Biochemistry of exercise-induced metabolic acidosis.

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The development of acidosis during intense exercise has traditionally been explained by the increased production of lactic acid, causing the release of a proton and the formation of the acid salt sodium lactate. On the basis of this explanation, if the rate of lactate production is high enough, the cellular proton buffering capacity can be exceeded, resulting in a decrease in cellular pH. These biochemical events have been termed lactic acidosis. The lactic acidosis of exercise has been a classic explanation of the biochemistry of acidosis for more than 80 years. This belief has led to the interpretation that lactate production causes acidosis and, in turn, that increased lactate production is one of the several causes of muscle fatigue during intense exercise. This review presents clear evidence that there is no biochemical support for lactate production causing acidosis. Lactate production retards, not causes, acidosis. Similarly, there is a wealth of research evidence to show that acidosis is caused by reactions other than lactate production. Every time ATP is broken down to ADP and P(i), a proton is released. When the ATP demand of muscle contraction is met by mitochondrial respiration, there is no proton accumulation in the cell, as protons are used by the mitochondria for oxidative phosphorylation and to maintain the proton gradient in the intermembranous space. It is only when the exercise intensity increases beyond steady state that there is a need for greater reliance on ATP regeneration from glycolysis and the phosphagen system. The ATP that is supplied from these nonmitochondrial sources and is eventually used to fuel muscle contraction increases proton release and causes the acidosis of intense exercise. Lactate production increases under these cellular conditions to prevent pyruvate accumulation and supply the NAD(+) needed for phase 2 of glycolysis. Thus increased lactate production coincides with cellular acidosis and remains a good indirect marker for cell metabolic conditions that induce metabolic acidosis. If muscle did not produce lactate, acidosis and muscle fatigue would occur more quickly and exercise performance would be severely impaired.