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The Case for Acidosis

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Abstract

Perhaps the strongest evidence that acidosis plays a major role in muscular fatigue has been provided by the research of Knuth and colleagues. Contradicting the results of other studies that showed little or no effect of acidosis on muscle performance at temperatures near those of the human body, they demonstrated that muscle contractile force, velocity and power were reduced by acidosis when muscle temperatures were 300C. Where muscle contraction force was concerned their results demonstrated, in agreement with other studies, that it was reduced less at higher, compared to lower temperatures. *The magnitude of that reduction differed by fiber type, however. The contraction force of slow twitch fibers was significantly reduced by 12% at a higher temperature compared to a 30% reduction at a lower temperature. In fast twitch muscle fibers, contractile force was not reduced significantly by acidosis at the higher temperature. The decrease was only 4% to 11% at a muscle temperature of 30oC versus 30% at a lower temperature (15oC).

Keywords: Acidosis; Muscular fatigue; Muscle contraction force; Muscle fibers

Introduction

The fact that the deleterious effect of acidosis on the contraction force of muscle fibers did not disappear entirely at higher temperatures in this and earlier research should not be dismissed lightly [1,2]. Reductions ranging from 4% to 11% [3] and 18% [4-7] for fast twitch fibers in two studies should have a detrimental effect on performance when the margin of victory is often measured in portions of an inch and hundredths of a second. Consequently, acidosis may have a significant detrimental effect on performance even if that effect is not as debilitating as once believed.

According to Knuth and associates, acidosis definitely had a detrimental effect on muscle contractile velocity at higher temperatures. In fact, performance was reduced more for slow twitch muscle fibers at a high compared to a low muscle temperature. They lost 25% of their contraction speed at a muscle temperature of 30°C and only 9% at a lower temperature (15°C). Reductions of contractile velocity were considerable for fast twitch fibers at both high and low temperatures although they were slightly, but non-significantly greater, at the higher temperature (27% at 15°C versus 32% at 30°C). Reductions of these magnitudes should be sufficient to reduce muscle performance at exercise body temperatures, therefore, they support the contention that acidosis can cause fatigue. This finding was in stark contrast to the results of Westerblad and associates [8], who found no slowing of fast twitch contraction velocity at higher temperatures during acidosis. They also differed with the result reported by Pate and colleagues [4] who reported a slight but non-significant increase of contractile velocity at higher muscle temperatures during acidosis.

Knuth and associates also looked at important aspects of muscle performance that were not included in previous studies. They measured the effect of acidosis on the amount of muscular force at various contraction velocities and they measured the effect of acidosis on muscle power (the product of force and velocity).

Peak power is considered the most discriminating measure of muscle performance because it reflects the ability to do work. Knuth and associates reported that power declined for both fiber types at high temperatures during acidosis, although the effect on fast twitch fibers was less pronounced. Their power diminished by 18% at 30°C versus 37% at 15°C. At the same time, slow twitch muscle fibers demonstrated a significantly greater loss of power at the high temperature. They lost 34% of their contraction power at 30°C versus only 17% at 15°C.

When the relationship of force exerted at different contraction velocities was compared, the force exerted by slow twitch muscle fibers was reduced by acidosis at each different contraction velocity when the muscle temperature was 30°C (-16%). This was less than the 25% reduction exhibited at a lower temperature but still represents a significant loss of force under loaded conditions. Fast twitch muscle fibers demonstrated less reduction in force at a higher temperature (-8% at 30°C vs. -28% at 15°C). Their contraction velocity was reduced similarly at both the higher and lower temperatures, however (-14% at 15°C vs. -11% at 30°C). Thus, when loaded, slow twitch muscle fibers lost a considerable amount of both force and velocity at a temperature of 30°C (-16% for force and -22% for velocity). On the other hand, reductions of both contractile force and velocity were considerably less for fast twitch fibers at the higher temperature (-8% for force and -11% for velocity)

Contrary to the results of others, Knuth and co-workers demonstrated that acidosis had a profoundly detrimental effect on muscle performance when that effect was differentiated according to fiber type. While the effect was more pronounced in slow twitch muscle fibers it was, nevertheless, also significant in fast twitch fibers. An 18% reduction in their muscular power at a higher temperature, although less than the 37% reduction at a lower temperature, should have been of sufficient magnitude to cause a reduction in performance.

Why did Knuth and co-workers find that acidosis caused muscular fatigue at high temperatures when the results of other researchers indicated otherwise? Perhaps it was because the studies that demonstrated little if any effect of acidosis on muscle contraction force and velocity were done with groups of mixed muscle fibers where the proportions of fast and slow twitch fibers were not identified, or with single fast twitch fibers whose contraction force and velocity were not hampered so greatly at higher temperatures. As mentioned earlier, this may have allowed fast twitch fibers to compensate for the reduced performance of slow twitch fibers in mixed samples so that muscle performance did not appear to be affected in an adverse manner. Whereas the profound effect of acidosis on single slow twitch muscle fibers was missed in studies where only single fast twitch fibers were used.

