



Midlife Clarity with Dr. Tracy Page

"Clarity Changes Everything"

Hosted by Dr. Tracy Page

Episode # 7 — Why Your GLP-1 Stopped Working

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Hi, I'm Dr. Tracy Page. Welcome to the podcast, Midlife Clarity. Today I want to talk to you about why your GLP-1 medication stopped working and what you can do about it. So, raise your hand if this sounds familiar to you. You started a GLP-1 medication. Maybe it was semaglutide, which is also known as Ozempic or Wegovy. One is for prediabetes and one is for weight loss. Or maybe it was Tirzepatide, which is also known as Mounjaro for diabetes, or Zepbound for weight loss. All of them are FDA approved. And you've been on it for a few months. And maybe your results were incredible. The appetite suppression felt like a superpower. I always say that the food noise or food chatter, it went away.

That's what a lot of my patients tell me. The scale is moving. And for some of my patients, you know, and you might be this one too, you felt hopeful for the first time in years that your body could actually lose weight. But then it stopped. I see this all the time in my practice. The scale didn't just slow down. It stopped. Or worse, the patient started gaining weight back. You're eating less than you ever had before. And all of a sudden now you're exhausted.

You feel puffy. You have zero interest in sex, zero energy, and zero motivation. And when my patient saw me, her doctor had suggested adding another medication to help her. So if this is you, or you notice that the scale has slowed down, and you're having trouble, and don't understand why, I need you to hear this. You're not failing your medication. Your medication is actually failing to address what's actually going on in your body. And today, we're going to change that. So welcome back to Midlife Clarity. This is where we don't settle for just band-aids. We want root cause solutions, and they're there to be found, and we're going to go over them today. So meet my patient. As you know, I change the names. I take bits and pieces from different patients to make up a patient case, and this patient we're going to call Diane. Let me introduce you to Diane. She's 54 years old. She works from home. She has a high-stress job.

She came in with three main complaints. Her weight loss had completely stalled, despite being on a GLP-1 medication that was working. She had zero, I mean zero, sex drive. And she was dealing with persistent puffiness and fluid retention that was worse



whenever she slept poorly. She had previously been on some supplements for cortisol. And these were to help her adrenal glands manage chronic stress. And she had recently stopped them. And almost immediately, she noticed things got worse. She was also on an antidepressant called Paxil. And she had a history of thyroid dysfunction.

She had a copper IUD. And she had a history of fibroids. Here's what stuck with me about Diane. She was generally trying. Genuinely trying. I mean, she was trying. She was on a GLP-1. She was motivated. She was showing up despite being stressed and tired. But her body was in a state of biological resistance, and nobody had helped her figure out why.

Let's look at Diane's labs in her full picture. So here's what her workup revealed and what each number means, because you may have some of these numbers yourself, and it helps us understand why she was on a plateau. First of all, her thyroid. So her free T3 was 2.5. This is low. Optimal is about 3.5 to 4.2 based on the lab standards that we use, and every lab is different. So this is an active thyroid hormone, and this runs your metabolism. Her free T4 was low — insufficient raw material to convert into active T3. You need free T4 to make T3. And her TSH was normal, which is exactly why this pattern gets missed all the time in standard labs. So often we just check a TSH, and I always say it's like only checking one of four tires on your car. Her hormones — her estrogen, which we measure as estradiol, was 51, down from 120, so there was a significant decline in driving symptoms. Her progesterone was low, unbalanced against estrogen, so it was contributing to fluid retention and her mood. And her testosterone was 15, which is low.

It dropped from 33. Her libido, her energy, her muscle mass were all affected. What do we see metabolically? So her glucose had improved. Her GLP-1 was helping here, as was her diet. Her hemoglobin A1c, which is a marker of her blood sugar over the last three months, had improved, and there was a positive trend going down. I like most of my patients to be between 4.9 and 5.2, and her level was just above 5.3 — she was at 5.4. Her protein intake was low — a critical gap that was actively blocking her weight loss, and we're going to talk about this more. And cardiometabolically, her advanced panel showed an LDL particle count of 1,435, which was high. It was elevated despite



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her successful weight loss. So her vascular risk still remained, and her HDL was low — insufficient cardioprotective cholesterol. That HDL cholesterol is so important for protecting us, and hers was not high enough.

