ExeLabs

RESEARCH MATERIALS GUIDE · 2026 EDITION

The Standard.

How to verify a material before you believe anything about it. Six families, twenty-seven materials, and the evidence in its place — including that of our own catalog.

EXELABS · WE DON'T PROMISE RESULTS. WE RUN A STANDARD.

RESEARCH USE ONLY

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Guide *map*

Four parts. The method before the catalog, because without verification nothing else holds up.

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BEFORE YOU BEGIN

What this guide is — *and isn't*

A working reference for understanding what each research material is, where its evidence actually stands, and, above all, how to check that what's in front of you is what it claims to be.

It's written backward from how these documents are usually written. Most open with the catalog and leave verification for an appendix at the end. Here the method comes first, because it's the one part that doesn't depend on the molecule: if a material can't be verified, no mechanism and no research line changes the decision. Everything else comes after.

The organization is also different. Instead of splitting the materials into thirteen effect categories — a taxonomy that follows commercial promise more than biology — we group them into **six families** by the system they act on. Some molecules move relative to how they're usually classified; when they do, we explain why.

What you won't find

No doses, no protocols, no promises of results. Not because we're short on space, but because these materials are **not intended for human consumption** and there is no reference dose we could legitimately offer. Any supplier who does give you one is stepping outside the framework they claim to operate in.

You also won't find claims of benefit. When we say a material "is studied in the context of" something, we're describing a line of research, not a proven effect in people. It's a distinction that runs through the entire document and is worth keeping in mind on every card.

CONFLICT-OF-INTEREST STATEMENT

ExeLabs distributes several of the materials described in this guide. We are not an independent observer, and we won't pretend to be. Precisely for that reason, every claim is documented with public, verifiable sources, and we flag just as clearly when the evidence is preclinical only — even for something in our own catalog. Cross-check what you read. It's the only sensible way to use a document like this one.

FRAMEWORK

Educational document within a research (RUO) framework. It does not sell products, does not recommend uses or doses, and does not constitute medical advice. Many of the compounds described are not approved for human use, and some are listed as prohibited substances in sport (WADA). Always consult a healthcare professional.



PART I

The *method*

First, not last. What a COA certifies, how to read it field by field, the five checks that aren't negotiable, and the eight signals that should make you stop.

01 **Why verification comes first**

02 **The COA, field by field**

03 **The five checks**

04 **Eight red flags**

Why verification *comes first*

Before asking what a molecule does, there is a prior question, and it's the one almost nobody asks: is this really the molecule it claims to be, and is it clean?

Recognition between a peptide and its receptor depends on shape. The exact order of amino acids defines the molecule's identity and its behavior. A wrong sequence, an impurity, or a poorly synthesized fragment might not fit the receptor, might fit a different one, or might behave unpredictably.

That makes identity and purity the foundation everything else rests on. It doesn't matter how elegant the proposed mechanism is or how promising a research line looks: if the material isn't what it's declared to be, all that reasoning applies to something else. That's why this guide starts here and not in the catalog.

The two questions are answered by two different techniques, and it's worth not confusing them. **Mass spectrometry** (LC-MS) measures the sample's molecular mass and compares it to the theoretical mass of the declared peptide: it answers "is this it?". **Liquid chromatography** (HPLC) separates the components and measures what proportion corresponds to the peptide versus impurities: it answers "how much of this is it?". A material can have correct identity and mediocre purity, or the reverse.

> WHAT A COA DOESN'T TELL YOU
>
> A certificate of analysis certifies **analytical quality, not safety in people**. A material can be 99.9% pure and still be an unapproved compound with no established human safety profile. Those are two different questions, and the paper only answers one of them.

This guide's evidence scale

To avoid repeating phrases like "limited evidence" on every card — which mean different things depending on who writes them — we use a five-level scale, applied with the same criteria to every material, including our own.

E0	**No data**	No specific research on this material as sold.
E1	**Preclinical**	In vitro and animal models. No result has been confirmed in people.
E2	**Limited clinical**	Small, early, or poorly replicated human studies.
E3	**Extensive clinical**	Phase 3 programs or extensive clinical development, without full approval.

E4	**Approved**	Authorized by a regulatory agency for a specific indication.

HOW TO READ IT

The level describes the **state of the research**, not the material's quality or how interesting it is. An E1 can be a perfectly synthesized compound that's heavily studied in the lab; what E1 says is that none of that has been confirmed in people. And an E4 doesn't mean the catalog version is approved: approval belongs to the drug, not to the research powder.

The COA, *field by field*

A certificate of analysis is a short document. These are its fields and what you should check in each before accepting it as valid.

1 **Product and synonyms**

The molecule's name and its aliases. Cross-check it against the CAS number: the trade name can vary, the CAS cannot.

2 **Lot number**

The link between the paper and the vial. Without an exact match to the label, the rest of the document is irrelevant.

3 **Analysis date**

A COA dated long before the lot, or with no date, tells you nothing about what was actually analyzed.

4 **Formula and molecular weight**

The theoretical value the mass measurement is compared against.

5 **Appearance**

Normally a white to off-white lyophilized powder. A description that doesn't match what you received is worth asking about.

6 **HPLC purity**

The percentage of the main peak's area. Research standard: ≥98%.

7 **Chromatogram**

The graph behind that percentage. Look for a dominant peak and a clean baseline.

8 **LC-MS identity**

The measured mass. It should match the stated molecular weight within the method's margin.

9 **Water / acetate content**

Residues from the process. They aren't peptide impurities, but they affect the true net weight.

10

Lab and signature

Who issues the analysis and stands behind it. It should be externally verifiable.

THE MOST COMMON MISTAKE

Looking only at the purity percentage. It's the easiest figure to read, and also the easiest to write into a PDF. **A number with no chromatogram, no matching lot, and no identifiable lab isn't a data point: it's a claim.**

The five *checks*

It doesn't matter which family, and it doesn't matter which molecule: the standard is the same. Five checks, and if one fails, you stop.

1 **Lot**

The COA corresponds to the exact lot you're about to receive, not to a generic code or a "representative" lot.

2 **Identity**

The mass measured by LC-MS matches the peptide's declared molecular weight, within the method's margin.

3 **Purity**

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

4 **Laboratory**

Issued by an independent laboratory that is identifiable and externally verifiable.

5 **Clinical judgment**

Quality is verified; use, indication, and follow-up belong to a licensed healthcare professional.

RULE OF THUMB

If a material can't be independently verified, **treat it as unaccredited**. Without that foundation, no mechanism and no research line changes the decision.

Eight *red flags*

Concrete signs that the document in front of you, or the vendor sending it, won't hold up under scrutiny. None is conclusive on its own; two together are already reason enough to stop.

01 **The COA carries no lot number**

Or one that doesn't match the vial's label. A certificate with no lot certifies nothing: it can't be linked to the material in your hand.

02 **No issuing lab is named**

Or a name appears that doesn't exist outside that website. The lab must be identifiable and independently contactable.

03 **The chromatogram is missing**

A purity figure with no supporting graph is a claim, not a data point. You should see a clean, dominant main peak.

