THE IMPORTANCE OF L_p-PLA_2 AS A CHD RISK ASSESSMENT BIOMARKER

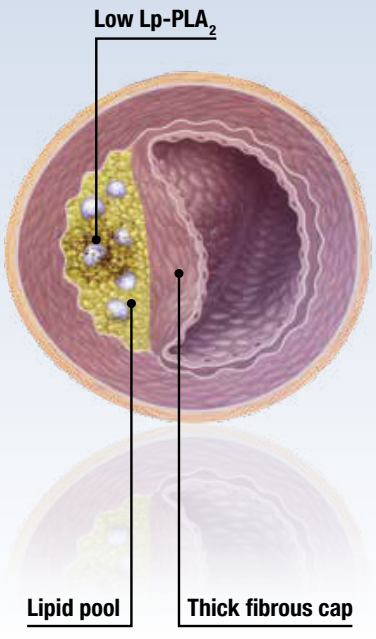
PLAC®

L_p-PLA_2

The PLAC® Test Clearly Identifies Active Cardiovascular Inflammatory Disease

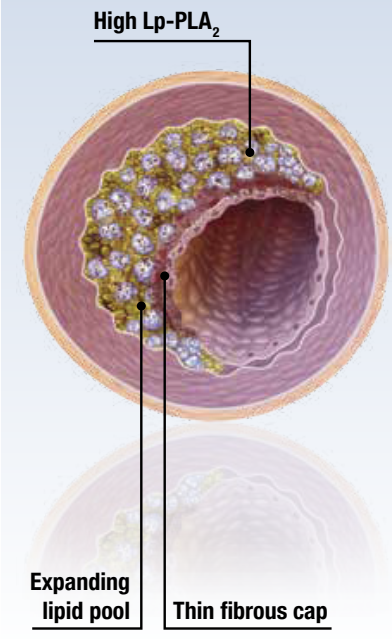
The PLAC® Test is the only blood test that measures Lp-PLA_2 – a vascular-specific inflammatory marker critical in the formation of rupture-prone plaque.[1]

Stable Plaque



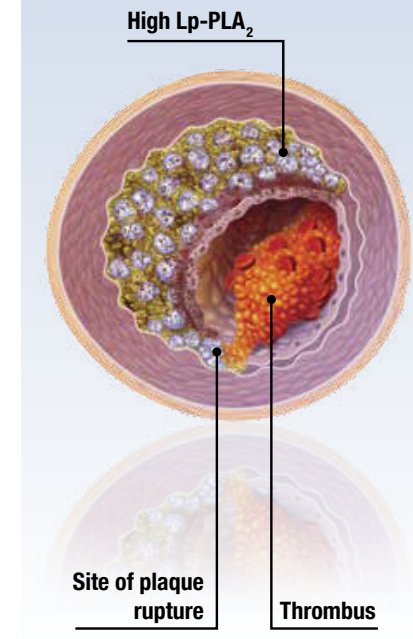
Cholesterol causes a fatty deposit called plaque that builds up inside the walls of the arteries

Rupture-Prone Plaque



When arterial walls become inflamed, the enzyme Lp-PLA_2 is produced within the plaque

Ruptured Plaque



If the amount of Lp-PLA_2 is high, the plaque is more likely to rupture through the inside lining of the artery into the bloodstream, leading to a dangerous blood clot that could result in heart attack or stroke[1]

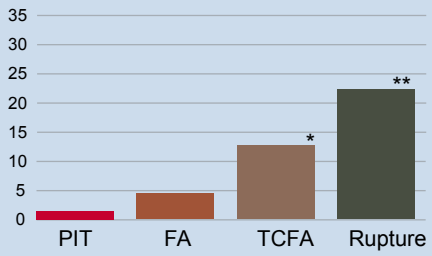
DIAZYME

Innovations in Clinical Diagnostics

PLAC® TEST FOR Lp-PLA$_2$

Lp-PLA$_2$ levels increase with plaque progression[1]

% Lp-PLA$_2$ staining in varying coronary plaque morphologies

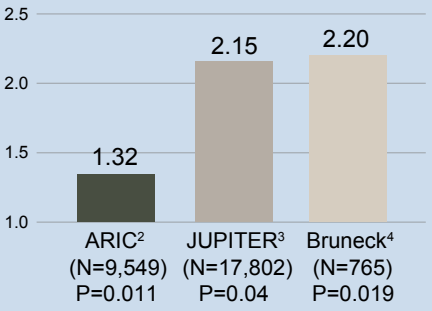


PIT = pathologic intimal thickening
FA = fibroatheroma
TCFA = thin-cap fibroatheroma

