NEW METRIC FOR THE HYPOXIC STIMULUS, NOT FOR THE RESPONSE

TO THE EDITOR: The proposal by our well-respected colleagues (2) to introduce a new metric—incorporating the altitude elevation and the total exposure duration, termed “kilometer hours”—for better describing the “hypoxic dose” is decidedly a step forward. By only quantifying the “external” stress, this metric presents several limitations: It suggests a linear relationship between altitude elevation and saturation decrease [but the Fick curve is curvilinear (3)] or that it applies to all athletes irrespectively of their training background [but elite endurance athletes suffer the largest decrease in VO2max (1)], altitude experience [but elite athletes who have had previous hypoxic exposure better adapt to hypoxic condition (4)], or type of hypoxia [but hypobaric vs. normobaric hypoxia induces larger desaturation (5)].

The large intersubject variability in the physiological responses to a given “hypoxic dose” implies that the magnitude of the stimulus rather than the altitude elevation should instead be considered. We therefore propose a new metric based on the sustained duration at a given arterial saturation level. Hence, desaturation levels in normoxia (exercise-induced arterial hypoxemia) or in hypoxia (3) predict the decrement in VO2max in this metric termed “saturation hours” is defined as %H = (98/s - 1) × h × 100, where s is the saturation value (in %) and h the time (in hours) sustained at any second level.

Practically, with the development of new sport gears incorporating the oximeter inside the textile, this metric will readily be measured without any disturbances to individuals.

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COMMENTARY ON VIEWPOINT: TIME FOR A NEW METRIC HYPOXIC DOSE?

TO THE EDITOR: It is a very good idea of Garvican-Lewis, Sharpe, and Gore (2) to initiate this discussion with a new metric hypoxic dose by combining living altitude in kilometers with hours spent at altitude (“kilometer hours”: km·h) in the field of altitude training science. However, is the altitude component of the dose really linear? Would exposure at lower altitudes overestimate the hypoxic dose because some sort of “altitude-threshold” exists? Physiological mechanisms behind a possible “altitude-threshold” could be associated with the s-shape of the oxyhemoglobin saturation curve: at altitudes above ~2,000 m the desaturation of athletes would occur on the steeper part of the curve resulting in more substantial increases in sEpo (~90% at 2,400 m compared with ~30% at 1,800 m after 24 h) (1). As the authors indicated, most recommendations for natural living altitudes are between 2,000 and 2,500 m (2, 5). Our Swiss experiences are that only ~1/6 of the endurance athletes living at 1,800 m (4) and 2/3 living at 2,200 m (3) have a substantially increased hemoglobin mass after a 3-week altitude training camp. Could the proposed method be “optimized,” if “kilometer” is weighted in a way, that there is a larger dose-difference for altitudes below and above an “altitude-threshold”? For example, start counting kilometers above 1,300 m, with double hours at 1,800 m needed to reach the same “dose” as compared with 2,300 m. Additionally, for elite sport settings, one should also keep in mind that the hemoglobin mass-response to a given “dose” has been shown to be largely idiosyncratic, thereby requiring individualized recommendations (3, 4).

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with all athletes showing a positive response. Siebenmann et al. (4) reported no change in average Hbmass after 1,328 km·h (4 wk, 16 h/day, 3,000 m). This greater dose was beneficial for some athletes, but trivial or detrimental for others, leading to no change on average. With an average 6% increase in red cell mass volume, Chapman et al. (1) did not show any dose response effect after 4 wk “living high, training high and low” between 1,780, 2,085, 2,454, and 2,800 m. Their study suggests that increasing the dose by increasing the altitude above optimum may not provide any benefit (1). After more extreme hypoxic dose, a 72-day self-supported Mt. Everest expedition (>9,000 km·h), Cheung et al. (2) reported a wide scale of positive, negative, and no change responses in Hbmass. Thus the suggested model and the present literature, analogously with our own unpublished data using the km·h approach, rather highlight the need for careful evaluation of all factors influencing athletes’ adaptation than solves the problem of how to determine hypoxic dose in elite sports.

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COMMENTARY ON VIEWPOINT: TIME FOR A NEW METRIC FOR HYPOXIC DOSE?

