Lp-PLA$_2$. A new risk factor for CVD.

The PLAC® Test was instrumental in landmark studies confirming the causal pathway between Lp-PLA$_2$ and CVD events.[1-3]


family history


diabetes

elevated Lp-PLA2


obesity


smoker


males aged ≥45 or females ≥55


high cholesterol


high blood pressure

PLAC® Test for Lp-PLA$_2$

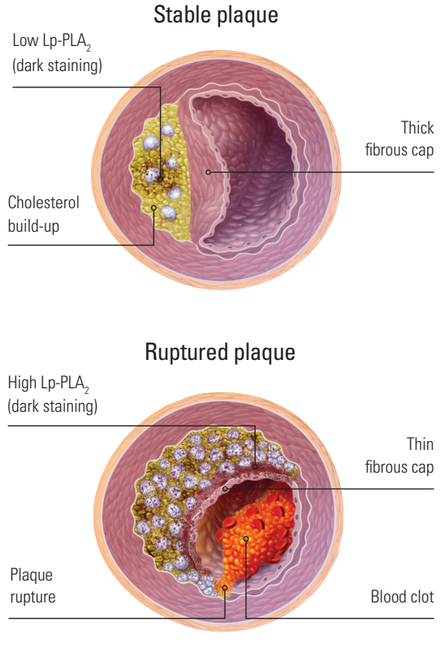
Landmark studies confirm the causal pathway.

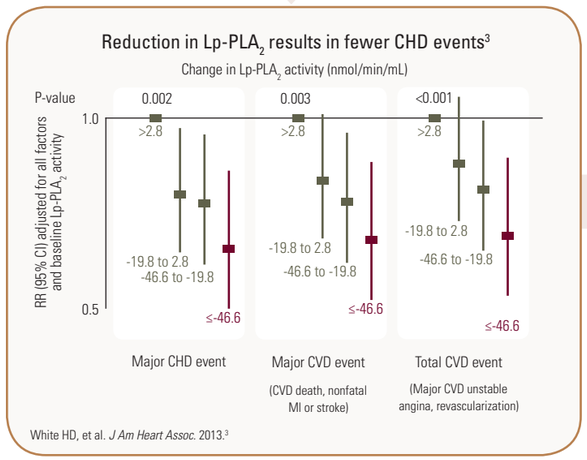
The PLAC® Test clearly identifies the atherosclerotic disease process and is the only evidence-based blood test that measures Lp-PLA$_2$—a vascular-specific inflammatory marker critical in the formation of rupture-prone plaque.[1-5]

The path to plaque rupture.

Lp-PLA$_2$ plays a key role in formation of rupture-prone plaque[5]

- Produced by macrophages in the vascular wall of atherosclerotic arteries, Lp-PLA$_2$ triggers hydrolysis of oxidizing LDL, which releases two highly inflammatory mediators
- When the arterial walls become inflamed, the enzyme Lp-PLA$_2$ produced within the plaque is elevated
- If the amount of Lp-PLA$_2$ is high, this may indicate that the plaque is more likely to rupture, leading to a dangerous blood clot that could result in cardiovascular disease (CVD) events

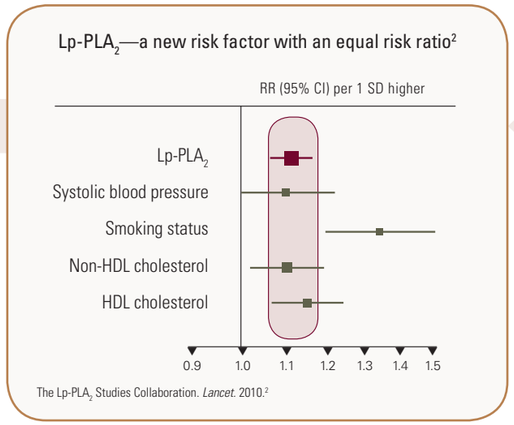




White HD, et al. *J Am Heart Assoc.* 2013.[3]

A natural connection.

New genetic evidence in certain populations shows that a natural deficiency of Lp-PLA$_2$ activity protects against coronary artery disease (CAD). A large-scale study demonstrated carriage of the V279F null allele within the gene encoding Lp-PLA$_2$ is protective from CAD. These results confirm the causal relationship between Lp-PLA$_2$ and CAD.[1]



The Lp-PLA$_2$ Studies Collaboration. *Lancet.* 2010.[2]

A powerful predictor.

