Product datasheet

Anti-Tau (phospho T205) antibody ab4841

Overview

Product name: Anti-Tau (phospho T205) antibody
Description: Rabbit polyclonal to Tau (phospho T205)
Host species: Rabbit
Tested applications:
- Suitable for: Flow Cyt, WB
Species reactivity:
- Reacts with: Mouse, Human
- Predicted to work with: Rat
Immunogen: Synthetic peptide (Human) derived from the region of human tau that contains threonine 205.

Properties

Form: Liquid
Storage instructions: Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
Storage buffer:
- pH: 7.30
- Preservative: 0.05% Sodium azide
- Constituents: PBS, 50% Glycerol, 0.1% BSA
Purity: Immunogen affinity purified
Purification notes: Purified from rabbit serum by sequential epitope-specific chromatography. The antibody has been negatively preadsorbed using a non-phosphopeptide corresponding to the site of phosphorylation to remove antibody that is reactive with non-phosphorylated tau. The final product is generated by affinity chromatography using a tau-derived peptide that is phosphorylated at threonine 205.
Clonality: Polyclonal
Isotype: IgG

Applications

Our Abpromise guarantee covers the use of ab4841 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
**Application**

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<td>Flow Cyt</td>
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<tr>
<td>WB</td>
<td>IHC-Fr: Use at an assay dependent dilution. WB: 1/1000. Predicted molecular weight: 62 kDa. Can be blocked with Tau peptide - phospho T205 (phospho and non-phospho pair). Not tested in other applications. Optimal dilutions/concentrations should be determined by the end user.</td>
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**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**
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, sevenfold 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**

Western blot - Anti-Tau (phospho T205) antibody (ab4841)

WB using Ab4841. The peptide corresponding to tau [pT205] blocks the antibody signal, thereby demonstrating the specificity of the antibody.
Flow cytometric analysis of 70% ethanol-fixed SH-SY5Y (human neuroblastoma cell line from bone marrow) cell line labeling Tau (phospho T205) with ab4841 at 5 μg/million cells (red) compared with a rabbit isotype control details (pink), an unlabelled control (purple) and a secondary only control (green). Alexa Fluor® 488 Goat Anti-Rabbit, at 1/400 dilution was used as the secondary antibody.

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