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Datasheet for 600-101-109

# APOLIPOPROTEIN A-I Antibody

## Overview

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| **Description:** | Anti-Apolipoprotein A-I (GOAT) Antibody - 600-101-109 |
| **Item No.:** | 600-101-109 |
| **Size:** | 1 mg |
| **Applications:** | IHC, WB, ELISA, IF |
| **Reactivity:** | Human |
| **Host Species:** | Goat |

## Product Details

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| **Background:** | Anti-Apolipoprotein A-I antibody recognizes the gene product of APOA1. Apolipoprotein promotes cholesterol efflux from tissues to the liver for excretion. Apolipoprotein A-I is the major protein component of high density lipoprotein (HDL) in the plasma. Synthesized in the liver and small intestine, it consists of two identical chains of 77 amino acids; an 18-amino acid signal peptide is removed co-translationally and a 6-amino acid propeptide is cleaved post-translationally. Variation in the latter step, in addition to modifications leading to so-called isoforms, is responsible for some of the polymorphism observed. APOA1 is a cofactor for lecithin cholesterolacyltransferase (LCAT) which is responsible for the formation of most plasma cholesteryl esters. The APOA1, APOC3 and APOA4 genes are closely linked in both rat and human genomes. The A-I and A-IV genes are transcribed from the same strand, while the C-III gene is transcribed convergently in relation to A-I. Defects in the apolipoprotein A-1 gene are associated with HDL deficiency and Tangier disease. Anti-Apolipoprotein A-I is useful for researchers interested in cardiovascular research. |
| **Synonyms:** | goat anti-Apolipoprotein A-I Antibody, goat anti-APOA1 antibody, goat anti-APO-A1 antibody, goat anti-APOA-1 antibody, APOA1/APOC3 fusion gene antibody, Apolipoprotein A I precursor antibody, Apolipoprotein AI antibody, Apolipoprotein of high density lipoprotein antibody, ProapoA-I, Proapolipoprotein A-I, ApoA-I |
| **Host Species:** | Goat |
| **Clonality:** | Polyclonal |
| **Format:** | IgG |

## Target Details

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| **Gene Name:** | APOA1 |
| **Reactivity:** | Human |
| **Immunogen Type:** | Native Protein |
| **Immunogen:** | apoLipoprotein Type A-I was isolated from human plasma by density gradient centrifugation followed by HPLC purification, followed by repeated immunizations in goat. |
| **Purity/Specificity:** | Goat Anti-Apolipoprotein A-I Antibody 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 A-I and has negligible cross-reactivity with Type A-II, B, 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:** | • 600-101-109 SDS<br>• UniProtKB - P02647<br>• NCBI - 4557321<br>• GeneID - 335 |

## Application Details

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| **Tested Applications:** | IHC, WB |
| **Suggested Applications:** | ELISA, IF (Based on references) |
| **Application Note:** | Anti-apoLipoprotein antibodies have been tested by Western blot and IHC and are suitable for indirect trapping ELISA for quantitation of antigen in serum using a standard curve, for immunoprecipitation, immunohistochemistry 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:** | 1:10,000 - 1:20,000 |
| **IF:** | User Optimized |
| **IHC:** | 1:50 - 1:200 |
| **IP:** | 1:100 |
| **WB:** | 1:1,000 - 1:2,000 |

## Formulation

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| **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

