THEPEPTIDELIST.COM

The Complete Peptide Protocol Guide

Evidence-based dosing, monitoring, and pharmacogenomics for 25 therapeutic peptides. A genetics-first clinical reference for practitioners and patients.

2026 EDITION · VERSION 1.0

How to Use This Guide

This is a **clinical reference**, not a textbook. Every peptide follows the same template so you can find what you need in seconds.

Who This Is For

- Consumers researching peptide options
- Clinicians prescribing peptide therapy
- Coaches and health practitioners
- Anyone tired of Reddit bro-science

What Each Page Contains

- Protocol box (dose, route, frequency, cycle)
- FDA status + evidence grade
- Key clinical trial results
- Bloodwork monitoring schedule
- Side effects (tiered by severity)
- The Genetic Variable (SNPs that matter)
- Best fit / not ideal guidance

Evidence Grades

A: STRONG EVIDENCE	Large RCTs, FDA-approved indications, robust human data
B: MODERATE EVIDENCE	Phase 2/3 trials with meaningful results; moderate human evidence
C: EARLY/MIXED	Early human data, small trials, mixed results, or off-label extrapolation
D: PRECLINICAL	Preclinical only (animal/cell studies). No controlled human trials.

Regulatory Status Badges

FDA APPROVED	Approved for at least one indication. Prescription required.
PHASE 3	In pivotal trials. Not yet approved. Clinical trial access only.
COMPOUNDABLE (CAT 1)	Legal to compound at 503A/503B pharmacies with a prescription.
RESEARCH ONLY	Not approved for human use. Available as research chemical.

IMPORTANT DISCLAIMER

This guide is for **educational purposes only** and does not constitute medical advice. Many peptides listed here are investigational and not FDA-approved. Dosing information reflects commonly discussed protocols in clinical and research literature, not individualized treatment recommendations. Always work with a qualified healthcare provider before starting any peptide protocol. Do not self-treat.

Start Here: Find Your Protocol

Not sure where to begin? Follow the arrows. Each path leads to specific peptide pages in this guide.

What is your primary goal?

I want to lose weight →

Weight Loss / GLP-1

Start with: Semaglutide (FDA approved, strongest evidence)
If cost is a barrier: Liraglutide (shorter acting, established)
Cutting edge: Tirzepatide (dual agonist) or Retatrutide (triple)
Coming soon: CagriSema (sema + amylin analog)

I want to build / preserve muscle →

Growth Hormone

Best starter: Ipamorelin + CJC-1295 (gold standard combo)
Gentler option: Sermorelin (shorter acting, fewer sides)
Oral convenience: MK-677 (not a peptide, oral, watch glucose)
Medical HRT: HGH 191aa (prescription, expensive, proven)

I want sharper cognition →

Cognitive / Neuroprotection

Low risk starter: Selank (anxiolytic + focus, intranasal)
Nootropic: Semax (BDNF booster, intranasal)
Serious neuro: Cerebrolysin (stroke/TBI, IV only)
Frontier: Dihexa (extreme potency, no human data)

I'm recovering from an injury →

Healing & Recovery

Go-to stack: BPC-157 + TB-500 ('Wolverine Stack')
Skin/topical: GHK-Cu (collagen, copper peptide)
Systemic repair: TB-500 alone (inflammation + tissue)

Hormones / sexual health →

Sexual Health & Hormones

Libido (on-demand): PT-141 (melanocortin, works centrally)
TRT fertility support: Gonadorelin or HCG
Testicular preservation: HCG (on TRT)

Longevity / anti-aging →

Anti-Aging & Immune

Telomeres: Epitalon (course-based, low side effects)
Mitochondria: MOTS-c (exercise mimetic)
Cellular energy: NAD+ (IV or oral precursors)
Zombie cells: FOXO4-DRI (senolytic, preclinical)
Immune: Thymosin Alpha-1 / LL-37 / KPV (gut)

Every path above has a genetic variable.

The same peptide works differently depending on your SNPs. Each peptide page in this guide includes a **Genetic Variable** section showing which genes matter. Your DNA report at **thepeptidelist.com/genetics** maps all of them.

Why Most Peptide Information is Garbage

There are over 130 peptides being discussed in clinics, forums, and Telegram groups right now. The information landscape looks like this:

Reddit & Forums

Anecdotal dosing. "I ran 500mcg BPC and my knee feels great." No bloodwork. No controls. No idea if it was the peptide, the rest, or placebo.

Vendor Websites

Marketing dressed as science. Cherry-picked studies. Dosing guides designed to maximize product sales, not outcomes. Anonymous authors.

What You Need

Evidence-graded protocols. Named clinical trials. Monitoring panels. Contraindications. And the part nobody talks about: which genetics change response.

The Problem Nobody Is Solving

Two people take the same peptide, same dose, same protocol. One responds beautifully. The other gets side effects and no results. The difference is not willpower or compliance. It is biology.

Genetic variants in receptor sensitivity, metabolic enzymes, and signaling pathways create a 30 to 70% variance in individual response to peptide therapy. This is well-established in pharmacogenomics for psychiatric medications, pain management, and oncology. The peptide space has ignored it entirely.

Until now. Every peptide in this guide includes a **Genetic Variable** section identifying the SNPs and pathways most likely to alter your response. This is the only peptide protocol reference that connects dosing to DNA.

For personalized genetic analysis across all 30 peptides, visit thepeptidelist.com/genetics

Master Quick Reference

All 30 peptides at a glance. Sorted by category.

Peptide	Primary Use	Route	Frequency	Evidence	FDA Status	Key Genes	Page
Semaglutide	Weight loss, T2D	SC / Oral	Weekly / Daily	A	Approved	GLP1R, MC4R	
Tirzepatide	Weight loss, T2D	SC	Weekly	A	Approved	GLP1R, GIPR	
Retatrutide	Weight loss	SC	Weekly	B	Phase 3	GLP1R, GCGR	
Liraglutide	Weight loss, T2D	SC	Daily	A	Approved	GLP1R, TCF7L2	
CagriSema	Weight loss	SC	Weekly	B	Phase 3	CALCR, GLP1R	
Ipamorelin	GH secretion	SC	2-3x daily	C	Cat 1	GHR, GHSR	
CJC-1295 no DAC	GH secretion	SC	2-3x daily	C	Cat 1	GHRHR, GHR	
Sermorelin	GH secretion	SC	Daily (5/7)	B	Approved*	GHRHR, SOCS2	
MK-677	GH/IGF-1	Oral	Daily	B	Research	GHSR, IGF1	
HGH 191aa	GH replacement	SC	Daily	A	Approved	GHR, IGF1R	
BPC-157	Tissue repair, GI	SC	2x daily	D	Cat 1	VEGF, NOS3	
TB-500	Tissue repair	SC	2x weekly	D	Cat 1	TMSB4X, ACTA2	
GHK-Cu	Skin, wound heal	SC/Topical	Daily	C	Cat 1	COL1A1, MMP2	
PT-141	Female HSDD	SC	As needed	A	Approved	MC4R, MC1R	
Gonadorelin	Fertility, TRT adj	SC	2-3x weekly	B	Approved*	GNRHR, FSHB	
HCG	Fertility, TRT adj	SC/IM	2-3x weekly	A	Approved	LHCGR, CYP19A1	
Selank	Anxiety, focus	Intranasal	2-3x daily	C	Cat 1	COMT, BDNF	
Semax	Cognition, stroke	Intranasal	2-3x daily	C	Approved (RU)	BDNF, NGF	
Cerebrolysin	Neuroregeneration	IV/IM	Daily	B	Approved (EU)	BDNF, APOE	
Dihexa	Cognition (exp.)	Oral	Daily	D	Research	MET, HGF	
Epitalon	Telomeres, sleep	SC	Daily (10-20d)	D	Cat 1	TERT, CLOCK	
MOTS-c	Metabolic aging	SC	2-3x weekly	D	Cat 1	AMPK, MT-RNR1	
NAD+	Cellular energy	IV / Oral	Varies	C	Supplement	NAMPT, SIRT1	
FOXO4-DRI	Senolytic	SC	EOD (3-6 doses)	D	Research	FOXO4, TP53	
LL-37	Antimicrobial	SC/Topical	Daily (5/7)	C	Research	VDR, CAMP	
KPV	Gut inflammation	SC / Oral	Daily	D	Research	MC1R, NFKB1	
Thymosin a-1	Immune modulation	SC	2x weekly	B	Cat 1	TLR2, HLA-A	

Approved* = Previously FDA-approved but may be discontinued as branded product. Still available via compounding. Evidence grades: A = large RCTs, B = moderate human data, C = early/mixed, D = preclinical only. Key Genes = primary pharmacogenomic targets. Full genetics analysis at thepeptidelist.com/genetics.

Reconstitution Cheat Sheet

The #1 question people ask when they get their first vial. Here is the math.

The Formula

Dose per tick mark = (Peptide amount in vial) / (Bacteriostatic water added in units)

A standard insulin syringe has **100 units (ticks)** per 1 mL.

Common Reconstitution Examples

Vial Size	Water Added	Per 1 unit (tick)	250 mcg dose	Notes
5 mg vial	2 mL (200 units)	25 mcg	10 units (ticks)	Most common for BPC-157, Ipamorelin, CJC-1295
5 mg vial	1 mL (100 units)	50 mcg	5 units (ticks)	More concentrated. Smaller injection volume.
10 mg vial	2 mL (200 units)	50 mcg	5 units (ticks)	Common for GHK-Cu, TB-500
10 mg vial	1 mL (100 units)	100 mcg	2.5 units (ticks)	Very concentrated. Precise syringe needed.
2 mg vial	1 mL (100 units)	20 mcg	12.5 units (ticks)	Typical for CJC-1295 DAC

Storage After Reconstitution

- Refrigerate at 2-8°C (36-46°F)
- Use within 2-4 weeks (varies by peptide)
- Never freeze reconstituted solution
- Protect from light
- Use bacteriostatic water (not sterile water) for multi-dose use

Common Mistakes

- Using sterile water instead of bacteriostatic water (no preservative = bacterial growth)
- Spraying water directly onto the powder (trickle down the side of the vial)
- Shaking the vial (swirl gently)
- Leaving reconstituted peptide at room temperature
- Using past the expiration window

Master Bloodwork Panel

Pull these before starting any protocol. Recheck at the intervals listed.

Universal Baseline (Every Protocol)

Marker	Why	Recheck
CBC with differential	Baseline immune/blood status	If symptoms arise
Comprehensive Metabolic Panel	Liver, kidney, electrolytes	4-6 weeks
Fasting Glucose + HbA1c	Glycemic baseline	12 weeks
Lipid Panel	Cardiovascular markers	12 weeks
hsCRP	Systemic inflammation	8-12 weeks

Weight Loss / GLP-1 Add-On

Marker	Notes
Thyroid (TSH, fT4)	GLP-1 class MTC warning
Amylase + Lipase	Pancreatitis screen
Fasting Insulin	Insulin sensitivity change

Growth Hormone Add-On

Marker	Notes
IGF-1	Primary GH marker. Recheck 4-6 wks
Fasting Insulin	GH affects glucose handling
Thyroid (TSH, fT3, fT4)	GH can suppress conversion

Hormones / Fertility Add-On

Marker	Notes
Total + Free Testosterone	Baseline reproductive axis
Estradiol (sensitive)	HCG raises E2
LH + FSH	Pituitary function
Semen Analysis	If fertility goal

Immune / Healing Add-On

Marker	Notes
ESR + CRP	Baseline inflammation
Vitamin D (25-OH)	Regulates LL-37 expression
Fecal Calprotectin	If gut protocol (KPV, BPC)

WEIGHT LOSS & METABOLIC HEALTH

Weight Loss & Metabolic Health

GLP-1 receptor agonists and next-generation metabolic peptides. The most evidence-rich category in the guide.

