

**THE EFFECT OF THE GASEOUS ENVIRONMENT
ON THE CARDIOVASCULAR DEVELOPMENT
OF TILAPIA, *Oreochromis niloticus***

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Introduction

We are interested in the developmental plasticity of the cardiovascular system. Cardiovascular development has drawn a lot of attention recently, probably because the heart is one of the first organs to function and the cardiovascular system one of the first organ systems to operate in any vertebrate embryo. Even so, few data exist for heart rate during development in fish. Even fewer data exist for the development of stroke volume and cardiac output, the latter being the most important variable for oxygen transport. A few studies have looked at the effects of altering the environment of developing fish embryos and larva, although most of these focus on the effects of acute changes to the environment, rather than morphological or physiological plasticity induced by prolonged exposure.

Methods

The purpose of this experiment was to test whether convective blood flow is necessary at early life stages for the delivery of oxygen to the tissues, and whether the development of the cardiovascular system is sensitive to the environment through feedback from O₂ delivery. In order to explore these

questions, tilapia (*Oreochromus niloticus*) larvae were raised in one of four environments: normoxia, 8% O₂, 2% CO, and a combination of 8%O₂ and 2%CO.

Tilapia eggs were obtained from the tilapia hatchery at Dalhousie University, Halifax, N.S., immediately after fertilization. Each spawning was separated into four groups and placed in one of four 10 gallon closed system treatment tanks: normoxia (control); 8% O₂ (hypoxia); 2% CO (which acts to functionally ablate Hb); and a combination of 8% O₂ and 2% CO (combination). Starting at 120 hours post fertilization, the larval hearts were video taped through an inverted microscope, and heart rate (hr = beats/min, bpm), stroke volume (sv = μ l), and cardiac output determined (co = hr*sv = μ l/min). The experiment was terminated when heart was no longer visible through the pericardial wall. Dry mass of the individual larvae was also determined (mg).

Results

Larval growth rate was affected by respiratory environment right from the start of the experiment, with the control fish growing faster than the other three treatments, CO and hypoxic fish being the same size and falling in the middle of the 4 treatments, and combined CO and hypoxic fish being the smallest of the treatments. This is probably due to the fact that tissue hypoxia decreased metabolism, and thus anabolic pathways, and the effect increased with increased limitation.

Heart rate of the tilapia larvae was unaffected by neither age nor treatment, however the effects of altering the respiratory environment on stroke volume and cardiac output (figure 1) are seen right from the start of the sampling. The data indicate that the cardiovascular system is required for delivery of oxygen to tissue at early stages of development in tilapia, and this need is dependant on stage and environment. Hemoglobin appears to be more necessary at later life stages than earlier ones and this is probably due increased importance of the cardiovascular system in delivering oxygen to tissues. Hypoxia, however, has more of an effect at the earlier stages. The effect of the combined CO and hypoxic environment was greater than hypoxia alone, indicating that cardiovascular oxygen transport seems to be important in tilapia at all life stages in hypoxic environments.

Discussion

The data obtained in this experiment on the effect of the respiratory environment on cardiac output suggest that cardiac output in larval fish is regulated by local tissue-blood vessel interactions acting through Starling effects of venous return. The absence of the bradycardia seen in adult fish in response to hypoxia implies that there is little neural input to the cardiovascular system in these larval fish. The greatly increased stroke volume is probably due to an enlargement of the heart. Growth regulation of the heart could be metabolic or mechanical. For example, hypoxia can dilate vascular beds, resulting in increased blood volume returning to the heart, and an increased stroke volume due to the Starling mechanism (Wadsworth 1994). Increased blood flow and hypoxia inducible factor 1 (HIF1) have been suggested to affect cardiovascular growth and remodeling during normal development, through factors such as vascular endothelial growth factor (VEGF) (Wenger and Gassmann 1997, Ratcliffe et al. 1998).

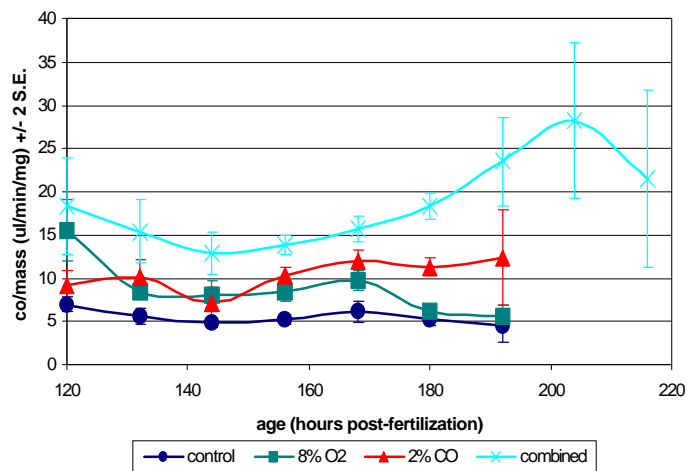


Figure 1. Mass specific cardiac output (co/mass) over time post fertilization for tilapia larvae exposed to four separate gaseous environments during development: normoxia (control); 8% O₂; 2% CO; a combination of 8% O₂ and 2% CO (combined). Standard error bars that do not overlap indicate significant differences between points.

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