Ivor Cummins 00:47 I'm here in the Royal College of general practitioners and I have a very special guest today who I'm honored and privileged to finally meet with. Dr. Robert Lustig, welcome.

Robert Lustig 00:59 My pleasure. Thank you for having me, Ivor.

Ivor 01:01 Not at all. Early in my research journey way back, I found pretty early on your bitter truth. It's now at 8 million views.

Robert 01:08 So I'm told.

Ivor 01:09 Yeah, I checked last night. And it was incredible. I was enthralled because it actually appeal to a mass audience, which is highly unusual for such a complex lecture. For someone like me who is biochemical background, it was... I watched it several times in a row. And I'll have to admit, I used your slides in my talks with engineers without getting permission.

Robert 01:30 It's called the academic binge watch.

Ivor 01:33 Oh, big time. Yeah. Big time.

Robert 01:34 Yeah. I have no idea why anyone would watch a 90 minute lecture on carbohydrate biochemistry. So the fact that anyone watched it was surprising to me. Nevermind 8.2 million. I mean, that's just crazy.

Ivor 01:49 It's crazy but it's crazy good. The passion actually was part of it, because most lectures in biochemistry obviously are “dry as sawdust”. The passion was enormous. The conviction. That was a big part, I think.

Robert 02:02 Well, the thing is… look, I'm a practicing physician, and a scientist. You know, for every one patient I took care of and got better, 10 more would show at my door. There was no way I was ever going to fix this. And the thing that really, really bothered me was I learned virtually everything I know about nutrition in college, because I majored in Nutrition and Food Science at MIT. And then I went to medical school and they beat it out of me, and told me that everything I'd learned was irrelevant, had no place in patient care, and it wasn't

necessary and that really all I had to do was focus on calories. I figured, well, these are the clinicians, you know, I'm going to be close, I better like listen to them. And so I practiced that way for like 20 years and then I started doing research because my patients weren't getting better. And I started doing research to try to figure out what's going on and it like all came rushing back to me, kind of like Post Traumatic Stress Disorder. It's like, "Oh my God, I knew this stuff back in 1975." So I got pissed off.

03:10 So I think part of the passion actually is sort of being dumbfounded, and the anger of what I see going on in medicine today. So, I'm glad it translates in a positive way and that people appreciate the passion. But I'm just, like really ticked off.

Ivor 03:31 Yeah, and rightly so. And I know when I originally discovered in my own small way that my blood tests were way out of whack, and three doctors in succession couldn't give me any convincing feedback, when I actually discovered how it essentially worked. I was really angry too, because I thought of all the hundreds of millions of people who are suffering, whatever that may, and no one seems to know how it works even at a basic level. Which is crazy.

03:58 So one thing that occurs to me and a couple of people have wondered it too, so I'll ask it. That original video, and the fat chance was the bulk essentially around that. Years later now, is there anything in there that you would change, which are further research or possibly emphasize in different relative ways, do you think?

Robert 04:18 Well, we have more data. If I rewrote it today, there'd be so much more that I could add, you know, in terms of fuel to the fire. There's nothing in the book that's wrong. So there's nothing I would retract. I would add certain things. And I've also recognized that the role of fiber, and there's a whole chapter on fiber, so it's not like I discounted it. But I've come to realize just how important the fiber story really is. Ultimately, I can sum up healthy eating in two clauses: protect the liver, feed the gut. If you protect the liver so that it's not getting the tsunami, a mono and disaccharides, that come from ultra-processed food, digestion and absorption early on in the duodenum, then you protect the liver. If you can move that food through the intestine, so that it doesn't get absorbed in the duodenum and gets further down to the jejunum so that the intestinal microbiome can chew it up. That means even though you ate it, you didn't get it. Since this is a macro nutrient

excess problem, this chronic metabolic disease problem, if you can get your bacteria to chew it up instead of you, then it doesn't really matter what past here; it matters what past here, Right? It doesn't matter what you ate, it matters what you absorbed. So if you feed the gut, you solve the problem. Protect the liver, feed the gut. Real food does that. Processed food doesn't.

