Introduction to Rob Lustig and his nutrition background

Welcome to this episode of PCOS SOS Summit. I'm your host, Dr. Felice Gersh. With me now is Dr. Robert Lustig He is a pediatric neuro endocrinologist with an expertize in metabolism, obesity and nutrition.

And he is now working to really change the food industry and is working very heavily to educate on the damage that can occur with the consumption of ultra processed food and sugar.

So welcome, Dr. Lustig. I'm so thrilled to have you join me on this summit, and I would love for you to tell us a little bit about how you got involved in this field of nutrition and sugar, what you've been doing and what you're working on now.

Well, thank you, Felice. But number one, it's Rob. It's not Dr. Lustig. I am not your doctor, and I am not anybody's doctor who's listening to this because I am clinically retired.

So take the moniker, Dr. Lustig, and throw it out the window. Okay? It's just me. Yes, I am a pediatric neuroendocrine ologist. Now clinically retired, I was always interested in how the brain controls hormones and how hormones control the brain.

When I was in seventh grade, it was 1968. Gilman and Shelley identified and isolated the first hypothalamic releasing factor, called TRF at the time thyroid open releasing factor.

And I did a term paper on it and I said, This is really cool. How cool is that? That the brain controls everything else that goes on in the body? And so I became a neuroendocrine ologist when I was 12 years old.

And you know, sometimes it sticks and it did for whatever reason. I went to M. I. T. for undergrad and majored in nutrition with the idea that ultimately, you know, I would be interested in growth, puberty.

And, you know, I was thinking about pediatrics even back then. And so I learned a whole lot of nutrition, you know, right from scratch. And then I went to medical school and they beat it out of me.

And they told me none of that stuff was important. This is how you take care of patients. You know, it was basically pills, prescriptions and procedures and, you know, don't worry about all that other junk.

And, you know, I believed them for better or worse, and I practiced. So the first 20 years of my career, basically in that same mode, you know, you know, match the the symptoms to the diagnosis, to the treatment and, you know, meet them, treat them and street them, get them out.

You know, and my patients were getting better. And, you know, then I started doing research on this issue. Around 1995, when the hormone leptin was discovered.

Leptin, of course, is the hormone that goes from your fat cell to your brain that tells your brain you've had you have enough energy on board so you can burn energy.

How neuroendocrine is that? So I was taking care of all these kids at St Jude Children's Research Hospital, all these kids who survived their brain tumors but became massively obese because of either the tumor or the surgery or the radiation.

This phenomenon we call hypothalamic obesity.

Hypothalamic obesity and insulin-suppressing treatment

And I had about 40 of these kids and, you know, parents would, you know, basically cry in my office. You know, my kid survived the tumor only to succumb to the therapy.

This is like double jeopardy. And it behooved me and it fell on me to try to do something about this. And these kids were 350-400 pounds. And when they when they before they got their tumor, they were perfectly normal weight for height and they became massively obese because of the tumor.

So this has been known for a hundred years, you know, but Binskin freely described this in 1901 and no one knew what caused it. Well, because I'm a neuro endocrinologist, I did know that there's a connection between the brain and the pancreas as well, the vagus nerve.

You've heard of it. And I knew that if you lesion the brain of an animal, they become massively obese because that vagus nerve is sending information to the beta cell of the pancreas to release more insulin.

And the reason now, because of leptin was obvious, because they couldn't see their leptin, because of the brain damage. Their brains thought they were starving and because they were starving, their brains put out a signal to store more energy in the form of it by releasing more insulin.

So here I had these 40 kids with the same problem as these, you know, lesion animals and what was I going to do for them? So I can't fix a brain and I can't cut a vagus nerve, but I could give a medicine that suppressed insulin release.

And so we did a study using a medicine called octreotide, which mostly inhibits growth hormone release, but it also inhibits insulin release. So we repurposed it for that reason.

And lo and behold, these kids started losing weight. And not only did they lose weight, but something even more remarkable happened. They started exercising spontaneously.

So we had eight kids in the original pilot study. One kids became a competitive swimmer. Two kids started lifting weights at home. One kid became the manager of his high school basketball team.

