ABSTRACT

THE CARDIOVASCULAR AND METABOLIC EFFECTS OF HIGH INTENSITY INTERVAL TRAINING WITH AND WITHOUT HIGH ALTITUDE SIMULATION AND EITHER WITH AND WITHOUT HIGH CONCENTRATION OXYGEN RECOVERY ASSISTANCE

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Purpose: The study investigated the acute responses of altitude (Denver, CO) simulation during high intensity interval training and the subsequent supplementation of oxygen to facilitate greater recovery. Lacking literature on the subject matter is a major consideration for completion of the study. We hypothesize that oxygen supplementation during an acute bout of high intensity interval training with accompanying altitude will allow for greater recovery.
Methods: Seven healthy cyclists aged 40.9 ± 7.01 (Height: 68.4 ± 4.98: Weight: 171.3 ± 33.29: 19.3% ±7.41%: VO2 Max L/min 4.12 ± 1.17) performed baseline VO2max testing and three subsequent separate randomized trials consisting of three HIIT and recovery intervals with varying conditions. Session A: altitude intervals / supplemental oxygen recovery. Session B: sea level HIIT / sea level recovery. Session C: altitude HIIT / sea level recovery. Trial intensity will be established by cardiac output prediction and set at 75% HIIT and 50% recovery in watts
Results: Supplemental oxygen following HIIT elicited significant responses in HR ($p<0.035$, $p<0.012$), VO2 ($p<0.029$, $p<0.030$, $p<0.004$, $p<0.001$), cardiac output ($p<0.012$, $p<0.002$), and right quadricep oxygen saturation ($p<0.011$, $p<0.013$, $p<0.009$)
Conclusion: The implementation of supplemental oxygen following altitude simulation with HIIT will facilitate greater recovery. Although significance was found among multiple variables, more subjects are needed for other to become significant.

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THE CARDIOVASCULAR AND METABOLIC EFFECTS OF HIGH-INTENSITY INTERVAL TRAINING WITH AND WITHOUT HIGH-ALTITUDE SIMULATION AND EITHER WITH AND WITHOUT HIGH-CONCENTRATION OXYGEN RECOVERY ASSISTANCE

BY

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CHAPTER 1

INTRODUCTION

The physiological and metabolic responses for meeting the demands of exercise are complex. Additionally, with increasing exercise intensity, how well a person meets these demands requires a highly integrated response across each major system involved. Limitations in any key system (i.e. heart function) play a major factor in how coordinating systems need to respond (Porcari, Bryant, & Comana, 2015).

For example, endurance capacity (i.e. $VO_{2\,max}$) is dependent on three major systems (Figure 1): the respiratory system, the cardiovascular system, and muscle metabolism (Wilmore, 1969). Individuals with high maximal oxygen consumption values (i.e. $\geq 90^{th}$ percentile) usually show similar and well–coordinated responses as exercise intensity goes from low to maximal effort (Ekblom & Hermansen, 1968). In healthy people, $VO_{2\,max}$ is primarily determined by the cardiovascular system's ability to maintain cardiac output and stroke volume (Brooks, Fahey, & Baldwin, 2005). In rare situations with elite endurance athletes, the cardiovascular system is so well developed that, like in elite race horses, arterial desaturation occurs because the rate of blood passing through the lung exceeds the respiratory gas exchange oxygen uptake transit time (Powers, Lawler, Dempsey, Dodd, & Landry, 1989). Thus, in these athletes, $VO_{2\,max}$ is limited by the cardiorespiratory system together (Brooks et al., 2005; Evans & Rose, 1988).

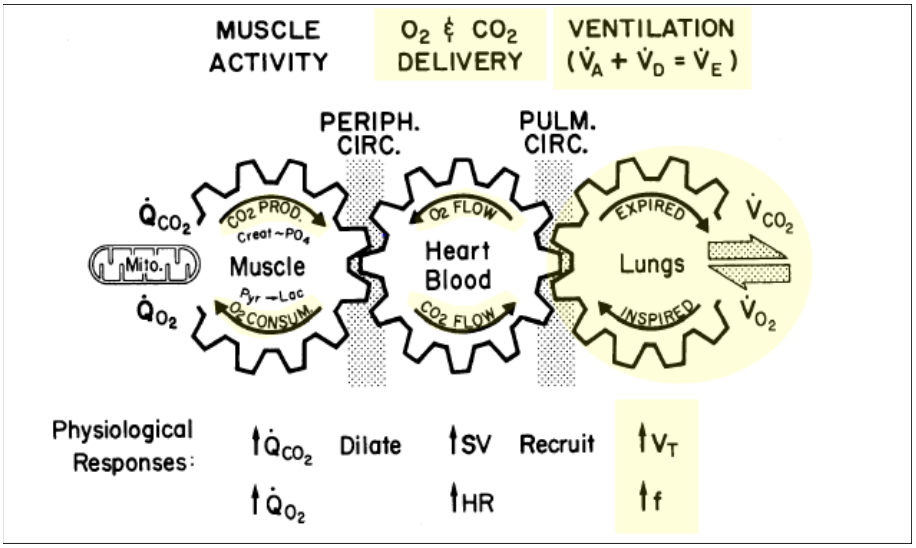


Figure 1: Body systems. Integration of bodily mechanisms during exercise.

The primary focus of this project is to understand the metabolic and physiological responses to a training modality called "oxygen contrast" training during moderate-to high-intensity exercise (Wilson & Welch, 1974). Oxygen contrast exercise training involves having a person exercise for a short duration (i.e. 60 seconds) at approximately 75% of $VO_{2\,max}$ while the air a subject consumes is at an oxygen concentration representing 10-15,000ft above sea level or a Pio_2 equal to 99.5mmHg. After completing the interval under reduced oxygen driving pressures, a person continues the interval (15 seconds additionally) followed by a 2-minute recovery at 50% of $VO_{2\,max}$ breathing air at 48% oxygen concentration to enhance recovery. Practitioners believe that with this type of training, individuals can gain the same metabolic and physiological benefits as performing high-intensity interval training (HIIT) with more resultant volume. HIIT sessions usually require a person to exercise above their $VO_{2\,max}$ workload by 15-30% for short periods (10-60seconds) with contrasting active recovery periods (Astorino &

Robergs, 2003; Gibala, Little, MacDonald, & Hawley, 2012). Previous research has shown that HIIT can "serve as an effective alternate to endurance training producing similar or even superior changes in a range of physiological, performance, and health-related markers in both healthy individuals and diseased populations" (Gibala et al, 2012, p. 1). The physiological benefits of HIIT training include increasing the maximal activity of mitochondrial enzymes, reducing glycogen utilization and lactate accumulation during matched-work exercise, and improving performance on tasks that rely on aerobic metabolism. Usually, HIIT requires less total training time compared to high-volume endurance training. However, HIIT can result in similar training adaptions and performance outcomes (Gibala et al., 2006).

Practitioners of contrast training believe that by using this type of modality in combination with moderately high-intensity exercise, individuals can display the same benefits of HIIT workouts, but at a much lower risk for injury. They also believe that contrast training may have greater applications in people with limited exercise capacity by creating a HIIT-like training effect while keeping the overall exercise moderate in duration and intensity.

To date, there are no well-controlled oxygen contrast training (OCT) studies characterizing the acute or chronic metabolic, cardiovascular, and respiratory effects of this training modality. Thus, the purpose of this thesis is to discover what specific effects oxygen-contrast HIIT training produces as implemented in sports performance and wellness patients at the EnergyBar Wellness Center in Naperville, IL.

CHAPTER 2

LITERATURE REVIEW

Physiological Acute Responses to Altitude

At any given steady-state (i.e. 70% of max) exercise (compared to sea level) for performance to be maintained, one would see at altitude an increase in cardiac output, a decrease in stroke volume, and an increase in heart rate (Kenny, Wilmore, & Costill, 2015; Noakes, Peltonen, & Rusko, 2001; Ploutz-Snyder, Simoneau, Gilders, Staron, & Hagerman, 1996). Figure 2 illustrates these physiological responses. The principles of Henry's law (i.e. gas moves from high to low pressure) leads to these physiological responses due to a decrease in oxygen driving pressure at altitude, which influences the amount of oxygen entering the body.

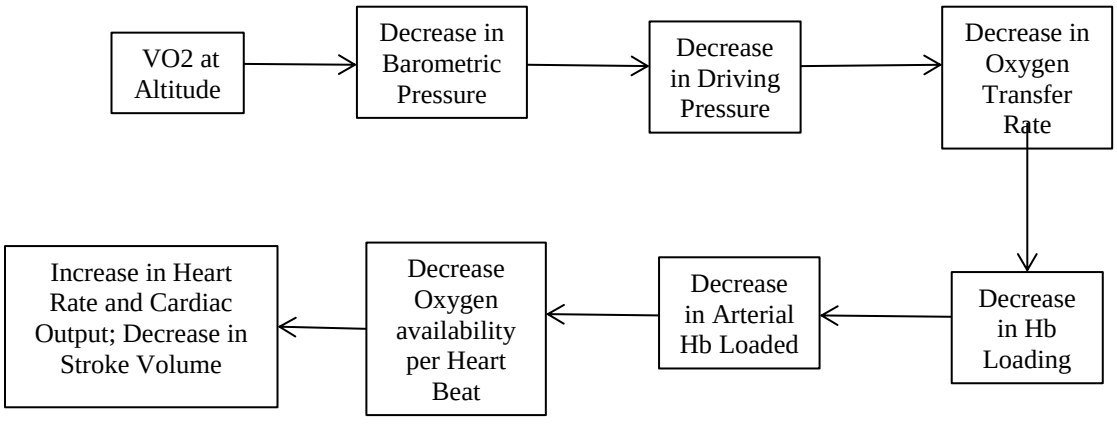


Figure 2: Altitude physiology. The physiological responses of the cardiovascular system via acute exposure altitude.

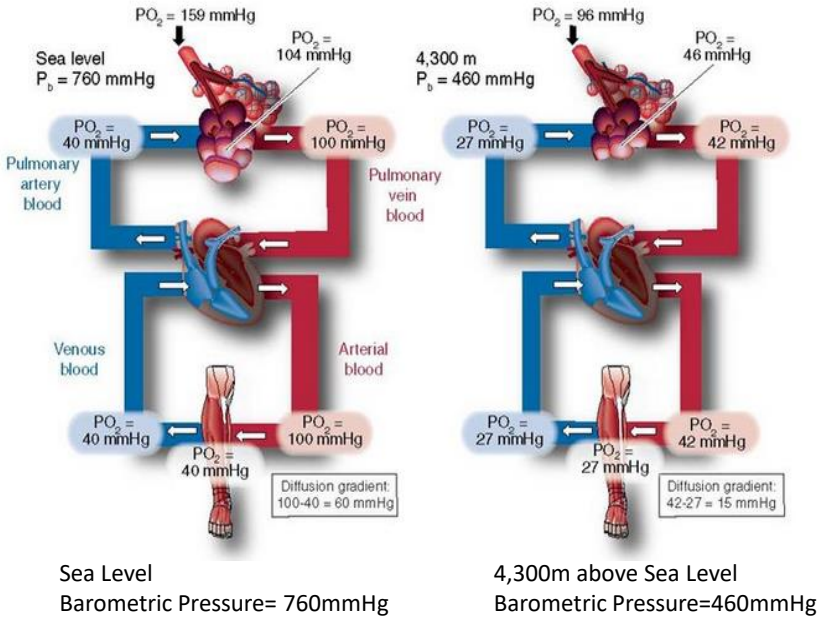


Figure 3: Partial pressures. Depiction of partial pressures upon exposure to sea level and altitude's barometric pressure.

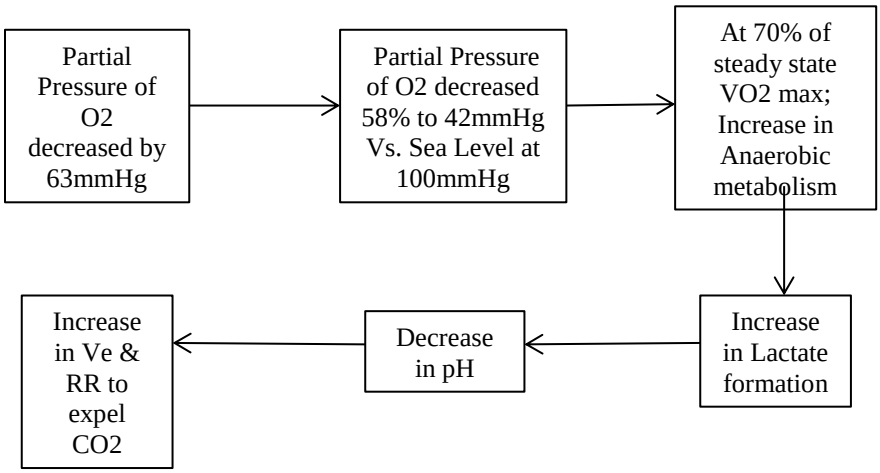


Figure 4: Altitude and partial pressure. Physiological responses to a decrease in barometric pressure accompanied by an increase in altitude to 4,300m.

