THESIS

INFLUENCE OF EXERCISE MODALITY AND MODALITY-SPECIFIC TRAINING ON ENDURANCE EXERCISE PERFORMANCE IN HYPOXIA

Submitted by

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ABSTRACT

INFLUENCE OF EXERCISE MODALITY AND MODALITY-SPECIFIC TRAINING ON ENDURANCE EXERCISE PERFORMANCE IN HYPOXIA

INTRODUCTION: In hypoxia, endurance exercise performance is impaired. The magnitude of impairment may be variable between individuals and exercise modalities. The purpose of this study was to determine the influence of exercise modality and modality-specific training on hypoxia-mediated performance decrements.

METHODS: In a randomized cross-over design, endurance trained cyclists (4 males, 3 females) and rowers (5 males, 3 females) performed exercise on both cycling and rowing ergometers. On separate occasions, participants completed graded exercise tests in normoxia (FiO_2= 0.21), and standardized exercise (15 minutes, 100 W) and time trials (4 km cycling, 2 km rowing) in normoxia and hypoxia (FiO_2= 0.15).

RESULTS: Hypoxia-mediated performance decrements were not different between cyclists and rowers (17±1 vs. 18±1%, p=0.189), cycling and rowing (18±2 vs. 16±2%, p=0473), or any combination of training or test modality (p=0.138). In rowers, peripheral oxygen saturation (SpO_2) was lower at the end of rowing compared to cycling time trials (78±1 vs. 83±1%, p=0.002), and lower than that of cyclists at the end of rowing time trials (78±1 vs. 83±1%, p<0.001).

DISCUSSION: Hypoxia-mediated performance decrements were not different between training modalities, test modalities, or any combination of the factors. We speculate that

reduced SpO_2 in rowers at the end of rowing time trials may be related to a greater active muscle mass, causing a rightward shift in the oxyhemoglobin dissociation curve and reduced transit time of blood in pulmonary capillaries. In conclusion, SpO_2 may be related to active muscle mass during exercise and could potentially modulate performance in hypoxia.

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TABLE OF CONTENTS

CHAPTER I

LITERATURE REVIEW

Hypoxia-mediated performance decrements

With acute exposure to increasing altitude/hypoxia, endurance-exercise performance is progressively attenuated. Hypoxia-mediated performance decrements have been observed both in laboratory and controlled field-based experiments (Amann et al., 2006; Gore et al., 1996; Romer et al., 2007), and by comparing race times of athletes at sea-level to their race-times at higher altitude [4].

Prime examples of the effect hypoxia has on performance were the 1968 summer Olympic games in Mexico City at 2,240 m (7,350 ft.), during which, surprisingly slow race times were recorded for many endurance-type events. In particular, athletes of rowing events seemed to be the especially affected as many cases of dyspnea-related loss of consciousness were reported of rowers and some teams could not even finish their event [6]. Failing to complete a race for any reason, save for mechanical failures, is atypical of world-class athletes, and clearly demonstrates the considerable adverse effects of altitude and hypoxia on endurance- exercise performance. However, the effects of hypoxia are not universal and vary between individual athletes and different events, depending on factors including the degree of hypoxia [7], type/duration of exercise [3], and the athlete's aerobic capacity [5,8]. As such, the strategy used to maximize performance in one situation, may not be appropriate in another.

To aid professionals and athletes in preparing for and maximizing performance at altitude, this study aims to elucidate how factors such as exercise modality and training specificity may influence hypoxia-mediated performance decrements. In order to understand

the interaction between these factors and performance in hypoxia, proper context is gained by reviewing the physiological effects of hypoxia on endurance exercise and the differences between cycling and rowing.

Hypoxia and maximal aerobic capacity (VO_{2max})

Endurance exercise, or any event beyond two minutes in duration is heavily dependent on aerobic metabolism [3]. Therefore, there is a positive relationship between performance and one's VO_{2max} [9], and hypoxia-mediated performance decrements are proportional to the total energy provided through aerobic pathways[3].

As altitude increases and the pressure of inspired oxygen (PiO_2) falls, VO_{2max} is reduced at a rate between five to seven percent for every 1000m gained [3], with significant reductions becoming apparent at altitudes as low as 580m [5]. Across various degrees of hypoxia, the metabolic cost of a specific exercise at a given workload does not change (Figure 1.1). Therefore, a reduction in VO_{2max} either limits maximal power achievable through aerobic pathways or increases the relative difficulty of submaximal exercise because the O_2 cost of a given workload represents a greater percentage of the reduced VO_{2max} [10].

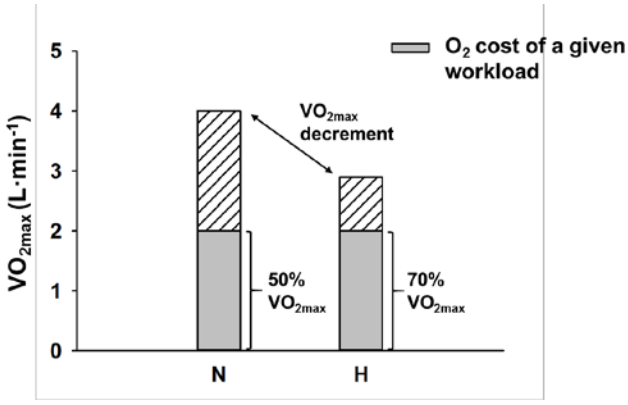

Figure 1.1. VO_{2max} is reduced from normoxia (N) to hypoxia (H), while the metabolic (O_2) cost of given workload does not change. Thus, the O_2 cost of exercise in hypoxia represents a greater percentage of VO_{2max}, increasing the relative difficulty (adapted from McArdle, et al., 2010).

The reduction of VO_{2max} subsequently entrains a proportional reduction of anaerobic threshold [8]. In hypoxia, as exercising muscles reach their capacity to produce aerobic power, a greater proportion of type II muscle fibers and anaerobic metabolism are required to achieve the same power output produced in normoxia [11,12]. However, anaerobic metabolism is not sustainable for long durations. Thus, in hypoxia, either power output must be scaled down or high-intensity exercise will lead to premature fatigue and exercise cessation.

Reduced VO_{2max} in acute hypoxia is a direct result of decreased oxygen delivery (DO_2) to exercising muscle, or the product of arterial oxygen content (CaO_2) and blood flow (BF) ($DO_2 = CaO_2 \cdot BF$). In the investigation of hypoxia-mediated performance decrements, it is necessary to examine both variables.

Decreased PiO_2 may account for approximately 41% of the reduction in CaO_2 [13], but there are other important factors that also affect CaO_2. Once air is inhaled into the lungs, the next stage in respiration is pulmonary gas exchange in which O_2 diffuses across the respiratory membrane into the circulation and CO_2 is removed. At rest, the alveolar to arterial O_2 tension difference ((A-a)DO_2) is very low (0-4 Torr), indicating efficient gas exchange [14]. However, (A-a)DO_2 begins to widen (15-20 Torr) at the onset of exercise and is exacerbated by increasing intensity [15] and decreased FiO_2 [13]. Although ventilation perfusion mismatching occurs during high-intensity exercise, its effect on (A-a)DO_2 is not substantial in acute hypoxia [16] and other research indicates that gas exchange during intense exercise in acute hypoxia is primarily impaired due to diffusion limitation [17].

At rest in normoxia, the diffusion rate of O_2 in the lungs is such that O_2 tension (PO_2) quickly reaches equilibrium between alveolar air and blood. As exercise intensity increases, cardiac output ($\dot{Q}$) increases, reducing red blood cell transit time in the pulmonary circuit [15]. In some cases, in highly trained athletes with high $\dot{Q}$ and greater muscle mass recruitment, red blood cell transit time is reduced to less than the 0.35 seconds necessary for complete equilibrium in normoxia [18], leading to exercise induced arterial hypoxemia (EIAH) [19] (Figure 1.2).

