THE

FAT LOSS PEPTIDE BIBLE

The Complete Research Guide to

Metabolic Optimization

40+ PEPTIDES 10 CHAPTERS 20+ STACKS

"Science That Can't Be Silenced."

BY REAL PEPTIDES RESEARCH TEAM

www.realpeptides.co

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All peptides discussed are research-grade materials for laboratory use only.

PREFACE

From the Research Desk at Real Peptides

"Science That Can't Be Silenced."

At Real Peptides, our mission is simple: To advance the world's understanding of peptide science through precision, purity, and education.

We exist for the serious researcher — those who refuse to accept surface-level answers or 'bro-science' explanations when it comes to human performance and fat loss.

This Bible was created to fill a gap. While peptides have exploded in popularity, true scientific literacy around their mechanisms, structures, and metabolic pathways remains limited. The internet is flooded with noise — conflicting data, exaggerated claims, and misinformation that discredits legitimate research.

Our goal is to change that.

Every compound in this guide has been carefully selected for its relevance to fat-loss research and metabolic optimization — supported by data from clinical and pre-clinical studies. You'll find molecular formulas, amino-acid sequences, mechanisms, and key findings presented in a clear, consistent format for scientific exploration.

This guide isn't about hype. It's about biology, chemistry, and truth. It's for the researcher who demands transparency. It's for the forward-thinker who wants to understand why a compound works — not just what it does.

IMPORTANT DISCLAIMER

All information provided is for educational and research purposes only. Real Peptides does not promote or imply personal use under any circumstance. All compounds discussed are research-grade materials, not approved for human consumption or therapeutic use unless otherwise specified.

Through knowledge, we empower better research. Through better research, we accelerate discovery. And through discovery — we redefine what's possible for human performance and longevity.

Welcome to Real Peptides. Welcome to The Fat Loss Peptide Bible.

INTRODUCTION

The Science, the Signal, and the Future of Fat-Loss Research

For decades, fat-loss discussion has been dominated by calorie math and surface-level diet tricks. Modern research tells a different story: the body doesn't burn fat because of willpower alone — it burns fat when the right biological switches are activated.

Peptides represent one of the most promising frontiers in metabolic and longevity science — short chains of amino acids that can precisely influence biological pathways governing lipolysis, appetite regulation, energy expenditure, and cellular repair.

Unlike stimulants or restrictive diets, these compounds work at the molecular level, improving the efficiency of fat oxidation rather than forcing the process. The field has advanced dramatically — we now have GLP-1 agonists producing 15-25% weight loss, triple agonists approaching surgical outcomes, and mitochondrial peptides that mimic exercise at the cellular level.

At Real Peptides, we're dedicated to advancing literacy around these compounds — helping researchers understand their mechanisms, synergies, and distinctions.

GUIDE ORGANIZATION	
Chapters 1-2	Direct fat-loss mechanisms (lipolysis, incretins)
Chapter 3	Growth hormone axis (anabolic/catabolic balance)
Chapter 4	Mitochondrial function (cellular energy)
Chapters 5-6	Appetite regulation and support peptides
Chapters 7-10	Practical application and reference

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Welcome to the next evolution in fat-loss research. Welcome to The Fat Loss Peptide Bible.

CHAPTER 1

Foundational Fat-Loss Peptides

Laying the Metabolic Foundation

Before the modern era of GLP-1s and mitochondrial enhancers, these were the compounds that opened the door. They target fat breakdown, energy use, and appetite at the root of metabolism — not by masking symptoms but by improving how the body mobilizes and processes stored energy.

AOD-9604

"The Fat Fragment"

Lipolytic / Fat Metabolism Peptide

Stimulates lipolysis (fat breakdown) while inhibiting lipogenesis (fat formation) without affecting blood glucose or IGF-1 levels.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{78}H_{123}N_{23}O_{23}S_2$	1815.08 Da	~2 hours	4-12 weeks typical research cycles

AMINO SEQUENCE

Tyr-Leu-Arg-Ile-Val-Gln-Cys-Arg-Ser-Val-Glu-Gly-Ser-Cys-Gly-Phe (cyclic disulfide 7– 14)

MECHANISM OF ACTION

AOD-9604 is a synthetic 16-amino acid fragment corresponding to the C-terminal region (residues 176-191) of human growth hormone, with an added N-terminal tyrosine. It selectively upregulates beta-3 adrenergic receptors in adipose tissue, which triggers the breakdown of stored triglycerides into free fatty acids. Unlike full-length HGH, AOD-9604 does not bind to growth hormone receptors, thus avoiding effects on IGF-1, glucose metabolism, or tissue growth. Research in mice lacking beta-3 receptors showed no fat-loss response, confirming this pathway is essential to its mechanism.

RESEARCH FINDINGS

- Phase 2 clinical trials (900+ participants) demonstrated favorable safety profile with no serious adverse effects
- 12-week human trial: 1mg daily showed ~2.6 kg weight loss vs 0.8 kg placebo (~1.8 kg additional loss)
- Pre-clinical: Reduces adipocyte size and epididymal fat mass in ob/ob mice without affecting food intake
- Emerging research suggests potential benefits for cartilage repair and osteoarthritis

RESEARCH STACK COMBINATIONS

- AOD-9604 + Ipamorelin GH pulse stimulation + targeted lipolysis
- AOD-9604 + MOTS-c fat mobilization + mitochondrial oxidation
- AOD-9604 + BPC-157 fat metabolism + tissue repair (especially joints)

KEY DIFFERENTIATOR

Isolates the fat-burning region of GH without growth, glucose, or IGF-1 effects. One of the most extensively safety-tested peptides in human trials.

Research Status: Developed by Metabolic Pharmaceuticals (Australia) in the 1990s. Phase 2 trials completed; development halted 2007 due to modest efficacy. Now GRAS (Generally Recognized as Safe) in the US for food applications.

LAB INSIGHT

"The 'fat fragment' — over 900 humans studied with no serious adverse effects. Think of it as the precision tool that tells fat cells to release their contents."

EXPLORE AOD-9604 realpeptides.co/products/aod9604/

5-Amino-1MQ

"The Metabolic Switch"

NNMT Inhibitor / NAD^+ Modulator

Inhibits nicotinamide N-methyltransferase (NNMT), increasing NAD^+ levels and shifting metabolism toward fat oxidation.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{10}H_{11}N_2^+$	~175 Da (as cation)	Short-acting (exact half-life not fully characterized)	4-8 weeks typical research cycles

MECHANISM OF ACTION

5-Amino-1MQ is a small-molecule inhibitor of NNMT, an enzyme overexpressed in adipose tissue during obesity. NNMT consumes nicotinamide (vitamin B3) and methyl donors, reducing NAD^+ availability. By blocking NNMT, 5-Amino-1MQ preserves nicotinamide for NAD^+ synthesis, boosting cellular energy metabolism. This increases fat oxidation, activates SIRT1 ('longevity gene'), promotes white-to-brown fat conversion, and improves insulin sensitivity — all without affecting appetite or food intake.

RESEARCH FINDINGS

- 11-day treatment in diet-induced obese mice: significant reductions in body weight, white adipose mass, and adipocyte size
- Treated mice showed 50-60% reduction in serum insulin levels
- No reduction in food intake observed — weight loss driven by increased energy expenditure
- 2024 study: Improved grip strength and exercise performance in aged mice, suggesting muscle benefits

RESEARCH STACK COMBINATIONS

- 5-Amino-1MQ + MOTS-c → dual NAD^+/AMPK activation for maximum metabolic enhancement
- 5-Amino-1MQ + SS-31 → cellular energy + mitochondrial protection
- 5-Amino-1MQ + AOD-9604 → enzyme-level + receptor-level fat targeting

KEY DIFFERENTIATOR

Works at the enzyme level rather than receptor level — a 'metabolic reset' that changes how cells process energy without hormonal manipulation.

Research Status: Developed at University of Texas Medical Branch; first characterized 2017. No FDA approval; research compound only.

LAB INSIGHT

"Flipping the switch that tells cells to burn fat for fuel. Unlike appetite suppressants, it increases metabolic rate rather than forcing calorie restriction."

EXPLORE 5-AMINO-1MQ → realpeptides.co/products/5-amino-1mq/

Tesofensine

"The Cognitive Craving Controller"

Triple Monoamine Reuptake Inhibitor

Suppresses appetite and enhances motivation by modulating dopamine, serotonin, and norepinephrine neurotransmission.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{17}H_{23}ClN_2O$	306.83 Da	~200-300 hours (extremely long)	12-24 weeks in clinical trials

MECHANISM OF ACTION

Tesofensine inhibits presynaptic reuptake of dopamine, norepinephrine, and serotonin — the three major neurotransmitters governing appetite, reward, and energy. Unlike stimulants, it doesn't cause significant release of these neurotransmitters, reducing abuse potential. The dopamine effect reduces food reward and cravings; serotonin promotes satiety; norepinephrine increases thermogenesis and energy expenditure.

RESEARCH FINDINGS

- Phase 2 (24 weeks): 0.5mg dose produced 10.6% mean body weight loss vs 2.2% placebo
- Phase 2 extension: Weight loss maintained at 12.8% at 48 weeks with continued treatment
- Improved mood and cognitive function reported in trials
- Originally developed for Parkinson's and Alzheimer's; repurposed after unexpected weight loss

RESEARCH STACK COMBINATIONS

- Tesofensine + Cagrilintide central (brain) + peripheral (gut) appetite control
- Tesofensine + Oxytocin mood stabilization + emotional eating reduction
- Tesofensine + MOTS-c behavioral control + metabolic enhancement

KEY DIFFERENTIATOR

Addresses the psychological dimension of fat loss — reducing cravings and enhancing discipline at the neurological level. Extremely long half-life allows once-daily dosing.

Research Status: Developed by NeuroSearch (Denmark) for Parkinson's disease. Saniona currently holds rights. Approved in Mexico (2023) for obesity.

LAB INSIGHT

"Discipline in a molecule — addresses why willpower fails by rebalancing the brain's reward circuitry."

