

THESIS

A SINGLE HIGH-ALTITUDE TRAINING BOUT IMPROVES HIGH-ALTITUDE
AEROBIC PERFORMANCE FOLLOWING ONE WEEK OF
LOW-ALTITUDE TRAINING

Submitted by

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ABSTRACT

A SINGLE HIGH-ALTITUDE TRAINING BOUT IMPROVES HIGH-ALTITUDE AEROBIC PERFORMANCE FOLLOWING ONE WEEK OF LOW-ALTITUDE TRAINING

Consecutive bouts of aerobic exercise at high-altitude are known to improve subsequent aerobic exercise performance at high altitude due to a variety of acute and chronic adaptations referred to as altitude acclimatization. However, it is unclear whether these benefits can be elicited by a single bout of high-altitude exercise followed by several days of training at lower altitude. **PURPOSE:** We investigated whether a single bout of hill running exercise performed at high-altitude improves running performance, arterial oxygen saturation (SaO_2), hematocrit (HCT) and perceived exertion (RPE) during a strenuous timed run at high-altitude performed 1 week following the training stimulus. **METHODS:** Participants were well-trained competitive runners (VO_2 max 52 ± 5 ml/kg/min, aged 42 ± 14 yrs, $n = 8$) living in Fort Collins, CO (5,003 ft) that were naïve to higher altitudes for at least 8 weeks. All were training regularly (66 ± 3 miles/wk) and refrained from any non-prescribed altitude exposure for the duration of the study. Baseline testing consisted of a timed run on the Mt. Evans Scenic Byway in Colorado

(11.5 miles at 11,500-14,100 ft) followed by one week of training in Fort Collins. Runners were then randomized to perform a 12 mile hill running workout in Fort Collins (LOW; 5,550-7,170 ft, n = 4) or Mt. Evans (HIGH; 12,750-14,100 ft, n = 4). All runners then engaged in 1 week of routine training in Fort Collins followed by a post-test run at Mt. Evans that was identical to the baseline test. **RESULTS:** There were no significant differences in run-time from pre to post-test for either group. Pre-run (11,500 ft) percent change in SaO₂ was significantly different between groups ($+3.70 \pm 2.95\%$ in High; $-3.95 \pm 1.62\%$ in Low, $P = 0.029$). The high group experienced a $4.01 \pm 2.72\%$ mean increase in exercising SaO₂ from pre to post-test, while the Low group decreased $1.21 \pm 1.41\%$ from pre to post-test ($P = 0.029$). In addition, the High group experience a $8.45 \pm 8.13\%$ increase in recovery SaO₂ from pre to post-test at an elevation of 14,100 feet, while the Low group had a $2.21 \pm 2.82\%$ decrease in recovery SaO₂ from pre to post-test at an elevation of 14,100 feet ($P = 0.057$). HCT increased from pre post-test to post post-test in the Low group only ($43.55 \pm 1.04\%$ & $45.67 \pm 0.79\%$ respectively; $P = 0.68$). There were no differences in HR or RPE within or between High and Low groups.

CONCLUSION: A single high-altitude training bout prior to 1 week of low-altitude training improves subsequent aerobic performance and arterial O₂ saturation at high-altitude, while a single bout performed 2 weeks prior to testing is ineffective.

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CHAPTER I

INTRODUCTION

High altitude trail running has become an increasingly popular sport in Colorado and around the United States. The sport challenges athletes to perform at extremely high aerobic exercise intensities in moderate to high altitude conditions which pose extreme demands on our physiology. The stressors placed on athletes who participate in high altitude trail running greatly exceed those that are experienced in flat races and races at sea-level. The Pikes Peak Marathon is an annual race held in August which epitomizes the sport of high altitude trail running. The Pikes Peak Marathon is the third oldest marathon in the United States, dating back to 1966 (Simpson 2004). Starting at an elevation of 6,300 feet and finishing at the 14,115 foot summit, the Pikes Peak Marathon features a 7,815 vertical foot elevation gain. Year in and year out, athletes come from across the globe to compete in The Pikes Peak Marathon to pursue “America’s Ultimate Challenge”.

Several training regimes have been implemented in an effort to optimally prepare for a high altitude endurance event such as The Pikes Peak Marathon. Optimal aerobic performance at high altitude is thought to require multiple days to weeks of high-altitude exposure prior to the event for full acclimatization, but this is not feasible for most

athletes living at lower altitudes. It is presently unclear whether shorter acclimatization protocols, such as a single high-altitude workout performed the weekend before an event, are sufficient to elicit a sustained physiological response and improve subsequent high-altitude running performance. Members of the Fort Collins Trail Runners (FCTR) and other local trail runners routinely engage in weekend training runs on the Barr Trail of Pikes Peak during the 4-6 week period prior to The Pikes Peak Marathon in hopes of optimizing their preparation for the event in August. One training routine that they implement is called a “3-2-1” workout, developed and practiced by the Incline Club in Manitou Springs, CO. This routine consists of an initial three mile descent from the summit of the Barr Trail and immediate ascent back to the summit as the “3” leg, followed by a two mile descent and immediate ascent to the summit as the “2” leg, and a final one mile descent and immediate ascent to the summit as the “1” leg. While several members feel that these weekly training bouts are useful, they require many hours of travel between Fort Collins and Colorado Springs for 2-3 hours of training, and conclusive evidence of their performance enhancing effects is lacking.

The present study aims to investigate the physiological (SaO_2 , HR & Hct) and performance (run-time & RPE) effects of a single high altitude training bout (“3-2-1”) performed on the Mount Evans Scenic Byway (12,750-14,100 feet) one week before a timed max effort high altitude run compared to a similar “3-2-1” training bout performed at Horsetooth Mountain Park in Fort Collins (5,550-7,170 feet).

Statement of the Problem

It is presently unclear whether a single “3-2-1” high altitude training bout performed one week before a high altitude event elicits physiological and performance enhancing effects that exceed those of a “3-2-1” training bout performed at lower altitude.

Hypothesis

Implementation of the “3-2-1” training routine at high altitude (Mount Evans Scenic Byway outside of Idaho Springs, Colorado) one week before a timed high altitude running event will elicit physiological acclimatization and enhance performance superior to that of a “3-2-1” training routine performed at lower altitude (Horsetooth Mountain Park in Fort Collins, Colorado).

Delimitations, Limitations and Assumptions

The present study is delimited to 18-65 year old competitive trail runners residing in Fort Collins, Colorado. All subjects are healthy with a minimum VO_2 max of 45 ml/kg/min. A sample size of eight (four per group) would be considered a limitation of the study which minimizes the statistical power and ability to extrapolate findings to a broad population of trail runners. This study is a field study conducted at Mount Evans and Horsetooth Mountain Park, which poses another limitation as the weather conditions are out of the researchers control during test-runs. Failure to collect data during the first attempted test-run at Mount Evans due to weather conditions led to scheduling conflicts which resulted in a mismatch in mean VO_2 max between HIGH and LOW altitude groups, creating another significant limitation. We assume that all subjects were well

rested and fully hydrated during each test-run, and that each subject trained/prepared for each test run equally.

CHAPTER II

LITERATURE REVIEW

Trail runners have been exploring multiple different training methods in preparation for the Pikes Peak Marathon for many years. Many local trail runners travel to Pikes Peak multiple for weekend training runs prior to the marathon in an attempt to acclimatize for enhanced performance during the race. However, it is unclear whether these weekly training bouts superior to similar training routines performed at lower altitudes such as Horsetooth Mountain Park in Fort Collins, Colorado. The following review will identify and discuss the physiological stressors faced by endurance athletes at altitude, and previous evidence of various strategies employed to optimally prepare for an endurance event at altitude.

Physiological Challenges Imposed at Altitude

Aerobic exercise relies on adequate delivery of oxygen (O_2) to mitochondria in working muscle for oxidative metabolism (Calbet & Lundby, 2009). Consequently, maximal aerobic capacity is often defined as an individual's maximal rate of oxygen consumption (VO_2 max), which reflects the maximal rate of oxidative phosphorylation of ADP by body tissues. Therefore, optimal aerobic exercise requires matching tissue O_2 demands with O_2 supply by the cardiopulmonary system. Acute and chronic altitude exposures impose physiological challenges that significantly impair aerobic performance

by depriving working muscle of O₂ required for oxidative metabolism. The mechanisms and implications of these changes are discussed briefly below.

Hypobaria, Hypoxia and Hypoxemia

Barometric pressure decreases as altitude increases, leading to a reduced partial pressure (PO₂) of oxygen at higher elevations (Green, 2000). Low barometric pressure environments are considered to be “hypobaric”. Figure 1 describes how the lower partial pressure of inspired air (P_IO₂) at altitude lead to lower partial pressures of oxygen in the alveoli (P_AO₂), arterial blood (P_aO₂) and venous blood (P_vO₂) effectively reducing the PO₂ gradient that drives O₂ diffusion from atmospheric air to mitochondria in working muscle (Fulco, Rock, & Cymerman, 1998).

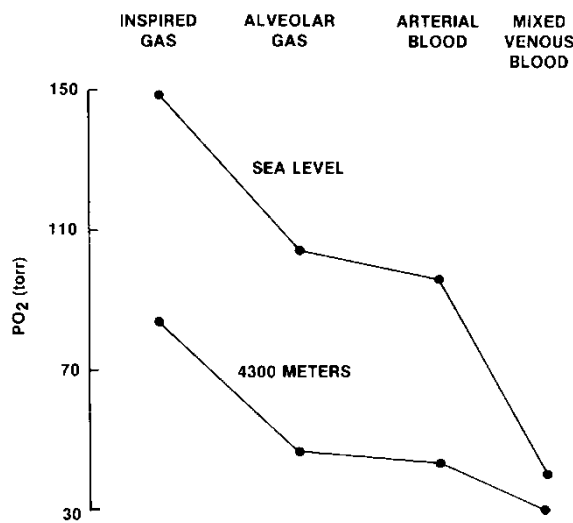


Figure 1: Differences in PO₂ at sea level and altitude (4,300 meters)

Although the fractional concentration of oxygen (F_IO₂) remains the same as at sea level, 20.93%, the reduced PO₂ results in less oxygen available at altitude. The decrease in partial pressure of oxygen in the blood or tissues is defined as “hypoxia”. “Hypoxemia”

is another, more specific term related to altitude, which refers to the decrease in oxygen content of arterial blood (CaO_2) (*Hypoxia and Exercise*, 2006).

Altitude Illness

Altitude may also lead to various forms of illness with increased exposure. The most common form of altitude illness is termed “Acute Mountain Sickness” (AMS). The most common symptoms of AMS include headache, fatigue, nausea, dizziness and vomiting. Onset and severity of symptoms are dependent upon individual variation, speed of ascent and elevation attained (West, 1981). High-altitude cerebral edema (HACE) is a more serious form of altitude illness. HACE is associated with filling of fluid in the head which impedes blood flow to the brain (West, 1981). Symptoms include drowsiness, confusion, discoordination and possible loss of consciousness. Onset of HACE typically occurs five days after rapid ascents above 9,000 feet. High-altitude pulmonary edema (HAPE) is similar to HACE, but occurs in the lungs which leads to a greater degree of hypoxia due to the inability to properly ventilate and transfer oxygen into the pulmonary capillaries (West, 1981). Symptoms include hyperventilation, rapid heart rate and dyspnea. It is well known that altitude acclimatization is the most effective strategy in preventing these various forms of altitude illness (Muza, Beidleman, & Fulco).

Acute Adaptations to Altitude

Cardiovascular

There are many acute physiological adaptations experienced with the exposure to altitude. With a decrease in CaO_2 , cardiac output (Q) must increase in order to maintain oxygen delivery. Cardiac output is defined as the product of heart rate and stroke

volume. With acute exposure to altitude, stroke volume remains the same or decreases slightly, requiring an increase in heart rate to maintain cardiac output. In addition, sympathetic drive of blood flow is increased at altitude. Acute adaptations of heart rate, stroke volume and cardiac output are provided in figure 2(a), 2(b) and 2(c) respectively (George A. Brooks, 1996).