TO THE EDITOR: Although exposure to some effective dose of hypobaric hypoxia provides a clear stimulus to increase hemoglobin (Hb) mass (3), numerous physiological responses to normobaric hypoxia have well documented differences to hypobaric hypoxia (4). Because of these discrepancies, we believe the conditions should not be treated as equal, and other meta-analyses (e.g., Ref. 1) have differentiated between “natural” and “artificial” hypoxic exposures. Additionally, given that all but one of the included studies consisted of highly trained subjects, the authors may wish to exclude the Siebenmann et al. study (5), which described subjects as “sedentary to moderately trained individuals who were not involved in high-level sport.” Finally, the model would benefit from a clear establishment of a minimum threshold, both from an altitude and a duration perspective, as the authors note both short duration high/extreme altitude exposure and chronic residence at mild altitude are each ineffective at increasing Hb mass.

We would be excited to see an expanded model that accounts for the above concerns, thus addressing the sensitivity in what is already a thin air of certainty in regards to hypoxic training.

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COMMENTARY ON VIEWPOINT: TIME FOR A NEW HYPOXIC DOSE?

TO THE EDITOR: Guidelines for simulated altitude exposure suggest athletes should spend around 14 h per day at 3,000 m for 3 weeks (300 h of exposure) to observe a mean increase in hemoglobin mass of 3–5% (3). Similarly, hypoxic exposure for 3–4 weeks at >2,200 m altitude will elicit a 3–5% increase in hemoglobin mass (2), with 4 weeks exposure believed to accelerate erythropoiesis (4). Hypoxia in both these occasions is influenced by altitude and the duration of hypoxia. The new metric of hypoxic dosing (1) addresses this problem, ensuring standardization of the hypoxic dose at various altitudes and hence will allow for comparing physiologic and nonphysiologic effects on body systems. The hypoxic dose as per the new metric for the studies mentioned above will be 882-1,478 km-hour (2, 3). There have been questions regarding the minimum altitude and the extent of duration that results in “hypoxic dose.” There is influenced by altitude and the duration of hypoxia. The new metric of hypoxic dosing (1) addresses this problem, ensuring standardization of the hypoxic dose at various altitudes and hence will allow for comparing physiologic and nonphysiologic effects on body systems. The hypoxic dose as per the new metric for the studies mentioned above will be 882-1,478 km·h (2, 3). There have been questions regarding the minimum altitude and the extent of duration that results in “hypoxic dose” for physiologic changes to occur. The new metric is a good starting point that combines altitude and duration to measure outcomes across studies. The hypoxic dose per the new metric is predominantly in the range of 600-1,500 km-hour that results in 3–6% change in hemoglobin mass across multiple studies (1). As the relationship between altitude and hypoxia is not exactly linear and various factors could influence physiologic adaptation or training performance, knowing the baseline (“hypoxic dose”) will make interpretation more well defined. The new metric may help to further characterize the minimum “dose” required for optimal performance, percent change in hemoglobin mass and other measures of physiologic adaptation.
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COMMENTARY ON VIEWPOINT: TIME FOR A NEW METRIC FOR HYPOXIC DOSE?

TO THE EDITOR: The actual model which includes the degree of altitude as an equivalent parameter as the exposure time to hypoxia (1) is a systematic further development of the former model using just the exposure time (2), which was only valid for athletes training at a relatively narrow range of altitude.

The authors correctly mention possible limitations concerning the minimum hypoxic dose for altitude and hypoxic exposure time. It seems to be also interesting if the new model is applicable to athletes living permanently in hypoxia. Whenever it is almost not possible to compare identical athletes under normoxic and chronic hypoxic conditions cross-sectional studies on elite cyclists show bigger increases under chronic altitude conditions (2,600 m) than calculated by the model [11 vs. 7.7% (4)]. For these cases a modification of the model should be considered.

Following the idea of the authors that athletes who want to increase their Hb-mass by altitude training may choose between a relatively long stay at lower or a shorter stay at higher altitude for a fixed increase in Hb-mass they have to consider if the hemoglobin gained at altitude can be transferred to low altitude, where the competition takes place. As demonstrated by Ryan et al. (3) a strong increase in Hb-mass after 16 days at high altitude (5,260 m) is almost completely abolished after some days at lower altitude. As the return from moderate altitude is not associated with remarkable red cell destruction, for practical reasons an altitude threshold for red cell cytolysis has to be determined.

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