

Diet, Diabetes and Exercise

RM - Dr Richard Mackenzie, DM – Dr Duane Mellor.

RM Welcome to the second podcast of the Human performance and health series. The aim of this series of talks is help likeminded people bridge the gap between the hard science and its application. And what I mean by that is, what does this data and literature mean and how can we best use it in the real world

So the title of todays talk is more to do /with diet and diabetes risk and type 2 diabetes: What macronutrients should these people consume and avoid. We will also explore the use of exercise, in particularly high intensity exercise in these populations.

RM Firstly a couple of quick introductions. I'm Dr Rich Mackenzie, a senior lecturer at Roehampton specializing in insulin resistance in muscle and I'm joined here today by Dr Duane Mellor – please can you give us a quick background as to your qualifications and experience?

DM Thank you, I'm Dr Duane Mellor, I'm currently a senior lecturer in human nutrition at Coventry University. I've been a diabetes and nutrition specialist in the past. I undertook my PhD in diabetes and endocrinology – looking at the effects of chocolate in type 2 diabetes. Since then I've worked on various groups looking at dietary guidelines in diabetes with Diabetes UK which were published in 2011 and part of the new review of that is due for publication next year.

RM Thank you Duane. You've been a busy man.

RM During this podcast I want to cover a few key points surrounding diabetes risk (or pre-diabetes) and type 2 diabetes. First, it would be useful to define or discuss what type 2 diabetes is to avoid confusion.

DM

A classic definition of type 2 diabetes is chronic hypoglycemia or chronic high glucose levels. There's a couple of ways of diagnosing it – one is looking at a marker called HbA1C which is the haemoglobin floating in our blood cells and the more glucose we have in our system, the more sticks to it, there's a threshold for that.

A little bit confusing to look at a definition because there's a cut off 48 mmols per mols which suggests that not diabetes – I think it's somewhere in the 50s. So it's a bit vague. The traditional way is using glucose levels – fasting glucose levels at 7 mmol or above is diabetes and then you decide whether it's type I or Type II or gestations of any individual – gestation has to be present. Or the two hours after a glucose load of 75 grams being above 11.1 or a random glucose load being above 11.1...if there's one reading if you have no symptoms or if you have symptoms to suggest two readings – there's lots of caveats and little niggles to be clear on.

I think some of the things to think about are:

How easy is it to fast someone for a test, it's always a problem. So that's where the HbA1C is a good marker. Apologies for not remember the cutoff points for those – think 48 is the diagnosis one (Yes you're in the right area!) it's come back to me now – when you try to remember numbers when you're talking...48 is a diagnostic, 42 is not likely to be the case. One of the problems you get with some of these screenings as you've seen

in the past, is that they do a random blood glucose on people and they find it's not diagnostic. So does it mean that the person hasn't got diabetes or that they're not showing that a level at that time? So that's why the HbA1c can be a bit better – fasting is a hard thing to do – is it 10 hours, 11, 8 hour fast. Some people – their fasting glucose can be fine but their afternoon one can be very high so it's a bit of problematic one. So in developed healthcare systems such as the UK probably a combination of the two is the best way of confirming a diagnosis of diabetes. Type II is generally when there is still insulin production so there should be an absence of ketones generally with a few caveats and exceptions in that. And gestational diabetes is any diabetes which occurs in pregnancy so there's many nuances and it needs to be confirmed by a laboratory sample not by your friend's finger pricking machine testing kit at home.

RM What you have highlighted there even to someone who works in diabetes is that there's lots of different caveats, aspects and crossover from one to another. If we take Type II diabetes, generally it's the case of perhaps physical and activity, poor lifestyle and obesity linked but not always the case. In this population it's generally categorized by elevated blood glucose above the norm and within that – what do you think is causing the elevated blood glucose in Type II diabetes?

