Sugar Addiction:
A Special Interview with Dr. Robert Lustig

By Joseph Mercola

DM: Joseph Merola

RL: Dr. Robert Lustig

DM: What is the proper role of sugar in our society? Can it be considered a poison? Hi, this is Dr. Mercola, helping you to take control of your health. Today, we are joined by Dr. Robert Lustig, who is a Professor of Pediatric Endocrinologist at the University of California in San Francisco (USCF). You might know him for one of the more popular YouTube videos on sugar that has over 5 million views. Now, that’s not as many as Psy’s Gangnam Style, which has 3 billion. But it’s certainly very popular. You might have also seen him on 60 Minutes. We’re really honored and privileged to have him join us today for discussion on this really important topic. Welcome, and thank you for joining us today.

RL: It’s my pleasure, Dr. Mercola.

DM: I’m wondering if you could, perhaps, expand or comment on your view about the role of sugar in society? Its proper role, that might be a better term for it.

RL: Well, once upon a time, sugar was a condiment and it had been a condiment from 1200 BC when the Indian subcontinent first learned how to extract cane juice. It got shipped around as a fruit extract, which was called Canda, which of course got ultimately shortened to candy. It was for nobility and it was hard to come by, until about year 1700, when the pot still allowed for mass production of refined sugar. It was still extraordinarily expensive until the middle of 18th-19th century.

At that point, we started seeing it appearing in various venues. We started seeing the growth of American sugar industry in Louisiana, Texas, and Hawaii. That’s when we started seeing chronic metabolic disease [inaudible 02:09] at about the same time. In fact, the very first demonstration of an increase in chronic metabolic disease was in 1924, when Haden Emerson, the commissioner of health of New York City, noticed the seven-fold increase in diabetes rate in New York City population. Then in 1931, Paul Dudley White called attention to the fact that we had an epidemic of heart disease. In 1988, we learned about the advent of adolescent type 2 diabetes. These are the three seminal hallmarks of chronic metabolic disease pervading our population. It goes up in lockstep with our increase in per capita sugar consumption.

The bottom line is sugar used to be something we added ourselves to coffee and tea, something where we had control over. Now, we are consuming virtually 25 times more sugar than our ancestors did, and we don’t have control over [it]. It has gone from condiment to diet staple. It is now anywhere from 15 to 18 percent of our total calories. Our liver, which process sugar, in terms of its metabolism, can’t handle the load that it’s being presented with. And when you overload your liver, you get bad chronic metabolic diseases. That’s what the data show. That’s what the data on sugar show.

Basically, sugar is metabolize virtually identically to that of alcohol, and we are now seeing diseases in children that we never saw before and they are alcohol-related diseases. Like for instance, non-alcoholic fatty liver disease and type 2 diabetes. These are diseases that we’re never seen in children and these are diseases of alcohol. Kids don’t drink alcohol but they certainly consume sugar, and that’s the point.
The point is we are overdosed. We have gone beyond our limits and we are now evidencing this massive increase in chronic metabolic disease which is chewing through the health care resources of every developed and developing country on the planet, and this is unsustainable.

**DM:** I couldn’t agree more. As a Pediatric Endocrinologist, I was wondering if we could [04:49] dive a bit into the metabolism of the sugar damage. I’m sure you would agree that one of the primary issues is metabolic syndrome. But at the core of the cause of metabolic syndrome, would it be fair to assume that insulin resistance plays a major role in that fact, in that syndrome?

**RL:** Insulin resistance is a hallmark of metabolic syndrome. Virtually, whatever organ becomes insulin resistant, it becomes an organ that manifests its own metabolic syndrome. When you have insulin resistance of, for instance, the liver, you end up with type 2 diabetes. When you have insulin resistance of the brain, you end up with Alzheimer’s disease. When you have an insulin resistance of the kidney, you end up with chronic renal disease, and so forth. The point is that all of these are due to insulin resistance states. The question is what causes this in the first place.

While the data is not completely in yet, we have some new data that we are very excited about, which demonstrate that if you overload the mitochondria, the little energy-burning factories within cells, in any given organ, you’ll end up manifesting these various forms of chronic metabolic disease. The thing that overloads the mitochondria best is trans fats. The thing that overloads the mitochondria next best is sugar. Trans fats and sugar pretty much characterize the processed food diet.

Trans fats are going away because now the FDA, just last year, finally declared trans fats as generally recognized as not safe. For the first time, they took trans fats off the Generally Recognize as Safe (GRAS) list. That’s wonderful and terrific; but sugar is still there and sugar is going to be a tough one to dislodge from that list. The bottom line is, as long as it’s on that list, the food industry has license to use as much as it wants to in any given food stuff. So, sugar has become the biggest problem in our diet since the advent of trans fats.

