

Retatrutide

What it is | Calorie-burn claims | Visceral and liver fat | Tirzepatide comparison | Stacking claims | Human trial dosing

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SUMMARY

1. What Retatrutide Is

Retatrutide is a long-acting investigational peptide drug that activates GIP, GLP-1 and glucagon receptors. Human trials show unusually large weight loss, improved glucose control, lower visceral abdominal fat and large reductions in liver fat in people with MASLD. It is designed for once-weekly subcutaneous dosing and has a human half-life of about 6 days. [1-8]

3

RECEPTORS

GIP + GLP-1 + glucagon

~6 days

HALF-LIFE

supports weekly dosing

5

POSITIVE PHASE 3 READOUTS

disclosed by Lilly as of Aug 2026

Who has it been studied in?

The strongest evidence is in adults with obesity or overweight. Trials also include people with type 2 diabetes, severe obesity with cardiovascular disease, obstructive sleep apnea, knee osteoarthritis and MASLD. There is no good evidence that Retatrutide should be chosen simply because someone is male or female. [6-8,19]

● IMPORTANT: TIRZEPATIDE + RETATRUTIDE HAS NOT BEEN TESTED AS A COMBINATION

Some people keep Tirzepatide for appetite suppression and add Retatrutide for a supposed metabolism boost. No controlled human trial has tested this combination. The drugs overlap at GIP and GLP-1 receptors, and the extra calorie burn often used to justify the stack has not been measured in a published human Retatrutide calorimetry study. [3,10-12,17,18]

THE BIGGEST CURRENT CLAIM

2. Does Retatrutide Really Make You Burn Hundreds of Extra Calories a Day?

No published human Retatrutide study has established a fixed daily calorie-burn bonus. In the community discussions reviewed for this guide, the most common concrete estimate was about 100-200 kcal/day; 150-300 also appears. I could not verify 400-600 kcal/day as the dominant figure. None of these numbers comes from published human Retatrutide calorimetry. [17]

Evidence snapshot

Community estimate	One of the most widespread myths online is that Retatrutide burns an extra 100-200 kcal/day.
Retatrutide calorimetry	Completed 85-person study; results have not been posted.
Human glucagon studies	Mixed: ~50-70 kcal/day higher expenditure in one study; no increase in another.

How the claim became convincing

1 Glucagon biology Glucagon can affect energy expenditure and fuel use.	2 Retatrutide adds GCGR It adds glucagon-receptor activity to GIP + GLP-1.	3 Weight loss was very large A hidden metabolism boost became an attractive explanation.	4 Exact numbers appeared A plausible idea turned into precise calorie claims without calorimetry.
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What the human evidence actually says

Retatrutide	A completed 85-person Phase 1 study measured 24-hour energy expenditure, sleep metabolic rate, food intake and appetite, but ClinicalTrials.gov still lists no results posted as of Aug 21, 2026. [21]
Glucagon alone	One controlled infusion study found roughly 50-70 kcal/day higher measured expenditure; another 72-hour whole-room study found no increase in sleeping, basal or 24-hour expenditure. Neither used Retatrutide. [10,20]
Tirzepatide	Human calorimetry found lower food intake and higher fat oxidation, but no increase in 24-hour or sleeping expenditure after accounting for body-composition change. [11]

