Effect of supplementary hypoxic training on physiological characteristics and ergometer performance of elite rowers

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Abstract Sixteen elite rowers supplemented their normal training with 30-60 min of rowing ergometer work daily for 19 consecutive days. The ergometer sessions were performed at 70-75% of maximum heart rate. During these sessions, eight of the rowers (control group) breathed medical air (20.93% oxygen). The other eight breathed a mixture of 15.2±0.2% oxygen in nitrogen, providing an inspiratory oxygen pressure equivalent to an altitude of 3100 m. The study was conducted in a single-blind fashion. Both before and after the period of supplementary training, the rowers completed two separate tests on a Concept II rowing ergometer. One was a discontinuous incremental test to exhaustion, and entailed measurements of oxygen uptake and blood lactate concentration. The other involved measurement of the time taken to complete a set amount of work (corresponding to 2500 m on the ergometer display). Resting venous blood samples were drawn from all rowers before, during and after the training period for determination of red blood cell count, haematocrit, haemoglobin concentration, reticulocyte levels, whole blood viscosity and serum concentrations of ferritin and haptoglobin.

Repeated measures analysis of variance showed that the rowers significantly improved their work outputs in the ergometer tests over the course of the study (alpha level = 0.05), with many attaining best-ever results. However, the magnitude of the improvement did not differ significantly between the control and hypoxic groups. Similar results were obtained for several physiological parameters. With the additional volume of training, there was a significant increase in the peak blood lactate concentration after the incremental ergometer test and in the concentration of reticulocytes in resting venous blood. There was a significant decrease in whole blood viscosity and serum ferritin concentration. Red blood cell count, haematocrit and haemoglobin concentration fell slightly during the initial stages of increased training, and then recovered. In no case was there a significant difference in the physiological responses of the control and hypoxic groups. Neither group showed a significant change in maximum oxygen uptake or resting serum haptoglobin concentration over the period of the study. It is concluded that the rowers gained significant physiological and performance benefits from the additional training, but that breathing of hypoxic gas during the supplementary sessions did not enhance the benefits.

Key words: altitude training, hypoxic training, red blood cells, reticulocytes, rowing.

INTRODUCTION

Polycythaemia induced by infusion of autologous red blood cells (RBC) or administration of erythropoietin can lead to increased maximum oxygen uptake and improved performance in endurance events.1-4 Such artificial procedures for increasing RBC numbers are banned under international sports doping policies. However, their demonstrated effectiveness has aroused new interest amongst coaches and athletes in identifying natural means for stimulating RBC increase.

There is strong evidence that training is effective in this regard, although its tendency to evoke an even greater increase in plasma volume often results in depression of RBC count and haematocrit.5-7
Another well-known stimulus to RBC increase is hypoxia. Total RBC mass is significantly increased after 3–4 weeks chronic exposure to altitudes of 4100–4300 m, and a rise in serum erythropoietin concentration can be detected after less than 90 min in a decompression chamber simulating an altitude of 4000 m.8,9–10

It is reasonable to suggest that a combination of training and hypoxic exposure might have a greater effect on RBC production than either stimulus alone. Certainly, altitude training is a common practice amongst endurance athletes. However, as indicated in several recent reviews, the scientific evidence relating to the matter is equivocal.11–13 Most studies have involved only small numbers of subjects and have lacked control groups. There has been variation between studies with regard to the initial physical condition of the subjects, the altitudes employed, training loads, and the tests used to assess performance changes. Consequently, results have been difficult to interpret.

Living and training at high altitudes has some disadvantages. In particular, the need for marked reduction of training intensity may lead to loss of specific neuromuscular capabilities.14 Lean body mass is typically reduced, and this too can be counter-productive.15 Runners trained for 8–9 weeks at approximately 4000 m showed no improvement in maximum oxygen uptake on return to sea level, and their running performance was worse.16 Thus many athletes have elected to train at more moderate altitudes of about 2000 m. There is uncertainty as to whether the hypoxic stimulus at these altitudes is sufficient to cause an increase in RBC numbers.

A better solution for the athletes may be to live and train at sea level but to perform some sessions under conditions of moderately severe hypoxia. We recently assessed the effects of 19 days supplementary hypoxic training on ergometer performance and certain physiological characteristics of elite rowers.