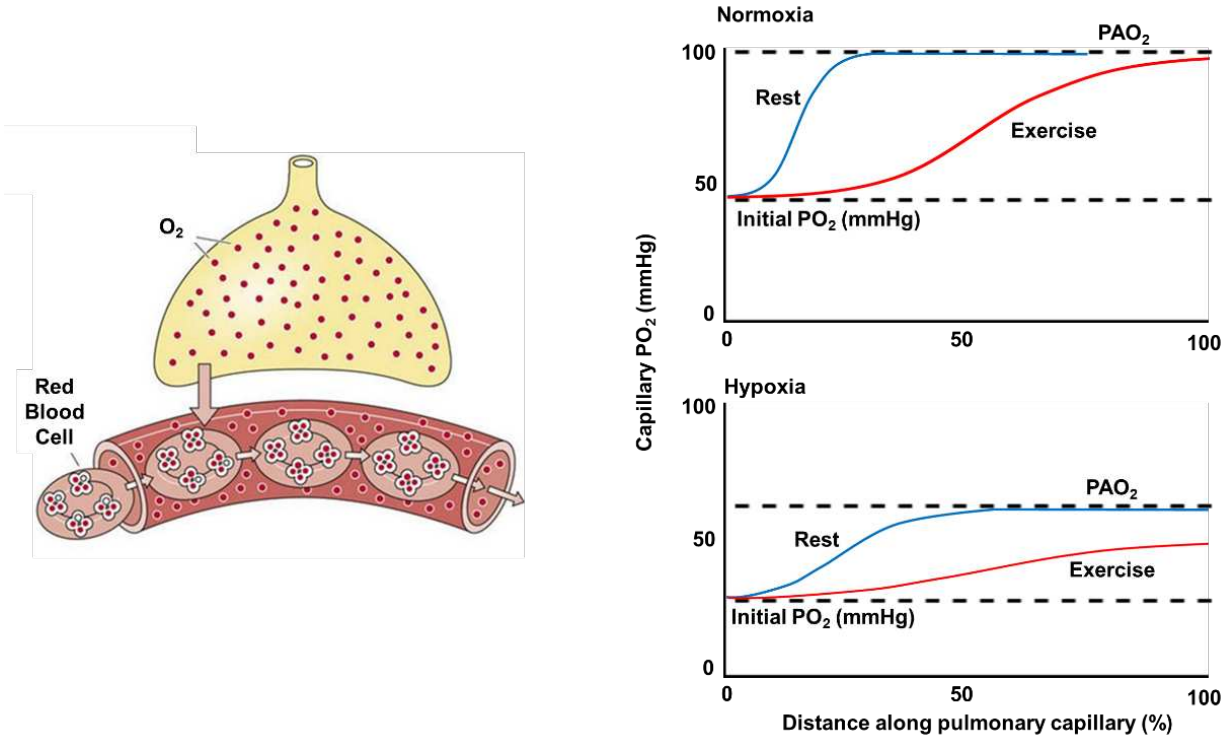

Figure 1.2. Capillary Oxygen tension (PO_2) along the distance of a pulmonary capillary at rest (blue lines) and during exercise (red lines). In normoxia (top), when the PO_2 range is higher, blood entering the pulmonary capillary equilibrates with alveolar PO_2 (PAO_2) quickly. However, during high-intensity exercise, increasing cardiac output (Q) limits red blood cell transit time in pulmonary capillaries and equilibration may not occur before the end of the pulmonary capillary. At altitude (bottom), the diffusion limitation during exercise is even more prominent due to a lower PO_2 range (adapted from Boron & Boulpaep, 2011).

Still, in normoxia, PO_2 remains close to the plateaued upper-section of the oxyhemoglobin dissociation curve and EIAH typically only leads to mild to moderate arterial desaturation (88-92% SaO_2) [18]. However, in hypoxia, when resting PO_2 is closer to the steep portion of the oxyhemoglobin dissociation curve, high-intensity exercise may lead to severe desaturation (<88% SaO_2) [17] (Figure 1.2), resulting in substantially reduced VO_{2max} and endurance exercise performance [5].

Additionally, CaO_2 is influenced not only by PO_2, but by [hemoglobin] and factors such as pH, PCO_2 and temperature. Athletes recruiting greater relative muscle mass or exercise models that require greater muscle mass may experience greater blood pH and temperature disturbances, causing a greater right-ward shift in the oxyhemoglobin dissociation curve [20] and thus further reduced CaO_2. In accordance, EIAH in hypoxia is greater in more aerobically fit individuals [5,7] or exercise models that require greater relative muscle mass [20], such that decrements in VO_{2max} may be commensurate with the amount of muscle mass that is recruited. It is important to note that more aerobically fit individuals still achieve greater VO_{2max} and generally better performance. However, a disparity in physical fitness or muscle mass recruitment, and varying degrees of EIAH between athletes could, in part, explain differences in hypoxia-mediated performance decrements.

The next important factor dictating VO_2 is the amount of oxygen that is delivered to exercising muscle (DO_2, again, the product of CaO_2 and BF) [21]. Muscle BF closely matches metabolic demand during rest and exercise [22] and has a linear relationship with workload without an obvious plateau (Andersen & Saltin, 1985). However, maximal $\dot{Q}$ is limited: healthy males may reach ~30 $L \cdot min^{-1}$ [22–24] and highly trained athletes may reach ~36 $L \cdot min^{-1}$ [25]. Still,

maximal muscle BF may reach values between 300-400 $mL \cdot min^{-1} \cdot 100\ g^{-1}$ [26–28]. This means that if all muscle were maximally perfused, cardiac output would be outstripped by the demand [22,28]. For example, to maximally perfuse all the muscle of an athlete with 30kg lean muscle mass, the necessary $\dot{Q}$ would be ~90-120 $L \cdot min^{-1}$ (~3 times higher than physiological values). So, as cardiac output nears maximal values, vascular conductance is restrained in order to maintain tolerable systemic perfusion pressure and adequate BF to vital organs [29]. Therefore, VO_{2max} and performance in any condition are limited by $\dot{Q}$ and the breadth of BF distribution.

During exercise in acute hypoxia, CaO_2 is reduced and maximal $\dot{Q}$ is attenuated [23,30] and in combination result in reductions of VO_{2max} in hypoxia. The reduction of maximal $\dot{Q}$ in hypoxia is not thoroughly understood, but may be mediated by factors including autonomic changes or myocardial dysfunction [31]. In the face of reduced CaO_2 and $\dot{Q}$, BF may be withheld from exercising muscle in attempt to maintain adequate DO_2 to vital organs, leading to attenuated aerobic exercise capacity. However, certain exercise models may allow adequate DO_2 in hypoxia if there is sufficient BF or reserve $\dot{Q}$. For example, research has shown that in hypoxia, adequate DO_2 is possible during exercise of low intensity [10] or a relatively small muscle mass [20] (Figure. 1.2). Therefore, differences in hypoxia-mediated performance decrements may exist between exercise modalities with different BF or $\dot{Q}$ requirements based on differing relative intensities and muscle mass recruitment.

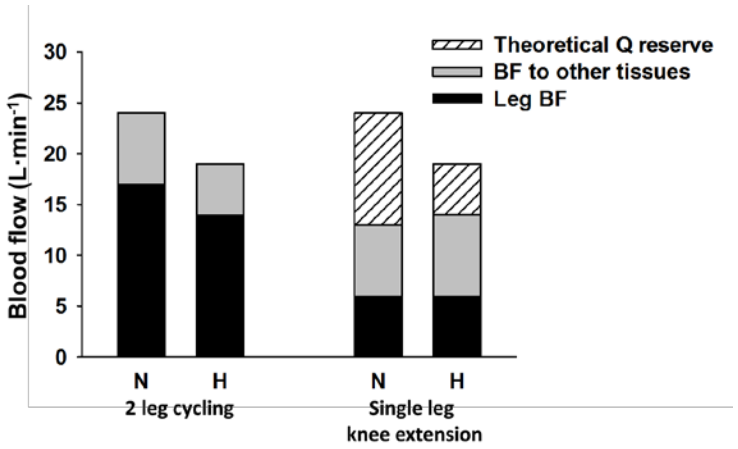

Figure 1.3. Maximal cardiac output (Q̇) is reduced from normoxia (N) to hypoxia (H) and limits blood flow (BF) to exercising muscle and other tissue. However, exercise with a relatively smaller muscle mass does not require maximal Q̇ and in hypoxia, there is adequate reserve Q̇ to maintain BF to exercising muscle and other tissue (adapted from Calbet, et al., 2009).

Cycling and rowing

Cycling and rowing are examples of non-weight bearing and rhythmic aerobic exercise. Participation in both modalities ranges from recreational to world class competition. While some similarities exist between the two modalities, the unique characteristics of each modality result in different physiological responses and subsequently, distinct training adaptations. Between the two modalities, obvious differences are 1) muscle groups engaged, and 2) technique. Cycling exercise primarily engages muscles of the lower body with most of the forces produced in the quadriceps muscle group [32]. Rowing also relies principally on force produced in the lower body but additionally engages muscles of the trunk and upper extremities [33]. During competition (cycling 4-km or rowing 2-km races), aerobic metabolism contributes 70-80% of the total energy during both cycling and rowing [34–37].

Cycling is a relatively simple movement pattern that is easily learned and familiar to many people. In cycling, each leg alternately applies forces to pedals attached to a short crank

arm that turns a flywheel attached by chain. The alternating and relatively short duty cycles of each leg allows nearly continuous force production. World class male track cyclists (4-km) optimize their pedaling rate near 130 rpms [38] and subsequently, relatively small forces applied rapidly result in substantial power output [39].

Conversely, rowing is a more complex movement that may be less familiar to many people, compared to cycling [33]. Rowing technique may be quickly learned to an adequate degree [33], but high levels of performance are the result of extensive practice and training [40]. Rowing involves coordination of the upper and lower body in which forces must be transferred through the legs, torso and arms to a handle and chain attached to a flywheel. The rowing stroke begins with the extension of both legs and the hip which contributes the majority of the power in the stroke followed by a relatively less powerful contribution from the arms and back [41]. Compared to cycling, the rowing duty cycle is a greater duration that involves a force generating "drive" phase, accounting for approximately 53% of the entire cycle, followed by a passive "recovery" phase [42]. Elite oarsmen choose a cadence near 35 strokes per minute and thus rowing invokes a greater relative force per stroke, but at a slower relative rate compared to cycling.

Between cycling and rowing, some physiological responses are shared, while some are distinct. In subjects previously naïve to the rowing technique (but given instruction and practice), peak heart rate and VO_{2max} were not different between the modalities but peak power was lower while rowing [43]. During incremental exercise, heart rate, ventilation and VO_2 are higher at each workload during rowing [33]. The differences in physiological response are explained in part, by the greater relative muscle mass recruited during rowing [43,44]. Also,

calculated efficiency (Δwork rate/ΔVO_2) is lower during rowing: 10-20% [33,45] compared to cycling: 22-26% [46]. Therefore, it is possible that proportional hypoxia-mediated performance decrements may be greater during rowing compared to cycling due to decreased efficiency. It may also be possible that inefficiency impairs rowing performance to such a degree that the added insult of hypoxia may be less apparent during rowing compared to cycling.

Different techniques and physiological responses also lead to different training programs and physical/physiological profiles between cyclists and rowers. Cyclists focus on lower body training occurring primarily on-bike and may include lower body resistance training [47], while upper body training is deemphasized. Conversely, rowers train both muscles of the upper and lower body in specific (rowing) and non-specific (running, cycling, resistance training) modalities [35]. Muscle mass that does not contribute to power production in cycling (i.e. upper body) would be extraneous, potentially negatively affecting efficiency [48] and performance [49]. In rowers, however, all major muscle groups contribute to the rowing effort and there is a positive correlation ($r=0.56$; $p<0.001$) [50] between overall body mass and performance. As such, on average, elite male cyclists are typically smaller in overall mass compared to age and sex matched elite rowers (approximately 60-75kg vs. 70-90kg, respectively)[51,52].

