Why Cholesterol Has Very Little to Do with Heart Attacks
And Why Silent Inflammation Does
by Barry Sears, Ph.D.

ONE OF THE BEST WAYS TO LIVE A LONGER AND BETTER LIFE IS TO reduce your likelihood of dying from heart disease. If we could eliminate heart disease tomorrow, the average life expectancy of every American would increase by an estimated ten years. Although mortality from heart disease has decreased due to medical advances, the incidence of heart disease is on the rise. More of us are getting heart disease because we aren’t doing enough to address the underlying cause: inflammation in the arteries. Like all silent inflammation, this arterial inflammation results from an increased production of “bad” eicosanoids. Rather than pinning your hopes on some new surgery or drug that may or may not be developed in the future, why not just avoid getting heart disease in the first place?

We are led to believe that elevated cholesterol is the cause of heart disease. As a result we have declared war on dietary cholesterol, and that has also meant a war on dietary fat. The result of that dietary approach has been an epidemic of obesity. That is why the focus of the medical community has shifted to reducing blood cholesterol levels to the lowest levels possible. Not surprisingly, the most profitable drugs (statins) known to the pharmaceutical industry are the primary weapons in this continuing war. But what if cholesterol were only a minor, secondary player in heart disease?

Protecting yourself against heart disease requires far more than simply lowering your cholesterol levels. In fact, 50 percent of the people who are hospitalized with heart attacks have normal cholesterol levels. What’s more, 25 percent of people who develop premature heart attacks have no traditional risk factors at all. So, if elevated cholesterol isn’t the primary cause of heart disease, what is?

Silent Inflammation = Bad Heart

A heart attack is simply the death of the muscle cells in the heart, due to a lack of oxygen caused by a constriction in blood flow. If this lack of oxygen is prolonged, enough heart muscle cells die, and your heart attack becomes a fatal one.

There are several things that can cause the stoppage of oxygen flow to the heart. A rupture could occur in a piece of unstable plaque lining the artery wall. This causes the activation of platelets, which clump together and block blood flow. You could have a spasm in the artery, that blocks blood flow to the heart. More often, it may be due to an electrical flutter, which disrupts the synchronized beating and causes the heart to stop functioning altogether. None of these heart attack causes has much to do with increased cholesterol levels, but they have everything to do with silent inflammation.

You may be asking yourself, “What on earth is silent inflammation?” Even more perplexing, how can inflammation be silent? Silent inflammation is simply inflammation that falls below the threshold of perceived pain. That’s what makes it so dangerous. You don’t take any steps to stop it as it smolders for years, if not decades, eventually erupting into what we call chronic disease.

Eicosanoids and Heart Disease

Eicosanoids were the first hormones developed by living organisms and are produced by every cell in your body. They control everything from your immune system to your brain to your heart. There are two kinds of eicosanoids: those that promote inflammation (pro-inflammatory) and tissue destruction and those that stop inflammation (anti-inflammatory) and promote healing. You need to have both kinds in the proper balance in order to be in a state of wellness. Unfortunately, most of us produce too many pro-inflammatory eicosanoids, which leads to increasing levels of silent inflammation and, eventually, to chronic disease. The Zone Diet was developed primarily to put these hormones back in proper balance.

A variety of factors forge the linkage between silent inflammation and fatal heart attacks. First of all, pro-inflammatory eicosanoids inside an unstable plaque can trigger the inflammation that increases the likelihood of rupture. Often these unstable plaques are so small that they can’t be detected by conventional technology, like an angiogram. When such a plaque bursts, cellular debris is released and platelets rush to the site in an attempt to repair the rupture, just as they would a wound. New blood clots formed from aggregated platelets may plug up the artery, stopping blood flow completely. This helps explain why many people do not die of heart attacks even though they have highly clogged arteries, whereas others do, even though they have seemingly normal arteries. It all depends on the levels of inflammation in these small, unstable plaques.

