

PEPTIDE DUE DILIGENCE

THE PEPTIDE RESEARCH FIELD GUIDE

What researchers study.

What the evidence can actually support.



12 PROFILES / 5 RESEARCH AREAS / PRIMARY-SOURCE LINKS

A curated research atlas with evidence labels, annotated study anchors, naming distinctions and a cross-peptide comparison matrix.

Research education only. No human or veterinary dosing, administration, protocols or treatment recommendations. Clinical studies are summarized to explain evidence, not to recommend use.

Start with the question.

01 / RESEARCH NAVIGATOR

The categories below describe research themes, not proven benefits. Several compounds span categories; no placement is a recommendation.

Research area	Profiles / page	Examples of measured questions
Tissue repair and wound healing	BPC-157 / 4 Thymosin beta-4 / TB-500 / 5	Cell migration, repair signaling and ocular-surface outcomes.
Skin and extracellular matrix	GHK-Cu / 6	Collagen synthesis and connective-tissue accumulation.
Metabolism and energy regulation	MOTS-c / 7 Semaglutide / 8 Retatrutide / 9	Endogenous exercise responses versus clinical body-weight outcomes.
Cognition and neurological research	Semax / 10 Selank / 11	Gene expression, functional connectivity and anxiety-related measures.
Growth hormone signaling	CJC-1295 / 12 Ipamorelin / 13 Sermorelin / 14 Tesamorelin / 15	Endocrine responses, pediatric growth, gut recovery and visceral fat.

Useful comparisons, not a shopping list

Page 16 contrasts commonly confused research claims. Page 17 is a question index and glossary. Page 18 gives you a reusable study-reading card. References are on pages 19-21.

The added value is synthesis: different compounds, models and endpoints placed side by side, with the missing inference made explicit. This is a selected evidence guide, not a systematic review or a complete list of peptides.

Follow the model and endpoint.

02 / READING EVIDENCE

Evidence is specific to a question. A small human imaging study and a large randomized clinical outcome trial are both human research, but they do not carry the same meaning.

Evidence type	What it can tell you	What it cannot settle alone
CELL / EXPLANT	Mechanisms and measurable behavior in a controlled experimental system.	A clinical benefit or long-term risk in people.
ANIMAL	Responses in a whole organism and a defined experimental model.	Transfer to a different species, condition or setting.
HUMAN BIOLOGY	Hormones, endogenous levels, imaging or other measured biological changes.	A meaningful clinical improvement merely because a marker changed.
HUMAN OUTCOME	Effects on specified clinical or functional outcomes in studied participants.	Effects in every population, with every formulation or on every outcome.

Read the primary endpoint before the headline

A primary endpoint is the prespecified main test. Secondary results can be informative, but selecting only a positive secondary result after a null primary test can overstate the finding. An exploratory signal calls for confirmation.

Count uncertainty, not just participants

Check comparison groups, masking, missing data, follow-up, adverse events and independent replication. "Not statistically significant" does not prove identical effects; it means the analysis did not establish the claimed difference.

How to use the labels

Profile labels describe the selected studies, not an approval status, certainty grade or safety rating. Study results belong to the defined molecule and formulation. A supplier name or RUO label does not establish equivalence to a trial product.

BPC-157

01 / TISSUE REPAIR AND WOUND HEALING

A synthetic 15-amino-acid peptide investigated in experimental repair biology.

PRECLINICAL FOCUS

What researchers are asking

How does it affect tendon-cell migration, survival and repair-related signaling in laboratory models?

Annotated study anchor

In a 2011 study, tendon explants and fibroblast experiments showed greater outgrowth and migration, with changes in FAK-paxillin signaling. This is a mechanistic observation in experimental systems. [S1]

Where the human evidence fits

The selected anchor is preclinical. It does not establish a clinical benefit in people. This guide does not treat it as a complete account of every human report.

The inference to avoid

A migration assay is not a healed human tendon. Injury models, assay conditions and tissue outcomes must be distinguished; a proposed pathway is not proof of a clinical effect.

Common misconception

"Repairs injuries" removes the model and outcome from the finding. Prefer: "studied for repair-related cell behavior in tendon models."

Compare with: Compare its model and endpoint with thymosin beta-4 and GHK-Cu; shared repair vocabulary does not imply interchangeable molecules or effects.

Ask while reading: Does the paper measure cell movement, tissue mechanics, or a patient outcome? Which of those is the headline claiming?

