Clin Nutr. 2011 Aug;30(4):416-21.

Diet-induced metabolic acidosis.

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The modern Western-type diet is deficient in fruits and vegetables and contains excessive animal products, generating the accumulation of non-metabolizable anions and a lifespan state of overlooked metabolic acidosis, whose magnitude increases progressively with aging due to the physiological decline in kidney function. In response to this state of diet-derived metabolic acidosis, the kidney implements compensating mechanisms aimed to restore the acid-base balance, such as the removal of the non-metabolizable anions, the conservation of citrate, and the enhancement of kidney ammoniagenesis and urinary excretion of ammonium ions. These adaptive processes lower the urine pH and induce an extensive change in urine composition, including hypocitraturia, hypercalciuria, and nitrogen and phosphate wasting. Low urine pH predisposes to uric acid stone formation. Hypocitraturia and hypercalciuria are risk factors for calcium stone disease. Even a very mild degree of metabolic acidosis induces skeletal muscle resistance to the insulin action and dietary acid load may be an important variable in predicting the metabolic abnormalities and the cardiovascular risk of the general population, the overweight and obese persons, and other patient populations including diabetes and chronic kidney failure. High dietary acid load is more likely to result in diabetes and systemic hypertension and may increase the cardiovascular risk. Results of recent observational studies confirm an association between insulin resistance and metabolic acidosis markers, including low serum bicarbonate, high serum anion gap, hypocitraturia, and low urine pH.