

**Running Performance after Acutely Intermittent
Simulated Altitude Exposure**

Matthew R. Wood, Martin N. Dowson, and Will G. Hopkins

Sport and Recreation, Auckland University of Technology, Auckland, NZ

Running performance and altitude exposure

Address for correspondence:

Will G Hopkins, PhD

Health Science/Sport and Recreation

Auckland University of Technology

Private Bag 92006, Auckland 1020, New Zealand

Direct dial +64 9 917 9793, Fax +64 9 917 9960, Cell +64 27 427 2518

will@clear.net.nz

About the Authors

Matthew Wood lectures exercise physiology at Auckland University of Technology. He also works as a consultant exercise scientist. His current research focuses on enhancement of athletic performance using simulated altitude exposure.

Martin Dowson is a senior lecturer in exercise physiology and applied use of sports science for elite performance at Auckland University of Technology. His main research interest is high-intensity team sports. He is a consultant for rugby union, hockey and soccer at national level.

Will Hopkins is a professor in exercise science at Auckland University of Technology and a fellow of the American College in Sports Medicine. His main interests are factors affecting athletic performance and statistical issues in the design and analysis of studies in exercise and sport science.

Running Performance after Acutely Intermittent Simulated Altitude Exposure

Matthew R. Wood, Martin N. Dowson, and Will G. Hopkins

Abstract

To quantify the effects of acutely intermittent simulated altitude exposure on running performance, we randomized 29 trained male hockey and soccer players in double-blind fashion to altitude or placebo groups for 15 d of daily use of a functional or placebo hypoxic re-breathing device. 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 with pulse oximeters and progressively reduced in the altitude group (90% on Day 1, 77% on Day 15; equivalent altitudes ~3600-6000 m). Performance tests were an incremental run to maximum speed followed by six maximal-effort running sprints; tests were performed 1 d before, 3 d after, and 12 d after the 15-d treatment. 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 relatively faster by 1.5% in the first sprint through 6.9% in the last ($\pm 1.9\%$); there were also substantial reductions in exercise lactate concentration and resting and exercise heart rate. Large effects on performance were still present 9 d later. Thus, acutely intermittent simulated altitude exposure substantially improves high-intensity running performance.

Key Words: hockey, hypoxia, re-breathing, reliability, soccer

Key Points:

1. Exposure to altitude simulated with a rebreathing device for an hour a day for 15 days enhanced running performance.
2. Three days after exposure, the enhancement of endurance speed was ~2%; in repeated sprints the enhancement was ~1-7%.
3. Substantial effects were still present 12 d after exposure.
4. These enhancements should be useful for athletes in high-intensity team sports.

Introduction

Adaptation to the reduced availability of oxygen at altitude has the potential to benefit endurance performance at sea level (21). Effects on such performance in controlled studies of athletes living and training at altitude have generally been unclear, possibly because the shortage of oxygen at altitude causes a reduction in intensity of training that offsets any beneficial adaptations (21). To maintain the benefits of high-intensity training, researchers devised the live-high train-low approach to altitude exposure, whereby athletes live at moderate altitude and train closer to sea level. This approach, which has produced gains in endurance running speed of 1-2% (2,22,30), has stimulated interest in the effects of exposing athletes to simulated rather than real altitude while they continue their normal training at sea level.

Studies of the effects of simulated altitude on performance have made use of hypobaric chambers or nitrogen dilution to reduce the availability of oxygen. In a study using hypobaric chambers, athletes exposed to short periods (3 h.d⁻¹) of low pressure equivalent to moderately high altitude (4000-5500 m) for two weeks improved their 200-m swimming performance time by ~1% (26). Nitrogen dilution can be achieved with stored nitrogen or a filtration machine to separate oxygen and nitrogen. With this approach, athletes typically reside or sleep for 8-18 hours per day at moderate levels of altitude (2000-3000 m) and achieve gains in performance equivalent to ~0.6-1.5% in mean power (11,15,23). Smaller volumes of nitrogen-diluted air can also be pumped to a mask for use with periods of exposure that, for obvious practical reasons, have to be relatively short. Exposures are at the equivalent of high altitudes (3000-6000 m) for 5-7 min alternating with similar periods of normal air for a total of 1-2 hours per day for several weeks. Some researchers using this approach in the former Soviet Union, where it was known as interval hypoxic training, made claims for unrealistic benefits on performance (17,28); others have found little or no benefit (3,16,19).

Rebreathing devices can also be used to produce nitrogen-diluted air. The devices incorporate a chemical absorbent to prevent accumulation of expired carbon dioxide, which, by stimulating ventilation, would otherwise prevent development of hypoxia. Research with these devices has been limited apparently to a few studies on ventilatory adaptations to hypoxia (29). The main aim of the present study was therefore to investigate the effects of altitude simulated with such a device on exercise performance.