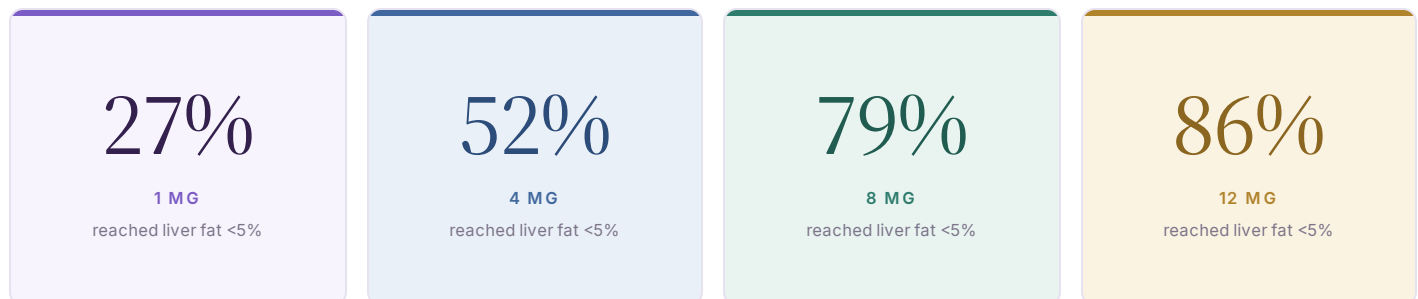
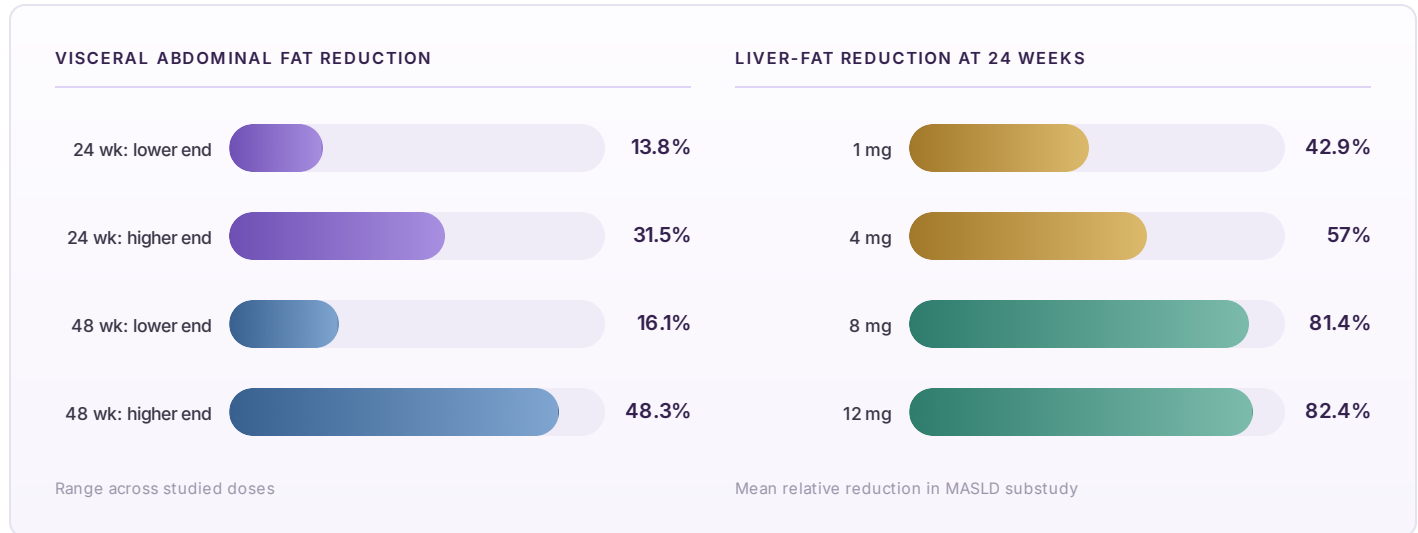
Where walking / NEAT fits If the only goal is to add roughly 100-200 calories of daily expenditure, extra walking, steps or other non-exercise movement can often reach that general range depending on body size and activity. That does not reproduce Retatrutide pharmacology. It does show why an unmeasured "metabolism bonus" is a weak reason to stack two overlapping incretin drugs.

Heart rate is not a calorie meter Retatrutide can raise heart rate, but a faster pulse cannot be converted into a daily calorie-burn number. The human data do not show that the heart-rate increase explains hundreds of extra calories per day. [3,10,20]

WHAT "FAT BURNING" ACTUALLY MEANS

3. Visceral Fat and Fatty Liver: These Claims Have Real Human Data

These effects were measured with imaging in humans. The useful distinction is that Retatrutide does reduce visceral fat and liver fat, but the studies do not show selective spot-fat burning or proof of fibrosis reversal. [5]



What the study shows

Retatrutide substantially reduced MRI-measured liver fat in people with obesity and MASLD, and it substantially reduced visceral abdominal fat.

What it does not show

It did not prove that Retatrutide independently "heals" every form of fatty liver disease, reverses fibrosis, or selectively burns visceral fat while sparing subcutaneous fat.

Why the result makes sense

The liver responds quickly to changes in energy balance, insulin sensitivity and fatty-acid flow. In the substudy, liver-fat reduction tracked strongly with body-weight and abdominal-fat loss. That fits the human data better than the idea that glucagon simply "melts liver fat" on its own.

