A Statin Nation: Damaging Millions in a Brave New Post-Health World: A Special Interview With Dr. Malcolm Kendrick

By Dr. Joseph Mercola

MK: Dr. Malcolm Kendrick
JM: Dr. Joseph Mercola

JM: Welcome, everyone. This is Dr. Mercola, helping you take control of your health. Today we are joined from across the ocean by Dr. Malcolm Kendrick, who we've interviewed previously for his book, "Doctoring Data: How to Sort Out Medical Advice From Medical Nonsense" – or data, depending on which way you like to pronounce it. But today, we're going to talk about his new book, "A Statin Nation: Damaging Millions in a Brave New Post-Health World", which really goes deep into helping us understand some of the challenges with the conventional approach on this. Welcome and thank you for joining us today, Dr. Kendrick.

MK: Thank you for inviting me.

JM: Yeah. It was great to read your book. You had written – I think this is your third book, if I'm not mistaken.

MK: Yup.

JM: A previous book that you wrote is "The Great Cholesterol Con: The Truth About What Really Causes Heart Disease and How to Avoid It," which, I guess, more or less addresses the cholesterol controversy. Can you discuss the reasons why you wanted to write this book, "A Statin Nation?"

MK: I think a number of people say a lot of things have changed in the last 10 years, so it's 10 years since I wrote this book. It seems ages.

JM: Okay. That's a good reason.

MK: I said, "I'll do a little bit of an update on some of the things that have happened." As you probably know, there's been a number of new cholesterol-lowering agents that have arrived, which drive low-density lipoprotein even lower than before. There's quite a few things that have changed. A lot of things have stayed the same. But I wanted to also add in a bit more information about sort of what you could do to hopefully prevent heart disease as well.

JM: Okay. Great. Now, I guess before we go there, I want to give a broader perspective because you describe yourself as a skeptic, but I think a good skeptic, because we have some skeptics who are disputing what we say, but I think we've turned that around. I guess because of your positions, you've been deleted from Wikipedia, just recently actually. Do you want to comment on that at all or?

MK: Well, I didn't even know I was on Wikipedia. I only found out because someone said, "You're about to be deleted." I'm like, "This is terrible. My life has come to an end." I wasn't really bothered about me being deleted, if you like, because these things are what they are. But I think it was more of a principle of the thing, because it seems that there's a move out there. There seems to be people out there whose entire function seems to be to try and just destroy anyone who disagrees with them.

They have a thing called Rational Wiki now, which they're putting up stuff about how idiotic I am and how stupid I am and how I know nothing. I mean really just some quite, personally, insulting stuff, which doesn't bother me, but you do sort of feel like you got into a little bit of a maelstrom. I'm sure you've had more than your fair share of this kind of nonsense.

JM: Yeah. I guess you've got two potential possibilities. One is you could not be on the page at all, or otherwise you could be on the page and they can just blast the heck out of you like they do to me. They'll disparage you and discredit you and make everyone believe that you're not worthy of looking at or even worse than that, because you're heralding nonsense and misinformation. In some ways, it's good that you're not on it.

Wikipedia is run by the skeptics. They're an organization that is dedicated to helping or really discrediting people who have our types of positions and really focus on natural therapies and point out the flaws and harm that can come from applying many of these conventional approaches, like drugs, that you're going to talk about in your book, "A Statin Nation"

MK: Yeah. I think, also in the U.K., one of the widest-read papers, Mail on Sunday, two weeks ago had a real go at – I think you maybe know this – Zoe Harcombe, saying that we caused hundreds of thousands of people to stop taking their statins and that we've caused, therefore, thousands of people to die. I think the headline was "There should be a special place in hell for people who deny the use of statins," really quite horrible stuff.

But I went and looked up the data on deaths from heart disease when the article came out. This is some years ago. I didn't write it, by the way. They I did. Their facts were, as usual, complete nonsense. I then looked up the fact that in the year after this article came out – it was supposed to frighten people of taking statins – there was actually a reduction in death rate from heart disease of 4.5 percent, compared to the previous year and compared to the year after. It looked like all that happened was there was a dip in the level of deaths from heart disease, which would kind of counteract their argument, which is all based on modelling nonsense.

But these people come at you hard and they don't pull their punches. One of the things that they said to me was, "We can find no evidence that you work as a doctor in the United Kingdom or registered as a doctor." I went, "If you want to put that, then that's fine. But I will then sue you because I am a doctor. I work in the U.K. This is just nonsense." They really don't pull back on it, do they? As if somehow they have the righteousness behind them. It's really quite appalling.

JM: Well, they certainly have the revenue stream behind them and backing from the pharmaceutical companies. I guess most of the article that they wrote about you was correct. In fact, they just got the words mixed up a little bit, so that when all these people went off of their

statins, instead of hundreds or hundreds of thousands of people dying, you actually saved their lives. A special place in hell should not be reserved for you. It should be reserved for them, who only misunderstood that because of their complete ignorance of the reality.

MK: Absolutely. I mean they rely very heavily. You may or may not know that there's a group in the U.K. who call themselves the Cholesterol Treatment Trialists' (CTT) Collaboration. They've got all the data – or data, as they say in America – from the Statin Trials. They hold it. They won't let anyone else look at it ever. They keep producing these meta-analyses showing how wonderful statins are and that they don't have adverse effects, and we're supposed to believe them. Although they run a clinical trials unit, last time I looked, they earned well over 400 million dollars in funding from pharmaceutical companies almost entirely – those companies that produce cholesterol-lowering agents.

I mean we have a completely biased organization paying hundreds of millions to hold all the data, and then tell us, "No one else can look at it. By the way, you should believe everything we say." I mean, how on Earth can this be allowed to happen? It happened in any other area of the world. It's happened where journalism was a legal profession or anywhere, you'd just be laughed at. It would not be allowed. Yet somehow, these people have got themselves such a standing and status that we're supposed to go, "Well, you said it. It must be right." This is ridiculous.

JM: I'm not sure that it's just restricted to health or medicine. I think it's really incumbent upon most industries. I'm writing a book on electromagnetic fields (EMFs). It's certainly prevalent in wireless telecommunications industry. It was certainly present in the tobacco industry. It's just this whole process that they go about.

