Heart Rate Variability in Responders and Non-Responders to Live-Moderate, Train-Low Altitude Training

Michael J. Hamlin¹, Apiwan Manimmanakorn, Gavin R. Sandercock², Jenny J. Ross, Robert H. Creasy³, John Hellemans⁴

Abstract—The aim of this study was to compare the effects of an altitude training camp on heart rate variability and performance in elite triathletes. Ten athletes completed 20 days of live-high, train-low training at 1650m. Athletes underwent pre and post 800-m swim time trials at sea-level, and two heart rate variability tests at 1650m on the first and last day of the training camp. Based on their time trial results, athletes were divided into responders and non-responders. Relative to the non-responders, the responders sympathetic-to-parasympathetic ratio decreased substantially after 20 days of altitude training (-0.68 ± 1.08 and -1.2 ± 0.96, mean ± 90% confidence interval for supine and standing respectively). In addition, sympathetic activity while standing was also substantially lower post-altitude in the responders compared to the non-responders (-1869 ± 4764 ms²). Results indicate that responders demonstrated a change to more vagal predominance compared to non-responders.

Keywords—parasympathetic predominance, poor performance, triathlon, 800-m swim

I. INTRODUCTION

AFTER the 1968 summer Olympic Games in Mexico City (2300 m) altitude training has become popular with athletes and coaches, but debate continues as to the effectiveness of real or simulated altitude training on subsequent sea-level performance [1]. Chapman et al. (1998) reported in a retrospective study that 17 athletes significantly improved sea-level performance, while 15 showed a decrement after 28 days of live-high (2500m) train-low (1250m) (LHTL) [2]. Evidence also exists for performance decrement after 28 days of live-high, train-low training at 1650m. Athletes underwent pre and post 800-m swim time trials at sea-level, and two heart rate variability tests at 1650m on the first and last day of the training camp. Based on their time trial results, athletes were divided into responders and non-responders. Relative to the non-responders, the responders sympathetic-to-parasympathetic ratio decreased substantially after 20 days of altitude training (-0.68 ± 1.08 and -1.2 ± 0.96, mean ± 90% confidence interval for supine and standing respectively). In addition, sympathetic activity while standing was also substantially lower post-altitude in the responders compared to the non-responders (-1869 ± 4764 ms²). Results indicate that responders demonstrated a change to more vagal predominance compared to non-responders.

TABLE I

<table>
<thead>
<tr>
<th>Characteristics of study participants</th>
<th>Responders (n = 6)</th>
<th>Non-responders (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>23.5 ± 4.2</td>
<td>21 ± 2.0</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>64.7 ± 7.4</td>
<td>66.1 ± 7.5</td>
</tr>
<tr>
<td>Height (m)</td>
<td>173.2 ± 6.4</td>
<td>175.3 ± 5.1</td>
</tr>
<tr>
<td>Sex</td>
<td>male 3, female 3</td>
<td>male 3, female 1</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.8 ± 1.3</td>
<td>21.8 ± 3.1</td>
</tr>
</tbody>
</table>

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Data are raw means ± SD.

Ten elite triathletes with international experience from the New Zealand Academy of Sport programme gave their written informed consent to participate in the study which met appropriate ethical standards [13] and was approved by the Lincoln University Human Ethics Committee. Participants’ characteristics are presented in Table 1. All participants were in their competition phase of training, and were using the altitude camp as a means of tapering towards the New Zealand National Triathlon champs in Wellington in March 2009.

