SANTA CRUZ BIOTECHNOLOGY, INC.

PrP siRNA (h): sc-36318

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 ovine, BSE in cattle and Cruetzfeldt-Jakob 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.

PRODUCT

PrP siRNA (h) is a pool of 3 target-specific 19-25 nt siRNAs designed to knock down gene expression. Each vial contains 3.3 nmol of lyophilized siRNA, sufficient for a 10 µM solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see PrP shRNA Plasmid (h): sc-36318-SH and PrP shRNA (h) Lentiviral Particles: sc-36318-V as alternate gene silencing products.

For independent verification of PrP (h) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-36318A, sc-36318B and sc-36318C.

STORAGE AND RESUSPENSION

Store lyophilized siRNA duplex at -20° C with desiccant. Stable for at least one year from the date of shipment. Once resuspended, store at -20° C, avoid contact with RNAses and repeated freeze thaw cycles.

Resuspend lyophilized siRNA duplex in 330 µl of the RNAse-free water provided. Resuspension of the siRNA duplex in 330 µl of RNAse-free water makes a 10 µM solution in a 10 µM Tris-HCl, pH 8.0, 20 mM NaCl, 1 mM EDTA buffered solution.

APPLICATIONS

PrP siRNA (h) is recommended for the inhibition of PrP expression in human cells.

SUPPORT REAGENTS

For optimal siRNA transfection efficiency, Santa Cruz Biotechnology's siRNA Transfection Reagent: sc-29528 (0.3 ml), siRNA Transfection Medium: sc-36868 (20 ml) and siRNA Dilution Buffer: sc-29527 (1.5 ml) are recommended. Control siRNAs or Fluorescein Conjugated Control siRNAs are available as 10 µM in 66 µl. Each contain a scrambled sequence that will not lead to the specific degradation of any known cellular mRNA. Fluorescein Conjugated Control siRNAs include: sc-36869, sc-44239, sc-44240 and sc-44241. Control siRNAs include: sc-37007, sc-44230, sc-44231, sc-44232, sc-44233, sc-44234, sc-44235, sc-44236, sc-44237 and sc-44238.

GENE EXPRESSION MONITORING

PrP (5B2): sc-47730 is recommended as a control antibody for monitoring of PrP gene expression knockdown by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) or immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor PrP gene expression knockdown using RT-PCR Primer: PrP (h)-PR: sc-36318-PR (20 µl, 445 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

SELECT PRODUCT CITATIONS

1. Déry, M.A., et al. 2013. Endoplasmic reticulum stress induces PRNP prion protein gene expression in breast cancer. Breast Cancer Res. 15: R22.
2. Cheng, Y., et al. 2014. CD44/cellular prion protein interact in multidrug resistant breast cancer cells and correlate with responses to neoadjuvant chemotherapy in breast cancer patients. Mol. Carcinog. 53: 686-697.
3. Park, J.Y., et al. 2015. Induction of cellular prion protein (PrPc) under hypoxia inhibits apoptosis caused by TRAIL treatment. Oncotarget 6: 5342-5353.
4. Asthana, A., et al. 2017. Prion protein facilitates retinal iron uptake and is cleaved at the β-site: implications for retinal iron homeostasis in prion disorders. Sci. Rep. 7: 9600.
5. Déry, M.A. and LeBlanc, A.C. 2017. Luman contributes to brefeldin A-induced prion protein gene expression by interacting with the ERSE26 element. Sci. Rep. 7: 42285.
6. Ashok, A. and Singh, N. 2018. Prion protein modulates glucose homeostasis by altering intracellular iron. Sci. Rep. 8: 6556.
7. Ashok, A., et al. 2019. Prion protein modulates endothelial to mesenchyme-like transition in trabecular meshwork cells: Implications for primary open angle glaucoma. Sci. Rep. 9: 13090.
8. Silva De Castro, I., et al. 2023. HTLV-1 p12 modulates the levels of prion protein (PrPC) in $CD4^+$ T cells. Front. Microbiol. 14: 1175679.

RESEARCH USE

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