

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ihda.ie](https://ihda.ie) and TheFatEmperor.com

Ivor Cummins	00:00:40	Today, I have someone who has arguably done more research into the deeper mechanisms and root causes of heart disease than anyone else on the planet. And I'm of course speaking of Dr. Malcolm Kendrick. Great to talk to you again, Malcolm.
Malcolm Kendrick	00:00:54	No, nice to speak to you Ivor. We'll have a few arguments on various things I'm sure...!
Ivor	00:00:58	Ahh, I think we'll align on quite a few vectors, but we'll see. You know, I was reading most of I think your series on heart disease root cause and it went up to about 52 or something at this stage.
Malcolm	00:01:10	Yeah, I'm up to 60, something.
Ivor	00:01:13	Oh, wow. Okay well, I'm a busy guy too, in fairness. But extraordinary, now you cover so many potential root causes that for some people, it may get a little kind of complex as you're in the middle of the series. But then in the latter stages, you begin to close in on your primary ones. But maybe we could start off with just looking at the genuine root causes of heart disease as a kind of pareto. So the top contenders for being primary drivers (although, of course, there are many) - the kind of top primary ones in your mind, and then we work through the list perhaps.
Malcolm	00:01:53	That's a big one. One of the things I've tried to say, and I've written a whole series of articles about what causes heart disease, one of the things I've tried to make clear is that the search for our cause has actually created a lot of confusion. Because what we're looking at really is a process. Heart disease is a process. And it's looking at it as a process that allows you to understand how does that risk factor? Terminology, I do know, you can probably find yourself a language, is the greatest barrier to try to explain things that there is because you use a word and it means it creates a whole series of thought processes. So I've tried to say heart disease is a process, you've got to go back from saying, "What's the cause?" Because then you end up with a situation where people say, "Yes, smoking is a cause. But do you have to smoke in order to get heart disease?" No. You know, diabetes is a cause. You have to have diabetes in order to get heart disease? No. Air pollution is a cause, you know, etc, etc. So you can find all these things. Epidemiology says we have a concept of "necessary and sufficient". And that is where the thinking of medicine has gone for a long time. So is a factor necessary? Does it have to be there? So if you had say, tuberculosis, you have to have the tuberculosis bacillus,

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

otherwise you can't get TB. It's impossible. That's a necessary cause. All right.

00:03:22 Then there's sufficient, which is, is it enough by itself to cause a disease so you can be infected? I mean, we know when the flu is going around, most people don't get the flu. So the flu virus is necessary, but it's not sufficient. Other things stop you getting the flu. So this is where some of the complexities come in. So when you look at heart diseases is to say, is there any necessary cause for heart diseases? Is there any single thing without which heart disease or cardiovascular disease cannot occur? And the answer is no, I have not found one. So there's no necessary cause for heart disease.

Ivor 00:03:59 Now, there's an interesting point. And I've argued with this with Dr. Peter Attia before. We didn't see eye to eye, but one thing in principle I might agree with all right is he said, "ApoB or LDL particles are necessary but not sufficient." Now, in one sense, if you have zero particles, I guess nothing will happen in your body. You might not exist, so they are necessary.

Malcolm 00:04:26 You will be dead.

Ivor 00:04:28 Yeah. But the idea is to use that phraseology that you described, to try and make ApoB or LDL particles appear to be crucial or core, even though not sufficient. But I think in a way, that's a twisting of the principle.

Malcolm 00:04:45 Well, of course it is, you can say that breathing is necessary or metabolism is necessary. I mean, yes, you've got to be alive in order for it to happen. And being alive consists of all sorts of processes that means you're alive. But that is reductio ad absurdum, if you like, and you can get into these ridiculous arguments about this sort of thing with people. When I say initially, when people are looking at disease causation in the mid-19th century, this is when people started looking at disease this way, they were initially looking at infectious diseases, because that's what killed people. So they were looking for things like syphilis and gonorrhoea and TB and whatever, whatever. And that worked pretty well as a model, because you could then isolate the necessary cause. So you do have a problem with heart disease, there is no necessary cause, there is no sufficient cause. So therefore, you got to say, "Okay, is there a necessary process that has to occur that without which nothing can happen?" So you start to focus down on process rather than cause. I mean, yes, of course there are. And this thing it causes is an accelerating factors and additional factor. You know, something like smoking, the Japanese for example,

smoke an enormous amount. They are, currently I think the heaviest smokers, there are probably other countries, but in the western, what you call an industrialized, westernized world, they're the heavy smokers. At the moment, I think 60% of Japanese men still smoke, and yet they have an incredibly low rate of cardiovascular disease. It's always been really, really small and it's got smaller.

00:06:25

So smoking in and of itself is not a necessary cause but kind of accelerated. I always like to say to people, "If you've got other things going on, that means that you're increasing your risk of dying of heart disease, if you smoke, if your underlying risk is this size, then smoking will do it to this. The Japanese risk is down here. So smoking actually doesn't make a huge difference. So I think when you're looking at a process, you're also saying, it's about a number of factors operating together, some of which are more important than others. I have only found one disease condition, which without any other risk factors being present, can cause rapidly accelerating heart disease. And that condition is a weird one, which is sickle cell disease. And people would never look at sickle cell disease as heart disease, because why would you feel like if they don't appear to have any correlation whatsoever. And you said, "Well, how on earth can sickle cell disease pulls out heart disease?" I was looking at a case history of a 14-year-old boy. He was admitted to a hospital with gangrene of his right foot. He had gangrene in his right foot because his blood supply in his leg was so compromised due to advanced atherosclerosis, that his foot eventually had to be amputated. This is a boy who also had a stroke when he was five. His brother died when he was three, of cardiovascular disease. So you're thinking, "Well, there must be something going on." What we have with this person is multiple arteries in their body, full of atherosclerosis, calcified atherosclerosis, late stage atherosclerosis, and he's 14. There wasn't an artery in his body that wasn't calcified to the atherosclerosis. And by the way, there's like no other risk factors. That's one of the things that they thought was that this boy had no other risk factors for heart disease.

00:08:20

And the other interesting thing about sickle cell disease is you can get atherosclerosis in the arteries in your lungs, which is extraordinary unusual things. As you age in your lungs, because the veins and the arteries of the other way around in your lungs, things leave the heart. Anyway, never mind on that. So you're thinking what we're looking here at what is actually there was no other, this was unnecessary, it was a sufficient cause of heart disease, nothing else was required. And I like to look at these extremes, because I think when you look at these super

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

extremes, you think there's something going on here that we should be interested, into which I said, "Well, what on earth is happening in sickle cell disease that could cause significant advanced, calcified atherosclerosis throughout his entire body? And if you think of it in terms of smoking, or LDL, or any of these other factors, they didn't exist, they weren't there. So you're left saying it can happen to somebody with nothing else but one condition. And I think that's sort of interesting to me, or should be interesting to anyone.

- Ivor 00:09:33 It certainly will be interesting to any engineering problem solver. And I found a fastening when I heard about it from you, actually, not too long ago. So when you find something dramatic and single factor for a massively multi factor problem, it is hugely important because it brings you towards the root of what's going on mechanistically or the process. Absolutely.
- 00:09:56 Now, when you pick something like high cholesterol or hypercholesterolemia that shows an increased risk, that's a very different thing and is of less value. Right? But what you're talking about is extreme and unique factor that is sufficient to cause enormous atherosclerosis in a guy who's a kid - with no other factors. Let's dwell and talk a little about what that does say or go through the mechanisms of that sickle cell.
- Malcolm 00:10:26 Yeah. Well, that also relates to and I hate to jump. Sorry, I'm being like a politician here. Let me answer another question, which is not the one you asked me, but I'd like to answer anyway. I also like to say, "When else can you get children dying of heart disease, heart attacks, myocardial infarctions?" There is another condition where this happens. It's called Kawasaki disease, which most doctors all have heard of. Most other people haven't called Kawasaki, not after the motivate motorcycle manufacturer. But Dr. Kawasaki from Japan because Kawasaki disease is far more common in Japan than anywhere else. No one knows why, no one even knows what the disease is. However, what it is, is a disease that comes in and is an acute, what they call a vasculitis, it causes an acute vascular inflammation for about two to three weeks, and then it goes away. And then most children then recover, everyone recovers, most of whom have no further problem, but some of them are left with such severe damage to the coronary arteries, That the arteries have got aneurysms which is big...kind of "balloon areas" - which can then burst. So it's not quite the same as a normal heart attack. But an aneurysm is normally a very late stage development heart disease. It would be something you would see in, you may have heard of ruptured aortic aneurysms, which is where you get a ballooning of the

aneurysm in your chest or abdomen, and they can burst which is obviously not a very good thing to have happen. Because then the bleeding is intention; you die. But that is a very late stage of atherosclerosis. And here we have four-year-old children who can diverse aneurysm, having had a three-week or four-week disease, which all its doing is damaging the artery itself quite severely for a short period of time.

00:12:12

So there is actually a link between Kawasaki disease and sickle cell disease, which is that the lining of the arteries, the vascular lining, vasculitis. Itis means inflammation. So appendicitis is inflammation of the appendix, and tonsillitis is infection of the inflammation of your tonsils. So vasculitis is inflammation of your vascular system, which basically means the lining of the arteries and your veins is damaged. So when you say, "How does that link to sickle cell disease?" The answer being that sickle cells is why it's called sickle cell disease, sickle as an sickle moon, as in sickle shaped like a sickle, hard pointy end. If you ever want to look at the picture of a sickle cell, it's got basically sharp ends, a normal red blood cells nice and round and squiggy, and donut-like, and it's designed to switch and move into smaller blood vessels. A sickle cell is harsh and abrasive. It's like putting a sandpaper into your blood vessels. It's not hard to imagine that a sickle cell bashing through your arteries at high Speed is crashing into the lining of your arteries and is stripping off and damaging the artery wall as it does so. In fact, that's what this article says. Basically the sickle cell is causing mechanical damage to the lining of the arteries and it's basically causing repeated damage all the time going on within the arteries. I mean, this 14-year-old boy is not the only one. I mean, normally sickle cell disease kills people when they're very young, of various other things. So no one's really looking for cardiovascular disease. And I must say if you live long enough to die of cardiovascular disease, you've done quite well.

