Peptide Pens Europe · educational reference

Peptide Atlas · research framework (RUO)

Peptide Atlas

What each peptide is, how it is classified, what is being studied and how it is verified. Category by category, with the evidence kept in context. Information, not sales.

2026 Edition · Scientific review: Equipo editorial

research use only

Before we begin

What this atlas is

An educational reference to the best-documented peptides, organised by category of effect. It is not a sales catalogue or a usage guide: it is a map for understanding what each molecule is and what the research says, read with a critical eye.

Each category brings together the peptides that share an area of interest, explains the biology behind them and presents a profile for each molecule: what it is, how it is classified, its proposed mechanism, what it is studied for, the state of that evidence and how its quality is verified. Always with sources.

The same standard runs through the atlas from start to finish. Most of these peptides are **for research use only**: the evidence is usually preclinical and many are not approved for human use. We say so in each case, without alarmism and without overstating the opposite.

> **Important** Research framework (RUO). This document is educational; it does not sell products, recommend uses or doses, and does not replace the advice of a healthcare professional. "Is studied in the context of..." does not mean "is used for...".

Contents

Part I

Fundamentals

Before the catalogue, the essentials: what a peptide is, how it acts, what "research use only" means and how we confirm that a molecule is what it claims to be.

What a peptide is

A peptide is a short chain of amino acids, the same building blocks the body uses to assemble proteins. The difference is one of size and of function.

Amino acids link one after another through a peptide bond and form a chain. When that chain is short we speak of a peptide; when it is long and folds into a complex three-dimensional structure, of a protein. The boundary is conventional and is usually placed at around fifty amino acids. Insulin, for example, sits right at that limit: a small molecule with a very precise job.

Messengers, not materials

Many peptides do not build tissue: they coordinate it. They act as chemical messengers that dock onto a specific receptor and trigger a particular signal, like a key that opens a single lock. That specificity is the reason a peptide can have a well-defined effect and, at the same time, the reason its exact sequence matters so much.

Peptide and protein: the same chemistry, a different scale

Feature	Peptide	Protein
Length	Short chain (up to ~50 aa)	Long chain, hundreds of aa
Structure	Simple, little folding	Folded in 3D, complex
Typical role	Signal, messenger	Structure, enzyme, transport

Natural and synthetic

Many peptides occur naturally in the body: oxytocin, glucagon or GLP-1 itself. Others are synthesised in the laboratory, either by copying a natural sequence or by designing a new one with desired properties, such as greater stability. Most of the molecules in this atlas are synthetic and are studied within that framework.

How they work: the signal and the receptor

A signalling peptide acts by binding to a receptor. Understanding that relationship explains almost everything else: why they are specific, why purity matters and why the smallest change changes everything.

The receptor is a protein, usually on the cell surface, with a shape that recognises a particular ligand. When the peptide fits, the receptor changes its conformation and sets off a cascade of signals inside the cell. Depending on the case, the peptide may activate the receptor (agonist) or block it (antagonist).

Agonist

`activates`

Binds to the receptor and switches it on, mimicking or reinforcing the natural signal. Most peptides of therapeutic interest are agonists of some receptor.

Antagonist

`blocks`

Binds to the receptor and occupies it without activating it, preventing the natural signal from doing so. It serves to dampen a pathway rather than stimulate it.

Why the exact sequence is everything

Because recognition depends on shape, the precise order of the amino acids defines the molecule's identity and its effect. A wrong sequence, an impurity or a poorly synthesised fragment may fail to fit the receptor, fit a different one or behave unpredictably. That is why the first question to ask of any peptide is not what it does, but whether it really is what it claims to be. That is the bridge to verification.

Research peptides and the RUO framework

"Research use only" is not a decorative label. It defines what these molecules legally are and how everything said about them should be read.

A research peptide (RUO, for *research use only*) is a compound intended for laboratory study, not approved as a medicine for human use. That means it has not been through the full evaluation of efficacy and safety that a regulatory agency requires before authorising a drug. The distinction is enormous.

Two categories worth keeping apart

	Approved drug	Research peptide (RUO)
Regulatory evaluation	Complete (EMA, FDA…)	Not approved for human use
Evidence	Published clinical trials	Often preclinical or limited
Intended use	Treatment, by prescription	Research study

In the catalogue you will see that some molecules, such as semaglutide, are indeed the active ingredients of approved drugs, while most remain within the research domain. We flag this in every profile with the regulatory status, because it completely changes how the information should be read.

How to read it

When the atlas says that a peptide "is studied in the context of" something, it describes a line of research, not a promise of a result. Preclinical evidence is a starting point, not a conclusion about people.

And one constant: none of this is medical advice. Any decision about health rests with a healthcare professional who can assess the individual case.

Identity, purity and verification

Before talking about mechanisms, there is a prior question: is the molecule really what it claims to be, and is it clean? The certificate of analysis answers that.

The certificate of analysis (COA) is the laboratory report that establishes the identity and purity of a batch. It answers two different questions with two different techniques.

Identity

LC-MS

Mass spectrometry measures the molecular mass of the sample and compares it with the theoretical mass of the declared peptide. It confirms that what is inside matches the label.

Purity

HPLC

Liquid chromatography separates the components and measures what proportion is the peptide against the impurities. The accepted standard for research is ≥98%.

Throughout the atlas, each profile includes the actual purity of the client's reference batch, measured by HPLC. These are high, consistent figures (generally above 99%), which provides a solid basis on which to read the rest. The verification standard, in five steps, is the same for any molecule:

1 **Batch**
The COA corresponds to the exact batch you are going to receive.

2 **Identity**
The mass by LC-MS matches the declared molecular weight.

3 **Purity**
HPLC ≥98%, backed by a chromatogram with a single clean main peak.

4 **Laboratory**
Issued by an independent laboratory that can be identified and verified.

5 **Clinical judgement**
Quality can be verified; use, dosing and follow-up rest with a doctor.

Note There is a dedicated guide solely on reading a COA step by step and spotting counterfeits. This atlas assumes that standard and focuses on what each peptide is.

How to read this atlas

Every chapter of the catalogue follows the same structure, so you can compare categories and molecules on the same terms.

A category chapter opens with the **area of effect**: what those peptides have in common and the biology that links them. Then comes a **profile per peptide** with these fields:

- **Class.** The chemical or functional family it belongs to.
- **Proposed mechanism.** How it is believed to act, according to the available research.
- **Studied in the context of.** The lines of research, without claiming these are proven effects in people.
- **Level of evidence.** From preclinical through to clinical or approved.
- **Regulatory status.** Whether it is an approved drug or a research compound (RUO).
- **Purity and batch.** The real quality data for the reference batch.

The two fields that matter most

The **level of evidence** and the **regulatory status** are the compass. Read them first: they tell you how much weight to give what follows and what legal ground you are standing on.

Part II

The catalogue by category

Thirteen categories of effect, from metabolic control to immunity. Each with its biology, its molecules and the real state of the evidence.

Map

The 13 categories

An overview before the detail. The bar on the right marks the highest level of evidence in the category, from preclinical to approved.

06 GLP-1 and metabolic control
4 molecules

07 Recovery and tissue repair
3 molecules

08 Skin and anti-ageing
3 molecules

09 Growth hormone
4 molecules

10 Libido and sexual function
1 molecule

11 Tanning and melanocortin
1 molecule

12 Metabolic and mitochondrial
2 molecules

13 Longevity
3 molecules

14 Antioxidant
1 molecule

15 Cognitive and nootropic
2 molecules

16 Anti-inflammatory
1 molecule

17 Immune
1 molecule

18 Hormonal
1 molecule

Level of evidence: preclinical · early clinical · broad clinical · approved.

GLP-1 and metabolic control

06

Peptides that act on the axis of the incretin and satiety hormones (GLP-1, GIP, glucagon and amylin): the receptors through which the body coordinates insulin, gastric emptying and hunger.

When we eat, the gut and pancreas release incretin hormones such as GLP-1 and GIP, along with amylin. Together they modulate the glucose-dependent release of insulin, slow gastric emptying and signal fullness to the brain. The peptides in this class are synthetic analogues designed to activate those same receptors with an extended half-life, generally through acylation with a fatty acid that binds them to albumin and protects them from degradation.

The category runs from lower to higher complexity. Semaglutide is a selective GLP-1 agonist and the most established, approved by the FDA and the EMA. Tirzepatide adds a second target, the GIP receptor, and is also approved. Retatrutide is a triple agonist (GLP-1, GIP and glucagon), still in phase 3 and not approved. Cagrilintide belongs to a different pathway: it is a long-acting amylin analogue, studied above all alongside semaglutide (CagriSema). One class trait: the most common adverse effects are gastrointestinal, particularly when the dose is increased.

The molecules in the category

Peptide	Class	Evidence	Status	Purity
Semaglutide	GLP-1 agonist	■■■■ Phase 3	Approved	–
Tirzepatide	GLP-1 / GIP	■■■■ Phase 3	Approved	99.78%
Retatrutide	Triple GLP-1/GIP/glucagon	■■■□ Phase 3	RUO	99.91%
Cagrilintide	Amylin analogue	■■■□ Phase 2-3	RUO	–

Semaglutide

Ozempic · Wegovy · Rybelsus

Selective GLP-1 agonist

A synthetic analogue of GLP-1, the incretin hormone the body releases after eating, designed to resist degradation and remain active for several days. It is the most established molecule in the category.