MUSCLE PRESERVATION CLAIM

4. Does Retatrutide Preserve More Muscle Than Other GLP-1 Drugs?

A common claim is that Retatrutide is more muscle-sparing because its glucagon activity shifts more of the weight loss toward fat. Human data do show fat-predominant weight loss. They do not show a unique muscle-preserving effect. [9]

Why the claim sounds logical

1	2	3
Most of the loss is fat Retatrutide produces large fat-mass reductions, so body-fat percentage can fall quickly.	Glucagon changes fuel use That makes "burn more fat, spare muscle" sound plausible. But fuel use is not an anti-catabolic muscle signal.	A leaner look can fool the eye Less fat, glycogen and water can make someone look smaller or flatter without showing how much contractile muscle changed.

What the human body-composition study actually found

Study	36-week Phase 2 DXA substudy in adults with type 2 diabetes.
What changed	Fat mass fell substantially. Lean mass also fell; the study did not show that weight loss was exclusively fat.
What the authors concluded	The proportion of lean-mass loss relative to total weight loss was similar to other obesity treatments. [9]

Why glucagon does not prove muscle sparing	Glucagon receptor activity changes fuel and amino-acid metabolism. The Retatrutide body-composition paper even notes a theoretical concern that chronic glucagon signaling could lower circulating amino acids and reduce muscle protein synthesis. The human data were reassuring that lean loss was not disproportionate - but they did not show a special protective effect. [9]
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How it compares with other weight-loss approaches

Retatrutide	No proven advantage	Human substudy: lean-mass share was similar to other obesity treatments. [9]
Tirzepatide	~25% lean mass	About 75% of weight lost was fat and 25% lean mass in SURMOUNT-1 DXA. [26]
Semaglutide	~35% pooled estimate	A 2026 meta-analysis estimated 35.2% of weight loss was lean mass. [27]
Lifestyle + resistance training	~18% pooled estimate	The same meta-analysis found the lowest lean-mass share when resistance training was included. [27]

Do not treat these as a head-to-head ranking	The studies used different populations, durations and methods. DXA "lean mass" is also broader than skeletal muscle: it includes water, glycogen, organs and other non-fat tissue.
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Conclusion	Retatrutide appears fat-predominant, not uniquely muscle-sparing. There is no good evidence that choosing Reta instead of Tirzepatide or Semaglutide will preserve more skeletal muscle. If muscle retention matters, resistance training, adequate protein and avoiding an unnecessarily aggressive calorie deficit have a more direct rationale than relying on Retatrutide's glucagon activity. [27]
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TIRZEPATIDE VS RETATRUTIDE

5. Tirzepatide vs Retatrutide: What Is Actually Different?

Tirzepatide GIP GLP-1 Approved drug · dual agonist	Retatrutide GIP GLP-1 GCGR Investigational · triple agonist
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Appetite / food noise	Both reduce food intake. Tirzepatide has mature approved obesity data and a strong real-world appetite-suppression track record. Retatrutide also suppresses appetite, but community reports are more variable. No validated head-to-head food-noise score exists.
Energy expenditure	Tirzepatide calorimetry did not show an increase in 24-hour expenditure after accounting for body-composition change. Retatrutide has no published human calorimetry result establishing a fixed calorie-burn amount. [11,21]
Visceral / liver fat	Both have human imaging evidence. Retatrutide produced very large liver-fat reductions in a MASLD substudy; Tirzepatide also reduced liver fat, VAT and ASAT in SURPASS-3 MRI. [5,13]
Current status	Tirzepatide is FDA-approved for obesity/weight management, OSA in adults with obesity, and type 2 diabetes under Mounjaro. Retatrutide remains investigational. [18]
Head-to-head	TRIUMPH-5 directly compares the two in obesity and is scheduled through late 2026. Until it reports, separate-trial percentages are not a direct comparison. [14]

Who may be better suited to each one?

Need an approved, standardized treatment	TIRZEPATIDE	Established product, dosing, contraindications and monitoring. Retatrutide is still investigational.
Type 2 diabetes is a major issue	TIRZEPATIDE	Approved for T2D. Retatrutide has strong glucose-lowering trial results but no approval yet.
Obesity-related OSA	TIRZEPATIDE	Zepbound is approved for moderate-to-severe OSA in adults with obesity.
Obesity + knee osteoarthritis	RETATRUTIDE SIGNAL	Direct Phase 3 evidence for major weight loss plus better knee pain/function; still investigational. [19]
Largest average weight loss seen so far	RETATRUTIDE SIGNAL	Higher averages in separate trials, but this does not prove superiority before TRIUMPH-5.
Food noise is the main problem	NO PROVEN WINNER	Tirzepatide often has strong appetite suppression in practice; Retatrutide can also suppress appetite. Anecdotes are not a head-to-head test.
High resting heart rate / palpitations	EXTRA CAUTION	Do not choose Retatrutide for a "metabolism boost." A faster pulse is not a useful treatment target.

