric and eccentric muscle contractions. Clinical Science, 64, 55-62.

Newham, D.J., Jones, D.A., & Clarkson, P.M. (1987). Repeated high-force eccentric exercise: effects on muscle pain and damage. Journal of Applied Physiology, 63(4), 1381-1386.

Nosaka, K., & Clarkson, P.M. (1996). Changes in indicators of inflammation after eccentric exercise of the elbow flexors. . Medicine and Science in Sports and Exercise, 28, 953-961.

Nosaka, K., Clarkson, P.M., & Apple, F.S. (1992). Time course of serum protein changes after strenuous exercise of the forearm flexors. Journal of Laboratory and Clinical Medicine, 119, 119-183.

Nosaka, K., & Sakamoto, K. (2001). Effect of elbow joint angle on the magnitude of muscle damage to the elbow flexors. Medicine and Science in Sports and Exercise, 31(1), 22-29.

Nuttal, F.Q., & Jones, B. (1968). Creatine kinase and glutamic oxalacetic transaminase activity in serum: Kinetics of change with exercise and effects of physical conditioning. Journal of Laboratory and Clinical Medicine, 71, 847-854.

Nylander, G., Lewis, D., Nordstrom, H., Larsson, J. (1985). Reduction of postischemic edema with hyperbaric oxygen. Plastic Reconstruction Surgery, 76(4), 596-603.

Nylander, G., Nordstrom, H., Lewis, D., Larsson, J. (1987). Metabolic effects of hyperbaric oxygen in postischemic muscle. Plastic Reconstruction Surgery, 79(1), 91-97.

Peacock, E. E. (1984). Inflammation and cellular response to injury. In: Wound Repair (3rd Ed., pp. 1-59). Philadelphia, PA: W. B. Saunders.

Ren, X., Fatemian, M., & Robbins, P.A. (2000). Changes in respiratory control in humans induced by 8 h of hyperoxia. Journal of Applied Physiology, 89(2), 655-662.

Richardson, R., Noyszewski, E.A., Leigh, J.S., & Wagner, P.D. (1998). Lactate efflux from exercising human skeletal muscle: role of intracellular PO_2. Journal of Applied Physiology, 85, 627-634.

Robergs, R.A., Quintana, R., Parker, D.L., & Frankel, C.C. (1998). Multiple variables explain the variability in the decrement in $\dot{V}O_{2\ max}$ during acute hypobaric hypoxia. Medicine and Science in Sports and Exercise, 30(6), 869-879.

Rodenburg, J.P., Bar, P.R., & De Boer, R.W. (1993). Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. Journal of Applied Physiology, 74(6), 2976-2983.

Rutkove, S.B, Kothari, M.J., & Shefner, J.M. (1997). Nerve, muscle, and neuromuscular junction electrophysiology at high temperature. Muscle and Nerve, 20(4), 431-436.

Ryan, G.B., & Majno, G. (1977). Acute inflammation. American Journal of Pathology, 86, 185-264.

Schwane, J.A., Johnson, S.R., Vandenakker, C.B., & Armstrong, R.B. (1983). Delayed-onset muscular soreness and plasma CPK and LDH activities after downhill running. Medicine and Science in Sports and Exercise, 15(1), 51-56.

Smith, L.L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? Medicine and Science in Sports and Exercise, 23(5), 542-551.

Sueblinvong, T., Egtasaeng, N., & Sanguangrangsirikul, S. (2004). Hyperbaric oxygenation and blood lactate clearance: study in sixty male naval cadets. Journal of the Medical Association of Thailand, 87(2) 218-222.

Sutton, J.R., Jones, N.L., & Toews, C.J. (1981). Effects of PH on muscle glycolysis during exercise. Clinical Science, 61(3), 331-338.

Stacey, M.J. (1969). Free nerve endings in skeletal muscle of the cat. Journal of Anatomy, 105(2), 231-254.

Staples, J.R., Clement, D.B., Taunton, J.E., & McKenzie, D.C. (1999). Effects of hyperbaric oxygen on a human model of injury. American Journal of Sports Medicine, 27(5), 600-605.

Strauss, M.B., Hargens, A.R., Gershuni, D.H., Greenberg, D.A., Crenshaw, A.G., Hart, G.B., & Akeson, W.H. (1983). Reduction of skeletal muscle necrosis using intermittent hyperbaric oxygen in a model compartment syndrome. Journal of Bone and Joint Surgery of America, 65(5), 656-662.

Strauss, M.B., Hargens, A.R., Gershuni, D.H., Hart, G.B., & Akeson, W.H. (1986). Delayed use of hyperbaric oxygen for treatment of a model anterior compartment syndrome. Journal of Orthopedic Research, 4, 108-111.

Talag, T. (1973). Residual muscular soreness as influenced by concentric, eccentric and static contractions. Research Quarterly,12, 458-469.

Tiidus, P.M. (1997). Manual massage and recovery of muscle function following exercise: A literature review. Journal of Orthopedic and Sports Physical Therapy, 25(2), 107-112.

Tiidus, P.M. & Ianuzzo, C. D. (1983). Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. Medicine and Science in Sports and Exercise, 15, 461-465.

Warren, G.L., Hayes, D.A., Lowe, D.A., & Armstrong, R.B. (1993). Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. Journal of Physiology, 464 , 457-475.

Webster, A.L., Syrotuik, DG., Bell, G.J., Jones, R.L., & Hanstock, C.C. (2002). Effects of hyperbaric oxygen on recovery from exercise-induced muscle damage in humans. Clinical Journal of Sports Medicine, 12(3), 139-150.

Weiss, S.J. (1989). Tissue destruction by neutrophils. Journal of Medicine, 320, 365-376.

Appendix A

INFORMED CONSENT FORM

Project Title: **E*ffects of Hypoxia on Exercise Induced Muscle Damage.***

Purpose of the Investigation:
The purpose of this investigation is to determine if exposure to four hours of hypoxia, similar to that experienced during air-travel, delays the recovery from eccentric exercise-induced muscle damage. The recovery from muscle damage will be assessed by variables routinely used to diagnose muscle damage. It is hypothesized that exposure to hypoxia will prolong the recovery from muscle damage and cause larger and more sustained changes in all dependant variables over the ten-day study.

Procedures of the study:
This investigation will be conducted over an eight-day period at Edith Cowan University, Joondalup campus. Subjects participating in the investigation will be required to answer a health screening and risk stratification questionnaire (see Appendix B) as outlined by the American College of Sports Medicine.

The purposes of the pre-participation screening include:
- identify and exclude individuals with medical contraindications to exercise.
- identify individuals with disease symptoms and risk factors for disease development who should receive medical evaluation before starting an exercise program.
- identify persons with clinically significant disease considerations who should participate in a medically supervised exercise program.
- identify individuals with other special needs.

Once you, the potential subject, have been assessed and passed all pre-participation screening criteria you will then be invited to participate in the present investigation. Subject will then be randomly assigned to either test or control group.

Testing;
Testing will consist of seven visits to the laboratory. We may stop the test at any time because of signs of fatigue or observed changes indicating risks or extreme discomfort to the subject. It is important for you to realize that you may stop when you wish because of feelings of fatigue or any other discomfort.

Test 1 & 2;
Subject height (cm); body mass (Kg), test limb circumference (cm); Resting Heart Rate, Muscle soreness & tenderness; Creatine kinase; Isometric strength; Isokinetic torque & Vertical jump height (cm) will be measured and recorded as baseline measures.
You will perform an exercise warm-up; 5-minute cycle (ergometer) and 3-minute stretch on muscle groups of the leg; immediately followed by a isometric and isokinetic exercise test on subject dominant leg using a cybex 6000 dynamometer.

Test 3;
Subjects will be requested to perform an exercise warm-up (Test 1 warm-up), followed by an intermittent eccentric action of the knee extensor muscle group. All subjects will be requested to perform 10 reps x 6 sets of eccentric contractions, of the dominant leg at the angle from 0^0 to 90^0. Subjects will eccentric actions (extension/flexion) at a rate of 9^0 per second, with a 20-second inter-trail rest, the six sets will be separated by 2 minutes of rest. Immediately following the eccentric exercise protocol, a cool down exercise protocol (Test 1 warm-up, at a lower intensity) will be performed.

Immediately following the cool down, subject will be exposed to either normobaric hypoxia condition (test group) or normal ambient condition (control group) protocol. Subject in the test group will be requested to sit in an isolated room (heat chamber, SOBSS laboratory) and breath a medical grade gas; oxygen (15.37%) and nitrogen (84.63%) mild hypoxia, this gas will be pumped from a tissot tank into the isolated room. Subject in the control group will be requested to sit in a, different but similar, isolated room and breath normal ambient air.