*P<0.05 vs FA or PIT
**P<0.05 vs TCFA, FA and PIT

The higher the level of Lp-PLA$_2$, the higher the risk for a CV event – even with normal LDL

Coronary and CV event hazard ratios



Fully adjusted for traditional risk factors

1. 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(11):2523-2529.
2. Hoogeveen, R., et al. Lipoprotein-Associated Phospholipase A2 Activity and Risk for Coronary Heart Disease and Stroke: The ARIC Study. ATVB 2011.
3. Ridker, P. M., et al. Relationship of lipoprotein-associated phospholipase A(2) mass and activity with incident vascular events among primary prevention patients allocated to placebo or to statin therapy: an analysis from the JUPITER trial. Clin Chem 2012;58(5): 877-886.
4. Tsimikas, S., et al. Lipoprotein-associated phospholipase A2 activity, ferritin levels, metabolic syndrome, and 10-year cardiovascular and non-cardiovascular mortality: results from the Bruneck study. Eur Heart J. 2009;30(1): 107-115.
5. Falk E, Shah PK, Fuster V. Coronary plaque disruption. Circulation. 1995; 92:657-671.
6. Sachdeva A, Cannon CP, Deedwania PC, et al. Am Heart J. 2009; 157(1):111-117.e2.
7. Data on file.
8. 3rd Annual American Society for Preventative Cardiology Cardiovascular Disease Prevention Conference, 2015. Symposium of REGARDS Lp-PLA2 Substudy.

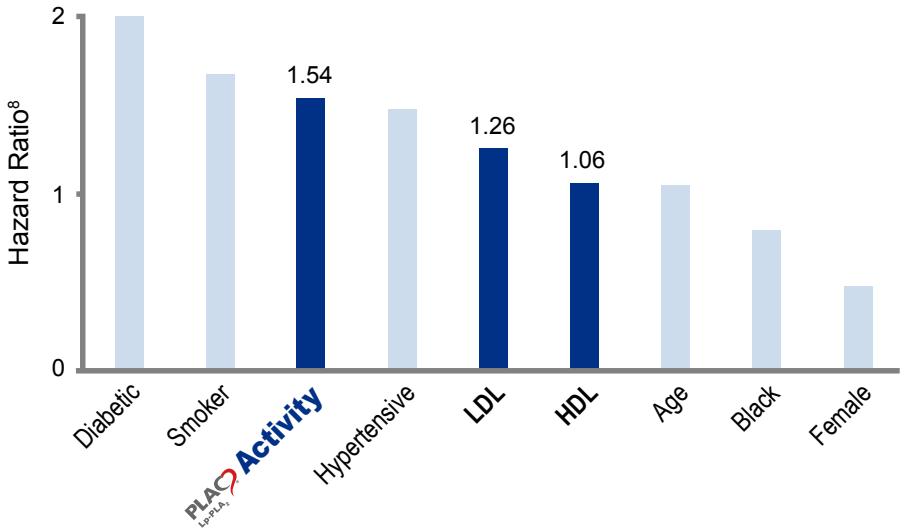
ASSOCIATION OF LP-PLA$_2$ AND CARDIOVASCULAR EVENTS

Coronary atherosclerosis is by far the most frequent cause of ischemic heart disease, and plaque disruption with superimposed thrombosis is the main cause of the acute coronary syndromes of unstable angina, myocardial infarction and sudden death.[2]

In a large cohort of patients hospitalized with coronary artery disease events, nearly 50% had admission LDL levels less than 100 mg/dL.[6]

The PLAC® Test for Lp-PLA$_2$ Activity is a strong and independent risk factor.

HR BY RISK FACTOR[7]



In a REGARDS multicenter sub study, high Lp-PLA$_2$ Activity was more closely associated with outcome than high LDL and low HDL. Only status of diabetes or smoking was more closely associated with events.[8]

Ask your Diazyme Representative for information on the PLAC® Test for Lp-PLA$_2$ Activity for your practice.

DIAZYME LABORATORIES, INC.
12889 Gregg Court, Poway, CA 92064 USA
PO Box 85608, San Diego, CA 92186 USA
Tel: +1-858-455-4768 +1-888-DIAZYME
www.diazyme.com sales@diazyme.com

DIAZYME EUROPE GMBH
Zum Windkanal 21, 01109 Dresden, Germany
Tel: +49-351-886-3300 Fax: +49-351-886-3366
sales@diazyme.de

DIAZYME SHANGHAI CO., LTD.
Room 201,1011 Halei Road, Zhangjiang Hi-tech Park
Shanghai, 201203, People's Republic of China
Tel: +86-21-51320668 Fax: +86-21-51320663
www.lanyuanbio.com service@lanyuanbio.com

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