Academy of PHYSICAL MEDICINE

The Evidence Base

with Malcolm Kendrick

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TRANSCRIPT

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Steven Bruce
In this show, we're going to be looking at evidence, evidence particularly for the treatment of cardiovascular disease, but also we're probably going to stray a little bit more widely in the field of healthcare. Because my guest is probably one of the best-known GPs in the UK, not least because of his prolific blog, which is largely about cardiovascular disease, but also because of a number of books he's written. And I've just actually realised that two of them are on the shelf behind me and the third one is in my office. That is The Great Cholesterol Con, A Statin Nation and Doctoring Data. And that last book is specifically about the way that the data relating to healthcare issues is manipulated by various parties. I think it's his third appearance on the show. His name is Malcolm Kendrick, and he is hugely entertaining, hugely knowledgeable and very down to earth in his somewhat quixotic quest to change the world's view of dealing with cardiovascular medicine. Malcolm, good to have you with us.

Malcolm Kendrick
Nice to be on in these strange times.

Steven Bruce
This is actually I think, the fourth time you've been on the show because on one occasion we did a show up in Manchester where the venue's internet completely let us down and we just had dinner instead, didn't we?

Malcolm Kendrick
That's right. It was much better!

Steven Bruce
Nevertheless, I promised people that I would explain this thing on my slide in the email I sent them out earlier on, because it tickled me. In one of your recent blogs, you said the way that we were receiving our information about COVID and cases and all the rest of it, which we might touch on later, was rather like it was comparing apples and oranges in order to work out how many bananas we actually had, just because you said that the data was being mishandled so badly and publicized so badly. Is that still your view?

Malcolm Kendrick
I think one of my skills, I like to believe, is looking at medical evidence and working out what's actually going on. And so, when you get data you think, right, okay, I understand people play games with it, but I'm usually pretty good at saying what game they played with this and what game have they played with that and how have they managed to get this figure? When it comes to the COVID statistics around the world it's like, what? You might remember recently that they just took 5000 deaths off the death toll in England and Wales. And you think, well, how can they do that? How can 5000 people suddenly decide that they did die of COVID or they didn't die of COVI? I had been arguing for some time that with COVID, if you ever got a diagnosis of COVID, say you got diagnosed with COVID on March the first and then you died on July the 30th. Got hit run over by a bus, actually literally true. Because you had a positive test from COVID you would be considered to be a death related to COVID. All right. That wasn't exact words, the terminology keeps changing, but essentially you would be put down as a COVID related death. And, well, this is clearly utterly ridiculous, I mean, it's still the case that with COVID in England and Wales, though not Scotland or Northern Ireland, you can't recover from COVID. So, if you get COVID, you've had COVID and you've still got COVID. Because when you look at the statistics, it says number of positive cases, whatever that means, 300,000 or 350,000, whatever that figure is, and then beside it, it's got a figure of a number who recovered. And in England and Wales, there's none, nobody has recovered. If you go to other countries like Germany or the United States, they'll have something like 2 million cases, 1,700,000 recovered, 250,000 dead or whatever it is. So, you've obviously got to

question, why is it you can't recover from COVID in England and Wales, and yet somebody's decided, this is the statistics? And again, when somebody dies, I've been working on the front line, you know, I have been going in and into nursing homes and elderly patients. I've seen people dying. I'm not suggesting that COVID is a non-existent thing, it's clearly existent. But early on, of course, when you went to a nursing home and somebody became unwell, you weren't allowed to do a test. You couldn't do a test. The only test available were for patients going into hospitals. So then somebody would die and then you were basically encouraged to say, well, they died of COVID. So, did we know they died of COVID? Well, I know I've written several death certificates. I've no idea of the accuracy of any of them. So at least some of those figures, I know are nonsense, because they're mine.

Steven Bruce
One of the things that might surprise a lot of people, I'm surprised that it surprised me in retrospect, but I think you pointed out early on that actually, even in the best of times, what's written on a death certificate is not necessarily as accurate as we might like to think it is.

Malcolm Kendrick
No, I think people are rather stunned. They think there's this sort of super, they've watched to any of these detective programs or they sort of work out every single cause of death. If somebody aged 35 dies a very strenuous effort will be made to find out what they died of. When somebody's in a nursing home and over 80, in fact, over the age of 80, you are allowed to write old age on a death certificate as the primary cause of death, because they just died and you're not quite sure what they died of. So what's the point in putting heart attack when you don't know? No one's going to do a post mortem. So as people become older, and of course, COVID has mainly affected the elderly, the average age of death in the UK is 82, most of these patients have not had, I don't want to say none, I don't know, but almost none of them have had a post mortem. Almost none of them have been further investigated. So how many people did die of COVID? How many died of something else, but just happen to have positive tests with COVID, which is another issue. There were people who were in intensive care, who were then swabbed and then someone said, Oh, my goodness, they've got COVID, and then they died. Well, what did they die of? These people were already extremely ill. I'll use the example of prostate cancer, a lot of men develop prostate cancer, it's quite slow growing, the vast majority of men who have got prostate cancer die with it, not of it. If that makes sense? And it's another question with COVID, did somebody die of COVID or did they die with it? Did it have some effect on their overall health? I mean the Centre for Disease Prevention and Control in the United States estimated only 6% of people who have died of COVID in the United States died purely from COVID. In other words, all the other people had significant comorbidities. So again, what does that figure tell us? Well, it is very difficult to know because it really depends on what the doctors themselves are deciding to put on and what they're told to put on as well. It's more in the states, people are more told to put COVID on that death certificate, because there are financial reasons for doing that in the states that don't exist in the UK. If in America a patient was just sent into the hospital, you get whatever it was, $7,000. If they were diagnosed with COVID, you get $14,000. If they were put on a ventilator, you got $36,000. So, there's an enormous pressure within the hospital system when the other patients aren't coming in and you're not doing the other elective work to make as much money as you can. So, there's a tremendous pressure to get doctors to state that these patients had COVID and needed to be on a ventilator because they had COVID.

Steven Bruce
So, given that those financial pressures don't exist in the UK, why did you think it is that we are perhaps over estimating the number of cases?

Malcolm Kendrick

I think there's many, many reasons. I'll tell you about, there's a condition called sepsis that you may or may not have heard of. If you go back to when I started in medicine, nobody had ever really heard of it. In the last few years there's been a tremendous drive to make people more aware of sepsis, to make people diagnose it earlier and so on. And the number of deaths, if you look at it, has trebled from sepsis over a five-year period on death certificates. Now, there's no way that three times as many people are dying of sepsis as it did before, but people become aware of it, once they're aware of it, that becomes the primary diagnosis. That's what's put on the death certificate. And so, when something becomes the overwhelming thing, if you like, then everyone starts to look at it and focus in on it, this becomes this confirmation bias and all sorts of other biases that were horribly capable of falling into. And if nothing else, I mean, instead of putting old age you put COVID. So, there's an artificially increased COVID death rate. But there's also an artificially decreased COVID death rate because some doctors didn't put it on, because they didn't do a swab and they didn't know it was COVID. So, you're looking at the statistics, it's like, I can't really make head or tail of what I'm looking at here because nothing is making sense. I mean even a concept, which I've written quite widely about, with infectious diseases there's a thing called, well there used to be a thing, apparently, they're no longer is, called the case fatality rate. Which used to mean people who had symptoms, who are unwell enough, usually to go into hospital. So, these were clearly quite unwell people. So, at the start of the epidemic in the United Kingdom, right at the very start, because not many people were being swabbed and only the illest were being swabbed. Well, the case fatality rate was in the order of 20%. So, 20% of people who are diagnosed with COVID died going into hospital or just overall. Well, clearly, as we've done more and more and more and more testing, we're testing people who are less and less and less and less unwell. So, what's happening is of course, the case fatality rate is falling and falling and falling and falling and falling and will continue to fall. Throughout all pandemics throughout history the rates of fatality fall. So, what we're seeing is a kind of combination of all sorts of factors making it virtually impossible to work out what's going on. I mean, just in this thing of the cases we're seeing at the moment, we're told there's now, on average 3000 new COVID cases in the United Kingdom every day. Well, of course, you're gonna say, the vast majority of these people have no symptoms at all, they're not even aware that they've got COVID. So clearly, they're not gonna die of it. But are they a case of COVID? Is this a case of COVID? The definitions have become meaningless almost. And people will say to me, Well, that's not a case. Well, that is a case because there's a positive swab. Well, that's not how people used to define these things, it used to be very clear, there was a case fatality rate and an infection fatality rate. Because a lot of people get infected, they don't even know they're infected. There aren't any symptoms and, in the past, we would never have been swabbing these people, we would never have noticed this level of disease in the community. So, if in an influenza epidemic, if we swabbed 200,000 people a day, every day for a month, we'd find influenza probably kicking around in a lot of people, just there.

