Supression of the STK15 oncogenic activity requires a transactivation-independent P53 function.

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Abstract

Using a transactivation-defective p53 derivative as bait, STK15, a centrosome-associated oncogenic serine/threonine kinase, was isolated as a p53 partner. The p53–STK15 interaction was confirmed further by co-immunoprecipitation and GST pull-down studies. In co-transfection experiments, p53 suppressed STK15-induced centrosome amplification and cellular transformation in a transactivation-independent manner. The suppression of STK15 oncogenic activity by p53 might be explained in part by the finding that p53 inhibited STK15 kinase activity via direct interaction with the latter's Aurora box. Taken together, these findings revealed a novel mechanism for the tumor suppressor function of p53.

Keywords: Aurora box/centrosome amplification/oncogenic kinase/p53/STK15