

Human Tau ELISA Kit, Fluorescent ab229394

Recombinant CatchPoint SimpleStep ELISA

4 Images

Overview

Product name Human Tau ELISA Kit, Fluorescent

Detection method Fluorescent

Precision Intra-assay

Sample	n	Mean	SD	CV%
Serum	8			5.5%

Inter-assay

Sample	n	Mean	SD	CV%
Serum	3			2.7%

Sample type Serum, Cell culture extracts, Tissue Extracts, Cell Lysate, Hep Plasma, EDTA Plasma, Cerebral Spinal Fluid

Assay type Sandwich (quantitative)

Sensitivity 2.9 pg/ml

Range 5.08 pg/ml - 20800 pg/ml

Recovery Sample specific recovery

Sample type	Average %	Range
Serum	79	76% - 84%
Tissue Extracts	108	101% - 111%
Cell Lysate	97	94% - 101%
Cell culture media	86	84% - 91%
Hep Plasma	75	70% - 82%
EDTA Plasma	78	75% - 83%

Sample type	Average %	Range
Cerebral Spinal Fluid	90	89% - 90%

Assay time

1h 30m

Assay duration

One step assay

Species reactivity

Reacts with: Human

Product overview

Tau *in vitro* CatchPoint 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.

This CatchPoint SimpleStep ELISA kit has been **optimized for Molecular Devices Microplate Readers**. Click [here](#) for a list of recommended Microplate Readers.

If using a Molecular Devices' plate reader supported by SoftMax® Pro software, a preconfigured protocol for these CatchPoint SimpleStep ELISA Kits is available with all the protocol and analysis settings at www.softmaxpro.org.

The CatchPoint 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. CatchPoint HRP Development Solution containing the Stoplight Red Substrate is added. During incubation, the substrate is catalyzed by HRP generating a fluorescent product. Signal is generated proportionally to the amount of bound analyte and the intensity is measured in a fluorescence plate reader at 530/570/590 nm Excitation/Cutoff/Emission.

Notes

Tau proteins constitute nine isoforms from a single transcript from the MAPT gene that range from 33-81 kDa. Tau proteins are expressed mainly in the neurons of the central nervous systems. They promote microtubule assembly and stability and might be involved in the establishment and maintenance of neuronal polarity. The C-terminus of Tau binds axonal microtubules while the N-terminus binds neural plasma membrane components, suggesting that Tau functions as a linker protein between both. 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, including Alzheimer disease, Pick disease of the brain, Progressive supranuclear palsy 1 and Parkinson-dementia syndrome. Based on the immunogen design of this ELISA antibody pair, this kit should detect all nine human Tau isoforms and it should have equal affinity towards human, mouse and rat Tau proteins.

Abcam has not and does not intend to apply for the REACH Authorisation of customers' uses of products that contain European Authorisation list (Annex XIV) substances.

It is the responsibility of our customers to check the necessity of application of REACH Authorisation, and any other relevant authorisations, for their intended uses.

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
100X Stoplight Red Substrate	1 x 120µl
10X Human Tau Capture Antibody	1 x 600µl
10X Human Tau Detector Antibody	1 x 600µl
10X Wash Buffer PT (ab206977)	1 x 20ml
500X Hydrogen Peroxide (H2O2, 3%)	1 x 50µl
5X Cell Extraction Buffer PTR (ab193970)	1 x 10ml
Antibody Diluent 5BI	1 x 6ml
Human Tau Lyophilized Recombinant Protein	2 vials
Plate Seals	1 unit
Sample Diluent NS (ab193972)	1 x 50ml
SimpleStep Pre-Coated Black 96-Well Microplate	1 unit
Stoplight Red Substrate Buffer	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 (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.

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.

