Dear Editor-in-chief

We read with profound interest the article titled ‘Increased hypoxic dose after training at low altitude with 9h per night at 3000m normobaric hypoxia’ by Carr et al. (2015). Authors have concluded that low altitude (1380 m) combined with normobaric hypoxia of 3000 m improves total haemoglobin mass (Hbmax) and is an effective alternate method for training. Like other studies on elite athletes, the authors of present work have brought out that a major limitation was non-availability of a control group consisting of subjects undertaking same supervised training at normoxia. The total number of ‘possible’ subjects for control group which were taken from a previous study (Saunders et al., 2010) was 11 i.e placebo group (n = 6; 3 male and 3 female) and Nocebo group (n = 5; 3 female and 2 male). It seems likely that authors of the present study have chosen only 10 subjects out of those 11. The criteria for exclusion of one subject and selection of 10 out of 11 subjects from the previous study to form the control group of the present study may require further elaboration.

Authors have mentioned that Hbmax of the subjects was measured within 1 week after the training period but have inadvertently not mentioned the time period between the end of training period (21 days) at Thredbo (1380m) and evaluation for VO2max at Australian Institute of Sport, Canberra (600m). The period between high altitude (HA) training and assessment of VO2max is likely to have an impact on effect of training on VO2max. Description of this period could enable the readers to have a better understanding of the phenomenon of effects of HA training on VO2max.

It is a well-known fact that VO2max is influenced by intrinsic motivation of an athlete undertaking the exercise test (Shin et al., 2014). In the present work, elite athletes of both low altitude-training (lowHH) and low-altitude and simulated altitude (lowHH+NHNight) groups were not blinded and were expectedly well-versed with the benefits of hypoxia training. This knowledge would have probably affected their performance on the treadmill. This possible bias could have been avoided by blinding the subjects of lowHH group of their ‘hypoxia exposure’ status by use of hypoxic tents for them as well but retaining the fraction of inspired Oxygen at 21%.

An important aspect of HA Physiology is HA related illnesses occurring in lowlanders on sudden exposure to HA. As Physiologists with areas of interest in HA and exercise Physiology, knowing the ethnic characteristics of the subjects would enhance application of findings of this study in other ethnic groups. Hypoxia training of individuals of genetically different groups can give varied outcomes in terms of improvement in Hbmax and VO2max (West 2004).

Also, it would have been interesting to know if any of the race-walkers from lowHH+NHNight group, who were given exposure to hypoxia of 3000m, reported for acute mountain sickness (AMS) on the morning of day2. AMS is a common HA illness occurring after 6-12 h of hypoxia exposure in unacclimatized lowlanders (Bärtsch and Swenson et al., 2013; West, 2004). The negative effects (in terms of AMS) of simulated hypoxia equivalent to ascents above 2000-2500m are seen in athletes especially if they are exposed to hypoxia at ascent rates faster than 100m/day (Schommer et al., 2012). A description of symptoms of AMS experienced by lowHH+NHNight group in this case would have comprehensively covered the possible limitations associated with hypoxia training in athletes.

Authors have reported the results of their work in terms of post minus pre intervention changes in each parameter of the three groups (Table 1 of the study). It would have been nice if actual mean values of these measures (before and after intervention) were also presented in the article. This would have further elucidated the beneficial effects of combination of low altitude training and hypoxic tents in athletes and also facilitated better comparison with other studies.

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References

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