04 **The LC-MS mass doesn't add up**

Compare it against the theoretical molecular weight on the spec sheet. An unexplained deviation is a reason to stop, however high the stated purity.

05 **The same COA for several products**

Certificates reused with the name changed. Cross-check dates, lot numbers, and chromatogram traces.

06 **The seller suggests doses or protocols**

If a research-material distributor is guiding you on human use, they are stepping outside the framework they claim to operate in. It's a signal about the vendor, not a service.

07 **Promises of results**

"Guaranteed results," before-and-afters, transformation testimonials. None of those claims is compatible with a research-only framework.

08 **Purity declared at 100%**

No synthesis and purification process reaches 100%. A perfectly round figure suggests the number didn't come from an instrument.

PART II

The *biology*

The essentials for reading the catalog with judgment: what a peptide is, how it acts on a receptor, what "research use only" actually means, and how to interpret an evidence claim.

05 **What a peptide is**

06 **The signal and the receptor**

07 **The RUO framework**

08 **How to read an evidence claim**

What a *peptide is*

A short chain of amino acids: the same pieces the body uses to build proteins. The difference is one of size and function.

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

Messengers, not building blocks

Many peptides don't build tissue: they coordinate it. They function 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 why a peptide can have a defined effect, and at the same time, why its exact sequence matters so much.

TRAIT	PEPTIDE	PROTEIN
Length	Short chain (up to ~50 aa)	Long chain, hundreds of aa
Structure	Simple, minimally folded	Folded in 3D, complex
Typical role	Signal, messenger	Structure, enzyme, transport
Stability	Low; degrades quickly	Higher, depending on folding

Natural and synthetic

Many peptides occur naturally in the body: oxytocin, glucagon, or GLP-1 itself. Others are synthesized in the lab, either by copying a natural sequence or by designing a new one with sought-after properties, such as greater stability. Most of the molecules in this guide are synthetic and are studied within that framework. A minority — NAD+, glutathione, HCG — are endogenous molecules, and that difference changes how their evidence should be read.

The signal *and the receptor*

A signaling peptide acts by binding to a receptor. Understanding that relationship explains almost everything else.

The receptor is a protein, usually on the cell surface, with a shape that recognizes a specific ligand. When the peptide fits, the receptor changes conformation and triggers a cascade of signals inside the cell. Most of the receptors in this guide are GPCRs: G-protein-coupled receptors.

MODE	WHAT IT DOES	WHAT IT'S FOR
Agonist	Binds the receptor and switches it on, mimicking or reinforcing the natural signal.	Stimulating a pathway. Most materials in this guide.
Antagonist	Binds and occupies it without activating it, preventing the natural signal from doing so.	Blocking a pathway instead of stimulating it.
Selective	Acts on one specific receptor within a family.	A narrower effect, with fewer side consequences.
Non-selective	Acts on several receptors of the family at once.	A broad profile, and therefore harder to predict.

> WHY THIS MATTERS WHEN BUYING
>
> The distinction between selective and non-selective isn't a technicality: it explains why two related molecules — ipamorelin and other secretagogues, PT-141 and Melanotan II — have such different profiles. **The more receptors a molecule touches, the broader and less predictable its behavior.**

The *RUO* framework

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

An RUO material is a compound intended for laboratory study, not approved as a drug for human use. That means it hasn't gone through the complete efficacy and safety evaluation process a regulatory agency requires before authorizing a drug. The distinction is enormous, and yet it's the one most often blurred in the sector's marketing communications.

	APPROVED DRUG	RESEARCH MATERIAL (RUO)
Evaluation	Complete (EMA, FDA…)	Not approved for human use
Evidence	Published clinical trials	Often preclinical or limited
Intended use	Treatment, by prescription	Laboratory study
Responsible party	The authorization holder	Whoever uses it in research

Three situations worth not mixing up

Approved in another form. Semaglutide, tirzepatide, and tesamorelin are active ingredients of authorized drugs. That doesn't turn the research powder into a medicine: the approval covers a specific product, with its formulation, route, and indication.

Approved in another jurisdiction. Thymosin alpha-1 is approved in more than thirty countries but not by the FDA or EMA. Semax and Selank are registered in Russia. A national registration doesn't equal approval under FDA/EMA standards, nor proof of safety under those criteria.

Approved in a close relative. Afamelanotide is approved for one specific rare disease; Melanotan II is not approved in any jurisdiction. Chemical resemblance doesn't transfer regulatory status.

How to read *an evidence claim*

Most of the misunderstandings in this sector don't come from false data, but from true data read with the wrong grammar.

WHEN YOU READ…	IT MEANS…	IT DOESN'T MEAN…
"Studied in the context of X"	There is a line of research on X.	That it works for X.
"Proposed mechanism"	A reasoned hypothesis, often from the lab.	A process demonstrated in people.
"In preclinical models"	Cell cultures or animals.	That it replicates in humans.
"Phase 3"	Advanced clinical development underway.	Approval.
"Registered in [country]"	National authorization under local criteria.	FDA/EMA approval.
"99.9% purity"	The lot's analytical quality.	Safety in people.

THE BIAS THAT RUNS THROUGH THE FIELD

Several reviews note **likely publication bias** in the preclinical peptide literature: positive results are published more readily than negative ones. In practice, this means the overall impression left by a literature search is more optimistic than the field's actual state. When a molecule accumulates dozens of enthusiastic animal studies and no human trial in twenty years, the absence is itself a data point.

PART III

The *materials*

Twenty-seven materials in six families, grouped by the biological system they act on rather than by the promise they're usually sold with. Each card carries its proposed mechanism, evidence level, regulatory status, and reference lot.

F1	**Metabolism and weight** — 6 materials
F2	**Tissue repair and inflammation** — 4 materials
F3	**Skin and dermal matrix** — 3 materials
F4	**Somatotropic axis** — 4 materials
F5	**Melanocortin and central nervous system** — 4 materials
F6	**Longevity, redox, and immunity** — 6 materials

F1

Metabolism and weight

Materials that act on the signals the body uses to coordinate insulin, gastric emptying, and hunger — and on the cell's energy machinery.

The gut and pancreas release incretin hormones (GLP-1, GIP) alongside amylin. Together they modulate glucose-dependent insulin release, slow gastric emptying, and signal satiety to the brain. The synthetic analogs in this family are designed to activate those same receptors with an extended half-life, generally through fatty-acid acylation that binds them to albumin and protects them from degradation.

Alongside them we group two compounds that reach metabolism through a different door: the cell's energy machinery. AMPK is the sensor that switches on when energy runs short; NAD+ is the redox cofactor that connects a cell's state to its capacity to produce energy. That second half of the family is far less settled: almost all the evidence is cellular and from rodents.