Tests 4-8;
Subject will be requested to visit the laboratory on 5 different (24, 48, 72, 96 &120 Hr post eccentric exercise protocol) occasions. Subjects variables to be measured; limb circumference (cm); Muscle soreness & tenderness; Creatine kinase levels; Isometric strength; Isokinetic torque & Vertical jump height (cm) to evaluate recovery and compare post-exercise measures to baseline measures.

Subject risks and discomforts;
Subject is likely to experience leg soreness, tenderness and stiffness, in the thigh and gluteal region, in the days following the eccentric exercise protocol. However, this should dissipate within 5-7 days post exercise. Every effort will be made to minimize the risks of abnormal blood pressure, fainting, heart attack, stroke, or death by evaluation of preliminary information relating to your health and fitness and by observation during testing. Emergency equipment and trained personal are available to deal with unusual situations that may arise.

- The investigator(s) ask that you **do not** make any changes to your **daily activities**, **eating plan** or **take any medication, anti-inflammation drug(s**) or similar **as this may influence results**.

Responsibilities of the subject;
The information you, the subject, posses about your health status or previous experiences of unusual feelings with physical effort may affect the safety and value of your exercise test. Your prompt reporting of feelings with effort during exercise test itself is also of great importance. You are responsible for fully disclosing such information when requested by the researcher(s).

Benefits to the subject;
The results and experiences obtained from the exercise test and participation in the present investigation may assist you in understanding recovery methods and recovery times

following exercise-induced muscle damage and the effects of hypoxia (air-travel) on muscle damage recovery. Information gained may provide the subject with periodization strategies for training following competition(s).

Inquiries;
Questions concerning this investigation may be directed to:

Trevor Farr BSc. (Hons); MSc. candidate; email: [redacted]

Dr Fiona Naumann Supervisor; email: f.naumann@ecu.edu.au
9400 5012

Freedom of consent;
Your permission to perform this exercise test is voluntary. You are free to stop the test at any point, if you so desire, and you may withdraw at any time for any reason.
All information will be secured under lock and key by the researchers. Data entry and analysis of that data will not include any name or information that may identify or link an individual as a subject.

I agree that the research data gathered for this study may be published provided I am not identifiable.

I (the participant) have read this form, and I understand the test procedures that I will perform and the associated risks and discomforts. Knowing these risks and discomforts and having had an opportunity to ask questions that have been answered to my satisfaction. I agree to participate in this research project.

Signature of Participant: _____ Date:_____:_____:_____

Signature of Investigator: **Trevor Farr BSc (Hons);**

_____ Date: ____:_____:_____

Appendix B

MEDICAL QUESTIONNAIRE

The following questionnaire is designed to establish a background of your medical history, and identify any injury or illness that may influence your testing or performance. Please answer all questions as accurately as possible, if you are unsure about anything please ask investigator(s) (see Appendix, p. 3 Inquiries). All information provided is strictly confidential.

Personal Details

Name: _____

DOB: _____:_____:_____

Height: _____cm Body Mass: _____Kg

Medical History

Have you ever had, or do you currently have any of the following?

If you answer "YES" please give details

High or abnormal blood pressure YN

_____

High cholesterol YN

_____

Rheumatic fever YN

_____

Heart abnormalities YN

_____

Asthma YN _____

Diabetes YN

_____

Epilepsy YN

_____

Back pain YN

_____

Neck pain YN

_____

Severe allergies YN _____

Any infectious diseases YN _____

If you answered YES please give details

Are you currently on any medication(s)? Y N

_____

Have you had the flu in the last two weeks? Y N

_____

Have you recently had any injuries or accidents? Y N

_____

Do you have a recurring muscle or joint injury? Y N

_____

Suffer any condition(s) not previously mentioned which may affect your exercise performance? Y N

_____

Family History

Are there any of the following known exist in your family?

If you answer "YES" please give details

Cardiac disease Y N

_____

Pulmonary disease Y N

_____

Stroke Y N

_____

Lifestyle Habits

Do you exercise regularly? Y N
If YES how many hours per week? _____

Do you smoke tobacco or any
other nicotine products? Y N
If YES, please indicate how many per day _____

Do you consume alcohol? Y N
If YES how many standard drinks
per week? _____

Do you consume tea or coffee? Y N
If YES how many cups per day? _____

Declaration

I acknowledge that the information provided on this form, is to the best of my knowledge, a true and accurate indication of my current state of health.

Name: _____ Date: _____:_____:_____

Signature: _____

Witness: Trevor Farr Date: _____:_____:_____

Signature: _____

Appendix C

Rating Muscle Soreness

Scale: 1 = normal, 10 = very, very sore

1	-	Normal
2	-	
3	-	Somewhat Sore
4	-	
5	-	Moderately Sore
6	-	
7	-	
8	-	Very Sore
9	-	
10	-	Very, Very Sore

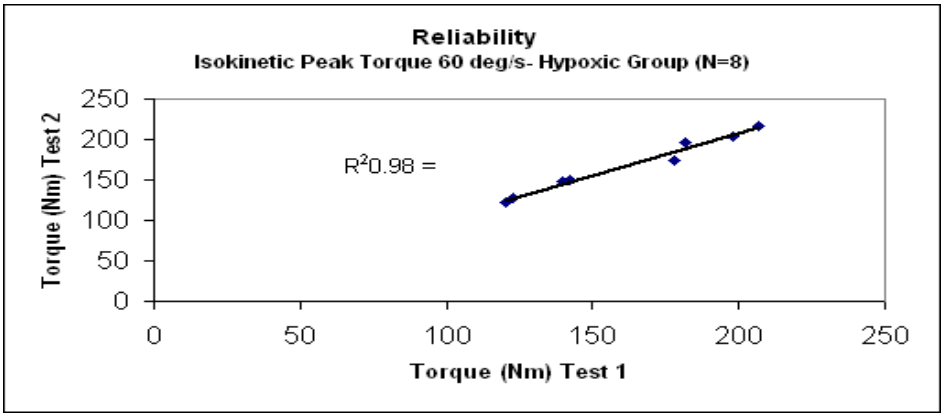
Adapted from Clarkson, P.M., & Tremblay, I. (1988). Exercise-induced muscle damage, repair, and adaptation in humans. Journal of Applied Physiology, 65 (1), 1-6.

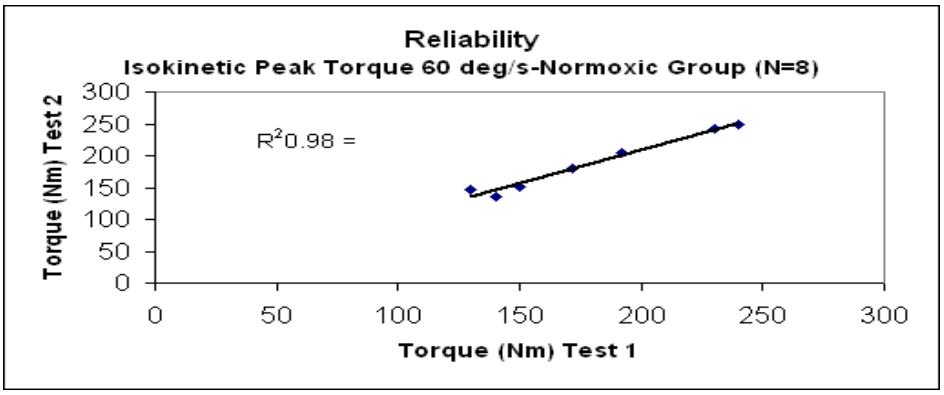
Appendix D

Reliability, Raw and Normalised Data

Isokinetic Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 60 deg/s

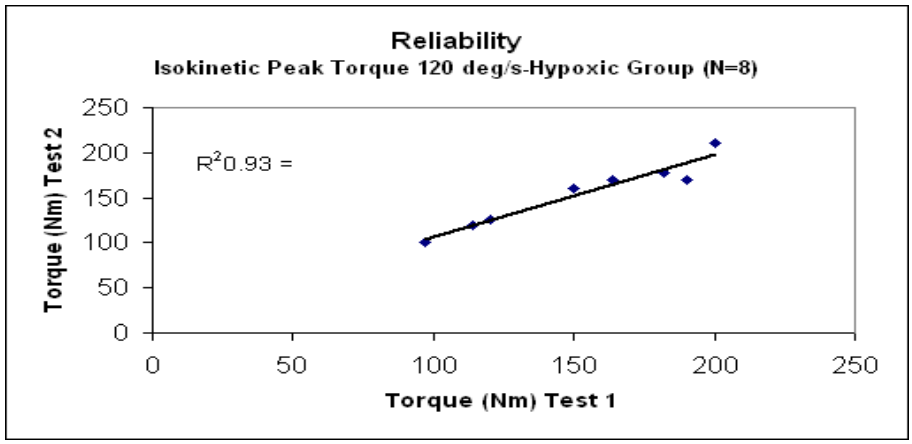
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	182	197	15	192	204	12
2	207	217	10	172	180	8
3	178	174	-4	230	242	12
4	140	148	8	150	152	2
5	123	127	4	192	205	13
6	120	122	2	130	146	16
7	198	204	6	140	136	-4
8	142	150	8	240	250	10
Mean	161.25	167.38	-6.13	180.75	189.38	-8.63
SD	34.10	36.04	5.67	40.44	43.29	6.57
SEM	12.06	12.74	2.00	14.30	15.31	2.32
ME			4.01			4.64
V			2.44			2.51
Correlation coefficient (*r*)			-3.06			-3.71

