

**EFFECT OF BRIEF-INTERMITTENT HYPOXIC  
EXPOSURE ON HIGH-INTENSITY KAYAKING  
AND CYCLING PERFORMANCE**

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**Attestation of Authorship**

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made.

Darrell Bonetti -----

Date -----

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## **Publications and conference presentations arising from this PhD Thesis**

Chapters 2-5 of this thesis represent four separate papers that have been published or submitted to peer-reviewed journals for consideration for publication. These papers were prepared in collaboration with my supervisors Professor Will Hopkins and Dr Andrew Kilding. The percentage of my own work in these papers is noted in brackets.

### **Published peer-reviewed articles**

Bonetti DL, Hopkins WG, & Kilding AE (2006) High-intensity kayak performance after adaptation to intermittent hypoxia. *International Journal of Sports Physiology and Performance*, 1, 246-260. (Darrell Bonetti 75%, Will Hopkins and Andrew Kilding 25%)

### **Peer-reviewed articles currently under review**

Bonetti DL, Hopkins WG, Kilding AE, Lowe T (2007) Cycling performance following adaptation to two protocols of acutely intermittent hypoxia. *International Journal of Sports Physiology and Performance*: Submitted in December 2007. (Darrell Bonetti 75%, Will Hopkins and Andrew Kilding 25%)

Bonetti DL, Hopkins WG (2007) Sea-level exercise performance following exposure to natural and artificial altitude- a meta-analysis: *Sports Medicine*: Submitted in December 2007. (Darrell Bonetti 60%, Will Hopkins 40%)

Bonetti DL, Hopkins WG (2007) Variation in performance in elite flat-water canoeists from race to race: *European Journal of Sport Science*: Submitted in July 2007. (Darrell Bonetti 60%, Will Hopkins 40%)

### **Conference Presentations**

Bonetti, DL & Hopkins WG (2003). Effective use of Incremental Step Tests. *Proceedings of the New Zealand Conference of Sports Medicine, Nelson, New Zealand.*

Bonetti, DL, Hopkins WG & Kilding AE (2005). High intensity kayak performance after adaptation to intermittent hypoxia. *Proceedings of the New Zealand Conference of Sports Medicine, Queenstown, New Zealand.*

Bonetti, DL, Hopkins WG, Kilding AE & Lowe TE (2007). Cycling performance following adaptation to two protocols of acutely intermittent hypoxia. *Proceedings of the European College of Sports Science, Jyvaskyla, Finland.*

Bonetti, DL, Hopkins WG, Kilding AE & Lowe TE (2007). Cycling performance following adaptation to two protocols of acutely intermittent hypoxia. *Proceedings of the New Zealand Conference of Sports Medicine, Hamilton,, New Zealand.*

Bonetti, DL & Hopkins WG (2007). Sea-level exercise performance following adaptation to hypoxia: a Meta-Analysis. *Proceedings of the New Zealand Conference of Sports Medicine, Hamilton, New Zealand.*

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## Abstract

Adaptation to the shortage of oxygen at altitude (hypoxia) promotes physiological changes which could enhance endurance performance. Consequently, altitude training has become a popular practice among competitive endurance athletes. Since its inception, the live-high train-low paradigm (LHTL) has been widely regarded as the most effective approach to altitude training. Over the past decade, brief intermittent simulation of LHTL via the use of hypoxic inhalers and re-breathing devices has gained increased popularity, but the evidence supporting their use is limited and conflicting. The experimental studies in this thesis investigated the response of sea level exercise performance and related physiological measures following adaptation to the usual and a novel protocol of brief intermittent hypoxia. I intended to perform all experimental studies on flat-water kayakers. Therefore, an initial requirement of this thesis was to establish the smallest worthwhile effect in performance for this sport. The final study utilising a meta-analytic approach was conducted to compare the effectiveness of brief intermittent hypoxia to other natural and simulated protocols, and to investigate the topical issue of what physiological responses mediate performance changes following hypoxic exposure.

In Study 1, the typical variation in competitive performance of elite flat-water canoeists was investigated using a repeated-measures analysis of race times. For individual flat-water canoeing events, the smallest worthwhile change in performance time was ~0.5%. In two separate experimental studies, adaptation to 60 min per day of brief intermittent hypoxia consisting of alternating 5 min intervals of hypoxia and normoxia for 3 weeks (5 days per week) using a nitrogen filtration device resulted in clear enhancement of endurance performance (~5%) for kayakers (Study 2) and cyclists (Study 3). Clear enhancements in repeat sprint performance were observed for kayaking only. The physiological mechanisms underlying performance changes were unclear. Modification of the hypoxic and normoxic intervals (Study 3) did not result in any clear alterations in performance or physiological mechanisms. The meta-analysis (Study 4) revealed clear enhancements in endurance power output of 1-3% in sub-elites following adaptation to hypoxia with the natural altitude protocols, and with two of the artificial-altitude protocols (LHTL-long and LHTL-brief-intermittent). In elite athletes the enhancements tended to be smaller and were clear only for the natural protocols. These enhancements could be mediated by VO<sub>2</sub>max, although other mechanisms may be possible.

# CHAPTER 1

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## Preface

### Thesis Rationale

Living and training at altitude or more recently living at altitude and training at sea level is a practise employed by numerous athletes attempting to improve endurance performance. For athletes who reside in New Zealand limited venues exist for altitude training, with the "Snow farm" based in Wanaka the most popular and practical venue utilised. It offers an altitude range of 1500-1980m and relatively quick access to near sea level altitudes (300 m). However, for many athletes living at this venue can cause disruption to their normal training environment and lifestyle, additionally appropriate facilities for some athletes are not available. There is therefore considerable interest from New Zealand sport scientists, coaches and athletes to consider simulated altitude exposure using tents and intermittent hypoxic devices.

The primary focus of this thesis was to investigate performance adaptations to intermittent hypoxia and I had originally intended to use flat-water kayakers for all my experimental studies. For the investigation of the performance enhancement in any sport, researchers need to know the smallest worthwhile change in performance, which is derived from the typical within athlete variation between competitions that will increase an athletes chance of medalling. However, at the onset of this thesis no published research on the variation in performance for flat-water kayakers existed. In Study 1, utilising a repeated measures analysis of race times for elite flat-water canoeists over a four year period I was able to determine the variability in competitive performance and consequently calculate the smallest worthwhile change in performance. Knowledge of the smallest worthwhile change in performance would inform interpretation of results in subsequent intervention studies as part of this thesis.

Acutely intermittent hypoxia is a method of simulating the live-high train-low model which has gained increased popularity in New Zealand over the past 5 years. During intermittent hypoxia the stimulus is provided either by adding additional nitrogen to the ambient air, oxygen filtration of the air, or by using re-breathing devices. These methods reduce the partial pressure of oxygen to levels that are experienced at medium to high altitude (3000-6000 m). Therefore, the athlete's normal training routine and

environment remains unaltered but they are exposed to a hypoxic environment for intervals of 5-7 minutes followed by a similar period of ambient air for a total of 60-90 minutes per day over several weeks. While this method has been extensively used in New Zealand, only limited research has been conducted supporting its effect on sea level exercise performance. Some investigators have reported enhancements in endurance performance (Hamlin & Hellemans, 2004; Wood, Dowson, & Hopkins, 2006) and anaerobic performance (Wood, Dowson, & Hopkins, 2006). Conversely others investigators report little or no change in endurance performance (Clark, Dixon, Gore, Martin, & Hahn, 1999; Julian et al., 2004 and Tadibi et al., 2007).

The physiological demands of flat-water kayaking require development of both aerobic and anaerobic capacities, making them an ideal population to study. To date no studies have been conducted on flat-water kayakers. Therefore, the focus of Study 2 was to determine the effect of acute intermittent hypoxia on exercise performance in flat-water kayakers.

The results of Study 2 provided good evidence that acute intermittent hypoxic exposure improves both high intensity and submaximal exercise performance. It also brought about some questioning of the mechanisms mediating the performance change. Recently, it has been shown that hypoxia promotes an inflammatory response (Shah, Allen, Wood, & Gonzalez, 2003). Furthermore, inflammation is also present following strenuous exercise (Camus et al., 1994), and it is possible that this inflammation may be a contributing mechanism in adaptation to the stress of exercise. Whether an inflammatory response is linked to physiological or performance adaptations following intermittent hypoxia is unknown, but investigating cytokine and general inflammatory responses to hypoxia may help to address this question. Therefore it is likely that a number of mechanisms could contribute to enhancing exercise performance following hypoxic exposure. Investigation of both traditional and alternative mechanisms would enhance knowledge of the adaptation process to hypoxia and potentially influence the prescription of hypoxic exposure protocols for athletes.

Currently, all brief intermittent hypoxia research has utilised a protocol of repeated 5- or 6-min hypoxic intervals followed by a similar period of normoxia. It is possible that manipulation of the duration of the hypoxic and normoxic intervals could result in larger performance gains. For example, shorter hypoxic intervals with less normoxic recovery would increase the number of on-off transitions for a given time period,

which could increase activity of hypoxia-inducible factor, a transcription factor mediating physiological responses to hypoxia (Semenza et al., 2000). Therefore the focus of Study 3 was to investigate the effect of manipulating the hypoxic and normoxic interval duration and to determine whether any change in performance was associated with changes in inflammatory, haematological or hormonal markers.

I decided to use cyclists rather than kayakers since an analysis of reliability of the performance measures following the previous study revealed a higher than anticipated error of measurement for kayak ergometry. A further small pilot study revealed that some minor modifications to the kayak ergometer may improve the error of measurement. A reliability study (Appendix B) was conducted to determine the reliability of the ergometer with its modifications. Unfortunately reliability was only slightly improved and the decision was made to change to a different mode of exercise. Cyclists were selected as the participants of this study due to their known low typical error of measurement in laboratory performance tests (Paton & Hopkins, 2006). The cyclists were competitive over long distance races and so arguably did not possess the same anaerobic attributes as the kayakers. However, this was not an issue for the second experimental study since the focus was on endurance, inflammatory, hormonal and haematological adaptation to intermittent hypoxia. An added bonus of selecting cyclists was that we were able to access a far larger sample size than we could with kayakers, an important consideration especially for the analysis of haematological and hormonal parameters, which are known to have a considerable error of measurement.

Various reviewers have concluded that living at altitude and training at sea-level (live high train low) can have small benefits for sea-level endurance performance, while the effects for living and training at altitude are unclear (Baker & Hopkins, 1998; Levine, 2002; Rusko, Tikkanen, & Peltonen, 2004; Wilber, 2001). Reviewers have also been uncertain about the effectiveness of simulating the live high train low paradigm, with long continuous application of the model regarded as having effects on sea-level performance similar to that experienced in a natural altitude environment (Levine, 2002; Rusko, Tikkanen, & Peltonen, 2004; Wilber, 2001). The efficacy of shorter intermittent application of LHTL, which was originally pioneered in the former Soviet Union (Serebrovskaya, 2002), is unclear (Levine, 2002; Rusko, Tikkanen, & Peltonen, 2004; Wilber, 2001). To date, no review of adaptation to natural and artificial hypoxic exposure has used a meta-analytic approach to investigate changes in sea-level

performance or to investigate the topical issue of what physiological responses mediate the changes. Therefore, for the final study we chose to conduct a meta-analysis specifically addressing these issues. This decision was made after a traditional literature review had been performed as part of my PhD proposal (Appendix A) and after all experimental studies were completed. By performing the meta-analysis towards the end of my PhD I was able to incorporate a number of recent studies (including my own) investigating exercise performance following adaptation to intermittent hypoxic exposure.

### **Originality of the Thesis**

- Currently, there is limited (and contradictory) research on the effects of brief intermittent hypoxia on high-intensity sea-level performance, the outcomes of this thesis contribute substantially to the body of knowledge.
- Apart from one conference abstract, there have been no published attempts to refine or improve the protocols via altering the hypoxic interval and recovery ratio.
- No other study has investigated the inflammatory and hormonal response following adaptation to intermittent hypoxia.
- No other study has investigated the magnitude of the smallest worthwhile enhancement in performance for elite flat-water canoeists competing in different race distances.
- This is the first study to present a detailed meta analysis of all studies (both natural and simulated) that have investigated the effect of hypoxic exposure on exercise performance and physiological mechanisms

### **Thesis Organisation**

This thesis consists of five chapters. The chapters are presented in the format of the journal for which they were written. The references for each chapter are retained in the respective chapters and are collated at the end of the thesis in APA format.

Chapter 2 is a statistical analysis investigating the variability of competitive kayak performance which has been submitted to the *European Journal of Sport Science*. Chapters 3 and 4 are the two experimental studies. Study 2 has been published in *International Journal of Sports Physiology and Performance* and Study 3 has been

submitted to the *International Journal of Sports Physiology and Performance*. Chapter 5 is the main review of the literature for the thesis, on the effect of natural and simulated altitude exposure on sea level performance. This is in the form of a meta-analysis paper and has been submitted to *Sports Medicine*. The final chapter consists of a general discussion of the implications of the results of this thesis for athletes, coaches and sport scientists.

The appendices are presented either in hard form or with the attached CD. Appendix A contains a summary of my original literature review from my PhD proposal. Appendix B contains a reliability study (written as a short technical report) which was completed to determine test retest reliability of the kayak ergometer after some modifications which were considered after the first experimental study. Appendices C-E contain subject information and ethical approval for the two experimental studies and the reliability study. The attached CD contains data and statistical analyses from studies 1-4.

## CHAPTER 2

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### Variation in Performance of Elite Flat-Water Canoeists from Race to Race

#### Abstract

In sports where athletes compete for the best time or distance, the smallest change in performance affecting a top athlete's chances of winning is  $\sim 0.5\times$  of the race-to-race within-athlete variation in performance. We report here the variations in performance and smallest worthwhile changes for elite athletes competing in 200-, 500- or 1000-m flat-water canoeing events (men) and kayaking events (men and women) at international regattas. The events were the A and B finals held at 7-13 regattas in 2003-2007, with a total of 49-82 athletes and a mean of 1.7-2.7 entries per athlete. A linear mixed-model analysis of log-transformed official race times provided estimates as coefficients of variation and included terms to account for changes in performance between years, venues, and A and B finals. For men the within-athlete variation in A finals was similar in canoeing and kayaking events, with a possible small increase in variation for increasing distance (0.9%, 1.1% and 1.2% for 200-m, 500-m and 1000-m events; 90% confidence limits  $\times/\div 1.34$ ,  $\times/\div 1.12$  and  $\times/\div 1.25$  respectively) that may reflect differences in pacing strategies. For women kayakers in A finals the within-athlete variation in the 500-m event (0.7%;  $\times/\div 1.22$ ) was very likely less than the variation in the 1000-m (1.2%;  $\times/\div 1.33$ ) and 200-m (1.5%;  $\times/\div 1.29$ ) events, possibly because of differences in competitive experience and depth of competition between these events. Within-athlete variation in the B finals was generally greater than in the A finals for the three distances for men, probably reflecting less consistency in motivation or pacing, but there was no clear pattern for women. We conclude that the smallest worthwhile changes in performance time in canoeing and kayaking are  $\sim 0.3$ - $0.6\%$ . Effects of 1-2% in power output would be required to achieve such changes, because power output is proportional to the cube of speed in this sport.

## Introduction

One of the aims of sport research is to enhance performance of elite athletes. To achieve that aim researchers need to know the smallest worthwhile change in competitive performance: that is, the change that would increase their medal prospects substantially. Statistical simulations have shown that the smallest worthwhile change is about half the within-athlete variation in performance between competitions (Hopkins, Hawley, & Burke, 1999). An estimate of the smallest worthwhile change can therefore be determined from an analysis of a series of competitive performances. The previous published studies in this area for elite athletes have been limited to swimmers (Pyne, Trewin, & Hopkins, 2004), triathletes (Paton & Hopkins, 2005), track and field athletes (Hopkins, 2005) and cyclists (Paton & Hopkins, 2006). We address here the magnitude of variability in performance time for elite flat-water canoeists.

## Methods

### *Subjects*

Individual flat-water canoeing is contested in still water over three distances: 200 m, 500 m and 1000 m, using either a canoe (men only) or kayak (men and women). Official race times for all distances and events were obtained from websites for all flat-water World Cup, World Championship and Olympic Games regattas held between 2003–2007. Data were collected and analysed only for A and B finals, as they represented the races in which all athletes would make a maximal effort. The descriptive statistics for the races we were able to analyse are shown in Table 2.1.

### *Statistical analysis*

We derived estimates of variability in performance of individual canoeists from race to race using procedures described previously for cyclists (Paton & Hopkins, 2006). Flat-water canoeing competitions present a novel challenge for the analysis in that there is an “A” and “B” final at most competitions. We included both finals in the analysis to compare estimates of variability of athletes of the highest calibre with those in the next tier. Analyses were performed with the mixed linear modelling procedure (Proc Mixed) of the Statistical Analysis System (Version 9.1, SAS Institute, Cary, NC). The only fixed effect in

the model was a term to estimate the mean time for A and B finals. The random effects were as follows: athlete identity (to estimate true differences in mean ability between athletes); the interaction of athlete identity with year of competition (to estimate within-athlete variation between seasons); competition identity (to estimate variability in competition mean time between competitions); and the interaction of competition identity with the A and B final term (to estimate variability in the difference between A and B final mean times between competitions).

Race times were log transformed for the analysis, because this approach yields variability as a percent of the mean (coefficient of variation), which is the natural metric for most measures of athletic performance (Hopkins, 2000). The coefficients of variation were derived by back transformation of variances representing random effects in the mixed model. The difference between the mean times for the A and B finals was also expressed as a percent by back transformation.

We reported uncertainty in estimates as 90% confidence limits, For comparisons of coefficients of variation we made probabilistic magnitude-based inferences as described elsewhere (Batterham & Hopkins, 2006) using a published spreadsheet (Hopkins, 2006). The comparison was performed using the ratio of the coefficients of variation, which was presumed to have a log-normal sampling distribution. The thresholds for smallest worthwhile differences were factors of  $\times 1.15$  and  $\div 1.15$  (Hopkins & Hewson, 2001). A ratio was deemed unclear if its confidence interval overlapped these thresholds; otherwise the magnitude of the effect was reported as the magnitude of its observed value, sometimes with an assertion about the probability the effect was substantial, as given by the spreadsheet.

We dealt with outliers at two stages of the analysis. The raw performance times for each sex and event were displayed as frequency distributions and normal probability plots for identification of obvious coding errors and of athletes who clearly “threw in the towel”. This process resulted in deletion of five canoeing times, three male kayak times and one female kayak time. During the modelling stages performance times with residuals  $>4.0$  were deleted before re-analysis (three observations for the men’s canoeing and two for women’s kayaking).

TABLE 2.1. Simple statistics for the number of A or B race finals entered by flat-water canoeists and kayakers who competed in international regattas in 2003-2007.

	Number of athletes	Race entries per athlete		
		Mean	Maximum	Sum
<b>Men's Canoe</b>				
1000 m	82	2.4	12	193
500 m	74	2.6	12	194
200 m	61	1.7	6	104
<b>Men's Kayak</b>				
1000 m	82	2.6	13	214
500 m	79	2.7	12	213
200 m	72	1.8	8	132
<b>Women's Kayak</b>				
1000 m	72	1.9	6	134
500 m	70	2.6	9	179
200 m	49	2.0	6	97

## Results

### *Effect of ability*

It is apparent from Table 2.2 that the B final was slower than the A final in all distances and events: the mean difference was 1.3-4.9% for men and 2.3-3.2% for women (90% confidence limits;  $\pm 0.7$  to  $\pm 3.1$ ). Table 2.2 shows that the B finalists were also more variable in their race-to-race performance, by an overall factor of 1.39 ( $\times/\div 1.15$ ). The remaining results are mainly for the A finals, because the smallest worthwhile changes come from an analysis of the best athletes.

### *Effect of event*

As shown in Table 2.3, the within-athlete variation was similar in canoeing and kayaking, although the 500-m was possibly more variable in kayakers. When the three distances were averaged, kayaking was slightly more variable than canoeing, but the difference was unclear (ratio 1.09; 90% confidence limits  $\times/\div 1.29$ ).

### *Effect of gender*

Overall, there was little difference between the variability for the men's and women's kayaking (means both 1.1%), but the difference was unclear (ratio 1.04;  $\times/\div 1.22$ ).

### *Effect of distance*

In the A finals there was a different pattern in the variability over different distances for the men in comparison with the women. Averaging the variability of the men's canoeing and kayaking for each distance revealed a trend towards a reduction in variation between the 1000-m (1.2%;  $\times/\div 1.25$ ), 500-m (1.1%;  $\times/\div 1.12$ ) and 200-m (0.9%;  $\times/\div 1.34$ ). A substantial difference between the 1000-m and 200-m was likely, while substantial differences between the 1000-m and 500-m and between the 500-m and 200-m were possible. In contrast, for women there was no trend towards a reduction in performance with shorter event duration: the within-athlete variation in the 500-m event for women was smallest (0.7%;  $\times/\div 1.22$ ) and was very likely less than the variability in the 1000-m (1.2%;  $\times/\div 1.33$ ) and in the 200-m (1.5%;  $\times/\div 1.29$ ).

TABLE 2.2. Descriptive statistics for competitive performance times of elite flat-water canoeists and kayakers in the A and B finals. The coefficient of variation (CV) is the between-athlete variation in each race averaged over all races in a given event.

	A final				B final		
	Number of races	Mean time (s)	CV (%)	Best time	Number of races	Mean time (s)	CV (%)
<b>Men's Canoe</b>							
1000 m	12	241.2	2.0	226.2	10	251.6	2.0
500 m	12	112.2	1.7	106.4	10	116.5	2.5
200 m	7	42.0	2.2	39.6	5	43.9	3.8
<b>Men's Kayak</b>							
1000 m	13	216.1	2.2	204.5	11	221.3	1.7
500 m	13	101.5	1.5	96.9	11	103.2	1.9
200 m	8	36.9	1.8	35.2	7	38.2	2.2
<b>Women's Kayak</b>							
1000 m	10	248.1	2.1	234.7	5	261.6	1.8
500 m	12	113.7	1.8	107.7	8	117.6	1.5
200 m	8	43.1	2.4	40.2	3	43.7	1.6

### *Season-to-season variation*

Athletes who competed in events for more than one year showed additional variation from one season to another that was estimable as a positive coefficient of variation only for five of the nine events (range 0.6–1.7% ; 90% confidence limits  $\sim \times/\div 2.0$ ).

### *Variation in mean race time*

Mean race time at each competition (including A and B finals) displayed a variation between competitions with a range of 1.3-3.1% ( $\sim \times/\div 1.7$ ) over the nine canoeing and kayaking events. The mean variation was 2.5% ( $\times/\div 1.19$ ).

TABLE 2.3. Within-athlete variability of elite flat-water canoeists between international competitions in A and B finals, expressed as a coefficient of variation (CV) with factor 90% confidence limits ( $\times/\div$ CL).

	A final		B final	
	CV (%)	$\times/\div$ CL (%)	CV (%)	$\times/\div$ CL (%)
<b>Men's Canoe</b>				
1000 m	1.2	1.47	1.5	1.29
500 m	1.0	1.17	1.7	1.25
200 m	0.9	1.66	3.2	1.33
<b>Men's Kayak</b>				
1000 m	1.3	1.27	1.1	1.23
500 m	1.2	1.17	1.5	1.25
200 m	0.9	1.33	1.9	1.23
<b>Women's Kayak</b>				
1000 m	1.2	1.33	1.1	1.73
500 m	0.7	1.22	1.5	1.26
200 m	1.5	1.29	0.9	1.57

## **Discussion**

We performed this study primarily to determine the variability in competitive performance of elite flat-water canoeists from race to race. We have found interesting effects on variability that shed light on the nature of flat-water canoeing. The estimates of variability also provide estimates of the smallest worthwhile changes in performance.

Our finding that the A finalists were less variable than the B finalists is consistent with other studies showing that the better athletes have smaller variability in performance (Hopkins, 2005; Paton & Hopkins, 2006; Pyne et al., 2004). This difference in variability has been attributed to better pacing, more consistent preparation, or more consistent motivation on the part of the better athletes (Hopkins, 2005). For canoeing and kayaking it seems reasonable that all these factors could contribute to the difference in variability shown between the A and B finalists. It should be noted however that the present analysis

differs a little from that of previous studies of competitive performance, because the times for the B finalists contain times for athletes who are sometimes in A finals. To some extent therefore the greater variability of the B finals represents less consistency in the performance of athletes when they are in a B final compared to when they are in an A final.

The comparisons of the within-athlete variabilities of men vs women and canoeing vs kayaking were unclear, but the observed differences with this sample of athletes and races were trivial. In the absence of more data it is reasonable to assume a single value of ~1% at the elite level in this sport. The pattern of increasing variability with increasing distance for men is similar to that observed in swimming (Pyne et al., 2004), track running (Hopkins, 2005) and track cycling (Paton & Hopkins, 2006), possibly reflecting the fact that the shorter events are performed all-out, whereas pacing could create additional variation as the event duration increases. There may be a similar underlying trend with women, but we found the greatest variability in the 200-m event, possibly due to less competitive experience or depth of competition in this event.

The previous published analyses of competitive performance have not addressed the issue of variation in an athlete's performance between seasons. Some of our estimates are at best approximate because of the limited number of athletes competing for more than one season, but the average of ~1% probably applies to all events.

The smallest worthwhile change in performance for competitive athletes is derived from their race-to-race variability under the assumption that they compete as independent individuals and that they attempt to perform their best in each race (Hopkins et al., 1999). Flat-water canoeists race in lanes and are not permitted to ride the wakes of other paddlers to assist their progress along the length of the course, so there is no concern about the independence of their performances through physical influences. It is also the experience of one of the authors (D.L.B.) that competition at the elite level is so close that athletes invariably give their best effort. The smallest worthwhile changes in performance time derived from the variability in competitive performance for the 1000-m, 500-m and 200-m events are therefore ~0.6%, ~0.6% and ~0.4% for men, and ~0.6%, ~0.3% and ~0.7% for women. When averaged, the smallest worthwhile effect is ~0.5%. The estimate of within-athlete variability between seasons gives rise to another smallest worthwhile effect of 0.5%,

which represents the performance enhancement required to improve medal prospects consistently the following season.

Although the smallest worthwhile effect on performance time for canoeists is similar to those for competitive swimming (Pyne et al., 2004), track cycling (Paton & Hopkins, 2006) and track running (Hopkins, 2005), there is a fundamental difference between running and these other sports, including canoeing. In running there is a direct linear relationship between changes in running speed or time in time trials and an athlete's physiological ability to output power, but in sports where the athlete is using power output to overcome the effects of fluid resistance, changes in power are not directly proportional to changes in speed or time. Currently there are no published data on the relationship for canoeists, but it is likely to be similar to rowing, where a given percent change in time requires a threefold greater percent change in power output (Hopkins, Schabort, & Hawley, 2001). One practical consequence for canoeists is that strategies aimed at enhancing medal prospects by increasing physiological power output will need to increase power output by at least  $\sim 1.5\%$  ( $\sim 3 \times 0.5$ ).

The relationship between changes in power and performance time also has implications for our understanding of canoeing events. Canoeists are presumably no different from runners or indeed any other highly trained competitive athletes in the variability of their physiological power output from race to race. Elite track runners vary by 0.8% in their performance time from race to race, so the variability in their physiological power output is at most 0.8%. If the variability in a canoeist's time was only physiological, their race time should therefore vary by only  $\sim 0.3\%$  ( $0.8/3$ ), but in our analysis they vary by  $\sim 1\%$ . The additional variability must be something other than physiological. We believe it arises from environmental effects in this sport, because it is well known that in canoeing the lane draw impacts performance, depending on the environmental conditions. Different environmental conditions could also increase variability by favouring some athletes more than others; for example, some athletes perform better in a tail wind. Other evidence for environmental effects in canoeing comes from the variation in mean race time between races (2.5%). This variation has no other plausible source, because it is derived from a statistical model that adjusts for any difference in the calibre of athletes competing in each race.

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## CHAPTER 3

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### High-intensity kayak performance following adaptation to intermittent hypoxia

#### Abstract

Live-high train-low altitude training produces worthwhile gains in performance for endurance athletes, but the benefits of adaptation to various forms of artificial altitude are less clear. **Purpose:** To quantify the effects of intermittent hypoxic exposure on kayak performance. **Methods:** In a crossover design with a 6-week washout, we randomized 10 subelite male sprint kayak paddlers to hypoxia or control groups for 3 weeks (5 days per week) of intermittent hypoxic exposure using a nitrogen filtration device. Each day's exposure consisted of alternately breathing hypoxic and ambient air for 5 and 5 min respectively over 1 h. Performance tests were: an incremental step test to estimate peak power, maximal oxygen uptake, exercise economy and lactate threshold; a 500-m time trial; and 5× 100-m sprints. All tests were performed on a wind-braked kayak ergometer 7 and 3 days pre-treatment and 3 and 10 days post-treatment. Hemoglobin concentration was measured at 1 day pre-treatment, 5 and 10 days during treatment and 3 days following treatment. **Results:** Relative to control, at 3 days post-treatment the hypoxia group showed the following increases: peak power 6.8% (90% confidence limits,  $\pm 5.2\%$ ), mean repeat sprint power 8.3% ( $\pm 6.7\%$ ) and hemoglobin concentration 3.6% ( $\pm 3.2\%$ ). Changes in lactate threshold, mean 500-m power, maximal oxygen uptake and exercise economy were unclear. Large effects for peak power and mean sprint speed were still present 10 days post-hypoxia. **Conclusion:** These effects of intermittent hypoxic exposure should enhance performance in kayak racing. The effects may be mediated via changes in oxygen transport.

