Abstract

Chronic hypobaric hypoxia is a physiological environmental stressor. While its effects on most major organ systems have been extensively studied, few works have addressed hypoxia-induced changes in intestinal transport. The effects of cyclooxygenase blockade with indomethacin on short-circuit current (Isc) and oxygen consumption (QO2) of the distal colonic epithelium of control rats and rats submitted to hypoxia for 10 days at 0.52 atm were studied. Isolated mucosae were mounted in an Ussing chamber modified for measuring QO2 while preserving transepithelial vectorial transport. Amiloride was added to the mucosal hemichamber to block a sodium component of Isc present in hypoxic rats. In this condition, basal Isc did not differ between the hypoxic and the control group, but QO2 was higher in the former. Indomethacin (30 ¿mol/L) reduced Isc to the same extent in both groups, but QO2 reduction was larger in the hypoxic group. Pharmacological blockade of chloride secretion and a low-chloride solution abolished the indomethacin-induced reductions of Isc in both groups, and the reduction of QO2 in controls, and attenuated but did not suppress the QO2 reduction in the hypoxic group. Linear regression analysis of QO2 changes versus Isc changes yielded a significant correlation for both groups, with regression lines with the same slope, but a higher position in hypoxic animals. Results suggest that spontaneously released prostaglandins are equally important for maintaining colonic chloride secretion in hypoxic as in normoxic rats, but that, in the former, indomethacin has an additional effect on QO2 which is unrelated to ion transport.

Keywords

Colon, Hypoxia, Indomethacin, Ion transport, Oxygen consumption.