Research Compound Comparison Tables 2026

A consolidated, print-ready reference consolidating the side-by-side specifications and evidence-quality assessments from Condor Research comparison articles. Each table links to the full article; each data point is sourced from peer-reviewed literature cited on that page.

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Contact: info@condorresearch.com
Supplier: Atrio Sciences s.r.o., Nitra (SK)

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PAIR 01 BPC-157 vs TB-500

Two of the most-studied tissue-repair peptides in preclinical research — chemically unrelated, mechanistically complementary. BPC-157 is a 15-aa gastric pentadecapeptide; TB-500 (as supplied by Condor Research) is full-length thymosin β4, a 43-aa actin-sequestering protein.

Full article: BPC-157 vs TB-500: A Research-Use Comparison · References 1–9 (#references)

Attribute	.....	..... (full-length Tβ4)
Chemical class	Synthetic pentadecapeptide (15 aa, gastric-sequence derived)	Full-length thymosin β4 (43 aa, actin-sequestering protein)
CAS / sequence	137525-51-0 · Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val	77591-33-4 · 43-amino-acid full-length Tβ4
Primary mechanism studied (preclinical)	Cytoprotection; FAK–paxillin-mediated fibroblast migration; angiogenic/vascular endpoints; GH-receptor upregulation in tendon fibroblasts[1,4] (#references)	G-actin sequestration; cytoskeletal remodelling; cell migration; angiogenesis via defined actin-binding motif[7,9] (#references)
Main preclinical research contexts	Tendon, ligament, muscle, GI, vascular models[2,3,5] (#references)	Wound healing, corneal/dermal repair, cardiac and cell-migration models[8,9] (#references)
Format (Condor Research)	10 mg lyophilised vial · also available as BPC-157 Arginate Capsules	10 mg lyophilised vial · also TB-500 Capsules
Co-formulated blend	BPC-157 + TB-500 (10 mg each); also in GLOW and KLOW blends	
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL ONLY Mostly rodent / in vitro; no robust human RCTs; not an approved drug	PRECLINICAL ONLY Mostly rodent / in vitro; thymosin β4 in some early clinical contexts; Tβ4 as RUO material lacks human efficacy data
How to choose	Connective-tissue repair, fibroblast behaviour, GI/vascular cytoprotection models	Actin dynamics, cell migration, angiogenesis, epithelial repair models

Matched on format and ≥99% HPLC purity; differentiated by chemical class, molecular size, and the research-model literature each peptide is primarily studied within.

PAIR 02

GHK-Cu vs AHK-Cu

Two copper-binding tripeptides separated by a single N-terminal residue substitution (Gly→Ala). GHK-Cu carries nearly five decades of preclinical literature; AHK-Cu is studied in a narrow set of in vitro and ex vivo models. Same histidine-anchored copper pocket; very different weight of evidence.

Full article: GHK-Cu vs AHK-Cu: A Research-Use Comparison of Two Copper-Binding Tripeptides · References 10–17 (#references)

Attribute	.....	.....
Chemical class	Copper(II) tripeptide complex	Copper(II) tripeptide complex
Sequence	Gly-His-Lys · Cu^{2+}	Ala-His-Lys · Cu^{2+}
CAS	49557-75-7	682809-81-0
Molecular formula / MW	$C_{14}H_{24}CuN_6O_4$ · 403.92 g/mol	$C_{15}H_{24}ClCuN_6O_4$ · 451.39 g/mol
Appearance	Blue-green lyophilised powder	Blue lyophilised powder
Primary mechanism studied (preclinical)	Copper delivery; matrix remodelling gene-expression models; cell signalling[11,12] (#references); Cu(II) binding thermodynamics well-characterised[13] (#references)	Hair-follicle and osteogenic in vitro models[14,15] (#references); coordination geometry studied in related Ala-His-His scaffold (family context, not direct evidence)[16] (#references)
Depth of literature	Extensive — decades, hundreds of papers; first identified 1977[10] (#references)	Limited — small number of in vitro/ex vivo studies
Format (Condor Research)	50 mg/vial · GHK-Cu Capsules	100 mg/vial · AHK-Cu Capsules
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL ONLY Rich preclinical corpus; limited randomised clinical data; not an approved drug in RUO context	PRECLINICAL ONLY Thin direct evidence; much inferred from related scaffolds; no clinical data
How to choose	Most-characterised copper-delivery peptide; comparative reference standard	N-terminal residue / coordination-geometry comparative chemistry; hair-follicle models

GHK-Cu carries a far larger and older preclinical literature. AHK-Cu is primarily studied as a structural analogue for comparative chemistry or in narrow follicle-model contexts. Both supplied at ≥99% HPLC purity with COA.

