Introduction and Betty Murrayu2019s Background

Hi, everyone. Welcome to the Reversing Type 2 Diabetes Summit. I'm your host, Dr. Beverly Yates, ND and I'm delighted to interview today Betty Murray, who has expertise that is specific to these issues around reversing type 2 diabetes, hormonal management, weight, hormonal metabolism, and all of the things that come together.

Betty, would you please introduce yourself to our audience? Sure. So I'm Betty Murray. I'm a clinical nutritionist and a PhD candidate. So I've spent the last five or six years of my life really digging into the metabolism, specifically around women's hormones and and how the body really operates as we head through that transition to menopause and the impact those hormones have on mitochondrial function.

I'm also a practicing clinician, and so I work with patients every day, and I own a very large integrative medical clinic, and I'm certified by the Institute for Functional Medicine as a functional medicine practitioner.

So I'm an O. G. in this town around for a while. Right? Right. We are OGs together, I hear. I hear you. That's great. All right, so let's start off with a question to get us going here on their journey.

So the audience really gets a picture of what the situation is and how they can recognize themselves and get some hope, some inspiration and clarity. So what is the interaction that sex hormones play when they interact with insulin, weight gain and type 2 diabetes and pre-diabetes?

Yeah. So, you know, it's it's interesting because if you look at a man and a woman, I think almost any time you get two of them together and let's say they're working on trying to lose weight, you know, both of them say, okay, I'm going to give up all the processed stuff.

Maybe I'm giving up donuts and bagels and and and sodas. And for the most part, a man is going to lose weight more quickly. Right? Statistically, it's just the way the everything seems to go.

How Sex Hormones Affect Insulin and Weight

And so there is a profound effect of our sex hormones, not only on what makes a woman a woman biologically. Right. So it's it's our hormones, estrogen.

We have three of those that we make estradiol estriol and estrogen. And then we have another one called progesterone. And it's the fluctuation of those hormones throughout the course of our month that cause the cycle. Right.

So for anybody that's kind of like, what is that? And then men have testosterone and women have actually a lot of testosterone. We have about 3 to 4 times the amount of testosterone.

And then we do estrogen when our estrogens at its highest. But men's testosterone is significantly higher. And so testosterone causes a larger growth spurt.

We see more muscle mass. It has its own metabolic effects also. But women, when we are fertile, right, and we're healthy and we have balanced hormones, it's the act of estrogen really being balanced that actually helps us maintain lean body mass.

So let's say in your twenties and early thirties, that happens to be the case that as women enter that late thirties, early forties, you enter a period that we call perimenopause.

It's that it can be anywhere. On average it's eight years. That means some people double that 15 in some people,

you know, maybe they don't get a whole lot of experience of it.

But really what that is, is the start of the change in these hormones that affect fertility. But in that perimenopausal state, one of the hormones, progesterone starts decline fairly quickly.

And it's and it's why we see fertility issues in our late thirties and especially in our forties, because progesterone is really there to progress, to help the beat, you know, help basically the fetus grow.

And so with that decline in progesterone, what we see is a state of where estrogen is still circulating. At a normal level. It's starting, you know, it's still popping up very high.

The second half of your cycle. Often women get more symptoms like, you know, peri menopausal symptoms like heavier periods, more PMS, weepy, angry sleep problems.

All those things start to appear because these hormones are shifting. What, in that time period, because estrogen is elevated, we can be not always, but can be in a state where that we're estrogen dominant relative to these other hormones.

And there's an interplay between estrogen and insulin because insulin's job obviously is to either shuttle glucose, sugar from the bloodstream into a muscle cell or other cell to burn immediately, or it's to store it as fat.

Well, in our fat cells, we have the capacity to take testosterone and androgens like androstenedione, which are two of the kind of male hormones that we'd like to talk about.

And we have an enzyme called aromatase that can convert that into estrogen in the fat cells so we can actually make estrogen out of our fat cells. So there's this intimate relationship with insulin and estrogen.

And so as we become antigen dominant, we start to see more insulin resistance, meaning the muscle cells in particular become less capable of using glucose as its primary fuel.

And that's the that's the disposal vessel. You know, so think of it this way, The muscles are going to be your big burning engine. So if they start to become resistant and then you are a woman, that's the hormones are changing behind it.

We start to see weight gain despite probably doing all the right things. I would say that's that's my experience. I hit 40 and it was like everything fell apart.

I was like, What is going on? What used to work great for me isn't working anymore. And a lot of it was this hormonal thing going on behind the scenes.

So when you're in that early stage perimenopause, that estrogen dominance will lead to it. Well, when we get into menopause, which is, you know, officially one one day after one full year of no period.

Right. But it's once you're in menopause, you're always in menopause. It's not like something magical happens and it changes. Well, the loss of that estrogen also leads to insulin resistance and weight gain.

So it's about it's kind of like the the Goldilocks story. You want it to be just right. Not too much, not too little. And so women have a unique metabolic situation where the change in their sex hormones is changing their metabolism truly, honestly, at a cellular level, that's amazing.

