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THE EFFECT OF MANIPULATING THE PRE-EXERCISE TEMPLATE ON TIME-
TRIAL PERFORMANCE

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Degree of Master of Science in Clinical Exercise Physiology

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THE EFFECT OF MANIPULATING THE PRE-EXERCISE TEMPLATE ON TIME-
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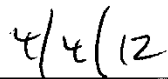
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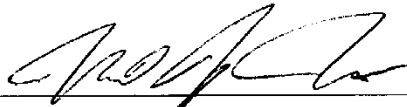
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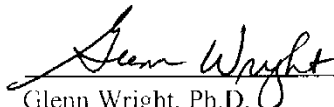
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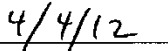
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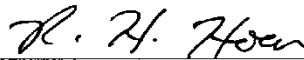


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ABSTRACT

Jaime, S.J. The effect of manipulating the pre-exercise template on time-trial performance. MS in Clinical Exercise Physiology, December 2012, 45pp. (C. Foster)

Introduction: The purpose of this experiment was to understand whether homeostatic disturbances, such as arterial oxygen saturation (S_aO_2), prior to exercise would reset the pre-exercise template, reflected by the power output (PO) during the first 1 km of a 5 km time trial (TT). **Methods:** Trained cyclists performed four randomly assigned trials with differing warm up (WU) and TT conditions, including NN, NH, HH, or HN ($H-F_{I}O_2 = 0.15$, $N-F_{I}O_2 = 0.21$) with the first and second letters depicting WU and TT conditions respectively. **Results:** Using either end-WU HLa, RPE, $\%S_aO_2$, or a multivariable product now known as the Desaturation Strain Index ($DSI=HR*HLa*(100-S_aO_2)$), the hypoxic WU was significantly harder than the control ($p<0.05$). There was no significant difference ($p>0.05$) between the WU conditions and the PO during the first 500m of the TT. **Discussion:** The central governor concept suggests that PO might be reduced following a more challenging WU, attributable to hypoxia, on the premise that the exerciser might expect a greater challenge to homeostasis. However, the results demonstrate that despite much more challenging conditions during the hypoxic WU, the PO during the opening part of the TT was not affected, demonstrating the robustness of the pre-exercise template.

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INTRODUCTION

Pacing strategy has been defined as the regulation of energy expenditure and resources to optimize performance during a race, with minimal wasted energy (Abbiss & Laursen 2008; Ansley et al. 2004; deKoning et al. 2011; Foster et al. 2002; Johnson et al. 2009; Joseph et al. 2007; Mauger, Jones, & Williams 2009a; Mauger, Jones, & Williams 2009b; Tucker & Noakes 2009). Pacing depends on a complex series of feed-forward/feedback mechanisms from both central and peripheral systems that detect what is experienced as fatigue, and regulate motor output accordingly. Pacing is also determined by other factors as well, such as anticipated race distance, feedback mechanisms, environmental changes, and individual capacity (Henslin et al. 2012; Johnson et al. 2009). Evidence has also been found that these pre-race plans are in place prior to the race (Ulmer 1996), and are learned by prior experiences with similar events (Foster et al. 2009; Mauger, Jones, & Williams 2009b). Ulmer (1996) has coined the term as teleoanticipation to explain this process. Ulmer demonstrated that a given athlete, with prior knowledge of their race distance, would be able to anticipate their pacing and initial exercise intensity prior to the race so as to optimize their time and energy efficiency.

In order to have an efficient pacing strategy and optimal race time, an athlete must be able to maintain homeostasis within the body so as to finish the race without unreasonably large homeostatic disturbances (Abbiss & Laursen 2008; Foster et al. 2002). However, the race must not be run in such a manner that the athlete becomes fatigued prior to the finish line. AV Hill, a Nobel Laureate in exercise physiology, presented data (Noakes, St Clair Gibson, & Lambert

2005; Swart et al. 2008) leading to the popular belief that athletes are unable to continue exercise due to some sort of catastrophic failure of internal homeostasis due to the lack of oxygen to the activated muscles or to depletion of energy reserves. This concept has been countered very strongly by Noakes et al. (2008). Evidence is accumulating in the favor of the Noakes' viewpoint, as he and his colleagues proposed what is now known as the Central Governor Model (Noakes 1998; Noakes, St Clair Gibson, & Lambert 2005; Noakes 2011; Tucker & Noakes 2009). The premise of this model is that there is a central governor that regulates the amount of motor units recruited for any given exercise. According to the viewpoint, it is not the catastrophic failure of peripheral muscles, but rather a decrease in motor unit recruitment as a consequence of afferent feedback that we perceive as fatigue (Noakes 1998; Noakes, St Clair Gibson, & Lambert 2005; Noakes 2011; Tucker & Noakes 2009).

To understand the concept of the Central Governor Model (Noakes 1998), we must be able to quantify what we perceive as fatigue. In 1982, the rating of perceived exertion (RPE) was created by studying a group of lumberjacks, and how hard they perceived to be working (Borg, 1982). Rating of perceived exertion was originally thought to be correlated with the subject's heart rate (HR) as the scale went from 6-20 (denoting 60-200 beats per minute). It was later realized that RPE was an accurate tool to predict the duration of an open-loop exercise (Crewe, 2008). Rating of perceived exertion is a great tool to measure the amount of effort one is putting into the task at hand and has been shown to increase during simulated competition (Joseph et al. 2008). Mauger, Jones, and Williams (2009c) found that exercise duration is regulated by pain levels, and these pain levels are correlated with RPE. Knowing this, along with the Central Governor Model, it is fair to state that fatigue is a complex and dynamic feedback mechanism used to communicate between the periphery to the central motor center in

order to regulate the intensity of the exercise. This helps the body to stay within the “safe” limit of homeostatic disturbance so as to avoid any catastrophe induced during maximal exercise.

Finally, it is known that different environment disturbances (such as altitude) can have detrimental effects on long-term aerobic performance. Mollard et al. (2007) observed this decrease in maximal oxygen ($\text{VO}_{2\text{max}}$) at higher altitude in trained and untrained subjects. This is because as altitude increases, the hemoglobin saturation is proportional to the decrease in the fraction of inspired oxygen ($\text{F}_\text{I}\text{O}_2$). Because the $\text{VO}_{2\text{max}}$ is the product of cardiac output and the difference in arterial and venous oxygen (a-vO_2) difference, changes in arterial oxygen content will affect the $\text{VO}_{2\text{max}}$. In contrast, it is note-worthy that when athletes were competing in supplemental hyperoxic conditions, performance was significantly better than at sea-level oxygen concentration (Amann et al. 2006; Tucker et al. 2007; Peltonen, Tikkanen, & Rusko 2001).

A recent study looking at the rapidity of response to a hypoxic challenge during a closed-loop exercise test was conducted in our human performance lab (Johnson et al. 2009). It was seen that PO and arterial oxygen saturation ($\text{S}_\text{a}\text{O}_2$) decreased with the initiation of hypoxic air during a 5-km time trial (TT). The abrupt decrease in PO during hypoxia suggests that the central governor can regulate PO very quickly from various receptors that are signaling low $\text{S}_\text{a}\text{O}_2$. A similar study administered a similar hypoxic challenge prior to the start of the closed-loop exercise test to observe the time required it took for the body to regulate PO, based on the fractional oxygen concentration (Henslin et al 2012). In both studies, the time required to decrease PO was about 40 seconds, and even though the PO was decreased, RPE was higher in the hypoxic conditions. These results suggest that there is a delay in the feedback/anticipation

mechanism when controlling workload. This increase in RPE can be seen as an increase in physiological stress on the subject during an exercise bout in a hypoxic environment.