The GLP-1 agonist class has the strongest clinical evidence of any peptide category, with multiple large Phase 3 trials, FDA approvals, and cardiovascular outcome data. Response varies significantly by genetics: variants in GLP1R, MC4R, FTO, and TCF7L2 affect appetite suppression, weight loss magnitude, GI tolerability, and glycemic response.

Semaglutide

Ozempic (T2D) • Wegovy (Obesity) • Rybelsus (Oral)

FDA APPROVED | A: STRONG EVIDENCE

The most prescribed GLP-1 agonist. 15% average weight loss. Weekly injection or daily oral tablet.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC injection / Oral	~7 days	Weekly (SC) / Daily (oral)	Ongoing chronic therapy

PROTOCOL

Starting Dose: 0.25 mg/week SC (Ozempic/Wegovy) or 3 mg/day oral (Rybelsus)
Titration: Escalate every 4 weeks: 0.25 → 0.5 → 1.0 → 1.7 → 2.4 mg
Maintenance: 2.4 mg/week (Wegovy) or 2.0 mg/week (Ozempic)
Oral Protocol: Take fasted with ≤4 oz water, 30 min before food
Timing: Same day each week, any time of day (SC)
Duration: Ongoing. Weight regain typical upon discontinuation.

🧬 GENES THAT ALTER GLP-1 RESPONSE

GLP1R (rs6923761) – Receptor sensitivity. A/A genotype = stronger response to semaglutide.
MC4R (rs17782313) – Appetite regulation. Risk allele = weaker satiety signaling.
FTO (rs9939609) – Obesity predisposition. A allele = higher baseline BMI, may need higher dose.
TCF7L2 (rs7903146) – Incretin biology. T allele = impaired glucose response to GLP-1.

→ GLP1R A/A carriers may lose 3-5% more body weight
→ MC4R risk carriers may plateau earlier
→ FTO carriers benefit more from appetite suppression
→ TCF7L2 T carriers may need tirzepatide (dual mechanism) instead

Know your GLP1R and MC4R variants before choosing between semaglutide and tirzepatide. Get tested at thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· STEP 1: 14.9% mean weight loss vs 2.4% placebo at 68 weeks (1,961 patients)
· SELECT: 20% reduction in major cardiac events (HR 0.80, p<0.001, 17,604 patients)
· SUSTAIN-6: 26% MACE reduction, 39% non-fatal stroke reduction in T2D
· STEP 5: Weight loss maintained at 104 weeks with continued treatment

BLOODWORK

HbA1c + Fasting Glucose – Baseline, 12 weeks
Lipid Panel – Baseline, 12 weeks
Thyroid (TSH, fT4) – Baseline (MTC box warning)
Amylase + Lipase – Baseline (pancreatitis screen)
CMP (liver/kidney) – Baseline, 12 weeks

COMMON: Nausea (44%) · Diarrhea (30%) · Vomiting (24%) · Constipation (24%) · Decreased appetite

CAUTION: Pancreatitis (rare, monitor) · Gallbladder events · MTC risk (box warning) · Severe GI if escalated too fast

BEST FIT: BMI ≥30 (or ≥27 with comorbidity) · Insulin resistance / prediabetes · Appetite-driven weight gain · Cardiovascular risk reduction

NOT IDEAL: History of MTC or MEN2 · Severe GI conditions · History of pancreatitis · Underweight / eating disorders

STACKING

Often combined with exercise + protein optimization. Enobosarm (SARM) being trialed to prevent lean mass loss during GLP-1 therapy.

BOTTOM LINE

The gold standard for weight loss pharmacotherapy. Strong evidence, proven CV benefit. Response varies by genetics. Start low, titrate slow.

Tirzepatide

Mounjaro (T2D) • Zepbound (Obesity) • LY3298176

FDA APPROVED | A: STRONG EVIDENCE

First-in-class dual GIP/GLP-1 agonist. Up to 22.5% weight loss. Outperforms semaglutide in head-to-head trials.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~5 days	Weekly	Ongoing chronic therapy

PROTOCOL

Starting Dose: 2.5 mg/week
Titration: Escalate every 4 weeks: 2.5 → 5 → 7.5 → 10 → 12.5 → 15 mg
Maintenance: 5-15 mg/week (dose-dependent efficacy)
Timing: Same day each week, any time, with or without food
Missed Dose: Take within 4 days; otherwise skip to next scheduled dose
Storage: Refrigerate. Room temp ≤30°C up to 21 days if unopened.

🧬 DUAL-RECEPTOR GENETICS

GLP1R (rs6923761) – GLP-1 receptor arm response.
GIPR (rs10423928) – GIP receptor sensitivity. Key to dual mechanism advantage.
TCF7L2 (rs7903146) – Incretin signaling. Dual agonism may overcome TCF7L2-related GLP-1 resistance.
PCSK1 (rs6232) – Prohormone processing affecting GLP-1/GIP activation.

→ GIPR variants may explain why some patients respond better to tirzepatide than semaglutide
→ TCF7L2 T carriers may benefit more from dual mechanism
→ Patients with GLP1R low-response genotype may do better on tirzepatide

Tirzepatide may be the better choice for GLP-1 partial responders. Genetic testing can identify who benefits from dual agonism. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· SURMOUNT-1: 20.9% weight loss at 15 mg vs 3.1% placebo (72 weeks, 2,539 patients)
· SURMOUNT-1: 36% of 15 mg patients achieved ≥25% weight loss
· SURPASS-2: Superior to semaglutide 1 mg for HbA1c and weight loss (head-to-head)
· HbA1c reductions of 1.87-2.58% across doses in T2D trials

BLOODWORK

HbA1c + Fasting Glucose – Baseline, 12 weeks
Lipid Panel – Baseline, 12 weeks
Thyroid (TSH, fT4) – Baseline (MTC box warning)
Amylase + Lipase – Baseline
CMP – Baseline

COMMON: Nausea (up to 33%) · Diarrhea (23%) · Constipation (up to 25%) · Decreased appetite · Injection site reactions

CAUTION: Pancreatitis (rare) · Gallbladder events · MTC risk (box warning) · Hypoglycemia (with insulin)

BEST FIT: Suboptimal response to semaglutide · Type 2 diabetes + obesity · Higher baseline HbA1c (>8%) · Strong appetite-driven eating patterns

NOT IDEAL: MTC/MEN2 family history · Severe gastroparesis · Pregnancy/breast-feeding · Cost-sensitive (branded only currently)

STACKING

Not combined with other GLP-1 agonists. Ongoing trials combining with enobosarm and bimagrumab for lean mass preservation.

BOTTOM LINE

The most effective single-agent weight loss therapy available. Dual mechanism provides an edge over pure GLP-1 agonists, especially for partial responders.

Retatrutide

LY3437943 • Triple G (GIP/GLP-1/Glucagon)

PHASE 3 B: MODERATE EVIDENCE

Triple receptor agonist. 28.7% weight loss in Phase 3. The most powerful obesity drug in development.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~6 days (est.)	Weekly	48-68 weeks (trials)

PROTOCOL

Starting Dose: 0.5 mg/week
Titration: Step-wise escalation over 12 weeks to target dose
Target Dose: 8-12 mg/week
Timing: Same day each week, any time
FDA Timeline: Phase 3 readouts 2026. Earliest approval: mid-2027.
Access: Clinical trial only. Not available commercially.

🧬 TRIPLE RECEPTOR PHARMACOGENOMICS

GLP1R – GLP-1 arm response.
GIPR – GIP arm response (shared with tirzepatide).
GCGR (rs1801483) – Glucagon receptor. Third mechanism unique to retatrutide.
FTO – Higher baseline BMI may see larger absolute loss.

→ GCGR variants may influence the hepatic fat metabolism advantage
→ Glucagon arm provides liver-specific benefits not seen with GLP-1 alone
→ Triple mechanism may overcome resistance patterns in dual-agonist partial responders

The glucagon receptor adds a metabolic dimension absent in semaglutide and tirzepatide. GCGR pharmacogenomics are still emerging. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· TRIUMPH-4: 28.7% mean weight loss at 12 mg (68 weeks)
· 58.6% achieved ≥25% weight loss at 12 mg
· 39.4% achieved ≥30% weight loss at 12 mg
· Phase 2: HbA1c reductions up to 2.0% in diabetes cohort

BLOODWORK

HbA1c + Fasting Glucose – Baseline, 12 weeks
Lipid Panel – Baseline
CMP (liver enzymes) – Baseline, 12 weeks (glucagon component)
Thyroid (TSH, fT4) – Baseline
Amylase + Lipase – Baseline

COMMON: Nausea (43%) · Diarrhea (33%) · Vomiting (21%) · Constipation (25%) · Decreased appetite

CAUTION: Dysesthesia (21% at 12 mg, novel signal) · Higher discontinuation (18%) than other GLP-1s · MTC risk (GLP-1 class warning)

BEST FIT: Severe obesity (BMI ≥40) · Partial response to GLP-1 or dual agonists · Hepatic steatosis / fatty liver · Willing to accept investigational status

NOT IDEAL: Anyone wanting an approved medication now · Low GI tolerance · Mild overweight (limited data) · History of dysesthesia/neuropathy

STACKING

Not combined with other incretin therapies. Standalone use only in clinical trials.

BOTTOM LINE

The most powerful weight loss peptide ever tested. 28.7% mean loss. Not yet available outside trials. Watch for 2026-2027 approval.

Liraglutide

Victoza (T2D) • Saxenda (Obesity)

FDA APPROVED | A: STRONG EVIDENCE

First-generation daily GLP-1 agonist. 8% weight loss. Proven cardiovascular mortality benefit. Now available as generic.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~13 hours	Daily	Ongoing chronic therapy

PROTOCOL

Starting Dose: 0.6 mg/day (Saxenda)
Titration: Increase by 0.6 mg/week over 5 weeks
Maintenance: 3.0 mg/day (Saxenda) or 1.2-1.8 mg/day (Victoza)
Discontinue: If <4% weight loss after 16 weeks at 3.0 mg
Timing: Any time of day, independent of meals
Generic: First generic GLP-1 approved 2025. Lower cost option.

🧬 GLP-1 RECEPTOR PHARMACOGENOMICS

GLP1R (rs6923761) – Receptor sensitivity determines response magnitude.
TCF7L2 (rs7903146) – Incretin biology. T allele = weaker GLP-1 response.
SORT1 (rs646776) – Lipid metabolism. May influence CV benefit magnitude.

→ Same GLP1R genetics as semaglutide, but daily dosing = less room for dose optimization
→ Generic availability makes it the most cost-effective GLP-1 option
→ Better tolerated than semaglutide for some GI-sensitive patients due to shorter half-life

If you are a GLP1R strong responder who is cost-sensitive, generic liraglutide may be the best value. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· SCALE: 8.0% weight loss vs 2.6% placebo at 56 weeks
· LEADER: 13% MACE reduction, 22% CV death reduction, 15% all-cause mortality reduction
· SCALE Teens: 4.64% BMI reduction in adolescents
· 88% of prediabetic patients returned to normoglycemia (SCALE)

BLOODWORK

HbA1c + Fasting Glucose – Baseline, 12 weeks
Lipid Panel – Baseline
Thyroid (TSH, fT4) – Baseline
Amylase + Lipase – Baseline
CMP – Baseline

COMMON: Nausea (dose-dependent) · Diarrhea · Constipation · Headache · Injection site reactions

CAUTION: Pancreatitis (rare) · MTC risk (box warning) · Gallbladder events

BEST FIT: Cost-sensitive patients (generic available) · Those who prefer daily dosing control · Adolescent obesity (FDA-approved) · Cardiovascular risk reduction

NOT IDEAL: Patients wanting maximum weight loss (semaglutide/tirzepatide are stronger) · Poor daily adherence · MTC/MEN2 history

STACKING

Not combined with other GLP-1 agonists or insulin (Saxenda indication).

