



## Rabbit Anti-phospho-Tau protein (Ser198) antibody

SL4199R

<b>Product Name:</b>	phospho-Tau protein (Ser198)
<b>Chinese Name:</b>	磷酸化微管相关蛋白抗体
<b>Alias:</b>	Tau (phospho Ser198); p-Tau (phospho S198); Tau (phospho S198); MAPT(phospho S198); MAPT; Microtubule-associated protein Tau; AI413597; AW045860; DDPAC; Disinhibition dementia parkinsonism amyotrophy complex; FLJ31424; FTDP 17; FTDP17; G Protein beta 1 gamma 2 subunit interacting factor 1; G protein beta1/gamma2 subunit interacting factor 1; MAPTL; MGC134287; MGC138549; MGC156663; Microtubule associated protein tau isoform 4; MSTD; Mtapt; MTBT1; MTBT2; Neurofibrillary tangle protein; Paired helical filament tau; PHF tau; PHF-tau; PPND; pTau; RNPTAU; Tauopathy and respiratory failure, included; TAU HUMAN.
<b>Organism Species:</b>	Rabbit
<b>Clonality:</b>	Polyclonal
<b>React Species:</b>	Human,Mouse,Rat,Dog,Horse,Rabbit,
<b>Applications:</b>	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800Flow-Cyt=1µg/TestICC=1:100-500IF=1:100-500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
<b>Molecular weight:</b>	52/79kDa
<b>Cellular localization:</b>	cytoplasmicThe cell membrane
<b>Form:</b>	Lyophilized or Liquid
<b>Concentration:</b>	1mg/ml
<b>immunogen:</b>	KLH conjugated Synthesised phosphopeptide derived from human Tau protein around the phosphorylation site of Ser198:GY(P-S)SP
<b>Lsotype:</b>	IgG
<b>Purification:</b>	affinity purified by Protein A
<b>Storage Buffer:</b>	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
<b>Storage:</b>	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized

	antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
<b>PubMed:</b>	<a href="#">PubMed</a>
<b>Product Detail:</b>	<p>This gene encodes the microtubule-associated protein tau (MAPT) whose transcript undergoes complex, regulated alternative splicing, giving rise to several mRNA species. MAPT transcripts are differentially expressed in the nervous system, depending on stage of neuronal maturation and neuron type. MAPT gene mutations have been associated with several neurodegenerative disorders such as Alzheimer's disease, Pick's disease, frontotemporal dementia, cortico-basal degeneration and progressive supranuclear palsy. [provided by RefSeq, Jul 2008].</p> <p><b>Function:</b> 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/MAPT 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.</p> <p><b>Subunit:</b> Interacts with PSMC2 through SQSTM1. Interacts with SQSTM1 when polyubiquitinated. Interacts with FKBP4. Binds to CSNK1D. Interacts with SGK1.</p> <p><b>Subcellular Location:</b> Cytoplasm, cytosol. Cell membrane; Peripheral membrane protein; Cytoplasmic side. Cytoplasm, cytoskeleton. Cell projection, axon. Note=Mostly found in the axons of neurons, in the cytosol and in association with plasma membrane components.</p> <p><b>Tissue Specificity:</b> Expressed in neurons. Isoform PNS-tau is expressed in the peripheral nervous system while the others are expressed in the central nervous system.</p> <p><b>Post-translational modifications:</b> Phosphorylation at serine and threonine residues in S-P or T-P motifs by proline-directed protein kinases (PDPK1: CDK1, CDK5, GSK3, MAPK) (only 2-3 sites per protein in interphase, seven-fold increase in mitosis, and in the form associated with paired helical filaments (PHF-tau)), and at serine residues in K-X-G-S motifs by MAP/microtubule affinity-regulating kinase (MARK1 or MARK2), causing detachment from microtubules, and their disassembly. Phosphorylation decreases with age. Phosphorylation within tau/MAP's repeat domain or in flanking regions seems to reduce tAU/MAP'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. Phosphorylation at Ser-548 by GSK3B reduces ability to bind</p>

and stabilize microtubules. Phosphorylation at Ser-579 by BRSK1 and BRSK2 in neurons affects ability to bind microtubules and plays a role in neuron polarization. Phosphorylated at Ser-554, Ser-579, Ser-602, Ser-606 and Ser-669 by PHK. Phosphorylation at Ser-214 by SGK1 mediates microtubule depolymerization and neurite formation in hippocampal neurons. There is a reciprocal down-regulation of phosphorylation and O-GlcNAcylation. Phosphorylation on Ser-717 completely abolishes the O-GlcNAcylation on this site, while phosphorylation on Ser-713 and Ser-721 reduces glycosylation by a factor of 2 and 4 respectively. Phosphorylation on Ser-721 is reduced by about 41.5% by GlcNAcylation on Ser-717.

Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the proteasome. 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.

O-glycosylated. O-GlcNAcylation content is around 8.2%. There is reciprocal down-regulation of phosphorylation and O-GlcNAcylation. Phosphorylation on Ser-717 completely abolishes the O-GlcNAcylation on this site, while phosphorylation on Ser-713 and Ser-721 reduces O-GlcNAcylation by a factor of 2 and 4 respectively. O-GlcNAcylation on Ser-717 decreases the phosphorylation on Ser-721 by about 41.5%.