PAIR 03 Ipamorelin vs CJC-1295 (no DAC)

Two GH-axis secretagogues acting through different receptors. Ipamorelin is a selective GHS-R1a (ghrelin-receptor) pentapeptide agonist; CJC-1295 (no DAC / Modified GRF 1-29) is a GHRH-receptor analogue. Often studied in parallel because the receptor-level distinction is precisely the research question.

Full article: Ipamorelin vs CJC-1295: Two Routes Into the Growth-Hormone Axis · References [18–25] (#references)

Attribute		
Chemical class	Pentapeptide GHS-R1a agonist (ghrelin mimetic)	GHRH-receptor analogue (Modified GRF 1-29); CAS 863288-34-0
Sequence	Aib-His-D-2-Nal-D-Phe-Lys-NH_2	Modified GRF(1-29) with D-Ala² substitution
Receptor target	GHS-R1a (ghrelin receptor) — pituitary[18] (#references)	GHRH receptor — pituitary[21] (#references)
Key selectivity feature	Does not significantly release cortisol, ACTH, or prolactin at studied doses[18] (#references)	Lacks DAC albumin-binding group; shorter circulating half-life than CJC-1295 DAC[21] (#references)
Primary mechanism studied (preclinical)	GHS-R1a activation → GH pulse; GI motility models (rodent postoperative ileus)[20] (#references)	GHRH-R activation → GH/IGF-1 axis; pulsatility preservation; GH-knockout rescue[23,25] (#references)
Human data available?	Pharmacokinetic and limited PD work; no robust human efficacy RCTs[19] (#references)	Small human studies report GH/IGF-1 responses and preserved pulsatility[22,23] (#references) — not a drug approval basis
Format (Condor Research)	10 mg lyophilised vial	10 mg lyophilised vial
Co-formulated blend	CJC-1295 no DAC + Ipamorelin (10 mg each)	
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
WADA status	Prohibited in-competition as GH secretagogue (S2)	Prohibited in-competition as GH secretagogue (S2)
Evidence quality	PREDOMINANTLY PRECLINICAL Pharmacokinetics described; no approved-drug status	EARLY HUMAN DATA Small human GH/IGF-1 studies; not approved as a drug; extrapolation to clinical efficacy unsupported
How to choose	GHS-R1a biology; selective ghrelin-receptor agonism; GI motility models	GHRH-receptor biology; pulsatility questions; GH/IGF-1 axis pharmacology

Different receptors, complementary axis — which is why they appear together in co-formulated blends. The research choice follows the receptor target, not a potency ranking.

PAIR 04

Selank vs Semax

Two Russian regulatory-lineage neuropeptides with a shared BDNF/neurotrophin signal in some models, but distinct parent sequences and the preclinical mechanisms primarily studied diverge: Selank (tuftsin analogue) into GABAergic / anxiolytic models; Semax (ACTH(4-10) analogue) into neuroprotection and cognitive models.

Full article: Selank vs Semax: A Research-Use Comparison of Two Russian Regulatory Neuropeptides · References [26–35] (#references)

