SANTA CRUZ BIOTECHNOLOGY, INC.

PrP (FL-253): sc-15312

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.

CHROMOSOMAL LOCATION

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

SOURCE

PrP (FL-253) is a rabbit polyclonal antibody raised against amino acids 1-253 representing full length PrP of human origin.

PRODUCT

Each vial contains 200 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.

RESEARCH USE

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

APPLICATIONS

PrP (FL-253) is recommended for detection of PrP of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

PrP (FL-253) is also recommended for detection of PrP in additional species, including equine, canine and bovine.

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.

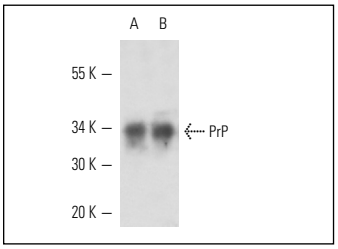
Molecular Weight of PrP: 30 kDa.

Positive Controls: mouse brain extract: sc-2253 or rat brain extract: sc-2392.

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 A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use goat anti-rabbit IgG-FITC: sc-2012 (dilution range: 1:100-1:400) or goat anti-rabbit IgG-TR: sc-2780 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

DATA



PrP (FL-253): sc-15312. Western blot analysis of PrP expression in rat brain (**A**) and mouse brain (**B**) tissue extracts.

SELECT PRODUCT CITATIONS

1. Morel, E., et al. 2004. The cellular prion protein PrPc is expressed in human enterocytes in cell-cell junctional domains. J. Biol. Chem. 279: 1499-1505.
2. Shyu, W.C., et al. 2004. Hyperbaric oxygen enhances the expression of prion protein and heat shock protein 70 in a mouse neuroblastoma cell line. Cell. Mol. Neurobiol. 24: 257-268.
3. Wong, H.K., et al. 2005. β subunits of voltage-gated sodium channels are novel substrates of β-site amyloid precursor protein-cleaving enzyme (BACE1) and γ-secretase. J. Biol. Chem. 280: 23009-23017.
4. Robertson, C., et al. 2006. Cellular prion protein is released on exosomes from activated platelets. Blood 107: 3907-3911.
5. Corsaro, A., et al. 2011. High hydrophobic amino acid exposure is responsible of the neurotoxic effects induced by E200K or D202N disease-related mutations of the human prion protein. Int. J. Biochem. Cell Biol. 43: 372-382.