**DM:** That’s an interesting comment on the trans fat myth. That FDA action was implemented by a lawsuit by Dr. Fred Kummerow, who is a long-time researcher of trans fats. The unfortunate challenge with that or problem is that they are removing trans fats and they are replacing it with other oils, these non-saturated vegetable oils. [And when] they are heated to high temperatures, they are producing even worse substances like cyclic aldehydes, which is probably going to make trans fats look good and we won’t realize the full damage of this for 10 years down the road.

**RL:** Indeed. The bottom line is polyunsaturated fatty acids, which are in a lot of seed oils that we consume, are actually reasonably good for us until they’re heated. When they’re heated, then they actually form what are tantamount to trans fats. It’s a real crapshoot and it’s a real problem in terms of how one deals with processed food. Processed food has so many problems with it and sugar’s just one of the problems. There are many problems with processed food. There’s too much of five things and too little of three things.

The too much things are: too much trans fats; too much omega-6 fatty acids, which are pro-inflammatory; too much branched-chain amino acids, which also overload your liver and cause chronic metabolic disease, and you get that from corn-fed beef, chicken, and fish – well, that’s processed food; too much alcohol, and too much sugar.

On the too little side: too little fiber, too little micronutrients, and too little omega-3 fatty acids, which are anti-inflammatory. Processed food has a zillion things wrong with it. Unfortunately, processed food is what we subsidize. Processed food is what we expect people to consume because of 1) expense and 2) shelf life. That’s making a fortune for the food industry but it’s killing us.
DM: Back to the issue of the trans fats or the insulin resistance, I was a bit surprised to learn, from your perspective, that trans fats appear to be a more significant contributor to insulin resistance than sugar exposure. Is that what you’re saying?

RL: Again, trans fats have been going down, so the fact of the manifestation of the increase in insulin resistance that we’ve seen over the last, I would say, 10 to 15 years, we cannot attribute to trans fats. There is no question that trans fats drove insulin resistance in part because we couldn’t digest the trans double trans fats became so popular was because bacteria couldn’t digest them, and that increased the shelf life for pastries, cakes, and things that sat on the shelf; vis-à-vis the 10 year old Twinky. But the fact of the matter is the same reason that the bacteria couldn’t digest them.

Our mitochondria are refurbished bacteria and we couldn’t digest them either. So what would happen is, we knocked off the two carbon fragments to turn them into fatty acid oxidation products until we got to the double bond, then we couldn’t go any further. And so then the rest of it lined our arteries and liver. You could actually see trans fats moieties in the arterial lesions of people with atherosclerosis. There’s no question that trans fats are a disaster. They continue to be a disaster. They are still used in foods although certainly, at much lower levels than before.

But again, as we talked about… When you heat polyunsaturates, you can make them. They’re never going to go away completely. Trans fats are, without question, consumable poison. There’s just no way around it. The question is what about sugar? Is sugar as bad as trans fats? The answer is no. I don’t think that sugar is as bad as trans fats. Because there is no threshold where trans fats are safe. There probably is a threshold where sugar is safe.

And that threshold probably, we can argue this in terms of individual people, but in general, that threshold probably is around six to nine teaspoons of added sugar per day, somewhere between 25-38 grams of added sugar per day. That’s what the data suggests, because our livers do have the capacity to metabolize fructose, this sweet molecule of sugar, as long as the mitochondria don’t get overwhelmed. So as long as you keep it below the threshold, above which toxicity would occur, I think that probably sugar is okay. I don’t believe that’s a straight linear relationship. I believe it’s got a curvilinear relationship and I think there probably is a safe threshold.

DM: I like to expand that because I think it is important point. I suspect insulin resistance plays a big role in making a decision. Would you agree with the fact, the belief, that insulin resistance plays a larger role in the incidence of overweight or obesity?

RL: No question that insulin resistance generates hyperinsulinemia. Hyperinsulinemia means that there’s more insulin at the fat cell, and more insulin at the fat cell means that you’re going to shunt more energy into those fat cells because that’s insulin’s job. Insulin resistance is clearly associated with weight gain. A lot of people think that the insulin resistance comes from the weight gain. Actually, the data, I would say in the past two years, argue strenuously against that: that actually, the insulin drives the weight gain.

There are basic science studies and clinical studies that show mechanistically that it’s the insulin driving the weight gain. This is one of the things that endocrinologists, nutritionists, gastroneurologists, and general physicians have not yet [inaudible 13:22] to, but the data are very clearly there. That insulin is the instigator of this phenomenon. When your liver becomes insulin resistant, which is what sugar does because of the way it’s metabolized, that generates hyperinsulinemia, and hyperinsulinemia drives energy storage into fat.

