

How the immune system causes heart disease

July 21 2017, by Rahul Kurup



Many suffer from heart disease despite the fact they don't smoke, have healthy diets, and are of a healthy weight. Credit: Tim Marshall/Unsplash, CC BY-SA

Heart disease is among the [leading causes of death globally](#) and imposes a significant burden on the health-care system. We know some of the causes of heart disease: smoking, unhealthy diet, obesity, high blood pressure, diabetes and [genes](#).

But there are also a [lot of people who die](#) from [heart disease](#) who don't have any of these risk factors. [Studies](#) are trying to find out why, and it appears the immune system and [inflammation](#) in the body could be to

blame.

The most common type of heart [disease](#) is coronary artery disease, which affects the [blood vessels](#) of the heart. Coronary artery disease is mainly caused by blockages that affect [blood](#) flow to the heart muscle, which interrupts the supply of oxygen and other important nutrients.

The [most common cause](#) of this blockage is the build-up of fatty molecules called lipids (which largely consist of cholesterol) [leading to plaque forming inside the vessels](#), and swelling (inflammation) within the walls of the blood vessels. This process is called atherosclerosis.

These plaques don't usually cause any symptoms for the first few decades, until there is significant narrowing of the vessel or there is disruption of the [plaque](#) surface (a piece of the plaque breaks off, causing a blood clot) which leads to a [heart attack](#).

How does the immune system affect the heart?

[Recent evidence](#) suggests cholesterol crystals in the plaque that builds up in atherosclerosis trigger the release of molecules from the immune system. These molecules cause inflammation and promote blood vessel injury and plaque instability, leading to heart attacks, strokes and death.

Immune cells have receptors that serve as sentry guards. They sense various potentially harmful molecules (such as foreign proteins, cell debris or damaged DNA), then send out molecules (like soldiers) to remove these "threats". The strength of this response can be heightened as a result of a person's genes - as is the case in some autoimmune diseases.

Elevated levels of these soldier molecules, "cytokines", [have been linked](#) with coronary artery disease. These cytokines can actually do us harm

though, by overstimulating the immune system to try to remove cholesterol crystals in the blood vessel walls. This overstimulation causes inflammation in the layers of the blood vessel wall.

Lowering these cytokines with cholesterol medications (statins) [has been shown to reduce](#) the progression of atherosclerosis, as well as reducing the number of heart events, including heart attacks that cause death.

So this means we now have a marker to tell us who will be at higher risk of heart attack and stroke, even if they have healthy lifestyles. Doctors can perform a simple blood test to measure the presence of this inflammation marker (one of which is "C-reactive protein" or CRP), which can tell us who is likely to be at risk.

Treatment

Heart attack victims are particularly vulnerable in the period just after their attack, because the inflammation in the arteries can lead to more plaque becoming unstable and chipping off. This instability and subsequent plaque rupture is the triggering event that [leads to most heart attacks](#).

Even with optimal medical therapy, the incidence of further adverse events in patients after the initial heart attack is [as high as 20%](#). This is possibly because the current treatment options aren't targeting these cytokines that cause inflammation well enough.

Statins and aspirin both exert indirect anti-inflammatory effects and are [first-line treatment options](#) in patients with coronary artery disease.

Colchicine, the oldest anti-inflammatory drug after aspirin that is still in routine clinical use [has been shown in an Australian study](#) to reduce heart attacks in patients with stable [coronary artery disease](#). Colchicine is

currently used in the treatment of gout and Familial Mediterranean Fever, both of which are inflammatory disorders. There's also [emerging evidence](#) another drug, Canakinumab, blocks a specific type of cytokine, reducing the risk of follow-up heart attacks.

A [recent study published in the British Medical Journal](#) showed an increased risk of heart attacks in patients who were taking non-steroid anti-inflammatory drugs, which are commonly used painkillers. It's important to note this study is observational and demonstrates an association, with the drugs not proved to be a direct cause of heart attack. It advises caution when prescribing or taking these painkillers, until more definitive studies are carried out.

More research needs to be done to better understand the exact immune processes in heart disease so we can find out exactly what the risks are. Targeting risk factors for atherosclerosis has reduced the death rate, but new therapies are needed to stabilise atherosclerotic plaques and to treat heart blood vessel inflammation to reduce the risk of recurrent [heart](#) attacks.

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