Product datasheet

Anti-Apolipoprotein E antibody ab7620

Overview

**Product name**
Anti-Apolipoprotein E antibody

**Description**
Goat polyclonal to Apolipoprotein E

**Host species**
Goat

**Specificity**
Typically less than 1% cross reactivity against other types of apoLipoprotein was detected by ELISA. This antibody reacts with human apoLipoprotein E and has negligible cross-reactivity with Type A-I, A-II, B, C-I, C-II, C-III and J apoLipoproteins.

**Tested applications**
Suitable for: Indirect ELISA, WB, ICC/IF

**Species reactivity**
Reacts with: Rat, Human

**Immunogen**
Full length native protein (purified)(Human) (isolated from human plasma by density gradient centrifugation followed by HPLC purification).

**Positive control**
Purified apolipoprotein E This antibody gave a positive result when used in the following methanol fixed cell lines: MCF-7.

**General notes**
This antibody has been used to determine that atherosclerotic lesions in the human aorta contain considerable amounts of lipoproteins. These lipoproteins were observed to be complexed with components of the extracellular matrix (especially LDL and proteoglycans). The role of these matrix-lipoprotein complexes is not entirely clear, however, animal models of atherosclerosis have shown that increased cellular proliferation and increased production of extracellular matrix components occur following injury to the intimal layer of the aorta.

Properties

**Form**
Liquid

**Storage instructions**
Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.

**Storage buffer**
pH: 8.00
Preservative: 0.01% Sodium azide
Constituents: 4.7625% Sodium borate, 0.146% EDTA, 0.435% Sodium chloride

**Purity**
Immunogen affinity purified

**Purification notes**
This product has been prepared by immunoaffinity chromatography using immobilized antigens followed by extensive cross-adsorption against other apoLipoproteins and human serum proteins to remove any unwanted specificities.

**Clonality**
Polyclonal

2 Abreviews 11 References 2 Images
Isotype

IgG

Applications

Our Abpromise guarantee covers the use of ab7620 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

<table>
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<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
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</thead>
<tbody>
<tr>
<td>Indirect ELISA</td>
<td>1/4000 - 1/8000.</td>
<td></td>
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<tr>
<td>WB</td>
<td>1/5000 - 1/10000. Predicted molecular weight: 36 kDa.</td>
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<tr>
<td>ICC/IF</td>
<td>Use a concentration of 10 µg/ml.</td>
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Target

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. 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.

**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 (ab7620) at 1/1000 dilution (in PBS/Tween (0.01%) + 3% milk for 12 hours) + Whole cell lysate of Rat glial cells at 40 µg

**Secondary**
An HRP-conjugated Rabbit anti-goat Polyclonal at 1/200 dilution

Developed using the ECL technique.

Performed under reducing conditions.

**Predicted band size:** 36 kDa

**Observed band size:** 35-36 kDa

**why is the actual band size different from the predicted?**

**Additional bands at:** 55 kDa. We are unsure as to the identity of these extra bands.

**Blocking Step:** 3% Milk for 20 minutes at room temperature
ICC/IF image of ab7620 stained MCF-7 cells. The cells were 100% methanol fixed (5 min) and then incubated in 1%BSA / 10% normal donkey serum / 0.3M glycine in 0.1% PBS-Tween for 1h to permeabilise the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody ab7620 at 10µg/ml overnight at +4°C. The secondary antibody (green) was DyLight® 488 donkey anti- goat (ab96931) IgG (H+L) used at a 1/250 dilution for 1h. Alexa Fluor® 594 WGA was used to label plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43µM.

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