00:14:00

That's not the case anymore. Because they do things like plasma apheresis and they change your red blood cells over, and people are living longer. So it's now being more increasingly recognized that you've got a long term problem. The other thing with sickle cell is, is that your spleen, because the spleen is the thing that gets rid of red blood cells, it breaks them down, it's one of his jobs; does other things. Splens become very large and sickle cell disease sort of become much, much bigger. And they're quite often removed, because there's a danger of rupturing them, and then they burst and then you bleed to death. And if you remove sickle splens from sickle cell disease patients, and then look at their arteries, they're full of atherosclerosis. This

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

has been known for some time. It's not a unique finding, this is a consistent finding of sickle cells.

Ivor 00:14:45 And just one quick question not to interject, but did you say veins with atherosclerosis or arteries?

Malcolm 00:14:52 Well, they don't... there is no condition. I have found anywhere ever. And I've looked, and I really looked. There is a thing called venosclerosis, which is sclerosis of the veins, but that's actually a completely different disease. And atherosclerosis, it isn't atherosclerosis of your veins.

Ivor 00:15:15 Exactly. I was just wondering for a moment there. Was there a very rare exception? Did the sickle cell actually managed to cause a problem with veins? But no, it's still arteries in that case.

Malcolm 00:15:26 it's still arteries, and it's in the lungs. I mean, the blood pressure in your lungs is normally about a fifth quarter, a fifth of that in the rest of the body. But in the veins, blood pressure is about a 15th or a 20th [of the arterial pressure]. And also the blood flow is very much slower. I like to think of it as an artery is a "white-water". You know, if you went whitewater rafting in the north, or you'd be bouncing all over the place, if you went whitewater rafting in a vein, you'd be sort of drifting gently along as the vein used its way back to the heart. So I mean, essentially the speed, the biomechanical stresses on veins are enormously less than in the arteries.

00:16:04 So I've never yet found atherosclerosis. So, in answer to my previous question, is a necessary factor for atherosclerosis and cardiovascular disease? The answer is, you gotta have a high blood pressure - because otherwise you can't get it. Of course, what you can do is you can take a vein from the leg, and stick it in the heart as a coronary artery bypass. So you can turn a vein into an artery. And this is what a coronary artery bypass is, essentially. And then what you find is that the vein rapidly develops atherosclerosis. So within, I think, five to seven years, 70% are blocked up again with atherosclerosis.

00:16:44 So, make a vein do the job of an artery, and it becomes atherosclerotic. And that basically means putting it into a high blood pressure, high turbulent, and biomechanical environment, and the same thing happens to it. So the necessary factor you would have to say is, enough blood pressure or biomechanical stress or whatever term you want to use for that, without that nothing happens.

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ihda.ie](https://www.ihda.ie) and [TheFatEmperor.com](https://www.TheFatEmperor.com)

- Ivor 00:17:06 Yeah, Malcolm, and this is mirrored in many other papers, investigations that they acknowledge. So the turbulent flow that you're talking about the whitewater versus the laminar flow in the veins, very sluggish. But they also know many other mechanisms beyond direct damage, that laminar flow, you know, with the glycocalyx and release of nitric oxide, it's just a much more favorable environment than the turbulent flow, say, at junction points in arteries, where there's a localized turbulence, and that's where atherosclerosis much more likely develops. So I think all of these processes tied together beautifully. And there's no real caveats or exception to what you described there in principle.
- Malcolm 00:17:52 I don't think so. I mean, yeah, you can say the word is atherosclerosis whether these thickening is in plaques, develop in the artery system. The common displaces are the arteries in your heart. And you can imagine your heart is pumping away 70 times a day. Someone described it as like stomping on a garden. That was 70 times, it's 70 times a minute. In fact, the blood flow in the arteries stops when the heart contracts, in the rest of the body, obviously, it goes through. The blood flow is, because the heart is contracting so hard, contracts the arteries so tightly, the blood flow stops when the heart is contracting, and it can only flow and the heart relaxes. There's almost the opposite way around for the rest of the body.
- 00:18:33 So you can see that if you're squishing an artery hard, 70 times a minute, that the poor little lining of the little endothelial cells that sit there are being put under a heavy-duty amount of stress. So that's the most common place where that happen. The second most common place is in the arteries and the neck or the carotid arteries. And this is where the blood comes out of the heart, around the big aorta (the aortic arch), maximum pressure, changes direction, heads up into the brain. So the arteries here, the carotid arteries is a second most common place to develop atherosclerosis and plaques. And then you've got renal arteries, and blah, blah, blah, but it tends to be at points of maximum, biomechanical stress, whatever word you want, if you're an engineer, you probably think of a better word than I can. But it's not surprising at a point where the blood is going like that. And then that's the term direction and then down like that. That right around there is where a lot of this stuff is happening.
- 00:19:35 This all again, fits with, it's about putting the arteries because the arteries are really the only place it developed under greater stress. And that's where it happens. But obviously, there's other factors as well, because not everybody... it doesn't happen to

everybody. So you have to have other things on top of that for it to happen. And that's kind of, I think, the link together, isn't it, where were you saying, we're looking at a process, how many factors do you have to have operating before that really becomes an issue? And the answer is, I don't know for sure. But it has to be more than one.

Ivor 00:20:14 Yeah, but we can infer from all the literature and all your research what the major ones are and roughly an order of importance, I guess. But like you say there, you could have all that stress or biomechanical stress and turbulence in an archery in someone who's running and doing healthy exercise. But if they don't have the other problems, X, Y, Z, W, they may have no atherosclerosis in their 60s, even though they have the necessary component, like the stress in their arteries, but it's not sufficient really, because they don't have other problems taking advantage of what the stress does to the artery.

Malcolm 00:20:51 Well, absolutely. I mean, you also got to remember, the body is constantly able to repair itself. We have to remember that we're not... and sometimes like the potholes on the road. I mean, potholes are gradually forming cars, go over them, the rain falls. Well, if there's freezing temperature and ice forms, and the road will crack. And if the council's got lots of money, they'll come along and they'll smooth it over. If the council says, "I can't be bothered repairing this" then pretty soon you're going to get ba-doosh....! And so, it's also a fair system that you have to bear in mind. Because yes, there's no doubt in my mind that the endothelial cell, which is the cell that lines the arteries is under constant pressure, and we're losing them quite regularly, repeatedly. I'm sure that's happening. You can see evidence. In fact, I was just looking at a paper that showed if you smoke, (non-smokers) you smoke one cigarette, right, then you can spot so-called "micro particles". And micro particles are released into the circulation when endothelial cells die. This is a dead endothelial cell.

00:21:58 So one cigarette causes an increase in micro particles. But at the same time, it causes an increase in another type of cell, called "endothelial progenitor cell", these are progenitor cells that are created in the bone marrow. They wander around the circulation and if they see an area of damage, or is necessary because a cell is missing or whatever - they cover it over and regrow and reform the endothelial barrier. So if you smoke a cigarette, you both get micro particles, you're killing endothelial cells - and then you get replacement, i.e. endothelial cells are being produced. So this is the body's healing system.

00:22:35 That's happening all the time. And the things that cause if you damage the healing system, or if you downgrade the healing system, this is just as bad as increasing the damage. If you do both at the same time it's really bad. So then really what I would say is to consider that atherosclerosis is a balance between damaging happening processes, and repair processes. And if a repair is happening, or keeping pace with it, you've got better prognosis. If the damage starts to outpace repair, you're running into problems, because then the repair doesn't happen properly. The damaged area becomes gradually increasingly an area of focus for further damage and you just develop a large pothole. I mean it's not a pothole, but that's what I'm kind of talking about here in a way.

00:23:20 So so long as the council comes in, repairs the roads sufficiently quickly, you're not going to have a problem. If they stopped doing that, you start running into problems. So when you look at things and say, "What are the things that can interfere with the repair systems that would be repairing this damage? What are those things?" Well, one of them is age, unfortunately. Which is why as people get older, it is the number one risk factor for cardiovascular disease, because I'm afraid as you get older this happens. Chronic kidney disease, which is known - is a severe problem for people dying of heart disease. And chronic kidney disease, you also get a reduction in endothelial cell production in the bone marrow and see cardiovascular disease accelerates steroids, which, you know, we're using a number of conditions. Steroids or corticosteroids produced, well they're synthetic steroids, corticosteroids - so called because they come from cortisone, which is the steroid produced in the adrenal glands. And the artificial steroids, they are fantastic at dampening down inflammation, because they use them and transplant patients. They use them in things like inflammatory bowel disease, Crohn's disease, asthma, etc. Really widely used.

00:24:34 Unfortunately, if you take long-term steroids, it stamps down the repair processes in your body, and they increase the risk of heart disease, greatly – by 400% 500%. And in fact, people who've got a condition known as Cushing's syndrome or Cushing's disease, where they overproduced steroids due to tumors in the adrenal glands, these people have 600, 700, 800% increased risk of dying of heart disease. So I do look at people who tell me that heart disease is an inflammatory condition. And I say, "Well, the most potent anti-inflammatory known to nature, the most potent anti-inflammatory we have - increases the risk of dying of heart disease by 600 to 800%, Probably more than this. So please don't tell me how "heart disease is due to inflammation."

