Physiological and Performance Responses to a Preseason Altitude-Training Camp in Elite Team-Sport Athletes

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Purpose: Little research has been done on the physiological and performance effects of altitude training on team-sport athletes. Therefore, this study examined changes in 2000-m time-trial running performance (TT), hemoglobin mass (Hbmass), and intramuscular carnosine content of elite Australian Football (AF) players after a preseason altitude camp. Methods: Thirty elite AF players completed 19 days of living and training at either moderate altitude (~2130 m; ALT, n = 21) or sea level (CON, n = 9). TT performance and Hbmass were assessed preintervention (PRE) and postintervention (POST1) in both groups and at 4 wk after returning to sea level (POST2) in ALT only. Results: Improvement in TT performance after altitude was likely 1.5% (± 4.8–90%CL) greater in ALT than in CON, with an individual responsiveness of 0.8%. Improvements in TT were maintained at POST2 in ALT. Hbmass after altitude was very likely increased in ALT compared with CON (2.8% ± 3.5%), with an individual responsiveness of 1.3%. Hbmass returned to baseline at POST2. Intramuscular carnosine did not change in either gastrocnemius or soleus from PRE to POST1. Conclusions: A preseason altitude camp improved TT performance and Hbmass in elite AF players to a magnitude similar to that demonstrated by elite endurance athletes undertaking altitude training. The individual responsiveness of both TT and Hbmass was approximately half the group mean effect, indicating that most players gained benefit. The maintenance of running performance for 4 wk, despite Hbmass returning to baseline, suggests that altitude training is a valuable preparation for AF players leading into the competitive season.

Keywords: football, hypoxia, hemoglobin mass, carnosine

High-intensity-running performance is vital to success in team sports, requiring athletes to have a highly developed aerobic system together with a large anaerobic capacity. Therefore, training modalities that increase an athlete’s ability to perform high-intensity work should benefit overall team performance. A recent modality adopted by professional teams in an attempt to improve performance is altitude training, where athletes are exposed to a hypobaric, hypoxic environment. Altitude training has been reported to induce a range of physiological adaptations, and 2 of these responses—increases in oxygen-carrying capacity of the blood and increases in muscle buffering capacity—would be expected to improve high-intensity-running ability. For improved oxygen transport, the primary physiological adaptation after altitude exposure is an increase in total hemoglobin mass (Hbmass), and this can lead to increases in VO2max. With respect to increases in muscle buffering capacity after altitude exposure, the underpinning mechanism is unknown, but several authors propose that an increase in an intramuscular buffer, carnosine, is likely responsible.

The live-high, train-high (LHTH) model, involving living and training at moderate altitudes in the range of 2000–3000 m, is a common practice for hypobaric, hypoxic exposure. Endurance athletes have used this model for more than half a century, attempting to improve performance at sea level. Similarly, for professional team sports, the ultimate goal of a preseason altitude camp is to elicit performance improvements and increase training capacity on return to sea level. However, there are no published data describing the physiological or performance responses after altitude exposure in elite team-sport athletes. Given the unique physical attributes and performance demands of these athletes, it is important to determine how they respond to an altitude camp and whether there are performance gains on return to sea level.

Therefore, the current study examined the physiological and performance changes in professional
Australian Football players after a 19-day altitude training camp. These data were compared with those of a control group of elite Australian Football players undertaking similar training at sea level. We hypothesized that increases in Hbmass and intramuscular carnosine content would occur after altitude exposure, and after an LHTH camp athletes would show greater improvements in running performance than their matched controls completing similar training at sea level.

**Methods**

**Subjects**

Thirty elite Australian Football players (mean ± SD; age 23± 3 y, height 188.2 ± 8.0 cm, body mass 88.0 ± 9.0 kg, sum of 7 skinfolds = 44.9 ± 4.7 mm) were examined throughout an 8-week training block (see Figure 1). Subjects were split into altitude (ALT, n = 21) and control (CON, n = 9) training groups. All training and testing took place during the Australian Football League preseason from November to January, after a 6-week off-season break. All subjects provided written informed consent, and this study was approved by the human research ethics committee at Australian Catholic University.

