SunLong Biotech Co.,LTD
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Rabbit Anti-APOA1 antibody

SL0849R

Product Name	APOA1
Chinese Name	载 LipoproteinA1 抗体
Alias	Apo-AI; ApoA I; ApoA-I; APOA1_HUMAN; Apolipoprotein A-I(1-242); Apolipoprotein A1; Apolipoprotein A 1; Apolipoprotein AI; Apolipoprotein A I; Brp14; Ltw1; Lvtw1; Sep1; Sep2.
Research Area	Cardiovascular immunology Diabetes Lipoprotein
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	Mouse,Rat (predicted:Human)
Applications	WB=1:500-2000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Theoretical molecular weight	28kDa
Cellular localization	Secretory protein
Form	Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human APOA1: 51-150/267
Lsotype	IgG
Purification	affinity purified by Protein A
Buffer Solution	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Attention	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
PubMed	PubMed
Product	This gene encodes apolipoprotein A-I, which is the major protein component of high

Detail

density lipoprotein (HDL) in plasma. The protein promotes cholesterol efflux from tissues to the liver for excretion, and it is a cofactor for lecithin cholesterolacyltransferase (LCAT) which is responsible for the formation of most plasma cholesteryl esters. This gene is closely linked with two other apolipoprotein genes on chromosome 11. Defects in this gene are associated with HDL deficiencies, including Tangier disease, and with systemic non-neuropathic amyloidosis. [provided by RefSeq, Jul 2008]

Function:
Participates in the reverse transport of cholesterol from tissues to the liver for excretion by promoting cholesterol efflux from tissues and by acting as a cofactor for the lecithin cholesterol acyltransferase (LCAT). As part of the SPAP complex, activates spermatozoa motility.

Subunit:
Interacts with APOA1BP and CLU. Component of a sperm activating protein complex (SPAP), consisting of APOA1, an immunoglobulin heavy chain, an immunoglobulin light chain and albumin. Interacts with NDRG1.

Subcellular Location:
Secreted.

Tissue Specificity:
Major protein of plasma HDL, also found in chylomicrons. Synthesized in the liver and small intestine. The oxidized form at Met-110 and Met-136 is increased in individuals with increased risk for coronary artery disease, such as in carrier of the eNOSa/b genotype and exposure to cigarette smoking. It is also present in increased levels in aortic lesions relative to native ApoA-I and increased levels are seen with increasing severity of disease.

Post-translational modifications:
Palmitoylated.
Met-110 and Met-136 are oxidized to methionine sulfoxides.
Phosphorylation sites are present in the extracelllular medium.

DISEASE:
Defects in APOA1 are a cause of high density lipoprotein deficiency type 2 (HDLD2) [MIM:604091]; also known as familial hypoalphalipoproteinemia (FHA). Inheritance is autosomal dominant.
Defects in APOA1 are a cause of the low HDL levels observed in high density lipoprotein deficiency type 1 (HDLD1) [MIM:205400]; also known as analphalipoproteinemia or Tangier disease (TGD). HDLD1 is a recessive disorder characterized by the absence of plasma HDL, accumulation of cholesteryl esters, premature coronary artery disease, hepatosplenomegaly, recurrent peripheral

neuropathy and progressive muscle wasting and weakness. In HDLD1 patients, ApoA-I fails to associate with HDL probably because of the faulty conversion of pro-ApoA-I molecules into mature chains, either due to a defect in the converting enzyme activity or a specific structural defect in Tangier ApoA-I.
Note=A mutation in APOA1 is the cause of amyloid polyneuropathy-nephropathy Iowa type (AMYLIOWA); also known as amyloidosis van Allen type or familial amyloid polyneuropathy type III. AMYLIOWA is a hereditary generalized amyloidosis due to deposition of amyloid mainly constituted by apolipoprotein A1. The clinical picture is dominated by neuropathy in the early stages of the disease and nephropathy late in the course. Death is due in most cases to renal amyloidosis. Severe peptic ulcer disease can occurr in some and hearing loss is frequent. Cataracts is present in several, but vitreous opacities are not observed.
Defects in APOA1 are a cause of amyloidosis type 8 (AMYL8) [MIM:105200]; also known as systemic non-neuropathic amyloidosis or Ostertag-type amyloidosis. AMYL8 is a hereditary generalized amyloidosis due to deposition of apolipoprotein A1, fibrinogen and lysozyme amyloids. Viscera are particularly affected. There is no involvement of the nervous system. Clinical features include renal amyloidosis resulting in nephrotic syndrome, arterial hypertension, hepatosplenomegaly, cholestasis, petechial skin rash.