Typically, it's largely a result of the massive amount of revenues that are created from these products that are backing this. One of the comments you make in your book is that statins are the most profitable drugs ever created in the history of mankind, grossing more than a trillion. That's 1,000 billion dollars. Why don't you elaborate on that? Because I thought that was an astounding statistic.

MK: Well, it is an astounding statistic. I mean I'm not entirely sure if my trillion figure is exactly right, but it's not far off. Other people have tried to calculate it. We know that Lipitor – it's called in the States atorvastatin, the Pfizer drug – at its max, it was making right about 35 billion dollars a year. That's just one statin in one year. A lot of the others have made billions – Almost all of them have made billions each year. Atorvastatin, Lipitor made the most.

When you look at those amounts of money, that really funds an awful lot of marketing. An awful lot of people can be paid very large sums of money to go to attend meetings and run guidelines. When I last looked at the guidelines of NCEP, National Cholesterol Education Panel, when they came out with the latest guidelines saying cholesterol should be lowered even more – out of nine I met of people in the committee, there were 124 conflicts of interest with companies making statins or other cholesterol-lowering agents. It's not surprising that we get the answers that we do, is it really? I mean what else do we expect is going to happen?

JM: Yeah. Precisely. As I mentioned earlier, you wrote the book, which I interviewed you for, "Doctoring Data." I think it might be wise to review one of the central concepts of that book, because it's certainly pertinent to this issue. That is the manipulation of data, primarily with respect to the industry confusing absolute and relative risk.

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Well, not maybe confusing it but using it to their advantage in two ways. They use it to increase the perception of benefit, and then when they were looking at trying to market that and convince physicians and patients to take the drugs and they use the other version to decrease the perception of risk and side effect. Why don't you review that? Because it's fascinating.

MK: You'd think there would be some way of saying there's one way to present the benefits of a drug or an intervention and it's going to be the same and it could be measured and it means something. What we managed to do – In fact, I've asked about 50 medical colleagues this question, "What's the difference between absolute and relative risk?" None of them has given me the correct answer. That's the sort of worrying thing. When people go to their doctors, they expect their doctor to tell them what the benefits are, and the doctors don't understand the benefits themselves, which is true also to various areas, like screening etc.

But the difference – I've tried to explain this as simply as possible. Someone said, "The difference between absolute and relative risk is the difference between multiplication and addition." If you say 100 people – we'll just use that as 100 people – and started them on a medication, say blood pressure-lowering tablet or a placebo, so 100 people on the blood-pressure lowering, 100 people on the placebo. You run the trial. We'll call it for a year. At the end of which, one person died in the treatment arm and two people died in the placebo arm.

What you have there is the difference in deaths between one and two. That's a 50-percent difference. The absolute risk is that 98 people were still alive in the placebo arm, 99 people were alive in the treatment arm. That's a 1-percent difference. The absolute difference is 1 percent. The relative difference is between one and two. That's 50 percent. I think I'm making that clear.

But if you run an experiment and then do it with 1,000 people instead of 100 people, at the end of the trial you get the same result, that's one person died in the treatment arm and two people died in the placebo arm, then there's still a relative difference of one and two is 50 percent, between one and two. But the absolute difference is between 999 and 998. That is 0.100 percent. If you keep running these figures, the relative risks can look incredibly impressive, so there can be a 50-percent reduction in something, relative reduction, but the absolute reduction could be 0.0000001 or 0.0000002.

What the industry has recognized is that people see these figures like a 36-percent difference in heart attack rate. The other thing that they do is they don't even mention overall mortality. They will say the difference in heart attack rates. There's a very famous advert for Lipitor saying a 36-percent reduction in heart attacks. When you examine the figures, the difference is – I can't remember it off the top of my head, but I think it was 1.1 percent absolute risk in the heart attack rate. Then you ask the next question: "What was the difference in death rate?"

JM: The most important question.

MK: Well, of course it's the most important question because you don't take a drug just to die of one thing and not to die of another thing. I'll use the example of when you push people off cliffs, then 100 percent of them will avoid dying from heart disease. You could say, "I could reduce the risk of dying of heart disease to 0 percent by pushing people off cliffs." You may not think it's a good intervention, but this thing of the overall mortality is the key important thing, because it's another thing. You can only die of one thing, so people die more of liver cancer or kidney failure or muscle breakdown.

We see this happening when people discuss the new medications. Repatha is a new lipid-lowering medication. It was presented as being absolutely fantastic in reducing the rate of heart disease death and myocardial infarction by 20 percent. When you looked at the absolute figures for death, more people died on the Repatha than there were in the placebo. More people died, and more people died of heart disease as well. This was presented as if it was a fantastic success.

We have a drug that you give to people. It's enormously expensive and more people will be dead if they take it than if they don't take it. Yet, the pharmaceutical industry manages to present this as a resounding, outstanding success. It's quite extraordinary.

JM: Yes. The Repatha is, I believe, a drug in the PSCK9 category. Isn't it thousands of dollars a month?

MK: I don't know what it is in the States, but the last time I looked, it was 14,000 dollars a month – No. Fourteen thousand dollars a year. That's right.

JM: Yeah. So it's over 1,000 dollars a month.

MK: It's over 1,000 dollars a month. They're trying to promote this. They're doing it in the U.K. as well. Obviously they're doing the usual marketing thing of saying we need to treat people who've got familial hypercholesterolemia, because these are the people at greatest risk, and there's only 1 in 250 people who've got this or whatever people they've come up with. It looks like, "Oh, that's quite a small target," but this is what they always do.

Because initially, when cholesterol guidelines came along, high cholesterol was – you use different figures in the States – at 7.5 millimoles in Europe. That would be approximately 300 milligrams per deciliter in the States. That was what was considered high. They then realized, "Well, we cannot have that figure because there's not very many people who've got a level that high." They had a meeting and they brought down the level of what we called normal to 200. Just like that, not based on any evidence whatsoever.

Of course, what you find is the vast majority of the population have a low-density lipoprotein (LDL) cholesterol level higher than that. At a stroke, we've turned the majority of people in the world into having a disease that needs to be given a drug that needs to be given for the rest of their lives. This is just – How can it possibly be? I looked at the figures in the U.K. In one population

group, 82 percent of people have a cholesterol level that is higher than that, which is considered average.