EXPLORE TESOFENSINE realpeptides.co/products/tesofensine/

Cagrilintide

"The Satiety Anchor"

Long-Acting Amylin Analog

Activates amylin receptors to slow gastric emptying, reduce glucagon secretion, and promote satiety signaling to the brain.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{170}H_{267}N_{47}O_{53}S_2$ (approximate)	~3900 Da	~7 days (designed for weekly dosing)	12-48 weeks in clinical trials

MECHANISM OF ACTION

Cagrilintide is a long-acting analog of amylin, a hormone co-secreted with insulin from pancreatic beta cells. It acts on the area postrema and nucleus tractus solitarius in the brainstem to reduce food intake, slow gastric emptying, and suppress post-meal glucagon release. The weekly formulation uses fatty acid acylation for prolonged albumin binding, similar to semaglutide's design.

RESEARCH FINDINGS

- CagriSema trial (cagrilintide + semaglutide): Up to 15.6% weight loss at 32 weeks
- Synergistic with GLP-1 agonists — combined therapy shows greater efficacy than either alone
- Phase 2: Dose-dependent weight loss up to 10.8% with cagrilintide monotherapy
- Well-tolerated; GI side effects similar to GLP-1 class

RESEARCH STACK COMBINATIONS

- Cagrilintide + Semaglutide CagriSema combination (now in Phase 3 as fixed-dose)
- Cagrilintide + Retatrutide triple-agonist + amylin for maximum gut-brain coverage
- Cagrilintide + Oxytocin physiological + emotional satiety

KEY DIFFERENTIATOR

The only long-acting amylin analog in advanced development. Complements GLP-1s rather than competing — together they engage distinct but synergistic satiety pathways.

Research Status: Novo Nordisk. Currently in Phase 3 trials. CagriSema (cagrilintide + semaglutide combination) showing strong results.

LAB INSIGHT

"The long-game satiety signal — designed to work alongside GLP-1s for next-generation combination therapy."

EXPLORE CAGRILINTIDE → realpeptides.co/products/calgrilintide-10mg/

Melanotan II

"The Dual-Action Modulator"

Non-Selective Melanocortin Receptor Agonist

Activates MC1R (tanning), MC3R/MC4R (appetite/metabolism), and MC5R (exocrine function) with effects on appetite, libido, and pigmentation.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{50}H_{69}N_{15}O_9$	1024.18 Da	~1 hour	4-8 weeks (intermittent dosing common)

AMINO SEQUENCE

Ac-Nle-cyclo[Asp-His-D-Phe-Arg-Trp-Lys]-NH_2

MECHANISM OF ACTION

Melanotan II is a cyclic heptapeptide analog of alpha-MSH that binds to multiple melanocortin receptors. MC4R activation in the hypothalamus reduces food intake and increases energy expenditure. MC3R modulates feeding efficiency and nutrient partitioning. The compound also stimulates melanogenesis (MC1R) and has well-documented effects on sexual function. Its broad receptor profile creates multiple metabolic effects but also limits selectivity.

RESEARCH FINDINGS

- Clinical trials showed reduced food intake and increased satiety in healthy volunteers
- Significant appetite suppression observed at sub-tanning doses
- Weight loss effects confounded by side effects limiting practical application
- Precursor compound for development of selective MC4R agonists

RESEARCH STACK COMBINATIONS

- Melanotan II + Tesofensine → dual appetite suppression pathways
- Melanotan II + AOD-9604 → central appetite control + peripheral lipolysis
- Note: Many researchers prefer selective compounds (PT-141, Setmelanotide) for targeted effects

KEY DIFFERENTIATOR

The original multi-pathway melanocortin agonist — paved the way for selective compounds. Demonstrates proof-of-concept for MC4R-mediated weight loss.

Research Status: Originally developed at University of Arizona (1980s). Never FDA-approved due to side effect profile. Research compound only.

LAB INSIGHT

"The pioneer — showed what's possible through melanocortin pathways, inspiring an entire class of selective drugs."

EXPLORE MELANOTAN II → realpeptides.co/products/melanotan-2-mt2-10mg/

LIPO-C

"The Lipotropic Blend"

Lipotropic Injection Complex

Combination of methionine, inositol, choline, and B-vitamins designed to support hepatic fat processing and energy metabolism.

FORMULA	WEIGHT	HALF-LIFE	DURATION
Variable (multi-component)	Variable	Component-dependent	8-12 weeks typical protocols

MECHANISM OF ACTION

LIPO-C combines lipotropic agents that support the liver's ability to process fats. Methionine is an essential amino acid that aids detoxification and fat metabolism. Inositol helps redistribute body fat and supports insulin signaling. Choline prevents fat accumulation in the liver and supports cell membrane integrity. B-vitamins (B12, B6) are essential cofactors for energy metabolism and methylation reactions.

RESEARCH FINDINGS

- Individual components have established roles in hepatic fat metabolism
- Choline deficiency clearly linked to fatty liver development
- B12 supplementation improves energy in deficient individuals
- Combination effects less studied than individual components

RESEARCH STACK COMBINATIONS

- LIPO-C + 5-Amino-1MQ → methylation support + NAD^+ optimization
- LIPO-C + AOD-9604 → hepatic processing + fat mobilization
- LIPO-C + MOTS-c → metabolic cofactors + mitochondrial function

KEY DIFFERENTIATOR

Not a peptide but a synergistic nutrient blend. Provides foundational support for fat metabolism pathways that peptides activate.

Research Status: Components are established supplements. Often used in medical weight-loss clinics as adjunct therapy.

LAB INSIGHT

"The foundation — ensures the machinery of fat metabolism has the fuel it needs to function optimally."

EXPLORE LIPO-C → realpeptides.co/products/lipo-c/

CHAPTER 2

GLP-1, GIP & Glucagon Pathways

The Modern Revolution in Metabolic Research

Incretin-based peptides have fundamentally transformed fat-loss research. These compounds don't just suppress appetite — they reprogram the body's metabolic set point, creating sustained changes in how energy is stored and utilized.

Semaglutide

"The Appetite Reset"

GLP-1 Receptor Agonist

Activates GLP-1 receptors to reduce appetite, slow gastric emptying, improve insulin sensitivity, and decrease hepatic glucose output.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{187}H_{291}N_{45}O_{59}$	4113.58 Da	~7 days (enabling weekly dosing)	52-68 weeks in major trials

AMINO SEQUENCE

Modified GLP-1 (7-37) with Aib at position 8, Arg Lyssubstitution at 34, and C18 fatty diacid at Lys26

MECHANISM OF ACTION

Semaglutide is a long-acting GLP-1 analog engineered for weekly dosing through strategic amino acid modifications and fatty acid acylation that enables albumin binding. It activates GLP-1 receptors in the pancreas (enhancing insulin secretion), gut (slowing gastric emptying), and brain (reducing appetite via hypothalamic nuclei). The compound reduces food intake by 20-30% while improving glycemic control independently of weight loss.

RESEARCH FINDINGS

- STEP trials: 14.9-17.4% mean weight loss at 68 weeks (2.4mg weekly)
- SELECT trial: 20% reduction in major cardiovascular events in obese patients without diabetes

- STEP 5: Weight loss maintained at 15.2% through 2 years of treatment
- Significant improvements in HbA1c, blood pressure, and lipid profiles

RESEARCH STACK COMBINATIONS

- Semaglutide + Cagrilintide CagriSema combination for enhanced efficacy
- Semaglutide + MOTS-c appetite control + mitochondrial enhancement
- Semaglutide + Tesamorelin metabolic optimization + lean mass preservation

KEY DIFFERENTIATOR

The most extensively studied weight-loss compound in history. FDA-approved for both diabetes (Ozempic) and obesity (Wegovy). Demonstrated cardiovascular mortality benefits.

Research Status: Novo Nordisk. FDA-approved 2021 for chronic weight management. SELECT cardiovascular outcome trial completed 2023.

LAB INSIGHT

"The gold standard — proof that sustained 15%+ weight loss is achievable with pharmacotherapy."

EXPLORE SEMAGLUTIDE realpeptides.co/products/semaglutide/

Tirzepatide

"The Dual Agonist"

GLP-1/GIP Dual Receptor Agonist

Simultaneously activates GLP-1 and GIP receptors, creating synergistic effects on appetite, insulin secretion, and fat metabolism.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{225}H_{348}N_{48}O_{68}$	4813.45 Da	~5 days	72-88 weeks in Phase 3 trials

AMINO SEQUENCE

Synthetic 39-amino acid peptide with GIP backbone incorporating GLP-1 activity, C20 fatty diacid moiety

MECHANISM OF ACTION

Tirzepatide is an engineered 'twincretin' that activates both GLP-1 and GIP receptors. GLP-1 activation provides appetite suppression and glycemic control. GIP adds complementary effects: enhanced insulin secretion, improved beta-cell function, and potentially increased energy expenditure through brown adipose tissue activation. The dual mechanism produces greater weight loss than GLP-1 mono-agonists while maintaining favorable tolerability.

RESEARCH FINDINGS

- SURMOUNT-1: 22.5% mean weight loss at 72 weeks with 15mg dose (vs 2.4% placebo)
- SURMOUNT-2: 15.7% weight loss in patients with type 2 diabetes
- 36.2% of SURMOUNT-1 participants achieved ≥25% weight loss
- Superior to semaglutide 1mg in head-to-head SURPASS trials for diabetes

RESEARCH STACK COMBINATIONS

- Tirzepatide + MOTS-c dual incretin + mitochondrial support
- Tirzepatide + Tesamorelin metabolic control + body composition
- Tirzepatide + BPC-157 metabolic + tissue repair support

KEY DIFFERENTIATOR

First approved dual agonist. Produces ~5-7% greater weight loss than semaglutide in comparable studies. Represents the leading edge of incretin therapy.

Research Status: Eli Lilly. FDA-approved 2023 for chronic weight management (Zepbound) and 2022 for diabetes (Mounjaro).

LAB INSIGHT

"The evolution — proof that multi-receptor activation enhances efficacy. Setting new benchmarks for pharmacological weight loss."

