HelixNova
PEPTIDES

THE PEPTIDE ENCYCLOPEDIA

A complete educational breakdown of the science, the categories, the protocols, and the pharmacology behind the modern peptide world.

EDITION · 2026 RESEARCH REFERENCE
SCOPE · MECHANISMS · CATEGORIES · DOSING FRAMEWORKS · STACKING · CYCLING · APPENDICES
CLASSIFICATION · EDUCATIONAL / RESEARCH USE ONLY

READ FIRST

PURPOSE & DISCLAIMER

This document is an educational and scientific reference. It exists to explain how peptides work, how they are categorized, and how protocols are discussed in the research and clinical literature.

NOT MEDICAL ADVICE

Nothing here is a prescription, a recommendation to self-administer, or a substitute for a licensed physician. Many compounds in this document are not approved for human use and are sold legally only as research chemicals. Several are prescription-only drugs. Dosing ranges are reported because they appear in published literature, manufacturer data, and clinical practice, not as instructions. Hormonal and metabolic interventions carry real risks and belong under medical supervision with bloodwork.

The compounds covered range from well-studied, FDA-approved pharmaceuticals (semaglutide, tirzepatide, tesamorelin, insulin) to peptides with limited or animal-only human data (BPC-157, many others). Wherever possible, this guide flags what is actually known versus what is extrapolated, because in the peptide world the marketing almost always runs ahead of the evidence.

Read it to get the full picture. Make real decisions with a doctor who can run your labs.

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PART ONE

THE SCIENCE OF PEPTIDES

Before any protocol makes sense, you have to understand what these molecules are and why a signaling peptide behaves nothing like a steroid.

1.1 · FUNDAMENTALS

WHAT A PEPTIDE ACTUALLY IS

A peptide is a short chain of amino acids linked by peptide bonds. That's the whole definition, and the length is what separates the categories.

Amino acids are the building blocks of all proteins. When you link a handful of them together you get a peptide; link hundreds and fold them into a 3-D shape and you get a protein. The dividing line is loose, but the convention runs like this:

CLASS	LENGTH	EXAMPLES
OLIGOPEPTIDE	2–20 amino acids	BPC-157 (15 aa), KPV (3 aa), GHK-Cu (3 aa)
POLYPEPTIDE	20–50 amino acids	Tesamorelin (44 aa), CJC-1295

CLASS	LENGTH	EXAMPLES
SMALL PROTEIN	50+ amino acids	Insulin (51 aa), IGF-1 (70 aa)

The key insight: your body is already running on peptides every second. Insulin is a peptide. Growth hormone is a peptide. Glucagon, oxytocin, ghrelin, the GLP-1 your gut releases after a meal, all peptides. They are the body's native signaling language. The peptides in this guide are either exact copies of those signals, slightly modified analogs designed to last longer, or fragments engineered to trigger one specific effect.

THE SIMPLE VERSION

A steroid is a key that forces a lock. A peptide is a text message. It tells a receptor to do something the cell already knows how to do. That single distinction explains almost everything about how differently they behave, dose, and cycle.

1.2 · MECHANISM

WHAT PEPTIDES DO

Peptides work almost entirely through receptor signaling. A peptide floats to a target cell, binds a receptor shaped to fit it, and that binding flips a switch inside the cell, triggering a cascade of downstream events (gene transcription, enzyme activation, hormone release, blood-vessel growth, immune modulation).

Because the effect depends on a receptor recognizing a specific shape, peptides are highly selective. A growth-hormone-releasing peptide talks to the pituitary. A GLP-1 analog talks to receptors in the pancreas, gut, and brain. BPC-157 appears to upregulate growth-factor and nitric-oxide pathways at sites of injury. Each one has a narrow, defined job.

THE FOUR JOBS PEPTIDES ARE USED FOR

- **Release a hormone**, secretagogues like ipamorelin or CJC-1295 prompt your own pituitary to pulse more GH.
- **Mimic a hormone**, GLP-1 analogs imitate a natural gut hormone for a much longer duration than the real thing.
- **Repair & remodel tissue**, BPC-157, TB-500, GHK-Cu accelerate the body's own healing machinery (angiogenesis, collagen, growth factors).
- **Modulate a system**, KPV calms inflammation, MOTS-c tunes mitochondrial metabolism, Epithalon influences the aging/telomere axis.

WHY THIS MATTERS FOR RESULTS

Because peptides ask the body to do something rather than override it, they generally cannot force a result the body isn't capable of. A GH secretagogue does nothing if your pituitary is exhausted. A healing peptide won't repair tissue with no blood supply. They amplify and accelerate native processes, which is exactly why they tend to be gentler, but also why "more" frequently does nothing.

1.3 · THE EDGE

WHAT MAKES THEM SPECIAL

Three properties make peptides uniquely interesting compared with nearly every other class of performance or wellness compound.

1 · SPECIFICITY

A peptide hits a defined receptor and does a defined thing. That precision means you can target visceral fat (tesamorelin), tendon healing (BPC-157), or appetite (GLP-1s) without the broad, body-wide hormonal disruption a steroid causes.

2 · THEY RUN ON THE BODY'S OWN MACHINERY

Most peptides work with existing feedback loops rather than bulldozing them. A secretagogue still respects the pituitary's pulsatile rhythm and somatostatin brake, which is why GH peptides don't shut down your axis the way exogenous HGH can.

3 · TUNABLE HALF-LIVES

Medicinal chemists can engineer a peptide to last minutes or weeks. Native GLP-1 lasts ~2 minutes; semaglutide was modified to last about a week. That tunability is why the same biological signal can become a daily shot, a weekly shot, or a continuous infusion.

THE HONEST CAVEAT

"Special" doesn't mean "proven." The approved GLP-1s and GH analogs sit on mountains of human data. A large fraction of the "research peptide" market (BPC-157, TB-500, many longevity peptides) rests on animal studies, anecdote, and mechanism, promising, but not the same evidentiary tier. A serious operator keeps those two buckets separate in their head at all times.

1.4 · THE COMPARISON EVERYONE ASKS ABOUT

PEPTIDES VS. STEROIDS

This is the single most misunderstood point in the space, so it deserves a real breakdown. Peptides are not steroids, and the difference is structural, not just a matter of degree.

PROPERTY	ANABOLIC STEROIDS	PEPTIDES
CHEMISTRY	Lipid / cholesterol-derived hormones (testosterone & derivatives)	Amino-acid chains
MECHANISM	Enter the cell, bind the androgen receptor, directly drive protein synthesis	Bind surface receptors, signal the cell to act
FORCE VS. SIGNAL	Force a result, anabolism happens whether the body "wants" it or not	Signal a request, amplify native processes
HPTA SUPPRESSION	Strong; shuts down natural testosterone, needs PCT	Most don't suppress the gonadal axis at all
RAW MUSCLE BUILDING	Powerful, direct, dose-dependent	Indirect and modest (except IGF-1 / insulin)
ORGAN & LIPID STRAIN	Significant, liver, lipids, BP, heart	Generally lower, category-dependent
PRIMARY USE CASE	Maximal size & strength	Recovery, fat loss, healing, GH support, longevity

A practical way to hold it: steroids build the house; peptides are the contractors, supply chain, and cleanup crew. Bodybuilders who use both treat steroids as the anabolic engine and peptides as recovery, healing, GH optimization, fat loss, and damage-control around that engine. Peptides alone will not produce a steroid-like physique, and anyone selling them that way is selling fiction.

IMPORTANT NUANCE

"Gentler than steroids" is not "harmless." GLP-1s have real GI and gallbladder risks. Melanotan II affects moles and melanoma risk. GH secretagogues raise IGF-1 and can affect glucose. Insulin can kill in minutes if mis-dosed. Lower ceiling of harm ≠ no harm.

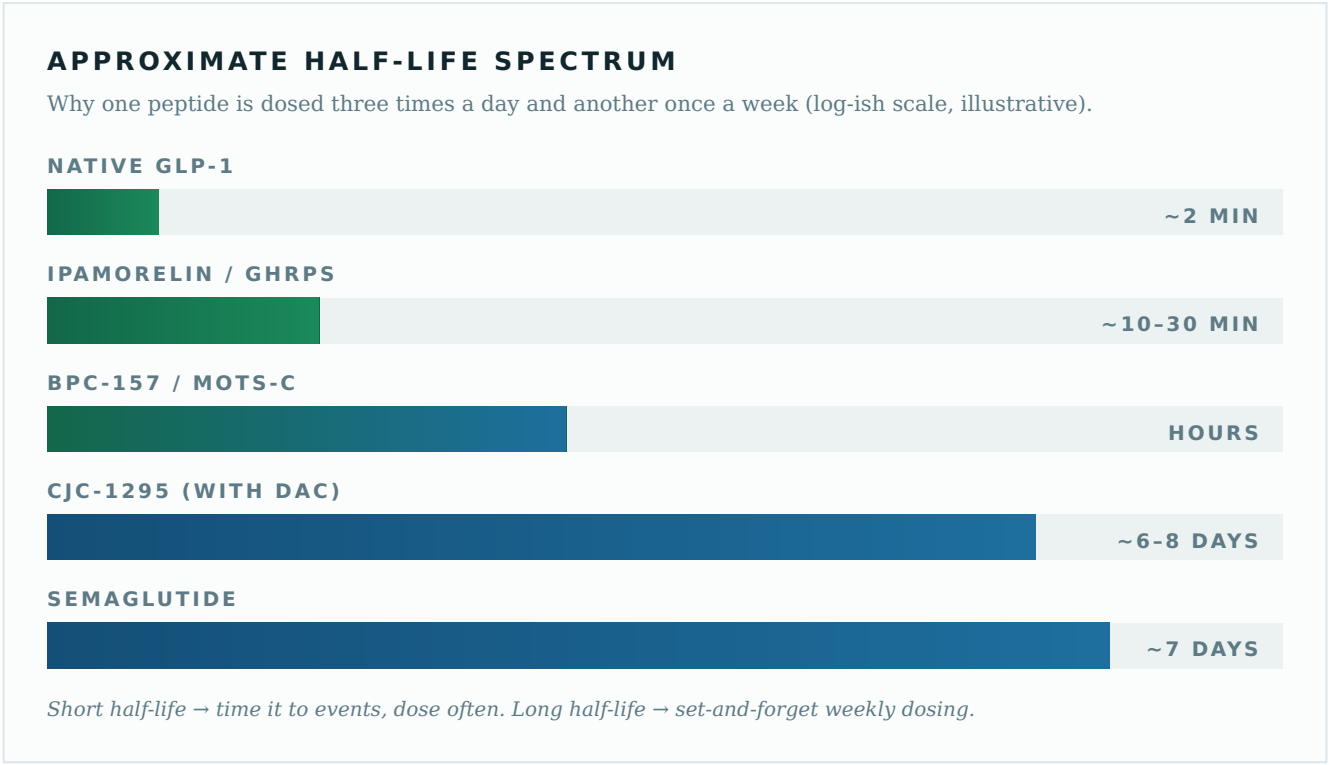
HALF-LIFE, RECONSTITUTION & DELIVERY

WHY MOST PEPTIDES ARE INJECTED

Peptides are made of amino acids, exactly what your digestive system is built to break down. Swallow most of them and stomach acid and enzymes destroy them before they reach the bloodstream. That's why the majority are given subcutaneously (into the fat layer) with a small insulin syringe. A handful are orally bioavailable or specially formulated (oral semaglutide, MK-677, 5-Amino-1MQ, SLU-PP-332 in research forms), and some are delivered nasally (Semax, Selank, PT-141).

