Weight Loss Next Level ~ Dose Protocol

SYSTEMS-LEVEL RECOMP ARCHITECTURE

This protocol represents a hierarchical, systems-based approach to metabolic optimization and body recomposition. Rather than targeting isolated symptoms, it addresses the underlying physiological control mechanisms that govern fat mobilization, energy expenditure, and lean tissue preservation.

Retatrutide

Remove fat-loss friction at the hormonal command center. If appetite and energy expenditure aren't aligned, nothing downstream works. Retatrutide fixes the control panel—not the symptoms.

Mito Stack

Upgrade the engine before you press the gas. You don't burn more fat by forcing it—you burn more fat by improving mitochondrial throughput. This increases efficiency so every calorie has somewhere to go.

Tesamorelin / Ipamorelin

Activate growth hormone—don't replace it. GH isn't about size, it's about signaling rhythm. Restore the pulse and fat mobilizes cleaner while lean tissue holds during the cut.

Ipamorelin + MT2

Pulse the axis, turn up the burn. Ipamorelin sharpens GH output while MT2 amplifies melanocortin-driven lipolysis. Two separate pathways, one coordinated push.

Core Principle: Stabilize insulin. Increase mitochondrial output. Restore GH rhythm. Amplify fat signaling. Layered physiology with no shortcuts, no replacement dependency—just clean systems execution.

Retatrutide (Reta)

FOUNDATION AGENT

Triple agonist targeting GLP-1, GIP, and Glucagon receptors to drive appetite suppression and increased energy expenditure.

Axis
Incretin / Metabolic Signaling

Timing
Same day weekly

Route
SubQ (abdomen preferred)

Standard Titration Model

1–4	0.5 mg weekly	Initial titration phase
5–8	1.0 mg weekly	First escalation
9–12	2.0 mg weekly	Therapeutic maintenance
Advanced	4–6 mg weekly	Clinical discretion only

Clinical Considerations

- Titrate slowly to minimize gastrointestinal burden and improve patient tolerance
- Hold dose for minimum 4 weeks before escalating to next tier
- Ideal foundation agent before initiating GH-axis optimization protocols
- Best clinical results when insulin stability is addressed first in treatment hierarchy

Mito Stack

MOTS-C + SS-31 + 5-AMINO-1MQ ± SLU-PP-332

The mitochondrial stack enhances cellular energy efficiency and fat oxidation capacity through multiple complementary mechanisms. By targeting AMPK signaling and mitochondrial function directly, this combination improves substrate utilization and metabolic flexibility without relying on hormonal suppression.

Primary Role

Improve mitochondrial efficiency and fat oxidation capacity

Axis

Mitochondrial / AMPK / Cellular Energy

Timing

AM fasted state preferred

Route

Subcutaneous injection

Sequential Reconstitution Model

Combine 20 mg MOTS-C → into 30 mg SS-31 → into 10 mg 5-Amino-1MQ using sequential transfer method. Reconstitute with 2 mL bacteriostatic water into first vial, then transfer sequentially through remaining compounds.

Dosing Tiers by Clinical Goal

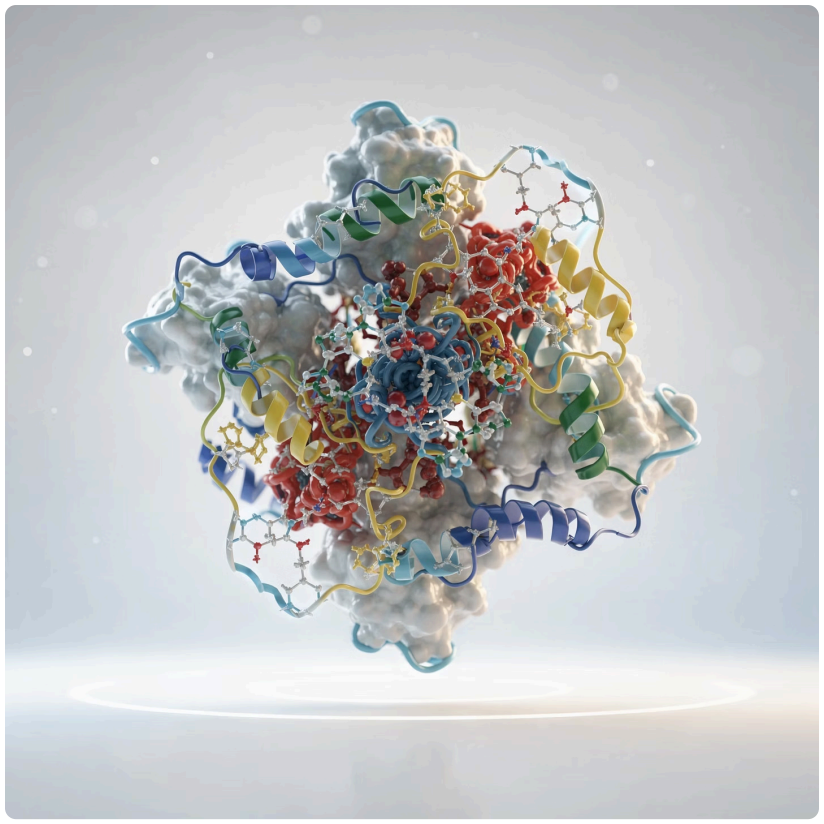
Longevity / Metabolic Health	20 units	3x weekly
Fat Loss / Performance	30–40 units	5x weekly
Aggressive Recomp	50 units	5–6x weekly

Protocol Duration: Run for 6–8 weeks. Layer after insulin stabilization. Enhances GLP-class efficacy and improves growth hormone fat-mobilization response. Monitor for synergistic effects with other metabolic agents.