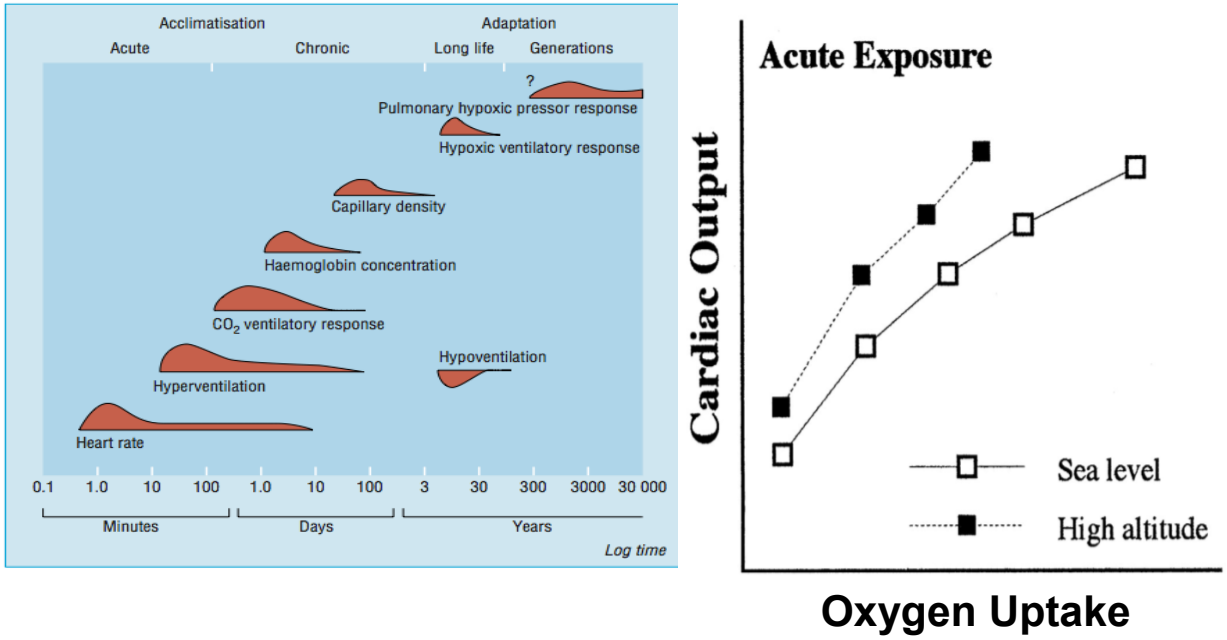


Figure 5: Altitude responses. (Left) Cardiorespiratory responses to the accompanied time that they occur. (Right) Cardiac output and response to acute exposure to altitude.

For an individual exposed to altitude (as shown in Figures 3 & 4), the driving pressure is decreased by 63 mmHg and thus the oxygen transfer rate diminishes. When the oxygen transfer rate declines, the result is reduced hemoglobin loading of oxygen and consequently lower arterial oxygen saturation levels. This transfer rate is also impacted with the lowering of blood pH due to tissue hypoxia from altitude. The Bohr effect, due to lower pH at altitude, limits the amount of oxygen that can remain bound to hemoglobin at a given partial pressure (Bursaux, 1974). Arterial hemoglobin oxygen saturation at sea level (i.e. partial pressure at the alveoli= 104mmHg) is typically around 97% to 96%. With a decrease in partial pressure due to exposure to altitude (i.e. 4,300m= 46mmHg), oxygen saturation drops to roughly 80% (Kenny et al.,

2015). Thus, when a person has lower oxygen availability, this arterial oxygen reduction creates a reliance on more anaerobic processes (i.e. increased lactate). With increases in blood lactate, blood pH lowers to a greater degree, and the resultant response to buffer high blood acidity is an increase in both that person's respiration rate and total ventilatory volume. These compensatory responses increase the volume of carbon dioxide expelled from the lungs in an attempt to offset the lowering blood pH (i.e. termed the respiratory compensatory buffering response). Correspondingly, as ventilation rates increase due to the reduced oxygen availability, heart rate will be disproportionally elevated at a given fixed work rate. Also, cardiac output (Figure 5) will be slightly higher than expected for the given intensity (Hsia, 2001). According to Kenny, Wilmore, and Costill (2015), these responses will occur within the first few hours of being exposed to altitude. The reliance on anaerobic processes, due to the increase in ventilation, will create more dependence on glycogen stores and further limit the expected performance.

Figure 6 demonstrates that oxygen saturation of blood (i.e. low to high kPa is representative of low to high saturation) is incomplete at altitude compared to sea-level conditions. Figure 6 further demonstrates that blood's time spent at the alveolar capillaries slows to compensate for the lower driving pressures, as depicted by the Everest condition. Unfortunately, as capillary transit time decreases in an attempt to maintain oxygen saturation at sea-level levels, it is accompanied by pulmonary vasoconstriction. Although the reasoning why pulmonary vasoconstriction occurs is debated still, this response implies that it helps to better match lung perfusion and ventilation (Peacock, 1998) as needed while living or working at altitude. This perfusion and ventilation mismatch, created by hypoxic pulmonary vasoconstriction (HPV), allows blood that is located in poorly oxygenated areas of the lung to be

shunted to areas of high oxygenation, and thus improvement of alveoli efficiency (Moudgil, Michelakis, & Archer, 2005). This increase of vasoconstriction increases the pulmonary arterial pressure (PAP) and increases the likelihood of pulmonary edema (Bärtsch & Gibbs, 2007; Peacock, 1998). This effect, though, is reversed immediately upon return to sea-level barometric pressure (Moudgil et al., 2005). As seen in Figure 6, the transient time to saturation is severely elongated and incomplete with higher altitudes. This response contributes to the limitations of performance as previously discussed.

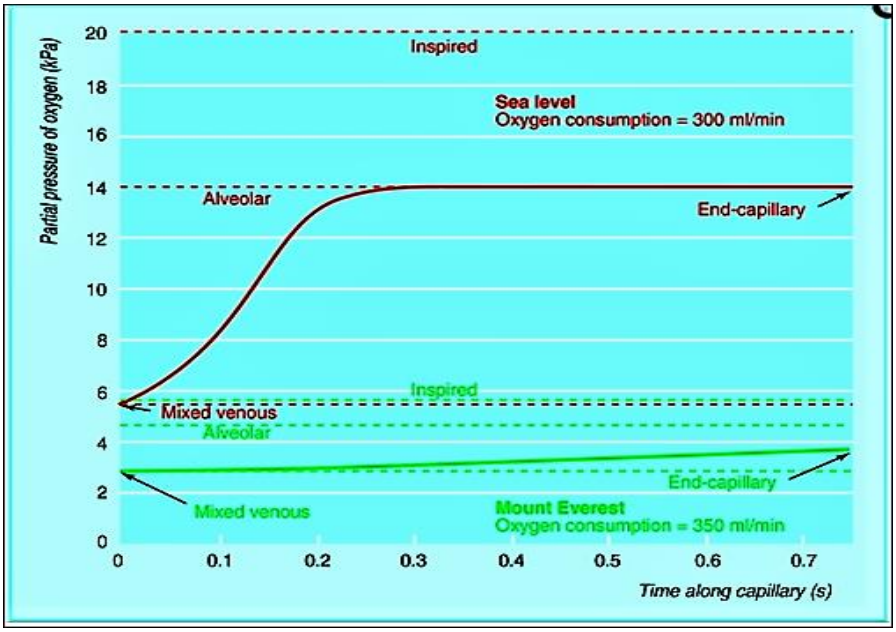


Figure 6: Incomplete saturation. Depiction of an acute response to hypoxic pulmonary vasoconstriction (HPV) and its effect on oxygen transfer rate.

Acute Responses of Interval Training

Interval training as defined by the American College of Sports Medicine (ACSM, 2014), involves varying exercise intensity and duration at fixed intervals during a single exercise bout. The duration and intensity of the intervals can be varied depending on the goals of the training

session and physical fitness level of the subject. Therefore, duration and intensity are the determinants of the physiological acute responses that occur during the said exercise bout. These two factors are inversely related. This allows for intensity during the exercise to be high with low duration and vice versa. ACSM defines intensity of exercise by listing five separate categories ranging from very light to near maximal. The descriptions of intensity are defined by Table 1.

Table 1: ACSM Definitions of Exercise Intensity

Intensity	**% of HR Max**	**% of VO2 max**
Very Light	<57	<37
Light	57-64	37-45
Moderate	64-76	46-64
Vigorous	76-96	64-91
Near Max/At Max	≥96	≥91

HIIT, which this study encompassed, utilizes working at a high intensity (i.e. vigorous or near max) for short durations (15 seconds to 2 minutes) and a recovery portion estimated at a light or very light intensity of equal or greater duration. For our purposes, the recovery segment of the HIIT interval was of greater duration than that of the higher intensity portion. This method facilitates recovery for preparation of the next interval by having a greater rest-to-work ratio. The objective of HIIT is an attempt to maximize volume of training at a high intensity. This maximized volume is achieved due to the greater amount of time spent at a high intensity than that of a comparable steady-state exercise (SSE). The advantages of practicing this method are that the acute responses can be maximized due to the intensity and volume. Also, the potential long-term adaptions can occur quicker than that of SSE. This concept is further described by the limits of a SSE at a high intensity. During SSE at high intensity, with an attempt for a long

duration, fatigue occurs quickly due to physiological limits, and thus the adaptions that occur as a result of working at the said intensity are minimized compared to HIIT (ACSM, 2014).

The acute responses that occur during a HIIT session can be manipulated in a variety of ways. These responses can vary by changing the peak workload, peak workload duration, recovery workload, recovery workload duration, or mean workload. High peak workloads (i.e. 95% of max oxygen consumption) and long durations (i.e. 4 min intervals; 4 min recovery) at the prescribed workload produce a higher peak response of heart rate and lactate levels contrasted to short duration HIIT (i.e. 20 second intervals) and SSE (i.e. SSE mean workload equivalent to mean workload of both HIIT scenarios) (Tschakert et al., 2015). This response coincides with the findings that mean workload (i.e. duration and intensity) is the sole determinant of acute cardiorespiratory responses (ACSM, 2014; Åstrand, Åstrand, Christensen, & Hedman, 1960; Tschakert & Hofmann, 2013). Figure 7 graphically illustrates and Table 2 quantifies the response of heart rate (HR) and lactate (La) during these three protocols. Therefore, the conclusion of these findings is that longer duration HIIT intervals exhibit anaerobic responses, and conversely, aerobic responses for short duration HIIT intervals (Tschakert et al., 2015; Wallner, Simi, Tschakert, & Hofmann, 2014). The rationale behind the determination of anaerobic or aerobic work, in which intervals are classified, is whether the lactate accumulation can be cleared during a recovery portion of an interval (Christensen, Hedman, & Saltin, 1960). The determinant of the La accumulation rate is defined by the peak workload of the interval and creation is defined by the time at the said peak workload (Tschakert & Hofmann, 2013). The Tschakert study protocol of a longer, then typical, duration HIIT (i.e. 4 minutes at 95% of HR_{max} in watts during interval

segments and 4 minutes at 70% of HR_{max} in watts during recovery segments) mimics the responses that will potentially be observed, as later discussed in our study description below.

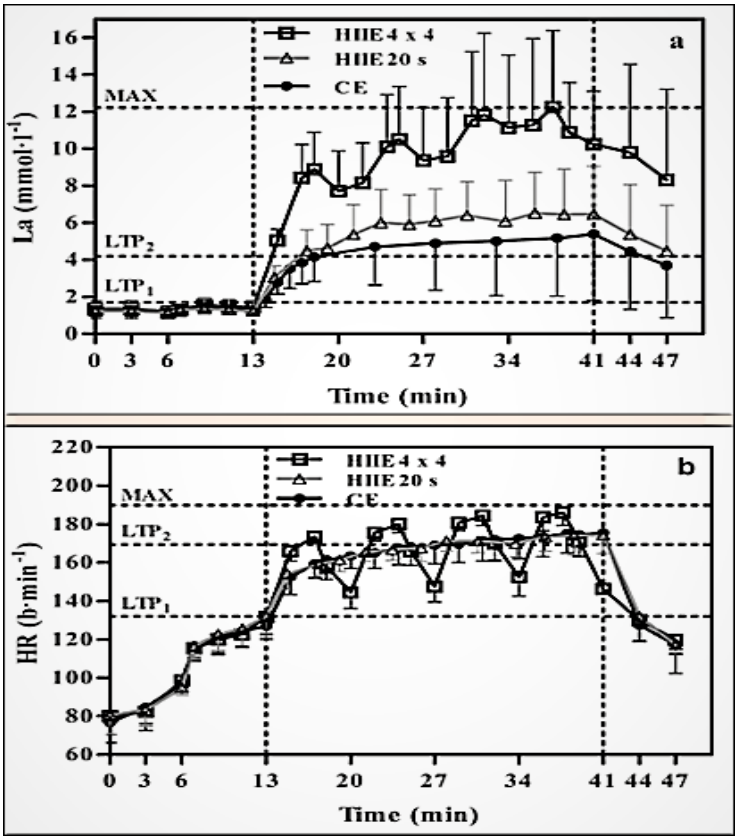


Figure 7: HIIT physiology. Heart rate and lactate during differing HIIT and steady-state protocols (CE is defined as SSE).

Another facet of the Tschakert study was the significant difference in peak HR for longer duration HIIT protocols (Table 2). What is also important to note is that mean HR stays relatively low (i.e. with respect to long duration HIIT) for SSE and short-duration HIIT due to a short duration at peak workload. This is extremely important because the peak HR of both these two protocols was similar, but the peak workload was higher. This result allows for individuals

to gain the long-term cardiovascular adaptions that occur after higher workloads with reduced stress and a more efficient use of time (Åstrand et al., 1960; Burgomaster et al., 2008; Meyer et al., 1997). The implementation of this response via short-duration HIIT trials is currently being used in diseased populations for rehabilitation, as a substitute for SSE (Meyer et al., 1997; Tschakert & Hofmann, 2013; Tschakert et al., 2015).

