

# JIP1 Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP9014b

# **Specification**

# JIP1 Antibody (C-term) Blocking Peptide - Product Information

**Primary Accession** 

Q9UQF2

# JIP1 Antibody (C-term) Blocking Peptide - Additional Information

**Gene ID 9479** 

#### **Other Names**

C-Jun-amino-terminal kinase-interacting protein 1, JIP-1, JNK-interacting protein 1, Islet-brain 1, IB-1, JNK MAP kinase scaffold protein 1, Mitogen-activated protein kinase 8-interacting protein 1, MAPK8IP1, IB1, JIP1, PRKM8IP

### Target/Specificity

The synthetic peptide sequence used to generate the antibody <a href=/products/AP9014b>AP9014b</a> was selected from the C-term region of human JIP1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

#### **Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

#### Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

#### **Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

# JIP1 Antibody (C-term) Blocking Peptide - Protein Information

Name MAPK8IP1

Synonyms IB1, JIP1, PRKM8IP

#### **Function**

The JNK-interacting protein (JIP) group of scaffold proteins selectively mediates JNK signaling by aggregating specific components of the MAPK cascade to form a functional JNK signaling module. Required for JNK activation in response to excitotoxic stress. Cytoplasmic MAPK8IP1 causes inhibition of JNK-regulated activity by retaining JNK in the cytoplasm and inhibiting JNK phosphorylation of c-Jun. May also participate in ApoER2-specific reelin signaling. Directly, or indirectly, regulates GLUT2 gene expression and beta-cell function. Appears to have a role in cell signaling in mature and developing nerve terminals. May function as a regulator of vesicle transport, through interactions with the JNK-signaling components and motor proteins. Functions



Tel: 858.875.1900 Fax: 858.875.1999

as an anti-apoptotic protein and whose level seems to influence the beta-cell death or survival response. Acts as a scaffold protein that coordinates with SH3RF1 in organizing different components of the INK pathway, including RAC1 or RAC2, MAP3K11/MLK3 or MAP3K7/TAK1, MAP2K7/MKK7, MAPK8/INK1 and/or MAPK9/INK2 into a functional multiprotein complex to ensure the effective activation of the JNK signaling pathway. Regulates the activation of MAPK8/JNK1 and differentiation of CD8(+) T-cells.

#### **Cellular Location**

Cytoplasm. Cytoplasm, perinuclear region. Nucleus. Endoplasmic reticulum membrane. Mitochondrion membrane. Note=Accumulates in cell surface projections. Under certain stress conditions, translocates to the perinuclear region of neurons. In insulin-secreting cells, detected in both the cytoplasm and nucleus (By similarity).

#### **Tissue Location**

Highly expressed in brain. Expressed in neurons, localizing to neurite tips in differentiating cells. Also expressed in the pancreas, testis and prostate. Low levels in heart, ovary and small intestine. Decreased levels in pancreatic beta cells sensitize cells to IL-1-beta-induced apoptosis

# JIP1 Antibody (C-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

# • Blocking Peptides

JIP1 Antibody (C-term) Blocking Peptide - Images

### JIP1 Antibody (C-term) Blocking Peptide - Background

JIP1 is a regulator of the pancreatic beta-cell function. It is highly similar to JIP-1, a mouse protein known to be a regulator of c-Jun amino-terminal kinase (Mapk8). This protein has been shown to prevent MAPK8 mediated activation of transcription factors, and decrease IL-1 beta and MAP kinase kinase 1 (MEKK1) induced apoptosis in pancreatic beta cells. This protein also functions as a DNA-binding transactivator of the glucose transporter GLUT2.

# JIP1 Antibody (C-term) Blocking Peptide - References

Mooser, V., et.al., Genomics 55 (2), 202-208 (1999)