EXPLORE TIRZEPATIDE realpeptides.co/products/tirzepatide/

Retatrutide

"The Triple Agonist"

GLP-1/GIP/Glucagon Triple Receptor Agonist

Activates all three metabolic hormone receptors — GLP-1 (appetite), GIP (insulin), and Glucagon (energy

expenditure) — for maximum fat loss.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{286}H_{452}N_{72}O_{86}$	~6485 Da	~6 days	48-96 weeks in clinical development

MECHANISM OF ACTION

Retatrutide represents the most comprehensive incretin-based approach to obesity. GLP-1 and GIP provide appetite suppression and glycemic benefits. The addition of glucagon receptor activation increases hepatic glucose output, lipolysis, and thermogenesis — essentially telling the body to burn more energy. This 'triple play' addresses both sides of the energy equation: reducing intake while increasing expenditure.

RESEARCH FINDINGS

- Phase 2: 24.2% mean weight loss at 48 weeks with 12mg dose
- TRIUMPH-4 Phase 3: 28.7% weight loss at optimal dosing — approaching surgical outcomes
- Dose-dependent liver fat reduction (up to 86% in NAFLD substudy)
- No unexpected safety signals; GI tolerability similar to GLP-1 class

RESEARCH STACK COMBINATIONS

- Retatrutide + MOTS-c triple receptor + mitochondrial enhancement
- Retatrutide + SS-31 metabolic activation + cellular protection
- Retatrutide + Cagrilintide maximum gut hormone coverage

KEY DIFFERENTIATOR

The first triple agonist in advanced development. Approaching the efficacy threshold previously only seen with bariatric surgery. May represent the ceiling for incretin-based weight loss.

Research Status: Eli Lilly. Phase 3 trials ongoing. Expected FDA submission 2025-2026.

LAB INSIGHT

"The ceiling breaker — demonstrating that 25-30% pharmacological weight loss is achievable. The future of metabolic pharmacotherapy."

EXPLORE RETATRUTIDE realpeptides.co/products/retatrutide/

Survodutide

"The Liver Optimizer"

GLP-1/Glucagon Dual Receptor Agonist

Dual activation of GLP-1 and glucagon receptors with particular efficacy for hepatic fat reduction and metabolic liver disease.

FORMULA	WEIGHT	HALF-LIFE	DURATION
Variable (proprietary)	~4500 Da	~7 days	48-72 weeks in trials

MECHANISM OF ACTION

Survodutide combines GLP-1 receptor activation (appetite suppression, glycemic control) with glucagon receptor activation (increased hepatic lipid oxidation, thermogenesis). The glucagon component particularly targets liver metabolism, making it promising for NAFLD/NASH. Unlike pure GLP-1 agonists, the glucagon activity increases energy expenditure rather than just reducing intake.

RESEARCH FINDINGS

- Phase 2: Up to 19% weight loss at 46 weeks
- MASH/NAFLD studies: >80% reduction in liver fat in some cohorts
- Significant improvements in liver enzymes and fibrosis markers
- Well-tolerated with manageable GI side effects

RESEARCH STACK COMBINATIONS

- Survodutide + MOTS-c hepatic + mitochondrial focus
- Survodutide + BPC-157 liver protection + tissue repair
- Survodutide + NAD+ metabolic + cellular energy support

KEY DIFFERENTIATOR

Specifically designed for metabolic liver disease. May become first-line for NAFLD/NASH with obesity. Glucagon component provides energy expenditure benefits.

Research Status: Boehringer Ingelheim. Phase 3 trials for obesity and MASH ongoing. Breakthrough therapy designation for MASH.

LAB INSIGHT

"The liver specialist — addressing fatty liver disease that often accompanies obesity. A targeted approach for metabolic dysfunction."

EXPLORE SURVODUTIDE → realpeptides.co/products/survodutide-peptide-fat-loss-research/

Mazdutide

"The Eastern Advance"

GLP-1/Glucagon Dual Receptor Agonist

Dual GLP-1 and glucagon receptor activation developed for Asian populations with particular focus on type 2 diabetes and obesity.

FORMULA	WEIGHT	HALF-LIFE	DURATION
Proprietary	~4400 Da	~6-7 days	24-48 weeks in trials

MECHANISM OF ACTION

Mazdutide activates both GLP-1 and glucagon receptors with a balanced profile optimized for both glycemic control and weight loss. The compound was developed with specific consideration for Asian populations, who often develop metabolic disease at lower BMIs and may respond differently to Western-developed therapies.

RESEARCH FINDINGS

- Phase 2 (China): Up to 11.1% weight loss at 24 weeks
- Phase 3: Up to 15% weight loss reported in Chinese populations
- Significant HbA1c reductions in diabetic cohorts
- Generally well-tolerated in Asian populations

RESEARCH STACK COMBINATIONS

- Mazdutide + MOTS-c → dual agonist + metabolic enhancement
- Mazdutide + 5-Amino-1MQ → receptor + enzyme level targeting
- Mazdutide + SS-31 → metabolic + mitochondrial support

KEY DIFFERENTIATOR

Developed specifically with Asian populations in mind. May fill an important gap in obesity treatment for East Asian patients.

Research Status: Innovent Biologics (China). Phase 3 trials in China completed. Regulatory submissions planned for China and potentially global markets.

LAB INSIGHT

"The regional pioneer — recognizing that metabolic therapies may need population-specific optimization."

EXPLORE MAZDUTIDE → realpeptides.co/products/mazdutide-peptide/

Cotadutide

"The Metabolic Balancer"

GLP-1/Glucagon Dual Receptor Agonist

Balanced dual activation of GLP-1 and glucagon receptors for comprehensive metabolic disease treatment.

FORMULA	WEIGHT	HALF-LIFE	DURATION
Proprietary	~4300 Da	~12-15 hours (daily dosing)	24-54 weeks in trials

MECHANISM OF ACTION

Cotadutide provides balanced activation of GLP-1 and glucagon receptors. The GLP-1 component reduces appetite and improves insulin sensitivity, while glucagon activation increases hepatic fat oxidation and energy expenditure. The shorter half-life requires daily dosing but may provide more physiological hormone patterns.

RESEARCH FINDINGS

- Phase 2b: Up to 5.4 kg weight loss at 54 weeks in NAFLD patients
- Significant liver fat reduction (mean 43% relative reduction)
- Improved glycemic control in type 2 diabetes cohorts
- Hepatic benefits independent of weight loss effects

RESEARCH STACK COMBINATIONS

- Cotadutide + MOTS-c → daily dosing pattern + mitochondrial support

- Cotadutide + BPC-157 → hepatic focus + tissue protection
- Cotadutide + NAD+ → metabolic + cellular energy

KEY DIFFERENTIATOR

Daily dosing may provide more consistent metabolic effects. Strong hepatic focus makes it promising for liver disease.

Research Status: AstraZeneca. Phase 2 trials completed. Development focused on NASH and chronic kidney disease with type 2 diabetes.

LAB INSIGHT

"The daily option — providing consistent dual-receptor activation with strong liver benefits."

CHAPTER 3

GH / IGF-1 Modulators

The Hormonal Catalysts of Fat Loss

Growth hormone and its downstream mediators are fundamental to body composition. These peptides stimulate natural GH release, promoting lipolysis, preserving lean tissue, and enhancing recovery — without the risks of exogenous GH administration.

CJC-1295 (no DAC)

"The Precision Pulse"

GHRH Analog (Modified)

Stimulates pulsatile GH release from the pituitary gland by mimicking growth hormone-releasing hormone (GHRH) without prolonged GH elevation.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{152}H_{252}N_{44}O_{42}$	3367.97 Da	~30 minutes (without DAC)	8-12 weeks typical research cycles

AMINO SEQUENCE

Tyr-D-Ala-Asp-Ala-Ile-Phe-Thr-Gln-Ser-Tyr-Arg-Lys-Val-Leu-Ala-Gln-Leu-Ser-Ala-Arg-Lys-Leu-Leu-Gln-Asp-Ile-Leu-Ser-Arg-NH_2

MECHANISM OF ACTION

CJC-1295 (no DAC) is a modified GHRH analog with improved stability due to amino acid substitutions. It binds to GHRH receptors on pituitary somatotrophs, triggering GH secretion. Without the Drug Affinity Complex (DAC), it produces discrete GH pulses rather than sustained elevation, more closely mimicking physiological patterns. This pulsatile release is important because continuous GH exposure can cause receptor desensitization.

RESEARCH FINDINGS

- Significant increases in GH and IGF-1 levels in research subjects
- Enhanced fat oxidation and improved body composition

- Improved sleep quality (GH secretion is tied to sleep cycles)
- Synergistic effects when combined with GHRPs like Ipamorelin

RESEARCH STACK COMBINATIONS

- CJC-1295 + Ipamorelin □ complete GH axis stimulation (the 'classic stack')
- CJC-1295 + Tesamorelin □ enhanced GH pulse + visceral fat targeting
- CJC-1295 + MOTS-c □ hormonal + mitochondrial optimization

KEY DIFFERENTIATOR

Produces natural, pulsatile GH release rather than constant elevation. Mimics physiological GHRH patterns for safer, more sustainable effects.

Research Status: Research compound developed for GH deficiency studies. Not FDA-approved.

LAB INSIGHT

"The timer — sets the rhythm for natural GH secretion without overriding the body's feedback systems."

EXPLORE CJC-1295 (NO DAC) □ realpeptides.co/products/cjc-1295-no-dac/

Ipamorelin

"The Clean Secretagogue"

Growth Hormone Secretagogue (Ghrelin Mimetic)

Stimulates GH release through ghrelin receptor activation without affecting cortisol, prolactin, or appetite — the 'cleanest' GHRP available.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{38}H_{49}N_9O_5$	711.85 Da	~2 hours	8-12 weeks typical research cycles

AMINO SEQUENCE

Aib-His-D-2-Nal-D-Phe-Lys-NH_2

MECHANISM OF ACTION

Ipamorelin is a pentapeptide that selectively binds to the ghrelin receptor (GHS-R1a) on pituitary somatotrophs. Unlike other GHRPs (GHRP-2, GHRP-6, Hexarelin), it has minimal effect on cortisol and prolactin secretion — hormones that can interfere with fat loss and cause side effects. It also lacks the appetite-stimulating effects of GHRP-6, making it ideal for fat-loss research.

