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

ProductInformation

Anti-Amyloid Peptide b, Cleavage Site 43
Developed in Rabbit, Affinity Isolated Antibody

Product Number **A 2101**

Product Description

Anti-Amyloid Peptide β, Cleavage Site 43 (Aβ43) is developed in rabbit using as immunogen a synthetic peptide of approximately 7-10 amino acid residues at the C-terminus of Aβ 43. The antibody is cleavage site specific and its binding to the Aβ peptide requires the presence of the free carboxyl group at the C- terminus of the peptide. The rabbit serum is affinity purified using epitope-specific affinity chromatography. The antibody is preabsorbed to remove any reactivity towards full-length Aβ40 (1-40 amino acids) and Aβ42 (1-42 amino acids). Anti- Aβ43 specifically recognizes Aβ, cleavage site 43. The antibody detects human and mouse Aβ43. It has been used in RIA, ELISA and dot blots applications.

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 Aβ peptide, a 4 kDa peptide of 39-43 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 Aβ peptide formation leads to the elevated extracellular concentrations of the "longer forms" of Aβ peptide, Aβ42 or Aβ43, a common effect of the mutations of genes in Alzheimer's disease. These peptides have a greater tendency to aggregate than Aβ40 and therefore are considered to be pathological.[1,2,3] In addition Aβ43 acts as an inhibitor of the ubiquitin-dependent protein degradation *in vitro.* The increased release of Aβ42/Aβ43 leads to the abnormal deposition of Aβ and the associated neurotoxicity in the brains of affected individuals.[6]

Reagent

Anti-Amyloid Peptide β43 is supplied as a solution in phosphate buffer, pH 7.4. One vial contains 25 µg of antibody.

Storage/Stability

Store at –70 °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. Working dilution samples 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 µg/ml is determined by dot blot.

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

References

1. 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).
2. Cotman, C.W. The β-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).
3. Sambamurti, K., et al. Advances in the cellular and molecular biology of the β-amyloid protein in Alzheimer's disease., Neuromolecular Med., **1**, 1-31 (2002).
4. Soreghan, B., et al. The influence of the carboxyl terminus of the Alzheimer Abeta peptide on its conformation, aggregation, and neurotoxic properties. Neuromolecular Med., 1,81-94 (2002)
5. Savage, M.J., et al. Turnover of amyloid β-protein in mouse brain and acute reduction of its level by phorbol ester. J. Neurosci., **18**, 1743-1752 (1998).
6. Lorenzo, A., et al., Amyloid β interacts with the amyloid precursor protein: a potential toxic mechanism in Alzheimer's disease. Nat. Neurosci., **3**, 460-464 (2000).

AH 10/02