MECHANISM	Proposed: binds selectively to the GLP-1 receptor and activates it; associated with glucose-dependent insulin secretion, lower postprandial glucagon, slower gastric emptying and signalling in appetite-related brain regions.
STUDIED FOR	Glycaemic control in type 2 diabetes, chronic weight management and cardiovascular risk reduction in selected populations.
EVIDENCE	High: broad phase 3 and an approved molecule in specific indications.
STATUS	Approved by the FDA and the EMA (diabetes as Ozempic/Rybelsus; weight and cardiovascular risk as Wegovy). The RUO catalogue versions are not for human use.
SAFETY	Boxed warning for thyroid C-cell tumours in rodents; contraindicated with a history of medullary thyroid carcinoma or MEN 2. Acute pancreatitis reported. Gastrointestinal effects are common.

CAS 910463-68-2 · C187H291N45O59 · 4113.58 g/mol · ~31 aa

Tirzepatide

Mounjaro · Zepbound · LY3298176

Dual GIP / GLP-1 agonist

A synthetic dual-target peptide: it activates both the GIP and GLP-1 receptors at once. Described as first-in-class among the dual incretin agonists.

MECHANISM	Proposed: simultaneously activates GIP and GLP-1, with greater affinity for GIP and cAMP-biased agonism at GLP-1. Associated with glucose-dependent insulin, lower glucagon, slowed emptying and satiety.
STUDIED FOR	Glycaemia in type 2 diabetes, weight management and, more recently, obstructive sleep apnoea in adults with obesity.
EVIDENCE	High: phase 3 programme (SURPASS / SURMOUNT) and approvals across several indications.
STATUS	Approved by the FDA (Mounjaro 2022; Zepbound 2023; sleep apnoea 2024) and the EMA (2022). The RUO versions are not for human use.
SAFETY	Same class warning for thyroid C-cells (contraindicated with a history of medullary carcinoma or MEN 2). Signals of pancreatitis and gallbladder events. Gastrointestinal effects are common when escalating the dose.

CAS 2023788-19-2 · C225H348N48O68 · 4813.45 g/mol · 39 aa · purity 99.78% · batch GT10-032026-1

Retatrutide LY3437943 · triple agonist

Triple GIP / GLP-1 / glucagon agonist

An investigational molecule, the first triple agonist of its class: it acts on three receptors (GLP-1, GIP and glucagon). It adds the glucagon receptor to the scheme of semaglutide and tirzepatide.

MECHANISM	Proposed: activates GIP, GLP-1 and glucagon at once. The glucagon component is thought to influence energy expenditure, in addition to the incretin effects. Still being characterised.
STUDIED FOR	Weight management in obesity, glycaemia in type 2 diabetes and, in the phase 3 TRIUMPH programme, also sleep apnoea and pain from knee osteoarthritis.
EVIDENCE	Moderate and evolving: phase 2 published (NEJM, 2023) and phase 3 (TRIUMPH) under way. Not approved.
STATUS	Not approved in any country (Jun 2026); in phase 3, with no regulatory submission filed. RUO framework: not intended for human use.
SAFETY	In phase 2, adverse events were mainly gastrointestinal (mild to moderate), more so at high doses; discontinuations ~6-16% depending on dose. Long-term profile not established.

CAS 2381089-83-2 · ~4731.33 g/mol · 39 aa · purity 99.91% · batch K3BQAA

Cagrilintide AM833 · NN9838 · CagriSema (with semaglutide)

Long-acting amylin analogue

A long-acting synthetic version of amylin, the hormone the pancreas co-secretes with insulin when we eat. It belongs to a different pathway from the GLP-1s and is studied above all alongside semaglutide (CagriSema).

MECHANISM	Proposed: a non-selective agonist of the amylin (AMY1-3R) and calcitonin receptors, mainly in the area postrema and hypothalamus, modulating appetite and satiety; an action on reward pathways is also postulated.
STUDIED FOR	Weight management and obesity, as monotherapy and in co-formulation with semaglutide, and glycaemic control.
EVIDENCE	Moderate: phase 2 as monotherapy and phase 3 of CagriSema (REDEFINE). No standalone approval.
STATUS	No standalone authorisation from the FDA or the EMA (Jun 2026). Novo Nordisk filed an application (NDA) for CagriSema in Dec 2025, under review. RUO framework.
SAFETY	Shares the gastrointestinal profile of the class, with a nausea and vomiting signal possibly lower than semaglutide as monotherapy. Long-term data still accumulating.

CAS 1415456-99-3 · ~4409 g/mol · 37-aa amylin analogue

› DrugBank — Semaglutide (DB13928) · FDA — Wegovy Prescribing Information (boxed warning).

› Eli Lilly / FDA — tirzepatide approvals (Mounjaro, Zepbound).

› NEJM 2023 — Triple-Hormone-Receptor Agonist Retatrutide for Obesity (phase 2).

› PMC / Novo Nordisk — cagrilintide and CagriSema (mechanism and regulatory status).

RUO Semaglutide and tirzepatide are the active ingredients of approved drugs; retatrutide, cagrilintide and the catalogue versions are research material (RUO), not intended for human use. This is not medical advice.

Recovery and tissue repair

07

Peptides that preclinical research studies for their possible role in wound healing: angiogenesis, fibroblast migration, matrix remodelling and modulation of inflammation. This is a largely preclinical field.

Repairing injured tissue is one of biology's most finely tuned feats of orchestration. After a wound, the body stems the bleeding, recruits immune cells, builds new vessels to bring oxygen to the area (angiogenesis), mobilises fibroblasts that weave collagen and remodels that scaffold until function is restored to the tissue. Molecular signals act on every step, and that is where research turns its attention.

This chapter is best read with a clear compass: almost everything known about these compounds comes from preclinical studies (rodents and cell cultures). Validation in humans is very limited today, with no large, rigorous trials. That is why you will find no promises or protocols here, only an honest account of what is being studied and on how much evidence. They are not approved for human use and are listed as prohibited substances in sport (WADA).

The molecules in the category

Material	Class	Evidence	Status	Purity
BPC-157	Pentadecapeptide (15 aa)	●○○○ Preclinical	RUO · WADA	99.51%
TB-500	Thymosin β4 frag.	●○○○ Preclinical	RUO · WADA	99.58%
BPC-157 + TB-500	Blend	●○○○ No data	RUO · WADA	99.29%

BPC-157

Body Protection Compound 157 · gastric pentadecapeptide

Synthetic peptide (15 aa)

A synthetic 15-amino-acid peptide whose sequence corresponds to a fragment of a protein that protects the gastric juice. The preclinical literature describes it as cytoprotective. Research material, not approved.

MECHANISM	Proposed, mainly in animals and cell cultures: interaction with the nitric oxide pathway (Akt-eNOS) and angiogenesis via VEGFR2 / VEGF; effects on fibroblast migration and an anti-inflammatory profile. Research hypotheses, not demonstrated in people.
STUDIED FOR	In preclinical models: repair of tendons and ligaments, muscle regeneration, skin wound healing, protection of the gastrointestinal mucosa and angiogenesis.
EVIDENCE	Mostly preclinical. A stark contrast between the abundant animal research and the near-absence of evidence in humans (a few small pilot studies). Reviews note probable publication bias.
STATUS	Not approved by any agency. In the US it was an FDA Category 2 substance (compounding concerns) and was removed from that category in Apr 2026, which does not amount to approval. Prohibited in sport (WADA, S0). RUO.
SAFETY	No robust evidence of safety in humans. Unconfirmed theoretical risks, such as pathological angiogenesis. Market purity can vary.

CAS 137525-51-0 · C62H98N16O22 · ~1419.5 g/mol · 15 aa · purity 99.51% · batch 2FRBRG

TB-500

Thymosin β4 fragment · name used ambiguously

Research peptide (thymosin β4)

Research material linked to thymosin β4, a natural actin-binding protein involved in cell migration, angiogenesis and wound healing. The name "TB-500" is applied ambiguously to either the full protein or its Ac-LKKTETQ fragment.

MECHANISM	Proposed: thymosin β4 sequesters G-actin and regulates the balance between globular and filamentous actin, associated in models with cell migration, re-epithelialisation and angiogenesis. Laboratory mechanisms, not demonstrated in people with "TB-500".
STUDIED FOR	In preclinical models: wound healing, tissue repair and recovery after injury. Thymosin β4 as a molecule (not the fragment) has been trialled in limited ophthalmic indications; it is unwise to extrapolate from one to the other.
EVIDENCE	For "TB-500" as such: preclinical, with no controlled trials. The most advanced clinical development relates to the protein formulated as an investigational drug, not to the RUO material.
STATUS	Not approved by the FDA or the EMA. Treated as Category 2 (compounding). Prohibited in sport (WADA, growth factors S2.3 and S0). RUO.
SAFETY	No large-scale or long-term safety studies in humans. Purity and composition can vary between products.

CAS 77591-33-4 (thymosin β4) · thymosin β4: 43 aa · ~4963 g/mol · purity 99.58% · batch PQ7MWD

BPC-157 + TB-500 recovery blend

Combination of two research peptides

A mixture of the two materials above in a single preparation. It is not a new molecule or an approved drug, but the co-formulation of both components.

MECHANISM	No mechanism of its own: the sum of each component's proposed mechanisms is assumed. No study characterises a combined effect in humans; any synergy is hypothetical.
STUDIED FOR	The interest comes from the tissue-repair context, but there is no evidence specific to the combination: the backing, preclinical, relates to the components separately.
EVIDENCE	No evidence specific to the mixture. It is the entry with the least direct backing in the section.
STATUS	Not approved. It inherits the status of its components: not approved and prohibited in sport (WADA). RUO.
SAFETY	Combining two compounds with no studies of their own adds uncertainty: there are no data on interactions or on the safety of the mixture in humans.

Combination without a single CAS · purity 99.29% · batch BPTB20-052026-4

› PMC — Multifunctionality and Possible Medical Application of the BPC 157 Peptide (review).

