Models of Urinary Acidification

by

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Abstract

The model of proximal tubule bicarbonate reabsorption developed by Wang and Deen has been modified to include: (1) axial variation of intracellular pH; (2) hydrogen secretion kinetics of the form \( J_H = P_H (\alpha C_{4C} - C_{4L} ) \), where \( P_H \) is apparent H\(^+\) permeability, \( \alpha \) is a measure of the secretion driving force and \( C_{4C} \) and \( C_{4L} \) are the intracellular and luminal hydronium concentrations; (3) a paracellular bicarbonate leak from the interstitial fluids to the tubule lumen; and (4) ammonia and phosphate buffers. The model predicts the intracellular pH to be higher than luminal pH everywhere except in the initial portion of the proximal tubule. Hydrogen secretion decreases with increasing luminal acidity. The ammonia and phosphate buffer systems and the paracellular bicarbonate leak result in higher luminal pH in the distal tubule segments. The paracellular leak reaches 12-15% of the bicarbonate efflux into the interstitial fluid by the proximal tubule end. By increasing the paracellular permeability coefficient, a steady state luminal pH is attained. Reabsorption rates of \( tCO_2 \) of 120-140 pmol/min/mm are predicted. Fractional reabsorption of phosphate is greater than 70%. The luminal concentration of monovalent phosphate increases to levels higher than plasma values so that passive transport of monovalent phosphate is sustained over the length of the proximal tubule. Total ammonia content increases to .4mM by the tubule end. A 50% increase in SNGFR results in a 7-23% increase in absolute \( tCO_2 \) reabsorption but a 30-33% decrease in fractional \( tCO_2 \).

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