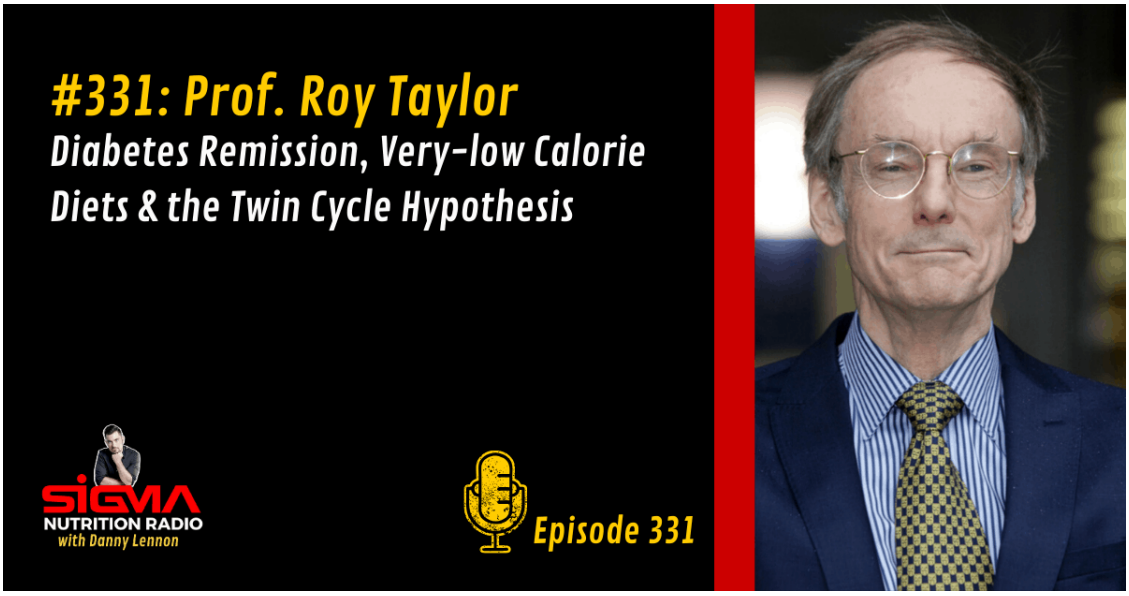

DANNY LENNON: I thank you so much for joining me on the podcast.

ROY TAYLOR: Oh, it's a pleasure to talk about this subject. Danny.

DANNY LENNON: We have a lot to get into. And before we get into any specific publications that you've put out over the years, and the work that you've done in the lab, maybe to get listeners on the same page, when we're thinking about Type 2 diabetes, what is the most accurate way to explain that as a condition that will kind of set the stage for maybe some of the rest of this conversation?

ROY TAYLOR: Sure. Well, diabetes just means like glucose control is not adequate. And so the levels of glucose in the blood, sugar in the blood will go above normal and that can cause all sorts of damage, especially in the long term. But the two main kinds of diabetes, and it's easiest to start off describing Type 1 diabetes first, because that is the kind it usually comes on in childhood, but in fact, can come on at any time of life, and always needs insulin injection therapy. So that's absolute and without those objections, people are likely to become extremely ill and indeed die over a period of just a few days. So that's Type 1 diabetes. And it's sharp contrast Type 2 diabetes can be controlled by diet. It can be improved a bit by tablets and tends to come on in later life. It also tends to be associated with being heavier than the average. Although that's by no means absolute as we'll touch

on later. So Type 2 diabetes has fallen away the corners kind of diabetes advocate for some 90% of diabetes in the community. But before just leaving it at that it's worthwhile also making a point about some very rare kinds of diabetes. Because Type 2 diabetes is not a disease that can be diagnosed absolutely. There's no one test that a doctor can say, I'll run this test and come back to you with a definite answer. It's always a diagnosis of exclusion. So Type 1 diabetes can be excluded to a large extent, if the various antibody tests that can be run on negative, well, that's only about a 70% exclusion. The important kind to recognize is about 1% of diabetes that is a genetically determined form. And that is quite different Type 2 diabetes that is easily confused with it. So in everything I say on this podcast, I'll be talking about typical Type 2 diabetes and sometimes we're surprised after a year or so it turns out that someone doesn't have Type 2, they have an unusual form. So it's worthwhile just setting that wider scene first.