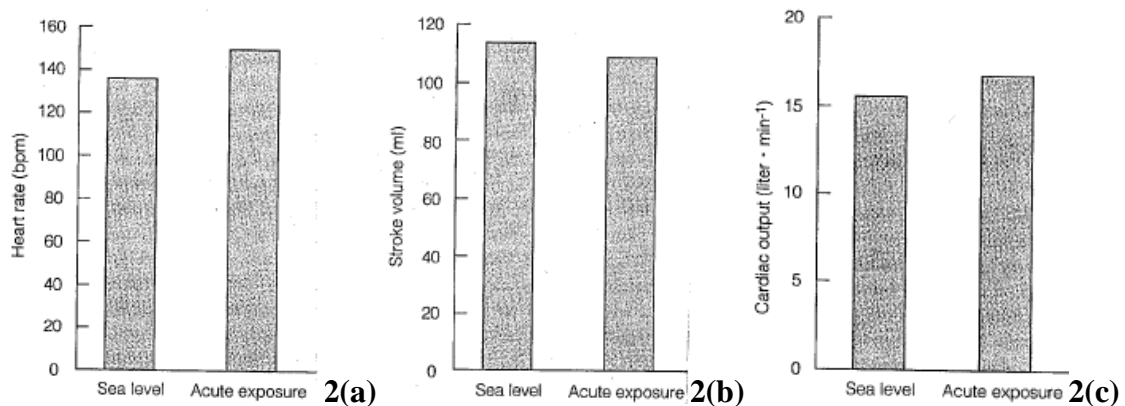


Figure 2: Cardiovascular adaptations following acute altitude exposure
2(a): Heart rate (bpm) **2(b):** Stroke volume (ml) **2(c):** Cardiac output (L/min)

The increase in heart rate will place a greater physiological strain on the heart muscle. An increase in cardiac output is evident during both rest and moderate exercise at altitude. A decrease in plasma volume will also result in an increase in red blood cell and hemoglobin concentration which corresponds with a larger diffusion area within the blood (Boning). Altitude exposure can also stimulate erythropoiesis which contributes to an increase in red blood cell concentration (Rodriguez et al., 2000). The net effect of acute and chronic increases in hemoglobin (Hb) is an increased O₂ carrying capacity of the blood.

$\dot{V}O_2 \text{ max}$

The state of hypoxia induced by altitude exposure steadily decreases hemoglobin saturation with increasing elevation. It is estimated that maximal oxygen consumption ($\dot{V}O_2 \text{ max}$) is reduced by 15% at an altitude of 2300 m, and will steadily decrease with increasing altitude (Drust & Waterhouse). With acute exposure, cardiac output (Q) and venous content of oxygen (C_vO_2) are reduced minimally, if at all. According to the Fick Principle, $\dot{V}O_2 \text{ max} = Q * (a-v O_2 \text{ difference})$. Therefore, the reduction in CaO_2 is the main influence in reduction of $\dot{V}O_2 \text{ max}$ (Michael P. Ward, 1995). As altitude increases, the ability to exert maximal effort becomes progressively impaired, thus a given amount of work represents a higher percentage of $\dot{V}O_2 \text{ max}$, making steady state aerobic exercise much more difficult, as depicted in figure 3 (George A. Brooks, 1996). Furthermore, evidence suggests that the largest reductions in $\dot{V}O_2 \text{ max}$ at altitude are seen in the most aerobically trained individuals when compared to un-trained individuals (Blomqvist, Johnson, & Saltin, 1969).

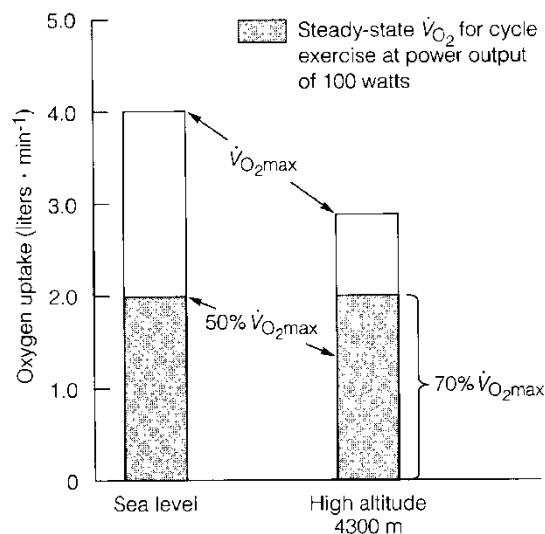


Figure 3: Effects of altitude on maximal and submaximal oxygen consumption

Pulmonary Ventilation

PCO_2 is the main regulator of pulmonary ventilation at sea level. With acute altitude exposure, the low PO_2 begins to control pulmonary ventilation by activating chemoreceptors which results in hyperventilation in an attempt to compensate for the low PO_2 of ambient air (Adamczyk et al., 2006). This compensatory increase in ventilation is referred to as the “Hypoxic Ventilatory Response” (HVR) and is depicted in figure 4 (Andersen).

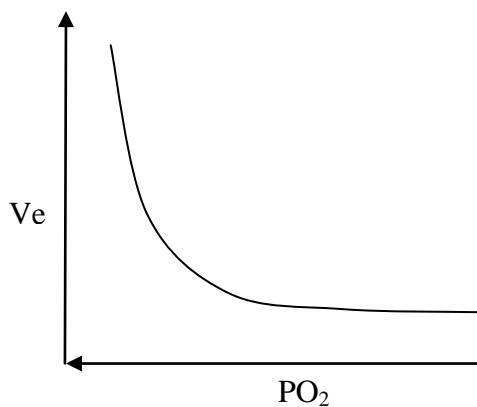


Figure 4: PO_2 control over pulmonary ventilation

Hyperventilation results in increases in pH (alkalosis), PaO_2 and O_2 saturation as PO_2 increases due to an increase in O_2 consumption, decrease in PaCO_2 as you blow more CO_2 off, and is critical in preserving arterial oxygen content (CaO_2) (Sutton & Gregg, 1994). The partial pressure of oxygen facilitates the drive of oxygen from the lungs into the blood (Hultgren, 1997). With a low PO_2 at altitude, this driving force is dramatically diminished, which impairs the rate of diffusion from the ambient air to the lungs and into the blood (Calbet & Lundby, 2009).

Respiratory responses to hypoxia are dependent on multiple factors. First, the severity and duration of hypoxia result in modifications in the ventilatory response. Furthermore, one study observed a blunted HVR in more trained individuals when compared to un-trained controls (Adamczyk, et al., 2006). This study suggests that exercise and training induced decreases in chemoreceptor sensitivity which reduced the ventilatory responsiveness to hypoxia. Therefore, well-trained athletes exhibit an elevated tolerance to hypoxia due to chemoreceptor adaptations resulting from years of physical activity and training (Adamczyk, et al., 2006).

Oxy-hemoglobin

A vast majority of O₂ transported in blood is carried by the hemoglobin molecule (Hb). The relationship between PO₂ and the affinity of Hb to bind to O₂ is described by three oxy-hemoglobin saturation (HbO₂) dissociation curves (figure 5). Acute exposure to altitude will cause a left shift in the oxy-hemoglobin dissociation curve (George A. Brooks, 1996). A left shift in the oxy-hemoglobin dissociation curve allows for a higher HbO₂ at a given PO₂.

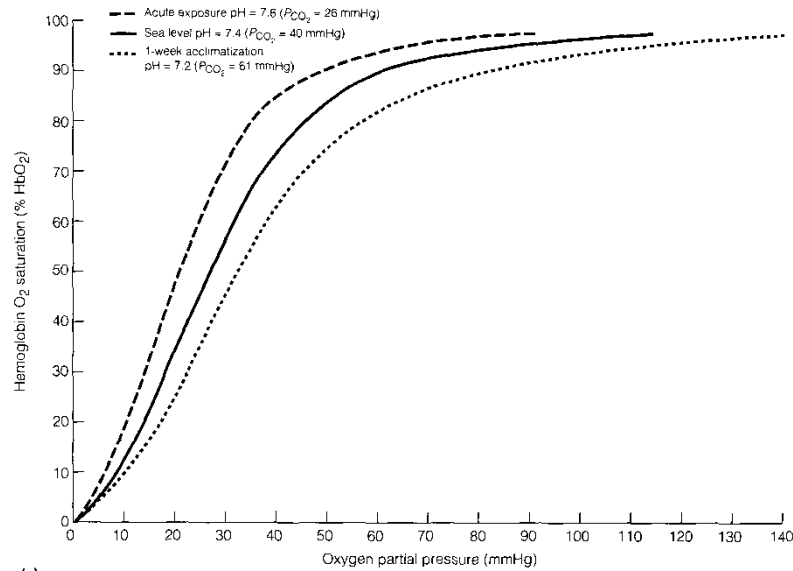


Figure 5: The oxy-hemoglobin dissociation curve following acute and chronic adaptations to altitude

This results from a decrease in PCO_2 , increased pH (alkalosis), and an increase in oxy-hemoglobin saturation. Increased hemoglobin saturation results in oxygen binding to hemoglobin more tightly, requiring a lower PO_2 necessary to release oxygen to the tissues (Michael P. Ward, 1995).

Chronic Adaptations to Altitude

Cardiovascular Adaptations

Prolonged exposure to altitude leads to more sustained physiological adaptations that differ from those seen with acute exposure. Heart rate continues to remain elevated above sea-level values, although cardiac output will decrease up to 20-25% with chronic exposure due to a continual decrease in stroke volume (*Hypoxia and Exercise*, 2006). Stroke volume will continue to decrease with prolonged exposure due to a low plasma volume that decreases filling of the heart, or reduces end diastolic volume (EDV), which consequently decreases stroke volume (SV) according to Starling Law. This decrease in

plasma volume results from dehydration associated with the HVR & respiratory water loss compounded by low relative humidity at altitude, along with a diuretic effect which reduces thirst and fluid intake while increasing urine production and excretion (*Hypoxia and Mountain Medicine*, 1991). A decreased plasma volume will lead to an increase in hematocrit, or red blood cell and hemoglobin concentration (West, 1981). The increase in red blood cell concentration increases the O₂ carrying capacity of blood, serving to improve oxygen delivery to tissues without an increase in cardiac output. Red blood cell concentration will continue to rise upon chronic exposure to altitude, while hemoglobin plateaus after several months of exposure as described in figure 6 (Hultgren 1997).

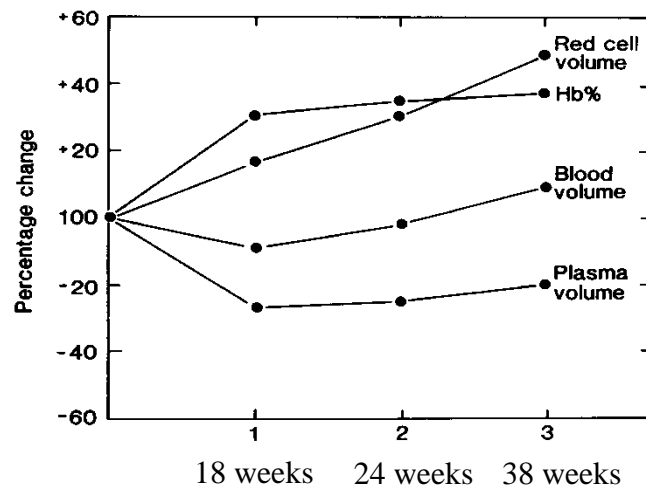


Figure 6: Mean changes in hemoglobin, red cell volume, plasma volume and total blood volume in four subjects during an eight month Himalayan expedition

$VO_2 \text{ max}$

$VO_2 \text{ max}$ will increase slightly from the depressed level seen during acute altitude exposure, although it remains well below $VO_2 \text{ max}$ levels seen at sea level. As previously mentioned, the reduction in $VO_2 \text{ max}$ seen with acute exposure is directly related to the decrease in arterial oxygen content (CaO_2). With chronic altitude exposure, CaO_2 will begin to normalize to sea level values. However, despite the normalization of CaO_2 , $VO_2 \text{ max}$ remains depressed as figure 7 describes (Gotshall).

