

PRODUCT INFORMATION



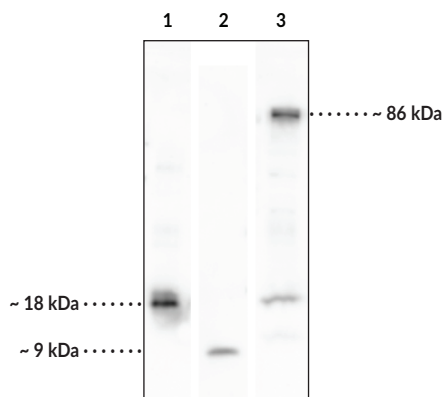
APP (C99 Fragment) (C-Term) Polyclonal Antibody

Item No. 28637

Overview and Properties

Contents:	This vial contains 500 µl of peptide affinity-purified polyclonal antibody.
Synonyms:	ABPP, Alzheimer's Disease Amyloid Protein, Amyloid-β A4 Protein, Amyloid Precursor Protein, APPI, Cerebral Vascular Amyloid Peptide, β C-terminal Fragment, βCTF, CVAP, PN-II, PreA4, Protease Nexin-II
Immunogen:	Synthetic peptide corresponding to the C-terminal region of the C99 fragment of human APP
Cross Reactivity:	(+) APP, C99 fragment, C47 fragment
Species Reactivity:	(+) Human
Uniprot No.:	P05067
Form:	Liquid
Storage:	-20°C (as supplied)
Stability:	≥3 years
Storage Buffer:	PBS, pH 7.2, with 50% glycerol and 0.02% sodium azide
Host:	Rabbit
Applications:	ELISA and Western blot (WB); the recommended starting dilution is 1:200 for ELISA and WB. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically.

Image



Lane 1: C99 (APP fragment) (10 ng)
Lane 2: C47 (APP fragment) (25 ng)
Lane 3: APP Overexpression Lysate (10 µg)

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

WARRANTY AND LIMITATION OF REMEDY
Buyer agrees to purchase the material subject to Cayman's Terms and Conditions. Complete Terms and Conditions including Warranty and Limitation of Liability information can be found on our website.

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Description

Amyloid precursor protein (APP) is a type I transmembrane protein that has a central role in the pathogenesis of Alzheimer's disease, as well as additional roles in brain development, neuronal plasticity, and memory.¹ APP is cleaved by β -secretase (BACE) in neuronal endosomes during amyloidogenic processing of APP, generating the C-terminal C99 fragment, which is localized to the endoplasmic membrane.^{2,3} C99 is further cleaved by γ -secretase, liberating the APP intracellular domain (AICD) and generating amyloid- β (A β) peptides of various lengths, including A β 40 (Item No. 21617) and A β 42 (Item No. 20574), which are hallmarks of Alzheimer's disease. APP can also be cleaved by α -secretase during non-amyloidogenic processing of APP, which occurs at the neuronal plasma membrane and generates the neuroprotective soluble APP fragment sAPP α , as well as a variety of other fragments, including the C47 fragment.^{1,4} Transgenic mice expressing mutant forms of APP exhibit extracellular A β deposits in the brain, as well as cognitive dysfunction, and are widely used models of Alzheimer's disease.⁵ Intraneuronal C99 levels are increased in the transgenic APP^{E693Q} mouse model of Alzheimer's disease, as well as postmortem frontal cortex from patients with sporadic Alzheimer's disease.^{3,6} Cayman's APP (C99 Fragment) (C-Term) Polyclonal Antibody can be used for ELISA and Western blot applications. The antibody recognizes the C-terminal region corresponding to the C99 fragment to detect intact APP, as well as the C47 APP fragment, at 18 kDa and 9 kDa, respectively, from human samples.

References

1. Müller, U.C., Deller, T., and Korte, M. Not just amyloid: Physiological functions of the amyloid precursor protein family. *Nat. Rev. Neurosci.* **18**(5), 281-298 (2017).
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3. Lauritzen, I., Pardossi-Piquard, R., Bourgeois, A., *et al.* Does intraneuronal accumulation of carboxyl-terminal fragments of the amyloid precursor protein trigger early neurotoxicity in Alzheimer's disease? *Curr. Alzheimer Res.* **16**(5), 453-457 (2019).
4. Schrader-Fischer, G., Staufienbiel, M., and Paganetti, P.A. Insertion of lysosomal targeting sequences to the amyloid precursor protein reduces secretion of β A4. *J. Neurochem.* **68**(4), 1571-1580 (1997).
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6. Pulina, M.V., Hopkins, M., Haroutunian, V., *et al.* C99 selectively accumulates in vulnerable neurons in Alzheimer's disease. *Alzheimers Dement.* **16**(2), 273-282 (2020).

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