One class trait worth keeping in mind: in the incretin-mimetic analogs, the most frequently reported adverse effects are gastrointestinal, especially when escalating the dose.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
Semaglutide	Selective GLP-1 agonist	■■■■ E4	Approved	—
Tirzepatide	Dual GIP / GLP-1 agonist	■■■■ E4	Approved	99.78%
Retatrutide	Triple GIP / GLP-1 / glucagon agonist	■■■□ E3	RUO	99.91%
Cagrilintide	Long-acting amylin analog	■■■□ E3	RUO	—
MOTS-c	Mitochondrial peptide · AMPK activator	■□□□ E1	RUO · WADA	—
5-Amino-1MQ	Small molecule · NNMT inhibitor (not a peptide)	■□□□ E1	RUO	99.94%

Semaglutide

Ozempic · Wegovy · Rybelsus

Approved

Selective GLP-1 agonist

■■■■ E4 · APPROVED

Synthetic analog of GLP-1, the incretin hormone the body releases after eating, engineered to resist degradation and stay active for several days. It is the most established molecule in the family.

MECHANISM	Binds selectively to the GLP-1 receptor and activates it. Associated with glucose-dependent insulin secretion, lower postprandial glucagon, slower gastric emptying, and signaling in appetite-related brain regions.
STUDIED FOR	Glycemic control in type 2 diabetes, chronic weight management, and cardiovascular risk reduction in selected populations.
EVIDENCE	High. Extensive Phase 3 program and a molecule approved for specific indications.
STATUS	Approved by the FDA and EMA (Ozempic/Rybelsus for diabetes; Wegovy for weight and cardiovascular risk). Catalog versions sold as RUO are not for human use.
SAFETY	Boxed warning for thyroid C-cell tumors in rodents; contraindicated with a history of medullary thyroid carcinoma or MEN 2. Acute pancreatitis has been reported. Gastrointestinal effects are common.

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

Tirzepatide

Mounjaro · Zepbound · LY3298176

Approved

Dual GIP / GLP-1 agonist

E4 · APPROVED · PURITY 99.78%

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

MECHANISM	Activates GIP and GLP-1 simultaneously, with higher affinity for GIP and cAMP-biased agonism at GLP-1. Associated with glucose-dependent insulin, lower glucagon, slower emptying, and satiety.
STUDIED FOR	Glycemia in type 2 diabetes, weight management, and more recently obstructive sleep apnea in adults with obesity.
EVIDENCE	High. Phase 3 program (SURPASS / SURMOUNT) and approvals across several indications.
STATUS	Approved by the FDA (Mounjaro 2022; Zepbound 2023; sleep apnea 2024) and EMA (2022). RUO versions are not for human use.
SAFETY	Same class warning for thyroid C-cell tumors. Signals of pancreatitis and gallbladder events. Gastrointestinal effects common when escalating dose.

CAS 2023788-19-2 · C225H348N48O68 · 4813.45 g/mol · 39 aa · lot GT10-032026-1

Retatrutide

LY3437943 · triple agonist

RUO

Triple GIP / GLP-1 / glucagon agonist

E3 · EXTENSIVE CLINICAL · PURITY 99.91%

Investigational molecule, the first triple agonist in its class: it acts on three receptors. It adds the glucagon receptor to the scheme of semaglutide and tirzepatide.

MECHANISM	Activates GIP, GLP-1, and glucagon at once. The glucagon component is theorized to affect energy expenditure, in addition to the incretin effects. Still under characterization.
STUDIED FOR	Weight management in obesity, glycemia in type 2 diabetes, and, in the Phase 3 TRIUMPH program, also sleep apnea and knee osteoarthritis pain.
EVIDENCE	Moderate and evolving. Published Phase 2 data (NEJM, 2023) and Phase 3 ongoing. Not approved.
STATUS	Not approved in any country; in Phase 3, with no regulatory filing submitted. RUO framework: not intended for human use.
SAFETY	In Phase 2, adverse events were mostly gastrointestinal (mild to moderate), more frequent at higher doses; discontinuations ~6-16% depending on dose. Long-term profile not established.

CAS 2381089-83-2 · ~4731.33 g/mol · 39 aa · lot K3BQAA

Cagrilintide

AM833 · NN9838 · CagriSema (with semaglutide)

RUO

Long-acting amylin analog

E3 · EXTENSIVE CLINICAL

Long-acting synthetic version of amylin, the hormone the pancreas co-secretes with insulin at mealtime. It belongs to a pathway distinct from the GLP-1s and is studied mostly alongside semaglutide.

MECHANISM	Non-selective agonist of amylin receptors (AMY1-3R) and calcitonin receptors, mainly in the area postrema and hypothalamus, modulating appetite and satiety; action on reward pathways has also been proposed.
STUDIED FOR	Weight management and obesity, as monotherapy and in co-formulation with semaglutide, and glycemic control.
EVIDENCE	Moderate. Phase 2 as monotherapy and Phase 3 for CagriSema (REDEFINE). No stand-alone approval.
STATUS	No stand-alone authorization from the FDA or EMA. Novo Nordisk filed a CagriSema application in Dec-2025, under review. RUO framework.
SAFETY	Shares the class's gastrointestinal profile, with a nausea/vomiting signal possibly milder than semaglutide as monotherapy. Long-term data still accumulating.

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

MOTS-c

Mitochondrial ORF of the 12S rRNA type-c

RUO WADA

Mitochondrial peptide · AMPK activator

E1 · PRECLINICAL

Short 16-amino-acid peptide encoded within the mitochondrial 12S ribosomal RNA gene, first described in 2015. It is naturally detectable in blood and rises with exercise.

MECHANISM	Proposed, not fully established: under metabolic stress it translocates to the nucleus and is associated with AMPK activation; in muscle it has been proposed to interfere with the folate cycle. In models it increases glucose uptake in a way reminiscent of exercise.
STUDIED FOR	Metabolic homeostasis, insulin sensitivity, diet-induced obesity, mitochondrial health, and metabolic aging. Human data are mostly observational.
EVIDENCE	Low to moderate. Mostly preclinical and observational. The only relevant human data come from an analog (CB4211) whose development was discontinued.
STATUS	Not approved by the FDA or EMA. Banned by WADA since Jan-1-2024 (section S4, AMPK activators).
SAFETY	The safety profile of MOTS-c administered in humans is not characterized: no long-term studies. Being 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

RUO

Small molecule · NNMT inhibitor (not a peptide)

E1 · PRECLINICAL · PURITY 99.94%

Small-molecule inhibitor of the enzyme nicotinamide N-methyltransferase (NNMT). It is not a peptide; it is included here for its shared metabolic biology.

MECHANISM	Selectively and competitively inhibits NNMT, which methylates nicotinamide using SAM. By slowing that methylation, in models nicotinamide remains available for recycling into NAD+, and in adipocytes it is associated with a metabolic shift toward oxidation.
STUDIED FOR	Diet-induced obesity, regulation of adipose tissue metabolism, NAD+ and SAM biology, and muscle stem cell function.
EVIDENCE	Low (preclinical). In vitro and rodent data. No published human clinical trials.

STATUS	Not approved by the FDA or EMA, with no known active IND. Sold only as a laboratory reagent.
SAFETY	No human trials: its safety, toxicity, and adverse effects in people are unknown.

CAS 42464-96-0 (iodide salt) · cation C10H11N2+ · ~159.21 g/mol · NNMT IC50 ~1.2 µM · lot JRZTDB

F2

Tissue repair and inflammation

Compounds studied preclinically for their possible role in wound healing — angiogenesis, fibroblast migration, matrix remodeling — and in modulating the inflammatory response.