Steven Bruce
What you say, Malcolm, seems so blindingly obvious that it's kind of disturbing that you don't see any mention of it coming out of the scientific advisory group.

Malcolm Kendrick
No, you see nothing of this at all. I think people like to be right and they get a narrative. And if they start saying, you know what, we have no idea what these tests mean anymore. Then they lose authority. So, they've kind of got stuck, in my opinion. They're stuck on a kind of a big dipper of doom, they're stuck and they can't get off it. Now they're on it, that's it. And if any individual was to stand up and go, you know what, this isn't right any longer. They would no longer be on stage. They would be, bye! You're not telling the story that is the one that we're trying to get. You know, if we said, well, a case is not is not a serious thing, a positive swap might just be picking up viral material in somebody's nose. And that's being shown in fact a study in China was looking at people who had had COVID and then gone negative, and then they did swabs on them. I think something like 10% of those people have now got a

positive swab. Because the test that we used is called polymerase chain reaction, invented by Kary Mullis, about 30 years ago. Essentially what it is it gets particles of DNA and bits of the virus. It's not multiplying up an entire virus particle. It's bits of the virus. So, you stick a swab in, you pick up some bits of a Coronavirus. You then put them in a device that multiplies by billions of times. Billions and billions of multiplications until you've got enough that you can say ah looks like some Coronavirus in there. What is it? Is it infective? Is it dead bits of the Coronavirus? Is it some other Coronavirus that looks very much like COVID-19? Because there's many, many different strains of Coronavirus, most of which cause the common cold. So as children are going back to school and potentially picking up all sorts of Coronaviruses and spreading them around, are these also being misinterpreted?

Steven Bruce
Dare I say also, of course, that the press love scary numbers, don't they? So, they don't want to hear that the statistics indicate things are not as bad as they like to paint it.

Malcolm Kendrick
Well, yes, of course. You know, it's a story. It's a frightening story. Everyone likes to see the story about the great white shark eating people off the Australian coast, nobody wants to know that more people die being killed by bee stings or something. You know, it's some things that grab the attention and some things that don't. And this is one of the ones and of course the press themselves are kind of trapped in it as well, they've got their death counter that clicks up 20 a day or whatever it is, oh my god we're now at 41,923 or whatever it is. It's just, you should really just stop doing this, what you should look at instead is say, okay, the metric or the outcome that I was interested in from the start was a thing called the overall mortality rate. And the overall mortality rate is measured, there's a group called EuroMOMO. I can never remember what the MOMO stands for.

Steven Bruce
It's the diagnosis that's hard to get wrong, isn't it? I seem to remember-

Malcolm Kendrick
Well being dead or alive. Although with COVID apparently you can get it wrong. No, if you're dead, you're dead. If you're alive, your alive. And it's very hard to go, Oh, I thought they were dead but actually, they're alive. It's probably been done, probably done it myself. In fact, I went to a nursing home one time to verify two deaths and they were both lying in the beds almost side by side, this is going back a few years. And I went to one said, yup, they're dead. Then I went to the next one and said, no, they're not yet dead, bit early on that one, I think. So, leaving that aside, yes, that's the accurate statistic. So, there are groups that look at overall mortality that's going on, and they monitor this, part of the MO is monitoring, can't remember what the MO stands for. And what they look at is they average the previous five years and they have got a line. So, in the winter more people die, in the summer less people die. This is always the case.

Steven Bruce
Isn't there a problem with that too, though, because the overall mortality is going to be affected by the number of people who weren't treated for things that killed them because of COVID.

Malcolm Kendrick
Well of course. You're slightly jumping ahead, I think, there. I'm just trying to explain the overall mortality in the UK every year about 670,000 people die, something like that, so around about 1700 a day. And that figure is the figure. So, what happens is if that figure jumps up more than what they call five standard deviations from the norm, that raises

an alarm. It says something is going on. We don't know what it is, but we know something is going on. This is normally for influenza and things like that. So you watch the graph go like that, and then it goes like that, and it goes back down like that and you think okay, so there was an increase in overall mortality. Interestingly across Europe, and not all of Europe is in this system, but the majority of Europe is, some countries showed a serious spike in overall mortality. Spain, Italy, Britain- although it separates us into England, Scotland, Northern Ireland and Wales- Belgium, the Netherlands, maybe, but anyway there were several countries where nothing happened. There was no change in overall mortality. Germany was one, I think, I don't remember. Definitely Ireland. Nothing showed at all in Ireland. So, it's interesting that although the COVID arrives in all of Europe approximately at the same time, and most countries showed a rise, no, actually 50% of countries showed a rise and a fall, 50% of countries didn't show anything at all. You have to say, so what did they do? Well, obviously, that's a debate. I don't know what they did, again, we look at the figures and what the hell do they mean?

Steven Bruce
Malcolm I ought to turn to our audience fairly shortly, because we've had some questions come in. Jan has said that weren't the asymptomatic positive COVID cases capable of passing it on to more vulnerable people? Isn't that the risk and the difference?

Malcolm Kendrick
Well, yeah, I mean, we don't know for sure. But it does look like asymptomatic people, the case has always been with many infectious diseases, what they call a prodromal phase, i.e. you've got it in your system, it's multiplying, you can start to shed it, then you get symptoms. That's sort of the case with influenza, although it seems that the time lag between the virus arriving and you getting symptoms is about two days, whereas with COVID it's a bit longer. So yes, asymptomatic people can potentially spread it to other people. Absolutely. That as a problem, it depends what you want to do about that, I suppose. But yes, asymptomatic people can get it. My own ideas, possibly people have read my blog will know that, I do believe that we should have a period of shielding the vulnerable people for about a month and letting it rip throughout the rest of the population. If there is any more rip to be had. I mean, there's a certain amount of information that says this is over effectively. And any deaths that we're seeing in hospitals are the kind of deaths that I was talking about, which is somebody elderly is a bit unwell and they have a swab, they're found to have COVID and end up in hospital. They then die. That will be a COVID related death. Had we not done the swab, would we have even known they had COVID? And was it the thing that killed them? So, there's almost always going to be this trickle level of deaths. Just if you like due to testing. Personally, I think it's gone effectively from Britain. I think it's gone from most countries in Europe, it's gone from all countries in Europe. In France, they've got more cases currently than they had in March, when they were in the middle of their high levels of death. So in March they were getting, I can't remember the exact figures, let's say there's a 14 day lag, I was looking at one day where they had the highest day of infections in March, late March, it was, I think, 5000. And 14 days later they had 1400 deaths. All right, we've now got 10,000 and at no point has the death rate, total number of deaths, gone above 30 in one day. You're getting twice as many positive cases and whatever 30 into 1400 is.

Steven Bruce
You can understand why this is quite difficult for the layperson and even actually professional medics to understand, isn't it? One of the reasons I love getting you on the show and why I've worked so hard to get you back on again, is because not only are you an avid reader of all the evidence, but you're capable of looking at this detailed stuff and

turning it into a language which is both entertaining and informative and is understandable by the layperson, because most of us can't understand the statistical analysis that goes on in research.

Malcolm Kendrick
Well, yeah, I mean, I'm a geek.

Steven Bruce
Thank goodness, we've got people like you who are because every time I speak to anybody I recommend your blog and I recommend your books because, although I suspect they're intended for the layperson, I'll bet there's a huge number of professional medics who read them because it makes the stuff understandable. Pip has asked a question. Pip says, Is there not an argument be made that if people were COVID positive, the effect that it had on their body could have made their other comorbidities worse than they would have been. Had they not been COVID positive?

Malcolm Kendrick
Well, yes, of course. I think the fact that the majority of people who've died, the vast majority of people have died, have been elderly and have had comorbidities. Which means that you are going to be more vulnerable to this virus if it gets you. There's no doubt about that. I mean, the correlation with type two diabetes is very, very strong, there's correlation with other heart disease issues. In fact, I was going to do an experiment which - are you not hearing me? Sorry.

Steven Bruce
I was talking to my technician.

