

Code No. 18961

**Anti-Human
APP (C) Rabbit IgG Affinity Purify**

Volume : 100 µg

Introduction : Amyloid precursor protein (APP) is precursor protein of Amyloid β which is major constituent of senile plaque in Alzheimer's disease. It is known that there are three major isoforms, APP695, APP751 and APP770, and are generated from alternative splicing of common precursor mRNA. Processing of APP occurs by two major pathways, non-amyloidogenic pathway and amyloidogenic pathway. The non-amyloidogenic pathway is mediated by α and γ-secretases and gives rise to a large fragment known as soluble APPα (sAPPα) and a small 3 kDa peptide known as p3. On the other hand, the Amyloidogenic pathway is mediated by β- and γ-secretases and yields soluble APPβ (sAPPβ) and Amyloid β. The physiologic function of APP itself is not clear, however, it is supposed that the function of APP in neuron system is different from that in other organ.

Antigen : Synthetic peptide of the C terminal part of Human APP
(NGYENPTYKFFEQMQN)

Purification : Purified with antigen peptide

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

How to use : 1.0 mL deionized water will be added to the product (the conc. comes up 100 µg /mL)

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

Application : This antibody can be used for western blotting in concentration of 1 - 2 µg /mL.
: This antibody can be used for immuno-precipitation in concentration of about 3 µg /test.

Specificity : Cross-reacts with mouse and rat. Not cross-react to sAPPα and sAPPβ
All isoforms (APP695, APP751, APP770) are detectable.

Reference : 1. Citron M, Oltersdorf T, Haass C, McConlogue L, Hung AY, Seubert P, Vigo-Pelfrey C, Lieberburg I, Selkoe DJ. Mutation of the beta-amyloid precursor protein in amilial Alzheimer's disease increases beta-protein production. *Nature* 360 (6405): 672-674 (1992)
2. Goate A, Chartier-Harlin MC, Mullan M, et al. Segregation of a missense mutation in the amyloid precursor protein gene with familial Alzheimer's disease. *Nature* 349: 704-706 (1991)
3. Selkoe DJ. Normal and abnormal biology of the beta-amyloid precursor protein. *Annu Rev Neurosci.* 1994;17:489-517.
4. Hsiao K, Chapman P, Nilsen S, Eckman C, Harigaya Y, Younkin S, Yang F, Cole G. Correlative memory deficits, Abeta elevation, and amyloid plaques in transgenic mice. *Science* 274 (5284): 99-102 (1996)
5. Iijima K, Ando K, Takeda S, Satoh Y, Seki T, Itohara S, Greengard P, Kirino Y, Nairn AC, Suzuki T. Neuron-specific phosphorylation of Alzheimer's beta-amyloid precursor protein by cyclin-dependent kinase 5. *J Neurochem.* 2000 Sep;75(3):1085-91.
6. Steinhilb mL, Turner RS, Gaut JR. ELISA analysis of beta-secretase cleavage of the Swedish amyloid precursor protein in the secretory and endocytic pathways. *J Neurochem* 80 (6): 1019-28 (2002)
7. Taru H, Suzuki T. Facilitation of stress-induced phosphorylation of beta-amyloid precursor protein family members by X11-like/Mint2 protein. *J Biol Chem.* 2004 May 14; 279(20): 21628-36.
8. Ando K, Oishi M, Takeda S, Iijima K, Isohara T, Nairn AC, Kirino Y, Greengard P, Suzuki T. Role of phosphorylation of Alzheimer's amyloid precursor protein during neuronal differentiation. *J Neurosci.* 1999 Jun 1; 19(11): 4421-7.
9. Lee MS, Kao SC, Lemere CA, Xia W, Tseng HC, Zhou Y, Neve R, Ahljiarian MK, Tsai LH. APP processing is regulated by cytoplasmic phosphorylation. *J Cell Biol.* 2003 Oct 13; 163(1): 83-95.
10. Nakaya T, Suzuki T. Role of APP phosphorylation in FE65-dependent gene transactivation mediated by AICD. *Genes Cells.* 2006 Jun; 11(6): 633-45.

For research use only, not for use in diagnostic procedures.