Academy of PHYSICAL MEDICINE

Transcript

Cardiovascular Disease: Recognition, Communication and Usual 'Care' Ref 239

Steven Bruce
Good evening, great to have you with us this evening for what I think is going to be a really interesting 90 minutes of CPD. We often get orthopaedic consultancy and rehab specialists. We have practical demonstrations and that sort of thing, which of course, is very much akin to what we do in our own clinics. But this evening, I'm going to have a fireside chat as it were with somebody who I admire greatly and have done ever since I first came across his blog posts. And I think they might have been the first of the books that he wrote, which is the great cholesterol com, amongst others. And we'll talk about the books a little bit later on. This is Dr. Malcolm Kendrick, he has been on the show a couple of times before. He's made his name by criticising the pharmacological industry's drive to get everybody on statins, the general evidence surrounding cardiovascular glare, and you might ask, what is what's cardiovascular care got to do with us in our practices? I guess, you know, we're going to see people who are worried about their cardiovascular health, we'll see people on various forms of medication. And what Malcolm has to say this evening is going to be extremely useful, possibly very thought provoking. Because as I say, he spent a lot of time pursuing the evidence. He knows the evidence really well, really well. We're not talking here about someone who is an anti Vaxxer, or something of that sort. We're talking about somebody who is putting himself up against the bulk of medical opinion, it seems on the basis of good quality evidence. So Malcolm, welcome. It's great to have you with us in the studio. Again, I'm really looking forward to this evening, and I know we'll cover stuff that we have done before. But I've just said I mean, you're not the equivalent of an anti Vaxxer. On the cardiovascular front, you have got a court case brewing at the moment, I think how many

Malcolm Kendrick
Yes. Now fought well brewing four years ago, The Mail on Sunday, wrote an article saying that myself and a couple of others were causing potentially hundreds of

1000s of people to die, because we don't believe that statins are as effective as the generally made out. And also that they have more adverse effects than

Steven Bruce
say, why would the Mail on Sunday do this? Because journalists aren't generally particularly experts in medicine. They're good at reading abstracts and sticking those into their headlines. But what provoked them to do this? Was there someone else behind it?

Malcolm Kendrick
Well, Far be it for me to know any any lawyers listening? But no, I think they were doing a thing called fake science or a fake news series. And I think they've done something. A few other things that and this was one of the things on their radar, but it's such a big subject in such a big area and there's A lot of money and readers and people on statins, I think they just thought this is an approach. I mean, ironically, before this, they actually published sort of articles on things that I'd written, they covered the great cholesterol con, they covered other books. And in fact, they were thinking of covering my latest book, and then decided now that we're going to take the reverse, which was, this isn't a good idea. This isn't good stuff. This is a maverick fool who should be crushed. And, you know, that's how newspapers work. They don't take positions other than, I suppose what they think is going to sell papers. And, you know, ironically, of course,

Steven Bruce
which means they have to appeal to the widest popular beliefs rather than drive those beliefs.

Malcolm Kendrick
Well, they don't drive the beliefs. Interestingly, after the articles in the comment section, I think 95% were anti the article and enter the industry and pro what we had to say. So it possibly backfired.

Steven Bruce
Well, I was gonna suggest save. Possibly this could backfire in a big way on them, couldn't it? Because assuming that you you win, in your case against the Daily Mail, yes. Mail on Sunday, that will itself be big headlines.

Malcolm Kendrick
Well, probably won't be great big headlines. I'll do what I can to get people that I know and other and other journalists, but it's quite difficult because the Mail on Sunday associated newspapers are really the biggest newspaper group in the UK. If you're a journalist, it doesn't behove your career to start going against what the what the Sent Mail on Sunday has to say. So I'm not holding out.

Steven Bruce

Huge hope. Okay. I mentioned your books. We've mentioned your books, and I talked about the great classes. This. Is it fair to say there's a progression here, the great cholesterol com is about what was wrong with the cholesterol? What is wrong with the cholesterol hypothesis? Yeah, a statin nation is about what's driving us to be on statins. I think the guidelines are pretty much know that if you're over about 50, you're going to be found to be the need of statin Yeah. Well, if

Malcolm Kendrick
you breathe, yeah. And

Steven Bruce
then the latest the clock thickens is, instead of the cholesterol hypothesis, there's an alternative, which has got some fairly decent evidence behind it.

Malcolm Kendrick
Yeah, I think it is. Well, it's a progression in that. Yes. You know, a lot of people have said, well, if it isn't cholesterol, and if it isn't statins, and then what is it? You know? And I couldn't really answer that for a long time, because I didn't really know for sure. Because there's a there's

Steven Bruce
a tendency isn't there? If someone says to you, okay, if it's not this Yeah, what is it? And you say, I don't know, they say, Well, you must be wrong.

Malcolm Kendrick
There. Well, yeah, it doesn't get their thinking anywhere to go. It's like, yes, let's blow this up. And then where do you go? Well, there's nowhere to go. But I think the fascinating thing for me is that once I started looking into an alternative ideas, which which I've known about for some time, is how long they've been around. And how many people have proposed that the idea that is the central idea, this book, and, and yes, never taken off. I mean, it was first proposed 170 years ago, in Vienna, you know, all those years ago, and you think, you know, and it didn't work. And it's been tried again, and again. And it's never grabbed the attention. I think more recently, because there's so much incentive and financial pressure to lower cholesterol and statins and a new cholesterol lowering agents that it's very difficult to go against it. But there was a long time when these things didn't exist. And yet still, the idea, the central idea, which you can go back into the history, it's like, there's a concept of the ghost in the machine. If you look back, you can see there was something in there. And it's been there all the time. And yet somehow, it's just been kept in the background. So it's fascinating to read about these people have been pushing the central idea here for 170 years, and yet it's never stuck. It's just never happened. It's really fascinating why that happens.

Steven Bruce
And I've sometimes I've struggled with with what you see in your books, because of course, I read those books, and I forgot to mention doctoring data, which is another

cracking read, because it's all about how the data has been manipulated to show whatever the pharmaceutical company is wanting to show. Yeah. And it's not the only book to show that's either. No. But I struggle because I read your book, and I think, right, this works. And then the next next week in one of the newspapers, or in one of the medical reports, there'll be this new study that shows that actually no, statins are good. And I think well is Malcolm wrong after all. Yeah, well, well. In the past, it's a Malcolm, what about this? Yeah, well, I

Malcolm Kendrick
never think that now, of course, it's very difficult to go against the tribe and it gets, I mean, an awful lot of this stuff that comes out. The problem is it sounds so plausible, but actually the reality of statins is that there's one organisation in in the world that holds all the data on status that people We say this to people. It's like a hack conspiracy theorist, go and check it out. And they're in Oxford in the UK is the Centre for evidence based medicines and known and different that they that's also there. Ironically, they've called the clinical trial service unit. And they run clinical trials. But actually, within that there's a thing called the cholesterol treatment trials collaboration. And they went to all the pharmaceutical companies and said, We will hold all the data on statins from all these competing companies. And we will have it and we will look at it. And we will produce these reports. And no one else is allowed to look at it, no one else can see the data on status as one group in the world hold all the data. When the Cochrane Collaboration sent someone to look at the data, they wouldn't let them look at it. And they hold all the data, they won't let anyone else see it. They do these reports. And upon these reports, rest everything. Well, no one else can do these reports, because no one else has got the data. And when they've been asked and pressurise, they say well, it was commercially sensitive information, and you can't see it. And we signed agreements to say you can't see it. So this is to me, the antithesis of science, where science is supposed to be debate. It's supposed to be everyone gets to see the data. Everyone can review the data. Well, here, you can't, they've got it. They won't let anyone else see it. One group of people Oxford University in the UK, that's it, no one else. But

Steven Bruce
that said, these are presumably at Oxford University, there are a bunch of well respected, impartial academics who's been whose opinion we should trust? Well, I agree

Malcolm Kendrick
with certain of those statements. weren't sure which one? Well, of course, they're hugely respected. And they go when they speak, and they publish papers, and they, then they do all this stuff. And when you look at, I'll just give you one example of how ridiculous some of the that is. They looked at the use of statins in diabetes. And it is now widespread, if you've got diabetes, you must take a statin based on a study that they did. Now, in their study, there's not there's been two studies directly done on on statins and diabetes. That was the only thing it studied. Most of them have looked at other people and then gathered together the diabetic patients out of those studies, which you shouldn't really do, but they did it. And and then, when they did their

analysis, they did not include the only two studies done on the use of statins and diabetes, they said, We have excluded these studies, because they wouldn't have made any difference to our results. Which is fascinating, because the only two studies that have been done specifically to look at statins and diabetes, found they had no beneficial effect. And yet the paper came out, which is basically statins are wonderful in diabetes. Everyone with diabetes should be on statins. And you'll look at that and think, how on earth can this be allowed to happen?

