The Genetics of GLP-1s: Why Two People on the Same Dose Get Different Results Transcript

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Intro and GLP-1 Genetics Overview

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And here's what I will tell. For me, this was so true because I used to like to have a glass of wine or two in the evenings. Once I use the GLp-I, I completely lost that craving for it.

I'm like, "I don't even want to touch it." I don"t want it, so it's really interesting what genetics could tell and how beneficial it can be above and beyond weight loss.

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Further, information about specific products or treatments within this Podcast are not to diagnose, treat, cure, or prevent disease. Hey guys, how are you?

Again, I have Dr. Piper Gibson. Dr Gibson, thanks for joining us again today. Thank you for having me. You're welcome. So guys we're doing this series on the Aimprint.

And if you guys are following us since the first episode, this is our fourth or fifth episode. We saved I think one of the best things for last on this.

And that is the genetics of weight loss medications, right? Because how awesome would it be if I told you Piper that I could wave a magic wand and say, you're going to do good on a GLP if you are not?

Well, I am waging here is the magic one because that is The Aimprint, right? And it's so neat because I'm seeing it even in practice, because we have obviously a very large weight loss practice here in Pittsburgh.

And we see our patients on GLP, some do magnificent and some don't do well. No matter how much elaborate blood work we order, we saw that, wow, there's something else and that something is definitely genetics.

That's what we're going to get into today, aren't we? We are, and I will tell you, I'm so excited. I think this is an amazing, juicy topic. And it's something I usually talk about in my own practice.

So to be able to sit down and nerd out with you on this, i'm, so, excited, absolutely. She's not BSing us because I got two emails on a phone call of how excited she is today.

By the way, it makes me feel excited that you're excited because to me, Today's video is one of the coolest topics we'll go over in all seriousness, guys.

It's going to be very, very educational. And again, Piper, thanks for coming on. So I want to get right into it. We're going talk about a GLP receptor sensitivity medication selection.

You know, because you have all these things, somaglutide, terzepatide and all this, right? So first and foremost, What does the GLP1R tell us about medication response?

So, by the way, guys, that GLp1r is the gene that we test on the imprint, right? And Piper, you know this. You were very instrumental in helping us develop it.

So yes, we can test how a GL p drug will do on you. Again, what's it tell about us, please?

GLP-1 Receptor Sensitivity and Drug Choice

So as we start to look at GLP1R, GLp1r encodes the receptor for a GL p1. So medications like semiglutide and what they actually bind to. If that receptor is less functional genetically, the medication has less to work with.

So we looked at a couple of your patient's reports. And so if you have a patient who has both of the genes that you're looking at in the green, it indicates normal receptor sensitivity for blood sugar reduction and body weight response, respectively.

So before investing in expensive medications with significant side effects, knowing those receptor sensitivities is really clinically responsible because the patient is genetically primed to respond, which changes the cost benefit conversation entirely.

It truly does. And you know this just by being a practitioner, how many people are buying these weight loss medications online, right? And it's really a crap shoot.

Like, hey, one, is it going to work? Right? Two, should I be on semiglutide or terzepatide? Which one would work best? I know that's my next question I'm going ask you, but The genetic testing and name print right there in that paragraph, Dr.

Gibson, you would agree is, that's paramount. Everyone should have it done to test this darn gene to see how well you do. So like you said, instead of spending and investing all this time and money on a medication that may not work for you, get the damn test done.

Right. And not only that, your test is going to tell you so much more above and beyond just your response to a GLP-1. This is where you start to build that roadmap.

Absolutely. So again, guys, as we go through this today, we're going to break down GLP ones first, and then we'll get into some other things. Let's go on that question I was going ask you.

Does it matter genetically which GL P1 medication someone is on? Yeah, so this to me is one of the least discussed and most important questions in clinical practice right now.

So terzepatide hits two receptors, a GLP1R and a GIPR, the receptor for a glp, which is a completely separate gut hormone. So GIPR genetic variants determine how well someone responds to that second mechanism.

So a patient with a strong GLP1R sensitivity but a poor G. I. P. R signaling will respond differently to terzepotide than to semiglutide. And the genetics can actually inform which drug is the better first choice.