Abstract—The aim of this study was to compare the effects of an altitude training camp on heart rate variability and performance in elite triathletes. Ten athletes completed 20 days of live-high, train-low training at 1650m. Athletes underwent pre and post 800-m swim time trials at sea-level, and two heart rate variability tests at 1650m on the first and last day of the training camp. Based on their time trial results, athletes were divided into responders and non-responders. Relative to the non-responders, the responders sympathetic-to-parasympathetic ratio decreased substantially after 20 days of altitude training (-0.68 ± 1.08 and -1.2 ± 0.96, mean ± 90% confidence interval for supine and standing respectively). In addition, sympathetic activity while standing was also substantially lower post-altitude in the responders compared to the non-responders (-1869 ± 4764 ms²). Results indicate that responders demonstrated a change to more vagal predominance compared to non-responders.
The athletes trained for 20 days under the supervision of their coach and team physiologist at the Snow Farm (altitude 1650m, Wanaka, New Zealand). The athletes lived at altitude and trained twice a day close to sea-level (Wanaka village, ~300m) on most days. On the first and last day of training athletes completed a 6-min heart rate variability test. These tests were performed in the morning, prior to training and before breakfast and were conducted in the same room, at temperatures that were similar throughout the study (15-18°C). Subjects were relaxed and had not ingested any coffee or tea prior to the testing. Performance was assessed by individual 800-m time trials at sea-level in a standard indoor 25 m pool conducted approximately 7 days before and 7 days after the altitude camp.

Training loads were calculated via the training impulse (TRIMP) method [14], which was expressed as a product of stress (duration of activity) and strain a 5-point Likert-type scale based on exercise heart rate; easy=1, steady=2, moderately hard=3, hard=4, very hard=5. The heart rate training zones were identified initially from laboratory-based cycling and running lactate tests, and confirmed or adjusted by training sessions performed in the field prior to the altitude training camp. The following training zones were established; easy, corresponding to heart rates where lactate was ≤ resting lactate concentration, steady = heart rate at the first lactate turnpoint, moderately hard = heart rate range between the first and second lactate turnpoint, hard = heart rates that matched the second lactate turnpoint (~4 mmol.L-1) and very hard = heart rate from the second lactate turnpoint to maximum heart rate.

Each test lasted for 6 min (3 in supine position and 3 in standing). The RR interval (time between two R waves of the recorded cardiac electrical activity) was measured with a Polar S810 heart rate monitor (Polar, Kempele, Finland) which shows good agreement with HRV determined from ECG [15]. The R-R series were analysed using Kubios HRV software (version 2.0, Biosignal Analysis and Medical Imaging Group, University of Kuopio, Finland) which was developed in accordance with published recommendations [16]. The software shows excellent validity [17], and is able to account for non-linear trends often present in beat-to-beat recordings by detrending the filtered R-R data using the smoothness priors approach [18]. Data were interpolated at a rate of 4 Hz in accordance with the software’s recommendations [17]. In the frequency domain, total power (TP) low frequency (LF) and high frequency (HF) spectral power were calculated in raw units using internationally agreed spectral bands [16]. The ratio of low to high frequency power (LF/HF) was also calculated. In the time domain, the root mean square of the standard deviation of normal to normal interval differences (RMSSD) were obtained. All HRV indices were calculated from R-R interval recordings made in both the supine (su) and standing (st) positions.

We used a contemporary statistical approach because small performance changes can be beneficial for elite athletes [19], whereas conventional statistics can be less sensitive to such small but worthwhile changes. Specifically, we used magnitude-based inferences about effect sizes, and then to make inferences about true (population) values of the effect, the uncertainty in the effect was expressed as 90% confidence limits (CL). The unequal variances t statistic was used to analyse differences in the mean change between groups. The probability that the true value of the effect was practically negative, trivial, or positive accounted for the observed difference, and typical error of measurement [20]. The smallest worthwhile change in 800 m performance was assumed to be a reduction or increase in performance time of more than 1.0%, based on previous research into elite triathletes swim section [21]. For the HRV measures, the value was determined by multiplying the baseline between-subject standard deviation by Cohen’s value of the smallest worthwhile effect of 0.2 [22]. Effects that were simultaneously both >75% likely positive and <5% negative were considered substantial and beneficial. An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative, or beneficial and harmful. The relationship between the post-pre change in performance and the change in neurovegetative state (LF/HF ratio) was analysed using linear regression analysis.

