

Product Information

Anti-phospho-Tau (pSer⁴⁰⁰)

produced in rabbit, affinity isolated antibody

Catalog Number **T1700**

Product Description

Anti-phospho-Tau (pSer⁴⁰⁰) is produced in rabbit using as immunogen a synthetic phosphopeptide derived from a region of human Tau that contains Ser⁴⁰⁰. The sequence is conserved in many species including mouse, rat, baboon, rhesus monkey, cow, and goat. The serum is affinity purified using epitope-specific affinity chromatography. The antibody is preadsorbed to remove any reactivity towards a non-phosphorylated tau.

Anti-phospho-Tau (pSer⁴⁰⁰) recognizes human Tau. Mouse and rat, 100% homologous, have not been tested but are expected to cross-react. The antibody has been used in immunoblotting applications.¹

Tau is a neuronal microtubule-associated protein found predominantly on axons. The function of tau is to promote tubulin polymerization and stabilize microtubules. Tau, in its hyperphosphorylated form, is the major component of paired helical filaments (PHF), the building block of neurofibrillary lesions in Alzheimer's disease (AD) brain. Hyperphosphorylated tau is also found in neurofibrillary lesions in a range of other central nervous system disorders. Hyperphosphorylation impairs the microtubule binding function of tau, resulting in the destabilization of microtubules in AD brains, ultimately leading to the degeneration of the affected neurons.

Numerous serine/threonine kinases, including GSK-3 β , protein kinase A (PKA), cyclin-dependent kinase 5 and casein kinase II, phosphorylate tau. To date, a total of 25 abnormal phosphorylation sites have been identified on hyperphosphorylated Tau in AD brain. Normal Tau has ~8 phosphorylation sites. The abnormal phosphorylation occurs usually on serine and threonine residues. Specifically, TPKII phosphorylates serines 202 and 404. GSK-3 β transfection phosphorylates serines 199, 202, 235, 396, 404 and 413, and threonines 205 and 231. These sites are among the major abnormal phosphorylation sites of tau. Phosphorylation on these sites reduces the ability of given tau species to promote microtubule self-assembly. Ser⁴⁰⁰ is phosphorylated by GSK-3 β *in vitro* and *in vivo*.

Reagent

Supplied as a solution in Dulbecco's phosphate buffered saline (without Mg²⁺ and Ca²⁺), 50% glycerol with 1 mg/mL BSA (IgG, protease free), and 0.05% sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at -20 °C. For extended storage, upon initial thawing, freeze in working aliquots. Avoid repeated freezing and thawing to prevent denaturing the antibody. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: A minimum working dilution of 1:1,000 is recommended using recombinant human tau treated with GSK-3 β .

Note: In order to obtain best results in various techniques and preparations, we recommend determining optimal working concentration by titration.

References

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2. Sawamura, N., et al., Site-specific phosphorylation of tau accompanied by activation of mitogen-activated protein kinase (MAPK) in brains of Niemann-Pick type C mice. *J. Biol. Chem.* **276**, 10314-10319 (2001).
3. Mandelkow, E., Alzheimer's disease. The tangled tale of tau. *Nature*, **402**, 588-589 (1999).
4. Sontag, E., et al., Molecular interactions among protein phosphatase 2A, tau, and microtubules. Implications for the regulation of tau phosphorylation and the development of tauopathies. *J. Biol. Chem.*, **274**, 25490-25498 (1999).

5. Wang, J.Z., et al., Tau is phosphorylated by GSK-3 at several sites found in Alzheimer disease and its biological activity markedly inhibited only after it is prephosphorylated by Akinase. *FEBS Lett.* **436**, 28-34 (1998).
6. Papasozomenos, S.C., et al., Testosterone prevents the heat shock-induced overactivation of glycogen synthase kinase-3 β but not of cyclin-dependent kinase 5 and c-Jun NH2-terminal kinase and concomitantly abolishes hyperphosphorylation of tau: implications for Alzheimer's disease. *Proc. Natl. Acad. Sci. USA*, **99**, 1140-1145 (2002).

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