› PMC — Regeneration or Risk? A Narrative Review of BPC-157 for Musculoskeletal Healing.

› USADA / US DoD (OPSS) — BPC-157, experimental peptide and unapproved drug.

› WADA — The Prohibited List · BSCG — TB-500: status, risks and bans.

RUO All the compounds in this category are research material (RUO), not approved for human use, and appear on WADA's list of prohibited substances in sport. The evidence is mostly preclinical. This is not medical advice.

Catalogue · Part II

Skin and anti-ageing

08

Peptides studied for their interaction with the dermal matrix (collagen, elastin, glycosaminoglycans), where fibroblasts and the vascular network determine firmness, hydration and wound healing. An area of preclinical research and, in a few cases, of topical cosmetics.

Beneath the epidermis, the dermis is a living scaffold of collagen and elastin held together by fibroblasts and nourished by capillaries. Over the years, matrix synthesis declines, oxidative damage accumulates and enzymes such as elastase break down the fibres that provide firmness. Against that backdrop, science is exploring small peptides as candidates to modulate the repair signals. It is worth saying upfront: apart from the use of GHK-Cu as a cosmetic ingredient, these are research materials with no approval for human use.

The central molecule is GHK-Cu, a copper tripeptide that occurs naturally in plasma and whose concentration declines with age. The other two entries are mixtures: GLOW combines GHK-Cu with BPC-157 and TB-500; KLOW adds KPV, an anti-inflammatory tripeptide. There are no clinical trials validating the mixtures as such: what is known comes from each component separately, mostly at preclinical stages. BPC-157 and TB-500, moreover, are prohibited in sport (WADA).

The molecules in the category

Material	Class	Evidence	Status	Purity
GHK-Cu	Copper tripeptide	●●○○ Preclinical + topical	RUO	99.75%
GLOW Blend	Mixture (GHK-Cu + BPC-157 + TB-500)	●○○○ No data	RUO	99.60%
KLOW Blend	Mixture (KPV + GHK-Cu + BPC-157 + TB-500)	●○○○ No data	RUO	99.76%

GHK-Cu

Copper tripeptide-1 · Gly-His-Lys:Cu

Copper tripeptide

A copper tripeptide (glycyl-histidyl-lysine bound to copper) present naturally in human plasma, whose concentration falls with age (from ~200 ng/mL at age 20 to ~80 ng/mL at 60). It binds copper(II) with high affinity.

MECHANISM	Proposed: it delivers copper into the cell and, in fibroblasts and animal models, is associated with the expression of collagen, elastin and glycosaminoglycan genes, with inhibition of elastase and with angiogenesis. It does not act through a single classical receptor.
STUDIED FOR	Wound healing and turnover of the dermal matrix, collagen and elastin synthesis, antioxidant and anti-inflammatory activity, and cosmetic applications in skin ageing.
EVIDENCE	Relatively solid preclinical evidence (in vitro and animal) and some small topical studies in cosmetics. Evidence for systemic use is practically non-existent.
STATUS	In topical cosmetics it is used as an ingredient (INCI Copper tripeptide-1). As a lyophilised research material for systemic use it is not approved by the FDA or the EMA. RUO framework.
SAFETY	Research material, not approved for systemic administration. It supplies copper, an excess of which can be problematic.

CAS 89030-95-5 (complex) · 49557-75-7 (free peptide) · Gly-His-Lys, 3 aa · free C14H24N6O4, 340.38 g/mol · purity 99.75% · batch C3M9KQ

GLOW Blend

GHK-Cu + BPC-157 + TB-500

Multi-component mixture

A research mixture that combines the copper tripeptide GHK-Cu with two peptides studied in the context of tissue repair: BPC-157 and TB-500. It is not a single molecule but a formulation of several components.

MECHANISM	Proposed per component, not validated for the mixture: GHK-Cu and the dermal matrix via copper; BPC-157 with nitric oxide and VEGF / angiogenesis; TB-500 with actin dynamics and cell migration. No combined mechanism has been characterised.
STUDIED FOR	Tissue and skin repair, drawing together the lines of research for each component. The combination itself has no published research to support it.
EVIDENCE	No clinical evidence on the mixture. What is known comes from the components separately: solid preclinical evidence for GHK-Cu, and animal/cell evidence for BPC-157 and TB-500.
STATUS	Not approved. Its components BPC-157 and TB-500 are research materials (RUO) with no FDA or EMA approval and prohibited in sport (WADA).
SAFETY	A mixture of unapproved materials; the safety profile of the combination has not been characterised.

Combination without a single CAS · components: GHK-Cu, BPC-157, TB-500 · purity 99.60% · batch GLOW-062026-4

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

Multi-component mixture

Adds, to the GLOW components, the tripeptide KPV, derived from the C-terminal end of α-MSH. It thus combines a component studied in the anti-inflammatory context with the copper tripeptide and the repair peptides. It is not a single molecule.

MECHANISM	Proposed per component: KPV is associated with modulation of the NF-κB pathway and of melanocortin receptors, with fewer pro-inflammatory cytokines and without the pigmentary effects of α-MSH; the rest, as in GLOW. No combined mechanism characterised.
STUDIED FOR	Skin inflammation and tissue repair, adding the anti-inflammatory line of KPV to that of the other components. The combination has no research of its own.
EVIDENCE	No clinical evidence on the mixture. It comes from the individual components: preclinical for KPV and GHK-Cu, and animal/cell for BPC-157 and TB-500.
STATUS	Not approved. KPV is sold for research only; BPC-157 and TB-500 are RUO, unapproved and prohibited in sport (WADA).
SAFETY	A mixture of unapproved research materials; the safety profile of the combination has not been characterised.

Combination without a single CAS · adds KPV (CAS 67727-97-3) to the GLOW components · purity 99.76% · batch XX1EZC

› PMC — GHK Peptide as a Natural Modulator of Multiple Cellular Pathways in Skin Regeneration.

› PubMed — Regenerative and Protective Actions of the GHK-Cu Peptide (new gene data).

› PMC — Exploring the Role of Tripeptides in Wound Healing and Skin Regeneration.

› USADA / WADA — status of BPC-157 and TB-500 in sport.

RUO Apart from GHK-Cu as a topical cosmetic ingredient, these materials are not approved for human use. The mixtures have no clinical evidence of their own. BPC-157 and TB-500 are prohibited in sport (WADA). This is not medical advice.

Growth hormone (secretagogues) 09

Molecules studied for their ability to stimulate the release of growth hormone (GH) from the pituitary itself, rather than supplying external GH. They act on two axes: the GHRH receptor and the ghrelin receptor (GHS-R1a).

The growth hormone axis is one of the most studied neuroendocrine systems. Unlike recombinant GH, these peptides do not inject the hormone: they prompt the pituitary itself to release it, and are studied for their potential to reproduce the physiological pulsatile secretion. Two families coexist: the GHRH analogues (tesamorelin, CJC-1295 without DAC), which mimic the GH-releasing hormone, and the ghrelin-receptor agonists (ipamorelin), which act through a complementary pathway.

A recurring hypothesis is that combining both mechanisms would give a stronger stimulus than either alone; hence the combined formulations. This is a research hypothesis, not a demonstrated effect in humans. The level of evidence is very uneven: tesamorelin is the only member approved by the FDA, and only for visceral fat in HIV-associated lipodystrophy; ipamorelin reached phase 2 (postoperative ileus) without demonstrating efficacy; CJC-1295 without DAC is essentially preclinical.

The molecules in the category

Material	Family	Evidence	Status	Purity
Tesamorelin	GHRH analogue	●●●● Phase 3	Appr. (HIV)	99.93%
Ipamorelin	Ghrelin agonist	●●○○ Phase 2	RUO · WADA	99.90%
CJC-1295 without DAC + Ipamorelin	GHRH + ghrelin combo	●○○○ Preclinical	RUO	99.96%
Tesamorelin + Ipamorelin	GHRH + ghrelin combo	●○○○ No data	RUO	99.71%

Tesamorelin EGRIFTA · TH9507

GHRH analogue · approved (specific indication)

A stabilised synthetic analogue of human GHRH. It is the only GH secretagogue in this category with regulatory approval: the FDA approved it in 2010 (EGRIFTA) for a specific indication.

MECHANISM	Proposed: it binds to the GHRH receptor in the pituitary and stimulates the production and release of endogenous GH, which in turn raises IGF-1. In its indication, that increase is associated with greater lipolysis of visceral adipose tissue.
STUDIED FOR	Reduction of excess visceral abdominal fat in people with HIV and lipodystrophy (its only approved indication). In research it was explored in other metabolic contexts and hepatic steatosis, off-label.
EVIDENCE	High for its approved indication: phase 3 (more than 800 participants). It is the peptide with the most clinical evidence in the category.
STATUS	Approved by the FDA (2010; EGRIFTA SV/WR reformulations) for visceral fat in HIV-associated lipodystrophy. NOT approved by the EMA (the application was withdrawn in 2012). Outside that indication, not approved.
SAFETY	Contraindicated in pregnancy, active malignancy and disorders of the pituitary axis. Warnings: glucose intolerance / increased diabetes risk, fluid retention, oedema, arthralgia and carpal tunnel syndrome.

CAS 218949-48-5 · C221H366N72O67S · ~5135.86 g/mol · 44 aa · purity 99.93%

Ipamorelin NNC 26-0161

Selective ghrelin agonist (GHS-R1a)

A synthetic pentapeptide, described in 1998 as the first truly selective GH secretagogue. Originally developed by Novo Nordisk. Research material, not approved.

