



Rabbit Anti-Apolipoprotein E antibody

SL0167R

Product Name:	Apolipoprotein E
Chinese Name:	载LipoproteinE抗体
Alias:	Apolipoprotein E precursor; AD2; Alzheimer disease 2; Apo E; ApoE; APOEA; ApolipoproteinE; Apolipoprotein-E; Apoprotein; MGC1571; Apo E2; ApoE2; APOE 2; Apolipoprotein E2; LDLCQ5; LPG; APOE_HUMAN; Apo-E.
文献引用 PubMed :	Specific References(1) SL0167R has been referenced in 1 publications. [IF=3.11]Chiu, Chen-Yuan, et al. "Supplementation of chitosan alleviates high-fat diet-enhanced lipogenesis in rats via AMP-activated protein kinase activation and inhibition of lipogenesis-associated genes." Journal of Agricultural and Food Chemistry (2015).WB;Rat. PubMed:25756465
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Mouse,Rat,
Applications:	WB=1:500-2000ELISA=1:500-1000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	32kDa
Cellular localization:	Secretory protein
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from mouse Apo E:211-311/311
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year

	when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	<p>Apolipoprotein E, a main apoprotein of the chylomicron, binds to a specific receptor on liver cells and peripheral cells and is essential for the normal catabolism of triglyceride-rich lipoprotein constituents. ApoE exists in three major isoforms; E2, E3, and E4, which differ from one another by a single amino-acid substitution. Compared with E3 and E4, E2 exhibits the lowest receptor binding affinity. Defects in ApoE are a cause of hyperlipoproteinemia type III due to increased plasma cholesterol and triglycerides levels which are the consequence of impaired clearance of chylomicron and VLDL remnants.</p> <p>Summary: Chylomicron remnants and very low density lipoprotein (VLDL) remnants are rapidly removed from the circulation by receptor-mediated endocytosis in the liver. Apolipoprotein E, a main apoprotein of the chylomicron, binds to a specific receptor on liver cells and peripheral cells. ApoE is essential for the normal catabolism of triglyceride-rich lipoprotein constituents. The APOE gene is mapped to chromosome 19 in a cluster with APOC1 and APOC2. Defects in apolipoprotein E result in familial dysbetalipoproteinemia, or type III hyperlipoproteinemia (HLP III), in which increased plasma cholesterol and triglycerides are the consequence of impaired clearance of chylomicron and VLDL remnants. [provided by RefSeq, Jul 2008].</p> <p>Function: Mediates the binding, internalization, and catabolism of lipoprotein particles. It can serve as a ligand for the LDL (apo B/E) receptor and for the specific apo-E receptor (chylomicron remnant) of hepatic tissues.</p> <p>Subcellular Location: Secreted.</p> <p>Tissue Specificity: Occurs in all lipoprotein fractions in plasma. It constitutes 10-20% of very low density lipoproteins (VLDL) and 1-2% of high density lipoproteins (HDL). APOE is produced in most organs. Significant quantities are produced in liver, brain, spleen, lung, adrenal, ovary, kidney and muscle.</p> <p>Post-translational modifications: Synthesized with the sialic acid attached by O-glycosidic linkage and is subsequently desialylated in plasma. O-glycosylated with core 1 or possibly core 8 glycans. Thr-307 is a minor glycosylation site compared to Ser-308. Glycated in plasma VLDL of normal subjects, and of hyperglycemic diabetic patients at a higher level (2-3 fold). Phosphorylation sites are present in the extracellular medium.</p> <p>DISEASE: Defects in APOE are a cause of hyperlipoproteinemia type 3 (HLPP3) [MIM:107741]; also known as familial dysbetalipoproteinemia. Individuals with HLPP3 are clinically</p>

characterized by xanthomas, yellowish lipid deposits in the palmar crease, or less specific on tendons and on elbows. The disorder rarely manifests before the third decade in men. In women, it is usually expressed only after the menopause. The vast majority of the patients are homozygous for APOE*2 alleles. More severe cases of HLPP3 have also been observed in individuals heterozygous for rare APOE variants. The influence of APOE on lipid levels is often suggested to have major implications for the risk of coronary artery disease (CAD). Individuals carrying the common APOE*4 variant are at higher risk of CAD.

Genetic variations in APOE are associated with Alzheimer disease type 2 (AD2) [MIM:104310]. It is a late-onset 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. Note=The APOE*4 allele is genetically associated with the common late onset familial and sporadic forms of Alzheimer disease. Risk for AD increased from 20% to 90% and mean age at onset decreased from 84 to 68 years with increasing number of APOE*4 alleles in 42 families with late onset AD. Thus APOE*4 gene dose is a major risk factor for late onset AD and, in these families, homozygosity for APOE*4 was virtually sufficient to cause AD by age 80. The mechanism by which APOE*4 participates in pathogenesis is not known.

Defects in APOE are a cause of sea-blue histiocyte disease (SBHD) [MIM:269600]; also known as sea-blue histiocytosis. This disorder is characterized by splenomegaly, mild thrombocytopenia and, in the bone marrow, numerous histiocytes containing cytoplasmic granules which stain bright blue with the usual hematologic stains. The syndrome is the consequence of an inherited metabolic defect analogous to Gaucher disease and other sphingolipidoses.

Defects in APOE are a cause of lipoprotein glomerulopathy (LPG) [MIM:611771]. LPG is an uncommon kidney disease characterized by proteinuria, progressive kidney failure, and distinctive lipoprotein thrombi in glomerular capillaries. It mainly affects people of Japanese and Chinese origin. The disorder has rarely been described in Caucasians.

Similarity:

Belongs to the apolipoprotein A1/A4/E family.

SWISS:

P08226

Gene ID:

11816

Database links:

[Entrez Gene: 348](#)Human

[Entrez Gene: 11816](#)Mouse

[Oimim: 107741](#)Human

[SwissProt: P02649](#)Human

[SwissProt: P08226](#)Mouse

[Unigene: 654439](#)Human

[Unigene: 305152](#)Mouse

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

ApoE

是在肝脏中合成的极低密度Lipoprotein的组分, 也是在细胞间转运胆固醇的高密度Lipoprotein的一种亚类.

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