## Introduction

Hypobaric and normobaric hypoxic exposure in both real and simulated environments is commonly used by athletes in an attempt to improve high-intensity endurance performance. Several mechanisms linked to the transport and utilization of oxygen have been proposed as potential mediators of performance enhancement following hypoxic exposure: increased red-blood cell mass <sup>1</sup>, increased capillarisation of muscle <sup>2</sup>, increased myoglobin concentration <sup>3</sup>, increased muscle mitochondrial volume and aerobic enzyme activities <sup>2</sup>, and elevated muscle buffering capacity <sup>4</sup>.

Studies on athletes living and training at altitude have found an enhancement of endurance performance at altitude <sup>5,6</sup>. However since the athlete cannot exercise at the same intensity while at altitude a relative detraining effect can occur <sup>1</sup>. Currently, uncertainty exists as to whether sea-level performance is improved with this method. To prevent the detraining effect the live-high train-low method was devised <sup>7</sup>. The performance enhancements with this method are ~1-2% when athletes return to sea level <sup>8</sup>. However this method can be expensive and disruptive to the athlete's normal training and living environment. Over recent years, several forms of hypoxic exposure have been devised to reproduce the effects of living high and training low without the problems associated with real altitude exposure. Continuous daily exposures of 1.5-8 hours, simulating low to medium altitude utilizing either normobaric (nitrogen houses and tents) or hypobaric hypoxia (barometric chambers) have been extensively investigated. Enhancements in exercise economy <sup>9,10</sup>, endurance performance <sup>11-13</sup> and performance related hematology <sup>11-14</sup> have been reported with this approach.

Intermittent hypoxic exposure offers a more time- and perhaps cost-efficient method of simulating the hypoxia experienced at high altitude while the athlete remains at sea level. During intermittent hypoxia the stimulus is provided by adding additional nitrogen to the ambient air, oxygen filtration of the air, or by using re-breathing devices. These methods reduce the partial pressure of oxygen to quantities that are experienced at medium to high altitude (3000-6000 m). Thus, the athlete's normal training routine and environment remains unaltered but they are exposed to a hypoxic environment for intervals of 5-7 minutes followed by a similar period of ambient air for a total of 60-90 minutes per day.

While intermittent hypoxia appears to be a promising training method, there has been limited research supporting its efficacy or physiological adaptations. Some investigators have found enhancements in endurance performance <sup>15,16</sup>, repeat sprint performance <sup>15,17</sup> and performance related hematology <sup>16,18</sup>. Conversely, other investigators have found

little or no change in measures of aerobic or anaerobic performance<sup>19, 20</sup>. Given these inconsistencies, we have assessed the effect of intermittent hypoxia with sprint kayak paddlers. Individual sprint kayaking over the 500-m distance is an Olympic high-intensity endurance event taking 96-105 s to complete. Research has indicated that this event requires approximately a 65% contribution from the aerobic energy system and 35% from the anaerobic energy systems<sup>21</sup>, making it an ideal event to investigate the effects of intermittent hypoxic exposure.

## **Methods**

### **Study Design**

This study employed a crossover design. Subjects (n=10) were randomly assigned to two groups balanced for best on-water K1 500-m performance in the previous year. Once placed in the groups, subjects then performed 1 week of pre testing followed by a 3 week intervention of kayak training and 5 days per week of intermittent hypoxic exposure or kayak training alone. The same testing procedures were repeated at 3 and 10 days post intervention. Following a 6-week washout period each group received the other treatment.

### **Subjects**

The subjects were sub-elite kayak paddlers who had at least 2 years of national or international race experience and a 500-m time of <2 min. Prior to the start of the study all subjects had been training consistently for at least three months. The study took place during the competitive season. All gave voluntary informed consent as required by the institutional ethics committee.

### **Training and Diet**

During the study all subjects followed a prescribed training program. The weekly schedule consisted of a 7-km race, 6 paddling sessions at aerobic threshold, 4 interval paddles at race-specific intensities, and 2 resistance-training sessions. The subjects maintained their normal diet during the course of both interventions and were instructed to have an easy day of training prior to each testing session. All subjects underwent an assay for ferritin status before the start of the study. All subjects were found to be in the normal range and therefore no iron supplementation was administered.

## **Hypoxic Treatment**

The BodyO<sub>2</sub> ESR-10 (Altitude Science, Auckland, New Zealand) was used to create the normobaric intermittent hypoxic exposure. This device uses nitrogen filtration to reduce the oxygen content of the air that the subject breathes when connected to the system via a face mask. The degree of hypoxia was gradually increased throughout the duration of the experimental period by reducing the fraction of inspired oxygen (F<sub>I</sub>O<sub>2</sub>) and peripheral oxygen saturation as follows:

- Days 1-5: oxygen saturation = 90,88,86,86, 84%; F<sub>I</sub>O<sub>2</sub> = 12%
- Days 6-10: oxygen saturation = 82, 82, 80, 80, 78%; F<sub>I</sub>O<sub>2</sub> = 11%
- Days 11-15: oxygen saturation = 78, 78, 76,76,76%; F<sub>I</sub>O<sub>2</sub> = 10, 9%

This protocol was based on previous research<sup>15</sup> and previous experience of the manufacturer of the ESR-10. The athletes breathed hypoxic air for a duration of 5 mins followed by 5 minutes of ambient air, for a period of 60 minutes, 5 times per week. Peripheral oxygen saturation was monitored individually with pulse oximeters (Sport-Stat, Nonin Medical, Minneapolis, MN; accuracy claimed to be a standard deviation of  $\pm 2$  units of percent saturation for saturations of 70-100%) throughout each interval of exposure. Subjects were advised to remove the mask if their oxygen saturation dropped below the target level and then immediately reposition the mask when the oxygen saturation had returned to the target level. If the subject could not reach the desired level of oxygen saturation, the F<sub>I</sub>O<sub>2</sub> was reduced at the Body O<sub>2</sub> ESR-10. This individual monitoring ensured that all subjects received the same hypoxic stimulus.

## **Exercise Performance Tests**

All physiological and performance tests were conducted in a temperature controlled laboratory (19-21°C) over a 2 day period. A calibrated, wind-braked kayak ergometer (Dansprint, Hvidovre, Denmark) was used in all tests. The foot-bar position of the kayak ergometer was adjusted to resemble the paddler's own kayak prior to each test. The ergometer was interfaced with a computer that continuously measured, calculated and stored accumulated work and other associated work indices, using specifically designed software. Day 1 consisted of an incremental step test to exhaustion and Day 2 a 500-m time trial followed 20 min later by 5× 100-m sprints. Each athlete completed four of these sessions (two pre- and two post-treatment) for each treatment (hypoxia or control), making a total of 8 testing sessions for the entire study.

The incremental step test commenced at a workload of 50-110 W and increased by 20 W every 4 min until exhaustion. There was a 1-min rest period between steps, where capillary blood was sampled from an earlobe for measurement of blood lactate (Lactate Pro, Arkray, Japan). Breath-by-breath oxygen uptake (Metamax 3b, Cortex, Leipzig, Germany) and heart rate (Polar A1, Polar Electro, Kempele, Finland) were measured continuously throughout the test. Maximum oxygen uptake ( $\text{VO}_2\text{max}$ ) was determined as the highest 30-s value obtained during the test. A measure representing the individual lactate-threshold power was derived from the step tests as follows. We assumed a log-log relationship between lactate concentration and power output<sup>22</sup>. We used the Trend function in Microsoft Excel to fit straight lines to the pre- and post-treatment lactate plots, then predicted power output corresponding to a "midpoint" of lactate concentration in the step tests. The midpoint was found by averaging the minimum and maximum values of the log-transformed lactate concentrations from all four tests. A similar procedure was used to create individual power profiles of heart rate and exercise economy; these variables did not require log transformation and power was calculated at fixed percentages of the individual's maximum value (heart rate 90%, exercise economy 70%).

The following day, each subject completed a simulated 500-m race on the kayak ergometer. Prior to the start of each test the subject had a 15 minute warm up period. This consisted of 2 mins of easy paddling then 8 mins of paddling at 70% of their peak power, then 5× 10-s efforts at 200% of peak power, performed every minute. The subject was then allowed 5 min to rest prior to the start of the race simulation. To ensure pacing was consistent throughout the 500-m simulation, subjects used an identical pacing strategy for each simulation. The strategy required each athlete to work at a maximum effort for 10 s followed by a 5-s transition to even pace, which was then held for the remainder of the first minute. In the final minute the athlete was encouraged to complete as much work as possible. Even pace was calculated from the subject's first 500-m race simulation. Recent research by Bishop et al.<sup>21</sup> supports the validity of this procedure. Simulated speed and power output were recorded via a computer interfaced with the ergometer. Twenty minutes after the conclusion of the 500-m race simulation, each athlete completed 5× 100-m maximal efforts followed by a 15 s of passive recovery. Interval time, average and peak power were recorded.

## Whole Blood Measurements

Subjects visited a medical center on 4 occasions per intervention. During each visit, blood from a venepuncture in a forearm vein was collected into tubes and analyzed by a commercial laboratory (Southern Cross Community Laboratories, Auckland, NZ) for the following variables: hemoglobin concentration, hematocrit, ferritin, erythrocyte sedimentation rate and white cell count. The blood tests were conducted at the same time, 1 day pre-treatment, 5 and 10 days mid-treatment, and 3 days post-treatment.

## Statistics

For the measures of performance, errors of measurement and individual responses were estimated using the appropriate mixed model (Proc Mixed) in the Statistical Analysis System (Version 8.2, SAS Institute, Cary NC). The fixed effects (and their levels) were the interaction of the testing session (*8 levels: pre 1, pre 2, post1 and post2* for each of the two arms of the crossover) with the crossover group (*hypoxia first, control first*). The random effects were subject variance, residual variance (representing error of measurement between both the two pre- and two post-tests), and additional within-subject variance for the first testing session (familiarization trial), for both post-hypoxia testing sessions (individual response to hypoxia), and for sessions separated by the treatment and the washout (errors for sessions 4 and 7 weeks apart).

Simple group statistics are shown as means  $\pm$  between-subject standard deviations. To make inferences about true (population) values of the effect hypoxia on performance, the uncertainty in the effect was expressed as 90% confidence limits and as likelihoods that the true value of the effect represents substantial change (harm or benefit)<sup>23</sup>. An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative, or beneficial and harmful. An estimate of the smallest substantial change in power output is required to make these inferences. The estimate is based on variability in performance of top athletes between competitions<sup>24</sup>. As yet there has been no published research on the variability of competitive kayaking performance, but in other sprint and endurance sports the smallest change is in the range of 0.5-1.5%<sup>25</sup>. For the present study we therefore assumed a smallest worthwhile effect of 1.0%.

## Results

### Subject characteristics

The characteristics and baseline exercise performance (mean of the two pre-tests) of the 10 sub-elite kayak athletes are shown in Table 3.1. In the second arm of the crossover 3 subjects pulled out of the study (2 hypoxia and 1 control).

Table 3.1. Characteristics and baseline measures of the subjects

	Mean $\pm$ SD
<b>Characteristics</b>	
Age (Yrs)	23.2 $\pm$ 8.3
Height (cm)	180.1 $\pm$ 4.0
Body mass (kg)	81.2 $\pm$ 7.2
Best on-water 500-m time in previous year (s)	113.1 $\pm$ 5.3
Hemoglobin (g.L <sup>-1</sup> )	146.4 $\pm$ 5.7
Hematocrit (%)	43.8 $\pm$ 1.3
Ferritin ( $\mu$ g.L <sup>-1</sup> )	64.2 $\pm$ 42.4
<b>Incremental step test</b>	
Peak power (W)	179 $\pm$ 26
Lactate mid point (W)	137 $\pm$ 23
Power @ 90% max HR (W)	139 $\pm$ 20
Economy @ 70% VO <sub>2</sub> max(W)	109 $\pm$ 20
Peak VO <sub>2</sub> (l /min)	4.0 $\pm$ 0.5
Peak lactate (mM)	10.8 $\pm$ 2.4
<b>500-m simulation</b>	
Mean Power (W)	239 $\pm$ 43
Mean power (% Peak aerobic power)	134.5 $\pm$ 8.0
Time (s)	124.2 $\pm$ 6.8
Mean power 0 - 10 s (W)	359 $\pm$ 76
Peak lactate (mM)	12.0 $\pm$ 2.1
<b>Repeat sprint test</b>	
1 <sup>st</sup> sprint power (W)	349 $\pm$ 81
1 <sup>st</sup> sprint time (s)	22.1 $\pm$ 1.5
Final sprint power (W)	239 $\pm$ 32
Final sprint time (s)	25.4 $\pm$ 1.0
Mean sprint power (W)	265 $\pm$ 42
Mean sprint time (s)	24.6 $\pm$ 1.1
Peak Lactate (mM)	13.2 $\pm$ 2.5

## Effects on performance

Table 3.2 shows the mean changes in the performance tests for the hypoxic relative to the control condition and statistics for the difference in the changes. At 3 days post-treatment there were substantial improvements in peak power and mean repeat sprint power in the hypoxic condition. The effect on individual sprints is outlined in Figure 3.1. The most substantial change between conditions in sprint power occurred at Sprints 2, 3 and 5. Lactate-threshold and 500-m power demonstrated improvements, but these were unclear. At 10 days post-treatment, the effects on all performance measures were unclear. However, there was still a strong trend towards improvement in measures of peak power, mean sprint power and 500-m power. Not shown in the Table 3.2 are the percent effects for performance time in the 500-m simulation and repeat sprint test; these were all  $\sim 0.38$  of the percent effects for power.

The standard errors of measurement for measures of performance were: peak aerobic power, 3.0%; lactate threshold, 4.6%; heart rate profile, 3.5%; exercise economy, 5.0%; mean repeat sprint power, 4.3%; 500-m mean power 2.3%; and mean power, 1st 10 s 2.8%.

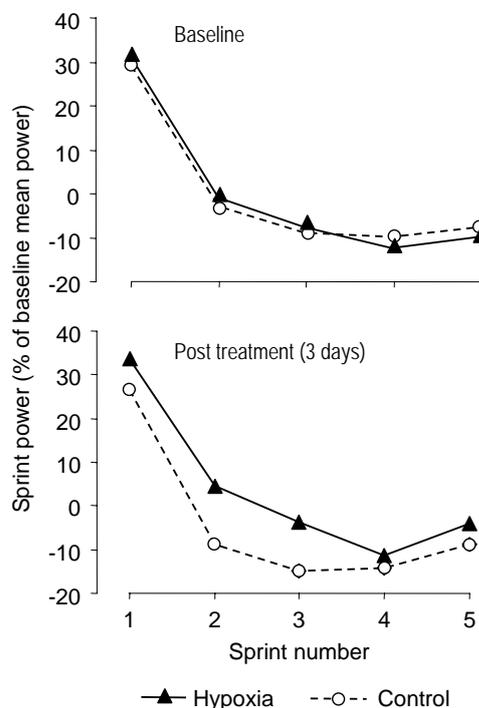


FIGURE 3.1. Effect of intermittent hypoxic and control treatments on repeated 100-m sprint power at baseline and 3 days post treatment. Power in each sprint is expressed as percent of the mean of all five baseline sprints.

TABLE 3.2. Mean changes in performance at 3 d and 10 d post hypoxia and control, and chances that the true difference in the changes is substantial.

	Post-test day	Change in measure <sup>a</sup>			Practical inference <sup>b</sup>
		Intermittent hypoxia mean $\pm$ SD	Control mean $\pm$ SD	Difference; $\pm$ 90%CL	
<b>Incremental step test</b>					
Peak power	3	8.2 $\pm$ 5.7	1.3 $\pm$ 5.7	6.8; $\pm$ 5.2	Benefit very likely
	10	5.8 $\pm$ 5.7	3.2 $\pm$ 5.7	3.5; $\pm$ 5.6	Unclear
Lactate threshold	3	6.7 $\pm$ 8.1	3.1 $\pm$ 8.2	3.5; $\pm$ 7.0	Unclear
	10	5.3 $\pm$ 8.1	6.0 $\pm$ 8.2	-0.7; $\pm$ 7.5	Unclear
Heart rate profile	3	5.9 $\pm$ 6.4	5.0 $\pm$ 6.3	0.9; $\pm$ 5.6	Unclear
	10	5.2 $\pm$ 6.4	5.1 $\pm$ 6.3	0.1; $\pm$ 6.1	Unclear
<b>Repeat sprint test</b>					
Mean repeat power	3	3.9 $\pm$ 6.4	-4.1 $\pm$ 7.6	8.3; $\pm$ 6.7	Benefit very likely
	10	3.1 $\pm$ 6.4	0.1 $\pm$ 7.6	3.0; $\pm$ 7.2	Unclear
<b>500-m simulation</b>					
500-m power	3	4.6 $\pm$ 5.5	2.2 $\pm$ 2.8	2.4; $\pm$ 4.1	Unclear
	10	6.0 $\pm$ 5.5	3.9 $\pm$ 2.8	2.2; $\pm$ 4.3	Unclear
Mean power 0-10 s	3	6.1 $\pm$ 8.5	2.9 $\pm$ 6.2	3.1; $\pm$ 8.2	Unclear
	10	0.8 $\pm$ 8.5	9.3 $\pm$ 6.2	-7.8; $\pm$ 8.5	Unclear

<sup>a</sup> Units of change are % for all measures.

<sup>b</sup> Based on a smallest beneficial or harmful change in performance of 1%

$\pm$ 90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

## Effects on Physiological Measures

Table 3.3 shows the mean changes in physiological measures for the hypoxic relative to the control condition and statistics for the difference in the changes. Intermittent hypoxia produced substantial effects on some measures of hematology. Hemoglobin concentration and hematocrit were both substantially elevated in the hypoxic condition 10 days during the intervention and 3 days post-treatment. Although ferritin showed a trend of decrement, it was not substantially different between the control and hypoxic condition until 3 days post-treatment. Effects on other blood parameters assayed but not shown in Table 3.3 (erythrocyte sedimentation rate and white blood cells count) were unclear. The effect on the other physiological measures was less pronounced, with only one measure (peak aerobic power obtained during the 500-m time trial) showing a substantial reduction in the hypoxic condition at 3 days post-treatment.

The standard errors of measurement for physiological measures were:  $\text{VO}_2\text{max}$ , 4.7%; exercise economy, 5.0%; peak lactate (step test), 11%; peak lactate (repeat sprints),

6.4%; 500-m power (% peak aerobic power), 2.5%; peak lactate (500-m), 14%; hemoglobin, 1.6%; hematocrit, 1.9% and ferritin, 13%.

TABLE 3.3. Mean changes in physiological measures at 3 d and 10 d post hypoxia and control, and chances that the true difference in the changes is substantial.

	Post-test day	Change in measure (%)			Qualitative inference <sup>a</sup>
		Hypoxia mean $\pm$ SD	Control mean $\pm$ SD	Difference; $\pm$ 90%CL	
<b>Incremental step test</b>					
VO <sub>2</sub> max	3	-0.5 $\pm$ 6.5	-0.4 $\pm$ 5.8	-0.1; $\pm$ 5.2	Unclear
	10	2.9 $\pm$ 6.5	2.2 $\pm$ 5.8	0.7; $\pm$ 6.3	Unclear
Exercise economy	3	1.7 $\pm$ 7.6	3.1 $\pm$ 6.2	-1.4; $\pm$ 6.4	Unclear
	10	2.7 $\pm$ 7.6	-5.7 $\pm$ 6.2	9.0; $\pm$ 12.6	Unclear
Peak Lactate	3	5 $\pm$ 15.5	6.1 $\pm$ 22.6	-0.7; $\pm$ 17	Unclear
	10	4.3 $\pm$ 15.5	-0.5 $\pm$ 22.6	3.0; $\pm$ 9.9	Unclear
<b>Repeat sprint test</b>					
Peak lactate	3	1 $\pm$ 17	-5 $\pm$ 11	6; $\pm$ 14	Unclear
	10	-6 $\pm$ 17	-6 $\pm$ 11	-1; $\pm$ 15	Unclear
<b>500-m time trial</b>					
Mean power (% peak aerobic power)	3	-3.7 $\pm$ 3.8	1.7 $\pm$ 7.1	-5.2; $\pm$ 4.5	Likely negative
	10	-0.4 $\pm$ 3.8	2.4 $\pm$ 7.1	-2.6; $\pm$ 5.0	Unclear
Peak Lactate	3	3.1 $\pm$ 18.3	8.3 $\pm$ 17	-4.8; $\pm$ 15	Unclear
	10	-6 $\pm$ 18	1 $\pm$ 17	-7; $\pm$ 16	Unclear
<b>Blood parameters</b>					
Hemoglobin	-16 <sup>b</sup>	0.4 $\pm$ 3.4	1 $\pm$ 2.5	-0.5; $\pm$ 1.9	Unclear
	-11 <sup>b</sup>	1.9 $\pm$ 3.4	-2.0 $\pm$ 3.4	4.0; $\pm$ 2.1	Almost certainly positive
	3	1.8 $\pm$ 3.4	-1.7 $\pm$ 3.4	3.6; $\pm$ 3.2	Likely positive
Hematocrit	-16 <sup>b</sup>	-0.8 $\pm$ 3.1	0.8 $\pm$ 3.9	-1.5; $\pm$ 2.3	Unclear
	-11 <sup>b</sup>	1.4 $\pm$ 3.1	-2.5 $\pm$ 3.1	4.1; $\pm$ 2.5	Almost certainly positive
	3	0.3 $\pm$ 3.1	-2.3 $\pm$ 3.1	2.7; $\pm$ 3.4	Unclear
Ferritin	-16 <sup>b</sup>	-5.1 $\pm$ 19	-0.4 $\pm$ 21	-4.7; $\pm$ 15	Unclear
	-11 <sup>b</sup>	-6.8 $\pm$ 19	-8.9 $\pm$ 21	2.3; $\pm$ 15	Unclear
	3	-18.5 $\pm$ 19	0.3 $\pm$ 21	-19; $\pm$ 15	Likely negative

<sup>a</sup>Based on a smallest substantial change of 1% for VO<sub>2</sub>max, economy and mean power (% peak aerobic power), and 0.2 of the baseline between-subject standard deviation for all other measures.

<sup>b</sup>Negative test days represent 5 & 10 days mid-intervention.

$\pm$ 90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

## Discussion

The major finding of this study is that intermittent hypoxic exposure for 15 days over a three-week period substantially enhanced peak power and repeat sprint performance on a kayak ergometer in sub-elite kayak paddlers three days after the treatment period. In addition, there was a substantial increase in hemoglobin concentration, hematocrit and a substantial reduction in ferritin following the treatment. Effects on all other measures, including those representing anaerobic power and all measures at 10 days post-treatment, were unclear.

The lack of clarity for most measures was due in part to a larger-than-expected error of measurement. For example, the errors of measurement for the performance measures in the comparable study of Wood et al.<sup>15</sup> were ~1-2%, whereas the errors were 2-5% in the present study. These differences in error could be due to kayak ergometry being less reliable than running, which has been used to assess performance in similar studies<sup>15, 19</sup>. The reduced reliability may result from small differences in technique between kayak ergometry and on-water kayaking and the athletes using the kayak ergometer only during performance assessment. Given these larger errors of measurement, we would need a larger sample size than was available to get clear outcomes when the true effect is a change in performance of a few percent.

Overall, the clear effects of adaptation to hypoxia on performance were somewhat greater than reported in similar studies<sup>15, 16, 19</sup> and may be viewed with skepticism by fellow exercise physiologists. It is possible that brief waves of hypoxia equivalent to moderately high altitude provide a more effective stimulus than longer periods at the equivalent of a lower altitude<sup>15</sup>. Furthermore, the level of intensity was individually monitored and controlled via pulse oximetry, which ensured all our subjects received a similar stimulus. This approach was either not utilized or not possible in most studies. It is also likely that the greater magnitude is partly a result of greater uncertainty in the estimates. If the true effect of the adaptation is a few percent, sampling variation will result in the effect being either unclear or beneficial (or, rarely, harmful). The beneficial effects will therefore be biased higher than the true effect. Similar bias occurs when inferences are based on statistical significance rather than precision of estimation, a well-known phenomenon in meta-analysis<sup>26</sup>. It is only when all measures are taken into account that the mean effect of a treatment is unbiased. As can be seen from Table 3.2, the overall effect is ~3-4% three days post-exposure and somewhat less by 10 days post-exposure, which is similar to those of Wood et al.<sup>15</sup> and to some extent Hamlin and Hellemans<sup>16</sup>.

Our findings are more difficult to reconcile with those of Julian et al.<sup>19</sup>, who found that 4 weeks of intermittent hypoxic exposure using a device and protocol similar to those of the present study failed to improve exercise performance or change hematology. Some important differences between the studies may explain the contrasting findings. Firstly, we monitored and adjusted the level of hypoxia individually via pulse oximeters to reach the target saturation, which progressively decreased every 1-2 days. In contrast, the subjects of Julian et al. maintained a preset level of hypoxia for 5 days, without frequent monitoring of individual oxygen saturations. In addition, the lowest saturation reached in Julian et al.'s study was 82% compared to 76% in the present study. Therefore, their subjects may have received a reduced hypoxic stimulus compared to ours. Furthermore, Julian et al.'s subjects, who were top-level competitors in individual endurance sports, may have been in a more highly trained state and may therefore have had less potential for enhancement of performance than our national-level kayak paddlers.

The observed physiological changes following adaptation to the hypoxic exposure provide several plausible mechanisms for the performance enhancement. The substantial enhancement in both peak power and repeat sprint speed in the present study are likely to have resulted from an improvement in oxygen carrying capacity, mediated by an erythropoietic mechanism. In support of this explanation, at 10 days during and 3 days post-treatment there was a substantial increase in hemoglobin concentration in the hypoxic condition. These changes suggest an enhancement of the oxygen carrying capacity of the blood occurred and are consistent with changes in hematology following adaptation to intermittent hypoxia exposure in other studies<sup>16, 18</sup> and in studies investigating the live-high train-low model<sup>1, 27</sup>. The change in hemoglobin was accompanied by a substantial decrease in ferritin at 3 days post-treatment in the hypoxia condition. Since it is known that ferritin levels substantially decrease when humans move to high altitude<sup>28</sup>, our ferritin results provide further indirect evidence of an erythropoietic stimulus. Direct evidence will require measurement of physiological parameters more closely related to erythropoiesis, such as erythropoietin, reticulocytes and hemoglobin mass.

Enhancements of performance resulting from erythropoiesis would normally be accompanied by a change in  $\text{VO}_2\text{max}$ . Such changes may have occurred in our study, but our uncertainty of change in oxygen uptake measures makes interpretation of these measures difficult. It is also possible that  $\text{VO}_2\text{max}$  was unaffected and that exercise economy improved. Improvements in economy of 3-6% have been observed after

various hypoxic interventions with athletes<sup>9, 10, 29-31</sup>. It has been theorized that this adaptation is a direct response to hypoxia at the tissue level, and that a suitable regulatory system mediated by changes in hypoxia inducible factor exists in most cells<sup>8</sup>. While the effect on economy was unclear in our study, improvements in this variable along with  $\text{VO}_2\text{max}$  cannot be dismissed as potential mechanisms mediating our performance enhancement.

Changes in lactate threshold also provide a potential mechanism for performance enhancement. Although statistically unclear, there was a substantial rightward shift in the lactate profile following the hypoxic intervention, similar to that reported by Wood et al.<sup>15</sup>. It is possible that this shift resulted from a change in substrate utilization or that the adaptation to the hypoxic stimulus simply enabled the athletes to train harder, thereby further enhancing lactate threshold.

A change in anaerobic power has also been suggested as an adaptation to intermittent hypoxia mediated, for example, by an increase in muscle buffering capacity<sup>4, 29</sup> and maximal accumulated oxygen deficit<sup>32</sup>. While we did not measure these variables, our indirect measures of anaerobic power are not consistent with this notion. The percentage of peak aerobic power obtained during the 500-m time trial *reduced*, despite 500-m performance *improving* in the hypoxic condition. This ratio would be expected to increase if anaerobic power improved. Other measure of anaerobic power in the present study that could provide evidence in support of this trend were mean power in the first 10 s of the 500-m time trial and mean power in the first of the 5 repeat sprints. Peak lactates would also provide an indirect measure of muscle buffering following the incremental step test, 500-m time trial and repeat sprint test. Unfortunately the changes in all these measures were unclear following the hypoxia intervention. These data collectively suggest that adaptation to intermittent hypoxic exposure does not enhance anaerobic power.

A potential limitation to the study design is that the subjects were not blind to the treatment they were receiving. However, motivation levels and effort were high, because the testing sessions occurred during the competitive season and replaced normal race-specific training sessions. Furthermore, the observed improvements in lactate threshold, hemoglobin, hematocrit and ferritin, which would all be unaffected by any potential placebo effect, provide evidence that intermittent hypoxia produced some kind of physiological adaptation. Finally it has been proposed that crossovers eliminate or reduce the biases arising from placebo and other patient-preference effects: because all subjects receive all treatments, it is in their interest to comply with and perform well

for all treatments, if they want to know how well the treatments work for them<sup>33</sup>. Therefore, it is unlikely that the non-blinding of our subjects would have influenced their motivation and intent in the testing sessions.

In conclusion, this investigation demonstrated that the use of intermittent hypoxic exposure at rest in 5 minute intervals for 60 minutes per day, 5 times per week for 3 weeks is sufficient to elicit substantial and worthwhile improvements in peak power, repeat sprint speed and performance-related hematology. Further research is required to clarify the mechanisms mediating the performance changes.