HALF-LIFE DRIVES FREQUENCY

Half-life, how long it takes the body to clear half a dose, is the single biggest driver of dosing frequency:



- **Very short (minutes):** ipamorelin, native GLP-1, dosed timed to events, often multiple times daily.
- **Short (hours):** BPC-157, TB-500, MOTS-c, daily or several times weekly.
- **Long (days–week):** semaglutide, CJC-1295 with DAC, tesamorelin, weekly or daily depending on design.

RECONSTITUTION IN PLAIN TERMS

Most research peptides ship as a lyophilized (freeze-dried) powder that must be reconstituted with bacteriostatic water before use. The math that trips people up:

THE ONLY FORMULA YOU NEED

Concentration = total mcg in vial ÷ mL of water added. Then units to draw = desired dose ÷ concentration per unit (on an insulin syringe, 100 units = 1 mL). Example: a 5 mg (5000 mcg) vial + 2 mL water = 2500 mcg/mL = 25 mcg per unit. A 250 mcg dose = 10 units.

Reconstituted peptides are fragile: keep them refrigerated, use bacteriostatic (not sterile) water for multi-use vials, and respect that potency degrades over weeks. This is also where quality collapses, underdosed, mislabeled, or contaminated product is rampant in the gray market, which is the entire reason third-party testing exists.

PART TWO

GLP-1 & THE INCRETIN REVOLUTION

The most important class in modern metabolic medicine. Semaglutide, tirzepatide, and retatrutide rewrote what's possible, here's the actual biology behind the mains.

2.1 · THE FOUNDATION

THE INCRETIN SYSTEM

To understand why these drugs are so effective, you have to understand the gut hormones they're built from. The "incretin effect" is one of the most elegant feedback loops in human physiology.

When you eat, your intestine releases hormones called incretins. The two that matter are **GLP-1** (glucagon-like peptide-1) and **GIP** (glucose-dependent insulinotropic polypeptide).

Their job is to prepare the body for incoming nutrients. Native GLP-1 does several things at once:

- **Stimulates insulin**, but only when blood glucose is elevated (glucose-dependent), which is why GLP-1 drugs rarely cause hypoglycemia on their own.
- **Suppresses glucagon**, the hormone that raises blood sugar.
- **Slows gastric emptying**, food leaves the stomach more slowly, so you feel full longer.
- **Acts on the brain**, GLP-1 receptors in the hypothalamus and reward centers reduce appetite and the "food noise" of constant cravings.

The catch: native GLP-1 is destroyed by the enzyme DPP-4 in about two minutes. The entire drug class exists to solve that one problem, modify the molecule so it resists DPP-4 and lasts long enough to work as a once-weekly (or once-daily) medication.

THE THREE RECEPTORS IN PLAY

The newest drugs are defined by how many incretin receptors they hit. **GLP-1** (appetite + insulin), **GIP** (insulin sensitivity + fat metabolism), and **glucagon** (energy expenditure + fat oxidation). Single, dual, and triple agonists are the story of the entire category's evolution.

2.2 · THE THREE GENERATIONS

SEMAGLUTIDE → TIRZEPATIDE → RETATRUTIDE

Each generation added a receptor and, with it, more weight-loss power. This is the clearest "evolution" in the peptide world.

SEMAGLUTIDE

GLP-1 · GEN 1

Brand names: Ozempic / Wegovy / Rybelsus (oral). The drug that started the cultural wave. A single GLP-1 receptor agonist engineered with a fatty-acid chain that binds albumin, extending half-life to ~7 days.

MECHANISM	Pure GLP-1 agonist, appetite suppression, glucose-dependent insulin, slowed gastric emptying, reduced food noise.
TYPICAL WEIGHT LOSS	~15% of body weight at top dose in trials (STEP program).
FREQUENCY	Once weekly (injectable); daily (oral form).
APPROVED FOR	Type 2 diabetes & chronic weight management.

TIRZEPATIDE

GLP-1 + GIP · GEN 2

Brand names: Mounjaro / Zepbound. The first dual agonist. It hits both GLP-1 and GIP. The GIP component appears to improve insulin sensitivity and fat handling while also blunting some of the nausea GLP-1 causes, letting people tolerate higher effective doses.

MECHANISM	Dual GLP-1 + GIP agonist, stronger appetite and glucose effects plus improved insulin sensitivity.
TYPICAL WEIGHT LOSS	~20–22% at top dose (SURMOUNT trials), meaningfully more than semaglutide head-to-head.
FREQUENCY	Once weekly.
APPROVED FOR	Type 2 diabetes & chronic weight management.

RETATRUTIDE

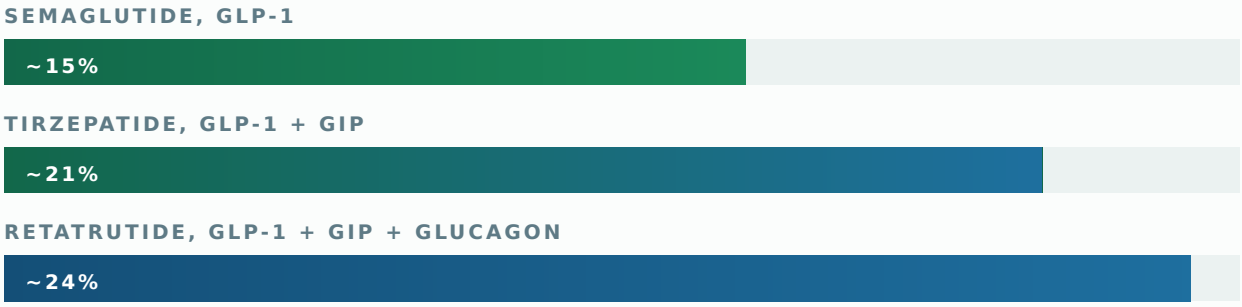
GLP-1 + GIP + GLUCAGON · GEN 3

The triple agonist, sometimes nicknamed "triple G." It adds glucagon-receptor agonism to the mix. That's counterintuitive (glucagon raises blood sugar), but at the receptor level glucagon agonism increases energy expenditure and fat oxidation, while the GLP-1/GIP components keep glucose controlled. The result in trials is the most dramatic fat loss yet seen from an injectable.

MECHANISM	Triple GLP-1 + GIP + glucagon agonist, appetite suppression plus a genuine metabolic-rate / fat-burning component.
TYPICAL WEIGHT LOSS	~24% at 48 weeks in Phase 2, with the curve not yet plateauing, the standout figure in the field.
FREQUENCY	Once weekly (trial design).
STATUS	Investigational, in late-stage trials, not FDA-approved. Anything sold now is gray-market research material.

AVERAGE WEIGHT LOSS BY GENERATION

Peak-dose results from pivotal trials (% of body weight). Each added receptor pushed the ceiling higher.



Figures are approximate peak-dose trial averages (STEP, SURMOUNT, and Phase 2 retatrutide). Retatrutide's curve had not fully plateaued at measurement, the standout signal in the field.

WHY RETATRUTIDE IS THE ONE TO WATCH

The glucagon arm means it's not only an appetite drug. It has a thermogenic, fat-oxidizing component the others lack. That's also why it demands respect: more pathways engaged means more that can go sideways (heart rate, glucose dynamics, GI). It is the most powerful and the least battle-tested of the three.

2.3 · PRACTICAL USE

TITRATION, DOSING & SIDE EFFECTS

The single most important principle with every GLP-1 drug: *start low, go slow.* Side effects are almost entirely a function of dose escalation speed, not the drug itself. The body adapts to slowed gastric emptying over weeks; rush it and you get the nausea, vomiting, and misery that make people quit.

STANDARD TITRATION LOGIC (APPROVED DRUGS)

Clinical protocols start at a sub-therapeutic dose purely for tolerance, then step up roughly every 4 weeks. The escalation exists to let the gut adjust, the early doses aren't meant to maximize weight loss, they're meant to keep you on the drug.

DRUG	TYPICAL START	ESCALATION	COMMON TOP RANGE
SEMAGLUTIDE	0.25 mg/wk	↑ ~monthly	2.0–2.4 mg/wk
TIRZEPATIDE	2.5 mg/wk	↑ ~monthly	10–15 mg/wk
RETATRUTIDE*	2 mg/wk (trial)	↑ over months	8–12 mg/wk (trial)

**Trial-derived figures; not an approved protocol.*

THE SIDE-EFFECT MAP

- **Common:** nausea, constipation or diarrhea, reduced appetite (the point), early fullness, fatigue during titration.
- **Manageable:** "sulfur burps," reflux, transient heart-rate bump. Smaller meals, lower fat/fiber timing, and hydration help.
- **Serious / see a doctor:** persistent severe abdominal pain (pancreatitis risk), gallbladder issues (rapid weight loss raises gallstone risk), and the boxed warning around thyroid C-cell tumors seen in rodents (contraindicated with personal/family history of medullary thyroid carcinoma or MEN2).

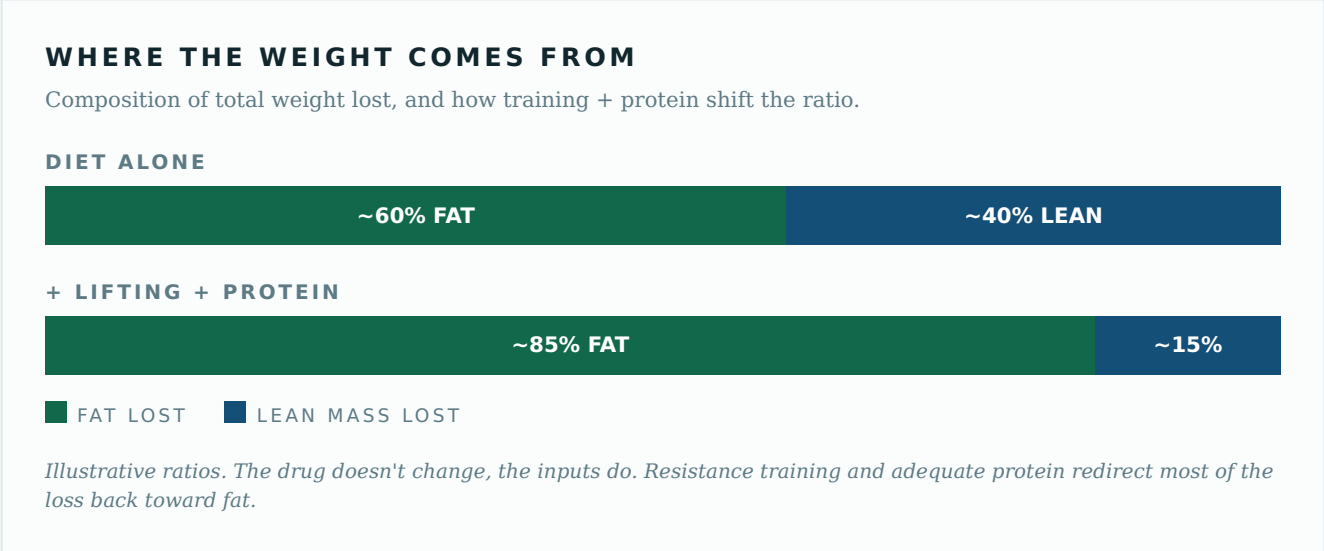
> **TWO PRACTICAL REALITIES**
>
> Hydration & electrolytes get neglected because appetite, and thirst, drop. Dehydration drives a lot of the fatigue and headaches. And protein intake becomes a deliberate effort, because total food drops so far that people unknowingly under-eat protein, which feeds directly into the next section.

