

THE PRACTICAL PEPTIDE GUIDE

A benefit-first, evidence-based overview of key peptides for metabolic health, skin, and recovery

Updated April 2026 | [Peptide Stack Research Series](#)

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What are peptides?

Peptides are short chains of amino acids, typically between 2 and 50, that act as signaling molecules in the body. They bind to specific receptors on cells and trigger targeted biological responses: fat metabolism, tissue repair, hormone release, immune modulation, skin regeneration.

Your body already produces thousands of them naturally. The compounds in this guide are either synthetic versions of those natural signals or modified analogs designed for greater stability, longer half-life, or stronger receptor binding.

Unlike broad-spectrum pharmaceuticals that affect multiple systems at once, peptides tend to act on specific pathways. That specificity is a big part of why researchers find them interesting.

How to prepare and use research peptides

Most research peptides arrive as a freeze-dried powder (lyophilized) in a sealed vial. Before use, they need to be reconstituted with bacteriostatic water.

What you need:

Peptide vial (lyophilized powder)

Bacteriostatic water (BAC water)

Alcohol swabs

Sterile syringe with needle

Insulin syringes for dosing

Reconstitution steps:

1. Clean the rubber stoppers on both the peptide vial and the BAC water vial with an alcohol swab. Let them dry.
2. Draw the required amount of bacteriostatic water into a syringe. A common ratio is 1 mL or 2 mL per vial, depending on the desired concentration.
3. Insert the needle into the peptide vial and direct the water slowly down the inside wall of the glass. Do not shoot it directly onto the powder. Let it trickle down gently.
4. Do not shake the vial. Roll it gently between your palms until the powder is fully dissolved and the solution is clear.
5. Store the reconstituted peptide in the refrigerator (2-8 C). Most reconstituted peptides remain stable for 4-6 weeks when refrigerated.

Administration:

Most peptides in this guide are administered via subcutaneous injection, typically into the abdomen, thigh, or upper arm. Use an insulin syringe (29-31 gauge) for minimal discomfort.

A few things to keep in mind:

Always check the expiration date on both the peptide and bacteriostatic water before use

If the reconstituted solution appears cloudy, discolored, or contains particles, do not use it

Dispose of needles and syringes safely according to local regulations

Keep unreconstituted peptides stored in a cool, dry place. Most lyophilized peptides are fine at room temperature, but refrigeration extends shelf life

About this guide

This is not casual internet advice. Everything here is sourced from primary literature, official prescribing information, Eli Lilly press releases, FDA material, and peer-reviewed publications.

We wrote it because the peptide space is full of noise. Bold claims, recycled forum posts, and marketing dressed up as science. This guide separates what the research actually shows from what the internet wants you to believe.

Three things to know before reading:

- 1. If your goal is better blood sugar, weight, lipids, and blood pressure, the strongest evidence is with the incretin class: retatrutide, tirzepatide, and semaglutide.*
- 2. If your goal is skin quality, GHK-Cu has the cleanest signal, especially in topical use.*
- 3. If your goal is tendon recovery, tanning, anti-anxiety, or growth-hormone effects, the online claims are much stronger than the human evidence. We'll be honest about that throughout.*



Metabolic Health

the strongest evidence is with the **incretin class**: retatrutide, tirzepatide, and semaglutide.



Skin Quality

GHK-Cu has the cleanest signal, especially in topical use.



Recovery & Other

the online claims are much stronger than the human evidence.

A note on retatrutide

Retatrutide is still investigational as of April 2026. Eli Lilly confirms it is not FDA-approved and not publicly available. When this guide references retatrutide dosing, we are describing published study protocols, not an approved label.

This distinction matters. Keep it in mind.

How this guide is organized

We cover ten compounds, organized by evidence strength:

Deep chapters (full analysis):

- 1. Retatrutide**
- 2. Tirzepatide**
- 3. Semaglutide**

4. GHK-Cu

5. BPC-157

Quick-reference profiles:

6. TB-500

7. Ipamorelin

8. Melanotan II

9. Selank

10. NAD+

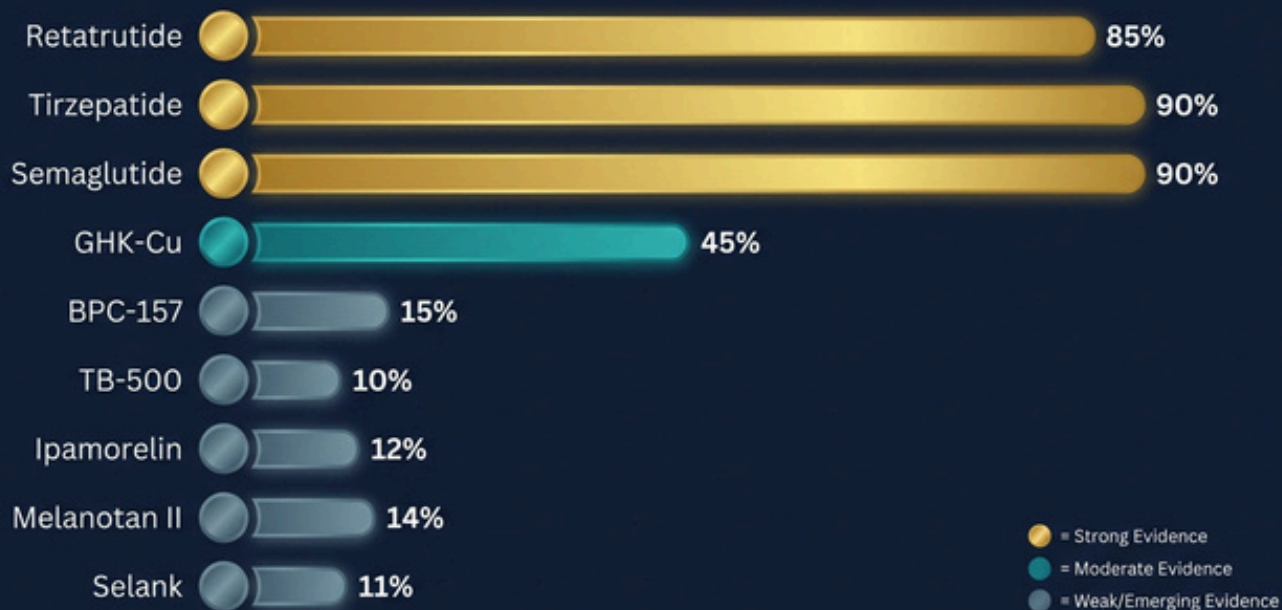
Each chapter follows the same structure: how the compound works, why people are interested, what the research actually says, practical takeaways, and an honest assessment of limitations.



