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Datasheet for 600-101-111

APOLIPOPROTEIN B Antibody

Overview

Description:	Anti-Apolipoprotein B (GOAT) Antibody - 600-101-111
Item No.:	600-101-111
Size:	1 mg
Applications:	ELISA, EM, IF, IHC, IP, Multiplex, WB
Reactivity:	Human
Host Species:	Goat

Product Details

Background:	Anti Apolipoprotein B antibody recognizes the gene product of APOB that is the primary apolipoprotein of low-density lipoproteins, which is responsible for carrying cholesterol to tissues. While it is unclear exactly what functional role APOB plays in LDL, it is the primary apolipoprotein component and is absolutely required for its formation. What is clear is that the APOB on the LDL particle acts as a ligand for LDL receptors in various cells throughout the body. Through a mechanism that is not fully understood, high levels of APOB can lead to plaques that cause vascular disease (atherosclerosis), leading to heart disease. There is considerable evidence that levels of APOB are a better indicator of heart disease and cardiovascular risk than total cholesterol or LDL. However, primarily for historic reasons, cholesterol, and more specifically, LDL-cholesterol, remains the primary lipid test for the risk factor of atherosclerosis.
Synonyms:	goat anti-Apolipoprotein B antibody, Apo B-100, Apo B-48, APOB protein antibody, Apolipoprotein-B 100 antibody, Apolipoprotein B 48 antibody, Apolipoprotein B antibody, FLDB antibody
Host Species:	Goat
Clonality:	Polyclonal
Format:	IgG

Target Details

Gene Name:	APOB
Reactivity:	Human

Immunogen Type:	Native Protein
Immunogen:	apoLipoprotein Type B 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 B and has negligible cross-reactivity with Type A-I, A-II, C-I, C-II, C-III, E and J 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:	• 600-101-111 SDS • UniProtKB - Q7Z7Q0 • NCBI - AAH51278.1 • GeneID - 338

Application Details

Suggested Applications:	ELISA, EM, IF, IHC, IP, Multiplex, 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:	1:2,000 - 1:10,000
IF:	User Optimized
IHC:	1:50 - 1:500
IP:	1:100
WB:	1:200 - 1:1,000

Formulation

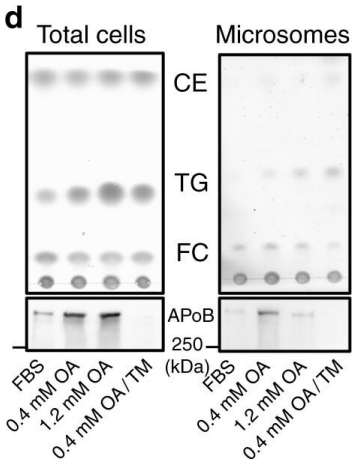
Physical State:	Liquid (sterile filtered)
Concentration:	1.0 mg/mL by UV absorbance at 280 nm
Buffer:	0.125 M Sodium Borate, 0.075 M Sodium Chloride, 0.005 M EDTA, pH 8.0

Preservative:	0.01% (w/v) Sodium Azide
Stabilizer:	None

Shipping & Handling

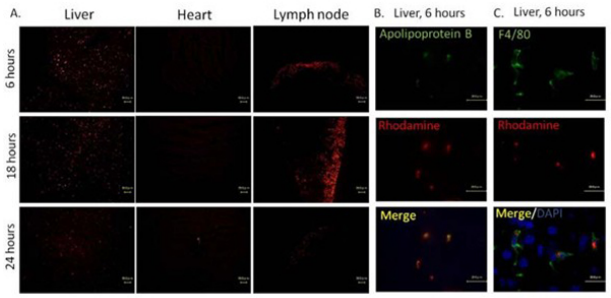
Shipping Condition:	Wet Ice
Storage Condition:	Store vial at 4° C prior to opening. This product is stable 4° C as an undiluted liquid. Dilute only prior to immediate use. For extended storage mix with an equal volume of glycerol, aliquot contents and freeze at -20° C or below. Avoid cycles of freezing and thawing.
Expiration:	Expiration date is one (1) year from date of receipt.

