# Maximal lactic capacity at altitude: effect of bicarbonate loading

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KAYSER, BENGT, GUIDO FERRETTI, BRUNO GRASSI, TIZIANO BINZONI, AND PAOLO CERRETELLI. *Maximal lactic capacity at altitude: effect of bicarbonate loading.* J. Appl. Physiol. 75(3): 1070–1074, 1993.—The aim of the present study was to test the hypothesis that the net maximal blood lactate accumulation ($[La]_{max}$) during heavy exercise in lowlanders acclimatized to chronic hypoxia may be limited by the reduced bicarbonate stores. Six men [age 32 ± 4 (SD) yr] performed supramaximal exercise until voluntary exhaustion at sea level (204 ± 54 W) and after sojourning for 1 mo at 5,050 m (175 ± 23 W), without (C) and with (B) oral sodium-bicarbonate loading (0.3 g/kg body wt). Exhaustion time, arterial blood lactate concentration, arterial pH ($pH_a$), arterial $P_{CO_2}$, and intramuscular pH were measured at rest and after exercise. At sea level, exhaustion time increased from 6.5 ± 2.8 min in C to 7.5 ± 2.7 min in B ($P < 0.05$). At altitude, exhaustion times were similar to the sea level C values and the same in C and B. At sea level, resting $pH_a$ increased from 7.41 ± 0.02 in C to 7.46 ± 0.03 in B ($P < 0.001$); the corresponding values at altitude were 7.46 ± 0.04 and 7.55 ± 0.03 ($P < 0.001$). Postexercise $pH_a$ at sea level was 7.22 ± 0.02 in C and 7.25 ± 0.08 in B (NS). After exercise at altitude, $pH_a$ was 7.32 ± 0.04 and 7.44 ± 0.03 in C and B, respectively ($P < 0.001$). $[La]_{max}$ increased from 12.86 ± 1.45 mM in C to 16.63 ± 1.76 mM in B ($P < 0.01$) at sea level and from 6.85 ± 1.40 mM in C to 7.95 ± 1.74 mM in B (NS) at altitude. Postexercise intramuscular pH was significantly higher at altitude than at sea level ($P < 0.05$), independent of bicarbonate loading. It is concluded that the decreased buffer capacity accompanying high-altitude acclimatization is not responsible for the observed decrease in $[La]_{max}$ in acclimatized lowlanders.

acclimatization; alkalosis; anaerobic metabolism; buffering; climbing; exercise; lactate

MAXIMAL NET (postexercise − resting) blood lactate concentration ($[La]_{max}$) after strenuous exercise in acute hypoxia attains the same value as in normoxia (8, 12). By contrast, in chronic hypoxia, $[La]_{max}$ is known to be greatly reduced (2, 7, 8, 12, 16, 26, 28–30), its level decreasing with altitude (8, 12, 26). This change is reversible on return to sea level, with a half time of ~5–10 days (15). High-altitude natives are also characterized by low levels of $[La]_{max}$ at exhaustion (8), although the underlying mechanism might be different from that for acclimatized lowlanders. Indeed, Matheson et al. (22), measuring muscle pH and blood lactate after heavy work in a group of altitude natives after a 6-wk deacclimatization found a persistent blunting of anaerobic glycolysis that was attributed to a possibly genetic factor.

A reduction of the buffer capacity in both the blood and the tissues is known to occur in chronic hypoxia as a consequence of the progressive metabolic compensation of repiratory alkalosis. Thus a given accumulation of lactic acid may generate a sizeable drop of intracellular pH, thereby impairing the activity of some key enzymes of anaerobic glycolysis (8, 14, 26). The reduced $[La]_{max}$ could therefore be an adaptive reaction. Should this be the case, an increase in buffer capacity by administration of bicarbonate might be compatible with greater $[La]_{max}$ levels. Indeed, bicarbonate administration was shown to increase $[La]_{max}$ in acclimated rats (14).