Kitamura et al. (1972) and Nelson et al. (1974) were able to approximate how much a person's myocardial oxygen demand was by simply multiplying the HR by systolic blood pressure (SBP). This is called the double product or rate pressure product. This type of calculation has been utilized in other studies to understand the amount of physiological stress is endured by the subject given different variables, such as heat stress in firefighters. Rodriguez-Morroyo et al. (2011) was able to measure the stress induced by the work-load and heat during wildfire activities among firefighters. They were able to measure this based on a modified equation by Moran et al. (1998) and Frank et al. (2001) in order to calculate physiological strain index and cumulative heat strain index, respectively, and were significant enough to provide further evidence to use these equations are valid measurement techniques. These studies provide enough support to rationalize the use of combining multiple relatable variables to show the amount of physiological strain induced on a subject in hypoxic conditions. We call this product the Desaturation Strain Index (DSI), which is defined by multiplying HR, blood lactate (HLA), and O_2 desaturation ($100 - S_aO_2$). The higher the DSI, the more strenuous the workload perceived by the subject at any point, effectively showing how much their pre-exercise template might be altered and how much physiological stress is placed on the subject in regards to oxygen desaturation. The amount of DSI experienced by the subjects in regards to the initial power output at the start of the time trial will paint a clear picture to how strong their pacing template really is.

The purpose of this experiment was to understand how strong the pacing template really is when manipulated prior to competition by using hypoxic and normoxic conditions and how

humans regulate exercise intensity accordingly via afferent feedback. As the protocol calls for two separate experimental conditions, the hypotheses are two-fold. (1) It is expected that when warming up in a normoxic environment ($F_{I}O_2 = 21\%$), the initial spike in PO will remain unchanged regardless of the atmospheric environment at the start of the TT due to a lower DSI during the WU and (2) when warming up in a hypoxic environment ($F_{I}O_2 = 15\%$), the initial spike in PO will be significantly attenuated at the start of the TT due to a higher DSI during the WU.

MATERIALS AND METHODS

Subjects

Seven well-trained cyclists were recruited and provided written informed consent prior to participation. The protocol was approved by the University of Wisconsin – La Crosse Institutional Review Board for the Protection of Human Subjects. Table 1 contains the descriptive statistics of the subjects.

Table 1. Descriptive Statistics (mean \pm SD)

	Male	n=5	Female	n=2
Age(years)	27.8	± 5.02	22	± 1.41
Mass (kg)	75.4	± 5.69	64.4	± 2.17
VO _{2max} (l/min)	4.70	± 0.47	3.32	± 0.03
HR _{max} (bpm)	177	± 11.5	181	± 4.24
PO _{max} (watts)	360	± 28.5	288	± 17.7

Protocol

An initial incremental exercise test was conducted to evaluate the subject's VO_{2max}, peak power output (PO_{max}), and heart rate max (HR_{max}). This test was performed on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). The testing protocol

consisted of the subject pedaling at 25W for 2 minutes, and increased by 25W every minute until exhaustion. Respiratory gas exchange was measured using open circuit spirometry (AEI Technologies, Pittsburgh, PA). HR was recorded using radiotelemetry (Polar Vantage XL, Polar Instruments, Port Washington, NY).

Subsequently, each subject completed two habituation protocols, practicing the experimental protocol that consists of a 15 minute warm up (WU) on the same cycle ergometer utilized for the $\text{VO}_{2\text{max}}$ test (5 minutes at 100W, 5 minutes at 50% PO_{max} , 3 minutes at 75% PO_{max} , 2 minutes recovery at 100W), a 3 minute transition period at 25W (Foster et al. 2009), followed by a 5-km TT on another cycle ergometer (Velotron, Racermate, Seattle, WA). This habituation period served to familiarize the subjects with the protocol, the measurements, as well as the small amount of resistance by the respiratory apparatus.

The subjects then repeated the protocol 4 times, with at least 48 hours of rest in between each trial, with one of the TT's being a control and the other three experimental tests. During the trials, the subjects were exposed to either normoxic or hypoxic air through the intake valve of the respiratory mask during the 15 minute WU. During the final minute of the recovery period, the experimenter changed the fractional concentration of oxygen from the intake valve of the breathing valve in order to ensure appropriate S_aO_2 at the beginning of the TT (Johnson et al. 2009) which was measured with a pulse oximeter (Allegiance Oxi-Reader 2000, Allegiance Health Care, McGraw Park, IL). The experimenter did not change the F_iO_2 on two trials, but did on the other two. The subjects were single-blinded to the treatment. Each subject was randomly assigned to a normoxic WU and normoxic TT (NN) (Control); normoxic WU and hypoxic TT (NH); hypoxic WU and hypoxic TT (HH); hypoxic WU and normoxic TT (HN).

In order to further elucidate the challenge provided by the hypoxic WU, we used the aforementioned DSI as a similar analysis to the rate pressure product (Kitamura et al. 1972; Nelson et al. 1974) to gain a clear understanding of how difficult the hypoxic WU was compared to the normoxic WU.

Statistical Analysis

Data was analyzed using multivariate Analysis of Variance (MANOVA) in order to identify statistically significant differences in HR, RPE, HLa, S_aO₂, and DSI between warm-up conditions. Repeated measures Analysis of Variance (ANOVA) in order to identify statistically significant differences in PO, HR, RPE, HLa, and S_aO₂ between time trials. A P-value < 0.05 is considered to be statistically significant. Post hoc tests were performed when justified by ANOVA using Fisher's LSD test.

RESULTS

Warm Up

The results for both WU conditions are reported below in tables 2, 3, and 4. There was no significant difference (p=0.430) in HR between WU conditions (Figure 1). There was a significant difference (p=0.044) in RPE between conditions (Figure 2). Blood lactate was significantly higher (p=0.002) than normoxic conditions during the hypoxic WU (Figure 3). S_aO₂ was significantly lower (p=0.000) during the hypoxic WU (Figure 4). The DSI was significantly higher (p=0.000) during the hypoxic WU (Figure 5).

Table 2. Warm-Up Results

Time (minutes)	Mean HR (bpm) (N)	Mean HR (bpm) (H)	Mean O ₂ (%) (N)*	Mean O ₂ (%) (H)*
5	110 ± 8	115 ± 18	98.2 ± 1.7	88.3 ± 2.8
10	139 ± 11	143 ± 22	97.5 ± 2.3	85.9 ± 2.3

	13	162 ± 8	163 ± 19	97.1 ± 1.6	84.1 ± 2.2
	15	127 ± 8	130 ± 13	98.1 ± 1.4	87.2 ± 2.9

*p≤0.05

Table 3. Warm-Up Results (Cont.)

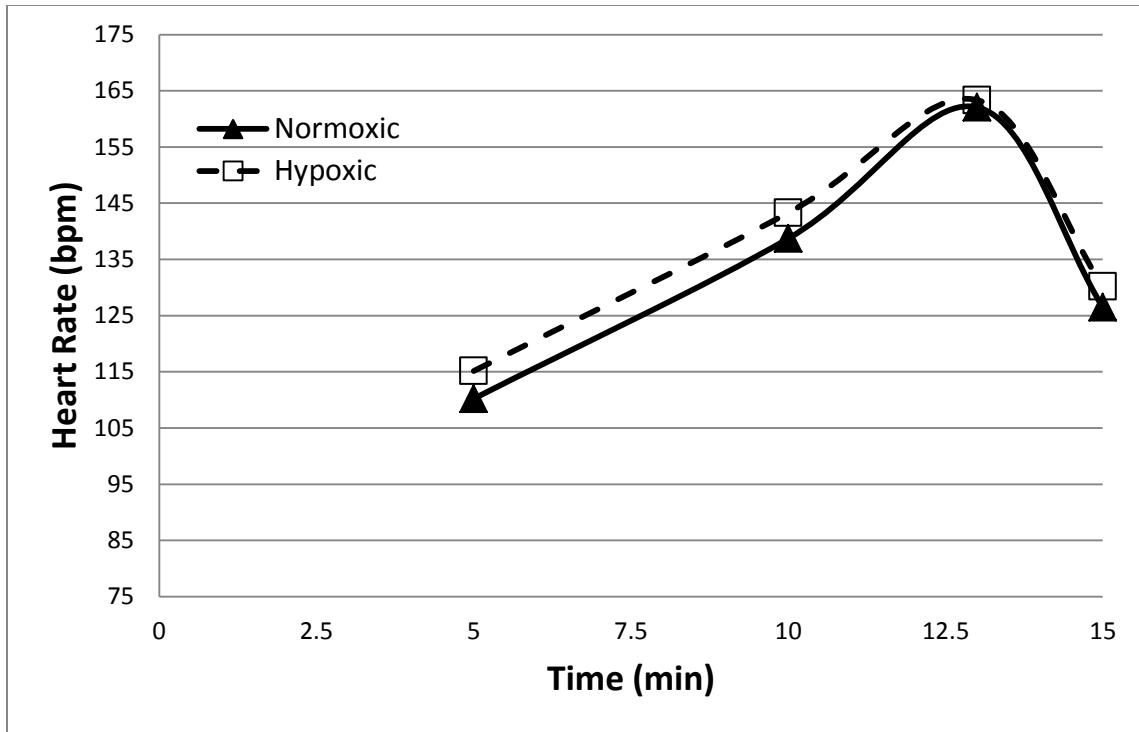
Time (minutes)	Mean HLa (mmol/L) (N)*	Mean HLa (mmol/L) (H)*	Mean RPE (N)	Mean RPE (H)
5	1.7 ± 0.6	2.4 ± 1.0	1.1 ± 0.5	1.3 ± 0.6
10	2.5 ± 0.5	3.9 ± 0.8	2.4 ± 0.8	3.3 ± 1.0
13	5.2 ± 1.2	7.7 ± 1.9	4.2 ± 1.3	5.9 ± 2.2
15	5.5 ± 1.5	8.2 ± 2.2	1.7 ± 0.5	2.4 ± 0.8

