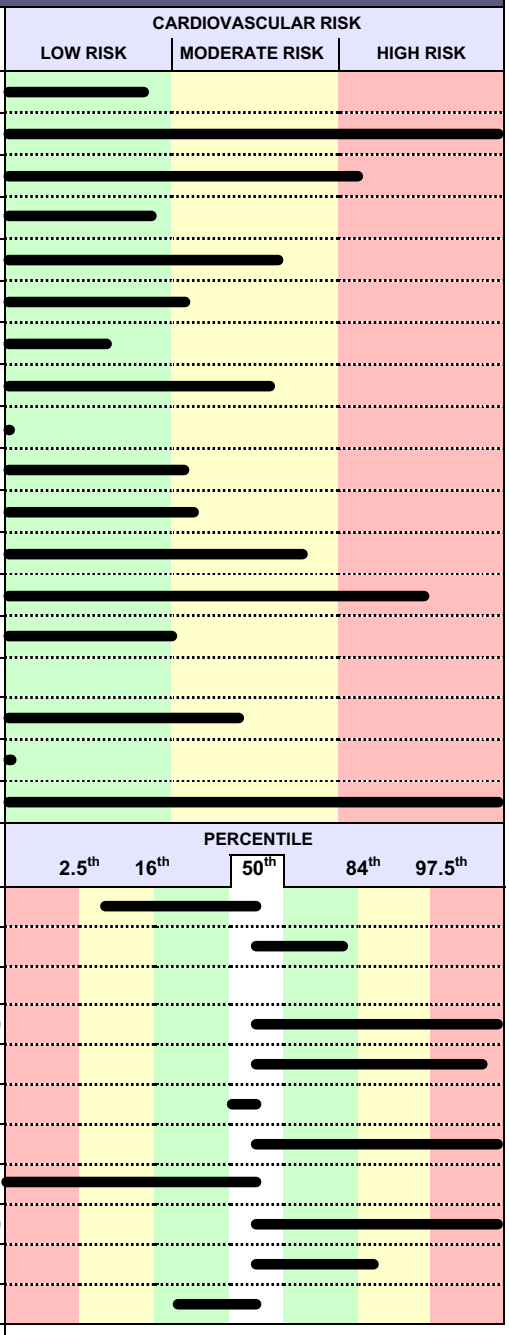
LAB #: Sample Report
PATIENT: Sample Patient
ID: Sample ID
SEX: Female
DOB: 00/00/1976 AGE: 48

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

CardioMetabolic Profile; serum

LIPIDS/RATIOS	RESULT	UNIT	REFERENCE INTERVAL
Total Cholesterol	173	mg/dL	< 200
Triglycerides	269	mg/dL	< 150
HDL Cholesterol	41	mg/dL	> 60
LDL Cholesterol (calculated)	90.0	mg/dL	< 100
VLDL Cholesterol (calculated)	43.0	mg/dL	< 30.0
Non-HDL Cholesterol (calculated)	134	mg/dL	< 130
OxLDL Cholesterol	44	U/L	< 60
sdLDL Cholesterol (calculcated)	41	mg/dL	< 35
Lp(a)	< 4	mg/dL	< 30
Total Cholesterol : HDL-C	4.3		< 4.0
LDL-C : HDL-C	2.2		< 2.0
OxLDL-C : LDL-C	0.49		< 0.45
sdLDL-C : LDL-C	0.45		< 0.34
Apo B : Apo A-1	0.80		< 0.80
RISK FACTORS/INFLAMMATORY MARKERS			
PLAC (LP-PLA$_2$ Activity)	168	U/L	< 151
Homocysteine	5.1	µmol/L	< 11.0
hsCRP	22	mg/L	< 1.0
APOLIPOPROTEINS			
Apolipoprotein A-1	126	mg/dL	115 - 220
Apolipoprotein B	104	mg/dL	50 - 130
METABOLIC RISK MARKERS			
Insulin	43.8	µIU/mL	2.8 - 18.0
Glucose	146	mg/dL	70.0 - 100
1,5-Anhydroglucitol (1,5 AG)	17	ug/mL	6.8 - 29
*Leptin	49	ng/mL	4.0 - 39
*Adiponectin	1.0	µg/mL	4.0 - 20
Leptin : Adiponectin ratio	48.3		0.20 - 3.40
Cystatin C	1.1	mg/L	0.5 - 1.5
Creatinine	0.8	mg/dL	0.6 - 1.3
eGFR (calculated)	81	mL/min	> 60