Right now, most prescribers are choosing medications based on availability and insurance. But the genetics, in my opinion, should really be driving that decision because practitioners who understand this are going to have so much more meaningful and better outcomes.

And really, that is the edge that the imprint creates. And a grin, I agree. And I see that just with our practitioners in recommending the right GLP for the person, when our patients have that test done from the imprint, and we follow up on them, obviously, right?

Because they're working with health coaches, they are working on us, the results are pretty profound. Pretty neat stuff. I want to get into a couple of the other genes on the test we have.

So, especially the hunger signaling genes, right? So if Sayadee genes are blunt, do GLP-1 drugs really even work then? Yeah, and I think this is a great question.

So GLP-1 medications enhance satiety signals. However, those signals still need functional downstream receptors to land. For example, one of the reports we looked at with one your patients was they had a leptin receptor and an HTR2C both in the red.

So, meaning that the downstream reception for these drugs is compromised.

Satiety Genes and Hunger Signaling

So the medication may produce less perceived fullness than expected in this patient, explaining why some people on GLP-1s actually describe still feeling hungry even when they're on a therapeutic dose.

So what's the fix? The fixes with this person, we would layer in high protein, high fiber and structured meal timing to provide independent satiety input while the medication works on the appetite hormone side.

So the genetics are telling you exactly what to stack. Yeah, exactly. And you know, having all of our health coaches, our certified health, coaches and working with our patients, because again, Piper, this is another issue we're seeing in the United States.

People just taking this medication and just thinking it's a cure all end all. They're not working what their food, they have no idea what they're should be eating, right?

Right. So working a certified practitioner and helping you through the food components, because it's still daunting to me how some people still to this day don't know what a protein is.

Don't what carbohydrate is, right? But education is paramount with these people. Dopamine, the reward eating and addiction wild card. You know and that's a title that i that we had for this next series of questioning but, you know one of the big jeans that you and i really get geeked out on as we say is that calm variant right and how it interacts with glp so the c o m t variance how did they interact with the g l p medications.

Yeah, this one for me was huge because this is me in a nutshell and how I felt with a GLP-1 and it is because of my COMT function. So GLp-2 medications are thought to reduce food reward signals in the brain, but not just suppress hunger, but dampen the drive to seek out pleasurable foods.

So this is part of why some patients lose interest in things like alcohol or highly palatable foods on these medications, which is very, very true for me.

So for a patient with intermediate COMT clearance, dopamine lingers. They're slightly longer than normal. It keeps the food reward signals a little bit elevated.

The GLP-1 medication may be partially counteracting this, Which could explain why COMt intermediate patients are often particularly good responders. However, when we start to look at some of the other factors, maybe your dopamine receptor or your DDC genes, is we can see if you're going to be predisposed to binge eating or overeating.

And so for this person that we were looking at, both of their genes for binge-eating and overeat came back normal, which is really a favorable finding, and it really makes that dopamine component here really manageable.

Yeah. And just like Dr. Gibson said, guys, you know, it's really amazing when you're asking patients, Hey, do you have any alcohol cravings now? And by the way, I have full transparency.

I, i have tried GLP drugs as well and medications and they've worked wonderfully. and it is funny. Sometimes doc, my craving really didn't go away. But let's say if I had a bite of pizza, I was good with the one.

It's like I really never had to have the two, the three, or the four other pieces. You know what I mean? But that comp gene makes sense now since I have my own imprint done on myself.

Reward Pathways, Alcohol Cravings, and COMT

Why do some patients on GLP suddenly stop drinking or lose interest in some of these behaviors, right? Is it genetic? is that something we can tell them on the imprint?

So when we start to take a look at it, this is one of the most fascinating emerging areas for GLP-1 medications because they appear to dampen the general reward response.

So not just to food, but to alcohol, nicotine, compulsive behaviors. The mechanism runs through the same dopamine pathways that drive that food reward.

So when we're looking at something like the AimPrint and we are looking COMT and DRD2 and ANKK1 variants, they start to determine that dopamine receptor density and clearance rate and predicts who gets cross-addiction benefit most strongly.

So patients with, like we said, that intermediate COmT who already have that elevated dopamine tone are the ones most likely to notice those reduced alcohol cravings and side effects.

It's just so fascinating to me. I'm sorry to interrupt. It's just literally mind-blowing how this works genetically. I love it. Yeah. So this matters because it changes the informed consent conversation you're having.

