

**ON-LINE MEASUREMENT OF VENOUS P<sub>O</sub><sub>2</sub>**  
**IN EXERCISING RAINBOW TROUT**

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**EXTENDED ABSTRACT ONLY – DO NOT CITE**

The vertebrate heart needs oxygen to survive. For fish such as trout and salmon, oxygen is delivered to the heart through two routes: a coronary circulation to the outer myocardium directly from the gills and a cardiac circulation which delivers partially deoxygenated venous blood to the inner myocardium of the heart (Davie and Farrell, 1991). Of these two circulations the oxygenation of cardiac tissue is more reliant on the venous blood returning to the heart. When fish exercise and due to increases in cardiac output and ventral aortic pressure, the oxygen needs of the heart increase. This, in association with increased oxygen extraction from the blood perfusing locomotory muscles could cause an imbalance in the supply and demand relationship at the heart, i.e. a compromise in the O<sub>2</sub> delivery to the heart when its demand is at its greatest.

Previously, examination of oxygen delivery to the heart has involved monitoring venous Po<sub>2</sub> via either extracorporeal circulation, which limits the locomotory capabilities of the fish, or blood sampling through a cannula, which does not allow for fine temporal resolution of Po<sub>2</sub> changes. In this study we utilized a fibre-optic micro-optode that once implanted in a blood vessel allowed us to monitor venous Po<sub>2</sub> on-line with a response time of < 3 s, with limited interference to the movement of the fish.

## Methods

Experiments were conducted at two acclimation temperatures, according to seasonal ground water temperature (spring, 6-10°C, N=6; summer, 13-15 °C, N=5). Rainbow trout (840 ± 122 g) were anesthetized and the optode was implanted into the ductus Cuvier. Venous Po<sub>2</sub> values are recorded every 1 s. Post-surgery fish were placed into a Brett-type respirometer for a 2-h recovery period that was followed by critical speed (U<sub>crit</sub>) test used to habituate the fish to the respirometer and testing protocol. Fish were then given an overnight recovery and a second U<sub>crit</sub> was performed (all results reported are for the second U<sub>crit</sub> test).

## Results

Venous Po<sub>2</sub> during swimming was very labile, as abrupt changes in venous Po<sub>2</sub> can be seen as a reaction to the fish struggling (Fig. 1). Along with these temporary changes in venous Po<sub>2</sub>, there was a general decrease associated with increased swimming speed. For cold-acclimated fish the threshold was reached prior to the fish quitting swimming (86-96 % of U<sub>crit</sub>) and even the most severe struggles rarely decreases venous Po<sub>2</sub> below the threshold. For warm-acclimated fish the venous Po<sub>2</sub> threshold at U<sub>crit</sub> was less apparent and was significantly higher at U<sub>crit</sub> (28.9 ± 3.5 torr) than the threshold for cold-acclimated fish (15.3 ± 3.7 torr) (Fig. 2); as well it is significantly higher for all points beyond 50% of U<sub>crit</sub>.

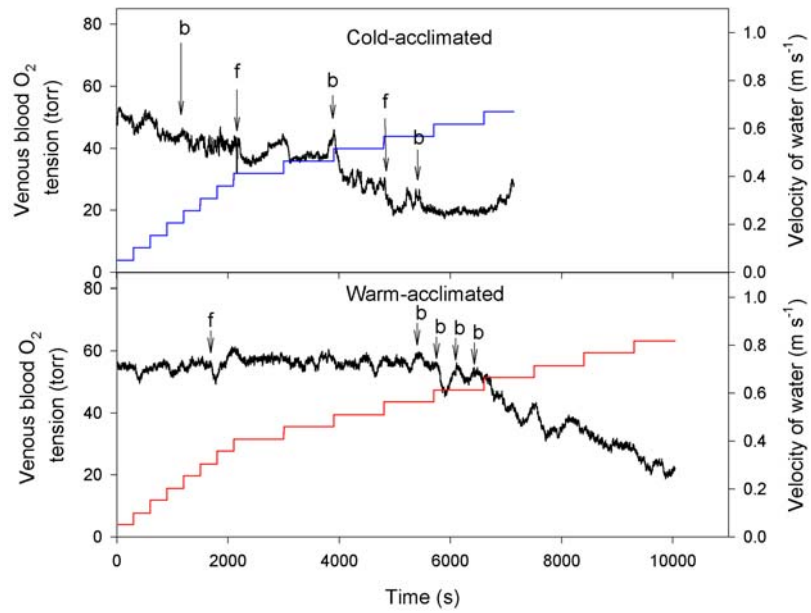


Fig. 1. Individual, on-line venous Po<sub>2</sub> during an U<sub>crit</sub> test. “b” and “f” represent bursting and fighting events during steady state swimming.