Any type of endurance-type training leads to central and peripheral adaptations that improve aerobic capacity as well as muscular strength and endurance, although adaptations may be regionalized with different training modalities. Central adaptations, such as improved stroke volume [53], blood volume and hemoglobin [54], and vascular function [55–57], enhance blood flow and DO_2 while increased motor output allows more muscle mass to be recruited during exercise [58,59].

Peripheral adaptations include increased muscle capillary density[60], improving O_2 diffusion into active muscle [61], and improved oxidative capacity of muscle via increased mitochondrial density[62] and oxidative enzymes [63] within trained limbs only [64] . Cross sectional area of muscle fibers and contractility also increases with training allowing for greater force production [65]. As a result of training, elite cyclists and rowers possess great aerobic capacity with typical VO_{2max} values of approximately 5.5 and 3.85 $L \cdot min^{-1}$ for male and female cyclists, respectively [51,66] and 6.3 and 4.5 $L \cdot min^{-1}$ for male and female rowers, respectively [35]. Maximal aerobic power output is also great with typical values of 470 and 333 W for male and female cyclists, respectively[66] and 422 and 305 W for male and female rowers, respectively [35].

Due to modality specific training, peripheral adaptations may occur regionally such that cyclists experience adaptations primarily in the lower body, while rowers make adaptations in the lower and upper body. Therefore, during rowing exercise in hypoxia, rowers may experience a greater hypoxia-mediated performance decrement due to greater active muscle mass or conversely, they may experience a less severe hypoxia-mediated performance decrement compared to cyclists because the trained muscles of their upper body may be better able to extract O_2 from the blood. Another possibility is that the additional muscle mass cyclists might engage during rowing is not appreciable enough for hypoxia-mediated performance decrements to become apparent.

Summary

This review has investigated factors that could potentially influence hypoxia-mediated performance differences between cycling and rowing exercise as well as between athletes that train in one modality or the other. Answers to these questions are informed by examining

literature that describes the effect of hypoxia on endurance exercise, as well as the differences between the two modalities and athletes of one or the other. Based on the literature, it seems that hypoxia-mediated performance decrements between cycling and rowing may be influenced by differences in relative active muscle mass requirements and mechanical efficiency. Further, the magnitude of hypoxia-mediated performance decrements may be different between cyclists and rowers during rowing due to modality-specific training adaptations.

In the following study, trained cyclists and rowers have performed both cycling and rowing exercise tests in normoxia and hypoxia to discern the influence of exercise modality and training modality on hypoxia-mediated performance decrements.

CHAPTER II

INTRODUCTION

Acute exposure to hypobaric or normobaric hypoxia is accompanied by impaired physiological function and reduced exercise capacity/performance. These impairments may have important implications for individuals who must make rapid transitions from low to high altitude, such as the military or professional athletes. As altitude increases or the pressure of inspired oxygen (PiO_2) falls, maximal aerobic capacity (VO_{2max}) is reduced by five to seven percent for every 1000 m elevation gain above sea-level [3]. Thereby, hypoxia-mediated performance decrements are most apparent in endurance-type events or any activity that is heavily reliant on aerobic metabolism. However, the magnitude of impairment is not universal and may vary between different athletes and exercise modalities.

Cycling and rowing are both aerobic events of which high-level competition takes place in venues world-wide, including many that are above sea-level. During competition (cycling 4-km or rowing 2-km races), aerobic metabolism contributes 70-80% of the total energy required [34–37] and thus performance decrements would be expected in hypoxia [3]. Interestingly though, evidence from the 1968 Summer Olympics (Mexico City, elevation: 2,240 m) suggests that rowers'/rowing performance may be more severely affected by hypoxia compared to other endurance-type athletes or events, including cyclists'/cycling [6].

Key differences exist between the two exercise modalities and the athletes of each that may explain variations in hypoxia-mediated performance decrements. Cycling and rowing both rely principally on the force produced in the muscles of the lower body, although rowing engages additional muscle mass of the trunk and upper extremities [32,33]. In subjects previously

naïve to the rowing technique, heart rate, ventilation and oxygen consumption (VO_2) are higher during rowing compared to cycling at a given submaximal workload [33], and this may be explained in part by the greater relative muscle mass recruited during rowing [67,68]. Also, calculated efficiency (Δworkrate/ΔVO_2) is lower during rowing: 10-20% [33,45] compared to cycling: 22-26% [46]. Greater O_2 demand due to increased active muscle mass and decreased efficiency may then lead to greater proportional performance decrements in hypoxia during rowing compared to cycling.

Further, modality-specific adaptations, as well as traits that predispose individuals to one modality or the other may lead to different physical profiles between cyclists and rowers that may help explain any differences in hypoxia-mediated performance decrements. For example, on average, elite male rowers are heavier than age-matched elite male cyclists (approximately 60-75kg vs. 70-90kg, respectively) [51,52]. Concurrently, with more potential muscle to recruit during exercise, typical absolute VO_{2max} values also tend to be greater rowers, compared to cyclist counterparts (6.3 vs. 5.5 $L \cdot min^{-1}$) [35,51,66]. As the magnitude of decrements in absolute and relative VO_{2max} in hypoxia has been observed to be positively related to VO_{2max} in normoxia[5,7], hypoxia-mediated performance decrements may be expected to be greater in rowers compared to cyclists.

The purpose of this study was to determine the influence of exercise modality and training specificity on the proportional magnitude of hypoxia-mediated performance decrements. Identifying factors that influence performance in hypoxia, such as exercise modality or training specificity, would be of physiological interest to researchers and may benefit professionals and those who must perform at high levels during sojourns at high

altitude. We hypothesize that 1) hypoxia-mediated performance decrements will be greater during rowing time trials compared to cycling time-trials and 2) hypoxia-mediated performance decrements will be greater in rowers compared to cyclists due to a greater amount of active muscle mass recruited in rowers/rowing.

METHODS

Subjects

From July 2016 through April 2017, 27 healthy adults were recruited for the current study. Inclusion criteria consisted of current participation in either cycling or rowing training with at least 11 months of experience; age within the range of 18-55; assessment of blood pressure, and 12-lead electrocardiogram during incremental exercise; and physician approval. Exclusion criteria included self-reported time-trial times (cycling 4km or rowing 2km) of greater than 7:30 (min:sec) for males and 8:30 (min:sec) for females; history of acute mountain sickness; and contraindications to performing vigorous exercise. Each subject gave written informed consent after a detailed explanation of the study procedures. This study was approved by the Institutional Review Board at Colorado State University.

Overview

After screening, subjects reported to the Human Performance Clinical Research Laboratory on 8 separate occasions; subjects performed a graded exercise test on both cycling and rowing ergometers, then 3 time trials (practice, normoxic, hypoxic) of both exercise modalities. The study design followed a random cross-over design: Subjects were randomly assigned to perform all tests of either modality first, then the other; subjects were also

randomly assigned to perform hypoxic or normoxic time trials first, then the other (Figure 2.1). Subjects were instructed to abstain from vigorous exercise at least 24 hours before each visit.

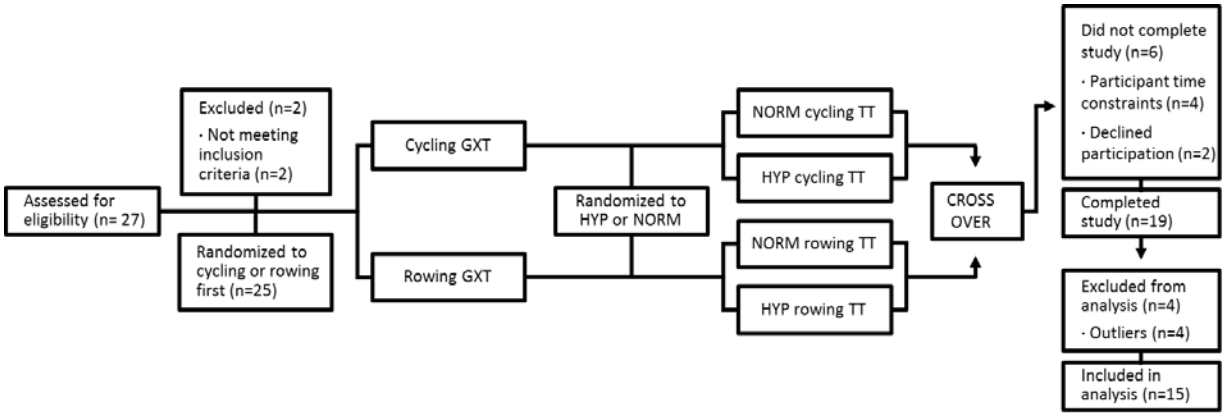

Figure 2.1. Participant flow diagram. Not all participants completed the study and not all data from completed participants were included in analysis: Data from 4 participants were removed; 3 as outliers in age; 1 as an outlier in hypoxia-mediated time trial performance.

Screening

During their first visit to the Human Performance Clinical Research Laboratory (HPCRL), subjects underwent a screening protocol to determine the safety and suitability of their participation: first, females provided urine samples for pregnancy testing; then blood was collected via finger prick and capillary tubes to measure hematocrit and hemoglobin values (Hb 201+; Hemocue, Ängelholm, Sweden); body composition was assessed via dual-energy x-ray absorptiometry (Lunar Radiation Corp, Madison WI; software v. 4.1); and lastly, subjects performed a graded exercise test (20-40 W/min continuous ramp until fatigue) on an electrically braked cycle ergometer (Velotron; Racermate Inc, Seattle, WA) while electrocardiogram tracings (Quinton Q-stress; Mortara Instrument, Millwaukee, WI) were

monitored and recorded, and expiratory gases were analyzed via an automated metabolic cart (TrueOne 2400, Parvo medics, Sandy, UT).

