Product datasheet

Anti-Tau (phospho T181) antibody ab75679

Overview

Product name: Anti-Tau (phospho T181) antibody
Description: Rabbit polyclonal to Tau (phospho T181)
Host species: Rabbit
Specificity: ab75679 detects endogenous levels of isoform Tau-F, which is a truncated form of the unprocessed precursor isoform PNS-tau (http://www.uniprot.org/uniprot/P10636), where the phosphorylation site is located at threonine 181.

Tested applications: Suitable for: WB, IHC-P
Species reactivity: Reacts with: Mouse, Rat, Human
Immunogen: Synthetic peptide corresponding to Human Tau. (PKT(phospho)PP)
Positive control: Rat hippocampal region tissue from a model with Alzheimer’s Disease and extracts from mouse brain tissue.

Properties

Form: Liquid
Storage instructions: Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer: pH: 7.40
Preservative: 0.02% Sodium azide
Constituents: 50% Glycerol, 0.87% Sodium chloride, PBS

Without Mg2+ and Ca2+

Purity: Immunogen affinity purified

Purification notes: ab75679 was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phosphorylation site.

Clonality: Polyclonal
Isotype: IgG

Applications

Our Abpromise guarantee covers the use of ab75679 in the following tested applications.
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, anoma, 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**
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.

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.

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.

**Cellular localization**

**Form**
There are 9 isoforms produced by alternative splicing.

**Images**

ab75679 at 1/50 dilution staining Tau in rat hippocampal region tissue from a model with Alzheimer's Disease by Immunohistochemistry, Paraffin-embedded tissue.
**Western blot - Anti-Tau (phospho T181) antibody (ab75679)**

**All lanes:** Anti-Tau (phospho T181) antibody (ab75679) at 1/500 dilution

**Lane 1:** mouse brain tissue extracts with immunising peptide

**Lane 2:** mouse brain tissue extracts

**Predicted band size:** 46 kDa

**Observed band size:** 46 kDa

**Additional bands at:** 35 kDa. We are unsure as to the identity of these extra bands.

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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