METHODS

Sixteen rowers (eight male, eight female) gave informed consent to take part in the study, which was approved by the Ethics Committee of the Australian Sports Commission. All were scholarship holders at the Australian Institute of Sport and had resided in Canberra (at an altitude of approximately 600 m) for at least the 5 months preceding commencement of the study. Their ages ranged from 17.5 to 23 years, with a mean of 20.7 years (s.d. = 1.8 years).

Preliminary testing

Four days after the 1991 Australian rowing championships, the athletes attended the laboratory in a rested state. In each case, 20 mL of blood was drawn from an antecubital vein with minimal stasis and with the athlete in a supine position. Whole blood viscosity was measured at high and low shear rates (100 s⁻¹ and 0.1 s⁻¹) using a Silverson rotational viscometer (Dandenong, Vic., Aust.). A Coulter Counter Model S550 (Hialeah, FL, USA) was used to determine RBC count, haemoglobin concentration, and haematocrit. Reticulocyte levels were determined using a Becton-Dickinson FACScan flow cytometer (Franklin Lakes, NJ, USA) after staining of the sample with Molecular Probes thiazole orange reticulocyte dye (Eugene, OR, USA). Serum haptoglobin concentration was measured by rate nephelometry using a Beckman Array (Schiller Park, IL, USA) and an antibody specific to haptoglobin. Serum ferritin concentration was determined by radio-immunoassay (Ciba-Corning Magic Ferritin [125I]; Australian Diagnostics, Melbourne, Vic., Aust.).

After the venous blood sampling, each rower was tested on a Concept II rowing ergometer (large cog, vent closed; Morrisville, VT, USA). Measurement was made of the time taken to complete a distance of 2500 m, as indicated by the ergometer display. The distance display is indicative of work done. The 2500 m test is an international standard in rowing, and is performed regularly by Australian rowers as a requirement for national selection.

On the following day, each rower completed a discontinuous incremental test on the Concept II ergometer. This test involved alternation of 3 min efforts with 1 min recovery periods. Target power outputs for the 3 min efforts increased in steps of 50 W from an initial load of 200 W for the men, and in steps of 25 W from 175 W for the women. Rowers able to meet the targets for five successive work periods were asked to make a maximal effort on the sixth. Performance was quantified in terms...
of the highest 3 min power output achieved (regardless of whether this occurred on the fifth or sixth workload). Earlobe blood samples were collected immediately after each effort during the test, and 2 and 4 min after the final effort. In each case, 25 μL of whole blood was added to 50 μL of a solution containing YSI Lactate Buffer (YSI 2357; Yellow Springs, OH, USA), YSI Cell Lysing Agent (YSI 1515), and sodium fluoride (in a concentration of 2.5 mg mL⁻¹). After complete lysis of RBC, the lactate concentration of the solution was determined using a YSI 2300L Lactate Analyser. The result was multiplied by three to obtain the whole blood lactate concentration. Throughout the test, oxygen uptake and other respiratory variables were measured each minute by means of an indirect calorimetry system incorporating Applied Electrochemistry oxygen and carbon dioxide analysers (Ametek, Pittsburgh, USA) and a P.K. Morgan ventilation meter (large turbine; Chatham, Kent, UK). The analysers were calibrated before and after each test against three alpha standard gravimetric gases that spanned the physiological range. The ventilation meter was calibrated before each test using a 1L syringe. The details of the gas analysis system have been described previously. Heart rates were also recorded minute-by-minute using a standard electrocardiograph (Siemens CardioStat 701; Medical Applications Pty Ltd, Adelaide, SA, Aust.) monitored by a Digital MicroVax computer.

For the purposes of the latter test, the ergometer was instrumented to measure the force applied to the chain connecting the 'oar' handle to the flywheel, and the horizontal linear displacement of the handle. Force applied to the chain was measured via a Kistler pre-calibrated quartz force link (model 9331A; Zurich, Switzerland) mounted within a modified ergometer handle in a way that ensured there was no interference with normal stroke length. A specially designed mechanical device, consisting of a cog in contact with the chain and a 360° potentiometer, was used to determine chain travel and consequently horizontal displacement of the handle throughout the stroke cycle. Force and displacement data were sampled continuously at 50 Hz/channel by a Digital MicroVax computer. This system allowed precise determination of average peak force, average stroke length and total mechanical work performed during each 3 min effort. It also enabled calculation of average work per stroke and stroke rate.