RESEARCH FINDINGS

- Dose-dependent GH release without cortisol or prolactin elevation
- Improved body composition in animal models
- Enhanced recovery and sleep quality in research observations
- Synergistic GH amplification when combined with GHRH analogs

RESEARCH STACK COMBINATIONS

- Ipamorelin + CJC-1295 (no DAC) □ the 'gold standard' GH stack
- Ipamorelin + Tesamorelin □ clean GH release + visceral fat targeting
- Ipamorelin + AOD-9604 □ GH pulse + direct lipolysis

KEY DIFFERENTIATOR

The most selective GHRP — stimulates GH without the hormonal 'noise' of cortisol, prolactin, or appetite changes. Allows precise GH axis support.

Research Status: Developed by Novo Nordisk; extensively studied in clinical trials for GH deficiency and bone health.

LAB INSIGHT

"The gentleman of GH secretagogues — gets the job done without side effects that derail research protocols."

EXPLORE IPAMORELIN □ realpeptides.co/products/ipamorelin/

Tesamorelin

"The Visceral Fat Target"

GHRH Analog (FDA-Approved)

Selectively reduces visceral adipose tissue through enhanced GH secretion — the only FDA-approved peptide specifically for fat reduction.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{221}H_{366}N_{72}O_{67}S$	5135.89 Da	26-38 minutes	12-26 weeks in clinical trials

MECHANISM OF ACTION

Tesamorelin is a modified GHRH(1-44) with a trans-3-hexenoic acid group that improves stability. It stimulates pituitary GH release, which increases IGF-1 and promotes lipolysis — particularly in visceral fat. The visceral fat selectivity may relate to regional differences in GH receptor sensitivity and beta-adrenergic responsiveness in different fat depots.

RESEARCH FINDINGS

- FDA-approved for HIV-associated lipodystrophy — the only approved indication
- 11.8% reduction in trunk fat at 26 weeks vs 0.4% placebo (pivotal trial)
- Reduces liver fat in NAFLD patients (investigational)
- Improves metabolic markers without significant effects on peripheral fat

RESEARCH STACK COMBINATIONS

- Tesamorelin + Ipamorelin → GHRH + GHRP synergy for enhanced GH pulse
- Tesamorelin + AOD-9604 → visceral + subcutaneous fat targeting
- Tesamorelin + MOTS-c → hormonal + mitochondrial fat oxidation

KEY DIFFERENTIATOR

The only peptide FDA-approved for fat reduction. Specifically targets dangerous visceral fat rather than general weight loss.

Research Status: Theratechnologies. FDA-approved 2010 for HIV lipodystrophy. Studied for NAFLD and general obesity.

LAB INSIGHT

"The precision instrument — targets the belly fat that matters most for metabolic health."

EXPLORE TESAMORELIN → realpeptides.co/products/tesamorelin-peptide/

GHRP-2

"The Classic Secretagogue"

Growth Hormone Releasing Peptide

Potent GH release through ghrelin receptor activation with moderate effects on cortisol and appetite.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{45}H_{55}N_9O_6$	817.97 Da	~25-30 minutes	8-12 weeks typical research cycles

AMINO SEQUENCE

D-Ala-D-2-Nal-Ala-Trp-D-Phe-Lys-NH_2

MECHANISM OF ACTION

GHRP-2 is a synthetic hexapeptide that strongly activates ghrelin receptors on pituitary somatotrophs. It produces robust GH release — stronger than Ipamorelin — but also has effects on cortisol and prolactin (though less than GHRP-6). The moderate appetite stimulation may be beneficial for research subjects requiring caloric intake.

RESEARCH FINDINGS

- Among the most potent GHRPs for GH release
- Moderate cortisol and prolactin elevation (less than GHRP-6)
- Significant IGF-1 increases in research subjects
- Well-studied since the 1990s with extensive data

RESEARCH STACK COMBINATIONS

- GHRP-2 + CJC-1295 □ classic high-output GH stack
- GHRP-2 + Tesamorelin □ potent GH + visceral targeting
- GHRP-2 + Ipamorelin □ balanced GH secretion

KEY DIFFERENTIATOR

The workhorse — provides robust GH release with an acceptable side-effect profile. A middle ground between Ipamorelin (mild) and Hexarelin (potent).

Research Status: Developed in the 1990s. Extensively studied. Research compound only.

LAB INSIGHT

"The reliable classic — decades of data supporting its GH-releasing efficacy."

EXPLORE GHRP-2 → realpeptides.co/products/ghrp-2/

GHRP-6

"The Appetite Activator"

Growth Hormone Releasing Peptide

Potent GH release with significant appetite stimulation — useful when caloric intake needs support.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{46}H_{56}N_{12}O_6$	873.01 Da	~20-30 minutes	8-12 weeks typical research cycles

AMINO SEQUENCE

His-D-Trp-Ala-Trp-D-Phe-Lys-NH_2

MECHANISM OF ACTION

GHRP-6 is a synthetic hexapeptide that activates ghrelin receptors with high potency. Beyond GH release, it significantly stimulates appetite through the same ghrelin pathway. It also raises cortisol and prolactin more than other GHRPs. The appetite effect can be advantageous for research subjects needing to maintain caloric intake during demanding protocols.

RESEARCH FINDINGS

- Strong GH secretion — comparable to GHRP-2
- Significant appetite stimulation (useful for muscle-building research)
- Notable cortisol and prolactin increases
- Extensive safety data from decades of research

RESEARCH STACK COMBINATIONS

- GHRP-6 + CJC-1295 → GH support + appetite when needed
- GHRP-6 + Ipamorelin → balanced appetite effects
- Not typically recommended for pure fat-loss protocols due to appetite effects

KEY DIFFERENTIATOR

The appetite stimulator — when maintaining food intake is as important as GH release. Less suitable for fat-loss-focused research.

Research Status: One of the first GHRPs developed. Extensively studied since the 1980s.

LAB INSIGHT

"The double-edged sword — powerful GH release but appetite effects may complicate fat-loss research."

EXPLORE GHRP-6 → realpeptides.co/products/ghrp-6/

Hexarelin

"The Potent Activator"

Growth Hormone Releasing Hexapeptide

The most potent GHRP for GH release — also has cardioprotective effects independent of GH.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{47}H_{58}N_{12}O_6$	887.04 Da	~70 minutes	4-8 weeks (desensitization may occur)

AMINO SEQUENCE

His-D-2-Me-Trp-Ala-Trp-D-Phe-Lys-NH_2

MECHANISM OF ACTION

Hexarelin is the most potent GHRP, producing the largest GH releases. Beyond GH effects, it has unique cardioprotective properties — it binds to CD36 receptors on cardiac tissue, independent of GH, providing direct myocardial benefits. However, rapid receptor desensitization can occur with continuous use, limiting its utility for long-term protocols.

RESEARCH FINDINGS

- Highest peak GH levels among GHRPs
- Cardioprotective effects independent of GH (CD36 pathway)
- Significant cortisol and prolactin elevation

- Desensitization occurs with continuous use — cycling required

RESEARCH STACK COMBINATIONS

- Hexarelin + CJC-1295 → maximum GH output (short-term)
- Hexarelin cycling with Ipamorelin → power + maintenance phases
- Hexarelin + cardiac research protocols → unique CD36 benefits

KEY DIFFERENTIATOR

The powerhouse — maximum GH release plus unique heart benefits. But desensitization limits long-term use.

Research Status: Studied for cardiac applications. Research compound.

LAB INSIGHT

"The heavy hitter — for short-term maximum GH or cardiac research, not sustained protocols."

EXPLORE HEXARELIN → realpeptides.co/products/hexarelin/

Sermorelin

"The Rebalancer"

GHRH Analog (Original)

Restores natural GH rhythm by stimulating pituitary GH release — the gentlest GHRH option with the longest safety record.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{149}H_{246}N_{44}O_{42}S$	3357.96 Da	10-20 minutes	3-6 months typical protocols

AMINO SEQUENCE

GHRH(1-29) with C-terminal amide

MECHANISM OF ACTION

Sermorelin is the original GHRH analog, representing the first 29 amino acids of natural GHRH. It binds to GHRH

receptors on the pituitary, stimulating GH release in a physiological pattern. Its short half-life means effects are gentle and transient — mimicking natural GHRH pulses. This makes it ideal for long-term protocols aimed at restoring GH rhythm rather than maximizing output.

RESEARCH FINDINGS

- Previously FDA-approved for GH deficiency diagnosis and treatment (discontinued for commercial reasons)
- Restores natural GH pulsatility in aging subjects
- Improves sleep quality — GH is released primarily during sleep
- Excellent long-term safety profile from clinical use

RESEARCH STACK COMBINATIONS

- Sermorelin + Ipamorelin gentle rhythm restoration
- Sermorelin + GHRP-2 enhanced but still physiological GH support
- Sermorelin solo for research focused on natural GH pattern restoration

KEY DIFFERENTIATOR

The original — gentlest approach to GH axis support. Ideal for long-term, physiological restoration rather than maximum GH output.

Research Status: Previously FDA-approved. Discontinued for commercial reasons (2008). Still available as research compound.

LAB INSIGHT

"The grandfather — the longest safety record and most physiological approach to GH support."

EXPLORE SERMORELIN realpeptides.co/products/sermorelin/

CHAPTER 4

Mitochondrial Optimization

The Cellular Engine of Fat Loss

Mitochondria are where fat is actually burned — the cellular furnaces that convert fatty acids into ATP. These peptides enhance mitochondrial function, efficiency, and protection, improving the body's fundamental capacity to oxidize fat.