BOTTOM LINE

The budget-friendly GLP-1 with proven mortality benefit. Less potent than semaglutide but now generic and well-tolerated.

CagriSema

Cagrilintide 2.4 mg + Semaglutide 2.4 mg

PHASE 3 B: MODERATE EVIDENCE

Amylin + GLP-1 combination in one injection. 20.4% weight loss. Novo Nordisk's answer to tirzepatide.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~7 days (both components)	Weekly	68 weeks (trials)

PROTOCOL

Components: Cagrilintide (amylin analog) + Semaglutide (GLP-1 agonist)
Titration: 16-20 week escalation to full dose
Target Dose: 2.4 mg cagrilintide + 2.4 mg semaglutide
Delivery: Single pre-filled pen combining both agents
FDA Timeline: NDA filed 2025. Decision expected 2026.
Access: Clinical trial only until approved.

🧬 DUAL PATHWAY GENETICS

GLP1R – Semaglutide component response.
CALCR (Calcitonin Receptor) – Amylin receptor. Cagrilintide binding affinity.
IAPP – Amylin gene variants may affect satiety signaling.

→ Amylin pathway adds a satiety mechanism independent of GLP-1
→ Patients with weak GLP1R response may benefit from the amylin component
→ CALCR pharmacogenomics are largely unstudied, representing frontier science

CagriSema adds amylin-pathway satiety on top of GLP-1. Genetic testing can identify GLP-1 partial responders who may benefit most. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· REDEFINE 1: 20.4% weight loss vs 14.9% semaglutide alone vs 3.0% placebo (3,417 patients, 68 weeks)
· 60% achieved ≥20% weight loss; 23% achieved ≥30%
· 88% with prediabetes returned to normoglycemia
· REDEFINE 2: 13.7% weight loss in T2D population vs 3.4% placebo

BLOODWORK

HbA1c + Fasting Glucose – Baseline, 16 weeks
Lipid Panel – Baseline, 16 weeks
CMP – Baseline
Thyroid Panel – Baseline

COMMON: GI events in 79.6% of patients · Nausea (dose-dependent) · Diarrhea · Vomiting · Constipation

CAUTION: Higher GI rates than semaglutide alone · MTC risk (GLP-1 class warning) · No CV outcomes data yet

BEST FIT: Suboptimal response to semaglutide alone · Severe obesity requiring >15% weight loss · Prediabetes reversal goal · Comfortable with investigational status

NOT IDEAL: GI-sensitive patients (79.6% GI event rate) · Need for medication now (not yet approved) · Cost-sensitive (will be premium priced)

STACKING

Self-contained combination. Not combined with other GLP-1 or amylin agents.

BOTTOM LINE

Novo Nordisk's next-gen combination outperforms semaglutide alone. Approval expected 2026. The amylin pathway is the new frontier.

GROWTH HORMONE & BODY COMPOSITION

Growth Hormone & Body Composition

GH secretagogues, GHRH analogs, and direct GH therapy. For recovery, body composition, sleep, and anti-aging.

Growth hormone peptides work by stimulating your pituitary gland to produce more GH naturally (secretagogues) or by directly replacing GH (HGH 191aa). The key genetic variables are GH receptor sensitivity (GHR), IGF-1 signaling, and somatostatin tone. Response to GH peptides is highly individual: IGF-1 levels, age, body fat, and sleep quality all modulate outcomes.

Ipamorelin + CJC-1295 (no DAC)

Ipamorelin + Mod GRF 1-29 • The GH Stack

COMPOUNDABLE (CAT 1) C: EARLY/MIXED

The most popular GH secretagogue combination. Synergistic 3-5x GH amplification with clean side effect profile.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	Ipa: ~2 hrs / CJC: ~30 min	2-3x daily	8-12 weeks on, 2-4 weeks off

PROTOCOL

Ipamorelin: 200-300 mcg per injection
CJC-1295 no DAC: 100 mcg per injection (1-2 mcg/kg)
Combined Dose: Both drawn in same syringe, injected together
Frequency: 2-3x daily: morning fasted, post-workout, before bed
Timing: Empty stomach (2+ hrs after eating, no food 30 min after)
Cycle: 8-12 weeks on, then 2-4 weeks off to maintain sensitivity

🧬 GH AXIS PHARMACOGENOMICS

GHR (rs6873545) – GH receptor sensitivity. d3-GHR deletion = enhanced response.
GHRHR – GHRH receptor. Determines CJC-1295 binding efficacy.
GHSR – Ghrelin receptor. Ipamorelin binding target.
SOCS2 – Negative regulator of GH signaling. Variants affect IGF-1 response ceiling.

→ d3-GHR carriers may see stronger IGF-1 elevation at lower doses
→ GHRHR variants affect the CJC-1295 arm specifically
→ SOCS2 high-expression variants may limit maximum IGF-1 response
→ Combine genetic data with baseline IGF-1 for optimal dosing

GHR d3 deletion status is the strongest predictor of GH peptide response. Get tested before investing in an 8-12 week cycle. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Beck et al. 2014: Ipamorelin 0.03 mg/kg showed trend toward faster GI recovery post-surgery (p=0.16, not significant)
· CJC-1295 Phase 1-2: Dose-dependent GH increases of 2-10x with sustained IGF-1 elevation
· Combined: 3-5x GH amplification exceeding either agent alone (synergistic, not additive)
· No completed RCTs for body composition. Evidence is Phase 1-2 and clinical observation.

BLOODWORK

IGF-1 – Baseline, 4-6 weeks (primary marker)
Fasting Glucose + HbA1c – Baseline, 4 weeks
Fasting Insulin – Baseline
Thyroid (TSH, fT3, fT4) – Baseline
CMP – Baseline

COMMON: Injection site redness · Mild water retention · Increased hunger (Ipamorelin) · Vivid dreams · Tingling/numbness (transient)

CAUTION: IGF-1 above age-adjusted range · Glucose intolerance · Joint pain (reduce dose) · Carpal tunnel symptoms

BEST FIT: Age-related GH decline (30+) · Body recomposition goals · Sleep quality improvement · Recovery from training · Prefer natural pulsatile GH release

NOT IDEAL: Active malignancy (any type) · Diabetes with poor glucose control · Expecting fast, dramatic results (3-6 month timeline) · Budget-constrained (requires 2-3 daily injections)

STACKING

Most common GH stack. Often combined with a healing peptide (BPC-157) or a longevity peptide (Epitalon). Do not stack with other GH secretagogues (choose one combo).

BOTTOM LINE

The workhorse GH stack. Clean profile, synergistic mechanism, minimal side effects. Patience required: meaningful body composition changes take 3-6 months.

Sermorelin

Geref (discontinued brand) • GRF 1-29

COMPOUNDABLE (CAT 1) B: MODERATE EVIDENCE

The original GHRH analog. Once-daily bedtime injection. Gentler GH stimulation with preserved pulsatility.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~10-20 minutes	Daily (5 nights/week)	3-6 months, cyclical

PROTOCOL

Dose: 200-300 mcg/day
Timing: 30 min before bedtime on empty stomach (90+ min fast)
Schedule: 5 nights on, 2 nights off
Cycle: 3-6 months, then reassess IGF-1
Pediatric: 30 mcg/kg at bedtime (GH-deficient children)
Often Combined: Ipamorelin 100-300 mcg for synergistic GHRH + GHRP effect

🧬 GHRH PATHWAY GENETICS

GHRHR – GHRH receptor. Direct target of sermorelin. Variants affect binding.
GHR (d3-GHR) – GH receptor. Determines downstream signaling from GH release.
SOCS2 – GH signaling brake. High expression = lower IGF-1 ceiling.

→ GHRHR variants directly determine sermorelin efficacy
→ Hypothyroidism blunts GHRH response (check thyroid first)
→ Obesity reduces GH response; higher doses may be needed

Sermorelin response depends heavily on GHRH receptor function and thyroid status. Genetic testing identifies optimal candidates. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Previously FDA-approved for pediatric GH deficiency (Geref, now discontinued as branded product)
· Sleep quality improvements within 1-2 weeks of initiation
· IGF-1 elevation measurable at 4-8 weeks
· Body composition changes (fat loss, lean mass) require 3-6 months

BLOODWORK

IGF-1 – Baseline, 4-8 weeks
Fasting Glucose + HbA1c – Baseline (GH affects insulin)
Thyroid (TSH, fT3, fT4) – Baseline (hypothyroidism blunts GHRH)
CMP + liver enzymes – Baseline
Lipid Panel – Baseline (GH improves lipids)

COMMON: Injection site reactions · Facial flushing · Headache · Dizziness (transient)

CAUTION: Rarely causes supraphysiological GH (self-limiting via feedback) · Glucose intolerance in predisposed patients

BEST FIT: GH optimization with minimal side effects · Sleep quality as primary goal · Those who prefer once-daily dosing · Long-term GH support (3-6 month cycles) · Patients new to GH peptides (gentle entry)

NOT IDEAL: Active cancer · Obesity (reduced response, may need higher dose) · Those wanting aggressive GH elevation (HGH or combo stack better)

STACKING

Pairs well with Ipamorelin for GHRH + GHRP synergy. Can be used as a standalone bedtime GH peptide.

BOTTOM LINE

The gentlest GH peptide. Best for sleep, slow body recomposition, and those who want to stay within physiological GH ranges. Previously FDA-approved.

MK-677 (Ibutamoren)

Ibutamoren Mesylate • Nutrobal

RESEARCH ONLY | B: MODERATE EVIDENCE

Oral GH secretagogue. 97% increase in GH secretion. Convenient but watch glucose and appetite.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Oral (capsule)	~4-6 hours (effects last 24 hrs)	Once daily	Up to 12 months studied

PROTOCOL

Dose: 25 mg/day (standard)
Timing: Evening administration (enhances nocturnal GH pulse)
Titration: Some start at 12.5 mg to assess appetite/glucose response
Cycle: 12 months in clinical trials; some use 8-12 weeks cyclically
No injection: Oral capsule. No reconstitution needed.
WADA: Prohibited substance in sport.

🧬 GHRELIN RECEPTOR + METABOLIC GENETICS

GHSR (rs509035) – Ghrelin/GHS receptor. MK-677's primary target.
IGF1 (rs35767) – IGF-1 gene. Baseline level affects ceiling.
IRS1 (rs2943641) – Insulin receptor substrate. Predicts glucose deterioration risk.

→ GHSR variants affect MK-677 binding and GH release magnitude
→ IRS1 risk carriers may experience worse glucose side effects
→ Monitor glucose closely regardless of genotype

Oral convenience comes with metabolic tradeoffs. IRS1 and GHSR testing can identify who will tolerate MK-677 vs who should choose injectable GH peptides. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Nass et al. 2008: 97% increase in GH secretion, restored GH/IGF-1 to young-adult levels in elderly
· 12 months: +1.6 kg fat-free mass vs placebo in healthy older adults
· Significant increase in appetite (therapeutic for some, problematic for others)
· Hip fracture trial terminated: CHF risk 6.5% vs 1.7% placebo in frail elderly

BLOODWORK

IGF-1 – Baseline, 4 weeks, every 3 months
Fasting Glucose + HbA1c – Baseline, every 3 months (critical)
Fasting Insulin – Baseline, 3 months
Echocardiogram/BNP – Baseline if at-risk for heart failure

COMMON: Significant appetite increase · Water retention / edema · Elevated fasting glucose · Muscle pain (transient) · Lethargy in some users

CAUTION: Congestive heart failure (6.5% in elderly trial) · Insulin resistance (monitor closely) · Elevated cortisol levels · Development discontinued for safety

BEST FIT: Needle-averse patients wanting GH elevation · Appetite stimulation (cancer cachexia, underweight) · Convenience as top priority · Younger, metabolically healthy individuals

NOT IDEAL: Heart failure risk factors · Diabetes or prediabetes · Weight loss goals (appetite increase counterproductive) · Competitive athletes (WADA prohibited) · Elderly/frail (safety signal in this population)

STACKING

Sometimes stacked with a GHRH analog, but this increases side effect burden. Generally used standalone for simplicity.