Traditionally researchers have investigated the effects of altitude exposure on endurance performance, presumably because they expected the physiological adaptations arising from the shortage of oxygen to affect primarily aerobic power. However, there is tentative evidence that altitude can benefit sprinting or other short-term high-intensity performance that requires a substantial contribution from anaerobic mechanisms. In an uncontrolled study by Fornasiero et al. (7), the mean power output of 3 sets of six maximal 15 s cycle sprints increased by 9.6% in national road cyclists exposed to 7 d living and training at 1600 m followed by 10 d residence in a nitrogen house at the equivalent of 2700 m. Gains in performance of a single shuttle sprint have been less impressive: Latyshkevich et al. (19) found a 1.0% improvement in 91-m running shuttle sprint speed following 24 d of acute intermittent altitude exposure (ending with ~10% oxygen), and in a similar study from the same laboratory, sprint speed apparently changed little following 14 d of exposure (28). If the benefit for repeated sprinting can be confirmed in a controlled trial, altitude exposure would likely enhance performance of team-sport athletes, who make many repeated high-intensity sprints in the course of a game. Thus the present study was also designed to evaluate the effects of acute intermittent simulated high-altitude exposure on repeated sprint performance and endurance performance in trained team-sport athletes.

Methods

Study Design

The study was a randomized controlled trial in which subjects in two cohorts were randomized to simulated altitude and placebo groups. Subjects did not know which group they had been assigned to, and almost all performance tests were administered by assistants who were blind to the assignment. The design was therefore effectively double-blind. Subjects performed all exercise tests on four occasions: a familiarization trial, a baseline trial one week later on the day before the beginning of daily simulated altitude or placebo exposures, and trials on Days 3 and 12 after the completion of the exposures. The altitude group received 1 h of progressive intermittent simulated altitude exposure for 15 consecutive days, while the control group received placebo exposure. A haematological profile via flow cytometry was performed 1 day before and after altitude exposure. Measures of blood acid-base status were taken immediately before and after the first and last exposure sessions only for the first cohort of subjects (owing to limited financial support for this project).

Subjects

The first cohort consisted of 20 national, open provincial and age-group provincial male hockey players recruited by contact with coaches. Owing to unforeseen team commitments, drop-outs soon after exposures began left only 4 in the placebo group and 7 in the altitude group. The second cohort consisted of 18 premier club soccer players randomized to give near equal final numbers in the groups. All subjects in the second cohort completed the study. All subjects in both cohorts were in a trained state at the end of a pre-competition training phase prior to international or representative games. All gave voluntary informed consent as required by the institutional ethics committee.

Training and Diet

Subjects were instructed to maintain their existing fitness training beginning two weeks prior to baseline testing and ending with the last performance trial. Team training involved game-specific drills and practice games. Subjects were instructed to refrain from strenuous physical activity for 24 hours prior to the performance trials. At the familiarization trial they were also instructed about appropriate dietary intake (high carbohydrate) in the 24 hours prior to subsequent performance tests. At the baseline trial, each subject recorded dietary intake in the previous 24 hours on a simple structured sheet. The subject was given a photocopy of the sheet and was instructed to use it to replicate the intake before each subsequent performance test.

In accord with previous studies of altitude exposure, iron supplementation was included to facilitate any increase in red-cell production. An assay for ferritin was therefore included in the blood test 1 d before exposure to determine if any subjects should not receive the supplement. One subject, who apparently had a genetically high ferritin concentration, was excluded; the remainder took one 18-mg carbonyl iron tablet (Douglas Pharmaceuticals, Auckland, NZ) every second day of the exposure period.

Subjects completed a daily questionnaire, consisting of training, dietary, sleep and general health and well-being questions during the period of exposure. After the completion of the final performance trial, subjects were asked whether they thought they had received altitude treatment or placebo.

Altitude Treatment

Altitude was simulated with a re-breathing device (ALTO₂Lab, Douglas Pharmaceuticals, Auckland, NZ). The device consisted of a breathing tube attached to an open-ended silo containing a sodalime (Spherasorb, Intersurgical Ltd, Wokingham, UK) to absorb carbon dioxide (CO₂). Additional foam-filled silos were added to increase respiratory dead space and thereby increase the altitude stimulus. Subjects wore a nose clip to prevent nasal breathing.

The placebo device was identical in construction but did not include the absorbent for CO₂. The build-up of CO₂ and the warmth of the expired gas in the dead space apparently persuaded the subjects in the placebo group that they were in the altitude group: after the last performance trial, all subjects reported that they thought they had been in the altitude group.

In the treatment sessions, subjects alternated 6 min of breathing through the device with 4 min of breathing air six times, for a total of 60 min. Peripheral oxygen saturation was monitored individually with pulse oximeters provided with each ALTO₂Lab device (Sport-Stat, Nonin Medical, Minneapolis, MN; accuracy claimed to be a standard deviation of ± 2 units of percent saturation for saturations of 70-100%). Saturation was reduced progressively in the altitude group, starting at ~90% on Day 1 and finishing at ~77% on Day 15. These saturations would occur with adaptation to altitudes of approximately 3600 and 6000 m respectively (<http://www.high-altitude-medicine.com/SaO2-table.html>, from Reference (9)). Oxygen saturation was recorded after each 6 min bout. A custom-designed screen was placed in front of subjects during treatment sessions to prevent them from seeing the oximeter and re-breathing device.

Exercise Performance Tests

Subjects were instructed to eat high carbohydrate meals on the evening before and on the day of each test. They performed the tests on a wooden surface in a covered stadium in performance-matched pairs. Testing occurred at approximately the same time of day for a given pair of subjects. Each testing session consisted of a warm-up, an incremental running test (~10-14 min), a rest of 30 min, another warm-up, and the repeated-sprint test (3 min). The warm-ups consisted of a jog for 3 min followed by 2 min of dynamic stretches.

