# abcam

# Product datasheet

# Anti-Tau (phospho S202 + T205) antibody [EPR20390] ab210703

Recombinant RabMAb

#### Overview

Product name Anti-Tau (phospho S202 + T205) antibody [EPR20390]

**Description** Rabbit monoclonal [EPR20390] to Tau (phospho S202 + T205)

Host species Rabbit

**Specificity** The specificity of this antibody refers to P10636-8.

**Tested applications** Suitable for: Dot blot, WB, IP

Species reactivity Reacts with: Human

**Immunogen** Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.

**Positive control** WB: Human brain lysate. IP: Human brain lysate.

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<sup>®</sup> technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb**<sup>®</sup> **patents**.

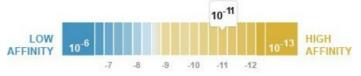
### **Properties**

Form Liquid

**Storage instructions** Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long

term. Avoid freeze / thaw cycle.

**Dissociation constant (K<sub>D</sub>)**  $K_D = 1.65 \times 10^{-11} M$ 



Learn more about K<sub>D</sub>

Storage buffer pH: 7.2

Preservative: 0.01% Sodium azide

Constituents: PBS, 40% Glycerol (glycerin, glycerine), 0.05% BSA

Purity Protein A purified

Clonality Monoclonal
Clone number EPR20390

**Isotype** IgG

#### **Applications**

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab210703 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
Dot blot		1/1000.
WB		1/1000. Detects a band of approximately 50-80 kDa (predicted molecular weight: 78 kDa).
IP		1/30.

#### **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.

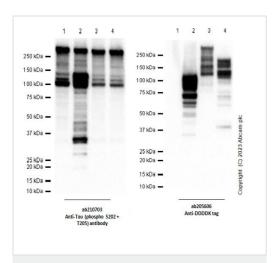
**Cellular localization** 

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 + T205) antibody [EPR20390] (ab210703)

**All lanes :** Anti-Tau (phospho S202 + T205) antibody [EPR20390] (ab210703) at 1/1000 dilution

**Lane 1 :** 293T cells transfected with an empty vector containing a flag tag whole cell lysate

**Lane 2**: 293T cells transfected with a human Tau expression vector containing a flag whole cell lysate

**Lane 3**: 293T cells transfected with a human MAP2 expression vector containing a flag whole cell lysate

**Lane 4**: 293T cells transfected with a human MAP4 expression vector containing a flag whole cell lysate

Lysates/proteins at 20 µg per lane.

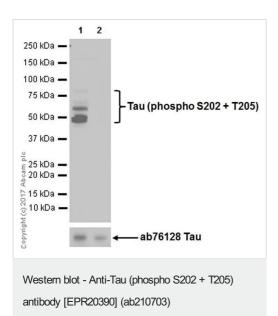
#### Secondary

**All lanes :** Goat Anti-Rabbit IgG H&L (HRP) (<u>ab97051</u>) at 1/20000 dilution

**Predicted band size:** 78 kDa **Observed band size:** 55-100 kDa

Exposure time: 1 second

Blocking/dilution buffer: 5% NFDM/TBST



**All lanes :** Anti-Tau (phospho S202 + T205) antibody [EPR20390] (ab210703) at 1/1000 dilution

Lane 1: Human brain tissue lysate

Lane 2: Alkaline phosphatase treated human brain tissue lysate (1

hour)

Lysates/proteins at 10 µg per lane.

#### **Secondary**

**All lanes :** VeriBlot for IP Detection Reagent (HRP) (<u>ab131366</u>) at 1/100000 dilution

Developed using the ECL technique.

**Predicted band size:** 78 kDa **Observed band size:** 50-80 kDa

Exposure time: 3 minutes

Blocking and dilution buffer: 5% NFDM/TBST.

The molecular weight observed is consistent with what has been described in the literature (PMID: 21932121, PMID: 20660113).



Dot Blot - Anti-Tau (phospho S202 + T205) antibody [EPR20390] (ab210703) Dot blot analysis of Tau (phospho S202+ T205) labeled with ab210703 at 1/1000 dilution.

Lane 1: Tau (phospho S202 + T205) peptide.

Lane 2: Tau (phospho S202) peptide.

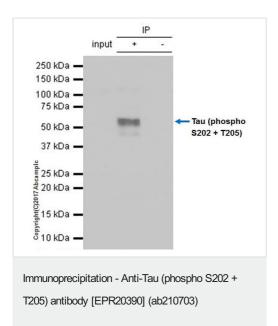
Lane 3: Tau (phospho T205) peptide.

Lane 4: Tau non-phospho peptide.

Goat Anti-Rabbit IgG H&L (HRP) (<u>ab97051</u>) at 1/100000 dilution was used as secondary antibody.

Blocking and dilution buffer: 5% NFDM/TBST.

Exposure time: 3 minutes.



Tau (phospho S202 + T205) was immunoprecipitated from 0.35 mg of human brain lysate with ab210703 at 1/30 dilution. Western blot was performed from the immunoprecipitate using ab210703 at 1/1000 dilution. VeriBlot for IP Detection Reagent (HRP) (ab131366), was used for detection at 1/10000 dilution.

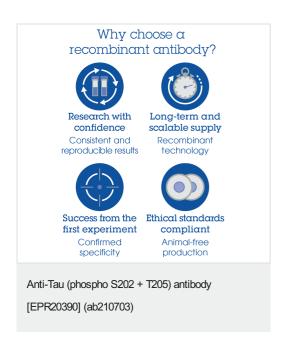
Lane 1: Human brain lysate 10 µg (Input).

Lane 2: ab210703 IP in human brain lysate.

**Lane 3:** Rabbit monoclonal lgG (<u>ab172730</u>) instead of ab210703 in human brain lysate.

Blocking and dilution buffer: 5% NFDM/TBST.

Exposure time: 8 seconds.



Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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