EFFECTS OF EXPOSURE TO SUB-LETHAL CONCENTRATIONS OF AMMONIA AND HYPOXIA ON THE SWIMMING PERFORMANCE OF BROWN TROUT (*SALMO TRUTTA*)

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Introduction

Ammonia is toxic to all vertebrates. It has become a pervasive pollutant of aquatic habitats but is also, for the majority of aquatic organisms, an end-product of protein metabolism. In teleost fish ammonia can, therefore, accumulate to toxic levels either as a consequence of exposure to elevated water ammonia concentrations or as a consequence of impaired excretion of the endogenous metabolite.

Ammonia accumulates in fish during exposure to sub-lethal concentrations of heavy metals such as copper and, in brown trout, this has been linked statistically to a decline in the ability to perform exercise (Beaumont et al., 1995). Beaumont et al. (1995) found a negative linear relationship between plasma ammonia concentrations and maximum sustainable swimming speed (U_{crit}) in brown trout exposed to a sublethal combination of copper and acidic water, and went on to demonstrate that the ammonia accumulation caused a partial depolarisation of muscle membrane potential (Beaumont et al., 2000). Shingles et al. (2001) demonstrated that exposure to elevated water ammonia alone was sufficient to reduce U_{crit} in rainbow trout (*Oncorhynchus mykiss*), with evidence that this was linked to a partial depolarisation of muscle.

Objectives of the current study included to investigate whether a linear relationship between plasma ammonia concentration and U_{crit} could be elicited in brown trout by ammonia exposure alone, and to investigate the mechanisms for impaired performance.

Hypoxia is a growing problem in many aquatic habitats and, therefore, can be expected to occur concurrently to elevated ammonia. Hypoxia impairs swimming performance in salmonids by limiting aerobic scope (Bushnell et al., 1984). Thus, a further objective of the current study was to determine how concurrent exposure to ammonia and hypoxia influenced exercise performance in brown trout.

Methods

To investigate the relationship between plasma ammonia concentration and swimming performance, adult brown trout (mean mass approx. 500g) were exposed to two different sub-lethal concentrations of NH_4Cl in the water, nominally 100 μmol l^{-1} and 200 μmol l^{-1}, for 24h in hard water at 15 °C and pH 8.2. Their swimming performance, and associated respirometry, were then investigated in a Brett-type swimming respirometer (Shingles et al., 2001), in comparison to controls in normal water. In parallel, the effects of the ammonia exposure regimes on plasma ammonia and on the membrane potentials (E_M) of red muscle, white muscle, heart and brain were investigated on trout with an indwelling catheter in the dorsal aorta.

To investigate the effects of ammonia and hypoxia, the experimental series were repeated, but trout were exposed to mild hypoxia at a nominal water O_2 partial pressure of 80 mmHg for 1h prior to, and then during, the various measurements.

Results

Exposure to either 100 μmol l^{-1} or 200 μmol l^{-1} NH_4Cl caused an increase in plasma total ammonia to 386±42 μmol l^{-1} or 771±92 μmol l^{-1}, respectively, compared with 133±29 μmol l^{-1} in control fish (mean ± SE, n = 6). This ammonia accumulation was associated with a significant decline in U_{crit} from 2.24±0.15 bodylengths s^{-1} (BL s^{-1}) in control trout to 1.46±0.09 BL s^{-1} or 1.08±0.16 BL s^{-1} in trout exposed to 100 μmol l^{-1} or 200 μmol l^{-1} NH_4Cl, revealing a direct negative relationship between plasma ammonia concentration and U_{crit} (Figure 1). The linear relationship was surprisingly similar to that observed by Beaumont et al. (1995) in brown trout exposed to sublethal copper at low pH, which is shown on the figure for comparison.

