

Your GLP-1 & Metabolic Response results, explained plainly first.

12 genes and 14 variants backed by peer-reviewed research, explained in plain language first, with the full technical data and a physician summary later in this same report.

PREPARED FOR

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REPORT ID

DEMO-000142

REPORT DATE

October 30, 2026

PANEL VERSION

3.0 | Shareable

Sample report for demonstration only. "Alex Morgan (demo)" is a fictional demo profile, not a real person. This shareable copy hides rsIDs and exact genotypes.

PART 1 | UNDERSTANDING YOUR RESULTS

How your results relate to each medication

This is not a recommendation for or against any drug and not a prediction of your outcome. It is a plain count of how many genes tied to each medication came back with no watch-out finding (favorable findings count as no watch-out). The gene pages explain what each finding means, and a licensed prescriber makes the call.

Semaglutide

Ozempic, Wegovy, Rybelsus

2 of 4 genes

showed no watch-out finding

GLP1R ARRB1 TCF7L2 FTO

Highest alignment in this panel

Tirzepatide

Mounjaro, Zepbound

3 of 4 genes

showed no watch-out finding

GLP1R GIPR TCF7L2 MC4R

Not a recommendation for this medication.

Liraglutide

Saxenda, Victoza

2 of 3 genes

showed no watch-out finding

GLP1R ARRB1 TCF7L2

GENE BY GENE

What each gene tells you



The dots show how many copies of the studied variant you carry: 0, 1 or 2. They are not a good or bad rating. Some variants are linked to a watch-out, some to a favorable finding, and some are still being studied; the text says which.

LAYER 1 Will it work? Genes in the drug's direct pathway**GLP1R** GLP-1 Receptor**1 copy**

Heterozygous

GLP1R makes the receptor every GLP-1 medication attaches to, the lock the drug's key fits into. You carry one copy of a variant that, in the largest study to date, came with a little more weight loss on average (about 0.76 kg, or 1.7 lb, per copy) and a bit more nausea while the dose is being raised.

Your result: hidden 1 copy of the studied variant

GLP1R GLP-1 Receptor



0 copies

Common / Common

A second spot in the same receptor gene. You carry the common form, so nothing here shifts your expected response.

Your result: hidden 0 copies of the studied variant

GLP1R GLP-1 Receptor



0 copies

Common / Common

A third spot in the receptor gene that may affect how much receptor is made. You carry the common form.

Your result: hidden 0 copies of the studied variant

ARRB1 Beta-Arrestin-1



0 copies

Common / Common

ARRB1 helps reset the GLP-1 receptor after it fires. You carry the common form, so this gene does not shift your expected response.

Your result: hidden 0 copies of the studied variant

TCF7L2 Transcription Factor 7-Like 2



0 copies

Common / Common

TCF7L2 is the common gene most strongly linked to type 2 diabetes. You carry the common form here.

Your result: hidden 0 copies of the studied variant

LAYER 2 Why you eat the way you do Appetite, cravings and fullness signals

FTO Fat Mass & Obesity-Associated



2 copies

Homozygous

FTO helps set how strongly your brain's hunger and reward circuits respond to food. You carry two copies of a variant linked to stronger food cues and higher body weight (about 3 kg on average). GLP-1 drugs act on these same appetite circuits, so this explains part of why you eat the way you do. It does not predict how well a drug will work.

Your result: hidden 2 copies of the studied variant

MC4R Melanocortin 4 Receptor



0 copies

Common / Common

MC4R is a key fullness switch in the brain. You carry the common form at this spot.

Your result: hidden 0 copies of the studied variant

CNR1 Cannabinoid Receptor 1



1 copy

Heterozygous

CNR1 makes the brain receptor behind cravings and food reward. You carry one copy of a variant that some studies link to appetite and weight traits. The evidence is early and it does not predict drug response.

Your result: hidden 1 copy of the studied variant

LEP Leptin



0 copies

Common / Common

LEP makes leptin, the hormone that tells your brain you have enough energy stored. You carry the common form.

Your result: hidden 0 copies of the studied variant

LEPR Leptin Receptor



0 copies

Common / Common

LEPR makes the brain's leptin receptor. You carry the common form.

Your result: hidden 0 copies of the studied variant

GIPR GIP Receptor



0 copies
Common / Common

GIPR only matters for tirzepatide (Mounjaro, Zepbound), the drug that also activates this second receptor. You do not carry the variant tied to more nausea on tirzepatide.

