K^+ in plasma is regulated through variations in K^+ secretion

cells secrete K^+ to an extent that normalizes its concentration in plasma.

As outlined above, the K^+ concentration in the extracellular fluid is mainly regulated by adjustments of K^+ secretion in the last part of the renal nephrons. Changes in GFR, on the other hand, are not normally of particular significance for K^+ excretion. However, when GFR is reduced in renal disorders, the amounts of K^+ filtered may become too low to counterbalance the dietary intake when the K^+ content of the feed is high. The concentration of K^+ in plasma may then become abnormally high (hyperkalemia).

In herbivores, the amounts of K^+ in the food may exceed the amounts filtered by the kidneys

The renal handling of K^+ may appear unnecessarily complex when compared with the handling of Na^+. However, the amounts that have to be excreted relative to those filtered may differ considerably for the two ions. The concentration of Na^+ in extracellular fluid is high (approximately 145 mmol/L), and there is never any need for urinary excretion of Na^+ to exceed the amounts filtered. For K^+, however, more than 98 % of the total body content is contained within the cells, and its concentration in extracellular fluid (approximately 4 mmol/L) is low compared with that of Na^+. If the diet contains large amounts of K^+, as it typically does for herbivores, it may be necessary to excrete more K^+ than the amounts filtered. In such situations, it would be impossible to maintain K^+ homeostasis if renal handling of K^+ occurred in the same manner as for Na^+.

K^+ secretion is regulated by aldosterone

Secretion of K^+ in the distal tubules and the collecting ducts is coupled to the Na^+-K^+ pump in the basolateral membrane of the epithelial cells. When Na^+ is pumped out of the cells, K^+ is simultaneously pumped in. The intracellular concentration of K^+ is therefore maintained at a high level, thereby providing a driving force for diffusion of K^+ out of the cells. The diffusion of K^+ into the tubular lumen takes place through selective K^+ channels in the apical membrane of the epithelial cells in the distal tubule and collecting duct (Fig. 13.20). In contrast, the apical membrane of the tubular cells in the proximal tubule does not contain K^+ channels. Consequently, there is no secretion of K^+ in this part of the nephron.

Several factors influence the secretion of K^+. The most important regulator is aldosterone, which simultaneously increases the reabsorption of Na^+ and the secretion of K^+. Besides increasing the activity of the Na^+-K^+ pump in the basolateral membrane, aldosterone increases the number of K^+ channels in the apical membrane of the epithelial cells in the distal part of the nephron (Fig. 13.20). A rise in the concentration of K^+ in plasma stimulates secretion of aldosterone, thereby increasing the excretion of K^+, which gradually normalizes its plasma concentration (Fig. 13.21).

Aldosterone enables the kidneys to regulate the concentration of K^+ in plasma very precisely. This is important, because fluctuations in the concentrations of K^+ in plasma can have adverse effects. The ratio between the extracellular and cytosolic concentrations of K^+ plays a key role in determining the value of the resting membrane potential (p. 67). Heart arrhythmias and weak heart contractions readily arise if the concentration of K^+ in plasma more than doubles.

The interplay between renal handling of Na^+ and K^+

Because aldosterone always increases reabsorption of Na^+ and secretion of K^+ (Fig. 13.22), the homeostatic regulation of the two ions could,

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74 Where are ANP, BNP, and urodilatin produced, and what are their effects on renal excretion of Na^+ and water?

75 Compare the renal handling of Na^+ and K^+.

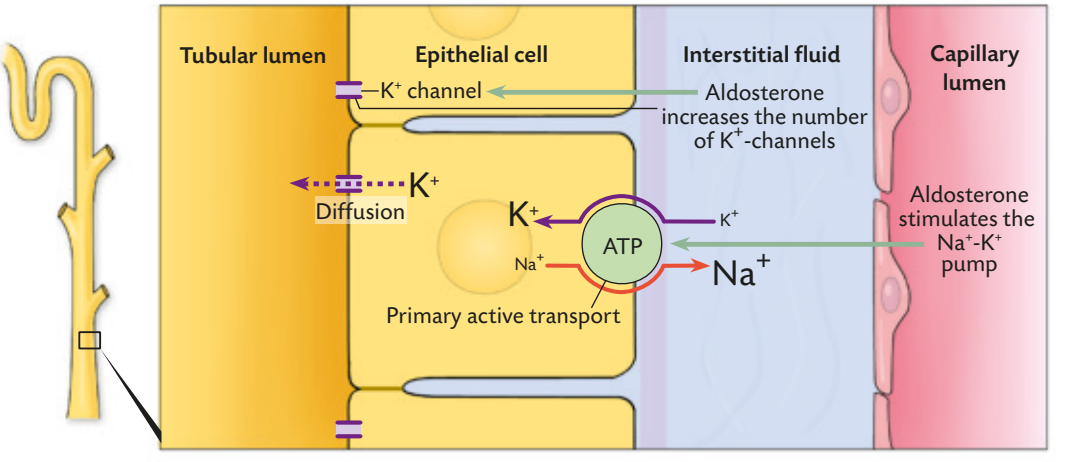

Figure 13.20 K^+ secretion in the distal tubules and the collecting ducts. Na^+-K^+ pumps transport K^+ from the interstitial fluid into the epithelial cells. K^+ then diffuses into the lumen through ion channels in the apical membrane.