

APOA1 Rabbit pAb

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Catalog # AP52072

Product Information

Application	WB
Primary Accession	P02647
Reactivity	Rat, Mouse
Predicted	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	30778
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human APOA1
Epitope Specificity	51-150/267
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Secreted.
DISEASE	Defects in APOA1 are a cause of high density lipoprotein deficiency type 2 (HDL2) [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 (HDL1) [MIM:205400]; also known as analphalipoproteinemia or Tangier disease (TGD). HDL1 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 HDL1 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. This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Important Note	

Background Descriptions

This gene encodes apolipoprotein A-I, which is the major protein component of high 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]

Additional Information

Gene ID	335
Other Names	Apolipoprotein A-I, Apo-AI, ApoA-I, Apolipoprotein A1, Proapolipoprotein A-I, ProapoA-I, Truncated apolipoprotein A-I, Apolipoprotein A-I(1-242), APOA1 (HGNC:600)
Dilution	WB=1:500-2000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	APOA1 (HGNC:600)
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.
Cellular Location	Secreted.
Tissue Location	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

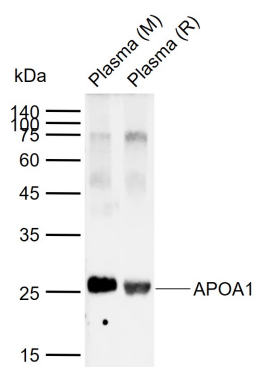
Background

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References

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Cheung P.,et al.Nucleic Acids Res. 11:3703-3715(1983).
Karathanasis S.K.,et al.Proc. Natl. Acad. Sci. U.S.A. 80:6147-6151(1983).
Seilhamer J.J.,et al.DNA 3:309-317(1984).
Sharpe C.R.,et al.Nucleic Acids Res. 12:3917-3932(1984).

Images



Sample:

Lane 1: Mouse Plasma

Lane 2: Rat Plasma

Primary: Anti-APOA1 (AP52072) at 1/1000 dilution

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

Predicted band size: 28 kDa

Observed band size: 27 kDa

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.