Malcolm Kendrick
I was trying to do a little calculation on, because there's a thing that if you go to your GP, they may or may not do called a QRISK score, which is quantifiable risk for heart disease. So, they ask you things like, obviously your age, your sex, whether you smoke, whether you're diabetic, what your blood pressure is, and then add it all together on this little scoring thing. And they say your risk of having a cardiovascular event in the next 10 years is some percent, 10%, 20%, 30%, whatever percent. But if you actually get the QRISK score, and you use it for COVID patients, there's an almost perfect correlation. I haven't done it exactly yet, but I've been looking at it. If you've got diabetes, your risk of cardiovascular disease is doubled. Well, your risk of dying of COVID is doubled. If you've got high blood pressure, your risk of dying is 50% higher. Well, if you've got COVID 50% higher. These are not the actual figures, these are just examples. If you are from a black, Asian, ethnic minority group, your risk is up by about double. Well COVID doubles your risk. So, it's quite extraordinary actually that the QRISK score, and the COVID sort of symptom score of people who are likely to become seriously ill or die, it's almost a perfect match. Which means that COVID is, to a certain extent, although it's a respiratory virus and it gets in your lungs, the majority of damage and the majority of people who die from this are people who've got a high cardiovascular risk. And it is in fact, causing a lot of doctors to think well what on earth is going on here? How can a virus infect you through your lungs and cause you to die of a heart attack?

Steven Bruce
I guess we're in slightly awkward, if not dangerous, territory here. Pip's followed up on that by saying that she's actually getting quite a bit upset by this because her 87-year-old mother was admitted to hospital for suspected stroke, turned out to be Alzheimer's, but they picked up COVID in hospital. One week later, she developed bacterial

pneumonia and passed away a few weeks later. And she's got no doubt in her mind, had she not picked up the COVID, she would have been discharged or would have been back in the care center.

Malcolm Kendrick
Yeah, that's quite likely true, yes. She's absolutely right. And these are the tragic cases. These are the people that I was seeing, in my own practice I'm quite often dealing with a high percentage of elderly patients. But these are the people who are really being hit by it. And I worked in a nursing home where 18 patients died, some of them very quickly. In fact, I've personally been involved in the care of 36 patients who died of COVID. So, I see it firsthand, and I see the tragedy of what it can cause. That's why to an extent, that's why we need to protect these people, if we can, at all costs. Whereas once people are younger and fitter and healthier, for the vast majority, this this is not a significant condition. It's less far less serious than flu. If you remember the Spanish flu epidemic of 1918-1919 the average age of death in that epidemic was 28 years old. The average age of death in this epidemic is 82 years old. So, it's gigantically biased towards the elderly population, especially the elderly vulnerable population. So, I think it is something we need to be aware of. And yes, I've got a 92-year-old mother, I don't want to be giving her COVID. That's the last thing I want to do, although she's of the opinion that she's much more gung ho than perhaps some people and said, "I'm 92, I've lived long enough. If I get it, I get it If I die, die. I don't care."

Steven Bruce
And my elderly father is exactly the same, I think. But again, it's the people who are left behind who often have to suffer the consequences in many ways, isn't it? Matthew has asked whether the interpretation of viral fragments in nasal swabs as an actual positive result is the main cause of false positive findings, or are there more significant reasons for false positives inherent in the test itself?

Malcolm Kendrick
Well there are inherent in any test, no test is 100% accurate. There's test code sensitivity and specificity. I always get them mixed and have to go and look in a book and remember which way they are. But sensitivity means false negatives, so you get a swab and it says you don't have COVID but you do. Now what is the rate of that? Well, I've seen figures suggesting if the swab technique is not very good, and in some studies, up to a third of tests can be wrong. So, you can be told you don't have COVID and you do. Which is of course a problem in and of itself because those people then go off and charge about, maybe infecting their elderly relatives. The other one is there's a false positive rate, which is you don't have the disease but you're told that you do. Now that that's inherent in any test, virtually. There's no test that's 100%, I don't think. Most cancer testing for example, the specificity can be down at 70%. Because it's considered a much, much worse thing to miss a case than to say to somebody, you've got it when you don't. But of course, saying to somebody, you got it when you don't carry a number of implications, but the false positivity rate is, in part, we don't know how many of these swabs that are showing viral particles really mean somebody who's got an infection that can then give it to anyone else, who is really infected. So, I sometimes liken viral infection to the storming of a castle, if you like, there's a moat and then there's the castle walls, and even if you get in the castle walls there's the keep in the middle and everyone will get into this keep in the middle. So, the virus can be present in your nose, does that mean it gets into your body? You're probably aware of MRSA, methicillin-resistant Staphylococcus aureus, which is the thing that can affect elderly people who get operations so they need to make sure they haven't got it. But you can test quite a lot of healthcare professionals and find that they've got MRSA in their nose, it's there. Are they a case of MRSA? No. Do they suffer any symptoms? No. Will they suffer any symptoms? No. So are they a case of MRSA or are they an asymptomatic carrier? And can they pass it on? You know, I think one thing we've realised is the terminology is imprecise for infectious diseases. To say that you're infected I would say, Well, what does infect mean? Because I was working with two nurses, as close as you can be in a small room, doing

some stuff and sorting out stuff for patients, both of them are unwell, both were coughing, nobody had masks on. I was in a room with them. They'd had a test the day before, as it turns out, both tests were positive, and they were moderately unwell for a week or so afterwards. So clearly, I was exposed at a high level, very high level, to the virus at that point, possibly before that. I have never had a symptom. I've had a swab, it was negative. I've had an antibody test, it was negative. What does that mean? Does that mean I've never been infected? Well then what does infected mean? We're running into problems here in that no one's ever done this level of testing. No one's ever done this before ever. So, we're kind of saying, Oh, look at all these things that are happening that are completely new, and you say, Well, we never knew if this was the case before because we've never done this before. So what happened in 1918, that had a case fatality rate of 3% that killed 50 million people around the world. That was the flu. COVID has currently killed about a million people around the world and the current population of the world is four times what it was then. So, the actual death rate of the flu in 1918 is in the order of 300 times what it is currently. So, when people say, what would have happened had we been swabbing people and testing people and what we have seen, we've no idea. And yet we see this now and we say, Oh, this means that. Well, we don't know this means that. And the other thing that can happen just with false positives, there's a guy who works as a senior lab technician I'm in communication with who says that if you do the 360,000 swabs that have been done, or whatever it is, in the last month, if you have a false positive rate of 0.05% you're still going to get 15,000 false positive results. I.e. we will get the majority of people who are told that they have it. It's not just finding particles, it's just the test was done wrong and the results come out wrong because it's over sensitive, if you like. You start multiplying out viral particles again and again and again, 40 cycles that this runs through, it's very likely that you're going to be multiplying up stuff that doesn't count for anything or it's from somewhere else. Or it's been introduced into the test by mistakes or contamination. Once you're at this level of multiplication, I hate to quote Donald Trump, but the more you test, the worse the disease gets. This is true. What you need to be looking at is how many people are going to hospital, how many people are dying? What's happened to that?

Steven Bruce
I suppose the hard part of this, particularly for someone like Pip whose mother has died and for many others who have suffered for one reason or another through this, is that if COVID was responsible for that, that feels really significant. But overall, globally, it isn't having a greater effect perhaps than a severe strain of the flu might have had in its place and yet we are taking much more severe preventative protective measures because of it.

Malcolm Kendrick
Well, absolutely. If they thought at the start, and I believe they got the figures wrong, but time will tell. However, this is not just a one-way street. As I say to people, this is not a zero-sum game we are playing at the moment. We are not playing a zero-sum game with this. Every person who, through lockdown, we prevent dying from COVID, however many that may be, we know for example, the statistics from the Office of National Statistics say, that up to now 16,000 people have been killed as a result of the actions taken. Lockdown, in other words. Because people do not turn up to the hospital with potential heart attacks, we know that rates gone down hugely. We know they estimate 6000 people have died of heart attacks that would have normally been treated. We know that cancer patients have not been treated, I watched two patients turn their faces to the wall and die after their cancer treatment was just stopped. The Lancet had an article paper showing that at least 70,000, what they call, life years are now going to be lost through delayed diagnosis and delayed treatment of cancer. It's estimated that the economic ramifications of COVID will in the next year kill 17 and a half thousand people and that will be likely to continue for the next four or five years. So, you can't just switch off the health service which they effectively did for several months, stop people being treated for other things and expect this is going to have no effect on health.

Steven Bruce
We've had a couple of orthopedic consultants on the show over the last few months and they've said that not only did they not treat people for those several months, but they can't suddenly treat them all now because they've now got a huge waiting list. And like the cancer patients, they'll all still be waiting a very long time possibly before they can be seen because there's so many left.

