

Anti-Apolipoprotein A I antibody [G2] ab58924

1 References

Overview

Product name	Anti-Apolipoprotein A I antibody [G2]
Description	Mouse monoclonal [G2] to Apolipoprotein A I
Host species	Mouse
Specificity	This antibody reacts with both free human Apolipoprotein AI and High Density Lipoprotein (HDL) bearing Apolipoprotein AI, but does not cross react with Apolipoprotein E, B or Albumin.
Tested applications	Suitable for: ELISA, IHC-Fr, WB
Species reactivity	Reacts with: Human
Immunogen	Apolipoprotein AI from human plasma.
General notes	Affinity: Kd = 3nM (for human apolipoprotein A1). 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. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
Storage buffer	Constituents: 1% Dextran, 1% Mannitol, 0.381% Sodium borate, 0.164% Sodium phosphate, 0.87% Sodium chloride And salts
Purity	Protein A purified
Clonality	Monoclonal
Clone number	G2
Isotype	IgG1

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab58924 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.
IHC-Fr		Use at an assay dependent concentration.
WB		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 occur 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 extracellular medium.

Cellular localization

Secreted.

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