Graded Exercise Tests

After screening and randomization, subjects reported to the laboratory to perform a graded exercise test (GXT) on either a cycle ergometer (Velotron; Racermate Inc., Seattle, WA) or rowing ergometer (Model E; Concept2 Inc., Morrisville, VT). On arrival, participants were instrumented for measurement of heart rate (Polar F21, Kempele, Finland) and peripheral oxygen saturation (SpO_2, finger pulse oximeter, Cardiocap 5, GE Datex-Ohmeda, Madison, WI). Heart rate, blood pressure and SpO_2 were measured at rest in the supine, sitting and standing positions. Participants were then instructed in proper form if they were not familiar with the modality and given time to practice until their form was adequate. Participants were then allowed to warm up at a self-selected, but moderate pace, for up to five minutes. Before the start of the test, subjects were fitted with headgear and a mouthpiece attached to a 3-way, non-rebreathing valve (2730 Series; Hans Rudolph, Inc, Shawnee, KS), connected to an automated metabolic cart (TrueOne 2400, Parvo medics, Sandy, UT). The test consisted of 2-minute work stages followed by a 30-second rest period. The test began at 100 W for females or 150 W for males and increased by 50 W each stage. The test was terminated either at volitional fatigue or once the subject was unable to maintain a cadence above 40 revolutions per minute on the cycle ergometer or power output within 10% of the current stage on the row ergometer. Metabolic data was measured continuously as 30-second averages. Heart rate was measured within 15 seconds before the end of each stage and blood pressure, SpO_2 and rating of perceived exertion (RPE) were measured immediately after each stage.

Time Trials

After the GXT of one modality, subjects returned to the laboratory on separate occasions to perform three time trials (4km cycling ergometry or 2km rowing ergometry) of the same modality. First, a practice time trial was performed in normoxia followed by normoxic (FiO_2=0.21) and hypoxic (FiO_2=0.15) time trials in randomized order. Time trials were performed in an environmental chamber within which researchers could manipulate the inspired oxygen concentration (Colorado Altitude Training, Louisville, CO). The elevation of Colorado State University (Fort Collins campus) is approximately 1525 m (~5000 ft.) and is mildly hypobaric (~640 mmHg). As such, "normoxia" represents ambient conditions at 1525 m and hypoxia represents a simulation of approximately 4300 m (~14000 ft.).

On arrival, subjects were again instrumented for measurement heart rate (3-lead electrocardiogram) (Cardiocap 5, GE Datex-Ohmeda, Madison, WI) and SpO_2. Resting heart rate, blood pressure and SpO_2 were measured in the supine, sitting and standing positions. Subjects then began 15 minutes of standardized exercise at a constant workload of 100 W. Based on the training status of the research participants, this workload was supposed to represent exercise that could be completed at low- to moderate-intensity in both hypoxia and normoxia. Heart rate was measured within the last 15 seconds of exercise and blood pressure, SpO_2 and RPE were measured immediately upon completion. Subjects were then allowed up to 5 minutes to rest before beginning the time trial. During the time trial, participants were allowed to self-pace, but were encouraged to complete the time trial as quickly as possible. Again, heart rate was measured within the last 15 seconds of exercise and blood pressure, SpO_2 and RPE were measured immediately upon completion.

Statistical Analysis

Students T-test was used to compare subject characteristics between cyclists and rowers. Two-way repeated measures analysis of variance (ANOVA) was used to determine the influence of exercise modality and training specificity on primary outcome data collected during GXTs. Three-way ANOVA was used to determine the influence of and interaction between exercise modality, training specificity and FiO_2 condition on primary outcome data collected during steady state exercise and time trials. The level of statistical significance was set at $p<0.05$. Throughout this manuscript, data are expressed as mean and standard error.

RESULTS

Participants

Nineteen research participants completed all nine study visits and data from fifteen were analyzed. Data from four participants was excluded from analysis due to outliers in age (n=3) and performance during the rowing time trials (n=1). Selected physiological characteristics of the research participants are presented in Table 1.

Table 2.1. Select Participant Characteristics

	Cyclists	Rowers	p value
n	7(3 female)	8(3 female)	
Age (yrs)	26±2	21±1	0.002
Height (m)	1.74±0.02	1.79±0.03	0.271
Mass (kg)	68.5±2.4	83.8±7.3	0.082
Body Mass Index (kg/m^2)	22.6±0.6	26.2±2.0	0.126
Lean mass (kg)	51.1±2.5	60.1±5.2	0.142
Arm lean mass (kg)	5.8±0.7	7.6±0.9	0.128
Upper body lean mass (kg)	30.6±1.6	36.7±3.2	0.165
Lower body lean mass (kg)	17.3±0.9	20.0±1.9	0.249

Data are mean±SE.
n, Number of participants.

One participant was unable to complete the rowing time trial in hypoxia in the first attempt, but returned on a separate occasion and was successful. Aside from this incident, exercise in hypoxia was well tolerated. On average, cyclists were 5 years older than rowers, and rowers were slightly heavier than cyclists, but the difference in mass (~ 15 kg) did not attain statistical significance (p=0.082). Other characteristics were similar between the groups.

Resting data

Resting heart rate, mean arterial pressure (MAP) and SpO_2 measured in the supine position during normoxia and hypoxia are presented in Table 2. In general, compared to rowers, cyclists' resting heart rate and MAP were lower and SpO_2 was greater. In both cyclists and rowers, hypoxia increased resting heart rate and reduced SpO_2, while there was no influence on MAP. There were no interactions in these resting variables between the groups and FiO_2 conditions (p<0.05).

Graded Exercise Tests

Between cyclists and rowers, VO_{2max} was not different during cycling (3.86±0.25 vs. 3.79±0.38 L·min^{-1}, p=0.869) or rowing (3.44±0.22 vs. 3.9±0.34 L·min^{-1}, p=0.317) (Figure 2.1A). However, VO_{2max} within cyclists was reduced from cycling to rowing (3.86±0.25 vs. 3.44±0.25 L·min^{-1}, p=0.002), while within rowers, there was no difference between cycling and rowing (3.79±0.38 vs. 3.9±0.34 L·min^{-1}, p=0.238) (Figure 2.1A).

Power output at VO_{2max} was not different between the cyclists and rowers during cycling (379±24 vs. 331±27 W, p=0.211) or rowing (264±18 vs. 325±23 W, p=0.114) (Figure 2.1B).

Table 2.2. Average Hemodynamic Responses in Normoxia and Hypoxia During Rest, Graded Exercise Tests, Standardized Exercise (15 Minutes at 100 W) and Time Trials (4-km cycling and 2-km rowing)

	Normoxia				Hypoxia				*p* values			
	Cycling		Rowing		Cycling		Rowing		FiO_2			
	Cyclists	Rowers	Cyclists	Rowers	Cyclists	Rowers	Cyclists	Rowers				
HR_{rest}	63±4	69±1	63±5	70±4	67±3	76±3	74±3	73±4	0.012	0.036	0.566	
HR_{GXT}	184±3	185±2	178±5	187±4	-	-	-	-	0.217	0.406	0.206	
HR_{100W}	117±7	124±4	129±7	134±7	133±4	143±6	142±4	152±6	<0.001	0.055	0.014	
HR_{TT}	179±4	185±4	181±3	188±2	177±2	185±3	176±2	184±3	0.195	0.002	0.655	
MAP_{rest}	84±2	89±3	87±1	92±3	83±2	85±2	83±2	88±2	0.183	0.009	0.039	
MAP_{GXT}	121±4	115±3	106±3	115±3	-	-	-	-	-	0.536	0.068	
MAP_{100W}	96±3	99±2	94±3	98±3	97±3	102±2	96±3	98±2	0.349	0.058	0.409	
MAP_{TT}	110±4	104±3	99±4	107±3	105±4	103±3	98±4	107±3	0.484	0.327	0.285	[a,b]
$SpO_{2\ rest}$	99±0	98±1	98±1	98±0	91±0	89±1	91±1	89±1	0.001	0.01	0.597	
$SpO_{2\ GXT}$	90±3	94±1	95±1	92±1	-	-	-	-	0.14	0.758	0.542	
$SpO_{2\ 100\ W}$	97±0	97±1	97±0	96±1	80±1	80±2	80±2	77±2	<0.001	0.165	0.383	
$SpO_{2\ TT}$	93±1	91±2	93±1	88±2	71±1	75±1	74±2	68±1	<0.001	0.033	0.109	[b,c]
RPE_{GXT}	18±1	18±0	18±1	18±0	-	-	-	-	-	0.933	0.665	
RPE_{100W}	9±1	9±1	11±1	9±1	11±1	9±1	11±1	10±1	0.046	0.039	0.222	
RPE_{TT}	18±1	18±0	18±1	18±1	18±1	18±0	18±1	18±0	0.394	0.736	0.621	

Data are mean±SE

[a] interaction within cycling between cyclists and rowers

[b] interaction within rowing between cyclists and rowers

[c] interaction within rowers between cycling and rowing

Rest, average resting values; GXT, average values at VO_{2max} during graded exercise tests; 100W, average values at the end of 15 minutes of standardized exercise at 100 W; TT, average values at the end of time trials; HR, heart rate (beats per minute); MAP, mean arterial pressure; SpO_2, oxyhemoglobin saturation, RPE, rating of perceived exertion (Borg scale; 6-20).

Also, cyclists' power output at VO_{2max} was reduced from cycling to rowing (379±24 vs. 264±18 W, p<0.001), while within rowers, there was no difference between cycling and rowing (331±27 vs. 325±23 W, p=0.666) (Figure 2.2B). There were no differences in peak heart rate, MAP, SpO_2 or RPE between groups or modalities (Table 2) or any combination of the two factors (p>0.05).

