Midlife Clarity with Dr. Tracy Page

"Clarity Changes Everything"

Hosted by Dr. Tracy Page

Episode #14 — The Peptide that Targets Visceral Fat

TESAMORELIN

Listen Here

Welcome. Hi, I'm Dr. Tracy Page, and this is MidLife Clarity. And today we're going to continue our peptide series, and we're talking about Tesamorelin. This is a peptide that I love because it goes after that visceral fat, that fat around our organs. It's a very dangerous fat. So, we're going to talk about why the visceral fat around your organs is the most dangerous fat and the peptide that actually goes after it. Because not all fat is the same. The fat you can pinch on your hips or in your tummy is mostly cosmetic; underneath your arms, you know, when we have the chicken wings. The fat you cannot see, wrapped around your liver and your pancreas and your intestines and your heart, is the fat that drives every modern chronic disease, and we need to talk about it today. Most weight loss interventions take fat from everywhere pretty much indiscriminately, including the muscle you cannot afford to lose, okay? Tesamorelin, this peptide, is different.

It is the FDA-approved peptide. So, that's pretty cool. It's a precision tool. And in midlife, for women whose waistlines are creeping up more and more despite stable weight, it's often the most important peptide we could add here in midlife. Now, the FDA approval is going to be interesting because it was FDA-approved for visceral fat for a different reason. And we're going to talk about that in the podcast today. So, don't get your hopes up. It's not going to be covered by insurance unless you have HIV. But this peptide is pretty amazing. You'll want to learn more about it. So, today we're going to talk about it. And as you know, I always use a patient to explain the peptides because I think it just hits home and helps you understand it even more. Okay, so we're going to talk about Beth today. And she is 53 years old. She's weighed within five pounds of the same number since she was 28. She wears the same dress size she wore in her 30s. But every metric a primary care doctor or even I used to measure would say she's healthy, and she's within a healthy weight normal range. But now that we can measure visceral fat, we can do that on the InBody. We know if visceral fat is over a certain level, you are not healthy.

So, she came into my office last summer because of an incidental finding on a routine ultrasound she had for actually a different reason. The radiologist had noted almost in passing that she had moderate hepatic steatosis. And fatty liver is what it's called. So, she had fat around the liver. She had no risk factors for it. She did not drink heavily. She was not diabetic. Her cholesterol actually was fine. She came to me; actually, she was referred by another patient, and she was a little confused because she was told she needed to lose some weight. But again, remember, she's within five pounds of her weight at 28. So, that was a confusing instruction for her, especially since she weighed 138 pounds. And in her wedding photo, she weighed 135. So, she was like, I don't get it, right?

So, let me tell you what we found when we actually measured her. As I said, we had an in-body scale that measures body composition. You can also do this through a body composition scan. So, you can get this information two different ways. Beth's total body fat percentage was 33%, which is high for her weight, but not alarming. I mean, we try to keep our patients 28% or less, kind of in that healthy range. But the story was in how her fat was distributed. Her visceral fat, the deep abdominal fat that wraps around the internal organs, was significantly elevated. Her trunk-to-leg fat ratio was the kind of pattern we usually see in postmenopausal, menopausal, or sometimes even perimenopausal women.

So, that menopause word, it's in there everywhere, right? um her trunk like i said her trunk fat to her leg fat ratio was the kind that we don't like right her waist circumference which she had not measured in years because again she's only a few pounds since her wedding pictures had grown from 20 inches in her 40s to 32 inches now four inches of expansion that her dress size hadn't fully revealed because dresses you know they stretch just like scrubs just like a lot of other stretchy pants and they're forgiving so it kind of hid the change. She didn't really realize until we started talking about this visceral fat. Her labs told the rest of the story. Her high-sensitivity CRP was elevated at 4.8, and it indicated, you know, chronic inflammation. Her fasting insulin was 14, and remember, I

like that less than 7, and she was on her way to insulin resistance. Her triglycerides were elevated at 168, and her ALT was 42, just above the upper limit, consistent with the fatty liver, and her hemoglobin A1C was 5.7, which in some labs is considered prediabetes or on the cusp of prediabetes.

Okay, so Beth was metabolically sick. She did not know it. Her weight had been hiding it. Her dress size had definitely changed. And her primary care visits, they measured the basic things as I learned when I was going to medical school. And you would measure things like weight, the finger-stick glucose, and you can't really pick it up. So, it had been hiding. And the fat that was driving all of it was the kind of fat she'd never been told to worry about, and I'm sure you haven't been told to be worried about it either, but it's visceral fat.

There are two completely different types or kinds of body fat, and they have completely different consequences for your health. Subcutaneous fat, the soft tissue you can pinch, we don't love that one either, is largely metabolically quiet. It stores energy, so you know, when we overeat, we store excess energy. It cushions our body so we can do our sit-ups without our tailbone hurting. It's not the fat that's going to kill you. But this real fat, the fat that has built up around your liver, your pancreas, your intestines, the amount that is in, I would say, your abdomen area, is metabolically active. It produces inflammatory cytokines.

It drives insulin resistance. It is the fat that fuels heart disease, type 2 diabetes, fatty liver, dementia, and certain cancers. And here's what nobody has told Beth, or most patients don't know, that Tesamorelin is the only FDA-approved peptide that specifically and selectively targets visceral fat. Phase 3 clinical trial data show a 15-18% reduction of visceral fat over 26 weeks, while simultaneously increasing lean body mass. So, it's a pretty awesome peptide. Today, we're going to talk about Tesamorelin, why visceral fat is biologically different from the fat under your skin, how Tesamorelin selectively targets it, the generally impressive clinical evidence behind it, how it differs from GLP-1

medications like semaglutide and Tirzepatide, and critically, because patients always ask, why the scale will lie to you on this protocol and what you should be measuring instead.