A patient with active alcohol use or compulsive eating patterns may get far more clinical benefit from a GLP-1 than just the weight alone. Their genetics can really tell you in advance and here's what I will tell you, for me this was so true because I used to like to have a glass of wine or two in the evenings and once I use the GLP-1 I completely lost that craving for it.

I'm like I don't even want to touch it, I dont want it so it's really interesting what genetics can tell and how beneficial it can be above and beyond weight loss.

A hundred percent. I mean, if we're reading the current literature and even just the news right now, a lot of big pharmas looking how to have these GLPs utilized for people addicted to alcohol and other things.

And again, I think you have the best population of people right. Now just ask them of, Hey, how much have your cravings declined? And they really have, you know, even myself when I was on them, I maybe, like you said, have a drink or two.

I was like, no, I'm good. You know, you just didn't want it. And moving forward, Like you've said it's getting away from just weight loss, even a body by aim, it, its longevity, right?

It's how can this aim print be so significant to help me on other facets of life? Again, other than just wait. But today, again, I really am glad we're focusing on these very sensitive genes and specific genes talking about the medication.

I want to talk about insulin resistant genes because I definitely have some insulin resistance, unfortunately. Which genes most clearly predict insulin-resistance on this panel?

Yeah, and you know, I think this is something that's so critical for so many people in the world right now is this insulin resistance effect. And so everybody that is listening to this podcast, yes, we are giving out some names for genes and they're usually like little code words, but Darren can guide you through all of that.

So when we're looking at insulin-resistance genes, TCF7L2 and CDKN2A are two genes that really lend to either a representation of elevated type 2 diabetes genetic risk or not.

And so when we're looking at, you know, a certain report, we can see that this individual actually has this elevated risk. But you also want to layer that with mitochondrial findings because mitochondria, right?

They're the energy in your cell, they're your workhorse. So you look at several variants. You look UCP variants and a TFAM variant. These people have a genetic profile where Energy can be stored more readily, and we can look at mitochondrial efficiency.

So the urgency here is really real. It isn't lifestyle optimization conversations. it's really about metabolic disease prevention and genetics. And we could look these genes and say, see how well people are going to use and burn energy, or see well how people will handle that insulin resistance.

Above and beyond the GLP-1, this panel does incredible things. Now, I appreciate you saying that because again, just even my passion, my family history, but even with our patent pending and our pattern hopefully being approved, coming out with and treating diabetics with the AIM method of how to circumvent some of these things of being insulin resistant.

You know, one of the items that happened the other day, and I know this is going to be our next topic to talk about.

Insulin Resistance and Metabolic Risk Genes

I want to get into this next is, The whole skinny fat skinny looking emaciated version of that person right like you know it's coming now to a point where we're saying oh that personal must be on the shot right.

You know even yourself you may be guilty of saying how they're on it you now. The muscle loss problem that no one is talking about is what I want to get into next.

Why do some people come off a GLP looking so depleted instead of lean? Yes, and this, since day one, has been something that I have said about GLP-1s.

And so muscle is so critically important for everybody, especially women over 40, muscle. But GLp-one medications don't selectively burn fat. They're not like, oh, you have a big butt, we're going to burn right here.

Uh-uh, not how it happens. So, you know what? Say that whole thing, Piper, again, because it's that important. GLP medications don't what again? GLp-1 medications.

Don't selectively burn fat. You got it. That's right. People don' t get it, people don t understand that. Yeah. They don''t get. It''s going to, burn the fat off your face, it'''s gonna burn.

The fat of your legs and your arms, and it' s not gonna selectly burn it off. Your booty, where you probably have a good bunch of it right? Yeah. So research shows a significant portion of weight loss on a GLP-1 is lean mass.

That means your muscle, everybody. And that percentage gets worse without intentional intervention. going on, lifting weights, building muscle, eating protein.

Variants in the TRHR, which is a thyroid hormone receptor, and your IGF2, an insulin-like growth factor, on the imprint directly govern how well someone preserves muscle during caloric deficit.

So people with red variants here mean a genetic predisposition toward muscle catabolism when eating less, so your body basically eating up your own muscle.

