THE COMPOUND

a sunday briefing

The 2026 Peptide Stack Map

14 compounds, their mechanisms, half-lives, and where the literature lives.

for founder-operators

FOR RESEARCH USE ONLY. NOT MEDICAL ADVICE.

Nothing in this document is a recommendation to administer, prescribe, or self-administer any compound.

14 compounds, their mechanisms, half-lives, and where the literature lives.

By The Compound — the Sunday briefing on peptides for founder-operators.

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What this is

A working reference. Not a protocol. Not a sales sheet.

If you already buy peptides and you read six newsletters on Sunday to assemble the picture, this is the picture — assembled. Fourteen compounds, one page each, mechanism through literature. Built for the operator who wants to know what each molecule actually does before he reads anyone's stack post.

We do not list doses anywhere in this document. Doses live behind the email gate, in cited issue summaries, where they belong. Mechanism, half-life, and literature pointers are the rails this document runs on.

If you want the weekly briefing — what's new, what's worth the vial, what survived peer review this week — subscribe at the link at the end.

Disclosure: The operator who publishes The Compound also owns heroxbio.com, an RUO peptide vendor. Full FTC disclosure on the About page.

How to read this map

Each section is structured the same way:

• **What it is** — mechanism class in one line.

• **Mechanism** — plain English, one technical term unpacked, no further.

• **Half-life** — numeric where the literature is clean; range where it isn't.

• **Route** — how it's been administered in published work. Not a recommendation.

• **Primary literature** — two to four PubMed-indexable studies. Where we're unsure of an exact citation, we point to the literature class instead of fabricating an author-year.

• **What the founder-biohacker community reports** — neutral summary of forum and N=1 chatter. Not endorsements.

• **What we don't know yet** — the gap. Where the literature thins and bro-science fills the vacuum.

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1. BPC-157

Body Protection Compound 157.

What it is. A 15-amino-acid synthetic peptide derived from a protective sequence in human gastric juice. Recovery and soft-tissue research compound. Often called the recovery anchor of the founder stack.

Mechanism. BPC-157 appears to upregulate growth factor receptors and modulate the nitric oxide system, with downstream effects on angiogenesis — the formation of new blood vessels — at injury sites. Animal models also show modulation of the gut–brain axis and dopaminergic signaling, which is why the literature spans tendons, gut wall, and CNS in ways that surprise people new to it.

Half-life. Short. Animal pharmacokinetic work places systemic half-life under an hour for injected forms; oral stability is debated and is one of the open questions below.

Route. Subcutaneous injection is the dominant route in published work. Oral administration appears in some gastrointestinal studies. Topical and intranasal preparations exist in community circles but the published support for those routes is thin.

Primary literature.

• Sikiric et al. — multiple papers across two decades on gut, tendon, and vascular endpoints in rodent models. This is the foundational corpus; search "Sikiric BPC-157" on PubMed and you have most of it.

• Chang et al., 2011 (Journal of Applied Physiology) on the promoting effect of BPC-157 on tendon healing — outgrowth, cell survival, and cell migration in rat tendon explants.

• Reviews: search "BPC-157 review" for several 2020–2024 narrative reviews summarizing the animal corpus.

What the founder-biohacker community reports. Tendon and joint pain often the first reported change inside two to four weeks. Gut symptoms — particularly NSAID-induced — frequently mentioned. PIP (post-injection pain) is generally low.

What we don't know yet. No large human RCTs. Almost the entire evidence base is preclinical. Long-term safety in humans is unstudied. Oral bioavailability remains contested.

2. TB-500 (Thymosin Beta-4 fragment)

Often shorthand for the active fragment of TB-4.

What it is. A synthetic peptide based on a region of thymosin beta-4, an actin-binding protein. Recovery and soft-tissue research compound, frequently paired with BPC-157 in what forum users call the "Wolverine stack."

Mechanism. TB-4 binds G-actin, the monomer form of the cytoskeletal protein actin, and is implicated in cell migration, angiogenesis, and tissue repair. The TB-500 research peptide is a fragment derived from this sequence, intended to capture the migration-and-repair signaling without the full protein.

