

Lecithin-Cholesterol Acyltransferase Human HEK293

Product Data Sheet

Type: Recombinant
Source: HEK293
Species: Human
Other names: LCAT, Phosphatidylcholine-sterol acyltransferase, Phospholipid-cholesterol acyltransferase

Cat. No.:
 RD172122100-HEK (0.1 mg)

Description

Total 429 AA. MW: 48.5 kDa (calculated). UniProtKB/Swiss-Prot P04180 Phe25-Glu440 with N-Terminal Flag-tag, 13 extra AA (highlighted).

Introduction to the Molecule

Human Lecithin: cholesterol acyltransferase (LCAT) is a glycoprotein with a molecular mass of approximately 58 kDa. It is the key enzyme responsible for esterification of free cholesterol to cholesteryl esters in circulating plasma lipoproteins, primarily in high density lipoprotein (HDL). The tertiary structure of LCAT is maintained by two disulfide bridges, similar to lipases and other proteins of the alpha/beta hydrolase fold family. Mature LCAT protein is synthesized from a 440 residue precursor by following cleavage of a 24 residue signal peptide. The mature protein contains 416 amino acids and is heavily N-glycosylated. LCAT is abundant in blood plasma and it is present in other organs, including liver, brain and testes. In plasma LCAT is associated with ApoD which frequently co-purify. A recent study suggests that LCAT can act as an antioxidant and prevent the accumulation of oxidized lipid in plasma lipoproteins. LCAT performs a central role in HDL metabolism by catalyzing the formation of cholesteryl esters on HDL through the transfer of fatty acids from the sn-2 positions of phosphatidylcholine (PC) to cholesterol. The role of LCAT in atherosclerosis is unclear. Dullaart et al. showed that plasma LCAT activity is elevated in metabolic syndrome and may be a marker of subclinical atherosclerosis. Sethi et al. demonstrated that low lecithin-cholesterol acyltransferase (LCAT) activities and high pre- β 1-HDL concentrations are strong positive risk markers for ischemic heart disease and are independent of HDLcholesterol. Miida et al. demonstrated that plasma pre beta1-HDL concentration increase in subjects with low LCAT activity. They also reported that patients with coronary artery disease (CAD) had higher pre- β 1-HDL concentrations than did normolipidemic subjects. Holleboom et al. showed that low plasma LCAT levels (reflecting low LCAT activity) are not associated with an increased risk of future (CAD) in the general population. However, other studies showed a positive association of LCAT levels with carotid atherosclerosis in patients with the metabolic syndrome as well as in control subjects whereas, LCAT activity was reduced in patients with CAD and in patients with acute myocardial infarction. In summary, LCAT activity might be reduced in the acute phase of a myocardial infarction. Mutations of LCAT on chromosome 16 resulting in homozygous or compound heterozygous form can cause two major phenotypes: FLD (familial LCAT deficiency) and FED (Fish Eye Disease). Patients with FLD have a complete loss of both alpha-LCAT activity and beta-LCAT activity and an increased proportion of unesterified cholesterol in plasma. In FED is partial loss of alpha-LCAT activity with normal elevated free cholesterol in plasma. Both FLD and FED are characterized by the development of corneal opacities.

Research topic

Cardiovascular disease, Lipoprotein metabolism

Amino Acid Sequence

HVDYKDDDDK **PAG**FWLLNVL FPPHTTPKAE LSNHTRPVIL VPGCLGNQLE AKLDKPDVVN WMCYRKTEDF FTIWLDLNMF
 LPLGVDCWID NTRVVYNRSS GLVSNAPGVQ IRVPGFGKTY SVEYLDSSKL AGYLHTLVQN LVNNGYVRDE TVRAAPYDWR
 LEPGQQEYY RKLGLVEEM HAAYGKPVFL IGHSLGCLHL LYFLLRQPQA WKDRFIDGFI SLGAPWGGSI KPMLVLASGD
 NQGIPIMSSI KLKEEQRIIT TSPWMFPSRM AWPEDHVFIS TPSFNVTGRD FQRFADLHF EEGWYMWLQS RDLLAGLPAP
 GVEVYCLYGV GLPTPRTYIY DHGFPYTDPV GVLIEDGDDT VATRSTELCG LWQGRQPQPV HLLPLHGIQH LNMVFSNLT
 EHINAILLGA YRQGPASPT ASPEPPPE

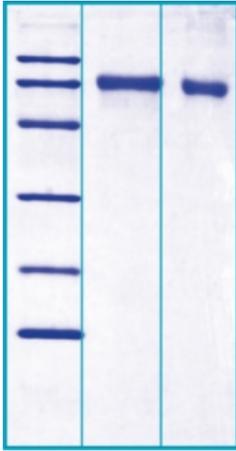
Source

HEK293

Purity

Purity as determined by densitometric image analysis: >95%

SDS-PAGE gel



14% SDS-PAGE separation of Human LCAT

1. M.W. marker - 14, 21, 31, 45, 66, 97 kDa
2. reduced and heated sample, 5µg/lane
3. non-reduced and non-heated sample, 5µg/lane

Endotoxin

< 0.1EU/ug

Formulation

Filtered (0.4 µm) and lyophilized from 0.5 mg/ml in 20mM Tris buffer, 50mM NaCl, pH 7.5

Reconstitution

Add deionized water to prepare a working stock solution of approximately 0.5 mg/mL and let the lyophilized pellet dissolve completely. Product is not sterile! Please filter the product by an appropriate sterile filter before using it in the cell culture.

Shipping

At ambient temperature. Upon receipt, store the product at the temperature recommended below.

Storage, Stability/Shelf Life

Store lyophilized protein at -80°C. Lyophilized protein remains stable until the expiry date when stored at -80°C. Aliquot reconstituted protein to avoid repeated freezing/thawing cycles and store at -80°C for long term storage. Reconstituted protein can be stored at 4°C for a limited period of time; it does not show any change after one week at 4°C.

Quality Control Test

BCA to determine quantity of the protein.

SDS PAGE to determine purity of the protein.

LAL to determine quantity of endotoxin.

Applications

Cell culture and/or animal studies, ELISA, Western blotting

Note

This product is intended for research use only. The Certificate of Analysis is available on www.biovendor.com

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