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Cross Tolerance to Environmental Stressors: Effects of Hypoxic Acclimation on Cardiovascular Responses of Channel Catfish (*Ictalurus punctatus*) to a Thermal Challenge

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Abstract

Hypoxia and temperature are two major, interactive environmental variables that affect cardiovascular function in fishes. The purpose of this study was to determine if acclimation to hypoxia increases thermal tolerance by measuring cardiovascular responses to increasing temperature in two groups of channel catfish. The hypoxic group was acclimatized to moderate hypoxia (50% air saturation, a P_{O_2} of approximately 75 torr) at a temperature of 22° C for seven days. The normoxic (i.e. control) group was maintained the same, but under normoxic conditions (a P_{O_2} of approximately 150 torr). After acclimation, fish were decerebrated, fitted with dorsal aorta cannulae, and then exposed to increasing temperature while cardiovascular variables were recorded. The end point (critical thermal maximum, CTMax) was defined as a temperature at which heart rate and blood pressure sharply decreased indicating cardiovascular collapse. Fish acclimatized to moderate hypoxia had higher resting heart rate than controls. Hypoxic acclimatized fish had a significantly higher CTMax. Acclimation to hypoxia increases the cardiovascular ability of channel catfish to withstand an acute temperature increase.

Keywords

teleost; heat coma; thermal stress; environment; oxygen; acclimatization

1. Introduction

Arguably, the two most important environmental variables that affect the overall biology of fishes are temperature and O_2 availability. The physico-chemical properties of water and the respiratory physiology of fishes dictate that these variables interact. High temperatures and limited O_2 availability can occur simultaneously or separately in nature and have likely continuously shaped the evolutionary history of cardio-ventilatory physiology of vertebrates. In addition, human activities are having large and rapid effects on temperature and O_2 availability in many waters. Therefore, if we can understand the cumulative effects of hypoxia and temperature on cardiovascular function in fishes, we may gain insights into the evolution of cardiovascular control in vertebrates and better understand how human activity impacts aquatic organisms.

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Acute hypoxia elicits a host of cardioventilatory responses in fishes, mediated by O₂-sensitive chemoreceptors and the release of catecholamines, to maintain O₂ uptake in the face of decreased environmental O₂ availability. The patterns of response to long-term, or chronic hypoxia vary, depending upon species, the time-scale and perhaps degree of the exposure. In a previous study, channel catfish held in moderate hypoxia (P_{O₂} = 75 torr) for one week showed increased ventilatory sensitivity to hypoxia and, although heart rate was significantly greater, as compared to controls, the cardiac sensitivity to hypoxia was unchanged (Burleson et al., 2002). The ventilatory response of zebra fish (*Danio rerio*), acclimatized to severe hypoxia (P_{O₂} = 30 torr) for 28 days, was blunted (Vulesevic et. al., 2006). Atlantic cod (*Gadus morhua*) acclimatized to hypoxia (P_{O₂} = 60–68 torr) for 6–12 weeks showed reduced cardiac output at rest and during exercise; however, metabolic rate, scope and swimming speed were unchanged when compared to controls (Petersen and Gamperl, 2010a).

In contrast to hypoxia, which typically elicits bradycardia, increasing temperature increases heart rate in fishes, as part of and overall increase in cardiac output (Farrell, 2002; Mendonça and Gamperl, 2010). This along with increased ventilation serves to maintain O₂ uptake in the face of the increasing metabolic demand stimulated by increasing temperature. Like the cardio-ventilatory responses to hypoxia, responses to increased temperature vary in pattern and magnitude among different fish species and with acclimation conditions/history. Increased cardiac output is achieved primarily by increases in rate (Aguiar et al., 2002; Mendonça and Gamperl, 2010). Cardiovascular variables begin to decrease with increasing temperature as fish reach their critical thermal maximum (CTMax) (Steinhausen et al., 2008) culminating in cardiovascular collapse.

The CTMax of an animal is a “predefined sub-lethal endpoint” such as loss of equilibrium or muscle spasms that prevents escape from lethal temperatures (Beitinger and Bennett, 2000). The CTMax of fishes vary as greatly as the cardiovascular responses to hypoxia and temperature as described above and depends largely on species and acclimation history (Beitinger et al., 2000). CTMax provide useful information for assessing and predicting the effects of increasing temperatures on populations of fish.