There's a lot of complexity there, but in some ways it's kind of simple. I think of our reproductive years as being the time when we have the potential to bring life into the world, and then when we shift on the other end of it, it's like puberty turns that on.

Menopause is now turning that off. All of these things have to shift. And I would imagine that if at that time when a

woman is in perimenopause approaching menopause, the end of menstrual cycle, it would be really important for her to have a fantastic lifestyle and quality of life.

And it seems to me she'd be far more vulnerable. If she has a lot of stress, then if she's struggling with poor sleep, maybe hot flashes, maybe too many worries or anxiety, all these other factors that can come in, it seems like that would be really unfair.

What what are your thoughts on that? I agree. You know, it's like we play this really we pay this extraordinary this extraordinary expense for having the ability to really to really bring another life into this planet.

You know. So I think I think some of the things that we see, whether it's the sleep deprivation, it truly is let's let's put it this way, like the things that lead to stress, right?

Whether it's a sleep, the toxins or this or that, is that our bodies, more so than even a man, is hardwired to see that change in stress chemistry. Right.

The increase of particularly a prolonged protracted stress and cortisol. And then what ultimately happens from that is, oh, gosh, you know, we have this increase in cortisol.

How does the body interpret that? So the body is going to interpret that situation as I'm starving on the Serengeti because all all mammals, so all mammals on the planet, I don't care if it's a sperm whale, a monkey, an ape or a human, The only time their cortisol is going to be high in the natural world is when they are starving.

And the cortisol message to the brain is you need to forage outside of your normal foraging time because we need food. And so so all of the things that stress us out as women and then the hormones changing behind it is driving a lot of this because our body is getting this biochemical method message from our stress that says, Hey, you know, you're starving on the Serengeti, which starvation response drives an insulin resistance response.

It's an well, that's the thing is the disease. Diabetes is a natural response to our bodies. Broken perception at the cell, in the powerhouse and in the hormones.

It's, you know, it's the. Powerhouse, the mitochondria. Yes, Yes. Yeah.

Stress, Cortisol, and the Menopause Metabolic Shift

Yeah, absolutely. Absolutely. It's so interesting. Thank you for laying it out so clearly and tying together those pieces, because I think a lot of times people aren't aware of the connections and they feel like their body has betrayed them.

And so, you know, women, we just wanna be really clear. Your body isn't broken, it hasn't betrayed you. It is responding to your situation. Yeah. And it thinks it's doing the right thing. Right?

The chemical messages our body is giving itself is is these messages that are setting off these metabolic changes and it thinks it's doing the right thing.

It just doesn't realize that it's not working the way it should. All right, cool. So then that leads us to our next question. Right? Which is what other hormones become dysregulated with type 2 diabetes and pre-diabetes is and also are dysregulated at the same time doing perimenopause and menopause.

Yeah. So there's so there's several hormones. So we have, you know, hormones that handle satiety and hormones that handle gastric emptying and appetite regulation.

So, you know, if we if we think of these hormones and they're actually peptides, truly. So if we would look at them and look at what the body's really thinking about, well, these regulatory activities are happening, we see that appetite regulation that can be controlled by things like ghrelin and gastro.

And so these are all in glucagon like peptide one, GLP one, which has become very, very popular. These gastric hormones become just regulated because they are in themselves dysregulated during diabetes.

So let me kind of back up and explain that. So, so in diabetes, obviously we have the start of it's going to be insulin resistance, right? The cells are no longer listening to the different tissues in the body, whether it's muscle or liver or or or your brain.

They get dysregulated and insulin resistant at different levels. Right. So muscles first and then eventually the liver is usually the last to know. So the liver is thinking that everything is working properly and the liver is the one that's producing some of these hormones.

So so when we become insulin resistant, all of a sudden now the liver thinks things are okay, but the rest of the body isn't. So we see things like the gastric gastric, which is a hormone produced by the stomach that primarily stimulates the secretion of gastric acid.

So it's the stuff that helps you break down your foods, right? Proteins. And that changes even the passage of food from the stomach into the small intestines.

So gastric and get slowed down. We get a gastric slowing. And so your food sits here longer and you and you don't digest as well. So you can get things like heartburn.

But it's also the stimulation of gastric and and another hormone called ghrelin that then say, hey, I'm full, right? Hey, I'm full. They report back to the brain and say, hypothalamus, Whoa, we're good.

I don't need anymore. So we get this message that we're not full because food is being slowed, right? So if all of a sudden ghrelin, which is the hunger hormone, is produced in the stomach, that stimulates appetite and promotes gastric emptying, if we get disconnection of that message, we may also be hungrier and have more more inability for the body to actually digest and also manage manage blood sugar.

There's also a hormone called motility, and it's a produced in the small intestine and it stimulates gastric motility. So it moves what they call the migrating motor complex.

