PRODUCT INFORMATION

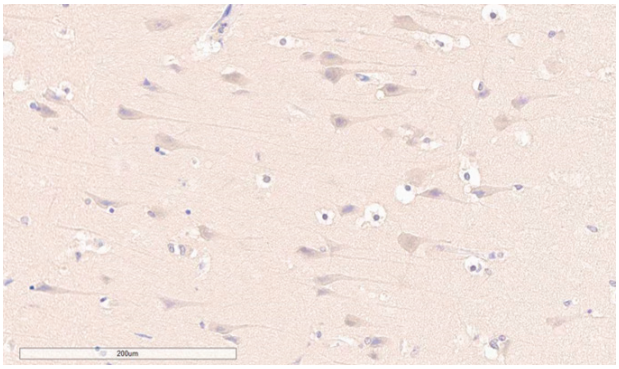
APP (C99 Fragment) Monoclonal Antibody (Clone 8G4)

Item No. 28636

Overview and Properties

Contents:	This vial contains 100 µg of protein G-purified monoclonal 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:	Peptide corresponding to the C99 fragment of human APP
Cross Reactivity:	(+) APP, C99 fragment, C47 fragment
Species Reactivity:	(+) Human; other species not tested
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
Clone:	8G4
Host:	Mouse
Isotype:	IgG2a
Applications:	ELISA and Immunohistochemistry (IHC); the recommended starting dilution is 1:1,000 for ELISA and 1:200 for IHC. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically.

Image



Immunohistochemistry analysis of formalin-fixed, paraffin-embedded (FFPE) human Alzheimer's disease brain tissue after heat-induced antigen retrieval in pH 6.0 citrate buffer. After incubation with APP (C99 Fragment) Monoclonal Antibody (Clone 8G4) at a dilution of 1:200, slides were incubated with a biotinylated secondary antibody, followed by alkaline phosphatase-streptavidin and chromogen (DAB).

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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 (Aβ) peptides of various lengths, including Aβ40 (Item No. 21617) and Aβ42 (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 sAPPα, as well as a variety of other fragments, including the C47 fragment.[1,4] Transgenic mice expressing mutant forms of *APP* exhibit extracellular Aβ 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) Monoclonal Antibody (Clone 8G4) can be used for ELISA and immunohistochemistry (IHC) applications. The antibody recognizes the C-terminal region corresponding to the C99 fragment to detect intact APP, as well as the C47 APP fragment.

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).
2. Zhao, J., Liu, X., Xia, W., *et al.* Targeting amyloidogenic processing of APP in Alzheimer's disease. *Front. Mol. Neurosci.* **13**, 137 (2020).
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., Staufenbiel, M., and Paganetti, P.A. Insertion of lysosomal targeting sequences to the amyloid precursor protein reduces secretion of βA4. *J. Neurochem.* **68(4)**, 1571-1580 (1997).
5. Sasaguri, H., Nilsson, P., Hashimoto, S., *et al.* APP mouse models for Alzheimer's disease preclinical studies. *EMBO J.* **36(17)**, 2473-2487 (2017).
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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