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Acid-base balance may influence risk for insulin resistance syndrome by modulating cortisol output.

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Frank metabolic acidosis is known to promote renal excretion of hydrogen ion by induction of glutaminase and other enzymes in the renal tubules. This induction, at least in part, reflects an increase in pituitary output of ACTH and a consequent increased production of cortisol and aldosterone; these latter hormones act on the renal tubules to promote generation of ammonia, which expedites renal acid excretion. Recent evidence suggests that the moderate metabolic acidosis associated with a protein-rich diet low in organic potassium salts - quantifiable by net acid output in daily urine - can likewise evoke a modest increase in cortisol production. Since cortisol promotes development of visceral obesity, and has a direct negative impact on insulin function throughout the body, even a modest sustained up-regulation of cortisol production may have the potential to increase risk for insulin resistance syndrome and type 2 diabetes. This thesis appears to be consistent with previous epidemiological reports correlating high potassium consumption, or a high intake of fruits and vegetables, with reduced risk for diabetes and coronary disease. Future prospective epidemiology should assess whether the estimated acid-base balance of habitual diets - calculated from the ratio of dietary protein and potassium - correlates with risk for insulin resistance syndrome and diabetes.