Images

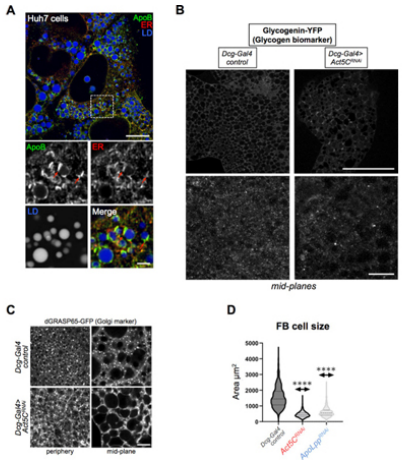


Western Blot

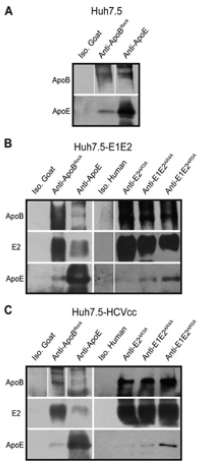
LDs are present in the lumen of the type I NR. a Huh7 treated with OA/TM for 24–48 h harbored LDs in the lumen of the ER (left; arrowheads) and within the type I NR (right; arrows mark the NR). INM: inner nuclear membrane, ONM: outer nuclear membrane. Bars, 0.2 µm. b Huh7-expressing HRP-KDEL treated with OA/TM for 24 h. DAB precipitated in the type I NR lumen (arrows). Bar, 0.5 µm. c Mouse hepatocytes in vivo after high-fat diet feeding for 6 weeks and TM injection. Lumenal LDs were observed in the ER (arrowheads), the nuclear cistern (arrowhead in the inset) (left figure; Bar, 0.5 µm), and in the type I NR (arrows mark the NR) (right figure; Bar, 0.2 µm). They contained more nuclear LDs than the control fed the high-fat diet and injected with vehicle alone. Mean ± SD of three independent experiments. *$p < 0.01$, Student's t test. d Microsomes of Huh7 treated with none, 0.4 mm OA, 1.2 mm OA, or OA/TM for 48 h. The OA/TM-treated cell microsome contained triglycerides (TG) and cholesterol esters (CE) most abundantly (by thin layer chromatography), but showed the lowest amount of ApoB (by Western blotting). e Three different kinds of LDs in the nuclear area: Nucleoplasmic LDs (A), NR-lumenal LDs (within the type I NR) (B), and cytoplasmic LDs (within the type II NR) (C). f Nucleoplasmic LDs (arrowheads) and NR-lumenal LDs (arrows) are distinguished by whether they are outside of or within LBR rings, respectively. Huh7 treated with OA/TM for 48 h. Both LDs were reduced by MTPi (100 nm BAY 13-9952). Bar, 10 µm. See also Supplementary Fig. 2e. g The number of nucleoplasmic LDs, NR-lumenal LDs, and cytoplasmic LDs within the type II NR were counted in randomly taken electron micrographs of Huh7 treated with OA/TM for 48–72 h. Box plot of pooled data from three independent experiments. The average is shown by +. Number of nuclei examined = 128. Source data are provided as a Source data file Figure provided by CiteAb. Source: Nat Commun, PMID: 30692541.



