



LAB #: Sample Report
 PATIENT: Sample Patient
 ID:
 SEX: Female
 DOB: 01/01/1951 AGE: 67

CLIENT #: 12345
 DOCTOR: Sample Doctor
 Doctor's Data, Inc.
 3755 Illinois Ave.
 St. Charles, IL 60174 U.S.A.

Cardiovascular Risk Profile; serum

LIPIDS/RATIOS	RESULT / UNIT	REFERENCE INTERVAL	CARDIOVASCULAR RISK				
			LOW RISK	MODERATE RISK	HIGH RISK		
Total Cholesterol	196 mg/dL	< 200					
Triglycerides; serum	90.0 mg/dL	< 150					
HDL Cholesterol	81 mg/dL	> 60					
LDL Cholesterol (calculated)	97.0 mg/dL	< 100					
VLDL Cholesterol (calculated)	18.0 mg/dL	< 30.0					
Non-HDL Cholesterol (calculated)	115 mg/dL	< 130					
Oxidized LDL	35 U/L	< 45					
Small dense LDL Cholesterol*	62 mg/dL	< 35					
Lp(a)	< 5 mg/dL	< 30					
Total Cholesterol : HDL-C	2.4	< 4.0					
LDL-C : HDL-C	1.2	< 2.0					
Oxidized LDL : HDL-C	0.4	< 0.8					
Small dense LDL-C : LDL-C	0.64	< 0.34					
Apo B : Apo A-1	0.9	< 0.8					
RISK FACTORS/INFLAMMATORY MARKERS							
PLAC (LP-PLA ₂ Activity)	166 nmol/mn/mL	< 151					
Homocysteine; serum	18.9 μmole/L	< 11.0					
CRP (Hs); serum	0.4 mg/L	< 1.0					
			PERCENTILE				
			2.5 th	16 th	50 th	94 th	97.5 th
APOLIPOPROTEINS							
Apolipoprotein A-1; serum	161 mg/dL	115- 220					
Apolipoprotein B; serum	148 mg/dL	50- 130					

SPECIMEN DATA

Comments:
 Date Collected: 05/30/2018 Time Collected: 08:00 AM <d!: less than detection limit
 Date Received: 06/01/2018 Fasting: Fasting *For Research Use Only. Not for use in diagnostic procedures.
 Date Completed: 06/06/2018 BMI: N/A
 Methodology: Chemistry Analyzer, Oxidized LDL by EIA

Small dense LDL Cholesterol High

Small dense LDL (sdLDL) is an extremely atherogenic LDL subtype that is associated with about 3-times greater risk for CVD than normal-size LDL particles. SdLDL-C levels are also independently associated with increased risk for Type-II diabetes. SdLDL-C is associated with elevated triglycerides and low HDL-C (mechanistically), obesity, metabolic syndrome, pre-diabetes, insulin resistance, renal dysfunction, hepatic steatosis and dietary trans-fatty acids.

The level of sdLDL-C is not proportional to the level of total LDL-C. The sdLDL more readily penetrate the arterial endothelial wall and are more prone to oxidation.

Elevated sdLDL-C may be lowered with lifestyle modifications and niacin that lower TG levels, as well appropriate control of blood glucose. Pharmaceuticals that lower sdLDL-C include, fenofibrate and combinations of fibrates and statins.

Small dense LDL : LDL-C High

A high ratio of small dense low density lipoprotein cholesterol (sdLDL-C) to total LDL-C indicates increased risk for CVD and diabetes type 2. Although both LDL-C and sdLDL-C levels are both independent risk factors for CVD, sdLDL are far more atherogenic than normal, larger LDL. Further, the level of sdLDL-C is not at all proportional to the level of LDL-C. Therefore the ratio of the two factors provides more sensitive assessment of risk for CVD and diabetes type 2 than either factor alone.

ApoB : ApoA1 Ratio High

A high ratio of apo B to apo-A1 is a very strong risk factor for CVD and acute myocardial infraction. Apo B levels provide a direct indication of the particle number of all atherogenic non-HDL lipoproteins, including VLDL, IDL, Lp(a) and LDL. Apo-A1 provides a direct indication of anti-atherogenic HDL particles. Therefore the apo B to apo-A1 ratio provides functional insight into so called cholesterol balance, or estimation of net reverse cholesterol transport.

Homocysteine High

High levels of serum homocysteine have long been thought to be an independent risk factor for CVD. Consensus has changed as a result of further evaluation, and presently homocysteine may be regarded as a weak risk factor for coronary heart disease. There is a lack of direct causal relationship between hyperhomocysteinemia and CVD. However elevated levels of homocysteine indicate significant disruption of essential methionine metabolism that can impair all essential methylation reactions, and impair the transsulfuration pathway with potentially diminished redox potential and increased oxidative stress. Methionine metabolism can be disrupted by genetic and epigenetic factors; the latter include deficiencies of vitamins B-6, B-12 and folate. The Plasma Methylation Profile can help identify causes of disrupted methionine and folate metabolism.

Apolipoprotein B High

A high level of apolipoprotein B (apo B) is a strong risk factor for CVD. Further, elevated levels of Apo B appear to indicate increased risk of fatal MI even when LDL levels are within normal. Elevated apo B is a better indicator of risk for CVD than either total cholesterol or LDL-cholesterol levels.

Apo B is the only protein constituent of low-density lipoproteins (LDL); apo B is also a component of all atherogenic non-HDL particles including very low density lipoproteins, intermediate density lipoproteins, Lp(a) and lipoprotein remnant particles. As such apo B levels provide a relative indication of atherogenic lipoprotein particle number.

PLAC High

High levels of lipoprotein phospholipase A2 activity (PLAC) are associated with increased risk of coronary artery disease (CAD) disease progression, plaque instability and cardiovascular events. High PLAC is indicative of very significant atherogenic disease activity within coronary arteries and increased risk for rupture of advanced plaque. High levels of PLAC are associated with double the risk of CAD regardless of the level of atherogenic non-HDL cholesterol levels, as well as a higher risk for myocardial infarction and CAD-related morbidity and mortality. PLAC interacts with oxidized LDL. It participates in the breakdown of oxidized LDL in the vascular wall by hydrolyzing the oxidized phospholipid, producing lysophosphatidylcholine and oxidized free fatty acids, both of which are potent pro-inflammatory products that contribute to the formation of atherosclerotic plaques.

PLAC is bound primarily to circulating LDL, and is enriched in atherosclerotic plaque. Lipid-laden macrophages within the artery release PLAC, further inflammation ensues, and calcified atherosclerotic plaques become unstable. Clinical management may include beginning or intensifying risk reduction strategies.