EXERCISE TRAINING ATTENUATES THE PROGRESSION OF ENDOTHELIAL DYSFUNCTION AND ARTERIAL CALCIFICATION INDUCED BY VITAMIN D(3) PLUS NICOTINE TREATMENT IN OVARIECTOMIZED RATS
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Backgrounds The increases in arterial stiffness and calcification are causal factors of atherosclerosis. The risks in these factors are dramatically elevated by loss of estrogen synthesis capacity after menopause in females. Exercise training may improve aging-induced attenuation of endothelial function, resulting in less arterial stiffness or calcification (1, 2). However, an underlying mechanism of exercise training-induced improvement of arterial stiffness and calcification remains unclear. Endothelin-1 (ET-1), an endothelium-derived vasoconstrictor peptide, is a regulator of arterial calcification and endothelial function (3). We hypothesized that exercise training after menopause in females would improve the progression of endothelial dysfunction and arterial calcification and its improvement may participate in the decrease in ET-1 levels.

Methods We tested to our hypothesis by using thoracic aortas of sham-operated rats (Sham Control; SC), ovarie-tomized (OVX) rats (OVX Control; OC), OVX plus treatment with vitamin D(3) plus nicotine (VDN) rats (OV Sedentary; OVSe), which is a model animal of an endothelial dysfunction and arterial calcification, and voluntary running wheel exercise for 8 weeks with OVX plus VDN rats (OV Exercise; OVEx).

Results In the result of an arterial tissue stain, OVSe induced arterial calcification, but not in OVEx. The arterial calcium and ET-1 levels were significantly higher in the OVSe compared with the SC and OC, whereas these levels in the OVEx were significantly lower than in the OVSe. Additionally, arterial eNOS expression, which is an enzyme to produce nitric oxide (NO: a vasodilator substance), was reduced in OVSe. However, the eNOS level was increased by exercise training. Moreover, there was a significant positive correlation between arterial calcium levels and arterial ET-1 levels (r = 0.677, p < 0.0001).

Conclusions These findings suggest that a mechanism of exercise training-induced an improvement in the progression of endothelial dysfunction and arterial calcification in OVX plus VDN model rats participates in the decrease in ET-1 levels and elevation of eNOS expression in the aorta.

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