

Comprehensive Cardiovascular Profile

Overview Genova Diagnostics has developed a revolutionary new Cardiovascular Assessment on the cutting edge of molecular medicine. Based on the latest advancements in cardiovascular research, this groundbreaking assessment measures the most important CVD markers available, including crucial new independent factors, a diverse array of lipoproteins, and two computed ratios.

All of these advanced markers play a critical role in assessing the biochemical environment underlying cardiovascular health. This information allows the clinician to accurately address abnormalities relating to heart and vascular diseases.

Using this state-of-the-art assessment, you can now better identify individuals who don't show abnormalities in traditional markers of CVD—those who comprise almost 50% of all heart attack victims. A clear, color-coded test report will allow you to quickly interpret results, and design a unique, customized nutrition therapy program that can dramatically improve a patient's quality of life, and possibly even save it.

This dynamic new assessment comes at a much-needed time. The number one killer in America today, cardiovascular disease claims as many lives as the next 8 leading causes of death combined, including cancer, accidents and AIDS.¹ No other modern illness comes close to reaping such a grim toll on human life: Over 950,000 deaths a year in the U.S. alone—or an average of about 2600 deaths each day.² And the scope of this insidious health problem is world-wide. Globally, cardiovascular disease accounts for almost 50% of all deaths.³

The current medical intervention for advanced coronary diseases includes balloon angioplasty, bypass surgery, heart transplants, and cardiac catheterization, all extremely traumatic procedures with prohibitive price tags. Bypass surgery has grown into a multi-billion dollar industry, with approximately 500,000 bypasses performed annually in the U.S. alone, at an average cost of about \$45,000 each.⁴ In fact, the total cost incurred by cardiovascular disease and stroke in this country was most recently estimated at an astounding \$259.1 billion for one year, and the figures continue to climb.⁵

Perhaps the greatest tragedy is that the deleterious consequences of this deadly disease are very often preventable. With advances in medicine, researchers have discovered important early indicators that can significantly change the direction of prophylactic and therapeutic treatment, decreasing the incidence and halting the progression of the disease.

Status of CV Disease It is currently estimated that 57 million Americans—over one-fifth of the entire U.S. population—suffer from some form of cardiovascular disease.⁶ Testing is strongly recommended for patients with the following history or medical conditions:

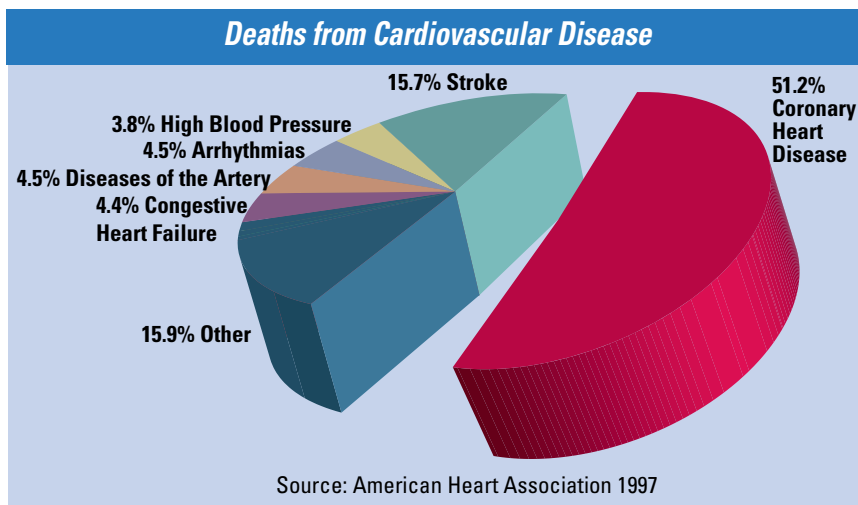
- Family history of cardiovascular disease
- Personal history of myocardial infarction, peripheral artery disease, or coronary artery disease
- Obesity
- Diabetes
- Hypertension
- Thyroid disorders
- Stress
- Chronic illness (including chronic fatigue)
- Diets high in saturated and trans fats
- Sedentary lifestyle
- Use of alcohol, nicotine, drugs (including certain medications)
- Use of oral contraceptives
- Hormonal imbalances (estradiol, cortisol, insulin, melatonin)
- Post-menopause
- Renal disease