At a glance: the comparison table

Compound	Primary interest	Best-fit goal	Evidence strength	Status
<u>Retatrutide</u>	Very large weight reduction, strong metabolic signal	Weight, A1c, triglycerides, LDL, blood pressure	Strong (Phase 2 + Phase 3 topline)	Investigational
<u>Tirzepatide</u>	Excellent weight reduction + glucose control	Weight, A1c, lipids, blood pressure	Strong (approved drug)	FDA approved
<u>Semaglutide</u>	Broad metabolic + cardiovascular benefit	Weight, A1c, CV risk, MASH	Strong (approved drug)	FDA approved
<u>GHK-Cu</u>	Skin texture, firmness, wrinkle depth	Skin quality, potential hair interest	Small but real human data	Topical use most defensible
<u>BPC-157</u>	Recovery reputation	Experimental recovery interest	Very weak human evidence	Unapproved
<u>TB-500</u>	Tissue repair narrative	Experimental recovery interest	No real human evidence for injectable use	Unapproved
<u>Ipamorelin</u>	GH secretagogue appeal	Body composition / recovery interest	Weak and narrow	Unapproved
<u>Melanotan II</u>	Tanning + libido	Cosmetic tanning	Small efficacy signal, safety concerns	Unapproved
<u>Selank</u>	Anti-anxiety, calm focus	Experimental mood support	Limited human evidence	Unapproved
<u>NAD+</u>	Cellular energy, longevity	Anti-aging, mitochondrial function, cognitive support	Established biochemistry, growing clinical interest	Coenzyme (naturally occurring)

Peptide Evidence Strength: A Comparative Analysis



CHAPTER 1

Retatrutide



How it works

Retatrutide is a triple agonist. It activates three receptor pathways simultaneously.

GLP-1 receptor: Slows gastric emptying so food stays in the stomach longer. Signals satiety to the brain. Improves insulin sensitivity and stabilizes blood sugar. This is the same pathway that makes semaglutide effective.

GIP receptor: Improves how the body handles post-meal glucose spikes. Enhances insulin response to food. Reduces the severity of GI side effects compared to GLP-1 alone. This is what makes tirzepatide more tolerable than semaglutide for most people.

Glucagon receptor: This is the differentiator. Glucagon signals the liver to break down stored glycogen, directly stimulates fat oxidation at the cellular level, and increases resting metabolic rate through thermogenesis. Your body burns more energy at rest.

The combination means retatrutide simultaneously reduces food intake, improves metabolic processing, and actively increases energy expenditure. No other compound in this guide acts on all three pathways at once.

Why people are excited

The weight reduction signal is unusually strong, and the metabolic story goes well beyond the scale.

In published Lilly and peer-reviewed data, retatrutide has shown:

Very large average weight reduction

Strong HbA1c reduction

Favorable movement in triglycerides, LDL, total cholesterol, and blood pressure

Continued trajectory without a clear early plateau in some readouts

For anyone focused on metabolic markers, this is the most interesting investigational compound on this list.

What the research actually shows

Phase 2 obesity data (published in The New England Journal of Medicine):

Mean weight reduction up to 24.2% at 48 weeks

Exploratory improvements in blood pressure, triglycerides, LDL, total cholesterol, HbA1c, fasting glucose, and insulin

Phase 2 type 2 diabetes data (published in The Lancet):

HbA1c reduction up to 2.02%

Body weight reduction up to 16.94%

Phase 3 diabetes update (Lilly, March 19, 2026):

A1c reduction of 1.7% to 2.0% at 40 weeks

Weight reduction up to 16.8%

TRIUMPH-4 release (Lilly, December 11, 2025):

Weight reduction up to 28.7% at 68 weeks (placebo-adjusted: 26.6%)

Average loss of 71.2 lbs from a baseline of 248.5 lbs

75% reduction in knee osteoarthritis pain scores (vs. 40% placebo)

14.1% of patients on 9 mg and 12.0% on 12 mg were completely pain-free

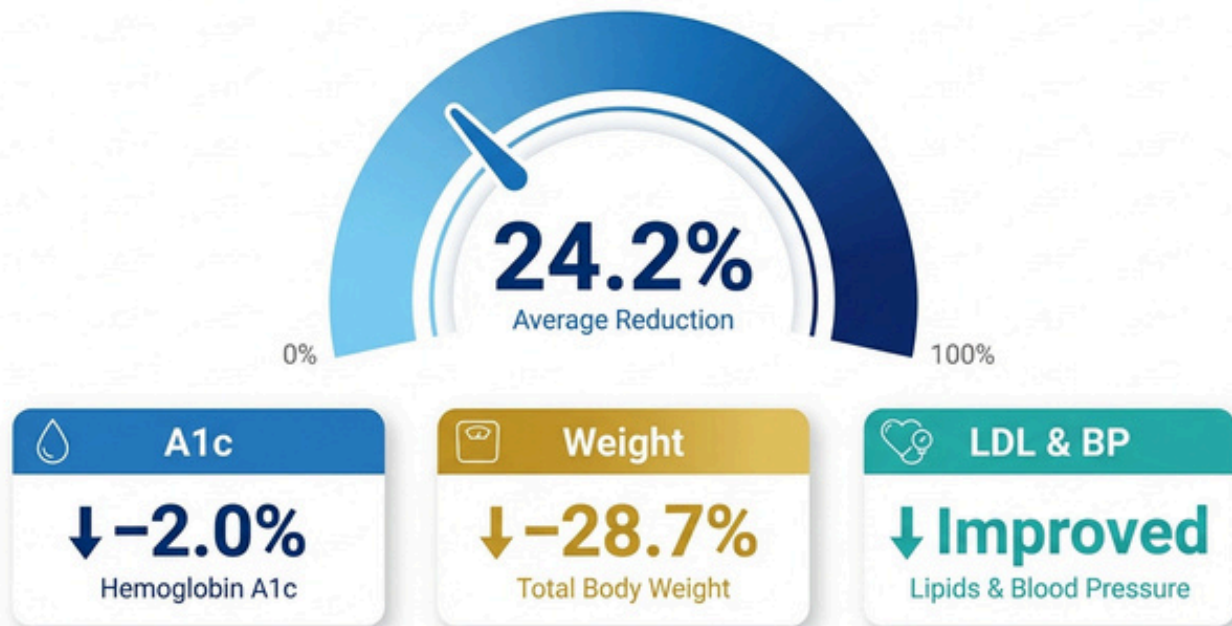
Lower non-HDL cholesterol, triglycerides, and systolic blood pressure

Six more late-stage TRIUMPH studies are underway with around 5,800 total participants. Readouts expected by end of 2026, covering obesity, obstructive sleep apnea, chronic low back pain, and liver disease

KEY TAKEAWAY

Retatrutide's combined metabolic signal across weight, glucose, lipids, and blood pressure is stronger than any other single compound in this guide. But it remains investigational. No approved label exists yet.

CLINICAL OUTCOMES: KEY METRICS IMPROVEMENT



Dosing from study protocols

What the published trials describe:

Once-weekly subcutaneous administration

Slow titration schedule

Starting point commonly 2 mg once weekly

Some Phase 2 arms used 4 mg starts depending on the target maintenance level

Phase 3 diabetes arms escalated every 4 weeks: 2 mg, then 4 mg, then 9 mg, then 12 mg

What about splitting the weekly amount? We did not find Lilly trial evidence supporting split dosing. All published studies describe once-weekly administration. "Splitting the dose" is a community practice, not a protocol-backed standard.

A NOTE ON REAL-WORLD STARTING DOSES

While Lilly's trials use 2 mg as the starting point, many independent researchers have found that 2 mg in week one is too aggressive, especially for people new to incretin compounds. The GI side effects (nausea, diarrhea, vomiting) can be significant at that dose without prior exposure.

A widely adopted community approach is to start much lower:

Week 1: 250 mcg (0.25 mg)

Week 2: 500 mcg (0.5 mg)

Week 3: 1 mg

Week 4: 1.5 mg

Week 5: 2 mg

Increase by 0.5 mg per week until reaching 2 mg. From there, many researchers report that 2 mg per week is their long-term sweet spot, with meaningful results and manageable side effects, without needing to push to the higher trial doses of 9 mg or 12 mg.

