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The Effect of Land-Based Repeated Sprint Training in Hypoxia compared to Normoxia on Anaerobic Capacity, Repeated Sprint Ability, and 100-Yard Swimming Performance: A Single-Blind Crossover Study

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Abstract

For over fifty years, athletes have attempted to induce additional improvements in middle and distance race performance by supplementing their sea-level conditioning programs with hypoxic training to improve aerobic capacity. The apparent success of this practice fostered an interest in applying hypoxic training to improve single-sprint, repeated sprint, and team sports performance. In response to this interest, a novel hypoxic training approach referred to as repeated sprinting in hypoxia (RSH), was proposed. RSH training is similar to training in normoxia (RSN). Both consist of performing multiple short sprints (≤ 30 seconds) on a work-to-rest ratio that does not permit complete recovery. The sole difference between them being the fraction of inspired oxygen (FiO_2) during training. RSN training takes place in normoxia (FiO_2 = 20.93 %) while during RSH training the FiO_2 is between 13.0% to 15.5%. To date, some evidence in the scientific literature suggests that RSH training induces small to moderate additional improvements in performance compared to the equivalent training in normoxia (RSN); however, there is contrasting evidence that postulates no additional performance improvements occur following RSH training. **PURPOSE:** The primary purpose of this dissertation was to determine if RSH training stimulated additional improvements in anaerobic capacity and swimming performance compared to the equivalent RSN training. **HYPOTHESIS**: We hypothesized that the increased hypoxic stress during RSH training, compared to the equivalent RSN training, would induce additional improvements in anaerobic capacity and swimming performance. **METHODS: A** randomized, single-blind, crossover design was used to train 12 Division II college swimmers from the same team. The twelve enrolled participants were 19.6 ± 1.4 years old, weighed 78.7 ± 10.6 kg, and measured 182.3 ± 6.9 cm in height. The study consisted of two 24-day training blocks separated by a 3-week washout period. All participants completed 7 RSH

workouts, or 7 RSN workouts, over each of the 24-day blocks, crossing over after the washout. Each workout included two sets of 8 x 20-second maximal effort sprints, on a 1-minute interval, with 2 minutes of passive rest between sets. Workouts consisted of two sets of repeated sprinting on the bicycle ergometer and two sets on the swimming ergometer with 5 minutes of passive rest between exercise modalities. During RSH training, the FiO_2 was 14.4 ± 0.2% maintained in an Altitude Chamber by replacement of O_2 with N_2 using Hypoxico Systems high flow hypoxic generators (Hypoxico Inc., NY, NY). RSN training was in ambient room air at 20.93% O_2 dry and atmospheric conditions of Syracuse, NY. The hypoxic stress during each workout was estimated from arterial oxygen saturation (%SpO_2) data collected using a forehead sensor. Training intensity was estimated from the average workload and peak heart rate achieved during each workout. Swimming performance was assessed with a 100-yard swimming time trial and an in-the-water repeated sprint test. Anaerobic capacity was estimated from laboratory-based tests that included a Wingate anaerobic test performed on a cycle ergometer (WAnT) and a novel modified Wingate test performed on a swimming ergometer (WAnT-Swim). Peak power (PP), mean power (MP), low power (LP) and the fatigue index (FI) were measured. In addition, peak heart rate (bpm) was measured during the WAnT and WAnT-Swim, and peak lactate ($mmol \cdot L^{-1}$) was assessed six minutes after the tests. **RESULTS:** Training intensity quantified as the mean power maintained during the workout, expressed as a percentage of the respective pre-training Wingate mean power score, did not differ significantly for simulated swim repeated sprinting during RSH (64.2 % ± 11.0%) vs. RSN (73.% ± 20.8%). Correspondingly, during repeated sprint cycling, the training intensity during RSH (19.5% ± 1.6%) was almost identical to RSN training (19.9% ± 1.3). In addition, peak heart rate during simulated swim RSH (138 ± 11bpm) vs. RSN (142 ± 14bpm) were not significantly different, nor were they between hypoxic conditions during

cycling, RSH (165 ± 11bpm) vs. RSN (170 ± 11). The hypoxic stress quantified as $\%SpO_2$ was the only apparent difference between hypoxic training conditions during simulated swim training, RSH (84.2% ± 2.2%) vs. RSN (92.5% ± 2.8%) and cycling, RSH (83.3% ± 2.2%) vs. RSN (95.6% ±0.8%). The main findings of this study were 1) RSH training significantly improved 100-yard time trial by 0.6 seconds ($p < 0.05$); however, following RSN training, there was a nonsignificant 0.31second increase (slower) in 100-yard performance. 2) Following RSH and RSN training, measures of repeated sprint swimming performance improved significantly ($p < 0.05$). 3) Following RSH and RSN training, anaerobic capacity based on WAnT and WAnT-Swim performance improved significantly ($p < 0.01$). 3) Compared to the equivalent RSN training, the increased hypoxic stress during RSH training did not induce additional improvements in swimming performance or anaerobic capacity. C**ONCLUSION:** Therefore, including a land-based, swim-specific repeated sprint training program in the yearly conditioning program of college swimmers may be beneficial. However, until further research establishes that RSH training stimulates additional improvements in swimming performance and anaerobic capacity, RSN training is the pragmatic and cost-effective option.
Keywords, hypoxic training, swimming ergometer, WAnT, WAnT-Swim, repeated sprints in hypoxia.

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Key Concepts

This section aims to provide background information for readers with limited exposure to the hypoxic training literature. Therefore, the key concepts discussed throughout the dissertation are defined below.

Altitude Chamber.

An altitude chamber is an enclosure that simulates conditions at a specific altitude. There are two types, Normobaric, which adjusts the concentration of oxygen and Hypobaric, which adjusts the pressure within the chamber. Chambers can be constructed as a free-standing structure or using an existing room.

Anaerobic Energy System.

The anaerobic energy system is the primary energy source for maximal exercise lasting up to 2.5 minutes. The system comprises two components, the phosphagen system and anaerobic glycolysis. In general, the phosphagen system is the primary energy source for the first 10 seconds of intense work. Then, as the duration of the exercise increases, the contribution from anaerobic glycolysis increases.

Elite competitive swimmer.

In this study, elite competitive swimmer refers to athletes that are current members of a college or university competitive swimming team.

Energy System Training.

Energy system training is a method for conditioning athletes based on analyzing the primary metabolic pathways involved in meeting the athlete's energy demands during

competition. For example, energy for a 10-second sprint is provided through the phosphagen system. Therefore, the training program is tailored to maximize the capacity of the phosphagen system.

Ergometers.

A machine capable of measuring work performed during exercise. Bicycling, rowing, skiing, arm crank, and swim ergometers are used.

Erythropoietin (EPO).

EPO is a hormone primarily produced in the kidney that prevents the destruction of red blood cells and stimulates their production. Of relevance for this study, EPO levels are increased in response to Live High training resulting in an increase in red blood cells and maximal oxygen consumption.

Erythropoiesis.

Erythropoiesis is the name for the process that creates new red blood cells.

Finger stick-Lactate Measurement.

A finger-stick is used to obtain a small quantity of blood and immediately analyzed using a portable lactate analyzer to determine the lactate concentration in the capillary blood. Higher lactate levels are associated with increased levels of anaerobic metabolism. Arterial blood in the capillaries is oxygenated and travels from the heart to the body. Therefore, samples taken immediately after exercise do not reflect lactate levels at the level of the muscle. For an accurate estimate of the lactate level in muscle, time is required for the lactate to diffuse from the muscle

into the blood. Studies have shown that maximal values are achieved 5 to 8 minutes after completing a maximal sprint exercise (Felippe LC, 2017).

Hypoxic Training.

Hypoxic training consists of two primary types, Live High (LH) and Live Low (LL), which consist of several approaches. Their common aim is to use the reduced availability of oxygen concurrent with exercise training to stimulate additional improvements in athletic performance.

Normoxia.

In this dissertation, normoxia refers to room air at 20.93% O_2 dry and atmospheric conditions of Syracuse, NY (522 feet above sea level).

Low Altitude.

In this dissertation, low altitude refers to an elevation that does not produce hypoxic stress sufficient to stimulate EPO production, accepted as an altitude of < 1250 meters. The term seal-level is often used as a synonym for low altitude.

Hypobaric Hypoxia vs. Normobaric Hypoxia

Altitude training in the laboratory depends on delivering air with a reduced PO_2 to training participants. The desired hypoxic stress can be achieved by reducing the fraction of inspired oxygen (FiO_2) or reducing the pressure (hypobaria) because the partial pressure of oxygen (PO_2) = the barometric pressure (P_B) x fraction of oxygen (FO_2). $PO_2 = P_B \times FO_2$. This study employed normobaric hypoxia (NH) using an altitude chamber and Hypoxico Systems generators.

Repeated Sprint Ability (RSA)

RSA is defined as maintaining a high percentage of maximal speed or power throughout a contest, game, meet, or test set, with incomplete recovery between sprints. Measures of RSA are often included as dependent variables in RSH training studies (Hancock, Sparks, & Kullman, 2015). Applying this definition necessitates understanding the sport-specific nature of RSA tests and the specificity of individual training protocols. For example, swimmers and soccer players sprint during competition. However, swimmers completely recover between races, while soccer players do not fully recover between sprints during a game. Therefore, they have different performance goals necessitating performance-specific test sets and training protocols.

Repeated Sprint Training (RST)

RST refers to repeated sprint training regardless of the hypoxic condition. Repeated sprint training is also referred to as repeated sprint exercise (RSE). RST has been defined throughout this dissertation as repeated sprints ≤ 30 seconds in duration, performed with incomplete recovery between sprints. Normally a work-to-rest ratio ≤ 1:4 is used (Brocherie, Girard, Faiss, & Millet, 2017; Faiss, Girard, & Millet, 2013). Another version of RSH training focused on repeated sprint performance during competition in Team Sports. This variation of RST defines RST as repeated bouts of maximal exercise for 10 seconds or less (Spencer, Bishop, Dawson, & Goodman, 2005).

Repeated Sprint Training.

Repeated sprint training in hypoxia (RSH): is a new approach to hypoxic training and the primary topic of this dissertation. RSH training was designed to improve sprint and repeated sprint performance in athletes that compete in short-duration events (less than 2 minutes) or team

sports that involve repeated sprinting (i.e., soccer). Workouts consist of 2-3 sets of high-intensity short sprints (less than 30 seconds) conducted 2-3 times weekly. **Repeated Sprint Training in Normoxia (RSN**): RSN refers to performing repeated sprint training while breathing air with a FiO_2 of 20.93%. **Repeated Sprints in Hypoxia versus Repeated Sprints in Normoxia:** This dissertation aims to assess the reality of RSH-induced additional improvements in performance beyond the equivalent RSN training. Therefore, the RSH protocols reviewed consisted of repeated sprints ≤ 30 seconds in duration, without complete recovery between repetitions. The sole distinction between RSH and RSN training was the lower oxygen concentration during RSH training.

Responder.

Responder is a term used to indicate an individual's likelihood to demonstrate a training adaptation after completing a specific training program. This concept implies that the response to exercise training is highly individualized. In other words, everyone responds uniquely to a given type of training. **Non-Responder.** This designation indicates that the individual does not respond to a specific type of exercise training. **Super-Responder. Super-Responder.** An individual whose response to a given training stimulus is amplified beyond that of a typical person. Elite athletes are often assumed to be super-responders.

Sea-Level.

Sea-level is the reference point for measuring elevation on earth. It refers to the ocean's surface, which is assumed to be approximately the same level worldwide. The term refers to an altitude that does not create hypoxic stress significant enough to stimulate EPO and erythropoiesis. This study was performed at sea-level.

Sprinting.

Sprinting is a high-intensity exercise performed at maximal speed or power output for short periods, generally less than 30 seconds. Energy for sprinting is supplied primarily through anaerobic pathways.

Swimming Ergometer.

A swimming ergometer is a machine that measures work while the person performs a swimming stroke-specific pulling pattern against resistance. For this dissertation a VASA swimming ergometer was used for swim-specific training and testing.

Team Sports.

Within the context of RSH training studies, Team Sports refer to sports that involve repeated sprinting with various periods of reduced speed or rest between sprints. Examples are soccer, basketball, or American football.

Training Adaptation.

Workouts and training programs are designed to evoke a specific response from the body. Superficially, physical enhancements such as increased muscle mass in response to strength training. These responses are called adaptations and are associated with metabolic and physiological changes.

Wingate Anaerobic Test (WAnT).

The WAnT, used in this dissertation, is a 30-second all-out sprint test typically performed on a cycle ergometer that quantifies anaerobic capacity using three power output scores (defined below).

Peak Power (PP).

PP is the highest power output recorded during the WAnT. Procedures vary, but the PP score is typically the average of the five highest outputs achieved during the test.

Mean Power (MP).

MP is the average power output over the 30-second test Wingate Test.

Low Power (LP).

LP is the lowest power output recorded during the test. Procedures vary between studies, but typically, the five lowest power outputs are averaged.

Fatigue Index (FI).

The FI calculated using the following formula [FI= (PP-LP)PP*100]. The Fi attempts to measure the rate of fatigue over the 30-second Wingate test.

Wingate Anaerobic Arm Test (WAAT).

The WAAT is an arm-based modification of the WAnT used to measure an individual's upper body anaerobic capacity. The WAAT is often administered using an arm-crank ergometer.

Wingate Anaerobic Swim Test (WAnT-Swim).

The WAnT-Swim is a novel modification of the WAnT used to measure the anaerobic capacity of a swimmer. The test utilizes a swimming ergometer, allowing the participant to simulate the swimming pull on land.

Non-Technical Summary

What is Known

For over fifty years, athletes have supplemented their yearly conditioning program with one of the Live High (LH) hypoxic training approaches, expecting additional improvements in sea-level performance compared to the equivalent training in normoxia. LH training involves living and training at altitude or by simulating altitude. The emphasis has been on performance in middle and distance events. Therefore, endurance athletes have been the primary beneficiaries of this training, evidenced by improved sea-level performance in runners and cyclists (D. L. Bonetti, Hopkins, Lowe, Boussana, & Kilding, 2009; Stray-Gundersen, Chapman, & Levine, 2001) although, in a few studies, contradictory results have been demonstrated (Hahn & Gore, 2001; D. Montero & Lundby, 2017). In addition, several LH training studies that investigated additional improvements in swimming performance following LH training were inconclusive (Robertson, Aughey, Anson, Hopkins, & Pyne, 2010; Siewierski, Slominski, Bialecki, & Adamczyk, 2012).

The popularly assumed benefit of LH training on athletic performance has fueled experimentation with new hypoxia training approaches (B Friedmann et al., 2005). The most recent approach utilizes repetitions of short sprint sets in hypoxia (RSH) to evoke additional improvements in a single sprint, repeated sprint and team sports performance beyond the same training in normoxia (RSN) (Czuba et al., 2013; Faiss, Girard, et al., 2013; Sharp, Troup, & Costill, 1982). Typically, RSH training utilizes sprints of 6 to 30 seconds in length, 2 to 3 times per week, with a work-to-rest ratio of less than 1:4 (Brocherie et al., 2017; Faiss, Girard, et al.,

2013). In the scientific literature, the duration of the RSH training programs ranged from 1-6 weeks (Brocherie et al., 2017; Puype, Van Proeyen, Raymackers, Deldicque, & Hespel, 2013).

RSH training targets improvements in athletic performance through adaptations in the pathways (anaerobic) that supply the energy needed to sprint. During shorter sprints (< 10 seconds), anaerobic contributions may be as high as 94% (PCr degradation 44%, anaerobic glycolysis 50%) (Gaitanos, Williams, Boobis, & Brooks, 1993). As the sprint duration or the number of sprints increases, the reliance on the aerobic system progressively increases (Faiss, Leger, et al., 2013). Although studied in several sports, the existence of RSH-induced additional improvements in anaerobic capacity compared to the corresponding RSN training remains unclear. Therefore, to determine the reality of RSH-induced additional improvements, future research with athletes from various sports and abilities (athletic status) is needed. Furthermore, if RSH proves to evoke additional improvements, the optimal training protocol and hypoxic dose must be elucidated.

Due to the novel nature of RSH training, a limited number of participants and sports have been studied, and its effectiveness remains to be established. Furthermore, to date, only one study has investigated swimming performance. Therefore, this study aimed to investigate the impact of RSH training compared to RSN training on swimming and laboratory-based performance measures in a cohort of Division II college swimmers. We hypothesized that improvement in performance following RSH training would be significantly greater than after RSN training.

The specific aims of this study were 1) to investigate if land-based, swim-specific RSH training stimulated additional improvement in 100-yard swimming performance compared to the equivalent RSN training. 2) to investigate if land-based, swim-specific RSH training stimulated

additional improvement in repeated sprint swimming performance compared to the equivalent RSN training. Finally, 3) to assess the impact of RSH vs. RSN training on anaerobic capacity measured in the laboratory using the Wingate Anaerobic Capacity Test (WAnT) conducted on a bicycle ergometer and the WAnT-Swim, a swimming modification of the WAnT performed on a swimming ergometer.

What is New and Noteworthy From Our Results

The first step in this dissertation assessed the reliability of the WAnT-Swim and determined that the test was reliable when administered using the VASA swimming ergometer. This land-based simulated swimming test eliminated multiple challenges associated with collecting data in an aquatic environment. In addition, based on our preliminary data, the WAnT-Swim appears to be a valid measure of anaerobic capacity; however, additional research is needed to confirm our findings.

Next**,** the hypothesis for Aim 1 that RSH training would improve 100-yard swimming performance beyond the same training in normoxia was tested. One-hundred-yard swimming performance improved significantly following RSH training. However, performance in the time trial following RSN training was slower. Although swimmers were faster following RSH vs. RSN training, the difference in improvement did not reach the significance level. In other words, there was no additional RSH-induced improvement in 100-yard swimming performance.

In Aim 2, we investigated the hypothesis that RSH training would improve repeated sprint swimming performance beyond the corresponding RSN training. Unlike the 100-yard time trial, repeated sprint performance improved following RSH and RSN, with no significant difference in improvement between RSH and RSN training.

Finally, in Aim 3, we assessed the potential additional benefits of RSH vs. RSN training on anaerobic capacity estimated from performance on the WAnT and WAnT-Swim. In addition, as a second measure of anaerobic capacity, peak lactate levels were measured after each Wingate test. Our results indicated no significant difference in improvement in WAnT or WAnT-Swim scores between RSH and RSN training. Moreover, peak lactate levels following training were essentially unchanged from pre-training levels. However, the power outputs at those lactate levels were significantly higher, with no significant difference between RSH and RSN training suggesting improvements in anaerobic capacity or management of lactate. Thus, swimming performance and anaerobic capacity improved significantly following RSH and RSN training, but our hypothesis that additional RSH-induced improvements would occur was not supported.

Implications

This study demonstrated that the WAnt-Swim was a reliable test when conducted using the VASA swimming ergometer. In addition, preliminary data supported the validity of the WAnT-Swim as a measure of anaerobic capacity. Therefore, additional research is needed to confirm the validity of the WAnT-Swim. Pragmatically, the WAnT-Swim and the swimming ergometer provide sports scientists and swimming coaches with a reliable land-based method to assess and train swim-specific components of swimming performance.

The main implication from the current study is that RSH training does not induce additional improvements beyond the corresponding RSN training in 100-yard swimming, repeated sprint swimming, and anaerobic capacity. Therefore, supplementing college swimmers' yearly conditioning program with RSH training is not warranted at this time.

Chapter I: Introduction and Specific Aims

Coaches and athletes have included hypoxic training in their yearly conditioning programs for over fifty years. Their goal was to use the increased hypoxic stress to stimulate additional improvements in sea-level athletic performance (Flaherty, O'Connor, & Johnston, 2016; Lundby, Millet, Calbet, Bärtsch, & Subudhi, 2012). The first hypoxic training approach consisted of ascending to altitude to live and train (LHTH) (Bärtsch & Saltin, 2008; Saunders, Pyne, & Gore, 2009). Subsequently, new live high (LH) approaches were developed. Today they include live high-train high (LHTH), live high-train low (LHTL), live high train low and high (LHTLH), and intermittent hypoxia training (IHT) (B Friedmann et al., 2005; Gough et al., 2012). Experimental evidence suggested that the mechanism underlying performance improvements in the middle distance and distance events following LH training was a hypoxia-induced enhancement in the oxygen-carrying capacity of the blood and a concomitant improvement in maximal oxygen capacity (VO_{2max}) (D. L. Bonetti et al., 2009; Brocherie et al., 2017; Stray-Gundersen et al., 2001). However, despite hundreds of studies investigating the live high training approaches, a definitive conclusion concerning hypoxia-induced additional improvements in performance beyond the equivalent training in normoxia remains elusive.

Notwithstanding the lack of a scientific consensus, athletes continue to ascend to altitude camps or use simulated hypoxic training to prepare for competition. Moreover, a recently developed Live Low (LL) hypoxic training approach, repeated sprint training in hypoxia (RSH), was proposed as a method to improve single sprint, repeated sprint, and team sport performance (Brocherie et al., 2017; Faiss, Leger, et al., 2013). The LL approaches utilize simulated altitude at sea-level and target adaptations that enhance anaerobic capacity (Faiss, Leger, et al., 2013). However, is the continued use of hypoxic training and the development of new approaches

simply the result of athletes following a fad, or do real hypoxic training-induced additional benefits on athletic performance occur? This study addressed that question relative to RSH training.

The novel LL hypoxic approach, RSH training**,** utilizes repetitions of short sprint sets of 6-30 seconds in length, performed two to three times per week, with a work-to-rest ratio of less than 1:4 (Brocherie et al., 2017; Faiss, Girard, et al., 2013). In the scientific literature, the overall duration of RSH training ranged from one to six weeks (Faiss, Girard, et al.; Puype et al., 2013). Currently, a small to moderate RSH-induced additional improvement in single sprint and repeated sprint performance compared to the same training in normoxia is supported in the literature (Brocherie et al., 2017). However, this conclusion was based on nine studies encompassing approximately three hundred participants. Furthermore, training protocols, athletic status (elite to novice), experimental design, and hypoxic dose varied between studies. In addition, several authors reported no additional benefit of RSH over RSN training (D. Montero & Lundby, 2017). Therefore, the additional benefits of RSH compared to RSN training on performance in athletes remains an open question.

Repeated sprint training in normoxia aims to improve single-sprint and repeated-sprint performance. What is unknown is whether limiting oxygen availability during repeated sprint training evokes additional improvements in performance for a specific sport or event. Moreover, the evidence suggests that fitness level may impact the efficacy of RSH training (B Friedmann et al., 2005; Grégoire P Millet, Roels, Schmitt, Woorons, & Richalet, 2010). To broaden the number of sports and athletic status of the participants investigated, we assessed the impact of RSH training on performance in college swimmers. To quantify their athletic status, we ranked the participants within Division II college swimming.

Swimming is contested across all three Divisions (I, II, III) of the National Collegiate Athletic Association (NCAA, 2015). In collegiate swimming, event distances vary from 50 to 1,650 yards (45.7 – 1508.8 m) and can last approximately 18 seconds to 18 minutes, with the majority of events completed in less than 2.5 minutes (NCAA, 2015). Due to the distance and duration of the events, swimmers typically rely on anaerobic metabolic pathways to generate and sustain most of the propulsive force needed to race (Hawley, Williams, Vickovic, & Handcock, 1992; Holmer, 1983; Mercier, Granier, Mercier, Trouquet, & Préfaut, 1993; Morouco, Marinho, Izquierdo, Neiva, & Marques, 2015). Therefore, given the anaerobic nature of most swimming events, swimmers represent an ideal pool of participants for a RSH training study.

Anaerobic capacity is typically measured using the Wingate Anaerobic Capacity Test (WAnT) (Bar-Or, 1987). The WAnT is conducted on a bicycle ergometer, and four power output scores are measured, peak Power (PP), mean power (MP), low power (LP), and fatigue index (FI). To estimate upper body anaerobic capacity, the WAnT is performed using an arm crank (Green, 1995). Theoretically, an upper body modification of the WAnT can be performed on any reliable ergometer (Green, 1995; Klasnja et al., 2010). Therefore, we developed a novel modification of the WAnT, the Wingate Anaerobic Capacity Swim Test (WAnT-Swim), to determine upper-body anaerobic capacity in swimmers. Using a swimming ergometer, the WAnT-Swim permits an athlete to simulate a swimming pull in a dry environment. After conducting a reliability study, we found that the WAnT-Swim conducted with a VASA swimming ergometer was a reliable test (Chapter III). Furthermore, our preliminary data suggest that the WAnT-Swim is a valid measure of upper body anaerobic capacity.

In the literature, RSH raining protocols varied between studies; however, a consensus emerged that during RSH training, using a fraction of oxygen in inspired air (FiO_2) between

13.0% and 15.5% was effective (Brocherie et al., 2017). Therefore, to maximize the usefulness of the results from this study, we adhered to the established range using an FiO_2 of 14.5%. In addition, our repeated sprint training protocol incorporated similar repetitions and sets as those used in other studies. However, this study uniquely included upper body, swim-specific testing, and training, which may provide new information and insights into the adaptations underlying any additional performance gains following RSH compared to RSN training.

According to Montero and Lundby, to conclude that RSH training induces additional improvements in performance beyond the equivalent RSN training requires additional controlled studies (David Montero & Lundby, 2016). To address this issue, we employed a single-blind, repeated measures crossover design in this study. In a crossover design, each participant completes RSH and RSN training with a washout period between each training bock. In this way, participants serve as their own controls minimizing the effect of inter-individual variability on the statistical analysis (Lim & In, 2021). In addition, the crossover design has higher statistical power with fewer participants than in a parallel study (Chow & Liu, 1999; Senn, 2002). Therefore, findings from our study will add to the existing pool of reliable data used to assess the impact of RSH training on performance and assist in determining if RSH training evokes additional improvements in performance compared to the equivalent RSN training.

Therefore, the primary purpose of this study was to investigate the plausibility of RSH training to induce additional improvements in swimming and laboratory-based performance measures compared to the equivalent RSN training in a cohort of Division II college swimmers. Using a single-blind crossover design, the main study of this Dissertation tested the hypothesis that a land-based, swim-specific RSH training program vs. the equivalent RSN training would

induce additional improvements in a 100-yard time trial, a repeated sprint swimming test, and in laboratory-based measures of anaerobic capacity.

The Dissertation is presented in five chapters, plus an appendix and structured as follows: Chapter 1 introduces the topic. A review of the literature relative to the LH training approaches and the LL training approaches with specific emphasis on repeated sprint training in hypoxia (RSH) is presented in Chapter II. The next two Chapters (III-IV) are presented as papers intended for submission to a journal for publication. Chapter III presents the study that assessed the reliability and preliminary validity of the WAnT-Swim. The focal study of this Dissertation, the investigation into the impact of RSH versus RSN training on swimming and laboratory-based performance outcomes, is presented in Chapter IV. The final chapter (V) summarizes the research findings, discusses the implications of our findings, and forecasts future directions in hypoxic training research, followed by an Appendix.

Specific Aims

The Aims of this Dissertation were:

Aim 1: To determine, in a cohort of Division II college swimmers, if RSH training stimulated additional improvement in 100-yard swimming performance beyond the improvement following RSN training.

H_A: Performance in a 100-yard swimming time trial will demonstrate significantly greater improvement following RSH training compared to RSN training. Rationale: The 100-yard distance takes between 40 and 70 seconds to complete and is assumed to rely primarily on anaerobic energy sources. This distance links to the underlying assumption that RSH training will enhance anaerobic capacity and sprint performance.

Aim 2: To determine, in a cohort of Division II college swimmers, if RSH training stimulated additional improvements in repeated sprint swimming performance beyond the improvements following RSN training.

H_A: In college swimmers, RSH training will improve performance operationalized as times on a repeated sprint swimming test to a greater degree than RSN training. Rationale: Repeated sprint performance will provide a second metric to examine the potential additional benefits of RSH versus RSN training on a component of swimming performance.

Aim 3: To determine, in a cohort of Division II college swimmers, if RSH training stimulated additional improvements beyond the equivalent RSN training in anaerobic capacity measured using the traditional WAnT and the WAnT-Swim.

H_A: The increased hypoxic stress during RSH training will induce additional improvement in anaerobic capacity compared to the corresponding RSN training. Rationale: anaerobic capacity is a determinant of performance for most swimming races. Anaerobic capacity is estimated from WAnT and WAnt-Swim power outputs. Therefore, assessing if the increased hypoxic stress during RSH training evoked additional improvements in anaerobic capacity compared to the same RSN training is warranted.

The results of this study will provide new data, which, when coupled with the existing literature, may help answer questions concerning the additional benefits of RSH training compared to RSN training on athletic performance. Furthermore, our results may provide useful information concerning the desirability of incorporating RSH training in the yearly program of college swimmers.

Chapter II: Review of the Literature

Introduction

The 1968 Olympic Games were held at an altitude of approximately 2,240 m in Mexico City, Mexico. Before the games, scientists, coaches, and athletes were concerned that the reduced availability of oxygen at higher altitudes would impair athletic performance, especially in distance running and swimming events (Jack Daniels, 1979; Flaherty et al., 2016; Pugh, 1967; Shephard, 1973; Wrynn, 2006). Prior to the games, establishing the optimal time needed to adapt to the low-oxygen environment was a priority (Balke, 1964; Jack Daniels, 1979; Fulco, Rock, & Cymerman, 2000; Suchy, 2017). However, during the Games, the athletes who lived and trained at altitude outperformed native lowland competitors, especially in distance events (Flaherty et al., 2016; Lundby et al., 2012). Due to the appearance of an altitude-enhanced benefit on performance, coaches and athletes quickly incorporated altitude training into their yearly conditioning programs (Flaherty et al., 2016; Lundby et al., 2012). For scientists, the results generated interest in assessing the reality of hypoxia-linked additional improvements in performance beyond the equivalent training in normoxia (Jack Daniels, 1979). However, over fifty years and hundreds of studies later, whether altitude/hypoxic training stimulates additional benefits in athletic performance remains controversial (Lundby et al., 2012; R. L. Wilber, 2001). Therefore, this review critically examined the scientific literature in reference to the hypothesis that hypoxic training stimulates additional improvements in athletic performance compared to the same training in normoxia.

For the remainder of this review, altitude/hypoxic training was referred to as hypoxic training. In the literature, hypoxic training was described based on the length of exposure to hypoxia (Grégoire P Millet et al., 2010). Ambiguously, researchers used the terms; approaches,

methods, models, strategies, versions, and types to describe hypoxic training. Based on the length of exposure to hypoxia, hypoxic training was divided into two types. The two types are Live High (LH) and Live Low (LL) hypoxic training. Variations within each type were called an approach, for example, the live high train high hypoxic training approach (LHTH). This review is organized into five sections. Section I defines the LH and LL hypoxic training approaches. Sections II and III review the literature relative to the impact of the LH training approaches (II) and LL training approaches (III) on anaerobic capacity, aerobic capacity, and athletic performance. Section IV briefly examines the potential mechanism underlying adaptations to hypoxic training, followed by a summary and conclusion in Section V.

Section I

Overview of Hypoxic Training

Based on the duration of exposure to hypoxia, hypoxic training is divided into two types; live high (LH) and live low (LL). Live high training incorporates long exposure to hypoxia (weeks) and includes three approaches: live high train high (LHTH), live high train low (LHTL), and live high train low and high (LHTLH). The Live Low approaches include live low and train high (LLTH), repeated sprint training in hypoxia (RSH), and a modification of RSH, voluntary hypoventilation at low lung volume (RSH-VHL) (Faiss, Girard, et al., 2013; Grégoire P Millet, Girard, Beard, & Brocherie, 2019; Grégoire P Millet et al., 2010; Randall L. Wilber, 2007). A third approach, intermittent hypoxia training (IHT), incorporates components of LH and LL training. Table 2.1 summarizes each hypoxic training approach, the energy system and performance targeted, the typical altitude or fraction of inspired oxygen (FiO_2) employed during training, and the duration of training.

Table 2.1 Hypoxic Training Approaches

Hypoxic Approach	**Description**	**Energy System**	**Performance**	**Altitude/FiO_2**	**Hypoxic Exposure**
LHTH	Train and live at altitude	Aerobic	Distance	2,500 – 3,000 m 14-15.5%	2-4 weeks
LHTL	Live at altitude, train at sea level	Aerobic	Distance	2,500 – 3,000 m 14-15%	2-4 weeks
LHTLH	Live at altitude, train at altitude and sea level	Aerobic	Distance	2,500 -3,000 m 14-15%	2-4 weeks
IHT	Live at sea-level with training in hypoxia and normoxia	Aerobic	Distance	10.5 – 15%	Up to 3 hours per day for 1 to 4 weeks
RSH	Sprints < 30 seconds in hypoxia	Anaerobic	Repeated and Single Sprint	13-15.5%	Minutes per day for 1- 4 weeks
RSH-VHL	Voluntary hypoventilation at low lung volume	Anaerobic	Repeated and Single Sprint	NA	Minutes per day for 1-4 weeks

Note. LHTH = live high train high; LHTL= live high train low; LHTLH = live high train low and high; LLTH = live low and train high; IHT= intermittent hypoxia training; RSH = repeated sprints in hypoxia; RSH-VHL = voluntary hypoventilation at low lung volume; NA = not applicable FiO_2= fraction of oxygen in inspired air. *Adapted from Millet et al. 2010.*

Section II

Live High Train High Training Approach (LHTH)

The LHTH hypoxic training approach, often called traditional or classic altitude training became popular after the 1968 Olympics (Flaherty et al., 2016; Grégoire P Millet et al., 2010). At that time, athletes typically ascended to a moderate altitude (2,000 to 3,000 m) to live and train for two to four weeks, often multiple times per year (Bärtsch & Saltin, 2008; Saunders, Pyne, et al., 2009). Currently, numerous hypoxic training camps worldwide are used regularly by athletes (J. Daniels & Oldridge, 1970; Grégoire P Millet et al., 2010; Paul Robach et al., 2014).

Based on reports from coaches of improved athletic performance following altitude training, scientists investigated four distinct phases of LHTH training. According to Millet et al., the validity of these phases remains under debate by scientists (Robert F. Chapman, Stickford, Lundby, & Levine, 2014; Grégoire P Millet et al., 2010). Phase 1 is an acclimatization period that lasts for 7-10 days. While the athletes acclimatize to the reduced partial pressure of inspired oxygen (PiO_2), fluids are increased with exercise limited to mild aerobic work. A training phase (2) follows acclimatization. During phase 2, training volume and intensity progressively increase to sea-level values. Training continues for two to four weeks, followed by two to three days of reduced training while still at altitude (Phase 3). After the reduced training phase, the athletes return to sea level.

Once the athletes returned to sea-level, the timing of peak performance was difficult to predict. Peak performances occurred across a continuum ranging from the first few days after descent to three weeks later at the major competition (Issurin, Kaufman, Lustig, & Tenenbaum, 2008; Grégoire P Millet et al., 2010; Rodríguez et al., 2015). The factors responsible for the timing of peak performance following hypoxic training are unclear. Potential causes include the

longevity of the physiological adaptations induced during hypoxic training, individual variation in response to training, and differences in post-camp training between athletes (Grégoire P Millet et al., 2010).

Interestingly several authors reported that after LHTH training, athletes returned to sea-level in a deconditioned state (Buskirk, Kollias, Akers, Prokop, & Reategui, 1967; Randall L. Wilber, 2007). They suggested that above 1500 meters (exceeded during LHTH), aerobic power declined at a rate of 1% for every 100-meter increase in elevation. During LHTH training, the decline in aerobic power caused a reduction in training intensity and a subsequent decrease in fitness (B. D. Levine & Stray-Gundersen, 1992; Robergs, Quintana, Parker, & Frankel, 1998; Saltin, 1967). Evidence also suggested that the fittest athletes with the highest maximal oxygen consumption (VO_{2max}) experienced the largest declines. However, interindividual differences were also reported (Koistinen, Takala, Martikkala, & Leppäluoto, 1995; Robergs et al., 1998). To maintain the adaptations induced during acclimatization and counter the decreased training intensity, Levine and Stray-Gundersen proposed the LHTL training approach (B. D. Levine & Stray-Gundersen, 1997).

Live High Train Low Training Approach (LHTL)

The LHTL approach involves living and sleeping at elevation (2000-3000 m) where the fraction of oxygen in inspired air (FiO_2) is between 13% and 15.5% and training low (< 1500 m), where the FiO_2 is greater than 17% (D. L. Bonetti et al., 2009; B. D. Levine & Stray-Gundersen, 1997; Randall L. Wilber, 2007). Today it is no longer necessary to ascend to altitude because normobaric hypoxic tents and chambers are commercially available and reasonably priced. Due to the enhanced logistics, affordability, and apparent performance gains, normobaric LHTL training has become the hypoxic approach preferred by professional and elite endurance athletes (Grégoire P Millet et al., 2010). Chronic hypoxic exposure, however, can induce

decreased muscle function, negatively impacting athletic performance at sea-level (B. D. Levine & Stray-Gundersen, 1997). Therefore, to prevent the loss of muscle function and maintain the benefits of LHTL training, Millet et al. proposed adding training in normoxia and called the new approach live high train low and high (LHTLH) (Grégoire P Millet et al., 2010).