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](https://www.ihda.ie) and [TheFatEmperor.com](https://www.TheFatEmperor.com)

- Ivor 00:25:22 You know, that's a great point worth reiterating constantly. So people think, "Oh, inflammation is bad." And in one sense, they're right. Anything that's driving inflammation in your body is not a good thing and it can also drive a lot of problems and disease. But the inflammation is only the response to the injury, really. So you got to focus on the injury.
- Malcolm 00:25:43 Yes. Well, I call inflammation...healing.
- Ivor 00:25:50 Mostly. And the classic example is - I think they did one trial on badly swollen ankles, when they were twisted. And for a long time, they used to cool them and wrap them up and raised them and tried to minimize the swelling. And then they did a trial and found that actually, you're kind of better off leaving it do its job and not intervening too much with drugs or trying to cool or minimize, and it will actually self-repair and resolve a little faster. So yeah, "good inflammation". But I think chronic inflammation from constant chronic injuries to your body through various root causes, that that's where the issue is, because the inflammation response can then partially contribute to the problem if it's really chronic and everything goes haywire...
- Malcolm 00:26:36 Well, I think you're right. I mean, I wish it was straightforward. Like everything, a little something, a lot of something, you have to look at it from more than just a simplistic point of view. Yeah, chronic inflammatory process itself, or whatever it's triggered by in things like Crohn's disease, or asthma, can become the problem in itself. So there are certain conditions where you have to dampen down the inflammation, or it will kill you. And that's why steroids are widely used. I mean, you get immunosuppressants, obviously. Clearly, immunosuppressants are used to prevent rejection in transplant patients, and what they're needed, because otherwise, you know, you're going to lose or reject the transplant. But actually, interestingly, they vastly increase the risk of heart disease. So immunosuppressants, well, these are double edged sword. If you can get rid of the thing that's causing inflammation, great. If the inflammation is causing more damage, then it is resolving its own problems, then great. But you have to be very careful when you're dealing with this stuff. You're best to look at why you're getting inflamed in the first place. Try and reduce that in other ways. And if it's due to some people carbohydrates or whatever, or if it's due to some people with omega 6, fatty acid balance or whatever, these things can trigger an unnatural inflammatory process. So get rid of them. You know, get rid of them. Because then there's no inflammation to be "damped

down". You don't need to give anti-inflammatories and then you won't have a double problem on your hands.

00:28:12

I mean I know you're clearly very interested in the in the blood glucose insulin resistance issue and I think this is extremely important. There's no doubt to that, in that we know that if you have a raised blood sugar level - this severely damages...you talked before about "the Glycocalyx", most people have never heard of it. Most doctors, in fact have no idea - I haven't yet spoken to a doctor who's ever heard of it. And when I mention it they're like, "Gosh, really?" "Yes, it really exists. It's not some kind of weird woo-woo medicine thing." The glycocalyx as you know is a little forest of glycoproteins. And I said, "If you were to pick up a fish, you know what happens? Flips straight through your hand. Not all fish do this, but most do. And the reason for that is they're covered in glycocalyx. That is their protection mechanism. It's slippery, it allows them to swim quicker, it prevents nasty things getting into them. It stops infection and pollutants getting into them. So we as humans are lined, our arteries and our veins and all our blood vessels are lined with glycocalyx. It's extremely important. It's anticoagulant. It's got vasodilator effects and it'll make your blood vessels open up. It's got enzymes inside it called nitric oxide synthase, which produces nitric oxide. You know, people take GTN tablets to open up their arteries and get rid of angina. So, all these things are going on on the glycocalyx.

00:29:37

If your glycocalyx says is this thick, then your endothelium, your arteries are being protected. If it narrows down to this thickness, then the lining of your arteries is becoming exposed. And one thing that really does this is diabetes or high blood sugar level, or pre diabetes, or metabolic syndrome, or whatever terminology you want to use for it. So you really, really need to ensure that your blood sugar level is kept under control. And I think this is one of the... it is a very important... I can say, "What's the most important factor for heart disease?" If you say, "sickle cell disease." But most of us don't have sickle cell disease, it's not an issue. You're talking about our population on the planet Earth, which of the factors that is existing that is most likely to affect most people? And then we're probably talking about - whatever term you use - insulin resistance, will raise blood sugar levels. That raised blood sugar level. Also insulin itself is quite a significant problem. A raised insulin, well everyone thinks insulin is good so they don't worry. Because if you go back to 1920, when children aged whenever died of diabetes, type 1 diabetes - and then insulin came along and suddenly they were alive. So we had this thing of "blood sugar bad, insulin good". And that sort of duality still exists in

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

- everybody's mind. Well, the answer is blood sugar is bad, and blood insulin levels are also bad. You have to keep them both down as much as you possibly can, because they're both damaging to the system.
- 00:31:18 And so on a mass population point of view, yes! Is it the most important single factor, which you have to look at and keep under control? I think probably it is - now - in the West, where whatever proportion is overweight, or pre-diabetic, and blood sugars is a big part of the problem. This is something that is absolutely, really important. So what's this? I said, "Look at process, don't look at cause." I said, "There are some causes more important than others. You know, if you smoke -then stop smoking you idiot; you know?"
- 00:31:54 There are other things that are incredibly important. Air pollution is remarkably important. And then people say, "Well, how does air pollution cause heart disease? How can that cause heart disease? Where's the link there in your hypothesis of endothelial damage?" I say, Well, I can show you paper after paper that shows what they called DM 25, I think is the term I might have got wrong there, maybe it's DM 2.5, which is particulate size of pollutants. Once you get enough small pollutant, particulate size pollutants that go into your lungs, get into your bloodstream, and you can see them. You know, I talked about micro particles, you can see micro particles shedding all over the place. Air Pollution strips your endothelium, like smoking does. Same process is happening. Air pollution is as bad as smoking. I think if you have really badly polluted environment, probably, yeah?
- Ivor 00:32:53 And there was even a question mark. I mean, sometimes we see these ancient people found with arteriosclerosis. Again, the data is not very strong, but people suggest, "Oh, well, they were hunter gatherers, so why did they get it?" But of course, in cold times, they were in these tiny huts with fires internally, constantly. So I mean, even when you look there, not to say that's the explanation for ancient people getting atherosclerosis by any means, but it's just one you can throw out there. And it has a lot of science behind it.
- Malcolm 00:33:25 Well, absolutely. I mean, you can make any argument about why people 10,000 with atherosclerosis and you'll never know the answer to it. But there were factors around it, you can still say, would have been there around there, then. I mean, one of the very interesting ones I looked at was lead pollution from petrol. Obviously, when it first came in, as usual, the experts told us, "Lead is completely safe. In fact, look, here's my daughter standing behind an automobile. She's been doing so

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

for five years, and at the moment, she's still alive," sort of stuff. And the guy that put lead into petrol? At one point over 200,000 tons of lead were being pushed out into the atmosphere, in the US alone. Lead, right? Now, when you look at it, you say, "Well, how does lead cause a problem with heart disease?" Well, the answer is that lead gets into your body and doesn't clear easily, you can see that it damages, there is very strong evidence of lead poisoning or chronic lead, whatever term you want to use, causing damage to the endothelial cells. And it sits there and it continues to damage them. Year after year.

00:34:36 I read a paper recently saying they believe that lead poisoning from exhaust fumes cause more deaths in the US than smoking over that period. Whether this is true or not, I don't know. But it comes to another wonky thing when I was thought this was were medicine. It's where you put them on chelation therapy. Have you ever heard of it?

Ivor 00:34:57 Yeah.

Malcolm 00:34:58 Which I thought, "Yeah, right. This is definitely woo-woo stuff." So you put stuff into the body – EDTA - which is actually the same stuff as you put into blood bottles to stop blood clotting, if you send them to the laboratory, which sounds like just nonsense. But apparently, it can take heavy metals out of your system. It binds to heavy metals and takes them out of your system. And they did a study, I can't remember the name of it, in the states, to prove that chelation therapy doesn't work. And what they managed to prove was that it does work. It worked.

Ivor 00:35:30 Oops!

Malcolm 00:35:30 Oops! They also were like, "Well, we'd like to double check this because we don't, ah...?" Well you know it was hilarious, kind of. They did this study to prove that it was nonsense, found that it worked, and then tried to say that their study was nonsense. You know? Actually, I just thought, "Crikey". It works, you know?

Ivor 00:35:50 Well, the bias is actually extraordinary, I would say the bias out there, and a lot of people are naive and they think "Oh, well, you know, all the scientists are just doing the science and whatever comes out comes out." But you go through thousands of papers. And it's incredible at the cognitive bias or desire of the researchers tilts things, puts the finger on the scale at every turn in terms of dosages for example. You know, if they want something to work, they make sure they max dose in the trial. If

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

they want something like you say to not work, they make sure they stay really careful with a teeny dose, and then they won't reach significance. So it's fascinating. But there was a trial, I'm not sure if it's the one you're referring to. It was called COMET, I think.

- Malcolm 00:36:34 No. I can't remember what mine was called, but it wasn't that.
- Ivor 00:36:37 It wasn't that. Well, I could be incorrect too with the name...
- Malcolm 00:36:40 Yeah. Well, there may have been another one, so I'm not going to contradict you. As you know, somebody once pointed out to me, "If you read two clinical paper per day, by at the end of the year, you'll be 55,000 years behind in your medical reading." And that was about 20 years ago, so probably a million years. But there was no way you could keep up with all this stuff.
- Ivor 00:37:01 Absolutely. Well, this trial, what was interesting to me is the last few weeks, I was doing a little research on calcification regression, because we've seen some phenomena in some documentary work we're doing where we see some regression. And the only two published papers really out there demonstrating regression in humans - for whatever it means or its implications - but demonstrating....are Dr. William Davis, (cardiologist Wheat Belly), and he used as it happened, a mild statin, which won't really reduce calcification, so you can put that aside vitamin D, low carb diet, magnesium, fish oil, and the synergistic mix of things.
- 00:37:40 So that was one that demonstrated regression in half of the people, but the other one actually was chelation. And this is a small trial, and they got an average, I think, 38% reduction in calcification with EDTA. I think it was a specialized form or high dose, but they also put in a whole array of micronutrients on fairness that's confounded. But still, it's the only other regression in human study. That's an actual trial that I know off. So, interesting.
- Malcolm 00:38:12 Well, I think it is interesting. I mean, interestingly, of course statin is increased calcification. So putting the statin, it wasn't beneficial. Again, I always refer people to the fact that the body is capable of repair. It doesn't just sit there going, "Oh, I've got cancer fighter." I mean, I think it's difficult to get rid of the calcification in arteries, it definitely seems to be because it's kind of like, I've got a scar on my wrist, and I didn't try to commit suicide, but I did try to break into my own house one day, and cut my wrist on a window. And it's now whitish, it's whitish because it's got calcified. That's never going to go away.