These were kids who sat on the couch, ate Doritos and slept. There were lumps on a log. And because we gave them this medicine that suppressed insulin, all of a sudden they were active.

And the parents would say things like, I got my kid back, and the kids would say, This is the first time my head hasn't been in the clouds since the tumor.

This was really remarkable. So we did a double blind, placebo controlled trial, showed that this was indeed the case, that this was reproducible. We asked the question, okay, are there adults without brain tumors who have the same problem?

And so we gave octreotide to a group of adults, 44 adults. It turned out eight not not all of them, but a small percentage, about 20% of the total. When we gave them the drug.

The same thing happened to them that happened to the kids. And when we looked at their insulin response, it was the same as the kids. So what we had done was we had identified a subset of obesity, a specific pathology that causes obesity that is different from, so, shall we say, general obesity.

And so this basically showed me two things. Number one, obesity is not a disease. It is a syndrome of diseases. And that if you treat the patient's pathology, you can actually get results.

That was the first thing. And the second thing it showed me was that the behavior we associate with obesity, the gluttony in the sloth, is actually secondary to the biochemistry.

And when you fix the biochemistry, you fix the behavior. And I have been working off that hypothesis for the last 20

years, and that's why I'm here talking with you today.

Well, that is quite a phenomenal detective story, really. And it's just amazing what you found out about sort of this underlying neuroendocrine pathways that are involved in this subset of obesity.

So while you're explaining that, I'm thinking, okay, so all of those women out there who have been successful and just like these other women, their children survived a brain tumor.

And then look what happened. So I'm thinking of all the women out there who managed to get pregnant and have a baby, you know, after maybe they had IVF and other infertility treatments because they're suffering from PCOS now, they have their child and their child is maybe already having some problems with metabolic functions.

They're gaining too much weight, even though their young children or they're looking to the future and they know they've heard that women who have a daughter who has the the mom in the mom has PCOS, that their child has a higher risk of developing the PCOS issues, which include tremendous problems involving insulin resistance and metabolism issues, obesity.

So what should this mom do who has a young child and she doesn't want her child, male or female, to become metabolically unhealthy and for the women to develop PCOS?

Well, before we get to what women should do, they sort of need to understand what might be going on. You know, and it's likely that different things are going on.

So it might be that different things need to be done. And, you know, not everybody's the same. Even within PCOS. PCOS is a syndrome, right? Polycystic ovarian syndrome, which means that it's likely that there are multiple causes and multiple mechanisms of action and pathology.

So we need to understand that and we need to appreciate that going into this. Now, the big question, is this genetic? Maybe, maybe not. You know, people have looked for genetic markers for metabolic syndrome for a while now.

And best data I've seen says that about 15% of the risk for metabolic syndrome is genetic. The rest they call dark matter, meaning environment, meaning something about the environment.

So that doesn't mean that genetics plays no role. It just means that it might not be the whole enchilada. In addition, you have to remember that most kids are exposed to the same thing the parents are exposed to.

So the expose home, if you will, things that are in the food, things that are in the air, things that are in the dirt, things that are in the packaging, things that are in the house.

You know, just because a mother has PCOS and the kid is at risk for PCOS doesn't mean that it was genetically vertically transmitted. It might be environmentally vertically transmitted.

PCOS, insulin resistance, and environmental causes

And so, you know, I don't want people to walk away saying, oh, there's nothing I can do because it's genetic. No, there's actually a lot you can do because 85% of it appears to be environmental.

The question is, what about the environment? And that's where things get a little complicated and a little difficult to explain. But let's let's see what we can do here.

But one thing that ties together all of polycystic ovarian syndrome is insulin resistance. All patients with polycystic ovarian syndrome have insulin resistance.

So the question is what's causing the insulin resistance? Now, the standard mantra, if you ask any doctor or any

scientist for that matter, is, oh, it's obesity.

Obesity is the cause of the insulin resistance. And the insulin resistance is the precursor to metabolic syndrome. Garbage. That's total complete trash.

And the reason we know that's code total complete trash is because 30% of women with polycystic ovary syndrome are not obese. Their normal weight, they're still insulin resistance, but they're not obese.