Her MPO, myeloperoxidase — an advanced marker of inflammation in the vasculature — was elevated, so she had active vascular inflammation occurring. And we also looked at her micronutrients. Her vitamin D3 was low at 28 — low because we consider optimal for metabolism and immune function to be between 60 and closer to 80. Her B12 was 1,159, which was slightly high due to supplementation, but not a concern — we'll still monitor her because she was in what we consider the normal range. Now, here's the thing I want you to notice. Diane's glucose and hemoglobin A1c are actually improving. The GLP-1 is doing its part of the job, but her weight has stalled. Her inflammation is elevated. Her thyroid is suppressed. Her sex hormones have fallen off a cliff, and her protein is too low to support the muscle mass she needs to keep her metabolism running.

So this is not a dose problem. This is not a lack-of-medication problem. This is really a system problem. Okay, so why do GLP-1s plateau? Why does this happen? Why don't they just continue working? And do you always have to go up in dose to continue to get weight loss? So here's the education block. Understanding the biology of stalled weight loss will really help you as you're working on your own weight loss journey, whether or not you're using a GLP-1 or no medication at all.

So let's get into the real science of what's happening in Diane's body, because this is where most people — and honestly, most medical professionals — get it wrong. And I think I got it wrong too when I first started. I have seen a lot of patients. I've had the privilege to take care of many patients, and they have taught me so much about the mechanisms of weight loss, the foundational tools of weight loss, and what to do when you hit a plateau. So GLP-1 receptor agonists are powerful medications, but they are not metabolic free passes. They work with your biology, and when your biology is fighting back, the medication will always lose. So reason number one: you're eating less, but you're eating wrong.



The GLP-1 medications dramatically suppress our appetite. That's the point of the medication. But what happens when a 54-year-old woman eats dramatically less without making sure that what she does eat is almost entirely protein? Think of it this way. Think of your metabolism as a campfire. The GLP-1 is reducing how much wood, which would be food, you can put on. But if the wood you're putting on is damp and low quality, the fire gets smaller, not hotter. Eventually, the fire starts to go out. That's what happens when we don't put quality food in. When protein intake drops too low, your body breaks down muscle to meet its amino acid requirements. Less muscle means a lower resting metabolic rate. A lower metabolic rate means fewer calories are burned at rest.

And fewer calories burned at rest means, even on a GLP-1, the scale stops moving. Or worse, you start losing the very tissue you need to keep the weight off long-term, which is muscle. And I always say muscle is the endocrine organ. So, protein requirements on a GLP-1: the minimum amount of protein you should be getting is 1.2 to 1.5 grams per kilogram of body weight daily to preserve your muscle. Every pound of muscle lost reduces resting metabolism by approximately 6 to 10 calories per day. Compounding over months, that can be pretty significant. Women over 50 are already losing 1 to 2 percent of muscle mass per year just naturally, just from aging. So inadequate protein on a GLP-1 dramatically accelerates this loss. Studies on GLP-1s plus inadequate protein show up to 39% of total weight loss can come from lean muscle mass, not fat, without strategic protein intake. So think about this — if you lose your muscle but you don't lose body fat, sure, you'll take up less space, but you'll be "skinny fat."

And it does not look good, and it certainly isn't good for you, and it doesn't feel good. So adequate protein on a GLP-1 preserves muscle, maintains resting metabolic rate, and sustains weight loss beyond the initial phase. Diane's protein was low — not a little low. It was clinically insufficient. This single factor is likely contributing to more of her plateau than any other variable, and it's one she can fix. Now, I have some patients who have been on semaglutide — Wegovy for weight loss — and some who've been on Tirzepatide, which is Zepbound. And I will tell you that if their appetite is suppressed



too low, this is the number one thing that happens. The scale stops moving, they're eating less, they're not eating protein, they say "I have no appetite," and then we see them either lose muscle or not lose weight at all. Reason number two why the scale stopped for Diane: her thyroid was quietly shutting down the furnace. Diane's TSH was normal, and if her doctor only checked her TSH, which is the most standard panel, they would have told her her thyroid was fine. But her free T3 was only 2.5.