The incremental running test was a 20-m shuttle run (20) modified by inclusion of 1-min rests every 3 min to allow sampling of finger-prick blood for determination of lactate concentration.

Subjects maintained a constant running speed during each 3-min stage by reaching the 20-m line in time with a pre-recorded audible beep. The running speed started at 11 km.h⁻¹ and increased by 1 km.h⁻¹ for each stage until subjects could not reach the line in time with the beep on two consecutive shuttles. Subjects then walked slowly for a further 4 min, when a final blood sample was taken for measurement of lactate. Time to maximum effort was converted by interpolation to the running speed equivalent to the speed at exhaustion if the speed had increased linearly during each stage. Heart rate (Polar A1, Polar Electro, Kempele, Finland) and perceived exertion (Reference (1): 15-point linear scale; minimum effort=6, maximum=20) were also recorded at the beginning of each 1-min rest period and as soon as the subjects finished the test. Mean and maximum values of heart rate and perceived exertion during the test were derived for each subject for further analysis. For each subject we also derived a new measure representing horizontal shift of the lactate profile, as follows: values of lactate concentration were plotted against running speed on a single set of axes for the baseline and post tests; points for each test were connected by smooth curves by choosing the appropriate XY (Scatter) chart type in Microsoft Excel; horizontal lines were drawn at either 0.5-mM or 1-mM intervals of lactate concentration to intersect all three curves; the change in speed from baseline to each post test for each lactate concentration was then expressed as a percent of baseline, and all such changes were averaged for each post test. The resulting measure is analogous to change in 4-mM lactate-threshold speed, but it is the average of such changes over a range of lactate concentrations. We repeated this process for plots of heart-rate against running speed, using horizontal lines drawn at 2.5-min⁻¹ or 5-min⁻¹ intervals.

The repeated sprint test consisted of six repeated maximal effort sprints, preceded by a sub-maximal familiarization sprint. Times for each sprint were recorded using two sets of electronic speed-timing lights (Speed-Light, Swift Performance Equipment, Goonellabah, Australia). Subjects began each sprint 30 cm behind the first set of timing lights. Each sprint involved running three shuttle distances (5, 10 and 20 m, each there and back, for a total of 70 m) at maximum effort. Each of the six sprints began every 30 s. Heart rate and perceived exertion were recorded at rest and immediately after the completion of each sprint. Mean and maximum values of heart rate and perceived exertion during the test were derived for each subject for further analysis. Blood lactate was assayed 4 min after the final sprint. For each subject time for each sprint was plotted against sprint number. A strong linear trend was apparent (range in Pearson

correlation coefficients, 0.87-1.00), so for further analysis we derived times for the first and last sprint predicted from the line of best fit for each subject, along with the mean time for the six sprints.

Blood measurements

The index finger of the subject's right hand was used as the sample site for blood lactate and acid-base status. The skin was punctured with a sterile lancet and blood was drawn into a capillary tube. Blood was transferred to the strip of a portable lactate analyser (Lactate Pro, Kyoto, Japan) or to the sampling well of a blood analyser (PCA, i-STAT, East Windsor, NJ) for measurement of blood pH and bicarbonate.

Subjects visited an independent professional testing laboratory (Diagnostic Medlab, Auckland, NZ) for determination of hematocrit, haemoglobin, and a white-cell count. Subjects were seated for several minutes with their arm at heart level while a vene-puncture blood sample was drawn from an antecubital vein of the left arm with use of a tourniquet. A 200 μ L blood sample was analysed using a haematology analyser (XE-2100, Sysmex, Kobe, Japan).

Statistics

Simple group statistics are shown as means \pm between-subject standard deviations. Confidence limits for the true mean values of effects were estimated with a spreadsheet (12) via the unequal-variances t statistic computed for change scores between baseline and post-exposure tests in the two groups. Raw values of change scores were used in the analyses only for heart rate; otherwise, each subject's change score was expressed as a percent of baseline score via analysis of log-transformed values, to reduce bias arising from non-uniformity of error.

The spreadsheet also computes chances that the true effects are substantial, when a value for the smallest worthwhile change is entered. For sprint speeds in the repeated-sprint test, we chose 0.8% as the smallest change that can affect gaining possession of the ball in a team sport (24). We do not know how a change in maximum speed or profile speeds in the incremental run would affect a team athlete's performance, so we chose instead 0.5%; this value is approximately the smallest that would affect a top track athlete's chances of a medal, on the assumption that these measures are proportional to mean power in competitive endurance events (13,14). To estimate chances that effects were substantial for the physiological variables, we expressed the effect sizes as fractions or multiples of the baseline between-subject standard deviation (for heart rates) or

coefficient of variation (for percent effects with all other variables), chose the smallest worthwhile value of this statistic as 0.2 (5), and assumed the sample size was sufficiently large that uncertainty in the standard deviation would not affect the estimates of the chances provided by the spreadsheet. We also interpreted larger values of this statistic as follows: ≥ 0.2 and < 0.6 is small, ≥ 0.6 and < 1.2 is moderate, ≥ 1.2 and < 2.0 is large, and ≥ 2.0 is very large (12).