Similarity:
Belongs to the apolipoprotein A1/A4/E family.

SWISS:
P02647

Gene ID:
335

Database links:

Entrez Gene: 335 Human

Omim: 107680 Human

SwissProt: P02647 Human

Unigene: 93194 Human

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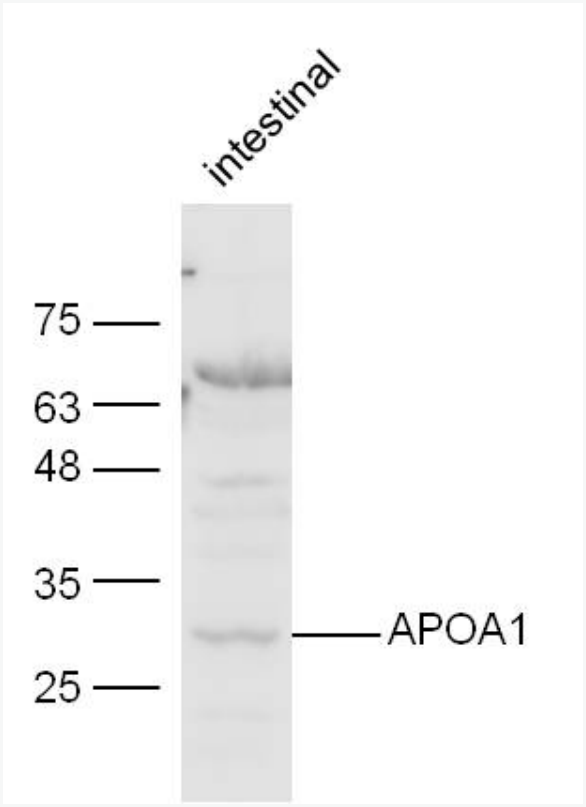
Product Picture



Sample:

intestinal (Mouse) Lysate at 40 ug

Primary: Anti-APOA1 (SL0849R) at 1/300 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 28 kD

Observed band size: 28 kD

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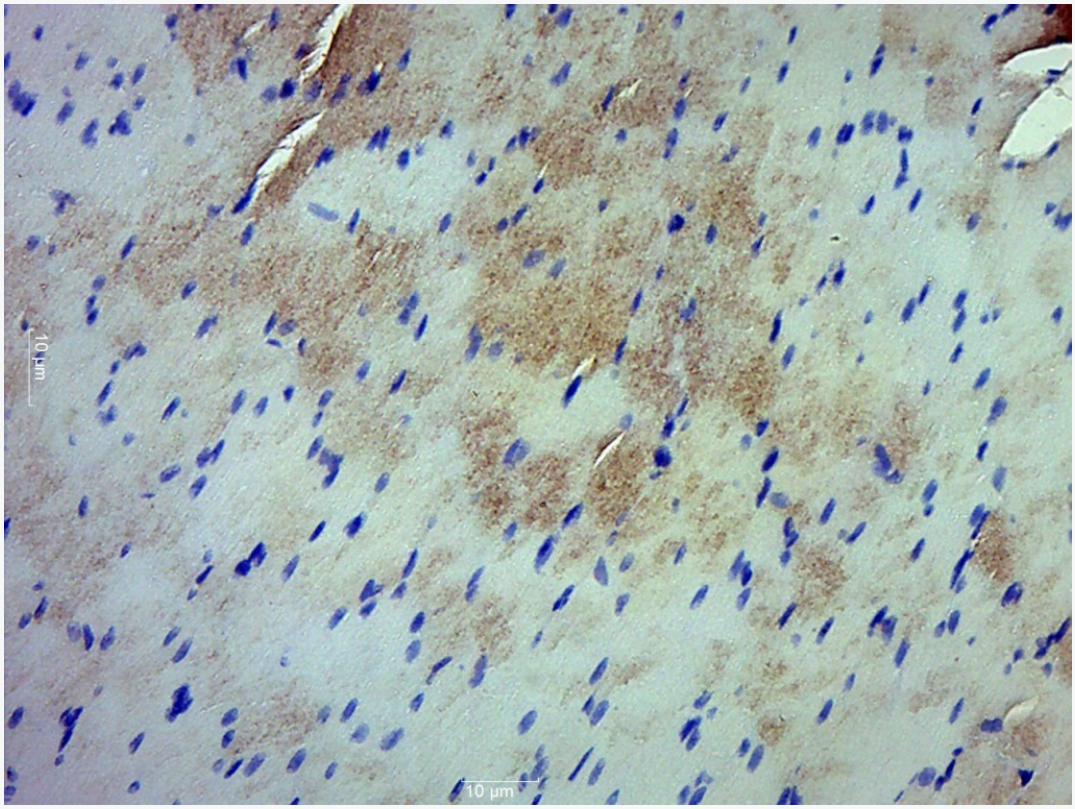