That's pretty low. Free T3 is the hormone that actually runs your metabolism. Think of the thyroid system as a thermostat and a furnace. TSH is the thermostat reading — it tells you what the setting is. Free T3 is the actual heat coming out of the furnace. Diane's thermostat looked fine, but her furnace was running at half capacity. There was no heat. And why does this happen? It's because T3, the active form, has to be converted from T4, the storage form. And that conversion requires adequate calories, adequate protein, low inflammation, and low cortisol. When any of those factors are compromised — and in Diane's case, all of them were — the body preferentially makes something called reverse T3 instead. Reverse T3 is a metabolic decoy. That's how I think about it. It occupies the thyroid receptor but does nothing. It's a parking spot taken by a car that will never move. Or sometimes I explain it to my patients this way:

Free T3 and reverse T3 are twins. There's only one seat at school. When reverse T3 sits there, it does nothing — it sleeps. And when free T3 sits there, it gets all its homework done. It's a superstar, runs track, and has so much energy. And that's why we need those thyroid receptors to see free T3. Optimal range for free T3 is 3.5 to 4.2 for metabolic function. Diane's was at 2.5, which represents roughly 30 to 40% below optimal.

Her low-T3 state reduces basal metabolic rate by about 15 to up to 40%, and this alone can fully account for a weight loss plateau. So you have to know what your thyroid numbers are if you're on a weight loss plateau, or even before you start losing weight. Like I said, check all four tires — all the numbers. Free T3, free T4, reverse T3, TSH — and then you have your two spare tires, which are your TPO antibody and your TgAb, or thyroglobulin antibody. Calorie restriction plus low T3 is a recognized physiological



combination that makes the body aggressively defend fat stores. So when you restrict, which you think is going to help you lose weight, and your T3 is low, your body is going to purposely hold onto body fat.

It is protecting itself. A normal TSH with a low free T3 occurs in up to 40% of clinically hypothyroid women, and standard thyroid panels miss this entirely — they only check a TSH, and if TSH is high, then they only check a free T4. GLP-1 medications do not correct underlying thyroid suppression. Appetite reduction without thyroid optimization leads to metabolic adaptation, and that's what's happening to her. Her metabolic system is adapting to its environment. A normal TSH does not mean a healthy thyroid. That's super important here.

It means the pituitary is satisfied. Your metabolism requires more than just that one number. Okay? Reason number three why she's not losing weight: cortisol is holding the weight on. Diane stopped her cortisol support supplements, and almost immediately, everything got worse. The puffiness increased, the scale moved in the wrong direction, and the fatigue deepened. And all of that was not a coincidence. Cortisol is your body's emergency response system. Think of it that way.

When it's chronically elevated, as it is under constant stress, and there's a lot of stress out there, it's like the fire alarm is going off nonstop. Your body cannot focus on anything other than surviving that alarm. Fat burning? That's a luxury. Repair? A luxury. Sex drive? An absolute luxury. The body is in survival mode, and it holds onto every calorie it can, and it doesn't want to function in these other departments. There's a specific physiological mechanism at play here with Diane's puffiness. Elevated cortisol increases a hormone called aldosterone, which tells the kidneys to hold onto sodium and water. This results in what we see in her — fluid retention. The puffy, inflamed feeling that gets worse with poor sleep and worse with stress. It's not fat, but it adds to weight gain, and it adds to the scale, which doesn't make her happy. And it makes people feel like failures when they actually are just cortisol-flooded. Now, I'm not saying she hasn't gained any weight back, but this is certainly not helping her in terms of the number on the scale.