Live High Train Low and High Training Approach (LHTLH)

Millet et al. described the LHTLH approach as a combination of LHTL training and intermittent hypoxia exposure (IHE) (Grégoire P Millet et al., 2010; Powell & Garcia, 2000). The hematological benefits (described below) were maintained during LHTLH training by alternating five nights of sleeping in hypoxia with two nights in normoxia. Furthermore, to take advantage of the hypoxia-induced anaerobic and aerobic adaptations, the LHTLH approach utilized high-intensity training in normoxia and hypoxia (Brocherie, Millet, et al., 2015; Robertson, Saunders, Pyne, Gore, & Anson, 2010). LHTLH training targeted improving performance in endurance events and was the first hypoxic approach to focus on repeated sprint performance (Brocherie, Millet, et al., 2015). To date, a limited number of LHTLH studies have been conducted. Therefore, investigations into the effect of LHTHL training on athletic performance are included in the review of the LHTL literature.

The Rationale for the LH Training Approaches

In 1992, Levine and Stray-Gundersen proposed that improvements in athletic performance following the LH hypoxic training approaches resulted from the physiological adaptations resulting from acclimatization, the increased training stimulus from the hypoxic stress, or a combination of both (B. D. Levine & Stray-Gundersen, 1992). Years later, they concluded that acclimatization was the more influential (B. D. Levine & Stray-Gundersen, 2005). Acclimatization is the physiological process of adapting to the demands imposed on the

body during exposure to hypoxia. Specifically, at natural altitude, the reduced partial pressure of inspired oxygen (PiO_2) stimulates physiological adaptations that compensate for the reduced availability of oxygen (Faiss, Leger, et al., 2013; B. D. Levine & Stray-Gundersen, 1997). One of those adaptions is an increase in red blood cells. There is a consensus, although not universal acceptance (B. D. Levine & Stray-Gundersen, 2005), that the increase in erythrocyte count is the key hypoxic-induced adaptation underlying additional improvements in athletic performance and the primary reason that athletes use the LH training approaches (B. D. Levine & Stray-Gundersen, 1992, 1997, 2005; Randall L. Wilber, 2007). Therefore, the goal of maximizing the hypoxia-induced increase in erythrocyte volume is common to all the LH training approaches (Saunders, Pyne, et al., 2009).

Beginning with a hypoxia-induced increase in erythropoietin (EPO), LH training stimulates a physiological cascade that leads to an increase in red blood cells. Erythropoietin, a glycoprotein hormone produced from renal EPO-producing cells (Souma, Suzuki, & Yamamoto, 2015), controls red blood cell production (Jelkmann, 2016). During LH training, the reduced partial pressure of oxygen causes a compensatory increase in EPO levels, stimulating the bone marrow to increase red blood cell production. Figure 2.1 summarizes this hematological response to LH training (B. D. Levine & Stray-Gundersen, 1997; Saunders, Pyne, et al., 2009).

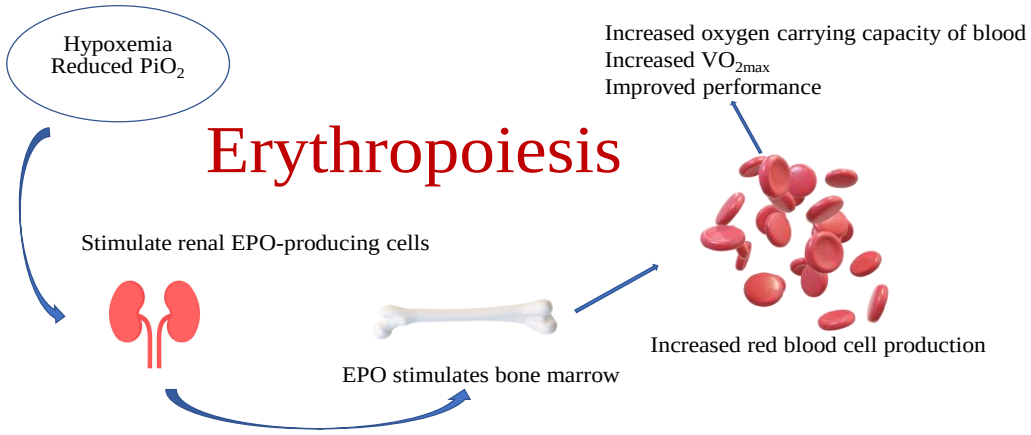


Figure 2.1: Hematological Response to LH training adapted from Souma 2015, Jelkmann 2016.

Erythrocyte or red blood cell count is an important determinant of aerobic capacity. Aerobic capacity is quantified as maximal oxygen consumption ($\dot{V}O_{2max}$). $\dot{V}O_{2max}$ is a whole-body assessment of cardiorespiratory fitness, measured in absolute ($L \cdot min^{-1}$) or relative terms ($mL \cdot min^{-1} \cdot kg^{-1}$) (Brutsaert, 2008). A higher $\dot{V}O_{2max}$ directly correlates with athletic performance in middle and distance events (Brutsaert, 2008; Grégoire P Millet et al., 2010; Paul Robach et al., 2014; Rodríguez et al., 2015; Wachsmuth, Volzke, et al., 2013; Wehrlin & Marti, 2006). Therefore, LH training aims to improve performance in the middle and distance events by increasing red blood cell volume generating a concurrent improvement in VO_{2max}. The effects of LH training on EPO levels, red blood cell volume, VO_{2max}, and athletic performance are presented below.

LH Training: Impact on EPO Levels

Following numerous LHTH studies, a general agreement emerged that EPO levels increased following two to four weeks of exposure to a moderate altitude of approximately 2,200 m (B. D. Levine & Stray-Gundersen, 1997; Saunders, Pyne, et al., 2009; Stray-Gundersen et al., 2001). However, due to a detraining effect linked to hypoxia and poor athletic performance following LHTH training, the LHTL approach was proposed. LHTL training was designed to preserve the increase in EPO levels while permitting the athlete to train at sea-level intensity.

Results of the LHTL studies conducted in the early 1990s confirmed increased EPO levels comparable to those observed after LHTH training. Subsequent research provided additional support for an EPO response to LHTL training (Brugniaux, Schmitt, Robach, Jeanvoine, et al., 2006; Dehnert et al., 2002; Garvican-Lewis, Clark, Polglaze, McFadden, & Gore, 2013; Gough et al., 2012; B. D. Levine & Stray-Gundersen, 1997; Grégoire P Millet et al., 2010; Rodríguez et al., 2015; Stray-Gundersen & Levine, 2008; Wehrlin & Marti, 2006). For example, Levine et al. (B. D. Levine & Stray-Gundersen, 1997) demonstrated an approximate doubling in EPO levels after athletes lived for 27 days at 2,500 m and trained at 1,250 m. Similarly, Stray-Gundersen et al. found EPO levels increased by 92% within the first 20 hours of hypoxic exposure (Stray-Gundersen & Levine, 1999). Four years later, Stray-Gundersen et al. (Stray-Gundersen et al., 2001) duplicated their earlier finding of EPO levels almost doubling after LHTL (2500 m, 1250 m) training in elite athletes. Table 2.2 presents findings on the impact of LHTL training on Hb levels, VO_{2max}, and performance compared to the equivalent training in normoxia.

When the hypoxic dose consisted of short exposures to hypoxia at lower altitudes, the increase in EPO levels was smaller and transitory. For example, Dehnert et al. (Dehnert et al., 2002) trained fifteen male and six female endurance athletes for two weeks. High-intensity

training was performed at low altitudes (800 m) and moderate training at 1960 m. Initially, EPO increased by 30% ($p < 0.012$), far less than in the studies mentioned above and then quickly (2 days) declined to sea-level values. The control group showed no change in EPO levels. Contrastingly, at higher altitudes (2500 m) and over a shorter timeframe (7 days), Laitinen et al. (Laitinen H, 1995) observed an 84% increase in serum EPO levels in male runners. Furthermore, the runners spent 16-18 hours daily at 2500 m, 13 hours longer and higher than in the Dehnert et al. study (B. D. Levine & Stray-Gundersen, 2006). As a result of these and numerous other studies, there is general agreement that LHTL training does stimulate EPO production (Grégoire P Millet et al., 2010). However, there is one caveat, for the increase in EPO to occur, the hypoxic dose must be sufficient.

The recommendation for an optimal hypoxic dose varied depending on the hypoxic approach and the type of exposure, natural altitude or normobaric hypoxia. For instance, Levine and Stray-Gundersen estimated that an altitude of 2,000 to 2,500 m is high enough and ~20 hours per day for three to four weeks long enough to maximize the erythropoietic effect during LHTL training (B. D. Levine & Stray-Gundersen, 2005, 2006; R. L. Wilber, Stray-Gundersen, & Levine, 2007). On the other hand, Wilber suggested 12-16 hours per day of hypoxic exposure at an equivalent altitude of 2,000-2,500 m for normobaric hypoxia LHTL training (R. L. Wilber et al., 2007). Although some studies demonstrated increased EPO levels at altitudes below 2,000 m, the consensus range for LH training is 2,000 to 3,000 m (R. F. Chapman et al., 2014). If the minimum hypoxic dose was utilized during LHTH training, there was general agreement that EPO levels would increase. However, the impact of the LHTH approach on hemoglobin mass and red blood cell volume was less clear.

LH Training: Impact on Hemoglobin (Hb) and Red Blood Cells

Theoretically, the key hypoxia-induced adaptation underlying improvements in aerobic capacity following LH training is augmented hemoglobin mass (Lundby et al., 2012; Grégoire P Millet et al., 2010). Many hypoxic training studies quantified aerobic capacity as VO_{2max}. $\dot{V}O_{2max}$ is the product of cardiac output ($\dot{Q}$), and the extraction of oxygen from the blood defined as the arterial-venous oxygen difference (A- VO_2 difference) and is calculated using the Fick equation (1) (Wilmore J., 1994).

$$VO_{2max} = (\dot{Q} \text{ x A} - VO_2 \text{ difference}) \quad (1)$$

The arterial-venous oxygen difference depends partly on the oxygen-carrying capacity of the blood (CaO_2). In circulation, oxygen is carried bound to hemoglobin (Hb). Therefore, any factor influencing Hb levels will impact CaO_2 and VO_{2max}. Wachsmuth et al. estimated an approximately 4 mL/min increase in VO_{2max} for every 1 g increase in Hb_{mass} (Wachsmuth, Völzke, et al., 2013). Thus, the rationale for the prevailing theory of a hypoxia-induced hematological improvement in VO_{2max} following LH training. However, accepting this rationale may be premature (B. D. Levine & Stray-Gundersen, 2005).

Indeed, several LH hypoxic training studies demonstrated increased red blood cell volume and hemoglobin levels following training (R. F. Chapman et al., 2014; Mounier et al., 2006; Ploszczyca, Langfort, & Czuba, 2018; Wehrlin & Marti, 2006; R. L. Wilber et al., 2007). However, after hypoxic training, others demonstrated no change in red blood cell volume and hemoglobin content (Ashenden, Gore, Dobson, & Hahn, 1999; Dehnert et al., 2002; B. Friedmann et al., 1999). Notably, Brocherie et al. found Hb_{mass} increased significantly following LHTHL (+ 4%, $p < 0.001$) and LHTL training (+ 3%, $p < 0.001$) compared to a LLTL group (no change). Contrastingly, the LHTHL group in the Altitude Project showed no significant change in Hb_{mass} from pre-training measurements. However, in the same study, Hb_{mass} for the two LHTH

groups (3 & 4 w) significantly increased through the duration of the study (766 ± 187 g to 810 ± 204 g, $p = 0.02$ and 816 ± 204 g to 857 ± 223 g, $p < 0.001$ respectively).

Reaching a consensus concerning the hypoxia-induced change in Hb_{mass} and red blood cell volume following LH training is complex due to numerous confounding factors. For example, changes in Hb_{mass} appeared to be inversely related to the participant's initial $Hb_{mass,}$ and the response varied in the same individual from year to year (Lundby et al., 2012; McLean, Buttifant, Gore, White, & Kemp, 2013). However, this initial claim was refuted (G. P. Millet, Chapman, Girard, & Brocherie, 2019). Moreover, elite athletes with pre-existing high Hb_{mass} demonstrated 3% to 4% gains in Hb_{mass} following LHTL training (Grégoire P Millet et al., 2019). Additional confounding factors include variations in hypoxic dose, type of training, the athletic status of the participants, previous hypoxic exposure, and interindividual variability (B Friedmann et al., 2005; McLean et al., 2013; Mujika, Sharma, & Stellingwerff, 2019). Therefore, to clarify the impact of LH training on Hb_{mass} and red blood cell volume, future studies must account for these confounding factors.

Given the consensus that following LH training, EPO levels increased, but the changes in red blood cell volume and hemoglobin content were uncertain, what does the literature suggest about the impact of the LH training approaches on improvements in VO_{2max}? Following LH training, improvements in VO_{2max} varied depending on the training approach used in the study. Albeit, in studies evaluating identical approaches, the improvement in VO_{2max} beyond the same training in normoxia fluctuated widely (Darrell L. Bonetti & Hopkins, 2009b). The impact of each LH training approach on changes in VO_{2max} is summarized below.

LHTH Training Approach: Impact on VO_{2max}

The LHTH studies conducted in the 1970s reported hypoxia-induced additional improvements in VO_{2max} beyond the same training in normoxia. However, the sample size for those studies was small and lacked appropriate controls (B. D. Levine & Stray-Gundersen, 1997). Subsequent research questioned the additional benefit of LHTH training on improvement in VO_{2max} beyond the same training in normoxia (Adams, Bernauer, Dill, & J. B. Bomar, 1975). For example, in three recent swimming studies, the differences in swim-specific VO_{2max} or VO_{2peak} following LHTH training did not reach significance despite significant increases in Hb_{mass} (Bonne et al., 2014; Rodríguez et al., 2015; Wachsmuth, Volzke, et al., 2013). Furthermore, in their meta-analysis, Bonetti and Hopkins concluded that LHTH training improved VO_{2max} in subelite athletes with possible impairment in elite athletes (Darrell L. Bonetti & Hopkins, 2009b). The impairment was associated with decreased training intensity due to a hypoxemia-linked reduction in maximal aerobic power of ~ 1% for every additional 100 m above 1500 m (Buskirk et al., 1967). As previously noted, the LHTL approach was proposed to mitigate the detraining effect observed following LHTH training.

Table 2.2 Hemoglobin, VO_{2max} and Performance After LHTH Training

Study	Design	Participants f or m, Sport	Altitude FiO_2	Duration x frequency weeks (w) x sessions per week (s/w) or days (d)	Training	Hb_{mass} (g/dl)	Δ VO_{2max} ($ml \cdot min^{-1} \cdot kg^{-1}$)	Δ Performance
Buskirk et al. (1967)	Case	(*n* = 6,) m	4,000 m 2,300 m	~ 8 x 6 d ~ 1 x 6 d	individual	NM	NSC from SL	NSC from SL
Adams et al. (1975)	Crossover no washout	(*n* = 12) ,m runners	2,300 m	2 w x 3 s/w	75% VO_{2max}	NM	NSC	NSC
Burtscher et al. (1996)	Control one year later	(*n* = 10) , m amateur runners (n = 12) control	2350 m	12 d	aerobic and interval training	NM	↑10% $p < 0.05$ post 16 d H > N $p = 0.005$	↑ 8% $p < 0.05$ post 3 d ↑ 8% $p < 0.05$ post 16 d
Levine et al. (1997)	Parallel	(*n* = 13) f = 4 m = 9 runners competitive	2500 m	28 d	ND	↑ 9% $p < 0.05$	↑ 5% $p < 0.05$	5,000 m TT (+) 3.3 ± 9 s, NS
Gore et al. (1998)	Case no control	(*n* = 8), m cyclists, elite	2690 m	31 d	ND	NC	NC	IP_{4000} (+) 4.3% $p = 0.009$

Wachsmuth et al. (2013)	Parallel	(*n* = 19) f = 6, m = 13 swim	2,320 m	4 w x 6 s/w	ND	+ 7.2 ± 3.3 %	NM	Significant improvement
Bonne et al. (2014)	Parallel	(*n* =10) f = 5 m = 5 swim	3,090 m 2,130 m	6 d 3 w x 6 s/w	ND	+ 6.2 ± 3.9 %	NSC	NSC
Rodriguez et al. (2015)	Parallel	(*n* = 16) f= 10 m = 6 swim	2,320 m	4 w x 6 s/w	ND	+ 6.2 ± 1.1 %	NSC	Unclear
Rodriguez et al. (2015)	Parallel	(*n* = 15) f= 8 m = 7 swim	2,320 m	3 w x 6 s/w	ND	+ 3.8 ± 2.3 %	NSC ($ml \cdot min^{-1}$)	NSC

Note, ND = not defined; NM = not measured; NSC = no significant difference in post-training improvement between hypoxia and normoxia; TT = time trial; IP_{4000} = individual pursuit cycling performance; SL = sea-level.

LHTL Training Approach: Impact on VO_{2max}

During the 1990s, LHTL was the preferred hypoxic training approach for athletes and the focus of numerous studies (Darrell L. Bonetti & Hopkins, 2009b; Grégoire P Millet et al., 2010; Saunders, Garvican-Lewis, Schmidt, & Gore, 2013). The change in VO_{2max} following LHTL training was often investigated. Based on the results reported in the scientific literature, the impact of LHTL training on improvements in VO_{2max} beyond the equivalent training in normoxia remained unclear. For example, Levine and Stray-Gundersen conducted one of the first LHTL studies. In that study, after four weeks of LHTL training (2,500 m, 1250 m), 39 runners (27 m, 12 f) increased their VO_{2max} ($ml \cdot min^{-1} \cdot kg^{-1}$) by 5% over sea-level controls ($p < 0.05$). In addition, red blood cell mass increased by approximately 10%. The authors credited the improved VO_{2max} to an “acute erythrocyte infusion" (B. D. Levine & Stray-Gundersen, 1997). Several studies demonstrated similar results (Mellerowicz et al., 1970; Stray-Gundersen et al., 2001).

The conclusion that LHTL training enhanced VO_{2max} was not unanimous. For example, the opposite conclusion was reached by the Altitude Project, a controlled, nonrandomized, parallel-group (4) study with swimmers that examined several physiological outcomes, including VO_{2max}. Participants were divided into two groups of LHTH (2320 m), one each of LHTLH (2320 m, 690 m), and SL (sea-level). Training duration between the two LHTH groups differed (3 or 4 weeks). Pre- and post-measurements of VO_{2max} did not differ significantly between any groups. As a result, they concluded that the significant increases in total hemoglobin mass (tHb_{mass}) following training in the two LHTH groups were not associated with changes in VO_{2max} (Rodríguez et al., 2015), supporting similar findings from several LHTL studies (Brugniaux, Schmitt, Robach, Nicolet, et al., 2006; Piehl Aulin, Svedenhag, Wide, Berglund, & Saltin, 1998; Saunders, Telford, Pyne, Hahn, & Gore, 2009). Additionally, Bonetti and Hopkins conducted a

Meta-Analysis of the effect of the LH training approaches on exercise performance. Of the 51 hypoxic training studies included, 12 were LHTL investigations. Based on their analysis, Bonetti and Hopkins could not determine the effect of LHTL training on improvements in VO_{2max} in subelite or elite athletes. Several factors moderated VO_{2max}. The most notable was that the highest VO_{2max} values occurred two weeks post-LHTL training (Darrell L. Bonetti & Hopkins, 2009b). Therefore, LH studies that did not measure VO_{2max} two weeks post-hypoxic training may have missed a significant training effect.

Athletes continued use of the LH training approaches is prima facie evidence of their expectation that hypoxic training will improve performance in distance events such as the 5,000-meter run (Siebenmann & Dempsey, 2020). However, given the lack of a consensus in the scientific literature regarding the additional benefit of the LH training approaches on hemoglobin mass and $VO_{2max,}$ is their expectation justified? In other words, does LH training improve performance in distance events beyond the same training in normoxia?

LHTH Training Approach: Impact on Athletic Performance

Early LHTH studies demonstrated a moderate positive effect of LHTH training on VO_{2max} and performance. Those studies were limited by their small sample sizes and lack of control groups. However, two early studies included a control group (Friedmann-Bette, 2008). Burtscher et al. demonstrated significantly greater improvement in performance on an incremental bike test in absolute VO_{2max} ($l \cdot min^{-1}$) and relative VO_{2max} ($ml \cdot min^{-1} \cdot kg^{-1}$) after LHTH (2315 m) compared to LLTL training (187 m) in a cohort of amateur runners (Burtscher, Nachbauer, Baumgartl, & Philadelphy, 1996). Three days after returning to sea-level, the improvement in VO_{2max} between groups was no longer significant. However, 16 days post descent, VO_{2max} in the LHTH group was significantly greater than in the LLTL group (p =

0.005). Notably, the normoxia portion of the study was conducted one year after LHTH training. In addition, training intensity was based on the heart rate at 4 mmol/L during an incremental bike test performed at sea-level. Training at the same heart rate during LHTH and LLTL workouts would produce relatively higher exercise stress at lower absolute workloads during LHTH training. In other words, the impact of the training protocol was different between the two groups, potentially influencing improvements in VO_{2max} and performance (Friedmann-Bette, 2008; B Friedmann et al., 2005; Koistinen et al., 1995).

In contrast, in a cohort of distance runners, Levin and Stray-Gundersen demonstrated a 5% improvement in VO_{2max} ($p < 0.05$) following LHTH training compared to no change in the sea-level control group. Furthermore, performance in the 5,000 m run time trail in the LHTH group increased by 3.3 ± 9 s compared to an increase of 26. 7 ± 13 s in sea-level controls. The slower performance in the LHTH group occurred despite the improvement in VO_{2max} ($p < 0.05$). On the other hand, VO_{2max} in the sea-level groups did not change. The authors concluded that LHTH training did not provide an additional benefit on performance compared to sea-level training but did improve VO_{2maz}. Their conclusion supported the results from previous studies. However, in the earlier studies, the altitude and timeframe used for training (1800 and 2000 m for 2 weeks or less) were below the current recommendations for LH training of an altitude between 2,000 to 3,000 m for 3-4 weeks (Adams, Bernauer, Dill, & Bomar, 1975; Bailey et al., 1998; R. F. Chapman et al., 2014; Ingjer & Myhre, 1992; Jensen et al., 1993; Svedenhagl, Saltin, Johanssonz, & Kaijserl).

Subsequent studies presented conflicting findings, with most demonstrating no additional benefit of LHTH training on performance (Bonne et al., 2014; Rodríguez et al., 2015). Only one study, whose participants were among the top 58 swimmers from Germany (27 participated in

the Olympics), concluded that LHTH training improved performance beyond the same training in normoxia (Wachsmuth, Volzke, et al., 2013). However, a control group was not included in the study. Furthermore, the duration of altitude training varied between participants. Both factors weaken their conclusion of an LHTH-induced improvement in swimming performance. Currently, there is no consensus regarding the effect of LHTH training on improvement in athletic performance compared to the same training in normoxia. Due in part to these equivocal findings, LHTH training has largely been replaced by the LHTL approach (Darrell L. Bonetti & Hopkins, 2009a; Grégoire P Millet et al., 2010)

LHTL Training Approach: Impact on Athletic Performance

The initial LHTL study is often credited to Levine and Stray-Gundersen. However, an earlier study completed in 1970 may have been the first to use the LHTL approach. Mellerowicz et al. trained two groups (n = 22, sea-level, and LHTL) of middle and distance runners in that study. The LHTL group slept at 2020 m and trained at 1,800 m and 2,500 m. LHTL training compared to sea-level training resulted in significantly greater improvement in VO_{2max} (16% vs. 4%, $p < 0.001$) and performance in a 3,000 m time trial (5% vs. 3%, $p < 0.05$). In the Levine and Stray-Gundersen LHTL study previously described in this review, 5,000 m run time was significantly faster (-13.4 ± 10 s, $p < 0.05$) in the LHTL group compared to both the LHTH and sea-level groups. A subsequent study (2001) showed a 1.1 to 1.2% improvement in 3,000 m run time in elite male and female runners following LHTL training. They concluded that elite athletes benefit from LHTL training acknowledging that the lack of a control group was a major limitation (Stray-Gundersen et al., 2001). However, previous studies reported that the fittest athletes did not benefit from LHTL training. Therefore, Levine and Stray-Gunderson justified their study design based on the novelty of their findings.

In contrast, numerous studies reported that LHTL hypoxic training did not induce additional improvements in athletic performance compared to the equivalent training in normoxia. For example, Robach et al. found no significant difference in 2,000-meter swimming performance and swim-specific VO_{2max} in a cohort of French National Team swimmers following 5, 8, and 16 days of LHTL training compared to controls (P. Robach et al., 2006). Similarly, after LHTL training, Robertson et al. found no additional benefit on 5-km performance in elite runners. They tested the reproducibility of their findings using a five-week washout period. Subsequently, they repeated the same training program with similar results (Robertson, Saunders, Pyne, Aughey, et al., 2010).

Throughout the scientific literature, a pattern of uncertainty regarding LH training-induced additional improvements in red blood cell volume, $VO_{2max,}$ and athletic performance was evident. Table 2.3 presents the results from the studies reviewed here relative to the impact of the LHTL training approaches on EPO levels, Hb levels, VO_{2max}, and athletic performance. Study designs ranged from double-blind with controls to those without blinding or control groups. Participants included athletes from track and field, swimming, cycling, kayaking, and several were classified as endurance athletes. Participants were further defined by their competitive status as elite, subelite, or well-trained. To be classified as elite, the athlete had to have competed in national or international competitions, often at the Olympic level. It is important to note that elite, subelite, and well-trained were not consistently defined between studies. Additional differences between studies included the duration of the training (5 days to 4 weeks), duration of hypoxic exposure, and type of hypoxia (hypobaric or normobaric). Furthermore, between studies, performance assessments included sport-specific time trials, athletic competitions, and

specific aerobic tests. These variations in study design and methodology complicate reconciling contradictory conclusions between studies.

In their 2009 meta-analysis, Bonetti and Hopkins attempted to account for the numerous moderating factors that influenced the likelihood that LHTL training evoked additional improvement in performance beyond the corresponding training in normoxia (D. L. Bonetti et al., 2009). Their analysis addressed the link between physiological variables and performance improvements following LHTL. Fifty-one studies published through 2007 that focused on post-hypoxia training performance at or near sea-level (<1000 m) following LHTL training were included in the analysis. Studies without control groups were also included. Rather than disqualifying the 55% of studies that lacked inferential information to support p-value inequality, they derived a weighting factor (study sample size)/(mean study sample size) from the sample size for each study estimate.

Runners, swimmers, rowers, cyclists, triathletes, skiers, kayakers, hockey players, and multi-sport athletes were investigated. Based on their classification in the original study, participants were categorized as elite, sub-elite, or trained athletes. The exact sample size for each mean effect was not published. The analysis was conducted using modeling procedures in SAS. Assessments of athletic performance averaged between 1-4 minutes in duration. Bonetti and Hopkins concluded that the performance of sub-elite athletes benefited (1-4%) from both brief natural exposure and simulated altitude over various timeframes. Elite athletes benefited only from LHTH when conducted at natural altitude. They could not determine if an increase in VO_{2max} caused improvement in performance following hypoxia training. The authors concluded that improved study designs (double-blind) and performance measures with smaller measurement errors were necessary to clarify the efficacy of the LHTL hypoxic training

approach. Finally, they suggested that their analysis indicated a small to moderate hypoxia-induced benefit on performance in the middle distance and distance events.

Live High Train Low and High Training Approach: Impact on Athletic Performance

To maintain the beneficial aspects of LHTH and LHTL training, Millet et al. proposed the live high train low and high training approach (LHTLH) (Grégoire P Millet et al., 2010). The expected benefits included an increase in red blood cell volume and an improvement in VO_{2max}. LHTLH training was also designed to minimize the reduction in training intensity associated with the LH approaches. Therefore, during LHTLH training, athletes combine LHTL with intermittent hypoxic training (IHT). They also suggested alternating sleeping at night in hypoxia and normoxia on a 5 to 2 or 6 to 1 rotation.

The LHTLH training approach marked the first attempt to use hypoxic training to improve repeated sprint performance. In 2015 Brocherie et al. tested the effect of LHTLH training on performance in team-sport athletes (Brocherie, Millet, et al., 2015). They assessed hematological and performance changes using a randomized, double-blind study following 14 days of LHTLH, LHTL, and LLTL training combined with repeated sprint training in hypoxia or normoxia. Both hypoxia groups improved Hb_{mass} compared to no change in the control group (LHTLH: +4.0%, $p < 0.001$, LHTL: +3.0% $p < 0.001$). In addition, cumulated sprint time significantly improved in the LHTLH and LHTL groups (3.6%, $p < 0.001$ vs. 1.9%, $p < 0.01$, respectively). Interestingly the benefit persisted for three weeks post-training only in the LHTLH group ($p < 0.001$). Thus, the timeframe for improvement following LHTLH training appears to be very different from LHTH and LHTL training. Therefore, future studies should consider the timing of the post-training performance assessments. To date, few studies have investigated the

LHTLH training approach making it impossible to draw any conclusions as to the efficacy of the training.

Table 2.3 EPO, Hemoglobin, VO_{2max} and Performance Following LHTL Training

Author	Participants	Low Altitude (m)	High Altitude (m)	Duration	EPO	Hb_{mass} (g/dL)	VO_{2max} ($mL \cdot min^{-1} \cdot kg^{-1}$)	Performance
Levine and Stray-Gundersen 1997	(*n* = 13) runners	1250	2,500	28 days	NM	↑ 9% $p < 0.05$	↑~ 4 $p < 0.05$	5000 m (-) 13.4 ± 10 s, $p < 0.05$
Phiel-Aulin et al. 1998	(*n* = 6) (*n* = 9) endurance	sea-level sea-level	2,000 2,700	10 days, simulated, 12 hr/day	↑80% $p < 0.05$	NC NC	NC NC	NM NM
Ashenden et al. 1999	(*n* = 6), M endurance	600	3,000	23 days, 8-10 hours/night	NM	NC	NM	NM
Ashenden et al. 1999	(*n* = 6) , F endurance	600	2,650	12 days, 8-10 hours/night	NM	NC	NM	NM
Ashenden et al. 2000	(*n* = 6), M runners	600	2,650	5 nights, 8-11 hours	↑57% $p < 0.05$	NC	NM	NM
Hahn et al. 2000, multiple studies	*(n* = 12) , F cyclists (*n* = 9), 5 F, 4 m kayakers (*n* = 13), M triathletes (*n* = 11) M runners	Sea-level	2,650-3,000 m	23 d	NM	NS	NS	NS
Stray-Gundersen et al. 2001 no control	(*n* = 14), M (*n* = 8), F elite runners	1,250	2,500	27 days	↑2x $p < 0.05$	↑ 7% $p < 0.05$	↑ 3% 72.1 ± 6.9 to 74.4 ± 6.8 $p < 0.05$	3000 m (-) 5.8 s $p < 0.5$

Dehnert et al. 2002	(*n* = 21) triathletes well trained	800	~1,960	14 days	↑30% ↑14.3 +/- 8.7 (mU· ml^{-1}) p < 0.012	NC	NS *p* = 0.074	NS *p* = 0.068 to exhaustion
Roberts et al. 2002	(*n* = 19) 14, M 5, F cyclists well trained	610	2,650	8 to 10 hours/night 15 days	NM	NM	NS	NS $MMPO_4$
Hinckson et al. 2005	(*n* = 20) F = 4, M = 16 subelite run	Sea-level	2,500-3,500 m	4 w 9.8 h/day	ND	NS	NS	NS
Robach et al. 2006	(*n* = 18) elite swim	1,200 m	2,500 m 3,000 m	5 d 8 d 16 h day/d	NC	↑8.5% *p* = 0.03	NS	NS
Brugniaux et al**. 2005**	(*n* =11) elite middle-distance	1,200	2,500 3,000	6 nights 12 nights	↑ 27% transferrin receptor	↑ 10.1% *p* < 0.05	NS	NM
Saunders et al. 2007	(*n* =18), M elite run,	600	2,860	46 nights, 9 hours/night training at 600 m	NM	↑~5% *p* = 0.01	NS	NM
Robertson et al. 2010	(*n* = 16), F = 5 M = 11 elite run	600	3,000	14 h/d 3 weeks	↑ 90% at day 6	↑~ 3%	NS	NS 4.5-km TT

Siebenmann et al. 2012	(*n* = 16) cyclists, triathletes	1,020	3,000	4 weeks 16 h/d	14.9 ng/L^{-1} vs. NC $p < 0.05$	NS	NS ($mL \cdot min^{-1}$)	NS TT 26.15 km
Rodriguez et al. 2013	(*n* = 12) elite swim	690	2,320	4 weeks	NM	NS	NS	NS

Note. NM = not measured; ND = not defined; NC = no change after training; NS = no significant difference between groups; F = female; M = Male; TT = time trial; $MMPO_4$ = maximal mean power output during minute 4 of a cycle ergometer test; $mU \cdot ml^{-1}$ = enzyme units per mL *Adapted from Millet et al. 2010 and Bonetti and Hopkins 2009 plus individual studies*

Section III

Live Low Hypoxic Training Approaches

Intermittent Hypoxia Training (IHT)

IHT Impact on EPO Levels, Hematological Response and Performance

IHT was designed to induce an increase in EPO and stimulate a hematological response while minimizing the risk of decreased fitness associated with LH training (Benjamin D. Levine, 2002). The same hematological cascade observed following the LH training approaches was the proposed mechanism for improvements in VO_{2max} and athletic performance. Currently, the evidence does not support a hematological response following IHT. For example, in their 2006 study, Gore et al. investigated the effects of exposure to simulated hypoxia (4,000 to 5,000 m) on EPO levels for four weeks, three hours per day, five days per week. Despite an almost doubling in EPO levels, this double-blind, placebo-controlled study found no evidence of a hematological response (no change in Hb_{mass}) (Truijens et al., 2007). Furthermore, following IHT, serval research teams reported an EPO response without a change in Hb_{mass} (Grégoire P Millet et al., 2010; Sanchez & Borrani, 2018; Vallier, Chateau, & Guezennec, 1996).

Furthermore, in their 2012 review of IHT, Faiss et al., (Faiss, Girard, et al., 2013) provided substantial evidence that IHT did not improve athletic performance. In that review, performance gains beyond the same training in normoxia were demonstrated in only 4 of the 23 studies. However, control groups and training data were not reported for all four studies demonstrating performance improvement. Therefore, the authors could not attribute the performance gains to IHT (J. Daniels & Oldridge, 1970; B Friedmann et al., 2005; Gore et al., 1998; Mizuno et al., 1990). Following their extensive review, Faiss et al. concluded that there

was no benefit to IHT on sea-level performance and possibly a decrement after IHT, a conclusion supported by Lundby et al. (Lundby et al., 2012).

Given the lack of an increase in Hb_{mass} and performance improvement following IHT, the significant improvement in swimming and Wingate performance observed in a study conducted by Czuba et al. was unexpected. They trained 16 male swimmers randomly divided into hypoxia (n = 8; age 19.1 ± 1.3 years; $VO_{2max,}$ 4.25 ± 0.29 l· min^{-1}) and control groups (n = 7; age 20.5 ± 1.3 years; $VO_{2max,}$ 3.97 ± 0.41 l· min^{-1}). Both groups significantly improved swimming performance in the 100 m and 200 m freestyle time trails (IHT 2.1%, 1.8%) and controls (1.1%, 0.8%) ($p < 0.05$) following four weeks of IHT at a FiO_2 of 15.5% or in normoxia. There was no improvement in absolute VO_{2max} or hematological variables in either group. Based on the post-training decrease in blood lactate levels (hypoxia group) following a ramp test to exhaustion ($p < 0.01$) and improved Wingate mean power scores ($p < 0.001$), the authors suggested that enhanced anaerobic capacity accounted for the improvement in swimming performance (Czuba et al., 2013).

Repeated Sprint Training in Hypoxia (RSH)

The RSH training approach was proposed as a method to improve single sprint, repeated sprint performance and competitive outcomes in team sports that required repeated sprinting, such as soccer, tennis, rugby, hockey, and cross-country skiing (Aebi, Willis, Girard, Borrani, & Millet; Beard, Ashby, Chambers, Brocherie, & Millet, 2019; Billaut, Gore, & Aughey, 2012; C. Brechbuhl, Brocherie, Millet, & Schmitt, 2018; Buchheit, Mendez-Villanueva, Delhomel, Brughelli, & Ahmaidi, 2010; Faiss et al., 2015; Galvin, Cooke, Sumners, Mileva, & Bowtell, 2013). RSH was defined as multiple sprints ≤ 30 seconds in duration, performed on a work-to-rest ratio of 1:4 (Brocherie et al., 2017; Faiss, Leger, et al., 2013). Recently a modification of

RSH, RSH-VHL, has been introduced. RSH-VHL creates hypoxia through voluntary hypoventilation at low lung volume. Since its inception in 2013, several research teams have investigated the effect of RSH training on athletic performance.