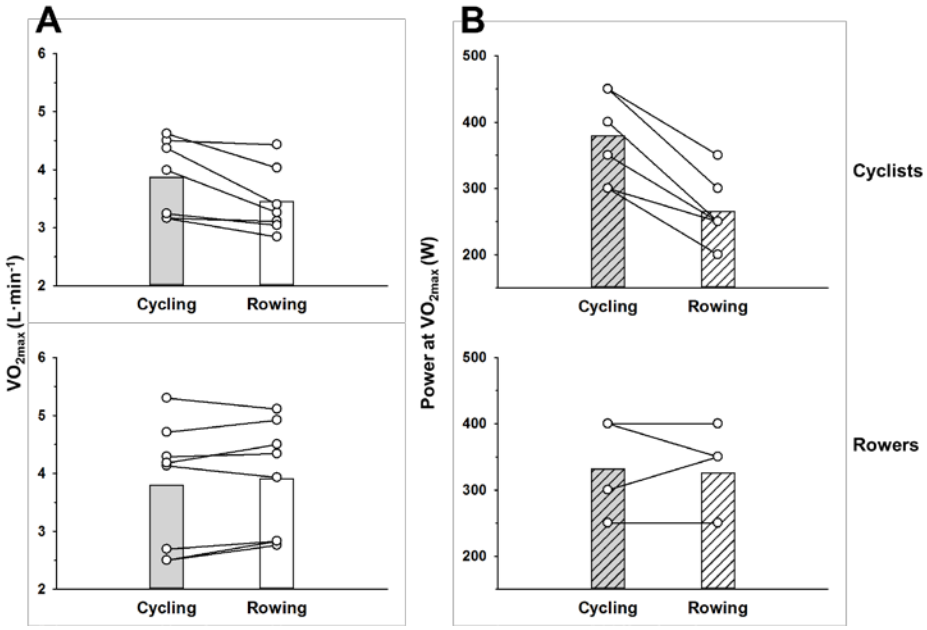

Figure 2.2. *A:* Maximal Oxygen uptake (VO_{2max}) during GXTs. *B:* Power achieved at VO_{2max}. Cyclists' data are on top; rowers' data are on bottom. Bars represent data mean values; circles represent individual data points and lines connect data points of the same individual between cycling and rowing.

VO_2 during submaximal GXT stages was lower during cycling compared to rowing (Figure 2.3, p<0.001) but was not different between cyclists and rowers. Further, efficiency (Δwork-rate/ΔVO_2), calculated within the first and penultimate stages of cycling and rowing GXTs, was not different between cyclists and rowers (30.9±1.2 vs. 31.2 ±1.0, p=0.614), nor cycling and rowing (31.6±1.3 vs. 31.0±1.4, p=0.766), and there were no interactions between the groups and modalities (p=0.252).

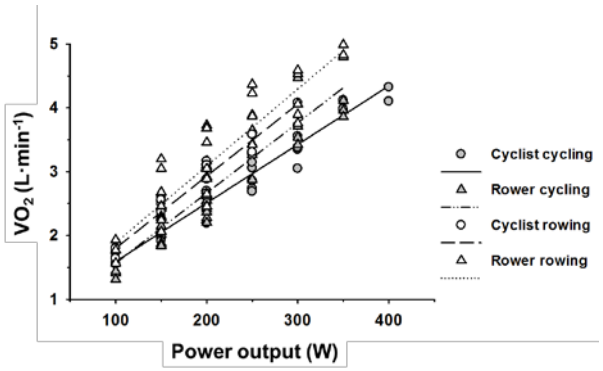

Figure 2.3. Oxygen uptake (VO_2) vs. Power output during cycling and rowing time trials between the first and penultimate stages. Individual data points are represented by circles (cyclists) or triangle (rowers). Lines of best fit are displayed.

Standardized exercise

The standardized bout of 100 W for 15 minutes represented low-moderate-intensity exercise (~ 22-40% of maximal work rate). Heart rate, MAP, SpO_2, and RPE data from the end of the standardized exercise are presented in Table 2. In general, at the end of standardized cycling and rowing exercise, heart rate was lower in cyclists compared to rowers, and lower after cycling compared to rowing. Hypoxia increased the heart rate response, but there was no interaction between the groups, modalities and FiO_2 conditions (p=0.928). MAP at the end of standardized exercise was not different between the groups, modalities or FiO_2 conditions, and there were no interactions between them (p=0.583). Hypoxia decreased SpO_2 at the end of both cycling and rowing standardized exercise, but there was no difference between the two groups or modalities, nor were there any interactions between the groups, modalities or FiO_2 conditions (p=0.399). Rating of perceived exertion after the standardized exercise was increased in hypoxia and was greater in cyclists compared to rowers, but similar between cycling and rowing. RPE was not influenced by any combination of group, modality or FiO_2 condition (p=0.280).

Time Trials

Normoxic time trials were not different from habituation trials during cycling or rowing time trials (p=0.728). Cycling and rowing time trial performance was slower in hypoxia for both cyclists and rowers (7.13±0.12 vs 7.63±0.12 minutes, p=0.005), although in both groups, cycling times were faster than rowing times (6.76±0.12 vs. 7.99±0.12minutes, p<0.001) (Figure 2.4A, 2.4B). In general, cycling time trials were similar between cyclists and rowers (6.65±0.18 vs 6.87±0.17 minutes, p=0.359), and cyclists completed rowing time trials slower than rowers (8.27±0.18 vs. 7.70±0.17 minutes, p=0.39) (Figure 2.4A, 2.4B). However, there were no interactions between the groups, modalities and FiO_2 conditions (p=0.798). Examination of the proportional magnitude of hypoxia-mediated performance decrements ((Δhypoxia time trial-normoxia time trial) · normoxia time trial^{-1}) revealed no difference between cyclists and rowers (7±0 vs 8±0%, p=0.059), nor between cycling and rowing (8±1 vs. 6±1%, p=0.135), and no interactions (p=0.138) (Figure 2.4C).

Average power output data during the time trials were also considered as an index of aerobic performance and thus were also analyzed. In alignment with time to completion data, hypoxia, as a main effect, reduced the average power output during both cycling and rowing (256±10 vs. 210±10 W, p=0.003), average power output was greater while cycling (253±10 vs. 212±10 W, p=0.007); and there was no overall difference between cyclists and rowers (227±11 vs. 238±10 W, p=0.441) (Figure 2.5A, 2.5B). However, cyclists and rowers maintained similar power output during cycling (262±15 vs. 244±14 W, p=0.579) (Figure 2.5A), while, cyclists' average power output was slightly lower than rowers' during rowing (191±15 vs. 233±14, p=0.053) (Figure 2.5B) although this difference did not attain statistical significance.

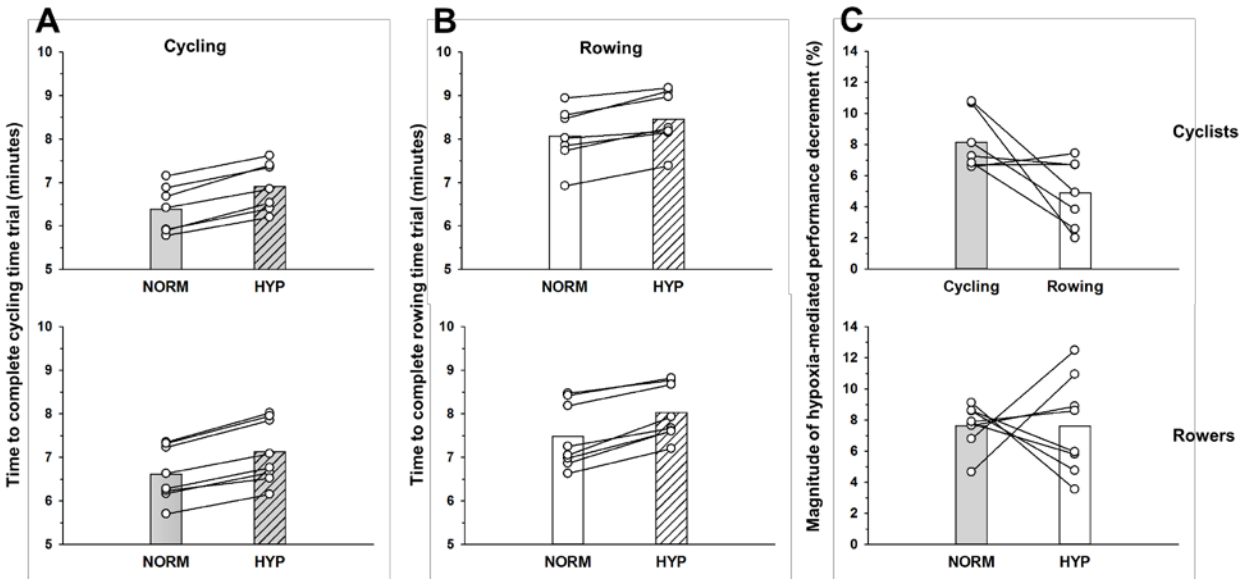

Figure 2.4. *A:* Time to complete the cycling time trials (4-km). *B:* Time to complete the rowing time trials (2-km). *C*: Magnitude of the hypoxia-mediated performance decrement during cycling and rowing time trials. Cyclists' data are on top; rowers' data are on bottom. Bars represent data mean values; circles represent individual data points and lines connect data points of the same individual in normoxia and hypoxia.

