Danny Lennon: A big welcome to the podcast to professor Norman Temple. How are you doing today?

Norman Temple: I am doing very well today. How are you doing and greetings from beautiful Vancouver, British Columbia, on the Pacific Ocean.

Danny Lennon: I'm sure that it is beautiful as you describe. I'm very much looking forward to this conversation as and as I think I've mentioned to you in some of our correspondence, some of your work has been very thought provoking reading to me and a number of the topics touch on themes that some of our regular listeners may have heard discussed on this podcast before and have been really quite intrigued by some of the ideas you've presented.

But before getting to any of that. Can you maybe walk us through your background in nutrition science and maybe some of the big overview things that might be relevant for people to be aware of?

Norman Temple: Well, I was always interested in areas of health and medicine and disease and what causes disease. And I to get further into it, I did a degree and a PhD in biochemistry, and I was of the opinion as are most people staff who studied biochemistry, that this is the secret for understanding disease. But as I went along, I realized it's actually much, much too complex. The body is so super, super complex. We can't manipulate the body by understanding biochemistry the way for instance, a car mechanic can understand how a car engine works, manipulate the car engine and make it work better. The body is

so much more complex and what is a much more fruitful way to understand health and disease prevention and treatment is nutrition, also related lifestyle things; don't smoke, exercise and so on. But for many of these areas, nutrition, understanding the food we eat, its relation to whether you get sick or not this is of tremendous importance. So I moved into over the years into nutrition and health, and I've been working in that area for many years, really since the 1980s. So going right back to the 1980s, I've been very interested in that area.

Danny Lennon: One thing in particular that maybe we can get into is thinking about different types of studies we can look to and different types of areas of research, which actually relates to what you've just said maybe, about: if we focus in on biochemistry and look at these individual biochemical reactions, it may not actually get us down the way of understanding human health as much as we would wish.

And it was in one of the papers you wrote, I think 2015 piece in the Journal of Nutrition and Health Sciences you said, and I have a quote here, which I'll read, you outlined this view that said: "most of our information of practical value in the area of nutrition in relation to health and disease has come from cohort studies and randomized control trials. By contrast, relatively little of it has come from mechanistic research."

But I would love just to open up and let you expand on that particular idea of first of all, why did you want to write this; what was the idea, the main kind of core idea that you wanted to get across to people? And what was the pitfall I suppose you were trying to make people aware of:Of looking in at mechanistic research in a way that might be not that productive.

Norman Temple: I worked for two years at the University of Surrey in Guildford, England. If people haven't realized it yet from my accent, I come from London. And I'd sit through these meetings and people are talking about the cause of cancer. It was a cancer group I was in and it was so very complicated that I found it hard to see how studying this in great detail would lead to a real understanding of the course of cancer and how to prevent it. In theory, it should: that you have all these very, very clever scientists around the world, whose looking at what's going on in cancer, the changes in DNA and the biochemistry, et cetera. And you can say, "ah, this is a malfunction". The way your motor mechanic can diagnose the noise coming out of the engine, what's going wrong and how he can fix it, or she can fix it. But no, this doesn't work with cancer and the same with many other diseases because it's simply too complicated.

But on the other hand, you look at simple studies of the relationship between lifestyle and health. And that is where all the information came from. You've got smoking and cancer: Some British and. American researchers back in 1950, compared people with the lung cancer and people who didn't have lung cancer. And they found the big difference between them was the vast majority, like 95% or more of the people with lung cancer were smokers. And from that we learned that smoking cause is cancer. Likewise, with exercise, it prevents cancer. Being obese is a significant risk factor for cancer and different aspects of the diet.

That is where, if somebody came and asked me, how do I prevent cancer? Well, I'd say to them, "don't smoke. Try not to be too overweight, try to control your weight". Things like that, lifestyle. This is a secret to preventing cancer, whereas. What can I tell them? That's useful from biochemistry, basically nothing that comes to mind.

Danny Lennon: One of the actual examples that I remember you gave in that piece, that ties in with these diet health relationships relates to cancer. And I think you talked about red meat and cancer risk. And this is a really fascinating question to consider, because this is still an area of nutrition science, where there is some degree of open debate, because while the evidence is much clearer around processed meat, for example, if we take unprocessed red meat and its risk for this, there's still more unanswered questions and there's this kind of gray area of, well, how exactly should we well, what conclusions can we actually make for human health and in particular cancer risk?