MOTS-c

"The Exercise Mimetic"

Mitochondrial-Derived Peptide

Activates AMPK pathway to enhance glucose uptake, fat oxidation, and metabolic flexibility — mimicking the effects of exercise at the cellular level.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{102}H_{152}N_{28}O_{31}S_2$	2174.63 Da	~2-4 hours	6-12 weeks typical research cycles

AMINO SEQUENCE

MRWQEMGYIFYPRKLR

MECHANISM OF ACTION

MOTS-c is a 16-amino acid peptide encoded within the mitochondrial genome. It's released into circulation in response to metabolic stress and exercise. Once in target tissues, MOTS-c activates AMPK (the cellular 'energy sensor'), promoting glucose uptake, fatty acid oxidation, and mitochondrial biogenesis. It essentially tells cells to behave as if they're exercising — even at rest.

RESEARCH FINDINGS

- Prevents diet-induced obesity in mouse models
- Improves glucose tolerance and insulin sensitivity
- Enhances exercise capacity and endurance

- Declining MOTS-c levels associated with aging and metabolic disease

RESEARCH STACK COMBINATIONS

- MOTS-c + SS-31 → mitochondrial function + protection
- MOTS-c + AOD-9604 → cellular oxidation + fat mobilization
- MOTS-c + 5-Amino-1MQ → AMPK + NAD^+ dual activation

KEY DIFFERENTIATOR

The first identified 'exercise mimetic' peptide — provides metabolic benefits of exercise at the molecular level.

Research Status: Discovered at USC (Dr. Pinchas Cohen). No FDA approval. Research compound with growing evidence base.

LAB INSIGHT

"The exercise pill — the closest thing to bottled workout benefits for cellular metabolism."

EXPLORE MOTS-C → realpeptides.co/products/mots-c-peptide/

SS-31 (Elamipretide)

"The Mitochondrial Shield"

Mitochondria-Targeted Peptide

Stabilizes cardiolipin in the inner mitochondrial membrane, improving electron transport chain efficiency and reducing oxidative stress.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{32}H_{49}N_9O_5$	639.79 Da	~1-2 hours	4-12 weeks typical protocols

AMINO SEQUENCE

D-Arg-Dmt-Lys-Phe-NH_2 (Dmt = 2',6'-dimethyltyrosine)

MECHANISM OF ACTION

SS-31 is a small tetrapeptide that concentrates 1000-fold in mitochondria. It binds to cardiolipin — a phospholipid

essential for electron transport chain function — stabilizing its structure and optimizing ATP production. This reduces electron leak (which causes oxidative stress) while increasing energy output. It essentially makes mitochondria run more efficiently.

RESEARCH FINDINGS

- Improves mitochondrial respiration in multiple tissue types
- Reduces reactive oxygen species (ROS) production
- Enhances exercise tolerance in heart failure patients (clinical trials)
- Protects against ischemia-reperfusion injury

RESEARCH STACK COMBINATIONS

- SS-31 + MOTS-c mitochondrial protection + activation
- SS-31 + 5-Amino-1MQ cellular efficiency + NAD^+ support
- SS-31 + BPC-157 cellular + tissue protection

KEY DIFFERENTIATOR

The only peptide that directly targets mitochondrial structure. Improves the engine itself, not just the fuel.

Research Status: Stealth BioTherapeutics. Phase 3 trials for Barth syndrome and heart failure. Orphan drug designation.

LAB INSIGHT

"The mechanic — fixes and optimizes the mitochondrial machinery that powers fat burning."

EXPLORE SS-31 (ELAMIPRETIDE) realpeptides.co/products/ss-31-elamipretide/

Humanin

"The Longevity Signal"

Mitochondrial-Derived Peptide

Cytoprotective peptide that protects against oxidative stress, apoptosis, and metabolic dysfunction — with emerging roles in insulin sensitivity.

FORMULA	WEIGHT	HALF-LIFE	DURATION

$C_{113}H_{177}N_{33}O_{31}S_1$	2687.01 Da	~30 minutes	4-12 weeks in research protocols

AMINO SEQUENCE

MAPRGFSCLLLLTSEIDLPVKRRA

MECHANISM OF ACTION

Humanin is a 24-amino acid peptide encoded in the mitochondrial genome. It was discovered for its ability to protect neurons from Alzheimer's-related toxicity. Since then, research has revealed metabolic benefits: improved insulin sensitivity, protection against oxidative stress, and anti-apoptotic effects. It signals cellular resilience and longevity — increasingly linked to metabolic health.

RESEARCH FINDINGS

- Improves insulin sensitivity in animal models of diabetes
- Protects against oxidative stress and cellular death
- Circulating levels decline with age and correlate with metabolic health
- Protective effects in ischemia and neurodegenerative models

RESEARCH STACK COMBINATIONS

- Humanin + MOTS-c → dual mitochondrial peptides for cellular protection
- Humanin + SS-31 → comprehensive mitochondrial support
- Humanin + NAD^+ → longevity + metabolic optimization

KEY DIFFERENTIATOR

A longevity peptide with metabolic benefits — bridges the gap between aging research and fat-loss science.

Research Status: Academic research compound. Discovered at USC. No FDA approval or clinical development.

LAB INSIGHT

"The survivor — protects cells under metabolic stress while improving their energy processing."

EXPLORE HUMANIN → realpeptides.co/products/humanin-peptide/

BPC-157

"The Healer"

Body Protection Compound

Accelerates tissue healing, protects against damage, and supports gastrointestinal health — with emerging evidence for metabolic protection.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{62}H_{98}N_{16}O_{22}$	1419.53 Da	~4-6 hours (estimated)	4-8 weeks typical protocols

AMINO SEQUENCE

Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val

MECHANISM OF ACTION

BPC-157 is a 15-amino acid fragment of a protein found in gastric juice. It promotes healing through multiple pathways: upregulating growth factors (VEGF, EGF), modulating nitric oxide systems, and protecting mitochondria from damage. Its effects on the gut-brain axis may also influence metabolism. The compound accelerates recovery from injury — allowing more consistent training and activity.

RESEARCH FINDINGS

- Accelerates healing of tendons, ligaments, muscles, and gut
- Protects against NSAID-induced gut damage
- Neuroprotective and mood-stabilizing effects in animal models
- Promotes angiogenesis (new blood vessel formation)

RESEARCH STACK COMBINATIONS

- BPC-157 + TB-500 → the 'gold standard' healing stack
- BPC-157 + AOD-9604 → joint protection + fat loss support
- BPC-157 + MOTS-c → tissue + mitochondrial protection

KEY DIFFERENTIATOR

The most versatile healing peptide — supports the physical foundation that makes fat-loss activity possible.

Research Status: Research compound studied since the 1990s. No FDA approval. Extensive animal data, limited human studies.

LAB INSIGHT

"The repair crew — keeps the body functional and resilient during demanding research protocols."

EXPLORE BPC-157 → realpeptides.co/products/bpc-157-peptide/

Thymosin Alpha-1 (TA-1)

"The Immune Modulator"

Immunomodulatory Peptide

Balances immune function and reduces chronic inflammation — addressing immune-metabolic dysfunction that impairs fat loss.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{129}H_{215}N_{33}O_{55}$	3108.27 Da	~2 hours	4-12 weeks typical protocols

AMINO SEQUENCE

Ac-SDAAVDTSSEITTKDLKEKKEVVEEAEN

MECHANISM OF ACTION

Thymosin Alpha-1 is a 28-amino acid peptide naturally produced by the thymus gland. It modulates T-cell function, enhances dendritic cell maturation, and balances Th1/Th2 immune responses. Chronic inflammation — common in obesity — impairs metabolic function. TA-1 restores immune balance, potentially removing an inflammatory brake on metabolism.

RESEARCH FINDINGS

- FDA-approved in multiple countries for hepatitis and immune deficiency
- Reduces chronic inflammatory markers
- Improves immune response to vaccines and infections
- Emerging research on metabolic benefits in inflammatory states

RESEARCH STACK COMBINATIONS

- TA-1 + BPC-157 → immune + tissue healing
- TA-1 + MOTS-c → immune balance + metabolic enhancement
- TA-1 + NAD⁺ → immune + cellular optimization

KEY DIFFERENTIATOR

Addresses immune dysfunction that often accompanies metabolic disease — inflammation is a hidden brake on fat loss.

Research Status: FDA-approved in 35+ countries (not US) for hepatitis B/C and immune support. SciClone Pharmaceuticals.

LAB INSIGHT

"The balancer — when immune dysfunction is blocking metabolic progress."

EXPLORE THYMOSIN ALPHA-1 (TA-1) → realpeptides.co/products/thymosin-alpha-1-peptide/

ARA-290

"The Anti-Inflammatory Regulator"

Erythropoietin-Derived Peptide

Activates innate repair receptor (IRR) to reduce inflammation and neuropathy — without erythropoietin's blood effects.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{53}H_{92}N_{14}O_{15}$	1173.37 Da	~2 minutes (rapid)	4-8 weeks in clinical trials

AMINO SEQUENCE

pGlu-EQLERALNSS

MECHANISM OF ACTION

ARA-290 is an 11-amino acid peptide derived from the structure of erythropoietin. Unlike EPO, it doesn't affect red blood cell production. Instead, it activates the innate repair receptor (IRR), reducing inflammation and promoting tissue protection. It's particularly promising for neuropathy and chronic inflammatory conditions that impair metabolic function.

RESEARCH FINDINGS

- Phase 2 trials show improvements in diabetic neuropathy symptoms
- Reduces inflammatory markers in multiple conditions
- Tissue-protective effects without EPO's hematological effects
- Emerging interest for metabolic inflammation

RESEARCH STACK COMBINATIONS

- ARA-290 + TA-1 → dual anti-inflammatory approach
- ARA-290 + BPC-157 → neuroprotection + tissue healing
- ARA-290 + MOTS-c → inflammation control + metabolic support

KEY DIFFERENTIATOR

Targets the inflammatory-neuropathic axis common in metabolic disease — addresses pain and dysfunction that limit activity.

Research Status: Araim Pharmaceuticals. Phase 2 trials for diabetic neuropathy completed. Orphan drug designation for sarcoidosis.

LAB INSIGHT

"The nerve protector — when metabolic inflammation causes neuropathy and pain."