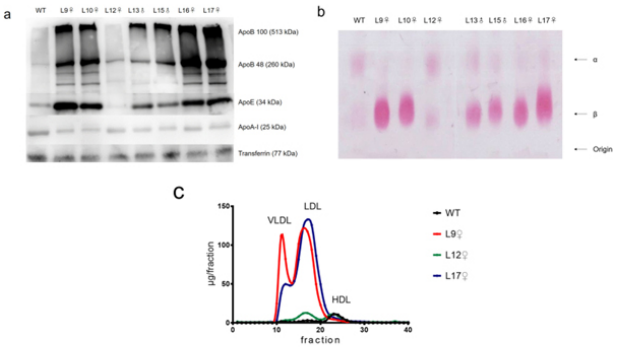
Immunofluorescence Microscopy
Rhodamine-labeled LDL in liver, heart and lymph node.
(A) Distribution of rhodamine-labeled LDL in liver, heart and lymph node 6, 18 and 24 hours after rhodamine-labeled LDL injection.
(B-C) Rhodamine signal co-localized with apolipoprotein B immunofluorescence (B) and inside F4/80 positive cells (C) in liver 6 hours after injection of rhodamine-labeled LDL. Figure 3.
PMID: 23756663



Immunofluorescence Microscopy
(A) Immuno-fluorescence (IF) confocal micrograph of human Huh7 hepatocytes immuno-stained for ApoB (green), the ER network (anti: calnexin, red), and co-stained for LDs (MDH, blue). Red arrows indicate ApoB. Scale bar is 4 µm. (B) Confocal mid-plane sections of larval FBs expressing glycogen binding protein glycogenin (Dcg-Gal4 >Glycogenin YFP). Glycogenin-YFP is distributed throughout FB cells. Scale bar 100 µm (top) and 10 µm (bottom). (C) Confocal images of L3 larvae from control or Act5CRNAi samples expressing Golgi-marker UAS-dGRASP65-GFP. Scale bar is 10 µm. (D) Violin plots of larval FB cell sizes for Dcg-Gal4 control, Dcg-Gal4 >Act5CRNAi, and Dcg-Gal4 >ApoLppRNAi. Figure 9—figure supplement 1.
PMID: 37144872

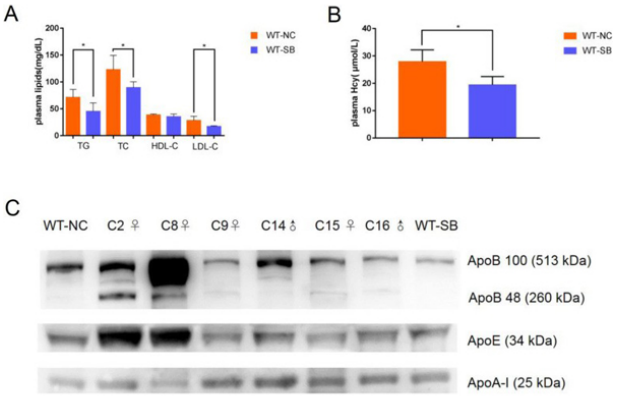


Western Blot
HCV E2, ApoB and ApoE form stable associations. Immunoprecipitations were performed on cell lysates from naïves, HCV glycoproteins transduced (Huh7.5-E1E2) or HCVcc JFH1 infected (Huh7.5-HCVcc) Huh7.5 cells, using indicated antibodies (top). The immunoprecipitates were separated by SDS-PAGE and analyzed by Western blotting with antibodies against the indicated proteins (left). Iso. Ab (Goat or Human): control with specific isotypic Ab. ApoE, ApoB, and E2 immunoprecipitations were performed separately but under the same experimental conditions. Fig 1.
PMID: 24838241