Of course, the other thing that's happened is there is no normal level of cholesterol anymore. There is no average. There used to be, but there isn't anymore. If you look at anything, there's raised cholesterol or raised LDL. There's optimal, but there's no lower limit. It's gone. We've reached a point whereby any level of cholesterol is now considered to be too high and any level can benefit from being lower, which is completely bonkers. But this is where we've reached. It seems that we're just going to accept that, "This is OK. This is fine. Everybody's got a high level of something, which is essential to their physiology." It's completely bonkers.

JM: Yes, it is indeed. I just want to mention here that if you and others are successful on helping people understand the dangers of these statins and eventually most people stop them, that it's important to realize that this other drug you mentioned, Repatha, is in the proprotein convertase substilisin/kexin type 9 (PSCK9) category. That's just a whole category, just like statin. Just like there are loads of drugs within the statin category. There are loads of drugs or there will be loads of drugs in the PSCK9 category.

What's true of Repatha will probably be true for most of the other ones – They're going to increase the mortality rate. But I want to get back to the cholesterol level, because you really touched on a question, which I think I'd like you to expand on, which is, "What do you believe to be the ideal, optimal, normal, healthy cholesterol level in milligrams per deciliter?"

MK: I have to do a conversion. It's 38.72 or whatever it is in my head. The normal total cholesterol level – because then you start to build subsections of subsections and subsections of subsections – The normal healthy level is – I do say to people, I'm going to change this slightly – When people say to me, "Oh. My cholesterol's high," "You're not worried about your height." I say, "Anyone who's between about 4'8" and 6'10" is okay." if you move out of that, you bump your head too many times when you're on that top. What's a normal, healthy cholesterol level is probably almost whatever you've got.

I'm not worried about it. I know what my cholesterol level is. It's 300. I didn't want it to be tanked but they did it by mistake when I went to GPs--. "You know, your cholesterol level's 300." I went, "Fine. It's not quite high. I'd like it a little bit higher." Because one of the things we wrote a paper with a few other doctors where we looked at the cholesterol levels and death rate in populations. What we found is that once you reach the edge of that 55 to 60-ish, those people with higher cholesterol levels live longer than people with lower cholesterol levels. It's not huge. It's not a gigantic difference, but it exists.

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Essentially – that's true even with people who've got familial hypercholesterolemia. There's a small subgroup of people with familial hypercholesterolemia who will die young. We wrote a paper on that demonstrating that it has nothing to do with the cholesterol or LDL level. It has to do with blood-clotting factors. The two things that are closely related on your genome, some people get both, some people get one. If you have the clotting factor problem, it's the major thing.

JM: Interesting. So they have increased clotting risk?

MK: Very much so. Well, not all do, but some do, moving in a genetic argument. What you can find is you can have siblings, one of whom has the hypercholesterolemia, the other one does not, alright? They both have the clotting factor, and then they have the same risk of dying of heart disease. Because actually, the LDL receptor itself, the thing that takes LDL out of the system, also takes Factor VIII out of the system as well. If you have less LDL receptors, you have higher levels of clotting factors. The thing that is probably damaging in familial hypercholesterolemia in some people is not the high LDL or the high cholesterol level. It's the fact that the clotting factors are not being taken out by the receptors.

Everything in the body does more than one thing, as you know. Everything is hypercomplicated. Essentially, people have been weirdly – well, not weirdly – have been deliberately looking at the wrong thing. When you get young people who would die at the age of about 35 with high LDL in familial hypercholesterolemia, they're very different people than the other groups who can live as long. In fact, if you have familial hypercholesterolemia, even some of the strongest supporters of the whole cholesterol hypothesis have noted that you will live just as long as anybody else. This is not something that reduces your life expectancy, and yet we are made to be terrified of it. It's completely insane.

JM: That is fascinating. In fact, familial hypercholesterolemia has really been the only medical justification in my view to put people on hyperlipidemic agents like statins. I have never heard that before. I maybe heard it and forgot. But just to give the audience an idea of how common this is, it's less than 100,000. Most physicians will only see one or two patients in their lifetime, certainly less than 10 if they have a general practice. If a person has this or a physician is monitoring someone, what is the specific clinical test they can check if this anomaly exists?

MK: Well, there's nothing standard, because this is not widely accepted. I was speaking to – Or in fact on my blog, someone contacted me and they said that they had familial hypercholesterolemia. Their LDL level was 700 – no – 800 on average.

JM: Wow.

MK: They've been investigated because they've had this for such a long time. They could find no evidence of any heart disease in this individual or whatsoever. He's 72. They've done a paper on him. Of course the conclusion is he must be being protected by something because look at how high his LDL level is.

I speak to many – People communicate with me quite regularly saying, "My LDL level or my total cholesterol level is 600, 700 or 800." One lady came up to me after I've given a talk. She said, "My total cholesterol level is 800." She's 93. A friend of my mother has got a total cholesterol level of 600. She's 86. She has no heart disease. She's completely healthy. You can find example after example.

I think it's Karl Popper from his views on science says, "If your hypothesis is 'all swans are white,' finding another white swan proves very little. Find a black swan and your hypothesis is dead." I

know, directly, of at least more than 50 people who I've communicated with who've got cholesterol levels that are three to four times the normal. Their LDL levels can be five to six times the normal with no discernible heart disease. Some of them are very elderly indeed. Therefore, you've got to say, "Well, how can this be?" If you look at all the other data that comes from around the world –

There were researchers in Norway who contacted me. They've done the study called the Nord-Trondelag Healthy Study (HUNT 2), where they looked at 50,000 people over a period of 10 years, looked at their cholesterol levels and the rate of death, ischemic heart disease death. What they found was that basically for women, as their cholesterol level went up, the risk of death went down. We're not talking a small amount here. We're talking 40 percent reduced risk of death from ischemic heart disease. Women who had LDL levels of 300, 350 or 400 were 40 percent less likely to die from ischemic heart disease as women who had an LDL level or cholesterol level of 200 or less.

There was an enormous study done in Austria on hundreds of thousands of people. What they found was exactly the same thing. This was all-ages, ages 15 to 95. If your cholesterol level was lower, you were more likely to die younger. Now, the difference wasn't enormous, but it existed. The only population where they didn't find that was in younger men. But in younger men, there was a very slight increase in cardiovascular death than people with high cholesterol levels. That disappeared around the age of 50. In women of all ages, you would live longer if your cholesterol level was higher.