In the present study, the hypothesis was tested that the reported decrease of $[La]_{max}$ in acclimatized lowlanders may be mainly the consequence of a reduced buffer capacity.

## METHODS

*Subjects.* Six healthy male subjects [age 32 ± 4 (SD) yr] participated in the study. They were informed about the scope and risks of the experiments and gave their consent. Body weight was 75.9 ± 12.4 and 70.4 ± 12.9 kg at sea level and after 4 wk at an altitude of 5,050 m, respectively.

*Measurements and calculations.* Blood samples from an earlobe were drawn into heparinized 0.1-ml capillaries. Arterialization was achieved by prior application of a hyperemia-inducing ointment (Trafuril, Ciba-Geigy). Arterial pH ($pH_a$), blood $CO_2$ partial pressure ($Pa_{CO_2}$), hemoglobin concentration ([Hb]), and lactate concentration ([La]) were determined on the samples by means of a blood gas and hemoglobin analyzer (Ciba Corning 280) and a lactate analyzer (Kontron 640). Both analyzers were previously calibrated with samples of known composition. Arterial $O_2$ saturation ($Sa_{O_2}$, %) at altitude was continuously measured at rest and during the exercise tests by photometry (Biox, Ohmeda, CO). $Sa_{O_2}$ at sea level was assumed to be 97%. Blood bicarbonate concentration was calculated by the Henderson-Hasselbalch equation from $Pa_{CO_2}$ and $pH_a$. $Pa_{CO_2}$ was plotted as a function of $pH_a$ according to Davenport (10). In vivo base excess ($BE_a$) was computed automatically by the blood gas analyzer, which took into account [Hb] and $Sa_{O_2}$.

Intramuscular pH ($pH_m$) was measured by a miniature glass electrode method, with the results probably reflecting, like those obtained from needle biopsy specimens, a compromise between intracellular and extracellular pH (27). An incision of the skin and fascia over the midportion of the vastus lateralis muscle was made with a scal-

TABLE 1. *Maximal power, hemoglobin concentration, and change in exhaustion time after sodium bicarbonate loading at sea level and at altitude*

| Subj No. | $\dot{W}_{max}$, W | | [Hb], g/l | | $\Delta t_{ex}$, min | |
|---|---|---|---|---|---|---|
| | Sea level | Altitude | Sea level | Altitude | Sea level | Altitude |
| *1* | 300 | 210 | 12.4 | 19.3 | 2.67 | 2.98 |
| *2* | 215 | 180 | 12.7 | 15.6 | 2.00 | −0.62 |
| *3* | 240 | 180 | 15.5 | 19.7 | 0.05 | 2.75 |
| *4* | 180 | 150 | 14.8 | 20.4 | 0.79 | 6.58 |
| *5* | 180 | 150 | 13.3 | 19.7 | 0.00 | −1.86 |
| *6* | 210 | 180 | 12.2 | 17.8 | 0.33 | 2.52 |
| Mean ± SD | 204±54 | 175±23* | 13.5±1.4 | 18.8±1.8* | 0.97±1.11 | 2.06±2.98 |

$\dot{W}_{max}$, maximal power; [Hb], hemoglobin concn; $\Delta t_{ex}$, change in exhaustion time. * Significantly different from sea level ($P < 0.001$).

pel. After calibration with buffers of known pH, an Ag/AgCl reference electrode (Mere 1, World Precision Instruments) connected with an KCl agar salt bridge to a 21-gauge butterfly needle, together with a conventional glass pH electrode mounted in a 21-gauge hypodermic needle (SA 1B, World Precision Instruments), were introduced ~1.5 cm deep into the muscle ~5 mm apart. $pH_m$ was read from a pH meter (Metrohm 654). The electrodes were then withdrawn during the exercise and introduced again at the onset of the recovery.