SEPARATE-TRIAL AVERAGE WEIGHT LOSS



SURMOUNT-1: 72 wk vs TRIUMPH-1: 80 wk — not head-to-head

Do not subtract the two percentages and call Retatrutide 7.4% better. The trials used different participants and durations. [6,12,14]

If Retatrutide is not suppressing appetite enough

Adding Tirzepatide just to keep Retatrutide for a supposed calorie-burn bonus is hard to justify. Tirzepatide already covers GIP + GLP-1 signaling, and the fixed Retatrutide calorie bonus has not been demonstrated. Tirzepatide plus regular walking or more daily movement is not the same drug effect as Retatrutide, but it can address the narrower goal of strong appetite control plus extra activity-related calorie expenditure without stacking two overlapping incretin drugs. [11,17,18,21]

COMMUNITY STACKING CLAIMS

6. Tirzepatide + Retatrutide vs Retatrutide + Cagrilintide

These stacks are popular for different reasons. Tirzepatide + Retatrutide is usually justified as more appetite control + extra metabolism. Retatrutide + Cagrilintide is usually used when hunger or food noise remains a problem. Neither combination has controlled Retatrutide outcome data. [15,17]

Tirzepatide + Retatrutide



High overlap · GIP + GLP-1 are duplicated

Retatrutide + Cagrilintide



Lower overlap · adds an amylin satiety pathway

Tirzepatide + Retatrutide

HIGH OVERLAP

GIP and GLP-1 are duplicated. The popular appetite + calorie-burn rationale depends heavily on an unmeasured Retatrutide metabolism number.

Retatrutide + Cagrilintide

LESS OVERLAP

Adds amylin-based satiety signaling rather than a second GIP/GLP-1 drug. The rationale is clearer when hunger is the problem, but the combination is still untested.

More appetite suppression

NOT ALWAYS BETTER

Very strong satiety can make it harder to eat enough protein, fluids and micronutrients and may worsen nausea, constipation or dehydration.

Simplest decision rule

If the problem is food noise or hunger, fix that problem directly rather than assuming a second overlapping incretin is needed to preserve a metabolism bonus that has never been quantified in humans.

A SECOND MAJOR COMMUNITY MYTH

7. Why "Glucagon Does Not Turn On Until 4 mg" Became So Common

The claim sounds scientific because 4 mg appears repeatedly in the clinical program. The evidence does not show a binary receptor switch at that dose.

1

Trial doses created landmarks

Phase 2 used 1, 4, 8 and 12 mg, and 4 mg later became the lowest main Phase 3 target.

2

Some signals looked clearer at 4 mg+

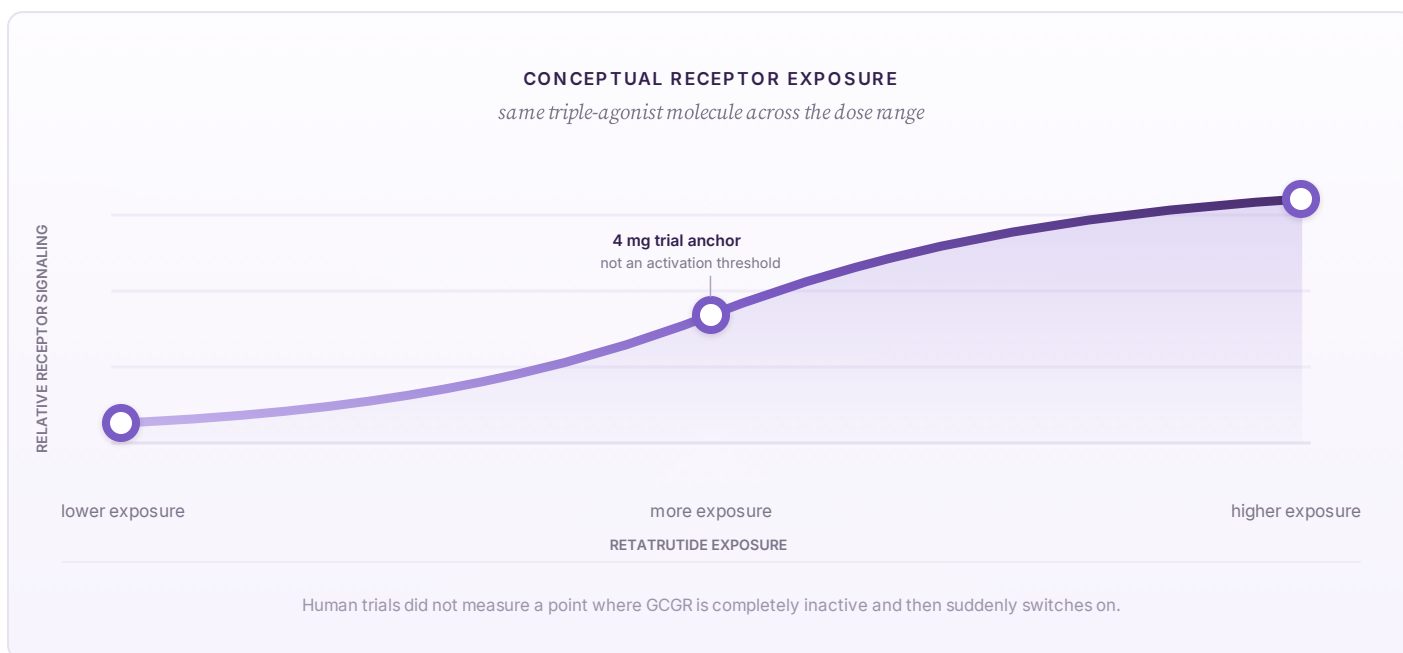
That made 4 mg feel biologically special even though receptor occupancy was not measured that way. [5]

