Cardiovascular effects of NO and Urotensin II in longhorn sculpin, *Myoxocephalus octodecimspinosus*

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Although both NO and Urotensin II are vasoactive in isolated teleost blood vessels, it is not clear what role these substances may play in gill hemodynamics[1]. To delineate more clearly specific cardiac vs. gill an/or systemic resistance effects, we have utilized the afferent and efferent branchial artery-cannulated sculpin preparation[4] to examine the effect of ventral aortic infusion of either sodium nitroprusside (SNP, NO donor; 10 μM) or Urotensin II (UTII; 0.1 μM). These nominal plasma concentrations were chosen because they had produced vascular tension changes in published studies.

Longhorn sculpin were prepared as described previously[4] and either SNP or UTII (in teleost Ringers) was infused into the afferent branchial cannula after an initial, control Ringers infusion of the same volume as a control. In both cases, an equivalent volume of blood was removed before the infusion to avoid volume effects. Measurements of cardiac output (CO) and ventral and dorsal aortic pressure (PVA, PDA), and calculations of gill resistance ((PVA-PDA)/CO) were made as described previously[3].

SNP produced no change in CO but it did stimulate bimodal alteration in both PVA and PDA, with a significant (and equivalent: ca. 30%) decline ($p = 0.01$ and $p = 0.04$, respectively) in both parameters ($N = 4$) within 5 minutes, followed by return to control levels ($p > 0.50$) within another 10 minutes. In contrast, UTII produced a significant (and equivalent; ca. 12%) increase ($p = 0.1$ and $p = 0.049$) in both parameters ($N = 3$-5) within 5 minutes, followed by a return to control levels ($p > 0.25$) within another 10 minutes. UTII did not alter CO. Since neither CO nor PVA-PDA was altered by either compound, these preliminary data suggest that both NO and UTII can produce significant vasoactive responses in the longhorn sculpin, but they appear to be secondary to alterations in systemic rather than branchial resistances. The bimodal responses to both SNP and UTII suggest either neuroendocrine, homeostatic reflexes or possibly the presence of receptor-specific, vasoconstrictory vs. vasodilatory pathways, as have been described recently for UTII in mammalian systems[2,5]. (Supported by NSF IBN-0089943 to DHE).

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