The Rationale for Repeated Sprint Training in Hypoxia

Performance improvements in middle-distance and distance events following IHT were poor and unpredictable (Brocherie et al., 2017). Researchers cited a lower training intensity during IHT as a possible cause for the lack of improvement (Faiss, Girard, et al., 2013). However, evidence suggested that IHT may improve anaerobic capacity and sprint performance. Furthermore, previous hypoxic research demonstrated that at FiO_2 levels above 13.3%, athletes were able to maintain sea-level training intensity for short exercise bouts (Bowtell, Cooke, Turner, Mileva, & Sumners, 2013; Feriche et al., 2007; Ogawa et al., 2005). As a result of these two factors, researchers hypothesized that intensity could be maintained during repeated sprint training in hypoxia if short sprints and a FiO_2 level above 13.3% were used (Faiss, Leger, et al., 2013). Furthermore, if equivalent exercise intensities were maintained during hypoxia and normoxia training, the hypoxic stress during RSH should provide an additional training stimulus. Moreover, the hypoxia-linked increased training stimulus would induce adaptations to compensate for the reduced availability of oxygen. Those adaptations would result in additional improvements in single and repeated sprint performance beyond the equivalent training in normoxia (Brocherie et al., 2017; Faiss, Leger, et al., 2013). Th adaptations were presumed to be in the anaerobic metabolic pathways and their regulatory mechanisms (Faiss, Leger, et al., 2013). Figure 2.2, presented at the end of this section, depicts the RSH-induced improvement in performance, the physiological adaptations and the underlying mechanism regulating adaptation supported in the current scientific literature.

Purpose of RSH Training

RSH training was designed to improve athletic performance in single sprints and team sports involving repeated sprinting throughout the game or contest. Admittedly, the duration of the performances encompassed in this objective was broad, ranging from a 4-second sprint to a more than hour-long soccer match where sprinting was interspersed with periods of reduced exercise intensity (Spencer et al., 2005). However, athletic performance is a function of multiple factors, and a complete analysis is beyond the scope of this review. Therefore, this review focused on the impact of RSH training compared to RSN training on two factors that impact performance, anaerobic and aerobic capacity.

Potential Physiological Adaptations to RSH Training

Athletes have used the LH training approaches for over fifty years. As a result, the physiological adaptations stimulated by LH training have been the subject of numerous studies. There is general agreement that a hypoxia-induced increase in red blood cell volume is responsible for improved athletic performance following LH training. On the other hand, repeated sprint training in hypoxia is a relatively new hypoxic training approach. Currently, the mechanisms responsible for improvements in athletic performance following RSH training are being explored and remain partially hypothetical (Grégoire P Millet et al., 2019). Potential improvements following RSH are not linked to a hypoxia-induced hematological response. To date, a few studies have compared the physiological adaptations following RSH training to the same training in normoxia. The results of those studies are discussed below. For easy reference, Table 2.4 presents the RSH training studies reviewed. The table includes details of the study design, methodology and results.

The Impact of RSH Training Compared With Equivalent RSN Training on Blood Flow

In 2013, Faiss et al. conducted one of the first studies to compare the physiological adaptations between RSH and RSN training (Faiss, Leger, et al., 2013). They reasoned that during RSH training, a constant oxygen supply to muscle was maintained through an increased blood flow achieved through compensatory vasodilatation resulting in enhanced blood perfusion (Casey & Joyner, 2012). Furthermore, the stress on oxygen delivery during RSH may stimulate unique adaptations designed to maintain muscle oxygenation, specifically benefitting fast twitch fibers (Faiss, Girard, et al., 2013). In their single-blind, parallel study, 50 moderately trained participants (age 35 ± 7 years) exercised either in hypoxia or normoxia for 4 weeks. A control group performed no specific training. Hypoxic exposure was approximately 40 minutes per training session (320 minutes for the 4 weeks). Each session consisted of 3 sets of 5 x 10 s all-out sprints on a bike. Following RSH training, they reported an increase in the number of sprints to volitional exhaustion (9.4 ± 4.8 vs. 13.0 ± 6.2, $p < 0.01$) with no increase following RSN training (9.3 ± 4.2 vs. 8.9 ± 3.5). Delayed fatigue was attributed to a greater increase in muscle blood flow following RSH than RSN training ($p < 0.01$).

In a subsequent study, Faiss et al. used near-infrared spectroscopy (NIRS) to estimate blood flow (Faiss et al., 2015). Several authors have described the NIRS technique in detail (Boushel & Piantadosi, 2000; De Blasi et al., 1994). Briefly, NIRS is a noninvasive optical technique that uses an algorithm to calculate blood flow from hemoglobin levels measured during the procedure (Livera, Spencer, Thorniley, Wickramasinghe, & Rolfe, 1991). Based on NIRS results, Faiss et al. inferred RSH-induced additional improvements in blood perfusion, with the caveat that caution is applied to interpreting NIRS results. However, the results supported their earlier findings of a greater improvement in blood perfusion following RSH

compared to RSN training (Faiss, Leger, et al., 2013). Similarly, Montero and Lundby estimated muscle blood perfusion measured by NIRS in a cohort of physically active male cyclists (D. Montero & Lundby, 2017). Their findings provided additional support for a significantly greater increase in blood flow following RSH (20.9%) compared to RSN training (13.8%).

Improvements in blood flow following RSH training were suggested as a beneficial adaptation relative to enhanced athletic performance. During exercise, inorganic phosphate levels (Pi) increase with a concurrent decrease in muscle force production (Westerblad, Allen, & Lännergren, 2002). Enhanced blood flow facilitates the removal of metabolic waste products such as P_i, leading to improved force production by the muscle. This adaptation may underlie the improvement in sprints to exhaustion following RSH training observed by Faiss et al. (Endo et al., 2005; Faiss et al., 2015).

The Impact of RSH Training Compared With The Equivalent RSN Training on Glycolytic Enzymes and Substrates

To assess the impact of RSH training on anaerobic capacity, pre-to-post changes in regulatory enzymes and the substrates involved in anaerobic metabolism have been investigated. For example, in one study, post-RSH phosphofructokinase (PFK) activity, a key regulator of glycolysis, increased significantly more ($p < 0.05$) compared to the equivalent training in normoxia (59% vs.17%). Furthermore, the increase was linked to improvement in the anaerobic threshold (Puype et al., 2013). Moreover, changes in muscle glycogen and phosphocreatine (PCr) content, both substrates for anaerobic metabolism, have been investigated (J. Bangsbo, Gollnick, Graham, & Saltin, 1991). For example, after five consecutive days of repeated sprint training in hypoxia (14.5%) or normoxia (20.9%), muscle glycogen content (RSH 79.9 ± 10.4%; RSN, 56.3 ± 11.2%) and PCr content (RSH 3.9 ± 1.4%, RSN 2.7 ± 1.1%) increased in 19 male sprinters

with no significant difference in the relative increase between groups (Kasai et al., 2017). This finding suggests that muscle glycogen and PCr content are similarly impacted by repeated sprint training in hypoxia and normoxia. Furthermore, additional RSH-induced improvements in pH regulation beyond the same training in normoxia are being investigated. (C. Brechbuhl et al., 2018; Faiss, Leger, et al., 2013; Puype et al., 2013). However, additional research is needed to clarify the mechanism(s) underlying additional improvement in anaerobic capacity beyond the equivalent training in normoxia.

The Impact of RSH Training Compared With The Equivalent RSN Training on Whole Body Anaerobic Capacity

In the literature, the definition of anaerobic capacity was often unclear (Green, 1994). Throughout this review, anaerobic capacity "refers to the maximal amount of ATP resynthesized via anaerobic metabolism (by the whole organism) during a specific type of short duration, maximal exercise" (J. Bangsbo et al., 1990; Green, 1994). The Wingate Anaerobic Capacity test (WAnT) was designed to estimate whole-body anaerobic capacity and is considered the gold standard (Bar-Or, 1987). The WAnT is an all-out 30-second sprint most often conducted on a bicycle ergometer, but any ergometer can be used (Bar-Or, 1987; Dotan & Bar-Or, 1983; Zupan et al., 2009). Anaerobic capacity is estimated from four WAnT power output scores. The scores include peak power (PP, highest average score over 5 seconds), mean power (MP, average power output over the 30-second test), low power (LP, lowest average low power over 5 seconds) and the fatigue index (FI, (PP-LP)*100) (Bar-Or, 1987). Upper-body anaerobic capacity is assessed using an arm-based modification of the WAnT. This modification originally employed an arm crank ergometer (Dotan & Bar-Or, 1983). Subsequently, researchers developed sport-specific

Wingate tests. For example, a rowing ergometer was used to evaluate oarsman and oarswoman (rowing) (Klasnja et al., 2010).

To date, few studies have compared the effect of RSH training and the same training in normoxia on changes in Wingate scores. In the first RSH study, Faiss et al. trained cyclists 2 times per week for 4 weeks in hypoxia (FiO_2 14.6%) or normoxia using 3 sets of 5 x 10 s maximal sprints with 5 min rest between sets (Faiss, Leger, et al., 2013). In a second study, Kasai et al. trained 19 male college track and field athletes twice each day for five consecutive days in hypoxia (14.5%) or normoxia (20.9%) using 4 sets of 20 s maximal sprints with 5 to 15 min rest between sprints plus a 10 s maximal sprint for the morning session. The afternoon session consisted of 5 sets of 6 s maximal sprints with 36 s rest between sprints on a bike and 4 x 20 s submaximal running sprints on a treadmill (Kasai et al., 2017). Both studies reported no significant difference between the improvement in WAnT scores following RSH and RSN training. Additional studies evaluating the impact of RSH training compared to RSN training on improvement in WAnT performance are needed before a definitive conclusion can be reached.

The Impact of RSH Training Compared With The Equivalent RSN Training on Repeated Sprint Ability (RSA)

The construct of repeated sprint ability (RSA) is closely related to whole-body anaerobic capacity. RSA is defined as the ability to maintain a high percentage of maximal speed or power throughout a contest, game, meet, or test set, with incomplete recovery between sprints (Hancock et al., 2015; Wadley & Le Rossignol, 1998). RSA may be an important factor in sports where success depends on maintaining a high percentage of maximal speed throughout the contest (Faiss, Girard, et al., 2013; Wadley & Le Rossignol, 1998). Unlike the WAnT with its standard

protocol, RSA was often tested using various sport-specific repeated sprint tests (RST). Most repeated sprint tests were designed to match the performance demands of a specific sport.

RSA may reflect both anaerobic and aerobic capacity. For example, repeated sprinting depletes phosphocreatine stores (PCr) and increases lactate levels, indicative of anaerobic metabolism (B. Dawson et al., 1997). However, removing metabolic waste and replenishing PCr stores depend partly on aerobic metabolism (Spencer et al., 2005). Furthermore, the ability to maintain performance during repeated sprinting has been linked to aerobic power (B Dawson, Fitzsimons, & Ward, 1993). Therefore, to estimate the aerobic and anaerobic components of RSA, several scores are often calculated from a repeated sprint test, including the fastest time, average time, the rate of fatigue, the number of sprints completed to exhaustion or until speed declines to less than 70% of the fastest sprint.

When RSA was quantified as delayed fatigued on a repeated sprint test, Faiss et al. found that RSH evoked significantly greater improvement ($p < 0.001$) compared to RSN training (Faiss, Leger, et al., 2013). On the other hand, using the same repeated sprint test, Montero and Lundby found no difference in the number of sprints to exhaustion (David Montero & Lundby, 2016). Several factors may have contributed to the conflicting results between studies. For example, the hypoxic exposure was longer in the Montero and Lundby study (14 workouts vs. 8), the FiO_2 lower (13.8% vs. 14.6%), and the number of sprints greater (20 vs.15). The 13.8% FiO_2 was marginally above the 13% level associated with decreased training intensity and may have impacted the effectiveness of the training (P. S. R. Goods, Dawson, Landers, Gore, & Peeling, 2014). Although the athletes were similar in that, they were moderately trained male cyclists, the Montero and Lundby participants (age 24.9 ± 3.7 y, body weight 71.7 ± 3.1 kg) were specifically endurance-trained and approximately 11 years younger than those in the Faiss et al. cohort (age

35. ± 7 years, mass 75 ± 69 kg) making it difficult to compare the studies. Interestingly, a RSH study that incorporated voluntary hypoventilation at low lung volume (RSH-VHL) to create hypoxia demonstrated significantly greater improvement in the number of 15 m swimming sprints to exhaustion in the RSH compared to the RSN group (Trincat, Woorons, & Millet, 2017). However, unlike RSH, where the athlete remains in the hypoxic environment during the rest period during RSH-VHL, the arterial saturation levels increase during the recovery in normoxia. For example, during RSH-VHL, the average SpO_2 was 94.5%, higher than in a typical RSH study. Similarly, Fornasier-Santos et al. demonstrated significantly greater improvement in the number of sprints to exhaustion in RSH-VHL trained rugby players compared to the same training in normoxia (Fornasier-Santos, Millet, & Woorons, 2018).

Researchers also quantified repeated sprint ability as peak power and mean power scores on a sprint-based cycle ergometer test. For example, improvements in repeated sprint ability were significantly greater following RSH than after RSN training in studies conducted by Kasai et al. and Beard et al. (Beard et al., 2019; Kasai et al., 2015). Both studies quantified repeated sprint ability in terms of peak power and mean power scores on a repeated sprint test composed of short sprints (< 10 s) with less than 30 seconds of rest between sprints. The participants, FiO_2, and training protocol were different between studies. Kasai et al. trained female soccer players using cycling in 8 workouts over 4 weeks at a FiO_2 of 14.5% using two sets of 10 x 7 s sprints with 30 s rest between sprints and a long 10–20-minute recovery between sets. On the other hand, Beard et al. trained world-ranked male rugby players using double polling (upper body exercise) at a FiO_2 of 13.8% for 4 workouts over two weeks using three sets of (8 x 10 s) with 20 s between sprints and 2 min between sets. In contrast, based on similar assessments of RSA, several studies found no significant difference in performance following RSH and RSN training

(Gatterer et al., 2015; P. S. Goods, Dawson, Landers, Gore, & Peeling, 2015; Kasai et al., 2015). Notably, Kasi et al. trained male sprinters for five consecutive days, twice daily, at a FiO_2 of 14.5%, with no difference in improvement between RSH and RSN. Similarly, in a third study, they found no significant difference in improvement in repeated sprint ability after six consecutive days of RSH (FiO_2, 14.5%) versus RSN training (FiO_2, 20.9) (Kasai et al., 2019). Kasi et al. employed the fewest training days in any of the studies included in this review. Their minimal training period may have factored into the lack of significant differences in improvement between RSH and RSN training.

Impact of RSH Training Compared to The Equivalent RSN Training on Aerobic Capacity

Throughout the RSH literature, aerobic capacity was quantified in three primary ways, performance on an incremental VO_{2max} test, performance on a sport-specific VO_{2max} test, and performance on the Yo-Yo Intermittent Recovery Test. Changes in VO_{2max} did not differ significantly between RSH and RSN training for any of the studies reviewed here. However, in one study, tennis-specific aerobic capacity improved to a greater extent following RSH versus RSN training. In that study, aerobic capacity was measured during a tennis-specific test to exhaustion (TEST) (C. Brechbuhl et al., 2018; Cyril Brechbuhl, Girard, Millet, & Schmitt, 2016, 2017). Maximal oxygen consumption ($mL \cdot min^{-1} \cdot kg^{-1}$) was assessed during a ball stroking accuracy test to volitional exhaustion or when a judge determined failure (national-level tennis coach). The time to exhaustion was significantly greater following RSH vs. RSN training ($p < 0.01$). However, VO_{2max} did not improve significantly in either group. Due to the tennis-specific nature of the study, the generalizability of the results to other sports is limited. In addition, the subjective nature of the termination of the test complicates comparisons with other studies, including those that used the same TEST methodology.

Several studies assessed aerobic capacity using the Yo-Yo intermittent recovery test. The Yo-Yo test was designed based on the anaerobic and aerobic energy demands of sports such as soccer (Jens Bangsbo, Iaia, & Krustrup, 2008), rugby (Atkins, 2006), and basketball (Castagna, Impellizzeri, Rampinini, D'Ottavio, & Manzi, 2008) where repeated sprint ability is required for success. The test has two levels. The authors described level one as an assessment of endurance capacity (YYIR1). Level two assesses repeated maximal exercise performance when aerobic and anaerobic energy supply is taxed (YYIR2) (Jens Bangsbo et al., 2008; Krustrup et al., 2003; Krustrup et al., 2006). Both versions of the test involve maximal stimulation of the aerobic energy system. However, they differ in the degree of anaerobic involvement. Specifically, the YYIR2 reflects greater reliance on anaerobic processes. Higher muscle lactate levels, lower creatine phosphate levels and increased glycogen depletion after the YYIR2 compared to the YYIR1 test support this assumption (Krustrup et al., 2006).

Two studies included the YYIR1 test in their methodology. In the first, two groups of rugby players trained for four weeks (12 workouts) at a FiO_2 of 13% or ~ 20.9%. Galvin et al. found significantly greater distances ($p < 0.002$) covered in the YYIR1 test in the RSH (33 ± 12%) compared to the RSN-trained group (14 ± 10%). In contrast, Hamlin et al. also trained rugby players but concluded that RSH training did not improve performance on the YYIR1 beyond RSN training. Notably, their participants trained at a higher FiO_2 (14.5% vs. 13%) and performed half as many workouts (6 vs. 12) (Hamlin, Olsen, Marshall, Lizamore, & Elliot, 2017). In addition, cycling was used for repeated sprint training, while Galvin et al. used a running program, arguably a more rugby-specific training modality. All three factors may have caused the unclear finding in the Hamline et al. study. A third study investigated the effect of RSH training on YYIR2 performance. In that study, Gatterer et al. found significantly greater

improvement in YYIR2 performance in the RSH group ($p < 0.024$) following soccer-specific shuttle run training. They suggested that the improved YYIR2 scores indicated an enhanced ability to maintain speed throughout a game (Gatterer et al., 2015). Based on these limited findings, the Yo-Yo Intermittent recovery test may be a valuable standardized test to include in future RSH training studies, especially those focused on performance in team sports.

The Impact of RSH versus RSN Training on Sport-Specific Time Trail Performance

Coaches and athletes want to know if RSH training will help them achieve their primary goal: winning. Therefore, the most pertinent performance assessment would be placement or scoring at the end of the season championship. In individual sports, with single sprint events, performance assessment is relatively straightforward (time and place). Assessments, in the case of team sports performance, are more complex. However, coaches and athletes are reluctant to participate in research during championships. Linking a winning performance to RSH training is complicated by the interplay between individual and team factors. Likely, these challenges are why none of the RSH studies reviewed assessed real-world championship performances following repeated sprint training. Instead, researchers developed sport-specific time trials performed under simulated competitive conditions as a proxy for real-world competition.

Two studies used time trials to estimate the impact of RSH compared to RSN training on athletic performance. Swimming performance in 100 m and 400 m freestyle was evaluated in the only study that trained swimmers with an in-the-water RSH training program. Marta Camacho-Cardenosa et al. used a crossover design composed of eight RSH training sessions conducted over four weeks at a F_iO_2 of 13.7% or in normoxia at 20.9% (Camacho-Cardenosa, Camacho-Cardenosa, González-Custodio, Zapata, & Olcina, 2020). Hypoxia was created by pumping reduced oxygen concentration air through a waterproof facial mask worn while swimming.

Training consisted of five sets of 15 m all-out sprints with 20 seconds of passive recovery between repetitions and a 200 m easy swim between sets. Time trail performance did not improve following RSH or RSN training. Importantly, the athletes performed repeated sprint training at the end of a two-hour long daily workout (~ 5,000 m) when the athletes were fatigued. In that state, glucose and glycogen levels would be depleted. Depleted substrate levels may have limited the energy supplied through glycolysis during repeated sprint training. With less anaerobic energy supply, the participants may not have maintained a sufficient training intensity to evoke a beneficial training effect (Chromiakº & Mulvaney, 1990; I. Jacobs, Kaiser, & Tesch, 1981; Saltin, 1981). Moreover, the authors suggested that the low SpO_2 levels (70.1± 4.8%) during RSH training and interference from the waterproof mask with stroke mechanics may have contributed to the lack of improvement.

Galvin et al. used time trails to assess rugby player performance changes following RSH and RSN training. The authors suggested that faster sprinting resulted in improved performance in rugby games. They trained 30 male rugby players in hypoxia (13%) or normoxia (~21%) using 10 x 6 s sprints with 30 s rest between sprints for 12 workouts over 4 weeks. Speed was evaluated at 5, 10, and 20-meter distances. However, with one exception (5 m sprint time), performance did not improve following RSH or RSN training. The lack of improvement in single sprint performance suggests that the repeated sprint training protocol was ineffective. They theorized that their repeated sprint protocol improved oxidative rather than non-oxidative metabolism (Galvin et al., 2013).

Meta-Analysis: The Impact of RSH versus RSN Training On Athletic Performance

Brocherie et al. using a meta-analysis, assessed the evidence of RSH-induced improvement in athletic performance beyond the equivalent training in normoxia (Brocherie et

al., 2017). They consolidated the research findings through 2017, beginning with the first RSH study by Faiss et al. in 2013. The selection criteria for the analysis conformed to Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA). Only randomized controlled trials that assessed the impact of RSH training protocols on sea-level performance were included. In addition, single-blind, double-blind, placebo-controlled, and crossover-design studies were accepted. Study participants had to be well-conditioned, defined as more than 2 hours a week of training. To be included, the training program had to consist of maximal or supramaximal sprint training (< 30 seconds) with less than 60 seconds of rest between repetitions. Studies were excluded if they did not incorporate a normoxia training group, failed to exclude participants with prior exposure to hypoxia or lacked performance outcome measures. Based on the final sample of 202 (mean age 22.6 ± 6.1 years; 180 males, 20 females), the authors concluded that RSH versus RSN training significantly improved performance (mean times) in the repeated sprint tests (SMD = 0.46, 95% CI -0.02 to 0.93; $p = 0.05$), with a small to moderate effect size. How this improvement transfers to real-world athletic performance was unclear.

Table 2.4 Repeated Sprint and Athletic Performance Following RSH vs. RSN Training

Author/Year/Design	Participants	RST Protocol Sets(repetitions x sprints, rest between sets) type of rest, number of workouts, number of weeks	FiO_2 %	Training Mode	RSA Tests	Aerobic Tests	Results RSH vs. RSN
Faiss et al. 2013, P, SB (*n* = 40)	M moderately trained cyclists	3(5 x 10 s, 5 min), A, 8, 4w	14.6	cycling	10 s all-out sprint, bike 20 s recovery until volitional exhaustion, WAnT	3 m all-out bike test	Delayed fatigue during RSA (*p* < 0.001)
Galvin et al. 2013, P, SB, (*n* = 30)	M, academy rugby union and league players	1(10 x 6 s, 30 s), na, P, 12, 4w	13	running	10 x 20 s 30 s passive recovery	YYIR1	YYIR1 (*p* < 0.002)
Puype et al. 2013, P (*n* = 29)	M, 29, healthy	1(4 x 30 s, 4:30) na, A, 18, 6w	14.5	cycling	none	VO_{2peak}	NS
Gatterer et al. 2014 P, SB (*n* = 10)	M adolescent soccer	3(5 x 10 s 20 s, 5 min), 8, 5w	14.8	shuttle run	6 x 40 m shuttle run passive recovery	YYIR2	RSA NS YYIR2 Fatigue slope lower after RSH (*p* = 0.024)

Brocherie et al. 2015, P, DB, (*n* = 16)	M, youth football	5(4x 5 s 45 s, 5 min), 10, 5 w	14.3	treadmill and shuttle run	10 x 30 s running sprints 30 s passive recovery	VAMEVAL	NS
Kasai et al. 2015 P, SB, *(n* = 32)	F, college club lacrosse college lacrosse	2(10x7 30 s 10-20 min), 8, 4 w	14.5	cycling	10 x 7 s cycle sprints 30 s passive recovery	VO_{2max}	PP and MP ($p < 0.05$) VO_{2Max} NS
Goods et al. 2015, P, SB, (*n* = 19)	M, semi-elite AFL players	3(7 x 5s, 15-35 s, 3 min), A, 15, 5w	14.5	cycling	running 3 x 6 x 20 m sprints, 25 s recovery cycling 3 x 6 x 4 s, 25 s recovery	20 m shuttle run test	NS
Montero and Lundby 2017 CO, DB (*n* = 15)	M, moderately trained endurance athletes	4(5 x 10 s, 20 s, 5 min), A, 12 4w	13.8	cycling	10 s all-out sprints, 20 s passive recovery to volitional exhaustion	VO_{2max}	NS
Hamlin et al. 2017, P. SB, (*n* = 19)	M, 19 players	4(5 x 5 s, 25 s, 5 min), A, 6, 3 w	14.5	cycling	8 ~ 4-sec sprints on 16-sec rest	YYIR1	YYIR1 Unclear RSA possibly beneficial
Kasai et al. 2017, P, SB (*n* = 19)	M, sprinters	2 sessions per day of sprint training for 5 consecutive days	14.5	run/cycling	10-s maximal sprint, repeated sprint ability (5x6-s sprints), WAnT	VO_{2max}	NS
Kasai et al. 2017, P, SB (*n* = 18)	M, college sprinters	6 consecutive days	14.5	cycling	10 x 6 s maximal	VO_{2max}	NS

		1-4 x 15 s with 5m rest/sprints 2-3 30 s sprints with 10 m rest/sprints			sprints, 30 s rest 2 x 60 m run		
Galvin et al. 2017 SP, P (*n* = 42)	M, well-trained academy rugby	1-3 sets of 5 x 6 s with 30 s rest/sprints 10 x 20 m sprints 30 r rest/sprints	13	running	3 x 20 m max sprints 3 min/sprints RSA 10 x 20 m sprints with 30 rest/sprints	YYIR1	Single sprint, NS RSA, NS YYIR1 ($p < 0.001$)
Brechbuhl et al 2018, P, DB (*n* = 16 m, 4 f)	M, F, well-trained tennis players	4(5 x 20 s) 7, ?,?, 5 12 d	14.5	running	10 x 20-m on 20 s	Tennis specific VO_{2manx} $mL \cdot min^{-1} \cdot kg^{-1}$	RSA NS Tennis specific VO_{2max} time to exhaustion ($p < 0.01$)
Beard et al. 2019 P, DB (*n* = 36)	M, national team, rugby players	3(8 x 10 s), 20s, 2 min) P, 4, 2w	13.8	double-polling	double polling 6 x 10 s maximal sprints	None	PP ($p = 0.002$) MP ($p < 0.001$)
Marta Camacho-Cardenosa et al. 2020 CO, SB		3(5 x 15 m), 20 s, P, 8, 4 w	13.7	swimming	100 m swim 400 m swim	VO_{2max}	NS

Note, P = parallel study design, DB = double-blind study design; SB = single-blind study design; CO = crossover study design; FiO_2 = fraction of inspired oxygen; A =active rest; P = passive rest; F= females; M = males; m = meters, s = seconds, min= minutes; ? = not reported; RST = repeated sprint training; RSA = repeated sprint ability; RSH = repeated sprints in hypoxia; RSN = repeated sprints in normoxia; YYIR1 = Yo-Yo intermittent recovery test level one; YYIR2 = Yo-Yo intermittent recovery test level 2; VAMEVAL = maximal incremental running test, a modified version of the University of Montreal Track Test. *Adapted from Brocherie et al. 2017 plus individual studies.*

Section IV

Mechanism Underlying Adaptations to Hypoxic Training

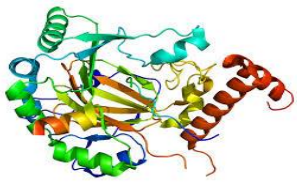
The common link between the hypoxic training approaches is the goal of inducing additional improvements in athletic performance by challenging oxygen homeostasis beyond the equivalent exercise in normoxia (Hoppeler & Vogt, 2001). The hypoxic dose, training protocols, performance goals, and the adaptations targeted by the training differ between approaches. Determining the mechanisms underlying the adaptations to each hypoxic training approach would enable scientists and coaches to design, implement and monitor training programs more effectively. Recently, oxygen-sensitive transcription factors in the hypoxia-inducible factor family (HIF) discovered in 1996 by Semenza (Kumar & Choi, 2015; G. L. Semenza, 1996), have emerged as the likely mediators of the molecular adaptations to hypoxic training (Faiss, Leger, et al., 2013; Serebrovs'ka et al., 2007; Vogt et al., 2001).

Specifically, within the HIF family, HIF-1α is the transcriptional activator and a master regulator for the expression of genes associated with adaptations to hypoxic training (Wiśniewska, Płoszczyca, & Czuba, 2020; Ziello, Jovin, & Huang, 2007). The HIF-1 pathway controls genes involved in erythropoiesis, new blood vessel development, and glycolysis (Kierans & Taylor, 2021; Nava et al., 2022; G. L. Semenza, 1996; Ziello et al., 2007). As previously mentioned, EPO is the main regulator of erythropoiesis, impacting the hematological response to training and likely a factor in the adaptations following LH training (Mounier et al., 2006). Following RSH training, the gene associated with Vascular endothelial growth factor (VEGF) is upregulated (Nava et al., 2022). VEGF stimulates new blood vessel growth (angiogenesis), which results in improved blood flow to exercising muscle, an important adaptation that may be relevant to both LH and LL training (Bloor, 2005; Wiśniewska et al.,

2020). In addition, following RSH training, genes associated with anaerobic glycolysis (PDK1) and pH regulation were upregulated and are thought to benefit repeated sprint performance (Faiss, Leger, et al., 2013; Nava et al., 2022). Concurrently after RSH training, the gene regulating mitochondrial biogenesis, peroxisome proliferator-activated receptor-gamma coactivator 1 alpha (PPARCG1A;PGC1A) was shown to be both upregulated (Faiss, Leger, et al., 2013) and downregulated (Brocherie et al., 2018). Importantly, Brocherie et al. 2018, performed RSH training coupled with normobaric hypoxic exposure (14 hr/day for 2 weeks), while Faiss et al. did not incorporate hypoxic exposure beyond RSH training. The difference in gene regulation may have been a function of the hypoxic dose rather than RSH training. However, investigations into the impact of hypoxic training compared to the equivalent training in normoxia on gene regulation are relatively recent. Continued research may improve our understanding of the role of the HIF-1 pathway in regulating the metabolic response to hypoxic training and lead to more effective methods of prescribing exercise (Nava et al., 2022).

Whole Body Adaptations
Improved anaerobic capacity
Increased repeated sprint ability
Increased number of sprints to exhaustion
Improved mean sprint time
Increased Peak Power
Increased Mean Power

Physiological Adaptations
Increased blood flow to muscle
Increased activity of PFK
Increased glycogen content
Increased PCr content
Improved pH management



Mechanism
HIF-1α Pathway
Increased angiogenesis
Enhanced glycolysis
Improved pH regulation

Figure 2. 2 Hypothesized RSH-induced additional improvement in performance, physiological adaptations, and the underlying mechanism supported by current research.

Section V

Summary and Conclusion

This review has critically evaluated the scientific literature regarding the hypothesis that training in hypoxia, compared to the equivalent training in normoxia, induced additional improvements in athletic performance. Two primary types of hypoxic training were reviewed. The LH training approaches utilized long exposure to hypoxia (weeks) to evoke a hematological response, enhance VO_{2max}, and potentially improve performance in aerobic or endurance events. On the other hand, the LL training approaches utilized acute exposure to hypoxia (minutes to hours), targeting improvements in anaerobic capacity and performance in single sprint events or team sports. Currently, no definitive conclusion has been reached concerning the additional

improvements in performance following hypoxic training compared to training in normoxia. However, the evidence suggests small to moderate additional performance improvements in middle-distance and distance events following LH training. Similarly, the evidence supports small to moderate gains in repeated sprint ability following RSH training.

Specifically, RSH training was defined as performing repeated sprints (30 s or less) in hypoxia on a 1:4 work-to-rest ratio. However, the optimal hypoxic dose has not been determined. There is a consensus that during RSH training, an effective range for FiO_2 is between 13% and 15.5%. Several studies found a small to moderate additional benefit on athletic performance following RSH training. However, a consensus concerning this conclusion has not been reached. Given the limited number of RSH training investigations, additional randomized, controlled studies with single or double-blinded conditions are needed to determine the benefits of RSH training on athletic performance (Brocherie et al., 2017). Continuing efforts to determine the optimum training program and hypoxic dose are important. Furthermore, agreeing on objective tests for quantifying participants' physical fitness and athletic status is necessary. In addition, methodologies that include standardized tests of repeated sprint ability will assist in comparing results and ultimately in answering the question of RSH-induced additional improvements in athletic performance beyond the equivalent training in normoxia.

Chapter III: The Reliability of the VASA Swimming Ergometer and the Validity of a Novel Laboratory-Based, Swim-Specific Anaerobic Capacity Test

Abstract

Introduction: Anaerobic capacity is a primary determinant of performance in many competitive swimming races. In the water, tests of anaerobic capacity are expensive and time-consuming. Sports scientists and coaches would benefit from a swim-specific, land-based method to assess and monitor anaerobic capacity in swimmers. Currently, a valid test and a reliable ergometer have not been identified. The VASA swimming ergometer measures power outputs during swim-specific training and is a potential option for conducting a swim-specific, laboratory-based modification of the Wingate upper body anaerobic capacity test, the WAnT-Swim**. Purpose:** This study aimed to assess the test-retest reliability of the WAnT-Swim and conduct a preliminary assessment of the validity of the test. **Hypothesis:** We hypothesized that the WAnT-Swim would reliably measure power outputs during a land-based, swimming-specific modification of the Wingate Anaerobic Capacity Test (WAnT) conducted on a swimming ergometer. Moreover, preliminary evidence would indicate that the WAnT-Swim is a valid modification of the WAnT warranting further research into the validity of the test. **Methods**: Ten competitive and fitness swimmers from the local college community completed four WAnT-Swim tests, two per day on two separate days conducted on the VASA swimming ergometer. The preliminary analysis of validity was conducted using peak heart rate and post-lactate level data from WAn-T swim tests performed by 12 competitive male college swimmers as part of another study in our laboratory. **Results:** Intra-class correlation coefficients (ICCs) calculated for Peak Power, Mean Power and Low Power were in the excellent range (0.98) with low variation (CV < 6%). A non-significant, one-sample t-test calculated by comparing lactate data from our laboratory with data from similar studies reported in the literature offered preliminary support for the validity of the WAnT-Swim. **Conclusion:** We conclude that the WAnT-Swim, administered using the VASA swimming ergometer, is a reliable test. Moreover, preliminary evidence provided initial support for the validity of the test. However, additional research is needed to confirm the validity of the WAnT-Swim.

Keywords: Anaerobic Capacity, Swimming Performance, Wingate Test

Introduction

Swimming is contested across all three Divisions (I, II, III) of the National Collegiate Athletic Association (NCAA) (NCAA, 2015). The four swimming strokes, freestyle, backstroke, breaststroke, and butterfly are performed as single races or combined as an individual medley or relay. In collegiate swimming, event distances vary from 50 to 1,650 yards (45.7 – 1508.8 m) and can last approximately 18 seconds to 18 minutes, with the majority of events completed in less than 2.5 minutes (NCAA, 2015). Due to the distance and duration of the events, swimmers typically rely on anaerobic metabolic pathways to generate and sustain most of the propulsive force needed to race (Hawley et al., 1992; Holmer, 1983; Mercier et al., 1993; Morouco et al., 2015). Additionally, the propulsive power produced by these athletes is generated primarily from the upper body (Hawley & Williams, 1991). Therefore, quantifying upper body fitness parameters is pertinent for monitoring training progress and talent identification (D. J. Smith, Norris, & Hogg, 2002).

Given that the majority of races in swimming take less than 2.5 minutes, anaerobic capacity is an important determinate of swimming performance (Campos et al., 2017; Demarie, Chirico, Gianfelici, & Vannozzi, 2019; Huub M Toussaint, 1992; Huub M. Toussaint & Hollander, 1994). Whole body anaerobic capacity is typically assessed in the laboratory using the Wingate Anaerobic Capacity Test (WAnT) (Bar-Or, 1987). The WAnT is an all-out, 30-second sprint conducted on a bicycle ergometer. However, sport-specific anaerobic capacity testing can be performed using any ergometer that permits the simulation of movements that occur in the sport (Bar-Or, 1987; Dotan & Bar-Or, 1983; Zupan et al., 2009). Modifications of the WAnT proved reliable and valid across ergometer types, athletes, and with clinical

populations (Bar-Or, 1987; P. L. Jacobs, Johnson, Somarriba, & Carter, 2005; P. M. Smith, Doherty, & Price, 2007; Tirosh, Rar-Or, & Rosenbaum, 1990). However, a feasible test for swimming anaerobic capacity is currently lacking, partially due to the unique swimming position (prone), the regulated respiratory rhythm, and water resistance encountered while racing (Aspenes & Karlsen, 2012).

