International Journal of Sports Physiology and Performance, 2006;1:246-260

High-Intensity Kayak Performance After Adaptation to Intermittent Hypoxia

Darrell L. Bonetti, Will G. Hopkins, and Andrew E. Kilding

Context: 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/week) of intermittent hypoxic exposure using a nitrogen-filtration device. Each day's exposure consisted of alternately breathing hypoxic and ambient air for 5 minutes each over 1 hour. 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 pretreatment and 3 and 10 days posttreatment. Hemoglobin concentration was measured at 1 day pretreatment, 5 and 10 days during treatment, and 3 days after treatment. ***Results:*** Relative to control, at 3 days posttreatment the hypoxia group showed the following increases: peak power 6.8% (90% confidence limits, ± 5.2%), mean repeat sprint power 8.3% (± 6.7%), and hemoglobin concentration 3.6% (± 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 posthypoxia. ***Conclusion:*** These effects of intermittent hypoxic exposure should enhance performance in kayak racing. The effects might be mediated via changes in oxygen transport. ***Key Words:*** altitude training, lactate threshold, peak power, sprint speed

Hypobaric and normobaric hypoxic exposure in both real and simulated environments are 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 after hypoxic exposure: increased red-blood-cell mass,[1] increased capillarization of muscle,[2] increased myoglobin concentration,[3] increased muscle mitochondrial volume and aerobic enzyme activities,[2] and elevated muscle buffering capacity.[4]

Studies on athletes living and training at altitude have found an enhancement of endurance performance at altitude.[5,6] Because athletes cannot exercise at the

The authors are with the Sport and Recreation Dept, AUT University, Private Bag 92006, Auckland, Auckland 1020 New Zealand.

same intensity while at altitude, however, a relative detraining effect can occur.[1] Currently, there is uncertainty as to whether sea-level performance is improved with this method. To prevent the detraining effect, the live-high train-low method was devised.[7] The performance enhancements with this method are ≈1% to 2% when athletes return to sea level.[8] This method can be expensive and disruptive to an 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 to 8 hours, simulating low to medium altitude using either normobaric (nitrogen houses and tents) or hypobaric hypoxia (barometric chambers) have been extensively investigated. Enhancements in exercise economy,[9,10] endurance performance,[11-13] and performance-related hematology[11-14] 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, by oxygen filtration of the air, or by using rebreathing devices. These methods reduce the partial pressure of oxygen to quantities that are experienced at medium to high altitude (3000 to 6000 m). Thus, the athlete's normal training routine and environment remain unaltered, but they are exposed to a hypoxic environment for intervals of 5 to 7 minutes followed by a similar period of ambient air, for a total of 60 to 90 minutes per day.

Although intermittent hypoxia appears to be a promising training method, there has been limited research supporting its efficacy or physiological adaptations to it. Some investigators have found enhancements in endurance performance,[15,16] repeat sprint performance,[15,17] and performance-related hematology.[16,18] Conversely, other investigators have found little or no change in measures of aerobic or anaerobic performance.[19,20] Given these inconsistencies, we have assessed the effect of intermittent hypoxia with sprint kayak paddlers. Individual sprint kayaking over a 500-m distance is an Olympic high-intensity endurance event taking 96 to 105 seconds to complete. Research has indicated that this event requires an approximately 65% contribution from the aerobic energy system and 35% from the anaerobic energy system,[21] making it an ideal event with which to investigate the effects of intermittent hypoxic exposure.

Methods

Study Design

This study employed a crossover design. Subjects (N = 10) were randomly assigned to 2 groups balanced for best on-water K1 500-m performance during the preceding year. Once placed in the groups, subjects performed 1 week of pretesting 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 postintervention. After a 6-week washout period each group received the other treatment.

Subjects

The subjects were subelite kayak paddlers who had at least 2 years of national or international race experience and a 500-m time of <2 minutes. Before the start of the study all subjects had been training consistently for at least 3 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 diets during the course of both interventions and were instructed to have an easy day of training before each testing session. All subjects underwent an assay for ferritin status before the start of the study. All were found to be in the normal range, and therefore no iron supplementation was administered.