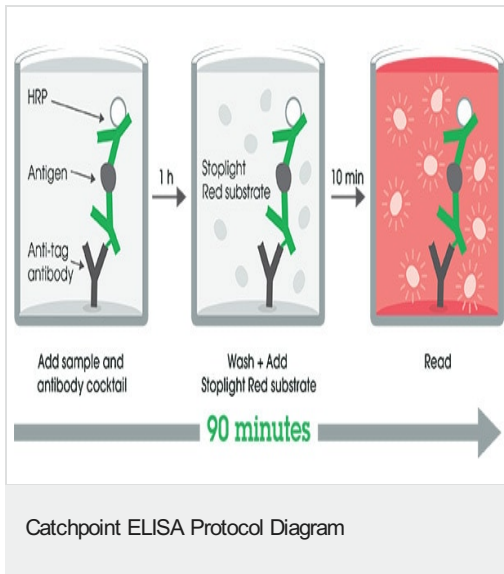
Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the proteasome (By similarity). PHF-tau can be modified by three different forms of polyubiquitination. 'Lys-48'-linked polyubiquitination is the major form, 'Lys-6'-linked and 'Lys-11'-linked polyubiquitination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

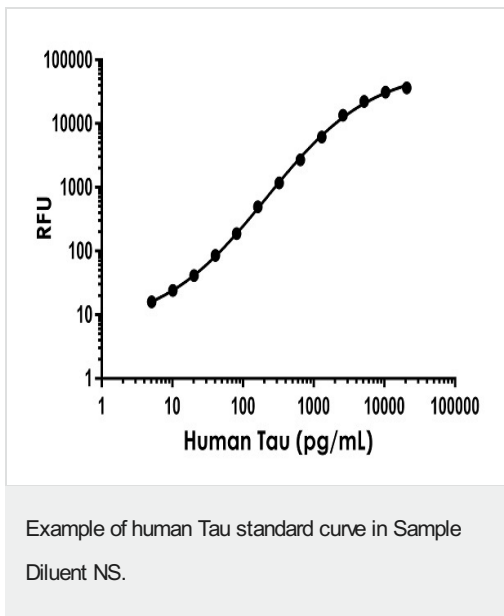
Cellular localization

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.

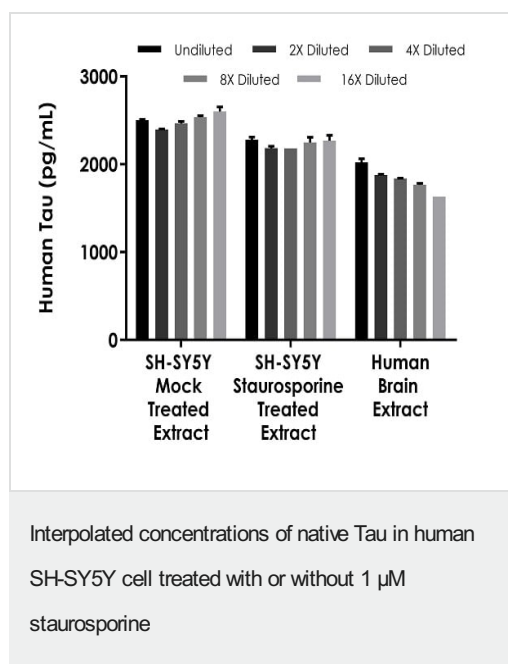
Images



SimpleStep ELISA technology allows the formation of the antibody-antigen complex in one single step, reducing assay time to 90 minutes. Add samples or standards and antibody mix to wells all at once, incubate, wash, and add your final substrate. See protocol for a detailed step-by-step guide.



The Tau standard curve was prepared as described in Section 10.



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 \pm SD, n=2). The mean Tau concentration was determined to be 2,500 pg/mL in SH-SY5Y mock treated extract, 2,233 pg/mL in SH-SY5Y staurosporine treated extract and 1,849 pg/mL in human brain extract. Staurosporine treatment induces Tau cleavage. [ab269557](#), the cleaved Tau fragment can be specifically measured using [ab269557](#), Cleaved Tau ELISA Kit (Human Asp738/Mouse Asp713).

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recombinant antibodies

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Recombinant technology

Success from the first experiment
Confirmed specificity

Ethical standards compliant
Animal-free production

Sandwich ELISA - Human Tau ELISA
Kit, Fluorescent (ab229394)

To learn more about the advantages of recombinant antibodies see [here](#).

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