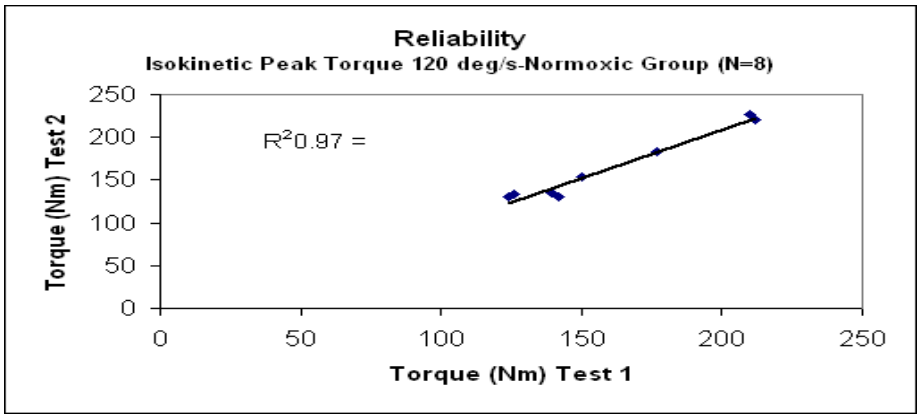




Isokinetic Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 120 deg/s

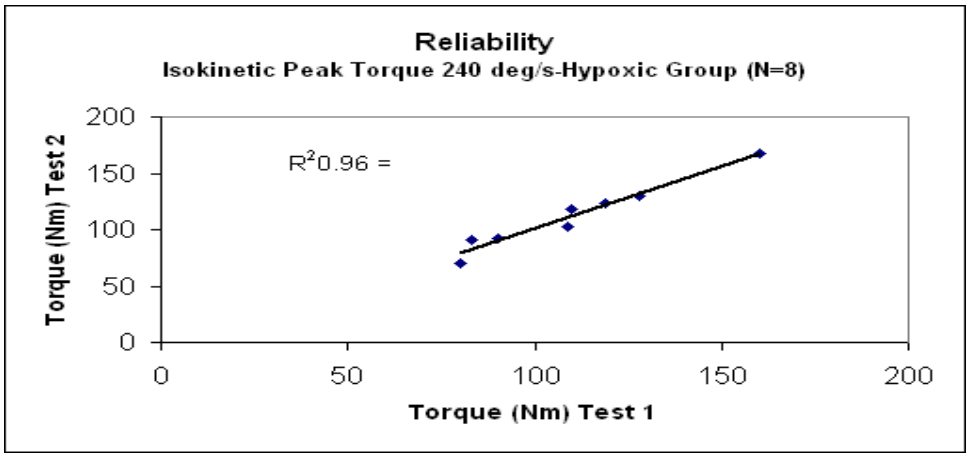
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	150	160	10	124	130	6
2	190	170	-20	177	183	6
3	182	178	-4	210	226	16
4	120	126	6	139	135	-4
5	114	120	6	150	154	4
6	97	101	4	126	133	7
7	200	210	10	142	130	-12
8	164	170	6	212	220	8
Mean	152.13	154.38	-2.25	160.00	163.88	-3.88
SD	38.34	35.87	10.00	35.48	40.63	8.43
SEM	13.55	12.68	3.53	12.54	14.36	2.98
ME			7.07			5.96
V			4.61			3.68
Correlation coefficient (*r)*			-0.64			-1.30

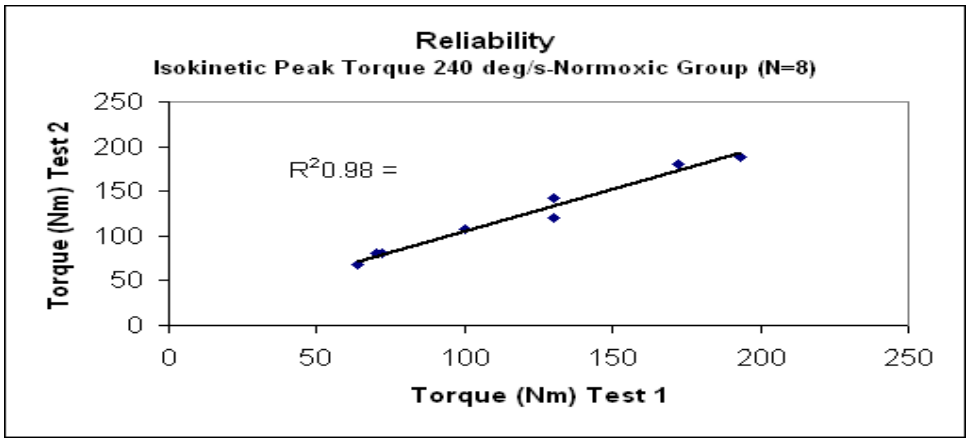




Isokinetic Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 240 deg/s

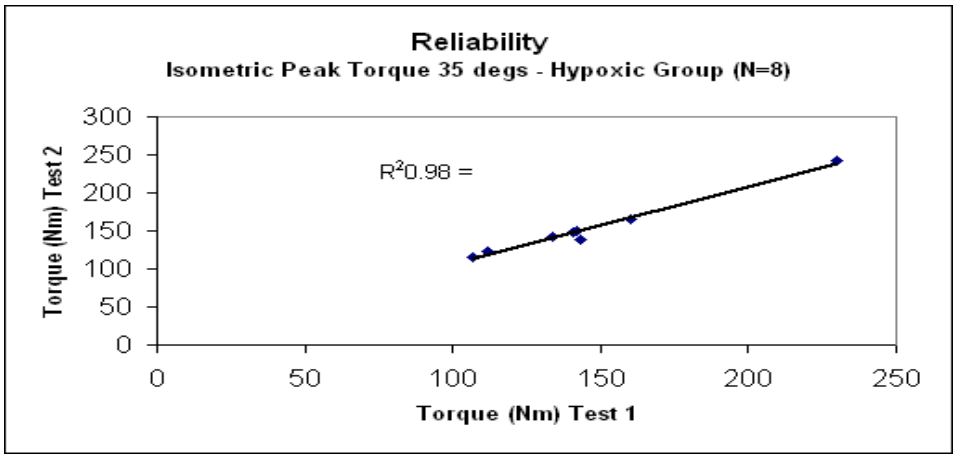
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	109	103	-6	72	80	8
2	128	130	2	130	142	12
3	119	123	4	193	189	-4
4	90	92	2	64	68	4
5	83	91	8	130	120	-10
6	80	70	-10	100	108	8
7	160	168	8	70	80	10
8	110	118	8	172	180	8
Mean	109.88	111.88	-2.00	116.38	120.88	-4.50
SD	26.55	30.01	6.76	48.50	46.12	7.62
SEM	9.39	10.61	2.39	17.15	16.30	2.69
ME			4.78			5.39
V			4.31			4.54
Correlation coefficient (*r)*			-0.84			-1.67