*Experimental protocol.* The maximal mechanical power output ($\dot{W}_{max}$), defined as the highest work rate that a subject could sustain for 4 min, was determined on a mechanically braked cycle ergometer (Monark) during a continuous incremental exercise on *day 1*. Starting from 30 W, the power was increased by 30-W steps until $\dot{W}_{max}$ was obtained. On *day 2*, the subjects exercised at $\dot{W}_{max}$ until voluntary exhaustion, defined as the subjects' inability to keep pedaling at the selected frequency of 60/min, the control condition (C). Time to exhaustion ($t_{ex}$) was measured by a stopwatch. On *day 3* metabolic alkalosis was induced by ingestion of sodium bicarbonate ($NaHCO_3$) at the selected dose of 0.3 g/kg body wt, diluted in 400 ml water. No adverse effects from the ingestion of $NaHCO_3$ were experienced, with the exception, in two cases, of some minor gastrointestinal complaints. One to 1.5 h after $NaHCO_3$ ingestion, the subjects repeated the exercise at $\dot{W}_{max}$, the bicarbonate condition (B). On *days 2* and *3*, resting arterialized blood samples for blood gases and [La] were taken from the earlobe of the subject while he was sitting on the cycle ergometer before the exercise test. Additional samples were drawn at 2-min intervals after the end of exercise. The resting values and the peak [La] with the corresponding $pH_a$ were used for further calculations. $pH_m$ was measured at rest before exercise and every 2 min from *minute 2* through *minute 24* into the recovery.

The above experimental protocol was performed twice, at sea level (Milano, Italy, 122 m) and at the end of a 1-mo sojourn at 5,050 m. The altitude experiments were conducted in the "Pyramid," the Italian Research Laboratory at Lobuche, Nepal (5,050 m). The laboratory was equipped with a stabilized source of electricity by a generator powered by a water turbine. Temperature inside the laboratory during the experiments ranged between 15 and 22°C.

*Statistics.* Values are given as means ± SD. Sea level and altitude measurements were compared by the *t* test for paired observations. $pH_m$ values after exercise in the different conditions were compared using a three-way analysis of variance [dependent variable: $pH_m$; independent variables: time from exhaustion until measurement (2, 4, 6, 8, . . .24 min), location (altitude or sea level), and condition (C or B)]. A post hoc test (Bonferroni) was used to discriminate between significantly different pairs. Normal distribution of errors was assumed. The differences were considered significant at $P < 0.05$.

## RESULTS

$\dot{W}_{max}$, [Hb], and the differences in exhaustion times ($\Delta t_{ex}$) between conditions B and C at sea level and at altitude are shown in Table 1. Average $t_{ex}$ at $\dot{W}_{max}$ in C at sea level was 6.5 ± 2.8 min and increased to 7.5 ± 2.7 min ($P < 0.05$) in B. At altitude, $t_{ex}$ values were 5.1 ± 1.9 and 7.1 ± 2.7 min (NS) in C and B, respectively. The altitude figures were not significantly different from the corresponding sea level values.

The mean experimental blood acid-base balance values are shown in Table 2. $[La]_{max}$ at sea level increased from 12.86 ± 1.45 mM in C to 16.63 ± 1.76 mM in B ($P < 0.01$). At high altitude the values were 6.85 ± 1.40 in C and 7.95 ± 1.74 in B (NS). It appears (Fig. 1) that, at sea level for an equal change in $[H^+]$, $[La]_{max}$ at exhaustion was higher in B than in C. By contrast, at altitude, $[La]_{max}$ at exhaustion was about the same in B and C and was accompanied by a significant difference in $[H^+]$ between B and C. $BE_a$, as expected, was significantly increased in B compared with C, both at sea level and at altitude. It is noteworthy that resting $BE_a$ in B at altitude was close to zero.

Altitude $Pa_{CO_2}$ values at rest are indicative of respiratory alkalosis. The lower $Pa_{CO_2}$ values after exercise, compared with rest, reflect a partial respiratory compensation of the metabolic acidosis. The decrease of $Pa_{CO_2}$ was more pronounced at sea level than at high altitude (see Table 2 and Fig. 2).