*p≤0.05

Table 4. Warm-Up Results (Cont.)

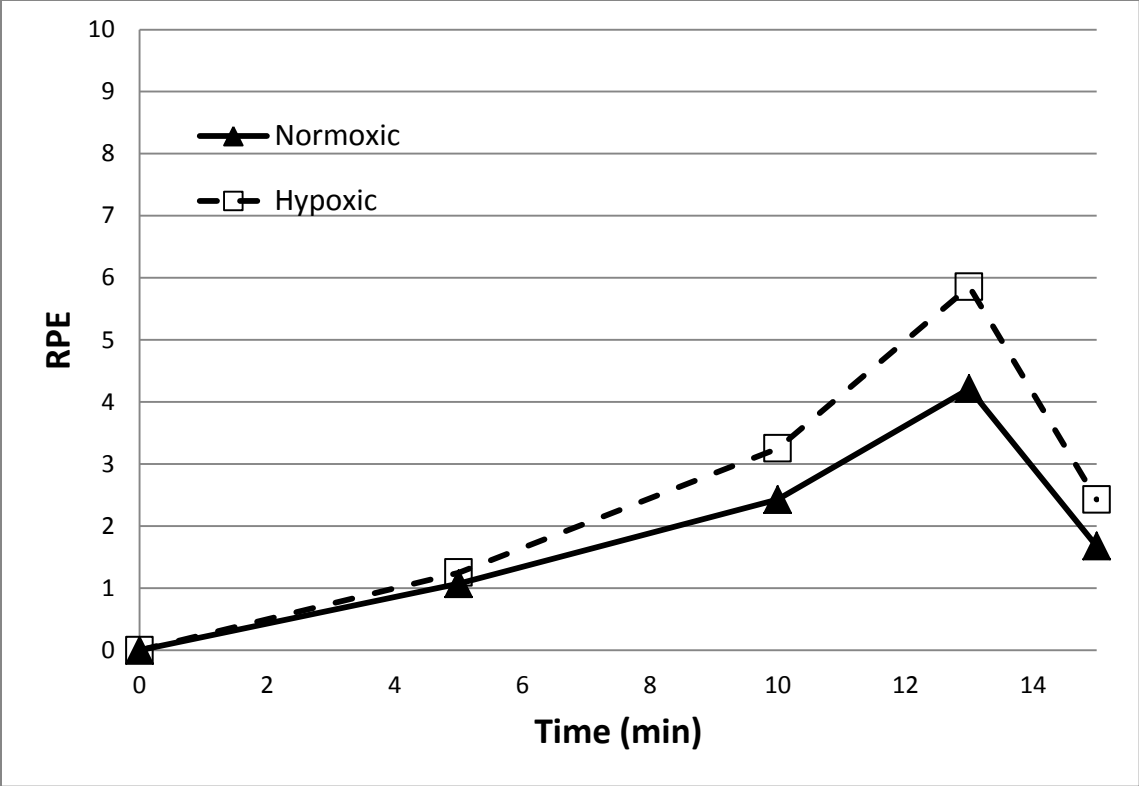
Time (minutes)	Mean DSI (N)*	Mean DSI (H)*
5	351.1 ± 167.2	3321.2 ± 1681.3
10	875.2 ± 565.6	8005.4 ± 2477.0
13	2465.3 ± 1555.2	20364.2 ± 8046.9
15	1364.7 ± 914.5	14511.7 ± 7065.1

