methazolamide concentration of 1 μM after forty minutes of drug free perfusion. Maren and Friedland (Bull. MDIBL, 1978) showed that the concentration of carbonic anhydrase in the rectal gland is 9.8 μM; thus our value of 1 μM is consistent with drug binding to gland enzyme and subsequent dissociation during control periods.

It is therefore reasonable to suppose that in these and similar experiments the enzyme is fully inhibited, certainly as much as in mammalian systems (i.e., kidney, aqueous humor, CSF, pancreas) in which a clear-cut response is elicited. A failure of response in dogfish rectal gland to carbonic anhydrase inhibitors does not appear due to lack of sensitivity of the enzyme or lack of access of drug.

It may be noted, however, that the absolute rates of sodium turnover in the rectal gland are rather low compared with rates in organs that are affected by carbonic anhydrase inhibition. Rectal gland in the basal state secretes about 5 μeq Na/min per gram of gland; this is increased some 6-fold by stimulation with theophylline + dibutyryl cyclic AMP (Silva *et al.*, *vide supra*) or by vasoactive intestinal peptide (Stoff *et al.*, Am. J. Physiol. 237:F138, 1979). Even these highest rates, however, are considerably lower than renal sodium reabsorption (about 150 μeq/min per gram kidney) or avian salt gland secretion (about 300 μeq/min per gram gland). Such comparisons invite the suggestion that catalysis is unnecessary in the rectal gland because of the low rate. On the other hand the enzyme *is* present, and there may be a buried or potential role of carbonic anhydrase still not revealed by present experiments. Reasons for continuing this assumption include the facts that high pCO_2 (Siegel *et al.*, *vide supra*) and HCO_3 removal (F. H. Epstein, personal communication) both lower secretion. Additionally, cytoplasmic and membrane associated carbonic anhydrase in the rectal gland (Maren and Friedland, *vide supra*) are highly resistant to inhibition by chloride, $I_{50} \sim 1.6$ M, while for dogfish red cell carbonic anhydrase the $I_{50} = 70$ mM. Thus the rectal gland enzyme displays a striking and unusual characteristic necessary for a role in the secretion of a highly saline fluid. It is still tenable that within the rectal gland a relatively high HCO_3 or alkaline milieu involving carbonic anhydrase subserves NaCl secretion, as it does in the avian salt gland and certain other secretory systems (Maren, Physiol. Rev. 47:595, 1967). Failure of response to inhibition of the enzyme, however, remains unexplained.

We thank Mr. Jonathan Epstein for carrying out the perfusion studies. Supported by NIH Grant NIH HL 22258.

TRANSPORT OF 2-DEOXY-D-GLUCOSE AND D-MANNITOL IN THE WINTER FLOUNDER INTESTINE

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The bidirectional transepitnelial fluxes of 2-deoxy-D-glucose and D-mannitol were studied in a continuation of previous work. The fluxes were measured as previously described (Naftalin, R., D. E. Pew, and A. Kleinzeller, Bull. MDIBL 18:107, 1978). The surface area of each port of system was 0.94 cm^2. The serosal-mucosal flux (J_{sm}), the mucosal-serosal flux (J_{ms}), and the net flux (J_{net}) were determined. The unidirectional entry and exit fluxes across both faces of the epithelium (R) were also determined (Naftalin, R. and Curran, P.E., J. Mem. Biol. 16:257-258, 1974).

The flux of 2-deoxy-D-glucose was not affected by 0.1 mM ouabain, 0.1 mM phlorizin, or the addition of glucose (5 mM) to either the serosal side or to both sides of the intestine. The addition of ouabain did however cause a slight decrease in the mucosal uptake as indicated by the R value of 0.261 ± 0.023 as compared to the control value of 0.460 ± 0.009 which may indicate a slight sodium dependence of the transport. Ouabain also caused a small decrease in the phosphorylation of the sugar.

TABLE 1

Uptake of 2-Deoxy-D-Glucose in winter flounder intestine

	Sugar Fluxes			Tissue Sugar		R
	J_{ms}	J_{sm}	J_{net}	Total	Free	
	nmol/cm^{2}hr			μmole/gWW		
Control (n=12)	25±2	22±1	3±3	0.749±0.029	0.410±0.018	0.460±0.009
Glucose (5mM) serosal (n=3)	26±3	31±3	-5±4	0.682±0.065	0.701±0.067	0.495±0.103
Glucose (5mM) s + m (n=3)	27±2	31±3	-4±2	0.718±0.069	0.744±0.069	0.524±0.068
Phlorizin serosal (0.1mM) (n=3)	36±2	32±4	4±2	0.760±0.045	0.322±0.031	0.538±0.028
Ouabain 0.1mM serosal (n=3)	25±4	30±	-5±3	0.733±0.90	0.517±0.028	0.261±0.023

All values are mean values ± S.E.M. (n = number of fish).

m: mucosal s: Serosal

TABLE 2

Uptake of D-mannitol by winter flounder intestine

	Tissue Sugar Free (μmol/gWW)	R
Control (n=4)	0.579 ± 0.039	0.435 ± 0.017
Glucose, 11mM (n=4)	0.685 ± 0.085	0.434 ± 0.027

No phosphorylation products; values of J_{ms}, J_{sm}, and J_{net} were zero (n=6).

The mannitol fluxes were found to be zero for J_{ms}, J_{sm}, and J_{net}. This observation contrasts with the results of H. N. Nellans and D. V. Kimberg (Am. J. Physiol. 236 (5) E474, 1979) who studied mannitol transport in the rat intestine and found a significant secretion of this carbohydrate. The addition of glucose (11 mM) did not significantly increase the uptake of mannitol into the tissue and the R values, indicating a higher rate of entry from the serosal side, were not affected by the presence of glucose on both sides of the epithelial membrane. Tables 1 and 2.

This work was supported in part by USPHS Grant AM 12619 and the Whitehall Foundation.