The mechanisms that ultimately determine CTMax are not completely understood, but current evidence, from diverse species, indicates that O₂ delivery limitations may play a pivotal role in setting upper CTMax in some fishes (Pörtner, 2010). Data on salmonids suggest that O₂ supply to the myocardium directly sets the upper limit on cardiac function in response to high temperatures (Farrell, 2002). It follows then, that physiological responses that enhance O₂ uptake, i.e. acclimation to hypoxia, should increase the CTMax.

Previous studies, in a variety of organisms, have demonstrated cross tolerances between temperature and O₂ availability and the responses to these two environmental variables appear to be mediated through several common mechanisms at the molecular level. Experiments at the organismal/reflex level, however, are lagging behind biochemical studies. The purpose of this study is to assess the functional consequences of acclimation to hypoxia on cardiovascular responses to acute temperature increase in channel catfish.

2. Material and Methods

2.1 Animal Preparation

Channel catfish (228 – 733 g, n = 16) were obtained from local suppliers. All were adults and either sex were used randomly. Catfish were maintained in fiberglass holding tanks filled with dechlorinated tap water and equipped with recirculating gravel filtration systems. The holding tanks were maintained at 22° C in a temperature controlled room on a 12 hour/

dark cycle, and the fish were fed commercial animal food once a week. Procedures were approved under IACUC #03-018.

2.2 Normoxic and Hypoxic Acclimation

Catfish were exposed to one of two levels of O₂ tension prior to surgery. Fish were placed in 76 liter glass aquaria kept at either 100% air saturation (approximately 150 torr P_{O₂}) for the normoxic control group (n=8) or 50% air saturation (75 torr P_{O₂}) for the hypoxic experimental group (n=8). The aquaria were covered with plastic lids to reduce gas exchange with the atmosphere. The O₂ concentration was controlled by using flowmeters to control the mix of air and nitrogen being fed into the water through the filtration system. O₂ levels were measured twice daily using a dissolved O₂ meter (Yellow Springs Instruments Model 58). Biological foam filters were used to filter the water and keep ammonia levels low. Fish were held for six days and were unfed during this period. On the sixth day the fish underwent surgery and then were transferred to the experimental chamber and allowed to habituate for 24 hours. If the fish were hypoxic the experimental chamber was kept hypoxic (50% oxygen saturation) using a 50/50 mixture of pure nitrogen and air. The nitrogen was adjusted using a flow meter (Cole Parmer Instruments) until the appropriate O₂ level was maintained. The O₂ levels were monitored with an O₂ electrode and associated meter (Microelectrodes Inc.).

2.3 Surgical Procedure

On the sixth day of holding, fish underwent surgery to implant a dorsal aorta cannula for the measurement of blood pressure and heart rate and decerebrated to remove the forebrain. Decerebration insured that the cardiac responses were not a result of a behavioral response to temperature. Fish were anesthetized in a 0.01% dissolved solution of MS-222 and dechlorinated tap water bubbled with O₂. The fish were then transferred to the surgery table where they were artificially ventilated with the same oxygenated anesthetic solution. The dorsal aorta was cannulated with polyethylene tubing (PE 50), secured to the roof of the mouth with sutures, guided out of the mouth through a hole drilled at the snout between the mental barbells, and finally secured with a sleeve and cuff of polyethylene tubing. Heparinized saline was injected in the cannula to avoid blood clotting once the cannula had been successfully inserted, and the cannula was plugged with a blunted 23 gauge hypodermic needle filled with wax. Decerebration was performed by cutting a 1×1 cm² hole on the top of the head just above the forebrain of the catfish and removing the forebrain by suction. After decerebration, the cut-away piece of skull was resituated, overlaid and sealed with a molded thermoplastic cover and secured to the head using stainless steel surgical screws.