But that's how it ended up. So I think getting rid of calcification is tricky, but regression of plaques, but I'm sure this is going on all the time, if your repair systems are operating well. So I think that you know, give the body a chance, put the things that it needs. If it's lacking micronutrient, give it some micronutrients.

00:39:14

And you know for well that Kilmer McCully was the man who years ago found children who had too much homocysteine, which is just a breakdown of product of various proteins. If you add vitamin B into their diet, then the homocysteine level drops. And this is true for other people. And in fact, I was just looking at a study the other day, from Cambridge, not heart disease, vitamin B, B1, B6, and folate, I think because that is a B vitamin, you could prevent the progression of Alzheimer's and people with high homocysteine levels. Yeah, people going, "Oh my god, what is it about Alzheimer's disease?" You know what, actually there have been studies showing you that for about 30% of people, this is what you do. You reduce the homocysteine level." Homocysteine, we know damages blood vessels, etc, etc. So I mean, we are looking at... anyway, you know yourself, as you say, going back to your original comment on this, is the research unbiased? Are the researchers out there pure objective searchers after the truth? The answer is, if you believe that, I have a really good offshore account, I'd like you to pay money. And it's a fantastic 50% interest rate every year, until you find your money's gone. I mean, the reality is, I mean, I just did a quote the other day from Richard Horton. And Richard horton is the Editor of The Lancet. "50% of the scientific literature published is false." Okay. 50%. I think it's higher than that. But if he's saying it, this is the problem that we're running into, you know? I look at things like sickle cell disease, and think that's interesting. Has anyone else done any work on it? No. It's just a dead end.

00:41:20

Kilmer McCully who wrote homocysteine, and the damage that it caused the arteries and the risk of heart disease, he got booted out of Harvard. And then when he dared to tell people about his ideas, Harvard University phoned up the people he've been speaking to and said, "Do not listen to this man. He is not associated with Harvard. We have nothing to do with him." He was hounded and then secretly, when people try to interview him for another job, if Harvard found out about him, that he was going for another job, he phoned the institution up and said, "Do not give this man a job." I am not making this up. This is being published. It was in the New York Times about three years ago. I mean, it is extraordinary, because we're up against this. The thing is that people who are trying to actually find out

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](https://ihda.ie) and TheFatEmperor.com

what causes heart disease, look into it to go into interesting work, their funding is just...

Ivor

00:42:17

Yeah. It's not conspiracy theory, it's just a modern capitalist society and everything is profit driven. And naturally, finding out things that undermine existing highly profitable and accepted orthodox opinions on how heart disease works, well, those things are a massive threat, not just to profit centers, but also to the academics and their pride, having said something for 30 years, and now something else is going to make that thing look trivial. So everyone involved in the leadership of these matters, that's another key point, everyone involved in the senior leadership of heart disease has something to lose if something comes out that undermines it all, like if we rediscover or discover that there's vastly more important factors, and they don't relate to what we've been saying for decades. You know, so it's a natural thing. It's not weird or unusual, or people, you know, taking envelopes under a desk. It's literally just the whole massive organ of academia and industry, who are involved into disease, they just will not tolerate something that undermines them. I mean, this is so obvious to me.

Malcolm

00:43:29

Well, it's so obvious because it's true. You know, people say, "Oh, you're a conspiracy theorist." I go, "Not a theory. I have facts. I have data." So I'm not a conspiracy scientist, I'm just pointing out to you that there is..." It's not a conspiracy as in like Doctor Know. Somebody sitting with a white cat on their lap going, "The world domination will be mine." No, but everyone kind of acts together. Because, as you say, also, sometimes I like it too, because I live near Manchester, and saying it, "You grew up as a Manchester United supporter, you support Manchester United, you hate Manchester City." It's not rational, it's just the thing that you do. I mean, no Manchester United supporter will ever say, "You know what, I think Manchester City are a better football team. I'm going to start supporting them, because they play better football and I think I'll enjoy it more." At which point it's like, he would just be like, "What? You gotta do that? Manchester City!" to all his mates, and everybody knows, and the social network of supporting [Inaudible 00:44:33] will be gone.

00:44:34

You can't do it. There's no financial thing there. It's just, you grown up with this, you brought up with it. And if you're surrounded by people who all believe in cholesterol hypothesis, and all tell you, it's all the same things and statins all the way, and lowering cholesterol all the way, if you stood up and said, at a meeting, "You know what, I've come to the conclusion that this is all a crock. It's just wrong. And I'm now going to go look

at something else and you're all wrong." That would be the end of their career. It would be end of their personal relationships, their department would never get any more money from the pharmaceutical industry, they would become just obliterated on the face of the earth, effectively. Who's going to do that?

00:45:18 So there are people unfortunately who are going to do that, are people who are outside of that world. And so you can immediately be described as a maverick, as [Inaudible 00:45:27], somebody who doesn't understand science, blah, blah, blah. You know, the usual criticisms? Well, you're not a professor of cardiology, what do you know? Which I'm often tempted to say, "I forgot more about heart disease and you will never know." Right? But I tried to not say that, because then people will just dismiss you an arrogant idiot. But I mean, you have to try and keep your cool. But even though you know that why do you believe this, why do you believe the cholesterol hypothesis? Did you think of it first? Why have you just been told it? And you've never really questioned it. It's just the way it is, isn't it? You grew out with it.

00:46:01 So yeah, you're right is it's not money necessarily although money does very major. It definitely shuts up people like Kilmer McCully who just was basically got rid of. Shuts up by the researchers. I was looking at the researcher, you'll never have heard of her; no one's ever heard of her. I'm writing a book on [Inaudible 00:46:20] called [Inaudible 00:46:23] Smith, who actually taught me cardiology when I was at Aberdeen University. And she said a thing to me in 1982. She said, "LDL cannot cross the endothelium," in a sort of small group tutorial. Nobody knew what she was talking about. I never heard of the endothelium. I vaguely knew LDL was. But there was something about the way she said it. I thought that's like I've just been told something, almost like a secret. Like, like the matrix, blue pill, red pill, you know? Kind of which way around it, is take the blue pill and you will live in this wonderful made up world and never question anything. Take the red pill, and your life will become really hard and difficult, and you'll be attacked by everybody for the rest of your life. So which pill do you want to take? Unfortunately, I took the red pill, because I went off and try to find that one she'd been talking about all those years ago.

00:47:25 What did that mean? And that's how I started questioning. I would never question that. I was a medical student. I was like, "Tell me stuff and I'll believe it." We do what we're told. No, I was never quite like that. But I wouldn't... I had no reason to question it. Even then it was, "You eat saturated fat, it raises your cholesterol, cholesterol goes into your arteries and kills

you.” And had somebody not come along and just put that... it was weird. It was like an electric shock. But it just went in my brain and just wouldn't go away, forever since then. And then I looked at her research and she'd been working on basically the endothelial damage and repair, Apo lipoprotein, a1, etc. all sorts of things that really are critically important. And I think in my opinion, she never quite said it, but she was saying what I'm saying is heart disease is about blood clotting on the artery walls due to damage in the artery walls, and etc. It's very much the same theory. A theory first proposed in 1842 by Carl von Rokitansky called the Encrustation Hypothesis. So this is not used up that we're talking about here. This hypothesis has kind of been wandering around in the background for ages, for as long as people have been looking at heart disease. It was a Scottish doctor in 1946 who basically said, “When I look at plaques, atherosclerosis, this is the remnants of blood clots.”

Ivor 00:49:40 This one, Malcolm, I'm going to just for a moment stop, this one is really central and it speaks to the heart of what you described earlier. Absolutely correctly, it's a process. So investigate, understand the process and the inputs to the process. And then there's beneficial inputs that are the healing process affects the process in the other direction. And so they're important too and you've made that point.

00:50:05 So if we briefly check back before we get into the blood clot creating atheroma or atheromatous plaque, the describing this process briefly go back, we've got the endothelium, the single cell, or arguably a couple of cell layer on the inside of your artery, pivotal, crucial and central to atherosclerosis is the health of that layer. And the cells turnover, bad things can make them die, become depleted, bad things can damage, good things will make them healthy. But if we take that list of factors, again, recap on what damages the endothelium, and then move into the clot hypothesis based on the damaged endothelium.

Malcolm 00:50:52 Yeah, no, no, I think that it's quite right. I mean, I have a tendency to leap around of it, because I get over enthusiastic about things. I mean, yeah, you're right. They're saying the endothelium is a lining of all your blood vessels, whether it's one cell thick, or to 279. I've seen places where it's more than one cell. You've got to keep that healthy. Because that's the protection. If that's healthy and the glycocalyx is healthy, and everything's going fine, we don't get atherosclerosis. This doesn't happen. So what you're saying is, “What can damage it?” Well, the things that damage it, obviously, if you got a really high blood pressure, that's going to create rid of biomechanical stress. If you smoke, that's bad for it. If you've got a high blood

sugar level, that's bad for it. If you've been exposed to pollutants, air pollution, various things that go into your bloodstream, like mercury, whatever poisoning that is bad for it.

00:51:45 So those are some of the bad things if you like, things that damage your endothelium include, and if you look at the latest list of what they call the QRISK factor thing that your GPU in the UK might have stuck with a computer, there are different factors you put in, cause heart disease, and that include things like rheumatoid arthritis, and systemic lupus, Hiroshima to it. It's even got these things in it and no one's ever heard of. It's like, these are things that can also damage because it causes vasculitis. So anything that can cause vascular inflammation, whatever that might be, then you can look at those things. Those are the things that can damage endothelium.