Half-life. Reported in the multi-day range for the parent thymosin beta-4 in some pharmacokinetic work; the synthetic fragment's exact half-life in humans is less cleanly characterized.

Route. Subcutaneous injection is the dominant administered route in research contexts.

Primary literature.

• Goldstein et al. — foundational papers on thymosin beta-4 biology.

• RegeneRx clinical trials on thymosin beta-4 (the parent peptide, not the TB-500 fragment) for dry eye and wound healing. These are human trials of TB-4, not the research-fragment, and the distinction matters.

• Search "thymosin beta-4 wound healing" and "thymosin beta-4 cardiac" on PubMed for the substantive corpus.

What the founder-biohacker community reports. Often stacked with BPC-157 for soft-tissue and joint complaints. Reported subjective effects build slower than BPC. Some users describe it as the "systemic" piece and BPC as the "local" piece, though this is community framing, not literature framing.

What we don't know yet. Whether the TB-500 fragment recapitulates full TB-4 activity in humans is not well-characterized. Most published work is on the full protein, not the fragment that vendors sell.

3. GHK-Cu

Glycyl-L-histidyl-L-lysine, copper-bound.

What it is. A naturally occurring tripeptide with high copper-binding affinity. Skin, hair, and tissue-remodeling research compound. The "GLOW stack" cornerstone in community parlance.

Mechanism. GHK forms a complex with copper(II) ions and acts as a copper chaperone — a molecule that delivers a metal ion to specific cellular destinations. Downstream effects in published work include modulation of gene expression related to extracellular matrix remodeling, antioxidant response, and wound repair.

Half-life. Short in plasma — minutes — though tissue residence and copper-delivery effects appear to outlast plasma clearance.

Route. Topical preparations dominate the cosmetic literature. Subcutaneous injection appears in research contexts. Oral GHK is poorly characterized; the peptide is degraded by digestive enzymes.

Primary literature.

• Pickart et al. — multi-decade body of work on GHK-Cu in wound healing, skin, and gene-expression modulation. Loren Pickart is the foundational author; his published reviews are the entry point.

• Pickart and Margolina, 2018 (International Journal of Molecular Sciences) — "Regenerative and Protective Actions of the GHK-Cu Peptide in the Light of the New Gene Data," the gene-expression-era summary of the GHK-Cu corpus.

• Search "GHK copper peptide" on PubMed for cosmetic and dermatologic work.

What the founder-biohacker community reports. Skin texture changes commonly reported within four to eight weeks of consistent topical use. Hair density anecdotes are noisier. Injection-route reports often pair GHK-Cu with BPC and TB-500 in recovery stacks.

What we don't know yet. Systemic effects of injected GHK-Cu in humans are under-characterized. Copper accumulation risk at sustained injected exposure has not been well-studied. Dose-response relationships are unclear outside topical applications.

4. Semaglutide

Brand-name: Ozempic, Wegovy. The compound that broke the GLP-1 conversation open.

What it is. A long-acting GLP-1 receptor agonist. Body composition and metabolic research compound. Approved as a prescription drug in multiple jurisdictions; appears in RUO research markets in non-prescription form.

Mechanism. Semaglutide is a synthetic analog of glucagon-like peptide-1 (GLP-1), a gut hormone released in response to food. It binds the GLP-1 receptor — a G-protein-coupled receptor expressed in pancreatic beta cells, brain, and gut — and produces glucose-dependent insulin secretion, slowed gastric emptying, and central effects on satiety. The molecule is engineered with a fatty acid side chain that binds albumin, dramatically extending its half-life.

Half-life. Approximately 7 days in humans. This is the longest half-life of the GLP-1 agonists in current use and is what enables once-weekly administration.

Route. Subcutaneous injection is dominant. An oral form (Rybelsus) exists but uses a permeation-enhancer formulation and has different pharmacokinetics.

Primary literature.

• STEP trials (STEP 1 through STEP 8) — the registration package for semaglutide in obesity. Wilding et al., 2021 (NEJM, STEP 1) is the highest-cited entry point.