Chronic elevated cortisol suppresses T3 conversion by up to 20 to 30%, worsening the thyroid suppression already present. Here's another statistic:

Cortisol and insulin together promote preferential fat storage in the abdomen, even with a calorie deficit. So she's eating less than she ever has before, but she has high cortisol, and if she has high insulin, it's going to promote holding fat in her abdomen. Cortisol-driven fluid retention can account for 2 to 5 additional pounds on the scale, independent of fat mass. And poor sleep, which is Diane's trigger, raises cortisol by 15 to up to 37% the following day. This creates a vicious cycle — disrupted sleep, more cortisol, more water retention, worse sleep. HPA axis dysregulation — that's hypothalamus, pituitary, adrenal — is found in up to 90% of women who report chronic high stress with physical symptoms. They have dysregulation.

So 90% of women — and I see this in my practice all the time — women take care of everything. Women take care of everybody. Women make sure everything happens. And they usually are under a lot of stress and not taking care of themselves. Okay, so what's her reason for the scale to stop moving? The SSRI nobody talked to her about. This is the one that I want to highlight specifically, because it was the biggest miss — or one of the biggest misses, I should say — in Diane's care. And it's one of the most common mistakes I see in women on antidepressants who are also trying to lose weight and restore libido. Diane is on Paxil for depression. Paxil belongs to a class of antidepressants called SSRIs.

And Paxil specifically is one of the most potent libido killers and weight-promoting agents in the entire class of antidepressants. Paxil is associated with the highest rate of sexual dysfunction of all the SSRIs — up to 70% of patients report a significant reduction in libido. SSRIs as a class cause weight gain or weight resistance in approximately 25, and you can't believe this, up to 65% of patients, depending on the specific agent and the dose. Paxil specifically is associated with an average weight gain of 2 to 6 pounds a year and can cause weight resistance that is independent of calorie intake. SSRIs plus a GLP-1 — the weight-promoting effects of the SSRI can partially blunt the GLP-1's outcomes.



The combination is understudied and under-discussed, but it does exist, and I've seen it in the clinic. I get someone off their SSRI — I never start with that — but as they get better and healthier and their hormones are balanced and they feel good, they actually come to me wanting to get off of it, not realizing that it is one of the reasons their sex drive is so low and is also preventing weight loss. But eventually we do talk about it, because it's important for them to make informed decisions. SSRI-induced sexual dysfunction does not improve simply by addressing hormones. The root cause must be acknowledged. Alternatives have to be discussed, because if we don't address it, I can balance her hormones all day long, make sure her testosterone is good, and her libido may still be poor. So I'm not saying Diane should stop her Paxil right now — her mental health does need to be protected — but this conversation needs to happen openly, because optimizing her testosterone and progesterone while leaving her on a libido-crushing antidepressant won't solve her problem.

There are alternative antidepressants with far more favorable profiles for both weight and sexual function, and that conversation was long overdue — and we did have it. Okay, let's talk about the cardiometabolic warning. So she's having weight loss. She was having weight loss — it's plateaued, but she'd had a significant amount of weight loss already. That doesn't always equal metabolic health. So this is really important — the number that's hiding behind the scale. I want to pause here and talk about something that doesn't get nearly enough attention in the GLP-1 conversation, because there's a myth circulating that if you're losing weight, your heart is healthy, your cardiovascular function has improved. And sometimes it has, but in Diane's case, it wasn't the truth. Diane's LDL particle count was 1,435.

That's high. And her MPO, myeloperoxidase — the real-time vascular inflammation marker — was elevated, meaning she had active arterial inflammation occurring as she was losing weight. And even as the scale has been moving downward, she's still having this. So think about it this way. If total cholesterol is the number of cars on the highway — and I've used this example before in another episode — so, if total cholesterol is the number of cars on the highway, LDL particle count is the number of those cars that are



subcompact, poorly made, and prone to accidents. And MPO is the ambulance siren telling you accidents are already happening.

Okay, so I used that a little bit differently before, but let me tell you that again. So if total cholesterol is the number of cars on the highway, LDL particle count is the number of those cars that are subcompact, poorly made, and prone to accidents. And in her case, prone to a cardiovascular event. And MPO, myeloperoxidase, is the ambulance — it's the siren telling you accidents are already happening, because it's active inflammation in the vasculature that's already occurring. Here's why this matters so profoundly in the context of GLP-1 therapy. When women are under-eating protein, the body breaks down muscle, not just for fuel, but it also releases inflammatory byproducts.