[S1] Chang et al. (2011). The promoting effect of pentadecapeptide BPC 157 on tendon healing involves tendon outgrowth, cell survival, and cell migration. J Appl Physiol.

Thymosin beta-4 / TB-500

02 / TISSUE REPAIR AND WOUND HEALING

Thymosin beta-4 and the fragment identified as TB-500 should not be treated as one interchangeable material. FDA identifies a TB-500 fragment of thymosin beta-4. [S3]

IDENTITY MUST COME FIRST

What researchers are asking

Thymosin beta-4 research includes ocular-surface repair and dry-eye outcomes; first establish exactly which molecule a paper studied.

Annotated study anchor

A 2015 randomized, masked phase 2 study enrolled 72 people with dry eye. Neither primary endpoint differed significantly at the specified visit; some secondary measures did. Reporting only the positive secondary results would conceal the main test. [S2]

Where the human evidence fits

Human trial evidence exists for a specific thymosin beta-4 ophthalmic formulation. It cannot be assigned automatically to a differently defined TB-500 fragment or supplier material.

The inference to avoid

Formulation, molecular identity, population and endpoint all matter. A dry-eye study is not a general demonstration of tendon repair.

Common misconception

"TB-500 has clinical trials" is incomplete unless the trial molecule matches the claim.

Compare with: Treat full-length protein versus fragment as an identity question before asking which has stronger evidence.

Ask while reading: Can I match the sequence or defined active entity in the study to the name in the claim?

[S2] Sosne and Ousler (2015). Thymosin beta 4 ophthalmic solution for dry eye: randomized Phase II trial. Clin Ophthalmol.

[S3] FDA GSRS. TB-500 substance record.

GHK-Cu

03 / SKIN AND EXTRACELLULAR MATRIX BIOLOGY

A copper-binding tripeptide complex studied in connective-tissue biology. Free GHK and its copper complex are distinct descriptions.

CELL + ANIMAL ANCHORS

What researchers are asking

How might copper-peptide signaling affect collagen production and extracellular matrix accumulation?

Annotated study anchor

A 1988 fibroblast-culture experiment examined collagen synthesis. A 1993 rat wound-model study examined connective tissue accumulation. These original experiments provide different levels of evidence: cell behavior and an animal tissue response. [S4, S5]

Where the human evidence fits

The selected anchors are not human cosmetic or clinical efficacy trials. They cannot establish how much a finished skin product changes a human outcome.

The inference to avoid

A peptide complex, a delivery system and a finished formulation are different research objects. Concentration at a target, copper coordination and formulation cannot be assumed equivalent.

Common misconception

"Increased collagen in cells" is not the same as "reverses skin aging". That larger claim needs appropriate human evidence.

Compare with: Compare the endpoint, not a generic "regeneration" label: collagen synthesis differs from migration, wound closure or appearance ratings.

Ask while reading: Was the active entity GHK-Cu, another copper complex, or a multi-ingredient formulation?

[S4] Maquart et al. (1988). Stimulation of collagen synthesis in fibroblast cultures by the tripeptide-copper complex glycyl-L-histidyl-L-lysine-Cu²⁺. FEBS Lett.

[S5] Maquart et al. (1993). In vivo stimulation of connective tissue accumulation by GHK-Cu in rat experimental wounds. J Clin Invest.

MOTS-c

04 / METABOLISM AND ENERGY REGULATION

A mitochondrial-derived peptide studied as a signal connecting cellular stress, metabolism and muscle biology.

HUMAN BIOLOGY + MOUSE INTERVENTION

What researchers are asking

How does endogenous MOTS-c respond to exercise, and what happens in experimental models when the peptide is administered?

Annotated study anchor

A 2021 paper measured endogenous MOTS-c in human skeletal muscle and circulation following exercise and separately tested interventions in mice. Its human exercise component involved 10 young men. [S6]

Where the human evidence fits

Human measurements show what the body did in that study. They do not demonstrate that administering a research material improves human exercise performance.

The inference to avoid

Keep the human observational/physiological arm separate from the animal intervention arm. A naturally occurring molecule is not automatically a safe or effective administered product.

Common misconception

"Exercise peptide" is shorthand for a research theme, not a proven substitute for exercise.

Compare with: Semaglutide and retatrutide trial outcomes concern clinical interventions and defined populations; MOTS-c exercise measurements are a different evidence type.

Ask while reading: Which results came from humans, which from mice, and which involved administered peptide?