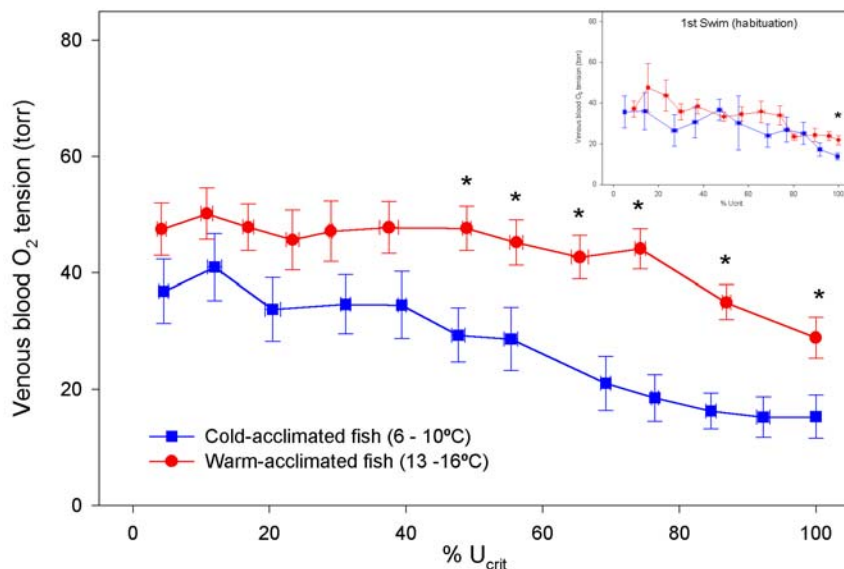


Fig. 2. Mean venous  $P_{O_2}$  for cold- and warm-acclimated fish, derived from periods of steady state swimming at each step of a critical speed test, expressed as a percentage of  $U_{crit}$ . \* represents significant differences between cold-acclimated and warm-acclimated fish ( $P < 0.05$ ). **Inset:** Results for the initial habituation swim test.

### Discussion and Conclusions

Oxygen delivery to the heart via the venous blood can be estimated using a hemoglobin-oxygen ( $Hb-O_2$ ) dissociation curve (Thomas et al., 1994),  $[Hb]$  of rainbow trout (Gallaughan et al., 1995) and cardiac output during exercise (Thorarensen et al., 1996), which is assumed to increase 2.5 fold. As venous  $P_{O_2}$  decreases to threshold from routine and titrates down the  $Hb-O_2$  curve, the increase in cardiac output will compensate for the decreased oxygen content in the blood allowing the oxygen delivery to the heart via venous blood to remain constant. An increase in temperature will shift the  $Hb-O_2$  curve to the right, therefore making it necessary for the venous  $P_{O_2}$  threshold to increase, which is in accordance with the findings of this study (Fig. 3).

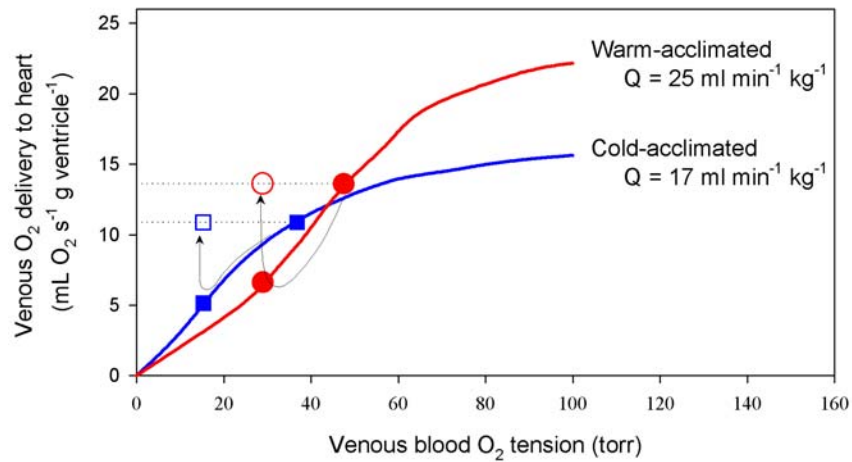


Fig. 3. Venous O<sub>2</sub> delivery curve to the heart using routine and exercise PVO<sub>2</sub> and Q.

### References

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