P86

Opposite Effects of Hepatitis B Virus X Protein on the Transcription of $p21^{waf1/cip1}$ Depending on the Status of Cellular p53[1]

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Despite of the extensive studies on the roles of hepatitis B virus-X protein (HBx) in the hepatocarcinogenesis, the mechanisms by which HBx contributes to HCC remain controversial. In this study, we investigated the effect of HBx on the transcription of $p21^{waf1/cip1}$ depending on the status of p53. Transcription of the $p21^{waf1/cip1}$ was activated by HBx in the presence of functional p53 by a p53-dependent manner. However, it was repressed by HBx via a p53-independent pathway when p53 was absent or present in a low level. Furthermore, growth rate of the HBx-expressing NIH3T3 cell lines compared to that of the parent cells was approximately two fold faster but more rapidly decreased when p53 was up-regulated by the cisplatin treatment. These results indicate that the status of p53 might determine whether HBx induces cell death or stimulates cell growth.