Abstract

Introduction. The colonic epithelium is a classical aldosterone target, but the effect of the hormone on the oxygen consumption rate (QO2) of this tissue is unknown. Objectives. We aimed at assessing, in the rectal epithelium of rats fed with diets of different sodium content, the effect of epithelial sodium channel (ENaC) blockade on short-circuit current (ISC) and QO2, and the acute effect of aldosterone incubation on ISC and QO2. Methods. Adult male rats were fed with normal, low or high-sodium diets for 8 days. Plasma sodium and serum aldosterone were measured. Isolated mucosa preparations from the rectal portion of the colon were mounted in Ussing chambers modified to measure ISC and QO2. Results. Baseline ISC and QO2 were highest in sodium-deprived rats. Both were proportionally reduced by amiloride (0.1 mM) in this group and in the normal sodium group, but not in sodium-loaded rats. In separate experiments, incubation with aldosterone (10 nM) for 7 h increased ISC and QO2 in all groups; increases were larger in the normal and sodium-loaded groups. Amiloride decreased both ISC and QO2, abolishing the differences between groups. Linear regression of the decrease in QO2 and ISC after amiloride showed the steepest slope for the sodium-deprived group and the flattest one for the sodium-loaded group. Conclusions. Baseline epithelial QO2 of sodium-deprived and control rats is reduced by ENaC blockade. Aldosterone increased QO2 proportionally to ISC augmentation in all groups, but the coupling between aerobic metabolism and electrogenic transport seems more efficient in sodium-deprived animals.

Keywords

Aldosterone, ENaC, oxygen consumption, rat rectal colon, short-circuit current, sodium intake.