ABSTRACT

Effect of swimming training on the arterial vasomotor function in spontaneously hypertensive rats (SHR): role of reactive oxygen and nitrogen species

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Experimental and clinical evidence indicates that vascular superoxide (O$_2^-$) play an important pathophysiological role in the development of hypertension, associated with decreased nitric oxide (NO) bioavailability and impaired endothelial-dependent vasorelaxation. Aerobic exercise training (AET) decreases blood pressure and improves endothelial-dependent vasorelaxation in hypertensive. However, the molecular mechanism of training-induced improvement of vasomotricity is not completely understood. The purpose of this study was examine the effect of AET (swimming, 10 wk) on the aorta function in SHR rats, emphasizing the O$_2^-$ and NO production indices. Results: SHR rats showed decreased maximal vasorelaxation and paradoxical vasoconstriction at high doses of ACh. This response was related to increased O$_2^-$ and decreased NO bioavailability, associated with increased expression of eNOS and NOX-4. On the other hand, swimming AET improved the endothelial-dependent vasorelaxation, which were associated with decreased O$_2^-$ and improved NO bioavailability. Conclusion: the results show that swimming AET reduces vascular oxidative stress, improves NO biodisponibility and increases endotelium-dependent vasorelaxation in SHR rats. These changes may be involved in blood pressure reduction in trained SHR rats.
Keywords: aerobic exercise training, hypertension, SHR, oxidative stress, endothelial function, rat.