Table 2: HIIT physiology data. Responses to exercise of three different protocols (also represented by Figure 7). CE is defined as SSE.

		P (W)	La ($mmol \cdot l^{-1}$)	HR ($b \cdot min^{-1}$)	VO_2 ($l \cdot min^{-1}$)
Mean Values	**CE**	213.2 (42)	4.14 (1.84)	167 (8.6)	3.32 (.59)
	HIIE 20 sec	217.2 (42.2)	5.22 (1.41)	168 (5.7)	3.37 (.58)
	HIIE 4x4 min	214.5 (43.1)	9.83*‡ (2.78)	167 (4.9)	3.19 (.53)
	at LTP_1	130.0 (24.0)	1.73 (0.63)	132 (13.4)	2.30 (.55)
	at LTP_2	241.3 (36.3)	4.17 (1.33)	169 (4.8)	3.60 (.45)
Peak Values	**CE**	213.2 (42.0)	5.54 (3.45)	177 (10.7)	4.03 (.70)
	HIIE 20 sec	340.0 (47.3) *	7.14 (2.48)	181 (6.7)	4.17 (.74)
	HIIE 4x4 min	279.2 (51.8) *‡	12.37*‡ (4.17)	188 (5.7) *‡	4.22 (.67)
	at P_{max} IET	340.0 (47.3)	12.17 (2.50)	190 (4.3)	4.33 (.71)

P, power output; La, blood lactate concentration; HR, heart rate; VO_2, oxygen uptake; CE, continuous exercise; HIIE, high-intensity interval exercise; LTP_1, first lactate turn point; LTP_2, second lactate turn point; P_{max}, maximum power output; IET, incremental exercise test. * significant ($p < 0.05$) difference to CE, ‡ significant ($p < 0.05$) difference to short HIIE

Although this trend that was described above in relation to HR, La, duration, and intensity is established, there is inadequate research completed on longer duration intervals above lactate steady state (LaSS), which this study sought to understand (Tschakert et al., 2015). The majority of the research on HIIT encompasses improvements in maximal oxygen consumption, not acute cardiovascular and metabolic responses (Daussin et al., 2008; Helgerud et al., 2007; Tschakert et al., 2015). These acute responses are of great importance to be researched due to the strong likelihood of them determining the particular muscular and systemic adaptations (healthy and diseased populations) that take place over the course of a training regimen. These responses also help display the potential health risks and benefits to individuals

while performing this type of exercise (Hawley, 2004, 2008; Meyer et al., 1997; Rognmo et al., 2012; Tschakert et al., 2015).

Physiological Acute Responses to Altitude and Intervals

The acute individual responses to altitude and HIIT physiologically, as discussed previously, are amplified when the two said conditions are implemented together with respect to perceived maximal intensity. The conditions of this study aim to present hypoxia (i.e. low oxygen driving pressure) during the interval portion of HIIT and present hyperoxia (i.e. high oxygen driving pressure) during the recovery portion of HIIT. Also, normobaric (i.e. sea-level) conditions were used in order to compare oxygen deprivation versus oxygen hyperoxia after the physiological responses of this study's HIIT protocol's effect on each subject. These combinations helped to establish a knowledge based upon which further research may be conducted, since the literature on the matter is limited (Ploutz-Snyder et al., 1996).

While working at an interval of the present study (i.e. 75% of $VO_{2\,max}$ in watts), the added hypoxia should result in amplified responses (i.e. simulating near or above max) in comparison to normobaric conditions. The acute responses that should be seen under the hypoxic condition of the HIIT are increases in cardiac output (i.e. increase in HR and decrease in SV) and reliance on more anaerobic processes. These effects result in higher lactate (La) levels, lower pH, increased ventilatory compensation, and increased resulting fatigue perception (i.e. increased RPEs during the HIIT trials with hypoxia) (Hill, Long, & Lupton, 1924; Noakes et al., 2001; Ploutz-Snyder et al., 1996). The low driving pressure from the hypoxic environment has a

pronounced detriment on arterial saturation of oxygen. This response, as outlined in Figure 2, will allow for the intensity of the exercise to arise to perceived maximal levels, although working at 75% of $VO_{2\,max}$ during an interval segment (Wilson & Welch, 1974). Few studies have researched the acute effects of hypoxia and HIIT, but recently many studies have begun to understand the long-term adaptions (i.e. 2 to 4 weeks) associated with this type of training (i.e. pro-inflammatory cytokines increase, increase in capillarization, and heart remodeling) (Faiss, Girard, & Millet, 2013; Faiss et al., 2015; Richardson & Gibson, 2015; Richardson, Relf, Saunders, & Gibson, 2016). These studies lay out the inherent need to understand and study the acute responses of HIIT and hypoxia that create these long-term adaptions.

During recovery of a HIIT interval (i.e. 50% of $VO_{2\,max}$ in watts) for our study, a subject experienced either a hyperoxic or normobaric condition. The protocol for the current study is outlined in the next chapter. This recovery segment of the HIIT protocol allowed for the responses brought on by hypoxia and HIIT to decrease (i.e. recover) until the onset of the next HIIT interval. The responses observed should indicate that an improved recovery will occur with the implementation of hyperoxia, then that of a normoxia (i.e. sea level). Observations that should be seen with the added hyperoxia, contrasted against similar intervals of the alternative condition of recovery (i.e. normoxia; sea level), are a lower RPE (i.e. rating of perceived exertion), lower lactate levels, decreased heart rate, decreased cardiac output, and higher muscle oxygen saturation. The physiological interpretation behind these responses are simply due to a greater concentration of oxygen within the arterial blood supply, leading to a reduction of blood being pumped and lower levels of lactate creation.

The amount of oxygen supplementation needed to produce responses, previously discussed, has been researched by numerous studies. A study conducted by Wilson and Welch in 1974 found significance to implicate that at least 40% concentration of supplemental oxygen (i.e. hyperoxic environment) allows for increased performance in regard to duration of the exercise (18% increase). Significant reductions in ventilation were also observed at the same work rate under the hyperoxic condition compared to normobaric conditions, implying increased arterial oxygen saturation. These findings suggest that the hyperoxia condition reduced the physiological metabolic demand of the HIIT workload. Consequently, as expected, each subject perceived the HIIT exercise as less intense, allowing them to complete more HIIT intervals. It was concluded that the increased exercise duration seen in Wilson and Welch's study was primarily related to the greater arterial oxygen saturation stimulated by the supplemental oxygen condition. This finding was so significant that other studies have built upon this and used it in conjunction with HIIT (Linossier et al., 2000; Morris, Kearney, & Burke, 2000). Morris, Kearney, and Burke, demonstrated that power outputs improved with the increase in supplemental oxygen (i.e. hyperoxia), and thus training at a higher intensity could be obtained. Linossier et al., (2000), demonstrated the metabolic impact of hyperoxia (60% oxygen concentration) during maximal cycling exercise. Their results showed glycogen depletion was significantly greater and lactate accumulation was less than normoxia (i.e. 21% oxygen). The data suggests that a higher oxidation rate for pyruvate and NADH in mitochondria exists, which allows for a lower metabolic acidosis (i.e. better functioning of the glycolitic processes that delays time to exhaustion). This study showed a 38% improvement in time to exhaustion under hyperoxia.

CHAPTER 3

METHODS

Subject Demographics

Nine healthy subjects with fitness levels exceeding the 50th percentile of maximal oxygen consumption (i.e. Female > 37.8ml/kg/min for 50 yrs. of age; Male >43.9ml/kg/min for 50 yrs. of age) were recruited to participate in this study (ACSM, 2014). Exclusions for participants was anyone who was outside of the testing age range (i.e. 18-50 years of age), currently had an implanted pacemaker, pregnant or expecting women, medical history of cardiovascular disease, currently had cardiovascular disease, and did not meet the 50th percentile of maximal oxygen consumption.

Subject Qualification Process

The study design and procedures were approved by the Northern Illinois University Institutional Review Board. Figure 8 shows the baseline qualification process. Potential subjects received a study orientation overview and had the opportunity to ask questions. Subjects who then wanted to participate in the study completed an IRB-approved consent document that explained the study requirements, including each subject's participation requirements and risks associated with being in the study. After each subject signed the study's consent, the subject completed a comprehensive medical and exercise history to verify that they were medically eligible to participate as outlined in the consent and in accordance with the guidelines of the

American College of Sports Medicine. Although specific subject characteristics (i.e. height, weight, and body composition) were not required to meet qualification standards, these variables were measured on each subject during the baseline screening process. These demographic variables were used to describe the study's participant's general physical characteristics (i.e. percent body fat to lean muscle mass ratio). Each subject completed a standardized cycling-based maximal oxygen consumption test to ensure each subject met or exceeded the main inclusion criteria for the $VO_{2\,max}$ 50th percentile for their age and gender.

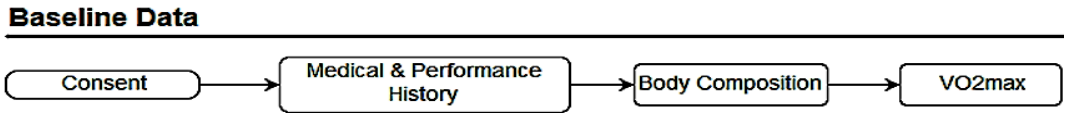


Figure 8: Baseline data qualification procedure.

Experimental Design

All testing was performed at Exercising Nutritionally's physiology performance laboratory in Lisle, IL. The testing sequence was comprised of three treatment sessions and two rest days (Figure 9). Sessions A and C used a randomized matched pair design. Session B was always the second treatment session. For example, a typical match pairing was as follows: Subject 1-Treatment Order = ABC and Subject 2-Teatment Order = CBA.

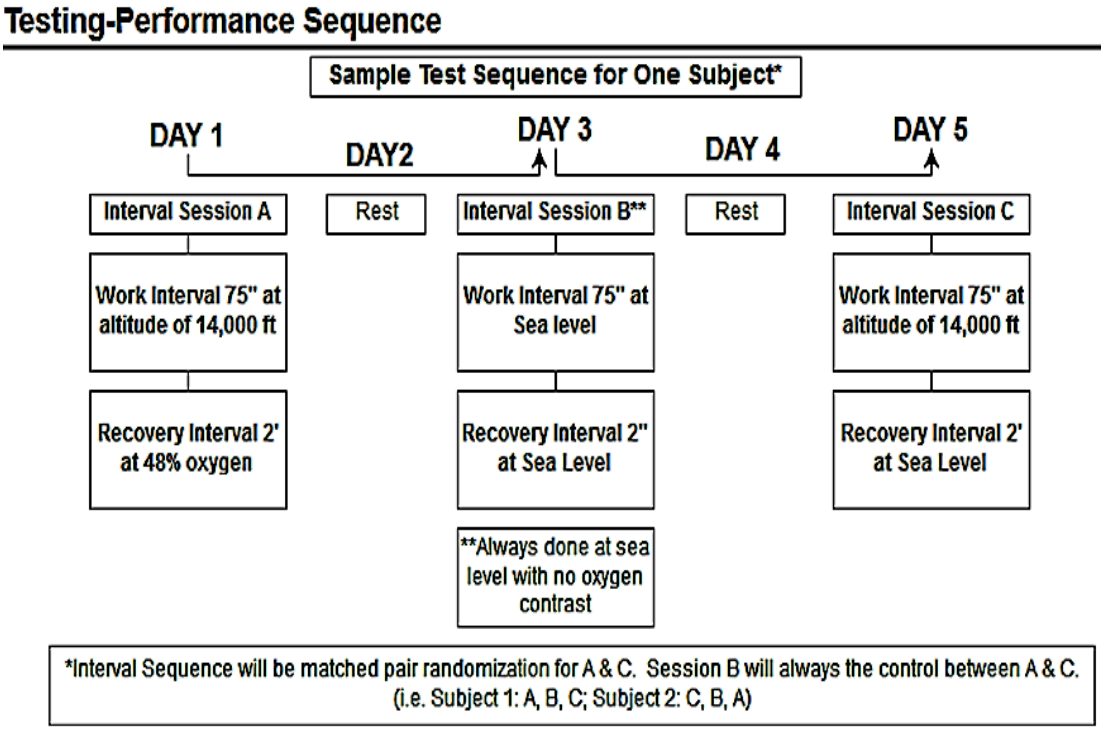


Figure 9: Testing performance sequence.

For this study, the comparisons of the three sessions was the basis by which our conclusions were made. The test sequence allowed us to demonstrate the physiological responses that occur when working at altitude compared with sea-level conditions and the strain that is placed upon the body. The sequence also allowed us to see if the responses and strains of the altitude can be mitigated by the supplementation of oxygen (i.e. 48%) during recovery compared to recovery at sea-level conditions.

Oxygen Contrast and HIIT Trial

Figure 10 and Table 3 illustrate the specific implementation of each session and the variables that were examined. Figure 11 depicts the LiveO2 oxygen contrast simulation machine and setup.