DANNY LENNON: Pretty much most people even in the lay public understand or correlate diabetes with an issue around blood sugar. But from a more detailed look at maybe some of the pathophysiology, what is the underlying mechanisms that drive blood glucose to be not in control, essentially? What is actually happening within Type 2 diabetes?

ROY TAYLOR: It's probably appropriate to start off with what might say is a conventional view of the cause of Type 2 diabetes. And it's been recognized quite correctly for a long time. But there are two aspects to this. On the one hand, we have insulin resistance. Insulin just doesn't work as well as it normally should. On the other hand, we have the fact that insulin is not made by the beta cells in the pancreas as rapidly or as effectively as it should be. So we have two conditions but confusion has crept in, because it's been felt that this is a very heterogeneous disease, different people seem to exhibit different features and is being regarded as a big confusing area. So the conditions being regarded as being due to multiple possible causes, both affecting insulin resistance side and affecting the matter of making insulin secretion side. But that's exactly where the story starts. Because the back in 2006, I was sitting at my desk, and suddenly,

the final piece of the jigsaw dropped into my lap or dropped onto my desk, you might say, because it was possible to see in a flash that in fact Type 2 diabetes could be quite simply explained. Yes, it could be complicated, but the complexity was not in the disease. The complexity was in the background population. Just look in the high street, all sorts of folk out there. But it's just one cause. And what I had envisaged was that it's just too much fat inside the liver that's causing the insulin resistance and too much fat inside the pancreas that's actually causing the defectiveness on secretion. So that was something that I published as the twin cycle hypothesis. It was possible on the basis of what we knew to say, how were the liver might slowly get more and more than more fat building up as a vicious cycle, but also has a might be a vicious cycle in the pancreas that was started off by the fat spilling out of the liver and slowly silting up in the pancreas. And then another vicious cycle, with insulin not being made so well after meals and eventually the system breaks down. But the beautiful thing about having a hypothesis is that you can test how wrong you are and so this is how science proceeds. Scientists have got to try and knock down the hypothesis as effectively as they can. And if it's complete failure, and the hypothesis is still standing, well, it might be right. And I have completely failed to demolish this hypothesis over 14 years of work now.

We set off to test it, and we could test it quite easily because the hypothesis predicted that if we took people with ordinary Type 2 diabetes, and asked them to eat much less than usual then these twin cycles would spin in the reverse direction, and we could say the diabetes will be reversed to normal. So it's postulated the glucose level or sugar levels would return to normal in quite a short time, after starting a low calorie diet, and the response of the insulin producing cells would go back to normal. And so we ran the Counterpoint study, this was back in 2008 and by 2011, were able to publish, to my utter astonishment, that that's exactly what we observed. We treat people with very ordinary Type 2 diabetes and it has to be a homogenous group we study in any test like this. And so we took people on diet alone, or a diet plus Metformin treatment. And the men and women just ordinary age, ordinary weight, but not too

heavy, because to do the tests, they had to fit into my magnetic scanner. And so it was a body mass index up to 45. And just in the first four years of Type 2 diabetes. So we took a very distinct group of people and went and put on a diet that we can talk about more later, they returned to normal glucose control first thing in the morning, within seven days. Now that was just astonishing because there's no other treatment for Type 2 diabetes that restore normal, like glucose in such a short time. And over the next few weeks, we saw something even more remarkable, because the insulin producing cells; these beta cells in the pancreas, gradually woke up and went back to be able to respond to glucose as fast as normal beta cells works. So it demonstrated right away that first of all, Type 2 diabetes is a simple disease of fat excess. It affects the liver and the pancreas, and that it's potentially reversible, at least in short duration disease. So that was the starting point for all this.

DANNY LENNON: First thing that comes to mind and we'll probably explore this a bit later on, is, I'm just interested to know off the back of that 2008 paper where you publish the hypothesis, what the initial reaction was from the research community, and then how or if that has changed over time in light of the Counterpoint study, or maybe even some of the newer work that we're probably going to discuss later in this conversation.

