Cholesterol & heart disease - there is a relationship, but it’s not what you think.

by Zoë Harcombe
I do a fortnightly newsletter called “Diet & Health Today” in our online support club. The club is there to help people to lose weight by eating food – real food! Apparently that makes us radical and controversial. The main article in Diet & Health Today is called “The Big Issues” and we have tackled things from “how we have misapplied thermodynamics to weight loss” to “what role does exercise play in losing weight”.

This is a copy of possibly one of the most serious Diet & Health Today articles that I may do. It is dedicated to Anne (Annem in the club) who asked me a great question about cholesterol. It made me do what I had been meaning to do ever since I read Dr Malcolm Kendrick’s The Great Cholesterol Con...

Dr MK ran some analysis on World Health Organisation (WHO) data. The WHO has extensive data from almost 200 countries on more health measures than you could imagine – definitely worth a look one rainy, wintry afternoon. This is where Dr MK presented the world with two different Seven Country Studies – (for those of you who aren’t familiar with the history, it was the Ancel Keys’ Seven Countries Study that started all the fat heart hypothesis stuff). Dr MK took the seven countries with the lowest saturated fat intake and then the seven countries with the highest saturated fat intake. You may need to read this twice – but he found: “Every single one of the seven countries with the lowest saturated fat consumption has significantly higher rates of heart disease than every single one of the countries with the highest saturated fat consumption.”

The next chapter in The Great Cholesterol Con goes on to look at cholesterol and heart disease (and overall death rates) and quotes many great studies where it is shown that lower cholesterol is associated with higher mortality. However, it did leave me thinking – having run the data on saturated fat and heart disease, let’s just run all the data on the cholesterol and heart disease and get to the bottom of this hypothesis from all parts of the allegations.

It actually didn’t take that long – less than a couple of hours one Saturday afternoon. You go to the WHO statistics area of their web site and then pick data for cholesterol from risk factors (how judgemental to start with!) and then look under: Global burden of disease (mortality); All causes; Non communicable diseases and then G Cardiovascular disease (shortened to CVD). CVD deaths include ischemic heart disease and cerebrovascular disease – that means fatal heart attacks and fatal strokes to us. You find the most recent year where you can get both sets of data to compare like with like. This turns out to be 2002. You download their very user friendly spreadsheet data (CSV) – cut and paste it into an excel file and then try to remember how the heck to do scatter diagrams in excel!

Before telling you the results, we need to go back for a quick reminder on what we know about cholesterol and then hopefully this can serve as a factsheet for all the cholesterol questions we continually get.

**The role of cholesterol**

It is virtually impossible to explain how vital cholesterol is to the human body. If you had no cholesterol in your body you would be dead. Every single cell of your body is covered by a membrane (think of a membrane as the ‘skin’ or protective barrier around each cell). This membrane is made largely of cholesterol, fat and protein. Membranes are porous
structures, not solid walls, letting nutrients and hormones in while keeping waste and toxins out. If cholesterol were removed from cell membranes they would literally explode from their internal water pressure. Human beings quite simply die without cholesterol.

Cholesterol is vital for hormone production – the sex hormones and therefore the entire human reproductive system are totally dependent on cholesterol. Hence, not only would humans die without cholesterol, the human race would die out.

Cholesterol is vital for digestion. The human body uses cholesterol to synthesise bile acids. Without cholesterol-rich, bile salts, the human body could not absorb essential fatty acids or the fat soluble vitamins (A, D, E and K) and serious, even life threatening, deficiencies could develop. (It is interesting, therefore, that nature puts cholesterol in virtually every food that contains fat – providing a digestion mechanism in tandem).

Cholesterol is vital for the brain, central nervous system and memory functions (hence how the side effects of statins include memory loss, mental confusion and people generally just not feeling themselves). Even though the brain is only 2% of the body’s weight, it contains approximately 25% of the body’s cholesterol. The vital connections between nerve endings in the brain, which help to conduct the electrical impulses that make movement, sensation, thinking, learning, and remembering possible, are largely made up of cholesterol.

