SANTA CRUZ BIOTECHNOLOGY, INC.

Aph-1 (H-50): sc-98469

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Four proteins comprise the γ-secretase complex: Presenilin, nicastrin, Aph-1 and PEN-2. Together, these proteins mediate cell surface signaling pathways for a variety of type I membrane proteins, notably β-Amyloid precursor protein, a protein implicated in the development of Alzheimer's disease, via intramembrane proteolysis. The proteins assemble into a proteolytically active complex in the Golgi/*trans*-Golgi network (TGN) compartments. Assembly leads to autocleavage of Presenilin into two subunits to create the active site of γ-secretase, an important step in understanding the mechanisms involved in the etiology and possible treatment of Alzheimer's disease.

REFERENCES

1. Kimberly, W.T. and Wolfe, M.S. 2003. Identity and function of γ-secretase. J. Neurosci. Res. 74: 353-360.
2. Baulac, S., et al. 2003. Functional γ-secretase complex assembly in Golgi/*trans*-Golgi network: interactions among Presenilin, nicastrin, Aph-1, PEN-2, and γ-secretase substrates. Neurobiol. Dis. 14: 194-204.
3. Wolfe, M.S. 2003. γ-secretase—intramembrane protease with a complex. Sci. Aging Knowledge Environ. 11: 7.
4. Fortna, R.R., et al. 2004. Membrane topology and nicastrin-enhanced endoproteolysis of Aph-1, a component of the γ-secretase complex. J. Biol. Chem. 279: 3685-3693.
5. Shirotani, K., et al. 2004. Identification of distinct γ-secretase complexes with different Aph-1 variants. J. Biol. Chem. 279: 41340-41345.
6. Hansson, E.M., et al. 2005. Aph-1 interacts at the cell surface with proteins in the active γ-secretase complex and membrane-tethered Notch. J. Neurochem. 92: 1010-1020.
7. Ma, G., et al. 2005. Aph-1a is the principal mammalian Aph-1 isoform present in γ-secretase complexes during embryonic development. J. Neurosci. 25: 192-198.

CHROMOSOMAL LOCATION

Genetic locus: APH1A (human) mapping to 1q21.2; Aph1a (mouse) mapping to 3 F2.1.

SOURCE

Aph-1 (H-50) is a rabbit polyclonal antibody raised against amino acids 181-230 mapping near the C-terminus of Aph-1 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

Aph-1 (H-50) is recommended for detection of Aph-1 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).

Aph-1 (H-50) is also recommended for detection of Aph-1 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for Aph-1 siRNA (h): sc-105081, Aph-1 siRNA (m): sc-141150, Aph-1 shRNA Plasmid (h): sc-105081-SH, Aph-1 shRNA Plasmid (m): sc-141150-SH, Aph-1 shRNA (h) Lentiviral Particles: sc-105081-V and Aph-1 shRNA (m) Lentiviral Particles: sc-141150-V.

Molecular Weight of Aph-1: 18 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200.

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.

SELECT PRODUCT CITATIONS

1. Sutinen, E.M., et al. 2012. Pro-inflammatory interleukin-18 increases Alzheimer's disease-associated amyloid-β production in human neuron-like cells. J. Neuroinflammation 9: 199.

PROTOCOLS

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