MEMBRANE POTENTIALS IN THE ISOLATED SMALL INTESTINE OF THE BULLFROG

by

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V. SUMMARY

Using microelectrodes filled with 1 molar potassium citrate and with tip resistances between 50 and 150 megohms, mucosal and serosal membrane potentials were measured in epithelial cells of isolated bullfrog small intestine. Simultaneously, the potential across the entire epithelium (the transmural potential) was measured. Experiments were conducted in the presence and absence of the amino acid valine and a number of sugars.

In sodium sulfate Ringer's, in the absence of transported solutes, the mucosal membrane potential was -49 ± 2 mV (SEM) (cell interior negative to mucosal and serosal bathing solutions) and the serosal membrane potential was -51 ± 1 mV. In sodium chloride media, the mucosal membrane potential was -43 ± 2 mV and the serosal membrane potential was -44 ± 2 mV. In all instances, the recorded transmural P.D. equalled the algebraic sum of the mucosal and serosal P.D.'s measured in the same preparation.

Several solutes were tested for their effect on the membrane potentials in sodium sulfate media. The actively transported sugars, glucose and galactose, decreased the mucosal membrane potential and elevated the transmural potential (the serosa becoming more positive with respect to the mucosa). The decrease in the mucosal membrane

potential exceeded the increase in the transmural potential implying a real but smaller decrease in the serosal membrane potential. The sugar sorbose, which is not actively transported, did not alter either the membrane potentials or the transmural potential. Valine, an actively transported amino acid, altered these electrical potentials in a manner qualitatively similar to that of the actively transported sugars.

Phlorizin, an inhibitor of intestinal sugar transport, prevented or reversed these electrical changes. The membrane and transmural potentials were unaltered when the tissue was exposed to Ringer's containing glucose and phlorizin. Phlorizin, which reduces the transmural potential elevated by galactose, increased the mucosal and serosal membrane potentials previously reduced by the same sugar. The electrical responses produced by the amino acid valine were not inhibited by phlorizin. This inhibitor alone did not alter these electrical parameters at the concentrations used.

The depolarization by actively transported solutes of the mucosal membrane potential, found in this investigation, is consistent with an increased permeability of this membrane to sodium. The overall effect of these two solutes on the mucosal and serosal membrane potentials are interpreted in terms of three possible physiological mechanisms:

1. Actively transported solutes decrease both membrane potentials equally, the transmural potential arising from current flow across series resistance elements located below the cell surface.

2. Actively transported solutes elevate the transmural potential by reducing both membrane potentials, the change in the mucosal membrane potential exceeding that of the serosal.

3. Actively transported solutes reduce the mucosal membrane potential but attenuation of the resulting increase in E_{tr} occurs due to the existence of extracellular ionic shunts.

These findings are not consistent with the hypothesis that an electrogenic sodium pump in the serosal membrane is solely responsible for the elevation of the transmural potential in the presence of actively transported solutes.

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