Product Datasheet

ABCA1 Antibody - BSA Free
NB400-105

Unit Size: 0.1 ml

Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.

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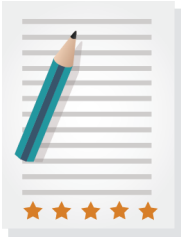
Updated 2/17/2025 v.20.1



NB400-105

ABCA1 Antibody - BSA Free

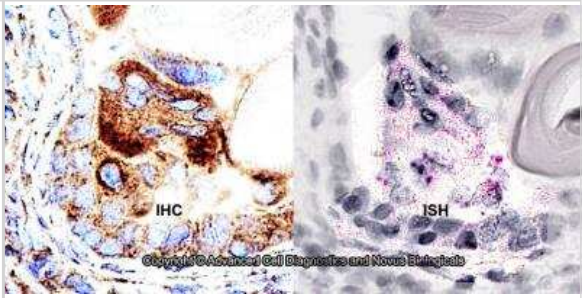
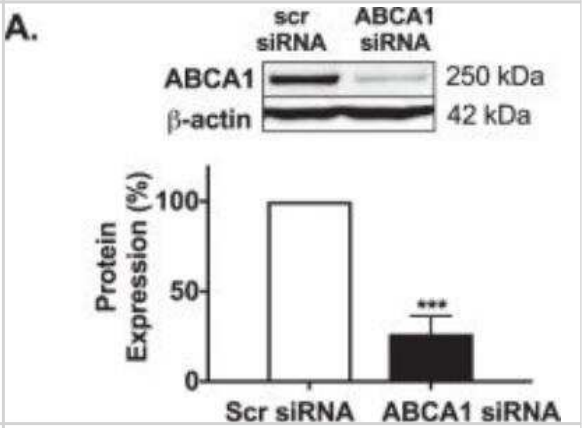
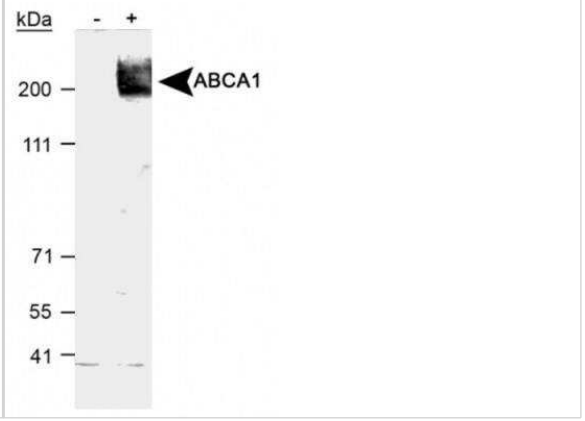
Product Information	
Unit Size	0.1 ml
Concentration	1 mg/ml
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	PBS
Target Molecular Weight	220 kDa

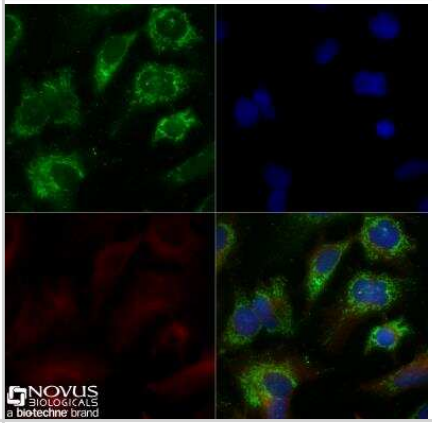
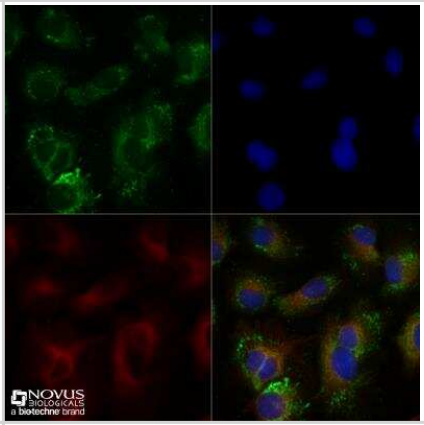
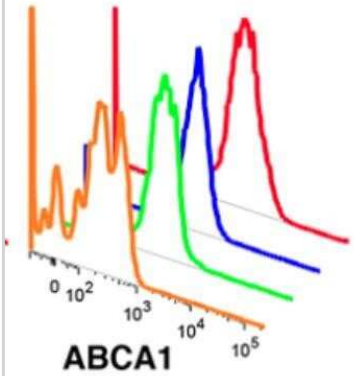
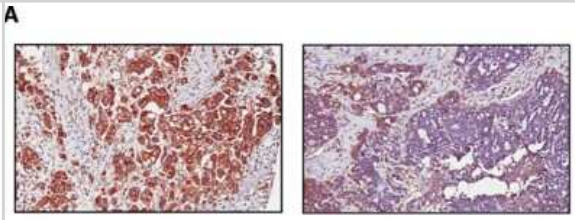
Product Description	
Host	Rabbit
Gene ID	19
Gene Symbol	ABCA1
Species	Human, Mouse, Rat, Porcine, Canine, Chicken, Chinese Hamster, Equine, Hamster, Mustelid, Primate, Rabbit
Reactivity Notes	Canine reactivity reported in scientific literature (PMID: 24612239). Rabbit reactivity reported in scientific literature (PMID: 26444796). Chinese Hamster reactivity reported in scientific literature (PMID: 27902765). Primate reactivity reported in scientific literature (PMID: 25440061). Equine reactivity reported in scientific literature (PMID: 26711702).
Immunogen	Partial peptide sequence (within residues 1100-1300) of human ABCA1 Antibody [UniProt# O95477]. Actual immunogen sequence is proprietary information.

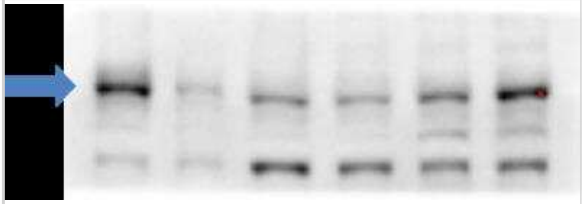
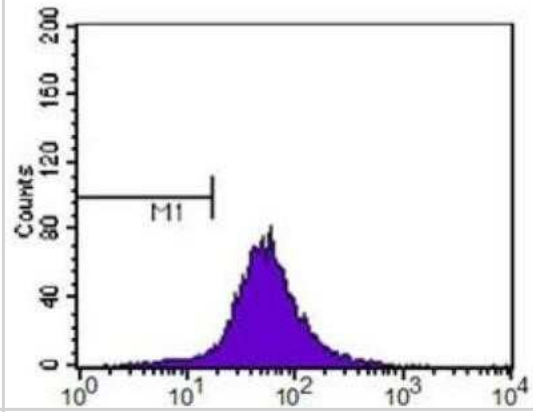
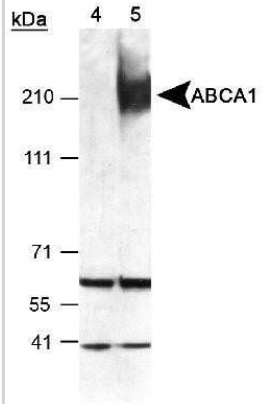
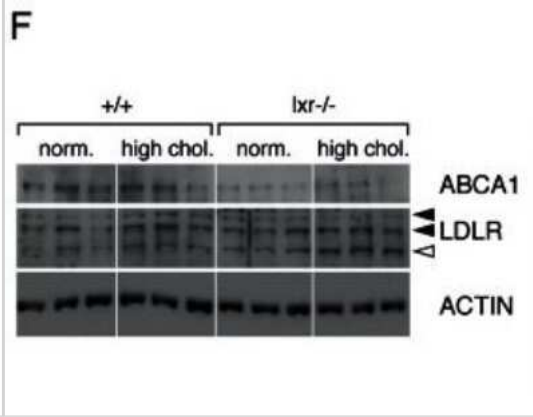
Product Application Details	
Applications	Western Blot, Simple Western, Chromatin Immunoprecipitation, ELISA, Flow Cytometry, Gel Super Shift Assays, Immunoblotting, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry, Immunohistochemistry-Frozen, Immunohistochemistry-Paraffin, Immunoprecipitation, Block/Neutralize, Chromatin Immunoprecipitation (ChIP), Dual RNAscope ISH-IHC, Gel Supershift Assay, Knockdown Validated, Knockout Validated, PCR
Recommended Dilutions	Western Blot 1:500, Simple Western 1:50, Chromatin Immunoprecipitation reported in scientific literature (PMID 19515742), Flow Cytometry 1:400. Use reported in scientific literature (PMID 21501868), ELISA reported in scientific literature (PMID 18541924), Immunohistochemistry 1:200, Immunocytochemistry/ Immunofluorescence 1:100. Use reported in scientific literature (PMID 21501868), Immunoprecipitation 1:10-1:500. Use reported in scientific literature (PMID 21846716; 21106520), Immunohistochemistry-Paraffin 1:200, Immunohistochemistry-Frozen, Immunoblotting reported in scientific literature (PMID 27599291), Gel Super Shift Assays reported in scientific literature (PMID 15684432), Gel Supershift Assay, Chromatin Immunoprecipitation (ChIP), Knockout Validated reported in scientific literature (PMID 32273567), Knockdown Validated reported in scientific literature (PMID 31666189), Block/Neutralize reported in scientific literature (PMID 30821416), PCR reported in scientific literature (PMID 27406916), Dual RNAscope ISH-IHC

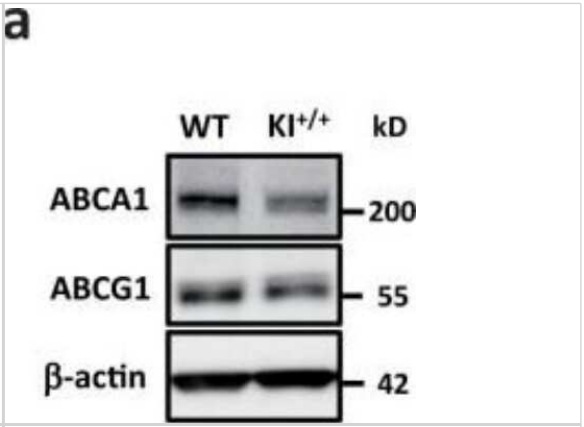
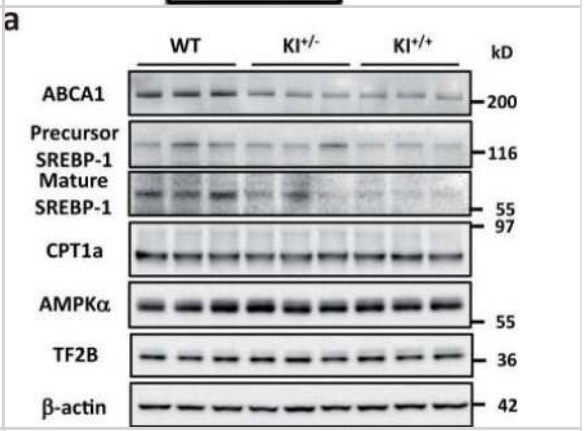
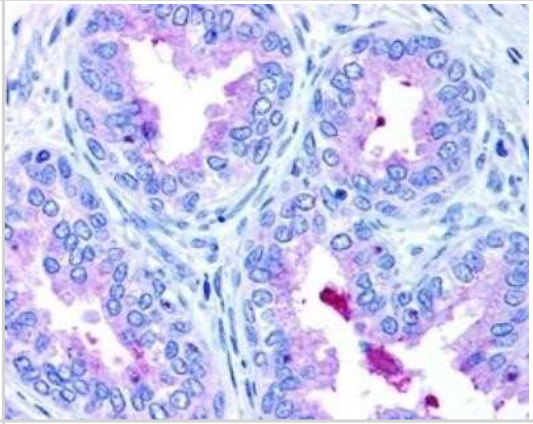
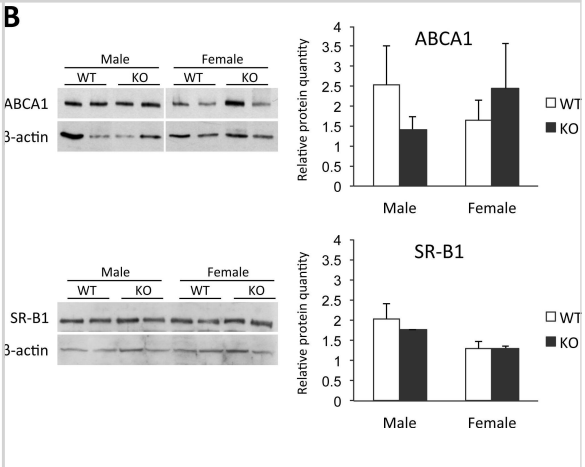
Application Notes	Western Blot band representing ABCA1 is observed at approx. 220 kDa. Additional non-specific bands are seen at lower molecular weights, but do not interfere with the ABCA1 signal. In Simple Western only 10-15 uL of the recommended dilution is used per data point. See Simple Western Antibody Database for Simple Western validation: separated by Charge, antibody dilution of 1:50Separated by Charge.