So, by the end of this episode, you're going to understand why Beth's waist had grown 4 inches without her weight changing and what we had to do to reverse it. Okay, so let me tell you something that will reframe how you think about body composition, hopefully for the rest of your life. Body fat is not one thing. It's not a single tissue. It's at least two functionally distinct organs that happen to share a name. So, there's subcutaneous fat, like I said, that fat directly under your skin, and then there's visceral fat, the fat deep in your abdominal cavity, wrapped around your intestinal organs.

They behave so differently that researchers increasingly think of them as entirely separate organs. They have different cell biology, different hormonal signaling, different vulnerabilities, and dramatically different consequences for your long-term health. So, subcutaneous fat- the storage closet is what we'll call that. So, subcutaneous fat is a soft fat you can pinch on your hip, your thigh, your inner thighs, your upper arm; it's what gives bodies their, I would honestly say, their feminine shape, right? It produces leptin, which signals satiety. And it extends, right? So, if you have extra body fat in your body, you should make more leptin because you don't need more energy stores, right?

So it actually signals that you feel full. That's why you have satiety. It cushions and it insulates. So, we do need some, you know, subcutaneous fat. It does keep us warm. In perimenopause and menopause, it actually appears to be metabolically protective. Women who carry their fat in the hips and the thighs have lower cardiovascular disease risk than women who carry it in the abdomen. Even with the same total body fat percentage, it doesn't matter. And you know, we hear the apple and the pear.

So, the patient who carries most of their fat like a pear, they're going to be more cardiac-protected than the woman who carries it like an apple. So, she carries everything in the trunk, okay? Subcutaneous fat is largely a storage organ. It receives energy when you have excess. So, that's what you're storing. And it releases it when you need it. So, if you're fasting, I always joke around when I was doing intermittent fasting because I was trying to change my body composition. I'd be like, oh, my right thigh right here, this little saddlebag, it's feeding me right now, right?

When I was fasting, because it was. It was breaking down fat to release energy. Okay. It does not aggressively interfere with your metabolism. Fat alone doesn't drive chronic disease. So, that's really important, okay? But we don't like having it anyway. You do need some of it. And so my ladies who are too thin, have no body fat, as I do worry about them. So, we do need some.

We want a healthy range. 18 to 28% of subcutaneous fat is healthy, all right? How low your percentage is, your body fat percentage is how lean do you want to look, okay? And I like my muscles to show in my arms. So, I don't think there's anything wrong with looking lean. But when we get under 18%, we get a little bit nervous, right? I like to call visceral fat the inflammatory engine. So, visceral fat is a different animal entirely. It sits deep in the abdominal cavity.

It wraps around the liver, the pancreas, the kidneys, and the intestines. You cannot pinch it. You cannot see it from the outside. And a woman like Beth, with a flat-looking abdomen by external appearances, can have substantially elevated visceral fat and never even know it. So, it's a phenomenon researchers call T-O-F-I, thin on the outside, fat on the inside. So, T-O-F-I, thin on the outside, fat on the inside.

And nobody likes that acronym, just like no one likes to be called skinny fat, right? So, visceral fat is metabolically active in the worst possible way. It releases free fatty acids

directly into the portal circulation, the blood supply that goes straight to the liver. So, this drives the fatty liver Beth has already been diagnosed with. So, that's how it happens, right? So, visceral fat is metabolically active; it releases free fatty acids directly into the portal circulation. That's the blood supply straight to the liver. So, I want to repeat that so you understand how fatty liver occurs. This drives fatty liver, and Beth has already been diagnosed with it.

It produces inflammatory cytokines like TNF-alpha and IL-6 constantly around the clock. is implicated in insulin resistance, type 2 diabetes, atherosclerosis, hypertension, fatty liver disease, and certain cancers. And the dementia spectrum is even affected by visceral fat. It's the fat your cardiologist should be measuring and almost never does, just like we never knew to. And so when I always say your primary care didn't order it or your cardiologist didn't get that or whatever, I'm not pointing fingers because, you know, I was brought up in the traditional medical system, and a lot of these things I learned by my integrative medicine certification and also my board certification in functional medicine and then even my board certification in obesity medicine. So, these are the things I learned after medical school when I went on to get more education. So, I never blame others for not knowing it. I just feel like, you know, we know more when we study more and we learn more, and we expose ourselves to more, right?

Like anyone. Okay. So, picture subcutaneous fat as the IV growing on the outside of your house. It's mostly cosmetic. Some people like the look. Some don't, right? Some people like a little more fat. They look feminine. Some of them want to look like hard bodies and be as lean as can be, right? So, everyone has a different, you know, desire and a different look.

But it isn't doing structural damage. It's just sitting on the bricks. That's what IV does, right? Just like subcutaneous fat. Just sits on your body. Visceral fat is the ivy growing on the inside of your house. It wraps around the electrical wiring, creeps into the plumbing,

and wedges itself between the support beams. It's the same plant. Completely different problems.

You could pretend the ivy inside the house isn't there because you can't see it from the curb, but every year it grows, and every year the structural damage compounds. Do you get it? Okay. The numbers on visceral fat and midlife. So, here's the data. I want you to change the way you think about your body. I want you to think about your body and body composition. So, often we get on the scale, and the only thing we see is that number, right? 120, 130, 140, 200.