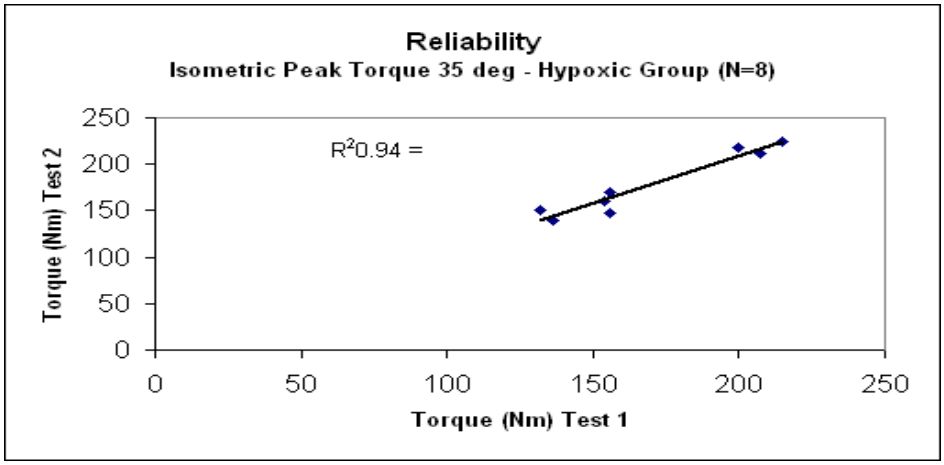




Isometric Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 35 deg

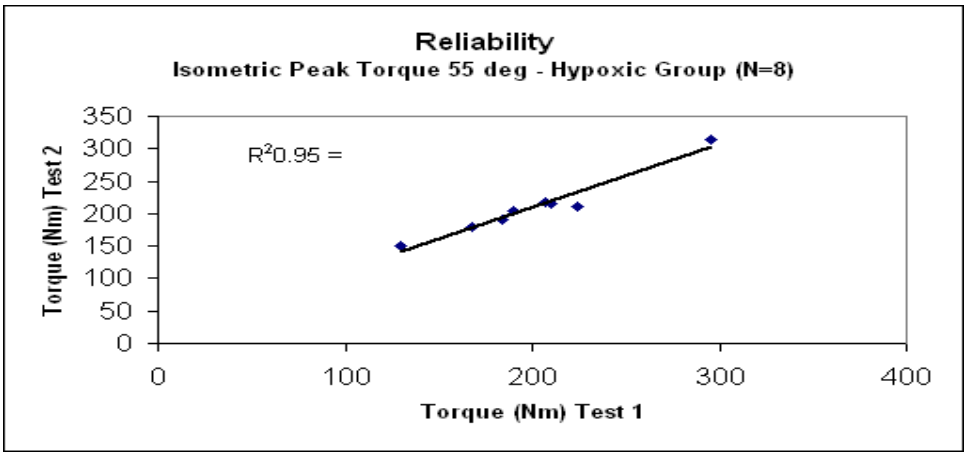
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	143	139	-4	215	225	10
2	142	150	8	156	170	14
3	112	124	12	207	211	4
4	107	115	8	136	140	4
5	141	149	8	156	148	-8
6	134	142	8	154	160	6
7	230	242	12	132	150	18
8	160	166	6	200	218	18
Mean	146.13	153.38	-7.25	169.50	177.75	-8.25
SD	38.03	39.13	5.01	32.82	34.67	8.71
SEM	13.44	13.83	1.77	11.60	12.26	3.08
ME			3.54			6.16
V			2.36			3.55
Correlation coefficient (*r)*			-4.10			-2.68

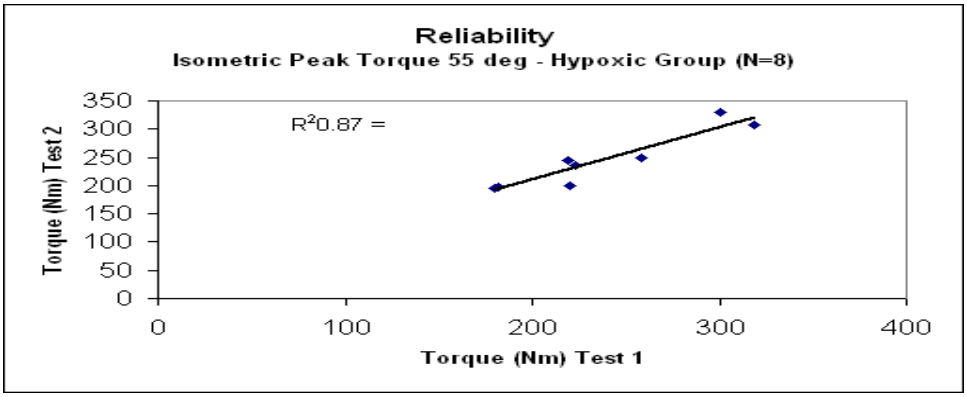




Isometric Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 55 deg

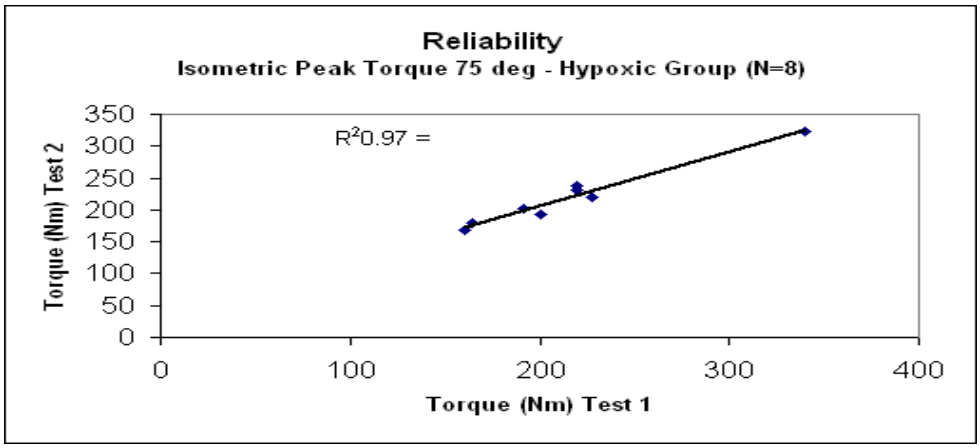
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	224	212	-12	319	307	-12
2	207	217	10	223	236	13
3	190	204	14	300	330	30
4	130	150	20	180	196	16
5	168	180	12	219	245	26
6	184	190	6	182	198	16
7	295	315	20	220	200	-20
8	210	216	6	258	250	-8
Mean	201.00	210.50	-9.50	237.63	245.25	-7.63
SD	47.84	47.88	10.24	51.01	50.44	18.52
SEM	16.91	16.93	3.62	18.03	17.83	6.55
ME			7.24			13.09
V			3.52			5.42
Correlation coefficient (*r)*			-2.62			-1.16

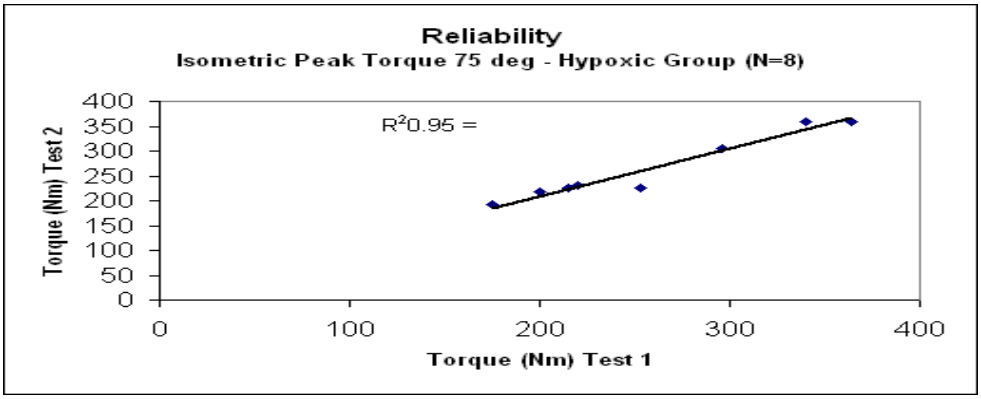




Isometric Peak Torque (Nm) for Hypoxic Group and Normoxic Group at 75 deg

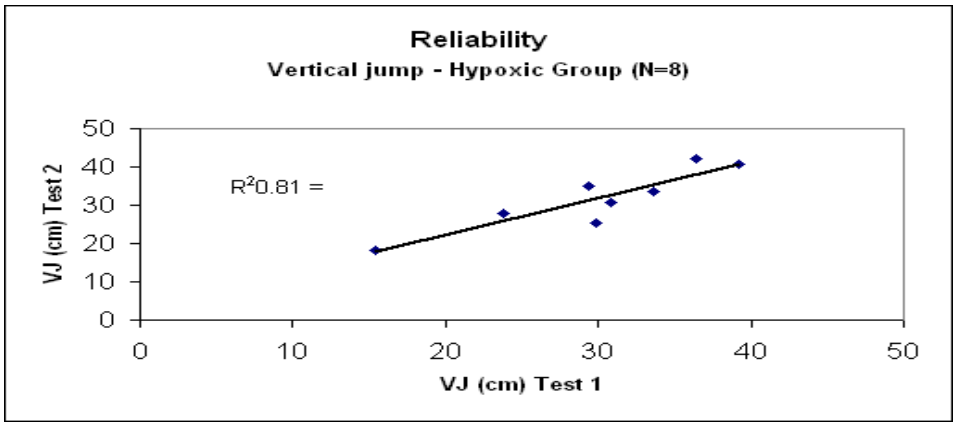
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	220	238	18	340	360	20
2	191	203	12	200	218	18
3	220	230	10	364	360	-4
4	164	180	16	215	225	10
5	160	168	8	296	306	10
6	200	194	-6	175	193	18
7	340	324	-16	253	225	-28
8	228	220	-8	220	230	10
Mean	215.38	219.63	-4.25	257.88	264.63	-6.75
SD	56.40	48.56	12.53	68.63	67.13	15.96
SEM	19.94	17.17	4.43	24.27	23.74	5.64
ME			8.86			11.29
V			4.07			4.32
Correlation coefficient (*r)*			-0.96			-1.20

