

APEX 100 · 2026 EDITION



FOR RESEARCH USE ONLY

The Peptide *Research* Guide

The science, signaling & standards behind the compounds most studied in modern metabolic, regenerative and cellular research.

AN INDEPENDENT RESEARCH REFERENCE · FOR RESEARCH USE ONLY — NOT FOR HUMAN CONSUMPTION

How to use this guide

This is a research reference. It surveys the receptor biology, published literature, and laboratory standards behind the peptide compounds most studied in metabolic, regenerative, and cellular research today.

Where clinical findings are cited, they describe what the published literature reports about a molecule — the study, its design, its results — provided for scientific context only. Nothing here is a protocol, a dose, or a recommendation for use in humans.

Important — Research Use Only

All compounds referenced are supplied strictly for laboratory and research purposes. They are not drugs, are not approved by the FDA, and are not intended to diagnose, treat, cure, or prevent any condition, or for human or veterinary use.

Reading a research peptide

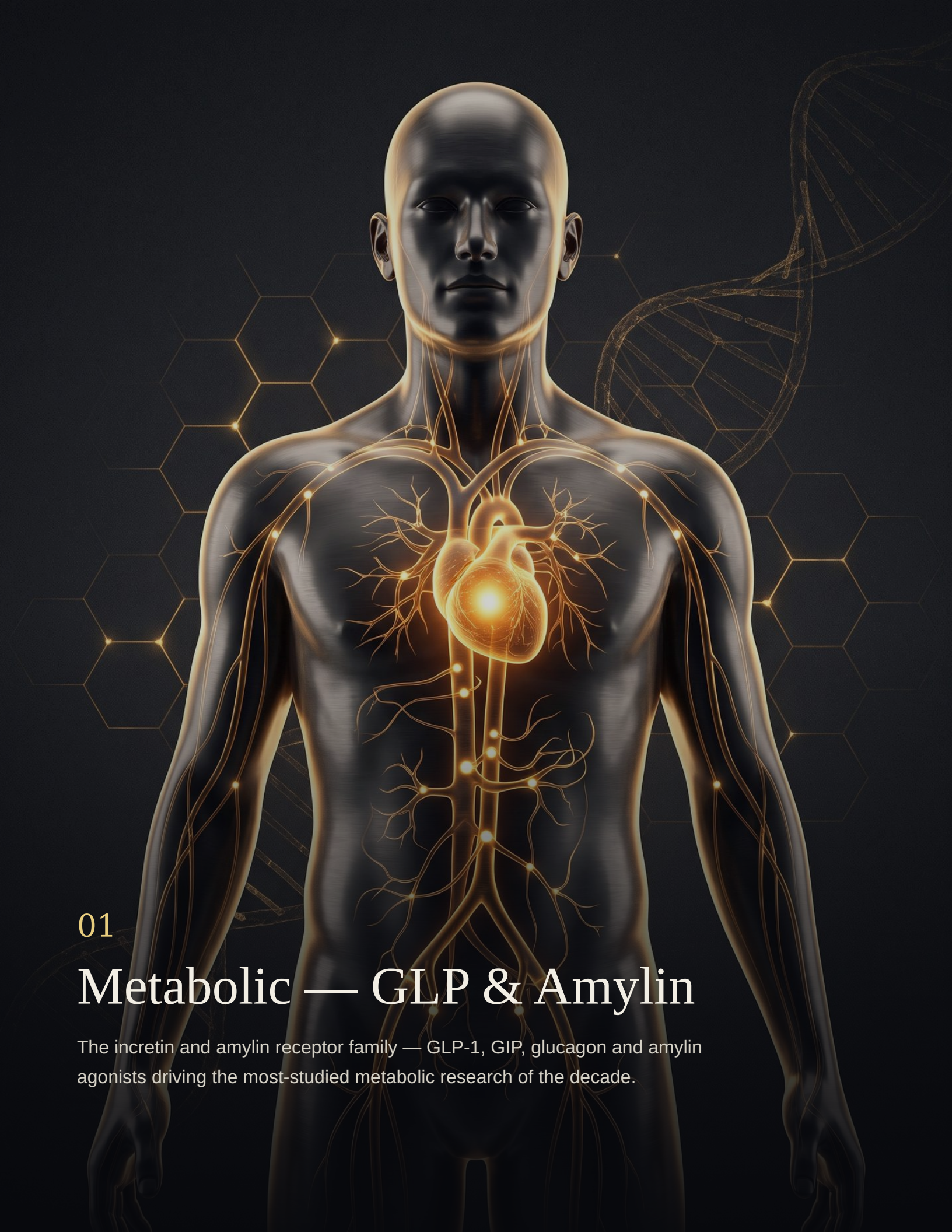
Understanding any research compound means answering three questions: **what is it** (its structure and origin), **how does it work** (its receptor target and signaling), and **what does the evidence actually show** (the depth and quality of the research behind it). Each entry is organized around those questions.

Handling & reconstitution standards

Research peptides are supplied as lyophilized powder for stability, then reconstituted with bacteriostatic water before laboratory use; a reconstituted vial remains usable for roughly 28 days when refrigerated. Lyophilized material is stored at -20°C , protected from light. Purity is verified by HPLC and mass spectrometry, with a Certificate of Analysis available per batch.

How this guide is organized

The compounds are grouped into five research classes: **Metabolic (GLP & Amylin)**, **Healing & Tissue Repair**, **Growth Hormone & IGF-1 Axis**, **Longevity & Mitochondrial**, and **Cosmetic, Sexual & Specialty**.



01

Metabolic — GLP & Amylin

The incretin and amylin receptor family — GLP-1, GIP, glucagon and amylin agonists driving the most-studied metabolic research of the decade.



Retatrutide

LY3437943 · triple agonist

Retatrutide is a synthetic 39–amino-acid peptide investigated as a first-in-class *triple* incretin agonist. Where earlier metabolic peptides engage one or two receptors, retatrutide is engineered to activate three simultaneously, making it one of the most studied experimental metabolic compounds of the decade.

What does Retatrutide do?

Retatrutide (developmental designation LY3437943) represents the current frontier of incretin research: it is a *triple agonist*, engaging three receptors simultaneously — the GLP-1 receptor, the GIP receptor, and the glucagon receptor. This three-way mechanism distinguishes it from every earlier compound in the class and is the primary focus of scientific interest.

Each receptor contributes a distinct effect. GLP-1 receptor activation — shared with semaglutide — is studied for reducing appetite through central signaling in the hypothalamus and brainstem, slowing gastric emptying, and enhancing glucose-dependent insulin secretion. GIP receptor activation, shared with tirzepatide, is examined for potentiating insulin response and influencing how fat tissue handles nutrients. The glucagon receptor component is what makes retatrutide unique: whereas GLP-1 and GIP primarily reduce energy *intake*, glucagon receptor activation is investigated for increasing energy *expenditure* — stimulating hepatic fat oxidation, promoting thermogenesis, and influencing liver-fat metabolism.

Researchers describe this as a "push–pull" system: the GLP-1 and GIP arms lower caloric intake while the glucagon arm raises caloric output. In preclinical and early clinical research this combination has been associated with the largest metabolic effects observed in the incretin class, drawing particular attention in models of metabolic dysfunction–associated steatotic liver disease (MASLD), where glucagon-driven reduction in liver fat is a key research endpoint. The scientific rationale traces to work such as Coskun and colleagues (2022), who published the preclinical development of LY3437943 and established the case for triple agonism over dual- and single-receptor approaches.

Context matters. Retatrutide is investigational — not FDA-approved and still in clinical development — and its human evidence base, while growing rapidly and attracting significant attention, is far younger than semaglutide's or tirzepatide's. Its profile is best understood as high-potential and mechanistically novel, but still maturing. For metabolic researchers, its appeal is precisely that it adds an entirely new dimension — energy expenditure — to a class previously defined by appetite suppression alone, making it a frequent reference point in studies of obesity, insulin sensitivity, and hepatic fat metabolism.

Mechanism & receptor target

In receptor-pharmacology studies it acts as an agonist at the glucagon-like peptide-1 (GLP-1), glucose-dependent insulintropic polypeptide (GIP) and glucagon (GCGR) receptors. This combined engagement is the

focus of research into how simultaneous incretin and glucagon signaling influences energy-balance pathways in model systems.

In the research

Preclinical and early-phase literature examines retatrutide's receptor binding, signaling kinetics and metabolic-pathway modulation. In a laboratory setting it is used as a reference tool for studying multi-receptor incretin biology.

Reference specifications

Sequence	39-residue acylated peptide
Molecular formula	C ₂₂₁ H ₃₄₂ N ₄₆ O ₆₈
Molecular weight	4731.33 g/mol
CAS number	2381089-83-2
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

Retatrutide is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 01 · METABOLIC — GLP & AMYLIN

Tirzepatide

LY3298176 · dual agonist

Tirzepatide is a 39–amino-acid synthetic peptide and the archetype of the “twincretin” class — a single molecule designed to engage two incretin receptors at once. It is among the most extensively characterized metabolic research peptides available.

What does Tirzepatide do?

Tirzepatide is a synthetic dual agonist that activates two incretin receptors at once — the GIP receptor and the GLP-1 receptor. This combination is the defining feature of the compound and the reason it became one of the most-studied molecules in modern metabolic research. By engaging two pathways that independently regulate appetite, insulin secretion, and energy balance, tirzepatide is investigated as a mechanistic step beyond single-pathway GLP-1 agonists such as semaglutide.

