Lp-PLA$_2$ Activity

CPT Code **83698***
Order Code **C570**
Sample Type **EDTA Plasma or Serum**
Tube Type **Lavender Top or Tiger Top**
LCD-CGS **L36139**

Increased activity of Lp-PLA$_2$ may lead to increased risk of:

- Coronary heart disease (CHD)
- Myocardial infarction (MI)

Lp-PLA$_2$ Activity can be reduced by:

- Treatment with lipid-lowering therapies
- Increased omega-3 fatty acid
- Lifestyle modifications

Description

Lp-PLA$_2$, or lipoprotein-associated phospholipase-A$_2$, measures disease activity within the artery wall below the collagen or calcified cap due to the activation of macrophages. Lp-PLA$_2$ is not an acute-phase reactant. When disease is active in the artery, increased levels of Lp-PLA$_2$ are produced by macrophages and foam cells within the intima of the artery.[1] Lp-PLA$_2$ also interacts with oxidized low-density lipoprotein (oxLDL), which increases inflammation and enhances a proatherogenic state, as well as plaque vulnerability.[2]

Clinical Use

The Lp-PLA$_2$ Activity test may be performed on individuals at intermediate or high risk for developing cardiovascular disease (CVD).

Clinical Significance

- Lp-PLA$_2$ accumulates within human atherosclerotic plaques and vulnerable lesions.[3]
- Individuals with elevated Lp-PLA$_2$ Activity are nearly twice as likely to develop CHD at 7 years regardless of non-high-density lipoprotein cholesterol levels.[4]
- Individuals with elevated Lp-PLA$_2$ Activity are twice as likely to experience a CHD event (MI, coronary revascularization or CHD-related death) at 5 years.[5]

Testing Frequency

Lp-PLA$_2$ testing is determined by an individual's medical history, but may be performed semi-annually or annually as necessary. If the initial test result is abnormal, then follow-up testing may be performed within 3-6 months following treatment.

Sample Type

The Lp-PLA$_2$ Activity test should be performed on a serum or EDTA plasma sample. Fasting is not required.

Commercial Insurance or Medicare Coverage

Coverage guidelines, also known as NCD (National Coverage Determination) or LCD (Local Coverage Determination), have been established or posted by CMS (Medicare & Medicaid). Guidelines should be reviewed for coverage and limitations. Limited information has been provided by the majority of the larger carriers (Aetna, United HealthCare, Cigna, Blues).

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RELATIVE RISK

Lp-PLA$_2$ Activity
(nmol/min/mL)

<75 Low	≥75 High

Treatment Considerations†

These treatment considerations are for educational purposes only. Specific treatment plans should be provided and reviewed by the treating practitioner.

✓ **Assess lifestyle habits.**

- Consider diet/exercise/weight reduction efforts if appropriate.[6]

✓ **Assess LDL-C levels.**

- If not at an optimal level, consider lipid-lowering therapies described in the National Cholesterol Education Program/Adult Treatment Panel III (NCEP ATP III) Guidelines.[7-9]

✓ **Assess blood pressure.**

- If not at an optimal level, consider initiating or titrating antihypertensive therapy.[10]

✓ **Assess omega-3 fatty acid levels.**

- If not at an optimal level, consider fish oil supplements, other dietary supplements, and dietary recommendations for increasing omega-3 fatty acid levels.[11,12]

Assess the presence of coronary artery disease (CAD) with imaging techniques such as carotid intima-media thickness (CIMT) testing.[13]

✓ **Assess clotting risk.**

- Consider antiplatelet therapy if history of CAD (i.e., myocardial infarction or revascularization) and/or a history of cerebrovascular disease (i.e., transient ischemic attack or stroke).[14]

✓ **Assess dental health (periodontal disease).**

- Refer to dentist to identify gum disease. Poor dental health may cause significant inflammation and is associated with the presence of atherosclerosis.[15,16]

** The CPT codes provided are based on AMA guidelines and are for informational purposes only. CPT coding is the sole responsibility of the billing party. Please direct any questions regarding coding to the payer being billed.*

† The treatment considerations are provided for informational purposes only and are not intended as medical advice. A physician's test selection and interpretation, diagnosis, and patient management decisions should be based on his/her education, clinical expertise, and assessment of the patient.

