The following letters are in response to the Point:Counterpoint “The lactate paradox does/does not occur during exercise at high altitude” that appears in this issue.

To the Editor: I found the argument put forth by Van Hall (5) to contain a number of inaccurate and misleading statements. First, his statement that a “controversy on sympathetic activity” exists is based solely on his incorrect review of one paper (2) in which sympathetic activity was not even reported. In fact, in all of the Pikes Peak investigations in which various markers of sympathetic activity were measured (arterial norepinephrine levels, muscle norepinephrine release and, 24-h urinary norepinephrine excretion), it was consistently found that sympathetic activity was reliably elevated over time at high altitude (3). Furthermore, Van Hall states that they previously observed “much higher blood levels of epinephrine” with acclimatization, yet epinephrine levels are not reported in the first study (1) cited and the second study (4) showed no differences in epinephrine levels between chronic hypoxia and acute normoxia, casting doubt on the validity of his argument. Second, no acute measurements on lactate were performed at Chacaltaya, which limits the data interpretation of this frequently cited study by Van Hall (4). Lastly, in the Chacaltaya study, subjects lost on average 7.3 kg of lean body mass (~16 lb) over the 9-wk period at altitude. Such a large loss of muscle mass would clearly confound data interpretation and certainly lactate metabolism for a given submaximal as well as maximal mass would clearly confound data interpretation and certainly lactate production during exercise at high altitude. J Appl Physiol 76: 610–615, 1994; 77: 2408–2412, 1994.

To the Editor: The initial question of this Point:Counterpoint is not about mechanisms but whether a phenomenon occurs at all (4, 6). The numerous studies reporting changes in exercise blood lactate in acute and chronic hypoxia appear rather convincing (2, 5, 6). The ensuing question then is why some studies have not found such changes (4).

Oxygen supply to muscle is not regarded anymore as the direct determinant of lactate production during exercise (1). There are good reasons to postulate that changes of blood lactate during exercise at altitude are related to mass effect and sympathetic drive; hypoxia decreases maximum power output and increases sympathetic drive, glycogenolysis is under adrenergic influence, and for a given rate of Krebs cycling, excess pyruvate will lead to more lactate formation and vice versa (2, 5).

In addition, because of orderly recruitment, incremental exercise begins with predominantly slow-twitch fibers, whereas fast-twitch fibers contract at higher intensities. This partly explains greater lactate efflux from muscle at higher intensities (3). At altitude, peak power is lower and thus the proportion of slow- vs. fast-twitch fibers activated at peak is higher than at sea level. Since humans can differ to quite an extent in fiber type composition, the differences between studies...
ies could perhaps in part be related to differences in fiber type composition of the subjects’ muscles.

Finally, blood lactate results from appearance and disappearance rates and does not directly reflect production.

REFERENCES

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To the Editor: I read with interest the West/Van Hall debate on the existence of the lactate paradox (2, 3) and wish to raise two issues. First, why did Van Hall neglect the 3,800 m study of Pronk et al. (1)? That study was similar in many ways to the Copenhagen group’s project at 4,100 m at El Alto, Bolivia. Arterial and not capillary blood was taken, data were collected several times over the course of 8 wk at a fixed altitude, evidence of constant fitness was obtained, and there were no weight changes. Pronk was fastidious about reproducing exercise duration and intensity and also about precise timing of blood sampling during exercise throughout the 8 wk of acclimatization. Pronk found that the curve relating arterial blood lactate to power output (including lactate at the end of intense exercise) was higher early after reaching altitude and then fell over time, demonstrating (again) what has become known as the paradox, now under precisely controlled conditions. The outcome was the same whether whole blood or plasma lactate was examined. Second, Van Hall’s discussion about lactate release, muscle lactate, tracer studies, and so on is interesting, but is completely peripheral to the question at hand. Studies lacking muscle biopsies or muscle effluent blood lactate levels or studies criticized concerning unrelated methods are not inferior to Van Hall’s for the question on the table and cannot be dismissed in the context of this question.