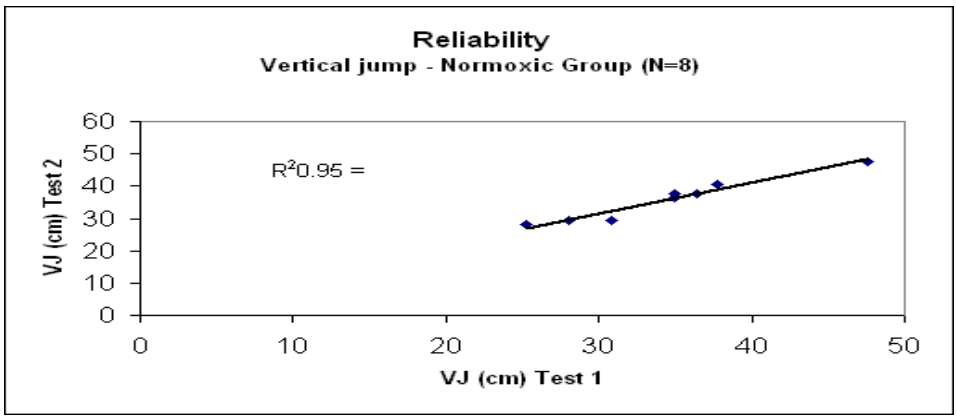




Vertical Jump (cm) measures for Hypoxic Group and Normoxic Group

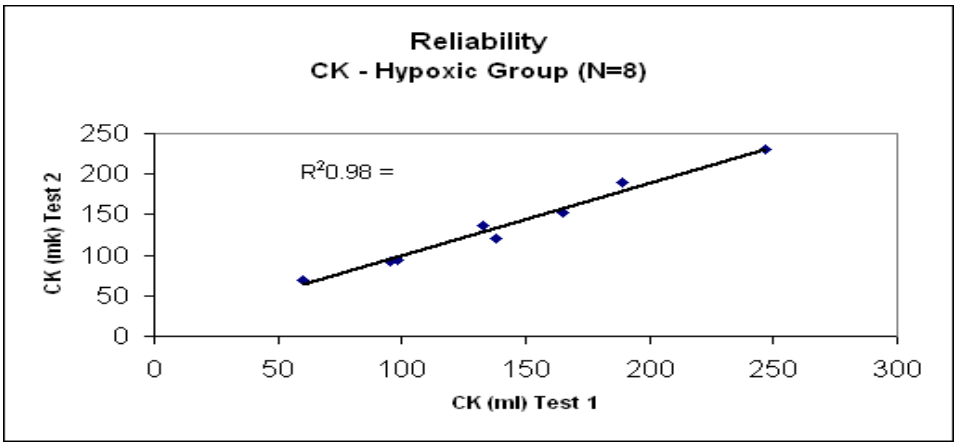
	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	29.4	35	5.60	30.8	29.4	-1.40
2	15.4	18.2	2.80	35	36.4	1.40
3	36.4	42	5.60	35	37.8	2.80
4	33.6	33.6	0.00	36.4	37.8	1.40
5	23.8	28	4.20	28	29.4	1.40
6	29.8	25.2	-4.60	25.2	28	2.80
7	30.8	30.8	0.00	37.8	40.6	2.80
8	39.2	40.6	1.40	47.6	47.6	0.00
Mean	29.80	31.68	-1.88	34.48	35.88	-1.40
SD	7.47	7.92	3.45	6.86	6.69	1.50
SEM	2.64	2.80	1.22	2.42	2.37	0.53
ME			2.44			1.06
V			7.93			3.01
Correlation coefficient (*r)*			-1.54			-2.65

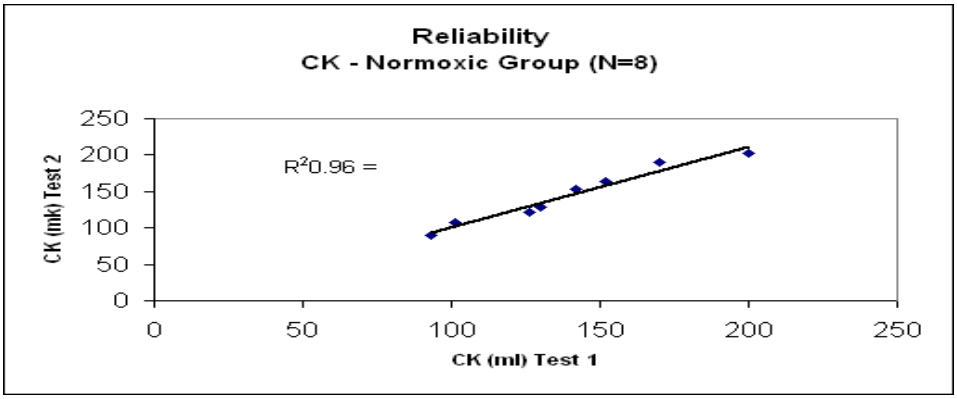




Plasma CK (mmol) measures for Hypoxic Group and Normoxic Group

	Hypoxic Group			Normoxic Group		
Subject	Test 1	Test 2	Difference	Test 1	Test 2	Difference
1	247	230	-17	90	93	3
2	98.60	94	-5	163	152	-11
3	133	137	4	121	126	5
4	165	153	-12	153	142	-11
5	189	190	1	190	170	-20
6	95.30	92	-3	107	101	-6
7	138	120	-18	128	130	2
8	60	70	10	202	200	-2
Mean	140.74	135.75	4.99	144.25	139.25	5.00
SD	59.32	53.92	10.05	39.63	35.19	8.62
SEM	20.97	19.06	3.55	14.01	12.44	3.05
ME			7.11			6.09
V			5.14			4.30
Correlation coefficient (*r)*			1.40			1.64





Creatine kinase (IU/L) activity

	Hypoxia Group							Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	239	254	314	348	308	453	N1	92	193	140	168	238	140
H2	97	180	176	114	176	101	N2	158	375	283	306	515	387
H3	135	288	174	190	167	337	N3	124	209	177	218	98	97
H4	159	658	631	682	661	328	N4	148	188	557	225	286	108
H5	190	237	148	156	219	241	N5	180	448	303	256	170	165
H6	94	133	161	408	466	414	N6	104	226	180	151	120	125
H7	129	230	273	262	321	235	N7	129	241	232	186	184	205
H8	65	101	61	67.60	62.90	90.2	N8	201	345	371	598	330	398
Mean	*138.25*	*260.13*	*242.25*	*278.45*	*297.61*	*274.90*		*141.75*	*278.13*	*280.38*	*263.50*	*242.68*	*203.09*
SD	*56.46*	*172.57*	*175.09*	*199.66*	*190.56*	*133.55*		*37.23*	*97.80*	*135.26*	*143.97*	*135.37*	*121.72*
SE_M	*19.96*	*61.01*	*61.90*	*70.59*	*67.37*	*47.22*		*13.16*	*34.58*	*47.82*	*50.90*	*47.86*	*43.03*
t test	time	0.07	0.44	0.30	0.27	0.70			0.00	0.97	0.77	0.69	0.23
t test	group	0.83	0.50	0.89	0.56	0.44							

Percent Change for Creatine kinase (IU/L) activity

	Hypoxia Group							Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	6.50	31.66	45.91	29.14	89.94	N1	0.00	110.93	53.01	83.61	160.11	53.01
H2	0.00	86.53	82.38	18.13	82.38	4.66	N2	0.00	138.10	79.68	94.29	226.98	145.71
H3	0.00	113.33	28.89	40.74	23.70	149.63	N3	0.00	69.23	43.32	76.52	-20.32	-21.70
H4	0.00	313.84	296.86	328.93	315.72	106.29	N4	0.00	27.46	277.63	52.54	93.90	-26.78
H5	0.00	25.07	-21.90	-17.68	15.57	27.18	N5	0.00	148.89	68.33	42.22	-5.56	-8.33
H6	0.00	42.25	72.19	336.36	398.40	342.78	N6	0.00	117.31	73.08	45.19	15.38	20.19
H7	0.00	78.29	111.63	103.10	148.84	82.17	N7	0.00	86.82	79.84	44.19	42.64	58.91
H8	0.00	55.38	-6.15	4.00	-3.23	38.77	N8	0.00	71.64	84.58	197.51	64.18	98.01
Mean	*0.00*	*97.92*	*81.68*	*125.40*	*144.15*	*120.08*		*0.00*	*101.98*	*99.17*	*65.73*	*78.42*	*27.02*
SD	*0.00*	*96.71*	*100.43*	*143.45*	*151.83*	*106.77*		*0.00*	*40.25*	*75.17*	*51.70*	*85.30*	*61.02*
SE_M	*0.00*	*34.19*	*35.51*	*50.72*	*53.68*	*37.75*		*0.00*	*14.23*	*26.58*	*18.28*	*30.16*	*21.57*
t test	time	*0.03*	*0.36*	*0.37*	*0.18*	*0.58*			*0.00*	*0.97*	*0.67*	*0.82*	*0.18*
t test	group	*0.90*	*0.25*	*0.66*	*0.42*	*0.24*							