And I think you kind of talked about, well, if we think about. How we could go about trying to answer that question. What is more likely for us to come to some pragmatic conclusions? Is it looking at prospective cohort studies or would it be from trying to look at every potential mechanism by which red meat could have a link to cancer risk and investigating these from a bottom-up perspective. Can you maybe just touch on that as one specific example the red meat and cancer, because I think it really does a good job of outlining what would be the most fruitful way to try and answer some of these very pragmatic questions for population health

Norman Temple: We have from the cohort studies, there's been these big cohort studies; that means you recruit somewhere between 20,000 and 100,000 people, and you ask them, say, people (in their) thirties, forties, fifties. And you ask them all sorts of detailed questions. How much do you weigh? What's your diet? How much alcohol do you have? Do you exercise? Do you smoke? And then you monitor them for the next 10, 15 years. And then you see who gets

different diseases, who gets diabetes, cancer, heart disease, and so on. And you try to relate the lifestyle that they're living with, the diseases down the road that they end up with and what's come out of this. As you correctly said, processed meat, you get this consistent, strong relationship. And then this is both cancer and heart disease. That processed meat; bacon, ham, sausages, that type of meat will significantly increase risk of heart disease and cancer. Red meat; so lamb, beef - the relationship, what we see is a pattern where there is this relationship, but it's not quite as strong because it's somewhat weaker.

Some studies will show it, but others won't. But if you, what people do because of this variation in cohort studies, there can be any number of reasons for the variation error in measuring people's diet. Maybe doing it in different countries, maybe what's true in a study in Germany is not so true in Australia and so on.

And so you have to take a batting average, so what they do these days is called a systematic review, followed by a meta analysis. So the systematic review means looking for all the studies in this area. So if you wanna study the relationship between meat and the risk of cancer, firstly, you do a search which is called a systematic review to find all the relevant papers.

And then you tabulate the findings. You give more weight to the big studies over the small studies and it's basically... like public opinion polls: "Is Labour (Party) ahead of the Conservatives?" There may be six polls and the numbers are somewhat scattered, but you take weighted average.

They do the same thing here in a meta analysis. What is the relationship between meat and the risk of cancer? And what we find is for processed meat; it's quite strong and it's quite clear, red meat, some studies show it, others don't, but the batting average is yes, there's a relationship. Now getting to the causation. That's an example. Like I said, talking more generally cancer.

What causes it, trying to understand what it is about red meat that causes cancer is a whole lot of guesswork, maybe barbecued meat. When you, if you barbecue red meat, it heats up to a high temperature that causes the formation of nasty chemicals. Maybe there's too much salt in it. Maybe preservatives. We don't really. We can take a lot of guesses, but we don't really know for sure. Which is why that comes back to why we need this simple relationships. What is your lifestyle and what is your risk of disease? You eat a lot of process meat, you get cancer and heart disease. You people have been looking at these things for years and years and years without coming to any clear. I am reminded of the line by Woody Allen in one of his movies where he says "I've been seeing my therapist for 13 years. We're making good progress."

Danny Lennon: Yeah. I mean there's so much within that because even when we look at, for example, the international agency for research on cancer or the IARC that a lot was made when they came out with some of their tentative conclusions around this classification for unprocessed meat as "probably carcinogenic to humans". But of course when you look into that report, that's based on the fact that, well, we don't really have this really clear pattern from nutritional epidemiology as you just outlined. And so we're relying a bit on the mechanistic work, but of course as I think one of the things that you pointed out in some of your writing as well, that if you have this reliance on mechanistic research because of how that's done and the way you would actually want to do it is for a mechanistic study.

If we're looking at a certain exposure of interest or a certain specific mechanistic process, you end up trying to exclude other protective or mediating compounds within that, because that would allow you to assess it better. But of course this gets further and further away from. Overall whole diet patterns, which is actually what we care about.

And so if you have an emphasis on these mechanistic studies, you end up over inflating that relationship between exposure and outcome potentially, but it's certainly not answering a very pragmatic question of if we have a certain diet that looks like this containing these types of foods in these typical amounts, what is that doing for a risk?