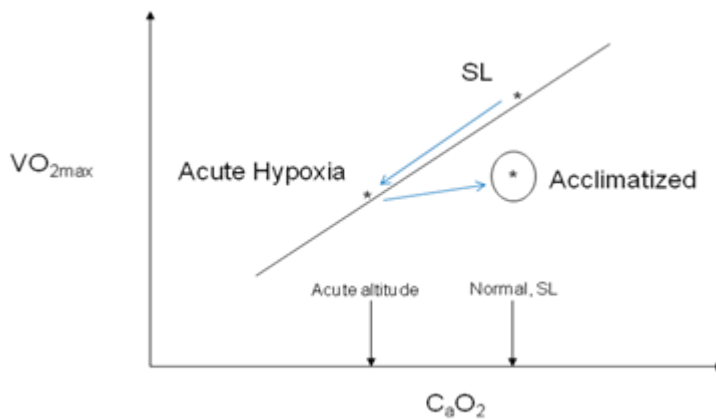


Figure 7: Chronic altitude adaptations to CaO_2 and $VO_2 \text{ max}$

Again, $VO_2 \text{ max} = Q * (a-v O_2 \text{ difference})$. Looking at the $VO_2 \text{ max}$ formula again, it is apparent that the cardiac output (Q) must have an impact in maintaining a depressed $VO_2 \text{ max}$. One study observed that with chronic altitude exposure, a competition for cardiac output occurs between exercising tissues and non-exercising tissues (Calbet et al., 2003). Therefore, blood flow and oxygen delivery is not being distributed to exercising tissues in proportions that will allow an increase in $VO_2 \text{ max}$ (Calbet, et al., 2003). Figure 8 shows cardiac output distribution at sea level and with chronic exposure to altitude (Gotshall).

In addition, the work required for ventilation requires a great deal of oxygen which blunts delivery to exercising muscles and impairs performance (figure 9) (Hultgren, 1997).

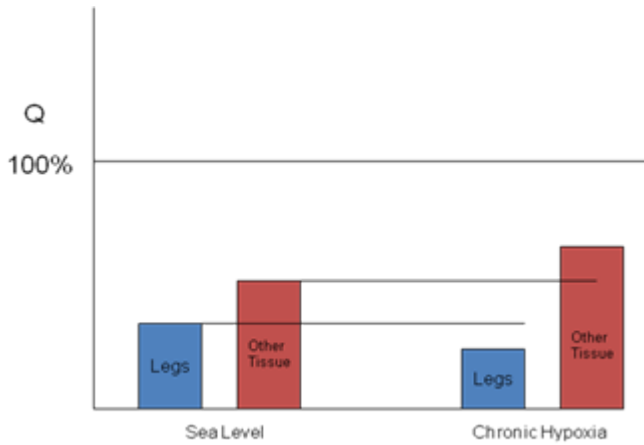


Figure 8: Cardiac output distribution at sea level and with chronic exposure to altitude

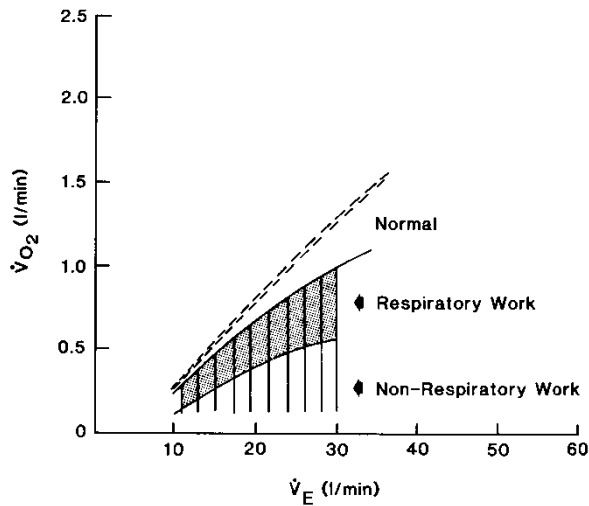


Figure 9: Amount of oxygen consumed by the respiratory muscles during exercise in chronic hypoxia at high altitude

Pulmonary Ventilation

Control over pulmonary ventilation returns to PCO_2 with prolonged exposure to altitude. As PCO_2 is reduced with increased ventilation at altitude, alkalosis occurs, which is an increase in pH. Chronic exposure to altitude will facilitate excretion of

bicarbonate by the kidneys to decrease pH caused by acute exposure. This brings the pH back to normal levels, restoring central chemoreceptor sensitivity and ventilatory control to PCO_2 (Michael P. Ward, 1995). Ventilation will increase with increased PCO_2 , allowing one to “blow off” extra CO_2 and inspire more O_2 . Figure 10 shows the PCO_2 control over pulmonary ventilation.

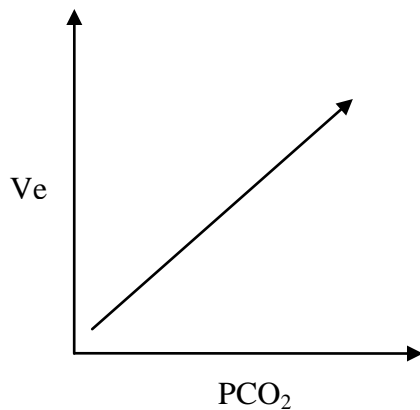


Figure 10: PCO_2 control over pulmonary ventilation

Pulmonary ventilation is also increased for a given PO_2 and PCO_2 with prolonged exposure to altitude, and is referred to as the “Hypoxic Ventilatory Response” as previously mentioned. Chronic exposure to altitude allows for an increased HVR at rest, and greater inspiration of oxygen to increase PaO_2 and reduce PaCO_2 at rest and during exercise in hypoxic conditions (Katayama et al., 2001). The process of ventilatory acclimatization can result in as little as four days of exposure, but can take up to 30 days or more to observe the full effects (Adamczyk, et al., 2006). Figures’ 10 and 11 show ventilatory acclimatization following chronic exposure to altitude (Gotshall).

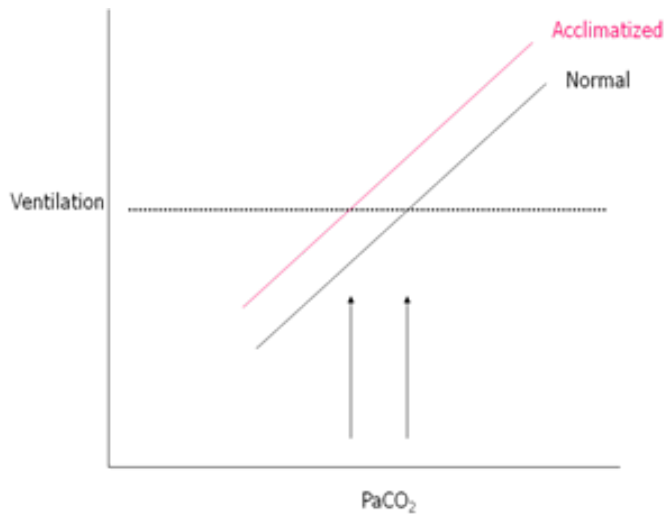


Figure 10: PaCO₂ control over pulmonary ventilation following chronic altitude exposure

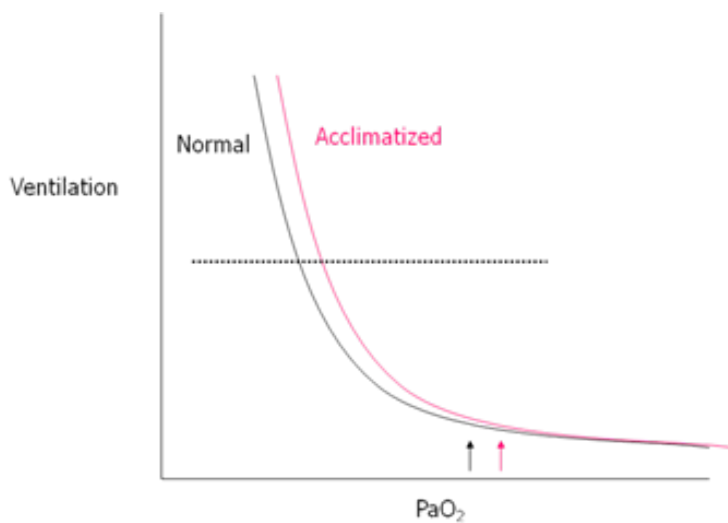


Figure 11: PaO₂ control over pulmonary ventilation following chronic altitude exposure

Oxy-hemoglobin

Prolonged exposure to altitude results in a right shift in the oxy-hemoglobin dissociation curve (figure 12) (George A. Brooks, 1996). This results from an increase in PCO₂, decrease in pH (acidosis), resulting in a decrease in hemoglobin saturation.

Addition of more hemoglobin will also be present during a right shift in the oxy-hemoglobin dissociation curve. Decreased hemoglobin saturation leads to a “looser” bond of oxygen and hemoglobin, leading to enhanced unloading of O₂ from Hb to the tissues at a given PO₂ (George A. Brooks, 1996).

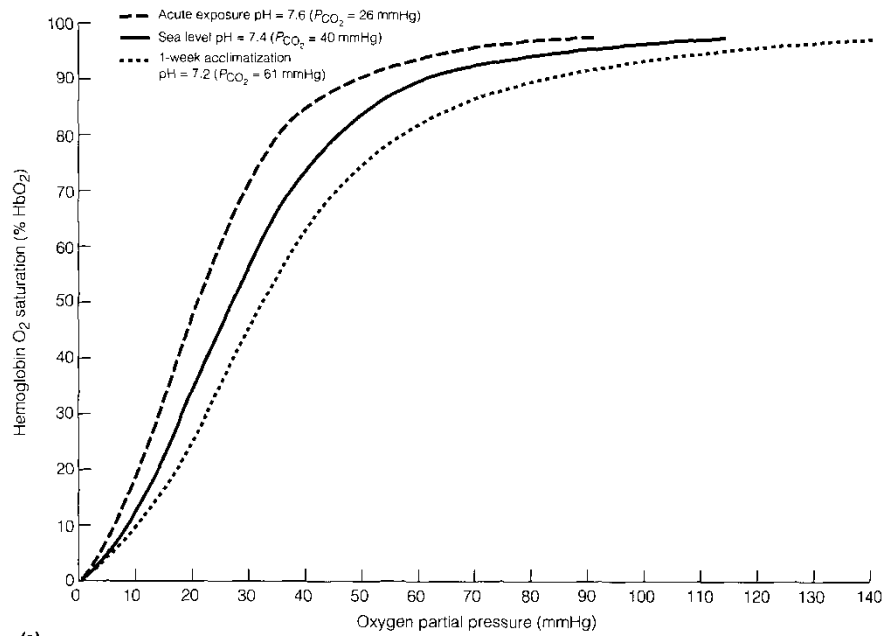


Figure 12: The oxy-hemoglobin dissociation curve following acute and chronic adaptations to altitude

Strategies for Improved Performance at Altitude

While chronic exposure to altitude is well known to elicit improvements in aerobic performance at altitude, endurance athletes have been seeking more effective and efficient methods for eliciting this response for many years. Several different methods have been employed to identify the optimal performance enhancing training routine. One method that has been studied is the implementation of intermittent hypoxic training (IHT). Studies draw comparisons between training sessions at or just above sea level with training sessions at altitude. Another method that has been previously researched is

intermittent hypoxic exposure (IHE). This method focuses on various physiologic acclimatization aspects with intermittent hypoxic exposure at rest. The final previously studied method is the Live High, Train Low concept (HiLo). With this method, athletes reside or sleep in high altitude or hypobaric environments, and train at or just above sea level. Although each method has been previously studied, results are controversial, possibly due to variations in individual responsiveness and the extent and duration of altitude exposure and/or training regime employed (Green, 2000). The most advantageous method of enhancing performance at altitude has yet to be identified. Moreover, the effect of a single short-duration exposure, such as the protocol examined in the present study, has not been investigated.

Intermittent Hypoxic Training (IHT)

Intermittent hypoxic training (IHT) is a technique used to enhance performance at altitude and at sea level. IHT specifically refers to the implementation of discontinuous training sessions in hypoxic or hypobaric conditions in an attempt to achieve physiologic altitude acclimatization effects to enhance performance at altitude, and possibly sea level as well (Levine, 2002). However, this strategy relies on the appropriate hypoxic dose, or extent and duration of exposure to elicit the acclimatization features sufficient enough to improve subsequent altitude performance (Debevec et al.). The idea is that training at altitude is much more demanding due to the challenges previously mentioned, such as an increase in ventilation and heart rate. However, chronic training at altitude can actually compromise performance by reducing an athlete's ability to train a high aerobic intensities due to the decrease in VO_2 max (Saunders, Pyne, & Gore, 2009). Therefore,

IHT has been hypothesized to elicit some acclimatization benefits associated with chronic altitude exposure without chronically reducing aerobic training intensity.

