
Product Name: Amyloid- β Rabbit Polyclonal Antibody**Catalog #: APRab06870**

For research use only.

Summary

Description	Rabbit polyclonal Antibody
Host	Rabbit
Application	WB,IHC,ICC/IF,ELISA
Reactivity	Human,Mouse,Rat
Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Concentration	1mg/ml
Storage	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Shipping	Ice bags
Buffer	Liquid in PBS containing 50% glycerol, 0.5% protective protein and 0.02% New type preservative N.
Purification	Affinity purification

Application

Dilution Ratio	WB 1:500-1:2000,IHC 1:50-1:300,ICC/IF 1:50-1:200,ELISA 1:10000-1:20000
Molecular Weight	87kDa

Antigen Information

Gene Name	APP A4 AD1
Alternative Names	amyloid beta (A4) precursor protein
Gene ID	351.0
SwissProt ID	P05067
Immunogen	Synthesized peptide derived from Amyloid- β at AA range: 221-270

Background

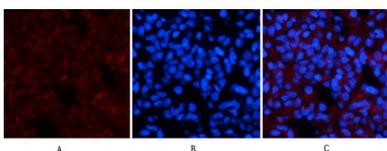
This gene encodes a cell surface receptor and transmembrane precursor protein that is cleaved by secretases to form a number of peptides. Some of these peptides are secreted and can bind to the acetyltransferase complex APBB1/TIP60 to promote

transcriptional activation, while others form the protein basis of the amyloid plaques found in the brains of patients with Alzheimer disease. In addition, two of the peptides are antimicrobial peptides, having been shown to have bacteriocidal and antifungal activities. Mutations in this gene have been implicated in autosomal dominant Alzheimer disease and cerebroarterial amyloidosis (cerebral amyloid angiopathy). Multiple transcript variants encoding several different isoforms have been found for this gene. [provided by RefSeq, Aug 2014],alternative products:Additional isoforms seem to exist. Experimental confirmation may be lacking for some isoforms,disease:Defects in APP are the cause of Alzheimer disease type 1 (AD1) [MIM:104300]. AD1 is a familial early-onset form of Alzheimer disease. It can be associated with cerebral amyloid angiopathy. Alzheimer disease is a neurodegenerative disorder characterized by progressive dementia, loss of cognitive abilities, and deposition of fibrillar amyloid proteins as intraneuronal neurofibrillary tangles, extracellular amyloid plaques and vascular amyloid deposits. The major constituent of these plaques is the neurotoxic amyloid-beta-APP 40-42 peptide (s), derived proteolytically from the transmembrane precursor protein APP by sequential secretase processing. The cytotoxic C-terminal fragments (CTFs) and the caspase-cleaved products such as C31 derived from APP, are also implicated in neuronal death.,disease:Defects in APP are the cause of amyloidosis cerebroarterial Dutch type (AMYL CAD) [MIM:605714]; also known as hereditary cerebral hemorrhage with amyloidosis Dutch type (HCHWAD). AMYL CAD is a hereditary localized amyloidosis due to amyloid-beta A4 peptide(s) deposition in the cerebral vessels. Beta-APP40 is the predominant form of cerebrovascular amyloid. Amyloid is not found outside the nervous system. The principal clinical characteristics are recurrent cerebral and cerebellar hemorrhages, recurrent strokes, cerebral ischemia, cerebral infarction, and progressive mental deterioration. Onset of the disease is in middle age (44 to 60 years). Patients develop cerebral hemorrhage because of the severe cerebral amyloid angiopathy. Parenchymal amyloid deposits are rare and largely in the form of pre-amyloid lesions or diffuse plaque-like structures. They are Congo red negative and lack the dense amyloid cores commonly present in Alzheimer disease.,disease:Defects in APP are the cause of amyloidosis cerebroarterial Iowa type (AMYLCAIW) [MIM:605714]. AMYLCAIW is a hereditary amyloidosis due to amyloid-beta A4 peptide(s) deposition. Patients have progressive aphasic dementia, leukoencephalopathy, and occipital calcifications.,disease:Defects in APP are the cause of amyloidosis cerebroarterial Italian type (AMYLCAIT) [MIM:605714]. AMYLCAIT is a hereditary localized amyloidosis due to amyloid-beta A4 peptide(s) deposition in the cerebral vessels, resulting in cerebral amyloid angiopathy. Amyloid is not found outside the nervous system. It is a condition very similar to AMYL CAD, but the clinical course is less severe. Patients manifest mild cognitive decline, recurrent strokes, and epilepsy in some cases.

