**p130 Mediates TGF-β-induced Cell-Cycle Arrest in Cervical Carcinoma Cells**

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**Background** The transforming growth factor-β (TGF-β) signaling pathway exerts an essential tumor suppressor function in various cell types. However, it has never been reported that TGF-β regulates the cellular proliferation in cervical cancer cells.

**Methods** The responses of 4 cervical carcinoma cancer cell lines (HT-3, CaSki, HeLaS3, and ME-180) by TGF-β were examined. Change of cell cycle distribution was analyzed, and the level of each protein and its association were assayed by Western blotting analysis and immunoprecipitation.

**Results** In the present study, we analyzed the effect of TGF-β on cervical carcinoma cell lines. TGF-β inhibited the proliferation of HT-3 cells expressing mutant Rb protein in a time-dependent manner. TGF-β (5 ng/ml) efficiently induced G1 arrest of the cell cycle. Protein level of p21 was increased in a time-dependent manner, but other G1 regulatory protein levels were not changed. TGF-β markedly enhanced the binding of p21 with cdk2 and decreased that of cdk2 with cyclin E. TGF-β inhibited the phosphorylation of p130 but did not change Rb and p107 protein status. We found that E2F-1 protein level was decreased in a time-dependent manner in TGF-β treated cells, and it might be resulted from the enhanced binding of E2F-4 with p130.

**Conclusion** Our results demonstrate that not Rb but p130 can mediate growth inhibition by TGF-β in Rb mutant HT-3 cells.