The GLP-1 arm is the more familiar of the two. In research models it is associated with reduced appetite through central hypothalamic and brainstem signaling, slowed gastric emptying, and glucose-dependent insulin secretion that improves glycemic control without the hypoglycemia risk seen with older agents. The GIP arm is what differentiates tirzepatide. GIP is studied for potentiating the insulin response and for distinct effects on adipose tissue and lipid handling — effects that may complement, rather than simply duplicate, GLP-1 activity.

A notable point in the literature is that tirzepatide is an *imbalanced* agonist: it shows relatively stronger activity at the GIP receptor than at the GLP-1 receptor. Researchers have proposed that this receptor bias may partly explain the differentiated outcomes observed in head-to-head studies against semaglutide, where dual agonism has been associated with larger metabolic and weight-related endpoints. The compound's evidence base is anchored to extensive clinical trial programs, making it one of the best-characterized dual incretins in the field.

In practical research interpretation, tirzepatide is evaluated less by short-term change and more by the stability of its metabolic signals over time — sustained directional shifts in body weight, improvements in insulin sensitivity, and consistency of appetite regulation across models. It is best understood as a metabolic-behavior support framework studied over weeks and months rather than a rapid single-effect compound.

For researchers, tirzepatide occupies an important middle position in the incretin class: more mechanistically complex than a single GLP-1 agonist, but with a deeper and more mature evidence base than the newer triple agonists. That makes it a common reference point in comparative studies of appetite, glucose metabolism, and body composition.

Mechanism & receptor target

It acts as a dual agonist at the GIP and GLP-1 receptors. Research focuses on how balanced co-activation of these two pathways affects insulinotropic signaling and downstream metabolic cascades relative to single-receptor GLP-1 agonists.

In the research

The compound is used in receptor-signaling and comparative-pharmacology research to benchmark dual-versus mono-agonist activity. Its long fatty-acid modification, which extends circulating half-life in models, is itself a subject of study.

Reference specifications

Sequence	39-residue acylated peptide
Molecular formula	C ₂₂₅ H ₃₄₈ N ₄₈ O ₆₈
Molecular weight	4813.53 g/mol
CAS number	2023788-19-2
Form	Lyophilized powder

For research purposes, semaglutide's value lies in that combination of a clean single mechanism and an unmatched depth of clinical characterization within this class. It is best interpreted as the well-mapped foundation of incretin science — the compound whose behavior is most thoroughly understood and against which the metabolic effects of everything newer are benchmarked.

Mechanism & receptor target

It binds and activates the GLP-1 receptor, a class-B G-protein-coupled receptor. Studies examine its receptor affinity, cAMP signaling and the structural modifications (an Aib substitution and a C18 fatty-diacid chain) that give it a long half-life in model systems.

In the research

Semaglutide is used in laboratory research as a well-characterized GLP-1R reference agonist for receptor assays and comparative signaling work.

Reference specifications

Sequence	31-residue acylated analog
Molecular formula	C ₁₈₇ H ₂₉₁ N ₄₅ O ₅₉
Molecular weight	4113.58 g/mol
CAS number	910463-68-2
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

Semaglutide is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



02

Healing & Tissue Repair

Regenerative and cytoprotection peptides — the tissue-repair compounds researchers investigate first, from BPC-157 and TB-500 to copper and α -MSH-derived signals.



BPC-157

Body Protection Compound-157

BPC-157 is a stable synthetic pentadecapeptide corresponding to a partial sequence of a body-protection compound identified in gastric juice. It is one of the most widely investigated peptides in regenerative and cytoprotection research.

What does BPC-157 do?

BPC-157, a synthetic 15–amino-acid sequence derived from a protein identified in gastric juice, is one of the most extensively studied peptides in tissue-repair and regenerative research. In the laboratory, its defining feature is stability — it remains intact under gastric conditions that would degrade most peptides — which is part of why researchers have returned to it across such a wide range of experimental models.

The central research theme around BPC-157 is angiogenesis: the formation of new blood vessels. In cell and animal studies, BPC-157 has been observed to promote vascular network growth at sites of injury, the process that supplies damaged tissue with oxygen and nutrients during repair. Investigators have connected this to upregulation of vascular endothelial growth factor (VEGF) and to signaling through the nitric oxide (NO) system — two pathways central to how new vessels form and how blood flow is regulated.

Beyond vascular effects, a large body of preclinical work has examined BPC-157 in connective tissue. Studies in cultured tendon and ligament cells report increased cell migration and survival alongside activation of the FAK–paxillin pathway, which governs how cells attach and move — mechanisms relevant to how tendon, ligament, and muscle reorganize after damage. Separate rodent studies have investigated BPC-157 in models of muscle injury, bone healing, and nerve regeneration, consistently framing it as a broadly cytoprotective agent rather than one acting on a single tissue. Reflecting its gastric origin, it has also been heavily studied in animal models of ulceration and gut-lining injury, where researchers have proposed interactions with the gut–brain axis and dopaminergic signaling.

It's important to frame this evidence honestly. The overwhelming majority of BPC-157 data comes from in-vitro and animal research — much of it from a small number of research groups — and human clinical data remains early-stage. BPC-157 is not an approved therapeutic in any jurisdiction. Its profile is best described as *broad but shallow*: consistent tissue-protective signals across an unusually wide range of injury models, but a depth of rigorous human evidence that does not yet match that breadth. For researchers, that combination of stability and multi-tissue activity is precisely what makes it a valuable reference compound in wound-healing and angiogenesis studies.

Mechanism & receptor target

Preclinical literature explores its interaction with angiogenic and growth-factor pathways, including effects on VEGFR2 signaling and nitric-oxide systems, in models of tissue integrity. Its notable stability in aqueous and acidic conditions makes it a practical laboratory research tool.

In the research

Animal-model studies examine roles in vascular, connective-tissue and gastrointestinal research contexts. It is handled strictly as a reference research material.

Reference specifications

Sequence	Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val
Molecular formula	C ₆₂ H ₉₈ N ₁₆ O ₂₂
Molecular weight	1419.54 g/mol
CAS number	137525-51-0
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

BPC-157 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 02 · HEALING & TISSUE REPAIR

TB-500

Thymosin β4 active region

TB-500 is a synthetic peptide corresponding to the biologically active, actin-binding region of Thymosin β4, a naturally occurring regulatory protein. It is investigated alongside BPC-157 as a core tissue-repair research compound.

What does TB-500 do?

TB-500 is a synthetic peptide corresponding to the active region of Thymosin Beta-4 (TB4), one of the most abundant and well-studied members of the beta-thymosin family. Specifically, it replicates the 17–23 amino-acid sequence (LKKTETQ) that mediates much of TB4's biological activity. Because the parent protein plays fundamental roles in cellular architecture and tissue repair, TB-500 is investigated primarily through a recovery and regeneration lens.

The mechanistic core of TB-500 is actin regulation. It sequesters G-actin monomers — the building blocks of the cell's cytoskeleton — and in doing so influences the assembly of new actin filaments that drive cell migration. This matters because directed cell migration toward sites of injury is a foundational step in nearly all tissue repair. By modulating this process, TB-500 has been studied for its ability to help cells move into and reorganize damaged tissue across multiple organ systems.

Preclinical research has examined several distinct effects. Wound-closure studies report accelerated epithelialization and granulation tissue formation in dermal, corneal, and cardiac models, sometimes with improved tensile strength in healed tissue. Cardiac research has investigated improved ventricular function and reduced scar size in models of ischemic heart injury, an area where the first human cardiac data has drawn particular interest. Anti-inflammatory activity — downregulation of pro-inflammatory cytokines and modulation of immune-cell infiltration at injury sites — is a recurring theme, and separate work has looked at angiogenesis and even hair-follicle activation via stem-cell migration in animal models.

As with many repair peptides, honest framing is essential. TB-500 shows a notably *broad* range of tissue responses across the literature, but the depth of rigorous human evidence remains limited; most of the data is animal-derived. It is best interpreted as a support-context compound for recovery rhythm across tissue types rather than a guaranteed structural-repair tool.

For researchers, TB-500's appeal is its position at the center of cytoskeletal biology: because actin dynamics underlie migration, healing, and remodeling in so many tissues, it serves as a useful reference peptide in wound-repair, cardiac, and angiogenesis research — and it is frequently studied alongside BPC-157, with which it shares complementary repair mechanisms.

Mechanism & receptor target

Research focuses on its interaction with G-actin and the promotion of cell migration and angiogenesis in model systems. The parent protein, Thymosin β 4, is a well-characterized actin-sequestering peptide, and TB-500 is studied as its accessible research fragment.

In the research

Used in laboratory studies of cell motility, vascular formation and connective-tissue biology. Frequently combined with BPC-157 in blends (see Wolverine, GLOW, KLOW).

Reference specifications

Sequence	Thymosin β 4 active fragment (e.g., Ac-LKKTETQ)
Molecular formula	Fragment of T β 4 (C ₂₁₂ H ₃₅₀ N ₅₆ O ₇₈ S)
Molecular weight	varies by fragment
CAS number	77591-33-4 (T β 4)
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

TB-500 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



GHK-Cu

copper tripeptide-1

GHK-Cu is the tripeptide glycyl-L-histidyl-L-lysine bound to a copper(II) ion. First isolated from human plasma, it is a heavily studied research compound in tissue-remodeling and cosmetic science.

What does GHK-Cu do?