Malcolm Kendrick
Yes. I think that because the statistics are all over the place, that you can make a very strong case, and I have made a very strong case, that lockdown has probably been responsible for killing more people than COVID has and will continue to do this for some time because of the longer-term effects of lockdown. So, it's not just health on one side or people dying on one side versus inconvenience or not being able to meet up with your friends or whatever on the other side. This is not how it works. Most tragically, in my opinion, is that we've already seen a rise in cases of domestic abuse deaths and children being killed by their parents in their own houses because you can imagine the impact of lockdown on an abusive parent or partner has been dreadful. People pointed this out, this is now going on. So I know people like to see it as just one thing: it's COVID and we must protect everyone from COVID. That's fine, maybe that's fine. But it's not fine if by doing that you are ignoring 95% of what a health service should be doing on the other side. It's not right. It's not happening. If we were to be getting death rates in the hundreds of thousands, well, that's a different matter. We've had 42,000 deaths, 50% of them, the excess deaths, were in elderly care homes. So, at the start of this pandemic, and I got very upset by my own area, they were discharging elderly patients into care homes who they knew had COVID. They were seeding COVID into care homes. They said, Oh, we've got to get the hospitals cleared out. We must get everyone out of hospital. The government's already apologised in Sweden because, although they didn't do lockdown like we did and they've had less deaths proportionately by a considerable margin, they admit that at least half of the deaths occurred in care homes and they could have been prevented. And so, we utterly destroyed the health of lots and lots of elderly people through the actions of lockdown. So, yes, if you could exactly pick out the people who are likely to die, you can do this reasonably accurately and then we can shield these people or ask them if they wish to be shielded because I think it is a decision that people should be allowed to make. The total number of children have died of this under the age of 20, in the UK, since the start of lockdown is 14 and all but one had serious underlying medical conditions. So effectively we've had one death in a population of 20 million, which is unheard of. I mean, that's just nothing. And even when you move up to the next age group 20 to 40, the total number of deaths is far less than die normally from things like road traffic accidents and suicides and poisonings and things like this.

Steven Bruce
Malcolm, we've done pretty well, considering we weren't going to talk about COVID because we're now half an hour in. There's one more question, Elspeth's had a question on my board for quite some time. She says, what percentage is it of the alleged 500,000 that die per year from all sorts of causes, 500,000 people die per year anyway, is there any overlap and what percentage is that? Looking at the figures you'd think that only just under 500,000 people have died since March? I'm not quite sure I follow the question to be honest.

Malcolm Kendrick
Well, I think she's just sort of saying, this is this excess mortality, over and above mortality, so 670,000 people die every year in the UK. The excess deaths in the period from March, well actually it went between March, April and early May as a spike, and then the death rate fell back to essentially normal. But during that period, I think it was 65 additional unexpected deaths or above what you normally expect to see at this time of year. The number is now

obviously levelled off and coming down. I don't know if that answers that question but that's what the statistics tell us.

Steven Bruce
Somebody has shoved a whole lot more questions in and one of them I do want to ask, maybe we'll have time to come back to the others. Sam has asked a question about PPE and this is bound to be emotive because of course we're all governed by Public Health England's regulations, their guidelines, but Sam's asked whether you think we should be behaving differently with regard to PPE, should we should we be doing all this cleaning and fogging and wearing PPE and stuff?

Malcolm Kendrick
That's a very large question. When it first hit a lot of health staff in the care homes were told not to wear a mask and not to wear PPE, because there was very little of it around. I think if you're gonna wear it, you have to make sure that it is actually going to do the job. I think these surgical masks, they were never designed to prevent infections spreading from one person to another. Surgical masks were designed to stop blood splattering into surgeons' faces and to stop the surgeon dribbling into the patient's wounds. That's what they are there for, they did not ever expect that they would ever prevent the transmission of infectious disease. And you will notice that early on the WHO and all the authorities said masks are not of any use and that's because any research that had been done had not been able to demonstrate any benefit from wearing a mask on the transmission of things like influenza, because obviously COVID hadn't happened before. There's been no studies or evidence since then done on account of there being just too many variables. Someone said to me, Well, expecting one of these ones that loop around your ears masks to prevent COVID from spreading is like expecting you're going to be able to pick up sand on a tennis racket. When they talk about viral particle sizes, the viral particle size is far, far smaller than any of the mesh on any mask that you wear. If you wear what they call an N95, that particulate matter, it's 95% of particulate matter is stopped. When you move on to the FFP3 masks, they've got a valve because they're tight around, because they have to be tight, so the air has to be able to blow out. So, it stops it from coming into you but it doesn't stop you blowing it out. So even the most protective masks, actually the most protective masks are the most dangerous, if you've got it you will be spreading it. And there's other risks, if it is spread through aerosol, we used to have this ridiculous thing of aerosol generating procedures in the hospital. I said, breathing is an aerosol generating procedure. This idea that the particles, yes, if you, and I've always told my children, if you're about to cough or sneeze, do this or sneeze into your elbow. So, then it's not on your hand, you prevent it from being thrown out, if you like, and then it's dealt with. It may still be sitting there but so long as you're careful about there. But with the masks, you've seen people throwing them off their faces. There's also clear evidence that this can be spread fecally and people who've been to the toilet and wipe themselves and then put a mask on, will be sticking viral particles all over the front of their mask. They will then be touching it, they will then be touching their face. I say if you're going to say that everyone should do something, and the whole world should do it, then you have to have very strong evidence that it works and there is no such strong evidence that mask wearing works. If, however, it helps everyone get back to living their normal lives and makes people feel safer, fine, but I would also be careful just because you feel safe, doesn't mean it's actually doing anything at all.

Steven Bruce
Can I assume that in your practice you personally are having to adhere to the PHE guidelines, whatever is appropriate to the procedures you're doing?

Malcolm Kendrick
Yes.

Steven Bruce
But anyway, you talked there about getting evidence for what we're doing. Should we move on to your favourite topic? Should we talk a bit about cardiovascular disease? You very kindly sent me half of your soon to be published book, which is great because it means I only have to buy half of it when it does get published.

Malcolm Kendrick
No, no, you have to buy several copies.

Steven Bruce
What's the title of the book? Do you know yet?

Malcolm Kendrick
No. I've thought of many ideas but I think it's going to be something on the terms of, you know, I was going to title it What the LDL?

Steven Bruce
That might need explaining.

Malcolm Kendrick
People wouldn't get that. Okay. Okay, if it's not cholesterol, what is it? I suppose? Well, I've been working in this area for years and years and with all sorts of other people who've been trying to convince the world-

Steven Bruce
When we spoke a several months ago, possibly a year ago now, I got the impression that you were gradually, you and others in the anti-cholesterol hypothesis movement, if I can call it that, were being taken more seriously. Is that the case? Or are people getting fed up with Malcolm Kendrick banging his gong?

Malcolm Kendrick
A bit of both probably. Of course, I've got an awful lot of confirmation bias going on so probably I'm the worst person to ask. And I think that there have been more questions. Interestingly, there's a new set of drugs have come along fairly recently called PCSK9 inhibitors. Statins lower your cholesterol by say 30% or whatever, your LDL by 30% but never mind, we'll not get into that discussion or we'll be here forever. They lower your cholesterol by 30% and they do reduce the risk of heart disease by a very small amount. I've always believed that impact was due to the other effects that statins have in your body, and I stick to that but that's another discussion. But with these new ones that have come along, they're injectable, they're actually a monoclonal antibody. They, what they do is they trigger a thing, normally cholesterol gets into your cell via an LDL receptor that links onto the LDL, pulls it into the cell and then both the receptor and the LDL are got rid of. What this does is it stops the disintegration of the receptor. So, it pulls the LDL in and then the receptor then pops out and pulls another one in, pops out, pulls another one in. So, each receptor is working however many times as hard. So basically, what they're doing is pulling LDL out of your blood stream out. I said, Well, if cholesterol is so bad for you, how come it is a great idea to have it pulled out of the bloodstream and into the cells? Surely that should be doing harm. Anyway, that's another discussion. But they lower your cholesterol by 40%. So, if you take a statin it's 30%, these will lower it by another 30-40%, or whatever, I'm just using these figures roughly, but they're approximately correct. So, the idea was, well, this is fantastic, because we'll see

huge effects, because we'll get the cholesterol right down with these things. But the analysis is that they can see no correlation. They can see no effect on a) overall mortality from these drugs or b) cardiovascular mortality.

Steven Bruce
This is slightly odd, isn't it? Because we know, don't we, that plaques in the arteries are caused by cholesterol.