Resting $pH_m$ values were similar in all conditions and independent of altitude exposure or bicarbonate ingestion. Analysis of variance showed no significant effect for postexercise $pH_m$ in B compared with C either at sea level or at altitude. The effect of altitude, however, was significant ($P < 0.05$), and the means were significantly different at *minutes 6, 8, 10, 12, 14,* and *16* into the recovery (Fig. 3).

TABLE 2. *Measured and calculated variables in arterialized blood with and without bicarbonate administration*

| | Sea Level | | | | Altitude | | | |
|---|---|---|---|---|---|---|---|---|
| | Rest | | Exercise | | Rest | | Exercise | |
| | C | B | C | B | C | B | C | B |
| $pH_a$ | 7.41±0.02 | 7.46±0.02‡ | 7.22±0.02 | 7.25±0.08 | 7.46±0.04 | 7.55±0.03† | 7.32±0.04 | 7.44±0.03‡ |
| $[La]_a$, mM | 1.11±0.11 | 1.62±0.16* | 13.97±1.45 | 17.40±2.60† | 1.40±0.49 | 1.41±0.38 | 8.24±1.13 | 9.36±1.64 |
| $Pa_{CO_2}$, Torr | 41.3±6.5 | 43.0±4.4 | 27.3±2.5 | 31.2±6.0 | 22.0±3.2 | 24.4±3.0 | 19.2±3.0 | 19.0±2.1 |
| $[HCO_3^-]_a$, mM | 26.0±3.5 | 30.8±2.6* | 11.1±1.8 | 14.1±5.2 | 15.7±1.8 | 21.4±3.6† | 9.9±1.4 | 12.8±0.8† |
| $[H^+]_a$, nM | 38.96±1.96 | 34.43±1.29‡ | 60.60±7.09 | 56.78±10.08 | 34.51±2.71 | 28.35±1.89† | 47.89±5.15 | 36.81±2.77† |
| $BE_a$, mM | 1.9±3.3 | 7.1±2.4† | −14.7±2.1 | −11.5±5.3* | −7.6±1.8 | −1.1±3.6‡ | −14.8±1.4 | −10.8±1.0† |

Values are means ± SD of 6 subjs. $pH_a$, arterial pH; $[La]_a$, arterial lactate concn; $Pa_{CO_2}$, arterial $Pco_2$; $[HCO_3^-]_a$, arterial bicarbonate concn; $[H^+]_a$, arterial proton concn; $BE_a$, arterial base excess; B and C, with and without bicarbonate administration, respectively. Significantly different from C: * $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$.

## DISCUSSION

At high altitude $[La]_{max}$ levels in B were not higher than in C. In fact, exercise was interrupted at $pH_a$ values well above those attained in C, despite the availability of greater bicarbonate buffers. If, as hypothesized, metabolic acidosis were the limiting factor to $[La]_{max}$ also at altitude, then bicarbonate loading would have buffered $H^+$, thus leading to increased $[La]_{max}$ levels well above that observed in C, and exhaustion should have been attained at $pH_a$ and $pH_m$ values close to those in C at sea level. Indeed, this was not the case.

There still is considerable debate about the efficacy of bicarbonate administration in improving performance (i.e., $t_{ex}$) at sea level. Some studies have reported an increase, whereas others have indicated no change at all (4, 9, 18, 25). Apart from being irrelevant to the aim of the present study, the observed small, but significant, increase in $t_{ex}$ must therefore be interpreted with caution. In any case, the greater increase in [La], after bicarbonate administration, coupled to unchanged postexercise $pH_a$, seems to indicate that, at sea level, the bicarbonate-induced increase in buffer capacity did contribute to buffer extra lactic acid in blood.

Figure 2 shows that, at altitude after ingestion of bicarbonate, bicarbonate buffers were normalized and base excess was almost zero such that the subjects were back to the normal buffer line, although in a condition of respiratory alkalosis. According to Davies et al. (11), respiratory alkalosis may lead to an increase in $[La]_{max}$ at sea level. However, this was not the case for the present study at altitude. Moreover, after exhaustive exercise $pH_m$ was higher at altitude than at sea level, confirming previous results (16, 29). Therefore the present data allow us to reject the hypothesis that the lower $[La]_{max}$ in chronic hypoxia may be a consequence of reduced buffer capacity.

