

Product datasheet

Anti-Tau antibody [5B10] ab278070

2 Images

Overview

Product name	Anti-Tau antibody [5B10]
Description	Mouse monoclonal [5B10] to Tau
Host species	Mouse
Tested applications	Suitable for: WB, ICC
Species reactivity	Reacts with: Mouse, Rat
Immunogen	Recombinant full length protein corresponding to Human Tau aa 1-758. Database link: P10636
Epitope	HVPGGGNKKIETHKLFREN
Positive control	WB: Rat brain and Spinal cord, Mouse brain and Spinal cord. ICC: Embryonic rat E20 cortical neuron-glia cells.

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. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.03% Sodium azide Constituents: 49.07% PBS, 50% Glycerol (glycerin, glycerine)
Purity	Protein G purified
Purification notes	Purified from Tissue Culture supernatant.
Clonality	Monoclonal
Clone number	5B10
Isotype	IgG1

Applications

Our [Abpromise guarantee](#) covers the use of **ab278070** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		1/2000 - 1/10000. Predicted molecular weight: 78 kDa.
ICC		1/1000 - 1/2000.

Target

Function

Promotes microtubule assembly and stability, and might be involved in the establishment and maintenance of neuronal polarity. The C-terminus binds axonal microtubules while the N-terminus binds neural plasma membrane components, suggesting that tau functions as a linker protein between both. Axonal polarity is predetermined by tau localization (in the neuronal cell) in the domain of the cell body defined by the centrosome. The short isoforms allow plasticity of the cytoskeleton whereas the longer isoforms may preferentially play a role in its stabilization.

Tissue specificity

Expressed in neurons. Isoform PNS-tau is expressed in the peripheral nervous system while the others are expressed in the central nervous system.

Involvement in disease

Note=In Alzheimer disease, the neuronal cytoskeleton in the brain is progressively disrupted and replaced by tangles of paired helical filaments (PHF) and straight filaments, mainly composed of hyperphosphorylated forms of TAU (PHF-TAU or AD P-TAU).

Defects in MAPT are a cause of frontotemporal dementia (FTD) [MIM:600274]; also called frontotemporal dementia (FTD), pallido-ponto-nigral degeneration (PPND) or historically termed Pick complex. This form of frontotemporal dementia is characterized by presenile dementia with behavioral changes, deterioration of cognitive capacities and loss of memory. In some cases, parkinsonian symptoms are prominent. Neuropathological changes include frontotemporal atrophy often associated with atrophy of the basal ganglia, substantia nigra, amygdala. In most cases, protein tau deposits are found in glial cells and/or neurons.

Defects in MAPT are a cause of Pick disease of the brain (PIDB) [MIM:172700]. It is a rare form of dementia pathologically defined by severe atrophy, neuronal loss and gliosis. It is characterized by the occurrence of tau-positive inclusions, swollen neurons (Pick cells) and argentophilic neuronal inclusions known as Pick bodies that disproportionately affect the frontal and temporal cortical regions. Clinical features include aphasia, apraxia, confusion, anomia, memory loss and personality deterioration.

Note=Defects in MAPT are a cause of corticobasal degeneration (CBD). It is marked by extrapyramidal signs and apraxia and can be associated with memory loss. Neuropathologic features may overlap Alzheimer disease, progressive supranuclear palsy, and Parkinson disease.

Defects in MAPT are a cause of progressive supranuclear palsy type 1 (PSNP1) [MIM:601104, 260540]; also abbreviated as PSP and also known as Steele-Richardson-Olszewski syndrome. PSNP1 is characterized by akinetic-rigid syndrome, supranuclear gaze palsy, pyramidal tract dysfunction, pseudobulbar signs and cognitive capacities deterioration. Neurofibrillary tangles and gliosis but no amyloid plaques are found in diseased brains. Most cases appear to be sporadic, with a significant association with a common haplotype including the MAPT gene and the flanking regions. Familial cases show an autosomal dominant pattern of transmission with incomplete penetrance; genetic analysis of a few cases showed the occurrence of tau mutations, including a deletion of Asn-613.