Images

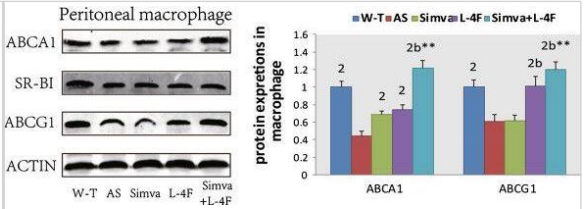
Formalin-fixed paraffin-embedded tissue sections of human prostate cancer were probed for ABCA1 mRNA (ACD RNAScope Probe, catalog # 432291; Fast Red chromogen, ACD catalog # 322360). Adjacent tissue section was processed for immunohistochemistry using rabbit polyclonal (Novus Biologicals catalog # NB400-105) at 1.5ug/mL with overnight incubation at 4 degrees Celsius followed by incubation with anti-rabbit IgG VisUCyte HRP Polymer Antibody (Catalog # VC003) and DAB chromogen (yellow-brown). Tissue was counterstained with hematoxylin (blue). Specific staining was localized to glandular cells.	
HSKMCs were transfected with scrambled siRNA (scr siRNA) or ABCA1 siRNA. The cells were lysed and ABCA1 (A) protein levels were quantified by immunoblotting. Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/s41598-018-38014-3) licensed under under a CC-BY license.	
Analysis of ABCA1 in total cell lysates of RAW264.9 cells treated with vehicle (-) or 9-cisretinoic acid and 22Rhydroxycholesterol (+). Samples used for this testing were 40 ug of total cell post-nuclear lysate from each group.	

HepG2 cells were grown to 60% confluency, serum starved for 24 hours, and then treated with 1uM TO9 for 24 hours prior to being fixed for 10 minutes using 10% formalin and then permeabilized for 5 minutes using 1X TBS + 0.5% Triton-X100. The cells were incubated with anti-ABCA1 at 5.0ug/ml overnight at 4C and detected with an anti-rabbit Dylight 488 (Green) at a 1:500 dilution. Alpha tubulin (DM1A) NB100-690 was used as a co-stain at a 1:1000 dilution and detected with an anti-mouse Dylight 550 (Red) at a 1:500 dilution. Nuclei were counterstained with DAPI (Blue). Cells were imaged using a 40X objective.	
Untreated HepG2 cells were grown to 60% confluency, and serum starved for 24 hours prior to being fixed for 10 minutes using 10% formalin and then permeabilized for 5 minutes using 1X TBS + 0.5% Triton-X100. The cells were incubated with anti-ABCA1 at 5.0ug/ml overnight at 4C and detected with an anti-rabbit Dylight 488 (Green) at a 1:500 dilution. Alpha tubulin (DM1A) NB100-690 was used as a co-stain at a 1:1000 dilution and detected with an anti-mouse Dylight 550 (Red) at a 1:500 dilution. Nuclei were counterstained with DAPI (Blue). Cells were imaged using a 40X objective.	
ABCA-1 FITC conjugated antibody of human adipose tissue macrophage subsets by flow cytometry. Image from verified customer review.	
Association between expression of ABCA1 and survival in ovarian cancer patients. Expression of ABCA1 in 55 ovarian cancer patient samples was determined by IHC in tissue microarray. Representative image of ovarian cancer showing high (left panel) and low (right panel) ABCA1 expression on the cell membrane or cytoplasm (x400). Image collected and cropped by CiteAb from the following publication (https://www.clinicalepigeneticsjournal.com/content/7/1/1), licensed under a CC-BY license.	

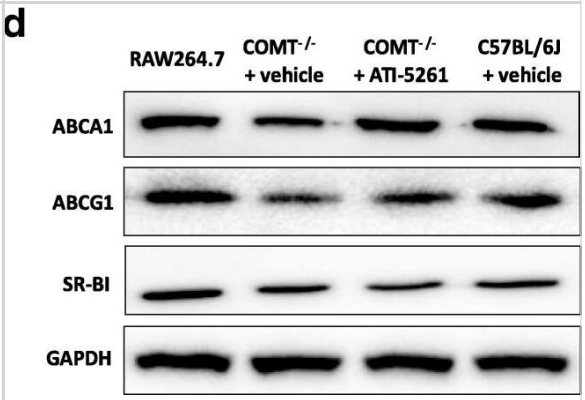
ABCA1 in human primary fibroblasts in culture. 10 ug of total protein. 7.5% TGX gel. Blocking 5% milk in PBST 1h RT. 1:1000 primary ab in BSA 3% PBST O/N at 4C. Secondary 1:5000 HRP 1h RT. Arrow shows around 250 kDa. WB image submitted by a verified customer review.	
ABCA1 antibody was tested at 1: 400 in HeLa cells using an Alexa Fluor 488 secondary (shown in purple). M1 is defined by unstained cells.	
Detection of ABCA1 in mouse peritoneal macrophages using NB 400-105 (Lot L). ECL exposure, 1 min. Lane 4: T09 uninduced lysate Lane 5: T09 induced lysate.	
Prostates of LXR mutant mice accumulate cholesterol esters through inappropriate LXR target genes regulation. Total protein lysates of WT and LXR null mice under normal or high cholesterol diet were analyzed by western blotting with antibodies against ABCA1, LDLR and ACTIN as a loading control (left panel), quantification of ABCA1 and LDLR protein accumulation levels (right panel). * p<0.05, *** p<0.001 in Student's t test. Error bars represent the +/- mean SEM. Image collected and cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pgen.1003483), licensed under a CC-BY license.	

miR-33b reduces cellular cholesterol efflux and serum HDL-C levels. Western blotting for ABCA1 (using NB400-105) and ABCG1 proteins in peritoneal macrophages from WT and KI+/+ mice. Representative images are shown. beta-actin was used as the loading control. Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/srep05312), licensed under a CC-BY license.	
miR-33b regulates ABCA1 and SREBP-1. Analysis for ABCA1, SREBP-1, CPT1a, and AMPK alpha protein levels in the livers of WT, KI+/-, and KI+/+ mice. Representative images are shown. TF2B and beta-actin were used as loading controls. Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/srep05312), licensed under a CC-BY license.	
Detection of ABCA1 in human prostate epithelium showing luminal and membrane staining.	
Liver mRNA & protein expression analysis of chow-fed Osbpl8KO mice.A: qPCR analysis of the quantity of the mRNAs identified at the bottom in chow-fed KO females (open bars) & males (closed bars). The mRNAs were quantified using ribosomal protein 36B4 message as a housekeeping reference. The data are expressed relative to quantity in littermate WT animals of the same gender, & represent mean ± s.e.m. (n =6; *p<0.05, **p<0.01, T-test). B: Western blot analysis of ABCA1 & SR-B1 proteins in WT & KO mouse liver. The blots were probed with anti-β-actin as a loading control. Densitometric quantification of the Western blot data is shown on the right. The results were normalized against β-actin. The data represents mean ± s.e.m. (n=4). Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0058856), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

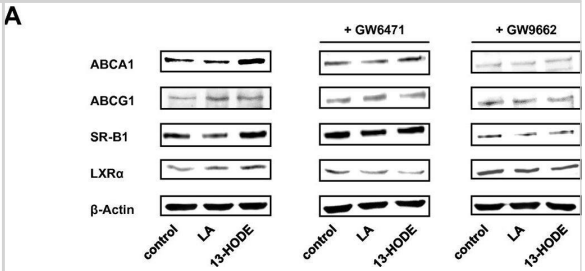
Protein expressions of ABCA1, SR-BI & ABCG1 in liver & macrophage by Western blot. Simvastatin increased the expressions of ABCA1 & ABCG1 in liver & ABCA1 in macrophage, Both L-4F & the combination group improved the expressions of ABCA1, SR-BI & ABCG1 in liver & ABCA1 & ABCG1 in macrophage. 1P < 0.05, 2P < 0.001, vs. AS group; aP < 0.05, bP < 0.001, vs. Simva group;*P < 0.05, **P < 0.001, vs. L-4F group. Image collected & cropped by CiteAb from the following publication (https://lipidworld.biomedcentral.com/articles/10.1186/1476-511X-12-180), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



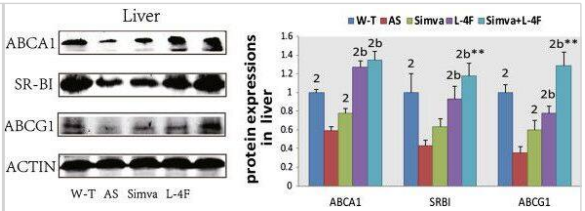
ABCA1, ABCG1 & SR-BI protein expressions. a & b Hepatic protein expressions of ABCA1, ABCG1 & SR-BI were significantly decreased in COMT−/− mice at GD 18.5, compared to C57BL/6 J mice. Decreased hepatic ABCA1 expression was also observed at 10 days postpartum. ATI-5261 increased ABCA1 & ABCG1 expression in the liver at 10 days postpartum. c Placental protein expressions of ABCA1 & ABCG1 was reduced in COMT−/− mice, compared to C57BL/6 J mice. ATI-5261 treatment significantly increased ABCA1 levels in the placenta of COMT−/− mice. d Representative immunoblots of the corresponding proteins in the placenta with mouse RAW264.7 cell lysate included as positive control. Similar results were obtained when the experiment was repeated with lysates prepared from three batches of tissues. Data are presented as mean ± SEM. Groups (n = 8 in all groups) were compared using one-way ANOVA with post-hoc analysis (Tukey's procedure). *, p < 0.05 Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/30237900), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



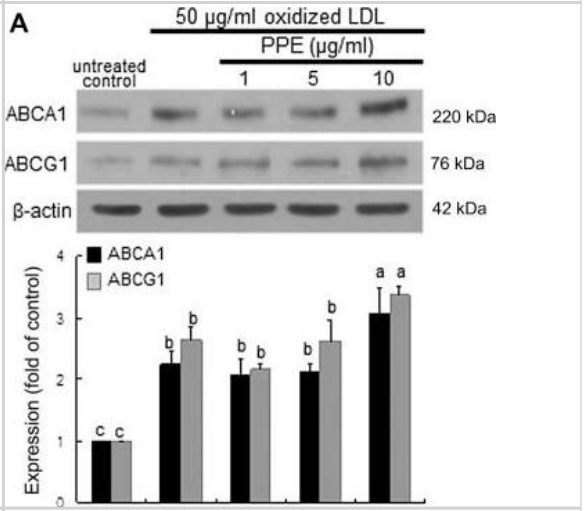
Effects of 13-HODE & LA in the presence & absence of PPARα & PPARγ selective antagonists on molecular markers of cholesterol homeostasis in RAW264.7 macrophages. RAW264.7 cells were pre-treated without or with the PPARα selective antagonist GW6471 or the PPARγ selective antagonist GW9662 & subsequently treated without (vehicle control) or with 2.5 μmol/L 13-HODE or 100 μmol/L LA for 24 h. Afterwards, cells were lysed & subsequently processed for western blotting as described in the materials & methods section. A, Representative immunoblots specific for ABCA1, ABCG1, SR-BI, LXRα, & β-actin which was used for normalization are shown. B, Bars represent data from densitometric analysis & are means ± SD from three independent experiments (n = 3). Data are expressed as percentage of protein concentration of vehicle control cells. Results from statistical analysis are indicated: Significant effects are denoted with superscript letters. Bars marked without a common superscript letter significantly differ (P < 0.05). Image collected & cropped by CiteAb from the following publication (https://lipidworld.biomedcentral.com/articles/10.1186/1476-511X-10-222), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



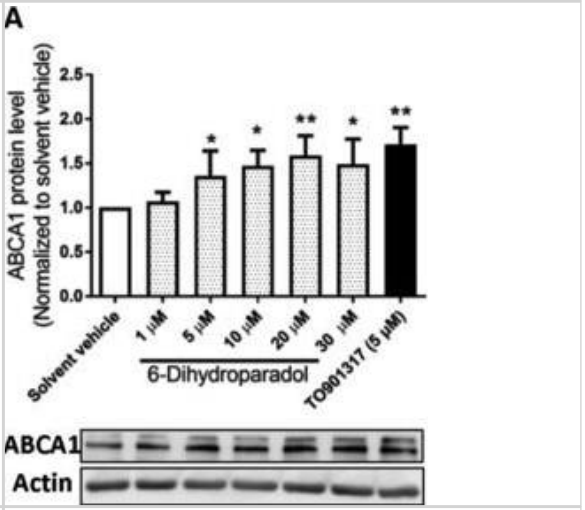
Protein expressions of ABCA1, SR-BI & ABCG1 in liver & macrophage by Western blot. Simvastatin increased the expressions of ABCA1 & ABCG1 in liver & ABCA1 in macrophage, Both L-4F & the combination group improved the expressions of ABCA1, SR-BI & ABCG1 in liver & ABCA1 & ABCG1 in macrophage. 1P < 0.05, 2P < 0.001, vs. AS group; aP < 0.05, bP < 0.001, vs. Simva group;*P < 0.05, **P < 0.001, vs. L-4F group. Image collected & cropped by CiteAb from the following publication (https://lipidworld.biomedcentral.com/articles/10.1186/1476-511X-12-180), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



