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

MONOCLONAL ANTI-HUMAN β AMYLOID PROTEIN [17-24]
Clone 4G8
Purified Mouse Immunoglobulin

Product Number **A 1349**

Product Description

Monoclonal Anti-Human β Amyloid Protein [17-24] (mouse IgG2b isotype) is derived from the hybridoma produced by the fusion of mouse myeloma cells and splenocytes from a BALB/c mouse immunized with a synthetic peptide corresponding to amino acids 1-24 of the β amyloid peptide[1], with Glu substituted at position 11, conjugated to KLH. The immunoglobulin is isolated from mouse ascites fluid by Protein A chromatography.

Monoclonal Anti-Human β Amyloid Protein [17-24] recognizes amino acid residues 17-24 of the human β amyloid peptide by immunoblotting, ELISA, immunoaffinity purification, immunoprecipitation and immunohistochemistry on fixed sections. This antibody may react with mouse β amyloid protein at higher IgG and/or antigen concentrations. Monoclonal Anti-Human β Amyloid Protein [17-24] also recognizes the precursor forms and the abnormally processed isoforms.

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 $A\beta_{1-42}$ and the $A\beta_{1-40}$ forms being the most prevalent. Secreted Aβ peptides can bind to scavenger receptors and the receptor for advanced glycation end-products. Aβ peptides are degraded either via a reuptake mechanism followed by endosomal degradation or by an extracellular insulin-degrading enzyme. Extracellular 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 AD research has been focused on determining the underlying mechanism(s) of Aβ protein toxicity. One possible mechanism of Aβ protein toxicity is 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, lastly, they 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]

Reagent

Monoclonal Anti-Human β Amyloid Protein [17-24] is supplied as 100 µl purified immunoglobulin at 1 mg/ml in phosphate buffered saline. The solution is sterile filtered.

Storage/Stability

Store at –20 °C. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. 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

The recommended working dilutions are $1:10^3$ to $1:10^5$ for ELISA; 1:100 to 1:1000 for immunoblotting; 1:10 to 1:100 for immunoprecipitation, and 1:100 to 1:1000 for immunohistochemistry.

Immunohistochemistry has been performed on formalin-fixed human and animal brains or paraffin-embedded and Immunogold EM embedded Alzheimer or animal brain sections. The epitope must be re-exposed in fixed tissues by pretreatment of tissue with 70 % formic acid for 10-30 minutes at room temperature.

Note: In order to obtain best results and assay sensitivities of different techniques and preparations, we recommend determining optimal working dilutions by titration test.

References

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3. Kawahara, M. and Kuroda, Y., Brain Res. Bulletin, **53**, 389-397 (2000).
4. Zhu, Y.J., et al., FASEB, **14**, 1244-1254 (2000).
5. Bhatia, R., et al., FASEB, **14**, 1233-1243 (2000).
6. Curtain, C.C., et al., J. Biol. Chem., **276**, 20466-20473 (2001).

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