DM

Well it's an inability of insulin to actually go into the circulation so there's resistance to the action of insulin. It may not be deficiency of insulin, there may be a lack of insulin but quite often in Type II diabetes, higher levels are normal. I think probably one of the nicest theoretical models is from Professor Lloyd Taylor at Newcastle, the twins cycle hypothesis. And we spend a lot of time focusing on glucose, but often it can be fats, triglycerides not talking about cholesterol here, can actually accumulate in tissues it shouldn't be in. Quite often associated with Type II diabetes is excess fat in the liver.

The work in Newcastle has also suggested that there can be excess fat in the pancreas too, around beta cells, so in the liver it leads to liver resistance of insulin so the insulin doesn't work as well. The fat infiltration in the cells in the pancreas mean it can't secrete as well so the twin cycle. With some of his work looking at the very low calorie diets and rapid weight loss over 8 weeks, that shows some signs of putting diabetes into remission – using the word remission carefully, rather than reversing diabetes. But it reverses metabolic pathways so diabetes is in remission. But if lifestyle returns and the weight gain returns, the situation will return so not permanently gone away. Not a cure in my view based on looking at the signs. Puts in a control for as long as that lifestyle is maintained.

RM – that's a good distinction between the words because I've often used reverse and I often get asked about reversal of Type II diabetes. I think you're absolutely right. Looking at some of the work that's done on longitudinal studies where the weight comes back, that it's likely the metabolic dysfunction comes back. So Type II diabetes is complex, you mentioned the liver, pancreas, tissue, glucose and storage of triglycerides and fat in the different tissues. I think it's important for me to highlight that it's not a matter of one macronutrient so it's not one food type in terms of carbohydrates, fats, proteins, it's a complexity there. And how different tissue types respond to a given situation.

DM

Exactly and we tend to focus on diet or physical activity in isolation then obviously if you've got an exercised individual we know they'll deplete glycogen stores in the muscle and to a point glycogen stores in the liver, which will mean there will be less diversion of excess macronutrients whether carbohydrate, fat or protein which inhibit the fat storage pathways. So it's a combination of food and physical activity, not just either / or in isolation.

RM – yes, I completely agree, couldn't agree more on a calorie balance / calorie deficit being a healthy pathway / process to control for Type II diabetes. Thank you for that, hopefully that's cleared some things up for those who are listening.

So my main question for today and we'll see how we get on with this is the carbohydrate-insulin hypothesis. Can you describe what your understanding of that hypothesis is and why carbohydrate restriction might give people a metabolic advantage?

DM

Well the argument is that in theory if you consume carbohydrate, according to this theory that this leads to a rapid increase in insulin. Insulin is an anabolic storage hormone so anything that's not used will drive towards storage and fat accumulation which lead to the problems we mentioned before – the resistance of insulin and poor secretion of insulin leading to diabetes.

The problems with this argument is that it forgets the fact that things like whey protein will also cause a big spike in insulin and there were studies done in the 90s, in Australia, by Holt that showed that things like croissants - a combination of fat, carbohydrate and protein, gave the biggest spike in insulin per 1000 kilojoules (kJ). So food interacts and it's not just carbohydrate that increases insulin.

A few other problems. Looking at why insulin is high. If insulin in the unfed state (known as hyperinsulemia) is associated with disease risk, cardiovascular disease and diabetes insulin resistance. Having an efficient way of secreting insulin in the fed state to then process the glucose out of the circulation and the muscle and other tissues, that's a good response in a healthy individual that's sort of physically active – with the muscles, liver insulin responsive, that's normal. So the insulin will go through and go back down. There's a lot of confusion about this idea of 'carbohydrate drives insulin and that drives the obesity'. So it's all about hormones not energy intake, not about macronutrients.

The other problem with it is reductionism, I think one of the failings that we probably have in the field of health and nutrition is this reductionist randomized control trial model for too long. It works very well in medicine where you have a pill – if you go back to 'The Matrix' you have your red pill and your blue pill. And you take one - something has an effect, the other is a placebo. In food we cannot do that because you cannot just change one thing, it's a multiple, complex type of intervention. You can't just take a nutrient out and expect it to be a placebo because another nutrient will go in there. And if you take fat out of the food, what is going in place.