DM: Okay, so we have about two-thirds of the population that are overweight. And then of course, about maybe a quarter to a third that are diabetic or prediabetic, another quarter who are hypertensive, and a fair
number who are high in cholesterol. All of which seem to have insulin resistance as a component of that. So, what would be your estimates, as a clinician, as to the prevalence of insulin resistance in American society? Is it at least 80 percent?

RL: There’s no question, in the obese we’re talking about 80 percent. If you did a Venn diagram of the United States population, you have 240 million adults in that Venn diagram and you could basically have two circles, one about as twice as big as the other. You have the obese population which is about 30 percent, and then you have the larger non-obese population which will be about 70 percent.

What we know is that 80 percent of the 30 percent, 80 percent of the obese population, are metabolically ill. They have insulin resistance and that manifests itself in many ways [like] type 2 diabetes, hypertension, dyslipidemia, heart disease, cancer, and dementia. These 80 percent of the obese population is 57 million people, who are sick. They all get these diseases, and the standard mantra is, “Well gee, if they would just diet and exercise, they wouldn’t be obese and we could solve this problem.” This is patently untrue.

Number one: it is true that 80 percent of the obese population is metabolically ill. That is true. But that means that 20 percent of the obese population is not. They’re metabolically healthy. They are called metabolically healthy obese. They will live a completely normal life, die at a completely normal age, and not cost the taxpayer a dime. They are just fat. They take it on the chin because they are fat. But the fact is they’re not contributing to our runaway medical train, as it were.

Conversely, let’s talk about the 70 percent that are normal weight. So that’s 168 million people in that circle. Turns out, 40 percent of the normal weight population – 40 percent of the 70 percent – 67 million people have insulin resistance on lab test, and they manifest different manifestation of metabolic syndrome as well. Normal weight people get type 2 diabetes. They get hypertension, dyslipidemia, cardiovascular disease, cancer, and dementia also. Now, they don’t get it at the same prevalence as the obese, of course, 40 percent versus 80 percent. But they get it and there are more of them.

When you do the math, there are more thin sick people than there are fat sick people. The thin sick people are actually costing more, and when you do the math on the two together, the sick population is 124 million, that’s more than half of the US adult population. It turns out, the thin people are costing us more and the thin people… You can’t attribute this to gluttony and sloth or diet and exercise, because they’re normal weight. [If] it’s not about behavior, then there’s only one other option: it must be about exposure. This is an exposure.

This is an exposure that obese people are exposed to and it’s an exposure that even normal weight people are exposed to. That is called the Western diet. The Western diet is replete with sugar. Sugar is mechanistically the thing that drives this insulin resistance.

DM: Well, processed foods, although sugar is a major component, but I think the [inaudible 17:34] in general eat…

RL: Sugar is the marker for processed foods. Basically, processed foods don’t taste all that good. A lot of the things that made food worth eating were actually in the fractions that were taken out, like the fiber fraction. In order to mitigate the effects of bitter, sour, umami, and salty, what you do is cover them up with sugar. The food industry has figured out numerous concoctions of sugar containing items in order to be able to obfuscate the negative taste the processed food delivers. Sugar is the marker of processed food.

DM: What’s your best estimate of the prevalence of insulin resistance in the US population?

RL: It’s 80 percent of 30 percent plus 40 percent of 70 percent; basically, it’s about 50 percent.
DM: So it’s one-half from your perspective. So for those who are insulin-resistant who are really at the challenge… As a clinician, you’re still actively seeing patients in addition to educating individuals. What is the best therapeutic strategy that you learned about to effectively make a difference in resolving insulin resistance?

RL: Two words: real food. What we do in our clinic, here at the University of California, San Francisco (UCSF), the Weight Assessment for Teen and Child Health Clinic, what we do is assess patients, we determine what the issue is. But I would say for the overwhelming majority, 60 to 70 percent of patients, is because of their processed food diet. We basically teach them what it is that they are supposed to do. What it is that their grandparent did and we basically… Just like Michael Pollan said, “If your grandparent wouldn’t recognize it as food [then] it isn’t.” It’s the same concept. We get kids and we get parents off processed food.

Now, processed food has taken over our lives. If you look at the Mother Jones’ article that Cristi Kearns Couzens and Gary Taubes wrote back in 2012, they showed that our food dollar, they divided up into different bins, and it turned out that processed foods and sweets bin doubled in the span of 30 years; from 1982 to 2012. That’s exactly what’s wrong.

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What we have to do is we have to move them back, and what we do is we explain what real food is. Lot of kids don’t even know what real food is. Lot of kids think that fruit flavored yogurt is real food, it is not. We explained that yogurt is sour milk; the milk that sours in your refrigerator. And yogurts are virtual identical except that for yogurt, you get to choose the bacteria that does the sour. So if you want yogurt, have plain yogurt and throw whole fruit in, just like what Europeans do. That’s called real food.