Malcolm Kendrick
Well, everybody knows that. Yeah. Like everybody knows that masks work as well.

Steven Bruce
And that cholesterol is produced by trapped by saturated fat, isn't it?

Malcolm Kendrick
Well, yeah, well, of course. Of course, it is not.

Steven Bruce
I should just explain to people they should read your book about the cholesterol con and your other books, because in there you've got a lovely diagram which shows, I think it's a saturated fat molecule and a cholesterol molecule. And you point out that this one cannot, under any circumstances, become that one. Even with the complicated wizardry of our body.

Malcolm Kendrick
Well, it's 99.9% true that statement, like everything. But the fact is, when you eat fat, your fat is absorbed directly from your guts, it never goes into your liver and it travels up through a thing called the thoracic duct. It goes into your bloodstream and it is absorbed directly into fat cells and other cells. It travels through what's called a chylomicron, which is like a big, wobbly lipoprotein. So your fat basically gets stripped out of that and that ends up at the liver, where the fragment is taken out of the bloodstream. So, fat that you eat doesn't go through the liver, right? It just doesn't. And of course, the liver's where cholesterol is produced, right? So where does the cholesterol come from? It's produced by the liver, the liver makes it, right? Makes five times as much as you'll ever eat. But when people say, if you eat fat, it will raise your cholesterol level. Well, two things don't ever meet, they never meet, and they only ever meet inside a lipoprotein. So, when you start picking these things apart, and saying, how does this lead to this and how does this cause this? There's no explanation. It's just it was decided that this was the case and then they reverse engineered the hypothesis to fit. But it's never made any sense.

Steven Bruce
I was just going to say, and moving slightly ahead of that, in the draft of the book that you sent me, and I haven't had time to read it as I explained earlier on, not fully, you kind of blow apart the assumption that the plaques in our arteries are made of cholesterol. Because I think in fact many people talking about arterial vascular disease, will talk about cholesterol plaques in the arteries.

Malcolm Kendrick
Yes, they talk about them being almost like a big lump of cholesterol in your artery. Which is absolute nonsense, it's just complete nonsense. I mean, it is. So, when you actually look at these things and examine them, I did a review of many studies, and you'll find what they say is within a plaque there's all sorts of things, sometimes a bit of cholesterol. Actually what is fascinating is that the cholesterol, such cholesterol as you can find in plaques and you can actually

find cholesterol crystals, you can actually find sharp pointy crystals of cholesterol in the plaque, and you go right, the cholesterol that is carried around in the lipoprotein that we call cholesterol, which is LDL, is actually a thing called a cholesterol ester. It's a cholesterol molecule attached to a fatty acid molecule. And when you attach these two things together, they become what is known as an ester. So, it's not pure cholesterol, it's a cholesterol ester. You can't make cholesterol crystals from cholesterol ester, because chemistry doesn't work like that. You can only make crystals from the pure substance. Right? Then where does the cholesterol come from? Whereby you've got these cholesterol crystals that have been found in plaques. Well, the only place you can get it from, took me a while to find this out, is actually the membranes of red blood cells, which have the highest level of cholesterol, free cholesterol, in them of any tissue in the body. And in fact, they have demonstrated that the cholesterol crystals, certain researchers that have bothered to look, have said the cholesterol crystals come from red blood cell membranes, because they can't come from cholesterol esters. One of the first things that was ever seen in a plaque was actually, going back 170 years, by the Austrians, was cholesterol crystals because you can see them. It's one of the few things they knew what it was, I'm still quite impressed they knew what cholesterol was in 1840 or whenever it was, it was quite clever. But anyway, I think they knew what it was because it used to be in gallstones, gallstones are quite often made of pretty high concentrations of cholesterol, some of them are virtually pure cholesterol. So, I think they kind of said, that's the same as that, so that must be what that is. So, when you start looking at that and say, well, they you are there's a cholesterol crystal and then you say fine. That doesn't come from the cholesterol in your blood it can't come from it, it's the wrong substance.

Malcolm Kendrick

How many doctors know this? One, that's me. Maybe it's two now if another doctor's watching. And then they say that the lipoprotein LDL is the lipoprotein found inside the plaque. And I go, actually, no it's not and people go, what do you mean? So, can I do this in 30 seconds? LDL, low density lipoprotein, is what we call bad cholesterol. It's not cholesterol, it's a lipoprotein, it's like a taxi carrying cholesterol esters around the body in it. And it has an evil twin brother called Lp(a) which is lipoprotein(a), which some people may have heard of, most people have not. Lp(a) is the same as LDL, it's got an extra protein wrapped around it called an apolipoprotein(a). Now if you're looking in a plaque and you find what looks like an LDL particle, you can say that's an LDL particle. Well, yes, how do you know it's an LDL particle? And how do you know, it's not one of these? Because they are exactly the same, apart from these extra proteins. So how would you identify if it was one of these or not one of these? Well, you look specifically for the protein. And when they look specifically for the protein, they find it. And they find it alongside what they call LDL particles. Well it's not LDL particles. The only way that you can find that is because this is Lp(a) and not LDL. So again, it's misidentification. A couple of researchers have gone quite long way down this Lp(a). It's now becoming a bit more of an issue that people with high Lp(a) levels are at greatly increased risk of dying of heart disease, right? No one's ever measured this or monitors it or even bothers about it, but it's quite a significant thing, right? And when you look inside plaques, and you look for what they call lipoproteins or remnants of lipoproteins, a bit like particles of COVID, and you say, look, we find an LDL. No, you haven't. You found an Lp(a). So even there, they're wrong. Right? So, once you start teasing it apart and saying, Well, how on earth did you think any of these things? This is the ultimate confirmation bias, you know, you eat cholesterol, cholesterol in your blood goes up, it's absorbed into your artery walls and thickens them and clogs them up. It's a beautifully simple story. It couldn't be simpler, as I say any five-year-old can understand it. And unfortunately, science is not really for five-year-old to understand because it's very often counterintuitive. The simple answer, I will say to people, beware the simple answer, beware what Daniel Kahneman would call fast thinking. Fast thinking is a disaster. Fast thinking has got us to where we are with COVID. Fast thinking takes us to the very simple solution, the very simple answer, and then we cling to it and then, have you ever seen the film Inception? Great film, in my opinion. The strapline, in a way, is the most resilient parasite is an idea. Once you've got an idea in someone's head, cholesterol, cholesterol, cholesterol, cholesterol, it's like, that's it, you

know? And this is one of the problems that you have is because well, what else is it? It's one of the reasons I wrote the book, I know you all think its cholesterol and nothing has really taken the place of cholesterol to explain what is a complex disease. Other people have come up with different ideas, but frankly, have not really been coherent. So, what I've attempted to do is to say it isn't cholesterol, right? Just accept that for now. In Chinese philosophy they have a thing called Po, which is try to come to this discussion without holding a view for certain. Try to come to this discussion with ideas that may or may not be correct but do not come to this discussion already knowing the answer or believing you already know the answer.

Steven Bruce
A lot of people could benefit from listening to that advice. Am I right in thinking that part of your hypothesis is that there is an association between heart disease and cholesterol, but it's not necessarily the cholesterol level which is causing the heart disease, it might be the other way around? It might be a marker rather than a cause.