Tesamorelin / Ipamorelin Blend

10 MG / 3 MG FORMULATION

This dual-peptide formulation provides endogenous growth hormone stimulation through complementary mechanisms: GHRH analog action (tesamorelin) with specific visceral fat targeting properties, combined with ghrelin receptor activation (ipamorelin) for pulsatile GH release optimization.



Axis

Growth Hormone (GHRH + GHSR)

Timing

Evening (empty stomach)

Route

Subcutaneous

Reconstitution & Standard Dosing Protocol

Preparation

- Add 1 mL bacteriostatic water
- Final concentration: 13 mg/mL total peptide
- Gently swirl—do not shake vigorously
- Store refrigerated between uses

Dosing Parameters

Tesamorelin	2.0 mg
Ipamorelin	0.6 mg
Total Volume	0.2 mL (≈20 units)
Frequency	5 nights/week
Duration	12 weeks

Clinical Implementation Notes

- Initiate only after metabolic foundation layer is stabilized with incretin therapy
- Administer in fasted state, preferably 60–90 minutes post-last meal
- Protects lean mass during caloric deficit and enhances preferential fat mobilization
- Avoid pairing with late evening carbohydrate intake to optimize GH pulse amplitude

Ipamorelin + MT2 Combination

DUAL-AXIS OPTIMIZATION

This combination leverages two distinct but synergistic pathways: growth hormone pulse amplification via ghrelin receptor stimulation (ipamorelin), coupled with melanocortin-driven lipolysis and appetite modulation (melanotan II). The result is enhanced fat mobilization through both GH-mediated and MC4R-mediated mechanisms.

Primary Mechanisms of Action

Ipamorelin

GH pulse amplification, sleep architecture optimization, lean mass preservation

Melanotan II

Melanocortin-driven lipolysis via MC4R activation, appetite suppression, increased energy expenditure

Axis	Ipa Timing
GH + Melanocortin	PM (sleep optimization)

MT2 Timing

Earlier evening or 3–4x weekly

Standard Dosing Protocol

Ipamorelin

- **Dose range:** 300–500 mcg per administration
- **Frequency:** 5 nights per week
- **Timing:** Pre-sleep for optimal GH release

Melanotan II

- **Dose range:** 250–500 mcg per administration
- **Frequency:** 3–5 times weekly
- **Initiation:** Start at lower dose to assess individual tolerance

Clinical Considerations & Monitoring

Phase Specificity: Most useful during aggressive fat-loss phases when maximal lipolytic signaling is desired

Mechanism: MT2 increases lipolytic signaling through melanocortin 4 receptor (MC4R) pathway activation

Tolerability: Monitor for nausea and other GI symptoms during initial titration phases

Stacking Caution: Avoid combining with high-dose GLP agonists initially to assess individual response and minimize additive GI effects

Systems-Level Sequencing Recommendation

HIERARCHICAL IMPLEMENTATION

Successful body recomposition requires sequential implementation of interventions in a specific physiological hierarchy. Each layer builds upon the previous, creating a stable foundation before advancing to the next level of metabolic optimization.

Boost Mitochondria

Implement Mito Stack to increase throughput.

Activate GH Axis

Use Tesamorelin/Ipamorelin for signaling optimization.

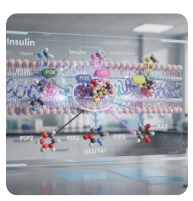
Stabilize Insulin

Begin Retatrutide to secure metabolic foundation.

Add MT2

Introduce MT2 for aggressive body recomposition.

This stepwise approach ensures that upstream metabolic control is established before introducing downstream interventions. Attempting to optimize growth hormone signaling or melanocortin pathways without first stabilizing insulin and improving mitochondrial function often results in suboptimal outcomes and increased side effect burden.

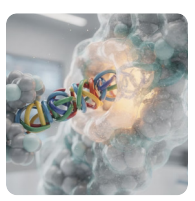


Phase 1: Metabolic Foundation

Establish incretin-based appetite control and energy expenditure optimization. Retatrutide addresses the hormonal command center, creating the necessary substrate for downstream interventions.

Phase 2: Cellular Efficiency

Enhance mitochondrial function and oxidative capacity. The Mito Stack ensures that increased fat mobilization from subsequent phases has adequate metabolic machinery for processing.



Phase 3: Hormonal Signaling

Restore physiological growth hormone pulsatility. Tesamorelin and ipamorelin activate endogenous GH pathways, improving body composition without exogenous hormone replacement.

Phase 4: Advanced Optimization

Layer melanocortin pathway activation for maximal lipolytic drive. MT2 provides an additional fat-mobilization signal when aggressive recomposition goals are pursued.

Key Principle: Each phase typically requires 4–8 weeks for stabilization and response assessment before advancing. Patient-specific factors, tolerance profiles, and clinical goals should guide advancement timing and dose selection within each tier.