Paraformaldehyde-fixed, paraffin embedded (Rat skeletal muscle); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (APOA1) Polyclonal Antibody, Unconjugated (SL0849R) at 1:500 overnight at 4°C, followed by a conjugated secondary (sp-0023) for 20 minutes and DAB staining.

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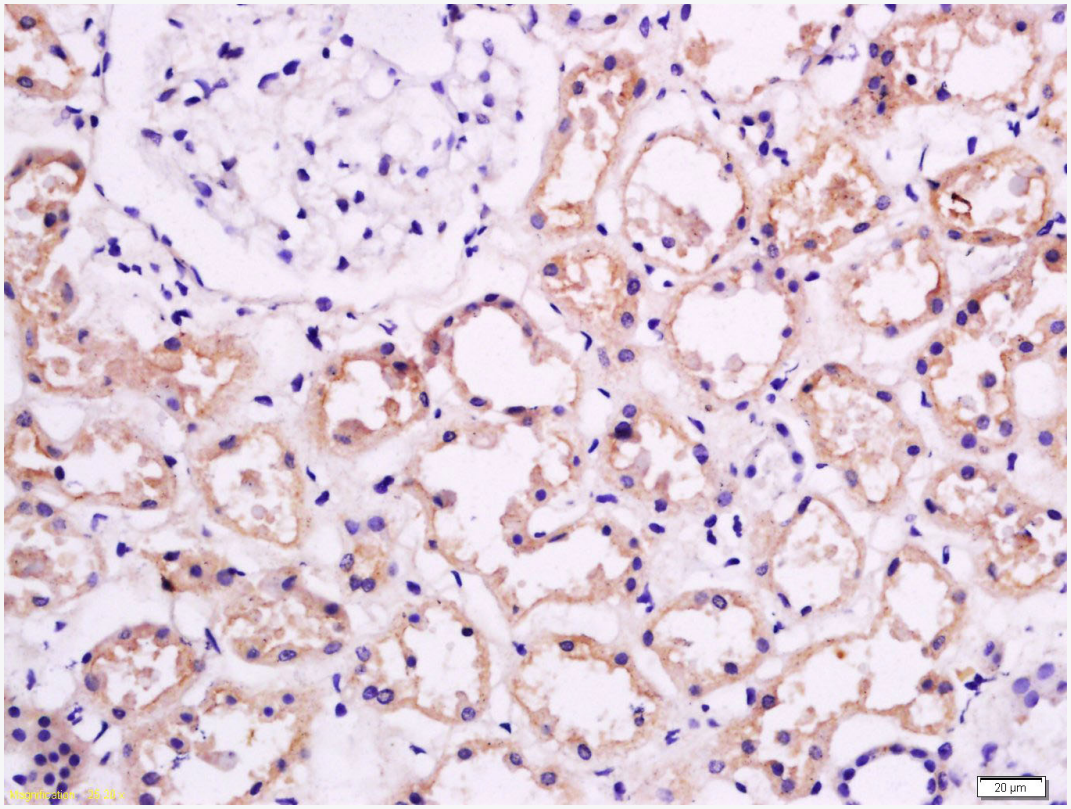

Tissue/cell: Human kidney tissue; 4% Paraformaldehyde-fixed and paraffin-embedded;

Antigen retrieval: citrate buffer (1M, pH 6.0), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37℃ for 20 min;

Incubation: Anti-APOA1 Polyclonal Antibody, Unconjugated(SL0849R) 1:400, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining

