

Product datasheet

Human Tau ELISA Kit ab210972

SimpleStep ELISA

[4 Images](#)

Overview

Product name Human Tau ELISA Kit

Detection method Colorimetric

Precision

Intra-assay

Sample	n	Mean	SD	CV%
Brain ext	5			2.9%

Inter-assay

Sample	n	Mean	SD	CV%
Brain ext	3			4.8%

Sample type

Cell culture supernatant, Serum, Cell culture extracts, Heparin Plasma, EDTA Plasma, Citrate Plasma, Cerebral Spinal Fluid

Assay type

Sandwich (quantitative)

Sensitivity

3.3 pg/ml

Range

31.25 pg/ml - 2000 pg/ml

Recovery

Sample specific recovery

Sample type	Average %	Range
Serum	116	116% - 117%
Cell culture extracts	111	107% - 117%
Tissue Extracts	106	103% - 108%
Heparin Plasma	114	113% - 116%
EDTA Plasma	107	105% - 109%

Sample type	Average %	Range
Cerebral Spinal Fluid	103	101% - 104%

Assay time

1h 30m

Assay duration

One step assay

Species reactivity

Reacts with: Rat, Cow, Human

Product overview

Tau *in vitro* SimpleStep ELISA[®] (Enzyme-Linked Immunosorbent Assay) kit is designed for the quantitative measurement of Tau protein in human serum, plasmas, cerebrospinal fluid, and cell and tissue extracts.

The SimpleStep ELISA[®] employs an affinity tag labeled capture antibody and a reporter conjugated detector antibody which immunocapture the sample analyte in solution. This entire complex (capture antibody/analyte/detector antibody) is in turn immobilized via immunoaffinity of an anti-tag antibody coating the well. To perform the assay, samples or standards are added to the wells, followed by the antibody mix. After incubation, the wells are washed to remove unbound material. TMB substrate is added and during incubation is catalyzed by HRP, generating blue coloration. This reaction is then stopped by addition of Stop Solution completing any color change from blue to yellow. Signal is generated proportionally to the amount of bound analyte and the intensity is measured at 450 nm. Optionally, instead of the endpoint reading, development of TMB can be recorded kinetically at 600 nm.

Notes

Tau proteins constitute six isoforms from a single transcript from the MAPT gene that range from 48-68 kDa. Tau proteins are expressed mainly in the neurons of the central nervous systems, and are implicated in the assistance of microtubule assembly and stability. Hyperphosphorylation of Tau proteins can result in destabilization of microtubule organization, Tau aggregation, and tangle formation. Defective Tau proteins may play a role in diseases of the nervous systems.

The protein standard for the kit is Tau-F, and the kit can detect Tau-A. Other isoforms have not been detected.

Tested applications

Suitable for: Sandwich ELISA

Platform

Pre-coated microplate (12 x 8 well strips)

Properties

Storage instructions

Store at +4°C. Please refer to protocols.

Components	1 x 96 tests
10X Human Tau Capture Antibody	1 x 600µl
10X Human Tau Detector Antibody	1 x 600µl
10X Wash Buffer PT (ab206977)	1 x 20ml
50X Cell Extraction Enhancer Solution (ab193971)	1 x 1ml

Components	1 x 96 tests
5X Cell Extraction Buffer PTR (ab193970)	1 x 10ml
Antibody Diluent CPI	1 x 6ml
Human Tau Lyophilized Recombinant Protein	2 vials
Plate Seals	1 unit
Sample Diluent NS	1 x 50ml
SimpleStep Pre-Coated 96-Well Microplate (ab206978)	1 unit
Stop Solution	1 x 12ml
TMB Development Solution	1 x 12ml

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 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.

