

Datasheet for 600-101-198

APOLIPOPROTEIN J Antibody**Overview**

Description:	Anti-Apolipoprotein J (GOAT) Antibody - 600-101-198
Item No.:	600-101-198
Size:	500 µg
Applications:	WB
Reactivity:	Human
Host Species:	Goat

Product Details

Background:	Apolipoprotein J Antibody functions as a secreted chaperone that prevents aggregation of nonnative proteins. It prevents stress-induced aggregation of blood plasma proteins and inhibits the formations of amyloid fibrils. Apolipoprotein J does not require ATP or refold proteins by itself. It maintains partially unfolded proteins in a state for subsequent refolding by other chaperones. It is shown to be involved in several basic biological events such as cell death, tumor progression, and neurodegenerative disorders. Binding to cell surface receptors triggers internalization of chaperone-client complex and subsequent lysosomal or proteasomal degradation. It modulates NF-kappa-B transcriptional activity. Nuclear isoforms promote apoptosis while mitochondrial isoforms suppress BAX-dependent release of cytochrome c into the cytoplasm and inhibit apoptosis. Anti-Apolipoprotein J Antibody is useful for researchers interested in the immune system, Ubiquitin pathways, and cardiovascular research.
Synonyms:	goat anti-Apolipoprotein J antibody, Clusterin, Complement-associated protein SP-40, Complement cytolysis inhibitor, CLI, NA1/NA2, Apo-J, Testosterone-repressed prostate message 2, TRPM-2, Ku70-binding protein 1, Aging-associated gene 4 protein, Clusterin beta chain, Clusterin alpha chain, Complement cytolysis inhibitor a chain, Complement cytolysis inhibitor b chain, ApoJ alpha, ApoJ beta, CLU, CLI, KUB1, AAG4
Host Species:	Goat
Clonality:	Polyclonal
Format:	IgG

Target Details

Gene Name:	CLU
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Reactivity:	Human
Immunogen Type:	Native Protein
Immunogen:	apoLipoprotein Type J was isolated from human plasma by density gradient centrifugation followed by HPLC purification.
Purity/Specificity:	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. Typically less than 1% cross reactivity against other types of apoLipoprotein was detected by ELISA against purified standards. This antibody reacts with human apoLipoprotein J and has negligible cross-reactivity with Type A-I, A-II, B, C-I, C-II, C-III and E apoLipoproteins. Specific cross reaction of anti-apoLipoprotein antibodies with antigens from other species has not been determined. Non-specific cross reaction of anti-apoLipoprotein antibodies with other human serum proteins is negligible.
Relevant Links:	• 600-101-198 SDS • UniProtKB - P10909 • NCBI - NP_001822.3 • GenelD - 1191

Application Details

Suggested Applications:	WB (Based on references)
Application Note:	Anti-apolipoprotein antibodies have been used for indirect trapping ELISA for quantitation of antigen in serum using a standard curve, for immunoprecipitation and for western blotting for highly sensitive qualitative analysis.
Assay Dilutions:	All assays should be optimized by the user. Recommended dilutions (if any) may be listed below.
ELISA:	1:5,000 - 1:10,000
IHC:	1:50 - 1:200
IP:	1:100
WB:	1:5,000 - 1:10,000

Formulation

Physical State:	Liquid (sterile filtered)
Concentration:	1.0 mg/mL by UV absorbance at 280 nm
Buffer:	0.125 M Sodium Borate, 0.075 M Sodium Chloride, 0.005 M EDTA, pH 8.0

Preservative: 0.01% (w/v) Sodium Azide

Stabilizer: None

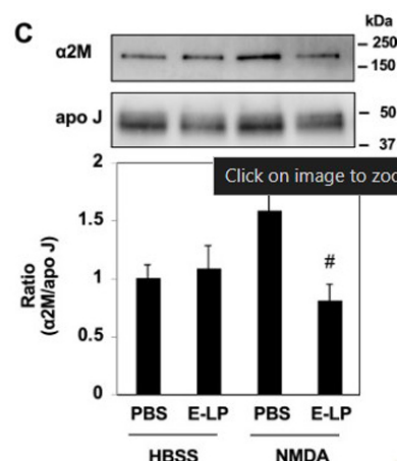
Shipping & Handling

Shipping Condition: Wet Ice

Storage Condition: Store vial at 4° C prior to opening. This product is stable at 4° C as an undiluted liquid. Dilute only prior to immediate use. For extended storage mix with an equal volume of glycerol, aliquot contents and freeze at -20° C or below. Avoid cycles of freezing and thawing.

Expiration: Expiration date is one (1) year from date of receipt.

Images

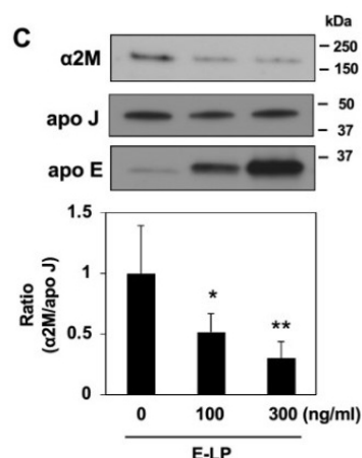


Western Blot

Western Blot of Anti-Apolipoprotein J Antibody.

