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Blood Clots May Be the Root Cause of All Heart Disease

Analysis by Dr. Joseph Mercola | April 14, 2024

STORY AT-A-GLANCE

- The thrombogenic hypothesis asserts that blood clotting is the basic underlying pathological process that causes all heart disease
- When a blood clot forms on your artery wall, it will typically be covered over and broken down. A problem arises, however, when the blood clot is not fully eliminated and becomes a 'vulnerable' point, and another blood clot forms at the same point. Over time this grows and becomes what's conventionally referred to as atherosclerotic plaque
- A clot will form where endothelial cells have been stripped away, or are seriously damaged The blood clot will then be covered over by endothelial progenitor cells, which float around in your blood stream at all times. When progenitor cells find an area of damage, where a blood clot has formed, they attach themselves to that area, creating the new endothelial layer. This repair process can gradually create a thickening inside the artery wall itself
- In almost everyone, the process of endothelial damage and blood clotting is an ongoing process. Which means that problems only occur when the damage/blood clotting process occurs faster than the repair process, at which point you will end up with plaque buildup. This thickens the arterial wall, forcing blood flow through a narrower gap. When a large blood clot forms on top of an existing plaque, in this already narrowed area, you can end up with a heart attack or stroke
- Common causes of endothelial damage include such things as viral infections, high blood sugar levels, smoking, diabetes, heavy metals such as lead and aluminum, and high blood pressure

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In this interview, repeat guest Dr. Malcolm Kendrick, a board-certified family physician and author of the book, "**The Clot Thickens: The Enduring Mystery of Heart Disease**," reviews the underlying mechanisms for heart disease, which for the last century has been the leading cause of death in the U.S.

Of all the books he's written, this is my favorite, as it goes into great detail, giving you the biological understanding of the process of atherosclerosis leading to heart attacks and strokes. He also has solid strategies for lowering your cardiovascular disease risk.

Incidentally, once you understand the disease process, then you can also understand how both COVID-19 and the COVID jab can contribute to heart disease. When asked why he's taken such an interest in heart disease, Kendrick replies:

> *"When I was training as a student in medicine, Scotland had the highest rate of heart disease in the world. Early on the answer for why was, 'Oh, well, it's because we have such terrible diet, and we eat rubbish food like deep fried Mars bars.'*
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> *So, you eat too much saturated fat, the saturated fat gets turned into cholesterol in your bloodstream, and then it's absorbed into arteries and forms narrowings and thickenings, which all sounds plausible if you don't think about it too hard.*
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> *But I also happen to go to France quite a lot, and what I noticed about France was, they eat a lot of saturated fat. They eat more, in fact, than anyone else in Europe, and certainly more than Scotland. So, [this saturated fat] hypothesis certainly didn't work for the French. They have the highest saturated fat intake in Europe and lowest rate of heart disease, and this has been the case for decades.*
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> *If you took all the risk factors for France and Scotland [such as smoking, high blood pressure and diabetes], then the French had slightly [higher risk],*

according to conventional thinking. But, in fact, they had one-fifth [the rate among age-matched men].

So, I thought, this is interesting. It doesn't make much sense according to what we're told. Then while I was in medical school, a tutor in cardiology said ... LDL cannot cross the endothelium. At the time, I didn't know what LDL was, nor did I know what the endothelium was, but it sounded important.

She had been looking at heart disease as a different process for decades ... So, I think that's really where I got started. Once you start questioning what the problem is, you end up questioning more and more and you start thinking, gosh, this is just nonsense, isn't it? This whole hypothesis is just nonsense. So, I started picking it apart."

The Thrombogenic Hypothesis

"The Clot Thickens" is Kendrick's effort to explain an alternative hypothesis for what actually causes heart disease. If it's not saturated fat and cholesterol, what is it? In 1852, a Viennese researcher, Karl von Rokitansky, developed what he called the encrustation hypothesis of heart disease.

Today, this hypothesis has been renamed the thrombogenic hypothesis. 'Thrombo' stands for thrombosis, i.e., blood clots, and 'genesis' means the cause of, or the start of. So, the thrombogenic hypothesis is that blood clots are the basic pathology that causes all heart disease.