Like, that's all we see. But we're made up of muscle, fat, and there's subcutaneous fat, and there's visceral fat, right? And we're made up of water, and there's just so many things that make up our bones, our organs. So, it's not just that number on the scale. Menopausal women gain distal fat at approximately twice the rate of premenopausal or perimenopausal women, even when the total body weight stays stable. Isn't that crazy? The shift from a hip-and-thigh distribution to a central abdominal distribution is one of the most reliable markers of declining estrogen, and it's part of what makes midlife metabolic health so much harder than it looks on the outside. I can tell you, I have so many women, so many women, which is why I love this work, because you can change this. That come to me, and they're like, I don't know what's happening to my body. I don't know where this fat came from. I always work out. I eat well. Like, I can't get rid of it. And these are women who just want to take care of themselves. This could be you. So visceral fat increases inflammatory cytokines that elevate high-sensitivity CRP, or C-reactive protein. So, that's an inflammatory marker. It drives insulin resistance independently of total body weight.

It is more strongly correlated with cardiovascular risk than total cholesterol. Isn't that crazy? Total fat percentage or even body mass index. So, correlated with cardiovascular risk more than total cholesterol, total fat percentage, or even body fat mass. A woman with a normal BMI and elevated visceral fat has a higher

cardiovascular risk than a woman with an elevated BMI and normal visceral fat. So, you can weigh more and have less visceral fat, and you're more cardiac protected. The number on the scale by itself tells you almost nothing useful about your metabolic future. We believe so much in the InBody and what it tells us that we just got our second one.

So, now we have two. We have two InBody machines to weigh more people and give them more body composition information because this really is foundational and you need to know. And I will be honest, I just went into mine over the weekend. Wasn't too happy with my results. As I have mentioned in a couple of podcasts, I've been working a lot. My deep sleep fitness is good. Not working out quite as much, but I do. I am working on changing this. And I lost some muscle.

And sure enough, my subcutaneous fat went up, and my visceral fat went up too. So, it happens to all of us. And the link to the fatty liver- so that's what Beth was diagnosed with- it's mechanistic. So, visceral fat dumps free fatty acids into the portal vein. I just told you that. The liver, receiving more fat than it can process, starts storing it. So, that's how we get fatty liver. Over the years, the storage becomes non-alcoholic fatty liver disease. Because you can have alcoholic liver disease, right? But this is non-alcoholic fatty liver disease.

It actually can cause even cirrhosis in the liver. So, you get changes to liver function, right? And in some patients, it can even progress to fibrosis. Roughly 25 to 30% of midlife adults in the United States have some degree of fatty liver. Isn't that crazy? Most of them, they're undiagnosed. Because remember, hers was an incidental finding. Like she didn't even go in for this, right? Beth's ultrasound caught hers by accident. And most patients are never caught, like, until it becomes a problem.

So, why does this matter, especially for women? Again, it goes back to estrogen. So, in perimenopausal and menopausal women, preferential routes for fat storage go to subcutaneous compartments. So, it goes to the hips, the thighs, the buttocks when we have estrogen. This is part of why young women have a different body shape than men with similar body fat percentages, and also older women. And I don't know about you, but when I go to buy clothes, they always ask me, like, you're trying to figure out your size, and they always ask, like, what's your age range? Because they know your body changes as you get older.

And sometimes they don't want to mark my age because I'm like, well, hey, I'm working on my body. So, I don't want you to put me in a category I don't belong in. But it's true. Our body shifts, our composition shifts, okay? As estrogen declines through perimenopause into menopause, that preferential route weakens. Fat that would have gone to the hips starts going to the abdomen. Your body shape shifts, and your metabolic risk profile shifts with it. So, now you know why they ask you your age when you try to figure out your size. This is why the 4-inch waist expansion Beth experienced with no change in her weight, it wasn't anything that she was doing.

It wasn't a lack of effort or what she was eating. It is a textbook signature of perimenopausal hormonal shift, redirecting fat storage to a more dangerous compartment. So, she wasn't gaining weight. She was just redistributing it. And I hear this all the time. My patients come to me, women, and they're saying, oh my God, I have all this fat in my belly. Like I never had this before. Why do I have it?

So, visceral fat is a smoldering basement fire. You've heard me talk about basement fires before. It does not produce dramatic flames. The smoke is slow. The heat is low. From upstairs, the house looks totally fine, right? You look at her; she looks fine. But the fire is burning around the clock. It's releasing inflammatory smoke into every floor of the house. You can paint the walls upstairs.

You can have a... And so painting the walls upstairs equates to a cleaner diet, more cardio, more willpower, and the smoke keeps rising. Until you go down to the basement and you put out the actual fire, so you have to get rid of the visceral fat, every other intervention is just cosmetic. Tesamorelin is the fire crew that knows the work has to happen in the basement. So, that's what I love about Tesamorelin. Okay, I've used that kind of fire-in-the-basement analogy before. So, again, you can clean, you can paint, you can do all the beautiful things, but unless you put the fire out, it won't result in what you want.

Okay, so let's talk about Tesamorelin and actually why there's clinical evidence that stands out and what tesamorelin actually is. So, tesamorelin is a synthetic analog of growth hormone-releasing hormone. So, you heard me mention this previously in CJC above. So, GHRH, growth hormone-releasing hormone. It is structurally similar to GHRH, which your hypothalamus naturally produces. But with a chemical modification, it gives it a longer half-life and stronger pituitary activity, okay? So, when you inject Tesamorelin sub-Q, it travels to the pituitary and tells the gland to release growth hormone, which travels to the liver- we talked about this before- and triggers IGF-1 production, insulin-like growth factor type 1. The downstream cascade is identical to what your body did naturally at 25, just driven by a synthetic signal, okay? This is similar in mechanism to, like I said, CJC-IPA, which we covered in the last episode. Both are GHRH analogs.

