Anti-BAX Antibody [PSH0-20] - BSA and Azide free

HA610038

Product Type:	Recombinant Mouse monoclonal IgG1, primary antibodies
Species reactivity:	Human
Applications:	WB, IHC-P
Molecular Wt:	Predicted band size: 21 kDa
Clone number:	PSH0-20

Description: Apoptosis regulator BAX, also known as bcl-2-like protein 4, is a protein that in humans is encoded by the BAX gene. BAX is a member of the Bcl-2 gene family. BCL2 family members form hetero- or homodimers and act as anti- or pro-apoptotic regulators that are involved in a wide variety of cellular activities. This protein forms a heterodimer with BCL2, and functions as an apoptotic activator. This protein is reported to interact with, and increase the opening of, the mitochondrial voltage-dependent anion channel (VDAC), which leads to the loss in membrane potential and the release of cytochrome c. The expression of this gene is regulated by the tumor suppressor P53 and has been shown to be involved in P53-mediated apoptosis. In healthy mammalian cells, the majority of BAX is found in the cytosol, but upon initiation of apoptotic signaling, Bax undergoes a conformational shift. Upon induction of apoptosis, BAX becomes organelle membrane-associated, and in particular, mitochondrial membrane associated. BAX is believed to interact with, and induce the opening of the mitochondrial voltage-dependent anion channel, VDAC. Alternatively, growing evidence also suggests that activated BAX and/or Bak form an oligomeric pore, MAC in the MOM (mitochondrial outer membrane). This results in the release of cytochrome c and other pro-apoptotic factors from the mitochondria, often referred to as mitochondrial outer membrane permeabilization, leading to activation of caspases. This defines a direct role for BAX in mitochondrial outer membrane permeabilization. BAX activation is stimulated by various abiotic factors, including heat, hydrogen peroxide, low or high pH, and mitochondrial membrane remodeling. In addition, it can become activated by binding BCL-2, as well as non-BCL-2 proteins such as p53 and Bif-1. Conversely, BAX can become inactivated by interacting with VDAC2, Pin1, and IBRDC2.

Immunogen:	Synthetic peptide within Human Bax aa 1-50 / 192.
Positive control:	Human kidney tissue lysate, Daudi cell lysate, Raji cell lysate, HepG2 cell lysate, human liver carcinoma tissue.
Subcellular location:	Mitochondrion outer membrane, Cytoplasm.
Database links:	SwissProt: Q07812 Human
Recommended Dilutions:	
WB	1:5,000
IHC-P	1:200
Storage Buffer:	PBS (pH7.4).
Storage Instruction:	Store at +4℃ after thawing. Aliquot store at -20℃. Avoid repeated freeze / thaw cycles.
Purity:	Protein A affinity purified.

Images

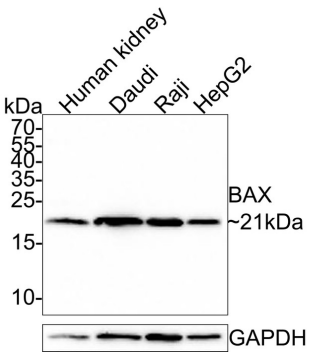

Fig1: Western blot analysis of BAX on different lysates with Mouse anti-BAX antibody (HA610038) at 1/5,000 dilution.

Lane 1: Human kidney tissue lysate (30 µg/Lane)
Lane 2: Daudi cell lysate (15 µg/Lane)
Lane 3: Raji cell lysate (15 µg/Lane)
Lane 4: HepG2 cell lysate (15 µg/Lane)

Predicted band size: 21 kDa
Observed band size: 21 kDa

Exposure time: 30 seconds;

15% SDS-PAGE gel.

Proteins were transferred to a PVDF membrane and blocked with 5% NFDM/TBST for 1 hour at room temperature. The primary antibody (HA610038) at 1/5,000 dilution was used in 5% NFDM/TBST at room temperature for 2 hours. Goat Anti-Mouse IgG - HRP Secondary Antibody (HA1006) at 1:150,000 dilution was used for 1 hour at room temperature.

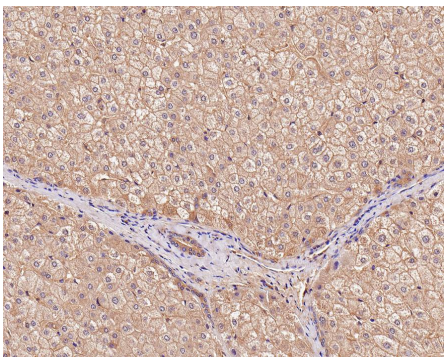

Fig2: Immunohistochemical analysis of paraffin-embedded human liver carcinoma tissue with Mouse anti-BAX antibody (HA610038) at 1/200 dilution.

The section was pre-treated using heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0) for 20 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH_2O and PBS, and then probed with the primary antibody (HA610038) at 1/200 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

Background References

1. Spitz AZ et al. Physiological and pharmacological modulation of BAX. Trends Pharmacol Sci. 2022 Mar
2. Lebeaupin C et al. BAX inhibitor-1: between stress and survival. FEBS J. 2020 May