[S6] Reynolds et al. (2021). MOTS-c is an exercise-induced mitochondrial-encoded regulator of age-dependent physical decline and muscle homeostasis. Nat Commun.

Semaglutide

05 / METABOLISM AND ENERGY REGULATION

A GLP-1 receptor agonist with a substantial clinical research program. This page uses STEP 1 as a selected anchor, not a full indication or labeling review.

LARGE RANDOMIZED HUMAN TRIAL

What researchers are asking

Can a defined clinical intervention change body weight in adults with overweight or obesity without diabetes?

Annotated study anchor

STEP 1 randomized 1,961 adults and included lifestyle intervention in both groups. Mean weight change at 68 weeks was -14.9% with semaglutide versus -2.4% with placebo. Gastrointestinal events were common, and discontinuation due to these events was more frequent with semaglutide. [S7]

Where the human evidence fits

This is direct clinical outcome evidence in a defined population. It is different from a cell marker or a hormone-only study.

The inference to avoid

The finding belongs to the studied intervention and population. It is not an authenticity, equivalence or quality finding for a supplier vial.

Common misconception

"Same peptide name" does not establish that an unapproved product is the studied medicine. FDA distinguishes unapproved versions from reviewed products. [S9]

Compare with: Do not rank it against retatrutide by comparing percentages from separate trials with different designs.

Ask while reading: What population, comparator, follow-up, adverse events and treatment-discontinuation assumptions shaped the result?

[S7] Wilding et al. (2021). Once-Weekly Semaglutide in Adults with Overweight or Obesity. *N Engl J Med*.

[S9] FDA. Concerns with Unapproved GLP-1 Drugs Used for Weight Loss.

Retatrutide

06 / METABOLISM AND ENERGY REGULATION

An investigational agonist of GIP, GLP-1 and glucagon receptors.

SELECTED PHASE 2 HUMAN TRIAL

What researchers are asking

How does multi-receptor agonism affect body-weight outcomes in adults with obesity?

Annotated study anchor

The 2023 phase 2 obesity trial tested retatrutide against placebo across several study groups and followed body weight through 48 weeks. It reported substantial weight reductions; gastrointestinal events and heart-rate changes were part of the safety findings. [S8]

Where the human evidence fits

The anchor is a randomized human trial. "Phase 2" describes this paper, not a claim that development has stopped there. Later trials require their own review.

The inference to avoid

A positive trial is not regulatory approval or validation of online material. FDA currently states retatrutide is not a component of an FDA-approved drug. [S9]

Common misconception

"Three receptors means three times better" is not a scientific comparison. Receptor count is not a clinical ranking.

Compare with: A meaningful comparison with semaglutide needs compatible populations, endpoints and follow-up; preferably a direct trial.

Ask while reading: Am I reading peer-reviewed results, a trial registration, or a company announcement? They provide different kinds of information.

[S8] Jastreboff et al. (2023). Triple-Hormone-Receptor Agonist Retatrutide for Obesity - A Phase 2 Trial. N Engl J Med.

[S9] FDA. Concerns with Unapproved GLP-1 Drugs Used for Weight Loss.

Semax

07 / COGNITION AND NEUROLOGICAL RESEARCH

A synthetic ACTH-fragment-related peptide investigated in neurological research.

ANIMAL MECHANISMS + HUMAN IMAGING

What researchers are asking

How does it affect gene-expression responses after experimental brain ischemia, and are short-term brain-network changes measurable in humans?

Annotated study anchor

A 2014 rat focal-ischemia paper examined immune- and vascular-related gene expression. A separate 2020 study of Semax, Selank and placebo in 52 healthy participants examined resting-state functional connectivity. [S10, S11]

Where the human evidence fits

The cited human study concerns short-term imaging. A connectivity difference is not a demonstration of improved memory, productivity or stroke recovery.

The inference to avoid

Species, injury context and outcome matter. A response in injured rat brain cannot be equated with enhancement in healthy people.

Common misconception

"Nootropic" names a claimed research interest; it does not specify a validated benefit.

Compare with: Semax and Selank appear together in an imaging study but have different molecular backgrounds and research questions.

Ask while reading: Is the outcome a molecular signal, imaging measure, cognitive test or clinical function? Does the claim preserve that distinction?

[S10] Medvedeva et al. (2014). The peptide semax affects expression of genes related to immune and vascular systems in rat brain focal ischemia. BMC Genomics.