Both increase endogenous growth hormone inside the body, and when you increase growth hormone, you increase IGF-1. But CJC-IPA and Tesamorelin differ in critical ways, including their clinical track records, which makes each appropriate for different patients. We're Okay, so how did it get approved? Tesamorelin has something almost no other peptide in this series has. It has full FDA approval for a finished pharmaceutical drug. Okay, so this is important. It was approved in 2010 under the brand name Egrifta. So, for the reduction of excess abdominal fat in adults with HIV-associated

lipodystrophy. The approval was based on Phase 3 clinical trial data that I want to walk you through because it is generally very impressive.

In the original New England Journal of Medicine trial published in 2007, 412 HIV-infected patients with abdominal fat accumulation were randomized to either tesamoran, 2 mg subcutaneously daily, or a placebo for 26 weeks. Visceral adipose tissue, measured by CT imaging, so actually measured it by CT scan, decreased significantly in the tesamoran group. The least-squares means were different from placebo; in one trial, it was about 19.6%, with trunk fat reductions around 18%. That's huge, like 20% of your visceral fat was reduced. IGF-1 levels increased by approximately 81%, triglycerides improved, the total cholesterol-to-HDL ratio improved, and self-assessed body image, of course, improved. In the 2014 JAMA-published trial (Journal of the American Medical Association), Tesamorelin 2 mg daily for 6 months reduced visceral adipose tissue by a mean of 34 cm2, compared with an 8 cm2 increase in the placebo group.

And the between-group difference was 42 cm2. That same study found a 40% relative reduction in liver fat in the Tesamorelin group, versus a 27% increase in the placebo group. This is a peptide where measurable hepatoprotective effects, so protection of the liver, happen, not just cosmetic outcomes. So, this is super important. So, all of those patients who have non-alcoholic fatty liver disease should be on Tesamorelin, right? If you have high visceral fat, you should be on Tesamorelin. Okay, so it's not just kind of cosmetic, like I said.

Critically, lean body mass increased; visceral fat decreased. In pooled trial data, approximately 1.42 kilograms- that's the definition of body recomposition. Most weight loss interventions take fat and muscle indiscriminately. So, we always worry about this. When patients are on GLP-1s, they'll lose fat, but they'll also lose muscle. Tesamorelin specifically directs fat loss to the visceral compartment while supporting lean tissue. Okay? So, what is the off-label application? Here's where it gets clinically relevant for the patients in front of me that I see every day because the FDA approved the

indication for HIV-associated lipodystrophy, lipomatosis, and dystrophy, reducing a relatively narrow population.

So, like I said at the beginning, don't get too excited because unless you have HIV, it's not FDA-indicated for you, okay? And because HIV patients - they have a lot of visceral fat, so that's why it was approved for them. Beth did not have HIV. Most of you listening do not have HIV, but the underlying biology that Tesamorelin targets doesn't change. Visceral fat accumulation, low growth hormone secretion, fatty liver, and cardiometabolic dysfunction is shared across many populations, including the perimenopausal and menopausal women that I see every day.

Okay. In specialties like functional medicine, Tesamorelin is increasingly used off-label for patients with significant visceral adiposity, fatty liver concerns, or escalating cardiometabolic risk that has not yet responded to lifestyle interventions. So, we do use it. This use is not FDA-approved. The dosing logic follows the same 2 mg daily framework studied in trials. Though some clinicians use lower doses in non-HIV patients, which I have done, the off-label use is increasingly mainstream. So, I see this used all the time. Surgeon volume for Tesamorelin in the United States grew approximately 49% in just six months from late 2025 to early 2026. Isn't that crazy? And as I said, I think peptides are having their day. And it's because they got taken off the list recently. Now they're going to face more scrutiny in July. We'll find out more about regulations. So, they're on the Category II list. They got taken off. And we're waiting to hear what happens on July 23rd and 24th. Okay, so it reflects how rapidly this conversation is shifting and how much benefit we're seeing from these peptides. And remember, peptides are just strings of amino acids put together, shorter strings. A 2026 systematic review, so that's this year, a meta-analysis of multiple randomized trials confirmed a visceral fat reduction effect across multiple studies with no significant reduction in subcutaneous adipose tissue or BMI.

So, this isn't for my patient who's trying to lose subcutaneous fat, right? They want smaller thighs. This is my patient who needs to lose visceral fat. And sometimes it's one of the same. I do have some patients who need to lose subcutaneous and visceral fat, and they might have cardiac risk. And so they're a person I might use it in, but I'm definitely going to use it in my patient who has high visceral fat, okay, and might not need to lose weight. That's skinny fat, right? So this is not a generic weight-loss drug. This is a targeted visceral fat intervention with the strongest peptide-level clinical evidence in its class. All right. So, picture the difference between a sniper and a shotgun. A shotgun, it's widespread, right?

It just pulls up everything. It hits a lot of targets. Some are the ones you want, and some are the ones you may not want, especially if I'm shooting it, right? But GLP-1 medications like semaglutide and tirzepatide, they're kind of like shotguns in this example, okay? They're very powerful. They produce dramatic weight loss, but they take fat from everywhere, including lean muscle you cannot afford to lose. All right.

They do not preferentially target visceral fat over subcutaneous fat. So we do see loss of both with GLP-1s. But Tesamorelin is like a sniper. It's like a precision tool. The data shows it goes specifically after the dangerous visceral fat while sparing or even building lean tissue. So, for a woman in midlife whose problem is metabolically dangerous because she has a fatty liver and central adiposity, not generalized obesity, the precision tool is often the better tool. So, that's really important. So, that's why I think this podcast series is relevant because you need to understand why certain peptides are used and how they benefit you in midlife, not just, hey, that's, you know, you should take this peptide.