*p≤0.05



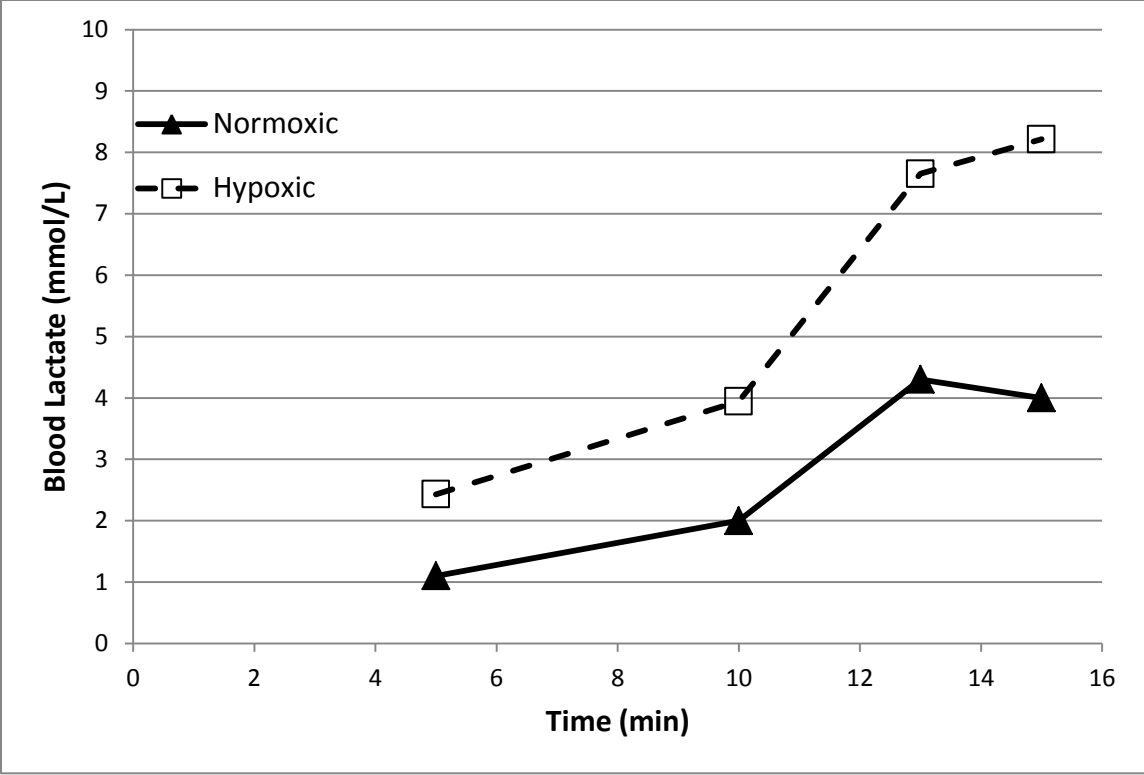
Figure

1. Heart rate vs. Time during warm up.



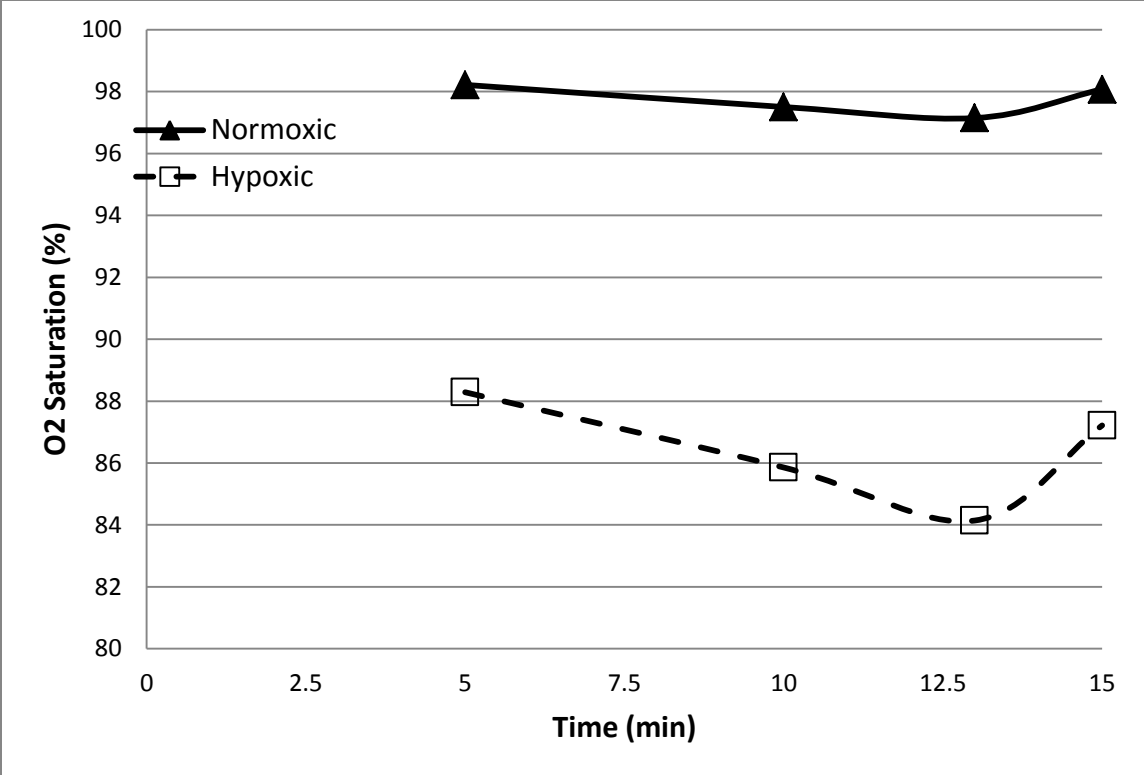
Figure

2. Rating of perceived exertion vs. Time during warm up.



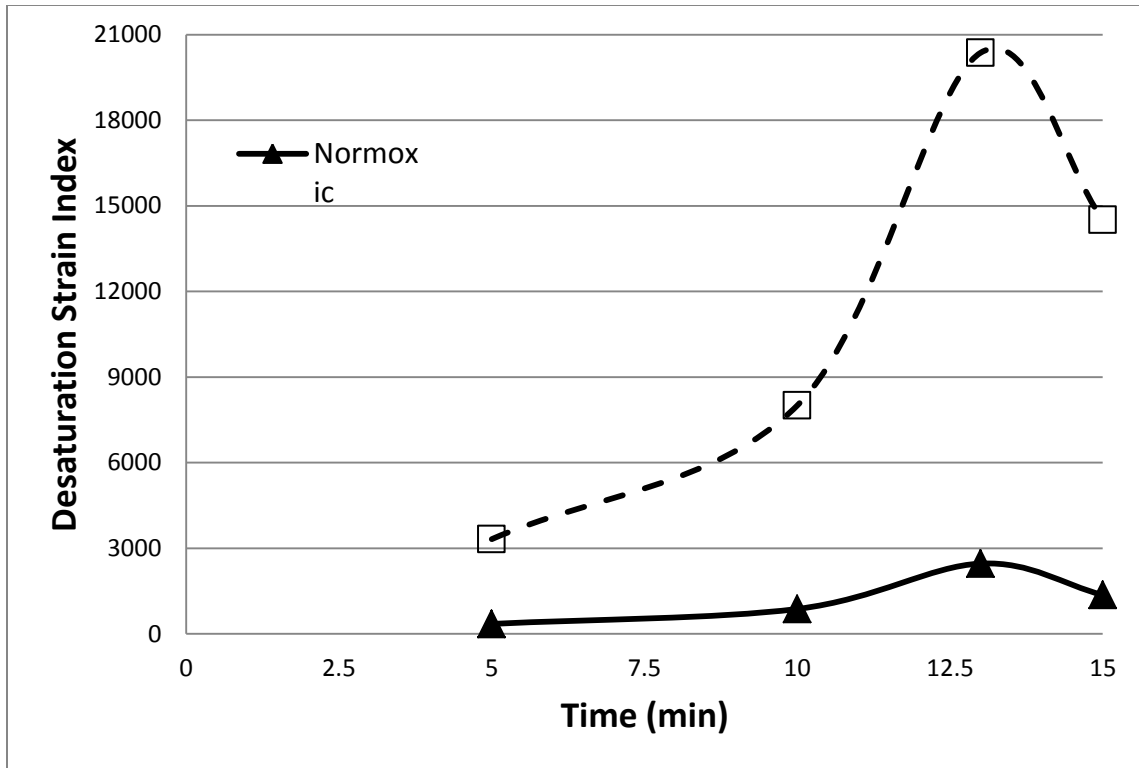
Figure

3. Blood lactate vs. Time during warm up.



Figure

4. Arterial oxygen saturation vs. Time during warm up.

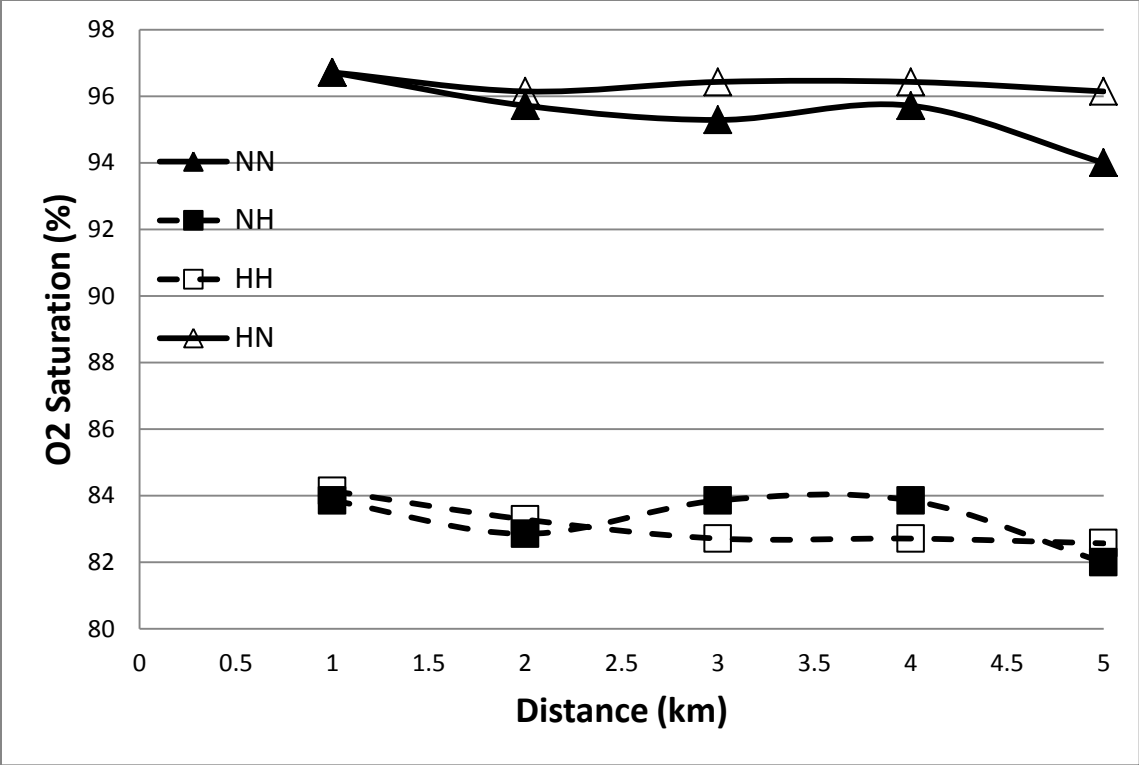


Figure

5. Desaturation strain index vs. Time during warm up.

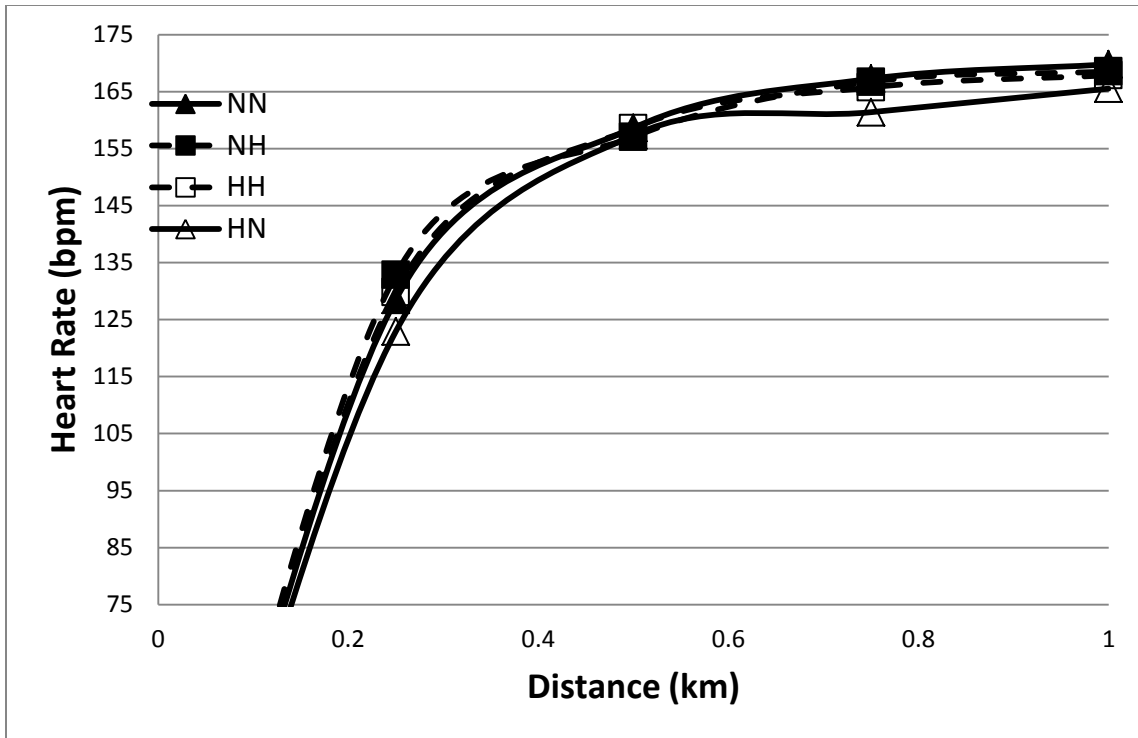
Time Trial

During the TT, S_aO_2 was significantly lower in the hypoxic condition, regardless of WU (Figure 6), yet still no significant difference in HR was seen between conditions (Figure 7). No significant difference was seen in PO between conditions during the first km, as the WU condition had no significant effect on initial PO (Figure 8). There begins a trend in decreasing PO during the TT (after 750 meters) between the hypoxic and normoxic conditions (Figure 9). During the TT, when in normoxia, the subjects were able to maintain a mean PO above 75% PO_{max} , whereas during hypoxia, the mean PO was below 75% PO_{max} (Figure 10).



