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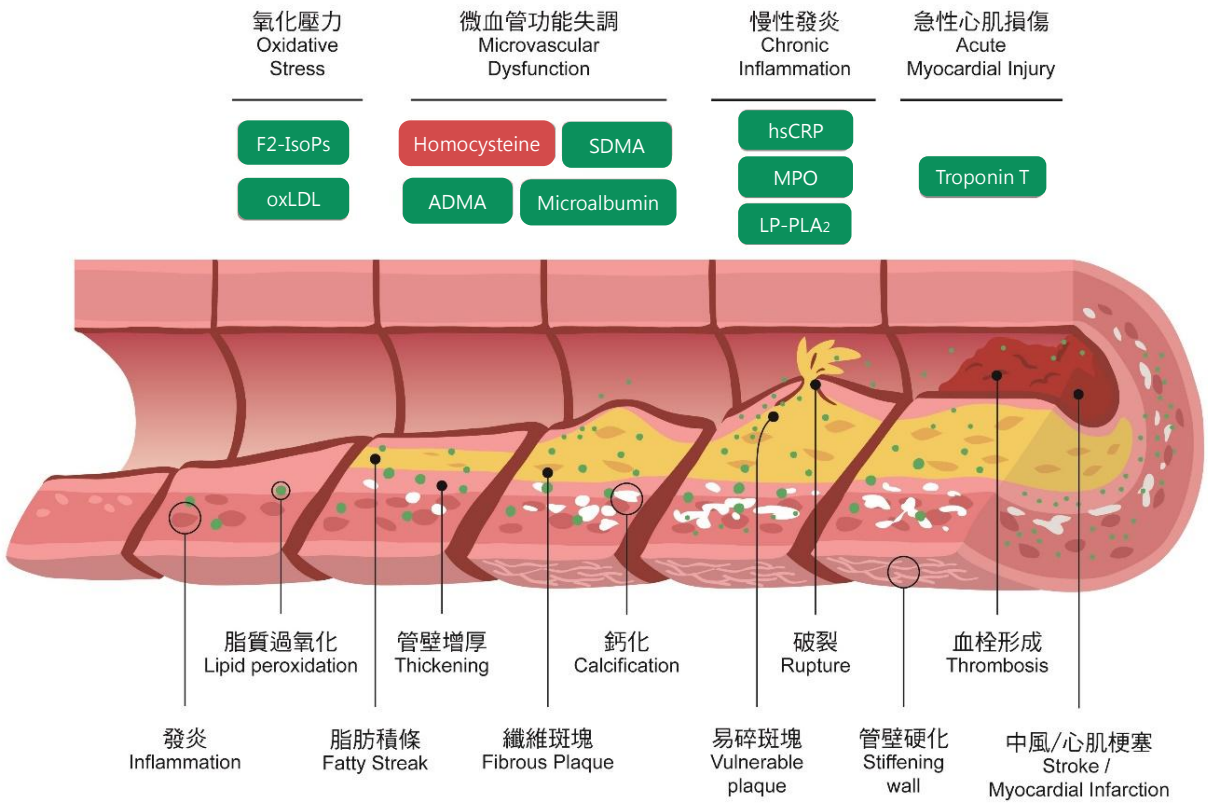
SAMPLE REPORT

Profile: : FNATS: Atherosclerosis Progression Analysis

Atherosclerosis Progression-META®



	Result		Reference range	
#1 F2-IsoPs	1.82		<3.35	µg/gCr.
#2 oxLDL	136		<231	ng/mL
#3 Homocysteine	18.2 ⬆		6.8-17.2 (6.8-12)	µmol/L
#4 ADMA	0.55		<0.61	µmol/L
#5 SDMA	0.56		0.36-0.67	µmol/L
#6 Microalbumin	2.17		<16.0	mg/gCr
#7 hsCRP	0.06		<0.2	mg/dL
#8 MPO	118		<134	ng/mL
#9 LP-PLA2	423		<639	U/L
#10 Troponin T	8.20		<14	pg/mL

Atherosclerosis Progression-META®

The inflammatory reaction is the cause of atherosclerosis, which can damage the inner wall of the artery, oxidized lipoprotein, and cause fat plaque accumulation. At first, the fat plaque caused by the endothelial layer continues to accumulate, leading to the outer layer of the artery to expand outward, allowing the blood vessels to maintain normal blood flow in abnormal lumens. However, as the fat plaque continues to grow and the artery can no longer expand outward, the fatty plaque will begin to protrude into the lumen, causing the diameter of the tube to shrink, leading to atherosclerosis. When the fat plaque ruptures, it will form a thrombus in the blood, further causing obstruction of the artery, bringing on diseases such as angina pectoris, myocardial infarction, and stroke.

Clinical research found that cholesterol in more than 30% of patients with cardiovascular disease are in the normal range, which means that cholesterol is an important but not the only risk factor in the formation of atherosclerosis. Recent studies also suggested that the "inflammatory response" has a non-negligible importance in the process of atherosclerosis, and further makes the "inflammation indicator" more and more valuable in the assessment of cardiovascular health risks.