GHK-Cu is a naturally occurring copper-binding tripeptide (glycyl-L-histidyl-L-lysine complexed with copper) that the body produces endogenously, with levels that decline notably with age. This age-related decline is part of what made GHK-Cu a durable focus of skin, tissue-remodeling, and longevity research. Its activity clusters around a few well-defined themes, and it is one of the better-characterized peptides in this guide at the cellular level.

The most reproducible finding in the GHK-Cu literature is its effect on the extracellular matrix. In fibroblast culture studies, GHK-Cu consistently upregulates the production of collagen types I, III, and IV alongside elastin — the structural proteins that give tissue its density, firmness, and elasticity. This is the cell-level foundation for its extensive study in skin research, where topical GHK-Cu preparations have been examined for measurable improvements in skin density and reductions in fine-line depth over multi-week periods. This topical and skin-focused evidence base is meaningfully stronger than the injectable tissue-repair evidence.

Beyond structural proteins, GHK-Cu functions as a biological signal molecule and copper transport vehicle. It binds copper(II) ions with exceptionally high affinity and delivers copper to enzymes such as superoxide dismutase (SOD), supporting antioxidant defense and reactive-oxygen-species clearance in cell and animal models. Gene-expression research is a particularly active area: array data show GHK-Cu can modulate large numbers of genes associated with tissue remodeling, inflammation, and aging, including downregulation of pro-inflammatory pathways — findings that have been functionally examined in models of lung and liver inflammation.

There is also emerging interest in neuroprotective activity, though this area is earlier-stage. Across all of these themes, the critical distinction to keep in mind is that GHK-Cu's skin and topical evidence base is considerably more developed than its injectable tissue-repair evidence, most of which is animal-derived and has not yet reached clinical-trial standard.

For researchers, GHK-Cu is valuable precisely because it sits at the intersection of copper biology, matrix remodeling, and gene-expression modulation — a rare combination that makes it a reference compound across skin science, wound healing, and cellular-aging studies.

Mechanism & receptor target

Research explores its high-affinity copper binding and its influence on extracellular-matrix genes, including collagen and metalloproteinase expression, in fibroblast models. The copper complex is considered the biologically relevant research form.

In the research

Used in dermatological, wound-model and matrix-biology research. Appears in cosmetic repair blends (see GLOW, KLOW).

Reference specifications

Sequence	Gly-His-Lys · Cu ²⁺
Molecular formula	C ₁₄ H ₂₄ N ₆ O ₄ · Cu
Molecular weight	≈403.9 g/mol (complex)
CAS number	49557-75-7
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

GHK-Cu is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 02 · HEALING & TISSUE REPAIR

Wolverine

BPC-157 + TB-500 blend

“Wolverine” is a research blend that combines BPC-157 and TB-500 — the two most-studied tissue-repair peptides — in a single preparation for parallel investigation.

What does Wolverine do?

Wolverine is a combination formulation pairing two of the most-studied repair peptides — BPC-157 and TB-500 (a Thymosin Beta-4 fragment) — in a single blend. The research rationale for combining them is complementary mechanisms: the two peptides are studied for overlapping but distinct contributions to tissue repair, and the blend is investigated as a way to engage multiple arms of the healing process at once.

BPC-157 contributes its well-documented angiogenic and cytoprotective profile: promotion of new blood-vessel formation (via VEGF and nitric-oxide signaling), activation of connective-tissue repair pathways (FAK–paxillin), and broad protective effects across muscle, tendon, ligament, nerve, and gut-injury models. TB-500 contributes actin-regulation and cell-migration activity: by sequestering G-actin and influencing cytoskeletal assembly, it is studied for driving the directed movement of cells into damaged tissue — a foundational step in wound closure and remodeling.

The theoretical logic of the combination is that these mechanisms address different stages and dimensions of repair. Angiogenesis (BPC-157) rebuilds the blood supply that healing tissue requires, while cell migration and cytoskeletal remodeling (TB-500) drive the physical reorganization of tissue at the injury site. Add TB-500's anti-inflammatory activity and BPC-157's protective signaling, and researchers describe the pairing as engaging repair from several angles simultaneously rather than through a single pathway.

Honest framing is important here. Because Wolverine is a blend, its evidence base is essentially the sum of the two individual peptides' research — both predominantly preclinical, animal-derived, and early-stage in humans. There is limited controlled research on the *combination* specifically, as opposed to each component alone, so any synergy is largely inferred from the complementary mechanisms rather than demonstrated head-to-head. Neither component is an approved therapeutic.

For researchers, the appeal of the blend is efficiency of study design: it allows the two leading repair peptides to be examined together as a combined recovery-support context. It is best interpreted as a multi-mechanism reference formulation for tissue-repair research, not as a compound with a distinct mechanism of its own beyond those of its two constituents.

Mechanism & receptor target

It brings together BPC-157's angiogenic / cytoprotection pathways and TB-500's actin-binding, cell-migration activity. The research rationale is to study whether the two complementary mechanisms are informative when examined together.

Reference specifications

Sequence	Two-peptide research blend
Molecular formula	Blend — see BPC-157 & TB-500
Molecular weight	—
CAS number	137525-51-0 / 77591-33-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

Wolverine is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 02 · HEALING & TISSUE REPAIR

GLOW

GHK-Cu + BPC-157 + TB-500

“GLOW” is a research blend of GHK-Cu, BPC-157 and TB-500, prepared to study matrix-remodeling and tissue-repair signaling together — a combination frequently referenced in cosmetic and regenerative research.

What does GLOW do?

GLOW is a combination formulation blending three well-studied peptides — BPC-157, GHK-Cu, and TB-500 — chosen for complementary activity across tissue-repair and skin/aesthetic research. The name reflects its research positioning: a multi-peptide blend investigated primarily for effects on skin quality, wound healing, and connective-tissue support, drawing on three distinct but overlapping mechanisms.

Each component brings a different research profile. GHK-Cu, the copper-binding tripeptide, is the aesthetic anchor of the blend: it is the most reproducible driver of collagen and elastin synthesis in fibroblast studies, with the strongest topical/skin evidence base of any peptide here, plus antioxidant (copper-to-SOD delivery) and gene-expression-modulating activity. BPC-157 contributes its angiogenic and cytoprotective profile — new blood-vessel formation via VEGF and nitric-oxide signaling, and broad protective effects across connective tissue. TB-500 contributes actin-regulation and cell-migration activity, driving the movement of cells into tissue during repair, plus anti-inflammatory signaling.

The research rationale for combining them is that skin and tissue quality depend on several processes at once: structural-protein production (GHK-Cu), a healthy blood supply (BPC-157), and cellular migration and remodeling (TB-500). By pairing a matrix-building peptide with two repair-and-vascular peptides, GLOW is studied as a blend that engages the extracellular matrix, angiogenesis, and cell-migration arms of tissue biology simultaneously — with a particular tilt toward skin and cosmetic-research endpoints given GHK-Cu's dominance in that area.

As with all blends, the evidence should be framed honestly. GLOW's research base is essentially the combined profiles of its three components, each predominantly preclinical (with GHK-Cu carrying the most human-relevant topical data). Controlled research on the specific three-peptide combination is limited, so synergy is inferred from complementary mechanisms rather than demonstrated directly, and none of the components is an approved therapeutic.

For researchers, GLOW is best interpreted as a matrix-and-repair reference blend — a way to study collagen/elastin support, angiogenesis, and tissue remodeling together — with its center of gravity in skin and connective-tissue research thanks to the GHK-Cu component.

Mechanism & receptor target

It brings together GHK-Cu's extracellular-matrix and copper-transport activity, BPC-157's angiogenic pathways and TB-500's actin-binding, cell-migration activity.

In the research

As a blend it has no single formula; each component is independently characterized and handled as research material.

Reference specifications

Sequence	Three-peptide research blend
Molecular formula	Blend — see components
Molecular weight	—
CAS number	49557-75-7 / 137525-51-0 / 77591-33-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

GLOW is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 02 · HEALING & TISSUE REPAIR

KLOW

GHK-Cu + KPV + BPC-157 + TB-500

“KLOW” is a four-component research blend of GHK-Cu, KPV, BPC-157 and TB-500. It extends the GLOW combination by adding KPV, an α-MSH-derived tripeptide studied in inflammatory-signaling research.

What does KLOW do?

KLOW is a four-peptide combination formulation building on the same repair-and-aesthetic foundation as GLOW — GHK-Cu, BPC-157, and TB-500 — with the addition of a fourth component, KPV. That addition is the defining feature of KLOW: it layers a dedicated anti-inflammatory peptide on top of the matrix-building, angiogenic, and cell-migration mechanisms of the other three.

The three shared components carry the profiles described elsewhere in this guide. GHK-Cu drives collagen and elastin synthesis and antioxidant/gene-expression activity, anchoring the blend's skin and connective-

tissue relevance. BPC-157 contributes angiogenesis (VEGF and nitric-oxide signaling) and broad cytoprotection. TB-500 contributes actin-regulated cell migration and anti-inflammatory signaling. Together these engage the extracellular matrix, blood supply, and cellular-remodeling arms of tissue repair.

KPV is what distinguishes KLOW. KPV is a small tripeptide (lysine-proline-valine) derived from the C-terminal region of α -MSH, and it is studied specifically for anti-inflammatory activity. Research examines its ability to downregulate pro-inflammatory signaling — including NF- κ B pathway activity — and to reduce inflammatory responses at both systemic and local tissue levels, with particular attention in models of gut and mucosal inflammation. By adding KPV, the KLOW blend is positioned as a repair formulation with a stronger explicit anti-inflammatory dimension than GLOW: the research logic is that resolving inflammation supports the environment in which the matrix-building and repair components can act.