• SUSTAIN trials — diabetes registration program.

• SELECT trial — Lincoff et al., 2023 (NEJM) — semaglutide and cardiovascular outcomes in obesity without diabetes (n=17,604).

What the founder-biohacker community reports. Body composition change is dramatic for most users. GI side effects — nausea, reflux, constipation — are near-universal at initiation and titration steps. Loss of lean mass alongside fat is a common concern; community reports most often describe pairing with resistance training in response. Sleep changes (both ways) come up often.

What we don't know yet. Long-term effects on body composition after discontinuation. Optimal use patterns for non-obese metabolically healthy users (much of the founder cohort) versus the trial populations. Interactions with GH-axis compounds are under-characterized.

5. Tirzepatide

Brand-name: Mounjaro, Zepbound. The dual-agonist that outperformed semaglutide head-to-head.

What it is. A dual GIP and GLP-1 receptor agonist. Body composition and metabolic research compound.

Mechanism. Tirzepatide activates two incretin receptors: GLP-1 (same target as semaglutide) and GIP (glucose-dependent insulinotropic polypeptide), a second incretin hormone. The dual mechanism appears to produce greater weight loss and glycemic effects than GLP-1 mono-agonism in head-to-head trials. The exact contribution of GIP agonism — versus theoretical antagonism — is an active area of mechanism research.

Half-life. Approximately 5 days in humans. Once-weekly administration in published trials.

Route. Subcutaneous injection.

Primary literature.

• SURPASS trials — diabetes registration program. SURPASS-2 head-to-head versus semaglutide is the most-cited.

• SURMOUNT trials — obesity registration. SURMOUNT-1, Jastreboff et al., 2022 (NEJM) is the entry point.

• Frías et al., 2021 (NEJM) — SURPASS-2 head-to-head publication.

What the founder-biohacker community reports. Stronger appetite suppression than semaglutide at equivalent weight-loss effect for many users. Some users report better sleep on tirz than sema; others report worse. PIP variable. The "bloat at titration" complaint is common in community reports.

What we don't know yet. Whether GIP agonism, antagonism, or partial agonism is the optimal pharmacology — there's a real scientific debate here. Long-term comparative outcomes versus semaglutide. Effects on lean mass relative to GLP-1 mono-agonists.

6. Retatrutide

The triple agonist. Phase 3 in 2026.

What it is. A triple agonist at GLP-1, GIP, and glucagon receptors. Investigational body composition and metabolic compound. The newest entry in the incretin pipeline and the one drawing the most operator attention.

Mechanism. Retatrutide adds glucagon receptor agonism to the dual-incretin profile of tirzepatide. Glucagon receptor activation increases energy expenditure and lipolysis — the breakdown of triglycerides into free fatty acids — which is the mechanism hypothesized to drive the larger weight-loss effect observed in early trials. The triple-agonist design is the most ambitious molecular bet in this class to date.

Half-life. Reported in the approximate 6-day range in early pharmacokinetic work. Once-weekly administration in trials.

Route. Subcutaneous injection.

Primary literature.

• Jastreboff et al., 2023 (NEJM) — Phase 2 obesity trial of retatrutide. This is the headline paper most operators have seen referenced.

• Rosenstock et al., 2023 (The Lancet) — Phase 2 retatrutide trial in type 2 diabetes.

• Phase 3 program (TRIUMPH) — ongoing as of this writing; topline reads will reshape the conversation.

What the founder-biohacker community reports. Substantially stronger appetite and weight effects than tirz at comparable titration positions in early N=1 reports. GI side effects also reported as more pronounced. Slower titration is what community reports describe doing in response. Sleep, energy, and resting heart rate changes appear in field reports more than they did with sema or tirz.

What we don't know yet. Almost everything compared to sema and tirz. Phase 3 outcomes, long-term safety, lean-mass preservation, cardiovascular outcomes — all pending. This is the most exciting and the least-characterized molecule in this document.

7. MK-677 (Ibutamoren)

The orally bioavailable growth hormone secretagogue.