Often things are taken out of food and not considered their replacement. And that gives a lot of misleading data to get in nutrition so food is more complicated than looking at single items. Unless you're eating powdered glucose or vegetable fat, it's going to be a complex mixture. Even if you're just looking at something like an oil, where there's a combination of several different types of fatty acids in there, it may have some bioactives in there as well as different things like Vitamin E so it's still not simple with nutrient type food. So we've got this reductionist view of the world where it's complicated so that doesn't condense down very well.

RM – no, I'm agreeing with a great deal of what you're saying. Just trying to clarify some things for the audience. The understanding from this hypothesis is that carbohydrates are driving elevated insulin and therefore insulin is driving obesity. What's you're saying is that's far too simplistic in how food interacts. We consume food rather than a pure

glucose meal / carbohydrate meal vs a pure fat meal. How that mean interacts with the different tissue types is also quite important.

Is that right?

DM – Also things like physical activity will have a massive impact there. You know, it's not simple and using a simple analogy now which is probably counter-intuitive – if you view each tissue as having its own battery supply and that could be glycogen or other stores of amino acid in muscle fluxes for example. They go up and down as in the fed state. Exercise will cause a different change and you have fluxes in and out. So if you view each tissue as having a variety of carbohydrate battery in your muscle you've got your myosin and also fat, because fat stored in the muscle.

They will go up and down and they've been designed for a biological function – for survival, that we get food, that we replenish those stores – they deplete – we replenish them. Possibly the modern environment which means that we don't have to deplete our stores before we replenish – we're having a constant potentially replenished store and that then leads to metabolic disturbances, not one food per se. Demonising one food doesn't really help. It's looking at what have we allowed our food system to become, how do we influence that then how do we become consciously physically active. Adjusting and running down these batteries before we recharge them again.

So you can use the analogy of nuts and cycles on a battery – if you don't manage those properly, you shorten the life of the battery. We've got multiple types of battery in each tissue and our modern life doesn't suit them particularly well.

RM – yes, I'm very interested in picking up on that and I'm very interested in muscle. So I use an analogy of the muscle cell or the muscle itself which has a maximum storage capacity for energy and it really depends on what that muscle's doing as to the percentage of carbohydrates vs fats that it can actually store but once it reaches capacity it can store no more. And that's what we would very basically term as insulin resistance. And then if that's a chronic state we secrete more insulin to try and compensate for that – to move more of the glucose which is harmful for us from the blood compartment into the muscle. So that goes back to what you were saying about habitual elevated insulemia or elevated insulin being a problem that's a consequence of over energy storage. And we link that to obesity.

My question around that and I don't know if you've got an opinion on this is the effects of these types of diets that would have on the muscles. So let's say that someone is following a high fat, low carbohydrate diet which is seemingly quite popular – with the view that this type of diet isn't going to cause as many spikes as insulin. So insulin is lower for longer and the theory is that wouldn't drive this particular hypothesis to be seen in Type II diabetes.

From my earlier studies and speaking to other people that I work with, a state where there is low insulin also drives an increase in protein degradation and reduces protein synthesis so in basic terms, causes the muscle to release more amino acids and break down. So if you have less muscle then your battery storage capacity is going to be lower.

Does that make sense?

DM – It does and I think if we take a step back from a food point of view, if you look at why a low carbohydrate diet works – people talk about ketones and appetites and data from the same study showed conflicting results depending on how it was interpreted from the papers and presentations. So the appetite thing – not convinced. If you look at

whether there is any mechanistic advantage of a low carbohydrate diet it seems that it's easier to get a bigger deficit.

It's mathematical and that's the Bazzano paper – the same paper that's done the appetite. Where if you reduce the fat intake from 40% of fat to 30%, you get a 10% energy deficit if you don't replace it. If you do the same with carbohydrate, theoretically you get 50% down to 5% - taking 45% of the energy out of the diet, you've got a bigger potential. Obviously that can be added in as other foods. And so the question is when you say high carb fat, are you talking high fat as in the total number of grams or are you eating a lot of fat or because they've reduced the amount of carbohydrate to a very low level. The fat that's left over is a large proportion of a low energy diet. It's relatively high fat and that's where some of the confusion starts.

