The following is the abstract of the article discussed in the subsequent letter:

Wagner, Peter D., Mauricio Araoz, Robert Boushel, José A. L. Calbet, Birgitte Jessen, Göran Rådegran, Hilde Spielvogel, Hans Søndegaard, Harrieth Wagner, and Bengt Saltin. Pulmonary gas exchange and acid-base state at 5,260 m in high-altitude Bolivians and acclimatized lowlanders. *J Appl Physiol* 92: 1393–1400, 2002. First published November 16, 2001; doi:10.1152/japplphysiol.00093.2001.—Pulmonary gas exchange and acid-base state were compared in nine Danish lowlanders (L) acclimatized to 5,260 m for 9 wk and seven native Bolivian residents (N) of La Paz (altitude 3,600–4,100 m) brought acutely to this altitude. We evaluated normalcy of arterial pH and assessed pulmonary gas exchange and acid-base balance at rest and during peak exercise when breathing room air and 55% O2. Despite 9 wk at 5,260 m and considerable renal bicarbonate excretion (arterial plasma HCO3 concentration = 15.1 meq/L), resting arterial pH in L was 7.48 ± 0.007 (significantly greater than 7.40). On the other hand, arterial pH in N was only 7.43 ± 0.004 (despite arterial O2 saturation of 77%) after ascent from 3,600–4,100 to 5,260 m in 2 h. Maximal power output was similar in the two groups breathing air, whereas on 55% O2 only L showed a significant increase. During exercise in air, arterial PCO2 was 8 Torr lower in L than in N (P < 0.001), yet PO2 was the same such that, at maximal O2 uptake, alveolar-arterial PO2 difference was lower in N (5.3 ± 1.3 Torr) than in L (10.5 ± 0.8 Torr). P = 0.004. Calculated O2 diffusing capacity was 40% higher in N than in L and, if referenced to maximal hypoxic work, capacity was 73% greater in N. Buffering of lactic acid was greater in N, with 20% less increase in base deficit per millimole per liter rise in lactate. These data show in L persistent alkalosis even after 9 wk at 5,260 m. In N, the data show 1) an insignificant reduction in exercise capacity when breathing air at 5,260 m compared with breathing 55% O2; 2) very little ventilatory response to acute hypoxemia (judged by arterial pH and arterial PCO2 responses to hypoxia); 3) during exercise, greater pulmonary diffusing capacity than in L, allowing maintenance of arterial PO2 despite lower ventilation; and 4) better buffering of lactic acid. These results support and extend similar observations concerning adaptation in lung function in these and other high-altitude native groups previously performed at much lower altitudes.

**What mechanism is responsible for the decreased BE/[La] ratio in exercise-induced metabolic acidosis?**

To the Editor: Wagner et al. (9) recently described an interesting phenomenon of the different slopes of the regression line for base excess (BE) on lactate concentration ([La]) in plasma during bicycle load for acclimatized lowlanders and high-altitude Bolivians. Exercise-induced metabolic acidosis in the acclimatized lowlanders was associated with a greater decrease in BE value than the increased [La] in plasma during and after exercise. However, a similar exercise in the high-altitude Bolivians was associated with a lower decrease in BE value than the increased [La]. Wagner et al. (9) concluded that lactate buffering appears more enhanced in high-altitude Bolivians, defending arterial pH in the face of both higher lactate and PCO2 levels, than in acclimatized lowlanders.

In a letter to the editor, this manuscript was commented on by Boning (1). Boning wrote: “Thus, despite my criticism, I feel that Wagner et al. (9) have observed an interesting phenomenon.” Wagner and Westen (10) in their reply wrote: “...we are not in a position to explain the entire nature of the complex differences in the acid-base behavior between the high-altitude natives and acclimatized lowlanders...we indeed agree that we have observed an interesting phenomenon, but this is one that requires further study.” On the one hand, the retrospective analysis of the relationship between BE and [La] during and after exercise-induced metabolic acidosis was not performed. On the other hand, there is a case history of the phenomenon.

Long time before, both Bouhuys et al. (2) and Osnes and Hermansen (5) investigated the relationship between BE and [La] in nonsportsmen healthy males and sportsmen under performance of high-intensity exercise. It was found in both cases that the BE value consistently overestimates the amounts of acid added to blood during high-intensity exercise (2, 5). When we carried out the analysis of the relationship between BE and [La] from Table 4 (2) in the sportsmen—members of a University rowing team under performance of high-intensity exercise—a significant increment was found: BE-to-[La] ratio (BE/[La]) increased from −1.23 ± 0.02 to −0.96 ± 0.01 (P = 0.03) when the same group was compared before and after a 2-mo training period. Kayser et al. (3) compared the effect of a bicarbonate loading on the exercise-induced metabolic acidosis in two physiological conditions: 1) at sea level and 2) at altitude. On the basis of results from Table 2 of Ref. 3, it was found that when the exercise is performed at sea level, average BE/[La] corresponds to −1.05, whereas after bicarbonate loading, BE/[La] corresponds to −0.66. When the similar exercise was performed at altitude, the BE/[La] decreased up to −1.79, whereas after the bicarbonate loading, the BE/[La] recovered up to −1.15.

Finally, during the period 1991–1993, we found that the value of BE/[La] strongly depends on physiological background, i.e. different degree of fatigue induced by the accumulated effect of the prolonged training program (6–8). The physiological background can be created by a prolonged training program within 3–6 wk, and it can be ranked by at least four different training programs: slight, middle, hard, and very hard. For example, changing the physiological background from a slight to very hard training program, the standard exercise-induced metabolic acidosis is associated with decreasing the value of BE/[La] from −1.0 to −4.0. We also found that there was a strong relationship between BE/[La] and PCO2. If BE/[La] decreased below −1.9, then PCO2 also decreased to a deep form of the respiratory alkalosis. It reflected the deceleration of the blood acid-base balance recovery after exercise (8). It was suggested that a transition from adaptation to deadaptation of the physiological state was accompanied by a decrease in HCO3 supply rate for blood and tissues due to a deceleration of anabolic reaction in the cell at resting period. The mechanism is based on metabolic regulation of the acid-base disorder in the cell (4). Therefore, we concluded that there are at least three forms of the metabolic acidosis induced by exercise: 1) an optimal form (BE/[La] corresponding to −1.5 and above), which reflects a good adaptation to exercise, 2) a nonoptimal form (BE/[La] corresponds to −1.9 and below), which reflects a deadaptation to exercise, and 3) a transition form (BE/[La] from −1.5 to −1.9), which reflects the intermediate state between adaptation and deadaptation to exercise (8).

**REFERENCES**


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