So think of that as a mechanical sort of thing. Your body goes through it. Know about 90 minutes after you eat, it starts at the top. I kind of think of this like a snake, and it sort of squishes everything down, squish all the food down, you know, and move it through the digestive tract so the motility changes and we see different contractility.

So all of a sudden you might get more digestive stuff, but the longer your digestion takes right?

Other Hormones Dysregulated in Diabetes and Menopause

So the longer your carbohydrate content is sitting in the spine since passing through the small intestines, particularly if you're eating more processed foods that turn to sugar more quickly, the faster and easier it is for it to get into the bloodstream, hit your bloodstream and be used. Right.

So that means your your ability for your blood sugar to come up after eating is going to be increased more quickly than somebody that has better gastric hormone control.

And then probably one of the other big ones that we hear a lot about now, because some of the medications for diabetes that are also very popular now for for weight loss is a hormone called glucagon, like peptide one or GLP one.

And it's called an increase in increase in hormone and it's created by the small intestines and it's in response to nutrients and it inhibits gastric emptying and in it and it increases kind of tone in the stomach, which moves the food to the small intestines.

Well, I always thought it was really interesting that changing that hormone in particular all of a sudden led to weight loss, number one. And an improvement in diabetes. Right.

Because the food, again, is not moving from the stomach into the small intestines where it can be mobilized into glucose as quickly. And so and then you have that same sort of messaging sort of thing going back and forth with the brain.

So those drugs actually change gastric function and digestive function. They're not directly working on insulin per say. Right. So those hormones change.

So as we go through estrogen or estrogen depletion through menopause, we see an impact of these to these hormones as well, because they work together.

But they but they also can be impaired and then one of the other areas that are really important is the activity of gluconeogenesis. So it's a big word, sounds, sounds really, really important.

Right. So gluconeogenesis is the process in which your body uses another form of fuel to make glucose. So at any given time, our body is going to have as an adult, about a teaspoon of sugar circulating.

Right. So one teaspoon now to be diabetic, it has to get up to to about a teaspoon and a half, I think is about right. Is that about right, Beverly, if you remember. Sounds about right.

Yeah. So teaspoon half. So everybody puts your put your thoughts on for a moment and just check it out. If you drink a soda, you know, it's going to be anywhere from 17 to 39, depending on the size.

Right. Of teaspoons. So so the body is going to freak out if anything's above that. So the other thing is, is is we don't have to have carbohydrate content in order to survive in our diet.

Now our bodies are made to use it. It's appropriate, but it's not an essential nutrient. We need proteins, we need fats. So what that means is the body has the capacity to make glucose with other fuel sources when we don't have it, i.e.

like in a starvation state or something like that, where food is not plentiful. So in the liver, glucose can get made out of non glucose ingredients, right?

So that's things like your amino acids, proteins broken down, that's things like fat. We can go lipo genesis and create fat and the body can then take it and make glucose out of it and transport it into the bloodstream to get used.

So in my research I was like, I think this is what's happening in women that are perimenopausal and menopausal. I think this is amplified. And so there is some studies, particularly looking at animals, showing that as estrogen declines, gluconeogenesis, the act of producing glucose at the liver is amplified.

So what that means is, is your body's doing this instead of burning fat, right? Because if I'm low carb or I'm fasting overnight, my body should be dipping into the fat stores, but instead it's going and creating its own glucose.

This would explain why at that moment in life there's such a change in terms of the fatty deposition. It's just so unfair. This is a real thing, especially in an era where we tend to have more access to food and if we aren't mindful about what we eat, if not the best quality food isn't nutrient dense, it isn't filling, and it often stimulates the desire for yet more calories, especially if we make the mistake of eating Say anything with artificial sweeteners and fake sugars.

This is trouble. This is real trouble. It's complete trouble. So so the take home message for everybody, they're like, okay, that's super technical. So I'll break it down very quickly.

You could be eating really well, right? You could be doing those things and maybe and maybe you're just starting on this journey and your blood sugar may or may not be out of whack, right?

So let's say maybe you're pre-diabetic and it doesn't look terrible. You know, if you have some degree of insulin resistance, let's say you're pre-diabetic, you're probably going through glucose, neo genesis.

And if you're an perimenopausal, menopausal woman, that is probably amplified. And and that even if you have removed some carbohydrate. So maybe you said, okay, I'm cutting back on my breads, pastas, crackers, cookies, and I'm going to eat more of my healthy vegetables like broccoli, Brussels sprouts, sweet potato, maybe you're doing all that and maybe you're not seeing things shift yet.

And it may be because your body is literally going behind the scenes and making glucose for you. Right. And I think this is also very much tied. So if we sort of wrap this up and look at it, okay, this is a survival mechanism, This this make sure we can survive because red blood cells and the brain, but particularly red blood cells must have glucose.

So if we back it up and say, okay, 95% of us are usually stressed and we have elevated cortisol, probably a good significant time of the day, that's going to drive gluconeogenesis, that's going to drive the liver to do this.

So when we look at all the things that you recommend and the stuff we were talking about just a minute ago with lifestyle, we have to protect our sleep.