Malcolm Kendrick
I don't want to go down that rabbit hole with people too far. There is an association, statins lower cholesterol and they reduce the risk of heart disease by a small amount. So how do they do that? If it's not through lowering your cholesterol level? Well, the answer is, almost certainly, what they also do is they stimulate a little hormonal compound in your blood called nitric oxide, which some people may or may not have heard of. If you take Viagra, that is what has happened is that Viagra increases nitric oxide synthesis in the penis and this causes an erection. It was originally developed as an angina drug. But while they were doing it clinical trials, the volunteers didn't hand their tablets back, which is very unusual. And then they went and said, why aren't you handing your tablets back? And eventually discovered this was what happening. You will have patients that take glyceryl trinitrate tablets or GTN sprays or anything that says nitrate on it. What nitrates do is they stimulate nitric oxide synthesis within the arteries, within the endothelial cells that line the arteries, and this causes the arteries to dilate so it improves blood flow. And it also has many effects: it's anticoagulant, increases synthesis of protective cells in the bone marrow, etc. It is the number one key most important chemical for cardiovascular health that there is. And the other thing that's very for increasing nitric oxide is going in the sun. Because if you go in the sun nitrates are released from your skin, produce high levels of nitric oxide, it lowers your blood pressure, opens up your arteries, protects your whole cardiovascular system. And that's why that works. So, what I've been trying to do is to say, how does that work? How does that fit? How does that fit? How does that fit? How does it all fit together? So how do these things all come together? Essentially, what I've said is you don't need to look for causes because you'll spend your whole life doing that and getting incredibly confused. And if you look for causes, you will always find yourself going down one route and not seeing the wider picture. So essentially, what I'm trying to say to people is the atherosclerosis, the buildup of thickenings and narrowings in your arteries and then the final blood clot that forms, is a process that's going on all the time in your system, right? And you've got to understand that process before you can understand why something might cause heart disease. So I've asked the question, how can smoking, raised blood pressure and being Asian cause the same disease? If you're going to explain cardiovascular disease, you have to be able to say that there is a process into which they can all be plugged to say, Oh, I understand. So, if you smoke, what happens is small particles get into your lungs, they get into the bloodstream. These nanoparticles attack the endothelial cells, which are the lining of the arteries, they damage them. They damage them such that if you smoke one cigarette, you can actually measure particles of dead endothelial cells floating around in the bloodstream. One cigarette, one volunteer, once. What's happening now is you get damage to the artery and a blood clot forms, small blood clot that gets incorporated into the artery wall. So, we move on, how does being, this is a complicated one, how does being of ethnic Asian origin in the United Kingdom, increase your risk of dying of heart disease? Well, that's a much more complicated set of pathways, but it ends up in the same place. The people from an ethnic Asian origin, especially Muslims, more than Hindus and Sikhs, much more, are under

extreme social stress. They become stressed, you can measure this stress. When the stress goes up, the cortisol level goes up, cortisol damages the arterial wall, raises the blood pressure and lowers nitric oxide levels.

Malcolm Kendrick
So, once you start looking at the process model, you can say, Okay, so how does that cause it? How does diabetes cause heart disease? Doesn't necessarily raise your blood pressure, doesn't raise your cholesterol level, but it definitely increases the risk of heart disease. So, what's happening now? Most people don't know, again, this is something no doctor knows. If you try and pick up a fish, it slips straight through your hand, or can do. Unless it's a shark because they're sort of different. But most fish have a lining on them called a glycocalyx, which is sugar protein layer, a bit like a miniature forest. If you look at it under a super microscope it's like a lawn. And that sits on fish and when you try and pick them up, they slip. That same lawn is present on all your arteries and veins and blood vessels. It's on the inside, it's a little lawn and within it are millions of chemicals and it forms a layer that protects the underlying cells, the endothelial cells, from damage. So as red blood cells and other things come across, it keeps them away from bashing and scraping at them. And if you've got diabetes, the high sugar level degrades that glycocalyx such that it becomes very thin. So, your endothelial cells become much more open to damage by external agents of any sort. So then again, you have this as endothelial damage caused by high blood sugar damaging the glycocalyx. What we're looking at essentially, is a process called endothelial damage. Endothelial cells are very thin cells, they line all your blood vessels in your body. If you damage these, all hell can break loose, so you must protect your endothelial cells. The next thing that will tend to happen is a blood clot forms if you damage the artery wall, and the size and nastiness of that blood clot is dependent on all sorts of factors. You know, stress, high blood sugar levels as well again do that, Lp(a) is a blood clotting factor. It sticks to artery walls very readily. So, then the blood clot forms. You want a small blood clot, you don't want a big blood clot. And then you have the repair systems. So once this is all happened, everything starts to repair itself. So, I say to people, what you've got in your system is that it's been continuously attacked. So, I liken it to say, a road surface. The road, there's sunlight and there's rain and cars going across it and things bashing into it, and eventually it breaks down and potholes develop. And eventually, if you don't repair it, all hell breaks loose. The arterial system is under constant attack from everything that's going on inside, you know, bacteria release exotoxins that attack your endothelial cells, there's all sorts of nasty things that can do damage your endothelial cells. Once that damage is at a high level, and the damage overcomes the repair, then you get the potholes and get the damage. So, what you're trying to do is ensure that the repair systems are working faster than the damaging systems. And if you can achieve that, then you can at least hold it or maybe even reverse it.

Steven Bruce
That was a question which came in ages ago by Dawn and I've been waiting to ask it. She's asked whether you can reverse the damage to the arteries?

Malcolm Kendrick
It's very unclear this one, there's a thing called a coronary artery calcium score. I don't know if you ever heard it, but you do a scan of the heart, CT scan, and it can show you the amount of calcium that's built up in your arteries. Now calcium is a late stage atherosclerotic development where calcium builds up, if you get scars, they've got calcium in them, and if you get a lot of calcium in your arteries, that means you've got a lot of underlying atherosclerosis has been happening for years and years. Now, they have looked at measuring this, the problem is of course, you're really looking at cardiovascular past, not necessarily your cardiovascular present. But if you've got a high calcium score, and some people have said with high calcium scores, if people have lowered their blood pressure and lowered their diabetes risk and taken exercise, that they can stabilise it at worst. So, if the score was 300, you can stay at 300. I know some people have seen some diminishment in these scores, which suggests that the plaques are regressing. So, I think

it is possible. I mean, the repair systems are happening all the time, your body's always trying to repair itself. Once it's reached a certain stage, once you get calcium in your arteries, it's quite difficult for your body to do much about that. But at least if you can stop that figure from going up, it means nothing else is going on. And then the repair systems that are already operating will stabilise what's happening. And therefore, you have definitely reduced your risk considerably. So, it is possible to do things. That's the final bit of the book I'm writing at the moment as well. Okay, so what can you do? What actually works, what will be helpful to tell people to direct them in useful ways?

Steven Bruce
Jan has asked a question about diabetes and statins. She wants to know your opinion of statins for type one diabetics. "Even though my cholesterol levels were good, what is it that makes my GP think that statins have a wider beneficial effect for cardiovascular risk in type one diabetics?"

Malcolm Kendrick
Well, I think that type one diabetics are at a greater risk of cardiovascular disease, although it's not a huge risk. And if you can keep your blood sugar within good target ranges, you know, that's the thing to do. There hasn't been a study on statins and type one diabetes. Never. So, there isn't any evidence. But basically, we've reached the point where, if your doctor thinks you're at increased risk of cardiovascular disease, they will recommend you take a statin. No matter if it has beneficial or other effects. However, there is no doubt that statins increase the risk of diabetes, not type one diabetes, but type two diabetes because they interfere with them with the function of insulin around your body. So, I would caution, I would ask for the evidence from that doctor and say, could you show me the evidence or the benefit of statins and type one diabetes? You can ask them that sure in the certain knowledge that there isn't any.

Steven Bruce
I suppose part of being part of what you've said so far would make people believe that you've have said that statins do lower the risk of death from cardiovascular disease a certain amount. I guess we should infer from that, that what you're saying is there are probably other drugs with fewer adverse side effects that could do the job just as well.

Malcolm Kendrick
I think it's not drugs, really, that I'd be looking at. I'd be looking at lifestyle issues here. There's evidence that people who takes statins end up then taking less exercise because they think they're protected. There's evidence that people who take statins do become more obese than people not taking statins. So, what you're much better to do, and these things are more likely: diabetes increases your risk of cardiovascular disease by say threefold, right? Approximately. And a statin might reduce your risk of cardiovascular disease by about 0.1%. These are absolute figures. So, you'd be better off looking at what's causing you to have a high blood sugar level and try and organise that, like reduce carbohydrate intake, take some exercise, go out in the sun, relax, do some meditation. These things will have measurably important and far more important effects than taking medications. I always advise in these situations, medications last, if nothing else is working and you can't get anywhere with anything else. I don't want to I don't want to be saying publicly, don't bother. But the benefits are really pretty small. And there's so many other things you could be doing. There's a study from Sweden, for example, showing that women who went out in the sun more, the highest sun exposure women, protected themselves against dying of cardiovascular disease. And the effect was as great as if it's caused by smoking. So, women who go out in the sun more and get sunshine and get nitric oxide levels up have an increased life expectancy and are far less likely to die of cardiovascular disease than women who don't go in the sun. And that difference is as great as smoking 20 cigarettes a day in the other direction. So, there are some things you can do that are definitely beneficial.

Steven Bruce
Elspeth has asked whether you've written a paper where this is all written down because she'd love to learn more. I thought I plugged your books hard enough at the beginning of the show but I would recommend starting with The Great Cholesterol Con, I think A Statin Nation is great, even Doctoring Data, which is really about the statistics of all this, it's eminently readable stuff. Pretty soon there'll be a fourth one.