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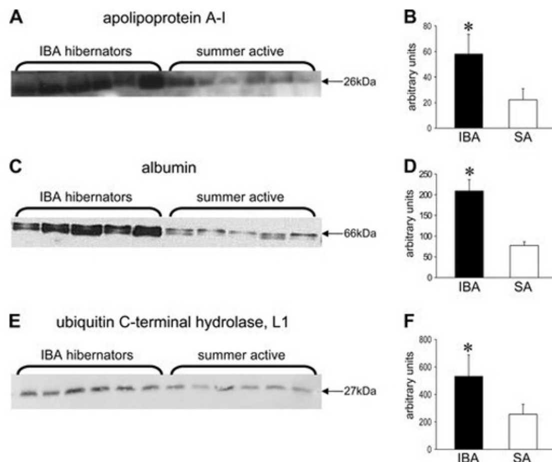
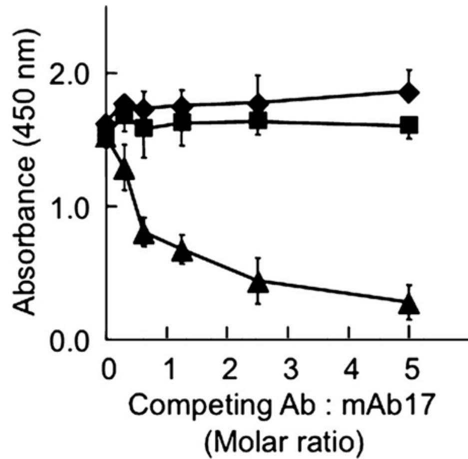
| **Shipping Condition:** | Wet Ice |
| **Storage Condition:** | Store vial at 4° C prior to opening. This product is stable at 4° C as an undiluted liquid. Dilute only prior to immediate use. For extended storage mix with an equal volume of glycerol, aliquot contents and freeze at -20° C or below. Avoid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

## Images

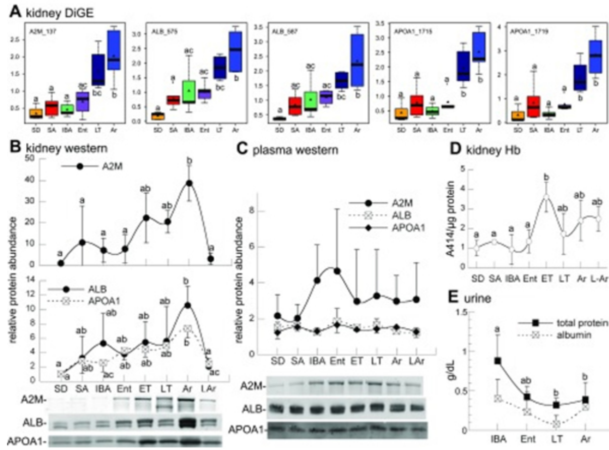



**Western Blot**
Western blot analysis of intestinal proteins in sham-treated hibernating (IBA) and SA ground squirrels. A, C, and E: representative immunoblots of apolipoprotein A-I (APOA1), albumin (ALB), and ubiquitin carboxyl-terminal hydrolase L1 (UCHL1) with bands at the indicated molecular size based on markers run on the same gel. For ALB and UCHL1, only the bands shown were detected; for APOA1, the band shown was the major band detected and the only one near the correct apparent molecular mass. B, D, and F: graphs plot mean and 1 SD obtained after quantifying protein bands in sham-treated summer and IBA animal groups. B: APOA1 (2.6-fold increased in IBA, n = 6; P = 0.001); D: ALB (2.7-fold increased in IBA, n = 5, P = 0.00046); F: UCHL1 (2.08-fold increased in IBA, n = 6, P = 0.003), Student's t-test. Fig 4. PMID: 18434441



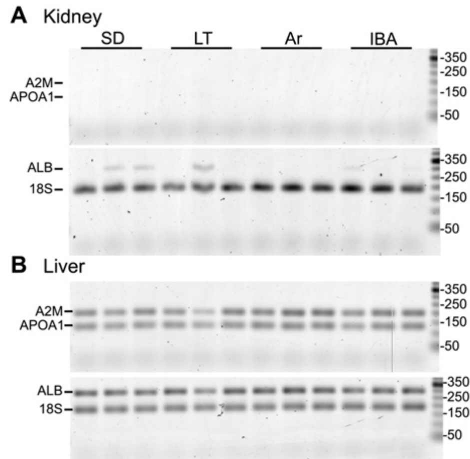

### ELISA

Mouse monoclonal anti-human apoA-I (MAb010-A/11) and anti apoA-I+32 (MOA-I) antibodies recognize different epitopes. Goat polyclonal anti human apoA-I (1:10,000 dilutions) was used as the capture antibody. ApoA-I+32 (400 ng/ml) was then added, followed by competitive ELISA as described in Materials and Methods and using biotinylated mAb17 (600 ng/ml) together with either MAb010-A/11 (squares), MOA-I (triangles) or control antibody (diamonds, anti-human smooth muscle actin) as competing antibody at the molar ratio indicated. The results show mean ± SEM of four separate experiments, each carried out in triplicates. Fig 2. PMID: 18832772