And what the clinical implication really means is that muscle preservation risk has to drive protein targets and resistance training recommendations before the medication even starts.

This is a conversation most prescribers are not having, and it's why some people look worse at their goal weight than they did when they were heavier.

They lost all of their lean muscle body weight. I really have to be honest with you. I don't know any practitioners in my area or anyone I've even spoken to of any practitioner is talking about this stuff, right?

Everyone is so focused on that goal of losing weight, but no one knows what's going to happen and what that person is going look like after. You know, we mandate some of our patients being on at least a creatine, a collagen, something where protein is going to be readily available to atleast build protein, right?

Because, like you said, the number one thing, Piper, for females, especially over the age of 50, We talk about bone loss. We talked about muscle loss, and then you have that whole downward spiral.

of osteopenia, breaking the hip and the story, right? So I'm all for these medications done properly, but the nutritional aspects have to be there too, you know?

Muscle Loss and Nutrient Depletion on GLP-1s

100%. So the side effect of certain things, meaning, and you and I just talked about this before we went on air. Who gets nauseated? Who doesn't? who's going to have diarrhea.

Who's gonna be constipated, right? So can genetics honestly predict who is going have miserable experiences on GLPs? Yeah, nausea, vomiting, and gastroparesis risk with GLP-1 medications.

It really isn't random. So that GLp-2 receptor that we talked about in the beginning, these receptors are expressed heavily in your gut and the vagus nerve.

it governs that gastric emptying. Your gut sends about 10 signals to your brain and your brains send about one signal to you gut. So your gut is extremely bossy.

In variants that affect vagal tone and GI motility pathways influence how severe GI response will be to these. We talked about COMT previously, but COMT variants are relevant here too.

So slower catecholamine clearance affects gut motility directly. Intermediate or slow COMTs patients may have more pronounced GI side effects because the signaling pathway stays activated longer.

Knowing this in advance changes the titration conversation, so how much you're going to increase over a period of time, right? It's not a reason to avoid the medication.

It is actually a reasons to start lower, go slower, and have a specific plan. Most patients My mom being one of them who abandoned GLP ones in the first 30 days because of the GI side effects.

And there's really a gentler way to approach it when we know your genetics. Yeah, 100%. And like you said, even with us, Piper, like your mother, the number one reason people quit these GI GLPs is because GI issues, right?

Or some sort of something. I just didn't feel well. So again, guys, I'm going to continually saying this on this episode. Before you start spending money on GLP's you have the imprint on and know what you're up against and like Piper said, there's so many different genomes we're talking about.

COMT, TKRM, you know, it sounds like a bunch of code words, right? but that's why there's practitioners like us to go over it and talk about it. Piper, a lot of people don't know we already have GLP sensors in our body, right?

You know, the GLPs sensor only last five or 10 seconds, but we have it, so your gut's already making GLp. Can we optimize it? Can someone actually boost their body's own GL p production?

Yeah, the human body is the coolest. The gut, like you said, produces GLP-1 naturally from the L cells in your small intestine. And those are triggered by fiber, protein, and fermented foods.

So, FUT2 secretor status matters. Let me just tell you real quick what F-U-T-2 is for people that are listening. FUT2, is your fucosal sugars in you gut.

You make these little sticky fuchosals sugars, in the gut that actually the probiotics that bacteria like to stick to and then proliferate, you want them to grow.

If you have an FUT2 variant that kind of you don't secrete these sugars as well, things like probiotic, your microbiome really may have a less favorable environment.

So, F-U-T-2 secretor status matters and that shows up in that gut microbiom work we're doing. It directly influences the microbial environment that drives this production of GLP-1s.

So non-secretors of FUT2 variants have a less diverse microbiome and produce less short-chain fatty acids that then in turn stimulate natural GLP-1 release.

They are starting from this compromised baseline before the medication even enters the picture. And really that practical application would be FUT2 variants informed targeted probiotic and prebiotic strategies that can meaningfully boost endogenous GLP1.

It's part of why two people on the same dose have totally different outcomes and why the full genomic picture matters before you even write the prescription.

Yeah, and you know, it's funny, even in the same family, I see people reacting differently, right? Sisters, mother, daughters, still acting differently.

Right? And that genetic testing, the genome looking at the FUT2 genes is pretty profound. It's so fascinating and the science behind it right now is just, like I said, it's just so exciting.