BOTTOM LINE

The easiest GH peptide to use (just swallow a pill). But the metabolic side effects are real. Best for younger, metabolically healthy users who hate needles.

HGH 191aa

Somatropin • Human Growth Hormone • Genotropin • Norditropin • Humatrope

FDA APPROVED | A: STRONG EVIDENCE

Direct growth hormone replacement. The gold standard for GH deficiency. Prescription required.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	2-5 hours	Daily	3-12 months or ongoing

PROTOCOL

Anti-aging: 1-3 IU/day
Body composition: 4-6 IU/day
Titration: Start 1-2 IU, increase by 0.5-1 IU per month
Timing: Before bed (anti-aging) or AM fasted + post-workout (performance)
Avoid: Injecting within 2 hrs of high-carb meals
Storage: Refrigerate. Do not freeze.

🧬 GH RECEPTOR AND IGF-1 GENETICS

`GHR (d3-GHR deletion)` – GH receptor. d3-GHR carriers have enhanced signaling.
`IGF1 (rs35767)` – IGF-1 production. Baseline levels affect dose requirement.
`IGF1R` – IGF-1 receptor sensitivity determines downstream effects.
`SOCS2` – GH signal terminator. High expression = diminished returns at higher doses.

→ d3-GHR carriers may achieve target IGF-1 at lower doses (cost savings)
→ IGF1 variants determine baseline level and dose titration requirements
→ Genetic testing can optimize the dose-finding phase (typically months of trial-and-error)

GHR d3 deletion is the most validated pharmacogenomic marker for GH therapy. Knowing your status can save months of dose titration. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Pediatric GHD: Significant height velocity increases vs control (multiple RCTs)
· Adult GHD: Consistent lean mass increase and IGF-1 normalization (meta-analytic evidence)
· Healthy elderly: Modest lean mass gains but no functional improvement; harms often outweigh benefits
· Athletic performance: Increases lean mass but no meaningful strength or aerobic gains

BLOODWORK

IGF-1 – Baseline, monthly during titration, quarterly
Fasting Glucose + HbA1c + Insulin – Baseline, monthly initially
Thyroid (TSH, fT3, fT4) – Baseline, 3 months (GH suppresses T4→T3)
CBC – Baseline
CMP + Lipid Panel – Baseline, quarterly

COMMON: Edema (17.3% vs 4.4% placebo) · Arthralgia / myalgia (≥10%) · Carpal tunnel syndrome · Injection site reactions · Glucose intolerance

CAUTION: Carpal tunnel (19% in elderly) · Impaired glucose tolerance (significant) · Gynecomastia · Theoretical cancer risk at supraphysiological levels

BEST FIT: Diagnosed GH deficiency · Age-related GH decline with confirmed low IGF-1 · HIV-associated wasting (approved indication) · Pediatric growth failure

NOT IDEAL: Active cancer or cancer history · Diabetes with poor control · Healthy individuals seeking performance gains (risk > benefit) · Cost-sensitive (branded HGH is expensive)

STACKING

Not typically stacked with GH secretagogues (redundant). May combine with testosterone replacement in GH + hypogonadism.

BOTTOM LINE

The most potent and evidence-backed GH therapy. Reserved for diagnosed deficiency or supervised anti-aging protocols. Prescription only. Expensive.

Healing, Recovery & Regeneration

Tissue repair peptides for injuries, gut healing, and wound recovery. High demand, limited human evidence.

BPC-157

Body Protection Compound-157 • Bepecin • PL 14736

COMPOUNDABLE (CAT 1) | D: PRECLINICAL

The most popular healing peptide. Studied for tendon, ligament, GI, and soft tissue repair. Extensive preclinical data, limited human trials.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~4 hours (estimated)	2x daily	4-6 weeks, repeatable

PROTOCOL

Dose: 250-500 mcg per injection
Frequency: Twice daily (morning + evening, ~12 hours apart)
Injection Site: Can inject near injury site for localized effect
Cycle: 4-6 weeks on, then reassess
Reconstitution: Bacteriostatic water. Store reconstituted at 2-8°C for up to 4 weeks.
Oral option: Oral BPC-157 studied for GI applications (capsule form).

🧬 HEALING PATHWAY GENETICS

VEGFA (rs2010963) – Vascular endothelial growth factor. BPC-157 upregulates VEGF.
NOS3 (rs1799983) – Nitric oxide synthase. Key to BPC-157's vascular repair mechanism.
COL1A1 (rs1800012) – Collagen Type I. Determines baseline tendon/ligament repair capacity.
MMP2 – Matrix metalloproteinase. Tissue remodeling enzyme in wound repair.

→ VEGFA high-expression variants may respond more robustly
→ NOS3 G894T carriers may have impaired NO-mediated healing (slower response)
→ COL1A1 variants affect tendon injury susceptibility and repair rate
→ Genetic testing can identify patients with impaired baseline healing capacity

BPC-157 works through VEGF and NO pathways. Variants in these genes affect your baseline healing biology. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Phase 1: No quantifiable BPC-157 detected in plasma after oral dosing (bioavailability uncertainty)
· Extensive animal data: accelerated healing of tendons, ligaments, muscle, bone, GI mucosa
· No completed Phase 2/3 RCTs in humans for any indication
· Clinical popularity far exceeds clinical evidence (most-prescribed research peptide globally)

BLOODWORK

CBC with differential – Baseline
CMP (liver/kidney) – Baseline, 4 weeks
No specific biomarker – Monitor symptoms and injury progression

COMMON: Injection site irritation · Nausea (mild, rare) · Headache (rare) · Generally well-tolerated

CAUTION: Angiogenic potential (avoid with active cancer) · No long-term human safety data · Unknown interactions with other medications

BEST FIT: Soft tissue injuries (tendon, ligament, muscle) · GI issues (gut lining support) · Post-surgical recovery · Athletes with recurring injuries

NOT IDEAL: Active cancer (angiogenesis concern) · Expecting pharma-grade evidence (it doesn't exist yet) · Pregnancy · Those unwilling to accept D-grade evidence

STACKING

The 'Wolverine Stack': BPC-157 + TB-500 (lacks controlled combination studies but widely used). Also paired with GHK-Cu for skin/wound healing.

BOTTOM LINE

The most popular healing peptide in the world. Enormous preclinical promise. Zero Phase 3 data. Use with appropriate expectations and an honest understanding of the evidence gap.

TB-500

Thymosin Beta-4 Fragment • Tβ4

COMPOUNDABLE (CAT 1) | D: PRECLINICAL

Systemic tissue repair peptide. Promotes cell migration and blood vessel formation. The other half of the 'Wolverine Stack.'

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	Unknown (peptide fragment)	2x weekly	4-8 weeks, repeatable

PROTOCOL

Loading Phase: 750 mcg twice weekly for 4 weeks
Maintenance: 750 mcg once weekly
Injection Site: Subcutaneous (systemic effect, not site-specific)
Cycle: 4-8 weeks loading, then maintenance or cycle off
Reconstitution: Bacteriostatic water. Refrigerate after mixing.
Distinction: Unlike BPC-157, TB-500 works systemically, not locally.

🧬 CELL MIGRATION AND REPAIR GENETICS

TMSB4X – Thymosin Beta-4 gene. Endogenous production levels vary.
ACTA2 – Actin gene. TB-500 works by sequestering G-actin for cell migration.
VEGFA – Angiogenesis. TB-500 promotes blood vessel formation.

→ TMSB4X expression levels may affect additional benefit from exogenous TB-500
→ ACTA2 variants influence cell migration mechanics
→ High endogenous Thymosin Beta-4 producers may see diminished returns

TB-500 works through actin dynamics and angiogenesis. Your baseline healing genetics determine how much additional benefit exogenous TB-500 provides. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Thymosin Beta-4 (parent compound): Phase 2 trials for corneal wound healing showed accelerated repair
· TB-500 fragment: No human RCTs published
· Animal data: accelerated dermal wound healing, cardiac repair post-MI, hair regrowth
· Originally studied in equine medicine for racehorse tissue repair

BLOODWORK

CBC – Baseline
CMP – Baseline, 4 weeks
No specific biomarker – Symptom-based monitoring

COMMON: Injection site irritation · Headache (rare) · Mild fatigue · Generally well-tolerated

CAUTION: Theoretical: may promote tumor migration (actin polymerization) · No long-term human safety data

BEST FIT: Systemic tissue repair (multiple injury sites) · Cardiac repair support · Hair regrowth protocols · Chronic injury patterns

NOT IDEAL: Active cancer (cell migration concern) · Localized injury (BPC-157 may be better for site-specific) · Those requiring strong evidence base

STACKING

Most commonly combined with BPC-157 ('Wolverine Stack'). BPC-157 = local repair, TB-500 = systemic repair.

BOTTOM LINE

The systemic healing peptide. Works through different pathways than BPC-157, which is why they stack well. Evidence is preclinical. Popular in sports medicine.

GHK-Cu

Copper Peptide • GHK-Copper

COMPOUNDABLE (CAT 1) C: EARLY/MIXED

Copper-binding tripeptide. Skin rejuvenation, wound healing, collagen synthesis. Available topical and injectable.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC / Topical	Short (minutes)	Daily (SC) / 1-2x daily (topical)	4-8 weeks SC / 8-12 weeks topical

PROTOCOL

Injectable: 1-2 mg/day subcutaneous
Topical: 0.01-1% cream/serum, 1-2x daily
SC Cycle: 4-8 weeks on, 2-4 weeks off (prevent copper dysregulation)
Topical Cycle: 8-12 weeks continuous for visible skin results
Reconstitution: Bacteriostatic water. Refrigerate. Use within 2 weeks.
Key: Cycle injectable use to maintain receptor sensitivity and copper balance.

🧬 COLLAGEN AND COPPER GENETICS

COL1A1 (rs1800012) – Collagen Type I. Baseline skin/tendon collagen quality.
MMP2 / MMP9 – Matrix metalloproteinases. Tissue remodeling capacity.
ATP7B – Copper transporter (Wilson's disease gene). Variants affect copper handling.

→ COL1A1 variants affect baseline collagen production and skin aging rate
→ MMP variants influence tissue remodeling response to GHK-Cu
→ ATP7B screening is critical before injectable copper peptide use

GHK-Cu delivers copper to repair pathways. Your collagen genetics and copper metabolism determine response. Screen for ATP7B before injectable use. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Topical studies: significant improvement in skin elasticity, thickness, and collagen density
· Wound healing: accelerated closure in multiple preclinical models
· Gene expression: modulates 4,000+ human genes related to repair and inflammation
· Limited controlled human trials for injectable route

BLOODWORK

Serum Copper + Ceruloplasmin – Baseline (critical for SC route)
CMP – Baseline, 4 weeks
Serum Copper – 4 weeks, then ongoing if injecting
CBC – Baseline

COMMON: Injection site reactions · Skin irritation (topical, test small area first) · Mild and transient generally

CAUTION: Copper accumulation risk with injectable route · Discontinue if serum copper exceeds normal range · Avoid in Wilson's disease

BEST FIT: Skin rejuvenation and anti-aging (topical) · Post-procedure wound healing · Hair thinning protocols · Collagen stimulation

NOT IDEAL: Wilson's disease or copper overload · Long-term injectable use without copper monitoring · Those seeking rapid dramatic results (topical takes 8-12 weeks)

STACKING

Topical GHK-Cu + injectable BPC-157 for comprehensive healing. Also pairs with TB-500 for systemic + skin protocols.