Ivor 06:00 Yeah. I love the way you summarize that. And also, of course, when the bacteria take these foods that do pass down to the lower intestine, you know, they will make beneficial things.

Robert 06:09 They make short chain fatty acids even more to the point. Because those are anti-inflammatory and anti-insulin, they suppress insulin. So yeah, short chain fatty acids have turned out to be an enormous help in terms of understanding the role of the microbiome. It was just an article in I forget, it was nature, on the role of short chain fatty acids and where they come from and how they work. And basically, you got to get the food down there in order to be able to get those bacteria to do it. And they actually use soluble fiber as their substrate for turning into short chain fatty acids. So again, I think there are a whole host of reasons why real food works. And there's a whole host of reasons why process who doesn't. And that is the crux of the problem. And the question is, how are we going to fix that especially when the food industry doesn't want to fix it?

Ivor 07:02 Yeah, that is a killer. Now, I actually ended up in RTE, Ireland's primetime television in a debate last year with a professor of nutrition, who actually works with the ILSI, the International Life Sciences Institute and has published multiple papers, essentially attacking and undermining De Novo European body who are trying to simply categorized ultra processed foods so there is some guidance.

Robert 07:31 Very aware. So there's a paper be coming out shortly about in the UK, looking at the various swaths of De Novo classification. So going from non processed all the way up to ultra processed. And basically 56% of the calories and 62% of the sugar in the UK diet is in that ultra processed food category. That is the goal. That is the target. That is the problem. That ultra processed food category. And the question is, what do you do about it? Because that's where the money gets made. And it's even worse than that. Because if you take those ultra processed foods, only 19% of the dollars you pay for pays for

the food. 40% pays for the marketing, and the last of the 40%, that's their profit.

Ivor 08:23 Wow, I actually didn't really have any of those figures. Now, I would have guessed it, not a mile off that because I know it's insane but it's great to hear those figures actually quoted. Shocking, really. And you're absolutely right, Robert, well you're obviously right, but the thing about the processed food industry, they need shelf life, they need to scale enormously across the world and they need dirt cheap ingredients and refined carbs, grains, seed oils…

Robert 08:50 ... doesn't come any cheaper.

Ivor 08:51 … it doesn't come any cheaper either. And also, the shelf life…

Robert 08:55 It also doesn't come any more dangerous.

Ivor 08:58 Yeah, absolutely. And actually, circling back what you were saying about a duodenum and that I had interviews recently with Gabor Erdoshi who's an extraordinary individual and hungry who does deep research, but he exposed me to all of the GIP in the upper intestine and the over exciting of that, versus proper foods that go down the intestine and excite GLP-1 and PYY gets satiety and all the benefits. So maybe talk a little.

Robert 09:25 I mean, they increase insulin release, but they go to the brain and reduce total food intake. So yes, I mean, GLP-1 agonists are now being used for diabetes and showing some effects on weight loss. They're now, you know, primary mode of therapy for type 2 diabetes in the United States.

09:45 My point is, yes, that's great and wonderful, except we could be doing that so much cheaper, so much easier, and so much across the board. And even though these GLP-1 agonists existed, they're breaking the bank. So this is not a sustainable method for dealing with the problem. Ultimately, we cannot treat our way out of this problem, we have to prevent our way out of this problem. It's the only way to be able to recoup medical resources that are being thrown at this that's breaking the healthcare bank and the budgets of every developed and developing country around the world. There are no options.

10:27 When you look at the six cellular pathways within the cell that are associated with longevity, they are also the same six cellular pathways of chronic disease. They are glycation, oxidative stress, inflammation, mitochondrial dysfunction, insulin resistance, and membrane instability. When you look at the actual metabolic pathways of each of those, none of them are druggable, except maybe inflammation and that's actually downstream of the others. They're all “foodable”, but not druggable. And they're only “foodable” with real food. In fact, processed food is what actually causes those dysfunctions.