MECHANISM	Proposed: a selective agonist of the ghrelin receptor (GHS-R1a) in the anterior pituitary, a different pathway from GHRH; associated with an increase in plasma GH. It is described as selective because, at the doses studied, it does not significantly raise ACTH, cortisol, prolactin or other hormones.
STUDIED FOR	Stimulation of the GH axis; in preclinical work, weight gain and bone growth in rodents and gastrointestinal motility. It reached phase 2 for postoperative ileus.
EVIDENCE	Mixed: solid preclinical characterisation and some human data, but development for postoperative ileus was discontinued for lack of efficacy. Not approved.
STATUS	Not approved by the FDA or the EMA. Research peptide (RUO). Prohibited in sport (WADA, S2).
SAFETY	As it is not approved for human use, its long-term safety profile is not established.

CAS 170851-70-4 · C38H49N9O5 · ~711.9 g/mol · 5 aa · purity 99.90% · batch VNA7TB

CJC-1295 without DAC + Ipamorelin mod GRF 1-29 + ipamorelin

GHRH + ghrelin combination (research)

A research formulation that combines a GHRH analogue (CJC-1295 without DAC, or mod GRF 1-29) with a ghrelin-receptor agonist (ipamorelin). CJC-1295 without DAC has a short half-life (~30 min) because it lacks the albumin-binding DAC complex. Research material (RUO).

MECHANISM	Proposed, dual: CJC-1295 without DAC activates the GHRH receptor (via adenylate cyclase / cAMP) and ipamorelin acts in parallel on the ghrelin receptor. The hypothesis is that combining both pathways would give a stronger GH stimulus while preserving the pulsatile pattern; not demonstrated in clinical trials.
STUDIED FOR	Stimulation of the GH/IGF-1 axis and body composition in experimental models. The combination has not been characterised in formal clinical trials.
EVIDENCE	Low for the combination: essentially preclinical and theoretical. CJC-1295 without DAC has no approved clinical development.
STATUS	Not approved by the FDA or the EMA. Both components are research material (RUO); CJC-1295 without DAC is described as for laboratory use only.
SAFETY	A combination with no approval and no established long-term safety profile.

CJC-1295 without DAC: CAS 446262-90-4 · C152H252N44O42 · ~3367.9 g/mol · 29 aa · ipamorelin: CAS 170851-70-4 · purity 99.96%

Tesamorelin + Ipamorelin GHRH + ghrelin combo

GHRH + ghrelin combination (research)

Combines a GHRH analogue (tesamorelin) with a ghrelin agonist (ipamorelin). Although tesamorelin on its own is approved for a specific indication, this combination as such is not an approved product and is handled as research material (RUO).

MECHANISM	Proposed, dual: tesamorelin activates the GHRH receptor (GH release and a secondary rise in IGF-1) and ipamorelin the ghrelin receptor. The research idea is that the combination strengthens the stimulus more than either agent alone; this is a hypothesis, not a demonstrated effect in humans.
STUDIED FOR	Stimulation of the GH/IGF-1 axis and body composition, drawing on the mechanisms of its components. The combination has not been characterised in clinical trials.
EVIDENCE	Low for the combination. Solid clinical evidence exists only for tesamorelin in its approved indication; ipamorelin did not reach approval.
STATUS	The combination is not approved by the FDA or the EMA (RUO). The FDA approval applies only to tesamorelin on its own in its authorised indication.
SAFETY	No safety profile of its own. Bear in mind the warnings for tesamorelin (glucose, fluid retention, arthralgia) and its contraindications.

tesamorelin: CAS 218949-48-5 · ipamorelin: CAS 170851-70-4 · purity 99.71%

› Raun K. et al. (1998) — Ipamorelin, the first selective growth hormone secretagogue (Eur J Endocrinol).

› PubChem — Tesamorelin (CID 16137828) · FDA — EGRIFTA Prescribing Information.

› EMA — withdrawal of the marketing authorisation application for Egrifta (tesamorelin), 2012.

› Ishida J. et al. (2020) — Growth hormone secretagogues: history, mechanism and clinical development.

RUO Apart from tesamorelin in its approved indication (visceral fat in HIV-associated lipodystrophy), all the material in this category is research-grade (RUO), not approved for human use. Ipamorelin is prohibited in sport (WADA). The combinations are not approved products. This is not medical advice.

Libido and sexual function

10

Desire combines signals from the central nervous system (motivation) with peripheral vascular and hormonal responses. This category focuses on the brain's melanocortin system, a pathway distinct from that of the PDE5 inhibitors (such as sildenafil): it targets centrally driven desire rather than the mechanics of erection.

Sexual desire begins in the brain. Unlike the mechanics of erection or lubrication, which are largely vascular, motivation is handled in circuits of the central nervous system, among them the hypothalamus. That is where research interest in the peptides of this category is concentrated. The central piece is the melanocortin system: a family of POMC-derived messengers that act on five receptors (MC1R to MC5R). MC4R, abundant in the hypothalamus, has been linked to the regulation of sexual behaviour.

PT-141 (bremelanotide) is a synthetic cyclic heptapeptide, derived from an α-MSH analogue, studied as an agonist of these receptors with a preference for MC3R and MC4R. What sets it apart is that it crossed the line between the experimental and the regulated: in 2019 the FDA approved bremelanotide (brand name Vyleesi) for hypoactive sexual desire disorder in premenopausal women. That approval is specific to a product, an indication and a route; it does not extend to the EU or to the research-grade powder presentations (RUO). And the evidence is best read with nuance: the phase 3 effects were significant but modest in magnitude, with nausea and a transient rise in blood pressure.

The molecule in the category

Material	Class	Evidence	Status	Purity
PT-141 (bremelanotide)	Cyclic heptapeptide · melanocortin agonist	●●●● Phase 3	FDA drug RUO powder	99.91%

PT-141 (Bremelanotide) Vyleesi (approved drug) · PT-141

Melanocortin agonist (MC3R / MC4R)

A synthetic cyclic heptapeptide, related to melanotan II (it is its active metabolite). An α-MSH analogue studied on the central melanocortin system. Unlike the PDE5 inhibitors, its proposed mechanism is central (on desire) rather than vascular.

MECHANISM	Proposed: an agonist of melanocortin receptors, with greater potency at MC3R/MC4R than at MC1R. Activation of MC4R in the hypothalamus and limbic regions is thought to modulate the pathways of motivation and desire. The details of downstream signalling are not fully established.
STUDIED FOR	Hypoactive sexual desire disorder (HSDD) in premenopausal women (the approved drug's indication) and, in earlier studies, erectile and sexual dysfunction more broadly.
EVIDENCE	Mixed. For HSDD, phase 3 (the RECONNECT trials): significant differences versus placebo, modest in magnitude and with a high placebo effect. For other uses, more preliminary. The RUO presentations have no safety or efficacy backing for human use.
STATUS	The drug bremelanotide was approved by the FDA in 2019 (Vyleesi, autoinjector) for HSDD in premenopausal women. It has no current EMA authorisation. Research-grade PT-141 powder is RUO: not approved for human use and not equivalent to the medicine.
SAFETY	In trials: nausea (~40%), flushing (~20%), headache (~11%) and injection-site reactions. Transient rise in blood pressure: contraindicated with uncontrolled hypertension or cardiovascular disease. Repeated use may be associated with focal hyperpigmentation. The research product has not been evaluated for safety in humans.

CAS 189691-06-3 · C50H68N14O10 · ~1025.2 Da · cyclic heptapeptide (7 aa) · purity 99.91% · batch YGAFK7

› FDA — VYLEESI (bremelanotide) prescribing information (2019).

› PubChem — Bremelanotide (CID 9941379) · BOC Sciences (CAS 189691-06-3).

› PMC — Bremelanotide for HSDD: review of the RECONNECT trials.

RUO Bremelanotide is approved by the FDA only as a pharmaceutical product (Vyleesi), with a specific indication and route, and not by the EMA. Research-grade PT-141 powder is RUO, not approved for human use. This is not medical advice.

Tanning and melanocortin

Synthetic peptides that mimic alpha-melanocyte-stimulating hormone (α-MSH) and act on the melanocortin receptors (MC1R to MC5R). In the skin, MC1R regulates melanin synthesis; activation of other receptors touches appetite, sexual function and inflammation, which makes the field broad and delicate.

The melanocortin family grew out of the study of α-MSH, a natural peptide that binds to five G-protein-coupled receptors (MC1R to MC5R). Each serves distinct functions: MC1R, in the melanocytes, regulates melanin production, which is why melanocortin was studied in the context of pigmentation and photoprotection; MC4R and MC3R take part in appetite and sexual-behaviour circuits. Melanotan II is a cyclic heptapeptide that binds non-selectively to several of these receptors at once, and that lack of selectivity defines its profile.

It is worth understanding that the area is under investigation but unresolved. A close relative, afamelanotide (sometimes called Melanotan I), did reach approval in the US and the EU for a specific rare disease (erythropoietic protoporphyria), thanks to a more selective action on MC1R. Melanotan II, by contrast, has never gained regulatory approval in any jurisdiction: several health agencies (the FDA, Australia's TGA) have warned against unregulated products sold online. Everything documented here belongs to preclinical research and early human studies, not to an approved medicine.

The molecule in the category

Material	Class	Evidence	Status	Purity
Melanotan II	Cyclic heptapeptide · non-selective melanocortin agonist	●●○○ Preclinical / I	RUO · ● Warnings	99.81%

Melanotan II MT-II · MT-2

Non-selective melanocortin agonist

A synthetic analogue of α-MSH, more stable and compact than the natural hormone. Developed from the 1980s at the University of Arizona, as a successor to afamelanotide (Melanotan I). Research material, not approved for human consumption.