< Oxidative Stress>

A free radicals is an oxygen containing molecule that has one or more unpaired electrons, making it highly reactive with other molecules. This may result in significant damage to cell structures. Cumulatively, this is known as oxidative stress. In general, to counteract oxidative stress, the body produces an armoury of antioxidants to defend itself. But, an imbalance between the production of free radicals and the antioxidants may lead to many pathophysiological conditions in the body. That is, an imbalance between oxidants and antioxidants is the underlying basis of oxidative stress.

1. F2-IsoPs

F2-Isoprostanes are produced by non-enzymatic free radical-catalyzed peroxidation of arachidonic acid. The level may differ according to the status of anti-oxidation in our body. These products may cause may cause vasoconstriction and platelet aggregation, even the cell proliferation of vascular smooth muscle cells and endothelial cells. Studies have shown that with the rising level of F2-IsoPs brings out eye problems, cardiovascular diseases, liver diseases, kidney diseases, neurological problem and even inflammatory diseases.

2. oxLDL

oxLDL are produced from the oxidation of low density lipoprotein (LDL) by macrophages and endothelial cells, becoming cytotoxic and immunogenic. They are also a potent inhibitor of the macrophages mobility, contributing to the vascular wall progressive inflammatory infiltration and atherosclerosis formation. Weight loss can prevent atherosclerotic plaque progression of and acute coronary events in obese subjects, so that oxLDL could be used a biomarker of cardiovascular diseases.

<Vascular Endothelial Injury >

Homocysteine is one of the typical vascular endothelial injury factors. The increase in the concentration of homocysteine is positively correlated with the incidence of ischemic heart disease, deep vein thrombosis, pulmonary embolism and stroke. Nitric oxide plays an important role in preventing the occurrence and progression of cardiovascular disease. Cardioprotective effects of NO include regulation of blood pressure and vascular tone,

inhibition of platelet aggregation and adhesion of white blood cells to the vessel wall, and prevention of smooth muscle cell proliferation. The generation of NO requires the drive of a methylation cycle. Arginine is converted into citrulline in the endothelial cells via the coupling of eNOS and BH4 and simultaneously produce NO.

3. Homocysteine

Homocysteine is an amino acid that functions as an intermediate in methionine metabolism. The latest medical research shows that homocysteine levels in the blood are very closely related to atherosclerosis. According to statistics, at least 10% of coronary artery disease is related to an excess of homocysteine. Homocysteine produces a shearing force on the endothelial cells of arteries, which induces lipid deposition in the arterial wall, thereby causing atherosclerosis.

Elevated Please check further if the vascular inflammatory index is ideal and whether the antioxidant enzyme synthesis in the body is normal. You can also check whether the diet contains a large amount of methionine (such as red meat, chicken, turkey, dairy products, etc.). If necessary, you should supplement the B complex to help normal metabolism of protein and amino acids in the body, and check the concentration of homocysteine in the blood, also maintain the ideal status which can effectively prevent the risk of cardiovascular endothelial injury.

4. ADMA

ADMA is the principal endogenous inhibitor of nitric oxide synthase. Thus, it regulates rates of nitric oxide (NO) formation. Nitric oxide acts as a signal molecule in the nervous system, as a weapon against infections, as a regulator of blood pressure, and as a gate keeper of blood flow to the organs.

5. SDMA

SDMA is an endogenously produced inhibitor of nitric oxide synthase. However, elevated levels of SDMA occur in patients with vascular disease, especially suffering end-stage renal disease.

6. Microalbumin

Microalbumin is considered to be an important indicator of vascular endothelial injury, and it is significantly increased in vascular injury or inflammation of the heart, reflecting systemic vascular endothelial dysfunction. Studies have found that elevated levels of microalbumin in normal urine increase the risk of arteriosclerosis and insulin resistance; if the microalbuminuria in diabetic patients increases, it may increase the risk of cardiovascular disease and death rate.

< Chronic inflammation>

Inflammatory reactions often occur silently. It can cause cardiovascular diseases such as arteriosclerosis, obesity and metabolic syndrome, which promotes the growth and metastasis of cancer cells, insulin resistance and diabetes. Chronic inflammation promotes hardening of blood vessels, forming atherosclerotic plaques on the wall of the tube. After the plaque ruptures, it causes platelet aggregation and thrombotic factor coagulation, forming a thrombus to block the coronary arteries. Inflammation of arteriosclerosis does not have typical characteristics, so it is considered to be a silent inflammation. How to regulate the occurrence of these inflammatory factors is also an important issue.

7. High sensitivity C-reactive protein(hsCRP)

C-reactive protein (CRP) is made from the liver and is an indicator of acute inflammation in the body, allowing early detection of inflammation and infection. C-reactive protein is also involved in leukocyte phagocytosis and initiation of the immune system, providing an early role in defense within the body. Studies have shown that normal human hs-CRP is positively associated with future cardiovascular risk, and the risk of cardiovascular disease is twice as high as that of low-risk groups. hs-CRP is a powerful independent predictor independent of traditional risk factors such as hypertension, diabetes, age, family history, smoking history, and cholesterol values.