Hypoxic Treatment

The BodyO_2 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_IO_2) and peripheral oxygen saturation as follows:

- Days 1 to 5: oxygen saturation = 90%, 88%, 86%, 86%, 84%; F_IO_2 = 12%
- Days 6 to 10: oxygen saturation = 82%, 82%, 80%, 80%, 78%; F_IO_2 = 11%
- Days 11 to 15: oxygen saturation = 78%, 78%, 76%, 76%, 76%; F_IO_2 = 10, 9%

This protocol was based on previous research[15] and previous experience of the manufacturer of the ESR-10. The athletes breathed hypoxic air for a duration of 5 minutes 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, Minn; accuracy claimed to be a standard deviation of ±2 units of percent saturation for saturations of 70% to 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 a subject could not reach the desired level of oxygen saturation, the F_IO_2 was reduced at the Body O_2 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°C to 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 each paddler's own kayak before 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 minutes later by 5 × 100-m sprints. Each athlete completed 4 of these sessions (2 pretreatment and 2 posttreatment) 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 to 110 W, which was increased by 20 W every 4 minutes until the athlete reached exhaustion. There was a 1-minute rest period between steps, when 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 (Vo_2max) was determined as the highest 30-second 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.[22] We used the Trend function in Microsoft Excel to fit straight lines to the pretreatment and posttreatment 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 4 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 each 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. Before the start of each test the subject had a 15-minute warm-up period. This consisted of 2 minutes of easy paddling, then 8 minutes of paddling at 70% of peak power, then 5 × 10-second efforts at 200% of peak power, performed every minute. The subject was then allowed 5 minutes to rest before the start of the race simulation. To ensure that 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 seconds followed by a 5-second 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[21] 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 15 seconds of passive recovery. Interval time and 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 venipuncture 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 pretreatment, 5 and 10 days midtreatment, and 3 days posttreatment.

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, post 1, and post 2 for each of the 2 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 2 pretests and 2 posttests), and additional within-subject variance for the first testing session (familiarization trial), for both posthypoxia 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 ± between-subject SDs. 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).[23] 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.[24] 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% to 1.5%.[25] 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 2 pretests) of the 10 subelite kayak athletes are shown in Table 1. In the second arm of the crossover 3 subjects pulled out of the study (2 hypoxia and 1 control).

Effects on Performance

Table 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 posttreatment 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 1. The most substantial change between conditions in sprint power

Table 1 Characteristics and Baseline Measures of the Subjects, Mean ± SD

Characteristic	Measure
Age (y)	23.2 ± 8.3
Height (cm)	180.1 ± 4.0
Body mass (kg)	81.2 ± 7.2
Best on-water 500-m time in previous year (s)	113.1 ± 5.3
Hemoglobin (g/L)	146.4 ± 5.7
Hematocrit (%)	43.8 ± 1.3
Ferritin (µg/L)	64.2 ± 42.4
Incremental step test	
peak power (W)	179 ± 26
lactate midpoint (W)	137 ± 23
power @ 90% HR_{max} (W)	139 ± 20
economy @ 70% VO_{2max} (W)	109 ± 20
peak VO_2 (L/min)	4.0 ± 0.5
peak lactate (mM)	10.8 ± 2.4
500-m simulation	
mean power (W)	239 ± 43
mean power (% peak aerobic power)	134.5 ± 8.0
time (s)	124.2 ± 6.8
mean power 0–10 s (W)	359 ± 76
peak lactate (mM)	12.0 ± 2.1
Repeat sprint test	
1st sprint power (W)	349 ± 81
1st sprint time (s)	22.1 ± 1.5
final sprint power (W)	239 ± 32
final sprint time (s)	25.4 ± 1.0
mean sprint power (W)	265 ± 42
mean sprint time (s)	24.6 ± 1.1
peak lactate (mM)	13.2 ± 2.5

occurred at sprints 2, 3, and 5. Lactate threshold and 500-m power demonstrated improvements, but these were unclear. At 10 days posttreatment, the effects on all performance measures were unclear. Nonetheless, there was still a strong trend toward improvement in measures of peak power, mean sprint power, and 500-m power. Not shown in Table 2 are the percentage effects for performance time in the 500-m simulation and repeat sprint test; these were all ≈0.38 of the percentage 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, first 10 seconds, 2.8%.

