Effect of NDC80 in human hepatocellular carcinoma

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1 What did this study explore?

The aim of this study was to investigate the role of NDC80 in human hepatocellular carcinogenesis.

2 How did the authors perform all experiments?

NDC80 gene expression was analyzed by real-time PCR (RT-PCR) in 47 paired HCC tissues and adjacent tissues. The HCC cell line SMMC-7721 was lentivirally transfected to silence endogenous NDC80 gene expression, which was confirmed by RT-PCR and western blotting analysis. The effects of NDC80 silencing on SMMC-7721 cell proliferation were evaluated by Cellomics ArrayScan VTI imaging. Cell cycle analysis and cell apoptosis were detected with flow cytometric technique. Colony formation was assessed by fluorescence microscopy.

3 How did the authors process all experimental data?

The GraphPad Prism software version 6.0 was used for data analysis. Statistical significance was defined as $P<0.05$. All data were presented as the mean ± standard deviation (SD). All the experiments were repeated at least three times.

4 How did the authors deal with the pre-study hypothesis?

They monitored the lentiviral infection efficiency by fluorescence microscope, and confirmed the target gene knockdown by western blot and real-time PCR, which provided a basis for the continued observation of the role of NDC80 in SMMC-7721cells. Real-time PCR analysis showed that the NDC80 expression levels in the tumor tissues were significantly increased compared with those in the adjacent tissues. They found that cell proliferation and cell colony formation were significantly inhibited in NDC80-silenced HCC cells. Moreover, apoptosis was significantly increased in NDC80-silenced HCC cells. They performed cell cycle assay to illustrate the mechanism by which NDC80 promotes cell proliferation. They found that attenuation of NDC80 expression in carcinoma cells delayed cell cycle progression through S phase of the cell cycle signifying cell arrest at S phase. This suggested that abnormal NDC80 expression might lead to severe disruption of cell cycle progression. The present study demonstrated that NDC80 knockdown induced the HCC cell apoptosis. The apoptosis caused by NDC80-knockdown might be due to incompletion of mitosis caused by severe mitotic spindle checkpoint dysfunction

5 What are the novel findings of this study?

This is the first study that NDC80 contributes to the pathogenesis of HCC through its proliferative and antiapoptotic effects. The critical role of NDC80 in HCC development could provide evidence for development of novel therapeutics against NDC80 for the early detection and treatment of HCC.