And a lot of times you don't even know why. Okay. So, how does it compare to CJC IPA or CJC? Let's see. So, patients ask me that all the time. How is it different from CJC? Both are, like I said, GHRH analogs, and both increase growth hormone and IGF-1. But the honest answer is that they're biologically similar but have different clinical profiles. So, we use CJC-1295 when the goal is broad anti-aging. We're talking sleep, recovery,

body composition, repair. We use tesamorelin when the goal is specifically visceral fat reduction in a patient with metabolic concerns. So, Tesamorelin has stronger published data for visceral fat outcomes specifically, including, like I said, FDA approval-grade phase three trial data measuring CT-visualized visceral fat as a primary endpoint. If a patient comes in with best picture, stable weight, growing waist, fatty liver, rising inflammatory markers, Tesamorelin is usually the better match tool for her than CJC-Ipamorelin. So, because we're specifically treating that visceral fat. All right. Okay. So, what Tesamorelin actually changes and why the scale will lie to you. All right. So, let me walk you through what patients actually experience on Tesamorelin, because the pattern of change is unlike almost any other intervention in our toolkit. If you don't understand it going in, you will misread your results and get frustrated. Okay. The scale is not going to move on Tesamorelin. This is the most important thing I tell patients before they start. Body weight is not the right metric for Tesamorelin. Okay. So, keep that in mind because you're going to be highly disappointed if you think that's it. So, the most important thing is that the scale will barely move.

Okay. When patients start Tesamorelin, body weight isn't the right metric, like I said, for testosterone. In the registration trials, total body weight changes were modest. Some patients lost a few pounds. Some patients actually stayed the same. Some patients actually gained a pound or two because they, again, gained lean body mass, which is great, while losing visceral fat. So they had no net change on the scale but dramatic changes in their body composition, including visceral fat. Okay, and we can measure that on an InBody or you can measure it on a DEXA scan. If you weigh yourself daily and decide whether tessimoralin is working based on the bathroom scale, you'll conclude it's not working when, in fact, it may be working beautifully. We measure, like I said, with the in-body or DEXA scan. You can also measure with the CT or MRI, but I'm not going to do that.

It's pretty expensive. With waist circumference, you can also see that because visceral fat sometimes can show up there. And with metabolic markers. The scale is the worst

possible measurement on this protocol. So, throw it out during the Tesamorelin iteration, or at least be aware, because I think that's where a lot of practitioners forget to educate their patients. And then they get on Tesamorelin, and they're like, well, my body weight didn't change. This peptide doesn't work. But it could have been working great, reducing the visceral fat.

And so that's the number you want to focus on. Okay. So, think about this as recomposition to reveal. Imagine two suitcases on a luggage scale. Okay. So, you're getting ready to travel. They put your luggage up on the scale. One is full of dirty laundry. It's bulky, it's taking up space, and it weighs eight pounds, let's say, okay? The other one is full of folded, clean clothes, organized, taking up less space, but weighing approximately the same eight pounds.

The scale reads identically. The contents are completely different, right? The messy, dirty laundry, all bunched up. The clean, organized laundry in the second suitcase, taking up less space, okay? Tesamorelin swapping the dirty laundry of visceral fat for the folded clean clothes of lean tissue. All right. Same number on the scale. Totally different body. The bathroom scale was always going to lie to you on this protocol because it doesn't know what's inside the suitcase. So, hopefully you get that.

Okay. Waist circumference will decrease. And usually that happens about weeks four to eight. So, this is the only way we see it on the outside. So, this is usually the first visible change patients notice. Within the first month or two, waist circumference begins to drop, which is great. Not dramatically- typically half an inch to an inch per week, I'd say by week eight, and continued reduction over the full course. Pants fit differently. The belt moves down a notch or two.

The clothes in the closet that were getting tight start to fit again. Patients describe this as noticing they're losing inches without losing weight, which is exactly what we expect.

Around month three or six, visceral fat on imaging starts to change. So, if they were actually getting a DEXA to measure visceral fat or coming in for the InBody, the visceral fat reduction is what we really want to see and what trials measured. Roughly 15 to 18% reduction over six months. So, this is the deep change that drives everything else. Because remember, visceral fat is very inflammatory. You cannot see this in the mirror.

You cannot feel it day to day. But this is a metabolic reset. And it affects your total health in so many ways. This is what the protocol is built on: getting rid of that visceral fat. Liver markers improve. So, for patients with fatty liver, tessamorin has a measurable hepatoprotective, so liver-protective, effect. ALT often drops within the first 8 to 12 weeks. Liver fat is measured by MRI, but you're not going to get an MRI, remember. But they measured it.

Bone density, fat fraction, or ultrasound estimation typically improves over 3 to 6 months. The 40% relative reduction in liver fat measured in the JAMA trial (Journal of the American Medical Association) is a meaningful clinical outcome, particularly for patients heading toward non-alcoholic fatty liver disease. You know, it's huge. Okay. Another key change on Tesamorelin is that the lipid panel improves. So, triglycerides typically drop. The total cholesterol-to-HDL ratio improves. LDL particle number often improves.

These changes are modest but consistent across the trials and aligned with visceral fat reduction. So, that's what we expect. Because visceral fat is what was driving much of the lipid abnormality to begin with. And sometimes we forget this as providers because we're so focused on helping patients lose weight, and their numbers look better. But if their visceral fat is high and their cholesterol is still high, it doesn't matter what they're putting in their diet; they need to get rid of the visceral fat, right? So, it's not just the diet. Okay, inflammatory markers decline as well. The high-sensitivity CRP often comes down.

Inflammatory cytokines downstream of visceral fat decrease. The chronic low-grade inflammation visceral fat has been generating begins to resolve. And patients report feeling less heavy in a way they cannot describe. Because think about it. When inflammation goes down, your body just feels better. And that's because that systemic inflammatory burden is starting to decrease. I even hear this on GLPs, right? So, inflammation goes down. IGF-1 will increase when you're on Tesamorelin, and we have to manage it. This is the part that requires careful clinical management.

