

Product datasheet

LCAT Activity Assay Kit ab242306

2 Images

Overview

Product name	LCAT Activity Assay Kit
Detection method	Fluorescent
Sample type	Serum, Plasma, Cell Lysate, Tissue Lysate
Product overview	The LCAT Activity Assay Kit (ab242306) is a simple, fluorometric assay that quantitatively measures LCAT phospholipase activity in plasma, serum, and lysates in a 96-well microtiter plate format.

Each kit provides sufficient reagents to perform up to 100 assays, including blanks and unknown samples. Besides LCAT, this assay can also be used to detect other calcium independent phospholipase activities such as lipoprotein phospholipase A2 (LP-PLA2).

Properties

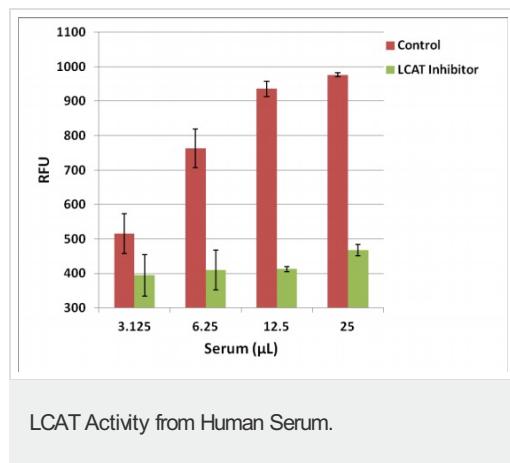
Storage instructions	Store at -20°C. Please refer to protocols.
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Components	
LCAT Fluorometric Substrate	1 x 50µl
10X LCAT Assay Buffer	2 x 1.5ml
2X Stop Solution	1 x 12ml
50X LCAT Inhibitor	1 x 100µl

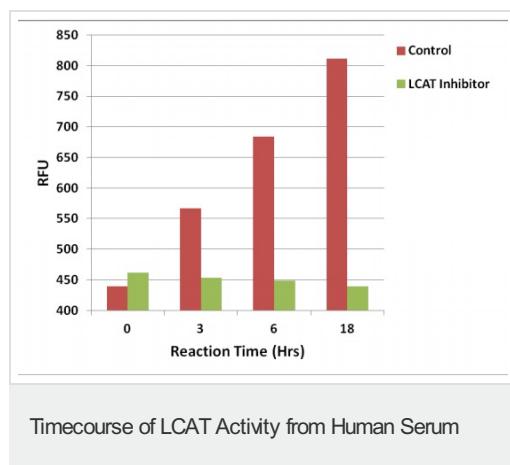
Function	Central enzyme in the extracellular metabolism of plasma lipoproteins. Synthesized mainly in the liver and secreted into plasma where it converts cholesterol and phosphatidylcholines (lecithins) to cholesteryl esters and lysophosphatidylcholines on the surface of high and low density lipoproteins (HDLs and LDLs). The cholesterol ester is then transported back to the liver. Has a preference for plasma 16:0-18:2 or 18:0-18:2 phosphatidylcholines. Also produced in the brain by primary astrocytes, and esterifies free cholesterol on nascent APOE-containing lipoproteins secreted from glia and influences cerebral spinal fluid (CSF) APOE- and APOA1 levels. Together with APOE and the cholesterol transporter ABCA1, plays a key role in the maturation of glial-derived, nascent lipoproteins. Required for remodeling high-density lipoprotein particles into their spherical forms.
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Tissue specificity	Expressed mainly in brain, liver and testes. Secreted into plasma and cerebral spinal fluid. In liver, expressed in HEPG2 hepatocytes.
Involvement in disease	Defects in LCAT are the cause of lecithin-cholesterol acyltransferase deficiency (LCATD) [MIM:245900]; also called Norum disease. LCATD is a disorder of lipoprotein metabolism characterized by inadequate esterification of plasmatic cholesterol. Two clinical forms are recognized: familial LCAT deficiency and fish-eye disease. Familial LCAT deficiency is associated with a complete absence of alpha and beta LCAT activities and results in esterification anomalies involving both HDL (alpha-LCAT activity) and LDL (beta-LCAT activity). It causes a typical triad of diffuse corneal opacities, target cell hemolytic anemia, and proteinuria with renal failure. Defects in LCAT are a cause of fish-eye disease (FED) [MIM:136120]; also known as dyslipoproteinemic corneal dystrophy or alpha-LCAT deficiency. FED is due to a partial LCAT deficiency that affects only alpha-LCAT activity. It is characterized by low plasma HDL and corneal opacities due to accumulation of cholesterol deposits in the cornea ('fish-eye').
Sequence similarities	Belongs to the AB hydrolase superfamily. Lipase family.
Post-translational modifications	O- and N-glycosylated. O-glycosylation on Thr-431 and Ser-433 consists of sialylated galactose beta 1-->3N-acetylgalactosamine structures. N-glycosylated sites contain sialylated triantennary and/or biantennary complex structures.
Cellular localization	Secreted. Secreted into blood plasma. Produced in astrocytes and secreted into cerebral spinal fluid.

Images



Various amounts of normal human serum samples were either untreated (control) or pretreated with LCAT Inhibitor (2 mM IAA) before incubation with LCAT substrate for 18 hrs at 37°C. The LCAT activity was determined as described in the Assay Protocol



25 μL of normal human serum samples were either untreated (control) or pretreated with LCAT Inhibitor (2 mM IAA) before incubation with LCAT substrate at 37°C. The LCAT activity was determined as described in the Assay Protocol.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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