11:11 So when we treat various diseases like GLP-1 analogues for diabetes, or any hypertensives for high blood pressure, or hypoglycemic agents for, you know, high hypoglycemia or statins for LDLs, basically what we're doing is we're treating symptoms of a disease, not the disease itself. And so if you treat a symptom and you haven't actually fixed the disease, guess what? The disease is still there.

Ivor 11:37 And that's essentially…

Robert 11:39 … getting worse.

Ivor 11:40 Yeah. And if you take fructose then which is a major problem in processed food and not in real foods nearly so much, obviously, and you take refined carbohydrates or essentially glucose, fast flash glucose, they'll hit the GIP-1, how would you see those in terms of deleterious potential? So a lot of fructose or a lot of refined glucose, or is it really the mixture together with one pushing up insulin and the other acting true ATP in the liver?

Robert 12:12 In fact, you've got it exactly right. They both are problematic. It's not that one is safe and the other one is not. They’re problematic in different ways. So, glucose is the energy of life. Every cell on the planet burns glucose for energy. Glucose is so important that if you don't consume it, your body makes it. So people on ketogenic diets still have a certain glucose level. The Inuit, they didn't have any carbohydrate, they didn't have any place to grow a carbohydrate. They had whale blubber, okay? They also didn't get cancer or heart disease. All right? The fact matters, they still had a serum glucose level. So it's proven back in 1928, that they still had a serum glucose level. So where the glucose come from? Well, it's so important that their livers turned fat or protein into glucose so that their bodies could and would function.

13:04 So it's not that glucose in and of itself per se is problematic. It's the insulin response to glucose. That is problematic because insulin while lowering blood glucose also causes cell proliferation, causes vascular smooth muscle proliferation, causes cancer cell promotion. So glucose causes insulin secretion, and it's the insulin secretion that drives these chronic metabolic diseases and also drives weight gain.

13:40 So it's not like glucose is off the hook. But glucose is a walk in the park compared to fructose. So fructose does not generate an insulin response. Unless you so overwhelmed the liver that you get a certain fructose level. In which case then, that fructose circulates goes to the beta cells of the pancreas. And you do get an insulin response because there is a fructose receptor in the beta cell, which only kicks in when your liver gets overwhelmed. And then you've really got a fructose rise. But mostly what happens is the fructose goes to the liver, overwhelms the livers capacity to metabolize it, turns the excess into liver fat through this process called the novo lipogenesis that we've studied, and that liver fat accumulation causes liver dysfunction and insulin resistance.

14:38 So, glucose causes insulin secretion. fructose causes insulin resistance. They both end up with high insulin levels, but for different reasons, and ultimately have different implications for chronic metabolic disease. So they're both important.

Ivor 14:56 And especially as both synergistically together overloading both together is the worst case. And that's most processed foods.

Robert 15:03 Absolutely.

Ivor 15:03 Have an element of that. And sucrose itself of course is the straight 50/50.

Robert 15:07 Absolutely! And high fibrous corn syrups are even worse.

Ivor 15:10 Yeah. Oh, it's around 65 or something fructose.

Robert 15:13 It can be. It can be up to 65% fructose depending on which distributor and which food maker. Yeah.

Ivor 15:20 Yeah. Well you actually mentioned insulin or hyperinsulinemia and some resistance, obviously a favorite

topic of mine. Dr. Joseph Kraft who died last year, who did the 15,000 Insulin Assays, his quote, one of them I loved was, “Hyperinsulinemia and insulin resistance, they are not combatants, they are one and the same.” What he really meant is for pathological states they're two sides of a coin. But on that thorny question, well for some people who are nerds, is it more the hyper insulin, the getting the insulin resistance with time or insulin resistance building which can happen for other reasons driving hyperinsulinemia? Or is that kind of both together?