The other thing is with our modern food supply which has had 30 nearly 40 years of the low fat message, we've had an evolved ecosystem which has found effective ways of reducing fat, keeping them palatable and high energy. So you can replace with refined carbohydrates and sugar which possibly has a metabolic disadvantage. The main thing is it's easy to consume, cheap ingredient and it's added back into food.

There isn't anything magically evil about sugar, it's just that we like it, in a similar way, like fat. So by taking the fat out of food, you've created a niche to add in refined carbohydrates and sugar with it to make the food palatable again and taste like it will still be high in energy.

Is it by then taking the carbohydrate out, our food supply is inherently low fat, meaning that it's easier to get an energy deficit in the environment. What will be interesting in the future as we get more low carbohydrate, fat junk type foods – whether that message continues – that you can have free-for-all low carbohydrate diet and still lose weight. And we're starting to see some messaging coming round from the low carbohydrate communities – that you still need to watch things carefully.

I think the point you made about protein is also important because if you go to the physiology – the brain can reduce its demands on glucose, which is the reason for having carbohydrate and replace it with ketones which can make them fat. So the red blood cells can't do that because they can only metabolise glucose and there's certain parts of the kidney which do the same. So you need to make that glucose from somewhere. You don't physiologically need to get it from the diet but you do need to make it. You can make some of it from glycerol which is in the fatty acids which stick together. The rest you need to make from amino acids.

So if you've not got enough amino acids coming in from the diet, under the low insulin levels that is why muscle release amino acids. So you've got that pool of building products to make the glucose you need to keep your red blood cells working otherwise you die, your kidneys working otherwise you die and that residual requirement in the brain otherwise you die.

So it makes sense that low insulin levels release that whether having more protein in the diet or whether you'd just be eating expensive substrate to make glucose out of because you basically take the nitrogen off it and why that needs a less refined sort of carbohydrate in the first place. There is that question. There's a lot of debate you see in these forums with people wanting to eat low carb, dropping the protein or not dropping the protein. And I think that's where you've kind of hit on – that if you've got low sodium levels and you don't have enough protein in the pool, which with that diet you're going to start release more from the muscle; you don't have enough dairy which I haven't covered yet. We talk about adaptation and adaptation taking a long time. The studies

that are over a month period are the endurance walkers in a study done at IAS in Australia suggest that low carbohydrate diets are effective. If you actually look at the work done by Jeff in the US who's a low carbohydrate diet advocate, says there's some sort of equivalence of low carbohydrate diets in cyclists but their initial performance drops.

So there are some questions there and some people do seem to perform at quite a high level on very low carbohydrate diets. International athletes are doing this but whether it's the best way for them or whether they are just incredibly talented individuals that can perform anyway, we don't know.

RM – yes, that's another big question isn't it – the pre-exercise diet for performance rather than the part which we're mainly focusing on today which is health. From my understanding you're fairly on the mark in terms of – some people are just more efficient at turning over a fat molecule or fatty acids to produce much more energy at a given exercise intensity. That's another topic for another time perhaps, some really interesting points that you're making.

Just trying to draw some key information away from this and simplify things a little. Am I right in saying, from your understanding **the healthy active person, should they be at least considering a low carbohydrate diet or is the standard diet – the composition of whatever carbohydrates, fats, amino acids – that's recommended to most not something that they should be pursuing?**

DM – that is a million dollar question. I think we should move away from focusing on macronutrient ratios and looking at eating foods is my personal opinion. The PrediMed diet - a 40% energy from fat – the majority of that being from monounsaturated – olive oil, nuts, seeds - is a good one for long term health. That gave 40% of energy from carbohydrate which is slightly less than guidelines of 50.