And I don't think a lot of people know that. When the thyroid is suppressed, lipid metabolism is impaired and LDL particle production increases. This is a correlation I'll be honest — I stumbled upon it because I had some patients who were having crazy increases in their cholesterol despite changes in their diet, and the only common denominator was their thyroid. There is a direct relationship between lipid metabolism and thyroid function — it gets impaired and LDL particle production increases when the thyroid isn't working, especially when it's suppressed. When cortisol is chronically elevated, it promotes the formation of small, dense LDL particles — the dangerous kind.

And if you don't know about those, go back and listen to the episode on the five markers to know your cardiovascular risk — I believe that's episode number five. So, when cortisol is chronically elevated, it promotes the formation of small, dense LDL particles, the dangerous kind, and increases MPO activity, which is inflammation in the vasculature. So Diane is losing weight by the scale — it's plateaued — but she may be getting metabolically worse beneath the surface. This is the most important reason why GLP-1 therapy without comprehensive metabolic monitoring is not good enough.

You can't just take medication and think it's a magic pass to health and wellness. I have this conversation every day, because these are amazing tools, but they are not the only tool in the toolbox. You cannot fix things with just one tool, okay? The LDL particle count



of 1,435 is high. The optimum is below 1,000. Elevated LDL particle count is a stronger predictor of cardiovascular events than LDL cholesterol alone. MPO elevation means active vascular inflammation — endothelial damage is occurring and is not corrected by weight loss alone.

Protein-adequate GLP-1 users show improved lipid particle profiles. Protein-deficient users show worsening despite weight loss. Thyroid optimization directly improves LDL receptor activity in the liver. Correcting low T3 alone can meaningfully reduce LDL particle number. And omega-3s — EPA and DHA, one of my favorite supplements, a foundational supplement — 2 to 4 grams a day has been shown to reduce LDL particle number, lower MPO activity, and reduce triglycerides substantially. Okay? The scale is only one data point. Please remember that.

Your particle count, your inflammation, your thyroid — those are all numbers that predict the next 10 years of your life, not just the scale. Okay, the clinical plan — fix the foundation first. That's always important. Here's the priority-by-priority roadmap. You can use this on yourself, but you should have your numbers checked, and you should know if this applies to you. Again, this isn't medical advice — this is education, and you should see your provider.

This is the part I want you to write down, because what I'm going to walk through is a sequence that matters. This is not a throw-everything-at-it approach. This is a specific order of operations, because in functional medicine — and this is what I teach my colleagues — sequence is strategy. When and how we do something matters as much as what we do. So priority number one: rebuild the metabolic foundation — protein first. Before we talk about medications, before we talk about hormones, before we talk about anything else, we have to get protein right.

This is the single most important intervention for a GLP-1 plateau. Not a higher dose, not a different medication — it's simple. It's just protein. Target 1.2 to 1.5 grams of protein per kilogram of body weight daily. It's non-negotiable. Practical translation for, let's say, a 150-pound woman: approximately 82 to 102 grams of protein per day, distributed



across multiple meals. Protein timing — at least 30 grams at breakfast. This sets the metabolic tone for the entire day and reduces hunger-driving hormones.

And I'll tell you, this was a hard one for me — I'm not much of a breakfast eater, but I started making myself eat breakfast. The more research I did and the more I counseled my patients, the more I realized it's so important. It sets the tone for the day. Protein sources — think complete proteins: eggs, chicken, fish, Greek yogurt, collagen peptides. Actually, I don't think collagen peptides count toward your total protein, but there are all kinds of sources out there. Try to stay away from protein bars with higher grams of sugar — more than 10 grams, probably stay away from them. I'll be honest, I use protein bars because sometimes it's the only way I can get all my protein in if I'm busy. Hydration — a minimum of 80 to 100 ounces of water daily. Dehydration mimics hunger and worsens cortisol signaling. You'll actually eat more if you're thirsty. Here's a clinical reality: I have seen women on GLP-1 medications get their weight moving again with zero medication change, simply by getting protein to target. The medication was never the problem — the fuel was. It's the very first place we start, the very first question we ask. We have foundational questions we go through with all of our patients on GLP-1s. They're even in our refill forms, because they're so important for success. It takes a while for patients to pick up on that sometimes, but protein is definitely number one.