Cholesterol is critical for bones and for all the roles performed by vitamin D. Vitamin D is best known for its role in calcium and phosphorus metabolism, and thus bone health, but we are continually learning more about potential additional health benefits of vitamin D from mental health to immune health. Vitamin D can be ingested (and is, interestingly again, found in foods high in cholesterol) and it can be made from skin cholesterol. Modern ‘health’ advice to avoid the sun, take cholesterol-lowering drugs, eat a low cholesterol diet (whatever the heck that is supposed to be) – combined with there not even being a recommended dietary allowance for vitamin D – is undoubtedly contributing to avoidable modern illness.

One of the key reasons that we need to spend approximately one third of our lives sleeping is to give the body time to produce cholesterol, repair cells and perform other essential maintenance.

This gives you the headlines of the vital functions that cholesterol performs, but hang on to that bottom line – it is utterly vital and we die instantly without it.

You may be familiar with the term essential fatty acids or essential amino acids (proteins break down into amino acids). The term ‘essential’ used like this in nutrition means that it is essential that we consume it in our diet because the body can’t make it. The body makes cholesterol. That says to me that cholesterol is even more vital than essential fatty acids or essential amino acids – even though these too are life critical – and therefore the design of the human body is such that it was not left to chance that we needed to get cholesterol from food. Of the 500 or so roles that the liver has – one is to produce cholesterol. It is too vital to be left to chance.

What went wrong?
So, how did something so life vital become
more vilified than a mass murderer? I think it comes down to three things (and I don’t take credit for this view – it is there to be worked out by anyone who traces back the history and Kendrick, Uffe Ravnskov and all the things.org guys have led the way):

1) Rabbits;
2) Ancel Keys;
3) Money!

1) In 1913, a Russian chap called Nikolai Anitschkow decided to feed rabbits purified cholesterol and he managed to get their blood cholesterol levels in excess of 1,000 mg/dl (nearly 26 mmol/L! Most UK people have levels of 5-7 mmol/L). He then noticed the formation of “vascular lesions closely resembling those of human atherosclerosis” forming in the arteries of the rabbits. The obvious flaw in the experiment should have been that rabbits are strict herbivores. They do not eat animal products, which is the only source of cholesterol. Hence rabbits are in no way designed to digest cholesterol or animal fat and no one should be surprised if cholesterol or animal fat ended up stuck in any part of the poor rabbit. The only surprise is that no one thought to ask Anitschkow why he was feeding cholesterol and animal fat to herbivores. Interestingly, far less well known is that a parallel test was done on rats and dogs (omnivores) and feeding cholesterol to these species failed to produce lesions.

2) Ancel Keys. Remember the Minnesota experiment that I so often refer to? A brilliant and unbiased piece of research, which has given the world one of the best insights into low calorie dieting ever done – it was pure genius. This study made Ancel Keys the man of the moment and I guess he wanted to follow it with something equally impactful. There is an anecdote in The Great Cholesterol

Con and on p113 of The Obesity Epidemic where Henry Blackburn, one of Keys’ closest colleagues, tries to explain what may have fuelled Keys drive to find a connection between diet and heart disease.

What is little known is that Keys originally tried to establish a link between cholesterol in food and cholesterol in the blood (our cholesterol levels when we have a blood test) because he thought (probably because of poor Bugs Bunny) that cholesterol in the blood causes heart disease.

Keys did multiples of studies, changing the diets of his human ‘guinea pigs’, and he presented his conclusions in The Journal of Nutrition, November 1955: “It is concluded that in adult men the serum cholesterol level is essentially independent of the cholesterol intake over the whole range of natural human diets. It is probable that infants, children and women are similar.” i.e. I only tested adult men and there is no relationship between cholesterol eaten and cholesterol in the blood and it is probable that there will similarly be no relationship for women or children.

