Alteration in Distribution of Focal Adhesion Components by Wortmannin in Hepatic Stellate Cell Culture on Type I Collagen Gel
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Aim: To investigate the mechanism by which hepatic stellate cells (HSCs) cultured on interstitial collagen gel are induced to extend long cellular processes, we examined the effects of phosphatidylinositol (PI) 3-kinase inhibitor, wortmannin, on the distribution and level of several components of focal adhesion.

Materials and Methods: Human HSCs were cultured overnight on type I collagen gel, and then incubated for up to 30 min in the presence of 100 nM wortmannin. Cells were immuno-fluorescently stained for vinculin, talin, α-actinin, Rho, or tyrosine-phosphorylated proteins. F-actin was stained using FITC-phalloidin. Stained cells were analyzed by using confocal laser scanning microscopy. The levels of focal adhesion components were analyzed by immunoblotting.

Results: HSCs cultured on type I collagen gel were induced to elongate many long cellular processes, but not in the presence of wortmannin. Once elongated cellular processes were retracted by the addition of wortmannin and the distribution and level of focal adhesion components were also altered by the wortmannin-treatment.

Discussion: Inhibition of cellular process extension and retraction of elongated cellular processes by wortmannin, as well as herbimycin A or staurosporin (our previous reports), suggest that continuous signal transduction processes including protein and PI phosphorylation are necessary to induce and maintain long cellular process architecture in HSC culture.

The affinity of type II collagen for heparin compared to that of type I and type V collagens.
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