

JIP-3 Polyclonal Antibody
Catalog # AP70619**Specification**

JIP-3 Polyclonal Antibody - Product Information

Application	WB, IHC-P, IF
Primary Accession	Q9UPT6
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal

JIP-3 Polyclonal Antibody - Additional Information**Gene ID** 23162**Other Names**

MAPK8IP3; JIP3; KIAA1066; C-Jun-amino-terminal kinase-interacting protein 3; JIP-3; JNK-interacting protein 3; JNK MAP kinase scaffold protein 3; Mitogen-activated protein kinase 8-interacting protein 3

Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/20000. Not yet tested in other applications.
IHC-P~~N/A
IF~~1:50~200

Format

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

Storage Conditions

-20°C

JIP-3 Polyclonal Antibody - Protein Information**Name** MAPK8IP3**Synonyms** JIP3, KIAA1066**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 (PubMed:12189133). May function as a regulator of vesicle transport, through interactions with the JNK-signaling components and motor proteins (By similarity). Promotes neuronal axon elongation in a kinesin- and JNK-dependent manner. Activates cofilin at axon tips via local activation of JNK, thereby regulating filopodial dynamics and enhancing axon elongation. Its binding to kinesin heavy chains (KHC), promotes kinesin-1 motility along microtubules and is essential for axon elongation and regeneration. Regulates cortical neuronal migration by mediating NTRK2/TRKB anterograde axonal

transport during brain development (By similarity). Acts as an adapter that bridges the interaction between NTRK2/TRKB and KLC1 and drives NTRK2/TRKB axonal but not dendritic anterograde transport, which is essential for subsequent BDNF-triggered signaling and filopodia formation (PubMed:21775604).

Cellular Location

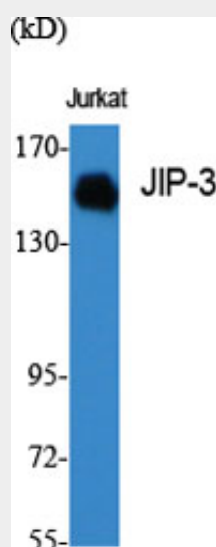
Cytoplasm {ECO:0000250|UniProtKB:Q9ESN9}. Golgi apparatus {ECO:0000250|UniProtKB:Q9ESN9}. Cytoplasmic vesicle {ECO:0000250|UniProtKB:Q9ESN9}. Cell projection, growth cone {ECO:0000250|UniProtKB:Q9ESN9}. Cell projection, axon {ECO:0000250|UniProtKB:E9PSK7}. Cell projection, dendrite {ECO:0000250|UniProtKB:E9PSK7}. Cytoplasm, perinuclear region {ECO:0000250|UniProtKB:E9PSK7}. Note=Localized in the soma and growth cones of differentiated neurites and the Golgi and vesicles of the early secretory compartment of epithelial cells. KIF5A/B/C-mediated transportation to axon tips is essential for its function in enhancing neuronal axon elongation. {ECO:0000250|UniProtKB:E9PSK7, ECO:0000250|UniProtKB:Q9ESN9}

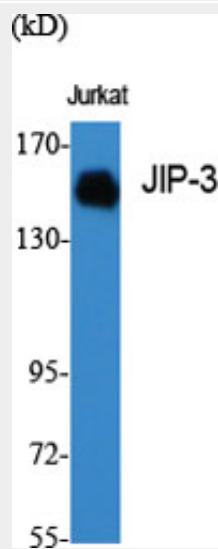
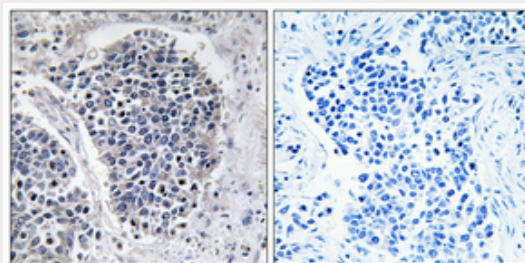
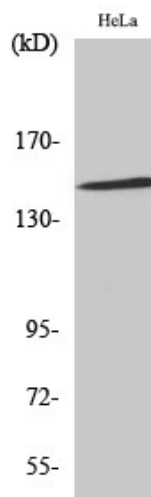
JIP-3 Polyclonal Antibody - Protocols

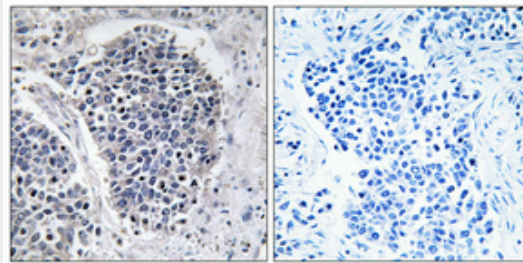
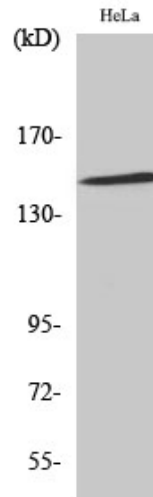
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

JIP-3 Polyclonal Antibody - Images







JIP-3 Polyclonal Antibody - Background

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 (PubMed:12189133). May function as a regulator of vesicle transport, through interactions with the JNK-signaling components and motor proteins (By similarity). Promotes neuronal axon elongation in a kinesin- and JNK-dependent manner. Activates cofilin at axon tips via local activation of JNK, thereby regulating filopodial dynamics and enhancing axon elongation. Its binding to kinesin heavy chains (KHC), promotes kinesin-1 motility along microtubules and is essential for axon elongation and regeneration. Regulates cortical neuronal migration by mediating NTRK2/TRKB anterograde axonal transport during brain development (By similarity). Acts as an adapter that bridges the interaction between NTRK2/TRKB and KLC1 and drives NTRK2/TRKB axonal but not dendritic anterograde transport, which is essential for subsequent BDNF-triggered signaling and filopodia formation (PubMed:21775604).