

HBV X

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

Catalog #: HBPS-278

Source: Escherichia Coli.

For research use only.

Amino Acid Sequence: MAARVCCQLD PARDVLCLRP VGAESRGRPVGSGPFGTLPSPPSSSAVPADHG AHLSLRGLPV CAFSSAGPCA LRFTSARRME TTVNAHQVLP KVLHKRTLGL SAMSTTDLEA YFKDCLFKDW EELGEEIRLK VFVLGGCRHK LVCSPAPCNFFTSA.

Purity: Greater than 98% as determined by SDS-PAGE.

Formulation:

Filtered (0.4

Stability:

Store lyophilized protein 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.

Usage:

NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drugs, agricultural or pesticidal products, food additives or household chemicals.

Solubility:

It is recommended to add deionized water to prepare a working stock solution of 0.5mg/ml and let the lyophilized pellet dissolve completely. Product is not sterile! Please filter the product by an appropriate sterile filter before using it in the cell culture.

Introduction:

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.

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