00:52:22 Things that will protect it: Exercise protects it, because exercise stimulates the thing called nitric oxide, which is a molecule which is incredibly important for your health. It sits in the glycocalyx, it's anticoagulant, it causes vasodilation, it increases the production of endothelial progenitor cells in the bone marrow. Your nitric oxide is really super important. So anything that can raise nitric oxide is a good thing. And you say, "What does that?" Exercise does that. Sunlight does that. Sunshine is very good for you. Sunshine is, I mean, everyone goes on with sunshine, skin cancer.

00:53:08 Leaving that to one side, you know, let's have another debate about that. But from a cardiovascular perspective, they did a study in Denmark, and I think also in Sweden, when he showed that, and I think it was just a woman rather than men, but they found that women who had increased solar exposure or whatever the term is, had a reduced risk of overall mortality, massive reduce in cardiovascular mortality. And this has been found in study after study. Sunlight makes you feel good. The reason why it makes you feel good is because it is good for you. And it increases nitric oxide synthesis. It lowers your blood pressure by more than most blood pressure lowering tablets. And it just generally protects you. So that's good for you. Definitely, absolutely. So there are two things that can... and also, if you think about stress, release the stress hormones, the stress hormones are bad for you. So relaxation, mindfulness, meditation, these things have been demonstrated to have benefits in this area. So these are good things to do as well.

00:54:11 Interestingly, one of the things, the medication that most increases your nitric oxide synthesis is Viagra. And that's how it works. And it works. It was actually designed for angina. It was

designed as a treatment for angina, right? And during the middle of the trials, they found that the participants who were actually American prisoners and American servicemen, especially the servicemen were not giving a Viagra back at the end of the trial, and then they've said, "Why not?" And they found eventually, hey admitted what was happening, and then they realized that actually stimulates nitric oxide synthesis in the penis, which creates an erection. And that is what it does. And also they find if you give Viagra, there have been studies showing people with heart disease and diabetes who take Viagra are up to three to 400, or three-fold reduction in the risk of dying of heart disease in the future. So they have shown that this is a thing that can happen.

00:55:20 So I'm not recommending medication for people who don't have a problem if you like. But the whole other issue and one of the major risk factors for heart disease, it's considered a risk factor, is erectile dysfunction. In other words, you can't get an erection. That's a sign usually that you don't have enough nitric oxide, which is a sign of endothelial damage, which is a sign that your vascular system is under threat for whatever reason or other. So I mean, people with diabetes, for instance, or high blood sugar levels are very likely to have erectile dysfunction. It's not a cure, but it's a way of reversing it as nitric oxide.

Ivor 00:55:55 And actually on that one, Dr. Joseph Kraft, I know you're familiar with, he saw PED or Penile Erectile Dysfunction as almost pseudo diagnostic of diabetes on less pulse glucose insulin test ruled it out. So absolute, very intimately related to the vascular destruction.

00:56:15 So we're talking now, you've listed out some great drivers of vascular endothelial distress which is the core of atherosclerosis and some great things that would help avoid it right up to and including Viagra, which is a good one, but not recommending it. And maybe another thing you could throw in, which is debatable, but various vitamins and minerals that assist generally in the health of all your body's tissues like maybe useful magnesium, selenium, whatever, but there's no point going down a hole on that because there's so many.

Malcolm 00:56:53 Yeah, it's like an endless list in a way. I mean, if you're going to put them on top of the list, magnesium, yes, I'm going to leave a room for potassium, which I try not to get into too much detail. But essentially, people have said, "Cut down on sodium, or salt." But actually, what most people need if you increase your potassium level... so anyway, potassium are so closely interlinked in the body. They're kind of synergistic in a way. If

you reduce your sodium too much, what actually happens is the body will fire up various systems to try and retain the sodium, one of which is getting rid of potassium. And the other one of which is actually it stimulates hormones which affect and reduce nitric oxide synthesis. And that creates vasoconstriction, the arteries narrow, and this is quite damaging to the cardiovascular system. And so if you reduce sodium, you will cause damage to the cardiovascular system. You'll create the very problems you're trying to solve. And as you may know, that the latest [Inaudible 00:58:02] Nutritional American, whatever it stands for, Study found that there was a very clear U shaped, in fact, it wasn't really U shaped, it was more the people who really restricted sodium intake were the ones with the highest mortality rate. By quite a considerable margin. And in fact, one of the, I forgot his name, it begins with A, who was the President of the American Society of Hypertension at one time, did a study on people with heart failure and reducing sodium intake. And what he found was that it actually quadrupled the mortality rate. So sodium restriction, just bonkers, because you need a potassium, so you keep those things in balance. That keeps nitric oxide levels in a good place as well and kicks down a whole bunch of hormones that are produced by the kidney that trigger your blood pressure to be hard. One of them is angiotensinogen. You may have heard a lot of people may be on an ACE inhibitor to lower their blood pressure, Angiotensin-Converting Enzyme inhibitor. Now, that's the most widely used antihypertensive drug that we have. And actually, I'm quite in favor of it because I know that it stops the production of this angiotensinogen which damages nitric oxide synthesis and damages the endothelium.

00:59:22

We can show this. So ACE inhibitors. Interestingly, and I know this goes off for another tangent, there is a drug called the Vascular Endothelial Growth Factor inhibitor. VEGF inhibitor. They're used in cancer treatment to stop the cancers from producing their own blood vessels to feed themselves blood cell. This stops what they call angiogenesis (production of new blood cells), this stops the tumor growing and and prevents the cancer from growing. So VEGF inhibitors are used in cancer treatment. The VEGF inhibitors, the problem with them is they increase the risk of heart disease usually. Anything that stops the endothelium from growing and repairing is going to cause heart disease, and it does. So, in fact, the treatment used to stop that being so severe, they're ACE inhibitors. And that focuses on nitric oxide, which actually blocks some of the damaging impact of the VEGF inhibitors.

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](https://ihda.ie) and TheFatEmperor.com

01:00:23 So, as you can see, when he starts looking at it with regard to, "Is nitric oxides positive, glycocalyx is a positive, endothelial progenitor cells are necessary for protecting the health of everything?" So you start looking at things on the basis of, if you damage these or improve these, do you see benefits? Do you see harm? The answer is yes, in every single case. I was pointed at a very commonly prescribed group of drugs. It's called proton pump inhibitors, which most people now called losec, omeprazole, pantoprazole, lansoprazole, things ending in zole is one of these. low And, they have an effect of reducing nitric oxide synthesis. And there's a pathway and you've seen it. And proton pump inhibitors increase the risk of dying of heart disease by about 200%. So you think, "Well, how can that be the case?" But once as soon as you start saying, well, let's focus in on endothelial health, you immediately understand how these things can happen. To me, it's almost like... this is so obvious. It's like, "Hold on, you give me a factor that causes increased risk of heart disease, and I'll tell you does it damage the endothelium or does it interfere with healing? There is a blood clot formation thing in the middle of that that's also quite important, but we'll ignore that for the moment. And if it does one of these two things, it will increase the risk of heart disease. And if it improves one of these two things, it reduces the risk of heart disease. I have not found a contradiction to this.

Ivor

01:02:06 That is crucial that any hypothesis are certainly a description of a very complex process, you need to look for exceptions, to better tune your model, you need to look for contradictions. Now, unfortunately, the orthodox take one champion, which turns out to be a dude, and then they find everything they can to support it, which is just confirmation bias and affirmation bias. But with this theory, which makes absolute mechanistic sense agrees with all of the literature that's available. And there's almost no exceptions, if not no exceptions. Of course, you generally there have arrived at the kernel, or the most important kernel of any big problem, which you have.

01:02:49 So we go now to the clot, and I know you don't want to go into the detail but I I love the description of the process where you do get endothelial damage, you get problems with the glycocalyx, and you begin the process of what we've been talking about based in the factors pro driving, proatherogenic, or anti. But the actual description of the blood clot becoming the atheroma and the proof, if you will, over the decades and way back 100 years ago, I believe. But the proof of that that the atheromas is essentially a blood clot that's been recovered. You know, that process is fascinating to me, that description.

Malcolm

01:03:32 And essentially, of course, one of the objections to this hypothesis, always been, "Oh well, atherosclerosis, it's full of fat and cholesterol, isn't it?" The answer is, "Well, they're there, yes. I agree." There's all sorts of things there as well, because blood clots are... almost everything that's in the blood becomes part of a blood clot. I was trying to explain to my daughter who's doing medicine, I said, "Don't get involved in it. It's too complicated. No one understands it." Except, one of the things that you've got to understand is that, obviously, a blood clot, well, if you strip a bit of endothelium, which is the lining off the artery wall, that all the things that were there before then the stopping of blood clot forming have gone, and actually you're exposing the blood to the artery wall. Now, not unnaturally, the body says, "If blood is being exposed on artery walls, something quite nasty probably just happened, I better form a blood clot. And I better do it bloody quick." So it does.

01:04:34 Essentially, there're two major players in blood clotting. There's platelets, which are little cells, about quarter of the size of a red blood cell. They float around in your bloodstream. And the moment they see an area of damage, these chemical signals, and they just go back. And they form what they call a platelet plug, which develops people throwing themselves at the dike in Holland. [Inaudible 01:04:58] boy with his finger. Well, this is like 500 people jump at the block, and then all will link arms, and then say the blood isn't going to get passed to you. So you get this plaque thing that forms. Then, slightly later on comes what they call the fibrin is also floating around your bloodstream. Little small fibrinogen is what it's called. Protein strands, say that that long, I've no idea proportionately. The moment all this, the platelets start sticking together, they trigger off what they call the clotting cascade, which everyone's heard of, [Inaudible 01:05:31], millions of them. And then the end result is that fibrinogen is linked together end to end to form a sticky form of fishing line, really strong. It goes in and holds the whole thing together really tightly. Along with this come, and this is where this gets complicated, but the surfaces on which fibrin is actually created our lipoproteins, the lipid surfaces, which are things like LDL and VLDL, and that's where things are formed.