In 1997 Keys put this even more assertively: “There’s no connection whatsoever between cholesterol in food and cholesterol in blood. And we’ve known that all along. Cholesterol in the diet doesn’t matter at all unless you happen to be a chicken or a rabbit.”

Did you know – even the UK Food Standards Agency (FSA) and UK National Health Service (NHS) admit this?
- “However, dietary cholesterol has little effect on blood cholesterol. More important is the amount of saturated fat in your diet”. (National Health Service). (Notice the second sentence? They just couldn’t let the theory go).
- “But the cholesterol we get from our food has much less effect on the level of cholesterol in our blood than the amount of saturated fat we eat”. (Food Standards Agency). (This link may disappear, as the FSA is bowing out of giving nutritional advice).

What the government advice should say is: The body makes cholesterol. The cholesterol you eat has no impact on the level of cholesterol in your blood – not “little”, but “no” – (and we’ve known that all along). And they should also explain how saturated fat can determine blood cholesterol levels and then provide irrefutable evidence that it does. But it must be hard for public health bodies to even go this far. As we saw in a recent thread – the FSA also now accept that there is no limit on the number of eggs we can eat:

If only Keys had stopped here, but he wanted to find an explanation for heart disease and he was not about to be deterred. For some reason, which I find inexplicable, he then turned to fat (the entire literature on this topic is very vague about “fat” vs. “saturated fat” so his early writings are also very vague on the topic). Here’s a bit of Mensa logic for those who like this kind of thing:

i) Only animal foods contain cholesterol (meat, fish, eggs, dairy). NO non animal foods contain cholesterol.

ii) All animal foods contain fat – saturated and unsaturated. Some may be very low in fat (e.g. white fish), but they all contain some fat.

iii) If there is no link whatsoever between increased consumption of foods containing cholesterol and blood cholesterol levels, there can be no link whatsoever between increased consumption of animal foods and blood cholesterol levels since only animal foods can be increased in consumption to increase consumption of cholesterol!

So, Keys first did the graph that was presented at the Mount Sinai hospital (which is the one shown in the Tom Naughton video and in Dr Robert Lustig’s “Sugar: The Bitter Truth”) and then went on to do the Seven Countries study – which I have read all twenty volumes of and take apart piece by piece in Chapter Eight of The Obesity Epidemic: What caused it? How can we stop it (on this page).

As Kendrick’s two unbiased seven country studies showed – there is not even an association between saturated fat and heart disease – let alone a causation. However, Keys published his seven countries study and the rest, as they say, is history.

3) The Robert Redford film All the Presidents’ Men that had the memorable quote “follow the money”. This is absolutely at the heart of everything in the diet industry from national dietary organisations to the food, drink and drug industries and individuals in between.

The Ancel Keys work interestingly claimed that saturated fat consumption (A) caused heart disease (C) not directly, but by raising cholesterol (B). Hence A was supposed to cause C through B. For this to even get off the starting blocks, A and C have to be related (plot one against the other and there has to be a clear relationship); A and B have to be related and B and C have to be related. None of these in fact holds. The Kendrick study shows that A and C are not related. There is no logic that A and B could be related – because of the problem of fat and cholesterol being found in the same foods and Kendrick presented many studies that showed B and C were not related. I aim in this article to put the nail in the coffin for any idea that high
cholesterol is even associated with high heart disease. We will, in fact, show that the evidence confirms the opposite.

By having cholesterol as this middle-man, this has allowed an entire pharmaceutical industry (and stupid cook books) to come up with ways of lowering cholesterol. The most lucrative of these has clearly been statins – drugs designed to stop the body producing the cholesterol that it is designed to produce. It never hurts to remind people that one statin alone, Lipitor, is worth $12 billion to Pfizer. Taubes has a deeply troubling passage in The Diet Delusion where he looked at the committee who approved a lowering of the target cholesterol levels for the USA population. From memory (it’s a big book to find a reference!), a number of people were on the committee and all but one were funded by pharma companies and one didn’t want the target cholesterol level lowered. I wonder which one! (Anyone reading this – if you can find the page number I’d be so grateful – my copy has so many scribbles on I can barely read it).