**Nutritional Supplementation**

Nutritional supplements were prescribed throughout the study by club dietitians. All subjects were supplemented with oral ferrous sulfate (325 mg/d) throughout the intervention period, and athletes identified as having low serum ferritin preintervention (serum ferritin ≤ 30 μg/L; n = 2) were given a single, 2-mL ferrum H injection (equivalent to 100 mg of iron) before the altitude camp. No supplements containing β-alanine were prescribed, and no subjects had used β-alanine within the preceding 6 months. As β-alanine is known to increase intramuscular carnosine concentrations,10 players were asked to refrain from consuming any supplements containing β-alanine throughout the study, and they reported 100% compliance.

**Training**

During an 8-week training block, the ALT training group completed 19 days living and training at altitude, involving endurance- and resistance-exercise training, in Flagstaff, AZ (elevation = 2130 m). The CON group training in Melbourne, Australia (elevation = 30 m), completed training similar to that of the ALT group for the first 4 weeks of the intervention; thereafter training differed between groups. As such, data for the CON group are only reported through the first 4 weeks of the intervention period. All training was prescribed by team coaches and monitored via session rating of perceived exertion (RPE).11 This method calculates a total load (arbitrary units [AU]) by multiplying the session RPE (Borg’s CR 10-scale) by the session duration.

**Running Performance**

To assess high-intensity-running performance, subjects completed a 2000-m running time trial (TT) at the commencement (PRE) and 4 weeks into the training block (POST1). In addition, TT performance was assessed again in ALT subjects 8 weeks into the training block (POST2). TT performance was assessed on the same outdoor running track at sea level in Melbourne, Australia, between 8 and 9 AM (temperature 18–26°C, humidity 50–80%) and measured via handheld stop watches. All players were familiar with the TT from previous years—most had completed 6 to 10 (minimum 3) similar time trials in the preceding 1 to 3 years.

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**Figure 1** — Schematic timeline of study design. The altitude-training group (ALT) was measured preintervention (PRE), postintervention (POST1), and 4 weeks after returning to sea level (POST2). The sea-level control group (CON) was measured at PRE and POST1. Abbreviations: MRS, magnetic resonance spectroscopy.
Hematological Measures

All subjects were measured for Hbmass using the optimized carbon monoxide rebreathing technique\textsuperscript{12} at PRE and POST1 (see Figure 1). In addition, Hbmass was measured again in ALT subjects at POST2. Briefly, subjects rebreathed a bolus of 99\% CO equivalent to 1.0 mL/kg of body mass through a glass spirometer (BloodTec, Bayreuth, Germany) for 2 minutes. Percent carboxy-hemoglobin (%HbCO) in fingertip capillary blood was measured using an OSM3 hemoximeter (Radiometer, Copenhagen, Denmark) before and 7 minutes after administration of the CO dose. Six repeat measures of %HbCO were made for improved precision in Hbmass estimation.\textsuperscript{13} Venous blood samples were sent to a local hospital laboratory (St Vincent’s Pathology, Fitzroy, VIC, Australia) for assessment of reticulocyte count in both groups at PRE and POST1 and in the ALT group only at POST2. Preceding venous blood-sample collection, subjects’ ambulation was limited for 15 minutes, with the majority of this time spent sitting. Concentration of serum ferritin was also assessed at PRE to identify subjects who might be iron deficient.

Magnetic Resonance Spectroscopy

Magnetic resonance spectra were acquired in the soleus and gastrocnemius of ALT only at a field strength of 3T on a Philips Achieva system (Philips Medical Systems, Best, The Netherlands) using the point-resolved spectroscopy technique\textsuperscript{14,15} with a repetition time of 2000 milliseconds and an echo time of 33 milliseconds. Voxel sizes varied depending on the size of the muscle but were generally in the range of 10 mm × 20 mm × 40 mm. Care was taken to position the voxel in such a way as to avoid contributions from fasciae across the chemical shift range of the voxel. Voxels were shimmed to between 12 and 19 Hz; 256 water-suppressed and 8 non-water-suppressed averages were acquired at the same receiver gain. Spectral data analysis was carried out using JMRUI version 4.0\textsuperscript{16} after zero filling and line broadening by 5Hz; metabolite peaks were fitted and expressed relative to the non-water-suppressed signal. Peaks were assigned as follows: carnosine C2-H (8 ppm) and C4-H (7 ppm), residual water (around 4.7 ppm), phosphocreatine and creatine (3.05 and 3.95 ppm), containing metabolites (3.20 ppm). All analyses presented in this article refer to the carnosine C2-H peak at 8 ppm and the carnitine and choline peak at 3.20 ppm. No relaxation-time corrections were made.