SPECIMEN DATA

Comments:
Date Collected: 04/28/2025 Time Collected: 09:30 AM
Date Received: 04/30/2025 Fasting: Fasting
Date Reported: 05/07/2025 BMI: 22
Methodology: Immunoturbidimetric Assay; Leptin by ELISA

<dl: less than detection limit
*For Research Use Only. Not for use in diagnostic procedures.

v1

HDL Cholesterol Low

A low level of high-density lipoprotein cholesterol (HDL-C) is considered to be an independent risk factor for CVD. Low levels of HDL-C increase the risk for atherosclerotic disease. Interpretation of the relative risk associated with low HDL-C should include consideration of the LDL-C to HDL-C ratio , and non-HDL-C in this report. Check for elevated triglycerides as they are a major factor adversely affecting HDL metabolism and size.

Diet and lifestyle changes that have been shown to decrease triglycerides and increase HDL-C include loss of body fat, increased routine aerobic exercise, smoking cessation, better control of blood glucose, omega-3 fatty acids (fish oil), decreased intake of trans-fatty acids, moderation in alcohol consumption, and possibly supplementation with nicotinic acid.

VLDL High

Elevated levels of very low density lipoprotein cholesterol (VLDL-C) have been associated with the atherosclerotic process. Very low density lipoproteins (VLDL) are triglyceride-rich particles secreted by the liver. With lipolysis of the core triglycerides (TG) free fatty acids are delivered to peripheral tissues. In the process intermediate density lipoproteins become enriched with cholesteryl esters and ultimately become cholesterol-enriched low density lipoproteins (LDL). Accumulation of VLDL-C indicates abnormal metabolism of lipids and lipoproteins. The best way to lower VLDL-C is to lower triglycerides by losing body fat, exercising regularly, reducing simple sugars and carbohydrates in the diet, and improving blood glucose levels. Normalizing the levels of adiponectin and leptin decrease fatty acid biosynthesis and increase fatty acid oxidation in the liver.

Non-HDL Cholesterol High

A high level of non-HDL cholesterol (NHDL-C) is a stronger CVD risk factor than LDL or triglycerides for patients with high triglycerides or diabetes. NHDL-C has become the new ”bad cholesterol,” as it reflects the sum of serum cholesterol carried by all of the potentially atherogenic apo-B containing lipoproteins including LDL, VLDL, IDL, Lp(a) and other remnant lipoproteins. Reductions in NHDL-C may improve endothelial function and reduce inflammatory reactions that contribute to atherosclerosis. NHDL-C is calculated from direct measurement of total and HDL cholesterol levels and is not influenced by serum triglyceride levels.

The recommended NHDL-C goal of less than 130 mg/dL is higher than the LDL-C target of 100 mg/dL.

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.

Triglycerides High

High levels of fasting triglycerides are associated with risk for CVD primarily due to their negative role in the regulation of the metabolism and size of high and low density lipoproteins. High levels of TG are associated with low levels of total HDL- cholesterol (HDL-C), and a preponderance of less anti-atherogenic smaller HDL-3. By a common mechanism, the activity of the plasma cholesterol ester transfer protein and lipolysis, high levels of TG are also associated with increased levels of atherogenic small dense LDL (sdLDL). Check the levels of HDL-C, and the sdLDL-C to LDL-C on this report.

High carbohydrate diets, excess simple sugar intake, hyperglycemia / hyperinsulinemia, metabolic syndrome, type II diabetes, excess abdominal fat, low adiponectin, high leptin, a high ratio of leptin to adiponectin, and excessive alcohol intake are all contributing factors to high serum TG levels.

