THE COMPOUND

a sunday briefing

The Wolverine Stack, Re-Examined

BPC-157, TB-500, and the recovery lane — what the literature actually shows.

for the lifter, the runner, the rebuilder

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

Receptor mechanics, route, and the gap between what the marketing says and what the corpus actually contains.

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

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

What this is

A working reference for the recovery lane. Five compounds that get stacked together by athletes, lifters, runners, jiu-jitsu players, and post-surgery rehab seekers — usually under the search term "Wolverine stack." This document is mechanism-first. It sorts the peer-reviewed corpus from the forum corpus, names what is Tier 1 evidence and what is Tier 3, and flags where the marketing has run ahead of the science. Doses live behind the email gate, in cited issue summaries, where they belong.

What the Wolverine stack is and isn't

The phrase "Wolverine stack" did not come from a clinic or a paper. It came from forums — Reddit threads, X posts, Telegram groups — sometime around 2018 to 2020, attached to a stack of two peptides (BPC-157 and TB-500) that users reported produced soft-tissue and joint changes in weeks. The branding referenced the Marvel character whose mutation is rapid tissue repair. The branding stuck. The science it pointed at is more interesting and more limited than the name suggests.

Here is what the corpus actually contains, in plain terms.

The bulk of published BPC-157 work is animal research — rat tendon, rat gut, rat cardiovascular endpoints — produced over roughly two decades by Predrag Sikiric and his collaborators at the University of Zagreb. This is real, peer-reviewed, mechanistically detailed work. It is also almost entirely preclinical. There are no large randomized human trials of BPC-157 for tendon, ligament, or joint endpoints. There are no FDA-approved BPC-157 products. The compound is sold for research use only.

The bulk of published TB-500 work is, technically, not TB-500 work at all. Most of the corpus is on thymosin beta-4 — the full, naturally occurring 43-amino-acid protein. TB-500 as sold by RUO vendors is a synthetic fragment of that protein. Whether the fragment fully recapitulates the parent protein's behavior in human tissue is not cleanly characterized in the literature. The fragment-versus-parent distinction is the single most important thing readers of forum posts on this stack tend to miss.

The mechanism the literature actually supports — for both compounds — is angiogenesis modulation. New blood vessel formation at injury sites. VEGF (vascular endothelial growth factor) pathway involvement. Cell migration into the injury zone. Extracellular matrix activity. None of this is "regenerates tendons" in the comic-book sense. It is "appears to support the tissue's existing repair machinery in animal models, with mechanistic plausibility for translation to humans, and incomplete human evidence." That is a much narrower claim than the one circulating on social media. It is also — for the operator-cohort reading this — a more useful one, because it tells you what to actually look for when you read a paper.

Tier-wise: most of what this lane rests on is Tier 3. Animal models. Mechanistic in vitro work. A scattering of Tier 2 open-label or small-cohort human work, mostly on the parent protein TB-4 rather than the fragment. Tier 4 — operator and forum n=1 — is where the loud reports live. Tier 1 RCTs in this lane are thin to absent for the fragment versions actually sold.

That is the real shape of the evidence. The rest of this document walks the five compounds in the recovery lane on those terms.

The five compounds in the recovery lane

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. The recovery anchor of the founder stack, and the most-discussed compound in the lane.

Mechanism. BPC-157 appears to upregulate growth factor receptors and modulate the nitric oxide system. Downstream effects in animal models include angiogenesis at injury sites — the formation of new blood vessels, which is one of the rate-limiting steps in tendon and ligament repair. Animal work also shows modulation of the gut–brain axis and protection of gastric tissue against NSAID damage, 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 animal 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. 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 outgrowth, cell survival, and cell migration in rat tendon explants. Several 2020–2024 narrative reviews summarize the animal corpus.

What the community reports. Tendon and joint pain often the first reported change inside two to four weeks of consistent use. Gut symptoms — particularly NSAID-related — frequently mentioned. Post-injection pain is generally low.

What we don't know. No large human RCTs. Almost the entire evidence base is preclinical (Tier 3). Long-term human safety is unstudied. Oral bioavailability remains contested. Whether the rat tendon results translate to human tendon at the doses and routes used by operators is the central open question in this lane.

2. TB-500 / Thymosin Beta-4

Often shorthand for the active fragment of TB-4. The fragment-versus-parent distinction is load-bearing.

What it is. A synthetic peptide based on a region of thymosin beta-4, an actin-binding protein found throughout human tissue. Recovery and soft-tissue research compound, almost always paired with BPC-157 in 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. Cells need to move into the injury zone to repair it; TB-4 appears to support that movement. The TB-500 research peptide is a fragment derived from this sequence, intended to capture the migration-and-repair signaling without administering 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 in published literature.

