PRODUCT INFORMATION

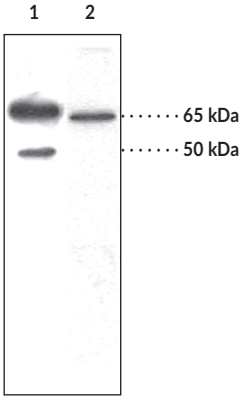
SOAT-1/ACAT-1 Polyclonal Antibody

Item No. 100028

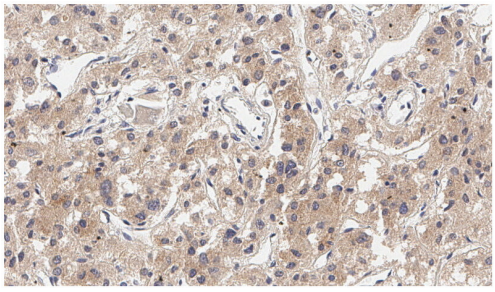
Overview and Properties

Contents:	This vial contains 500 µl of peptide affinity-purified polyclonal antibody.
Synonyms:	Acyl-coenzyme A: Cholesterol Acyltransferase-1, Cholesterol Acyltransferase 1, Sterol O-Acyltransferase 1
Immunogen:	Synthetic peptide from the N-terminal region of human SOAT-1/ACAT-1
Species Reactivity:	(+) Human, mouse, porcine, rat; other species not tested
Uniprot No.:	P35610
Form:	Liquid
Storage:	-20°C (as supplied)
Stability:	≥3 years
Storage Buffer:	PBS, pH 7.2, with 50% glycerol and 0.02% sodium azide
Host:	Rabbit
Applications:	Immunoflouresence (IF), Immunohistochemistry (IHC) and Western blot (WB); the recommended starting dilution is 1:200. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically.

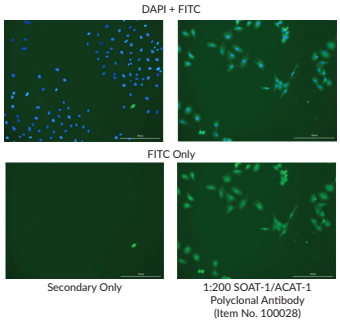
Images



Lane 1: Rat liver (30 µg)
Lane 2: HepG2 cell lysate (30 µg)



Immunohistochemistry analysis of formalin-fixed, paraffin-embedded (FFPE) human adrenal tissue after heat-induced antigen retrieval in pH 6.0 citrate buffer. After incubation with SOAT-1/ACAT-1 Polyclonal Antibody, at a 1:200 dilution, slides were incubated with biotinylated secondary antibody, followed by alkaline phosphatase-streptavidin and chromogen (DAB).



Immunofluorescence analysis of paraformaldehyde-fixed A549 cells. After incubation with SOAT-1/ACAT-1 Polyclonal Antibody (Item No. 100028) at a 1:200 dilution (or negative control), cells were incubated with FITC-labeled anti-rabbit IgG (Item No. 10006588), followed by DAPI nuclear stain. Images show FITC alone or both fluorescence channels to highlight nuclear staining (where applicable).

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PRODUCT INFORMATION

Description

Sterol O-acyltransferase 1 (SOAT-1), also known as acyl-coenzyme A:cholesterol acyltransferase-1 (ACAT-1), is an enzyme encoded by *ACAT1* in humans that catalyzes the intracellular formation of cholesterol esters from cholesterol and long-chain fatty acyl-coenzyme A.[1] It is ubiquitously expressed and localized to the rough endoplasmic reticulum where it preferentially utilizes oleic acid (Item Nos. 90260 | 24659) or palmitic acid (Item No. 10006627) as fatty acid substrates for the synthesis of cholesterol esters, which are stored intracellularly or packaged into chylomicrons or VLDL and secreted into the blood stream. SOAT-1/ACAT-1 protein levels are increased in macrophages under various pathological conditions, including atherosclerosis.[2] SOAT-1/ACAT-1 activity is increased by cholesterol *in vitro*, and *ACAT1* expression is increased by stimulation with the pro-inflammatory cytokines TNF-α or IFN-γ in isolated human and THP-1 monocytes, respectively.[1,3] *ACAT1* silencing in human H4 neuroglioma cells overexpressing amyloid-β precursor protein (APP) reduces secretion of soluble amyloid-β (Aβ) and Aβ42 (Item No. 20574).[4] Genome-wide deletion of *Acat1* reduces macrophage infiltration and neutral lipid deposition in atherosclerotic aortic lesions and decreases serum total cholesterol levels, but increases brain cholesterol deposition, in *ApoE*$^{-/-}$ mice fed a Western diet.[5] Cayman's SOAT-1/ACAT-1 Polyclonal Antibody can be used for immunofluoresence (IF), immunohistochemistry (IHC) and Western blot (WB) applications. The antibody recognizes the N-terminus to detect full-length SOAT-1/ACAT-1 at approximately 65 kDa from human, mouse, porcine, and rat samples.

References

1. Pramfalk, C., Eriksson, M., and Parini, P. Cholesteryl esters and ACAT. *Eur. J. Lipid Sci. Technol.* **114(6)**, 624-633 (2012).
2. Sakashita, N., Miyazaki, A., Chang, C.C.Y., *et al.* Acyl-coenzyme A:cholesterol acyltransferase 2 (ACAT2) is induced in monocyte-derived macrophages: In vivo and in vitro studies. *Lab. Invest.* **83(11)**, 1569-1581 (2003).
3. Chang, T.-Y., Li, B.-L., Chang, C.Y., *et al.* Acyl-coenzyme A:cholesterol acyltransferases. *Am. J. Physiol. Endocrinol. Metab.* **297(1)**, E1-E9 (2009).
4. Huttunen, H.J., Greco, C., and Kovacs, D.M. Knockdown of ACAT-1 reduces amyloidogenic processing of APP. *FEBS Lett.* **581(8)**, 1688-1692 (2007).
5. Accad, M., Smith, S.J., Newland, D.L., *et al.* Massive xanthomatosis and altered composition of atherosclerotic lesions in hyperlipidemic mice lacking acyl CoA:cholesterol acyltransferase 1. *J. Clin. Invest.* **105(6)**, 711-719 (2000).

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