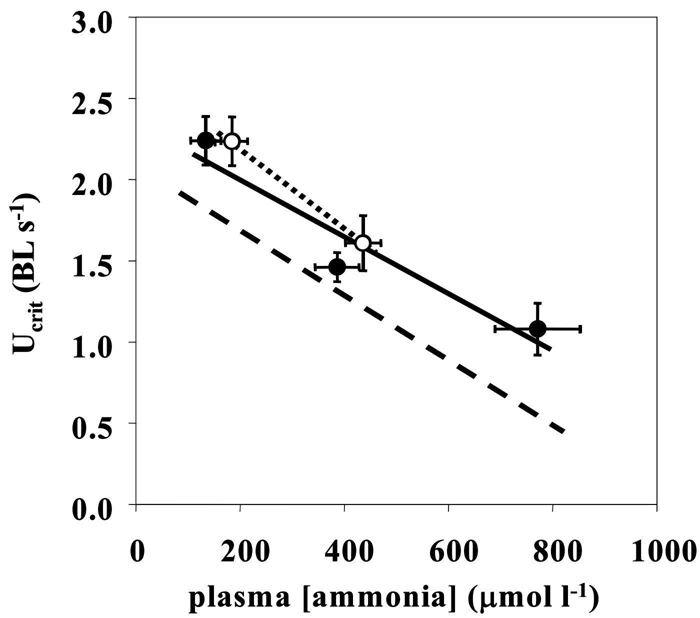

Figure 1. The least squares linear regression relationship between plasma ammonia concentration and maximum sustainable swimming speed (U_{crit}) in brown trout exposed to three water concentrations of ammonia (this study, solid symbols, solid line); as reported for brown trout that accumulate ammonia following exposure to a sub-lethal combination of copper and low pH (Beaumont et al., 1995, dashed line), and in rainbow trout exposed to two water concentrations of ammonia (plotted from data reported in Shingles et al. 2001, open symbols, dotted line). The error bars on the symbols are ± 1 SEM of the mean values for plasma ammonia and U_{crit}, n = 6 or 7. For brown trout exposed to ammonia (this study), the linear regression was described by the equation: mean U_{crit} = -0.0018(mean[ammonia]) + 2.347 (R^2 = 0.903, n = 3). Beaumont et al. (1995) reported an equation based on individual values rather than means: U_{crit} = -0.0020[ammonia] + 2.089 (R^2 = 0.670, n = 30). For rainbow trout (Shingles et al., 2001), the equation was: mean U_{crit} = -0.0024(mean[ammonia] + 2.674 (n = 2).

Also, the figure shows the relationship between plasma ammonia and U_{crit} in rainbow trout, replotted from the data reported in Shingles et al. (2001). The relationship between the plasma ammonia concentration and U_{crit} was very similar in the two species. Interestingly, however, a significantly higher water ammonia concentration was required in rainbow trout to elicit the same plasma ammonia accumulation (Shingles et al., 2001).

The respirometry measurements revealed that the impaired performance in the brown trout exposed to ammonia was associated with reduced swimming efficiency, and with a partial depolarisation of E_M in white muscle and the brain, although there were no significant effects on E_M of the red muscle and heart.

Exposure to hypoxia caused a 45% decline in U_{crit} in control animals, down to 1.23±0.09 BL s^{-1}, as a consequence of the expected limitation to aerobic scope. Hypoxia did not, however, cause the same proportional decline in performance in the ammonia-exposed fish; both groups had a U_{crit} of approximately 1 BL s^{-1}. Therefore, hypoxia had no further effect on the reduced performance of animals exposed to 200 μmol^{-1} NH_4Cl.

Conclusions

The results provide further evidence that the impaired swimming performance of trout following exposure to sub-lethal concentrations of copper in acid water (Beaumont et al., 1995) can be attributed to the accumulation of ammonia. Ammonia accumulation has similar effects on performance in both rainbow and brown trout, but rainbow trout appear better able to limit plasma ammonia accumulation during exposure to elevated water ammonia. The fact that hypoxia did not elicit any further decline in U_{crit} in trout exposed to NH_4Cl indicates that ammonia impairs performance by a mechanism unrelated to oxygen supply in brown trout, perhaps through effects on nerve and white muscle function.

References

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