Steven Bruce
But hang on, you must be paraphrasing here because you can't say these studies would have made no difference if you haven't included them in the study surely?

Malcolm Kendrick
Well. Because you don't know that's not my paraphrasing. That's what they said, really, in the paper itself. What you have to do in this world is you have abstracts, which are short things like what does this paper show, you have some discussion. But then you have methodology, which no one reads because it's really, really boring. And you have this this testicle analysis which no one reads because it's incredibly boring, usually incomprehensible. And then you have other stuff, it's all in there. But if you don't read the paper, I'll tell you a story about a paper which is it's a years ago, it was written. And in the methodology section, the author had written. If anyone reads this sentence in front of this telephone number, I will send them a case of champagne. Okay. No one ever found. And remember, this got through editorial review. This was published, no one even saw it. From the editorial perspective. No one ever phoned him. He said no one ever for me. The reality is people don't read these papers properly. I do and it's down there. It's in black and white. We did not account for the exact words, we did not include these two studies, as it would have made no difference to our results. The only two studies that have ever been done on use of statins and diabetes. And these two studies were both completely negative. Now, you may find that you think this is impossible to believe surely there are people out there who must have noticed this and gone. You can't do that. But you come to do more and more of this stuff and you read it. I did a pro bono research into MECFS at one time 650 pages of a nice report right? And I looked through the whole damn thing there was there were tables where people had written this, this figure should be in table two, whatever. Anyway, the editorial notes in it, this is a nice report from whichever one bases how they treat, and no one had even read this, I think I'm the only person apart from the person who wrote the thing in the first place. I'm the only person who's ever read that report ever, ever.

Steven Bruce
It makes you think that perhaps for nice, the aim of the exercise is to have a report not to have a meaningful report that anyone does anything with?

Malcolm Kendrick
Well, there's I know that in the legal terminology is like if you bombard people with so much stuff that they can't possibly read it, they just give up on what's this, oh, my

God, you know, and a nice do these enormous, I mean, in this report on me and CFS, which we now go, anyway, whatever you call it, the the decided to look at whether graded exercise therapy and cognitive behavioural treatment was, was beneficial, and they decided it was alright. And, and it was like 600 papers and all these just went on and on and on. But in the end, all the other papers were completely irrelevant, because they hadn't looked at this really, they'd looked at other endpoints and other things. There was one study in this entire paper of 600. That was to do with whether graded exercise therapy, and CBT, had a benefit on quality of life, which is the thing you were looking at study itself was done in Belgium is on 200 people. And at the start, the two groups were mismatched. Differently much. So one group had a higher quality of life than the other. In fact, the group that had the higher quality of life of the people who didn't get the treatment that people the lower quality of life, for the people that did get the treatment, at the end of the study, the quality of life of both groups was the same. Now, any statistician will tell you, that's just regression to the mean. That means nothing happened in this study, the author's admitted, nothing happened in the study. And the entire nice guidance was based on that one paper that was wrong. And when that went to the court, and I said, it's all based on one paper and paper was wrong. And by the way, they got the nominator and the denominator upside down when they did the calculations. The judge said, I can't decide on this. This is scientific stuff. All right. I'm just here to decide whether the process of doing the report was fine. It did involve the stakeholders in blah, blah, blah, which they did, of course, and and then they said the only people who can decide on whether this report is valid or nice. I won't. But they're the ones that did the report. That was rubbish in the first place. It was just like, honestly, the grown ups aren't. There are grown ups out there. But the grown ups aren't out there. People just let this rubbish get through. And if you go, this is just rubbish. How can you allow a study on statins in diabetes to not include the only two papers studies ever done on statins and diabetes? Well, no one, you know, surely the editorial people should be going. Hold on. This is this concept of peer review at a conference in the States. We were discussing peer review. I hate peer review. I think it's the worst and stupidest thing. It's not the worst, stupidest thing, but it is a completely ridiculous concept

Steven Bruce
in practice or in theory, or both. In theory, it's

Malcolm Kendrick
good idea. Oh, yeah, get people to read it. And is it wonderful, but no one reads the bloody papers. Yeah, I get asked the peer review all the time, by the way, in our peer review some things. And I look at the other peer reviewers comments, and I think you haven't read this paper. You have not you cannot read this paper. If you're saying that. If I do pure I don't do much peer reviewing, because I just say no, I'm good. I've read the bloody paper.

Steven Bruce
It must be incredibly time consuming. You've got to check their references actually add up to what they say they do. You have

Malcolm Kendrick
to check the references. You have to you have to check the figures. I'm not very good at statistics. I mean, I understand there's a broad concept. But when someone says some equation, it's 53 pages long and using chi, I have no idea. I have some friends who are very good statisticians and go.

Steven Bruce
So we're getting I'm getting off the top. So to some extent, but I mean, the medicine ought to be based on evidence. But actually, if the evidence is so difficult to find your way through or to deceive the good from the bad. How does it make progress? Well, tell us a little bit about that.

Malcolm Kendrick
Well, I mean, well, you know, you can read Richard Horton who published a paper, the lancet published a paper on what's it called hydroxychloroquine is a big, big, huge debate about hydroxychloroquine at the start COVID. And there's a group called surges fear, who said we have this fantastic research and we've shown that hydroxychloroquine harms people and kills them, right. And the peer reviewers and the journal itself had let all that go through it was only some other people I think it was Australian researchers who said, I work in that hospital. And we never had any of these patients. So and then Have they got the data from? Turned out? They just completely made it up. They had completely made it up. And it got published in The Lancet. And Richard Horton said, Well, how are we supposed to know if they made it up or not? Well, you can try asking your hospital or to, you know, or something. It's your job, surely. And recently, I mean, you just to scare people out there that the level of data fraud I was just reading a paper is, is at least 20% of data are just completely fraudulent now, isn't it?

Steven Bruce
I'm sure it's the editor of the lungs. And I presume it was Richard Horton. Isn't he the one who said that you just can't trust? Yes. And medical evidence? Now? We

Malcolm Kendrick
have no idea. Well, I think he said as well, we don't know. How are we supposed to know? And saying, Well, if you don't know, how does anybody know? How do we know that this? I mean, there's a big monoblock thing. You know, the whole thing about Alzheimer's recently was based on amyloid beta amyloid plaques are the thing that build up in your brain and cause you to get Alzheimer's. Now, that was based that entire hypothesis, which rules the world of Alzheimer's disease was based on and they now admitted it was fraudulent research. They just made it up. They're the ones that have this. And they've said, Yeah, we're looking at, no, here's direct evidence that they made up their results. But we're, we're looking at it, no, get rid of it. Billions have been spent on this huge amounts of a drug was recently approved in the United States, because it reduced beta amyloid plaque buildup to zero, in fact, so it got launched, it's hugely expensive. And they had no clinical data at all other than reduction of beta amyloid plaques. And when you looked at the data, some of the

data, it's it showed that the dementia progression was more rapid in the treated group. So that actually damaging and harming people, based on the assumption that the results on beta amyloid in Alzheimer's were true, when they were made up now become part of a organisation called Broken sights initiative in the States, whether we'll be successful or not, is to try and highlight the fact that this stuff is just being made up, I was looking at a paper on this as cholesterol lowering, not statins, a new drug. Where, as it turns out, 35% of the deaths in the study group were when nobody knew what they died off, right? They didn't have a cause of death. But there were sudden deaths. And they assumed they decided to assume there were cardiovascular deaths, no post mortems. They call them autopsies in the states were done. So they basically just made up the data, they made it up. They're making it up. And if they can do that, on this drug, these drugs, which are really high profile, and and then people say, yeah, they made it up, and it's almost like, well, and so so you know, it you're gonna say is all the data been made up? No, clearly, not all the data has been made up. There are a really good researchers out there doing really good work. But there are other researchers out there. And specifically doing not very good work. And, and, and the regulatory authorities are almost just going. Yeah. Yeah,

Steven Bruce
which is, so I've got lots of questions. And I want to sort of, I want to save some of them until a bit later on. So now we're gonna get on to these topics. So can you talk what, what is the current pathway for someone, which leads to their diagnosis of cardiovascular dysfunction of some sort? I'm not gonna say they've got cannabis. But they're told they have

Malcolm Kendrick
Well, well, yeah. It's like these questions. Listen to what we say. And you asked me a question like this. Kaboom. You know, it's everywhere. But now, it cardiovascular disease is, of course, a really wide spectrum. And it includes many things. But what most people think of cardiovascular disease would be atherosclerotic cardiovascular disease, thickening of the arteries in your heart, your neck and around your body that reduce blood supply, and then can cause heart attacks and strokes include strokes. Yeah. Oh, absolutely. I mean, it's the same. You know, it's fascinating. So you can either had a heart attack or a stroke, and then obviously, you're going to be, you know, diagnosed with it. That tends to be where the diagnosis starts, you've had an episode, maybe you get angina, where you get pain because of lack of oxygen. It's applied to the heart. Other things can happen as well. It can damage your kidneys, it can damage your eyes, it can do all sorts of things, but generally, someone will have to have a symptom that will then be diagnosed. Not always, but that's sort of how it goes. So that would be it once you've had a symptom. Some you're called someone who's had diagnose heart disease. In the clinical trials, they would call that secondary prevention, you've had a primary event or trying to stop a secondary event, people who are treated before they have any diagnosed cardiovascular disease. That used to be called primary prevention, which is what most people are, we don't know you've got cardiovascular disease, but our risk calculator says you are at high risk of it. And they're now called low risk and high risk is,