BOTTOM LINE

The best-studied peptide for skin. Topical is low-risk and well-supported. Injectable requires copper monitoring. Excellent for post-procedure recovery.

SEXUAL HEALTH & HORMONES

Sexual Health & Hormones

Peptides for libido, arousal, fertility support, and hormone optimization during TRT.

PT-141 (Bremelanotide)

Vyleesi • Bremelanotide Acetate

FDA APPROVED A: STRONG EVIDENCE

FDA-approved for female hypoactive sexual desire disorder. Works through the brain, not the vasculature. As-needed dosing.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~2.7 hours	As needed (PRN)	On-demand, max 8x/month

PROTOCOL

Dose: 1.75 mg subcutaneous (fixed dose)
Timing: At least 45 minutes before anticipated activity
Max Frequency: No more than once per 24 hours, max 8 doses/month
Peak Effect: ~1 hour post-injection
Delivery: Pre-filled auto-injector (Vyleesi). Ready to use.
Storage: Room temperature (20-25°C). Protect from light.

🧬 MELANOCORTIN RECEPTOR GENETICS

MC4R (rs17782313) – Primary target. Melanocortin-4 receptor in hypothalamus. Drives sexual motivation.
MC1R (rs1805007) – Skin pigmentation receptor. Off-target activation causes hyperpigmentation.
DRD2 (rs6277) – Dopamine D2 receptor. PT-141 triggers dopamine release in reward circuits.

→ MC4R variants affect the magnitude of sexual desire response
→ MC1R red-hair variants may experience more or less hyperpigmentation
→ DRD2 variants influence dopamine-mediated arousal pathways

PT-141 works through MC4R → dopamine. Your melanocortin receptor genetics predict response strength and hyperpigmentation risk. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· RECONNECT Phase 3 (1,267 women): Significant increase in sexual desire vs placebo (P<.001)
· Significant reduction in HSDD-related distress (P<.001)
· 52-week extension: sustained efficacy over long-term use
· Onset within 45-60 minutes; effects can persist several hours

BLOODWORK

Blood Pressure – Baseline, with first doses, ongoing
CBC + CMP – Baseline
Skin Exam – Baseline (MC1R activation → focal hyperpigmentation)

COMMON: Nausea (40% vs 1.3% placebo) · Flushing (20.6%) · Headache (12%) · Transient BP increase (1.9/1.7 mmHg)

CAUTION: Focal hyperpigmentation (melanocyte activation) · Persistent BP elevation → discontinue · Not approved for men (off-label only)

BEST FIT: Premenopausal women with HSDD (FDA indication) · Desire-based (not arousal-based) sexual dysfunction · Non-vascular mechanism (unlike PDE5 inhibitors) · As-needed dosing preference

NOT IDEAL: Uncontrolled hypertension · Cardiovascular disease · Arousal dysfunction (PDE5 inhibitors better for vascular causes) · Nausea-sensitive patients (40% incidence)

STACKING

Not typically combined with other sexual health peptides. Complements PDE5 inhibitors for different mechanism.

BOTTOM LINE

The only FDA-approved peptide for sexual desire. Works on the brain, not blood flow. Nausea is the main barrier. As-needed convenience is a major advantage.

Gonadorelin

GnRH • Factrel (diagnostic) • Lutrepulse

FDA APPROVED B: MODERATE EVIDENCE

Natural GnRH analog. Preserves fertility during TRT. Pulsatile dosing is critical: continuous use causes the opposite effect.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~2-4 minutes	2-3x weekly	Ongoing during TRT

PROTOCOL

TRT Adjunct: 100-200 mcg, 2-3x weekly subcutaneous
Fertility: Pulsatile pump: 10 mcg every 90 minutes (clinical setting)
Critical Rule: Must be pulsatile (intermittent). Continuous = receptor shutdown.
Timing: Consistent schedule; specific time of day less important
Duration: Ongoing while on TRT; 4-8 weeks for diagnostic/fertility protocols
Reconstitution: Bacteriostatic water.

🧬 HPG AXIS GENETICS

GNRHR – GnRH receptor. Direct binding target. Variants affect signaling.
FSHB (rs10835638) – FSH beta subunit. Affects FSH production in response to GnRH.
LHB – LH beta subunit. Determines LH pulse amplitude.

→ GNRHR variants can cause partial resistance to gonadorelin
→ FSHB variants affect the fertility response specifically
→ Genetic testing can predict who will respond adequately to 100 mcg vs needing 200 mcg

GnRH receptor genetics determine whether gonadorelin adequately maintains fertility during TRT. Test before committing to a protocol. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Spermatogenesis rate: 76% in hypogonadotropic hypogonadism (95% CI 65-86%)
· Time to first sperm: 6 months (vs 18 months with HCG/HMG)
· Female functional amenorrhea: 96% ovulation rate per cycle
· Cumulative live birth rate: 65.9% per treatment course

BLOODWORK

LH + FSH – Baseline, 4-6 weeks
Total + Free Testosterone – Baseline, 4-6 weeks
Estradiol (sensitive) – Baseline
Semen Analysis – Baseline if fertility goal

COMMON: Injection site reactions · Gynecomastia (~4%) · Acne (~5%) · Generally well-tolerated

CAUTION: Continuous (non-pulsatile) dosing → paradoxical LH/FSH suppression · Essentially zero with correct pulsatile protocol

BEST FIT: Fertility preservation during TRT · Testicular atrophy prevention on TRT · Hypogonadotropic hypogonadism · Patients who prefer GnRH over HCG

NOT IDEAL: Pituitary failure (needs functional pituitary) · Those who cannot maintain consistent dosing schedule · Hormone-sensitive cancers

STACKING

Used alongside TRT (testosterone). Alternative to HCG for maintaining testicular function on exogenous testosterone.

BOTTOM LINE

The physiological choice for fertility preservation on TRT. Works with your pituitary, not around it. Pulsatile dosing is non-negotiable.

HCG

Human Chorionic Gonadotropin • Pregnyl • Ovidrel

FDA APPROVED A: STRONG EVIDENCE

LH analog. Gold standard for fertility support during TRT and spermatogenesis induction. Also used for ovulation triggering.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC / IM	~24-36 hours	Every other day or 2-3x weekly	Ongoing during TRT / 6-12 weeks for fertility

PROTOCOL

TRT Adjunct: 250-500 IU every other day or 2-3x weekly
Fertility/PCT: 1,500-5,000 IU, 2-3x weekly for 6-12 weeks
Ovulation Trigger: Single dose per ART cycle (5,000-10,000 IU)
Route: SC preferred for TRT adjunct; IM for higher doses
Timing: Consistent schedule; IM yields higher Cmax than SC
Storage: Refrigerate after reconstitution. Bacteriostatic water.

🧬 LH RECEPTOR AND AROMATASE GENETICS

LHCGR (rs4539842) – LH/CG receptor. Direct target of HCG. Variants affect Leydig cell response.
CYP19A1 (rs4646) – Aromatase gene. Determines E2 conversion rate from HCG-stimulated testosterone.
SHBG (rs6257) – Sex hormone binding globulin. Affects free testosterone levels.

→ LHCGR variants determine testicular response to HCG (some need higher doses)
→ CYP19A1 high-activity variants = more estrogen conversion = may need AI
→ SHBG variants affect how much of the HCG-stimulated testosterone is bioavailable

HCG response varies by LH receptor genetics and aromatase activity. Knowing your CYP19A1 status predicts whether you will need estrogen management. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Spermatogenesis preservation during TRT: Zero azoospermia in 26-man series; 9/26 partner pregnancies
· Hypogonadotropic hypogonadism: 77.8% achieved spermatogenesis ≥1M/mL
· Testicular volume: 8.6 → 17.8 mL in HH treatment
· Dual trigger (GnRH-agonist + HCG): Live birth rate 36.2% vs 22.0% HCG alone in ART

BLOODWORK

Total + Free Testosterone – Baseline, 4-6 weeks
Estradiol (sensitive) – Baseline, 4-6 weeks (HCG raises E2)
LH + FSH – Baseline
CBC – Baseline
Semen Analysis – Baseline if fertility goal

COMMON: Estradiol elevation (intratesticular aromatization) · Gynecomastia · Acne · Water retention · Injection site reactions

CAUTION: OHSS in women (ovarian hyperstimulation) · Venous thromboembolism (rare case reports) · May need aromatase inhibitor if E2 gets too high

BEST FIT: Fertility preservation during TRT · Spermatogenesis induction · Testicular atrophy prevention · ART/IVF ovulation triggering

NOT IDEAL: Androgen-dependent cancers · History of venous thromboembolism · Estrogen-sensitive conditions without AI management

STACKING

Used alongside testosterone replacement. Can be combined with FSH analogs for enhanced fertility outcomes. Alternative to gonadorelin.

BOTTOM LINE

The most proven fertility support peptide. Works. Raises testosterone and preserves testicular function. Watch estrogen: get CYP19A1 tested.

Cognitive & Neuroprotection

Peptides targeting focus, anxiety, mood, neuroplasticity, and neuroprotection. Evidence ranges from approved (Russia) to purely preclinical.

Selank

TP-7 • Thr-Lys-Pro-Arg-Pro-Gly-Pro

COMPOUNDABLE (CAT 1) C: EARLY/MIXED

Anxiolytic peptide approved in Russia. Intranasal delivery. Reduces anxiety without sedation or dependence.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Intranasal / SC	Minutes (enhanced by D-Arg modification)	2-3x daily	30 days on, 30 days off

PROTOCOL

Intranasal: 300-600 mcg per dose, 2-3x daily
Subcutaneous: 200-400 mcg/day, 5 days/week
Timing: Morning and early afternoon. Avoid evening (may be activating).
Cycle: 30 days on, 30 days off. ~6 cycles per year.
Storage: Reconstitute with bacteriostatic water. Refrigerate.

🧬 ANXIETY AND GABA/SEROTONIN GENETICS

COMT (rs4680) – Catechol-O-methyltransferase. Val/Met affects dopamine/norepinephrine clearance.
BDNF (rs6265) – Brain-derived neurotrophic factor. Val66Met affects neuroplasticity.
GAD1 – Glutamic acid decarboxylase. GABA synthesis rate.

→ COMT Met/Met (worrier phenotype) may respond most strongly to anxiolytics
→ BDNF Met carriers have reduced neuroplasticity (may need longer cycles)
→ GAD1 variants affect baseline GABA tone and anxiety susceptibility

Selank modulates GABA and enkephalin systems. COMT and BDNF status predict anxiety susceptibility and neuroplasticity response. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· GAD study (n=20): 40% rapid responders by day 3, remaining 60% by day 14
· Single 900 mcg dose produced measurable EEG changes (increased beta, decreased theta)
· Adjustment disorder (n=30): 2-week improvement in somatic symptoms vs untreated controls
· No controlled trials outside Russian regulatory framework

BLOODWORK

CBC with differential – Baseline (immune modulation)
CMP – Baseline
Thyroid Panel – Baseline (rule out thyroid anxiety)
AM Cortisol – Baseline (HPA axis)
CBC – End of cycle

COMMON: Minimal reported side effects · Nasal irritation (intranasal route) · No sedation or dependence reported

CAUTION: Limited safety data outside Russian framework · Caution in bleeding disorders (tuftsin analog) · Long-term human safety unknown

BEST FIT: Generalized anxiety without desire for sedation · Focus enhancement alongside anxiolysis · Those avoiding benzodiazepine-class medications · Intranasal preference (no injection needed)

NOT IDEAL: Those requiring strong evidence base (small trials only) · Severe anxiety requiring immediate pharmaceutical intervention · Bleeding disorders

STACKING

Often paired with Semax (cognitive + anxiolytic combo). Can complement adaptogens and lifestyle interventions.