Statistical Analysis

A contemporary analytical approach involving magnitude-based inferences\textsuperscript{17} was used to detect small effects of practical importance. All data were log-transformed to account for nonuniformity error. The percentage changes in the mean TT and Hbmass from prealtitude to each time point after altitude were calculated. The differences within and between groups were assessed with dependent and independent t tests for unequal variance.\textsuperscript{17} The magnitudes of changes were assessed in relation to the smallest worthwhile change (SWC), which was set to 2\% for TT and Hbmass, and a small effect size (d = 0.2) was used for all other variables. Analysis of overall training load revealed differences between the CON (3229 ± 447 AU per week; mean ± SD) and ALT (4249 ± 351) groups. As a result, training load was used as a covariate in the analysis of changes in TT and Hbmass. The observed effects were reported as the mean change or difference ± 90\% confidence limits (CL). Effects were termed positive, trivial, or negative depending on the magnitude of the change relative to the SWC and were assigned a qualitative descriptor according to the likelihood of the change exceeding the SWC as follows: 50\% to 74\%, possible; 75\% to 94\%, likely; 95\% to 99\%, very likely; and >99\%, almost certainly.\textsuperscript{18} Effects where the 90\% confidence interval overlapped simultaneously the substantially positive and the negative thresholds were deemed unclear. The individual response was also quantified for TT performance and Hbmass. For each, the magnitude of individual responses was calculated from the square root of the difference in the variance of the change scores of the CON and ALT groups.\textsuperscript{19} A Pearson correlation coefficient was used to examine the relationship between initial Hbmass and change in Hbmass at POST1—however, 2 subjects reported illness during the study and were subsequently removed from the correlation analysis, as proinflammatory cytokines that are increased with infection are known to suppress EPO production.\textsuperscript{20}

Results

Training Load

Training load, training duration, and RPE are presented in Table 1. Throughout the first 4 weeks of the intervention period, overall training load was almost certainly 24.5\% ± 10\% higher in ALT than in CON. Training duration was very likely 11.5\% ± 7.2\% higher and mean RPE was almost certainly 13.3\% ± 5.4\% higher in ALT than in CON.

2000-m Time-Trial Performance

Time-trial performance was possibly faster in ALT (413 ± 14 s) than in CON (422 ± 23 s) before the altitude-training camp (Figure 2). The mean improvement in TT performance (± 90\% CL) at POST1 was likely 2.1\% ± 2.1\% greater in ALT than in CON. When training load was used as a covariate, change in TT was possibly 1.5\% ± 4.8\% greater in ALT than in CON at POST1. The individual variation in TT performance at POST1 was 0.9\% without training load used as a covariate and 0.8\% when load was used as a covariate. Thirty days postdescent (POST2), improvement in TT performance in ALT had been maintained, with the change from POST1 to POST2 trivial (–0.8\% ± 0.9\%).
### Table 1  Training Load, Training Duration, and Rating of Perceived Exertion (Mean ± SD) in ALT and CON During the First 4 Weeks of the Intervention

