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The Viral Oncoprotein HBx of Hepatitis B virus Promotes the Growth of Hepatocellular Carcinoma through Cooperating with the Cellular Oncoprotein RMP

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Abstarc :

The smallest gene HBx of Hepatitis B virus (HBV) is recognized as an important viral oncogene (V-oncogene) in the hepatocarcinogenesis. Our previous work demonstrated that RMP is a cellular oncogene (C-oncogene) required for the proliferation of hepatocellular carcinoma (HCC) cells. Here we presented the collaboration between V-oncogene HBx and C-oncogene RMP in the development of HCC. The coexpression of HBx and RMP resulted in the cooperative effect of antiapoptosis and proliferation of HCC cells. In vivo, overexpression of RMP accelerated the growth of HBx-induced xenograft tumors in nude mice and vice versa HBx promoted the growth of RMP-driven xenograft tumors. Although HBx didn't regulate the expression of RMP, HBx and RMP interact with each other and collocalized in the cytoplasm of HCC cells. HBx and RMP collaboratively inhibited the expression of apoptotic factors and promoted the expression of antiapoptotic factors. This finding suggests that HBV may induce, or at least partially contributes to the carcinogenesis of HCC, through its V-oncoprotein HBx interacting with the C-oncoprotein RMP.

Key Word :
HBx, viral oncogene

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