Muscle Soreness

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	1	5	3	2	1	1	N1	1	1	1	1	1	1
H2	1	2	1	1	1	1	N2	1	2	3	1	1	1
H3	1	2	2	2	1	1	N3	1	2	1	1	1	1
H4	1	3	3	1	1	1	N4	1	3	2	2	1	1
H5	1	6	4	3	2	2	N5	1	3	2	1	1	1
H6	1	4	5	2	1.5	1	N6	1	2.5	1	1	1	1
H7	1	2	4	2	1	1	N7	1	3	1	1	1	1
H8	1	1	3	1	1	1	N8	1	2	2	1	1	2
Mean	*1.00*	*3.67*	*3.00*	*1.83*	*1.25*	*1.17*		*1.00*	*2.25*	*1.67*	*1.17*	*1.00*	*1.00*
SD	*0.00*	*1.63*	*1.41*	*0.75*	*0.42*	*0.41*		*0.00*	*0.76*	*0.82*	*0.41*	*0.00*	*0.00*
SE_M	*0.00*	*0.58*	*0.50*	*0.27*	*0.15*	*0.14*		*0.00*	*0.27*	*0.29*	*0.14*	*0.00*	*0.00*
t test	*time*	0.01	1.00	0.01	0.01	0.35			0.00	0.08	0.10	0.35	0.35
t test	*group*	0.25	0.05	0.09	0.20	1.00							

Percent Change for Muscle Soreness

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	400.00	200.00	100.00	0.00	0.00	N1	0.00	0.00	0.00	0.00	0.00	0.00
H2	0.00	100.00	0.00	0.00	0.00	0.00	N2	0.00	100.00	200.00	0.00	0.00	0.00
H3	0.00	100.00	100.00	100.00	0.00	0.00	N3	0.00	100.00	0.00	0.00	0.00	0.00
H4	0.00	200.00	200.00	0.00	0.00	0.00	N4	0.00	200.00	100.00	100.00	0.00	0.00
H5	0.00	500.00	300.00	200.00	100.00	100.00	N5	0.00	200.00	100.00	0.00	0.00	0.00
H6	0.00	300.00	400.00	100.00	50.00	0.00	N6	0.00	150.00	0.00	0.00	0.00	0.00
H7	0.00	100.00	300.00	100.00	0.00	0.00	N7	0.00	200.00	0.00	0.00	0.00	0.00
H8	0.00	0.00	200.00	0.00	0.00	0.00	N8	0.00	100.00	100.00	0.00	0.00	100.00
Mean	*0.00*	*266.67*	*200.00*	*83.33*	*25.00*	*16.67*		*0.00*	*125.00*	*66.67*	*16.67*	*0.00*	*0.00*
SD	*0.00*	*163.30*	*141.42*	*75.28*	*41.83*	*40.82*		*0.00*	*75.83*	*81.65*	*40.82*	*0.00*	*0.00*
SE_M	*0.00*	*57.74*	*50.00*	*26.61*	*14.79*	*14.43*		*0.00*	*26.81*	*28.87*	*14.43*	*0.00*	*0.00*
t test	*time*	0.01	1.00	0.01	0.01	0.35			0.00	0.08	0.10	0.35	0.35
t test	*group*	0.25	0.05	0.09	0.20	1.00							

Muscle Tenderness (mean of 6 sites)

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	100	50.80	22.10	48	70.80	87	N1	100	62	62	95	95.80	97.50
H2	100	82.60	98	98	100	100	N2	100	70.80	79	80.80	94	100
H3	100	98	94	100	100	96.60	N3	100	95	95.40	96.60	100	100
H4	100	95	95	96.60	100	100	N4	100	82.90	100	100	100	100
H5	100	93	87.50	88.75	98	100	N5	100	97.50	98.60	100	100	100
H6	100	74	77.50	91.60	97.50	100	N6	100	64	75.80	88.80	96.30	98.60
H7	100	114.16	100	100	100	100	N7	100	100	100	100	100	100
H8	100	97.50	98.30	100	100	100	N8	100	100	99.16	100	100	100
Mean	*100.00*	*82.23*	*79.02*	*87.16*	*94.38*	*97.27*		*100.00*	*78.70*	*85.13*	*93.53*	*97.68*	*99.35*
SD	*0.00*	*17.80*	*28.82*	*19.63*	*11.61*	*5.21*		*0.00*	*15.45*	*15.28*	*7.48*	*2.65*	*1.07*
SE_M	*0.00*	*6.29*	*10.19*	*6.94*	*4.10*	*1.84*		*0.00*	*5.46*	*5.40*	*2.64*	*0.94*	*0.38*
t test	*time*	0.12	0.40	0.09	0.09	0.34			0.03	0.09	0.16	0.11	0.14
t test	*group*	0.25	0.45	0.50	0.48	0.29							

Percent Change for Muscle Tenderness (mean of 6 sites)

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	50.80	22.10	94.49	70.80	87.00	N1	0.00	62.00	62.00	95.00	154.52	97.50
H2	0.00	82.60	98.00	118.64	100.00	100.00	N2	0.00	70.80	79.00	80.80	118.99	100.00
H3	0.00	98.00	94.00	102.04	100.00	96.60	N3	0.00	95.00	95.40	96.60	104.82	100.00
H4	0.00	95.00	95.00	101.68	100.00	100.00	N4	0.00	82.90	100.00	100.00	100.00	100.00
H5	0.00	93.00	87.50	95.43	98.00	100.00	N5	0.00	97.50	98.60	100.00	101.42	100.00
H6	0.00	74.00	77.50	123.78	97.50	100.00	N6	0.00	64.00	75.80	88.80	127.04	98.60
H7	0.00	114.16	100.00	87.60	100.00	100.00	N7	0.00	100.00	100.00	100.00	100.00	100.00
H8	0.00	97.50	98.30	102.56	100.00	100.00	N8	0.00	100.00	99.16	100.00	100.85	100.00
Mean	*0.00*	*82.23*	*79.02*	*106.01*	*94.38*	*97.27*		*0.00*	*78.70*	*85.13*	*93.53*	*117.80*	*99.35*
SD	*0.00*	*17.80*	*28.82*	*12.29*	*11.61*	*5.21*		*0.00*	*15.45*	*15.28*	*7.48*	*20.92*	*1.07*
SE_M	*0.00*	*6.29*	*10.19*	*4.34*	*4.10*	*1.84*		*0.00*	*5.46*	*5.40*	*2.64*	*7.40*	*0.38*
t test	*time*	0.00	0.40	0.09	0.17	0.34			0.00	0.09	0.16	0.06	0.09
t test	*group*	0.25	0.45	0.25	0.13	0.29							

Extensor Isokinetic Peak Torque (Nm) for 120 deg/sec

	Hypoxia Group							Normoxia Group						
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	155	155	193	151	134	156	N1	127	167	165	176	167	186
H2	180	174	173	160	183	186	N2	180	160	144	180	178	170
H3	180	186	183	191	206	197	N3	218	205	235	245	232	225
H4	123	129	130	127	138	125	N4	137	164	182	142	170	171
H5	117	79	104	94	104	115	N5	152	167	175	184	184	179
H6	99	83	92	85	75	77	N6	129	119	138	123	138	152
H7	205	182	201	197	187	199	N7	136	111	99	125	126	137
H8	167	164	157	156	174	159	N8	216	241	254	244	220	209
Mean	*153.25*	*144.00*	*154.13*	*145.13*	*150.13*	*151.75*		*161.88*	*166.75*	*174.00*	*177.38*	*176.88*	*178.63*
SD	*36.79*	*42.76*	*41.16*	*40.93*	*45.22*	*43.39*		*37.97*	*42.12*	*50.83*	*47.83*	*36.24*	*28.54*
SE_M	*13.01*	*15.12*	*14.55*	*14.47*	*15.99*	*15.34*		*13.42*	*14.89*	*17.97*	*16.91*	*12.81*	*10.09*
t test	*time*	*0.13*	*0.11*	*0.13*	*0.38*	*0.74*			*0.59*	*0.24*	*0.71*	*0.93*	*0.68*
t test	*group*	*0.24*	*0.43*	*0.12*	*0.13*	*0.14*							