For the measures of performance, errors of measurement and individual responses were also estimated with the spreadsheet and checked 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 trial (pre, post1, post2), group (altitude, placebo) and their interaction. The random effects were subject variance, residual variance, and additional within-subject variance for each of the two post-exposure trials for the altitude group. The square root of the residual variance is a standard deviation equivalent to within-subject error of measurement in a reliability study, and the square root of the additional within-subject variance is a standard deviation representing typical between-subject variation in the effect of altitude relative to placebo. The analyses were performed with an option that allowed estimates of variance and their confidence limits to be negative. Negative variances were transformed to negative standard deviations by a change of sign before and after taking the square root.

Results

Subject characteristics

The characteristics and baseline exercise performance of the 11 hockey and 18 soccer players are shown in Table 1. Differences between the groups in terms of fractions of a between-subject standard deviation were mostly small or moderate.

Table 1 Characteristics and baseline measures of performance of hockey and soccer players in the two training groups

	Placebo (n=14)	Altitude (n=15)
Sample size by competitive level		
National representative	3	3
Regional representative	6	8
Premier Club	5	4
Age (y)	23.4 ± 3.5	24.2 ± 3.8
Weight (kg)	75.7 ± 3.2	77.4 ± 3.5
Height (m)	1.69 ± 0.14	1.73 ± 0.10
Training ^a (h.wk ⁻¹)	4.1 ± 0.8	4.2 ± 1.2
Incremental shuttle run		
Maximum speed (km.h ⁻¹)	14.0 ± 0.4	13.8 ± 0.5
Repeated 70-m shuttle sprints		
Sprint 1 (s)	15.5 ± 0.9	15.2 ± 0.6
Sprint 6 (s)	19.5 ± 1.3	19.1 ± 0.5

Data other than sample sizes are mean ± between-subject standard deviation.

^aPredominantly high-intensity interval training and practice games.

Exposure Sessions

The oxygen-hemoglobin saturation in the altitude group at the end of the 6-min periods of exposure in the first exposure session was 89.8 ± 2.5 percent (mean of the six periods and mean of the between-subject standard deviations for each period). On Days 8 and 15 the saturations were 80.6 ± 1.8 and 76.6 ± 1.1 percent respectively. The numbers of spacers required to produce these saturations were 1.0 ± 0.4 , 2.5 ± 0.4 and 3.8 ± 0.4 respectively. A similar progression in spacers was used with the control group (1.2 ± 0.6 , 2.2 ± 0.6 , and 3.3 ± 0.6), but their saturations were stable throughout the period of exposure (97.2 ± 0.9 , 97.4 ± 0.8 , 97.4 ± 0.9 percent on Days 1, 8 and 15 respectively).

Effects on Performance

Table 2 shows the mean changes in the performance tests for altitude and placebo groups, and statistics for the difference in the changes. All observed effects of altitude on performance represent substantial improvements in performance at 3 and 12 d post exposure, and for most effects the true values were almost certain to be substantial.

Table 2 Mean changes in performance at 3 d and 12 d post altitude and placebo exposures, and chances that the true difference in the changes is substantial.

	Post-test day	Change in performance (%)			Chance that true difference is substantial ^a	
		Altitude	Placebo	Difference; $\pm 90\%CL$	%	Qualitative
Incremental shuttle run						
Lactate profile speed	3	4.4	0.4	4.0; ± 1.9	99.8	Almost certain
	12	3.3	-0.4	3.7; ± 1.8	99.8	Almost certain
Heart rate profile speed	3	2.4	-0.4	2.8; ± 1.7	98	Very likely
	12	1.8	0.8	1.1; ± 2.1	68	Possible
Maximum speed	3	1.8	-0.3	2.0; ± 0.5	>99.9	Almost certain
	12	1.4	-0.1	1.5; ± 0.4	>99.9	Almost certain
Repeated-sprint shuttle run						
First sprint time	3	-1.9	-0.4	-1.5; ± 1.3	91	Likely
	12	-0.8	0.2	-1.0; ± 2.0	67	Possible
Last sprint time	3	-7.5	-0.6	-6.9; ± 1.6	>99.9	Almost certain
	12	-6.2	0.0	-6.1; ± 1.3	>99.9	Almost certain
Total sprint time	3	-5.1	-0.5	-4.6; ± 1.1	>99.9	Almost certain
	12	-3.8	0.1	-3.9; ± 1.2	>99.9	Almost certain

^a *Substantial* is an improvement in performance of >0.5% for measures in the shuttle run and >0.8% for sprints.

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

The observed first sprint was faster than the predicted first sprint on each test day (by 1.4-1.6%), reflecting relatively greater effort in the first sprint. There was a similar relatively greater effort on the last sprint (by 1.6-1.8%). The effects of altitude exposure on these observed first and last sprints were larger by Day 3 post exposure (2.5% and 7.3% respectively, both almost

certainly substantial) than those on the predicted sprints shown in Table 2, but by Day 12 there was little difference between the effects on the observed and predicted sprints.

Mean perceived exertion fell by 0.4-0.7 units of the 15-point scale in the altitude group relative to the placebo group in both performance tests on Days 3 and 12 post exposure relative to baseline (90% confidence limits between ± 0.4 and ± 0.7). Means of maximum perceived exertion in each test at each time point were between 19.5 and 20, and there were no substantial differences in the change following exposure.