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To the Editor: In our opinion, the arguments in favor of the existence of the lactate paradox (LP; Ref. 6) overwhelm those against it (5). It is, however, true that despite several attempts (2), a clear explanation for LP still lacks. Hence the question is whether maximal exercise at altitude represents a good probe to get insight into LP. The obvious second question: which is the best probe? Historically, animal studies often provided clues to understand physiological processes in humans, but we are not aware of such studies that may be useful to better understand LP. Alternatively, in CHF patients, and very likely in COPD patients, the increased resistance to gas transfer across the alveolar-capillary interface usually yields to a number of consequences, among which is peripheral hypoxia (3). Although exercise training is an elective therapy for CHF patients (4), only one study, to our knowledge, hitherto addressed the issue of lactate release during exercise (1). These authors found that, for each fraction of maximal exercise intensity, lactate release decreases as a function of the severity of the disease, thereby supporting the occurrence of some form of LP in CHF patients. In conclusion, LP exists, but the study of additional pathophysiological models would help not only to fill actual knowledge gaps, but also to address the question whether LP has any relevant clinical implications.

REFERENCES

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To the Editor: In our experiments we saw the “lactate paradox” (LP). Measurements were carried out on 10 Caucasians staying for 5 wk at 5,050 m in a fully equipped laboratory with excellent living conditions. At the end of incremental exercise leading to exhaustion in ~20 min (2), peak blood lactate ([La]bpeak) was lower at altitude [8.0 ± 3.0 (means ± SD) mM at 1 wk, 6.4 ± 1.4 at 3 wk, 6.3 ± 0.9 at 5 wk] vs. sea level (11.5 ± 2.2). Interestingly, [La]bpeak was still lower during the first 2 wk after return from the expedition (2). Moreover, induction of acute hypobaric normoxia (FiO2 ~0.40) at 5,050 m increased peak workload, but not [La]+peak (2). Thus LP was not directly related to the lower peak workload. In other experiments at 5,050 m we observed a clear LP during exercise to exhaustion at 100% VO2 peak (4), as well as during 45-s supramaximal exercise (200% VO2 peak; Ref. 1); in the latter case, the LP was less pronounced after 4 wk of acclimatization. We did not see a LP, again at 5,050 m, after “all out” exercise...
lasting 10 s, during which maximal mechanical power output was not reduced vs. sea level (3). So, does the LP depend, at least in part, on the exercise protocol? Yes, according to our data. Can the LP be reversed in some cases and at least in part during prolonged acclimatization? Yes, according to our data. Does the LP occur? Yes, according to our data. Possible causes and the meaning of LP remain topics of further research and discussion.

REFERENCES


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To the Editor: In the debate over the existence of the lactate paradox during exercise at high altitude (4, 6), it is important to recall that lactate is not distributed equally between red blood cells (RBCs) and plasma (e.g., Refs. 1, 3). Because RBCs have a lactate concentration ([La−]) that is about one-half that of plasma at equilibrium (both corrected for water content), whole blood [La−] typically averages ~70% of plasma [La−] (neither corrected for water content). With increasing hematocrit, such as occurs with prolonged stay at altitude, this percentage will likely decrease somewhat. Obviously this creates a problem in quantifying the magnitude of a lactate response to exercise under different conditions. However, it is also a problem when attempting to measure lactate release or uptake across exercising muscle groups (e.g., Ref. 5). In such cases, the actual arteriovenous [La−] difference (and therefore lactate release or uptake) is likely to be only 70% of that reported on the basis of plasma [La−] measures, perhaps less if the hematocrit is elevated. These issues are compounded by the facts that 1) different automatic analyzers have different accuracies and reliabilities (see Ref. 2) and 2) when a whole blood sample is introduced to automatic analyzers, some of these instruments measure the plasma phase and report that value, while others measure the plasma phase and make a correction for hematocrit (entered by the user), and still others measure the plasma phase and use an internal algorithm to correct to a presumed whole blood value.

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