2.4 · THE BODYBUILDER'S CONCERN

THE MUSCLE-LOSS PROBLEM

Any rapid weight loss strips some lean mass, GLP-1s are no exception, and for a physique athlete this is *the* issue that matters.

In weight-loss trials, a meaningful share of total weight lost on GLP-1s is lean mass, often cited in the range of 25–40% of the loss, similar to aggressive dieting in general. The drug doesn't specifically attack muscle; it just makes you eat dramatically less, and severe deficits without a protein-and-training stimulus cost muscle. For a sedentary patient that's an acceptable trade. For someone whose entire goal is muscle, it's the central problem.



HOW IT'S MITIGATED IN PRACTICE

- **Protein first, non-negotiable.** The standard target lands around 1 g per pound of goal bodyweight. When total food is suppressed, protein has to be prioritized over everything else on the plate.
- **Resistance training as the signal.** Lifting is what tells the body to keep muscle in a deficit. Without it, lean loss accelerates.
- **Moderate the deficit / dose.** Using the lowest effective dose that still drives appetite control, rather than maxing out, keeps the deficit from becoming so steep that muscle goes with it.
- **Pairing with GH support or anabolics.** This is why enhanced lifters often run GLP-1s alongside GH secretagogues, GH, or a TRT/anabolic base, the muscle-sparing signal offsets the deficit's lean cost. (Combination = combination risk; covered in Part V.)

WORTH KEEPING IN MIND

GLP-1s are an appetite tool, not a fat-loss magic bullet. They make a deficit effortless to maintain. Everything that protects muscle in a normal diet, high protein, hard training, a controlled deficit, protects muscle here too. The drug just removes the hunger that usually breaks adherence.

2.5 · ON THE HORIZON

THE NEXT WAVE

The incretin field is moving fast. Beyond the big three, a cluster of next-generation molecules and combinations is already in trials, worth knowing because the gray market sells them long before approval.

SURVODUTIDE

GLP-1 + GLUCAGON

A dual GLP-1 + glucagon agonist (skipping GIP). The glucagon arm drives energy expenditure and shows particular promise for liver fat / MASH (fatty liver disease) alongside weight loss.

MAZDUTIDE

GLP-1 + GLUCAGON

Another GLP-1/glucagon dual agonist (based on oxyntomodulin), advancing notably in Asian-market trials with strong weight and metabolic results.

CAGRISEMA

COMBINATION

Cagrilintide (amylin analog) + semaglutide in one pen. Pairs two satiety mechanisms, incretin + amylin, and has posted some of the highest trial weight-loss figures yet, rivaling tirzepatide.

ORAL GLP-1S

DELIVERY SHIFT

Oral semaglutide already exists; next-gen oral small-molecule GLP-1 agonists (e.g., the orforglipron class) aim to deliver GLP-1 benefits in a daily pill without injection, potentially the biggest accessibility shift in the category.

THE TRAJECTORY

The pattern is clear: more receptors, more combinations, easier delivery. Triple agonists, amylin pairings, and oral formats are converging on the same goal, maximal fat loss with maximal adherence. For a research reference, the takeaway is that "the GLP-1s" is no longer three drugs; it's a rapidly expanding class.

PART THREE

THE CATEGORIES

Every major peptide, sorted by what it's actually for, with mechanism, dosing ranges, timing, cycling, and how each is commonly stacked.

HOW TO READ THESE ENTRIES

THE CARD FORMAT

Every compound below uses the same structure so you can scan fast. Dosing ranges are **reported from literature and common practice for educational reference**. They are not instructions, and they assume a context most people shouldn't replicate without supervision.

MECHANISM	what it does at the receptor
DOSE RANGE	figures cited in research / practice
FREQUENCY	how often
TIMING	AM / PM / pre-workout logic
CYCLE	on-vs-off convention
STACKS WITH	common pairings

3.1 · CATEGORY

HEALING & RECOVERY

The "repair crew." These peptides accelerate the body's native healing, angiogenesis, collagen synthesis, growth-factor signaling, and inflammation control. The most popular real-world category, and the one with the most anecdotal love and the thinnest human trial data.

BPC-157

HEALING · GUT · TENDON

A 15-amino-acid fragment derived from a protein found in gastric juice. The most popular healing peptide, prized for tendon, ligament, muscle, and gut repair. Strongly upregulates the VEGF/nitric-oxide pathway, promoting new blood-vessel growth at injury sites.

MECHANISM	Angiogenesis, growth-factor upregulation, gut-lining repair, anti-inflammatory.
DOSE RANGE	250–500 mcg, 1–2× daily (some run up to ~10 mcg/kg).
TIMING	Flexible; often near the injury site or split AM/PM. Oral forms favored for gut issues.
CYCLE	Run through the healing window, commonly 4–8 weeks, then off.
STACKS WITH	TB-500 (the "Wolverine" duo), KPV (gut), GHK-Cu.
EVIDENCE NOTE	Compelling animal data; human data is largely anecdotal. Not FDA-approved.

TB-500 / THYMOSIN BETA-4

HEALING · INFLAMMATION

A synthetic fragment of thymosin beta-4. Works on a different axis than BPC-157. It regulates actin, the protein behind cell migration, helping cells travel to and rebuild damaged tissue. Favored for larger muscle groups, flexibility, and systemic recovery.

MECHANISM	Actin regulation, cell migration, angiogenesis, reduced inflammation.
DOSE RANGE	Loading ~2–2.5 mg 2×/week, then a lower maintenance dose.
TIMING	Time-of-day insensitive; usually fixed days each week.
CYCLE	4–6 week loading phase, then maintenance or off.
STACKS WITH	BPC-157 (synergistic healing), GHK-Cu.

KPV

GUT · ANTI-INFLAMMATORY

A tiny 3-amino-acid C-terminal fragment of alpha-MSH. A focused anti-inflammatory, especially for the gut and skin, used for IBS, colitis-type inflammation, psoriasis/eczema, and as a calming agent in gut-repair stacks.

MECHANISM	Downregulates inflammatory signaling (NF-κB), mast-cell calming, immune balance.
DOSE RANGE	~200–500 mcg daily (oral capsule forms common for gut targeting).
TIMING	Daily; oral with the gut as target.
CYCLE	Run during a flare or repair phase (weeks), then taper.
STACKS WITH	BPC-157 (the gut-repair pairing), TB-500 ("KLOW" family).

GHK-CU

SKIN · HAIR · TISSUE

A copper-binding tripeptide naturally present in plasma that declines sharply with age. A genuine crossover compound, sits in both healing and looksmaxing. Drives collagen and elastin, wound healing, and hair-follicle support. Often used topically and injectable.

MECHANISM	Collagen/elastin synthesis, antioxidant, wound remodeling, follicle stimulation.
DOSE RANGE	Injectable ~1–2 mg/day; widely used topically in serums.
TIMING	Flexible; topical often AM/PM skincare.
CYCLE	Can run continuously at low dose or in 4–8 week blocks.
STACKS WITH	BPC-157 + TB-500 (the "GLOW" stack), Epithalon, Melanotan I.

THE SIGNATURE STACKS OF THIS CATEGORY

Wolverine = BPC-157 + TB-500 (injury & recovery). **GLOW** = BPC-157 + TB-500 + GHK-Cu (skin, joints, anti-inflammatory). **KLOW** = GLOW + KPV (adds gut + full-body anti-inflammatory). These show up constantly because the mechanisms genuinely complement each other.

3.2 · CATEGORY

GROWTH HORMONE SECRETAGOGUES

Instead of injecting GH directly, these prompt your own pituitary to release more of it. The advantage: they preserve the natural pulsatile rhythm and don't shut down your axis the way synthetic HGH can. They split into two families that are usually combined.

THE TWO FAMILIES, AND WHY THEY'RE PAIRED

- **GHRH analogs** (Sermorelin, CJC-1295, Tesamorelin), mimic growth-hormone-releasing hormone; they tell the pituitary "release GH."
- **GHRPs / ghrelin mimetics** (Ipamorelin, GHRP-2/6, Hexarelin, MK-677), work through the ghrelin receptor on a separate pathway and also suppress somatostatin (the GH brake).

Pairing one from each family (classic: CJC-1295 + Ipamorelin) produces a synergistic GH pulse larger than either alone, which is why that combo is the backbone of the whole category.

IPAMORELIN

GHRP · CLEAN PULSE

The "clean" GHRP. Stimulates a strong, selective GH pulse with minimal effect on cortisol, prolactin, or hunger, the reason it's the default GHRP for most people.

MECHANISM	Ghrelin-receptor agonist → GH pulse; suppresses somatostatin.
DOSE RANGE	~100–300 mcg per dose, 1–3× daily.
TIMING	Short half-life → dose on an empty stomach; best before bed (rides the natural nocturnal GH pulse) and/or post-workout. Avoid food/carbs ~30–60 min around it.
CYCLE	Often run continuously or in 12–16 week blocks; some cycle off periodically.
STACKS WITH	CJC-1295 (no DAC), the signature pairing.

CJC-1295 (NO DAC) / MOD GRF 1-29

GHRH · SHORT-ACTING

A short-acting GHRH analog. "No DAC" means it clears in hours, producing a clean pulse that mirrors natural GH release rather than a flat elevation. Pairs with a GHRP to amplify each pulse.

MECHANISM	GHRH-receptor agonist → tells pituitary to release GH.
DOSE RANGE	~100 mcg per dose, matched to GHRP timing, 1–3× daily.
TIMING	Empty stomach; before bed and/or post-workout, co-injected with ipamorelin.
CYCLE	Continuous or 12–16 week blocks.
STACKS WITH	Ipamorelin (the backbone combo).

CJC-1295 (WITH DAC)

GHRH · LONG-ACTING

The DAC (drug-affinity complex) binds albumin and extends half-life to ~6–8 days, creating a sustained "GH bleed" rather than discrete pulses. Convenient (weekly-ish dosing) but trades away the natural pulsatility some consider important.

MECHANISM	Long-acting GHRH analog → elevated baseline GH/IGF-1.
DOSE RANGE	~1–2 mg per week (often split).
TIMING	Timing-insensitive due to long half-life.
CYCLE	8–16 week blocks.
STACKS WITH	Ipamorelin or GHRP-2.

TESAMORELIN

GHRH · VISCERAL FAT

An FDA-approved GHRH analog (for HIV-associated lipodystrophy) with the strongest data of the secretagogues for one specific job: **reducing visceral (organ) fat**. The go-to when stubborn belly/visceral fat is the target.

MECHANISM	GHRH analog → GH/IGF-1 rise with pronounced visceral-fat reduction.
DOSE RANGE	~1–2 mg daily (2 mg is the clinical dose).
TIMING	Typically before bed, empty stomach.
CYCLE	Run in blocks (e.g., 12+ weeks) toward a body-comp goal.
STACKS WITH	Ipamorelin; sits inside fat-loss + GH protocols ("Level Up X" style stacks add it to CJC/ipa).

SERMORELIN

GHRH · GENTLE / ANTI-AGING

The original GHRH analog, milder and shorter-acting than CJC-1295, popular in anti-aging/ longevity clinics for gentle GH support, sleep, and recovery.

MECHANISM	GHRH agonist → modest GH pulse.
DOSE RANGE	~200–500 mcg before bed.
TIMING	Nightly, empty stomach, to support the nocturnal pulse.
CYCLE	Often run long-term at low dose.
STACKS WITH	GHRP / ipamorelin; GHK-Cu for skin/longevity.

MK-677 / IBUTAMOREN

ORAL · GHRELIN MIMETIC

An orally active ghrelin mimetic, no injection. Produces a sustained rise in GH and IGF-1. Loved for sleep, recovery, and fullness; the catch is it sharply increases appetite, can cause water retention and lethargy, and may raise blood glucose.