3

A dose anchor became a switch

Community repetition turned "4 mg is an important study dose" into "glucagon is off below 4 mg."

Dose-response is a continuum, not an on/off switch



What the biology supports

Retatrutide is the same triple-agonist molecule at every dose. Receptor signaling changes with concentration; it does not become a different drug at 4 mg.

What the data cannot tell us

The trials do not isolate exactly how much of each dose's effect came from GIP, GLP-1 or glucagon, and they do not establish a human receptor-occupancy threshold at 4 mg.

Key point

4 mg is a real, effective human trial dose. It is not a demonstrated glucagon activation threshold.

HUMAN DOSE EVIDENCE

8. Dosing: What Was Studied, What People Do Online, and What Makes Sense



How to read the dose numbers

2 mg	A genuine Phase 3 starting dose. It is not the universal maintenance dose.
4 mg, 9 mg, 12 mg	Principal target doses in the initial Phase 3 obesity program.
6 mg	A genuine Phase 3 escalation step on the way to 9 or 12 mg; not one of the principal TRIUMPH-1 targets.
8 mg	A genuine Phase 2 target dose. The pivotal Phase 3 program shifted its main targets to 4, 9 and 12 mg.
0.5-1 mg	Low-dose human exposure exists in Phase 2, but that does not make every community microdose schedule optimized.

Studied Phase 3 dose paths



Why waiting matters

Retatrutide has a mean half-life of about 6 days. With weekly injections, exposure accumulates over several weeks. A four-week step gives the current dose time to build and gives side effects time to become clear. [1,2]

Why faster escalation is weak logic

Not feeling strong appetite suppression in week one does not prove the dose failed. Increasing after only 1-2 weeks can add more drug while exposure from the current dose is still building.

Most evidence-aligned dosing framework

The clinical program supports subcutaneous dosing once weekly, beginning with the studied 2 mg Phase 3 starting dose and allowing about four weeks before the next planned step. That framework fits the roughly 6-day half-life, accumulation and tolerability data. These are trial exposures, not an FDA-approved public dosing schedule. [1-3,6-8]

COMMUNITY PROTOCOLS VS HUMAN RESEARCH

9. Split Dosing, Microdosing, Faster Titration and Cycling

Once weekly	BEST SUPPORTED	The route/frequency used throughout the controlled clinical program.
Twice-weekly split dosing	PLAUSIBLE, UNVALIDATED	Could smooth peaks and troughs on paper, but no controlled trial shows better efficacy or tolerability.
Every 5-6 days	COMMUNITY SCHEDULE	Half-life is not an instruction to redose when half remains; the drug was engineered around weekly dosing.
0.25-1 mg microdosing	COMMUNITY STRATEGY	0.5 and 1 mg have human exposure precedent, but chronic microdosing has not been shown to reproduce Phase 3 outcomes.
Escalating after 1-2 weeks	POORLY SUPPORTED	Exposure is still accumulating, so early increases can outrun tolerability assessment.

Why online protocols can look more clinical than they are

1

Borrow real trial numbers

Several individual doses used online really did appear somewhere in development.