Tesamorelin is more potent than CJC, and I always say CJC IPA because they're usually used together, but it's more potent at driving IGF-1 elevation. So, in the registration trials, IGF-1 increased by approximately 81% in the treatment group. So, we monitor IGF-1 monthly initially, then quarterly. We want IGF-1 in the upper portion of the age-adjusted normal range, not above upper limits. IGF-1 trends above the range. We reduce the dose or pause the protocol. So, that's really important because remember, too much IGF-1 can cause insulin resistance, right? We don't want that.

Theoretically, risk of supraphysiological IGF-1, including potential effects on insulin sensitivity and a small association with certain cancers. So, that's why a little is good; more is not always better. I always say this to my patients, and we never want to cause a problem while we're fixing one. So, those are two important things to keep in mind as we monitor and as you go through protocols. I will tell you, so many patients are taking peptides they got off the internet, and they have no clue what they're doing. And these peptides are not without problems. Like, you have to know what you're doing.

You can't just be injecting things and thinking it's okay. Like, so I'm not a big fan of people just getting them, injecting them with no guidance, no medical oversight, and not knowing where they come from. So, I will never endorse that. Okay. Think of the GHRH signal in your midlife body as a thermostat that's gotten stuck in the off position. Okay. Your pituitary still has growth hormone in storage. The downstream system is still capable, but the upstream signal that tells it when to release is the dial that got stuck.

So, natural GHRH is a brief tap on the dial that wears off in minutes. All right. So, the natural GHRH we produce is a tap on the dial, but it wears off really quickly. Tesamorelin is a longer-acting version of the same tap that we would give it naturally. It presses the button and holds it long enough for the message to actually register. The system was always capable. So, you had everything there you needed. You just needed someone to lean on the dial a little bit. And that's what testosterone does. Okay.

All right. Let's talk about the metabolic cascade that resets when you go on testosterone. So, here's the bigger picture. The reason it all matters. Visceral fat does not exist in isolation. It is the upstream driver of insulin resistance, fatty liver non-alcoholic fatty liver disease, dyslipidemia high cholesterol, hypertension, and chronic inflammation. When you reduce visceral fat by 15 to 18%, you do not just improve one number. You reset the entire cascade. Insulin sensitivity improves.

The liver heals. Lipids normalize. Blood pressure comes down. Inflammatory markers go down quietly. This is what we mean when we talk about restoring metabolic health. That's really what we're doing. We're not just losing visceral fat, right? They're not the same thing. So, losing weight is great. semaglutide and tirzepatide. Great, great, great, right? Tesamorelin, we're looking at visceral fat because visceral fat is the worst type of fat in our body. Okay, so imagine your metabolic health is a retirement account, right? Every day you have visceral fat dumping inflammatory cytokines into your bloodstream, all right? You are making a small withdrawal every time that happens.

Most people have been making these withdrawals for 20, maybe 30 years without even realizing it. Okay, that's all that visceral fat, right? By midlife, the account is depleted. The next 20 years of your life depend on what you do with that account starting right now. So, Tess and Moreland doesn't just stop the withdrawals. It begins making deposits back into the account. Your future self in your 70s, your 80s, your 60s even, is the one

whose health depends on whether you reverse this trend in the years you're in right now. So, that's what I'm talking about. This is a metabolic intervention with a long-arc payoff, right? Just like our 401k. We put into it, we put into it, put into it for that long payoff when we retire. So every time you've been taken out of your retirement account, you need something to put back in so we can restore it, right? So, how do we use Tesamorelin in practice? Again, let me get specific about how I prescribe it because this is the peptide that requires the most careful clinical management of the ones we've covered. But again, this is not me giving you a protocol. So, please know that. This is medical information. It's just information for teaching. It's not for diagnosis or treatment. All right?

So, Tesamorelin is administered as a subcutaneous injection. There's no oral form. The molecule is too fragile to survive the digestive tract, just like CJC IPA. Patients self-administer at home using a small insulin syringe, similar to the other peptide protocols we've discussed. Site rotation matters - abdomen, thigh, upper arm - to prevent local lipohypertrophy. So, at injection sites, we don't want hypertrophy of fat in your injection site, okay? Timing. Tesamorelin is typically dosed once daily because it primarily works through driving overnight pituitary signaling, just like CJC. Evening dosing is most common, typically 30 to 60 minutes before bed on a relatively empty stomach.

Some clinicians prefer morning dosing for patients who have insomnia, since growth hormone elevation can occasionally affect sleep onset in sensitive patients. Okay, because remember, it elevates IGF-1 by 80%, so much more than CJC IPA. But it's great for sleep, right? CJC IPA is. But a five-on, two-off cycle pattern is common in most protocols. So, we usually say five nights on, take the weekends off. Okay. Though the registration trials did use continuous daily dosing for 26 to 52 weeks, the right cadence depends on the patient and the goals, and I would say you look to your physician or your provider to guide you- someone who has peptide training.

The FDA-approved dosing is 2 mg subcutaneously daily. Many functional medicine clinicians use lower doses for off-label visceral fat reduction in non-HIV patients, sometimes starting as low as 1 mg daily and titrating based on the response in IGF-1. As always, I won't give specific dosing on a podcast. Dosing requires clinical context, patient size, IGF-1 response, glucose response, and other variables, so please keep that in mind. Most patients run an initial 12- to 26-week course to evaluate the peptide's true response. Some patients can continue beyond with appropriate monitoring.