2.4 Protocol

Following surgery, the fish were placed in the experimental setup for 24 hours at the proper O₂ level. The experimental setup consisted of a plexiglass chamber painted black to reduce disturbance of the fish. Dechlorinated water was cycled through the chamber using a circulating water temperature controller (MGW Lauda RM20). On the seventh day of holding, 30 min before beginning the protocol, O₂ content was raised to 100% air saturation if the fish was hypoxic. The dorsal aorta cannula was led out of a small hole on the top of the chamber and connected to a blood pressure transducer (Colbourn Instruments) which in turn was connected via an analog to digital data interface (DATAQ) to view and record blood pressure on a computer data acquisition program (Windaq). Water temperature was also measured and recorded using a telethermometer (Yellow Springs Instruments). Output of the telethermometer was calibrated against a mercury thermometer. O₂ partial pressure was measured by feeding water from the circulating temperature controller's reservoir

through a peristaltic pump (Buchler Instruments) across an oxygen electrode (Microelectrodes Inc.) connected to a oxygen meter (Cameron Instruments).

Once a fish was connected to the proper experimental equipment, the temperature of the water was raised using the Lauda temperature control from the initial starting temp of 25 ± 1.33 °C at a rate of 1°C every 2 minutes, until the critical thermal maximum (CTMax) occurred. CTMax has been defined in various ways (see Beitinger et al., 2000). The definition used in this experiment was the onset of muscular spasms that immediately precede heat shock and cardiovascular collapse (Kowalski et al., 1978).

2.4.3. Cardiovascular Measurements

Dorsal aorta blood pressure (P_{DA}) (mean arterial, systolic and diastolic pressures, cm H₂O) and heart rate (fH, beats/min) were obtained from Windaq recordings of the blood pressure trace during the experiment using the postacquisition analysis program Windaq Waveform Browser. Mean arterial blood pressure was calculated as the sum of the systolic and diastolic pressures divided by two. The CTMax was obtained by observing when the catfish went into muscle spasms; this was observed visually and confirmed by the Windaq blood pressure trace.

2.5 Statistical Analyses

The effects of hypoxic acclimation on mean thermal tolerance was analyzed with a 2-way, repeated measures ANOVA. Normoxic and hypoxic variables were compared at each temperature point beginning at 26.0°C until 36.0°C by 2°C increments. The Scheffé test for *post hoc* comparisons was used to determine differences between control and experimental treatments at specific temperatures. A student's t-test was performed to determine if acclimation to chronic hypoxia had a significant effect on mean CTMax. Statistical significance was considered to be $p \leq 0.05$ for all analyses. All data are expressed as mean $\pm$ S.E.M.

3. Results

Channel catfish responded to increasing temperature with an overall increase in all monitored cardiovascular variables until CTMax was reached. Heart rate became increasingly irregular with increasing temperature. A representative cardiovascular trace for an individual fish responding to increasing temperature is shown in Figure 1.

Mean arterial pressure (P_{DA}) increased significantly with increasing temperature but normoxic and hypoxic acclimatized groups were not significantly different (Fig. 2). Diastolic pressure showed the same pattern of response in both acclimation groups (Fig. 3), increasing significantly with increasing temperature but there was no significant difference between acclimation groups. Systolic pressure increased significantly with increasing temperature in both groups, however, there was a significant decrease in the normoxic group when temperature reached 36.0°C (Fig. 4). Fish acclimatized to hypoxia were able to maintain systolic pressure at higher temperatures.

Heart rate increased with increasing temperature and became more irregular at higher temperatures (Fig 5). Mean heart rate was significantly higher in hypoxic acclimatized fish between 26–30°C. Above 30°C, the heart rates of both groups of fish were the same and increased significantly with increasing temperature. Critical thermal maximum, measured at the point of cardiovascular collapse was significantly ($P = 0.029$) higher in hypoxia acclimatized fish (36.09 ± 0.22 °C) than normoxic (35.47 ± 0.39 °C).

Although increasing temperature decreases the solubility of O₂ in water, and consequently O₂ content, calculated content of normoxic water dropped only from 8.1 mg/l at 26°C to 6.8 mg/l at 36°C. The U.S. Geological Service defines aquatic hypoxia as O₂ content less than 2 mg/l (<http://toxics.usgs.gov/definitions/hypoxia.html>).

