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Roy Taylor

Emeritus Professor of Medicine and Metabolism, Newcastle University, UK

Q1 For decades, Type 2 diabetes was described as a chronic progressive disease. You argued it can be reversible. When you first proposed the Twin Cycle Hypothesis, how radical did it seem at the time?

Putting together the hypothesis did not seem radical at all, because we had assembled various pieces of new information that were necessary. Then, in a single morning during a brainstorm, it suddenly occurred to me that Type 2 diabetes would fit into a simple pattern.

I spent several months scribbling it down and moving the boxes around, and the Twin Cycle Hypothesis emerged. The critical thing about a hypothesis is that you need to test it to destruction, and, if it will not be destroyed, then it might be right. That is where we are now.

However, it was revolutionary. It was flying against the tide, because everybody knew, or thought they knew, that Type 2 diabetes was lifelong, inevitably progressive, and would inevitably cause serious complications in later years.

Q2 Your idea of the Personal Fat Threshold challenges the assumption that Type 2 diabetes is mainly a disease of obesity. Why has BMI been such a misleading metric?

BMI has effectively taken over as the standard by which individuals are judged, but what has been forgotten is that it was developed very precisely to measure populations. It describes the average and the distribution within populations, and it is very good at that.

When we come down to the level of the individual, however, it falls apart. For instance, you would not go up to a front-row forward of the England rugby team and call them obese, even though they may have a BMI of 34. At the other end of the scale, there are people like me who have had a BMI of 18 throughout life, and putting on weight to a BMI of 21 may cause Type 2 diabetes.

We do, however, have a way of introducing a more relevant personal measurement. This is highly individual and genetically determined. At present, there is no way of detecting it in advance, but the critical measurement is not weight or BMI, it is waist circumference.

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Over time, most adults unfortunately put on inches at the waist. If that waist measurement is reduced, typically to somewhere between 36–34 inches, an individual may cross their personal fat threshold, and the diabetes will switch off.

So, that's how the personal fat threshold can be a practically useful measurement.

Q3 What did the ReTUNE study teach us about people who develop Type 2 diabetes despite appearing slim?

This is really important, because, after all our initial studies, we knew that people could reduce their BMI from a high level, even

modestly, and eliminate diabetes. However, we had been faced for a long time with apparently slim people in the clinic who had Type 2 diabetes.

It seemed likely they had the same underlying problem, but that BMI was the wrong metric. In other words, BMI was misleading. So, what we did was take a group of people of normal BMI (between 21–27) with diabetes, and they were given a weight loss diet. They lost weight as expected, and the underlying mechanisms of diabetes returned to normal, exactly as seen in heavier individuals. In the ReTune study, it wasn't just half the people who responded; it was 70%. People who are not very overweight often find it easier to lose a small amount of weight, which is an important message.

And even better, in ReTune, we came out with a really practical answer, because we had the waist measurements of these people. Even after weight loss and remission, when matched with individuals without diabetes and the same BMI, waist circumference remained higher in those who had previously had diabetes. We could see on the MRI scans that there was still excess fat inside the abdomen, even though people at that time had lost an average of 10% of their body weight. So, individuals developing Type 2 diabetes have a significant excess of fat relative to their personal threshold. Losing around 10–15 kg, or even 8–10 kg in lighter individuals, can bring them below that threshold, restoring metabolic control and avoiding complications. Again, it's such a glittering insight. For people who are slim, just check the waist.

Q4 You showed that remission can occur rapidly after substantial weight loss. Why do some people achieve remission and others do not, even when they lose similar amounts of weight?

There are some other factors. If someone has had Type 2 diabetes for too long, the critical recovery may not occur. The insulin-producing β cells in the pancreas, when exposed to excess fat for prolonged periods, may be too damaged to recover. How long is 'too long' varies. Genetics play a role. Some individuals may become unresponsive after as little as 3 years, while others can have diabetes for over 20 years and still return to normal. I have seen people with 23 years of Type 2 diabetes, treated with tablets, who lost weight, stopped medication on Day 1 of the diet, and maintained normal glucose levels thereafter. We are therefore dealing with individuals, not averages. Epidemiology has limited relevance in the clinic.

Q5 Your early studies used rapid calorie restriction to show that reducing liver and pancreatic fat could put Type 2 diabetes into remission. Do you see glucagon-like peptide-1 medicines as a transformational new chapter in diabetes care, or mainly as a more practical tool for achieving the same biological result?

Well, we're just at the start of this story, so I don't have any definite

views on this, but we can say several things clearly. The first thing is, these amazing medicines achieve what's been wanted for a long time, a direct effect on appetite. When appetite is wound down, everybody loses weight. Now, the importance of that critical observation is that it cuts through much of the confusion that has surrounded obesity research. There's no doubt that when you reduce body weight, you will produce the physiological changes that we have shown. And so, the reason for the improvement in diabetes is exactly the same as what we've shown.

