abcam

Product datasheet

Anti-Apolipoprotein E antibody [E6D7] ab1907

★★★★★ 7 Abreviews 13 References 2 图像

概述

产**品名称** Anti-Apolipoprotein E**抗体**[E6D7]

小鼠单克隆抗体[E6D7] to Apolipoprotein E

宿主 Mouse

经测试应用 适用于: IHC-P, ICC/IF

种属反应性 与反应: Human

免疫原 Synthetic peptide corresponding to Apolipoprotein E. Spans the polymorphic amino acid position

158.

阳性对照 IHC-P: Human liver tissue. ICC/IF: HepG2 cells. WB: Human THP-1 macrophage whole cell lysate

常规说明 This product was changed from ascites to tissue culture supernatant on 20/05/2019. Please note

that the dilutions may need to be adjusted accordingly. If you have any questions, please do not

hesitate to contact our scientific support team.

The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets

your needs before purchasing.

If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be

found below, along with publications, customer reviews and Q&As

性能

形式 Liquid

存放说明 Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw

cycles.

存储溶液 Constituent: PBS

纯**度** Affinity purified

克隆 单克隆

克隆编号 E6D7

同种型 lgG1

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The Abpromise guarantee

Abpromise™承诺保证使用ab1907于以下的经测试应用

"应用说明"部分下显示的仅为推荐的起始稀释度;实际最佳的稀释度/浓度应由使用者检定。

应用	Ab评论	说明
IHC-P	★★★★ (2)	Use at an assay dependent concentration. Perform heat mediated antigen retrieval before commencing with IHC staining protocol.
ICC/IF		Use at an assay dependent concentration.

靶标

功能

组织特异性

疾病相关

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.

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.

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 plagues is the neurotoxic amyloid-beta-APP 40-42 peptide (s), derived proteolytically from the transmembrane precursor protein APP by sequential secretase processing. The cytotoxic Cterminal 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.

Belongs to the apolipoprotein A1/A4/E family.

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).

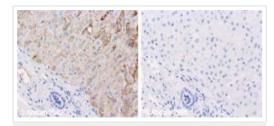
Phosphorylation sites are present in the extracellular medium.

细胞定位 Secreted.

图片

序列相似性

翻译后修饰



Immunohistochemistry (Formalin/PFA-fixed paraffinembedded sections) - Anti-Apolipoprotein E antibody [E6D7] (ab1907)

Ab1907 staining Human liver. Staining is localised to cytoplasmic and extracelular regions.

Left panel: with primary antibody at 1 ug/ml. Right panel: isotype control.

Sections were stained using an automated system DAKO Autostainer Plus, at room temperature: sections were rehydrated and antigen retrieved with the Dako 3 in 1 AR buffers citrate EDTA pH 9.0 in a DAKO PT Link. Slides were peroxidase blocked in 3% H2O2 in methanol for 10 mins. They were then blocked with Dako Protein block for 10 minutes (containing casein 0.25% in PBS) then incubated with primary antibody for 20 min and detected with Dako envision flex amplification kit for 30 minutes. Colorimetric detection was completed with Diaminobenzidine for 5 minutes. Slides were counterstained with Haematoxylin and coverslipped under DePeX. Please note that for manual staining we recommend to optimize the primary antibody concentration and incubation time (overnight incubation), and amplification may be required.

This image was generated using the ascites version of the product.

ICC/IF image of ab1907 stained HepG2 cells. The cells were 100%

methanol fixed (5 min) and then incubated in 1%BSA / 10% normal Apolipoprotein E antibody [E6D7] (ab1907) goat 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 (ab1907, 1µg/ml) overnight at +4°C. The secondary antibody (green) was Alexa Fluor® 488 goat anti-mouse IgG (H+L) used at a 1/1000 dilution for 1h. Alexa Fluor® 594 WGA was used to label

This image was generated using the ascites version of the product.

to stain the cell nuclei (blue) at a concentration of 1.43µM.

plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used

Immunocytochemistry/ Immunofluorescence - Anti-

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