5. Discussion

Exposure of fish to high temperature or hypoxia will increase their tolerance to extreme changes in the acclimatized variable. For example, acclimation to high temperature increases CT_{Max} (Beitinger et al., 2000) and preconditioning to sub-lethal hypoxia have been shown to have increased survival time during exposure to lethal hypoxia (Rees et al., 2001). This study now demonstrates cross tolerance of the cardiovascular system of channel catfish to acute temperature increase after acclimation to moderate hypoxia resulting in an elevated CT_{Max}.

Acclimation to hypoxia alters a number of physiological systems depending upon the time-course and level of hypoxia. Perhaps the most studied of these is ventilatory acclimation to hypoxia which is a time-dependent increase in ventilatory sensitivity to hypoxia and occurs in response to long-term hypoxia. The mechanisms that underlie ventilatory acclimation to hypoxia are not completely understood, but experimental evidence suggests that it involves changes in the sensitivity of both central and peripheral neural components that control breathing (Hainsworth et al., 2007). All vertebrates that have been examined, including channel catfish, show ventilatory acclimation to hypoxia (Burleson et al., 2002). Acclimation to hypoxia also alters neural and biochemical control and function of the heart and vasculature (for reviews see Hainsworth et al., 2007; Ostadal and Kolar, 2007). Most studies are on mammals, with only a few having examined how acclimation to hypoxia affects cardioventilatory control in fishes (Burleson et al., 2002; Vulesevic et al., 2006; Petersen and Gamperl, 2010a).

Previous studies have shown cross tolerance between hypoxia/anoxia and high temperature stress in a variety of taxa: mammals (rats) (Wen et al., 2002; Tetievsky et al., 2008), insects (*Locusta migratoria*) (Wu et al., 2002), nematodes (*C. elegans*) (Treinin et al., 2003) and plants (*Arabidopsis*) (Banti et al., 2008). Although cross tolerance appears to be a widespread phenomenon, similarities and/or differences in the physiological mechanisms across different organisms have yet to be determined.

The reported effects of temperature increase on blood pressure in fishes are variable. Catfish, like rainbow trout (*Oncorhynchus mykiss*) (Heath and Hughes, 1973; Sandblom and Axelsson, 2007) increase P_{DA} in response to increasing temperature. Chinook salmon (*Oncorhynchus tshawytscha*) (Clark et al., 2008) show no change and winter flounder (*Pseudopleuronectes americanus*) decrease P_{DA} in response to increasing temperature (Mendonça and Gamperl, 2010). There was not a significant difference in mean arterial pressure (MAP) between treatment groups of catfish, but when the two components of MAP, systolic and diastolic pressure, were analyzed separately, different patterns of response were observed (Figs 3 and 4). The significant drop in systolic pressure in the normoxic group at the highest temperature is a good indication that the heart is beginning to fail earlier than in the hypoxic group.

Hypoxic bradycardia in fishes is an acute cardiac response to decreasing environmental O₂ availability, whereas tachycardia is the cardiac response to increasing O₂ demand. Although the responses address the same physiological goal, maintenance of aerobic metabolism, they are mediated by different reflex mechanisms and neural pathways. Cardiovascular responses of channel catfish to increasing temperature were similar to previously published data on

various fish species (Stevens et al., 1972; Heath and Hughes, 1973; Aguiar et al., 2002; Gollock et al., 2006; Steinhausen et al., 2008; Mendonça and Gamperl, 2010). Increased heart rate appears to be the usual response to increasing temperature. Increased rate, but often not stroke volume (Mendonça and Gamperl, 2010a), serves to increase cardiac output to meet increased metabolic demand in response to increasing temperature.

The reported effects of acclimation to hypoxia on heart rate in fishes are variable. This and a previous study (Burleson et al., 2002) show, contrary to predictions, that channel catfish acclimatized to hypoxia have significantly higher heart rate than normoxic controls. Resting fH of Atlantic cod (*Gadus morhua*) are no different in hypoxic and normoxic acclimatized groups *in vivo* and *in situ* (Petersen and Gamperl, 2010a&b); however, hypoxic cod had significantly greater fH at high critical swimming speeds (Petersen and Gamperl, 2010a). Differences in fH between treatment groups were maintained during hypoxia (Burleson et al., 2002), however, the only effect of acclimation to hypoxia on the fH response to increasing temperature was to delay the increase that brought both treatment groups to the same trajectory (Fig. 5).

