3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Anti-β-Amyloid [13-28] antibody
Mouse monoclonal, clone BAM90.1
purified from hybridoma cell culture

Product Number **A8978**

Product Description

Anti-β-Amyloid [13-28] antibody, Mouse monoclonal (mouse IgG1 isotype) is derived from the BAM90.1 hybridoma produced by the fusion of mouse myeloma cells (NS1 cells) and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to a fragment of human β-Amyloid protein, conjugated to KLH . The isotype is determined by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2.

Anti-β-Amyloid [13-28] antibody, Mouse monoclonal recognizes the β-Amyloid peptide. The product is useful in ELISA, immunoblotting, immunoprecipitation, immunohistochemistry, and in *in vivo* sequestration of endogenous plasma human β-Amyloid peptide (1-40).

The β-amyloid precursor protein (APP) is cleaved sequentially by the proteolytic enzymes β-secretase and γ-secretase to produce β-amyloid (Aβ) peptides with the Aβ1-42 and the Aβ1-40 forms being the most prevalent. Secreted Aβ peptides are degraded either via a re-uptake mechanism followed by endosomal degradation, or by an extracellular insulin-degrading enzyme.

Extra cellular accumulation of Aβ leads to formation of aggregates, fibrils, and eventually amyloid deposits called neuritic plaques, a hallmark of Alzheimer's disease (AD).[1] Much of the AD research has focused on determining the underlying mechanism(s) of Aβ protein toxicity. Of the many proposed mechanisms, one possible mechanism of Aβ protein toxicity may be through calcium-mediated neurotoxicity. Aβ peptides can increase calcium influx through voltage-gated calcium channels (N- and L-type), reduce the magnesium blockade of NMDA receptors to allow increased calcium influx, and can form a cation-selective ion channel after their incorporation into the cell membrane.[2-4] Cation channels are induced by both nascent and globular Aβ peptides.[5] Thus, Aβ peptides may elicit toxic effects prior to fibril formation.

Recent evidence suggests that copper and zinc may modulate the structure of the pleimorphic Aβ peptides to induce either pore formation or peptide precipitation.[6] In other models it was found that the Aβ peptides exhibit superoxidase dismutase activity thus producing hydrogen peroxidase that may be responsible for neurotoxicity.[7]

Reagent

The product is supplied as a 0.2 μm filtered solution in 0.01 M phosphate buffered saline, pH 7.4.

Antibody Concentration: ~2 mg/mL

Storage/Stability

For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

ELISA: a working concentration of 0.2-.0.4 μg/mL is determined using Amyloid β-Protein Fragment 1-40 (Product Number A1075) as the antigen.

Note: In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration.

References

1. Law, A., et al., *Brain Res. Rev*., **35**, 73-96 (2001).
2. Pearson, H.A., in Alzheimer's Disease: Methods and Protocols, Hooper, N.M. (Ed.) pp. 113-138, Humana Press, NJ (2000).
3. Kawahara, M., and Kuroda, Y., *Brain Res. Bulletin*, **53**, 389-397 (2000).
4. Zhu, Y.J., et al., *FASEB J*., **14**, 1244-1254 (2000).
5. Bhatia, R., et al., *FASEB J*., **14**, 1233-1243 (2000).
6. Curtain, C.C., et al., *J. Biol. Chem*., **276**, 20466-20473 (2001)
7. Veurink, G., et al., *Ann. Hum. Biol*., **30**, 639-667 (2003).

VS,DS,PHC,MAM 08/19-1