**Product datasheet**

**Anti-Apolipoprotein E antibody [D6E10] ab1906**

★★★★☆ 8 Abreviews  34 References  画像数 5

製品の概要

製品名
Anti-Apolipoprotein E antibody [D6E10]

製品の詳細
Mouse monoclonal [D6E10] to Apolipoprotein E

由来種
Mouse

特異性
Mouse reactivity. Please be aware that we have received positive as well as negative feedback for reactivity of this antibody with mouse samples. The antibody is not being batch-tested in the mouse samples. Anti-Apolipoprotein E antibody [D6E10] recognizes the E2, E3 and E4 isoforms of apolipoprotein E. It was raised against a peptide sequence corresponding to aa 141-160 of human Apo-E.

アプリケーション
適用あり:  

IHC-P

交差種
Human

免疫原
Synthetic peptide corresponding to Apolipoprotein E aa 141-160.

Sequence:
QAMLGQSTEE LRVRLASHLR

Run BLAST with

Run BLAST with

特記事項

This product was changed from ascites to tissue culture supernatant on 2nd February 2018. 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.

製品の特性

製品の状態
Liquid

保存方法
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.

バッファー
Constituent: PBS

特記事項(精製)
Purified from TCS

ポリモノ
モノクローナル

クローニ名
D6E10

アイソタイプ
IgG1

アプリケーション
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 have been clinically characterized by xanthomas, yellowish lipid deposits in the palm and 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.

Our Abpromise guarantee covers the use of ab1906 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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<th>アプリケーション</th>
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<tr>
<td>WB</td>
<td>★★★★☆</td>
<td>Use a concentration of 0.5 - 1 µg/ml. Predicted molecular weight: 38 kDa.</td>
</tr>
<tr>
<td>IHC-P</td>
<td>★★★★★☆</td>
<td>Use a concentration of 5 - 10 µg/ml. Antigen retrieval is not essential but may optimise staining. The staining intensity of formalin-fixed paraffin embedded tissues may be significantly improved by pretreatment methods such as: 70% Formic acid for 10-30 minutes at room temperature or Hydrolytic autoclaving.</td>
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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.

Secreted.

**Western blot - Anti-Apolipoprotein E antibody [D6E10] (ab1906)**

All lanes: Anti-Apolipoprotein E antibody [D6E10] (ab1906) at 1 µg/ml

Lane 1: Recombinant Apolipoprotein E2 protein, 40 ng
Lane 2: Recombinant Apolipoprotein E3 protein, 40 ng
Lane 3: Recombinant Apolipoprotein E4 protein, 40 ng
Lane 4: Human plasma at 40 µg

Developed using the ECL technique.

**Predicted band size:** 38 kDa

**Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Apolipoprotein E antibody [D6E10] (ab1906)**

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of Alzheimer’s disease brain tissue labelling Apolipoprotein E with ab1906. The tissue was incubated with 5 µg/mL of the primary antibody overnight at 4°C. Antigen retrieval was performed using Sodium Citrate H.I.E.R. Counterstained with hematoxylin. The image was captured with a 40X objective. Scale bar: 50 µm.
Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human cerebellum tissue labelling Apolipoprotein E with ab1906. The tissue was incubated with 10 µg/mL of the primary antibody overnight at 4°C. Antigen retrieval was performed using Sodium Citrate H.I.E.R. Counterstained with hematoxylin. The image was captured with a 40X objective. Scale bar: 50 µm.

All lanes: Anti-Apolipoprotein E antibody [D6E10] (ab1906) at 1/500 dilution

All lanes: SCC61 whole cell lysate

Lysates/proteins at 30 µg per lane.

Secondary

All lanes: HRP-conjugated goat anti-mouse IgG at 1/5000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 38 kDa

Observed band size: 40 kDa

why is the actual band size different from the predicted?

Additional bands at: 60 kDa (possible non-specific binding)

Exposure time: 2 minutes

Blocked with 5% milk for 1 hour at 22°C.

Incubated with the primary antibody in 5% milk for 16 hours at 4°C.
ab1906 staining Apolipoprotein E in human testis tissue sections by Immunohistochemistry (IHC-P - paraformaldehyde-fixed, paraffin-embedded sections). Tissue was fixed with formaldehyde and blocked with 1% BSA for 10 minutes at 21°C; antigen retrieval was by heat mediation in citric acid. Samples were incubated with primary antibody (1/200 in TBS/BSA/azide) for 2 hours at 21°C. An undiluted biotin-conjugated goat anti-mouse IgG polyclonal was used as the secondary antibody.

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