What this test does:

Helps identify patients with hidden CVR abnormalities—even those with normal lipid levels

Provides a solid foundation for effective nutritional therapy

Turn-around Time 7 days



Cardiovascular System

A small muscular organ about the size of a clenched fist, the heart beats more than 100,000 times each day.⁷ Although the heart has the inherent ability to spontaneously initiate cardiac impulse, the autonomic nervous system plays an important role in regulating the impulse formation. Cardiac performance is influenced by many factors including heart rate, ventricular contraction, atrial function, neural control, drugs, and hormonal and metabolic products.⁸

The cardiovascular system has three crucial functions:

- 1) To transport oxygen and other nutrients to the cells of the body
- 2) To remove metabolic waste products from cells
- 3) To carry substances such as hormones from one part of the body to another

To accomplish these tasks, the heart pumps nearly 2000 gallons of blood throughout the body's circulatory system every day. The vast network of blood vessels, including arteries, capillaries and veins, is over 60,000 miles long—more than twice the distance around the earth.⁷

Atherosclerosis and Arteriosclerosis

Ongoing molecular and cellular research over the last 30 years reveals that atherogenesis is a complex disorder arising from the combined interaction of lipid accumulation and cell proliferation, involving numerous cell types including arterial medial smooth muscle cells, endothelial cells, and macrophages.

Atherosclerosis occurs when fat deposits form on the walls of major arteries throughout the body, particularly on the walls of coronary arteries. This process often begins in childhood with the development of lipid-rich lesions, called fatty streaks or atheromas. These lesions undergo pathological changes over time, often beginning to appear as fibrous plaque—fat debris that proliferates smooth-muscle cells and connective tissue—at about age 25.⁹

Once lesions reach an advanced stage, they may calcify, hardening in a process known as arteriosclerosis. If thrombosis (clot formation) occurs on the surface of a plaque of an arteriosclerotic coronary artery, the result can be angina or a heart attack. Atherosclerosis of the cerebral arteries, on the other hand, may cause strokes and transient ischemic attacks.

Clinical Markers Blood Lipids

Lipids are vital for the survival of the body. An integral part of the cell membrane, lipids also function to protect the body from the entry of water soluble substances through the skin and to prevent evaporation of water from the body. There are five major classes of lipids in blood plasma: cholesterol, cholesteryl ester, phospholipid,

triglyceride, and unesterified fatty acids. All other lipids are bound together in larger complexes and are called lipoproteins.¹⁰

Over the last 30 years, a plethora of evidence has been found to substantiate the relationship between various blood lipids and coronary disease. Although these factors generally increase with age, they strongly relate to cardiovascular health throughout an individual's life. A case-controlled study of young adults who died suddenly and unexpectedly from atherosclerotic coronary artery disease found blood lipid and lipoprotein values that were markedly higher in the victims. Researchers concluded that levels of plasma cholesterol, triglycerides, and LDL were all important markers for cardiovascular assessment, even for young adults.¹¹

Total Cholesterol

The most abundant steroid in animal tissue, cholesterol is the pre-cursor to major steroid hormones such as cortisol, DHEA, testosterone, and estrogen. The body synthesizes 60 to 80 percent of its cholesterol, primarily in the liver and intestine, and derives the remainder from the diet. Although elevated cholesterol is linked to increased atherosclerosis, cholesterol also functions as a natural protector of cell membranes due to its free radical scavenging activity. Widely dispersed in cell membranes throughout the body, unoxidized cholesterol protects against cancer and other free-radical-induced diseases. Total cholesterol thus serves as a marker for both cardiovascular disease and oxidative stress.

