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Product Information

Monoclonal Anti-β-Amyloid antibody produced in mouse
clone NAB 228, purified from hybridoma cell culture

Catalog Number **A8354**

Synonym: Anti-Aβ

Monoclonal Anti-β-Amyloid (mouse IgG2a isotype) is derived from the NAB 228 hybridoma produced by the fusion of mouse myeloma cells (SP2 cells) and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to amino acids 1-11 of human β-amyloid protein, conjugated to KLH.[1] The isotype is determined by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Catalog Number ISO2.

Monoclonal Anti-β-Amyloid recognizes human β-amyloid peptide, full-length amyloid precursor protein (APP) (approx 110kDa), soluble-APP (sAPPβ' and sAPPα) C99 cleavage form and Aβ (1-40/42), but not the soluble-APP form sAPPβ. The product is useful in ELISA, immunoblotting,[1] immunoprecipitation and immunohistochemistry.

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).[2] 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.[3-5] Cation channels are induced by both nascent and globular Aβ peptides.[6] 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.[7] In other models it was found that the Aβ peptides exhibit superoxidase dismutase activity thus producing hydrogen peroxidase that may be responsible for neurotoxicity.[8]

Reagent
The antibody is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody Concentration: ~2 mg/ml.

Precautions and Disclaimer
This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

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
Immunoblotting: a working concentration of 2-4 μg/ml is determined using cell extract of human embryonal carcinoma NTERA-2 (NT2/D1) cells, treated for 2-3 weeks with 10 μM retinoic acid.

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

References

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3. Pearson, H.A., in: Alzheimer's Disease: Methods and Protocols, pp. 113-138, Ed.: Hooper, N.M., Humana Press, NJ (2000).
4. Kawahara, M., and Kuroda, Y., *Brain Res. Bulletin*, **53**, 389-397 (2000).
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