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## CHAPTER 4

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### Cycling performance following adaptation to two protocols of acutely intermittent hypoxia

#### Abstract

**Purpose:** Adaptation to acutely intermittent hypoxic exposure appears to produce worthwhile enhancements in endurance performance, but the current 5-min duration of hypoxia and recovery intervals may not be optimal. **Methods:** Eighteen male competitive cyclists and triathletes were randomized to one of two intermittent-hypoxia groups, while nine similar athletes represented a control group. Athletes in the hypoxia groups were exposed to 60 min per day of intermittent hypoxia consisting of alternating intervals of hypoxia and normoxia lasting either 3 or 5 min. Exposures were performed at rest for 5 consecutive days per week for 3 wk. Oxygen saturation, monitored with pulse oximetry, was reduced progressively from 90% (Day 1) to 76% (Day 15). All athletes maintained their usual competitive-season training throughout the study. Incremental and repeated-sprint tests were performed pre-, 3 d post-, and 14 d post-intervention. Venous blood at rest was sampled pre-, mid- and post-intervention. **Results:** There were no clear differences between effects of the two hypoxic treatments on performance or various measures of oxygen transport, haematopoiesis and inflammation. Compared with control, the combined hypoxic groups showed clear enhancements in peak power (4.7%; 90% confidence limits,  $\pm 3.1\%$ ), lactate-profile power (4.4%;  $\pm 3.0\%$ ) and heart-rate profile power (6.5%;  $\pm 5.3\%$ ) at 3 d post-intervention, but at 14 d the effects were unclear. Changes in other measures at 3 and 14 d post-intervention were either unclear or unremarkable. **Conclusion:** Acutely intermittent hypoxia produced substantial enhancement in endurance performance, but the relative benefit of 3- vs 5-min exposure intervals remains unclear.

## Introduction

Exposure to hypoxia in both natural and simulated environments is commonly used by athletes in an attempt to improve high-intensity endurance performance. Traditionally, an increase in red-cell mass, with its associated improvement in  $\text{VO}_2\text{max}$ , has been proposed as the primary mediator of performance enhancement.<sup>1</sup> More recently several investigators have found enhancement of performance after hypoxia without substantial changes in oxygen transport-related measures such as hemoglobin mass, haematocrit and serum soluble transferrin receptor.<sup>2-4</sup> Therefore it seems likely that alternative mechanisms such as enhanced exercise economy,<sup>2,4</sup> elevated muscle buffer capacity<sup>2</sup> and enhanced mitochondrial function<sup>5</sup> play a substantial role in the adaptation process following hypoxia.

While the mechanisms mediating performance enhancement following adaptation to hypoxia continue to be debated,<sup>1,6</sup> researchers have consistently found an enhancement in endurance power of 1-2% with the live-high train-low model of adaptation to hypoxia in a natural environment. Furthermore, this model of exposure has been simulated using hypobaric chambers<sup>3</sup> and nitrogen houses<sup>2,4</sup> with comparable success. Acutely intermittent hypoxia offers a more convenient method of simulating the live-high train-low model. With this method, athletes breathe hypoxic gas and normal air for alternating periods of a few minutes for 60-90 min daily over several weeks. Some researchers investigating acutely intermittent hypoxia have reported considerable enhancements in endurance performance<sup>7-10</sup> and repeated sprint performance,<sup>7,10</sup> but others have found little or no change in measures of endurance performance.<sup>11,12</sup>

To date, all acutely intermittent hypoxia research has utilised a protocol of repeated 5- or 6-min hypoxic intervals followed by a similar period of normoxia. Manipulation of the duration of the hypoxic and normoxic intervals could result in greater performance gains, if the number of on-off hypoxia transitions in a given time period regulates the physiological responses to hypoxia. A response that warrants further investigation is inflammation, which is associated with hypoxia.<sup>13</sup> Furthermore, inflammation is also present following strenuous exercise<sup>14</sup> and may contribute to the adaptation process with physical training. Whether an inflammatory response is linked to physiological or performance adaptations following intermittent hypoxia is unknown, but investigating cytokine and general inflammatory responses to hypoxia would help to address this question. Thus, the aim of the present study was to determine the effects of altering the hypoxic and normoxic interval duration and to

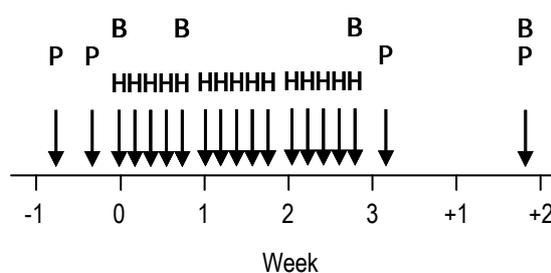
determine whether any change in performance was associated with changes in inflammatory markers.

## Methods

### Study Design

The time line of the study is shown in Figure 4.1. This design was a parallel-groups controlled trial. Male subjects ( $n=27$ ) were randomly assigned to three groups (3-min, 5-min or control), balanced for their incremental peak power achieved in two pre-intervention testing sessions separated by 3-5 d in the week before the start of the intervention. Subjects then performed a three week intervention of intermittent hypoxic exposure 5 days per week plus their normal cycle training (3-min and 5-min groups), or their normal cycle training alone (control). The same testing procedures were repeated at 3 and 14 d post-intervention. Blood samples were taken at rest, 1 d pre-treatment, after 5 d of treatment, on the last day of treatment, and 14 d post-treatment, for evidence of mechanisms of any performance effects. The subjects were studied in two cohorts of 11 and 16, staggered by one week, to accommodate constraints on availability of resources for testing.

FIGURE 4.1. Time line of performance tests (P), blood tests (B) and hypoxic-exposure sessions (H).



### Subjects

The subjects were well-trained, competitive endurance cyclists and triathletes who had been competing in road cycle racing for at least two years and training consistently for at least three months prior to the start of the study. Subject characteristics, including training volume immediately prior to the intervention, are shown in Table 4.1. The study took place during the competitive phase of each athlete's periodised season. All gave voluntary informed consent as required by the institutional ethics committee.

Table 4.1. Baseline subject characteristics of the 3-min and 5-min hypoxic groups and of the control group.

	3-min (n=9)	5-min (n=9)	Control (n=9)
Age (y)	34 ± 9	37 ± 7	36 ± 6
Height (cm)	177 ± 6	178 ± 7	182 ± 7
Body mass (kg)	83 ± 8	81 ± 6	85 ± 10
Training (h.wk <sup>-1</sup> )	12 ± 4	12 ± 6	10 ± 2

Data are mean ± standard deviation.

## Training and Diet

During the study all athletes were in a period of medium volume training with the majority competing in endurance races of 2-3 h duration each weekend. None of the athletes was tapering for any major competitions during the intervention or in the performance test post intervention. The athletes were instructed to maintain their normal diet during the course of the intervention and to have an easy day of training prior to each testing day. Training diaries were distributed to the athletes for the intervention period, but compliance (~50%) was not adequate in any groups to justify their analysis. To ensure adequate iron storage was present, all athletes underwent an assay for ferritin status before the start of the study. All athletes were found to be in the normal range and therefore no iron supplementation was administered.

## Hypoxic Treatment

Athletes underwent 15 d of intermittent hypoxic exposure created by an io™ altitude simulator (io, Queenstown, New Zealand). This device uses oxygen filtration to reduce the oxygen content of the air that the subject breathes when connected to the system via a face mask. The degree of hypoxia was gradually increased throughout the duration of the experimental period by reducing the fraction of inspired oxygen ( $F_{I}O_2$ ) and saturation of haemoglobin with oxygen as follows:

- Days 1-5: saturation = 90,88,86,86, 84%;  $F_{I}O_2$  = 12%
- Days 6-10: saturation = 82, 82, 80, 80, 78%;  $F_{I}O_2$  = 11%
- Days 11-15: saturation = 78, 78, 76,76,76%;  $F_{I}O_2$  = 10, 9%

This incremental hypoxic exposure protocol was based on previous research<sup>7,10</sup>. Athletes breathed hypoxic air intermittently for a period of 60 min five days per week using either 3-min or 5-min periods of alternating hypoxia and normoxia. Haemoglobin saturation was monitored individually on a forefinger with pulse oximeters (Sport-Stat,

Nonin Medical, Minneapolis, MN; accuracy claimed to be a standard deviation of  $\pm 2$  units of percent saturation in the range 70-100%) throughout each interval of exposure. Athletes were advised to remove the mask if saturation dropped below the target level and then immediately reposition the mask when the saturation had returned to the target level. If the athlete could not reach the desired level of saturation, the  $F_{I}O_2$  was reduced at the altitude simulator. This individual monitoring ensured that all athletes remained at the same saturation and had similar exposure.

### **Exercise Performance Tests**

All physiological and performance tests were conducted in a temperature controlled laboratory (19-21°C) using a electro-magnetically braked cycle ergometer (Velotron, Racermate, Seattle, USA). Prior to the first test, the ergometer was set to replicate the athlete's road bike set-up as closely as possible. The settings were recorded and used for each subsequent testing session. The ergometer was interfaced with a computer that continuously measured, calculated and stored accumulated work and other associated work indices. In each testing session the ergometer was calibrated in accordance with the manufacturers instructions prior to the athlete performing an incremental step test to exhaustion and a repeated 30-s sprint test. These tests were separated by a 20-min recovery period consisting of 5 min of rest, 10 min cycling at 50% of peak power, and 5 min of rest. Each athlete completed four test sessions (two pre-treatment and two post-treatment). The first pre-test was intended as a familiarization, but consistent with the approach of Bonetti et al.<sup>7</sup>, performance in the two pre-tests was averaged and used as baseline. This approach resulted overall in a marginal improvement in precision of estimation of treatment effects compared with use of only the second pre-test.

The continuous, incremental step test commenced at a workload of 150-180 W, depending on fitness, and increased by 30 W every 3 min until volitional exhaustion. At the end of each 3-min stage, capillary blood was sampled from an earlobe for measurement of blood lactate using a hand-held analyser (Lactate Pro, Arkray, Japan). In addition, pulmonary oxygen uptake ( $VO_2$ ) was measured continuously using a breath-by-breath metabolic system (Metamax 3b, Cortex, Leipzig, Germany), which was calibrated using a standard two-point gas check immediately before and after each incremental test. Heart rate was continuously measured using a short-range telemetry device (Polar A1, Polar Electro, Kempele, Finland). The average heart rate obtained in

the final 10 s of each stage was recorded. Maximum oxygen uptake ( $\text{VO}_2\text{max}$ ) was determined as the highest 30-s value obtained during the test. Due to a technical fault encountered with our metabolic cart on the final series of performance tests (14 d post-intervention, Cohort 2) we were unable to measure  $\text{VO}_2$ . We therefore had sufficient data to analyse  $\text{VO}_2$  only at 3 d post intervention. A measure of lactate-profile power (analogous to the individual lactate-threshold power) was derived from the step tests as follows. We assumed a log-log relationship between lactate concentration and power output.<sup>15</sup> We used the Trend function in Microsoft Excel to fit straight lines to the pre- and post-treatment lactate plots, then predicted power output corresponding to a "midpoint" of lactate concentration in the step tests. The midpoint was found by averaging the minimum and maximum values of the log-transformed lactate concentrations from all four tests. A similar procedure was used to create individual power profiles of heart rate and exercise economy; these variables did not require log transformation and power was calculated at fixed percentages of the individual's maximum value (heart rate 90%, exercise economy 70%).

The repeated sprint test consisted of four 30-s all-out sprints against a load of 0.09 kg per kg of body mass.<sup>16</sup> Athletes pedalled at a cadence of  $100 \text{ min}^{-1}$  for 10 s prior to the start of each repetition and were then instructed to exert maximal pedal cadence throughout the 30-s period once the resistance was applied. Immediately following each repetition athletes were instructed to pedal unloaded at a self-selected cadence for a period of 50 s, after which they repeated the procedure. Athletes were verbally encouraged, given time updates, and instructed to remain seated throughout each 30-s repetition. Measures of peak and mean power were obtained using specifically designed software (Velotron Wingate, Seattle, USA). Baseline performance and physiological variables are shown in Table 4.2.

### **Whole Blood Measurements**

All athletes were required to fast overnight prior to the blood test the preceding morning, which was conducted at the same time for each athlete (between 06:00 – 09:00) and before any hypoxic exposure session. On all occasions, 7.5 ml of whole blood was drawn from the median cubital vein and collected into tubes (Vacutainer SST 2, 3.5 ml and Vacutainer K2E, 4 ml; BD, Franklin Lakes, NJ) by an experienced phlebotomist. Standard haematological parameters (hemoglobin concentration, haematocrit, ferritin, reticulocytes and white cell count) were determined from these

TABLE 4.2 Baseline values of performance and physiological variables in the 3-min and 5-min hypoxic groups and in the control group.

	3-min	5-min	Control	Measurement error <sup>a</sup>
<b>Incremental step test (mean ± coefficient of variation)</b>				
Peak power (W)	347 ± 9%	349 ± 12%	349 ± 12%	±1.8%
Lactate-profile power (W)	270 ± 13%	266 ± 21%	277 ± 10%	±5.2%
Heart-rate profile power (W)	294 ± 13%	281 ± 16%	286 ± 14%	±4.2%
VO <sub>2</sub> max (L.min <sup>-1</sup> )	4.32 ± 10%	4.10 ± 12%	4.13 ± 13%	±6.8%
Economy (W)	223 ± 14%	226 ± 10%	223 ± 16%	±5.0%
Peak lactate (mmol.L <sup>-1</sup> )	11.3 ± 22%	11.5 ± 27%	10.4 ± 24%	±15%
Peak heart rate (beats.min <sup>-1</sup> )	182 ± 7.2%	181 ± 5.6%	189 ± 4.4%	±1.6%
<b>Repeated sprint test (mean ± coefficient of variation)</b>				
Mean sprint power (W)	610 ± 9.5%	608 ± 6.0%	640 ± 8.8%	±1.9%
First sprint power (W)	705 ± 10%	675 ± 9%	763 ± 12%	±4.1%
Final sprint power (W)	553 ± 9.1%	553 ± 13%	564 ± 8.0%	±3.4%
First sprint (% of peak power)	203 ± 9.6%	193 ± 18%	219 ± 14%	±5.1%
Peak lactate (mmol.L <sup>-1</sup> )	13.9 ± 12%	12.9 ± 22%	13.7 ± 13%	±9.8%
<b>Hematology (mean ± coefficient of variation)</b>				
Hemoglobin (g.L <sup>-1</sup> )	153 ± 4.7%	148 ± 3.8%	156 ± 5.4%	±2.9%
Hematocrit (%)	0.47 ± 3.3%	0.47 ± 2.9%	0.47 ± 4.9%	±4.1%
Ferritin (µg.L <sup>-1</sup> )	164 ± 84%	211 ± 61%	197 ± 85%	±9.7%
2,3-DPG (mmol.L <sup>-1</sup> )	2.4 ± 13%	2.2 ± 6.6%	2.4 ± 11%	±4.8%
Reticulocyte count (10 <sup>9</sup> . L <sup>-1</sup> )	40 ± 26%	45 ± 21%	40 ± 40%	±12%
White blood-cell count (10 <sup>9</sup> . L <sup>-1</sup> )	5.1 ± 16%	5.3 ± 22%	6.3 ± 43%	±16%
<b>Inflammatory markers (mean ×/÷ factor variation)</b>				
C-reactive protein (µg.ml <sup>-1</sup> )	3.5 ×/÷ 3.1	1.8 ×/÷ 6.8	2.5 ×/÷ 8.0	×/÷2.4
IL-1β (pg.ml <sup>-1</sup> )	4.1 ×/÷ 10	6.4 ×/÷ 12	3.9 ×/÷ 13	×/÷1.7
SOD (U.mg <sup>-1</sup> Hb)	1.69 ×/÷ 1.22	1.56 ×/÷ 1.11	1.61 ×/÷ 1.19	×/÷1.08

Data are back-transformed means ± coefficients of variation or ×/÷ factor variations.

<sup>a</sup>Uncertainties in these errors: ×/÷1.26 (factor 90% confidence limits).

samples by an independent, commercial laboratory (Southern Cross Community Laboratories, Auckland, NZ). In addition, a further 12 ml of whole blood was drawn from the same puncture site and collected into tubes (Vacutainer K2E, 6 ml; BD, Franklin Lakes, NJ) and spun at 2000 g for 10 min. The separated plasma and packed red blood cells were aliquoted in volumes of 1.5 ml into Eppendorf tubes and immediately frozen using dry ice. These samples were subsequently stored at -80°C and used for determination of 2,3-diphosphoglycerate (2,3-DPG), C-reactive protein (CRP), erythrocyte superoxide dismutase (SOD) and interleukin-1 beta (IL-1β). 2,3-DPG was measured using an enzymatic UV spectrophotometer kit (Cat. No. 10 148 334 001, Roche, Sydney, Australia). Hemoglobin concentration was measured in these same samples using Drabkins solution as described by Dacie.<sup>17</sup> CRP was measured

using an ELISA kit (DSL-10-42100, Diagnostic Systems Laboratories, Webster, TX). Plasma IL-1 $\beta$ , was measured using a DuoSet ELISA development kit (R&D Systems, Minneapolis, MN). Red blood cell superoxide dismutase was measured using a modification of the method described by Kakkar.<sup>18</sup> The sodium pyrophosphate buffer had 5% Tween-20 added to increase the solubility of the nitroblue tetrazolium. The method was adapted for use in a Fluostar Optima microtitre plate reader equipped with two reagent injectors. The reaction rate was measured for 10 min. Red blood cell extracts were prepared as described by Peskin<sup>19</sup> and diluted 4-fold for SOD measurement.

### **Statistical Analysis**

Analyses were performed with the mixed model (Proc Mixed) in the Statistical Analysis System (Version 8.2, SAS Institute, Cary NC). The programme used the methodology of Hopkins<sup>20</sup> for analysis of controlled trials, including: adjustment for any differences in pre-test means between groups and estimation of effect of pre-test score, by including the pre-test score as a covariate; estimation of individual responses from the difference in the variances of change scores; and estimation of standard errors of measurement. Effects were calculated for the difference between the 3-min and 5-min groups and for the difference between the mean effect of the hypoxic groups and control. Errors of measurement were calculated from the change scores between the first two trials for all subjects (Table 4.2).

Means and between-subject standard deviations for subject characteristics were derived from the raw values of the measures; for all other measures they were derived by back transformation of the log-transformed values. Standard deviations and effects for measures of performance and most other measures are shown as percentages. Inflammatory markers had large between-subject variations (some >100%), so their standard deviations and effects are shown as factors. To make inferences about true (population) values of the effect of hypoxia on performance, the uncertainty in the effect was expressed as 90% confidence limits and as likelihoods that the true value of the effect represents substantial change<sup>21</sup>. An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative. An estimate of the smallest substantial change in power output was required to make these inferences. Paton and Hopkins<sup>22</sup> estimated smallest effects of 0.5-1.5% in mean power, based on variability in

competitive performance of elite cyclists in various time trials where drafting and group tactics did not contribute. We therefore assumed a smallest worthwhile effect of 1.0%, which also applied to physiological measures directly related to performance. For all other measures we used 0.2 of the baseline between-subject standard deviation<sup>20</sup>.

## Results

Table 4.3 shows the effects on mean performance in the three groups. Differences between the effects in the 3-min and 5-min hypoxic groups for all performance measures obtained during the incremental step test and repeated sprint test at 3 and 14 d post-treatment were unclear, with no consistent trend in the observed differences for the various measures. When the hypoxic groups were combined, there were substantial improvements in peak power, lactate-profile power and heart-rate profile power in the combined hypoxic groups relative to the control at 3 d post-treatment. These improvements tended to be moderated by the pre-test score, two standard deviations of which accounted for reductions in peak power, lactate-profile power and heart-rate profile power of -1.9%, -5.4%, and -2.5% respectively; however, none of these effects was clear (90% confidence limits  $\pm 5.7\%$ ,  $\pm 7.2\%$  and  $\pm 10\%$ ). After adjustment for these effects of pre-test score, remaining individual responses were not consistent across the three measures of endurance performance (standard deviations of 1.7%, -1.9% and -5.7%; 90% confidence limits  $\sim \pm 6\%$ ).

At 14 d post-treatment, the effects in the combined hypoxic groups relative to the control for all performance measures were unclear, although the observed effects represented substantial improvements (on average about half of the 3-d effects). There was a substantial impairment in mean repeated sprint power and in the final sprint in the combined hypoxic groups relative to the control at 3 d post-treatment. The other effects on sprint power for the combined hypoxic groups relative to control were unclear, but with one exception the observed effects represented substantial impairments. Power in the first sprint expressed as a percent of peak power in the incremental test showed a clear impairment at 3 d post-treatment.

A comparison of the physiological measures from the performance tests in the two hypoxic groups also revealed no clear differences (Table 4.4). The only clear effects in the comparison of the hypoxic groups with control were possible trivial differences in peak heart rate in both post-treatment tests and probable reduction in peak lactate for the repeat sprint test at 3 d post-treatment. For measures of hematology (Table 4.4)

there was a possibly lower reticulocyte count and higher white blood-cell count in the 3-min group relative to the 5-min group at the end of the exposure period, and a probably lower 2,3-DPG concentration and higher reticulocyte count 14 d later. Differences between the hypoxic groups were otherwise unclear. The only clear differences in the combined hypoxic groups relative to control were a possibly lower ferritin concentration and higher reticulocyte count at the end of the exposure period, and a probably higher concentration of hemoglobin and very likely higher reticulocyte count 14 d after exposure.

There was a substantial reduction in C-reactive protein (by a factor of 0.5; 90% confidence limits  $\times/\div 2.3$ ) and a substantial increase in IL-1 $\beta$  (1.9;  $\times/\div 2.0$ ) in the 3-min group relative to the 5-min group on Day 5 of the intervention. These differences were more-or-less maintained through to the last blood test. Differences for SOD were unclear. The only clear effect for the comparison of the hypoxia groups with the control group was a consistently trivial difference in IL-1 $\beta$ . A table of changes and comparisons for the inflammatory markers can be obtained from the authors.

TABLE 4.3. Percent changes in performance in the 3-min and 5-min hypoxic groups and in the control group between baseline and post-treatment tests.

	Post-treatment day	Changes in each group			Comparisons of changes in groups			
		3-min mean $\pm$ SD	5-min mean $\pm$ SD	Control mean $\pm$ SD	3-min – 5-min; $\pm$ 90%CL	Practical inference <sup>a</sup>	Hypoxia –control; $\pm$ 90%CL	Practical inference <sup>a</sup>
<b>Incremental step test</b>								
Peak power	3	3.5 $\pm$ 5.9	1.3 $\pm$ 2.3	-2.2 $\pm$ 4.0	2.2; $\pm$ 3.9	Unclear	4.7; $\pm$ 3.1	Very likely +ive
	14	2.0 $\pm$ 3.5	0.6 $\pm$ 2.5	-0.3; $\pm$ 6.0	1.3; $\pm$ 2.6	Unclear	1.6; $\pm$ 4.6	Unclear
Lactate-profile power	3	2.4 $\pm$ 3.1	3.7 $\pm$ 4.3	-1.3 $\pm$ 4.0	-1.3; $\pm$ 3.0	Unclear	4.4; $\pm$ 3.0	Very likely +ive
	14	1.3 $\pm$ 4.5	1.1 $\pm$ 5.5	-2.0 $\pm$ 5.8	0.2; $\pm$ 4.2	Unclear	3.3; $\pm$ 4.8	Unclear
Heart-rate profile power	3	2.4 $\pm$ 4.3	3.9 $\pm$ 5.2	-3.2; $\pm$ 7.7	-1.4; $\pm$ 3.9	Unclear	6.5; $\pm$ 5.3	Very likely +ive
	14	0.9 $\pm$ 7.4	3.2 $\pm$ 6.4	0.6; $\pm$ 3.7	-2.1; $\pm$ 5.7	Unclear	1.4; $\pm$ 3.8	Unclear
<b>Repeated sprint test</b>								
Mean sprint power	3	1.0 $\pm$ 2.8	-0.4 $\pm$ 3.2	2.4 $\pm$ 3.6	1.4; $\pm$ 2.9	Unclear	-2.0; $\pm$ 2.6	Possibly -ive
	14	1.2 $\pm$ 2.0	0.3 $\pm$ 4.6	2.7 $\pm$ 4.2	0.9; $\pm$ 3.7	Unclear	-1.9; $\pm$ 4.8	Unclear
First sprint power	3	-0.7 $\pm$ 4.4	-3.9 $\pm$ 5.1	-0.2 $\pm$ 4.9	3.3; $\pm$ 5.5	Unclear	-2.1; $\pm$ 4.1	Unclear
	14	0.0 $\pm$ 3.0	-2.1 $\pm$ 5.1	-1.8 $\pm$ 3.3	2.2; $\pm$ 5.3	Unclear	0.8; $\pm$ 4.7	Unclear
Final sprint power	3	1.8 $\pm$ 4.5	0.7 $\pm$ 5.7	4.6 $\pm$ 7.3	1.1; $\pm$ 5.1	Unclear	-3.2; $\pm$ 4.8	Unclear
	14	1.1 $\pm$ 6.3	1.3 $\pm$ 5.3	7.1 $\pm$ 5.0	-0.2; $\pm$ 5.4	Unclear	-5.5 $\pm$ 4.7	Probably -ive
First sprint (% peak power)	3	-4.2 $\pm$ 8.2	3.3 $\pm$ 6.2	1.1 $\pm$ 5.5	-1.0; $\pm$ 7.1	Unclear	-4.8; $\pm$ 4.7	Probably -ive
	14	-1.7 $\pm$ 5.9	-2.2 $\pm$ 5.4	-0.9 $\pm$ 8.0	0.6; $\pm$ 5.8	Unclear	-1.1; $\pm$ 7.9	Unclear

Data are changes in each group (mean  $\pm$  SD) and comparison of the changes (difference in the means;  $\pm$ 90% confidence limits) between the hypoxic groups and between the mean of the hypoxic groups and control. The practical inference is the qualitative assessment of the chances that the true effect is substantially positive (+ive) or negative (-ive).

<sup>a</sup> Based on a smallest beneficial or harmful change in performance of 1%.

$\pm$ 90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

TABLE 4.4. Percent changes in physiological measures from the performance tests and hematology in the 3-min and 5-min hypoxic groups and in the control group between baseline and post-treatment tests.

	Post-treatment day <sup>a</sup>	Changes in each group			Comparisons of changes in groups			
		3-min mean $\pm$ SD	5-min mean $\pm$ SD	Control mean $\pm$ SD	3-min – 5-min; $\pm$ 90%CL	Practical inference <sup>b</sup>	Hypoxia – control; $\pm$ 90%CL	Practical inference <sup>b</sup>
<b>Incremental step test</b>								
VO <sub>2</sub> max	3	-3.1 $\pm$ 5.4	-3.3 $\pm$ 4.2	-5.0 $\pm$ 9.1	0.2; $\pm$ 4.5	Unclear	1.9; $\pm$ 6.9	Unclear
Economy	3	8.8 $\pm$ 9.6	6.2 $\pm$ 9.0	5.1 $\pm$ 8.0	2.5; $\pm$ 7.9	Unclear	2.2; $\pm$ 6.8	Unclear
Peak lactate	3	0 $\pm$ 24	-2 $\pm$ 21	-2 $\pm$ 28	2; $\pm$ 17	Unclear	1; $\pm$ 18	Unclear
	14	-1 $\pm$ 24	2 $\pm$ 12	4 $\pm$ 22	-2; $\pm$ 14	Unclear	-3; $\pm$ 21	Unclear
Peak heart rate	3	0.4 $\pm$ 2.6	-0.1 $\pm$ 2.3	-1.3 $\pm$ 2.7	0.5; $\pm$ 2.0	Unclear	1.4; $\pm$ 1.9	Possibly trivial
	14	0.4 $\pm$ 2.6	-0.2 $\pm$ 2.7	-1.3 $\pm$ 3.3	0.5 $\pm$ 2.3	Unclear	1.4; $\pm$ 2.7	Possibly trivial
<b>Repeated sprint test</b>								
Peak lactate	3	-7 $\pm$ 12	-6 $\pm$ 26	1.2 $\pm$ 7.7	-1; $\pm$ 20	Unclear	-8 $\pm$ 10	Probably -ive
	14	-7 $\pm$ 17	-3 $\pm$ 21	-3 $\pm$ 24	-5; $\pm$ 16	Unclear	-2; $\pm$ 19	Unclear
<b>Hematology</b>								
Hemoglobin	-14	1.3 $\pm$ 4.9	1.5 $\pm$ 4.3	1.5 $\pm$ 3.1	-0.2; $\pm$ 4.0	Unclear	-0.1; $\pm$ 3.0	Unclear
	0	0.7 $\pm$ 4.6	0.9 $\pm$ 3.6	-0.2 $\pm$ 4.5	-0.2; $\pm$ 3.5	Unclear	1.0; $\pm$ 3.4	Unclear
	14	0.3 $\pm$ 3.4	-0.3 $\pm$ 4.3	-2.8 $\pm$ 3.2	0.5; $\pm$ 3.6	Unclear	2.9; $\pm$ 3.4	Probably +ive
Hematocrit	-14	0.0 $\pm$ 4.1	2.2 $\pm$ 8.1	1.7 $\pm$ 3.9	-2.2; $\pm$ 6.4	Unclear	-0.6; $\pm$ 4.2	Unclear
	0	0.5 $\pm$ 3.3	0.6 $\pm$ 3.5	-1.5 $\pm$ 5.3	-0.1; $\pm$ 3.2	Unclear	2.0; $\pm$ 3.9	Unclear
	14	-0.9 $\pm$ 3.1	-1.6 $\pm$ 4.2	-2.1 $\pm$ 3.8	0.7; $\pm$ 3.8	Unclear	0.9; $\pm$ 4.3	Unclear
Ferritin	-14	-17 $\pm$ 16	0 $\pm$ 18	-2 $\pm$ 11	-16; $\pm$ 45	Unclear	-7; $\pm$ 24	Unclear
	0	1 $\pm$ 24	-2 $\pm$ 22	16 $\pm$ 17	3; $\pm$ 27	Unclear	-15; $\pm$ 16	Possibly -ive
	14	-9 $\pm$ 6	9 $\pm$ 5	36 $\pm$ 67	-1; $\pm$ 17	Unclear	-33; $\pm$ 38	Unclear
2,3-DPG	-14	3.7 $\pm$ 7.7	4.3 $\pm$ 7.8	0.9 $\pm$ 6.6	-0.5; $\pm$ 7.7	Unclear	3.1; $\pm$ 6.2	Unclear
	0	-1.6 $\pm$ 7.7	1.7 $\pm$ 9.8	-1.3 $\pm$ 6.1	-3.3; $\pm$ 9.1	Unclear	1.4; $\pm$ 6.0	Unclear
	14	-6.2	1.2	-5.1	-7.2; $\pm$ 7.2	Probably -ive	2.6; $\pm$ 6.8	Unclear
Reticulocyte count	-14	3 $\pm$ 18	13 $\pm$ 20	5 $\pm$ 16	-9; $\pm$ 16	Unclear	3; $\pm$ 14	Unclear
	0	4 $\pm$ 10	13 $\pm$ 15	-5 $\pm$ 23	-8; $\pm$ 11	Possibly -ive	14; $\pm$ 17	Probably +ive
	14	25 $\pm$ 26	-2 $\pm$ 32	-21 $\pm$ 27	27; $\pm$ 30	Probably +ive	40; $\pm$ 32	Very likely +ive
White blood-cell count	-14	11 $\pm$ 39	5 $\pm$ 13	7 $\pm$ 16	5; $\pm$ 28	Unclear	1; $\pm$ 16	Unclear
	0	7 $\pm$ 13	-3 $\pm$ 14	10 $\pm$ 18	10; $\pm$ 13	Possibly +ive	-7; $\pm$ 12	Unclear
	14	0 $\pm$ 27	-3 $\pm$ 12	4 $\pm$ 16	3; $\pm$ 19	Unclear	-5; $\pm$ 15	Unclear

Data are changes in each group (mean  $\pm$  SD) and comparison of the changes (difference in the means;  $\pm$ 90% confidence limits) between the hypoxic groups and between the mean of the hypoxic groups and control. The practical inference is the qualitative assessment of the chances that the true effect is substantially positive (+ive) or negative (-ive).