Okay, priority number two: restore thyroid signaling. In her case, thyroid was a big component. With a free T3 of 2.5, Diane's metabolic furnace is running at a fraction of its capacity. Fixing this requires two things: optimizing the conversion environment — remember, T4 converts to T3 — and, if that's not enough, adjusting her liothyronine dose, which is what she was on. T3 conversion optimization requires adequate protein — there's that P word again — reduced inflammation, cortisol control, and in her case, selenium. We supplemented with 200 micrograms a day. And I've said this before — please, please have your selenium level checked before you start supplementing, because there is such a thing as selenium toxicity, and you won't know if you're toxic if you don't know your levels. Target her free T3 above 3.2 as a minimum; optimal, as I said, is 3.5 to 4.2 for metabolic function. We adjusted her liothyronine with incremental increases and repeated her labs at eight weeks to make sure her level was in the



optimal range. The key clinical insight here is that a stimulant medication like phentermine, without first fixing a low T3, is like pressing the accelerator in a car with a clogged fuel line. That's how I describe it.

I have a lot of car analogies — I don't know why. You're not going to get where you want to go. You're going to strain the engine. Early in my career, I may have even done this — added something like phentermine, an FDA-approved medication for weight loss. But what I've learned is that foundations are key. Take care of the foundational things first. It might be necessary later on, but not when you haven't even fixed thyroid first. So remember, adding a stimulant medication, or any medication, to a patient who has thyroid dysfunction is like pressing the accelerator in a car with a clogged fuel line. The car is not going to go anywhere — your engine is just going to be strained.

Okay, priority number three: address cortisol and stress physiology. Diane stopped her cortisol support and got worse. The solution is not complex, but it requires consistency, and we all get pill fatigue. So that's why I'm very intentional about what supplements I put my patients on, and I try to make sure that list isn't too long. We resumed her cortisol support supplement — it gives her adaptogenic support, and these typically include things like ashwagandha, phosphatidylserine, rhodiola. These are just examples — everyone responds differently to different types of cortisol support. I'm not here to tell you which one or which supplements, because that's something you should talk to your provider about. But the supplements help buffer the HPA axis response to chronic stress. What else did I ask her to do in addressing cortisol and stress physiology? Morning light exposure — 10 minutes within 30 minutes of waking. It resets the cortisol rhythm and reduces evening and overnight cortisol spikes. Phosphatidylserine is one of my favorite cortisol-reducing supplements — 200 to 400 milligrams at night, specifically shown to blunt cortisol-driven sleep disruption.

And one of my other favorite supplements — I think this one is foundational — magnesium glycinate, 400 milligrams at night. It reduces cortisol, supports deep sleep, and reduces the fluid retention driven by that aldosterone excess we talked about with cortisol. And the last thing: post-meal walks. 10 to 15 minutes after meals, just walking. It



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reduces cortisol-driven glucose spikes, improves insulin sensitivity, and meaningfully supports weight loss momentum. It's such a simple thing to do. I tell my patients I would rather they do three 10-to-15-minute walks a day after eating than go to the gym for 30 minutes — you're going to get more out of it, because it's working in real time to help reduce your glucose and your insulin.

I want to say something about stress that rarely gets said in the medical setting. The stress load that high-achieving women carry is physiologically real — work from home or not, with constant demands. I tell my female patients, it's not a mindset problem. It's a cortisol problem, and it needs to be treated. You have to treat the stress that goes on in your life, because it does affect you, and we don't really talk about it. At our practice, we talk about it a lot — it's one of those foundational questions we ask our patients all the time.