Robert 16:01 Yes. It’s both. It can go either way. So insulin resistance at the level of the liver can cause hyperinsulinemia and chronic disease, or insulin secretion at the level of the pancreas can drive weight gain, which will then lead to insulin resistance too. In other words if you have a circle of insulin, insulin resistance and weight gain, it can go in either direction. And different people go in different directions. And to be honest with you, in terms of treatment, you actually have to be able to parse that. You have to be able to figure out where the origin of the problem is in order to direct your therapy to that target in order to get beneficial effects. If you enter the pathway at some other node, it won't work. And this is why it's so essential to understand you know, nutritional biochemistry completely and also see each patient individually and know their physiology in order to be able to target the therapy to the pathology, which is what we did in our obesity clinic every day for 17 years that I ran it.

Ivor 17:10 Excellent. Yeah, Rob. And I often say to people when I'm trying to explain, the arrow of cause can go in multiple directions depending on the scenario. And exactly if you don't, for an individual case, if you don't find out where you are in it, you know, you're not going to be effective.

Robert 17:27 But the great thing is that real food works for virtually everybody.

Ivor 17:31 Yes.

Robert 17:32 Now, there are outliers. There are people where real food is not going to be the answer. They have maybe genetic defects or epigenetic abnormalities or potentially developmental programming issues, or something even more extreme may be required, including, you know, shall we say altered or multimodal dietary therapies and/or drugs, and we use them as we needed to, but if you're looking for a general

public health message that will help virtually, I would say 90 to 95% of the population right off the bat, it's real food.

Ivor 18:10 Yes. And myself and Dr. Garber's who say three eliminations - sugar, refined carbon seed oils, and then processed food is…

Robert 18:17 That is processed food.

Ivor 18:18 That is processed food. So it's a process food, it's stuffed with all three is what I always say. If people only did that, yes, there will be people who have diabetic dysfunction who can benefit from low carb and keto, you know, or weight loss and all that's true. But for the vast majority, just that switch away from processed food would have an enormous bang for the buck.

Robert 18:39 Exactly. So I think there are people who require a, shall we say very low carb or even sometimes ketogenic diet in order to suppress their insulin so severely that it's not becomes necessary in order to turn them around metabolically. And I have taken care of patients in clinic who had insulin responses to an oral glucose tolerance test that were in the hundreds, even thousands of serum insulin at 30 minutes of 900 and then at 60 minutes of 1300. Enormous insulin responses to glucose. We take those patients, we put them on a very low carb or even in some cases ketogenic diet, and the process reverses. And the parents kiss my feet.

Ivor 19:30 Wow, that's so rewarding to be able to take such entrenched cases that the vast majority of doctors couldn't help with, Robert. The vast majority of doctors and medics and even specialists, endocrinologists do not really have a full grasp of what you're saying.

Robert 19:47 Unfortunately, they still think it's about calories.

Ivor 19:49 Or glucose, blood glucose.

Robert 19:51 Well, so… and I will tell you that we have a problem. And you just mentioned it. We have this thing called sugar, and sugar really has two definitions. There is blood sugar, and there is dietary sugar, and they are not the same. Blood sugars, blood glucose, dietary sugar is glucose fructose. We treat them like they are the same thing. They're not. We also do the same thing for another word. Fat. We have body fat and we have dietary fat. And they are not the same. And within dietary

fat, we have a whole host of different things like for instance, saturated fat and saturated fats, not one thing. It's two. It's red meat saturated fat, and it's also dairy saturated fat. And they're not the same. And then of course, we have a whole host of other fats like omega 3s, monounsaturates, polyunsaturates, medium chain triglycerides, omega 6s, and of course, the ever ubiquitous and ever demonized transfat.

20:55 So, the fact the matter is, fat is not fat. Sugar is not sugar. A calorie is not a calorie. The only way that doctors can help their patients is to understand nutritional biochemistry. Yet, it is the one thing doctors don't learn in medical school - nutrition.

Ivor 21:14 And it will be slow to change in fairness.

Robert 21:18 Well, we're trying to fix that.

Ivor 21:20 Yeah.And if we just take the fats there and there are myriad different fats and they've been like stearic acid is supposedly not problematic, and this other one is and all that kind of thing. There's a lot of science.