<sup>a</sup>-14 d post-treatment is 5 d into the intervention.

<sup>b</sup>Based on a smallest beneficial or harmful change in performance of 1% for VO<sub>2</sub>max and exercise economy and 0.2 of the baseline between-subject standard deviation for all other measures.

$\pm$ 90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

## Discussion

This study was the first of its kind to examine the effect of different hypoxic exposure interval lengths on exercise performance and the first to report on any inflammatory effects associated with adaptation to hypoxia. We proposed that increasing the number of hypoxic transitions during an exposure period might result in a greater enhancement of performance. There were, however, no clear differences for any maximal or sub-maximal performance measures between the hypoxic treatments. There were also no clear differences in hematology between the hypoxic groups. Evidence for a difference in inflammatory response in the 3-min group relative to the 5-min group was inconsistent: IL-1 $\beta$  was elevated, but CRP was attenuated—a surprising finding, given that CRP is regulated by IL-1 $\beta$ .<sup>23</sup> Collectively, these results do not exclude the possibility of different effects of 3- vs 5-min intervals of hypoxia, but any differences are likely to be small.

The substantial enhancement in aerobic performance (~4%) in the combined hypoxic conditions is of magnitude similar to that in studies using similar protocols and athletes of similar ability (2-7%).<sup>7,8,10</sup> In contrast, studies with elite athletes have produced smaller or even negative effects. Hinckson et al.<sup>11</sup> observed only a 0.6% enhancement of power over 5000 m and an impairment of 2.2% over 500 m in ergometer trials with elite rowers, while Julian et al.<sup>12</sup> observed a 0.8% impairment in 3000-m speed with elite runners. Although differences in protocols could account for some of these differences in performance, it appears likely that highly trained athletes have less potential for enhancement than sub-elites with adaptation to hypoxia. In support of this notion, Hamlin and Hellemans<sup>8</sup> reported a trend towards less enhancement in 3-km running performance following adaptation to intermittent hypoxia in faster athletes (~1% vs 3% for the fastest vs the slowest). We have observed a similar trend, although there is too much uncertainty for the trend to be anything more than suggestive.

The 2% impairment in repeated sprint performance observed in the combined hypoxic groups at 3 d post-treatment is not consistent with the substantial improvement of 5-8% previously reported,<sup>7,10</sup> even allowing for uncertainty in the estimates. Repeated sprint performance is determined partly by aerobic capacity and lactate threshold,<sup>24</sup> so the gains in peak power and lactate-profile power in the hypoxic groups could have led to improved performance in the last few sprints. More research is required to clarify the effect of intermittent hypoxic exposure on repeated sprint performance.

The observed physiological changes following adaptation to hypoxic exposure provide several plausible mechanisms for the performance enhancement observed in the combined hypoxic groups. It is possible that the increase in peak power in part resulted from erythropoiesis leading to an increase in oxygen transport. On the final day of the intervention and 14 d post-intervention reticulocytes were substantially elevated in the combined hypoxic groups relative to the control, although these changes could in part reflect differences in training between groups<sup>25</sup>. In addition, hemoglobin concentration was elevated at 14 d post intervention. These findings are consistent with changes in hematology following adaptation to intermittent hypoxia in other studies<sup>7-10</sup>. Furthermore, these changes were accompanied by a substantial reduction in ferritin on the final day of the intervention, a finding reported consistently in previous studies.<sup>7,8</sup> This response is consistent with the decrement in ferritin observed while adapting to a natural altitude environment.<sup>26</sup> Collectively, these changes suggest that erythropoiesis could have contributed to an enhancement in the oxygen carrying capacity, but it is doubtful whether these effects alone accounted for the observed changes in endurance performance. Other researchers have found no direct evidence of erythropoiesis using similar protocols of intermittent hypoxia<sup>11,12</sup> or longer-exposure continuous protocols.<sup>2-4</sup>

Enhancements of performance resulting from erythropoiesis are typically accompanied by a change in  $\text{VO}_2\text{max}$ .<sup>27</sup> Such changes may have occurred in our study, but our uncertainty of change in  $\text{VO}_2$  measures makes interpretation of these measures difficult. An increase in 2,3-DPG would also facilitate greater offloading of oxygen to the tissues. While the change in 2,3-DPG for the combined groups was unclear, the change was at most (upper confidence limit 9.3%) about half that previously reported for continuous or overnight exposure to hypoxia, ~18%.<sup>28</sup> It is also possible that  $\text{VO}_2\text{max}$  was unaffected and that exercise economy improved, as reported by numerous investigators following adaptation to various hypoxic interventions with athletes<sup>2,4</sup>. While the effect of hypoxia on economy was unclear in our study, improvements in this measure along with  $\text{VO}_2\text{max}$  cannot be dismissed as mechanisms mediating our performance enhancement.

The substantial changes in lactate-profile and heart-rate profile power observed at 3 d post-intervention are consistent with previous studies.<sup>2,3,7,10</sup> Any enhancement in peak power as observed in the present study is likely to manifest itself as an improvement in lactate-profile and heart-rate profile power, but other mechanisms are possible. A change in lactate profile could also be due to changes in lactate metabolism, influenced by erythropoietic adaptations. It is known that altitude acclimatisation improves the

transport capacity for lactate and H<sup>+</sup> across the erythrocyte membrane.<sup>29</sup> Furthermore any increase in red-cell mass with acclimatisation<sup>1,29</sup> would increase the dilution space for lactate and protons released in the plasma by the exercising muscles; substantial erythropoiesis would therefore enhance the capacity of red cells to pick up lactate and protons from the plasma, as suggested by Messonnier.<sup>30</sup> The effect on heart-rate profile power could also result from increase in circulating blood volume, as suggested by Wood et al.<sup>10</sup> Any such increase would need substantial erythropoiesis, because an increase in plasma volume alone would lead to a decrease in hemoglobin concentration rather than the observed probable increase.

Finally, a change in anaerobic power has also being suggested as an adaptation to intermittent hypoxia mediated, for example, by an increase in muscle buffering capacity<sup>2</sup> and maximal accumulated oxygen deficit.<sup>31</sup> While we did not measure these variables directly, our indirect measures of anaerobic power were inconsistent with this notion. Power in the first sprint as a percent of peak incremental power *declined* by 4.8%, whereas this ratio would be expected to increase if anaerobic power improved. The finding is consistent with recent research reporting a 5.2 % decrease in the same variable following intermittent hypoxia<sup>7</sup>. In increase in peak lactate in the repeated-sprint test would also provide an indirect measure of an increase in muscle buffering, but peak lactate was substantially lower in the combined hypoxic groups. These data collectively suggest that adaptation to intermittent hypoxic exposure does not enhance anaerobic power.

Although hypoxia in tissues promotes an inflammatory response,<sup>13</sup> we found no clear differences in markers of inflammation between the combined hypoxic groups and control, and one of the markers (IL-1 $\beta$ ) demonstrated a trivial difference throughout the intervention. The most likely explanation is that the hypoxic stimulus was not of sufficient duration and intensity to elicit substantial changes that would be clear in our assays. In any further study of a potential role of inflammation, it would be important to take blood samples soon after exposure sessions to detect any acute inflammatory effects that subside overnight.

A limitation in the present study is the lack of blinding, which may have resulted in a contribution of the placebo effect to performance in the two experimental groups via harder training during the exposure period and more effort in the post tests. Together, these effects could account for at least part of the difference between the changes in performance in our experimental and control subjects. Controlled blind trials of intermittent hypoxia on average have produced little effect on endurance performance,

but the outcomes have been inconsistent, ranging between enhancements and impairments of up to 4% in mean power or its equivalent.<sup>8,10,11,32</sup> A meta-analysis would help account for these inconsistencies and, by comparing blinded with unblinded studies, provide an estimate of the placebo effect. The magnitude of the placebo effect in altitude training is an important issue, because all published studies of real altitude and of simulated altitude with long continuous exposures to hypoxia were performed without blinding. The published effects on performance for these protocols are therefore all potentially biased high.

In summary, the data in this investigation suggest that reducing the hypoxic interval length and recovery time during acutely intermittent hypoxia has little effect on exercise performance, hematology or inflammatory markers. Utilising either the 3-min or 5-min protocols employed in the present study is sufficient to elicit substantial improvements in endurance performance and some changes in hematology. Any role of inflammatory responses is as yet unclear. Further research is required to clarify the mechanisms mediating the performance changes.

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## CHAPTER 5

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### Sea-level exercise performance following adaptation to hypoxia: a Meta-Analysis

#### Abstract

Adaptation to living or training in hypoxic environments (altitude training) continues to gain interest from sport scientists and endurance athletes. Here we present the first meta-analytic review of the effects on performance and related physiological measures following adaptation to six protocols of natural or artificial hypoxia: live-high train-high (LHTH), live-high train-low (LHTL), artificial LHTL with long or brief continuous or intermittent daily hypoxia, and artificial live-low train-high (LLTH). The 51 qualifying studies provided 11-33 estimates for effects on power output with each protocol and up to 20 estimates for effects on maximal oxygen uptake and other potential mediators. The meta-analytic random-effect models included covariates to adjust for and estimate moderating effects of study characteristics such as altitude level and days of exposure. Poor reporting of inferential statistics limited the weighting factor in the models to sample size. Probabilistic inferences were derived using a smallest worthwhile effect on performance of 1%. Substantial enhancement of maximal endurance power output in controlled studies of subelite athletes was very likely with artificial brief intermittent LHTL (2.6%; 90% confidence limits  $\pm 1.2\%$ ), likely with LHTL (4.2%;  $\pm 2.9\%$ ), possible with artificial long continuous LHTL (1.4;  $\pm 2.0\%$ ), but unclear with LHTH (0.9;  $\pm 3.4\%$ ), artificial brief continuous LHTL (0.7%;  $\pm 2.5\%$ ) and LLTH (0.9%;  $\pm 2.4\%$ ). In elite athletes enhancement was possible with natural LHTL (4.0%;  $\pm 3.7\%$ ) but unclear with other protocols. There was evidence that these effects were mediated at least partly by substantial placebo, nocebo and training-camp effects with some protocols. Enhancing protocols by appropriate manipulation of study characteristics produced clear effects with all protocols (3.5 to 6.8%) in subelites, but only with LHTH (5.2%) and LHTL (4.3%) in elites. For maximal oxygen uptake increases were very likely with LHTH (4.3%;  $\pm 2.6\%$ ) in subelites, whereas in elites a *reduction* was possible with LHTH (-1.5%;  $\pm 2.0\%$ ); changes with other protocols were unclear. Effects on erythropoietic and other physiological mediators provided little additional insight into mechanisms. In conclusion, natural live-high train-low currently provides the best protocol for enhancing endurance performance in elite and subelite athletes, while some artificial protocols are effective in subelites. Likely mediators include maximal oxygen

uptake and the placebo, nocebo and training-camp effects. Modification of the protocols presents the possibility of further enhancements, which should be the focus of future research.

## Introduction

When an athlete ascends from sea level to moderate altitude, the shortage of oxygen (hypoxia) initially impairs endurance training and performance. After a few weeks at altitude, training and performance recover to some extent as the athlete adapts. If the athlete then returns to sea level, do the adaptations lead to enhancement of endurance performance? Coaches have long thought so, but studies aimed at this question appeared to be inconclusive, leading researchers to suspect that any benefit from adaptation to hypoxia was offset by loss of endurance fitness consequent to the reduction in training intensity <sup>[1]</sup>. The focus of research on altitude training then moved from this traditional “live-high train-high” approach (LHTH) to live-high train-low (LHTL), in which athletes live and sleep at altitude but descend regularly to lower altitude for training sessions <sup>[2]</sup>. LHTL appeared to be more successful, and interest has grown in the use of nitrogen houses, hypobaric chambers, altitude tents or hypoxic inhalers to adapt to hypoxia and train normally without having to travel up and down a mountain <sup>[1]</sup>. Researchers have investigated three such approaches to artificial LHTL: continuous exposure to a simulated moderate altitude for periods of 8-18 hours per day (artificial long continuous LHTL); continuous exposure to a simulated moderate-high altitude for 1.5-5 hours per day (artificial brief continuous LHTL); and intermittent exposure to a simulated high altitude for <1.5 hours per day (artificial brief intermittent LHTL). The same devices have also been used to simulate moderate altitude while the athlete exercises continuously or intermittently for at least 0.5 hours per session (artificial LLTH).

While there is general agreement that adaptation to some forms of hypoxia can enhance sea-level performance, there has been considerable debate recently about the physiological mechanisms <sup>[3-5]</sup>. Gore and Hopkins <sup>[4]</sup> provided a rationale for understanding the mechanisms underlying effects on maximal performance of differing durations. Exercise intensities below maximal oxygen uptake (>10 min duration) are sustained essentially by aerobic power, whereas exercise intensities above maximal oxygen uptake are sustained by a combination of aerobic and anaerobic power. Aerobic power consists of three components: maximal oxygen uptake, the fraction of maximal

uptake that can be sustained during the exercise, and economy or efficiency of conversion of oxygen consumption into power output <sup>[6]</sup>. Changes in endurance performance following adaptation to hypoxia could therefore be due to changes in any of these three components, along with any changes in the contribution of anaerobic power for supramaximal exercise. Researchers who are interested in the mechanisms underlying the performance effects of hypoxic adaptation measure one or more of these components or the more fundamental physiological variables underlying them.

There has been no previous meta-analytic review of the effects on performance or related physiological measures following adaptation to any of the artificial or natural forms of altitude training. The current review addresses this deficit. There were sufficient studies to allow us to meta-analyze separately the effects on performance of the six natural and artificial altitude protocols. By far the most popular potential mechanism variable has been maximal oxygen uptake, and we have also been able to meta-analyze this variable with all six protocols. Researchers have long argued that enhancements in maximal oxygen uptake are mediated by erythropoiesis <sup>[1,5,7-9]</sup>, so measurements of erythropoietin, reticulocytes, red cell mass, hemoglobin mass, hemoglobin concentration, and ferritin have also been reported. We have been able to meta-analyze hemoglobin concentration for LHTH and artificial brief intermittent LHTL, but we had to meta-analyze hemoglobin mass and red cell mass by combining them across all protocols. We were able to perform only a graphical analysis for erythropoietin, reticulocytes, and ferritin, owing to the small number of estimates for these variables. Mechanisms underlying anaerobic power are less popular with researchers, and only the indirect measure of anaerobic power represented by peak blood lactate following an exercise test was reported in sufficient studies for meta-analysis in LHTH and artificial brief intermittent LHTL.

## **Methodology**

### **1.1 Study Selection**

Searches of Pubmed, SportDiscus and Google Scholar were performed for studies published in English up to and including April 2007. Reference lists in review and original-research articles identified were also examined. The primary focus of the meta-analysis was performance. We therefore included studies of performance measured at or near sea level (<1000 m). Studies published only as conference abstracts were not excluded. We included studies with measures of oxygen consumption directly related to

endurance performance, but studies reporting hematological or other parameters not directly related to performance and lacking a performance measure were excluded. Several studies were excluded due to poor reporting of data or for not assessing performance at or near sea level <sup>[10-24]</sup>. Other reasons for excluding studies were: a performance enhancement of 19% in 5-mM lactate speed in elite runners, when other measures of performance increased by 0.6% and 1.1% <sup>[25]</sup>; the only uncontrolled study in LLTH and with only 5 athletes <sup>[26]</sup>; and the only uncontrolled study of the brief continuous LHTL protocol <sup>[27]</sup>. The descriptive statistics for the 51 qualifying studies are shown in Table 5.1.

## 1.2 Data Extraction

The study-estimates for the treatment effect were calculated for estimates without a control group by dividing the mean post-score by the mean pre-score for the experimental group and expressing the ratio as a percent; for estimates with a control group the post/pre ratio in the experimental group was divided by the post-pre ratio in the control group before converting to a percent. Percent change in performance time in time trials was converted to change in mean power output by multiplying by an appropriate factor derived from power-velocity relationships <sup>[28]</sup>. For running the factor was -1; for cycling the factor was -2.5; for swimming the factor was -2.0, which was an index  $x$  derived from first principles <sup>[28]</sup> by fitting the power-velocity relationship  $P=kV^x$  to published data <sup>[29]</sup>. For any exercise modality, the percent change in time to exhaustion at a constant power was converted to percent change in power output in an equivalent time trial of the same duration by multiplying by a factor derived from models for the power-duration relationship of human performance, as follows: for supramaximal tests (<7.5 min), the factor was  $0.50/T$ , where  $T$  is the time in minutes <sup>[30]</sup>; for submaximal tests (>7.5 min), the factor was approximately  $1/15$  <sup>[28]</sup>. Percent change in time to exhaustion in incremental tests was converted to percent change in peak power by multiplying by a factor  $1-f$ , where  $f$  was the power of the first stage of the test expressed as a fraction of the peak power, under the assumption that the load increased linearly to maximum. A spreadsheet containing all study-estimates can be obtained from the authors.

## 1.3 Meta-analyses

The main outcome from a meta-analysis is a weighted mean of values of an outcome statistic from various studies, where the weighting factor is usually the inverse of the

square of the sampling standard error of the statistic. The standard error is derived from either the confidence interval or p value of the statistic or from standard deviations of change scores in control and experimental groups. Unfortunately, 55% of the study-estimates for performance that would have otherwise qualified for inclusion in our meta-analyses did not have sufficient information to derive the standard error; for estimates from studies other than of intermittent artificial LHTL, the figure is 71%. The main problem was reporting of statistical significance or non-significance as a P value inequality without any further inferential information. To exclude all these studies from the meta-analyses would have resulted in unacceptable bias, akin to the publication bias that arises from failure of authors to submit studies with non-significant outcomes or failure of journal editors to accept them. We therefore performed the meta-analyses with a weighting factor derived from the sample size for each study-estimate. The factor was (study sample size)/(mean study sample size). For a controlled trial with groups of unequal size  $n_1$  and  $n_2$ , we used an effective sample size of  $4n_1n_2/(n_1+n_2)$ . The sample size of uncontrolled trials was inflated by a factor of 4 to give them the effective sample size of a controlled trial. (An uncontrolled trial would effectively have such a sample size, when the weighting factor is the inverse of the sampling variance.) To ensure studies with different numbers of estimates would have equal weighting, each study's weighting factor was divided by the number of estimates it provided and multiplied by the mean number of estimates in all the studies contributing to the meta-analysis. The resulting meta-analyzed effect is equivalent to that produced in a random-effect meta-analysis, with the assumption that the dependent variable giving rise to the study-estimates has the same error of measurement in all studies.

The meta-analyses were performed with the mixed modeling procedure (Proc Mixed) in the Statistical Analysis System (Version 9.2, SAS Institute, Cary, NC). Percent effects were converted to factors ( $= 1 + \text{effect}/100$ ), log transformed for the analysis, then back transformed to percents. Study characteristics were the fixed effects in the model; these were included as main effects only, because of the limited number of study-estimates. We limited the characteristics to those that were included in most studies and that might be expected on physiological or psychological grounds to moderate the effect of hypoxia: competitive status (elite vs subelite); design characteristics (uncontrolled vs controlled trial, non-blind vs blind trial); sex (males as a fraction of sample); training phase (competitive vs non-competitive or unknown); altitude level or its equivalent for artificial altitude (m); hours of hypoxia per day (for LHTH, LHTL and artificial long-duration LHTL); minutes of hypoxia per day (for the remaining protocols, not counting

minutes spent in normoxia between intervals of hypoxia); count of days when any exposure to hypoxia occurred; total count of treatment days, including any days resting from exposure; ratio of exposure/treatment days; day post-exposure when performance was tested; training intensity on a 1 to 4 scale (for LLTH); type of performance test (submaximal vs maximal); and duration of maximal exercise tests (min). Missing values for sex of 9 and 10 subelite runners experiencing LHTH<sup>[31]</sup> and of six subelite runners experiencing LHTL<sup>[32]</sup> were assigned the mean value of proportion of males for their protocols. Competitive status was deemed elite if the athletes were in a national team and competing at international level. The four points of the training-intensity scale for LLTH were: above maximal oxygen uptake, 4; around maximal oxygen uptake, 3; around anaerobic threshold, 2; below anaerobic threshold, 1. Post-exposure test day and duration of maximum exercise tests were log transformed before analysis and included as simple linear predictors. Supplementary analyses (not shown in Table 5.2) were also performed, where possible, with post-exposure test day included as a quadratic or cubic polynomial in  $\times/\div$ SD units, to investigate the possibility of peaks or troughs in performance.

An effect of a study characteristic is not shown in the tables for one or more of the following reasons: there was insufficient variation in the characteristic between study-estimates to estimate the effect; collinearity with other study characteristics prevented its estimation; and the small number of study-estimates limited the analysis to only a few characteristics. To compare effectiveness of protocols on performance, the meta-analyzed effects are shown for subelite athletes (all protocols) and elite athletes (four protocols) and are adjusted to 100% controlled trials and 100% maximal tests. For all the other study characteristics we could not adjust to the same common value, so the effects on performance for each protocol are shown evaluated at the mean values of the study characteristics for that protocol.

In most models it was possible to include a random effect to estimate pure between-study variation in the effect of the treatment, expressed as a standard deviation. In principle this measure of between-study variation is free of sampling variation arising from error of measurement in the dependent variable, but use of sample size as the weighting factor does not produce clean partitioning of random error into pure between-study variation and residual error. The standard deviation representing the residual error in such models is the standard error of a study-estimate with the mean sample size ( $n$ ) of the meta-analyzed estimates; this standard error was multiplied by  $\sqrt{(n/8)}$  to provide an estimate of the mean standard error of measurement of the dependent variable. When

there were insufficient study-estimates to include a pure between-study random effect, the residual random effect is shown as the between-study standard deviation.

For each outcome measure, a novel funnel plot of the inverse of an estimate's weighting factor (Y axis) vs the value of the estimate's random effect (X axis) was examined qualitatively for evidence of outliers (points judged visually to be more than about four standard deviations of horizontal scatter away from the center of the plot) and publication bias towards positive effects (positive trend in the scatter). This procedure did not result in exclusion of any estimates.

We reported uncertainty in the meta-analyzed estimates as 90% confidence limits, and we made probabilistic magnitude-based inferences about the true (large-sample) values of outcomes, as described elsewhere <sup>[33]</sup>. In brief, an outcome was deemed unclear if its confidence interval overlapped the thresholds for smallest worthwhile positive and negative effects; equivalently, effects were unclear if chances of the true value being substantially positive and negative were both >5%. The magnitude of a clear effect was reported as the magnitude of its observed value, sometimes with an assertion about the probability the true value was substantial. The probabilities for each meta-analyzed effect and for pairwise comparisons of effects were derived using a published spreadsheet <sup>[34]</sup>. The thresholds for smallest effects on performance were assumed to be  $\pm 1\%$ , which is an approximate average across a range of sports <sup>[35-37]</sup>. Smallest effects on maximal oxygen uptake, hemoglobin or red-cell mass, and exercise economy were also assumed to be  $\pm 1\%$ , on the assumption that a 1% change in these measures would result in a similar change in endurance performance. For hemoglobin and peak lactate concentration there is no direct relationship with performance; effects were therefore standardized by dividing by the mean between-subject standard deviation of these variables in the studies that contributed to their meta-analyses, and a modified Cohen scale was used to make inferences <sup>[38]</sup>.

## Results

### 2.1 Exercise Performance

The meta-analyzed outcomes for the six protocols of natural and artificial altitude are shown in Table 5.2. Substantial enhancement of power output in subelite athletes was very likely with artificial brief intermittent LHTL, likely with LHTL, possible with artificial long continuous LHTL, but unclear for LHTH, artificial brief continuous LHTL and LLTH. Comparisons between the protocols for subelites revealed that LHTL

was likely better than all protocols, with the exception of artificial brief intermittent LHTL, where the difference was unclear. Artificial brief intermittent LHTL was possibly better than artificial long continuous LHTL, artificial brief continuous LHTL and LLTH. All other differences between protocols were unclear. Enhancements of mean power in elite athletes were likely with LHTL, but unclear for all other protocols. In comparison with the other protocols in elites, LHTL was likely better than artificial long continuous LHTL and artificial brief intermittent LHTL. All other differences between protocols were unclear.

Several of the study characteristics listed in Table 5.2 moderated the effects of hypoxia; performance was better in controlled relative to uncontrolled studies for LHTL, but the opposite was observed for LHTH; subelite athletes had a clear enhancement in performance relative to elites with artificial brief intermittent LHTL, and submaximal exercise performance was clearly impaired relative to maximal with artificial long continuous LHTL. Effects for blinding, competitive phase, and sex were unclear for the few protocols where these effects could be estimated. Post-exposure test day had a substantial clear positive linear effect for LLTH and trivial or unclear effects with the other protocols. Quadratic or cubic effects of post-exposure test day (not shown in Table 5.2) could not be modeled with the two shortest protocols of artificial altitude, and the polynomials revealed little curvature with LHTL ( $<0.3\%$  over  $\times/\div SD^2$  either side of the mean time). However, relative to the effect at the mean post-test time, LHTH showed some evidence of enhancement at very short times (1.8% at  $\div SD^2$  or  $\sim 2.5$  d) followed by impairment (-1.5% at  $\div SD$  or 5 d), enhancement (1.4% at  $\times SD$  or 17 d) and impairment (-2.3% at  $\times SD^2$  or 33 d); artificial long continuous LHTL showed a peak at the mean post-test time with a relative impairment of 1-2% either side of the mean (at  $\times/\div SD$  or 2.5 and 13 d); and LLTH showed a trough at the mean time with relative enhancements either side (3.8% at  $\div SD$ , or 1.5 d; and 1.0% at  $\times SD$  or 5 d).

The moderating effect of study characteristics provides an avenue for enhancing each protocol, as shown in Table 5.2 for the effects on performance after changing selected characteristics by  $\pm$  or  $\times/\div$  one standard deviation. Improvements in power output were observed in subelite athletes for all protocols after these theoretical enhancements, the increase ranging from 0.4% for LHTL through 5.9% for LLTH. The resulting effects were all clearly beneficial for subelite athletes, but beneficial effects for elites were clear only for LHTH and LHTL. Alterations to the altitude level, days of exposure and daily exposure hours had the biggest contribution to the enhanced protocols, whereas effects for other characteristics were generally trivial or unclear. Modifying test duration

by one standard deviation would also have produced substantial enhancements in performance, especially for LHTH, but this characteristic was not included, because the mean duration of tests was reasonably similar across the protocols, and changing the performance test does not represent a change to an exposure protocol.