> **"We know blood clots cause the final event in cardiovascular disease. We know blood clots cause plaques to grow. Why won't you accept that blood clots are the thing that starts heart disease in the first place? Because then we have one process all the way through,**

> **and it makes sense, because it fits with what you can see." ~ Dr. Malcolm Kendrick**

In a nutshell, when a blood clot forms on your artery wall, which can happen for a number of reasons, it will typically be covered over and dissolved. A problem arises, however, if the blood clot is not fully eliminated and another blood clot forms in the same 'vulnerable' area. This then becomes what's conventionally referred to as atherosclerotic plaque.

> *"The atherosclerotic plaque is basically a buildup of blood clot, repair, blood clot, repair, blood clot, repair,"* Kendrick explains. *"If the blood clotting process is faster than the repair process, you have a plaque that gradually grows and eventually thickens the artery wall until it narrows sufficiently that the final blood clot, on top of the existing plaque, is the thing that can cause a heart attack or stroke ...*
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> *If you cut through the plaque and look at it, it almost looks like tree rings. You can see there's been a clot, repair, clot, repair, clot, repair, clock, repair over the years.*
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> *It's widely accepted that a blood clot forming on an existing plaque will cause the plaque to grow in size. You can find 10,000 papers saying that this is the case. What the mainstream won't accept is that a blood clot on a healthy artery wall can initiate the whole process.*
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> *So, to an extent, all I'm saying to people is, well, we know blood clots cause the final event. We know blood clots cause plaques to grow. Why won't you accept that blood clots are the thing that starts it in the first place? Because then we have one process all the way through, and it makes sense, because it fits with what you can see."*

As noted by Kendrick, the conventional view is that low-density lipoprotein or LDL gets into the artery wall where it initiates plaque formation. It then, inexplicably, stops

initiating plaque, and the plaque continues to grow through the addition of repeated clots.

However, Kendrick says, once you start drilling down into the cholesterol, aka LDL hypothesis, the whole thing starts to fall apart. LDL simply cannot explain the disease progression. Yet despite the many holes in the theory, the idea that LDL causes heart disease is touted as an absolute, indisputable fact.

What's the Mechanism?

In order to justify a hypothesis, you need to have a mechanism of action. Once you understand the mechanism of the actual disease process, then you can put the puzzle pieces together. Kendrick begins his explanation:

> *"Your blood vessels are lined with endothelial cells, a bit like tiles on a wall. Endothelial cells are also covered themselves in a thing called glycocalyx. If you try to pick up a fish, it'll slip through your fingers; it's very slippery. The reason it's slippery is because it's covered in glycocalyx and the glycocalyx is incredibly slippery. It's nature's Teflon.*
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> *So basically, in our case, the glycocalyx [is inside] our blood vessels, to allow the blood to travel through without it sticking, without damage occurring. So, you have this kind of damage-repellent layer on top of your endothelial cells.*
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> *Now, if that layer is damaged, and then the endothelial cell itself underneath is damaged, then the body will say, 'Oh, we've got damage to a blood vessel, we must have a blood clot there because we could bleed out.' So, a blood clot forms on the area of damage, and immediately stops [the bleeding]."*

The blood clot doesn't just keep on growing and growing. If it did, you'd die anytime you had a blood clot. Instead, when a clot forms, other processes step in to prevent it getting too big, which is why every blood clot doesn't cause a stroke or heart attack. Once the clot has stabilized, and has been shaved down, the area is covered over by endothelial progenitor cells, made in the bone marrow, that float around in your blood stream.

When a progenitor cell finds an area that has been damaged, it attaches itself to that area, along with others, forming a new endothelial layer. The remaining blood clot is now lying 'within' the artery wall itself. So, basically, it's the repair process that can lead to plaque buildup within the artery wall. In time, if damage outstrips repair, this can narrow the artery and reduce blood flow.

What Damages Endothelial Cells?