We have to get good physical exercise to burn off that stress chemistry. We have to learn to say no. We have to do these things that help protect the body's perception of what's going on so the hormones can respond appropriately.

Because when we're in perimenopause and menopause, we have a less than stellar metabolism because our body is going into conservation. Makes total sense, you know, And word no is a complete thems.

Absolutely. All right. So, Betty, a little bit earlier you talked you brought up the topic of mitochondria. Would you share with us what the function is of the mitochondria that you referenced it as a powerhouse before it?

And I have a specific question to ask you about mitochondrial function as we continue to look at these issues that affect health and the development of blood sugar dysregulation.

Yeah, somatic, yeah. So mitochondria are known as the powerhouse of the cell, so depending on the type of cell you have, you may have thousands of little powerhouses in it and then other cells may have less.

And so but, but they also produce things. Right? So but for this, the argument of this conversation will focus mostly on their powerhouse activities. So inside, inside the powerhouse, we can we can burn different types of fuel so we can burn glucose, right?

Blood sugar and we can burn fat. The most important thing about the mitochondria is the mitochondria must have oxygen. So think of it as a coal burning power.

Power plant or like a fire. You can't burn a fire without oxygen. Right. And actually, the fastest way to put out a fire is deprive it of oxygen. So. So we have a coal burning plant.

It's going to it's going to start firing up. And the more fuel we bring in, the faster and hotter it's going to get. So the mitochondria are our biggest place to make energy.

So think of that as your high pass sports car, right? We make if it's running perfectly and we're doing glucose burning really, really well, we'll come out with about 36 molecules of the energy molecule called ATP.

Now we have the capacity to burn fuel outside of the powerhouse when we need to in a squishy part of the cell out by the nucleus. So think of that as kind of a little area outside the powerhouse.

And I like to refer to it as the campfire, right? We have the capacity in the cytosol to make a little campfire, generate some heat. We can do that, but we can't do it for very long because in most cases, that campfire can be can be created and used in a robot condition where we've got a lot of oxygen, but it's generally anaerobic.

So what that means to everybody is we don't have enough oxygen. So our body is designed to do either one because there are times when we will need to use that campfire.

So I'll give a really good example. If I'm running up a flight of stairs as fast as I can, I'm going to start huffing and puffing. And when I get to that last step at the top, I'm probably out of air.

You know, that's almost the worst breath you take. It's like, right when I get to the point where it's difficult to breathe and I'm losing oxygen, my body has shifted the the activity of burning into the campfire and it and it uses it uses glucose.

And another thing called phosphate creatine. You can only do that for a short period of time. That's why you can't sprint for 20 minutes. It's that it's like you're going to burn through that really, really quickly and it produces a ton of lactic acid, right?

So, so we can't do that for very long, which interesting is like interval exercise causes you to flip back and forth between the powerhouse to the campfire, to the powerhouse, to the campfire.

And it's that switching that tells your body heat, go make more mitochondria biogenesis. Right. Cool. Make them. But when I'm out in the campfire, that's the equivalent of being in a four banger Hyundai from 1984.

So so sorry. Since it's just the reality of it, everybody that's older on this is like, Oh, totally. I remember those. And so so what it means is as I produce four molecules of ATP in the campfire, I produce 36 in the mitochondria.

So mitochondrial function is vital to your body being able to make energy appropriately. And diabetes to some degree is is also a mitochondrial problem.

It's we're not getting glucose into the powerhouse like we're supposed to because it's getting blocked because insulin is resistant at the receptor. And before that we become glucose toxic.

So it's like we shove too much coal in the plant and they can't get it moving quick enough. So so mitochondria are really important not only to produce energy, but it's part of that entire picture.

And I think that it's an underlying piece that doesn't get explained around one of the key hallmark symptoms of diabetes. Really, any kind of diabetes, you know, type 2 diabetes, prediabetes, type 1.5 diabetes, type one diabetes, metabolic syndrome, PCOS, and often resistance is this issue of fatigue, of tiredness.

Their mitochondrial powerhouse production is compromised and this is part of what's going on. Okay. So with this in mind, now you're talking about these key changes in mitochondrial function that can lead to like to diabetes and metabolic derangements along the way, particularly for women in menopause.

So what's going on here with this setup? So so when we look at women in mitochondria, so obviously the mitochondria are slowing down, right? So your powerhouses are all at half mast, right?

So and they're not quite as effective. So estrogen affects the mitochondria in seven several different ways. So estrogen has receptors on the mitochondrial wall.

So so estrogen actually enters into a cell, goes in to the powerhouse and sort of clicks in. And so estrogen affects the signaling of several different pathways.

So one of them is the the passive transport of glucose into the mitochondria called the gloop for transport.

Mitochondria, Energy Production, and Fat Storage

So think of this as I like to think of it as a game that we used to play as kids Chutes and Ladders, you know, And so you get to the ladder, you get to slide down the ladder.

