Z304 Anti-angiogenic activities of tabanus and cnidii rhizoma

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This study investigated the anti-angiogenic activities of cnidii rhizoma and tabanus by using cultured glomerular capillary endothelial (GEC) and cancer cells, chorioallantoic membrane (CAM) and rat corne-a. Teatment of GECs with several concentrations (5-50 μg /ml) of cnidii rhizoma and tabanus extracts for 24 h ingibited angiotenisin II (10-8M)induced increases of [3H]thymidine uptake and cell numbers in a dosedependent Herbal extracts also inhibited the proliferation of 4 different types of cancer cells, and the order of inhibitory ratio was ECV 304 (endothelial carcinoma cells) SNU 638(gastric carcinoma cells)> HL-60 (promyelocytic leukemia cells) SNU 449 (hepatic carcinoma cells). In contrast to the normal branching of vascular vessels, blood vessel patterns in CAMs treated with extracts (50μg/egg) of cnidii rhizoma and tabanus were ran parallel to each other without much branching. Moreover, oral administration of herbal extracts (20 mg/kg body weight/day) for 4 weeks significantly inhibited the rat corneal cularization induced by suture, and the length of blood vessels in herbal medicine treated rat cornea was conspicuously lower than that in control animals.

Z305 Role of FGF signaling pathway in the ascidian embryo

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In the ascidian embryo, a fibroblast growth factor (FGF)-like signal from endoderm precursors between the 32- and early 64-cell stages induces the formation of notochord and mesenchyme cells. However, it has not been known whether endogenous FGF signaling is involved in the process. Here we show that 64-cell embryos exhibited a marked increase in endogenous MAP kinase activity. The increase in MAP kinase activity was reduced by treatment with an FGF receptor inhibitor, SU5402, and a MAP kinase kinase inhibitor, U0126. Both drugs blocked the formation of notochord and mesenchyme when embryos were treated at the 32-cell stage, but not at the 2- or 110-cell stages. The dominant negative mutant of Ras also suppressed the formation. Both inhibitors suppressed the induction by exogenous basic FGF. These results indicate that the FGF signaling cascade is necessary for the formation of notochord and mesenchyme cells in the ascidian embryonic development. We also show that FGF signaling is required for formation of the secondary notochord, secondary muscle, and neural tissues, and at least MAP kinase activity is necessary for the formation of trunk lateral cells and posterior endoderm.