I'm just excited about it. You know, there's times in medicine where you're on the forefront of certain things and there are times where we're really seeing, not categories, I see people falling into different categories and it is really neat guiding them on different aspects of this.

You know, even one of the things, as you know and it leads me to this, is a lot of what we do here is even with our custom IVs, right? Remember, when I have all this blood work, Piper, and I this gene testing, our IV's are able to be made custom for you when you're in the office.

So, are there nutrient deficiencies practitioners should really be worried about on long-term GLPs? Like, do you have a top two or three that we should talk about?

Yeah, I do. And I think this is another great conversation because it is one that is not being had. GLP-1 medications, yeah, they reduce food intake, which sounds obvious, but the nutrients lost aren't distributed evenly.

So reduced protein intake accelerates muscle loss. We went back to that TRHR and that IGF2 that we looked at. But reduced animal product intake is going to deplete B12.

And so if you have someone who has a TCN2 variant that is compromised, they are going have issues with B-12 transport. So you're already getting less B 12 and they already have an issue transporting this B twelve.

We also have to look at vitamin D production and how it falls because less dietary fat means less absorption of fat soluble vitamins. I don't know if anyone's ever told you, but if you're going to take a fat-soluble vitamin, take it with something that has fat in it.

So when we start to looks at this and we look the imprint, you are looking at three different variants in the vitamin-D pathway, which can be critical to know, hey, is this person already in a hole before we started this medication?

So the genetic panel isn't just telling you what to supplement. It's telling which depletions will happen faster and hit harder in a specific person on this specific medication.

GI Side Effects and Titration Strategy

This to me is really proactive care. That's what prevents people from feeling great for six months and then just completely crashing. I agree. Not only crashing, it's how about gaining weight after they get off these things, right?

Yeah. I mean, I will tell you this much. Hi, am seeing not our patients, but patients coming from other clinics coming in who had no nutritional guidance, who just took a shot.

They didn't talk about nutrition, supplementation, anything like that. Is that rebound effect? And I'm going to tell, you guys, holy moly, is that a real thing?

I think the number one fear with GLPs is regaining all the weight. is that fear genetic you think yeah you know some of that really can be so when the medication stops the biological landscape it was masking snaps right back into shape if you have an fto or a leptin receptor or an htr2 that are in the red the drug compensates for those variants.

It's not correcting them. You still have those variance. They still are doing the same things in your body. The drug is just kind of masking that. So people, for example, who have the heaviest satiety burden, you know, stacked across like the leptin receptor and the HTR2C, they have this steepest rebound risk because they were the most dependent on the medication to feel full at all.

This is something families and patients deserve to know before they start. The argument is for building the full protocol during the medication phase, not after.

So you use the appetite suppression window to rewire the eating patterns, establish that circadian structure, and address those nutrient depletions. The genetics are going to tell you what needs to be in place before anyone considers tapering them off.

These are things that you need to know. I don't think there's enough push for patients to know or education wise that letting people know, Hey, when you go up on some of these higher doses and you stop cold Turkey, you're going to have a problem, right?

You're. Going to gain weight back. And it's almost like, You know when people were on certain SSRIs and certain doses on you and I both know this. If you do not just tie trade down and you just completely take yourself off of or stop, there's going to be hell to pay.

Right? Yeah. Yeah, you're not going feel so hot. No, not at all. And, and it's just not only that it I've seen people have gained weight back like I'd never seen before when they're no tie trading down correctly.

All right. Lastly, the whole body by aim, that the entire way we built this company is customization and accountability. When we talk about stacking genetics with medication and coaching, I believe that's, That's the win-win here body buy aim.

So I want to hear it from you, Dr. Gibson. Why do some people lose 40 pounds on this?

Boosting Natural GLP-1 and Gut Health

stuff and some people lose eight. Yeah. And wouldn't you like to know this before you go into taking this medication and you take it for months and then you're like, I only lost eight pounds, like I would love to notice prior to that.

So a 40 pound responder typically have good receptors sensitivity to these drugs, which the person that we were looking at really does. They have manageable satiety genetics.

And you have this clinical team that personalizes the surrounding protocols. So we know they have the good genes going into it, and you the genes to build that roadmap and that plan.