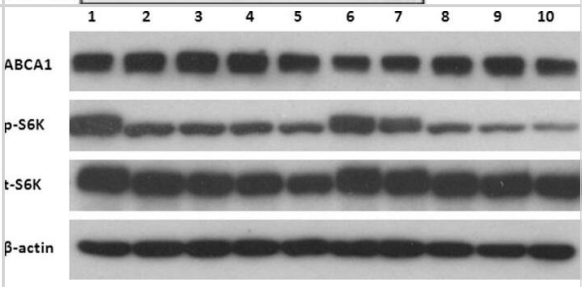
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Upregulation of ABCA1 & ABCG1 & (B) enhancement of cholesterol efflux by purple Perilla frutescens extracts (PPE) in 50 µg/ml Cu2+-oxidized low-density lipoproteins (LDL)-exposed J774A.1 murine macrophages. (A) For the measurement of ABCA1 & ABCG1 expression, total cell lysates were subjected to western blot analysis with a primary antibody against ABCA1 or ABCG1. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. (B) Cholesterol efflux was expressed as the percentage of fluorescence in the medium relative to the total fluorescence. Bar graphs denoted without a common letter indicate significant difference, P<0.05. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



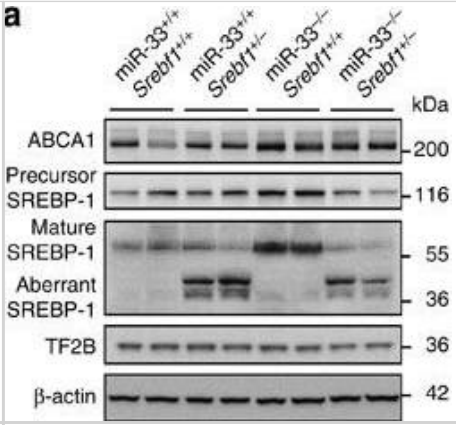
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - 6□Dihydroparadol increases A) ABCA1 & B) ABCG1, but not C) SR□BI protein levels in cholesterol□loaded THP□1□derived macrophages. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with increasing concentrations of 6□dihydroparadol (1–30 µm) for another 24 h. The protein levels of ABCA1, ABCG1, & SR□B1 were detected by Western blot analysis. The control was treated with solvent vehicle (0.1% DMSO). As a positive control, TO901317 (5 µm, 24 h) was used. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



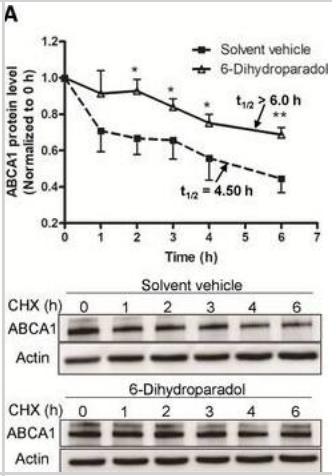
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Akt inhibition suppresses mTORC1 activity.BHK-ABCA1 cells were induced overnight with mifepristone (10 nM) & then incubated with DEBC, LY294002 or rapamycin at indicated concentrations for 2 h. Cells were then lysed & analyzed for ABCA1 & phosphorylated S6K by Western blotting. Actin was also blotted as loading control. Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0113789), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



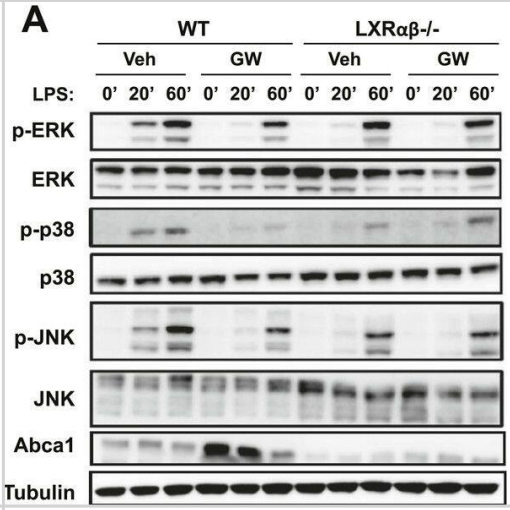
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Reversal of hepatic steatosis by reduction of SREBP-1 levels.(a) Western blotting analysis of SREBP-1 & ABCA1 levels in livers of miR-33+/+Srebf1+/+, miR-33+/+Srebf1+/−, miR-33−/−Srebf1+/+ & miR-33−/−Srebf1+/− mice. Representative western blot images are shown (n=4). Image collected & cropped by CiteAb from following publication (https://pubmed.ncbi.nlm.nih.gov/24300912), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



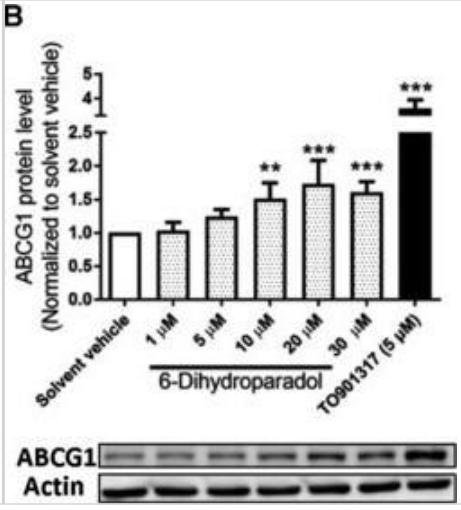
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - 6□Dihydroparadol increases A) ABCA1 & B) ABCG1 protein stability. THP□1 cells were differentiated as described in Figure 2. Then cells were loaded with unlabeled cholesterol & treated with 6□dihydroparadol (10 μm), or solvent vehicle (0.1% DMSO, control) for 24 h. Cells were lysed at different time points (0, 1, 2, 3, 4, & 6 h) after treatment with the protein synthesis inhibitor cycloheximide (CHX, 100 μm). The protein levels of both ABCA1 & ABCG1 were detected by Western blot analysis. The data points represent mean ± SD from three independent experiments. *p < 0.05 & **p < 0.01 versus control at the same time point (determined by Student's t□test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

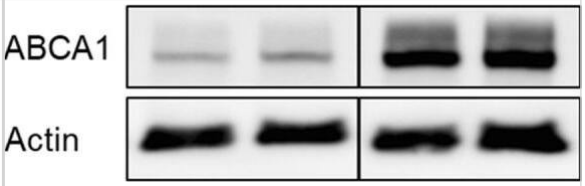
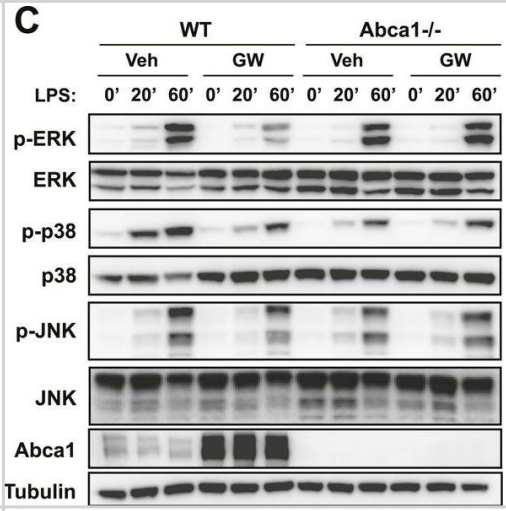
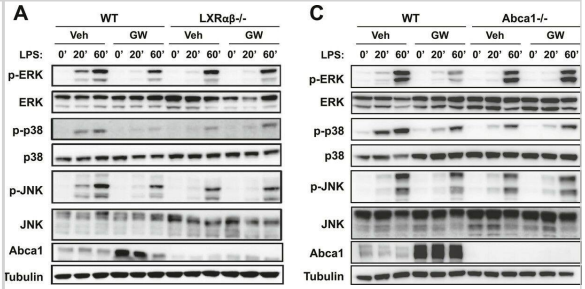


Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Ligand activation of LXR inhibits LPS-induced MAP kinase activation through Abca1 induction.(A–D) Bone marrow-derived macrophages from Lxrα−/−Lxrβ−/− & control wild-type mice (A, B), or bone marrow-derived macrophages from myeloid-specific Abca1−/− & control wild-type mice (C, D) were pretreated with GW3965 (1 μM) overnight, followed by stimulation with LPS (10 ng/ml) for 20 min or 1 hr. Whole cell lysates were harvested & protein expression was analyzed by immunoblotting with the indicated antibodies (A, C). Protein expression was quantified by Image Quant TL7.0 (B, D). N = 4–6 per group. *p < 0.05, **p < 0.01, NS, not significant. Error bars represent means ± SEM.DOI:http://dx.doi.org/10.7554/eLife.08009.012 Image collected & cropped by CiteAb from the following publication (https://elifesciences.org/articles/08009), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

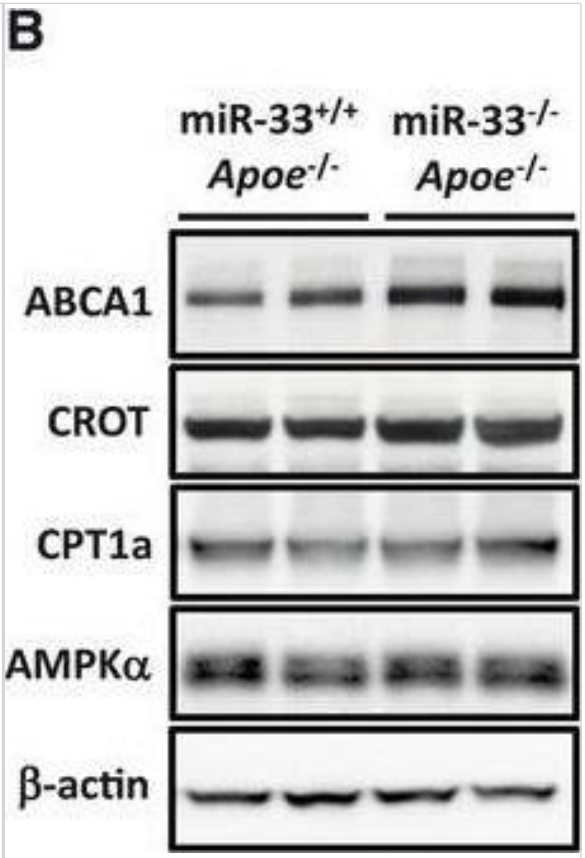


Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - 6□Dihydroparadol increases A) ABCA1 & B) ABCG1, but not C) SR□BI protein levels in cholesterol□loaded THP□1□derived macrophages. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with increasing concentrations of 6□dihydroparadol (1–30 μm) for another 24 h. The protein levels of ABCA1, ABCG1, & SR□B1 were detected by Western blot analysis. The control was treated with solvent vehicle (0.1% DMSO). As a positive control, TO901317 (5 μm, 24 h) was used. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