Unfortunately, while several studies have examined the effects of IHT on performance, well designed and controlled studies looking at the effects of IHT are controversial and lacking (Friedmann-Bette, 2008). One study compared the effects of IHT at a simulated altitude of 2750 m with similar training sessions at sea level (SLT). Training sessions were performed by each group over a four week period, three times per week. The results from this study revealed significant increases in VO₂ max, mean power, peak power and absolute power in both groups following the four week training period (Morton & Cable, 2005a). Although performance increased, there were no significant differences between the SLT group and the IHT group. In addition, hematological parameters did not reveal any significant changes in either group (Morton & Cable, 2005b). This study concluded that increases in performance elicited by IHT were not significantly greater than increases in performance elicited by SLT.

Other studies have suggested benefits of IHT on aerobic performance at altitude. One study compared the effects of IHT and normoxic training (NT) on sea level and altitude performance following a three week training program. No significant differences were observed in peak power output (PPO) measured in normoxia in either group. However, when measured in hypoxic conditions, only the IHT group increased PPO, as the NT group did not show any improvements (Roels, Bentley, Coste, Mercier, & Millet, 2007). This study concluded that IHT has superior training induced effects when compared to normoxic or sea level training in preparation for competition at altitude.

However, studies suggesting enhanced performance at altitude following an IHT protocol are very limited and quite controversial.

Intermittent Hypoxic Exposure (IHE)

Intermittent hypoxic exposure (IHE) refers to the discontinuous exposure of hypoxia under resting conditions to elicit altitude acclimatization effects and enhance subsequent performance at altitude (Rusko, Tikkanen, & Peltonen, 2004). Studies have shown that there is a certain duration and elevation required to elicit such acclimatization effects. Exposure to altitudes greater than 4000 m for at least an hour and a half per day, five to six days per week has shown to stimulate ventilatory acclimatization effects (Beidleman et al., 2009). However, one week of this IHE protocol along with exercise training at sea-level did not improve endurance at altitude (Beidleman, et al., 2009). Another study examined the differences between IHE and prolonged hypoxic exposure (PHE). IHE consisted of hypoxic exposure for five to six minutes, alternating with normoxic exposure for four to five minutes for a total duration of 60-90 minutes. PHE consisted of hypoxic exposure up to three hours per day. While IHE did not elicit any performance enhancing effects, PHE elicited increases in VO_2 max and improvements in time trial performance at 4300 m (Bartsch, Dehnert, Friedmann-Bette, & Tadibi, 2008).

Some studies have demonstrated positive effects of IHE. A seven week IHE protocol consisting of 30.8 total hours of exposure elicited a significant increase in oxygen saturation from 4570 – 6400 m compared to 2470 – 4570 m (Hetzler et al., 2009). Furthermore, an experiment exposing subjects to simulated altitude of 5500 m for 90 minutes per day, three days per week for three weeks resulted in increases in all hematological indicators including red cell mass, red blood cell count, hemoglobin

concentration along with an increase in SaO₂ measured at 5500 m (Rodriguez, et al., 2000). However, this study did not examine subsequent performance effects related to the IHE protocol. Physical performance enhancements have also been evaluated following IHE protocols. Three days after one hour of exposure per day for 15 days resulted in increases in sea-level endurance speed time trials by ~2%, although altitude endurance speed time trials were not assessed (Wood, Dowson, & Hopkins, 2006). Results also marked significant reductions in resting and exercise heart rates following the protocol (Wood, et al., 2006). Finally, one study examined the effects of simulated altitude exposure of 4500 m one hour per day for seven days on ventilatory responses (Katayama, et al., 2001). Researchers observed significant increases in ventilation and SaO₂ at rest and during exercise in simulated altitudes of 4500 m. In addition, the effects on ventilation and SaO₂ following the IHE protocol were maintained for seven days following cessation of the hypoxic exposure (Katayama, et al., 2001). In summary, it is known that IHE can elicit acclimatization effects that improve subsequent aerobic performance; however the available literature examining effects of IHE are mixed and controversial (Green, 2000).

Live High Train Low (HiLo)

It has been hypothesized that high altitude natives (HAN) such as Tibetans from the Himalaya Mountains may have enhanced work capacities in hypoxic conditions due to acclimatization and genetic factors (Brutsaert, 2008). Residing at such high elevations, HAN have been known to feature enhanced oxygen consumption (VO₂ max) at altitude and smaller decrements of VO₂ max with increased levels of hypoxia (Brutsaert, 2008). In the early 1990's, Dr. Benjamin Levine and James Stray-Gundersen developed the live

high, train low (HiLo) model which consists of athletes residing at high altitudes while training at lower altitudes (Wilber, 2007). The essential features of this method include living high enough, for long enough to produce essential altitude acclimatization features similar to HAN while training at lower altitudes to ensure high intensity training sessions for enhanced performance both at sea level and altitude (*Hypoxia and Exercise*, 2006). The most important altitude acclimatization effects elicited by the HiLo method include increased pulmonary ventilation (HVR), increased hemoglobin, and enhanced oxygen extraction by the tissues (Sutton & Gregg, 1994). The question that remains to be answered is what altitude and duration is required to produce the most optimal altitude acclimatization effects.

To address the previous question, some studies have implemented multiple different altitudes and durations. One study examined individuals residing at four different altitudes, 1780 m, 2085 m, 2454 m and 2805 m. Results showed increases in performance in all four altitude groups; however, the most marked improvements were seen in the middle two elevations, while the lowest and highest elevations showed minimal improvements (*Hypoxia and Exercise*, 2006). Erythropoiesis accelerates between weeks 3-4 of living at altitude, producing significant increases in hemoglobin and hematocrit (*Hypoxia and Exercise*, 2006). Therefore, durations of less than two weeks are not able to generate improvements in performance.

The HiLo method is an increasingly popular one among athletic teams in the United States and around the world. The US speed skating team took advantage of the HiLo method while training for the 2002 Winter Olympics in Salt Lake City, Utah. Skaters on the team lived in Park City, Utah, an elevation of 2000-2500 m, and practiced

at the Olympic Oval at an elevation of 1400 m (*Hypoxia and Exercise*, 2006). This method seemed to be advantageous, as the US speed skating team was awarded the largest number of medals to the most number of skaters in US speed skating history (*Hypoxia and Exercise*, 2006)

Periodic Acute Altitude Training

Periodic, acute altitude training defined as acute bouts of exercise at altitude separated by several days of training in native/lower altitude environments, is a method which has not yet been previously researched. The aim of this study will be to identify whether periodic hill running sessions at altitude are superior to similar hill running sessions performed at low altitude in preparation for a high altitude endurance race. In addition, whether periodically training at altitude elicits beneficial physiological acclimatization effects that are superior to those seen with training at lower altitudes was also examined.

CHAPTER III

METHODS AND PROCEDURES

Prior to recruitment of subjects, the research team obtained approval to conduct the study by the Institutional Review Board (IRB) at Colorado State University in Fort Collins, Colorado. Once selected, subjects provided informed consent to all protocol methods and procedures as explained by a research team member at the Human Performance Clinical/Research Laboratory (HPCRL) at Colorado State University.

Subject Selection

Subjects were recruited through a recruitment flyer posted on the Fort Collins Trail Runners Group website and in the Department of Health and Exercise Science at Colorado State University. Exclusion criteria consisted of anyone under the age of 18, or over the age of 65 and/or a VO_2 max under 45 ml/kg/min. Eight competitive trail runners (VO_2 max 52 ± 5 ml/kg/min; Range: 45.4-59.9 ml/kg/min) participated in the study. Subjects consisted of seven males and one female (aged 42 ± 14 years; Range: 22-61 years) residing in Fort Collins, CO (5,003 feet). All subjects refrained from altitude exposure above 7,000 feet at least four weeks prior to the beginning of the study, and were instructed to refrain from any non-prescribed altitude exposure for the duration of the study. Subject characteristics are presented in Table 1.

Table 1: Subject Characteristics

Subject	Age	Height (in.)	Weight (lbs.)	VO2 Max (ml/kg/min)
1	31	69	178.5	49.2
2	22	69	121	45.4
3	46	72	153	51.9
4	59	69	135.5	46.2
5	32	69.5	151	59.9
6	61	70.5	139	51.7
7	35	71	150	59.7
8	49	70	141.5	56.1

Experimental Groups

Following selection into the study, subjects were semi-randomly assigned to one of two groups receiving a high or low altitude training intervention described below.

Initially, subjects were matched on the above parameters to ensure similar age, weight and baseline levels of aerobic fitness. However, failure to collect data during the first attempted pre-test run due to weather conditions resulted in scheduling conflicts for many subjects. Therefore, re-assigning the groups became necessary to accommodate the research team and subjects schedules ultimately resulting in the group assignments described in Table 2.

Table 2: Baseline Group Characteristics

Group	Age	Height (in.)	Weight (lbs.)	VO ₂ max (ml/kg/min)
LOW (n = 4)	44 ± 7	70.25 ± 0.3	145 ± 3	56.8 ± 1.9
HIGH (n = 4)	40 ± 8	69.75 ± 0.75	147 ± 12	48.2 ± 1.5

Pre-treatment Assessments

History

Subjects completed a general health history questionnaire and a detailed running history questionnaire including years training, current average miles per week running flat vs. hills and racing experience. Questionnaires are included in the Appendix section.

Aerobic Fitness Assessment

Prior to beginning the study, a baseline graded exercise treadmill VO_2 max assessment was administered at the HPCRL at Colorado State University for determination of aerobic capacity. VO_2 max assessments were administered using a Cardiac Science TM65 treadmill (Bothell, Washington) and a Parvo Medics TrueOne 2400 Metabolic Measuring System (Sandy, Utah).

Hematocrit Assessment

Prior to beginning the study, a hematocrit assessment was administered at the HPCRL through a venous blood sample from a finger prick using an International Equipment Company Model MB micro-capillary centrifuge (Needham Heights, Massachusetts). A small amount of blood was collected into a capillary tube and spun down in the micro-capillary centrifuge. Once spun in the centrifuge, total blood content and red blood cells were measured using a millimeter ruler. The length of red blood cells was divided into the total blood volume to assess the percentage of red blood cells to total blood volume, or hematocrit. Hematocrit was assessed throughout the study one day prior to and one day following each test or training run. Subjects were instructed to be adequately hydrated when coming in for hematocrit assessments.

Pre-treatment High-Altitude Hill Run

All subjects completed a timed max effort high-altitude hill run on the Mount Evans Scenic Byway outside of Idaho Springs, Colorado. The test-run consisted of 11.5 miles starting at an elevation of 11,500 feet and ending at the summit at an elevation of 14,100 feet (Average grade: 4.23%; Range: 1.2 – 6.1%). Water and Powerade© were provided to all subjects *ad libitum* at designated points during the test-run. Test-run profile is presented in figure 13.

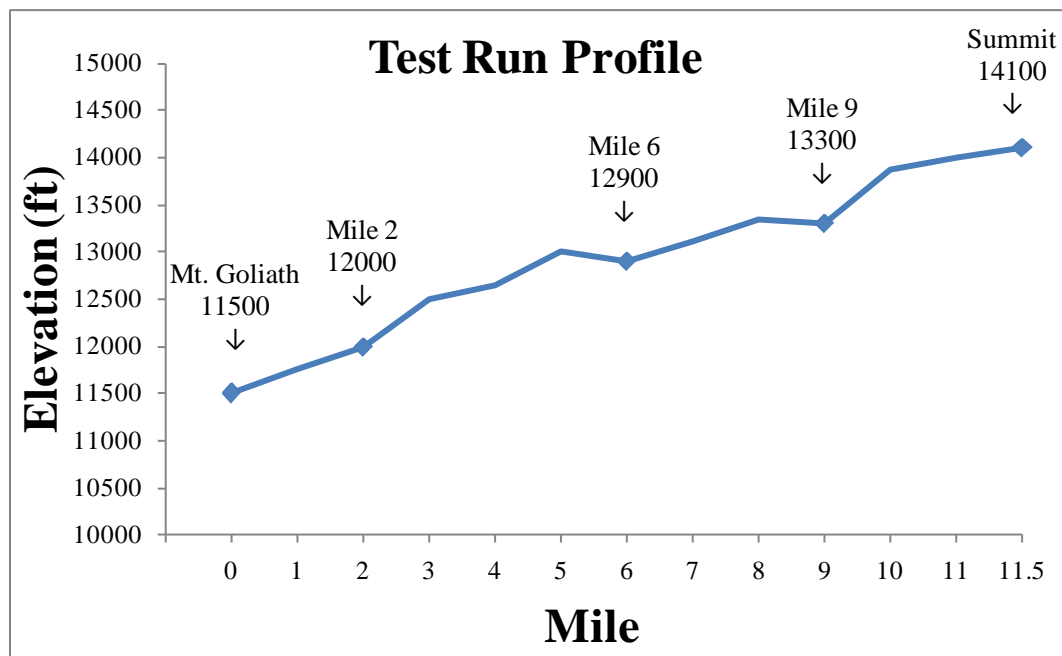


Figure 13: Test-Run Profile

Outcome Measurements

Elapsed run-time, arterial oxygen saturation (SaO₂), heart rate (HR) and rate of perceived exertion (RPE) were all measured at the start, miles 2, 6, 9 and 11.5 (summit). SaO₂ was also measured at the summit after 20 minutes of rest upon completion of the test-run to determine the extent of re-saturation at 14,100 feet following exercise. In

addition, SaO₂ was measured at each data collection mile marker on the way down from the summit to determine the extent to which recovery SaO₂ was impacted by the descent. Elapsed run-time was measured using a Garmin Ltd. Forerunner 305 trainer watch (Olathe, Kansas) and recorded by study personnel at each indicated mile marker. SaO₂ and HR were measured using a Nonin Medical Inc. GO₂ LED pulse-oximeter (Plymouth, Minnesota). RPE was measured using the standard Borg 6-20 RPE scale that was described in full to each subject before beginning the test-run.

