ACID-BASE REGULATION IN LONG-HORNED SCULPIN (Myoxocephalus octodecimspinosus) DURING EXPOSURE TO LOW SALINITIES

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To survive the transition from seawater to fresh water, a fish must be able to make a number of osmoregulatory adjustments. Clearly, many euryhaline species are capable of adapting to lower salinities (Evans, in "Fish Physiology", eds. W.S. Hoar & D.J. Randall, Vol. Xb, pp. 239-283, 1984). To permit habitation of a more dilute environment, transbranchial Na^+/NH_4^+, Na^+/H^+, and Cl^-/HCO_3^- exchanges (thought to be utilized by these animals) must be modified. Since these exchanges affect both salt and acid-base movements, the need to alter ion transfers may also perturbate acid-base regulation. In a previous report (Claiborne & Evans, Bull. MDIBL 25:32-34, 1985), we showed that long-horned sculpin (Myoxocephalus octodecimspinosus) cannot withstand drastic changes in water salinity. Though these animals were able to survive in 8% seawater for up to 24 hours, we noted a large apparent efflux of HCO_3^- from the animal to the water. It was proposed that this acid-base transfer was responsible for the limitations placed on some species when entering brackish or fresh water. To further quantify this phenomenon, we have examined the role of acid-base regulation in long-horned sculpin during long-term exposure to dilute water salinities.

Sculpin were cannulated (Claiborne & Evans, Bull. MDIBL 24:24-25, 1984) and placed in darkened plexiglas experimental chambers that were supplied with running seawater. A recovery period of 8-12 hours was allowed before each chamber was closed and continuous aeration was begun. After a 10 hour control period, animals either remained in seawater (control group; ~480 mM) or were exposed to a 20-60 hour period in tap-water diluted seawater of one of the following salinities: 160 mM, 40 mM, or 20 mM (measured as Cl^- concentration). Blood samples were collected through the aortic cannula and analyzed for $[Cl^-]$, pH and T_{CO_2}. From these values, plasma P_{CO_2} and $[HCO_3^-]$ were calculated according to the methods of Boutilier et. al. (in "Fish Physiology", eds. W.S. Hoar & D.J. Randall, Vol. Xa, pp. 401-426, 1984). Water samples were collected periodically and were analyzed for ΔNH_4^+, ΔHCO_3^-, and net ΔH^+ transfers between the fish and the surrounding water (see Claiborne & Evans, Ibid., 1985).

While fish exposed to the 160 mM dilution lived for the length of the experiment (up to 10 days), the 40 and 20 mM groups exhibited significant mortality within 24-48 hours. Plasma $[Cl^-]$ (control: 160 mM $\pm$ 2.25, mean $\pm$ S.E., n=7) decreased significantly over time in the 40 and 20 mM dilution groups with the maximum decrease observed in the 20 mM fish as $[Cl^-]$ fell to 104.6 $\pm$ 3.84, n=3, at hour 24. Preliminary data from several animals (n=3) indicate that plasma $[Na^+]$ decreased in a similar manner. Plasma $[HCO_3^-]$ did not change significantly for any group (control = 4.56 $\pm$ 0.62, n=7) and plasma pH values showed little change from the control value of 7.75 $\pm$ 0.03 (n=7). Analysis of water samples revealed a moderate increase in ΔNH_4^+ excretion with each water dilution. The maximum increase was noted in the 20 mM group at hour 20, as ΔNH_4 loss was 1.38 mM/kg higher than the respective control animals. A large concurrent increase in the amount of HCO_3^- excreted from the fish was also recorded. ΔHCO_3^- loss was again directly related to the external dilution (eg., after 20 hours, ΔHCO_3^- control: 1.73 $\pm$ 0.23, 160

mM: 2.06, 40 mM: 3.24 ± 0.69, 20 mM: 6.48 ± 0.62). On examination of net ΔH^+ (calculated as the difference between ΔNH_4^+ and ΔHCO_3^-) it becomes evident that animals are gaining H^+ from (or losing HCO_3^- to) the dilute water, and the rate of this net base loss is proportional to the decrease in water salinity (Fig. 1).

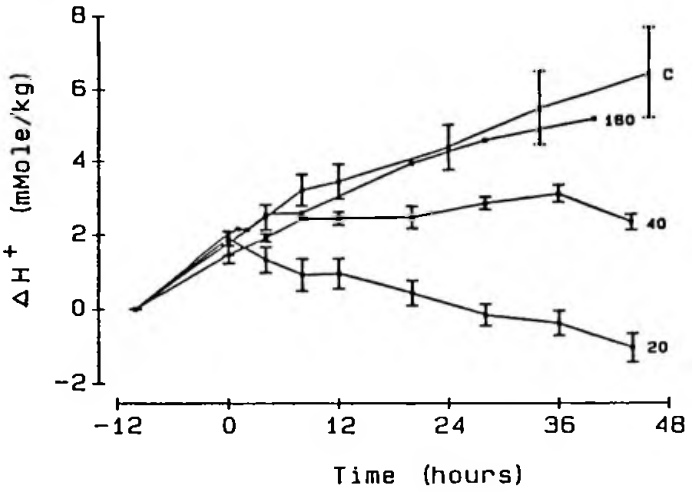

Fig 1. Cumulative ΔH^+ transfers by sculpin during exposures to various water dilutions (mean ± S.E.; C: Control; 160, 40, 20: water $[Cl^-]$ of dilution groups).

Since teleosts are restricted to an aqueous respiratory media, they cannot freely modify plasma P_{CO_2}. Thus, the adjustment of $[HCO_3^-]$ becomes the predominant acid-base regulatory mechanism in these animals (Heisler, in "Fish Physiology", eds. W.S. Hoar & D.J. Randall, Vol. Xa, p. 315-401, 1984). It is intriguing that the net base loss observed in the sculpin exposed to dilute water, had no observable effect on blood acid-base balance. One interpretation of these data is that HCO_3^- is leaving intracellular or bone compartments, transiently passing through the extracellular space, and finally being lost to the water. While a fraction of the observed decrease in plasma $[Cl^-]$ could have been due to exchange with intracellular HCO_3^- (as in erythrocytic HCO_3^-/Cl^- exchange; Obaid & Crandall, J. Membrane Biol., 50, 23-41, 1979), the majority was most likely lost diffusively to the dilute environment along with Na^+. Determination of NaCl transfers between the fish and the external water may provide essential information in identifying the mechanisms involved. Likewise, the measurement of intracellular pH during exposure to low salinities might indicate if intracellular movements of H^+ or HCO_3^- are responsible for the observed acid-base transfers. It remains to be seen whether loss of acid-base balance is the cause of the final expiration of the fish, or if this is just a secondary effect linked to the diffusive loss of NaCl. (Funded by NSF DCM 86-02905 to JBC).