Repairing an injured tissue is one of biology's most finely tuned orchestrations. After a wound, the body contains bleeding, recruits immune cells, builds new vessels to bring oxygen to the site, mobilizes 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 this research points its attention.

This family is best read with a clear compass: almost everything known about these compounds comes from preclinical studies. Human validation is today very limited, with no large or rigorous trials. That is why you won't find promises or protocols here, but rather the honest context of what is being studied and with how much evidence. Several are also listed as prohibited substances in sport.

We add KPV to this family, which in the original organization lived apart. It shares the underlying biology with the others — modulating an injury response — and its evidence sits on the same preclinical step, so separating them hid more than it clarified.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
BPC-157	Synthetic pentadecapeptide (15 aa)	E1	RUO · WADA	99.51%
TB-500	Research peptide (thymosin β4)	E1	RUO · WADA	99.58%
BPC-157 + TB-500	Combination of two research peptides	E0	RUO · WADA	99.29%
KPV	Melanocortin-derived tripeptide	E1	RUO	99.75%

BPC-157 Body Protection Compound 157

RUO WADA

Synthetic pentadecapeptide (15 aa)

E1 · PRECLINICAL · PURITY 99.51%

Synthetic 15-amino-acid peptide whose sequence corresponds to a fragment of a gastric-juice protective protein. The preclinical literature describes it as cytoprotective.

MECHANISM

Proposed, mostly in animals and 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: tendon and ligament repair, muscle regeneration, skin wound healing, gastrointestinal mucosal protection, and angiogenesis.
EVIDENCE	Mostly preclinical. Sharp contrast between abundant animal research and the near-absence of human evidence. Reviews note likely publication bias.
STATUS	Not approved by any agency. In the U.S. it was FDA Category 2 and was removed from that category in Apr-2026, which does not equal approval. Prohibited in sport (WADA, S0).
SAFETY	No solid evidence of safety in humans. Theoretical, unconfirmed risks such as pathological angiogenesis. Market purity may vary.

CAS 137525-51-0 · C62H98N16O22 · ~1419.5 g/mol · 15 aa · lot 2FRBRG

TB-500 Thymosin β4 fragment

RUO WADA

Research peptide (thymosin β4)

E1 · PRECLINICAL · PURITY 99.58%

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 the whole protein or to 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-epithelialization, 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 has had trials in narrow ophthalmic indications; extrapolating from one to the other is not advisable.
EVIDENCE	For "TB-500" as such: preclinical, with no controlled trials. The most advanced clinical development corresponds to the protein formulated as an investigational drug, not to the RUO material.
STATUS	Not approved by the FDA or EMA. Treated as Category 2 (compounding). Prohibited in sport (WADA, growth factors S2.3 and S0).
SAFETY	No large-scale or long-term human safety studies. Purity and composition can vary between products.

CAS 77591-33-4 (thymosin β4) · 43 aa · ~4963 g/mol · lot PQ7MWD

BPC-157 + TB-500 recovery blend

RUO WADA

Combination of two research peptides

E0 · NO DATA · PURITY 99.29%

A blend 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 characterizes a combined effect in humans; any synergy is hypothetical.
STUDIED FOR	Interest comes from the tissue-repair context, but there is no specific evidence for the combination: the preclinical backing corresponds to the components separately.
EVIDENCE	No specific evidence for the blend. This is the entry with the least direct backing in the guide.
STATUS	Not approved. Inherits its components' status: not approved and prohibited in sport (WADA).
SAFETY	Combining two compounds with no dedicated studies adds uncertainty: no data exist on interactions or on the safety of the blend in humans.

Combination with no single CAS · lot BPTB20-052026-4

KPV Lys-Pro-Val · MSH(11-13)

RUO

Melanocortin-derived tripeptide

E1 · PRECLINICAL · PURITY 99.75%

Tripeptide corresponding to residues 11-13 of the C-terminus 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: its anti-inflammatory action would not depend on melanocortin receptors. The peptide enters the cell (in intestinal epithelium, via the PepT1 transporter) and would interfere with NF-κB and MAP-kinase (ERK/p38) activation.
STUDIED FOR	Modulation of inflammatory processes. The most developed line: preclinical models of intestinal inflammation (DSS- and TNBS-induced colitis in mice). Also models of skin inflammation and topical administration.
EVIDENCE	Preclinical. In vitro and animal models. No human clinical trials support therapeutic uses.
STATUS	Not approved for human use. RUO framework. Not a drug approved by the FDA, EMA, or other agencies for any indication.
SAFETY	With no human clinical trials, its safety profile, dosing, and interactions in people are not established.

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

F3

Skin and dermal matrix

Materials studied for their interaction with the dermal matrix — collagen, elastin, glycosaminoglycans — where fibroblasts and the vascular network determine firmness, hydration, and wound healing.

Beneath the epidermis, the dermis is a living scaffold of collagen and elastin supported by fibroblasts and nourished by capillaries. With age, matrix synthesis declines, oxidative damage accumulates, and enzymes like elastase degrade the fibers that give firmness. Against that backdrop, science explores small candidate peptides that may modulate repair signals.

Worth clarifying up front: except for the use of GHK-Cu as a topical cosmetic ingredient, these are research materials with no approval for human use. And the two blends in this family have no dedicated research of their own — what is known comes from each component separately, mostly at preclinical stages.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
GHK-Cu	Copper tripeptide	■■□□ E2	RUO	99.75%
GLOW Blend	Multi-component blend	□□□□ E0	RUO · WADA	99.60%
KLOW Blend	Multi-component blend	□□□□ E0	RUO · WADA	99.76%

GHK-Cu

Copper tripeptide-1 · Gly-His-Lys:Cu

RUO

Copper tripeptide

■■□□ E2 · LIMITED CLINICAL · PURITY 99.75%

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

MECHANISM	Proposed: delivers copper into the cell and, in fibroblasts and animal models, is associated with expression of collagen, elastin, and glycosaminoglycan genes, elastase inhibition, and angiogenesis. It does not act through a single classical receptor.
STUDIED FOR	Wound repair and dermal matrix turnover, collagen and elastin synthesis, antioxidant and anti-inflammatory activity, and cosmetic applications for skin aging.
EVIDENCE	Relatively solid preclinical evidence (in vitro and animal) and some small topical cosmetic studies. Evidence for systemic use is virtually nonexistent.

STATUS	Used as an ingredient in topical cosmetics (INCI Copper tripeptide-1). As a lyophilized research material for systemic use, it is not approved by the FDA or EMA. RUO framework.
SAFETY	Research material, not approved for systemic administration. Delivers copper, whose excess can be problematic.

CAS 89030-95-5 (complex) · 49557-75-7 (free peptide) · 3 aa · 340.38 g/mol (free) · lot C3M9KQ

GLOW Blend

GHK-Cu + BPC-157 + TB-500

RUO WADA

Multi-component blend

E0 · NO DATA · PURITY 99.60%

Research blend combining the copper tripeptide GHK-Cu with two peptides studied in the context of tissue repair. It is not a single molecule but a multi-component formulation.