ROY TAYLOR: I think it's fair to say the response to the hypothesis paper was polite interest, but disbelief among scientists, and of course, there's a whole range of reactions. Some people got it right away and said, that's really exciting. We've got to see the results of this test. And most people say, no, no, no, you don't understand. Type 2 Diabetes is really complex and you've missed the point. Well, the range was illustrated by the response of the audience because I'd actually presented it to the diabetes UK meeting in 2007. So I had a plenary lecture, and I decided to announce this in the plenary lecture. And most of my colleagues were politely skeptical. But fortunately, the editor of the European Diabetes Journal was in the audience and he came up to me afterwards and said, that's absolutely fascinating. Why don't you write it up as a review for the journal. Now, that was actually

fantastic because to write up a hypothesis on paper is much more difficult than just doing it in a lecture where you can take shortcuts and just refer to papers you've read. This has got to be tied down. And so there's 2008 paper, the hypothesis paper is a result of one person being extremely excited on hearing this hypothesis. So a range of responses

DANNY LENNON: And with those twin cycles, we're talking about accumulation of fat at the liver and pancreas specifically, which has to be preceded by positive energy bonds over some sort of chronic period. So presumably there's a lag time from when that positive energy balance is in place to when we start being able to detect higher elevated levels of blood glucose consistently in someone. Do we know what that typical lag time looks like in terms of the time period?

ROY TAYLOR: Well, yes, we do. It wasn't clear at all in 2006. But in around 2009, that sort of time, the results of the Whitehall II study were published. And this very interesting study that followed up by a large number of British government employees who were middle aged, and across a number of them got diabetes during the study. And it was possible to follow them year-on-year until they develop their diabetes because this was a long term study, and all the blood samples were taken, and the sugar was measured. And the people who got diabetes started tracking up their blood glucose very slowly over about 10 years. And then, in just the 18 months before the diabetes was recognized, their sugar levels rose very rapidly. So there we have it all in a nutshell. We can see these twin cycles spinning onwards over the next 10 years. But then there's a big acceleration as the beta cells are being battered into submission every 18 months. So yes, we've got a handle on this.

DANNY LENNON: And that I think ties into one issue that has been highlighted by several people at this point of when a lot of people get a diagnosis of Type 2 diabetes. And we know that's tied to this decreased beta cell function, that there's actually been a decline in that beta cell function for a long time proceeding that diagnosis. I think it might have been the UK perspective diabetes study that showed was like 50% decline, I think, on average, before diagnosis, which

kind of shows the magnitude of the difficulty in practice that medical professionals are facing with this issue.

ROY TAYLOR: That's true. And it was both observation of people in the clinic, as well as the United Kingdom prospective diabetes study. That led to the idea that Type 2 diabetes was inevitably progressive pieces of function and gradually fell off. Even by the time of diagnosis, it was already down to 50%. And almost any test that was used, including looking at the post mortem pancreas people with diabetes, the number of beta cells still present seem to be about 50%. So everything pointed to this slow, steady loss and the fact that the beta cells have gone on the histological studies seem to underpin this. They've gone, they were “dead”. But in fact, just like Mark Twain said, rumors of my death have been much exaggerated. So these beta cells were not actually dead. They were just dormant. And the reason why they can't be seen on the histological sections is that we would use a stain to detect a cell as a beta cell across to the cell was there minding his own business, but not making any insulin. You don't stain for insulin, you're not a beta cell. So we can actually explain why this misconception about inevitable beta cell decline came with that. We know from our in fact, paper published just a couple of months ago in diabetes care that the beta cell recovery can be really convincingly complete and stabilize so that 50% maximum capacity goes back to complete normality. That's a remarkable finding. It takes us all 12 months to get back up there after reversing the diabetes. But once it's attained, provided people keep the weight off, then the beta cells are just fine. It does depend on this being reasonably short term Type 2 diabetes. The longer the duration of diabetes before the weight loss, then the greater the chances that beta cells won't come back completely to normal. So if we look at people with less than six years duration of diabetes than 9 out of 10 people achieving 33 pounds weight loss 50 kilograms of weight loss will get rid of the diabetes just to put a frame on it.

