

Code No. 18591

**Anti-Human
Amyloid β (N3pE) Rabbit IgG Affinity Purify**Volume : 50 μ g

Introduction : Alzheimer's disease (AD) is characterized by the presence of extracellular plaques and intracellular neurofibrillary tangles (NFTs) in the brain. The major protein component of these plaques is beta amyloid (A β) peptide, a 40 to 43 amino acid peptide cleaved from amyloid precursor protein by β -secretase and γ -secretase. Increased release of A β 42 or A β 43, both of which exhibit a greater tendency to aggregate than A β 40, occurs in individuals expressing certain genetic mutations, ApoE alleles or may involve other undiscovered factors. Many researchers theorize that it is this increased release of A β 42/A β 43 which leads to the abnormal deposition of A β and the associated neurotoxicity in the brains of affected individuals.

It is reported that a distinct A β peptide, A β (N3pE), is deposited in senile plaques in a dominant and differential manner as compared with the standard A β peptide.

Antigen : Synthetic peptide of the N terminal part of A β (N3pE): Amyloid β which the 3rd N-terminal residue, glutamate is converted to pyroglutamate.

Purification : Purified with antigen peptide

Form : Lyophilized product from 1 % BSA in PBS containing 0.05 % NaN₃

How to use : 1.0 mL deionized water will be added to the product (the conc. comes up 50 μ g /mL)

Stability : Lyophilized product, 5 years at 2 - 8 °C
: Solution, 2 years at -20 °C

Application : This antibody can be used for immunohistochemistry with formalin fixed paraffin embedded tissues after formic acid treatment*1 by several techniques such as Avidin Biotin Complex (ABC) Method. The optimal concentration is 1 - 2 μ g/mL, however, the concentration should be optimized by each laboratory.

*1 Rinsing by running water after formic acid treatment for 5 minutes following de-paraffin.

: This antibody can be used for western blotting in concentration of 1 - 5 μ g /mL.

Specificity : Human Amyloid β (N3pE) specific.
Not cross-react with Human Amyloid β (1-40), (1-42) and (1-43).

Reference : 1. Cynis H, Scheel E, Saido TC, Schilling S, Demuth HU. Amyloidogenic processing of amyloid precursor protein: evidence of a pivotal role of glutaminyl cyclase in generation of pyroglutamate-modified amyloid-beta. *Biochemistry*. 2008 Jul 15;47(28):7405-13.
2. Shirotani K, Tsubuki S, Lee HJ, Maruyama K, Saido TC. Generation of amyloid beta peptide with pyroglutamate at position 3 in primary cortical neurons. *Neurosci Lett*. 2002 Jul 12;327(1):25-8.
3. Harigaya Y, Saido TC, Eckman CB, Prada CM, Shoji M, Younkin SG. Amyloid beta protein starting pyroglutamate at position 3 is a major component of the amyloid deposits in the Alzheimer's disease brain. *Biochem Biophys Res Commun*. 2000 Sep 24;276(2):422-7.
4. Tekirian TL, Saido TC, Markesbery WR, Russell MJ, Wekstein DR, Patel E, Geddes JW. N-terminal heterogeneity of parenchymal and cerebrovascular Abeta deposits. *J Neuropathol Exp Neurol*. 1998 Jan;57(1):76-94.
5. Russo C, Saido TC, DeBusk LM, Tabaton M, Gambetti P, Teller JK. Heterogeneity of water-soluble amyloid beta-peptide in Alzheimer's disease and Down's syndrome brains. *FEBS Lett*. 1997 Jun 16;409(3):411-6.
6. Saido TC, Iwatsubo T, Mann DM, Shimada H, Ihara Y, Kawashima S. Dominant and differential deposition of distinct beta-amyloid peptide species, A beta N3(pE), in senile plaques. *Neuron*. 1995 Feb;14(2):457-66.

For Non-Clinical Research Use Only