MECHANISM	Oral ghrelin-receptor agonist → sustained GH/IGF-1 elevation.
DOSE RANGE	~10–25 mg daily.
TIMING	Often at night (sleep/GH), but daytime if night-time grogginess is an issue. Consistent daily timing matters more than the hour.
CYCLE	8–16 weeks; some run longer with glucose monitoring.
WATCH	Appetite, water retention, fasting glucose / insulin sensitivity.

GHRP-2 · GHRP-6 · HEXARELIN

GHRP FAMILY · OLDER GEN

The older generation of growth-hormone-releasing peptides, mostly superseded by ipamorelin's cleaner profile but still used. They produce strong GH pulses with more "baggage": GHRP-6 causes intense hunger (sometimes used deliberately to drive appetite), GHRP-2 is more potent but raises prolactin/cortisol somewhat, and Hexarelin is the most potent but desensitizes fastest.

MECHANISM	Ghrelin-receptor agonists → GH pulse (same family as ipamorelin, less selective).
DOSE RANGE	~100–300 mcg per dose, 1–3× daily.
TIMING	Empty stomach; same logic as ipamorelin.
CYCLE	Hexarelin especially needs breaks (rapid desensitization).
PICK WHEN	GHRP-6 if appetite stimulation is wanted; otherwise ipamorelin usually wins.

THE GH-SECRETAGOGUE CAVEAT

Raising GH and IGF-1 is not free. It can worsen insulin sensitivity, cause water retention and carpal-tunnel-like symptoms, and, at the IGF-1 level, is theoretically relevant to anyone with cancer risk. The "natural and safe" framing oversells it. This category earns regular bloodwork (IGF-1, fasting glucose, HbA1c).

3.3 · CATEGORY

FAT LOSS

Beyond the GLP-1s (covered in Part II), this group works through fat oxidation, metabolic-rate, and mitochondrial pathways rather than appetite. They tend to be subtler tools, useful as accelerators inside a real diet, not replacements for one.

AOD-9604 / HGH FRAG 176-191

LIPOLYSIS · NO IGF RISE

A fragment of the GH molecule (the 176-191 tail) isolated to keep the fat-burning effect without the blood-sugar or IGF-1 effects of full GH. Promotes lipolysis and may inhibit new fat formation. Gentle, long-tolerated, often used for stubborn/localized fat.

MECHANISM	Stimulates lipolysis, inhibits lipogenesis, no IGF-1 rise.
DOSE RANGE	~250–500 mcg SC daily.
TIMING	Fasted AM or pre-workout, when free fatty acids can be burned.
CYCLE	Continuous or 8–12 week blocks.
STACKS WITH	CJC-1295/ipamorelin, L-carnitine, GLP-1s.

5-AMINO-1MQ

ORAL · NNMT INHIBITOR

An oral small molecule that inhibits the enzyme NNMT, which is overactive in fat tissue. Inhibiting it raises cellular NAD+ and pushes fat cells toward burning rather than storing, targeting white fat and metabolic slowdown.

MECHANISM	NNMT inhibition → ↑ NAD+, ↑ fat-cell energy expenditure, targets white adipose.
DOSE RANGE	~50–100 mg orally daily.
TIMING	Daily, often AM.
CYCLE	~4–8 week blocks.
STACKS WITH	NAD+ / NMN, MOTS-c, mitochondrial boosters.

MOTS-C

MITOCHONDRIAL · METABOLIC

A mitochondrial-derived peptide, a signal your own mitochondria produce. It improves metabolic flexibility and insulin sensitivity and acts almost like "exercise in a vial," activating AMPK (the same energy-sensing pathway training and fasting hit). Crosses into the longevity category too.

MECHANISM	AMPK activation → improved glucose handling, fat metabolism, mitochondrial function.
DOSE RANGE	~5–10 mg, 2–3× per week.
TIMING	Often pre-workout to amplify the exercise effect.
CYCLE	~4–8 week blocks, several times weekly.
STACKS WITH	5-Amino-1MQ, NAD+, SLU-PP-332.

SLU-PP-332

RESEARCH · "EXERCISE MIMETIC"

An early-stage research compound (ERR agonist) generating buzz as an "exercise mimetic", in rodents it boosts fat oxidation and endurance by upregulating the genes exercise activates. Very early; essentially no human data. Included for completeness, not endorsement.

MECHANISM	Estrogen-related-receptor (ERRα) agonist → ↑ fat oxidation, mitochondrial biogenesis, endurance genes.
DOSE RANGE	No established human dosing, animal research only.
TIMING	Unknown in humans.
CYCLE	N/A, investigational.
EVIDENCE NOTE	Rodent data only. Highest uncertainty in this section.

TESOFENSINE

ORAL · APPETITE / CNS

Not a peptide, a triple monoamine reuptake inhibitor (serotonin, dopamine, noradrenaline) that strongly suppresses appetite via the CNS. Potent in obesity trials. Stimulant-class effects mean cardiovascular and mood considerations matter.

MECHANISM	Serotonin/dopamine/noradrenaline reuptake inhibition → appetite suppression.
DOSE RANGE	~0.25–0.5 mg daily (trial range).
TIMING	Morning (stimulant profile can disrupt sleep).
CYCLE	Short blocks; watch BP and heart rate.
WATCH	Heart rate, blood pressure, mood, sleep.

CAGRILINTIDE

AMYLIN ANALOG · EMERGING

A long-acting amylin analog. Amylin works alongside insulin to signal fullness and slow gastric emptying. Most notable as the partner in CagriSema (cagrilintide + semaglutide), a combination posting standout weight-loss numbers in trials.

MECHANISM	Amylin-receptor agonist → satiety, slowed gastric emptying, complements GLP-1.
DOSE RANGE	Weekly (trial-defined).
TIMING	Once weekly.
STATUS	Investigational, pairs with semaglutide in late-stage trials.

ADIPOTIDE (FTPP)

RESEARCH · AGGRESSIVE · HIGH RISK

A targeted peptide that homes to the blood vessels feeding fat tissue and triggers their apoptosis (programmed death), essentially starving fat cells of blood supply. Dramatic fat loss in primate studies, but it came with **kidney toxicity**, which is the reason it never advanced and why it sits firmly in the high-caution column.

MECHANISM	Targets prohibitin on fat-vessel endothelium → adipose apoptosis.
DOSE RANGE	~1–2 mg SC daily (cited), short courses only.
CYCLE	2–4 weeks maximum in references, kidney strain limits duration.
WATCH	Renal toxicity, dehydration. Contraindicated with any kidney issue.
EVIDENCE NOTE	Discontinued in development. Aggressive and under-studied in humans.

GW-501516 (CARDARINE)

PPARΔ · ENDURANCE · CAUTION

Not a peptide, a PPARδ agonist (a "SARM-adjacent" research chemical) that shifts the body toward burning fat for fuel and dramatically boosts endurance. Beloved for cardio capacity and fat oxidation. The asterisk is large: it was **dropped from development after cancer findings** in long-term high-dose rodent studies, which keeps it controversial.

MECHANISM	PPARδ activation → ↑ fat oxidation, endurance gene expression.
DOSE RANGE	~10–20 mg oral daily (cited).
TIMING	Often pre-cardio; daily.
CYCLE	Short blocks; long-term use is where the concern lies.
WATCH	The rodent carcinogenicity signal at high chronic doses is the main caveat.

HOW THIS CATEGORY IS ACTUALLY USED

These rarely work as standalone "fat burners." In practice they're accelerators layered onto a deficit: AOD or carnitine for fasted-cardio fat mobilization, MOTS-c / 5-Amino-1MQ for the metabolic/mitochondrial angle, GLP-1s for appetite, GH peptides for the GH-driven lipolysis. The diet and training still do the heavy lifting.

3.4 · CATEGORY

MUSCLE BUILDING & BULKING

This is the category that most resembles steroids in directness, and the one carrying the most serious risk. These compounds drive growth through IGF-1, mechano-growth signaling, nutrient partitioning, and myostatin pathways. Several (IGF-1, MGF, insulin) are genuinely advanced and genuinely powerful.

IGF-1 LR3

ANABOLIC · ADVANCED

A modified, long-acting version of insulin-like growth factor 1, the hormone GH actually works through. The "LR3" modification extends its half-life dramatically. Directly drives muscle-cell growth (hyperplasia and hypertrophy) and nutrient uptake. Powerful, and not benign, IGF-1 promotes growth indiscriminately, which is the same reason it carries theoretical cancer-risk concerns.

MECHANISM	IGF-1 receptor agonist → direct muscle growth, satellite-cell activation, nutrient uptake.
DOSE RANGE	~20–50 mcg daily (cited ranges; highly individual).
TIMING	Often post-workout or near meals.
CYCLE	Short blocks (commonly ~4 weeks) due to receptor and risk concerns.
WATCH	Hypoglycemia, organ growth concerns, cancer-risk theory. Advanced-only.

PEG-MGF

MECHANO GROWTH · REPAIR

MGF (Mechano Growth Factor) is a splice variant of IGF-1 the body releases locally in muscle in response to mechanical overload, it's the signal that activates satellite cells to repair and grow trained tissue. The "PEG" (pegylated) version adds a coating that extends its short native half-life so a dose actually lasts. Think of it as the "recruit new muscle cells" signal that works downstream of the training stimulus.

MECHANISM	Activates & proliferates satellite cells → new myonuclei, local repair/ growth after overload.
DOSE RANGE	~200–400 mcg per dose (cited); pegylation allows less frequent dosing than non-PEG MGF.
TIMING	Post-workout, into the trained muscle, to ride the mechanical-damage window.
CYCLE	Short blocks, training-driven.
STACKS WITH	IGF-1 LR3 (systemic vs. local growth), GH secretagogues.

IGF-1 DES

ANABOLIC · LOCALIZED

A truncated, very short-acting IGF-1 variant that's even more potent at the receptor but clears fast, used by advanced lifters for a localized, site-specific pump-and-grow effect when injected into the trained muscle, rather than the systemic action of LR3. Same IGF-1 risk family, same advanced-only caution.

MECHANISM	Highly potent IGF-1 analog, short half-life → strong local muscle signaling.
DOSE RANGE	~50–150 mcg (cited), site-injected post-workout.
CYCLE	Short blocks; advanced use.
WATCH	Same hypoglycemia / growth concerns as the IGF-1 family.

FOLLISTATIN / FOLLISTATIN-344

MYOSTATIN · EXPERIMENTAL

Targets myostatin, the body's brake on muscle growth. Inhibiting myostatin theoretically removes a ceiling on hypertrophy (the basis of the famously muscular "myostatin-null" animals). In practice the injectable peptide's real-world efficacy and durability in humans are unproven and debated, and quality control is a major problem.

MECHANISM	Myostatin inhibition → removes a limiter on muscle growth.
DOSE RANGE	No reliable human protocol; product quality is the bigger issue than dose.
CYCLE	Short experimental blocks.
EVIDENCE NOTE	Heavily hyped, thinly supported in humans. High skepticism warranted.

INSULIN

ANABOLIC · HIGH RISK

The most anabolic hormone in the body and, used for physique purposes, **the single most dangerous compound in this entire document**. It drives nutrient partitioning, shuttling glucose and amino acids into muscle for fullness and growth. It is also the one peptide where a dosing error can cause unconsciousness and death within an hour.

MECHANISM	Insulin-receptor agonist → glucose/amino-acid uptake, glycogen loading, nutrient partitioning.
MEDICAL USE	Essential, life-saving for diabetics.
PERFORMANCE USE	Advanced bodybuilding only, and disproportionately responsible for fatal accidents in the sport.

