Inhibition of Aerobic Exercise on PKC/CaV1.2 pathway enhanced the function of vascular smooth muscle in hypertension

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Objective The purpose of this study was to investigate the effects of aerobic exercise on PKC/CaV1.2 pathway in mesenteric arterial smooth muscle from spontaneously hypertensive rats (SHRs).

Methods Twelve-week-old male normotensive Wistar-Kyoto (WKY) rats and SHRs were randomly assigned to sedentary groups (SHR-SED, WKY-SED) and exercise training groups (SHR-EX, WKY-EX). Exercise groups were performed an 8-week moderate-intensity treadmill running. After 8 weeks, vascular contractility of mesenteric arteries was measured. Vascular smooth muscle cells (VSMCs) were obtained with an enzymatic isolation method. CaV1.2 channel currents were examined by using whole-cell patch clamp recording technique.

Results 1) Body weight and systolic blood pressure (SBP) in both WKY-EX and SHR-EX were significantly lower than those of their sedentary counterparts (both P < 0.05). Body weight in SHR-SED was remarkably lower than WKY-SED (P < 0.05), while SBP was much higher than WKY-SED (P < 0.05). 2) PDBu (PKC activator) elicited a tension increase, and Gö6976 (PKC inhibitor) induced vasodilation. Both the responses of PDBu and Gö6976 in SHR-SED were notably increased compared with WKY-SED (both P < 0.05), however, exercise training significantly suppressed these increases (both P < 0.05). 3) Nifedipine (CaV1.2 inhibitor) induced vasodilation. Response to nifedipine in SHR-SED was more sensitive than both SHR-EX and WKY-SED (both P < 0.05). 4) The current density of SHR-SED and WKY-EX exhibited an increase compared to the WKY-SED (both P < 0.05), and the current density of the SHR-EX decreased obviously in contrast with SHR-SED (P < 0.05). Besides, PDBu enlarged current density of all the groups, while Gö6976 decreased current density. The increase or decrease amplitude in SHR-SED was significantly higher than WKY-SED (both P < 0.05), whereas exercise training markedly inhibited those responses (both P < 0.05).

Conclusions Aerobic exercise efficiently prevents the upregulation of PKC/CaV1.2 pathway in hypertension, and enhances the function of vascular smooth muscle.