The cardiovascular and branchial perfusion effects of endothelin in the longhorn sculpin (*Myoxocephalus octodecimspinous*)

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Endothelin is a potent vasoconstrictor of a variety of isolated fish vascular rings[2] and produces an increase in branchial resistance in the cod[4], which may be mediated by contraction of the lamellar pillar cells[5]. This study was undertaken to observe and quantify the cardiovascular and intra-gill perfusion effects of ET-1 and the endothelin agonist sarafotoxin S6c in the sculpin.

The procedure for preparation of the animals for study was described in detail previously[3]. An additional cannula not listed in that procedure was used to measure post-branchial (dorsal aortic) blood pressure. The last gill arch of each gill was used for either the pre or post-branchial cannula. Infusions of endothelin (ET-1), SRX S6c (an ET_B - specific agonist), or a control (saline) were done through the pre-branchial cannula. A volume of blood equal to the infusion volume (0.8% total blood volume) was removed prior to delivery of the agonist. The infusions were given in a stepwise manner of increasing cumulative concentration (control, 1, 10, 100, 316, 1000 pmol/kg). Measurements of cardiac output (CO), ventral aortic blood pressure (P_{VA}), and dorsal aortic blood pressure (P_{DA}) were made at time points including 1 minute pre injection and 1, 5, and 10 minutes post injection. These readings were similar to cardiovascular measurements made in other species[1].

CO and P_{DA} were not affected significantly by the infusion of either ET-1 or SRXS6c. The P_{VA} was significantly increased with the infusion of ET (mean 12.33 ± 3.72 cm H_2O increase with 1000 pmol/kg infusion, N = 6) but unaffected by SRX or the control. This increase in P_{VA} and unchanged CO and P_{DA} is evidence of an increased gill resistance, which was calculated by (VA-DA)/CO. The video clip (http://www.zoo.ufl.edu/dhefish/ETeffect.htm) demonstrates that ET shuts down flow in the afferent lamellar arteriole, but the site of action (pillar cells vs. afferemt filamental artery constriction) was not evident. Infusion of SRX did not affect blood flow in the filament, suggesting that the effective receptor is an ET_A-type. Further experiments are warranted, but it is clear that the increased gill resistance is secondary to blockage of blood flow in the filament. (Supported by NSF IBN-0089943 and an REU Supplement to DHE).

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