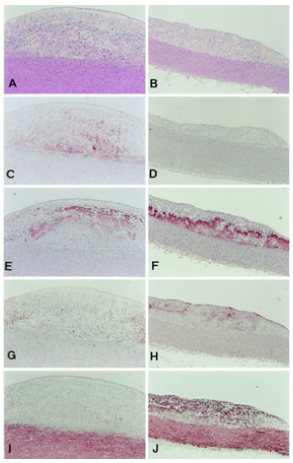
Western Blot

Analysis of plasma TC, TG, LDL-C, and HDL-C in the LDLR-KO rabbits.
A: Analysis of plasma apolipoproteins by Western blot. Plasma samples (0.5 μL) were fractionated on 4–15% SDS-PAGE and transferred to a PVDF membrane probed with Abs anti ApoA-I, ApoB, and ApoE as described in the Materials and Methods section.
B: Agarose gel electrophoresis of plasma lipoproteins. 4 μL of plasma was loaded in each well, fractionated on 1% agarose gel, and stained with fat red 7 B for neutral lipids. Lipoprotein migration positions are indicated by arrows.
C: Analysis of plasma lipoprotein profiles by fast-protein liquid chromatography (FPLC). Black, red, green, and blue lines showed the fractions of WT, L9♀, L12♀, and L17♀ respectively; VLDL, very low-density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.
Fig 3. PMID: 30243490

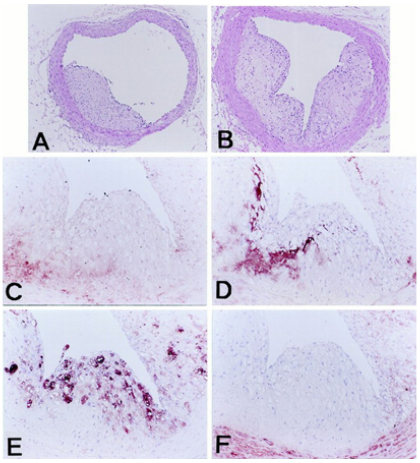


Western Blot

CBS-KO rabbits are hyperlipidemic. a Plasma TG, TC, HDL-C, LDL-C and b Hcy levels in 6 weeks-old WT rabbits (n = 6). Mean ± SD, *P < 0.05. c Immunoblot showing levels of plasma apolipoproteins in the indicated groups. WT-NC: wild type rabbits fed normal chow. WT-SB: wild type rabbits fed normal chow supplemented with the vitamin B and betaine complex. C2♀, C8♀: CBS-KO rabbits on a normal chow diet. C9♀, C14♂, C15♀, C16♂: CBS-KO rabbits fed normal chow supplemented with the vitamin B and betaine complex. Fig 3.
PMID: 33054837



Immunohistochemistry
Histological and immunohistochemical analysis of aortic atherosclerosis in transgenic and nontransgenic rabbits. Serial sections (5 µm thick) were taken at the thoracic aorta at the same position for each aorta and stained with hematoxylin-eosin (A and B), anti-human apo(a) (C and D), anti-apoB (E and F), anti-macrophage (G and H), anti–smooth muscle α-actin (I and J), anti-vimentin antibodies (K and L), anti-human apo(a) (M), anti-macrophage (N), and anti–smooth muscle α-actin antibodies (O). Magnification was as follows: panels A to J, ×50; panels K and L, ×100; and panels M to O, ×200. Note that mature SMCs in the media are clearly stained with anti–α-actin mAb. In foam cell–rich areas, there is apo(a) deposition in the extracellular matrix, but it is not associated with macrophages or SMCs (M to O). Figure 2. PMID: 11145938



Immunohistochemistry
Coronary artery micrographs. A and B, Coronary arteries with elevated fibrofatty plaque lesions from nontransgenic (A) and transgenic (B) rabbits fed a cholesterol diet for 16 weeks (hematoxylin-eosin staining). Transgenic rabbit coronary lesions were more extensive than those of control rabbits, as quantified in Table 2. C to F, Serial sections of transgenic rabbit coronary arteries stained with anti-human apo(a) (C), anti-apoB (D), anti-rabbit macrophage (E), and anti-rabbit smooth muscle α-actin (F) antibodies. Original magnification ×200. Apo(a) deposits are present in the center of the lesion core (C) and are partially associated with apoB (D). However, these areas are not directly associated with macrophages (E) or α-actin–positive SMCs (F). Note that intimal cells are partially positive for smooth muscle α-actin on the right side, where there is no apo(a) deposition.
Figure 3. PMID: 11145938

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