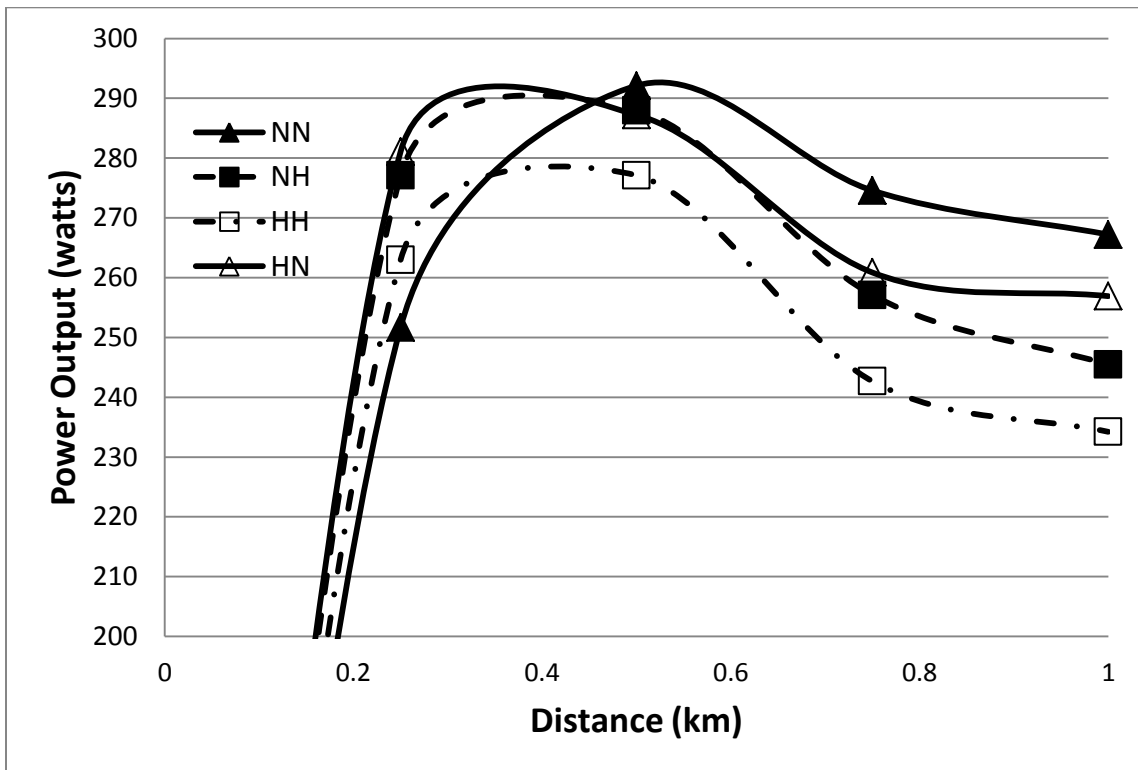
Figure

6. Arterial oxygen saturation vs. Distance during time trial.



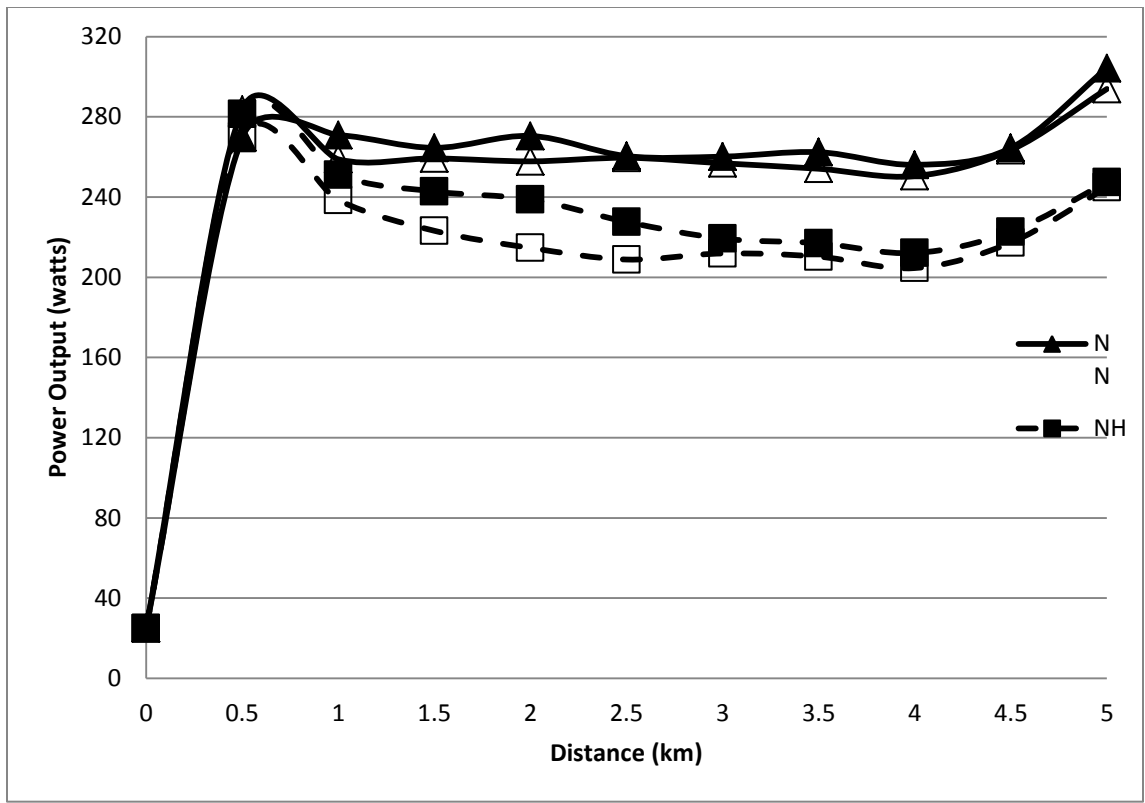
Figure

7. Heart rate vs. Distance during time trial.



Figure

8. Power output vs. Distance during initial km of time trial.



Figure

9. Power output vs. Distance during time trial.

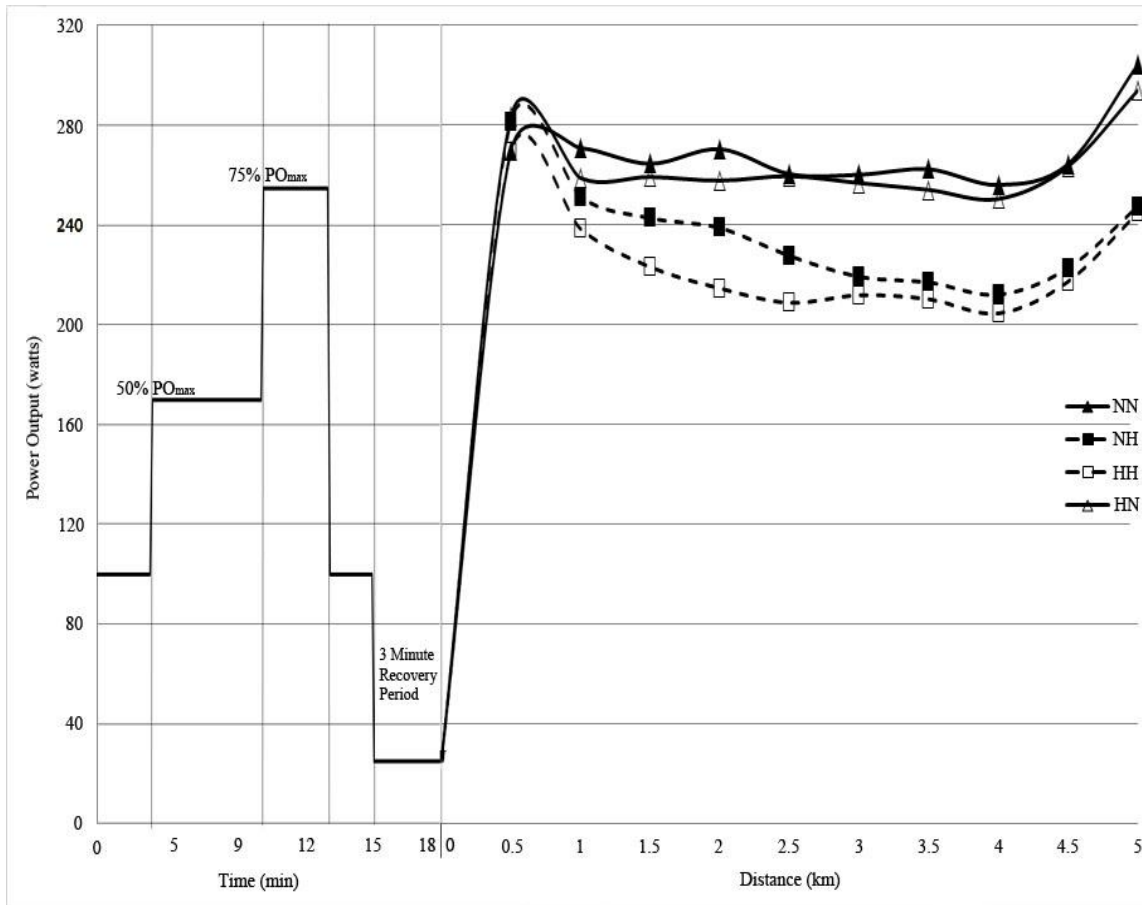


Figure 10. Power output vs. Time during warm up as it transitions to power output vs. distance during time trial.

DISCUSSION

The evidence supports our first hypothesis, in that the initial PO at the beginning of the TT was not changed post-normoxic WU regardless of TT condition. However, our second hypothesis is refuted because the initial PO at the beginning of the TT was not attenuated post-hypoxic WU. This is a significant finding because the RPE, HLa, and DSI were all significantly higher during the hypoxic WU. The WU was hard enough to potentially cause modification of the pre-exercise template. Because of the 5 minutes of recovery time at the end of the warm up and during the transfer to the time trial, it can be reasoned that PCr levels would replenish prior to the start of the time trial. Because is the source of the initial energy reserves, one may think that the athlete would tap into this in order to begin the time trial at the expected exercise intensity. However, based on the central governor theory (Noakes 1998), the hypoxic WU should have been expected to attenuate the PO at the beginning of the TT as enough time was given to acclimate for the change in $F_{I}O_2$ and the pre-exercise template was altered immensely. Not only were we able to make the hypoxic WU significantly more difficult for the subject, we also induced hypoxic air a minute before the start of the TT. Johnson et al. (2009) found that it took about 40 seconds post-hypoxic respiration for PO to decline. However S_aO_2 declined after 20 seconds, allowing for a 20 second delay before the central governor reduced PO. However, even in our study, when the WU was under hypoxic conditions as well as during the TT, the initial PO was still not attenuated. If the central governor was the main mechanism in controlling exercise intensity, it was not apparent in this study. Henslin et al. (2012) found that the time it took for the PO to decrease after administering the hypoxic air, was 40 seconds after the start of the TT further supporting our results.

When comparing the results of the mean PO during the TT to the PO during the WU, it was seen that those in normoxia were able to maintain a significantly higher PO ($>75\% PO_{\max}$) when compared to those in hypoxia ($<75\% PO_{\max}$). This is significant because it did not matter what the WU condition was, as long as there was normoxia during the TT.

Limitations of our findings could have been the effect of sea-level barometric pressure when inspiring hypoxic air. The respiration through the mask could have proved a challenge for some of the cyclists as they are not normally accustomed to having to breathe through a constricted airway. Finally, the cyclists were all physically fit, however it cannot be overlooked that they were most likely not all in the same category, nor were they all elite athletes.

The main finding in this study is that there was no significant difference in initial PO in the TT regardless of the how difficult the WU condition was perceived to be by the subject, providing evidence that the pre-exercise template is not easily changed and that the controlling mechanism that limits exercise intensity is not exclusive to the central governor model or peripheral fatigue model. Rather it seems to be a complex and dynamic interaction of both.