Just because you're insulin resistant doesn't make you obese. And just because you're obese doesn't make you insulin resistant. They are two separate things. They overlap.

I don't argue that, that's for sure, but they are not the same. And it turns out the things that causes insulin resistance can also cause obesity. In other words, the insulin resistance very well may be an epi phenomenon and that the obesity may actually be a secondary phenomenon rather than a primary phenomena.

So we have a lot of data to support that. Insulin resistance is a problem, but it may not be the obesity that caused it. It may be the other way around. Now, what causes insulin resistance?

That's a really good question. And of course, everybody and his brother and sister are working on trying to figure that out. What I can say is that there are three fat depots in the body and they are not all the same.

Obesity is BMI. BMI only measures really one fatty depot, the subcutaneous or big fat as it does this bathing suit make me look fat. Fat. Okay. And of course, I know as a male and as like husband and as a father, you never answer that question.

Okay. Now, having said that, that big but fat is actually metabolically protective. That's where your body wants to put extra energy is in the big fat.

So it may be cosmetically undesirable, but it is metabolically inert. So, Felice, how many pounds of subcutaneous is fat you have to gain before you become metabolically ill before you develop insulin resistance?

Well, I didn't even think there was a clear cut correlation there. On average, about £22, about ten kilos. Some people more, some people less. But, you know, around ten, around ten kilos right now.

The second fat people is the visceral fat, the big belly fat. And it turns out the big belly fat has nothing to do with diet. The big belly fat has to do with stress, cortisol.

How do we know this? Because we can take patients who have endogenous clinical depression, who are suicidal, who have to be admitted to the hospital to save them from themselves.

And we stick them in a scanner and it turns out they're not eating, they're losing weight. Weight loss is a hallmark of endogenous clinical depression, a major depressive disorder, because they're anhedonia, so they don't eat, so they lose weight, but they increase their visceral fat.

The reason is because their cortisol is sky high, so that visceral fat is actually responsive not to food or to exercise, but rather to stress. And when the stress goes down, then the visceral fat goes down.

And we've actually shown that in our studies at UCSF. One mindfulness, it's called the shine study by my colleagues Alissa Apple and Rick Hecht showed that if we take post-menopausal women with visceral fat and teach them mindfulness both at six months and 18 months, we can show that there's no change in weight, but there's a change in visceral fat and change in insulin sensitivity improvements in both.

So that big belly fat is a major contributor to disease. So how many pounds of visceral fat do you have to gain before you get sick? About five. So £22 of subcutaneous fat versus £5 of visceral fat.

And now the third fatty liver fat. And it turns out liver fat is the worst fat of all because it's it's affecting ground zero. It's affecting the liver, which is where insulin resistance starts.

So what makes that liver fat? Turns out sugar and alcohol, that's what makes liver fat, not other calories, sugar and alcohol. So how many pounds of liver fat do you have to gain before you get metabolic rate of £1?

So £22 of subcu fat equals £5 of visceral fat equals £1 of liver fat. Now, can you measure £1 of liver fat on the scale? No. Impossible. Can you even measure £5 of visceral fat on this scale?

Not really. So the fact of the matter is, this obesity thing, this whole subcu fat thing, this whole BMI thing, this whole get the weight down thing is actually missing the point entirely.

Let's fix the fat that's actually causing the problem, because that's what's driving the insulin resistance is the fat in the liver. And that has to do directly with the composition of the diet.

Well, I'm thrilled to have you bring all of that to light, because when you look at women with PCOS, there is a few really key things that are completely interrelated to what you just discussed.

Number one, they even the lean ones, although we call them Lean PCOS, when they've done body compositions like CT scans and such. They are. And someone coined this term, which sounds terrible, like the skinny fat, you know, because they have high amounts of this visceral, this really deadly, inflammatory fat.

Even though by weight, if they stood on a scale or BMI, they're actually normal or even at the low end, but they are very unhealthy. In addition, women with PCOS do have those high rates of of the nonalcoholic fatty liver, which is, you know, so, so awful for their overall metabolic well-being, their overall status.