INSULIN: READ THIS, NOT A PROTOCOL

Exogenous insulin can drop blood glucose to fatal levels. Hypoglycemia escalates from sweating and confusion to seizure, coma, and death, and it can happen during sleep with no warning. There is no "safe beginner insulin cycle" for muscle building; experienced competitors have died from miscalculations. **This guide deliberately gives no performance dosing for insulin.** It belongs only under direct medical management. The educational takeaway is understanding why it's anabolic and why it's feared.

HONEST FRAMING FOR THE WHOLE CATEGORY

Note what's missing: there is no peptide that builds steroid-like muscle safely and easily. The compounds that come closest (IGF-1, MGF, insulin) are precisely the ones with the steepest risk curves. The muscle most people attribute to "peptides" actually comes from **better recovery** (healing peptides), **better training capacity** (GH support, sleep), and **better body composition** (fat loss), not direct anabolism. MGF and IGF are the real local growth signals here; everything else is support around them.

3.5 · CATEGORY

LOOKSMAXING: SKIN, HAIR & TAN

The aesthetics cluster: collagen and skin quality, hair, and tan. This is purely about how you look, built around copper-peptide, melanocortin (tanning), and pineal pathways. Anything sexual lives in its own category (§3.9).

GHK-CU

SKIN · HAIR · COLLAGEN · LEAD

The cornerstone looksmaxing peptide. A copper-binding tripeptide that's naturally abundant in young skin and plummets with age. It drives collagen and elastin synthesis, skin firmness and tone, wound/scar remodeling, and hair-follicle support, used both injectable and topical (serums). Also a genuine healing compound (see §3.1), but aesthetics are its claim to fame.

MECHANISM	Collagen/elastin synthesis, antioxidant, skin remodeling, follicle stimulation.
DOSE RANGE	Injectable ~1–2 mg/day; widely used topically in serums.
TIMING	Flexible; topical typically AM/PM skincare.
CYCLE	Low-dose continuous or 4–8 week blocks.
STACKS WITH	Epithalon, Melanotan, NAD+ (the skin/anti-aging cluster); BPC-157/ TB-500 for the "GLOW" blend.

MELANOTAN I (AFAMELANOTIDE)

TAN · MILDER

An alpha-MSH analog that stimulates melanin to give a gradual, even tan with less UV exposure. The "cleaner" tanning peptide, slower and milder than MT-II, without the strong libido/appetite side effects, but also less dramatic.

MECHANISM	Melanocortin-1 receptor agonist → melanin production.
DOSE RANGE	Low daily dosing during a build-up phase, then maintenance.
TIMING	Daily; tan deepens with some UV exposure.
CYCLE	Build-up phase, then occasional top-ups.
WATCH	Existing moles can darken, baseline skin check matters.

MELANOTAN II

TAN · LIBIDO · APPETITE

A broader melanocortin agonist, stronger tanning than MT-I, plus notable libido enhancement and appetite suppression (it hits multiple melanocortin receptors). More side effects: nausea, flushing, and the classic spontaneous erections from MC4 activity.

MECHANISM	Non-selective melanocortin agonist → tan + libido + appetite effects.
DOSE RANGE	~0.25–1 mg SC daily or every other day.
TIMING	Often evening (nausea is easier to sleep through); paired with measured UV.
CYCLE	~6–8 week build, then pause.
WATCH	**Melanoma risk & mole changes, the main safety concern.** Nausea, flushing, darkening of moles.

NAD+ (SKIN / ANTI-AGING ANGLE)

CELLULAR REPAIR · GLOW

Primarily a longevity compound (full card in §3.6), but it earns a place here for the aesthetic angle: NAD+ supports cellular repair and the sirtuin pathways tied to skin quality, energy, and that overall "well-rested glow." Often folded into anti-aging skin protocols alongside GHK-Cu and Epithalon.

MECHANISM	Restores NAD+ → cellular repair, sirtuin activation, skin/energy support.
USE HERE	The anti-aging / radiance layer of a looksmaxing stack.
STACKS WITH	GHK-Cu, Epithalon.

EPITHALON (EPITALON)

SKIN · LONGEVITY CROSSOVER

A pineal-gland peptide (Russian research) that influences telomerase and circadian/melatonin regulation. Used for skin elasticity, sleep, and as a flagship "longevity" peptide. Strong claims, mostly older Russian data, genuine longevity crossover.

MECHANISM	Telomerase activation, pineal/melatonin regulation, antioxidant.
DOSE RANGE	~5–10 mg/day in short courses.
TIMING	Often evening; run as a short "course."
CYCLE	Pulsed courses (e.g., 10–20 days) a few times per year.
STACKS WITH	GHK-Cu, NAD+ (the skin/longevity cluster).

THE LOOKSMAXING STACK LOGIC

Skin & collagen → GHK-Cu (topical + injectable) and Epithalon. Hair → GHK-Cu plus the GH axis. Anti-aging glow → add NAD+. Tan → Melanotan I (controlled) over II for safety. This is the pure-aesthetics group, libido and sexual performance are deliberately split out into Sexual Health (§3.9), since PT-141 and the like have nothing to do with how you look.

3.6 · CATEGORY

LONGEVITY & CELLULAR HEALTH

The anti-aging cluster, aimed at mitochondria, NAD+ metabolism, and telomeres. The most speculative category for "lifespan" claims, but several members have solid mechanistic and clinical footing for cellular function and recovery. (Immune compounds now live in §3.10.)

NAD+ (INJECTABLE)

CELLULAR ENERGY · REPAIR

NAD+ is a coenzyme central to energy production and DNA repair that declines with age. Injectable NAD+ (or precursors NMN/NR) aims to restore it, supporting mitochondrial function, cellular repair, and the sirtuin "longevity" enzymes. The infusion itself is infamous for a flushing/chest-pressure sensation if pushed too fast.

MECHANISM	Restores NAD+ → energy metabolism, DNA repair, sirtuin activation.
DOSE RANGE	Injectable protocols vary widely; oral precursors (NMN/NR) ~250–1000 mg/day.
TIMING	Slow administration is the rule, speed drives the side effects.
CYCLE	Periodic loading then maintenance.
STACKS WITH	MOTS-c, 5-Amino-1MQ, Epithalon.

SS-31 (ELAMIPRETIDE)

MITOCHONDRIAL · CLINICAL

A mitochondria-targeting peptide that binds cardiolipin in the inner mitochondrial membrane, stabilizing the energy-production machinery and cutting oxidative stress. One of the more clinically advanced longevity peptides (studied in mitochondrial disease and heart conditions).

MECHANISM	Cardiolipin stabilization → restored mitochondrial efficiency, reduced ROS.
DOSE RANGE	Clinical/trial-defined; varies by indication.
CYCLE	Block-based.
STACKS WITH	NAD+, MOTS-c (the mitochondrial trio).

HUMANIN

MITOCHONDRIAL · CYTOPROTECTIVE

A mitochondrial-derived peptide (the same family as MOTS-c) with cytoprotective and metabolic-protective signaling. Studied for neuroprotection, cardiovascular protection, and the biology of aging. It helps cells survive metabolic stress, which is why it sits squarely in the longevity axis.

MECHANISM	Cytoprotective mitochondrial signaling → cell survival, metabolic protection.
STATUS	Research-stage; part of the mitochondrial-longevity family with MOTS-c.
STACKS WITH	MOTS-c, NAD+, SS-31 (the mitochondrial cluster).

Also living here: **Epithalon** (telomerase/pineal, see Looksmaxing) and **MOTS-c** (mitochondrial/metabolic, see Fat Loss). These crossovers are why "longevity stacks" so often blend the same handful of mitochondrial and NAD-axis compounds.

> **REALITY CHECK ON "LONGEVITY"**
>
> Almost none of these have human lifespan data, that study would take decades. What they have is mechanistic plausibility and improvements in markers of cellular function. Treat "longevity" claims as "supports cellular health on paper," not "proven to extend your life."

3.7 · CATEGORY

COGNITIVE & NEURO

The nootropic peptides, focus, mood, neuroprotection, and stress resilience. Mostly Russian-developed, often delivered nasally, and frequently of interest for both performance and early cognitive-decline support.

SEMAX

FOCUS · NEUROPROTECTION

An ACTH-fragment peptide that raises BDNF (brain-derived neurotrophic factor) and modulates dopamine/serotonin. Used for focus, mental clarity, and neuroprotection. Delivered nasally.

MECHANISM	↑ BDNF, neurotransmitter modulation → focus, neuroprotection.
DOSE RANGE	Nasal, daily during use; low microgram-to-mg drops.
TIMING	AM/daytime for the stimulating effect.
CYCLE	Short courses or as-needed.

SELANK

ANXIETY · CALM FOCUS

Semax's calming counterpart, an anxiolytic peptide that takes the edge off anxiety without sedation. Often paired with Semax for "calm, clear focus." Also nasal.

MECHANISM	Modulates GABA/serotonin, ↑ BDNF → reduced anxiety, stable mood.
DOSE RANGE	Nasal, daily during use.
TIMING	Daytime or before stress; non-sedating.
STACKS WITH	Semax (focus + calm).

CEREBROLYSIN

NEURORESTORATIVE · CLINICAL

A mixture of neurotrophic peptide fragments used clinically (in several countries) for stroke, dementia, and TBI recovery. The most "medical" of the cognitive peptides, with real clinical trials behind it, often discussed for serious neuro support and early cognitive decline.

MECHANISM	Neurotrophic support → neuronal survival, repair, plasticity.
DOSE RANGE	Clinical IM/IV courses (e.g., daily for a set number of days).
CYCLE	Defined courses, repeated periodically.
NOTE	The cognitive peptide with the strongest clinical footing.

DIHEXA

EXPERIMENTAL · POTENT

An extremely potent synaptogenesis compound (angiotensin IV derivative) reported in animal work to dramatically boost new synapse formation. Very experimental, essentially **no human safety data**, included for completeness with maximum caution.

MECHANISM	HGF/c-Met pathway → synapse formation (animal data).
EVIDENCE NOTE	Preclinical only. Highest uncertainty in this category.

3.8 · CATEGORY

SUPPORT COMPOUNDS

Not main performance peptides, but constant supporting players, antioxidant, detox, and fat-transport tools that round out protocols, especially for enhanced athletes managing the strain of heavier compounds.

GLUTATHIONE

ANTIOXIDANT · DETOX · LIVER

The body's master antioxidant. Supplemented (IV, IM, or oral/liposomal) for oxidative-stress control, liver support, immune function, and skin brightening. Common in enhanced-athlete protocols to offset the oxidative load of other compounds.

MECHANISM	Master antioxidant → neutralizes free radicals, supports detox/liver.
DOSE RANGE	IM/IV protocols vary; liposomal oral for daily support.
TIMING	Flexible; often paired with NAD+ or vitamin infusions.
STACKS WITH	NAD+, vitamin C, the broader recovery stack.

L-CARNITINE (INJECTABLE)

FAT TRANSPORT · ENERGY

Not a peptide, an amino-acid derivative that shuttles fatty acids into the mitochondria to be burned. Injectable forms (SubQ or IM) bypass the poor absorption of oral carnitine. A staple alongside fasted cardio and fat-loss peptides.

MECHANISM	Transports fatty acids into mitochondria → fat oxidation, energy, recovery.
DOSE RANGE	Injectable ~200–500 mg, often pre-cardio.
TIMING	Pre-workout / pre-fasted cardio, when fat-burning is active.
STACKS WITH	AOD-9604, GH peptides, GLP-1 protocols.

