INSTITUTIONEN FÖR BIOLOGI OCH MILJÖVETENSKAP

Thermal Tolerance in Teleost Fish
Importance of Cardiac Oxygen Supply, ATP Production and Autonomic Control

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Dissertation Abstract

Temperature tolerance is a key determinant of the resilience and adaptability of fish facing a warmer and more thermally variable future with climate change. Yet, the underlying physiological mechanisms determining the critical thermal maximum (CT_{max}) are poorly understood. This thesis investigated the physiological determinants of CT_{max} in teleost fish, focusing on cardiovascular function. An inability of the heart to pump and supply the body tissues with oxygenated blood could constrain whole animal tolerance to high temperatures. This has been hypothesized to be related to an oxygen limitation of the heart, which receives its oxygen supply via the venous blood (luminal circulation), and in some species also via a coronary circulation.

This hypothesis was first tested by evaluating the relationship between luminal oxygen supply, via continuous recordings of the venous oxygen tension (P_{VO2}), and *in vivo* cardiovascular performance and CT_{max} in European perch (*Perca fluviatilis*). Perch were sampled from the Baltic Sea (reference, 18°C) and the Biotest enclosure (24°C, Biotest) that is chronically warmed by cooling water effluents from a nuclear power plant. While CT_{max} was 2.2°C higher in Biotest compared to reference perch, cardiac failure (*i.e.* reduced heart rate and cardiac output) occurred at similar P_{VO2}. By artificially increasing the oxygen availability to the heart through water hyperoxia (200% air saturation), it was revealed that while heart rate still declined at high temperatures, cardiac stroke volume and cardiac output were maintained. This demonstrates that mainly stroke volume is sensitive to limitations in luminal oxygen supply. In rainbow trout (*Onchorhynchus mykiss*), coronary blood flow first increased with moderate warming, but plateaued at higher temperatures suggesting limitations to the coronary vasodilatory reserve. Ligation of the coronary artery reduced CT_{max} and impaired cardiac performance during warming, which was reflected in an elevated heart rate across temperatures, possibly to compensate for an impaired myocardial contractility and stroke volume of the oxygen deprived ventricle.

A thermal impairment of mitochondrial ATP production could also explain reductions in cardiac performance of acutely warmed fish. This hypothesis was tested by evaluating the catalytic capacities of key enzymes involved in ATP production in the perch heart. The main findings suggest that mitochondrial function is impaired at critically high temperatures by a reduced production of NADH and $FADH_2$ in the tricarboxylic acid cycle, which provides the electrons necessary for driving mitochondrial ATP production. Moreover, a temperature dependent failure of several complexes in the electron transport chain was observed, which would also limit the synthesis of ATP at high temperatures. Indications of an increase in oxidative capacity were observed in the warm acclimated Biotest perch, which may be associated with their improved cardiac thermal performance and elevated CT_{max}.

Finally, it was hypothesized that cholinergic inhibition of heart rate could improve cardiac oxygenation during warming, and that adrenergic stimulation may improve cardiac contractility at high temperatures and reduced cardiac oxygen availability. These hypotheses were tested in rainbow trout by pharmacologically blocking the cholinergic and adrenergic input to the heart. However, neither of the treatments resulted in earlier onset of cardiac failure during acute warming, or a reduced CT_{max}. This could reflect that the heart was adequately oxygenated via compensatory increases in coronary flow, and/or that an increased cardiac filling pressure served to maintain cardiac output.

Collectively, these findings provide novel insights into the causal factors underlying thermal tolerance and cardiac failure during acute warming in teleost fish *in vivo*. While whole animal thermal tolerance limits likely involve thermal failure at several levels of physiological organization, a failing heart undoubtedly plays a crucial role for the sensitivity of fish to a warmer and more thermally extreme future.