Robert 21:34 Well, we think palmitate is particularly egregious compound. But palmitate really does not come from your diet. It comes from the novo lipogenesis, is the sole fat that the novo lipogenesis makes - free palmitate. We think that's the particularly egregious guy in the story.

Ivor 21:57 And I know Professor Volek did some beautiful work in human studies, and that feeding very high saturated fat, low carb, and the blood flat levels of palmitate were way lower than the healthy, high carb low fat diet.

22:47 But there's so much data now actually, we in the sense, my own sponsor David Bobbett, Irish Heart Disease Awareness, he feels that what's already in the literature, there's plenty there to answer these questions.

Robert 22:58 There is.

Ivor 22:59 More research is not really going to change much. It's a battle not based on producing more papers per se.

Robert 23:06 So this is a problem of science is what level of proof do you need to act? Where is the line? And I will tell you

that the food industry moves the goalposts. What they say is, “We need randomized controlled trials,” and then you produce the randomized controls, “We need longer term,” “We need something,” because they don't want to change. They're doing their absolute level best to maintain their position and their market share. So, they can always say, “We don't have enough research.” And you know what, we'll never have enough research, but that doesn't matter. The point is, at some point, you just have to set a bite the bullet and say, “When do we have enough to act?” And the answer says, “We have way enough to act now.”

Ivor 23:52 It's very reminiscent of the tobacco, a playbook of course.

Robert 23:56 It’s tobacco all over again.

Ivor 23:58 And I believe that the tobacco people moved into processed food. I interviewed a lady…

Robert 24:03 Actually, it's really the other way around. The processed food people moved into tobacco. So name was John Hockett. He was an MIT professor. I hate saying that since that's my alma mater. But he actually worked for the sugar industry and then went to work for the tobacco industry in the 1950s.

Ivor 24:22 Okay. Yeah, there may have been cross pollination then back the other way. I think when tobacco got a bit squeezed, I think tobacco companies began to invest in processed food.

Robert 24:33 Absolutely. Altria, you know, Philip Morris was craft, in general foods. R.J. Reynolds did that, Nabisco, you know? Absolutely, they were diversifying their portfolio and they were bringing what they had learned in terms of tobacco to how to market processed food. Snackwells, perfect example.

Ivor 24:53 And I interviewed a lady, a fantastic lady Joan Ifland last well.

Robert 24:58 Oh! Very well.

Ivor 24:58 I was guessing you might. And we had a lovely interview. She was fantastic talk.

Robert 25:03 Lovely lady.

Ivor 25:04 Yeah, and she has this textbook, an actual textbook now not a populist, “Processed Food Addiction.”

Robert 25:09 Correct?

Ivor 25:09 Yeah. And that sounds like one I really gotta get a hold on.

Robert 25:11 We're actually going to be using that as a textbook at Touro University, California, where I teach. It's an osteopathic school in Vallejo, California.

Ivor 25:20 Excellent. Yeah, because she spoke highly of you. But it was briefly in the conversation amongst other things, and I didn't know how well you're connected.

Robert 25:27 We are connected.

Ivor 25:28 Lovely. And if we take, just things popped into my head, but what pops into my head is we have the sugar and refined carb explosion, and lust and fiber and processing and food in the 20th century, and we've got our epidemics, diabetes, obesity and all the rest. The seed oils, the soy and not just trans fats, but omega six rich seed oils have also gone up by an enormous magnitude and there's a lot of science around there being obesogenic. I mean a lot of animal studies and very impressive ones. How do you feel those to stack up as causal?