DANNY LENNON: When earlier you referenced the Counterpoint study, and mentioned that we see over time this gradual return up that first phase insulin response and knowing that the Counterpoint study has this low

calorie diet used in the intervention, maybe an intuitive question people would have as well, what's the long term implications of that going beyond the period of just that acute diet? But as you said, there seemed to be evidence off the back of that afterwards that has suggested that it does actually, we can actually keep it maintained that success that's ---

ROY TAYLOR: That's correct. And that was the most personal question that was fired at me when I presented these results. People say but this is just showing the situation of severe calorie lack, these people are in a state of severe negative counterbalance. The diabetes is certain to come back as soon as you go back to calorie balance. So we run a study, the next study was called Counterbalance. And that aimed to keep the weight steady for six months after the acute weight loss. And we reproduce the initial findings of diabetes going away and in short duration disease, but we actually managed to keep weight absolutely steady. And that study, we got something like 30.5 kilograms of weight loss just a bit less. But it was rock steady for six months after that, and we demonstrated that the beta cell recovery was there and allow normal glucose control in people with short duration diabetes, and it would be absolutely there for six months of course the insulin resistance remained awake. That's had disappeared with the initial weight loss. So we did answer this early on, but even the early data from the Counterbalance study, took us on to the big question, which was okay, this is all very well with clever doctors in a research institute doing this. But could this be used in the wider health service in a family practitioners office, for instance. And so that's why we ran the Direct study. And so in this study, it was just the primary care nurses or dietitians that we had them but the primary care staff who actually administered the low calorie diet and changing to an isochoric diet. And in this study, we were able to show that in the general population, we could get admission of Type 2 diabetes in 50% of people in 9 figures 46%, actually and that was largely maintained at two years 33% sorry, 36% after two years duration after the weight loss.

DANNY LENNON: And I think Direct was one of the studies that had massive feedback beyond just the academic world and

obviously generated a lot of interest because of the implications of those findings. Just to clarify for maybe some people who haven't read that study yet, can you maybe give some more specifics on the actual intervention of that low calorie phase? And what that look like?

ROY TAYLOR: Sure. It's probably worthwhile just talking about the whole approach to this low calorie business. Because back in 2006, I was faced with a problem was how we persuade people with Type 2 diabetes to lose about 15 kilograms in weight, like calculator does about 15 kilograms should be about the right amount to achieve this reversal of the trend cycles. Now I knew all of the old data on the very low calorie diets and the striking thing about that era was that people reported relatively left hunger want to actually be established on it. Now, as a doctor, I've been told by many people over 44 years of medical practice, that the difficulties of losing weight are the terrible hunger. You just can't live with hunger nodding away here. And the second thing is, the reason why diets don't work is that you get absolutely exhausted with the day-to-day burden of making decisions. Can I eat this? What should I eat. Is that half a cup full or two spoon full? It's, it's awful. It just gets to you after a while. So I knew how I had to make it a bit simple. And by virtue of using a low calorie diet, we avoided the hunger problem in the long term. And by virtue of using a liquid formula diet we avoided the decision problem of what could you eat. Well, you just eat that packet for lunch, that packet for dinner, etc. But life isn't all about metabolism. There's also the rest of the body and especially the gut and because of that, we introduced non-starchy vegetables. So salad foods, other leafy green vegetables, for instance, and advise people to have a good portion of that every day. So that was the original diet we used, and it's a diet we still use in our ongoing research subjects studies. But during Direct, we use the modification of this diet and that is because Direct was a joint study between myself, my colleague Mike Lean [PH] and Glasgow. Mike is an obesity expert. And he had actually used a low calorie diet to manage obesity in primary care and had the logistics setup to deliver what he was already using. And he was using a liquid formula diet only. Now we choose this over and we figured that for the simplicity