Currently, there is no generally accepted laboratory-based, swim-specific anaerobic capacity test. Although several field-based tests exist (Baldari et al., 2013; Kjendlie & Thorsvald, 2006), these tests are costly, and the aquatic environment presents numerous challenges (Aebi et al., 2019; Hawley & Williams, 1991). Therefore, a laboratory-based, swim-specific anaerobic capacity test is warranted (I. L. Swaine & Winter, 1999). The VASA swimming ergometer (VASA Inc.) is a swimming-specific ergometer that measures power output (W) and can be used for training and performance testing. Individuals can lie prone or supine on the ergometer and complete a swimming-specific pull against resistance with power measured second-by-second during each pull. The VASA swimming ergometer reliably measured power outputs generated during a pulling motion akin to paddling during surfing (Loveless & Minahan, 2010), but the test-retest reliability of power outputs measured during simulated swimming is unknown. Given that the majority of races in swimming take less than 2.5 minutes, anaerobic capacity is an important determinate of swimming performance (Campos et al., 2017; Demarie et al., 2019; Huub M Toussaint, 1992; Huub M. Toussaint & Hollander, 1994). Whole body anaerobic capacity is typically assessed in the laboratory using the Wingate Anaerobic Capacity Test (WAnT) (Bar-Or, 1987). The WAnT is an all-out, 30-second sprint conducted on a bicycle ergometer. However, sport-specific anaerobic capacity testing can be performed using any ergometer that permits the simulation of movements that occur in the sport (Bar-Or, 1987; Dotan

& Bar-Or, 1983; Zupan et al., 2009). For example, an arm-based modification of the WAnT utilizes an arm crank ergometer to measure upper body anaerobic capacity (Dotan & Bar-Or, 1983; Julio et al., 2019). Modifications of the WAnT proved reliable and valid across ergometer types, athletes, and with clinical populations (Bar-Or, 1987; P. L. Jacobs et al., 2005; Paul M. Smith, Chapman, Hazlehurst, & Goss-Sampson, 2008; Tirosh et al., 1990). However, a feasible test for swimming anaerobic capacity is currently lacking, partially due to the unique swimming position (prone), the regulated respiratory rhythm, and water resistance encountered while racing (Aspenes & Karlsen, 2012).

Therefore, this study aimed to 1) determine the test-retest reliability of the WAnT-Swim by measuring second-by-second power outputs measured with the VASA swimming ergometer and 2) perform a preliminary evaluation of the validity of the test.

Methods

Participants

A group of 10 swimmers was recruited to participate in this study. Swimmers were either current Division II NCAA athletes (n = 6) or recreational fitness swimmers (n = 4). Including competitive and recreational swimmers provided a range of power outputs to evaluate test reliability and validity while also ensuring participants had the requisite skill needed to complete the task (i.e., minimize learning effects). All participants were recruited from the local college community. Exclusion criteria included shoulder surgery or injury within the last 12 months, known cardiovascular disease, a sedentary lifestyle (less than three 30-minute workouts per week), and medication that affects heart rate response to exercise. All participants provided

written informed consent prior to study enrollment. The Institutional Review Boards of Syracuse University (IRB# 16-121) and LeMoyne College (IRB # 2016-298) approved this study.

Study design and procedure

All testing was conducted in the Altitude Simulation Laboratory at Syracuse University. Participants visited the laboratory at approximately the same time of day on two separate visits. Study visits were separated by at least two but no more than seven days. Each study visit consisted of two separate WAnT-Swim tests (Tests A and B on Visit 1; Tests C and D on Visit 2) for a total of four WAnT-Swim tests performed throughout study enrollment. Prior to study visits, participants were instructed to refrain from caffeine, alcohol, and strenuous exercise for 24 hours. Before the first test (Test A), participants watched an instructional video demonstrating the correct use of the swimming ergometer. After the video, participants were allowed to familiarize themselves with the ergometer until they felt comfortable with their pulling motion and stroke rate. Heart rate was recorded continuously throughout the WAnT-Swim test using a heart rate monitor (Polar H7: Polar Inc, Kempele, Finland) and stored with the Polar Beat Multi-Sport Fitness Tracker application. Following a 5-min rest period to establish resting heart rate, participants warmed up on the ergometer for 5-min prior to commencing the 30-sec WAnT-Swim test. Immediately following the WAnT-Swim, participants reported a rating of perceived exertion (RPE; Borg 6-20 scale) and then completed a 5-min cool-down (Gunnar Borg, 1970). This process was repeated after 15 min of passive rest. Peak lactate data measured from another study in our laboratory was used for the preliminary assessment of test validity. In that study, peak lactate levels from 12 male competitive swimmers were measured six minutes post-WAnt-Swim using a finger stick blood sample and analyzed with a Nova Biomedical Lactate Plus Meter (Nova Biomedical, Waltham MA).

Instrument- the swimming ergometer

The design of the VASA swimming ergometer utilizes an approximately 8-foot-long monorail, which attaches to the front of the ergometer. A flat padded bench equipped with rollers slides freely on the rail. This configuration permits participants to lie in a supine or prone position and fully extend their arms and legs as they would when swimming. Paddles for each arm are attached to separate drive chords that feed into the ergometer. Resistance on the VASA swimming ergometer is controlled by airflow and adjusted using a damper door with seven settings. Software within the power meter calculates power by sampling the force and velocity of the pull "many times per second" (Loveless & Minahan, 2010; VASA, 2016).

WAnT-Swim Test

Participants were instructed to find a comfortable prone position on the swimming ergometer, extend their arms forward and grasp the hand paddles. Against minimal resistance, they executed a double arm freestyle pull from this position until their hands were at thigh level. The return to the starting position consisted of reversing the pull pattern (no resistance) with the hands kept below the ergometer monorail. Using this pulling motion, participants completed a five-minute warmup with resistance on the flywheel set at three per the manufacturer's recommendations (VASA, 2016). A 10-sec lead-in phase was initiated toward the end of the 5-min warmup, in which participants gradually increased their stroke rate to a self-determined optimum rate. The WAnT-Swim consisted of 30-sec all-out double-arm pulling. Throughout the entirety of the test, participants were verbally encouraged to pull as hard as possible against the resistance. Immediately following the sprint phase of the test, participants were asked to rate the intensity of the test and then were allowed to cool down for 5 min by pulling slowly.

Calculation of WAnT-Swim power scores

The WAnT-Swim data was collected using VASA's ANT Power Meter (VASA Inc, Essex Junction, VT). The meter was linked to a Garmin USB ANT Stick for Garmin Fitness Devices (Garmin International, Olathe, KS) and the TrainerRoad application (TrainerRoad, Reno, NV). The Garmin USB ANT Stick transmitted data wirelessly from the VASA ANT Power Meter to TrainerRoad. The system measured, displayed, and stored test data, including the exercise duration, distance (meters), average watts, stroke rate, and power in watts per second during the test. In addition, detailed documentation of second-by-second measurements was retrieved through TrainerRoad and exported to Microsoft Excel datasheets for computation and evaluation, as described below.

For each WAnT-Swim, Peak Power (PP), Mean Power (MP), Low Power (LP), and Fatigue Index (FI) (Bar-Or, 1987) were calculated. The average of the five highest outputs was used to determine PP, and the average of the lowest five power outputs was used to determine LP, thereby minimizing the effect of a single aberrant power output measure (Wilmore J., 1994). MP was calculated as the average of all power outputs over the full 30-sec test. FI was calculated as [(PP – LP)/PP] x 100.

Statistical analysis

A priori sample size of ten was estimated using the Intraclass Correlation (ICC) Sample Size Package of *R* with a moderate ICC of 0.70 (Team, 2013). All data collected were analyzed with SPSS version 28 (IBM, Armonk, NY). Data were assessed for normality using the Shapiro-Wilkes test. Non-normally distributed data were $\log^{10}$-transformed. Visual inspection of boxplots was used to determine outliers, defined as values greater than 1.5 box lengths from the edge of

the box (Williamson, Parker, & Kendrick, 1989). Where outliers were identified, a Friedman test was performed (Conover, 1999). The reliability of the VASA swimming ergometer was assessed in several ways. First, all power outputs (PP, MP, LP, FI) from each test (A-D) were compared using a one-way ANOVA with repeated measures. Second, test-retest reliability of power outputs, stroke rate, heart rate, and RPE were assessed through a two-way random effects (consistency) analysis of variance (ANOVA) model (Koo & Li). ICC scores were used to indicate moderate (ICC = 0.50 to 0.75), good (ICC = 0.75 to 0.90), or excellent (ICC > 0.90) test-retest reliability (Atkinson & Nevill, 1998; Koo & Li, 2016). The coefficient of variation (CV = standard deviation/mean, expressed as a percent) (Bruton, Conway, & Holgate, 2000) was calculated for all variables across the four separate tests (Tests A through D).

While the primary purpose of this study was to evaluate the reliability of the VASA swimming ergometer, a preliminary assessment of the criterion validity of the WAnT-Swim was also conducted. A one-sample t-test was used to compare our data with the results reported in the literature. This approach was limited and was used solely to estimate the similarity between means. The mean peak heart rate and lactate levels from our data were compared with "reference" data from WAnT upper body tests reported in the scientific literature to conduct the analysis.

Results

Participant descriptive characteristics are displayed in Table 3.1. Overall, a majority of participants were men from a Division II team. Peak heart rate was non-normally distributed and was $\log^{10}$-transformed to better align with normality assumptions for analysis. Outliers were noted for LP, peak heart rate, and stroke rate; however, according to Friedman test results, the

outliers did not statistically influence the means and, therefore, were included in the analysis ($\chi^2 \geq 3.36$; $p \geq 0.09$).

Table 3.1 Participant Descriptive Characteristics

Participant descriptive characteristics (n = 10).	
Sex (n men/women)	7/3
Swimming level (n DII, rec.)	6, 4
Age (yrs)	21 ± 2
Height (cm)	177.0 ± 9.8
Weight (kg)	75.1 ± 13.2
Body Mass Index ($kg \cdot m^{-2}$)	23.8 ± 2.4

Note. Data are n or mean ± SD. DII = Division II NCAA athlete; rec = recreational swimmer.

Reliability

Table 3.2 summarizes WAnT-Swim power outputs, stroke rates, peak heart rate, and RPE with CV and ICC across study tests. One-way repeated measures ANOVA showed no significant difference between Tests A-D for measures of PP, MP, LP, Stroke Rate, Peak Heart Rate and RPE. Post hoc analysis with a Bonferroni adjustment for all the dependent variables (Maxwell, Delaney, & Kelley, 2017) demonstrated lower PP, MP, and LP for Test A compared to Tests B, C, and D, however, the differences did not reach significance. Generally, test-retest reliability for PP, MP, LP and stroke rate was in the excellent range (ICC > 0.90). In contrast, FI, peak heart rate, and RPE were all considered moderate for reliability based on ICC (ICC > 0.50). Regardless, low variation based on CV was observed for all variables.

Table 3.2 WAnT-Swim, test-retest reliability

	Peak Power (watts)	Mean Power (watts)	Low Power (watts)	Fatigue Index (%)	Stroke Rate (per minute)	Peak Heart Rate (bpm)	RPE
Day 1							
Test A	182 ± 60	157 ± 55	137 ± 51	25.3 ± 10.8	54 ± 7	154 ± 6	12.3 ± 2.1
Test B	197 ± 66	170 ± 58	149 ± 50	24.2 ± 8.6	56 ± 9	156 ± 5	12.8 ± 1.8
Day 2							
Test C	197 ± 56	169 ± 52	150 ± 51	24.4 ± 8.0	52 ± 9	149 ± 10	14.0 ± 1.3
Test D	196 ± 57	168 ± 52	148 ± 48	25.2 ± 8.2	54 ± 8	154 ± 10	14.4 ± 2.3
CV (%)	3.8 ± 7.3	5.3 ± 9.0	4.1 ± 6.1	3.9 ± 1.0	3.0 ± 1.6	1.9 ± 3.0	7.2 ± 0.9
ICC	0.98	0.98	0.98	0.65	0.96	0.70	0.55

Note. Test A = first test on testing day one; Test B = second test on testing day one; Test C = first test on testing day two; Test D = second test on testing day two; CV = correlation coefficient; ICC = intraclass correlation coefficient

Validity

Table 3.3 summarizes the data from studies that conducted the WAnT upper body test. Testing equipment used in the studies included arm crank ergometers, modified cycle ergometers, and the biokinetic swim bench. Table 3.4 presents studies that tested competitive swimmers and includes data from this study and some unpublished data from our laboratory. When the participants were tested in the prone position, PP on the swim bench ranged from 164 ± 12 W (female) to 304 ± 22 W (males). Comparably on the swimming ergometer, PP ranged from 117 ± 47 W (female) to 239 ± 46 W (males). Only one study using the swim bench measured MP while the participants were prone (225 ± 31 W males). In comparison, MP on the swimming ergometer ranged from 97 ± 33 W (females) to 201 ± 39 W (males). In addition, only

our study measured LP, FI, and RPE while the participants were prone. Peak heart rate while exercising in the prone position ranged from 153 ± 10 bpm (male, swim ergometer) to 174 bpm (males, swim bench, no standard deviation reported). FI for the WAnT-Swim ranged from 23 ± 8 to 30 ± 8.

Criterion validity of the WAnT-Swim was estimated by comparing the means for peak heart and lactate levels from WAnT upper body tests reported in the literature with mean peak heart rate data from this study and mean peak lactate levels from our unpublished study. Based on a one-sample t-test, the peak heart rates in this study were significantly lower than those measured in similar published studies ($p < 0.001$, 2-tail). On the other hand, the post-WAnT-Swim peak lactates measured in our laboratory were not significantly different than those measured by Guglielmo and Denadai (Guglielmo & Denadai, 2000). Conversely, our peak lactates were significantly lower (7.3 $mmol \cdot L^{-1}$ vs. 10.1 $mmol \cdot L^{-1}$, $p < 0.001$) than those measured by Ogonowska (Ogonowska, Hübner-Wozniak, Kosmol, & Gromisz, 2009).

Table 3.3 Upper Body WAnT Test Results

Author	Sample	Test/Ergometer	Peak Heart Rate (beats/min)	RPE	Lactate (mmol·L^{-1})	Peak Power (W)	Mean Power (W)	Low Power (W)	FI (%)
Sharpe et al. (1982)	(*n* = 18) m (*n* = 22) f swimmers	Power test swim bench	nm	nm	nm	286 ± 14 m 164 ± 12 f	nm	nm	nm
Kounalakis et al. (2008)	(*n* = 21) me (*n* = 9) m con handball	WAnT arm crank	158 ± 13 170 ± 20	nm	nm	655 ± 25 571 ± 12	nm	nm	nm
Talbot et al. (2014)	(*n* = 11) m (*n* = 9) f non-specific	WAnT arm crank	166 m 162 f	nm	nm	586 m 246 f	412 m 186 f	nm	nm
Swaine et al. (1997)	(*n* = 12) m swimmers	Swim Bench	170 ± 3	nm	nm	nm	nm	nm	nm
Swaine (2000)	(*n* = 22) m swimmers	Swim Bench	174 sb 161 ac	nm	nm	304 ± 22	225 ± 31	nm	nm
Guglielmo and Denadai (2000)	(*n* = 9) m swimmers/ triathletes	WAnT arm ergometer	nm	nm	7.8 ± 1.2	527 ± 79 7.6 ± 0.95 W·kg^{-1}	421 ± 63 6.19 ± 0.70 W·kg^{-1}	nm	42.9 ± 6.4
Hawley (1992)	(*n* = 12) m (*n* = 10) f swimmers	WAnT\ arm crank	nm	nm		4.89 W·kg^{-1} m 3.65 W·kg^{-1} f	3.74 W·kg^{-1} m 2.82 W·kg^{-1} f	nm	nm
Ogita (1995)	(*n* = 8) m swimmers	VO_{2max} swim bench	162 ± 10	nm	8.5 ± 2.2	nm	nm	nm	nm
Ogonowska et al. (2009)	(*n* = 9) m (*n* = 6) f	WAnT arm crank	162 ± 7 m	nm	10.1 ± 0.8 m	7.96 ± 1.1 m 5.14 ± 0.4 f	5.97 ± 0.8 m 4.27 ± 0.3 f	nm	nm

			172 ± 10 f		6.7 ± 2.0 f				
Unnithan et al. (2004)	(*n* = 15) f swimmers	WAnT arm crank	nm	nm	nm	298 ± 64	176 ± 34	131 ± 28	55 ± 9
Garcia-Pallares et al. (2011)	(*n* = 18) me (*n* = 15) ma wrestlers	WAnT/arm crank standing	nm	nm	nm	630 ± 86 me 429 ± 146 ma	nm	nm	nm
Price et al. (2014)	(*n* = 8) m mod fit	WAnT arm crank	181 ± 10	17.9 ± 1	nm	419 ± 139	322 ± 50	222 ± 26	47 ± 16
Crawley et al. (2016)	(*n* = 61) m (*n* =7) f police	WAnT arm crank	nm	nm	nm	2.2 ± 0.07 $W \cdot kg^{-1}$	nm	nm	nm
Kachaunov & Petrov (2020)	(*n* = 37) m (*n* = 15) f	WAnT arm crank				538 ± 138 m 282 ± 75 f	378 ± 69 m 209 ± 48 f	249 ± 47 m 148 ± 33 f	
Holohan et al. (2022)	(*n* = 7) m (*n* = 3) f swimmers	WAnT-Swim swim ergometer	153 ± 10 m 155 ± 10 f	14 ± 3 m 12 ± 3 f	nm	220 ± 44 m 117 ± 47 f 2.67 ± 0.09 $W \cdot kg^{-1}$ m 2.23 ± 0.15 $W \cdot kg^{-1}$ f	191 ± 39 m 97 ± 33 f 2.31 ± 0.07 $W \cdot kg^{-1}$ m 1.80 ± 0.14 $W \cdot kg^{-1}$ f	169 ± 36 m 81 ± 32 f	23 ± 8 m 30 ± 8 f
Holohan et al.[a] (2022)	(*n* = 12) m swimmers	WAnT-Swim swim ergometer	160 ± 12	nm	7.3 ± 1.4	263 ± 46	219 ± 40	187 ± 40	29 ± 7

Note. WAnT/arm crank = WAnT using an arm crank; Swim Bench = power test using the swim bench; WAnT-Swim = swim-specific modification of the Wingate test; m = male; f = female; VO_{2max} = maximal oxygen consumption; RPE = rating of perceived exertion; me = male elite; ma = male amateur; con = control; nm = not measured; [a] = unpublished data.

Table 3.4 WAnT/Arm Crank and Swim Bench Test Results in Swimmers

Author	Sample (*n*)	Peak Heart Rate (bpm)	Peak Lactate ($mmol \cdot L^{-1}$)	Peak Power (W)	Mean Power (W)	Low Power (W)	FI
WAnT/Arm Crank							
Talbot et al. 2014	(*n* = 11) m (*n* = 9) f	166 m 162 f	nm	586 m 246 f	412 m 186 f	nm	nm
Ogonowska et al. 2009	(*n* = 9) m (*n* = 6) f	162 ± 7 m 172 ± 10 f	10.1 ± 0.8 m 6.7 ± 2.0 f	nm	nm	nm	nm
Unnithan et al. 2004	(*n* = 15) f	nm	nm	298 ± 64 f	176 ± 34 f	131 ± 28 f	55 ± 9 f
Swim Bench							
Guglielmo and Denadai 2000	(*n* = 9) m	nm	7.8 ± 1.2	527 ± 79 m	421 ± 63 m	nm	42.9 ± 6.4 m
Swaine et al. 1997	(*n* = 12) m	170 ± 3 m	nm	nm	nm	nm	nm
Swaine 2000	(*n* = 22) m	174 m	nm	304 ± 22 m	225 ± 31 m	nm	nm
WAnT-Swim/Swim Ergometer							
Holohan et al. 2022	(*n* = 7) m (*n* = 3) f	153 ± 10 m 155 ± 10 f	nm	220 ± 44 m 117 ± 47 f	191 ± 39 m 97 ± 33 f	169 ± 36 m 81 ± 32 f	23 ± 8 m 30 ± 8 f
Holohan et al.[a] 2022	(*n* = 12)	160 ± 12 m	7.3 ± 1.4	263 ± 46	219 ± 40	187 ± 40	29 ± 7

Note. WAnT/arm crank = WAnT using an arm crank; Swim bench = power test using the swim bench; WAnT-Swim = swim-specific modification of the Wingate test; m = male; f = female; W = watts; PP = peak power, MP = mean power; LP = low power; FI = fatigue index; nm = not measured; [a] = unpublished data.

Discussion

This study assessed the test-retest reliability of the VASA swimming ergometer to measure power outputs during a swim-specific modification of the WAnT. Additionally, a preliminary assessment of the validity of the modification, the WAnT-Swim, was conducted. The main findings of this study were that the VASA swimming ergometer reliably measured all WAnT-Swim power outputs with low variation (CV) and excellent test-retest reliability (ICC). In addition, the similarity between the peak lactate levels discussed in this study with those reported in the scientific literature following upper body anaerobic capacity testing offers preliminary support for the criterion validity of the WAnT-Swim.

Reliability

A variety of arm crank ergometers have been deemed reliable machines for measuring both submaximal and maximal upper body fitness capacity during the WAnT (Bulthuis, Drossaers-Bakker, Oosterveld, van der Palen, & van de Laar, 2010; Flueck, Lienert, Schaufelberger, & Perret, 2015; Patrick L Jacobs, Mahoney, & Johnson, 2003; Mitropoulos, Gumber, Crank, & Klonizakis, 2017; Shaw et al., 1974). Our study assessed the reliability of a novel swimming ergometer to measure swim-specific power outputs during the WAnt-Swim, a modification of the WAnT. CV was less than 7.5% for all variables, a threshold generally considered acceptable in sport science research (Stokes, 1985). As absolute power outputs decreased from PP to LP, CV remained low with little change in CV between WAnT-Swim power scores (3.8 ± 7.3% to 5.3 ± 9.0%, respectively). In addition, the ICC values for power outputs were excellent (0.98), consistent with previously reported ICC values for bicycle ergometers and arm crank Wingate tests (Bar-Or, 1987). ICC for the FI was also in the moderate range (0.65) and the CV low (3.9 ± 1.0%). FI is often unreliable and not reported (Vandewalle,

Peres, & Monod, 1987). However, the moderate ICC and low CV noted in this study suggest the WAnT-Swim provides a moderately reliable measure of FI. On the other hand, RPE was somewhat reliable (ICC = 0.55, CV = 7.2 ± 0.9%) and should not be used as the sole measure of exercise intensity during the WAnT-Swim.

There are unique aspects to the WAnT-Swim relative to the upper body WAnT that could impact the reliability of data measured on the swim ergometer. During an arm crank WAnT, subjects sit or stand and typically crank a modified bicycle ergometer. The load setting on the flywheel of the cycle ergometer is determined based on the individual's body weight, sex, age, and training status (Bar-Or, 1987; Forbes, Kennedy, Boule, & Bell, 2014). Loads are applied on the bicycle ergometer through mechanical or electromechanical braking (Astorino & Cottrell, 2012). Load on the swimming ergometer cannot be applied in the same manner. Resistance on the swimming ergometer's flywheel is adjusted by opening or closing a damper door which modifies the airflow and increases or decreases the resistance. Power output measured by the ergometer is the value of the force of each pull times the velocity of the pull. The VASA ergometer samples the force and velocity "multiple times per second" (VASA, 2016). Despite the various ways in which braking forces can be applied during a WAnT, two studies found that the type of braking force does not impact the reliability of the WAnT (Jaafar et al., 2014; Vandewalle, Peres, Heller, & Monod, 1985). Therefore, the braking method used to apply force on the swimming ergometer should not impact reliability.

The selection of stroke rate when testing with the swimming ergometer was another potential source of measurement error. The stroke rate for the WAnT-Swim is self-selected. Therefore, the main question concerns the impact of self-selected vs. a predetermined or imposed stroke rate on reliability. Initial evidence indicated that an imposed crank rate of 60-80

revolutions per minute was optimal for WAnT testing (Paul M Smith, McCrindle, Doherty, Price, & Jones, 2006; P M Smith, Price, & Doherty, 2001). However, other studies contradicted those assertions concluding that there was no difference in upper body fitness test results between an imposed and self-selected crank rate (G Marais, Dupont, Garcin, Vanvelcenaher, & Pelayo, 2001; Weissland, Marais, Robin, Vanvelcenaher, & Pelayo, 1999). To further investigate the question, Smith et al. (P. M. Smith et al., 2007) assessed the impact of a self-selected crank rate compared to an imposed rate on several performance metrics. They reported a wide interindividual variation in the self-selected rate; however, the differences did not influence test outcomes. Furthermore, PP, time to exhaustion, and peak oxygen consumption were not significantly different between self-selected and imposed crank rates. Therefore, they concluded that either crank rate procedure was acceptable but cautioned against mixing imposed and self-selected rate methods within the same study (Paul M Smith et al., 2006). In addition, Marais et al. found no difference in power output between an imposed and self-selected crank rate (G. Marais, Dupont, Vanvelcenaher, Clarys, & Pelayo, 2004). Finally, in a swimming-specific study, Swaine et al. demonstrated that a self-selected stroke rate resulted in the highest power outputs on a swim bench (I. Swaine & Reilly, 1983). During this study, CV for self-selected stroke rate was low (3.0 ± 1.6), and ICC was excellent (0.96), indicating reliability in self-selected stroke rates using the VASA ergometer. Therefore, it is likely that the reliability of the power outputs measured using the swimming ergometer is not impacted by a self-selected stroke rate.

Validity

While not the primary purpose of this study, a preliminary assessment of the criterion validity of the WAnT-Swim as a modification of the WAnT was conducted. For the WAnT-Swim to be considered a valid modification of the WAnT, a favorable comparison with a gold

standard is required (Bar-Or, 1987). The standard is the Wingate upper body anaerobic capacity test in this case. However, determining the validity of the WAnT-Swim is not straightforward. Complications arise due to between-study differences in the type of ergometer used, body position during all-out exercise, sample size, and the characteristics of the participants.

One possible approach to estimating the validity of the WAnT-Swim is to compare power outputs between the WAnT upper body test and WAnT-Swim. However, power output varies with the type of ergometer used for the test, rendering comparisons difficult. For example, Micklewright et al. compared electromagnetically and mechanically braked bicycle ergometers and found significant differences between devices for LP ($r = 0.54$, $p < 0.05$) and MP ($r = 0.60$, $p < 0.05$). (Micklewright, Alkhatib, & Beneke, 2006). When dissimilar types of ergometers were used, the differences in power outputs increased. For example, the data presented in Table 3.3 shows the wide range in PP between the WAnT arm crank (655 ± 25 W) and swim ergometer (220 ± 44 W) tests. Therefore, comparisons of power outputs obtained from arm crank, swim bench, and swim ergometer tests are not useful to evaluate the validity of the WAnT-Swim.

RPE is a psychophysical method of assessing the perception of effort during exercise (Scherr et al., 2013). The scale has been used for over 40 years and is considered reliable and valid (Dunbar et al., 1992). Therefore, a comparison of RPE between the WAnT-Swim and WAnT upper body test may be useful in evaluating the criterion validity of the WAnT-Swim. As a measure of intensity, RPE correlates with physiological measures often associated with exercise intensity, such as heart rate and lactate levels (G Borg, 1982; M. J. Chen, Fan, & Moe, 2002). However, RPE is not normally recorded during short maximal sprinting (Doherty, Smith, Hughes, & Collins, 2001). As a result, only one of the studies summarized in Table 3.3 reported RPE after a WAnT upper body test. In that study, Price et al. reported RPE values of 17.9 ± 1

following an arm crank WAnT (Price et al., 2014). In our study, RPE values following the WAnT-Swim were lower (14 ±3 in men and 12 ±3 in women). However, fitness level impacts RPE scores, with less fit participants giving higher ratings to the same relative intensity exercise (M. J. Chen et al., 2002). Our participants were younger, included competitive and recreational swimmers, and were likely more fit than the moderately fit participants in the Price et al. study, which may explain the lower RPE. Therefore, comparing RPE scores from the two studies is not highly informative in determining the validity of the WAnT-Swim.

A more accurate method to estimate criterion validity is to compare physiological data between studies. For example, peak heart rate during exercise testing is often used as a proxy for intensity. Table 3.3 shows peak heart rates during the WAnT upper body tests ranging from 162 bpm to 181 bpm compared to 153 bpm to 160 bpm during the WAnT-Swim. A one-sample t-test was used to compare our data with the mean data reported for each of the relevant studies in Table 3.3. Due to the lack of variability information related to the mean, the test results can only be used to estimate the similarity between means. Peak heart rates from the studies reported in the literature were significantly higher than those measured during the WAnT-Swim, suggesting that the intensity of the tests was dissimilar. Therefore, peak heart rate does not support the criterion validity of the WAnT-Swim.

Being prone during the WAnT-Swim may partially explain the lower peak heart rates during the test compared to seated exercise during the arm crank WAnT. However, during prone stroke-specific exercise to voluntary exhaustion on a swim bench, the peak heart rates were closer to rates during WAnT arm cranking tests (174 bpm to 170 bpm, respectively). Like the swimming ergometer, the swim bench permits swim-specific single and double-arm pulling. Resistance on the swim bench has been described as pseudo-accommodating (Sharp et al., 1982;

I. L. Swaine & Zanker, 1996). In addition, on the swim bench, the pulling speed is predetermined and fixed, while the pulling speed is not restricted on the swimming ergometer. These factors may explain the higher heart rates on the swim bench. However, additional research is required to determine the reason for the lower peak heart rates measured during the WAnT-Swim.

Comparison with a second physiological variable, post-test peak lactate levels, which is used as a proxy for exercise intensity and to estimate the contribution of anaerobic metabolism, may be a viable approach to determining the criterion validity of the WAnT-Swim. One study by Guglielmo and Denadai et al. reported lactate levels of 7.8 ± 1.2 mmol·L^{-1} in swimmers following an arm cranking WAnT (Guglielmo & Denadai, 2000). In addition, Ogonowska et al. measured lactate concentrations of (10.1 ± 0.8 mmol·L^{-1}) in male swimmers after an upper body WAnT using an arm crank (Ogonowska et al., 2009). For comparison, we used unpublished data from our laboratory. In that study, peak lactates (7.3 ± 1.4 mmol·L^{-1}) were measured 6-min post-WAnT-Swim in a cohort of 12 competitive male college swimmers.

Exercising while prone (WAnT-Swim) compared to seated (WAnT) may account for the lower peak heart rates measured during the WAnT-Swim (Lazar, Khanna, Chesler, & Salciccioli, 2013). However, peak heart rates during prone exercise to voluntary exhaustion on a swim bench were similar to rates measured while performing a WAnT arm cranking test (174 bpm to 170 bpm, respectively) but significantly higher than during the swimming ergometer test (WAnT-Swim). Therefore, factors other than body position underly the significantly higher peak heart rates during exercise on the swim bench.

A portion of the variation may be due to differences in how the machines create resistance and regulate pulling velocity. For example, resistance on the swim bench was described as pseudo-accommodating (Sharp et al., 1982; I. L. Swaine & Zanker, 1996). In

contrast, resistance on the swimming ergometer varies with pulling velocity (similar to swimming). Furthermore, pulling velocity is predetermined and fixed (imposed) on the swim bench while self-selected and variable on the swimming ergometer. These factors may account for the differences in peak heart rates. However, there is limited data. As a result, empirically determining the reason for the different peak heart rates during testing on the swim bench and the swimming ergometer isn't feasible without additional research.

Between test comparison with a second physiological variable, post-test peak lactate levels, which are used as a proxy for exercise intensity and to estimate the contribution of anaerobic metabolism, may be a viable approach to determining the criterion validity of the WAnT-Swim. One study by Guglielmo and Denadai et al. reported lactate levels of 7.8 ± 1.2 $mmol \cdot L^{-1}$ in male swimmers following an arm cranking WAnT (Guglielmo & Denadai, 2000). In addition, Ogonowska et al. measured lactate concentrations of 10.1 ± 0.8 $mmol \cdot L^{-1}$ in male swimmers after an arm crank upper body WAnT (Ogonowska et al., 2009). For comparison, we used unpublished data from our laboratory. In that study, peak lactates (7.3 ± 1.4 $mmol \cdot L^{-1}$) were measured at 6-min post-WAnT-Swim in a cohort of 12 competitive male college swimmers.

The difference between WAnT upper body peak lactate levels measured by Guglielmo and Denadai (Guglielmo & Denadai, 2000) and peak lactates post-WAnT-Swim did not reach the significance level, supporting the validity of the WAnT-Swim. However, peak lactates after the WAnT-Swim were significantly lower ($p < 0.001$) than those measured by Ogonowska et al. The higher peak lactates measured in the Ognowska study may be related to their methodology. Lactate levels were measured at four-time points post-test (3, 5, 7 and 9 minutes). The authors stated that lactate levels continued to increase until the ninth minute (Ogonowska et al., 2009). Contrastingly, in this study, WAnT-Swim peak lactates were measured at a single point, 6

minutes post-test. Our lactate values may have shown a similar trend and been higher at 9 minutes post-test. In addition, differences in posture (sitting vs. prone) may account for some of the variation in lactate levels between studies (Sawka, 1986). Given the limited data available, this study offers initial support for the criterion validity of the WAnT-Swim based on comparison with peak lactates following the upper body WAnT.

Limitations

According to the engineer who designed the VASA, the ergometer consists of load cells certified by the supplier with a certain accuracy (+/- 0.5%) and linearity (+/- 0.1%). Velocity, measured with timing magnet pulses from the drive spool, is the major source of error. Velocity is measured accurately, but there is likely a delay between the initiation of movement and the detection of the pulse. The delay equates to missed power. Therefore, absolute power scores may be higher, complicating comparing VASA-measured power outputs with outputs from different ergometers. However, the reliability of the VASA is not impacted.

The WAnT-Swim can be used for swim-specific training or evaluation on the pool deck in the laboratory or gym. However, contrary to actual swimming, the test is limited to upper-body exercise and does not include leg work. In addition, the regulation of respiratory rate due to water immersion and the resistance provided by the water are not accounted for during the test. However, VASA claims that the application of force during the pull and the resistance created by air resistance closely approximate resistance while swimming (VASA, 2016). The current study was not designed to assess each of these aspects but rather as the first step in developing a test that truly encapsulates the swimming experience, mainly the body position in the water and a swim-specific stroking movement. Additional studies are warranted to expand these findings and develop methods to emulate actual swimming on land.

Conclusion

The results from this study indicate that the VASA swimming ergometer reliably measured power outputs during a swim-specific modification of the WAnT in a group of ten physically fit college male and female students with varying degrees of swimming ability. Our initial evidence supports the validity of the WAnT-Swim as a modification of the WAnT. Therefore, the WAnT-Swim is likely a reliable and valid test of upper body anaerobic capacity.

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Chapter IV: The Effect of Land-Based Repeated Sprint Hypoxic Training on Anaerobic Capacity and Swimming Performance

Abstract

Performance in a single sprint, repeated sprints, and team sports are partly dependent on anaerobic capacity. Therefore, conditioning programs for these sports target improving anaerobic capacity through various repeated sprint training approaches. A novel training approach combines limiting oxygen availability with repeated sprint training. This hypoxic training approach, repeated sprint training in hypoxia (RSH), aims to induce additional improvements in sea-level performance beyond the same training in normoxia. Typically, RSH training utilizes sprints of 6 to 30 seconds in length, two to three times per week, with a work-to-rest ratio of less than 1:4. To date, the additional benefit of RSH over the well-established effects of repeated sprint training in normoxia (RSN) remains unclear. **PURPOSE:** To determine if RSH training stimulated additional improvements in swimming performance and anaerobic capacity compared to the equivalent RSN training. Performance measures included a 100-yard swimming time trial, a water-based test of repeated sprint swimming ability, a traditional Wingate anaerobic test on a cycle ergometer (WAnT), and a modification of the Wingate test performed on a swimming ergometer (WAnT-Swim). **HYPOTHESIS**: We hypothesized that the increased hypoxic stress during RSH training would stimulate additional improvements in swimming performance and anaerobic capacity compared to the corresponding training conducted in normoxia (RSN). **METHODS:** Twelve Division II male college swimmers from the same team participated in a randomized, single-blind, crossover study comparing the effect of RSH and RSN training on swimming and laboratory-based performance. During RSH, the fraction of inspired oxygen (FiO_2) was maintained at 14.4 ± 0.2%. RSN training was performed in ambient room air. Assessment of swimming performance consisted of a 100-yard time trial and a repeated sprint swimming test. Laboratory-based assessments of anaerobic capacity included the WAnT and a newly developed modification, the WAnT-Swim. Peak power (PP), mean power (MP), low power (LP), and fatigue index (FI) were measured for all Wingate tests. **RESULTS:** The main findings of this study were (1) repeated sprint training significantly improved performance in a 100-yard time trial following RSH (-0.61 seconds, $p < 0.0245$, 1-tail). Following RSN training, time trial performance showed a non-significant increase of 0.30 seconds, a 0.91 second difference between RSH and RSN training. There was no additional benefit of hypoxia over normoxia on time trial performance. (2) Repeated sprint swimming performance significantly improved following RSH and RSN training. Improvements following RSH (total time -1.8 seconds, average decrement -1.1 seconds, mean time -0.5 seconds) and RSH (total time -2.6 seconds, average decrement -1.2 seconds, mean time -0.5 seconds) were significant ($p < 0.05$, 1-tail) for all repeated sprint scores with no additional hypoxia-induced benefits. (3) Following RSH training PP and MP scores measured on the WAnT improved significantly ($p < .001$) correspondingly after RSN training MP and LP improved significantly ($p < 0.001$). PP, MP, and LP measured during the WAnT-Swim improved significantly ($p < 0.001$) after RSH and RSN training with no RSH-induced additional improvement. **CONCLUSION:** land-based repeated sprint training in hypoxia and normoxia using a cycle ergometer and swimming ergometer induced significant improvement in swimming performance and anaerobic capacity. However, during RSH training the increased hypoxic stress did not induce additional improvements in swimming performance or laboratory-based performance measures of anaerobic capacity beyond the same training in normoxia. Therefore, including land-based swim specific and cycle repeated sprint training in the yearly conditioning program of swimmers is warranted with RSN training

being the cost-effective approach. However, additional research is needed to determine if RSH training induces additional improvements in swimming performance compared to the equivalent training in normoxia.

