

data allow prediction of the brain space which ^{22}Na would occupy in all brain slices (cpm/mg telencephalon \pm cpm/ μl EDF) were the EDF to supply all of the sodium gained by the brain during this stress. As seen in Table 1,

Penetration of ^{22}Na and ^{125}I -HSA From EDF Into Skate Brain

Experiment	Isotope	Slice#	Tissue : EDF distribution ratio, cpm/mg tissue : cpm/ μl EDF									
			1	2	3	4	5	6	7	8	9	10
Control	^{22}Na		.023	.011	.007	.007	.005	.004	.004	.003	.003	.002
Hypertonic	^{22}Na		.026	.018	.017	.016	.012	.017	.021	.028	.026	.016
Control + Hypertonic	^{125}I -HSA		.006	.003	.003	.002	.001	.002	.002	.003	.004	.004
Predicted Value of ^{22}Na space			.095	.095	.095	.095	.095	.095	.095	.095	.095	.095

All values are means of five to seven animals. Brain slices are numbered dorsal to ventral.

Although ^{22}Na values are consistently higher in hypertonic skates, differences are insignificant with the exception of the 10th slice due to the large SD in the hypertonic group.

the predicted distribution ratio (0.095) is 4-20 times greater than that measured in any brain slice under hypertonic conditions. There appears to be a tendency for skates exposed to hypernatremia to have more ^{22}Na penetration into the brain than controls, but differences between the two groups were not significant in all but one slice due to large standard deviations, particularly in the hypernatremic data. For RISA, penetration into the brain was identical in control and hypernatremic skates and, for the purposes of Table 1, the values from both RISA groups have been averaged. The results of these experiments clearly show that EDF is not the major source of the sodium taken up by the brain during hypernatremia. Supported by PHS NS 11050.

CHLORIDE EFFLUXES FROM THE PERFUSED DOGFISH HEAD

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Our recent studies with the intact pup of Squalus acanthias have indicated that the rectal gland plays an important but not unique role in NaCl extrusion. This conclusion is prompted by our finding that blood Na and Cl concentrations rise, after removal of the rectal gland, at rates significantly below those which can be accounted for by loss of this salt extrusion mechanism (Evans & Mansburger, Bull. MDIBL 19:101-103, 1979; Evans et al., J. Exp. Biol. in press, 1982). Similar results have been reported for the same species by Burger (Physiol. Zool. 38:191-196, 1965) and Forrest et al (Bull. MDIBL 13:41-42, 1973), for the lip shark, Hemiscyllium plagiosum (Chan et al., Comp. Biochem. Physiol. 23:185-198, 1967), and for the striped dogfish, Paroderma africanum (Haywood, J. Exp. Zool., 193:167-176, 1973). It has therefore been proposed that elasmobranchs may excrete salts via branchial transport mechanisms (Evans, Am. J. Physiol. 238:R224-R230, 1980). We therefore decided to initiate studies of the mechanisms of transport of Cl across the dogfish gill epithelium utilizing the perfused pup head which we recently described (Evans and Claiborne, Bull. MDIBL 20:9-11, 1981; Evans and Claiborne, J. Exp. Biol., in press, 1983).

The perfused pup head was prepared as described previously (Evans & Claiborne, *ibid*) except that in some experiments the plastic collar was not utilized and the head was suspended by plastic strips so that irrigation fluid drained into another container. No significant differences were seen in the pressure/flow or flux characteristics of heads suspended in the two different manners. Heads were perfused at approximately 750 $\mu\text{l}/\text{min}$ (resulting in afferent pressures of 15 to 30 torr), and irrigated at 40 ml/min. Cl effluxes were measured by adding 3-5 μCi of ^{36}Cl to the recirculated perfusate (10-20 mis), and removing 1 ml samples of the irrigation bath (50 - 100 mis at various times thereafter and measuring the radioactivity via liquid scintillation counting. Drugs (ouabain or furosemide) were added directly to the perfusate after a 20 minute control period. Thus, each perfused head served as its own control. All data are expressed as mean \pm s.e. (N).

