Anti-Apolipoprotein E antibody ab20874

Overview

Product name
Anti-Apolipoprotein E antibody

Description
Rabbit polyclonal to Apolipoprotein E

Host species
Rabbit

Specificity
Ab20874 is extremely low recognition of human Apo E. No data available regarding rat activity. We have data to indicate that this antibody may not cross react with Human. However, this has not been conclusively tested and expression levels may vary in certain cell lines/tissues.

Tested applications
Suitable for: RID, Immunodiffusion, Immunelectrophoresis, WB, IHC-Fr

Species reactivity
Reacts with: Mouse, Rat

Immunogen
Full length native protein (purified) corresponding to Mouse Apolipoprotein E.

Properties

Form
Liquid

Storage instructions
Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.

Storage buffer
Preservative: 0.05% Sodium Azide

Purity
Whole antiserum

Purification notes
The product is heat inactivated and 0.22um filtered.

Clonality
Polyclonal

Isotype
IgG

Applications

Our Abpromise guarantee covers the use of ab20874 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

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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.

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.

Involvement in disease

Defects in APOE are a cause of hyperlipoproteinemia type 3 (HLPP3) [MIM:107741]; also known as familial dysbetalipoproteinemia. Individuals with HLPP3 are clinically 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. 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.

Sequence similarities

Belongs to the apolipoprotein A1/A4/E family.
**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.

**Cellular localization**

Secreted.

**Images**

Anti-Apolipoprotein E antibody (ab20874) + 20ug rat hippocampal lysate

**Secondary**

anti-rabbit HRP-labeled secondary antibody

**Predicted band size:** 36 kDa

Rabbit polyclonal to Apolipoprotein E (ab20874) in western blot analysis of proteins isolated from 100mg of rat brain hippocampus. Tissue was homogenized in a 5V of gentle lysis buffer (10mM HEPES, 1mM EGTA, 5mM MgCl2, 142.5mM KCl, 0.25% NP-40, with Roche complete Protease Inhibitor Cocktail Tablets) and centrifuged 20 mins/14000rpm/4°C. Supernatants reserved and protein concentrations were assigned by Bradford method. Proteins were resolved onto 10% SDS-PAGE and blotted on nitrocellulose membranes (Amersham- Hybond C). The membranes were incubated with anti-ApoE (ab20874 at 1/400) for 2h at rm temp in 0.05% TBST (150 mM NaCl, 50 mM Tris, pH 7.0, and 0.05% Tween 20) with 3% milk. Following incubation with the anti-rabbit HRP-labeled secondary antibody (1/10000 in 0.05% TBST, 1h rm temp), blots were developed using the enhanced chemiluminescent substrate (ECL, Amersham Bioscience).
ab20874 staining Apolipoprotein E in rat brain tissue by Immunohistochemistry (Frozen sections). After fixation in 4% PFA in PBS (pH 7.4), brains were cryoprotected in 30% sucrose in PBS at +4°C, frozen, and thereafter kept at -80°C until used. For the immunostaining, brains were cryosectioned in 30 µm slices, collected in PBS (pH 7.4), and processed in a free-floating system. Slices were first incubated in the blocking solution (BS) containing 2% bovine serum albumin, 2% goat serum, and 0.1% Triton X-100 in PBS (pH 7.4) for 1 hour at room temperature, and thereafter with the primary antibody for 24–48 hours at +4°C in BS at a 1/1000 dilution. After washing in PBS, slices were incubated with the biotin-conjugated secondary antibody (1/4000) in BS, rinsed with PBS, and incubated with the avidin-peroxidase conjugate in BS for 1 hour at room temperature. The staining was detected using 3,3-diaminobenzidinetetrahydrochloride as a chromogen.

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