The weird thing is I can get these studies from around the world. There's a group in Japan who've looked at this. There's a study – I can't remember what it's called, Ishikawa, I think – where they looked at women. In Japan, they have a very low rate of heart disease anyway. But they found that this was a population of 12,000 women and they had a total cholesterol level of over 300. Over a 12-year period, not one of them died from ischemic heart disease, not one.

When people say to me raised cholesterol causes heart disease, the evidence just doesn't say this. I don't know if you know Zoe Harcombe. She's another researcher. She took all the countries in the world that measure such things – World Health Organization (WHO) – and did a graph of the cholesterol level and the rate of cardiovascular death in 186 or however many countries she can find. It was about 160. Basically, in all of these countries, you drew the line. As the cholesterol went up, the rate of heart disease went down.

When people say that it has been proven that cholesterol causes heart disease, there's so much evidence that says that it does not. Even the Framingham study itself, which I presume everybody knows about, Framingham, near Boston, which is the one that started the whole thing up. What they found was that over a 32-year period of research, the most dangerous thing that could happen to you is your cholesterol level started to fall.

When they took it at the beginning of the study and then they measured it over the years and then to about 14 years, if memory serves, for about a 10-percent reduction in your cholesterol level, the risk of cardiovascular death went up 500 percent. That's a relative risk, not an absolute risk. But they're still pretty gigantic. Even the Framingham study itself contradicts itself. They said that for

every 1 percent fall an LDL or cholesterol was a 2-percent fall in cardiovascular death. That figure doesn't exists from anywhere. Where does it come from? It's just been made up, and it's widely quoted everywhere. You'll read that everywhere. And you say, "Where did it come from?" It's just made up. People believe things where the facts don't exist. They're not supported. You do find yourself thinking, "Am I the only person – Have I gone mad? Am I looking at everything upside down?

JM: You've got approximately a trillion dollars in revenues perpetuating this myth. Interestingly, you've got not one, not two, not ten, but 50 black swans that disprove this myth. I'm wondering if you could tell us – What's even more remarkable about those 50 black swans? It's that they somehow managed to get to an elderly age and not be convinced by their physician to go on a statin drug or another agent to lower their cholesterol. That's even more remarkable. I don't know how they escaped that, but they did, which is really interesting. But had they been placed on a statin drug, tell us how they would have died prematurely.

MK: Well, the statin drugs – I mean, they're not – My problem with them is they cause quite a lot of adverse effects in a lot of people that are usually dismissed. Some people say, "I've got aching muscles. I've got aching joints. My memory's gotten a bit fuzzy or I've become impotent or whatever." And then the doctor just says, "Well, what do you expect? You're getting older," especially to men, and to women.

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But the problem that I have with the drugs is – I mean you're dealing with a really fundamental process of the human body, making cholesterol, which is a 37- or 39-step process or whatever it is. You're hacking off like the base of the tree, and the tree doesn't just make cholesterol. It makes another 10 or 15 important things for your body, like Coenzyme Q10 (CoQ10), which is, as you know, used by the mitochondria to create adenosine triphosphate (ATP), which is the energy. It's basically the energy in your body. If you get rid of ATP, you die.

Well, the pharmaceutical companies knew when they first started looking at statins that it reduced CoQ10 by 40 to 50 percent. At one time, they had a patent, which was going to be "If you're going to give someone statins, you have to give the CoQ10 at the same time; otherwise you might suffer from all these unpleasant effects." They decided not to go ahead with that patent, I think for obvious reasons. If you put the antidote to your drug and with your drug, people might think, "Maybe this isn't so wonderful after all."

The whole CoQ10 thing is there. CoQ10 is essential for your body, and it's essential for energy. There's a number of people – I think one of the most worrying thing is the statistics for heart failure have really started to rise and go through the roof. And yet, these people are not looking at this in association with statins. But your heart's a muscle. Your heart needs CoQ10. Statins, not CoQ10 on the head. People are getting heart failure. Why is this not being picked up?

Another really worrying side effect – well, it's not a side effect – an adverse effect that has been picked up – WHO first noted this – was an association with amyotrophic lateral sclerosis, which is, in America, known as Lou Gehrig's disease, I think. It's a really horrible motoneuron disease. More recently – I've blogged about this early last year – it was found that the statins were

associated – Taking statins was associated with, in some cases, a 20,000-percent increase in the risk – 20,000 percent. It's not a very common condition, and it's not a notifiable disease if people aren't looking for the association.

Just yesterday, I had someone write to me saying that their father took statins and developed amyotrophic lateral sclerosis. He's now dead. I think there was an association. You, like me, have this frustration of going, "Well, in an individual case, I can't say this is true. All I can say is I know it increases risk. Did it cause it in your father's case? I can't say that, because I can't. I have to say I don't know. And I know it will never stand up in court." But these are major, major problems.

Our bodies need cholesterol. Our neurons need cholesterol. Our brain synthesizes cholesterol in cells, specific cells. They put it in your myelin sheathes, which are the things that protect the neurons. Without it, who knows what's going to happen? There's definitely an increase in Parkinson's disease and other neurological conditions. That's been shown in several studies. It's been dismissed. I know it's been dismissed.

In fact, you'll read articles saying, "Statins protect against Parkinson's disease and multiple sclerosis," but that's based on the nonsense thing that in the past, people with higher cholesterol levels were given statins. People with higher cholesterol levels are less likely to get Parkinson's, less likely to get multiple sclerosis, less likely to get neurodegenerative conditions. What you do is you get a group of people who were less likely to get a neurological condition. Give them a statin and they say, "Well, the statins stop them from getting a neurological condition." This is the opposite of science. This is utterly nonsensical.

This is where I'm really worried. It's that people are getting serious, particularly neurological problems from statins, and it's just being dismissed. In fact, people are now – They're trying to set up a clinical trial to give people with multiple sclerosis statins. This is just not going to end well. I think that might have been dropped. We have some really serious problems with statins. They're being just swept under the carpet.