BOTTOM LINE

The gentlest anxiolytic peptide. No sedation, no dependence, intranasal convenience. Evidence is limited to small Russian trials. Approved in Russia since 2009.

Semax

ACTH(4-10) analog • Heptapeptide

COMPOUNDABLE (CAT 1) C: EARLY/MIXED

Nootropic peptide approved in Russia for stroke recovery and cognitive enhancement. Intranasal. Does not affect cortisol despite ACTH origin.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Intranasal	Hours (extended vs ACTH)	2-3x daily	10-14 days on, 4 weeks off

PROTOCOL

Nootropic (0.1%): 200-600 mcg/dose, 2-3x daily intranasal
Stroke (1%): 3-6 mg/dose, 2-3x daily (supervised)
Timing: Morning/early afternoon. Avoid evening.
Cycle: 10-14 days on, 4 wks off. 2-4 courses/yr.

🧬 NEUROTROPHIN AND COGNITIVE GENETICS

BDNF (rs6265) – Val66Met affects neuroplasticity response to Semax
NGF – Semax enhances NGF expression
COMT (rs4680) – Dopamine clearance; baseline cognitive phenotype

→ BDNF Val/Val: stronger neuroplasticity benefit
→ BDNF Met carriers: may need longer courses
→ COMT genotype shifts cognitive vs mood effects

BDNF genotype predicts neuroplasticity response magnitude. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Approved in Russia since 2011 for stroke and cognitive disorders
· BDNF upregulation demonstrated in human studies
· ~7% of diabetics report glucose elevation; no Western trials

BLOODWORK

CBC / CMP – Baseline
Fasting Glucose – Baseline + end of course
Thyroid Panel – Baseline
AM Cortisol – Baseline (does NOT elevate cortisol)

COMMON: Nasal irritation · Mild and transient generally · Glucose elevation in ~7% of diabetics

CAUTION: Caution with diabetes (glucose effect) · Caution with hypertension · No Western safety surveillance data

BEST FIT: Cognitive enhancement (focus, memory) · Post-stroke neurological recovery · Short course protocols (10-14 days)

NOT IDEAL: Diabetes (7% glucose elevation risk) · Uncontrolled hypertension · Those needing Western regulatory approval

STACKING

Often combined with Selank (cognitive + anxiolytic). The 'Russian nootropic stack.'

BOTTOM LINE

Russia's premier nootropic peptide. BDNF upregulation is the mechanism. Short courses, intranasal delivery, minimal side effects. No Western approval.

Cerebrolysin

FPF-1070 • Brain-Derived Peptide Mixture

RESEARCH ONLY | B: MODERATE EVIDENCE

Porcine brain-derived peptide mixture mimicking endogenous neurotrophic factors. Approved in 40+ countries for stroke and TBI. IV/IM only.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
IV / IM	Short (peptide fragments cleared in hours)	Daily for 10-20 days	10-20 day courses, 2-4x/year

PROTOCOL

IV Infusion: 10-30 mL in 100 mL saline, daily, 10-20 days
IM (lower dose): 5-10 mL daily, 10-20 days
Post-Stroke Acute: 30-50 mL IV daily, 21 days (start within 72h)

🧬 THE NEUROTROPHIC RESPONSE VARIABLE

BDNF (rs6265) – Met carriers have reduced BDNF secretion; Cerebrolysin compensates
APOE (rs429358/rs7412) – APOE4 carriers: accelerated neurodegeneration, may benefit most
COMT (rs4680) – Dopamine clearance rate affects cognitive response ceiling

→ BDNF Met carriers: greater relative benefit (compensating for low endogenous BDNF)
→ APOE4: higher neurodegeneration risk makes neuroprotection more urgent
→ COMT Val/Val: faster dopamine clearance, more pronounced improvement

APOE and BDNF status predict who benefits most from neurotrophic compensation. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Cochrane meta-analysis (2020): modest cognitive benefit in vascular dementia
· CASTA trial: 30 mL/day post-stroke improved NIHSS scores at 90 days (n=1,070)
· Multiple RCTs show improved ADL scores in moderate Alzheimer's disease
· TBI trials: accelerated recovery in GCS 9-12 patients when administered within 24h

BLOODWORK

CBC with Differential – Baseline (monitor for allergic reactions)
LFTs (ALT, AST) – Baseline + post-course (porcine-derived product)
CRP – Inflammation marker. Should decrease.
Renal Panel – If high-dose IV protocol

COMMON: Dizziness · Headache · Injection site reaction · Mild agitation

CAUTION: Allergic reaction (porcine-derived) · Seizure risk in epilepsy patients · Hyperthermia (rare)

BEST FIT: Post-stroke cognitive recovery (within 72h onset) · Moderate Alzheimer's/vascular dementia · TBI rehabilitation protocols · BDNF Met carriers seeking neuroplasticity support

NOT IDEAL: Epilepsy patients (seizure threshold lowered) · Those uncomfortable with porcine-derived products · Mild cognitive complaints (overkill) · No IV/IM access

STACKING

Can complement Semax (intranasal) for dual-route neurotrophic support. Not typically combined with other IV neuropeptides.

BOTTOM LINE

The most evidence-backed neuropeptide for serious neurological conditions. Approved in 40+ countries. IV-only delivery limits accessibility. Not a nootropic for healthy brains. This is medicine for damaged brains.

Dihexa

N-hexanoic-Tyr-Ile-(6)-aminohexanoic amide

RESEARCH ONLY | D: PRECLINICAL

Hepatocyte growth factor (HGF) mimetic, 10 million times more potent than BDNF at forming new synapses in vitro. Preclinical only. Extremely potent.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Oral / SC / Intranasal	Estimated 6-12 hours	Daily (short courses)	2-4 weeks on, 4+ weeks off. No established protocol.

PROTOCOL

Oral: 10-20 mg daily, 2-4 weeks
SC Injection: 2-5 mg daily, 2-4 weeks
Intranasal: 5-10 mg daily, 2-4 weeks

🧬 THE GROWTH FACTOR VARIABLE

`MET (c-Met receptor)` – Dihexa's target. Polymorphisms affect receptor sensitivity and expression.
`HGF` – Endogenous HGF levels vary. Low producers may see more benefit from exogenous HGF mimetic.
`BDNF (rs6265)` – Indirect relevance. If BDNF pathway is impaired, HGF pathway gains importance.

→ MET receptor variants may alter Dihexa binding and efficacy
→ Low endogenous HGF producers: theoretically greater response
→ BDNF Met carriers: HGF pathway may serve as alternative neuroplasticity route

Dihexa bypasses BDNF entirely, using HGF/c-Met for synaptogenesis. Genetics affecting both pathways matter. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· NO human clinical trials. All data is animal/cell studies.
· Rat studies (2013): reversed scopolamine-induced cognitive deficits
· Animal models: restored spatial memory in aged rats comparable to young controls
· Mechanism paper (Bhatt et al., 2014): confirmed HGF/c-Met pathway activation

BLOODWORK

LFTs (ALT, AST, GGT) – HGF pathway. Monitor liver function closely.
CBC – Baseline safety check
Blood Pressure – HGF affects vascular growth; monitor.

COMMON: Headache · Vivid dreams (reported anecdotally) · Mild anxiety/overstimulation

CAUTION: Unknown long-term safety profile · Theoretical tumor promotion risk (HGF/c-Met involved in some cancers) · No human safety data

BEST FIT: Researchers studying HGF/c-Met pathway · Those who've exhausted standard nootropic peptides · Individuals with full informed consent of risk profile

NOT IDEAL: Anyone with cancer history (HGF/c-Met oncogenic potential) · Risk-averse individuals (no human safety data) · Those expecting evidence-backed protocols (none exist) · Anyone under 40 without cognitive complaints

STACKING

Not recommended to stack with other potent nootropics until safety is established. Some anecdotally combine with Semax, but no data supports this.

BOTTOM LINE

The most potent synaptogenic compound ever tested. 10 million times more active than BDNF in vitro. But zero human trials, unknown safety profile, and HGF/c-Met oncogenic concerns. This is frontier science. Treat it accordingly.

Anti-Aging & Longevity

Peptides targeting telomeres, mitochondrial function, cellular senescence, and NAD+ metabolism. Frontier science with limited human data.

Epitalon

Epithalon • Epithalone • AEDG Peptide

COMPOUNDABLE (CAT 1) D: PRECLINICAL

Telomerase-activating tetrapeptide. Studied for telomere elongation and pineal gland function. Course-based dosing.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	Short (minutes)	Daily (course-based)	10-20 days, repeat every 4-6 months

PROTOCOL

Dose: 5-10 mg/day subcutaneous
Cycle: 10-20 consecutive days per course
Frequency: 2-3 courses per year (every 4-6 months)
Timing: Evening/before bed (aligns with pineal melatonin cycle)
Reconstitution: Bacteriostatic water. Store at -20°C (lyophilized), 2-8°C (reconstituted).
Protect from light.

🧬 TELOMERE AND CIRCADIAN GENETICS

TERT (rs2736100) – Telomerase reverse transcriptase. Epitalon's primary target.
CLOCK (rs1801260) – Master circadian gene. Pineal function modulation.
TERC – Telomerase RNA component. Determines telomere maintenance capacity.

→ TERT variants affect baseline telomerase activity and elongation potential
→ CLOCK variants influence circadian rhythm response to pineal peptides
→ Short baseline telomeres (measurable) may indicate higher potential benefit

Epitalon targets telomerase. Your TERT genotype and baseline telomere length determine potential benefit. Measure before and after. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Khavinson et al.: Telomerase activation and telomere elongation in human cell cultures
· Observational studies (Khavinson group): improved circadian rhythm and melatonin secretion in elderly
· No registered clinical trials in ClinicalTrials.gov or EU registries
· All published evidence from a single research group (replication needed)

BLOODWORK

CBC with differential – Baseline
CMP – Baseline, end of course
Melatonin Level (optional) – Baseline (overnight/salivary)
Telomere Length (optional) – Baseline (for tracking over courses)

COMMON: Injection site reactions · Generally well-tolerated in limited data

CAUTION: Theoretical: telomerase activation could promote cancer cell survival · No long-term safety data in humans · Single research group; no independent replication

BEST FIT: Longevity-focused protocols · Circadian rhythm / sleep optimization · Those tracking telomere length over time · Course-based approach (not daily long-term)

NOT IDEAL: Active cancer or high cancer risk · Those requiring replicated clinical trial evidence · Expecting rapid visible results

STACKING

Often combined with MOTS-c (mitochondrial) for comprehensive anti-aging. Can pair with melatonin optimization protocols.

BOTTOM LINE

The telomere peptide. Fascinating mechanism, single-lab evidence. Course-based dosing is convenient. Cancer safety question remains unresolved.

MOTS-c

Mitochondrial ORF of the Twelve S rRNA Type-c

COMPOUNDABLE (CAT 1) D: PRECLINICAL

Mitochondrial-derived peptide. Activates AMPK for metabolic regulation. Exercise mimetic properties. Entirely preclinical.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	Unknown	2-3x weekly	4-6 weeks, repeat 2-4x/year

PROTOCOL

Dose: 5-10 mg per injection
Frequency: 2-3 times per week or every 5 days
Cycle: 4-6 weeks (20-30 day protocols), repeat 2-4x per year
Timing: Morning or pre-exercise for metabolic activation
Reconstitution: Bacteriostatic water. Refrigerate.
Course-based: Treatment repeatable with rest periods.