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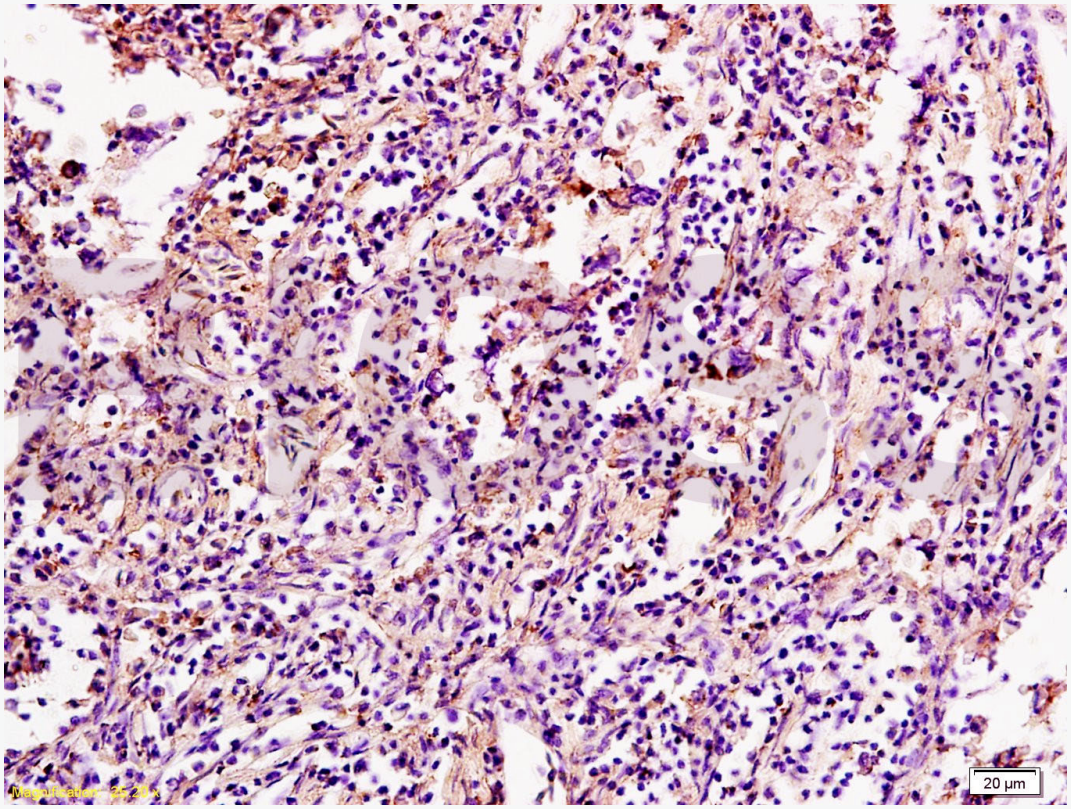

Tissue/cell: chicken intestine tissue; 4% Paraformaldehyde-fixed and paraffin-embedded;

Antigen retrieval: citrate buffer (1M, pH 6.0), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37℃ for 20 min;

Incubation: Anti-APOA1 Polyclonal Antibody, Unconjugated(SL0849R) 1:200, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining

