SANTA CRUZ BIOTECHNOLOGY, INC.

PrP (6D11): sc-58581

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Prion diseases, or transmissible spongiform encephalopathies (TSEs), are manifested as genetic, infectious or sporadic, lethal neurodegenerative disorders involving alterations of the prion protein (PrP). Characteristic of prion diseases, cellular PrP (PrPc) is converted to the disease form, PrPSc, through alterations in the protein folding conformations. PrPc is constitutively expressed in normal adult brain and is sensitive to proteinase K digestion, while the altered PrPSc conformation is resistant to proteases, resulting in a distinct molecular mass after PK treatment. Consistent with the transient infection process of prion diseases, incubation of PrPc with PrPSc both *in vitro* and *in vivo* produces PrPc that is resistant to protease degradation. Infectious PrPSc is found at high levels in the brains of animals affected by TSEs, including scrapie in sheep, BSE in cattle and Cruetzfeldt-Jacob disease in humans.

REFERENCES

1. Bessen, R.A. and Marsh, R.F. 1992. Biochemical and physical properties of the prion protein from two strains of the transmissible mink encephalopathy agent. J. Virol. 66: 2096-2101.
2. Bessen, R.A., et al. 1995. Non-genetic propagation of strain-specific properties of scrapie prion protein. Nature 375: 698-700.
3. Weiss, S., et al. 1996. Recombinant prion protein rPrP27-30 from Syrian golden hamster reveals proteinase K sensitivity. Biochem. Biophys. Res. Commun. 219: 173-179.

CHROMOSOMAL LOCATION

Genetic locus: PRNP (human) mapping to 20p13; Prnp (mouse) mapping to 2 F2.

SOURCE

PrP (6D11) is a mouse monoclonal antibody rasied against non-denatured prion protein (PrP) of mouse origin.

PRODUCT

Each vial contains 50 µg IgG_{2a} in 0.5 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

PrP (6D11) is recommended for detection of PrPc and PrPsc of mouse, rat, human and bovine origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for PrP siRNA (h): sc-36318, PrP siRNA (m): sc-36319, PrP shRNA Plasmid (h): sc-36318-SH, PrP shRNA Plasmid (m): sc-36319-SH, PrP shRNA (h) Lentiviral Particles: sc-36318-V and PrP shRNA (m) Lentiviral Particles: sc-36319-V.

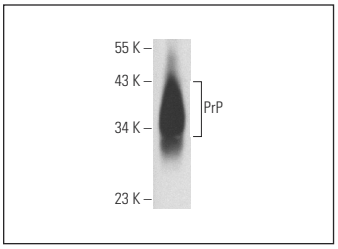
Molecular Weight of PrP: 30 kDa.

Positive Controls: mouse brain extract: sc-2253.

STORAGE

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

DATA



PrP (6D11): sc-58581. Western blot analysis of PrP expression in mouse brain tissue extract.

SELECT PRODUCT CITATIONS

1. Hughes, M.M., et al. 2010. Microglia in the degenerating brain are capable of phagocytosis of beads and of apoptotic cells, but do not efficiently remove PrPSc, even upon LPS stimulation. Glia 58: 2017-2030.
2. Fan, X.Y., et al. 2015. Activation of the AMPK-ULK1 pathway plays an important role in autophagy during prion infection. Sci. Rep. 5: 14728.
3. Wei, W., et al. 2016. Expression of prion protein is closely associated with pathological and clinical progression and abnormalities of p53 in head and neck squamous cell carcinomas. Oncol. Rep. 35: 817-824.
4. Xiao, K., et al. 2016. Re-infection of the prion from the scrapie-infected cell line SMB-S15 in three strains of mice, CD1, C57BL/6 and Balb/c. Int. J. Mol. Med. 37: 716-726.
5. Wang, J., et al. 2016. Treatment of SMB-S15 cells with resveratrol efficiently removes the PrP(Sc) accumulation *in vitro* and prion infectivity *in vivo*. Mol. Neurobiol. 53: 5367-5376.
6. Wang, T.T., et al. 2016. Down-regulation of brain-derived neurotrophic factor and its signaling components in the brain tissues of scrapie experimental animals. Int. J. Biochem. Cell Biol. 79: 318-326.
7. Xu, Y., et al. 2016. FBXW7-induced MTOR degradation forces autophagy to counteract persistent prion infection. Mol. Neurobiol. 53: 706-719.
8. Wang, H., et al. 2016. Overexpression of PLK3 mediates the degradation of abnormal prion proteins dependent on chaperone-mediated autophagy. Mol. Neurobiol. E-published.

RESEARCH USE

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

CONJUGATES

See **PrP (5B2): sc-47730** for PrP antibody conjugates, including AC, HRP, FITC, PE, Alexa Fluor® 488 and Alexa Fluor® 647.