Lactate transport over the sarcolemmal membrane seems to occur by means of facilitated exchange along $H^+$ and lactate concentration gradients involving a ubiquitous lactate transport protein (5, 13, 24). Proton transmembrane transport is an active process and involves an exchange of $Na^+$-$H^+$ and $HCO_3^-$-$Cl^-$ (1, 21). Extracellular $[HCO_3^-]$ is therefore important for proton transport and in theory could become a limiting factor to anaerobic me-

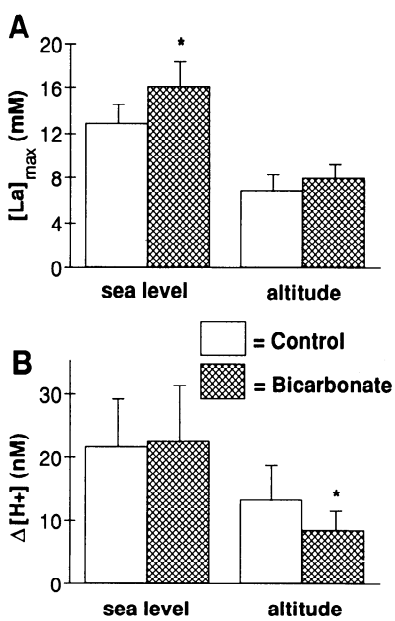


FIG. 1. Average net increase in maximal lactate concn ($[La]_{max}$; *A*) and net increase in proton concn ($\Delta[H^+]$; *B*) measured at end of exhaustive maximal exercise at sea level and at altitude. Values are means ± SD. * Significantly different from control after bicarbonate loading ($P < 0.05$).

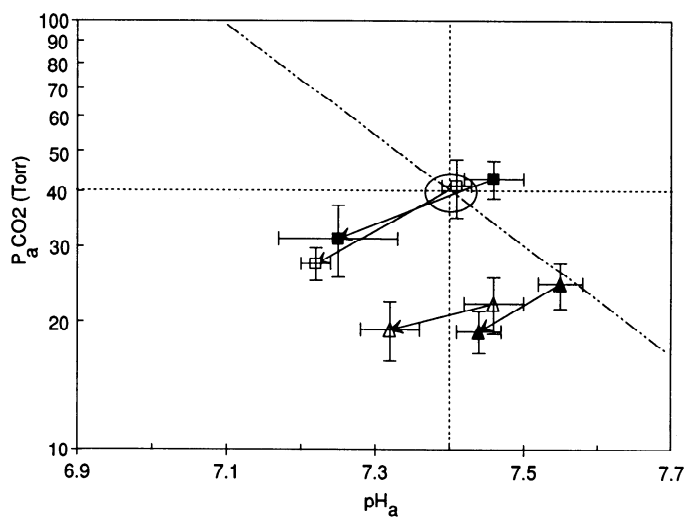


FIG. 2. Average arterial $Pco_2$ ($Pa_{CO_2}$) and arterial pH ($pH_a$) data plotted on a Davenport diagram (10). Dot-dash line, normal buffer line; circle, region of normal acid-base status for resting subjects at sea level; dotted lines, isopleths for $Pa_{CO_2}$ = 40 Torr and $pH_a$ = 7.4. □, Sea level without bicarbonate; ■, sea level with bicarbonate; △, altitude without bicarbonate; ▲, altitude with bicarbonate. Arrows, changes in acid-base status occurring with movement from rest to exercise.