<table>
<thead>
<tr>
<th></th>
<th>ALT (n = 21)</th>
<th>CON (n = 9)</th>
<th>% Chances for ALT to be greater/similar/smaller than the SWC compared with CON</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Training load (arbitrary units)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>preintervention week</td>
<td>2260 ± 571</td>
<td>2553 ± 952</td>
<td>25/8/67 (CON possibly higher)</td>
</tr>
<tr>
<td>week 1</td>
<td>4765 ± 685</td>
<td>2961 ± 382</td>
<td>100/0/0 (ALT almost certainly higher)</td>
</tr>
<tr>
<td>week 2</td>
<td>4962 ± 313</td>
<td>3771 ± 848</td>
<td>99/0/0 (ALT very likely higher)</td>
</tr>
<tr>
<td>week 3</td>
<td>4536 ± 240</td>
<td>3631 ± 477</td>
<td>100/0/0 (ALT almost certainly higher)</td>
</tr>
<tr>
<td><strong>Training duration (min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>preintervention week</td>
<td>536 ± 118</td>
<td>446 ± 157</td>
<td>87/4/9 (ALT likely higher)</td>
</tr>
<tr>
<td>week 1</td>
<td>702 ± 95</td>
<td>562 ± 59</td>
<td>100/0/0 (ALT almost certainly higher)</td>
</tr>
<tr>
<td>week 2</td>
<td>748 ± 41</td>
<td>663 ± 115</td>
<td>92/4/4 (ALT likely higher)</td>
</tr>
<tr>
<td>week 3</td>
<td>597 ± 9</td>
<td>623 ± 29</td>
<td>0/11/88 (CON likely higher)</td>
</tr>
<tr>
<td><strong>Rating of perceived exertion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>preintervention week</td>
<td>4.84 ± 0.58</td>
<td>5.38 ± 0.68</td>
<td>1/4/95 (CON likely higher)</td>
</tr>
<tr>
<td>week 1</td>
<td>6.97 ± 0.26</td>
<td>5.01 ± 0.75</td>
<td>100/0/0 (ALT almost certainly higher)</td>
</tr>
<tr>
<td>week 2</td>
<td>6.35 ± 0.13</td>
<td>5.38 ± 0.48</td>
<td>100/0/0 (ALT almost certainly higher)</td>
</tr>
<tr>
<td>week 3</td>
<td>6.49 ± 0.38</td>
<td>5.64 ± 0.65</td>
<td>99/1/0 (ALT very likely higher)</td>
</tr>
</tbody>
</table>

Abbreviations: ALT, altitude-training group; CON, sea-level control group; SWC, smallest worthwhile change.

### Figure 2
Performance in 2000-m time-trial running at sea level (mean ± SD). Abbreviations: ALT, altitude-training group; CON, sea-level control group.
**Hbmass and Reticulocytes**

The mean (± SD) Hbmass in the ALT and CON groups before the intervention were 992 ± 129 g and 980 ± 151 g, respectively. Percentage change in Hbmass (ΔHbmass) from baseline is displayed in Figure 3. At POST1, mean %ΔHbmass (± 90% CL) was very likely increased in ALT (3.6% ± 1.6%), while changes in CON were trivial (0.5% ± 2.4%). The changes in Hbmass at POST1 were likely 2.8% ± 3.5% greater in ALT than in CON. When training load was used as a covariate, the mean %ΔHbmass was possibly 2.2% ± 7.7% higher in ALT than in CON. The individual variation in Hbmass at POST1 was 1.7% without training load used as a covariate and 1.3% (90% CL –4.2 to 4.8) when load was used as a covariate. Thirty days after the camp, Hbmass was not likely different from PRE in ALT (0.3% ± 1.6%). A Pearson correlation revealed a significant negative correlation (R = −0.484, P = .036) between initial Hbmass relative to body mass (RelHbmass) and %ΔHbmass postaltitude exposure in ALT (2 subjects who reported illness throughout the study removed from this analyses). Reticulocytes were almost certainly lower in ALT than in CON at POST1 (−26.2% ± 8.6%) and were almost certainly lower than PRE at POST2 in ALT (−64.5% ± 12.6%).

**Intramuscular Metabolites**

Intramuscular carnosine of ALT was almost certainly 35.5% ± 15.0% higher in the gastrocnemius (34.9 ± 8.6 AU) than in the soleus (22.4 ± 5.2 AU) at PRE. Changes in carnosine were trivial from PRE to POST1 in gastrocnemius (3.9% ± 9.0%) and unclear in soleus (−1.6 ± 13.1). The pooled carnitine and choline was likely 26.3% ± 15.5% lower in gastrocnemius (228 ± 60 AU) than in soleus (283 ± 50 AU) at PRE. Pooled carnitine and choline was likely increased in soleus (8.8% ± 6.1%) but trivial in gastrocnemius from PRE to POST1 (0.6% ± 14.1%).