Training

After completion of the initial testing, the rowers returned to full training in preparation for a national selection regatta. Throughout the season, they had been training twice daily, with each session lasting about 2 h. The training consisted of rowing in pairs and fours, and exercises with weights. Now, for a period of 19 consecutive days, a third training session was added. This involved continuous exercise on a Concept II rowing ergometer, usually for two 30 min periods separated by a 3 min ‘drinks break’. However, on the first day, the rowers completed a total of only 50 min ergometer work, and on the last 3 days, the times were 45, 45 and 30 min. The reduced exercise times towards the end of the programme reflected a general tapering of training in preparation for the national selection regatta. During ergometer work, the rowers wore Polar KY Sports Tester heart rate monitors (Hakamaantie, Kempele, Finland), and were asked to keep their heart rates at 70–75% of the maximum value recorded during the incremental test. They were prevented from seeing the digital display unit of the ergometer, and were not informed of their power outputs. However, their average power outputs were recorded at the end of each session.

Composition of inspired air during the additional training sessions

Throughout the ergometer sessions, the rowers breathed through a Hans Rudolph 2700 valve (Kansas City, MO, USA). The inspiratory side of the valve was connected to a 12 L balloon reservoir receiving flow from a cylinder of compressed gas (Fig. 1). A technician was responsible for adjusting the flow so that the balloon reservoir was kept almost (but not quite) full. Thus the gas was brought to ambient pressure prior to its inspiration.

The rowers were divided into two groups, with those who competed as a pair being separated. One group always breathed standard medical air (20.93% oxygen) during the ergometer work. The other breathed a mixture of 15.2 ± 0.2% oxygen in nitrogen. Since the average barometric pressure in Canberra is 710 mmHg, the latter provided an
Blood analyses during the training period

Venous blood samples were again collected on the mornings of the days 7 and 14 of training, before any exercise had been performed. These samples were subjected to the same analysis as those collected at the beginning of the study, except that reticulocyte levels were not measured, haptoglobin concentration was measured only for day 7, and whole blood viscosity was determined only for day 14.

On day 7, an additional venous blood sample was drawn 3–10 min after the ergometer session. Serum erythropoietin concentration was measured by radio-immunoassay (Diagnostic Systems Laboratories Erythropoietin Kit, Immunodiagnostics, Sydney, NSW, Aust.) for both the pre-exercise and post-exercise samples. The measurements were aimed at assessing the relative effects of exercise and hypoxia on erythropoietin levels during a single training session. They were also aimed at determining whether the breathing of hypoxic gas had led to comparative elevation of resting erythropoietin levels over a period of 1 week.

Testing at the end of the training period

On the day after completion of the training period, resting venous blood samples were again drawn from the rowers who, with one exception, then repeated the discontinuous incremental test on the instrumented Concept II ergometer. The one exception was a female member of the control group who had developed lower back pain. The blood samples were analysed for viscosity, RBC count, haemoglobin concentration, haematocrit, reticulocyte levels, and serum concentrations of ferritin and haptoglobin, as before.

Six days later, and 2 days after the finish of the national selection regatta, all but two of the rowers performed a 2500 m test on the Concept II ergometer. This test was performed in Melbourne, as part of the national selection process, but each rower used the same ergometer as in the original Canberra test. Again, a record was made of the time taken to complete the 2500 m, as indicated by the digital display unit of the ergometer.

Of the two rowers who missed the final 2500 m test, one was ill and the other had been informed by the selectors that national representation was
no longer a possibility. In the latter case, the athlete was obviously not motivated to perform the test.

Statistical analysis

Most variables included in the study were measured serially across time. The aim was to determine whether they were significantly affected by the training, and whether the response differed between the hypoxic and standard air (control) groups. To achieve this, the data were assessed by two-way analysis of variance with repeated measures on the time factor. Where appropriate, Tukey post-hoc comparisons were used for more precise location of significant differences.