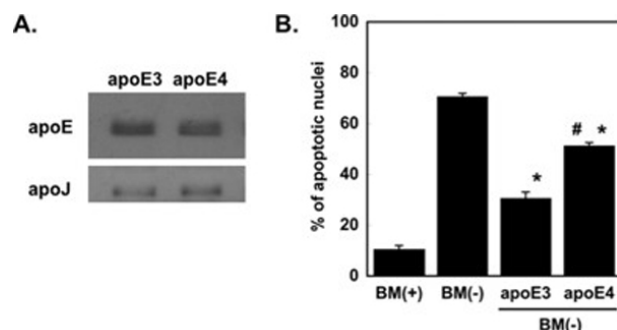
E-LPs attenuate the increased secretion of $\alpha 2$ -macroglobulin induced by NMDA in primary cultures of mixed retinal cells.

(C) Conditioned media (HBSS + PBS, n = 5 cell cultures; HBSS + E-LP, n = 5 cell cultures; NMDA + PBS, n = 6 cell cultures; and NMDA + E-LP, n = 6 cell cultures) of the mixed retinal cells were collected 24 hours after treatment with HBSS or 300 μ M NMDA treatment with PBS or 300 ng/mL E-LP, and subjected to immunoblotting with antibodies directed against $\alpha 2$ -macroglobulin or apo J. The protein levels were normalized by the corresponding apo J levels. Data are means $\pm$ SD. *, P = 0.0007 for HBSS + PBS versus NMDA + PBS. #, P < 0.0001 for NMDA + PBS versus NMDA + 300 ng/mL E-LP. Data were analyzed with the 1-way ANOVA, Tukey's multiple comparisons test. Figure 3. PMID: 34698771



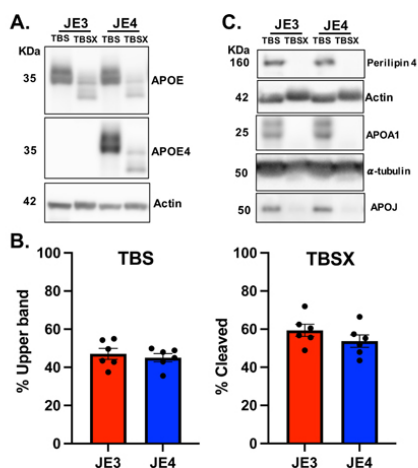
Western Blot

Western Blot of Anti-Apolipoprotein J Antibody. E-LPs reduce the expression and secretion of $\alpha 2$ -macroglobulin in primary cultured Müller glia. (C) Conditioned media C of Müller glia were collected 24 hours after 0, 100, or 300 ng/mL E-LP treatment (n = 5 cell cultures), and subjected to immunoblotting with antibodies directed against $\alpha 2$ -macroglobulin, apolipoprotein J, apolipoprotein E, or β -actin. The protein levels were normalized by the corresponding apo J C or β -actin D levels. Data are means $\pm$ SD. C *, P = 0.0483 or **, P = 0.0059 for 0 ng/mL E-LP versus 100 or 300 ng/mL E-LP, respectively. D *, P < 0.0001 for 0 ng/mL E-LP versus 100 or 300 ng/mL E-LP. Data were analyzed with the 1-way ANOVA, Tukey's multiple comparisons test. Figure 4. PMID: 34698771



Western Blot

ApoE4-containing LpE less effectively protect RGCs against apoptosis than do apoE3-containing LpE. Cortical glia were isolated from mice lacking endogenous apoE but expressing human apoE3 or human apoE4. LpE were isolated from conditioned medium. A, equal amounts of LpE protein (10 μ g) were separated by polyacrylamide gel electrophoresis and immunoblotted with antibodies directed against apoE and apoJ. The experiment was repeated two additional times with similar results. B, LpE secreted by mouse glia expressing human apoE3 or apoE4 were given to RGCs for 24 h in medium lacking trophic additives (BM(-)). Control cultures were supplied with trophic additives (BM(+)). The number of apoptotic neurons was determined by Hoechst staining and is given as percentage of total number of neurons. Data are the means $\pm$ S.E. from four independent experiments. *, p < 0.005 for BM(-) versus BM (+) + apoE3 and for BM(-) + apoE4. #, p < 0.001 for apoE3 versus apoE4. Statistical analysis was performed using one-way analysis of variance followed by Bonferroni's multiple comparison. Fig 6. PMID: 19717566



Western Blot

Western blot analysis of cortical brain fractions demonstrated brain APOE from JAX-E3 (JE3) and JAX-E4 (JE4) as a doublet in the TBS fraction as a series of full-length and smaller bands in the TBSX fraction (A). Double band from TBS and TBSX fraction were quantified and expressed as percentage of upper bands with respect of total APOE detected (B). Western blot of cortical TBS and TBSX fractions showed the TBS fraction contains proteins in lipid droplets (Perilipin 4), and in brain lipoproteins (APOA1, and APOJ) (C). Bar graphs represent the mean ± SEM (n = 6 JE3; n = 6 JE4), n = number of animals. A two-tailed student t-test was used to assess outcome from percentage of upper band in TBS and TBSX soluble APOE. Fig 2.

PMID: 35838553

References

- Sepulveda J et al. Independent APOE4 knock-in mouse models display reduced brain APOE protein, altered neuroinflammation, and simplification of dendritic spines. *J Neurochem.* (2022)
- Hayashi H et al. Apolipoprotein E-Containing Lipoproteins and LRP1 Protect From NMDA-Induced Excitotoxicity Associated With Reducing α2-Macroglobulin in Müller Glia. *Invest Ophthalmol Vis Sci.* (2021)
- Hayashi H et al. Protection of neurons from apoptosis by apolipoprotein E-containing lipoproteins does not require lipoprotein uptake and involves activation of phospholipase Cγ1 and inhibition of calcineurin. *J Biol Chem.* (2009)

Disclaimer

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