Table 2 Mean Changes in Performance at 3 Days and 10 Days Posthypoxia and Control and Chances That the True Difference in the Changes Is Substantial

		Change in Measure*			
	Posttest day	Intermittent hypoxia, mean ± SD	Control, mean ± SD	Difference; ± 90% CL†	Practical inference‡
Incremental step test					
peak power	3	8.2 ± 5.7	1.3 ± 5.7	6.8; ± 5.2	Benefit very likely
	10	5.8 ± 5.7	3.2 ± 5.7	3.5; ± 5.6	Unclear
lactate threshold	3	6.7 ± 8.1	3.1 ± 8.2	3.5; ± 7.0	Unclear
	10	5.3 ± 8.1	6.0 ± 8.2	–0.7; ± 7.5	Unclear
heart-rate profile	3	5.9 ± 6.4	5.0 ± 6.3	0.9; ± 5.6	Unclear
	10	5.2 ± 6.4	5.1 ± 6.3	0.1; ± 6.1	Unclear
Repeat sprint test					
mean repeat power	3	3.9 ± 6.4	–4.1 ± 7.6	8.3; ± 6.7	Benefit very likely
	10	3.1 ± 6.4	0.1± 7.6	3.0; ± 7.2	Unclear
500-m simulation					
500-m power	3	4.6 ± 5.5	2.2 ± 2.8	2.4; ± 4.1	Unclear
	10	6.0 ± 5.5	3.9 ± 2.8	2.2; ± 4.3	Unclear
mean power 0–10 s	3	6.1 ± 8.5	2.9 ± 6.2	3.1; ± 8.2	Unclear
	10	0.8 ± 8.5	9.3 ± 6.2	–7.8; ± 8.5	Unclear

*Units of change are percentages for all measures.

†±90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

‡Based on a smallest beneficial or harmful change in performance of 1%.

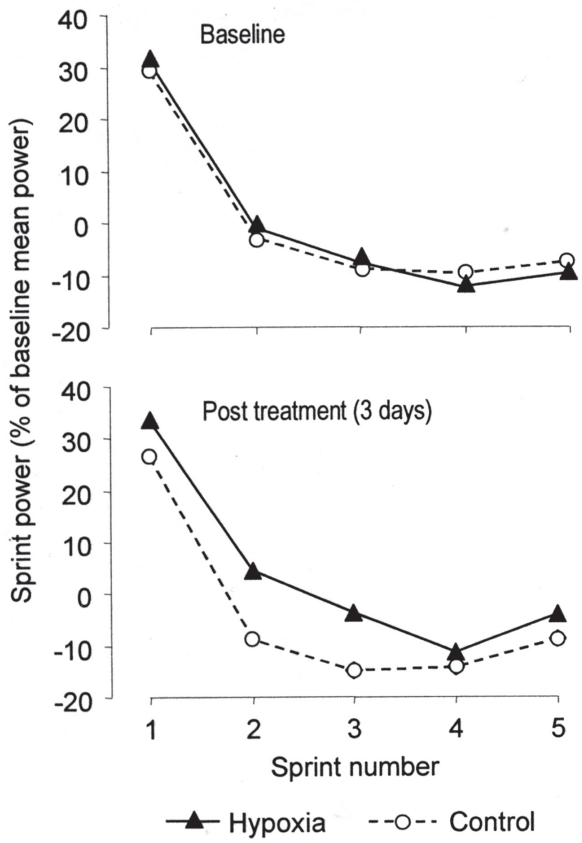

Figure 1 — Effect of intermittent hypoxic and control treatments on repeated 100-m sprint power at baseline and 3 days posttreatment. Power in each sprint is expressed as a percentage of the mean of all 5 baseline sprints.

Effects on Physiological Measures

Table 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 into the intervention and 3 days posttreatment. Although ferritin showed a trend of decrement, it was not substantially different between the control and hypoxic condition until 3 days posttreatment. Effects on other blood parameters assayed but not shown in Table 2 (erythrocyte sedimentation rate and white-blood-cell count) were unclear. The effect on the other physiological measures was less pronounced, with only 1 measure (peak aerobic power obtained during

Table 3 Mean Changes in Physiological Measures at 3 and 10 days Posthypoxia and Control and Chances That the True Difference in the Changes Is Substantial