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

Combination with no single CAS · lot GLOW-062026-4

KLOW Blend

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

RUO WADA

Multi-component blend

E0 · NO DATA · PURITY 99.76%

Adds the tripeptide KPV to GLOW's components. It thus combines a compound 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, with fewer pro-inflammatory cytokines and without α-MSH's pigmentary effects; the rest, as in GLOW. No combined mechanism has been characterized.

STUDIED FOR	Skin inflammation and tissue repair, adding KPV's anti-inflammatory line to that of the other components.
EVIDENCE	No clinical evidence for the blend. It draws on the individual components.
STATUS	Not approved. KPV is sold only for research; BPC-157 and TB-500 are RUO with no approval and are prohibited in sport.
SAFETY	Blend of unapproved research materials; the safety profile of the combination is not characterized.

Combination with no single CAS · adds KPV (CAS 67727-97-3) to the GLOW components · lot XX1EZC

F4

Somatotropic axis

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

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 physiological pulsatile secretion.

Two groups coexist: GHRH analogs (tesamorelin, CJC-1295 without DAC), which mimic the GH-releasing hormone, and 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 without demonstrating efficacy; CJC-1295 without DAC is essentially preclinical.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
Tesamorelin	GHRH analog · approved (specific indication)	■■■■ E4	FDA approved	99.93%
Ipamorelin	Selective ghrelin agonist (GHS-R1a)	■■□□ E2	RUO · WADA	99.90%
CJC-1295 without DAC + Ipamorelin	GHRH + ghrelin combination (research)	■□□□ E1	RUO · WADA	99.96%
Tesamorelin + Ipamorelin	GHRH + ghrelin combination (research)	■□□□ E1	RUO · WADA	99.71%

Tesamorelin

EGRIFTA · TH9507

FDA approved

GHRH analog · approved (specific indication)

■■■■ E4 · APPROVED · PURITY 99.93%

Stabilized synthetic analog of human GHRH. It is the only GH secretagogue in this family with regulatory approval: the FDA approved it in 2010 for a specific indication.

MECHANISM

Binds the GHRH receptor in the pituitary and stimulates 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). It has been explored off-label in other metabolic contexts and hepatic steatosis.
EVIDENCE	High for its approved indication: Phase 3 with more than 800 participants. It is the peptide with the strongest clinical evidence in the family.
STATUS	Approved by the FDA (2010; reformulations EGRIFTA SV/WR). NOT approved by the EMA (the application was withdrawn in 2012). Outside that indication, not approved.
SAFETY	Contraindicated in pregnancy, active malignancy, and pituitary axis disorders. Warnings: glucose intolerance / increased diabetes risk, fluid retention, edema, arthralgia, and carpal tunnel syndrome.

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

Ipamorelin NNC 26-0161

RUO WADA

Selective ghrelin agonist (GHS-R1a)

E2 · LIMITED CLINICAL · PURITY 99.90%

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

MECHANISM	Proposed: selective agonist of the ghrelin receptor (GHS-R1a) in the anterior pituitary, a pathway distinct from GHRH; associated with increased plasma GH. It is described as selective because, at the doses studied, it does not significantly raise ACTH, cortisol, or prolactin.
STUDIED FOR	Stimulation of the GH axis; preclinically, weight gain and bone growth in rodents and gastrointestinal motility. It reached Phase 2 for postoperative ileus.
EVIDENCE	Mixed: solid preclinical characterization and some human data, but development for postoperative ileus was discontinued for lack of efficacy. Not approved.
STATUS	Not approved by the FDA or EMA. Research peptide (RUO). Prohibited in sport (WADA, S2).
SAFETY	Since 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 · lot VNA7TB

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

RUO WADA

GHRH + ghrelin combination (research)

E1 · PRECLINICAL · PURITY 99.96%

Research formulation combining a GHRH analog (CJC-1295 without DAC, or mod GRF 1-29) with a ghrelin receptor agonist. CJC-1295 without DAC has a short half-life (~30 min) because it lacks the albumin-binding DAC moiety.

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 is not characterized in formal clinical trials.
EVIDENCE	Low for the combination: essentially preclinical and theoretical. CJC-1295 without DAC lacks approved clinical development.
STATUS	Not approved by the FDA or EMA. Both components are research material (RUO); CJC-1295 without DAC is described as laboratory use only.
SAFETY	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

Tesamorelin + Ipamorelin

GHRH + ghrelin combo

RUO WADA

GHRH + ghrelin combination (research)

E1 · PRECLINICAL · PURITY 99.71%

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

MECHANISM	Proposed, dual: tesamorelin activates the GHRH receptor (GH release and secondary IGF-1 increase) and ipamorelin the ghrelin receptor. The research idea is that the combination would potentiate 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 is not characterized in clinical trials.
EVIDENCE	Low for the combination. The solid clinical evidence belongs only to tesamorelin in its approved indication; ipamorelin did not reach approval.
STATUS	The combination is not approved by the FDA or EMA (RUO). FDA approval applies only to tesamorelin alone in its authorized indication.
SAFETY	No safety profile of its own. Keep in mind tesamorelin's warnings (glucose, fluid retention, arthralgia) and its contraindications.

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

F5

Melanocortin and central nervous system

Materials acting on central nervous system circuits: the melanocortin family — derived from POMC — and the short Russian-origin peptides studied for their action on neuronal plasticity and the stress response.

The melanocortin family originates in the study of α-MSH, a natural peptide that binds five G-protein-coupled receptors (MC1R through MC5R). Each serves a different function: MC1R, in melanocytes, regulates melanin production; MC4R and MC3R take part in appetite and sexual-behavior circuits. That multiplicity explains why a poorly selective molecule has a broad and delicate profile.

Alongside them we group two short Russian-origin peptides that share a chemical strategy: taking an active fragment of an endogenous molecule and adding a stabilizing Pro-Gly-Pro tail that protects it from enzymes. Both are registered as medicines in Russia, but that status does not equal FDA or EMA approval: clinical research is of limited scope, with small samples and little independent replication.

Here we gather what the original organization split across three chapters. The criterion running through the whole guide is the same: group by real biology, not by commercial promise.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
PT-141 (Bremelanotide)	Melanocortin agonist (MC3R / MC4R)	■■■■ E4	FDA drug · RUO powder	99.91%
Melanotan II	Non-selective melanocortin agonist	■■□□ E2	RUO · Warnings	99.81%
Semax (N-acetyl amidate)	Heptapeptide neuropeptide (derived from ACTH 4-7)	■■□□ E2	Reg. Russia · RUO	99.11%
Selank	Tuftsin-analog heptapeptide	■■□□ E2	Reg. Russia · RUO	99.74%

PT-141 (Bremelanotide)

Vyleesi (approved drug) · PT-141

FDA drug · RUO powder

Melanocortin agonist (MC3R / MC4R)

■■■■ E4 · APPROVED · PURITY 99.91%

Synthetic cyclic heptapeptide, related to melanotan II (it is its active metabolite). An α-MSH analog studied in the central melanocortin system. Unlike PDE5 inhibitors, its proposed mechanism is central, not vascular.