Steven Bruce
I suppose that's the population that I'm most interested in. Those are the ones who are going along and are going to be given some advice on how they avoid cardiovascular disease. And that's going to start with tests, which are going to tell us about Yeah, well, why would why would a GP put someone out test their cholesterol levels? Is that something you do as a routine

Malcolm Kendrick
standard, it's routine. Well, the UK they've got a thing called quaff, which is quality outcome framework. You get paid for doing things like measuring their cholesterol, measuring their blood pressure measuring this that the next thing and you get paid for doing so doing and if their cholesterol is here, you get paid for putting them on a statin. If your blood pressure's here, you get paid for lowering their blood pressure. If they've got diabetes, you get paid for putting them on diabetes medication and bringing their sugar

Steven Bruce
down people who would say that's not a good model for healthcare.

Malcolm Kendrick
There are a lot of people who would say that's not a good model. And I would be amongst them. In fact, when quaff was being coming in, I was part of the BMA negotiation committees, blah, blah, blah. And, and I threw myself on the tracks on this one and said, Don't do it. And then the train ran me over that was that because everyone thought this was going to be wonderful, you know, based on evidence. It's, it's fantastic. And the review on coffees, it hasn't achieved anything at all. Nothing. In fact, all we can see is it might, of course, some damage. This is not just it's not me. It's been published in the BMJ in The Lancet, and did it dark cardiovascular screening. Is cough still, no, it's still going. It's gone in Scotland. People have been trying to get rid of it in England and Wales, but there's clinging on as if this is this will be the isn't the like Putin hanging on in Ukraine that we cannot give you in other ways we will have, it will demonstrate we were wrong about it on the first. So given

Steven Bruce
that you're going to be paid for taking somebody measuring someone's cholesterol. What's the what's the trigger point when you say I'm going to measure your cluster? Or is it seems that

Malcolm Kendrick
you do a good job, keep changing the regulations. So I'll probably say something wrong here. But as if you're a bloke, you'll get a cardiovascular screening thing done when you think it is 55 Women 60. And this testing will be done on old tests, your cholesterol, your blood pressure, and blah, blah, blah, all the usual things, and then start treating you for various things. So that's kind of how it works. Yeah, yeah.

Steven Bruce

Right. And you're being paid to do this. And now you're being paid. Because I mean, what is you know, what? I suspect you do what the current goal for cholesterol level was,

Malcolm Kendrick
was part of it. There's a thing called vaguely discussed, called Q risk two, and three, which is a risk calculator. You can go on the internet and type it if you typing Q, risk two or Q risk three, it'll come up straight away. And it's basically with curious, Korea's the latest one, and it's 20 factors. Most GPS don't know this, but I separated them out and said, What are the factors are they looking at? And so then it'd be awaited. So you'll have, you know, actually, you do not get your LDL level checked. By the way, this is not part of the calculation, LDL being the bad cholesterol. So you say, Have you got diabetes? Yes or no? Do you smoke? Yes or no? How old? Are you? What sex? Are you? Are you from an ethnic minority, which is a slightly trickier one? What is your postcode? There's another one. And you put all these in, and it will, and then you press go, what's your blood pressure data, and it will say your risk of having a cardiovascular event in the next 10 years is, whatever it is. And if it's above 10%, you will be advised to take a statin for the rest of your life in the US at 7.5%. Because they use the same calculator. No, they use it and called ascvd. Right, but it doesn't use as many factors. And then at that point, they'll say you need to, they say we tell everyone to take exercise and dietary stuff and blah, blah, blah, lifestyle things are first but the reality is that never happens. They just want you on a statin and that's it, and it or if your blood pressure's high, you get your blood pressure lowered. If you've got diabetes, or low your sugar level, and that's kind of about it, really. There's a

Steven Bruce
lot of people who will be very critical of GPS for doing that. But presumably a GP is seeing however many dozen patients a day at five minutes each and the simplest route is to say the NICE guidelines say do this, that's what I'll do.

Malcolm Kendrick
Yeah, yeah. Well, the cough is not quite the same. Let's not go down that route. Let's say it's nice. Yes, yeah. And, yes, as a GP, if you don't do quaff, you'll lose, you will, you will go bankrupt and lose all your money and you won't work anymore. Because you won't be able to make a profit. And you won't make a profit. You can't pay yourself and you don't pay yourself and you can't feed your children. So there's quite a lot of incentive to do this. It's not just cardiovascular, it's also there's other things that are not cardiovascular. But this is quite a major part of it all. So you get paid a lot of money for doing this. Yeah, I'm, anyway, you've probably gathered what my views on this.

Steven Bruce
And I know it'll go down well with the audience, because, of course, everyone likes a bit of controversy. But also people like me is backed up with evidence. And so I knew we would have got to this eventually, anyway. But Keith asked earlier on the statins

cause weight loss and is that how they affected type two diabetes, I know that is covered in the the clot thickens, that can actually

Malcolm Kendrick
starting to increase weight can actually cause weight gain. In fact, there's quite a lot of studies that have shown that people take statins start to think I'm protected. So I'm not going to do anything else. They take less exercise, they do other unhealthy lifestyle things. That's, that's and they make diabetes worse. It's actually a warning on the drug insert. People may develop diabetes taking statins, and there's biochemical reasons for that, which I have covered. But no statins will make diabetes worse, or they'll raise your blood sugar, they will not help with weight loss. They don't that's not how they're supposed to work. The argument for statins in diabetes is if you've got type two diabetes, we're not talking about type one here, you're in your three fold increased risk of dying of cardiovascular disease, possibly higher. Now, statins reduce the risk of cardiovascular disease, therefore, that all people with diabetes must take statins, it has nothing to do with your blood sugar level. That's not what they do. So whatever your blood cholesterol is, you will be advised to take a statin if you've got type two diabetes, if your cholesterol was one of point one, or zero, you'd be told to take a statin.

Steven Bruce
So do they have other effects than lowering

Malcolm Kendrick
cholesterol? Well, we've discussed this before. Yes, they do. I was just reading a paper showing that basically, starting last time I looked, I counted 38 off target effects of status. All drugs do more than one thing. They all they all crash around human physiology started, the main benefit of statins in such as they have is that they are actually quite reasonably strong anticoagulants around about the same strengths as aspirin, right. And that effect happens very quickly. So they have an anticoagulant effect, they stop blood clots. And as we know, drugs that stopped blood clots reduce the risk of heart disease, they also lower blood pressure, they do it through the same mechanism, probably won't discuss that year, but they lower blood pressure, they lower your blood clotting. And therefore these are probably the two main impacts that they have on reducing cardiovascular disease risk.

Steven Bruce
So this sounds contradictory, because now you're saying well, they do reduce cardiovascular risks, and therefore they are a good drug.

Malcolm Kendrick
Well, aspirin reduces cardiovascular risk, but there's a long argument about the adverse effects of aspirin outweigh the benefits because the benefit is small. And then the adverse effects are

Steven Bruce

greater. And we're starting to see that the same

Malcolm Kendrick
Well, I believe that the adverse effects of statins are far greater than our at the moment we've been given this message that statins cause no adverse effects. It's all in no SIBO effects and others, we think they're going to cause an effect therefore they do. But that's based on this same research group in Oxford, essentially. Now, I have looked at more recent studies where they've been double blind, placebo, whatever, for whatever that means. Crossover studies, where 43% of people who are given statins reported adverse effects, ranging from pretty minor to pretty major. And I've seen people die from taking statins, they can kill you. This is known. I'm not saying anything that is not known here. This is not controversial. statins can cause liver failure such that leads to death. It's not common, but it can happen. And statins can cause a thing called rhabdomyolysis where your muscles basically dissolve. They break down the head for the kidneys because there's so much crap basically arrives at the kidneys, your kidneys fail, and then you die of kidney failure. rhabdomyolysis has a 25% mortality rate. And statins cause it in one in however many 100,000 people, doses, whichever metric you're going to use. So yes. You know, you say these things people say, Oh, this will be yes. But it's so rare. It doesn't matter and you go, I've seen three people died renting. Absolutely not cause I'm one GP. There's like 40,000 GPUs in the country, if every one of them I've seen three, that's 120,000.