## 2.2 Physiological Measures

The meta-analyzed effects on sea-level maximal oxygen uptake are shown in Table 5.3. There was a very likely enhancement with LHTH and a possible enhancement with LLTH in subelite athletes. The trivial effect for artificial LHTL with predominantly subelite athletes is very unlikely to have arisen from a substantial true positive effect. The unclear effects for the remaining two artificial protocols represent changes in maximal oxygen uptake that were either unlikely to be positive (brief continuous LHTL) for subelite athletes or were possibly positive (brief intermittent LHTL) for predominantly subelite athletes. For elite athletes there was a possible *impairment* with LHTH but an unclear effect for LHTL. It was not possible to estimate effects for elites alone in the other protocols.

Study characteristics moderating maximal oxygen uptake are also shown in Table 5.3. The most interesting effect of characteristics with the natural protocols was the increase in maximal oxygen uptake with increasing time post exposure (clear for LHTH, unclear for LHTL), indicating that there is more benefit at least for maximal oxygen uptake around two weeks after the intervention period. The trivial effect in artificial long continuous LHTL can be converted into a positive effect by increasing the hours of exposure; there is also a possibility of less benefit from *more* days of exposure, even though the mean days of exposure is already about a week less than for the natural protocols. A reduction in training intensity with LLTH would promote a further increase in maximal oxygen uptake. The remaining effects of study characteristics on maximal oxygen uptake were unclear.

Hemoglobin mass (including red-cell mass) and exercise economy were meta-analyzed for all studies collectively because of the lack of study-estimates. Effects for hemoglobin mass were unclear, but an increase in exposure days and possibly an increase in altitude would produce a clear increase, whereas delaying the test day later by more than 1 SD (>10 d) would offset the increase. The effect on exercise economy was trivial, but a substantial increase could accrue from reducing exposure days and increasing altitude (Table 5.4).

Hemoglobin concentration and peak lactate could be meta-analyzed only for LHTH and brief intermittent artificial LHTL. For the interpretation of magnitude, the average pre-test between-subject standard deviation for hemoglobin concentration was 6.2%, while that for peak lactate was 21%. Hemoglobin concentration demonstrated a likely moderate increase for LHTH and a possible small increase for artificial brief intermittent LHTL. The moderating effect of post-exercise test day shows that the increase in hemoglobin concentration was lost 3-4 weeks after exposure. The effect for peak lactate was unclear with LHTH, but an increase in altitude would produce a clear small to moderate decrease, whereas delaying the test day would produce a similar (but unexpected) decrease. Peak lactate showed a trivial decrease for artificial LHTL. The effect for peak lactate in artificial brief intermittent LHTL was trivial, but the uncertainty allows for the possibility of a small negative true effect.

Effects for other physiological measures that could not be meta-analyzed due to insufficient data are shown in Figure 5.1. Erythropoietin was elevated during the hypoxic interventions and possibly showed a small elevation afterwards. Reticulocytes appeared to be elevated in a few studies during the intervention. The scatter in the plot for ferritin makes any conclusion about trend difficult. Plots of performance vs maximal oxygen uptake, hemoglobin or red-cell mass and exercise economy are shown in Figure 5.2. There was a modest linear relationship between performance and maximal oxygen uptake, but no evidence of such a relationship between performance and the other two variables.

## Discussion

In this first meta-analysis of sea-level exercise performance following adaptation to hypoxic exposure, we observed clear enhancements in endurance power output of 1-4% in subelite athletes with LHTL and with two of the artificial-altitude protocols (long continuous and brief intermittent LHTL). In elite athletes the enhancements were clear only with LHTL. Modification of study characteristics might result in clear enhancements of 3-7% with all protocols in subelite athletes, but effects in elites would be clear only for LHTH and LHTL.

Following the development of the LHTL approach, the use of LHTH has received little support from sport scientists. There is enough uncertainty in our estimates of the effect of LHTH to allow for enhancements in elites and subelites with this protocol. Furthermore, our estimates are for controlled trials, whereas athletes in an altitude camp would experience the equivalent of an uncontrolled trial, giving a possible further

increase of ~3% (Table 5.2). The LHTH protocol also showed effects of post-exposure test day consistent with anecdotal reports of coaches that performance is enhanced immediately after altitude and peaks again several weeks later. Taken together these results provide reasonable support for what is still a widely accepted practice among many elite coaches and athletes. LHTH was also one of only two protocols that produced clear enhancements in endurance performance for elites with appropriate manipulation of study characteristics. These moderating effects show that it may be better for athletes to go to higher altitudes (~2400 m) for shorter periods (~16 d) around 2-3 wk before an important competition.

Our results provide good evidence for the effectiveness of LHTL which was clearly better than all but one protocol in subelites (brief intermittent LHTL) and elites (LHTH). The only moderating effect of study characteristics with LHTL was unexpected: uncontrolled trials showed a clear negative effect relative to controlled trials. According to conventional wisdom, uncontrolled studies should show *larger* enhancements due to the so called “training-camp effect”, which in principle is adjusted for in a controlled trial. What may happen in reality is that subjects in the control group of a controlled trial experience less of the training-camp effect, because they do not train as hard. There could also be a contribution from a “nocebo effect”, whereby subjects in a control group perform worse, because they know they are in the control group. There is evidence of a nocebo effect in the classic natural LHTL study of Levine and Stray-Gundersen <sup>[2]</sup> that is especially clear when the data are presented graphically as percent changes (see Figure 1 at <http://www.sportsci.org/traintech/altitude/wgh.html>). Indeed, data for the effect of uncontrolled vs controlled LHTL studies came entirely from this study. Therefore our meta-analyzed effect of ~4% for controlled studies needs to be interpreted with caution. When performance is predicted for uncontrolled studies (as previously mentioned, the way athletes train) the effect becomes a more realistic ~1.5%. The only design that avoids the nocebo problem is a blind trial, which is not possible with natural LHTL. Further research with controlled trials is warranted to assess the potential of LHTL.

Artificial LHTL with long continuous exposures was developed to simulate LHTL, and our analysis provides some support for its efficacy. The limitation with this approach appears to be insufficient exposure to hypoxia, because the moderating effects of study characteristics show that the effect on performance can be increased by increasing altitude and adding daily exposure hours. This result is consistent with the suggestions of researchers who believe that at least 12 hours of daily exposure is critical

for the success of this protocol <sup>[7,39]</sup>. Another substantial moderating effect was a reduction in performance with increasing days of exposure, similar to that with LHTH. This result for both protocols seems counter-intuitive, although a ready explanation is a short-acting placebo effect. The only other moderating effect was a substantial downward adjustment for submaximal performance, which again implicates a placebo effect. More studies are needed to clarify the role of placebo effects with this and other protocols.

At the opposite end of the spectrum of daily hypoxic exposure, artificial LHTL with brief intermittent exposures was one of the best protocols in subelites. The moderating effects of study characteristics provided only marginal improvements of 1%, mainly through maximizing the exposure days in the intervention period. The equivalent altitude of this protocol is already at the limit for ethical approval, so there is no option to investigate higher altitudes. Alteration of the hypoxic and normoxic intervals is a possible avenue for improvement, although we have found no clear difference between the effects of 3- and 5-min intervals <sup>[40]</sup>. The clear difference between the effect on subelite and elite athletes suggests that the waves of hypoxia are less effective in elite athletes, possibly because elites experience more hypoxia in their muscles from higher intensities of training in comparison with subelites.

With the remaining two forms of artificial altitude exposure, the uncertainty in the meta-analyzed estimates was too large for their trivial magnitudes to be clear, although clear enhancements were possible with adjustment of appropriate study characteristics. With brief continuous LHTL, the average altitude appears to have been too high, since a reduction in altitude by 1 SD could produce a substantial enhancement in performance. Reducing the altitude may seem an implausible way to enhance this protocol, but the reduction by 1 SD would bring the altitude to ~3700 m, which is still well above that of the other continuous protocols and which could conceivably provide a sufficient hypoxic stimulus without the negative sequelae of continuous exposure to high altitude. A reduction in altitude along with a reduction in training intensity would also enhance performance with LLTH, but the main enhancement for this protocol would come from the more reasonable strategy of increasing days of exposure. LLTH also showed evidence that performance could be better either side of the mean post-exposure test day (~3 d), but it seems to us that this protocol is the least likely to produce performance enhancement.

A study characteristic not included in the above discussion of the individual protocols was test duration, because altering this characteristic does not alter the

exposure protocol. There are nevertheless implications for the effects on aerobic vs anaerobic performance. Our results demonstrate that performance could be improved in LHTH and brief intermittent LHTL with tests of longer duration. In all other protocols performance could be better by a trivial margin for shorter tests. The average test duration in all protocols was 4-11 min, making all tests highly aerobic, but with only one of the protocols (LLTH) would a 1-SD reduction in test duration make the tests substantially anaerobic. We need more studies with shorter tests to clarify the effect on anaerobic performance.

Insights into the practical application of the findings of the meta-analyses can also be gleaned from a consideration of the between-study standard deviations (bottom of Table 5.2). These standard deviations represent unexplained variation in the mean effect of the protocol from study to study; as such, their magnitude is the typical deviation from the meta-analyzed mean effects that a researcher or practitioner can expect to experience in another study using the mean protocol with a group or squad. For natural and artificial brief intermittent LHTL these standard deviations in combination with the uncertainties in the mean effects imply that most researchers and practitioners will observe substantial enhancements in performance with a group or squad of subelite athletes. A beneficial outcome is less certain for elites with the natural protocols and for subelites with artificial long continuous LHTL; for the remaining protocols with subelites or elites the outcomes could be good, bad or indifferent. However, if the enhanced protocols are as good as shown, the influence of the between-study standard deviation could be nullified for all protocols.

The standard errors of measurement estimated from the meta-analyses (bottom of Table 5.2) do not have an immediate practical application, but they do provide evidence that the uncertainties in the meta-analyzed mean effects and in the moderating effects of study characteristics are trustworthy. These uncertainties are estimated from a combination of the between-study standard deviations and the standard errors of measurement, so it is important that the standard errors of measurement estimated from the meta-analysis are realistic. The low value for natural LHTL (0.7%) is a reflection of the fact that almost all of the performance tests in these studies were time trials with runners. This value and the other values for error of measurement, given their uncertainties, are within the normal range for tests of endurance performance <sup>[28]</sup>.

It is important to understand that some individual athletes may obtain no benefit or even impairment in performance from adaptation to hypoxia, even with those protocols that are clearly beneficial. Meta-analysis cannot address the question of individual

responses to treatments until researchers provide complete inferential information about experimental and control groups in the form of confidence limits, exact p values, or best of all, standard deviations of change scores. Such information would also allow the use of the inverse of sampling variance instead of sample size as a weighting factor in the meta-analysis, which would result in more trustworthy and probably narrower uncertainties in the meta-analyzed mean and between-study standard deviations.

Turning to the analysis of potential mechanism variables, it is clear from the findings in Table 5.3 that adaptation to hypoxia can result in enhancements in maximal oxygen uptake. The usual mechanism suggested for an increase in this variable is erythropoiesis, which would effect a change in hemoglobin or red cell mass with a resulting increase in blood volume, cardiac output or oxygen-carrying capacity. Our meta-analyses provide limited evidence for this mechanism: the meta-analyzed effect on hemoglobin mass was unclear on average, although extra exposure to hypoxia and a higher altitude level could result in a substantial increase. The meta-analyzed effects on hemoglobin concentration provide some additional indirect evidence for an increase in hemoglobin mass, but an alternative explanation for the increase in hemoglobin concentration often mentioned by researchers is a dehydrating effect of acclimatization to altitude <sup>[1]</sup>. Direct evidence of erythropoiesis from levels of EPO and reticulocytes could not be provided by meta-analysis, owing to insufficient data, but it is reasonably clear from Figure 5.2 that these variables increase transiently to some extent in some studies. Any erythropoiesis that did occur was not accompanied by clear reductions in ferritin.

Do the changes in maximal oxygen uptake mediate the changes in performance? The pattern of the effects on maximal oxygen uptake in Table 5.3 across different protocols for elite and subelite athletes and for the moderating effects of study characteristics does not mirror closely the effects on performance in Table 5.2. Figure 5.2 also shows only a modest positive relationship between the study estimates of maximal oxygen uptake and performance. Furthermore, a positive relationship between changes in hemoglobin mass and changes in performance—the expected outcome if the changes in maximal oxygen uptake were mediated by erythropoiesis—was not observed (Figure 5.2), although the noise with hemoglobin mass measurement (which manifests as a large between study CV, Table 5.4) could attenuate a true substantial relationship. Thus, it is possible that variables other than maximal oxygen uptake also mediate the effects on performance.

There were insufficient data to meta-analyze the effects of exercise economy for each protocol, but a single analysis for all protocols and the relationship between exercise economy and sea-level performance (Figure 5.2) provided little evidence for this mechanism. The only other physiological variable we meta-analyzed, peak lactate concentration, is not a contender as a primary mechanism of performance enhancement, but an increase in peak blood lactate would indirectly implicate buffering capacity. For the two protocols we meta-analyzed, a substantial increase in peak blood lactate was either unlikely (LHTH) or very unlikely (brief intermittent LHTL), so an increase in buffering capacity is presumably not involved with adaptation to these protocols. Other mechanisms therefore need to be identified, particularly for the artificial LHTL protocols, where gains in performance appear to be due at least partly to placebo or nocebo effects and where an increase in maximum oxygen uptake apparently does not contribute. The suggestion of a change in cardiovascular regulation resulting in more cardiac output to exercising muscle<sup>[4]</sup> is plausible but will be hard to investigate.

## **Conclusions**

There is now good evidence that subelite athletes can experience endurance performance enhancements with adaptation to natural altitude exposure and to brief intermittent and long continuous protocols of artificial altitude exposure. For elites, enhancements in endurance performance were possible only with the natural live-high train-low protocol. The enhancements with natural altitude could be mediated in part by maximal oxygen uptake, but placebo effects, nocebo effects, training-camp effects, and other mechanisms may be involved with these and the artificial protocols. Modification of the protocols presents the possibility of further enhancements, which should be the focus of future research using double-blind designs, performance measures with smaller errors of measurement, and putative physiological mediators. Reviewers and editors should ensure that studies accepted for publication contain complete inferential information about the effects in treatment and control groups.

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**Table 5.1.** Characteristics of study groups included in the meta-analysis sorted by protocol and first author.

Study	Subjects	Sample Size <sup>a</sup>	Design <sup>b</sup>	Competitive Level	Training Phase	Hypoxic Hours per Day <sup>c</sup>	Exposure/ Intervention Days <sup>d</sup>	Altitude Level <sup>e</sup> (m)	Hypoxia Device
<b>Live-high train-high</b>									
Bailey, 1998 <sup>[41]</sup>	Runners	8M,2F;14M,5F	C	Elite	?	24	28	1640	-
Bailey, 1998 <sup>[41]</sup>	Runners	9M,5F;6M,3F	C	Elite	?	24	28	1750	-
Burtscher, 1996 <sup>[42]</sup>	Runners	10M;12M	C	Subelite	?	24	12	2315	-
Friedmann, 1999 <sup>[43]</sup>	Boxers+Fe <sup>f</sup>	9M	U	Subelite	Off-season	24	18	1800	-
Friedmann, 1999 <sup>[43]</sup>	Boxers-Fe <sup>f</sup>	7M	U	Subelite	Off-season	24	18	1800	-
Gore, 1998 <sup>[44]</sup>	Cyclists	8M	U	Elite	?	24	31	2690	-
Ingjer, 1992 <sup>[45]</sup>	Skiers	7M;7M	U	Elite	Competitive	24	21	1900	-
Jensen, 1993 <sup>[46]</sup>	Rowers	9M;9M	C	Elite	Competitive	24	21	1822	-
Levine, 1992 <sup>[31]</sup>	Runners	10?	U	Subelite	?	24	28	1200	-
Levine, 1992 <sup>[31]</sup>	Runners	9?	U	Subelite	?	24	28	2500	-
Levine, 1997 <sup>[2]</sup>	Runners	9M,4F;9M,4F	C	Subelite	Competitive	24	28	2500	-
Miyashita, 1988 <sup>[47]</sup>	Swimmers	12M,8F	U	Elite	Competitive	24	21	2300	-
Pyne, 1998 <sup>[48]</sup>	Swimmers	14M,8F	U	Elite	Competitive	24	21	2102	-
Rusko, 1999 <sup>[49]</sup>	Skiers	14M;7M	C	Elite	?	24	22	1700	-
Saunders, 2004 <sup>[50]</sup>	Runners	10M;13M	C	Elite	?	24	20	1750	-
Svedenhag, 1991 <sup>[51]</sup>	Runners	5M;4M,2F	C	Elite	?	24	14	2000	-
Svedenhag, 1997 <sup>[52]</sup>	Skiers	5M,2F	U	Elite	?	24	30	1900	-

### Live high train low

Dehnert, 2002 <sup>[53]</sup>	Triathletes	6?;10?	C	Subelite	?	~18-24	13	1956/800	-
Levine, 1997 <sup>[2]</sup>	Runners	9M,4F;9M,4F	C	Subelite	Competitive	~18-24	28	2500/1200	-
Stray-Gundersen, 1994 <sup>[32]</sup>	Runners	6?	U	Subelite	?	~18-24	28	2500/1200	-
Stray-Gundersen, 2001 <sup>[8]</sup>	Runners	8F,14M	U	Elite	Competitive	~18-24	27	2500/1200	-
Wherlin, 2006 <sup>[54]</sup>	Orienteers	5M,5F	U	Elite	Pre-season	~18-24	24	2456/1000	-
Witkowski, 2001 <sup>[55]</sup>	Runners	8M,4F	U	Subelite	?	~18-24	28	1780/1200	-

### Artificial long continuous live-high train-low

Clark, 2004 <sup>[56]</sup>	Cyclists, triathletes	9M;10M	C	Subelite	?	9-10	20	2650	N <sub>2</sub> house
Clark, 2004 <sup>[56]</sup>	Cyclists, triathletes	10M;10M	C	Subelite	?	9-10	20/24	2650	N <sub>2</sub> house
Gore, 2001; Hahn, 2001 <sup>[57,58]</sup>	Triathletes	6M;6M	C	Elite	?	8-10	23	3000	N <sub>2</sub> house
Hahn, 2001 <sup>[58]</sup>	Cyclists	5F;6F	C	Elite	?	8-10	12	2650	N <sub>2</sub> house
Hinckson, 2005 <sup>[59]</sup>	Runners, triathletes	11M;11M	Xover	Subelite	?	8	25	2500-3500	N <sub>2</sub> tent
Hinckson, 2005 <sup>[60]</sup>	Runners	8M,2F	U	Subelite	?	10	24-30/30	2500-3500	N <sub>2</sub> tent
Martin, 2002 <sup>[61]</sup>	Cyclists	5F;6F	C	Elite	?	8-10	12	2650	N <sub>2</sub> house
Mattila, 1996 <sup>[62]</sup>	Cyclists	5M	U	Elite	Competitive	18	11	3000	N <sub>2</sub> house
Nummela, 2000 <sup>[25]</sup>	Runners	6M,2F;10M	C	Elite	?	16-17	17	2200	N <sub>2</sub> house
Roberts, 2003 <sup>[63]</sup>	Cyclists	14M,5F;14M,5F	Xover	Subelite	?	8-10	5-15	2650	N <sub>2</sub> house
Rusko, 1999 <sup>[49]</sup>	Skiers	9M,3F;8M,2F	?	?	?	12-16	25	2500	N <sub>2</sub> house
Saunders, 2004 <sup>[50]</sup>	Runners	10M;13M	C	Elite	?	9-12	19/25	2000-3100	N <sub>2</sub> house
Witkowski, 2001 <sup>[55]</sup>	Runners	8M,4F	U	Subelite	?	~18-24	28	2085/1200	-
Witkowski, 2001 <sup>[55]</sup>	Runners	8M,4F	U	Subelite	?	~18-24	28	2454/1200	-
Witkowski, 2001 <sup>[55]</sup>	Runners	8M,4F	U	Subelite	?	~18-24	28	2805/1200	-

**Artificial short continuous live-high train-low**

Basset, 2006 <sup>[64]</sup>	Skiers, skaters	7M,5F;7M,5F	Xover, B	Subelite	Off-season	3	6/19	3650	N <sub>2</sub> tent
Katayama, 2003 <sup>[65]</sup>	Runners	6M;6M	C	Subelite	?	1.5	9/19	4000	Chamber
Katayama, 2004 <sup>[66]</sup>	Runners	8M;7M	C	Subelite	Competitive	3	14	4000	N <sub>2</sub> tent
Rodríguez, 2000 <sup>[27]</sup>	Non-athletes	13M	U	Trained	?	1.5	9/19	4000-5500	Chamber
Rodríguez, 2003, 2006 <sup>[67,68]</sup>	Swimmers	3M,3F;4M,3F	C	Subelite	?	3-5	9	4000- 5500	Chamber
Rodríguez, 2003, 2006 <sup>[67,68]</sup>	Runners	2M,3F;3M,2F	C	Subelite	?	3-5	9	4000- 5500	Chamber

**Artificial brief intermittent live-high train-low**

Bonetti, 2006 <sup>[69]</sup>	Kayakers	10M;10M	Xover	Subelite	Competitive	0.5/1	15/19	3600-6000	Inhaler
Bonetti, 2007 <sup>[40]</sup>	Cyclists	18M;9M	C	Subelite	Competitive	0.5/1	15/19	3600-6000	Inhaler
Hamlin, 2006 <sup>[70]</sup>	Multisport athletes	5M,7F;8M,2F	C, B	Subelite	?	0.75/1.5	15/19	3600-6000	Inhaler
Hinckson, 2006 <sup>[71]</sup>	Rowers	2M,5F;1M,4F	C, B	Elite	?	0.9/1.5	15/19	3600-6000	Inhaler
Julian, 2004 <sup>[72]</sup>	Runners	7M;7M	C, B	Elite	Competitive	0.75/1.5	20/26	3600-6000	Inhaler
Wood, 2006 <sup>[9]</sup>	Hockey players	15M;14M	C, B	Subelite	Competitive	0.6/1	15/19	3600-6000	Inhaler

### Live-low train-high

Dufour, 2006 <sup>[73]</sup>	Runners	9M;9M	C	Subelite	Pre-season	0.2-0.33/0.33	12/40	3000	Inhaler
Hendriksen, 2003 <sup>[74]</sup>	Triathletes	12M;12M	Xover, ?	Subelite	Pre-season	2	10	2500	Chamber
Katayama, 1999 <sup>[75]</sup>	Non-athletes	7M;7M	C	Trained	?	0.5	10/12	4500	Chamber
Morton, 2005 <sup>[76]</sup>	Team sports	8M;8M	C	Trained	?	0.17/0.5	9/19	2750	N <sub>2</sub> house
Roels, 2005 <sup>[77]</sup>	Cyclists, triathletes	11M;11M	C, ?	Subelite	Pre-season	0.2-0.5/0.5	14/47	3000	Inhaler
Terrados, 1988 <sup>[78]</sup>	Cyclists	4M;4M	C	Subelite	?	2	20/20-26	2300	Chamber
Truijens, 2003 <sup>[79]</sup>	Swimmers	3M,5F;3M,5F	C, B	Subelite	?	0.21/0.5	15/33	2500	Inhaler
Vallier, 1996 <sup>[26]</sup>	Triathletes	3M,2F	U	Elite	?	1	9/19	4000	Chamber
Ventura, 2003 <sup>[80]</sup>	Cyclists	6M;5M,1F	C, ?	Subelite	Competitive	0.5	18/40	3200	Inhaler

<sup>a</sup>M, male; F, female; data separated by ";" are controlled trials with sample size in experimental and control groups.

<sup>b</sup>C, controlled trial; U, uncontrolled trial; Xover, crossover; B, blind; ?, blinding uncertain (assumed not blind).

<sup>c</sup>Numbers separate by "/" indicate sum of time in bouts of hypoxia and sum of recovery time per session.

<sup>d</sup>Numbers after "/" indicate intervention period, if longer than exposure period.

<sup>e</sup>Numbers separate by "/" indicate live-high and train-low altitudes

<sup>f</sup>Groups with and without iron supplementation.

C, controlled trial; U= uncontrolled trial; Xover, crossover; B, subjects blinded to treatment.

**Table 5.2.** Meta-analysis of effects on sea-level mean power output following adaptation to hypoxia experienced in studies with various protocols of natural and artificial altitude. Effects of mean and enhanced protocols are those predicted for controlled trials and maximal tests.

	Natural-Altitude Protocols		Artificial-Altitude Protocols			
	Live High, Train High	Live High, Train Low	Live High 8-18 h.d <sup>-1</sup> Continuous, Train Low	Live High 1.5-5 h.d <sup>-1</sup> Continuous, Train Low	Live High <1.5 h.d <sup>-1</sup> Intermittent, Train Low	Live Low, Train High 0.5-2 h.d <sup>-1</sup>
<b>Effect of Mean Protocol<sup>a</sup> (%); ±90%CL<sup>b</sup></b>						
Elite	1.6; ±2.7	4.0; ±3.7	0.6; ±2.0	-	0.2; ±1.8	-
Subelite	0.9; ±3.4	4.2; ±2.9	1.4; ±2.0	0.7; ±2.5	2.6; ±1.2	0.9; ±2.4
<b>Effect of Enhanced Protocol<sup>c</sup> (%); ±90%CL</b>						
Elite	5.2; ±4.1	4.3; ±4.1	4.0; ±5.5	-	1.2; ±2.5	-
Subelite	4.5; ±4.1	4.6; ±3.3	4.8; ±5.3	3.5; ±3.5	3.6; ±2.1	6.8; ±4.9
Study characteristics changed by +1 SD or -1 SD for enhanced protocol	+altitude -days exposure +test day	-altitude -test day	+altitude +hours hypoxia -days exposure	-altitude -test day	+exposure ratio -test day	-altitude -train intensity +days exposure +test day
<b>Study Characteristics (mean ± SD)</b>						
References	10	5	9	4	6	7
Study groups	13	9	10	5	5	7
Study-estimates	33	13	17	11	33	19
Subjects.estimate <sup>-1</sup>	16 ± 7	12 ± 6	17 ± 9	15 ± 5	20 ± 6	17 ± 5
Effective subjects.estimate <sup>-1</sup>	36 ± 22	41 ± 11	20 ± 9	15 ± 5	20 ± 6	17 ± 5
Elite athletes (%)	54	33	50	0	33	0
Controlled trials (%)	46	11	85	100	100	100
Blind trials (%)	0	0	0	20	67	14
Males (%)	84	61	80	72	81	90
Competitive phase (%)	31	33	10	20	67	14
Phase unknown (%)	54	56	90	60	0	43
Maximal tests (%)	78	100	85	100	71	92
Altitude level (m)	2030 ± 410	2400 ± 290	2890 ± 420	4530 ± 840	6000	2750 ± 310
Hours of hypoxia per day	24	-18-24	11 ± 3	-	-	-
Minutes of hypoxia per day	-	-	-	210 ± 84	40 ± 9	47 ± 48
Days of exposure	23 ± 6	27 ± 1	18 ± 7	9 ± 3	16 ± 2	14 ± 4
Total period of treatment (d)	23 ± 6	27 ± 1	19 ± 7	14 ± 5	20 ± 3	30 ± 13
Exposure/treatment ratio (%)	100	100	96 ± 7	76 ± 32	82 ± 8	55 ± 27
Training intensity (1-4)	-	-	-	-	-	2.3 ± 1.1
Post-exposure test day <sup>d</sup>	9.1 ×/÷ 1.9	5.4 ×/÷ 2.2	2.2 ×/÷ 2.7	4.4 ×/÷ 1.9	6.4 ×/÷ 2.3	2.8 ×/÷ 1.9
Duration of max. tests <sup>d</sup> (min)	6.9 ×/÷ 2.4	11 ×/÷ 1.3	5.6 ×/÷ 2.3	5.2 ×/÷ 3.0	6.1 ×/÷ 2.5	3.9 ×/÷ 4.9
<b>Effects of Study Characteristics (%); ±90%CL</b>						
Subelite - elite	0.7; ±3.8	0.3; ±2.2	0.8; ±3.2	-	2.4; ±2.8	-
Uncontrolled - controlled	3.3; ±3.6	-2.6; ±3.0	-1.6; ±3.4	-	-	-
Blind - not blind	-	-	-	-	-1.4; ±4.5	-
Female - male	-	-0.3; ±3.9	-	-	-	-
Competitive - unknown phase	0.5; ±3.8	-	-	-	-	-
Submaximal - maximal test	0.0; ±1.6	-	-3.3; ±2.4	-	-0.3; ±1.8	1.4; ±3.2
1 SD altitude level	1.2; ±1.6	-0.1; ±1.0	1.5; ±2.5	-2.3; ±2.5	-	-0.9; ±2.5
1 SD hours hypoxia	-	-	0.8; ±1.8	-	-	-
1 SD minutes hypoxia	-	-	-	-	0.4; ±2.3	-
1 SD days exposure	-1.8; ±1.7	-	-1.0; ±1.7	-	-	2.4; ±2.5
1 SD exposure/treatment ratio	-	-	-	-	0.6; ±1.2	-
1 SD training intensity	-	-	-	-	-	-1.2; ±2.5
1 SD post-exposure test day	0.5; ±0.7	-0.2; ±0.3	0.1; ±2.1	-0.5; ±0.8	-0.4; ±0.6	1.2; ±1.5
1 SD duration of max. test	3.0; ±2.5	-	-0.9; ±1.2	-0.3; ±0.6	0.6; ±1.3	-0.2; ±1.1
<b>Random Variation (%); ±90%CL or ×/÷90%CL factor</b>						
Between-study SD	2.7; ±2.3	1.3; ±1.3	1.0; ±1.9	2.2; ±3.5	-0.6; ±0.9	2.4; ±3.1
Standard error of measurement	2.4; ×/÷1.7	0.7; ×/÷2.2	2.2; ×/÷1.9	1.2; ×/÷1.9	3.2; ×/÷1.3	2.8; ×/÷1.5

Effects in cells shaded grey are unclear (>5% chance of enhancement and >5% chance of impairment); otherwise bold indicates ≥50% chance of enhancement, *italic* indicates ≥50% chance of impairment, and plain font indicates ≥50% chance of a trivial effect. These probabilistic outcomes are computed with reference to a smallest important change of 1%.