Normalised Extensor Isokinetic Peak Torque (Nm) for 120 deg/sec

	Hypoxia Group							Normoxia Group						
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	0.00	24.52	-2.58	-13.55	0.65	N1	0.00	31.50	29.92	38.58	31.50	46.46
H2	0.00	-3.33	-3.89	-11.11	1.67	3.33	N2	0.00	-11.11	-20.00	0.00	-1.11	-5.56
H3	0.00	3.33	1.67	6.11	14.44	9.44	N3	0.00	-5.96	7.80	12.39	6.42	3.21
H4	0.00	4.88	5.69	3.25	12.20	1.63	N4	0.00	19.71	32.85	3.65	24.09	24.82
H5	0.00	-32.48	-11.11	-19.66	-11.11	-1.71	N5	0.00	9.87	15.13	21.05	21.05	17.76
H6	0.00	-16.16	-7.07	-14.14	-24.24	-22.22	N6	0.00	-7.75	6.98	-4.65	6.98	17.83
H7	0.00	-11.22	-1.95	-3.90	-8.78	-2.93	N7	0.00	-18.38	-27.21	-8.09	-7.35	0.74
H8	0.00	-1.80	-5.99	-6.59	4.19	-4.79	N8	0.00	11.57	17.59	12.96	1.85	-3.24
Mean	*0.00*	*-7.10*	*0.23*	*-6.08*	*-3.15*	*-2.07*		*0.00*	*3.68*	*7.88*	*9.49*	*10.43*	*12.75*
SD	*0.00*	*12.47*	*11.11*	*8.68*	*13.47*	*9.24*		*0.00*	*17.16*	*21.60*	*15.28*	*13.60*	*17.56*
SE_M	*0.00*	*4.41*	*3.93*	*3.07*	*4.76*	*3.27*		*0.00*	*6.07*	*7.64*	*5.40*	*4.81*	*6.21*
t test	*time*	*0.15*	*0.10*	*0.10*	*0.43*	*0.74*			*0.56*	*0.26*	*0.79*	*0.81*	*0.43*
t test	*group*	*0.15*	*0.30*	*0.04*	*0.10*	*0.08*							

Extensor Isokinetic Peak Torque (Nm) for 240 deg/sec

	Hypoxia Group							Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	106	99	110	99	99	100	N1	76	127	125	127	114	133
H2	129	145	134	118	132	145	N2	136	136	161	142	156	149
H3	121	137	132	140	144	141	N3	191	180	184	191	183	176
H4	91	126	98	113	130	117	N4	66	103	111	81	113	130
H5	87	64	79	68	77	81	N5	125	103	121	104	106	132
H6	65	68	72	60	61	57	N6	104	85	96	107	99	103
H7	164	133	167	159	126	167	N7	75	61	61	57	75	69
H8	114	118	119	145	130	127	N8	176	160	197	182	182	179
Mean	*109.63*	*111.25*	*113.88*	*112.75*	*112.38*	*116.88*		*118.63*	*119.38*	*132.00*	*123.88*	*128.50*	*133.88*
SD	*30.10*	*31.15*	*31.21*	*35.70*	*29.91*	*36.14*		*47.17*	*39.25*	*45.85*	*46.62*	*40.14*	*36.29*
SE_M	*10.64*	*11.01*	*11.03*	*12.62*	*10.57*	*12.78*		*16.68*	*13.88*	*16.21*	*16.48*	*14.19*	*12.83*
t test	*time*	*0.84*	*0.70*	*0.84*	*0.95*	*0.47*			*0.94*	*0.03*	*0.15*	*0.43*	*0.29*
t test	*group*	*0.59*	*0.38*	*0.57*	*0.22*	*0.36*							

Normalised Extensor Isokinetic Peak Torque (Nm) for 240 deg/sec

	Hypoxia Group							Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	-6.60	3.77	-6.60	-6.60	-5.66	N1	0.00	67.11	64.47	67.11	50.00	75.00
H2	0.00	12.40	3.88	-8.53	2.33	12.40	N2	0.00	0.00	18.38	4.41	14.71	9.56
H3	0.00	13.22	9.09	15.70	19.01	16.53	N3	0.00	-5.76	-3.66	0.00	-4.19	-7.85
H4	0.00	38.46	7.69	24.18	42.86	28.57	N4	0.00	56.06	68.18	22.73	71.21	96.97
H5	0.00	-26.44	-9.20	-21.84	-11.49	-6.90	N5	0.00	-17.60	-3.20	-16.80	-15.20	5.60
H6	0.00	4.62	10.77	-7.69	-6.15	-12.31	N6	0.00	-18.27	-7.69	2.88	-4.81	-0.96
H7	0.00	-18.90	1.83	-3.05	-23.17	1.83	N7	0.00	-18.67	-18.67	-24.00	0.00	-8.00
H8	0.00	3.51	4.39	27.19	14.04	11.40	N8	0.00	-9.09	11.93	3.41	3.41	1.70
Mean	*0.00*	*2.53*	*4.03*	*2.42*	*3.85*	*5.73*		*0.00*	*6.72*	*16.22*	*7.47*	*14.39*	*21.50*
SD	*0.00*	*20.32*	*6.15*	*17.67*	*20.81*	*13.86*		*0.00*	*34.62*	*32.98*	*27.96*	*30.27*	*40.68*
SE_M	*0.00*	*7.18*	*2.17*	*6.25*	*7.36*	*4.90*		*0.00*	*12.24*	*11.66*	*9.88*	*10.70*	*14.38*
t test	*time*	*0.73*	*0.80*	*0.77*	*0.76*	*0.67*			*0.60*	*0.02*	*0.19*	*0.38*	*0.20*
t test	*group*	*0.72*	*0.32*	*0.66*	*0.28*	*0.28*							

Isometric Peak Strength (Nm/kg) at 75 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	229.00	201.00	231.00	217.00	254.00	258.00	N1	350.00	301.00	370.00	363.00	363.00	366.00
H2	197.00	277.00	292.00	275.00	312.00	316.00	N2	209.00	197.00	278.00	259.00	275.00	335.00
H3	225.00	245.00	247.00	267.00	264.00	267.00	N3	362.00	316.00	355.00	342.00	320.00	331.00
H4	172.00	197.00	207.00	213.00	221.00	206.00	N4	220.00	243.00	275.00	243.00	283.00	287.00
H5	164.00	159.00	122.00	127.00	161.00	216.00	N5	301.00	344.00	377.00	391.00	395.00	420.00
H6	197.00	178.00	225.00	233.00	240.00	252.00	N6	184.00	179.00	206.00	213.00	216.00	245.00
H7	332.00	336.00	365.00	411.00	365.00	359.00	N7	239.00	222.00	182.00	235.00	214.00	225.00
H8	224.00	251.00	232.00	264.00	264.00	277.00	N8	225.00	275.00	271.00	321.00	335.00	347.00
Mean	*217.50*	*230.50*	*240.13*	*250.88*	*260.13*	*268.88*		*261.25*	*259.63*	*289.25*	*295.88*	*300.13*	*319.50*
SD	*52.25*	*58.29*	*69.45*	*80.09*	*60.38*	*50.09*		*67.42*	*58.99*	*73.33*	*66.59*	*65.45*	*64.29*
SE_M	*18.47*	*20.61*	*24.55*	*28.32*	*21.35*	*17.71*		*23.84*	*20.86*	*25.93*	*23.54*	*23.14*	*22.73*
t test	*time*	0.31	0.35	0.20	0.38	0.27			0.91	0.07	0.57	0.57	0.02
t test	*group*	*0.42*	*0.32*	*0.36*	*0.35*	*0.19*							

Percent Change for Isometric Peak Strength at 75 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	-12.23	0.87	-5.24	10.92	12.66	N1	0.00	-14.00	5.71	3.71	3.71	4.57
H2	0.00	40.61	48.22	39.59	58.38	60.41	N2	0.00	-5.74	33.01	23.92	31.58	60.29
H3	0.00	8.89	9.78	18.67	17.33	18.67	N3	0.00	-12.71	-1.93	-5.52	-11.60	-8.56
H4	0.00	14.53	20.35	23.84	28.49	19.77	N4	0.00	10.45	25.00	10.45	28.64	30.45
H5	0.00	-3.05	-25.61	-22.56	-1.83	31.71	N5	0.00	14.29	25.25	29.90	31.23	39.53
H6	0.00	-9.64	14.21	18.27	21.83	27.92	N6	0.00	-2.72	11.96	15.76	17.39	33.15
H7	0.00	1.20	9.94	23.80	9.94	8.13	N7	0.00	-7.11	-23.85	-1.67	-10.46	-5.86
H8	0.00	12.05	3.57	17.86	17.86	23.66	N8	0.00	22.22	20.44	42.67	48.89	54.22
Mean	*0.00*	*6.55*	*10.17*	*14.28*	*20.36*	*25.37*		*0.00*	*0.59*	*11.95*	*14.90*	*17.42*	*25.98*
SD	*0.00*	*16.88*	*20.63*	*19.31*	*17.80*	*16.09*		*0.00*	*13.38*	*18.42*	*16.54*	*21.76*	*26.44*
SE_M	*0.00*	*5.97*	*7.29*	*6.83*	*6.29*	*5.69*		*0.00*	*4.73*	*6.51*	*5.85*	*7.69*	*9.35*
t test	*time*	*0.31*	*0.49*	*0.21*	*0.19*	*0.29*			*0.90*	*0.09*	*0.55*	*0.43*	*0.04*
t test	group	*0.43*	*0.85*	*0.95*	*0.74*	*0.92*							