Looking at how you formulate carbohydrate guidelines, quite often it tends to be – we know this level of fat intake is between 30-40%, it's gone up slightly with the PrediMed study being the best for cardiovascular health. We need this much protein, is that the optimum amount of protein to just avoid deficiency and the remainder of energy has been built from carbohydrate historically. We know that probably around 40% to a ceiling of around 50% in the average individual is probably appropriate. Exercise in the individual is going to vary according to their need. In diabetes probably over 40% is not unrealistic as a good starting point but then again you've got to think of the energy restriction to try and reduce – it's not just the carbohydrates, just the fat, just the protein you've got to try to bring down. So I think 50% is probably the upper limit for the average population.

I'd probably look at the PrediMed type diet which is a 40% and also confirmed by the big Swedish review of weight management. I suggest about 40% because longer term there is no evidence of benefit of reducing it down much lower.

RM – just for clarity, we're talking about carbohydrates?

DM – yes, for a not too active general population. I think the caveat there is that we should focus on food and not eating too much food. Eating more fresh and minimally processed food but some processing of food is useful to keep it safe. Less of the highly processed food, particularly refined carbohydrates and sugar. I think looking at the amount that we eat is also important. We've probably been a bit too cautious in the past about protein for health, probably 20% based on the data I've seen is sensible and probably try to split that between the plants and non-red meat sources as well as red

meat sources. Increasing the low energy density foods such as vegetables in particular.

And there's good evidence for the inclusion of leafy vegetables in the diet to reduce risk of diabetes than the root vegetables and the fruit so I think we need to get something that fits with again this Mediterranean style. Not saying a Mediterranean diet because you need to adapt to what that means for that person's culture, their lifestyle and their food practices. So it might be eating more cabbage than some of what you get in other vegetables in the Mediterranean diet.

RM – There's some good advice and what I'm gathering from what you've said – that a balanced diet rather than a low carbohydrate diet is the way forward, regardless of which states. And I'm going to use states in terms of the populations I mentioned before which is Type II diabetics, pre-TypeII diabetics and a healthy active person. And of course each different population will have different things to look at but it's on a person by person basis rather than a blanket low carbohydrate diet.

DM – and on a person by person basis I would see that some individuals might find it easier to manage their condition with a lower carbohydrate intake. There are cases of some people managing on a high carbohydrate intake, particularly looking at some data on vegan diets – they seem to do quite well so it's very much what are the best diets someone can stick to very long term and getting exact percentages of carbohydrate can be problematic. It's more finding a way of eating that helps you achieve those goals and not try to say one is better than the other.

It's the best one for you which fits with your way of eating – the sort of food that you enjoy. That could be a very low carbohydrate diet in some cases but I think for the majority – approximately 40% - it can be broken down to a simple message – the amount of carbohydrate should be pretty unrefined – wholegrain and round about the size of your fist on a plate – the clenched fist. It's probably a simple way of visualizing it. Then you have two good handfuls of the vegetables there and a palm size amount of meat or alternative – fish, lentils, beans.

RM – I'm listening to you and I'm nodding to myself – obviously you can't see me at the moment. I'm thinking about things that I've heard that frustrate me really quite passionately. I heard on an ITV programme with people that probably don't have the knowledge that you have, are saying things and I won't quote the person by name but 'glucose causes cancer' – so let's not eat glucose. And I heard on the radio today on my four hour commute from home that we should 'avoid all carbs' and I hate using the word or abbreviation 'carbs' , just because it's got such an evil connotation associated with it.

I wonder how much of the popular social media is playing by controlling or at least influencing people's diets. You have to be very careful about how that information is received because I don't think we can control its output too much because people will have their own opinions.

DM – I think there's a whole host of problems there, there's a misunderstanding of the biology with 'glucose causes cancer' and whilst it's true that some cancers may thrive very well on glucose but others will thrive equally well and divide on ketones. It depends on the type of cancer cell. That's been known for 40+ years and we know that one of the biggest risks for cancer patients is malnutrition so we need to weigh that up as well. Fortunately in the UK we have the cancer act so if anyone does promote a low carbohydrate diet which doesn't have the proof in humans at the moment. Some interest in the brain tumour areas particularly on symptom control. In general cancer treatment they are probably committing an offence and it needs to be taken seriously.

