



3050 Spruce Street  
Saint Louis, Missouri 63103 USA  
Telephone (800) 325-5832 (314) 771-5765  
Fax (314) 286-7828  
email: techserv@sial.com  
sigma-aldrich.com

## Product Information

### Anti-Amyloid Peptide $\beta$ , Cleavage Site 42

Developed in Rabbit, Affinity Isolated Antibody

Product Number **A 1976**

#### Product Description

Anti-Amyloid Peptide  $\beta$ , Cleavage Site 42 (A $\beta$ 42) is developed in rabbit using as immunogen a synthetic peptide of approximately 10 amino acid residues at the C-terminus of A $\beta$ 42. The antibody is cleavage site specific and its binding to the A $\beta$  peptide requires the presence of the free carboxyl group at the C-terminus of the peptide. It does not bind to Amyloid Precursor Protein (APP), which lacks this free carboxyl group. The rabbit serum is affinity purified using epitope-specific affinity chromatography. The antibody is preabsorbed to remove any reactivity towards full-length A $\beta$ 40 (1-40 amino acids) and amyloid peptide A $\beta$ 43 (1-43 amino acids).

Anti-A $\beta$ 42 specifically recognizes A $\beta$ , cleavage site 42. The antibody detects human, mouse and rat A $\beta$ 42. It has been used in RIA, ELISA<sup>1,2</sup>, dot blots, immunoprecipitation<sup>3</sup> and immunohistochemistry applications.<sup>4</sup>

Alzheimer's Disease (AD) is characterized by the deposition of extracellular plaques and intracellular neurofibrillary tangles (NFTs) in the brain. The major component of these senile extracellular plaques is the amyloid  $\beta$  peptide (A $\beta$  peptide), a 4 kDa peptide of 39-42 amino acid residues. This peptide is cleaved by caspases from amyloid precursor protein (APP) during apoptosis. Mutations in APP are the key triggers for the pathogenesis of AD. Increased amyloid  $\beta$  peptide formation leads to the elevated extracellular concentrations of the "longer forms" of A $\beta$  peptide, A $\beta$ 42 or A $\beta$ 43, a common effect of the mutations of genes in Alzheimer's disease. These peptides have a greater tendency to aggregate than A $\beta$ 40 and, therefore, are considered to be pathological.<sup>4,5</sup> The increased release of A $\beta$ 42/A $\beta$ 43 leads to the abnormal deposition of A $\beta$  and the associated neurotoxicity in the brains of affected individuals.<sup>6</sup>

#### Reagent

Anti-Amyloid Peptide  $\beta$ 42 is supplied as a solution in phosphate buffer, pH 7.4. One vial contains approximately 25  $\mu$ g of antibody.

#### Storage/Stability

Store at  $-70^{\circ}\text{C}$ . For extended storage, upon initial thawing, freeze in working aliquots. Do not store in frost-free freezers. Avoid repeated freezing and thawing to prevent denaturing the antibody. Samples at working dilution should be discarded if not used within 12 hours. The antibody is stable for at least 6 months when stored appropriately.

#### Product Profile

A recommended working concentration of 0.05 to 1.0  $\mu$ g/ml is determined by dot blot. In ELISA a 1:200 working dilution is recommended.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

#### References

1. Vassar, R., et al.,  $\beta$ -secretase cleavage of Alzheimer's amyloid precursor protein by the transmembrane aspartic protease BACE. *Science*, **286**, 735-741 (1999).
2. Savage, M. J., et al., Turnover of amyloid  $\beta$ -protein in mouse brain and acute reduction of its level by phorbol ester. *J. Neurosci.*, **18**, 1743-1752 (1998).
3. Russo, C., et al., Opposite roles of apolipoprotein E in normal brains and in Alzheimer's disease. *Proc. Natl. Acad. Sci. U S A.*, **95**, 15598-15602 (1998).
4. Borchelt, D.R., et al. Accelerated amyloid deposition in the brains of transgenic mice coexpressing mutant presenilin 1 and amyloid precursor proteins. *Neuron*, **19**, 939-945 (1997).
5. Cotman, C.W. The  $\beta$ -amyloid peptide, peptide self-assembly, and the emergence of biological activities. A new principle in peptide function and the induction of neuropathology. *Ann. N. Y. Acad. Sci.*, **814**, 1-16 (1997).

6. Sambamurti, K., et al. Advances in the cellular and molecular biology of the  $\beta$ -amyloid protein in Alzheimer's disease. *Neuromolecular Med.*, **1**, 1-31 (2002).
7. Lorenzo, A., et al., Amyloid  $\beta$  interacts with the amyloid precursor protein: a potential toxic mechanism in Alzheimer's disease. *Nat. Neurosci.*, **3**, 460-464 (2000).

AH 10/02

Sigma brand products are sold through Sigma-Aldrich, Inc.

Sigma-Aldrich, Inc. warrants that its products conform to the information contained in this and other Sigma-Aldrich publications. Purchaser must determine the suitability of the product(s) for their particular use. Additional terms and conditions may apply. Please see reverse side of the invoice or packing slip.