You may be aware of the study that was done in the U.K. saying all statin adverse effects are what we call nocebo effect. Placebo effect is you think it's going to do you good and it does. Nocebo effect is you think it's going to cause harm and it does. When someone says I'm getting muscle pain, they go out and say, "It's just because you thought you're going to get muscle pain." I mean you can use this for all drugs and then say, "Well, actually there's no such thing as an adverse effect of any drug. You're just making it up in your head." This is just utterly ridiculous.

The study itself – I have written about it. I did have a look at it. But I mean, what can you say? With a lot of the statins, you can only look at the evidence from the clinical trials. A lot of the statin trials have what we call lead-in periods, where in one case, in the Heart Protection Study, they removed 36 percent of people from the study in a month of the lead-in period. They haven't said why. They said it was because of people who couldn't maybe take the drug well. The other explanation is anyone who looked like they were going to suffer from an adverse effect was removed from the trial.

We can't find that out because the organization, the CTT Collaboration, won't let anyone see that data. In fact, the last time I said it to them, they said, "We don't want anything – We didn't even have that data in the first place." Yet, they've written papers saying statins have no adverse effects. How did you manage that if you don't have the data?

It's like having a little naughty boy and a broken window and you say, "Did you throw the ball?" "No, no. Not me. No, no, no, no, no, no." "But you're here, there's a hole in the window. I saw you throwing a ball. You must have broken it." "No, no, no." It is constantly frustrating. I have to bite my tongue at times and just not tell them what I really think about them, because I would probably end up getting a libel case against me.

I mean you probably have the same thing. You just think, "How can you people get away with saying this stuff, which is just monumentally ridiculous? Beyond monumentally ridiculous."

JM: Yeah. It all goes back to the income. But there's a concept called pleiotropy, which means that a drug or an intervention could have multiple effects, not just the primary one. Physicians typically prescribe statins to lower cholesterol. You provided some very compelling evidence that lowering cholesterol does not decrease your risk of cardiovascular disease, or, more importantly, death. But there have been some observations that prescribing statins do seem to be providing some benefit not related to cholesterol.

Some people believe it may reduce inflammation. You have a speculation in your book that it may be due to increase in nitric oxide. I have another passable thesis on this, but I'll discuss that after you share with us your insights on the connection between statins and nitric oxide increase.

MK: As you say quite rightly. I mean one of the arguments I use I've never had an answer to is that no study ever has raised cholesterol level seem to be associated with an increased risk of stroke. You say this and people will look at you and go, "You're mad." But this is the truth. But if you give a statin, it does reduce the risk of stroke by a small amount, but it exists as a finding.

You say to people, "Well, if a raised cholesterol doesn't cause stroke, and your lower cholesterol lowers the risk of stroke, how does that work?" Well, it can't work. Something else is obviously going on. That's something else that's going on in my opinion.

Now, statin has a pleiotropic effect as you say. Statins do all sorts of things. In fact, whenever you look at them, they do something. But one of the important things that they do in my opinion, the most important, is that they increase nitric oxide synthesis in the endothelial cells, which, as you know, has both an anticoagulant effect, as nitric oxide is a strong anticoagulant. It causes vasodilation. It increases the health of the endothelial cells. It also stimulates the production of new endothelial cells in the bone marrow.

I think that that, on its own, could explain any benefits you see from statins. Because when you look at the clinical trials as well, the benefits of statins are seen almost immediately, like after a week or two weeks. And then it kind of plateaus out. If it were to do with lowering cholesterol and stopping plaques from forming, it should surely take years to see any benefit, which was actually when the first study came out.

The Scandinavian Simvastatin Survival Study (4S) study was the first of the major studies to come out. Interestingly, it showed no benefit whatsoever for 18 months. The two lines with the placebo and the statin were plot together, then suddenly they split apart at 18 months, which in it and of itself was something that you had to look at for a bit of sense. Scans saying that's interesting. But I do think that there is benefit there. It's from nitric oxide. Because as you know, there are other agents that could raise your nitric oxide level.

JM: Sure.

MK: For instance, ace inhibitors blood pressure lowers, which have a benefit on cardiovascular diseases. It seems to be completely unrelated to their effect on blood pressure, whereas other agents that can lower blood pressure by as much or more, which can actually increase the risk of cardiovascular disease so there is something else going on with all these medications. Sorry I interrupted you there.

[-----40:00-----]

JM: No, no. I'm sorry I interrupted you. But I was just going to say that the other agents could be natural agents like arginine, sertraline or fermented beets, which absorbs nitrate that the microbiome converts. But I'm wondering if you're aware of the mechanism that the statins impair or actually increase nitric oxide production. Is it in the endothelial nitric oxide or is it inducible nitric oxide, iNOS? What enzyme are they facilitating or augmenting?

MK: It appears to be associated with the renin-angiotensin system.

JM: Interesting.

MK: Because if you trigger off the renin-angiotensin system as the way of raising your blood pressure that the body has, you lose fluids. It's a series of complicated mechanisms. But the renin-angiotensin system is quite damaging. I think it's the iNOS effect that it has. Because that's causing more vessels to constrict in order to push the blood pressure up. If you interfere with that process, then that's a problem. That's mediated through an enzyme called bradykinin.

What statins do is they actually upregulate bradykinin or downregulate bradykinin. Sorry. I need to have it in front of me to remember these things. But it's through the bradykinin effect on inhibiting the angiotensin effect on the cardiovascular system. I was getting a bit technical about that. But that, I believe, is how it works.

JM: Yeah. I'm sorry to have to pass you up for some of the details, but it is a very intriguing concept and one that is not commonly promoted. Thank you for explaining that. I want to run my theory past you to get your thoughts on it. As you mentioned, we all know statins are designed to lower cholesterol. They do that by inhibiting an enzyme in the liver, 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase (HMG-coenzyme A reductase), which is responsible for making cholesterol, which converts to a lot of almost all of your steroid hormones, including vitamin D. That has problems. And also CoQ10.

But as you said, it's going to lower the cholesterol production in the body. One of the things that your body uses in addition to this enzyme, to making these fatty acids is another coenzyme called nicotinamide adenine dinucleotide phosphate (NADPH), which is in the family of nicotinamide adenine dinucleotide (NAD) coenzymes. NADPH is just crucial for your body. It's essentially the battery of your body stores electrons and serves as a reservoir to recharge your antioxidant system. It's like glutathione. Once you utilize glutathione once, it's dead, unless you recharge it, and NADPH is what recharges it.

