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Metabolic and endocrine effects of metabolic acidosis in humans.

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Metabolic acidosis is an important acid-base disturbance in humans. It is characterised by a primary decrease in body bicarbonate stores and is known to induce multiple endocrine and metabolic alterations. Metabolic acidosis induces nitrogen wasting and, in humans, depresses protein metabolism. The acidosis-induced alterations in various endocrine systems include decreases in IGF-1 levels due to peripheral growth hormone insensitivity, a mild form of primary hypothyroidism and hyperglucocorticoidism. Metabolic acidosis induces a negative calcium balance (resorption from bone) with hypercalciuria and a propensity to develop kidney stones. Metabolic acidosis also results in hypophosphataemia due to renal phosphate wasting. Negative calcium balance and phosphate depletion combine to induce a metabolic bone disease that exhibits features of both osteoporosis and osteomalacia. In humans at least, 1,25-(OH)₂ vitamin D levels increase, probably through phosphate depletion-induced stimulation of 1-alpha hydroxylase. The production rate of 1,25-(OH)₂ vitamin D is thus stimulated, and parathyroid hormone decreases secondarily. There is experimental evidence to support the notion that even mild degrees of acidosis, such as that occurring by ingestion of a high animal protein diet, induces some of these metabolic and endocrine effects. The possible role of diet-induced acid loads in nephrolithiasis, age-related loss of lean body mass and osteoporosis is discussed.