



Source

Monoclonal Anti-Human Phospho-Tau (Thr181) Antibody, Mouse IgG2a (8D8C10) is a Mouse monoclonal antibody produced from a hybridoma created by fusing SP2/0 myeloma and Mouse B-lymphocytes.

Clone

8D8C10

Species

Mouse

Isotype

Mouse IgG2a | Mouse Kappa

Conjugate

Unconjugated

Antibody Type

Hybridoma Monoclonal

Reactivity

Human

Immunogen

Recombinant phosphorylated Tau181(pTau181) polypeptide.

Specificity

Specifically recognizes Human p-Tau181 Protein.

Application

Application	Recommended Usage
IF	1:50-1:500

Purity

>95% as determined by SDS-PAGE.

Purification

Protein A purified/ Protein G purified

Formulation

Lyophilized from 0.22 µm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

Reconstitution

Please see Certificate of Analysis for specific instructions.

For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.

Storage

For long term storage, the product should be stored at lyophilized state at -20°C or lower.

Please avoid repeated freeze-thaw cycles.

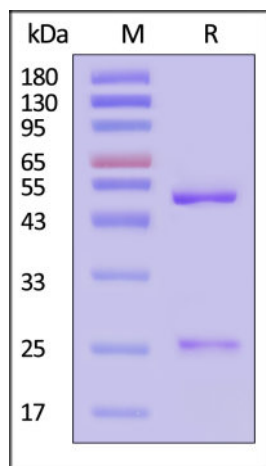
This product is stable after storage at:

- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

SDS-PAGE

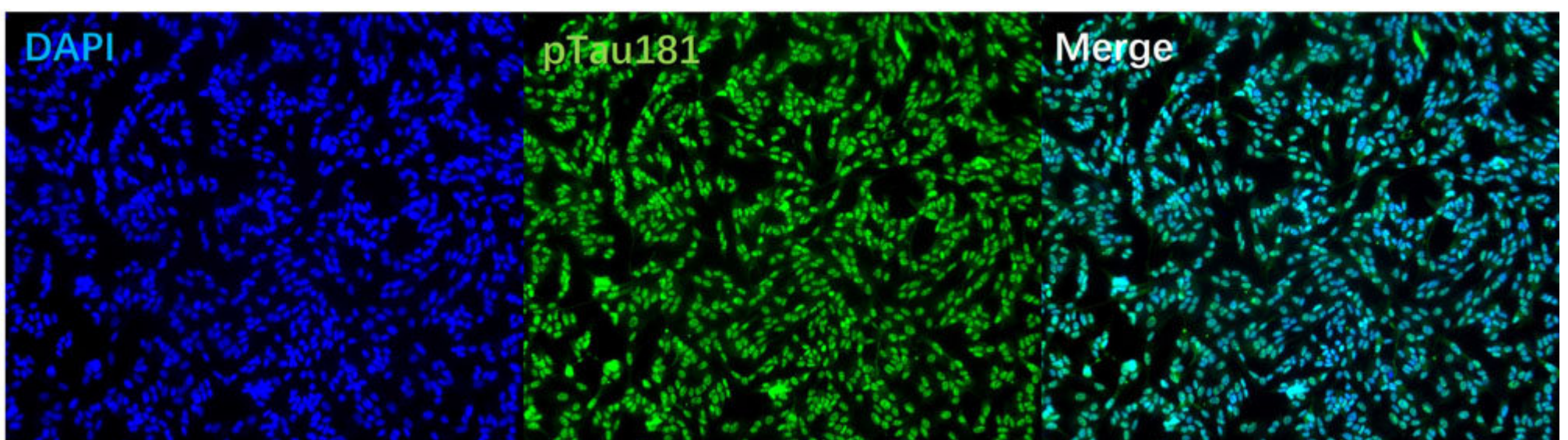
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Monoclonal Anti-Human Phospho-Tau (Thr181) Antibody, Mouse IgG2a (8D8C10) on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 95% (With [Star Ribbon Pre-stained Protein Marker](#)).

Immunofluorescence



2D cell staining: Immunofluorescent staining (10X) of phosphorylated tau in treated SH-SY5Y neuroblastoma cells with purified PT1-Y2073 at 1:200 dilution. DAPI (blue) was used as nuclear counterstain.

Background

Tau, the microtubule-associated protein, forms insoluble filaments that accumulate as neurofibrillary tangles in Alzheimer's disease (AD) and related tauopathies. Under physiological conditions, tau regulates the assembly and maintenance of the structural stability of microtubules. In the diseased brain, however, tau becomes abnormally hyperphosphorylated, which ultimately causes the microtubules to disassemble, and the free tau molecules aggregate into paired helical filaments.

Clinical and Translational Updates

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