And women with PCOS and there are published studies on this. They tend to run high levels of cortisol and they tend to run higher rates of anxiety and stress and circadian rhythm dysfunction.

And going back, you know, in terms of how things went awry at the very beginning, there is a fair amount published now of endocrine disruptors in utero which alter the receptors and the whole endocrine formation of the endocrine system and so on.

And so everything you said is so incredibly relevant to to all women at every age, whether it's post-menopausal in the reproductive age and even in the probably pre diagnostic stage of childhood of you know, of the PCOS spectrum.

So I'm just very appreciative that you brought that to light. A paper just came out yesterday in Frontiers in Nutrition out of Tel Aviv University, where they took a whole bunch of women and did MRI scans on their bellies and found that 33% of normal weight women are skinny, fat, which we have a name tofu.

Tofu, thin on the outside, fat on the inside. Real medical term coined by Dr. Jimmy Bell in neuro imager at University College London. So that's the big question is, you know, it's not the obesity.

The obesity is just the fat. You can see it's actually the fat you can't see. That's causing the problem. And the question is, how did it get there and what's it doing there and how do you fix that?

That's a very different question than how do you get into a bathing suit? Well, this is the ultimate challenge because the conventional medical world is doing starvation, diets, pharmaceuticals with no exit strategy whatsoever.

And and women are just so lost and they're so lost, what can they do for their children? What can they do for themselves? And and, you know, it's not simple, right?

Visceral fat, liver fat, and metabolic disease

There's no magic pill to the ill for this. So maybe you could talk about, well, where do you even start, you know, in terms of trying to turn around? Like, one thing is prevention, which we definitely want to do everything we can to try to stem this tide of of metabolic dysfunction, obesity, insulin resistance.

And then once it's established, though, how can we do some reversal? So where would you like to start? Because this is a gigantic topic. It is more than gigantic.

It is a systems biology issue. And it is the single biggest issue in the entire world, this side of climate change. And actually it's contributing to climate change in terms of the food system, basically, we need to rethink our entire food system in order to solve this problem.

And that ain't easy to do, as you can imagine. So here's the way to think of it. And I, you know, recognizing it's not easy. Okay. You said something very important.

You said, how do you prevent it? That's exactly right. You have to prevent this. You can't treat it so you can treat PCOS with estrogen. But that's not really treating the insulin resistance.

That's not treating the primary pathology. That is only treating the symptoms. That's treating the hyperandrogenism. That's treating the balding, that's treating the hair, that's treating the abnormal menses.

It's not treating the insulin resistance. You have to fix the insulin resistance. And in order to fix the insulin resistance, you kind of have to know what caused the insulin resistance.

And that's where things get complicated. So there are environmental obesity, genes, chemicals in our environment that actually make you gain fat. We published on this last year three articles in biochemical pharmacology showing how these different chemicals work.

Some of them are in the water, some of them are in the air, some of them are in the food packaging and some of some of them are in the frying pans and some of them are in the food itself.

All right. Example, Teflon Teflon is an obesity. You write PBS poorly fluoridated alkali substances, obesity. Another example, cadmium. Cadmium is an obesity. Okay.

Where did you get cadmium from and why isn't it listed on the side of the box or on the label? Well, cadmium is in cocoa because it's in everything in South America.

So if you're eating chocolate from South America, whether it's dark chocolate or milk chocolate, you're getting a big dose academy. Now, the question is, why does cadmium cause weight gain?

Why does cadmium cause obesity? What is going on with these obesity? We just submit a paper for review that suggests that it's the free radicals that are made by these various chemicals and also by dietary components that ultimately impact that insulin resistance at the level of the liver insulin secretion and the level of the kidney, the microbiome and also the brain.

And they're all in synchrony in order to make you gain more weight. So the goal is get rid of the free radicals, get rid of the reactive oxygen species.

Well, how do you do that? The best way is avoid those things that generate the most of the thing that we are exposed to every single day that contributes the most to free radical generation is air pollution.

