

## Product datasheet

# Recombinant human Tau (mutated P301S) protein aggregate (Active) ab246003

[1 References](#) [4 Images](#)

### Description

<b>Product name</b>	Recombinant human Tau (mutated P301S) protein aggregate (Active)	
<b>Biological activity</b>	Thioflavin T emission curve shows increased fluorescence (correlated to tau protein fibrillation) when ab246003 is combined with active tau monomers.	
<b>Purity</b>	> 95 % Ion Exchange Chromatography.	
<b>Expression system</b>	Escherichia coli	
<b>Accession</b>	<b><u>P10636</u></b>	
<b>Protein length</b>	Full length protein	
<b>Animal free</b>	No	
<b>Nature</b>	Recombinant	
<b>Species</b>	Human	
<b>Sequence</b>	MAEPRQEFEVMEHDAGTYGLGDRKDQGGYTMHQDQEG DTDAGLKESPLQT PTEDGSEEPGSETSDAKSTPTAEDVTAPLVDEGAPGKQ AAAQPHTIPEG TTAAEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDD KKAKGADGKTK IATPRGAAPPGQKQANATRIPAKTPPAPKTPPSSGEPK SGDRSGYSSP GSPGTPGSRRTPSLPTPPTREPKKVAVVRTPPKSPSSA KSRLQTAPVPM PDLKNVSKIGSTENLKHQPGGKQIINKKLDLSNVQSK CGSKDNIHV SGGSVQMYKPVDSLKVTSCGSLGNIHHPGGGQVEV KSEKLDKDRV QSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEI VYKSPVVS GDTSPRHLSNVSSSTGSIDMVDSPQLATLADEVASLAKQ GL	
<b>Predicted molecular weight</b>	46 kDa	
<b>Amino acids</b>	1 to 441	

<b>Modifications</b>	mutated P301S
<b>Additional sequence information</b>	NP_005901.2
<b>Description</b>	Recombinant human Tau (mutated P301S) protein (Active)

## Specifications

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Our **Abpromise guarantee** covers the use of **ab246003** 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>	Electron Microscopy Functional Studies SDS-PAGE
<b>Form</b>	Liquid
<b>Additional notes</b>	Active Human Recombinant Tau441 (2N4R), P301S mutant Protein Pre-formed Fibrils.

## Preparation and Storage

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<b>Stability and Storage</b>	Shipped on Dry Ice. Upon delivery aliquot. Store at -80°C. Avoid freeze / thaw cycle. pH: 7.40 Constituents: 0.24% HEPES, 0.58% Sodium chloride This product is an active protein and may elicit a biological response in vivo, handle with caution.
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## General Info

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<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>	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 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, 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.

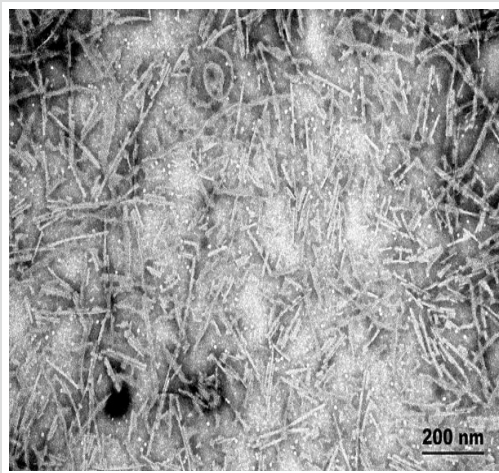
Glycation of PHF-tau, but not normal brain tau. Glycation is a non-enzymatic post-translational modification that involves a covalent linkage between a sugar and an amino group of a protein molecule forming ketoamine. Subsequent oxidation, fragmentation and/or cross-linking of ketoamine leads to the 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.

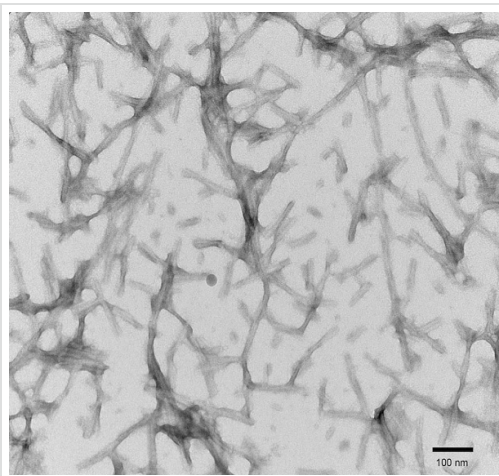
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**Images**



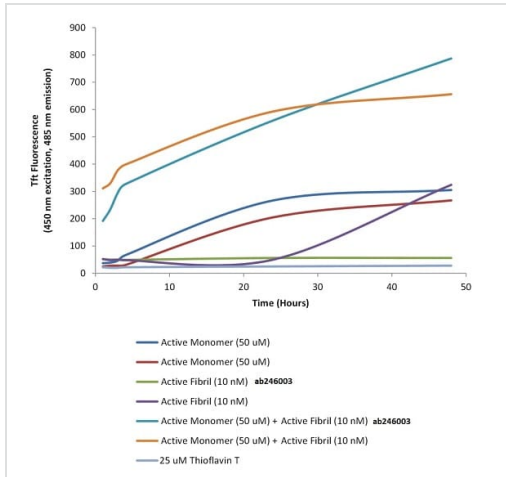
Electron Microscopy - Recombinant human Tau  
(mutated P301S) protein aggregate (Active)  
(ab246003)

TEM of ab246003. Fibrils were sonicated and stained with uranyl acetate.



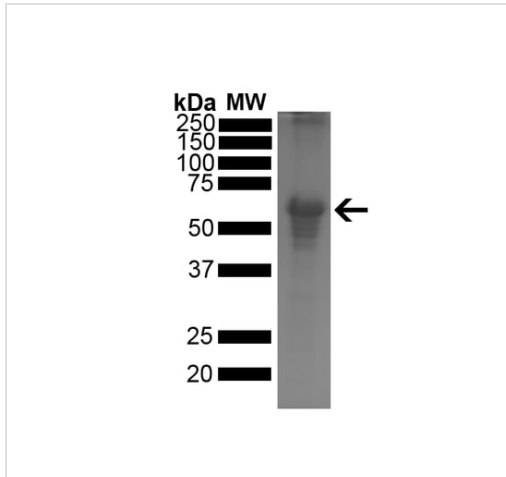
Electron Microscopy - Recombinant human Tau  
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(ab246003)

TEM of ab246003 at 150kx magnification. HV = 80kV. Fibrils were sonicated and stained with uranyl acetate.



Thioflavin T is a fluorescent dye that binds to beta sheet-rich structures, such as those in tau fibrils. Upon binding, the emission spectrum of the dye experiences a red-shift, and increased fluorescence intensity. Thioflavin T emission curves show increased fluorescence (correlated to tau aggregation) when ab246003 is combined with active tau monomers. The preformed fibrils seed the formation of new fibrils from a pool of active monomers. Thioflavin T  $\text{ex} = 450 \text{ nm}$ ,  $\text{em} = 485 \text{ nm}$ .

Functional Studies - Recombinant human Tau (mutated P301S) protein aggregate (Active) (ab246003)



SDS-PAGE analysis of ab246003.

SDS-PAGE - Recombinant human Tau (mutated P301S) protein aggregate (Active) (ab246003)

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