ARG22203
anti-Amyloid Oligomers (A11) antibody

Package: 50 µl
Store at: -20°C

Summary

Product Description	Rabbit Polyclonal antibody recognizes Amyloid Oligomers (A11)
Tested Reactivity	Hu, Ms, Rat
Tested Application	Dot, ELISA, ICC/IF, IHC-P, IP, WB
Specificity	Recognizes all types of amyloid oligomers. Appears to recognize a peptide backbone epitope that is common to amyloid oligomers, but is not found in native proteins, amyloidogenic monomer.
Host	Rabbit
Clonality	Polyclonal
Target Name	Amyloid Oligomers (A11)
Species	Human
Immunogen	Synthetic molecular mimic of soluble oligomers
Conjugation	Un-conjugated
Alternate Names	AAA; AD1; PN2; ABPP; APPI; CVAP; ABETA; PN-II; CTFgamma

Application Instructions

Application table

Application	Dilution
Dot	Assay-dependent
ELISA	Assay-dependent
ICC/IF	Assay-dependent
IHC-P	Assay-dependent
IP	1:1000
WB	1:1000

Application Note: * The dilutions indicate recommended starting dilutions and the optimal dilutions or concentrations should be determined by the scientist.

Properties

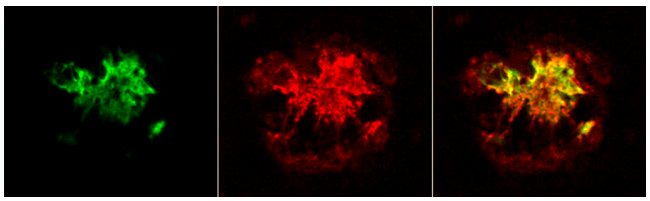
Form	Liquid
Purification	Purification with Protein A.
Buffer	PBS, 0.09% Sodium azide and 50% Glycerol
Preservative	0.09% Sodium azide
Stabilizer	50% Glycerol
Storage instruction	For continuous use, store undiluted antibody at 2-8°C for up to a week. For long-term storage, aliquot and store at -20°C. 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	APP
Gene Full Name	amyloid beta precursor protein
Calculated Mw	87 kDa. (79 - 120 kDa depending on glycosylation level)
PTM	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.
Cellular Localization	Membrane

Images



ARG22203 anti-Amyloid Oligomers (A11) antibody IHC image

Immunohistochemistry: Formalin-fixed Human Alzheimer's Disease brain stained with ARG22203 anti-Amyloid Oligomers (A11) antibody at 1:1000. Secondary Antibody: Goat anti-Rabbit ATTO 594 (red). (A) ARG22204 anti-Amyloid Fibrils (OC) antibody. (B) ARG22203 anti-Amyloid Oligomers (A11) antibody. (C) Composite.