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APPENDIX A
INFORMED CONSENT

Protocol Title: The Effect of Manipulating the Pre-Exercise Template on Time-Trial Performance

Principal Investigator: Salvador Jaime
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(503)621-6422

Emergency Contact: Carl Foster
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- Purpose and Procedure
 - The purpose of this study is to determine the effect of pre-exercise template manipulation and its effect on time-trial performance.
 - My participation will involve one maximal exercise test, a habituation time-trial, and four separate time-trials (competitive simulation), all of which will be very fatiguing.
 - The total time requirement is 6 hours over a three-week period.
 - Testing will take place in the Human Performance Laboratory in Mitchell Hall at the University of Wisconsin-La Crosse.
 - During all tests, I will wear a snorkel-like apparatus to simulate normoxic and hypoxic conditions, as well as measure the respiration ratio. I will also wear a heart rate monitor strapped around my chest to ensure I am safely monitored during the exercise test.
- Potential Risks
 - I may experience fatigue and muscle soreness on the day of the test, and may experience it the following hours post-test.
 - Individuals trained in CPR, Advanced Cardiac Life Support, and First Aid will be monitoring my test, and it will be terminated should any complications occur.
 - The risk of serious or life-threatening complications, for health individuals like me, is near zero.
- Rights & Confidentiality
 - My participation is voluntary.
 - I can withdraw myself from this study at any time without the need of reason and without penalty.
 - The results of this study may be published in scientific literature or presented at professional meetings, however all data will be number-coded.
 - All information will be kept confidential and data sheets will be kept in a locked cabinet. My data will not be linked with personal information.
- Possible Benefits
 - There are no benefits to my participation in the study.

Questions regarding study procedures may be directed to Salvador Jaime (503-621-6422), the principal investigator, or the study advisor Dr. Carl Foster, Department of Exercise and Sport Science, UW-L (608-785-8689). Questions regarding the protection of human subjects may be addressed to the UW-L Institutional Review Board for the Protection of Human Subjects, (608-785-8124 or irb@uwlax.edu).

Participant: _____ Date: _____

Researcher: _____ Date: _____

APPENDIX B
REVIEW OF LITERATURE

INTRODUCTION

Pacing strategy has been defined as the regulation of energy expenditure and resources in a manner to optimize performance with minimal wasted energy at the end of the race, but not expend the energy too soon in order to avoid unreasonable homeostatic disturbances and become fatigued prior to the finish. This behavior to optimize time in a competitive event is particularly important because of the sheer amount of athletes that aspire to be the best in their respective sport. Though it is well known that athletes do in fact regulate their pace to avoid fatiguing prior to the finish line, the mechanism in which the body regulates exercise capacity is still obscure. Thus, pacing strategies are dependent on a multitude of factors that impact the performance of an athlete by regulating physiological responses throughout a race that include individual limits, anticipatory factors, prior experiences, feedback mechanisms, and environmental factors.

EXERCISE AND THE ENVIRONMENT

In order to understand the reasoning for some of the pacing strategies at altitude, we must first understand why it is we cannot perform as well at altitude. Between 1968-1971 there were a series of studies conducted on young and healthy athletes at a wide range of altitudes (Buskirk 1967; Faulkner 1968; Daniels & Oldridge 1970; Dill and Adams 1971). It was seen that VO_{2max} was reduced due to a decreased arterial PO_2 and a decreased maximal cardiac output (decrease SV due to decrease in plasma volume and increase in pressure, decrease HR due to increase parasympathetic tone and decrease O_2 to the heart). Trend of these studies were that athletes had a decrease in performance (VO_2 , Mile times, mean power output, etc...) while at altitude when compared to pre-altitude performance levels, however once they returned to sea level, their post-altitude performance was much better. It was these studies that have suggested the concept of

“live high, train low” theory that many endurance runners believe in. In theory, if you live high, your body makes the necessary physiological changes to maximize the amount of oxygen transported in your system (i.e. increased red cell mass) and training low allows the athlete to maximize the amount of training they can do. Though these studies are slightly older, they still hold value as the beginnings of exercise at altitude examinations. Recent studies over the years have shown that exercise performance decreased and rate of fatigue increased in direct relationship with increasing altitude (Peltonen 2001; Amann 2006a; Crewe 2008; Johnson 2009; Mollard 2007; Henslin 2012).

Peltonen and colleagues observed the determinants of maximal oxygen consumption at altitude and found that there was a decrease in VO_{2max} , power output (PO), maximal cardiac output (Q_{max}) in hypoxic conditions, with increases in hyperoxic conditions in relation to normoxic conditions. Differences between arterial and venous oxygen saturation (oxygen extraction) and a decrease in cardiac output (oxygen transportation) can explain the reduction in VO_{2max} . The reduction in PO may be due to the reduction in skeletal muscle recruitment. If this is the case, the central nervous system has down regulated the exercise capacity in order to limit the increase in cardiac output.

Mollard et al (2007) compared 8 endurance trained subjects to 8 untrained subjects. Arterial oxygen saturation decrease at altitudes at and above 1000 meters, as well as decreased in VO_{2max} , HR_{max} , and oxygen transport, with a small increase in oxygen extraction. Stroke volume and cardiac output stayed constant, despite the decrease in HR in response to an increase in altitude. Aerobic training increases the athlete’s oxygen transportation through an increased heart rate max and the ability to extract oxygen. In this study, it was seen that oxygen extraction

had reached a maximal limit yet it was not enough to compensate for the significant decrease in oxygen transportation.

Knowing that athletes have a large decrease in aerobic performance during exercise at altitude, it can be used as a great tool to understand what other factors influence pacing strategies when there is a large decrease in arterial oxygen saturation at altitude.

PACING STRATEGIES AND RATING OF PERCEIVED EXERTION

In 1982, the rating of perceived exertion (RPE) was created by studying a group of lumberjacks, and how hard they perceived to be working (Borg, 1982). RPE was originally thought to be correlated with the subject's heart rate as the scale went from 6-20 (denoting 60-200 beats per minute).

Foster et al. (2003, 2004) observed subjects during middle-distance time trials. The athletes were given only one instruction, and that was to finish the race as fast as possible. It was seen that the strategy used was to preserve enough anaerobic energy so as to have an end-spurt at the end of the race. Athletes seem to manage their energy resources so as to not become fatigued too early in the race and still have enough anaerobic energy to accelerate near the end of the race.

Crewe, Tucker & Noakes (2008) observed that the rate of increase in RPE throughout an exercise bout is indicative of exercise duration in different environmental conditions. RPE was seen to increase during prolonged exercise at a fixed workload. Because exercise is terminated when RPE reaches maximal levels, the exercise duration can be predicated from the slope of increasing RPE. This signifies that RPE is an accurate tool to predict the duration of open-loop exercise, but closed-loop simulated competition was still in question.

Joseph et al. (2008) demonstrated that subjects have a linear increase in RPE throughout the duration of a closed-loop exercise bout in both normoxic and hypoxic conditions. This is significant because even though the hypoxic conditions significantly decreased the power output in the respective trials, RPE was consistent in both the control and experimental trials demonstrating that RPE has scalar properties related to duration of the exercise. It is seen that the RPE scale is a great tool to measure the amount of effort one is putting into the task at hand and has been shown to increase during simulated competition

TELEOANTICIPATION AND PRIOR EXPERIENCES

To establish an efficient pacing strategy, an athlete must have prior experience in a similar event. This prior experience will allow the athlete to develop an anticipatory mechanism to judge their pace depending on the amount of distance that is remaining in the current exercise bout. This is known as teleoanticipation (Ulmer 1996). In his study, the rise in RPE progresses in a linear fashion from the beginning of exercise showing that the pace is set prior to the exercise bout so as to anticipate the difficulty of the race and maintain homeostasis and reduce the decrease in performance prior to the finish.

For a clearer understanding of teleoanticipation and its involvement in pacing strategies Foster et al. (2003, 2004) observed how athletes regulated their metabolic reserves. With the data of these studies, they coined the term 'Internal negotiation' which is the preventative effect of the central governor to inhibit catastrophic failure due to homeostatic disturbances using feed-forward mechanisms and afferent feedback from the peripheral system. The athlete is dynamically comparing how they feel to how they should feel bases on a prior experience learned from a similar event. This expectation of how they should feel is considered the pre-

exercise template and is the basis to how the athlete is to distribute his or her energy throughout an exercise bout limiting the amount of homeostatic disturbances. This is quantified by the use of RPE in order to understand the sensation of fatigue induced by several factors during exercise.

