ACID-BASE TRANSFERS IN THE LONG-HORNED SCULPIN (MYOXOCEPHALUS OCTODECIMSPINOSUS) FOLLOWING ACID INFUSION: EFFECT OF LOW EXTERNAL CHLORIDE

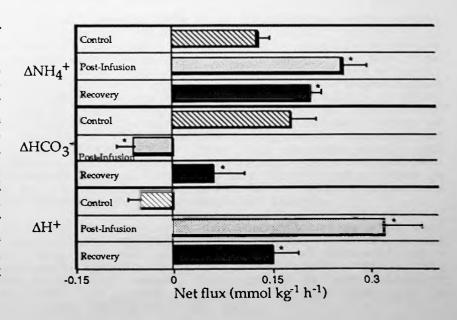
James B. Claiborne¹ and Shenna Bellows²
¹Department of Biology, Georgia Southern University, Statesboro, GA 30460
²The Mount Desert Island Biological Laboratory, Salsbury Cove, ME 04672

Long horned sculpin (Myoxocephalus octodecimspinosus) can excrete an infused acid load over 12-24 h when in seawater or dilute (20%) seawater, but the excretion is impaired when external [Na+] is reduced (Claiborne, Perry, & Bellows, Bull. MDIBL 32:95-97, 1993). Surprisingly, net HCO₃- loss to the water increased during the low Na+ period. We have hypothesized that both presumed acid-base transfers (Na+/H+, and Cl-/HCO₃- exchange; Evans, in "Fish Physiology", eds. W. S. Hoar & D. J. Randall, Vol Xb, pp. 239-283, 1984) were active in the sculpin gill and that Cl-/HCO₃- exchange was revealed only when Na+/H+ transfer was inhibited by low external [Na+] (Claiborne et al., 1993, ibid.; Claiborne & Perry, Bull. MDIBL 30:107-108, 1991). To investigate the role of potential Cl-/HCO₃- transfer mechanisms in these fish, we have studied acid-base transfers in animals exposed to low external [Cl-] (<1% of normal seawater) following acid infusion (2 meq kg-1 HCl).

Long-horned sculpin (Myoxocephalus octodecimspinosus) were pre-adapted for 7-13 days in 20% seawater and then cannulated and placed in experimental chambers according to the methods of Claiborne, Walton & Compton-McCullough (J. Exp. Biol., in press, 1994) and Claiborne & Perry (Bull. MDIBL 31:54-56, 1992). The infusion protocol followed the methods described by Claiborne et al.(1993, ibid.) except that water Cl^- rather than Na^+ was substituted with 100 mM sodium isethionate (final [Cl-]: 1.5 ± 0.6 mM, mean \pm S.E., n=5).

Figure 1 shows the transfer rates of NH₄+, HCO₃-, and H+ between the fish and the ambient water measured over the pre-infusion, post-infusion (low Cl⁻; 4 h), and recovery (9 h) periods. ΔNH₄+ increased both during and after the low Cl⁻ period. ΔHCO₃- loss was reversed to an uptake during the low Cl⁻ period and then returned to a reduced excretion once again during

Figure 1. Transfer rates of NH₄+, HCO₃-, and H+ (mmol kg-1 h-1) before (Pre-infusion;), immediately after (Post-infusion), and then 13 h after (Recovery) an acid infusion in fish which were pre-adapted to 20% (~100 mM NaCl) seawater. During the Post-infusion period the fish were exposed to 100 mM sodium isethionate (low Cl-). A positive transfer rate indicates a net efflux from the animal. mean \pm s.E., n=5; *: flux is significantly different from the Pre-infusion value.



the recovery. The sum of these transfers resulted in a large positive ΔH^+ (excretion) of 0.32 \pm 0.06 mmol kg⁻¹ hr⁻¹ during the post-infusion period. When the external Cl⁻ was restored in the recovery period, H⁺ excretion continued. Thus, during the 4 h low Cl⁻ exposure, the animals exhibited a net (post-infusion minus control) H⁺ excretion of ~1.5 meq kg⁻¹ which amounted to ~75% of the infused load. When ambient Cl⁻ was again available, the rate of net H⁺ loss was reduced, but an additional 1.8 meq kg⁻¹ of acid was still lost during the subsequent 9 h recovery period.

Interestingly, the rate of net H⁺ loss during the Post-infusion period (~100 mM Na⁺, 1.5 mM external Cl⁻) was >50% higher than that measured in animals which were allowed to remain in 20% seawater (~100 mM NaCl; Claiborne et al., 1993, ibid.) and even 15% higher than in seawater adapted animals (Claiborne & Perry, 1992, ibid.). Thus, a reduction in available ambient Cl⁻ increased the ability of the sculpin to excrete the administered acid load. These data support our hypothesis that a transbranchial Cl⁻/HCO₃⁻ exchange (Cl⁻ influx for HCO₃⁻ efflux) is operating in opposition to a gill Na⁺/H⁺ transfer (Na⁺ influx for H⁺ efflux; Claiborne et al., 1993, ibid.) and the loss of HCO₃⁻ continues even in the face of an internal acidosis as long as significant external Cl⁻ is available. It is likely that these transfers are responsible for the rapid and sometimes lethal loss of HCO₃⁻ to the water observed in this species as it is exposed to dilutions greater than 20% seawater (Claiborne, Walton & Compton-McCullough, 1994, ibid.). It remains to be seen whether these transfers can be regulated by the animal, or if the persistent base loss is an important limitation to true euryhaline adaptation in the sculpin.

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