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EFFECTS OF XERODERMA PIGMENTOSUM B GENE ON PROLIFERATION AND APOPTOSIS CONDUCTED BY INTERLEUKIN-6 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, interleukin-6 (IL-6) enhance excessive proliferation of VSMC. As studies have showed, up-regulation of xeroderma pigmentosum B (XPB) gene could induce apoptosis of a variety of cells. However, it has not been reported whether there is any effect of XPB on proliferation and apoptosis of VSMC. To investigate effects of XPB on proliferation induced by IL-6 in human VSMC and its association with atherosclerosis, this study was carried out.

Methods

1. Recombinant plasmid pcDNA3.1-XPB and vacant vector plasmid pcDNA3.1 were transfected stably into VSMC by liposome, and these cells were incubated with IL-6 at a 100 U/ml concentration for 48 h. The experiments were divided into six groups: blank control group; pcDNA3.1 group; pcDNA3.1-XPB group; IL-6 group; IL-6+pcDNA3.1 group; IL-6+pcDNA3.1-XPB group.
2. Through RT-PCR and Western blot, the expression levels of XPB, Bcl-2, Bax and wild type p53 (wt-p53) were detected.
3. The cell growth was detected by MTT. 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 pcDNA3.1-XPB increased the expression of XPB, Bax and wt-p53 ($p < 0.05$ or $p < 0.01$), decreased the expression of Bcl-2 ($p < 0.05$ or $p < 0.01$), and reduced the effects that IL-6 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 overexpression of XPB inhibited the cell growth ($p < 0.05$), and reduced the positive effects of IL-6 on VSMC growth ($p < 0.05$).
3. Flow cytometry results showed that overexpression of XPB increased the survival 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 IL-6 decreased the survival rate of VSMC and the cell amounts of G₀/G₁ phase, increased the cell amounts of S phase (P respectively < 0.01).

Conclusions XPB gene can inhibit VSMC proliferation, promote VSMC apoptosis, and reduce the effects that IL-6 promotes VSMC proliferation and inhibits VSMC apoptosis. Therefore, XPB gene is likely to be potential molecular target for treatment of atherosclerosis.