SANTA CRUZ BIOTECHNOLOGY, INC.

p-Tau (Ser 235): sc-101812

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The Power to Question

BACKGROUND

Tau can be phosphorylated by several protein kinases. Phosphorylation by Tau protein kinase II at Serine 404 is primarily responsible for the functional loss of Tau-mediated Tubulin polymerization. In addition, phosphorylation of microtubule-associated Tau results in the dissociation of Tau from the microtubules and Tubulin depolymerization. Serine 412 of Tau is modified by Tau protein kinase I/glycogen synthase kinase-3 β (TPKI/GSK-3β) to disrupt neuronal metabolism in anatomical areas vulnerable to Alzheimer's disease. TPKI/GSK-3β is expressed primarily in neurons and especially in neurites early in development, whereafter the distribution is concentrated mostly in the cell soma and the proximal neurite region.

REFERENCES

1. Tashiro, K., et al. 1997. Somatodendritic localization of phosphorylated Tau in neonatal and adult rat cerebral cortex. Neuroreport 8: 2797-2801.
2. Iqbal, K., et al. 1998. Mechanisms of neurofibrillary degeneration and the formation of neurofibrillary tangles. J. Neural Transm. Suppl. 53: 169-180.
3. Zhong, J., et al. 1999. Hyperphosphorylated Tau in SY5Y cells: similarities and dissimilarities to abnormally hyperphosphorylated Tau from Alzheimer disease brain. FEBS Lett. 453: 224-228.
4. Hashiguchi, M., et al. 2000. 14-3-3 ζ is an effector of Tau protein phosphorylation. J. Biol. Chem. 275: 25247-25254.
5. Lesort, M., et al. 2000. Insulin-like growth factor-1 and insulin mediate transient site-selective increases in Tau phosphorylation in primary cortical neurons. Neuroscience 99: 305-316.
6. Iqbal, K., et al. 2000. Mechanism of neurofibrillary degeneration and pharmacologic therapeutic approach. J. Neural Transm. Suppl. 59: 213-222.
7. Taniguchi, T., et al. 2001. Phosphorylation of Tau is regulated by PKN. J. Biol. Chem. 276: 10025-10031.
8. Alonso, A., et al. 2001. Hyperphosphorylation induces self-assembly of Tau into tangles of paired helical filaments/straight filaments. Proc. Natl. Acad. Sci. USA 98: 6923-6928.

CHROMOSOMAL LOCATION

Genetic locus: MAPT (human) mapping to 17q21.31; Mapt (mouse) mapping to 11 E1.

SOURCE

p-Tau (Ser 235) is a rabbit polyclonal antibody raised against a short amino acid sequence containing phosphorylated Ser 235 of Tau of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

p-Tau (Ser 235) is recommended for detection of Ser 235 phosphorylated Tau of human origin, correspondingly phosphorylated Ser 526 of mouse origin and correspondingly phosphorylated Ser 545 of rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1–2 µg per 100–500 µg of total protein (1 ml of cell lysate)].

Suitable for use as control antibody for Tau siRNA (h): sc-36614 and Tau siRNA (m): sc-36615; and as shRNA Plasmid control antibody for Tau shRNA Plasmid (h): sc-36614-SH and Tau shRNA Plasmid (m): sc-36615-SH.

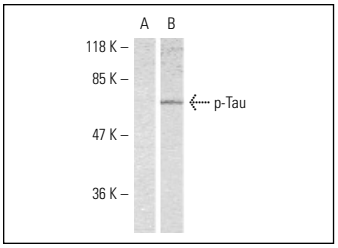
Molecular Weight of p-Tau: 46-68 kDa.

Positive Controls: mouse brain tissue extract.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible goat anti-rabbit IgG-HRP: sc-2030 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto B Blocking Reagent: sc-2335 (use 50 mM NaF, sc-24988, as diluent) and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



Western blot analysis of phosphorylated Tau expression in mouse brain tissue whole cell lysates. Blots were probed with p-Tau (Ser 235): sc-101812 preincubated with cognate phosphorylated peptide (**A**) and p-Tau (Ser 235): sc-101812 (**B**).

SELECT PRODUCT CITATIONS

1. Ding, Y., et al. 2008. Retinoic acid attenuates β-amyloid deposition and rescues memory deficits in an Alzheimer's disease transgenic mouse model. J. Neurosci. 28: 11622-11634.
2. Ding, Y., et al. 2010. Indirubin-3'-monoxime rescues spatial memory deficits and attenuates β-amyloid-associated neuropathology in a mouse model of Alzheimer's disease. Neurobiol. Dis. 39: 156-168.

RESEARCH USE

For research use only, not for use in diagnostic procedures.