

32-13592: HBV-X

Alternative Name :

Hepatitis B virus X protein (HBx) is a 17 kD transcriptional coactivator that plays a significant role in the regulation of genes involved in inflammation and cell survival. It regulates many transcription factors including nuclear factor kappa B (NF-kappaB) and plays a key role in hepatocarcinogenesis. rHBx facilitates the binding of cAMP response element binding protein (CREB) to its responsive element. rHBx stabilizes the cellular coactivator ASC-2 through direct protein-protein interaction, affecting the regulation of genes actively transcribed in liver cancer cells. HBx transactivates both JNK and MAPK signal transduction pathways in association with the mobilization of cytosolic Ca²⁺. The communication between HBx and general transcription factor TFIIB is also one of the mechanisms which account for its transcriptional transactivation. HBx decreased the expression of PTEN a known tumor suppressor and a negative regulator of phosphatidylinositol 3'-kinase/AKT and HBx decreased the expression of PTEN in HBx-transfected cells. The etiology of hepatocellular carcinoma (HCC) is involved with hepatitis B virus (HBV) infection and HBx in particular plays a role in the development of HBV-related HCC. The persistence of HBx is important to the pathogenesis of early HCC and HBx expression in the liver during chronic HBV infection may be an important prognostic marker for the development of HCC.

Description

Source: Escherichia Coli.

Hepatitis B virus X protein (HBx) is a 17 kD transcriptional coactivator that plays a significant role in the regulation of genes involved in inflammation and cell survival. It regulates many transcription factors including nuclear factor kappa B (NF-kappaB) and plays a key role in hepatocarcinogenesis. rHBx facilitates the binding of cAMP response element binding protein (CREB) to its responsive element. rHBx stabilizes the cellular coactivator ASC-2 through direct protein-protein interaction, affecting the regulation of genes actively transcribed in liver cancer cells. HBx transactivates both JNK and MAPK signal transduction pathways in association with the mobilization of cytosolic Ca²⁺. The communication between HBx and general transcription factor TFIIB is also one of the mechanisms which account for its transcriptional transactivation. HBx decreased the expression of PTEN a known tumor suppressor and a negative regulator of phosphatidylinositol 3'-kinase/AKT and HBx decreased the expression of PTEN in HBx-transfected cells. The etiology of hepatocellular carcinoma (HCC) is involved with hepatitis B virus (HBV) infection and HBx in particular plays a role in the development of HBV-related HCC. The persistence of HBx is important to the pathogenesis of early HCC and HBx expression in the liver during chronic HBV infection may be an important prognostic marker for the development of HCC.

Hepatitis B Virus Protein X is a 17kDa protein containing 154 amino acid residues and purified by proprietary chromatographic techniques.

Product Info

Amount :	2 µg / 10 µg
Purification :	Greater than 95% as determined by SDS-PAGE. Filtered (0.4µm) and lyophilized from 0.5mg/ml in 30mM acetate buffer pH4 and 5% trehalose. It is recommended to add 0.1M Acetate buffer pH4 to prepare a working stock solution of approximately 0.5mg/ml and let the lyophilized pellet dissolve completely. For conversion into higher pH value, we recommend intensive dilution by relevant buffer to a concentration of 10µg/ml. In higher concentrations the solubility of the HBV X antigen is limited. Filter sterilize your culture media/working solutions containing this non-sterile product before using in cell culture.
Content :	
Storage condition :	For long term storage lyophilized protein should be stored at -20°C. Aliquot the product after reconstitution to avoid repeated freezing/thawing cycles. Reconstituted protein can be stored at 4°C for a limited period of time; it does not show any change after two weeks at 4°C.
Amino Acid :	MAARVCCQLD PARDVLC LRP VGAESRGRP VSGPFGTL PSP SSSAVPADHG AHLSLRGLPV CAFSSAGPCA LRFTSARRME TTVNAHQVLP KVLHKRTLGL SAMSTTDLEA YFKDCLFKDW EELGEEIRLK VFVLGGCRHK LVCSPAPCNF FTSA.