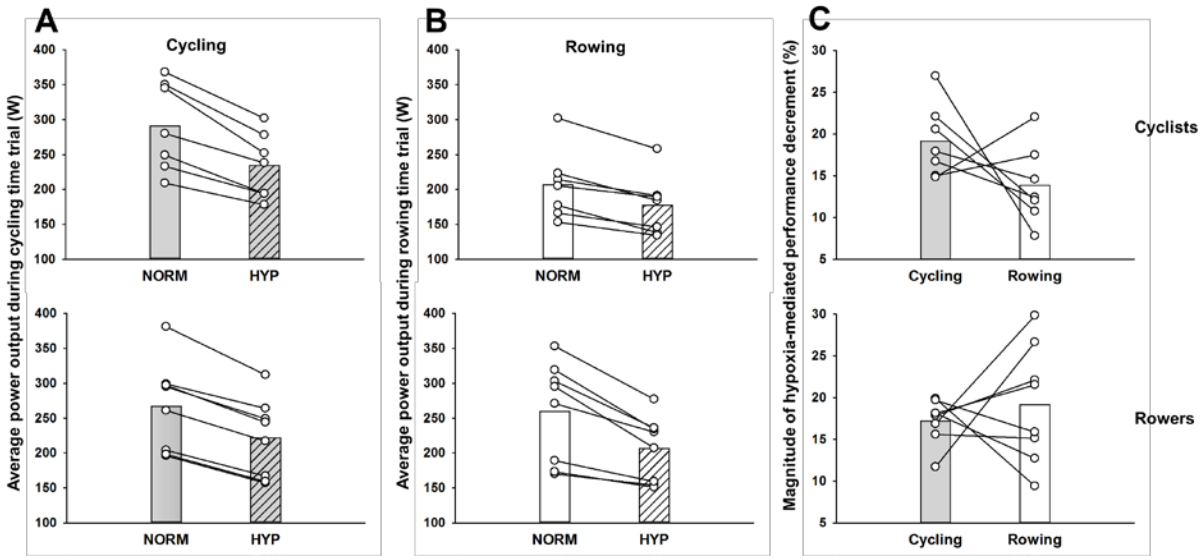

Figure 2.5. *A:* Average power output (W) during the cycling time trials (4-km). *B:* Average power during the rowing time trials (2-km). *C*: Magnitude of hypoxia-mediated performance decrement during cycling and rowing time trials. Cyclists' data are on top; rowers' data are on bottom. Bars represent data mean values; circles represent individual data points and lines connect data points of the same individual in normoxia and hypoxia.

Also, between the two modalities, cyclists' power output was much greater during cycling compared to rowing (191±15 vs. 233±14 W, p=0.002) (Figure 2.5A, 2.5B), while rowers' power output was similar between cycling and rowing (244±15 vs. 233±14 W, p=0.579). There was no interaction between groups, modalities and FiO_2 conditions (p=0.540). The proportional magnitude of hypoxia-mediated reduction in average power output during the time trials was not different between cyclists and rowers (17±1 vs 18±1%, p=0.189), nor between cycling and rowing (18±2 vs. 16±2%, p=0.473) (Figure 2.5C) and there was no interaction between groups, modalities and FiO_2 conditions (p=0.138).

Values of heart rate, MAP, SpO_2 and RPE at the end of the time trials are presented in Table 2. Heart rate at the end of the time trials was lower in cyclists compared to rowers, but similar between the two modalities and FiO_2 conditions, and there was no interaction between the groups, modalities and FiO_2 conditions (p=0.979). MAP at the end of the time trials was not different between groups, modalities, nor FiO_2 conditions. However, MAP in cyclists was greater after cycling but lower than rowers after rowing. No interaction existed between the groups, modalities and FiO_2 conditions (p=0.979). As a main effect, SpO_2 in both cyclists and rowers at the end of the time trials was reduced in hypoxia (Figure 2.6). Cyclists desaturated less than rowers, but there was no difference between the modalities (Figure 2.6). Specific to rowers, SpO_2 was lower after rowing compared to cycling, and during rowing, cyclists' SpO_2 was greater than that of rowers (Figure 2.6). No interaction existed though when groups and modalities were also compared with FiO_2 conditions (p=0.096). RPE after the time trials was not different between the groups, modalities or FiO_2 conditions, and there were no interactions (p=0.982).

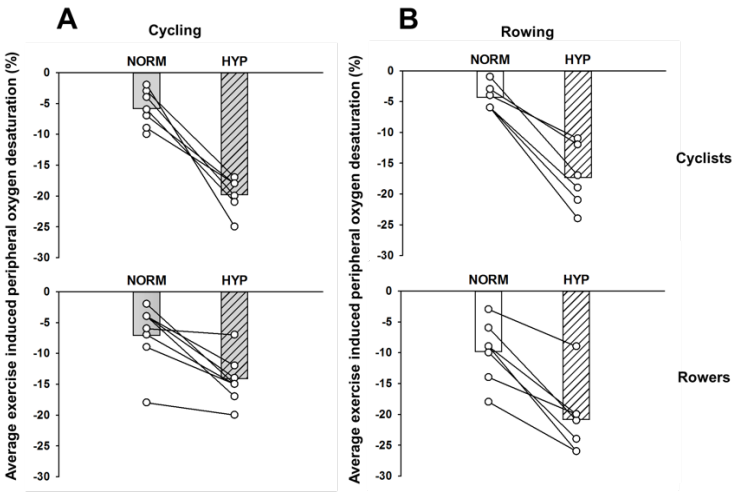

Figure 2.6. *A:* Average magnitude of exercise induced arterial oxygen desaturation (%)($SpO2_{rest}$-SpO_{2endTT}) during cycling time trials (4-km). *B:* Average magnitude of exercise induced arterial oxygen desaturation during the rowing time trials (2-km). Cyclists' data are on top; rowers' data are on bottom. Bars represent data mean values; circles represent individual data points and lines connect data points of the same individual in normoxia and hypoxia.

DISCUSSION

The purpose of the current study was to determine whether hypoxia-mediated performance decrements are influenced by exercise modality and training specificity. The major findings of our study were: 1) Hypoxia-mediated performance decrements during cycling (4-km) and rowing (2-km) time trials were not significantly different between the cyclists and rowers, or between any combination of group and modality. 2) SpO_2 at the end of cycling and rowing time trials in normoxia and hypoxia was lower in rowers compared to cyclists, and lower at the end of cycling compared to rowing. 3) Specifically, SpO_2 after rowing was lower in rowers compared to cyclists, and rowers' SpO_2 was lower after rowing compared to cycling. Although these differences in SpO_2 did not account for differences in the functional outcomes of time trial performance (time to completion or average power output), these data may help explain

how hypoxia-mediated performance decrements could be greater in rowers/rowing compared to cyclists/cycling in other situations (i.e. longer-duration time trials, or elite-level athletes).

Hypoxia-mediated performance decrements are determined by the body's ability to: 1) transfer O_2 from the lungs into circulation, 2) deliver O_2 to exercising muscle, and 3) utilize O_2. The differences between cycling and rowing, and between the physical characteristics of cyclists and rowers could influence these capabilities, so as to elicit greater performance decrements in hypoxia during rowing compared to cycling, or in rowers compared to cyclists.

During exercise in hypoxia, the magnitude of pulmonary O_2 diffusion impairment can be influenced by the amount of muscle mass recruited during exercise. Greater muscle mass recruitment requires greater $\dot{Q}$ causing a greater disturbance in blood pH and temperature leading to a rightward shift in the oxyhemoglobin dissociation curve leading to less SaO_2 for a given PO_2. Greater $\dot{Q}$ also reduces red blood cell transit time through pulmonary capillaries, potentially disallowing complete oxygen tension (PO_2) equilibration [18]. In combination, these mechanisms may explain the modulating effect of muscle mass on SaO_2.

It is likely that differences existed in the amount of active muscle mass used by cyclists and rowers during cycling and rowing that influenced pulmonary O_2 diffusion, contributing to the observed differences in SpO_2. Unfortunately, electromyogram data were not included in this study, but possible differences in active muscle mass may be alluded to by anthropometric, GXT and time trial data. In this study, there were no statistical differences between cyclists and rowers in body mass, but rowers tended to be heavier (p=0.082) and in other studies profiling elite cyclists and rowers, elite rowers are generally greater in mass [51,52]. By itself, mass may not be a useful parameter, but combined with greater VO_{2max} values in elite rowers compared to

elite cyclists [35,51,66], it may be evidence of greater potential muscle mass capable of performing work. In the current study, VO_{2max} was not different between cyclists and rowers within either modality, but it is important to acknowledge that these participants were *well-trained*, but not *elite* athletes. However, VO_{2max} and power output at VO_{2max} of cyclists was reduced from cycling to rowing, indicating an attenuation of active muscle mass from cycling to rowing. This is further supported by longer time to completion and lower average power output of cyclists during rowing time trials. Cycling exercise primarily engages muscles of the lower body with most of the forces produced in the quadriceps muscle group [32], and extensive training in this modality may lead to a relative structural underdevelopment of cyclists' arms compared to their well-developed legs; that is, the ligaments and tendons of the arms may not be able to tolerate the maximal force potential of the legs. It is then possible, that within cyclists, the additional muscle mass of the upper body recruited during rowing does not outweigh the unexploited muscle mass of the legs. In contrast, rowers, who train both upper and lower body [35], may have better coordination of simultaneous leg-extension [69], and better developed arm structure, thus allowing rowers to recruit a greater proportion of muscle mass during rowing. This may be supported by the rowers' faster time to completion during rowing time trials with a slightly greater average power output compared to cyclists (191±15 vs. 233±14, p=0.053) (Figure 2.5B). While it is clear that SpO_2 was lowest in rowers while rowing, definitive conclusions as to the cause of reduced SpO_2 cannot be drawn from these data. However, these data align with previous research implicating active muscle mass as a factor influencing SaO_2 [5,7,20] and could indicate a potential for greater hypoxia-mediated decrements in rowers'/rowing compared to cyclists'/cycling performance. Another explanation for decreased SpO_2 during

rowing could be relative hypoventilation due to the cramped position of rowing and impaired diaphragmatic excursion. However, ventilatory data were not collected during time trials, and GXT data as well as prior research suggests that ventilation is not impaired during rowing [70]. Future investigations including measurements of muscle activation, $\dot{Q}$, blood pH and temperature would help elucidate the influence of active muscle mass on pulmonary O_2 diffusion between these two types of athlete and exercise modality.