Steven Bruce
So which I suppose the obvious question is, will be about how many people didn't die early? Because they would say,

Malcolm Kendrick
Yeah, well, what? Well, I know the answer that question in primary prevention, the answer that question is, is zero. It doesn't prevent you from dying. They don't prevent you from dying. There's another way of looking at the

Steven Bruce
story that didn't prevent you from dying early, because obviously, nothing is going to prevent you from dying.

Malcolm Kendrick
That's true. You catch me? No, no, no, no, I'm not trying to catch up. It's something I always say to people, you're gonna die. Nothing will prevent you dying. It just depends how long you extra they give you a study by Christiansen in BMJ open and they looked at this and said, Well, how many extra days do you get, whether that's statistically significant or not. And they found that if you took a statin in primary prevention, looking at nine of the major studies that they had, the increase in life expectancy was three days for over five years of treatment. So that point, seven, five days a year for taking a statin. Now, that is as beneficial as you can get. There's a group in America called NNT, which is number needed to treat who make the

statement on their site. Statins do not affect overall, or cardiovascular mortality in primary prevention. 95% of people who are treated to statins are primary prevention, taking it without known cardiovascular disease.

Steven Bruce
I think you did seem it might have been the cholesterol con, I'm not sure that actually if you've had a prior cardiovascular event, then they can be beneficial. Is that still?

Malcolm Kendrick
Yeah, yeah. I do agree that they have shown some benefits and secondary prevention. But the benefits are, I mean, we're talking here about really minut amounts of time in secondary prevention, the figure was 4.1 days of increased survival, per five year, five year treatment is point eight of a day, a year. Now, you may think that's worthwhile. People think, Oh, well look for an extra year. But remember, are two things. What the statin promoters say is, yeah, well, obviously people are taking them for 30 years, and these benefits will get greater and greater. And I will say is, well, you don't know that. And the other thing is, are they causing damage? If you if you did a study on people smoking cigarettes for five years, you would find no difference in overall mortality in the two groups. Does that mean it's that there's no danger or no damage? I've seen people who have become ghosts of themselves taking status for 20 years. They are just become a bit like Gollum. And

Steven Bruce
I don't want us to just go over stuff that we've talked about before. But I think some things are definitely worth emphasising. And one of the points you made on, I think the broadcasts we did in Manchester, was that one of the reasons that the side effects the adverse side effects of statins are possibly not as great it would seem is that GPS don't ask the questions correctly. So if you if you if you say to somebody, is your memory, is your memory going or something like that? They're gonna they're not gonna say, they probably can say, No, it's fine. But then their wife or their partner, I say, No, he's been a lot worse.

Malcolm Kendrick
Absolutely, you are some one of the things going back in the world of cholesterol was they noticed the first drugs that were given to lower cholesterol. This is 1970s people were more likely to die of violent death, accident or murder or whatever. And they said, Well, this, is this just a coincidence? Or is it a real effect? Where if you go, you can find criminals. 75% of criminals have got low cholesterol levels. In their blood, violent criminals have low cholesterol levels in their blood. And if you ask people who are on statins, about their sense of irritation and aggression and anger, if you ask those questions, every single one of them, or their close relative more likely will say they've been almost impossible to live with since they've been taking these bloody drugs. I was doing an interview in my house with a Dutch crew, and I was talking about this. And there's a guy doing it. It was curtains in the back. And I was talking about the same. That's exactly what happened to my wife. She's that she became she's become unbearable. She's so angry and irritable. Of course, it's not

done as an adverse effect. It's not something you'll tell, no one will make those connections. But there's, there's there's a researcher in the states BEATRICE GOLOMB has been looking at statin adverse effects for the last 40 years. And she says this is one of the primary things that she's noticed as people become really irritable and angry. about things, and when they stop, it goes away. Now, you can argue about why that happens. mechanisms are there and potentially clear and yes, 25% of brains made of cholesterol, dry weight, it's essential for the production of sinuses. It's just an essential neuronal function. And you knock that down, you're gonna what's gonna happen in your brain? Well, it's not going to be good, is it? And, you know, I believe there is a huge amount of not done research out there. Demonstrating, and muscle pains in joints, you'll get this such a lot. Muscle pain, 40% of people. So if you're talking about this group, why is it important? Someone's getting muscle pain and joint pain and difficulty gripping? And you say, are you on a statin? Well, let's see what happens if you stop it for a bit.

Steven Bruce
Again, we I asked you this before, where do we stand as physical therapists in saying to a patient? Well, I've tried stopping aesthetic? Well. Of course, he's outside our scope of practice. It is

Malcolm Kendrick
outside your scope of practice, but I think you can you can suggest or seen a lot of people like this. Maybe you want to go and speak to your

Steven Bruce
GP? Yeah. It's a lot of them. You might want to read one of these books.

Malcolm Kendrick
I mean, it's ridiculous, isn't it? You should be looking at causes of things. Here's a potential cause of a thing. And you're not allowed to discuss it. I mean.

Steven Bruce
Can I turn to a few of the questions? Yeah. I'm going to ask, Pippa sent this one in earlier on, she says as someone with familial hypercholesterolemia, I'm staring down the barrel of statins having been told that diet will play only a very small part in cholesterol levels. I've been taking plant sterols, a gramme a day, as well as omega oils and turmeric with curcumin, but haven't seen any real decrease in cholesterol levels. 8.8 At last check. I do 10,000 steps every day as well as on the exercise as well on the assessment and obviously tried to eat healthily also, what else could people like her do to reduce their cholesterol level? Well,

Malcolm Kendrick
well, well, you can take a statin they'll reduce it. Or you can take one of the new injectable pcsp Nine inhibitors that will reduce it even more. Or what you could say is what I would say to you is stop worrying about it.

Steven Bruce
Familial Hypercholesterolemia go tell us a bit about that though, because it is single out I think, because a group who are prone to cardiovascular diseases and

Malcolm Kendrick
yeah, it is single that it singled out due to illness I hate to go back and all the research is rubbish. I've written a couple of papers on this. And the study, the study where this comes from is obviously originally, it was Goldstein and brown looking at the thing called LDL receptor sits on all your cells, millions of them in your liver, and they pull LDL out of the bloodstream. LDL is what your low density lipoprotein is what people are talking about when they're talking about cholesterol, in reality, so although it's called familial hypercholesterolemia, it's actually familiar LDL EMIA. The terminology is terribly

Steven Bruce
stupid and confusing. But basically, it's a genetic

Malcolm Kendrick
genetic population, about one in 500. People have the heterozygous form of this. So there's different forms of it. About one in a million people have homozygous familial hypercholesterolemia and their cholesterol levels are like 40 and stuff. And they die young have heart disease, it's like our prove it. But in the UK, we have a thing called the Simon broom registry where they've looked at people with familial hypercholesterolemia, and monitor them over years and years to see what happened. Now, there is a small group of people who died young of cardiovascular disease with familial hypercholesterolemia. I think the total number out of the 1000s, it was eight. So we're talking minut numbers here. What they also found was after the age of 50, if you have FH, you live longer, and you're less likely to get heart disease. So to me, this is like saying, Well, if you smoke before, you're 50, it's going to kill you. But after 50 actually protects you from lung cancer. Let's think that through again. So what what, what we looked at was, this is the yellow fingers and lung cancer argument, again, is that people who have yellow fingers are more likely to die from lung cancer. Yes. Why have they got yellow fingers? Because they smoke, not the yellow fingers causing the lung cancer. This is when you have what's actually the causal agent is going on here. Now, when you look at familial hypercholesterolemia, there are subgroups of people within this who have a blood clotting factor abnormality. Now, when they looked at identical twins, not identical twins, when they looked at twins, one of whom had the the gene FH gene and had the high cholesterol, and the other one who didn't have that gene and didn't have the high cholesterol. They have the same risk of cardiovascular disease. So Another gene, this is smoking. But you're looking at his yellow fingers. When you're looking at the cholesterol level, there's something else going on in there, it's actually causing heart disease, and that is blood clotting factor abnormalities. And you can show them, because I'm not going into the exact details of this take too long. But the LDL receptor itself, which sits on cells and pools, LDL molecules out of the bloodstream, also takes that factor eight, it has a very important blood clotting factor control issue with with the LDL receptor society. What is a receptor do? Well, it takes LDL out.

That's it. No, it does all sorts of other things as well, which are very important. And some people have this and some people don't. And if you split the sections out 98% of people with familiar however many years, we have this double gene, and nine and 5% of them. So don't have this double gene and 5%, do these 5% who are at risk. It's not because of the LDL, because of the clotting factors. So so you can go by the way, she can go to her own doctor who will know none of this, understand it less well dismissing that company completely.

Steven Bruce
That's where I was going with this because I knew he, quite rightly, who said you're not going to give medical diagnosis over the Internet to anybody who you haven't actually had in your treatment room and examine so people can't take this as her diagnosis or recommendation now, if so, who should she go to? Where she can get a reliable?

Malcolm Kendrick
She can't there's no one.

