The TEP of the IPHP in seawater was 7.7 ± 1.4 mV (N = 7), while that recorded in vivo was 7.2 ± 1.6 (N = 6). The similarity between these values may suggest that the IPHP exhibits the same overall differential ion permeability and electrogenic ion transport as in vivo. Table 1 shows the effect of Na- and Cl-free seawater on the TEP in vitro and in vivo. Na-free seawater depolarized the TEP of the sculpin to a much greater extent than the hyperpolarization by Cl-free media, in both the IPHP and the whole animal. This is an indication that the gills of the IPHP of the sculpin (and in vivo) possess a higher permeability to Na than to Cl. This is not surprising since the branchial epithelium of several other teleosts which are serosally electropositive exhibit a Na/Cl permeability ratio greater than 1 (see review by Kirschner, Am. J. Physiol., 7(2):R219-R223, 1980). Cl-free seawater did not effect the TEP of the IPHP, but hyperpolarized the TEP of the whole animal. This suggests that the IPHP retains a lower Cl permeability than in vivo, thereby limiting the rate of Cl efflux from the preparation. We have shown previously that this is indeed the case, in that the rate of isotopic Cl efflux from the IPHP appears to be below in vivo rates (Claiborne and Evans, 1981, op.cit.), though the underlying reasons for the Cl permeability changes in the IPHP of the sculpin are yet to be resolved.

To date, the TEP across an isolated head preparation has not been reported in the literature. This parameter is a good test of the viability and branchial permeability of the IPHP and should provide useful information when monitored concurrent with gill ion transport studies. This research was supported by NSF grant PCM 81-04046 to DHE.

INHIBITION OF CHLORIDE SECRETION BY $BaCl_2$ IN THE RECTAL GLAND OF THE SPINY DOGFISH, SQUALUS ACANTHIAS

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Soluble barium salts are known to produce poisoning, fatal if untreated, that is characterized by hypokalemia and muscle paralysis. The hypokalemia is thought to be due to the capacity of barium to decrease the efflux of potassium out of cells, although inhibition of both influx and efflux of potassium has been noted. The capacity of barium to reduce passive efflux of potassium has been used experimentally to probe transport characteristics of different epithelia. Inhibition of potassium efflux should result in depolarization of the cell membrane with consequent effects on the membrane transport of other ions. We have postulated a model for the secretion of chloride by the rectal gland in which the negative intracellular potential facilitates the movement of chloride across the luminal cell membrane into the duct. Barium salts, by decreasing the membrane potential should inhibit the secretion of chloride by the rectal gland. In the experiments reported here barium chloride was added to the perfusate of isolated rectal glands to test this hypothesis.

Rectal glands were perfused as previously described (Silva et al., Am. J. Physiol. 233:F298, 1977), except that the perfusate was prepared without sulfate, to avoid precipitation of barium sulfate. Barium chloride was added to the perfusate at concentrations ranging from 10^{-4}M to 5×10^{-3}M. The results are summarized in Table 1. Barium inhibits chloride transport by the rectal gland in a dose dependent manner. No effect is seen at 10^{-4}M, the lowest concentration used, and inhibition was most marked (82%) at a concentration of 5×10^{-3}M. Since the latter was the highest concentration used it is not known whether that is the maximal inhibition achievable. The effects of barium on chloride secretion are completely reversible as shown in the last row of the Table where, upon return to a perfusate that does not contain barium, chloride secretion returned to normal. Electrical potential difference measured across the gland is also inhibited by barium in a dose dependent way. The reduction in the electrical potential across the gland correlates well with the reduction in chloride secretory rate as evidenced by a correlation coefficient of .84 $p < .05$. As was the case with chloride secretion, the electrical potential difference across the gland returned to normal when the barium was removed from the perfusate.

Table 1.--Effect of $BaCl_2$ on chloride secretion and electrical potential in the shark rectal gland

Perfusate $BaCl_2$ concentration M	Chloride secretion micro Eq/hr/g	PD* -mV
0	1567 ± 134 (10)	6.7 ± .9 (6)
10^{-4}	1297 ± 142 (6)	4.2 ± 1.0 (5)
5×10^{-4}	941 ± 77 (6)	2.7 ± 1.3 (4)
10^{-3}	906 ± 144 (6)	1.5 ± .4 (5)
2×10^{-3}	491 ± 74 (2)	NM**
5×10^{-3}	280 ± 25 (6)	1.0 ± .5 (4)
0	1487 ± 144 (10)	5.1 ± 1.1 (5)

Values are mean ± SEM; *Potential difference across the rectal gland is lumen negative
**NM: not measured.

The model for chloride secretion for the rectal gland predicts that a reduction in cellular potassium efflux produced by by barium should lower the potential difference across the luminal membrane, resulting in a reduction in the driving force for chloride across that membrane and a consequent reduction in the rate of chloride secretion and in the transglandular electrical potential. The positive correlation between the rate of chloride secretion and the transglandular electrical potential is consistent with this hypothesis. Although it is tempting to ascribe the effects of barium to a reduction in intracellular potential, this remains an unproven assumption since membrane potential was not measured. The possibility remains that barium has unappreciated effects on cellular metabolism that might be responsible for its effect on potassium efflux and ultimately, in the rectal gland, on chloride transport.

ION REQUIREMENTS FOR OXYGEN CONSUMPTION BY ISOLATED RECTAL GLAND CELLS

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Cells were isolated from rectal glands using the technique previously described (MDIBL Bull. 20:38-39, 1980), with only minor modifications. The cells were kept on ice until used in a buffer of the following composition (in mM): Na 280; K 5: Cl 295; Mg 3; Ca 2.5; SO_4 0.5 ; phosphate 1; Urea 350; Hepes 40, pH 7.6. The same buffer was used for measuring oxygen consumption. When sodium was removed from the bath it was replaced by lithium. When chloride was removed it was replaced by nitrate. When both sodium and chloride were removed they were replaced by lithium nitrate. Cells that were stored in buffer containing sodium and chloride were washed twice in buffer of the same composition as that used for measuring oxygen consumption. Oxygen consumption was measured in a constant temperature (25°C) chamber using a Clark type polarographic oxygen electrode (YSI) connected to a recorder. The electrode was calibrated using the known solubility of oxygen at 25°C and the barometric pressure. The rate of oxygen consumption was calculated from the tangent of the recorded slope of the oxygen consumption, the volume of buffer in the measuring chamber, and the wet weight of a measured aliquot of the cells removed from the measuring chamber at the end of the experiment. Glucose 5×10^{-3}M, pyruvate 10^{-2}M, and acetate 2.5×10^{-3}M were used as metabolic substrates. Theophylline 2×10^{-3}M, dibutyryl cyclic AMP 2×10^{-3}M, ouabain 10^{-4}M, and bumetanide 3×10^{-5}M (final concentrations), were all added in a volume not exceeding 1% of the volume of