Glycation of PHF-tau, but not normal brain TAU/MAPT. 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.

#### **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). O-GlcNAcylation is greatly reduced in Alzheimer disease brain cerebral cortex leading to an increase in TAU/MAPT phosphorylations.

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 disproportionately 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]; 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.

Defects in MAPT are a cause of Parkinson-dementia syndrome (PARDE) [MIM:260540]. A syndrome characterized by parkinsonism tremor, rigidity, dementia, ophthalmoparesis and pyramidal signs. Neurofibrillary degeneration occurs in the hippocampus, basal ganglia and brainstem nuclei.

**Similarity:**

Contains 4 Tau/MAP repeats.

**SWISS:**

P10636

**Gene ID:**

4137

**Database links:**

[Entrez Gene: 281296](#)Cow

[Entrez Gene: 4137](#)Human

[Entrez Gene: 17762](#)Mouse

[Entrez Gene: 29477](#)Rat

[Omim: 157140](#)Human

[SwissProt: P29172](#)Cow

[SwissProt: P10636](#)Human

[SwissProt: P10637](#)Mouse

[SwissProt: P19332](#)Rat

[Unigene: 101174](#)Human

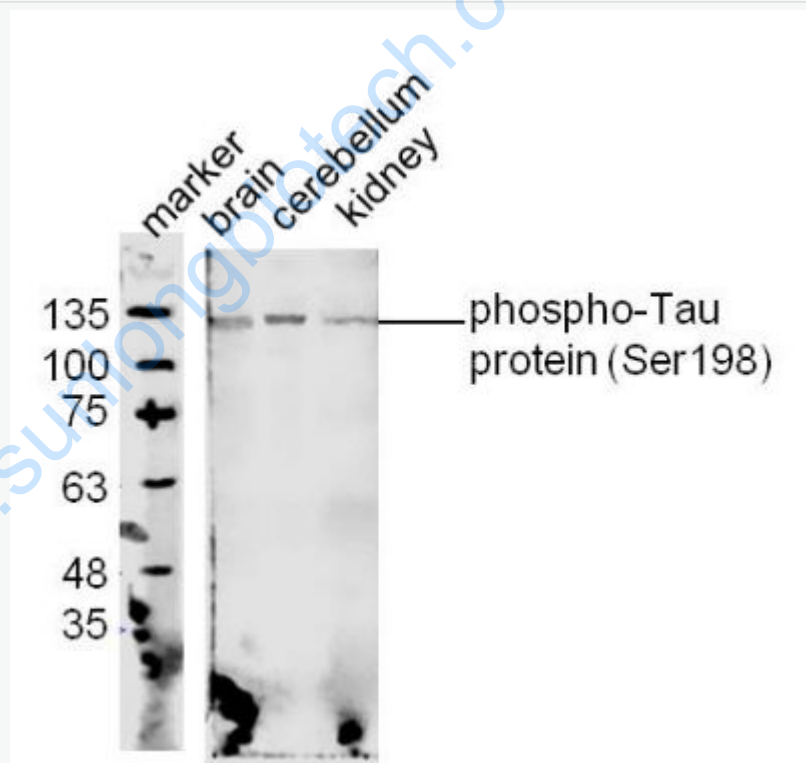
[Unigene: 1287](#)Mouse

[Unigene: 2455](#)Rat

**Important Note:**

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Picture:

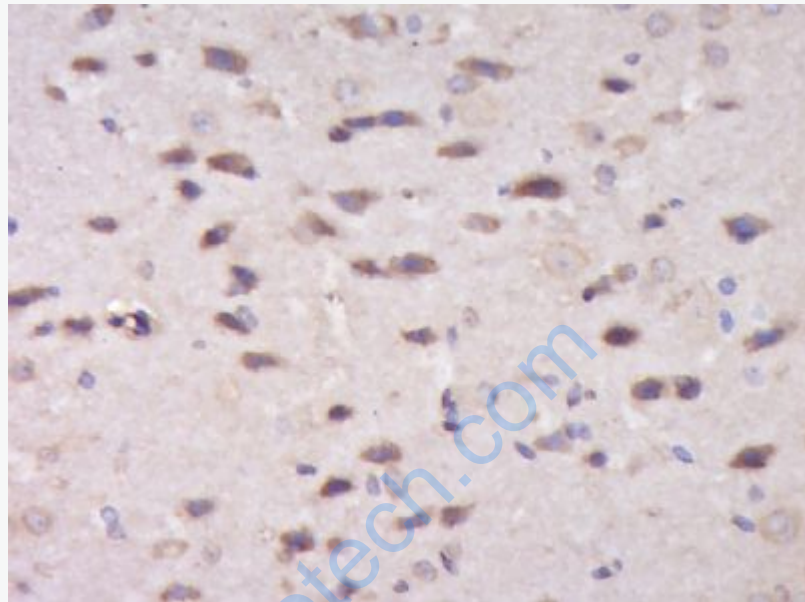


Protein: 1.brain lysates(mo); 2.cerebellum lysates(mo);3.kidney lysates (mo);

