sponges and performance were mainly conducted in HH (7, 16, 28, 30). Nevertheless, it remains to be confirmed whether the benefits of training would be greater following training in HH compared with NH as suggested by the current literature (13). This assumption is supported by the results of a meta-analysis (4) in which a “terrestrial” LHTL protocol (i.e., HH) induced additional benefits in performance (estimated by change in power output) of 4.0% and 4.2% for elite and non-elite athletes vs. 0.6% and 1.4% with “artificial” LHTL (i.e., NH).

On the basis of the existing data relating to ventilatory responses, fluid balance, AMS severity, NO metabolism, and performance improvement in HH vs. NH, there is no doubt that hypobaric hypoxia induces different physiological responses from normobaric hypoxia. However, the main mechanisms remain unclear.

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COUNTERPOINT: HYPOBARIC HYPOXIA DOES NOT INDUCE DIFFERENT RESPONSES FROM NORMOBARIC HYPOXIA

Studies on hypoxia are performed by lowering ambient oxygen partial pressure (PO2) either by reducing the barometric pressure (hypobaric hypoxia) or by lowering the O2 fraction [normobaric hypoxia at the prevailing barometric pressure (PaO2)]. Upon reflection we can see that many land-
Semantics considerations. The first remark we can make is that the physiological responses induced by hypoxic or normobaric hypoxia are different, whereas this Counterpoint will present evidence arguing that these physiological responses are indeed equivalent.

Interchangeability between normobaric and hypobaric hypoxia. The carotid bodies, located at the bifurcation between the internal and external carotid arteries, are oxygen sensors. As such, they respond to a wide range of arterial partial pressure of O2 (Pao2) available to any cell, tissue, or organism (21) and in that respect is independent of changes in PaO2. Hypoxia can be either continuous or intermittent; continuous hypoxia being generally encountered during high altitude exposure, i.e., hypobaric hypoxia. On the other hand, intermittent or transient hypoxia as experienced under various clinical conditions, such as obstructive sleep apnea (OSA) or stroke, is always characterized by hypoxic/ischemic episode(s) irrespective of the ambient pressure. These two conditions also highlight the two extremes of the spectrum of hypoxic levels, OSA representing a systemic hypoxia whereas stroke is more local.

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proposed to explain the putative physiological differences between these two modalities of hypoxia.

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REBUTTAL FROM MILLET, FAISS, AND PIALOUX

Mounier and Brugniaux began their Counterpoint (5) by defending the idea that hypobaric (HH) and normobaric (NH) hypoxia induced equivalent physiological responses and concluded that if differences did exist, they were too small to be clinically relevant. Regardless of the semantic considerations proposed by our opponents, we are convinced that differences exist between HH and NH (4).

We agree that oxygen sensing is an important key to altitude adaptations as it was highlighted by Brugniaux and Mounier (2), and we are in agreement with the pivotal importance of HIF-1α in these adaptations. Epo data drawn from the meta-analysis of Bonetti and Hopkins [(1) Fig. 1a] may suggest a higher response of Epo production in natural altitude than in normobaric artificial altitude. However, the number of studies analyzed (n = 11) was too low to conclude any difference between NH and HH. In addition, the very large intervariability in HIF-1α responses to hypoxia (6) suggests that only a protocol designed for a paired statistical analysis using perfectly matched high “hypoxic doses” may provide an answer regarding the different HIF-1α responses between HH and NH. A similar scientific approach may also be relevant to assess the differences between NH and HH individual susceptibility to acute mountain sickness (AMS). In fact, although the individual history in real altitude conditions remains the best predictor of AMS (8), different equations have been proposed for both HH and NH tests (7). This kind of protocol is also necessary to compare the efficiency of HH and NH for the pre-acclimatization treatment for AMS because there are not any internationally recognized “gold standard” protocols or recommendations. Because, for practical reasons, NH interventions will continue to be recommended in many circumstances, it is time to investigate beyond the “oxygen sensing” or “equivalent air altitude” (2) paradigms. This may prevent the reproduction of past errors done in the field of altitude physiology (10) because the physiological adaptations to hypoxia are very complex and not limited to a single function (3, 9). So, we encourage further investigations to better understand the clinical implications of the observed differences between HH and NH.

To conclude, we agree that the clinical evidence regarding the differences between HH and NH is still lacking in this field of medicine and sport performance. This may due to very large interindividual variability in the responses to hypoxia. Out of the few studies directly comparing HH vs. NH, none were