THE ROLE OF THE KIDNEY IN ACID-BASE REGULATION IN THE LONG-HORNED SCULPIN (MYOXOCEPHALUS OCTODECIMSPINOSUS) DURING EXPOSURE TO LOW SALINITIES

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In teleost fish, the gills are normally considered to be the main site of acid-base regulation, while the possible role of the kidney has often been neglected (Heisler, in "Environmental Physiology of Fishes", eds. M.A. Ali pp. 123-162, 1980). Though Hodler et al. (Am. J. Physiol., 183,155-162, 1955) suggested that the pH of urine in marine teleosts is fixed at 5.8, Hickman and Trump (in "Fish Physiology", eds. W.S. Hoar & D.J. Randall, Vol. I, pp. 91-239,1969) demonstrated that urine pH (in the southern flounder) can range from 5.68 to 8.24. More recent experiments have reported a significant capacity for renal acid-base excretion in freshwater trout following acid or base infusions, or hypoxia (Kobayashi & Wood, J. Exp. Biol. 84, 227-244, 1980) with the renal response accounting for 16 to 18% of the total measured transfers (in the catfish; Cameron & Kormanik, J. Exp. Biol., 99, 143-160, 1982). In contrast, little is known about the role of the seawater teleost kidney in acid-base regulation. Our previous study showed that the long-horn sculpin, a marine teleost, is unable to maintain normal acid-base transfers when exposed to lower salinities, presumably secondary to impaired transbranchial H^+ and/or HCO_3^- movements (Walton & Claiborne, Bull. MDIBL 27,4-5, 1987). The present investigation was undertaken to measure the contribution of the kidneys to acid-base and ion balance while sculpin are in normal seawater or are exposed to a very dilute environment.

Long-horned sculpin (Myoxocephalus octodecimspinosus; 205-444 g, N=4) were anesthetized (MS-222 1:10,000) and the opening of the urinary papillae was catheterized with PE-50. The end of the catheter was perforated pushed well within the bladder (2-4 cm) before the papillae of the fish were ligated. Fish were placed in darkened plexiglass experimental chambers, supplied with running seawater, and allowed to recover for 12 to 24 hours. The external end of each catheter was placed in a pre-weighed vial attached to the outside of the experimental chamber. Each vial was pre-treated with 10 µl of streptomycin solution (0.25 g/l) to inhibit bacterial growth (Calla, J. Gen. Physiol. 69, 537-555, 1977). Urine was then collected during a 12-24 hour seawater period, a 24 hour dilution period (~20 mM seawater; Walton & Claiborne, ibid.), and another 12-24 hours in normal seawater. Throughout the experiment, collection vials were periodically changed, weighed, and the urine was then decanted for analysis of pH, $[Na^+]$, $[Cl^-]$, and $[NH_4^+]$. A portion of the urine (0.5-1.0 ml) was titrated with 0.1N NaOH to a pH of 7.80 for a measure of titratable acidity (as modified from Cameron & Kormanik, ibid.).

Urine pH values ranged from 6.21 to 7.61 in seawater, 6.50 to 7.04 in 20 mM seawater, and 6.33 to 7.21 after the fish were returned to seawater once again. Urine pH, flow rate, Na^+, Cl^-, NH_4^+ and H^+ excretion between all three exposure periods were not significantly different ($p>0.2$). Whole body efflux of Na^+ from the sculpin in seawater is approximately 15 mmol kg^{-1} hr^{-1} (Claiborne and Evans,

Mar. Biol. Lett. 2, 123-130, 1981). In the present study, the average rate of Na^+ efflux in the urine over the three periods was 0.03 ± 0.01 mmol kg^{-1} hr^{-1} (n=9, measured in 4 animals) a value only 0.2% of the whole body (presumably branchial) efflux. Renal Cl^- and NH_4^+ efflux also proved to be less than 1% of the gill efflux for these ions.

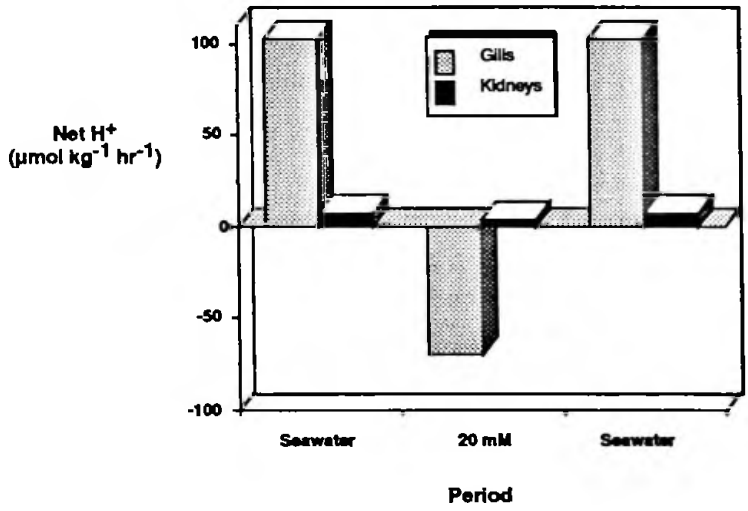

Fig 1. Renal and branchial (calculated from Walton & Claiborne, op cit.) net H^+ excretion by the sculpin in seawater and dilute 20 mM seawater. Negative H^+ values indicate an acid uptake or HCO_3^- loss.

Net whole body H^+ excretion in the seawater sculpin is 110 µmol kg^{-1} hr^{-1} and is reversed to an uptake (or loss of HCO_3^-) of -65 µmol kg^{-1} hr^{-1} when fish are exposed to 20 mM seawater (Walton & Claiborne ibid.). The net H^+ excretion in the urine measured under these same conditions was 7.8 and 4.0 µmol kg^{-1} hr^{-1}, respectively (Fig. 1). Thus, renal H^+ loss in seawater made up ~7% of the measured whole body excretion. When branchial H^+ transfers were reversed in low salinities, the continued renal H^+ efflux ameliorated the net H^+ load by ~6%. Therefore, the contribution of the kidney to pH regulation in these animals is less than that measured in freshwater species (Cameron & Kormanik,op. cit.), but still may assist the animal in times of acid-base imbalance. In contrast, renal excretion of Na^+, and Cl^-, and NH_4^+ is insignificant when compared to branchial transfers of these ions. While a more complete analysis of the renal contribution is required, it is intriguing that urine H^+ excretion appears to remain relatively constant during an acid stress in these animals. It remains to be seen whether the ratio of renal to branchial partitioning is retained when fish are exposed to other acid-base perturbations. (Funded by NSF DCM 86-02905 to JBC).