EXPLORE ARA-290 → realpeptides.co/products/ara-290/

SLU-PP-332

"The Exercise Amplifier"

ERRα/γ Agonist (Small Molecule)

Activates estrogen-related receptors (ERRs) to enhance mitochondrial function and exercise capacity — a chemical exercise mimetic.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{21}H_{22}FN_3O_3$	383.42 Da	~4-8 hours (estimated)	4-8 weeks in research protocols

MECHANISM OF ACTION

SLU-PP-332 is a small molecule (not technically a peptide) that activates ERRα and ERRγ — nuclear receptors that

control genes involved in mitochondrial biogenesis, oxidative metabolism, and exercise adaptation. Activation mimics the transcriptional changes that occur with endurance training, increasing fatigue resistance and metabolic capacity.

RESEARCH FINDINGS

- Increases running endurance by 50-70% in mice without training
- Enhances mitochondrial gene expression in muscle
- Improves metabolic markers independent of exercise
- Reduces muscle atrophy in immobilization models

RESEARCH STACK COMBINATIONS

- SLU-PP-332 + MOTS-c → dual exercise mimetic pathways
- SLU-PP-332 + SS-31 → ERR activation + mitochondrial protection
- SLU-PP-332 + AOD-9604 → exercise adaptation + fat mobilization

KEY DIFFERENTIATOR

Provides endurance and metabolic benefits of exercise through transcriptional activation — true 'exercise in a pill' potential.

Research Status: Academic research (Washington University, St. Louis). Pre-clinical stage. High interest from pharmaceutical companies.

LAB INSIGHT

"The trainer's substitute — activates exercise genes even when the body can't train."

EXPLORE SLU-PP-332 → realpeptides.co/products/slu-pp-332-peptide/

CHAPTER 5

Appetite & Satiety Regulators

The Control Mechanisms of Modern Metabolic Research

These compounds influence neurochemical and hormonal networks that control reward, hunger, and fullness — achieving less intake, better output, and stable energy through precise biological signaling.

Oxytocin

"The Emotional Regulator"

Neuroendocrine Peptide

Reduces food intake, especially reward-driven eating, while improving mood and stress resilience — the 'social hormone' with metabolic effects.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{43}H_{66}N_{12}O_{12}S_2$	1007.19 Da	~3-5 minutes (endogenous)	4-12 weeks in clinical research

AMINO SEQUENCE

Cys-Tyr-Ile-Gln-Asn-Cys-Pro-Leu-Gly-NH_2 (cyclic 1-6)

MECHANISM OF ACTION

Oxytocin acts on hypothalamic nuclei to reduce food intake, particularly high-calorie/reward foods. It also modulates stress response and social bonding. For metabolic research, oxytocin addresses emotional and reward-driven eating — a major driver of obesity that's resistant to willpower. The compound may also increase energy expenditure and fat oxidation through peripheral effects.

RESEARCH FINDINGS

- Intranasal oxytocin reduces caloric intake, especially from fats
- Decreases reward-driven eating behavior in human studies
- Improves glucose tolerance in some populations

- Reduces stress and cortisol — known metabolic disruptors

RESEARCH STACK COMBINATIONS

- Oxytocin + Tesofensine emotional + neurological appetite control
- Oxytocin + Cagrilintide psychological + physiological satiety
- Oxytocin + Selank stress reduction + mood stabilization

KEY DIFFERENTIATOR

Targets the emotional and social dimensions of eating — addresses why people eat, not just hunger signals.

Research Status: Investigational for obesity and binge eating. Approved for obstetric uses. Extensive safety data.

LAB INSIGHT

"The relationship hormone — fixes the emotional eating that willpower can't touch."

EXPLORE OXYTOCIN realpeptides.co/products/oxytocin/

PT-141 (Bremelanotide)

"The Reward Reset"

Melanocortin Receptor Agonist

Modulates central reward pathways through MC4R activation — FDA-approved for sexual dysfunction, with emerging evidence for appetite effects.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{50}H_{68}N_{14}O_{10}$	1025.17 Da	~2-3 hours	4-8 weeks in research protocols

AMINO SEQUENCE

Ac-Nle-cyclo[Asp-His-D-Phe-Arg-Trp-Lys]-OH (derived from Melanotan II)

MECHANISM OF ACTION

PT-141 is a cyclic heptapeptide derived from Melanotan II with improved selectivity for MC4R. MC4R activation in the

hypothalamus affects both sexual function and appetite — explaining why many GLP-1 users report libido changes. PT-141 primarily activates dopaminergic pathways, influencing motivation and reward. The metabolic implications are under investigation.

RESEARCH FINDINGS

- FDA-approved (Vyleesi) for hypoactive sexual desire disorder
- Activates central dopaminergic pathways
- MC4R pathway clearly linked to appetite regulation
- Research ongoing into direct metabolic effects

RESEARCH STACK COMBINATIONS

- PT-141 + Tesofensine → dopamine pathway optimization
- PT-141 + Oxytocin → reward + bonding pathway synergy
- PT-141 + Kisspeptin-10 → HPG axis + metabolic effects

KEY DIFFERENTIATOR

Targets the reward system that drives compulsive eating — a selective melanocortin agonist with FDA approval.

Research Status: Palatin Technologies. FDA-approved 2019 for HSDD. Emerging interest for metabolic applications.

LAB INSIGHT

"The reward rewirer — when dopamine dysfunction drives overeating."

EXPLORE PT-141 (BREMELANOTIDE) → realpeptides.co/products/pt-141-bremelanotide/

Kisspeptin-10

"The Metabolic Switch"

Hypothalamic Neuropeptide

Regulates the HPG axis (reproductive hormones) with emerging evidence for direct metabolic effects on glucose and appetite.

FORMULA	WEIGHT	HALF-LIFE	DURATION

$C_{63}H_{83}N_{17}O_{14}$	1302.46 Da	~4-5 minutes	4-8 weeks in research protocols

AMINO SEQUENCE

Tyr-Asn-Trp-Asn-Ser-Phe-Gly-Leu-Arg-Phe-NH_2

MECHANISM OF ACTION

Kisspeptin-10 is the active fragment of the larger kisspeptin protein, acting through the KISS1R (GPR54) receptor. It's the master regulator of the HPG axis — controlling release of GnRH, LH, and FSH. Beyond reproduction, kisspeptin influences glucose homeostasis, insulin secretion, and appetite. Metabolic status affects fertility, and these pathways are linked.

RESEARCH FINDINGS

- Regulates reproductive hormones in both sexes
- Improves glucose-stimulated insulin secretion
- Affects food intake and body weight in animal models
- Links metabolic status to reproductive function

RESEARCH STACK COMBINATIONS

- Kisspeptin-10 + GH secretagogues hormonal optimization
- Kisspeptin-10 + PT-141 reproductive + reward pathways
- Kisspeptin-10 + MOTS-c hormonal + metabolic support

KEY DIFFERENTIATOR

Links the reproductive and metabolic systems — may be particularly relevant for metabolic dysfunction affecting fertility.

Research Status: Academic research compound. Investigational for infertility and metabolic disorders.

LAB INSIGHT

"The fertility-metabolism bridge — when hormonal dysfunction connects to metabolic problems."

EXPLORE KISSPEPTIN-10 realpeptides.co/products/kisspeptin-10/

Setmelanotide

"The MC4R Specialist"

Selective MC4R Agonist

Highly selective MC4R activation for genetic obesity syndromes — the proof-of-concept for melanocortin weight-loss therapy.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{56}H_{78}N_{16}O_{10}$	1117.33 Da	~11 hours	52+ weeks in clinical trials

AMINO SEQUENCE

Ac-Arg-Cys(1)-D-Ala-His-D-Phe-Arg-Trp-Cys(1)-NH_2

MECHANISM OF ACTION

Setmelanotide is a highly selective MC4R agonist that bypasses upstream genetic defects in the leptin-melanocortin pathway. For patients with POMC, PCSK1, or LEPR deficiency, it restores the satiety signal that's genetically broken. This demonstrates that MC4R activation alone can produce substantial weight loss — validating the pathway for broader obesity treatment.

RESEARCH FINDINGS

- FDA-approved for POMC, PCSK1, and LEPR deficiency obesity
- Mean 25% weight reduction in approved indications
- Normalizes hunger and reduces hyperphagia
- Proof that MC4R activation drives weight loss when pathway is intact

RESEARCH STACK COMBINATIONS

- Setmelanotide is typically used as monotherapy for genetic indications
- Research interest in MC4R activation + GLP-1 combinations
- May inform development of broader MC4R-targeting therapies

KEY DIFFERENTIATOR

The first approved MC4R agonist — validates melanocortin pathway for weight loss. Currently limited to rare genetic obesities.

Research Status: Rhythm Pharmaceuticals. FDA-approved 2020 for genetic obesity syndromes. Expanding indications under study.

LAB INSIGHT

"The pathway validator — proves that MC4R activation produces profound weight loss in the right genetic context."

CHAPTER 6

Supportive & Synergistic Peptides

The Amplifiers of Fat-Loss Research

These peptides enhance stress regulation, recovery, sleep, and focus — making other fat-loss pathways perform at their best. They address the foundation that enables metabolic optimization.

Semax

"The Cognitive Edge"

Nootropic / Neuroprotective Peptide

Enhances focus, memory, and mood during caloric restriction — maintains cognitive performance when energy is limited.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{37}H_{51}N_9O_{10}$	753.87 Da	~60 minutes	4-8 weeks typical protocols

AMINO SEQUENCE

Met-Glu-His-Phe-Pro-Gly-Pro

MECHANISM OF ACTION

Semax is a synthetic analog of ACTH(4-10) that enhances BDNF expression, modulates dopaminergic transmission, and protects neurons from stress. During caloric restriction, cognitive function often suffers. Semax maintains mental clarity and motivation, supporting adherence to demanding protocols.

RESEARCH FINDINGS

- Approved in Russia for cognitive enhancement and stroke recovery
- Increases BDNF and neurotrophic signaling
- Improves attention, memory, and stress resilience
- No significant side effects in clinical use

RESEARCH STACK COMBINATIONS

- Semax + Selank → cognitive + anxiolytic support
- Semax + MOTS-c → mental + metabolic optimization
- Semax + Tesofensine → neurotransmitter balance

KEY DIFFERENTIATOR

Maintains the cognitive performance that makes fat-loss protocols sustainable — addresses the mental side of metabolic research.