Keywords: Repeated sprints in hypoxia, altitude training, sprint swimming performance, repeated sprints in normoxia

Abbreviations

BMI, body mass index

BPM, beats per minute

FI, rate of fatigue or fatigue index from a Wingate Test

HR, heart rate

LP, low power during a Wingate test

MP, mean power during a Wingate Test

Mode, the type of exercise, i.e., biking or swimming

PP, peak power during a Wingate test

RHS, repeated sprints in hypoxia

RSN, repeated sprints in normoxia

RSA, repeated sprint ability

RST, repeated sprint training

SaO_2, arterial oxygen saturation

$\%SpO_2$, arterial oxygen saturation measured by pulse oximetry

WAnT-S, Wingate arm crank test modified for swimming

WAnT, Wingate Anaerobic test

WAAT, Wingate arm crank test

Introduction

For the last fifty years, athletes have attempted to induce additional improvements in middle and distance race performance by ascending to altitude once or twice a year for two to four weeks to live and train (Flaherty et al., 2016; Lundby et al., 2012). This Live High Train High (LHTH) hypoxic training approach combines the increased hypoxic stress at altitude with aerobic training to stimulate additional enhancements in maximal oxygen consumption (VO_{2max}). The resultant improvements in race performance were attributed to the LHTH approach and fostered an interest in applying hypoxic training to single sprints, repeated sprints, and team sports (Faiss, Girard, et al., 2013). In response to this interest, repeated sprint training in hypoxia (RSH), a live low (LL) hypoxic training approach, was proposed. RSH training is defined as multiple sprints ≤ 30 seconds duration, conducted in hypoxia, and performed on a work-to-rest ratio that does not permit complete recovery (Faiss, Leger, et al., 2013). Athletic performance is operationalized as a single sprint time trial, a sport-specific repeated sprint or a laboratory-based test. A 2017 meta-analysis, based on nine studies with 202 participants, demonstrated small to moderate improvement in mean repeated sprint performance following RSH compared to repeated sprint training in normoxia (Brocherie et al., 2017; G. P. Millet et al., 2019). Conversely, several studies demonstrated that RSH training did not induce additional improvement in performance beyond the corresponding RSN training (Camacho-Cardenosa et al., 2020; P. S. Goods et al., 2015; David Montero & Lundby, 2016). As a result, Montero and Lundby cited a need for additional controlled studies and recommended caution when supplementing existing conditioning programs with RSH training (David Montero & Lundby, 2016). Currently, the reality of RSH-induced additional improvements in a single sprint, repeated

sprints, and team sport performance compared to the same training in normoxia remains an open question (Siebenmann & Dempsey, 2020).

The common link between the hypoxic training approaches is the goal of inducing additional improvements in athletic performance by challenging oxygen homeostasis beyond the equivalent training in normoxia (Hoppeler & Vogt, 2001). RSH training focuses on repeated sprint training to stimulate additional improvements in anaerobic capacity beyond the equivalent RSN training. Recently, oxygen-sensitive transcription factors in the hypoxia-inducible factor family (HIF) have emerged as the likely mediators of the molecular adaptations to hypoxic training (Faiss, Leger, et al., 2013; Serebrovs'ka et al., 2007; Vogt et al., 2001). However, despite confirmation of RSH-induced molecular adaptations within the anaerobic pathway and improved anaerobic capacity, studies demonstrated no coincident improvement in performance (D. Montero & Lundby, 2017) while improved performance has also been observed (Faiss, Leger, et al., 2013; Faiss et al., 2015). The reason for the discrepancy in performance outcomes is unknown. However, factors including the hypoxic dose, individual variation in response to hypoxia (Bonafiglia et al., 2016; R. F. Chapman et al., 2014) and athletic status are being investigated (Gore et al., 2013; P. Robach et al., 2006; Robertson, Aughey, et al., 2010; Siewierski et al., 2012).

Competitive swimmers were among the athletes that supplemented their yearly conditioning programs with LHTH hypoxic training (Bonne et al., 2014; Pyne, 1998; Siewierski et al., 2012). However, improvements in sea-level performances were inconsistent (Gough et al., 2012; Miller & George, 2012), potentially reflecting a wide variation in the individual response to hypoxic training (R. F. Chapman et al., 2014). Moreover, the LH training approaches evoke a hematological-induced improvement in VO_{2max}, which would theoretically benefit performance

in events that rely on a high aerobic capacity (Brutsaert, 2008; B. D. Levine & Stray-Gundersen, 2005; Grégoire P Millet et al., 2010; Paul Robach et al., 2014; Rodríguez et al., 2015; Wachsmuth, Volzke, et al., 2013; Wehrlin & Marti, 2006). In contrast, the ATP-PC system and anaerobic glycolysis supply a significant portion of the energy needed to sustain propulsion during a majority of races in competitive swimming (Hawley et al., 1992; Holmer, 1983; Mercier et al., 1993; Morouco et al., 2015). Therefore, RSH training, which targets improvements in the ATP-PC and glycolytic systems (Medbo JL, 1999; Withers RT, 1991), is potentially a more effective training strategy for competitive swimmers.

To date, only one study assessed the impact of RSH training on swimming performance. In that study, hypoxia was created by pumping air with reduced oxygen concentration (FiO_2 = 13.7%) through a waterproof face mask worn while swimming (Camacho-Cardenosa et al., 2020). The 4-week training protocol consisted of twice weekly completion of 5 sets of 15-meter all-out sprints with 20 seconds of passive recovery between repetitions and a 200-meter easy swim between sets. Time trial performance did not improve following RSH or RSN training. The authors stated that the facemask interfered with stroke mechanics and negatively impacted repeated sprint swim training (Camacho-Cardenosa et al., 2020). Furthermore, during in-the-water RSH swim training, the $\%SpO_2$ (70.1 ± 4.8%) levels were low, similar to high altitude (> 4,000 m) where the resultant reduction in training intensity caused a loss of fitness (P. S. R. Goods et al., 2014; Sweeting et al., 2017). In addition to the hypoxic stress, the participants were fatigued at the beginning of repeated sprint training. Specifically, in-the-water repeated sprint swim training was performed at the end of a two-hour workout (~ 5,000 m) (Camacho-Cardenosa et al., 2020). At that point, glucose and glycogen levels were strained, potentially

reducing training intensity to a level below the threshold needed to evoke a training effect (Chromiakº & Mulvaney, 1990; I. Jacobs et al., 1981; Saltin, 1981).

Therefore, based on the uncertainty of RSH-induced additional improvements in performance, the limited number of sports investigated, and the need for controlled or crossover studies, this study aimed to investigate if RSH training-induced additional improvements compared to the equivalent RSN training in swimming performance and laboratory-based measures of anaerobic capacity in a cohort of highly trained male college swimmers. We hypothesized that the increased hypoxic stress during RSH training would induce additional improvements in swimming performance and anaerobic capacity compared to the equivalent RSN training.

Methods

Participants, Training, Athletic Status, and Anthropometrics

All participants were recruited from a combined Women's and Men's Division II college swimming team. A detailed explanation of the risks, benefits, and expectations of participating in the study was made during a presentation to the team, followed by a question-and-answer period. Exclusion criteria included exposure to altitude, shoulder surgery or injury within the previous 12 months, known cardiovascular disease, a sedentary lifestyle, medications that affected heart rate, and health screening results which required a physician's approval before enrollment. Of the seventy athletes, twelve male swimmers volunteered to participate in this study. Prior to study enrollment, all participants provided written informed consent. The Institutional Review Boards of Syracuse University (IRB # 18-138) and LeMoyne College (IRB # 2018-218) approved this study.

According to self-report, all the participants had trained daily for at least the prior twelve months. Training consisted of at least five swimming and three strength training workouts per week. Swimming practices included three aerobic, one anaerobic threshold, and two anaerobic workouts per week. The average weekly swimming yardage was approximately 35,000 yards. Strength training took place three times per week and consisted of weightlifting and flexibility exercises. The study took place at the beginning of the college swimming season, and the participants continued their regular swimming training while participating in the study.

Athletic status for each participant was estimated by expressing the swimmer's best time, in the 100-yard distance, as a percent of the NCAA Division II Championship qualifying standard. This metric, named Rank, indicated a mean rank of 91.0 ± 4.6% for our sample, meaning they were slightly slower than the national cut-off time. To put that in perspective, nationally, only 30 to 50 swimmers qualify per event for the NCAA DII championship. The breakdown for the primary stroke of the participants was as follows: freestyle = 7, butterfly = 2, breaststroke = 2, and backstroke = 1. Height and weight were measured using a standard physicians' scale with stadiometer. Percent body fat was estimated from Jackson & Pollock 3-site caliper method (Jackson & Pollock, 1985).

Table 4.1 Participant Descriptive Characteristics (*n* = 12)

Variable	Mean ± SD
Age (yrs)	19.7 ± 1.4
Height (cm)	182 ± 6.9
Weight (kg)	78.7 ± 10.6
% Fat	10.9 ± 2.8
BMI ($kg \cdot m^{-2}$)	23.6 ± 2.5
Rank (%)	91 ± 4.6
Weekly swimming distance (yards)	35,000

Note. % Fat= estimated from Jackson & Pollock 3-Site Caliper Method (Jackson & Pollock, 1985) ; BMI= [wt(kg)/ht(cm^2)]*100; Rank=best time as a percent of DII Championship qualifying time

Study Design

Table 4.1 depicts the timeline for the randomized, single-blind crossover study. All participants completed 2, 24-day repeated sprint training blocks (A and B). Each block consisted of seven repeated sprint training workouts. Prior to the first workout, participants were randomly assigned to RSH training (FiO_2 = 14.4 ± 0.2%) or RSN training (normoxia). Partners were randomly assigned and trained together in the altitude chamber. All performance assessments were conducted in normobaric normoxia near sea-level in Syracuse, NY (551 ft.). A 3-week washout period was employed between training blocks A and B as this timeline was associated with reductions in repeat sprint ability and regression of hypoxia-induced molecular changes following hypoxic training (Beard et al., 2019; Brocherie et al., 2017). After the washout period,

the participants completed the remaining training block. Pre-training swim and laboratory-based tests were conducted within five days of the beginning of each 24-day training block. Post-training assessments were performed within three but no later than five days after completion of the training. The dependent performance variables were 100-yard swimming and repeated sprint swimming performance. Anaerobic capacity was assessed using peak power (PP), mean power (MP), low power (LP) and fatigue index (FI) scores on a Wingate anaerobic capacity test (cycle ergometer) and the WAnT-Swim, a swim-specific modification of the WAnT, conducted using a swimming ergometer.

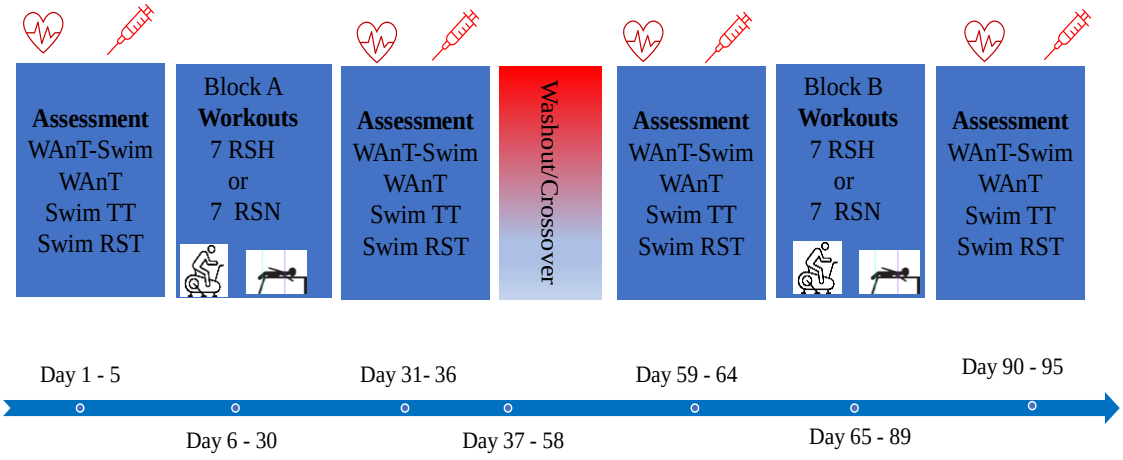


Figure 4.1: Study design

Note. WAnT-Swim = Wingate anaerobic capacity test for swimming; WAnT = Wingate anaerobic capacity test; RSH = repeated sprints in hypoxia; RSN = repeated sprints in normoxia; Swim TT = 100-yard time trial; Swim RST = repeated sprint swim test;
= peak heart rate; = peak lactate

Repeated Sprint Training

RSH and RSN repeated sprint training workouts were conducted in a free-standing normobaric Altitude Chamber with the FiO_2 maintained at approximately 14.5% by replacement of O_2 with N_2 using Hypoxico Systems high flow hypoxic generators (Hypoxico Inc., NY, NY). Two participants exercised in the chamber during each workout. To blind the participants to hypoxia, the Hypoxico system, which made an audible sound, was operated during RSH and RSN workouts. Hypoxic conditions within the chamber were monitored using the Hypoxico System internal monitor and via a sample line from the chamber to an external gas analyzer (VacuMed, model 17500 O_2, CO_2 Dual Gas Analyzer, Ventura, CA). In addition, participants' arterial oxygen saturation levels ($\%SpO_2$) were measured during each workout using a forehead sensor (Nellcor, OxiMax N-600x, Medtronic Minneapolis, MN). Data was collected via LabChart Version 7 and PowerLab 8/30 (AD Instruments, Colorado Springs, CO).

Each workout consisted of repeated sprint training on the bicycle ergometer and simulated swim repeated sprinting on the swimming ergometer. Before the first workout, the order of repeated sprint training was randomized and alternated for each subsequent workout. The specific training protocol (Figure 4.2) consisted of 2 sets of 8 x 20-second sprints on a 1-minute interval with 2 minutes of passive rest between sets. Upon completing the first two sets, the participants rested for 5 minutes, after which they completed training on the remaining ergometer. Exposure to hypoxia was approximately 41 minutes per workout and 287 minutes over the 24-day training program. The dependent variables were 100-yard swimming performance, repeated sprint swimming performance, WAnT and WAnT-Swim power outputs.

Swim-specific repeated sprint training was conducted by simulating the swimming stroke using the VASA swimming ergometer (VASA Inc, Essex Junction, VT). For this study, the

stroke rate was self-selected and consisted of a double arm pulling motion. To begin the stroke, participants were prone on the swim ergometer with their arms fully extended. From their extended position, they pulled under their body with both arms until the hands reached the thigh. The recovery to the starting position consisted of reversing the pull pattern (no resistance) with the hands kept below the ergometer monorail. For all workouts, the damper door of the swimming ergometer was set at level 3 per the manufacturer's guidelines.

During repeated sprint training on the Velotron bicycle ergometer (SRAM Inc., Chicago, Il), the seat height was adjusted until the participant's leg approached full extension during pedaling. For all workouts, the resistance on the bicycle ergometer was set at 50% of the participant's pre-training WAnT PP output [(.075 x weight) x 0.50]. The pedal rate was self-selected. Participants were verbally encouraged throughout the workout to maintain maximal intensity.

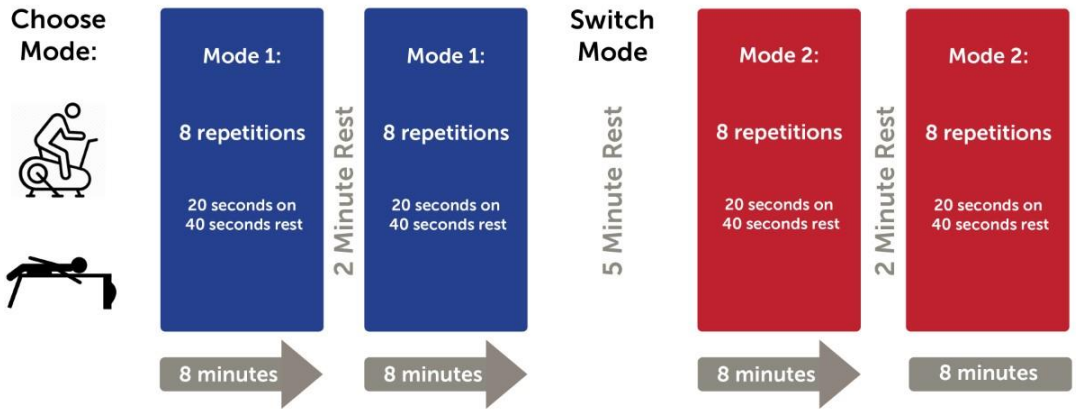


Figure 4.2**:** Repeated sprint workout for RSH and RSN training on the bicycle and swimming ergometers.

Swimming-based Performance Evaluations

1. Time Trial – 100 Yards

Time trials are a common measure of performance in swimming studies (Paul Robach et al., 2014; Rodríguez et al., 2015). Therefore, a 100-yard time trial (primary stroke) was used to assess swimming performance. All the testing, including the time trials used for this study, were conducted by the author, an experienced swimming coach (30 years). Time trials were performed in a six-lane, 25-yard swimming pool, which was used to host NCAA-sanctioned meets. The time for each trial was obtained using a handheld stopwatch.

2. Repeated Sprint Swim Test

Swimming performance was assessed using a previously validated repeated sprint swimming test (Tan, Polglaze, & Dawson, 2010). The test protocol consisted of 6 x 15-meter sprints. Swimmers completed the sprint and started the next within 17 seconds. Clear markings on the deck and lane line indicated the finish lines. Testing was conducted in an outside lane to improve communication and enhance the swimmer's ability to sight the finish line. The swimmers had to tread water to maintain their position at the start line while resting between repetitions. In addition, they were not allowed to push off the wall or bottom of the pool throughout the test. The test measured several components of repeated sprint performance, including Total Time (sum of the six sprint times), Ideal Time (fastest time x 6), Absolute Decrement (total time-ideal time), and Relative Percent Decrement [(total time/ideal time x 100)-100] (Fitzsimons, Dawson, Ward, & Wilkinson, 1993). The time for each trial was obtained using a handheld stopwatch.

Laboratory-based Performance Evaluations

1. WAnT-Swim Procedures

The WAnT-Swim, a swimming-specific methodology for administering a modification of the Wingate Anaerobic Capacity Test (WAnT), was developed for this study. The WAnT-Swim was conducted in normoxia using the VASA swimming ergometer. All the participants had previous experience training on the swimming ergometer. Before conducting the WAnT-Swim, a Polar H7 Bluetooth Heart Rate Sensor & Fitness Tracker (Polar Electro Inc, Bethpage, NY) was secured around the participant's chest using a strap. After 5 minutes of quiet sitting, the resting

heart rate was recorded. During the WAnT-Swim, heart rate was measured continuously using the heart rate monitor and stored with the Polar Beat Multi-Sport Fitness Tracker application.

To reacquaint themselves with the ergometer, participants practiced until they were comfortable with the stroking movement. Once satisfied with their stroking technique, they performed a three-minute warm-up at a self-selected, moderate pace. Following the warm-up, participants rested for one minute. Next, participants were instructed to find a comfortable prone position on the swimming ergometer, extend their arms forward and grasp the hand paddles. Against minimal resistance, they executed a double arm freestyle pull from this position until their hands were at thigh level. The return to the starting position consisted of reversing the pull pattern (no resistance) with the hands kept below the ergometer monorail. Using this pulling motion, participants completed a five-minute warmup with resistance on the flywheel set at three per the manufacturer's recommendations (VASA, 2016). A 10-sec lead-in phase was initiated toward the end of the 5-min warmup, in which participants gradually increased their stroke rate to a self-determined optimum rate. The WAnT-Swim consisted of 30-sec all-out double-arm pulling. Throughout the entirety of the test, participants were verbally encouraged to pull as hard as possible against the resistance. Immediately following the sprint, participants were asked to rate the intensity of the test (RPE: Borg 6-20) and then were allowed to cool down for 5 min by pulling slowly.

During the WAnT-Swim, power output data was collected using the VASA ANT Power Meter in communication with a Garmin USB ANT Stick for Garmin Fitness Devices (Garmin International Inc, KS) and the TrainerRoad application. The VASA ANT Power Meter takes second-to-second measures of time, distance in meters, average watts, stroke rate, and power in watts. The Garmin USB ANT Stick transmits data wirelessly from the VASA ANT Power Meter

to TrainerRoad (Trainer Road LLC, NV). TrainerRoad stores the data, which can be retrieved or exported to Excel. Power output was displayed on a laptop computer, within the investigator and participants' sight, as a motivational and safety tool. In addition, heart rate was monitored via a Polar H7 Bluetooth Heart Rate Sensor & Fitness Tracker (Polar Electro Inc, Bethpage, NY), worn throughout each training session.

2. WAnT Procedures

Using their proprietary software, the WAnT was conducted in normoxia on the Velotron bicycle ergometer (SRAM Inc., Chicago, Il). The load setting for the WAnT was 7.5% of a participant's weight (Bar-Or, 1987; Vandewalle et al., 1985). Participants were allowed to familiarize themselves with the bike operation before the WAnT began. Next, the bike handlebars and seats were adjusted until the participant was comfortable. Once satisfied with their position, athletes completed a three-minute warm-up at 75 watts, a minimal workload. After three minutes, the participants began a 30-second lead-in phase ending with a 10-second phased-in acceleration to their maximum pedal rate. At the end of the 10-second countdown, the load on the flywheel was automatically added. Participants were verbally encouraged throughout the test to maintain maximal intensity. Heart rate was monitored via a Polar H7 Bluetooth Heart Rate Sensor & Fitness Tracker (Polar Electro Inc, Bethpage, NY), worn throughout each training session.

Study Measures

Peak Lactate Level

Previous research demonstrated that peak blood lactate levels occurred within three to eight minutes of completing a 30-120 second sprint test (Gass, Rogers, & Mitchell, 1981).

Therefore, peak lactate levels were measured six minutes after the WAnT and WAnT-Swim tests using a finger stick blood sample and immediately analyzed with a Nova Biomedical Lactate Plus Meter (Nova Biomedical, Waltham MA). In addition, peak lactate levels were measured before and after each 24-day training block.

Minimum Oxygen Saturation (%SpO_2) During Repeated Sprinting

To determine hypoxic stress during the repeated sprint workouts, oxygen saturation data were collected continuously using a forehead sensor, LabChart Version 7, PowerLab 8/30 and exported to Excel for analysis. From that data, the average minimum oxygen saturation level for the simulated swim and cycle workouts was calculated for each athlete. Subsequently, the mean minimum %SpO_2 level during RSH-cycling, RSH-simulated swim, RSN-cycling and RSN-simulated swim workouts was calculated and used to compare hypoxic stress between conditions.

Workload and Heart Rate During Repeated Sprint Training

The training intensity of each workout was estimated from the average power maintained during the repeated sprint cycle and simulated swim workouts. First, the average power for a workout was quantified as the percent of the participant's pre-training MP output on the respective WAnT or WAnT-Swim (average workout power/pre-training MP) x 100. Subsequently, the average power for simulated swimming and cycling workouts was calculated for RSH and RSN training.

The training intensity for each RSH and RSN workout was also estimated from the peak heart rate reached during repeated sprint cycling and simulated swimming. Heart rate was measured continuously and stored for analysis. Stored data from each workout was used to

compute a participant's average peak heart rate for the seven workouts. Two average peak heart rate values were recorded for each workout, one for repeated sprint cycling and one for simulated swimming.

Statistical Analysis

WAnT and WAnT-Swim scores were calculated for peak power (PP), mean power (MP), low power (LP), and fatigue index (FI) (Bar-Or, 1987). Several studies suggested averaging the five highest or lowest power outputs within a 30-second test to determine PP and LP (Wilmore J., 1994). For this study, PP equaled the average of the five highest power readings achieved in the first 10 seconds of the test. MP was the average power output over the 30-second test. LP was calculated as the average of the five lowest power outputs. The FI, also called the rate of fatigue, was calculated using the formula; FI = [(Peak Power-Low Power)/Peak Power] x 100 (Bar-Or, 1987). In addition, the workload for each workout was quantified as the mean power score maintained during the workout expressed as a percent of the participant's first pre-training WAnT or WAnT-Swim mean power score.

A priori sample size of 11 for a Repeated Measures ANOVA was estimated using G-Power 3.1 for the primary dependent variable, 100-yard time trial, with stipulations for a moderate effect size, power of 0.80, and Alpha of 0.05. All data collected were analyzed with SPSS version 29 (IBM, Armonk, NY). Data were assessed for normality using the Shapiro-Wilkes test. Non-normally distributed data were log10-transformed. Visual inspection of boxplots was used to determine outliers, defined as values greater than 1.5 box lengths from the edge of the box (Williamson et al., 1989). To determine their impact on the analysis, where outliers were detected, they were removed, and the analysis was conducted again. Data are reported as mean ± standard deviation. To assess if a training effect occurred following RSH and

RSN repeated sprint training, a paired samples t-test was conducted for WaNT power outputs, WaNT-Swim power outputs, 100-yard-swim time trial, repeated sprint times, peak lactate levels, and peak heart rates. The effect size was estimated using Cohen's *d* (*d*) and Hedges *g* (*g*). Based on the value of *d*, effect sizes were classified as small (*d* = .20), medium (*d* = .50), or large (*d* = .80) (Cohen, 1988, pg. 1310). To determine if hypoxia training induced additional benefits on performance compared to the corresponding RSN training, an analysis of covariance (ANCOVA) was conducted using the GLM Univariate procedure in SPSS 29. In the model, to adjust for differences in baselines, the pre-training performance scores were entered as a covariate (Kaiser, 1989; Kenward & Roger, 2010). Performance change scores (pre–post) were entered as the dependent variable, with hypoxia versus normoxia as fixed factors. Statistical significance was determined a priori as $p \leq 0.05$.

Results

Swimming-Based Tests

Swimming 100-yard Time Trial Performance

One-hundred-yard time trial performance before and after RSH and RSN training is presented in Table 4.2. Performance in the 100-yard time trial improved significantly following hypoxia training (M = 0.61s, $p = 0.0245$, 1-tail, $d = .640$, $g = .595$). Conversely, after normoxia training, 100-yard time trial performance was unchanged. The difference in performance following RSN and RSH was not statistically significant.

Repeated Sprint Swimming Performance

The pre-training, post-training, and change in repeated sprint swimming performance outcomes are presented in Table 4.2. Prior to conducting a paired samples t-test, outliers for pre-

training RSIT (n =1), pre-training RSRD (n = 1), pre-training Total Time (n = 2), and post-training TT (n = 1) were detected. Although inspection of their values did not reveal them to be extreme, therefore, they were kept in the analysis. However, as a precaution, the analysis was conducted with the outliers removed, resulting in no change in statistical significance. In addition, the assumption of normality was violated for the repeated sprint ideal time pre-test as assessed by Shapiro-Wilk's test (RSH, $p < 0.013$) (RSN, $p < 0.007$). Therefore, for analysis, the variable was $\log^{10}$-transformed to better align with normality assumptions.

Two repeated sprint measures improved significantly following RSH training, total time (M = 1.735, $p = 0.034$, 1-tail, d= 0.586, g = .545) and mean time (M = .300, $p = 0.036$, 1-tail, d = 0.575, g = .534). These same measures also improved significantly following RSN training, total time (M = 2.590, $p = 0.036$, 1-tail, $d = 0.574$, g = .534), mean time (M =.500, p = .039, 1-tail, $d = 0.561$, g = .521). In addition, the absolute decrement improved significantly (M = 1.162, $p = 0.041$, 1-tail, $d = 0.552$, g = .513). An ANCOVA was conducted to determine the effect of RSH training compared to RSN training on change in repeated sprint performance. The ANCOVA model indicated that RSH did not induce a statistically significant difference in repeated sprint swimming performance compared to the corresponding RSN training. The between-subjects ANCOVA results are presented in the appendix.

Table 4.2 Swimming Performance Following RSN and RSH Training

Training	100 yard (seconds)	Total Time (seconds)	Ideal Time (seconds)	Absolute Decrement (seconds)	Relative Decline (%)	Mean Time (seconds)
RSN						
Pre-training	57.2 ± 7.3	69.6 ± 10.0	62.6 ± 8.3	7.0 ± 2.4	11.0 ± 3.3	11.6 ± 1.7
Post-training	57.5 ± 7.0	67.0 ± 6.4*	61.2 ± 5.4	5.8 ± 1.8*	9.5 ±.6	11.1 ± 1.1*
Δ-Training	0.3	-2.6	-1.4	-1.2	-1.5	-0.5
RSH						
Pre-training	57.4 ± 7.0	68.7 ± 8.2	61.7 ± 6.8	7.0 ± 2.7	11.3 ± 4.4	11.5 ± 1.4
Post-training	56.8 ± 6.6*	66.9 ± 7.2*	61.0 ± 7.9	5.9 ± 2.0*	10.0 ± 4.0	11.2 ± 1.2*
Δ-training	-0.61	-1.7	-0.7	-1.1	-1.3	-0.3

Note. * = indicates a significant difference between the pre and post-training performance scores indicative of a training effect, $p < 0.05$, 1-tail, paired samples t-test

Laboratory-Based Tests

WAnT-Swim Performance

The pre-training, post-training, and change scores for the laboratory-based performance tests are presented in Table 4.3. A paired samples t-test was used to determine whether a statistically significant difference existed between the pre-training and post-training WAnT-Swim power outputs following RSH and RSN training. No outliers were detected that were more than 1.5 box-lengths from the edge of the box in a boxplot. The assumption of normality was not violated, as assessed by Shapiro-Wilk's test. The results of the paired samples t-tests for performance following RSH showed a significant training effect for PP (M = 54.9, $p < 0.001$, 2 -tail, d= 1.292, g = 1.048), MP (M = 39.83, p =0.001, 1-tail, d= 1.127, g = 1.048), and LP (M = 44.16, p = 0.001 2-tail, d= 1.292, g = 1.202). The trend was similar after RSN with significant

improvement in PP (M = 56.33, p = 0.03, 1-tail, d = .973, g = .905), MP (M = 46.07, p = 0.004, 1-tail, d = .951, g = .885), and LP (M = 37.83, p = 0.008, 1-tail, d= 0.816, g = .759). The ANCOVA model indicated that RSH training did not induce additional improvements in performance for any of the WAnT-Swim power outputs compared to the equivalent RSN training.

WAnT Performance

A paired-sample t-test was used to determine whether there was a statistically significant difference between the change in WAnT power outputs following RSH and RSN training. No outliers were detected that were more than 1.5 box-lengths from the edge of the box in a boxplot. The assumption of normality was not violated, as assessed by Shapiro-Wilk's test. Results of paired samples t-tests indicated a significant training effect following RSH for PP (M = 79.66, p < 0.001, d = 1.233, g = 1.147) and MP (M = 24.90, p < 0.001, d = .293, g = .272). Following RSN, significant training effects were observed for MP (M= 36.00, p = 0.034, 1-tail, d = .699, g =.650) and LP (M = 28.17, p = 0.005 2-tail, d = .998, g =.928). The ANCOVA model indicated that RSH training did not induce additional improvements in performance for any of the WAnT power outputs compared to the equivalent RSN training.

Study Measures

Peak Lactate

Table 4.3 presents the change in peak lactate levels following RSH and RSN training. Potential outliers were noted for peak lactate levels (n = 3) during post-RSH training WAnT-Swim (n = 2) and pre-RSH training WAnT (n = 1). Analyses did not differ with these outliers included or excluded; therefore, the outliers were included for all subsequent analyses. Peak

lactate levels did not change significantly from baseline following the WAnT or WAnT-Swim. However, peak lactate levels did differ between the WAnT and WAnT-Swim tests. Before the start of RSH training, the pre-training peak lactate levels measured after the WAnT were significantly higher than levels measured after the WAnT-Swim (M= 4.45, $p = 0.001$, $d = 1.315$, $g = 1.223$). The same was true for RSH post-training peak lactate levels (M = 4.091, $p < 0.001$, 2-tail, $d = 1.765$, $g = 1.641$). Comparably, for RSN training, the pre-training (M = 4.500, $p < 0.001$, 2-tail, $d = 1.562$, $g = 1.453$) and post-training peak lactates (M = 5.366, $p < 0.001$, 2-tail, $d = 1.859$, $g = 1.729$) were significantly higher when measured after the WAnT than the WAnT-Swim.

Peak Heart Rates

Heart rate was measured continuously during the WAnT and WAnT-Swim. Eight peak heart rates were determined per participant, one for each Wingate test (4 WAnT, 4 WAnT-Swim). Test results were used to calculate the pre-to-post-change in peak heart presented in Table 4.3. Peak heart rates measured during the WAnT and WAnT-Swim did not change significantly following RSH, or RSN repeated sprint cycle or simulated swim training. However, pre- and post-training peak heart rates measured during the WAnT were significantly higher than those measured during the WAnT-Swim. The differences in peak heart rates between the WAnT and WAnT-Swim for all conditions were: RSH pre-training (M = 15.42, $p < 0.002$, 2-tail, $d = 1.187$, $g = 1.104$), post-training (M = 13.33, $p < 0.002$, 2-tail, $d = 1.366$, $g = 1.270$). Similar results were observed for RSN pretraining (M = 13.08, $p < 0.001$, 2-tail, $d = 1.65$, $g = 1.53$) and post-training (M = 14.58, $p < 0.001$, 2-tail, $d = 1.98$, $g = 1.84$).

Table 4.3 Change in WAnT, WAnT-Swim, Peak Lactate, Peak Heart Rate following RSH and RSN Training

Condition/Test	Peak Lactate (mmol/L)	PP(W)	MP(W)	LP (W)	FI	Peak HR (bpm)
RSN WAnT						
Pre-Training	11.9 ± 1.8	800.3 ± 124.7	684.9 ± 99.0	535.4 ± 84.1	32.4 ± 10.1	173 ± 10
Post-Training	12.5 ± 1.4	814.9 ± 106.4	720.9 ± 91.4**	563.6 ± 91.4**	30.4 ± 8.4	173 ± 9
Δ-Training	0.6	14.6	36	28.2	-2.0	0
RSH WAnT						
Pre-Training	10.6 ± 2.2	744.5 ± 91.6	664.3 ± 74.3	522.3 ± 122.0	29.4 ± 16.5	173 ± 11
Post-Training	11.9 ± 1.9	824.2 ± 102.3**	708.8 ± 69.6**	547.2 ± 69.7	33.1 ± 8.7	172 ± 10
Δ-Training	1.3	79.7	44.5	24.9	3.7	1.0
RSN WAnT-S						
Pre-Training	7.5 ± 3.5	232.6 ± 80.1	194.3 ± 68.3	165.9 ± 66.9	29.2 ± 9.8	160 ± 12
Post-Training	7.0 ± 1.5	288.9 ± 42.2**	240.4 ± 37.7**	203.7 ± 42.3**	29.5 ± 9.6	159 ± 13
Δ-Training	-0.3	56.3	46.1	37.8	0.3	-1.0
RSH WAnT-S						
Pre-Training	6.7 ± 1.7	239.1 ± 46	201.3± 38.6	166.6 ± 34.3	30.2 ± 7.6	157 ± 13
Post-Training	7.4 ± 1.6	294.0 ± 33.1**	241.2 ± 31.0**	210.7 ± 30.1**	28.2 ± 8.2	159 ± 12
Δ-Training	0.7	54.9	39.9	44.1	-2.0	2.0

Note. WAnT= Wingate anaerobic capacity test; WAnT-S = Wingate anaerobic capacity swim test; * = indicates a significant difference $p < .05$, between the pre and post-training performance scores indicative of a training effect 1-tail paired samples t-test; ** = indicates a significant difference between the pre and post-training performance scores indicative of a training effect $p < 0.01$, 2-tail paired samples t-test.