Okay, priority number four: pause on adding additional medication. Something in Diane's care plan that gave me pause was the instinct to add more medication — whether phentermine, metformin, or another agent — to help with weight loss on top of the GLP-1, without first fixing protein, thyroid, and cortisol. It's like treating a symptom and ignoring the mechanism that created it. Let's talk about phentermine, because it's a stimulant — it stimulates the adrenal system, increases dopamine and norepinephrine. In a patient who already has cortisol dysregulation, adding adrenal stimulation risks worsening HPA axis dysfunction. It worsens or increases the risk of sleep disturbance, and it makes the adaptive plateau worse — she'll have an even harder time losing weight because it messes with that HPA axis. Again, it's treating the symptom and ignoring the mechanism. This medication may have a role, but only after the metabolic foundation is solid. GLP-1 plus phentermine combinations show modest additional weight loss in some patients, but increase heart rate, sleep disruption, and adrenal load. You have to be careful with whom you use it and why. Phentermine is most effective when protein is adequate, thyroid is optimized, and cortisol is stable — it's not a first response to a weight loss plateau.



Okay, priority number five: address the libido root cause, and do it in the right order. Zero libido in a woman with low testosterone, low progesterone, and on an antidepressant known to suppress sexual function is not really a mystery — at least not for a physician who does this work — but the solution requires honesty and proper sequencing. Step one: have an open conversation with your doctor about your medications, and ask them — do my medications cause weight gain? Do my medications cause libido suppression? You have every right to ask that. There are alternative antidepressants with lower sexual side-effect profiles — Wellbutrin, which is bupropion, for example, is associated with improved libido and is weight-neutral; it doesn't cause weight gain or weight loss. You want to optimize the thyroid and cortisol first — both directly suppress testosterone and libido when dysregulated. Step three: consider low-dose testosterone optimization. You can use a cream, a pellet, or an injection — those are my three mainstays. I'm not a big fan of troches, because they cause such high spikes in serum levels. But cream, pellet, or injection — we target free testosterone in the upper quartile of the normal range for a woman. Step four: add progesterone support, cyclic or continuous depending on her cycle status, which reduces fluid retention, improves sleep quality tremendously, and supports mood alongside libido. Testosterone is the hormone of desire; progesterone is the hormone of calm. Both matter a lot, but neither will be fully effective if cortisol is flooding the system and an SSRI is blocking the receptor. You have to take care of those two things.

Priority number six: cardiometabolic cleanup. This is the longer game plan, but it starts now, and it has to be discussed with anyone on a weight loss plateau. Omega-3 fatty acids, EPA and DHA — 2 to 4 grams a day — reduce LDL particle count, lower MPO, reduce triglycerides, and have direct anti-inflammatory effects on the vessel wall. I'm a huge, huge fan of good-quality omega-3 fatty acids. Increased dietary fiber, 25 to 35 grams a day, reduces LDL particle number through bile acid sequestration and supports gut health and cortisol metabolism. So you'll hear me, podcast after podcast, talk about fiber. Fiber, fiber, fiber — you cannot get enough of it. And vitamin D3, 5,000 IU a day with food — I always recommend D3 with K2 — will correct a level like her 28 to a target of 60 to 80, closer to 80. Vitamin D directly modulates LDL receptor activity and immune-mediated inflammation, so it's so important in getting her healthy. We recheck



her LDL particles and MPO about three to four months after metabolic correction, and I'd expect a meaningful improvement in her thyroid numbers and her inflammation numbers as well.

Okay, your action plan — what do you do this week? Here are seven steps for women on a GLP-1, and for women who aren't — it really applies to both. Sometimes we think, "well, I'm not on a GLP-1," but if you're on a weight loss plateau, or feeling like your body is working against you, these steps apply to everyone. So here's your roadmap — and if you're doing them, check the box.

Number one: track your protein for one week. No calories, just protein. Use a free app like Cronometer or MacroFactor. I guarantee most of you will be shocked at how far below target you actually are. For a 150-pound woman, the target is roughly 85 to 100 grams of protein per day. And if you're on a GLP-1 eating 1,200 calories of crackers and soup, you're burning your own muscle — please stop that.