The second most important thing that we are exposed to that generates free radical formation is sugar. Those two things, air pollution and sugar. Now, fortunately, we can't do a whole lot about air pollution and you can't do anything about it alone. And you know, if you live close to a freeway, you are much more likely to be obese.

Now, people think that's in part because of lower socioeconomic status. You know, it's a living close to a freeway is

a sign of poverty. That's true. But it turns out it's not related to poverty.

It's actually related to the parts per million of the particulate matter, what we call PM 2.5. And this was found in the Sally a study in Germany where they don't have, you know, the same freeways we do.

They have autobahns and they actually go through nice areas of town. But turns out sugar is the biggest generator of free radicals of reactive oxygen species.

Sugar is a primary driver of that liver fat. Sugar is a primary driver of insulin resistance. And the problem is, of course, is that 73% of all the items in the grocery store are spiked with added sugar on purpose.

So the food industry's reasons not for yours. So the first thing is, you know, if you can do something about your air pollution, great. Why don't you change the air filter in your house to start with?

And then the second thing is clean out your refrigerator and get rid of all the things that have added sugar in them. It used to be that trans fats were the highest generator of reactive oxygen species and trans fats were the biggest contributor to heart disease.

Well, we learned back in 2006 that trans fats were bad. They by 2013, they were gone from our diet. You know, the FDA finally banned them, but they haven't done a damn thing about sugar.

So that's where you start. And that doesn't matter if you're obese or if you're thin because PCOS is ovarian insulin resistance. It's insulin resistance at the level of the ovary.

Now, you might also have insulin resistance at the level of liver, you might have insulin resistance at the level of the brain, you might have insulin resistance at the level of the heart.

You might have insulin resistance at one of many different organs. And if you do, each one of those will give you a different set of symptoms. And that's why PCOS is a syndrome of multiple pathologies, because it depends on where the insulin resistance is.

But if you have PCOS, you have insulin resistance at the level of the ovary. That's what's causing the hyperandrogenism that's what's causing a lot of amenorrhea.

That's what's causing the changes in balding. That's also what's causing changes in cognition and emotion because of the change in estrogen, because you're not making it, because the androgens not getting converted to estrogen.

So fix the insulin resistance any way you can and it starts with diet. So, you know, you talked about sugar. And I know we're going to talk more about sugar, but I created a motto.

I just want to know if you agree with this motto, because I have patients and they're very confused about sugar. So instead of thinking it as a, you know, an additive, that's part of processed foods that they're finding in the area of the grocery store that's packaged with boxes and bags as opposed to in the produce section in the organic area.

And they'll say, oh, I don't eat beets and I don't eat a lot of fruit because it has so much sugar. So the motto I said is, Don't be afraid of fruit. You know, the problem that we're having with all of these metabolic dysfunctions isn't because just too many apples in your diet or, you know, you ate one too many grapefruits or something, you know, it's like the processed sugar.

So maybe you could comment on what is sugar anyway and where do we get it? I mean, is fruit really a problem or is my motto, don't be afraid of fruit.

Okay. Now, you are correct. Don't be afraid of fruit. So, yes, fruit has sucrose true? I'm not arguing. That's why it's sweet. Fruit has sucrose. Sucrose is two molecules, once called glucose, once called fructose.

We consume fructose and not the same. The fruit industry will tell you they are the same. They will tell you a calorie is a calorie and a sugar is a sugar.

And the reason they will tell you that is because they don't want you to know, because if you knew, then you wouldn't be eating their food, right? They think that if they hide behind calories, well, you have to eat something and, you know, you get discretionary calories and why wouldn't you spend them on our sugar?

Sugar, fructose, fiber, and why fruit is different

So calories work for the food industry. It assuages their culpability. But in fact, glucose and fructose are not the same because as a six member, green fructose, as a five member drink, they are handled completely differently by the liver.

And it turns out that glucose stimulates the metabolism of energy in your cells. It actually increases mitochondrial beta oxidation. It causes an increase in ATP generation, which is what your cells need in order to power themselves.

So glucose, for lack of a better word, we can call good fructose. On the other hand, this five membered ring actually inhibits mitochondrial function.

