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Molecular mechanism of tumorigenesis induced by FUSE binding protein 1: A potential target for anticancer drug development

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Persistent Hepatitis C virus (HCV) infection leads to chronic hepatitis C (CHC), which often progresses to liver cirrhosis (LC) and hepatocellular carcinoma (HCC). We recently identified an oncogenic cellular factor, FUSE binding protein 1 (FuBP1), which antagonizes the function of tumor suppressor p53 and promotes persistent HCV replication and associated pathogenesis. We found that the direct target of FuBP1 in cancer cells is the tumor suppressor p53, the function of which is actively suppressed by FuBP1. Knockdown of FuBP1 in cancer cells significantly activates p53, increases their sensitivity to apoptotic stimuli, and drastically reduces cell proliferation and migration. We found that FuBP1 physically interacts with the wild-type p53 as well as all the p53 isoforms found in cancers. FuBP1 interaction with p53 strongly inhibits the target DNA binding function of p53. Mapping of the FuBP1-interaction site on p53 molecule indicated DNA binding domain of p53 as the site of FuBP1 interaction. Since FuBP1 expression is undetectable in normal differentiated cells, its overexpression in most cancers including HCC suggests that it is a potential target for drug development.

Biography

Virendra N Pandey received his PhD in 1985 from the University of Mumbai at Bhabha Atomic Research Center (BARC), India. He served as Professor (Scientist-F) at BARC until 1994 and received the most prestigious Shanti Swaroop Bhatnagar Award in 1991 for his research contribution in Life Sciences. He has published more than 90 research articles in reputed peer reviewed journals and has received continuous research funding from the National Institute of Health for the past 20 years. Currently, he is tenured senior faculty and a member of the Rutgers University Senate.

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