The role of serum cholesterol levels in the development and progression of heart disease has been well-established by research over the last forty years. The relationship between cholesterol levels and CVD appears to be a continuum, i.e., the higher the serum cholesterol, the more frequently associated heart disease is found.¹²⁻¹⁵ The Framingham study found that individuals with serum cholesterol levels below 175 mg/dl had less than half the rate of infarction as those with levels of 250 to 275 mg/dl. For certain age groups, cardiovascular deaths increased 10% for each 10 mg/dl increase in cholesterol.¹⁶ Elevated cholesterol levels have also been specifically linked with increased heterogeneous carotid plaque and the development of cerebrovascular diseases.^{17,18}

Although total cholesterol can provide a very general assessment of cardiovascular health, additional consideration of other lipid markers is crucial for accurate investigation of coronary disease, particularly for people over age 60.^{19,20}

LDL

Low density lipoproteins (LDL) deliver cholesterol to cells for membrane synthesis and steroid hormone synthesis, via LDL receptors. In healthy humans, more than 70 percent of the LDL circulating in plasma is removed each day through LDL receptors.¹⁰ Diets high in saturated fats and cholesterol decrease the liver's endogenous synthesis of cholesterol and can cause chronic suppression of the LDL receptors, resulting in elevated circulatory levels of LDL.

Excess LDL is removed from the bloodstream by cells of the reticuloendothelial system, via the scavenger-cell pathway. This removal results in the peroxidation of LDL, and may promote the cholesterol and cholesteryl ester accumulations in macrophages and smooth muscle cells that lead to the development of atherosclerotic plaque.

An abundance of research has established that elevated serum levels of LDL are a major cause of coronary heart disease.²¹⁻²⁴ Thus, measuring LDL is critical for complete interpretation of total cholesterol level.²⁵ A long-term epidemiological research project of incredible magnitude, the Göttingen Risk, Incidence and Prevalence study investigated the relationship of lipid factors with the development of cardiovascular disease in approximately 6000 middle aged men, and found that LDL cholesterol, followed by plasma concentration of total cholesterol and apolipoprotein B, were the predominant markers associated with coronary artery disease.²⁶

HDL

Commonly called the "good cholesterol," HDL is not secreted into the circulation as a mature lipoprotein but is assembled in the blood from components derived from the

intestine, liver, cell membranes, and triglyceride-rich lipoproteins during lipolysis. Cholesterol and phospholipids originating from membranes during cellular renewal and death become associated with HDL.

Two mechanisms have been proposed to explain how HDL might be a deterrent against atherosclerosis. One theory says that HDL acts as a scavenger by removing cholesterol from peripheral cells such as smooth muscle. In this process, free cholesterol in plasma membranes of peripheral tissues comes into contact with the surface of HDL, where it is esterified by lecithin-cholesterol acyltransferase (LCAT) and stored in the core of the HDL particle. HDL could also provide protective benefits from its apparent ability to influence the binding and absorption of LDL by cells such as smooth muscle.⁹

An inverse relationship between HDL and the incidence of coronary disease has been well-established.²⁷ One study found that individuals at the 80th percentile of HDL had one-half the likelihood of developing coronary heart disease when compared to those at the 20th percentile of HDL.²⁸ An extensive study that examined over 8000 subjects from the Framingham Study, the Coronary Prevention Trial, and the LRC Population Prevalence study found total cholesterol/HDL ratio to be quite strongly associated with coronary heart disease.²⁹

Homocysteine

In a healthy artery, the endothelial cells form a continuous protective layer that regulates the passage of substances from the plasma to the underlying artery wall. Injury to the endothelial cells, however, may alter their permeability and allow direct interaction between elements from the blood and the artery wall. Homocysteine acts as a molecular abrasive by scraping the inner layer of blood vessels. Thus high levels of homocysteine have been correlated with damaged endothelium, increased platelet utilization, and the formation of atherosclerotic lesions.⁹ One study found that men with extremely high homocysteine levels were three times more likely to have an associated myocardial infarction, even when adjustments for other factors such as blood lipids were considered.³⁰

Homocysteine is an intermediate in the biosynthesis of cysteine from methionine, via cystathionine. Deficiency of vitamin B12, folic acid and/or vitamin B6 can affect the enzyme pathways involved in cysteine formation, resulting in increased circulating homocysteine levels in the blood.

