MODULATION OF TAURINE TRANSPORT IN RECTAL GLAND CELLS OF THE SHARK (SQUALUS ACANTHIAS)

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Cell taurine (50 mM) in the shark rectal gland may contribute to cell volume maintenance. We studied several factors that may modulate uptake and efflux of taurine and the accompanying changes in cell volume. The methods for work with rectal gland slices were as previously described (Ziyadeh et al., Biochim. Biophys. Acta 943:43, 1988).

Taurine uptake is achieved by Na^+-dependent cotransport carrier, presumably located at the basolateral membrane (Ziyadeh et al., loc.cit.). We now report that the stoichiometry of the transport system is $2Na^+$/1taurine (Hill plot) as described previously for erythrocytes of euryhaline fish (Fincham et al., J. Membr. Biol. 96:45, 1987) and rabbit kidney (Wolff & Kinne, ibid. 102:131, 1988). Removal of media Cl^- inhibited taurine uptake by 30% (Ziyadeh et al., loc.cit.). Whether in the rectal gland Cl^- solely provides catalytic activation of Na^+-dependent carrier or is also cotransported via $2Na^+/1Cl^-$/1taurine remains to be elucidated.

The efflux of taurine is relatively slow despite very steep gradient favoring exit from the cell. Addition of 0.1 mM DIDS did not affect taurine efflux significantly suggesting that exit does not involve an anion exchange process. The transport system responsible for taurine exit remains to be determined although passive diffusion may account for this mechanism.

The influence of media pH (Tris/Tes buffers) on the kinetics of taurine uptake revealed that alkaline pH increased Vmax and Km (lowered affinity) of the physiologically-relevant high-affinity (Km = 60μM) taurine uptake system. Interaction between the amine group of taurine (pKa = 9) and the β-amine membrane carrier may involve an ionizable group(s) on the carrier and/or altered bonding energetics (cf. Fairley & Walker, J. Membr. Biol. 98:191, 1987).

Addition of 0.1 mM 8-p-chlorophenylthio-cyclic AMP (cAMP), which stimulates Cl^- secretion, reduced taurine uptake by 35% at 3 h, tended to slightly increase the fast rate constant of taurine efflux (without affecting the slow rate constant) and shrank the cells by 5%. The Cl^--secretagogue VIP (1 μg/ml) reproduced the effects of cAMP; taurine uptake was reduced by 25%. However, the combined effect of bumetanide (0.2 mM) plus VIP abrogated the decrement in uptake. Bumetanide alone did not affect taurine uptake (cf. Ziyadeh et al., loc.cit.). Hence, cAMP-inhibition of taurine uptake may be related to the intact operation of the 1Na-1K-2Cl cotransport pathway.

The role of Ca^{++} in rectal gland secretory function remains incompletely understood, in contrast to its central role in other Cl^--secretory epithelia (for a review see Ziyadeh & Agus, Miner. Electrol. Metab. 14:71, 1988). We found no effect of Ca^{++}-ionophore A23187 (10μM) or phorbol 12-myristate 13-acetate (1μM) on taurine uptake and efflux, and on cell H_2O, Na^+ and K^+ content at 2 and 3h. This is contrasted with the marked stimulation of taurine efflux in skate erythrocytes by A23187 and phorbol ester (Leite & Goldstein, J. Exp. Zool. 242:95, 1987). We also found that Ca^{++}-free media (plus 0.1 mM EGTA) were without effect on taurine efflux; however, taurine uptake was reduced by 25% at 3h possibly reflecting altered Ca^{++}-related interaction of taurine with its membrane carrier.

These data show that taurine transport by rectal gland cells can be modulated by media composition (Na^+, Cl^-, Ca^{++}, pH) and by VIP and cAMP.

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