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Transcriptional Activation of the Cdk Inhibitor p27 during the Differentiation of Human Neuroblastoma Cell Line SH-SY5Y

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p27, an inhibitor of cyclin-dependent kinases(Cdks), plays a role in cell cycle exit by TGF-β, cell-cell contact or differentiation. p27 protein markedly accumulated in quiescent cells compared with proliferating cells, and prevented the activation of cyclin E-Cdk2 complexes. The expression of p27 protein has been found to be regulated mainly post-transcriptionally by translational control and ubiquitin-proteasome pathway in a variety of cells. In this study, we investigated the regulation of p27 expression in SH-SY5Y human neuroblastoma cells that undergo differentiation in response to retinoic acid and BDNF. During differentiation, an accumulation of p27 protein occurred 48 hr following the treatment. Unexpectedly, p27 mRNA significantly increased 24 hr prior to the accumulation of p27 protein. These results suggest that the expression of p27 is regulated by transcriptional activation during the differentiation of human neuroblastoma cells, in addition to the post-transcriptional controls.

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NF-κB Activation Induced by Disruption of Microtuble Network during Myogenesis

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Recently, we have found that NF-κB is involved in an early stage of skeletal muscle differentiation. However, the mechanism that leads to NF-κB activation during myoblast differentiation is yet to be elucidated. One of the early events in myogenic differentiation is morphological changes accompanied by massive cytoskeletal reorganization. Here we show that the activity of NF-κB markedly increased in the L6 rat myogenic cells that just initiated morphorogical changes and then gradually declined thereafter. In addition, a variety of agents that depolymerize microtubules stimulated differentiation as well as the activation of NF-κB in the L6 cells. On the other hand, a microtubule-stabilizing agent, taxol, reversed the effects of microtubule depolymerizing agents and also inhibited the spontaneous differentiation of the L6 cells. These results suggest that the reorganization of cytoskeleton in myoblast acts as a signal for NF-κB activation.