Discerning normobaric and hypobaric hypoxia: significance of exposure duration

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TO THE EDITOR: We read a recently published article (4) with great interest. The authors performed a rigorously controlled study scrutinizing the possible differences in cardiorespiratory responses, control of breathing, and acute mountain sickness (AMS) incidence during short-term (6 h) exposures to hypobaric (HH) and normobaric hypoxia (NH). We commend the authors for their smart experimental design, which in addition to a normobaric normoxic (placebo) exposure also incorporated a hypobaric normoxic exposure, often lacking in these kinds of experiments. Although this work significantly advances our current understanding of the potential effects of hypobaria per se and adds an important step to unfolding the highly debated potential differences between “real” (HH) and “fake” (NH) altitude (3), we would like to briefly comment on two aspects of their study.

First, although the authors provide rationale for the chosen exposure duration (6 h) and also comment on that feature in the discussion, we believe that the duration of the exposure (i.e., total hypoxic dose) might very well be one of the key factors influencing the subsequent results for both cardiorespiratory responses and AMS. Although the differences between HH and NH in cardiorespiratory responses have already been demonstrated after exposures as short as 40 min (5), recent work suggests that the disparity in the effects of the two might predominantly be observed after exposures of more than 6 h in duration (2). Indeed, the study by Faiss et al. (2) clearly demonstrates that the main differences between HH and NH in ventilation and tidal volume are noted after 8 h during a continuous 1-day hypoxic exposure.

Second, the exposure duration might have an even greater impact when assessing the possible differences between HH and NH in AMS incidence and severity. Previous studies showing higher AMS in HH compared with NH have used longer (>8 h) exposures compared with Richard et al. (4) and that might explain the discrepancies in the findings. This notion is further supported by the fact that, in humans, AMS (at least at moderate to high altitudes) becomes fully manifested after 6–96 h of the hypoxic/altitude exposure.

Although the final passage of the Richard et al. (4) paper states that “the differences between responses to NH and HH may not be as significant as previously observed” and this conclusion is fully supported by their data, we believe that this notion cannot be directly translated to exposures of longer duration (e.g., higher cumulative hypoxic dose) or generalized across a broad range of hypoxia/altitude application. Based on the above, we strongly believe that further strictly controlled studies comparing HH and NH during exposures of longer duration are warranted to provide additional information on the possible differences between the two that might be of applied and/or clinical value. For example, a recent meta-analysis (1) suggested that the living-high training-low (LHTL) method would induce greater performance gains in athletes when sleeping in HH than NH, but the recommended repeated hypoxic/altitude exposures are of significantly longer duration (typically ≥12 h/day) than those used in the study by Richard et al. (4). This merits further focus on the topic.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

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