Luminal Na-independent K secretion in the renal cortical collecting duct

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Summary

We previously demonstrated that raising bath K from 2.5 to 8.5 mM in the presence of luminal Na in the isolated perfused rabbit cortical collecting duct (CCD) resulted in an initial hyperpolarization of transepithelial voltage (V_T) and basolateral membrane voltage (V_B), followed by a delayed depolarization, and that the initial phase was due to basolateral electrogenic Na pump stimulation and the late phase was due to stimulation of the apical Na and K conductances (Am J Physiol 276: F143-F158, 1999). In the present study, we used the microelectrode technique to determine the effects of raising bath K on apical Na and K conductances, and basolateral K conductance and Na/H exchange (NHE) in the absence of luminal Na in the isolated perfused rabbit CCD. Acute increase in the bath K from 2.5 to 8.5 mM in the absence of luminal Na abolished the early phase of hyperpolarization of V_T and V_B, but still depolarized V_T and V_B with increased both transepithelial conductance (G_T) and fractional apical membrane resistance (fR_A). The high bath K-induced depolarization of V_T and V_B and increased G_T and fR_A were partially inhibited by addition of Ba (a K channel inhibitor) in either the luminal or the basolateral side and by addition of ethylisopropylamiloride (a specific inhibitor of the NHE) in the basolateral side, but were not affected by luminal addition of amiloride (a Na channel inhibitor). Acute increase in the bath K also increased net K secretion. We conclude that, in the rabbit CCD, raising bath K in the absence of luminal Na leads to an increase in K secretion, which is mediated by Na pump, K conductance, and NHE in the basolateral membrane and by K conductance in the apical membrane.