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 lysophosphatidycholine 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 athero-sclerotic plaques become unstable. Clinical management may include beginning or intensifying risk reduction strategies.

Elevated hsCRP

An elevated level of hsCRP is a well-established indicator of arterial inflammation that is associated with substantial risk of coronary artery disease and cardiovascular events. It is an independent risk factor for future heart attack, stroke and death for asymptomatic men and woman. Elevated CRP has also been related to risk for metabolic syndrome; it tracks well with a high leptin to adiponectin ratio. Reductions in hsCRP levels along with other CVD risk factors such as non-HDL cholesterol levels has been associated with decreased progression of atherosclerosis and better clinical outcomes.

Guidelines for cardiovascular risk related to levels of CRP are: moderate; 1-3 mg/dL, high; 3-10 mg/dL. Levels greater than 10 are likely associated with non-cardiovascular inflammation (e.g. acute infection), and the hsCRP test should be repeated in about three weeks. Some suggested interventions to lower hsCRP levels include statins, decreasing adiposity, aspirin, and low-dose methotrexate.

High Serum Glucose

High fasting serum glucose greater than 125 mg/dL indicates hyperglycemia and diabetes, and diabetes is a risk factor for CVD. Serum glucose levels may also be elevated if the sample was collected non-fasting. Typical symptoms associated with diabetes may include excessive urination, abnormally elevated blood and urine glucose values, excessive thirst, persistent hunger, sudden weight loss, or elevated blood and urine ketones. Diabetes is associated with increased risk of kidney disease. Glucose levels > 400 mg/dL are considered critical and require immediate action to lower serum glucose levels.

Serum glucose levels > 90 mg/dL have been associated with an increased risk of developing type II diabetes, and may be associated with other cardiovascular risk factors such as low HDL cholesterol, hypertension and elevated serum triglycerides. Additional factors associated with elevated serum glucose include obesity or excessive abdominal fat, a prior history of gestational diabetes, or a family history of diabetes. Low insulin secretion or insulin resistance also increases the risk of developing diabetes.

Leptin High

High levels of leptin are associated with the development of metabolic syndrome and pre-diabetes, and may contribute to hypertension, atherosclerosis, and coronary heart disease, acute cardiovascular events and stroke. Leptin stimulates the sympathetic nervous system, adrenal function, vascular inflammation and increases oxidative stress. High levels of leptin have also been associated with collagen-related arthritis and joint inflammation, and symptoms of depression.

Lifestyle changes to lower leptin may include body fat loss, routine exercise, and smoking cessation, and a ”heart healthy diet.” Obstructive sleep apnea should be treated, if present.

Adiponectin Low

Low levels of adiponectin are associated with marked increases in risk for developing metabolic syndrome, type II diabetes and coronary artery disease. Adiponectin is a hormone /adipokine produced by fat cells, and is normally one of the most abundant hormones in circulation. However, with excess adiposity less adiponectin is released into circulation. Adiponectin has a major role in the regulation of blood glucose, insulin sensitivity, fatty acid oxidation, and triglyceride and lipoprotein metabolism. It also has anti-inflammatory, anti-atherogenic, anti-diabetic, anti-oxidative properties, and facilitates proper endothelial functioning. Lifestyle changes that decrease body fat mitigate low adiponectin and the associated metabolic consequences.

Total Cholesterol : HDL-C High

A high ratio of plasma total cholesterol (TC) to high-density lipoprotein cholesterol (HDL-C) is considered to be a CVD risk factor. Blood cholesterol is transported predominantly by low-density lipoproteins (LDL) and high-density lipoproteins (HDL). The majority of circulating TC is associated of LDL, and an elevated level of TC is considered to be CVD risk factors. HDL-C is inversely associated with CVD risk. The clinical significance of a level of TC is more predictive when viewed in context with the associated level of anti-atherogenic HDL-C.

