Modulation by extracellular Cl- of volume-activated organic osmolyte and halide permeabilities in HeLa cells

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Organic osmolyte and halide permeability pathways activated in epithelial HeLa cells by osmotically induced cell swelling were studied using electrophysiological and radiotracer efflux techniques. On hypotonic challenge, HeLa cells responded by activating an efflux pathway for [3H]taurine and a swelling-induced outwardly rectifying Cl- channel. Removal of extracellular Cl-, or its replacement by a less permeable anion, enhanced taurine efflux and decreased the inward current (Cl- efflux). The effect of Cl- removal on taurine efflux was not a consequence of changes in membrane potential. The degree of deactivation of the Cl- current at depolarized potentials was also Cl- dependent, suggesting that external Cl- is necessary for channel activity. The Cl- channel inhibitors 1,9-dideoxyforskolin, tamoxifen, and 4,4'-diisothiocyanostilbene-2,2'-disulfonic acid (DIDS) inhibited swelling-activated taurine efflux, with DIDS being the most potent, at variance with sensitivity of the Cl- channel