Catalpol suppressed proliferation, growth and invasion of CT26 colon cancer by inhibiting inflammation and tumor angiogenesis

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Tumor angiogenesis and inflammation, which play important roles in mediating tumor proliferation and growth, should be inhibited to effectively regulate tumor progression. Catalpol, a main active ingredient extracted from Rehmannia glutinosa, has various pharmacological actions including anti-apoptotic, anti-inflammatory, hypoglycemic and anti-cancer properties. However, the pharmacological effect of catalpol on colon cancer remains largely unknown. The aim of this study was to investigate the effects of catalpol on the proliferation, growth, invasion, tumor angiogenesis and inflammation of CT26 colon cancer in vitro and in vivo. Catalpol inhibited the proliferation and growth of CT26 cells in concentration- and dose-dependent manners in vitro and in vivo, respectively. Catalpol suppressed the invasion of CT26 cells in vitro. Tumor cell-induced vascularization of endothelial cells and rat aortic ring angiogenesis were impaired by catalpol. Catalpol reduced the secretions of several angiogenic markers in the culture supernatant of CT26 cells. Immunohistochemical assay showed that catalpol inhibited the expressions of angiogenic markers vascular endothelial growth factor (VEGF), vascular endothelial growth factor receptor 2 (VEGFR2), hypoxia inducible factor-1a (HIF-1a) and basic fibroblast growth factor (bFGF) in colon cancer tissues. Moreover, catalpol inhibited the expressions of inflammatory factors interleukin (IL), IL-6, IL-8, cyclooxygenase (COX-2) and inducible nitric oxide synthase (iNOS).

Taken together, catalpol suppressed the growth and invasion of CT26 colon cancer cells mainly by inhibiting inflammation and tumor angiogenesis, as a promising candidate compound for treating colon cancer.