Cite this as: Yuan LI, Hai-tao REN, 2017. Endostatin inhibits fibrosis by modulating the PDGFR/ERK signal pathway: an in vitro study. *Journal of Zhejiang University-Science B (Biomedicine & Biotechnology)*, 18(11):994-1001.
http://dx.doi.org/10.1631/jzus.B1700052

Endostatin inhibits fibrosis by modulating the PDGFR/ERK signal pathway: an in vitro study

Keywords: Endostatin, Hypertrophic scar, Phosphorylated platelet-derived growth factor receptor (p-PDGFR), Extracellular signal-regulated kinase (ERK), Signal pathway

Research Summary

In this study, we evaluated the effect of endostatin on fibrosis by using PDGF-BB- (Platelet derived growth factor, PDGF) and TGF-β_1 - (transforming growth factor-β, TGF-β) induced human skin fibroblasts fibrosis models and further elucidated its underlying mechanisms.

- We found that endostatin could alleviate human skin fibroblast fibrosis via inhibition of the PDGFR/ERK signal pathway. Our study demonstrated the potential of endostatin as a potent drug for the treatment of hypertrophic scar and skin fibrosis disease.

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Innovation points

We investigated the effect of endostatin on fibrosis and elucidated the potential mechanisms of modulating PDGFR/ERK signal pathway for the first time.

1. Endostatin significantly attenuated both the PDGF-BB- and TGF-β_1- induced over-expression of collagen I, hydroxyproline, and α-SMA.
2. PDGF-BB and TGF-β_1 both promoted the expression of PDGFR, ERK and p-ERK
3. . Endostatin inhibited the expression of PDGFR and p-ERK but did not affect the expression of total ERK.

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Figure 4