EFFECTS OF ACUTE AND CHRONIC HYPERCAPNIA ON HEART FUNCTION IN EELS

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Background and Aims

Eel reared intensively in systems with recirculating water can experience chronic hypercapnia. Metabolic CO_2 may accumulate, and water CO_2 partial pressures (pCO_2) may exceed 30 torr (Steffensen & Lomholt, 1990), far above what is considered physiologically normal in fish (Heisler, 1984).

Main aim of the present study was to evaluate the effects of high levels of environmental hypercapnia and consequent plasma acidosis on eel heart performance. Specific aims were: i) evaluation of the effect of hypercapnic acidosis on the *in vitro* heart function; ii) evaluation of the consequences of chronic exposure to constant or fluctuating levels of hypercapnia of eels, on *in vitro* heart performance.

Methods

Animals (*Anguilla anguilla*) were maintained at the Experimental Thermal Aquaculture Plant "La Casella" (Sarmato, PC, Italy) in tanks with a continuous supply of fresh biofiltered water at 23±1°C, both under normocapnic or controlled hypercapnic conditions. Hearts were perfused at 20°C according to

Houlihan et al. (1988); a saline modified from Davie et al. (1992) was used. The effects of hypercapnic acidosis on cardiac function were studied on hearts from normocapnic acclimated animals submitted to a stepwise increase of Pco_2 from 4 torr to 10, 20, 40 and 60 torr. Two different oxygen levels were used: oxygenated saline (Po_2 = 590 torr) and aerated saline (Po_2 = 150 torr that, under the perfusion conditions used, represented a hypoxic stimulus to the heart). At each step, cardiac output (CO), input and output pressures and oxygen consumption (Vo_2) were measured. Maximal volume loading (VL, i.e. the maximal increase in CO and power output, PO, via the Starling mechanism) and pressure loading (PL, with the consequent shift from volume displacement to pressure development) were determined.

The *in vitro* heart performance of eel chronically exposed to constant (three groups, 15 torr, 30 torr and 45 torr Pco_2, respectively) or fluctuating (two groups, 5-15 and 5-25 torr, respectively, daily oscillation) was studied. Perfusion saline was modified to match *in vivo* plasma levels of bicarbonate and pH. On each heart Starling Index (= the ratio between maximal power output obtained by volume loading and basal PO), cardiac scope (=maximal *in vitro* PO), and mechanical efficiency were evaluated. The effect of *in vitro* acute hypecapnia on hearts from acclimated animals was also tested.

Results and Discussion

Under oxygenated conditions, eel heart was able to maintain basal PO up to 60 torr Pco_2. At this level of hypercapnia a significant reduction in PO (consequent to the combination of both negative inotropic and chronotropic effects) was observed in the loaded hearts, more evident under pressure loading than under volume loading. Hypoxia strongly affected the sensitivity to hyercapnia of hearts, which were unable to maintain basal performance at Pco_2 values higher than 20 torr. This higher sensitivity was particularly evident under pressure loading, where PO was close to zero at 20 torr. Mechanical efficiency of hearts significantly reduced under hypercapnia (increase in the energetic cost of cardiac work). The above results suggest that the eel heart is inclined to produce volume work rather than pressure work.

Heart rate, which in the normocapnic acclimated animals is negatively affected by an *in vitro* (acute) hypercapnic insult, was not affected by *in vitro* hypercapnia in the animals chronically exposed to high levels of Pco_2, either constant or fluctuating. Also, intrinsic frequency under isocapnic conditions was not different among the acclimated groups and with respect to the controls.

Chronic exposure to hypercapnia did not affect the heart's ability to respond to volume loading when perfused under isocapnic conditions. Moreover, 30 and 5-25 torr acclimated animals displayed a higher Starling response than the other groups when perfused under hypercapnia. *In vitro* hearts mechanical efficiency was high in all eels exposed to chronic hypercapnia, when compared with the normocapnic animals. This suggests a lower effect of acute hypercapnia *in vitro* in the chronically exposed animals than in the normocapnic animals.

Conclusion

The results here reported indicate that the heart from normocapnic acclimated eels displays a particularly high tolerance to hypercapnic acidosis. However, under hypoxic conditions tolerance is strongly reduced. Chronic exposure of eels to constant hypercapnia significantly affects intrinsic heart performance. In particular, adaptations are observed in the intrinsic chronotropism (the typical bradycardic response to hypercapnia disappears) and in the ability to tolerate an acute hypercapnic insult. The latter is manifest in the ability to maintain high Starling response and mechanical efficiency.

References

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