Because homocysteine is located at a key metabolic pathway, it affects methyl and sulfur group metabolism throughout the body, and may serve as a critical functional marker for assessing the status of the skeletal and nervous systems. A wide array of health conditions, including depression, multiple sclerosis, diabetes, birth defects, Alzheimer's disease, rheumatoid arthritis and osteoporosis are associated with elevated levels of homocysteine.³¹

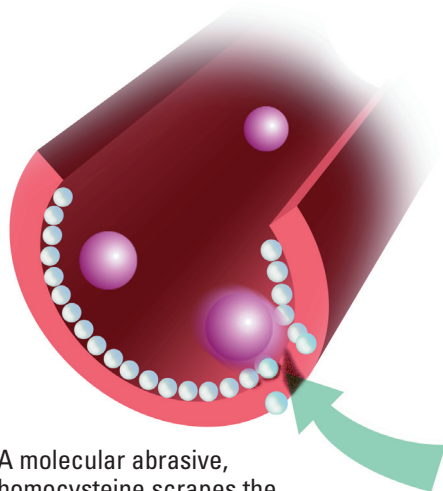
Triglycerides

Triglycerides are esterified fatty oils that predominate in the core of chylomicrons and VLDL. Elevated blood levels of triglycerides, but not cholesterol, have been associated with an impaired fibrinolytic system³²—thus accounting for another possible etiology in the development of cardiovascular disease. Recent angiographic studies have implicated triglycerides in the progression of both coronary and peripheral atherosclerosis, independent of LDL.^{33,34}

Koren and others found that triglyceride levels reflect the severity of coronary artery disease.³⁵ In addition, elevated serum triglycerides have been specifically tied to the occurrence of atherothrombotic stroke and transient ischemic attacks.³⁶ In a recent article for JAMA, researchers noted the powerful role of triglycerides as predictors of myocardial infarction.³⁷ A diet high in saturated fats can raise serum triglycerides. Since triglycerides are also derived from excess glucose in the bloodstream, excess carbohydrates may also result in elevated serum triglycerides.

Lipoprotein(a)

Lipoproteins are high-molecular-weight particles that transport water-insoluble lipids (primarily triglycerides and cholesterol esters) through the plasma. Lipoprotein (a), or Lp(a), consists of an LDL molecule covalently bound to the protein component apolipoprotein(a).



A molecular abrasive, homocysteine scrapes the endothelial cells, causing atherosclerotic lesion

Research over the last twenty years has underscored the critical relationship between Lp(a) and coronary artery disease, delineating its causative role in atherothrombogenesis and its strong association with both coronary and peripheral cardiac events.³⁸⁻⁴² As many as 40% of individuals who suffer myocardial infarction do not display conventional factors of concern such as fatty diets, lack of exercise, hypertension, smoking and high cholesterol.⁴³ Largely hereditary, Lp(a) is unaffected by many of these external influences associated with heart disease,^{44,45} and thus helps explain why a seemingly “healthy” patient may experience a MI, while an “unhealthy” patient, with a preponderance of various other biochemical and lifestyle factors, may not.

Lp(a) has been cited as a better predictor of coronary disease severity than most other lipid parameters.^{46,47} Doetch, Roheim, and Thompson referred to Lp(a) as the most important genetic factor associated with early atherosclerosis and coronary artery disease.⁴⁸

Although its exact physiological mechanisms have not yet been fully determined, there are a variety of ways that Lp(a) could promote the development of atherosclerosis and thrombosis. Lp(a) binds to endothelial and macrophage cells, fibrinogen and fibrin, promoting the deposit of cholesterol and other fatty waste in the vascular endothelium. Lp(a) also prevents clot lysis, adding fibrin and other debris to atherosclerotic plaque. Another theory is that by inhibiting plasminogen activity, Lp(a) prevents the activation by plasmin of latent transforming growth factor-beta. This action suppresses smooth muscle cell growth, encouraging the proliferation of muscle cells commonly seen in atherosclerotic lesions.^{49,50}

Lp(a) is also an accurate indicator for assessing the extent of carotid atherosclerosis, and an elevated serum level can serve as the most significant indicator of patients in which cerebral infarction is a concern.⁵¹⁻⁵⁴

