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Mechanisms of ENaC Life Time Regulated by Protease

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Summary

Although it is well known that aldosterone and osmotic stress regulate the epithelial Na^+ channel (ENaC)-mediated Na^+ transport in renal epithelial cells, no information is available on the interaction of aldosterone and osmotic stress on stimulation of ENaC-mediated Na^+ transport in renal epithelium. In this report, we investigated how aldosterone of 1 μM applied for 1 day modifies the action of hypotonic stress on the ENaC-mediated Na^+ transport in renal A6 epithelial cells by measuring the benzamil (a specific inhibitor for ENaC)-sensitive short-circuit current. Further, we studied the effect of protease inhibitor, aprotinin (40 μg/ml), on the hypotonic stress-induced benzamil-sensitive short-circuit current. This study indicates that: 1) in the cells without aldosterone treatment most ENaCs are translocated to Golgi apparatus, 2) in aldosterone-treated cells major parts of ENaCs are located at the endoplasmic reticulum, 3) the endocytosis rate of ENaCs from the apical membrane is diminished by aldosterone without any significant changes in the insertion rate of ENaCs into the apical membrane, and 4) aprotinin diminishes both the insertion rate of ENaCs into the apical membrane and the endocytosis rate of ENaCs from the apical membrane.