This is not from Lilly's protocol. It is based on widespread community experience. The logic is simple: a gentler ramp means fewer GI issues, better adherence, and for many people, 2 mg is enough.

The carbohydrate question

There is no primary-source evidence showing that people on retatrutide do better with purposefully higher carb intake. But there is a practical case for it.

What the evidence supports: smaller meals, slower eating, lower-fat meals when nausea is active, steady hydration, and adequate protein to preserve lean mass.

What the trial data does confirm: Retatrutide significantly improves insulin sensitivity. Your body becomes markedly more efficient at shuttling glucose into muscle glycogen. That is a published finding from the Phase 2 and Phase 3 data, not speculation.

Why that matters for training: If your insulin sensitivity is meaningfully improved, your muscles can utilize carbohydrates more efficiently. For anyone training consistently, this creates a practical window to increase clean carbohydrate intake and take advantage of the improved metabolic environment. Better glycogen replenishment, better workout fuel, better recovery.

PRACTICAL MACRO ADJUSTMENT

A commonly discussed approach among researchers who train while on retatrutide:

Before retatrutide (example baseline):

15% fat / 35% protein / 50% carbs

Adjusted for improved insulin sensitivity:

13% fat / 30% protein / 57% carbs

- Shift 5-8% of total calories toward clean carbs (rice, oats, potatoes, fruit)
- Concentrate the extra carbs around training windows (pre and post workout)
- On heavy training days, carbs can push toward 60% if appetite allows
- Keep protein at 30%+ minimum. Lean mass preservation is critical on any GLP-1 compound, since weight reduction can pull from muscle if protein is too low
- Keep fat at 12-15% minimum for hormonal health

The practical challenge: retatrutide's appetite suppression can make it hard to eat enough, period. Easy-to-digest carbs around training become doubly important. They are the most practical way to maintain energy and performance when total food intake drops.

Honest tradeoffs

Still investigational, no approved retail label

GI side effects are significant during escalation:

Nausea: 43% of participants

Diarrhea: 33%

Vomiting: 21%

Dysesthesia (abnormal skin sensations) emerged as a new safety signal in Phase 3, something not seen in the earlier mid-stage trial:

8.8% on the 9 mg dose

20.9% on the 12 mg dose

0.7% on placebo

Lilly reports it was mild for most patients and rarely caused discontinuation

Discontinuation rates were notable: 12.2% at 9 mg, 18.2% at 12 mg (vs. 4% placebo). Some discontinuations were attributed to "perceived excessive weight loss"

No long-term safety data beyond study durations

What's coming next (2026 pipeline)

Retatrutide's Phase 3 story is far from over. Six additional TRIUMPH trials are expected to report by end of 2026, covering:

Obesity (broader population beyond osteoarthritis)

Obstructive sleep apnea

Chronic low back pain

Metabolic dysfunction-associated steatotic liver disease (MASLD)

Cardiometabolic outcomes

Maintenance dosing strategies

With around 5,800 participants enrolled across these trials, the evidence base will expand dramatically this year.

BOTTOM LINE

Retatrutide looks like the most powerful metabolic compound on this list. If the question is "which one moves body weight and metabolic markers the most?" then retatrutide is the answer. If the question is "which one has an approved label and real-world guidance?" that's tirzepatide and semaglutide.

7 non-negotiable retatrutide rules

The data on retatrutide is impressive. 24% average body weight reduction in 48 weeks. Triple receptor activity. Metabolic markers moving across the board.

But none of that matters if the compound is used without a system around it.

We've seen it over and over in the community: someone gets access, injects, and assumes the drug will handle the rest. Three months later they've dropped 30 or 40 pounds and somehow look worse. Softer. Flatter. The scale went down but the mirror tells a different story.

That's what happens when aggressive fat loss runs without muscle protection, hormonal optimization, or an exit strategy. On retatrutide or any GLP-1, it happens fast.

Retatrutide is not a weight loss tool. It is a metabolic reprogramming tool. But only if you treat it like one.

Here are seven rules we consider non-negotiable.

This section is for educational purposes only and is not medical advice. Always consult a qualified healthcare professional before starting any protocol.

Rule 1: The compound is the accelerant, not the plan

Retatrutide suppresses appetite, improves insulin sensitivity, and speeds up fat mobilization. What it does not do: build muscle, protect lean tissue, or teach your body to maintain results after the last injection.

Three things need to be in place before anything goes in:

Structured resistance training running the entire time

A daily protein target that's established and tracked

A defined plan for what happens when the compound stops

Without all three, there is no protocol. There's just a weekly injection and a hope that it works out.

Rule 2: Hormonal environment first

Running a deep caloric deficit without adequate anabolic signaling doesn't selectively burn fat. It burns everything. Muscle is metabolically expensive tissue, and in a low-testosterone environment your body has zero incentive to keep it.

Before starting retatrutide, confirm:

Total testosterone is actually optimized, not just "within range." 400 ng/dL and 900 ng/dL both read as normal on a lab report. The difference in outcome is enormous.

Free testosterone has been checked. Elevated SHBG can make solid total numbers functionally irrelevant.

If free T is suboptimal and you're not on replacement therapy, that gets addressed first or at the same time.

Coming out the other side leaner and stronger requires the hormonal foundation to support it. Without that foundation, the deficit just eats into your muscle.

(This is not medical advice. Work with a physician before making any hormonal decisions.)

Rule 3: Baseline bloodwork and slow titration

The common approach is to source retatrutide, pick a dose based on a forum thread, and inject. No labs. No titration schedule. No real understanding of where things stand before the compound enters the picture.

Retatrutide activates three receptor pathways at once. The glucagon receptor in particular raises resting heart rate and increases energy expenditure. If cardiovascular markers are already off, the compound will make that worse before it improves anything.

Recommended pre-cycle panel:

Hormones: Total T, free T, estradiol, SHBG, LH, FSH

Metabolic: Fasting glucose, insulin, HbA1c, ALT/AST

Cardiovascular: Full lipid panel, CRP, resting heart rate, blood pressure

Titration approach:

Begin at 0.5 mg for those new to incretin compounds

Increase no more frequently than every two weeks

Most researchers settle between 3 and 4 mg/week. Upper ceiling: 6 mg.

In the Phase 2 data, GI side effects roughly doubled when participants started at 8 mg compared to those who titrated up from 1 mg. Going slower costs nothing. Going too fast costs weeks of nausea and potentially abandoned protocols.

Rule 4: This is not tirzepatide

Anyone transitioning from tirzepatide needs to drop their assumptions at the door.

Tirzepatide acts on two receptors: GLP-1 and GIP. Retatrutide acts on three: GLP-1, GIP, and glucagon. That third receptor is a different animal. Glucagon drives glycogen breakdown in the liver, directly promotes fat oxidation, and raises resting metabolic rate through thermogenesis.

That triple activity is why retatrutide outperforms tirzepatide in head-to-head Phase 2 comparisons. It's also why the dosing logic is completely different.

Tirzepatide escalation schedules do not apply here

The Phase 2 data showed 8 mg and 12 mg delivering nearly identical fat loss outcomes, with fewer adverse events at the lower dose

Sensible target range: 3 to 6 mg/week, reached gradually over 8 to 12 weeks

Rule 5: Eat on a schedule, not by feel

Retatrutide can suppress appetite so thoroughly that 700 or 800 calories a day feels normal. That doesn't make it safe. The body will quietly catabolize lean tissue to compensate, and by the time it shows, the damage is already weeks old.