What it is. A non-peptide ghrelin receptor agonist that drives endogenous growth hormone (GH) and IGF-1 release. Body composition, sleep, and recovery research compound. Technically not a peptide; included here because it occupies the same shelf in the operator's stack and the same conversation in the literature.

Mechanism. MK-677 binds the growth hormone secretagogue receptor (GHSR), the same receptor activated by ghrelin, and stimulates the pituitary to release pulsatile GH. Downstream, the liver releases IGF-1. The mechanism mimics endogenous GH release rather than supplying exogenous GH directly.

Half-life. Approximately 4–6 hours in humans, with effects on GH/IGF-1 lasting longer than plasma half-life would suggest due to the cascade lag.

Route. Oral. This is one of MK-677's defining features versus injectable GH secretagogue peptides.

Primary literature.

• Murphy et al., 1999 (Journal of Bone and Mineral Research) — oral MK-677 and markers of bone turnover in healthy and functionally impaired elderly adults; also the broader Murphy et al. 1998 corpus on MK-677 and diet-induced catabolism (J Clin Endocrinol Metab).

• Nass et al., 2008 (Annals of Internal Medicine) — long-term oral administration in older adults.

• Search "MK-677" or "ibutamoren" on PubMed for the substantive corpus.

What the founder-biohacker community reports. Appetite increase is near-universal and is the most common reason users discontinue. Sleep — particularly slow-wave sleep — is the most common reported benefit. Water retention and finger stiffness appear at higher exposures. IGF-1 elevation on bloodwork is consistent and detectable.

What we don't know yet. Long-term effects of sustained IGF-1 elevation on cancer risk are theoretical concerns rather than well-characterized ones, but they are the standing literature debate. Optimal use duration and cycling patterns are not literature-supported.

8. CJC-1295 / Ipamorelin

The injectable GH secretagogue stack.

What it is. A pairing of two peptides: CJC-1295 (a GHRH analog) and ipamorelin (a selective ghrelin-receptor agonist). The injectable counterpart to MK-677, with a different pharmacology and a different operator profile.

Mechanism. CJC-1295 mimics growth hormone–releasing hormone (GHRH) and stimulates the pituitary's GH-releasing pathway. Ipamorelin binds the ghrelin receptor and stimulates a separate GH-release pathway. The two peptides target different receptors that converge on GH release, which is why the stack is more than additive in some published work. Two important versions of CJC-1295 exist — with and without DAC (Drug Affinity Complex) — and they have very different half-lives.

Half-life. Ipamorelin: approximately 2 hours. CJC-1295 without DAC ("Mod GRF 1-29"): minutes to about half an hour. CJC-1295 with DAC: approximately 6–8 days due to albumin binding. Operators who don't know which version they have are flying blind on dose timing.

Route. Subcutaneous injection.

Primary literature.

• Teichman et al., 2006 (Journal of Clinical Endocrinology & Metabolism) — prolonged stimulation of GH and IGF-1 secretion by CJC-1295 in healthy adults; estimated half-life 5.8–8.1 days.

• Raun et al., 1998 (European Journal of Endocrinology) — ipamorelin as the first GHRP-receptor agonist with GH-release selectivity comparable to GHRH; no elevation of ACTH, cortisol, FSH, LH, PRL, or TSH at doses 200-fold above the GH-releasing ED50.

• Search "CJC-1295" and "ipamorelin" on PubMed for the substantive corpus.

What the founder-biohacker community reports. Sleep and recovery are the most common reported benefits. Less appetite increase than MK-677. The "with DAC vs. without DAC" version question is the single most common confusion in community threads. Site-specific PIP is variable.

What we don't know yet. Long-term effects on the GH/IGF-1 axis with sustained use. Whether the stack outperforms either peptide alone in well-controlled human trials is under-characterized.

9. Selank

A short Russian-developed anxiolytic peptide.

What it is. A synthetic heptapeptide based on the immunomodulatory peptide tuftsin. Anxiolytic and cognitive research compound. One of the two "Russian peptides" most likely to appear in a founder cognition stack.