[S11] Panikratova et al. (2020). Functional Connectomic Approach to Studying Selank and Semax Effects. Dokl Biol Sci.

Selank

08 / COGNITION AND NEUROLOGICAL RESEARCH

A tuftsin-related synthetic peptide investigated in anxiety-related and neurobiological research.

SMALL HUMAN REPORTS + IMAGING

What researchers are asking

What changes are reported in anxiety-related clinical measures and in short-term functional brain connectivity?

Annotated study anchor

A 2008 clinical report compared Selank with medazepam in patients with generalized anxiety disorder or neurasthenia. The authors reported similar anxiolytic effects. A separate 2020 study examined Selank and Semax effects on imaging measures in healthy participants. [S12, S11]

Where the human evidence fits

Human publications exist, but a clinical report and a short imaging study do not amount to a broadly replicated, long-term evidence base.

The inference to avoid

For the clinical anchor, this guide relies on the indexed English abstract of a Russian-language paper. Full methods, masking, allocation and longer follow-up should be checked before stronger conclusions.

Common misconception

"Comparable to an anxiety medicine" does not establish equivalence, a replacement indication or a safety advantage.

Compare with: Brain connectivity findings and symptom scores are different outcomes. Do not combine them into an unsupported overall effectiveness score.

Ask while reading: Was there placebo control, an active comparator, adequate masking and independent replication?

[S12] Zozulia et al. (2008). Efficacy and possible mechanisms of a new peptide anxiolytic selank in generalized anxiety disorders and neurasthenia.

[S11] Panikratova et al. (2020). Functional Connectomic Approach to Studying Selank and Semax Effects. Dokl Biol Sci.

CJC-1295

09 / GROWTH HORMONE SIGNALING

A long-acting growth hormone-releasing hormone analog studied for sustained stimulation of GH and IGF-I.

EARLY HUMAN ENDOCRINE RESPONSE

What researchers are asking

Can a longer-acting GHRH analog produce sustained endocrine changes in healthy adults?

Annotated study anchor

A 2006 study reported sustained increases in GH and IGF-I in healthy adults. The paper concerns the long-acting CJC-1295 entity. [S13]

Where the human evidence fits

Hormone-response evidence is direct human biology evidence, but it is not a trial demonstrating longer life, injury repair or improved athletic performance.

The inference to avoid

A biomarker is not a clinical outcome. A short study cannot settle long-term risk or benefits in a different population.

Common misconception

"Higher GH" is not synonymous with better health. A direction of biological change is not a value judgment.

Compare with: Do not assume a product labeled "CJC-1295 no DAC" matches this long-acting trial molecule. Require the exact structure and modification before transferring findings.

Ask while reading: Did the paper study the exact analog being discussed, and did it measure hormones or a meaningful clinical outcome?

[S13] Teichman et al. (2006). Prolonged stimulation of GH and IGF-I secretion by CJC-1295 in healthy adults. J Clin Endocrinol Metab.

Ipamorelin

10 / GROWTH HORMONE SIGNALING

A ghrelin-receptor agonist studied as a growth hormone secretagogue and in gastrointestinal-motility research. [S20]

HUMAN PHARMACOLOGY + MIXED RESULTS

What researchers are asking

What is its relationship to GH release, and does ghrelin-receptor stimulation improve postoperative gut recovery?

Annotated study anchor

A 1999 healthy-volunteer study modeled exposure and GH response. In a 2014 randomized postoperative-ileus trial, 117 patients enrolled; key and secondary efficacy analyses did not differ significantly from placebo. [S14, S15]

Where the human evidence fits

Human evidence is present, but its questions range from hormone pharmacology to surgical recovery. Neither is a controlled demonstration of anti-aging or muscle-building benefit.

The inference to avoid

A null clinical result matters even if the mechanism sounds plausible. It does not prove every future use fails, but it prevents calling that trial a success.

Common misconception

"Selective GH release" does not mean risk-free or beneficial for every purpose.

Compare with: Its receptor pathway differs from GHRH analogs such as sermorelin or CJC-1295; combinations require their own evidence.

Ask while reading: Does the headline mention the nonsignificant efficacy finding, or only the mechanistic rationale?