<sup>a</sup>Effects are the means predicted for controlled trials and maximal tests, but are otherwise evaluated at the mean values of the study characteristics for which effects are shown.

<sup>b</sup>90%CL: subtract and add this number to the observed effect to obtain the 90% confidence limits for the true (large-sample) effect.

<sup>c</sup>Effects are the predicted means in maximal tests adjusted to ±1 SD away from the mean for selected study characteristics shown.

**Table 5.3.** Meta-analysis of effects on sea-level maximum oxygen uptake following adaptation to hypoxia experienced in studies with various protocols of natural and artificial altitude. Effects of mean protocol are those predicted for controlled trials.

	Natural Altitude		Artificial Altitude			
	Live High, Train High	Live High, Train Low	Continuous Long Hypoxia (8-18 h.d <sup>-1</sup> ), Train Low	Continuous Brief Hypoxia (1.5-5 h.d <sup>-1</sup> ), Train Low	Intermittent Brief Hypoxia (<1.5 h.d <sup>-1</sup> ), Train Low	Live Low, Train High (0.5-2 h.d <sup>-1</sup> )
<b>Effect of Mean Protocol<sup>a</sup> (%); ±90%CL<sup>b</sup></b>						
Elite	<i>-1.5; ±2.0</i>	<b>6.4; ±11.2</b>	-0.5; ±1.4	-	<b>0.1; ±2.8</b>	-
Subelite	<b>4.3; ±2.6</b>	<b>6.4; ±9.4</b>		<b>-1.1; ±3.5</b>		<b>1.1; ±2.0</b>
<b>Study Characteristics (mean ± SD)<sup>c</sup></b>						
References	12	5	5	4	3	8
Study groups	15	9	6	5	3	8
Study-estimates	20	10	7	6	5	10
Subjects per estimate	15 ± 7	12 ± 6	20 ± 10	15 ± 5	19 ± 5	16 ± 5
Effective subjects per estimate <sup>d</sup>	33 ± 19	41 ± 11	20 ± 10	15 ± 5	19 ± 5	16 ± 5
Elite athletes (%)	57	33	33	0	33	0
Controlled trials (%)	43	11	100	100	100	100
Blind trials (%)	0	0	0	20	33	13
Males (%)	87	61	75	72	100	91
Competitive phase (%)	29	33	0	20	100	13
Phase unknown (%)	57	56	100	60	0	50
Altitude level (m)	1990 ± 400	2400 ± 290	2680 ± 160	4530 ± 880	6000	2970 ± 680
Hours of hypoxia per day	-	-	10 ± 2	-	-	-
Minutes of hypoxia per day	-	-	-	210 ± 90	35 ± 8	45 ± 46
Days of exposure	23 ± 6	27 ± 1	18 ± 6	9 ± 3	17 ± 3	14 ± 4
Total period of treatment (d)	23 ± 6	27 ± 1	19 ± 7	14 ± 5	21 ± 4	28 ± 14
Exposure/treatment ratio (%)	100	100	96 ± 9	76 ± 33	78 ± 1	59 ± 27
Training intensity (1-4)	-	-	-	-	-	2.1 ± 1.1
Post-exposure test day <sup>e</sup>	8.0 ×/÷ 1.8	4.8 ×/÷ 2.1	1.2 ×/÷ 2.3	4.4 ×/÷ 2.0	4.7 ×/÷ 2.5	2.9 ×/÷ 2.2
<b>Effects of Study Characteristics (%); ±90%CL</b>						
Uncontrolled - controlled	0.3; ±2.4	<b>-2.7; ±9.3</b>	-	-	-	-
Competitive - unknown phase	<b>1.3; ±2.7</b>	-	-	-	-	-
Subelite - elite	<b>5.5; ±2.4</b>	<b>-0.0; ±5.2</b>	-	-	-	-
1 SD altitude level	0.3; ±1.2	-	-	-	-	<b>0.0; ±2.3</b>
1 SD hours exposure	-	-	<b>2.5; ±1.9</b>	-	-	-
1 SD days exposure	0.5; ±1.3	-	-0.9; ±1.7	<b>1.1; ±4.3</b>	-	<b>1.0; ±2.2</b>
1 SD training intensity	-	-	-	-	-	<i>-1.2; ±2.1</i>
1 SD post-exposure test day	<b>1.0; ±1.0</b>	<b>1.1; ±2.8</b>	-	-	<b>-0.5; ±3.3</b>	-
<b>Random Variation (%); ±90%CL or ×/÷90%CL factor</b>						
Between-study SD	1.8; ±2.4	3.8; ×/÷1.7	1.7; ×/÷1.9	3.3; ×/÷2.5	2.6; ×/÷2.2	2.1; ±2.8
Standard error of measurement	2.9; ×/÷1.8	<sup>e</sup>	<sup>e</sup>	<sup>e</sup>	<sup>e</sup>	2.5; ×/÷2.6

Effects in cells shaded grey are unclear (>5% chance of increase and >5% chance of decrease); otherwise bold indicates ≥50% chance of increase, *italic* indicates ≥50% chance of decrease, and plain font indicates ≥50% chance of a trivial effect. These probabilistic outcomes are computed with reference to a smallest important change of 1%.

<sup>a</sup>Effects are the means predicted for controlled trials but otherwise evaluated at the mean values of the study characteristics for which effects are shown.

<sup>b</sup>90%CL: subtract and add this number to the observed effect to obtain the 90% confidence limits for the true (large-sample) effect.

<sup>c</sup>SD shown as ×/÷ factor derived from log-transformed times.

<sup>d</sup>Derived by adjusting all sample sizes to those of controlled trials with equal numbers in control and experimental groups.

<sup>e</sup>Insufficient within-study clusters to estimate error of measurement; between-study SD includes within-study sampling variation.

**Table 5.4.** Meta-analysis of effects on sea-level hemoglobin or red-cell mass (Hb mass), exercise economy, Hb concentration, and peak lactate in an exercise test following adaptation to hypoxia experienced in studies with various protocols of natural and artificial altitude.

	Hb Mass, Where Measured <sup>a</sup>	Economy, Where Measured <sup>b</sup>	Hb Concentration		Peak Lactate	
			Live High, Train High	Intermittent Brief Hypoxia, Train Low	Live High, Train High	Intermittent Brief Hypoxia, Train Low
<b>Effect of Mean Protocol<sup>c</sup> (%); ±90%CL<sup>d</sup></b>	<b>1.3; ±2.4</b>	0.4; ±1.3	<b>4.8; ±2.7</b>	<b>2.3; ±1.2</b>	<b>0.7; ±5.7</b>	-3.5; ±4.7
<b>Study Characteristics (mean ± SD)<sup>c</sup></b>						
References	12	14	5	4	5	5
Study groups	14	15	7	4	7	7
Study-estimates	18	19	8	8	9	14
Subjects.estimate <sup>-1</sup>	15 ± 7	19 ± 5	16 ± 9	22 ± 5	19 ± 8	24 ± 3
Effective subjects.estimate <sup>-1</sup>	25 ± 9	31 ± 27	32 ± 11	22 ± 5	35 ± 23	24 ± 3
Elite athletes (%)	46	33	57	20	43	0
Controlled trials (%)	62	80	43	100	57	100
Blind trials (%)	0	7	0	60	0	50
Males (%)	74	91	83	92	82	90
Competitive phase (%)	15	53	29	80	29	75
Phase unknown (%)	62	40	43	0	43	0
Altitude level (m)	2540 ± 970	3410 ± 1460	1900 ± 280	6000	1990 ± 320	6000
Minutes of hypoxia per day	-	-	-	37 ± 7	-	35 ± 6
Days of exposure	21 ± 7	20 ± 6	24 ± 5	16 ± 2	22 ± 6	15
Total period of treatment (d)	21 ± 7	24 ± 6	24 ± 5	20 ± 4	22 ± 6	18 ± 2
Exposure/treatment ratio (%)	100	86 ± 22	100	83 ± 9	100	84 ± 9
Post-exposure test day <sup>e</sup>	3.9 ×/÷ 2.6	3.3 ×/÷ 2.8	9.1 ×/÷ 2.1	4.3 ×/÷ 2.3	8.3 ×/÷ 2.2	5.9 ×/÷ 2.3
<b>Effects of Study Characteristics (%); ±90%CL</b>						
1 SD altitude level	<b>1.5; ±2.6</b>	0.6; ±1.6	<b>-1.8; ±4.0</b>	-	<i>-12.4; ±7.0</i>	-
1 SD exposure days	<b>2.7; ±2.7</b>	-0.8 ±1.6	-	-	<b>1.4; ±7.7</b>	-
1 SD post-exposure test day	-0.9; ±1.0	<b>0.1; ±1.4</b>	<i>-3.3; ±3.9</i>	0.6; ±1.3	<i>-10.5; ±7.7</i>	-0.6; ±2.1
<b>Random variation (%); ±90%CL or ×/÷90%CL factor</b>						
Between-study SD	4.6; ±2.2	-1.0; ±2.5	3.6; ×/÷1.8	1.7; ×/÷1.7	8.7; ×/÷1.7	3.6; ±4.8
Standard error of measurement	2.1; ×/÷1.9	6.0; ×/÷1.7	<sup>f</sup>	<sup>f</sup>	<sup>f</sup>	7.3; ×/÷1.5

Effects in cells shaded grey are unclear (>5% chance of increase and >5% chance of decrease); otherwise bold indicates ≥50% chance of increase, *italic* indicates ≥50% chance of decrease, and plain font indicates ≥50% chance of a trivial effect. These probabilistic outcomes are computed with reference to a smallest important change of 1% for Hb mass, 1% for economy, and 0.20 of baseline between-subject SD for Hb concentration and peak lactate.

<sup>a</sup>No. of estimates: LHTH, 10; LHTL, 3; artificial long continuous LHTL, 3; artificial brief continuous LHTL, 2.

<sup>b</sup>No. of estimates: LHTH, 4; LHTL, 3; artificial long continuous LHTL, 3; artificial brief continuous LHTL, 3; artificial brief intermittent LHTL, 5; LLTH, 1.

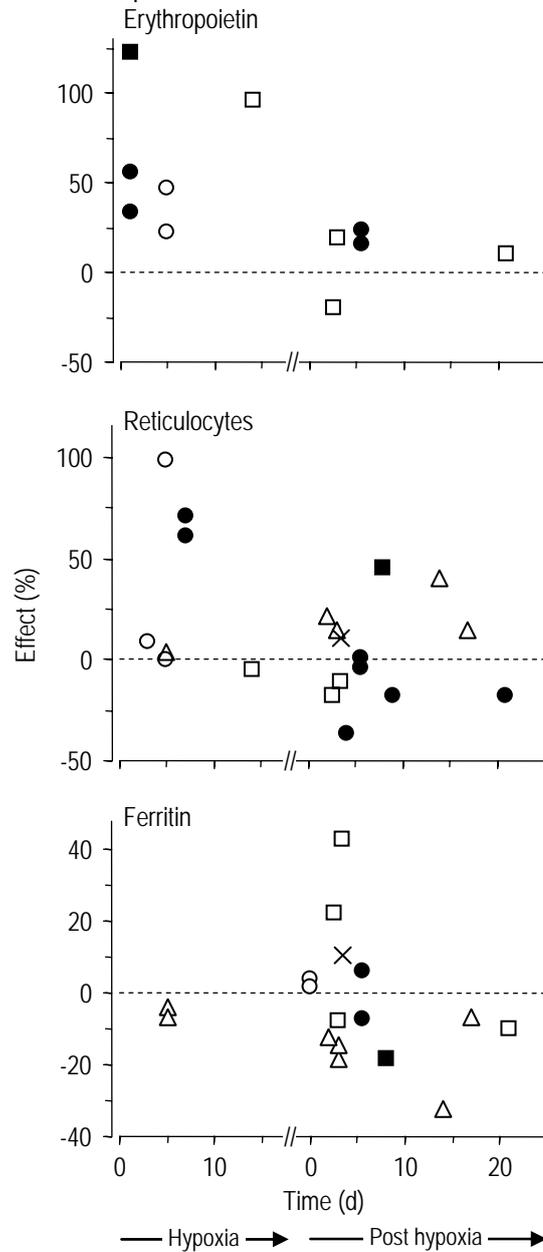
<sup>c</sup>Effects are the predicted means evaluated at the mean values of the study characteristics for which effects are shown.

<sup>d</sup>90%CL: subtract and add this number to the observed effect to obtain the 90% confidence limits for the true (large-sample) effect.

<sup>e</sup>SD shown as ×/÷ factor derived from log-transformed times.

<sup>f</sup>Insufficient within-study clusters to estimate error of measurement; between-study SD includes within-study sampling variation.

**Fig. 5.1.** Individual study-estimates of effects on erythropoietin, reticulocytes and ferritin sampled in blood during and following exposure to hypoxia with the various protocols of natural and artificial altitude.



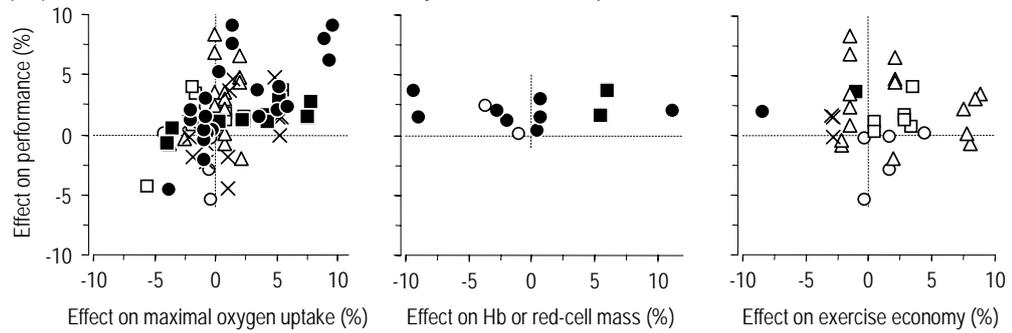
**Natural altitude:**

- Live high, train high
- Live high, train low

**Artificial altitude:**

- Live high 8-18 h.d<sup>-1</sup> continuous, train low
- Live high 1.5-5 h.d<sup>-1</sup> continuous, train low
- △ Live high <1.5 h.d<sup>-1</sup> intermittent, train low
- × Live low, train high 0.5-2 h.d<sup>-1</sup>

**Fig. 5.2.** Individual study-estimates of effects on performance plotted against maximal oxygen uptake, hemoglobin (Hb) or red-cell mass, and exercise economy with the various protocols of natural and artificial altitude.



**Natural altitude:**

- Live high, train high
- Live high, train low

**Artificial altitude:**

- Live high 8-18 h.d<sup>-1</sup> continuous, train low
- Live high 1.5-5 h.d<sup>-1</sup> continuous, train low
- △ Live high <1.5 h.d<sup>-1</sup> intermittent, train low
- × Live low, train high 0.5-2 h.d<sup>-1</sup>

## CHAPTER 6

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### General Summary

My PhD research projects addressed a number of questions relevant to the application and understanding of simulated altitude training as a method of enhancing sea-level exercise performance.

The initial focus of my PhD was to use flat-water kayakers for my experimental studies. To determine the magnitude of the smallest worthwhile improvement in competitive performance that matters for kayakers it is necessary to establish the kayakers typical variation in competitive performance. Therefore, the first study involved the investigation of variation in elite flat-water canoeing performance using official race times. Data were analysed from World Cup, World Championships and Olympic Games regattas held between 2003-2007. Variability in performance was greater for athletes in the “B” finals compared to those in the “A” finals, illustrating that the higher ranked athletes demonstrate less variability in performance. The comparisons of the within-athlete variabilities of men vs women and canoeing vs kayaking were unclear, but the observed differences with this sample of athletes and races were trivial. For men within athlete variation was similar in canoeing and kayaking events and displayed a trend towards reduction as the duration of the event decreased. There may be a similar underlying trend in women, but we found a greater variation in the shortest event (200-m) which could be due to less competitive experience or depth of competition in this event. Overall, the variability in competitive performance for flat-water canoeists is ~1%. Therefore, the smallest worthwhile change in performance (approximately half of the within athlete variation between competitions) for elite flat-water canoeists are ~0.5%.

There has been limited research investigating the effects of intermittent hypoxic exposure on sea level performance. Additionally only two studies had investigated the effect on anaerobic performance and no study had used flat-water kayakers. Therefore, the focus of the second study was to examine the effect of acute intermittent hypoxia on aerobic and anaerobic exercise performance. Utilising a crossover design, 10 sub-elite male flat-water kayakers, were assigned to altitude or control groups for 3 weeks (5 days per week) of intermittent hypoxic exposure. Each day’s exposure consisted of

breathing hypoxic air for a duration of 5 mins followed by 5 minutes of ambient air, for a period of 60 minutes. Following a 6-week washout the groups were reversed. Relative to the control group, at 3 days post-treatment the hypoxic group demonstrated improvements in peak power 6.8%, mean repeat sprint power 8.3% and hemoglobin concentration 3.6%. These improvements were associated with small although unclear changes in lactate threshold and mean 500-m power. Effects for all other measures including those representing anaerobic power were unclear. At 10 days post-treatment, although statistically unclear, there was a possible elevation in peak power and mean sprint power in the hypoxic group. Effects for all other measures were also unclear.

I concluded that the standard protocol of acute intermittent hypoxia should enhance performance in flat-water kayak racing. It is very likely that the performance in other sports requiring a large proportion of aerobic metabolism would also achieve similar benefits, effects on anaerobic power are unclear and require further investigation. The mechanisms mediating these changes also were unclear, although there with some indirect evidence of erythropoiesis.

The results of my first experimental study demonstrated that clear improvements in endurance performance can be observed following adaptation to the standard protocol of acute intermittent hypoxia. However due to the small sample size and relatively large error of measurement for our mechanism measures, we were unable to draw any clear conclusions as to what was mediating our performance changes. Additionally there was reasonable evidence to suggest that alternative mechanisms such as inflammatory responses may play a role in the adaptation process following intermittent hypoxia. Furthermore I believed that the performance effect could possibly be enhanced by altering the hypoxic and normoxic interval durations. In order to address these issues we needed a larger sample size, greater reliability in our measures and a new protocol to experiment with. As previously rationalised cyclists were chosen instead of kayakers to improve precision of estimate and sample size. Therefore the focus of Study 3 was to investigate the effect of altering the normoxic and hypoxic intervals and to identify whether haematological, hormonal and inflammatory response is altered.

Utilising a controlled design, 18 male competitive cyclists and triathletes were randomized to one of two intermittent-hypoxia groups, for 3 weeks of acute intermittent hypoxic exposure. Nine similar athletes represented a control group. The same hypoxic device and intervention period as the previous study was employed. Athletes in the hypoxia groups were exposed to 60 min per day of intermittent hypoxia consisting of alternating intervals of hypoxia and normoxia lasting either 3 or 5 min. In all post test occasions there were no clear differences between effects of the two hypoxic treatments on performance or various measures of oxygen transport, haematology and inflammation. However when the hypoxic groups were combined and compared against the control, there were clear improvements in peak power (4.7%), Lactate profile power 4.4% and heart rate profile power (6.5%) at 3-d post-intervention. Unlike the previous study there were no improvements in repeat sprint power, in fact a small although unclear impairment was observed. Consistent with the previous study effects for performance in the second series of post tests (14 days compared to 12 in Study 3) were no longer clear. Reinforcing that adaptation to this type of hypoxic intervention is temporary.

While there was indirect evidence of a small erythropoietic effect as demonstrated by a substantial increases in haemoglobin concentration and substantial reductions in ferritin post-intervention, there were no substantial changes in hormonal or inflammatory responses. I conclude that the relative benefit of 3- vs 5-min exposure intervals remains unclear. However, consistent with my previous study acutely intermittent hypoxia produced substantial enhancement in endurance related performance. The performance enhancement may be partially mediated by changes via oxygen transport, the effect of inflammatory responses or alternative mechanisms on the adaptation process is unclear.

The final study in this thesis was the first literature review to employ a meta-analytic approach to compare the effects on sea level performance and related physiology following adaptation to various protocols of natural or artificial altitude exposure: 51 qualifying studies (including the two experimental studies from this) thesis were compared using the meta-analytic mixed model. The analysis revealed that in sub-elite athletes enhancements in endurance power output were very likely utilising either live high train high or intermittent brief hypoxic exposure (as investigated in this thesis).

Smaller enhancements were likely for natural live high train low and artificial live high train low with long continuous exposures. For elites only natural live high train high and live high train low produced substantial enhancements in performance. Additionally each protocol was able to show further enhancements in endurance power by manipulation of certain study characteristics. Changes in maximum oxygen uptake were limited to the natural protocols and only for live high train low in elite athletes. Our results only provided limited evidence for erythropoiesis as the mediator of the changes in VO<sub>2</sub> max, and were inconclusive for the role of erythropoiesis as a mediator of the observed performance enhancements. Effects for other mechanism variables including exercise economy were unclear.

I conclude that there is now good evidence that sub-elite athletes experience endurance performance enhancements with adaptation to natural altitude exposure and to brief intermittent and long continuous protocols of artificial altitude exposure. Effects for elites are currently limited to the natural protocols. Modification of the protocols presents the possibility of further enhancements. The enhancements could be mediated by VO<sub>2</sub>max, although other mechanisms may be involved.

## **(De)Limitations**

There are several (de)limitations to the findings of this thesis, specifically:

- The small improvement observed in 500-m performance in flat-water kayakers was performed on a kayak ergometer. Although the reliability of this test is reasonable the validity of this test with regard to on-water kayaking performance remains to be investigated.
- Sub-elite kayakers and cyclists were used for both experimental studies. It is clear from our meta-analysis that performance enhancements following adaptation to various protocols of natural and artificial altitude are less for elites when compared to sub-elites. Therefore elite athletes can not expect the same improvement in performance as was experienced by the sub-elite athletes recruited for the two experimental studies.
- Meta-analysis cannot address the question of individual responses to treatments until researchers provide complete inferential information about experimental and control groups in the form of confidence limits, exact p values, or standard deviations of change scores.
- The estimates of smallest worthwhile changes in performance for elite flat-water canoeists are not thresholds for changes in the laboratory tests we used, because the relationship between change in performance in these laboratory tests and change in performance time in the canoeing events is unknown.
- The high error of measurement in both experimental studies for measures of oxygen uptake using the meta-max system made any potential changes in oxygen uptake difficult to quantify.
- Endurance performance in both experimental studies was measured by peak power in an incremental test, rather than performance time trials. Although peak power is highly correlated with endurance performance, the changes in peak power observed may not cause an equal change in real life endurance performance.

## **Future research**

In this thesis I have examined the influence of hypoxic exposure on sea level aerobic and anaerobic exercise performance. While I have addressed a number of questions specific to this area, further research addressing the following issues is warranted:

- Determine the relationships between change in power in kayak ergometry tests and change in actual on-water racing performance.
- Further investigate the response of hormonal and molecular biology markers to hypoxic exposure, in particular: Growth hormone, testosterone, cortisol, vascular endothelial growth factor, hypoxia-inducible factor 1, mitochondrial transcription factor A, and carbonic anhydrase 3. Researchers investigating these responses should design their studies appropriately to allow timing of blood or saliva samples immediately following each exposure session and at several time points throughout the day.
- Although the studies in this thesis were equivocal regarding anaerobic performance following adaptation to intermittent hypoxia, research in this area is still very limited and requires further investigation.
- The “enhanced protocols” that were derived in the meta-analysis may provide researchers and practitioners an ideal starting point for investigation and modification of contemporary protocols of simulated and natural altitude training.
- Wherever possible “elite” athletes should be used for future studies since our meta-analysis has clearly shown that performance adaptations between elites and sub-elite athletes are of a differing magnitude.

## Conclusions

- The smallest worthwhile enhancements are ~0.5% for elite flat-water canoeist
- Intermittent hypoxic exposure using the standard protocol of 5-min hypoxia, 5-min normoxia, produced worthwhile gains in endurance performance for sub-elite sprint kayakers and road cyclists. The effect on anaerobic performance for the kayakers was unclear and negative for the cyclists
- The performance effects following adaptation to intermittent hypoxia are most noticeable immediately following the hypoxic exposure period. Therefore coaches and athletes need to program the conclusion of any hypoxic intervention as close as possible to their competition.
- Altering the hypoxic and normoxic interval duration in Study 3 had little effect on exercise performance, haematology or inflammatory markers. Therefore, utilising periods of either 3-min hypoxia, 3-min normoxia or 5-min hypoxia, 5-min normoxia are sufficient to elicit substantial improvements in endurance performance
- While some changes in haematology were found to result from both experimental studies of intermittent hypoxia, the true effect of this mechanism on exercise performance remains unclear. Additionally the roles of other mechanisms such as exercise economy, buffering and inflammatory responses require further investigation.
- As meta-analysed, clear enhancements in endurance power output can be observed in sub-elite athletes following adaptation to various protocols of either natural or artificial altitude exposure. In elite athletes smaller enhancements in endurance power output are observed and limited to the natural protocols
- As meta-analysed, there is limited evidence supporting the role of erythropoiesis as a mediator of changes in VO<sub>2</sub> max. The role of erythropoiesis as a mediator of changes in endurance performance is unclear. Effects for other mechanism variables including exercise economy were unclear.

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## Appendix A

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### Literature Review from PhD proposal

#### Introduction

This chapter will begin with a review of supramaximal exercise metabolism and the related physiological processes that produce fatigue and limit performance. The physiological rationale and methodologies of hypoxic exposure will then be presented, followed by a review of the effects of hypoxic exposure on exercise performance.

#### Sprint kayak performance

Before considering the influence of hypoxic exposure on kayak performance, it is important to consider the physiological requirements of a supramaximal task, such as the K1 500 m. Similar in duration to 800 m track running and 200 m freestyle swimming, the K1 500 is completed in 96-105 s for elite men and 109 -115 s for elite women (International Canoe Federation statistics, 2004). Research has indicated that this event requires approximately a 65% contribution from the aerobic energy system and 35% from the anaerobic energy systems (Bishop et al., 2001). Thus, the K1 500, places a major demand on the aerobic system, although the anaerobic systems (particularly the glycolytic energy system) also play a significant role in the provision of ATP for this event.

From the onset of supramaximal exercise, there is a metabolic demand for oxygen, which is greater than the cardiorespiratory system can supply. This aerobic energy shortfall, apparent in the difference between the amount of oxygen theoretically required to produce the required power output and that which is actually consumed during high energy exercise, is met by anaerobic energy supply systems (Gutin et al., 1976). This shortfall can be expressed in oxygen equivalents and is known as the accumulated oxygen deficit (AOD) (Gastin, 1995).

The rapid synthesis of ATP (both aerobic and anaerobic) results in a dramatic fall in intracellular pH. Intracellular H<sup>+</sup> accumulation has been proposed to reduce power output and to produce fatigue via a number of mechanisms. Accumulation of H<sup>+</sup> may inhibit the enzyme phosphofructokinase (PFK), therefore slowing glycolysis and reducing energy producing capacity (Trivedi and Danforth, 1966). However, the

importance of this mechanism may be questionable, since the inhibition of PFK may be overcome by increased adenosine monophosphate (AMP) or adenosine diphosphate (ADP) levels during intense exercise (Sahlin, 1986). Accumulation of  $H^+$  may also affect the contractile apparatus. Katz (1970) has suggested that increased concentrations of  $H^+$  could compete with  $Ca^{++}$  for the binding sites on the actomyosin. This results in a decreased number of active cross bridges, causing a decrease in contractile force and decrease in performance. Thus, an increase in  $H^+$  concentration may impair supramaximal performance via inhibition of anaerobic glycolysis and / or interference with muscle contractile processes.

While increased  $H^+$  concentrations contribute to fatigue and loss of power output during supramaximal exercise, it is also known that disturbances in muscle pH become greater as the intensity of exercise increases (Medbo & Tabata, 1993). Therefore, the more intensely an athlete starts an event, the greater the disturbances in muscle pH are likely to be during the latter stages of the event. However, recent research by Bishop et al., (2001) has demonstrated that performance is superior during simulated K1 500 racing if an all-out start strategy rather than an even start strategy is utilised, despite increased acidity with the all-out strategy. Not surprisingly this is the most common racing strategy adopted by elite K1 500 athletes. Therefore it should be expected that K1 500 athletes will adopt a strategy that will invoke large disturbances in muscle pH. Thus, their ability to buffer acidity in addition to their aerobic capacity could be a significant factor influencing their competitive performance. Hypoxic exposure is a training aid which has been shown to improve both aerobic (Levine et al., 2001) and anaerobic capacity (Roberts et al., 2003). Since these capacities are vital for success in K1 500 racing it could be a useful training strategy for kayak athletes to adopt

### **Hypoxic exposure**

Hypoxic exposure or as it is more commonly known “altitude training”, relates to any training or non training method whereby the athlete is exposed to an oxygen deficient environment. There are two ways that athletes are exposed to hypoxia: hypobaric and normobaric. In hypobaric hypoxia a change in barometric pressure alters the partial pressure of oxygen. The most common environment employed here is natural altitude. Upon ascent to a higher altitude barometric pressure is reduced. The change in barometric pressure does not affect the mixture of gases in the air. However,

the pressure that oxygen molecules exert at various altitudes is directly affected. This causes a reduction in the partial pressure of oxygen as the altitude increases. For example at sea level the partial pressure of oxygen is 159 mmHg but decreases to 125 mmHg at 2000 m and 48 mmHg at 5000 m (Wilmore and Costill, 2003). Hypobaric chambers can also be utilised to alter barometric pressure and create hypoxia.