The area is clouded by the manufacturers of these medicines. These drugs are often presented as acting through incretin effects on insulin secretion, but their primary impact is reducing body fat.

What will happen after treatment stops? There haven't been many studies of this so far. Weight often returns, because individuals have not learned how to regulate their intake. That learning cannot be taught didactically; it requires experience. If weight loss is externally imposed via medication, that learning process may be absent. We've got a learning curve here, and that learning curve is absent when people have been taking an injection and are basically having health administered to them artificially. They don't know this essential skill of how much to eat.

So, that's the problem. How can we approach it? It strikes me that there are at least two approaches. One is to try and mimic our food reintroduction programme, so that there is an intensive learning period with careful monitoring of body weight, with management by a doctor and nurse over the first 6 weeks of stopping the tablet or injection. The second approach is to consider continuing just a low dose of the medicine. Now, we don't know which is better, but there's one thing that's very human: many people with Type 2 diabetes absolutely hate their condition, but many patients wish to avoid lifelong medicalisation, which is an important consideration. This is something that doctors have been slow to realise by and large. This is a problem with using a tablet or a medicine approach. So, I see these drugs as being useful for diabetes potentially, but only when the learning has been appreciated and we've got in place some reasonable long-term measures.

Q6 The number of children and young people with Type 2 diabetes is rising in the UK. Are we seeing a fundamentally different form of Type 2 diabetes in adolescents, or simply earlier exposure to the same drivers?

It is earlier exposure to the same drivers. However, the consequences are more severe. Younger individuals are more susceptible to complications, likely because the vascular endothelium is more vulnerable to hyperglycaemia. Type 2 diabetes at age 20 years can lead to retinopathy within a few years and renal failure within a decade or so. By contrast, onset after age 70 years carries a far lower risk of serious complications. So, Type 2 diabetes in the young is extremely serious and, until politicians and legislators grasp that the food environment has to be legislated for, we'll continue to see this. Prevention is always better than cure, and we know how this can be prevented. It's not through trying to stimulate even more activity. Children leap around quite a lot anyway. What is abnormal is the dysregulated food environment.

The critical thing to mention to your politicians, your legislators, is that excess food is toxic. It's poisonous. While we regulate substances like lead, we have not adequately addressed the toxicity of excess caloric intake.

International food companies have been astonishingly successful in persuading politicians to look the other way. How often has the childhood obesity strategy been abandoned since it was going to be launched in 2017? Even now, with the banning of food adverts that might be seen by young people before 9pm, it turns out to only be the banning of images. The adverts can run if they're just in print and mention the name of the product. This is the kind of persuasive power that the big food companies have.

We have had a great start with the soft drinks levy on sugar. It's taken 37 tonnes of sugar out of the UK consumption every year, which is spectacular and has had an impact on childhood rates of obesity that is measurable. Politicians have got to be held to account, and we shouldn't be dragged into the folly of assuming that exercise will head this off.

Q7 Looking ahead,what important questions about Type 2 diabetes remain unanswered?

There are many, but, first, what is it about the β cell that causes it to be susceptible to fat? Can we detect these people in advance? Not everyone develops diabetes, even with substantial weight gain. Among White European populations, only about one-third will develop diabetes under such conditions; in some Asian populations, it is closer to half. This reflects genetic differences in personal fat thresholds. So,

what is it that determines that personal fat threshold and the initial susceptibility? The second question is, how can we measure the personal fat threshold? I've tried to measure it to get an index of how we could measure it with blood tests, but attempts to identify inflammatory markers have not yielded clinically useful signals, particularly in relatively lean individuals. So, this is an open question. At present, the most practical method remains observation: individuals can do it for themselves by measuring their waist circumference, substantial weight loss, and monitoring glucose response.

So, those are important things, but I'll circle back round to prevention. Preventing Type 2 diabetes is so important. The economic burden of overweight and obesity in the UK is estimated at 93 billion GBP annually, according to the Institute of Fiscal Studies. It gives an idea of the extent of what we're turning a blind eye to at the moment.

Q8 What practical tools and resources are available to support clinicians?

The NHS England national remission programme is now well underway, although eligibility criteria mean that not all patients qualify. I had lots of inquiries about this, so I put together a toolkit to be used by nurses and doctors in primary care.[1]

It provides background information, suggested language for patient communication, and structured guidance for implementation. There is also the book 'Life Without Diabetes',[2] which brings together the evidence and clinical approach, including a dedicated chapter on Type 2 diabetes in young people, and all the proceeds of this book go to Diabetes UK to provide research money not only for what we've already done, but for research in the future.

References

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