So, cholesterol will remain the mass murderer for as long as statins are as lucrative as they are or until the public are enlightened and courageous enough to say no to doctors who try to put them on this medication (like my mum was after reading Dr MK!)

A small technicality
On p35 of The Great Cholesterol Con, Kendrick says: “How can eating saturated fat raise LDL levels? It is not merely biologically implausible, it is biologically impossible. Boy does that statement make me a hostage to fortune!”

I arranged to meet a biochemist at a local university to try to get to the bottom of this statement. The biochemist (who has more qualifications than I’ve had dark chocolate) was sadly so brainwashed in the ‘fat is bad’ theory that he just kept saying eating fat raises cholesterol. When I asked him to talk me through the biochemical pathway from fat digestion through to how this impacts cholesterol he said he didn’t know the digestive process well enough – we would need to add a dietician into the conversation. This was alarming enough. I then said – we eat 39 grams of butter per person per week in the UK and about 1.4 kilos of flour – didn’t he think it was more likely that the flour was making us fat and sick. He said it only took a drop of arsenic to kill us. I left shortly afterwards.

Kendrick has to be right (isn’t he always?) LDL (remember this is not cholesterol – it is a low density lipoprotein) is the left over from IDL (intermediate density lipoprotein), which is the left over from VLDL (very low density lipoprotein). VLDL is one of the measures you get in your blood cholesterol test (actually they estimate it – they don’t measure it – they only measure total cholesterol and HDL leaving two other unknowns in an equation with four variables and you thought this was scientific). (They also call VLDL ‘triglyceride’, which is confusing and unhelpful). Cutting a complex story short (it is explained in my book in different passages), carbohydrates can impact VLDL levels (starter for 10: Acetyl-CoA being the start of the process by which the body makes cholesterol and part of the Kreb’s cycle whereby the body turns glucose into ATP), but I really have found no way in which the fat that we eat can do so. Because fat is not water soluble, it is packaged into a lipoprotein in the digestive system. The lipoprotein that fat goes into is the biggest
one – the chylomicron – and then it travels off into the body to go and do the essential repair and maintenance jobs that fat does. Does the fat say – hang on Mr chylomicron – we need to go via the liver and see if we can mess up the body’s VLDL production in some way?! Do ask this ‘how’ question (in detail) of someone who thinks that this is possible. I am still open to someone answering this, but I’m not holding my breath.

Fructose, on the other hand, we do know goes straight to the liver to be metabolised. Could that, and other carbs, impact VLDL production? The evidence I have already seen is strong that they do.

The serious bit
The WHO data is split into men and women. I first did the scatter diagrams for average (mean) cholesterol levels and CVD deaths.

Then I ran the Pearson correlation coefficient on these numbers. This gives us the term called “r”. “r” tells us if there is some kind of a relationship: an r score of 0 would indicate no relationship; an r score of 1 would indicate a perfect relationship. A negative r score is called an inverse relationship e.g. the price of concert tickets is likely to be inversely related to the number of concert tickets bought – fewer tickets being bought at higher prices.

The “r” score for men revealed that there was a small relationship of 0.13 – however this relationship was inverse. The diagram and correlation shows that higher cholesterol levels are associated with lower CVD deaths and lower cholesterol levels are associated with higher CVD deaths. In women, the relationship is stronger – to the point of being meaningful. The r score was 0.52 – but, again, inverse. For women, higher cholesterol levels
are quite significantly associated with lower CVD deaths and lower cholesterol levels are quite significantly associated with higher CVD deaths. Please note that I have added r squared on the graphs below (excel can do this for us) and it can confirm that you’ve got your r numbers right and r squared tells us the strength of any relationship we have observed.

