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ProductInformation

MONOCLONAL ANTI-b-AMYLOID PROTEIN, CLONE BAM-10
Mouse Ascites Fluid

Product No. **A5213**

Product Description

Monoclonal Anti-β-Amyloid Protein (mouse IgG1 isotype) is derived from the BAM-10 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from an immunized BALB/c mouse. A synthetic β-amyloid peptide (1-40) conjugated to KLH was used as the immunogen. The isotype is determined using Sigma ImmunoType Kit (Product Code ISO-1) and by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents (Product Code ISO-2).

Monoclonal Anti-β-Amyloid Protein 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.

Monoclonal Anti-β Amyloid Protein may be used for the localization of β-amyloid protein using various immunochemical assays such as ELISA, competitive ELISA and immunohistochemistry.

Alzheimer's disease (AD) is the most common progressive age-related cause of senile dementia in man, characterized by abnormal filamentous protein deposits in the brain.[1] The abnormal filaments appear within neurons as neurofibrillary tangles and as extracellular deposits. The extracellular filaments are seen in the characteristic senile plaques within the cortex, and in blood vessel walls of the meninges, and are having the staining properties of amyloid. These extracellular amyloid deposits are composed of a 4.2 kDa protein (42-43 amino acids), first described as β-protein and subsequently referred to as A4 (βA4, Aβ4). Molecular biological studies have established that β-amyloid protein/amyloid A4 is derived from larger precursors which are members of a large family of a 70 kDa transmembrane glycoproteins (amyloid precursor proteins, APP). These are produced as a variety of isoforms by alternative splicing.[2] The precursors are synthesized by many tissues as well as in the brain.[3] APP undergo post-translational processing includeing N- and O- linked glycosylation, tyrosine phosphorylation and sulfatation, through a secretory pathway.[4]

β-Amyloid deposits are also detected in Lewy body dementia, Down's syndrome, amyloidosis (Dutch type) and in the Guam-Parkinson dementia complex. Several lines of evidence indicate that β-amyloid fragments are amyloidogenic and neurotoxic both *in vitro* and *in vivo*.[5,6] The presence of a large number of neuritic (senile) plaques and neurofibrillary tangles in the cerebral cortex is used as a pathological marker for a disease state and presents the major criterion for the diagnosis of Alzheimer's disease at autopsy. A monoclonal antibody reacting specifically with β-amyloid protein is valuable for studying the nature of the β-amyloid protein by enabling detection and localization of β-amyloid protein and fragments.

Reagents

Monoclonal Anti-β-Amyloid Protein is provided as ascites fluid with 15 mM sodium azide as a preservative.

Precautions

Due to the sodium azide content a material safety 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 practices.

Storage
For continuous use, store at 2-8 °C for no more than one month. 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.

Product Profile
A minimum working dilution of 1:2,000 is determined by indirect immunoperoxidase staining of formic acid-treated, formalin-fixed, paraffin-embedded tissue sections of human Alzheimer disease (AD) brain tissue.

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

References

1. Selkoe, D.J., Sc. Am., **265**, 40 (1991).
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5. Yanker, B.A., et al., Science, **245**, 417 (1989).
6. Neve, R.L., et al., Proc. Natl. Acad. Sci. USA, **89**, 3448 (1992).

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