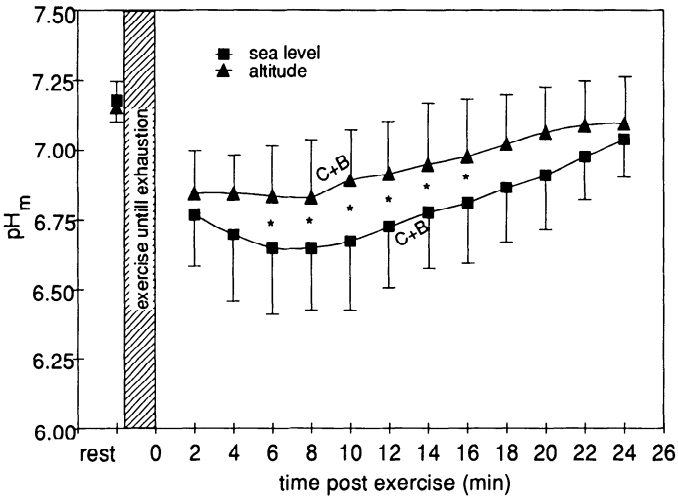


FIG. 3. Intramuscular pH ($pH_m$) at rest and after exercise as a function of time. Hatched area, exhaustive exercise; C+B, average of control (without bicarbonate) and bicarbonate values. * Significantly different pairs ($P < 0.05$).

tabolism at high altitude. In that case one would have expected to observe postexercise $pH_m$ values close to or lower than those observed at sea level both in C and in B. Again, this was not the case.

The observed greater $[La]_{max}$ after bicarbonate administration at sea level may have resulted not only from increased lactate transport from the muscle into the blood but also from increased lactate production in the muscle or from decreased lactate uptake by other tissues (11). In this study neither intramuscular lactate nor lactate clearance rate was measured, so a conclusive answer cannot be given. It may also be argued that, in conditions of chronic hypoxia, lactate and/or $H^+$ transport from the intracellular space to the blood could be reduced compared with normoxia. The latter argument, however, appears in contrast to the following observations, all compatible with normal lactate and proton transport from the cell into the blood in chronic hypoxia: *1*) the distribution of lactate among the intra- and extracellular body compartments is similar to that during normoxia (7, 8), *2*) net kinetics of arterial [La] recovery after supramaximal exhaustive exercise is unchanged (20), *3*) muscle pH at exhaustion is more alkaline (29; this study), and *4*) at exhaustion both $[La]_{max}$ and muscle lactate are lower than in normoxia (16, 29).

Because reduced buffer capacity of the body in the acclimatized state is not responsible for the decreased $[La]_{max}$ at altitude, other mechanisms must be called on to explain this phenomenon. Diminished muscle glycogen stores and impaired activity of several glycolytic enzymes have been excluded as possible determinants (16, 19, 28). According to Bigland-Ritchie and Vollestadl (3), complete neuromuscular activation may not be achieved in chronic hypobaric hypoxia. It has been hypothesized that in these conditions the central nervous system could become a limiting factor to muscle contraction (3, 16, 20, 23). Alternatively, it was shown that the rate of appearance of lactate both at sea level and high altitude is closely related to adrenergic activity (6, 23). In fact, Reeves et al. (23) and Young et al. (30) argue that the higher [La] values found at a given absolute submaximal work load in acute hypoxia compared with normoxia are apparently mediated by catecholamines. By contrast, the reduction of $[La]_{max}$ after acclimatization appears to be catecholamine independent. The explanation proposed by Matheson et al. (22) for the low $[La]_{max}$ observed in high-altitude natives, although appealing, is not applicable to acclimatized lowlanders, because the latter show rapid normalization of $[La]_{max}$ during deacclimatization (15). Further research is therefore necessary to elucidate the mechanisms controlling the rate and the capacity of anaerobic glycolysis at high altitude.

In conclusion, no increase in $[La]_{max}$ after bicarbonate loading was found in altitude-acclimatized subjects. Although at altitude, after bicarbonate loading, the preexercise alkali reserve was restored to the sea level value, $[La]_{max}$ did not attain preacclimatization values, and blood pH was higher than in any other tested condition. In addition, muscle pH after exhaustive exercise was higher at altitude than at sea level. As a consequence, decreased intra- and extracellular buffer capacities do not appear to be responsible for the decrease in $[La]_{max}$ usually observed in lowlanders acclimatized to high altitude.

