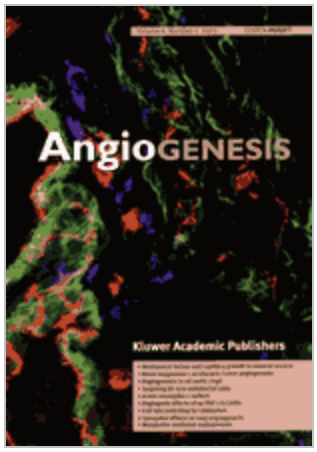
Article



Angiogenesis

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Inhibition of B16 Melanoma Growth *in vivo* by Retroviral Vector-Mediated Human Ribonuclease Inhibitor

Ting Wang[1], Mingjie Yang[1], Junxia Chen[1], Tonya Watkins[2] and Cui Xiuyun[1]

(1) Department of Biochemistry and Molecular Biology, Dalian Medical University, Dalian, 116027, China

(2) Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

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Abstract Human ribonuclease inhibitor (hRI) can inhibit angiogenesis by reversibly binding angiogenin, a member of the RNaseA superfamily, and by suppressing the expression of basic fibroblast growth factor (bFGF). Angiogenesis is necessary for the growth and metastasis of tumors. To study the links between hRI, angiogenesis, and melanoma growth, the hRI gene was intravenously administered to mice in a recombinant retroviral vector, and expression of the hRI gene was induced to block melanoma angiogenesis. Expression, distribution, and contribution of the target gene in mice were assayed. The results showed that the tumors of mice in the hRI treatment group grew slower with less vascularity than those of mice in control groups. The introduced hRI gene inhibited tumor growth without causing significant side effects in the animals. More hRI expression in vimentin-positive cells of the tumor than in melanoma cells suggested that mesenchymal cells in the fibrous envelope of the tumor play important roles in this gene therapy.

Key words anti-angiogenesis - B16 melanoma - gene therapy - retroviral vector - ribonuclease inhibitor

Cui Xiuyun

Email: cuixy@dlmedu.edu.cn
Phone: +86-411-84720648
Fax: +86-411-84672153

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