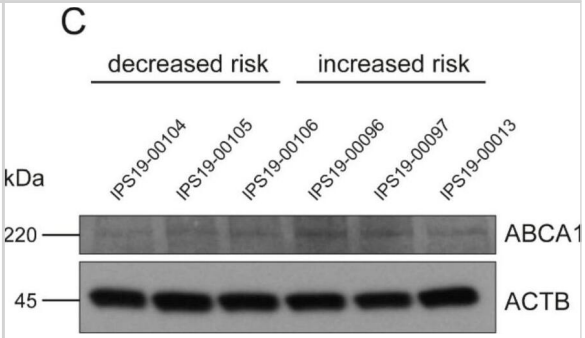


Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Western Blotting for ABCA1, as detailed in Supplementary Protocol #5 (Additional file 1). Caco-2 cells cultivated on filter inserts were treated for 48 h with bexarotene (RXR agonist, 5 μM), T0901317 (LXR agonist, 10 μM), GW3965 (LXR agonist, 10 μM) or the vehicle control (DMSO 0.1%). Protein expression was then determined by western blot analysis. Results were normalized for interexperimental variance & then set in relation to the vehicle control. Bar graphs represent mean ± SD from three independent experiments. A representative blot is shown - the bands stem from the same membrane & the vertical line indicates excised irrelevant bands. ns not significant, *P < 0.05, **P < 0.01 versus solvent vehicle (determined by paired t-test) Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/32308567), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Ligand activation of LXR inhibits LPS-induced MAP kinase activation through Abca1 induction.(A–D) Bone marrow-derived macrophages from Lxrα−/−Lxrβ−/− & control wild-type mice (A, B), or bone marrow-derived macrophages from myeloid-specific Abca1−/− & control wild-type mice (C, D) were pretreated with GW3965 (1 μM) overnight, followed by stimulation with LPS (10 ng/ml) for 20 min or 1 hr. Whole cell lysates were harvested & protein expression was analyzed by immunoblotting with the indicated antibodies (A, C). Protein expression was quantified by Image Quant TL7.0 (B, D). N = 4–6 per group. *p < 0.05, **p < 0.01, NS, not significant. Error bars represent means ± SEM.DOI:http://dx.doi.org/10.7554/eLife.08009.012 Image collected & cropped by CiteAb from the following publication (https://elifesciences.org/articles/08009), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Ligand activation of LXR inhibits LPS-induced MAP kinase activation through Abca1 induction.(A–D) Bone marrow-derived macrophages from Lxrα−/−Lxrβ−/− & control wild-type mice (A, B), or bone marrow-derived macrophages from myeloid-specific Abca1−/− & control wild-type mice (C, D) were pretreated with GW3965 (1 μM) overnight, followed by stimulation with LPS (10 ng/ml) for 20 min or 1 hr. Whole cell lysates were harvested & protein expression was analyzed by immunoblotting with the indicated antibodies (A, C). Protein expression was quantified by Image Quant TL7.0 (B, D). N = 4–6 per group. *p < 0.05, **p < 0.01, NS, not significant. Error bars represent means ± SEM.DOI:http://dx.doi.org/10.7554/eLife.08009.012 Image collected & cropped by CiteAb from the following publication (https://elifesciences.org/articles/08009), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

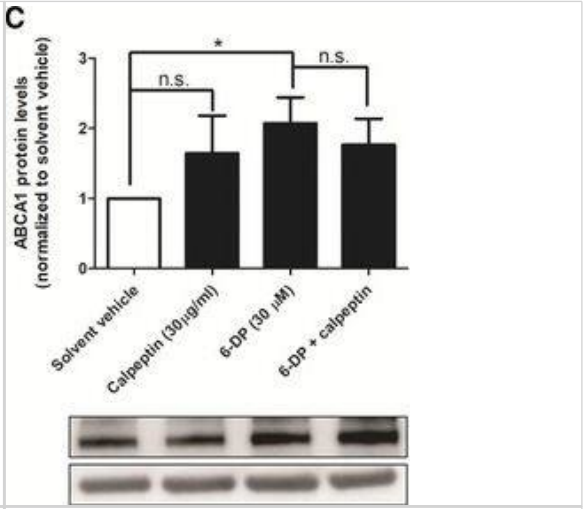
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Expression of ABCA1 & CROT in livers & RIP140 in macrophages is elevated in miR-33−/−Apoe−/− mice compared with miR-33+/+Apoe−/− mice. A, Quantitative real-time PCR analysis of Abca1, Crot, Cpt1a, & Prkaa1in livers from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values from miR-33+/+Apoe−/− mice were set at 100%. Values are mean±SE (n=9 to 11each); **P<0.01. B, Western analysis of ABCA1, CROT, CPT1a, & AMPKα in livers from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. β-actin was used as a loading control. C, Densitometry of ABCA1, CROT, CPT1a, & AMPKα in livers from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values from miR-33+/+Apoe−/− mice were set at 100%. Values are mean±SE (n=4 each); *P<0.05. D, Total cholesterol, free cholesterol, cholesterol ester, & triglyceride levels in livers of miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values are mean±SE (n=9 to 11 each). E, HE staining of livers of miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice at age 20 weeks fed NC. Scale bar: 100 μm. F, Quantitative real-time PCR analysis of Nrip1 (RIP140) in peritoneal macrophages from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values from miR-33+/+Apoe−/− mice were set at 100%. Values are mean±SE (n=7 each). G, Western analysis of NRIP1 (RIP140) in peritoneal macrophages from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. GAPDH was used as a loading control. H, Densitometry of NRIP1 (RIP140) in peritoneal macrophages from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values from miR-33+/+Apoe−/− mice were set at 100%. Values are mean±SE (n=4 each); *P<0.05. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/23316322), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



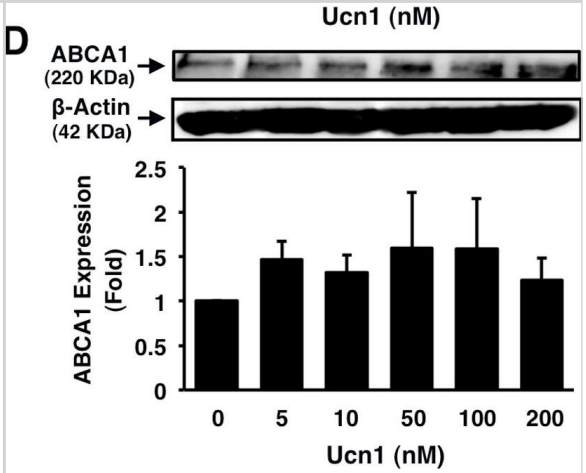
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Basal ABCA1 expression & function in patient-derived iRPEs. (A) Representation of patient-derived iPSC lines & genotypes harboring polymorphisms in ABCA1 that are associated with decreased or increased risk for AMD development. (B) Relative expression of ABCA1 mRNA in patient-derived iRPEs under basal conditions normalized to RPL28 & decreased risk group. (C) Western blot analysis of ABCA1 levels in patient-derived iRPEs under basal conditions. Actin was detected as loading control. ABCA1 expression was quantified & normalized to actin. (D,E) Relative expression of ABCG1 (D) & NR1H3 (E) mRNA in patient-derived iRPEs under basal conditions normalized to RPL28 & decreased risk group. (F) Cholesterol efflux in patient-derived iRPEs after direct cell labeling & in the presence of ApoAI. (G) Cholesterol efflux in patient-derived iRPEs after phagocytosis of BODIPY-cholesterol-loaded POSs & in the presence of ApoAI. (H) Quantification of phagocytosed POSs per nuclei in patient-derived iRPEs. Values shown are means ± SD (n = 3). * p < 0.05; *** p < 0.001. Unpaired Student's t-test. (I) Representative fluorescence microscopy images of phagocytosed FITC-labeled POSs (green) & staining for ZO-1 (red) & DAPI (blue) of increased risk cell line IPS19-00096. Scale bar = 100 μm. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/35328615), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



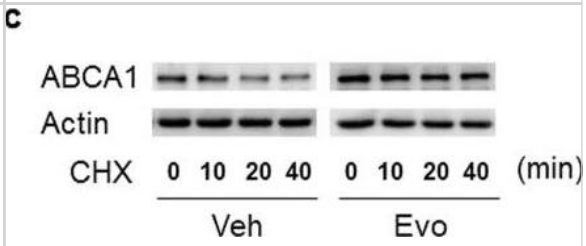
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - A–C) Effects of 6□dihydroparadol & specific proteasomal, lysosomal, & calpain inhibitors on the ABCA1 protein levels. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with or without 6□dihydroparadol (6□DP, 30 μm) for 24 h & incubated for another 3 h with or without the proteasome inhibitor lactacystin (10 μm), the lysosomal inhibitor chloroquine (100 μm), or the calpain inhibitor calpeptin (30 μg mL−1). The ABCA1 protein levels were detected by Western blot analysis. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control; n.s., not significant versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



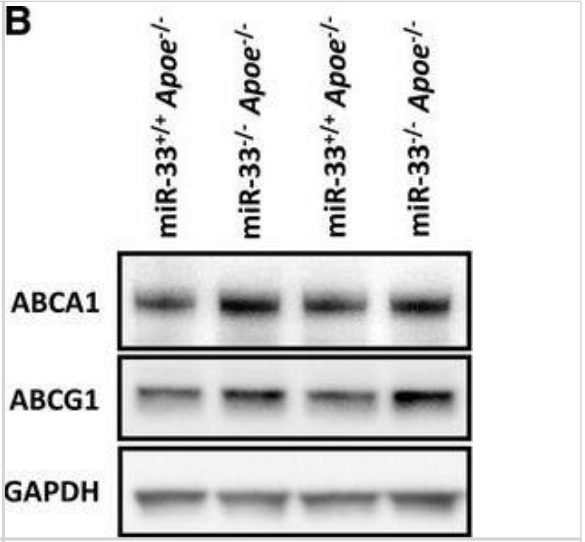
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Suppressive effects of Ucn1 on foam cell formation & related protein expression in human monocyte-derived macrophages.Human monocytes were incubated for 7 days with RPMI-1640 supplemented with 10% human serum & the indicated concentrations of Ucn1, followed by a 19 h-incubation with 50 μg/ml oxLDL in the presence of 0.1 mmol/l [3H]oleate. Intracellular CE accumulation was determined from the radioactivity of cholesterol-[3H]oleate. Otherwise, before the addition of oxLDL, cells were harvested & subjected to immunoblotting analyses for CD36, ACAT1, or ABCA1. β-Actin served as a loading control. Data are expressed as means ± SEM from 4–6 independent experiments with monocytes from 4–6 different donors. Baseline (1 fold)=8.4±2.2 nmol/mg cell protein. *P<0.05, †P<0.001 vs. 0 nmol/l of Ucn1. Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0110866), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



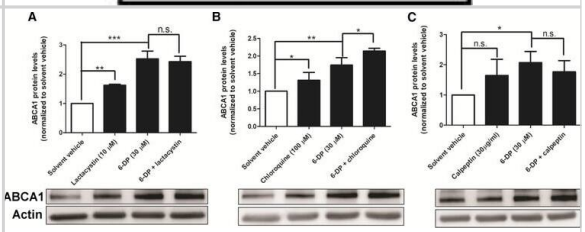
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Effect of evodiamine on ABCA1 transcription & the degradation rate of ABCA1 protein. (a) Differentiated THP-1 macrophages were incubated with 10 μM evodiamine or 10 μM pioglitazone as positive control for 24 h. Total RNA was extracted & ABCA1 mRNA expression levels were quantified by qRT-PCR. (b) 293 T cells were transfected with a luciferase reporter construct driven by the human ABCA1 promoter as described in the Materials & Methods section. After transfection, cells were treated with 10 μM Evodiamine or 1 μM T0901317 as positive control for 24 h. (c) Differentiated THP-1 macrophages were incubated for 24 h with (black circle) or without (Veh; white circle) evodiamine (10 μM) & lysed after addition of cycloheximide (CHX; 140 μM) at different time points (0, 10, 20, 40 min). Western blot analysis shows the decline of ABCA1 protein level with cycloheximide in the presence & absence of evodiamine. All data are means ± S.D. (n = 3) vs. solvent vehicle control (DMSO), *p < 0.05, **p < 0.01, n.s. no significance (ANOVA/Bonferroni). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/30038271), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



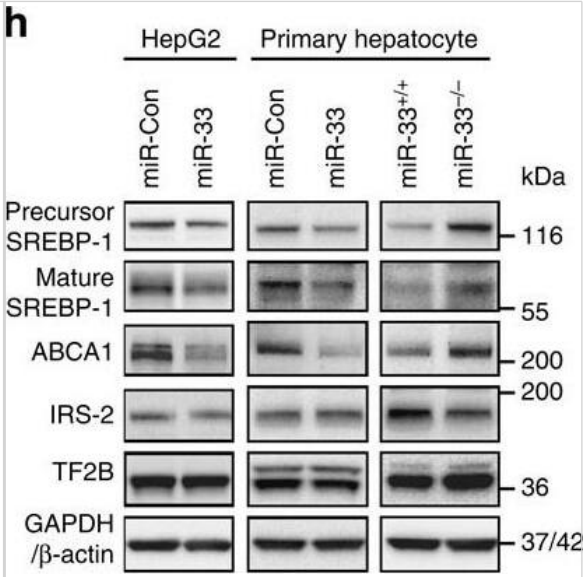
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - miR-33 deficiency improved cholesterol efflux in macrophages. A, Quantitative real-time PCR analysis of Abca1 & Abcg1 in macrophages from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. Values from miR-33+/+Apoe−/− were set at 100%. Values are mean±SE (n=7 each); ***P<0.001. B, Western blotting analysis of ABCA1 & ABCG1 in thioglycollate-elicited peritoneal macrophages from miR-33+/+Apoe−/− & miR-33−/−Apoe−/− mice. GAPDH was used as a loading control. C, Cholesterol efflux from thioglycollate-elicited peritoneal macrophages in the presence or absence of apoA-I (5 or 10 μg/mL). Values are mean ±SE (n=6 each); **P<0.01. D, Cholesterol efflux from thioglycollate-elicited peritoneal macrophages in the presence or absence of HDL-C (50 or 100 μg/mL). Values are mean±SE (n=6 each); **P<0.01. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/23316322), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