		Change in Measure (%)			
	Posttest day*	Hypoxia, mean ± SD	Control, mean ± SD	Difference; ± 90% CL†	Qualitative inference‡
Incremental step test					
Vo_2max	3	–0.5 ± 6.5	–0.4 ± 5.8	–0.1; ± 5.2	Unclear
	10	2.9 ± 6.5	2.2 ± 5.8	0.7; ± 6.3	Unclear
exercise economy	3	1.7 ± 7.6	3.1 ± 6.2	–1.4; ± 6.4	Unclear
	10	2.7 ± 7.6	–5.7 ± 6.2	9.0; ± 12.6	Unclear
peak lactate	3	5 ± 15.5	6.1 ± 22.6	–0.7; ± 17	Unclear
	10	4.3 ± 15.5	–0.5 ± 22.6	3.0; ± 9.9	Unclear
Repeat sprint test					
peak lactate	3	1 ± 17	–5 ± 11	6; ± 14	Unclear
	10	–6 ± 17	–6 ± 11	–1; ± 15	Unclear
500-m time trial					
mean power (% peak aerobic power)	3	–3.7 ± 3.8	1.7 ± 7.1	–5.2; ± 4.5	Likely negative
	10	–0.4 ± 3.8	2.4 ± 7.1	–2.6; ± 5.0	Unclear

peak lactate	3	3.1 ± 18.3	8.3 ± 17	–4.8; ± 15	Unclear
	10	–6 ± 18	1 ± 17	–7; ± 16	Unclear
Blood parameters					
hemoglobin	–16[b]	0.4 ± 3.4	1 ± 2.5	–0.5; ± 1.9	Unclear
	–11[b]	1.9 ± 3.4	–2.0 ± 3.4	4.0; ± 2.1	Almost certainly positive
	3	1.8 ± 3.4	–1.7 ± 3.4	3.6; ± 3.2	Likely positive
hematocrit	–16[b]	–0.8 ± 3.1	0.8 ± 3.9	–1.5; ± 2.3	Unclear
	–11[b]	1.4 ± 3.1	–2.5 ± 3.1	4.1; ± 2.5	Almost certainly positive
	3	0.3 ± 3.1	–2.3 ± 3.1	2.7; ± 3.4	Unclear
ferritin	–16[b]	–5.1 ± 19	–0.4 ± 21	–4.7; ± 15	Unclear
	–11[b]	–6.8 ± 19	–8.9 ± 21	2.3; ± 15	Unclear
	3	–18.5 ± 19	0.3 ± 21	–19 ; ± 15	Likely negative

*Negative test days represent 5 and 10 days midintervention.

†±90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.

‡Based on a smallest substantial change of 1% for Vo_2max, economy, and mean power (% peak aerobic power, and 0.2 of the baseline between-subject SD for all other measures.

the 500-m time trial) showing a substantial reduction in the hypoxic condition at 3 days posttreatment.

The standard errors of measurement for physiological measures were Vo_2max, 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%.

Discussion

The major finding of this study is that intermittent hypoxic exposure for 15 days over a 3-week period substantially enhanced peak power and repeat sprint performance on a kayak ergometer in subelite kayak paddlers 3 days after the treatment period. In addition, there was a substantial increase in hemoglobin concentration and hematocrit and a substantial reduction in ferritin after the treatment. Effects on all other measures, including those representing anaerobic power and all measures at 10 days posttreatment, were unclear.

The lack of clarity for most measures was caused in part by 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[15] were ≈1% to 2%, whereas the errors were 2% to 5% in the present study. These differences in error could be a result of kayak ergometry being less reliable than running, which has been used to assess performance in similar studies.[15,19] The reduced reliability might 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[15,16,19] and might 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.[15] Furthermore, the level of intensity was individually monitored and controlled via pulse oximetry, which ensured that all our subjects received a similar stimulus. This approach was either not used or not possible in most studies. It is also likely that the greater magnitude partly results from 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.[26] 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 2, the overall effect is ≈3% to 4% 3 days postexposure and somewhat less by 10 days postexposure, which is similar to the results of Wood et al[15] and to some extent Hamlin and Hellemans.[16]

Our findings are more difficult to reconcile with those of Julian et al,[19] 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 might explain the contrasting findings. First, we monitored and adjusted the level of hypoxia individually via pulse oximeters to reach the target saturation, which progressively decreased every 1 to 2 days. In contrast, the subjects of Julian et al[19] maintained a preset level of hypoxia for 5 days, without frequent monitoring of individual oxygen saturations. In addition, the lowest saturation reached in the study of Julian et al was 82%, compared with 76% in the present study. Therefore, their subjects might have received a lesser hypoxic stimulus than ours did. Furthermore, the subjects of Julian et al, who were top-level competitors in individual endurance sports, might have been in a more highly trained state and might therefore have had less potential for performance enhancement than our national-level kayak paddlers.