2

Change the schedule

The ladder, interval or split-dosing pattern is then changed from the trial.

3

The whole plan looks clinical

The numbers are real, but the combined protocol may never have been tested.

Rule to remember

A real trial dose does not automatically make the surrounding community protocol a clinical protocol. Dose, frequency, escalation interval, formulation and treatment goal all need to match the study before its evidence can be transferred.

Do Reta, Tirz or Sema need to be cycled to "rest" the receptors?

104 wk

SEMAGLUTIDE

sustained treatment remained effective

~2/3

WEIGHT REGAINED

within a year after Semaglutide withdrawal

14.0%

REGAINED

after Tirzepatide was switched to placebo in SURMOUNT-4

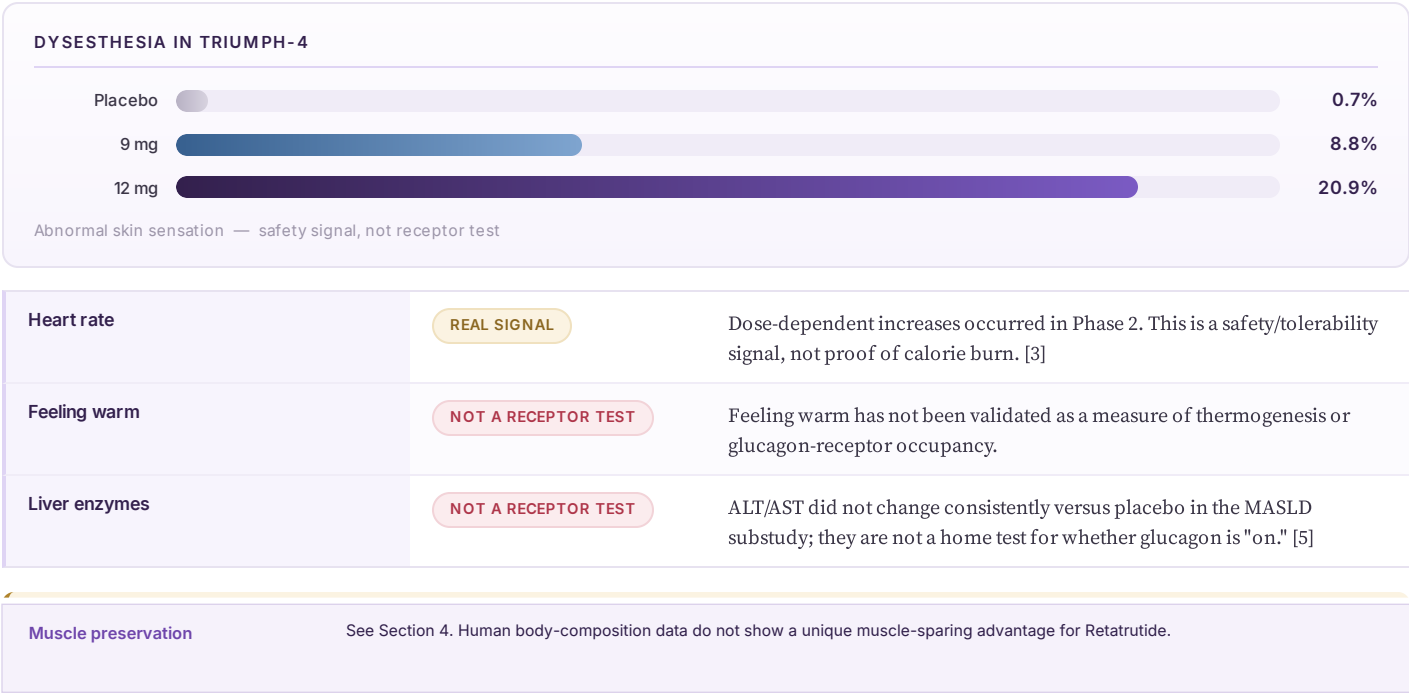
What this means

A plateau or weaker appetite sensation does not prove GLP-1, GIP or glucagon receptors are "burned out." Some individual effects can adapt over time, including part of the gastric-emptying effect, but there is no human evidence that routine drug holidays prevent receptor damage or make these drugs work better when restarted. [22-25]

SYMPTOMS ARE NOT RECEPTOR TESTS

10. Skin Sensitivity, Heat and Heart Rate Do Not Prove "Glucagon Kicked In"

Community discussions sometimes treat side effects as receptor biomarkers. Human trials do not validate that approach.