Daily nutrition framework:

Protein floor: 1 g per pound of lean body mass (typically 160 to 220 g/day)

Lead every meal with the protein source

Total daily intake stays above 1,600 to 1,800 calories no matter how little hunger there is

Carbohydrates concentrated around training sessions

Meals go on a schedule with alarms. Appetite is no longer a reliable signal.

Anything below that calorie floor is not optimized fat loss. It's a starvation pattern: suppressed thyroid output, spiking cortisol, accelerating muscle loss, and setting up the exact weight rebound the protocol was supposed to prevent.

Rule 6: Monitor cardiovascular markers throughout

Most people running retatrutide fixate on the scale. Meanwhile resting heart rate is climbing, HRV is trending down, and blood pressure is drifting upward without anyone noticing.

Phase 2 trial data shows a dose-dependent rise in resting heart rate on retatrutide, peaking around 6 to 7 BPM above baseline at 12 mg, typically between weeks 20 and 24.

What to track:

Morning resting heart rate, measured before getting out of bed

If resting HR increases more than 8 to 10 BPM over your pre-cycle number, pause and reassess

Weekly blood pressure readings, not just when something feels off

HRV if you have the tools for it. A sustained downward trend is the earliest warning sign, before anything else becomes obvious.

Without the baseline from Rule 3, none of these numbers have context.

Rule 7: The exit strategy comes first

Clinical data on GLP-1 discontinuation is clear: weight regain averages 0.4 kg per month after stopping, with full return to pre-treatment weight projected within 1.7 years. Once retatrutide clears the system, appetite regulation doesn't taper. It vanishes, and what replaces it is rebound hunger stronger than anything before the protocol started.

The exit has to be designed before the first injection, not figured out after the last one.

Tapering framework:

No cold stops. Reduce dose over the final 4 to 6 weeks.

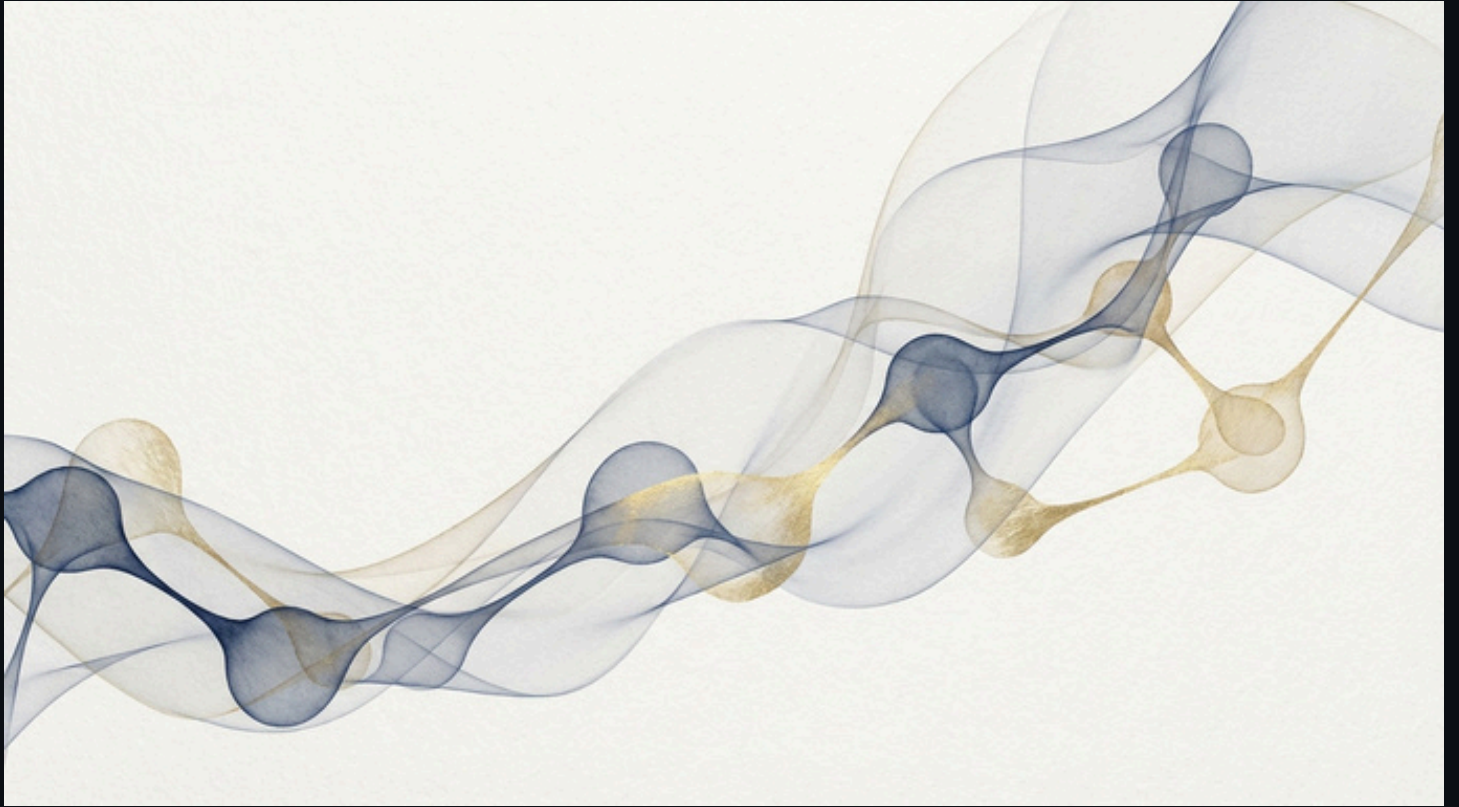
Two weeks at half the working dose, then two weeks at a quarter

Begin moving calories back toward maintenance during the last four weeks of the taper, not after

Identify true maintenance intake for the new body composition

Training consistency has to be a locked-in habit before the compound is gone

Retatrutide opens a window. Whether the results last depends entirely on what gets built during that window.



CHAPTER 2

Tirzepatide



How it works

Tirzepatide is a dual agonist that activates two receptor pathways.

GLP-1 receptor: Slows gastric emptying, signals fullness to the brain, and improves insulin sensitivity. The same mechanism behind semaglutide, but here it works in tandem with a second pathway.

GIP receptor: Glucose-dependent insulintropic polypeptide. It enhances insulin secretion in response to food, improves how the body processes post-meal blood sugar, and appears to buffer against some of the GI side effects that pure GLP-1 agonists cause. This is why tirzepatide tends to be better tolerated than semaglutide at equivalent weight loss levels.

The dual mechanism produces additive metabolic effects: appetite suppression from GLP-1, plus improved glucose handling and reduced nausea from GIP. Stronger weight and glucose outcomes than single-agonist drugs, with a more manageable side effect profile.

Why it stands out

Tirzepatide is Lilly's currently approved heavyweight. It combines strong blood sugar control with unusually large weight reduction for an approved medicine, and it touches multiple markers at once: HbA1c, body weight, triglycerides, LDL trend, and systolic blood pressure.