MECHANISM	Proposed: a non-selective agonist of the melanocortin receptors (MC1R, MC3R, MC4R, MC5R). Binding to MC1R is studied in relation to melanogenesis; central activation of MC3R/MC4R is associated with its systemic effects on appetite, sexual behaviour and autonomic responses. The exact order of affinity varies by source.
STUDIED FOR	The biology of pigmentation and photoprotection (via MC1R) and as a tool to study the melanocortin circuits linked to appetite and erectile function (via MC4R). Its commercial development was abandoned because of regulatory restrictions and concerns about promoting tanning.
EVIDENCE	Low to moderate depending on the outcome. Mostly preclinical and from some early phase I studies; it did not complete clinical development or reach the market. Much real-world usage information comes from case reports involving unregulated products.
STATUS	Not approved for human use in any jurisdiction (RUO). Several agencies (FDA, TGA) have warned against unregulated products; in several countries its supply without prescription is illegal. Its relative afamelanotide (Scenesse) is approved, but only for erythropoietic protoporphyria, not for tanning.
SAFETY	Documented: nausea, vomiting, facial flushing, yawning and loss of appetite (central melanocortin activation, MC4R), and prolonged erection (priapism) in men, which may require urgent care. Darkening of moles and the appearance of new ones. The link with melanoma has not been conclusively demonstrated. Unregulated products may contain impurities.

CAS 121062-08-6 · C50H69N15O9 · ~1024.18 g/mol · cyclic heptapeptide · purity 99.81% · batch MT210-052026-7

› PubChem / Wikipedia — Melanotan II (identity, CAS, non-selective agonism, regulatory status).

› DermNet NZ — Melanotan II (clinical and safety summary: priapism, moles).

› TGA (Australia) — warning against melanotan products · EMA — Scenesse (afamelanotide, for comparison).

RUO Melanotan II is not approved for human use in any jurisdiction (RUO) and several health agencies have warned against unregulated products. Do not confuse it with afamelanotide, approved only for a rare disease. This is not medical advice.

Metabolic and mitochondrial

12

Compounds studied for their action on the cell's energy metabolism, with the mitochondria and the redox cofactors at the centre. Interest concentrates on two levers: the energy sensor AMPK and NAD+ homeostasis. These are targets explored in obesity, insulin resistance and metabolic ageing, still mostly at the preclinical stage.

This area gathers compounds whose common denominator is not a chemical structure but a shared biology: how the cell produces, stores and spends energy. At the centre are the mitochondria and two master signals: AMPK, the sensor that switches on when energy runs low and reorients metabolism towards oxidation, and NAD+, the redox cofactor that connects the cell's state to its capacity to produce energy and whose availability falls with age and in obesity.

Its two representatives reach that biology by opposite routes. MOTS-c is a 16-amino-acid peptide encoded within the mitochondrial genome itself, described in 2015; it is studied as a messenger released with exercise that, according to the dominant hypothesis, activates AMPK, hence its description as an exercise mimetic. 5-Amino-1MQ is not a peptide but a small molecule that inhibits the enzyme NNMT, altering the availability of NAD+ and SAM. The field is far from settled: almost all the evidence is cellular and from rodents; MOTS-c has barely any direct human data and is prohibited by WADA; 5-Amino-1MQ has no published clinical trial at all.

The molecules in the category

Material	Class	Evidence	Status	Purity
MOTS-c	Mitochondrial peptide (16 aa) · AMPK activator	●●○○ Preclin. + observ.	RUO · WADA	–
5-Amino-1MQ	Small molecule · NNMT inhibitor	●○○○ Preclinical	RUO	99.94%

MOTS-c

Mitochondrial ORF of the 12S rRNA type-c

Mitochondrial peptide · AMPK activator

A short 16-amino-acid peptide encoded within the mitochondrial 12S ribosomal RNA gene, described in 2015. It belongs to the mitochondria-derived peptides (MDPs). It is detected naturally in the blood and rises with exercise. The research material is a synthetic peptide identical to the endogenous one (RUO).

MECHANISM	Proposed, not fully established: under metabolic stress it translocates to the nucleus and is associated with AMPK activation; in muscle it was proposed to interfere with the folate cycle, accumulating AICAR (an AMPK activator). In models it increases glucose uptake in a way reminiscent of exercise. Mechanisms under investigation, with no demonstrated benefit in people.
STUDIED FOR	Metabolic homeostasis, insulin sensitivity, diet-induced obesity, mitochondrial health and metabolic ageing. In humans the data are mostly observational (lower levels in diabetes and obesity; they rise with exercise).
EVIDENCE	Low to moderate. Mostly preclinical and observational. The only relevant human data come from an analogue (CB4211, CohBar; phase 1a/1b) that met the safety endpoint but did not separate from placebo on liver fat, and whose development was discontinued.
STATUS	Not approved by the FDA or the EMA. Prohibited by WADA since 1 Jan 2024 (section S4, hormone and metabolic modulators, as an example of an AMPK activator). It is distributed as research material.
SAFETY	The safety profile of MOTS-c administered in humans has not been characterised: no long-term studies. Research products may contain impurities. As a prohibited substance, its use carries consequences in sport.

CAS 1627580-64-6 · C101H152N28O22S2 · ~2174.62 g/mol · 16 aa · MT-RNR1 gene

5-Amino-1MQ

5-Amino-1-methylquinolinium · 5A1MQ

Small molecule · NNMT inhibitor (not a peptide)

A small-molecule inhibitor of the enzyme nicotinamide N-methyltransferase (NNMT). It is not a peptide; it is included for its shared metabolic biology. Characterised by Stan Watowich's group (Neelakantan et al., 2018). Research material (RUO).

MECHANISM	Proposed: it selectively and competitively inhibits NNMT, which methylates nicotinamide using SAM. By curbing that methylation, in models the nicotinamide becomes available to recycle towards NAD+, with a rise in intracellular NAD+ and SAM; in adipocytes it is associated with a shift in metabolism from storage towards oxidation. Mechanisms under investigation, with no demonstrated benefit in people.
STUDIED FOR	Diet-induced obesity, regulation of adipose-tissue metabolism, the biology of NAD+ and SAM, and the function of muscle stem cells. In models: adipocyte differentiation, lipogenesis and muscle regeneration in aged mice.
EVIDENCE	Low (preclinical). In vitro and rodents. There are no published clinical trials in humans: no phase 1 studies and no human pharmacokinetic data.
STATUS	Not approved by the FDA or the EMA, with no known active IND. It is sold only as a reagent or laboratory research material.
SAFETY	No human trials: its safety, toxicity and adverse effects in people are unknown; the data come from animal models. Research products may contain impurities.

CAS 42464-96-0 (iodide salt) · cation C10H11N2+ · ~159.21 g/mol (free) · NNMT IC50 ~1.2 µM · purity 99.94% · batch JRZTDB

› Lee C. et al. (2015) — The Mitochondrial-Derived Peptide MOTS-c (Cell Metabolism).

› USADA / WADA — MOTS-c prohibited since 2024 (S4, AMPK activators).

› Neelakantan H. et al. (2018) — NNMT inhibitors reverse diet-induced obesity in mice · Sigma-Aldrich SML2832.

RUO None is approved for human use (RUO). MOTS-c is prohibited by WADA (S4). 5-Amino-1MQ has no published clinical trials. All the evidence is preclinical. This is not medical advice.

Longevity

13

Compounds studied in relation to the mechanisms of cellular ageing: energy metabolism and the sirtuins (via NAD+), telomere maintenance and epigenetic regulation (pineal peptides). A well-characterised endogenous molecule sits alongside synthetic peptides whose evidence is mostly preclinical.

Ageing is not a single process but the sum of several mechanisms that deteriorate. This category brings together three very different compounds. NAD+ is a coenzyme the body produces naturally, central to metabolism and a substrate for the sirtuins (enzymes linked to DNA repair); its levels fall with age, hence the interest. Unlike the other two, it is a well-characterised endogenous molecule, although its exogenous administration lacks solid backing in large trials.

The other two are synthetic peptides developed at the St Petersburg Institute of Bioregulation and Gerontology (Vladimir Khavinson's group), the bioregulator peptides. Epitalon is a tetrapeptide (Ala-Glu-Asp-Gly) modelled on a pineal-gland extract, studied in relation to telomerase, epigenetics and melatonin. Pinealon is a tripeptide (Glu-Asp-Arg) studied for its possible neuroprotective action against oxidative stress. The evidence for both is mostly preclinical and comes largely from a single group; the human studies are small. Neither is approved as a medicine.

The molecules in the category

Material	Class	Evidence	Status	Purity
NAD+	Endogenous coenzyme (not a peptide)	●●○○ Endogenous	RUO / suppl.	99.85%
Epitalon	Tetrapeptide (pineal bioregulator)	●○○○ Preclinical	RUO	99.75%
Pinealon	Tripeptide (bioregulator)	●○○○ Preclinical	RUO	99.84%

NAD+ Nicotinamide adenine dinucleotide · β-NAD+

Endogenous coenzyme (not a peptide)

A coenzyme the body synthesises naturally, present in every cell. Central to energy metabolism (the NAD+/NADH pair) and a substrate for enzymes such as the sirtuins and the PARPs. Its availability declines with age. It is included for its relevance to longevity research, although by structure it is a dinucleotide, not a peptide.