Apolipoprotein A-1

A protein component of various lipoprotein complexes, Apo A-1 is the major constituent of HDL, and higher levels of this protein are predictive of a decreased incidence of cardiovascular disease. In a controlled study of adolescents with a family history of coronary heart disease, French researchers found that in young men, Apo A-1 was the best predictor of family history of early myocardial infarction, while in young women it was HDL-C.⁵⁵ In a Mayo Clinic study, cardiovascular specialists argued that plasma apolipoprotein levels—particularly A-1 and A-2—may be considerably better markers than traditional lipid determinants.⁵⁶

Apolipoprotein B

Another protein component of lipoproteins, Apo B is the primary substance in LDL and is thus associated with an increased incidence of coronary artery disease.²⁶ Reinhart and others concluded that both apolipoproteins A-1 and B provide important information about the presence of coronary artery disease.⁵⁷ Researchers at Johns Hopkins went even further, asserting that the “non-traditional” markers apo A-1 and Apo B were better indicators of premature coronary atherosclerosis than plasma lipoproteins.⁵⁸

Ratio of Apo B/Apo A-1

One study on 225 patients with angiographic evidence of coronary artery disease concluded that the strongest association between coronary artery disease and blood analytes was found in the ratio of Apo B/A-1.⁵⁹ Van Stiphout and his colleagues compared lipid levels in two groups: children of fathers with severe coronary atherosclerosis and children whose fathers were free of atherosclerosis. They found a higher ratio of Apo B/Apo A-1 in sons of fathers with severe coronary atherosclerosis, and suggested that the ratio be used to detect children who have an increased probability of developing severe atherosclerosis in later life.⁶⁰ These findings were supported by a study done by Beigel and others, which identified the Apo B/Apo A-1 ratio as the best predictor of a family history of coronary artery disease in children, and an important consideration in their cardiovascular health.⁶¹

Over 57 million Americans have one or more types of cardiovascular disease according to current estimates.

- High Blood Pressure—50,000,000
- Coronary Heart Disease—13,670,000
- Stroke—3,890,000
- Rheumatic Fever/
Rheumatic Heart Disease—1,380,000

Fibrinogen

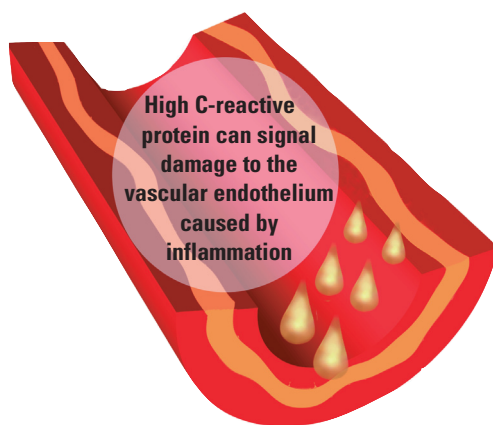
A globulin synthesized in the liver, fibrinogen strongly affects blood coagulation, blood rheology and platelet aggregation. It has direct effects on the vascular wall and is a prominent acute-phase reactant. Fibrinogen plays a key role in arterial occlusion by promoting atherosclerotic plaque, thrombus formation, endothelial injury, and hyperviscosity.⁶²⁻⁶⁶ Recently, researchers discovered fibrinogen involvement in the subclinical phase of extracoronary and coronary atherosclerosis. They postulated that a synergistic effect existed between the total cholesterol/HDL ratio and fibrinogen, with fibrinogen acting as a potential trigger for the atherogenic effect of hyperlipidemia.⁶⁷ This discovery underscores the importance of using comprehensive testing to accurately assess cardiovascular status.