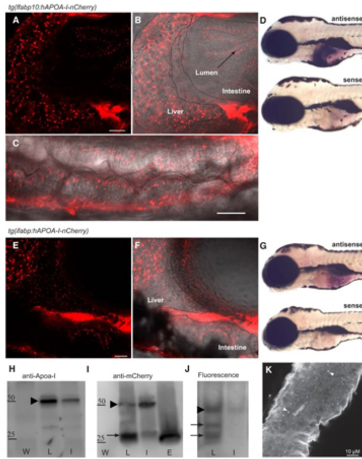

### Western Blot

Analysis of plasma proteins. Small letters on graphs distinguish significantly different groups (Tukey, $P < 0.05$). A: box plots of kidney protein spot intensities in 6 physiological stages determined by DiGE. Plots show mean (⍰), median (thick horizontal line), center 25th–75th percentile (colored boxes), 0 and 100th percentile (capped thin lines), and outliers (○) for each group of protein abundance measurements. B: Western blot images and corresponding quantitative analysis of A2M, ALB, and APOA1 in kidney, adding ET and LAr (Fig. 1). Western blots confirm that all 3 proteins differ among stages: A2M $P = 0.0013$; ALB $P = 0.03$; APOA1 $P = 0.0005$. C: no significant differences were detected for any of these 3 proteins in plasma. D: plot of relative hemoglobin (Hb) as measured by A414/µg total protein in kidney extracts. Kidney Hb varies ($P = 0.004$). E: urine total protein and ALB. Total protein varied ($P = 0.0047$), but no change was detected in ALB. B–D show means ± SD for n = 3; E is for n = 5 for IBA and LT, 3 for Ent, and 6 for Ar. Fig 5. PMID: 22643061



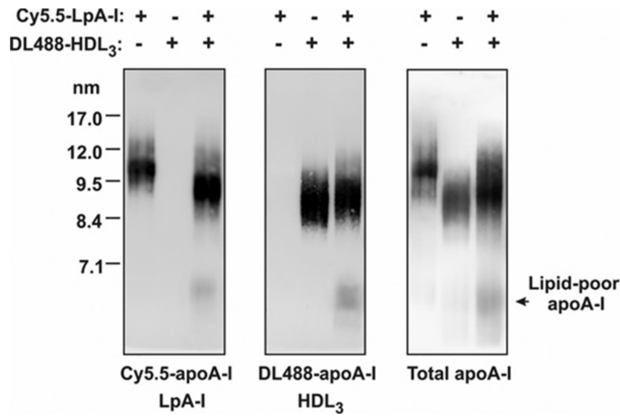

**Western Blot**

Expression of A2M, APOA1, and ALB occurs primarily in the liver. We assessed 4 RNAs for presence in kidney (A) and liver (B) from 12 ground squirrels (n = 3 in SD, LT, Ar, and IBA) by RT-PCR. Reactions were combined after the final PCR step into 1 gel lane/individual for A2M and APOA1, and for ALB and 18S. The expected sizes of PCR products are: A2M, 199 bp; APOA1, 134; ALB, 293 bp; 18S rRNA 187 bp. Marker sizes are indicated on the right in bp. Fig 6. PMID: 22643061