[S14] Gobburu et al. (1999). Pharmacokinetic-pharmacodynamic modeling of ipamorelin in healthy volunteers. *Pharm Res.*

[S15] Beck et al. (2014). Randomized proof-of-concept study of ipamorelin for postoperative ileus in bowel resection patients. *Int J Colorectal Dis.*

[S20] Raun et al. (1998). Ipamorelin, the first selective growth hormone secretagogue. *Eur J Endocrinol.*

Sermorelin

11 / GROWTH HORMONE SIGNALING

GHRH(1-29)-amide, a biologically active fragment of growth hormone-releasing hormone. [S17]

HISTORICAL PEDIATRIC HUMAN RESEARCH

What researchers are asking

Can GHRH stimulation alter growth outcomes in children with growth hormone deficiency?

Annotated study anchor

The 1996 Geref International Study Group report evaluated one year of GHRH therapy in GH-deficient children. Of 110 patients, 86 were eligible for efficacy analysis; the report described accelerated growth during the first year. [S16]

Where the human evidence fits

This is human research in a specific pediatric endocrine setting. It should not be presented as evidence for adult longevity or general wellness.

The inference to avoid

Age, endocrine function, study design and analysis eligibility influence interpretation. Pediatric growth is not a substitute endpoint for adult benefit.

Common misconception

"Studied in humans" is incomplete unless it states which humans and for what outcome.

Compare with: Sermorelin and long-acting CJC-1295 share a signaling theme but are not the same analog. Tesamorelin has a separate formulation and clinical context.

Ask while reading: Are the population and endpoint relevant to the claim, or merely connected by the phrase "growth hormone"?

[S16] Geref International Study Group (1996). GHRH therapy and growth in GH-deficient children during the first year. J Clin Endocrinol Metab.

[S17] NLM MeSH. Sermorelin.

Tesamorelin

12 / GROWTH HORMONE SIGNALING / METABOLISM

A growth hormone-releasing factor analog with research in HIV-associated abdominal fat accumulation.

RANDOMIZED HUMAN OUTCOMES

What researchers are asking

Can it reduce visceral adipose tissue in adults with HIV and excess abdominal fat?

Annotated study anchor

A 2007 randomized trial enrolled 412 patients with HIV. At 26 weeks, visceral adipose tissue decreased 15.2% with tesamorelin and increased 5.0% with placebo. [S18]

Where the human evidence fits

The anchor measures a defined body-composition outcome in a specific clinical population, not just a hormone concentration.

The inference to avoid

Visceral fat, subcutaneous fat and total body weight are different outcomes. The population cannot be generalized to every person seeking fat loss.

Common misconception

"A weight-loss peptide" blurs the indication. FDA labeling for EGRIFTA WR states it is not indicated for weight-loss management and notes that long-term cardiovascular safety is not established. [S19]

Compare with: Compare its outcome and population with semaglutide, rather than placing all metabolic peptides in a single effectiveness ranking.

Ask while reading: Is the claim about an imaging-defined fat compartment, total weight, or a health outcome? Which one did the study test?

[S18] Falutz et al. (2007). Metabolic Effects of a Growth Hormone-Releasing Factor in Patients with HIV. *N Engl J Med*.

[S19] FDA (2025). EGRIFTA WR prescribing information.

Similar headlines, different evidence.

03 / COMPARISON MATRIX

Use the distinction in the final column before making any cross-compound claim. This is not an effectiveness ranking.

Comparison	Selected evidence	Important distinction
BPC-157 vs thymosin beta-4	Tendon-cell work [S1] vs an ocular formulation trial [S2].	Different molecule, model, tissue and endpoint. TB-500 identity needs its own check.
GHK-Cu vs repair peptides	Collagen and wound-model anchors [S4, S5].	A shared repair theme does not establish a shared effect or interchangeable formulation.
MOTS-c vs metabolic trial drugs	Endogenous human exercise measurement [S6] vs clinical interventions [S7, S8].	A natural association is not evidence that external administration reproduces it.
Semaglutide vs retatrutide	Separate randomized studies [S7, S8].	Different populations, designs and follow-up; percentages are not head-to-head estimates.
Semax vs Selank	Rat mechanisms, imaging and a clinical report [S10-S12].	Imaging changes are not durable symptom relief or cognitive improvement.
CJC-1295 vs sermorelin	Early endocrine response vs pediatric growth [S13, S16].	Shared signaling does not mean the same entity, duration or demonstrated outcome.
Ipamorelin vs GHRH analogs	Ghrelin-receptor pharmacology [S20] and clinical studies [S14, S15].	Different receptor pathway. Combination claims require combination-specific evidence.
Tesamorelin vs general weight loss	HIV-associated visceral-fat research [S18, S19].	A specific fat compartment and population cannot stand in for all weight-loss outcomes.