Robert 26:05 So it's very (interesting), I mean, this is a complex subject with a lot of data and a lot of people on either side of the argument. I've talked at great length about this with my colleague, Dr. Dariush Mozaffarian at Tufts, who's probably the world's expert on this. We all agree to a nutrition researcher, you know, that omega 3s are good. We all agree on that. No one doubts that. The question is are omega 6 is bad? And you know, people have talked about the omega 6 to omega 3 ratio. It used to be 1:1, now it’s 25:1. We also know that omega 6s are the precursors of arachidonic acid, which is, you know, the precursor to prostaglandins involved in inflammation. And we need inflammation because we have to fight off the maggots, you know? You can't not have inflammation, you have to have it. The question is how much of it and does increased omega 6 drive it? And the answer is maybe. We don't know that. Dari thinks not. He doesn't think. He thinks that omega 6s are not nearly as bad a rap as people give them.

27:23 Here's the thing that's really important. Omega 6s are in everything, because they're stable, they're cheap and they don't smell. Omega 3 smell. They smell like fish. So you don't find them in a lot of processed foods. We use omega 6s, they don't smell. The problem is when you take an omega 6 fatty acid and you heat it, you'll supply energy to that double bond and you will basically transit. You will turn it into a trans fat. And because so much processed food has so much omega sixes in it, and because they've been subjected to to heat a various methods, you know, in terms of the processing, it very well could be that those omega 6s are now trans fats. And that's the reason so that it's actually an epiphenomenon of you'll make 6s as being high in these foods, but it's actually the mechanism is that of a trans fats. We don't know that.

28:23 I think there are data implicating omega 6s in disease. But what I don't know is, is it really the omega six or is it what happened to the omega 6? Because food processing, food engineering matters in this case

Ivor 28:38 But I expect that the food manufacturers and producers of vegetable oils should be extremely helpful to help us get this answer. They will they would want to sit on that.

Robert 28:46 Don’t bet on it.

Ivor 28:48 I know. Sarcasm there for sure. No, I hosted the debate between Dariush and Gary Taubes a month ago and it was very interesting because we kind of went down a bit of a rabbit hole in that and of course I had to bring up Sydney heart and Minnesota and the Helsinki Businessmen Trial. All the trials were… the extra non trans generally it's assumed went the wrong way.

Robert 29:11 Well, we don't know.

Ivor 29:12 But we don’t know for sure.

Robert 29:12 We don’t know. I mean, I understand. But you know, what you think you know, you don't know.

Ivor 29:21 I think the most we know about those oils, even non trans is from animal experiments, and there's a lot of those, the carcinogenesis and the obese genesis. But they are animals. So we don't know yet what to watch.

Robert 29:33 It's complicated.

Ivor 29:34 Yeah, yeah. So… oh, you had to study out one of the things since your book and the lecture is certainly, you did run a small study on children with the key intervention of removing fructose rather than adding in a lot of things. Maybe just run through that.

Robert 29:49 Sure. So what we did, and I'm very proud of this study, and it's the gift that keeps on giving it - the data, just phenomenal. What we did was we took 43 children from our obesity clinic at UCSF with metabolic syndrome. So obesity plus at least one comorbidity. Latino and African American. Well, high processed food consumers. And what we do is we figured out what they were eating on their home diet. We studied them on their home diet. And then for the next nine days, we catered their meals. No added sugar. We gave them fruit. That was their sugar, but no other added sugar in any of the foods that we catered.

Ivor 30:32 Which would be a big change for them, I'm guessing.

Robert 30:34 It's a 350 to 400 calorie reduction per day. Now, we took their percent of calories as sugar from 28% to 10%. Now if you do that, you're losing 350-400 calories. That could cause weight loss. And so if the patient's got better, people say, “Well, of course they got better, they lost weight.” We didn't want them to lose weight. So then we had to re substitute the same number of calories we were excluding as sugar in something that was equity caloric so we gave them processed starch. So, in the vernacular, we took the pastries out we put the bagels in. We took the sweetened yogurt out, we put the big potato chips in. We took the chicken teriyaki out, we put the turkey hotdogs in. So we didn’t give them good food, we give them crappy food. Give them processed food. We give them kid food, food kids would eat, but it was no added sugar food.

Ivor 31:32 And specifically it was not resistant starch and good starch as it was. As you say…

Robert 31:37 Not at all.