Your result: hidden 0 copies of the studied variant

CTRB1 Chymotrypsinogen B1



0 copies
Common / Common

CTRB1 makes a pancreatic digestive enzyme. You carry the common form. Full disclosure: this gene matters for a different diabetes drug class (DPP-4 inhibitors) and did not predict GLP-1 response in testing.

Your result: hidden 0 copies of the studied variant

CYP2D6 Cytochrome P450 2D6



0 copies
Common / Common

GLP-1 drugs are not processed by CYP2D6, but some medicines often taken alongside them are. You carry the common form here.

Your result: hidden 0 copies of the studied variant

CYP3A4 Cytochrome P450 3A4



0 copies
Common / Common

CYP3A4 processes about half of all prescription drugs, though not GLP-1 drugs themselves. You carry the common form.

Your result: hidden 0 copies of the studied variant

REFERENCE

Terms used in this report

rsID

A reference number (like rs9939609) for one specific spot in DNA that researchers have studied.

Receptor agonist

A drug that binds to and switches on a receptor, the lock and key a medication uses to work.

GWAS

Genome-wide association study: a large study scanning DNA for differences linked to a trait or drug response.

Evidence level (A / B)

Level A: large or replicated studies back the finding. Level B: smaller or single studies, or a plausible mechanism.

Heterozygous / homozygous

Carrying one copy (heterozygous) or two copies (homozygous) of a variant. One copy comes from each parent.

Research Use Only

General wellness insight, not a diagnosis. Discuss findings with a licensed clinician.

All assessed variants

GENE	VARIANT	EVIDENCE	YOUR RESULT	TECHNICAL NOTE CITATION
GLP1R	<div>hidden</div> <div>Pro7Leu</div>	LEVEL A	1 copy <div>hidden</div>	<p>Missense variant in the receptor signal peptide. Each effect allele associated with ~0.76 kg additional weight loss on GLP-1 RAs and higher nausea/vomiting incidence (genome-wide significant; replicated in All of Us). Effect is modest relative to dose, drug, sex and duration.</p> <p><i>Su QJ et al., Nature 2026 (N=27,885)</i></p>
GLP1R	<div>hidden</div> <div>Gly168Ser</div>	LEVEL B	0 copies <div>hidden</div>	<p>Ser allele: 0.08% (0.9 mmol/mol) smaller HbA1c reduction per allele across 4,571 patients (p=6.0x10-5). A prospective semaglutide study (n=112) reported faster weight loss in AA homozygotes, modified by sex. Direction for weight outcomes is not settled.</p> <p><i>Dawed AY et al., Lancet Diabetes Endocrinol 2023; Phan A et al., Obesity 2025</i></p>
GLP1R	<div>hidden</div> <div>regulatory</div>	LEVEL B	0 copies <div>hidden</div>	<p>Associated with obesity and metabolic parameters in a Polish cohort. No replicated association with GLP-1 RA response; included as receptor-expression context.</p> <p><i>Michalowska J et al., Front Endocrinol 2022</i></p>
ARRB1	<div>hidden</div> <div>Thr370Met</div>	LEVEL A	0 copies <div>hidden</div>	<p>Met allele: 0.25% (2.7 mmol/mol) greater HbA1c reduction per allele (p=5.2x10-5). More common in Hispanic and American Indian / Alaska Native populations (6-11%) than in Europeans, with similar effect size. Glycemic endpoint; weight effect not reported.</p> <p><i>Dawed AY et al., Lancet Diabetes Endocrinol 2023 (n=4,571)</i></p>
TCF7L2	<div>hidden</div> <div>intronic</div>	LEVEL B	0 copies <div>hidden</div>	<p>T allele is the strongest common T2D risk variant and reduces incretin-stimulated insulin secretion. Reports of attenuated glycemic response to GLP-1 RAs are inconsistent; no evidence of effect on weight loss or cardiovascular outcomes.</p> <p><i>Pearson ER et al., Diabetologia 2017</i></p>
FTO	<div>hidden</div> <div>intronic</div>	LEVEL A	2 copies <div>hidden</div>	<p>A allele: ~0.4 kg/m2 higher BMI per allele; AA homozygotes ~3 kg heavier on average. fMRI shows stronger food-cue reward responses in AA carriers. Association with GLP-1 RA response is not established.</p> <p><i>Frayling TM et al., Science 2007; Melhorn SJ et al., Am J Clin Nutr 2018</i></p>
MC4R	<div>hidden</div> <div>near MC4R</div>	LEVEL A	0 copies <div>hidden</div>	<p>Common GWAS variant ~188 kb downstream of MC4R; C allele ~0.22 kg/m2 higher BMI per allele. Not a monogenic MC4R deficiency call: rare loss-of-function variants require sequencing and are not assessed here.</p> <p><i>Loos RJF et al., Nat Genet 2008</i></p>
CNR1	<div>hidden</div> <div>1359G>A</div>	LEVEL B	1 copy <div>hidden</div>	<p>Synonymous variant associated with obesity-related traits in some candidate-gene studies; findings inconsistent. CB1 and GLP-1 signaling converge on hypothalamic appetite circuits (mechanistic). No GLP-1 RA response data.</p> <p><i>Candidate-gene literature; mechanistic (emerging)</i></p>
LEP	<div>hidden</div> <div>-2548G>A</div>	LEVEL B	0 copies <div>hidden</div>	<p>Promoter variant associated with circulating leptin levels in some cohorts. Context for leptin signaling; no direct GLP-1 RA response data.</p> <p><i>Hu W et al., Endocr Connect 2025 (review)</i></p>
LEPR	<div>hidden</div> <div>Gln223Arg</div>	LEVEL B	0 copies <div>hidden</div>	<p>Among the most studied obesity candidate SNPs; associations with BMI vary by population. Leptin resistance and GLP-1 signaling share hypothalamic circuits (mechanistic). No direct GLP-1 RA response data.</p> <p><i>Hu W et al., Endocr Connect 2025 (review)</i></p>

