

# Product datasheet

info@arigobio.com

# ARG54671 anti-Amyloid Precursor Protein antibody

Package: 50 μg Store at: -20°C

### Summary

Product Description Rabbit Polyclonal antibody recognizes Amyloid Precursor Protein

Tested Reactivity Hu, Ms, Rat

Tested Application ELISA, IHC-P, WB

Host Rabbit

**Clonality** Polyclonal

Isotype IgG

Target Name Amyloid Precursor Protein

Immunogen Synthetic peptide (10 aa) within aa. 653-662 of Human APP protein (DAEFRHDSGY).

Conjugation Un-conjugated

Alternate Names CVAP; AAA; AICD-50; PN2; 50; Beta-APP42; AID; Gamma-CTF; S-APP-alpha; 57; AD1; PN-II; Beta-APP40;

42; 40; APPI; Alzheimer disease amyloid protein; Amyloid beta A4 protein; PreA4; ABETA; Amyloid intracellular domain 50; CTFgamma; Amyloid intracellular domain 57; 59; AICD-59; S-APP-beta; APP; AICD-57; Amyloid intracellular domain 59; ABPP; Protease nexin-II; Cerebral vascular amyloid peptide

#### **Application Instructions**

Application table	Application	Dilution
	ELISA	Assay-Dependent
	IHC-P	Assay-Dependent
	WB	1 μg/mL
Application Note	* The dilutions indicate recommended starting dilutions and the optimal dilutions or concentrations should be determined by the scientist.	
Positive Control	Mouse Brain Tissue Lysate	
Observed Size	115 kDa	

#### **Properties**

Form Liquid

Purification Affinity purification with immunogen.

Buffer PBS and 0.02% Sodium azide

Preservative 0.02% Sodium azide

Concentration 1 mg/ml

Storage instruction For continuous use, store undiluted antibody at 2-8°C for up to a week. For long-term storage, aliquot

and store at -20°C or below. Storage in frost free freezers is not recommended. Avoid repeated freeze/thaw cycles. Suggest spin the vial prior to opening. The antibody solution should be gently mixed

before use.

Note

For laboratory research only, not for drug, diagnostic or other use.

#### Bioinformation

Gene Symbol Gene Full Name Background APP

amyloid beta (A4) precursor protein

APP Antibody: Accumulation of the amyloid-beta peptide (Abeta) in the cerebral cortex is a critical event in the pathogenesis of Alzheimer's disease. The beta-amyloid protein precursor (APP) is cleaved by beta-secretase, producing a soluble derivative of the protein and a membrane anchored 99-amino acid carboxy-terminal fragment (C99). The C99 fragment serves as substrate for gamma-secretase to generate the 4 kDa amyloid-beta peptide (Abeta), which is deposited in the brains of all suffers of Alzheimer's disease.

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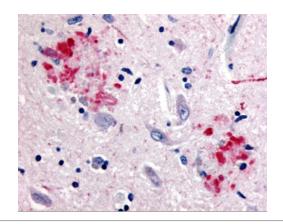
87 kDa. (79 - 120 kDa depending on glycosylation level)

Proteolytically processed under normal cellular conditions. Cleavage either by alpha-secretase, beta-secretase or theta-secretase leads to generation and extracellular release of soluble APP peptides, S-APP-alpha and S-APP-beta, and the retention of corresponding membrane-anchored C-terminal fragments, C80, C83 and C99. Subsequent processing of C80 and C83 by gamma-secretase yields P3 peptides. This is the major secretory pathway and is non-amyloidogenic. Alternatively, presenilin/nicastrin-mediated gamma-secretase processing of C99 releases the amyloid beta proteins, amyloid-beta 40 (Abeta40) and amyloid-beta 42 (Abeta42), major components of amyloid plaques, and the cytotoxic C-terminal fragments, gamma-CTF(50), gamma-CTF(57) and gamma-CTF(59). Many other minor beta-amyloid peptides, beta-amyloid 1-X peptides, are found in cerebral spinal fluid (CSF) including the beta-amyloid X-15 peptides, produced from the cleavage by alpha-secretase and all terminating at Gln-686. Proteolytically cleaved by caspases during neuronal apoptosis. Cleavage at Asp-739 by either caspase-6, -8 or -9 results in the production of the neurotoxic C31 peptide and the increased production of beta-amyloid peptides.

N- and O-glycosylated. O-glycosylation on Ser and Thr residues with core 1 or possibly core 8 glycans. Partial tyrosine glycosylation (Tyr-681) is found on some minor, short beta-amyloid peptides (beta-amyloid 1-15, 1-16, 1-17, 1-18, 1-19 and 1-20) but not found on beta-amyloid 38, beta-amyloid 40 nor on beta-amyloid 42. Modification on a tyrosine is unusual and is more prevelant in AD patients. Glycans had Neu5AcHex(Neu5Ac)HexNAc-O-Tyr, Neu5AcNeu5AcHex(Neu5Ac)HexNAc-O-Tyr and O-AcNeu5AcNeu5AcHex(Neu5Ac)HexNAc-O-Tyr structures, where O-Ac is O-acetylation of Neu5Ac. Neu5AcNeu5Ac is most likely Neu5Ac 2,8Neu5Ac linked. O-glycosylations in the vicinity of the cleavage sites may influence the proteolytic processing. Appicans are L-APP isoforms with O-linked chondroitin sulfate.

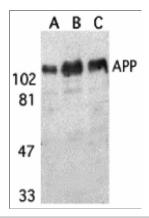
Phosphorylation in the C-terminal on tyrosine, threonine and serine residues is neuron-specific. Phosphorylation can affect APP processing, neuronal differentiation and interaction with other proteins. Phosphorylated on Thr-743 in neuronal cells by Cdc5 kinase and Mapk10, in dividing cells by Cdc2 kinase in a cell-cycle dependent manner with maximal levels at the G2/M phase and, in vitro, by GSK-3-beta. The Thr-743 phosphorylated form causes a conformational change which reduces binding of Fe65 family members. Phosphorylation on Tyr-757 is required for SHC binding. Phosphorylated in the extracellular domain by casein kinases on both soluble and membrane-bound APP. This phosphorylation is inhibited by heparin.

Extracellular binding and reduction of copper, results in a corresponding oxidation of Cys-144 and Cys-158, and the formation of a disulfide bond. In vitro, the APP-Cu(+) complex in the presence of hydrogen peroxide results in an increased production of beta-amyloid-containing peptides. Trophic-factor deprivation triggers the cleavage of surface APP by beta-secretase to release sAPP-beta which is further cleaved to release an N-terminal fragment of APP (N-APP). Beta-amyloid peptides are degraded by IDE.



# ARG54671 anti-APP antibody IHC image

Immunohistochemistry: human brain (Alzheimer's disease) tissue stained with ARG54671 anti-APP antibody at 10  $\mu g/ml.$ 



# ARG54671 anti-APP antibody WB image

Western blot: (A) human, (B) mouse, and (C) rat brain tissue lysates stained with ARG54671 anti-APP antibody at 1  $\mu g/ml$ .