**Discussion**

The main finding of the current study is that professional team-sport athletes undertaking an LHTH preseason training camp at moderate altitude had ~1.5% greater improvements in running performance than matched controls living and training at sea level. These performance improvements were accompanied by ~3% increase in Hbmass not observed in control subjects. Changes in Hbmass returned to baseline 4 weeks postaltitude, but improvements in running performance were maintained. A novel finding of the current study is that there was no clear change in intramuscular carnosine concentration after altitude exposure, suggesting that changes in this intramuscular protein may not be responsible for previously reported changes in muscle buffering capacity or that the changes were too small for magnetic resonance spectroscopy (MRS) measurements to identify.

**Performance**

The ~1.5% greater improvement in running performance in ALT over that observed in CON subjects in the current investigation is of similar magnitude to improvements seen in endurance athletes after altitude-training interventions. However, the individual variability in this study was approximately half of the mean change in TT performance, which is less than previously reported in endurance athletes. This suggests that team-sport athletes may experience more consistent improvements in performance than endurance athletes after an altitude intervention, which may be related to some endurance athletes’ approaching their physiological limits, with only small opportunities for improvements. It has previously been suggested that, after altitude exposure, athletes are able to train at higher intensities, thereby increasing the training stimuli and consequent improvements in performance. As improvements in running performance were maintained for at least 4 weeks postdescent in our subjects, we propose that an altitude-training camp may positively influence subsequent preseason training in team-sport athletes, thus improving preparation leading into the competitive season.

**Hbmass**

A change in the oxygen-carrying capacity of the blood is often proposed as the major physiological adaptation leading to improved performance after altitude exposure. In the current study, athletes achieved a mean increase in Hbmass of 3.6%, which is similar to changes observed in endurance cyclists after 19 days residing at 2760 m. Hbmass returned to baseline 30 days postdescent, which is also similar to the results of Garvican et al, suggesting that team-sport athletes can achieve improvements in Hbmass after an altitude intervention comparable to those observed in endurance athletes and that such changes follow a similar time course with ascent to altitude and descent to sea level. The depression of reticulocytes on return to sea level in the ALT group provides additional evidence of accelerated erythropoiesis after altitude exposure. Pottgiesser et al observed an ~20% depression of reticulocytes 9 days after 26 nights spent at 3000 m, which is similar to the 26% depression we observed 7 days postaltitude.

Although it is commonly accepted that prolonged exposure to hypoxic conditions can elicit increases in Hbmass, it is also acknowledged that there is high variability in this response between individuals. Similar to previous findings, our data demonstrate high interindividual variability, with individual responsiveness in Hbmass approximately half the magnitude of the mean change in ALT. The causes of such variability between individuals are not well understood and may be related to a number of factors. The proinflammatory cytokine interleukin 1 (IL-1) suppresses the release of erythropoietin, so athletes experiencing infections that lead to increases in IL-1 before or during an altitude camp may...
Figure 3 — Changes in hemoglobin mass (Hbmass) after altitude exposure (ALT, n = 21) or control conditions (CON, n = 9). Black circles and error bars show group change (%) as mean ± SD, and gray circles show individual responses. The letters a and b depict responses of 2 subjects who reported illness before (b) and during (a) altitude exposure.
have a blunted erythropoietic response. This is supported by data from the current study in which 2 athletes reported illness, either before or during altitude exposure, and neither athlete demonstrated an increase in Hbmass after the altitude-training camp (ΔHbmass = −0.8% and −2.7%, see Figure 3).

However, there is still large variability in the responses of our other subjects. Some have proposed that athletes starting with high initial Hbmass may have a limited ability to stimulate further increases.26 Robach and Lundby26 combined data from 9 altitude-training studies involving elite endurance athletes and found an inverse correlation between initial RelHbmass and change in Hbmass (R = .86, P < .01). Our results also show a significant inverse correlation between initial RelHbmass and percentage change in Hbmass after altitude exposure, but the magnitude was only about half that of Robach and Lundby.

