ANTI-β-AMYLOID  (1-40)
Developed in Rabbit
Delipidized, Whole Antiserum

Product No. A 8326

Product Description
Anti-β- Amyloid (1-40) is developed in rabbit using a synthetic peptide (Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys-Leu-Val-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys-Gly-Ala-Ile-Gly-Leu-Met-Val-Gly-Gly-Val-Val) corresponding to β-Amyloid precursor protein (672-711) (β-APP_{672-711}) conjugated to BSA as the immunogen. The antiserum has been treated to remove lipoproteins.

Anti-β- Amyloid (1-40) reacts in dot blot immunoassay with β-Amyloid (1-40) peptide conjugated to KLH. Weak cross-reactivity is observed with β-Amyloid (1-28) and β-Amyloid (12-28) peptides conjugated to KLH. The antiserum shows no cross-reactivity with calcitonin gene related peptide (CGRP)-KLH conjugate. Anti-β- Amyloid (1-40) specifically stains amyloid plaques in formalin-fixed, paraffin-embedded or Methacarn-fixed brain sections (hippocampus) of Alzheimer's disease (AD) patients. Specific staining is inhibited by preincubation of diluted antiserum with β-Amyloid (1-40) peptide (200 µg/ml). No staining is observed with control sections of normal brain tissue.

The deposition of amyloid in senile plaques and on cerebral vasculature is a prominent feature in the neuropathology of Alzheimer's disease (AD) and Down's syndrome (DS). The principal constituent of amyloid deposits is β-amyloid peptide (βA4, Aβ4), a 4.2 kDa peptide fragment of 42-43 amino acids, that results from abnormal proteolytic cleavage of β-amyloid precursor proteins (APP). APP are members of a large family of 70 kDa transmembrane glycoproteins existing as three major isoforms (APP_{695}, APP_{751}, and APP_{770}) derived from alternative splicing of precursor mRNA. Both APP_{751} and APP_{770} contain a sequence of high homology to the Kunitz type serine proteinase inhibitors (KPI), whereas APP_{695} lacks this sequence. β-Amyloid fragments are amyloidogenic and non-neural tissue, while APP_{695} is preferentially expressed in the central nervous system (CNS). APP undergoes post-translational processing including N- and O- glycosylation, tyrosine phosphorylation and sulfonation, through a secretory pathway. In vivo, lesions in nerve cells are caused by a variety of mechanisms including excitotoxins (e.g., kainic acid, ibotenic acid), physical injury and ischemia and is accompanied by an increase in APP mRNA and immunoreactivity near the lesion. APP is rapidly induced particularly in astrocytes and hippocampal neurons following neuronal damage as well as in microglia/macrophages. The mechanisms responsible for APP processing are not completely understood. Normal processing of mature APP generates non-amyloidogenic, soluble amino terminal fragments following proteolytic cleavage between Lys and Leu peptide bond within the β-amyloid domain. Alternatively, APP is thought to be internalized and degraded by an endosomal-lysosomal pathway to yield carboxyl-terminal derivatives, including intact β-amyloid. Several lines of evidence indicate that these β-amyloid fragments are amyloidogenic and neurotoxic both in vitro and in vivo, and therefore may play a role in the neuropathology of AD. Antibodies that react specifically with β-amyloid may be used to study the intracellular and extracellular localization of β-amyloid fragments and APP processing in the neuropathology of AD.

Reagents
Rabbit Anti-β- Amyloid (1-40) is supplied as a liquid containing 0.1% sodium azide as preservative.

Precautions and Disclaimer
Due to the sodium azide 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 hazards and safe handling practices.
Storage
For continuous use, store at 2-8 °C for up to one month. For extended storage, the solution may be frozen 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.

References