🧬 MITOCHONDRIAL AND METABOLIC GENETICS

`MT-RNR1` – Mitochondrial 12S rRNA gene. MOTS-c is encoded within this gene.
`AMPK (PRKAA2)` – AMP-activated protein kinase. MOTS-c's primary signaling target.
`PPARGC1A (rs8192678)` – PGC-1alpha. Mitochondrial biogenesis master regulator.

→ MT-RNR1 variants affect endogenous MOTS-c production (maternal inheritance)
→ AMPK pathway genetics determine metabolic response to exercise mimetics
→ PGC-1alpha variants influence mitochondrial biogenesis capacity

MOTS-c is encoded in your mitochondrial DNA. Variants in MT-RNR1 affect your baseline production. Mitochondrial genetics are maternally inherited. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· No completed human clinical trials (entirely preclinical)
· Mouse studies: improved glucose tolerance, insulin sensitivity, exercise capacity
· AMPK activation demonstrated as primary mechanism
· Translocation to nucleus during metabolic stress (unique among mitochondrial peptides)

BLOODWORK

Fasting Glucose + HbA1c – Baseline, 4 weeks
Fasting Insulin – Baseline, 4 weeks
CMP – Baseline
Lipid Panel – Baseline
hsCRP – Baseline (inflammation marker)

COMMON: No human safety data available · Complete absence of documented side effects (untested, not safe)

CAUTION: Entirely unknown safety profile in humans · No formal contraindications (because no human data exists)

BEST FIT: Metabolic aging / insulin sensitivity goals · Exercise mimetic interest · Mitochondrial optimization protocols · Those comfortable with preclinical-only evidence

NOT IDEAL: Anyone expecting proven results (zero human trials) · Those needing evidence-based protocols · Competitive athletes (untested substance risk)

STACKING

Often paired with Epitalon (telomeres + mitochondria). Can complement NAD+ protocols.

BOTTOM LINE

The most scientifically fascinating anti-aging peptide. Mitochondrial-encoded, AMPK-activating, exercise-mimetic. Zero human data. Pure frontier science.

NAD+

Nicotinamide Adenine Dinucleotide • NR (Nicotinamide Riboside) • NMN

RESEARCH ONLY C: EARLY/MIXED

Cellular energy currency. IV infusion or oral precursors (NR/NMN). Central to sirtuins, DNA repair, and mitochondrial function.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
IV / Oral	~45 min (IV NAD+) / varies (oral precursors)	1-2x weekly IV / daily oral	4-8 weeks IV loading, then maintenance

PROTOCOL

IV Loading: 250-750 mg, 1-2x/wk for 4-8 wks. Start 1-2 mg/min.
IV Maintenance: Monthly sessions
Oral (NR/NMN): 300-1,000 mg daily, morning, ongoing

🧬 NAD+ METABOLISM GENETICS

NAMPT (rs61330082) – Rate-limiting salvage enzyme; determines baseline NAD+
SIRT1 (rs7895833) – NAD+-dependent deacetylase; longevity gene
PARP1 – DNA repair; high activity = faster NAD+ depletion
NNMT – Diverts NAD+ precursors from salvage pathway

→ Low NAMPT expression = lower baseline NAD+ = greater supplementation benefit
→ SIRT1 variants affect NAD+-to-longevity signal conversion
→ High PARP1 activity may need higher doses

NAD+ declines 50% by age 60. NAMPT and SIRT1 genetics determine depletion rate and response. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· NR 1,000 mg/day: Safe and well-tolerated up to 2,000 mg/day in healthy adults
· Liposomal GSH + NAD+ precursors: up to 400% NK cell cytotoxicity increase (pilot study)
· Oxaliplatin neuropathy: IV reduced glutathione 1,500 mg/m² reduced Grade 2-4 neurotoxicity
· Disease-specific outcomes remain largely preclinical for NAD+ itself

BLOODWORK

CMP + LFTs – Baseline, 4-6 weeks
CBC – Baseline
Fasting Glucose + Insulin – Baseline
NAD+ metabolite panel – 4-8 weeks (if available)

COMMON: Nausea (IV infusion) · Flushing and warmth (IV) · GI discomfort (oral) · Headache

CAUTION: Avoid during active cancer treatment (especially PARP inhibitors) · Rapid IV infusion can cause severe chest tightness · Paradoxical liver enzyme elevation (monitor)

BEST FIT: Age-related energy/cognition decline · Post-addiction recovery protocols (IV NAD+) · Mitochondrial optimization · Those willing to invest in IV sessions

NOT IDEAL: Active cancer on PARP inhibitors · Budget-constrained (IV is expensive) · Those expecting oral NR/NMN to match IV effects (bioavailability differs)

STACKING

Often combined with resveratrol (SIRT1 activator) and TMG (methylation support). Can pair with MOTS-c for mitochondrial stack.

BOTTOM LINE

The foundational longevity molecule. IV is potent but expensive and time-consuming. Oral precursors (NR/NMN) are convenient but less dramatic. Genetics determine your depletion rate.

FOXO4-DRI

Proxofim • FOXO4-D-Retro-Inverso

RESEARCH ONLY D: PRECLINICAL

Senolytic peptide that selectively induces apoptosis in senescent cells by disrupting the FOXO4-p53 interaction. Preclinical. The 'zombie cell killer.'

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC / IV	Estimated hours (D-amino acids resist proteolysis)	Pulsed protocols (3 days on, 2 weeks off)	3-day pulses every 2-4 weeks. No established human protocol.

PROTOCOL

SC (discussed): 5-10 mg/day, 3 consecutive days, repeat every 2-4 wks
IV (clinical): Higher dose, provider-dependent, 3 days, research only

🧬 THE SENESCENCE VARIABLE

`TP53 (rs1042522)` – p53 is FOXO4-DRI's released effector. Arg72Pro variant affects apoptotic potency.
`CDKN2A (p16INK4a locus)` – Primary senescence marker. Genetic variation affects senescent cell accumulation rate.
`FOXO3 (rs2802292)` – Longevity-associated FOXO variant. Affects cellular stress response and senescence pathways.

→ TP53 Arg/Arg: stronger apoptotic response when p53 is released from FOXO4
→ CDKN2A variants affecting p16 expression: higher senescent cell burden may mean more to clear
→ FOXO3 longevity allele (G): already enhanced stress response, may have lower senescent burden

FOXO4-DRI's mechanism is p53-dependent. Your TP53 genotype affects apoptotic efficiency, and CDKN2A affects senescent cell burden. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· NO human clinical trials. All data is animal/cell studies.
· Baar et al. (2017, Cell): reversed chemotherapy-induced senescence in mice
· Restored fur density, renal function, and fitness in aged mice
· Selectively killed senescent cells while sparing healthy cells in vitro
· Mouse lifespan extension data is promising but not yet replicated

BLOODWORK

Senescence Markers (p16INK4a, p21) – Research panels only. Not widely available.
CRP, IL-6 – Inflammatory markers. Senolytic clearance should reduce chronic inflammation.
CBC with Differential – Safety monitoring
Comprehensive Metabolic Panel – Kidney and liver function

COMMON: Injection site pain · Fatigue during clearance window · Mild flu-like symptoms (senescent cell clearance)

CAUTION: Unknown long-term safety · Theoretical wound healing impairment (senescence plays role in tissue repair) · No human toxicology data

BEST FIT: Researchers studying senolytics · Those with high senescent cell burden markers · Individuals who understand and accept experimental risk · Anti-aging protocols where dasatinib+quercetin was insufficient

NOT IDEAL: Active cancer (senescence is a tumor-suppressive mechanism) · Active wound healing or surgery recovery · Risk-averse individuals (no human data) · Anyone expecting proven longevity outcomes

STACKING

Theoretically complementary with dasatinib+quercetin (different senolytic mechanisms). Often discussed alongside Epitalon (telomere maintenance after senescent clearance) and MOTS-c (mitochondrial support).

BOTTOM LINE

The most targeted senolytic peptide known. Kills zombie cells while leaving healthy cells alone. But only proven in mice. D-retro-inverso design is clever chemistry. This is the frontier of longevity science. Treat it as such.

Immune & Gut Health

Antimicrobial peptides, immune modulators, and gut-healing compounds. Evidence ranges from clinical to preclinical.

LL-37

Cathelicidin • hCAP-18 (precursor)

RESEARCH ONLY C: EARLY/MIXED

Human antimicrobial peptide. Broad-spectrum pathogen defense. Promotes wound healing and angiogenesis. Bell-shaped dose response.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC / Topical	Short (minutes, rapidly degraded)	Daily (5 days/week)	2-4 weeks on, 2 weeks off

PROTOCOL

Subcutaneous: 100-200 mcg daily, 5 days per week
Topical: 0.5-1.6 mg/mL per formulation (wound healing)
Cycle: 2-4 weeks on, 2 weeks off
Critical: Bell-shaped dose response. Higher is NOT better.
Reconstitution: Bacteriostatic water.
Do NOT exceed: 1.6 mg/mL topical (higher concentrations increase inflammation).

🧬 ANTIMICROBIAL DEFENSE GENETICS

VDR (rs2228570) – Vitamin D receptor. VDR activates CAMP gene → LL-37 production.
CAMP – Cathelicidin gene. Encodes LL-37 precursor. Expression varies.
TLR2 / TLR4 – Toll-like receptors. Innate immune recognition that LL-37 modulates.

→ VDR variants dramatically affect endogenous LL-37 production
→ Low Vitamin D + VDR risk variant = very low natural LL-37 (check Vitamin D first)
→ TLR variants influence inflammatory response to LL-37 supplementation

LL-37 production is controlled by Vitamin D and VDR genetics. Check your Vitamin D level and VDR genotype before considering exogenous LL-37. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· HEAL LL-37: Topical 0.5 mg/mL superior for venous leg ulcers ≥10 cm² (RCT)
· Diabetic foot ulcers: Faster granulation at days 7, 14, 21, 28 (0.5 mg/g cream)
· Higher concentrations (3.2 mg/mL): Increased inflammation without additional benefit
· Bell-shaped dose response: optimal at lower concentrations, harmful at higher

BLOODWORK

CBC with differential – Baseline (immune status)
hsCRP – Baseline, 2-4 weeks
CMP – Baseline
Vitamin D (25-OH) – Baseline (critical: Vitamin D regulates LL-37 expression)
CBC – 2 weeks (monitor immune response)

COMMON: Injection site reactions (redness, burning) · Local inflammation (immune-stimulatory) · Adjacent skin redness, warmth

CAUTION: Dose-dependent toxicity (bell-shaped curve) · Mast cell activation via MRGPRX2 · May exacerbate autoimmune conditions · Can worsen inflammatory skin conditions at high doses

BEST FIT: Chronic wound healing (venous/diabetic ulcers) · Antimicrobial support · Low Vitamin D status (may have low endogenous LL-37) · Topical application for wound care

NOT IDEAL: Autoimmune conditions (may flare) · Mast cell disorders · Injectable use without medical supervision · Higher doses (bell-shaped response curve)

STACKING

Optimize Vitamin D first (the natural LL-37 inducer). Can complement KPV for immune + gut protocols.

BOTTOM LINE

Your body's own antimicrobial peptide. Bell-shaped dose response means less is more. Check Vitamin D first: it may be all you need to boost endogenous LL-37.

KPV

Lys-Pro-Val • Alpha-MSH Fragment

RESEARCH ONLY | D: PRECLINICAL

Anti-inflammatory tripeptide derived from alpha-MSH. Targets NF-kB pathway. Studied for IBD and gut inflammation. Entirely preclinical.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
SC / Oral	Unknown	Daily	4-8 weeks, repeatable

PROTOCOL

Subcutaneous: 200-500 mcg per injection, once daily
Oral: Capsule form, 1-2x daily
Timing: No specific requirement. Oral on empty stomach may improve absorption.
Cycle: 4-8 weeks minimum, extendable for chronic conditions
Reconstitution: Bacteriostatic water for injectable form.
Key: PepT1 transporter enables intestinal epithelial uptake (oral may work for gut).