Keep each conclusion in the form: exact entity + model/population + comparator + measured outcome + important limitation.

Find the right research lens.

04 / QUESTION INDEX + GLOSSARY

Start with a measurable question rather than a broad promise such as "healing," "longevity" or "optimization."

If you are exploring...	Read	Look for
Tendon-cell behavior	BPC-157 / 4	Migration or survival versus tissue or patient outcomes.
Ocular-surface repair	Thymosin beta-4 / 5	Exact formulation and primary endpoint.
Collagen / matrix biology	GHK-Cu / 6	Cell cultures versus intact tissue and human outcomes.
Exercise-associated signals	MOTS-c / 7	Endogenous measurements versus interventions.
Clinical body-weight outcomes	Semaglutide / 8; retatrutide / 9	Trial population, duration, comparator and adverse events.
Neurological mechanisms	Semax / 10; Selank / 11	Gene expression, imaging, symptoms or function.
GH and IGF-I responses	CJC-1295 / 12; ipamorelin / 13	A biomarker shift versus a demonstrated benefit.
Pediatric endocrine growth	Sermorelin / 14	Defined deficiency, age and growth outcomes.
Visceral fat compartment	Tesamorelin / 15	Imaging-defined adipose tissue versus total weight.

Five terms worth knowing

Agonist: a molecule that activates a receptor.

Analog: a related molecule with structural changes; findings may not transfer.

Endogenous: produced within the body.

Surrogate / biomarker: a measured indicator that is not necessarily a clinical outcome.

Extracellular matrix: the structural and signaling environment outside cells.

Read one paper more carefully.

05 / REUSABLE STUDY-READING CARD

Print this page or copy the prompts into your notes. It is a research-literacy tool, not a protocol or procurement worksheet.

Identity + citation

Exact molecule / modification / formulation; title, year, DOI or PMID

Model + design

Cell type, animal model or human population; comparator, allocation and masking

Endpoint + finding

Primary outcome, direction and size of result; uncertainty or nonsignificant findings

Limits + missing information

Sample size, follow-up, missing data, adverse events, conflicts and replication

Bounded summary

In [model], [design] reported [outcome] versus [comparator]; it does not establish [broader claim].

Update the record if a correction, retraction or a stronger subsequent study changes the interpretation. A trial registration and a press release are useful leads, but not substitutes for reported study results.

Study anchors / 1

06 / PRIMARY SOURCES

Clickable source records for the numbered citations. Primary-study titles are shortened where needed; PMID links resolve the full bibliographic records.

[S1] Chang et al. (2011). The promoting effect of pentadecapeptide BPC 157 on tendon healing involves tendon outgrowth, cell survival, and cell migration. *J Appl Physiol*.

Cell and tendon-explant experiments; mechanism and model boundaries.

<https://pubmed.ncbi.nlm.nih.gov/21030672/>

[S2] Sosne and Ousler (2015). Thymosin beta 4 ophthalmic solution for dry eye: randomized Phase II trial. *Clin Ophthalmol*.

72-participant study; primary endpoints versus secondary findings.

<https://pubmed.ncbi.nlm.nih.gov/26056426/>

[S3] FDA GSRS. TB-500 substance record.

Identity context for a thymosin beta-4 fragment; not product approval.

<https://precision.fda.gov/ginas/app/ui/substances/e850a4ce-7777-4d25-ae69-ab7174c798a4>

[S4] Maquart et al. (1988). Stimulation of collagen synthesis in fibroblast cultures by the tripeptide-copper complex glycyl-L-histidyl-L-lysine-Cu²⁺. *FEBS Lett*.

Original cell-culture study of GHK-Cu and collagen synthesis.

<https://pubmed.ncbi.nlm.nih.gov/3169264/>

[S5] Maquart et al. (1993). In vivo stimulation of connective tissue accumulation by GHK-Cu in rat experimental wounds. *J Clin Invest*.

Original animal wound-model work; not a clinical outcome trial.

<https://pubmed.ncbi.nlm.nih.gov/8227353/>

[S6] Reynolds et al. (2021). MOTS-c is an exercise-induced mitochondrial-encoded regulator of age-dependent physical decline and muscle homeostasis. *Nat Commun*.

Human endogenous measurements and separate mouse intervention experiments.