It gets further away from that, which I think one of your points is if we want really pragmatic conclusions, then things like cohort studies and, or in combination with RCTs is much more likely to be the way to go than trying to work all the mechanisms out first, which is probably an impossible job.

Norman Temple: Well, yes, I agree with that. And, but there's no end to these. You can look at diabetes and obesity. And a bunch of other diseases and they all tell the same story. Diabetes has been investigated for a hundred years, insulin was discovered in Canada a hundred years ago, and people have been trying to understand what's going on in diabetes.

And after a hundred years, it still comes down to the simple fact that if you exercise, that's very good for prevention, the most important thing you can. To prevent diabetes is to keep your weight down and also cereal fiber. So having whole grain cereals, the cohort studies show this clear and strong protection that people who eat more cereal fiber, which means they're having more of this whole grain food in their diet, whole grain cereal food. They have this much lower risk of getting type two diabetes. So that comes from the cohort studies.

But why does cereal fiber prevent diabetes? We don't know. I wrote a paper on this with a colleague, we speculated on possible mechanisms and most likely it's, what's called a microbiome in the colon.

You have these billions and billions of bacteria and some of them are good and some are bad and you wanna have the right balance between them. But this is an area which is related to disease, and there's a lot of research going on and maybe down the road, people will be able to manipulate this more cleverly and understand what's going on. But at the moment, we don't know. The one thing we can say with confidence is that having your unrefined, whole grain, breakfast cereals, and whole wheat bread and oats and so forth; that lowers the risk of diabetes and the evidence is very strong for that. And when it boils down to it, who cares how it's working

Danny Lennon: Yeah, to make actionable food-based recommendations to people, we can get there first and then almost working out some of the mechanisms afterwards is in some case a nice luxury, or of course it can inform other work down the line, but to actually get to an initial recommendation, we actually have plenty to work with.

And this is actually something I wanted to ask you about because unfortunately it's sometimes the case where people will comment about the state of nutrition science and say, oh, well, we can't really we don't really know anything or there's so much disagreement between different work or we can't really rely on nutritional epidemiology.

And so while even if they agree with, yeah, we don't wanna rely on mechanistic work. There's this view of well, epidemiology is not really reliable. We need to go for the "gold standard" randomized control trial. And this is something we've talked about on this podcast. And I know you've written really well on this idea of, well, we need to be careful when it comes to nutrition science questions and how we think about randomized control trials, particularly in relation to things like cohort studies and be very careful about thinking as one of them as always inherently better than the other. And it very much seems to be about question that we're concerned with. So I have a number of questions on this. I suppose the first one would be from an overview level. Can you maybe just give your thoughts about that type of rhetoric that sometimes emerges where people think of randomized control trials as being inherently better quality evidence in all circumstances relative to a cohort study, simply because that's epidemiology and how we might need to have a more nuanced way of thinking about evidence and trial quality.

Norman Temple: That's a very good question. Now the official dogma is that randomized trials are the gold standard because you have no variables who can control everything. So for instance, you want to find out whether, shall we say having cereal fiber improves the digestion. So you get a hundred volunteers and you split them in two groups, 50, get the cereal fiber and the other half get something similar, but it's not cereal fiber. And you wait to say two or three weeks. And you check them to see what's happened. You could be looking at the digestive function and that's you get nice, reliable evidence because you've randomized them.

You've only got one variable and the results should be super reliable. Whereas an epidemiology study, you've got no controls over how people behaved. You are relying on they've chosen what diet they're eating and the diet you choose to have is related all sorts of other things, like your level of education and your interest in health and so on.

Everything is linked to everything else. So epidemiology does have these limitations and randomized trials. Really good and reliable. So they're often called a gold standard now in drugs, they work beautifully. So every drug that you, before you, any doctor is allowed to prescribe a drug, they do these randomized trials to see if it works or not.

So the COVID vaccine, for instance, they had clinical trials and they showed it works. It reduces your risk of getting COVID and then they authorized it. Now that works very well for drugs, but it doesn't work nearly as well for nutrition and health. And there's reasons for that. One reason is that you there's a lot of areas you can't do the trial, you can't do things which are too dangerous. You can't ask people to go and get drunk every night for six months. That's unethical. The other problem with it is that you are looking at a fairly brief period of time because of the problem and numbers. If you want to study, shall we say does cereal fiber prevent cancer?