The efflux of Cl from the perfused pup head was $200 \pm 45 \mu\text{M}$, $100\text{g}^{-1}\cdot\text{hr}^{-1}$ (17), which is not significantly different from that described for the intact pup ($219 \mu\text{M}\cdot 100\text{g}^{-1}\cdot\text{hr}^{-1}$, Evans et al., J. Exp. Biol., Op Cit., 1982). However, approximately 25% of the Cl efflux from the intact pup is via the rectal gland (Evans et al., *ibid*), so it appears that, while the Cl efflux from the perfused head approximates that from the intact fish, some 25% of this efflux is possibly from a mechanical leak. (We have recently monitored ^{14}C urea effluxes from perfused heads--the urea efflux from intact elasmobranchs is extremely low (Goldstein et al., Am. J. Physiol. 215:1493-1497, 1968)-- and found that extremely small mechanical leaks (approximately $3 \mu\text{l}/\text{min}$) are sometimes present and can account for approximately 25% of the Cl efflux.

Addition of ouabain (10^{-4}M) to the perfusate increased the efferent pressure slightly ($4.7 \pm 0.56 \text{ torr}$, $N = 3$), and stimulated the rate of Cl efflux by 222%, 329%, and 18% in three experiments. Addition of furosemide (10^{-4}M) to the perfusate reduced the afferent pressure slightly ($3.7 \pm 0.41 \text{ torr}$, $N=3$), but did not alter the rate of Cl efflux (control: $225 \pm 57 \mu\text{M}\cdot 100\text{g}^{-1}\cdot\text{hr}^{-1}$, furosemide: $221 \pm 122 \mu\text{M}\cdot 100\text{g}^{-1}\cdot\text{hr}^{-1}$, $n=5$). Unfortunately, we did not monitor urea fluxes in three experiments, so it is possible that slight changes in any structural leak (which even slight pressure changes could theoretically produce) could have masked any inhibition of Cl efflux produced by either ouabain or furosemide. Nevertheless these preliminary data are consistent with the proposition that Cl efflux from the perfused pup head is in the same range as that found *in vivo*, and is apparently not via a furosemide sensitive Na-Cl co-transport systems, which is secondarily sensitive to the inhibition of Na-K ATPase by ouabain (Fizzzell et al., Am. J. Physiol., 236:F1-F8, 1979). Further studies which will include measurement of the structural leak pathway by monitoring urea effluxes will enable us to separate passive, carrier-mediated, and structural leak pathways for Cl efflux across the gill epithelium of S. acanthias. This work was supported by NSF PCM 81-04046 to DHE.

PRELIMINARY INVESTIGATION OF THE ADRENERGIC CONTROL OF GILL HEMODYNAMICS IN THE SMOOTH DOGFISH, Mustelus canis

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Last year we examined the viability and hemodynamics of the perfused head of pups of Squalus acanthias (the spiny dogfish). We found that that preparation displayed long-term hemodynamic integrity and that addition of epinephrine (10^{-5}M) was followed by a significant reduction in gill resistance, which was inhibited by the concomitant addition of the beta-adrenergic receptor blocker propranolol. In some experiments this fall in gill resistance was preceded by a transient increase in gill resistance, which could be inhibited by the alpha-adrenergic blocker phentolamine (Evans and Claiborne, Bull. MDIBL 21:9-11, 1981). In addition, epinephrine stimulated the preferential flow of perfusate into the dorsal aorta, and this effect was blocked by propranolol. Thus, it appears that the branchial hemodynamics of the perfused S. acanthias pup head are similar to those described for a number of teleosts, with the exception that, in teleosts, the preferential flow into the dorsal aorta is mediated via alpha-adrenergic, rather than beta-adrenergic receptors (Claiborne and Evans, J. Comp. Physiol., 138:79-85, 1980, for relevant citations). A single pregnant female of the viviparous, smooth dogfish, Mustelus canis was caught this summer and we were able to perfuse the heads of the six pups (total body weights of from 103 to 122 g) which were removed from the sacrificed female. These experiments provided us with some comparative data to determine if the differences between the adrenergic control of gill hemodynamics in S. acanthias and teleosts was the result of species differences, or differences between teleosts and elasmobranchs. In addition, we wanted to determine if heads from pups from other available species could be routinely perfused and utilized for planned studies on elasmobranch gill solute transport (Evans et al., this volume).

The heads were perfused with elasmobranch Ringer's solution (Forster et al., Comp. Biochem. Physiol. 42A:3-13, 1972) using the perfusion and irrigation circuitry as described previously for the Squalus pups (Evans and Claiborne, 1981, *op. cit.*). The only major changes were the use of PE 90 for the entire cannula into the conus arteriosus, and the use of