

**OXYGEN PARTIAL PRESSURES
IN THE RED MUSCLE OF RAINBOW TROUT**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Oxygen-sensitive optical chemical fibre sensors (Pre-Sens micro-optodes, Precision Sensing GmbH, Germany) were used to measure the partial pressures of O₂ (pO₂) in the red muscle of rainbow trout (*Oncorhynchus mykiss*) during exposure to hypoxia and exercise to fatigue.

Methods

Data were collected from six trout with a mean (\pm SD) mass of 697 \pm 152 g. The micro-optode was inserted into red muscle just dorsal to the lateral line, and the trout cannulated in the dorsal aorta, under anaesthesia (MS-222). Fish were recovered for approximately 42h in normoxic water while swimming at a speed equivalent to 0.5 bodylengths s⁻¹ (BL s⁻¹) in a Brett-type swimming respirometer. The trout were exposed to mild hypoxia, comprising 30 min at 100 mmHg followed by 30 min at 75 mmHg, then 1h recovery to normoxia. Two hours later, at complete recovery from hypoxia, fish were exposed to 0.5 BL s⁻¹ increments in swimming speed every 30 min until fatigue. Measurements included water pO₂; arterial blood pO₂, pH and total O₂ content; red muscle pO₂, and fish O₂ uptake. The position of the probe in the red muscle was confirmed post-mortem.

Results

In all fish, red muscle pO₂ was close to zero under anaesthesia, but gradually rose over a few hours during recovery and then stabilised. At a normoxic water

pO₂ of 138 mmHg, and mean (\pm SE) plasma pO₂ of 119 ± 5 mmHg, mean red muscle pO₂ was 61 ± 10 mmHg. Although red muscle pO₂ was relatively stable in normoxia, sharp reductions were observed if the animal struggled in the respirometer, followed by a gradual return to the previous pO₂. During exposure to hypoxia and recovery to normoxia, red muscle pO₂ showed close temporal sensitivity to changes in water and blood pO₂. In hypoxia, net changes in muscle pO₂ were less than those in the blood, such that the arterial blood to muscle pO₂ gradient declined as hypoxia deepened (Figure 1). Proportional changes in pO₂ were also less in the muscle than in the blood. Hypoxia had no effect on blood total O₂ content or fish O₂ uptake.

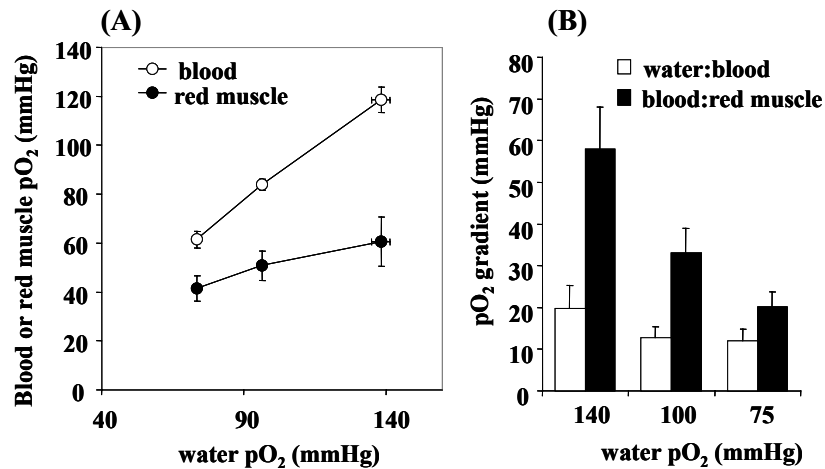


Figure 1. (A) Effects of mild hypoxia (30 min at a water pO₂ of 100 mmHg followed by 30 min at a pO₂ of 75 mmHg) on arterial blood and red muscle pO₂. (B) Gradients of pO₂ between water and arterial blood, or arterial blood and red muscle, in normoxia (pO₂ of 140 mmHg) and during exposure to mild hypoxia. Values are mean \pm 1 SEM of six measurements.