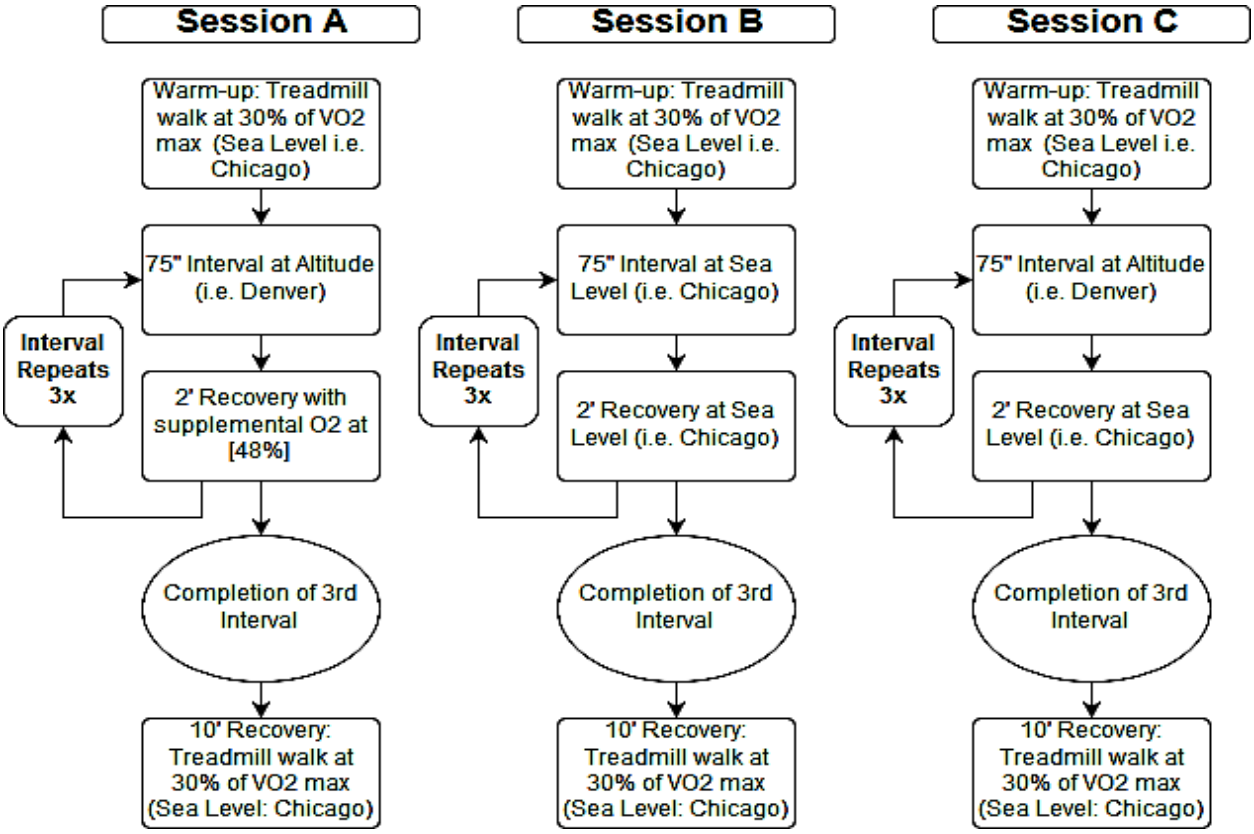


Figure 10: Individual session explanation. **Session A**: Altitude with Hyperoxia Recovery, **Session B**: Sea-Level Verification of Intensity, **Session C**: Altitude with Sea-Level Recovery.

Table 3: Experimental variables. List of variables measured during all HIIT sessions
† - When using the altitude simulation system, a subject cannot be measured simultaneously using a metabolic cart system. Thus, the variables were not directly measured.

Variables to be measured during HIIT sessions	**A**	**B**	**C**
Volume of Oxygen Consumption (ml/kg/min) †		**x**	
-efficiency of the body; cardio, respiratory, metabolism			
Heart Rate (b/min)	**x**	**x**	**x**
-definition of intensity			
Ventilatory Threshold†	**x**	**x**	**x**
-indication of increased lactate production			
Muscle Oxygen Saturation Percent	**x**	**x**	**x**
-quantifies demands and supply of oxygen			
Muscle Hemoglobin Levels	**x**	**x**	**x**
-quantifies blood flow to the area of work			
Blood Lactate	**x**	**x**	**x**
-quantifies anaerobic glycolysis			
Cardiac Output, Stroke Volume, Total Peripheral Resistance	**x**	**x**	**x**
-displays intensity and efficiency of the cardiovascular system			
Ventilation		**x**	
-increases to accommodate metabolism and lactate build-up			
Watts	**x**	**x**	**x**
-increase in intensity for exercise			
Time/ Number of Intervals	**x**	**x**	**x**
-explains the outcomes of trials			

Figure 11: LiveO2 oxygen contrast simulator setup.

Baseline Data: $VO_{2\,max}$ Design and Protocol

$VO_{2\,max}$ was assessed using a standardized cycling protocol on a Monark cycle ergometer (Model #: LC4) as outlined in Table 4.

Table 4: Maximal oxygen consumption study protocol

Stage	Watts	Time (min)
0	0	0:00
1	25	1:00
2	50	2:00
3	75	3:00
4	100	4:00
5	125	5:00
6	150	6:00
7	175	7:00
8	200	8:00
9	225	9:00
10	250	10:00
11	275	11:00
12	300	12:00
13	325	13:00
14	350	14:00
15	375	15:00

During the $VO_{2\,max}$ test, each subject's heart rate, VO2, RER, ventilatory response, cardiac output, stroke volume, total peripheral resistance, regional muscle oxygenation, and total hemoglobin were measured. Moxy near-infrared sensors (i.e. measures regional muscle oxygen saturation (SMO2) and total regional hemoglobin (THb) concentrations) were placed over the midpoint of each subject's medial left and right leg quadricep muscle to determine oxygen saturation changes and break points. Along with the leg sensors, a single Moxy sensor was placed the right carotid artery upon the neck to measure oxygen and blood flow to the brain (i.e. data to supplement the safety of the experiment). Previous research has shown that oxygen saturation break points can accurately predict a person's true lactate threshold (Perry et al.,

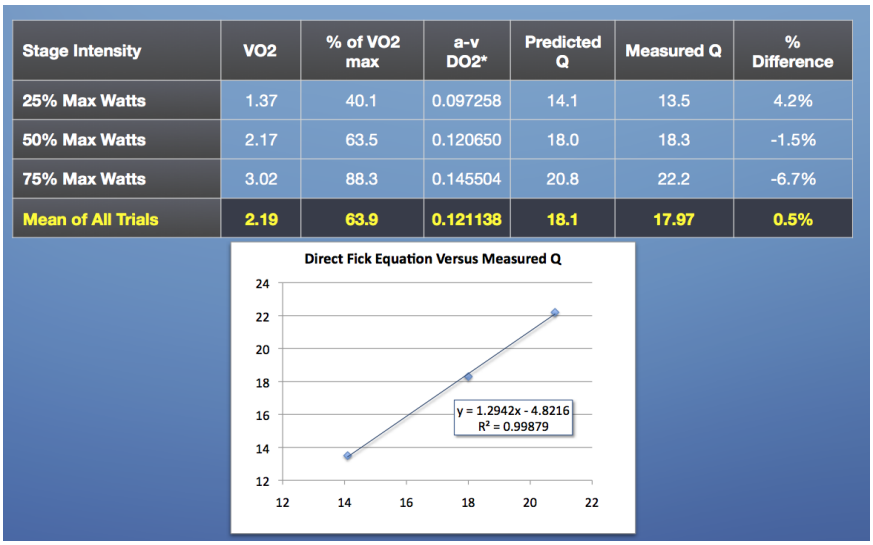
2017). Each subject was fitted for a Hans Rudolph, Inc., V2 maximal oxygen consumption mask connected to a Korr metabolic cart (i.e. VO2, RER, and ventilatory response). The Korr metabolic cart was calibrated as recommended by the manufacturer. The PhysioFlow PF-05 advanced impedance cardiac output measurement system was used to collect cardiac function data (i.e. heart rate, stroke volume, cardiac output, and total peripheral resistance (TPR)). This unit is a non-invasive cardiac monitoring system which provides hemodynamic parameters by measuring thoracic heart blood volumes using advanced electrical bioimpedance signal changes synced to each heart beat's ventricular activation (QRS wave). A back-up monitoring device for heart rate was also used (i.e. MYZONE MZ-3 heart rate chest monitor). Subjects began the prescribed warm-up upon completion of pre-trial preparation. The prescribed warm-up for the $VO_{2\,max}$ test required cycling on a Monark cycle ergometer for 10 minutes at subject self-selected wattage and RPMs. Testing ensued upon completion of the warm-up and ended at volitional fatigue.

The data obtained after the conclusion of the maximal oxygen consumption test was used to establish each subject's OCT intensities. During each of the OCT sessions, subjects performed the 75-second HIIT segments at the wattage equal to 75% of a subject's respective $VO_{2\,max}$. Each subject's recovery intensity was at 50% of his or her $VO_{2\,max}$ wattage. Physiological variables associated at these selected intensities (e.g., cardiac output, stroke volume, oxygen saturation, & heart rate) were key performance markers by which all subsequent trials were evaluated, since VO2 cannot be measured during Trials A and C.

The setup of oxygen contrast simulator (Live O2 Altitude Simulator) when used during trials A and C, simulating high altitude and to deliver oxygen recovery loads, was adapted from

manufactures specifications regarding the mask worn. The mask provided by the manufacture would not coincide with the Korr unit and did not allow for a closed system between the Live O2 simulator and the subject. Instead of the manufacture's mask, the Hans Rudolph mask with Korr attachments was used for all sessions. This also allowed for the subject to have consistent conditions (i.e. pressure on the face, resistance of flow, volume and specifications within the mask) across all trials. Since the direct metabolic and ventilatory measurements cannot be taken at during sessions A and C because of altitude, hyperoxia, and the measurement with the metabolic cart, prediction of VO2 had to be made. The comparison of the overall physiological strain of each respective treatment session was quantified by the primary variables monitored across all trials such as cardiac output, stroke volume, heart rate, and SMO2-THb at both the active muscle (medial quad) and neck (right carotid artery). In addition, blood lactate was measured prior to the start of each trial and immediately after completing the recovery period to further assess each trial's physiological responses. Previous research with the PhysioFlow system had demonstrated that this system can accurately assess exercise intensity during submaximal exercise in relationship to metabolic demand measured by direct oxygen uptake at the same exercise intensities in the current study (Broeder, Hickok, & Burditt, 2009). For example, Broeder et al. (2009) showed that the measurement of cardiac output using the PhysioFlow system in a linear regression had a R-squared value equal to 0.998 with respect to the direct Fick equation. The intensities of 25%, 50% and 75% of max effort were verified and indicated the PhysioFlow system can accurately quantify the HIIT training session with respective physiological demands induced during the high-altitude HIIT and oxygen-contrasting protocols compared to the control trial at sea level (see Figure 12). Each subject's trial exercise loads

during HIIT cycle ergometer sessions were derived directly from the linear relationship between external workload demand set on the cycle ergometer (watts per stage) and each person's oxygen uptake response to that work rate as shown in Figure 13 and estimated wattages in Table 5.



Stage Intensity	VO2	% of VO2 max	a-v DO2*	Predicted Q	Measured Q	% Difference
25% Max Watts	1.37	40.1	0.097258	14.1	13.5	4.2%
50% Max Watts	2.17	63.5	0.120650	18.0	18.3	-1.5%
75% Max Watts	3.02	88.3	0.145504	20.8	22.2	-6.7%
Mean of All Trials	2.19	63.9	0.121138	18.1	17.97	0.5%

Figure 12: Results of Broeder et al. (2009). Reliability of Q correlating with VO2 intensities.

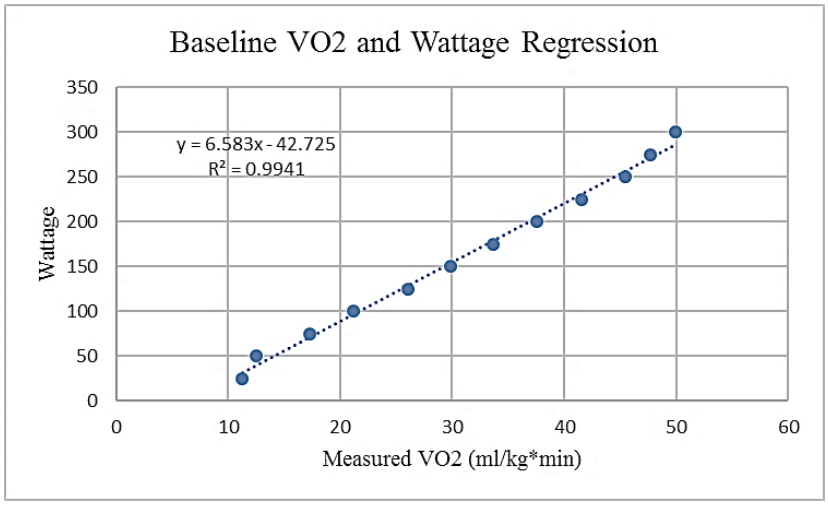


Figure 13: Intensity prediction. Prediction of interval and recovery wattages for Sessions A, B, and C.

Table 5: Estimated wattages for Sessions A, B, and C. Wattages are rounded to the nearest 5 watts.