MECHANISM	Proposed in longevity: a redox cofactor and obligatory co-substrate of the sirtuins, which deacetylate proteins by consuming NAD+ and are linked to metabolism, DNA repair and nucleus-mitochondria communication. The hypothesis is that its age-related decline reduces sirtuin activity; restoring it through precursors is studied as a possible route to counter that decline.
STUDIED FOR	Cellular ageing, sirtuin biology, mitochondrial metabolism and DNA repair. Its precursors (NMN, nicotinamide riboside) have been evaluated in early trials for their ability to raise blood NAD+.
EVIDENCE	Variable. The basic biology of NAD+ and the sirtuins is solidly established. Exogenous administration as an anti-ageing strategy has limited evidence (pilot studies, early trials with precursors), with no large pivotal trials. Commercial use (IV drips) outpaces the evidence.
STATUS	Not approved as a medicine for longevity indications. As an exogenous compound it is sold within a research framework (RUO) or as a supplement/infusion, without FDA or EMA approval to treat disease.
SAFETY	IV infusion is offered with limited evaluation of safety and efficacy (small pilot studies). Products not regulated as medicines: possible variability in quality. Discomfort reported during rapid infusion.

CAS 53-84-9 · C21H27N7O14P2 · ~663.43 g/mol · dinucleotide · purity 99.85% · batch J4Q90F

Epitalon Epithalon · AEDG (Ala-Glu-Asp-Gly)

Tetrapeptide (pineal bioregulator)

A short synthetic peptide developed in Russia (Khavinson's group) as a synthetic version of the pineal complex epithalamin. It belongs to the bioregulator peptides. Research material (RUO), not approved.

MECHANISM	Proposed: in cultures and human lymphocytes, activation of telomerase (TERT/TERC) with telomere elongation was described; also an influence on melatonin synthesis, epigenetic effects through DNA/histone binding and antioxidant activity. The literature notes that the mode of action is not fully clarified. Research hypotheses, not confirmed in humans.
STUDIED FOR	Geroprotection and ageing, telomere maintenance, circadian rhythms and melatonin, and, exploratorily, retinitis pigmentosa, tumour inhibition in rodents and cognitive function.
EVIDENCE	Low. Mostly preclinical (cultures and animal models) and drawn largely from a single Russian group. In humans, only two small studies. Insufficient for regulatory approval.
STATUS	Not approved in any jurisdiction (RUO). No FDA or EMA approval. Reviews note that short- and long-term toxicity studies would be essential before any approval.
SAFETY	A significant safety gap: no genotoxicity, carcinogenicity or interaction studies in humans. Short peptides tend to be unstable in vivo. Possible variability in quality.

CAS 307297-39-8 · C14H22N4O9 · ~390.35 g/mol · 4 aa (AEDG) · purity 99.75%

Pinealon EDR (Glu-Asp-Arg)

Tripeptide (ultrashort bioregulator)

A short synthetic peptide developed in Russia (Khavinson's group), documented as a geroprotector and bioregulator. Research material (RUO), not approved.

MECHANISM	Proposed: owing to its small size, it is hypothesised to cross the cell and nuclear membranes and interact directly with DNA and histones. In models it is credited with suppressing reactive oxygen species and inhibiting ERK1/2, with a possible protective effect against oxidative stress. Research hypothesis, not confirmed.
STUDIED FOR	Neuroprotection and ageing of the central nervous system, cell viability under oxidative stress and hypoxia, and regulation of gene expression. In animal models: prenatal hyperhomocysteinaemia, learning in experimental diabetes, cortical serotonin expression.
EVIDENCE	Low. Mostly preclinical (in vitro and rodents), with no controlled clinical trials in humans, and drawn largely from the same Russian group.
STATUS	Not approved in any jurisdiction (RUO). Sources indicate it lacks legal status and an ATC code. No FDA or EMA approval.
SAFETY	No safety studies in humans: toxicity, interactions and long-term effects unknown. Possible variability in quality; short peptides are unstable in vivo.

CAS 175175-23-2 · C15H26N6O8 · ~418.4 g/mol · 3 aa (EDR) · purity 99.84%

› PubChem — NAD+ (CID 5892) · Imai & Guarente — NAD+ and sirtuins in the control of ageing.

› PMC — Overview of Epitalon (pineal tetrapeptide) · PubChem — Epitalon (CAS 307297-39-8).

› Wikipedia / PMC — Pinealon (EDR): identity and gene-expression mechanism.

RUO NAD+ is an endogenous molecule; as an exogenous compound it is not an approved drug. Epitalon and Pinealon are research peptides (RUO) with mostly preclinical evidence, not approved for human use. This is not medical advice.

Antioxidant

14

Molecules studied for their interaction with the cell's redox balance: the equilibrium between the generation of reactive oxygen species (ROS) and the systems that neutralise them. The protagonist is an endogenous tripeptide, glutathione, the central piece of the intracellular antioxidant defence.

Energy production in the mitochondria generates, as an inevitable by-product, reactive oxygen species: unstable molecules that, in excess, oxidise membranes, inactivate enzymes and damage DNA. Against that threat, the body deploys a network of defences, and at its centre is glutathione, the most abundant non-protein thiol in animal tissues. L-glutathione is a tripeptide (glutamate, cysteine and glycine); the key lies in the thiol group of the cysteine, which donates electrons and neutralises free radicals. It acts directly (neutralising oxidants) and indirectly (as a cofactor for enzymes such as the glutathione peroxidases). The swing between reduced (GSH) and oxidised (GSSG) glutathione is the cell's redox buffer.

The evidence is best read carefully. At the laboratory level, glutathione's role in redox homeostasis is textbook biochemistry. The debated question is supplementation: oral glutathione has limited bioavailability because it is broken down in the digestive tract, and trials on whether raising its levels translates into functional effects have given mixed results, often in small studies. Framework: oral glutathione is sold as a supplement, not as a medicine; its injectable use (above all for cosmetic skin-lightening purposes) is not approved and prompted FDA warnings over contamination risks. As a research input it is RUO.

The molecule in the category

Material	Class	Evidence	Status	Purity
L-Glutathione (GSH)	Endogenous tripeptide (antioxidant thiol)	●●○○ Endogenous	Suppl. / RUO	99.73%

L-Glutathione

Reduced glutathione · GSH · γ-Glu-Cys-Gly

Endogenous tripeptide (antioxidant thiol)

The most abundant non-protein thiol in animal cells, present in almost all tissues at millimolar concentrations. A tripeptide (glutamate, cysteine and glycine) that the body synthesises in two enzymatic steps. It coexists in reduced (GSH) and oxidised (GSSG) forms; their ratio defines the cellular redox state.

MECHANISM	Proposed: antioxidant by two routes. Directly, the thiol group (-SH) of its cysteine donates electrons and neutralises ROS. Indirectly, it is a cofactor for antioxidant and detoxification enzymes (glutathione peroxidases, S-transferases, glyoxalases). GSH is oxidised to GSSG, which glutathione reductase regenerates using NADPH. This is biochemistry, not a claim of therapeutic benefit.
STUDIED FOR	Oxidative stress and redox homeostasis: antioxidant defence, detoxification, and models linked to cardiovascular, metabolic, infectious and neurological disease. In oral supplementation, whether it raises systemic levels and with what consequences is studied, with no conclusive results. In dermatology, controversially, its relationship with pigmentation.
EVIDENCE	The physiological role is solidly established at the biochemical level. The evidence for supplementation in humans is uneven: oral bioavailability is low owing to digestive breakdown, and trials give mixed results, often in small samples. Efficacy for specific indications (skin lightening) is considered insufficient.
STATUS	It is not an approved drug for treating disease. In the US it has GRAS notifications for food uses and is sold orally as a supplement (DSHEA). Injectable preparations do not qualify as a supplement and are not approved; in 2019 the FDA warned against compounding injectables with dietary-grade glutathione after adverse events from contamination. As a lyophilised research material it is RUO.
SAFETY	Although it is endogenous, its administration in unapproved forms (in particular injectables) is not endorsed and carries risks: in 2019 the FDA documented adverse events from endotoxins in injectables compounded with dietary glutathione (ranging from nausea to respiratory difficulty and at least one hospitalisation).

CAS 70-18-8 (GSH) · C10H17N3O6S · ~307.32 g/mol · 3 aa (γ-Glu-Cys-Gly) · purity 99.73%

› The antioxidant glutathione (PubMed 36707132) · Glutathione and Glutaredoxin in Cellular Redox Homeostasis (PMC).

› PubChem CID 124886 — Glutathione (identity, formula).

› FDA — concerns with dietary glutathione to compound sterile injectables (2019).

RUO Glutathione is an endogenous molecule; orally it is sold as a supplement, not as a medicine. Its injectable use is not approved and prompted FDA warnings. As a research input it is RUO, not suitable for human consumption. This is not medical advice.

Cognitive and nootropic

15

Peptides studied for their action on the central nervous system, in particular the pathways that regulate neuronal plasticity, attention, memory and the stress response. These are short peptides derived from fragments of hormones or endogenous peptides, modified to resist degradation. The research combines preclinical work with mostly Russian clinical trials of limited scope.

The brain depends on a fine balance between signals that stimulate and calm it, and on the plasticity of its neurons. Much cognitive research revolves around the neurotrophins (BDNF, NGF), which sustain neuronal survival and synapse formation, and around the neurotransmission systems (dopaminergic, serotonergic, GABAergic). The two peptides in this category, both of Russian origin, share a chemical strategy: take an active fragment of an endogenous molecule and add a stabilising Pro-Gly-Pro tail that protects it from enzymes.

Semax derives from a fragment of adrenocorticotropic hormone (ACTH 4-7, extended with Pro-Gly-Pro), with no hormonal activity on the adrenal axis, and is studied for its proposed effect on the expression of BDNF and NGF. The variant we document is the N-acetyl amidated one. Selank derives from tuftsin (a fragment of immunoglobulin G) and is studied as an anxiolytic with a proposed non-benzodiazepine mechanism. Both are registered as medicines in Russia, but the clinical research is limited in scope, with small samples and little independent replication. Neither is approved by the FDA or the EMA.

