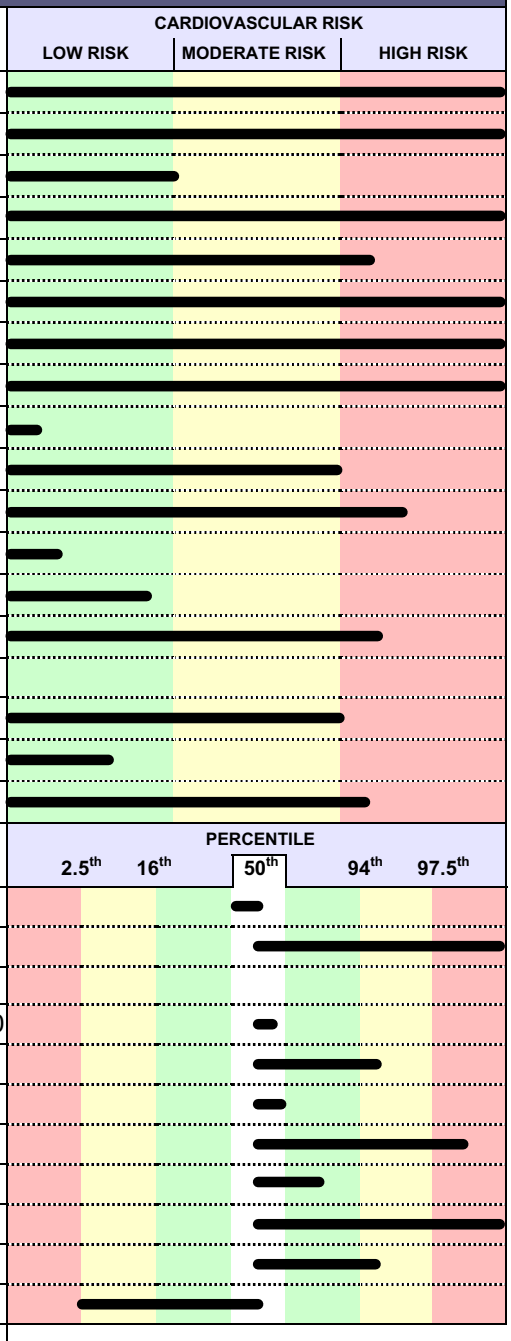
LAB #: Sample Report
PATIENT: Sample Patient
ID:
SEX: Female
DOB: 05/15/1960 AGE: 58

CLIENT #: 12345
DOCTOR: Sample Doctor
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	476	mg/dL	< 200
Triglycerides	281	mg/dL	< 150
HDL Cholesterol	60	mg/dL	> 60
LDL Cholesterol (calculated)	360	mg/dL	< 100
VLDL Cholesterol (calculated)	56.0	mg/dL	< 30.0
Non-HDL Cholesterol (calculated)	417	mg/dL	< 130
Oxidized LDL	96	U/L	< 45
Small dense LDL Cholesterol*	110	mg/dL	< 35
Lp(a)	9	mg/dL	< 30
Total Cholesterol : HDL-C	8.0		< 4.0
LDL-C : HDL-C	6.1		< 2.0
Oxidized LDL : HDL-C	0.3		< 0.8
Small dense LDL-C : LDL-C	0.30		< 0.34
Apo B : Apo A-1	1.4		< 0.8
RISK FACTORS/INFLAMMATORY MARKERS			
PLAC (LP-PLA$_2$ Activity)	199	nmol/min/mL	< 151
Homocysteine	8.6	µmole/L	< 11.0
CRP (hs)	5.9	mg/L	< 1.0
APOLIPOPROTEINS			
Apolipoprotein A-1	177	mg/dL	115 - 220
Apolipoprotein B	243	mg/dL	50 - 130
METABOLIC RISK MARKERS			
Insulin	7.0	µIU/mL	2.8 - 14.0
Glucose	109	mg/dL	70.0 - 100
Glycomark (1,5-Anhydroglucitol)	19	ug/mL	6.8 - 29
*Leptin	43	ng/mL	4.0 - 39
*Adiponectin	9.8	µg/mL	4.0 - 20
Leptin : Adiponectin ratio	4.4		1.5 - 3.2
Cystatin C	1.1	mg/L	0.5 - 1.5
Creatinine	0.6	mg/dL	0.6 - 1.3
eGFR (calculated)	95	mL/min	> 60