It inhibits three separate enzymes in mitochondria that make the mitochondria do its job inhibits an enzyme called AB kinase, which is the fuel gauge on the liver.

So it inhibits an enzyme called a catalase or COA dehydrogenase long chain, which is necessary to cut the fatty acids up into two carbon fragments. So you can oxidize them.

You can make ATP and also indirectly fructose increases uric acid, uric acid. It inhibits an enzyme at the outer mitochondrial membrane called carnitine pol mineral transferase one and CBT one.

This is the enzyme that regenerates carnitine and carnitine is the shuttle mechanism by which the fatty acids get from the outside into the mitochondria in the first place so they can be burned.

So every single thing fructose does to mitochondria inhibits their function. Well, if you inhibit mitochondrial function, what do you do with the excess energy when you turn it into fat?

And that's why fructose causes fatty liver. And that fatty liver then drives the insulin resistance. And the insulin resistance drives the chronic metabolic disease.

And when that's happening at the ovary, that's PCOS. Fruit. Yes, fruit has fructose. That's why it's sweet. It's not sweet because of the glucose. It's sweet because of the fructose, same chemical, same molecule.

It's true, but a whole lot less so. For instance, an apple has about four grams of fructose compared to a glass of apple juice, which has about 12 grams.

So it's one third as much number one. Number two, most importantly, the apple has fiber. And there are two kinds of fiber. There's soluble fiber like pectin or inulin, which holds jelly together, and there's insoluble fiber like cellulose, like the stringy stuff.

And so so imagine you have a spaghetti colander, you run, water goes right through. Okay, now imagine you take a blob of petroleum jelly and throw it into the center of a column.

And now you run. The war still runs right through, bounces off the jelly. Now take your finger and rub it all the way around the inside of the colander.

So that all that petroleum jelly gets into the nooks and crannies of that colander. Now run the war. Now the water

doesn't run. Now you got a barrier, you have created a secondary barrier because the petroleum jelly has blocked the holes.

Well, the same thing is happening in your intestine. So the cellulose, the stringy stuff and celery, also cardboard is acting like a latticework on the inside of your intestine, like a fishnet.

And the soluble fiber, which are globular, are plugging the holes in the fishnet. And together they form an impenetrable secondary barrier that prevents the absorption of glucose.

Fructose, simple starches renders them unavailable for absorption. So your glucose response is lower, therefore your insulin response is lower. You're keeping your insulin down, which is improving your insulin sensitivity.

That's what you want to do in addition, because you didn't absorb that mucus in fructose early, it goes further down the intestine. And what's in the second part of the intestine that's not in the first part.

The microbiome. The bacteria is bacteria can't live in the stomach or duodenum because the is too low, it's too acid. But as soon as you get into the second part, the jejuni after the ligament attracts the pancreatic juices which have the bicarbonate to neutralize that acid.

Now the bacteria grow like crazy. It's a sewer in there, and those bacteria will chew up the glucose and the fructose for their own purposes. So even though you consumed it, even though passed your lips, you didn't get it because you didn't absorb it.

So it's like getting fructose for free because you were feeding your gut, you were feeding your bacteria, which is what you need to do. You need to protect your liver, which the barrier does, and you need to feed your gut, which the barrier does.

So fiber is super important to metabolic health, but of course we throw the fiber in the garbage because we think it's the part of the food that doesn't matter.

It's actually the reason for eating the food. I can't tell you how much I loved that explanation and it matches with what I always talk about. You know, you have to nurture your gut microbiome.

Well, of course, nurturing your own cells. And that is so great. So everyone knows you eat whole foods. That's why they always talk about whole right as opposed to juicing it or you know, when you liquefy, I keep trying to tell people, stop juicing everything and eat it whole because number one, your brain won't know your eating if you don't chew and nothing's going to work quite right if you get it down into your gut so quickly and too, you're destroying the fiber, it really gets altered by that Vitamix or whatever you're putting it in that basically destroys the fiber, or you even do worse.

You strain it out right to a straining mechanism. So that is amazing. And one thing that you brought up that I just wanted to comment on was about mitochondria, know the energy producing factories.