You would need large numbers of people for a long period of time. That costs a lot of money. You can't do. It is researchers only have limited budgets. You can't hire a hundred thousand people and follow them for 10 years. That's just not feasible. So to try to get around there, they focus on people at very high risk.

So a heart disease study, very often will recruit people who have already had a heart disease, because if you've ever had a heart attack, once you are at high risk. So instead of getting somebody who's 30 and healthy, you need a huge number of people like that in order to do a proper experiment, a good randomized trial, you'll probably have to have 10,000 people and wait 20 years.

That's gonna be too much money. So instead you get people perhaps in their fifties and sixties who have had heart disease, and then a lot of them are gonna get another heart attack. Now that's often how they do these randomized trials, but then you've got a problem: if somebody's already had a heart attack, they're in a late stage. This is a disease that takes decades to develop; the unhealthy lifestyle started in childhood and gradually went on for about 50 years. And then finally, when you are in your sixties or seventies, that's when the heart attack happened. When you do your randomized trial and somebody who's 60 and has heart disease, you are looking at a very late stage in the disease.

A cohort study; the great advantage of that is you are starting much earlier, so you are recruiting somebody who's healthy and 40, and you follow them for the next 20 years. And you are following them during these stages of developing heart disease from early stage to advanced disease. And you may, it may be that the cereal fiber works, but only at the early stage of the disease, it doesn't work later in the disease process, which is why often we think the cohort studies for that reason may actually be more reliable.

And I wrote a paper on that, giving several examples. Fish oil is a good example of that. Does fish oil prevent heart disease? Where there's been lots of good studies, cohort studies has shown repeatedly people who have fish have lower risk of heart disease. That is based on following people, young, healthy people for about 10 or 20 years to see who develops heart disease.

And they're following the people through the different stages of heart disease. Causation... the randomized trials to keep the numbers down. So you, so you can do it within your budget. They recruit people. Who've already had heart disease and then it doesn't always work. And it may be that it doesn't always work because you are looking at people at a very advanced stage of heart disease. It may already be too late.

Danny Lennon: Yeah. I mean, that's a great example. And I think in a previous episode, we talked about this discrepancy between what we see in epidemiology around say high fatty fish intake, versus some of the many kind of null findings we're seeing with an omega three supplement trials and heart disease as this example where we're seeing difference.

And I think this really gets to one of the key points that you are making is that, and there's really, there's actually two sides of this when it comes to thinking about cohort studies and randomized control trials. First of all, a point we can certainly circle back to is that in many cases and on many topics, there's far more agreement between the literature base in both of those areas then people

maybe realize, but then on the other side of, in cases where there is this potential disagreement in findings, for example, you mentioned fish oil, another good one that you mentioned in that paper was dietary fiber and colorectal cancer. Is that when there is a disagreement, what we shouldn't do is immediately default to saying, oh, well, if there's disagreement, well, that means the randomized control trial has to be right and the epidemiology is definitely wrong. Rather, we need to look at what's going on and think about, and place those findings in context and see, well, Which set of these studies is better set up to answer this particular research question we're interested in and what could explain this seemingly disagreement.

And once we start looking through some of the context and caveats to that, we can kind of understand, well, why we're seeing that disagreement as opposed to immediately saying, oh, well, the epidemiology has to be wrong, right? The RCT is always gonna be right. Whereas that could lead us to some incorrect conclusions.

Norman Temple: Right this good point, the randomized trials, it would be very, very nice if there was... we could write nice, very, very simple rules: "if there's a disagreement between the findings of a cohort study and a randomized trials, one is gonna be right and the other is gonna be wrong." That is a big oversimplification. Because that breaks down in the face of reality. You've gotta look at the totality of the evidence and try to figure out which makes most sense. So with your fish oil often, it doesn't work. This another important difference is mostly it's giving people not fish, but fish oil.

And the cohort studies is mainly based on people choosing to eat fish. And fish is not the same as fish oil. It could be that the cohort studies work and fish because they're mostly not many healthy people have fish or supplements every day, but many people have fish every day. So mainly they're looking at fish is appears to be preventive heart disease, based on cohort studies, but the randomized trials produce this wishy wasy contradictory evidence that fish oil reduces the risk of a heart disease. But again, that's based on fish oil and it's based on people who have already had heart disease. Now in that case, I would say I would go with the fish and say fish prevents heart disease, because we've got this large body of consistent evidence and it's difficult to think of how it could have been done wrong, that we find time and time again in different countries. People who choose to have fish two or three times a week have this much lower rate of going heart disease.

