Transport and Reaction of CO_2 in the Kidney

by

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Abstract

Theoretical models were developed to examine the mechanisms of renal proximal tubule HCO_3^- reabsorption and blood buffering in terms of recent micropuncture measurements of single nephron bicarbonate reabsorption rate (R_B) and CO_2 partial pressure (PCO_2) in surface nephrons of the rat. Axial and radial variations in the concentrations of HCO_3^-, CO_2, and related species were included in the proximal tubule lumen and epithelial cells. Plasma and red cells of the surrounding peritubular capillaries were treated as distinct, well-mixed compartments. R_B is predicted to increase with the filtered HCO_3^- load (L_B) for $L_B < 1400$ pmole/min, and to be relatively constant beyond this value. The fraction of filtered HCO_3^- reabsorbed in therefore predicted to decrease significantly when L_B is elevated well above the normal level of about 1200 pmole/min, in excellent agreement with most of the available data. Capillary PCO_2 is predicted to depend critically on the amount of CO_2 generated metabolically in the epithelial cells, and on the amounts of HCO_3^- and water reabsorbed. Predictions of capillary and transepithelial PCO_2 from the model under normal conditions are consistent with those measured experimentally. Using the model, these predictions are extended to conditions of interest for which no data are yet available. A second model was developed to investigate the source of the large PCO_2 difference observed between the surface of the kidney and the renal artery. Countercurrent CO_2 exchange between interlobular arteries and veins, which supply and remove blood to and from the cortex, is shown to be responsible for this PCO_2 difference. Taken together the single nephron and countercurrent exchange models provide a reasonably complete description of the handling of HCO_3^- and CO_2 in the renal cortex, which forms the basis for renal control of blood bicarbonate levels.

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