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

Primary literature. Goldstein et al. — foundational papers on thymosin beta-4 biology going back several decades. RegeneRx clinical trials on thymosin beta-4 — the parent peptide, not the TB-500 fragment — for dry eye and wound healing endpoints. 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 community reports. Often stacked with BPC-157 for soft-tissue and joint complaints. Reported subjective effects build slower than BPC. Some users describe TB-500 as the "systemic" piece and BPC as the "local" piece, though this is community framing, not literature framing.

What we don't know. 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 actually sell. This is the single biggest gap in the Wolverine stack literature.

3. GHK-Cu

Glycyl-L-histidyl-L-lysine, copper-bound. The skin-and-tissue-remodeling tripeptide.

What it is. A naturally occurring tripeptide with high copper-binding affinity. Most often sold for topical skin use, but appears in injectable RUO catalogs and in recovery stacks among operators using it for soft-tissue and joint endpoints rather than skin.

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. The "remodeling" frame is the relevant one for the recovery lane: tendons and ligaments are extracellular matrix structures, and any compound modulating that machinery is at least mechanistically interesting in the recovery context.

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 some 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) is the gene-expression-era summary of the GHK-Cu corpus.

What the community reports. Skin texture changes commonly reported within four to eight weeks of consistent topical use. Injection-route reports from operators using GHK-Cu in recovery stacks — alongside BPC and TB-500 — are noisier but persistent. Reports often describe it as a "third lever" in the stack rather than a primary driver.

What we don't know. 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 outside topical applications are unclear.

4. Pentadeca Arginate (PDA)

The newer variant. Sometimes sold as "BPC-157 PA." The compound where the marketing is furthest ahead of the corpus.

What it is. A synthetic 15-amino-acid peptide that shares the BPC-157 sequence backbone with an arginate salt formulation. Marketed by some vendors as a more stable, more bioavailable version of BPC-157. Entered the RUO market in volume around 2023–2024.

Mechanism. The mechanistic claim — repeated across vendor pages and forum posts — is that PDA produces BPC-157-like effects with improved stability and a different pharmacokinetic profile from the arginate counterion. The mechanism for this stability claim is plausible. Arginate salts of small peptides do behave differently from acetate salts in some preparations. Whether the in-vivo effect profile actually

matches BPC-157 in humans is a separate question, and the human corpus answering it does not yet meaningfully exist.

Half-life. Not cleanly characterized in published literature. Vendor and forum estimates exist; peer-reviewed pharmacokinetic data on the arginate form specifically does not, as of this writing.

Route. Subcutaneous injection in community use. Oral preparations are marketed but unsupported by published human data on this specific salt form.

Primary literature. Thin. The BPC-157 parent corpus (Sikiric et al.) is the closest mechanistic anchor, but it is not PDA-specific. PDA-specific peer-reviewed papers are sparse to absent in the major indices as of early 2026. Most of what circulates is white-paper-style content from vendors and a small number of conference posters.

What the community reports. Operators report effects similar to BPC-157, with some claiming faster onset or more consistent effect. PIP reports vary by vendor. The reports are loud; the underlying corpus is not.

What we don't know. Most of it. Whether PDA is meaningfully different from BPC-157 in human tissue. Whether the arginate form's pharmacokinetic claims hold up. Long-term safety. Comparative effect size against the parent compound. This is the compound in the recovery lane where the gap between the marketing and the corpus is largest. Read vendor claims on PDA with the most skepticism.

5. Larazotide

The gut-recovery adjacent. Briefly, because it's relevant for the post-surgery and high-NSAID cohorts.

What it is. An octapeptide developed as a tight-junction regulator — a compound that modulates the integrity of the intestinal epithelial barrier. Studied in clinical trials for celiac disease as an adjunct to a gluten-free diet.

Mechanism. Larazotide appears to antagonize zonulin signaling at intestinal tight junctions, reducing paracellular permeability. In plain terms: it modulates how leaky the gut wall is. The cohort relevance for this document is narrow but real — athletes recovering from surgery often run high NSAID loads, and high NSAID loads degrade gut barrier integrity, and gut barrier integrity has downstream effects on systemic inflammation that affect tissue repair.

Half-life. Short. Published work on the orally administered form characterizes it as a locally acting compound rather than a systemic one.

Route. Oral. This is one of the few compounds in this neighborhood with meaningful oral bioavailability data.

Primary literature. Larazotide reached Phase 3 development for celiac disease (CeDLara program). Published Phase 2 data exists in peer-reviewed journals — Leffler et al. is the entry point. The Phase 3

readout did not meet its primary endpoint, which is part of the picture and worth reading the trial documents on.

What we don't know. Whether larazotide is useful in non-celiac populations as a recovery-adjunct compound. The published work is celiac-specific. Operator use outside that indication is fully Tier 4.

How operators sequence the stack — patterns from the field

This section describes what operators in forums and community channels report doing. It is not a protocol. It is field reporting on Tier 4 behavior, summarized so a reader of community posts has the shape of the conversation.