Well, think of it as that. Your ladder is not really tilted at a real high angle. The incline is sort of just barely like it's a 7% grade, just enough to feel it, but not enough to really get somewhere.

So so that that's passive transport of glute for is stimulated by estrogen. And so when we lose estrogen, we can't get glucose into the into the mitochondrial activity as well.

Right. So we see we see that we also see the signaling of of actual like muscle tissue, Right. So that the actual innervation of muscle tissue and fat tissue to mobilize fat is impaired.

And that's because the then the what what am I trying to say? It is the activity of your nerves talking to your fat cells that stimulate epinephrine to transport.

Right. Estrogen has an interplay there, too. It's why we get subcutaneous fat like on the outside of our thighs, the jiggly bits that we don't like, those those, the innovation, the discussion to those tissues to mobilize the jiggly bits on the outside, the subcutaneous fat become impaired.

And so we also can't get the fat out of the fat cell as efficiently. But even more importantly. So if we look at what's happening inside the cell, one of the mechanisms that again is a protective mechanism in the cell to allow the body to store fat actually came about millions of years ago when we moved from some transition from what looks like a to mankind.

There was an ice age and the apes that happened to be on the planet at that time, up until that point, really couldn't store body fat. So they had to had a constant supply of food and their food was all vegetable based.

So it was mostly fruits. There was a change in an enzyme called urea case. We were no longer capable of, of being able to clear uric acid out of out of our body most mammals can.

And it was this alteration in the case enzyme that allowed our great ancestors to survive, because for the first time they were able to store fat. So your case changes a molecule called uric acid.

So uric acid is produced inside the cell and it is a signaling mechanism inside the cell to also tell that powerhouse whether to amplify or decline in activity, and it's in relationship to what's perceived damage.

So things that contain RNA. So RNA is part of your cellular mechanics, part of your cellular parts. So if there's a lot of RNA being spilled inside the cell, it's like, oh, there's some sort of damage happening.

So for anybody thinking about it, it's like maybe somebody pulled into the parking lot outside the site of the powerhouse and forgot to hit the brakes and hit the gas and banged into the wall.

So now there's damage, right? And so everybody goes, oh, we've got to slow some production down because we've got to go fix the wall, Right? Yeah. So uric acid is this is this sort of cellular injury mechanism.

And so what we know now and it's and it's been studied pretty heavily at this point is as uric acid rises inside the cell and makes the powerhouse slow down, our body then becomes more insulin resistant, it then stores more fat and it allows us to act as if we're hibernating.

This was found in hibernating animals like bears and and squirrels. Right. This is what this is the mechanism in which they gain weight so they can sleep for months on end through the winter and not die.

Right. So uric acid is a hibernation response. So what's interesting is, is when you look at women, so go out and

you kind of dig through the studies automagically.

When we hit menopause, there seems to be this significant rise in uric acid levels when we look in bloodwork, right? So uric acid levels in a blood, a very easy lab test that your doctors can run can show whether uric acid climbs.

Most people think of uric acid for gout, it's associated with hypertension, cardiovascular disease, diabetes, metabolic syndrome, obesity, all of those different things.

And the higher it goes, the more it's going to cause these sort of problems. And it is causative, it causes hypertension, it causes diabetes. And it's through this mechanism.

And so uric acid, as it elevates, becomes a surrogate marker for is the powerhouse up or not Right. Is it working? And so what the research showed is that if it's over 4.2 on a serum test, that means that the so let's say it's a six, let's say somebody came back at a six, that means that you're you're fat switch is on your body thinks it needs to stop and it will do that with carbohydrate content particularly sugars fructose high fructose corn sirup and it'll do that with, you know, aged foods, alcohol, sugar.

But that's the big stuff that we see. And so a big change in our diets, particularly in the last 20, 30 years, is that increase in high fructose corn sirup and corn sugar and all the other names we have for it, because it's triggering this all the time.

Well, as a menopausal and perimenopause as a woman, we are already dealing with an amplification of that uric acid switch inside the cell, regardless of what we're consuming, which means we have less freedom within those food categories because we might be turning it on all the time.

I can see that this becomes a trap for sure. Absolutely. Yep. Yeah. Okay. Okay, great. Thank you so much for that. All right. So now that we've walk through these particular kinds of issues that circle back a bit to what you had started to talk about with GLP one and some of these peptides and receptors.

Right? So what about these current obesity drugs for a similar time and for terms appetite like they do provide people with amazing support and for people who struggle with these things where something's clearly broken, they're finally getting some practical support, something that's helpful.

But then of course other people are using it inappropriately. That's my opinion. So, you know, tell talk to us, please, about these obesity drugs to make the tide and to his appetite.

Yes. Yes. And so, so Semaglutide. And I will say I'm not the world's biggest expert on these, but, you know, so Semaglutide obviously designed for a diabetes drug.

Is it what they call a GLP one receptor agonist? And so what it does is it binds to receptors in the brain in the hypothalamus, which is your regulator.

The hypothalamus takes information from your environment, right? So it takes information from your senses, from all your chemistry, and it starts making decisions about what needs to happen.