What is actually established about safety

GI effects	COMMON	Nausea, diarrhea, vomiting, constipation and decreased appetite, especially during escalation. [3,6,7]
Dysesthesia	REPEATED SIGNAL	Now reproduced in Phase 3; generally mild to moderate but clearly dose-related in TRIUMPH-4. [19]
Heart-rate increase	REAL SIGNAL	Observed dose-dependently; do not treat it as a desirable metabolism target.
Excessive weight loss	PRACTICAL RISK	Some TRIUMPH-4 discontinuations were associated with perceived excessive weight loss. [19]

● NO APPROVED RETAIL RETATRUTIDE PRODUCT EXISTS

Clinical-trial Retatrutide uses a controlled investigational formulation. Lilly warns that products sold to consumers outside its trials are not Lilly clinical-trial Retatrutide and may contain the wrong ingredient, concentration, impurities or contaminants. There is no approved universal research-vial concentration, reconstitution method, shelf-life rule or public prescribing label. [8]

Current status	Phase 3 investigational; five positive readouts disclosed as of August 2026. Not FDA approved.
Planned U.S. submission	Lilly has stated a Q1 2027 BLA plan. A planned submission is not approval.

HOW STRONG IS THE EVIDENCE?

11. Evidence Summary

Obesity efficacy	VERY STRONG	Late-stage human evidence with multiple positive Phase 3 readouts.
Fixed extra-calorie claim	NOT ESTABLISHED	No published Retatrutide calorimetry result supports a fixed 100, 200, 400 or 600 kcal/day bonus.
Visceral fat	STRONG	Direct human imaging evidence for substantial VAT reduction.
Liver fat / MASLD	STRONG SIGNAL	Large human liver-fat reductions; not proof of fibrosis reversal.
Cardiovascular outcomes	NOT ESTABLISHED	Dedicated outcomes research is still needed for firm event-reduction conclusions.
Tirzepatide + Retatrutide	NO COMBO EVIDENCE	High pathway overlap and an unsupported calorie-burn rationale.
Retatrutide + Cagrilintide	UNVALIDATED	Less receptor overlap for appetite, but no Retatrutide-specific combination trial.
4 mg glucagon threshold	NOT ESTABLISHED	No human receptor-occupancy study identifies a binary switch at 4 mg.
Split dosing / microdosing	UNVALIDATED	Used in communities, but not compared against once-weekly dosing in controlled outcome trials.
Cycling / receptor burnout	NO RESET EVIDENCE	Long-term treatment remained effective; withdrawal studies showed substantial weight regain after stopping. [22-24]
Once-weekly SC	STRONG	The route and frequency used throughout the clinical program.

12. Conclusion

What is real

Retatrutide has unusually strong late-stage human evidence for weight loss, glucose improvement, visceral-fat reduction and liver-fat reduction. Once-weekly SC dosing with slow titration is the clear clinical-development framework.

What is still being overstated

A fixed metabolism bonus, a 4 mg glucagon switch, split-dosing superiority, receptor-reset cycling and untested stacks remain community claims rather than established Retatrutide pharmacology.

The practical bottom line

Do not confuse a real dose number with a validated protocol, a side effect with a receptor test, or a plausible mechanism with a measured clinical effect. The human trials support powerful results, but they also make it easier to see which online claims are unnecessary additions to the evidence.

