FURTHER STUDIES ON AMMONIA EXCRETION IN A MARINE TELEOST (MYOXOCEPHALUS OCTODECIMSPINOSUS)

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In a previous study, we examined the effects of NH_4Cl infusion on the acid-base balance of the long-horned sculpin (Claiborne & Evans, Bull. MDIBL 24:24-25, 1984). NH_4Cl induced a plasma acidosis which was due to a rapid transfer of NH_3 to the surrounding water while the remaining protons were eliminated more slowly. It was likely that NH_3 made up a significant proportion of the total ammonia ($T_{amm} = NH_3 + NH_4^+$) excreted. To further test this proposition, we have duplicated the experimental protocol described previously (Claiborne & Evans, ibid., 1984), but in the present experiments, NH_4HCO_3 was utilized as the carrier for the ammonia load. Following each infusion, internal acid-base balance (plasma pH and Tco_2, from which Pco_2, and $[HCO_3^-]$ were calculated), plasma T_{amm}, and the net transfers of ammonia ($\dot{}NH_4^+$) and bicarbonate ($\dot{}HCO_3^-$) between the animal and the external water, were monitored over the succeeding 20 hours (see Claiborne and Evans, this volume, for details).

In each of the sculpin (170 ± 15 grams, n=5, mean ± S.E.) infusion of 5 mMole/kg NH_4HCO_3 elicited several changes in the measured acid-base parameters. Both plasma T_{amm} and Tco_2 increased rapidly after the infusion (plasma T_{amm} control: 0.24 ± 0.06 mM, 5 min. post-infusion: 4.62 ± 0.34 mM; Tco_2 control: 4.78 ± 0.24 mM, post infusion: 11.50 ± 0.41). T_{amm} returned to control values within 2 hours, while Tco_2 required 4 hours to regain normal levels. Blood pH varied only slightly (from 7.81 at hour 2 to 7.73 at hour 8) around the pre-infusion pH of 7.78 ± 0.01. Plasma Pco_2 increased immediately after the infusion (from 1.8 to 4.7 torr), and then slowly declined to control levels within 4 hours. Both $\dot{}NH_4^+$ and $\dot{}HCO_3^-$ were elevated subsequent to the infusion, and within 2 hours, ~75% of the respective NH_4^+ and HCO_3^- load had appeared in the external water. The rate of HCO_3^- excretion remained significantly above the control rate for up to 8 hours post-infusion.

The present data agree with our earlier findings (Claiborne & Evans, ibid., 1984). Plasma T_{amm} rapidly declined within several hours of the infusion (via an increased loss of T_{amm} into the water). T_{amm} was eliminated more rapidly from the extracellular space than H^+ and/or HCO_3^-. Had a portion of the total infused ammonia been excreted in the form of NH_4^+, a plasma alkalosis should have resulted (due to the remaining infused HCO_3^-). Indeed, had all T_{amm} been lost as NH_4^+, the measured post-infusion plasma $[HCO_3^-]$ of 11.25 mM in combination with the control plasma Pco_2 of 1.8 torr would have effected a serosal pH value of ~8.12 (calculated using solubility and pK' values derived from Boutilier et. al., in "Fish Physiology", eds. W.S. Hoar and D.J. Randall, Vol Xa, pp. 401-430, 1984). In contrast, if a majority of the T_{amm} transfer was due to the release of NH_3, the resulting equimolar augmentation of plasma $[H^+]$ and $[HCO_3^-]$ should have been reflected by an increase in plasma Pco_2. Since the measured post-infusion blood pH was only elevated slightly (0.03 units) from control values, and a ~2.5x increase in plasma Pco_2 was concurrently observed, we would again propose that this species of marine teleost may utilize NH_3 excretion to compensate for an elevation of internal T_{amm}. (Funded by a Faculty Research Grant from GSC to JBC and NSF PCM 83-02621 to DHE)