It sets appetite. It also, but it also talks to the pituitary, who is the conductor for the entire endocrine system. So between the two of them, they're picking up messages and they're figuring out, hey, this is what the show is going to look like.

This is a look and feel. Is it a thriller? Is it a drama? Is it a comedy? Right. And then and then the pituitary is going to tell the the band how to play the orchestra.

So there's a lot of information that goes on there. Well, well, if we are taking a GLP one receptor and we've got a binding there, it's going to talk to the hypothalamus which regulates appetite.

So both Semaglutide and his appetite are appetite suppressing right through their mechanism. And so in some cases that's going to reduce and regulating appetite.

So we're going to probably eat less and it and when you activate these receptors, it also affects the satiety mechanism, right? So it feels like you're more satisfied.

You reduce hunger, so you tend to eat less. Semaglutide also slows gastric emptying, right. And so that means, again, that you're going to have food sitting in the stomach longer.

Are you going to feel fuller longer? It's going to trigger ghrelin. And so we're going to get this message, Hey, the stomach is stretched, I'm full and and then we're going to have food hitting the small intestines as quickly.

So we're going to have we're going to have a slowing of the speed in which particularly carbohydrate content can be made into sugar and mobilized into the blood or I won't say made into broken down into its simple sugars.

The GLP one receptor agonists also regulate blood glucose levels by stimulating insulin secretion and to some degree suppressing a little bit of gluconeogenesis.

But mostly it's stimulating that insulin activity. So it it can help improve glucose control, which will lead to weight loss and reducing ideally insulin resistance.

And then it has a little bit of energy expenditure changes where we see a little bit increased of fatty acid burn. Right? So the goal is if insulin comes down and we're more insulin sensitive and we're eating less.

So I have less food available in the bloodstream in the form of carbohydrate, Ultimately your body should be dipping into the tens of thousands of calories that we have sitting in the fat cells to take it to burn.

And it has to burn it in that mitochondria in the powerhouse like it has to burn there. It needs oxygen. So you eat those drugs end up losing weight loss for sure.

Now Semaglutide, what's interesting about that one is because of its impact, particularly on the gastric emptying, it has a pretty sizable issue with symptoms, right?

With side effects, like in some of the studies were as high as 40% experienced digestive problems, nausea, GERD, changes in bowel habit. So and and and so it does result in weight loss because it has this multi mechanism sort of effect.

And it is a drug that can help with diabetes, which I'm sure you could speak to much better than I can. But the side effects to some degree, the weight loss, I think to some degree, even if you sort of dig through the literature, is probably because you just don't want to eat because you don't feel good.

There's a tradeoff, right? If people are suffering or feeling miserable, if they're having the side effects and along with benefits, I think I think it's a mixed thing, we're sure.

But if that's if they've tried everything else, truly and this is what's working and they have symptoms that are tied to the issues with unregulated blood sugar and weight, you know, it's a like a cost benefit analysis for that person and their lived experience, you know, in their skin.

It's just a fascinating moment in human history for sure. Yeah. Yeah. And, you know, the thing is, is where when I because I was very interested, I'm always interested in biochemically how these things work and you know that there is appetite which I think just got didn't it just get approved as Maduro.

Yes. Recently for weight loss. Right And it's slightly different it's a gypsy GLP one drug but but both of them have very similar mechanisms.

GLP-1 Obesity Drugs and Appetite Control

Right. The thing that I found interesting is so there's significant weight loss. And I would say if somebody had to lose a lot of weight and let's say they're diabetic or maybe they're on the thing, but but that definitely weight is is contributing that it definitely resulted in weight loss and it was significant.

But they in many cases, as they took the drug out after that first year there was weight regain right less but so what was really interesting is the people that want to use it for ten £20, it doesn't have that positive effect as if somebody needed to lose 40, 50, 60, 70 or more.

Right. Meaning that it's probably it might be a good solution for somebody if if they're really struggling. They've tried everything else and they're not losing weight to know that.

But the thing is, it's expensive, it's injectable. You have to take it over a long period of time. You have to slowly titrate up to some of the symptoms, get better if you go very slow, it increasing the dose and that coming off of it is a very slow ride because because there is a weight regain and a rebound weight gain.

But in the person that's trying to use this for a £20 weight loss like I want to get ready for the summer because it's bikini season, the weight regain potential is is I think outweighs the use of that drug because it from the studies it doesn't look like it's very effective and that there is a rebound weight gain and I don't think it's something that should be used as a I'm just going to do it every spring and, you know, and drop that toe and then I'm good.

You know, I don't know what you think, but I was like, I'm a little cautious. I if it looks too good to be true, it probably is. I agree with you with that.

I think that those drugs should be for the people for whom they were designed and intended and where the strongest body of research is. So for the ones that are for people with type 2 diabetes, absolutely.

The ones who are obese and are not able to lose weight any other way. Yes. And I think for people who have just a few pounds to lose who are doing this almost like it's some sort of a sport or much more casually, it's not lifesaving for them.