What the label and trials show

Official Zepbound dosing:

Start: 2.5 mg once weekly for 4 weeks

Escalate by 2.5 mg increments every 4+ weeks

Range: 5 mg, 7.5 mg, 10 mg, 12.5 mg, 15 mg

Once weekly only. The evidence base does not support split dosing.

Trial results:

SURPASS-2: Tirzepatide lowered HbA1c more than semaglutide 1 mg, and produced more average weight reduction

SURMOUNT-1: Weight reduction reached approximately 22.5%

KEY TAKEAWAY

If retatrutide is the exciting next-wave molecule, tirzepatide is the "available now" version of that metabolic ambition. Approved, accessible, and meaningfully effective across multiple markers.

Food strategy

The most evidence-backed approach is not "eat more carbs." Better defaults: smaller meals, slower eating, lower-fat and easier-to-digest meals during nausea, steady hydration, and enough protein to support lean mass.

The image is split into two vertical panels. The left panel has a teal background and features a vial of Tirzepatide 10mg. Behind the vial is a teal line graph with an upward-pointing arrow. The right panel has a blue background and features a vial of Semaglutide 5mg. Behind the vial is a blue line graph with an upward-pointing arrow. Both vials are labeled 'PEPTIDE-STACK' at the top and 'FOR RESEARCH USE ONLY' at the bottom.

TIRZEPATIDE 10MG
Weight: -22.5% | A1c: Superior to Semaglutide

SEMAGLUTIDE 5MG
Weight: -14.9% | CV Risk + MASH Approved

CHAPTER 3

Semaglutide



How it works

Semaglutide is a single-agonist GLP-1 receptor agonist. It mimics the natural incretin hormone GLP-1, which the gut releases after eating.

What GLP-1 activation does: Slows gastric emptying so food stays in the stomach longer, producing sustained fullness. Signals the hypothalamus to reduce appetite. Enhances insulin secretion when blood sugar is elevated (glucose-dependent, so it doesn't cause hypoglycemia on its own). Suppresses glucagon release after meals, reducing excess glucose production by the liver.

Semaglutide is engineered with a fatty acid side chain that binds to albumin in the blood, extending its half-life to roughly 7 days. That's what allows once-weekly dosing instead of the multiple daily injections that older GLP-1 drugs required.

The compound also appears to have direct effects on cardiovascular risk markers and liver fat independent of weight loss, though the exact mechanisms are still being studied.

Why it still matters

Semaglutide may feel "older" compared to tirzepatide and retatrutide, but it remains one of the most deeply validated metabolic compounds available. The story has expanded well beyond weight:

The current Wegovy label reflects cardiovascular risk reduction

The 2026 label includes MASH (metabolic dysfunction-associated steatohepatitis)

The evidence base is enormous

Dosing

Wegovy injection, official escalation:

0.25 mg, then 0.5 mg, then 1.0 mg, then 1.7 mg, then 2.4 mg (once weekly, each step is 4 weeks)

2026 label update: in selected adults who tolerate 2.4 mg for 4+ weeks and need further reduction, increase up to 7.2 mg once weekly is permitted

Data

STEP 1: Approximately 14.9% weight reduction at 68 weeks

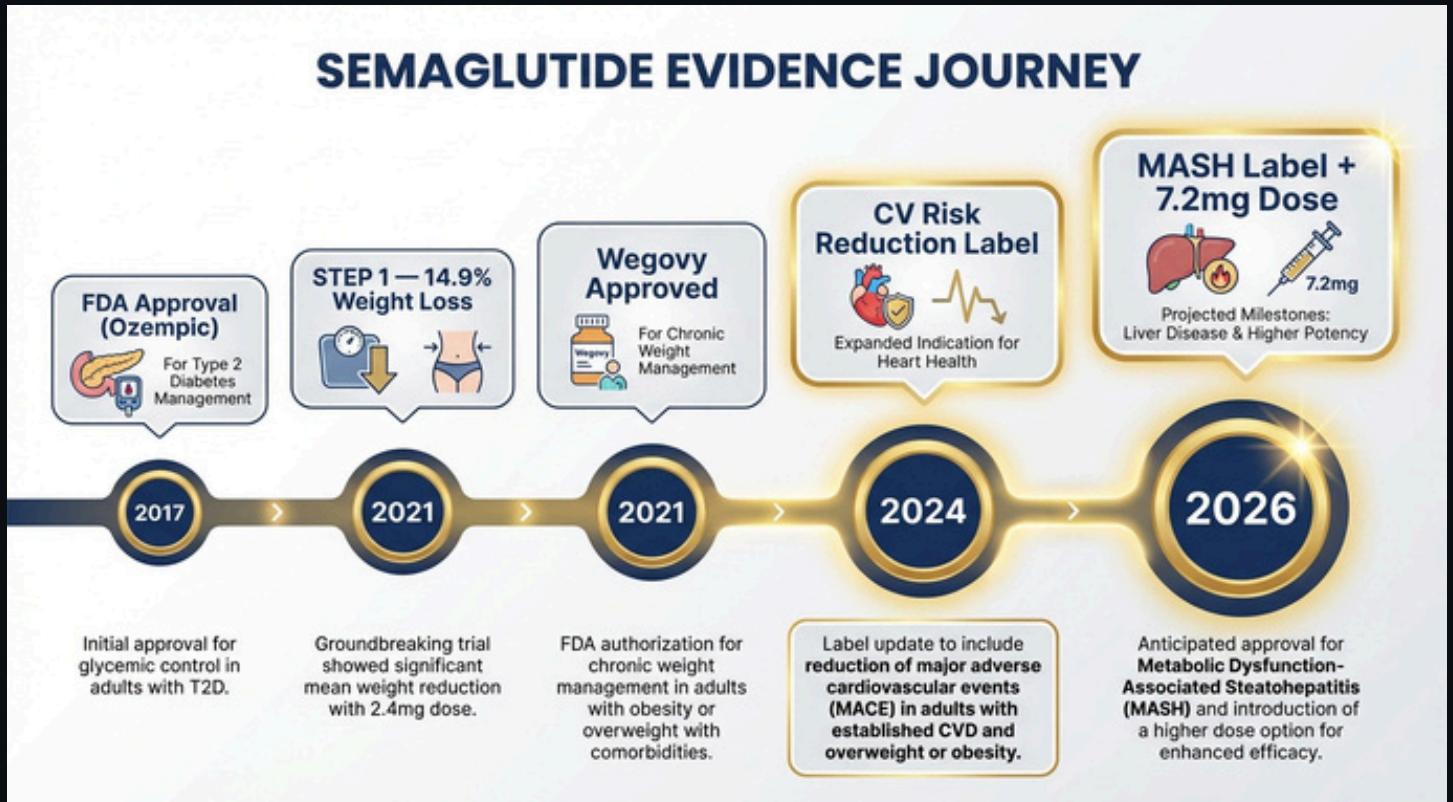
Long-term weight reduction

Cardiovascular risk reduction in adults with established CVD and obesity/overweight

MASH with moderate to advanced fibrosis (accelerated approval)

BOTTOM LINE

Semaglutide is one of the most useful compounds in real-world practice. Tirzepatide may have a stronger average weight profile, but semaglutide remains deeply validated and broadly useful.



GHK-Cu



How it works

GHK-Cu (glycyl-L-histidyl-L-lysine copper complex) is a naturally occurring tripeptide found in human plasma, saliva, and urine. It has a strong affinity for copper ions, and this copper-peptide complex is what drives its biological activity.

