The following letter is in response to the Point:Counterpoint series “Positive effects of intermittent hypoxia (live high:train low) on exercise performance are/are not mediated primarily by augmented red cell volume” that appeared in the November 2005 issue (vol. 99: 2053–2058, 2005; http://jap.physiol.org/content/vol99/issue5).

To the Editor: Excellent discussion (5). However, the evidence for [Hb] changes as being the main factor accounting for the effects of altitude training on performance is weak, because the increase in [Hb] (5-10%) explained only 16% of the variance in the increase in $V_{O_2,max}$. In addition, the increase in $V_{O_2,max}$ only minimally explained the variance of the improvement in performance. Moreover, the following facts deserve further attention. First, [Hb] increased by twice as much in the HiHi (live high:train high) than in the HiLo (live high:train low) groups, whereas the increase of $V_{O_2,max}$ was similar in both groups. Second, it is not clear why the HiHi group did not improve performance despite the increase of $V_{O_2,max}$ and [Hb]. Third, some subjects appear to improve their $V_{O_2,max}$ more than accountable only by the increase in [Hb]. Fourth, blood transfusion studies show more marked effects on endurance (performance) than on $V_{O_2,max}$ and report a reduction of submaximal exercise blood lactate concentration after transfusion. Altitude training appears not to have significant effects on submaximal blood lactate concentration (4). Fifth, isovolemic (1, 2) and hypervolemic (3) changes in [Hb] are consistently counterbalanced by changes in cardiac output and skeletal muscle blood flow such that for a given O2 demand, O2 delivery is maintained constant, but only during submaximal exercise. In Levine et al. (4), cardiac output was not affected by the increase of [Hb] when running at 8 and 10 mph (~60 and 76% of their posttraining $V_{O_2,max}$), but it was reduced when running at 12 mph (~90%). Thus the discussion remains open.

REFERENCES

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