I just think it's a it's a wrong approach. And that speaks to, I think, a different set of issues. That's that's my own both personal and professional opinion about that.

Yeah. Now to a tied had slightly less symptoms right so because it has a slightly different mechanism and so it seems to be I would say I think it's I think it's going to be a better drug overall, you know, in that respect.

But but I think that there's so many other solutions people can have is weight loss is their only goal. Right. Like I said, I agree with you. I think from a diabetes standpoint, it's it's it it's a really, really good drug used in the right population.

Yeah. And so far, the research is showing in terms of rebound weight gain. If people were taking the drug and it was prescribed, you know, and they're using it for the correct reasons that they rebound, weight gain is about two thirds.

So let's say they need to lose 100 pounds and then they go off of the drug about 66, 67 pounds, come back for a net loss of 33, just to make a clear, simple example, you know, And so I think in that case, perhaps it's buying people time and they can correct some other things and maybe start to get some success.

These things are today complex and multifactorial, as we'll talk about in other sessions with other experts brands. Just please understand that there's no one size fits all approach that works for everyone 100% of the time.

I wish it were that simple. You know, the 1950s advice was eat less and move more. And now in this era, we really have to all as health professionals up our game.

And for people who have the problem, you really will have to insist that whoever you're working with can meet you where you're at, helping, where you need to go.

Yeah, well, you know, the beauty of what you're doing here with this summit is, you know, people are going to get information about the different medication choices and other things, but they're going to get so much more from you and all the other experts as well about dietary changes and lifestyle changes that even if they're using this medication.

Right. You've got to remember that people that did these studies were not probably given any dietary thing. It was like, hey, just go on this medication, do it, you know, because they don't want any confounding variables, right?

They don't want to add anything else that might make it more effective. We're just going to do the drug. So they were still eating the same things. And then when they got off the medication, they went right back to the same standard American diet, which is a diet guaranteed for metabolic derangement.

Right. So people listening to this are going to learn so much about how to do things better. So even if they're using this drug to reverse or diabetes, they're going to be so much better off.

And what the research showed. Agree, agreed. Okay. So with that in mind, let's get to our next topic, which is what can someone do who's in transition to menopause?

So this is a woman. She's perimenopausal and the actual end of the road in terms of the menstrual cycle is coming. Right. So she's and clearly in transition, the menopause.

If she's struggling with blood sugar control, she's struggling with diabetes. What can be done to help her fix some of those underlying causes? Yeah, So, you know, a lot of it's going to, you know, follow probably along with with many of your other experts that are on here.

So I think the first thing that we have to understand is that 88% of us are insulin resistant and you can be insulin resistant and still body normative, meaning that your BMI is perfectly fine.

Right? You're not overweight. Right? That just means your cells, particularly muscle cells and different cells in your body, are not capable of using glucose properly.

So I think one of the most important things that is valuable for people to do is we need to move more, right? If our muscles are our primary target for glucose utilization.

Right. So I want to put a bunch of, you know, fuel cells into my Tesla. I need to I need to take the Tesla out for a ride. So so that means I need to move more.

Right. And that and that means that I need to do some interval training. So think I want to move back and forth between the powerhouse and the campfire right back and forth.

And what's cool about interval and verse training is it's different for each person. One person that's super athletic, they're going to be more intense than somebody else.

You're just looking for that. So we need to do a little bit of that fuel switching source and fire switching source, but we also just need to move more.

In general, I think people forget that, that we need to teach the cells in the muscles to be active. And and what that really means is that we need to do some low intensity to moderate intensity cardio, not cardio like my heart rates above the fat burning zone.

So most people, when they do cardio, they overdo it, right? So you can't truly have a conversation with somebody. You're not burning fat, right? So you have to be able to still have a conversation.

But so for most people, if you're in your forties to fifties, you're looking at like a 120 to 130 heart rate probably, you know, if you're de conditioned, maybe a little bit slower.

But we just need to do that, but we need do it for time. It is not really effective if you don't do it for at least an hour, you know which and it's in and it's almost better to do it one day and to say, okay, I'm going to do at least an hour one day.

It's like, teach your body that.

Lifestyle Strategies for Perimenopause and Blood Sugar Control

And then throw in these other exercise things so your muscles become sensitive because that's going to help you move glucose and recognize that if you're a woman in perimenopause menopause, that you're going to probably have to amp it up a little bit more because your messengers from your nerve cells aren't talking to your fat cells at the same level.

You know, half of them went on break, right? Are you saying things change. And they do. They do. Well, you know, because I hear this all the time. I know there's metabolic things going on, right?

Like if we were to look at bloodwork constantly, we could see glucose is getting more sensitive, all that other stuff. Well, we get mad about the jiggly bits on our butt and thighs, her abdomen, but that's the subcutaneous fat.

That's the first and last off and that's the stuff that's very hard to move. And so you've got to think about when your body becomes more insulin sensitive and you're more capable of burning fat because insulin is down, because it's got to come down in order for fat burning to come up is your your your muscles are like a prime rib.