Standard deviations representing observed individual responses in maximal-effort performance were positive only for maximum speed in the incremental run (0.6%) and for time in the last sprint (0.9%), both at 3 d post exposure. The variation between individuals represented by these values was small relative to the mean effect of altitude shown in Table 2. The uncertainty (90% confidence limits $\sim \pm 0.5$ -1.5%) in these and the other estimates of individual responses allows for at most modest individual responses for all the maximal-effort measures, relative to the mean effects. In contrast, standard deviations representing individual responses in the submaximal measures of performance (lactate and heart-rate profile speeds) were substantial at 3 and 12 d (1.6-2.8%), although the individual responses were substantial over the 90% confidence interval ($\sim \pm 1.5$ -2.5%) only for the lactate profile.

Observed standard errors of measurement for measures of performance were: maximum incremental speed, 0.4%; lactate profile speed, 1.0%; heart-rate profile speed, 2.0%; sprint total time, 1.5%; first sprint time, 2.1%.; last sprint time, 1.8% The 90% confidence limits for the true error were $\times/\div 1.35$ for the lactate profile speed and $\sim \times/\div 1.2$ for the other errors.

Effects on Physiological Measures

Effects of altitude exposure on physiological measures (Table 3) were not as clear as the effects on performance measures. Heart rate before the first performance test (the incremental run) and mean heart rate in both tests fell by 2-3 min^{-1} in the altitude relative to the placebo group on Day 3 after exposure and by 1-2 min^{-1} on Day 12. Effects on maximum heart rate were trivial in the repeated sprints, but there was a tendency for altitude to produce a $\sim 2 \text{ min}^{-1}$ fall in the incremental run (data not shown). Altitude produced a clear fall in maximum lactate after the incremental run, but a fall was unlikely after the repeated sprints. Substantial increases resulting from altitude exposure were possible for hemoglobin concentration and hematocrit and likely for white-cell count.

Table 3 Mean changes in physiological measures in the performance and blood tests after altitude and placebo exposures, and chances that the true difference in the changes is substantial

	Post-test day	Change in measure ^a			Chance that true difference is substantial ^b	
		Altitude	Placebo	Difference; $\pm 90\% \text{CL}$	%	Qualitative
Incremental shuttle run						
Pre-test heart rate (min^{-1})	3	-1.9	0.5	-2.4; ± 2.0	88	Likely
	12	-2.5	-0.7	-1.8; ± 1.3	84	Likely
Mean heart rate (min^{-1})	3	-3.2	0.1	-3.2; ± 2.5	92	Likely
	12	-2.1	-1.2	-0.9; ± 2.2	43	Possibly not
Maximum lactate (%)	3	-12.2	-0.4	-11.8; ± 6.0	99	Very likely
	12	-12.3	-3.1	-9.2; ± 7.8	89	Likely
Repeated-sprint shuttle run						
Pre-test heart rate (min^{-1})	3	-0.3	-0.1	-0.2; ± 1.8	18	Unlikely
	12	-0.2	0.2	-0.4; ± 1.3	15	Unlikely
Mean heart rate	3	-3.1	0.1	-3.2; ± 1.9	96	Very likely

(min ⁻¹)	12	-2.9	-0.3	-2.6; ±2.0	89	Likely
Maximum lactate (%)	3	-1.1	-2.6	1.5; ±2.4	6	Unlikely
	12	-0.9	-1.4	0.5; ±4.9	16	Unlikely
Blood parameters						
Hemoglobin (%)	1	1.9	0.7	1.2; ±1.9	52	Possible
Hematocrit (%)	1	1.2	-0.8	2.0; ±2.7	71	Possible
White cells (%)	1	13.0	-0.5	13.4; ±9.8	95	Likely

^a Units of change are min⁻¹ for heart rates and % for all other measures.

^b Chances for a substantial decrease in heart rate, decrease in lactate, and increase in blood parameters, where *substantial* is 0.2 of the baseline between-subject standard deviation for each measure.

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

Some effects of exposure on blood pH during and between exposure sessions were large, but the sample size was too small for the effects to be particularly clear. Blood became relatively more acidic in the placebo group during the first exposure session (difference in change in pH of 0.029 pH units; 90% confidence limits ±0.026). On Day 15, pre-exposure pH was relatively more acidic in the placebo group (0.050; ±0.049) and there was little difference between the groups in the change that occurred during the session (-0.004; ±0.044). Changes in blood bicarbonate concentration paralleled these changes in pH but were small-moderate (~1 mmol.L⁻¹) and less clear.

Effects on Training

Figure 1 shows the time course of the rating of training on the day before each day of altitude or placebo exposure. There was a decline in quality in the altitude group relative to the placebo group that was greatest on the fourth day of exposure, when it represented a large effect (1.5 between-subject pre-exposure standard deviations). By Day 7 of exposure the altitude group was reporting better training quality than the placebo group, and by the last day of exposure the difference had increased to a very large magnitude (2.6 standard deviations). The true values of the differences between groups (changes relative to Day 1) on Day 4 and 15 and on most other days were almost certainly substantial.