<https://pubmed.ncbi.nlm.nih.gov/33473109/>

[S7] Wilding et al. (2021). Once-Weekly Semaglutide in Adults with Overweight or Obesity. *N Engl J Med*.

STEP 1: randomized clinical outcomes, population and adverse-event context.

<https://pubmed.ncbi.nlm.nih.gov/33567185/>

Study anchors / 2

06 / PRIMARY SOURCES

Clickable source records for the numbered citations. Primary-study titles are shortened where needed; PMID links resolve the full bibliographic records.

[S8] Jastreboff et al. (2023). Triple-Hormone-Receptor Agonist Retatrutide for Obesity - A Phase 2 Trial. *N Engl J Med*.

Selected phase 2 trial; not a complete review of subsequent development.

<https://pubmed.ncbi.nlm.nih.gov/37366315/>

[S9] FDA. Concerns with Unapproved GLP-1 Drugs Used for Weight Loss.

Current regulatory distinction between studied or approved products and unapproved materials.

<https://www.fda.gov/drugs/drug-alerts-and-statements/fdas-concerns-unapproved-glp-1-drugs-used-weight-loss>

[S10] Medvedeva et al. (2014). The peptide semax affects expression of genes related to immune and vascular systems in rat brain focal ischemia. *BMC Genomics*.

Original rat ischemia and gene-expression study.

<https://pubmed.ncbi.nlm.nih.gov/24661604/>

[S11] Panikratova et al. (2020). Functional Connectomic Approach to Studying Selank and Semax Effects. *Dokl Biol Sci*.

52 healthy participants; short-term imaging, not durable clinical outcomes.

<https://pubmed.ncbi.nlm.nih.gov/32342318/>

[S12] Zozulia et al. (2008). Efficacy and possible mechanisms of a new peptide anxiolytic selank in generalized anxiety disorders and neurasthenia.

Russian-language clinical report with English abstract; active-comparator context.

<https://pubmed.ncbi.nlm.nih.gov/18454096/>

[S13] Teichman et al. (2006). Prolonged stimulation of GH and IGF-I secretion by CJC-1295 in healthy adults. *J Clin Endocrinol Metab*.

Early human endocrine-response work with the long-acting analog.

<https://pubmed.ncbi.nlm.nih.gov/16352683/>

[S14] Gobburu et al. (1999). Pharmacokinetic-pharmacodynamic modeling of ipamorelin in healthy volunteers. *Pharm Res*.

Human hormone response and exposure modeling.

<https://pubmed.ncbi.nlm.nih.gov/10496658/>

Study anchors / 3

06 / PRIMARY SOURCES

Clickable source records for the numbered citations. Primary-study titles are shortened where needed; PMID links resolve the full bibliographic records.

[S15] Beck et al. (2014). Randomized proof-of-concept study of ipamorelin for postoperative ileus in bowel resection patients. Int J Colorectal Dis.

Key and secondary efficacy analyses did not differ significantly from placebo.

<https://pubmed.ncbi.nlm.nih.gov/25331030/>

[S16] Geref International Study Group (1996). GHRH therapy and growth in GH-deficient children during the first year. J Clin Endocrinol Metab.

Pediatric study; limited transfer to unrelated adult claims.

<https://pubmed.ncbi.nlm.nih.gov/8772599/>

[S17] NLM MeSH. Sermorelin.

Identity: GHRH(1-29)-amide.

<https://www.ncbi.nlm.nih.gov/mesh/?term=D017337>

[S18] Falutz et al. (2007). Metabolic Effects of a Growth Hormone-Releasing Factor in Patients with HIV. N Engl J Med.

Randomized tesamorelin study; visceral fat outcome in a defined population.

<https://pubmed.ncbi.nlm.nih.gov/18057338/>

[S19] FDA (2025). EGRIFTA WR prescribing information.

Specific indication and limitations; not weight-loss management.

https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/022505s020lbl.pdf

[S20] Raun et al. (1998). Ipamorelin, the first selective growth hormone secretagogue. Eur J Endocrinol.

Original pharmacology; selectivity claims are bounded by the tested models.

<https://pubmed.ncbi.nlm.nih.gov/9849822/>

Scope: Selected primary studies and official records checked October 15, 2026. Historical anchors explain study design and inference; inclusion does not mean a study is the latest available. This is not a systematic review, exhaustive safety assessment or clinical recommendation. PubMed indexing is not endorsement.

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