What the yogurt companies are doing when they add evaporated cane juice to mitigate it, to actually get rid of the sourness, because it’s only in America [where] yogurt is a dessert. This is what we have to educate people. Yogurt isn’t supposed to taste good. Yogurt is supposed to taste sour, so we have a lot of work to do.

DM: We just published a report in Cornucopia exposing its fraud because the vast majority, well over 90 percent, probably closer to 95 percent of the commercially available yogurts, are nothing more than creamy junk food.

RL: Absolutely.

DM: I couldn’t agree more. I’m wondering if you could comment on the emerging number of clinical trial that seem to show very promising results with the use of intervention called intermittent fasting. There is a number of different strategies that one can use, but it seems to be a powerful tool that help resolve insulin resistance; ideally, integrative course with real food.

RL: Yeah. I’m not a fan of intermittent fasting but I’m also not a detractor. I have seen those studies. I have seen that they certainly can work. I think the reason they work is when you fast, your liver has to live off the liver fat that’s there. So what you’re doing is you are temporarily depleting your liver fat stores and you’re restoring metabolic stability to the liver, and improving hepatic insulin sensitivity, at least temporarily until you start eating again.

I think you can do this much, much more rationally, by eating properly all the way through the week rather than having to do intermittent fasting. I think, ultimately, the goal is get the liver fat down. And since the liver fat, the cost of the liver fat is dietary sugar, via the process of De novo lipogenesis which we have shown – which my colleagues Dr. Jean-Marc Schwarz, Dr. Kathy Mulligan, Dr. Alejandro
Gugliucci and I have shown – we think that once you get rid of the sugar, the liver fat will go down and we have data that supports that, both in adults and in children.

I personally don’t think you have to go to the extent of intermittent fasting. I think, ultimately, what you have to do is get the liver fat down. Will intermittent fasting do that? Yes, it will. But will eating properly do that? It does it even better.

DM: Well, it just seems to replicate an ancestral eating pattern that we have and that made our [inaudible 23:23-25] adapted to because our ancestors never had an access to a grocery 24/7. Most of them went through regular periods of feast and famine, so it seems we may be adapted to that type of eating style rather than eating three meals and snacks in between and the only time we’re not eating is when we are sleeping; even if it’s real food.

RL: Well, there’s no question that if you fast, you’re going to become ketonic. You’re lowering your insulin. You’re going to release fatty acids from fat cells into the blood stream. Those fatty acids are then going to the liver and get processed into ketones. Those ketones can then be used by the rest of the body for energy. That would certainly works, but the problem with fasting is, when those ketones first come out and the enzymes aren’t yet developed in order to be able to metabolize, you feel like crap; you feel lousy. People who become ketogenic acutely usually do not feel very good. I do it once a year [inaudible 24:18] and I tell you, I don’t feel so good; I get headaches.

But if you do it for five days, if you basically adapt the ketogenic diet – and this of course is the work of Dr. Jeff Volek and Dr. Stephen Phinney, they get this and understand this – what you do is upregulate those enzymes. And you can live off those ketones very nicely if you maintain that ketogenic diet and keep your insulin down at zero. I have nothing against that either. If you want to do that, that’s great. It certainly limits the kinds of foods and the availability of a real food diet; limit the types of food that you are consuming. You can’t have any carbohydrate if you’re going to do that.

I personally don’t think that you have to go to that extreme in most cases. Now, I do use a low carb diet for patient whom I cannot get the insulin down any other way. I’m not saying that low carb diets are bad. I’m not saying that low carb diets don’t work. They’re not bad and they do work and I do use them. But I use them in patients on a per patient basis. I don’t do, you know, sort of general advocacy of any given diet.

I practice what you might call personalized obesity medicine. The goal is to try to match the therapy with the pathology. And when we do that, we are usually very successful. I would put my numbers and my statistics in terms of our success in our clinic up against any other childhood obesity program in the country. We don’t see our patients nearly as often, so we don’t waste as much money in terms of personnel. We have a breakeven situation as opposed to a lot of programs that are throwing lots of money in to keep people employed, and they are not doing nearly as well. The reason is we match the pathology with the therapy.

DM: That’s great. Congratulations in your results. The issue with intermittent fasting, it doesn’t necessarily have to involve a low carbohydrate diet. In fact, most of the regimen that I’m familiar with [inaudible 26:29] so you don’t necessarily to go into ketosis. It’s just the matter of restricting the eating length of maybe eight to ten hours...

RL: Right.