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## REFERENCES

1. AICKIN, C. C., AND R. C. THOMAS. An investigation of the ionic mechanism of intracellular pH regulation in mouse soleus muscle fibers. *J. Physiol. Lond.* 273: 295–316, 1977.
2. BENDER, P. R., B. M. GROVES, R. E. MCCULLOUGH, R. G. MCCULLOUGH, L. TRAD, A. J. YOUNG, A. CYMERMAN, AND J. T. REEVES. Decreased exercise muscle lactate release after high altitude acclimatization. *J. Appl. Physiol.* 67: 1456–1462, 1989.
3. BIGLAND-RITCHIE, B., AND N. K. VOLLESTAD. Hypoxia and fatigue: how are they related? In: *Hypoxia. The Tolerable Limits*, edited by J. R. Sutton, C. S. Houston, and G. Coates. Indianapolis, IN: Benchmark Press, 1988, p. 315–328.
4. BRIEN, D. M., AND D. C. MCKENZIE. The effect of induced alkalosis and acidosis on plasma lactate and work output in elite oarsmen. *Eur. J. Appl. Physiol. Occup. Physiol.* 58: 797–802, 1989.
5. BROOKS, G. A. Current concepts in lactate exchange. *Med. Sci. Sports Exercise* 23: 895–906, 1991.
6. BROOKS, G. A., G. E. BUTTERFIELD, R. R. WOLFE, B. M. GROVES, R. S. MAZZEO, J. R. SUTTON, E. E. WOLFEL, AND J. T. REEVES. Decreased reliance on lactate during exercise after acclimatization to 4,300 m. *J. Appl. Physiol.* 71: 333–341, 1991.
7. CERRETELLI, P., AND T. BINZONI. The energetic significance of lactate accumulation in blood at altitude. *Int. J. Sports Med.* 11, *Suppl.* 1: S27–S30, 1990.
8. CERRETELLI, P., A. VEICSTEINAS, AND C. MARCONI. Anaerobic metabolism at high altitude: the lactacid mechanism. In: *High Altitude Physiology and Medicine*, edited by W. Brendel and R. A. Zink. Berlin: Springer, 1985, p. 94–102.
9. COSTILL, D. L., F. VERSTAPPEN, H. KUIPERS, E. JANSSEN, AND W. FINK. Acid-base balance during repeated bouts of exercise: influence of $HCO_3^-$. *Int. J. Sports Med.* 5: 228–231, 1984.
10. DAVENPORT, H. W. *The ABC of Acid-Base Chemistry*. Chicago, IL: Univ. of Chicago Press, 1974.
11. DAVIES, S. F., C. IBER, S. A. KEENE, C. D. MCARTHUR, AND M. J. PATH. Effect of respiratory alkalosis during exercise on blood lactate. *J. Appl. Physiol.* 61: 948–952, 1986.
12. EDWARDS, H. T. Lactic acid in rest and work at high altitude. *Am. J. Physiol.* 116: 367–375, 1936.