MECHANISM

Proposed: melanocortin receptor agonist, with greater potency at MC3R/MC4R than at MC1R. It is proposed that MC4R activation in the hypothalamus and limbic regions modulates motivation pathways. The details of downstream signaling 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 general sexual dysfunction.
EVIDENCE	Mixed. For HSDD, Phase 3 (RECONNECT trials): significant differences versus placebo, of modest magnitude and with a high placebo effect. For other uses, more preliminary.
STATUS	The drug bremelanotide was approved by the FDA in 2019 (Vyleesi, auto-injector) for HSDD in premenopausal women. It has no current EMA authorization. Research-grade PT-141 powder is RUO: not approved for human use and not equivalent to the drug.
SAFETY	In trials: nausea (~40%), flushing (~20%), headache (~11%), and injection-site reactions. Transient blood pressure increase: contraindicated with uncontrolled hypertension or cardiovascular disease. Repeated use may be associated with focal hyperpigmentation.

CAS 189691-06-3 · C50H68N14O10 · ~1025.2 Da · cyclic heptapeptide (7 aa) · lot YGAFK7

Melanotan II MT-II · MT-2

RUO Warnings

Non-selective melanocortin agonist

E2 · LIMITED CLINICAL · PURITY 99.81%

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

MECHANISM	Proposed: non-selective agonist of melanocortin receptors (MC1R, MC3R, MC4R, MC5R). Binding to MC1R is studied in relation to melanogenesis; central MC3R/MC4R activation is associated with its systemic effects on appetite and autonomic responses.
STUDIED FOR	Pigmentation and photoprotection biology (via MC1R) and as a tool to study appetite-linked melanocortin circuits (via MC4R). Its commercial development was abandoned due to regulatory restrictions.
EVIDENCE	Low to moderate depending on the outcome. Mostly preclinical plus some early Phase I studies; it did not complete clinical development or reach market. Much real-world use 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 sold as such. Its relative afamelanotide (Scenesse) is approved, but only for erythropoietic protoporphyria.
SAFETY	Documented nausea, vomiting, facial flushing, yawning, and appetite loss. Prolonged erection (priapism) in men, which may require urgent care. Darkening of moles and appearance of new ones; a link to melanoma has not been conclusively demonstrated.

CAS 121062-08-6 · C50H69N15O9 · ~1024.18 g/mol · cyclic heptapeptide · lot MT210-052026-7

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

Reg. Russia | RUO

Heptapeptide neuropeptide (derived from ACTH 4-7)

E2 · LIMITED CLINICAL · PURITY 99.11%

Synthetic heptapeptide developed in Russia from the ACTH 4-7 fragment, with a stabilizing Pro-Gly-Pro tripeptide. It retains neurotrophic effects but, per the literature, without hormonal activity on the adrenal axis.

MECHANISM	Proposed, neurotrophic and neuromodulatory: increases BDNF and NGF expression (mRNA) in the hippocampus and frontal cortex, linked to synaptic plasticity; also modulation of the dopaminergic and serotonergic systems. Mostly from cellular and animal models.
STUDIED FOR	Cognitive function (attention, memory) and neuroprotection. The most developed clinical line (Russian) is ischemic stroke and neurological recovery.
EVIDENCE	Mixed, with limitations. Small-sample studies, published in Russian, with little independent replication. The N-acetyl amidated variant has very scarce specific human data.
STATUS	Not approved by the FDA or EMA. In Russia, Semax (base form) is registered as a medicine; that national registration does not equal FDA/EMA approval. Outside Russia, RUO.
SAFETY	Its safety profile, interactions, and long-term behavior are not established to Western standards, especially for the N-acetyl amidated variant.

CAS 2920938-90-3 (N-acetyl amidate) · C39H54N10O10S · ~854.97 g/mol · 7 aa · Semax base CAS 80714-61-0 · lot SEMX10-042026-3

Selank TP-7 · tuftsin analog

Reg. Russia | RUO

Tuftsin-analog heptapeptide

E2 · LIMITED CLINICAL · PURITY 99.74%

Synthetic heptapeptide from the Institute of Molecular Genetics (Russian Academy of Sciences), based on tuftsin — an immunomodulatory tetrapeptide fragment of immunoglobulin G — with a stabilizing Pro-Gly-Pro tail.

MECHANISM	Proposed, multifactorial and non-benzodiazepine. On the GABAergic system the evidence is nuanced: in vitro, by itself it did not alter GABA-A receptor subunit expression but blunted GABA-induced changes, interpreted as indirect modulation. Also influences enkephalin metabolism and BDNF expression.
STUDIED FOR	Anxiety (generalized anxiety disorder, asthenic states) and cognitive function, described without sedation. Preclinically: enkephalins, monoaminergic systems, GABAergic gene expression.

EVIDENCE	Limited. The most-cited clinical evidence comes from small-sample Russian trials with narrow methodological reporting. No large controlled trials under Western standards.
STATUS	Not approved by the FDA or EMA. In Russia it is registered as a prescription anxiolytic/nootropic medicine. Outside Russia, RUO.
SAFETY	Human safety data are limited and mostly Russian; interaction profile and prolonged use are not established to Western standards.

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

F6

Longevity, redox, and immunity

Compounds studied in relation to the mechanisms of cellular aging, redox balance, and modulation of the immune response. Here well-characterized endogenous molecules coexist with peptides whose evidence is mostly preclinical.

Aging is not a single process but the sum of several mechanisms that decline: energy metabolism and sirtuins (via NAD+), telomere maintenance, epigenetic regulation, redox balance, and immune system competence. This family gathers the materials research associates with those processes.

The distance between its members is large, and that is precisely the point. NAD+ and glutathione are endogenous molecules whose biochemistry is textbook material: what is debated is not what they do inside the cell, but what happens when they are administered from outside. Epitalon and Pinealon are synthetic peptides with preclinical evidence coming largely from a single research group. Thymosin alpha-1, by contrast, has real clinical development and drug approval in more than thirty countries — although not by the FDA or EMA.

We close the family with HCG, which the original organization treated as a separate chapter. It shares with the rest the status of an endogenous hormone with its own pharmacological history, and it carries the same misunderstanding that runs through several of these materials: the gap between what is approved and what is promised.

MATERIAL	CLASS	EVIDENCE	STATUS	PURITY
NAD+	Endogenous coenzyme (not a peptide)	■■□□ E2	RUO / suppl.	99.85%
Epitalon	Tetrapeptide (pineal bioregulator)	■□□□ E1	RUO	99.75%
Pinealon	Tripeptide (ultra-short bioregulator)	■□□□ E1	RUO	99.84%
L-Glutathione (GSH)	Endogenous tripeptide (thiol antioxidant)	■■□□ E2	Suppl. / RUO	99.73%
Thymosin alpha-1	Thymic immunomodulatory peptide (28 aa, N-acetylated)	■■■□ E3	+30 countries · RUO	99.65%
HCG	Glycoprotein hormone · LH/CG receptor agonist	■■■■ E4	Approved (indic.) · WADA	—

NAD+

Nicotinamide adenine dinucleotide · β-NAD+

RUO / suppl.