Incremental exercise caused an exponential increase in fish O₂ uptake, and a drastic decline in blood pO₂ and pH at fatigue. Blood total O₂ content was, however, maintained throughout exercise, despite the profound blood acidosis. There was no significant effect on red muscle pO₂, which remained stable throughout exercise, and during recovery of fish O₂ uptake and acid-base balance post-fatigue (Figure 2).

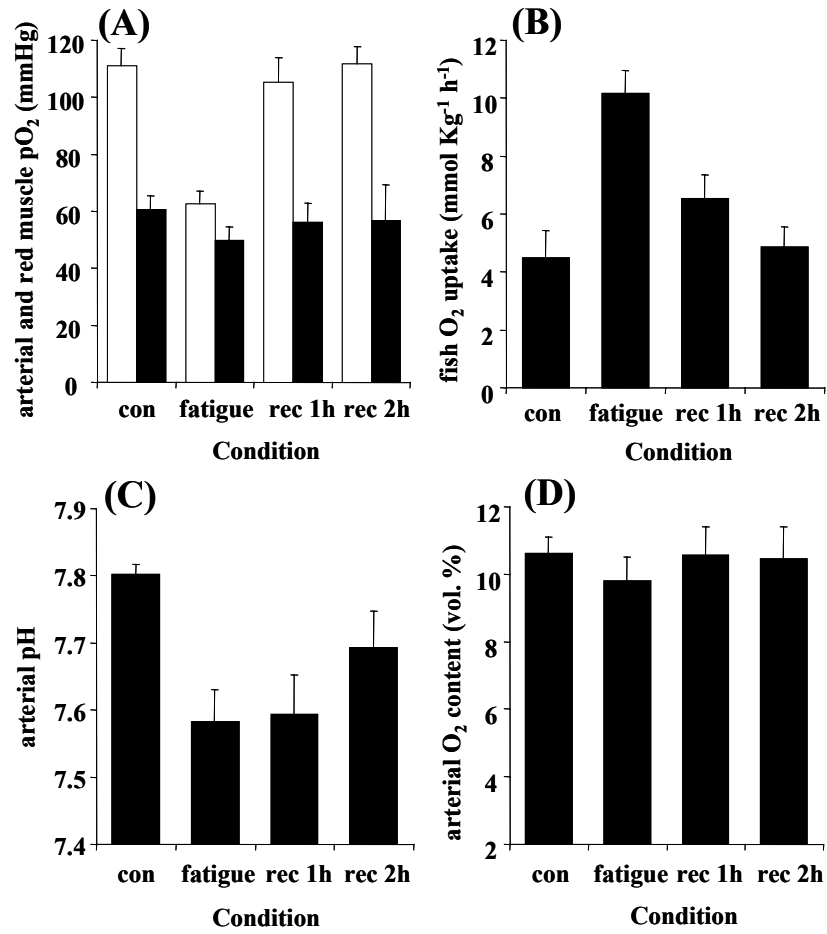


Figure 2. Effects of exercise to fatigue on (A) pO₂ of arterial blood (empty bars) and red muscle (closed bars); (B) mean rates of fish O₂ uptake; (C) arterial pH, and (D) arterial total O₂ content. Values are mean ± 1 SEM of six measurements. con, control pre-exercise (i.e. swimming at 0.5 BL s⁻¹); fatigue, measurement at fatigue; rec 1h and rec 2h, at one hour or two hours recovery from exercise to fatigue, respectively.

Conclusions

Muscle pO_2 can be very low under extreme conditions (MS-222 anaesthesia with air-exposure) and recovery of muscle homeostasis seems quite slow after surgery (~20 hours). Once recovery is achieved, muscle pO_2 can change quite rapidly when the fish struggles. The data indicate that red muscle pO_2 is defended when blood pO_2 declines during mild hypoxia. Red muscle pO_2 is regulated independently of changes in metabolic rate and blood acid-base status during and following exercise to fatigue.