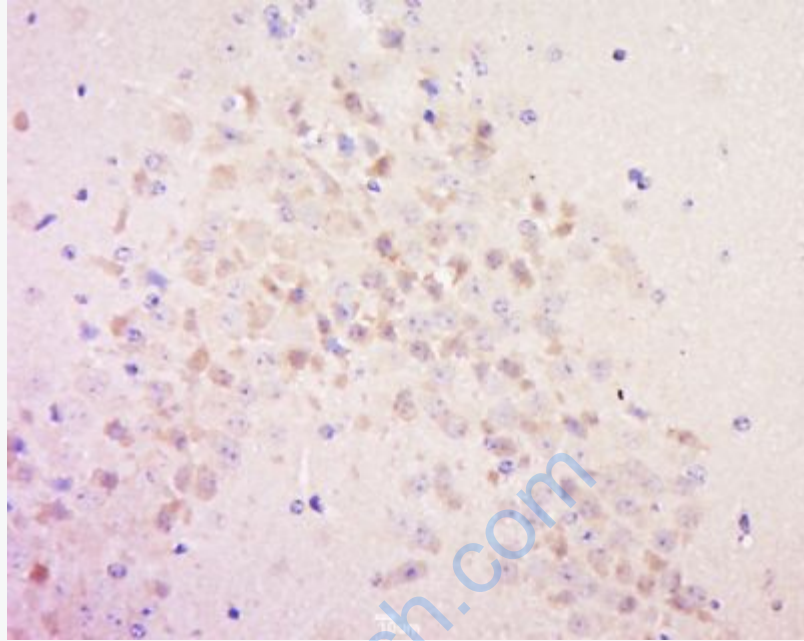
Primary: Rabbit Anti-phospho-Tau protein (Ser198) (SL4199R) at 1:300;

Secondary: 800CW Conjugated Goat (polyclonal) Anti-Rabbit IgG(H+L) at 1:10000;

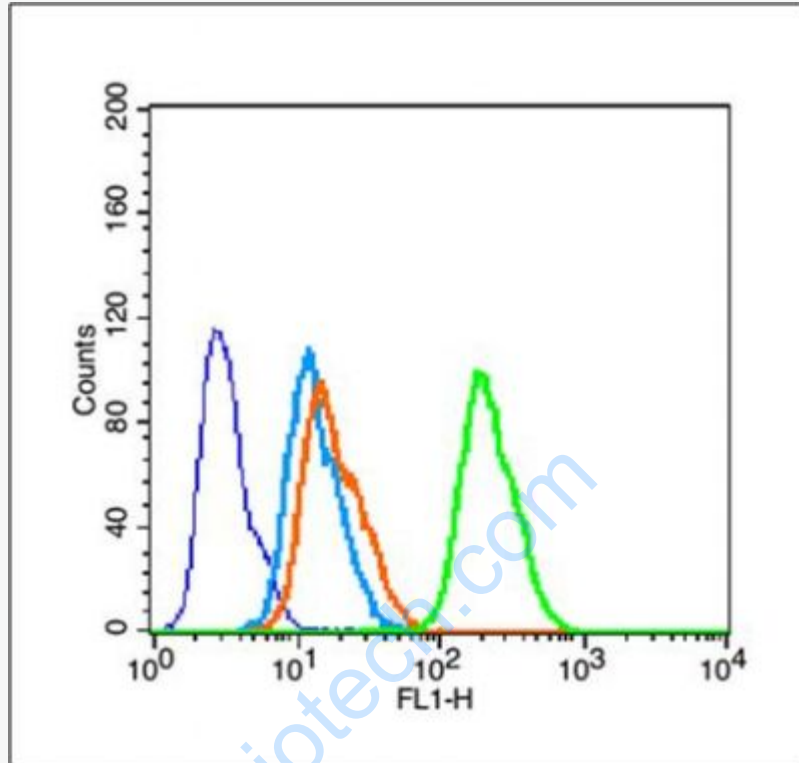
Predicted band size:79 kD Observed band size:128 kD



Paraformaldehyde-fixed, paraffin embedded (Rat brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (phospho-Tau protein (Ser198)) Polyclonal Antibody, Unconjugated (SL4199R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.



Tissue/cell: Mouse brain tissue; 4% Paraformaldehyde-fixed and paraffin-embedded;  
Antigen retrieval: citrate buffer ( 0.01M, pH 6.0 ), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37°C for 20 min;  
Incubation: Anti-MAPT Polyclonal Antibody, Unconjugated(SL4199R) 1:500, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining



Blank control (blue line): MCF 7 (fixed with 70% methanol (Overnight at 4°C) and then permeabilized with 90% ice-cold methanol for 30 min on ice).

Primary Antibody (green line): Rabbit Anti- phospho-Tau protein (Ser198)antibody (SL4199R),Dilution: 0.2µg /10<sup>5</sup> cells;

Isotype Control Antibody (orange line): Rabbit IgG .

Secondary Antibody (white blue line): Goat anti-rabbit IgG-FITC,Dilution: 1µg /test.