VO2 and Wattage Establishment				
	Max	**75%**	**50%**	**30%**
VO2	50	37.5	25	15
Wattage	300	205	120	55

HIIT Interval Session Technology Preparation Description

Prior to starting each HIIT interval session, a Moxy SMO2 sensor was placed at the following locations to assess changes in oxygen saturation and THb changes: right quadriceps, left quadriceps, and right carotid artery. Sensor placement allowed direct comparisons of oxygen saturation (SMO2) and THb changes in the muscle directly involved in the cycling exercise (quadricep) and saturation of blood headed to the brain for safety under each testing condition. These comparisons helped distinguish how the altitude simulation and oxygen contrasting affect the working muscle and the vitals of the brain. After proper cleansing skin preparation, several PhysioFlow EKG pads was placed on each subject for PhysioFlow data collection process as shown in Figure 14. A MY-Zone Heart Rate monitor was placed on each subject as a heart rate back-up. Finally, each subject was fitted for Hans Rudolph masks to ensure a snug fit around the face prior to starting the HIIT interval sessions but not be placed on each subject until after the warm-up period was completed.

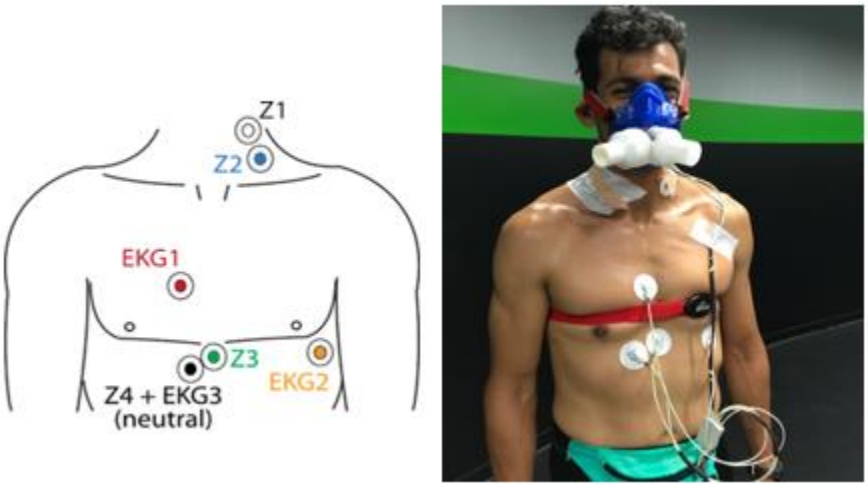


Figure 14: PhysioFlow electrode placement.

The LiveO2 oxygen contrast simulator was implemented for the duration of sessions A and C. Figure 15 depicts the completed setup of the testing setup. The main mechanism of the machine that allowed for storage of supplemental oxygen within the LiveO2 bag utilized a nitrogen extraction filter. The filtration process utilized a fan that pulls air within the unit and does not allow for the larger molecules of nitrogen to diffuse through the filter, only allowing the smaller oxygen molecules to enter the Live O2 bag. The system remained on throughout the sessions to ensure adequate supplemental oxygen was available for use. This also ensured that oxygen escape via backflow through the nitrogen filter would be minimized. The LiveO2 bag was connected to the Hans Rudolph facemask, similar of that used for standard metabolic testing. This mask surrounded the nose and mouth as shown in Figure 14. This allowed for a closed system between the subject and the bag (i.e. no ambient air or normoxia influences occur). With the aid of a contrasting switch located on the bag, a deprivation (i.e. altitude) or supplementation simulation mode could be switched on. This mode closes off the supplemental oxygen compartment and directs the subject's inhalable air to the smaller attached compartment within

the same closed system. As breathing progresses from this compartment, the driving pressure of oxygen decreases similar to the process of ascending to altitude. This process occurs due to the size of the compartment and the amount of oxygen being pumped into the compartment. The physiological challenges to the subject when the altitude simulation chamber is activated is equal to the subject exercising in Aspen, CO.

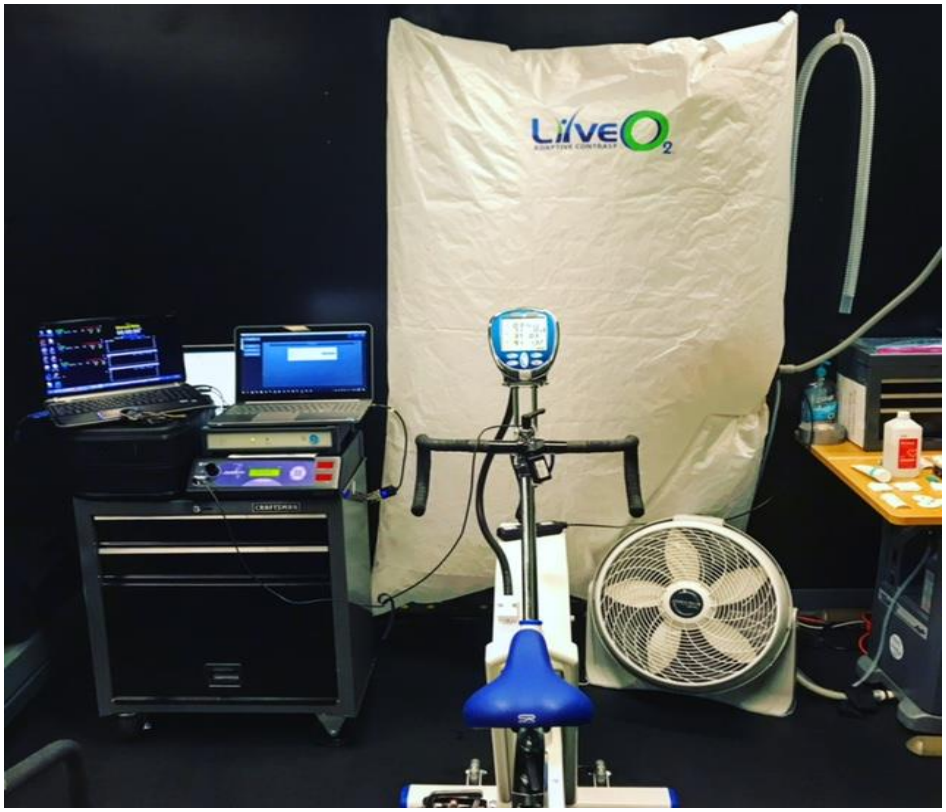


Figure 15: Project setup. LiveO2 oxygen contrast and altitude simulation setup.

Prior to starting each training session, each subject performed a standardized warm-up period before beginning the oxygen-contrasting interval trial segments. The warm-up was executed at 30% of $VO_{2\,max}$ in watts for 10 minutes on the Monark cycle ergometer. This warm-up period was completed under normal sea-level conditions. After the warm-up period was

completed, the Hans Rudolph mask was applied and the experimental protocol began. When the HIIT protocol had been completed, a cool-down session was completed at the same exercise intensity as the warm-up (i.e. 30% of $VO_{2\,max}$ for 10 minutes on a cycle ergometer) to ensure recovery from the physiological strain of altitude and HIIT.

Session A: Altitude with Hyperoxia Recovery

During Session A, each subject performed the HIIT cycle ergometer protocol under the conditions of altitude and supplemental oxygen recovery. The HIIT altitude simulation (LiveO2 altitude simulation unit) segment mimicked the oxygen driving pressure of Aspen, CO, and lasted 75 seconds. After completing the altitude-based interval, each subject completed a 2-minute recovery period with supplemental oxygen (i.e. 48%). Altogether, each subject completed three HIIT recovery combinations as described above. Refer to Figure 10 and Table 3 for a session overview and a complete list of the variables collected.

Session B: Sea-Level Verification of Intensity

During Session B, each subject performed the HIIT cycle ergometer protocol under normal sea-level conditions (i.e. Chicago, IL: 21% Oxygen saturation) for the interval (75 seconds) and recovery segments (2 minutes). This session was implemented to be used as a control condition for comparison of the experimental variables. No matter the treatment order assigned, Session B was always performed second to verify the intensities established from the baseline $VO_{2\,max}$ test. Each subject was asked to complete three of the HIIT intervals and recoveries under the conditions described above.

Session C: Altitude with Sea-Level Recovery

During Session C, each subject performed the HIIT cycle ergometer protocol under the conditions of altitude and sea-level recovery. The HIIT altitude simulation (LiveO2 altitude simulation unit) segment mimicked the oxygen driving pressure Aspen, CO, and lasted for 75 seconds. After completing the altitude-based interval, each subject completed a 2-minute recovery segment without supplemental oxygen (Sea Level Condition, Chicago) contrary to Session A. (i.e. Chicago, IL). Since, normal ambient conditions cannot be applied through the LiveO2 system, each subject had the hose connected to the mask removed for the recovery segment. This opened the closed system and allowed for the sea-level conditions to occur. The hose was reconnected immediately prior at the start of the next HIIT interval. Each subject was asked to complete three of the HIIT intervals and recoveries under the conditions described above.

Data Processing and Statistical Analysis

Each subject's trial data was stored in a secure ENLLC cloud server system. The trial data was postprocessed so that the total physiological demands of HIIT and recovery session could be compared across all three treatment sessions. Metabolic and physiological data for each session's mean full HIIT interval, mean HIIT last 15 seconds, full recovery, and recovery last 15 seconds data were compared between each treatment condition (i.e., sea level only, exercise at altitude with supplemental oxygen recovery, and exercise at altitude without supplemental oxygen). In addition, the metabolic and physiological effects of each HIIT session were compared across each treatment condition comparing similar time points (see Figure 16).

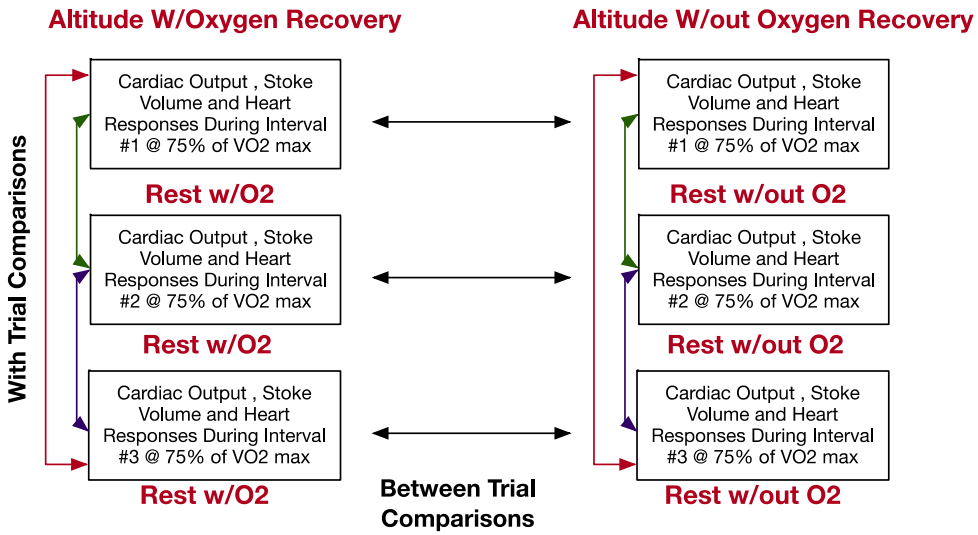


Figure 16: Statistical analysis. A sample of the within and between data variable comparisons for statistical analysis reporting.

Demographic and summary data are presented as the mean, standard deviation, and the minimum/maximum values. A 3 X 3 repeated-measures ANOVA procedure were used to analyze variables within and across each trial. When the ANOVA procedures identified a significant main effect, an orthogonal pair-wise post-hoc procedure will be used to identify its location (i.e., within a given trial period or between a give segment across the three treatments). For those comparisons identified as statistically significant following the post-hoc processing, Cohen's effect size procedures were used to quantify the overall strength of the comparison found to be significant. Effect size is considered good practice and helps facilitate the interpretation of the importance of a statistical finding, as opposed to simply stating that a given comparison is statistically significant. Effect size strength was defined as follows: small $\leq$ 0.20-0.50, medium = 0.51 to 0.79, and large $\geq$ 0.80 (Cumming & Finch, 2001).

CHAPTER 4

STUDY HYPOTHESES

Hypothesis 1–We hypothesized that the physiological responses of Session A (i.e. altitude intervals with supplemental O2 recoveries) in comparison to the same time points of Sessions B and C will exhibit the trends in Table 6.

Hypothesis 2 – We hypothesized that the physiological responses of Session C (i.e. altitude intervals with sea-level recoveries) in comparison to the same time points of Sessions A and B will exhibit the trends in Table 6.

Hypothesis 3 – We hypothesized that the physiological responses of Session B (i.e. sea-level intervals with sea-level recoveries) in comparison to the same time points of sessions A and C will exhibit the trends in Table 6.

Hypothesis 4 – We hypothesized that the verbalized RPE after the completion of Session C will be at or near max (i.e. Borg scale 18-20) compared to the RPE of Session A.

Hypothesis 5– We hypothesized that the measured intensity (i.e. heart rate, cardiac output, post-session lactate) of Session C will be at or near maximal values despite standardized work intensity.

Hypothesis 6– We hypothesized that the O2 saturation percentages after recovery, during Session A, will reach post-warm-up values.

Hypothesis 7– We hypothesized that the O2 supplementation during Session A recoveries will create reliance on more aerobic metabolism indicated by lower post-session lactate levels than Sessions B and C.

Hypothesis 8– We hypothesized that greatest difference in pre vs. post-lactate will occur with completion of Session C.

