abcam

Product datasheet

Anti-Tau (phospho S202) antibody [EPR2402] ab108387

Recombinant RabMAb

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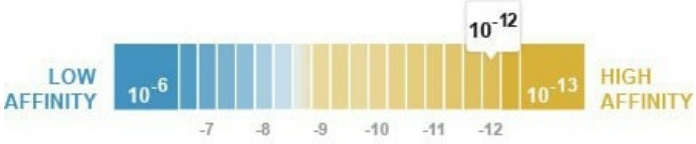
Overview

Product name	Anti-Tau (phospho S202) antibody [EPR2402]
Description	Rabbit monoclonal [EPR2402] to Tau (phospho S202)
Host species	Rabbit
Specificity	*Stimulation may be required to allowdetection of the phosphorylated protein. Please see images belowfor recommended treatment conditions and positive controls.* The specificity of this antibody refers to P10636-8.
Tested applications	**Suitable for:** WB, Dot blot **Unsuitable for:** Flow Cyt,ICC/IF or IHC-P
Species reactivity	**Reacts with:** Mouse, Rat, Human
Immunogen	Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.
Positive control	WB: Human brain lysate, mouse hippocampus, rat hippocampus and cerebral cortex lysates.
General notes	This product is a recombinant monoclonal antibody, which offers several advantages including: - High batch-to-batch consistency and reproducibility - Improved sensitivity and specificity - Long-term security of supply - Animal-free production For more information **see here**. Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb® patents**.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at -20°C. Stable for 12 months at -20°C.
Dissociation constant (K_D)	K_D = 4.13 x 10^{-12} M



Learn more about K_D

Storage buffer	pH: 7.20 Preservative: 0.01% Sodium azide Constituents: 0.05% BSA, 40% Glycerol (glycerin, glycerine), 59% PBS
Purity	Protein A purified
Clonality	Monoclonal
Clone number	EPR2402
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab108387 in the following tested applications.

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

Application	Abreviews	Notes
WB		1/5000 - 1/10000. Predicted molecular weight: 79 kDa.
Dot blot		1/1000.

Application notes Is unsuitable for Flow Cyt,ICC/IF or IHC-P.

Target

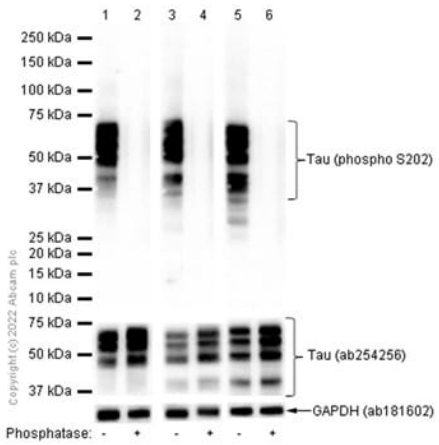
Function Promotes microtubule assembly and stability, and might be involved in the establishment and maintenance of neuronal polarity. The C-terminus binds axonal microtubules while the N-terminus binds neural plasma membrane components, suggesting that tau functions as a linker protein between both. 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 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.
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	Cytoplasm > cytosol. Cell membrane. Cytoplasm > cytoskeleton. Cell projection > axon. Mostly found in the axons of neurons, in the cytosol and in association with plasma membrane components.
Form	There are 9 isoforms produced by alternative splicing.

Images



Western blot - Anti-Tau (phospho S202) antibody [EPR2402] (ab108387)

All lanes : Anti-Tau (phospho S202) antibody [EPR2402] (ab108387) at 1/1000 dilution

Lane 1 : Rat hippocampus lysate
Lane 2 : Rat hippocampus lysate then the membrane treated with Alkaline Phosphatase for 1 hour
Lane 3 : Rat cerebral cortex lysate
Lane 4 : Rat cerebral cortex lysate then the membrane treated with Alkaline Phosphatase for 1 hour
Lane 5 : Mouse hippocampus lysate
Lane 6 : Mouse hippocampus lysate then the membrane treated with Alkaline Phosphatase for 1 hour

Lysates/proteins at 15 µg per lane.