References

1. Ferguson JF, Hinkle CC, Mehta NN, et al. Translational studies of lipoprotein-associated phospholipase A(2) in inflammation and atherosclerosis. *J Am Coll Cardio*. 2012; 59: 764-772. 2. Gonçalves I, Edsfeldt A, Ko NY, et al. Evidence supporting a key role of Lp-PLA$_2$-generated lysophosphatidylcholine in human atherosclerotic plaque inflammation. *Arterioscler Thromb Vasc Biol*. 2012; 32: 1505-1512. 3. Kolodgie FD, Burke AP, Skorija KS, et al. Lipoprotein-associated phospholipase A2 protein expression in the natural progression of human coronary atherosclerosis. *Arterioscler Thromb Vasc Biol*. 2006; 26: 2523-2529. 4. Oei HS, van der Meer IM, Hofman A, et al. Lipoprotein-associated phospholipase A2 activity is associated with risk of coronary heart disease and ischemic stroke: The Rotterdam Study. *Circulation*. 2005; 111: 570-575. 5. Cushman M, Judd S, Kissela B, et al. Lipoprotein-associated phospholipase A2 (Lp-PLA$_2$) activity and coronary heart disease risk in a biracial cohort: The reasons for geographic and racial differences in stroke (REGARDS) Cohort. *Atherosclerosis*. 2015; 241: e1-e31. 6. Hatoum IJ, Nelson JJ, Cook NR, Hu FB, Rimm EB. Dietary, lifestyle, and clinical predictors of lipoprotein-associated phospholipase A2 activity in individuals without coronary artery disease. *Am J Clin Nutr*. 2010; 91(3):786-793. 7. Davidson MH, Corson MA, Albers MJ, et al. Consensus Panel recommendation for Incorporating Lipoprotein-Associated Phospholipase A2 Testing into Cardiovascular Disease Risk Assessment Guidelines. *Am J Cardiol*. 2008;101(suppl):51F-57F. 8. Schaefer EJ, McNamara JR, Asztalos BF, et al. Effects of atorvastatin versus other statins on fasting and postprandial C-reactive protein and lipoprotein-associated phospholipase A2 in patients with coronary heart disease versus control subjects. *Am J Cardiol*. 2005; 95(9):1025-1032. 9. Third report of the National Cholesterol Education Program (NCEP). Expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *National Institutes of Health*. September 2002. NIH Publication No. 02-5215. 10. Kim M, Yoo HJ, Kim M, et al. Associations among oxidative stress, Lp-PLA$_2$ activity and arterial stiffness according to blood pressure status at a 3.5-year follow-up in subjects with prehypertension. *Atherosclerosis*. 2017; 257: 179-185. 11. Davidson MH, Stein EA, Bays HE, et al. Efficacy and tolerability of adding prescription omega-3 fatty acids 4 g/d to simvastatin 40 mg/d in hypertriglyceridemic patients: An 8-week, randomized, double-blind, placebo-controlled study. *Clin Ther*. 2007; 39: 1354-1367. 12. Gajos G, Zalewski J, Mostowik M, Konduracka E, Nessler J, Undas A. Polyunsaturated omega-3 fatty acids reduce lipoprotein-associated phospholipase A(2) in patients with stable angina. *Nutr Metab Cardiovasc Dis*. 2014; 24(4): 434-439. 13. Ikonomidis I, Kadoglou NNP, Tritakis V, et al. Association of Lp-PLA$_2$ with digital reactive hyperemia, coronary flow reserve, carotid atherosclerosis and arterial stiffness in coronary artery disease. *Atherosclerosis*. 2014; 234 :34-41. 14. Winkler K, Winkelmann BR, Scharnagl H, et al. Platelet-Activating Factor Acetylhydrolase Activity Indicates Angiographic Coronary Artery Disease Independently of Systemic Inflammation and Other Risk Factors: The Ludwigshafen Risk and Cardiovascular Health Study. *Circulation*. 2005; 111: 980-987. 15. Mochari H, Grbic JT, Mosca L. Usefulness of Self-Reported Periodontal Disease to Identify Individuals With Elevated Inflammatory Markers at Risk of Cardiovascular Disease. *Am J Cardiol*. 2008; 102: 1509-1513. 16. Buhlin K, Mäntylä P, Paju S, Peltola JS, Nieminen MS, Sinisalo J, Pussinen PJ. Periodontitis is associated with angiographically verified coronary artery disease. *J Clin Periodontol*. 2011; 38: 1007-1014.

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