

Cholesterol & Plaque

Empowering Information for Understanding How Your Body Works

What is the Role of Cholesterol?

- Cholesterol is an essential building block for the normal metabolism of the body.
- Cholesterol is a lipid (fat). The liver produces 90% of the body's cholesterol (usually during sleep). This is primarily a genetic factor; only 10% comes from food.
- Problem: Lipids cannot circulate alone in the blood stream (fat and water do not mix). They require a transport system.
- Solution: 'Water-soluble' proteins called lipoproteins transport cholesterol in the blood. Think of 'dump trucks' designed to carry specific types of cholesterol. The amount of these lipoprotein 'dump trucks' determines how much cholesterol can be transported.

- There are three main types of lipoproteins that transport cholesterol:
 - ♥ **HDL** – high-density lipoprotein. Referred to as "good" cholesterol, because it *removes* cholesterol from arterial plaque and transports it back to the liver to be metabolized.
 - ♦ **LDL** – low-density lipoprotein. Referred to as "bad" cholesterol, because it *deposits* cholesterol into the inflamed plaque of the artery wall.
 - **VLDL** - very low-density lipoprotein. This lipoprotein is directly related to the level of triglycerides.
 - **Total Cholesterol** is the sum of all three types of lipoproteins. Measuring these and the triglyceride level produces a lipid profile. The Ratio is the total cholesterol divided by the HDL level (the lower, the better).
- Women are frequently told by their physicians that their cholesterol levels and "ratio" are either normal, or if abnormal, their 'good' cholesterol is 'OK'. *The problem with this message is the fact that 50% of women who suffer their first heart attack or stroke have 'normal' cholesterol levels, according to the American Heart Association criteria.*

Why is CRP-HS Important?

- **CRP-HS** (C-Reactive Protein - Highly Sensitive) is a normal protein produced in the liver that circulates in the blood stream. It rises and falls in the presence or absence of inflammation occurring somewhere in the body, such as infections, inflammatory diseases (e.g. rheumatoid arthritis, cancers, diabetes, hypertension and Alzheimer's disease). It remains elevated as long as the inflammation is present, *including the walls of arteries.*
- **Environmental Factors** such as smoking, poor diet, lack of exercise and obesity also leads to *increased* CRP-HS levels. Whereas, avoiding tobacco, eating less saturated fat, and exercising 5 times a week can *reduce* CRP-HS levels.

- Many studies have shown that this simple blood test to measure the CRP-HS levels is *the most sensitive predictor* of a future heart attack or stroke, even more so than the cholesterol level.
- **Jupiter Study.** This study confirmed the important role of CRP-HS.
 - The Jupiter Study included 18,000 apparently healthy patients with *normal cholesterol* numbers and no known risk factors for heart attack or stroke. Study participants had only an *elevated* level of CRP-HS.
 - Participants were given either a placebo or a statin medication (Crestor) for the *Primary Prevention* of cardio-vascular events. After 1.9 years the study was stopped because the Crestor group had **50% fewer heart attacks and strokes** than the placebo group.
 - The Jupiter Study explains why cholesterol levels are “normal” in 50% of women who have a heart attack.
 - The Jupiter Study strongly suggests that when CRP-HS levels remain elevated, despite lifestyle changes (discussed previously), a statin medication should be considered. In fact, in December 2009 the FDA advisory panel recommended that Crestor be approved to prevent heart attacks in healthy persons age 50 and over with a normal cholesterol panel, but have an abnormal CRP-HS.

How is Atherosclerotic Plaque Detected?

- **Atherosclerotic plaque** is an active inflammatory process involving cholesterol molecules being deposited within the artery wall by the LDL lipoproteins and removed by the HDL lipoproteins. During this active phase the plaque contains a soft core and its capsular surface is subject to rupture. During the prolonged healing phase (years) of the inflammation, the non-calcified (soft) plaque becomes hardened with calcium deposits. Plaque progression tends to a slow process and varies widely. However, it has been demonstrated to progress significantly over the course of a single year.
- **Calcified Plaque:** Detected by X-ray, such as an Ultra Fast CT-Scan of the heart and is referred to as ‘hardening of the arteries’ and measures the ‘Calcium Score’. However, this type of CT-Scan does NOT detect the presence of any non-calcified (soft) plaque. It is the soft plaque that appears to present the greatest risk of sudden rupture and potential heart attack or stroke.
- **Non-Calcified (soft) plaque:** Detected with **vascular ultrasound**. Vascular (Doppler) Ultrasound is a simple, *non-invasive*, safe (no radiation) and very accurate office test used for detecting both non-calcified (soft) and calcified plaque. It is used for detection of any plaque within the carotid arteries in the neck (stroke risk), abdomen and femoral arteries (peripheral vascular risk). There is a high correlation between the peripheral arteries (neck and leg) and coronary (heart) arteries. The peripheral arteries are thus a representation of the entire arterial system.

Newer technologies, such as the Multi-Slice CT Angiography, will detect soft plaque, but uses radiation, is expensive, and is not a routine screening method.

Ultrasound of the coronary arteries requires an invasive procedure and is not done as a screening test.

- **CAD Screening:** Carotid Artery Doppler ultrasound is a safe, highly accurate and low-cost method to detect the presence of plaque. When plaque is detected, a full diagnostic ultrasound is performed to establish a baseline of the location, type, characteristics and extent of the plaque. This initial baseline screening is used by the Gunn Towbin Center Heart Healthy staff to chart the future progression or regression in the amount of plaque.

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