And you just can't ignore this data so that I would say has to be right now you mentioned fiber and colon cancer now, very similar example a gentleman who

comes from your part of the world called Dennis Burkitt, who became very famous for his work on dietary fiber. He was actually originally from Coleraine in Ireland. He was born before there was such a thing as Northern Ireland. So he's actually Irish. And he was the person behind dietary fiber and behind the idea that dietary fiber prevents colon cancer. And this idea is still debated. We're talking now 50 years later and it's still being debated and so the cohort studies produce just like the fish, not as strong as fish, but fairly good evidence that people who have plenty of fiber in their diet have a reduced risk of colon cancer. So what people then try to do is a randomized trial. But then you... ideally to do it, you'd recruit a hundred thousand people in their forties and randomize them and ask 50,000 of them to have fiber every day and the other 50,000 to continue with their regular diet. And then follow them for 20 years; that you cannot do. You'd need a gigantic, you'd need millions and millions of pounds in your research budget. This is not feasible. So how do you get around that? So just like the fish oil, they did it on people who have had a, his already had a heart attack. So with colon cancer, they did it on people who have had adenomas in the colon.

That's a benign type of cancer. And then check to see if the fiber over the course of about three years prevented the recurrence of adenomas. And it didn't now to say that fiber, therefore doesn't prevent colon cancer takes a huge leap of faith. We can't conclude from it there they're already in a fairly advanced stage on the road to getting colon cancer. They've got adenomas. That means something is already wrong in their colon. And then they're not looking at colon cancer, they're looking at benign tumors. So there's all sorts of problems. And we have to be very skeptical about making any real conclusions from that type of research list. You can't leap from that to say, "ah, fiber doesn't work in preventing colon cancer".

So they never looked at it. Whereas the cohort study just like the fish with heart disease, you're looking at over the very long. Starting at a very early stage on before colon cancer has appeared right to the late stage when person has colon cancer. That is why I'd prefer the cohort study to the randomized trial. There'd be plenty of other examples where randomized trials are really good.

Danny Lennon: Thank you so much for that Norman, that was a excellent explanation. I think maybe just around this out for people, because you mentioned it right at the end, there is we don't wanna go the other way and say, well, if there's any disagreement between cohort studies and randomized control trials then for all nutrition questions or any particular set of studies, then the cohort studies are always right. That would be equally a fallacy rather than actually looking into the specific nature of each trial and the specific nature of.

The particular question we're looking at: are there any examples that come to mind that might be illustrative of people where we do have maybe disagreement between them or at least some degree of conflicting findings where findings from RCTs have been really instructive in our understanding of a particular nutrition related question? Are there any examples that we could show people that might be useful?

Norman Temple: With vitamin supplements, mineral supplements, the results are more akin to a drug and some of the results are, if it's a short term effect, then the supplement can be very useful. Now, vitamin C and the common cold comes to mind because that's a short term effect.

So winter is coming up, will vitamin C reduce your risk of getting a cold or flu? So you can do it over maybe six or eight months. That's quite feasible for a randomized trial and the result would be reliable. A good one is beta carotene cause that's something I was involved in. I did studies on mice in the eighties and it prevented colon cancer in mice. Now the, this is where the cohort studies... There was a great learning lesson that was learned as a result of all this, that people saw that beta carotene is associated, has a negative association with the risk of different types of cancer.

And I wrote a paper, a review paper in about 1987, said, yes, the evidence is pretty clear. If you have more beta carotene in your diet, then you have a lower risk of all different types of cancer. And that was the opinion in the late eighties. Then people based on that did randomized controlled trials about maybe four of them and they all produced these negative results. Had no benefit. In some, actually it may have made things a bit worse. It may have actually in a few subgroups may even have increased the risk of cancer. Then people went back and scratched their head. What the hell is going on? Why do these studies contradict?

And the best explanation was the randomized trials have been done over several years. If the, if beta carotene does prevent cancer, they, we should at least see a hint of a benefit after several years, there was no hint of a benefit, not even a little bit of benefit. So that's strongly suggested to everybody it doesn't work.