The framing caveats mirror the other blends. KLOW's evidence base is the combined preclinical profiles of its four constituents, each studied largely in cell and animal models. There is little controlled research on the specific four-peptide combination, so its integrated activity is inferred from the complementary mechanisms rather than demonstrated head-to-head, and none of the components is an approved therapeutic.

For researchers, KLOW is best interpreted as a repair-plus-anti-inflammation reference blend — the most "complete" of the multi-peptide combinations in this guide in terms of the number of tissue-biology arms it engages: matrix synthesis, angiogenesis, cell migration, and inflammation resolution together.

Mechanism & receptor target

It combines matrix-remodeling (GHK-Cu), inflammatory-pathway modulation (KPV), angiogenic signaling (BPC-157) and actin-binding cell migration (TB-500) for parallel study.

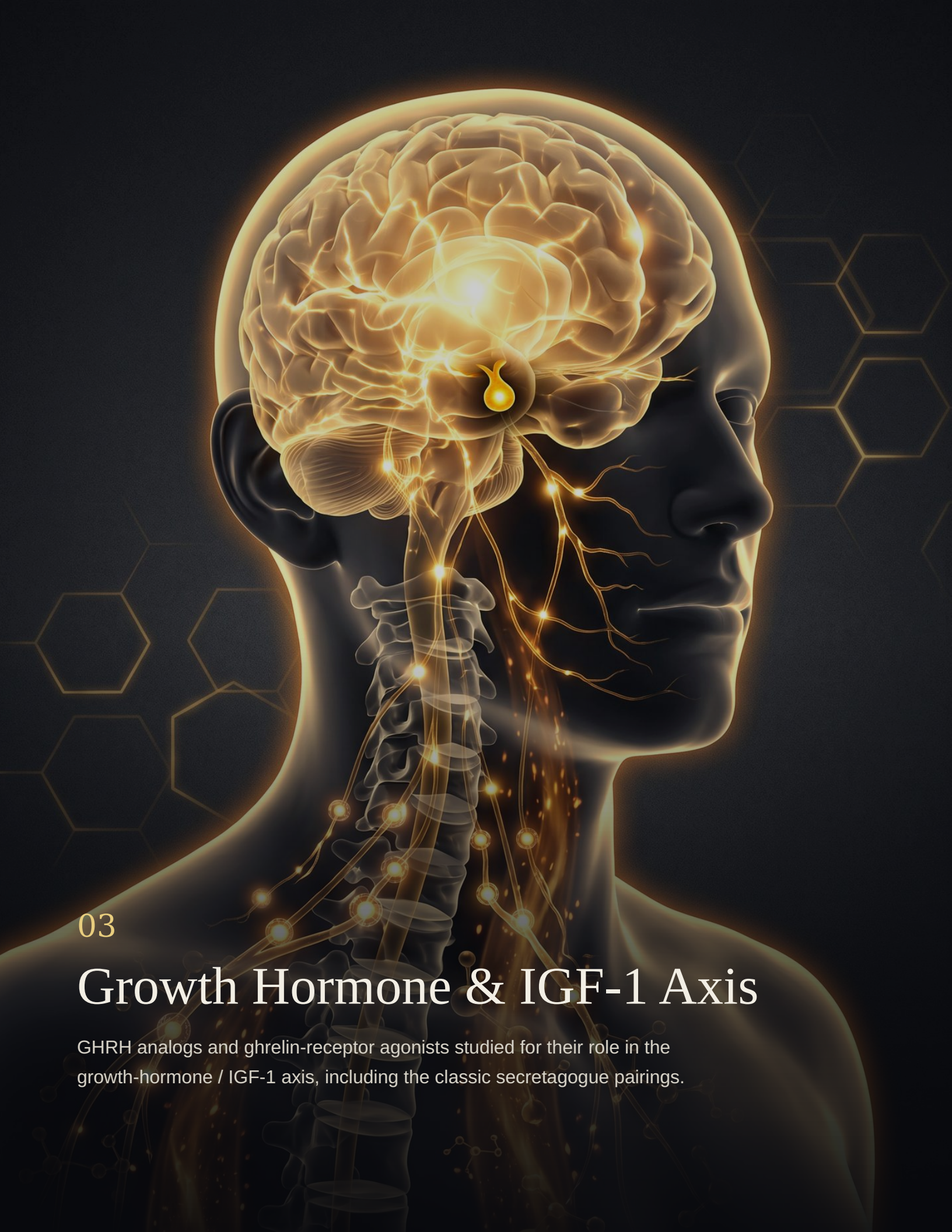
In the research

As a blend it has no single formula; each component is independently characterized and handled as research material.

Reference specifications

Sequence	Four-peptide research blend
Molecular formula	Blend — see components
Molecular weight	—
CAS number	49557-75-7 / 67727-97-3 / 137525-51-0 / 77591-33-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

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03

Growth Hormone & IGF-1 Axis

GHRH analogs and ghrelin-receptor agonists studied for their role in the growth-hormone / IGF-1 axis, including the classic secretagogue pairings.



Ipamorelin

selective GHRP

Ipamorelin is a synthetic pentapeptide and one of the most selective growth-hormone-releasing peptides (GHRPs) in research use. It is valued as a research tool for its selectivity relative to earlier GHRPs.

What does Ipamorelin do?

Ipamorelin is a synthetic growth-hormone secretagogue that acts on the ghrelin receptor (GHS-R1a) on somatotroph cells of the anterior pituitary, triggering release of the body's own growth hormone (GH). Rather than introducing exogenous GH, it is studied for prompting pulsatile, endogenous GH release — a pattern that more closely resembles natural secretion — with downstream elevation of insulin-like growth factor 1 (IGF-1).

Its defining research characteristic is selectivity. The foundational work (Raun and colleagues, 1998) demonstrated that ipamorelin produces dose-dependent GH release comparable to earlier secretagogues such as GHRP-6, but without meaningfully affecting cortisol, ACTH, prolactin, or aldosterone. This selectivity distinguished it from every previously characterized GH secretagogue and is the primary reason it became a preferred reference compound in the class: it isolates the GH-releasing signal from the broader hormonal "noise" that complicated earlier peptides.

Mechanistically, ipamorelin works through the ghrelin/GHS-R pathway, which is pharmacologically distinct from the GHRH-receptor pathway used by compounds such as somatostatin, CJC-1295, and tesamorelin. This distinction is what makes dual-pathway stimulation possible: because ipamorelin and GHRH analogs act through different receptors, they are frequently studied in combination, where their effects on GH release appear complementary and, in some models, synergistic.

Preclinical research has examined several downstream effects tied to GH/IGF-1 elevation. Studies report longitudinal bone growth in juvenile animal models, counteraction of glucocorticoid-induced bone loss in adults, and increases in bone mineral content — evidence connecting GH-secretagogue signaling to tissue-level anabolic and protective outcomes. Because the GH axis influences body composition, recovery, and metabolic function broadly, ipamorelin is studied across a range of endpoints rather than a single one.

For researchers, ipamorelin's value is that combination of a clean, selective mechanism and a distinct receptor pathway. It serves as a well-behaved model compound for studying pulsatile GH release without confounding hormonal effects, and as a natural partner in dual-pathway GH-axis research alongside GHRH analogs.

Mechanism & receptor target

It acts as an agonist at the ghrelin / growth-hormone-secretagogue receptor (GHS-R1a). Research highlights its selectivity — minimal impact on cortisol or prolactin pathways in models — which makes it a clean probe of GHS-R signaling.

In the research

Studied in the GH-axis research context and commonly combined with CJC-1295 to examine GHRH + ghrelin-receptor co-stimulation.

Reference specifications

Sequence	Aib-His-D-2-Nal-D-Phe-Lys-NH ₂
Molecular formula	C ₃₈ H ₄₉ N ₉ O ₅
Molecular weight	711.85 g/mol
CAS number	170851-70-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

Ipamorelin is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 03 · GROWTH HORMONE & IGF-1 AXIS

CJC-1295 / Ipamorelin

secretagogue blend

This is a research blend of CJC-1295 and ipamorelin — the most-studied secretagogue combination — prepared together to investigate two complementary arms of growth-hormone-axis signaling.

What does CJC-1295 / Ipamorelin do?

The CJC-1295 / Ipamorelin combination pairs two growth-hormone-axis peptides that act through *different* receptors, and the entire rationale for the blend rests on that difference. CJC-1295 is a growth-hormone-releasing hormone (GHRH) analog; ipamorelin is a ghrelin-receptor (GHS-R1a) agonist. Because they stimulate GH release through two independent pathways, they are studied together as a dual-pathway approach that engages the pituitary from two directions at once.

CJC-1295 mimics GHRH, the body's natural signal instructing the pituitary to produce and release GH. Its structural modifications extend its activity relative to native GHRH, providing a more sustained release signal. Ipamorelin, acting on the ghrelin receptor, provides a distinct, selective GH-releasing pulse without meaningfully disturbing cortisol or prolactin. The research logic is that a GHRH analog raises the baseline GH-release drive while a ghrelin agonist amplifies the pulse — two complementary levers on the same output.

