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PLAC – A Specific Marker for Vascular Inflammation

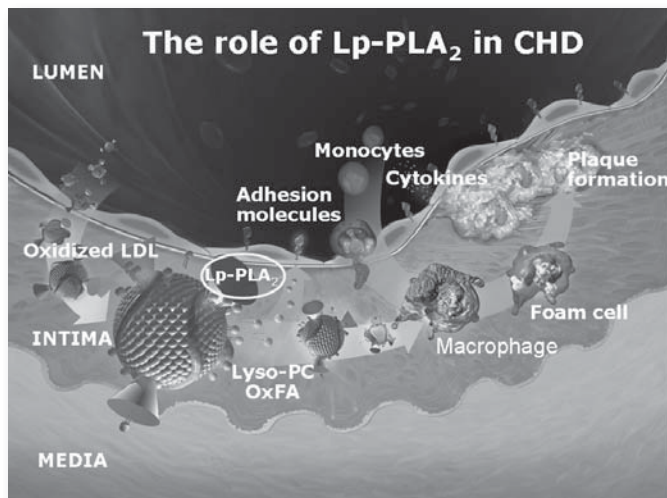
Lawrence M. Killingsworth, Ph.D., DABCC, Chief Science & Technical Officer

Some individuals with cardiovascular disease show normal levels of conventional risk factors, such as lipids. Recent studies have focused on the inflammatory aspects of the atherosclerotic process to provide a more complete picture of cardiovascular risk.

Lipoprotein-associated phospholipase A2 (Lp-PLA₂), also known as PLAC, is a novel risk marker, specific for vascular inflammation. Elevations of PLAC are associated with increased risk for cardiovascular disease and ischemic stroke.

The Role of PLAC in Cardiovascular Disease and Plaque Destabilization

- PLAC migrates with LDL in the circulation. The LDL-PLAC complex passes from the vascular lumen into the intima where LDL is oxidized.
- PLAC hydrolyzes the oxidized LDL to form lysophosphatidylcholine (Lyso-PC) and oxidized fatty acid (OxFA).
- Lyso-PC and OxFA are pro-inflammatory mediators that cause increased production of adhesion molecules and cytokines, which bring monocytes into the intima.
- The monocytes become activated macrophages that engulf the oxidized LDL and become foam cells.
- Foam cells aggregate to form atherosclerotic plaque, which produces cytokines and proteases that degrade the smooth muscle cells and collagen matrix of the fibrous cap.
- The plaque weakens and ruptures, leading to a thrombotic clot.



Clinical Cut Point for PLAC

In a population study of men and women, the central 90% range was found to be 131 to 376 ng/mL for PLAC. Recent published studies have demonstrated an increased risk of CHD and stroke associated with PLAC values in the second and third tertiles (upper 67%) of the studied populations. The recommended cut point for identifying individuals with significant risk is at the 50th percentile value, which corresponds to a cut point of 235 ng/mL.

Quick Facts

- ▶ **PLAC is a specific marker for vascular inflammation**
- ▶ **Elevated PLAC levels are associated with increased risk for CHD and stroke events.**
- ▶ **The recommended cut point for identifying individuals with significant risk attributable to PLAC is 235 ng/mL.**

Ordering PLAC

- ▶ **Clients have the option of ordering PLAC by itself or as part of the new LIPID2 profile which includes; PLAC, Cholesterol, Triglycerides, HDL and LDL (calculated).**
- ▶ **A lipid profile without PLAC (LIPID) can also be ordered.**
- ▶ **PAML's lipid profile conforms to the recommendations of the NIH sponsored National Cholesterol Education Project.**

Paml.com

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For more information, please contact your local representative.

Test Information

DESCRIPTION LPA2

METHOD ELISA

ORDER CODE PLACA2

CPT CODE 83698

SPECIMEN REQUIREMENTS 1 mL frozen serum or plasma (red top tube or PST). Separate serum or plasma from the cells within 4 hours of collection and put in separate plastic tube and freeze immediately. Store and transport frozen.

COMMENTS 1) Min Amt: 0.5 mL.
2) Other acceptable samples: EDTA or heparin plasma (lavender or green top tube).
3) Unacceptable conditions: ambient or unprocessed blood samples. Samples may be frozen and thawed up to six times.
4) Stability: RT-4 hours, Refrigerated-7 days, Frozen(-20C)-3 months. Frozen(-70C)-6 months.RANGES **LP-PLA2** 0-234 ng/mL
Patients with PLAC levels greater than 235 ng/mL are at increased risk for cardiovascular events, including myocardial infarction and stroke.**Test Information**

DESCRIPTION LIPID profile & LPA2 (PLAC)

METHOD Enzymatic, ELISA

ORDER CODE LIPID2

CPT CODE 86001, 83698

SPECIMEN REQUIREMENTS 2 mL refrigerated and 1 mL frozen serum (SST or PST tubes). Separate serum or plasma from cells within 4 hours of collection and put in two separate tubes. Freeze one immediately for LPA2 (PLAC) assay and refrigerate the other one. Store and transport each one appropriately.

COMMENTS 1) Min Amt: 1 mL refrigerated and 0.5 mL frozen.
2) Other acceptable specimens: EDTA or lithium heparin plasma (lavender or green top tube).
3) Unacceptable conditions: ambient or unprocessed blood samples. 4) Stability: RT-4 hours, Refrigerated-7 days, Frozen(-20C)-3 months, Frozen(-70C)-6 months.

RANGES	Cholesterol	LT 200	Desirable	mg/dL
		200-239	Borderline high	
		240 or more	High	
Triglycerides	LT 150	Normal	mg/dL	
	150-199	Borderline high		
	200-499	High		
	500 or more	Very High		
HDL	LT 40	Low	mg/dL	
	40-59	Within normal limits		
	60 or more	High		
LDL (calculated)	LT 100	Optimal	mg/dL	
	100-129	Near or above optimal		
	130-159	Borderline high		
	160-189	High		
	190 or more	Very high		

To calculate 10 year cardiac risk for this patient, go to <http://www.paml.com>, click on Testing, then on ranges/algorithms and then on lipid results.**LDL/HDL Ratio** No longer applicable or reported.**CHO/HDL Ratio** No longer applicable or reported.**LP-PLAC2** 0-234 ng/mL

Patients with PLAC levels greater than 235 ng/mL are at increased risk for cardiovascular events, including myocardial infarction and stroke.

Selected References

1. Garza, CA, et al. Association between lipoprotein-associated phospholipase A2 and cardiovascular disease: A systematic review. *Mayo Clin Proc* 2007;82(2):150-165.
2. Lanman, RB, et al. Lipoprotein-associated phospholipase A2: Review and recommendation of a clinical cut point for adults. *Prev Cardiol* 2006;9:138-43.
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4. Koenig, W, et al. Lipoprotein-associated phospholipase A2 predicts future cardiovascular events in patients with coronary heart disease independently of traditional risk factors, markers of inflammation, renal function and hemodynamic stress. *Arterioscler Thromb Vasc Biol* 2006;26:1-9.
5. Brilakis, ES, et al. Association of Lipoprotein-associated phospholipase A2 levels with coronary artery disease risk factors, angiographic coronary artery disease and major adverse events at follow-up. *Euro Heart J* 2005;26:137-44.
6. Packard, CJ, et al. Lipoprotein-associated phospholipase A2 as an independent predictor of coronary heart disease. *N Engl J Med* 2000;343:1148-55.

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