Table 6: Hypothesized physiological variable responses

Variables	Control Condition (B)		Session A		Session C	
	Sea Level Interval	Sea Level Recovery	Altitude Interval	Supplemental O2 Recovery	Altitude Interval	Sea Level Recovery
Cardiac Output	Increase	Decrease	Moderate Increase	Moderate Decrease	Moderate Increase	Little Decrease
Heart Rate	Increase	Decrease	Extreme Increase	Moderate Decrease	Extreme Increase	Little Decrease
Stroke Volume	Decrease	Increase	Moderate Decrease	Moderate Increase	Moderate Decrease	Little Increase
O2 Sat% Post-Int	Decrease	N/A	Extreme Decrease	N/A	Extreme Decrease	N/A
O2 Sat% Pre-Int	N/A	Increase	N/A	Extreme Increase	N/A	Little increase
TPR	Decrease	Increase	Moderate Decrease	Moderate Increase	Moderate Decrease	Little Increase
Post-Session Lactate	Moderate		Low		High	
Post-Pre-Lactate	Moderate		Low		High	
RPE (Borg)	Moderate	Low	Extreme	Low	Extreme	Moderate
Ventilation	Increase	Decrease	Extreme Increase	Moderate Decrease	Extreme Increase	Little Decrease

CHAPTER 5

RESULTS

Subject Demographics

Subect demographics are presented in Table 7. For this study the total number of participants was seven (i.e. n=7). The subject means coincide well within the norm for the specfic demographic selected and are determined to be an accurate representation of the approved subject requirement.

Table 7: Subject overall demographics

Subject Demographics			
Demographics	**Mean**	**Min**	**Max**
Age	40.9 ± 7.01	26	49
Ht	68.4 ± 4.98	63.0	75.0
Wt	171.3 ± 33.29	121.5	213.0
BMI	25.3 ± 2.72	21.5	30.1
%BF	19.3% ± 7.41%	7.4%	30.2%
FFM	139.4 ± 35.27	94.8	196.9
BFM	32.0 ± 12.17	15.7	48.1

Subject Fitness Demographics

The subject fitness demographics are presented in Table 8. The fitness profile of the subjects tested achieved above the threshold at which the minimum requirement was set (i.e. 50th percentile for $VO_{2\,max}$). Subjects obtained a mean 52.01 ± 7.5 ml/kg/min maximal oxygen consumption which is well above the norm (i.e. 43.9ml/kg/min) for the mean age (i.e. 40.9) of the study participants. Ventilatory threshold and respiratory compensation was determined from

each person's baseline maximal oxygen consumption test. The mean percent of max for these two values is considered normal for the athlete profile tested. Selection of this atheletic demographic was used to promote a safe enviroment due to fitness level for the OCT.

Table 8: Subject Fitness Demographics

Fitness Demographics			
Demographics	**Mean**	**Min**	**Max**
VO2Max ml/Kg	52.01 ± 7.5	41.1	62.4
VO2 Max L/min	4.12 ± 1.174	2.270	6.025
Max HR	170.4 ± 7.8	155.0	183.0
Max RER	1.085 ± 0.13	0.94	1.35
VTH	31.5 ± 5.7	25.3	41.1
VTH %MAX	57.03% ± 7.6%	45.9%	65.9%
RC	43.61 ± 5.5	39.2	55.2
RC % Max	79.05% ± 5.3	72.0%	88.5%

Full Trial Results

Entire trial results are the cumulation data of all the HIIT interval and recovery means for all subjects categorized by session. The first comparison that can be made from the results of the study is the predicted VO2 data and how it compares between Session A, B, and C. Results of this comparison are displayed by Figure 17. The only significance found from the variables collected over the entire trial was heart rate ($p<0.035$). Based upon the Tukey HSD results, Session A (i.e. altitude HIIT/ O2 recovery; mean of 127.25±12.7) was significantly lower compared to the B (i.e. sea-level HIIT/sea-level recovery) and C (i.e. altitude HIIT/ sea-level recovery). Sessions B (130.62±12.36) and C (132.96±11.49) were not significantly different from each other. Expected trends were reported, but no other variables were found significant ($p<0.05$). These trends are displayed by Figures 18, 19, 20, and Table 9. It is also important to note the safety of the overall study in regard to oxygen saturation of blood being pumped to the

brain during the OCT. Figure 21 and Table 9 show that there was no significant difference between sessions and that the desaturation that occurs is minimal. This displays that the exercise with OCT is deemed safe, and blood saturation is not affected for our purposes.

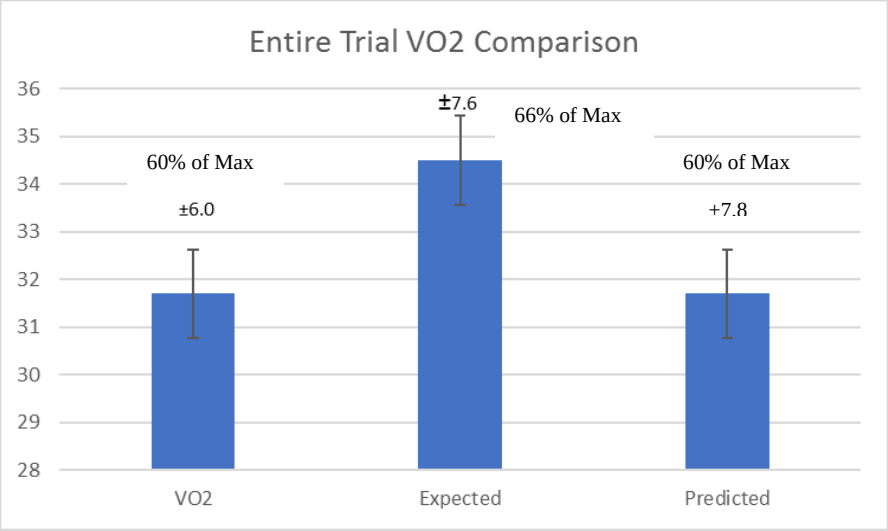


Figure 17: Entire trial VO2 comparison.

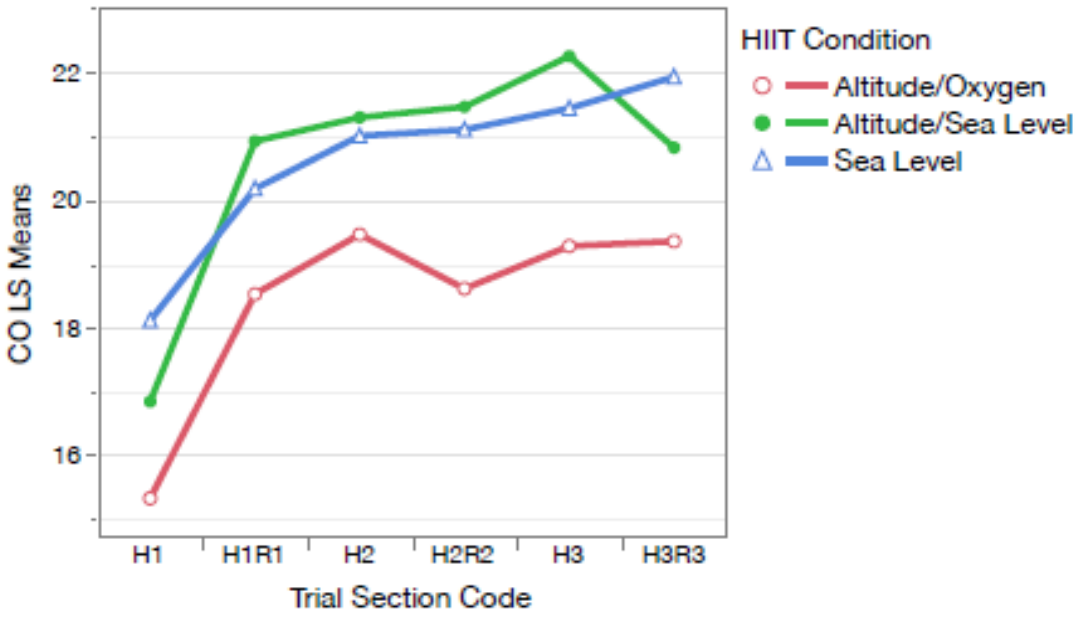


Figure 18: PhysioFlow cardiac output entire trial. H1=HIIT 1, H1R1=Recovery 1.

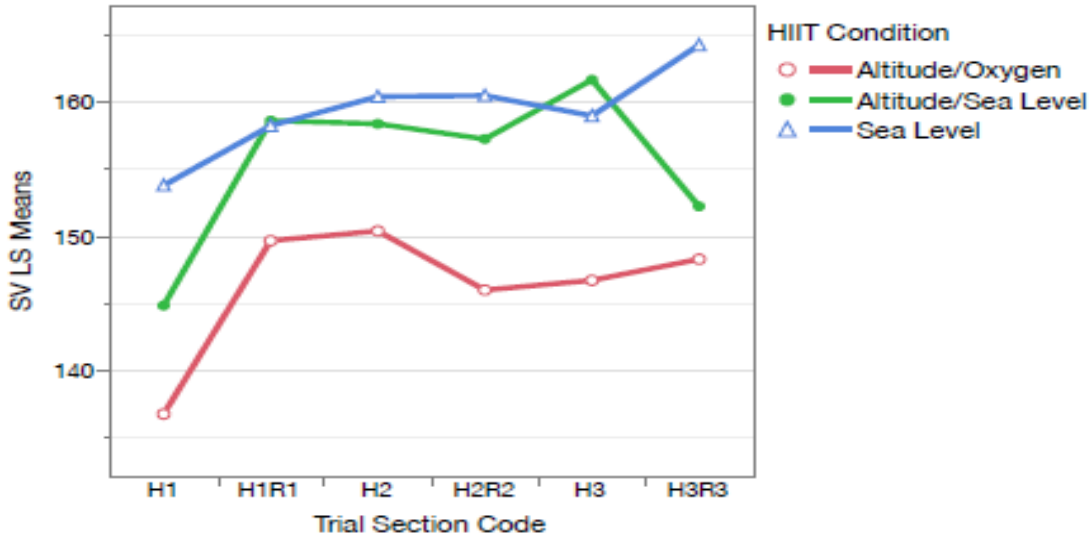


Figure 19: PhysioFlow stroke volume entire trial.

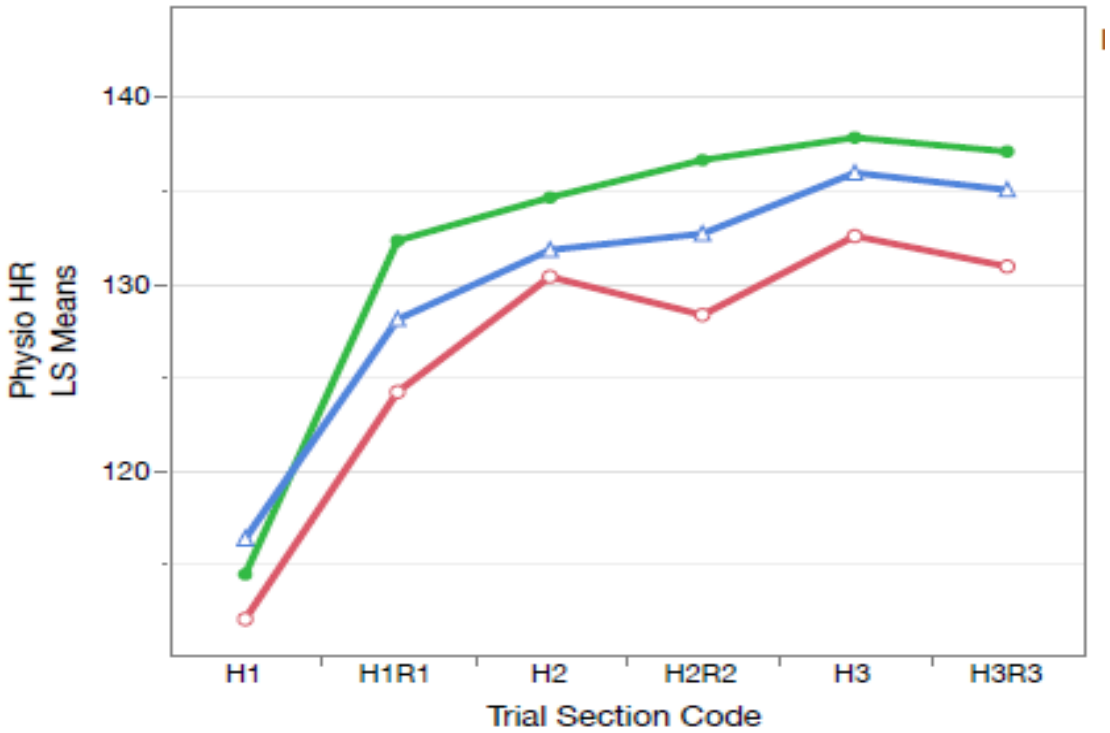


Figure 20: PhysioFlow heart rate entire trial.