Student’s t-test for independent samples was used to determine the significance of differences between the groups for variables on which there was a single result for each subject. Relationships between variables were evaluated by calculation of Pearson’s product-moment correlation coefficients.

Subjects with missing data points (due to absence from one or other of the final tests or, in one case, the clotting of a blood sample) were totally excluded from the statistical analysis of the variables in question.

RESULTS

On several occasions, individual rowers were permitted by their coaches to miss an ergometer training session. Nevertheless, the overall attendance rate was 97.4% for the hypoxic group and 95.4% for the control group. Power output during ergometer training averaged 150.4 W for the hypoxic subjects and 170.4 W for the controls. The difference was statistically significant (t = 5.217, \( P < 0.001 \)), and occurred despite the fact that under normoxic conditions the maximal work capacities of the two groups were similar. The power outputs did not tend to increase during the course of the study. There was no case of headache, nausea or other medical symptoms as a result of hypoxic exposure.

Times for 2500 m test

Times for the 2500 m test before and after the training period are shown in Table 1. For purposes of comparison, personal best times recorded before the training period are also presented. The rowers showed a significant improvement from the pre-test to the final test (\( P < 0.005 \)), but the magnitude of the change did not differ significantly between the hypoxic and control groups. Of the 14 rowers who completed the final 2500 m test, 11 (six members of the hypoxic group and five controls) improved on their previous best time. However, for the group as a whole, the difference between final and previous best times was not statistically significant (\( P > 0.05 \)).

TABLE 1. Individual times for the 2500 m rowing ergometer test before and after the period of supplementary training

<table>
<thead>
<tr>
<th>Rower</th>
<th>Gender</th>
<th>Pre-supp. training (Canberra)</th>
<th>Personal best*</th>
<th>Post-supp. training (Melbourne)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>M</td>
<td>7:42</td>
<td>7:40</td>
<td>7:37</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>7:57</td>
<td>7:53</td>
<td>7:49</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>7:58</td>
<td>7:53</td>
<td>7:49</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>7:45</td>
<td>7:36</td>
<td>7:31</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>9:02</td>
<td>9:02</td>
<td>8:55</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>8:49</td>
<td>8:49</td>
<td>8:43</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>8:59</td>
<td>8:48</td>
<td>8:55</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>9:02</td>
<td>9:02</td>
<td>0:03</td>
</tr>
<tr>
<td>Mean ± s.d.</td>
<td>8:24 ± 0:37</td>
<td>8:20 ± 0:38</td>
<td>8:18 ± 0:40</td>
<td></td>
</tr>
</tbody>
</table>

| Control group |
| 1 | M | 7:44 | 7:44 | 7:40 |
| 2 | M | 7:53 | 7:46 | 7:42 |
| 3 | F | 8:47 | 8:47 | 8:40 |
| 4 | F | 8:59 | 8:46 | 8:47 |
| 7 | F | 9:11 | 9:11 | 9:03 |
| 8 | F | 8:41 | 8:41 | 8:34 |
| Mean ± s.d. | 8:33 ± 0:36 | 8:29 ± 0:36 | 8:24 ± 0:35 |

* Recorded before supplementary training.

Progressive ergometer test

For the hypoxic group, the peak 3 min power output during the progressive ergometer test, as indicated by the ergometer display unit, averaged 356.6 ± 83.6 W at the start of the study and 376.1 ± 96.8 W at the end. The control group showed a mean increase from 358.9 ± 78.4 to 380.8 ± 76.2 W. Again, statistical analysis revealed a significant training effect (\( P < 0.005 \)), but no significant difference in the response of the two
FIG. 2. Physiological responses of (a) hypoxic \( n = 8 \) and (b) control \( n = 7 \) groups to a progressive rowing ergometer test before and after 19 days of supplementary training. \( \text{---} \) Pre-training; \( \text{---} \) post-training.
groups. Biomechanical evaluation of peak 3 min work output yielded a similar result. The additional work at the end of the training period was achieved mainly through a significant increase in stroke rate ($P<0.005$). Stroke length was slightly decreased ($P<0.05$), but this failed to cause a significant change in work per stroke. The average peak force generated during the stroke was not significantly altered ($P>0.05$).