The question is, what can damage the endothelium in the first place? Here, Kendrick uses the SARS-CoV-2 mechanism as an example:

> *"The COVID virus enters endothelial cells through the ACE2 receptor. It prefers endothelial cells because they've got ACE2 receptors on them. It gets into the endothelial cell and starts replicating, then bursts out, damaging the cell. Bingo, you've got an area of damage.*
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> *Of course, added to this, when cells have viruses within them, they send out distress signals to the immune system saying, 'I've been infected, come and kill me,' and so the immune system starts to have a go at the endothelial cells. This is why you can get a problem, because the endothelial cells are being damaged and stripped off.*
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> *Blood clotting occurs at the points of damage and hey, presto, you're having clotting, you're having strokes, you're having heart attacks, which is the thing that people at first couldn't understand [about COVID-19]. Yet it's very clear that what's happening is you've got damage to the endothelial cells.*
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> *Obviously, you and I both know that if you get a [COVID jab], the cells are triggered to produce the spike protein, and these cells are sending out distress messages saying, 'I'm infected.' You have to be very careful if you want to stick something into cells that then says to the immune system, 'Please come and destroy me,' because that's what the immune system is going to do.*

But moving on from that, what other thing can cause endothelial damage? The answer is things like smoking. Smoke particles get out of your lungs, they go into your blood vessels and they cause damage ... You smoke one cigarette and a whole bunch of microparticles appear in your bloodstream, which means endothelial cells are dying.

Luckily as endothelial cells die, another message is sent to the bone marrow saying, we need more endothelial cells and it stimulates endothelial progenitor cell production. These endothelial progenitor cells rush around covering over the areas of damage.

Some smokers have enough repair going on and when you're younger, it's okay. As you get older and your repair systems begin to fail a bit, cigarette smoking becomes more and more of a problem."

Other things that can cause endothelial damage include:

- **High blood sugar levels and diabetes —** The protective glycocalyx layer is made of proteins and sugars — High blood sugar damages the glycoprotein layer, thinning it down in a measurable way. High blood sugar can reduce the glycocalyx layer by as much as two-thirds. This, in turn, exposes the endothelial cells to the bloods and anything else damaging that might be there.

  The damage to the glycocalyx is why diabetics are prone to both arterial and capillary (small vessel) disease. You can't get atherosclerosis in the capillaries, as there's no room. Instead, the capillaries become broken down and destroyed. This in turn can cause ulcers, due to poor circulation in the skin of your legs and feet.

  Peripheral neuropathy as the ends of nerve cells are deprived of oxygen. Also visual problems (diabetic retinal damage) and kidney damage. Blood pressure may also become elevated as your heart has to work harder to push blood through a network of damaged/missing small blood vessels.

- **Heavy metals such as aluminum and lead**.

- **High blood pressure, as it puts stress on the endothelium —** Atherosclerotic plaques (atherosclerosis) doesn't occur unless the pressure is raised, adding biomechanical stress.

Repairing the Glycocalyx

As explained by Kendrick, the glycocalyx layer resembles a lawn, with slippery filaments that stick up. Within this glycocalyx layer you have nitric oxide synthase (NOS), which produces nitric oxide (NO), and you have NO itself, as well as a number of other anticoagulant proteins. The glycocalyx is actually a potent anticoagulant layer, so it stops blood clots forming. If glycocalyx is damaged, your risk of blood clotting increases.

"It's a very complicated layer," Kendrick says. "It's like a jungle full of things that say, 'Don't stick to this, stay away from this.'" Within it, you also have albumin, protein complex produced by the liver. Albumin contains the proteins that help maintain and repair the glycocalyx. A fact that most doctors are unaware of is that, if you have a low albumin level, you're significantly more likely to die of heart disease.

The good news is that while the glycocalyx layer can be rapidly destroyed, it can also be rapidly repaired. (Experiments have shown that in an area where the glycocalyx has been completely stripped off, it can be completely repaired in a single second.) Supplements like chondroitin sulfate and methylsulfonylmethane (MSM) can be helpful in this regard.