Collagen and skin remodeling: GHK-Cu stimulates the production of collagen types I and III, along with decorin and glycosaminoglycans. These are the structural components responsible for skin firmness, elasticity, and hydration. It also promotes fibroblast activity, the cells that build and maintain the extracellular matrix.

Anti-inflammatory signaling: GHK-Cu suppresses inflammatory cytokines including TNF-alpha and IL-6. This reduces chronic low-grade inflammation in aging skin and may accelerate wound healing.

Antioxidant support: The copper complex acts as a superoxide dismutase mimetic, helping neutralize free radicals that drive photoaging and cellular damage.

Tissue remodeling: GHK-Cu modulates the balance between metalloproteinases (which break down damaged tissue) and their inhibitors (which protect new tissue). This controlled demolition-and-rebuild cycle is what gives it potential across both skin and wound healing applications.

Topical delivery is the most defensible application. Injectable use is discussed in communities but has a much thinner evidence base.

A different goal entirely

If the first three chapters were about metabolic health, this chapter shifts to the surface. GHK-Cu's strongest case is cosmetic skin quality: texture improvement, increased elasticity, wrinkle depth reduction, and skin density and thickness.

What the human data says

The studies are small, but they are real:

Increased procollagen synthesis in a pilot study

Improvement in photoaged facial skin over 12 weeks

An 8-week randomized, double-blind study showing better wrinkle volume and wrinkle depth reduction than vehicle and a comparator peptide product

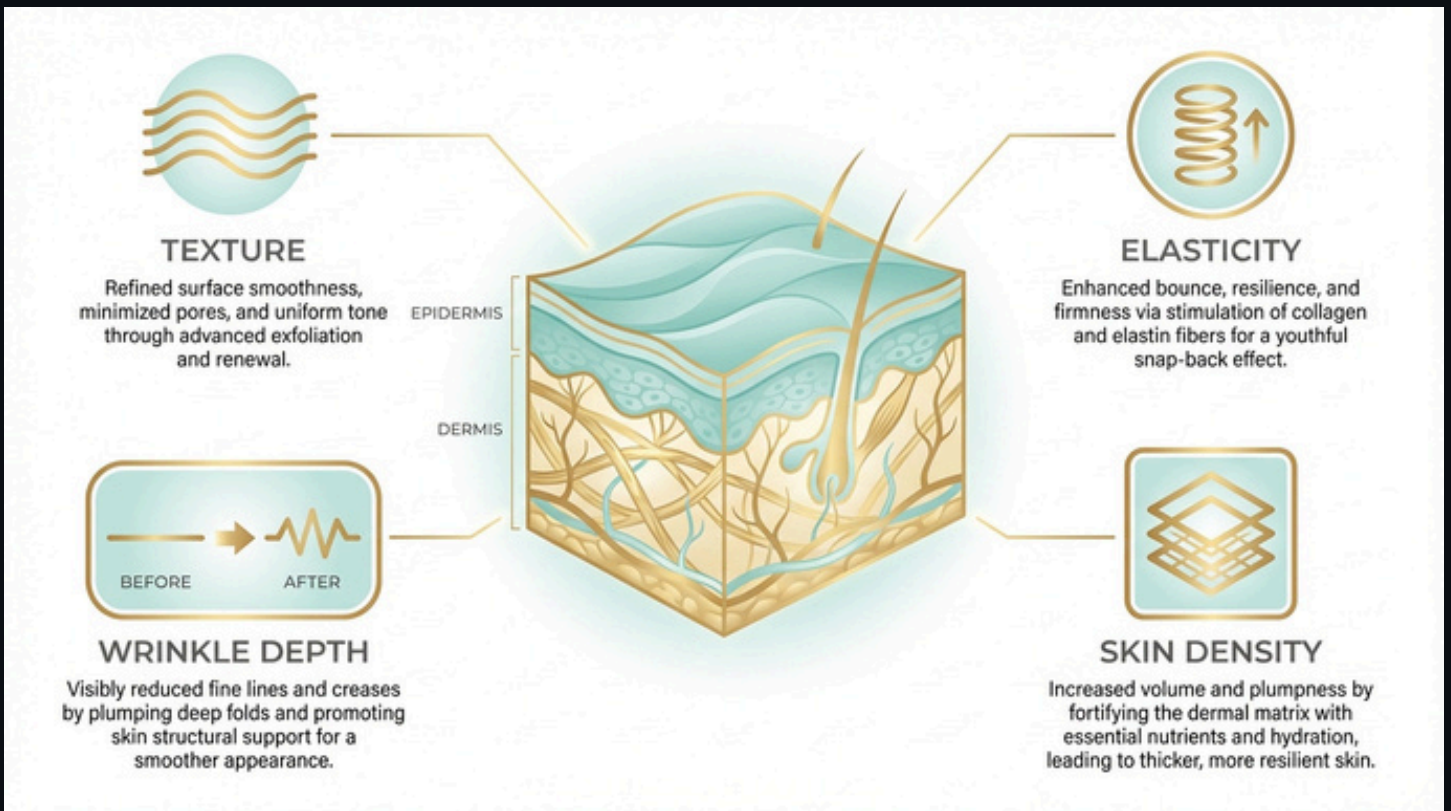
Where the evidence is weaker: hair growth, systemic (injectable) use, and oral use.

Formulation matters, a lot

The literature repeatedly emphasizes that GHK-Cu is not easy to deliver through skin. Carrier systems make a significant difference. Stable, well-formulated topical products are more credible than generic peptide marketing.

KEY TAKEAWAY

GHK-Cu is the best compound on this list for a skin-first goal. Promising for wrinkles, texture, and elasticity. Not as convincing for hair as the internet often claims, but still one of the few peptides with a believable skin quality story backed by human data.



CHAPTER 5

BPC-157



How it works (based on animal data)

BPC-157 (Body Protection Compound-157) is a synthetic pentadecapeptide derived from a protective protein found in human gastric juice. Its 15-amino-acid sequence does not occur naturally in this exact form, but it is based on a fragment of a larger protein the stomach produces.

Angiogenesis: In animal models, BPC-157 promotes the formation of new blood vessels at injury sites. Improved blood supply accelerates delivery of oxygen and nutrients to damaged tissue.

Nitric oxide system: BPC-157 appears to interact with the nitric oxide (NO) system, modulating blood vessel tone and promoting vascular repair. Animal studies show it can counteract the effects of NO-system blockade.

Growth factor upregulation: In rats, BPC-157 increases expression of growth hormone receptor and several growth factors involved in tendon, muscle, and gut tissue repair.

Gut cytoprotection: The compound is derived from gastric juice for a reason. Animal data suggests it protects gut lining integrity, reduces ulcer formation, and accelerates healing of damaged intestinal tissue.

Important caveat: Nearly all mechanism data comes from rat and mouse studies. The human evidence base for BPC-157 is extremely limited. The gap between animal findings and proven human efficacy remains large.

Why people keep asking about it

BPC-157 has a massive online reputation. People associate it with tendon recovery, ligament support, faster healing, and gut support.

That is the story people want to believe. Here is where we have to be direct about the gap between reputation and evidence.

What the human evidence actually looks like

As of April 2026, the human evidence is extremely thin:

One 2025 IV safety pilot in two healthy adults

No convincing human efficacy trial showing tendon, ligament, muscle, or gut-healing benefit

The marketing story is dramatically larger than the clinical story.