So then you go back and look at the cohort studies and then it dawned on people. There was a fundamental error and there's a thing called confounding that everything is associated with everything else. You have people who lead a healthy lifestyle. So they exercise, they don't smoke, they eat a healthy diet, it all goes together.

And then when you do a cohort studies, you have to be very, very careful that you are not having guilt by association, innocence by association, that when you show that exercise reduces your risk of disease, it's not because the exercise maybe has no effect, it's the other things that go along with exercise, like the fact that you don't smoke and you keep your body weight down and you eat a healthy diet.

This is all called confounding. Now looking at food specifically, where do you get your beta carotene from? You get it from fruit and vegetables. So people who will eat a lot of beta carotene , what you are really showing is they're eating a lot of fruit and vegetables. And then it dawned on people. No, it's not beta carotene that's preventing cancer. Everybody got it wrong. It's because you are eating a lot of fruit and vegetables, beta carotene , the same with vitamin C in about 1990. An American researcher did the same thing as they done on beta carotene. They looked at all the studies and found wonderful people who have a lot of vitamin C have a low risk of cancer.

This clearly shows that if you have vitamin more vitamin C in your diet, this will reduce your risk account. The next step would obviously be, have vitamin C supplements and that will stop you getting cancer. Then it dawned on people. No, that's seriously flawed. There is no evidence that vitamin C prevents cancer, it's that people who have a high intake of vitamin C, they're eating a lot of fruit and vegetables. It's the fruit and vegetables that prevents cancer. Vitamin C is just a marker to indicate how much you're having in the diet. That was a very good learning experience and people became much more conscious of the great importance of confounding.

You can't. You have to be very, very, before you start accepting the results of cohort studies. They're very good, very, very reliable, extremely valuable. Yes, but they are prone to this area due to confounding. So you have to be very cautious in interpreting the results of them. Beta keratin was really a great learning experience in this area.

Danny Lennon: That's a really useful example. And I think one thing that that naturally extends to and probably for regular listeners, they might be connecting to a couple of previous episodes of this podcast, maybe notably with professor David Jacobs, who I know you've published with and Anthony Fardet, where we have these ideas around food synergy, but also the problems of an overly reductionist approach. If we try and take isolating down everything down to say an individual nutrient, like you say there, rather than realizing that, okay, if we're gonna do that, we need to remember that. First of all, nutrients are gonna

be consumed in the context of overall foods and overall diet patterns and meals. And so there's synergy between nutrients.

And then on the flip side, we need to have this non-reductionist idea that food X is a proxy for a certain nutrient. For example, if we think, "oh, well, milk, that's just calcium, right"? Where that's obviously a problem it's been overly reductionist. And so some of those ideas that you have just brought up echoes some of that, those ideas that people may have been exposed to around thinking too reductionist in around single nutrients, as opposed to thinking about an overall diet.

As the exposure that we typically care about most. And so when we're investigating these questions of being really careful of how not only studies are designed, but then how we in turn are gonna interpret them right.

Norman Temple: A hundred years ago, from 1920 to 1960 in that period, that was a golden age of vitamins one after the other they had all these diseases and they realized. They were vitamin deficiency diseases. So, Beri Beri, and scurvy and other diseases directly related to lack of particular vitamins, one vitamin one disease, and that made people think that changed people's mindset in nutrition research, people started thinking very strongly about single substances. And now there's been this big move that... you refer to David Jacobs. So we've written about that food synergy. I wrote a paper on him on that subject to food synergy that it's all the diet as a whole talking about vitamins, single substances, potassium, or vitamin C or beta carotene, or dietary fiber. You wanna think about the whole diet? It's everything interacting with everything.

Yes, fiber is very important and it probably has many direct benefits on the body, particularly on the colon function. Potassium is very important. Folate is very important. Vitamin C is very important, but it seems what's much more important is all these things added up together and interacting, and then you've got the phytochemicals. Thousands of them are in tea and so, and fruits and vegetables, they're thousands of these substances and they are also very important. So when you are eating your diet, you are eating all of it. And now we think not of single substances so much, they can still be important in some areas. As a single substance fiber is important. Sodium is important. Sugar is important. They do these things either good for you or bad for you, but more, much more importantly it's the diet as a whole. So we think in terms of food patterns, you have to have a healthy food pattern now Here's an example. I'm writing a paper right now on the origin of the obesity epidemic particularly in the USA and the focus is on what caused it, you can't explain it in terms of a single substance, like fat and carbohydrate.