Isometric Peak Strength (Nm/kg) at 55 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	218.00	108.00	198.00	206.00	216.00	241.00	N1	313.00	260.00	306.00	308.00	305.00	312.00
H2	212.00	235.00	260.00	270.00	289.00	286.00	N2	229.00	241.00	271.00	296.00	315.00	296.00
H3	194.00	190.00	210.00	213.00	217.00	217.00	N3	315.00	312.00	332.00	361.00	330.00	323.00
H4	140.00	156.00	167.00	170.00	178.00	191.00	N4	188.00	232.00	236.00	225.00	247.00	225.00
H5	174.00	104.00	121.00	132.00	159.00	188.00	N5	232.00	287.00	287.00	263.00	287.00	301.00
H6	187.00	174.00	186.00	209.00	206.00	206.00	N6	190.00	193.00	172.00	210.00	117.00	138.00
H7	305.00	311.00	319.00	319.00	286.00	317.00	N7	210.00	216.00	222.00	221.00	271.00	264.00
H8	213.00	195.00	205.00	212.00	237.00	247.00	N8	254.00	250.00	296.00	336.00	334.00	324.00
Mean	*205.38*	*184.13*	*208.25*	*216.38*	*223.50*	*236.63*		*241.38*	*248.88*	*265.25*	*277.50*	*275.75*	*272.88*
SD	*47.68*	*67.56*	*59.61*	*57.16*	*46.31*	*45.97*		*49.89*	*38.01*	*52.08*	*56.55*	*70.62*	*63.86*
SE_M	*16.86*	*23.88*	*21.07*	*20.21*	*16.37*	*16.25*		*17.64*	*13.44*	*18.41*	*19.99*	*24.97*	*22.58*
t test	*time*	*0.23*	*0.04*	*0.01*	*0.33*	*0.03*			*0.54*	*0.09*	*0.19*	*0.91*	*0.61*
t test	*group*	*0.08*	*0.10*	*0.08*	*0.09*	*0.18*							

Percent Change for Isometric Peak Strength at 55 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	-50.46	83.33	4.04	4.85	11.57	N1	0.00	-16.93	-2.24	-1.60	-2.56	-0.32
H2	0.00	10.85	10.64	3.85	7.04	-1.04	N2	0.00	5.24	18.34	29.26	37.55	29.26
H3	0.00	-2.06	10.53	1.43	1.88	0.00	N3	0.00	-0.95	5.40	14.60	4.76	2.54
H4	0.00	11.43	7.05	1.80	4.71	7.30	N4	0.00	23.40	25.53	19.68	31.38	19.68
H5	0.00	-40.23	16.35	9.09	20.45	18.24	N5	0.00	23.71	23.71	13.36	23.71	29.74
H6	0.00	-6.95	6.90	12.37	-1.44	0.00	N6	0.00	1.58	-9.47	10.53	-38.42	-27.37
H7	0.00	1.97	2.57	0.00	-10.34	10.84	N7	0.00	2.86	5.71	5.24	29.05	25.71
H8	0.00	-8.45	5.13	3.41	11.79	4.22	N8	0.00	-1.57	16.54	32.28	31.50	27.56
Mean	*0.00*	*-10.49*	*17.81*	*4.50*	*4.87*	*6.39*		*0.00*	*4.67*	*10.44*	*15.42*	*14.62*	*13.35*
SD	*0.00*	*26.33*	*26.80*	*4.36*	*9.07*	*6.86*		*0.00*	*13.44*	*12.58*	*11.44*	*25.60*	*20.28*
SE_M	*0.00*	*9.31*	*9.48*	*1.54*	*3.21*	*2.43*		*0.00*	*4.75*	*4.45*	*4.04*	*9.05*	*7.17*
t test	*time*	*0.24*	*0.13*	*0.21*	*0.91*	*0.66*			*0.36*	*0.13*	*0.23*	*0.92*	*0.64*
t test	*group*	*0.10*	*0.55*	*0.04*	*0.30*	*0.33*							

Isometric Peak Strength (Nm/kg) at 35 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	141.00	138.00	137.00	155.00	165.00	213.00	N1	220.00	214.00	226.00	217.00	232.00	213.00
H2	146.00	151.00	159.00	176.00	198.00	186.00	N2	163.00	188.00	206.00	202.00	214.00	212.00
H3	118.00	137.00	129.00	134.00	137.00	138.00	N3	209.00	217.00	239.00	248.00	221.00	214.00
H4	111.00	107.00	104.00	98.00	108.00	108.00	N4	138.00	155.00	164.00	165.00	184.00	170.00
H5	145.00	83.00	104.00	96.00	122.00	140.00	N5	152.00	193.00	187.00	178.00	184.00	198.00
H6	138.00	113.00	132.00	159.00	156.00	152.00	N6	157.00	149.00	141.00	167.00	156.00	144.00
H7	236.00	232.00	220.00	220.00	243.00	236.00	N7	141.00	144.00	174.00	138.00	199.00	202.00
H8	213.00	213.00	213.00	213.00	213.00	213.00	N8	209.00	220.00	232.00	250.00	271.00	285.00
Mean	*156.00*	*146.75*	*149.75*	*156.38*	*167.75*	*173.25*		*173.63*	*185.00*	*196.13*	*195.63*	*207.63*	*204.75*
SD	*44.54*	*51.59*	*44.91*	*46.60*	*46.87*	*45.23*		*33.47*	*31.71*	*35.37*	*40.68*	*35.23*	*40.70*
SE_M	*15.75*	*18.24*	*15.88*	*16.48*	*16.57*	*15.99*		*11.84*	*11.21*	*12.51*	*14.38*	*12.45*	*14.39*
t test	*time*	*0.32*	*0.50*	*0.18*	*0.02*	*0.45*			*0.09*	*0.05*	*0.94*	*0.23*	*0.54*
t test	*group*	*0.12*	*0.04*	*0.10*	*0.04*	*0.07*							

Percent Change for Isometric Peak Strength at 35 deg of Knee Flexion

		Hypoxia Group						Normoxia Group					
Subject	0	24	48	72	96	120	Subject	0	24	48	72	96	120
H1	0.00	-2.13	-2.84	9.93	17.02	51.06	N1	0.00	-2.73	2.73	-1.36	5.45	-3.18
H2	0.00	3.42	8.90	20.55	35.62	27.40	N2	0.00	15.34	26.38	23.93	31.29	30.06
H3	0.00	16.10	9.32	13.56	16.10	16.95	N3	0.00	3.83	14.35	18.66	5.74	2.39
H4	0.00	-3.60	-6.31	-11.71	-2.70	-2.70	N4	0.00	12.32	18.84	19.57	33.33	23.19
H5	0.00	-42.76	-28.28	-33.79	-15.86	-3.45	N5	0.00	26.97	23.03	17.11	21.05	30.26
H6	0.00	-18.12	-4.35	15.22	13.04	10.14	N6	0.00	-5.10	-10.19	6.37	-0.64	-8.28
H7	0.00	-1.69	-6.78	-6.78	2.97	0.00	N7	0.00	2.13	23.40	-2.13	41.13	43.26
H8	0.00	0.00	0.00	0.00	0.00	0.00	N8	0.00	5.26	11.00	19.62	29.67	36.36
Mean	*0.00*	*-6.10*	*-3.79*	*0.87*	*8.27*	*12.43*		*0.00*	*7.25*	*13.69*	*12.72*	*20.88*	*19.26*
SD	*0.00*	*17.53*	*11.73*	*17.95*	*15.62*	*19.02*		*0.00*	*10.52*	*12.35*	*10.24*	*15.50*	*19.52*
SE_M	*0.00*	*6.20*	*4.15*	*6.35*	*5.52*	*6.72*		*0.00*	*3.72*	*4.37*	*3.62*	*5.48*	*6.90*
t test	*time*	*0.36*	*0.45*	*0.19*	*0.02*	*0.41*			*0.09*	*0.07*	*0.83*	*0.21*	*0.54*
t test	group	*0.17*	*0.03*	*0.16*	*0.19*	*0.59*							