If you're lowering cholesterol, which is not good, but one of the artifacts of that, maybe you actually increased NADPH, which is good. It may counteract some of the benefits. I'm wondering if you have any thoughts on that.

MK: The thing I do know is that the more I look at this area, the more I think, "Gosh, it's so complicated."

JM: Yeah.

MK: You'd think, "How on Earth could all of these ever got together and worked?" But I would say that I, like yourself, you could look at something and say, "Is this likely? That's likely. Could it be the cause? It could be the cause." It's very difficult to isolate out when you've got so many things going on. But definitely, NADPH, I do remember from – Was that my Krebs Cycle or is that another cycle?

JM: No. It's just recently gotten a lot of notoriety. It's been around. We've been aware of it for 70 years, but since this massive increase in longevity and discovery of sirtuins and NAD as a substrate for that, there's been a lot of increased interest in that. And the recognition that NADPH has a lot of other roles – It's primarily used in the body for the biggest consumers – fatty acid synthesis. Cholesterol's a subset of that. It is just massively important and probably almost every bit as important in NAD+, which gets a lot of press.

MK: Yeah. I think that when you ask things like this, I like to say, "Keep an open mind," because goodness knows.

JM: Right.

MK: What I do know is – What I believe I know for certain is the cholesterol-lowering effect of the statins is an unfortunate side effect. Other things that they might do. The problem is, over time, as you know, you block all these systems and your body starts to find ways around them. You can get damaged. The body doesn't produce cholesterol for fun. It's absolutely essential. It's absolutely an essential part of how we operate.

When I think of any of the secondary effects, like NADPH or nitric oxide, they could be beneficial by accident. I mean a lot of drugs, as you know, have been discovered. I mean aspirin itself was an anti-inflammatory and painkiller. Then it was found to reduce the risk of heart disease, although that's now being questioned. Now it's being used to protect against cancer. All drugs, when you start looking at them, you think, "Oh crikey. I never realized it did that. Blimey, I never realized it

did that." But I suppose the central question here is, "Could it be something else? Could it be a pleiotropic effect?" While I believe that it must be – I wouldn't say I'm going to battle you to death over saying which pleiotropic effect is more important.

JM: Yeah. I was just –

MK: I'm not bullying if you'd like em. But the whole nitric oxide thing is of interest to me. It is an area that I think –

JM: So, the bottom line is there may be some other pleiotropic benefit for taking statins unrelated at all to lowering cholesterol. But it's an expensive and dangerous way to get those benefits when you can easily achieve it, which is – I've got another softball question for you about a study that was published last week, which I'm sure you've heard of it.

But before I get there, heart disease, at least currently, is the No. 1 cause of death in the Western cultures. It's a big issue. That's why this is so important, because it's an absolutely perfect model for these types of drugs, because they do nothing to decrease. It's a lifelong prescription. But it's interesting.

I'm interviewing Chris Knobbe, who wrote a book on age-related macular degeneration. He did an extensive review of all the medical textbooks over the last 100 years and was able to document really precisely that prior to about 1930, age-related macular degeneration didn't exist. We think it's been around forever. He was able to nail it. This is an important tangent. He was able to nail it down to the introduction of processed foods, that when you eliminate the processed foods or the cause, it tends to disappear.

I think, similarly, in 1900, heart disease was an anomaly. It was a rare event. Now, it's an epidemic. Half the people are dying from it. The answer certainly isn't a magic pill. It's not a magic supplement. It's looking at the diet, at the foundational issue of correcting this thing, correcting the reason why people are dropping like flies from heart disease. It's not because of the cholesterol. It's because of these other issues.

MK: Absolutely. In fact, one of the interesting things about macular degeneration – I know it's going to go off on a slight tangent because I was looking at the use of Avastin, which is used for macular degeneration, which is, as you know, is a vascular endothelial growth factor inhibitor.

But interestingly, if you use that treatment, the vascular endothelium growth factor inhibitor, it destroys nitric oxide synthesis in the endothelium. It enormously increases the risk of dying of heart disease. In fact, I've just been looking at this area. Over a two-year period, if you used Avastin – and that's for cancer treatment, not for macular degeneration – it can increase your risk of dying of heart disease by up to 1,200 percent.

JM: But don't they use the Avastin for age-related macular degeneration in small amounts in the back of the eye? I don't know how they get it there.

MK: Yeah. They do. They inject it into your eye, which never sounds like a lovely thing to do. But even when you do that – They did the experiment on rabbits, where they injected it in the eye, then they looked at nitric oxide synthesis around the body, and it reduced it by about 70 percent in most organs.

JM: Wow. That's crazy.

MK: It really knocks you on the head. Again, you want to know these things, and everything becomes associated with everything else, doesn't it? The whole concept of what's going on in the body and what we're actually achieving – You're right. It's about healthy things, like exercise, sunshine –

JM: The basics.

MK: The basics. Maybe one or two supplements, a bit of vitamin D and C here or there to make sure that you're giving your body all the nutrients it needs, because I think we've mangled them out. We've stripped them out of food with the processing, and then we put them back in in weird fashions. It probably means it doesn't really work that way. It probably only works if you eat food that's got all these things in in the proportions and the associations that we were designed to eat.

When people say, "What should you eat?" I just say, "Well, eat something that looks like food. If it takes you more than two minutes to read the ingredients on the side of the packet, throw it away. Because it's not really going to do you any good, is it?"

[-----50:00-----]

JM: I want to ask your opinion about a study that was published last week in The Journal of the American Medical Association (JAMA), a really prominent U.S. medical journal. It was really popular in the media here. I suspect it was on your side of the ocean also. That was the definitive study on eggs, finally, that excluded all these other variables and was able to show that for every 300 milligrams of cholesterol, dietary cholesterol, which is about what you get in one egg, your increase of heart disease or cardiovascular disease went up by 17 or 18 percent. I've got my views, but I really am curious to hear what you were saying.