You have to look at the diet and it's the amount of what we now call Ultraprocessed foods, which is more or less the same thing as junk food. So, white bread, Coca-Cola similar foods, processed foods, the amount of it in the diet and in the British and American diet, more than half the calories are coming from Ultraprocessed food, your pizza, your burger and fries, your Coca-Cola and many, many other similar foods. And this seems to be the explanation for what triggered the obesity epidemic and has been behind it for the last 40

Danny Lennon: years. This could be something that extends into so many different areas and routes we could take with it. I feel that maybe a round two for this discussion would be worthwhile to get into some of the specifics of maybe even that question alone.

But as I'm cognizant of time, we're gonna start wrapping up here. So maybe if I. If we get to a point where we can turn this into some kind of clear conclusions for people that are most useful to take from what we said throughout this conversation today. If we were to think about how we can assess these diet health related questions through looking to research and we want to come to not only clear conclusions, but ones that are evidence based and that we can have some degree of confidence in what are some of the things that you would want to put on people's radar? What are some kind of clear takeaway messages that you would most like to leave people with from anything that we've discussed today?

What, what are some of the things that maybe come to mind that you would most likd to impress upon them and leave them with?

Norman Temple: Well, probably I would say two things. Number one, what I just said a few minutes ago, that the secret of understanding the relationship between diet and disease is seldom found in individual substances. Some of the individual substances can be fairly important in their own, right? The sugar in sugar-sweetened beverages, like Cola drinks. The amount of salt in the diet. These do have a direct effect, but by and large, it's the diet as a whole, the dietary pattern, a pattern of a health diet and the healthiest diet would be a Mediterranean diet. So a low amount of meat, olive oil on the food lots of fruit and vegetables that dietary pattern rather than be obsessed with single substances. So dietary patterns is crucially important. And the second thing is when you've got all this evidence, you've got your evidence from randomized studies and you've got your evidence from cohort studies and they don't there's contradictions and inconsistencies, and you look at it and you are pulling your hair out and you are confused.

You can't have very, very simple guidelines. I would love to give such a guideline. And if I was able to write a paper that made good sense, it would be a seriously brilliant paper, but I don't have such a simple guideline that I can make some. It would be really attractive to have a golden rule. This is how you interpret the results you follow these steps 1, 2, 3, this is the most reliable. This is the least reliable note. I'm sorry, I can't produce such a simple golden rule. Maybe some very clever person will come along in 10 years who will write that golden rule. But. At the moment, at least I'm not that person. And you have to take all the evidence and critically evaluate it.

That is the best golden rule we have right now. Look at the totality of the evidence and critically evaluate it. What are the strengths and weaknesses of the different areas of research.

Danny Lennon: Maybe as a way to finish with the very final question that I always end the podcast episode on, and this can be in relation to anything even outside of something we've discussed today, it can be outside of nutrition. It's something more broad. So apologies for putting you on the spot with it, but it's simply, if you could advise people to do one thing each day, that would have some positive impact on any area of their life. What might that one thing be?

Norman Temple: Okay. I'll give you a good answer. Don't have children. If you love this planet, if you're concerned about global warming, if you're concerned about the amount of plastic in the middle of the Pacific ocean, if you are concerned that Britain is a very grossly overcrowded island and it would be a much better place if the population was cut in half. And I live in a very beautiful place here in Vancouver, which is more densely packed than New York city. The world would be far better. If we had far less people in it.

Danny Lennon: That is certainly a unique answer. And one that probably for me at least is suitable . And one, one that I can get on board with. And thank you for not only your discussion today, but for the work you've done beyond that Norman, like I said, it's been quite interesting for me to read and it's been the very thought provoking around some ideas that I've discussed. And not only have I enjoyed reading that, but I've really enjoyed this conversation for our discussions around politics before we started recording. And then also for have we've wrapped things up here today as well. So I wanna thank you for all of that and for really taking the time out of your day to come and talk to me, it's been an absolute.

Norman Temple: Well, thank you very much for talking. It's been a pleasure to talk to you about all this. I hope I've contributed some useful comments for your listeners.