Oxygen Saturation (%SpO_2) During Repeated Sprint Workouts

The participant's oxygen saturation level (%SpO2) was continuously measured during the RSH and RSN workouts. For each workout, the second-to-second data were stored for analysis and used to determine the oxygen saturation level during the repeated sprint cycle and simulated swim workouts (not during rest). Table 4.4 presents the mean minimum %SpO_2 levels calculated for RSH and RSN training. As expected, %SpO_2 levels within ergometer type were significantly lower during RSH compared to RSN training (cycling M = -12.31, p = 0.001 2-tail, d = 4.021, g = 3.739) simulated swim (M = -12.308, $p < 0.001$, 2-tail, d = 5.079, g = 4.723). However, during RSN, repeated sprint simulated swim training, the minimum %SpO_2 levels were significantly lower than during cycle repeated sprint training (M = -3.13, p = 0.004, 2-tail, d= 1.06, g = .986).

Table 4.4 Mean Minimum %SpO_2 During RSH and RSN Training

Type of Training	**Minimum %SpO_2**
RSN Cycle	95.6 % ± 0.8
RSH Cycle	83.3 % ± 2.2 **
Δ **Hypoxic Condition**	-12.3 %
RSN Simulated Swim	92.5 % ± 2.8 *
RSH Simulated Swim	84.2 % ± 2.2 **
Δ **Hypoxic Condition**	-8.3 %

Note. %SpO_2 = arterial oxygen saturation measured using a forehead sensor; * = indicates a significant difference between the minimum % SpO_2 level during RSN simulated swim compared to RSN cycle, $p < 0.01$, 2-tail paired samples t-test; ** = indicates a significant difference between the minimum % SpO_2 level during RSN cycle vs. RSH cycle and RSN simulated swim vs. RSH simulated swim, $p < 0.001$, 2-tail t-test.

Workload During Repeated Sprint Workouts

Table 4.5 presents the mean values for the workload maintained during the RSH and RSN repeated sprint workouts. During RSH repeated sprint cycle training, the average workload was 19.5% ± 1.6 compared to 19.9% ± 1.3 during RSN training. The mean workload during RSH simulated swim repeated sprinting workouts was 64.2% ± 11.0 compared to 73.2% ± 20.8 during RSN training.

The paired samples t-test indicated that the difference between the workload maintained during RSH and RSN repeated sprint cycling workouts did not reach significance. Similarly, the workload maintained during simulated swim repeated sprinting workouts did not differ between hypoxic conditions. However, compared to repeated sprint cycling, the mean workload during the simulated swim workouts was significantly higher (M = 44.72, $p < 0.001$, 2-tail, $d = 4.21$, $g = 3.91$). Moreover, during RSN training, significantly higher mean workloads were maintained during the simulated swim vs. cycling workouts (M = 53.24, $p < 0.001$, 2-tail, $d = 2.68$, $g = 2.49$).

Heart Rate During Repeated Sprint Workouts

Table 4.5 presents the mean values for the peak heart rates recorded during RSH and RSN training. During RSH repeated sprint cycling workouts, the peak heart rate was 170 ± 11 bpm and 165 ± 11 bpm during the RSN workouts. The difference in heart rates was not significant. Similarly, the difference between peak heart rates during RSH (138 ± 11 bpm) and RSN (142 ± 14 bpm) repeated sprint simulated swimming workouts was not significant. However, significantly lower peak heart rates were recorded during repeated sprint simulated

swim training compared to cycle training in hypoxia (M = -26.67, $p < 0.001$, d= 3.47, g =3.206) and normoxia (M = -27.91, $p < 0.001$, d= 4.295, g = 3.994).

Table 4.5 Workload and Heart Rate During RSH an RSN Cycle and Simulated Swim Workouts

Training	**Mean Workload (% mean power)**	**Peak Heart Rate (bpm)**
RSN Cycle	19.9 ± 1.3	170 ± 11
RSH Cycle	19.5 ± 1.6	165 ± 11
Δ-Hypoxic Condition	0.40	5.0
RSN Simulated Swim	73.2 ± 20.8	142 ± 14
RSH Simulated Swim	64.2 ± 11.0	138 ± 11
Δ-Hypoxic Condition	-9.0	-4.0

Note. Mean workload = the percent of the pre-training Wingate mean power score maintained during the workout; There was no significant difference between training workload or heart rate in normoxia or hypoxia when using the same ergometer; peak heart rate was significantly higher while sprinting on the bike than during simulated swim (p = 0.000, 2-tail paired samples t-test)

Discussion

This study assessed if RSH training induced additional improvements in anaerobic capacity and swimming performance compared to the equivalent RSN training. A randomized, single-blind, crossover design consisting of two training blocks (A and B) separated by a 3-week washout period was employed. Simulated swim and cycle repeated sprint training consisted of seven workouts completed over 24 days in hypoxia (14.4± 0.2%) or normoxia. The main findings of this study were 1) One-hundred-yard time trial, repeated sprint swimming, WAnT, and WAnT-Swim performance improved significantly following RSH or RSN training. 2) The hypothesis that repeated sprint training in hypoxia induced significant additional improvement in

100-yard and repeated sprint swimming performance beyond the same training in normoxia was not confirmed. 3) Contrary to our hypothesis, the increased hypoxic stress during RSH training compared to the equivalent RSN training did not induce additional improvements in anaerobic capacity.

To determine the effectiveness of RSH training, the absolute training intensity must be equivalent between RSH and RSN training with the magnitude of hypoxic stress the only difference between them (Brocherie et al., 2017). In the current study, training intensity for a workout was quantified as the mean power expressed as a percentage of the respective pre-training Wingate mean power score. Mean power did not differ significantly between simulated swim repeated sprinting during RSH (64.2 % ± 11.0%) vs. RSN (73.% ± 20.8%). Correspondingly, during repeated sprint cycling, the training intensity during RSH (19.5% ± 1.6%) was almost identical to RSN training (19.9% ± 1.3). In addition, a second measure of intensity, peak heart rate during simulated swim RSH (138 ± 11bpm) vs. RSN (142 ± 14 bpm) were not significantly different, nor were they during repeated sprint cycling RSH (165 ± 11bpm) vs. RSN (170 ± 11). Finally, the hypoxic stress, quantified as $\%SpO_2$ was the only apparent difference between hypoxic training conditions during simulated swim training, RSH (84.2% ± 2.2%) vs. RSN (92.5% ± 2.8%) and cycling, RSH (83.3% ± 2.2%) vs. RSN (95.6% ±0.8%). Therefore, we are assured that hypoxic stress was the only difference between our sprint conditions and not differences in training intensity. Regardless, there were no RSH-induced additional improvements in performance or anaerobic capacity. These findings are in alignment with several studies (Camacho-Cardenosa et al., 2020; P. S. Goods et al., 2015; David Montero & Lundby, 2016), but contrary to others. The lack of influence of hypoxia on study outcomes between our study and others may be due to methodological considerations.

Specifically, to improve statistical power and to minimize variability we utilized a crossover rather than a parallel study design and adjusted the ANCOVA model for the treatment baseline (X. Chen, Meng, & Zhang, 2012; Kenward & Roger, 2010). Therefore, we are confident that individual difference in the training response and period related method effects had minimal influence on outcomes (X. Chen et al., 2012) In addition, repeated sprint training was conducted at a level of hypoxia deemed effective for RSH training (Bowtell et al., 2013; Brocherie et al., 2017; Feriche et al., 2007; Ogawa et al., 2005). Furthermore, participants were blind to the training condition. In contrast, many studies reporting RSH-induced additional improvements in performance did not incorporate blinding, controls, a crossover study design, measure $\%SpO_2$ during sprinting or utilize a FiO_2 within the suggested range of 13.0-15.5% (Faiss, Leger, et al., 2013; Galvin et al., 2013). Therefore, without control conditions attributing improvements to RSH training rather than differences between groups is questionable (Brocherie et al., 2017; D. Montero & Lundby, 2017).

The lack of RSH-induced additional improvement in performance found in this study, concurs with the findings of the only two crossover studies reported in the RSH training literature. (Camacho-Cardenosa et al., 2020; D. Montero & Lundby, 2017). Between the three studies, the lack of RSH-induced improvement was found along a range of FiO_2 levels, blinding procedures, training protocols, participants, performance assessments, washout periods, and statistical tests. For example, blinding procedures included double-blind, single-blind (participants) and single-blind (analysts). Importantly, a key element of a crossover study is the duration of the washout period. Given the recent development of RSH training, a consensus as to the duration of the washout period has not been reached. Therefore, the fact that each of the crossover studies used a different washout period (2, 3, 6 weeks) suggests that our 3-week

washout period was adequate and strengthens our conclusion that RSH did not induce additional improvements in performance.

The hypoxic dose differed between the three studies offering insight into the impact of dose on outcomes. Participants in the Montero and Lundby study completed 12 workouts equating to approximately 528 minutes of total exposure to hypoxia of which less than 4 minutes per workout was actual sprinting (48 min total) (D. Montero & Lundby, 2017) On the other hand, over 8 workouts swimmers in the Camacho-Cardenosa et al. study were exposed to hypoxia for 128 min of which 52 min was sprinting (Camacho-Cardenosa et al., 2020). By comparison, over 7 workouts swimmers in our study were exposed to hypoxia for approximately 287 min, of which 75 min was sprinting. Therefore, in the combined studies total exposure to hypoxia varied from 128 min to 528 min and sprinting in hypoxia from 48 min to 75 min with no difference in outcome between studies. In addition, the method of creating hypoxia (facemask vs. chamber) and the absolute FiO_2 (13.8% vs.14.4%) differed between studies. Suggesting that utilizing a lower FiO_2 or method of creating hypoxia in our study would not have altered the outcome.

Consistent with findings reported in the literature, in the current study, post-WAnT and WAnT-Swim peak lactates did not change significantly from baseline measures following training in either condition(C. Brechbuhl et al., 2018; B. Dawson et al., 1998). However, post-training PP, MP, and LP were significantly higher at the same peak lactate level than their pre-training counterparts, with no additional benefit from RSH training. Higher power outputs at the same peak lactate levels support the theory that repeated sprint training enhances anaerobic capacity, possibly through improved regulation of lactate. However, RSH training does not appear to provide additional improvement beyond the corresponding RSN training (C. Brechbuhl

et al., 2018; Faiss et al., 2015; Galvin et al., 2013; David Montero & Lundby, 2016). Therefore, given the similar improvement in anaerobic capacity between RSH and RSN training, RSN may be the cost-effective and pragmatic choice for training.

Individual variation in response to hypoxic training is known (R. F. Chapman, 2013). Various methods are used to minimize the impact of individual variation on study outcomes. One method classifies the participants by athletic status with the underlying assumption being that equivalent status implies a similar response to training. In the present study, athletic status was defined by ranking the athletes relative to the Division II qualifying time in their event. This approach may be a viable option for sports that measure performance by time. Alternatively, quantifying the participant's fitness level using standardized procedures such as a WAnT or the Yo-Yo test would provide a common metric between studies. In this regard, future studies may benefit by defining athletic phenotypes. Accounting for individual differences by phenotype rather than through the classification of athletic status or measuring fitness levels would improve analysis and eventually aid in exercise prescription and training design (Ehlert, Simon, & Moser, 2013; Feriche et al., 2007; Moir et al., 2019; Roth et al., 2012; Timmons et al., 2010; Wang et al., 2013).

The rationale underlying RSH training is that the challenge to cellular oxygen homeostasis created by the reduced availability of oxygen will enhance adaptations to training beyond the equivalent RSN training resulting in additional improvements in athletic performance (Brocherie et al., 2017; Faiss, Girard, et al., 2013; Faiss, Leger, et al., 2013). Various RSH-induced adaptations have been identified. For example, although not assessed in this study, several research teams have demonstrated RSH-induced additional increases in muscle blood perfusion coincident with and without improved performance (Faiss, Leger, et al., 2013; David

Montero & Lundby, 2016). In addition, the activity of phosphofructokinase (PFK), a key regulatory enzyme of glycolysis, has shown greater increases after RSH compared to RSN training (Puype et al., 2013), suggesting enhanced anaerobic capacity.

In studies where RSH training did result in additional improvements in anaerobic capacity, changes in hypoxia-inducible factor pathway (HIF) gene regulation were hypothesized as the underlying mechanism (Beard et al., 2019; Childebayeva et al., 2021; Faiss, Leger, et al., 2013). Specifically, RSH, compared to RSN training, may induce additional adaptations in the HIF-1 α pathway (Beard et al., 2019; Gregg L Semenza, Shimoda, & Prabhakar, 2006). HIF-1 α is a transcription factor that regulates genes associated with maintaining oxygen homeostasis. However, few studies have investigated the impact of RSH-induced modifications in genes regulated by the HIF-1 α pathway on performance (Brocherie et al., 2017; Faiss, Leger, et al., 2013; Nava et al., 2022).

One study demonstrated the upregulation of vascular endothelial growth factor (VEGF) following RSH training. VEGF is regulated by HIF-1 α and induces the formation of new blood vessels (angiogenesis) in response to hypoxia (Lindholm & Rundqvist, 2016; Ziello et al., 2007). Increased vascularity may underlie the enhanced muscle perfusion following RSH training observed by several research teams (Faiss, Leger, et al., 2013; David Montero & Lundby, 2016). In addition, in response to low tissue oxygen levels, genes associated with glucose transporters and glycolytic enzymes are upregulated by the HIF-1 α pathway (Kierans & Taylor, 2021). For example, following RSH training PDK1 was upregulated (Nava et al., 2022). The PDK1 gene regulates pyruvate dehydrogenase kinase (PDK), which inhibits the conversion of pyruvate to acetyl-CoA increasing energy production through anaerobic glycolysis, potentially resulting in improvements in repeated sprint performance (Nava et al., 2022). The fact that we did not find

additional improvements in performance suggests that changes in gene regulation may not have occurred or were not large enough to impact performance beyond RSN training. Future RSH studies designed to determine the optimal hypoxic dose and training program necessary to evoke changes in gene regulation sufficient to induce additional improvements in performance beyond RSN training are needed. Furthermore, elucidating the differential effects of RSH and RSN training on gene regulation may eventually lead to a better understanding of the efficacy of RSH training. However, given the polygenic nature of the response to training, significant additional research is necessary to determine the response of HIF-1α pathway genes to RSH and RSN training and their subsequent impact on athletic performance (Brocherie et al., 2018; Ehlert et al., 2013; Moreland et al., 2022; Pramkratok, Songsupap, & Yimlamai, 2022; Soori et al., 2020; Wang et al., 2013).

Unique aspects of this study

The workload during repeated sprint cycling and simulated swimming remained relatively constant between workouts. However, the difference in workload between cycling and simulated swim sprinting was significant ($p \leq 0.001$). Participants maintained ~19% of their WAnT pre-training mean power score during cycling. In contrast, participants maintained ~70% of their pre-training WAnT-Swim mean power score during repeated sprinting on the swim ergometer. The impact of the difference in workloads on performance outcomes is noteworthy. PP, MP, and LP scores from pre-to-post-training in our participants increased to a greater extent on the WAnT-Swim than on the WAnT; PP (26% vs. 11%), MP (22% vs. 7%), and LP (30% vs.12%) following RSH, with a similar differential following RSN. This improvement in WAnT-Swim scores is consistent with the expectation that the higher workloads maintained during simulated swimming would confer greater benefits. However, distilling the unique impact of

cycling and simulated swimming sprint training on performance outcomes is difficult because all the workout sessions included both simulated swim and cycle repeated sprinting.

Cardiovascular stress estimated from peak heart rate during repeated sprinting was significantly higher during cycling than during simulated swimming. However, differences in heart rates between RSH and RSN were not significant within the same training mode. Peak heart rates during repeated sprint cycling were similar to peak heart rates measured during the pre-training WAnT (1-3 bpm), coincident with lower relative workloads than during repeated sprint simulated swimming. During repeated sprint simulated swimming, peak heart rates were 15-19 beats less than those measured during the pre-training WAnT-Swim. Compared to simulated swimming, the higher training heart rates during cycling reflect the greater muscle mass involved (di Prampero, 2003). Regardless of the cause, the higher relative heart rates imply increased cardiorespiratory stress during cycling versus simulated swim sprinting. However, this does not explain the higher workloads at the lower relative heart rates maintained during simulated swim repeated sprinting. The difference in heart rate and workload between cycle and simulated swim sprint training may reflect long-term adaptations specific to highly trained swimmers (Secher, Ruberg-Larsen, Binkhorst, & Bonde-Petersen, 1974).

Limitations and Considerations

The results of our study did not support the hypothesis of RSH-induced additional improvement in performance. Two feasible explanations for this finding are 1) RSH does not induce additional benefits on performance compared to the equivalent RSN training, or 2) although a priori analysis indicated a sample size of 11 provided sufficient power to detect differences between RSH and RSN improvements, there may not have been adequate power to detect small differences. Post-hoc power analysis (table in the appendix) indicated that the

sample size of 12 was adequate to detect differences between RSH and RSN training on improvement in the 100-yard time trial. However, a larger sample size may have allowed the detection of RSH-induced additional improvement in the other dependent variables. For example, for three dependent variables, WAnT PP, WAnT peak lactates, and WAnT-Swim FI, minimal increases in sample size (2, 4, and 8, respectively) may have detected differences. The remaining variables required much larger increases in sample size. However, the effect size for those variables was small to trivial ($d < 0.20$). Therefore, based on the sample size alone, we don't believe any meaningful effects of RSH compared to RSN training were missed.

Male and female swimmers were recruited for this study; however, no female swimmers volunteered. Therefore, the results of this study cannot be generalized to female college swimmers. Currently, only one study included in the meta-analysis (Brocherie et al., 2017) investigated the impact of RSH training on female athletes ($n = 32$). In that study, the female college soccer players demonstrated significantly greater improvement in peak power and mean power scores on a repeated sprint test compared to the RSN trained women (peak power output, 5.0 +/- 0.7% vs. 1.5 +/- 0.9%, respectively; mean power output, 9.7 +/- 0.9% vs. 6.0 +/- 0.8%, respectively; $P < 0.05$) (Kasai et al., 2015). Therefore, female, and male athletes may respond differently to RSH training. Although the disparity in the number of studies conducted in the exercise science and biomedical fields with male compared to female participants has decreased, there is a significant need for research with female participants (Landen et al., 2023). Furthermore, given that sex-linked physiological differences impact adaptation to training, it is imperative to clarify these differences to improve exercise prescription and maximize performance in female and male athletes (Ansdell et al., 2020).

Finally, we followed the repeated sprint swim test procedure described by Tan et al. (Tan et al., 2010). Although the test was shown to be reliable and the same investigator measured time for all the tests, there is the possibility that human error affected the results.

Conclusion

This study assessed the potential for RSH training to induce additional improvement in anaerobic capacity, swim and repeated sprint swimming performance compared to the equivalent RSN training. Despite the increased hypoxic stress created by the reduced FiO_2 and similar training intensity, RSH training did not induce additional improvements in swimming performance or anaerobic capacity beyond the equivalent RSN training. Therefore, at this time, the inclusion of RSH training into the yearly conditioning program of college swimmers is not recommended. However, including a swim-specific land-based repeated sprint training program may be beneficial. Finally, due to the recent development of the RSH training approach, additional research that incorporates standardized tests of anaerobic capacity, methods that control for the confounding impact of athletic status and interindividual variation in response to hypoxia are needed to determine the effectiveness of RSH training in inducing additional improvements in athletic performance compared to the equivalent RSN training.

Chapter V: Summary, Future Directions and Considerations

Summary

The desire to win drives athletes and coaches to seek out innovative methods to improve performance. The LH hypoxic training approaches that emerged after the 1968 Olympics are a good example. The LH approaches focused on increasing aerobic capacity to enhance performance in middle and distance events. Today, one or more of the LH approaches are integral components of the training programs for many world-class endurance athletes. In addition, the purported hypoxia-induced improvements in performance outcomes stimulated interest in applying hypoxic training to improve performance in a single sprint, repeated sprint, and team sports. This interest resulted in the development of the RSH training approach.

Performance in a single sprint, repeated sprint and team sports rely, in part, on repeated sprint ability (RSA). RSA represents the capacity to maintain maximum or near-maximum speed (70% of max) for several repetitions with little or no rest between sprints. On the other hand, a single sprint requires explosive power applied over 6-30 seconds with complete recovery between sprints. The energy needed to compete in sprints and repeated sprints is supplied primarily through anaerobic metabolism.

Several potential adaptations within the skeletal muscle are being investigated as the reason for the RSH-induced additional improvement in performance beyond the equivalent RSN training. Among them is an enhanced rate of PCr resynthesis (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Kasai et al., 2017; Mendez-Villanueva, Edge, Suriano, Hamer, & Bishop, 2012), increased levels of PFK (Puype et al., 2013), increased levels of PC, increased glycogen, (J. Bangsbo et al., 1991; Kasai, Kojima, & Goto, 2018) and enhanced pH regulation (C. Brechbuhl et al., 2018; Puype et al., 2013). In addition, increased blood flow, selectively favoring fast

twitch muscle fibers, has been demonstrated (Brocherie, Girard, Faiss, & Millet, 2015; Faiss, Leger, et al., 2013; David Montero & Lundby, 2016). Furthermore, a hypoxia-induced stimulation of the HIF pathway, which regulates many genes associated with anaerobic metabolism, has been suggested as the mechanism regulating the adaptations to RSH training (Faiss, Girard, et al., 2013; Ziello et al., 2007). However, additional research is needed to confirm the adaptions to RSH compared to RSN training and the role of the HIF pathway in regulating the response.

The first step in this dissertation assessed the reliability of the WAnT-Swim test conducted using the VASA swimming ergometer. The ergometer permits the athlete to simulate an under-the-body, double-arm pulling motion used for the test. This land-based simulated swimming test solves multiple problems associated with collecting data from swimmers in an aquatic environment. Our results show high interclass correlation coefficients for all the Wingate Scores. The ICC for Peak Power (0.97) CI [0.932, 0.993], Mean Power (0.98) CI [.951, .995], and Low Power (0.977) CI [.941, .994) were high. However, the FI was moderately reliable based on the ICC [0.0.676) CI [.0.676 – 0.912]. However, similar to the other test scores, CV was low, suggesting that the WAnT-Swim is a reliable test. Furthermore, our preliminary analysis of the criterion validity of the WAnT-swim as a modification of the WAnT offered limited support for the validity of the test. Additional research is needed to confirm the validity of the WAnT-Swim as a modification of the WAnT and test of anaerobic capacity.

The second step tested our main hypothesis, defined in Aim 1, that RSH training would improve 100-yard swimming performance beyond the same training in normoxia (Figure 5.1). After completing a crossover study consisting of two 24-week training blocks, our hypothesis was not confirmed. However, RSH training significantly improved 100-yard time trial

performance by 0.61 seconds ($p < 0.0245$). On the other hand, RSN training resulted in a non-significant increase of 0.30 seconds; however, the difference between them was not significant. At the same time, this finding may indicate a trend toward a hypoxia-induced benefit on 100-yard swimming performance.

Our hypothesis in Aim 2 that RSH training would induce additional improvements beyond the corresponding RSN training in repeated sprint swimming performance was also unconfirmed (Figure 5.1). As with time in the 100-yard swim, repeated sprint performance improved following both RSH and RSN with no significant difference between the type of training. Notably, two components of the repeated sprint test did improve significantly following RSH or RSN training. The Ideal Time and Relative Decline improved following both training programs, but the improvement did not reach significance. Ideal time is a theoretical time calculated from the fastest time multiplied by six, and absolute decrement is the percent decline from sprint to sprint.

Our third hypothesis of a RSH-induced additional increase in anaerobic capacity beyond the corresponding RSN training was also unconfirmed. Anaerobic capacity estimated from WAnT and WAnT-Swim performance was not significantly different between RSH and RSN training. A part of the analysis of improvement following RSH and RSN training examined changes in the WAnT and WAnT swim performance measures (Figures 5.2 and 5.3). Here too, RSH training did not evoke benefits beyond RSN training. However, PP on the WAnT following RSN training did not improve significantly, while after RSH, there was a significant improvement and a large effect size ($p = 0.001$, $d = 1.233$). The finding of significant improvement and large effect in PP suggests that RSH may provide an additional real-world improvement in performance beyond RSN training and warrants additional research.

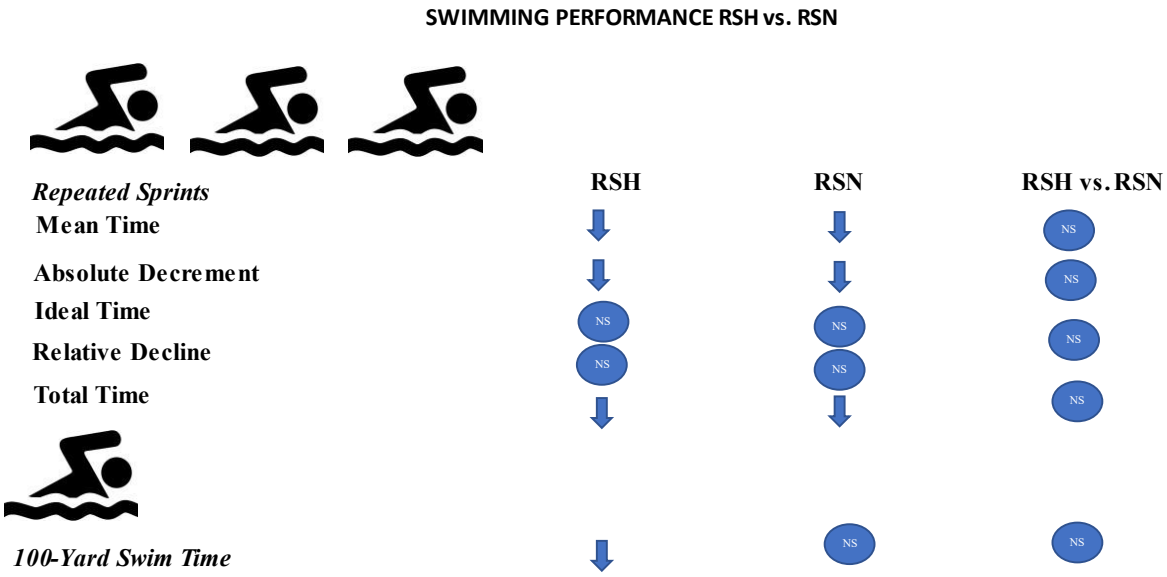


Figure 5.1: 100-yard and repeated sprint swimming performance following RSH and RSN.

= *significant improvement in time;* NS = *non-significant change in performance*

Figure 5.2: WAnT-Swim performance following RSH and RSN.

= *significant improvement in time;* NS = *non-significant change in performance*

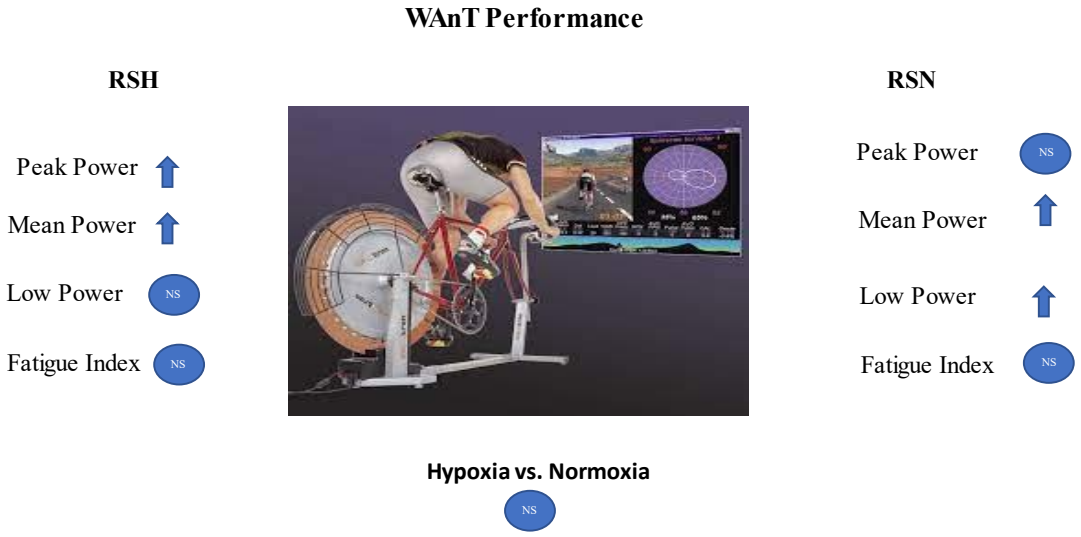


Figure 5.3: WAnT performance following RSH and RSN.

↓ = *significant improvement in time;* NS = *non-significant change in performance*

Implications

The main implication of our study is that RSH training may induce additional benefits of practical significance to athletes. Given the small differences in improvement between RSN and RSH training, additional research with larger sample sizes is needed. In fact, based on the scientific literature, the case can be made for an additional small to moderate RSH-induced benefit on performance. Furthermore, there is high interindividual variability in response to RSH training. In other words, RSH training may be extremely beneficial to one athlete and ineffective, possibly detrimental to another. Current work to identify athletic phenotypes may help differentiate between responders and non-responders. Moreover, there is a wide variation in the individual response to the same FiO_2 (R. F. Chapman, Stray-Gundersen, & Levine, 1998). For example, athletes using the same RSH training protocol may receive effective or ineffective

hypoxic stress. Therefore, controlling for this individual variation in response to a given FiO_2 within a study design is an important factor in determining the efficacy of RSH training. One research team suggested using the SpO_2 to FiO_2 ratio to individualize the hypoxic dose in studies (Soo, Girard, Ihsan, & Fairchild, 2020).

A second important implication from this study stems from the development of the WAnT-Swim, a land-based modification of the Wingate Anaerobic Capacity Test. This test was reliable when conducted on the VASA swimming ergometer. Moreover, our data provided preliminary albeit limited support for the criterion validity of the WAnT-Swim. Pragmatically, the WAnT-Swim provides sports scientists and swimming coaches with a reliable land-based method to assess swim-specific physiologic and performance components. In addition, the swimming ergometer offers an easily accessible alternative to in-the-water training. For example, during this study, two swimming practices per week were replaced with repeated sprint workouts using the ergometers, resulting in improved swimming performance.

The third implication underscores the importance of carefully classifying participants' athletic status using standardized methods. Currently, many investigations attempted to minimize the impact of the interindividual response to training using the construct of athletic status. Within the concept of athletic status is the assumption of a homogenous fitness level and similar response to training between participants. However, athletic performance results from the interaction between multiple factors, of which fitness is one (Drozdovska, Dosenko, Ahmetov, & Ilyin, 2013). As an alternative to athletic status, we suggest including standardized anaerobic and aerobic capacity assessments in study designs. Standardized tests would provide an objective fitness measure and facilitate comparison between studies.

Finally, although our study demonstrated no significant difference in performance improvement between RSH and RSN training, the seven-workout land-based training design stimulated rapid improvement in all performance tests. Thus, the repeated sprint protocol may be useful in retraining athletes after they return from an injury. In addition, repeated sprint training may offer an effective way to retrain after the end-of-the-season taper and peak. For example, before the year-end Championship, swimmers often reduce daily swimming yardage and intensity for 2- 4 weeks. After the Championship, a week of repeated sprint training before the break between seasons may limit the loss in fitness associated with the break (no training). The same repeated sprint training program could accelerate an athlete's return to their previous fitness level at the beginning of the season. Furthermore, two land-based repeated sprint training workouts per week (< 2 hours) resulted in improved swimming performance despite the elimination of two in-the-water swimming practices (4 hours). Therefore, based on the efficiency of the land-based repeated sprint training, coaches may consider including it in the training programs of time-strapped college student-athletes or as a substitute when in-the-water training isn’t an option.

Future directions

The development of the WAnT-Swim provides a valuable tool to sports scientists, coaches, and athletes for testing and training. Additional research to determine the validity of the tests is necessary. A follow-up study that includes male and female participants and measures such as lactate levels and heart rate is needed. Furthermore, determining the relationship between PP, MP, LP and FI scores and swimming performance over various distances is needed.

Our data indicate that RSH training did not induce additional improvement in swimming performance or anaerobic capacity beyond the same training in normoxia. However, our study

was limited to male college swimmers, a specific training protocol, and a sample size of 12. The next step is to repeat the study with a larger sample and include female participants. Manipulation of the training protocol and the hypoxic dose is also necessary. Similar to LH training, longer exposure may be better. For example, increasing the training to 12 weeks, more than double the longest study design used to date, may help determine if RSH stimulates additional improvements in performance beyond the equivalent RSN training. However, that study would require at least 29 weeks for the testing, training, crossover, and washout. A 29-week study, although pragmatically challenging, may also help determine differences in the rate of adaption to RSH compared to the corresponding RSN training.

There is a consensus in the literature that LH training causes an increase in EPO, leading to an increase in red blood cells and hemoglobin mass. The increased hemoglobin mass improves the oxygen-carrying capacity of the blood. As a result, an athlete's maximal oxygen consumption ($VO_{2\ max}$) improves. Furthermore, higher VO_{2max} levels are associated with enhanced performances in middle and distance events (Grégoire P Millet et al., 2010; Paul Robach et al., 2014; Rodríguez et al., 2015; Wachsmuth, Völzke, et al., 2013). This hematological cascade is generally accepted as the underlying mechanism for performance gains associated with the LH training approaches.

Contrastingly, there is a lack of consensus concerning the underlying mechanism for performance gains following RSH training. Currently, improvements in pH regulation (C. Brechbuhl et al., 2018), enhancements within the glycolytic system (Puype et al., 2013), and improved muscle perfusion (Casey & Joyner, 2012; Faiss, Leger, et al., 2013) are being investigated as potential mechanisms (Glaister, 2005). Furthermore, the role of HIF-pathway genes in the adaptive response to RSH training is the focus of ongoing research (Faiss, Leger, et

al., 2013; Nava et al., 2022). Therefore, a logical next step is to assess changes in gene regulation as part of a RSH training study.

In late 2019, the world entered a Pandemic due to COVID-19 the disease caused by the novel SARS-CoV-2 virus. The primary path of transmission is respiratory through droplets. In addition, proximity to the infected person and ventilation are key factors in transmission (Meyerowitz, Richterman, Gandhi, & Sax, 2021). Increased transmission has been reported during heavy exercise in a crowded gym (Jang, Han, & Rhee, 2020). Given this transmission route, how will normobaric hypoxia research, normally conducted in a small altitude chamber, often with multiple heavily exercising participants, be impacted? Modified training systems involving personal masks with inhalation and exhalation controlled by valves to prevent contamination of the common hypoxic source have been proposed and may be a requirement for future studies(Trapé et al., 2021). A second consideration related to COVID-19 concerns training athletes afflicted with long COVID.

Interestingly, intermittent hypoxia training has been proposed as a preventative and rehabilitative non-drug therapy to improve health outcomes related to COVID-19, the infectious disease caused by the SARS-CoV-2 virus (Trapé Á et al., 2021). A decreased risk of infection has been demonstrated in people living at altitude, potentially due to the virus's reduced viability and downregulation of the angiotensin-converting enzyme gene (ACE 2) associated with highlanders (Accinelli & Leon-Abarca, 2020; Arias-Reyes et al., 2020). Moreover, native highlanders exhibit downregulation of molecules in the cyclooxygenase-2 pathway (COX-2) with presumed beneficial effects on resistance to infection from the virus. Applying intermittent hypoxia

training to induce similar adaptions in native lowlanders is currently being investigated as therapeutic target (Supriya et al., 2023) As a result, intermittent hypoxia training has been proposed as rehabilitative therapy for COVID patients (Trapé Á et al., 2021). Investigation into the effectiveness of hypoxic induced changes in gene expression is in the developmental stages but may offer guidance for the clinical applications of the various hypoxic training approaches to the treatment and prevention of COVID-19 (Cai et al., 2021).

RSH training, specifically RSH-VHL which creates hypoxia through voluntary hypoventilation may also be useful in training and protecting the health and safety of competitive swimmers. Recently, the rules in competitive swimming were modified, permitting athletes to kick underwater for up to 12.5 meters in the freestyle, backstroke, and butterfly events. As a result, the ability to remain submerged while kicking at maximum speed (the fifth stroke) has become a necessity. However, without proper training, the fatigue resulting from the tactic is counterproductive during a race. The need to minimize fatigue prompted coaches to add underwater training to workouts. Tragically, several drownings occurred due to shallow water blackouts (Bart & Lau, 2020; Boyd et al., 2015; Pearn, Franklin, & Peden, 2015). As a result, USA Swimming, the governing body of the sport in the USA, issued hypoxic training guidelines. Land-based RSH training poses no risk of drowning and may offer a safer alternative for conditioning swimmers to the physiological demands of prolonged underwater kicking.

