

**REGULATION OF THE CARDIORESPIRATORY SYSTEM OF
COMMON CARP (*CYPRINUS CARPIO*) DURING SEVERE HYPOXIA
AT THREE ACCLIMATION TEMPERATURES**

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Introduction

Unlike the large amount of information that exists for short-term moderate hypoxia, *in vivo* measurements of the cardiovascular responses of anoxia-tolerant teleosts, such as the common carp, to a prolonged exposure of severe hypoxia are limited to indirect measurements of cardiac output (Q). For example, Garey (1970) reported that at 9-11°C, Q of two carp decreased by 26% and 39% when the water oxygen partial pressure gradually decreased to below 4.40 kPa over 2 h. In contrast, Itazawa and Takeda (1978) reported a 30% increase in Q in carp exposed to hypoxia at a temperature of 24.5°C. These small changes in Q certainly appear to be counterproductive for long-term survival under oxygen-limiting conditions because survival is dependent on the ability to depress energy demand relative to the glycolytic potential for ATP production. Conversely, the small changes in Q could simply reflect the short duration and moderate level of hypoxia used in the studies.

We recently aimed to resolve this uncertainty by directly monitoring Q in common carp during prolonged severe hypoxic exposure (Stecyk and Farrell, 2002). In contrast to the earlier studies, our measurements clearly revealed an appreciable cardiac depression with severe hypoxia at acclimation temperatures of 6, 10 and 15°C. In our ongoing study into the cardiorespiratory responses of common carp (*Cyprinus carpio*) to severe hypoxia, we have simultaneously

monitored changes in ventral aortic pressure and Q during hypoxic exposure, allowing for the calculation of cardiac power output and thus a quantitative insight into the problem of cardiac energy supply and demand expressed in terms of cardiac ATP demand. Furthermore, we were also interested in elucidating the underlying mechanisms of the reduced cardiac activity observed during prolonged severe hypoxic exposure. Specifically, we were interested in determining whether the depression in cardiac activity occurring with prolonged severe hypoxic exposure was due to extrinsic autonomic regulatory mechanisms or the intrinsic effects of severe hypoxia.

Methods

Experiments were conducted at three acclimation temperatures (5°C, 10°C and 15°C) in order to investigate the role of temperature on cardiovascular control during prolonged severe hypoxia. An ultrasonic flow probe (Transonic Systems., Ithaca, NY) was implanted around the ventral aorta to measure Q , heart rate, and stroke volume and the afferent branchial artery of the third gill arch was occlusively cannulated (PE 50) to measure ventral aortic pressure. In addition, respiration rate and amplitude were monitored through the cannulation of the buccal cavity. Cardiorespiratory regulation was examined through serial intra-arterial injections of α -adrenergic (phentolamine), cholinergic (atropine), purinergic (aminophylline), and β -adrenergic (propranolol) antagonistic drugs (1 mg kg⁻¹) after 12.5 h (5°C), 3.5 h (10°C) and 1.17 h (15°C) of severe hypoxia (0.1 – 0.3 mg O₂ L⁻¹; approximately 0.2 – 0.6 kPa).

Results and Discussion

Severe prolonged hypoxia resulted in a large cardiac depression similar to that previously documented (Stecyk and Farrell, 2002) and an increased peripheral resistance at acclimation temperatures of 5, 10, and 15°C. However, calculation of cardiac power output revealed that despite the large cardiac downregulation occurring with prolonged severe hypoxia, common carp are unable to reduce cardiac energy demands to a level that can be supported without an upregulation of glycolysis. Specifically, cardiac power output was reduced 4.6-fold (5°C), 7.6-fold (10°C) and 4.3-fold (15°C) with severe hypoxia, considerably less than the approximate 18-fold reduction in ATP yield per mol glucose with glycolysis compared to oxidative metabolism. As a result, cardiac energy stores will be depleted more rapidly than if there was no Pasteur effect. This conclusion is consistent with the finding that phosphocreatine and ATP levels continuously

decrease throughout anoxic exposure in the common carp without reaching a plateau, as found in other anoxia-tolerant species (van Waarde et al., 1990).

Serial intra-arterial injections of α -adrenergic, cholinergic, purinergic, and β -adrenergic antagonistic drugs during severe hypoxia revealed that autonomic control of the cardiovascular system was conserved at all three acclimation temperatures (Figure 1). Specifically, injection of the α -antagonist phentolamine, which decreased systemic resistance and increased heart rate, illustrated that a strong α -adrenergic tone and barostatic reflex were retained during severe hypoxia. Atropine injection after α -adrenergic blockade did not further increase cardiac activity, while injection of the β -adrenergic antagonist propranolol reduced cardiac status to routine hypoxic levels indicating a large β -adrenergic tonus on the heart. Injection of the purinergic antagonist aminophylline in hypoxic fish had no cardiovascular effects, but significantly increased respiration rate (Figure 2).