Steven Bruce
Where do you practice? You're in Manchester. I don't know where pepper is.

Malcolm Kendrick
There's no, there's no clinical practitioner that will even countenance this, even though the research shows that it is this. And everything shows that it is this. Alright.

Steven Bruce
So from what we're seeing here without having examined pepper yourself, should she be somewhat reassured but what you

Malcolm Kendrick
need to be reassured that people with familial hypercholesterolemia live just as long as everybody else, whether they die of cardiovascular disease. Go back was a study done in the Netherlands, ironically, by one of the greatest cholesterol lowering proponents and the roadside brands is his name. And they look back to sort of history said, Well, we know it's a genetic condition. So let's look back through history of people who died and what ages they died at who we know will have had familial hypercholesterolemia. So, they went and looked at records from 1815 1819, whatever. And what they found was that actually in 1850s, do by the turn of the century, people with familial hypercholesterolemia lived longer than the surrounding population from 1900 to about 1960, they lived shorter, over 1960 onwards, they are now living longer again. Now, the explanation for this is the LDL is quite a potent anti infective agent agent, it locks onto bacteria and viruses and and stabilises immune ism and then immune system comes along and kills them, it plays quite an a very important role. The studies with rats where you have rats with high LDL, you stick nasty substances into the bacterial substances into them. And, and those with higher LDL that if you ever have a thing called lethal dose, lethal dose fancy, you have to

give that what dose do 50% of the animals die? The LD 50 and those with higher LDL levels was eight times as high eight times this

Steven Bruce
is only for bacteria and viruses.

Malcolm Kendrick
That was only for bacteria. They will they will they are part of the immune system. Almost everything in the blood is actually part of the immune system. When you look at it, blood clotting factors are a key part of the immune system. And LDL is a key part of the immune system. And these these stick on to and basically stop them from being infected. And this research is well known, published and disbelieved by everybody to do with cholesterol. So you are less likely to dive in infectious disease if you have high LDL, it will protect you against infections and a small proportion of people will have this clotting factor problem which which is a problem

Steven Bruce
perhaps come back into so that it's very interesting she was also identified as having a haemoglobin variant of no consequence so she has told me she should look more into what that human well

Malcolm Kendrick
you got haemoglobin, well, you got you know, people with blood. Group o are less likely to die of heart disease. People with a B are more likely to die with heart disease. This again, is an immune complex blood clotting issue going on here. All right. All it is fascinating when you start looking at everything connects to everything else in ways that you initially think they don't but they do. This human physiology is just like a What, what, how is that ever happened? It's really fascinating. But the fascinating thing is that probably possibly FH was something that became more prevalent at a time. When infectious diseases were wiping out so many people on this planet, you know, because when people move from the countryside to the cities, and the populations crammed together, and you got all the people, the amount of people tied to things like syphilis and TB, and whatever, having a protective factor, that increased competitive factor in your blood is probably quite a good thing to have. And that's why from 1900 to 1960, it was actually didn't do any good. But once antibiotics came along, it's really fascinating, but don't get worried about it. I've got there's a guy in the states were up to me 10 years ago saying, My LDL, which is the bad cholesterol, is 18. Right? Which is like, and he said, and I've been investigated for many years because they can't work out why I have no detectable heart disease. No, he's been scanned. He's been screened is a disorder. He's looked at every possible upside down way. I thought, well, maybe. And then Then he sent me a paper where he's been studies a case history. Here's a man with the cholesterol, LDL and total cholesterol be about 25 of 18. No detectable heart disease. That's not just one by the way. I can give you example after example. Now the solution to this as apparently the experts go, oh, he must be being protected by something else. No,

the solution is LDL doesn't cause heart disease. That's the obvious and easiest solution to your problem. Something

Steven Bruce
else is causing the heart disease.

Malcolm Kendrick
It's not old it never was. It isn't it can't be it's impossible that it is.

Steven Bruce
So before we get on to what it might be what it is let me ask you one more question because scroll down first and Robin sent this one in I think yesterday but was worried that he might not be here but I'm told Robin is watching a bit too. So you love this is Robin. He's talking about Sally Norton's book toxic superfoods how oxalate overload is making you sick and how to get better and she says the blood vessels are printed damaged from oxalic acid and Crystal accumulation which can lead to tissue degeneration, including cataracts, vision problems, and fatal brain aneurysm. oxalate deposits are found in the arteries and in calcified arterial plaques. And the crystals are associated with blood vessel weakness, vasculitis, stroke and cardiac conduction abnormalities and arrhythmia. Could I ask you how significant you think oxalate damage is in heart disease.

Malcolm Kendrick
I'll quote my great mentor. If you're having a SCAF, who said it's fascinating why you find so many fire engines at the site of fires, they don't cause the fires. There's a lot of things you'll find associated with damaged arteries and all sorts of other parts of the body. Ask yourself the question, did he cause it? Or did it cause them oxidation and Oxalic the human immune system and also the clearing up system uses superoxide to destroy bacteria and viruses. That's what microfinish is do it also uses these to destroy damaged areas in the body. So if you have damage going on, your immune system goes in and oxidises the hell out of it, and then then invade your knees and takes it away. Of course, if you don't get rid of all of it, what you've got left is an awful lot of superoxide to kicking around. This is the body trying to heal itself. This is not a cord. This is somebody getting as everybody does get cause and effect the wrong way round. And this is the same reason why the cholesterol hypothesis is lasted so long. What is the most important repair system in your body? It's cholesterol. And this is

Steven Bruce
true is it though that if you've got cardiovascular disease, you are likely to have higher levels of cholesterol?

Malcolm Kendrick
No, it's not actually true at all.

Steven Bruce

So there's just it's a very strange hypothesis.

Malcolm Kendrick
Well, it now well, that hypothesis began, because when people looked arteries, and atherosclerosis, thickenings and damaged areas or whatever you want to call them, they found that there were a high percentage of cholesterol in them, right? And then they said, Well, where did this cholesterol come from? And the answer was, well, it must have come from the bloodstream, because where else can it come from? Which is, which is a reasonable hypothesis. Therefore, these, there is cholesterol that you find is the cause of the atherosclerosis. No, that's yellow fingers, and lung cancer all over again. This type of thinking is so prevalent in medicine, we find an abnormality and we decide the abnormality is the cause of the disease. We find beta amyloid plaques in the brain and decide that beta amyloid plaques are the cause of Alzheimer's. No Oh, this is the body trying to repair itself, you idiots. We find cholesterol in arterial plaques. This is the body trying to repair itself. This is you getting it 180 degrees the wrong way around as per usual, you idiots.

Steven Bruce
My audience loves it when people get off the fence and they're fairly emphatic about their view, well, I

Malcolm Kendrick
can't be more emphatic this is just such a stupid idea that's caused so much damage, but so many years. If

Steven Bruce
it's going to be it must be doubly frustrating for you because as we already said, It's outside our scope of practice to avoid advise people on what they do about their potential cardiovascular problems. But for you, trying to educate your fellow GPS and the rest of the medical world, because actually as an author, a cardiovascular consultants, presumably who sign up to the cholesterol

Malcolm Kendrick
now they all do. Well. 99.99

Steven Bruce
We'll move on to the clock thickens in a minute, but have there been occasions when you have sat down with the head of the unit at Oxford? So Rory,

Malcolm Kendrick
there was occasion when I was supposed to be debating with him one time when we Cox's Collins, Rory Collins where he pulled out, okay, ya know, you, you kind of debate that debate doesn't happen in science anymore. It's like, someone said, we should be playing tennis, you know, but we're all playing golf. We'll just play our game and come to the end, where we should be banging it back and forward. But you don't get this bang, it is very difficult to get debate with anybody. They usually

just say, Do you know who I am? You know, and that's kind of that's it? Or they are they produce these enormous reports when the European Society of Cardiology, which had like sort of 70 pages of why cholesterol causes heart disease, it's proven fact. And then they selectively pick every fact that they can. And then, you know, I wrote to them the ironically, the man who runs the UK Biobank study is also Rory Collins. You may never have heard of the UK Biobank study. But it's enormous study where they gather genetic and other data about people to try and work out what's causing illnesses, which is, at least in theory, quite a good idea. Although if you're a pharmaceutical company, you get first dibs on all the information for the first five years or whatever it is. And they looked at cardiovascular disease in the UK Biobank study and said, Well, what factors do we find increase cardiovascular disease? Number one was previously having had cardiovascular disease, big surprise. Number two is diabetes, essentially, big surprise, smoking was pretty high up there. And you go down all these factors, and then they came to cholesterol. For each one millimole increase in cholesterol, the increased risk of cardiovascular disease is one. And one is basically your I would say nothing, no risk, this is average risk. So there was no difference in cardiovascular disease death, with every one millimole increase in cholesterol, none zero. It was 1.01 and 1.02, which is essentially one of one is no increase in risk. So they produced a study. And it's in the BMJ about four years ago, and I read it. So there we go. You have seen no difference in cardiovascular risk, no matter what the cholesterol level is nothing. Zero zip nada. Okay, so what do you say about this? They didn't even mention it in the abstract. They didn't mention it in the discussion. They didn't mention it in the results. They did table, you had to go into like appendix 73, subsection four to find distinct, but it was there. I wrote to them. Of course, I didn't reply thing. Interesting. I noticed you found it was no increase in the risk of heart disease with an increased cholesterol level. And what's your explanation for this? Do you have an explanation?