I think the idea of social media is an interesting one because quite often these messages are being spun quite tightly – conscience bubbles, echo chambers. The problem is when food retailers and their staff start listening to these and then it gets amplified onto our supermarket shelves which isn't particularly helpful. The other one is when you get influence of politicians who are promoting potentially expensive low carbohydrate diets when they've got massive poverty in the cities they are serving. That is when we have real issues and we need to stand up and say this is not realistic – the average person in a British city cannot afford that way of eating. They're struggling to eat as it is. If we forget those basic things because of the position of privilege of those people that can afford.

RM – yes, I'd go one step further and say these echo chambers are influencing the marketing of certain foods, certain diet and the supermarkets are following that – that's a really big problem and something that needs to be addressed but it's probably well beyond our grades !

DM – personally I don't have a problem with corgetti – you can buy it and make it yourself but instead of replacing your spaghetti completely with it, why not mix whatever corgette, carrot or other vegetable you want to spiralise with normal pasta – ideally wholewheat pasta. You're reducing the energy density of the meal, you're adding more vegetables and you're getting more colour. You don't have to go all that way and you'll still improve the quality of the diet and hopefully it still tastes good.

RM – yes you've used one very good example – corgetti – it's a shame we don't have a picture of our meals tonight to post on Instagram or twitter. I just want for the last five minutes to consider exercise and I know we need to consider exercise in terms of one part of the energy balance and it also needs to incorporate the individual. I wanted to cover high-intensity exercise and in the same way that corgetti might have had a lot of media coverage, so has high intensity exercise. It's very useful in terms of the short duration, you can do less in terms of time but potentially expend more energy.

I wonder what your experience is on that type of exercise in a Type II diabetic population?

DM – I had 3 students who looked at metabolic syndrome traits and the literature on this a couple of years ago when I was at Nottingham. They actually dug into the research and one thing they found is that some of the reporting of the data was not very good. There were some of the statistics which were questionable in terms of the some of the standard deviation scores – the data did not look right in the paper, an implausible spread of the data. So either the sample was not large enough to make it meaningful or they confused how they'd analysed it. So that made the interpretation of the data quite hard – we kept strictly to some criteria and we found that – hardly surprising – exercise is good. I think that was the message.

HIT (high intensity exercise) – some were a little bit better, some were not quite the same but if you find it easier to do that really intensive exercise, there's no physical reason why you can't do it. And if it fits into your lifestyle, then great. If you prefer more of a stroll and you've got time to do it, then great – do it. I think we get too hung up and I guess it goes back to some of this stuff about macronutrients. We get very hung up on – this is better than that – when the fact is – we just need to get moving more. The detail is what fits with your lifestyle, what can you continue doing for a long period of time because that's what you need to do to get healthy. There's some minor bits and pieces with responders / non-responders and how you're measuring it is wrong with all the scientific navel gazing. The bottom line is – we just need to do something. Lifestyle around diabetes needs to be physiologically designed to consume lots of energy whilst

not able to move too much – our modern lifestyle. Choosing better options for food and then deliberately taking exercise when we don't have to. We can ask people to design our cities in a way to make that better but we also have to design our workplaces and our job roles to encourage us to move more frequently.

I think that's not very exciting because HIT is amazing – you can do everything you need to do in a couple of minutes but if you don't want to do that and want to go for a walk at lunchtime and you've got an hour – then do it.

RM – that goes back to a paper I read a long time ago by a guy called Frank Booth and I recommend to anyone. He did two papers 2000, 2002 – 'Waging the war on modern disease with diet and physical activity'. How we are designed for physical activity and we are designed to consume energy when its available. The problem is that now energy is very much available and the other side of it is that we do a lot less than we used to, therefore there's this imbalance in terms of energy and how we respond to it. Excessive energy in our bodies.