There's fat running through them and then there's fat on the outside that you see. It's got to burn through all that fat and the muscle tissue first before it touches the subcutaneous fat.

So we get frustrated if we work out for three weeks and we don't see a change in the way our pants fit. And it's like just, no, hey, I'm burning through the prime rib, and then it's going to hit that subcutaneous fat on my body and it'll slowly burn off some of the booty.

Right. That we have. We have to hit a little harder. We got to and we got to weight train right. Got a weight train. Ladies, I have a specialty in osteoporosis.

You got a weight drain. We're going to protect our bones and our muscles. Here's my kettlebell weight training. People do it. They got to do it right. You can't get bulky.

You can't get bulky. I promise you, it's impossible. Even if you took hormones. Yeah. So weight. Yeah. So the exercise is important. So. So the other side of it is that we got to make sure that we're not doing things that trigger some of those mitochondrial things.

Right. That I was talking about. I don't want to turn on the fat switch. I don't want to get my body using, you know, using just glucose or making glucose instead of burning fat.

So the first thing is obviously, everybody is going to say this processed foods, if it's got a bag box or a label on it, and especially if somebody is promoting it on TV, you don't eat it.

Stay out of the middle of the grocery store. There's nothing healthy for you there. Right? That's all profits for big food, right? So you want to shop the perimeter, You want to eat a lot of vegetables.

You want to eat healthy proteins, you want to eat healthy fats. You want to if you're going to do starchy carbohydrates, get them from either healthy legumes, your starchy vegetables, healthy fruits.

Right. And sticking to those arenas and stay away from things that have been processed, milled floured, that kind of thing, because that just makes it faster to turn into sugar.

But it also is going to pull that uric acid level up, those foods trigger it and you want to stay away from sugar table sugar, fructose, high fructose corn sirup don't fall for agave.

It's all fructose. You know, you want to you want to avoid those foods because they're going to turn up uric acid very quickly. Right. Because you got to remember, you're already you're already battling with a little bit of flat switch like sliding upwards.

So we can't get away with I'm having just one coke a day or Sweet tea, I had a conversation yesterday. She's like, Why do sweet tea? I'm like. Are you buying it? Like, it's sweet.

That's got fructose babe. It's like put some stevia or low hanging fruit in it. We just you can make your sweet just make it with something that's a non caloric sweetener that's healthy, right?

Yeah. So we off that all together so you're not checking your brain because the Facebook the brain thing. The whole percent. It's a nasty. Nasty nasty Well and it messes with your gut microbiome and it does dysregulate insulin.

But I do have people that are like, I'm dying if I don't have them. Like, okay, one packet of sweet stevia a day looks. Bargaining. Yeah, it's bargaining, right?

So, you know, work your way off of those things because you don't want to drink your calories right now. Give be another one. Don't drink your calories ever.

You know, because our brain doesn't register food as liquid calories. Right. You to your body, your brain registers it, you know, through the pre-op reception in the brain.

You know. And I'd say the other side of it is I hear from women a lot, you know, especially, let's say they've done a lot of low carb dieting, you know, cause there's a lot of women who are insulin resistant that are still low carbon it right.

Carbs aren't the only game and that a lot of times they're eating protein and then I'll hear, oh, I'm eating a lot of protein. I'm having a protein bar, I'm having a protein shake, I'm eating, you know, my protein at my meal.

And then I'm like, what? You know, what vegetable you have? And they're like, Well, I had one at dinner, so you got to remember your your liver can make glucose and your your liver can also make insulin a response to high protein meals and a big meal.

So it's appropriate protein, but we need lots of fiber, lots of veggies. Right. And we we want to make sure that that's balanced. So those would be my biggies.

Those would be my biggies. All right. Great. Great. Thank you so much for that wonderful summary. You know, to take homes along the way of like, how do you make this work in today's world?

Not in a theoretical world or in in a lab or in a lifestyle that very few people enjoy. How can you know the vast majority of people really benefit? You know, Betty, thank you so much for your advice, your expertise and your insights today.

If people want to get in touch with you, where can they find you? How do they get in touch? Sure. Sure, they can find me at BettyMurray.com. And that's B E T T Y M U R R A Y.

And you can also catch me on my podcast called Menopause Mastery. And it comes out once a week. And that's a great place to see me. Great. Thank you so much for being such a great guest and giving us, you know, all of these

pieces of the puzzle, the points of view and the aspects of something that I think sometimes people just feel really confused by or perhaps don't necessarily have the hope, inspiration that they need.

And I know today that you helped give people that hope and inspiration and clarity. Clarity. All right, friends, so thank you for watching this session.

Please share this with anyone that you know who cares about their health, anyone who's going through menopause or who has pre-diabetes. Type 2 diabetes, This affects so many people.

There's a lot of needless struggling and suffering. If you would be so kind as to share this with the people that you care about, that would be fantastic.

All right, Betty, thank you so much.