To the Editor: In response to the comments (4, 5) on our Point-Counterpoint debate (3), we point out that if there is action potential failure, there is no subsequent contraction whatsoever, and so it is untenable to argue, as Lindinger does (4), that some relatively minor inhibition of muscle performance is too big a price to pay to keep the whole E-C coupling process working. Yes, we fully agree with Sahlin (5) and Tupling (4) that an increase in intracellular acidity does have some downsides, slowing muscle relaxation to some degree. But we think that when a lion is chasing you, it is far better to have a small reduction in muscle performance than to have action-potential failure, such that your muscles fail to activate and you collapse in front of the lion! Clearly, lactic acid accumulation is a distinct “advantage” if it keeps your muscles working.

Sahlin’s comment (5) that activation failure is rarely seen in humans during voluntary exercise misses the point that is “rarely seen” for the very reason that the intracellular acidity is helping keep the action potential propagating in adverse circumstances and hence helping prevent “activation failure” from occurring. These adverse circumstances would be anything that hinders action potential propagation directly or indirectly, such as membrane depolarization by any cause (be it increased local extracellular [K+] or decreased Na-pump function, opening or closure of various ion channels), or increased intracellular [Na+]. No one seriously questions that fibers deep in human muscles in vivo, like animal muscle fibers in vitro, have a membrane potential, are activated by action potentials, and have an appreciable chloride conductance that is reduced by intracellular acidity. So surely then, without unequivocal proof to the contrary, one must expect that intracellular acidity will definitely be helping action potential propagation in human muscle in vivo too.

Concerning Tupling’s other point, recent results show that low-frequency fatigue is not caused by small increases in basal intracellular [Ca2+] but instead by large transient rises in local [Ca2+] near the triad junctions (7). Consequently, as we discussed previously (3), the rise in basal [Ca2+] at acid pH would be beneficial for force production, not deleterious. Just as we said concerning Juel’s study (2), the recent report (8) that exogenous lactic acid fails to slow the onset of fatigue in whole muscles stimulated in vitro simply reflects their finding that lack of tissue oxygenation makes whole muscle fatigue prematurely. Hence, this was not a test of whether intracellular acidity helps maintain excitability when a muscle is pushed hard. If testing a new wing design to enable a plane to fly at high altitude in thinner air, you wouldn’t give it a fuel line constriction and then conclude from the poor performance that the wing design was no good.

Finally, readers should not be distracted by the Editor’s headline in the Science commentary (1) on our article (6). There is no claim that lactic acid increases muscle performance above the peak level achieved in its absence. The point is that lactic acid has a vital “performance-sustaining” action.

REFERENCES


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central fatigue, which was discussed in our original study (1) so as not to exclude any potential factors, and, as argued, there was no sign of central fatigue with the subjects having a heart rate of about 120 beats/min, demonstrating that the stress on the central system was low. It is of great value to highlight this interesting topic and we should all contribute to a further understanding by posing relevant questions that can be attacked using different models. Then, when it comes to the interpretation, we have to understand the limitations in each of the models.

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