Normobaric hypoxia refers to a hypoxic environment created without an adjustment in barometric pressure. The most common method employed here is by adding nitrogen to the inspired air. This has the effect of diluting the oxygen content and reducing the partial pressure of oxygen, creating a hypoxic environment at sea level. This method is commonly used in altitude houses and tents. For example altitude houses in Finland use a ventilation system to pull in ambient air which is composed of 20.93% O<sub>2</sub> and 79.0% N<sub>2</sub>. A gas comprised of 100% N<sub>2</sub> is simultaneously introduced into the ventilation system, which results in an internal gas composition of 15.3% O<sub>2</sub> and 84.7% N<sub>2</sub>. This normobaric hypoxic environment simulates an altitude of 2500m/8200ft [partial pressure of inspired oxygen (PIO<sub>2</sub>) = 116 mmHg (Wilber 2001)]. The same method can be employed intermittently with a face mask connected to a generator pumping nitrogen enriched air. Alternatively, normobaric hypoxia can also be created using a rebreathing device. The devices incorporate a chemical absorbent to prevent accumulation of expired carbon dioxide, which, by stimulating ventilation, would otherwise prevent development of hypoxia (Wood et al., 2003). These devices are also used intermittently.

### **Physiological adaptations to hypoxia**

Both normobaric and hypobaric hypoxia have been shown to produce favourable changes in oxygen transport and utilisation mechanisms. In addition other oxygen independent mechanisms have also been proposed to improve as a result of normobaric and hypobaric hypoxia.

### **Oxygen transport**

The most commonly known adaptation to hypoxia is an increase in the total number of red blood cells, and the associated mass of haemoglobin. Research had indicated that 3 weeks of hypobaric hypoxia can increase haemoglobin concentration by 1-4 %

(Berglund 1992). This response is largely mediated by an erythrocyte-stimulating hormone, erythropoietin, that is produced primarily in the proximal tubule cells of the kidneys and to some extent in the liver (Erslev 1987, 1991). The availability of oxygen in the kidneys and liver is the main regulator for the production of EPO. Hypoxia increases the production of the hormone, whereas hyperoxia has the opposite effect. The activation of EPO receptors in the bone marrow enhances the mitosis and differentiation of specific erythroid progenitor cells, resulting in the production of new red blood cells (Krantz 1991). Red blood cells carry oxygen via hemoglobin from lungs to muscles (Baker and Hopkins 1998). Therefore, if you increase the amount of red cells your blood can carry more oxygen, which should enhance  $\text{VO}_2$  max and exercise performance.

A role for an increase in oxygen delivery following hypoxic exposure, mediated by a more rapid or marked vasodilatation in active muscle has been proposed by Wood et al. (2003). The response may be mediated by the “stress of hypoxia” which may enable the arterioles in active muscles to dilate more during exercise, thereby delivering more blood to the muscles.

### **Oxygen utilisation**

Oxygen utilisation via a number of mechanisms, has been demonstrated to improve in response to hypoxia. Capillary supply has been shown to increase in response to hypoxia (Desplanches et al. 1993). This modification in local circulation has been theorised to reduce the distance for oxygen diffusion between the blood and tissues (McCardle et al. 1998), which would enhance oxygen utilisation.

Muscle biopsies from humans living at high altitude indicate an increase in myoglobin by as much as 16% after acclimatisation to hypoxia. This was complemented by an increase in the number of mitochondria and the concentration of enzymes required for energy transfer (Macdougall et al., 1991; Renafajre, 1962) These adaptations would increase the available storage of oxygen in specific muscles. Finally, an increase in concentration of 2,3 - DPG in red blood cells occurs during long term acclimatisation to hypobaric hypoxia (Eaton et al 1969). 2,3 - DPG has the ability to combine reversibly with haemoglobin and thus alter its structure to release oxygen McCardle et al. (1998).

Therefore a greater concentration of 2,3 - DPG would allow more oxygen to be released from haemoglobin.

### **Oxygen independent**

Oxygen independent mechanisms have been shown to change as a result of hypoxia. Research has demonstrated an increase in the maximal accumulated oxygen deficit (MAOD) in response to normobaric and hypobaric hypoxia (Mizuno et al., 1990; Ogita and Tabata 1999; Roberts et al 2003). MAOD is an indirect measurement of anaerobic capacity; an increase in MAOD has been demonstrated to parallel improvements in supramaximal exercise performance (Roberts et al., 2003). Other research has indicated that increased muscle buffering (Gore et al 2001) and changes in acid base and lactate metabolism (Numela and Rusko 2000) occur in response to hypobaric hypoxic. It is possible that an increase in MAOD may be mediated by either of these mechanisms.

It is also possible that an enhanced rate of phosphocreatine resynthesis can occur in response to hypoxia. Wood et al. (2003) found an improvement in repeat sprint ability following 15 days of intermittent hypoxia. Greater rates of PCr resynthesis would provide a plausible explanation for this finding. Further research needs to be conducted with direct measurements of PCr resynthesis to establish this relationship.

The section has outlined the physiological changes which can occur in response to both normobaric and hypobaric hypoxia. The increasing popularity of this training method has led to a vast amount of scientific research into both the physiological and performance benefits of hypoxic exposure. However despite the large volume of research, it is yet to be conclusively established how effective hypoxic exposure is at improving both physiological capacities and exercise performance. Following is a review of this literature.

### **Live high / train high (hypobaric)**

Traditionally athletes have both lived and trained at altitude in an attempt to improve their sea level performance. This was based largely upon the assumption that an increase in red blood cells and other favourable hematological adaptations, that occur in response to hypobaric hypoxia would translate into improved sea level performance.

However despite the potential for enhanced hematological indices the majority of research studies conducted to date have proven to be inconclusive in their findings.

Since 1967, 17 studies have investigated the effect of hypobaric hypoxia on endurance performance. 8 studies showed a positive effect of 0.5 – 3% (Levine and Stray - Gundersen 92 & 97; Martino et al., 1995; Mizuno et al., 1990; Karvonen et al., 1986; Adams et al., 1975, Dill and Adams 1971, Daniels and Oldridge 1970). However 5 of the studies showed a definite negative effect of 0.5 – 7.5% (Gore et al. 1997a; Rusko et al, 1996; Jensen et al. 1993; Faulkner et al. 1967; Saltin 1967), and 4 showed a minimal effect  $\pm$  0.5% (Gore et al. 1997b; Telford et al. 1996; Rahkila and Rusko 1982; Buskirk et al 1967).

Analysis of these studies would suggest that there is a tendency towards a benefit from altitude training, but there is a large variation in the outcome between studies. Some of the variability could be a result of different altitudes (1300 to 4000 meters) and different periods of training (12 to 63 days). Six of these studies had no control group, therefore changes in performance could also have been the result of factors unrelated to living and training at altitude.

There is another plausible explanation for the inconclusive results. It is an established fact that athletes cannot train with the same intensity at altitude compared to sea level. For example VO<sub>2</sub> max decrease by 2% for every 300 m above 1500m (McCardle, Katch and Katch., 1998 ). It has been proposed that the lack of oxygen at altitude results in detraining through a reduction in training intensity (Hopkins and Baker 1998). Accordingly it has been theorised that this reduction in fitness may offset any positive physiological benefits gained from altitude exposure (Levine 2002). Therefore based upon the available evidence it would appear that the live high train high method is a questionable method to adopt.

### **Live high train low (hypobaric)**

The lack of consistent results obtained with live high train high training proved to be the catalyst for the live high train low approach. The ‘live high – train low’ model was initially investigated by Levine et al., (1991). At this time it was well established that living at altitude produced favorable gains in red blood cells and other hematological

indices. These authors theorised that athletes could improve sea level endurance performance by living high (2000 to 2700m) while simultaneously training at low elevation (=1000m). Simultaneous training at low altitude allows athletes to train at exercise intensities that are similar to sea level, thereby maintaining adequate training stimulus. It was proposed that the combination of adequate training intensity / recovery combined with hematological improvements that result from 'high-low' altitude training lead to the enhancement of sea level maximal oxygen uptake (VO<sub>2</sub>max) and endurance performance (Levine et al., 1991). This is indeed what occurred in Levine's initial investigation. Athletes experienced a 3.1% increase in VO<sub>2</sub> max and a 2.3% improvement in 5 km running performance (relative to live high train high or control?)

Since this initial investigation by Levine et al., (1991) 8 studies have investigated the effect of high / low hypobaric hypoxia on endurance performance and physiological measures mediating changes in performance. The altitude range in these studies for living was consistent. 6 studies used 2500 m and the remaining 2 used 2000 m (Dehnert et al. 2002) and a variety of ranges 17850 – 2850 m (Witkowski et al., 2001). The altitude used for high intensity training was below 1500 m with a far greater range of altitude utilised for low intensity training 1000 – 3000 m. All of these studies found a positive enhancement in endurance performance ranging from 1 - 4%. In the only study to have addressed supramaximal performance, Stray-Gundersen and Levine (1994) also found that 3 minute running speed was 2.7 % greater following live high train low.

Not surprisingly the studies which did assess physiological measures mediating changes in performance, found improvements of 4% in VO<sub>2</sub> max relative to a control group (Levine et al., 1992; Levine et al 1997). Increases in Red Cell Volume of 1 - 5% relative to a control group were also observed (Levine et al., 1992; Levine et al 1997; Stray-Gundersen and Levine 1997). Hemoglobin was also found to have increased by 9% in Stray-Gundersen and Levine (1997).

Analysis of these studies would suggest that athletes can live and perform low intensity training in a range of 2000 – 3000 m, with high intensity training performed below >1500 m and gain a performance enhancement in the order of 2-3 %. This performance improvement would appear to be mediated by an increase in red cell volume (and possibly other hematalogical changes) and as a direct consequence improve VO<sub>2</sub> max.

**Live high train low (normobaric)**

The success of the High / low model prompted research into the use of normobaric hypoxia as an aid to enhance sea level performance. This idea is very appealing since the athlete does not have to leave their normal training and living environment to receive a hypoxic stimulus. Studies which have investigated live high and train low in this manner have generally used nitrogen addition to create the hypoxic environment. Therefore instead of going to altitude the athlete remains at sea level in either a nitrogen house or tent.

To date 5 studies have investigated the influence of high / low normobaric hypoxia on endurance and supramaximal exercise performance. All of these studies with the exception of Rodriguez et al., (1999) used simulated altitudes of 2200 - 2600 m and trained at elevations of less than 600 m. Endurance performance was enhanced relative to the control groups in the range of 1 – 2.3%, which is smaller improvement in performance when compared to live high train high hypobaric. Supramaximal performance was assessed in 2 studies. Numela and Rusko (2000) found that 400 m running performance was improved by 1.1% and Roberts et al., 2003 found a 2.3% improvement in 4 min cycling performance.

Studies which investigated the effect of physiological measures mediating changes in performance have not observed a consistent pattern of change in VO<sub>2</sub> max or hematalogical indices. For example Roberts et al. (2003) found a 0.9% reduction in VO<sub>2</sub> max, but a 12.4% improvement in MAOD following 5 – 15 days of exposure to a simulated altitude of 2650 m for 8-10 hrs per day. In a n earlier investigation, Roberts et al. (2000), found a 2 % reduction in VO<sub>2</sub> max and 17% improvement in MAOD following 12 days of exposure to a simulated altitude of 2650 – 3000 m. Gore et al. (2001), and Hahn et al. (2001) also found small reductions in VO<sub>2</sub> max of 2.4% and 4.3 % respectively. The only study to contradict this trend was Rusko et al. (1999) who found a 5.5 % improvement in VO<sub>2</sub> max and a 6.4% improvement Red Cell Volume.

A possible explanation for the lack of improvement in VO<sub>2</sub> max is that the exposure periods in all of these studies which showed a decrement in VO<sub>2</sub> max were considerably shorter (8 -11 h day ) when compared to the exposure period (12 – 16 hrs per day) employed by Rusko et al. (1999). This statement is given further strength

when you consider that the average exposure period for Hypobaric hypoxia is between 20 – 22 hours per day. The shorter exposure time periods of (8-11 hrs per day) may not have been long enough to stimulate a noteworthy change in red cell volume or haemoglobin mass, and subsequently no increase in  $VO_2$ max was observed. Furthermore, for the same given level of altitude (partial pressure of oxygen), hypobaric hypoxia has been reported to lead to a lower arterial oxygen saturation, greater hypocapnia and blood alkalosis (Savourey et al., 2003). In summary, normobaric live high / train low can improve both endurance and supramaximal exercise performance. However the magnitude of effect is not as great as hypobaric live high / train low. Possibly this is due to a smaller amount of total exposure time. This warrants further investigation.

### **Intermittent hypoxia**

Initially investigated in Russia some 50 years ago, intermittent hypoxic exposure is an alternative method of stimulating hypoxia that has gained increased popularity over the past decade. In this method the athlete breathes hypoxic (generally normobaric) air through a mask intermittently for 60 – 90 minutes, per day for a period of 2 - 5 weeks. (Wood et al. 2003). This method poses only minimal interference to the athlete's living and training environment. Alternatively, rebreathing devices have been employed to produce hypoxia, with the same exposure protocol.

To date, 4 studies have investigated the effect of intermittent hypoxic exposure on endurance and supramaximal exercise performance. However, these studies are vastly different in terms of the volume and frequency of hypoxia used, although the relative intensity 9% - 12%  $FIO_2$  is similar in all studies. Two studies have shown a positive enhancement in performance and hematological indices following exposure to normobaric intermittent hypoxic exposure at rest.

Hellems (1999) conducted a study involving 10 elite endurance athletes who completed 20 days of Intermittent hypoxic training. The athletes breathed hypoxic air through a nose-face mask device (hypoxicator) in  $FIO_2$  concentrations of 10%  $O_2$  for the first 10 days and 9%  $O_2$  for the second 10 days. The hypoxic exposures were 5

minutes in length and followed by equivalent time periods of normoxic exposure. Each of the sessions lasted 60 minutes and were conducted twice per day. The athletes continued their normal training during the experimental period, but were instructed to take a minimum of 1 hour of recovery between an exposure sessions and a workout. The results from this study were limited but showed significant improvements in endurance performance (3%), reticulocyte count (29%), hemoglobin (4%), and hematocrit (5%). Unfortunately no control group was used to establish comparison.

Recently Wood et al (2003) found that normobaric intermittent hypoxia exposure via the use of a rebreathing device provided a substantial performance enhancement in aerobic and anaerobic exercise performance. In this study 29 trained male hockey and soccer players were divided in double-blind fashion to altitude (n=15) or placebo (n=14) groups for 15 d of daily use of a functional or placebo re-breathing device (Alto lab) to simulate the hypoxia of altitude. Each day's exposure consisted of alternately breathing stale and fresh air for 6 and 4 min respectively over 1 h. Oxygen saturation was monitored individually with pulse oximeters and progressively reduced in the altitude group (90% on Day 1, 77% on Day 15; equivalent altitudes ~3600-6000 m). These were similar to the oxygen saturations employed in all previously reviewed simulated altitude studies. Performance tests, which included blood-lactate and heart-rate measurements, were an incremental run to maximum speed followed by a set of six maximal-effort running sprints; tests were performed 1 d before, 3 d after, and 12 d after the 15-d treatment. Blood-cell counts were taken 1 d pre and post treatment. Measures of blood acid-base status were taken immediately pre and post exposure on Days 1 and 15.

Relative to placebo, at 3 d post treatment the altitude group showed a mean increase in maximum speed of 2.0% (90% confidence limits,  $\pm 0.5\%$ ); sprint speed was also relatively faster by 1.5% in the first sprint through 6.9% in the last ( $\pm 1.9\%$ ). Interestingly subjects individual perception of training quality was initially lower (Days 1-6) in the hypoxic group but then remained significantly higher than the placebo for the remainder of the study (Days 7-15). Noteworthy changes in blood measures in the altitude group relative to placebo were increases in hemoglobin concentration (1.2%,  $\pm 1.9\%$ ) and hematocrit (2.0%,  $\pm 2.7\%$ ), and substantial reductions in exercise lactates

and resting and exercise heart rates. Large effects on performance were still present 9 d later. These authors concluded that acutely intermittent simulated altitude exposure substantially improves high-intensity running performance, possibly via changes in oxygen transport.

In contrast to these studies, Frey et al. (2000) reported that normobaric intermittent hypoxic exposure had no effect on hematological indices and submaximal or maximal exercise responses in moderately trained females and males. These athletes were exposed to intermittent normobaric hypoxia for 75 minutes per day for 21 days, during which time the athletes were exposed to an inspired oxygen fraction ~9% (6400m/20992ft) using a hypoxicator device similar to that employed by Hellemons. Specific details of the exposure:recovery ratio were not reported. Initially 2 hours after the first IHT session there was a significant ( $p < 0.05$ ) increase in serum EPO levels (38%), However throughout the remainder of the study there were no other changes in serum EPO levels, reticulocyte counts, hemoglobin, or hematocrit. In addition, there were no differences in submaximal oxygen uptake at 2.0 and 4.0 mmol/L blood lactate levels, or in  $VO_{2max}$ . Unfortunately this study did not have a control group for comparison.

Most recently in a well designed and controlled study, Julian et al. 2003 examined the effect of 4 weeks of normobaric intermittent hypoxic exposure on 3000 m running performance. They recruited fourteen national class distance runners to complete a four week regimen (5:5 minute hypoxic:normoxic ratio for 70 minutes, 5 times per week) of either intermittent normobaric hypoxia (HYP) or placebo control (NORM) at rest. The experimental group was exposed to a graded decline in the fraction of inspired oxygen ( $FIO_2$ ) (week 1: = 0.12, week 2: = 0.11, weeks 3 and 4: = 0.10). The placebo control group was exposed to the same temporal regimen, but breathed an  $FIO_2 = 0.209$  for the entire four weeks.

Subjects were matched for training history, gender, and baseline measures of maximal oxygen uptake ( $VO_{2max}$ ) and 3000 m time trial (3000TT) performance in a randomized, balanced, double blind design. These measures, along with submaximal treadmill performance (economy, heart rate, lactate, ventilation) were made in duplicate prior to the intervention as well as one and three weeks post. Hematological indices including serum concentrations of erythropoietin (EPO), soluble transferrin receptor

(sTfr) and reticulocyte parameters (flow cytometry) were made twice before, on days 1, 5, 10 and 19 of the intervention and 10 and 25 days post. These authors found that there were no significant differences in VO<sub>2</sub>max, 3000TT, EPO, sTfr or reticulocyte parameters between groups at any time. They concluded that four weeks of a 5:5 minute normobaric hypoxia exposure at rest for 70 minutes, 5 days per week is not a sufficient stimulus to elicit improved performance, nor change the normal level of erythropoiesis in highly trained runners.

It is interesting to note that the 2 controlled studies, Wood et al. (2003) and Julian et al (2003) had conflicting results. In addition to improved hematological indices. Wood found an impressive improvement in peak aerobic speed and repeated sprint performance following only 15 days of normobaric intermittent hypoxia. However, Julian et al (2003) found no improvements in aerobic capacity, endurance performance or hematological indices. These authors proposed that 70 min of intermittent hypoxia is not an adequate stimulus for enhancement of endurance performance or hematological indices. However, Wood's result clearly refutes that rationale. Perhaps the 15 days employed by Wood is a sufficient time frame and the extra stimulus that was employed by Julian may have led to overtraining. However the results of Hellemans study contradict this since they used a 20 day exposure period and gained a substantial improvement in endurance performance and hematological indices. It is possible that other mechanisms, which cannot be appropriately assayed via hematology, could be responsible for performance enhancement following intermittent hypoxia. In addition no research has investigated the effects of altering the hypoxic interval length / recovery ratio or adding additional periods of hypoxia. Further research investigating these areas is warranted.

### **Summary and Recommendations**

A review of the available literature would suggest that much is still to be learnt about the physiological and performance responses of hypoxic exposure on exercise performance, particularly with regards to intermittent hypoxic exposure and its effect on supramaximal exercise. Hypoxic exposure has been shown to produce favourable changes in oxygen transport (Krantz et al, 1991; Berglund 1992), and utilisation mechanisms (Desplanches et al. 1993). In addition other oxygen independent mechanisms (Roberts et al 2003) have also been proposed to improve as a result of

hypoxic exposure. However despite these positive adaptations some methods of hypoxic exposure exhibit a questionable effect on exercise performance.

Traditionally hypoxic exposure has been carried out with the athlete residing and training in a natural hypobaric atmosphere (altitude). This hypoxic strategy has yielded inconsistent results. A possible explanation for the lack of consistent results is that VO<sub>2</sub> max is reduced at altitude (McCardle et al 1998). Accordingly it has been theorised that this reduction in fitness may offset any positive physiological benefits gained from altitude exposure (Levine 2002). The live high train low approach avoids detraining by exercising at low altitudes where normal exercise intensities can be maintained. This approach is generally very successful especially in a natural hypobaric environment with typical gains in the order of a 2-3 % enhancement in endurance performance. Smaller performance gains in the order of 1-2 % are generally observed when this approach is carried out in a normobaric hypoxic environment. A possible explanation for the discrepancy between these two methods is that in normobaric hypoxia the athlete will generally receive a smaller volume of hypoxic exposure per day.

Despite the potential for improved performance with these methods, there are some shortcomings. If the athlete chooses to use natural hypobaric hypoxia, they will have the financial cost of travelling to an appropriate altitude. In addition they may not have access to suitable training conditions. This is an obvious problem for kayak athletes. Normobaric hypoxia solves the problem of travel to altitude and should not interrupt the athletes normal training routine. However these devices are expensive and the athlete will still have to spend between 8 – 18 hours in either an altitude house or tent which could prove to be a major inconvenience for many athletes.

Intermittent hypoxic exposure involves the athlete performing short intervals of hypoxia followed by a recovery period of normoxia, for a duration of 60 – 120 minutes per day (Wilber et al 2001). This method does not alter the athletes normal environment and has been shown to produce favourable changes in exercise performance (Wood et al. 2003; Hellemans 1999). Of the four studies which have investigated intermittent hypoxic exposure only two had control groups (Wood et al., 2003; Julian et al., 2003). All of these studies employed similar hypoxic interval lengths/ recovery ratio. In addition, no studies have investigated the effects of stacking extra periods of hypoxia.

Modification of these two parameters alone could lead to an enhancement in endurance performance. In addition little is known about the mechanisms responsible for performance improvement following intermittent hypoxia. Therefore further research is warranted investigating the effects of intermittent hypoxic exposure on both endurance and supramaximal exercise performance, in particular we need to:

- Establish the effect of altering the interval length and recovery ratio.
- Establish the effect of adding additional periods of hypoxia
- Assess the effect of intermittent hypoxic exposure on supramaximal exercise performance
- Develop a greater understanding of potential mechanisms responsible for performance enhancement

This research will be the first series of studies systematically investigating alterations in the hypoxic interval length / recovery ratio. In addition the effects of these interventions will be measured by changes in flat-water kayak performance. Research on flat-water kayak performance and physiology is limited. This research will expand our knowledge in this field and create a deeper understanding of potential mechanisms which mediate performance changes following hypoxic exposure.

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## Appendix B

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### Reliability of Peak-Incremental and Repeat-Sprint Power in Kayak Ergometry

#### Introduction

Understanding the reliability of performance assessments is important to Sport Scientists. Flat-water kayaking is one such sport where the effect of wind, water depth, temperature and waves can alter the reliability and outcome of any performance assessment. For this reason laboratory based assessment using kayak ergometers are often favoured over on-water kayak assessments. It has been demonstrated that air braked kayak ergometry accurately reflects the on-water physiological and technical demands of flat-water kayaking (von Someren, Phillips, & Palmer, 2000). However, the reliability of power output generated during kayak ergometry is unknown. Some preliminary data from our laboratory using the Dansprint air-braked kayak ergometer (ref bonetti) has found relatively high errors of measurement for peak aerobic power (3.0%) in comparison to published reliability of <2% for peak aerobic power in cycle ergometry and treadmill running (Hopkins, Schabort, & Hawley, 2001)

In an attempt to improve the error of measurement of the Dansprint, we conducted a pilot study and made some small modifications to the ergometer shaft and air intake settings which appear to have improved the accuracy of the ergometer. Therefore, investigating the reliability of power output in a series of trials is warranted to identify measurement of error accurately with the modified ergometer and to ascertain whether it is a reliable enough to use in future studies.

Additionally, assessments of aerobic and anaerobic indices are usually conducted on different days. However, this results in extra time constraints for subjects and may restrict their willingness to participate in a study. We developed a time efficient protocol to measure anaerobic and aerobic variables of interest in one testing session and wanted to assess its efficacy to use in future studies. Therefore the aim of the present study was to ascertain the test-retest reliability of kayak ergometry and to investigate the efficacy of a test protocol designed to assess aerobic and anaerobic capacity within the same testing session.

## Methods

### *Subjects*

A group of 8 flat-water kayak paddlers: Age  $28 \pm 9$  y; height  $183 \pm 6$  cm;  $84 \pm 9$  kg; who had at least 2 years of national or international race experience and a 500-m time of  $<2$  min were recruited. This study took place during the competitive sprint racing season. Subjects gave informed consent as required by the institutions ethics committee.

### *Training and Diet*

During the study all subjects followed a similar training program. The weekly schedule consisted of a 5-km race, 4-6 paddling sessions at aerobic threshold, 2-4 interval paddles at race-specific intensities, and 2-3 resistance-training sessions. The subjects maintained their normal diet during the course of both interventions and were instructed to have an easy day of training prior to each testing session.

### *Performance Tests*

All physiological and performance tests were conducted in a temperature controlled laboratory ( $19-21^{\circ}\text{C}$ ) using a calibrated, wind-braked kayak ergometer (Dansprint, Hvidovre, Denmark) The foot-bar position of the kayak ergometer was adjusted to resemble each paddlers kayak. In each testing session the athletes performed an incremental step test to exhaustion and a repeated 30-s sprint test. These tests were separated by a 20-min recovery period consisting of 5 min of rest, 10 min paddling at 50% of peak power, and a final 5 min of rest. Each athlete was required to complete 4 testing sessions, separated by 5-7 days. The first testing session served as a familiarisation to the ergometer and protocols. Only 2 of the 8 athletes recruited for this study had no prior experience with the test ergometer, none of the subjects had prior experience with the repeats 30 s test.

The, incremental step test commenced at a workload of 50-90 W, and increased by 20 W every 4-min until volitional exhaustion. There was a 1-min rest period between stages where capillary blood was sampled from an earlobe for measurement of blood lactate using a hand-held analyser (Lactate Pro, Arkray, Japan). In addition, pulmonary

oxygen uptake ( $\text{VO}_2$ ) was measured continuously using a breath-by-breath metabolic system (Metamax 3b, Cortex, Leipzig, Germany). Heart rate was continuously measured using a short-range telemetry device (Polar A1, Polar Electro, Kempele, Finland). The average heart rate obtained in the final 10 s of each stage was recorded. Maximum oxygen uptake ( $\text{VO}_{2\text{max}}$ ) was determined as the highest 30-s value obtained during the test. A measure of lactate-profile power (analogous to the individual lactate-threshold power) was derived from the step tests as follows. We assumed a log-log relationship between lactate concentration and power output (Beaver, Wasserman, & Whipp, 1985). We used the Trend function in Microsoft Excel to fit straight lines to the pre- and post-treatment lactate plots, then predicted power output corresponding to a "midpoint" of lactate concentration in the step tests. The midpoint was found by averaging the minimum and maximum values of the log-transformed lactate concentrations from all four tests. A similar procedure was used to create individual power profiles of heart rate and exercise economy; these variables did not require log transformation and power was calculated at fixed percentages of the individual's maximum value (heart rate 90%, exercise economy 70%).

The repeated sprint test consisted of four 30-s all-out sprints. From a stationary start, the athletes were instructed to exert maximal stroke-rate and power-output throughout the 30-s period. Immediately following each repetition athletes were instructed to stop paddling for 15 s, they were then permitted to paddle at a self-selected pace for 30 s, and then rest for the remaining 15 s prior to the start. Athletes were verbally encouraged, and given time updates, during each 30-s repetition. Measures of peak and mean power were obtained using specifically designed software.

#### *Data Analysis*

Measures of reliability were derived using a spreadsheet downloaded from a website ([www.newstats.org/xrely.xls](http://www.newstats.org/xrely.xls)). The spreadsheet uses log-transformation to compute standard error of measurement as a coefficient of variation and changes in the mean between consecutive pairs of trials. The spreadsheet also calculates 90% confidence limits in the estimates.

## Results and Discussion

The mean results for both physiological and performance measures obtained during each testing session are shown in Table 1. From the total of 4 testing sessions, 5 of the 8 athletes completed 4 incremental step tests. Due to some ergometer problems experienced during the 30 s test, 3 out of 8 athletes, completed 4 repeat sprint tests and 6 out of 8 athletes completed 3 repeat sprint tests. Errors of measurement testing for performance and physiological measures are shown in Table 2.

Table 1. Performance in the four sessions of the reliability study and changes in the mean between sessions.

