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Focus On: Cholesterol — A change in thinking about atherosclerosis—how it forms, how it's treated

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FOCUS ON Cholesterol

ISSUE #5 OF 6 IN AN E-MAIL SERIES

HARVARD MEDICAL SCHOOL
Trusted advice for a healthier life

A change in thinking about atherosclerosis—how it forms, how it's treated

Atherosclerosis, the accumulation of fatty gunk in the arteries, is the underlying cause of most heart attacks. It's easy to think of it as a plumbing problem, where cholesterol-filled plaques stop up the "pipes" that bring blood to the heart (coronary arteries).

A flood of new findings show that it is time to trade in the plumbing analogy for a better one. The new view paints a picture of atherosclerosis as a chronic condition driven largely by inflammation. Blockages take a back seat to widespread damage to arteries in the heart and beyond, from those nourishing the brain, the feet, and everything in between. This new thinking explains how atherosclerosis advances steadily but can explode in an instant.

How arteries get 'sick'

The arteries of an active child who eats a healthful diet are smooth and supple. The inner lining, a layer known as the endothelium, is unblemished. The arteries respond instantly to the tissues' demand for oxygenated blood. But feed that child a typical Western diet and swap racing around the neighborhood playing hide-and-seek with playing video games, and this state of arterial innocence begins to fade. Whitish streaks begin staining the endothelium.

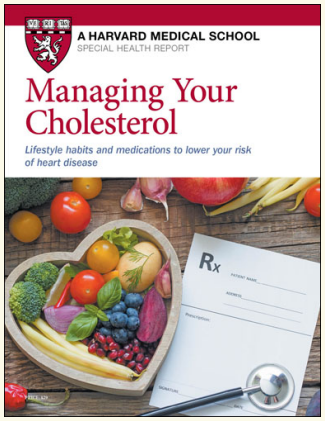
If the teen, then young adult, then adult follows a typical Western lifestyle, these streaks gradually evolve into atherosclerotic plaque—patches of toxic muck that can lead to angina, heart attack, stroke, kidney disease, memory loss, sexual problems, leg pain, and other cardiovascular woes.

From healthy artery to heart attack

Inflammation triggered by damage or stress in the inner lining of an artery sets off the steady growth of atherosclerosis, which can suddenly erupt, causing a heart attack.

In the old view, plaque formed because a person ate too much cholesterol, or his or her liver made too much of it. That made sense, because fatty

Featured Report



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Why do people on cholesterol-lowering drugs still have heart attacks? What role does cholesterol really play? How can you lower your risk of heart disease and stroke? What to Do About High Cholesterol answers these questions and explains why lowering your LDLs (the bad cholesterol) is even more important than previously thought. The report includes a step-by-step method to determine your risk level for heart disease and specific guidelines on how to lower your risk.

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streaks and plaque are full of cholesterol. But we now know that inflammation helps trigger plaque's accumulation.

Atherosclerosis starts with an insult or injury to the delicate endothelium. Any of the following could do it:

- a localized infection
- high blood pressure or high blood sugar
- a barrage of inflammatory signals from excess body fat
- damage from turbulent blood flow
- noxious chemicals from cigarette smoke
- too much fat or cholesterol in the bloodstream.

In the Next Issue

Your cholesterol test: What happens during and after?

Additional Resources

- **Hypertension: Controlling the "silent killer"**

This damage causes some cells in the endothelium to become sticky. Like flypaper, they snare passing white blood cells and entice them to burrow into the lining. These white blood cells stir up a torrent of inflammatory cells and signals.

Over time, a new type of tissue begins to form. It attracts cholesterol-carrying low-density lipoprotein (LDL) particles circulating through the blood. As the LDL slips inside cells, it gets oxidized by the cells' highly reactive contents. Circulating white blood cells sense this change as a threat to the body. They seek out the oxidized LDL and gorge on it. As they fill up and begin to die, they give off more inflammatory signals, perpetuating and accelerating the cycle.

This activity leads to gooey pools of plaque embedded here and there in the endothelium. Each one is separated from the bloodstream by a thin cap.

Spreading plaque

Plaque grows and spreads in different ways. Some plaque grows into the open space inside an artery (the lumen) through which blood flows. These are the bumps and narrowings that an angiogram can detect.

However, most patches of plaque bulge outward, away from the lumen. They don't get in the way of blood flow, and they're invisible to the prying eyes of an angiogram or stress test. In some arteries, especially small ones, plaque covers the entire wall. This symmetrical change is also invisible on an angiogram.

In the old view of atherosclerosis, the biggest bulges caused heart attacks. They can, of course. But the danger often comes from soft plaques with a thin cap that barely poke into the artery. Big plaques and small ones can break open, which can lead to a heart attack or stroke.

Shift in thinking, shift in treatment

The traditional view of coronary artery disease emphasizes blockages. Cardiologists look for them with stress tests and angiograms. If no blockage is found, people are often told they are "fine," sometimes in spite of chest pain and other symptoms.

If a blockage is found, treatment usually focuses on squashing the offending plaque with a balloon and placing a stent to prop open the newly widened section of artery (called balloon angioplasty). Surgery to bypass the blockage is another option.

Balloon angioplasty

In balloon angioplasty, a cardiac surgeon feeds a catheter (small tube) to the site of the blockage and threads a thin, flexible guide wire through the narrowing (**A**). The balloon catheter advances along the guide wire until it's positioned directly inside the narrowed area (**B**). As the balloon inflates, the plaque stretches and cracks, allowing freer passage of blood through the now-reopened artery (**C**).

Yet these treatments are more like delaying tactics than cures. Since they don't fix the underlying problem—atherosclerosis—another plaque in the coronary artery tree or elsewhere can enlarge or burst. Fighting both the atherosclerosis and inflammation is what's needed.

Almost all adults have some atherosclerosis. How do you know how hard to fight it and what to do to protect your heart and arteries?

heart and arteries?

If you are at low risk for having a heart attack, a healthful diet and daily exercise are your key protectors.

If you have heart disease or are at high risk for having a heart attack, you need to attack the disease on all fronts:

- Exercise and eat healthfully.
- Control blood pressure, cholesterol, and blood sugar.
- Take medications to stabilize plaque and stop blood clots from forming in the heart's arteries.

If you're at intermediate risk, eat right and exercise for sure, and choose other strategies based on your risk factor profile.

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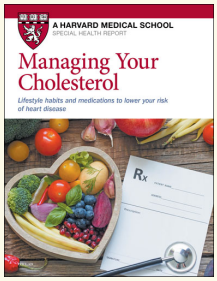

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