Endogenous coenzyme (not a peptide)

E2 · LIMITED CLINICAL · PURITY 99.85%

Coenzyme the body naturally synthesizes, present in every cell. Central to energy metabolism (the NAD+/NADH pair) and a substrate for enzymes such as sirtuins and PARPs. Its availability declines with age.

MECHANISM	Proposed in longevity research: redox cofactor and obligatory co-substrate for 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.
STUDIED FOR	Cellular aging, sirtuin biology, mitochondrial metabolism, and DNA repair. Its precursors (NMN, nicotinamide riboside) have been evaluated in early trials.
EVIDENCE	Variable. The basic biology of NAD+ and sirtuins is solidly established. Exogenous administration as an anti-aging strategy has limited evidence, with no large pivotal trials. Commercial use outpaces the evidence.
STATUS	Not approved as a drug for longevity indications. As an exogenous compound it is sold under a research framework (RUO) or as a supplement, with no 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 drugs: possible quality variability.

CAS 53-84-9 · C21H27N7O14P2 · ~663.43 g/mol · dinucleotide · lot J4Q90F

Epitalon

Epithalon · AEDG (Ala-Glu-Asp-Gly)

RUO

Tetrapeptide (pineal bioregulator)

E1 · PRECLINICAL · PURITY 99.75%

Short synthetic peptide developed in Russia (Khavinson group) as a synthetic version of the pineal complex epithalamin. Belongs to the bioregulator peptides. Research material, not approved.

MECHANISM	Proposed: in cultures and human lymphocytes, telomerase activation (TERT/TERC) with telomere elongation has been described; also influence on melatonin synthesis, epigenetic effects via DNA/histone binding, and antioxidant activity. The literature notes that the mode of action is not fully elucidated.
STUDIED FOR	Geroprotection and aging, telomere maintenance, circadian rhythms and melatonin, and, exploratorily, retinitis pigmentosa and cognitive function.
EVIDENCE	Low. Mostly preclinical (cultures and animal models) and largely from a single Russian research group. Only two small human studies.
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	Significant safety gap: no genotoxicity, carcinogenicity, or human interaction studies. Short peptides tend to be unstable in vivo.

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

Pinealon EDR (Glu-Asp-Arg)

RUO

Tripeptide (ultra-short bioregulator)

E1 · PRECLINICAL · PURITY 99.84%

Short synthetic peptide developed in Russia (Khavinson group), documented as a geroprotector and bioregulator. Research material, not approved.

MECHANISM	Proposed: because of its small size, it is hypothesized 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.
STUDIED FOR	Neuroprotection and aging of the central nervous system, cell viability under oxidative stress and hypoxia, and gene expression regulation.
EVIDENCE	Low. Mostly preclinical (in vitro and rodents), with no controlled human trials, and largely from the same Russian research 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 human safety studies: toxicity, interactions, and long-term effects unknown. Short peptides unstable in vivo.

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

L-Glutathione (GSH) Reduced glutathione · γ-Glu-Cys-Gly

Suppl. / RUO

Endogenous tripeptide (thiol antioxidant)

E2 · LIMITED CLINICAL · PURITY 99.73%

The most abundant non-protein thiol in animal cells, present in nearly every tissue at millimolar concentrations. A tripeptide the body synthesizes in two enzymatic steps. It coexists in reduced (GSH) and oxidized (GSSG) form; their ratio defines the cell's redox state.

MECHANISM	Proposed: antioxidant through two routes. Directly, its thiol group donates electrons and neutralizes ROS. Indirectly, it is a cofactor for detoxification enzymes (glutathione peroxidases, S-transferases, glyoxalases). This is biochemistry, not a claim of therapeutic benefit.

STUDIED FOR	Oxidative stress and redox homeostasis: antioxidant defense, detoxification, and models linked to cardiovascular, metabolic, infectious, and neurological disease.
EVIDENCE	The physiological role is solidly established at the biochemical level. Evidence for supplementation in humans is uneven: oral bioavailability is low due to digestive degradation, and trials give mixed results, often in small samples.
STATUS	Not an approved drug to treat disease. Sold orally as a supplement. 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.
SAFETY	Administration in unapproved forms (injectables in particular) is not endorsed and carries risk: in 2019 the FDA documented adverse events from endotoxins, ranging from nausea to respiratory difficulty and at least one hospitalization.

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

Thymosin alpha-1

Thymalfasin · Tα1 · Zadaxin (brand)

+30 countries · RUO

Thymic immunomodulatory peptide (28 aa, N-acetylated)

E3 · EXTENSIVE CLINICAL · PURITY 99.65%

Synthetic 28-amino-acid peptide identical to a portion of human prothymosin alpha, first isolated in 1977 from calf thymus extracts. As thymalfasin it is used as an immunomodulator in several countries; where not approved, it is distributed as a research material.

MECHANISM	Proposed: Toll-like receptor agonist (mainly TLR2 and TLR9) on dendritic and myeloid cells, activating pathways such as MyD88, IRF3/NF-κB, and p42/44 MAPK/JNK. Downstream, associated with T-lymphocyte maturation (CD4+ and CD8+), NK cell activation, and changes in cytokine profile.
STUDIED FOR	Immune modulation in chronic hepatitis B and C, chemotherapy-induced immunosuppression and certain cancers, sepsis, and as a vaccine adjuvant in immunocompromised people. Also COVID-19 and immune reconstitution.
EVIDENCE	Within this guide, one with the strongest backing: real clinical development, human trials, and drug approval in +30 countries. Strength varies by indication: most consistent in hepatitis B/C; heterogeneous in sepsis and oncology; mixed and controversial in COVID-19.
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) or by the EMA. 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 corresponds to the pharmaceutical product under medical supervision.

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

HCG

Human chorionic gonadotropin · Pregnyl · Novarel · Ovidrel

Approved (indic.) WADA

Glycoprotein hormone · LH/CG receptor agonist

■■■■ E4 · APPROVED

Glycoprotein hormone produced by the placenta during pregnancy. As a drug it exists in urine-derived (Pregnyl, Novarel) and recombinant (choriogonadotropin alfa, Ovidrel) forms. It is not a synthetic peptide but a native hormone with alpha and beta subunits.

MECHANISM	Agonist of the shared LH/HCG receptor (LHCGR, a GPCR). Activates the Gαs → adenylate cyclase → cAMP → PKA pathway, associated with increased steroidogenic enzymes. In men it stimulates Leydig cells; 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 male hypogonadotropic hypogonadism, and ovulation induction in selected anovulatory women.
EVIDENCE	Mixed depending on the context. High for its approved indications. Null or negative for weight loss: the FDA states there is no substantial evidence it increases weight loss beyond caloric restriction.
STATUS	A hormone with indications APPROVED by the FDA, by prescription. The FDA states it is NOT approved for weight loss and that OTC products (drops, sprays, homeopathic) marketed for weight loss are fraudulent and illegal. 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; in women, primary ovarian failure, hormone-dependent tumors, pregnancy. In ovulation induction: risk of ovarian hyperstimulation syndrome, multiple pregnancy, and thromboembolism.