In the literature, combining a GHRH analog with a ghrelin-receptor agonist is a well-established experimental strategy for maximizing GH release, and the two mechanisms are frequently described as synergistic rather than merely additive. The downstream endpoint of interest is elevated GH and, consequently, IGF-1, which connects the combination to research on recovery, body composition, bone, and metabolic function — all areas influenced by the GH/IGF-1 axis.

As with all blends, framing should be careful. The evidence base is largely the combination of the two individual peptides' preclinical profiles, and controlled human data on the specific pairing is limited. Neither component is an approved therapeutic, and the "synergy" is grounded in the complementary receptor mechanisms and animal-model data rather than large clinical trials.

For researchers, the CJC-1295 / Ipamorelin blend is a canonical example of rational dual-pathway design: it allows the GHRH and ghrelin arms of GH regulation to be studied together, making it a common reference formulation in growth-hormone-axis and secretagogue research.

Mechanism & receptor target

CJC-1295 engages the GHRH receptor while ipamorelin engages the ghrelin / GHS receptor. The research rationale is to study how simultaneous stimulation of both pathways compares with either alone in model systems.

In the research

As a blend it has no single formula; each component is independently characterized. Handled strictly as research material.

Reference specifications

Sequence	Two-peptide research blend
Molecular formula	Blend — see components
Molecular weight	—
CAS number	863288-34-0 / 170851-70-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

CJC-1295 / Ipamorelin is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



Tesamorelin

GRF analog

Tesamorelin is a synthetic analog of the full 44–amino-acid human growth-hormone-releasing factor, stabilized by an N-terminal hexenoyl modification. It is a well-characterized GHRH research peptide.

What does Tesamorelin do?

Tesamorelin is a stabilized analog of growth-hormone-releasing hormone (GHRH) — a modified form of human GHRH(1–44) with a trans-3-hexenoic acid group that improves its stability. Among the compounds in this guide, it holds a distinctive position: it is one of the few with formal regulatory approval (for HIV-associated lipodystrophy, 2010), which gives it a more developed human evidence base than most research peptides.

Its mechanism is to stimulate the anterior pituitary to release the body's own growth hormone (GH), which in turn drives IGF-1 production and downstream metabolic effects. Crucially, because tesamorelin acts through the GHRH pathway rather than supplying exogenous GH, it is studied for preserving the natural pulsatile pattern of GH secretion and maintaining hypothalamic–pituitary feedback regulation — a more physiological profile than direct GH administration.

The most robust research findings concern visceral fat. The landmark Stanley and colleagues (2014) trial demonstrated significant reductions in both visceral adipose tissue and liver fat in patients with abdominal fat accumulation, and subsequent work (Stanley 2019) showed reductions in hepatic fat fraction and improvements in liver-fibrosis markers in the context of NAFLD. More recent meta-analyses pooling multiple randomized trials have confirmed reductions in visceral adipose tissue, trunk fat, and waist circumference, often with concurrent improvements in lean body mass — an unusually well-replicated body-composition profile for a peptide.

Beyond fat, research has examined metabolic endpoints (improved triglyceride levels, reduced inflammatory markers, favorable adipose-tissue quality) and, intriguingly, cognitive function: work using GHRH-analog protocols has reported improvements in cognition in both healthy older adults and adults with mild cognitive impairment, broadening the compound's research relevance beyond body composition alone.

For researchers, tesamorelin is valuable precisely because it combines a clean GHRH mechanism with a genuine clinical evidence base — a rarity in this space. It is best understood as the most clinically validated GHRH analog available for research, and a frequent reference point in studies of visceral fat, the GH/IGF-1 axis, and metabolic health.

Mechanism & receptor target

It binds and activates the GHRH receptor, and research examines how its stabilizing modification affects potency and half-life relative to native GRF in model systems.

In the research

Used in endocrine research on the somatotropic axis as a defined, full-length GRF-analog reference compound.

Reference specifications

Sequence	Hexenoyl-GRF(1-44) analog
Molecular formula	C ₂₂₁ H ₃₆₆ N ₇₂ O ₆₇ S
Molecular weight	5135.86 g/mol
CAS number	218949-48-5
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

Tesamorelin is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 03 · GROWTH HORMONE & IGF-1 AXIS

IGF-1 LR3

Long R3 IGF-1

IGF-1 LR3 (Long R3 IGF-1) is an 83-amino-acid analog of insulin-like growth factor 1, carrying an Arg³ substitution and a 13-residue N-terminal extension that reduce its binding to IGF-binding proteins and extend its activity. It is a core reference material in growth-factor and cell-proliferation research.

What does IGF-1 LR3 do?

IGF-1 LR3 (Long R3 IGF-1) is an engineered, long-acting analog of insulin-like growth factor 1 — an 83-amino-acid variant carrying an arginine substitution at position 3 and a 13-residue N-terminal extension. Those two modifications are the entire point of the molecule: they dramatically reduce its binding to IGF-binding proteins and extend how long it remains active, making it a far more potent and durable research tool than native IGF-1.

Mechanistically, IGF-1 LR3 acts on the IGF-1 receptor (IGF-1R), a receptor tyrosine kinase structurally related to the insulin receptor. Binding triggers receptor autophosphorylation and activation of two primary downstream cascades. The PI3K/Akt/mTOR pathway drives protein synthesis, cell survival (anti-apoptotic signaling), and glucose uptake — the principal anabolic axis relevant to muscle-hypertrophy research. The Ras/MAPK/ERK pathway promotes cell proliferation and differentiation, with important implications for both tissue growth and

proliferative risk. These pathways operate identically whether activated by native IGF-1 or the LR3 variant; the difference is duration and magnitude of exposure, not the signaling itself.

The reason the LR3 modification matters comes down to the IGFBP system. In normal physiology, roughly 98% of circulating IGF-1 is bound to one of six IGF-binding proteins and held in reserve. IGF-1 LR3's reduced affinity for these binding proteins allows it to remain free and bioactive far longer than native IGF-1, bypassing the regulatory checkpoints that normally limit IGF-1 activity. This is what makes it such a consistent and powerful stimulus in cell-culture and animal research.

Because IGF-1 signaling sits at the center of growth, proliferation, and metabolism, IGF-1 LR3 is studied across muscle-biology, tissue-growth, and cell-proliferation research. Investigators value it for its predictability: it delivers a strong, sustained IGF-1R signal that is straightforward to study, which is why it has become a standard reference growth factor in laboratory settings. Its potency also makes it a compound in which the proliferative dimension of IGF-1 signaling is taken seriously in research design.

Mechanism & receptor target

Research examines its activation of the IGF-1 receptor and downstream PI3K/Akt and MAPK signaling, and because it largely evades IGFBP sequestration its prolonged availability in cell- and tissue-culture systems.

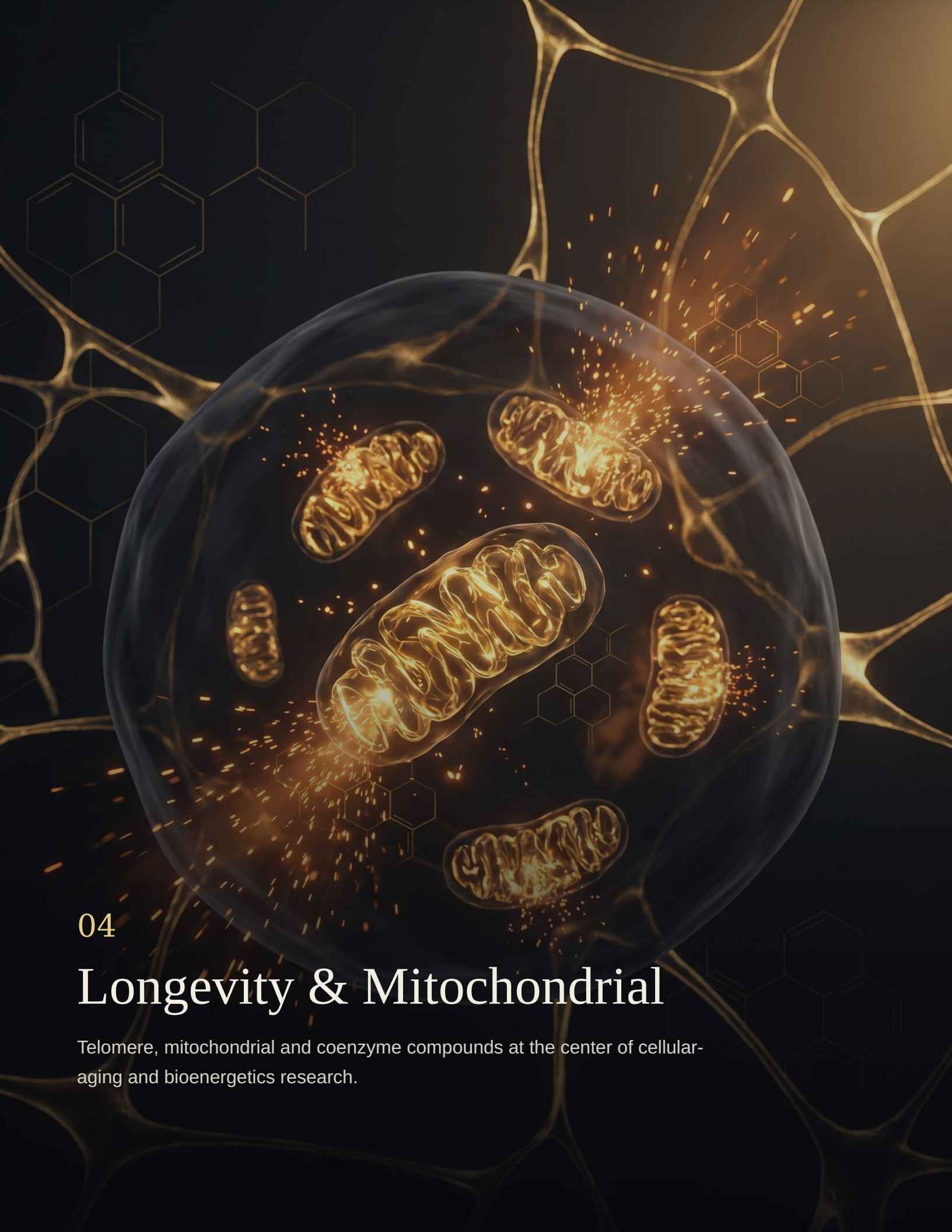
In the research

Used in cell-proliferation, muscle-biology and tissue-growth research as a long-acting IGF-1 reference analog.

