THE COMPOUND BRIEF

The Early Read Vault

Four emerging peptides, decoded before consensus.

The compounds your Discord won't cover for six months. The literature decoded against tier-evidence. Mechanism class, half-life, primary anchor, what the community will get wrong.

In this Vault

The brief is published Sundays, 9 AM ET. The Conflict Test applies: the publisher operates an RUO peptide vendor (heroxbio.com), and the framework holds whether or not heroxbio sells the compound covered.

Selank + Semax

CLASS: Russian-developed peptides · HALF-LIFE: <1 h plasma (intranasal) · TIER: 2 (Russian RCT) / 4 (Western)

"Modafinil is an addition. Selank is a subtraction. Both move the output. The founder who knows the difference is choosing deliberately."

What it is

Selank is a synthetic heptapeptide analog of tuftsin, developed at the Institute of Molecular Genetics. Anxiolytic via GABA-A modulation, plus BDNF elevation in rodent models. Semax is an ACTH(4-10) analog from the same lab, with NE/DA prefrontal-cortex effects and consistent BDNF upregulation. Both intranasal. Plasma half-life is short — minutes — but CNS effects persist longer, the same Issue 3 nuance.

What the literature actually shows

Selank carries the stronger anxiolytic signal. Multiple Russian RCTs (n=30-80 range) in generalized anxiety and mixed neurotic disorders show meaningful response vs placebo on HARS and HADS. Effect size is real. Therapeutic indication is anxiety-adjacent, not cognitive-enhancement-adjacent.

Semax has the stronger cognitive signal — but in impaired-baseline populations. The ischemia and stroke-recovery literature is deep by Russian clinical standards. Healthy-volunteer cognitive-optimization data is thin. The jump from ischemia-recovery (Tier 2) to a prediction about a healthy founder at a standing desk is Tier 4. Name it as such.

What the community gets wrong

The nootropic blog coverage treats Semax's ischemia literature as if it were a healthy-user cognitive-enhancement trial. The evidence is real. The population generalizability is not established. Those are different statements, and conflating them is how operators end up with misplaced confidence in a compound they do not fully understand.

Where to read further

Russian Academy of Sciences tuftsin and Selank work, 1990s-2010s. Goldstein / Garaci ACTH-fragment series for Semax. Issue 8 of The Compound Brief walks an 8-week n=1 protocol applying Issue 4's framework to both.

Retatrutide

CLASS: Triple GLP-1 / GIP / Gcg agonist · HALF-LIFE: ~6 days · TIER: 2 (active trials)

"The bloat complaint is most consistent with a titration-speed artifact. Accumulation mismatch before steady state. That's a protocol problem, not a compound problem."

What the community is reporting

Bloat complaints cluster at early cycle onset, weeks one through four. They appear disproportionately in threads where the poster describes moving up in dose on a weekly schedule. "Hit 2mg and the bloat started the same week" is a representative structure. The timing is not random. It is dose-step correlated.

The half-life math

Retatrutide has a ~6-day half-life per NEJM 2023 phase 2 PK. The five-half-life rule says steady state is reached at approximately 30 days on a fixed weekly dose. That is not a rounding error. It is five weeks on a stable dose before the receptor signal stabilizes.

An operator who moves from 1mg to 2mg to 3mg at weekly intervals is landing a new dose level before the previous dose has reached steady state. The compound is not behaving the way the trial data describes — the trial data describes a compound at steady state.

What the consensus gets wrong

The community has decided the bloat is a glucagon-axis side effect of triple-agonism. If that were true, bloat would be persistent and dose-proportional. Operators report it clears with slower titration at the same final dose. The glucagon theory loses that differentiating prediction. Accumulation mismatch fits the data; glucagon does not.

Where to read further

NEJM 2023 retatrutide phase 2 pharmacokinetics. STEP-1 (semaglutide) and SURMOUNT-1 (tirzepatide) for comparator titration ladders. Issue 9 of The Compound Brief is the contrarian read in full.

Kisspeptin-10

CLASS: KISS1R agonist (upstream of GnRH) · HALF-LIFE: minutes plasma · TIER: 2 (active reproductive-axis trials)

"Phase 2 reproductive-axis data is real. The longevity adjacency is the speculative layer. The mainstream press is missing this compound entirely."

What it is

Kisspeptin-10 is a synthetic 10-amino-acid fragment of the kisspeptin family, the upstream regulator of GnRH and the HPG axis. Discovered in metastasis-suppressor research, the reproductive role was characterized later by Dhillo, Seminara and others through the 2000s and 2010s. Investigational drug. Plasma minutes; CNS effect outlives plasma.

What the literature actually shows

Tier 2 in active reproductive-medicine trials — IVF triggering, hypogonadism diagnostics, FH-axis modulation. The Phase 2 fertility-clinic protocols are reproducible. The compound is real medicine in a defined clinical lane.

Where the community is running ahead of the data

The longevity-adjacency claim — that activating an upstream HPG regulator is a path to youthful endocrine function — is a Tier 4 mechanistic leap. The trials test fertility endpoints. They do not test a 38-year-old founder's subjective vitality at 8 weeks of nightly subq injections. The mechanism is plausible; the population data is simply not there yet.

Why this is an Early Read

Mainstream coverage of the "14 banned peptides" press wave missed kisspeptin almost entirely. The reclassification discussion at the FDA Pharmacy Compounding Advisory Committee is one of the most under-covered beats in the space. Operators who read the Phase 2 reproductive-axis literature today are six months ahead of the consensus.

GHK-Cu (parenteral)

CLASS: Copper tripeptide · HALF-LIFE: ~1 h plasma · TIER: 2/3 (topical strong, parenteral thin)

"The topical literature is real. The injection community is two decades ahead of the parenteral data. That gap is the story."

What it is

GHK-Cu (Glycyl-L-Histidyl-L-Lysine copper complex) is a 3-amino-acid peptide that occurs naturally in human plasma at declining concentrations after age 20. Loren Pickart's research from the 1970s onward built the topical literature: wound healing, hair-follicle modulation, skin remodeling, fibroblast and stem-cell behavior. Topical use is widespread and well-supported.

Where the data is

Tier 2 evidence — topical: wound healing, post-procedure recovery, scar attenuation. Pickart's body of work, plus dermatologic case series. Cosmetic industry adoption has been extensive.

Where the community is running past the data

The injection community is running GHK-Cu subq for systemic anti-aging effects extrapolated from rodent gene-expression work (Pickart's 2012 paper). The mechanism story — gene-expression modulation toward a younger transcriptome — is real. The human parenteral RCT data for that systemic effect is essentially absent. Operators are pinning a Tier 3 mechanistic claim as if it were a Tier 1 outcome.

Why this is an Early Read

Most peptide newsletters reprint the topical literature as if it carries over to injection use. It does not. The mechanism plausibility carries; the human-systemic effect-size at typical operator doses is not yet characterized. An operator running parenteral GHK-Cu in 2026 is in n=1 Tier-4 territory — fine, but they should name the tier they're working in.

THE EDITORIAL POSTURE

What you're subscribing to.

One compound every Sunday. The brief covers an emerging peptide each week — mechanism class, half-life, primary literature anchor, regulatory status, and what the community is getting wrong. 5 minutes to read.

Tier-evidence framework. Tier 1 = human RCT. Tier 2 = open-label or non-Western trial. Tier 3 = mechanistic + animal. Tier 4 = operator n=1 and community aggregation. Every claim is tagged.

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