Lactic acid is/is not the only physicochemical contributor to the acidosis of exercise

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extracellular space. However, ABE is biased under in vivo conditions (e.g., Refs. 2, 23) because blood can interchange substances with the interstitial fluid. During acidosis HCO₃⁻ originating from the erythrocytes leaves the blood while Cl⁻ is taken up. This explains nearly completely the difference between ABE and [La]blood changes after exercise (3). Cl⁻ is a substitute for those La⁻ ions that cannot enter the red blood cells because of the Donnan effect and therefore remain in the extracellular fluid.

Medbø et al. (17) applied standard BE (SBE, common BE of blood and interstitial fluid) to avoid the effects of ion shifts, but unfortunately compared it with [La]blood and obtained a similar bias. When SBE is compared with estimated [La] in the same volume ([La]blood−interstitial fluid), the difference in the changes disappears nearly completely (3). There is no evidence for appreciable differences in La⁻ and H⁺ transport across tissue cell membranes.

Thus there remain no arguments for other acids or other mechanisms of acidification. Lactic acid is the essential cause for nonrespiratory acidosis of exercise.

REFERENCES


COUNTERPOINT: LACTIC ACID IS NOT THE ONLY PHYSICOCHEMICAL CONTRIBUTOR TO THE ACIDOSIS OF EXERCISE

TO THE EDITOR: The acidification of blood during muscular exercise cannot be understood from an analysis of blood from a single site, nor from examination of only arterial (or arterialized) blood. Acidification of blood during muscular contraction occurs as a result of rapid and marked increases in skeletal muscle mitochondrial CO₂ production and decrease of cellular HCO₃⁻ (and other CO₂ stores; 2) resulting from intra- and extra-cellular acidification. Peak efflux of CO₂ occurs during the first 30 s of high (5, 8)- and moderate (7, 9, 13)-intensity exercise. With both high (5, 8)- and moderate-intensity (7, 9) exercise, blood is also acidified due to decreases in plasma strong ion difference, or [SID], and increases in plasma [protein]. The contributions of each of these independent variables of acid-base control to the changes in plasma [H⁺] and [HCO₃⁻] have been quantified in arterial, femoral, and antecubital venous bloods during leg bicycling exercise and recovery. In femoral venous plasma the increase in PCO₂ remains the primary contributor to the plasma acidosis during exercise and the initial recovery period. In mixed venous blood, increased PCO₂ is also the primary contributor to the plasma acidosis.