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Water-soluble chitosan regulates vascular remodeling in hypertension via NFATc1

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Objective: Hypertension is a public health problem in the world, and the disability and mortality rate is extremely high. Its important pathology foundation is vascular remodeling. Water-soluble chitosan (WSC) is the degradation product of chitosan, and have a role to control hypertension. The present study aims to investigate the regulatory effects of WSC on vascular remodeling in hypertension, and further to confirm the roles of nuclear factor c1 of activatied T cells (NFATc1) during this effect.
Methods: Primary cultured rat abdominal aortic smooth muscle cells were incubated with PBS, AngII, and AngII + WSC (0.1 mg/L) for 24 h. MTT and western blot methods were applied to analyzed the cell proliferation and c-myc protein expression, respectively, among all experimental groups. Successfully established spontaneously hypertensive Wistar-kyoto rats (SHR) were divided into two groups randomly: SHR group (n = 30) and SHR + WSC group (n = 30,WSC150 mg/kg/d). Another 15 Wistar-kyoto rats treated with PBS were served as control group. At the end of the experiments, the hemodynamic changes were analyzed using rat tail arterial pressure measuring instrument. H&E staining was performed to observe the morphological changes of abdominal aorta. Furthermore, immunohistochemical method, western blot and real-time quantitative PCR were applied to detected the expression of NAFTc1 protein and mRNA.
Results: WSC significantly reduced the cell viability in primary cultured rat abdominal aortic smooth muscle cells compare with PBS and AngII-treated cells. Compared with PBS group, the enhanced expression level of c-myc protein was observed in AngII-treated cells, which was significantly blocked by WSC incubation. Compared with control rats, the abnormally high blood pressure and membrane thickness/lumen diameter ratio of abdominal aorta were noted in SHR model rats, which strongly reduced after WSC administration. Hypertension resulted in an increment on expression of both NFATc1 protein and mRNA in abdominal aorta of Wistar-kyoto rats compared with control groups. Encouragingly, WSC strikingly suppressed the high levels of NFATc1 protein and even mRNA.
Conclusion: In both cell and animal experiments, we succefully confirmed the regulatory effects of WSC on vascular remodeling in hypertension. Based on the present results, WSC-inhibited vascular remodeling may be related to the modulation on NFATc1 expression. Our experiment provides a solid basis for the clinical application of WSC on hypertension.

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