Lactic Acid Is Not The Source of Protons in Metabolic Acidosis.

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The main biochemical explanation of the metabolic acidosis accompanying intense exercise has been the production of lactic acid (lactic acidosis), causing a pH dependent release of protons due to the low pKa (pH=3.87) of lactic acid. This explanation was developed based on the pioneering work of A.V. Hill and Otto Meyerhoff prior to 1922, and has remained a rigid fixture within academia and scientific research of muscle metabolism and acid-base physiology through to present time. Such a persistent acceptance of a lactic acidosis has been unfortunate for many reasons. Review of the organic biochemistry of intermediary metabolism reveals that no acid intermediates are produced from glycolysis, that lactate not lactic acid is produced from the lactate dehydrogenase reaction, and that lactate production consumes not releases a proton. Clearly, the lactic acidosis concept has no basis of support from applications of organic chemistry and metabolic biochemistry. The biochemical cause of metabolic acidosis is an increased dependence on cellular ATP hydrolysis that is not met by mitochondrial respiration. I have referred to this as an increased dependence on non-mitochondrial ATP turnover. While some exercise and acid-base physiologists have argued that the distinction between an ATP vs. lactate explanation of acidosis is trivial, the field of acid-base physiology is a good example of how such a distinction is vitally important. For example, it has been routine for physiologists to estimate proton release during muscle contraction from estimates of lactate production. Such proton release (proton load) estimates have been used to estimate tissue (structural and metabolic) buffering capacities. An incorrect acceptance of a lactic acidosis therefore causes errors in the estimation of the proton load, which in turn would cause errors in estimates of buffer capacities. I propose that the proton load of muscle contraction far exceeds lactate production, and that therefore the tissue buffer capacity (structural + metabolic) is actually far higher than previously recognized and published based on the acceptance of a lactic acidosis. The presentation ends with data resulting from my attempts to re-compute muscle buffer capacities derived from pH dependent proton loads estimated from non-mitochondrial ATP turnover.