The molecules in the category

Material	Class	Evidence	Status	Purity
Semax (N-acetyl amidate)	Heptapeptide derived from ACTH 4-7	●●○○ Russian clinical	Reg. Russia RUO	99.11%
Selank	Heptapeptide tuftsin analogue	●●○○ Russian clinical	Reg. Russia RUO	99.74%

Semax (N-acetyl amidate) Ac-Met-Glu-His-Phe-Pro-Gly-Pro-NH_2

Neuropeptide heptapeptide (derived from ACTH 4-7)

A synthetic heptapeptide developed in Russia from the 4-7 fragment of ACTH (the portion associated with that hormone's nootropic activity), with a stabilising Pro-Gly-Pro tripeptide. It retains neurotrophic effects but, according to the literature, without hormonal activity on the adrenal axis. The catalogue variant is the N-acetyl amidated one, with modifications intended to improve stability.

MECHANISM	Proposed, neurotrophic and neuromodulatory: it increases the expression (mRNA) of BDNF and NGF (and in some work VEGF) in the hippocampus and frontal cortex, linked to synaptic plasticity; also modulation of the dopaminergic and serotonergic systems. Mostly from cell and animal models, not fully confirmed in humans.
STUDIED FOR	Cognitive function (attention, memory) and neuroprotection. The most developed clinical line (Russian) is ischaemic stroke and neurological recovery; also cognitive decline and optic-nerve disorders.
EVIDENCE	Mixed, with limitations. Clinical research (Russian) in ischaemic stroke and abundant preclinical work. Small-sample studies, in Russian and with little independent replication. The N-acetyl amidated variant has very scarce specific human data; much of the evidence is for the base Semax.
STATUS	Not approved by the FDA or the EMA. In Russia, Semax (base form) is registered as a medicine (intranasal drops) and on the list of essential medicines; that national registration does not equate to FDA/EMA approval or to proof of safety/efficacy under those standards. Outside Russia, RUO.
SAFETY	Its safety profile, interactions and long-term behaviour are not established by Western standards, especially for the N-acetyl amidated variant.

CAS 2920938-90-3 (N-acetyl amidate) · $C_{39}H_{54}N_{10}O_{10}S$ · ~854.97 g/mol · 7 aa · base Semax CAS 80714-61-0 · purity 99.11% · batch SEMX10-042026-3

Selank TP-7 · tuftsin analogue · Thr-Lys-Pro-Arg-Pro-Gly-Pro

Heptapeptide tuftsin analogue

A synthetic heptapeptide from the Institute of Molecular Genetics (Russian Academy of Sciences), based on tuftsin (an immunomodulatory tetrapeptide, a fragment of immunoglobulin G), with a stabilising Pro-Gly-Pro tail. It is studied for its proposed anxiolytic and nootropic properties.

MECHANISM	Proposed, multifactorial and non-benzodiazepine (it does not bind directly to the benzodiazepine site). On the GABAergic system the evidence is nuanced: in vitro, on its own it did not alter the expression of GABA-A receptor subunits but attenuated GABA-induced changes, interpreted as indirect modulation. Also an influence on enkephalin metabolism, monoaminergic systems and BDNF expression. From cell and animal models, not confirmed in humans.
STUDIED FOR	Anxiety (generalised anxiety disorder, asthenic conditions) and cognitive function, with reports of an absence of sedation and of cognitive impairment. In preclinical work: enkephalins, monoaminergic systems, GABAergic gene expression.
EVIDENCE	Limited. The most cited clinical evidence comes from small-sample Russian trials (e.g. a comparison with medazepam in generalised anxiety), with limited methodological reporting and mostly in Russian. No large-scale controlled trials by Western standards.
STATUS	Not approved by the FDA or the EMA. In Russia it is registered as a prescription anxiolytic/nootropic medicine (intranasal form); that registration does not equate to FDA/EMA approval. Outside Russia, RUO.
SAFETY	Human safety data are limited and mostly Russian; the interaction profile and prolonged use are not established by Western standards.

CAS 129954-34-3 · C33H57N11O9 · ~751.9 g/mol · 7 aa · purity 99.74%

› Semax regulates BDNF/trkB in the rat hippocampus · Semax activates the dopaminergic and serotonergic systems (PubMed).

› PubChem — N-acetyl semax amidate (CAS 2920938-90-3) · Cayman — base Semax (CAS 80714-61-0).

› Filatova et al. (Frontiers, 2017) — GABA and Selank in GABAergic neurotransmission · PubChem — Selank.

RUO Semax and Selank are registered as medicines only in Russia, with clinical evidence of limited scope; they are not approved by the FDA or the EMA. Outside Russia they are research material (RUO), not suitable for human consumption. This is not medical advice.

Anti-inflammatory

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Peptides studied for their proposed ability to modulate the pathways that trigger and sustain inflammation, in particular the transcription factor NF-κB and the MAP-kinase cascades. Several derive from endogenous hormones and are studied for their action inside the epithelial or immune cell. The research is mostly preclinical.

Inflammation is an essential response: the body uses it to defend and repair. The problem arises when it becomes chronic or dysregulated (inflammatory bowel disease, dermatitis, tissue in a persistent inflammatory state). At the centre is NF-κB, a transcription factor that acts as a master switch: when activated, it travels to the nucleus and switches on the production of pro-inflammatory cytokines. Together with the MAP-kinases, it is one of the most studied targets.

The best-studied case in this category is KPV, a tripeptide (lysine-proline-valine) that corresponds to the C-terminal 11-13 fragment of α-MSH. What is interesting is that, in preclinical models, it appears to retain much of the parent hormone's anti-inflammatory activity without its pigmentary effect, with an action that would not depend on the melanocortin receptors: the peptide is thought to enter the cell and interfere with inflammatory signalling. The bulk of the data comes from in vitro studies and animal models of colitis; there are no human clinical trials. It is framed as RUO.

The molecule in the category

Material	Class	Evidence	Status	Purity
KPV	Tripeptide (α-MSH 11-13 fragment)	●○○○ Preclinical	RUO	99.75%

KPV Lys-Pro-Val · MSH(11-13) · α-MSH 11-13 fragment

Melanocortin-derived tripeptide

A tripeptide (lysine, proline, valine) that corresponds to residues 11-13 of the C-terminal end of α-MSH. It is studied as a minimal fragment that, in preclinical models, would retain the parent hormone's anti-inflammatory activity without its effect on pigmentation.

MECHANISM	Proposed, intracellular: according to the data, its anti-inflammatory action would not depend on the melanocortin receptors. The peptide enters the cell (in the intestinal epithelium, via the PepT1 transporter) and is thought to interfere with pro-inflammatory pathways, in particular the activation of NF-κB and the MAP-kinases (ERK/p38), reducing pro-inflammatory cytokines. From cell and animal models, not confirmed in humans.
STUDIED FOR	Modulation of inflammatory processes. The most developed line: preclinical models of intestinal inflammation (DSS and TNBS colitis in mice), uptake by PepT1 and inflammatory markers. Also models of skin inflammation (keratinocytes) and topical/transdermal administration.
EVIDENCE	Preclinical. In vitro (epithelial and immune cells) and animal models. There are no human clinical trials to support therapeutic uses.
STATUS	Not approved for human use. RUO framework. It is not a medicine approved by the FDA, the EMA or other agencies for any indication.
SAFETY	As there are no human clinical trials, its safety profile, dose and interactions in people are not established.

CAS 67727-97-3 · C16H30N4O4 · ~342.43 g/mol · 3 aa (Lys-Pro-Val) · purity 99.75%

› Dalmasso et al. (2008) — PepT1-Mediated Tripeptide KPV Uptake Reduces Intestinal Inflammation (Gastroenterology).

› PubChem CID 125672 — MSH(11-13) (KPV) · Ghazvini et al. (2025) — anti-inflammatory peptides in IBD (review).

RUO KPV is research material (RUO), not approved for human use. The evidence is preclinical, with no clinical trials. This is not medical advice.

Immune

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Peptides studied for their ability to modulate the immune system: maturation and activation of T lymphocytes, the activity of NK and dendritic cells, and the balance of cytokines. These are immunomodulatory molecules that turn the defensive response up or down. A particular case: here a real clinical development with approval in several countries sits alongside, in many other markets, distribution only as research material (RUO).

The immune system works like an orchestra: it needs each section to come in at exactly the right moment, and to know when to fall silent too. When that coordination goes awry (chronic infection, immunosuppression, ageing of the thymus), the response is weak where it should be firm or runs wild where it should be restrained. Immunomodulation studies how to restore the timing. The thymus is the gland where T lymphocytes mature; in the 1970s the thymosins were isolated from its extracts. One of them, thymosin alpha-1, is a 28-amino-acid fragment of prothymosin alpha, and it makes up this category.

It is credited with an immunomodulatory mechanism: it would act as an agonist of Toll-like receptors (TLR2, TLR9) on dendritic and myeloid cells, favouring the maturation of T lymphocytes, the activation of NK cells and a recalibration of the cytokines. That is why it is studied where defence is compromised: hepatitis B and C, immunosuppression from chemotherapy, sepsis, as a vaccine adjuvant and, with controversial results, in COVID-19. The level of evidence differs from the rest: it has real clinical development and is approved as a medicine (Zadaxin) in more than thirty countries for hepatitis and as an adjuvant. But it is not approved by the FDA or the EMA, and the strength of the evidence varies greatly by indication. Where it is not an approved drug, it is distributed only as RUO.

