Product Information

Anti-β-Amyloid Antibody, Mouse Monoclonal

Clone BAM-10, purified from hybridoma cell culture

A3981

Product Description

Anti-β-Amyloid (mouse IgG1 isotype) is derived from the BAM-10 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from a BALB/c mouse immunized with a synthetic β-amyloid peptide (1-40) (Gene ID: 351) conjugated to KLH. The isotype is determined using a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Cat. No. ISO2.

Anti-β-Amyloid reacts specifically with β-amyloid protein. The epitope recognized by the antibody resides within amino acid residues [1-12] of the β-amyloid protein. The antibody specifically stains amyloid plaques within the cortex, and amyloid deposits in blood vessels, in formic acid-treated, formalin-fixed, paraffin-embedded and Methacarn-fixed sections of human Alzheimer's disease (AD) brain tissue. The antibody is useful in immunohistochemistry, immunoblotting,[3] ELISA, and competitive ELISA. It has been used to neutralize Aβ assemblies in brains of transgenic mice expressing a mutant form of amyloid precursor protein,[1] and for *in vivo* deep tissue imaging using near-IR optical spectrum.[2]

The β-amyloid precursor protein (APP) is cleaved sequentially by the proteolytic enzymes β-secretase (BACE1) 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. Extracellular accumulation of Aβ leads to the formation of aggregates, fibrils and eventually amyloid deposits called neuritic plaques, a hallmark of Alzheimer's disease (AD).[4] 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.[5-7] Cation channels are induced by both nascent and globular Aβ peptides.[8] Thus, Aβ peptides may elicit toxic effects prior to fibril formation. Evidence suggests that copper and zinc may modulate the structure of the pleimorphic Aβ peptides to induce either pore formation or peptide precipitation.[9] In other models, it was found that the Aβ peptides exhibit superoxide dismutase activity thus producing hydrogen peroxidase that may be responsible for neurotoxicity.[10]

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody concentration: ~1.5 mg/mL

Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For extended storage, freeze at -20 °C 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

Immunohistochemistry

A working concentration of 2.5-5 µg/mL is recommended using formic acid treated, formalin-fixed and paraffin-embedded tissue sections of human Alzheimer disease (AD) brain tissue.

Note: In order to obtain the best results using various techniques and preparations, we recommend deter-mining optimal working dilutions by titration.

References

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