

**P031** Role of RhoC in motility and cervical carcinoma  
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RhoC, a Ras homologue, regulates actin cytoskeletal organization in cells. Reports implicate RhoC in metastasis of carcinomas including breast and lung. Progression of carcinogenesis involves epithelial-to-mesenchymal transition (EMT), increased motility and invasiveness. Since RhoC regulates actin organization and cell motility we investigated the role of RhoC activity in cervical carcinoma.

Immunohistochemical analysis of 30 patient samples showed increased cytosolic staining of RhoC in carcinoma tissue sections in contrast to very mild expression levels in normal tissue samples. Semi-quantitative RT-PCR on cervical cancer cell lines including SiHa and CaSki showed higher RhoC levels as compared to RhoB and RhoA. Wound healing assays with atorvastatin, inactive-RhoC and PI3K inhibitor, blocked wound healing as compared to untreated cells. The effective concentration of atorvastatin and LY294002 on EMT was observed to be different in both the cell lines. Results suggest that RhoC induced motility may be regulated in these cell lines by different signaling mechanisms. TGF-beta induced EMT, in CaSki and SiHa, could be blocked by expressing inactive-RhoC and atorvastatin, suggesting the presence of RhoC downstream of TGF-beta signaling. The addition of inactive-RhoC to TGF-beta treated cells inhibited actin stress filament formation and upregulated E-cadherin. These results collectively indicate that RhoC may be a potential metastatic factor in cervical carcinoma progression.