The other point being that HIT exercise and I'm probably being guilty as some looking into it, it can be useful but the key there in the popular media is that its sexy and it sells so therefore it gets a lot of attraction. Looking at a paper that we're just putting out at the moment, I won't plug it too much but I compared different types of exercise and pre-diabetics that are typically hyperinsulemic chronically and I wanted to understand what this type of exercise would do to them. In the short term, if the person is responding to the stress then HIT exercise will help them secrete more insulin. And I wanted to have a look at the effects that would have on the different components that causes Type II diabetes.

What I actually found I didn't expect because I come from the same mindset as yourself, is that HIT decreased hepatic glucose production . For people who don't know what that is, when we go from healthy to pre-diabetes to Type II diabetes, our liver stops responding to insulin as Duane mentioned earlier and the offshot is that it produces more glucose. I found that this type of exercise reduced the amount of glucose in pre-diabetics which is quite interesting. But I generally come back to the point that it doesn't really matter about the type of exercise, it comes more down to the energy deficit at the end of the exercise that's important long term.

I've realized I'm waffling on now and I've taken up quite a bit of your time Duane. I just wanted to finish off with a quick question before thanking you for your time.

What do you think the biggest problem is for controlling the progression from obese individuals to Type II diabetes in the modern society?

DM – I think Dr [Harry Rutter](#) has a complicated map with the individual in the middle and you've got activity, environment, food, society. You've got all these things – this complicated network of impacting factors. If we're going to do anything about it, we need to change how we view food, we probably need to look at moving from a consuming society to a more thoughtful society in how we eat. To enjoy food more, rather than just consuming which is philosophically quite challenging because it goes against the market forces we currently have set up. We need to make physical activity enjoyable and the norm. I don't think we can get people to eat better and be more physically active unless that's enjoyable and pleasurable. We can't keep telling people to stop doing what they're doing, behave themselves and they'll be healthier or they'll die because people don't like negative messages. We need to make the healthy choice more pleasurable and give it positive outcomes. We need to repackage, resell and learn from the marketeers about how we market healthier lifestyles, to adapt how we live.

Otherwise we're going to keep hitting the same problem. The reason obesity is rising is not because of carbohydrates, it's because our food environment is changing and we need to start reversing the way we're consuming. How we do that is complicated – taxation is a tiny part of it, reforming a tiny part of it, active cities a tiny part of it. We need lots of these tiny parts to make a big difference. And that's possibly why when individuals go from obesity to Type II diabetes, the all-or-nothing low carbohydrate diet works for them because it actually fights against the system and allows their bodies to change.

We need to get the food system as a whole to allow that to be easier rather than individuals having to make choices. It will work for a short time but may not work long-term because their enjoyment of food might not be there. For individuals who enjoy that type of diet – great but we need to make the whole food system, the physical activity system more suitable for a healthy lifestyle. And this is dreaming but maybe in a system where we don't look at financial equity but health, sustainable equity and social equity in equal values.

RM – you've covered some really good points and I picked up a few but I won't dwell on them because of time. The fad diet can be a problem, it can work for some people and for a short period of time but it's not a long term solution to the problems we face because of the food environment that you mentioned. Unless we change that radically, nothing is going to change in your opinion.

DM – we need to change how we see food and look at ways that we can enjoy being healthier. We can't do it by sanctions and threats, we don't respond well to being threatened.

RM – thanks Duane, we actually ran over, I know you've got some lectures to run to and I have too. I really appreciate your time and I hope to get you back to talk about some other issues. Thank you again Duane, it's been very interesting.

Duane Mellor links:

- <https://theconversation.com/profiles/duane-mellor-136502>
- <https://pureportal.coventry.ac.uk/en/persons/duane-mellor>
- [Am J Physiol Endocrinol Metab.](#) 2017
- Most recent book chapter ([Macronutrient composition for weight loss in obesity](#) (*Advanced Nutrition and Dietetics in Obesity*. Hankey, C. (ed.). [Wiley](#)

Frank Booth

- Waging war on modern chronic diseases: primary prevention through exercise biology: <https://www.ncbi.nlm.nih.gov/pubmed/10658050>
- Waging war on physical inactivity: using modern molecular ammunition against an ancient enemy: <https://www.ncbi.nlm.nih.gov/pubmed/12070181>