Training Intervention

All subjects were instructed to engage in their standard training routine 5-6 days per week during the study. Subjects also kept a log of their daily running activity. One week after the pre-treatment high-altitude hill run, subjects engaged in either a high intensity “3-2-1” hill training session on either Towers Road in Horsetooth Mountain Park (LOW) or the Mount Evans Scenic Byway (HIGH).

LOW Group:

Starting at the summit of Towers Road in Horsetooth Mountain Park (7,170 feet), subjects descend three miles to an elevation of 5,550 feet followed by an immediate ascent back to the summit. Subjects then descend two miles to an elevation of 6,146 feet followed by an immediate ascent back to the summit. A final descent of one mile to an elevation of 6,668 feet followed by an immediate ascent back to the summit completes the “3-2-1” training intervention. LOW group training intervention profile is presented in figure 14.

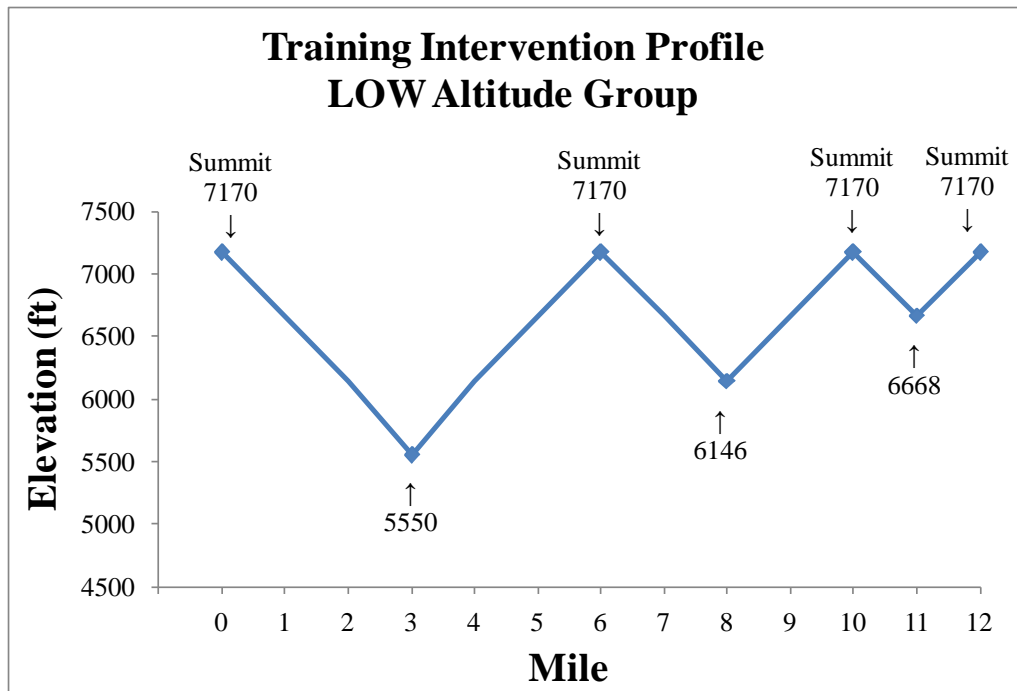


Figure 14: LOW Group Training Intervention Profile

HIGH Group:

Starting at the summit of the Mount Evans Scenic Byway (14,100 feet), subjects descend three miles to an elevation of 12,750 feet followed by an immediate ascent back to the summit. Subjects then descend two miles to an elevation of 13,250 feet followed by an immediate ascent back to the summit. A final descent of one mile to an elevation of 13,750 feet followed by an immediate ascent back to the summit completes the training intervention. HIGH group training intervention profile is presented in figure 15.

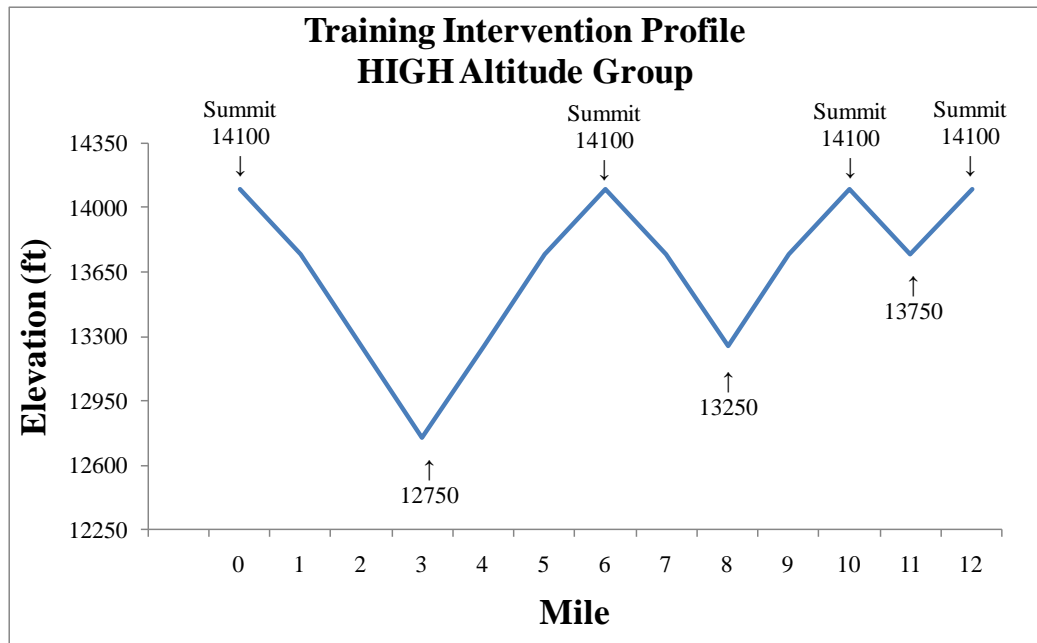


Figure 15: HIGH Group Training Intervention Profile

Post-treatment Assessments

Post-treatment High Altitude Hill Run:

One week after the training intervention, all subjects completed a max effort high-altitude hill run on the Mount Evans Scenic Byway identical to the pre-treatment test-run (11.5 miles; starting elevation: 11,500 feet, ending elevation: 14,100 feet; average grade: 4.23%; range: 1.2 – 6.1%). Water and Powerade© were provided to all subjects *ad libitum* during the test-run. Test-run profile is presented in figure 13.

Outcome Measurements:

Elapsed run-time, arterial oxygen saturation (SaO₂), heart rate (HR) and rate of perceived exertion (RPE) were all measured at the start, miles 2, 6, 9 and 11.5 (summit). SaO₂ was also measured at the summit after at least 20 minutes of rest upon completion of the test-run. In addition, SaO₂ was measured at each data collection mile marker on

the way down from the summit to determine the extent to which recovery SaO₂ was retained. Run-time was measured using a Garmin Ltd. Forerunner 305 trainer watch (Olathe, Kansas). SaO₂ and HR were measured using a Nonin Medical Inc. GO₂ LED pulse-oximeter (Plymouth, Minnesota). Data recorded represent an average of at least three SaO₂ and HR measurements obtained while subjects continued running. RPE was measured using the standard Borg 6-20 RPE scale that was described in full to each subject before beginning the test-run.

Post-study Aerobic Fitness Assessment

To determine whether there was any general training effect during the three week study protocol, a graded exercise treadmill VO₂ max assessment was administered at the HPCRL for determination of aerobic capacity within one week of the post-treatment high-altitude hill run.

Pulse-oximeter Validation

In order to validate the accuracy and reliability of the Nonin portable pulse-oximeter used in our study, we used a self-regulating partial rebreathe system developed by Banzett *et al.* (2000) in the HPCRL to simulate hypoxic conditions. Subjects (n = 4) breathed hypoxic air through the rebreathe system with nose clips on to prevent any nasal breathing. We gradually reduced the fraction of inspired oxygen (F_IO₂) from 18-10%. Arterial oxygen saturation (SaO₂) was measured every minute for ten minutes using the Nonin portable pulse-oximeter used during data collection and a Datex-Ohmeda Cardiocap/5 Oxy Tip pulse-oximeter (Louisville, Colorado) placed on opposite index fingers. Pulse-oximeter validation and reliability data is presented in table 3a and 3b respectively.

Table 3a: Pulse-oximeter Validation Data

FiO₂ (%)	PO₂ (mmHg)	SaO₂ (Nonin)	SaO₂ (D-O)	P-Value
17	107.4	95.5 ± 2.1	95.8 ± 2.4	0.879
16	101.1	94.3 ± 2.1	94.8 ± 3.3	0.806
15	94.8	88.3 ± 7.4	88.0 ± 11.5	0.968
14	88.5	92.0 ± 2.6	92.7 ± 4.0	0.823
13	82.2	87.3 ± 7.7	87.3 ± 10.0	1.000
12	75.8	86.4 ± 1.8	87.6 ± 2.7	0.434
11	69.5	79.4 ± 4.6	79.0 ± 6.1	0.884
10	63.2	78.7 ± 2.5	77.7 ± 3.6	0.586
Average P-Value:				<i>0.797 ± 0.194</i>

Table 3a illustrates data from pulse-oximeter validation study. FiO₂ = %O₂ of inspired air; PO₂= partial pressure of oxygen; SaO₂ = arterial HbO₂ % saturation; D-O = Datex-Ohmeda pulse-oximeter; SaO₂ values represent the average of values from 4 subjects' ± SD at a given FiO₂.

Mean % difference between the two devices was 0.9% with an R-value of 0.99. P-values at each given FiO₂ indicate that the two pulse-oximeters were not statistically different from one another. Therefore, we concluded that the Nonin pulse-oximeter is a sufficiently valid instrument in measuring SaO₂ in our subjects during the test/training runs.

Table 3b: Pulse-oximeter Reliability Data

FiO₂ (%)	PO₂ (mmHg)	SaO₂ (Nonin)	CV	SaO₂ (D-O)	CV	ΔCV
17	107.4	94.9 ± 0.8	0.008	95.8 ± 0.6	0.006	0.002
15	94.8	93.1 ± 1.1	0.012	92.0 ± 0.5	0.005	0.007
13	82.2	89.9 ± 0.9	0.010	88.6 ± 0.8	0.009	0.001
11	69.5	81.8 ± 3.3	0.041	79.4 ± 2.6	0.033	0.008
			<i>Average CV:</i>	<i>0.0178</i>	<i>0.0133</i>	

Table 3b represents data from the pulse-oximeter reliability study. SaO₂ values represent the average of 8 consecutive measurements at the indicated FiO₂ over 60 seconds to determine the extent to which the data obtained from each oximeter varies during a one minute data collection period. Coefficients of variation (CV = SD/mean) were calculated to determine the extent of variability over time.

Oximeter readings varied an average of 1.78% with the Nonin and 1.33% with the Datex-Ohmeda (P-value for difference = 0.672) over the course of the one minute data collection. Therefore, we concluded that the Nonin is a sufficiently reliable instrument in measuring SaO₂ in our subjects during the test/training runs.

Statistical Analysis

Differences within each group (pre-treatment vs. post-treatment) for high-altitude hill run variables (run-time, SaO₂, HR and RPE) were determined using nonparametric procedures. For absolute pre-post data within each group, the Wilcoxon Signed Ranks procedure was used. The analysis of both absolute and relative (%) change in any variable was analyzed using the Mann-Whitney U. All statistics were run in SPSS version 19.0. Statistical significance was established at P < 0.10 for all analysis.