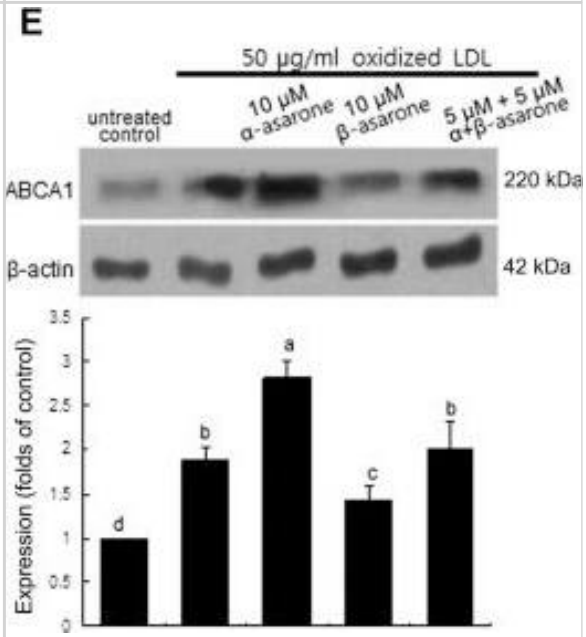
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - A–C) Effects of 6□dihydroparadol & specific proteasomal, lysosomal, & calpain inhibitors on the ABCA1 protein levels. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with or without 6□dihydroparadol (6□DP, 30 μm) for 24 h & incubated for another 3 h with or without the proteasome inhibitor lactacystin (10 μm), the lysosomal inhibitor chloroquine (100 μm), or the calpain inhibitor calpeptin (30 μg mL−1). The ABCA1 protein levels were detected by Western blot analysis. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control; n.s., not significant versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



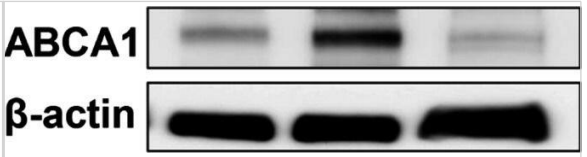
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Srebf1 is a miR-33 target gene.(a) Relative changes in lipid metabolism-related genes in the livers of miR-33−/− mice compared with miR-33+/+ mice fed NC at 16 weeks of age. (n=5–8 each,*P<0.05 in Student's t-test). (b) Conservation of miR-33 target regions in the 3′UTR of Srebf1. Underlined sequences are the potential binding site of miR-33 seed sequences. * indicates the conservation among spieces. (c) 3′UTR reporter assay used to verify the target. Luciferase reporter activity of human & mouse SREBP-1 gene 3′UTR constructs in 293T cells overexpressing miR-control (miR-Con) & miR-33 (n=4 each, *P<0.05 & ***P<0.001 in Student's t-test). (d) miR-33 dose-dependent changes in luciferase reporter activity of mouse Srebf1 3′UTR construct in 293T cells. miR-Con & miR-146a is used as a negative control (n=4 each, *P<0.05 & ***P<0.001 in one-way analysis of valiance test). (e) Luciferase reporter activity of the WT or mutant Srebf1 3′UTR at the potential miR-33 binding site in 293T cells (n=4 each, **P<0.01 in Student's t-test). (f,g) Luciferase reporter activity of SRE-promoter (f) or FAS-promoter (g) in 293T cells. 293T cells were co-transfected with mouse Srebf1 with the full-length 3′UTR or without the 3′UTR, along with expression plasmids for miR-negative control, or miR-33. Values are the mean±s.e. (n=4 each, **P<0.01 versus miR-Con. ***P<0.001 versus miR-Con in one-way analysis of valiance test). (h) Western blotting analysis of SREBP-1, ABCA1, & IRS-2 in miR-33 transduced HepG2 cells & primary hepatocytes & hepatocytes prepared from miR-33+/+ & miR-33−/− mice. Representative western blot images are shown (n=4). Values are the means±s.e.m. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/24300912), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



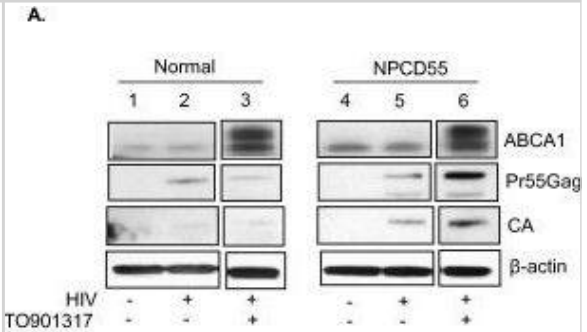
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Chemical structure, (B) cytotoxicity of α-asarone, (C) enhancement of cholesterol efflux by α-asarone, (D & E) upregulation of ABCA1 & ABCG1 by α-asarone & β-asarone, & (F) elevation of retinoid X receptor (RXR)α transcription. J774A.1 murine macrophages were exposed to 50 μg/ml oxidized low-density lipoprotein (LDL) & treated with 1–10 μM purple Perilla frutescens extracts (PPE)-α-asarone & 5–10 μM β-asarone. (B) MTT assay was performed for the measurement of α-asarone toxicity. Graph data represent 1 of 4 independent experiments with multiple estimations. Values are expressed as the percentage cell survival relative to the untreated control cells (cell viability, 100%). (C) Cholesterol efflux was expressed as the percentage fluorescence in the medium relative to total fluorescence. (D & E) For the measurement of ABCA1 & ABCG1 expression, total cell lysates were subjected to western blot analysis with a primary antibody against ABCG1 or ABCG1. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. Bar graphs denoted without a common letter indicate significant difference, P<0.05. (F) RXRα mRNA expression was measured by RT-PCR. GAPDH was used as a housekeeping gene for the co-amplification with RXRα. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



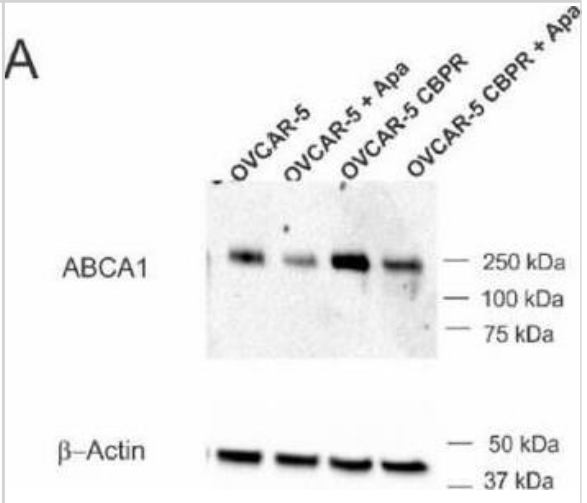
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Bilirubin significantly decreases expression of ATP□binding cassette transporter A1 (ABCA1) protein in THP□1 macrophages treated with LXR agonist (TO901317) to upregulate ABCA1 protein. THP□1 cells were differentiated for 72 hours with 200 nmol/L phorbol□12□myristate□13□acetate & then loaded with unlabeled cholesterol for another 24 hours. Cells were treated with TO901317 (5 µmol/L) or solvent vehicle (0.1% dimethyl sulfoxide [DMSO]) for 24 hours. Afterward, cells were treated with bilirubin (17.1 µmol/L) or solvent vehicle (0.1% DMSO) for 16 hours. The protein levels of ABCA1 were detected by western blot analysis. The bar graphs present mean±SD from 3 independent experiments. ***P<0.001 vs control (determined by ANOVA with Bonferroni post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/28455345), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



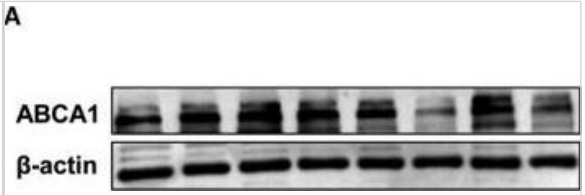
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Induction of cholesterol efflux attenuates the enhancement in HIV-1infectivity in NPCD55 cells. (A) Normal & NPCD55 cells were treated with 5 µM TO-901317 at 24 h post-infection & cultured for 72 h in the presence of the compound. At 96 h post-infection the cells were harvested & ABCA1, Gag, & β-actin expression was detected by Western blotting analysis. All samples were loaded on the same gel. (B) AmplexRed assay was performed to measure free cholesterol content of untreated (filled) & TO-901317-treated (slashed) cells. Cholesterol content was normalized to protein concentration. (C) AmplexRed assay was performed to measure virion-associated cholesterol from purified virus produced in untreated (filled) & TO-901317-treated (slashed) infected cells. Virion-associated cholesterol content was normalized to p24 concentration. (D) TZM-bl reporter assay was performed to measure virus infectivity when cholesterol efflux was induced via TO-901317 stimulation. The ρ values were calculated by performing student T-test (*denotes < 0.05). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/22273177), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



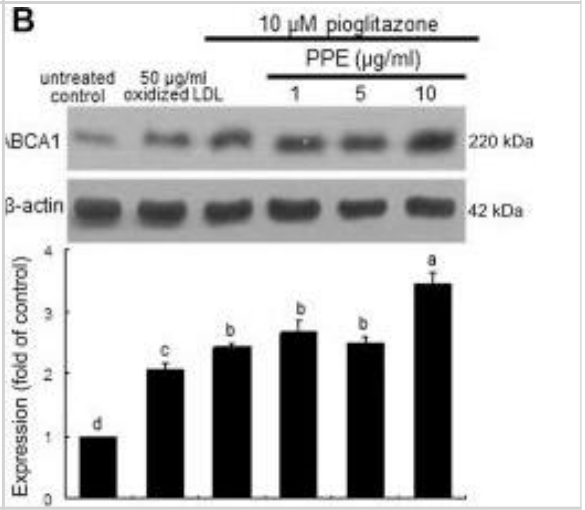
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Effects of apabetalone on ABCA1 expression & carboplatin response in ovarian cancer cells. (A) Apabetalone treatment reduced expression of ABCA1 in OVCAR-5 & OVCAR-5 CBPR cell lines. Cells were treated for 72 h with control medium (DMSO) or apabetalone (80 µM). Protein extracts from OVCAR-5 (~30 µg) were electrophoresed & immunoblotted with rabbit polyclonal ABCA1 antibody (1/1000, NB400-105, Novus Biological) & β-actin (1/2000, Abcam) was used as a loading control. (B) Quantification of Western blot. OVCAR-5 (C), CaOV3 (D), OVCAR-5 CBPR (E) & CaOV3 CBPR (F) cell survival following treatment with carboplatin (CBP) alone (0-200 µM, black line) & in combination with apabetalone (Apa, 80 µM, purple line). (G) Carboplatin IC50 for OVCAR-5 & OVCAR-5 CBPR cells ± apabetalone. Data are mean ± SEM from three independent experiments. *P < 0.05, Student's t-test. (H) Carboplatin IC50 for CaOV3 & CaOV3 CBPR cells ± apabetalone. Data are mean ± SEM from three independent experiments. P < 0.05, Student's t-test. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/35582032), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



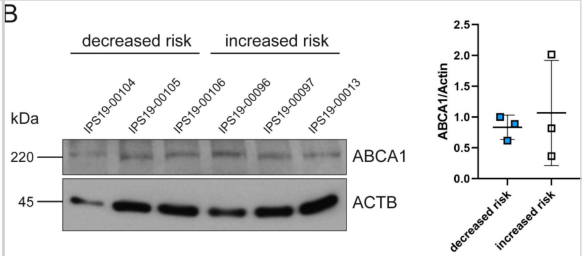
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Expression of ATP□binding cassette transporter A1 (ABCA1) protein in THP□1 macrophages treated with bilirubin & in peripheral blood mononuclear cells (PBMCs) from Gilbert syndrome (GS) patients. A, Bilirubin suppresses the expression of ABCA1 protein in THP□1□derived macrophages time□dependently. THP□1 cells were differentiated as described in Figure 1 & then loaded with unlabeled cholesterol for another 24 hours. Cells were treated with bilirubin (17.1 µmol/L) for 4, 8, 16, & 24 hours. The protein levels of ABCA1 were detected by western blotting. Control was treated with solvent vehicle (0.1% dimethyl sulfoxide). The bar graph presents mean±SD from 3 independent experiments. B, Expression of ABCA1 protein is decreased in PBMCs from participants with high bilirubin blood levels (GS) compared with healthy controls. The protein levels of ABCA1 were detected by western blotting. The bar graph presents mean±SEM (n=28 per group). *P<0.05, **P<0.01 & ***P<0.001 vs control. ns indicates not significant (determined by Student t test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/28455345), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



