

Product Information

Anti-Amyloid Precursor Protein, C-Terminal produced in rabbit, IgG fraction of antiserum

Catalog Number **A8717**

Synonym: Anti-APP

Product Description

Anti-Amyloid Precursor Protein, C-Terminal is produced in rabbit using as immunogen a synthetic peptide KMQQNGYENPTYKFFEQMQN corresponding to the C-terminal of human APP₆₉₅ (amino acids 676-695) conjugated to KLH. This sequence is identical in rat and mouse APP₆₉₅ and the isoforms APP₇₅₁ and APP₇₇₀. Whole antiserum is purified to provide an IgG fraction of antiserum

Anti-Amyloid Precursor Protein, C-Terminal recognizes human, mouse and rat APP₆₉₅, APP₇₅₁ and APP₇₇₀ (95-100 kDa) by immunoblotting. Staining of the APP bands (95-100 kDa) by immunoblotting is specifically inhibited with the APP immunizing peptide.

Anti-Amyloid Precursor Protein, C-Terminal may be used for the detection of neurite plaques in human Alzheimer's Disease (AD) brain by immunohistochemistry

Alzheimer's Disease is characterized by deposition of amyloid in the central nervous system, in neurite plaques and on cerebral vasculature. The principal constituent of amyloid deposits is the β -amyloid peptide (β A4, A β , A4) a 42-43 amino acids (4.2 kDa) fragment that is a cleavage product of the amyloid precursor proteins (APPs).^{1,2} APPs are members of a large family of transmembrane glycoproteins widely distributed in many tissues. APPs three major isoforms, APP₆₉₅, APP₇₅₁ and APP₇₇₀ (calculated MW 79-87 kDa), are derived from alternative splicing of common precursor mRNA. Both APP₇₅₁ and APP₇₇₀ contain a 56 amino acid domain that is highly homologous to the Kunitz family of serine protease inhibitors (KPI domain), whereas APP₆₉₅ lacks this insert.³ APP₆₉₅ is preferentially expressed in the central nervous system,

while APP₇₅₁ and APP₇₇₀ are more abundant in peripheral tissues.⁴ APPs undergo post-translational processing including N- and O-glycosylation, phosphorylation and sulfation. The function of APPs is unknown and the mechanisms underlying APP processing are not completely understood. Mutations in the APP gene are linked with rare forms of autosomal dominant familial Alzheimer's Disease (FAD). These mutations result in increased production of A β . In Alzheimer's Disease, APP is thought to be internalized and degraded by an endosomal-lysosomal pathway to yield amyloidogenic peptides.⁵ β -Amyloid is cytotoxic causing neuronal damage and degeneration *in vitro* and *in vivo*.^{6,7} β -Amyloid peptide induces cytotoxic oxidative stress, formation of reactive oxygen intermediates (ROI), promotes microglia activation, astrocytosis and microgliosis^{8,9,10} and it is therefore thought to play a central role in the neuropathology of AD.

Reagents

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

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.

Product Profile

Immunohistochemistry: a minimum working dilution of 1:1,000 is determined using formalin-fixed, paraffin-embedded, formic acid-treated sections of human Alzheimer's Disease (AD) brain.

Immunoblotting: a minimum working dilution of 1:4,000 is determined using a rat brain extract.

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

References

1. Goldgaber, D., et al., *Science*, **235**, 877 (1987).
2. Tanzi, R.E., et al., *Science*, **235**, 880 (1987).
3. Tanzi, R.E., et al., *Nature*, **331**, 528 (1988).
4. Tanaka, S., et al., *Biochem. Biophys. Res. Commun.*, **165**, 1406 (1989).
5. Golde, T.E., et al., *Science*, **255**, 728 (1992).
6. Yankner, B.A., et al., *Science*, **245**, 417 (1989).
7. Johnson-Wood, K., et al., *Proc. Natl. Acad. Sci. USA*, **94**, 1550 (1997).
8. Hensley, K., et al., *Proc. Natl. Acad. Sci. USA*, **91**, 3270 (1994).
9. Games, D., et al., *Nature*, **373**, 523 (1995).
10. Yan, S.D., et al., *Nature*, **382**, 685 (1996).

MG,KAA,PHC 02/09-1