

## Product datasheet

### Human Tau (phospho S214) peptide ab19123

[2 Images](#)

#### Description

<b>Product name</b>	Human Tau (phospho S214) peptide
<b>Purity</b>	> 70 % HPLC. 70 - 90% by HPLC
<b>Animal free</b>	No
<b>Nature</b>	Synthetic
<b>Species</b>	Human
<b>Modifications</b>	phospho S214

#### Specifications

Our **Abpromise guarantee** covers the use of **ab19123** in the following tested applications.

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

<b>Applications</b>	Blocking
<b>Form</b>	Lyophilized
<b>Additional notes</b>	<ul style="list-style-type: none"> <li>- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.</li> <li>- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.</li> <li>- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.</li> <li>- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.</li> <li>- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.</li> </ul>

#### Preparation and Storage

<b>Stability and Storage</b>	Shipped at 4°C. Store at -20°C.  Information available upon request.
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#### General Info

<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 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.
<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.
<b>Involvement in disease</b>	<p>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).</p> <p>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.</p> <p>Defects in MAPT are a cause of Pick disease of the brain (PDB) [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.</p> <p>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.</p> <p>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.</p>
<b>Sequence similarities</b>	Contains 4 Tau/MAP repeats.
<b>Developmental stage</b>	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.
<b>Domain</b>	The tau/MAP repeat binds to tubulin. Type I isoforms contain 3 repeats while type II isoforms contain 4 repeats.
<b>Post-translational modifications</b>	<p>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.</p> <p>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</p>

during mitosis.

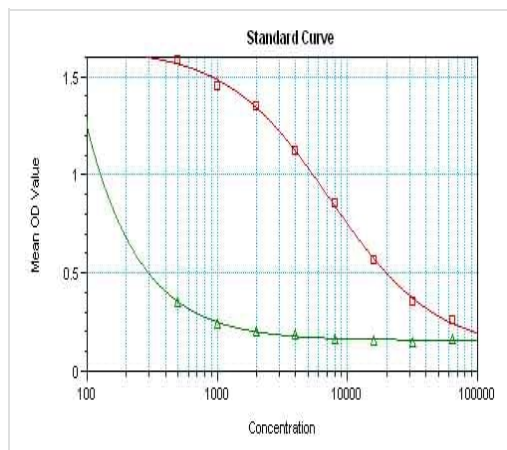
Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the proteasome (By similarity). PHF-tau can be modified by three different forms of polyubiquitination. 'Lys-48'-linked polyubiquitination is the major form, 'Lys-6'-linked and 'Lys-11'-linked polyubiquitination 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

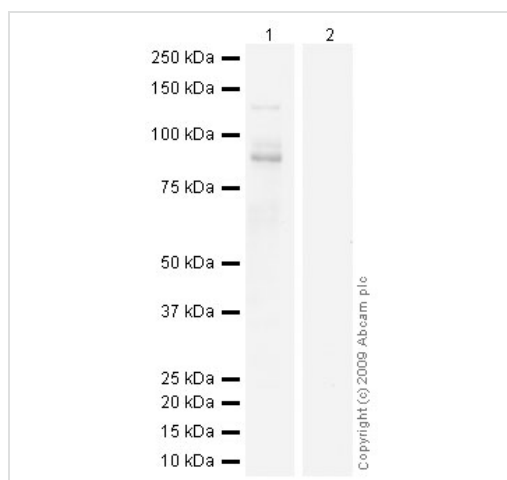
Cytoplasm > cytosol. Cell membrane. Cytoplasm > cytoskeleton. Cell projection > axon. Mostly found in the axons of neurons, in the cytosol and in association with plasma membrane components.

## Images



ELISA - Human Tau (phospho S214) peptide  
(ab19123)

**ab10891** gave a positive result in ELISA against the immunising peptide (ab19123). This ELISA shows that **ab10891** antibody has some cross-reactivity with the non-modified equivalent peptide (**ab23425**) when used at high concentrations. Not yet tested in other applications.



Western blot - Human Tau (phospho S214) peptide  
(ab19123)

**All lanes** : Anti-Tau (phospho S214) antibody (**ab10891**) at 1/250 dilution

**Lane 1** : HeLa (Human epithelial carcinoma cell line) Whole Cell Lysate

**Lane 2** : HeLa (Human epithelial carcinoma cell line) Whole Cell Lysate with Human Tau (phospho S214) peptide (ab19123) at 1 µg/ml

Lysates/proteins at 10 µg per lane.

## Secondary

**All lanes** : Goat polyclonal to Rabbit IgG - H&L - Pre-Adsorbed (HRP) at 1/3000 dilution

Performed under reducing conditions.

**Exposure time:** 2 minutes

Tau contains a number of potential phosphorylation and glycosylation sites (Swiss Prot data) which may explain its migration at a higher molecular weight than predicted.

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

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