Mauger et al (2009b) recently investigated the influence of external feedback and prior experiences on time trial performances. The control group knew distance of time trial and received distance feedback throughout each trial. The experimental group received neither feedback nor knew the distance needed to be traveled, but did know that each trial was equal in distance. Mean PO, VO_{2max} , end RPE, and $\%HR_{max}$ were all similar once the experimental group completed their 4th time trial. Peak power output was significantly less in the experimental group all the way through. Integrated Electromyography (iEMG) tracked mean PO was observed in the control group but not in the experiment group. iEMG research is needed with self-paced exercise. What was interesting is that they had the experimental group shout out their RPE after each kilometer they believed to have passed. They did not perform this task with any accuracy, but this appears to point to the fact that knowledge of absolute distance is not necessary, rather the athlete must create a relative distance to be completed subconsciously, allowing for the implementation of a basic pacing strategy. When no feedback is given, RPE seems to be the most important factor. This learning process will impact the next race if it is similar in duration, and is considered to be an athlete's pre-exercise template (Foster et al. 2009). The athlete must have this prior experience in order to anticipate the difficulty of the race, and understand how they will feel throughout the given bout. In order to full comprehend the pre-exercise template, Mauger et al. (2009c) studied the effect of time trial performance in well trained cyclists in which they were either given correct feedback during the race or incorrect feedback, designed to confuse their learned pre-exercise template in a competitive simulation

time trial. Those who were given correct feedback achieved greater performance than those who were given non-contingent feedback. It is seen that if no feedback is given, RPE is a useful tool in intratrial pacing, however if feedback is given and incorrect, then it confuses their momentary RPE when compared to pre-trial expectations.

Athletes whose goal in a race is optimized time will either decrease or increase their velocity at a given moment based on their “Hazard Score” (deKoning et al. 2011). deKoning et al. observed that an athlete is able to compare how they feel in certain point in the race to the perceived exertion from prior learning experiences in a similar event. Using this, they are able to either, increase their velocity if they are feeling better than expected, or decrease velocity if they are feeling a greater exertion than that of a similar event. Using the RPE, it was hypothesized that they were able to figure out whether or not an athlete will change their velocity using what was coined as the “Hazard Score”, which is the product of RPE and the fraction of remaining distance. This was a significant study because it allows us to understand the relationship of RPE and the amount of distance left in a race to an athlete’s pacing strategy and pre-exercise template. If they do not think that they feel as good as they should during that portion of the race, they will most likely slow down, or a high hazard score. But if they feel better than anticipated, as in their RPE is lower at that moment than previously experienced, a low hazard score is observed along with an increase in energy expenditure. They are making a conscious effort to maintain energy reserves until they are needed for an end spurt near the terminal portion of the race.

FEEDBACK MECHANISMS

AV Hill, a Nobel Laureate in exercise physiology, suggested that athletes are unable to continue exercise due to some sort of catastrophic failure to the internal homeostasis due to the

lack of oxygen to the active muscles or depletion of energy reserves (Noakes, St Clair Gibson, & Lambert 2005; Swart et al. 2008). Such claims were countered very strongly by Tim Noakes and many of his colleagues. Evidence is currently compiling in the favor of Noakes, as he and his colleagues proposed what is now known as the Central Governor Model (Noakes 1998; Noakes, St Clair Gibson, & Lambert 2005; Noakes 2011; Tucker & Noakes 2009). According the Hill's cardiovascular/anaerobic model, once a plateau has been reached in a maximal exercise test, cardiac output cannot increase anymore. If maximal CO is attained, then myocardial infarction will set in due to the inability to deliver enough oxygen for the heart to contract properly. However during a maximal exercise test, with no regulation system to stop the cardiac output from rising to maximal limits, why don't we see this regularly? Myocardial infarction and other cardiovascular events are rarely seen in healthy subjects, therefore must be another mechanism in which regulates exercise capacity. Noakes suggests that a limit to exercise capacity is not that simple, but a much more complex and dynamic mechanism. Though skeletal muscle anaerobiosis does have an effect on exercise capacity, it is not the limiting factor, but rather a piece in a much larger puzzle in which our body attempts to maintain a reasonable range of homeostasis in order to avoid catastrophic events. He states that if there is an absence of myocardial infarction during maximal exercise testing as high as 8848 meters, with the arterial oxygen saturation so low that it was almost not enough to provide enough oxygen to maintain cerebral function, then it can be concluded that there must be a central governor that reduces the exercise capacity.

Noakes is not the only one fighting the uphill battle against popular exercise physiology theory. Ansley et al. (2004) Studied power output, oxygen consumption, and muscle recruitment during 3 consecutive 4k cycling time trial. Subjects instructed to complete TT in fastest time

possible, the only feedback given was distance covered. Peak power output was highest in the first trial then the second and third, signifying that the pacing strategy is modified and implemented between trials. If peripheral fatigue actually manifested itself, power output would decrease throughout the trial; however an increase of power output was seen. iEMG activity should have risen unproportional to the power output if peripheral fatigue was the limiting factor. It is unknown whether or not pacing strategy is influenced by metabolic events or preemptive feedforward information.

Amann et al. (2006a) studied the effects of changing the concentration of arterial oxygen (17.6-24.4 ml O₂ dl⁻¹) over 4 trials of 5k cycling time trials (closed-loop exercise). Quadriceps fatigue pre- and post-test for every trial via nerve stimulation while central neural drive was measured via quadriceps electromyography. Central neural drive, time trial performance, and power output increased as concentration of arterial oxygen increased. Concentration of arterial oxygen saturation (C_aO₂) effects power output in a closed-loop exercise test. In addition to this study, Amann & Dempsey (2008) shed further light on the topic, observing the effect of this locomotor fatigue on central neural drive. Based on these results, it was hypothesized that the effect of C_aO₂ on locomotor muscle power output and exercise performance time is determined to a significant extent by the regulation of central motor output to the working muscle in order that peripheral muscle fatigue does not exceed a critical threshold.

Studies on drugs that have effects on the brain has also shed some light in favor of the central governor theory. Swart et al. (2008) examined the effects of amphetamines on exercise performance. The experimental group was given amphetamines and an open-loop exercise test to exhaustion was performed on both the experimental group and control group. The experimental group had a significantly greater performance than the control group. This

suggests that the central nervous system regulates exercise performance to regulate homeostasis. iEMG decrease in both groups as power output decreased. If peripheral fatigue was the main factor, the decrease in motor unit recruitment would not have occurred.

Mauger, Jones & Williams (2009a) also tested subjects who were under the influence of a considerable amount of acetaminophen, a pain killing ingredient found in many over-the-counter medications. It was seen that those subjects who were on acetaminophen were able to achieve better time trial performance times because they were not experiencing the same amount of pain that is relative to the perceived exertion. They were able to increase their speed because the RPE at a certain point in the race did not increase as much as anticipated prior and during the race. Finally, to drive in the point that exercise capacity is not driven and regulated by a single component, Swart et al. (2011) separated the physical from the mental aspect of RPE. To lessen the confusion, they named the physical aspect P-RPE and the mental aspect TEA. Subjects were able to efficiently differentiate the two from one another. During the maximal effort 100-km time trial, P-RPE reached near maximal values, supporting the notion that afferent feedback is the regulatory factor in exercise performance. In a closing statement, Noakes suggests that rather than having a single model of peripheral fatigue, that we should aim our research to a more complex concept of peripheral and central fatigue in which regulates performance to avoid catastrophic failure

CONCLUSION

There have been many studies observing the limiting effects of both peripheral and central fatigue with the main goal being trying to find the metaphorical golden ticket. Though much is known about pacing strategies in athletes during both simulated and recreational

exercise, little is known about the regulation of physical capacity during exercise or about the effects of how the body regulates pacing strategy when environmental conditions are rapidly shifted between the warm-up and exercise bout.

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