Steven Bruce
Aloo Simon, sent in a question a few minutes ago and Simon, I hope you'll forgive me for phrasing it this way. But it seems to me that what Simon is asking is possibly a very common response to what you were telling people because he says, Do you think that cholesterol particle size has any relevance in this because he's read that it's not your cholesterol count matters, but what makes up that count? And I suspect that lots of people will turn to you and say oh, yes, but it's something else in the cholesterol. Yeah.

Malcolm Kendrick
This is what I call throwing chaff into the air. You know, someone tries to shoot down an aeroplane it throws chaff up and go. Well, essentially, yes. We started out with cholesterol, right. And then, oh, no, it's not cholesterol. It's low density lipoprotein. Oh, no, it's not loaded. Is the lipid protein it's low density, low protein, high density lipid protein ratio. Oh no, it's the non, the non HDL level that counts. And then we had high light and fluffy LDL. And then we had small dense LDL, we still got all of these things, they still swirl around. And now we've got particle number, right? What was the difference in a particle number and the total number of molecules? Explain this to me. That is odd. It's not that it's the April be lipid protein, April, April routine is

attached to the molecule that counts. And then now it's the ratio of the site. Guys, guys. Stop. It's got nothing to do with any form of cost or whatsoever. In fact, you have no cholesterol in your bloodstream anyway, it's all carried around in lipoproteins. Like people are intact. It's a bit like saying, you know how many people are there on the motorways? I don't know their own cars was how much cholesterol you got in your blood. And I don't know it's all inside. lipoproteins. Oh, it's the particle particle, what? What's the difference in the number of particles and the actual total number? Because they seem to be suggesting something here that stretches the possibility of of logic snapped beyond every time you looked at these things. Isn't that the last time I looked at HDL, which is high density lipoproteins, I'll give you a story about high density lipoprotein is supposed to protect against heart disease, because it sucks cholesterol out of blacks, takes it to the nearest LDL molecule transfers it to that and it goes back to the liver, and it's taken out of the system. And they found a group of people living in Italy who had almost no HDL, and they had no heart disease. And they said, Oh, right. They have a special form of HDL, which is specially protective, even though they haven't got as much of it. They called April a one Mallanna. Alright. And they created a boy one Milano and a laboratory, injected it into people and said, Now, they're atherosclerosis will disappear. And certain people became very rich on this because this technology was sold for a billion dollars to Pfizer, and Pfizer on some early results by a couple of guys that I won't mention the names because otherwise I'll end up swearing at them who said, This is amazing. The plaques disappeared, like you know, snowing a dike or whatever. And, and then Pfizer did a study and they said actually, nothing happened. The proper study, so this product kind of died. So you know, particle sized particles oxidised LDL, D oxidised LDL, it is nothing to do with now, you can flip it around in a million different directions. And they have and it still continues. I keep reading papers about oh, it's whatever form of LDL or it's the amount of, of esterified cholesterol within the LDL was? No, you can't do this because it's nonsense. It is utter nonsense. It's the same thing. You're just changing the name and flipping it around and saying, Oh, well, it's not this. It's that? No, it's none of these things. It's nonsense.

Steven Bruce
I've got a few other questions here, which are all asking variations on the cholesterol theme is, is it on low total cholesterol is one of the questions here in a bit. But if we just leave it that cholesterol has nothing to do with heart disease, as far as your research shows, yeah, well,

Malcolm Kendrick
it has nothing to do with that. Yeah. Okay. So

Steven Bruce
we forgive me if I if I don't ask these questions specifically. No, no,

Malcolm Kendrick
no, no, I understand. You're trying to move people from something they've heard banged out for the last 30 years, by everybody, by the experts. How can this person

possibly be right? How can they say that what they're talking about is right? How can they be right? Because the evidence or not, the evidence says I'm right. All right. Is it drug out there called Go Repatha from survey, otherwise called ever Luca mob, which lowers cholesterol, LDL, by 60%. Alright, 60% more than any statin alright. And, and the study, study study was a four year study. And it was recently re re reviewed by by a group called the restoring abandoned and incomplete trials. There is a group that do this. And they went back and reanalyze the data from the trials and will actually it's even worse than we thought this drug reduces LDL by 60%. Right. And in this trial over two years, the difference between the people that took it and the people that didn't take it was that there were 114 heart attacks and the people taking the Repatha and 80 in the people on the placebo. And the overall mortality went up as well. The overall mortality went up I don't

Steven Bruce
know what the total numbers but that was a statistical statistically significant was

Malcolm Kendrick
not statistically significant okay. But then statistical significance is another one of

Steven Bruce
the ways in artificial,

Malcolm Kendrick
just men anyway p p values are just you know anyway, p values in peer review two peas we should be getting rid of. And anyway, more people died before statins came along there were other cholesterol lowering agents clofibrate that lowered not quite as much a similar amount, increased overall mortality and cardiovascular mortality and with overall mortality, it was statistically significant. There's a whole bunch of other drugs that no one's ever heard of, they will never launch them are called traps for craps. He was a trap towards the trap it reversed enemy. Billions were spent on reviewing reviewing this because they lowered LDL by as much as as statins. In one case, they increased HDL by 130%. And none of them at any benefit and cardiovascular disease. In fact, one of them increased cardiovascular disease by 65%.

Steven Bruce
Which just added some weights to what you said earlier on that if statins are beneficial, it's not the cholesterol lowering well.

Malcolm Kendrick
We have drugs that lower lower LDL more than statins, which have had no benefit on cardiovascular mortality. We have drugs that don't lower LDL at all that have a benefit on cardiovascular disease like aspirin and reduce blood pressure lowering drugs and, and things like anticoagulants. And then we have statins that lower LDL and lower the rate of heart disease. So what does this tell us? It's not the lowering of cholesterol. That is the thing. It can't be.

Steven Bruce
What is the thing? Dr. Kendrick?

Malcolm Kendrick
Well, the thing is something that people have been talking about for 170 years,

Steven Bruce
just very quietly.

Malcolm Kendrick
170 175 years now. I wonder somebody when you years. So it's essentially when you look at it. Heart disease, the plaques, when you look at plaques, the thickenings and blood vessels. And I've had pathologist come up to me and say you're right, or by the way, you know, this is what we are looking at. The idea is well, the first original person was Carl von rocky tan ski who looked at plaques in arteries in Vienna in 1850s. And said what I'm looking at are blood clots in various stages of metamorphosis and repair. He said that then he was like one of the first people ever to look at this under proper view, microscopic examination. And this is true, if you look at plaques, they are essentially blood clots in various stages of development, metamorphosis and repair. That's what they are, right. So you say well, how can how can other people not have noticed this? Because what happens obviously in repair, you know if you if you cut your skin or you can get keloid in skin, especially with the dark people, they develop these locks what you find in a key Lloyd is not it's not that there's been damaged, too skinny, she's a big lump of thing. All right. That's the body repairing itself inappropriately in this case, once the body starts to repair itself, what's in there looks nothing like what was in there originally. If you look at a blood clot, after two weeks, there's not that much stuff that you definitely say was blood clot but there are things in there if you look at them closely enough. See, ironically, one of the things that was first seen inside atherosclerotic plaques was cholesterol crystals, actual crystals of cholesterol. So like sharp pointy things. Yeah. And they're not that long,

Steven Bruce
small, sharp, pointy things,

Malcolm Kendrick
very small, sharp, pointy things. And that was what first directed people to think arts cholesterol, because they found that this was pure cholesterol, right? And you say, well, that's fine. Great. Yes, it's there. I'm not gonna say it's not there. I'm not going to say that's not cholesterol, because it is. But you cannot make a cholesterol crystal out of the cholesterol you find in a low density lipid protein molecule, because you can only make cholesterol crystals out of pure cholesterol. And cholesterol is carried in low density lipoproteins as a thing called a cholesterol Ester, which is attached end to end with a fatty molecule. That's a cholesterol ester. That's how it's carried about around the body. It's not carried free. It cannot be carried free. MIT chemically,

it can't. So where do you get cholesterol crystals from? If not LDL? The answer is there's only one tissue or substance or whatever the exact term is where you can find cholesterol of sufficient purity to create a clear crystal, and that is in the membranes of red blood cells. It's the only place in the body that produces sure your 100 papers, we, we know that the only place these crystals could could have come from was red, red blood cell membranes, can't make it out of LDL. So when you find cholesterol crystals, you know, there's been red blood cells in there. They have to have been in there. So where the red blood cells come from, well, you have red blood cells in clot. That's why if you cut yourself and you get a bleed, and then it forms itself into a scab. It's primarily dark red