So you mentioned also that women with PCOS don't make estrogen in the form of estradiol properly in their ovaries, which comes from the testosterone. And we know that estradiol is key to mitochondrial function.

So that's like an extra whammy for these women with PCOS, their mitochondria are being poisoned by their bad diet and then on top of that, by lack of adequate estradiol, which of course is comparable in some ways different etiology to like women in menopause who have an obvious estradiol deficiency state.

So this explains so much about why they feel tired. It's like living in a sea of energy that you can't access and it's such a different skill set to burn fat versus making and storing fat.

And that was just such a fantastic explanation. So thank you so much for that. Absolutely. There's another reason why they feel tired and irritable as well, and that is because the ovary, as you know, makes two hormones, not one

makes two.

It makes estrogen. It also makes progesterone. Now, progesterone is thought to be just, you know, important for pregnancy. No, actually, progesterone is important for the brain.

Progesterone is the precursor to alo pregnant, alone. And also pregnant alone is a hormone, a neurotransmitter. It's both. And what it does is it binds to the gab, a receptor.

So gamma aminobutyric acid and GABA is one of the neurotransmitters that puts your brain to rest. It's what's in Ambien, okay? And alo pregnant alone is like your own endogenous Ambien.

And so when you don't have your own endogenous neurotransmitter, you know, comar guess what you are, you're pretty irritable. So, you know, getting your ovary to make the estrogen and progesterone is pretty darn important.

But the problem is that in PCOS yes, you are basically stopping at the androgen level. So the androgen levels go up and that's not having any beneficial effect on your brain.

And all it's doing is increasing the risk for hair growth, baldness and taking away your curves and making you pretty darn miserable and increasing your risk, by the way, for heart disease, too.

So getting your ovary to be insulin sensitive is what therapy for PCOS is all about. It's not about bypassing the problem, and that's what you do with birth control pills.

So that is just a bypass. Now it's better than nothing, but it's not actually fixing the problem. It is bypassing the problem. You got to fix the problem.

And the problem is the insulin resistance and that is coming from your diet. Well, this is so absolutely true, because, you know, those of us who consider ourselves functional medicine doctors, we talk about the root cause and we're not just trying to cover up symptoms, although not that covering up symptoms has no value.

Real food, ultra-processed food, and practical next steps

Certainly symptoms can destroy people's quality of life. But we're really trying to get to the root cause and talking about, by the way, thank you so much for talking about progesterone as something that matters as a neuropathy detective hormone.

It's even a neuro produced hormone. You know, you make progesterone in the brain. It's so important. And estradiol also is neuroprotective. A lot of people don't realize that these are critically important hormones throughout the entire body, including in the brain and sleep and anxiety is also related to the actual levels.

And you're not making any of this properly. You don't ovulate. You're not making any progesterone in your ovary. So absolutely everything you said is resonating, I'm sure, with everyone out there who is listening, this is such key information.

So if they say Rob Och, where should I start? What should I do tomorrow? The mantra is All food is inherently good. It's what they do to the food. That's not so.

That means you got to eat real food, know what's real food and what's healthy. And in my book metabolically, I actually go through this. There are three things protect the liver, feed the gut, support the brain.

Any food that does those three things is by definition healthy. Any food that does none of those three things, by definition is poison. And any food that does one or two, but not all three is going to be somewhere in between.

So the goal is to consume food that protects the liver, feeds the gut, supports the brain, and all real food does all three things. That's where you start.

But of course, that means basically overhauling your entire refrigerator and pantry because most people don't even know what real food is. Most people in America think Cheetos is food.

And if you think Cheetos is food, all is lost. It's just that simple. It's so funny. You said that one time I was traveling, as we both do, and I'm sitting in the airport, people watching.

So I'm watching what people are eating at the airport. And I said, I don't think any of what's going in their mouth actually qualifies as food. So I had to go look up food.

The definition on Dictionary.com and it said it must have nutritional value. So basically all these people I'm watching at the airport are consuming non food.

So I would actually give you a slightly different definition of food because I've actually looked at this and I give a talk called is ultra processed food food.