Maximum oxygen uptake was not changed by training. The mean value for the hypoxic group was $4.63 \pm 0.94 \text{ L min}^{-1}$ in the pre-test, and $4.62 \pm 0.83 \text{ L min}^{-1}$ in the final test. The reading for the control group showed a non-significant decline from $4.90 \pm 0.90$ to $4.74 \pm 0.76 \text{ L min}^{-1}$. The difference in the response of the two groups did not approach statistical significance ($P>0.30$).

Oxygen uptakes recorded at submaximal work-load during the progressive test showed little change for either group, and the same was true of heart rates and respiratory exchange ratios (Fig. 2). Maximal values for the latter two parameters were also unchanged by training (Table 2). By contrast, whole blood lactate concentrations were elevated (though not always significantly) at each workload (Fig. 2). Before the training period, the peak value measured after the final stage of the progressive test averaged $11.4 \pm 2.8 \text{ mmol L}^{-1}$ for the hypoxic group and $10.7 \pm 3.0 \text{ mmol L}^{-1}$ for the controls. In the final test, the peak readings were $15.9 \pm 2.0$ and $14.4 \pm 1.9 \text{ mmol L}^{-1}$ respectively. The rise in the peak lactate concentration with training was statistically significant ($P<0.001$), but the response of the hypoxic group was not significantly different from that of the control group.

### Table 2. Peak values of various parameters measured during the progressive ergometer test before and after the supplementary training period

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hypoxic group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>3 min power output (W)</td>
<td>$356.6 \pm 83.6$</td>
<td>$376.1 \pm 96.8$</td>
</tr>
<tr>
<td>Oxygen uptake (L min$^{-1}$)</td>
<td>$4.63 \pm 0.94$</td>
<td>$4.62 \pm 0.83$</td>
</tr>
<tr>
<td>Respiratory exchange ratio</td>
<td>$1.09 \pm 0.04$</td>
<td>$1.11 \pm 0.04$</td>
</tr>
<tr>
<td>Heart rate (beats min$^{-1}$)</td>
<td>$195 \pm 7$</td>
<td>$194 \pm 5$</td>
</tr>
<tr>
<td>Blood lactate (mmol L$^{-1}$)</td>
<td>$11.4 \pm 2.8$</td>
<td>$15.9 \pm 2.0$</td>
</tr>
</tbody>
</table>

Data are presented as mean ± s.d.

### Table 3. Mean haematological values of hypoxic and control subjects before, during and after the supplementary training period

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Before</th>
<th>Day 7</th>
<th>Day 14</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC count ($10^{12}$ L$^{-1}$)</td>
<td>Hypoxic</td>
<td>$4.90 \pm 0.42$</td>
<td>$4.74 \pm 0.44$</td>
<td>$4.69 \pm 0.37$</td>
<td>$4.83 \pm 0.29$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$4.92 \pm 0.40$</td>
<td>$4.84 \pm 0.52$</td>
<td>$4.91 \pm 0.46$</td>
<td>$4.91 \pm 0.44$</td>
</tr>
<tr>
<td>Haemoglobin conc. (g 100 mL$^{-1}$)</td>
<td>Hypoxic</td>
<td>$14.9 \pm 1.1$</td>
<td>$14.5 \pm 1.2$</td>
<td>$14.3 \pm 1.2$</td>
<td>$14.8 \pm 0.8$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$14.9 \pm 0.8$</td>
<td>$14.7 \pm 1.0$</td>
<td>$14.9 \pm 0.9$</td>
<td>$14.9 \pm 0.8$</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>Hypoxic</td>
<td>$44.3 \pm 3.2$</td>
<td>$42.3 \pm 3.4$</td>
<td>$43.2 \pm 4.0$</td>
<td>$43.5 \pm 2.4$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$44.0 \pm 2.0$</td>
<td>$42.9 \pm 3.0$</td>
<td>$44.2 \pm 2.8$</td>
<td>$44.0 \pm 2.3$</td>
</tr>
<tr>
<td>Serum haptoglobin conc. (g L$^{-1}$)</td>
<td>Hypoxic</td>
<td>$0.9 \pm 0.4$</td>
<td>$0.8 \pm 0.4$</td>
<td>—</td>
<td>$0.8 \pm 0.5$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$0.9 \pm 0.4$</td>
<td>$0.8 \pm 0.4$</td>
<td>—</td>
<td>$0.7 \pm 0.3$</td>
</tr>
<tr>
<td>Serum ferritin conc. (ng mL$^{-1}$)</td>
<td>Hypoxic</td>
<td>$63 \pm 19$</td>
<td>$52 \pm 23$</td>
<td>$54 \pm 20$</td>
<td>$48 \pm 20$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$75 \pm 26$</td>
<td>$73 \pm 28$</td>
<td>$76 \pm 23$</td>
<td>$65 \pm 27$</td>
</tr>
<tr>
<td>Reticulocytes (% of RBC)</td>
<td>Hypoxic</td>
<td>$2.0 \pm 0.4$</td>
<td>—</td>
<td>—</td>
<td>$3.1 \pm 1.0$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$1.8 \pm 0.3$</td>
<td>—</td>
<td>—</td>
<td>$2.4 \pm 0.7$</td>
</tr>
<tr>
<td>Reticulocyte conc. ($10^9$ L$^{-1}$)</td>
<td>Hypoxic</td>
<td>$97 \pm 22$</td>
<td>—</td>
<td>—</td>
<td>$143 \pm 49$</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>$88 \pm 21$</td>
<td>—</td>
<td>—</td>
<td>$113 \pm 22$</td>
</tr>
</tbody>
</table>

