

**$\beta$ -Amyloid (pE3 Peptide) Antibody**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

| Applications: | Reactivity: | Sensitivity: | MW (kDa): | Source/Isotype: | UniProt ID: | Entrez-Gene Id: |
|---------------|-------------|--------------|-----------|-----------------|-------------|-----------------|
| W             | H           | Recombinant  | 4         | Rabbit          | #P05067     | 351             |

**Product Usage Information****Application**

Western Blotting

**Dilution**

1:1000

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100  $\mu$ g/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

$\beta$ -Amyloid (pE3 Peptide) Antibody recognizes recombinant pE3 form of  $\beta$ -amyloid peptides. This antibody does not cross-react with the non-pyroglutamate (E3) form of  $\beta$ -amyloid peptides.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human  $\beta$ -amyloid (pE3) peptide. Antibodies are purified by protein A and peptide affinity chromatography.

**Background**

Amyloid  $\beta$  (A $\beta$ ) precursor protein (APP) is a 100-140 kDa transmembrane glycoprotein that exists as several isoforms (1). The amino acid sequence of APP contains the amyloid domain, which can be released by a two-step proteolytic cleavage (1). The extracellular deposition and accumulation of the released A $\beta$  fragments form the main components of amyloid plaques in Alzheimer's disease (1). APP can be phosphorylated at several sites, which may affect the proteolytic processing and secretion of this protein (2-5). Phosphorylation at Thr668 (a position corresponding to the APP695 isoform) by cyclin-dependent kinase is cell-cycle dependent and peaks during G2/M phase (4). APP phosphorylated at Thr668 exists in adult rat brain and correlates with cultured neuronal differentiation (5,6). A $\beta$  peptides can be further modified by amino-terminal truncation that exposes a free glutamate residue to the enzyme glutaminyl cyclase, which catalyzes the formation of an amino-terminal pyroglutamate (pE) (7,8). A $\beta$  (pE3) peptides exhibit increased stability relative to non-modified peptides due to an enhanced resistance to peptidase-mediated degradation (9) and a higher propensity to form  $\beta$ -sheets and aggregate (10). Antibodies targeting A $\beta$  (pE3) peptides may be plaque-specific as there is no evidence for circulating A $\beta$  (pE3) peptides (11).

**Background References**

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**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting

**Cross-Reactivity Key**

**H:** Human

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