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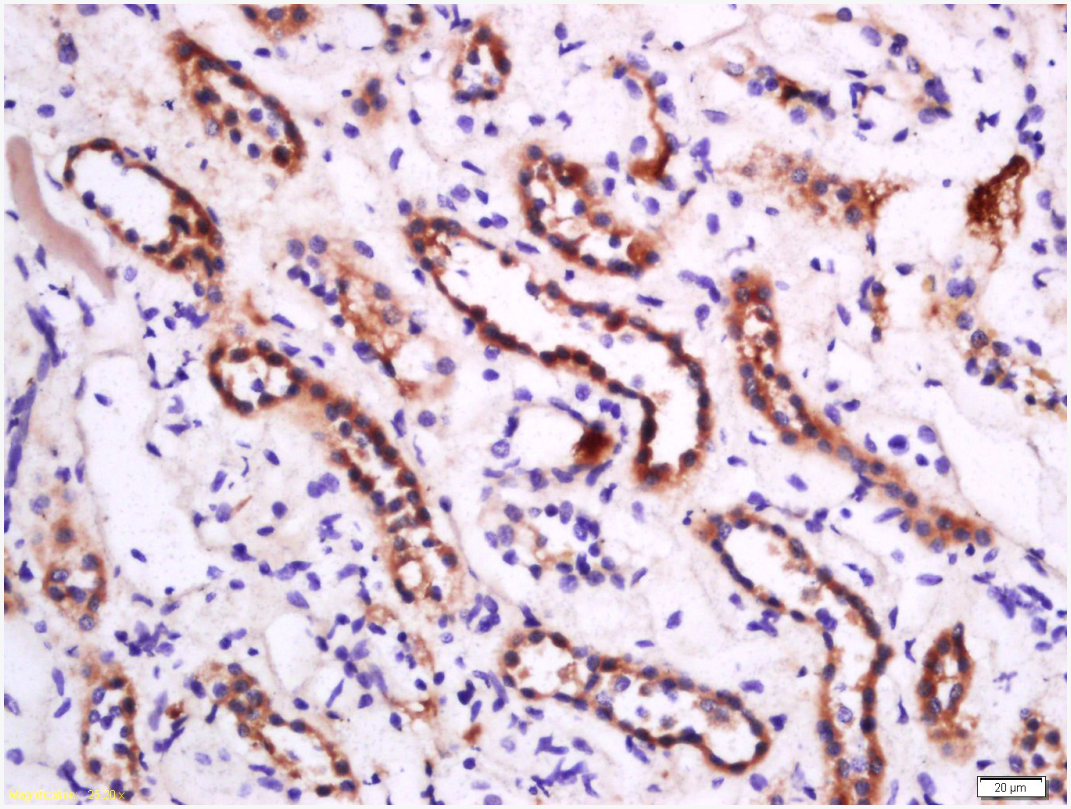
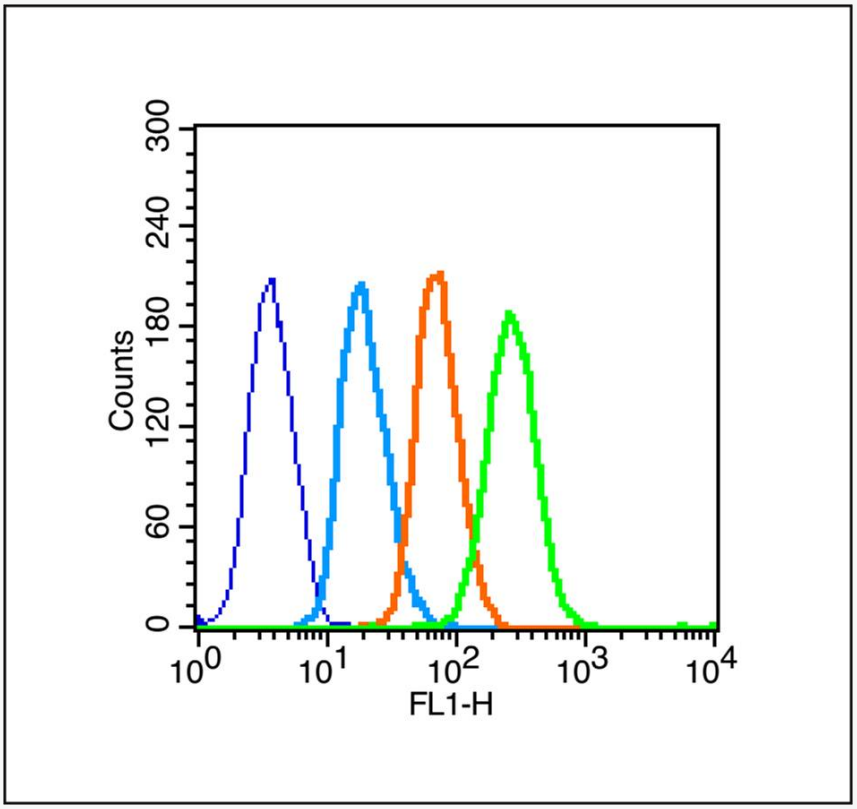


Tissue/cell: human kidney tissue; 4% Paraformaldehyde-fixed and paraffin-embedded;

Antigen retrieval: citrate buffer (1M, pH 6.0), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37℃ for 20 min;

Incubation: Anti-APOA1 Polyclonal Antibody, Unconjugated(SL0849R) 1:200, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining



Blank control (Black line): Raji (Black).

Primary Antibody (green line): Rabbit Anti-APOA1 antibody (SL0849R)

Dilution: 1μg /10^6 cells;

Isotype Control Antibody (orange line): Rabbit IgG .

Secondary Antibody (white blue line): Goat anti-rabbit IgG-PE

Dilution: 1μg /test.

Protocol

The cells were fixed with 70% ice-cold methanol overnight at -20℃ and then permeabilized with 0.1% PBS-Tween for 20 min at room temperature. Cells stained with Primary Antibody for 30 min at room temperature. The cells were

then incubated in 1 X PBS/2%BSA/10% goat serum to block non-specific protein-protein interactions followed by the antibody for 15 min at room temperature. The secondary antibody used for 40 min at room temperature. Acquisition of 20,000 events was performed.