

**ALL TROUT ARE NOT CREATED EQUAL:
HYPOXIA-TOLERANCE OF THE *IN SITU*
RAINBOW TROUT (*ONCORHYNCHUS MYKISS*) HEART**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Introduction

Fish have a wide range of myocardial hypoxia tolerance. For example, tuna can't maintain cardiac performance during modest hypoxia ($P_{O_2} = 50-140$ mmHg) (Bushnell et al., 1990), while hagfish hearts can function in severe hypoxia ($P_{O_2} = 11.25-16.5$ mmHg) (Axelsson et al., 1990). Furthermore, following 30 min. of hypoxia, ventricular strips from carp show full functional recovery whereas ventricular strips from trout recover only 40% of function (Gesser, 1977). Although many studies have examined interspecific differences in myocardial hypoxia tolerance, intraspecific variation has been largely overlooked. The trout heart is generally considered to be hypoxia intolerant (Gesser, 1977; Gamperl, unpublished). However, we have identified a hatchery population of rainbow trout that exhibits a significant degree of myocardial hypoxia tolerance. This paper reports significant functional recovery of *in situ* trout hearts following 10, 20, and 30 min. of severe hypoxia, and compares these results with previous data for the rainbow trout.

Methods

Rainbow trout (*Oncorhynchus mykiss*) were obtained from a local aquaculture supplier and held at Portland State University (PSU) for at least 10 days before experimental use. Trout hearts were isolated using the *in situ* procedures outlined by Farrell et al. (1986). Upon completion of surgery (15-25 min.), the anterior half of the animal was submersed in a temperature controlled saline bath (10°C), and the heart was perfused with oxygenated saline. Output pressure was set to a physiologically realistic level (50 cm of H₂O) and filling pressure was adjusted to maintain a resting cardiac output of 16 ml kg⁻¹min⁻¹. Fish were then exposed to 1 of 4 experimental protocols (N = 8 in all groups): control, 10, 20, or 30 min. of severe hypoxia (P_{O₂} 5-10 mmHg). Output pressure was maintained at 50 cm of H₂O throughout each of these treatments. Maximum cardiac output (Q_{max}) was measured before and after treatment, in order to assess the degree of functional myocardial damage caused by each protocol.

Results

Previous work, at Simon Fraser University (SFU), showed that the maximum cardiac function of *in situ* trout hearts decreased by approximately 30% following 15 min. of hypoxia at sub-physiological workloads (Figure 1) (Gamperl, unpublished). However, these protocols failed to functionally damage hearts from the population of trout used at PSU. Comparable decreases in cardiac function could only be achieved by extending the hypoxic period to 30 min. and increasing output pressure from 10 to 50 cm (Figure 1).

Discussion and Conclusions

The rainbow trout studied at PSU showed greater myocardial hypoxia tolerance than fish studied at SFU (Figure 1). Furthermore, hearts from fish examined at PSU maintained 77% of function following 30 min. of hypoxia; a value similar to that reported for myocardial strips from anoxia-tolerant carp, but dramatically greater than values measured for trout (Gesser, 1977) (Figure 2).

The factors leading to the enhanced hypoxia tolerance of trout hearts at PSU are presently unknown, however, they may be linked to low water quality or oxygen tensions at the aquacultural facility, and/or gill damage associated with branchial copepod infections.

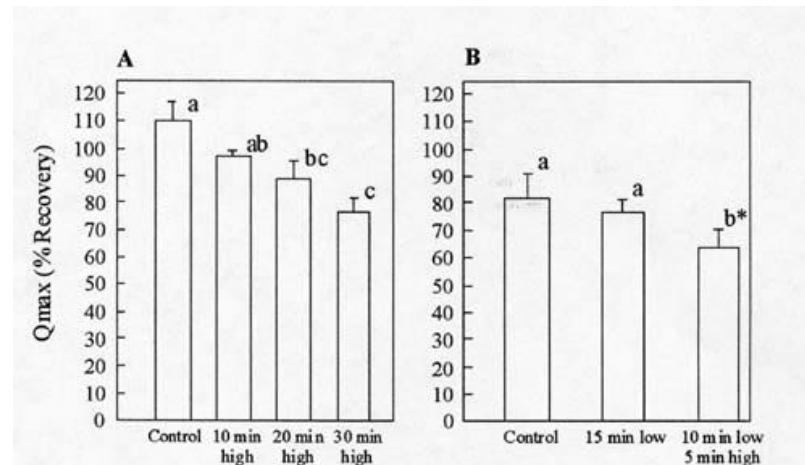


Figure 1. Recovery of maximum cardiac output (Q_{max}) following various treatment protocols at A) Portland State University and B) Simon Fraser University. Protocols are defined in terms of min. of exposure to severe hypoxia (P_{O_2} 5-10 mmHg), at either high (50 cm H_2O) or low (10 cm H_2O) output pressures. Dissimilar letters indicate values that are significantly different ($P < 0.05$), (*) indicates values that are significantly different at $P = 0.07$. $N = 8$ in all groups, except for the 15 min. low P_{out} group on graph B, where $N = 7$.

Our evidence for intraspecific differences in myocardial hypoxia tolerance is based solely on functional recovery, an indirect measurement of myocardial damage. Therefore, we are exploring whether the release of biochemical markers (myoglobin, creatine kinase, and lactate dehydrogenase) can be used to directly quantify irreversible myocyte injury. The release of myoglobin and creatine kinase has been strongly correlated with cardiac myocyte necrosis in mammals (Stokke et al., 1998; Liang, 1996).

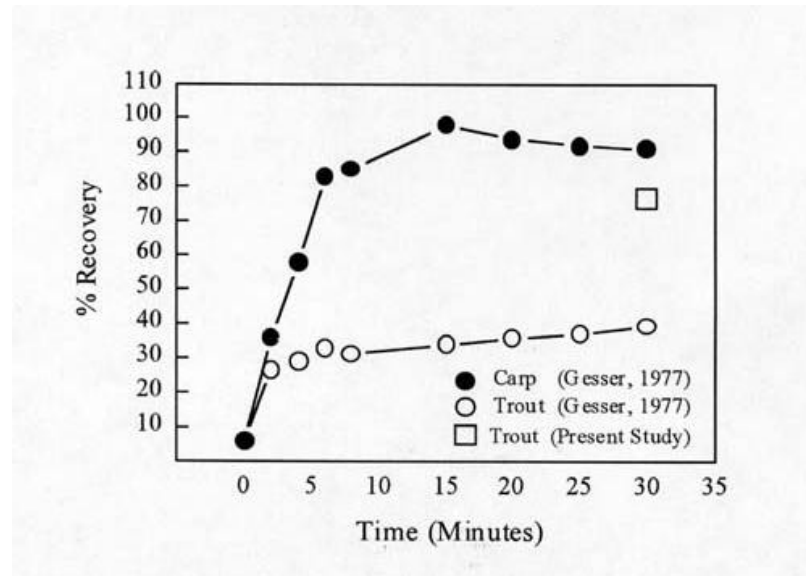


Figure 2. Percent recovery (force) of ventricular strips from carp and trout following 30 min. of hypoxia at 20°C (Gesser, 1977), along with the percent recovery (maximum cardiac output) of *in situ* trout hearts following 30 min. of hypoxia at 10°C (present study). During hypoxia, the myocardial strips were paced at 12 Hz, and output pressure of the *in situ* hearts was set to 50 cm H₂O.

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