13. FISHBEIN, W. N. Lactate transporter defect: a new disease of muscle. *Science Wash. DC* 234: 1254–1256, 1986.
14. GONZALEZ, N. C., M. ZAMAGNI, AND R. L. CLANCY. Effect of alkalosis on maximum oxygen uptake in rats acclimated to simulated altitude. *J. Appl. Physiol.* 71: 1050–1056, 1991.
15. GRASSI, B., B. KAYSER, T. BINZONI, M. MARZORATI, M. BORDINI, C. MARCONI, AND P. CERRETELLI. Peak blood lactate concentration during altitude acclimatization and deacclimatization in humans (Abstract). *Pfluegers Arch.* 420: R165, 1992.
16. GREEN, H. J., J. SUTTON, P. YOUNG, A. CYMERMAN, AND C. S. HOUSTON. Operation Everest II: muscle energetics during maximal exhaustive exercise. *J. Appl. Physiol.* 66: 142–150, 1989.
17. HOCHACHKA, P. W. The lactate paradox: analysis of underlying mechanisms. *Ann. Sports Med.* 4: 184–188, 1989.
18. HORSWILL, C. A., D. L. COSTILL, W. FINK, M. G. FLYNN, J. P. KIRWAN, J. B. MITCHELL, AND J. A. HOUMARD. Influence of sodium bicarbonate on sprint performance: relationship to dosage. *Med. Sci. Sports Exercise* 20: 566–569, 1988.
19. HOWALD, H., D. PETTE, J. A. SIMONEAU, H. HOPPELER, AND P. CERRETELLI. Effects of chronic hypoxia on muscle enzyme activities. *Int. J. Sports Med.* 11, *Suppl.* 1: S10–S14, 1990.
20. KAYSER, B., B. GRASSI, G. FERRETTI, A. COLOMBINI, C. MARCONI, AND P. CERRETELLI. Maximal rate of blood lactate accumulation during high altitude exposure in humans (Abstract). *Physiologist* 35: 211, 1992.
21. MAINWOOD, G. W., AND J. M. RENAUD. The effect of acid-base balance on fatigue of skeletal muscle. *Can. J. Physiol. Pharmacol.* 63: 403–416, 1985.
22. MATHESON, G. O., P. S. ALLEN, D. C. ELLINGER, C. C. HANSTOCK, D. GHEORGHIU, D. C. MCKENZIE, C. STANLEY, W. S. PARKHOUSE, AND P. W. HOCHACHKA. Skeletal muscle metabolism and work capacity: a $^{31}$P-NMR study of Andean natives and lowlanders. *J. Appl. Physiol.* 70: 1963–1976, 1991.
23. REEVES, J. T., L. G. MOORE, E. E. WOLFEL, R. S. MAZZEO, A. CYMERMAN, AND A. J. YOUNG. Activation of the sympathicoadrenal system at high altitude. In: *High Altitude Medicine*, edited by G. Ueda, J. T. Reeves, and M. Sekiguchi. Matsumoto, Japan: Shinshu University Press, 1992, p. 10–26.
24. ROTH, D. A. The sarcolemmal lactate transporter: transmembrane determinants of lactate flux. *Med. Sci. Sports Exercise* 23: 925–934, 1991.
25. SUTTON, J. R., N. L. JONES, AND C. J. TOEWS. Effect of pH on muscle glycolysis during exercise. *Clin. Sci. Lond.* 61: 331–338, 1981.
26. WEST, J. B. Lactate during exercise at extreme altitude. *Federation Proc.* 45: 2953–2957, 1986.
27. WILLIAMS, C., AND G. HALL. Continuous intramuscular pH measurement during the recovery from brief, maximal exercise in man. *Eur. J. Appl. Physiol. Occup. Physiol.* 59: 465–470, 1990.
28. YOUNG, A. J., W. J. EVANS, A. CYMERMAN, K. B. PANDOLF, J. J. KNAPIK, AND J. T. MAHER. Sparing effect of chronic high-altitude exposure on muscle glycogen utilization. *J. Appl. Physiol.* 52: 857–862, 1982.
29. YOUNG, A. J., W. J. EVANS, E. C. FISHER, R. L. SHARP, D. L. COSTILL, AND J. T. MAHER. Skeletal muscle metabolism of sea-level natives following short-term high-altitude residence. *Eur. J. Appl. Physiol. Occup. Physiol.* 52: 463–466, 1984.
30. YOUNG, A. T., P. M. YOUNG, R. E. MCCULLOUGH, L. G. MOORE, A. CYMERMAN, AND J. T. REEVES. Effect of beta-adrenergic blockade on plasma lactate concentration during exercise at high altitude. *Eur. J. Appl. Physiol. Occup. Physiol.* 63: 315–322, 1991.