SPECIMEN DATA

Comments:
Date Collected: **01/15/2019** Time Collected: **08:45 AM** <dl: **less than detection limit**
Date Received: **01/17/2019** Fasting: **Fasting**
Date Completed: **01/28/2019** BMI: **N/A**
Methodology: **Chemistry Analyzer; Oxidized LDL, Leptin, Adiponectin by EIA**

*For Research Use Only. Not for use in diagnostic procedures.

Total Cholesterol High

The level of plasma total cholesterol in this sample is higher than expected. A high level of plasma total cholesterol has been long considered to an independent risk factor for CVD. Modern day research indicates that much more sensitive CVD risk factors include small dense LDL (sdLDL) cholesterol, the ratio of sdLDL cholesterol to LDL cholesterol, non-HDL cholesterol, and the ratio of LDL to HDL cholesterol.

Dietary changes, in addition to other lifestyle modifications, may help reduce total cholesterol.

Total cholesterol levels may be lowered by reducing the consumption of saturated fat, and increasing consumption of omega-3 fatty acids (e.g. fish oil, algae oil).

LDL Cholesterol High

The level of low-density lipoprotein cholesterol (LDL-C) in this sample is higher than expected. LDL-C has long been considered to be an independent risk factor for CVD. However recent research indicates that sub-species of LDL pose a better indication of risk when LDL particles are metabolized to sdLDL and oxidized LDL (ox-LDL). The levels of sd LDL-C and oxidized LDL are not correlated with the level of LDL-C therefore all three factors should be considered in the assessment of CVD risk.

Statin drugs reduce high levels of LDL-C by inhibiting the enzyme HMG-CoA reductase which is the rate-limiting step in cholesterol biosynthesis, but also inhibit production of CoQ10. Supplementation with CoQ10 is essential with use of statin drugs. High LDL-C levels may be lowered by consumption of an appropriate amount of omega-3 fatty acids from fish oil. Niacin (vitamin B3) may lower LDL-C by decreasing the hepatic secretion of precursor very low density lipoproteins.

LDL-C was calculated using a formula that incorporates the patient's level of serum triglycerides (TG). As serum TG rise above 200 mg/dL, calculated levels of LDL-C will be less accurate (under estimated). In such a case it is best to pay closer attention to the reported level of non-HDL cholesterol and small dense LDL-C (sdLDL-C) in this report. Non-HDL cholesterol (calculated from direct measurements of total and HDL-C is) not influenced by level of TG; in fact non-HDL cholesterol is a better indicator of risk of CVD than LDL-C.

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 ôbad cholesterol,ö 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. Calculated LDL-C is less accurate for risk assessment when triglycerides are greater than 200 mg/d.

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

Oxidized LDL High

A high level of oxidized LDL (oxLDL) is a strong predictor of risk for coronary artery disease (CAD), and increasing levels of oxLDL are incrementally associated with the severity of CAD. High levels of oxLDL also markedly increase the risk for developing metabolic syndrome well within a decade.

The apo B protein constituent is oxidized when LDL particles (predominantly small dense LDL) penetrate the arterial wall. The modified apo B protein is then recognized as foreign, taken up in an unregulated manor by the scavenger receptors on resident macrophages. That process instigates an arterial inflammatory response with further recruitment of monocytes, and the initiation of foam cells. Oxidized LDL interacts with PLAC (Lp-PLAC2), which increases inflammation and enhances a pro-atherogenic state, as well as plaque vulnerability.

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 f 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.

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.

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 ôheart healthy diet.ö Obstructive sleep apnea should be treated, if present.

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. A low 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). 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.

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.

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 athe serum levels of leptin or adiponectin alone.

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