8. Myeloperoxidase(MPO)

It is mainly found in neutrophils and mononuclear spheres. It can use its ability to peroxidize to produce toxic free radicals and eliminate invading bacteria or molds. In recent years, studies have found that MPO, which is induced by long-term inflammatory reactions in the body, causes lipid peroxidation, which leads to atherosclerosis and cardiovascular disease. Therefore, MPO has become a new risk factor for predicting cardiovascular disease.

9. Lipoprotein-associated PLA_2 (Lp-PLA_2)

Lp-PLA_2 Synthesized and secreted by mature macrophages and lymphocytes. It can hydrolyze platelet activating factor to inactivate , and can also hydrolyze lecithin on LDL, causing inflammation. The reaction causes the mononuclear cells to aggregate into the inner membrane, and then the macrophages phagocytose and oxidize the LDL to form foam cells. The foam cells accumulate on the blood vessel wall to form plaques, causing atherosclerosis, which leads to cardiovascular disease.

<Acute Myocardial Injury>

10. Troponin T

Cardiac troponin is the preferred biomarker for the diagnosis of acute myocardial infarction (AMI). The recent development of a high-sensitive cardiac troponin T (hsTnT) assay permits detection of very low levels of cTnT. Using the hsTnT assay improves the overall diagnostic accuracy in patients with suspected AMI, while a negative result also has a high negative predictive value. Measurement of cardiac troponin T with the hsTnT assay may provide strong prognostic information in patients with acute coronary syndromes, stable coronary artery disease, heart failure and even in the general population.

Nutritional Requirements

- Vitamin C promotes the absorption and utilization of folic acid, prevents folic acid from being oxidized, maintains active folic acid, and helps folic acid to function normally.
- Vitamin E can prevent arterial plaque formation, reduce oxidative damage of LDL-C, strengthen blood vessels, reduce blood viscosity and platelet aggregation.
- Essential fatty acid, including alpha-linolenic acid (ALA) and DHA from fish oil, modulate blood lipids; inhibit plateletclumping; have anit-inflammatory activity; reduce fibrinogen, homocysteine, and C-reactive proteins levels; and improve insulin sensitivity.
- Sulfur compounds (e.g., methionine, N-acetylcysteine and taurine) may influence plasma cholesterol and

atherosclerosis.

- Supplement antioxidants (such as vitamins A, E, C, α-lipoic acid, glutathione, N-acetylcysteine, copper, zinc, manganese, selenium and CoQ10) to enhance antioxidant capacity and prevent blood lipid degeneration and deposition on the vessel wall causing obstruction.
- Consider herbal antioxidants supplementation, such as garlic, green tea, curcumin, resveratrol and grape seed.
- Choose more cherries, the flavonoids assist antioxidation.

The food source of nutrients as follows:

Catalog	Food Sources
Fat	
Ω-3 Fatty Acid	Deep-sea fish(Mackerel, Herring, Salmon, Tuna Trout, Flounder, Shrimp, Cod)
Vitamin	
Vitamin A	β-carotene (carrot, parsley, spinach, yam), animal (liver, egg yolk).
Vitamin E	Vegetable oils, nuts, seeds, green leafy vegetables, whole grains, α-tocopherols (almonds, hazelnuts, sunflower oil, avocado), γ-tocopherols (soybeans, corn, rapeseed oil).
Vitamin C	Guava, sweet pepper, vanilla (thyme and parsley), dark green leafy vegetables (cabbage, mustard, cress), broccoli, Brussels sprouts, kiwi, papaya, orange and citrus, strawberry.
Others	
Lycopene	Tomatoes, watermelons, grapefruits, sweet peppers, red heart guava, papaya, mango and other red fruits and vegetables.
Carotene	Carrots, sweet potatoes, mangoes, pumpkins, bell peppers, cantaloupe and green leafy vegetables.
Flavonoids	Blueberries, raspberries, cranberries, strawberries, cherries, grapes, citrus fruits.
CoQ10	Meat, poultry, fish, soybeans, canola oil and nuts.
Lipoic acid	Meat (liver and kidney), green vegetables (spinach, broccoli, peas), rice bran and brewer's yeast.

Lifestyle & Health

- Avoid smoking and excessive alcohol consumption.
- Exercise
- Avoid high-fat diet, less processed food and more fresh fruits and vegetables.
- Regular lifestyle, relax, keep exercising

Testing and Treatment Guidelines

- Oxidative Stress Analysis (#1131)
- Lipid Metabolism Profile 2.0 (#1164)

Atherosclerosis Progression-META®

- Endothelial Metabolic Function Profile (#1102)

Have a functional medical exam every 3-6 months to make sure that all the nutrient is being fully utilized.