MK: Well, the first thing, as you know, we're talking about an increase by 18 percent. That's a relative increased risk, which is – I have a personal philosophy, which is unless your hazard ratio – they call this hazard ratio, which is the thing they're talking about is increased by 17 percent. Unless your hazard ratio goes up by more than 200 percent in what they call an observational study, we should just take this study, you should grab it in both hands, crunch it up into a small ball, and you should throw it over your shoulder into the bend. Because it's absolutely worthless.

It wasn't me that came up with that figure. The person who works to discover that smoking can cause lung cancer, two people in Britain – I think most people knew it already, but they kind of proved it. Richard Doll was one of them, and Bradford Hill, who created this thing, of course, standards for causation. They wrote a paper saying, "If you've got an observational study and your hazard ratio is not greater than two, you really can't place no reliance on this whatsoever. There are so many variables that are going on." I mean someone sent me this study. In this study, what

they found was people who had never smoked were more at risk of dying of heart disease than people who smoked.

When you look at the study that's finding results like this, you realize to yourself that this is just a nonsense study. It's not even worth looking at. I have looked at it. As you may know, it takes a long time to go into studies like this and say, "Well, what did they do? What did they not do? What did they study and what didn't they study? How did they do this?"

I'm giving a talk next week, actually, where I'm talking about "Doctoring Data." It's just saying, "How do you analyze this study? What do you look for?" I say, "The first things you start looking for are, A, what's the difference we're looking at here? Could it be something else that's causing it?" Really, when we're looking at hazard ratios of 1.17, really, forget it. This is nonsense. The number of variables that could be causing this difference are enormous, and they didn't even account for most of the variables.

I can't tell you how rubbish that study was, because you have to go into the statistics and all the other things. But essentially, the finding was irrelevant and trivial. Just forget it. Don't worry about it. Forget about it. It was Ancel Keys himself who started the entire diet heart study nonsense, who fed volunteers up to 50 eggs a week, alright? What he found was he couldn't make any difference to their blood cholesterol level. He came to the conclusion – and he was quoted – I have this quote. I have written it down many times. "Cholesterol in the diet makes no difference to you unless you are a chicken or a rabbit." We are neither. We're human beings.

Your body can deal with wide, wide variations in cholesterol intake. Your body produces about 5 grams – No, it doesn't. Yes, it does – 5 grams of cholesterol a day. You can't get that in the diet unless you eat about 12 eggs. The idea that the body can't go with 300 grams of cholesterol, you can't control that. It goes against all known concepts of homeostasis.

What happens if you eat more cholesterol? Your body produces less cholesterol because it doesn't need to anymore. Perhaps you can overwhelm it, but I have seen studies where people have tried to overwhelm. What happens is that they're in the gut. There's a kind of shuttle system. Once your cholesterol levels are full and you don't need anymore, it doesn't absorb cholesterol anymore. It just shuttles it back out again. It just goes straight through you and out the other end. Your body can control these things. And the idea that some minute amount of added cholesterol is going to overwhelm your control systems, it just goes against all known human physiology. It's just complete nonsense, because that's what I think about it.

JM: Thank you for sharing your insights on that. The fact that 1 out of 4 adult Americans over the age of 40 is currently taking a statin is something I call TOS or tragic on steroids. I mean they've been able to convince, manipulate and deceive a massive millions, tens of millions of people into taking these drugs.

MK: I know. It's an idea. I think it was one of your journalists, H.L. Mencken who said, "For every complex problem there is an answer that clear, simple and wrong." This is the cholesterol hypothesis. It's simple. It's easy to understand. Anyone can understand it. As I say, a 5-year-old can understand it, which is why it shouldn't really be believed. It's just wrong. When you look at

it, well, what's supposed to happen? How is this supposed to cause heart disease? It just starts to turn into mush in front of your eyes.

I mean it took me quite a long time to work out it was rubbish. I listed this for many, many years. I realized the dietary product was nonsense pretty quickly, because it just couldn't make any sense. It took me a while to get rid of the raised cholesterol bit, because, well, "You get cholesterol in your arteries and cholesterol in your blood and di-da-di-da-di-da." It all sort of makes kind of sense if you don't think about it too deeply. Once you start looking at it, you think, "This is just preposterous."

JM: Well, what's confusing is the cholesterol in the plaque too.

MK: I'll tell you an interesting thing about cholesterol in the plaque. It was first noted by Rudolph Virchow in 1852. What he saw was cholesterol crystals in the plaque, because he couldn't see cholesterol at that time. There weren't sophisticated – "There's cholesterol in the plaque, that's interesting."

Now, the cholesterol crystal, the interesting thing is cholesterol crystal, you can't get a cholesterol crystal from the cholesterol that's carried around in LDL. Because the cholesterol that's carried around in LDL is what we call the cholesterol ester. It's a cholesterol attached to a fatty acid. Two molecules stuck together, that's what they call cholesterol ester. You can't make a crystal from that.

In fact, I've been reading papers recently that have agreed with this. Cholesterol crystals can't come from LDL, so where does it come from? The only place we could get a cholesterol crystal from in the body is from the membranes of red blood cells, because the membrane of a red blood cell contains more free cholesterol than anything else in the body. It's the only place you can make a crystal from.

If you want to say, "Where does the cholesterol crystal come from? Where does the cholesterol come from?" Well, it comes from red blood cells. "Where do red blood cells come from?" Well, it's got nothing to do with LDL. We know that.

The other interesting fact is that when we find things that look like LDL, they're almost certainly not LDL. They're almost certainly another lipoprotein called LPA, lipoprotein A, because lipoprotein A and LDL are exactly the same thing, except LPA has another protein attached to it called apolipoprotein A stuck to it. It floats around in the body and everyone says, "We don't know what it does," but we know what it does. Because apolipoprotein A is identical in structure, apart from one amino acid, to plasminogen. Plasminogen, as most people don't know, is a thing incorporated into our blood clots as they form. Plasminogen can be turned into plasmin by tissue plasminogen activator, and that's what splits clots apart.

However, if you have a clot with LPA in it with apolipoprotein A in it, the plasminogen activator cannot work, and that clot cannot be broken apart and it remains stuck where it is. Therefore, when you have LPA involved when you've got arterial damage, then you get a blood clot, you get LPA, you get red blood cells, and then you have a blood clot attached to the side of your blood vessel.

So you have red blood cells, you have LPA, you have almost everything actually. You have a lot of these things. Then you're left with a situation.