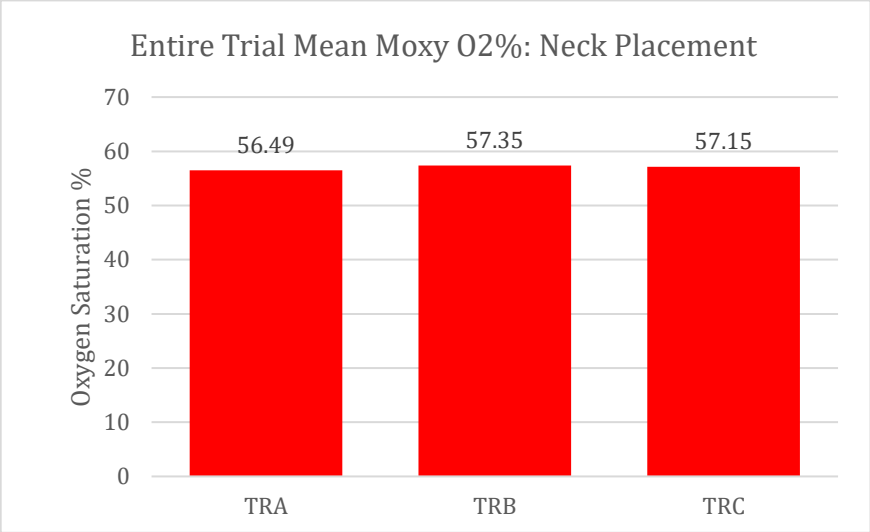


Figure 21: Neck O2% across all trials.

Table 9: Entire trial variable means and significance.

Entire Trial Variables				
Variables	**TRA**	**TRB**	**TRC**	**P-Val**
HR	**127.25±12.7**	**130.62±12.36**	**132.96±11.49**	**0.0348**
SV	146.91±27.45	160.14±21.9	155.70±25.31	0.4938
CO	18.6±3.54	20.81±2.51	20.73±3.65	0.4908
Neck O2%	56.49±16.07	57.35±11.6	57.15±9.25	0.9638
Neck THb	12.95±0.15	12.86±0.15	12.94±0.17	0.4119
LFQ O2%	54.99±20.73	58.27±16.85	48.84±21.88	0.1646
LFQ THb	12.22±0.43	12.24±0.34	12.11±0.34	0.3285
RTQ O2%	58.55±14.71	57.11±9.14	53.16±12.23	0.2537
RTQ THb	12.3±0.38	12.27±0.33	12.35±0.38	0.3695
Overall Sat%	55.99±12.32	57.57±9.07	55.1±10.12	0.4878
Overall THb	12.53±0.3	12.54±0.25	12.43±0.27	0.8777
Deoxy Hb	5.52±1.58	5.29±1.18	5.59±1.29	0.4931

Full HIIT and HIIT Last 15 Seconds

Table 10 displays the results of all HIIT interval data combined within a trial looking at the overall interval trial means across the three treatments. The results indicate that there was no

significant difference observed except for the predicted VO2 ($p<0.029$) or metabolic demand comparisons across the trials. Although there was no significance with variables such as cardiac output and right quadricep oxygen saturation percentage, the presumed effect trend of the implemented treatment did exist. The inclusion of more power via more participants should allow for significance to be found during this time period.

Table 10: Full HIIT variable means and significance

Full HIIT Trial Data				
Variables	**TRA**	**TRB**	**TRC**	**P-Val**
HR	124.98±12.81	128.01±12.77	128.93±13.42	0.59
SV	144.58±29.29	157.7±24.03	154.93±26.53	0.252
CO	18.02±3.89	20.19±3.02	20.13±4.09	0.1040
Neck O2%	58.42±3.06	60.26±2.83	60.11±3.06	0.891
Neck THb	12.94±0.12	12.83±0.15	12.92±0.17	0.072
LFQ O2%	53.29±19.16	59.76±14.68	49.18±19.92	0.169
LFQ THb	12.22±0.44	12.21±0.35	12.11±0.35	0.608
RTQ O2%	56.56±9.4	58.05±5.84	52.78±8.48	0.1000
RTQ THb	12.28±0.41	12.25±0.34	12.34±0.4	0.733
Predic VO2	**24.41±6.54**	**30.36±8.43**	**29.5±7.74**	**0.0290**
Overall Sat%	55.21±9.26	59.36±7.17	55.96±7.36	0.225
Overall THb	12.52±0.30	12.43±0.26	12.42±0.28	0.525
Deoxy Hb	5.61±1.18	5.06±0.95	5.48±0.97	0.23

When the last 15s of each HIIT interval were compared across the three trials, a much clearer picture of how supplemental oxygen and altitude affected the physiology responses to exercise treatment workloads can be seen (Table 11) . The purpose of this analysis was to see the physiological impact of the altitude condition near the end of the HIIT interval period; i.e., did it alter oxygen desaturtation or change the cardiovascular response to completing this exercise protocol at sea level. From these analyses, left quadricep oxygen saturation ($p<0.045$) and right quadricep oxygen saturation ($p<0.011$) for Sessions A and C are significantly lower than the

control of Session B. Overall saturation (i.e the mean of neck, LFQ, and RTQ O2 %) was also found under the altitude conditions (Trials A & C) to be significantly lower than the responses observed during the sea-level trial (Trial B, $p<0.003$). As one would expect, with the mean significant decrease of overall oxygen saturation, deoxygenated hemoglobin was significantly greater during the altitude HIIT trial segments than at sea level ($p<0.004$).

Table 11: Last 15 seconds of HIIT variable means and significance.

Last 15s HIIT Trial Data				
Variables	**TRA**	**TRB**	**TRC**	**P-Val**
HR	137.5±10.65	137.05±10.62	139.78±9.84	0.6591
SV	150.4±31.06	162.57±23.35	160.07±25.35	0.3055
CO	20.44±4.09	22.14±2.62	22.36±3.68	0.1609
Neck O2%	47.91±16.58	56.25±11.19	53.01±10.5	0.1427
Neck THb	12.95±0.18	12.85±0.17	12.93±0.19	0.2125
LFQ O2%	**37.92±20.36**	**51.36±16.9**	**36.25±25.01**	**0.0452**
LFQ THb	12.22±0.49	12.2±0.38	12.09±0.40	0.5541
RTQ O2%	**42.26±9.85**	**49±5.71**	**40.67±10.84**	**0.0105**
RTQ THb	12.29±0.43	12.24±0.36	12.34±0.41	0.7072
Predic VO2	**28.79±5.89**	**34.34±8.73**	**33.49±6..37**	**0.0300**
Overall Sat%	**42.41±9.11**	**52.19±6.92**	**45.59±10.32**	**0.0034**
Overall THb	12.52±0.33	12.43±0.27	12.40±0.29	0.4469
Deoxy Hb	**7.22±1.21**	**5.95±0.93**	**6.76±1.34**	**0.0043**

Full Recovery and Recovery Last 15 Seconds

Similar to the compilation of the HIIT intervals into a mean for each trial, the recovery data is displayed in Table 12. The full recovery allowed for comparison between trials to see if there was an implications to what effect the oxygen supplementation would have upon physiological variables. The only significantly different variables across trials were cardiac output ($p<0.012$), right quadricep oxygen saturation ($p<0.013$), and VO2 ($p<0.004$). Cardiac output (Figure 22) was significantly lower during Session A compared to both Sessions B and C,

indicating supplemental oxidation reduced the metabolic demand of the workload segments and enhanced recovery. Correspondingly, right quadricep oxygen saturation (Figure 23) during session A was significantly greater than the mean oxygen saturation responses during Sessions B and C. As a result, supplement oxygen usage during Session A led to significantly lower post-exercise VO2 uptake and the metabolic demand compared against Sessions B and C (Figure 24).

Table 12: Full recovery variable means and significance.

Full Rec Trial Data				
Variables	**TRA**	**TRB**	**TRC**	**P-Val**
HR	127.8±10.59	131.9±12.30	135.29±9.42	0.0891
SV	147.95±26.19	160.97±19.99	155.99±25.43	0.2165
CO	**18.83±2.89**	**21.07±1.83**	**21.07±3.28**	**0.0124**
Neck O2%	56.07±13.67	53.38±8.84	53.94±7.11	0.6941
Neck THb	12.98±0.16	12.89±0.13	12.97±0.15	0.1161
LFQ O2%	59.27±16.64	57.36±17.11	49.66±19.42	0.1768
LFQ THb	12.22±0.43	12.26±0.34	12.12±0.34	0.4607
RTQ O2%	**63.22±10.3**	**56.83±8.09**	**55.15±8.5**	**0.0128**
RTQ THb	12.31±0.37	12.28±0.34	12.36±0.39	0.7589
Predic VO2	**26.38±4.4**	**32.25±6.88**	**31.04±5.63**	**0.0037**
Overall Sat%	58.71±7.48	55.85±8.38	55.15±7.03	0.3412
Overall THb	12.54±0.29	12.47±0.25	12.44±0.27	0.5675
Deoxy Hb	5.19±1.03	5.52±1.10	5.59±0.94	0.4628

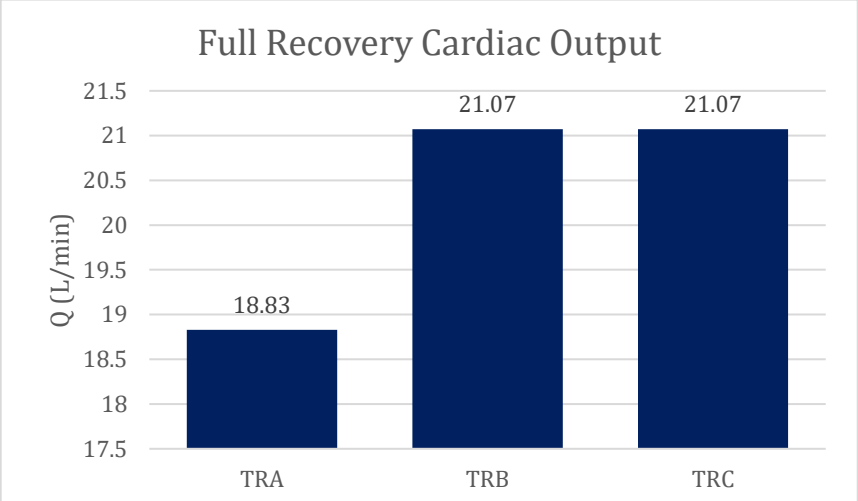


Figure 22: Cardiac output full recovery means across Sessions A, B,and C.

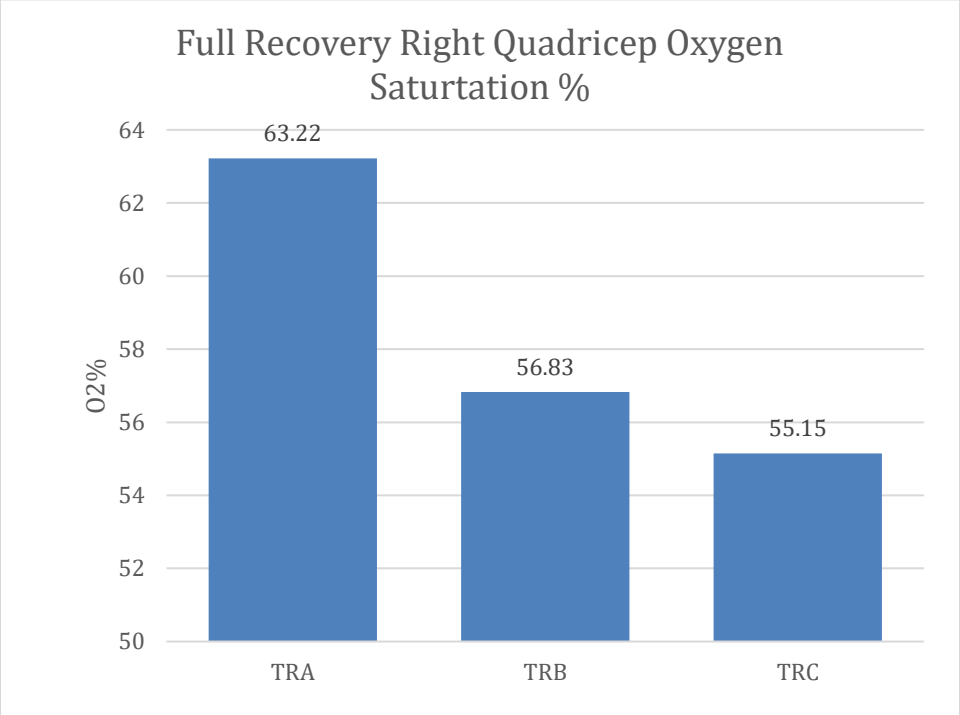


Figure 23: Right quadricep oxygen saturation full recovery means across Sessions A,B, and C.

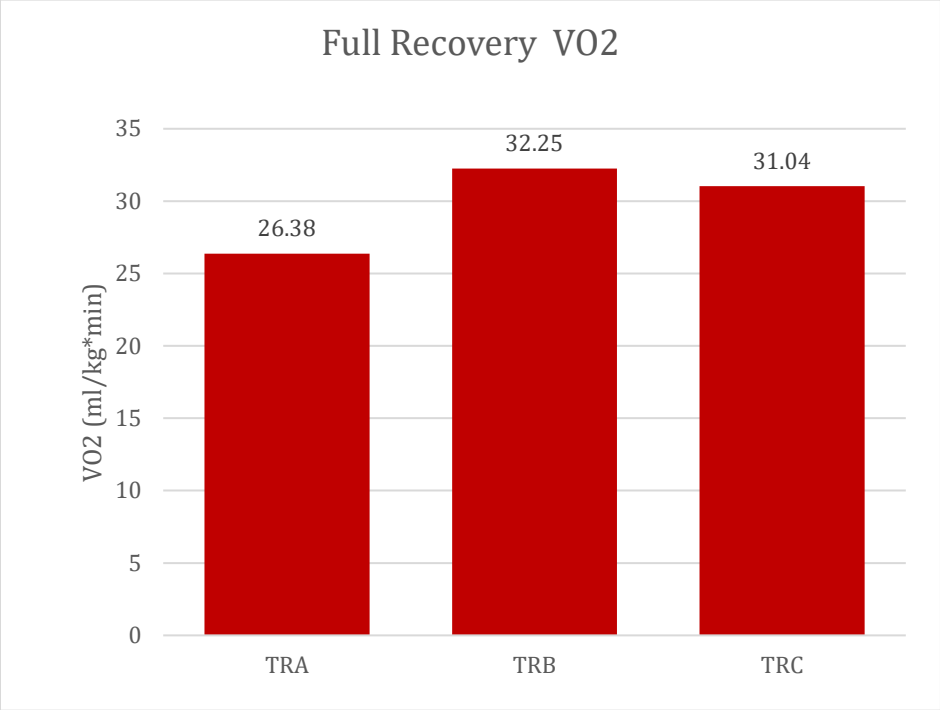


Figure 24: VO2 full recovery means across Sessions A, B, and C.

