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 workload on the cycle ergometer as fiber type recruitment would become a contributing factor. Thus, although interindividual differences exist, the lactate paradox is alive and well.

REFERENCES

Robert S. Mazzeo
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To the Editor: After acclimatization to high altitude, maximal exercise is associated often with a low blood lactate level (4, 5), but it remains to be evaluated whether this lactate paradox reflects a strategy to maximize work capacity. At the low arterial oxygen tension at high altitude, it is a disadvantage to produce lactate, because the Bohr effect on the oxyhemoglobin dissociation curve means that any decrease in pH markedly affects the arterial oxygen saturation, oxygen delivery to the working muscles, oxygen uptake, and work capacity (1–3). At high altitude, lactate production limits endurance capacity by right shift of the oxyhemoglobin dissociation curve, but that does not indicate an inability to produce lactate. Lactate production would not limit small muscle mass work capacity because it does not depend on pulmonary oxygen uptake. The debate on the lactate paradox may reflect that different questions are asked. If a subject is asked to perform maximal whole body exercise and is interested in performing as much work as possible, a wise strategy would be to produce as little lactate as possible and that becomes increasingly important at higher altitudes. On the other hand, if the subject is asked to work as intensively as possible without considering endurance, it appears possible to produce as much lactate as at sea level. To view oxygen transport from the perspective of the Bohr effect on the oxyhemoglobin dissociation curve may be what unites the various observations on the blood lactate level during maximal whole body exercise after acclimatization to high altitude.

REFERENCES

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 stud-
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.

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]peak) 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]peak 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% V O2peak (4), as well as during 45-s supramaximal exercise (200% V O2peak; 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


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, 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.

REFERENCES


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