



Figure 14.7 Reabsorption of filtered HCO_3^- in the proximal tubule, the ascending loop of Henle, and the initial part of the distal tubule. For every H^+ that reacts with filtered HCO_3^- in the tubular lumen, one HCO_3^- is transported from the epithelial cells and into the blood, together with Na^+ . c.a. = carbonic anhydrase.

the extracellular fluid (alkalosis), more HCO_3^- is lost in the urine, and the concentration of H^+ in the extracellular fluid is thereby raised towards normal. In contrast, during acidosis, the kidneys reabsorb all the filtered HCO_3^- and also generate new HCO_3^- (see later) that enters the blood. The concentration of H^+ in the extracellular fluid is thus restored towards normal.

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38 How much of the filtered HCO_3^- is normally reabsorbed in an omnivorous animal?

39 In which part of the nephron is most of the filtered HCO_3^- reabsorbed?

40 Discuss the mechanisms for renal reabsorption of HCO_3^- .

Generation of HCO_3^- in the Kidneys

As noted, carnivores continuously consume HCO_3^- for buffering the net production of H^+ during metabolism. In order to compensate for this loss of HCO_3^- , carnivores continuously generate HCO_3^- . As with reabsorption of filtered HCO_3^- , generation of new HCO_3^- is coupled to secretion of H^+ by tubular cells (intercalated cells) in the distal tubules and the collecting ducts (Fig. 14.8). In animals with a net production of acids, the tubular cells transport more H^+ into the renal tubules than is needed for complete reabsorption of filtered HCO_3^- .

The excess H^+ is excreted in the urine, and it is this excess production of H^+ that is coupled to *de novo* generation of HCO_3^- . In a stable situation, the generation of HCO_3^- keeps pace with the net production of non-volatile acids. In the intercalated cells, HCO_3^- is transported through the basolateral membrane in exchange with Cl^- .

The secondary active secretion of H^+ in the proximal tubule is driven by the electrochemical gradient for Na^+ between the tubular lumen and the cytosol of the epithelial cells. However, the energy provided by this gradient is insufficient to create a steep H^+ gradient. Therefore, the pH of the tubular fluid can only be lowered to about 6.8 in the proximal tubule, despite the fact that there is a large *transport capacity* for H^+ in this part of the nephron. This is in contrast to the situation in the last part of the distal tubules and in the collecting ducts, where primary active H^+ secretion takes place through the apical membranes of the *intercalated cells* (Fig. 14.8). The active H^+ secretion is driven either by an H^+ ATPase, or an H^+ - K^+ ATPase that reabsorbs one K^+ for each H^+ secreted. This primary active H^+ secretion can increase the H^+ concentration in the lumen about 900-fold.

Buffering of H^+ in the Renal Tubules

The lower pH limit for urine is about 4.5. However, lowering the tubular pH to 4.5 does not represent excretion of a large amount of *free* hydrogen ions. Therefore, in order for animals with a significant net production of acids to be able to

Renal synthesis of HCO_3^- replaces consumed HCO_3^-

The secretion of H^+ in the distal tubules and the collecting ducts is primary active

The lower pH limit for urine is about 4.5

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41 Compare H^+ secretion in the proximal tubule and in the last part of the nephron.

42 Compare the lower limits for pH in the proximal tubule and urine.