Sequence similarities

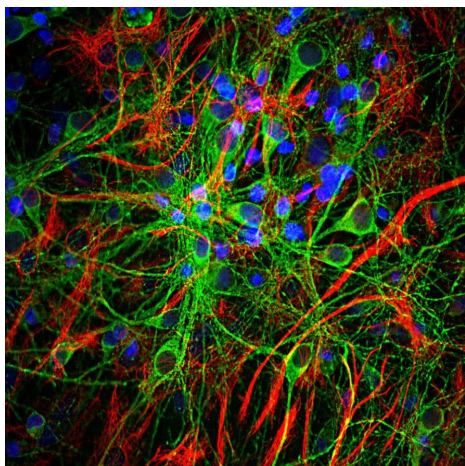
Contains 4 Tau/MAP repeats.

Developmental stage

Four-repeat (type II) tau is expressed in an adult-specific manner and is not found in fetal brain, whereas three-repeat (type I) tau is found in both adult and fetal brain.

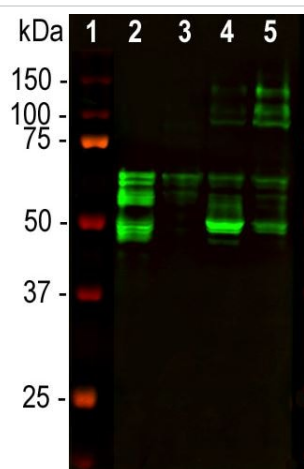
Domain	The tau/MAP repeat binds to tubulin. Type I isoforms contain 3 repeats while type II isoforms contain 4 repeats.
Post-translational modifications	<p>Phosphorylation at serine and threonine residues in S-P or T-P motifs by proline-directed protein kinases (PDPK: CDK1, CDK5, GSK-3, MAPK) (only 2-3 sites per protein in interphase, seven-fold increase in mitosis, and in PHF-tau), and at serine residues in K-X-G-S motifs by MAP/microtubule affinity-regulating kinase (MARK) in Alzheimer diseased brains. Phosphorylation decreases with age. Phosphorylation within tau's repeat domain or in flanking regions seems to reduce tau's interaction with, respectively, microtubules or plasma membrane components. Phosphorylation on Ser-610, Ser-622, Ser-641 and Ser-673 in several isoforms during mitosis.</p> <p>Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the proteasome (By similarity). PHF-tau can be modified by three different forms of polyubiquitination. 'Lys-48'-linked polyubiquitination is the major form, 'Lys-6'-linked and 'Lys-11'-linked polyubiquitination also occur.</p> <p>Glycation of PHF-tau, but not normal brain tau. Glycation is a non-enzymatic post-translational modification that involves a covalent linkage between a sugar and an amino group of a protein molecule forming ketoamine. Subsequent oxidation, fragmentation and/or cross-linking of ketoamine leads to the production of advanced glycation endproducts (AGES). Glycation may play a role in stabilizing PHF aggregation leading to tangle formation in AD.</p>
Cellular localization	Cytoplasm > cytosol. Cell membrane. Cytoplasm > cytoskeleton. Cell projection > axon. Mostly found in the axons of neurons, in the cytosol and in association with plasma membrane components.
Form	There are 9 isoforms produced by alternative splicing.

Images



Cortical neuron-glia cell culture from E20 rat stained for MAP-t (green) using ab278070 at 1/2000 dilution in immunocytochemistry. A chicken polyclonal antibody to GFAP (red) was used at 1/5000 dilution. The blue is DAPI staining of nuclear DNA.

Immunocytochemistry - Anti-Tau antibody [5B10] (ab278070)



Western blot - Anti-Tau antibody [5B10] (ab278070)

Lanes 2-5 : Anti-Tau antibody [5B10] (ab278070) at 1/2000 dilution

Lane 1 : Molecular weight ladder

Lane 2 : rat brain

Lane 3 : rat spinal cord

Lane 4 : mouse brain

Lane 5 : mouse spinal cord

Predicted band size: 78 kDa

Tau protein is expressed as up to 9 different isoforms of different molecular weight, and so appears as multiple closely spaced bands in the region of the blot from 48 kDa to 67 kDa with some higher molecular weight isoforms.

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