Synaptic Systems

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APP

Cat.No. 127 003; Polyclonal rabbit antibody, 50 µg specific antibody (lyophilized)

Data Sheet

Reconstitution/ Storage	50 µg specific antibody, lyophilized. Affinity purified with the immunogen. Rabbit serum albumin was added for stabilization. For reconstitution add 50 µl H_2O to get a 1mg/ml solution in PBS. Then aliquot and store at -20°C until use.
Applications	**WB**: 1 : 500 up to 1 : 1000 (AP staining) **IP**: not tested yet **ICC**: 1 : 500 **IHC**: 1 : 500 up to 1 : 1000 (see remarks) **IHC-P/FFPE**: 1 : 2000
Immunogen	Synthetic peptide corresponding to AA 756 to 770 from rat APP (UniProt Id: P08592)
Reactivity	Reacts with: rat (P08592), mouse (P12023), chicken, frog. Other species not tested yet.
Specificity	Specific for APP.
matching control	127-0P
Remarks	**IHC**: **IHC/IHC-P**: The affinity purified antibody is highly recommended.

TO BE USED IN VITRO / FOR RESEARCH ONLY
NOT TOXIC, NOT HAZARDOUS, NOT INFECTIOUS, NOT CONTAGIOUS

Alzheimer's disease is characterized by the accumulation of β-amyloid peptides in plaques and vessel walls and by the intraneuronal accumulation of paired helical filaments composed of hyperphosphorylated tau.
Amyloid **p**recursor **p**rotein **APP** is part of a super-family of transmembrane and secreted proteins. It appears to have a number of roles, including regulation of haemostasis and mediation of neuroprotection. APP also has metal and heparin-binding properties. Cleavage of amyloid precursor protein by β- and γ-secretases results in the generation of the Aβ (βA4)peptide, whereas α-secretase cleaves within the Aβ sequence and prevents formation from APP.
Recent findings indicate that the site of γ-secretase cleavage is critical to the development of amyloid deposits. Aβ1-42 is much more amyloidogenic than Aβ1-40. Aβ1-42 formation is favoured by mutations in the two presenilin genes (PS1 and PS2), and by the commonest amyloid precursor protein mutations.

Selected References SYSY Antibodies

Amyloid precursor protein is trafficked and secreted via synaptic vesicles.
Groemer TW, Thiel CS, Holt M, Riedel D, Hua Y, Hüve J, Wilhelm BG, Klingauf J
PloS one (2011) 6(4): e18754. **WB, ICC, EM**

The metalloprotease ADAMTS4 generates N-truncated Aβ4-x species and marks oligodendrocytes as a source of amyloidogenic peptides in Alzheimer's disease.
Walter S, Jumpertz T, Hüttenrauch M, Ogorek I, Gerber H, Storck SE, Zampar S, Dimitrov M, Lehmann S, Lepka K, Berndt C, et al.
Acta neuropathologica (2018) : . **IHC-P; tested species: mouse**

Transformation of diffuse beta-amyloid precursor protein and beta-amyloid deposits to plaques in the thalamus after transient occlusion of the middle cerebral artery in rats.
van Groen T, Puurunen K, Mäki HM, Sivenius J, Jolkkonen J
Stroke (2005) 36(7): 1551-6. **IHC; tested species: rat**

Surface Trafficking of APP and BACE in Live Cells.
Bauereiss A, Welzel O, Jung J, Grosse-Holz S, Lelental N, Lewczuk P, Wenzel EM, Kornhuber J, Groemer TW
Traffic (Copenhagen, Denmark) (2015) 16(6): 655-75. **ICC**

Selected General References

The amyloid precursor protein of Alzheimer's disease and the Abeta peptide.
Storey E, Cappai R
Neuropathology and applied neurobiology (1999) 25(2): 81-97.

Molecular genetics of Alzheimer's disease.
Cruts M, Van Broeckhoven C
Annals of medicine (1998) 30(6): 560-5.

Regulation of APP expression, biogenesis and metabolism by extracellular matrix and cytokines.
Beyreuther K, Multhaup G, Mönning U, Sandbrink R, Beher D, Hesse L, Small DH, Masters CL
Annals of the New York Academy of Sciences (1996) 777: 74-6.

The role of APP processing and trafficking pathways in the formation of amyloid beta-protein.
Selkoe DJ, Yamazaki T, Citron M, Podlisny MB, Koo EH, Teplow DB, Haass C
Annals of the New York Academy of Sciences (1996) 777: 57-64.