🧬 INFLAMMATION AND NF-KB GENETICS

MC1R (rs1805007) – Alpha-MSH receptor. KPV is an alpha-MSH fragment targeting this pathway.
NFKB1 (rs28362491) – NF-kB transcription factor. KPV inhibits NF-kB nuclear translocation.
IL10 (rs1800896) – Anti-inflammatory cytokine. Baseline IL-10 production affects inflammation control.

→ MC1R variants (common in red-haired individuals) may alter melanocortin pathway response
→ NFKB1 variants affect baseline NF-kB activity and inflammatory burden
→ Low IL-10 producers may have more room for anti-inflammatory benefit

KPV works through melanocortin receptors and NF-kB inhibition. Your MC1R and NFKB1 genetics affect baseline inflammation. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· No human clinical trials conducted (entirely preclinical)
· Rodent colitis models: significant reduction in intestinal inflammation
· NF-kB inhibition demonstrated in cell culture
· Distinct mechanism from conventional IBD medications (potential complementary use)

BLOODWORK

CRP + ESR – Baseline, 4-6 weeks
CBC with differential – Baseline
CMP with liver enzymes – Baseline
Fecal Calprotectin – Baseline, 4-6 weeks (if IBD)

COMMON: GI discomfort (reported) · Generally described as mild and transient

CAUTION: No human safety data exists · No established contraindications (because unstudied, not safe)

BEST FIT: Gut inflammation / IBD support (adjunctive) · NF-kB-driven inflammatory conditions · Those seeking oral peptide options · Complementary to conventional IBD therapy

NOT IDEAL: Sole treatment for IBD (no human evidence) · Immunocompromised patients · Anyone requiring evidence-based treatment

STACKING

Can complement BPC-157 for gut healing (different mechanisms). LL-37 for antimicrobial + anti-inflammatory combo.

BOTTOM LINE

Promising anti-inflammatory mechanism through NF-kB inhibition. Entirely preclinical. If you have IBD, do not replace proven treatments. May be a future adjunctive option.

Thymosin Alpha-1

Tα1 • Zadaxin • Thymalfasin

COMPOUNDABLE (CAT 1) B: MODERATE EVIDENCE

Immune modulator approved in 35+ countries. Used for hepatitis, cancer immunotherapy support, and immune deficiency. The most evidence-backed immune peptide.

ROUTE	HALF-LIFE	FREQUENCY	CYCLE
Subcutaneous	~2 hours	2x weekly	6-12 months or ongoing

PROTOCOL

Dose: 1.6 mg subcutaneous, twice weekly
Timing: No specific time requirement; consistent schedule preferred
Cycle: 6-12 months for hepatitis/immune indications; ongoing for immunodeficiency
Approved in: 35+ countries (not FDA-approved in US, but compoundable as Cat 1)
Brand: Zadaxin (SciClone Pharmaceuticals)
Storage: Refrigerate. Ready-to-use formulation available.

🧬 IMMUNE SYSTEM GENETICS

`TLR2 (rs5743708)` – Toll-like receptor 2. Innate immune recognition. Tα1 enhances TLR-mediated signaling.
`HLA-A / HLA-B` – Major histocompatibility complex. Antigen presentation capacity.
`IL2RA (rs2104286)` – IL-2 receptor. T-cell proliferation and activation.

→ TLR2 variants affect innate immune response to Tα1
→ HLA type determines which antigens T-cells can recognize (relevant for cancer immunotherapy)
→ IL2RA variants influence T-cell expansion response to immune modulation

Thymosin Alpha-1 enhances your existing immune architecture. HLA type and TLR genetics determine the ceiling of immune response improvement. thepeptidelist.com/genetics

KEY CLINICAL EVIDENCE

· Hepatitis B: Improved sustained virologic response when combined with interferon (multiple RCTs)
· Hepatitis C: Enhanced treatment response in combination therapy
· Cancer: Improved immune reconstitution during chemotherapy (multiple trials)
· COVID-19: Reduced mortality in severe cases (retrospective data; not RCT-proven)

BLOODWORK

CBC with differential – Baseline, 4-8 weeks, quarterly
CMP – Baseline
CD4/CD8 T-cell counts – Baseline, quarterly (immune reconstitution)
Viral load – If hepatitis indication

COMMON: Injection site reactions (mild) · Generally well-tolerated · Minimal reported side effects in trials

CAUTION: Rare allergic reactions · Theoretical autoimmune flare (immune activation)

BEST FIT: Chronic hepatitis B/C (combination therapy) · Immune reconstitution during/after chemotherapy · Chronic or recurrent infections · Primary immunodeficiency support

NOT IDEAL: Organ transplant recipients (rejection risk) · Active autoimmune disease · Healthy immune system with no specific indication

STACKING

Used alongside standard antiviral or chemotherapy regimens. Not typically stacked with other peptides.

BOTTOM LINE

The most clinically validated immune peptide. Approved in 35+ countries. Excellent safety profile. The serious choice for immune modulation, not a biohacking experiment.

Comparison: GLP-1 Agonists

Head-to-head: the four GLP-1 options and when to choose each.

	Semaglutide	Tirzepatide	Retatrutide	Liraglutide
Mechanism	GLP-1	GIP + GLP-1	GIP + GLP-1 + Glucagon	GLP-1
Max Weight Loss	~15% (STEP 1)	~21% (SURMOUNT-1)	~29% (TRIUMPH-4)	~8% (SCALE)
Dosing	Weekly SC or daily oral	Weekly SC	Weekly SC	Daily SC
FDA Status	Approved	Approved	Phase 3	Approved (generic)
CV Outcomes	Yes (SELECT)	Pending	Pending	Yes (LEADER)
GI Side Effects	Moderate (44% nausea)	Moderate (33% nausea)	High (43% nausea)	Moderate
Cost	$$$	$$$$	N/A (trial only)	$ (generic)
Key Advantage	Most data, oral option	Best approved efficacy	Highest efficacy ever	Cheapest, mortality data
Key Genes	GLP1R, MC4R, FTO	GLP1R, GIPR, PCSK1	GLP1R, GIPR, GCGR	GLP1R, TCF7L2

Decision Framework

Cost is primary concern? → Liraglutide (generic available)
Want maximum approved efficacy? → Tirzepatide (21% weight loss)
Need proven cardiovascular protection? → Semaglutide (SELECT trial)
Partial responder to GLP-1 alone? → Tirzepatide (add GIP) or trial for retatrutide
Oral option preferred? → Semaglutide (Rybelsus)
GLP1R low-responder genotype? → Consider dual/triple agonist (tirzepatide or retatrutide)

Comparison: Growth Hormone Options

Secretagogues vs direct GH replacement. Choosing the right approach.

	CJC/Ipamorelin	Sermorelin	MK-677	HGH 191aa
Type	GHRH + GHRP combo	GHRH analog	Oral GH secretagogue	Direct GH replacement
Route	SC 2-3x daily	SC daily (bedtime)	Oral daily	SC daily
GH Elevation	3-5x pulsatile	Moderate pulsatile	97% sustained	Supraphysiological possible
IGF-1 Impact	Moderate	Moderate	Significant	Highest
Glucose Impact	Minimal	Minimal	Significant (watch)	Significant
Pulsatility	Preserved	Preserved	Blunted	Not pulsatile
Evidence	Phase 1-2	Previously approved	Phase 2 (discontinued)	Approved (GHD)
Best For	Body recomp + recovery	Sleep + gentle GH	Needle-averse	Diagnosed GH deficiency
Key Risk	Low (clean profile)	Very low	CHF, glucose, appetite	Edema, carpal tunnel, glucose

Decision Framework

New to GH peptides? → Sermorelin (gentlest entry, once daily bedtime)
Want maximum natural GH elevation? → CJC-1295/Ipamorelin combo
Hate needles? → MK-677 (oral, but watch glucose and appetite)
Diagnosed GH deficiency? → HGH 191aa (direct replacement, most potent)
Sleep is primary goal? → Sermorelin or CJC/Ipa (bedtime dosing)
d3-GHR carrier? → Any secretagogue (you respond well to GH signaling)

Comparison: Healing Peptides

BPC-157 vs TB-500 vs GHK-Cu. Different mechanisms, different strengths.

	BPC-157	TB-500	GHK-Cu
Mechanism	VEGF + NO pathways	Actin dynamics + angiogenesis	Collagen + copper delivery
Scope	Local (inject near injury)	Systemic (whole body)	Skin + systemic
Best For	Tendons, ligaments, GI	Multiple injuries, cardiac	Skin, wounds, collagen
Route	SC (local injection)	SC (systemic)	SC or topical
Frequency	2x daily	2x weekly	Daily (SC) / 2x daily (topical)
Evidence	Preclinical (extensive)	Preclinical	Moderate (topical human data)
FDA Status	Cat 1 (compoundable)	Cat 1 (compoundable)	Cat 1 (compoundable)
Key Genes	VEGFA, NOS3, COL1A1	TMSB4X, ACTA2	COL1A1, MMP2, ATP7B
Stacking	Pairs with TB-500	Pairs with BPC-157	Pairs with either

Decision Framework

Single localized injury (tendon/ligament)? → BPC-157 (inject near site)
Multiple injury sites or systemic repair? → TB-500 (systemic action)
Skin rejuvenation or wound healing? → GHK-Cu (topical is low-risk)
Comprehensive healing protocol? → BPC-157 + TB-500 ("Wolverine Stack")
Post-surgical recovery? → BPC-157 + GHK-Cu (local repair + collagen)
All three? → Some practitioners use all three for comprehensive healing

Stack Synergy Matrix

Common combinations and what NOT to mix. Green = synergistic. Red = avoid. Gray = no interaction data.

PROVEN / COMMONLY USED STACKS

Stack	Rationale	Notes
BPC-157 + TB-500	Local repair + systemic repair	'Wolverine Stack.' Different mechanisms. No controlled combo studies.
CJC-1295 + Ipamorelin	GHRH + GHRP synergy	3-5x GH amplification. The standard GH combo.
Sermorelin + Ipamorelin	GHRH + GHRP (gentler)	Alternative to CJC combo. Bedtime protocol.
Semaglutide + Enobosarm	Weight loss + lean mass preservation	In clinical trials (QUALITY study). 71% less lean mass loss.
GHK-Cu (topical) + BPC-157	Skin repair + tissue repair	Complementary for post-procedure healing.
Selank + Semax	Anxiolytic + nootropic	'Russian nootropic stack.' Different cognitive targets.
Epitalon + MOTS-c	Telomeres + mitochondria	Comprehensive anti-aging. Both course-based.

DO NOT COMBINE

Combination	Why Not
GLP-1 agonist + GLP-1 agonist	Redundant mechanism. Increased GI side effects. No added benefit.
HCG + Gonadorelin (long-term)	Both stimulate LH pathway. Choose one for TRT adjunct.
MK-677 + Insulin	Both raise glucose. Dangerous hypoglycemia risk + insulin resistance compounding.
Multiple GH secretagogues	Receptor competition and desensitization. Pick one GHRH + one GHRP max.
NAD+ IV + PARP inhibitors	PARP inhibitors depend on NAD+ depletion for cancer cell death. NAD+ counteracts this.

THE MISSING PIECE

This Guide Tells You What the Protocols Are.

Your DNA Tells You Which One Is Yours.

Every peptide in this guide includes genetic variables that alter response.

But knowing the genes is only half the picture.

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