Another study on over 200 stroke patients established a link between fibrinogen plasma levels and brain infarction mortality.⁶⁸ Fibrinogen can be increased by smoking, obesity, inflammation, stress, oral contra-ceptives, and aging.⁶⁹⁻⁷³

C-Reactive Protein

Inflammation may be a crucial factor in the pathogenesis of atherothrombosis. C-reactive protein is a marker associated with production of inflammatory cytokines. These cytokines appear to encourage coagulation and damage to the vascular endothelium, increasing the potential threat to cardiovascular health.⁷⁴

A recent study published in the New England Journal of Medicine found that plasma C-reactive protein (CRP), a marker for systemic inflammation, is a strong predictor of myocardial infarction and stroke. Men with CRP values in the highest quartile had three times the incidence of myocardial infarction and two times the incidence of ischemic stroke. Significantly, these relationships remained steady over long periods, and were independent of other lipid and non-lipid factors, including smoking. Evidence suggests that previous infection with pathogens such as Chlamydia pneumoniae or Helicobacter pylori may initially trigger the chronic inflammation detected by CRP.⁷⁵ Researchers thus theorize that one way aspirin improves cardiovascular function is through its anti-inflammatory effect, and the subsequent lowering of C-reactive protein levels.



Diagnosis and Treatment

Although most of the blood analytes used in Genova Diagnostics' Cardiovascular Assessment are independent markers, a composite analysis is crucial to understand the full implication of the test results. Many of the markers exert a more powerful impact on coronary status in concert with one another; studies have shown, for example, that the combination of high LDL cholesterol, elevated triglycerides, and low HDL is of greater concern than elevated LDL alone.⁷⁶ Thus this assessment contains a composite Cardiovascular Index, determined by a multivariate analysis of all of the individual lipid scores and the independent CV factors.

Once test results have been carefully evaluated, numerous therapeutic interventions can be implemented to significantly improve cardiovascular health. Conditions such as hypertriglyceridemia and low HDL cholesterol, for example, can often be managed through a treatment program that addresses diet, weight control, exercise, and smoking cessation.⁷⁷ Homocysteine is a modifiable CV factor that can often be effectively treated with vitamin supplements such as B6, B12 and folic acid.⁷⁸ Niacin and neomycin have been shown to reduce serum Lp(a) levels in patients with hyperlipidaemia.⁷⁹⁻⁸¹ And combined hormone therapy using progesterone and estradiol effectively can also lower Lp(a).⁸²

In fact, evidence from randomized, controlled trials confirms that comprehensive lifestyle changes may be able to induce regression of even severe coronary atherosclerosis after one year, without the use of lipid-lowering drugs.⁸³ Please see our Comprehensive Cardiovascular Assessment Interpretive Guidelines for more detailed diagnostic interpretations and suggested treatment protocols.

Related Tests To Consider Adrenocortex Stress Profile

Stress is a well-known major contributing factor in the development of cardiovascular disease. The Adrenocortex Stress Profile analyzes adrenocortical function, a crucial indicator of the body's ability to handle stress, by measuring levels of the adrenal

hormones cortisol and DHEA-S. Researchers at Johns Hopkins demonstrated a “consistent, independent, inverse, dose-response relationship” between DHEA-S levels and coronary atherosclerosis in men. They suggested that DHEA-S could serve as an important, modifiable component in the development and progression of coronary atherosclerosis.⁸⁴ Oral administration of DHEA (50 mg/d) to post-menopausal women enhanced tissue insulin sensitivity and lowered serum triglycerides.⁸⁵ Fatigue, commonly associated with high cortisol levels, can also be an early warning symptom of coronary heart disease, making this profile an especially valuable analysis for patients exhibiting concomitant symptoms of fatigue and stress.

Male & Female Hormone Profiles

Estrogen replacement has been shown to improve endothelium-dependent vasodilation of coronary arteries in women, perhaps due to its antioxidant properties.⁸⁶ Similarly, testosterone therapy seems to decrease total cholesterol and atherogenic LDL cholesterol in elderly and hypogonadal men⁸⁷ and significantly lower elevated Lp(a) levels in healthy men.⁸⁸ Reduced levels of testosterone are associated with coronary heart disease, and oral doses improve angina pectoris in patients with coronary heart disease compared to controls.⁸⁹ Genova Diagnostics’ Male and Female Hormone Profiles are critical tools for effectively implementing and monitoring sex hormone replacement therapies.