But when we look at someone like an eight pound responder, they are often fighting their genetics without knowing it. Maybe they have late chronotype circadian disruption, which can undermine medication benefits and metabolic benefits.

So, you know, what is their eating schedule like? What is there sleep schedule? Like it can be blunted and those satiety receptors that were supposed to reduce fullness, maybe they don't mitochondrial inefficiency and limiting how much energy they can actually burn.

Right? So when we look at the medication, you can't just look it as a standalone. The medication is one lever on a 10 lever system. Pulling only one level while the others work against you explains the wildly variable outcomes that we're seeing and why data on GLP ones is so inconsistent in the literature.

Yeah. And it truly, truly truly is, what does a truly personalized protocol look to you? What's it look like to Yeah, that's really how I've built my business, too, is this personalization, this individualization.

And so when we are looking at specific patients, GLP-1 medications where they have a good receptor sensitivity, you can assist them with a low-carb diet structure when you start to look at their FTO genes or their UCP genes.

We know that maybe they need more morning light or a consistent sleep schedule maintenance. They need targeted supplementation based on their genes. For example, we talked about the D3 and the methylated B12.

And maybe, they needs some mitochondrial support.

Rebound Weight Gain and Long-Term Planning

So, you know, looking at that, it's not just one thing. It is multiple factors built around, hey, know these genetic results. That aim print gives you the blueprint.

Without it, You're just prescribing to the average patient. With it you are prescribing to a specific person. Agreed. So tell me what you believe is the single most important insight providers miss though when prescribing this stuff.

The medication operates inside a biological system shaped by dozens of genetic variants. Prescribing without understanding those variants is like tuning an engine without knowing what model the car is.

The genetics tell you what medication will hit, what it won't hit. What it needs to be addressed through diet, sleep, stress management, lifestyle, environment, and supplementation to make the medication work as well as it possibly can.

So, you know, one of the benefits is the way that you approach it. This is that differentiator. And it's the reason that have better, more meaningful outcomes.

Well, thank you. Thank you, and I mean, that's what we pride ourselves on, right? And having this aim print has been such a game changer in my practice.

I thought ordering over 80 biomarkers was huge. When you have that genetic factors and those genetics in front of you got it. You know, I'll give you an instance.

So Ali was like, well, you respond so well. One of the things guys we talked about was the fasting genes and we'll do that in another episode. And I am fantastic when I fast right I was all green funny enough when i do fast I feel

fantastic alley coincidentally genetics.

doesn't do well, son of a gun, when Allie tries to fast like I do, she does horribly, right? Even worse outcomes, headaches, not feeling well. Really lethargic.

On the other hand, I'm ready to rock and roll, Right? I like food. What food? Like I am that good. I want to thank you for today. I know we made this again, the genetics of weight loss medications, but like Dr.

Personalized Protocols and Closing Thoughts

Gibson said, guys, this can go in so many different variants. And we said a lot of different gene variants today as well. It sounds confusing. Trust me, until I really got involved in genetics and neutral genomics, some of the stuff was confusing to me.

But it's amazing. Dr Gibson, I appreciate your professionalism and your knowingness of all this. It is how amazing we can streamline a person by having their genes.

It's period. And it really, it's really is life changing, right? People say that nonchalantly. Oh, no, this really it is. I mean, I always say it on all my advertisements and every time I talk with a patient, the aim print is just not giving you data.

it Is absolutely giving a direction. on which way to take that information, right? And to prevent those certain things. I want to thank you again. This isn't going to be the last time we hear from you on the Aimprint.

Thank you for doing this initial series with me. Guys, again, beyond the scale, like us, share us with people who love this stuff. The TikTok, as you could see, behind Dr.

Gibson is a great book that you wrote. When did that come out? Can I ask you that? Oh, 2022. 2022, so guys, please also look up Dr. Piper Gibson. She's a genius in the world of what she does.

And I'm always humbled that you're saying yes to coming on to the show. So thank you so much. You are welcome. Thank you for being my really nerdy genetic bestie.

Your welcome, this is what we talk about as we're talking about genes. It's really neat stuff. Again, thank Thanks for spending time with us on Beyond the Scale.

If today's episode helped you see your health differently, like the show, share it, and we'll see you next time on beyond the scale.