CHAPTER IV

RESULTS AND DISCUSSION

Results

Elapsed Run-Time

Figure 16 represents absolute pre and post-test run-times for both High and Low groups. No statistically significant differences between High and Low groups run-time were observed. Figure 17 describes the change in elapsed run-time from pre to post-test for both High and Low groups. In addition, there were no statistically significant differences in the High group from pre to post-test in run-time ($-3.88 \pm 8.15\%$, or -4.25 ± 7.93 minutes). There were also no statistically significant differences in the Low group from pre to post-test in run-time ($1.25 \pm 4.56\%$, or 1.0 ± 4.76 minutes).

However, due to scheduling conflicts, one of the subjects in the High group was required to complete the post-test on a separate day than the rest of the subjects when there were unusually high winds on Mt. Evans (> 20 mph winds sustained). When we excluded the data from this subject, there was a strong trend for improvements in total high-altitude run-time in the High group ($-7.10 \pm 6.12\%$, or 7.33 ± 6.11 minutes faster), however this difference did not reach statistical significance ($P = 0.114$).

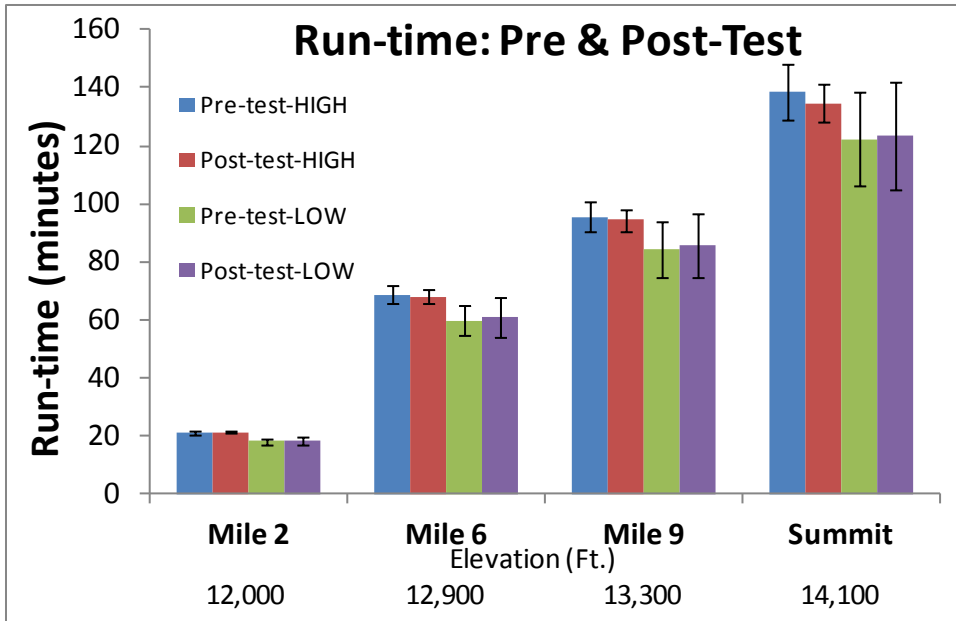


Figure 16: Absolute pre and post-test run-times for both High and Low groups

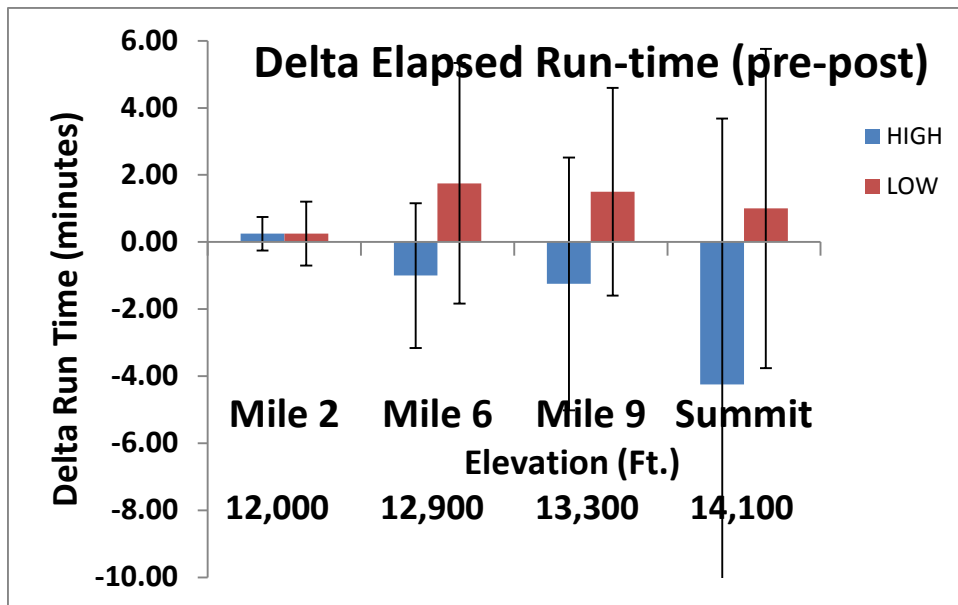


Figure 17: Delta Elapsed Run-time from pre to post-test for High and Low groups

Exercising SaO₂ and HR

Results from pre and post-test exercising SaO₂ and HR for both High and Low groups are presented in Table 4. Absolute exercise SaO₂ was significantly higher in the High group from pre to post-test at elevations 13,300 (mile 9) and 14,100 feet (mile 11.5, summit). In addition, absolute exercise SaO₂ was significantly lower from pre to post-test in the Low group at elevations 11,500 (start of the race) and 12,000 feet (mile 2).

Table 4: Exercising SaO₂ and HR; *P < 0.10 for pre vs. post

Elev. (ft.)	PO ₂ (mmHg)	<u>High Group</u>		<u>Low Group</u>	
		SaO ₂ (%)	HR (bpm)	SaO ₂ (%)	HR (bpm)
<i>Pre-test</i>					
5,003	133.1	97.8 ± 0.3	77.0 ± 12.5	97.3 ± 0.5	78.3 ± 10.0
11,500	105.1	89.3 ± 1.8	75.3 ± 5.0	93.5 ± 2.1	68.5 ± 10.0
12,000	102.7	78.0 ± 2.1	162.3 ± 6.3	79.0 ± 0.0	146.8 ± 37.3
12,900	100.3	75.5 ± 1.9	159.0 ± 5.7	75.3 ± 1.3	132.5 ± 20.6
13,300	98.6	72.5 ± 4.6	151.8 ± 20.8	75.8 ± 0.6	145.0 ± 23.4
14,100	98.0	75.5 ± 2.8	149.8 ± 20.9	73.0 ± 1.6	136.0 ± 35.2
<i>Post-test</i>					
5,003	133.1	98.0 ± 1.4	70.8 ± 8.7	95.0 ± 2.4	62.0 ± 15.2
11,500	105.1	82.5 ± 2.4	79.0 ± 2.0	*89.8 ± 2.5	66.5 ± 19.6
12,000	102.7	77.8 ± 4.3	130.8 ± 19.6	*76.3 ± 2.9	149.5 ± 17.2
12,900	100.3	*78.3 ± 4.1	148.8 ± 17.3	76.0 ± 2.6	133.3 ± 34.0
13,300	98.6	*77.8 ± 4.1	158.5 ± 10.6	76.3 ± 3.4	126.5 ± 20.8
14,100	98.0	79.8 ± 1.7	145.5 ± 9.9	73.5 ± 3.9	147.5 ± 15.8

Figures 18 & 19 represent % change in exercising SaO₂ and HR respectively for both High and Low groups from pre to post-test. The pre-run (11,500 ft) percent change in SaO₂ was significantly different between groups (+3.70 ± 2.95% in High; -3.95 ± 1.62% in Low, P = 0.029). When all altitudes are combined, the High group experienced a 4.01 ± 2.72% mean increase in exercising SaO₂ from pre to post-test, while the Low group decreased 1.21 ± 1.41% from pre to post-test (P = 0.029).

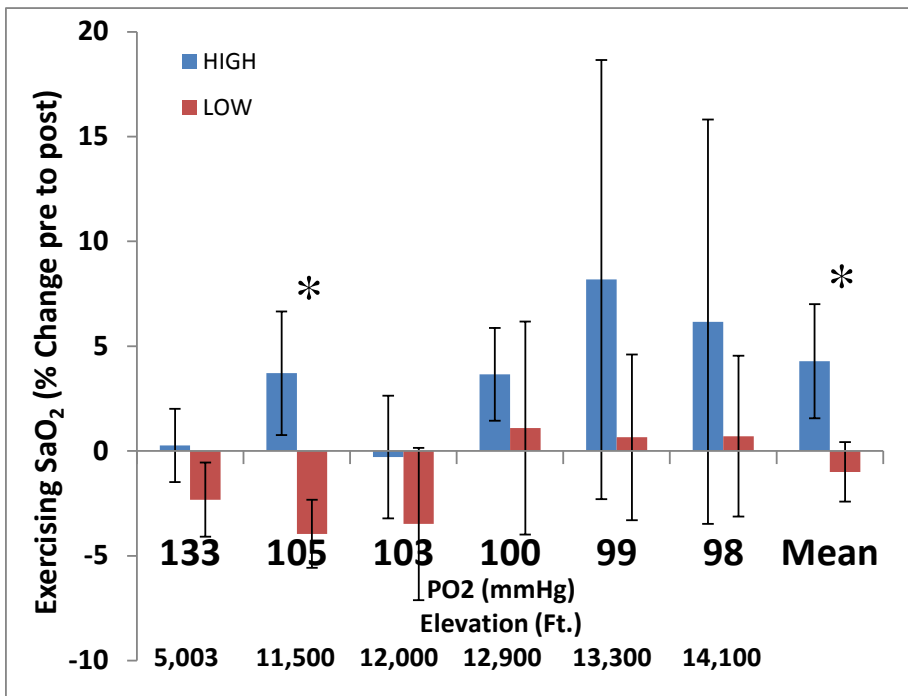


Figure 18: % Change in Exercising SaO₂ from Pre to Post-test
 *P < 0.05 for group difference

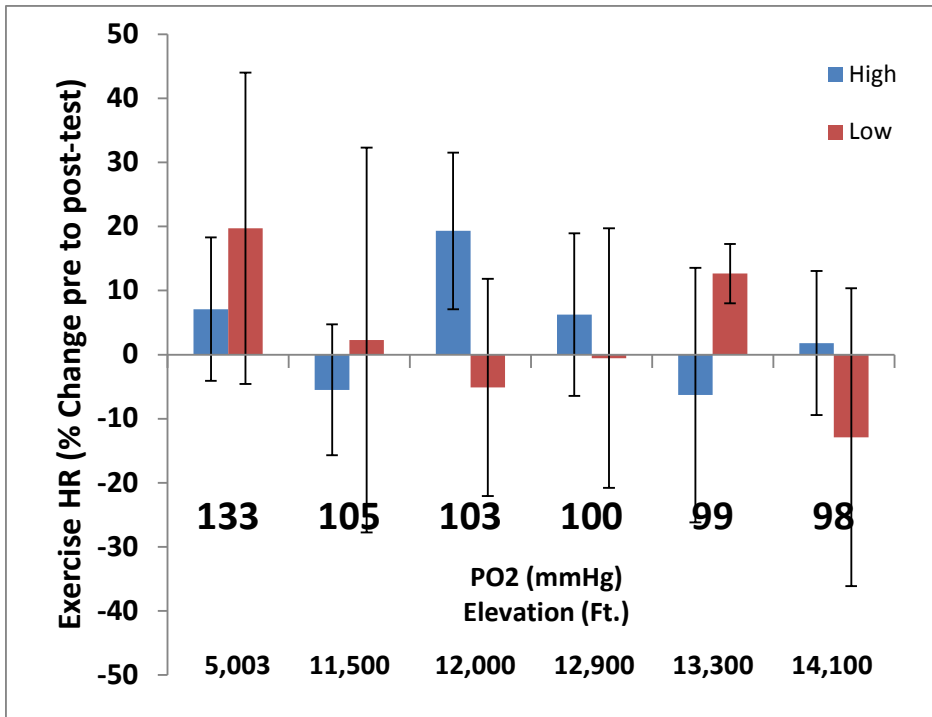


Figure 19: % Change in Exercising HR from Pre to Post-test

Recovery SaO₂ and HR

To determine if the High or Low altitude training intervention altered SaO₂ and HR responses to high altitude under recovery conditions, we evaluated these parameters at least 20 minutes following the pre and post-test runs. Results are presented in Table 5. Absolute recovery SaO₂ did not differ between groups at the summit of 14,100 feet.