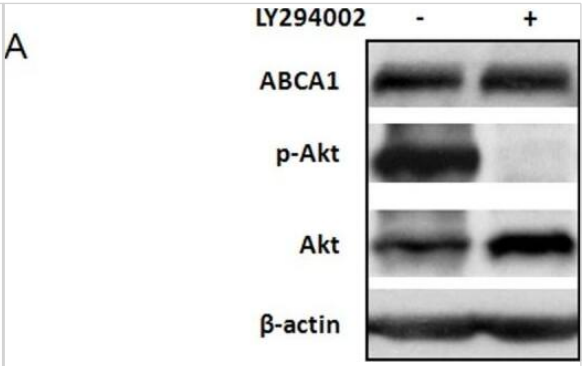
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Time course response of ABCA1 induction by pioglitazone, & (B) enhancement of ABCA1 & (C) peroxisome proliferator-activated receptor γ (PPARγ) by purple Perilla frutescens extracts (PPE). J774A.1 murine macrophages were incubated with 10 µM pioglitazone & 50 µg/ml oxidized low-density lipoprotein (LDL) in the absence or presence of 1–10 µg/ml PPE. For the measurement of expression of (A & B) ABCA1 & (C) PPARγ, total cell lysates were subjected to western blot analysis with a primary antibody against ABCA1 or PPARγ. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. Bar graphs denoted without a common letter indicate significant difference, P<0.05. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



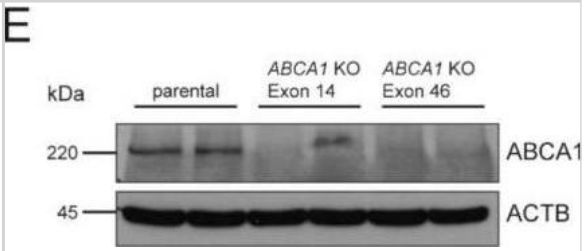
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - LXR agonist-stimulated ABCA1 expression & function in patient-derived iRPEs. Patient-derived iRPE lines were stimulated for 16 h with LXR agonist & are relative to unstimulated cells shown in Figure 2. (A) Expression of ABCA1 mRNA normalized to RPL28 & relative to unstimulated cells shown in Figure 2B. (B) Western blot analysis of ABCA1 protein levels. Actin was detected as loading control. ABCA1 expression was quantified & normalized to actin. (C) Cholesterol efflux assay after direct cell labeling & in the presence of ApoAI. (D) Cholesterol efflux assay after phagocytosis of BODIPY-cholesterol-loaded POSs & in the presence of ApoAI. Data are relative to unstimulated cells shown in Figure 2F,G, respectively. Data are presented as means ± SD (n = 3). Unpaired Student's t-test. * p < 0.05. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/35328615), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



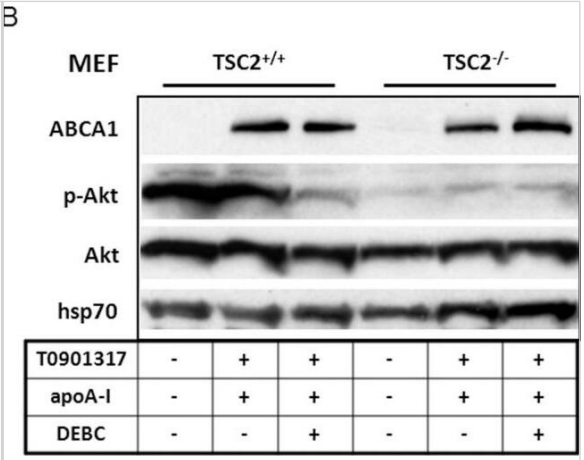
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Akt inhibition by LY294002 or Akt1/2 also enhances cholesterol efflux to apoA-I from ABCA1-expressing BHK cells.A) BHK cells were induced as in Fig. 1 & then incubated with LY294002 (200 μM) for 2 h. Cells were then lysed & analysed for ABCA1, phosphorylated Akt (p-Akt) & total Akt by Western-blotting. Hsp70 was also blotted as loading control. B & C) BHK cells were labeled with [3H] cholesterol & induced with 10 nM mifepristone overnight. Cholesterol efflux was measured after 2 h incubation with BSA (1 mg/ml) or BSA plus apoA-I (5 μg/ml). Some of cells were also incubated with indicated doses of LY294002 (B) or Akt1/2 (C), in addition to apoA-I, during 2 h efflux period. Results are presented as the average of triplicate wells with standard deviation & representative of more than three independent experiments. *** P<0.0001, **P<0.001 & *P<0.05 vs apoA-I only. Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0113789), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



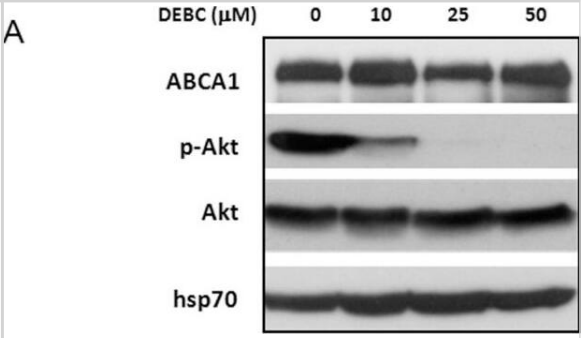
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Generation, differentiation & analysis of ABCA1-deficient iRPEs. (A) Immunofluorescence staining for ZO-1 (red) in 4-week cultured iRPEs. Nuclei were stained with DAPI (blue). Scale bar = 100 μm. (B) Bright-field microscopy of iRPEs. (C) Ct values of RPE marker genes BEST1, OTX2, RLBP1 & RPE65 in iRPEs & parental iPSCs obtained by qPCR. Values shown are means ± SD (n = 3). Unpaired Student's t-test. **** p < 0.0001. (D) Sequence alignment of parts of exon 14 (top) & exon 46 (bottom) of ABCA1-deficient iRPE cell clones & parental line. (E) Western blot of ABCA1 protein levels in ABCA1-deficient iRPE cell lines & parental cell line after 16 h of stimulation with 1 μM LXR agonist. Actin was detected as loading control. (F) Relative expression of ABCA1 mRNA in ABCA1-deficient iRPE cell lines & parental cell line normalized to ACTB. Values shown are means ± SD (n = 3). One-way ANOVA with Tukey's post hoc test. **** p < 0.0001. (G) Relative expression of ABCG1 mRNA in ABCA1-deficient iRPE cell lines & parental cell line normalized to ACTB. Values shown are means ± SD (n = 3). One-way ANOVA with Tukey's post hoc test. ** p < 0.01; *** p < 0.001. (H) Cholesterol efflux assay in iRPEs in the presence of ApoAI and/or LXR agonist (LXR ag). Values shown are means ± SD (n = 3). Two-way ANOVA with Bonferroni post hoc test vs. DMSO + BSA control. **** p < 0.0001. (I) Bright-field (BF) microscopy & Nile Red fluorescence microscopy (overview & close-up view marked with white squares) of 4-week cultured ABCA1-deficient iRPE cell lines & parental cell line. Relative fluorescence was quantified & is shown as mean ± SD (n = 8). One-way ANOVA with Tukey's post hoc test. * p < 0.05, **** p < 0.0001. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/35328615), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



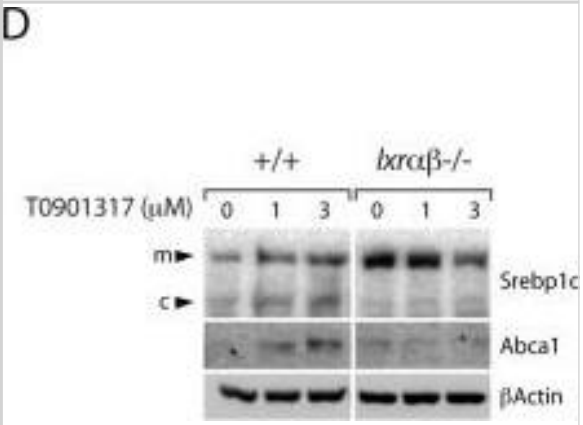
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Akt inhibition enhances cholesterol efflux to apoA-I from wt MEFs, but not from TSC2 -/- MEFs where mTORC1 is constitutively activated.A) Mouse embryonic fibroblasts (MEFs), wt & TSC2-/-, were induced overnight with T0901317 (10 µM). Cell lysates were collected for Western blotting for phosphorylated S6K & total S6K. B) wt & TSC2-/- MEFs were incubated with or without T0901317 (10 µM) overnight. Some of cells were then incubated with apoA-I (10 µg/ml) or apoA-I plus DEBC (25 µM) for 2 h. The expression of ABCA1, phosphorylated Akt, total Akt & loading control hsp70 were analyzed by Western blotting. C) wt & TSC2-/- were labeled with [3H] cholesterol for 1 day & then incubated with or without T0901317 (10 µM) overnight. Some of cells were then incubated with apoA-I (10 µg/ml) or apoA-I plus DEBC (25 µM) for 2 h to analyze cholesterol efflux. Data presented as the average of triplicate wells with standard deviation & representative of at least three independent experiments. *** P<0.0001 vs apoA-I only. Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0113789), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



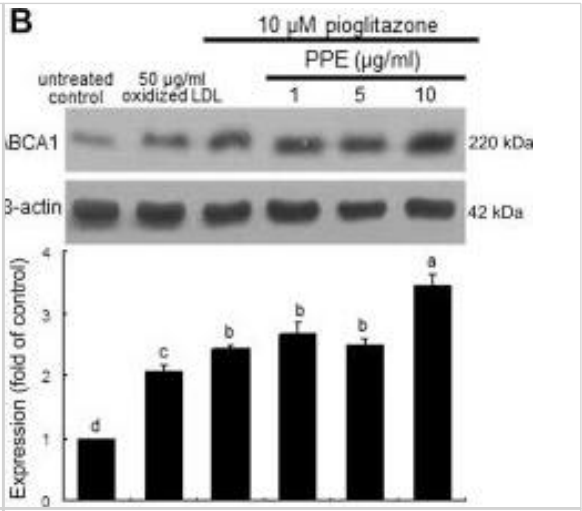
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Akt inhibition by DEBC enhances cholesterol efflux to apoA-I specifically from ABCA1-expressing BHK cells.A) BHK-ABCA1 cells were induced with mifepristone (10 nM) overnight & then incubated with indicated doses of DEBC for 2 h. Cells were then lysed & Western-blotted for ABCA1, phosphorylated Akt (p-Akt) & total Akt. Hsp70 was also blotted as loading control. B) BHK-ABCA1 cells were labeled with [3H] cholesterol & induced overnight as above. After 2 h incubation with BSA (1 mg/ml) or BSA plus apoA-I (5 µg/ml), cholesterol efflux was measured as described in the Methods section. Some of the cells were also incubated with indicated doses of DEBC, in addition to apoA-I, during 2 h efflux period. C) BHK-ABCA1 & BHK-A937V cells were induced with mifepristone (10 nM) overnight. 2 h Cholesterol efflux was measured as above either with or without DEBC (25 µM). Results are presented as the average of triplicate wells with standard deviation & representative of more than three independent experiments. *** P<0.0001 & **P<0.001 vs apoA-I only. Image collected & cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0113789), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



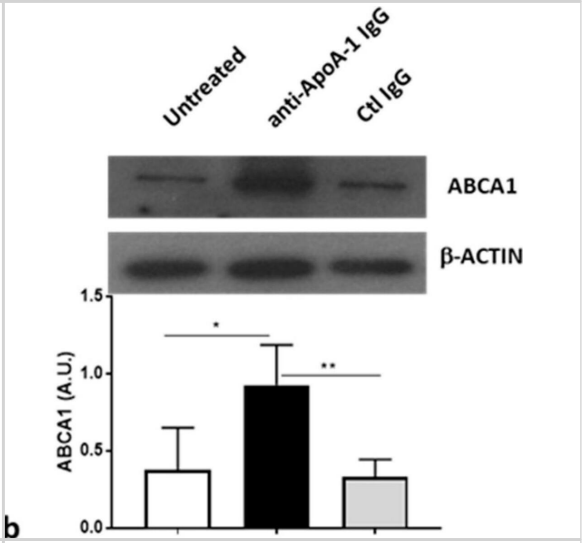
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - LXRs control expression of genes involved in cholesterol homeostasis & fatty acid synthesis in MPECs.(A) qPCR analysis of Abca1, Abcg1 & Idol levels in WT (+/+) & Lxrαβ−/− (lxr−/−) MPECs after DMSO (vehicle) or T0901317 stimulation (B) Effect of 9-cis retinoic acid and/or T0901317 stimulation on Abca1 accumulation levels in WT (+/+) & Lxrαβ−/− (lxr−/−) MPECs (C) qPCR analysis of Srebp1, Acc, & Fas levels in WT (+/+) & Lxrαβ−/− (lxr−/−) MPECs after DMSO (vehicle) or T0901317 stimulation. (qPCR analysis results from 4 independant experiments & was normalized using 36b4 gene). *p<0.05, **p<0.01, ***p<0.001 in Student's t test. Error bars represent mean ± SEM. (D) Western blot analysis was performed on WT (+/+) or Lxrαβ−/− MPECs using Srebp1c, Abca1 & β-Actin antibodies. (E) Oil-Red O staining (ORO) & Normarski/Dapi of WT (+/+) & Lxrαβ−/− MPECs, or WT MEFs, treated for 48h with DMSO (vehicle) or T0901317 (1 µM). Head arrows indicate lipid droplets. Scale bars 100 µm. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/23554947), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