Similar to the data collected for the last 15 seconds of each HIIT, the last 15 seconds of recovery data were also collected and analyzed. These data allow for the comparison of variables near the end of recovery, where one would assume provides the greatest degree of recovery across each of the trials. Similar to the findings with the last 15 seconds of HIIT, comparing the last 15s of the HIIT recovery sections allows us to observe the distinct effects of HIIT, altitude, and supplement oxygen during the recovery processes. Heart rate was found to be significantly attenuated in Session A ($p<0.012$) when compared to that of Sessions B and C, indicating enhanced recovery and less post-HIIT metabolic demand. Along with a lower HR for Session A, cardiac output was also reduced during Session A ($p<0.002$) when compared to the non-supplement oxygen exercise sessions (B and C). Correspondingly, right quadricep oxygen saturtation was enhanced as a result of the supplemental oxygen in recovery during Session A

($p<0.009$) when compared Sessions B and C. As expected, metabolic demand or VO2 was significantly reduced during Session A ($p<0.001$) compared to Sessions B and C. Table 13 displays the mean results and the corresponding significance.

Table 13: Last 15 seconds variable means and significance

Last 15s Recovery Trial Data				
Variables	**TRA**	**TRB**	**TRC**	**P-Val**
HR	**118.74±9.37**	**125.55±11.31**	**127.83±9.1**	**0.0124**
SV	144.7±24.4	159.32±21.36	151.83±25.08	0.1438
CO	**17.12±2.38**	**19.85±1.89**	**19.35±3.01**	**0.0015**
Neck O2%	63.53±14.65	59.53±12.91	61.8±7.07	0.5884
Neck THb	12.95±0.16	12.85±0.16	12.97±0.18	0.0842
LFQ O2%	69.49±14.43	64.59±17.01	60.28±16.82	0.1887
LFQ THb	12.23±0.39	12.28±0.31	12.13±0.3	0.3113
RTQ O2%	**72.18±10.30**	**64.60±9.19**	**64.03±8.22**	**0.0094**
RTQ THb	12.32±0.35	12.29±0.33	12.37±0.38	0.7858
Predic VO2	**23.35±4.32**	**29.91±6.69**	**27.91±5.48**	**0.0011**
Overall Sat%	67.65±7.80	62.91±10.24	64.20±5.86	0.2041
Overall THb	12.54±0.27	12.48±0.24	12.47±0.25	0.6804
Deoxy Hb	4.07±1.05	4.64±1.33	4.47±0.80	0.2713

CHAPTER 6

DISCUSSION

Purpose Revistited

The study investigated the acute physiological responses of altitude simulation during high-intensity interval training and the subsequent supplementation of oxygen to potentially facilitate greater recovery. One of the main purposes of the study was to expand the lacking literature on the acute responses of altitude, HIIT, and oxygen supplementation that might possibly lead to chronic adaptations as overall HIIT workload is increased. Research currently existing in relation to the current study (i.e. hyperoxia and sprint intervals) has shown improvements in power output achieved by lower heart rates at preset intensities under hyperoxic conditions (Nummela, Hämäläinen, & Rusko, 2002; C. G. Perry, Reid, Perry, & Wilson, 2005). This seems to coincide with our findings, although power output was not measured. Under the condition of altitude and supplemental oxygen, the physiological strain seemed to be reduced and is in accordance with the discussed research findigs. Besides the supplemental oxygen condition, our study supports these previous results across multiple physiological domains, time points, and variables. Contrasting research recently completed involving oxygen supplementation compared against altitude during an increasing incremental maximal load showed no change in the submaximal cardiovascular responses of this test (Bell & Subudhi, 2017). The current study design allowed us to easily control for which condition had an impact on recovery, i.e., altitude,

HIIT work, supplemental oxygen recovery. The purpose of Session B (i.e. sea-level HIIT/ sea-level recovery) was not only for control reasons but allowed us to verify the intensity. It also allowed us to observe what the normal recovery would be for our protocol. Session C (i.e. altitude HIIT/ sea level recovery) allowed us to measure the effects altitude had on HIIT interval physiological demand and its effect on the recovery processes. Finally, Session A (i.e. altitude HIIT/ O2 recovery) provided us with the measurable data to quantify if supplemental O2 would facilitate greater recovery than that of normal conditions (B) and normal conditions after altitude exercise (C).

Entire Trial Significance

Cardiovascular data showed the most promise due to the ability to easily collect and analyze multiple facets of the cardiovascular system. We observed, across the entire HIIT data collection period, heart rate responses were reduced with the addition of a hyperoxic (A) environment post-exercise at altitude compared to normal recovery at normobaric conditions (B and C) ($p<0.035$). This finding suggests that supplement oxgyen reduces metabolic demand and enhanced recovery compared to the non-supplemental trials. If the metabolic and cardiovascular demand on this system is reduced, performance will increase (Adams, Cashman, & Young, 1986; Morris et al., 2000). Although respiratory data could not be collected, we can infer based upon the literature existing and the significance with reduced heart rate that respiratory and metabolic demand would decrease with the addition of supplemental oxygen.

Because this is the first study to look at HIIT intervals with altitude in non-acclimatized subjects, it was important we determined what effects HIIT altitude training in our population of subjects had on maintaining a safe exercise environment. Previous research has shown HIIT

training to be a very safe exercise modality in special populations and patients. But, to date no one has determined if HIIT with altitude conditions reduced the safety of HIIT training. Based on the neck oxygen saturation or blood flow changes across all three trials, the brain oxygenation and blood flow, as well as cardiovascular changes, indicated that performing HIIT at altitude with and without supplemental oxygen did not create an unsafe exercise environment for the current subject population. Our results are in contrast to previous research published (Banchero et al., 1966; Roach, Greene, Schoene, & Hackett, 1998). The findings from previous studies show that with the exposure to altitude, arterial blood saturation imparement occurs. However, it is important to point out that we did not directly measure arterial blood gas, which may account for the between-study differences. With our study showing no significant difference between sessions of altitude, this demonstrates that the amount of exposure to altitude for each subject is not long enough to cause significant changes in desaturation levels that increased the overall exercise risk of our subjects (i.e. even when exaggerated by exercise).

HIIT Intervals Significance

In regards to the HIIT component of the trial (i.e. full HIIT and last 15 seconds of HIIT), one would only expect that the significance would be in the form of the two altitude conditions being different than that of the sea-level control. The significance that was observed supported this hypothesis as expected. With full HIIT (i.e. the mean of all HIIT segments compared to each condtion), supplemental oxygen reduced the metabolic demand of the HIIT intervals. Similar research supports this claim that hyperoxia , although not inhaled during the HIIT, improves VO2 efficiency by enhancing artial oxygen saturation (Astorino & Robergs, 2003; C. G. Perry et

al., 2005). Along with the uneven work-to-rest ratio of our study, having the extra time under the exposure to hyperoxia should allow for the same response (i.e., lower VO2) to occur when not breathing the supplemental O2 for the HIIT. Multiple variables were close to significance, such as right quadricep oxygen saturation ($p>0.100$). With more completed subjects, we believe that signifcance would likely occur and show higher mean saturation values for Session A.

With respect to the last 15 seconds of the HIIT intervals, the true effects of altitude were more clearly observed. The rational for this significance is that the comparison time frame is the point at which the physiological variables should be at their highest or most demanding point in the trial (Åstrand et al., 1960; Tschakert et al., 2015). With the analysis of the last 15 seconds, we see significance in left ($p<0.045$) and right quadriceps ($p<0.011$) O2%, VO2 ($p<0.030$), overall O2% ($p<0.003$), and deoxygenated hemoglobin ($p<0.004$). These findings show that altitude increased a subject's exercising metabolic demand and enhanced the need for a recovery stimulus. Previous research shows that oxygen saturation is reduced within the blood due to the inability to saturate within the lungs when a person performs intense exercise at altitude compared to that same workload at sea level (Bärtsch & Gibbs, 2007; Hsia, 2001; Moudgil et al., 2005; Peacock, 1998). Interestingly, we also observed, based on each subject's deoxygenated hemoglobin SMO2 muscle measurements, that each subject's muscles were extracting O2 at a greater level when exercising at sea level and altitude and not recovering with supplemental oxygen. It is important to note that the metabolic demand (VO2 uptake) with supplemental oxygen was significantly less than Sessions B and C. This further demonstates the effect that the oxygen supplementation during the recoveries had on the actual work demand of the HIIT intervals.

Recovery Interval Significance

Similar to the comparisons made for the time frames of the HIIT intervals, recovery analysis resulted in showing the true effect of HIIT intervals at altitude and sea level with and without supplemental oxygen recovery. The significance of the full recovery means clearly showed that cardiovascular metabolic demand was reduced (cardiac output, $p<0.012$; VO2, $p<0.004$) through an enhancement in the maintenance of oxygen saturation during the recovery process (right quadricep O2%, $p<0.013$). The practical significance of these results is that the subjects could resaturate better with more oxygen available, expend less energy, and have less demand on the cardiovascular system as previously shown in past research (Chick, Stark, & Murata, 1993; Linossier et al., 2000; Naeije, 2010; C. G. Perry et al., 2005). These findings were further verified when we looked at the final 15s of each HIIT recovery segment. With or without supplemental oxygen, one would expect the greatest recovery of the session to be as close to the end of the recovery segment. With the analysis of this time domain, the results indicated, as discussed above, supplemental oxygen enhanced cardiovascular and metabolic recovery when comparing the supplemental oxygen trial to the other two sessions: Session A versus B/C decreased heart rate ($p<0.012$), decreased cardiac output ($p<0.002$), enhanced right quadricep O2% ($p<0.009$), and reduced VO2 ($p<0.001$). These results agree well with previous research comparing supplemental oxygen on cardiovascular function and metabolic demand (Linossier et al., 2000; Morris et al., 2000; Naeije, 2010). In summary, these results show that supplemental oxygen following altitude-based HIIT training reduces the overall metabolic and cardiovascular demand during the actual intervals through the enhancement of the recovery process.

Practical Significance of Results

The significance of the data collected in this study has a variety of practical uses. First and foremost, the literature on the subject matter of altitude, HIIT, and oxygen supplementation is greatly expanded in the discipline where the literaure is lacking. Literature currently published lacks the combination of HIIT, altitude, and supplemental oxygen for recovery. The majority of research articles have two of the three variables mentioned. Far too many are focused on the steady-state singular responses to altitude and supplemental O2 at submax intensities or have too short a duration to actually promote a significant physiological effect (Armstrong, Jacks, Sowash, & Andres, 2000; Bell & Subudhi, 2017). Also, many studies are directed towards the long-term adaptations, when the acute responses for HIIT alone have yet to be consistantly established due to the subject and HIIT protocol variability (Gibala et al., 2012; Ploutz-Snyder et al., 1996; Tschakert & Hofmann, 2013; Tschakert et al., 2015). Second, the use of this methoodolgy can be implemented in cardiovascular rehabilitation facilities that already incorperate HIIT training to facilitate greater adaptation following post-surgical rehabilitaion (Bärtsch & Gibbs, 2007; Guiraud et al., 2012; Hsia, 2001; Naeije, 2010; Weston, Wisløff, & Coombes, 2014). The use of oxygen suplementation in these protocols will reduce the overall risk in these clinical populations while enhancing the potential to perfrom higher workloads, whether that be working at a higher percent of max or performing a longer duration interval at the prescibed percent of max (Gibala et al., 2012; Gibala et al., 2006). With the results from this study, it is in all likelihood that this treatment will facilitate greater recovery. Last, the performance aspect for atheletes using this treatment can be improved. Atheletes training with

the Live High Train Low programming can utilize O2 recovery for longer work at the altitude accustomed to in order to facilitate increased workload. Atheletes training with the Live Low Train High programming can utilize the O2 to facilitate greater workloads at altitude in preparation of an event at a unaccustomed altitude (Vogt & Hoppeler, 2010; Wehrlin, Zuest, Hallén, & Marti, 2006).

Future Considerations

There are many future considerations for expanding the knowledge within the disipline of altitude, HIIT, or oxygen supplementation. One specfic consideration would be an 8-week training program with the protocol implemented in this study. With this study, one could measure the chronic adaptations that take place with the combination of altitude, HIIT, and oxygen supplementation. Variables that could be collected include hematocrit levels and muscle biopses (i.e. fiber type, mitochondrial density, metabolic enzymes). Another consideration would be to keep the protocol similar but manipulate the recovery time or intensity of the HIIT interval to investigate if there are further significances.

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