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Immunotag™ Tau Polyclonal Antibody

Antibody Specification

Catalog No.	ITT4550
Product Description	Immunotag™ Tau Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	Tau
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	Western Blot: 1/500 - 1/2000. ELISA: 1/20000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat
Host Species	Rabbit
Immunogen	Synthesized peptide derived from Tau, at AA range: 240-320
Specificity	Tau Polyclonal Antibody detects endogenous levels of Tau protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	MAPT
Accession No.	P10636 P10637 P19332
Alternate Names	MAPT; MAPTL; MTBT1; TAU; Microtubule-associated protein tau; Neurofibrillary tangle protein; Paired helical filament-tau; PHF-tau

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Description	microtubule associated protein tau(MAPT) Homo sapiens 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],
Cell Pathway/ Category	MAPK_ERK_Growth,MAPK_G_Protein,Alzheimer's disease,
Protein Expression	Brain,Brain cortex,Epithelium,Fetal brain,Fetal brain cortex,
Subcellular Localization	cytoplasm,cytosol,microtubule,microtubule associated complex,plasma membrane,axoneme,postsynaptic density,axon,dendrite,growth cone,nuclear periphery,cytoplasmic ribonucleoprotein granule,cell body,

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Protein Function	Additional isoforms seem to exist. Isoforms differ from each other by the presence or absence of up to 5 of the 15 exons. One of these optional exons contains the additional tau/MAP repeat,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.,disease: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.,disease:Defects in MAPT are a cause of frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP17) [MIM:600274, 172700]; also called frontotemporal dementia (FTD) 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.,disease:Defects in MAPT are a cause of pallido-ponto-nigral degeneration (PPND) [MIM:168610]. The clinical features include ocular motility abnormalities, dystonia and urinary incontinence, besides progressive parkinsonism and dementia.,disease:Defects in MAPT are a cause of progressive supranuclear palsy (PSP) [MIM:601104, 260540]; also known as Steele-Richardson-Olszewski syndrome. PSP 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.,disease:Defects in MAPT may be a cause of hereditary dysphasic disinhibition dementia (HDDD) [MIM:607485]. HDDD is a frontotemporal dementia characterized by progressive cognitive deficits with memory loss and personality changes, severe dysphasic disturbances leading to mutism, and hyperphagia.,disease: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).,domain:The tau/MAP repeat binds to tubulin. Type I isoforms contain 3 repeats while type II isoforms contain 4 repeats.,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.,online information:Tau mutations,online information:Tau protein entry,online information:Vita minima - Issue 68 of March 2006,PTM: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.,PTM:Phosphorylation at serine and threonine residues in S-P or T-P motifs by proline-directed protein kinases (PDPK: CDC2, 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.,PTM:Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the

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