A meta-analysis of 79,036 participants in 32 prospective studies demonstrated that Lp-PLA$_2$ was a predictor of coronary heart disease (CHD), with a risk ratio similar in magnitude to that of systolic blood pressure and non-HDL cholesterol.[2]

An independent factor.

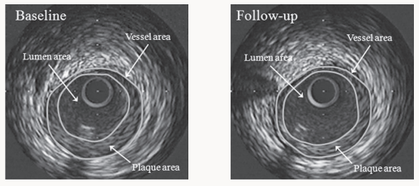
The PLAC® Test was instrumental in the LIPID* sub-study, a long-term outcome study in over 6,500 patients that showed that reduction of Lp-PLA$_2$ activity was a highly significant predictor of reduction in subsequent coronary events.[3]

- Patients who had the biggest decrease in Lp-PLA$_2$ activity levels on statin therapy were most likely to have the greatest benefits, with fewer CHD deaths and heart attacks, as well as significantly fewer total CVD events ($p<0.001$)
- Reduction in Lp-PLA$_2$ activity may account for over half of the benefits of pravastatin in the LIPID study
- This study suggests that in addition to measuring and treating LDL-C, Lp-PLA$_2$ should also be monitored—prior to and during treatment—to assess the potential efficacy of statin therapy and to help direct CVD risk management

In summary, reduction in Lp-PLA$_2$ predicted CHD events, establishing Lp-PLA$_2$ as a new risk factor.

*Long-term Intervention with Pravastatin in Ischemic Disease (LIPID)

Reduction in Lp-PLA$_2$ is associated with coronary plaque regression[4]



	Baseline	Follow-up
Plaque volume (mm^3)	100.1	89.2
Lp-PLA$_2$ (IU/L)	502	237

Images reprinted from Dohi T, et al, in *Atherosclerosis*; 2011[4], with permission from Elsevier Ireland, Ltd.

A closer relationship.

In an intravascular ultrasound (IVUS) study of acute coronary syndrome (ACS) patients at six-month follow-up, plaque volume and circulating levels of Lp-PLA$_2$ were reduced. The absolute change in plaque volume significantly correlated with the reduction in Lp-PLA$_2$ levels ($r=0.404$, $p=0.009$).[4]

PLAC® Test for Lp-PLA$_2$

Add the PLAC® Test to your CVD risk assessment strategy.

The PLAC® Test is recommended for patients with established CVD[3] or patients at moderate to intermediate risk for CVD, such as patients with, including but not limited to, two or more of the following risk factors:

- Family history
- Diabetes
- Obesity
- Smoker
- Males aged ≥45 or females ≥55
- High cholesterol
- High blood pressure

Lp-PLA$_2$ testing is recognized in the guidelines of the European Society of Cardiology, American Association of Clinical Endocrinologists, American Heart Association and American Stroke Association.

More than 125,000 published patient results confirm the PLAC Test for Lp-PLA$_2$ is the only evidence-based assay for measuring the new modifiable risk factor, lipoprotein-associated phospholipase A$_2$, for CVD events.

For more information, visit www.plactest.com, or call diaDexus at 1.650.246.6400 or your laboratory representative.

@plactest

REFERENCES:

1. Jang Y, Waterworth D, Lee J-E, et al. Carriage of the V279F null allele within the gene encoding Lp-PLA$_2$ is protective from coronary artery disease in South Korean males. *PLoS ONE.* 2011;6(4):e18208. doi:10.1371/journal.pone.0018208. **2.** The Lp-PLA$_2$ Studies Collaboration. Lipoprotein-associated phospholipase A$_2$ and risk of coronary disease, stroke, and mortality: collaborative analysis of 32 prospective studies. *Lancet.* 2010;375:1536-1544. **3.** White HD, Simes J, Stewart RAH, et al. Changes in lipoprotein-associated phospholipase A$_2$ activity predict coronary events and partly account for the treatment effect of pravastatin: results from the long-term intervention with pravastatin in ischemic disease study. *J Am Heart Assoc.* 2013;2:e000360. doi:10.1161/JAHA.113.000360. **4.** Dohi T, Miyauchi K, Okazaki S, et al. Decreased circulating lipoprotein-associated phospholipase A$_2$ levels are associated with coronary plaque regression in patients with acute coronary syndrome. *Atherosclerosis.* 2011. doi:10.1016/j.atherosclerosis.2011.09.019. **5.** Kolodgie FD, Burke AP, Skorija KS, et al. Lipoprotein-associated phospholipase A$_2$ protein expression in the natural progression of human coronary atherosclerosis. *Arterioscler Thromb Vasc Biol.* 2006;26(11):2523-2529.