"Well, what do you do with this blood clot? What does the body do with it?" Well, it can't fall off and travel down the artery. It would just block the artery further down. So, what the body does with it is that it shaves it off. Then when it shaved it down, new endothelial cells floating around in the blood cover it over, so you then have a blood clot lying underneath of a new layer of endothelium. That blood clot is the formation. The direct result of that over time – if it keeps happening on that spot over and over again – that becomes plaque-containing cholesterol crystals, things that look like LDL, LPA and everything else.

When people say, "What about oxidized LDL?" I say, "Where do you think the oxidation of LDL occurs? It occurs inside the artery wall, because that's the body trying to get rid of it. Because the first thing that the white blood cell does, the macrophage does when it comes across alien material, is it hits it with a superoxide burst, a superoxide burst is nitric oxide. It oxidizes it, then it engulfs it, then it gets rid of it." That's how the system works. When you find oxidized LDL or oxidized anything inside a plaque, oxidation is the body trying to kill you.

[-----1:00:00-----]

JM: Well, wouldn't it be superoxide? Because if it's going to oxidize it, it typically is through NADPH oxidase, or NOX, which releases superoxide. Superoxide can combine with nitric oxide and form peroxynitrite, and that blasts it even more.

MK: Nitric oxide is what macrophages use, at least that's what's on everything that I read.

JM: It's a free radical, but it's not very oxidizing.

MK: Yeah.

JM: Typically. It's more beneficial, especially – Well, I don't know. Macrophages are – Are they made by eNOS or iNOS? It must be iNOS.

MK: I think it's iNOS.

JM: Yeah.

MK: Yeah. But I don't know how you tell the different point to that. But I think that to an extent – I mean, I'm throwing a few different concepts here at you rather quickly. But to an extent, we've looked at the whole process. There have been people kicking around for hundreds of years who have always thought the cholesterol hypothesis was nonsense and have come up with different ideas what's actually going on and what's actually causing heart disease. Yet we can't move away from this cholesterol ideas. It seems we're absolutely trapped with it. As you say, we've managed to convince, around the world, hundreds of millions of people to take statins. I'm not quite sure how we get away from this. I don't know how we move away from this.

JM: Well, spreading the truth. Just a quick question on – Thank you so much for that elegant description of what's happening, because a lot of people are a bit confused with it. But ultimately, it's LPA, which is a far more potent risk factor than LDL.

MK: Yeah.

JM: Assessing that level. If you have these high LPA, essentially, it activates your own endogenous tissue-type plasminogen activator (tPA). The tPA, as many people may know, is what they give in the emergency rooms for people who have an acute heart attack or stroke if it's within a certain timeframe, usually a few hours. If they give it intravenously, tPA, because they're giving it in such massive doses, is it enough to bust through the clot?

MK: I think it is. I think that if you've got a large part of the clot even if it's not about LPA in it, if it does block the artery completely, if you can blow that part, then you're going to blow up the artery again.

JM: Okay.

MK: Does it overwhelm? It probably does overwhelm the amount of LPA in there and just blasts the whole thing to pieces. But I assume that. I don't know if anyone's ever studied that. That's a guess on my part really.

JM: Well, this has been great. Are there any other pearls you'd like to share from us from your magnificent book?

MK: Well, I think, as you probably know, we're very much in the same line in many things. I think that one of the pearls that the strongest risk factor for dying from heart disease is actually mental stress and mental illness by a huge, huge margin.

JM: You just wrote a blog post on that.

MK: Yeah. I've done a blogpost on that. I think that if you look at populations who have suffered really, really badly from heart disease, they tended to be under a great deal of psychosocial – if that word means anything – psychosocial stress. It did point out the Australian aboriginals, whose culture and whose lifestyle has been obliterated. I mean they don't eat very healthily and they smoke and they take drugs and they drink. But even taking all these things aside, an Australian aboriginal woman will have a rate of heart disease that is 1,000-percent that of the surrounding population.

I think when we're looking at heart disease, I think we have to look at mental effects as well, things like friendships, things like having a sense of purpose, things like having a good family around you, are usually important. I think mainstream medicine has gone off and sort of ignored this really. It's just a drug fest.

But I think that the mental and physical health, which I know is something you promote and promote, is just absolutely critical to what you call true health or proper health, not health coming

out of tablets and pills, which is just not the right way of getting people back on track. That would be my – I don't know if that's a pearl.

JM: Well, a subset of that too would be social community. The fact that – I think there's been some studies that point it out pretty clearly – that loneliness is another major factor. It's really sort of a subset of psychosocial stress.

MK: Absolutely. Yeah. Recently, even politicians have noticed that. Especially for elderly people, lack of social interaction and loneliness is deadly. I think they call these the diseases of civilization – what cancer and heart disease were used to be called. But in many ways, they are, because we live so differently than we used to, and it's the culmination of all these things: not moving and not eating what we used to, not having the social interactions that we did, and people become more isolated. It's a terrible kind of series of things that when you added one on top of the other, people find it difficult to deal with.

JM: Yes. Thank you so much for your time and writing your book. The book, again, the name is "A Statin Nation." It should be coming up very shortly. If you're – I think we'll put a link to it on the site, because there's actually another book that's called "Statin Nation," which was written prior to yours. You have to be careful and pick the right one.

MK: Yeah. I didn't want to call it "A Statin Nation." The publishers insisted. They wouldn't let me call it anything else. There we have it then. "A Statin Nation." Thanks very much, Joe.

JM: My very first book in 2004, literally 15 years ago, was "The No-Grain Diet." That was a name dictated by the publisher. I was so annoyed for many, many years about it. Because I didn't necessarily believe that at the time. But now I've come full-circle and I think, "Yeah. That's a great idea. I'm so glad it has a name." Maybe in another 10 years, you'll be just delighted with that.

MK: I don't like to put that name. Well maybe, you never know.

JM: Well, again, thank you so much. I really appreciate your commonsense wisdom, basic rational approach in helping us decipher these complex manipulations that industry uses to deceive us and really convince hundreds of millions of people around the world to go on these dangerous medications that don't do a darn thing to improve their long-term risk of dying from the disease that they're prescribed for.

MK: I know. Thank you, Joe. That's great. I hope people have got something from it.

JM: Okay.

MK: Cheers.

[END]