

**Acid-base status affects renal magnesium losses in healthy, elderly persons.**

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Magnesium and calcium deficiency in humans is related to a number of pathological phenomena such as arrhythmia, osteoporosis, migraine, and fatal myocardial infarction. Clinically established metabolic acidosis induces renal losses of calcium. In normal subjects, even moderate increases in net endogenous acid production (NEAP) impair renal calcium reabsorption but no information is available whether this also influences renal magnesium handling. The aim of the study was to examine the relation between NEAP and renal magnesium excretion in healthy, free-living, elderly subjects. The subjects (age 64 +/- 4.7 y, n = 85) were randomly selected from the population register in Gothenburg (Sweden). Magnesium, calcium, and potassium were measured in 24-h urine samples and NEAP was quantified as renal net acid excretion (NAE). NAE was positively correlated with excretions of magnesium ( $R(2) = 0.27$ ,  $P < 0.0001$ ) and calcium ( $R(2) = 0.30$ ,  $P < 0.0001$ ) but not potassium. When 24-h urinary magnesium excretion was adjusted for 24-h urinary potassium excretion, a biomarker for dietary potassium intake, the association between magnesium excretion and NAE remained significant ( $R(2) = 0.21$ ,  $P < 0.0001$ ). The significant association between potassium-adjusted magnesiuria and NAE suggests that the acid-base status affects renal magnesium losses, irrespectively of magnesium intake. Magnesium deficiency could thus, apart from an insufficient intake, partly be caused by the acid load in the body.