The observed physiological changes after 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 posttreatment there was a substantial increase in hemoglobin concentration in the hypoxic condition. These changes suggest that an enhancement of the blood's oxygen-carrying capacity occurred and are consistent with changes in hematology after adaptation to intermittent hypoxia exposure in other studies,[16,18] including studies investigating the live-high train-low model.[1,27] The change in hemoglobin was accompanied by a substantial decrease in ferritin at 3 days posttreatment in the hypoxia condition. Because it is known that ferritin levels substantially decrease when humans move to high altitude,[28] 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 Vo_2max. Such changes might 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 Vo_2max was unaffected and that exercise economy improved. Improvements in economy of 3% to 6% have been observed after various hypoxic interventions with athletes.[9,10,29-31] 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.[8] Although the effect on economy was unclear in our study, improvements in this variable along with Vo_2max 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 after the hypoxic intervention, similar to that reported by Wood et al.[15] 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 their lactate thresholds.

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[4,29] and maximal accumulated oxygen deficit.[32] Although 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 measures of anaerobic power in the present study that could provide evidence in support of this trend were mean power in the first 10 seconds 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 after the incremental step test, 500-m time trial, and repeat sprint test. Unfortunately the changes in all these measures were unclear after 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. Motivation levels and effort were high, however, 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.[33] Therefore, it is unlikely that the nonblinding 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.

References

1. Levine BD, Stray-Gundersen J. "Living high—training low": effect of moderate-altitude acclimatization with low-altitude training on performance. *J Appl Physiol*. 1997;83:102-112.
2. Desplanches D, Hoppeler H, Linoissier MT, et al. Effects of training in normoxia and normobaric hypoxia on human muscle ultra-structure. *Pflugers Arch*. 1993;425:263-267.
3. Terrados N, Jansson E, Sylven C, Kaijser L. Is hypoxia a stimulus for synthesis of oxidative enzymes and myoglobin? *J Appl Physiol*. 1990;68:2369-2372.
4. Mizuno M, Juel C, Bro-Rasmussen T, et al. Limb skeletal muscle adaptation in athletes after training at altitude. *J Appl Physiol*. 1990;68:496-502.
5. Jensen K, Nielsen TS, Fiskastrand A. High-altitude training does not increase maximal oxygen uptake or work capacity at sea level in rowers. *Scand J Med Sci Sports*. 1993;3:252-262.
6. Adams WC, Bernauer EM, Dill DB, Bomar JB. Effects of equivalent sea level and altitude training on Vo_2max and running performance. *J Appl Physiol*. 1975;39:262-266.