Table 5: Recovery SaO₂ and HR

Elev. (ft.)	PO ₂ (mmHg)	<u>High Group</u>		<u>Low Group</u>	
		SaO ₂ (%)	HR (bpm)	SaO ₂ (%)	HR (bpm)
<i>Pre-test</i>					
5,003	133.1	94.8 ± 1.0	86.5 ± 9.1	95.5 ± 2.4	77.5 ± 4.0
11,500	105.1	86.5 ± 5.0	85.5 ± 13.0	87.3 ± 3.9	81.5 ± 8.9
12,000	102.7	84.8 ± 2.6	84.5 ± 16.3	84.5 ± 5.7	80.5 ± 10.4
12,900	100.3	82.3 ± 5.9	87.0 ± 19.7	85.8 ± 7.6	79.8 ± 14.1
13,300	98.6	82.3 ± 7.5	82.8 ± 14.2	84.0 ± 3.7	75.3 ± 9.9
14,100	98.0	77.5 ± 5.0	85.0 ± 13.7	77.8 ± 1.7	77.3 ± 9.1
<i>Post-test</i>					
5,003	133.1	95.3 ± 0.5	80.3 ± 8.0	95.3 ± 1.0	66.5 ± 4.1
11,500	105.1	86.5 ± 1.7	80.5 ± 19.6	86.3 ± 5.6	75.8 ± 10.6
12,000	102.7	84.5 ± 2.9	80.8 ± 15.9	85.3 ± 4.2	77.8 ± 12.7
12,900	100.3	84.5 ± 5.4	82.3 ± 20.8	83.5 ± 2.4	77.5 ± 14.0
13,300	98.6	86.0 ± 1.6	87.5 ± 21.6	82.3 ± 4.1	77.0 ± 13.1
14,100	98.0	83.8 ± 1.0	82.3 ± 12.8	76.0 ± 1.4	77.8 ± 8.1

Figures 20 & 21 represent % change in recovery SaO₂ and HR respectively for both High and Low groups from pre to post-test. The High group experienced a 8.45 ± 8.13% increase in recovery SaO₂ from pre to post-test at an elevation of 14,100 feet, while the Low group had a 2.21 ± 2.82% decrease in recovery SaO₂ from pre to post-test at an elevation of 14,100 feet (P = 0.057). However, there were no differences in recovery SaO₂ from pre to post-test at elevations 13,300 feet and below for either group.

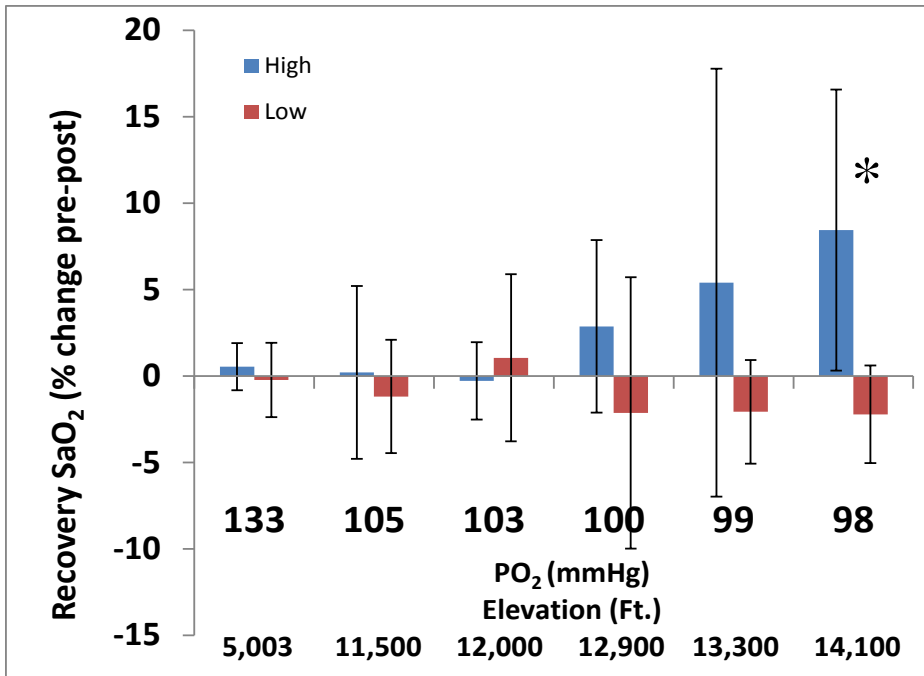


Figure 20: % Change in Recovery SaO₂ from Pre to Post-test

*P < 0.10 for group difference

No statistically significant differences in recovery HR from pre to post-test between or within groups were observed.

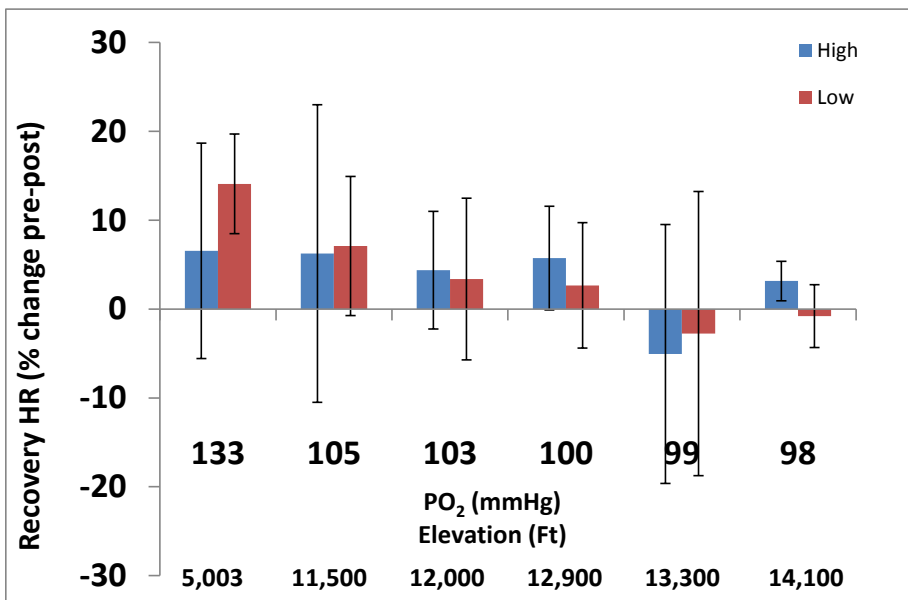


Figure 21: % Change in Recovery HR from pre to post-test

Hematocrit

Results from hematocrit measurements throughout the study are presented in Figure 22. The Low group experienced a significant increase in hematocrit from pre post-test to post post-test ($43.55 \pm 1.04\%$ & $45.67 \pm 0.79\%$ respectively; $P = 0.68$).

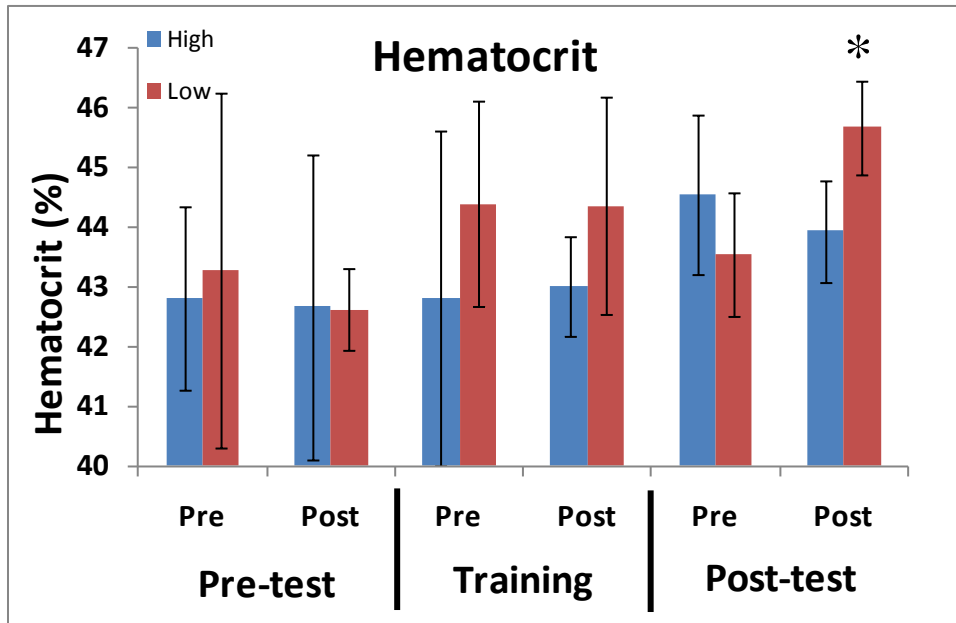


Figure 22: Hematocrit throughout the study for both High and Low groups
* $P < 0.10$ for pre post-test vs. post post-test in Low

RPE

Mean RPE values for both High and Low groups are presented in Table 6. Mean RPE values for the High group ranged between 16.5 – 18.5 (pre-test 17.5 ± 0.95 ; post-test 17.25 ± 1). Mean RPE values for the Low group ranged between 13.5 – 17.25 (pre-test 15.75 ± 1.5 ; post-test 15 ± 1.5). There were no significant differences within or between groups for RPE. Figure 23 represents the change in RPE from pre to post-test for both High and Low groups.

Table 6: Mean RPE values for High and Low pre and post-test runs

Elev. (ft.)	PO ₂ (mmHg)	<u>High Group</u>		<u>Low Group</u>	
		Pre-test	Post-test	Pre-test	Post-test
12,000	102.7	16.5 ± 1.7	16.5 ± 1.0	14.5 ± 2.5	13.5 ± 1.3
12,900	100.3	17.0 ± 1.7	16.5 ± 1.0	15.0 ± 1.6	14.5 ± 1.7
13,300	98.6	18.0 ± 1.3	17.5 ± 0.6	16.5 ± 2.9	15.5 ± 1.7
14,100	98.0	18.5 ± 1.3	18.5 ± 0.6	17.5 ± 1.5	17.0 ± 1.5

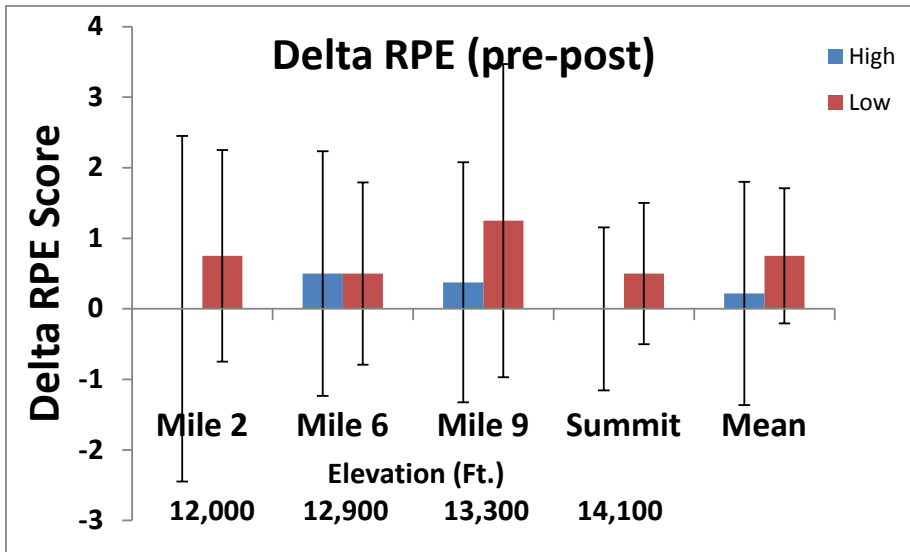


Figure 23: Delta RPE from pre to post-test for both High and Low groups

Post VO₂ Max

To determine whether the High or Low altitude training had an effect on aerobic exercise capacity, a post VO₂ max assessment was administered within one week of the high-altitude post-test run. Mean post VO₂ max for High and Low groups was 47.9 ± 1.2 ml/kg/min and 58.1 ± 3.0 ml/kg/min respectively. As in the baseline VO₂ max

assessment, mean values for the High group were significantly lower than the Low group ($P < 0.05$), but there were no significant differences between the baseline and post-test values for either group.