Mechanism. Selank appears to modulate GABAergic signaling — the brain's primary inhibitory neurotransmitter system — and influence BDNF (brain-derived neurotrophic factor) expression. The proposed anxiolytic effect operates without the sedation or dependence profile typical of benzodiazepines, which is the angle that draws operator interest.

Half-life. Short. Plasma half-life is reported in the minutes range; effects appear longer-lasting than plasma kinetics would predict.

Route. Intranasal is the dominant route in the published Russian literature. Subcutaneous injection appears in some research contexts.

Primary literature.

• Russian research corpus — primarily from the Institute of Molecular Genetics (Moscow) and collaborators. Much of the substantive work is in Russian-language journals; English-language reviews provide the entry point.

• Search "Selank anxiolytic" and "Selank BDNF" on PubMed for English-indexed work.

What the founder-biohacker community reports. Calm-without-sedation is the most common descriptor. Onset reported within minutes of intranasal administration. Tolerance development is debated. Often paired with semax in cognition stacks.

What we don't know yet. The English-language literature is thin compared to the Russian corpus, and translation gaps mean Western operators are working with a partial picture. Long-term effects, optimal use patterns, and tolerance dynamics are not well-characterized in indexed literature.

10. Semax

The cognitive-stimulant counterpart to Selank.

What it is. A synthetic heptapeptide derived from a fragment of ACTH (adrenocorticotropic hormone). Cognitive and neurotrophic research compound. The second of the two Russian peptides commonly seen in founder cognition stacks.

Mechanism. Semax is structurally related to ACTH(4–10) but lacks ACTH's hormonal activity. It appears to upregulate BDNF and NGF (nerve growth factor) and modulate dopaminergic and serotonergic signaling. Animal work suggests neuroprotective effects in models of ischemic injury.

Half-life. Short in plasma — minutes — though reported subjective effects last hours, suggesting downstream signaling outlasts the molecule.

Route. Intranasal is the dominant route in published Russian literature.

Primary literature.

• Russian clinical research on stroke recovery — Semax is in the Russian state pharmacopoeia for ischemic stroke and the substantive trials are in Russian-language journals.

• Search "Semax" on PubMed for English-indexed work, including BDNF and dopamine system studies.

What the founder-biohacker community reports. Focus and verbal fluency are the two most common reported subjective changes. Onset within minutes intranasally. Often stacked with selank — selank for anxiolytic balance, semax for activation. The "n-acetyl" variants (NA-Selank, NA-Semax) appear in community use but are less characterized in the literature than the parent peptides.

What we don't know yet. Same gap as Selank — the substantive literature is predominantly Russian-language, and the English-indexed corpus is incomplete. Long-term cognitive effects, tolerance, and optimal use cadence are under-characterized.

11. NAD+ (and precursors NMN, NR)

The longevity research compound that isn't a peptide. Included because every founder-operator stack includes it or asks about it.

What it is. Nicotinamide adenine dinucleotide — a coenzyme present in every cell — and its precursors NMN (nicotinamide mononucleotide) and NR (nicotinamide riboside). Cellular metabolism and longevity research compound.

Mechanism. NAD+ is a substrate for sirtuins (a family of NAD-dependent enzymes implicated in cellular aging) and for PARPs (poly-ADP-ribose polymerases involved in DNA repair). Cellular NAD+ levels decline with age, and the longevity hypothesis is that restoring them — directly or via precursors that the cell converts to NAD+ — supports the enzymes that depend on it.

Half-life. NAD+ itself has a complex pharmacokinetic profile and is not well-characterized as a circulating molecule with a clean half-life. NMN and NR oral pharmacokinetics show plasma elevation within an hour and conversion to cellular NAD+ over hours.

Route. NMN and NR: oral is dominant in published work. NAD+: intravenous infusion is the most-studied route; subcutaneous and intramuscular injection appear in research contexts.

Primary literature.

• Sinclair lab corpus on NAD+ biology and sirtuins — David Sinclair at Harvard is the foundational author.

• Yoshino et al., 2021, on NMN in postmenopausal women (Science).

• Martens et al., 2018, on NR in middle-aged adults.

• Brakedal et al., 2022, on NR in Parkinson's (clinical trial).