Research Status: Approved in Russia and Ukraine. Research compound elsewhere.

LAB INSIGHT

"The brain fuel — keeps you sharp when calories are low."

EXPLORE SEMAX → realpeptides.co/products/semax-amidate-peptide/

Selank Amidate

"The Calm Controller"

Anxiolytic Neuropeptide

Reduces anxiety and stress without sedation — addresses cortisol dysregulation that impairs fat loss.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{33}H_{57}N_{11}O_9$	751.88 Da	~2-3 hours	4-8 weeks typical protocols

AMINO SEQUENCE

Thr-Lys-Pro-Arg-Pro-Gly-Pro (tuftsin analog)

MECHANISM OF ACTION

Selank is a synthetic analog of tuftsin with added Pro-Gly-Pro. It modulates GABA-A receptor function, reduces cortisol, and enhances mood stability. Chronic stress and elevated cortisol promote fat storage (especially visceral) and muscle breakdown. Selank addresses this without the sedation or dependency of benzodiazepines.

RESEARCH FINDINGS

- Approved in Russia for anxiety and neurasthenia
- Reduces anxiety comparable to benzodiazepines without sedation
- Normalizes cortisol in stressed subjects
- Immunomodulatory benefits

RESEARCH STACK COMBINATIONS

- Selank + Semax anxiolytic + cognitive enhancement
- Selank + Oxytocin stress + emotional regulation
- Selank + Tesamorelin cortisol control + GH support

KEY DIFFERENTIATOR

Addresses the stress-cortisol axis that derails fat loss — calm without sedation or impairment.

Research Status: Approved in Russia. Research compound elsewhere.

LAB INSIGHT

"The chill pill — when stress hormones are sabotaging your metabolism."

EXPLORE SELANK AMIDATE realpeptides.co/products/selank-amidate-peptide/

NAD+

"The Cellular Currency"

Nicotinamide Adenine Dinucleotide

Essential cofactor for cellular energy production, DNA repair, and sirtuin activation — declines with age and metabolic stress.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{21}H_{27}N_7O_{14}P_2$	663.43 Da	Variable (depends on form and route)	4-12 weeks typical protocols

MECHANISM OF ACTION

NAD+ is required for mitochondrial electron transport, glycolysis, and hundreds of enzymatic reactions. It activates sirtuins (especially SIRT1), which enhance fat oxidation and mitochondrial biogenesis. NAD+ levels decline with age and obesity, impairing cellular metabolism. Supplementation restores these functions.

RESEARCH FINDINGS

- Improves mitochondrial function and exercise capacity
- Activates SIRT1 and other longevity pathways
- Enhances insulin sensitivity in animal models
- Multiple forms available: IV, sublingual, precursors (NR, NMN)

RESEARCH STACK COMBINATIONS

- NAD+ + 5-Amino-1MQ → dual NAD⁺ optimization
- NAD+ + MOTS-c → cellular energy + AMPK activation
- NAD+ + SS-31 → NAD⁺ + mitochondrial membrane support

KEY DIFFERENTIATOR

The fundamental fuel — everything else works better when NAD+ is optimized.

Research Status: Available as supplement and IV therapy. Precursors (NR, NMN) widely available.

LAB INSIGHT

"The foundation — without adequate NAD+, metabolic optimization hits a ceiling."

EXPLORE NAD+ → realpeptides.co/products/nad-100mg/

GHK-Cu

"The Regenerative Messenger"

Copper Tripeptide

Promotes tissue repair, collagen synthesis, and antioxidant response — with emerging evidence for metabolic signaling.

FORMULA	WEIGHT	HALF-LIFE	DURATION

$C_{14}H_{24}N_6O_4Cu$	403.92 Da	~30-60 minutes	4-12 weeks typical protocols

AMINO SEQUENCE

Gly-His-Lys (with copper ion)

MECHANISM OF ACTION

GHK-Cu is a naturally occurring tripeptide that binds copper with high affinity. It promotes wound healing, collagen synthesis, and stem cell activity. Beyond aesthetics, GHK-Cu modulates gene expression for hundreds of genes involved in tissue repair and may support metabolic tissue health.

RESEARCH FINDINGS

- Accelerates wound healing and tissue regeneration
- Enhances skin elasticity and appearance
- Activates stem cell and tissue repair pathways
- Anti-inflammatory and antioxidant effects

RESEARCH STACK COMBINATIONS

- GHK-Cu + BPC-157 → comprehensive tissue support
- GHK-Cu + TB-500 → regeneration + repair
- GHK-Cu + NAD+ → tissue + cellular optimization

KEY DIFFERENTIATOR

Supports the tissue health that enables activity — when the body is falling apart, fat loss stalls.

Research Status: Available topically and as research peptide. Studied for wound healing.

LAB INSIGHT

"The beauty signal that goes deeper — tissue quality matters for metabolic function."

EXPLORE GHK-CU → realpeptides.co/products/ghk-cu-copper-peptide/

KPV

"The Inflammation Tamer"

Anti-Inflammatory Tripeptide

Potent anti-inflammatory derived from alpha-MSH — reduces inflammation that impairs metabolic function.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{16}H_{30}N_6O_4$	386.45 Da	~30 minutes	4-8 weeks typical protocols

AMINO SEQUENCE

Lys-Pro-Val

MECHANISM OF ACTION

KPV is the C-terminal tripeptide of alpha-MSH with concentrated anti-inflammatory activity. It reduces pro-inflammatory cytokines (TNF-α, IL-6, IL-1β) without melanocortin receptor activation. Chronic inflammation drives metabolic dysfunction — KPV addresses this at the molecular level.

RESEARCH FINDINGS

- Reduces inflammatory markers in colitis and arthritis models
- Inhibits NF-κB signaling
- Promotes wound healing
- Well-tolerated with minimal systemic effects

RESEARCH STACK COMBINATIONS

- KPV + BPC-157 → gut + tissue inflammation control
- KPV + TA-1 → dual immune modulation
- KPV + MOTS-c → inflammation control + metabolic support

KEY DIFFERENTIATOR

Concentrated anti-inflammatory without melanocortin side effects — targets the inflammation that blocks fat loss.

Research Status: Research compound. Studied for inflammatory bowel disease and wound healing.

LAB INSIGHT

"The fire extinguisher — when chronic inflammation is the hidden brake on metabolism."

EXPLORE KPV → realpeptides.co/products/kpv-5mg/

TB-500

"The Tissue Architect"

Thymosin Beta-4 Fragment

Accelerates tissue repair through cell migration and angiogenesis — the systemic healing peptide.

FORMULA	WEIGHT	HALF-LIFE	DURATION
$C_{212}H_{350}N_{56}O_{78}S$	4963 Da	~2-3 hours	4-12 weeks typical protocols

AMINO SEQUENCE

LKKTETQ (active fragment of thymosin beta-4)

MECHANISM OF ACTION

TB-500 is a fragment of thymosin beta-4 that promotes cell migration, angiogenesis, and tissue repair. It upregulates actin — the protein that allows cells to move and rebuild tissue. By improving blood vessel formation and tissue regeneration, it supports the physical infrastructure that enables activity.

RESEARCH FINDINGS

- Accelerates healing of muscles, tendons, and ligaments
- Promotes cardiac tissue repair after injury
- Enhances angiogenesis (new blood vessel formation)
- Reduces inflammation and scar tissue

RESEARCH STACK COMBINATIONS

- TB-500 + BPC-157 → the 'gold standard' healing combination
- TB-500 + GHK-Cu → systemic + local tissue support
- TB-500 + MOTS-c → tissue repair + metabolic optimization

KEY DIFFERENTIATOR

The systemic healer — works throughout the body to repair and regenerate tissues that fat-loss activity demands.

Research Status: Research compound. Extensively used in veterinary medicine. Human data limited.

LAB INSIGHT

"The builder — keeps the physical machine running when demanding protocols break it down."

EXPLORE TB-500 → realpeptides.co/products/tb-500-thymosin-beta-4/

CHAPTER 7

Research Guidelines & Best Practices

The Framework for Credible, Controlled, and Repeatable Research

1. Storage & Stability

Proper storage is critical for maintaining peptide integrity:

- Lyophilized (freeze-dried) peptides: Store at -20°C or below, protected from light, in sealed vials until ready for use
- Reconstituted peptides: Refrigerate at 2-8°C, use within 2-4 weeks maximum
- Never freeze reconstituted peptides — freeze-thaw cycles destroy peptide bonds
- Protect from heat, light, and humidity at all stages
- Label everything: compound name, concentration, reconstitution date, expiration

Pro Tip: Use amber vials or wrap clear vials in foil to protect from light degradation.

2. Reconstitution Protocol

Standard Supplies:

- Bacteriostatic water (BAC water) — preferred for multi-use vials
- Sterile water — for single-use applications
- Insulin syringes (28-31 gauge) for precise measurement
- Alcohol wipes for sterilization

Procedure:

- Allow vial to reach room temperature before opening
- Wipe vial tops with alcohol and allow to dry
- Draw appropriate volume of BAC water into syringe
- Inject slowly along the glass wall — never spray directly onto powder
- Allow natural dissolution — gently swirl if needed, never shake
- Record: compound, reconstitution volume, concentration, date

Example Calculation: 10 mg vial + 2 mL BAC water = 5 mg/mL concentration. Each 0.1 mL (10 units on insulin syringe) = 500 mcg.