Reference specifications

Sequence	83-residue IGF-1 analog (Arg, N-terminal extension)
Molecular formula	C ₂₀₈₉ H ₂₀₈₁ N ₂₀₈₂ O ₂₀₈₃ S ₂₀₈₇
Molecular weight	9111 g/mol
CAS number	946870-92-4
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

IGF-1 LR3 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.

The background is a dark, textured surface with a network of glowing, golden-yellow lines that resemble a cellular or molecular structure. In the center, a large, semi-transparent blue sphere represents a cell. Inside this sphere, several glowing, golden-yellow mitochondria with visible internal folds (cristae) are depicted. Small, golden-yellow sparks or particles are scattered around the mitochondria, suggesting energy production. In the upper left corner, there are several hexagonal chemical structures, some of which are interconnected, representing molecular compounds. The overall color palette is dominated by dark blues, blacks, and golden-yellow, creating a high-tech, scientific aesthetic.

04

Longevity & Mitochondrial

Telomere, mitochondrial and coenzyme compounds at the center of cellular-aging and bioenergetics research.



NAD⁺

nicotinamide adenine dinucleotide

NAD⁺ is not a peptide but a coenzyme — nicotinamide adenine dinucleotide — present in every living cell. It is included here as a core research material in cellular-energy and aging science.

What does NAD⁺ do?

NAD⁺ (nicotinamide adenine dinucleotide) is the one entry in this guide that is not a peptide but a coenzyme — a fundamental cellular cofactor present in every living cell. It is included as a core research material because it sits at the absolute center of cellular energy metabolism and has become one of the most heavily studied molecules in aging and bioenergetics science.

NAD⁺ has two broad research roles. The first is its classical function as an electron carrier in redox reactions: it cycles between its oxidized (NAD⁺) and reduced (NADH) forms to shuttle electrons through the metabolic pathways that generate cellular energy, including glycolysis and oxidative phosphorylation. Without adequate NAD⁺, the cell's capacity to extract energy from nutrients is fundamentally constrained, which is why NAD⁺ availability is treated as a limiting factor in bioenergetics research.

The second role — and the one driving most contemporary interest — is as a required substrate for two major families of signaling enzymes. Sirtuins, a class of NAD⁺-dependent deacetylases, are central to research on cellular stress resistance, metabolic regulation, and longevity; they cannot function without NAD⁺. PARP enzymes, involved in DNA-damage detection and repair, also consume NAD⁺. Because both systems draw on the same NAD⁺ pool, research explores how cellular NAD⁺ levels influence the balance between energy metabolism, stress signaling, and DNA repair — a balance that appears to shift with age, as NAD⁺ levels are observed to decline in many tissues over the lifespan.

This age-associated decline is the foundation of the "NAD⁺ and aging" research field. Investigators study whether maintaining or restoring cellular NAD⁺ influences sirtuin activity, mitochondrial function, and markers of cellular aging in model systems. NAD⁺ and its precursors have consequently become reference materials across metabolic, mitochondrial, and longevity research.

For researchers, NAD⁺ is foundational rather than exotic: it is the cofactor that so many other pathways depend on, which is precisely why understanding and manipulating its availability is central to modern cellular-aging, sirtuin-biology, and bioenergetics research. It is best interpreted as an essential reference cofactor rather than a compound with a single targeted mechanism.

Mechanism & receptor target

It functions as an electron carrier in redox reactions and as a required substrate for sirtuins and PARP enzymes. Research explores how cellular NAD⁺ availability influences these signaling and DNA-repair pathways in model systems.

In the research

Used broadly in bioenergetics, sirtuin-biology and cellular-aging research as a fundamental reference cofactor.

Reference specifications

Sequence	Dinucleotide coenzyme (not a peptide)
Molecular formula	$C_{21}H_{27}N_7O_{14}P_2$
Molecular weight	663.43 g/mol
CAS number	53-84-9
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

NAD+ is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 04 · LONGEVITY & MITOCHONDRIAL

MOTS-c

mitochondrial-derived peptide

MOTS-c is a 16–amino-acid peptide encoded within the mitochondrial 12S rRNA gene — one of the first mitochondrial-derived peptides shown to act systemically as a “mitokine.” It is a major focus of metabolic-longevity research.

What does MOTS-c do?

MOTS-c is a 16–amino-acid peptide with an unusual origin: it is encoded within the mitochondrial genome itself, in the 12S ribosomal RNA gene. That makes it one of the first identified mitochondrial-derived peptides shown to act as a systemic signaling molecule — a “mitokine” — and it has become a major focus of metabolic and longevity research.

The primary mechanism through which MOTS-c exerts its effects is activation of AMPK (AMP-activated protein kinase), the cell's master energy sensor. AMPK activation initiates a cascade of metabolic responses: increased glucose uptake, enhanced fatty-acid oxidation, improved mitochondrial function, and inhibition of the folate–methionine one-carbon cycle. In the original discovery work (Lee and colleagues), MOTS-c was shown to inhibit the folate cycle by targeting AICAR, itself a well-known AMPK activator in exercise-physiology research — a finding that led investigators to describe MOTS-c as converging on the same energy-sensing pathways activated by physical exercise, earning it the “exercise-mimetic” framing that appears throughout the literature.

Perhaps the most striking mechanistic finding came in 2018, when Kim and colleagues demonstrated that under conditions of metabolic stress — glucose restriction, oxidative stress, serum deprivation — MOTS-c physically translocates from the cytoplasm into the nucleus. There it interacts with nuclear DNA and regulates the expression of genes involved in antioxidant defense and metabolic adaptation, particularly genes containing antioxidant response elements. This nuclear translocation represents a form of "mitonuclear communication" not previously observed for any mitochondrial-derived peptide, and it fundamentally expanded the understood scope of the molecule: a peptide encoded in the mitochondrial genome that can directly regulate nuclear gene expression.

The implications are significant for aging and metabolic science, positioning MOTS-c as a potential communication channel between the mitochondrial and nuclear genomes during periods of cellular stress. Research has connected its metabolic effects to insulin sensitivity, exercise capacity, and age-related metabolic decline in animal models.

For researchers, MOTS-c is compelling precisely because it links mitochondrial biology, AMPK energy signaling, and epigenetic gene regulation in a single small peptide — a rare combination that makes it a reference compound in metabolic-longevity and mitochondrial research.

Mechanism & receptor target

Research examines its activation of the AMPK energy-sensing pathway and its influence on glucose uptake and fatty-acid oxidation in skeletal-muscle models, effects often described as exercise-mimetic in the literature.

In the research

Used in metabolic and mitochondrial-aging research as a reference mitochondrial-derived peptide.

Reference specifications

Sequence	Met-Arg-Trp-Gln-Glu-Met-Gly-Tyr-Ile-Phe-Tyr-Pro-Arg-Lys-Leu-Arg
Molecular formula	C ₁₀₁ H ₁₅₂ N ₂₈ O ₂₂ S ₂
Molecular weight	2174.64 g/mol
CAS number	1627580-64-6
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

MOTS-c is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



SS-31

Elamipretide \u00b7 MTP-131

SS-31 (also known as elamipretide) is a cell-permeable, mitochondria-targeting tetrapeptide that concentrates on the inner mitochondrial membrane. It is one of the most-studied compounds in mitochondrial-bioenergetics research.

What does SS-31 do?

SS-31 (also known as elamipretide or MTP-131) is a mitochondria-targeting tetrapeptide, and it is one of the most mechanistically specific compounds in this guide. Where most peptides act on cell-surface receptors, SS-31 concentrates on the inner mitochondrial membrane and works at the biophysical level of mitochondrial organization itself.

Its mechanism centers on selective binding to cardiolipin, a distinctive tetra-acyl phospholipid found almost exclusively in the inner mitochondrial membrane. Cardiolipin plays indispensable roles in mitochondrial biology: it anchors and organizes the electron transport chain (ETC) supercomplexes, maintains the curvature of the cristae (the folded inner-membrane structures where energy production occurs), facilitates cytochrome c interactions, and supports ATP synthase assembly. Research by Szeto and colleagues demonstrated that SS-31 binds cardiolipin through electrostatic and hydrophobic interactions, stabilizing the cardiolipin–cytochrome c relationship and preventing cytochrome c from converting into a peroxidase.

