beta Amyloid (Abeta) (N-terminal). Rabbit Antigen Immunoaffinity Purified Polyclonal

APP, ABPP, Alzheimer disease amyloid protein, Cerebral vascular amyloid peptide, CVAP, Protease nexin-II, PN-II, APPI, PreA4

Background

Beta-amyloid peptides are lipophilic metal chelators with metal-reducing activity. Bind transient metals such as copper, zinc and iron. In vitro, can reduce Cu(2+) and Fe(3+) to Cu(+) and Fe(2+), respectively. Beta-amyloid 42 is a more effective reductant than beta-amyloid 40. Beta-amyloid peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma, inhibiting metal-catalyzed oxidation of lipoproteins. Beta-APP42 may activate mononuclear phagocytes in the brain and elicit inflammatory responses. Promotes both tau aggregation and TPK II-mediated phosphorylation. Interaction with overexpressed HADH2 leads to oxidative stress and neurotoxicity.

Ordering Information

Catalog Number
X1884P

Size
50 µg

Form
Unconjugated

Host/Clone
Rabbit

Formulation
Purified IgG in phosphate buffered saline, pH 7.4

Concentration
See vial for concentration

Isotype
IgG

Applications
Dot Blot, Radio Immunoassay, Enzyme Immunoassay

Species Reactivity
Human

Accession Number
P05067, Human

Immunogen

Synthetic peptide derived from the N-terminal region of the amyloid beta peptide.

Positive Control/Tissue Expression

No cross-reactivity against a negative spanning region of Aβ has been observed by ELISA.

Comments

For dot blot applications, we recommend using the antibody at 1.0 µg/mL.Optimal concentration should be evaluated by serial dilutions.

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PURIFICATION

Purified from rabbit serum by epitope-specific affinity chromatography.

SHIP CONDITIONS

Ship on dry ice, freeze upon arrival

STORAGE CUSTOMER

Product should be stored at -70ºC. Aliquot to avoid freeze/thaw cycles

STABILITY

Products are stable for one year from purchase when stored properly

REFERENCES

1. Head, E., Moffat, K., Das, P., Sarsoza, F., Poon, W.W., Landsberg, G., Cotman, C.W., Murphy, M.P. (2005) β-Amyloid deposition and tau phosphorylation in clinically characterized aged cats. Neurobiol Aging. 26(5): 749-763.

2. Borchelt, D.R. et al. (1997) Accelerated amyloid deposition in the brains of transgenic mice coexpressing mutant presenilin 1 and amyloid precursor proteins. Neuron 19:939-945.

3. Savage, M.J. et al. (1998) Turnover of amyloid β-protein in mouse brain and acute reduction of its level by phorbol ester. J. Neurosci. 18:1743-1752.

PRODUCT SPECIFIC REFERENCES

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