DM: And still having regular carbohydrates in that. It just seems to me, if a person’s highly insulin resistant, maybe not experiencing the results you’re anticipating in your typical program, that that may be a therapeutic option to accept, to accelerate the insulin resistance.
RL: I’m not for or against it. You know, the fact of the matter is half the Muslim world celebrates Ramadan, and they don’t necessarily lose weight while in their intermittent fasting each day. They’re going eight to 12 hours without eating either. I think it probably depends on the patient and depends on what’s going on. I’m not a fan and not a detractor. I think that probably intermittent fasting will work for some people and not for others. I think that some people can adapt it and some others can’t. I’m for real food. That’s what I am for.

DM: Okay, perfect. Do you actually monitor insulin resistance clinically or in a laboratory, and then they send the assessments? Do you change your guide recommendations?

RL: Absolutely. We do many lab tests on patients when they first come in, in order to establish what their metabolic health base line is. So we look at fasting insulin, and of course, we look at fasting glucosamine. We compute the HOMA, the homeostatic model of insulin resistance. We look at uric acid as a proxy of sugar consumption. We look at ALT as a proxy of liver fat. We look at lipid profile to look at the triglyceride levels, the HDL levels; that will tell you about insulin resistance. We look at LDL as well, although that is a secondary issue in our clinic as opposed to our primary issue; like it is in so many other places. Of course, we do hemoglobin A1c to determine what their degree of glucose tolerance over the last three months is.

So we do a whole host of laboratory studies in order to sort of give us [an idea] where people come in; where they are at base line. And then using those data, we will tailor what we recommend for the patient and what they are able to accomplish within their home. Remember, I’m taking care of children, so in order to do that I have to get the parents to do the right thing for themselves, not just for the kids. We have to work with the entire family together in order to make the home safe for the child. And usually, when we do that, we make the home safe for the adult as well.

DM: So when the insulin resistance improves, you allow more carbohydrate or sugar into the… Permit more. I mean, obviously, it’s not in good and large amounts but it’s something that is based on your assessment of patient.

RL: What we tell people is very simple, get rid of every sugar beverage in the house. Then, eat your carbohydrate with fiber. And if you do that, then whole food is okay because the fiber mitigates the negative effects of the fructose on hepatic metabolism. Because it reduces the rate of absorption, [that is] number one. Number two, it forms a gel in the inside of the intestine, which provides a secondary barrier and delivers food further down the intestine faster so you get the tiny signal sooner, and you also get the nutrients to the intestinal bacteria so that they can chew them up so you won’t get them.

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So basically when you consume your carbohydrate with fiber, you are limiting your absorption. You’re limiting your liver’s exposure and your liver can get healthier. We don’t tell people they can’t eat sugar. They have to eat sugar in a form that nature produced it, and it’s called whole fruit.

DM: Okay. [inaudible 30:29- 30:33] grains too, because there are many clinicians who, especially in an insulin resistant individual, would suggest limiting grains or eliminating it until the insulin resistance improves. I’m wondering what your [inaudible 30:41] is.

RL: What we say is that nature packaged all carbohydrate with its requisite fiber. If you eat it the way nature produced it, you’re getting the fiber that you need to mitigate the effect. What you’re referring to is the concept of glycemic index. The higher the blood sugar rise, the higher the insulin rise; therefore, the more energy will get shunted into fat. I do not believe in glycemic index. I believe in glycemic load. There’s a big difference between the two.
Glycemic index is how high will your blood sugar rise when you consume 50 grams of a carbohydrate in specific food. Glycemic low is how much food do you have to eat to get the 50 grams. The perfect example of how this is dichotomized is carrots. Carrots have a high glycemic index. If you eat 50 grams of carbohydrates in carrots, your blood sugar is going to get pretty high. And so carrots have a high glycemic index and a lot of dieticians tell their patients, “Don’t eat carrots because they have a high glycemic index.”

Garbage. Absolute trash, because carrots have very low glycemic load. In order to get 50 grams of carbohydrates in carrots, you would have to eat 1.3 pounds of carrots. You’d have to eat the whole frigging Grimmway truck. Okay? And no one’s going to do that. You’re going to get sick of carrots before you ever get there. The concept is, if you consume the food with its requisite fiber, the fiber mitigates the effects of the carbohydrates on that insulin response. Real food takes care of itself. That’s basically a low glycemic load diet; that is what we’re shooting for. There are two words that describe a low glycemic load diet. You don’t have to go to the store and say, “Is this low glycemic load or not?” All you have to say is, “Is this real food or not?” Because real food is automatically low glycemic load because it comes with its requisite fiber.

DM: The argument against the use of glycemic index is, one of your friend is fructose and its glycemic index, I believe, is zero. Right?

RL: Fructose has a glycemic index of 19.

