RETATRUTIDE PROTOCOL

Next-generation triple-agonist metabolic regulator driving aggressive fat loss with preserved lean mass signaling.
If GLP-1 was appetite control, Retatrutide is metabolic orchestration

What it is

Retatrutide is a long-acting, investigational synthetic peptide engineered to activate **three metabolic receptors simultaneously** GLP-1 receptor, GIP receptor, Glucagon receptor. Unlike single-pathway incretins, this compound operates across appetite regulation, insulin dynamics, and energy expenditure at the same time.

Result: Retatrutide drives significant fat loss by suppressing appetite and increasing energy expenditure, while largely preserving lean mass when paired with training and adequate protein.

This is not just a satiety drug. It is a central + peripheral metabolic reprogramming signal.

Axis: Metabolic / Incretin / Energy Expenditure Axis

Mechanism: Retatrutide activates GLP-1, GIP, and glucagon receptors simultaneously—reducing appetite, improving insulin signaling, and increasing energy expenditure. The combined effect lowers intake while accelerating fat metabolism

Vial Composition

Component	Amount
Retatrutide	20 mg
Total per vial	**20 mg**
Reconstitution: bacteriostatic water	2 mL
Final concentration: mg/mL (total peptide/ml)	10.0 mg/mL

Dosing Protocol

Parameter	Specification
Injection timing	Morning (AM)
Dose (total)	**1.0 mg**
Retatrutide	1.0 mg
Injection volume	0.1 mL (≈10 insulin units)
Frequency: days/week	2

Protocol Length

	Time Frame
Total duration: weeks	12
Active dosing days: days	24
Vials:	1.2

Supply Calculation

Item	Quantity
Total peptide required	24 mg
Vials required	2 vials (20 mg each)
Insulin syringes	24
BAC water	3 mL (recommended 1-10 mL vials

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RETATRUTIDE PROTOCOL NOTES

Retatrutide is a long-acting, once-weekly triple agonist that activates GLP-1, GIP, and glucagon receptors, producing coordinated effects across appetite regulation, insulin dynamics, and energy expenditure. GLP-1 signaling reduces caloric intake and slows gastric emptying, GIP enhances insulin sensitivity and supports nutrient partitioning, and glucagon receptor activation increases lipolysis and basal metabolic rate. The combined effect is simultaneous intake suppression and metabolic acceleration, distinguishing it from earlier single- or dual-pathway incretins.

Clinically, this translates to substantial reductions in total body weight and visceral adiposity, with improvements in glycemic control, waist circumference, hepatic fat markers, and cardiometabolic risk parameters. The glucagon component contributes to increased energy expenditure, which may reduce the degree of metabolic adaptation typically seen with aggressive weight loss. Lean mass preservation is achievable when therapy is paired with structured resistance training and adequate protein intake; without these, some lean mass reduction can occur, as expected with significant caloric deficit.

From a systems perspective, Retatrutide functions as a central appetite modulator and peripheral metabolic regulator, impacting hypothalamic signaling, pancreatic insulin secretion, hepatic fat metabolism, and adipose tissue mobilization. It is best positioned for patients with significant visceral adiposity, insulin resistance, or plateaued response to GLP-1 monotherapy, and should be implemented with deliberate titration to manage gastrointestinal tolerability and maintain adherence.