DISSERTATION ABSTRACT

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In fish and other vertebrates, venous capacitance changes have important implications on venous return and cardiac filling pressure. The main objective of this thesis was to gather information on venous haemodynamic responses and neurohumoral control mechanisms in two teleost species; the sea bass, *Dicentrarchus labrax*, and the rainbow trout, *Oncorhynchus mykiss*. As previous studies of venous function in fish have primarily focused on the pharmacology of the venous vasculature, special attention was paid to venous responses elicited by exercise, acute temperature changes and environmental hypoxia, which represent natural cardiovascular challenges in aquatic environments.

Methods: Cardiac output (Q), central venous (P_{ven}) and dorsal aortic (P_{da}) blood pressures were recorded *in vivo*. The mean circulatory filling pressure (MCFP), a measure of vascular capacitance, was measured as the venous plateau pressure during ventral aortic occlusion. In one study, vascular capacitance curves were also constructed by measuring MCFP at different blood volumes (between 80-120% of the assumed blood volume), to investigate changes in vascular compliance (C) and unstressed blood volume (USBV) during normoxia and hypoxia. In another study, blood volume was measured using dilution of ^{51}Cr-labelled red blood cells. Drugs were administered systemically to elucidate the role of adrenergic control systems and the renin-angiotensin system (RAS) in the observed cardiovascular responses.

Results and conclusions: Exercise, in both sea bass and rainbow trout, results in increased Q and increased MCFP. Although P_{ven} increases during exercise in both species, cardiac stroke volume (SV) only increases in rainbow trout, whereas increased heart rate (f_H) is exclusively responsible for the increased blood flow in sea bass. When ambient temperature was raised acutely from 10 to 13 and 16°C, rainbow trout respond with a significantly elevated Q which, in contrast to the exercise response, is exclusively mediated by tachycardia with an unchanged P_{ven} and SV. Similarly, however, MCFP increases which indicates an actively reduced vascular capacitance, especially since the blood volume does not change between 10 and 16°C. In both species, blockade of α-adrenoceptors delays the increase in P_{ven} during exercise, and in rainbow trout, additional blockade of angiotensin converting enzyme abolishes all venous exercise responses. Environmental hypoxia typically elicits bradycardia that is associated with reduced vascular capacitance and an increased P_{ven}. Q is unchanged or increased during hypoxia due to an increased SV. The capacitance responses during hypoxia are mainly due to changes in USBV that are mediated by both nervous and humoral α-adrenergic mechanisms.

In summary, it is shown that vascular capacitance decreases during exercise, acute temperature increase and hypoxia. This mobilizes blood to the central venous compartment which, depending on the heart rate response, results in maintained or increased P_{ven}, SV and Q. It is also suggested that the decrease in capacitance during exercise and acute temperature increase prevents blood from passively pooling in the venous periphery as blood flow increases. RAS is activated during exercise after α-blockade to increase P_{ven} and MCFP. Thus, RAS affects venous capacitance in fish and not only arterial tone as previously suggested.

Keywords: blood volume, catecholamines, central venous pressure, exercise, hypoxia, mean circulatory filling pressure, preload, renin-angiotensin, temperature, vascular capacitance.