DM: 19, very well.

RL: Yet, fructose’s negative effects have nothing to do with glycemic index. It has to do with how much your liver gets overloaded. Glycemic index is how high your serum glucose goes. Fructose doesn’t contribute to an increase in serum glucose, because it’s fructose. It contributes to a serum fructose level, which is way worse. And it causes reactive oxygen species (ROS) formation within the arterial wall. It causes plaque formation, [and] causes all sorts of negative consequences, lipid peroxidation and protein denaturation due to reactive oxygen species, having nothing to do with how high the insulin goes and how high the blood sugar goes. Fructose is toxic, irrespective of its glycemic index, because glycemic index is a canard. Anyone who believes in glycemic index needs to basically get re-educated, and I’m talking to all you dietitians out there. Get with the program.

DM: Great. I couldn’t agree more. I’m sure you’ve heard of the recent interest in the observation that sitting is the new smoking, and the fact that most of us are sitting up to eight hours a day and if you got an office job, it might be 13 hours or more. I’m wondering if you’ve integrated that component into your program by recommending people to walk after they eat to lower the blood sugar level.

RL: Sure. We very much promote exercise. We have four messages that come out of our general lifestyle intervention for every patient when they first come to clinic. Whether they do it or not, it’s a different issue. But the four messages are: 1) get rid of every sugar beverages in the house (by the way, all these are evidence-based); 2) eat your carbohydrate with fiber; 3) wait 20 minutes for second portions, in order to take advantage of the peptide YY rise to reduce second portion consumption; and 4) buy your screen time with activity. If you’re watching TV for half hour, that means you’re outside for half hour. If you’re texting for an hour, that means you’re playing soccer, for an hour, etc., etc.

Now, the question is why are activity and exercise important? Does exercise promote weight loss? The answer is no, it doesn’t. Exercise does not promote weight loss. There are no studies anywhere in the entire world’s literature that show that exercise alone – alone – promotes weight loss. It promotes weight stability, but it does not promote weight loss. If you believe that calorie is a calorie, if you increase your exercise, you should you should be burning calories. You should be losing weight, but you don’t, because
a calorie is not a calorie. Why does exercise does not promote weight loss? The answer is exercise promotes muscle gain. That’s the reason why exercise is good. Exercise promotes muscle gain.

There is a transcription factor known as Peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) which is the transcription factor that’s involved in mitochondrial biogenesis. In other words, when you turn up PGC-1α, you make more mitochondria. Increasing your sympathetic tone which is what exercise does, turns on PGC-1α. Increasing your exercise makes more mitochondria and the reason is you make more muscle, and muscle weighs more than fat. So, you don’t lose weight, what you do is, you get rid of fat and you build muscle. All of which improves insulin sensitivity. All of which are very good for you from a metabolic standpoint; from a quality of life standpoint, from a general health standpoint. All good, but it doesn’t show in the scale because muscle weighs more than fat.

Every doctor who tells their patient, “Well, if you’d exercise, you’d lose weight.” You know it’s basically guilty of malpractice. Because it isn’t true, there are no studies that show it. Yet, exercise is the single best thing you can do for yourself and we should be promoting it, but we have to explain to patients what the outcome variable they should be looking at is. And the outcome variable is belt size, because they will reduce their visceral fat. They will lose inches, not pounds. And losing inches means improved metabolic health, and that’s good and great. We should be explaining to patients what it is that they should be expecting from exercise.

DM: Great. Do you use weight-size measurement as a variable in your practice? What standards do you recommend people seek to achieve based on your experience?

RL: The problem is that waist circumference in children are not standardized. They’re very different across different population, different racial groups, and of course, different ages and different puberty stages. What we do is we use waist circumference at baseline, and then look at it further down the line. We don’t have specific standards to be able to point to. There’s really only one paper in the literature on children’s waist circumference. It’s from Jose Fernandez from The Journal of Pediatrics and unfortunately, it’s really quite lacking. So what we use it as a baseline measure for each patient rather than comparing them to a relative of distribution.

DM: Do you have any comments on the waist-to-hip ratio relative to waist circumference?

RL: The data on waist to hip ratio don’t seem to contribute any more than waist circumference itself. The big issue with waist circumference is where do you measure it? There are two places to measure it; the World Health Organization (WHO) measures it in one place, the National Health and Nutrition Examination Survey (NHANES) measures it in another place. Actually, the data I like the best is the data that was developed from Rudolph Leibel’s group a couple of years ago, where they look at different metabolic parameters and had computed tomography (CT) values at different levels and they quantitated the amount of fat within that CT cut and what they set was 10 centimeters above L45. Well you where that is? That’s the belly button. I kind of measured at the belly button.