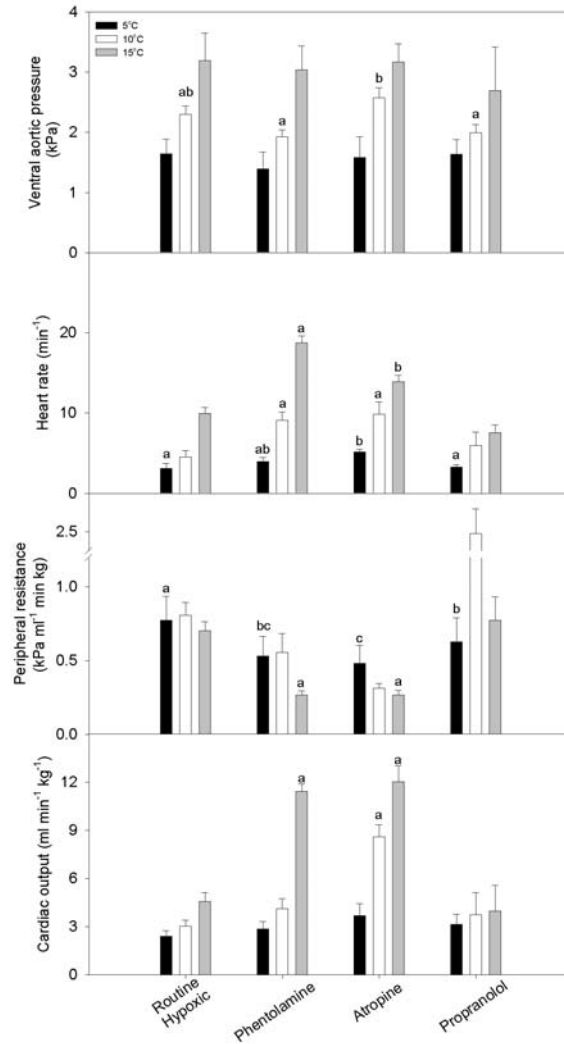


Figure 1. Changes in P_{VA} , f_{HP} , peripheral resistance and Q with α -adrenergic, cholinergic and β -adrenergic blockade during severe hypoxia. Significant differences ($P < 0.05$) among drugs within each temperature group are indicated by dissimilar letters. [$N=6$ (5°C, 10°C) and 3 (15°C)]

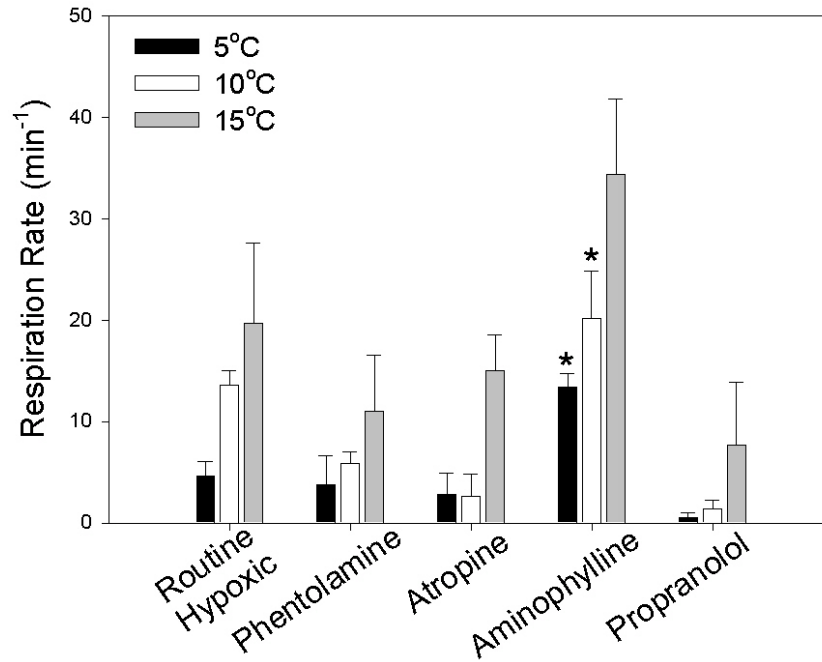


Figure 2. Changes in respiration rate with α -adrenergic, cholinergic, purinergic and β -adrenergic blockade during severe hypoxia. Significant differences ($P < 0.05$) among drugs within each temperature group are indicated by a *. [$N=6$ (5°C, 10°C) and 3 (15°C)]

References

- Garey, W. 1970. Cardiac output of the carp (*Cyprinus carpio*). *Comp. Biochem. Physiol.* 33:181-189
- Itazawa, Y. and T. Takeda. 1978. Gas exchange in the carp gills in normoxic and hypoxic conditions. *Respir. Physiol.* 35:263-269

Stecyk, J.A.W. and A.P. Farrell. 2002. Cardiorespiratory responses of the common carp (*Cyprinus carpio*) to severe hypoxia at three acclimation temperatures. *J. Exp. Biol.* 205:759-768

van Waarde, A., van den Thillart, G. Erkelens, C., Addink, A. and J. Lugtenburg. 1990. Functional coupling of glycolysis and phosphocreatine utilization in anoxic fish muscle. *J. Biol. Chem.* 265:914-923

Acknowledgments

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