What the founder-biohacker community reports. IV NAD+ is described as energizing and unpleasant during administration. Oral NMN and NR are reported as more subtle, with subjective effects on energy and sleep showing at sustained use. Cost is a frequent operator complaint and drives precursor selection.

What we don't know yet. Whether oral precursors (NMN, NR) raise cellular NAD+ to functionally relevant levels in the relevant tissues in healthy adults. Whether plasma NAD+ elevation translates to meaningful longevity outcomes. The clinical-outcome corpus lags the mechanism corpus.

12. Kisspeptin-10

The HPG-axis upstream signal.

What it is. A 10-amino-acid fragment of the kisspeptin protein, the upstream regulator of the hypothalamic-pituitary-gonadal axis. Hormonal and libido research compound.

Mechanism. Kisspeptin signals to GnRH (gonadotropin-releasing hormone) neurons in the hypothalamus, which in turn drive LH (luteinizing hormone) and FSH (follicle-stimulating hormone) release from the pituitary. In men, this drives downstream testosterone production. In women, it's a major regulator of the menstrual cycle. The molecule sits one step upstream of the hormonal cascade most operators are paying attention to.

Half-life. Short. Plasma half-life is reported in the minutes range, with hormonal effects measurable for hours after administration.

Route. Intravenous and subcutaneous routes appear in research contexts.

Primary literature.

• Dhillo et al. — multi-paper corpus from Imperial College London on kisspeptin in healthy men and women, including effects on LH and testosterone.

• Search "kisspeptin-10 LH" and "kisspeptin libido" on PubMed for the substantive corpus.

What the founder-biohacker community reports. Acute effects on libido and sexual response are the most commonly reported subjective changes. Use among men on TRT is debated in community discussion — some argue kisspeptin downstream of exogenous testosterone is redundant; others discuss using it as a way to address endogenous signaling concerns.

What we don't know yet. Whether sustained kisspeptin administration produces tachyphylaxis (receptor desensitization) is debated in the literature. Long-term effects on the HPG axis with chronic exposure are under-characterized. Optimal pulsatility is theoretically important but unclear in practice.

13. AOD-9604

A targeted fragment of human growth hormone.

What it is. A synthetic peptide corresponding to the 176–191 fragment of human growth hormone. Body composition and lipolysis research compound. Originally developed as a non-GH-active fat-loss molecule.

Mechanism. AOD-9604 is the C-terminal fragment of hGH that, in early animal work, appeared to retain the lipolytic effects of full-length GH without the IGF-1-mediated growth effects. The molecule is hypothesized to act on adipose tissue beta-3 adrenergic signaling, though the mechanism is not as cleanly established as the marketing language suggests.

Half-life. Reported in the short-to-moderate range; the published pharmacokinetic record is thinner than for the GLP-1 class.

Route. Subcutaneous injection is the dominant administered route. Oral preparations exist but bioavailability is poorly characterized.

Primary literature.

• Heffernan et al. — early 2000s work on the C-terminal hGH fragment in animal models of obesity.

• The clinical development program — AOD-9604 went through obesity trials in the 2000s without reaching approval; the pivotal data is in the Metabolic Pharmaceuticals trial record.

• Search "AOD-9604" and "hGH 177-191" on PubMed for the substantive corpus.

What the founder-biohacker community reports. Subjective effects on body composition are subtle compared to the GLP-1 class. Often stacked with GH secretagogues or used in cuts where GLP-1 agonism isn't desired. The "no IGF-1 effect" claim is the principal differentiator in operator framing.

What we don't know yet. Whether the lipolytic effect translates to clinically meaningful body-composition change in humans is the standing question — the clinical development program did not produce a clean affirmative answer. Optimal use patterns are not literature-supported.

14. Cerebrolysin

A peptide-and-amino-acid mixture, not a single compound. Included because it sits in the same conversation as Selank, Semax, and other cognitive research compounds.

What it is. A standardized mixture of low-molecular-weight peptides and free amino acids derived from porcine brain tissue. Neurotrophic and recovery research compound. Used clinically in several countries — particularly in Eastern Europe and Asia — for ischemic stroke and neurodegenerative conditions.