GENE	VARIANT	EVIDENCE	YOUR RESULT	TECHNICAL NOTE CITATION
GIPR	<div>hidden</div> Glu354Gln	LEVEL A	0 copies <div>hidden</div>	<p>Tirzepatide-specific nausea/vomiting signal; not observed in semaglutide users in the same cohort. Individuals homozygous for risk alleles at both GIPR rs1800437 and GLP1R rs10305420 had 14.8-fold odds of tirzepatide-associated vomiting (95% CI 6.2-35.8) versus double non-risk homozygotes.</p> <p><i>Su QJ et al., Nature 2026 (N=27,885)</i></p>
CTRB1	<div>hidden</div> eQTL	LEVEL B	0 copies <div>hidden</div>	<p>eQTL increasing CTRB1/2 expression. Strong DPP-4 inhibitor evidence (0.51% lower HbA1c response); GLP-1 RA weight response non-significant (OR 1.12, p=0.84, n=116). Reported as a null-result disclosure.</p> <p><i>t'Hart LM et al., Diabetes 2013; Kyriakidou A et al., J Pers Med 2022</i></p>
CYP2D6	<div>hidden</div> *4	LEVEL B	0 copies <div>hidden</div>	<p>CYP2D6*4 no-function allele (single-SNP call; does not capture copy-number variants or other star alleles). Relevance is indirect: delayed gastric emptying on GLP-1 RAs may alter absorption of co-prescribed CYP2D6 substrates (e.g., paroxetine, metoprolol).</p> <p><i>Jayathilaka NS et al., Clin Pharmacol Drug Dev 2025</i></p>
CYP3A4	<div>hidden</div> *22	LEVEL B	0 copies <div>hidden</div>	<p>CYP3A4*22 reduced-expression allele. GLP-1 RAs are cleared by proteolysis, not CYP3A4; relevance is for co-medications whose absorption may shift with delayed gastric emptying.</p> <p><i>PharmGKB CYP3A4*22 annotation</i></p>

Clinician summary

For prescriber use. This page restates the panel findings for a clinical encounter. It is a supplementary data point, not a substitute for history, examination or standard contraindication screening, and it does not recommend for or against any medication or dose. Tier tags (favorable / mixed / lower alignment: 70% or more, 55-69%, under 55%) summarize the share of each drug's relevant genes with no watch-out finding, as a starting point for clinician review.