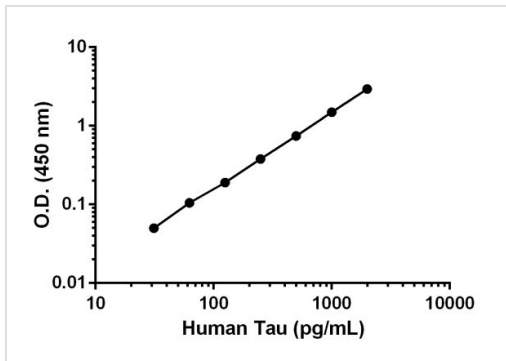
Applications

Our [Abpromise guarantee](#) covers the use of **ab210972** in the following tested applications.

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

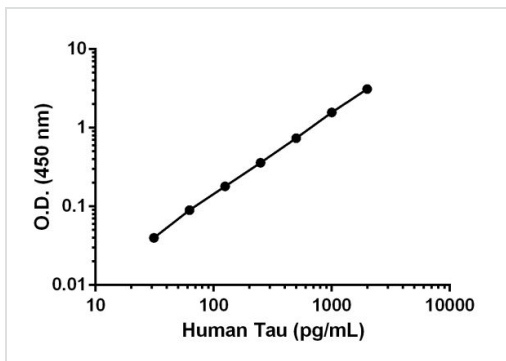
Application	Abreviews	Notes
Sandwich ELISA		Use at an assay dependent concentration.

Images



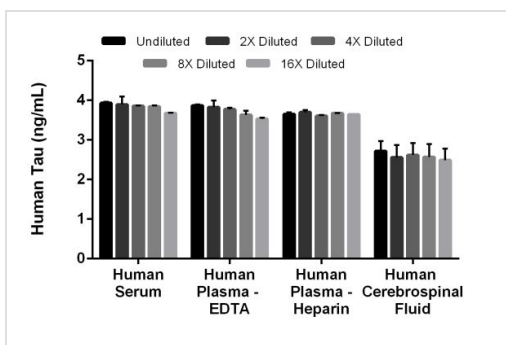
Background-subtracted data values (mean +/- SD) are graphed.

Example of human Tau standard curve in Sample Diluent NS.



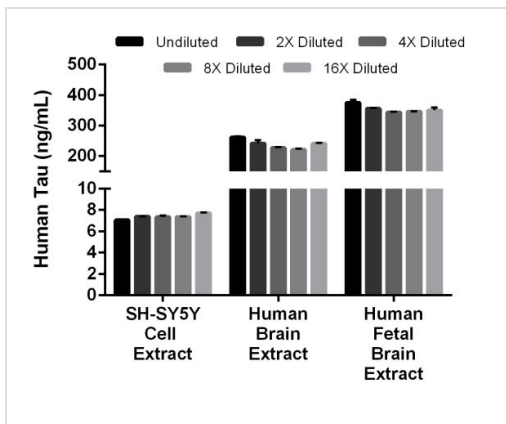
Background-subtracted data values (mean +/- SD) are graphed.

Example of human Tau standard curve in 1X Extraction Buffer PTR.



The concentrations of Tau were measured in duplicates, interpolated from the Tau standard curves and corrected for sample dilution. Undiluted samples are as follows: serum 18.75%, plasma (EDTA) 18.75%, plasma (heparin) 18.75%, and cerebrospinal fluid 37.5%. The interpolated dilution factor corrected values are plotted (mean +/- SD, n=2).

Interpolated concentrations of spiked Tau in human serum, plasma, and cerebrospinal fluid samples.



Interpolated concentrations of native Tau in SH-SY5Y cell extract, human brain extract and human fetal brain extract samples based on a 1,000 µg/mL extract load.

The concentrations of Tau were measured in duplicate and interpolated from the Tau standard curve and corrected for sample dilution. The interpolated dilution factor corrected values are plotted (mean +/- SD, n=2). The mean Tau concentration was determined to be 14.91 ng/mg in SH-SY5Y cell extract, 14.88 µg/mg in human brain extract and 92.05 µg/mg in human fetal brain extract.

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