of running a big study, it was worthwhile taking on the challenge of the extra constipation that would otherwise result. And that's what we did. So in Direct, we use four packets a day of liquid formula diet, that's just over 800 calories a day, and no ordinary foods. We had a fair amount of trouble with constipation. It's all laid out in the papers. And certainly, I would advise anyone to go with our original approach now, if we're taking off and doing it for themselves, but that's how we approach this. We knew that at the low calorie approach was associated with not very much hunger. It's also associated with what we discovered in our very first study encountering point, people were coming back to us and saying, I feel great. You know, I haven't felt like this for years. In fact, I feel 10 years younger. So then we were setting up trying to sort out Type 2 diabetes and inadvertently we discovered the elixir of eternal youth! People felt so much better and they felt better within two to three weeks usually, and also people reported increased energy levels. So who are feeling more energetic day to day, even though they're eating so little. I have to point out that about one person in 15 does feel a bit tired on the diet. But that's a tiny-tiny minority reaction, but vast majority of people are banding round like spring chickens chasing their grandchildren around the garden. So it really is quite an impressive effect and there is no disbenefit to the rapid weight loss. This has been one of these rumors that have run around nutrition circles for quite a while and it simply is not borne out. We've published all the observed adverse effects in our writings and Counterpoint, Counterbalance, Direct. It adds up to being just trivial, difficulty, boredom of the diet. Some people report feeling a bit cold because anyone feels cool during a period of weight loss, presumably the body mass, the basal metabolic rate is actually falling during that time and this is easy to put up with. On the other hand, the group of people who went on in the control end, who have the usual treatment for diabetes are the ones who had some major problems. Several major classical events, several other events and five weight related cancers. Now, that's dramatic. It's not that we're changing the cause of all these different cancers. It's we're changing time to presentation of suddenly dropping body weight produces a dramatic drop in plasma insulin levels. Insulin is a tumor promoter. And it's a no

brainer, that one is related to the other. I'm afraid and oncology circles not the heavy, whether it's negative, why some cancers are related to weight, but I really have no doubt that it's largely mediated through the insulin and we see a dramatic fall in fasting plasmas insulin levels from diabetes going back down to normal. So, side effects? Well, if you want side effects just carry on with your diabetes treatment. If you want freedom from side effects, then try some rapid weight loss.

DANNY LENNON: When it comes to diabetes remission, how was that defined by your group and is that a or when that's decided upon is that something that's consistent across groups? Or how does that number get arrived upon?

ROY TAYLOR: Originally, I simply used the definition of diabetes. It seemed to be the place to start. So an HbA1c over 6.5 or fasting plasma glucose is a bit out of date with regard to the US levels at the moment, but fasting plasma glucose of 7 or more, that's diabetes, and less than that, should all be regarded that as not diabetes. Now, of course, some people might be in the gray area where you might say, well, that's pre-diabetes, isn't it? Well, no, it's not. If you kind of go the other way, it's a completely different state, because you have normalized lipid metabolism. And it really express that in terms of 10 year risk of heart disease, but then calculate from indices just not indices at the time, then this measurement drops dramatically. In Counterbalance, it dropped from about 23% risk, I think it was down to 20% risk, dramatic drop. Now in direct, we're dealing with a group of people who had rather lesser degree of risk, but it still went down from about 16% to 8%. So there was a dramatic change. And it doesn't matter if plasma glucose is hanging around what might ordinarily be called pre-diabetic levels. That's pre-diabetic level, but it's not mean the same thing. So people need to be very careful about assigning a risk to purely looking at plasma glucose. So that's why we alight on less extreme simplicity of saying, okay, let's talk about readmission as being no longer in the universally accepted diabetes range. And this is something which has subsequently been accepted by the UK Primary Care Society and the UK Specialist Society in Diabetes that's published on the

web and in the journals. We're in discussion as to we, the ADA with the ESD and Diabetes UK to come to some agreement, and the discussions are underway, but I'm afraid, various things have gotten in the way of concluding them and they're still not concluded. But hopefully, we'll have consensus that we're looking at a simple diagnosis of not diabetes. In other words, what under the diagnostic levels for diabetes, and on no diabetes tablets or injections. So that's the number one thing from the point of view of my patients. They want to come off the next month. They don't want to be labeled as being that diabetic over there. They certainly don't want to pay 50% more for the holiday insurance with people with a diabetes label have. So they want off their tablets, which they don't like swallowing, and they want to be clear of the diagnosis. So that's where we're going with the definition. Hopefully, there will be a worldwide agreement that it's not diabetes below the diagnostic level [Inaudible] [00:35:01]

DANNY LENNON: With this evidence now that with the certain amount of weight loss and reduction in adiposity that we're seeing is this remission of diabetes, an improvement in that glucose response back towards normal. Maybe one intuitive question a lot of people will have is, well, sure, if we have people that could we see this high correlation between obesity and Type 2 diabetes. So it makes sense, we'll try and lose a lot of body weight and bring down adiposity. What happens in the case of someone who is typically determined to be normal weight or within a normal “BMI”? Does the same strategy apply to them and if so, why is that the case?