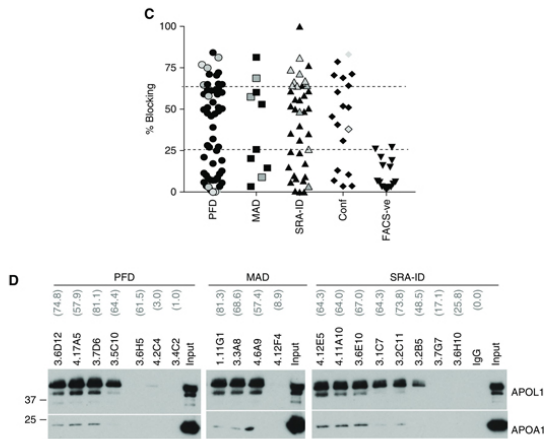

**Immunofluorescence Microscopy**

Transgenic zebrafish allows for visualization of human APOA-I in vivo. Live tg(lfabp10:hAPOA-I-mCherry) (A–C) and tg (ifabp:hAPOA-I-mCherry) (E–F) larvae show hAPOA-I-mCherry accumulation in the liver and intestine. hAPOA-I-mCherry derived from the liver (outlined by dotted line) accumulates in the intestine outlined by wavy line) and hAPOA-I-mCherry derived from the intestine accumulates in the liver (B and C). Tissue specificity of promoters used in APOA-I-mCherry transgenic fish revealed using whole mount in situ hybridization with antisense riboprobes to mCherry mRNA (D and G). No expression was detected with the sense probes (n = 10–12 fish); all larvae 6-dpf. ApoA-I-mCherry fusion protein is made in transgenic zebrafish and partially degraded (H). ApoA-I-mCherry fusion protein (arrow head) is made in transgenic zebrafish as expected size (50 kDa) (representative image from three experiments). mCherry antisera detected two different bands from Tg(lfabp:hApoA-I-mcherry) and Tg(ifabp:hApoA-I-mcherry) (I). The higher molecular weight bands (arrow head) represent ApoA-I-mCherry full-length protein. The lower molecular weight bands (arrow) is possibly a degradation product of similar size as mCherry protein made from Tg(ef1a:mcherry-CVLL) (representative image from two experiments). mCherry flourscence on native gel (representative image from four experiments) (J). Immunofluorescence for endogenous zApoA-Ia and zApoA-Ib in wild-type fish show punctae within the intestine similar to hAPOA-I-mCherry accumulation (representative image from three experiments) (K). APOA-I, apolipoprotein A-I; dpf, days postfertilization; E, Tg (ef1a:mcherry-CVLL); hAPOA-I, human APOA-I; I, Tg (ifabp:hApoA-I-mcherry); L, Tg(lfabp:hApoA-I-mcherry); W, wild-type; zAPOA-1, zebrafish APOA-1. Fig 4.
PMID: 30629468



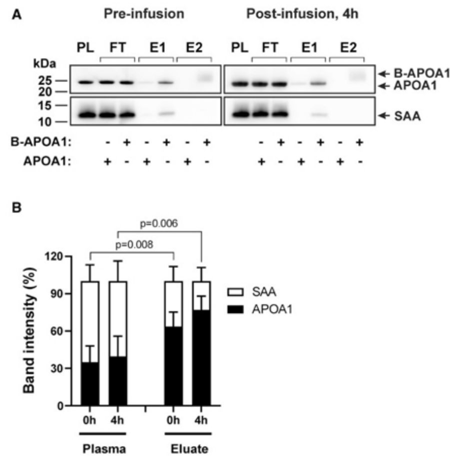

**Western Blot**

Lipoprotein particle remodeling is induced by the interaction of nascent LpA-I (apo AI [apolipoprotein AI] containing particles formed by incubating ABCA1 [ATP-binding cassette transporter 1]-expressing cells with apo AI) and HDL3 in vitro. Fluorescently labeled LpA-I (Cy5.5-LpA-I) and HDL3 (DL488-HDL3) were incubated for 1 h at 37°C, final concentration of each lipoprotein was 0.5 mg/mL. Lipoproteins were subjected to nondenaturing polyacrylamide gradient gel electrophoresis. Cy5.5–apo AI (left) and DL488–apo AI (middle) were visualized by fluorescent imaging, apo AI (right) was detected by anti–apo AI antibody. The migration of lipid-poor apo AI is also indicated. HDL indicates high-density lipoprotein. Fig 3. PMID: 32131613