Number two: ask for a complete thyroid panel, not just a TSH. Use these exact words with your doctor: "I'd like a free T3, free T4, reverse T3, and TSH, as well as TPO antibodies added to my next blood draw." If your doctor says TSH is all you need, that's information too. A TSH of 2.8 with a free T3 of 2.5 means your metabolism is running low. You deserve to know that. If they won't check a full panel — remember, checking just TSH is like checking one tire out of four, or in this case, we've got two spares as well — find a functional medicine doctor who will.

Number three: evaluate your medications honestly. If you're on an antidepressant and you have zero libido and can't lose weight despite putting in all the effort, have a conversation with the doctor who prescribed it. Ask about the side effects. Do your own research, because it might be one of the medications causing your lack of libido. There are plenty of alternatives, and there are opportunities to get off medications too.

Step four: take sleep seriously — it's a cortisol intervention. I always say sleep is the only thing I can give you that costs you nothing and will lower your cortisol. Diane's puffiness



got worse with poor sleep, because poor sleep spikes cortisol, which causes us to hold onto water, which blocks fat loss. Start with magnesium glycinate, 400 milligrams before bed, and 10 minutes of outdoor morning light within 30 minutes of waking. These aren't luxuries — they're hormonal medicine.

Step five: add omega-3s if you haven't — 2 to 4 grams of EPA and DHA daily. Not flaxseed oil, but fish oil or algae-based omega-3s with EPA and DHA directly. I'm a big fan of flaxseed and use it all the time, but for EPA and DHA specifically, you want algae or fish oil. These address LDL particle count, vascular inflammation, and support thyroid hormone conversion. It's one of the highest-value supplements in any woman's protocol — it lowers general inflammation too.

Step six: if you're on a GLP-1, do 10 minutes of walking after your two largest meals of the day. Start there. This isn't optional cardiovascular advice — this is metabolic medicine. Post-meal walking reduces glucose spikes by 20 to 30%, improves insulin sensitivity, and counteracts the cortisol-driven metabolic slowdown we see in patients using a GLP-1.

Step seven: ask your doctor to check your LDL particle count and MPO. Honestly, I want them to check all five — Lp(a), apoB, LDL particle count, MPO, and oxidized LDL — not just the standard cholesterol panel, which you still need. If you're losing weight and feeling good about your labs, but nobody's checked your cardiovascular system, you could have vascular inflammation and cardiovascular issues despite losing weight. You're driving without a speedometer, so you need to know. This advanced cardiometabolic panel exists, and you can ask for it by name — I believe I mentioned it, CardiolQ, in podcast number five.

Okay, so remember: you're not failing at your GLP-1. The GLP-1 is failing to get the support it needs from the system around it. GLP-1s work best in a good system. Fix the system, and the medication can do the job it's supposed to do.



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So, a few closing teaching pearls. I want to leave you with one thing to carry out of this episode. Weight loss plateaus on a GLP-1 medication are almost never solved by adding more medication or increasing your dose. They're usually solved by going upstream — to the protein that's too low, the thyroid that's suppressed and not working properly, or the stress that's causing your cortisol system to run in emergency mode, often for many years. Diane came in being told her only option was to increase her dose or go on a stimulant medication. But her body wasn't resistant to weight loss because she needed more pharmaceuticals. It was resistant because it was under-fueled, thyroid-suppressed, cortisol-flooded, and hormonally depleted. Those are all important foundational things to know if you're trying to lose weight.

Our medical system is incredible at finding individual problems, but what it often misses is the pattern — the way low protein, low T3, and high cortisol stack on top of each other to create a biological environment where no medication, honestly, can fully succeed, and no effort can fully succeed either. That's what functional medicine is really about. We like to see patterns. We like to look at systems — what's the right order to treat things, using the right tools? And we have many tools in our toolbox.

So if you're on a GLP-1 medication and it's not working the way you hoped, I hope you'll do some more investigation. Get a better roadmap. Get your thyroid checked. Get your cortisol checked. Make sure your metabolism is working. Make sure you're eating enough protein. These are all fixable things. If this episode spoke to you, share it with a woman who's been told her plateau is just part of the process of losing weight. It doesn't have to be. And if you want to dig into your own labs or connect with our team, you can find us. See you next week. Your body, remember, is always working for you. Just give it the right input.