Acute metabolic acidosis decreases muscle protein synthesis but not albumin synthesis in humans

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Chronic metabolic acidosis induces negative nitrogen balance by either increased protein breakdown or decreased protein synthesis. Few data exist regarding effects of acute metabolic acidosis on protein synthesis. We investigated fractional synthesis rates (FSRs) of muscle protein and albumin, plasma concentrations of insulin-like growth factor-I (IGF-I), thyroid-stimulating hormone (TSH), and thyroid hormones (free thyroxin [fT(4)] and triiodothyronine [fT(3)]) in seven healthy human volunteers after a stable controlled metabolic period of 5 days and again 48 hours later after inducing metabolic acidosis by oral ammonium chloride intake (4.2 mmol/kg/d divided in six daily doses). Muscle and albumin FSRs were obtained by the [(2)H(5)]phenylalanine flooding technique. Ammonium chloride induced a significant decrease in pH (7.43 +/- 0.02 versus 7.32 +/- 0.04; P < 0.0001) and bicarbonate concentration (24.6 +/- 1.6 versus 16.0 +/- 2.7 mmol/L; P < 0.0001) within 48 hours. Nitrogen balance decreased significantly on the second day of acidosis. The FSR of muscle protein decreased (1.94 +/- 0.25 versus 1.30 +/- 0.39; P < 0.02), whereas the FSR of albumin remained constant. TSH levels increased significantly (1.1 +/- 0.5 versus 1.9 +/- 1.1 mU/L; P = 0.03), whereas IGF-I, fT(4), and fT(3) levels showed no significant change. We conclude that acute metabolic acidosis for 48 hours in humans induces a decrease in muscle protein synthesis, which contributes substantially to a negative nitrogen balance. In contrast to prolonged metabolic acidosis of 7 days, a short period of acidosis in the present study did not downregulate albumin synthesis.

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