Overview

Product name: Anti-Hepatitis B Virus X antigen antibody [3F6-G10]

Description: Mouse monoclonal [3F6-G10] to Hepatitis B Virus X antigen

Host species: Mouse

Specificity: HB-X antigen (17 kD). We have received mixed reports regarding how ab235 works in Western blot, and we are not able to guarantee this antibody in WB.

Tested applications: Suitable for: ELISA, IHC-P, IHC-Fr

Species reactivity: Reacts with: Hepatitis B virus

Immunogen: HB-Xag-Protein A Fusion protein

Properties

Form: Liquid

Storage instructions: Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.

Storage buffer: Preservative: 0.09% Sodium azide
Constituent: 0.1% BSA

Purity: Immunogen affinity purified

Purification notes: Purified IgG prepared by affinity chromatography on protein G from tissue culture supernatant.

Clonality: Monoclonal

Clone number: 3F6-G10

Myeloma: unknown

Isotype: IgG2a

Light chain type: unknown

Applications

Our Abpromise guarantee covers the use of ab235 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
Relevance
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. HBx facilitates the binding of cAMP response element binding protein (CREB) to its responsive element. HBx 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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