Oxygen availability has a significant effect on thermal tolerance in many aquatic ectotherms (Rutledge and Beitinger, 1989; Pörtner, 2010). At the point of CTMax, a cascade of events related to O₂ demand and availability leads to a decrease in heart rate and cardiovascular collapse. Increased metabolic demand in the tissues is met by increased O₂ extraction from the blood. This leads to low venous P_{O2} which leads to insufficient O₂ supply to the myocardium (Farrell, 2002). The CTMax reported here are within the ranges reported previously for channel catfish summarized in Beitinger et al., 2000.

A variety of direct and indirect mechanisms have been hypothesized and/or demonstrated to contribute to CTMax. These include direct effects on the cardiovascular system, changes in hemoglobin oxygen affinity, disruption of acid-base and osmotic/ionic balance which can directly affect enzyme, nerve and muscle function (Crawshaw, 1979). Some cellular processes such as signal transduction pathways and myofilament Ca⁺² sensitivity show direct temperature sensitivity (Gamperl and Farrell, 2004). The physiological adjustments to long-term hypoxia are presumed to increase the ability to maintain O₂ delivery to the tissues in the face of decreased environmental O₂ availability. That acclimation to hypoxia improves thermal tolerance further demonstrates the central role of O₂ in the ability of fishes to withstand thermal stress.

The differences in heart rate, systolic blood pressure and CTMax in hypoxic acclimatized and control fish, although statistically significant, were not large. This may be explained by either the level of hypoxia used (50% air saturation), which is not severe for this species or the relatively short period allowed for acclimation (seven days). Other studies have used more severe hypoxia (10% saturation) for shorter time periods (48 hrs) (Rees et al., 2001, zebrafish, *Danio rerio*), more severe hypoxia (20–25% saturation) for longer time periods (28 days) (Vulesevic et al., 2006, zebrafish), or similar levels of hypoxia (40–45%) for longer time periods (6–12 weeks) (Petersen and Gamperl, 2010a, cod). These differences in protocol likely contribute, along with differences in species, to the variability of physiological responses reported in these different studies.

Despite the differences in the physiological responses to hypoxia and thermal stress, both stressors activate common molecular pathways at the cellular level. It has been shown in diverse organisms, from plants to vertebrates, that HIF (hypoxia-inducible factor) and HSP (heat shock proteins) are generically activated by either stress, and exposure to one often confers increased resistance to the other (Wen et al., 2002; Wu et al., 2002; Banti et al.,

2008; Tetievsky et al., 2008). This suggests that HSP and HIF are probably part of a more generalized response to stress and not specific to either hypoxia or temperature.

This study demonstrates that moderate hypoxia can alter the cardiovascular responses to an acute increase in temperature and significantly elevate the CTMax in a hypoxic-tolerant species over a relatively short period of exposure time (seven days). Genomic and proteomic studies in channel catfish should provide evidence for the molecular basis of this hypoxic/thermal stress cross tolerance.

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Figure 1. Representative trace showing the cardiovascular effects of increasing temperature. Top trace: blood pressure recorded from the dorsal aorta. Bottom trace: temperature recorded from probe.

Figure 2. Increasing temperature significantly increased mean arterial pressure but there was no significant difference between acclimation groups. Data in this and subsequent graphs are mean $\pm$ S.E.M. N = 8 at 26–34 °C. N = 6 at 36 °C in normoxic controls and 7 in the hypoxic group.

Figure 3.
Increasing temperature significantly increased diastolic pressure in both acclimation groups, but there was no significant difference between groups.

Figure 4. Increasing temperature significantly increased systolic pressure. There was no significant difference between acclimation groups until temperatures rose above 34°C and systolic pressure decreased in the normoxic group (*).

Figure 5.

Heart rate was initially, significantly higher in the hypoxic acclimatized group (*). As temperature increased, heart rate increased in both treatment groups until they were not significantly different from 30 °C and up.