Essential and Metabolic Fatty Acids Analysis

Essential fatty acids have recently elicited much attention from researchers for their potential to prevent and inhibit the progression of atherosclerosis. Epidemiological studies have confirmed the anti-atherogenic effect of both eicosapentaenoic (EPA) and docosahexaenoic acid (DHA), and spawned heightened interest in the role of their metabolic precursor, alpha-linolenic acid.⁹⁰ Fatty acids are excellent indicators of dietary intake and early, pre-symptomatic conditions leading to the development of atherosclerosis.^{91,92} In addition, elevated levels of alpha-linolenic acid appear to be protective against some forms of cardiovascular disease in men.⁹³ Genova Diagnostics’ Fatty Acids Analysis measures erythrocyte levels of over 40 fatty acids.

Elemental Analysis

Genova Diagnostics’ Elemental Analysis identifies levels of toxic and nutritional elements that may be contributing to coronary disease conditions. This test may be particularly indicated in patients with hypertension, as cadmium accumulation and low magnesium both may contribute to elevated blood pressure. Accumulation of toxic mercury, moreover, is associated with increased incidence of acute myocardial infarction and with death from cardiovascular disease in general.⁹⁴ Low hair levels of calcium have been correlated with cardiovascular disease pathology.^{95,96} Magnesium deficiencies have been strongly tied to the etiology of cardiovascular disease,⁹⁷ and higher hair magnesium levels are associated with increased rates of regional cerebral blood flow.⁹⁸

Amino Acids Analysis

Amino acid imbalances can impact cardiovascular health in a variety of ways. An antioxidant with anti-inflammatory effects, taurine modulates the cellular influx of calcium, sodium, potassium and, significantly, magnesium—sometimes called “nature’s calcium channel blocker.” By promoting potassium retention by the heart, taurine actually appears to reverse certain cardiac abnormalities.⁹⁹ Thus subnormal levels may indicate the course of increased cardiovascular symptoms.

Methionine is a crucial essential amino acid required for proper metabolism of carbohydrates and lipids. Impaired methionine metabolism, particularly homocysteine accumulation, leads to increased incidence of coronary artery disease,¹⁰⁰ peripheral and cerebral occlusive disease,¹⁰¹ myocardial infarction,^{102,103} and stroke.¹⁰⁴

Administration of arginine has been demonstrated to reduce intimal thickness and vascular reactivity in atherosclerosis,¹⁰⁵ as well as decrease platelet clumping and clot formation within arterial walls.¹⁰⁶ Arginine is a precursor to nitric oxide, which plays a key role in regulating blood pressure and platelet accumulation.

Other amino acid patterns can provide important clues about deficiencies of nutrients such as B6, B12, folic acid and serine, the major components in homocysteine metabolism. The Amino Acids Analysis evaluates over 40 metabolic markers and is available as a plasma or a 24-hour urine assay.

Comprehensive Melatonin Profile

A decrease in melatonin causes increased night-time sympathetic activity, which in turn is linked to coronary disease. One study found that patients with coronary heart disease had nocturnal melatonin levels five times lower than in healthy controls. Investigators surmised that lower levels of melatonin may act to increase circulating epinephrine and norepinephrine. Atherogenic uptake of LDL cholesterol is accelerated by these amines at pathophysiological concentrations.¹⁰⁷

Laboratory research has shown that melatonin treatment also exerts the beneficial effect of increasing the HDL/LDL cholesterol ratio, possibly by enhancing endogenous cholesterol clearance mechanisms.¹⁰⁸ In addition, melatonin appears to inhibit platelet aggregation, which plays a significant role in the progression of cardiovascular disease.¹⁰⁹ A non-invasive salivary test, the Comprehensive Melatonin Profile measures the circadian rhythm of melatonin secretion over a complete light-dark cycle, and is particularly recommended for patients who exhibit insomnia as part of their overall clinical condition.

Oxidative Stress Analysis

Oxidation of lipids in the bloodstream is a crucial mechanism for setting off the damaging process of atherosclerosis. The Oxidative Stress Analysis includes measurements of blood glutathione, lipid peroxides, and hydroxyl radical markers as important indicators of oxidative stress in the body.

How do I order this test?

For Comprehensive Cardiovascular Profile kits or information, please call a Client Services representative at 800-522-4762 or order online at www.GDX.net.

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