ROY TAYLOR: So for people who have Type 2 diabetes, but have a normal BMI, or just don't overweight BMI, the writings very much on the wall Now, this assumes that it's classical Type 2 diabetes, because with people with normal BMI, there is a rather greater risk that they may have a genetic form of diabetes, but that greater risk is only rising to about one in six, one in eight. Sorry, one in 10 will be 12 sort of chance of that being the case. So we come back to our person with say, a BMI of 24. I wonder how they got there. Now, if we're talking about a person in their 40s, 50s, 60s, it's highly likely that person at the age of 21 would have had a BMI of say 19. And if they get back towards that

BMI, they're highly likely to get diabetes. So in fact, they have become obese for their own body. Now, the body is an individual trying to lump people together and put things down firm lines anyone with a BMI over 30 isn't a disease category. That's awful. That's obesity. That's their fault. Well, it doesn't work like that. Some big guys have got a BMI over 30. But I'm coming in out of extra fat, probably American football players or indeed British rugby players. So we've got to get away from this population base metric of obesity. If a person has Type 2 diabetes then they've become too heavy for the body. If they share about for the leaner end of the spectrum, about 10% of their body weight, they highly likely to go back to normal provided they've had short duration diabetes. So we've got it for the individual and get away from this dreadful evidence base matrix so called which loops in the population and imagines we can treat everybody as "Mrs. Average" or "Mr. Average". So I've published the personal fat threshold concept, which is essentially what I've just described. In other words, it's not about obesity, forget obesity. Obesity does not cause Type 2 diabetes. Now that might cause a sharp intake of breath. Who is this? He doesn't know what he's talking about at all. Let's look at the data. And we take people who have very heavy, body mass index over 45, then about three quarters of them do not have diabetes. So it's not the obesity that's causing it. It's not crossing a threshold of the population that causes Type 2 diabetes. It's just carrying around and more weight, more fat for an individual can cope with.

DANNY LENNON: We've had on the podcast before discussion with Jason Gill, who's published in the area of looking at different ethnicities and their risk of metabolic health markers at different BMI, for example. So immediately we know that not everyone in the population carries this same risk. And then even beyond that, as you've just outlined, we all have an individual risk even within these smaller subgroups. So you've also mentioned previously that the ability for someone to go or that how easily remission is achieved seems to be related to how long they've had diabetes for. I'm wondering, are there any other factors that seem to determine the likelihood of remission beyond that other any other things that

would tie into how likely this individual is to achieve success in remission versus not?

ROY TAYLOR: Well, I knew only the starting grid is precious little that can be said to determine success apart from duration. That's the big feature. Age is not a big thing. Male, female doesn't really matter that much. But the one thing that really matters is motivation. And this is not something that can be judged in advance. There's some interesting aspects to this. Probably the single most important thing is that the individual themselves is absolutely clear in their mind that they hate their diabetes and refer to the congregation so much, but they want out of bed and they're prepared to take major steps to do that. That's the first thing, but close on its heels is something that almost nobody thinks about. It's the outlook of that person's partner, spouse, or close friends, close family. In other words, it's the people who will support this individual while they're going through what is a tough time, the process is simple to describe. But simple isn't the same as easy. So it's a challenging time for anyone to pass through, and having the help and support from a spouse or friend. That's actually very important. So it flagged up those things as being really the most important factors add to the whole level.

DANNY LENNON: And related to that, and that might be particularly important for the practitioners listening to this discussion. Now that we've seen that diabetes is possible, at least to be put into remission via various dietary interventions that lead to weight loss. I saw before in a previous talk that you gave to, it was something to the effect of saying that now that we know this, and we have this knowledge we shouldn't use as a stick to beat people with if they don't want to go this route of using dietary or lifestyle change. Can you maybe just expand on that and why that's so important?