It was mentioned earlier that a reduced rate of calcium release, and not acidosis, is thought by some to be the cause of muscle fatigue. However, a compelling bit of support for acidosis as the actual cause of fatigue is that peak force decreases during acidosis even when the rate of calcium release is high in muscle fibers [5]. If an increase of inorganic phosphate were the culprit, we would expect to see an inhibition of calcium release accompanying the loss of muscular force. This suggests that an increase of hydrogen ions will cause fatigue even when the release of inorganic phosphate is high in muscles. Along this same line, the finding that the reduction in calcium release that accompanies an increase of inorganic phosphate is intensified when accompanied by an increase of hydrogen ions is another point in support of acidosis as a major factor in muscular fatigue.

Discussion

Further support for acidosis as a major cause of muscular fatigue is found in the fact that increased amounts of calcium are required to produce a particular level of force when muscle pH is low [9]. Additionally, acidosis has also been demonstrated to cause a slowing of muscle relaxation time at exercise body temperatures [2]. In this respect, Westerblad and Lannergren [7] reported muscle fibers did not take longer to relax between fatiguing contractions when acidosis was prevented. This finding supports acidosis as a major cause of fatigue.

In another attempt to substantiate the role of acidosis in muscular fatigue, Bangsbo and colleagues [1] showed that the endurance of leg muscles was reduced if the lactate concentration of the legs was first increased by arm exercise prior to testing the legs. (lactate from the arms had been transported to the legs during arm exercise and prior to leg exercise.) Of course, it could be argued that the increase of lactate was coincidental, in that, exercise may have also increased other metabolites that were responsible for reducing leg endurance. Nevertheless, this is one more indication that acidosis may cause muscular fatigue.

Conclusion

Finally, there is a theory by [6] that muscle fatigue is the result of feedback mechanisms residing in the central nervous system. A precept of this theory is that the central nervous system (the brain and autonomic nervous system) sense unusual bodily distress during exercise and try to lessen that distress by reducing (inhibiting) the rate of muscular contraction and, perhaps, by changing the pattern of muscle activation toward greater recruitment of enduring slow twitch muscle fibers in preference to more rapidly fatiguing fast twitch fibers. The rate of work would necessarily slow down when slower-contracting fibers are substituted for fibers that contract more rapidly and have a greater potential for anaerobic energy release.

The brain can sense distress from several different sources during exercise. A low muscle pH, and an unusually high heart rate are two of the more prominent indicators of distress as are an unusually high respiratory rate and high body temperature. The brain is predisposed to a certain level of distress during a particular exercise through prior exposure and will send out inhibiting messages to the heart, lungs and muscles when that level is reached even if that level does not pose an immediate danger to life and limb.

And believes that improved performances occur when those inhibiting effects are removed by “re-programming” the brain to accept a previously inhibiting level of distress as manageable. In this respect, acidosis may be the most prominent distressing event that stimulates the feedback mechanisms of pain, nausea, breathlessness, and high heart rates to reduce muscle contraction rate and change the recruitment pattern toward greater reliance on slower and less powerful fiber types. Thus, if the onset of distress and/or the severity of inhibiting feedback can be delayed through training then athletes should be able to maintain a particular level of effort for a longer period of time.

It is also possible that the effect of acidosis on performance may be mitigated by psychological factors as much or more so than by those that are physiological in nature. That is, athletes may consciously slow their efforts and their paces as acidosis and other forms of distress progress because they lose the motivation to push on, or because they become frightened that they will not

be able to complete the event at a respectable level of effort. In the next section we will consider an important question, "What are the implications of this information for training athletes?".

References

1. Bangsbo J, Madsen K, Kiens B, Richter EA (1996) Effect of muscle acidity on muscle metabolism and fatigue during intense exercise in man. J Physiol 495: 587-596.

2. Bruton JD, Linnergren J, Westerblad H (1998) Effects of CO_2-induced acidification on the fatigue resistance of single mouse muscle fibers at 28°C. J Appl Physiol 85: 478-483.

3. Knuth ST, Dave H, Peters JR, Fitts RH (2006) Low cell pH depresses peak power in rat skeletal muscle fibers at both 30°C and 15°C: implications for muscle fatigue. J Physiol 575: 887-899.

4. Pate E, Bhimani M, Franks-Skiba K, Cooke R (1995) Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: implications for fatigue. J Physiol 486: 689-694.

5. Metzger JM, and Moss RL (1987) Greater hydrogen ion-induced depression of tension and velocity in skinned single fibres of rat fast and slow muscles. J Physiol 393: 727-742.

6. Noakes TD (2000) Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. Scand J Med Sci Sports 10: 123-145.

7. Westerblad H and Lannergren J (1991) Slowing of relaxation during fatigue in single mouse muscle fibers. J Physiol 434: 323-336.

8. Westerblad H, Burton JD, Lannergren H (1997) The effect of intracellular pH of contractile function in intact, single fibers of mouse muscle declines with increasing temperature. J Physiol 500: 193-204.

9. Westerblad H, Bruton JD, Allen DG, Lannergren J (2000) Functional of Ca^{2+} in long-lasting fatigue of skeletal muscle. Eur J Appl Physiol 83: 166-174.