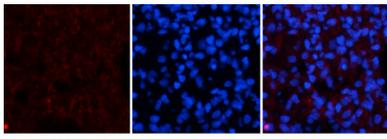
Research Area

Alzheimer's disease;

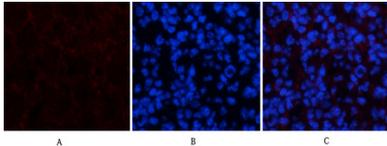
Image Data



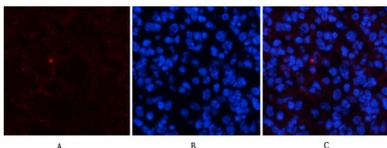
Immunofluorescence analysis of rat-lung tissue. 1, Amyloid- β Polyclonal Antibody (red) was diluted at 1:200 (4°C, overnight). 2, Cy3 labeled Secondary antibody was diluted at 1:300 (room temperature, 50min). 3, Picture B: DAPI (blue) 10min. Picture A: Target. Picture B: DAPI. Picture C: merge of A+B



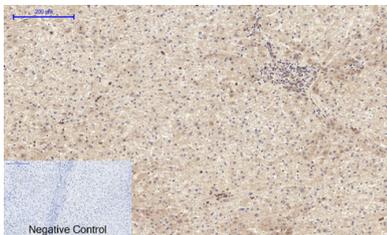
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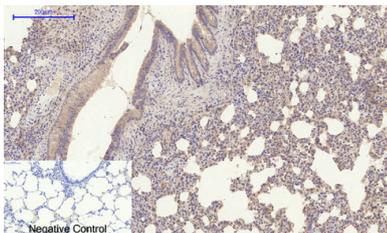
Immunofluorescence analysis of mouse-spleen tissue. 1, Amyloid- β Polyclonal Antibody (red) was diluted at 1:200 (4°C, overnight). 2, Cy3 labeled Secondary antibody was diluted at 1:300 (room temperature, 50min). 3, Picture B: DAPI (blue) 10min. Picture A: Target. Picture B: DAPI. Picture C: merge of A+B



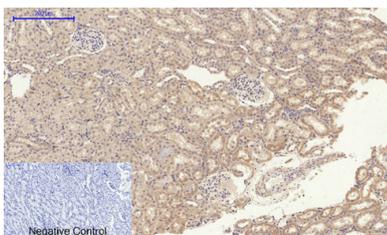
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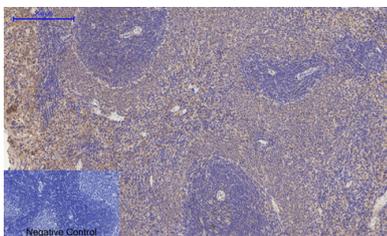
Immunohistochemical analysis of paraffin-embedded Human-liver tissue. 1, Amyloid- β Polyclonal Antibody was diluted at 1:200 (4°C, overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval (>98°C, 20min). 3, Secondary antibody was diluted at 1:200 (room temperature, 30min). Negative control was used by secondary antibody only.



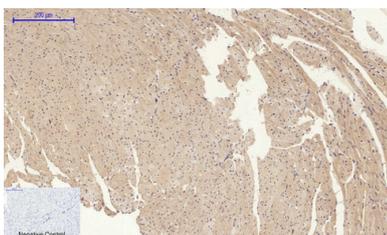
Immunohistochemical analysis of paraffin-embedded Rat-lung tissue. 1, Amyloid- β Polyclonal Antibody was diluted at 1:200 (4°C, overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval (>98°C, 20min). 3, Secondary antibody was diluted at 1:200 (room temperature, 30min). Negative control was used by secondary antibody only.



Immunohistochemical analysis of paraffin-embedded Rat-kidney tissue. 1, Amyloid- β Polyclonal Antibody was diluted at 1:200 (4°C, overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval (>98°C, 20min). 3, Secondary antibody was diluted at 1:200 (room temperature, 30min). Negative control was used by secondary antibody only.



Immunohistochemical analysis of paraffin-embedded Rat-spleen tissue. 1, Amyloid- β Polyclonal Antibody was diluted at 1:200 (4°C, overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval (>98°C, 20min). 3, Secondary antibody was diluted at 1:200 (room temperature, 30min). Negative control was used by secondary antibody only.



Immunohistochemical analysis of paraffin-embedded Mouse-heart tissue. 1, Amyloid- β Polyclonal Antibody was diluted at 1:200 (4°C, overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval (>98°C, 20min). 3, Secondary antibody was diluted at 1:200 (room temperature, 30min). Negative control was used by secondary antibody only.

