The challenge of assessing athlete performance after altitude training

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Chapman et al. (2) have compiled one of the largest ever altitude training studies in an attempt to quantify an optimal altitude. Their conclusion that the sweet spot for training is between 2,000 and 2,500 m is supported by their data but warrants further consideration. Four key areas for interrogation are red cell volume (RCV), maximal \( \dot{V}O_2 \) uptake (\( \dot{V}O_{2\text{max}} \)), performance, and measurement precision, which are interrelated.

**RCV**

The finding that 28 days of live high-train low (LHTL) increases RCV by \( \sim 7\% \) at 1,780 m to the same extent as at 2,800 m (2) suggests that the threshold to accelerate red cell production is lower than the 2,000 m suggested previously (9). A recent meta-analysis (7) concluded that the threshold is \( >3,000 \) m, but another attempt (5) using a more precise technique to estimate hemoglobin mass (Hbmass) suggests that, at 2,320 m, red blood cell production increases \( \sim 1\%/100 \) h of exposure. Collectively, the data of Chapman et al. (2) and Gore et al. (5) support the concept that altitude training increases red cell production, even among athletes who are already well-trained.

It is surprising that the increase in RCV immediately post-altitude was similar at all four altitudes [Chapman et al. (2), Fig. 8], despite a difference of 1,020 m. Rasmussen et al. (7) modeled a greater increase in RCV at higher altitudes, albeit only after the 3,000-m threshold is surpassed. It is mechanistically appealing that red cell production might be accelerated more aggressively at higher altitudes. In support of Chapman et al. (2), Gore et al. (5) found that Hbmass increased similarly at 2,320 m (live and train at altitude) and at 3,000 m (simulated altitude for 14 h/day), but both modes exceed the conventional 2,000-m threshold (9). Pooling the findings of Chapman et al. (2) for RCV and Gore et al. (5) for Hbmass suggests that the rate of red cell production is similar between 1,800 and 3,000 m. However, further data are needed to titrate the lower boundary at which an acceleration of red cell production is attained. Such data will require a technique with high precision; CO rebreathing for Hbmass or \( ^{51} \)Cr-labeled red blood cells are both suitable (3).

**\( \dot{V}O_{2\text{max}} \)**

Cross-sectional studies show that \( \dot{V}O_{2\text{max}} \) increases directly with Hbmass, such that each gram change in Hb is associated with a change in \( \dot{V}O_{2\text{max}} \) of \( \sim 4 \) ml \( O_2/\text{min} \) (11). Therefore, based on changes of RCV/Hbmass, one expects similar increases in \( \dot{V}O_{2\text{max}} \) for each of the four altitudes studied by Chapman et al. (2). Saunders et al. (10) pooled data of 145 athletes and determined that \( \dot{V}O_{2\text{max}} \) changes by about one-half the magnitude of the increase in Hbmass. It is thus incongruous that for 1,780 m the increase in \( \dot{V}O_{2\text{max}} \) was \( \sim 2\% \), whereas that at 2,800 m was \( \sim 5\% \), despite a similar 6–7% increase in RCV. This research group has previously reported a typical error (TE) of 1.6% for relative \( \dot{V}O_{2\text{max}} \) (8) and 6.7% for RCV (4); the latter consistent with a TE of \( \sim 7\% \) derived from meta-analysis (3). TE is the within-subject standard deviation, so the 95% confidence interval for change can be calculated as \( \pm \sqrt{2} \times \text{TE} \times 1.96 \). Therefore, for \( \dot{V}O_{2\text{max}} \), the 95% confidence interval for change is approximately \( \pm 4\% \), whereas that for RCV is approximately \( \pm 19\% \). That is, in the study of Chapman et al. (2), 5% of the time there will be random individual changes in \( \dot{V}O_{2\text{max}} \) of \( >4\% \) and of RCV \( >19\% \). So it follows that, with \( \dot{V}O_{2\text{max}} \), there is more precision about the result, whereas RCV has more imprecision, which, with small samples, will make it more difficult to discern the magnitude of the true response. The inference being that the results of Chapman et al. (2) for RCV may include some aberrant individual changes that have spuriously elevated the mean response. This interpretation is not to discount the finding that RCV increased at 1,780 m, rather that the error-free magnitude of increase may not have been as large as \( 7\% \) (5). Specifically, using the results of Saunders et al. (10), and based on the more precisely measured change in \( \dot{V}O_{2\text{max}} \) of \( 2\% \) by Chapman et al. (2), it is tempting to surmise that the error-free increase of RCV at 1,780 m was closer to \( \sim 4\% \).

**Performance**

Blood doping studies indicate that transfusion of two units of red blood cells (\( \sim 50 \) g of Hb per unit) increases performance in trained athletes; for instance, by 1.7% in 1,500-m run (1). It is, therefore, curious that only those groups from 2,085 and 2,454 m, improved 3,000-m time-trial performance, when all four groups, including the two from 1,780 and 2,800 m, increased RCV by a similar amount (\( \sim 6–7\% \)). A plot of all data, regardless of altitude, for the percent change in RCV vs. the percent change in time-trial performance might have been a relevant way to further interrogate the strength of association between the two measures.

There is good insight in the suggestion of Chapman et al. (2) that training response may have been poorer at 2,800 m, which compromised postaltitude performance. But why did the group from 1,780 m also fail to improve performance immediately postaltitude, despite increased RCV by \( \sim 7\% \) and when they would not have had relatively compromised training? One potential confounder is that there are limitations to simulating performance in the laboratory or field, compared with actual racing. For instance, Wilmore (12) demonstrated benefit of additional motivation for cycle ergometer performance. Many athletes attend World Championship or Olympic Games striving for a personal best performance, but generally \(<10\% \) manage to do so, such as at the 2013 World Championships (S. Hollings, Athletics New Zealand, personal communication). It is, therefore, unsurprising that, after an experimental intervention such as LHTL, there is a wide range of performance changes postaltitude; for example, that one-half of the 2,800-m
group ran faster and one-half slower after LHTL [Chapman et al. (2), Fig. 3]. In track running races, Hopkins and coworkers (6) have demonstrated that the coefficient of variation for performance (time) is ~1%, but is approximately twice as large (2–3%) in slower runners. It would have been interesting if Chapman et al. (2) used their two baseline time trials to quantify the within-subject variation for performance of their collegiate-level runners, since, in a simulated race setting, it is likely that there remains sufficient imprecision to detect small changes. One way to reduce the effects of variable motivation is to use actual race performances to understand the effects of altitude training, not just for running, but for a wide range of sports. However, to mitigate against the effects of tactics in some types of races would require a number of races to be considered, or the use of races with individual time-trials, such as cycling.

In conclusion, the similar increase in Hbmass across all studied altitudes is unexplained and moreover is associated with different performance outcomes, raising new questions about the physiological basis of improved sea level performance after LHTL. It is also probably premature based on this study to rule out 2,800 m (or higher) as being unsuitable for LHTL altitude training. The physiological changes of Hbmass suggest that 2,800 m could be just as efficacious as the lower altitudes, providing that training is conducted appropriately.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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