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ProductInformation

Monoclonal Anti-Human β Amyloid [1-17]
Clone 6E10
Purified Mouse Immunoglobulin

Product Number **A 1474**

Product Description

Monoclonal Anti-Human β Amyloid [1-17] (mouse IgG1 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 1-17 of the β amyloid peptide with Glu substituted at position 11, conjugated to KLH. The immunoglobulin is isolated from mouse ascites fluid by Protein G chromatography.

Monoclonal Anti-Human β Amyloid [1-17] recognizes amino acid residues 1-17 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 [1-17] 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 re-uptake 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 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 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 [1-17] is supplied as 100 µl purified immunoglobulin at 1 mg/ml in phosphate buffered saline with 0.03 % thimerosal as preservative.

Precautions and Disclaimer

Due to the thimerosal content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling

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 reexposed in fixed tissues by pretreatment of tissue with 70% formic acid for 10-30 minutes at room temperature.

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

References

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4. Zhu, Y.J., et al., FASEB, **14**, 1244-1254 (2000).
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6. Curtain, C.C., et al., J. Biol. Chem., **276**, 20466-20473 (2001).

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