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EFFECTS OF SUPPRESSOR OF CYTOKINE SIGNALLING-1 ON PROLIFERATION INDUCED BY OXIDATIVE LOW-DENSITY LIPOPROTEIN IN VASCULAR SMOOTH MUSCLE CELLS

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Objectives Excessive proliferation vascular smooth muscle cells (VSMC) can promote the development of atherosclerosis.And, in the development of atherosclerosis, oxidative low-density lipoprotein (oxLDL) enhance excessive proliferation of VSMC.As studies have showed, down-regulation of suppressor of cytokine signalling-1 (SOCS1)could induce apoptosis of a variety of cells. However, it has not been reported whether there is any effect of SOCS1 on proliferation and apoptosis of VSMC. To investigate effects of SOCS1on proliferation induced by oxLDL in human VSMC and its association with atherosclerosis, this study was carried out.

Methods

(1) SiRNA targeting SOCS1 gene (SOCS1-siRNA) and negative control siRNA (NC-siRNA) were transfected into VSMC by liposome, and these cells were incubated with oxLDL at a 100 μg/ml concentration for 48 h. The experiments were divided into six groups: blank control group; NC-siRNA group; SOCS1-siRNA group; oxLDL group; oxLDL+NC-siRNA group; oxLDL+SOCS1-siRNA group.
(2) Through RT-PCR and Western blot, the expression levels of SOCS1, Bcl-2, Bax and wild type p53 (wt-p53) were detected.
(3) The cell growth was detected by MTT.
(4) The cell cycle and apoptosis rate were examined with flow cytometry.

Results

1. RT-PCR results and western blot results showed that the transfection of SOCS1-siRNA increased the expression of Bax and wt-p53 ($p<0.05$), decreased the expression of SOCS1 and Bcl-2 ($p<0.05$ or $p<0.01$), and reduced the effects that oxLDL decreased the expression of Bax and wt-p53, increased the expression of Bcl-2($p<0.05$ or $p<0.01$).
2. MTT results showed that down-regulation of SOCS1 inhibited the cell growth ($p<0.05$), and reduced the positive effects of oxLDL on VSMC growth ($p<0.05$).
3. Flow cytometry results showed that down-regulation of SOCS1 increased the apoptosis rate of VSMC ($p<0.01$) and the cell amounts of G0/G1 phase ($p<0.05$), decreased the cell amounts of S phase ($p<0.05$), and reduced the effects that oxLDL decreased the apoptosis rate of VSMC and the cell amounts of

G0/G1 phase, increased the cell amounts of S phase (P respectively<0.01).

Conclusions Down-regulation of SOCS1 can inhibit VSMC proliferation, promote VSMC apoptosis, and reduce the effects that oxLDL promotes VSMC proliferation and inhibits VSMC apoptosis. Therefore, SOCS1 gene is likely to be potential molecular target for treatment of atherosclerosis.