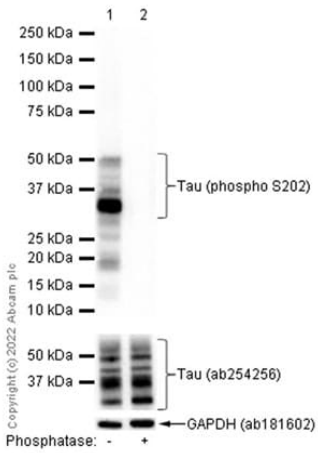
Secondary
All lanes : Goat Anti-Rabbit IgG (HRP) with minimal cross-reactivity with human IgG at 1/2000 dilution

Predicted band size: 79 kDa
Observed band size: 32-72 kDa

Exposure time: 3 seconds

The molecular weight observed is consistent with what has been described in the literature (PMID: 28382304, 32692785 and 30120733).

Blocking and diluting buffer: 5% NFDM/TBST



Western blot - Anti-Tau (phospho S202) antibody [EPR2402] (ab108387)

All lanes : Anti-Tau (phospho S202) antibody [EPR2402] (ab108387) at 1/1000 dilution

Lane 1 : Human brain lysate
Lane 2 : Human brain lysate then the membrane treated with Alkaline Phosphatase for 1 hour

Lysates/proteins at 15 µg per lane.

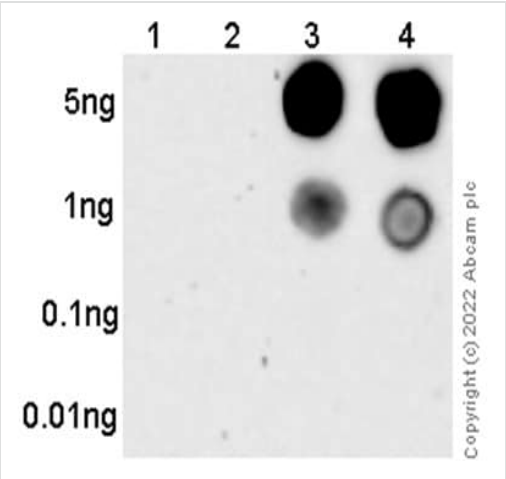
Secondary
All lanes : Goat Anti-Rabbit IgG (HRP) with minimal cross-reactivity with human IgG at 1/2000 dilution

Predicted band size: 79 kDa
Observed band size: 32-72 kDa

Exposure time: 5 seconds

The molecular weight observed is consistent with what has been described in the literature (PMID: 28382304, 32692785 and 30120733).

Blocking and diluting buffer: 5% NFDM/TBST



Dot Blot - Anti-Tau (phospho S202) antibody [EPR2402] (ab108387)

Dot blot analysis using 1/1000 dilution ab108387 and Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (**ab97051**) secondary at 1/100000 dilution.

Blocking and diluting buffer: 5% NFDM/TBST

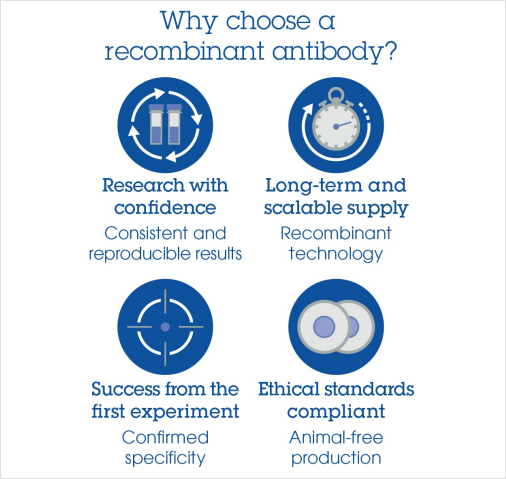
Lane 1: Tau non-phospho peptide

Lane 2: Tau S199 phospho peptide

Lane 3: Tau S202 phospho peptide

Lane 4: Tau S199+S202 phospho peptide

Exposure time: 3 minutes



Anti-Tau (phospho S202) antibody [EPR2402] (ab108387)

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