Steven Bruce
wrestlers, you'd have a drug to reduce blood red blood cells. And

Malcolm Kendrick
then there are people who say that if you'd use iron, you'd use the risk of heart disease because they found that people who are anaemic are less likely to die of heart disease. Yes, it's not the iron. Okay? No, you don't want to make people anaemic. But red blood cells, again, in Britain, it's amazing that the the link in red blood cells link to fibrin, which is the other part, and they they attach to it. And then the red blood cells shrink into sort of dodecahedron or shape and pull their blood clot really tight. Red blood cells are the tightener uppers of blood clots. Amazing. Anyway, you find red blood cells there, you find fibrin there, you find fibrin remnants. Where do you get fibrin from fibrin? There's two things that make up a blood clot. There's lots of things that make up a clot with this two essential things. The platelets start it they gather together, then red blood cells get drawn in and fibrin forms around it like a fishing line. And then the whole thing goes. And fibrin makes the clot really, really, really difficult to remove. And, and that's what holds it together. And if you find fibrin inside a blood vessel wall, which is you find it in blood vessel walls. Where does it come from? Well, it you're not going to form fibrin just spontaneously inside a blood vessel, it can only have come from a blood clot. So you've got the remnants of red blood cells, you've got the remnants of fibrin. Yes, you have cholesterol and and you have LDL molecules in there. So people have said unbeknownst to every doctor that I speak to, there is another form of LDL that floats around in the bloodstream. And it is identical to LDL except it has a protein attached to it. And this protein is called April what was the story about these horrible names April lipoprotein A. And therefore the LDL molecule with this attached to it, it's called LP a lipid protein, a small a LPA nobody knows this exists, nobody really knows what it does. Alright, so the body doesn't produce stuff that has no function. The fascinating thing about LPA is that when you have a damage to a blood vessel LPW A is attracted to it sticks to the area of damage forms very tight bonds with it. And therefore plugs is one of the original plugs for damaged or blood vessel. And then the April open Protein A comes in this is I just find this fascinating other people maybe don't but anyway, April I for protein A when a blood clot forms, all sorts of things get pulled in, like just forget, it's just, it's like what? Anyway, one of the things you get thrown into a blood clot is a thing called plasmin. Oh, Jen, like fibrinogen plasminogen is, is a pre enzyme, it's not active. It's drawn into every single blood clot. I don't know how much it's on it. And

this is amazing. But if you want to blow up a blood clot, you, you you tissue plasminogen activator is a thing that's produced by the body tissue plasminogen activator comes to the clock. It locks in it converts plasminogen to plasmin and plasmin slices fibrin apart. And so the blood clot disintegrates.

Steven Bruce
This is the stuff they inject into people having a heart

Malcolm Kendrick
used to be then I do other things. But in the older day, if you having a stroke, they'll give you TPA isn't called different things. And it busts the clot in your brain and stops the stroke from causing so much damage. Obviously, you don't want to give it to people who are having a bleeding stroke because otherwise it'll kill them. But if you're having a blood clot caused stroke in your brain, you give them TPA. So TPA tissue plasminogen activator activates plasminogen plasminogen turns into plasmin plasmin slices fibrin apart fibrin the clock starts to be dissolved back in the story April lipo Protein A is identical plasminogen apart from how it's folded at the end, right? To the TPA comes across an LD LPA molecule or the API protein a molecule comes, I'm going to activate you and it goes, you can't activate me. I'm not plasminogen. So that clot does not dissolve, fully, if at all. All right. But of course, you then have this clot, which is stuck to the artery wall, and it's got TPA, it's got an LPA in it, which is LDL way under the knee. And then what do you do with it? Well, you can dissolve it around a bit, because the your dissolve away. Because if the clock started that size, you reduce it to say that, whatever that size, but you still got to do something else with it, you can't let it break off and travel down the artery because it will just get jammed somewhere further down the system. And could cause a heart attack or stroke, a smaller heart attack or stroke. So your body has to do something with this clot that stuck to the artery wall can dissolve it so far. But then it runs across April, April, Protein A and it's stopped. Because obviously, if you could just keep dissolving it, all it happens is you got a blood clot, it completely dissolved. And then you say, Oh, my God is an exposed area. Now the blood clot dissolved blood clot, well, that is a stupid system that won't work. So it's a bit of the clot that's closest to the artery wall has been damaged, stays there and doesn't get broken down. That's the function of LPA. So then this thing that sitting near has to get covered over by a new layer, a new endothelial layer, which is the layer that lines all arteries, right? And we know this is one of the reasons why rocky tan skis was never an idea was never accepted, because another co worker said, but these are underneath the endothelial these blood clots underneath it and Aetherium How can a blood clot form underneath the endothelial when blood clots form within the blood itself? And rocket TASKI? couldn't answer that question. So like I said, Well, you're wrong. They're not you. And that was the end of rocket attacks these ideas. Okay, TASKI didn't know. Because how would you know that in our bloodstream float around endothelial progenitor cells EP, she sees an air they're present in your bloodstream all the time. And if they see an area of damage, they come across it, they stick to it, they grow into proper fully grown into thelia cells. And then at that point, everything's repaired. But you have a remnant blood clot stuck in the artery wall at that point. Now it's full of

Steven Bruce
fibrin, and that's causing a narrowing,

Malcolm Kendrick
but it will cause an earring. Well, an initial one and initial one probably doesn't work because there's very, very little of an ROI. But if you look at blood, if you look at plaques, and you'll find it in you chop them in half, you find a number of about 45% of them. It's like looking at tree rings, there's layer after layer after layer after layer after layer after layer after layer after layer. You say well, what could have caused all these layers to have formed? Well, it's block after block after block being shaved away and removed. Probably most of them are fully removed, but some of them will get stuck. And if you've got a stuck area, it's probably a vulnerable area where you're more likely to get another blood clot. So these become focuses of blood clotting. And their blood clots build up and build up and build up and build up.

Steven Bruce
So I think we missed out a stage anyway. Because you were talking about blood clots something well, why the hell is there a blood clot? endothelial damage? What's caused the damage? That surely is? Well, yeah,

Malcolm Kendrick
well, yeah. So step back, you're not going to get a blood clot until you've damaged the artery wall, or the blood vessel wall because there's no stimulus for a blood clot to form. Because your bloods system is enormously doesn't want blood clots forming.

Steven Bruce
This is where cholesterol comes in. It's bursting through the artery wall.

Malcolm Kendrick

Well, this is the idea is that is it cholesterol bursts through the artery wall to the endothelium. And then, and then what well, it gets stuck inside the artery wall in some way. And then, then that's where it all happens. Yep, being an idea of the idea might have some validity if you could get LDL through the artery to the endothelium. Now, I've looked at this as the most complicated part of this, I'm not going to go into any great detail, but what I will say to you is, all arteries, the arteries of the size in which atherosclerosis develops, have actually got their own blood vessels to supply them with blood that conveys a visual from the blood vessels of the blood vessels. And there they like form a latticework around major blood vessels. And anything in the bloodstream can come into the visa Museum, and then enter the artery wall from behind, including as many LDL molecules as you want. Because once once blood vessels reach a certain very small size, which is eyes of a visa visa forum is big enough for one, very small, they are no longer barriers to the movement of substances. But obviously, they can't be because otherwise nothing could move in and out of the bloodstream, and you'd just die. So they have what they call fenestrations go holes in them, and gaps in them in the basement membrane behind them start to loosen off. So in your kidneys, for instance, you've heard of the lamella your apparatus, and you've got all these blood vessels in this little cup, and they're capillaries. Well, clearly, this is the point at which all sorts of stuff leaks out, and then goes around your loop of Henle and all that. And if it couldn't leak out of here, then you couldn't just couldn't work. So at the smallest level, blood vessels are leaky. But at a larger level, blood vessels cannot be leaky, because if you allowed everything to leak out of your major arteries into the tissue underneath, you would be dead almost instantaneously. Because your body would just fall to pieces. Now we know this because you've heard of Ebola. And Ebola kills you. And how does it kill you? It's called hemorrhagic fever. As the other word for it, you start peeing blood and stuff like that. Why does this happen? Because the Ebola virus for reasons unknown, I don't understand why is there there's always really tight junctions between between cells, all cells in your body. And Ebola opens up these junctions, especially the junctions in your in your blood vessels. So it removes the tight junctions. And once it does this, all of the blood contents of the blood can go straight to the blood vessel wall into the surrounding tissue behind. And that's why you die of Ebola because it opens up the normal barriers in the endothelial cells and allow stuff to leak out. That's why you get so we've so so from that perspective, unless you have opened up the the barriers in the normal endothelial in the larger blood vessels. Nothing can get through that is not allowed to get through the body very carefully regulates how these things work. And yet people say well, LDL is completely different. It just goes through. Well, why does water go through? Why doesn't even smaller molecules go through? Why doesn't? Why don't they understand themselves? Just like anything go through? Because if they did, you'd be dead.