So, remember, the trials were 26 to 52 weeks, right? so up to a year. A critical point patients need to understand: the visceral fat reduction does not persist after stopping. In the registration trials, patients who discontinued Tesamorelin gradually reaccumulated visceral fat over the following months. This is not a cure-and-cure intervention, so that's important to know. It's a treatment for a chronic condition, and like most chronic condition treatments, the benefits last while you're using it. So, lifestyle changes layered alongside the peptide create sustainable change. So, I don't keep patients on this forever because I'm teaching them lifestyle changes alongside it. And if they are making lifestyle changes, their visceral fat is going to go down anyway. And so they're not going to stay on it long term. All right. So, we try, we do have some patients on some of the weight loss medications on a maintenance dose, but I'd say for my Tesamorelin patients, I really don't do maintenance. I don't do that that often. So, Tesamorelin requires more careful monitoring than other GHRH peptides, as we discussed. Baseline labs should include IGF-1, fasting glucose, hemoglobin A1c, fasting insulin, and lipid panel. Also, those liver markers, so ALT and AST, and your inflammatory marker, high-sensitivity CRP, and I even do a thyroid panel. We also typically obtain a baseline, and I wouldn't say we get a DEXA. I would say we always get a baseline in-body study because it measures visceral fat, and we have great machines that do that.

If you don't have that available, you could use a DEXA scan to measure what you need to know, but we don't do that. During treatment, we monitor IGF-1 at week 4 and week

8, and quarterly thereafter. We monitor fasting glucose, hemoglobin, and A1c because Tesamorelin can transiently affect insulin sensitivity, particularly in the first few weeks. So, that's what happens most often. We also monitor ALT and AST to track hepatic improvement. We adjust the dose based on the data, not the patient's enthusiasm or scale changes, right? So, we use data to guide us. Foundations first. You'll hear me say this in every peptide podcast. Tesamorelin is more dependent on lifestyle foundations than almost any other peptide we use.

Because the same lifestyle factors that drive visceral fat in the first place will continue to drive it through treatment if not addressed. So, adequate protein: one gram per pound of ideal body weight. Strength training, because lean body mass increases only if there's mechanical input telling the body what to build. Resolution of insulin-driven dietary patterns. So, typically lower carbohydrate diets, no simple carbs, whole foods with attention to glycemic control. CGM monitor. Alcohol restriction. So, I recommend elimination during the active treatment course. Because alcohol is one of the most potent visceral fat drivers and is a direct hepatotoxin. This is why you get liver disease with alcohol, okay?

Synergies. So, what can we pair with it? So, testosterone pairs powerfully with several other interventions. Strength training, protein optimization, and body composition outcomes improved dramatically, along with BPC-157 or Larazotide for patients who also have gut barrier issues. So, they have to have gut issues. The systemic inflammatory background improves more completely when you also treat the gut. All right? With bioidentical hormone therapy in perimenopausal women and menopause, the underlying driver of central fat redistribution is addressed at the source.

So, I know when I put patients on estrogen replacement, their fat distribution is going to change. That's why they lose belly fat. Okay? CJC-IPA, I don't know. The picture is a little bit more nuanced. We generally don't stack CJC-IPA. We generally don't stack two GHRH analogs simultaneously, so I wouldn't recommend that because the IGF-1

response can be excessive. And also, too, you want to get the best bang for your buck, so why waste if you don't need to go that high, okay?

Who should not use it? Contraindications matter on this peptide, just like with all the other peptides. Active malignancy is an absolute contraindication. Pregnancy and breastfeeding are contraindications. Active diabetic ketoacidosis or severely uncontrolled diabetes. We don't want to use it. Active proliferative diabetic retinopathy, so when there are changes to their vision, and also pituitary or hypothalamic disease. So, again, this is why you need a provider or physician who is overseeing your care. We use caution in patients with a history of certain cancers, particularly hormone-driven ones, just like CJC IPA talked about this. We also use caution in patients with significant insulin resistance because Tesamorelin can transiently worsen glucose tolerance early in treatment. That may require dosing adjustments or short-term glucose management strategies. So we want to stay on top of it, okay? As with every peptide we have covered, this should be prescribed, like I said, by a provider or physician, sourced from a licensed compounding pharmacy or pharmaceutical manufacturer, and monitored over the course of treatment.

The chain of command is just as important as the peptide and just as important as the sequence in which you would use it, okay? So, here's exactly what we did for Beth. And again, I always say sequence matters. First, comprehensive testing. Beyond the lab, she had already had to run. We did an in-body scan to measure her visceral fat, plus a fasting insulin. We can calculate the HOMA-IR; we also ran an advanced lipid panel, including APOB and LDL particle numbers, and a comprehensive thyroid panel and full sex hormone panel, including estradiol, progesterone, free testosterone, total testosterone, DHEA, sex hormone-binding globulin, and vitamin D3. So, we always get a really complete panel for our patients. Beth's baseline visceral fat was elevated.

Her fasting insulin was 14, confirming early insulin resistance. Her estradiol was consistent with early menopause. And her DHEA was below the reference range. Second, her

protein intake was again 110 grams a day. This has required a significant shift because she's been eating about 50 grams. And I know it's hard to get the protein in. I struggle with it myself. A lower glycemic dietary pattern with attention to protein first at all meals and fiber. Two strength training sessions weekly. Alcohol elimination for the duration of the protocol.

And a strict 9.30 p.m. window. Okay? So, it's really important that she gets to bed. All right? Vitamin D supplementation. Magnesium glycinate at night. And we also began bioidentical progesterone and topical estrogen to address her underlying hormonal drivers. So, that's really important too. Okay. Vitamin D, I said, magnesium. We also be in the hormones. And then her targeted protocol.