DM: Okay, as long as you’re consistent.

RL: I’m consistent.

DM: Very good. I want to applaud you for your efforts at penetrating the media with this really important message and educating many, many, millions of people about this. Because the traditional media manipulation has been toward [inaudible 39:42] is the result of the industry’s support to these ads and such. Congratulations on that. I’m wondering if you have gotten any negative…

RL: The bottom line is we have to speak truth to the power because that’s what is about, and we will not solve this problem until public is educated properly.
They have to know what the real story is. The good news is that there are now some resources out there that can give them that information: your blog, your website, as well, the movie Fed Up. You may know the www.sugarscience.org.

DM: We just did an article on that the other day.

RL: That’s 8,000 clinical research articles vetted by 11 scientists who don’t take money from the industry, of which I am one; still down to five messages for the general population. You may be aware of the public television special that we just produced called Sweet Revenge: Turning the Tables on Processed Foods, which is making its way around pledge break specials around the country right now. The point is that the information is now available. People need to understand that they have been fed a bill of goods and they need to take back their health because the food industry won’t do it for them.

DM: Again, I congratulate you for catalyzing that important message. So you’re really elevated in the academic community, I’m wondering if you get resistance from your colleagues about spreading this message and criticisms. What are you experiencing in that [inaudible 41:22]?

RL: Well, it’s kind of an interesting thing. I think there are few people who see this is sour grapes, like [they say], “Why does he get to do this?, “What does he have that I don’t have?,” or “Why is he promoting this? Is he trying to get famous?” I’m not at all. I’m a very reluctant participant in this. I was trapped in the ivory tower for many years, but the bottom line is this information has to get out there.

There are two things that have really changed my life. Two statements. Let me give them to your audience and they can, sort of, do what they will with them. The first was by Jeremiah Stamler, who is the father of cardiovascular epidemiology back in the early 20th century. He said –very famously– “If a researcher is not willing to follow his data into the policy arena, who will?” Well, the fact is we, scientists generate the data then we expect our policy people, our public health officials, and our politicians to use those data in order to improve the public good. Well, that stopped. That stopped about 40 years ago, and there are a lot of reasons why it stopped. But mostly, they center around money. We, physicians and scientists, now have to take this added role on as well.

The second is a statement that I heard from an Indian public health forum that occurred about five years ago. It is called [Hydrobod 42:58] statement and it states “All significant public health advances involve and require the use of law.” That’s exactly right. Every single public health debacle that we have faced in America has required a legal challenge. Usually because there’s somebody making money off the other end, whether it would be tuberculosis, pollution and asthma, vitamin deficiencies, teen pregnancy, guns, or HIV. Bottom line is they’ve all required legal challenges. Sugar and processed food are no different. In order to understand the nature of the arguments, understand the legal doctrines, and in order to be able to be a better and more effective policy wonk, I went to law school. I got a masters in law from UC Hastings College of the Law last year, very specifically, to be able to do this work and to be more effective at doing it.

We have to use the science to help formulate the policy, and we have to be able to know where the political imbroglios and the political problems occur in order to be able to either ram through them or circumvent them.

DM: Congratulations. I didn’t realize you had your… you’re now an attorney also.

RL: No, no. I’m not an attorney. I’m not [inaudible 44:26] just a Masters in Law, but I can talk to people who can. And they’ll listen.
**DM:** Perfect. What are your current strategies? Can you document or tell us any of these successes you have to date so far?

**RL:** Well, we are doing a whole bunch of things, but I will tell you most of the things that we are doing are sort of, right now, we’re being a little quiet about. But the first issue is public education. You can’t do anything until you do public education. Ultimately, you can’t see policy change, litigation change, and administrative law change; you can’t see any of these things unless the public is supportive and behind it. Right now, we’re not there yet.

It took a long time with cigarettes and alcohol for us to be able to get there. This is just really hitting the ground now. Mark Bittman, Michael Pollan, Ricardo Salvador, and Olivier De Schutter, this past year called for a national food policy. And I completely agree with that. Right now, we have 15 agencies and 51 separate agreements that control food regulatory activities in our country and no one knows what the other hand is doing. The food industry takes complete advantage of this.

We need a national food policy. Our non-profit Institute for Responsible Nutrition is participating in that discussion. We are doing bunch of things in order to try to level the playing field, and bring that to [inaudible 46:07] probably, over the course of the next three to five years. We also have some very specific efforts that we ourselves engage in, which I’m not in the liberty to discuss right now.

**DM:** I’m really curious as physician; we’re both physicians, we practice medicine, and we see patients. It becomes really obvious very clearly that food play essential role in almost all diseases, but why? Why are you so passionate about this, in making such a depth when the vast majority – forget the majority – we’re talking over 99 percent of physicians aren’t doing anything? To just going on with their day without saying a word about this and not really even addressing it, almost any way it should be performed.