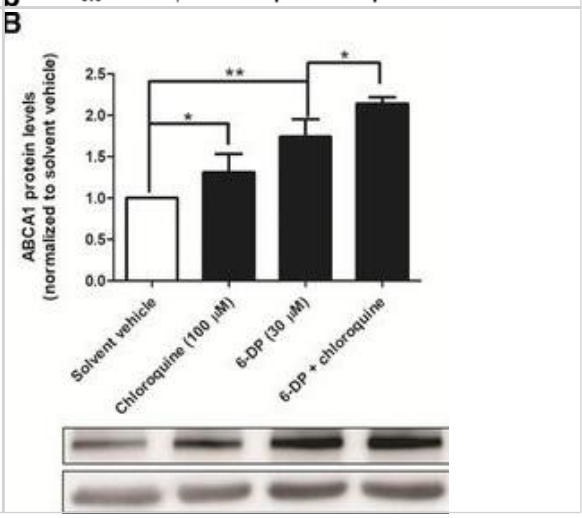
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Time course response of ABCA1 induction by pioglitazone, & (B) enhancement of ABCA1 & (C) peroxisome proliferator-activated receptor γ (PPARγ) by purple Perilla frutescens extracts (PPE). J774A.1 murine macrophages were incubated with 10 µM pioglitazone & 50 µg/ml oxidized low-density lipoprotein (LDL) in the absence or presence of 1–10 µg/ml PPE. For the measurement of expression of (A & B) ABCA1 & (C) PPARγ, total cell lysates were subjected to western blot analysis with a primary antibody against ABCA1 or PPARγ. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. Bar graphs denoted without a common letter indicate significant difference, P<0.05. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



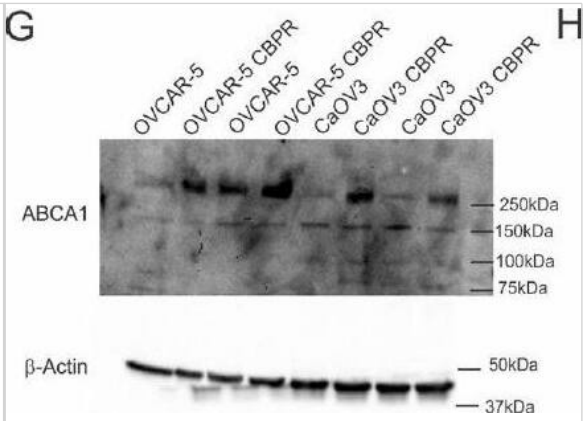
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Anti-ApoA-1 IgGs upregulate ABCA1. ABCA1 was increased after 16 & 24 h of anti-ApoA-1 IgG exposure to HMDM at the mRNA level as revealed by RT-PCR (a) as well as at the protein level after 24 h anti-apoA-1 IgG stimulation, as revealed by Western blot analysis (b). In panel a, data are expressed as fold change expression of the mean ± SD of ABCA1 calculated by ΔΔCT method of six independent experiments (n = 6) & values were normalized to untreated condition. p-values were calculated using the Student's t-test: * p < 0.05, ** p < 0.01, **** p < 0.0001. (b) One of four representative Western blots is shown. Data are the mean ± SD of band intensity volume/actin intensity volume of four independent experiments (n = 4). p-values were calculated using the Student's t-test: * p = 0.02, ** p = 0.005. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/31766415), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



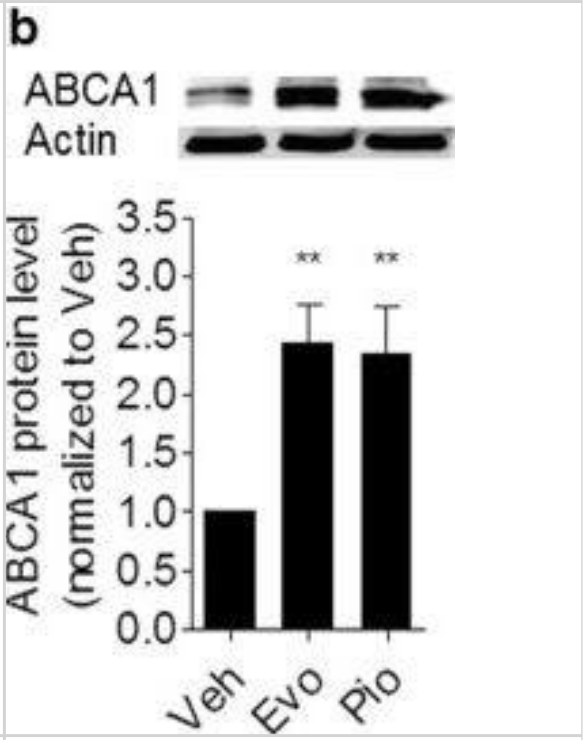
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - A–C) Effects of 6□dihydroparadol & specific proteasomal, lysosomal, & calpain inhibitors on the ABCA1 protein levels. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with or without 6□dihydroparadol (6□DP, 30 µm) for 24 h & incubated for another 3 h with or without the proteasome inhibitor lactacystin (10 µm), the lysosomal inhibitor chloroquine (100 µm), or the calpain inhibitor calpeptin (30 µg mL−1). The ABCA1 protein levels were detected by Western blot analysis. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control; n.s., not significant versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



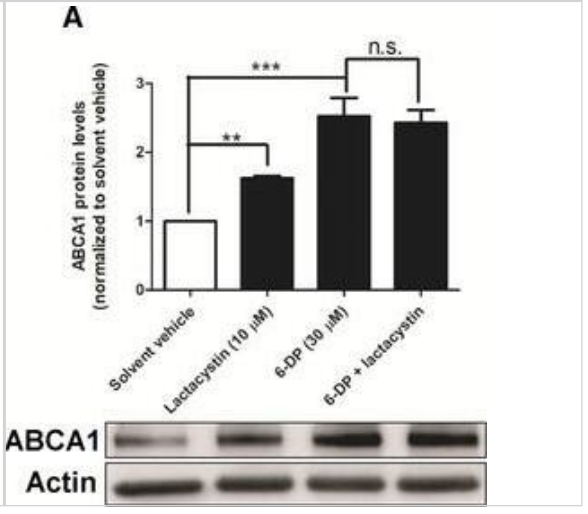
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - ABCA1 protein expression is upregulated in ovarian cancer cells with acquired carboplatin resistance. ABCA1 expression in OVCAR-5 (A) & CaOV3 (C) & carboplatin resistant OVCAR-5 CBPR (B) & CaOV3 CBPR (D) by immunocytochemistry using rabbit polyclonal ABCA1 antibody (1/100, NB400-105, Novus Biological). (E) OVCAR-5 cells with Rabbit IgG & (F) CaOV3 cells with Rabbit IgG. (G) Protein extracts from OVCAR-5 (~30 µg) & CaOV3 cell lines (~60 µg) were electrophoresed & immunoblotted with rabbit polyclonal ABCA1 antibody (1/1000, NB400-105, Novus Biological), & β-actin (1/2000, Abcam) was used as a loading control. A major band was detected at ~250 kDa, which is the predicted size for ABCA1. (H) Quantitation of ABCA1 Western blots. Data are from 2-4 independent experiments. Statistical significance was determined using the Student's t-test, *P < 0.05. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/35582032), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



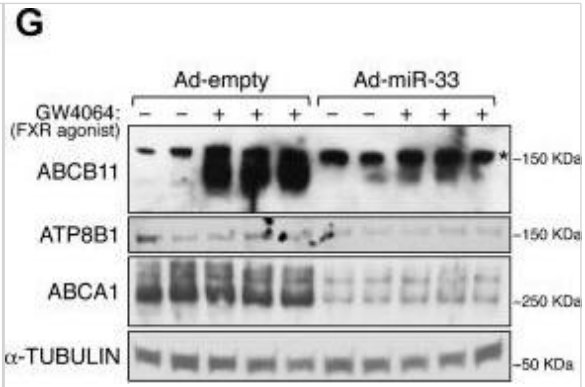
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Evodiamine enhances apo A1-mediated ChE from THP-1 macrophages & increases ABCA1 protein level. (a) Differentiated THP-1 cells were loaded with [3H]-cholesterol together with the indicated treatments for 24 h. On the next day, the cells were washed twice with PBS & incubated with the same compounds [solvent vehicle control (Veh; ≤0.1% DMSO), evodiamine (1–20 µM), & the PPARγ agonist pioglitazone (10 µM) as positive control] with or without 10 µg/mL apo A1. Extracellular as well as intracellular radioactivities were quantified with scintillation counter. Differentiated THP-1-derived macrophages were treated with solvent vehicle control (Veh; ≤0.1% DMSO), evodiamine (10 µM), & the PPARγ agonist pioglitazone (10 µM) as positive control. After 24 h incubation, the cells were lysed & 20 µg protein was resolved via SDS-PAGE. Immunodetection was performed with antibodies against the indicated proteins, ABCA1 (b), ABCG1 (c), & SR-B1 (d), & visualized by chemiluminescence detection. All experiments were performed at least three times & data are presented as means ± S.D. vs. solvent vehicle control, *p < 0.05, **p < 0.01, ***p < 0.001, n.s. no significance (ANOVA/Bonferroni). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/30038271), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



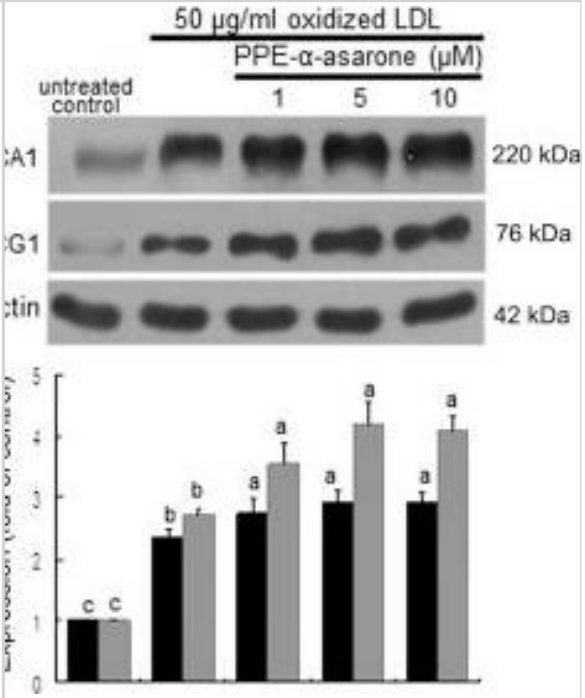
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - A–C) Effects of 6□dihydroparadol & specific proteasomal, lysosomal, & calpain inhibitors on the ABCA1 protein levels. THP□1 cells were differentiated as described in Figure 2, & then loaded with unlabeled cholesterol for 24 h. Cells were treated with or without 6□dihydroparadol (6□DP, 30 µm) for 24 h & incubated for another 3 h with or without the proteasome inhibitor lactacystin (10 µm), the lysosomal inhibitor chloroquine (100 µm), or the calpain inhibitor calpeptin (30 µg mL−1). The ABCA1 protein levels were detected by Western blot analysis. The bar graphs represent mean ± SD from three independent experiments. *p < 0.05, **p < 0.01, & ***p < 0.001 versus control; n.s., not significant versus control (determined by Student's t□test or ANOVA with Bonferroni's post hoc test). Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/29802792), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