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



PART IV

Appendices.

Glossary of the terms used in the cards, the guide's methodology, and the sources it was built on.

A **Glossary**

B **Methodology**

C **Sources**

APPENDIX A

Glossary

The terms that appear in the cards, in one line each.

Amino acid
The building block of peptides and proteins.

Agonist
Binds to a receptor and activates it.

COA
Certificate of Analysis. A report certifying a lot's identity and purity.

LC-MS
Liquid chromatography coupled to mass spectrometry. Confirms identity.

Lyophilized
Freeze-dried. The usual presentation form for these materials.

Preclinical
Research in cell cultures and animal models, prior to humans.

GPCR
G-protein-coupled receptor. The receptor family for most of these peptides.

Publication bias
The tendency to publish positive results, which inflates the impression of efficacy.

Peptide
A short chain of amino acids, conventionally up to about 50.

Antagonist
Binds to a receptor and blocks it without activating it.

HPLC
High-performance liquid chromatography. Measures purity.

CAS
A unique numeric identifier for a chemical substance.

RUO
Research use only. Material intended exclusively for research.

Phase 1 / 2 / 3
Stages of clinical development: safety, efficacy signal, broad confirmation.

WADA
World Anti-Doping Agency. Publishes the list of substances prohibited in sport.

APPENDIX B

Methodology

How this guide was built and under what criteria, so you can cross-check it.

Each family was documented from scientific literature and official sources. Identity data (CAS, formula, molecular weight) come from public databases; purity and lot data come from the reference catalog's certificates of analysis. Where the evidence is limited or preliminary, that is stated explicitly rather than rounded up toward a conclusion.

The E0–E4 scale was applied with the same criteria to every material. When a material has a different status depending on jurisdiction or formulation — thymosin alpha-1, PT-141, tesamorelin — that asymmetry is reflected rather than choosing the most favorable reading.

What differs from other classifications

This guide groups into six families what other references split into thirteen effect categories. The criterion is the biological system the material acts on, not the outcome it's commercially credited with. That's why KPV sits with the repair materials instead of having its own chapter; why the metabolic and mitochondrial compounds travel with the incretin mimetics; and why melanocortin and the centrally acting peptides share a family. Grouping by promise separates molecules that share biology and lumps together molecules that share nothing.

ABOUT THIS GUIDE

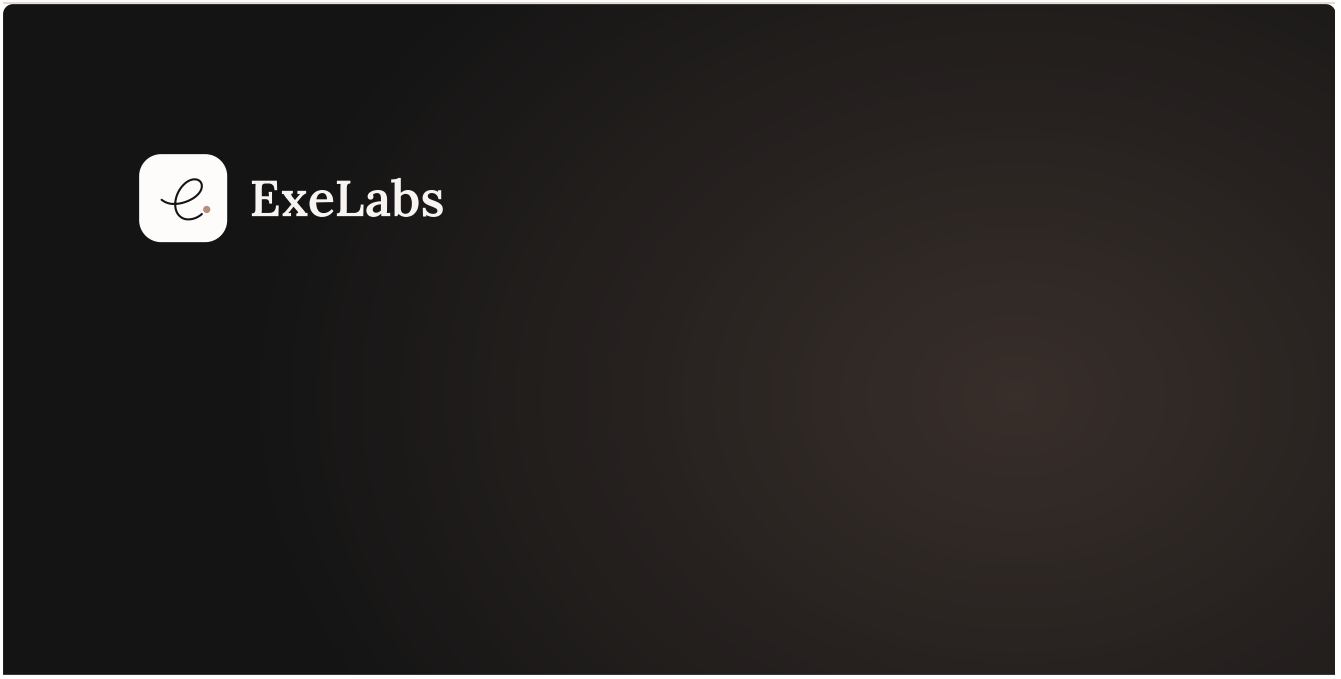
Educational material produced by ExeLabs, a distributor of materials for research use only. **We sell several of the materials described here**, and that's why the guide relies on publicly verifiable sources rather than on our own authority. 2026 edition · last updated: August 2026 · research (RUO) framework.

APPENDIX C

Sources

Primary sources. Molecule-specific ones appear on each card.

- World Health Organization — substandard and falsified medical products.
- European Medicines Agency (EMA) — regulatory status and product information.
- U.S. Food and Drug Administration (FDA) — prescribing information, warnings, and communications.
- World Anti-Doping Agency (WADA) — The Prohibited List.
- PubMed / PMC — peer-reviewed literature: mechanisms and preclinical and clinical studies.
- DrugBank and ClinicalTrials.gov — molecule identity and trial status.
- NEJM (2023) — Triple-Hormone-Receptor Agonist Retatrutide for Obesity (Phase 2).
- Raun K. et al. (1998) — Ipamorelin, the first selective growth hormone secretagogue.
- Dalmasso et al. (2008) — PepT1-Mediated Tripeptide KPV Reduces Intestinal Inflammation.
- Lee C. et al. (2015) — The Mitochondrial-Derived Peptide MOTS-c (Cell Metabolism).
- Neelakantan H. et al. (2018) — NNMT inhibitors in diet-induced obesity.
- Thymosin alpha 1: a comprehensive review (PMC7747025).



Understand the molecule. Verify the lot. *Decide with a professional.*

Notice. Informational and educational document within a research (RUO) framework. It does not sell products, does not recommend uses or doses, and does not constitute medical advice. Many of the compounds described are not approved for human use, and some are listed as prohibited substances in sport (WADA). Always consult a healthcare professional.

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