Malcolm Kendrick
Well, this one is if you like, I have written a blog, which has reached about part 69 saying, here's what causes heart disease and some of the things you can do to prevent it. The book I'm currently writing is more kind of bringing it all together because my first two books said essentially, what doesn't cause heart disease? Forget about these things. This is more about what does really cause heart disease and what you can do, as many things as you can without swamping people, what you can do to prevent it. I was just writing a section taking the mickey out of these lifestyle people. I think it was Mark Wahlberg who put his daily diary on the internet, like 2:30 get up, 2:45 in the gym, 3:00 under a cold shower or whatever, just a ridiculous kind of thing. Most people are never going to ever be able to put in that level. So, it has to be also things that people can find relatively easy and fun if you can imagine fun, rather than just grinding your way to health, because I think that's not the way that you want to see it.

Steven Bruce
In terms of your written material, again you mentioned your blog, if people google your name, Malcolm Kendrick, then you they will find a link to your blog and the blog comes out what, you must produce a one a week?

Malcolm Kendrick
It's around about that. Probably.

Steven Bruce
And again, they are immensely readable.

Malcolm Kendrick
Lots of COVID recently, but really, I think I'm trying to reduce the level of anxiety that people have about things. The level of anxiety about heart disease, the level of anxiety people have about these medical conditions. I mean, most of us do remain remarkably well and healthy. You know, these risks are overemphasised in many cases, you know.

Steven Bruce
Robin has asked a question, which I suspect you've got some fairly strong opinions about, which is why don't NICE know all this information?

Malcolm Kendrick
Well, there is a certain narrative- that's what I call it, is that the right word?- that people get into. And it is basically the narrative is: cholesterol is bad for you, you must lower it. That's it. And you can't step out of that world. Because you pretty soon become an ex-expert very quickly. I sometimes liken it to if you're Manchester United supporter, there's no way in God's Earth you're ever going to say, you know what, I think Manchester City played better football, I'm going to go up and support them. It's just never gonna happen. Now, why wouldn't you do that? Logically, if they're playing better football that you would enjoy more, why not go off and watch Man City? Because once you've got locked into an idea, the most resilient parasite is an idea. And once people have a very strongly held idea, they just won't see it. There's another famous American politician, whose name escapes me, but this famous quote, "It's very

difficult to get someone to believe in something if their salary depends on them and not believing in it." And I think there's all sorts of reasons why these things happen, you do end up with tribes, you do end up with ideas. I've read through the history of medical thinking and ideas that held sway for a long time. And throughout history, it's just been the case that an idea gains ascendancy and then it becomes incredibly difficult to shift it. One of the saddest ones was the radical mastectomy for breast cancer, which actually was first developed in late 19th century by a surgeon called Halsted who said, you must remove the tumour, the breast, the other breast, as much tissue as you can, all the nodes, everything. Horribly disfiguring operation. But if you as a surgeon didn't do that, you were ostracised, you lost your job, you were thrown out of the societies. In fact, John Maynard Keynes' brother was a surgeon who questioned the radical mastectomy, was thrown out of the Royal College of Surgeons, lost his job never and worked again. That's the kind of implacable barriers you come up against. Do they know this? Do NICE know this? Well, there's a bit of them probably does know this. Who's going to be the first to go, you know what? I don't know. Max Planck said, science progresses one funeral at a time.

Steven Bruce
You drew my attention in one of your blogs, I think, to what I think it's fair to call a scandal that Professor Peter Gotzsche was ousted from the Nordic Cochrane Centre, which he founded, because he refused to back down from his very, very robust studies which had shown that mammography screening was not beneficial.

Malcolm Kendrick
He didn't get chucked out for mammography screening, although he did get a lot of flack for saying that mammography screening does more harm than good. He got really attacked for that one. I happen to believe that he's right. That screening is presented as, a bit like all those things are presented, as a universal good. They don't take into account the downsides. You say someone screening is a good idea, it picks up the cancer early, then we can treat it early, then the woman is fully treated. Who could object to that? Well, no one could object to that. I couldn't object to that. If that's what actually happened, I'd be the number one supporter. However, when you start looking into it, all sorts of issues, false positives, false negatives. Going back to Peter Gotzsche, he got kicked out of the Cochrane Collaboration, because he did a review of the HPV vaccine, which is the one for preventing cervical cancer, and he said that basically there hadn't been sufficient studies done on safety and the safety studies were very disturbing. And at that point, he was removed from the Cochrane Collaboration. So, yes, criticising heart disease and criticising anything to do with COVID is bad enough, make one statement that could suggest that vaccines have any issues at all that could be concerning and you are toast.

Steven Bruce
Which I guess is at the nub of what I wanted to get to this evening about how much we can trust the evidence base, because when somebody who- and I've read Peter Gotzsche's book on mammography screening and I'm convinced by his credentials as an unbiased and very educated researcher, he's capable of understanding the statistics of what he's reading and if he doesn't, he gets an expert to do it- but when someone like him can be ousted for stating what I imagine is a completely objective opinion backed up by evidence, how much can we trust the evidence? How can we trust it when Richard Horton, the editor of The Lancet, says that he can't trust the evidence? And that must be, what, number two medical journal in the world?

Malcolm Kendrick
The editor of The New England Journal of Medicine for many years, Marcia Angell, basically, can't remember the exact words, but said I can no longer trust the evidence coming from opinion leaders or published research or the people in guidelines. And she was publishing the most widely rated medical journal in the world.

Steven Bruce
Where do you stand when you're giving advice to patients, and I must get to some of the questions from our audience very soon as I've been neglecting them and I apologise to them for that. You mentioned earlier on you would not want to say, certainly in public, to everybody stop taking statins. But at some point, you must be advising patients to do things which are not in keeping with the NICE guidelines or potentially you are.

Malcolm Kendrick
What I do is usually just say these are the facts as I understand them, other people in the medical profession have another view, I am just presenting you with the data as I understand it. The decision on what to do is entirely yours. So, I don't ever advise or tell anyone to do that. I just present them with what I consider the evidence and then it's up to them to make their own decision.

Steven Bruce
From our perspective as osteopaths and chiropractors, we are required to have a wider view of healthcare as a whole. When I'm asked questions like that, when people tell me about statins, I simply tell them to read your book.

Malcolm Kendrick
Yeah, I sometimes say just read my book and you'll know what advice I would give you. But you're allowed to still within the General Medical Council, aka Spanish Inquisition, as long as you don't give direct advice to patients, you can make generic states or general statements about things. So, I think we still have that flexibility if you like to do this, but it is becoming an increasingly guideline driven from evidence, that the evidence itself is often not terribly impressive, should we say. So, I think it does become issue I don't know where you draw the line. The most widely downloaded paper in the last 15 years was by John Ionnidis, a Greek who's now a professor in the states, and he wrote a paper called Why Most Published Data are False. He's saying most published data are false. And Richard Horton said at least 50% of the data that published is untrue. At least 50%, so that's a majority. So, this is a very worrying world, where a) the editor of a major journal doesn't even know if he's publishing stuff that's true or not, b) if at least 50% and perhaps the majority of the evidence, the medical research that's being published is false. The very foundations upon which you should be doing medical practice are no longer there, they've gone.

Steven Bruce
We in our professions would be castigated by our general councils if we allowed our beliefs about treatment to cause us to advise patients incorrectly against NHS guidelines. But it was seen that there are plenty of people who are formulating those guidelines who have beliefs about treatment, which are influencing their interpretation of the data.

Malcolm Kendrick
Well, of course, it's always been like this in the history of medicine. It's perhaps more biased now than it has ever been, I think would be possibly true due to the financial imperatives.

Steven Bruce
Can I drag you back to cardiovascular disease? Sylvie has asked, is it Lp(a)s which are blocking the arteries?

Malcolm Kendrick
Well, not exactly. The plaque, the thing that builds up in the artery and blocks it, consists of many different bits. So, you'll get Lp(a) in there, you'll get red blood cells in there, you get lipids is in there, you get macrophages in there, you

get smooth muscle cells. There's a whole lot of stuff in there. It just basically is a load of stuff, in some plaques there's more of one thing than another thing and more of another thing than another thing. But in general, no, the Lp(a) is a player but it's not by any means the number one player. Platelets are small blood vessels that are involved in blood clotting, you look inside plaques, you'll find them. You find fibrin, you find red blood cells, you'll find, essentially, almost anything that floats around in the blood can become incorporated into a plaque. So, you can't say for sure that any one substance is the number one substance that's causing it.