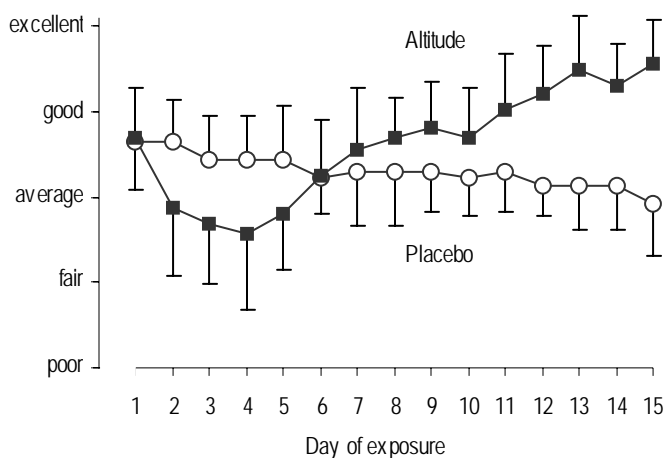


Figure 1–Retrospective rating of training quality for each day preceding the day of altitude and placebo exposure. Data are means and standard deviations for the two groups.

There was little change in the volume of training reported by the two groups during the period of exposure (altitude, 4.4 ± 1.2 h.wk⁻¹; placebo 4.0 ± 0.7 h.wk⁻¹) compared with baseline values shown in Table 1. The change was slightly greater in the second compared with the first week of exposure (data not shown). There was little difference in the reported hours of sleep (means of daily means and standard deviations: altitude, 7.8 ± 1.5 h.d⁻¹; placebo 7.8 ± 1.2 h.d⁻¹).

Discussion

The major finding in this study is that acute intermittent simulated high altitude exposure for 15 d considerably enhanced incremental-run and repeated-sprint performance for at least 12 d following the exposure. In relation to smallest worthwhile effects, the enhancements were almost certainly beneficial for a team-sport or track athlete. This high level of certainty is due to several factors: reasonably good sample size, small error of measurement for performance, small or negligible individual differences in the response to altitude, and large observed magnitude of the effect.

Our sample size of 15 in the altitude group and 14 in the placebo group was larger than that in any other published study of altitude exposure (typically 10 per group). Only in the classic study of Levine et al. (22) was the total sample size larger (39), but the subjects in that study were divided equally into a control and two different altitude groups.

The errors of measurement are amongst the smallest in the literature on reliability of performance (14). The following aspects of our study could be responsible for the small test-retest errors: the athletes were accustomed to performing similar if not identical performance tests as part of their usual battery of team fitness testing; they performed a complete familiarization trial before the baseline trial; they attempted to reproduce their day-to-day training and diet throughout the study; and they performed the tests in performance-matched pairs to maintain a high level of motivation. The latter aspect would not account for the small magnitude of error in our submaximal measures of performance, but it may be the crucial difference between our study and almost all studies of the reliability of measures of maximal performance.

Individual differences in the performance response to altitude were small or negligible for the maximal measures of performance, probably because we modified each subject's rebreathing device throughout the 15 d of exposure to produce virtually the same progressive decrease in oxygen-hemoglobin saturation. This explanation is, of course, predicated on the reasonable assumption that oxygen-hemoglobin saturation is closely related to the stimulus for adaptation to altitude. The extent of individual responses to altitude documented in other studies is unclear, because the researchers identified responders without taking into account variation in performance arising from error of measurement (2,30). Nevertheless, there appears to be more variation in the response to altitude in these studies, possibly because the real altitude to which

the athletes were exposed produced a wider variation in oxygen-hemoglobin saturation than occurred in our study of simulated altitude.

Finally, even the smallest effects on performance in our study were larger than the smallest beneficial effect for athletes, and it was only for the first of the repeated sprints that the benefit was unclear. All other effects were large enough for the benefit to be almost certain. Furthermore, these gains in performance were due to enhancements in the altitude group; there was little change in performance in the control group, and we consider it most unlikely that the control treatment (one hour of hypercapnic hyperventilation each day) somehow prevented an improvement of performance that would otherwise have occurred in the control group at this stage of their training.

Overall, the effects of altitude exposure on performance in our study are at the high end of the range of magnitudes in the literature. The effect on maximum speed in the incremental running test was generally larger than effects on measures of maximal endurance performance in other studies (2,6,11,16,22,30), although the effects are comparable when uncertainty in the estimates is taken into account. The effect on the first of the repeated sprints was also larger than, but comparable with, performance of a single sprint in other studies (19,28). The last of the repeated sprints showed a greater enhancement than all other measures of performance in our study, but it was somewhat less than the effect on repeated sprints in the uncontrolled study of Fornisiero et al. (7). A greater performance enhancement (~8%) has also been reported for lactate-related maximal running speeds in the study of Nummela and Rusko (23), although it is difficult to compare their measure with our lactate-profile and heart-rate profile speeds. In studies with comparable submaximal measures (3,6,8,15,25), the effects of altitude exposure were either smaller or negligible.

In comparing our findings with those in other studies, it is important to realize that subjects were blind to the identity of control and altitude treatments in only one other study (16). The magnitude of the effect of altitude on performance in the unblinded studies could therefore be inflated by the placebo effect, which is as high as ~4% for mean power in an endurance test lasting about an hour (4). The placebo effect in the shorter tests used in altitude studies is probably smaller, but it could easily account for a substantial proportion of the observed effects in these studies.

