

Product datasheet

Anti-Apolipoprotein E antibody (HRP) ab20261

★★★★☆ 1 Abreviews 5 References

Overview

Product name	Anti-Apolipoprotein E antibody (HRP)
Description	Goat polyclonal to Apolipoprotein E (HRP)
Host species	Goat
Conjugation	HRP
Specificity	No cross reactivity with human Apolipoprotein (Apo) AI, Apo AII, Apo B-100, Apo CI, Apo CII and Apo CIII.
Tested applications	Suitable for: WB, ELISA
Species reactivity	Reacts with: Cow, Human
Immunogen	Full length native protein (purified) (Human).

Properties

Form	Lyophilised: Add 0.5 ml distilled water and 0.5ml glycerol to final conc. of 50% glycerol and store at -20°C. Centrifuge product if not completely clear after standing for 1-2 hours at room temperature.
Storage instructions	Shipped at 4°C. Store at +4°C.
Storage buffer	Preservative: 0.005% Thimerosal (merthiolate) Constituents: 50% Glycerol added to give a final volume of 1mL, 5mg/ml BSA, 0.05M Sodium chloride, 25mM PBS, pH 7.4
Purity	Immunogen affinity purified
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab20261** in the following tested applications.

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

Application	Abreviews	Notes
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Application	Abreviews	Notes
WB	★★★★☆	Use at an assay dependent dilution. Predicted molecular weight: 36 kDa. Dilution optimised using Chromogenic detection.
ELISA		1/2000 - 1/8000.

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.

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