These same pro-inflammatory eicosanoids are also the culprits behind vasospasm, the second cause of fatal heart attacks. Pro-inflammatory eicosanoids act as powerful constrictors of your arteries and can lead to a vasospasm, a potentially fatal cramp or “charley-horse” that prevents blood flow to the heart.

As if all this weren’t enough, lack of sufficient levels of long-chain omega-3 fatty acids (found in fish oil) in the heart muscle can also lead to a fatal heart attack caused by chaotic electric rhythms in the heart. This condition, called sudden death, accounts for more than 50 percent of all fatal heart attacks.

The Cholesterol Myth

I am not saying that cholesterol has no role in heart disease, only that it is a secondary factor that plays a far lesser role in fatal heart attacks than silent inflammation. If your goal is to reduce the chances for a fatal heart attack, then it’s far more important to decrease silent inflammation than to decrease cholesterol. So how
did the importance of inflammation get lost, and, how did hype
over cholesterol get started? To answer that question, you have to
go back nearly 150 years.

One of the greatest physicians in the nineteenth century was
Rudolf Virchow. Nearly 150 years ago, he stated that atheroscle-
rosis is an inflammatory disease, based on his observations of
autopsies of the very rare number of people who had actually died
of heart attacks. At the turn of the twentieth century, the greatest
physician in America was Sir William Osler. When asked why he
didn’t include a chapter on heart disease in the classic textbook of
medicine, he replied, the disease is so rare that most physicians
would never see it. However, all this began to change.

In 1913, studies by a Russian scientist demonstrated that feeding
a large amount of cholesterol to rabbits induced atherosclerotic le-
sions. As a result of this experiment, physicians began to believe
that dietary cholesterol might be the primary cause of heart dis-
ease. Unfortunately, further studies found that dietary cholesterol
induced atherosclerosis in rabbits, because it depressed thyroid
function. If thyroid extracts were given at the same time as the di-
etary cholesterol, then there was no damage to the arteries. What’s
more, studies in primates suggested that a high-cholesterol diet
only led to accelerated lesions on the arteries, if the arteries were
significantly inflamed in the first place. Although these findings
should have put a damper on the primacy of the cholesterol con-
nection causing heart disease, this was not the case.

The High-Serum Cholesterol Myth

Physicians used to think that we only had to worry about our
total cholesterol levels. Then research found that this wasn’t such
a strong predictor of heart disease. The fact that the most important
drug (aspirin) to prevent heart attacks had no effect on reducing cho-
lesterol (but it does a great job of reducing inflammation) was not
going to get in the way of the great story on the benefits of lowering
serum cholesterol levels as much as possible. Today, lowering cho-
lesterol is the number-one priority of every cardiologist in America.

I challenge the Holy Grail of cardiology that high serum cho-
lesterol is the cause of cardiovascular mortality for one reason
only: the data. Various epidemiological studies have found that
increased serum cholesterol levels occurred more often in heart dis-
ease patients. But that increase was 5 to 10 percent higher in
those who developed heart disease than those who didn’t. To show
how small this difference is, 38 percent of the heart disease-free
patients in the Framingham study had a total cholesterol level of
220 mg/dl or less, whereas 32 percent of patients with heart disease
had a total cholesterol level of 220 mg/dl or less. Further analysis of
the same data indicated that high total cholesterol levels after age
forty-seven appeared to have no impact on cardiovascular death.
The MONICA study in Europe confirmed this lack of linkage
between high cholesterol and death from heart attacks. In France,
subjects with cholesterol levels of around 240 mg/dl had only one-
fifth the number of fatal heart attacks as subjects in Finland who
had the same cholesterol levels. This is called the French Paradox.
Really, though, it’s only a paradox if you choose to believe that total
serum cholesterol is the primary cause of death from heart disease.

Dr. Barry Sears, leading authority on the dietary control of hor-
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