Readers with insight into the adaptations that occur with altitude exposure may be skeptical about the magnitudes of the effects on performance that we have observed, given that acute intermittent simulated altitude exposure for only 15 days represents a much smaller total exposure to hypoxia than that of more traditional live-high train-low real or simulated approaches. Indeed, our own skepticism about the effects in the first cohort of subjects was partly the reason we investigated the reproducibility of the effects by studying a second cohort. We offer the following observations as possible explanations for our findings.

First, several aspects of our method of simulating altitude exposure could have contributed to performance enhancement. Although the total period in hypoxia was short, the brief waves of hypoxia equivalent to moderately high altitude may have been a more effective stimulus than longer periods at the equivalent of a lower altitude. The relatively rapid "ascent" to altitude in the first week may also have added to the effectiveness of the stimulus. Furthermore, the individualized monitoring of oxygen-hemoglobin desaturation ensured that all our subjects received a similar stimulus, whereas individual respiratory adaptations to hypoxia in other studies may have lessened the stimulus for some subjects. There is also a possibility that our rebreathing device, by virtue of its respiratory dead space or imperfect functioning of CO₂ absorbent, produced a relative hypercapnia or other changes in acid-base status that in some manner augmented the adaptive response to the hypoxic stimulus. We observed substantial effects of altitude on blood acid-base status, but the changes tended to occur in the placebo group. A larger sample size and a direct comparison with the effects of other forms of altitude exposure on blood CO₂-related parameters are needed to address this issue.

Secondly, skeptical readers should recognize that the physiological changes we observed following altitude exposure provide several plausible mechanisms for the enhancements in performance. The striking reduction in lactate concentration in the incremental run, including maximum lactate, suggests some kind of change in substrate metabolism that could modify whatever intramuscular fatigue process limits performance. (The modification apparently does not involve an increase in buffering capacity, which would result in an *increase* in maximum lactate concentration.) The reduction in resting and submaximal exercise heart rates are consistent with an increase in circulating blood volume, which would enhance performance by increasing cardiac output and oxygen delivery to the muscles at high exercise intensities. The changes in hemoglobin concentration and hematocrit were not clear cut but were also in the right direction to

help account for the enhancement of performance via an increase in oxygen-carrying capacity. It seems unlikely that these hematological changes were due simply to hemoconcentration through loss of plasma volume, as suggested for such changes in some altitude studies (10), because the resulting loss in blood volume would *increase* resting and exercise heart rates. Any increase in red-cell mass would presumably be mediated by erythropoietin, but it is unclear whether the intermittent exposure protocol we used would produce a sufficient increase in release of this hormone (18,27). Future studies of performance following use of the rebreathing device should include measurement of red-cell mass and other physiological parameters associated with endurance performance, such as exercise efficiency. A role for an increase in oxygen consumption mediated by a more rapid or marked vasodilatation in active muscle is also possible. Whether any increase in white-cell count is causally related to the changes in performance through hormones of the immune system also needs to be clarified.

Finally, the changes in rating of training quality, which have not been assayed or reported in similar studies of altitude, have no reasonable explanation other than a substantial effect of altitude exposure on exercise performance. The large impairment in training quality in the first week of exposure was presumably a response to the stress of hypoxia or a manifestation of acute mountain sickness. The even larger improvement in training quality in the second week may have contributed to the enhancements in the performance tests by increasing training-related fitness. The training sessions of these athletes generally reproduced the physical demands of competition, so the scale of improvement in training quality is a reasonably clear indication that the effects of altitude exposure on performance are likely to extend beyond tests to games. Modifications of the exposure protocol should lead to further enhancements.

Acknowledgments

We thank Graeme Sequeira, Monica Wong and AUT postgraduate students for their assistance with performance testing. This study was supported by a research grant from the Auckland University of Technology. Douglas Pharmaceuticals provided the rebreathing devices and pulse oximeters for the study without charge. The authors received no other financial incentives from Douglas Pharmaceuticals and there is no past, present or intended future financial association between the authors and Douglas Pharmaceuticals.