The most common pattern: BPC-157 and TB-500 stacked together at the start of a recovery window — acute injury, post-surgical, or aggravation of a chronic problem — with stack length running roughly four to eight weeks before operators describe stepping off. BPC is usually run more frequently than TB-500. Community reports describe subjective tendon and joint changes appearing earlier on BPC and later on TB-500, consistent with the half-life difference described in the literature.

GHK-Cu enters the stack as a third lever in some reports, often when the injury involves skin or scar tissue alongside the soft-tissue component (post-surgery cases, lacerations, road rash in the cycling and motorcycle cohorts).

Pentadeca arginate increasingly substitutes for BPC-157 in newer reports — driven, by the operator's read of the forums, more by vendor marketing than by comparative effect data. Whether this substitution is supported by the corpus is, at this writing, an open question.

Prophylactic use — running the stack continuously in the absence of an active injury, on the theory that it supports general tissue maintenance — appears in operator reports but is not well-supported by published work. The corpus is built on injury models, not maintenance models. Operators running prophylactic stacks are doing so on Tier 4 evidence.

This is the shape of what the field reports. None of it is a recommendation. Doses, frequencies, and stack timing live behind the email gate, with citations.

What mainstream sports-medicine writing gets wrong

Three things, repeatedly.

The universal-warning framing on RUO compounds. Mainstream sports-medicine outlets frequently treat RUO peptides as a single category and warn against the category. The framing collapses the difference between a compound with twenty years of preclinical work behind it (BPC-157) and a

compound with almost no peer-reviewed work at all (PDA). This is a category error. The corpus matters. The vendor matters. The route matters. A blanket "these are all unsafe" reads as authoritative and is, in practice, less useful than a per-compound read.

The TB-500 versus TB-4 conflation. Sports-medicine writing — and a great deal of forum writing — uses "TB-500" and "thymosin beta-4" interchangeably. They are not the same molecule. The published human data is largely on the parent protein. The fragment that vendors sell may or may not produce equivalent effects. Writing that conflates them is, at best, sloppy; at worst, it imports the credibility of TB-4 trials onto a fragment that has not been trialed at the same scale.

The Sikiric corpus read as definitive. The Sikiric body of work on BPC-157 is impressive in volume and consistency. It is also almost entirely rodent. Forum posts and some sports-medicine writing cite the Sikiric papers as if they settled the human question. They did not, because they were not designed to. They built mechanistic plausibility for human translation. That plausibility is real. It is not the same as a Phase 3 readout, and writing that treats it as one is overclaiming.

The honest read of the recovery lane is: mechanistically plausible, preclinically supported, human-trial-thin, vendor-quality-variable, and in the case of PDA, marketing-ahead-of-corpus. That is a more nuanced picture than either the cheerleaders or the warning-sirens tend to draw.

What separates a good vial from a bad vial in this lane

Recovery-lane compounds are predominantly injectable. That changes what matters in vendor selection.

A good vial in this lane will have, at minimum: a Certificate of Analysis (COA) from a third-party lab, identifying the peptide and its purity by mass spectrometry; a sterility test; an endotoxin test; and identity confirmation against the claimed sequence. Endotoxin and sterility are not optional for injectable use — they are the difference between a research compound and a contamination event. The Compound's Issue 2 covers how to read a COA in detail; the short version is that absence of these tests, or presence of a COA that does not name the lab, is a fail.

Vendor-side red flags in this lane: no COA on the product page; COAs that are clearly templated and unchanged across SKUs; sequence claims without identity testing; injectable products without endotoxin data. Any one of these is a reason to source elsewhere.

The operator's RUO vendor in this category is heroxbio.com; full disclosure on the About page. There are other vendors. The point is not to push one — the point is to tell the operator-cohort reading this what to look for, so they can evaluate any vendor on the same axes.

What the Brief covers next

Issues queued behind this lead magnet that extend the recovery-lane material:

• **Issue 4** — BPC-157 dosing summary: every published-rate-and-route from the Sikiric corpus, translated to human-equivalent reference points, with the gap-zones flagged. (Tier 2, behind the email gate.)

• **Issue 5** — TB-500 versus TB-4: a literature read on whether the fragment carries the parent's biology, what the RegeneRx trials actually showed, and what would need to be true for the fragment to behave like the protein.

• **Issue 6** — PDA versus BPC-157 head-to-head: the comparative read on Pentadeca Arginate, including a pharmacokinetic skepticism check on the stability claims.

• **Issue 7** — The post-surgery stack: how operators sequence recovery compounds in the four to twelve weeks following orthopedic surgery, with the gut-barrier-and-NSAID interaction surfaced.

• **Issue 9** — The COA-read, full version: every line on a peptide COA, what it means, and how to spot a forged or templated certificate.

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One issue per week. No clinic upsell. No bro-science. Mechanism, half-life, literature pointers, and the gap between what the marketing says and what the corpus actually contains.

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