The molecule in the category

Material	Class	Evidence	Status	Purity
Thymosin alpha-1 (thymalfasin)	Immunomodulatory thymic peptide (28 aa)	■■■□ Clinical	30+ countries; RUO	99.65%

Thymosin alpha-1

Thymalfasin · Tα1 · Zadaxin (brand) · thymalfasin (INN)

Immunomodulatory thymic peptide (28 aa, N-acetylated)

A synthetic 28-amino-acid peptide identical to a portion of human prothymosin alpha, isolated in 1977 from calf thymus extracts. As thymalfasin (brand name Zadaxin) it is used as an immunomodulator in several countries; where it is not approved, it is distributed as research material (RUO).

MECHANISM	Proposed: an agonist of Toll-like receptors (above all TLR2 and TLR9) on dendritic and myeloid cells, activating pathways such as MyD88, IRF3/NF-κB and p42/44 MAPK/JNK. Downstream it is associated with maturation of T lymphocytes (CD4+ and CD8+), activation of NK cells and changes in the cytokine profile (IFN-α, IFN-γ, IL-2, IL-12). It is an immunomodulatory effect, not indiscriminate immune stimulation. Mechanism under study, with no proven benefit.
STUDIED FOR	Immune modulation in chronic hepatitis B and C (alone or with interferon), immunosuppression from chemotherapy and certain cancers, sepsis, and as a vaccine adjuvant in immunocompromised people. Also COVID-19 and immune reconstitution. Describing these lines does not assert benefits outside the indications approved in each country.
EVIDENCE	Within this encyclopaedia, one of the better-supported: real clinical development, human trials and approval as a drug in 30+ countries for hepatitis B/C and as an adjuvant. The strength varies by indication: more consistent in hepatitis B/C; heterogeneous in sepsis and oncology; mixed and controversial in COVID-19 (meta-analyses with no significant effect on mortality).
STATUS	Heterogeneous. Approved (Zadaxin) in 30+ countries (China 1995, Argentina, Mexico, Peru…) for hepatitis B/C and as an adjuvant. NOT approved by the FDA (only orphan-drug designations, which are not approval) or by the EMA (orphan designation for hepatocellular carcinoma, with no marketing authorisation). In the US it was not cleared for compounding (PCAC voted against, Dec 2024). Where it is not an approved drug, it is sold only as RUO.
SAFETY	In approved clinical use it is generally described as well tolerated, with mild local adverse effects. Caution in immunosuppressed people. The established safety profile relates to the pharmaceutical product under medical supervision; RUO materials may vary in purity and are not intended for human use.

CAS 62304-98-7 (thymalfasin) · C129H215N33O55 · ~3108.3 g/mol · 28 aa (N-acetylated) · purity 99.65%

› Thymosin alpha 1: a comprehensive review (PMC7747025) · Tα1 in viral diseases (PMC, Molecules 2023).

› Thymosin alpha 1 in COVID-19: systematic review and meta-analysis (PMC9754924).

› PubChem — Thymalfasin (CAS 62304-98-7) · FDA — Tα1 bulk drug substances (503A evaluation).

RUO Thymosin alpha-1 is approved as a medicine (Zadaxin) in more than thirty countries, but NOT by the FDA or the EMA. Where it is not an approved drug it is distributed only as research material (RUO), not suitable for human consumption. This is not medical advice.

Hormonal

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The reproductive hormonal axis (hypothalamus-pituitary-gonads) regulates the sex hormones and reproductive function. The molecules in this category act as gonadotropins or analogues of the signals the pituitary sends to the gonads, and are studied for how they modulate testosterone synthesis or ovulation.

The hypothalamic-pituitary-gonadal axis governs the production of sex hormones. The hypothalamus releases GnRH, the pituitary responds with two gonadotropins (LH and FSH), and these regulate testosterone in the testis and ovulation in the ovary. The protagonist is HCG (human chorionic gonadotropin), a glycoprotein hormone produced by the placenta during pregnancy. It shares the alpha subunit with LH, FSH and TSH, and its own beta subunit lets it bind to the same receptor as LH (LHCGR): that is why it behaves as an agonist of the LH signal. In men it stimulates the Leydig cells to produce testosterone; in women it takes part in triggering ovulation.

Unlike many research peptides, HCG has a long pharmacological history and has FDA-approved indications (infertility, prepubertal cryptorchidism, selected hypogonadotropic hypogonadism). But around it lies an area of unapproved uses, above all weight loss (the HCG diet): the FDA itself states that HCG has no known effect on fat mobilisation, appetite or fat distribution, and considers over-the-counter weight-loss products fraudulent and illegal, with reports of serious adverse events. In sport it is prohibited (WADA, S2) in men. Any presentation labelled as a research peptide is RUO.

The molecule in the category

Material	Class	Evidence	Status	Purity
HCG (chorionic gonadotropin)	Glycoprotein hormone · LH/CG receptor agonist	●●●● Appr. (indic.)	Approved ● WADA	–

HCG (human chorionic gonadotropin) hCG · Pregnyl · Novarel · Ovidrel (recombinant)

Glycoprotein hormone · LH/CG receptor agonist

A glycoprotein hormone produced by the placenta during pregnancy, where it sustains the corpus luteum and progesterone in the first weeks. As a drug it exists in urine-derived form (Pregnyl, Novarel) and recombinant form (choriogonadotropin alfa, Ovidrel). It is the hormone that pregnancy tests detect. It is not a synthetic peptide but a native hormone with alpha and beta subunits.

MECHANISM	An agonist of the shared LH and HCG receptor (LHCGR, a GPCR). It activates the Gαs → adenylate cyclase → cAMP → PKA pathway, with phosphorylation of ERK1/2, associated with an increase in steroidogenic enzymes. In men it stimulates the Leydig cells to produce testosterone (mimicking LH); in women, in its approved use, it substitutes for the LH surge to trigger ovulation.
STUDIED FOR	Within the FDA-approved framework: prepubertal cryptorchidism, selected hypogonadotropic hypogonadism in men, and induction of ovulation in selected anovulatory women. Outside that framework: recovery of the gonadal axis after anabolic steroids (off-label, limited evidence). For weight loss (the HCG diet) it was studied and did not demonstrate efficacy.
EVIDENCE	Mixed depending on the context. High for its approved indications (infertility, cryptorchidism, selected hypogonadism). None or negative for weight loss: the FDA states there is no substantial evidence that it increases weight loss beyond caloric restriction. Post-steroid use is off-label, with limited evidence.
STATUS	A hormone with FDA-APPROVED indications (ATC G03GA01; recombinant G03GA08), prescription-only (Pregnyl, Novarel, Ovidrel). The FDA states it is NOT approved for weight loss and that OTC products (drops, sprays, homeopathic) for slimming are fraudulent and illegal. Outside its indications, off-label. Any presentation as a research peptide is RUO. Prohibited in sport (WADA, S2, in men).
SAFETY	Contraindicated in precocious puberty and in prostate carcinoma or other androgen-dependent neoplasms (and, in women, primary ovarian failure, hormone-dependent tumours, pregnancy). Warnings: in men it may induce precocious puberty; in ovulation induction, a risk of ovarian hyperstimulation syndrome, multiple pregnancy and thromboembolism. The FDA linked its use for slimming to serious adverse events.

CAS 9002-61-3 (native) · 177073-44-8 (recombinant) · heterodimeric glycoprotein · ~36.7 kDa · α subunit 92 aa + β ~145 aa

› FDA — Prescribing Information for Pregnyl / Novarel / Ovidrel (indications, contraindications, OHSS).

› Casarini et al. (2016) — LH vs hCG signalling in Leydig cells (Reprod Biol Endocrinol).

› FDA — Questions and Answers on HCG Products for Weight Loss (not approved; OTC products illegal).

RUO HCG is a drug approved for specific reproductive indications, but NOT for weight loss (the FDA considers OTC slimming products fraudulent). It is prohibited in sport (WADA). The research presentations (RUO) are not intended for human use. This is not medical advice.

Part III

Takeaways

The verification method on a single page, a glossary to keep the technical jargon clear, and how this atlas was built.

How to verify any peptide

The category is irrelevant: the quality standard is the same. Five checks, and if one fails, you stop.

1 Batch

The COA corresponds to the exact batch you will receive, not to a generic code.

2 Identity

The mass by LC-MS matches the molecular weight of the declared peptide.

3 Purity

HPLC ≥98%, with a clean, dominant main-peak chromatogram.

4 Laboratory

Independent, identifiable and externally verifiable.

5 Clinical judgement

You verify the quality yourself; use and monitoring belong to a licensed professional.

Rule of thumb

If a peptide cannot be independently verified, treat it as unaccredited. Without that foundation, no mechanism and no line of research changes the decision.

Methodology and sources

How this atlas was built and under what criteria, so you can trust what you read and check it against the sources.

Each category was documented from scientific literature and official sources, and reviewed independently before being included. The identity data (CAS, formula, molecular weight) and purity data come from the certificates of analysis of the reference catalogue. Where the evidence is limited or preliminary, this is stated explicitly rather than rounded up towards a conclusion.

Principal sources

› World Health Organization (WHO) — falsified and substandard medical products.

› European Medicines Agency (EMA) and FDA — regulatory status and product information.

› PubMed / peer-reviewed literature — mechanisms and preclinical and clinical studies.

› DrugBank and ClinicalTrials.gov — molecular identity and trial status.

The sources specific to each molecule appear at the foot of its category.

About this atlas

Educational material prepared and reviewed by the scientific review team at Peptide Pens Europe. We do not sell peptides: that is why we can describe the evidence as it stands, without promises. The aim is for you to understand the field and decide with sound judgement, always alongside a healthcare professional.

Author: Scientific review team · Equipo editorial · 2026 edition · Last updated: June 2026 · Research framework (RUO).

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Understand the molecule. Verify the quality. Decide with a doctor.

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