Beyond contributing to a pulmonary O_2 diffusion limitation, greater active muscle mass in rowers while rowing could negatively impact aerobic performance in hypoxia through limitations in BF distribution and DO_2. Greater muscle activation would require a greater $\dot{Q}$ to perfuse all the active muscle mass and is limited by maximal $\dot{Q}$ [22,26,28]. Research has also shown that maximal $\dot{Q}$ is reduced in acute hypoxia[30], perhaps as a mechanism to protect from myocardial hypoxia [23]. This may further reduce BF and DO_2, but if the exercise model does not elicit maximal $\dot{Q}$ in normoxia, such as during exercise with a small relative muscle mass, $\dot{Q}$, BF and DO_2 may be preserved in hypoxia [20]. If less muscle mass were recruited in cyclists/cycling compared to rowers/rowing, there may be a smaller perturbation to systemic BF and DO_2, thereby limiting the magnitude of hypoxia-mediated performance decrements in cyclists/cycling. Although there were no significant differences in the proportional hypoxia-mediated decrements in performance between the groups or modalities, performance (time to completion) was slightly more affected by hypoxia in rowers compared to cyclists (p=0.059), supporting the notion above. These data are interpreted cautiously though, due to the methodology used; participants were not blinded to their power output during the time trials, which would allow participants to pace themselves, adding a psychological aspect to their

performance, thereby negating a truly physiological response to hypoxia. However, these data may have validity still as athletes would have performance feedback during real-competition.

The third factor that could influence exercise performance in hypoxia is O_2 utilization. During cycling and rowing GXTs in this study, VO_2 at a given workload was consistently higher during rowing compared to cycling and efficiency between the first and penultimate stages was similar between the groups and modalities. Prior research has also shown the O_2 cost of submaximal exercise at a given workload to be greater during rowing compared to cycling [33,71,72]. Present efficiency data, though, contrasts with prior findings of lower efficiency values during rowing compared to cycling (10-20% vs. 22-26%) [33,45,46]. This difference is possibly due to our efficiency calculations beginning at either 100 or 150 W for females and males respectively, as we did not collect VO_2 from the start of exercise. If rowing is in fact less efficient than cycling, it may be due, in part, to a longer and more complex kinetic chain between the points of force application (the feet and the flywheel), thus requiring the recruitment of a greater amount of muscle mass for a given workload [72]. Further, the disparate patterns of force generation between cycling and rowing could influence the efficiency of either modality, although it is not clear how the quick and nearly continuous force production during cycling compares to the relatively longer duration of stroke and recovery during rowing. In any case, greater O_2 cost and decreased efficiency during rowing could conceivably lead to greater impairment of performance in O_2-scarce environments. Another consideration is that endurance training of any muscle improves O_2 utilization of that muscle through increased muscle capillary density [60], improving O_2 diffusion into active muscle [61], and improved oxidative capacity of muscle via increased mitochondrial density [62] and oxidative enzymes [63] within

trained limbs only [64]. In theory, peripheral adaptations in the upper bodies of rowers could endow them with greater tolerance to hypoxia during rowing compared to cyclists, who's capacity to utilize O_2 in the muscles of their upper body may not be as great. However, measurement of oxidative capacity of muscles was beyond the scope of this study and there were no differences in hypoxia-mediated performance decrements beside.

Although the results of the current study may provide insights to the modulation of performance in hypoxia, several limitations in the current study restrict full confidence in our results. Notably, the populations of each group were quite small and may not have represented explicitly distinct phenotypes of elite cyclists or rowers. Although, all participants were well-trained and physically fit, anthropometric and performance data is not comparable to elite-level athletes. Also, due to geographical location, in Fort Collins, CO., where high level rowing competition is not prolific, recruitment of well-trained rowers was limited to the CSU club rowing team, leading to a disparity in age, and possibly skill, between the two groups. Differences between the two groups would likely be clearer if they were of the highest caliber athlete in each modality and more closely age matched, with a greater number of participants. Further, the inherent differences between cycling and rowing technique may obscure the physiological responses to exercise in hypoxia. Although adequate cycling and rowing technique are easily learned [33], increased rowing experience would likely lead to greater proficiency and possibly different results than those of the current study. Additionally, the pattern of leg muscle activation is different between the two modalities. A cleaner model to investigate the modulating effect of muscle mass in hypoxia might use arm and leg cycle ergometry, in which arm cycling is added to leg cycling and there is no change in the technique

used to activate leg muscle mass. While this might be a more physiologically sound experiment, the comparison of cycling and rowing, two modalities of world-wide competition, has valid application. Also, allowing participants to view performance feedback during time trial performance severely limits the interpretation of the physiological response to exercise in hypoxia. However, as mentioned before, performance during real competition would involve the integration of feedback, thus representing an environment similar to competition. Further, since hypoxia had main effects on SpO_2 and time trial performance, we are confident in the validity of our hypoxic stimulus even though it was not a purely physiological response. Further, despite the mildly hypoxic ambient conditions of Fort Collins (elev. 1,525 m; 5,003 ft), the validity of our hypoxic stimulus is again supported by performance decrements in this study, as well as others conducted in similar conditions [73]. Lastly, pertaining to SpO_2 data, peripheral pulse oximetry has been shown to be valid and reliable, although with a ±1.5% standard error of estimation, it is not as accurate as direct arterio-venous blood sampling [18]. Some SpO_2 measurements from the end of exercise in this study were lower than SaO_2 in athletes at the end of maximal exercise in some other studies: 70% [7]; 77% [74]; and 86% [5], bringing the validity of our SpO_2 results into question. However, these studies all used cycling exercise, whereas the lowest SpO_2 values measured in this study were in rowers while rowing. In line with our hypothesis, it is possible that the increased muscle mass recruited by rowers during rowing influenced pulmonary O_2 diffusion, such that arterial blood is in fact less saturated at the end of intense exercise. However, it is also possible that SpO_2 measurements in rowers were influenced by having more callused hands than other athletes due to specificities within their training, although this notion is hypothetical.

In summary, rapid transition from normoxic to hypoxic environments is accompanied by substantial decrements in SpO_2 and exercise performance. In this study, hypoxia-mediated performance decrements during cycling and rowing were not different between cyclists and rowers, or any combination therein. However, SpO_2 at the end of the time trials was lower in rowers and specifically, lower after rowing compared to cycling and lower than in cyclists during rowing. It is possible that differences in SpO_2 during exercise are modulated by the amount of muscle mass utilized for a specific exercise modality and could lead to differences in hypoxia-mediated performance decrements. Although these notions were not substantiated in this study, differences in hypoxia-mediated performance decrements may be more pronounced in a greater hypoxic stimulus, longer-duration events, or highly specialized/elite athletes. Therefore, the strategy used to maximize performance in one instance may not be appropriate in another. These data may help inform those interested in exercise performance in hypoxic environments, but further investigation is necessary to discern the full modulating potential of muscle mass on SpO_2 and exercise performance in hypoxia.

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APENDIX

CONSENT FORM

Consent to Participate in a Research Study
Colorado State University

TITLE OF STUDY: Endurance Exercise in Low Oxygen: Influence of Muscle Mass and Modality-Specific Training Status

PRINCIPAL INVESTIGATOR:
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WHY AM I BEING INVITED TO TAKE PART IN THIS RESEARCH?
You are a healthy endurance trained adult man or woman aged between 18 and 50 years. You are a competitive cyclist or a competitive rower. You have been competing for at least one year.

WHO IS DOING THE STUDY?
Christopher Bell, Ph.D., an associate professor in the Department of Health and Exercise Science at Colorado State University will perform this research. Trained graduate and undergraduate students will assist Dr. Bell.

WHAT IS THE PURPOSE OF THIS STUDY?
In a low oxygen environment, such as high altitude, endurance exercise performance is impaired. The degree of impairment may be related to the amount of active muscle. That is, the more muscle needed to perform an exercise, the worse the effect of low oxygen on exercise performance. Cycling relies mostly on the muscles of the legs, but rowing involves legs, arms, and trunk muscles. We wish to find out if rowing performance will be impaired to a greater extent than cycling performance. In addition, as a result of endurance training, the ability to deliver blood and oxygen to active muscles during exercise is improved. We also wish to determine if frequent training on a specific exercise machine will influence the effect of low

oxygen. To do this we will study cyclists and rowers in normal oxygen, and in a low oxygen environment, while cycling and rowing.

WHERE IS THE STUDY GOING TO TAKE PLACE AND HOW LONG WILL IT LAST?
All of the procedures (unless otherwise stated) will take place in the Human Performance Clinical/Research laboratory (HPCRL) in the Department of Health & Exercise Science (Moby Complex). This whole research project will take place over a period of approximately 1 year. You will be asked to be involved for approximately 6-8 weeks. The total time of your participation will be approximately 10 hours spread over 9 visits.

WHAT WILL I BE ASKED TO DO?

Screening Visit ~ 2 Hours
The first visit to the HPCRL will be a screening visit. During this visit we will make sure that participation in this study is right for you.

Medical Questionnaire
You will be asked to answer several pages of questions related to your health, any illness you may have or have had, and medications you use or have used in the past.

Pregnancy Test
If you are female you will be required to have a sample of your urine tested for the presence of human chronic gonadotropin (HCG), a hormone that indicates whether you may be pregnant. This will require approximately 1 cup of your urine. If you are pregnant or the test indicates that you are pregnant you will not be able to participate in this study. (Duration: 5 minutes)

Body Composition
We will measure how much fat you have in your body using a test called dual energy x-ray absorptiometry (DEXA). The DEXA test requires you to lie quietly on a padded table while a small probe gives off low-level x-rays and sends them over your entire body. This test gives very accurate measurements of your body fat and bone mineral density. We will also measure the circumference of your waist and hip using a tape measure. (Duration: ~ 15 minutes)

Exercise Stress Test
You will be asked to perform a vigorous exercise test. This test will tell us if your heart is healthy. You will be asked to walk on a motorized treadmill or ride an exercise cycle (cycle ergometer) for approximately 10-15 minutes. The exercise will become more difficult every 2 minutes. While you are walking/riding we will measure your heart rate with an electrocardiogram (ECG) and your blood pressure with a cuff placed around your upper arm. We will also ask you to wear a nose clip (something that stops you breathing through your nose) and ask you to breathe through a mouthpiece. This will let us measure the gases you breathe. Depending on your age and health history, a physician may supervise the test. If we do not think your heart is healthy you will be referred to your primary care physician for

further testing. There is a chance that you may not be allowed to take part in our study. (Duration: ~ 60 minutes)

You will be asked to complete 4 cycling visits and 4 rowing visits. A coin toss will be used to decide if you will complete the cycling visits first, or the rowing visits first (e.g. heads=cycling, tails=rowing).

