Anti-Tau antibody ab64193

Product name: Anti-Tau antibody
Description: Rabbit polyclonal to Tau
Host species: Rabbit
Specificity: ab64193 recognizes both non-phosphorylated and phosphorylated Ser262.

Tested applications:
Suitable for: ICC, WB, Dot blot, ICC/IF, IHC-FoFr
Unsuitable for: IHC-P

Species reactivity:
Reacts with: Mouse, Cow, Human, Zebrafish, Apteronotus leptorhynchus
Predicted to work with: Rat

Immunogen:
Synthetic peptide corresponding to Human Tau aa 576-583.
Sequence: KIGSTENL

Positive control:
Recombinant Human Tau412 protein (ab125484) can be used as a positive control in WB.
Mouse brain tissue lysate.

Properties:
Form: Liquid
Storage buffer: pH: 7.40
Preservative: 0.05% Sodium azide
Constituents: 99% PBS, 0.5% BSA

Purity: Immunogen affinity purified
Clonality: Polyclonal
Isotype: IgG

Applications:
Our Abpromise guarantee covers the use of ab64193 in the following tested applications.
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>ICC</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
</tr>
<tr>
<td>WB</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use a concentration of 0.5 - 2 µg/ml. Detects a band of approximately 52 kDa (predicted molecular weight: 79 kDa).</td>
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<tr>
<td>Dot blot</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use a concentration of 0.5 - 2 µg/ml. By dot blot, this antibody only recognizes the immunizing peptide.</td>
</tr>
<tr>
<td>ICC/IF</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
</tr>
<tr>
<td>IHC-FoFr</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
</tr>
</tbody>
</table>

**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 argyrophilic 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. 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**

![Western blot - Anti-Tau antibody (ab64193)](image)

Anti-Tau antibody (ab64193) at 1/100 dilution + Mouse brain whole tissue lysate at 120 µg

**Secondary**
HRP-conjugated goat anti-rabbit IgG polyclonal at 1/10000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

**Predicted band size:** 79 kDa

**Observed band size:** 52 kDa
why is the actual band size different from the predicted?

**Exposure time:** 5 minutes

Immunohistochemical analysis of *Apterontus leptorhynchus* brain tissue, staining Tau with ab64193.

Tissue was fixed with paraformaldehyde, permeabilized with 0.3% Triton X-100 and blocked with 3% sheep serum (ab7489), 1% BSA and 1% teleostean gelatine in TBS for 1 hour at 24°C. Samples were incubated with primary antibody (1/20 in blocking solution) for 18 hours at 4°C. An AlexaFluor®546-conjugated goat anti-rabbit polyclonal IgG (1/200) was used as the secondary antibody.

Immunocytochemistry of Zebrafish Cultured Cells (primary neuron), labelling Tau with ab64193.
Immunocytochemistry/ Immunofluorescence analysis of mouse primary cortical neuronal cells labeling Tau with ab64193 at a dilution of 1/100. The cells were fixed with Ethanol and permeabilized with 0.2% Triton X-100. An AlexaFluor® 488-conjugated goat anti-rabbit polyclonal IgG (1/200) was used as the secondary antibody.

Anti-Tau antibody (ab64193) at 1/200 dilution + Mouse brain tissue lysate at 15 µg

**Predicted band size:** 79 kDa

**Observed band size:** 52 kDa  why is the actual band size different from the predicted?

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