Data are presented as mean ± s.d.
Red blood cell count, haematocrit and haemoglobin concentration

The mean RBC counts, haematocrits and haemoglobin concentrations recorded for the two groups over the course of the study are presented in Table 3. All of these variables showed a slight decline during the first week of the training period. For the control group, they had returned to normal by the end of the second week. By contrast, values for the hypoxic group remained depressed and, in the case of the RBC count and the haemoglobin concentration, even showed a slight further decrease. For the hypoxic group, a return towards starting levels did not occur until the third week. However, analyses of variance were unable to detect any significant difference between the groups in terms of temporal or other characteristics of the response. These analyses revealed only that, for the rowers in general, haematocrits after 1 week were significantly lower than at the outset.

Haptoglobin, reticulocytes and ferritin

Serum haptoglobin concentrations (Table 3) showed only a slight and non-significant decrease during the study. The trend was not detectably different for the hypoxic and control groups. At the start of the training period, reticulocyte levels for both groups were in the upper part of the normal range. On average, they constituted 2.0 ± 0.4% of all RBC for members of the hypoxic group, and 1.8 ± 0.5% for the controls (normal range = 0.2–2.0%). By the end of the study, the levels had risen to 3.1 ± 1.0% and 2.4 ± 0.7%, respectively. Overall, the change in reticulocytes was significant (P < 0.001), but the response of the hypoxic group was not statistically different from that of the control group. There was a significant decrease in serum ferritin concentration over the training period (P < 0.01; Table 3), but again the trend did not differ significantly between the two groups. When the results of all the rowers were pooled, there was a correlation of 0.65 (P < 0.01) between the rise in reticulocytes during the study and the reduction of serum ferritin concentration.

Whole blood viscosity

Whole blood viscosity at both high and low shear rates decreased significantly during the first 2 weeks of the training period (Table 4). There was an increase during the final week, but viscosity at the high shear rate remained significantly below the initial level (P < 0.01). The low shear rate viscosity at the end of the study was also below the starting point, but the difference was not statistically significant (P > 0.05). Changes in blood viscosity were not related to the gas mixture breathed during the ergometer training sessions.

Serum erythropoietin concentration at rest and after exercise

The serum erythropoietin concentration before exercise on day 7 of ergometer training averaged 22.6 ± 7.1 mU mL⁻¹ for the hypoxic group and 23.6 ± 5.9 mU mL⁻¹ for the controls. The difference was not statistically significant (P > 0.05). Furthermore, neither group showed a rise in the concentration at the end of 1 hour of exercise. The post-exercise reading for the hypoxic group was 22.6 ± 6.5 mU mL⁻¹, while that for the control group was 22.9 ± 5.4 mU mL⁻¹. Interestingly, serum erythropoietin concentrations were substantially higher for the women rowers than for the men (t = 5.29, P < 0.001). Obviously, the higher values could reflect a need to replace RBC lost through menstruation.