After six weeks of foundational work, we started Tesamorelin at 1 mg subcutaneously every evening. We monitored IGF-1 at week four and it moved from 102 to 174, within the upper range of normal for her age. Fasting glucose was stable. We continued the protocol. At 12 weeks, we added BPC-157 by injection at first, but then we switched her to oral because it was her gut, and that's the most important thing we wanted to treat. And so I feel like oral is better for the gut, and we wanted to see how her liver picture was. Okay. At six months, this is where Beth was. Her waist circumference has dropped. So, she dropped from 32 to 29.5. Her DEXA scan showed- not DEXA; you can do a DEXA, but she had an InBody- a 70% reduction in visceral fat. Her ALT had normalized to 22. A repeat liver ultrasound showed resolution of her non-alcoholic fatty liver disease.

So, she had gotten down. Her high-sensitivity CRP had dropped from 4.8 to 1.4, and her fasting insulin was 8. Her hemoglobin A1C was 5.3, and her triglycerides were only 88. Her weight was 137, essentially unchanged. But her body, by every metric that actually matters in this case, had been completely transformed. So, the bathroom scale, as I said, if you're using that to measure progress, you're measuring the wrong thing, all right? This is what tessomorlin does in the right patient, the right protocol, with the right

foundations in place, and you monitor closely, all right? It's not magic, just like I said about the other peptides.

It's precision biology applied at the most appropriate time for the most dangerous fat in the body. And it has measurable results that show up in imaging and in blood work, even when the change. So, that's so important. Okay. What can you do this week before you see a physician or provider if you want to work on your visceral fat? So, measure your waist circumference. Use a soft tape measure at the level of your navel at the end of a normal exhale. So, don't hold it in. Write the number down. For women, a circumference above 32 inches is associated with elevated metabolic risk. Above 35 inches is high risk. The scale tells you almost nothing useful about visceral fat. So, use the tape measure to tell you a lot. Okay. Stop weighing yourself daily. The scale is unreliable, and it changes. I mean, depending on what we eat, how much sodium, water, and if we slept.

So, I think once weekly is plenty. If you want to weigh yourself and tracking your waist circumference monthly tells you more than weighing yourself every day. So, that's the protocol I like to go with my patients. Eliminate or dramatically reduce alcohol for 60 days. Alcohol is one of the most potent drivers of visceral fat accumulation and a direct hepatotoxin. This single change alone can produce measurable visceral fat reduction in patients with no other intervention, especially if you drink alcohol on the regular. And I know this is a big ask, I know, but this is life-changing if you can do it. There's a great book by Annie Grace called The Naked Mind. Anyone who struggles with alcohol or is thinking about cutting back on alcohol or wants to get off it completely: great book to read. So, Annie Grace, The Naked Mind. Okay. Front-load protein at breakfast, 30 grams minimum. If you can get up to 40, even better. Protein in the morning improves satiety, supports lean muscle mass, and reduces the carbohydrate cravings that drive insulin spikes throughout the day. Ask your physician or your provider for an advanced metabolic workup that goes beyond the basic panel. So, for fasting insulin, ask for a HOMA, H-O-M-A-I-R. They can even calculate it; an advanced lipid panel with APOB,

lipoprotein A, particle numbers. And if you want to go back to episode five, I think it was where I talked about all the reasons that you should know your cardiovascular risk by knowing certain numbers. Ask for a liver panel so they can get a CMP that gives you ALT and AST. You also want high-sensitivity CRP, so HSCRP, and have a way to measure your visceral fat. The standard panel isn't enough to really tell you what's going on, especially in midlife. You have to ask for the right test, all right? If you recognize it in yourself, if the weight's been stable for years, but your clothes are fitting tighter, especially around the belly, now you know why, okay? The fat that has accumulated around your organs is real; it is measurable, and it's the fat that matters most in the next 30 years of your life if you're 40 years old, okay? The medical system has trained all of us to look at the bathroom scale as a primary measure of body composition. I even hate when BMI is used because that's just height over weight, or it's height and weight.

And if you have a lot of muscle, your BMI will be high. So it misses the signal that your dress size and your tape measure have been quietly carrying. So use your tape measure. Remember, above 32 inches is associated with elevated metabolic risk; above 35 inches is high risk, okay? Tesamorelin will not give you the body of a 25-year-old. No peptide will do that. You have to do work, right, to reach that. But in the right patient, with the right protocol and the right foundations in place, it will reverse dangerous fat accumulation in midlife and protect the metabolic future of your 60s, 70s, and 80s, okay?

It is the only FDA-approved peptide in our visceral fat toolkit. It's amazing. The phase three trial data is really strong. And in midlife, women with stable weight, growing waistlines and fatty liver, or escalating cardiometabolic risk, it's often, like I said, it's like a sniper. It's precision that turns a slow-moving disaster into the correct trajectory. So, it gets it out of that path and onto the right path. All right? In our next episode, we're going to talk about TB. You'll hear it called TB500 or thymosin beta 4 is also how I learned it.

The recovery and connective tissue peptide that pairs really nicely with BPC-157 for serious musculoskeletal repair. Also, with your gut sealed, your tissue repaired, your immune system back on track, I've gone through three of the peptides; if you can associate them, your master signaling restored, and your visceral fat reduced, the next layer is targeted regeneration, and that's what TB-500, or Thymosin Beta-4, focuses on. So, that will be in our next episode. If today's episode resonated with you, share it. Share it with a woman whose waist has been quietly growing larger, and she's complaining about it because you now know why, right?

And it's so much more than just visceral fat. So, share it with her so she can learn what to do to help her feel her best in her 60s, 70s, 80s, but also to decrease her metabolic risk, which is cardiovascular, it's liver, it's so many different things, okay? Thanks for listening. I'm Dr. Tracy Page at Wisconsin Institute of Functional Medicine. This is Midlife Clarity, and we're so grateful that you're here. We hope you share this information and help every woman you know and every man be healthy and well, live vibrantly, and have the best life possible inside and out. Thanks for listening.

Talk to you soon. Bye.