So the definition of food, according to Webster's, comes straight out of a dictionary substrate that contributes either to growth or burning of an organism.

Growth or burning either. So let's say burning first. Turns out because fructose inhibits three mitochondrial enzymes, it actually inhibits burning number two growth.

My colleague, Dr. Efrat months Inigo or non who's head of nutrition at Hebrew University Jerusalem, has shown that ultra processed food and sugar in particular actually inhibits linear growth.

It inhibits cortical and trabecular bone growth and actually stunts growth, and it actually increases cancer cell growth. It basically hijacks growth.

So bottom line, ultra processed food neither contributes to growth nor burning. Therefore, ultra processed food is not food. Now, how can you recognize ultra processed food?

So my colleague, Dr. Carlos Monteiro at the University of Sao Paulo, Brazil actually came up with a classification system called the NOVA System Doesn't stand for it.

There's a new system. The easiest way to explain it would be an example. So let's take an apple. So Nova class one would be an apple right off the tree of a class two would be apple slices.

These stemmed deseeded maybe de skimmed nova. Class three would be a jar of applesauce, macerated, possibly cooked, possibly sugar. Added Nova. Class four would be a McDonald's apple pie.

The fact is that only that Nova Class four grouping is associated with all these chronic metabolic diseases diabetes, hypertension, dyslipidemia, cardiovascular disease, cancer, dementia, fatty liver disease and of course, polycystic ovarian disease.

So get rid of the NOVA Class four, get rid of the ultra processed food. That's where you start and that's where you finish. Well, everyone go and say that to yourselves.

Right? Write it down. Put it on your refrigerator and and what I have found that when you stop eating ultra processed food, when you get away from it, it's like your taste buds change, don't you agree?

And then suddenly, so-called, you know, real food, whole food, food from Earth, it tastes wonderful. It doesn't taste the same as it you thought it did, you know.

And you hate the stuff that has that chemical tiny taste to it. You finally taste it for what it is, you know, fake food, faux food, you know, don't eat that stuff.

And then that you will learn to love real food, right? I mean, people say I mean, you have it. I'm sure you've seen it with the kids when you were practicing.

It's like they say, I hate vegetables. And mom say, I don't know what to do like what do you do with people who say, I hate real food, I only love garbage?

What do you do? You have to desensitize their taste buds or sorry, desensitize their taste buds. It takes about a week. And my colleague, Dr. Monica Duse at the University of Michigan has actually looked at the biochemistry of what happens on the tongue.

And bottom line is sugar downregulated a receptor. So you need to sugar detox for about a week and when you do that, a blueberry will taste like a, you know, fruit bomb going off in your mouth and you will say, whoa, that's terrific.

But if you taste that blueberry, you know, today, you would say, I need, you know, you know, shall we say some blueberry ice cream instead? Oh, absolutely.

So you have literally whetted the appetite of everyone out there to learn more. How can people find you? How can they find your books? And amazing again.

So I'm easy to find. RobertLustig.com. I wrote really four books for the general Public Fat Chance back in 2013, which basically said, You are not what you eat, you are what you do with what you eat, you are what you metabolize.

I wrote metabolic, which actually recast that it is not. You are what you do with what you eat as you are. What they did with what you eat. Because the industry has basically bastardized all of our food for their own purposes.

I wrote The Hacking of the American Mind in 2017, which basically talks about diet and mental health, including these issues that we brought up with estrogen.

And I also wrote a cookbook with my colleague Cindy Gershon called The Fat Chance Cookbook. So 140 recipes, no added sugar and all of them prepare kibble within a half an hour.

The most complex piece of equipment you need is a blender and they were all vetted by a mount Diablo High School student to be produced all consumable and delicious in 30 minutes or less.

Now, I know I'm going to go out by that tonight. I mean, that sounds great because we all love to have a greater array of easy to do recipes to create food that is real and really tasty.

So that is fantastic. Rob, I can't thank you enough for joining me. This has been so informative and I just hope everyone is going to go out there and go to your website and start buying your books and really making the changes they need to make.

Fix problem. That's the only answer. Fix the problem where it is right now. The problem is in our food supply.