**RL:** Because they didn’t want to help. They went to med school, I went to med school, you went to med school. Did you learn any nutrition in med school, really? I mean, nobody does and they still don’t. There is just a recent survey that said only 19 percent of medical schools actually teach nutrition in medical school. If food is medicine, and if all of these chronic metabolic diseases are diet-related, which they are and they’re preventable, shouldn’t physicians know something about food? What they’ve learned is they’ve learned about drugs because the drug industry has done a very good job of selling itself to the medical profession.

Whereas the food industry has basically gone the other route and basically remade itself because of subsidies into this consumable chemical industry, which we physicians don’t understand. The sad part of this, Dr. Mercola, is I knew all of these in college. I majored in nutritional biochemistry in college in Massachusetts Institute of Technology (MIT). In fact, my teacher was Sandy Miller, who ended up being one of the undersecretaries of the Food and Drug Administration (FDA). This was back in the mid-1970s, and then I went to medical school. They beat it out of me. I rediscovered all of these 35 years later based on the research I was doing. It was also like having a selective amnesia. I was having these sweats at night like, “Oh my god, I knew this.” It was coming out from my subconscious because it had been buried for so many years.

The fact is medical schools don’t teach this. Doctors don’t know this; they don’t know how to do this, they don’t know how to talk to their patients about this, they don’t even know what’s in the food themselves. I guess we shouldn’t be too surprised that the food industry has basically taken this part of medicine over from doctors. The problem is they’re using it to their own advantage. It’s time for us to take food back as a mode of therapy.

**DM:** I didn’t realize that you had a nutritional biochemistry background before we start this whole process. It makes perfect sense now. You know or maybe are aware that some experts believe that over a
century ago, that the Rockefeller and Carnegie foundations started to become involved in medical education, and actually were responsible for the confederal government actually licensing, formerly licensing and everything. But also, more importantly, and special context of this is that they are directing a medical curriculum more in favor of drugs and getting nutrition out of the curriculum by design.

**RL:** Well, that may be true. Certainly the Flexner report, from 1910…

**DM:** Which they subsidized.

**RL:** Which they subsidized. Certainly, that was true. But the fact of the matter is, I don’t think we did anything specific to undo the concept of food. I think it just sort of fell by the wayside. I think we left it to other people. I think you may remember Upton Sinclair’s *The Jungle* in early 1910s.

I think, we basically look at food safety, acute food toxicity as a major problem. We have not yet experience chronic food toxicity. It wasn’t in our ethos, it wasn’t in something that we knew about. Again, Haden Emerson noticed the changes in diabetes in 1924. It took a long time. Look at lead poisoning, the first paper that came out about lead poisoning was in 1892. We didn’t get lead out of paint until 1982. It took 90 years before the first paper and final action. These things, these chronic exposures, these long term chronic exposures, take a long time to fix. We shouldn’t be too surprised that this has happened.

But now, we have the data. It’s not like it’s a question anymore. It’s hard and fast, we have the data. We don’t have correlation anymore. Now, we have causation. We have causation for sugar and obesity. We have causation for sugar and diabetes, for heart disease, and for fatty liver disease. Now, we have correlation for cancer and dementia. We’re not quite there yet, for those two, although people are working on that. We have causation. It’s time to do something about it.

**DM:** You’re actively doing that and catalyzing others too with your efforts, it’s great to see that. You review the research on this area regularly and I’m wondering if there are any significant updates in the last year or so, that you think have been relative milestones or important pieces of information that people need to know?

**RL:** Well, of course, the meta-analysis that has shown that sugar contributes not just to weight gain but also to hypertriglyceridemia and other diseases. This past year, the paper from Yang, et al, from *JAMA Internal Medicine*, which look at consumption of added sugar over two decades as a percent of total calories and showed that it contributed to cardiovascular deaths in a big way, and if you were consuming 30 percent of your calories as added sugar, like our teenagers are, your risk for heart disease deaths is four fold greater. If you think we got a problem now, wait until our teenagers hit heart disease age; things are really going to be even worse shortly.

We have some data that we’re very excited about that we will be presenting at major meetings this coming year that will move the needle even more, which I’m not at a liberty to discuss right now.

**DM:** Terrific. Anything else you’d like to add before we end? Because it’s just been a real pleasure to connect and find out some of these updates.

**RL:** Just for your audience, food should confer wellness, not illness, and it used to. But then, the food industry got involved. And now it confers illness, not wellness. We have to take back our food.

**DM:** Terrific, short and simple. Congratulations again for all your efforts to help us take back our food. We’ll take it from there.