3.9 · CATEGORY

SEXUAL HEALTH & LIBIDO

Purely sexual-performance and desire compounds, nothing to do with how you look. Two mechanisms dominate: *central desire* (the brain wants it) and *peripheral blood flow* (the body can perform). The best results often combine one of each.

PT-141 (BREMELANOTIDE)

DESIRE · FDA-APPROVED

The flagship libido peptide. An MT-II offshoot FDA-approved specifically for sexual dysfunction. It works on **desire in the brain** (central melanocortin pathway), not blood flow like Viagra/Cialis. Works in both men and women, which is rare. This is a sexual-performance compound. It has no aesthetic or looksmaxing role.

MECHANISM	Central melanocortin (MC4) agonist → sexual desire/arousal.
DOSE RANGE	~1–2 mg, used as-needed before activity.
TIMING	A few hours ahead of desired effect; not daily.
WATCH	Nausea, transient blood-pressure rise, flushing.
STACKS WITH	Tadalafil, desire (PT-141) + blood flow (PDE5i) is the classic pairing.

TADALAFIL (CIALIS)

PDE5 INHIBITOR · BLOOD FLOW

Not a peptide, a PDE5 inhibitor and the blood-flow half of the equation. Long-acting (the "weekend" effect, ~36 hr) versus shorter sildenafil. Beyond erectile function, low daily doses are used for blood-flow benefits, "pumps," and even BPH/prostate symptoms. The performance complement to PT-141's desire effect.

MECHANISM	PDE5 inhibition → ↑ nitric-oxide signaling → vasodilation / blood flow.
DOSE RANGE	~5 mg daily (low-dose) up to ~10–20 mg as-needed.
TIMING	Daily low-dose for steady effect, or ahead of activity.
WATCH	Headache, flushing, nasal congestion; **never combine with nitrates** (dangerous BP drop).

KISSPEPTIN

LIBIDO · FERTILITY · UPSTREAM

The master upstream regulator of the reproductive axis. It sits above GnRH and kicks off the whole hormonal cascade. Research interest centers on libido, arousal, and fertility, studied as a more natural way to stimulate sex-hormone production and desire from the top of the chain.

MECHANISM	Stimulates GnRH neurons → drives LH/FSH/sex-hormone cascade → libido & fertility.
STATUS	Largely research/clinical; promising for libido and fertility.
STACKS WITH	Hormonal base; complements PT-141's central effect from the axis side.

THE TWO-LEVER MODEL

Sexual function splits cleanly into **desire** (brain) and **performance/blood flow** (body). PT-141 and Kisspeptin work the desire/hormonal lever; Tadalafil works the blood-flow lever. Note: Melanotan II also raises libido as a side effect (it's a PT-141 cousin), but its primary purpose is tanning, so it's carded under Looksmaxing, the libido is a bonus, not the reason to run it.

3.10 · CATEGORY

HORMONAL AXIS & IMMUNE SUPPORT

The support layer for anyone running a hormonal base (TRT/anabolics) and the immune peptides that keep a heavy protocol from running you down.

GONADORELIN

HPTA · TESTICULAR SUPPORT

A synthetic GnRH that prompts the pituitary to release LH and FSH, used to keep the testes active and maintain fertility/testicular size on TRT or a suppressive cycle. A modern, peptide-based alternative to HCG for axis support.

MECHANISM	GnRH agonist → LH/FSH release → testicular stimulation.
DOSE RANGE	Low microgram doses, often 2–3× weekly (clinically guided).
USE CASE	Preserving fertility/testicular function alongside exogenous hormones.

THYMOSIN ALPHA-1

IMMUNE · RESILIENCE

An immune-modulating peptide (approved in some countries for hepatitis and as an immune adjuvant). Balances and strengthens immune function, useful for staying healthy under the stress of a heavy training/compound load, and relevant to aging via immunosenescence.

MECHANISM	T-cell modulation → balanced, strengthened immune response.
DOSE RANGE	~1.5 mg, 2× weekly is a common reference.
CYCLE	Run during immune-support phases.
STACKS WITH	KPV, LL-37, the healing peptides for overall resilience.

LL-37

ANTIMICROBIAL · IMMUNE

A natural antimicrobial peptide of the innate immune system. Researched for antibacterial, anti-biofilm, and wound-healing roles, of interest for chronic infection, gut, and skin contexts. Powerful but can be pro-inflammatory, so it's a more advanced tool.

MECHANISM	Disrupts microbial membranes, modulates immune & wound-healing response.
USE CASE	Chronic/biofilm infection, immune-support contexts.

WHERE THIS FITS

For enhanced athletes, Gonadorelin is the practically relevant one, axis/fertility insurance on a suppressive cycle (often alongside or instead of HCG). Thymosin Alpha-1 and LL-37 are the immune layer for staying healthy under load. All belong under clinical guidance given how directly they touch the hormonal and immune systems.

PART FOUR

PROTOCOLS BY GOAL

The categories reorganized around what people actually want, so you can see which compounds cluster toward each objective and why.

PART IV · GOAL FRAMEWORKS

MAPPING COMPOUNDS TO OUTCOMES

These are **conceptual frameworks for how the field thinks about each goal**, not prescriptions. Each shows the primary driver, the support players, and the logic. Real protocols are individualized and supervised.

4.1, LOOKSMAXING

Primary: GHK-Cu (collagen, skin, hair) · **Support:** Epithalon (elasticity), NAD+ (anti-aging glow), Melanotan I (controlled tan), the GH axis (Sermorelin/CJC + ipamorelin for skin quality & recovery). **Logic:** aesthetics are a collagen + skin-turnover + tan problem; the GH peptides improve overall skin/hair as a byproduct of better recovery and IGF-1. GHK-Cu is the one compound nearly every skin protocol is built around, topical and injectable. Sexual compounds are deliberately not here. That's §4.6.

4.2, YOUTH & LONGEVITY

Primary: NAD+ axis (NAD+/NMN) + mitochondrial peptides (MOTS-c, SS-31, Humanin) · **Support:** Epithalon (telomere/sleep), Thymosin Alpha-1 (immune resilience), GHK-Cu. **Logic:** aging shows up first in mitochondria, NAD+ decline, immune drift, and collagen loss, so the longevity cluster targets exactly those. Pulsed courses (Epithalon, NAD+ loading) rather than continuous use is the common pattern.

4.3, INJURY REPAIR

Primary: BPC-157 + TB-500 (the "Wolverine" duo) · **Support:** GHK-Cu (tissue remodeling), the GH axis (CJC/ipamorelin for connective-tissue recovery), KPV if gut/inflammation is involved. **Logic:** healing is an angiogenesis + cell-migration + growth-factor problem, and BPC-157 and TB-500 hit complementary parts of it. This is the most popular real-world use of peptides, period, run through the healing window, then off.

4.4, FAT LOSS

Primary: a GLP-1 (semaglutide/tirzepatide/retatrutide) for appetite, or Tesamorelin for visceral fat · **Support:** AOD-9604 + L-carnitine (fasted-cardio fat mobilization), MOTS-c / 5-Amino-1MQ (metabolic/mitochondrial), GH peptides (GH-driven lipolysis). **Logic:** the GLP-1 removes the hunger that breaks diets; everything else accelerates oxidation. Protein + lifting are mandatory to protect muscle (Part II, §2.4).

4.5, MUSCLE BUILDING & BULKING

Primary: the GH axis (CJC-1295 + ipamorelin, or tesamorelin/MK-677) for recovery and IGF-1 · **Advanced/high-risk anabolic signaling:** IGF-1 LR3 (systemic), PEG-MGF / IGF-1 DES (local muscle growth), insulin (see the serious cautions in §3.4) · **Support:** BPC-157/TB-500 to train harder injury-free, MK-677 for sleep & fullness. **Logic:** the GH axis plus IGF/MGF signaling drives the actual growth; outside those, peptides build muscle indirectly by maximizing recovery, sleep, and training capacity. There's no safe peptide shortcut to steroid-level mass.

4.6, SEXUAL HEALTH

Primary: PT-141 (desire) + Tadalafil (blood flow), the two-lever combo · **Support:** Kisspeptin (libido from the hormonal axis), a solid hormonal base / healthy testosterone. **Logic:** sexual function is two separate problems, wanting it (brain) and performing (blood flow), so the strongest approach addresses both levers. This is entirely separate from looksmaxing; these compounds do nothing for appearance.

THE PATTERN ACROSS ALL SIX

Every goal has **one primary driver** and a small set of supporting players chosen because their mechanisms complement the primary. Stacking is not "more is better", it's covering different mechanistic angles of the same goal without redundancy. That principle is the next section.

PART FIVE

TIMING, STACKING & CYCLING

The operating principles that apply across every category, when to dose, how long to run, how to combine, and what changes between men and women.

5.1 · TIMING

TIME OF DAY: AM VS. MIDDAY VS. NIGHT

Timing isn't arbitrary. It follows the biology of each compound. Three principles drive almost every decision.

PRINCIPLE 1, RIDE THE NATURAL RHYTHM

GH releases in its biggest pulse during early deep sleep. So GH secretagogues (ipamorelin, CJC-1295, sermorelin, tesamorelin) are classically dosed **before bed on an empty stomach** to amplify that nocturnal pulse. MK-677 is also often nighttime for the same reason (though it can cause grogginess, pushing some people to daytime).

PRINCIPLE 2, MATCH THE FUEL STATE

Fat-mobilizing compounds work best when fat is available to burn and insulin is low. AOD-9604, L-carnitine, and MOTS-c are commonly dosed **fasted in the morning or pre-workout**. Conversely, food/carbs blunt GH secretagogues, so those need an empty stomach window (~30–60 min before and after).

PRINCIPLE 3, MATCH THE DESIRED EFFECT

Stimulating cognitives (Semax) → **daytime**. Calming ones (Selank) → flexible. Stimulant fat-loss agents (tesofensine) → **morning** to protect sleep. Libido (PT-141) → hours before activity. Long-half-life compounds (semaglutide, CJC-with-DAC) → timing-insensitive; pick a consistent day.

A DAY ON A STACK

How timing maps onto the clock when several categories run together.

The big GH pulse rides early deep sleep, so secretagogues anchor the night. Fat mobilizers anchor the fasted/empty windows. Weekly compounds float.

CLASS	BEST WINDOW	WHY
GH SECRETAGOGUES	Before bed (empty stomach)	Amplify nocturnal GH pulse
FAT MOBILIZERS	Fasted AM / pre-workout	Low insulin, fat available
HEALING PEPTIDES	Flexible, often split AM/PM	Steady tissue exposure
GLP-1S	Any consistent day	Weekly half-life
STIMULANT COGNITIVES	Morning/daytime	Avoid sleep disruption

5.2 · CYCLING

TIME ON VS. TIME OFF

"Do I need to cycle off?" depends entirely on the mechanism. The right answer differs across the categories.

RECEPTOR DESENSITIZATION IS THE MAIN REASON TO CYCLE

Some peptides lose effect if the receptor adapts to constant stimulation. GHRPs/ghrelin mimetics can show some receptor downregulation over long continuous use, which is part of why GH protocols are often run in 12–16 week blocks with breaks, or pulsed.

