Effects of ginsenoside Rg1 on the senescence of vascular smooth muscle cells

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ABSTRACT. The development of age-related cardiovascular disease is associated with the senescence of vascular cells. This study aimed to investigate the effect of ginsenoside Rg1 on vascular smooth muscle cell (VSMC) senescence. Primary VSMCs were cultured and divided into control, D-galactose (D-gal), Rg1-L, and Rg1-H groups, which were cultured without and with D-gal, and with low- and high-concentrations of Rg1, respectively. D-gal-induced cellular senescence was identified by β-galactosidase staining, and ultrastructural changes within the cells were observed. The expression of p16, p21, and p53 in the four groups of VSMCs was determined by western blotting, and the cell cycle was investigated by flow cytometry. Compared with the control group, there was an obvious change in the ultrastructure of VSMCs in the D-gal group, and the proportion of β-galactosidase-positive cells was significantly increased ($P < 0.05$). In addition, p16, p21, and p53 expression was significantly increased ($P < 0.05$) and the cell cycle was arrested in the G0/G1 phase. Compared with the D-gal

group, the percentage of positive cells was significantly reduced ($P < 0.05$) in the Rg1 groups, the expression of p16, p21, and p53 was significantly reduced ($P < 0.05$), and the number of cells in the G0/G1 phase decreased ($P < 0.05$). Ginsenoside Rg1 can inhibit VSMC senescence, and the mechanisms may be related to its partial inhibition of the $p16^{INK4a}$/Rb and p53-$p21^{Cip1/Waf1}$ signaling pathways during the cell cycle.

Key words: Vascular smooth muscle cells; Senescence; Ginsenoside Rg1; Signaling pathway