SANTA CRUZ BIOTECHNOLOGY, INC.

BACE (C-15): sc-10055

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Autosomal dominant Alzheimer's disease is caused by mutations in the gene encoding the β-amyloid protein precursor (APP). Amyloid β-peptide (Aβ), the major feature of amyloid plaques in Alzheimer's patients, is the product of APP cleavage by β- and γ-secretases. BACE is the transmembrane protease which cleaves Aβ from APP. BACE and the related protein Asp1 are both widely expressed in human tissue with the highest levels in the pancreas. BACE is localized within Golgi and endosomes

REFERENCES

1. Kang, J., et al. 1987. The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor. Nature 325: 733-736.
2. Goate, A., et al. 1991. Segregation of a missense mutation in the amyloid precursor protein gene with familial Alzheimer's disease. Nature 349: 704-706.
3. Mullan, M., et al. 1992. A pathogenic mutation for probable Alzheimer's disease in the APP gene at the N-terminus of β-amyloid. Nat. Genet. 1: 345-347.
4. Selkoe, D.J. 1998. The cell biology of β-amyloid precursor protein and presenilin in Alzheimer's disease. Trends. Cell Biol. 8: 447-453.
5. Yan, R., et al. 1999. Membrane-anchored aspartyl protease with Alzheimer's disease β-secretase activity. Nature 402: 533-537.
6. Vassar, R., et al. 1999. Beta-secretase cleavage of Alzheimer's amyloid precursor protein by the transmembrane aspartic protease BACE. Science 286: 735-741.
7. Hussain, I., et al. 1999 Identification of a novel aspartic protease (Asp 2) as β-secretase Molec. Cell Neurosci. 14: 419-427.

CHROMOSOMAL LOCATION

Genetic locus: BACE1 (human) mapping to 11q23.3; Bace1 (mouse) mapping to 9 A5.2.

SOURCE

BACE (C-15) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of BACE 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.

Blocking peptide available for competition studies, sc-10055 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

APPLICATIONS

BACE (C-15) is recommended for detection of BACE 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).

BACE (C-15) is also recommended for detection of BACE in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for BACE siRNA (h): sc-37224, BACE siRNA (m): sc-37225, BACE shRNA Plasmid (h): sc-37224-SH, BACE shRNA Plasmid (m): sc-37225-SH, BACE shRNA (h) Lentiviral Particles: sc-37224-V and BACE shRNA (m) Lentiviral Particles: sc-37225-V.

Molecular Weight of BACE: 70 kDa.

Positive Controls: SH-SY5Y cell lysate: sc-3812.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (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 donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Fuso, A., et al. 2007. γ-secretase is differentially modulated by alterations of homocysteine cycle in neuroblastoma and glioblastoma cells. J. Alzheimers Dis. 11: 275-290.
2. Fuso, A., et al. 2008. B-vitamin deprivation induces hyperhomocysteinemia and brain S-adenosylhomocysteine, depletes brain S-adenosylmethionine, and enhances PS1 and BACE expression and Amyloid-β deposition in mice. Mol. Cell. Neurosci. 37: 731-746.

RESEARCH USE

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