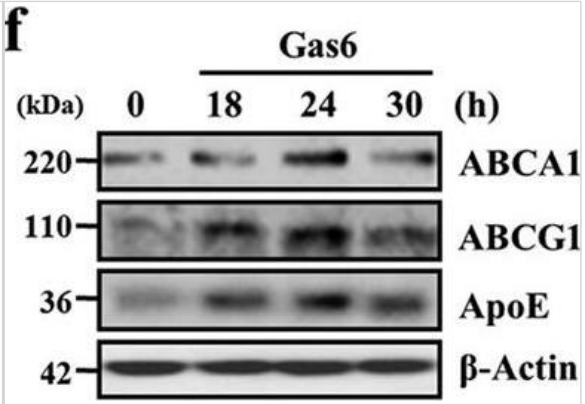
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Functional miR-33 responsive elements in the 3′UTR of ATP8B1 & ABCB11A,B. Conserved sequences in the 3′UTR of ATP8B1 & ABCB11 are partially complementary to miR-33. The element in human ATP8B1 is located 1877–1897 nt after the stop codon. The element in ABCB11 overlaps the stop codon in primates, while rodents show a conserved sequence 732–751 nt after the stop codon.C,D. Luciferase assays in HEK293 cells using the whole 3′UTR of human or murine ATP8B1 & ABCB11, or a single copy of the responsive elements (RE) identified above, or mutant responsive elements (RE*; where AATGCA was mutated to GGGTTG to prevent complementarity to the seed sequence of the miRNA), co-transfected with (closed bars) or without (open bars) a vector to overexpress miR-33. In grey, data from empty (negative control) & R33 (positive control containing a 100% match to miR-33) reporter vectors.E,F. Relative mRNA expression of canalicular transporters in primary murine hepatocytes (n = 4 dishes/condition) & human HuH-7 hepatoma cells (n = 3 dishes/condition) transduced 48 h with empty or miR-33 adenovirus.G. Relative protein levels in HuH7 cells transduced with empty or miR-33 adenovirus. Some cells were incubated for 16 h in the presence of FXR:RXR agonists (2 μmol/L GW4064 : 1 μmol/L 9-cis-retinoic acid) to induce ABCB11. Asterisk indicates a non-specific band. Data shown as mean ± SD; **p < 0.01. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/22767443), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



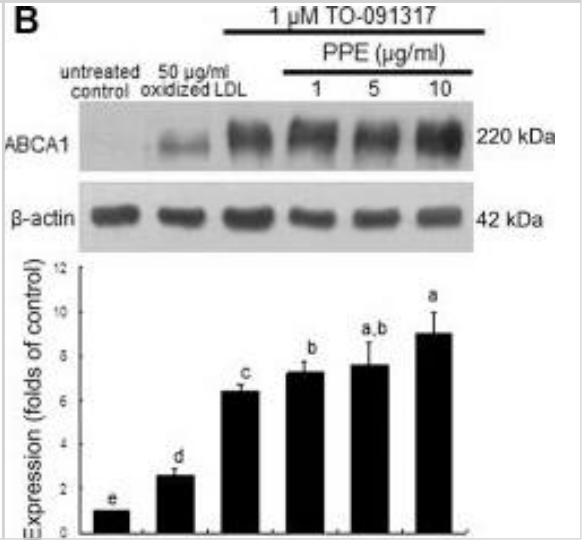
Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Chemical structure, (B) cytotoxicity of α-asarone, (C) enhancement of cholesterol efflux by α-asarone, (D & E) upregulation of ABCA1 & ABCG1 by α-asarone & β-asarone, & (F) elevation of retinoid X receptor (RXR)α transcription. J774A.1 murine macrophages were exposed to 50 μg/ml oxidized low-density lipoprotein (LDL) & treated with 1–10 μM purple Perilla frutescens extracts (PPE)-α-asarone & 5–10 μM β-asarone. (B) MTT assay was performed for the measurement of α-asarone toxicity. Graph data represent 1 of 4 independent experiments with multiple estimations. Values are expressed as the percentage cell survival relative to the untreated control cells (cell viability, 100%). (C) Cholesterol efflux was expressed as the percentage fluorescence in the medium relative to total fluorescence. (D & E) For the measurement of ABCA1 & ABCG1 expression, total cell lysates were subjected to western blot analysis with a primary antibody against ABCG1 or ABCG1. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. Bar graphs denoted without a common letter indicate significant difference, P<0.05. (F) RXRα mRNA expression was measured by RT-PCR. GAPDH was used as a housekeeping gene for the co-amplification with RXRα. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - Gas6 treatment enhances expression of LXRα & LXRβ & their target genes in BMDM.Mouse BMDM were stimulated with 400 ng/ml Gas6, 1 μM T0901317, 10 ng/ml interferon (IFN)-γ, 10 ng/ml IL-4, or 100 ng/ml LPS for 4 h (a,b) or 400 ng/ml Gas6 for the indicated times (c–g). (a,b,e) The amounts of the LXRα, LXRβ, ABCA1, ABCG1, ApoE, AIM, Arg2, VEGF, YM1, & Arg1 mRNAs were analyzed by real-time PCR & normalized to that of Hprt mRNA. (c,d,f,g) The relative abundances of LXRα, LXRβ, ABCA1, ABCG1, ApoE, Aim, Arg2, & VEGF proteins were determined by Western blotting analysis. The relative densitometric intensity was determined for each band & normalized to β-actin. Data in all bar graphs are means ± SEM of three independent experiments. *P < 0.05 compared with control. Image collected & cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/27406916), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Western Blot: ABCA1 Antibody - BSA Free [NB400-105] - (A) Time course response of ABCA1 induction by TO-091317 & (B) upregulation of ABCA1 by purple Perilla frutescens extracts (PPE), & (C) enhancement of liver X receptor (LXR)α induction by PPE. J774A.1 murine macrophages were cultured with 1 μM TO-091317 or 50 μg/ml Cu2+-oxidized low-density lipoprotein (LDL) in the absence or presence of 1–10 μg/ml PPE. For the measurement of expression of (A & B) ABCA1 & (C) LXRα, total cell lysates were subjected to western blot analysis with a primary antibody against ABCA1 or LXRα. β-actin was used as an internal control. Bar graphs (means ± SEM, n=3) represent quantitative densitometric results of the upper bands. Bar graphs denoted without a common letter indicate significant difference, P<0.05. Image collected & cropped by CiteAb from the following publication (https://www.spandidos-publications.com/10.3892/ijmm.2015.2101), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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More publications at http://www.novusbio.com/NB400-105

Procedures

Western Blot protocol for ABCA1 Antibody (NB400-105)

RAW macrophages were treated with 9-cis-retinoic acid and 22R-hydroxycholesterol, known inducers of ABCA1 expression in macrophages. The total cell post-nuclear lysate (40ug protein) was separated by SDS-PAGE and detected using a 1:1000 dilution of NB400-105 affinity purified ABCA1 antibody incubated for 1 hour at room temperature. ABCA1 has been found to run as 3 bands by many researchers; this is probably due to protein modifications such as glycosylation.

NOTE: An important factor in detecting ABCA1 is in the cell type used. ABCA1 is expressed in very low levels in most cell types. Therefore, ABCA1 expression needs to be induced by using 22-hydroxycholesterol and 9-cis-retinoic acid as ligands for the transcription factor LXR.

1. Without heating at all (leave at room temp for about 15 to 20 minutes with Beta-mercaptoethanol), load 40 ug post-nuclear lysates* to 7.5% or 4-15% Tris-HCL SDS gel (Bio-RAD) in sample buffer. Do NOT boil the samples. (NP-40 will not interfere with the running of the protein on SDS-PAGE.)
2. Transfer to nitrocellulose membrane at 100V 1hr or 30V overnight.
3. Block membrane in 5% milk in TBS-T for at least 1 hr. Wash with TBS-T 5 minutes.
4. Blot with anti-ABCA1 antibody in 3% milk in TBS-T for 1 hour.
5. Wash with TBS-T 3 times, 10 minutes each.
6. Blot with anti-rabbit secondary according to the recommended dilutions in 3% milk in TBS-T for 1 hour.
7. Wash with TBS-T 3 times, 10 minutes each.
8. Detect with chemiluminescent reagent (Pierce).
9. Image

TBS-T: Tris-buffered-saline with Tween-20

See also the specific references mentioned in the datasheet. *Post-nuclear lysate is the result of sonication or dounce homogenization of lysate, centrifugation at low-speed, and the removal of nuclei. The resulting supernatant is called post-nuclear and contains cytosolic and membrane proteins without any of the nuclear components.

Immunohistochemistry-Paraffin protocol for ABCA1 Antibody (NB400-105)
ABCA1 Antibody:
Immunohistochemistry-Formalin Fixed Paraffin Embedded sections

I. Deparaffinization:
A. Treat slides with Xylene: 3 changes for 5 minutes each. Drain slides for 10 seconds between changes.
B. Treat slides with 100% Reagent Alcohol: 3 changes for 5 minutes each. Drain slides for 10 seconds between changes.

II. Quench Endogenous Peroxidase:
A. Place slides in peroxidase quenching solution: 15-30 minutes.
To Prepare 200 ml of Quenching Solution:
-Add 3 ml of 30% Hydrogen Peroxide to 200 ml of Methanol.
-Use within 4 hours of preparation
B. Place slides in distilled water: 2 changes for 2 minutes each.

III. Retrieve Epitopes:
A. Preheat Citrate Buffer. Place 200 ml of Citrate Buffer Working Solution into container, cover and place into steamer. Heat to 90-96 degrees C.
B. Place rack of slides into hot Citrate Buffer for 20 minutes. Cover.
C. Carefully remove container with slides from steamer and cool on bench, uncovered, for 20 minutes.
D. Slowly add distilled water to further cool for 5 minutes.
E. Rinse slides with distilled water. 2 changes for 2 minutes each.

IV. Immunostaining Procedure:
A. Remove each slide from rack and circle tissue section with a hydrophobic barrier pen (e.g. Liquid Blocker-Super Pap Pen).
B. Flood slide with Wash Solution. Do not allow tissue sections to dry for the rest of the procedure.

C. Drain wash solution and apply 4 drops of Blocking Reagent to each slide and incubate for 15 minutes.
D. Drain Blocking Reagent (do not wash off the Blocking Reagent), apply 200 ul of Primary Antibody solution to each slide, and incubate for 1 hour.
E. Wash slides with Wash Solution: 3 changes for 5 minutes each.
F. Drain wash solution, apply 4 drops of Secondary antibody to each slide and incubate for 1 hour.
G. Wash slides with Wash Solution: 3 changes for 5 minutes each.
H. Drain wash solution, apply 4 drops of DAB Substrate to each slide and develop for 5-10 minutes. Check development with microscope.
I. Wash slides with Wash Solution: 3 changes for 5 minutes each.
J. Drain wash solution, apply 4 drops of Hematoxylin to each slide and stain for 1-3 minutes. Increase time if darker counterstaining is desired.
K. Wash slides with Wash Solution: 2-3 changes for 2 minutes each.
L. Drain wash solution and apply 4 drops of Bluing Solution to each slide for 1-2 minutes.
M. Rinse slides in distilled water.
N. Soak slides in 70% reagent alcohol: 3 minutes with intermittent agitation.
O. Soak slides in 95% reagent alcohol: 2 changes for 3 minutes each with intermittent agitation.
P. Soak slides in 100% reagent alcohol: 3 changes for 3 minutes each with intermittent agitation. Drain slides for 10 seconds between each change.
Q. Soak slides in Xylene: 3 changes for 3 minutes each with intermittent agitation. Drain slides for 10 seconds between each change.
R. Apply 2-3 drops of non-aqueous mounting media to each slide and mount coverslip.
S. Lay slides on a flat surface to dry prior to viewing under microscope.

NOTES:
-Use treated slides (e.g. HistoBond) to assure adherence of FFPE sections to slide.
-Prior to deparaffinization, heat slides overnight in a 60 degrees C oven.
-All steps in which Xylene is used should be performed in a fume hood.
For Epitope Retrieval, a microwave or pressure cooker may be substituted for the steamer method. Adjust times as necessary depending on conditions.
-For the initial IHC run with a new primary antibody, test tissues with and without Epitope Retrieval. In some instances, Epitope Retrieval may not be necessary.
-200 ul is the recommended maximum volume to apply to a slide for full coverage. Using more than 200 ul may allow solutions to wick off the slide and create drying artifacts. Small tissue sections less than 200 ul may be used.
-5 minutes of development with DAB Substrate should be sufficient. Do not develop for more than 10 minutes. If 5 minutes of development causes background staining, further dilution of the primary antibody may be necessary.
Hematoxylin should produce a light nuclear counterstain so as not to obscure the DAB staining. Counterstain for 1-1.5 minutes for nuclear antigens. Counterstain for 2-3 minutes for cytoplasmic and membranous antigens. If darker counterstaining is desired increase time (up to 10 minutes).

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Products Related to NB400-105

NBP1-30159	Raw 264.7 Whole Cell - T0901317 treated Assay Kit
NB400-105PEP	ABCA1 Antibody Blocking Peptide
HAF008	Goat anti-Rabbit IgG Secondary Antibody [HRP]
NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

Limitations

This product is for research use only and is not approved for use in humans or in clinical diagnosis. Primary Antibodies are guaranteed for 1 year from date of receipt.

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