RESEARCH SOURCES

13. Sources Used for This Guide

- 1 **Coskun T, et al. Cell Metabolism (2022).** Retatrutide discovery-to-clinical proof of concept; receptor profile, long half-life and once-weekly design.
- 2 **Retatrutide Phase 1 pharmacokinetic summaries.** Dose-proportional exposure, mean half-life about 6 days and Tmax roughly 12-49 hours.
- 3 **Jastreboff AM, et al. New England Journal of Medicine (2023).** PMID 37366315. Phase 2 obesity trial; dose-response, weight loss, GI tolerability and heart-rate findings.
- 4 **Rosenstock J, et al. The Lancet (2023).** PMID 37385280. Phase 2 type 2 diabetes trial; supports 0.5 mg and higher weekly human exposures.
- 5 **Sanyal AJ, et al. Nature Medicine (2024).** PMID 38858523. MASLD substudy; liver fat, visceral fat, subcutaneous abdominal fat, metabolic biomarkers and correlations with weight loss.
- 6 **Lilly TRIUMPH-1 / 2026 ADA materials.** Phase 3 obesity efficacy; 4, 9 and 12 mg target framework, weight loss and adverse events.
- 7 **Bajaj HS, et al. The Lancet (2026).** PMID 42250575; Lilly 2026 TRIUMPH updates. Peer-reviewed Phase 3 type 2 diabetes data and latest pivotal readouts.
- 8 **Eli Lilly. "What to know about retatrutide," updated 2026.** Investigational status, no legitimate retail product and planned US submission.
- 9 **The Lancet Diabetes & Endocrinology (2025), Retatrutide body-composition substudy.** Large fat-mass loss with lean-mass proportion similar to other obesity treatments.
- 10 **Nair KS, et al. Diabetes (2017), Effects of 13-Hour Hyperglucagonemia on Energy Expenditure.** Controlled human glucagon infusion; roughly 50-70 kcal/day increase under study conditions.
- 11 **Ravussin E, et al. Cell Metabolism (2025).** PMID 40203836. Tirzepatide human calorimetry: no increase in adjusted 24-hour expenditure, with increased fat oxidation and lower food intake.
- 12 **Jastreboff AM, et al. New England Journal of Medicine (2022).** PMID 35658024. SURMOUNT-1; Tirzepatide 15 mg mean weight change -20.9% at 72 weeks.
- 13 **Gastaldelli A, et al. Lancet Diabetes & Endocrinology (2022).** PMID 35468325. SURPASS-3 MRI; Tirzepatide reductions in liver fat, VAT and ASAT.
- 14 **TRIUMPH-5, NCT06662383 / Lilly trial registry.** Direct Retatrutide versus

Look M, et al. Diabetes Obesity and Metabolism (2025). PMID 39996356. SURMOUNT-1 DXA substudy; about 75% of Tirzepatide-associated weight loss was fat mass and about 25% lean mass.
- 15 **Garvey WT, et al. New England Journal of Medicine (2025).** PMID 40544433. CagriSema Phase 3; clinical activity of amylin + GLP-1 coadministration and frequent GI adverse events.
- 16 **Pearson MJ, et al. Journal of Clinical Endocrinology & Metabolism (2026).** PMID 42135195. Retatrutide metabolomics/lipidomics; fatty-acid oxidation-related signatures and metabolic changes.
- 17 **Community-source audit.** Current Reddit, GLP-1 Forum, PeptideDosages and PeptideFox discussions were reviewed for calorie-burn claims, stacking, food-noise strategies, the 4 mg glucagon threshold, microdosing, split dosing, competing dose ladders and receptor-reset claims. Community posts document beliefs and practices; they are not efficacy evidence.
- 18 **FDA Zepbound Prescribing Information, revised February 2026.** Tirzepatide indications, once-weekly dosing and warning that coadministration with another GLP-1 receptor agonist is not recommended.
- 19 **Eli Lilly TRIUMPH-4 Phase 3 results (2025-2026 updates).** Knee OA pain/weight outcomes; dysesthesia rates and discontinuations including perceived excessive weight loss.
- 20 **Whytock KL, et al. Obesity (2021).** PMID 34029448. 72-hour glucagon infusion with whole-room calorimetry; no significant increase in sleeping, basal or 24-hour energy expenditure.
- 21 **ClinicalTrials.gov NCT06313528 / Lilly J11-MC-GZBW.** Completed 85-person Retatrutide study measuring calorie intake, sleep metabolic rate, 24-hour energy expenditure and appetite; no results posted as of Aug 21, 2026.
- 22 **Garvey WT, et al. Nature Medicine (2022).** PMID 36216945. STEP 5; continuous Semaglutide produced sustained weight loss through 104 weeks without a planned receptor-reset cycle.
- 23 **Wilding JPH, et al. Diabetes, Obesity and Metabolism (2022).** PMID 35441470. STEP 1 extension; after Semaglutide withdrawal, participants regained about two-thirds of prior weight loss within a year.
- 24 **Aronne LJ, et al. JAMA (2024).** doi:10.1001/jama.2023.24945. SURMOUNT-4; people switched from Tirzepatide to placebo regained 14.0%, while continued treatment produced another 5.5% reduction.
- 25 **Jalleh RJ, et al. Journal of Clinical Endocrinology & Metabolism (2024).** PMID 39418085. Some gastric-emptying effects show tachyphylaxis over prolonged incretin treatment; this is not the same as receptor "burnout."
- 26 **Lean Mass Changes With Incretin Therapy Versus Lifestyle Intervention (2026).** PMID 41877354. Meta-analysis: lean mass accounted for about 35.2% of Semaglutide weight loss, 25.4% with Tirzepatide, 26.2% with lifestyle alone and 17.5% with lifestyle plus resistance training.