Mechanisms for K transport in the renal collecting duct

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Summary

Previous studies indicated that an acute elevation of peritubular K enhances K secretion and Na reabsorption in the isolated perfused cortical collecting duct (CCD) from rabbit kidneys. To determine the underlying cellular mechanisms, we used microelectrode techniques to assess the membrane properties of principal collecting duct cells in isolated perfused CCDs from rabbit kidneys after raising bath K from 2.5 to 8.5 mM. This induced, first, a short-lasting hyperpolarization and second, a sustained phase of depolarization of transepithelial, basolateral, and apical membrane voltages. Whereas the transepithelial conductance (GT) and fractional apical membrane resistance (fRA) remained unchanged during the initial phase of hyperpolarization, GT increased during depolarization and fRA decreased. Perfusion of the lumen with solutions containing either amiloride or Ba partially inhibited the high K-induced apical electrical changes, and basolateral strophanthidin abolished both apical and basolateral electrical responses during elevation of K in the bath. Addition of Ba to the bath had no effects on the high K-induced electrical changes during either phase. From these results we conclude that acute elevation of basolateral K activates the Na pump in the basolateral membrane, which secondarily elevates the activity of both Na and K channels in the apical membrane.