<table>
<thead>
<tr>
<th>Shear rate</th>
<th>Group</th>
<th>Before</th>
<th>Day 14</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>High (100 s⁻¹)</td>
<td>Hypoxic</td>
<td>4.088 ± 0.400</td>
<td>3.645 ± 0.314</td>
<td>3.845 ± 0.353</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>4.138 ± 0.230</td>
<td>3.828 ± 0.249</td>
<td>3.866 ± 0.357</td>
</tr>
<tr>
<td>Low (0.1 s⁻¹)</td>
<td>Hypoxic</td>
<td>66.254 ± 20.191</td>
<td>50.143 ± 11.933</td>
<td>55.116 ± 17.225</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>71.005 ± 16.118</td>
<td>59.067 ± 10.415</td>
<td>62.396 ± 9.143</td>
</tr>
</tbody>
</table>

Data are presented as mean ± s.d.


DISCUSSION

Hypoxic group versus control group

The rowers who breathed hypoxic gas during supplementary training gained no measurable advantage over those who breathed standard medical air. The responses of the two groups were not significantly different for any of the physiological or performance variables measured.

The inclusion of the control group was critical to interpretation of the results of the study. Without it, the increased reticulocyte levels and improved ergometer performances of the experimental subjects might have been wrongly attributed to the daily hypoxic exposures. It was the increased volume of training that seems to have been the explanation. Many of the studies reporting positive effects of hypoxic training have not incorporated control groups. Moreover, training volumes have often been increased during the period of hypoxic exposure.\textsuperscript{18,19} The present findings indicate that training effects can occur even in elite athletes at the height of their competitive season. This is contrary to assumptions sometimes made by previous researchers.\textsuperscript{20}

The lack of effect of hypoxia in the present study may have been due to the limited duration of the daily exposures. Eckardt et al.\textsuperscript{10} collected serial blood samples from six resting male subjects exposed to a simulated altitude of 3000 m. On average, it was 114 min before serum erythropoietin concentration began to rise. We expected that imposition of an exercise task would add to the hypoxic stress and lead to earlier erythropoietin increase. However, the rowers showed no rise in serum erythropoietin concentration after 1 h of ergometry. The final blood sample for measurement of erythropoietin was collected within 10 min of the completion of exercise. A response to hypoxia might have been observed if we had drawn another sample 1–2 h later. Erythropoietin is not stored, and must therefore be produced when a relevant stimulus is encountered. This entails a delay, but once production is set in train, serum erythropoietin concentration will eventually rise. Eckardt et al. have shown that the rise can occur even after the hypoxic stimulus has been removed.\textsuperscript{10} The present results may also have been influenced by the fact that erythropoietin measurements were not made until day 7 of the supplementary training programme. Milledge and Cotes reported that in subjects exposed for several days to constant moderate hypoxia, serum erythropoietin levels soon become unresponsive.\textsuperscript{21} Although the experimental group breathed hypoxic gas for only a brief period each day, a similar loss of sensitivity might have occurred. Yet if hypoxia had elicited an erythropoietin increase which was undetected for reasons of experimental protocol, the hypoxic and control groups should have differed significantly with regard to changes in RBC count, haemoglobin concentration, and reticulocyte levels. Such a difference was not observed.

A question can be raised as to whether the supplementary training was sufficiently intense. We chose a level of exercise which would not be expected to induce metabolic acidosis. This was necessary to ensure that the rowers could accommodate their other training requirements. Furthermore, recent studies indicate that metabolic acidosis inhibits erythropoietin release.\textsuperscript{22} We therefore believe that our choice of workload was appropriate.

There have been several other controlled studies of the effects of daily periods of hypoxic training on subjects living at or near sea level.\textsuperscript{23–26} They have generally indicated that short-term improvements in physical work capacity are greater with hypoxic training than with similar training in a normal environment. However, only one of these studies involved elite athletes.\textsuperscript{25} Even in this case, the subjects were clearly not in peak condition at the outset, since very large increases in work capacity were subsequently observed for both the hypoxic (33%) and control (22%) groups. It is possible that hypoxia accelerates early adaptations to training, but that the end-points are much the same as those eventually achieved through the normal training process. This would explain the apparently beneficial effect of hypoxic training on previously sedentary subjects, while also accounting for its lack of influence on already well-trained athletes such as the rowers of the present study.