CATEGORY	TYPICAL PATTERN	REASONING
HEALING (BPC/TB-500)	Run the injury window (4–8 wks), then off	Job is done when healed; no reason to continue
GH SECRETAGOGUES	12–16 wk blocks, then break	Receptor sensitivity; IGF-1 management
GLP-1S	Long-term, titrated; weight regain risk on stopping	Chronic appetite tool, not a cycle
LONGEVITY (EPITHALON, NAD+)	Pulsed courses a few times/year	Designed as periodic "courses"
IGF-1 LR3	Short (~4 wk) blocks	Risk & receptor concerns
MELANOTAN	Build-up then pause	Reach target tan, then maintain

> **THE GLP-1 EXCEPTION WORTH KNOWING**
>
> GLP-1s aren't really "cycled", they're a chronic management tool. The well-documented catch is that stopping often brings significant weight regain as appetite returns, unless habits and a maintenance plan are firmly in place. People should go in understanding it may be long-term, and plan the off-ramp deliberately.

5.3 · COMBINING

HOW TO STACK

Good stacking means combining compounds whose mechanisms complement each other to cover one goal from multiple angles, not piling on redundancy.

THE TWO REAL STACKING RULES

- **Pair across mechanisms, not within them.** A GHRH + a GHRP (CJC + ipamorelin) is synergistic because they hit two different pathways to the same outcome. Two GHRPs together mostly just compete.
- **One primary, supporting cast.** Pick the main driver for the goal, then add support compounds that fix the primary's gaps (e.g., GLP-1 for appetite + protein/training + GH support to protect muscle).

THE ESTABLISHED, NAMED STACKS

STACK	COMPONENTS	PURPOSE
WOLVERINE	BPC-157 + TB-500	Injury & recovery
GLOW	BPC-157 + TB-500 + GHK-Cu	Skin, joints, anti-inflammatory
KLOW	GLOW + KPV	Adds gut + full-body anti-inflammatory
CJC + IPAMORELIN	GHRH + GHRP	The GH-optimization backbone
LEVEL UP X	CJC/Ipa + Tesamorelin	Fat loss + lean muscle + visceral fat
SUMMER SHRED	CJC/Ipa + BPC-157	GH for body comp + recovery

> **WHAT NOT TO DO**
>
> Don't stack five compounds with overlapping mechanisms and assume more effect. Don't combine multiple things that all raise IGF-1 or all stress glucose without monitoring. And don't add the high-risk anabolics (IGF-1, insulin) to a stack to "round it out". Those change the risk profile of the entire protocol.

5.4 · SEX DIFFERENCES

MEN VS. WOMEN

For most peptides the mechanism is identical between sexes, but dosing, side-effect profiles, and a few specific compounds differ in important ways.

WHERE IT'S LARGELY THE SAME

Healing peptides (BPC-157, TB-500, GHK-Cu, KPV), GH secretagogues, and most longevity/cognitive peptides work through pathways present in both sexes. The biggest practical adjustment is usually just dose scaling to bodyweight, women, on average lighter, often sit at the lower end of cited ranges.

WHERE IT GENUINELY DIFFERS

- **GLP-1s:** Women tend to lose weight effectively but sometimes report more nausea; titration patience matters even more. Pregnancy is an absolute contraindication, and these can affect oral contraceptive absorption via slowed gastric emptying, a real, often-missed point.
- **IGF-1 & anabolics:** Women are far more sensitive to anything androgenic or strongly anabolic. IGF-1 LR3 and insulin carry the same dangers for everyone but require even more conservative handling in women.
- **Melanotan II:** The libido and appetite effects show up in both sexes; mole/melanoma vigilance applies to all.
- **PT-141:** Notably, FDA-approved for women's sexual dysfunction specifically, one of the few with a female-first indication.
- **GH peptides & water retention:** Side effects like water retention and carpal-tunnel symptoms can present differently; women often do well starting low.

> **PREGNANCY & FERTILITY FLAG**
>
> Most of these compounds are contraindicated in pregnancy and many have unknown effects on fertility. Anyone who could become pregnant should treat that as a hard stop and discuss it with a physician before starting anything in this guide.

PART SIX

QUALITY, SAFETY & MONITORING

The part the marketing skips. Sourcing, bloodwork, and an honest read on which compounds actually have evidence behind them.

6.1 · QUALITY

SOURCING & PURITY

"Quality and dosing matter a lot" is the most important fact on every peptide list, because the gray market is full of underdosed, mislabeled, and contaminated product.

Because most research peptides exist in a legal gray zone (sold "for research, not for human consumption"), there's no FDA oversight of what's actually in the vial. The same compound from two vendors can differ wildly in purity, actual dose, and contamination. This is where most bad outcomes and "it didn't work" stories come from, not the peptide failing, but the product not being what the label claimed.

WHAT SEPARATES REAL PRODUCT FROM GARBAGE

- **Third-party testing**, independent COAs (certificates of analysis) verifying identity and purity, ideally batch-specific.
- **Purity %**, reputable material is typically tested ≥98%; mystery purity is a red flag.
- **Proper lyophilization & handling**, correct freeze-drying, cold-chain shipping, accurate fill weights.
- **Reconstitution & storage discipline on your end**, bacteriostatic water, refrigeration, respecting degradation timelines.

> **THE CONTAMINATION RISK THAT'S UNDERRATED**
>
> Beyond wrong dose, gray-market vials can carry endotoxins or bacterial contamination from sloppy manufacturing. Injecting that is a real infection/immune risk. Sterile technique and verified-clean source material aren't optional details, they're the difference between a tool and a hazard.

6.2 · MONITORING

BLOODWORK & WHAT TO TRACK

If there's one habit that separates responsible use from reckless use, it's regular bloodwork. You can't manage what you don't measure.

IF RUNNING…	WATCH THESE MARKERS
GH SECRETAGOGUES / GH	IGF-1, fasting glucose, HbA1c, insulin sensitivity
GLP-1S	Hydration/electrolytes, lipase (pancreas), thyroid history, weight composition
IGF-1 / INSULIN	Glucose (constant), HbA1c, plus the acute hypoglycemia danger
ENHANCED-ATHLETE BASE	Lipids, blood pressure, hematocrit/viscosity, liver & kidney panels
MELANOTAN	Dermatology/skin checks for mole changes
GENERAL	CBC, CMP, lipids, baseline before, then periodically

The pattern: get a **baseline before starting**, recheck during, and adjust based on data rather than feel. For anyone stacking metabolic or GH-axis compounds, glucose regulation is

the recurring theme. That's the system these compounds most often nudge in the wrong direction over time.

6.3 · THE HONEST READ

EVIDENCE SCORECARD

The single most useful filter in this whole space: knowing which bucket each compound sits in. The marketing flattens these together; the evidence does not.

A **PROVEN · FDA-APPROVED** Mountains of human data. Known risk profiles.

B **LATE-STAGE / STRONG TRIALS** Promising human data, approval pending or partial.

C **MECHANISTIC + ANECDOTAL** Good rationale, animal data, heavy real-world use, thin RCTs

D **EARLY / SPECULATIVE** Animal-only or preclinical. Treat claims with maximum caution.

EVIDENCE TIER	COMPOUNDS
STRONG HUMAN DATA / FDA-APPROVED	Semaglutide, Tirzepatide, Tesamorelin, Insulin, PT-141, Cerebrolysin (where approved)
LATE-STAGE / PROMISING HUMAN TRIALS	Retatrutide, Cagrilintide, SS-31, Thymosin Alpha-1
MECHANISTIC + ANECDOTAL, THIN HUMAN RCTS	BPC-157, TB-500, GHK-Cu, KPV, the GH secretagogues, MOTS-c, AOD-9604, MK-677, Semax, Selank, Epithalon
EARLY / ANIMAL-ONLY / SPECULATIVE	SLU-PP-332, Follistatin (injectable), 5-Amino-1MQ (newer), Dihexa

HOW TO USE THIS TABLE

It doesn't mean tier 3 and 4 compounds don't work, many users swear by BPC-157. It means the **confidence and risk-awareness** you bring should scale to the evidence. The approved drugs have known risk profiles; the speculative ones have unknown ones. Respect the difference, and never let a confident marketing claim substitute for that distinction.

THE 10 THINGS TO ACTUALLY INTERNALIZE

1. Peptides are signaling molecules, not steroids. They ask, steroids force.
2. Your body already runs on peptides; most of these copy or extend native signals.
3. They work through receptors, so they're selective, and can't force impossible results.
4. GLP-1s are the most proven, most powerful, and genuinely revolutionary class.
5. The muscle-loss issue on GLP-1s is real and solved with protein + lifting.
6. Most muscle "from peptides" is actually better recovery, sleep, and body comp.
7. IGF-1 and insulin are the dangerous ones, insulin can be fatal. No shortcuts.
8. Timing follows biology: GH at night, fat mobilizers fasted, consistency over cleverness.
9. Stack across mechanisms, not within; one primary + supporting cast.
10. Quality, dosing, sourcing, and bloodwork decide outcomes more than the compound choice.

PART SEVEN

APPENDICES & REFERENCE TABLES

The working reference layer, reconstitution math, technique, master tables, a glossary, and a one-page cheat sheet.

APPENDIX A.1

RECONSTITUTION & DRAW-SIZE MATH

The math that intimidates people is genuinely simple once you see it worked out. Two formulas cover everything.

THE TWO FORMULAS

1. Concentration = total mcg in vial ÷ mL of water added.
2. Units to draw = desired dose (mcg) ÷ concentration per unit.

On a U-100 insulin syringe, 100 units = 1 mL, so concentration-per-unit = (mcg/mL) ÷ 100.

WORKED EXAMPLE, STEP BY STEP

A 5 mg vial (= 5000 mcg), reconstituted with 2 mL bacteriostatic water:

- Concentration = 5000 ÷ 2 = **2500 mcg/mL**
- Per insulin unit = 2500 ÷ 100 = **25 mcg/unit**
- Want a 250 mcg dose? → 250 ÷ 25 = **10 units**
- Want a 500 mcg dose? → 500 ÷ 25 = **20 units**

MASTER DRAW-SIZE TABLE

Units to draw on a U-100 insulin syringe, by vial size, water added, and target dose. Find your row, read across.

VIAL	WATER	CONC.	250 MCG	500 MCG	1 MG	2 MG
5 MG	1 mL	50 mcg/u	5 u	10 u	20 u	40 u
5 MG	2 mL	25 mcg/u	10 u	20 u	40 u	80 u
10 MG	2 mL	50 mcg/u	5 u	10 u	20 u	40 u
10 MG	1 mL	100 mcg/u	2.5 u	5 u	10 u	20 u
2 MG	1 mL	20 mcg/u	12.5 u	25 u	50 u	100 u
2 MG	2 mL	10 mcg/u	25 u	50 u	100 u	n/a

THE TWO MISTAKES PEOPLE MAKE

Confusing **mg and mcg** (1 mg = 1000 mcg) is the most common, and dangerous, error. And more water doesn't mean more peptide; it only changes how many units a dose works out to. The amount of active compound is fixed by the vial; water just sets the dilution.

APPENDIX A.2

INJECTION TECHNIQUE & SITES

Most research peptides are subcutaneous, into the fat layer, not muscle. The goal is consistency and sterility, not bravery.

SUBCUTANEOUS SITES (THE COMMON ONES)

- **Abdomen**, the classic; stay ~2 inches away from the navel, lots of real estate.
- **Love handles / flanks**, easy pinch, well-tolerated.
- **Outer/upper thigh and back of upper arm**, useful for rotation.

STERILE TECHNIQUE, THE NON-NEGOTIABLES

- Wash hands; alcohol-swab the vial top and the injection site, let it dry.
- Pinch the skin to lift the fat layer; insert at the angle the needle length calls for (short insulin needles often 90°, longer at 45°).
- Inject slowly, withdraw, light pressure. Rotate sites to avoid lumps and lipohypertrophy.
- One needle, one use. Never share. Dispose in a sharps container.