LDL-C : HDL-C High

The ratio of low-density lipoprotein cholesterol (LDL-C) to high-density lipoprotein cholesterol (HDL-C) is higher than expected in this sample. The LDL-C: HDL-C ratio is considered to be a CVD risk factor. Plasma cholesterol is transported predominantly by low-density (LDL) and to a lesser extent by high-density lipoproteins (HDL). The majority of total cholesterol is associated within the hydrophobic core of LDL and LDL-C is considered to be CVD risk factor. HDL-C is inversely associated with CVD risk but the clinical significance of the level of HDL-C has more value when viewed in context with LDL-C. For example if one has a normal level of HDL-C but an elevated level of LDL-C the predictive value of that level of HDL-C may be significantly marginalized.

Oxidized LDL : LDL-C High

The ratio of oxidized low-density lipoprotein (ox-LDL): total low-density lipoprotein cholesterol (LDL-C) is higher than expected in this sample. Ox-LDL is a very atherogenic form of LDL and is considered to be an independent CVD risk factor. When the apolipoprotein B moiety of LDL becomes oxidized the protein is recognized as a foreign antigen that is rapidly taken up by the unregulated ”scavenger” or ”ox-LDL receptors” on monocyte-derived macrophages. When the phagocytic cells residing in the arterial intima accumulate ox-LDL they release proinflammatory cytokines and chemotactic messengers that initiate and perpetuate the atherogenic process.

Clinical efforts to lower the ratio of ox-LDL: LDL-C might include exercise and body fat reduction, increased consumption of antioxidant-rich foods or supplements, decreased consumption of trans-fatty acids, and cessation of smoking, and decreased toxicant exposures and oxidative stress. Fish oil (EPA/DHA-rich oil), fibrates and statins have been shown to lower both ox-LDL and small dense LDL. An oral liposomal glutathione preparation has been demonstrated to decrease the extent of ox-LDL uptake, macrophage cholesterol mass, and decreased atherosclerotic lesion area in a rodent model of atherosclerosis.

Steinberg D. (1997) Oxidative modification of LDL and athersclerosis. Circulation (95):1062-71.

Holvoet P et al. (1998) Ox-LDL and malondialdehyde-modified LDL in patients with acute coronary syndromes and stable CAD. Circulation(98):1487-94.

Schulze PC, Lee RT. (2005) Oxidative stress and athersclerosis. Curr Atheroscler Rep (7):242-8.

Rosenblat M et al. (2007) Anti-oxidant and anti-atherogenic properties of liposomal glutathione: Studies in vitro, and in atherosclerotic apo-E deficient mice. Atherosclerosis (195):e61-e68.

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.

High Leptin to Adiponectin

High leptin to adiponectin (LAR) ratios have been associated with obesity, type II diabetes, insulin resistance, inflammation, and CVD. Recent evidence indicates that a high LAR is more clinically sensitive for risks of metabolic syndrome, type II diabetes and CVD than either the serum levels of leptin or adiponectin alone.

High LAR appears to be an independent predictor of arterial intimal medial thickness. There is increased concern with respect to CVD when hsCRP is elevated into the high risk range (3-10 mg/L).

Insulin High

High fasting levels of insulin are observed in the early stages of type 2 diabetes and metabolic syndrome due to insulin resistance. Insulin resistance has been associated with cardiovascular disease, excess adiposity, and non-alcoholic fatty liver disease. Hyperinsulinemia is often associated with a pro-atherogenic lipid profile consisting of high triglycerides and sdLDL-C, and, low levels of HDL-C.

Insulin is a peptide hormone that regulates the cellular uptake of glucose. Insulin also has major roles in the regulation of the synthesis of protein and fatty acids, and triglyceride (fat) storage. Other medical conditions that may result in elevated insulin levels include acromegaly, Cushing's syndrome (hypercortisolism), polycystic ovary syndrome, fructose intolerance or galactose intolerance. Pharmaceuticals such as corticosteroids, levodopa or oral contraceptives may raise insulin levels.