01:06:09 So they get drawn into the clot as well, along with red blood cells. I just saw a fantastic micro pictures of a clot which is red blood cells and fibrin. It's just like a fantastic sort of model of things. So anyway, all that gets dragged in, forms a plug, and sits there. And that's the clot. Obviously at the same time, every time a blood clot forms, it can't keep forming, otherwise, it's going to be a big blood clot and you die. So it stops forming very

quickly. So for every factor saying clot, this has been another factor going don't clot, clot, don't clot. The whole system is incredibly complicated. But essentially, you've now got a blood clot sitting on top of the artery wall. And then the other question is, "What are you going to do with it?" As I say to people, if you scratch your skin, they'll bleed for a bit, then a scab will form and then eventually the scab will just fall off. This is what happens. But of course the skin, underneath the surface of the skin, there's layers of cells that can grow up from underneath. So the very top layer of your skin, there's about 12 or 13 underlying layers, so they grew up and push the scab off your skin. Thought of it in your artery, obviously, a clot that's in the lining of your artery wall just got pushed off and then charged off down the artery, it would reduce the artery now and then just cause a blockage. And that's what we call a heart attack or a stroke or something of the sort. So you can't have that happen. So the blood clot can't be dealt with that way.

01:07:39

The other thing is, of course, the endothelial cells are one layer thick, and there's no cells underneath, they don't grow up. That's your layer. Underneath that is other stuff. But it's not further endothelial cells. So there's no way of creating a new layer that grow up from underneath. So then you have this clot stuck to your artery. Well, it not that big. I think it shaved down a bit as much as possible. And then of course, this is where the endothelial progenitor cells come along, is they come along to stick to it and then they grow into full blown endothelial cells and now you have the clot sitting underneath the endothelium. If you go back 150 years ago, this was 160 years ago, this is a point of argument between two guys. One of them says, "When I look at plaques, I'm seeing blood clots in various stages I prepare." Another doctor, co worker said, "Well, you can't get a blood clot forming underneath the (endothelium)." He didn't call endothelium at that time, "It can't form inside the artery wall. So your idea is obviously nonsense." But of course, now there are new about endothelial regenesis cells, they weren't discovered until 1990 something. People thought the endothelial cells regrew from the side, but they don't. That's not what happens. Like many cells in parts of your body, the mature cell can't become another cell. The endothelial cells are produced in your bone marrow.

01:09:05

So as soon you know this, you can understand the process of what's going on. What we have now is a bit of a blood clot stuck inside the artery wall. And then what happens? Well, generally, it's got rid of. Generally the repair processes get rid of it. So white cells and whatever chomp it away, and you will be left with no sign that was ever there. But if it keeps forming again

and again at the same point, which is more likely, I suppose, because this is an area of damage. And you can see that many, I would say all start this way, but most plaques have a kind of tree ring appearance if you look at them. This thing has grown layer upon layer upon layer upon layer upon layer upon layer upon layer. And said, "Well, how can that possibly be the case?" Well, the only way that can be the case, is it layer after layer after layer of blood clot has formed being partially got rid of and then another one's coming stuck itself on top. And then you say, "Well, how come there is LDL in there, how come there are lipoproteins in there? How come there is cholesterol in there?" And then one of the original discoveries by [Inaudible 01:10:16] was he saw cholesterol inside plaques, and said, "Well, there's cholesterol in there. Where does that come from?" And the answer was, "It must have come from LDL." Because that's the particle that carries cholesterol around. Well, the answer is, it can't have come from there. Because the cholesterol has carried an LDL. It's not pure cholesterol. It's actually called cholesterol ester. So it's a cholesterol attached to a fatty acid.

01:10:44 You can't make your cholesterol crystal from that. The only place you can get pure cholesterol from is actually the membrane of a red blood cell. Because actually, think of red blood cells, we call them red blood cells, we call lipoproteins lipoproteins, but a red blood cell, 40% of it is lipid, and a really high percentage of that is cholesterol. So actually the only possible source of the cholesterol crystals you see in atherosclerotic plaques is from red blood cells. And where do you get red blood cells from? Blood clots. So what you're seeing is a remnant of a blood flow. It's not the remnant of lots of little bits of LDL that gradually sort of gone through an end crystallized because they can't crystallize. Because you can't get a crystal crystal from what they call a cholesterol ester. It just chemically doesn't happen.

Ivor 01:11:36 Now, the cholesterol, I've yet to see a response to this discussion which you've brought up a while back from the more pro cholesterol [Inaudible 01:11:46]. I suppose you could argue that the lipoproteins become engaged in some manner, like you said, they do become engaged and maybe they could be [Inaudible 01:11:54] or broken up, or macrophage get involved and eventually, a conversion of cholesterol ester inside the lipoprotein into cholesterol. But we're putting a lot of things together now just to try and refute.

Malcolm 01:12:11 The papers I wrote are very clear that the cholesterol that you find in cholesterol crystals does not come, cannot come from LDL. The only possible source is a red blood cell. Then you're

going to ask yourself, "How does a red blood cell get inside the artery wall?" How can you get a red blood cell inside the artery wall? The only way you can get it is if there was a clot, or a plaque or a clot that formed at some point in the past, wherever that may come from. So I mean, the basic principles on which the LDL hypothesis are put together. Because the other thing and you know, you'll know this, but most people won't, is that they say that they're finding LDL inside plaques. But there was another lipoprotein called lipoprotein (a). And lipoprotein (a) is an LDL, with an additional protein attached. And if you don't look for that additional protein, you would say you're finding LDL. So it's a bit like finding a whole bunch of cars in a scrap yard and saying they're all [Inaudible 01:13:19] cars because they're rubbish. No, they're all [Inaudible 01:13:22] because they rust. And then someone says, "Yes, but actually, what we can find is there's a thing at the front. There's lots of these other things around and got off early on them." So you thought you were finding fears, but actually there are for [Inaudible 01:13:32]. And you wouldn't know the difference. You wouldn't know the difference between the two unless you found the badge. So you're not looking for the badge, you don't know what you're looking at. And if you look for the badge, which is this protein, you find that it's there. In other words, the lipoprotein that you find in the artery wall is not LDL, it's Lp(a).

- Ivor 01:13:56 You know what? I think it was circa, it would have been 2013. I spent a little while on Lp(a) because my Lp(a) is through the roof. It's 118 nanomole. It's huge, like my particle count, but I have zero CAC at 48. And I have my reasons for not worrying about that. But Lp(a), I found a paper and it was old enough to be photocopied rather than a proper electronic copy. I purchased it, and it showed exactly that. It showed that the LDL type components in the plaque are overwhelmingly of the Lp(a) type. How that fact is not openly discussed by lipidologists and poured over is extraordinary to me.
- Malcolm 01:14:44 Well, it's not extraordinary, because it's the same thing. Once you go down that route, you're left thinking, "So, we're wrong." We thought we were finding... without the cholesterol crystals coming from LDL, no, they don't. We thought that a lipoprotein we're finding is LDL. No, it isn't. Well, there's the hypothesis gone, isn't it?
- 01:15:05 I mean, yes. As you know, Lp(a), lots of people say we don't know why it's there. Well, you do because you've been told, right? You know why it's there. It's there, because. If you have a lack of vitamin C, which is called scurvy or called scorbutic. If you have a low level of vitamin C, or you have damaged the

artery walls due to a low level of vitamin C, Lp(a) is attracted to areas of damaging craps, sticks to it very tightly, and cannot be easily removed. And that stops you bleeding to death, because that's why you die if you've got scurvy. So people gums used to bleed and then it would bleed internally, and then they would die.

01:15:53 Vitamin C is required to make collagen, collagen is quite lightly reinforcing roads in concrete. Get rid of the collagen and your arteries start to splinter. So when that starts to happen, well, nature came along because we can't synthesize... humans can't synthesize vitamin C. We lost that ability, apparently, 40 million years ago. I never quite know how they can work out this up up 40. But what you were around at the time, did you see it happen, I presume is somewhere you can do this and track it back. But anyway, for some reason, human beings, great apes, couple of other animals I can't remember, and guinea pigs can't synthesize vitamin C. So we need it in the diet. If we don't get it, we die. Which seems a bad design flaw to me. There we go. You know, why would this happen? Anyway, there must be some...

01:16:46 Anyway, that's evolution for you. So we can't produce it. So therefore, the theory was by some people that we've got, most people have got ineffective, a lot of us are slightly vitamin C deficient at all times so our arteries are slightly cracking open. And then the Lp(a) comes along and sticks to it, and cannot be removed. This was Linus Paulings's idea, initially. And it wasn't Linus Pauling's ideas; Matthias Rath's idea, and Linus Pauling thought it was a really good idea. So he said, "If you want to prevent heart disease, eat lots of vitamin C, you won't get cracks in your arteries, and you will live forever." I don't know if you eat a lot of vitamin C or not. But of course, if you don't get cracks in your arteries, it doesn't matter. Because the Lp(a) does nothing, it just floats around.

01:17:31 This is found, the [Inaudible 01:17:36] don't synthesize vitamin C. That's it. And the Lp(a) is designed to cause any damage to the arteries to be covered over and not leak. And then it gets incorporated into the artery as it must do because it gets re-endothelialized. And the reason why Lp(a) is so effective at doing this is because that protein that's attached to it called Apolipoprotein(a) is almost exactly the same structure as another protein called plasminogen. Plasminogen is incorporated into all clots as they form. And when plasminogen is changing to plasmin, it then slices fibrin apart and chops the clots apart so it shaves it down. But if you've got lots of Apolipoprotein(a), this blocks the movement from plasminogen to plasmin, and therefore the clot can't be got rid of.

