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Product datasheet

Anti-Apolipoprotein A I antibody ab7614

5 Abreviews 15 References

Overview

Product name	Anti-Apolipoprotein A I antibody
Description	Goat polyclonal to Apolipoprotein A I
Host species	Goat
Specificity	Typically less than 1% cross reactivity against other types of apoLipoprotein was detected by ELISA. This antibody reacts with mouse apoLipoprotein A-I and has negligible cross-reactivity with Type A-II, B, C-I, C-II, C-III, E and J apoLipoproteins.
Tested applications	**Suitable for:** ELISA, IP, IHC-P, Sandwich ELISA, WB
Species reactivity	**Reacts with:** Mouse
Immunogen	Full length native apoLipoprotein Type A-I (purified).
General notes	This antibody has been used to determine that atherosclerotic lesions in the human aorta contain considerable amounts of lipoproteins. These lipoproteins were observed to be complexed with components of the extracellular matrix (especially LDL and proteoglycans). The role of these matrix-lipoprotein complexes is not entirely clear, however, animal models of atherosclerosis have shown that increased cellular proliferation and increased production of extracellular matrix components occur following injury to the intimal layer of the aorta. The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing. If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.01% Sodium azide Constituents: 0.44% Sodium chloride, 4.77% Sodium borate, 0.15% EDTA
Purity	Immunogen affinity purified

Purification notes	This product has been prepared by immunoaffinity chromatography using immobilized antigens followed by extensive cross-adsorption against other apoLipoproteins and human serum proteins to remove any unwanted specificities.
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab7614 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
ELISA		Use at an assay dependent concentration.
IP		Use at an assay dependent concentration.
IHC-P	★★★★★ (1)	Use at an assay dependent concentration.
Sandwich ELISA		Use at an assay dependent concentration.
WB	★★★☆☆ (3)	Use at an assay dependent concentration.

Target

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.
Tissue specificity	Major protein of plasma HDL, also found in chylomicrons. Synthesized in the liver and small intestine.
Involvement in 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. Defects in APOA1 are the cause of amyloid polyneuropathy-nephropathy Iowa type (AMYLIOWA) [MIM:107680]; 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.
Sequence similarities	Belongs to the apolipoprotein A1/A4/E family.
Post-translational modifications	Palmitoylated. Phosphorylation sites are present in the extracelllular medium.
Cellular localization	Secreted.

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