Why it still belongs in this guide

BPC-157 is included because people ask about it constantly. But it is not in the same evidence class as tirzepatide, semaglutide, retatrutide, or even topical GHK-Cu.

HONEST ASSESSMENT

Think of BPC-157 as an experimental recovery compound with far more hype than confirmed human benefit. The animal data is interesting. The human data barely exists.



QUICK-REFERENCE

Profiles



TB-500

How it works: TB-500 is a synthetic version of a 43-amino-acid region of thymosin beta-4, a protein involved in cell migration and blood vessel formation. In animal models, it promotes actin binding, which helps cells move to injury sites. It also upregulates angiogenesis and reduces inflammation at the tissue level.

Why people like it: Marketed for tissue repair and recovery.

What the evidence says: We did not find a published human clinical trial for injectable TB-500. The closest meaningful human data in the thymosin beta-4 family is ophthalmic (eye disease) research, which does not

validate systemic TB-500 for whole-body recovery.

Short read: Interesting biologic family. Very weak support for the common recovery claims.

Ipamorelin

How it works: Ipamorelin is a growth hormone secretagogue. It binds to the ghrelin receptor (GHSR) in the pituitary gland, triggering a pulse of growth hormone release. Unlike older secretagogues (GHRP-6, hexarelin), ipamorelin is selective. It stimulates GH release without significantly raising cortisol or prolactin. The GH pulse is dose-dependent and mimics the body's natural pulsatile release pattern.

Why people like it: Growth hormone release, recovery, lean mass appeal.

What the evidence says: The human evidence is narrow. A small randomized trial in postoperative ileus did not show a strong main efficacy signal.

Short read: Popular in peptide circles, but far less convincing in human outcomes than online marketing suggests.

Melanotan II

How it works: Melanotan II is a synthetic analog of alpha-melanocyte-stimulating hormone (alpha-MSH). It binds to melanocortin receptors, primarily MC1R (which stimulates melanin production in skin cells, producing a tan) and MC4R (which influences sexual arousal and appetite). The tanning effect comes from actual melanin synthesis, not UV exposure, though UV still accelerates the visible result.

Why people like it: Tanning effect and libido signal.

What the evidence says: Small human studies suggest physiologic activity. But safety concerns are meaningful enough that this is difficult to frame as a clean "skin health" compound.

Short read: More of a risky cosmetic/tanning compound than a skin improvement peptide. Approach with caution.

Selank

How it works: Selank is a synthetic analog of the natural immunomodulatory peptide tuftsin, with an added Pro-Gly-Pro sequence for stability. It modulates the balance of neurotransmitters including serotonin, dopamine, and norepinephrine. It also influences brain-derived neurotrophic factor (BDNF) expression, which plays a role in neuroplasticity and stress resilience. The compound has both anxiolytic and mild nootropic properties in animal and limited human data.

Why people like it: Calm focus, anti-anxiety reputation.

What the evidence says: Limited human evidence, mostly from small Russian studies. Some anxiolytic signal exists, but the evidence base is not deeply replicated.

Short read: The most psychologically interesting compound on this list, but the evidence doesn't compete with the incretin peptides for metabolic markers or with GHK-Cu for skin.



NAD+

How it works: NAD+ (nicotinamide adenine dinucleotide) is not a peptide. It is a coenzyme present in every cell, required for hundreds of metabolic reactions. It works in two main capacities.

Energy metabolism: NAD+ is essential for mitochondrial ATP production. It shuttles electrons through the electron transport chain, converting nutrients into usable cellular energy. Without adequate NAD+, mitochondria become less efficient and cells produce less energy.

Sirtuin activation: NAD+ is the required substrate for sirtuins (SIRT1-7), a family of enzymes that regulate DNA repair, inflammation, metabolism, and cellular stress responses. Sirtuins cannot function without NAD+. They are involved in gene silencing, circadian rhythm regulation, and inflammatory suppression.

DNA repair: NAD+ fuels PARP enzymes, which detect and repair DNA strand breaks. As NAD+ declines, DNA damage accumulates faster than it can be fixed.

The aging problem: NAD+ levels decline significantly with age, with studies reporting 30-50% reductions in key tissues by middle age. This decline contributes to mitochondrial dysfunction, reduced DNA repair capacity, and metabolic slowdown. Restoring NAD+ levels is one of the foundational targets in longevity research.

Delivery options: Subcutaneous injection, intravenous infusion, or oral precursors (NMN, NR). Injectable and IV routes offer higher bioavailability. Oral precursors are converted to NAD+ in the body but with variable efficiency.

Why people like it: Energy, cognitive clarity, anti-aging, and the idea that it makes other compounds work better by supporting the cellular machinery they depend on.

What the evidence says: The biochemistry is well established. NAD+ decline with age is documented, and its role in sirtuin activation and mitochondrial function is not disputed. Human clinical data on NAD+ supplementation (particularly injectable) for longevity outcomes is still growing. Oral precursors like NMN have shown promise in raising blood NAD+ levels in clinical trials, but long-term outcome data is limited.

Short read: Not flashy, but it supports the cellular infrastructure that everything else depends on. The science behind the decline is real. Whether supplementation meaningfully reverses aging outcomes in humans is still being answered.



TB-500 — Tissue
Repair Narrative



Ipamorelin — GH
Secretagogue



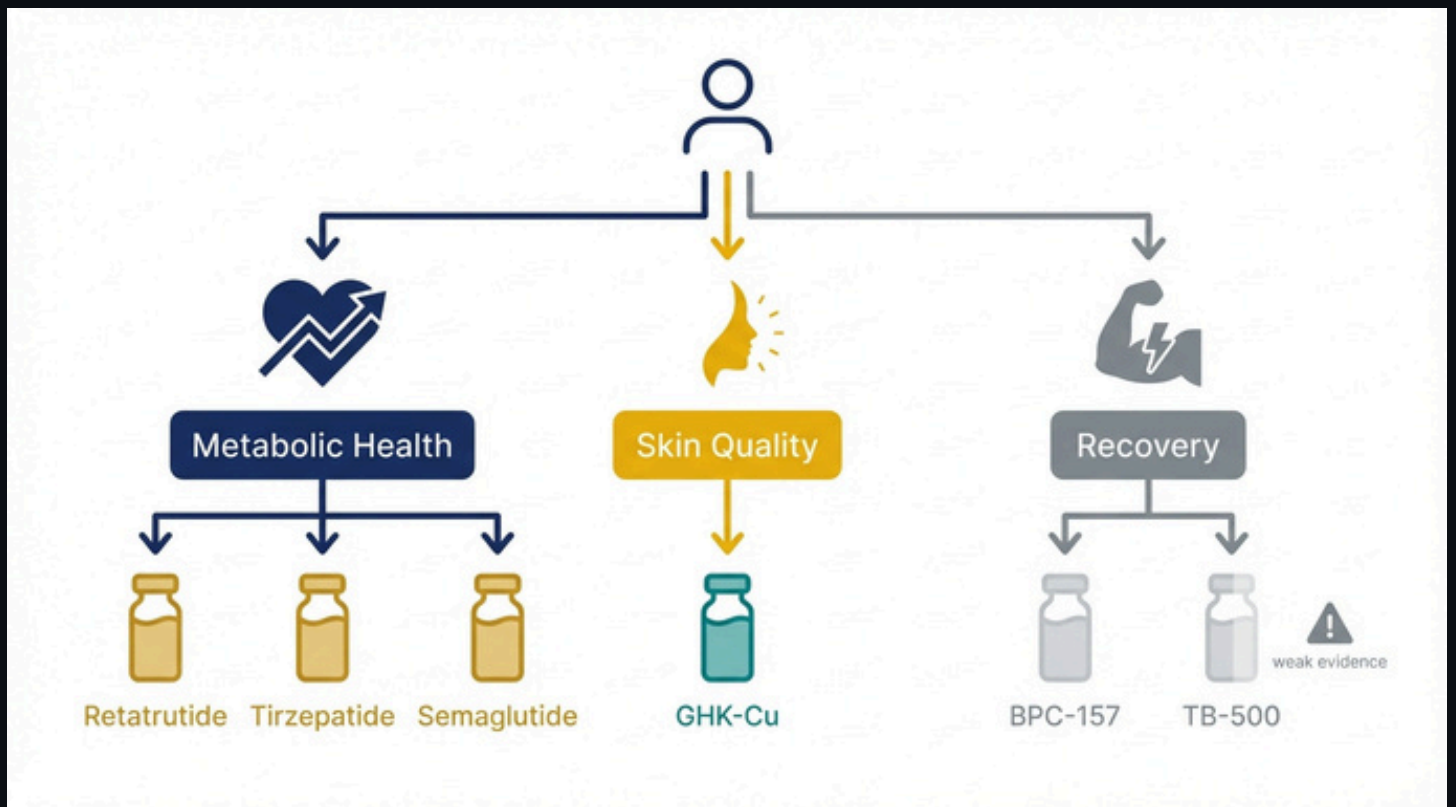
Melanotan II — Tanning
+ Safety Concerns