That last point is central to the compound's rationale. Under normal conditions, cytochrome c shuttles electrons between Complex III and Complex IV. However, when cardiolipin becomes oxidized — as occurs during ischemia, aging, and metabolic disease — cytochrome c binds more tightly to the damaged cardiolipin and acquires peroxidase activity, further oxidizing cardiolipin in a destructive positive-feedback loop. By stabilizing this relationship, SS-31 is studied for interrupting that cycle and restoring several downstream functions: improved electron-transport-chain efficiency (reduced electron leak and reactive-oxygen-species generation), enhanced ATP synthesis, preserved cristae architecture, and better mitochondrial membrane integrity supporting balanced fission and fusion.

This mechanism is distinct from peptides such as MOTS-c, which act as signaling molecules through pathways like AMPK. Whereas MOTS-c modulates cellular metabolism at the transcriptional level, SS-31 acts directly at the biophysical level of mitochondrial membrane organization. Much of its clinical-stage research attention has centered on mitochondrial diseases such as Barth syndrome, where cardiolipin biology is directly implicated.

For researchers, SS-31 is valuable as a precise molecular tool for studying mitochondrial membrane function, cardiolipin biology, and bioenergetics — a compound whose mechanism is unusually well-defined for this field.

Mechanism & receptor target

Research investigates its selective binding to cardiolipin on the inner mitochondrial membrane and the reported effects on cristae architecture, electron-transport efficiency and reactive-oxygen-species handling in model systems.

In the research

Used in mitochondrial-function, bioenergetics and cellular-aging research as a reference membrane-targeting peptide.

Reference specifications

Sequence	D-Arg-Dmt-Lys-Phe-NH
Molecular formula	C ₂₈₃ H ₂₈₄ N ₂₈₉ O ₂₈₅
Molecular weight	639.79 g/mol
CAS number	736992-21-5
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

SS-31 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 04 · LONGEVITY & MITOCHONDRIAL

Epithalon

Epitalon · AEDG

Epithalon is a synthetic tetrapeptide (Ala-Glu-Asp-Gly) modeled on the pineal peptide epithalamin. It is one of the most-cited compounds in experimental longevity and telomere research.

What does Epithalon do?

Epithalon (also written Epitalon; sequence Ala-Glu-Asp-Gly, or AEDG) is a synthetic tetrapeptide modeled on epithalamin, a natural peptide extract of the pineal gland. It sits at the center of experimental longevity research, and its investigation revolves around four interconnected mechanisms: telomerase activation, melatonin regulation, antioxidant activity, and gene-expression modulation.

The mechanism that draws the most attention is telomerase activation. Telomeres are the protective nucleotide caps at the ends of chromosomes that shorten with each cell division, and telomere shortening is a central hallmark of cellular aging. In preclinical models, epithalon has been reported to upregulate telomerase — the enzyme that maintains and extends telomeres — potentially slowing or partially offsetting this shortening. A frequently cited line of work has examined telomere elongation in human cell lines through both telomerase upregulation and alternative lengthening of telomeres (ALT) activity, providing the theoretical foundation for most anti-aging research associated with the peptide.

Beyond telomeres, epithalon is studied for stimulating melatonin production from the pineal gland, connecting it to circadian-rhythm regulation and endogenous antioxidant defense. Animal studies have reported normalization of melatonin secretion patterns in aging rodents, where pineal function naturally declines. This dual mechanism — telomere maintenance plus melatonin restoration — is what researchers use to frame its longevity relevance.

An essential context point concerns the evidence base itself. The majority of epithalon data originates from Russian research groups, particularly the laboratories associated with Khavinson and Anisimov. While the findings are internally consistent across multiple publications, independent replication from Western institutions has historically been limited. This does not invalidate the data, but it means the evidence carries a concentration risk that should inform interpretation.

For researchers, epithalon is compelling as a small, stable, well-defined tetrapeptide that touches several core mechanisms of aging science at once — telomere biology, chronobiology, and pineal signaling — making it a recurring reference compound in cellular-aging and longevity research.

Mechanism & receptor target

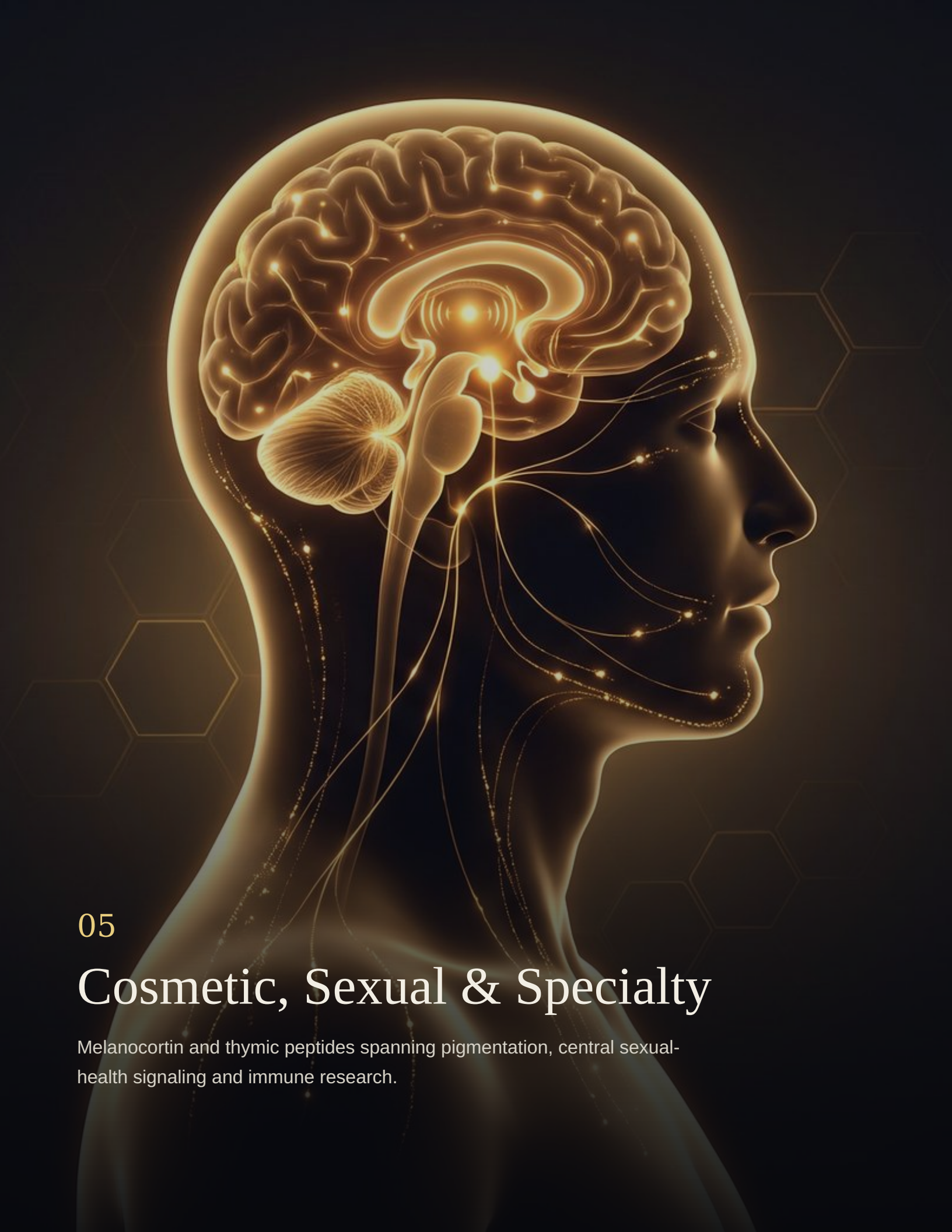
Research investigates its reported influence on telomerase expression and telomere maintenance in cultured cells, as well as its interaction with pineal and circadian regulatory pathways in model systems.

In the research

Used in cell-aging, telomere-biology and chronobiology research as a small, stable reference peptide.

Reference specifications

Sequence	Ala-Glu-Asp-Gly
Molecular formula	C ₁₄ H ₂₂ N ₄ O ₉
Molecular weight	390.35 g/mol
CAS number	307297-39-8
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light



05

Cosmetic, Sexual & Specialty

Melanocortin and thymic peptides spanning pigmentation, central sexual-health signaling and immune research.



Melanotan II

cyclic melanocortin agonist

Melanotan II is a synthetic cyclic heptapeptide analog of α -melanocyte-stimulating hormone. It is a widely studied research probe of the melanocortin-receptor family.

What does Melanotan II do?

Melanotan II (MT-II) is a synthetic cyclic analog of alpha-melanocyte-stimulating hormone (α -MSH), and its defining pharmacological feature is that it is a *non-selective* melanocortin receptor agonist — it activates four of the five known melanocortin receptor subtypes. Because those receptors govern very different physiological processes, MT-II produces a characteristically broad, multi-dimensional activity profile that researchers study across several distinct areas.

The most recognized mechanism is melanogenesis via the MC1R receptor. MC1R is expressed primarily on epidermal melanocytes; when activated by MT-II, it triggers a cAMP-dependent signaling cascade that upregulates tyrosinase activity and shifts melanin production from the lighter pheomelanin toward the darker eumelanin. This is the mechanism underlying the compound's well-documented tanning effect in research subjects — increased eumelanin synthesis producing visible skin darkening, notably in a manner that can occur independently of ultraviolet exposure, though it works synergistically with UV.