Conclusion

Sports scientists, coaches and athletes adopted RSH training as a sprint-based version of Hypoxic training. RSH training was incorporated into the yearly conditioning plan to improve speed and repeated sprint ability. Additional improvements in anaerobic capacity compared to the equivalent RSN training are considered the primary mechanism underlying the hypothesized

additional gains. The exact mechanism for the proposed additional improvements induced by RSH training remains under study. Our findings do not support the hypothesis that RSH training improves swimming performance and anaerobic capacity beyond the corresponding RSN training. However, our data and the data reported in the scientific literature suggest RSH-induced small to moderate additional improvements in athletic performance compared to the equivalent RSN training. Therefore, based on the conflicting evidence recommending the inclusion of RSH training in the yearly conditioning program of male college swimmers is unsupported at this time.

Appendix

Appendix 1 Syracuse University IRB # 16-121

SYRACUSE UNIVERSITY Institutional Review Board

Office of Research Integrity and Protections 121 Bowne Hall, Syracuse, New York 13244-1200 (Phone) 315.443.3013 ♦ (Fax) 315.443.9889 orip@syr.edu ♦ www.orip.syr.edu

MEMORANDUM
TO: Tom Brutsaert
DATE: April 27, 2016
SUBJECT: Expedited Protocol Review - Approval of Human Participants
IRB #: 16-121
TITLE: *Test to Retest Reliability of the Wingate Arm Crank Protocol When Using the VASA Swimming Ergometer*
The above referenced protocol was reviewed by the Syracuse University Institutional Review Board for the Protection of Human Subjects (IRB) and has been given **expedited approval.** The protocol has been determined to be of no more than minimal risk and has been evaluated for the following:
1. the rights and welfare of the individual(s) under investigation;
2. appropriate methods to secure informed consent; and
3. risks and potential benefits of the investigation.

The approval period is **April 27, 2016** through **April 26, 2017**. A continuing review of this protocol must be conducted before the end of this approval period. Although you will receive a request for a continuing renewal approximately 60 days before that date, it is your responsibility to submit the information in sufficient time to allow for review before the approval period ends.
Enclosed are the IRB approved date stamped consent and/or assent document/s related to this study that expire on **April 26, 2017**. **The IRB approved date stamped copy must be duplicated and used when enrolling new participants during the approval period** (may not be applicable for electronic consent or research projects conducted solely for data analysis)**.** Federal regulations require that each participant indicate their willingness to participate through the informed consent process and be provided with a copy of the consent form. Regulations also require that you keep a copy of this document for a minimum of three years after your study is closed.
Any changes to the protocol during the approval period cannot be initiated **prior** to IRB review and approval, except when such changes are essential to eliminate apparent immediate harm to the participants. In this instance, changes must be reported to the IRB within five days. Protocol changes must be submitted on an amendment request form available on the IRB web site. Any unanticipated problems involving risks to subjects or others must be reported to the IRB within 10 working days of occurrence.
Thank you for your cooperation in our shared efforts to assure that the rights and welfare of people participating in research are protected.
Katherine McDonald
IRB Chair
DEPT: Exercise Science, 201 Women's Bldg. **STUDENT:** John Holohan

EXERCISE SCIENCE
820 COMSTOCK AVENUE
201 WOMEN'S BUILDING
SYRACUSE, NY 13210
(315)-443-2114

Test to retest reliability of the Wingate Arm Crank Protocol when using the VASA Swimming Ergometer.

Principal Investigator: Tom Brutsaert, Ph.D.
Telephone: 315-443-9696
Email: tdbrutsa@syr.edu
IRB Protocol #:

We are inviting you to participate in a research study. Involvement in the study is voluntary, so you may choose to participate or not to participate. This sheet will explain the study to you and please feel free to ask questions about the research if you have any. I will be happy to explain anything in detail if you wish.

Purpose

Ergometers have been used to measure work and analyze performance in many sports. For example, in rowing and cycling. One of the benefits of using an Ergometer is that we can accurately measure the amount of work done and relate it to different variables that affect athletic performance. To date, there is no generally approved Ergometer used that simulates the strokes in swimming. This greatly limits the research undertaken in swimming because of the difficulty in collecting performance data while someone is swimming. A recent innovation, the VASA Swimming Ergometer (VASA) has shown promise as both a research and training tool.

The purpose of this study is to determine the value of the VASA in administering a standard sprint test called the Wingate Anaerobic Arm Crank Test (WAAT). Specifically, we need to determine if the test results from tests performed on different days will be similar. In other words, if you take the test on a Monday and on a Wednesday, will your test results be similar? This is called test to retest reliability. Our goal is to determine the test to retest reliability of the VASA using the WAAT. If we find that the VASA is a reliable instrument, our research team and others will be able to use it for research into swimming performance. This will benefit coaches, athletes, and fitness enthusiasts.

Who can participate?

- Men and women between the ages of 18-25

Do I have to participate?

- Your participation in this study is voluntary, which means you get to decide whether or not you want to participate
- Make sure that you read this entire form before making a decision and take as much time as you need.
- Feel free to ask as many questions about the study as you want. If you do not understand a term in the form, ask, and a researcher will explain it for you.
- If you decide to participate in the study you will be asked to sign a consent form.
- Do not sign the consent form until all of your questions have been answered and you understand what will happen in the study.
- Your signature means that you agree to participate in this study.
- You can ask for a copy of this form whether or not you agree to take part in the study.
- Your decision not to be in this research study will not result in any loss of benefits to which you are otherwise entitled.

Can I Withdraw From The Study Once It Has Started?

- At any time you may remove yourself from the study without giving any reason.
- If you are a student, withdrawing from the study will not affect your grade in courses in any way.

What Can I Expect From Participating?

For this study, you will need to visit the Altitude Lab located in the Women's Building at Syracuse University *once* for study screening and *twice* for the exercise test. The screening will take about 30 minutes, the second visit an hour, and the third visit, 30 minutes.

- At the ***initial visit,*** you will be given a copy of the informed consent. We will review the consent form and the study procedures with you. The conditions that would exclude you from the study will be explained. We will take all the time that you need to answer any questions you have about the study or the consent form. You will be given the consent form to take with you. You may review it with anyone you wish. After two days we will contact you to see if you want to participate in the study. If you do want to participate an appointment will be made for a second visit.
- For the ***second visit***, you will be asked again if you have any questions about the informed consent. Once your questions have been answered you will be asked to sign the consent form. Next we will complete the Health History and Activity Questionnaires. We will ask you to arrive not having eaten within the past 1 hour. Your performance on the WAAT can be affected by consuming food, caffeine or alcohol. Therefore, we will ask you to please refrain from exercising or consuming alcohol or caffeine (including caffeinated coffee, tea, soda or energy drinks) on the day that you will come into the lab.

- We will describe the stroking pattern on the VASA and show you a short video on how to use the VASA. You will be given enough time to practice the movement until you are comfortable with the movement.
- You will be asked to sit down and rest for 5 minutes. We will then place a heart rate monitor on your wrist. This monitor will continually measure your heart rate. This is a noninvasive procedure similar to wearing a watch. We will record your resting heart rate.
- After five minutes you will be positioned on the VASA. We will give you a five second countdown. On zero you will be instructed to stroke as quickly as possible for 30 seconds. We will encourage you throughout the test. If at any time during the test you feel the need to stop, please do so immediately. After 30 seconds you will be told to stop and sit quietly for at least 5 minutes or until your heart rate returns to within 10-15 beats of your pre-test resting heart rate. After 15 minutes of rest you will repeat the test.
- After the end the second test, you will sit quietly and be released when your heart rate returns to within 10-15 beats per minute of your pre-test resting heart rate.
- The second visit will take approximately 1 hour.
- The third and final visit will consist solely of a repeat of the exercise testing of day two. This should take approximately 30 minutes.
- If you wish to withdraw from the study at any time you are free to do so.

Can I be excluded from participation for any reason?

- Based on answers to the above mentioned health history and activity questionnaire you may be excluded from the study. If you regularly experience any signs or symptoms that suggest you may have a medical condition and your health care provider is not aware that you are experiencing these symptoms, we will exclude you from the study and ask that you contact your health care provider. We also will exclude you if you have a history of shoulder injury, take medications that affect your heart rate or do not exercise at least three times per week.
- If you are experiencing any signs or symptoms of a serious/significant health condition *at the time of consent* (i.e. severe chest pain, leg pain, dizziness, and feelings of heart palpitations) we will contact emergency medical services immediately and you will not be able to participate in the study.

What Benefits Can I Expect From Participating?

- A benefit from this study is helping us understand if the VASA is a reliable testing instrument.
- You will learn about the research process, which may aid you in your future studies.
- You may feel good about helping others with their research study by participating in this research study.
- You will receive information on your response to an upper body sprint test.
- ***These tests are not being used to diagnose a problem (NOT for medical/clinical purposes). These tests are for research purposes only.***

Are There Any Potential Risks From Participating In This Study?

- There are some risks associated with portions of this study.
- Communicating with the researcher throughout the protocol will reduce risks.
- If at any point you are uncomfortable or feel pain anywhere, please tell us immediately.
- All exercise brings with it some risk of injury and muscle soreness from the exercise protocol. In rare cases, exercise may cause an irregular heartbeat. All exercise sessions will be directly supervised to reduce risk for injury. Ensuring that you communicate with the researching throughout the protocol will reduce risks. If at any point you are uncomfortable or feel pain anywhere, please tell us immediately.
- There may be abnormal changes in heart rate and blood pressure associated with exercise. These abnormal changes are very rare and you will be asked to report any abnormal feelings throughout exercise sessions. We will also instruct you on proper breathing to help reduce this risk. We will monitor your heart rate to minimize your risk.
- In the event of illness or physical injury resulting from taking part in this research study, medical treatment will not be compensated for. You will be responsible for any costs not paid by your insurance company. No other compensation is offered by Syracuse University. You have not waived any of your legal rights by signing this form.

Are There Any Costs?

- There will be no costs to you for participating in this study.

Who Can See Information About This Study?

- ***The research records from this study will be confidential.*** Confidentiality means that it is our responsibility to keep any information you provide private and safe.
- We will conduct the study in the Altitude Lab. While you are present the door will be kept locked from the inside. There is still the possibility that during your test someone may enter the laboratory. We will do our best to prevent this but the possibility does exist.
- Only members of the trained research staff for this study with training in research ethics may look over your research records.
- The paperwork, results and records will be kept in a locked filing cabinet that only the researchers with training in research ethics will have access to.
- You will be given a study identification number (coded numbers, known only by primary researchers) and this will be entered into all research computers used to collect your blood pressure and blood flow. Your name will not appear anywhere on these computers or the data output from these computers.
- All information stored on computers requires a password access it. Only members of the research team with training in research ethics will have this password.
- The data and research record will be stored for up to 10 years.
- ***Your individual results will not be used in any way (we will average all results and display group averages only when presenting findings in papers and presentations)***

What Are My Rights In This Study?

- If at any point you wish to withdraw yourself from the study you may.
- You do not give up any of your legal rights by participating in this study.

Who Can I Contact For Questions Or More Information?

- If there are research related injuries or if you have any questions, concerns, or complaints about this study at any time, please feel free to contact:
  - John Holohan, jdholoha@syr.edu ,315-418-6828
  - Dr. Tom Brutsaert, tdbrutsa@syr.edu, 315-443-9696
- If you have any questions about your rights as a research participant, you have questions, concerns, or complaints that you wish to address to someone other than the investigator, if you cannot reach the investigator, or have experienced research related injuries, contact the Syracuse University Institutional Review Board at 315-443-3013.

By signing below you indicate that you have read and fully understood this informed consent form. You are fully aware of the purpose and procedures of this study as well as the risks, discomforts, and benefits associated with the experimental protocol and that you sign this document freely and voluntarily.

All of my questions have been answered, I am 18 years of age or older, and I wish to participate in this research study. I have received a copy of this consent form.

_____ _____

Signature of participant Date

_____

Printed name of participant

_____ _____

Signature of researcher Date

_____

Printed name of researcher

Syracuse University IRB Approved

APR 27 2016 APR 26 2017

Appendix 2 LeMoyne College IRB # 2016-298

LE MOYNE
SPIRIT. INQUIRY. LEADERSHIP. JESUIT.

INSTITUTIONAL REVIEW BOARD
FOR THE PROTECTION OF HUMAN SUBJECTS
1419 SALT SPRINGS ROAD | SYRACUSE, NY 13214 | WWW.LEMOYNE.EDU

MEMORANDUM

LeMoyne College APPROVED SEP 23 2016 Institutional Review Board

DATE: September 23, 2016

TO: Tom Brutsaert, Ph.D. and John Holohan, M.S.

FROM: Dr. Paul Blackley, Co-chair
Dr. Mary Zampini, Co-Chair

RE: IRB APPROVAL – Form B

Application Number: IRB2016-29B

Proposal Title: Test to retest reliability of the Wingate Arm Crank Protocol when using the VASA Swimming Ergometer

Your application has been reviewed and the study has been approved by the Le Moyne College IRB.

- The appropriate IRB form with required attachments has been presented.
- You may now recruit your subjects and begin to collect your data.
- Be advised of your responsibility to adhere to the approval protocol for the protection of human subjects involved in research projects. Any modifications or additions to the approved procedure must be approved by the IRB before they are implemented. In this case, you must submit a Request To Change An Approved Study, unless the change is immediately necessary in order to eliminate a hazard to a participant. The IRB should be notified immediately in the event of a serious hazard requiring change in the previously approved procedures.
- IRB approval is valid for one year from the date on this memorandum.
- If you intend to continue or resume this project after the current one year approval period, you must submit an Application for Continuing Approval to the IRB at least one month prior to the expiration of this approval.
- Upon the conclusion of your research project, a Project Closure Form must be submitted to the IRB.

The IRB wishes you success as you undertake this research project.

Appendix 3 Syracuse University IRB # 18-138

INSTITUTIONAL REVIEW BOARD MEMORANDUM

TO: Tom Brutsaert

DATE: June 7, 2018

SUBJECT: Expedited Protocol Review - Approval of Human Participants IRB #: 18-138

TITLE: The Effect of Repeated Sprint Training in Hypoxia on Swimming Performance

The above referenced protocol was reviewed by the Syracuse University Institutional Review Board for the Protection of Human Subjects (IRB) and has been given **expedited approval.** The protocol has been determined to be of no more than minimal risk and has been evaluated for the following:

1. the rights and welfare of the individual(s) under investigation;
2. appropriate methods to secure informed consent; and
3. risks and potential benefits of the investigation.

The approval period is **June 6, 2018** through **June 5, 2019**. A continuing review of this protocol must be conducted before the end of this approval period. Although you will receive a request for a continuing renewal approximately 60 days before that date, it is your responsibility to submit the information in sufficient time to allow for review before the approval period ends.

Enclosed are the IRB approved date stamped consent and/or assent document/s related to this study that expire on June 5, 2019. The IRB approved date stamped copy must be duplicated and used when enrolling new participants during the approval period (may not be applicable for electronic consent or research projects conducted solely for data analysis). Federal regulations require that each participant indicate their willingness to participate through the informed consent process and be provided with a copy of the consent form. Regulations also require that you keep a copy of this document for a minimum of three years after your study is closed.

Any changes to the protocol during the approval period cannot be initiated **prior** to IRB review and approval, except when such changes are essential to eliminate apparent immediate harm to the participants. In this instance, changes must be reported to the IRB within five days. Protocol changes must be submitted on an amendment request form available on the IRB web site. Any unanticipated problems involving risks to subjects or others must be reported to the IRB within 10 working days of occurrence.

Thank you for your cooperation in our shared efforts to assure that the rights and welfare of people participating in research are protected.

Katherine McDonald IRB Chair

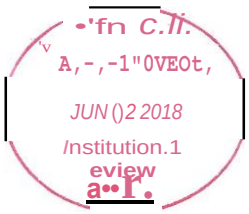
DEPT: Exercise Science, 201 Women's Bldg. **STUDENT:** John Holohan

Appendix 4 LeMoyne College IRB # 2018-218

LEMOYNE
SPIRIT. INQUIRY. LEADERSHIP. JESUIT.

INSTITUTIONAL REVIEW BOARD
FOR THE PROTECTION OF HUMAN SUBJECTS
1419 SALT SPRINGS ROAD, SYRACUSE, NY 13214 | WWW.LEMOYNE.EDU

MEMORANDUM



DATE: June 2, 2018

TO: Tom Brutsaert Ph.D. and John Holohan Ph.D. candidate

FROM: Dr. Paul Blackley, Co-chair

RE: **IRB APPROVAL-** Form B

Application Number: IRB2018-218

Proposal Title: The Effect of Repeated Sprint Training in Hypoxia on Swimming Performance

Your application has been reviewed and the study has been approved by the IRB. Regulations require that you keep a copy of this document for a minimum of three years after your study is closed.

- The appropriate IRB form with required attachments has been presented.
- You may now recruit your subjects and begin to collect your data.
- Be advised of your responsibility to adhere to the approval protocol for the protection of human subjects involved in research projects. **The approved date-stamped copy of your consent forms must be duplicated and used when enrolling new participants during the approval period** (may not be applicable for electronic consent or research projects conducted solely for data analysis). Each subject must sign the consent form and be provided with a copy for their records.
- Any modifications or additions to the approved procedure must be approved by the IRB before they are implemented. In this case, you must submit a Request To Change An Approved Study, unless the change is immediately necessary in order to eliminate a hazard to a participant. The IRB should be notified immediately if a serious hazard requires a change in the previously approved procedures. Any other unanticipated problems involving risks to subjects or others must be reported promptly to the IRB.
- If you intend to continue or resume this project after the current one year approval period, **June 2, 2018** through **June 1, 2019,** you must submit an Application for Continuing Approval to the IRB at least one month prior to the expiration date.
- Upon the conclusion of your research project, a Project Closure Form must be submitted to the IRB.

The IRB wishes you success as you undertake this research project.

Appendix 5 Informed Consent

SYRACUSE UNIVERSITY
SCHOOL OF EDUCATION
DEPARTMENT OF EXERCISE SCIENCE

INFORMED CONSENT

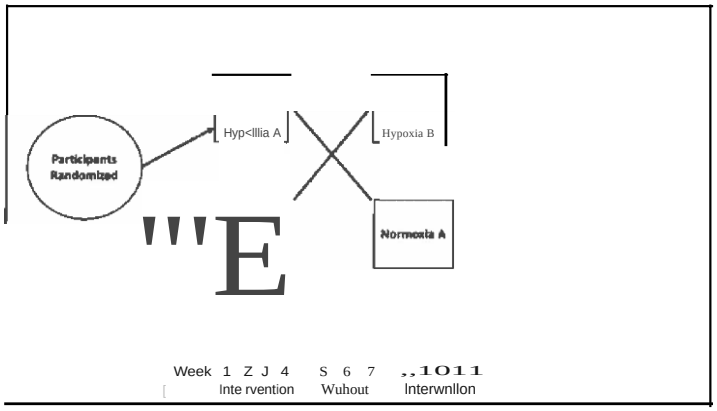
Effect of Repeated Sprints in Hypoxia Train ing on Swimming Performance

My name is John Holohan and I am a Ph.D. candidate in Exercise Science. Tom Brutsaert Ph.D., current Chair of the Department of Exercise Science at Syracuse University, is the principal investigator for this study. You are being invited to participate in a research study. Involvement in the study is voluntary, so you may choose whether or not to participate. This sheet will explain the study to you. Please feel free to ask questions about the research, if you have any. I will be happy to explain anything in further detail if you wish.

Purpose

Exercise training at elevation, where the amount of oxygen in the air is reduced (known as "hypoxia training) has been shown to improve athletic performance in some events. This has been especially true for endurance-based sports. Recently, researchers have been investigating the effect of hypoxia training on sprint performance using a repeated sprint training program in hypoxia. This is accomplished using an altitu de chamber where the concentrat ion of oxygen in the air is reduced as a way to mimic training at elevation. Although hypoxia train ing has been studied in compet itive college swimmers, repeated sprint t raining has not.

The purpose of this study is to assess the value of repeated sprints in hypoxia training on swimming performance. The trainab ility of the arms compared to the legs will also be investigated. To do this, both the arms and the legs will be tested on an all-out 30 second sprint test called the Wingate-bike (WaNT-bike) and the Wingate-swim (WAAT-swim) test. Then you will complete a four-week training program before completing both tests again. After each of the tests, a very small sample of blood will be collected from your finger using a fi nger-stick. The training will be conducted twice per week for the four weeks. Each training session should take approximately one hour. After the first four-week training intervention, you will take three weeks off and then return to complete another four-week repeated sprint training program. For one of the four-week blocks you will train in hypoxia (H) for the other you will train in normal air **(N).** You will not be told which condit ion you are exercising in. This is called a single-blind crossover design and is depicted in the diagram following this paragraph. This design will help us analyze the effectiveness of hypoxia training. If the resu lts indicate that training improves swimming performance, the information may benefit swimmers, coaches, and fitness enthusiasts.



Who can part icipate?

Male swimmers between the ages of 18-25 who are current members of their college swimming team. They must train at least 5 times per week and their school must be located within 50 miles of Syracuse University.

Can I be excluded from the study for any reason?

Yes, you can be excluded from the study for any of the following condition s. tf you are not a member of a college swimming team. If your school is located more than SO miles from Syracuse University. If you do not train year-round . If you do not tr ain at least 5 times per week. You may be excluded based on the answers to the health history and the activity questionnaire. If you regularly experience any signs or symptoms that suggest you may have a medical condition, and your health care provider is not aware that you are experiencing these symptoms, we will exclude you from the study and ask that you contact your health care provider. You will also be excluded if you have a history of shoulder injury, shoulder surgery within the last year, take medications that can affect your heart rate, or have known cardiovascular disease.

If you are experiencing any signs or symptoms of a serious/significant health condition *at the time* of *the consent* (i.e. severe chest pains, leg pain, dizziness, and feeling of heart palpitations) I will contact emergency medical services immediately and you will not be able to participate in the study.

Do I have to **participate?**

- Your participation in this study is voluntary, which means you get to decide whether or not you want to participate.
- Make sure that you read this ent ire form before deciding and take as much time as you need.

- Feel free to ask as many questions about the study as you want. If you do not understand a term in the form, ask, and a researcher will explain it to you.
- If you decide that you do want to participate in the study, you will be asked to sign a consent form.
- Your signature means that you agree to participate in the study.
- Do not sign the consent form until all your questions have been answered and you understand fully what to expect from the study.
- You can ask for a copy of this form whether or not you choose to participate in the study.
- Your decision not to be in this research study will not result in any loss of benefits to which you are otherwise entitled.

Can I withdraw from the study once it has started?

- At any time, you may remove yourself from the study without penalty without giving any reason.
- If you are a student, withdrawing from the study will not affect your grade in courses in any way.
- If you are a student-athlete, withdrawing from the study will not affect your team status in any way.

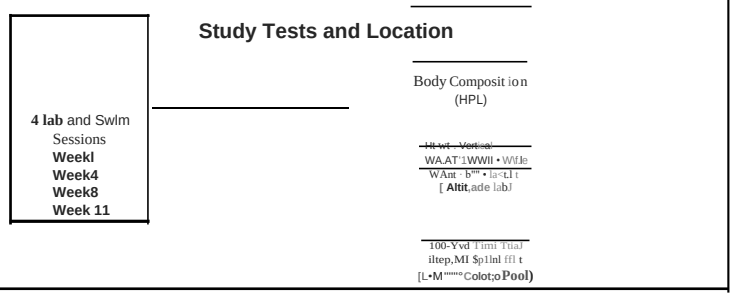
What can I expect from participation.

- Prior to your enrollment in the study, a general information and screening session will be offered at the LeMoyne College Swimming Pool. If you volunteer for this study, you will need to visit the Altitude Lab located in the Women's Building at Syracuse University, eight times for each of the four-week trainings, and four times in total for testing. Measurements of your body composition will take place in the Human Performance Lab located a floor above the Altitude Lab. You will also participate in 100-yard time trials and a repeated sprint test in your primary stroke at the LeMoyne CollegePool, where you normally practice, on four separate occasions.

- The *screening and general information session at the leMoyne Pool* wilt take about 30 minutes. Each lab visit will take approximately 1 hour. Each *training session* will take 45 minutes. The *time trials* at the LeMoyne College pool will take approximately 30 minutes.

- At the **screening session,** you will be given a copy of the informed consent form. I will review the consent form and the study procedures with you. Any conditions that might exclude you from the study will be explained. If you meet any of these conditions, you will not be able to participate in the study. I will take all the time that you need to answer any questions that you have about the study or the consent form. You may take the consent form with you when you leave and review it with anyone you wish. After two days, I will contact you to see if you want to participate in the study. If you do want to participate, an appointment will be made for your first lab visit.

- For **the *lab visit*.** Any remaining questions you may have will be answered. If you agree to participate you will sign the consent form. After you sign, the study will begin that day. Prior to the visit, if you know that you want to participate, please arrive not having eaten within the past 3 hours. As your performance on the WaNT-bike and WAAT-swim can be affected by food, alcohol, or caffeine. Please refrain from consuming any alcohol or caffeine (including caffeinated coffee, tea, soda, or energy drinks) on the day you come to the lab. During this first lab visit you will complete the Health History and Activity Questionnaires. Your height, weight, and body composition will be recorded. Using a simple jump test, your maximal jumping height (vertical jump) will be determined, and you will do the WAAT-swim and WAnT-bike tests. These procedures will be repeated at the end of the first four weeks of training, the beginning and end of the second four-week training session. The order of the data collection procedure and physical tests are depicted below and described in the following section.



- Before you perform any physical tests your **body composition** will be evaluated using the Bod Pod. As previously stated you will need to refrain from food or exercise for 3 hours prior to the Bod Pod procedure. In addition, please don't use any creams or lotions that day. You will be asked to remove your glasses and any removable jewelry. You will then be asked to sit in the Pod wearing your bathing suit and a swim cap, like what you wear when competing. The actual test takes about 3 minutes. During that time, the door to the Bod Pod will be opened and closed several times. You are always able to open the door to the Bod Pod on you own, however, if you are claustrophobic you will not participate in this test.

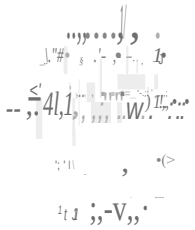
- After the body composition test you will go back downstairs to the Altitude Lab. Your height and weight will be recorded. You will perform the vertical jump test (described below). After the

jump test, you will be asked to sit down and rest for 5 minutes. A heart rate monitor will be placed on your chest. This monitor will continuously measure your heart rate. This is a noninvasive procedure very similar to wrapping an ace bandage around your chest. Your resting heart rate and blood pressure will be recorded at the beginning and end of each Wingate test.

- Before the Wingate tests you will be asked to perform a **vertical jump test.** This test provides information about the type of muscle fiber that you have in your lower body. Your fingers on their right hand will be marked with colored chalk. Standing flat footed, you will reach up and touch a wall on your right with your right hand at the highest possible point. Next you will complete a maximal jump by flexing your legs to no more than 90 degrees then jumping and touching the wall at the highest possible point. This test will be repeated three times with one-minute rest between jumps. Vertical jump score will be the highest score achieved.

- After five minutes of quiet sitting, I will record your heart rate and blood pressure. Prior to taking the **WAAT-swim** you will be positioned on the swimming ergometer. You will receive instructions on how to use the ergometer and allowed to practice. Once you are comfortable with the stroking technique, you will be allowed to warm-up for three minutes using an easy stroking rate, about half speed. Toward the end of the warm-up, you will be alerted that the test is about to begin and given a 10-second countdown. During the countdown, you will be told to increase your stroke rate to close to maximal speed. On zero you will be instructed to stroke as quickly as possible, moving through the full range of the pulling motion. You will be encouraged throughout the test. If at any time during the test you feel the need to stop, please do so immediately. After 30 seconds, you will be told to stop and sit quietly for six minutes. To access the amount of lactate that you generated during the test, at the six-minute mark a fingertip prick will be used to obtain a very small sample of blood from the tip of your finger. This will help estimate your anaerobic or sprint capacity. You will feel a small pinch. After, your finger will be cleaned with alcohol swab and will you hold the swab in place until the blood clots. You will be released from the test after your heart rate returns to within 10-15 beats per minute of your resting heart rate.

- After the WaNT-swim you will be given 15 minutes rest. After 15 minutes your heart rate and blood pressure will be taken. Then you will complete the WaNT-bike test. You will be positioned on the bike and the handle bars and seat height adjusted to your comfort level. You will receive instruction on how to use the bike and will be allowed to practice until you are comfortable with the movement. Once comfortable you will complete a 5-minute warmup at 75 watts, a very light workload. After 5 minutes you will pedal for 20 seconds more, followed by a 6 second phase where you will increase your pedaling speed to your fastest rate. After 6 seconds a load will be added to the bike based on your body weight. You will continue to pedal as fast as you are able for the next 30 seconds. I will encourage you throughout the test. If at any time during the test you feel the need to stop, please do so immediately. After 30 seconds have passed you



will be told to stop and sit quietly for six minutes. At the six-minute mark a fingertip prick will be used to obtain a very small sample of blood from the tip of your finger. You will feel a small pinch. After an alcohol swab will be used to clean the area. You will hold the swab in place for a few minutes. You will be released from the test after your heart rate returns to within 10-15 beats per minute of your resti ng heart rate. The order that you take the WAAT-swim and the WaNT-bike will be randomly determined. Therefore, you may take either test first. Regardless of test order the procedure will remain the same and you will receive a 15-minute rest between tests.

- The above testing procedure will be completed at the beginning of the study, the end of your first 4-week training session, and at the beginning and the end of the second four-week training session. Each lab visit should take approximately 1 hour.

- Each ***training session*** will take approximately 1 hour. All training sessions will take place in the altitude chamber at the Syracuse University Altitude lab. You will complete two, four-week training programs, with a 3-week break between the first and second four-week segments. During one of the segments you will exercise in normal air and in the other in reduced oxygen, equivalent to what you would feel at 3000 meters of elevation. You will not be told which condition that you are training in. You will conduct 2 sets of 8, 20 seconds all-out sprints on the bike with 40 seconds rest between sprints and 2 minutes between sets. Following 5 minutes rest you will complete 2 sets of 8, 20 second all-out sprints on the swimming ergometer. You will be given 5 minutes rest between the bike and swim workouts. Throughout the workout your heart rate and the amount of oxygen in your blood will be monitored. The order of the bike and swim workouts will be randomly determined. The level of intensity of the workout will be like a sprint swimming practice. You may stop the workout at any time.

- **Time trial procedure:** Time trials will be conducted at the leMoyne College Swimming Pool, in a meet format, using electronic timing that records performance to the nearest .001 of a second. After a 15-minute warm-up at approximat ely a comfortable pace, you will do 4 xS0 on 1 minute. You will increase your speed to approximately 90% of full speed over the 4 fifties, similar to how you would warm-up for a meet. Next you will complete an easy 200-yard swim at about half your top speed. Fina lly, you will be allowed to complete two full speed sprints from the blocks. After 10 minutes rest you will perform a 100-yard time trial in your primary stroke. After the t ime-t rial you will be complete a 10 -minute recovery swim at self-selected pace.

- **Repeated** sprint test: A validated repeated spr int swimming test consisting of 6 x 15- meter sprints on 17 seconds will be used in this study. Testing will be conducted at the leMoyne College Swimming Pool. A competition grade electronic timing system will be used. After a 15-minute warm-up at a self -selectedcomfortable pace, you will do 4 x50 yard swims on 1 minute.

You will increase your speed to approximately 90% of full speed over the four fifties, similar to how you would warm-up for a meet. Next you will complete an easy 200 swim. Finally, you will be allowed to complete two full speed sprints from the blocks. You will perform the repeated

sprint test in an outside lane. The start point will be clearly marked. Between repeats, you will tread water with your head on the start line. A recorded start cadence will be used (1, 2, 3 followed by an electronic beep) to standardize the take-off. On the beep, you will sprint 15 meters, time will be recorded when your head passes over the finish line. You will repeat a sprint every 17 seconds until six are completed. Following the six sprints, you will perform at 10-minute cooldown swim at a pace of your choosing If you wish to withdraw from the study, you may do so at any time.

What benefits can I expect from participation?

- You will help us understand if sprint training in hypoxia benefits swimming performance.
- You will learn about the research process, which may aid you in your future studies.
- You may feel good about helping others with their research study by participating in this research study.
- You will receive information about your sprint ability which may benefit your future swimming training.
- ***These tests are not to be used to diagnose a problem (NOT for medical/clinical purposes). These tests are for research purposes only.***

Are There Any Potential Risks From Participating In This Study?

- There are some risks associated with portions of this study. ***Communicating with the researchers throughout the protocol will reduce these risks.***
- All exercise brings with it some risk of injury to muscle and joints including soreness, strains, and irritation from the exercise protocol. In rare cases, exercise may cause an irregular heartbeat, nausea, dizziness, and potentially passing out. All exercise sessions will be directly supervised to reduce risk for injury. Again, ensuring that you communicate with the researchers, about how you feel, throughout the protocol will reduce risks. *If at any point you are uncomfortable or feel pain anywhere, please tell us immediately.*

- With intense exercise there is a very slight risk (1 in 100,000) of a cardiac incident. Some abnormal changes in heart rate and blood pressure can be associated with exercise. These abnormal changes are very rare, and you will be asked to report any abnormal feelings throughout exercise sessions. To minimize your risk, you will be instructed on proper breathing technique and your heart rate monitored.
- In the event of illness or physical injury resulting from taking part in this research study, medical treatment will not be compensated for. You will be responsible for any costs not paid by your insurance company. No other compensation is offered by Syracuse University. You have not waived any of your legal rights by signing this form.

Are There Any Costs?

- There will be no costs to you for participating in this study.

Who Can See Information About This Study?

- ***The research records from this study will be confidential.*** Confidentiality means that it is our responsibility to keep any information you provide private and safe.
- The study will be conducted in the Altitude Laboratory at Syracuse University. While you are present, the door will be kept locked from the inside. There is still the possibility that during your test someone may enter the laboratory. Precautions will be taken to prevent this, but the possibility does exist.
- Only John Holohan and Tom Brutsaret will have access to information that identifies you.
- The paperwork, results, and records will be kept in a locked filing cabinet that only John Holohan and Tom Brutsaert, both with training in research ethics, will have access to.
- You will be given a study identification number (coded numbers, known only by primary researchers) and this will be entered into all research computers used to collect your blood pressure and blood flow. Your name will not appear anywhere on these computers or the data output from these computers.
- All information stored on computers requires a password access it. Only John Holohan and Tom Brutsaert will have access to these records.
- The data and research record will be stored for up to 10 years.
- ***Your individual results will not be used in any way (all results will be averaged and only group averages will be displayed when presenting findings in papers and presentations)***

What Are My Rights In This Study?

- If at any point you wish to withdraw yourself from the study, you may.

SYRACUSE UNIVERSITY
SCHOOL OF EDUCATION
DEPARTMENT OF EXERCISE SCIENCE

Syracuse University IRB Approved
JUN 6-2018 JUN 5-2019

- You do not give up any of your legal rights by participating in this study.

Who Can I Contact For Questions Or More Information?

- If there are research related injuries or if you have any questions, concerns, or complaints about this study at any time, please feel free to contact:
  - John Holohan, jdholoha@syr.edu ,315-418-6828
  - Dr. Tom Brutsaert, tdbrutsa@syr.edu, 315-443-9696

- If you have any questions about your rights as a research participant, you have questions, concerns, or complaints that you wish to address to someone other than the investigator, if you cannot reach the investigator, or have experienced research related injuries, contact the Syracuse University Institutional Review Board at 315-443-3013.

By signing below, you indicate that you have read and fully understood this informed consent form. You are fully aware of the purpose and procedures of this study as well as the risks, discomforts, and benefits associated with the experimental protocol and that you sign this document freely and voluntarily.

All of my questions have been answered, I am 18 years of age or older, and I wish to participate in this research study. I have received a copy of this consent form.

_____ _____

Signature of participant Date

_____

Printed name of participant

_____ _____

Signature of researcher Date

_____

Printed name of researcher

Appendix 6 ACSM Health Status & History Questionnaire

This form includes several questions regarding your physical health – please answer every question as accurately as possible. Please ask us if you have any questions. Your responses will be treated in a confidential manner.

PERSONAL INFORMATION

Last Name:__First Name:__Gender: F M Mobile:_____Email: _____

Date of Birth_/_____/_____ Height_____ Weight _____

YES NO (ACSM HEALTH SCREEN)

- ☐ ☐ Do you have any personal history of heart disease (coronary or atherosclerotic disease)?
- ☐ ☐ Any personal history of diabetes or other metabolic disease (thyroid,renal,liver)?
- ☐ ☐ Any personal history of pulmonary disease, asthma, interstitial lung disease or cystic fibrosis?
- ☐ ☐ Have you experienced pain or discomfort in your chest apparently due to blood flow deficiency?
- ☐ ☐ Any unaccustomed shortness of breath *p*erhaps during light exercise)?
- ☐ ☐ Have you had any problems with dizziness or fainting?
- ☐ ☐ Do you have difficulty breathing while standing or sudden breathing problems at night?
- ☐ ☐ Have you experienced a rapid throbbing or fluttering of the heart?
- ☐ ☐ Do you suffer from ankle edema (swelling of the ankles)?