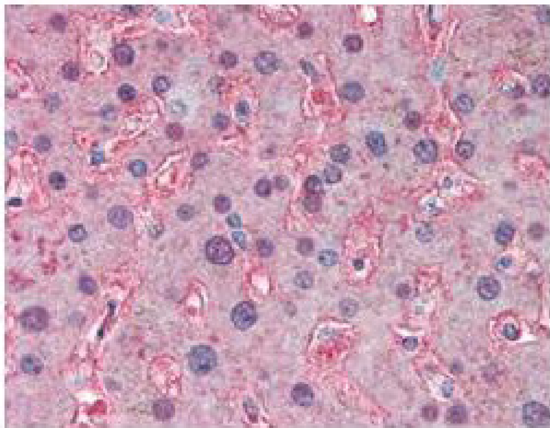

**Western Blot**

(C) Blocking of serum-associated APOL1-mediated trypanolytic activity in the presence of 1 µg/ml anti-APOL1 antibodies is plotted. Antibodies are grouped according to their domain recognition as determined in Figure 1. The percentage of blocking is calculated by normalizing to no antibody control (NHS only). Each symbol represents the average of two independent experiments for a given antibody. Antibodies used in the immunoprecipitation assay in (D) and Supplemental Figure 8 are plotted in gray. Note that all experiments were performed with 1 and 10 µg/ml antibodies in parallel, but only the 1 µg/ml data are shown due to higher stringency. The dashed line at 25% indicates the cut-off limit for blockers; the line at 60% indicates the cut-off for “strong” blockers.(D) Immunoprecipitation of APOL1 from NHS. A subset of the cloned anti-APOL1 antibodies to each domain [selected for a range of blocking activities, see (C)] were conjugated to Dynabeads and incubated with NHS. Immunoprecipitates were Western blotted with rabbit anti-APOL1 (Proteintech; top) or anti-APOA1 (Rockland Immunochemicals; bottom) on 4%–12% Bis-Tris gels. The strong blockers pulled down large quantities of APOL1, the weak blockers little or no APOL1, and the nonblockers no APOL1. Gray numbers in brackets indicate percentage blocking activity as plotted in (C). Because APOL1 is only present in HDL3b,32 which comprises only approximately 5% HDL particles,33 only a fraction of total APOA1 is coimmunoprecipitated by the best anti-APOL1 antibodies, as expected. Another experiment including some of the other antibodies, as well as HPR pull-down, is shown in Supplemental Figure 8. Fig 6.
PMID: 32764138



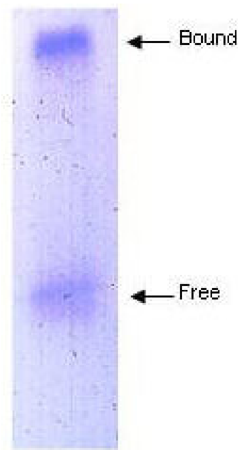

**Western Blot**

CSL112 infusion lowers SAA (serum amyloid A) content of HDL (high-density lipoprotein) particles involved in APOA1 (apolipoprotein A1) exchange. A, Patient plasma collected at pre-infusion or at 4 hours post-infusion with 6 g CSL112 was incubated with B-APOA1 (biotinylated APOA1) or unlabelled APOA1 as indicated. After incubation at 37 °C for 30 minutes and subsequent treatment with streptavidin sepharose, protein samples collected during the capture process were analyzed by SDS-PAAGE and Western blotting with an anti-APOA1 and anti-SAA1/2 antibodies. Untreated plasma (PL); flow through (FT); elution 1 with reducing LDS sample buffer for 5 minutes at 65 °C (E1); elution 2 with reducing LDS sample buffer supplemented with 5% SDS and 1 mM biotin for 5 minutes at 95 °C. The positions of migration of APOA1, SAA (serum amyloid A), and B-APOA1 and molecular weight standards are indicated. B, Band intensities of APOA1 and SAA (in untreated plasma and eluate (elution I) were semiquantified by densitometry. The intensities for APOA1 and SAA are shown as percentages of the summed intensity for corresponding 2 bands. Data values represented as the mean±SD are derived from the analysis of 5 patients infused with 6g CSL112 (shown in Figure S3; Table S5), P=0.008; P=0.006, vs corresponding plasma control by 1-way ANOVA followed by Dunnett post hoc test.
Figure 5. PMID: 36994730



**Immunohistochemistry**

Rockland's anti-APOA1 antibody was used at a 5 ug/ml to detect signal in human liver tissue. Tissue was formalin-fixed and paraffin embedded. Personal Communication, Tina Roush, LifeSpanBiosciences, Seattle, WA.




**SDS-PAGE**
Coomassie stained gel showing both free and HDL bound apoA-I eluted from a solid phase resin prepared using Rockland's anti-Human apoLipoprotein A-I antibody. The resin was reacted with human serum prior to washing and elution of bound proteins. The gel was composed of 0.75% agarose in a native buffer system. Separation occurred at room temperature.

## References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