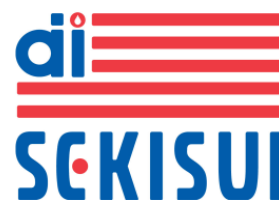


Lecithin-cholesterol acyltransferase (LCAT)



Description

High density lipoprotein (HDL) plays an important role in reverse cholesterol transport, a process by which excess cholesterol from peripheral tissues is returned to the liver for use or excretion.

There is a strong inverse correlation between plasma HDL cholesterol concentration and the incidence of atherosclerosis. Lecithin-cholesterol acyltransferase (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.

Indication

- Atherosclerosis
- LCAT deficiency syndrome

Pathophysiology

Genetic deficiencies of human LCAT have been recently reviewed by Kuivenhoven et al. Briefly, two classes of genetic deficiencies are known: familial LCAT deficiency (FLD) and fish-eye disease (FED). FLD is caused by either null or missense mutations. FED is caused by missense mutations only. Direct measurement of the enzyme mass and activity may contribute to the differentiation of LCAT defects.

The role of LCAT in atherosclerosis is not clearly established, studies have yielded conflicting results. 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 independent of HDL-cholesterol. 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. Other studies showed a positive association of LCAT levels with carotid atherosclerosis in patients with the metabolic syndrome as well as in control subjects, LCAT activity was reduced in patients with CAD and in patients with acute myocardial infarction. It can be reasoned that LCAT activity might be reduced in the acute phase of a myocardial infarction, but may normalize over time.

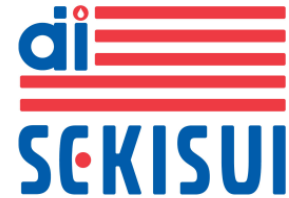
A specific and sensitive enzyme immunoassay for human plasma and serum LCAT is a useful tool to clarify the role of LCAT and to clinically investigate LCAT deficiency syndrome.

References

- Lecithin Cholesterol Acyltransferase: An anti- or pro-atherogenic Factor? Rousset X et al., Curr Athero Rep. 2011 Feb 18. [Epub ahead of print]
- High pre-beta1 HDL concentrations and low lecithin: cholesterol acyltransferase activities are strong positive risk markers for ischemic heart disease and independent of HDL-cholesterol. Sethi AA et al., Clin Chem 2010 Jul;56(7):1128-1137.
- Plasma levels of lecithin:cholesterol acyltransferase and risk of future coronary artery disease in apparently healthy men and women: a prospective case-control analysis nested in the EPIC-Norfolk population study. Holleboom AG et al., J Lipid Res. 2010 Feb;51(2):416-421.
- Plasma lecithin: cholesterol acyltransferase activity is elevated in metabolic syndrome and is an independent marker of increased carotid artery intima media thickness. Dullaart RP et al., J Clin Endocrinol Metab. 2008 Dec;93(12):4860-4866.
- Lecithin-cholesterol acyltransferase activity in patients with coronary artery disease examined by coronary angiography. Solajić-Bozicević N et al., Clin Investig. 1994 Dec;72(12):951-6.
- Reverse cholesterol transport: a review of the process and its clinical implications. Hill SA and McQueen. J Clin Biochem 1997; 30:517–525.
- Lecithin:cholesterol acyltransferase (LCAT) mass; its relationship to LCAT activity and cholesterol esterification rate. Albers JJ et al., J Lipid Res 1981; 22: 1206–1213.
- The molecular pathology of lecithin:cholesterol acyltransferase (LCAT) deficiency syndromes. Kuivenhoven JA et al. J Lipid Res 1997; 38:191–205.

Product information LCAT ELISAover

LCAT ELISA



Principle of the assay

The LCAT ELISA kit is intended for the quantitative determination of lecithin-cholesterol acyltransferase (LCAT) in human serum and plasma by utilizing a two-step sandwich method of enzyme-linked immuno-sorbent assay (ELISA).

Test wells are coated with anti-LCAT mAb (clone 36486, epitope C-terminus of LCAT), which binds with LCAT in the sample. After the first incubation and washes to remove all of the unbound material, horseradish peroxidase (HRP)-labeled anti-LCAT mAb (clone 36487, epitope located in the center of the LCAT primary structure) is added. The enzyme labeled mAb (36487) binds with LCAT immobilized on the well by the coated mAb (36486). After the second incubation and subsequent washes, the antibody / LCAT / enzyme complex is incubated with a substrate solution and terminated with a stop reagent. The intensity of color that develops in the enzyme reaction is measured by using a microplate reader. The absorbance is proportional to the concentration of LCAT in the sample.

A strong correlation between LCAT concentration (using the ELISA) and LCAT activity (as assessed with a liposome substrate) measurements in plasma has been demonstrated.

References

- A new enzyme-linked immunosorbent assay with two monoclonal antibodies to specific epitopes measures human lecithin-cholesterol acyltransferase. Kobori K et al. J Lipid Res, 2002; 43: 325-334.

Key Features

- **Format:** 96-well plate
2- step sandwich ELISA
- **Sample type:** human plasma and serum
- **Reference range:** 5.0 ~ 10.3 µg/ml (n = 60)
- **Linearity:** 0.5 ~ 35 µg/ml
- **Sensitivity:** 0 µg/ml ≤ 0.15 Abs
7.5 µg/ml 0.2 ~ 0.8 Abs
- **Specificity:** 85 ~ 115% of expected value
- **Reproducibility:** CV value less than 10%
- **Shelf life:** 24 months



[Scientific information on LCATover](#)