All you need to do is to look at the lines going down to the right and wonder how on earth we ever got away with telling people that cholesterol causes heart disease. High cholesterol is associated with lower heart disease and vice versa – for all the data available in the world. High cholesterol is not even associated with high heart disease, let alone does it cause it.

It gets worse. I then kept the cholesterol information and changed the death rates to total deaths – all deaths from any cause – cancer, heart disease, diabetes, strokes – all deaths. You can see the diagrams for men and women again below. This time there is a significant relationship for both men and women: 0.66 for men and 0.74 for women – again inverse. There is a significant association between higher cholesterol levels and lower deaths and lower cholesterol levels and higher deaths for men and an even more significant relationship for women.

This is serious. I’ve shown it to a couple of academics (Professor sort of things) with whom I’ve been having great debates, as I want to see what the view is from people who wholly believe the fat/cholesterol/heart/death hypothesis. (Kendrick talks in his book...
about what happened when he showed an intelligent colleague his two seven countries studies and the evidence was just dismissed instantly). It is most useful to know what the resistance arguments will be before starting to invite the resistance. The two arguments I got back were:

1) “Ah yes – but this is only an association.”

Ah yes – but
a) we changed global dietary advice back in 1977-1983 on the back of an association in Seven (carefully hand picked) Countries that miraculously became a causation even when the association was far from established and b) it is an association that’s the opposite to the one that the world currently holds true and c) that’s what epidemiology is supposed to be about – establish an association and then investigate if there could be any causation or useful learnings. So – go out with a new paradox – that high cholesterol is associated with low deaths and then see what dietary advice emerges.

2) “But that’s total cholesterol – the key thing is the ratio of good to bad cholesterol.”

Oh boy! The chemical formula for cholesterol is C27H46O. There is no good version or bad version. HDL and LDL are not even cholesterol, let alone good cholesterol or bad cholesterol. They are lipoproteins – see above – and they carry cholesterol, triglyceride, phospholipids and protein. Do you think that taxis are people? Or do you think that they are carriers of people (and luggage, and pets and fresh air and other things).

Back to – this is serious. Unless you have familial hypercholesterolemia (a hereditary condition suffered by c. 1 in 500 people), my personal view is that you can do real harm by lowering your cholesterol level – as measured by CVD deaths and total deaths. (New note – Uffe Ravnskov has emailed me to say high cholesterol is not a risk factor for people with FH either – I am sure he will be right. I’ll look at the papers he referenced asap and the “unless you have...” may disappear).

The doctors’ Hippocratic oath is “First do no harm”.

This also says to me – even though saturated fat has nothing to do with cholesterol, it doesn’t actually matter. Even if it did – cholesterol is only associated with CVD deaths in an inverse way. If fat did raise cholesterol – as public health officials like to claim – it could save lives! Please note I am always really careful with language in this area and never jump from association or relationship to causation. Someone may be in the bath and they may be singing – if we observe this in many cases, we may claim that there is an association. We cannot say that bathing causes singing or that singing causes bathing.

Our global dietary advice was changed in 1977 in the US and 1983 in the UK as a result of a biased study of seven handpicked counties. Had the data been available for the 192 countries we can analyse now, or had Keys even considered all the data that was available to him at the time (for France etc), our conclusion may have been that we need to protect cholesterol levels in the body. We may have realised that the last thing we should be trying to do is lowering cholesterol – unless we’re trying to lower life expectancy for some reason.

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About the Author

Zoë Harcombe is an author, researcher, speaker and founder of The Harcombe Diet®. She has 5 books published: ‘Why do you overeat? When all you want is to be slim’; ‘Stop Counting Calories & Start Losing weight: The Harcombe Diet’; The Harcombe Diet Recipe Book’; ‘The Obesity Epidemic. What Caused it, How can we stop it?’ and ‘The Harcombe Diet for Men: No more Mr Fat guy!’.

All her books and further information about her work are available at ZoeHarcombe.com.

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