Selank —
Anti-Anxiety Interest

BEST PICKS

By goal



If your goal is metabolic improvement

Top three from this list:

1. Retatrutide (strongest combined signal, investigational)
2. Tirzepatide (strongest approved option)
3. Semaglutide (most deeply validated)

Why: Strongest combined data for weight, glucose, triglycerides, LDL trend, and blood pressure.

If your goal is skin quality

Top pick: GHK-Cu

Why: Actual human topical skin data. Small studies, but real ones.

If your goal is hair

The honest answer: None of the listed compounds has strong human hair data. GHK-Cu has the most interesting indirect case, but the evidence is still preliminary.

If your goal is recovery

The honest ranking: BPC-157 and TB-500 are popular. Human proof is weak. They do not currently deserve the same confidence as the metabolic compounds.

If your goal is longevity and cellular health

Top pick: NAD+

Why: The biochemistry is established. NAD+ decline with age is documented, and its role in energy production, DNA repair, and sirtuin activation is not disputed. It also supports the cellular infrastructure that other compounds depend on.



The bottom line

If you want the most honest summary from this guide:

Retatrutide is the most interesting Lilly research compound for future metabolic applications

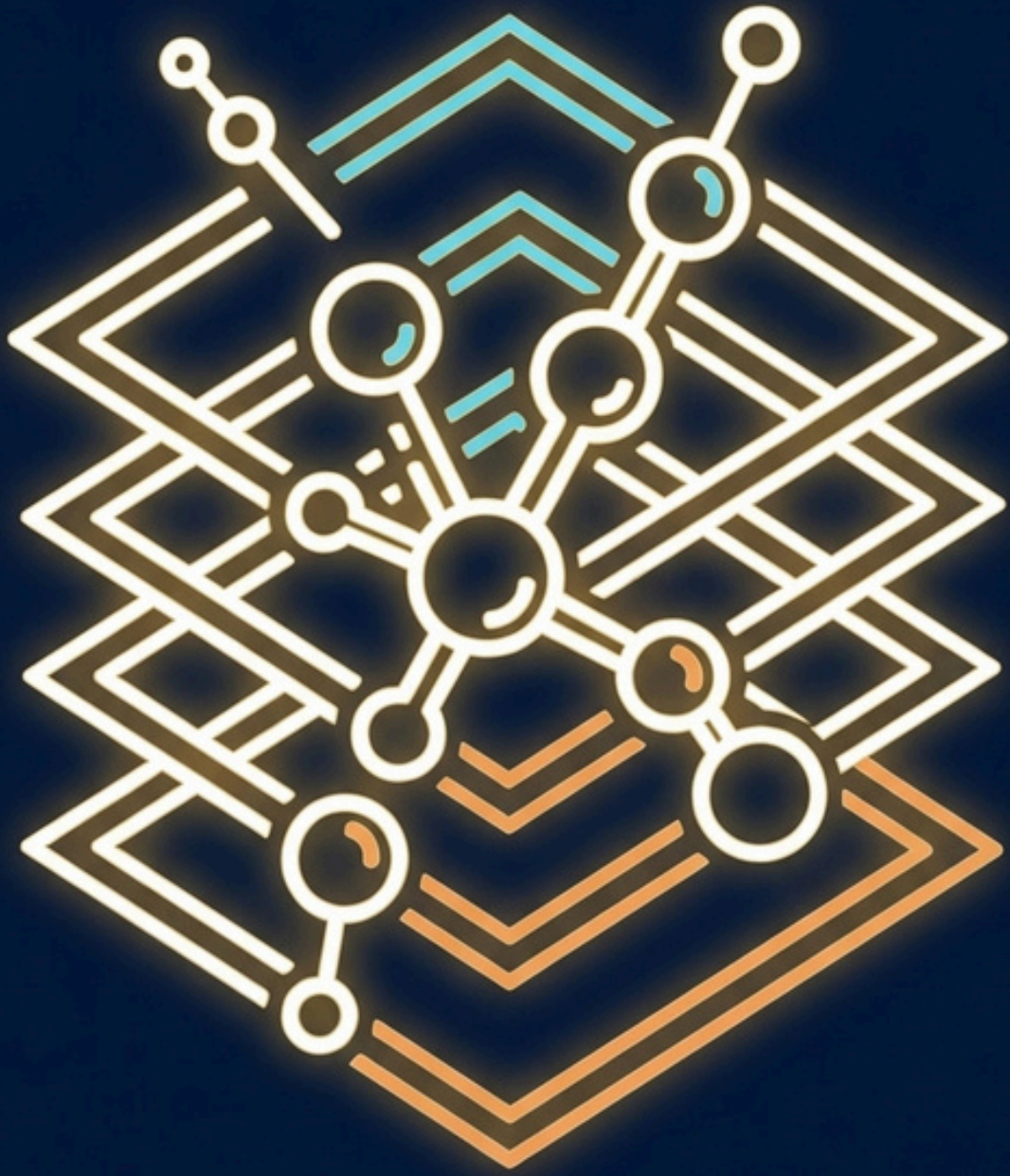
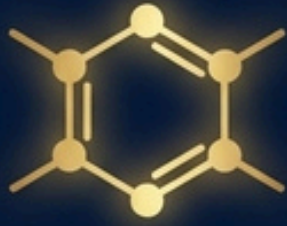
Tirzepatide is the strongest currently available option for weight reduction plus glucose improvement

Semaglutide remains one of the most validated metabolic compounds on the market

GHK-Cu is the best compound on this list for skin quality

NAD+ is the strongest candidate on this list for cellular health and longevity

The rest are interesting, but their online reputation outruns the human evidence



About Peptide Stack

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We built this guide because we believe informed researchers make better decisions, and the peptide space needs more honesty and less hype.

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