Intramuscular Metabolites

While increased erythropoiesis appears to be an important adaptation with altitude exposure, it cannot completely account for increases in performance after such exposures, even in situations where the strongest relationships are observed.4 Nonhematological adaptations may also play a role in improved performance,3 including increases in muscle buffering capacity.5,6,8 However, such findings are not universal,25 and none of the aforementioned studies have been able to elucidate the mechanism responsible for this adaptation. Despite this, Saltin et al6 proposed that changes in muscle buffering capacity after altitude training may be due to changes in intramuscular carnosine, a hypothesis further supported by others.8 The current study is the first to assess intramuscular carnosine levels before and after an altitude-training intervention. However, we found no clear changes in carnosine content in the gastrocnemius or soleus after the altitude intervention. This finding may be due to the availability of β-alanine, which limits carnosine synthesis.19 If altitude exposure were to alter carnosine concentrations within skeletal muscle, such exposure would need to alter the availability of β-alanine, but there is no apparent mechanism for this to occur during altitude exposure. The current results, combined with the current understanding of factors limiting carnosine synthesis, would suggest that changes in carnosine are not responsible for previously observed changes in muscle buffering capacity, although muscle buffering capacity was not directly measured in the current study.

An interesting finding in our data was an increase in the carnitine/choline peak observed in soleus after the intervention period. The mechanism for such an increase is unclear, but as pooled carnitine and choline increased only in soleus, and not in gastrocnemius, we propose that these increases are related to changes in the mitochondria, which are found in higher concentrations in soleus due to the greater proportion of type I muscle fibers.28 Carnitine acts as an acceptor for the acetyl groups by forming acetylcarnitine when the rate of acetyl coenzyme A formation from glycolysis is high during high-intensity exercise.29 Therefore, we propose that possible increases in carnitine may be related to improved capacity for high-intensity exercise. Further research is needed to fully understand this finding, including whether this change was due to a training effect or altitude exposure, as the control group was not included in the MRS analysis in this study.

Limitations

One limitation in this study was the difference in training load observed between groups. However, although ALT subjects completed higher training loads than controls during the intervention period, covariate analyses suggest that improvements in performance and changes in Hbmass were still greater in ALT subjects when controlling for training load. Furthermore, using RPE-based methods to assess training load during LTHH interventions may overestimate the mechanical and physiological training load. When training in hypoxic conditions, RPE is higher than in similar training completed in normoxic conditions, even when training velocities and markers of physiological load (eg, oxygen consumption and heart rate) are lower.30 RPE was higher in the ALT subjects throughout the intervention period, and it is not possible to determine if this is merely due to perception of effort or if the ALT group actually completed higher-intensity training throughout this period. Although differences in RPE may account for some of the observed differences in training load, training duration was also ~12% higher in the ALT group.

Possible placebo, nocebo, and training-camp effects also cannot be eliminated from having an influence on the current results. However, while placebo and training-camp effects may confound scientific results, these changes are nevertheless of interest to practitioners looking to achieve the best possible performance outcomes during the preseason period.

Practical Applications

The current investigation was conducted in an ecologically valid environment with professional team-sport athletes; therefore, the findings have strong practical relevance. Our results show improved running performance in team-sport athletes completing a preseason LTHH intervention. Altitude exposure also increased Hbmass, and this benefit may be greatest in athletes starting with lower initial RelHbmass. Such physiological changes may lead to improved quality of training on returning to sea level, and although we found performance benefit for a month, practitioners should not expect physiological changes to be maintained throughout the duration of a team-sport season. Athletes experiencing illness leading into or during an altitude-training camp are also unlikely to see erythropoietic benefit from the altitude exposure.
Conclusions

Team-sport athletes completing a 19-day altitude-training camp experience greater improvements in running performance than athletes completing similar training at sea level. Improvements in running performance are maintained for at least 4 weeks postdescent and may therefore lead to improved quality of training throughout this period, thereby improving preparation for competition. Therefore, we conclude that a preseason altitude-training camp is a worthwhile intervention for improving running performance in elite team-sport athletes.

Acknowledgments

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