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# Effects of extracellular chloride ion on epithelial sodium channel (ENaC) in arginine vasotocin (AVT)-stimulated renal epithelial cells

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## ABSTRACT

The epithelial $Na^+$ channel (ENaC) contributes to control of blood pressure by reabsorbing $Na^+$ in the cortical collecting duct of the kidney. The luminal $Cl^-$ concentration in the duct varies under physiological conditions. As the body $Na^+$ content is lower, the luminal $Cl^-$ concentration in the duct becomes lower. Thus, we hypothesized that the extracellular $Cl^-$ elevates ENaC activity in AVT-stimulated renal epithelial A6 cells (a model cell line of the cortical collecting duct) leading to recovery from a low body $Na^+$ content. To clarify this point, we studied effects of extracellular $Cl^-$ concentration on ENaC activity using cell-attached patch clamp technique. We found that ENaC had a single-channel conductance of 4.6 ± 0.1 pS (mean ± SE) and channel activity (open probability, Po) of 0.30 ± 0.02 at a pipette potential of 60 mV. Lowering pipette $Cl^-$ concentration diminished Po to 0.23 ± 0.02 associated with a significant decrease in open time from 0.78 ± 0.03 to 0.61 ± 0.02 s with no significant change in closed time, and shifted the current-voltage relationship leftward. These results suggest that the extracellular $Cl^-$ regulates the ENaC-mediated $Na^+$ reabsorption by affecting ENaC properties in AVT-stimulated renal epithelial cells.

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