Product datasheet

Anti-Tau antibody ab75714

Overview

<table>
<thead>
<tr>
<th>Product name</th>
<th>Anti-Tau antibody</th>
</tr>
</thead>
<tbody>
<tr>
<td>Description</td>
<td>Chicken polyclonal to Tau</td>
</tr>
<tr>
<td>Host species</td>
<td>Chicken</td>
</tr>
<tr>
<td>Tested applications</td>
<td>Suitable for: IHC-P</td>
</tr>
<tr>
<td>Species reactivity</td>
<td>Reacts with: Mouse, Rat, Human</td>
</tr>
<tr>
<td>Immunogen</td>
<td>Two synthetic peptides corresponding to two regions of the Tau gene product corresponding to sequences shared between the mouse (P01637, NCBI) and human (NP_05819, NCBI) proteins, and not containing any of the serine and threonine residues known to undergo phosphorylation.</td>
</tr>
</tbody>
</table>

General notes

Two different affinity purified anti peptide antibodies were combined to make this product. The concentrations of both of these antibodies was 100 µg/ml, making the total antibody concentration 200 µg/ml.

Reproducibility is key to advancing scientific discovery and accelerating scientists’ next breakthrough.

Abcam is leading the way with our range of recombinant antibodies, knockout-validated antibodies and knockout cell lines, all of which support improved reproducibility.

We are also planning to innovate the way in which we present recommended applications and species on our product datasheets, so that only applications & species that have been tested in our own labs, our suppliers or by selected trusted collaborators are covered by our Abpromise™ guarantee.

In preparation for this, we have started to update the applications & species that this product is Abpromise guaranteed for.

We are also updating the applications & species that this product has been "predicted to work with," however this information is not covered by our Abpromise guarantee.

Applications & species from publications and Abreviews that have not been tested in our own labs or in those of our suppliers are not covered by the Abpromise guarantee.

Please check that this product meets your needs before purchasing. If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, as well as customer reviews and Q&As.
**Form**  
Liquid

**Storage instructions**  

**Storage buffer**  
pH: 7  
Preservative: 0.02% Sodium azide  
Constituent: 0.0268% PBS  
PBS isotonic (0.9%, w/v)

**Purity**  
Immunogen affinity purified

**Purification notes**  
IgY fractions purified from immune egg yolks then affinity purified using a peptide column. Equal volumes of both affinity purified anti peptide antibodies were mixed, and the preparation was filter sterilized.

**Primary antibody notes**  
Two different affinity purified anti peptide antibodies were combined to make this product. The concentrations of both of these antibodies was 100 µg/ml, making the total antibody concentration 200 µg/ml.

**Clonality**  
Polyclonal

**Isotype**  
IgY

**Applications**

Our Abpromise guarantee covers the use of ab75714 in the following tested applications.

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

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHC-P</td>
<td></td>
<td>Use at an assay dependent concentration.</td>
</tr>
</tbody>
</table>

**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) [MM: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, 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.

<table>
<thead>
<tr>
<th>Sequence similarities</th>
<th>Contains 4 Tau/MAP repeats.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Developmental stage</td>
<td>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.</td>
</tr>
<tr>
<td>Domain</td>
<td>The tau/MAP repeat binds to tubulin. Type I isoforms contain 3 repeats while type II isoforms contain 4 repeats.</td>
</tr>
<tr>
<td>Post-translational modifications</td>
<td>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.</td>
</tr>
<tr>
<td>Form</td>
<td>There are 9 isoforms produced by alternative splicing.</td>
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</tbody>
</table>
Immunohistochemistry (10% Formalin-fixed paraffin-embedded sections) analysis of Human Alzheimer's disease brain (CA1 region of hippocampus) tissue labelling Tau (brown) with ab75714.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Tau antibody (ab75714)

IHC image of Tau staining in Human Hippocampus (Alzheimer's) (ab4583) formalin-fixed paraffin-embedded tissue section, performed on a Leica Bond™ system using the standard protocol F. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH6, epitope retrieval solution 1) for 20 mins. The section was then incubated with ab75714, 5 µg/ml, for 15 mins at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.

For other IHC staining systems (automated and non-automated) customers should optimize variable parameters such as antigen retrieval conditions, primary antibody concentration and antibody incubation times.

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