- 01:18:35 So if you've got quite a lot of Lp(a) in your system, and a lot of that is arriving in your arteries and covering over clots, then basically you have an immovable or less easily removable clot that is stuck the artery wall. Now, people have looked and said, you know, I've seen people say, "We don't really know Lp(a) does. But why does it look so much like plasminogen? In fact, why is it only changed by three amino acids in a tree ring sequence?" "Well, that's interesting. What's that got to do with heart disease?" Well, everything actually, because this is what happens. The Lp(a) is attracted to areas of arterial damage. It forms a clot that is much more difficult to remove. It becomes incorporated into the artery as part of the clot. It looks like LDL and all you idiot say is, "Look, we've got LDL inside the plaque." We go, "No, you don't. You've got Lp(a) inside, which is a completely different thing, or some completely different because it's exactly the same apart from this protein." And then they go, "Oh, look, LDL must cause heart disease." And you think, "Well, that's one way of looking at it." But it's almost like a weird... I remember giving a couple of talks and saying, don't know if you've ever seen the film 12 Angry Men.
- Ivor 01:19:50 Yes.
- Malcolm 01:19:52 Very good film.
- Ivor 01:19:53 Yeah.
- Malcolm 01:19:54 It started out as a play. Well, they've got this Latino lad who's been accused of stabbing his father to death. We've got this knife, and this knife was very unusual, no one's ever seen a knife like this before. And somebody said, "Oh, I heard him shout, "I'm going to kill you." Somebody saw him enter the apartment. You know, the evidence was overwhelming. Essentially, it's a film about prejudice and things like that. But it's this piece of evidence because the first thing is that Henry Fonda goes down to the local store, finds a knife exactly the same, sticks in the table and says, "Well, I found one in two minutes." And then he says, when the guy heard him say, "I'm going to kill him," there was a train going past at the time. One of the old trains, it makes so much noise, no one can hear anything. The woman that's torn the apartment [Inaudible 01:20:41] and couldn't see that she was wearing them.
- 01:20:43 Essentially all the evidence, it appears absolutely incontrovertible. This person is guilty. We've got all the evidence we need. Look at it. No one could possibly say this person isn't guilty. When you start looking at each piece of

evidence, it falls apart. And to me this is the current hypothesis, we've got the LDL hypothesis, it falls apart, you know? You eat saturated fat, it raises your LDL level. No, it doesn't. Well if it does, who cares, because it doesn't matter? You know, "Oh look, we find LDL inside atherosclerotic plaques." "No, you didn't. You found Lp(a)." "Oh, we've got cholesterol crystals. That must have come from LDL, because LDL carries the most cholesterol around." "Well, the cholesterol crystals mostly come from red blood cells because it can't come from LDL." So now, this piece of the evidence, there's more than that, of course, but it talks a bit. And when you look at it, it doesn't work and therefore, you said, "Well, what does work?" "What actually fits?" "Where does it fit?" You know? Okay, if you're going to find lipoproteins inside plaque, where did that come from? Well, it came from Lp(a).

01:21:47 Unifying crystals, plasmic crystals and hydroplaque, where did that come from? It came from red blood cell. I'm going to say to them, "If you find fibrin inside the plaque, where the hell did that come from?" The only place that's going to come from is a blood clot. So you're finding fibrin inside artery walls. Even artery walls with there's no obvious, where did that come from? The only place that came from was a blood clot, because that's the only place that ever happens like that. So it just seems to me, I mean, you know yourself that the arguments in favor of the cluster hypothesis are just repeated ad nauseum. And you may know that we got a paper, the [Inaudible 01:22:22] group got a paper published recently, which says that, "In people with familial hypercholesterolemia, which is usually the strongest argument in support of cholesterol hypothesis that the people with familial hypercholesterolemia who are genetically very high raised cholesterol levels are far more likely to die from cardiovascular disease."

01:22:45 Well, there's two answers to that is one, that's true when they're under the age of about 60. After that, their risk of heart disease becomes the same as everybody else, and in fact, slightly less than everybody else. So an age when you're most likely to get heart disease, people with high cholesterol are less likely to get heart disease. Okay, fingers. But the other important thing is that actually the gene loci for (FH) familial hypercholesterolemia is also very closely associated with blood clotting factor abnormalities. And what you can see is if you look at it, and you can say that, if you get the offspring of parents that got familial hypercholesterolemia, one of them has got the high cholesterol, the other one doesn't, the risk of heart disease is the same. It's got nothing to do with the cholesterol level.

- 01:23:35 And then you start looking at blood clotting factors. It's actually a blood clotting factor problem, because without going into all of the stuff, the LDL receptor itself, which is the thing that takes LDL out of circulation, and if you got less of them, that's called familial hypercholesterolemia in its simplest form. The LDL receptor itself removes blood clotting factors from the bloodstream as well, including factor VIII. So you can even easily explain from a hypercholesterolemia on the basis of blood clotting because it doesn't actually fit with the cholesterol hypothesis. Because people with hypercholesterolemia, the risk of dying of heart disease, there's no relationship to the absolute risk level of the LDL, when you've got familial hypercholesterolemia. So if you're level seven or 13, it doesn't matter. It's the same if you're talking about a higher level causes risk, well, it doesn't. You just have to have the [Inaudible 01:24:32] and your risk is exactly the same.
-
- 01:24:36 I mean, to me, the argument is a bit like 12 Angry Men. On one side we've got what is absolutely certain. This is the argument, look at all the evidence. The other side is you got people saying, "Well, this piece of that evidence, if you actually decide to analyze it, what I call correctly, disintegrates." And that's where we are.
- Ivor 01:24:54 And in 12 Angry Men, it was prejudiced on behalf of the jurists, or the 11 jurists anyway against the Latino kid, which is a certain amount of driver. But in this case with cholesterol we have as described earlier, pretty much everyone in authority, academia and business are all massively kind of incentivized to keep the hypothesis. So it's going to be even worse than the single trial.
- Malcolm 01:25:21 Yes. Well, in fact, the 12 Angry Men, it was actually a play about McCarthyism.
- Ivor 01:25:28 Ah, actually. I love the film or the movie with Peter Fonda. And people should really check that out. It's probably a few dollars on YouTube. It's an amazing movie to a play originally.
- Malcolm 01:25:39 No, no. Well, I think, absolutely. This is the thing of how already having made your mind up, which is incredibly important. We've already got a victim. I've got a few books, mistakes were made but not by me. And then it was called Why We Lie? which actually fit quite closely together. There are ways that we fool ourselves? We create stories and we like these stories and then it's incredibly difficult to get that changed. A story that we've come to know and love is like an emotional thing as well. It's powerful from that perspective. I sometimes use the example of

the film, Inception. I don't know if you've seen it, "The most powerful parasite is an idea." And I think that's it. There's also ideas, we love ideas, and this is an idea the cholesterol hypothesis is like a, it's almost... it's a story, isn't it? It's the body, it's this fat, this cholesterol, this heart disease and statin, whatever. And no one wants to change that, say, "Well, what's going on with fat in the diet? [Inaudible 01:26:45] cholesterol." Statins, well, we know that statins have some benefits. Where do those benefits come from? Well, I know that one of the things that statins do is they increase nitric oxide synthesis in endothelial cells in the glycocalyx.

01:27:00 This is a [Inaudible 01:27:02]. It weren't designed to do this in any way, shape, or form. It's just that we know that they do. And they do it in exactly the same way that a sonographers do and they have about the same benefit as a sonographers on cardiovascular disease. This is not a coincidence. So even when it comes to statins and how do they work, say, well, I can't explain it how they lower LDL, but I can explain it from a nitric oxide, endothelial protection, blood clotting perspective, because it fits exactly. I don't need I'd be scrambling around and pretending. No, I didn't do this research. You know, this is well established. If somebody's watching this wants to type in statins and increase nitric oxide synthesis into Google, be prepared for many, many papers from all over the place.

Ivor 01:27:52 And indeed, other mechanisms of statins that would be mechanistically beneficial that don't involve LDL. And I think once... I had a [Inaudible 01:28:00] a couple of years ago, there's a few papers out that show in human RCTs with statin, they tracked all the data, the lowered events in the people taking the statin, and they saw there was no correlation with the LDL value or lowering achieved. Now I know in England, you've got Rory Collins claiming there's a magical straight line relationship. But independent studies in the US have showed no real relationship with the degree of LDL lowering. And in fact, one paper that saw that also noted that there was a relationship with the lowering of ferritin and CRP and inflammatory markers caused by the statin, did track with the lowered events, but not the LDL.

Malcolm 01:28:45 No. Well, I mean, it is remarkable though people can look at papers and just decide it's confirmed everything they ever thought. It's like in the UK at the moment on the Brexit and [Inaudible 01:28:56], I mean just one side, whatever put you back into the middle, it's like, well, that just proves that we should leave. Absolutely proves you should remain. Same vibe, completely different interpretations. You're never going to get

any changes like you don't know these, just stop saying stupid things all the time. It's driving me insane. I just stop looking at the debate, my brain is going to blow up with frustration. But you can see this and people can take this in. What they've done is they've taken studies and looked at giving a statin and the starting level of LDL and the finishing level of LDL in the various studies, and they're all kind of been all over the place. And then they've just basically scrunch them together and draw a line. I mean, which School of Science did you go to? You do think, how on earth do they keep getting away with this nonsense? It's just ridiculous.

01:30:00

Because I've seen studies, it was a study in 1992. It was a report of a consensus meeting on cholesterol and whether it might increase the risk of cancer or the non cardiovascular diseases. So having a lower cholesterol level might be harmful or might otherwise. And when they looked at studies, every study and everything they could find about cholesterol done up to that point. And all they could find, in women there was absolutely no correlation whatsoever, except maybe a slight reduction in mortality as your cholesterol went up. With men, there was an increase of like 10% or 15% in cardiovascular events with a high cholesterol level. So this would be the actual data from people and yet Rory Collins comes along and draws you a graphic. It was a bit like this. All the data says that, if there is any association is very weak. And yet, you're showing a graph that shows the relationship is very strong, based on studies that were never designed to look at the degree of cholesterol lowering anyway. And then of course, the papers I produced in like 60 pages along with statistics that you have to go look up on a computer because no one can understand them. And it is really, I think there was Montaigne, he's one of my favorite philosophers who said, to paraphrase, "Bullshit baffles brains. You can't read these papers, it's impossible, you just fall into a coma after about page one. Whilst they put together, stitched together this enormously comprehensible series of statistical correlations that just make no sense at all.

