Damietta University
Faculty of Science
Zoology Department

Up date survey on

Kidney tissues affected by di methyl sulphoxide (DMSO)

Prepared by:

Nayra Ahmed Mohmed

B. Sc. Student

Zoology and chemistry division

Under supervision:

Dr .Nahed Ahmed Omar

Lecturer of histology & histochmistry

Faculty of Science, Damietta University

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Abstract

The effects of dimethylsulfoxide, DMSO, and mercurial sulfhydryl reagents have been studied on water and small solute permeability of rat renal brush border membrane vesicles. Water and solute permeability was measured by mixing membrane vesicles with hypertonic solutions in a stopped-flow apparatus and following osmotically-induced changes in vesicular volume via changes in scattered light intensity. The rate constant of the fast osmotic shrinkage is proportional to the osmotic water permeability, while the rate constant of the slow reswelling phase is proportional to the solute permeability. Using mannitol as the osmotic agent, the osmotic shrinkage of rat renal brush border membrane vesicles followed a biphasic time course. 80% of the vesicles shrunk with a rate constant of approx. 50 s^{-1} and 20% with a rate constant of approx. 2 s^{-1}, DMSO decreased dose-dependently the amplitude of the fast osmotic shrinkage, without affecting its rate constant. In contrast to DMSO, $HgCl_2$ decreased the rate constant but not the amplitude of the fast osmotic shrinkage of renal brush border vesicles. Between 40–50 μM $HgCl_2$, the inhibition of the fast osmotic shrinkage was completed. DMSO and $HgCl_2$ increase the activation energy of water permeation in renal membranes from 3 to 12–15 kcal/mol. DMSO and $HgCl_2$ did not affect the rate constant of the slow osmotic shrinkage of renal membrane vesicles and were also without effect on osmotic shrinkage of small intestinal brush border and pure phospholipid vesicles. In renal brush border membranes, $HgCl_2$ at low concentrations (<10 μM) increased by 15-fold the permeability to NaCl and urea but not to mannitol, an effect which precedes the inhibition of water permeability at higher $HgCl_2$ concentrations. The increase in small solute permeability was irreversible

while the inhibition of water permeability could be reversed with cysteine and dithiothreitol. We conclude that water and small solute pathways in rat renal brush border membranes are completely separate entities, which are effected differently by DMSO and $HgCl_2$. These pathways for water and solutes must be membrane proteins since neither DMSO nor $HgCl_2$ affect the permeability properties of pure phospholipid vesicles