General training effects

Some aspects of the response of our subjects to training are of particular interest. Since the response was independent of the condition under which the
supplementary sessions were performed, the results may be considered in a general sense. During the first week of the training period, there was a significant decrease in haematocrit, while RBC count and haemoglobin concentration were also moderately (although not significantly) reduced. These changes were probably due to increase of plasma volume rather than large-scale destruction of RBC, as serum haptoglobin concentration showed only a slight and non-significant fall. The work of Schmidt et al. suggests that reduced haematocrit may lead to an increase in reticulocyte levels; an increase which was demonstrated in the present investigation. A reduction in serum ferritin concentration was also observed. The fact that there was a strong negative correlation between rise in reticulocytes and reduction in ferritin suggests the incorporation of iron into new RBC, and provides further evidence for increased RBC production. The effect of reduced haematocrit on RBC production is thought to be mediated primarily through enhanced release of erythropoietin. As the serum erythropoietin concentrations of the rowers were not measured at the beginning of the training period, the mediating role of the hormone could not be assessed in the present investigation. However, the resting erythropoietin concentrations on day 7 of supplementary training did approach the upper limits of the normal range for the method used.

By the end of our experiment, the haematocrits, RBC counts and haemoglobin concentrations of the rowers had returned to normal. This probably reflected a regression towards the initial plasma volume, but the increased reticulocyte levels suggest that there might also have been some increase in total RBC mass. If so, it was not sufficient to cause an increase in maximum oxygen uptake, but it may have improved the capacity of the blood to buffer hydrogen ions, since haemoglobin has an important buffering role. This could partially explain the enhanced work outputs and increased peak blood lactate concentrations of the rowers after the training period. Such a mechanism has been proposed previously by Buick et al.1

The elevated reticulocyte levels of the rowers after training clearly indicate a decrease in the average age of the RBC population. This alone could lead to improved work capacity, since younger RBC are more deformable, and so gain easier access to small capillaries. They also have higher concentrations of 2,3-diphosphoglycerate, and therefore release oxygen more readily at the cellular level. Haematocrit is one of several factors influencing whole blood viscosity. Thus it is not surprising that the whole blood viscosity of the rowers decreased in the early part of the training period, then began to increase. However, even at the end of the study when haematocrit had returned to normal, viscosity remained below initial levels. This may have been partly due to increased RBC deformability, but training has also been found to influence other components of whole blood viscosity, such as plasma fibrinogen, yield stress and plasma viscosity. All else being equal, reduction in blood viscosity should allow better perfusion of the active muscles, conferring a performance advantage. Improved perfusion would not only increase oxygen supply to the muscle, but would also enhance transfer of metabolites between muscle and blood. Consequently, the increased blood lactate concentrations of our rowers at submaximal and maximal workloads after the training period might be partly attributable to their reduced whole blood viscosity. In any case, the elevated lactates are apparently not due to increased use of glycogen as a metabolic substrate, even though the rowers had started to taper their training before the final progressive test. Increased glycogen usage could be expected to result in elevated respiratory exchange ratios and reduced oxygen uptakes at submaximal workloads, but neither variable was significantly changed.

Conclusion

In summary, it appears that the rowers gained significant physiological and performance benefits from the supplementary training. Breathing of hypoxic gas during the supplementary sessions did not enhance the benefits. Nevertheless, the possibility remains that under different conditions (e.g. lower inspiratory oxygen content, longer periods of exposure) hypoxic training might be advantageous for endurance athletes. Further research is needed to clarify this matter.
ACKNOWLEDGEMENTS

This study was supported by grants from various agencies of the Australian Sports Commission, including the Applied Sports Research Program, the Centre for Sports Science and Sports Medicine, and the Sports Development Branch. Funds were also contributed by the Rowing Unit of the Australian Institute of Sport. The authors wish to acknowledge the excellent technical support provided by Ian Jamieson, Warren Roberts, Kaylene Hood, Stan Martin, Robert Shugg, Kate Cameron, Kim Putland and Michael Savage.

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