☐ ☐ Have you experienced severe pain in leg muscles during walking?

☐ ☐ Do you have a known heart murmur?

☐ ☐ Has your serum cholesterol been measured at greater than 200 mg/dl?

☐ ☐ Are you a cigarette smoker?

☐ ☐ Has your HDL (the "good" cholesterol) been measured at greater than 60 mg/dl?

☐ ☐ Would you characterise your lifestyle as "sedentary"?

☐ ☐ Have you had a high fasting blood glucose level on 2 or more occasions (>=110mg/dl)?

☐ ☐ Are you 20% or more overweight or have you been told your “BMI” was greater than 30?

☐ ☐ Have you been assessed as hypertensive on at least 2 occasions (systolic > 140mmHg or diastolic > 90mmHg)?

☐ ☐ Do you have any family history of cardiac or pulmonary disease prior to age 55?

MEDICAL HISTORY

☐ Are you currently being treated for high blood pressure?

If you know your average blood pressure, please enter:__/_____

Please check all conditions or diagnoses that apply:

- ☐ Abnormal EKG?
- ☐ Abnormal Chest X-Ray?
- ☐ Limited Range of Motion?
- ☐ Arthritis?
- ☐ Stroke?
- ☐ Do You Suffer from Epilepsy or Seizures?

☐ Rheumatic Fever? ☐ Bursitis? ☐ Chronic Headaches or Migraines?

☐ Low Blood Pressure? ☐ Swollen or Painful Joints? ☐ Persistent Fatigue?

☐ Asthma? ☐ Foot Problems? ☐ Stomach Problems?

☐ Bronchitis? ☐ Knee Problems? ☐ Hernia?

☐ Emphysema? ☐ Back Problems? ☐ Anemia?

☐ Other Lung Problems? ☐ Shoulder Problems? ☐ Are You Pregnant?

☐ Recently Broken Bones?

☐ Has a doctor imposed any activity restrictions? If so, please describe:

_____

_____

_____

_____

FAMILY HISTORY

Have your mother, father, or siblings suffered from *p*lease select all that apply):

☐ Heart attack or surgery prior to age 55. ☐ High cholesterol

☐ Stroke prior to age 50. ☐ Diabetes

☐ Congenital heart disease or left ventricular hypertrophy.

☐ Obesity

☐ Hypertension ☐ Asthma

☐ Leukemia or cancer prior to age 60. ☐ Osteoporosis

MEDICATIONS

Please Select Any Medications You Are Currently Using:

☐ Diuretics	☐ Other Cardiovascular
☐ Beta Blockers	☐ NSAIDS/Anti-inflammatories (Motrin, Advil)
☐ Vasodilators	☐ Cholesterol
☐ Alpha Blockers	☐ Diabetes/Insulin
☐ Calcium Channel Blockers	☐ Other Drugs (record below).

Please list the specific medications that you currently take:

_____

☐ Are you a cigarette smoker? If so, how many per day? _____

☐ Previously a cigarette smoker? If so, when did you quit? ___

How many years have you smoked or did you smoke before quitting? _

Do you/did you smoke (Circle one): Cigarettes Cigars Pipe Please Rate Your Daily Stress Levels (select one):

	☐ Low	☐ Moderate but I enjoy the challenge	☐ High	☐ High: sometimes difficult to handle	☐ High: often difficult to handle.

☐ Do you drink alcoholic beverages?

How many units of alcohol do you consume per week: ___

(see Alcohol Units Chart)

Alcohol Units Table

Type of Drink	Units
½ pint of beer	1
1 glass of wine	1
1 pub measure of spirits (Gin, Vodka etc.)	1
1 can of beer	1.5
1 bottle of strong lager	2.5
1 can of strong lager	4
1 bottle of wine	7
1 litre bottle of wine	10
1 bottle of fortified wine *p*ort, sherry etc.)	14
1 bottle of spirits	30

Dietary Habits. Please Select All That Apply.

☐ I seldom consume red or high-fat meats. ☐ I eat at least 5 servings of fruits/vegetables per day.

☐ I pursue a low-fat diet. ☐ I almost always eat a full, healthy breakfast.

☐ My diet includes many high-fiber foods. ☐ I rarely eat high-sugar or high-fat desserts.

[redacted]

Please indicate any other medical conditions or activity restrictions that you may have, or any other information you feel is critical to understanding your readiness for exercise. It is important that this information be as accurate and complete as possible

Please indicate your personal health and fitness-related goals (select all that apply):

☐ Cardiovascular Fitness	☐ Injury Rehab	☐ Muscular Strength
☐ Feel Better	☐ Look Better	☐ Reduce Stress
☐ General Fitness	☐ Lose Weight	☐ Reduce Back Pain
☐ Improve Diet	☐ Lower Cholesterol/Blood Pressure	☐ Sport-Specific Training
☐ Improve Flexibility	☐ Muscular Size	☐ Stop Smoking

Please tell us a little about your exercise patterns and goals: What is your exercise history?

What health improvements do you need?

What are your activity preferences?

What barriers to success do you anticipate?

How will you know that you are succeeding?

What is your *motivation* level?

What is your *confidence* level?

□ High□ Medium □ Low □ High□ Medium □ Low

I verify that all of the completed information is correct to the best of my knowledge. I declare that I am participating voluntarily in a performance fitness test. The maximum exertion during the test is at my discretion and I understand that I can stop the test at any time. I declare that I have no medical problems that prevent me from undertaking the fitness

test and that I am not currently taking any medication that could present a danger with the performance fitness test.

Printed Name Signature Date

_____ _____

Emergency Contact: Mobile: ____

Appendix 7 International Physical Activity Questionnaire

(August 2002)
SHORT LAST 7 DAYS SELF-ADMINISTERED FORMAT
FOR USE WITH YOUNG AND MIDDLE-AGED ADULTS (15-69 years)

The International Physical Activity Questionnaires (IPAQ) comprises a set of 4 questionnaires. Long (5 activity domains asked independently) and short (4 generic items) versions for use by either telephone or self-administered methods are available. The purpose of the questionnaires is to provide common instruments that can be used to obtain internationally comparable data on health–related physical activity.

Background on IPAQ

The development of an international measure for physical activity commenced in Geneva in 1998 and was followed by extensive reliability and validity testing undertaken across 12 countries (14 sites) during 2000. The final results suggest that these measures have acceptable measurement properties for use in many settings and in different languages and are suitable for national population-based prevalence studies of participation in physical activity.

Using IPAQ

Use of the IPAQ instruments for monitoring and research purposes is encouraged. It is recommended that no changes be made to the order or wording of the questions as this will affect the psychometric properties of the instruments.

Translation from English and Cultural Adaptation

Translation from English is supported to facilitate worldwide use of IPAQ. Information on the availability of IPAQ in different languages can be obtained at www.ipaq.ki.se. If a new translation is undertaken, we highly recommend using the prescribed back translation methods available on the IPAQ website. If possible, please consider making your translated version of IPAQ available to others by contributing it to the IPAQ website. Further details on translation and cultural adaptation can be downloaded from the website.

Further Developments of IPAQ

International collaboration on IPAQ is on-going and an ***International Physical Activity Prevalence Study*** is in progress. For further information see the IPAQ website.

More Information

More detailed information on the IPAQ process and the research methods used in the development of IPAQ instruments is available at www.ipaq.ki.se and Booth, M.L. (2000). *Assessment of Physical Activity: An International Perspective*. Research Quarterly for Exercise and Sport, 71 (2): s114-20. Other scientific publications and presentations on the use of IPAQ are summarized on the website.

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an

active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

_____ days per week

☐ No vigorous physical activities → ***Skip to question 3***

2. How much time did you usually spend doing **vigorous** physical activities on one of those days?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

____

days per week

☐ No moderate physical activities → ***Skip to question 5***

4. How much time did you usually spend doing **moderate** physical activities on one of those days?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

_____ days per week

☐ No walking → Skip to question 7

6. How much time did you usually spend **walking** on one of those days?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

This is the end of the questionnaire.

Appendix 8 RSH Data Sheets

Participant ID_____

Informed Consent Date_____

Wingate Tests

Pre-test 1 Date_____

WAnT-Swimwim

Condition _____ Resting HR_____ Resting BP_____ Age_____ Wt._____ Ht._____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

WAnt-bike

Condition_____Resting HR_____ Resting BP_____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

Post-test 1 Date_____

WAnT-Swim

Condition _____ Resting HR_____ Resting BP_____ Age_____ Wt._____ Ht._____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

WAnt-bike

Condition_____Resting HR_____ Resting BP_____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

Pre-test 2 Date_____

WAnT-Swim

Condition _____ Resting HR_____ Resting BP_____ Age_____ Wt._____ Ht._____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

WAnt-bike

Condition_____Resting HR_____ Resting BP_____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max LA_____

Post-test 2 Date_____

WAnT-Swim

Condition _____ Resting HR_____ Resting BP_____ Age_____ Wt._____ Ht._____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max

LA_____

WAnt-bike

Condition_____Resting HR_____ Resting BP_____

PP_____ MP_____ LP_____RF_____ Borg _____Max HR_____ BP_____ Max

LA_____

RSH study swimming performance data sheet

Participant ID_____

Informed Consent Date _____

Repeated Sprint Test: Date_____

Age_____ Ht_____ Wt_____

Repetition	Date	Pre 1	Date	Post 1	Date	Pre 2	Date	Post 2
1								
2								
3								
4								
5								
6								
Total Time (TT)								
Decrement (D)								
Ideal Time (I)								
% Decrement (%D)								

TT= sum of all reps, I= fastest timex6, D= TT-I, %D= (TT/I x 100)-100

Time Trial Primary Stroke, 100-yards

Pre-1 Date_____ Age_____ Ht_____ Wt_____

Pre-1 time_____

Post-1 Date_____ Age_____ Ht_____ Wt_____

Post- 1 time_____

Pre-2 Date_____ Age_____ Ht_____ Wt_____

Pre-1 time_____

Post-2 Date_____ Age_____ Ht_____ Wt_____

Pre-1 time_____

RSH study vertical jump test datasheet

Subject ID_____

Informed Consent Date _____

Post-1 Date_____ Age_____ Ht_____ Wt_____

Vertical Jump: Ht 1_____ Ht 2 _____ Ht 3_____ VT x mass_____

Post-1 Date_____ Age_____ Ht_____ Wt_____

Vertical Jump: Ht 1_____ Ht 2 _____ Ht 3_____ VT x mass_____

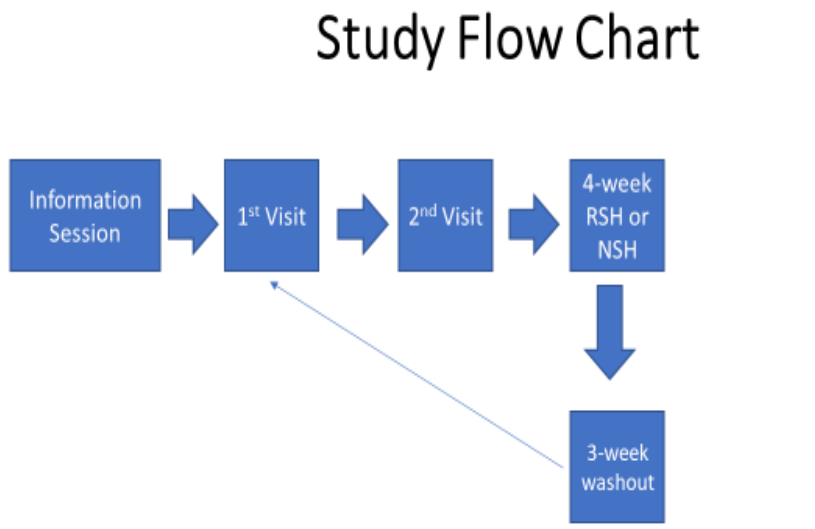
Pre-2 Date_____ Age_____ Ht_____ Wt_____

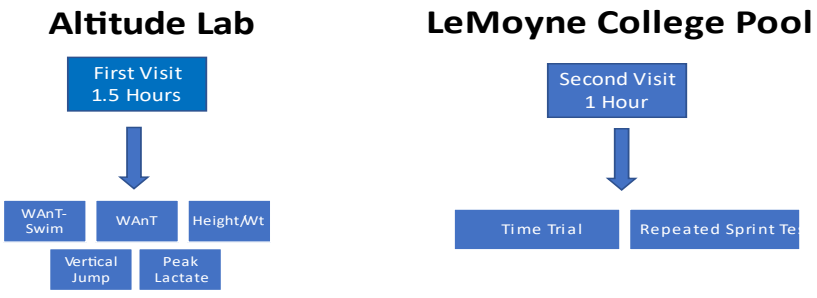
Vertical Jump: Ht 1_____ Ht 2 _____ Ht 3_____ VT x mass_____

Post-2 Date_____ Age_____ Ht_____ Wt_____

Vertical Jump: Ht 1_____ Ht 2 _____ Ht 3_____ VT x mass_____

Appendix 9 Study Flow Chart





Appendix 10 ANOVA Table Chapter III

ANOVA Table for PP

		Sum of Squares	df	Mean Square	F	Sig
Between People		121076.42	9.00	13452.94		
Within People	Between Items	1667.75	3.00	555.92	1.68	0.19

ANOVA table for MP

		Sum of Squares	df	Mean Square	F	Sig
Between People		103821.033	9	11535.670		
Within People	Between Items	1065.758	3	355.253	1.733	0.184

ANOVA Table for LP

		Sum of Squares	df	Mean Square	F	Sig
Between People		84008.761	9	9334.307		
Within People	Between Items	1131.859	3	377.286	1.946	0.146

ANOVA Table for FI

		Sum of Squares	df	Mean Square	F	Sig
Between People		1427.282	9	158.587		
Within People	Between Items	8.953	3	2.984	0.054	0.983

Note. PP=peak power; MP= mean power; LP= low power; Fi= fatigue index; Sig= p value

Appendix 4 ANOVA Table Chapter IVANCOVA table for the between subject effects for the Dependent Variables in Chapter IV.

Tests of Between-Subjects Effects

Dependent Variable: Change Peak LA WAnT-Swim

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	158.179[a]	2	79.089	23.023	<.001	.687
Intercept	134.781	1	134.781	39.235	<.001	.651
Pre-Peak LA WAnT-Swim	157.977	1	157.977	45.987	<.001	.687
HYPvsNORM	.330	1	.330	.096	.760	.005
Error	72.140	21	3.435			
Total	231.200	24				
Corrected Total	230.318	23				

Note. R Squared = .687 (Adjusted R Squared = .657): WAnT-Swim = Wingate Anaerobic Test on the swimming ergometer; Pre-Peal LA = peak lactate after the pretraining WAnT-Swim.

Tests of Between-Subjects Effects

Dependent Variable: Change Peak LA WAnT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	85.330[a]	2	42.665	19.367	<.001	.648
Intercept	93.407	1	93.407	42.400	<.001	.669
Pre-Peak LA WAnT	85.090	1	85.090	38.624	<.001	.648
HYPvsNORM	5.255	1	5.255	2.386	.137	.102
Error	46.263	21	2.203			
Total	142.800	24				
Corrected Total	131.593	23				

Note. R Squared = .648 (Adjusted R Squared = .615); WAnT = Wingate Anaerobic Capacity Test on the bicycle ergometer; Pre-Peak LA = peak lactate after the pretraining WAnT.

Tests of Between-Subjects Effects

Dependent Variable: Change in Peak Power WAnT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	53803.614[a]	2	26901.807	4.685	.021	.309
Intercept	39298.221	1	39298.221	6.844	.016	.246
Pre-WAnT PP	28453.614	1	28453.614	4.955	.037	.191
HYPvsNORM	12219.975	1	12219.975	2.128	.159	.092
Error	120589.719	21	5742.368			
Total	227786.000	24				
Corrected Total	174393.333	23				

Note. R Squared = .309 (Adjusted R Squared = .243); WAnT= Wingate Anaerobic Capacity Test on the bicycle ergometer; Pre-WAnT PP = pre-training peak power score on the WAnT.

Tests of Between-Subjects Effects

Dependent Variable: Change in MP WAnT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	5495.790[a]	2	2747.895	.726	.496	.065
Intercept	6463.024	1	6463.024	1.708	.205	.075
Pre-WAnT MP	4069.749	1	4069.749	1.075	.312	.049
HYPvsNORM	2048.272	1	2048.272	.541	.470	.025
Error	79471.168	21	3784.341			
Total	104177.000	24				
Corrected Total	84966.958	23				

Note. R Squared = .065 (Adjusted R Squared = -.024); WAnT= Wingate Anaerobic Capacity Test on the bicycle ergometer; Pre-WAnT= MP = pre-training mean power score on the WAnT.

Tests of Between-Subjects Effects

Dependent Variable: Change in LP WAnT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	45011.869[a]	2	22505.935	14.043	<.001	.572
Intercept	54069.456	1	54069.456	33.737	<.001	.616
Pre-WAnT LP	44948.494	1	44948.494	28.046	<.001	.572
HYPvsNORM	476.460	1	476.460	.297	.591	.014
Error	33656.089	21	1602.671			
Total	95575.000	24				
Corrected Total	78667.958	23				

Note. R Squared = .572 (Adjusted R Squared = .531); WAnT= Wingate Anaerobic Capacity Test on the bicycle ergometer; Pre-WAnT LP = pre training low power score on the WAnT.

Tests of Between-Subjects Effects

Dependent Variable: Change in Fi WAnT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	1861.757[a]	2	930.879	18.396	<.001	.637
Intercept	1531.916	1	1531.916	30.273	<.001	.590
Pre-WAnT Fi	1665.103	1	1665.103	32.905	<.001	.610
HYPvsNORM	86.566	1	86.566	1.711	.205	.075
Error	1062.662	21	50.603			
Total	2940.590	24				
Corrected Total	2924.420	23				

Note. R Squared = .637 (Adjusted R Squared = .602) WAnT= Wingate Anaerobic Capacity Test on the bicycle ergometer; Pre-WAnT Fi = pre training Fi score on the WAnT.

Tests of Between-Subjects Effects

Dependent Variable: Change in PP WAnT-Swim

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	38141.178[a]	2	19070.589	20.709	<.001	.664
Intercept	66856.955	1	66856.955	72.601	<.001	.776
PreWAnTSPP	38129.136	1	38129.136	41.405	<.001	.663
HYPvsNORM	44.534	1	44.534	.048	.828	.002
Error	19338.447	21	920.878			
Total	131739.000	24				
Corrected Total	57479.625	23				

Tests of Between-Subjects Effects

Dependent Variable: Change in MP WAnT-Swim

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	24479.220[a]	2	12239.610	16.798	<.001	.615
Intercept	42004.475	1	42004.475	57.647	<.001	.733
PreWAnTSMP	24245.469	1	24245.469	33.274	<.001	.613
HYPvsNORM	25.304	1	25.304	.035	.854	.002
Error	15301.740	21	728.654			
Total	84062.410	24				
Corrected Total	39780.960	23				

Note. WAnT-Swim = Wingate anaerobic capacity test on a swimming ergometer; Pre-WAnt-Swim MP = pre training MP score on the WAnT-Swim

Note. WAnT-Swim = Wingate anaerobic capacity test on a swimming ergometer; Pre-WAnt-Swim PP = pre training PP score on the WAnT-Swim

Tests of Between-Subjects Effects

Dependent Variable: Change in WAnT-Swim Fi

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	643.075[a]	2	321.538	4.569	.023	.303
Intercept	509.591	1	509.591	7.241	.014	.256
PreWAnTSFI	608.275	1	608.275	8.643	.008	.292
HYPvsNORM	18.734	1	18.734	.266	.611	.013
Error	1477.941	21	70.378			
Total	2138.870	24				
Corrected Total	2121.016	23				

Note. WAnT-Swim = Wingate anaerobic capacity test on a swimming ergometer; Pre-WAnt-Swim Fi = pre training Fi score on the WAnT-Swim; R Squared = .303 (Adjusted R Squared = .237)

Tests of Between-Subjects Effects

Dependent Variable: Change in LP WAnT-Swim

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	19418.251[a]	2	9709.125	11.776	<.001	.529
Intercept	36563.435	1	36563.435	44.348	<.001	.679
PreWAnTSLP	19177.584	1	19177.584	23.261	<.001	.526
HYPvsNORM	269.617	1	269.617	.327	.573	.015
Error	17313.749	21	824.464			
Total	77076.000	24				
Corrected Total	36732.000	23				

Note. R Squared = .529 (Adjusted R Squared = .484); WAnT-Swim = Wingate Anaerobic Capacity Test on the swimming ergometer; Pre-WAnT-Swim LP = pretraining low power score on the WAnT-Swim.

Tests of Between-Subjects Effects

Dependent Variable: Change in Time Trial

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	9.779[a]	2	4.890	3.136	.064	.230
Intercept	4.296	1	4.296	2.755	.112	.116
PreTIMETRIAL	4.710	1	4.710	3.021	.097	.126
HYPvsNORM	4.901	1	4.901	3.143	.091	.130
Error	32.745	21	1.559			
Total	43.003	24				
Corrected Total	42.524	23				

Note. a. R Squared = .230 (Adjusted R Squared = .157); Time trial = 100 yard time trial in primary stroke

Tests of Between-Subjects Effects

Dependent Variable: Change in repeated Sprint Ideal Time (RSIT)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	74.722[a]	2	37.361	3.091	.067	.227
Intercept	60.606	1	60.606	5.014	.036	.193
PreRSIT	71.142	1	71.142	5.885	.024	.219
HYPvsNORM	1.762	1	1.762	.146	.706	.007
Error	253.857	21	12.088			
Total	354.601	24				
Corrected Total	328.580	23				

Note. a. R Squared = .227 (Adjusted R Squared = .154); Pre-RSIT = repeated sprint ideal time (fastest time x 6)

Tests of Between-Subjects Effects

Dependent Variable: Change in Repeated Sprint Average Decrement (RSAD)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	133.255[a]	2	66.627	17.606	<.001	.626
Intercept	82.148	1	82.148	21.708	<.001	.508
PretrainingRSAD	133.214	1	133.214	35.202	<.001	.626
HYPvsNORM	.060	1	.060	.016	.901	.001
Error	79.470	21	3.784			
Total	242.920	24				
Corrected Total	212.725	23				

Note. R Squared = .626 (Adjusted R Squared = .591); Pre-RSAD = the pretraining repeated sprint average decrement (total time-ideal time).

Tests of Between-Subjects Effects

Dependent Variable: Change in Repeated Sprint Relative Percent Decrement (RSRD)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	413.802[a]	2	206.901	17.775	<.001	.629
Intercept	293.309	1	293.309	25.198	<.001	.545
PretrainingRSRD	413.466	1	413.466	35.520	<.001	.628
HYPvsNORM	1.958	1	1.958	.168	.686	.008
Error	244.446	21	11.640			
Total	706.812	24				
Corrected Total	658.248	23				

Note. R Squared = .629 (Adjusted R Squared = .593) Pre-RSRD = pretraining relative percent decrement (total time/ideal time x 100)-100.

Tests of Between-Subjects Effects

Dependent Variable: Change in Repeated Sprint Total Time

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	183.224[a]	2	91.612	13.577	<.001	.564
Intercept	142.377	1	142.377	21.100	<.001	.501
Pre-RSTT	178.847	1	178.847	26.505	<.001	.558
HYPvsNORM	1.870	1	1.870	.277	.604	.013
Error	141.702	21	6.748			
Total	437.203	24				
Corrected Total	324.926	23				

Note. R Squared = .564 (Adjusted R Squared = .522); Pre-RSTT= pretraining repeated sprint total time (sum of the six sprints).

Appendix 12 Power Analysis Chapter IV

Table 4 1: Observed η_p^2, *p* value, effect sizes, and required sample size to detect significant effects of Hypoxia over Normoxia at a power of 0.80

Hypoxia vs Normoxia	*p* value	Partial eta squared	Effect Size	Achieved Power	N
WAnT PP	0.159	0.092	0.318	0.685	16
WAnT MP	0.470	0.025	0.160	0.205	56
WAnT LP	0.591	0.014	0.119	0.130	98
WAnT FI	0.205	0.075	0.285	0.579	20
WAnT Peak Lactate	0.137	0.102	0.337	0.740	14
WAnT-S PP	0.828	0.002	0.044	0.059	682
WAnT-S MP	0.854	0.002	0.044	0.059	682
WAnT-S LP	0.573	0.015	0.123	0.136	92
WAnT-S FI	0.611	0.013	0.115	0.124	106
WAnT-S Peak Lactate	0.760	0.005	0.071	0.076	274
100-Time Trial	0.091	0.130	0.386	0.858	12
RS Ideal Time	0.760	0.007	0.084	0.087	196
RS Average Decrement	0.901	0.001	0.032	0.055	1364
RS Rate of Decline	0.686	0.008	0.089	0.093	172
RS Total Time	0.640	0.013	0.115	0.124	106

Note. WAnT = Wingate anaerobic capacity test on the bike; WAnT-Swim – Wingate anaerobic capacity on the swimming ergometer; PP = peak power score on the Wingate test; MP = mean power score on the Wingate test; LP =low power score on the Wingate test; FI = the fatigue index score on the Wingate; RS = the repeated sprint swim test; RS ideal time = the fastest time x 6; RS average decrement = repeated sprint average decrement (total time-ideal time); RS rate of decline = relative percent decrement (total time/ideal time x 100)-100; RS total time = sum of the six sprints; η_p^2 (partial eta squared) = a measure of effect size; N = sample size

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Appendix 14 Curriculum Vitae

Curriculum Vitae

John David Holohan

Contact Information: John D. Holohan
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Syracuse, NY 13224
Ph. 315-418-6828
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Education:

Syracuse University, Syracuse, New York,
School of Education
Ph.D. Candidate Science Education

While completing the dissertation portion of my doctoral program, I taught three to nine credit hours per semester for the Exercise Science Department at Syracuse University. Courses ranged from Health Sciences to Motor Behavior. In the Fall of 2019, I taught the Introduction to Exercise Science for Majors Course with five T.A.'s and over 70 students. Throughout my studies at S.U., I have mentored five students via the research experience credit program.

While planning and implementing my research, I refined my grant writing, data management, statistical analysis, and manuscript preparation skills. My strengths include developing the research question, IRB relationships, participant recruitment, and study execution. Currently, two manuscripts are in review for publication.

Research emphasis:
· Repeated sprints n hypoxia and performance in college swimmers
· The reliability of a swimming ergometer for the administration of the Wingate Anaerobic Test.
· Physiologic correlates to elite swimming performance in female adolescent swimmers
· The ACE gene insertion/deletion polymorphism and endurance performance
· Gene expression changes and cognition in Alzheimer's patients following 12 weeks of Aerobic Exercise
· Obesity and exercise in a beta-amyloid mouse model
· Olfactory function and exercise in an Alzheimer's disease mouse model

Cornell University, Ithaca, New York
The Johnson School- 6 graduate credits (Marketing and Executive Leadership)
2008-2009

Syracuse University, Syracuse, New York
School of Education
M.S. In Exercise Science
Thesis: Physiologic Correlates to Swimming Performance in Elite 12-16-Year-Old Female Swimmers. As a T.A., I taught Exercise Science labs and Aquatics.
2002-2005

LeMoyne College, Syracuse, New York
Graduate Business School- 3 credits (Entrepreneurship)
2001- 2001

Instructor and Coaching Related Employment:

Non-Tenure Track Faculty- Syracuse University, Syracuse NY
Courses:
Sole Instructor for Introduction to Exercise Science with Recitation (3 credits, one semester)
Managed five graduate assistants as part of the course.
Motor Behavior across the Lifespan (3 credits, three semesters) with one T.A.
Sports in Education (3 credits, one semester)
Health Science for Coaching (3 credits seven semesters)
Recitation, Introduction to Exercise Science (seven semesters).
October 2009-Present

Non-Tenure Track Faculty - Onondaga Community College, Syracuse NY
Courses: Health and Wellness (4 sections)
Strength Training for Life (3 sections)
Team Sports with emphasis on Pedagogy (2 credits)
Semester teaching load, 6-10 credits per semester
Five Year Service Award
September 2009-present

Exercise Physiologist/ Clinical Fitness Program- Upstate Medical University, Syracuse, NY. Developed and instructed water-based fitness programs for clinical illness and injury participants, ranging from orthopedic problems to Parkinson's disease.
November 2009-June 28, 2013.

Research Coordinator- Upstate Medical University, Syracuse, NY. Coordinate an NIA-funded study investigating the effect of exercise on gene expression and cognition in Alzheimer's disease. My specific tasks included monitoring protocol implementation for IRB compliance, subject exercise training, and maintenance of all subject records, HIPAA compliance, and coordination with our surgeons relative to biopsy procedures. August 2010-present.

Graduate Teaching and Research Assistant- Syracuse University, Syracuse NY
I am certified to teach the NYS Coaching certification curriculum in Sports Psychology and Exercise Science.
Developed the coursework and taught Allied Health Sciences for Coaches *p*PE 300). I also taught the following: PPE 447, Exercise Physiology Lab Section, PPE 295 Introduction to Exercise- Recitation, PPE 400 Biomechanics.
September 2009-present

Non-Tenure Track Faculty, Syracuse University, Aquatic Instructor- Beginning, intermediate and advanced aquatics courses, Syracuse University, Syracuse NY, Spring 2002, 2003

Head Coach - Cornell University Women's Swimming, Ithaca, NY

I was hired to rebuild an Ivy League Division I Women's Swimming and Diving Program. During my six-season tenure, 85% of the athletes had lifetime best performances while maintaining a team GPA of 3.2 or higher. Team accomplishments included: the first NCAA B cut time for women, two individual Ivy League champions, numerous school records, USA national and Olympic trial qualifications. For the student-athletes, balancing academic, athletic, and life demands was challenging. We developed a "Fuel for Performance Program," flexible practice schedules, and an online stroke refinement program that used underwater filming and Dartfish to help them thrive. I also worked in consultation with the University Counseling Center. The result was a healthier team culture. In 2009, four student-athletes qualified for the prestigious Cornell 400 Club (GPA of 4.0). That same year, the team's 3.44 GPA resulted in qualification to the Academic All-American Team. They were first in the Ivy League and 14th in the Nation. The Cornell Community is a vibrant academic environment. The intellectual growth of the young women I coached inspired my return to graduate studies.

Head Coach/Owner -The Sharks/Aquafit, Syracuse, NY
Founder, owner, and head coach of a USA Swimming Club with 150 athletes who ranged from 8-24 years of age. I was responsible for all aspects of club management, including hiring, supervision, and

training of 5-7 assistant coaches, parent communications, planning workouts, and conducting 25 hours of practice per week. I developed programs for all of our athletes and coached the Senior Athletes along with the advanced level of age group swimmers. Accomplishments included: Five world rankings, over 50 National Top 16 performances, 20 Junior National Qualifiers, 5 Senior National Qualifiers, 3 Olympic Trial Qualifiers, 2 Olympians (2000, 2020).
1988-2003

Assistant Swimming Coach- LeMoyne College, Syracuse NY
Coached the sprint group for Men's and Women's Swimming
1991-1992

Head Coach- Liverpool High School Boy's Swimming & Diving, Liverpool NY
Turned a faltering team around in one year going from 5-6 to 10-1
1991-1993.

Head Coach- Baldwinsville High School Girl's Swimming & Diving
The team broke all but two school records and went 27-6.
1992-1996

Assistant Swimming Coach, Men's and Women's Swimming- Syracuse University
Responsible for speed training, strength training, and recruiting
1980-1981

Head Coach- Water Polo**,** Syracuse University, Coach, Syracuse, NY
As a player-coach, I led the team to its highest Eastern league finish in school history (3rd).

Head Coach-Syracuse Water Polo Age Group Program (Men's and Women's). Women qualified for Nationals.

Head Coach- The Liverpool Jets Age Group Swimming Team. I developed numerous State, Regional, and National qualifiers.
1974-1976

Primary Business Employment:

Consultant InSourcing Inc. Assisted in the conceptualization, organization, completion of government applications, and implementation of a 401-C corporation. InSourcing provided training opportunities for the development of skilled labor in the manufacturing sector.
2007-2009

Stockbroker- Smith Barney, San Diego CA
Second Vice President, I was licensed in stocks, insurance, and foreign currencies.
1986-1987

Stockbroker- Prudential Bache Securities, Syracuse NY
Licensed broker (Series 7), Insurance and Foreign Currency
I developed a sales book with over 800 clients.
1980-1986

National Fitness Director/Special Assistant to the President, Sports Illustrated Court Clubs of America, Southfield MI.
I repositioned the firm as a fitness business through a mass marketing campaign and a redesign of club operations and programming. We installed the program into 22 clubs with an average increase in Net Income of 30%. I was promoted to Special Assistant to the President in charge of a 10-million-dollar turnaround project called Hamilton Place. I managed a million-dollar advertising campaign that resulted in a five-fold increase in income.
1977-1980

Secondary Employment

Aquatic Director-Onondaga Golf and Country Club, Fayetteville NY
Direct the summer operations of a country club recreational swimming program and competitive team.
1987-current

Personal Coach- Precision Coaching, Develop and implement wellness and athletic training programs for individual clients, includes technique analysis using Dartfish.
2000-current

Pool Management- Syracuse, NY
I was the owner-operator of a pool management company. Operated, staffed, and managed five pools in the Syracuse area.
1987-2000

Fitness Consulting-Pro-Fit Inc., Syracuse NY and Southfield MI
Designed, programmed, and staffed fitness centers. Repositioned Racquetball and Tennis Clubs from sport-specific businesses to fitness facilities.
1980-1983

Additional Teaching Experience

Guest Lecturer, Cornell University School of Human Ecology, Sports Nutrition
Repeat guest lecturer on the topic of Holistic Coaching of College Athletes.
2004-2009

Individual Retreat Guide, Spiritual Renewal Center, Syracuse NY
I guided individuals in the nine months long, non-denominational, 19th Annotation Retreat.
1995-2001

Graduate Assistant, Syracuse University, Syracuse NY
Water Polo head coach, highest Big East finish (3rd).
1975-1976

Volunteer Work:

Member of the Assembly of Syracuse University School of Education

Syracuse University Whitman School, liaison between MBA students enrolled in a course focused on developing workarounds for disabled entrepreneurs.

Spiritual Renewal Center, Syracuse, NY member of the financial planning committee

Publications/Presentations

MARC Conference 2012/PowerPoint Presentation/First Author: *"Muscle gene expression in Alzheimer's disease following 12 weeks of exercise". Holohan J., Hassan M., Brangman S., Middleton F., Keslacy S.*

Experimental Biology Conference 2011/Poster Presentation/First Author, *"The effect of exercise on olfactory function in a mouse model of Alzheimer's disease" Holohan J., Heckstell E, Ogaye K., Lloyd J.,*
Keslacy K.

Skeletal muscle: Missing link between exercise and NF-kappaB pathway regulation in Alzheimer's Disease? *Lloyd J., Kelleher A., Heckstall E., Holohan J., Keslacy K. 2010*

Guest Lecture, Tompkins County Eating Disorder Group, "*One Coaches Perspective on Eating Disorders in Female College Athletes," November* 2009

Second Author for my master's thesis, *"Aerobic Cost in Elite Female Adolescent Swimmers,"* in the March 2009 issue of the International Journal of Sports Medicine" Unnithan *V., Holohan J., Fernhall B., Wylegala J., Rowland T., Pendergast D.*

Presenter/First Author 2004, FINA World Sports Medicine Conference "*Physiologic Correlates to Swimming Performance in Elite 12-16-Year-Old Female Swimmers". Holohan J., Fernhall B., Wylegala J, Rowland T., Pendergast D., Unnithan V.*

Presenter 2004, NASPEM Annual Conference, Poster Presentation, "*Physiologic Correlates to Swimming Performance in Elite 12-16-Year-Old Female Swimmers". Holohan J., Fernhall B., Wylegala J, Rowland T., Pendergast D., Unnithan V.*

Article, ASCA Newsletter**,** *"Innovative Uses of the Vasa Trainer,"* John Holohan 1999

Grants:

CAPS Grant through the Maxwell School and NIH: With my advisor, I co-authored an NIH pilot grant application. We were awarded approximately $100,000.00 to investigate the molecular mechanism underlying the exercise-induced improvements in cognition in patients with Alzheimer's disease.
May 2011-present.

Certifications and Related Experience:

Blackboard
Blackboard Ultra
SPSS
CITI Certification, Human Subject Research

CPR
First Aid
Former ACSM Cardiac Graded Exercise Technician
Dartfish Software, movement analysis
ASCA Level 4 Certified Coach
First Team All-East Water Polo Selection
Anonymous Swimming Award, Syracuse University
Member, National Sports Festival Water Polo Team
Participant UC Irvine Water Polo
Coach Participant, USA Swimming National Swimming Camp
Coach Participant, USA Swimming National Team
Coach Participant USA Swimming Olympic Trials 1996, 2000