Ivor 31:37 ... the usual junk…

Robert 31:38 … usual junk, just not fructose. Glucose for fructose exchange, purely glucose for fructose exchange, no changing calories, no change in weight. And we then re studied them 10 days later on this diet. Every aspect of their metabolic

health improved. Blood glucose went down 5 points. Blood insulin went down 25%. Triglycerides went down 46%. APLC went down 49%, which is huge.

Ivor 32:12 Yeah, it's big.

Robert 32:14 And most importantly, we studied their fat depots. So their sub q fat didn't change at all because they didn't lose weight. Their visceral fat went down 7% and that's good. Their liver fat went down 22% and the change in the liver fat predicted the change in the insulin response. And now, we've just published a fourth paper on this study that just came out two weeks ago in Journal of Clinical Endocrinology Metabolism, where we have actually found the toxic metabolite in the liver that fructose drives to cause the novo lipogenesis and the insulin resistance. It is called methylglyoxal. It is an alpha dicarbonyl which means it is 250 times more dangerous than glucose at forming the mired reaction, like a drowning reaction. And every time that happens, a reactive oxygen species gets released, which has to be quenched. And if it doesn't get quenched, guess what? It drives all these chronic diseases. So we now have the toxic metabolite. We know why sugar is doing this. We know why sugar is bad for you. We know why sugar is toxic, that is detrimental unrelated to its calories, and we know why everyone's getting sick. And we know what to do about it, too. And I'm very comfortable with this.

Ivor 33:40 Excellent. And I was told this morning, and I'm very conscious you have to go back shortly. But I missed your talk. Because I flew in from Ireland. I was late, but I was told that you had revealed that exactly what you just said.

Robert 33:51 We're very excited. Because, you know, obviously, people have given us a lot of flak over this notion that, "How can sugar be toxic, it's just empty calories. Oh, no, no, no, no, not at all." It is metabolized differently. And one of its metabolic byproducts is toxic. And we have it.

Ivor 34:12 And now that you have it, you can expand the research and follow down the rabbit hole of… are you actually already know a lot of the rabbit holes?

Robert 34:20 We've already excavated the rabbit hole. The problem is the food industry tries to keep filling it in.

Ivor 34:28 Yeah, and they've got a lot of shovels. They've got mechanized shovels.

Robert 34:31 They sure do.

Ivor 34:32 Excellent. Well, I tell you what, we'll wrap it up one moment. There's one last thing. Oh, yes. Hyperinsulin's effect on appetite and the mechanisms, are they really well fleshed out now solidly? What's your thoughts on that?

Robert 34:47 So, insulin blocks leptin signaling. That is well flushed out. Several different labs have demonstrated this. Martin Myers lab, and people at Harvard showed it back in 2001. And Mike shorts lab. Mike Short still doesn't believe it. I don't know why.

35:11 So insulin blocks leptin signaling. So as insulin goes up, your brain doesn't see the leptin, in which case your brain thinks it's starving. Now the question is why? Why should insulin block leptin? And the answer is because there are two times in your life where you actually have to gain weight where you want insulin not to work. Sorry, we want leptin not to work, where you want to be leptin resistant, because you have to gain weight. If you were leptin sensitive all the time, you could never gain the weight. Those two are puberty in pregnancy. Well, those are the two insulin resistance states. So it doesn't make sense that the hormone that drives the weight gain peripherally should also be the hormone that blocks for leptin signaling centrally, so that those two phenomena, the weight gain and the hunger are yoked together by the same compound. So twice in your life, you want to be leptin resistant - puberty and pregnancy. The problem is we're now insulin resistant and therefore leptin resistant 24/7 365.

Ivor 36:14 Winter never comes.

Robert 36:16 That's right. Winter never comes.

Ivor 36:18 Well, it was an absolute pleasure and as a say, a privilege to finally talk to you, Robert, and hope to meet you again soon. And thank you.

Robert 36:25 My pleasure. Thank you for having me.

Ivor 36:27 Thank you.