7. Levine BD, Stray-Gundersen J, Duhaime G, Snell PG, Friedman DB. "Living high—training low": the effect of altitude acclimatization/normoxic training in trained runners [abstract]. *Med Sci Sports Exerc*. 1991;23:S25.
8. Gore CJ, Hopkins WG. Counterpoint: positive effects of intermittent hypoxia (live high—train low) on exercise performance are not mediated primarily by augmented red cell volume. *J Appl Physiol*. In press.
9. Katayama K, Matsuo H, Ishida K, Mori S, Miyamura M. Intermittent hypoxia improves endurance performance and submaximal exercise efficiency. *High Alt Med Biol*. 2003;4:291-304.
10. Katayama K, Sato K, Matsuo H, Ishida K, Iwasaki K, Miyamura M. Effect of intermittent hypoxia on oxygen uptake during submaximal exercise in endurance athletes. *Eur J Appl Physiol*. 2004;92:75-83.
11. Rodriguez FA, Murio J, Ventura JL. Effects of intermittent hypobaric hypoxia and altitude training on physiological and performance parameters in swimmers [abstract]. *Med Sci Sports Exerc*. 2003;35:S115.
12. Rodriguez FA, Casas H, Casas M, et al. Intermittent hypobaric hypoxia stimulates erythropoiesis and improves aerobic capacity. *Med Sci Sports Exerc*. 1999;31:264-268.
13. Casas M, Casas H, Pages T, et al. Intermittent hypobaric hypoxia induces altitude acclimation and improves the lactate threshold. *Aviat Space Environ Med*. 2000;71:125-130.
14. Rodriguez FA, Ventura JA, Casas M, et al. Erythropoietin acute reaction and haematological adaptations to short, intermittent hypobaric hypoxia. *Eur J Appl Physiol*. 2000;82:170-177.
15. Wood MR, Dowson MN, Hopkins WG. Running performance after adaptation to acutely intermittent hypoxia. *Eur J Sport Sci*. In press.
16. Hamlin MJ, Hellemans J. Effect of intermittent normobaric hypoxic exposure at rest on hematological, physiological and performance parameters in multi-sport athletes. *J Sports Sci*. In press.
17. Latyshkevich LA, Zakusylo MP, Shakhlina LH. The efficiency of interval hypoxic training in volley-ball. *Hypoxia Med J*. 1993;2:33-35.
18. Hellemans J. Intermittent hypoxic training: a pilot study. In: *Proceedings of the Second Annual International Altitude Training Symposium*. Flagstaff, Ariz; 1999:145-154.
19. Julian C, Gore C, Wilber R, et al. Intermittent normobaric hypoxia does not alter performance or erythropoietic markers in highly trained distance runners. *J Appl Physiol*. 2004;96:1800-1807.
20. Clark SA, Dixon J, Gore CJ, Martin DT, Hahn AG. Intermittent hypoxia fails to improve rowing ergometer performance [abstract]. In: *Fifth IOC World Congress on Sport Sciences*. Canberra: Sports Medicine Australia, 1999:85.
21. Bishop D, Bonetti D, Dawson B. The influence of pacing strategy on VO_2 and supramaximal kayak performance. *Med Sci Sports Exerc*. 2003;34:1041-1047.
22. Beaver WL, Wasserman K, Whipp BJ. Improved detection of lactate threshold during exercise using a log-log transformation. *J Appl Physiol*. 1985;59:1936-1940.
23. Batterham AM, Hopkins WG. Making meaningful inferences about magnitudes. *Int J Sports Physiol Perform*. 2006;1:50-57.
24. Hopkins WG, Hawley JA, Burke LM. Design and analysis of research on sport performance enhancement. *Med Sci Sports Exerc*. 1999;31:472-485.
25. Hopkins WG. How to interpret changes in an athletic performance test. *Sportscience*. 2004;8:1-7.
26. Higgins JPT, Green S. *Cochrane Handbook for Systematic Reviews of Interventions 4.2.5*. Chichester, UK: Wiley; 2005. Available at: http://www.cochrane.org/resources/handbook/hbook.htm. Accessed December 23, 2005.

27. Stray-Gundersen J, Chapman RF, Levine BD. "Living high—training low": altitude training improves sea level performance in male and female elite runners. *J Appl Physiol*. 2001;91:1113-1120.
28. Richalet JP, Souberbielle JC, Antezana AM, et al. Control of erythropoiesis in humans during prolonged exposure to the altitude of 6,542 m. *Am J Physiol*. 1994;266:R756-R764.
29. Gore CJ, Hahn AG, Aughey RJ, et al. Live high:train low increases muscle buffer capacity and submaximal cycling efficiency. *Acta Physiol Scand*. 2001;173:275-286.
30. Green HJ, Roy B, Hughson R, et al. Increases in submaximal cycling efficiency mediated by altitude acclimatization. *J Appl Physiol*. 2000;89:1189-1197.
31. Saunders PU, Telford RD, Pyne DB, et al. Improved running economy in elite runners after 20 days of moderate simulated altitude exposure. *J Appl Physiol*. 2004;96:931-937.
32. Roberts AD, Clark SA, Townsend NE, Anderson ME, Gore CJ, Hahn AG. Changes in performance, maximal oxygen uptake and maximal accumulated oxygen deficit after 5, 10 and 15 days of live high:train low altitude exposure. *Eur J Appl Physiol*. 2003;88:390-395.
33. Batterham AM, Hopkins WG. A decision tree for controlled trials. *Sportscience*. 2005;9:33-39.