	Performance in each session (mean $\pm$ SD)				Changes in mean between sessions (%)		
	Session 1	Session 2	Session 3	Session 4	2-1	3-2	4-3
<b>Incremental step test</b>							
Peak power (W)	163 $\pm$ 24	171 $\pm$ 24	176 $\pm$ 18	175 $\pm$ 18	4.9	2.9	-0.6
Lactate-profile power (W)	127 $\pm$ 19	135 $\pm$ 20	136 $\pm$ 19	134 $\pm$ 16	6.3	0.7	-1.5
Heart-rate profile power (W)	123 $\pm$ 21	134 $\pm$ 20	138 $\pm$ 23	141 $\pm$ 16	8.9	3.0	2.2
VO <sub>2</sub> max (L.min <sup>-1</sup> )	3.78 $\pm$ 0.37	3.90 $\pm$ 0.6	4.15 $\pm$ 0.48	3.96 $\pm$ 0.25	3.2	6.4	-4.6
Economy (W)	113 $\pm$ 15	107 $\pm$ 11	105 $\pm$ 10	107 $\pm$ 5	5.3	1.9	1.9
Peak lactate (mmol.L <sup>-1</sup> )	10.3 $\pm$ 2.6	9.8 $\pm$ 2.6	10.2 $\pm$ 2.2	10.2 $\pm$ 3.1	4.9	4.1	0.0
Peak heart rate (beats.min <sup>-1</sup> )	182 $\pm$ 11	179 $\pm$ 10	180 $\pm$ 8	180 $\pm$ 7	1.6	0.6	0.0
<b>Repeated sprint test</b>							
Mean sprint power (W)	265 $\pm$ 32	277 $\pm$ 39	277 $\pm$ 37	264 $\pm$ 34	4.5	0.0	4.7
First sprint power (W)	300 $\pm$ 50	302 $\pm$ 53	306 $\pm$ 50	298 $\pm$ 43	0.7	1.3	2.6
Final sprint power (W)	242 $\pm$ 26	256 $\pm$ 36	254 $\pm$ 27	271 $\pm$ 45	5.8	0.8	6.7
Peak lactate (mmol.L <sup>-1</sup> )	11.7 $\pm$ 2	10.9 $\pm$ 2.6	11.4 $\pm$ 3.0	11.1 $\pm$ 3.2	-6.8	4.6	2.6

There was evidence of a substantial learning effect between the first two testing sessions, which is consistent with reliability studies of other performance tests (Hopkins, Schabert, & Hawley, 2001): percent change in the mean (Table 1) and errors of measurement (Table 2) for nearly all measures were highest between the first two sessions. Collectively these data indicate that for aerobic and anaerobic measures on the

Dansprint ergometer, at least one familiarisation sessions should be performed prior to any physiological or performance baseline measures to overcome any learning effects.

Table 2. Standard error of measurement for performance and physiological measures expressed as coefficients of variation (%).

	Session 1 & 2	Session 2 & 3	Session 3 & 4	Mean of Sessions 2&3 and 3&4 <sup>a</sup>
<b>Incremental step test</b>				
Peak power	5.1	2.0	2.8	2.3
Lactate-profile power	4.7	4.3	3.8	4.1
Heart-rate profile power	7.4	5.8	3.7	5.1
VO <sub>2</sub> max	8.4	4.5	4.1	4.4
Economy	9.6	6.1	4.3	5.7
Peak lactate	13.1	19.7	5.5	16.1
Peak heart rate	1.7	2.0	1.8	1.9
<b>Repeated sprint test</b>				
Mean sprint power	4.6	2.1	4.0	3.0
First sprint power	7.8	2.5	4.8	3.7
Final sprint power	8.7	2.9	5.3	4.3
Peak lactate	11.2	11.1	4.4	8.8

<sup>a</sup>Range of 90% Confidence limits:  $\times/\div 1.4$  to  $\times/\div 1.6$

One of the objectives of this study was to determine whether modifications to the ergometer shaft and air resistance settings would reduce error of measurement. Relative to what has been previously reported by our laboratory (Bonetti, Hopkins, & Kilding, 2006) we were able to demonstrate improvements in error of measurement for peak power (2.3% vs 3.0%) repeat sprint power (3.0% vs 4.3%) and peak VO<sub>2</sub> (4.4% vs 4.7%). However, measurement error for economy (5.7% vs 5.0%), lactate threshold (4.6% vs 4.1%) and heart-rate profile (5.1% vs 3.5%) were not improved. Additionally, the measurement errors for the variables of most importance in terms of future research (peak power, lactate threshold) are still considerably greater than has been reported for other modes of exercise, such as cycling and running (Hopkins, Schabert, & Hawley, 2001).

Our data indicate that anaerobic indices of performance can be calculated with reasonable accuracy within the same testing session as maximal aerobic assessment in subelite flat-water kayakers. Peak lactates were similar to the range reported following 500-m sprint time trials in an earlier investigation (Bonetti, Hopkins, & Kilding, 2006) and substantially higher than those recorded in the incremental step test preceding the repeat sprints. These results provide reasonable evidence that the preload of the incremental step test did not incur excessive glycogen depletion or excessively fatigue the athletes for a series of repeat sprint test 20 min later.

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## **Appendix C**

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### **Ethical approval and relevant subject information for Study 2**

## M E M O R A N D U M

**Student Services Group - Academic Services**

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To: Will Hopkins  
From: **Madeline Banda**  
Date: 02 August 2004  
Subject: 04/137 The effects of intermittent simulated altitude exposure using the Body02 Sat device on high intensity sprint kayak performance

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Dear Will

Thank you for providing clarification and/or amendment of your ethics application as requested by AUTEK.

Your application is approved for a period of two years until 2 August 2006.

You are required to submit the following to AUTEK:

- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda  
**Executive Secretary**  
**AUTEK**  
Cc: Darrell Bonetti

# Participant Information Sheet

**Date Information Sheet Produced:** June 2004

## Project Title

The effects of intermittent simulated altitude exposure using the BodyO<sub>2</sub> simulated altitude training device (BodyO<sub>2</sub> Sat) on kayak performance and physiology.

## Invitation

As a promising national / international calibre kayak paddler you are invited to participate in this research.

## What is the purpose of the study?

To provide evidence that the BodyO<sub>2</sub> Sat enhances kayak performance, and to measure variables that might help us understand how the device enhances performance.

## What happens in the study?

You will either use the BodyO<sub>2</sub> Sat device for one hour a day for 15 days, or you will be in the control group, will do the same training as the altitude group but not receive the altitude treatment. On various occasions throughout the study you will do performance tests and provide venous blood samples. You will use the devices and provide blood samples under supervision at the North Shore Canoe Club. You will visit the Millennium Institute for all performance tests.

The times for the performance tests are 14 days and 1 day before the beginning of the 15-day period on the device, then 3 and 12 days after the 15-day period. Blood samples will be taken 1 day before the first period, 4 and 7 days into the first period, and 3 days after the first period.

You use the device by breathing through it for 5 minutes, followed by 5 minutes of breathing normal air. You repeat this process 6 times in a session. We monitor the amount (saturation) of oxygen in your blood with a pulse oximeter, which fits around a finger. Normally your blood is 98-99% saturated with oxygen. Over the 15 days we will

bring your saturation down to no less than 75%, if you are one of the subjects getting altitude. That's the equivalent of being at an altitude of about 5500 m. It's important for the design of the study that you are allocated to the altitude or control group. We need a control group to understand how effective the altitude device is. It is very important that you stick to the set training programme. This programme has been drafted up in consultation with your coach and will produce good results.

There are three performance tests. First you do an incremental paddle test to maximum effort on a kayak ergometer while you breathe into a facemask or mouthpiece so we can measure your oxygen uptake. The test is stopped every 4 minutes so a droplet of blood can be taken from your earlobe. The test lasts about 30 minutes altogether. On the following day you will then do a simulated 1000 m race on the kayak ergometer. This involves completing as much work as possible in four minutes. After a 30-min rest, you will then complete 4 x 15 s all-out sprints, with 15 s recovery. At the conclusion of this test the same blood sampling procedures will be used.

### **What are the discomforts and risks?**

When you breathe through the BodyO<sub>2</sub> Sat device, you may feel a bit breathless and dizzy or headachy from the shortage of oxygen. Your heart rate also increases a little. Thousands of people have used this device, apparently without any harm.

The exercise tests produce the feelings of effort and fatigue that you are used to in hard training and racing. Exercise tests increase the risk of a heart attack during or shortly after the test, but the risk for healthy young adults is very small and no greater than you would encounter during your normal training. The long-term effects of such exercise is an overall reduction in risk of heart attacks and other diseases.

Blood sampling produces a bit of pain from the puncturing of your skin with a needle for the venous blood samples or with a small lancet for the droplets of blood taken during the performance kayak tests.

### **How will these discomforts and risks be alleviated?**

We can't really do anything about the discomforts. If it gets too uncomfortable, you just stop doing it. As far as the risks are concerned, there will always be a trained qualified person present who knows how to deal with any incidents or accidents.

**What are the benefits ?**

As an athlete you will get some useful physiological information about your body which can be implemented into your training schedule. Your participation will help us understand the benefits of the BodyO<sub>2</sub> Sat for kayak performance. Understanding what's going on might also help us work out how to use it even more effectively.

**What compensation is available for injury or negligence?**

Only the usual ACC compensation.

**How will my privacy be protected?**

We don't put your name into a computer with your data. Hard copies of data are kept in a locked filing cabinet and are eventually destroyed.

**How do I join the study?**

Let your coach and Darrell Bonetti know.

**What are the costs of participating in the project? (including time)**

Time is the only cost. The daily sessions with the BodyO<sub>2</sub> Sat device take an hour. There are 15 sessions altogether, with the protocol been 5 days on (Monday – Friday) 2 days off (Saturday and Sunday). There are 6 visits to the Millennium Institute altogether. Transport to the Institute will be provided or the cost will be reimbursed as petrol vouchers. Each visit will take about an hour, plus travel time.

**Opportunity to consider invitation**

We need to get started by mid September, so we need a quick decision. There is a lead-in time of two weeks while we do baseline performance testing and training. You can change your mind about your participation in the study at any time throughout the study. Your participation is entirely voluntary. You can ask Darrell, or Will Hopkins, or any independent person for more information about the project before or after signing up. (An independent altitude researcher and sports physician in New Zealand with no ties to the present researchers is Dr Jon Hellemans in Christchurch.)

**Opportunity to receive feedback on results of research**

We will provide you with your own results and the average results of all participating paddlers.

**Participant Concerns**

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor. Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEK, Madeline Banda, [madeline.banda@aut.ac.nz](mailto:madeline.banda@aut.ac.nz), 917 9999 ext 8044.

**Researcher Contact Details:** Darrell Bonetti, Sport and Recreation, Auckland University of Technology, Phone 021 719 643, [dbonetti@aut.ac.nz](mailto:dbonetti@aut.ac.nz).

**Other Investigator Contact Details:** Will Hopkins, Sport and Recreation, Auckland University of Technology. Phone 917 9793, after hours 376 0198.

**Other Investigator Contact Details:** Charlotte Harrison, Altitude Science Ltd. 921 1487.



## Informed Consent Form

**Project Title:** The effects of intermittent simulated altitude exposure using the BodyO<sub>2</sub> simulated altitude training device (BodyO<sub>2</sub> Sat) on kayak performance and physiology.

**Principal Investigator:** Professor Will Hopkins, Tel: 09 917 9793

**Other Researchers:**

Darrell Bonetti

I have read and understood the subject information sheet for the above titled research project to be conducted by Darell Bonetti at Auckland University of Technology.

This is to certify that I \_\_\_\_\_ hereby agree to participate as a volunteer in the study described.

- I have read and understand what is required of me as a participation in this study. YES  NO  (please tick)
- I am aware of the benefits and risks associated with my participation in this study. YES  NO
- I am aware that I am free to withdraw my consent at any time without giving reason. YES  NO
- I understand that my name will not be associated with the research.  
YES  NO
- I have been given the opportunity to ask whatever questions I desire and all such questions have been answered to my satisfaction.  
YES  NO

Participant: \_\_\_\_\_ Date: \_\_\_\_\_

Witness: \_\_\_\_\_ Date: \_\_\_\_\_

Researcher: \_\_\_\_\_ Date: \_\_\_\_\_

## **Appendix D**

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Ethical approval and relevant subject information for  
reliability study



## MEMORANDUM

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To: Will Hopkins  
 From: **Madeline Banda** Executive Secretary, AUTECH  
 Date: 25 October 2005  
 Subject: Ethics Application Number 05/193 **The reliability of kayak ergometry and on-water kayak assessment.**

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Dear Will

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTECH) at their meeting on 10 October 2005. Your ethics application is now approved for a period of three years until 25 October 2008.

I advise that as part of the ethics approval process, you are required to submit to AUTECH the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2, which is available online through <http://www.aut.ac.nz/research/ethics>, including a request for extension of the approval if the project will not be completed by the above expiry date;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/ethics>. This report is to be submitted either when the approval expires on 25 October 2008 or on completion of the project, whichever comes sooner;

You are reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTECH for approval before that change is implemented.

Please note that AUTECH grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at [charles.grinter@aut.ac.nz](mailto:charles.grinter@aut.ac.nz) or by telephone on 921 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Madeline Banda  
**Executive Secretary**  
**Auckland University of Technology Ethics Committee**

Cc: Darrell Bonetti

# Participant Information Sheet



**Date Information Sheet Produced:** September 2005

## **Project Title**

“The reliability of kayak ergometry & on water kayak assessment”

## **Invitation**

As a competitive kayak paddler you are invited to participate in this study

## **What is the purpose of the study?**

This study will provide us with reliability information that is required to determine how reproducible results are on a day to day basis when performing exercise tests on the “Dansprint erg” and on-water using the athletes racing kayak.

## **What happens in the study?**

You will visit the Millenium Institute twice per week for 4 weeks. On each visit you will perform either an incremental step test or 4 x 30 s repeat sprint tests. On 3 other separate occasions we will perform 4 x 30 s sprints on water using your own racing kayak. (This test will be weather dependent)

The incremental step test involves you paddling to maximum effort on a kayak ergometer while you breathe into a facemask or mouthpiece so we can measure your oxygen uptake. The test is stopped every 4 minutes so a droplet of blood can be taken from your earlobe. The test lasts about 45 minutes altogether. 1 day later you will perform 4 x 30 s sprints on the kayak erg, with 1.5 minutes recovery between each test. At the conclusion of this test the same blood sampling procedures will be used.

## **What are the discomforts and risks?**

The exercise tests produce the feelings of effort and fatigue that you are used to in hard training and racing. Exercise tests increase the risk of a heart attack during or shortly after the test, but the risk for healthy young adults is very small and no greater than you would encounter during your normal training. The long-term effects of such exercise is an overall reduction in risk of heart attacks and other diseases.

Blood sampling produces a bit of pain from the puncturing of your skin with a needle for the venous blood samples or with a small lancet for the droplets of blood taken during the running performance tests.

### **How will these discomforts and risks be alleviated?**

We can't really do anything about the discomforts. If it gets too uncomfortable, you just stop doing it. As far as the risks are concerned, there will always be a trained qualified person present who knows how to deal with any incidents or accidents.

### **What are the benefits ?**

As an athlete you will get some useful physiological information about your body which can be implemented into your training schedule. Your participation will help us understand the reliability of these testing procedures.

### **What compensation is available for injury or negligence?**

Only the usual ACC compensation.

### **How will my privacy be protected?**

We don't put your name into a computer with your data. Hard copies of data are kept in a locked filing cabinet and are eventually destroyed.

### **How do I join the study?**

Let your coach and Darrell Bonetti know.

### **What are the costs of participating in the project? (including time)**

Time is the only cost. The daily sessions with the BodyO<sub>2</sub> Sat device take an hour. There are 15 sessions altogether, with the protocol been 5 days on (Monday – Friday) 2 days off (Saturday and Sunday). There are 6 visits to the Millennium Institute altogether. Transport to the Institute will be provided or the cost will be reimbursed as petrol vouchers. Each visit will take about an hour, plus travel time.

### **Opportunity to consider invitation**

We need to get started by Mid October, so we need a quick decision. There is a lead-in time of two weeks while we do baseline performance testing and training. You can change your mind about your participation in the study at any time throughout the study. Your participation is entirely voluntary. You can ask Darrell, or Will Hopkins, or

any independent person for more information about the project before or after signing up.

### **Opportunity to receive feedback on results of research**

We will provide you with your own results and the average results of all participating athletes.

### **Participant Concerns**

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor. Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEK, Madeline Banda, [madeline.banda@aut.ac.nz](mailto:madeline.banda@aut.ac.nz) , 917 9999 ext 8044.

**Researcher Contact Details:** Darrell Bonetti, Sport and Recreation, Auckland University of Technology, Phone 021 719 643, [dbonetti@aut.ac.nz](mailto:dbonetti@aut.ac.nz).

**Other Investigator Contact Details:** Will Hopkins, Sport and Recreation, Auckland University of Technology. Phone 917 9793, after hours 376 0198.

**Project Title:**

“The reliability of kayak ergometry & on water kayak assessment”

**Principal Investigator:** Professor Will Hopkins, Tel: 09 921 9793

**Other Researchers:**

Darrell Bonetti

I have read and understood the subject information sheet for the above titled research project to be conducted by Darrell Bonetti at Auckland University of Technology.

This is to certify that I \_\_\_\_\_ hereby agree to participate as a volunteer in the study described.

- I have read and understand what is required of me as a participation in this study. YES  NO  (please tick)
- I am aware of the benefits and risks associated with my participation in this study. YES  NO
- I am aware that I am free to withdraw my consent at any time without giving reason. YES  NO
- I understand that my name will not be associated with the research. YES  NO
- I have been given the opportunity to ask whatever questions I desire and all such questions have been answered to my satisfaction. YES  NO

Participant: \_\_\_\_\_ Date: \_\_\_\_\_

Witness: \_\_\_\_\_ Date: \_\_\_\_\_

Researcher: \_\_\_\_\_ Date: \_\_\_\_\_

## **Appendix E**

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Ethical approval and relevant subject information for Study

3



## MEMORANDUM

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To: Will Hopkins  
 From: **Madeline Banda** Executive Secretary, AUTECH  
 Date: 10 February 2006  
 Subject: Ethics Application Number 05/99 **The effects of altered hypoxic intervals on cycling performance.**

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Dear Will

The Executive Secretary, acting under delegated authority, has approved minor amendments altering the participant group from runners to cyclists and reducing the number of performance tests from three to two, subject to endorsement at AUTECH's meeting of 13 March 2006.

I am pleased to advise that the Executive Secretary, acting on delegated authority from the Auckland University of Technology Ethics Committee (AUTECH) in terms of section 5.3 of AUTECH's *Guidelines and Procedures*, has approved minor amendments altering the participant group from runners to cyclists and reducing the number of performance tests from three to two, subject to endorsement at AUTECH's meeting of 13 March 2006.

I remind you that as part of the ethics approval process, you are required to submit to AUTECH the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2, which is available online through <http://www.aut.ac.nz/research/ethics> including a request for extension of the approval if the project will not be completed by the above expiry date;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/ethics>. This report is to be submitted either when the approval expires on 9 June 2008 or on completion of the project, whichever comes sooner;

You are also reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTECH for approval before that change is implemented.

Please note that AUTECH grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at [charles.grinter@aut.ac.nz](mailto:charles.grinter@aut.ac.nz) or by telephone on 921 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Madeline Banda  
**Executive Secretary**  
**Auckland University of Technology Ethics Committee**  
 Cc: Darrell Bonetti [dbonetti@aut.ac.nz](mailto:dbonetti@aut.ac.nz)



## Participant Information Sheet

**Date Information Sheet Produced:** January 2006

### Project Title

The effects of altered hypoxic intervals on cycling performance

### Invitation

As a competitive cyclist you are invited to participate in this study

### What is the purpose of the study?

To provide evidence that the BodyO<sub>2</sub> Sat enhances cycling performance, and to measure variables that might help us understand how the device enhances performance.

### What happens in the study?

You will either use the BodyO<sub>2</sub> Sat device for one hour a day for 15 days. On various occasions throughout the study you will do performance tests and provide venous blood samples. You will use the devices and provide blood samples under supervision at the Millennium Institute. You will visit the Millennium Institute for all performance tests.

The times for the performance tests are 14 days and 1 day before the beginning of the 15-day period on the device, then 3 and 10 days after the 15-day period. Blood samples will be taken 1 day before the first period, 4 and 7 days into the first period, and 3 days after the first period.

You use the device by breathing through it for 5 minutes, followed by 5 minutes of breathing normal air. You repeat this process 6 times in a session. We monitor the amount (saturation) of oxygen in your blood with a pulse oximeter, which fits around a finger. Normally your blood is 98-99% saturated with oxygen. Over the 15 days we will bring your saturation down to no less than 75%, if you are one of the subjects getting altitude. That's the equivalent of being at an altitude of about 5500 m. It is very

important that you stick to the set training programme. This programme has been drafted up in consultation with your coach and will produce good results.

There are two performance tests, both of which are to be completed on the same day. First you do an incremental cycling test to maximum effort on a treadmill while you breathe into a facemask or mouthpiece so we can measure your oxygen uptake. The test is stopped every 4 minutes so a droplet of blood can be taken from your earlobe. The test lasts about 40 minutes altogether. Following a twenty minute rest you will then perform 4 x 30 s sprints, each separated by 1 min of recovery. At the conclusion of this test the same blood sampling procedures will be repeated. The total time in required to complete both of these performance tests will be 75 minutes.

### **What are the discomforts and risks?**

When you breathe through the BodyO<sub>2</sub> Sat device, you may feel a bit breathless and dizzy or headachy from the shortage of oxygen. Your heart rate also increases a little. Thousands of people have used this device, apparently without any harm.

The exercise tests produce the feelings of effort and fatigue that you are used to in hard training and racing. Exercise tests increase the risk of a heart attack during or shortly after the test, but the risk for healthy young adults is very small and no greater than you would encounter during your normal training. The long-term effects of such exercise is an overall reduction in risk of heart attacks and other diseases.

Blood sampling produces a bit of pain from the puncturing of your skin with a needle for the venous blood samples or with a small lancet for the droplets of blood taken during the cycling performance tests.

### **How will these discomforts and risks be alleviated?**

We can't really do anything about the discomforts. If it gets too uncomfortable, you just stop doing it. As far as the risks are concerned, there will always be a trained qualified person present who knows how to deal with any incidents or accidents.

### **What are the benefits ?**

As an athlete you will get some useful physiological information about your body which can be implemented into your training schedule. Your participation will help us understand the benefits of the BodyO<sub>2</sub> Sat for cycling performance. Understanding what's going on might also help us work out how to use it even more effectively.

**What compensation is available for injury or negligence?**

Only the usual ACC compensation.

**How will my privacy be protected?**

We don't put your name into a computer with your data. Hard copies of data are kept in a locked filing cabinet and are eventually destroyed.

**How do I join the study?**

Let your coach and Darrell Bonetti know.

**What are the costs of participating in the project? (including time)**

Time is the only cost. The daily sessions with the BodyO<sub>2</sub> Sat device take an hour. There are 15 sessions altogether, with the protocol been 5 days on (Monday – Friday) 2 days off (Saturday and Sunday). There are 4 visits to the Millennium Institute altogether. Transport to the Institute will be provided or the cost will be reimbursed as petrol vouchers. Each visit will take about an hour, plus travel time.

**Opportunity to consider invitation**

We need to get started by March so we need a quick decision. There is a lead-in time of two weeks while we do baseline performance testing and training. You can change your mind about your participation in the study at any time throughout the study. Your participation is entirely voluntary. You can ask Darrell, or Will Hopkins, or any independent person for more information about the project before or after signing up. (An independent altitude researcher and sports physician in New Zealand with no ties to the present researchers is Dr Jon Hellemans in Christchurch.)

**Opportunity to receive feedback on results of research**

We will provide you with your own results and the average results of all participating athletes.

**Participant Concerns**

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor. Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEK, Madeline Banda, [madeline.banda@aut.ac.nz](mailto:madeline.banda@aut.ac.nz), 917 9999 ext 8044.

**Researcher Contact Details:** Darrell Bonetti, Sport and Recreation, Auckland University of Technology, Phone 021 719 643, [dbonetti@aut.ac.nz](mailto:dbonetti@aut.ac.nz).

**Other Investigator Contact Details:** Will Hopkins, Sport and Recreation, Auckland University of Technology. Phone 917 9793, after hours 376 0198.

**Other Investigator Contact Details:** Mike Davis, Altitude Science Ltd. 921 1487.

## Informed Consent Form



### Project Title:

“The effects of altered hypoxic intervals on cycling performance”

**Principal Investigator:** Professor Will Hopkins, Tel: 09 921 9793

### Other Researchers:

Darrell Bonetti

I have read and understood the subject information sheet for the above titled research project to be conducted by Darrell Bonetti at Auckland University of Technology.

This is to certify that I \_\_\_\_\_ hereby agree to participate as a volunteer in the study described.

- I have read and understand what is required of me as a participation in this study. YES  NO  (please tick)
- I am aware of the benefits and risks associated with my participation in this study. YES  NO
- I am aware that I am free to withdraw my consent at any time without giving reason. YES  NO
- I understand that my name will not be associated with the research. YES  NO
- I have been given the opportunity to ask whatever questions I desire and all such questions have been answered to my satisfaction. YES  NO

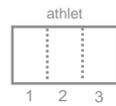
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Researcher: \_\_\_\_\_ Date: \_\_\_\_\_

# TRAINING DIARY

AUT University. Contact: Darrell Bonetti, 021 719 643.



- Fill in the date, the type of session (cycle, run, swim, weights, whatever...), and the duration in hours and minutes.
- Then tick boxes to show the intensity of the session and how tired you felt when you did it (compared with usual).
- Record training as soon as possible after each session.

Date	Type of session	Duration	How <b>intense</b> was the session? (Tick one or two boxes.)				How <b>tired</b> did you feel, compared with the way you usually feel for this kind of session? (Tick one or two boxes.)					ANY COMMENT?	
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**AUT University**Division of Sport and Recreation, Akoranga Campus  
Contact: Darrell Bonetti, 021 719 643

Code#: \_\_\_\_\_

# TRAINING AND PERFORMANCE QUESTIONNAIRE

This questionnaire is about you and your training.

Answer by ticking a box:

or by filling in **ALL** blank lines: like this

**Please put something on every blank line,  
so we know you haven't missed the question.**

Put a dash or a zero where necessary. For example:

Total time: — hours per week

**If you are not sure about something, please estimate.  
Don't spend too much time on it.**

## **CONFIDENTIAL**

This is a valuable document.

Please hand back to Darrell in the first testing session.

Many thanks for your help!

# GENERAL

1

What is your age? \_\_\_\_\_ years

What is your sex?    male         female

What is your height? \_\_\_\_\_ cm OR \_\_\_\_\_ feet \_\_\_\_\_ inches

What is your usual weight? \_\_\_\_\_ kg OR \_\_\_\_\_ stone \_\_\_\_\_ pounds

2

What is your main competitive sport? \_\_\_\_\_

How many years have you been training for this sport? \_\_\_\_\_ years

What specific event have you  
been training for in the last season  
(for example K1 500)? \_\_\_\_\_

What is your present age-group and/or competition class? \_\_\_\_\_

What is the highest level you are competing at in this class?

international     national     local or club

3

Please describe your best performances in competitions in the last 12 months.

approximate date	type of event (if applicable)	distance (if applicable)	time (if applicable)
/ /	_____	_____	_____
/ /	_____	_____	_____
/ /	_____	_____	_____
/ /	_____	_____	_____
/ /	_____	_____	_____

4

How often does a coach advise you about your training?

not at all     less than once a month     once or twice a month     about once a week     several times a week

# TRAINING PROBLEMS in the last 6 months

5

Here is a list of problems that make athletes stop or reduce their training:

**ILLNESS** (colds, flu, viruses, asthma, stomach upsets, infections etc)

**TRAINING INJURIES** (sprains, fractures, pain in muscles etc)

**OVERTRAINING** (You couldn't reach your usual training and performance targets; you may also have felt very tired, worried or lacking in confidence, or had sore muscles and bad moods.)

**or OTHER PROBLEMS** (accidents, family, travel or work problems, bad weather, exams, worry, apathy, loss of interest, etc)

Please list ALL your training problems in the last 6 months.

Show the number of days of LOST training (no training at all)

and/or the number of days of REDUCED training (lighter training than normal).

(An example is shown.)

reason for lost or reduced training	LOST training	REDUCED training
sore shoulder	3 days	5 days
_____	_____ days	_____ days
_____	_____ days	_____ days
_____	_____ days	_____ days
_____	_____ days	_____ days
_____	_____ days	_____ days
_____	_____ days	_____ days

**Please turn over**

# RECENT TRAINING

7

Describe your training for a typical week recently (in the last month) with NO training problems.

Total time spent in training each week: \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

## **endurance training** (non-stop exercise and intervals, each lasting **3 minutes or more**)

Show total time each week at...

...hard or fast pace: \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

...moderate and easy pace: \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

List your main endurance-training activities:

---

## **interval training** (each interval or rep lasting **less than 3 minutes**)

Show total time each week doing...

...hard or fast sets (including rests): \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

...moderate and easy sets (including rests): \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

List your main interval-training activities:

---

## **strength training** (also called resistance training)

Show total time each doing...

...hard or exhausting workouts (including rests): \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

...moderate and easy workouts (including rests): \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

List your main strength-training activities:

---

## **skill training** (also called technique or form training)

Total time each week (including rests): \_\_\_\_\_ hours and \_\_\_\_\_ minutes per week

List your main skill or technique training activities:

---

**Thank you!**