Cycling Visit 1 – less than 1 hour
You will be asked to perform a vigorous exercise test, also know as a VO2max test. You will be asked to ride an exercise cycle (cycle ergometer) for approximately 10-15 minutes. The exercise will be easy in the beginning but will become progressively harder until you are no longer able to exercise. While you are riding we will measure your heart rate. We will also ask you to wear a nose clip (something that stops you breathing through your nose) and ask you to breathe through a mouthpiece. This will let us measure the gases you breathe and let us calculate your VO2max.

Cycling Visit 2 – less than 1 hour
You will be asked to complete a practice test that will be the same as the tests you complete in visits 3 and 4. You will ride an exercise cycle for 15 minutes. The exercise will be easy. You will then be asked to ride the exercise cycle a distance equivalent to 4 km (a little less than 2.5 miles). The purpose of the test is for you to cycle this distance as fast as possible, as if it were a race.

During this test we will measure your heart rate, blood pressure, and the amount of oxygen in your blood. We also ask you how much effort you are putting in to the exercise.

Cycling Visits 3 and 4 – less than 1 hour each
These visits will take place in an environmental chamber, a special room in which the air you breathe can be changed. During one visit you will breathe normal air. Normal air contains 21% oxygen. During the other visit the amount of oxygen in the air will be decreased to approximately 15%. This might feel like being on top of a mountain that is 14,000 ft above sea level.

A coin toss will be used to decide if you will complete the normal air visit first, or the low oxygen visit first (e.g. heads=normal air, tails=low oxygen).

Each visit will begin with 15 minutes of sitting. The remainder of the visit will be exactly like cycling visit 2.

Rowing Visit 1 – less than 1 hour
You will be asked to perform a vigorous exercise test, also know as a VO2max test. You will be asked to exercise on a rowing machine for approximately 10-15 minutes. The exercise will be easy in the beginning but will become progressively harder until you are no longer able to

exercise. While you are rowing we will measure your heart rate. We will also ask you to wear a nose clip (something that stops you breathing through your nose) and ask you to breathe through a mouthpiece. This will let us measure the gases you breathe and let us calculate your VO2max.

Rowing Visit 2 – less than 1 hour
You will be asked to complete a practice test that will be the same as the tests you complete in visits 3 and 4. You will exercise on a rowing machine for 15 minutes. The exercise will be easy. You will then be asked to row a distance equivalent to 2 km (a little less than 1.25 miles). The purpose of the test is for you to row this distance as fast as possible, as if it were a race.

During this test we will measure your heart rate, blood pressure, and the amount of oxygen in your blood. We also ask you how much effort you are putting in to the exercise.

Rowing Visits 3 and 4 – less than 1 hour each
These visits will take place in an environmental chamber, a special room in which the air you breathe can be changed. During one visit you will breathe normal air. Normal air contains 21% oxygen. During the other visit the amount of oxygen in the air will be decreased to approximately 15%. This might feel like being on top of a mountain that is 14,000 ft above sea level.

A coin toss will be used to decide if you will complete the normal air visit first, or the low oxygen visit first (e.g. heads=normal air, tails=low oxygen).

Each visit will begin with 15 minutes of sitting. The remainder of the visit will be exactly like rowing visit 2.

Prizes
When you have finished the study we will add your exercise times from the cycle visits 3 and 4 to your exercise times from rowing visits 3 and 4. When the study is over for everyone, we will compare all of the exercise times. The man and woman with the fastest total times will each be given $50. The man and woman with the second fastest total times will each be given $30. The man and woman with the third fastest total times will each be given $20. The man or woman whose total time is in the middle (i.e. equal number of participants who finished faster and slower) will be given $10.

All of the times will be made available to all of the subjects, but the names of the study participants, including yours, will be in code. You will not know the identity of the other study participants, and they will not know your identity.

ARE THERE REASONS WHY I SHOULD NOT TAKE PART IN THIS STUDY?
You will not be allowed to participate in these studies for any of the following reasons:

1) You are pregnant.
2) You are younger than 18 or older than 50 years.
3) You suffer from a disease of the cardiovascular system.
4) You have asthma or any other type of lung/respiratory dysfunction.
5) Your resting oxygen saturation < 95% (we will measure this for you).
6) A contraindication was identified during the screening visit (e.g. positive stress test).
7) You have experienced and/or have been treated for seizures.
8) You use performance enhancing drugs.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS?
It is not possible to identify all potential risks in research procedures, but the researcher(s) have taken reasonable safeguards to minimize any known and potential, but unknown, risks. The Human Performance Clinical Research Laboratory has emergency supplies including a medicine trolley equipped with heart machines and supplemental oxygen. The investigator has a great deal of experience with all of the procedures. Some of the procedures for which you are being asked to volunteer have a number of associated risks:

Body Composition

The risks associated with the DEXA are very low. The maximum radiation dose you will receive in this study is less than 1/3000th of the federal and state occupational whole body dose limit allowed to radiation workers. Put another way, you will receive less than 1.3 mrem from this scan and you already receive approximately 450 mrem /year from normal background radiation dose in Colorado. The more radiation you receive over the course of your life, the more the risk increases of developing a fatal cancer or inducing changes in genes. The radiation dose you receive from this scan is not expected to significantly increase these risks, but the exact increase in such risks is not known. There are no discomforts associated with this procedure. Women who are or could be pregnant should receive no unnecessary radiation and should not participate in this study.

Breathing Air Containing Less Oxygen Than Normal Room Air

The risks associated with breathing a gas that contains less oxygen than normal room air include nervousness and shakiness, perspiration, dizziness or lightheadedness, nausea (feeling sick), sleepiness, confusion, difficulty speaking, and feeling anxious or weak. We will measure your heart rate, oxygen saturation (how oxygen you have in your blood), and blood pressure; if resting oxygen saturation falls below 70%, resting heart rate increases by more than 50 beats per minute, or resting blood pressure increases by more than 35 mmHg, the test will be terminated. Should you decide you no longer wish to continue breathing the low oxygen air, you will be able to leave the low-oxygen room and immediately begin breathing normal air. Supplemental air (100% oxygen) will be available on request or if needed. Symptoms associated with low oxygen can be treated very, very quickly by breathing normal room air and/or supplemental oxygen.

Exercise

During any exercise there is a risk that your heart will stop working. Exercise may make you feel tired and/or sick. Rowing exercise may hurt your hands (e.g. blisters). During cycle exercise the bike seat can be uncomfortable.

ARE THERE ANY BENEFITS FROM TAKING PART IN THIS STUDY?

There are no direct benefits in participating, however you will receive a copy of your results and information pertinent to your fitness and body composition (i.e. height and weight. You will be provided with a copy of your DEXA scan; you may wish to have this interpreted by a medically qualified professional.

DO I HAVE TO TAKE PART IN THE STUDY?

Your participation in this research is voluntary. If you decide to participate in the study, you may withdraw your consent and stop participating at any time without penalty or loss of benefits to which you are otherwise entitled.

WHAT WILL IT COST ME TO PARTICIPATE?

Other than transport to and from the lab, your participation should incur no costs.

WHO WILL SEE THE INFORMATION THAT I GIVE?

We will keep private all research records that identify you, to the extent allowed by law.

Your information will be combined with information from other people taking part in the study. When we write about the study to share with other researchers, we will write about the combined information we have gathered. You will not be identified in these written materials. We may publish the results of this study; however, we will keep your name and other identifying information private. We may be asked to share the research files with the CSU Institutional Review Board ethics committee for auditing purposes. If you receive a prize, your identity/record of receiving a prize (NOT your data) may be made available to CSU officials for financial audits.

CAN MY TAKING PART IN THE STUDY END EARLY?

Your participation in the study could end if you become pregnant, or if you miss any of the scheduled appointments.

WILL I RECEIVE ANY COMPENSATION FOR TAKING PART IN THIS STUDY?

You will not receive any compensation for taking part in this study.

Should your participation in the study end early, you will still receive feedback pertaining to your health and fitness.

WHAT HAPPENS IF I AM INJURED BECAUSE OF THE RESEARCH? We will arrange to get you medical care if you have an injury that is caused by this research. However, you or your insurance company will have to pay for that care. The Colorado Governmental Immunity Act determines and may limit Colorado
State University's legal responsibility if an injury happens because of this study. Claims against the University must be filed with Colorado State University within 180 days of the injury.

WHAT IF I HAVE QUESTIONS?
Before you decide whether to accept this invitation to take part in the study, please ask any questions that might come to mind now. Later, if you have questions about the study, you can contact the investigator, Christopher Bell at physiology@cahs.colostate.edu or 970-491-3495. If you have any questions about your rights as a volunteer in this research, contact the CSU IRB at: RICRO_IRB@mail.colostate.edu; 970-491-1553. We will give you a copy of this consent form to take with you.

WHAT ELSE DO I NEED TO KNOW?
Your signature acknowledges that you have read the information stated and willingly sign this consent form. Your signature also acknowledges that you have received, on the date signed, a copy of this document containing 8 pages.

_____ _____
Signature of person agreeing to take part in the study Date

_____ _____
Printed name of person agreeing to take part in the study Time of Day

_____ _____
Name of person providing information to participant Date

_____
Signature of Research Staff