HBXIP

Hepatitis B virus X-interacting protein is a protein that in humans is encoded by the HBXIP gene.

This gene encodes a protein that specifically complexes with the C-terminus of hepatitis B virus X protein (HBx). The function of this protein is to negatively regulate HBx activity and thus to alter the replication life cycle of the virus.

There is abundant evidence to indicate that HBXIP functions as an oncoprotein and transcription co-activator during the development and promotion of cancers. In multiple cancers, ZEB1 serves as a transcription activator to regulate gene expression.

Jiang et al. explored the roles of ZEB1 in HBXIP-induced breast cancer growth.

HBXIP regulation of ZEB1 was evaluated by reverse transcription PCR and immunoblotting. The stimulation of ZEB1 promoter by HBXIP and/or Sp1 was tested using luciferase reporter gene analysis. The alteration of cell proliferation mediated by HBXIP-induced ZEB1 was tested using methyl-thiazolyl-tetrazolium and 5-Ethynyl-2'-deoxyuridine (EdU) incorporation analysis. ZEB1 and HBXIP expression in human breast cancer tissues was analyzed using quantitative real-time PCR. The relationship between HBXIP and ZEB1 was confirmed by Pearson's correlation coefficient.

They observed dose-dependent upregulation of ZEB1 by HBXIP in breast cancer cells. HBXIP can activate the ZEB1 promoter by interacting with transcription factor Sp1. Cell viability and EdU incorporation analysis showed that HBXIP could drive cell proliferation by enhancing ZEB1 in breast cancer. Using quantitative real-time PCR, ZEB1 overexpression and a positive relationship between ZEB1 and HBXIP were observed in clinical breast cancer samples.

Oncogenic HBXIP controls the transcription regulation of ZEB1 by co-activating Sp1, thereby accelerating breast cancer growth [1].

Interactions

HBXIP has been shown to interact with NCOA6.

1)
Jiang Y, Wang D, Ren H, Shi Y, Gao Y. Oncogenic HBXIP enhances ZEB1 through Sp1 to accelerate breast cancer growth. Thorac Cancer. 2018 Oct 1. doi: 10.1111/1759-7714.12878. [Epub ahead of print] PubMed PMID: 30273966.