

β-Amyloid (H-43): sc-9129

BACKGROUND

Proteolytic cleavage of the amyloid protein precursor (APP) gives rise to the β-Amyloid and Amyloid A4 proteins, which are present in human platelets. Amyloid deposition is associated with type II diabetes, Down's syndrome and a variety of neurological disorders, including Alzheimer's disease. The amyloid precursor protein (APP) undergoes alternative splicing, resulting in several isoforms. Proteolytic cleavage of APP leads to the formation of the 4 kDa Amyloid-β/A4 protein. This protein is involved in the formation of neurofibrillary tangles and plaques that characterize the senile plaques of Alzheimer patients. APLP1 (amyloid precursor-like protein 1) and APLP2 are structurally similar to APP. Human APLP2 is a membrane-bound sperm protein that contains a region highly homologous to the transmembrane-cytoplasmic domains of APP found in brain plaques of Alzheimer disease patients.

REFERENCES

1. Kosik, K.S. 1992. Alzheimer's disease: a cell perspective. *Science* 256: 780-783.
2. Dyrks, T., et al. 1993. Generation of β A4 from the amyloid protein precursor and fragments thereof. *FEBS Lett.* 335: 89-93.
3. Hirai, S. and Okamoto, K. 1993. Amyloid-β/A4 peptide associated with Alzheimer's disease and cerebral amyloid angiopathy. *Intern. Med.* 32: 923-925.
4. Arendt, T., et al. 1995. Paired helical filament-like phosphorylation of tau, deposition of β/A4-Amyloid and memory impairment in rat induced by chronic inhibition of phosphatase 1 and 2A. *Neuroscience* 69: 691-698.
5. Gillmore, J.D., et al. 1997. Amyloidosis: a review of recent diagnostic and therapeutic developments. *British J. of Haematol.* 99: 245-256.

CHROMOSOMAL LOCATION

Genetic locus: APP (human) mapping to 21q21.3; App (mouse) mapping to 16 C3-qter.

SOURCE

β-Amyloid (H-43) is a rabbit polyclonal antibody raised against amino acids 672-714 of Amyloid A4 representing full length β-Amyloid protein 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

β-Amyloid (H-43) is recommended for detection of 4 kDa β-Amyloid and Amyloid A4 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).

Suitable for use as control antibody for β-Amyloid siRNA (h): sc-29677 and β-Amyloid siRNA (m): sc-29678.

Molecular Weight of β-Amyloid: 4-46 kDa (various forms).

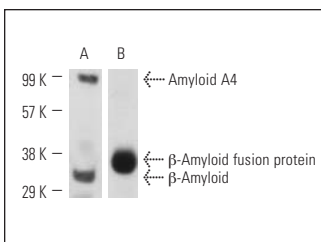
Molecular Weight of Amyloid A4: 100-125 kDa (various forms).

Positive Controls: H4 cell lysate: sc-2408, 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



β-Amyloid (H-43): sc-9129. Western blot analysis of β-Amyloid expression in H4 whole cell lysate (A) and human recombinant β-Amyloid fusion protein (B).

SELECT PRODUCT CITATIONS

1. Wolvetang, E.W., et al. 2003. The chromosome 21 transcription factor Ets-2 transactivates the β-APP promoter: implications for Down syndrome. *Biochem. Biophys. ACTA Rev.* 1628: 105-110.
2. Holginger, G.U., et al. 2005. The mitochondrial complex I inhibitor rotenone triggers a cerebral tauopathy. *J. Neurochem.* 95: 930-939.
3. Tang, K., et al. 2006. Platelet amyloid precursor protein processing: a biomarker for Alzheimer's disease. *J. Neurol. Sci.* 240: 53-58.