3. Dosing Accuracy & Documentation

- Use insulin syringes with 0.5 mL or 1.0 mL capacity for precision
- Measure by volume (mL), not by insulin units — units vary by syringe
- Calculate: Desired dose ÷ Concentration = Volume to draw
- Maintain detailed research logs: peptide, dose, timing, fasting state, observations
- Never mix peptides in the same vial without confirmed compatibility data

4. Timing Considerations by Category

GH/GHRP Peptides:

- Half-life: 30 minutes - 2 hours
- Optimal timing: AM upon waking (fasted) + before sleep
- Avoid within 2-3 hours of high-fat meals

GLP-1/GIP Analogs:

- Half-life: 5-7 days
- Weekly administration
- Consistent day and time each week

Mitochondrial Peptides:

- Half-life: 1-4 hours
- Pre-workout or morning administration
- Can be combined with fasted protocols

5. Safety & Integrity Principles

All research must follow laboratory safety protocols:

- Use appropriate personal protective equipment (gloves, lab coat)
- Dispose of sharps in approved biohazard containers
- Never reuse syringes or needles
- Store compounds away from food and accessible areas
- Maintain chain of custody documentation

Disclaimer: All materials are for in-vitro research and laboratory use only. Not for human consumption.

CHAPTER 8

Advanced Stacks & Synergies

Where Science Meets Strategy — The 5-Tier Stacking Hierarchy

The 5-Tier Stacking Hierarchy Framework

Build protocols systematically, starting with core mechanisms and adding layers for specific goals:

TIER 1 — Core Drivers (Pick 1-2):

The foundation — direct fat-loss mechanisms

- Retatrutide, Tirzepatide, or Semaglutide — GLP-1 pathway
- AOD-9604 — direct lipolysis
- Cagrilintide — amylin pathway

TIER 2 — Hormonal Amplifiers (Pick 1-2):

Optimize hormonal environment for fat loss

- CJC-1295 + Ipamorelin — GH axis support
- Tesamorelin — visceral fat targeting
- Kisspeptin-10 — hormonal optimization

TIER 3 — Behavioral Regulators (Pick 1-2):

Address the psychology of eating

- Tesofensine — appetite/motivation
- Oxytocin — emotional eating
- PT-141 — reward pathways
- Semax/Selank — cognitive/stress support

TIER 4 — Regenerative Support (Pick 1-2):

Keep the body functional during demanding protocols

- BPC-157 + TB-500 — tissue repair
- GHK-Cu — regeneration
- KPV — inflammation control
- TA-1 — immune support

TIER 5 — Mitochondrial Masters (Pick 1-2):

Enhance the cellular machinery of fat burning

- MOTS-c — exercise mimetic
- 5-Amino-1MQ — NAD^+/NNMT modulation
- SS-31 — mitochondrial protection

Example Protocol Stacks

1. The GLP-1 + Mitochondrial Stack

Semaglutide or Tirzepatide + MOTS-c + SS-31

Purpose: Appetite suppression + metabolic enhancement at cellular level

> *"Control intake, amplify output — the metabolic double helix."*

2. The Fat Fragment + GH Synergy Stack

AOD-9604 + CJC-1295 (no DAC) + Ipamorelin

Purpose: Direct lipolysis + optimized GH pulsatility

> *"Mobilize fat by day, recover and rebuild by night."*

3. The Endurance & Energy Stack

MOTS-c + SLU-PP-332 + NAD+

Purpose: Mitochondrial performance for those prioritizing activity

> *"The cellular equivalent of endurance training."*

4. The Cognitive Compliance Stack

Tesofensine + Semax + Selank

Purpose: Maintain focus and reduce stress during caloric restriction

> *"Because mindset drives metabolism."*

5. The Regenerative Fat-Loss Stack

BPC-157 + TB-500 + GHK-Cu + KPV

Purpose: Healing and anti-inflammatory support during demanding protocols

"Recover faster, perform longer."

6. The Maximum Efficacy Stack

Retatrutide + Cagrilintide + MOTS-c + BPC-157

Purpose: Approaching surgical-level outcomes with pharmaceutical intervention

"The ceiling breaker — for research aiming at maximum effect."

CHAPTER 9

FAQ & Troubleshooting

Answers to Common Research Questions

Q1: Why isn't my peptide dissolving?

Roll the vial gently between your palms — never shake. If refrigerated, allow to reach room temperature first. Most peptides dissolve within 5-10 minutes. If cloudiness persists, the peptide may be degraded.

Q2: What causes cloudiness in the reconstituted vial?

Cloudiness indicates potential contamination, oxidation, or precipitation. Do not use cloudy solutions — discard and use a fresh vial. Proper storage and sterile technique prevent this.

Q3: How long is reconstituted peptide stable?

2-4 weeks when refrigerated at 2-8°C using bacteriostatic water. Sterile water preparations should be used within 48-72 hours. Never freeze reconstituted peptides.

Q4: Can peptides be mixed in one syringe?

Only if pH, solubility, and stability data confirm compatibility. CJC-1295 + Ipamorelin are commonly combined. When uncertain, administer separately to avoid potential degradation or precipitation.

Q5: Do peptide protocols require cycling?

Depends on the compound:

- GHRPs (especially Hexarelin): Yes — receptor desensitization occurs. 4-8 weeks on, 2-4 weeks off.
- GHRH analogs (CJC-1295, Sermorelin): Less critical, but periodic breaks recommended.
- GLP-1 analogs: No cycling required — designed for continuous use.
- Mitochondrial peptides: Generally no cycling needed.

Q6: Why does fatigue increase early in mitochondrial peptide research?

Mitochondrial optimization can temporarily increase ROS (reactive oxygen species) as cells upregulate fat oxidation. This adaptation typically resolves within 1-2 weeks. Ensure adequate antioxidant support and hydration.

Q7: What's the difference between GHRH and GHRP peptides?

- GHRH analogs (CJC-1295, Sermorelin, Tesamorelin): Stimulate GH release by binding GHRH receptors. Set the timing/rhythm.
- GHRPs (Ipamorelin, GHRP-2, GHRP-6, Hexarelin): Stimulate GH via ghrelin receptors. Amplify the signal.
- Best together: GHRH + GHRP creates synergistic GH release greater than either alone.

Q8: Why do GLP-1 agonists cause nausea?

GLP-1 slows gastric emptying and affects central appetite circuits. Nausea typically improves with gradual dose escalation over weeks. Start low and increase slowly. Avoid large, fatty meals during titration.

Q9: Can peptides be used with other medications?

Potential interactions exist, particularly with:

- Diabetes medications — GLP-1s can cause hypoglycemia with insulin/sulfonylureas
- Thyroid medications — some peptides affect thyroid function
- Blood thinners — BPC-157 may affect coagulation
- Always document all compounds in research protocols

Q10: How do I know if a peptide is working?

Objective markers to track:

- Body composition: DEXA, body-fat percentage, waist circumference
- Metabolic markers: Fasting glucose, HbA1c, lipid panel
- Hormones: IGF-1, insulin, leptin (where applicable)
- Subjective: Appetite, energy, sleep quality, recovery

Document baseline values before starting any protocol. Measure at consistent intervals.

Key Insight

The best research results come from discipline, not chance. Peptides are precision tools — not shortcuts. Consistent application of proper protocols, documentation, and objective measurement separates

meaningful research from guesswork.

CHAPTER 10

Glossary, Reference & Final Notes

Core Terminology

- Peptide — A short chain of amino acids (2-50) linked by peptide bonds; larger chains are proteins
- Lipolysis — The breakdown of triglycerides into free fatty acids and glycerol for energy use
- Lipogenesis — The synthesis and storage of fat from excess energy substrates
- Mitochondria — Cellular organelles that oxidize fatty acids and produce ATP; the "powerhouses" of cells
- AMPK — AMP-activated protein kinase; master metabolic sensor that promotes fat oxidation when energy is low
- GH / IGF-1 Axis — The growth hormone pathway affecting body composition, metabolism, and tissue repair
- GLP-1 / GIP / Glucagon — Incretin hormones regulating appetite, insulin, and energy expenditure
- Reconstitution — The process of adding bacteriostatic water to lyophilized (freeze-dried) peptide powder
- Half-Life — Time for compound concentration to reduce by 50%; determines dosing frequency
- Secretagogue — A substance that stimulates secretion of a hormone (e.g., GH secretagogue)
- Agonist — A compound that binds to and activates a receptor (opposite: antagonist)
- Cytoprotection — Protection of cells against damage, stress, or death

Key Pathways Reference

GLP-1 Pathway: Reduces appetite → slows gastric emptying → improves insulin sensitivity → promotes satiety

GH/IGF-1 Axis: Pituitary GH release → liver IGF-1 production → lipolysis + tissue repair + lean mass support

AMPK Pathway: Energy sensor activation → increased fat oxidation → glucose uptake → mitochondrial biogenesis

Melanocortin Pathway: MC4R activation → appetite suppression → energy expenditure increase

Top 10 Research Rules — The Real Peptides Code

1. Test, Don't Guess. Data over dogma — always validate with objective measurement.
2. Respect the Molecule. Purity, storage, and handling define the accuracy of your results.
3. Build, Don't Stack. Add one compound at a time, document effects, then evolve your protocol.
4. Control Variables. Sleep, nutrition, training, and stress all influence outcomes.
5. Document Everything. A forgotten entry is a week of progress lost — maintain detailed logs.
6. Prioritize Mitochondria. They power every metabolic pathway — healthy mitochondria, healthy

metabolism.

7. Balance Stress and Recovery. Chronic cortisol elevation sabotages fat loss — manage stress actively.
8. Think Systems, Not Symptoms. Fat loss is metabolic communication — address root causes.
9. Never Chase Shortcuts. Precision beats intensity — sustainable results require sustainable methods.
10. Science That Can't Be Silenced. Real data speaks louder than marketing — trust evidence.

Real Peptides Brand Philosophy

At Real Peptides, science isn't just a product — it's a standard. Every compound we discuss is sourced from verified synthesis partners, tested for >99% purity via HPLC & mass spectrometry, and stored under strict environmental controls.

We exist to bring transparency, accuracy, and integrity back to the peptide industry — one researcher at a time.

Author's Closing Note

Every peptide inside these pages represents not just a molecule — but a mindset:

To seek truth, not trend. To measure, not assume. To build from data, not desire.

Fat loss isn't the goal. Mastery is.

The compounds discussed here are the most promising tools available for metabolic research. But tools are only as good as the hands that wield them. Your discipline, your documentation, your commitment to truth — that's what transforms potential into results.

Stay curious. Stay disciplined. And above all — keep testing.

— Real Peptides Research Team

Science That Can't Be Silenced.

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