Ivor

01:31:47

Yeah, if you have to do enormous statistical treatments in modeling and adjustment, in order to get a result, you have to ask yourself, how big the result really was in the first place and what extent it's real after all your adjustment, true. But I think the best thing to say about statins is yes, for demonstrated heart disease, like a high calcification score or a prior even, and if you if you're not in the position to fix the causes to your own satisfaction, and that takes a lot of scale, then a statin will reduce future events, etc, etc. So it's an intervention. But I think the big problem is statin is going to people with zero CAC scores

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](https://ihda.ie) and TheFatEmperor.com

because they never get the scan, people who have apparent risk factors, but they actually don't have any disease. So it's like a shotgun medication approach. And that has to be really fixed first.

Malcolm

01:32:38

Oh no, in the UK, they've just decided that high intensity statins can be prescribed or unprescribed. You can buy them over the counter. You just walk into campus and buy them. I mean, what are we doing here? I mean, yes, well, to say that there's an intervention that does score some benefit, the benefit is vanishingly small. As you know, there's a study in BMJ that showed if you taken a high statin for secondary prevention, which would be already established heart disease, particular for five years, the average increase in life expectancy is 4.1 days. 4.1 days. That is .75 on the day per year. Now, whether that benefit really exists or not, is in my opinion, still up for debate. Those are the fingers. Because that were presented with ridiculous manipulation of statistics, to an extent. It's virtually impossible to work out what anyone's saying to you. But the real benefit is pretty small. It exists, but it's very small. You've got to ask yourself is, can I be bothered taking a drug for the rest of my life that's it's going to give me .75 days a year, extra a year, and might cause all it has to do, because that's one, about 450th years of the year benefit. All it has to do is reduce your quality of life by more than one 450th. And it's not doing any good at all, is it?

Ivor

01:34:05

And I think that was mortality as opposed to reducing events. So the reduction of events, much more substantial. But the mortality true, there's no massive data. Now I do know that paper was hotly contested. But that's another whole argument, you know, as to the statistical methods, etc. Yeah, it's a complicated sphere and I think Malcolm does a massive problem with understanding that cholesterol is not nearly as causal as we are led to think in the process. And there's another separate problem as to statin and its action, whether it's cholesterol or not, and all the data there. So it's really hard for the person in the street to grasp anything around this.

Malcolm

01:34:48

Well, if you're fed a story by all the mainstream, if you like, and all the experts and all the opinion leaders and all that, you're naturally going to think, "Well, they can't all be wrong, can't they? They can't all be wrong. They must be right." I mean, they just must be right. The truth of it is, it's a gigantic conspiracy to kill us all. It's like, it is how we proceed if you go. Well, I wouldn't call it a conspiracy, but I would say it's a way of thinking that's just has come to be the way that people think. I don't know how you get around that. How do you chisel into

that? I suppose you just keep telling people the facts and you keep presenting the facts. And in the end, I mean, whether it happens, I know that sometimes use the example of building a cathedral is you know, people used to build cathedrals knowing that they'd never see it finished, because it was going to take 300 years. So what you're doing, building a cathedral?

01:35:54 I kind of see it as I'm building a cathedral, it may happen in my lifetime, but may not have in my lifetime. But as some of us aren't trying to build this, someone's aren't building the foundations now putting it together, they'll never get built. And I do fear, because I do believe that in the longer term, statins have some really significant unpleasant effects that are irreversible. And they're being swept under the carpet. And in fact, I've decided a [Inaudible 01:36:21] in the UK along with people like Fiona Godlee, she's the editor of the BMJ, asking for an independent review of the adverse effects, data, and overall benefit of statin in primary prevention. And this has gone to the head of Elephant Technology in the government. But obviously, that's probably going to not do much in the middle of all the chaos.

01:36:45 But there are people out there and we are out there. And we are saying we need to review. Because I mean, I say things to people that they don't believe, but it's true. And this is a statement that all of the safety data or all the data on statins has been collated by one organization in Oxford in the UK. And they won't let anyone else see it. So no one else can see their data. And they're going, "Well, you have to believe us because we're good guys." Whatever they are. And you think, "How could it possibly be that the world's most widely prescribed medication, that some people have significant concerns about longer term damaging promises, adverse effects, the data is held by one organization that won't let anyone else see it or review it at all." So it's difficult to be convinced about the safety of these drugs when no one's letting you see the data. I mean, that's quite astonishing. And just, I think just unbelievable that this is the case, but it is the case. And if anyone wants to say that isn't the case, well, I'll do another blog, and I'll put down all the information. Actually, on their own website say that they've got the data anymore and anyone will see it. They don't put it in those terms. You know, "We have signed agreements that we have the data and were the only people that had seen data." So it's not like a good point, but it's quite a good point. You say, "Isn't that conspiracy?" It doesn't have to be conspiracy, these things are happening anyway.

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](https://ihda.ie) and TheFatEmperor.com

- Ivor 01:38:16 And you know, it's a reality, people need to be less naive and more corporate aware in all those things, you know? If you don't have corporate experience or experience in PR, and spin doctoring, or media, the average person really, yeah, has no idea. You just mentioned there something which drives me crazy, actually, is, "They can't all be wrong." And of course, I've been listening to this for seven years since I started to research in this area. And I said, "Well, they are." Not if they're not wrong in everything. They're wrong in some huge fundamentals, which they think they're right on. But they actually like you said they don't have the data. They didn't do the research themselves. They just hear it. So everyone thinks everyone else must be right on the main theory, because we can't all be wrong, but it's self driving.
- 01:39:04 An example of it is I know a person quite close to me, actually, who will never listen to me, very strong minded, arguer, wrangler. And I've heard that for years, "They can't be wrong." I know you're highly technical, but they can't all be wrong. Well, that person recently has been diagnosed with chronic kidney disease. TOFI person. Yeah, stage three. So I look at the panel and I go, yeah, and the doc didn't even tell them. They have to email and said, "What's this CKD thing, stage three?" And then I said, "you look back," I said, "because I'd reckon you're probably diabetic for a decade or more, right?" And he looked back at his results. And he had stage two CKD, two and a half years ago. It was never even raised to him. So basically, he began to say, "Okay, well, how do I fix this?" So I began to explain to him and finally, he realized they're all wrong.
- Malcolm 01:39:58 Yes.
- Ivor 01:39:59 Because he has kidney disease now. What's it mean? I said, "Well, your organs will slowly begin to fail in the coming decade. You got a massive risk for cancer of organs, heart disease, heart attack, stroke, pretty much everything." What I've been saying for seven years is entirely correct, and it was all along. So I kind of rubbed it in a bit too. But yeah, finally finally realizes it.
- Malcolm 01:40:22 Never say I told you so. Yeah.
- Ivor 01:40:26 You kind of have to though, because it's an important lesson for people to learn also.
- Malcolm 01:40:30 Yeah. I mean, they can't all be wrong. Well, history says that very often, they're all wrong. Being wrong is the natural state of experts. Frankly, and yes. The difficulty is in a way is giving them

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ ihda.ie](http://ihda.ie) and TheFatEmperor.com

a stepping stone away from it, doesn't it? Because to say, you know, I've tried to think to myself, how do I get to say, "Well, you weren't wrong, really, you are just a little bit wrong." So if you accept the little bit wrong, then maybe it can bring you to saying, "Well, actually, I'm more comfortable, sort of moving this way." You have to give people an out, I think, and if you if you're too much, like, you can't admit you're wrong without saying and you were right. And I was completely wrong. And I base myself before you know that there is the great, Malcolm Kendrick was correct all along.

01:41:25 They're never going to do that. Ever. And I don't really care if they do that. It doesn't matter to me. I don't need them to, you know, do the walk of shame from like Game of Thrones. Naked out in the streets, where we throw shit at you, you know? We don't need that to happen.

01:41:43 I'd quite like to see it happen. What we need is somehow rather to find a trigger point that allows them to move away and come to the right place. Because the people in charge will always be the people in charge. They are the people that are always in charge in the matter. That's just the way it works. And what what you're going to try and do is we can still remain in charge. but you just got to say different things. Like the correct things for a while, you know? And that's the difficulty.

Ivor 01:42:22 Well, yeah, I know, we've all got hard stops coming up. So that's kind of it. Yeah. Hopefully they will get some kind of a note. I think that's the Chinese kind of thing. Is it cultural or Japanese where, "You don't humiliate someone, you give them away to get out gracefully." And we probably should do that. And I think for low carb, it's kind of morphing at the moment and there's the slowest acceptance. I think the cholesterol one is far too froth, they cannot let that one go because there's too much built on us. But, you know, it's good to be involved in building this cathedral, even if it's not finished too long after we're dead. What do you think, Malcolm? Is that the final kind of point of it?

Malcolm 01:43:04 We're building a cathedral of wisdom.

Ivor 01:43:07 I like it.

Malcolm 01:43:08 You like it? Yeah, I just came up with that.

Ivor 01:43:11 It will be a towering structure of magnificence. Yeah, I think so. Well, I think that's a good point to end. And there's more people joining the army, though, to build this cathedral all the time, which is good. I think it's rapidly expanding. You were a pioneer,

Dr. Malcolm Kendrick and the True Causes of Heart Disease

Find out more [@ihda](https://twitter.com/ihda) and TheFatEmperor.com

and many other names. I won't go through them all way back. But certainly the armies are growing and very heartening as more and more doctors and specialists, consultants, professors, cardiologists are actually growing and you can kind of see that in Twitter. A lot more people beginning to realize we kind of duped ourselves.

Malcolm 01:43:49 Yeah, well, I hope so. Anyway, as a bit of final thing, if you'd said in early 1989 at the Berlin [Inaudible 01:43:59] fall this year, no one would have believed you. And when things change, they do tend to change all of a sudden.

Ivor 01:44:06 Well, hopefully so. Great stat, Malcolm and anyone watching now, just stay to the end screen and make sure you hit the subscribe button. I get the impression lately that Google and Facebook and other kinds of media organizations are not exactly promoting on orthodox views. So you know, we need people to help get the message out there.

Malcolm 01:44:26 Okay.

[End of transcript]