Mechanism. Cerebrolysin is hypothesized to act as a multi-component neurotrophic factor mimic — supporting neuronal survival, synaptic plasticity, and BDNF expression. Because it is a mixture rather than a single molecule, the mechanism is not as cleanly definable as for the single-peptide compounds in this document, and that is one of the criticisms in the Western literature.

Half-life. Component-dependent. The mixture has no single half-life; individual peptide and amino acid components clear at different rates.

Route. Intramuscular and intravenous routes are dominant in clinical trial use.

Primary literature.

• CARS (Cerebrolysin and Recovery After Stroke) trials — clinical trial program in ischemic stroke.

• Cochrane reviews on Cerebrolysin in stroke and dementia — the meta-analytic record is the most useful entry point because individual trial quality varies.

• Search "Cerebrolysin" on PubMed for the substantive corpus, including the dementia and traumatic brain injury literature.

What the founder-biohacker community reports. Use is concentrated in cognitive recovery contexts — post-concussion, post-illness cognitive complaints — rather than baseline cognitive enhancement. Onset of subjective effects is reported as gradual over weeks.

What we don't know yet. The Western literature is more skeptical of Cerebrolysin than the Eastern European clinical record, and the question of whether the mixture is reproducible across manufacturers is a live one. Mechanism-of-action specificity is limited by the molecule's multi-component nature.

How operators in the literature and community have framed stacks

A note before you read anyone's stack post — including ours.

Stacking is the act of running multiple compounds at the same time. The operator question is never "is this compound interesting" — by the time you're reading this document, you've already answered yes to that

for half the list. The operator question is "what does adding this to what I'm already running do, and how would I know?"

A few rails for thinking about that.

One variable at a time. The discipline most often cited in N=1 community discussion is changing one thing per cycle. If you start retatrutide, MK-677, and BPC-157 in the same week, the labs and the journal are uninterpretable. The literature doesn't have to design clean experiments; you do, on yourself. Most operators don't. The ones who learn most do.

Mechanism stacking is a different question from "I read both of these on a forum." BPC-157 plus TB-500 is a defensible mechanism stack — local repair plus systemic migration signaling, with overlapping but non-identical pathways. CJC-1295/Ipamorelin plus MK-677 is a defensible mechanism question — two different routes to the same axis, and the question is whether you want both or whether you're just stacking redundancy. Selank plus Semax is a defensible balance question — calming plus activating modulators. Stacks built on mechanism converge with the literature; stacks built on forum momentum diverge from it.

Half-lives drive timing. A peptide with a one-hour half-life and a peptide with a one-week half-life cannot be administered on the same schedule. **The literature has a center and a frontier.** GLP-1 mono-agonism (semaglutide) is the center: phase 3 done, registration data, long-term safety record being built. Triple-agonism (retatrutide) is the frontier: phase 2 read, phase 3 in progress. BPC-157 is preclinical-only. Cerebrolysin has Eastern European clinical record and Western skepticism. Selank and Semax have a Russian-language corpus that doesn't cleanly translate. Knowing where each compound sits on this gradient is more useful than knowing the abstract of any single paper.

COA before everything. Community discussion treats a current third-party COA — confirming identity, purity, and absence of common contaminants — as the threshold below which mechanism discussion is moot.

What you don't know. Most of the compounds in this document have thin human safety records, particularly at the use patterns operators run. The literature you read is almost always describing a different population, route, or duration than your N=1 will. Mechanism is a strong starting point. It is not a substitute for caution.

We write a Sunday briefing on these compounds every week — what's new in the literature, what's worth the vial, what survived peer review, and what didn't. If this document was useful, the weekly is the next step.

Subscribe to The Compound

The 2026 Peptide Stack Map is the front door. The Sunday briefing is the room behind it.

Every Sunday morning, one email: what's new this week in the peptide literature, which compounds moved up or down on the operator stack, what to watch in the next phase 3 read, and the cited literature summaries and stack reasoning that fit better in subscriber issues than on a public page like this one.

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