III. RESULTS

One subject’s HRV data was not analysed due to large aberrations in the data that were probably caused by excessive movement. Training loads between groups (396.4 ± 171.4, 387.4 ± 164.1, mean ± SD, for the non-responders and responders respectively) over the study period were similar with little difference between groups observed (8.9 ± 52.4, mean ± 90% CL). However, in response to this similar training load, 6 athletes improved their performance (3.0 ± 2.4%, mean ± SD) and 4 showed decreased performance (-1.8 ± 1.2%).

Overall there was little change in any of the HRV parameters during the course of the altitude training camp (Table I). However, when comparing the responders to non-responders over the training period a number of substantial differences were found. Relative to the non-responders, the responders LF/HF ratio both in the supine and standing positions decreased substantially after the 20 days of altitude training (Table II). In addition, LFst (standing) was also substantially lower post-altitude training in the responders compared to the non-responders (-1869 ± 4764 ms², mean ± 90% CL).
Table II
Heart Rate Variability in Supine (SU) and Standing (ST) Positions and Swimming Performance in All Subjects Before and Immediately After 3 Weeks of Altitude Training

<table>
<thead>
<tr>
<th></th>
<th>All Subjects</th>
<th></th>
<th>Differences;</th>
<th>Chances that the true differences</th>
<th>Qualitative inference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>± 90% CL</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>800-m (s)</td>
<td>597.4 ± 35.6</td>
<td>591.9 ± 26.9</td>
<td>-5.6; 26.2</td>
<td>48</td>
<td>Unclear</td>
</tr>
<tr>
<td>LFsu</td>
<td>2279 ± 1537</td>
<td>1793 ± 2333</td>
<td>-485; 1744</td>
<td>53</td>
<td>Unclear</td>
</tr>
<tr>
<td>HFsu</td>
<td>2064 ± 1556</td>
<td>3295 ± 3239</td>
<td>1230; 2305</td>
<td>70</td>
<td>Unclear</td>
</tr>
<tr>
<td>LF/HFsu</td>
<td>1.22 ± 0.47</td>
<td>0.86 ± 0.87</td>
<td>-0.36; 0.62</td>
<td>72</td>
<td>Unclear</td>
</tr>
<tr>
<td>TPsu</td>
<td>6884 ± 4768</td>
<td>6949 ± 5288</td>
<td>65; 4322</td>
<td>35</td>
<td>Unclear</td>
</tr>
<tr>
<td>RMSSDsu</td>
<td>81.7 ± 40.5</td>
<td>86.9 ± 46.4</td>
<td>5.3; 37.4</td>
<td>43</td>
<td>Unclear</td>
</tr>
<tr>
<td>LFs</td>
<td>1660 ± 1151</td>
<td>2277 ± 2504</td>
<td>617; 1650</td>
<td>58</td>
<td>Unclear</td>
</tr>
<tr>
<td>HFst</td>
<td>1576 ± 1076</td>
<td>2813 ± 3026</td>
<td>1237; 1962</td>
<td>74</td>
<td>Unclear</td>
</tr>
<tr>
<td>LF/HFst</td>
<td>1.31 ± 0.78</td>
<td>1.13 ± 0.80</td>
<td>-0.18; 0.65</td>
<td>51</td>
<td>Unclear</td>
</tr>
<tr>
<td>TPs</td>
<td>4831 ± 2888</td>
<td>7099 ± 6323</td>
<td>2268; 4161</td>
<td>69</td>
<td>Unclear</td>
</tr>
<tr>
<td>RMSSDst</td>
<td>75.2 ± 39.1</td>
<td>82.0 ± 49.6</td>
<td>6.8; 36.9</td>
<td>45</td>
<td>Unclear</td>
</tr>
</tbody>
</table>

Data are raw means ± SD. Pre, day 1 of altitude training; Post, day 20 of altitude training; 800-m, time to complete 800-m swim time trial 1 week before (pre) and 1 week after (post) altitude camp; LF, low frequency reflecting sympathetic predominance; HF, high frequency reflecting parasympathetic predominance; LF/HF, sympathetic-parasympathetic neurovegetative balance; TP, total power (addition of LF and HF); RMSSD, root mean square of the standard deviation of the R-R intervals. aBased on a smallest substantial change of 1% for performance and 0.2 for all other measures. ± 90% CL: add and subtract this number to the mean effect to obtain confidence limits for the true difference.