A second dimension operates through the centrally expressed MC3R and MC4R receptors, which mediate effects on sexual arousal and appetite. MC4R activation in the hypothalamus has been shown to initiate sexual-response pathways and to suppress food intake — contributing to the anorexigenic (appetite-reducing) effects observed in melanocortin research. MC3R contributes to both sexual arousal and energy homeostasis, though its specific role is less well characterized. The MC5R receptor, expressed in exocrine glands, adds a further, less-studied dimension related to sebaceous and exocrine function.

The core research consideration with MT-II is precisely this non-selectivity: the desired melanogenic effect via MC1R cannot be cleanly separated from the centrally mediated sexual, appetite, and autonomic effects via MC3R/MC4R, or from the nausea and fatigue that appear to be dose-dependent class effects in the literature. This makes MT-II a compound of significant mechanistic interest but complex, overlapping activity.

For researchers, Melanotan II is a valuable tool for studying the melanocortin system as a whole — pigmentation, central appetite and sexual-function circuitry, and receptor cross-talk — precisely because it engages the majority of that receptor family at once.

Mechanism & receptor target

It acts as a non-selective agonist across melanocortin receptors (including MC1R and MC4R). Research examines MC1R-linked pigmentation pathways and MC4R-linked central signaling in model systems.

In the research

Used in melanocortin-pharmacology research as a broad-spectrum reference agonist.

Reference specifications

Sequence	Ac-Nle-cyclo[Asp-His-D-Phe-Arg-Trp-Lys]-NH ₂
Molecular formula	C ₅₀ H ₆₉ N ₁₅ O ₉
Molecular weight	1024.18 g/mol
CAS number	121062-08-6
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, -20°C, protect from light

Melanotan II is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



CLASS 05 · COSMETIC, SEXUAL & SPECIALTY

PT-141

Bremelanotide · MC4R agonist

PT-141 (bremelanotide) is a cyclic heptapeptide within the melanocortin family, studied for its relative preference for central melanocortin signaling.

What does PT-141 do?

PT-141 (bremelanotide) is a melanocortin receptor agonist derived from the same peptide family as Melanotan II, but it is distinguished by *where* and *how* it acts. Its defining characteristic is that it operates centrally — in the brain — rather than peripherally in vascular tissue, which sets it fundamentally apart from the mechanism of PDE5 inhibitors such as sildenafil and tadalafil. Those compounds increase blood flow to peripheral tissues; PT-141 instead activates melanocortin receptors in the hypothalamus, modulating the neural circuitry that governs sexual desire and arousal at its origin point.

Mechanistically, bremelanotide's activity centers on activation of the melanocortin-3 (MC3R) and melanocortin-4 (MC4R) receptors in the medial preoptic area and paraventricular nucleus of the hypothalamus. These regions are critical integration hubs for sexual motivation, connecting sensory input, hormonal status, and emotional context into behavioral output. In practical research terms, this means PT-141 targets the *desire* component of sexual function — the central nervous-system pathways that initiate arousal — rather than the *mechanical* component of blood flow and tissue response.

This distinction is why bremelanotide was investigated specifically in the context of hypoactive sexual desire disorder (HSDD), a condition characterized by persistently low sexual desire causing distress, rather than for erectile dysfunction or arousal disorders with purely vascular origins. Its relative specificity for the MC3R and MC4R subtypes gives it a more targeted sexual-function profile compared with broader melanocortin agonists like Melanotan II, which activate all five receptor subtypes and therefore carry the wider, overlapping effects of full melanocortin-system stimulation.

Because it was developed through formal clinical channels (marketed under the brand name Vyleesi by AMAG Pharmaceuticals for a specific indication), PT-141 has a more defined pharmacological and regulatory characterization than many research peptides — its key parameters were established through clinical development and regulatory review.

For researchers, PT-141 is a useful reference compound for studying the central, melanocortin-mediated pathways of sexual motivation, precisely because it isolates the neural "desire" circuitry from the peripheral vascular mechanisms targeted by other classes.

Mechanism & receptor target

Research focuses on its agonism at the melanocortin-4 receptor (MC4R) in the central nervous system — a pathway distinct from the peripheral pigmentation signaling of MC1R.

In the research

Investigated in melanocortin and central-signaling research as an MC4R-oriented reference peptide.

Reference specifications

Sequence	Ac-Nle-cyclo[Asp-His-D-Phe-Arg-Trp-Lys]-OH
Molecular formula	C ₅₀ H ₆₈ N ₁₄ O ₁₀
Molecular weight	1025.16 g/mol
CAS number	189691-06-3
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

PT-141 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.



Thymosin α -1

Thymalfasin

Thymosin alpha-1 (thymalfasin) is a 28–amino-acid acetylated peptide derived from the precursor prothymosin-alpha. It is one of the most-studied immune-regulatory research peptides.

What does Thymosin α -1 do?

Thymosin alpha-1 (T α 1) is a 28–amino-acid peptide originally isolated from thymic tissue, and it is fundamentally an immune-regulating compound. Where most peptides in this guide are studied for metabolic or tissue-repair effects, T α 1's research profile is defined by its influence on both the innate and adaptive arms of the immune system, acting through several interconnected immunological pathways.

The most established mechanism is its effect on T-cell maturation and differentiation. T α 1 promotes the maturation of T-cell precursors (thymocytes) into functional T-cells, enhancing the expression of key T-cell markers including CD3, CD4, and CD8. This supports the development of both helper and cytotoxic T-cell populations, strengthening adaptive immune surveillance — the mechanism most directly tied to the peptide's thymic origin.

A second major pathway is dendritic-cell activation. Dendritic cells are the principal antigen-presenting cells that initiate adaptive immune responses, and research (Romani and colleagues, 2007) has shown that T α 1 promotes their maturation and function through Toll-like receptor-dependent pathways, enhancing antigen presentation and downstream T-cell activation. This positions T α 1 as a bridge between innate pathogen detection and adaptive immune activation. Relatedly, T α 1 acts as an endogenous activator of Toll-like receptor signaling itself — particularly TLR2 and TLR9 — with work (Bozza and colleagues, 2007) demonstrating activation of the TLR9/MyD88/IRF7 pathway and induction of antiviral responses in viral-infection models. This connects the peptide to pattern-recognition receptor biology, the frontline system by which the immune system detects pathogen-associated molecular patterns.

Beyond these, T α 1 has been studied for enhancing innate immune activity, including macrophage activation and natural-killer (NK) cell cytotoxicity — components of the body's frontline surveillance against virally infected and transformed cells. The overall picture is of a coordinated immune-enhancement profile that touches T-cells, dendritic cells, TLR signaling, and innate effector cells simultaneously.

For researchers, thymosin alpha-1 is a valuable reference compound in immunology precisely because it modulates multiple immune pathways at once rather than a single target, making it relevant across studies of immune regulation, antiviral responses, and host defense.

Mechanism & receptor target

Research examines its modulation of T-cell maturation and innate-immune signaling, including interactions with Toll-like-receptor pathways and dendritic-cell function in model systems.

In the research

Used in immunology research as a well-characterized thymic-peptide reference material.

Reference specifications

Sequence	Ac-Ser-Asp-Ala-Ala-Val-Asp-Thr-Ser-Ser-Glu-Ile-Thr-Thr-Lys-Asp-Leu-Lys-Glu-Lys-Lys-Glu-Val-Val-Glu-Glu-Ala-Glu-Asn
Molecular formula	C ₁₂₉ H ₂₁₅ N ₃₃ O ₅₅
Molecular weight	3108.30 g/mol
CAS number	62304-98-7
Form	Lyophilized powder
Purity	≥99% (HPLC & mass spec)
Storage	Lyophilized, −20°C, protect from light

Thymosin α-1 is supplied strictly for research use only. Not for human or animal consumption. No dosing, protocol, or health-outcome guidance is provided.

Sourcing, purity & documentation

Every compound in this reference is sourced, tested, and documented to a single consistent laboratory standard. Materials are US-sourced and produced under controlled processes, then independently verified before release.

Independent analytical verification

Each batch is analyzed by a third-party laboratory rather than relying on in-house claims. Reversed-phase high-performance liquid chromatography (HPLC) establishes chromatographic purity — typically $\geq 99\%$ — by resolving the target peptide from process-related impurities, truncated sequences, and residual reagents. Mass spectrometry independently confirms molecular identity, matching the measured mass to the theoretical mass of the intended sequence. Used together, these orthogonal methods verify both that the material is what it claims to be and that it is as pure as stated.

Batch-specific Certificate of Analysis

A Certificate of Analysis (COA) accompanies every compound and is tied to the exact lot shipped. It records the product identity and sequence, the measured purity, the analytical methods used, and the lot number and date of analysis. Retaining the COA alongside experimental records supports reproducibility and lets any result be traced back to a documented, verified source material.

Form, stability, storage & traceability

Compounds are supplied as lyophilized (freeze-dried) powder — the most stable format for peptide storage and transport — and ship with appropriate cold-pack protection. Removing water slows the hydrolysis and aggregation that degrade peptides in solution, so material is intended to be stored lyophilized and frozen, protected from light and moisture, until a study calls for it. Once reconstituted with an appropriate solvent such as bacteriostatic water, working solutions are kept refrigerated and used within a limited window to minimize degradation. Because each vial carries a lot number linked to its COA, materials remain fully traceable from batch to bench — a prerequisite for rigorous, repeatable laboratory work.

For Research Use Only

The information in this guide is provided for scientific and educational context only. It is not medical advice. The compounds described are not approved for human use and are not intended to diagnose, treat, cure, or prevent any condition.

