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Student Sessions

Influence of long-term exposure of advanced glycation endproducts on vascular contraction in rat carotid artery

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Although there are several reports suggested that advanced glycation end-products (AGEs) cause vascular endothelial dysfunction, the direct relationship between AGEs and smooth muscle contractile function remains unclear. Therefore, we investigated the long-term effects of AGE-bovine serum albumin (AGE-BSA) on contractile responses in rat carotid arterial rings using organ-culture technique. After exposure of AGE-BSA (0.001-0.1 mg/mL) for approximately 1 day in carotid artery, concentration-response curves were investigated under endothelium denuded artery. Contractile responses of high K^+ or serotonin did not alter among groups treated with and without AGE-BSA. Treatment with AGE-BSA (0.1 mg/mL) (vs. control; PBS) increased thromboxane A_2 analog-induced contraction, whereas decreased noradrenaline-induced contraction. The decreased noradrenaline-induced contraction by AGE-BSA was prevented by co-treated with organic cation transporter-3 (OCT-3) inhibitor corticosterone. The protein expression of OCT-3 in endothelium-denuded carotid artery was similar between control and AGE-BSA groups. These results suggest that ligand specific alterations of contractile responses by AGE-BSA exposure were seen in carotid arteries, and that decreased noradrenaline-induced contraction by AGE-BSA may be partly due to increased OCT-3 activity rather than the expression.