Discussion

The present study tested the hypothesis that a single high altitude bout improves aerobic performance following one week of routine training at a lower native altitude. The primary findings of the study include an improvement in both exercising and recovery SaO₂ responses following the high altitude training intervention. There were no differences observed in HR, HCT, RPE or aerobic capacity (VO₂ max) throughout the study for either group.

Elapsed Run-time

Maximal aerobic exercise performance depends on the ability of working muscles to uptake, transport, deliver and utilize oxygen for oxidative phosphorylation of ADP to ATP. VO₂ max reflects the maximal rate of oxidative phosphorylation by the exercising tissues, and is therefore the primary index of aerobic exercise capacity. In the present study, the Low altitude training group was able to complete both pre and post-test runs in less time than the High altitude group, which is likely due to their higher aerobic capacity indicated by their higher VO₂ max vs. the High group (Table 2). However, despite no change in VO₂ max from pre to post testing, a strong trend for improvements in run-time were observed in the High, but not the Low altitude training group. The lack of statistical significance might have resulted from the small number of subjects per group, therefore more subjects will need to be tested to confirm this effect. Assuming the trend for a faster run time is a legitimate effect, the lack of any change in VO₂ max measured in Fort

Collins indicates that improvements were not due to increased in aerobic capacity, suggesting that other mechanisms must have enhanced exercise efficiency or oxygen availability at altitude. While it is plausible that improvements in biomechanical efficiency or course familiarity may have contributed to the faster run time, these parameters were not assessed in this study. The High altitude training group did experience improvements in SaO₂ responses from pre to post-test. A higher SaO₂ in the High group during the post-test would allow for more O₂ to be available for delivery to exercising tissues for oxidative metabolism by the mitochondria, which could allow for an improvement in aerobic running performance.

Exercising & Recovery SaO₂

There are at least two mechanisms that could explain the increase in SaO₂ observed from pre to post-test in the High altitude training group. The first is an increased ventilation rate in response to hypoxia. This phenomenon is commonly referred to as the “hypoxic ventilatory response”. An enhanced HVR results in a greater O₂ loading of Hb due to hyperventilation (Adamczyk et al., 2006). This would allow an increase in alveolar PO₂, effectively increasing ones SaO₂ and subsequently increasing the O₂ diffusion gradient from the arterial blood to working tissue.

The second possible mechanism relates to an enhanced HVR, and is referred to as a “left shift” in the oxy-hemoglobin dissociation curve associated with acute altitude exposure. A “left shift” represents an increased affinity of Hb for O₂ at a given PO₂, allowing O₂ to bind to Hb more tightly, effectively increasing arterial HbO₂ loading, leading to a greater SaO₂ at a given PO₂. This effect is known to result from a decrease in PCO₂ and/or increase in pH due to hyperventilation, and is typically associated with a

normal acute exposure to hypoxia. Increases in SaO₂ are associated with more O₂ available for delivery to the exercising tissues for oxidative metabolism by the mitochondria.

Regardless of the mechanism responsible, the observed increases in SaO₂ suggest that more O₂ was available for delivery to the exercising tissues for oxidative metabolism by the mitochondria in the High group during the post-test. However, additional studies are necessary to determine if the one-day intervention was sufficient to induce an enhanced HVR and the extent to which such adaptations contribute to enhanced aerobic exercise performance.

The ventilatory responsiveness to hypoxia is highly individually variable. One study identified a blunted HVR in trained individuals when compared to untrained controls (Adamczyk, et al., 2006). This study conducted by Adamczyk, et al. examined ventilatory responsiveness to hypoxia in trained individuals when compared to untrained, sedentary controls. However, participants in the present study were all highly trained individuals with high levels of aerobic capacity (VO₂ max range: 45.4 – 59.9 ml/kg/min). The extent to which different levels of aerobic capacity (VO₂ max) alter the HVR in highly trained individuals is unknown, and warrants further investigation.

Exercising & Recovery HR

During maximal exercise at high altitude, an increase in cardiac output is essential for maintaining O₂ delivery to the tissues, as the O₂ available progressively decreases with increasing altitude. Dehydration resulting from respiratory and evaporative water loss during and after test runs contributes to a decrease in blood plasma volume resulting in a decreased stroke volume (SV). As a result, HR might increase to compensate for a

decreased SV to maintain cardiac output and O₂ delivery to tissues (a phenomenon known as “cardiovascular drift”).

In the present study, resting HR tended to be higher from pre to post-test in both groups at elevations 12,900 feet and below, which may have been in compensation for a decreased stroke volume (SV). Therefore, it is possible that HR was increased from pre to post-test due to a decrease in SV. A decrease in SV might have resulted from a decrease in plasma volume of blood via evaporative respiratory and sweat water loss (i.e. dehydration) during the test runs; however these parameters were not assessed in this study. It is also possible that HR was higher due to a greater sympathetic stimulation during the post- vs. pre-test, perhaps due to enhanced anxiety or motivation associated with a post-training assessment, but this is not clear from our study.

Hematocrit

Increases in hematocrit through stimulation of erythropoiesis can take up to three weeks of altitude exposure (Rodriguez et al., 2000). Therefore, as seen in the present study, a single high altitude exposure will not be sufficient to elicit erythropoiesis responses to increase hematocrit. During exercise at high or low altitude, respiratory water loss and sweat loss can contribute to a decrease in blood plasma volume, which results in an increase in red blood cell concentration. Conversely, well hydrated subjects will have a lower hematocrit due to a greater blood plasma volume. Therefore, the wide variability of HCT values in the present study likely resulted from variations in hydration status of the subjects.

RPE

RPE values were not significantly different within or between groups from pre to post-test. RPE steadily increased at each individual data collection site, with the highest values being observed at the 14,100 foot summit. This would be expected as exercise becomes progressively more difficult as PO_2 drops with increasing altitude. Both pre and post-test runs were maximal effort, timed runs at an altitude of 11,500 – 14,100 feet. Therefore, it is evident that during a maximal effort timed run at high altitude, RPE values would tend to be towards the maximal perceived exertion score (17 – 20). Improvements in perceived exertion would not be expected during maximal effort runs at high altitude; rather, improvements would be seen in running performance, as the runner would just be able to run faster while still experiencing maximal RPE scores.

Post VO_2 max

Post VO_2 max assessment of aerobic capacity revealed that both High and Low altitude training interventions did not impact aerobic capacity throughout the duration of the study. Increases in aerobic capacity can be attained through high levels of aerobic training. All subjects were highly trained, fit individuals with relatively high aerobic capacities. Therefore, increases in VO_2 max were not expected to be seen throughout the study.

Implication of Results

We have observed that a single high altitude training bout can elicit improvements in SaO_2 responses in a subsequent high altitude endurance event one week later. In addition, although statistical significance was not reached for run-time results, the High group tended to improve running performance from pre to post-test. These findings may

have positive implications for athletes that are unable to properly acclimatize to high altitude multiple days to weeks before a high altitude event, such as athletes residing at or just above sea-level. Therefore, a shorter acclimatization protocol, such as a single high altitude bout separated by multiple bouts at lower altitude appears to be sufficient to elicit at least some acclimatization responses to altitude.

Suggestions for Future Studies

The results of the present study suggest that a single high altitude training bout may confer benefits to athletes performing at altitude one week later, possibly by increasing SaO₂ through an enhanced HVR. However, further studies with additional subjects should be conducted to improve the statistical power of the study and further elucidate the mechanism(s) responsible.

CHAPTER V

SUMMARY AND CONCLUSIONS

High altitude hill running events such as the Pikes Peak Marathon have become increasingly popular among athletes around the World. The sport of high altitude hill running imposes physiological challenges that greatly exceed those presented in flat land races at lower elevations. It is presently thought that to fully acclimatize for such an event, an individual must receive multiple days to weeks of altitude exposure. However, this is not feasible for many athletes who reside at or just above sea-level. Therefore, the aim of the present study was to examine whether a shorter altitude acclimatization protocol, such as a single high altitude training bout would be sufficient enough to elicit physiologic and performance enhancing benefits for improved performance in a subsequent high altitude event. This is an altitude acclimatization protocol that has not previously been examined.

To examine the question of interest, we implemented a single training bout performed either on the Mount Evans Scenic Byway outside of Idaho Springs, Colorado (High group; elevation range 12,750 – 14,100 feet) or on Towers Road in Horsetooth Mountain Park in Fort Collins, Colorado (Low group; elevation range 5,550 – 7,170 feet). Prior to the single training bout, subjects completed an 11.5 mile pre-test run on

the Mount Evans Scenic Byway. Run-time, SaO₂, HR and RPE were all assessed at miles 2, 6, 9 & 11.5 (summit). One week after the single training bout, subjects completed an identical 11.5 mile post-test run with run-time, SaO₂, HR and RPE being assessed at miles 2, 6, 9 & 11.5 (summit).

We were able to observe improvements in SaO₂ responses in the High altitude training group along with a trend for improvements in total run time, while no significant differences were observed in the Low altitude group. In addition, improvements in recovery SaO₂ responses within the High altitude training group were identified. Therefore, we were able to conclude that a single high altitude training bout performed on the Mount Evans Scenic Byway was able to improve SaO₂ responses in a subsequent high altitude event one week after the training routine, which may lead to improvements in running performance.

Study Limitations

The current study presented a couple of limitations which compromise the ability to extrapolate the findings to a wide range of athletes. First off, given the nature of the study being a field study, certain conditions are out of the research teams' control. Weather conditions during the first attempted pre-test resulted in failure to allow the subjects to test and failure to collect data. This led to scheduling conflicts which resulted in a mismatch in aerobic fitness levels (VO₂ max) between the two experimental groups. Therefore, the High altitude group was a less aerobically fit group, which compromises the end results because evidence suggests that altitude acclimatization may elicit superior benefits in a "less fit" population. In addition, certain runners experienced extremely high wind conditions (>20 mph) during the post-test which could have compromised

their running performance. Second, a sample size of 8 subjects (4 High, 4 Low) reduces the statistical power and the ability to extrapolate the findings to a wide range of athletes.

Future Directions

First and foremost, additional subjects should be added to the Low and High groups to confirm the validity of the observed trend for improvements in total run time in the High group. In addition, due to the fact that the two experimental groups had a mismatch in aerobic fitness levels, we would like to add some subjects with higher VO_2 max values to the High altitude group and lower VO_2 max values to the Low altitude group to better match the aerobic fitness levels between groups. This would allow us to extrapolate the findings to a wider range of athletes with varying fitness levels and rule out any effects of aerobic fitness in the observed effects. In addition, we would like to identify whether a “hypoxic ventilatory response” was induced in the High altitude group by measuring ventilation during the pre and post-test runs. To do this, we would need to change the nature of the study into a controlled environment such as a hypobaric chamber. Ideally, we would like to implement the same protocol but do so in a more controlled manner. Not only would this allow us to measure variables such as ventilation, but it would allow us to control the environmental conditions so that we don’t experience limitations such as high winds during the post-test.

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APENDIX I

Health History, Running History and Training Questionnaire

Name: _____ Phone #: _____

E-mail: _____

Address: _____ Sex: _____

_____ Age: _____

ID# (Randomly assigned by research group) _____

Health History:

1. Do you currently have any health conditions or injuries that might affect or limit your ability to participate in weekly, high-intensity hill training sessions this summer? Please explain.

2. Are you currently using medications that may affect your running?

Running History:

1. How many years have you been running?

2. How many years have you been trail running? Flat land running?

3. Please provide a list of race events and times (approximately) you have completed in the last 12 months:

- a. 5K:

- b. 10K:

- c. ½ Marathon:

- d. Marathon:

- e. Other:

Training History:

1. Describe *in detail* your last 4 weeks of training:

2. Briefly describe your last 12 months of training:

3. How many miles per week do you run presently (Trail vs. flat)?

4. What is your max mileage you have you run in 1 week?

5. What is your average pace per mile for an easy effort?

Miscellaneous:

1. Are you able to commit to weekly trail running sessions with study groups on Sundays during the months of June and July? Please note any weekends you know that you'll be away and unable to participate.
2. Participants will be semi-randomly assigned to groups engaging in weekly training sessions at high (Pikes Peak) or low (Horsetooth Mountain park) altitude training sites. While we cannot guarantee your placement in one or the other group, do you have a preference?
3. Are you registered for the Pikes Peak Marathon or Pikes Peak Ascent?
4. Do you own a Heart Rate monitor? Is it capable of keeping track of time, distance, altitude etc...?