ROY TAYLOR: Yes, it's so important because we come back to this individual. Now everybody is an individual. As a doctor, I only have one person in front of me in the consultation. My advice is absolutely focused upon that person. And what a doctor for? Well surely a doctor is to help people enjoy life to the full and to help them maximize their health because that allows

them to enjoy life to the full, but what enjoyment? Well, what's the individual's choice, and so I would very much want this to be seen as an option. Yes, we can offer you the possibility of getting rid of your diabetes entirely, returning to health actually feeling far better than you feel at the moment. On the other hand, we can say that we can give you tablets and medicines to manage the diabetes as best as we can. But you're going to have to face up to the fact that you still have diabetes and also you face the risk of long term complications. So there is a choice, but it should be able to be clearly explained in non-emotive terms just absolutely for the individual to decide. If the individual doesn't want to do that well, that's their choice. But it is important that people actually understand what's involved. And see, for instance, someone started on this, but was one of the very few people who really couldn't hack the low calorie diet. They've had an extremely few of that over the last 40 years. But In that case, well, that's again, their choice. And if with all efforts they've tried and discover they can't do it, or indeed lose the weight, but then just can't keep it down in the longer term, which is actually common well, we can offer rescue packages. But we've really got to get inside this person's skin and walk around them. As Atticus Finch said in that famous book To Kill a Mockingbird, you got to understand what it's like for these people to live us themselves. And if their decision is they want to live as a larger person, they want to just carry on having the tablets etc. the diabetes well, modern medicine can provide that. But it's got to be sympathetic medicine. You've got to remember the very basis of medical practice. It's not a matter of aesthetic. rules governance is enforcing.

DANNY LENNON: Dr. Taylor, there's so much complexity and work that people can go and dig into the details of. And for those listening, I will reference and link to all the particular studies we've mentioned throughout this conversation. For anywhere else on the internet that they can find you and your work where's the best place for them to go?

ROY TAYLOR: Well, the best single place is now in a book because just earlier this year, Life Without Diabetes was released in the states. In the US it's published by

HarperOne, and it's now readily available. In the UK it's published by Short Books. Easily available on Amazon. And in the book, I tried to lay out what I've been talking about in this interview, and in particular, try and explain to people, the practicality of how they might want to escape from type 2 diabetes. So that's the most complete results. Also, the basics of how to do this, available on my Newcastle University website. So the shorter cut to that is go to ncl.ac.uk/diabetes-reverse and that website contains the essential information as to how to use this approach with only understanding. It's so essential to make it a success.

DANNY LENNON: With that Dr. Taylor brings us to the very final question that we always round the podcast out on. And this can be completely separate from what we've discussed today. If you wish, it's a quite a broad, generic question. So apologies for throwing at you. But it's simply if you could advise people to do one thing each day that would have a positive impact on any area of their life what would that one thing be?

ROY TAYLOR: That's a complicated question, implying that one thing would be applicable to all. I wonder if I could cheat by making two observations.

DANNY LENNON: Absolutely.

ROY TAYLOR: One is that it's so important for people to set aside some time for them to just have a think about what they're about and what's going on. Some people call that meditation. I might call it riding my bike to work, wonderful time to let the mind go in to free association. It's helpful just to refocus and concentrate on what's important. But when we look at the population as a whole in the UK, while the most of these nations in Europe, now in the United States, overweight and obesity is a major problem, meaning it's very prevalent, I would suggest if there's one thing a person could do, it might be to avoid snacking between meals. It's all about the intake side. Our exercise is extremely good as a way of keeping the weight down long term, it's not great for losing weight. And we need to face up to that, because most overweight middle aged people just can't exercise to a degree where it would have a meaningful effect without spilling over and making them eat more. So

let's get real about this snack size. We need to regard meal times as a time when we put energy into our mouths. Why would you want to do that in between meals? I think that's a message I would leave.

DANNY LENNON: Wonderful. And with that, let me say thank you again so much for the wonderful information for your time today. But even more than that, the body of work that you've produced over your career. I know it's helped a lot of people in the field. And I want to say thank you for that and for coming and doing this today.

ROY TAYLOR: Thank you for putting out information.