> *"If you try and explain that through the LDL mechanism, it just doesn't work,"* Kendrick says. *"They have discovered that if you give chondroitin sulfate as a supplement — which normally is for arthritis and stuff like that — it reduces the risk of heart disease quite considerably. How do you explain that? Well, you can explain that because you're protecting your glycocalyx.*
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> *These are the sort of things that make no sense if you like looking at the conventional ideas of heart disease, but are immediately and easily explained if*

you say, 'We have to keep our glycocalyx healthy and we have to keep our endothelial cells underneath them healthy.

Otherwise they will be damaged and stripped off, and then we will get a blood clot, and if we keep getting blood clots at that point, we will end up with a plaque and eventually one of the blood clots on that plaque will kill you from a heart attack or a stroke."

Blood Flow Restriction Training

A lifestyle strategy that can help repair endothelial damage is blood flow restriction (BFR) training. In response to BFR, your body produces vascular endothelial growth factor (VEGF), which acts as "fertilizer" for the endothelium. You can learn the ins and outs of BFR in **my free BFR report**. VEGF also induces the synthesis of nitric oxide (NO), a potent vasodilator, and it stimulates endothelial progenitor cells.

"NO protects the endothelium. It is anticoagulant — the most potent anticoagulant we have in the body. It's really the magic molecule for cardiovascular health," Kendrick says.

"At one time NO was known as Endothelial Derived Relaxation Factor (EDRF) NO was something no one believed could possibly exist in the human body. NO is actually a free radical. Everyone says free radicals are terribly damaging and unhealthy.

To that I reply, 'Well, you may wish to know that the chemical that is the single most important protective chemical in the body for the cardiovascular system is an incredibly free radical called nitric oxide."

Some anticancer drugs are designed to block VEGF, as the tumor needs angiogenesis — which is the creation of new blood vessels that are required to provide sufficient 'nutrients' Without these new blood vessels, the tumor dies off. Unfortunately, if you block VEGF, you also block NO, which then raises your risk for heart disease.

"These drugs were almost removed from the market," Kendrick says, *"because despite their anticancer activity, they were procardiovascular disease to quite a scary degree.*

[That's why], if you are given bevacizumab or Avastin as an anticancer drug, they now give you angiotensin converting enzyme inhibitors (ACE inhibitors), which are blood pressure lowering tablets, and ACE inhibitors have a specific impact on bradykinin, which increases NO synthesis."

Strategies to Lower Your Thrombotic Risk

In his book, "**The Clot Thickens: The Enduring Mystery of Heart Disease**," Kendrick reviews many different strategies that can lower your disease risk. Here's a short-list of examples covered in far greater depth in the book, as well as some of my own recommendations that I bring up in the interview:

Avoid unnecessary use of nonsteroidal anti-inflammatories (NSAIDs) such as ibuprofen and naproxen — While they effectively inhibit inflammation, they can cause platelet aggregation by blocking COX-2. In other words, they activate your blood clotting system, making blood clots more likely.

Get plenty of sensible sun exposure — Sun exposure triggers NO that helps dilate your blood vessels, lowering your blood pressure. NO also protects your endothelium, and increases mitochondrial melatonin to improve cellular energy production.

Avoid seed oils and processed foods — Seed oils are a primary source of the omega-6 fat called linoleic acid (LA), which I believe may be far more harmful than sugar. Excessive intake is associated with most all chronic diseases, including high blood pressure, obesity, insulin resistance and diabetes.

LA gets embedded in your cell membranes, causing oxidative stress, and can remain there for up to seven years. Oxidative linoleic acid metabolites (OXLAMs) are what's causing the primary damage, including endothelial damage.

Lower your insulin and blood sugar levels — Simple strategies to accomplish this include time-restricted eating, eating a diet high in healthy fats and low in refined carbohydrates, significantly restricting your LA intake and getting regular exercise.

Address chronic stress, which raises both blood sugar and blood pressure, promotes blood clotting and impairs your repair systems. Cortisol, a key stress hormone, reduces endothelial cell production.

Quit smoking.