References

1. Borg G. 1970. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med* 2:92-8.
2. Chapman RF, Stray-Gundersen J, Levine BD. 1998. Individual variation in response to altitude training. *J Appl Physiol* 85:1448-56.
3. Clark SA, Dixon J, Gore CJ, Martin DT, Hahn AG. Intermittent hypoxia fails to improve rowing ergometer performance (Abstract); 1999; Canberra. *Sports Medicine Australia*. p 85 (<http://www.ausport.gov.au/fulltext/1999/iocwc/abs085.htm>).
4. Clark VR, Hopkins WG, Hawley JA, Burke LM. 2000. Placebo effect of carbohydrate feedings during a 40-km cycling time trial. *Med Sci Sports Exerc* 32:1642-7.
5. Cohen J. 1988. *Statistical Power Analysis for the Behavioral Sciences*. New Jersey: Lawrence Erlbaum. p.25 p.
6. Dehnert C, Huetler M, Liu Y, Menold E, Netzer C, Schick R, Kubanek B, Lehmann M, Boening D, Steinacker JM. 2002. Erythropoiesis and performance after two weeks of living high and training low in well trained triathletes. *Int J Sports Med* 23:561-6.
7. Fornasiero D, Martin DT, Brosnan MJ, Arkinstall MJ, Lee H, Trewin C, Moquin A, Stephens S, Gore CJ, Hahn AG. Effects of altitude training on repeat sprint and graded exercise test performance in female road cyclists (Abstract); 1999; Canberra. *Sports Medicine Australia*. p 90 (<http://www.ausport.gov.au/fulltext/1999/iocwc/abs090b.htm>).
8. Gore CJ, Hahn AG, Aughey RJ, Martin DT, Ashenden MJ, Clark SA, Garnham AP, Roberts AD, Slater GJ, McKenna MJ. 2001. Live high:train low increases muscle buffer capacity and submaximal cycling efficiency. *Acta Physiol Scand* 173:275-86.
9. Hackett PH, Roach R. 1995. High-altitude medicine. In: Auerbach PS, editor. *Wilderness Medicine*. 3rd ed. St. Louis, MO: Mosby. p 1-37.
10. Hahn AG, Gore CJ. 2001. The effect of altitude on cycling performance: a challenge to traditional concepts. *Sports Med* 31:533-57.

11. Hahn AG, Gore CJ, Martin DT, Ashenden MJ, Roberts AD, Logan PA. 2001. An evaluation of the concept of living at moderate altitude and training at sea level. *Comp Biochem Physiol A* 128:777-89.
12. Hopkins WG. 2003. A spreadsheet for analysis of straightforward controlled trials. *Sportscience* 7:sportsci.org/jour/03/wghtrials.htm.
13. Hopkins WG, Hawley JA, Burke LM. 1999. Design and analysis of research on sport performance enhancement. *Med Sci Sports Exerc* 31:472-85.
14. Hopkins WG, Schabort EJ, Hawley JA. 2001. Reliability of power in physical performance tests. *Sports Med* 31:211-34.
15. Ingham EA, Pfitzinger PD, Hellemans J, Bailey C, Fleming JS, Hopkins WG. 2001. Running performance following intermittent altitude exposure simulated with nitrogen tents (Abstract). *Med Sci Sports Exerc* 33:S11.
16. Julian CG, Levine BD, Stray-Gundersen J, Gore CJ, Wilber RL, Daniel JT, Fredericson M. 2003 (in press). Intermittent hypoxic training (IHT): effects on hematological and performance markers in elite distance runners. In: Roach RC, Wagner PD, Hackett PH, editors. *Hypoxia: through the Lifecycle*. New York: Kluwer/Plenum.
17. Khotoshkina IV, Statzenko MV. 1993. Interval hypoxic training as a means of physical fitness and working capacity of elite rowers improvement. *Hypoxia Med J* 2:38-40.
18. Knaupp W, Khilnani S, Sherwood J, Scharf S, Steinberg H. 1992. Erythropoietin response to acute normobaric hypoxia in humans. *J Appl Physiol* 73:837-40.
19. Latyshkevich LA, Zakusylo MP, Shakhlina LH. 1993. The efficiency of interval hypoxic training in volley-ball. *Hypoxia Med J* 2:33-5.
20. Leger LA, Lambert J. 1982. A maximal multistage 20-m shuttle run test to predict VO_2 max. *Eur J Appl Physiol* 49:1-12.
21. Levine BD. 2002. Intermittent hypoxic training: fact and fancy. *High Altit Med Biol* 3:177-93.
22. Levine BD, Stray-Gundersen J. 1997. "Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance. *J Appl Physiol* 83:102-12.
23. Nummela A, Rusko H. 2000. Acclimatisation to altitude and normoxic training improve 400m running performance at sea level. *J Sports Sci* 18:411-9.

24. Paton CD, Hopkins WG, Vollebregt L. 2001. Little effect of caffeine ingestion on repeated sprints in team-sport athletes. *Med Sci Sports Exerc* 33:822-5.
25. Piehl Aulin K, Svedenhag J, Wide L, Berglund B, Saltin B. 1998. Short-term intermittent normobaric hypoxia - hematological, physiological and mental effects. *Scand J Med Sci Sports* 15:132-7.
26. Rodriguez FA, Murio J, Ventura JL. 2003. Effects of intermittent hypobaric hypoxia and altitude training on physiological and performance parameters in swimmers (Abstract). *Med Sci Sports Exerc* 35:S115.
27. Rodriguez FA, Ventura JA, Casas M, Casas H, Pages T, Rama R, Ricart A, Palacios L, Viscor G. 2000. Erythropoietin acute reaction and haematological adaptations to short, intermittent hypobaric hypoxia. *Eur J Appl Physiol* 82:170-7.
28. Savchenko ZP, Yugai NV. 1993. Interval hypoxic training in volley-ball. *Hypoxia Med J* 3:32-4.
29. Serebrovskaya T. 2002. Intermittent hypoxia research in the former Soviet Union and the Commonwealth of Independent States: history and review of the concept and selected applications. *High Altit Med Biol* 3:205-21.
30. Stray-Gundersen J, Chapman RF, Levine BD. 2001. "Living high-training low" altitude training improves sea level performance in male and female elite runners. *J Appl Physiol* 91:1113-20.