IV. DISCUSSION
The aim of this study was to compare the effects of an altitude training camp on HRV and performance of elite triathletes. In addition, the study sought to determine whether HRV could indicate which athletes benefit from altitude training and improve performance and which athletes do not respond and suffer performance decrements. Results indicate that athletes who improved performance as a consequence of the altitude training also showed a negative shift in the LF/HF ratio.

The effect of LHTL training is a matter of debate with some researchers finding insignificant sea-level performance improvements [3], while others have found clear improvements [23], or both improvements and decrements in the same athletes under similar conditions [24]. A possible reason for this performance variation may be due to individual differences to the training and hypoxic load. Large inter-individual variations have been reported in many physiological responses to hypoxia including the degree of ventilatory drive [25] and erythropoietin release [26]. Such variation may create an environment where some athletes might find the demand of coping with altitude in addition to normal training too stressful and maladaptation may ensue, while others may thrive, giving rise to the performance variation observed.

In a recent study, Schmitt and colleagues (2006) reported decreased LF/HF ratio’s in both the supine and standing positions after 11 days training at 1200 or 1850 m, (significantly different only at 1850 m). Others have reported a decrease in LF coinciding with an increase in HF towards the end of altitude acclimatization [27]. These results are in agreement with the present study in which we found a substantial lowering of the mean overall LF/HF ratio after 20 days of live-high train-low training in the responders compared to the non-responders. Some of the lowered LF/HFst ratio in the responders was due to withdrawal of the sympathetic innervation, as witnessed by the substantially lower LFst in these subjects.
The lack of a substantial increase in the LF/HF ratio when moving from supine to standing (approximately 7% and 31% for the supine to stand increase for pre and post testing respectively) is surprising, given that such an orthostatic challenge would normally increase sympathetic stimulation significantly. We speculate that this may be due to the effects of hypoxia at altitude. Although the effects of acute and chronic hypoxia on cardiovascular changes during an orthostatic challenge are relatively un-researched, it is possible that the increase in respiration [28], [29] changes in cardiac output [30], or hormonal release [31], upon accent to altitude secondary to the decreased partial pressure of inspired oxygen may have led to changes in the cardiovascular response to such an orthostatic challenge in our athletes. Further research on these elite athletes during altitude training is required to understand such mechanisms.

The effects of hypoxia and physical training on HRV show similarities. An increase in short-term training load can negatively influence vagally-mediated HRV indices [32], whereas medium-term endurance training increases vagally mediated HRV indices [33]. The athletes in this study that did not respond positively to the 20-days LHTL showed a withdrawal of vagally-mediated HRV parameters similar to Mourot and colleagues (2004) observations, which suggests that HRV may be a useful tool at identifying athletes under too much stress, whether it is from training alone, or a combination of training and hypoxia.

It seems likely that the effects of high training loads with the addition of hypoxic stress has a cumulative effect, such that the normal adaptation to such stressors is overwhelmed and sympathetic activity predominates. If recovery is not sufficient and training and hypoxia continue, the athlete has little chance of recovery and gradually shifts into a sympathetically innervated overtress-type syndrome causing a reduction in performance potential. It would be expected that removal of some of this stress (either the hypoxia or the training load) may improve this condition and subsequent performance, however this is speculative and further research is required to elucidate these changes.

A limitation of the study is the lack of a sea-level control group which does not allow us to distinguish whether the observed effects are due to the altitude or to the effect of training. However, we consider our observations are worthwhile given the lack of information on such groups (elite
HRV during altitude training may be a useful tool in establishing adequate training (and hypoxic) stress. It is possible that with such a tool individual altitude and training stimuli may be adjusted appropriately for maximum effect.

REFERENCES