Semaglutide Ozempic, Wegovy, Rybelsus

LOWER ALIGNMENT

2/4 relevant genes with no watch-out finding | 50% alignment | Genes: GLP1R, ARRB1, TCF7L2, FTO

GLP1R: 1 copy. Literature association with modestly greater average weight loss and higher early nausea/vomiting incidence; titration tolerability may be relevant. FTO: 2 copies. Common adiposity variant; context for appetite, not a response predictor.

Tirzepatide Mounjaro, Zepbound

FAVORABLE ALIGNMENT

3/4 relevant genes with no watch-out finding | 75% alignment | Genes: GLP1R, GIPR, TCF7L2, MC4R

GLP1R: 1 copy. Literature association with modestly greater average weight loss and higher early nausea/vomiting incidence; titration tolerability may be relevant.

Liraglutide Saxenda, Victoza

MIXED ALIGNMENT

2/3 relevant genes with no watch-out finding | 67% alignment | Genes: GLP1R, ARRB1, TCF7L2

GLP1R: 1 copy. Literature association with modestly greater average weight loss and higher early nausea/vomiting incidence; titration tolerability may be relevant.

Co-medication watch: no reduced-function CYP2D6*4 or CYP3A4*22 alleles detected. Single-SNP calls only; CNVs and other star alleles are not assessed.

Standard screening reminders

This genetic panel does not screen for the items below. They are standard class-wide considerations for GLP-1/GIP receptor agonists and should be checked independently of genotype.

- Personal or family history of medullary thyroid carcinoma (MTC) or Multiple Endocrine Neoplasia type 2 (MEN2): boxed-warning contraindication across the class.
- History of pancreatitis.
- Severe gastroparesis or other significant GI motility disorders.
- Pregnancy or planning pregnancy.
- Concurrent insulin or sulfonylurea use (hypoglycemia risk).

RESEARCH USE ONLY (RUO)

This report is for Research Use Only and is not FDA-cleared, FDA-approved or CE-IVD certified. Findings reflect statistical associations from published studies; effect sizes are modest, and dose, drug choice, adherence, diet and sex usually matter more than any single variant. Most studies were in people of European ancestry. Not a diagnosis, prescription recommendation or prediction of individual outcome. Always consult a licensed physician before acting on any finding.

REFERENCES

1. Su QJ, Ashenurst JR, Xu W et al. Genetic predictors of GLP-1 receptor agonist weight loss and side effects. Nature. 2026. doi:10.1038/s41586-026-10330-z
2. Dawed AY et al. Pharmacogenomics of GLP-1 receptor agonists: a genome-wide analysis of observational data and large randomised controlled trials. Lancet Diabetes Endocrinol. 2023;11(1):33-41. doi:10.1016/S2213-8587(22)00340-0

3. Phan A et al. A GLP1R gene variant and sex influence the response to semaglutide treatment in patients with severe obesity. *Obesity*. 2025;33(7):1237-1242. doi:10.1002/oby.24300
4. Michalowska J et al. Association of GLP1R variants rs2268641 and rs6923761 with obesity and other metabolic parameters in a Polish cohort. *Front Endocrinol*. 2022.
5. Pearson ER et al. TCF7L2 genotype and response to GLP-1 receptor agonists. *Diabetologia*. 2017.
6. Frayling TM et al. A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science*. 2007;316:889-894.
7. Melhorn SJ et al. FTO genotype impacts food intake and corticolimbic activation. *Am J Clin Nutr*. 2018;107(2):145-154. doi:10.1093/ajcn/nqx029
8. Loos RJF et al. Common variants near MC4R are associated with fat mass, weight and risk of obesity. *Nat Genet*. 2008;40:768-775.
9. Hu W et al. Leptin and leptin resistance in obesity: current evidence, mechanisms and future directions. *Endocr Connect*. 2025. doi:10.1530/EC-25-0521
10. t'Hart LM et al. The CTRB1/2 locus affects diabetes susceptibility and treatment via the incretin pathway. *Diabetes*. 2013;62(9):3275-81. doi:10.2337/db13-0227
11. Kyriakidou A et al. Clinical and genetic predictors of glycemic control and weight loss response to liraglutide. *J Pers Med*. 2022;12(3):424. doi:10.3390/jpm12030424
12. Jayathilaka NS et al. Integrating pharmacogenomic insights in GLP-1 receptor agonist therapy. *Clin Pharmacol Drug Dev*. 2025. doi:10.1002/cpdd.1624