Steven Bruce
And Mel has asked about aspirin. The advice on aspirin seems to have changed recently and she's asking whether aspirin helps with cardiovascular disease?

Malcolm Kendrick
It does, it doesn't have a massive effect. If your risk is very low, it's not terribly beneficial. The other problem is that taking aspirin every day can cause adverse effects, such as stomach ulcers, even at a low dose. So, if you start adding up the benefits versus the disbenefits, then it's not hugely beneficial. A bit like statins as I always like to say, there's some benefit there, yes but is it worth taking off every day? Maybe. I can't be definitive on that one.

Steven Bruce
Mark has asked whether you could talk about the relationship between statins and dementia?

Malcolm Kendrick
There is evidence that having a low LDL level, cholesterol level, is associated with increased risk of neurological conditions such as Parkinson's and dementia. The problem we have here is, of course, when they've done the statin studies, they have tended to be done on people with high cholesterol levels, who are therefore at lower risk of developing dementia. And therefore, you're giving statins to people at a generally lower risk then finding that they are at low risk and then saying the statin has reduced the risk of dementia, which is, of course, nonsense. That's what we call a confounder. Do statins cause dementia? I've seen some patients who've been clearly affected by statins, mentally affected. In fact, we had one lady who was about to have a lawyer in, she was lacking capacity and therefore she was going to have power of attorney activated but she took the statin off and five days later was as bright as a button. So, I've seen that some patients are quite badly affected by the impact of statins. Which is not incredibly surprising because there is more cholesterol in your brain than in any other part of your body. And the myelin sheath around nerves is made up with a very high concentration of cholesterol. In fact, there are cells in your brain called glial cells, one of their primary purposes is to synthesise cholesterol to nurture the neurons themselves and some statins more than others can penetrate the blood brain barrier and can reduce cholesterol production in the brain. So, there's some quite strong theoretical possibility that statins can increase, say dementia or whatever term you want to use for it. So, I'm not sure the evidence is entirely powerful enough in either way to make any certain statements.

Steven Bruce
Mel has asked whether alendronic acid and calcium supplementation could have an adverse effect on arterial disease?

Malcolm Kendrick
Well, calcium can that's for sure. We already know that, that's been proven in many studies that calcium supplementation can increase the risk of cardiovascular disease.

Steven Bruce

Does it have to be massive supplementation or is it relatively low levels?

Malcolm Kendrick
I can't really say but a lot of people are on quite high levels of calcium supplementation. So, you got to be careful with this stuff. Alendronic acid. Well, I have my own issues with alendronic acid to do with microfractures in bones and making them more likely to fracture, even though they may be more dense. And there's some quite disturbing evidence coming out with regards to alendronic acid and the impact on bone strength. And essentially, you can make the bones more dense, but they are more likely to fracture and when they have these fractures are much more difficult to treat because the bone has become effectively more brittle. I think that alendronic acid, I've not really looked at its impact on cardiovascular disease, but I've looked at osteoporosis and I'm not impressed.

Steven Bruce
Elspeth has asked about libido and statins. She says she's found that in her patients, those who take statins also have problems with their sex lives. Is that a well-known adverse effect?

Malcolm Kendrick
It is known and written down in the BNF as an adverse effect. Yes. So, it definitely can occur. I've seen it myself in people, patients rarely complain about it but if you ask them about it, many of them will say it is an issue. So yes, there is a there is a correlation.

Steven Bruce
That's actually, something you said there, is a matter you raised in one of your books, I can't remember which one, is that actually many GPs possibly don't realise the adverse effects of statins because unless you ask about them, the patient won't mention them. And so, they'll measure the obvious cardiovascular things but, certainly in an osteopathic or a chiropractic practice, it would be less common for us to about to ask about sex life, for example.

Malcolm Kendrick
Yeah, well, it will be, you only got 10 minutes per patient or whatever it is. I'm trying to get out now of that nonsense of finding out what's really wrong with patients. So, I think, yes, that's true. I was actually doing a little interview, this is going back a few years, and talking about one of the adverse effects of statins was people getting very irritable and angry, which has been seen, and there was a guy who was actually sorting out something electrical next to me, he was just doing some work in the background, and when we finish he said, You know what? You're describing my wife, she's gone on statins and since she's been one statins, that's exactly what she's been like, irritable flying off the hook. He said, I'm going back and telling her to stop taking statins. And I was just thinking that he would never have made that association if he'd not heard me speaking to somebody else at the time. And of course, the problem is that mainstream medicine says if you ask about things, it's like leading questions. And then you're making people think there's gonna be a problem and then there's a problem and therefore there's not really a problem. So, it's kind of the ultimate cop out isn't it? You can't talk about any of the things or mention any of these things because it will just cause them to happen. Well, no, you can ask about these things. Because if you ask about it and say have you had problems with becoming more irritable or upset, well, they won't make that association themselves. And sometimes they won't say that to you then but they will go away and think about it in then maybe next time when they come back, they'll say, you know what? That has happened to me. So, people don't just tell you stuff straight up, straight off. They just don't, that's not what happens in the world. It takes them a while to decide whether or not they're going to tell you about stuff. So, you know, when you've got a subtle thing like irritability or loss of sex drive or feeling low or being a bit worried about your memory or whatever. The other thing I always say, that statins may add 15 years to

your life, they don't make you live 15 years longer, they just make you feel 15 years older. The problem is an awful lot of the adverse effects are exactly the same as happen to people as they get older, therefore it tends to get dismissed as well. You're getting old, what do you expect? So, it's very subtle many of the problems as well. So that makes it very difficult to pick it up.

Steven Bruce
Catarina has asked about Q10 supplementation and asks, is it true that it reduces the risk from blood pressure? And do patients continue taking medication if they're taking q 10?

Malcolm Kendrick
Well, yeah, I think you can take medication and Q10. Obviously, Co Q10, otherwise known as ubiquinone, is involved in the creation of ATP in your mitochondria. It's called ubiquinone, because it's ubiquitous and essential. Statins knock Q10 production down by about 50%. So, it should not really be any surprise that you might get things like muscle pain or weakness or fatigue or tiredness or loss of libido or all these things because basically someone's gone and turned your energy production down by whatever. So, if you're taking statins, definitely take Q10. Is it beneficial otherwise? I've never seen it do anyone any harm. But can you take other medications? There might be something? I'd have to look it up, I don't know if there's any specific contraindications or interactions with any specific drugs, but there may be some that do something. I'm not sure.

Steven Bruce
Well, Claire, who we were speaking to before we went live, has just sent in an observation saying that she's naturally irritable and I shouldn't blame it on any statins because she's not taking those. Matthew Davis has asked whether high levels of vitamin D3 supplement can aggravate cardiovascular disease?

Malcolm Kendrick
I've seen this mentioned, I've not seen any strong evidence one way or the other. My own tendency is to say that there are B vitamins that are beneficial, thyamine is definitely one of them. The problem with vitamin researcher is that it's hugely influenced by a very strong and enormously powerful pharmaceutical lobby, who seem to have at least part of their vast billions spent on trying to say that the vitamins are dangerous, they don't work but they're dangerous, you shouldn't take them. And there's a real major drive, there's a Codex whatever it is, trying to stop anybody being given vitamins or prescribed vitamins or being told to take vitamins at any level actually might do any good. You get a lot of vitamins studies, especially the vitamin D studies, at the moment and give people doses that are homoeopathic, essentially, and then go well, we didn't see any benefit for anyone taking vitamin D in our studies and I'm not surprised, you didn't give enough. They're very clever at doing this with setting up studies to deliberately prove something doesn't work by knowing beforehand what doesn't work and then doing a study and saying, well, we showed it didn't work. And then showing that things of danger. As far as I'm aware, in the last 10 years now people have communicated with me about this, the other thing about things that kill you in the States every year, it's estimated that 240,000 deaths due to medications, correctly prescribed medications, every year in the States. And over a period, I think they've managed to find of 10 years, they've had one person who had been killed due to overdosing on vitamins. Whether that was true or not.

Steven Bruce

Malcolm, we're at the end of our scheduled time, and as always, I've run out of time to ask all the questions that came in. Very, very grateful to you for giving up your time this evening and looking forward to seeing the new book when it comes out. But for now, hopefully you'll keep producing that blog and I can only recommend people very strongly that they follow you there.

Malcolm Kendrick
Well, thank you very much. I hope I've been of some assistance to some people this evening. I hope so. Anyway.

Steven Bruce
And it's been a great pleasure to have you on the show. Thank you again.

Malcolm Kendrick
Okay. Thank you.