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Dietary acid load: a novel nutritional target in chronic kidney disease?

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Nonvolatile acid is produced from the metabolism of organic sulfur in dietary protein and the production of organic anions during the combustion of neutral foods. Organic anion salts that are found primarily in plant foods are directly absorbed in the gastrointestinal tract and yield bicarbonate. The difference between endogenously produced nonvolatile acid and absorbed alkali precursors yields the dietary acid load, technically known as the net endogenous acid production, and must be excreted by the kidney to maintain acid-base balance. Although typically 1 mEq/kg/day, dietary acid load is lower with greater intake of fruits and vegetables. In the setting of CKD, a high dietary acid load invokes adaptive mechanisms to increase acid excretion despite reduced nephron number, such as increased per nephron ammoniagenesis and augmented distal acid excretion mediated by the renin-angiotensin system and endothelin-1. These adaptations may promote kidney injury. Additionally, high dietary acid loads produce low-grade, subclinical acidosis that may result in bone and muscle loss. Early studies suggest that lowering the dietary acid load can improve subclinical acidosis, preserve bone and muscle, and slow the decline of glomerular filtration rate in animal models and humans. Studies focusing on hard clinical outcomes are needed.