> **SUBQ VS IM: WHEN IT MATTERS**
>
> Most peptides (BPC-157, GH secretagogues, GLP-1s) are subcutaneous. A few compounds or preferences call for intramuscular (e.g., some run BPC near an injury site, some carnitine IM). SubQ is the default, slower-absorbing, and far more forgiving. When in doubt, the literature for that specific compound, not bravado, sets the route.

> **INSULIN IS THE EXCEPTION TO "FORGIVING"**
>
> Everything above describes low-risk subcutaneous peptides. Insulin's dosing and timing carry life-threatening hypoglycemia risk and are not covered as a self-administration procedure here, see §3.4.

APPENDIX A.3

STORAGE & HANDLING

Peptides are fragile proteins. Mishandling silently destroys potency, which is why "it didn't work" is often a storage problem, not a compound problem.

STATE	STORAGE	NOTES
LYOPHILIZED (POWDER)	Fridge ideal; freezer for long-term	Most stable form; protect from light & heat
RECONSTITUTED (LIQUID)	Refrigerated, 2–8°C	Use within weeks; potency declines over time
IN TRANSIT	Cold chain preferred	Brief room-temp exposure usually tolerated for powder

HANDLING RULES

- Reconstitute gently, aim the water stream down the vial wall, swirl, **don't shake** (shaking can shear/denature the peptide).
- Use bacteriostatic water for multi-use vials (the benzyl alcohol inhibits bacterial growth); sterile water is single-use only.
- Keep out of direct light and heat at all times.
- Discolored, cloudy, or particulate solution = discard.

WHY THIS IS IN THE SAME CONVERSATION AS QUALITY

You can buy verified, high-purity material and still end up with a dead dose if it's shaken, left warm, or kept reconstituted too long. Sourcing and handling are two halves of the same outcome.

APPENDIX A.4

MASTER REFERENCE TABLE

Every major compound at a glance, category, typical dose reference, frequency, and route. Dosing is reported for education, not as instruction.

COMPOUND	CATEGORY	TYPICAL DOSE REF.	FREQ.	ROUTE
BPC-157	Healing	250–500 mcg	1–2×/day	SC
TB-500	Healing	2–2.5 mg load	2×/wk	SC
KPV	Gut/anti-inflam	200–500 mcg	Daily	SC/oral

COMPOUND	CATEGORY	TYPICAL DOSE REF.	FREQ.	ROUTE
GHK-CU	Skin/healing	1–2 mg	Daily	SC/ topical
IPAMORELIN	GH secretagogue	100–300 mcg	1–3×/day	SC
CJC-1295 (NO DAC)	GH secretagogue	~100 mcg	1–3×/day	SC
CJC-1295 (DAC)	GH secretagogue	1–2 mg	Weekly	SC
TESAMORELIN	GH / visceral fat	1–2 mg	Daily	SC
SERMORELIN	GH secretagogue	200–500 mcg	Nightly	SC
MK-677	GH (oral)	10–25 mg	Daily	Oral
AOD-9604	Fat loss	250–500 mcg	Daily	SC
5-AMINO-1MQ	Fat loss	50–100 mg	Daily	Oral
MOTS-C	Metabolic/mito	5–10 mg	2–3×/wk	SC
SEMAGLUTIDE	GLP-1	0.25→2.4 mg	Weekly	SC/oral
TIRZEPATIDE	GLP-1+GIP	2.5→15 mg	Weekly	SC
RETATRUTIDE*	Triple agonist	trial-defined	Weekly	SC
IGF-1 LR3	Muscle (adv.)	20–50 mcg	Daily	SC
PEG-MGF	Muscle	200–400 mcg	Post-workout	SC/IM
IGF-1 DES	Muscle (local)	50–150 mcg	Post-workout	IM (site)
MELANOTAN I	Tan	low daily	Daily/build	SC
MELANOTAN II	Tan (+libido)	0.25–1 mg	Daily/EOD	SC
PT-141	Sexual health	1–2 mg	As needed	SC/nasal

COMPOUND	CATEGORY	TYPICAL DOSE REF.	FREQ.	ROUTE
TADALAFIL	Sexual health	5–20 mg	Daily/PRN	Oral
KISSPEPTIN	Sexual/fertility	research	varies	SC
EPITHALON	Longevity/skin	5–10 mg	Course	SC
NAD+	Longevity	varies	Course/ maint.	SC/IV
SEMAX / SELANK	Cognitive	nasal	Daily	Nasal
GLUTATHIONE	Support	varies	varies	IM/IV/ oral
L-CARNITINE	Support	200–500 mg	Pre-cardio	SC/IM
GONADORELIN	HPTA support	low mcg	2–3×/wk	SC
THYMOSIN A-1	Immune	~1.5 mg	2×/wk	SC

**Investigational. SC = subcutaneous, IM = intramuscular, IV = intravenous, EOD = every other day. All figures are literature/ practice references, not recommendations.*

APPENDIX A.5

SIDE-EFFECT QUICK REFERENCE

The realistic, category-level side-effect map. "Lower risk than steroids" never means "no risk."

COMPOUND / CLASS	COMMON / MANAGEABLE	SERIOUS, FLAG IT
GLP-1S	Nausea, constipation, fatigue, sulfur burps	Pancreatitis, gallstones, thyroid-tumor warning
GH SECRETAGOGUES	Water retention, tingling, hunger (some)	↑ glucose / insulin resistance, ↑ IGF-1
MK-677	Appetite, water retention, lethargy	Blood-sugar elevation over time
IGF-1 LR3 / DES / PEG-MGF	Hypoglycemia, fullness	Organ growth, cancer-risk theory (IGF family)

COMPOUND / CLASS	COMMON / MANAGEABLE	SERIOUS, FLAG IT
INSULIN	n/a	**Fatal hypoglycemia, highest-risk compound here**
MELANOTAN II	Nausea, flushing, freckling	Mole changes, melanoma vigilance
PT-141	Nausea, flushing, BP rise	Watch BP; not for uncontrolled hypertension
TADALAFIL	Headache, flushing, congestion	Never with nitrates (dangerous BP drop)
BPC-157 / TB-500	Injection-site reaction, head rush (TB)	Largely unknown long-term human data
NAD+	Flushing, chest pressure if infused fast	n/a
ADIPOTIDE / GW-501516	n/a	Kidney toxicity (Adipotide); rodent cancer signal (GW)
TESOFENSINE	Insomnia, dry mouth	↑ heart rate / BP, mood effects

THE COMMON THREAD

Two themes dominate the "serious" column: **glucose dysregulation** (GH axis, IGF-1, insulin) and **under-studied long-term effects** (the research peptides). Bloodwork addresses the first; humility addresses the second.

APPENDIX A.6

MYTHS VS. FACTS

MYTH: "PEPTIDES ARE JUST SAFER STEROIDS."

Fact: Different class entirely. They signal rather than force, mostly don't suppress your hormonal axis, and (outside IGF-1/insulin) don't build steroid-like muscle. Not "safer steroids", different tools for different jobs.

MYTH: "MORE PEPTIDE = MORE RESULTS."

Fact: Receptor-based signaling saturates. Past the effective dose you mostly add side effects, not benefit. Dose precision beats dose size.

MYTH: "NATURAL GH PEPTIDES HAVE NO DOWNSIDE."

Fact: Raising GH/IGF-1 affects glucose, water balance, and carries the same IGF-1-related theoretical risks as GH. "Natural" describes the mechanism, not the safety.

MYTH: "GLP-1S MELT FAT DIRECTLY."

Fact: They suppress appetite. The fat loss comes from the deficit that creates. They make a diet effortless. They aren't a metabolic magic trick (retatrutide's glucagon arm is the partial exception).

MYTH: "IF IT'S SOLD AS A RESEARCH PEPTIDE, IT'S TESTED AND CLEAN."

Fact: The gray market has no oversight. Purity, dose accuracy, and contamination vary wildly. Third-party COAs exist precisely because the label often lies.

MYTH: "HEALING PEPTIDES ARE CLINICALLY PROVEN."

Fact: BPC-157 and friends have strong animal data and huge anecdotal followings, but thin human RCTs. Promising ≠ proven. Keep the two buckets separate.

APPENDIX A.7

GLOSSARY

AGONIST	A molecule that activates a receptor (vs. antagonist, which blocks it).
BACTERIOSTATIC WATER	Water with benzyl alcohol that inhibits bacterial growth, for multi-use vials.
COA	Certificate of Analysis, third-party lab proof of identity and purity.
DPP-4	The enzyme that rapidly degrades native GLP-1; drugs are built to resist it.
GHRH	Growth-hormone-releasing hormone; analogs tell the pituitary to release GH.
GHRP	Growth-hormone-releasing peptide; ghrelin-receptor agonists that pulse GH.
GIP	An incretin hormone; the second receptor tirzepatide targets.
GLP-1	Gut incretin hormone driving insulin, satiety, and slowed gastric emptying.
HALF-LIFE	Time to clear half a dose; sets how often you dose.

HPTA	Hypothalamic-pituitary-testicular axis, the natural hormone feedback loop.
IGF-1	Insulin-like growth factor 1; the anabolic hormone GH works through.
INCRETIN	Gut hormones (GLP-1, GIP) released after eating to manage nutrients.
LIPOLYSIS	Breakdown of stored fat for fuel.
LYOPHILIZED	Freeze-dried; the powder form most peptides ship in.
RECONSTITUTION	Adding water to powder to make an injectable solution.
SECRETAGOGUE	A compound that makes a gland secrete its own hormone.
SOMATOSTATIN	The body's "brake" on GH release; some peptides suppress it.
SUBCUTANEOUS (SC)	Into the fat layer under the skin, the default peptide route.
TITRATION	Gradually increasing a dose to build tolerance (key for GLP-1s).
VISCERAL FAT	Deep fat around the organs; tesamorelin's specialty.

APPENDIX A.8

ONE-PAGE GOAL CHEAT SHEET

The whole guide compressed. Primary driver first, support cast after.

GOAL	PRIMARY	SUPPORT CAST
INJURY / RECOVERY	BPC-157 + TB-500	GHK-Cu, GH axis, KPV (gut)
FAT LOSS	GLP-1 or Tesamorelin	AOD-9604, L-carnitine, MOTS-c, GH axis
MUSCLE (RECOVERY-LED)	CJC-1295 + Ipamorelin	MK-677, BPC-157, sleep
MUSCLE / BULKING (ADVANCED)	IGF-1 LR3 + PEG-MGF	IGF-1 DES, insulin (see cautions)
LOOKSMAXING, SKIN/HAIR	GHK-Cu	Epithalon, NAD+, GH axis
LOOKSMAXING, TAN	Melanotan I	(MT-II if stronger effect wanted)
LONGEVITY	NAD+ + MOTS-c	Epithalon, SS-31, Humanin

GOAL	PRIMARY	SUPPORT CAST
COGNITIVE	Semax + Selank	Cerebrolysin (clinical)
SEXUAL HEALTH	PT-141 + Tadalafil	Kisspeptin, hormonal base
AXIS / IMMUNE SUPPORT	Gonadorelin	Thymosin α-1, LL-37

THE FOUR RULES TO CARRY OUT THE DOOR

1. Match the compound to the goal, one primary, not five overlapping. **2.** Time it to biology, GH at night, fat mobilizers fasted. **3.** Source verified, store correctly, dose precisely. **4.** Get baseline bloodwork and recheck. Everything else in this book is detail hanging off those four.

HelixNova Peptides, Research Division. Educational reference only; not medical advice, not a prescription, not an endorsement of self-administration. Many compounds described are not approved for human use. Decisions belong with a licensed physician and current bloodwork.