Steven Bruce

I got you. I got you started on a red herring that didn't like I cheekily said yes, it must be cholesterol when I knew very well, you were going to tell me your why. So what is causing the damage to the

Malcolm Kendrick

damages? Well, an endothelial cell is since this size, was quite thin, like a cell, now, obviously the bloods flowing past them all the time. And quite rapidly now all endothelial cells are also have got a thing attached to them called glycocalyx. Again, you ask 100 doctors, no one no one has any idea this

exists. This is a slippery stuff on this slippery stuff on fishy timepicker

Malcolm Kendrick

proficient in the office sharks don't do this. But the fishy catch. Normally a small fishing, grab hold of it straight out your finger. Why? Because it's covered in glycocalyx, which really slippery doesn't let anything stick to it. And it also acts as a barrier to bacteria and viruses getting into fish as glycocalyx is very important for not letting infections and why if you get too many fish together, like salmon farms, they bash into each other. They're not the glycocalyx off to get horrible infections. So glycocalyx has an anti infective anti clotting, it's got about 20 substances in our anticoagulant, it allows the blood to flow through really smoothly and fast and it doesn't have anything to clot to stick. If you damage the glycocalyx, then basically the endothelial cells are our risk, things start to become can either directly attack them, or bash against them, or do damage to them. And once you've damaged endothelial cells sufficiently that they break off, you expose the underlying blood vessel wall. And when you do that, that's like, that's a red alert. We think a blood vessel big blood vessel getting damaged the body's clock now, otherwise, you're going to bleed to death. So, if you damage the glycocalyx, if you damage the endothelial cells, you will get blood clot forming on that point. Now it may be quite a small blood clot, usually is pretty small blood clot. But we know this happens because if you get a healthy volunteer to smoke one cigarette and then you you look at what happens when they do that. You can see the glycocalyx is damaged. You can actually measure destroyed and dying endothelial cells in the bloodstream is called. It's called what's called microparticles microparticles are the remnants of dead endothelial cells, you smoked one cigarette and the microparticle goes like this in the ceiling at the glycocalyx does this and endothelial cells die around your whole vascular system,

Steven Bruce

which means clots are forming all around, the rest clots

Malcolm Kendrick

must be forming all around the micro clubs. Yes, you're interested see them with the naked eye. At the same time, luckily, this bone marrow goes, oops, things are happening to the endothelium stimulates it to produce more endothelial progenitor cells. So the repair troops come shooting out, find the areas, cover them or sort them out. And these really small clots all over the place, essentially just repaired almost completely probably. So if you smoke, like one cigarette, the other will be damaged, but it will be cleared up you smoked 20 Cigarettes, the other be damaged. But I've cleared up 40 Cigarettes, there'll be damaged but it'll be cleared up. You smoke 40 cigarettes for 40 years, you're screwed. Because the body can only do so much repair. So, you know without disease, it's, of course, if you smoke and you do nothing else, you're probably okay. You need to do other things. And the other thing that really damages endothelial glycocalyx is diabetes, high blood sugar levels strips, the glycocalyx say it's supposed to be that thick. You get diabetes, it's that thick. And you can measure this, you can see it you smoke as well, well, you're doubling your problems, aren't you? And this is why risk factors for heart disease are multiple KTF. Right? So you know, you do one thing wrong, you're probably all right, you do two things wrong. You see things wrong. Maybe four things, you do five things, and you are in deep. So people have to do one of the things you do wrong is getting old because as you get older, your repair systems don't work so well. So the things that you can get away with when you're 20 When you're 50 you know stop doing that right? If you've got diabetes in the background and you smoke right and then there's other things you know, that can cause damage. I was looking at all sorts of things that can damage the endothelium you know what what what what can I show damages that endothelial Alright, well I can show you we can show that smoking doesn't diabetes and these things you can show things that people don't consider led to heavy metal which people So how on earth can What's that doing? I can lead damage endothelial will lead if you if you inhale it, which we all used to do in exhaust fumes, gets into your lungs goes through your lungs, because it's a micro particle. The nanoparticle actually it's even smaller than it goes into your bloodstream and you can show it destroying endothelial cells. It's threatened in the states More people have died of heart disease from lead poisoning the died from smoking. People say how did light goes? Well, because lead does the same thing as smoking when you smoke. Now, particles in the smoke come out of your lungs, travel around your body and blow up your glycocalyx and your endothelial cells. So what else can cause cocaine? Right because 100 Cocaine cause heart disease? Well, if you snort cocaine, you know people snort kicking, what happens is the middle of your nose falls apart. Why does the middle of your nose fall apart? Because it can causes a really intense vasculitis it causes the blood vessels to inflame and die off. Alright. And that's why the llenos falls apart. Because once that once the blood vessels are gone. Once you've inhaled it into your lungs, it gets into your bloodstream. Once it's in your bloodstream, it causes an extreme vasculitis. And vasculitis just means inflammation of the vascular system. People who take your cane in an hour after taking cocaine are 20 to 30 times as likely to have a heart attack in that period of time. It's like smoking except on steroids.

Steven Bruce

We are at the end of our show, which is extraordinary. I mean, the time just flies by and I've literally got three minutes left, but I'm gonna try I'm gonna put you on earth that across here between medical diagnosis and speed dating, because I've got a bunch of questions here. See what you think of these dances? What do you suggest for someone who has cardiovascular disease? What should they do to avoid early mortality?

Malcolm Kendrick

Well, it depends what's causing it. And this is the thing I said there is no a cause that just isn't the cause of heart disease. There's, you know, I made a list once and it came to hundreds of things that can cause it. The important thing is to try and work out what it is for you. I'm talking to someone about setting up a clinic where we're going to be looking at these things in more detail. But essentially, what is it for you and you might be different for you than anybody else? You have to work out why it's happening to you, and what your risks are and then what to do to mitigate them. I can't go through that at the moment because it's like hundreds of things but

Steven Bruce

we're not here. You're not here. Well, I'm not here to plug your You might be here to plug your books. I'm sure you're not. But I mean, there's a good start in reading those books as well.

Malcolm Kendrick

I'm trying to say here are the X number of really the most important causes of heart disease. And what you can do about it, you know, if you get diesel fuel inhalation or vehicle fume inhalation, so if your next big road and you're breathing in diesel particles, yes, they get through your lungs, they cause damage to your blood vessels. So that might be your cause. I don't know with individuals. That's why it's so complex. Really,

Steven Bruce

one very quick one last one here from specie. specie. So she has been told by both a consultant and her GP that she needs to be on statins, then copper doggerel. So the correct pronunciation I call it clopidogrel girl, right. Okay. For the rest of her life, a CT scan revealed what she is told is the result of a stroke that occurred at some stage in the past, therefore, she needs to be on those meds. Does that sound reasonable to you?

Malcolm Kendrick

Now? Because you have to know what caused the stroke again, you know, you can have a stroke, because you've got a hole in your heart.

Steven Bruce

So it could be good advice, but

Malcolm Kendrick

it might not be. It could be terrible advice. What caused it? You know? Is it blood clotting it goes a hole in your heart means means clots that would normally get stuck in your lungs, travels through your heart and go up to your brain. And that's quite common as people call it cryptogenic on cause stroke. Some people have strokes, they never know what caused it. But if you don't know what caused it, how can you know what to do to reduce your risk? You have to get down to the detail. What is it in your case? I don't know what it is in our case. And we don't do the proper screening. We don't do the proper screening in the NHS. So you probably will. So they come to the simplest provision, a statin and comparator go, Well, yeah, well, that might work for you. Because you might it might be that that's those are the things well, the statin won't work. The competitor might be effective, though, you might be getting the right treatment. I don't know. Uh, neither do they. Yeah. Well,

Steven Bruce

let's hope that that helps us, Becky in some way. I know. She's not a smoker. So she's not that risky. But yeah, Malcolm, thanks very much. I'm sorry. We've got some questions here. We've had over 500 people watching and just in case you were uncertain, everybody's loving it as much as literally I really can't recommend enough that you're gonna have a look at these books, though. The clock thickens. The latest one is a fantastic summary of what is likely to cause heart disease, would you say it's certain?

Malcolm Kendrick

It doesn't cover everything. But it covered hopefully 95% of the things that on a population basis visa thing, but

Steven Bruce

it answers the question. Well, if it's not cholesterol, what could it be? Well, there's a very good, very well-reasoned argument in the book about what could cause heart disease. And, you know, I recommend these books to my patients as well, because they're the people who need to be reading them. So when they go to their GP, they're armed with the information that you've just given us and so on.