Product datasheet

Anti-Tau antibody ab62639

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Overview

Product name  Anti-Tau antibody
Description  Sheep polyclonal to Tau
Host species  Sheep
Tested applications  Suitable for: IHC-P, IHC-Fr, ICC/IF
Species reactivity  Reacts with: Human
Predicted to work with: Chimpanzee, Rhesus monkey, Gorilla, Orangutan
Immunogen  Synthetic peptide corresponding to Human Tau aa 12-30.
Sequence: EDHAGTYGLGDRKDQGGYTC

Properties

Form  Liquid
Storage buffer  Preservative: 0.02% Thimerosal (merthiolate)
Constituent: Whole serum
Purity  Whole antiserum
Clonality  Polyclonal
Isotype  IgG

Applications

Our Abpromise guarantee covers the use of ab62639 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

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Application notes

This antibody achieves excellent staining of Tau aggregations within neurons (tangles), and distrophic neurons within plaques and also stains Tau threads within axons.

Not yet tested in other applications.
Optimal dilutions/concentrations should be determined by the end user.

**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 disproportionally affect the frontal and temporal cortical regions. Clinical features include aphasia, apraxia, confusion, anemia, memory loss and personality deterioration.

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
IHC-P image of Tau staining on Human colon tissue section using ab62639 (1/4000). The section were subjected to heat mediated antigen retrieval using citric acid (pH 6). The sections were then blocked using 1% BSA for 10 min at 21°C. The primary antibody was incubated for 2 hours at 21°C.

ab62639 at 1/400 dilution staining Tau in neurons by confocal Immunohistochemistry. Jackson Cy2 secondary antibody (1/100).

ab62639 at 1/400 dilution staining Tau in dystrophic neurites within plaques by confocal Immunohistochemistry. Jackson Cy2 secondary antibody (1/100).
ab62639 at 1/8000 staining Tau in neurons within neuronal axons Tau threads (as indicated by arrows), by Light Immunohistochemistry. Jackson biotinylated donkey anti rabbit secondary antibody (1/2000) followed by Sigma Extra avidin peroxidase (1/400) and DAB substrate.

ab62639 at 1/8000 staining Tau in dystrophic neurites within plaques, within neuronal axons Tau threads (as indicated by arrows), by Light Immunohistochemistry. Jackson biotinylated donkey anti rabbit secondary antibody (1/2000) followed by Sigma Extra avidin peroxidase (1/400) and DAB substrate.

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