Attribute	.....	.....
Chemical class	Synthetic heptapeptide analogue of tuftsin (Thr-Lys-Pro-Arg-Pro-Gly-Pro)	Synthetic hexapeptide analogue of ACTH(4-10) (Met-Glu-His-Phe-Pro-Gly-Pro)
Parent / origin	Tuftsin (immunopeptide Thr-Lys-Pro-Arg) + stabilising Pro-Gly-Pro extension	ACTH(4-10) core with C-terminal Pro-Gly-Pro extension for stability
Primary mechanism studied (preclinical)	GABAergic modulation[27] (#references); anxiolytic effects in animal models[26,28,29] (#references); BDNF interaction in hippocampus[28] (#references)	Neuroprotection in cerebral ischaemia models[35] (#references); BDNF/neurotrophin modulation; anti-depressant-like effects in chronic stress models[33] (#references); Aβ aggregation interference (in vitro)[31] (#references)
Regulatory status (Russia)	Registered anxiolytic in Russia	Registered nootropic / neuroprotectant in Russia
Shared model overlap	Both studied using functional connectomic approaches in the same paper[34] (#references); some BDNF pathway overlap in rodent work	
Co-formulated blend	Semax + Selank (combination vial)	
Format (Condor Research)	Vial (lyophilised)	Vial (lyophilised)
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL + LIMITED CLINICAL Rodent models; one small Russian anxiolytic trial[30] (#references); not approved outside Russia in RUO context	PRECLINICAL + LIMITED CLINICAL Rodent neuroprotection; small Russian stroke study[35] (#references); not approved outside Russia in RUO context
How to choose	GABAergic / anxiety / stress-model biology	Neuroprotection / cognitive / ischaemia-model biology

Both carry a Russian regulatory lineage not recognised by EMA/FDA. All human observations cited above are from small, non-randomised or Russian-only studies and do not establish EMA/FDA-standard efficacy.

PAIR 05

NMN vs NAD+

One enzymatic step apart on the same salvage pathway: NMN is the mononucleotide precursor; NAD+ is the finished dinucleotide coenzyme. The research choice follows where in the pathway the experiment needs to intervene, not which molecule is intrinsically superior.

Full article: NMN vs NAD+: A Research-Use Comparison of NAD Metabolism Precursors · References [36–45] (#references)

Attribute	.....	.....
Chemical class	Mononucleotide — NAD+ precursor (salvage pathway substrate)	Dinucleotide — redox coenzyme and signalling substrate
Full name / CAS	β-Nicotinamide mononucleotide · 1094-61-7	Nicotinamide adenine dinucleotide · 53-84-9
Molecular formula	$C_{11}H_{16}N_2O_8P$	$C_{21}H_{27}N_7O_{14}P_2$
Pathway position	Substrate — one NMNAT-catalysed adenylylation upstream of NAD+[36] (#references)	Product — central redox coenzyme and sirtuin/CD38/PARP substrate[36,37] (#references)
Primary mechanism studied (preclinical)	Precursor supplementation → NAD+ pool elevation; SIRT/PARP activation; metabolic and mitochondrial endpoints in aged rodents[38,39] (#references)	Redox electron transfer; direct sirtuin/CD38 substrate; mitochondrial function; age-related NAD+ decline models[37] (#references)
Notable human data	Single-dose tolerability and metabolite changes[43] (#references); one RCT in prediabetic women: increased muscle insulin sensitivity[42] (#references); systematic review: physical performance not statistically significant[44] (#references)	Less direct human supplementation literature in RUO context; NAD+ precursor class reviewed in Nat Metab 2025[45] (#references)
Format (Condor Research)	60 HPMC (vegan) capsules · 500 mg each	1,000 mg/vial · white lyophilised powder
Handling	Capsule — no reconstitution required	Reconstitute with sterile water for solution-based bench work
Purity / QC	≥99% HPLC; COA available	≥99% HPLC; third-party tested; COA
Evidence quality	EARLY HUMAN DATA (INCONSISTENT) Rich rodent mechanism; promising but unsettled early human dataset	PREDOMINANTLY PRECLINICAL Strong cell/biochemistry base; limited direct human supplementation RCTs in RUO context
How to choose	Oral-availability / pharmacokinetic animal models; precursor-leverage questions	Bench reconstitution; enzyme assays requiring intact coenzyme; direct NAD+-delivery models

Format follows function: capsules for ingestion-route animal models, lyophilised vial for bench reconstitution. The human evidence base for both is early and unsettled — the honest reading is that the preclinical signal has not been matched by a settled clinical picture.

PAIR 06

Epitalon vs Pinealon

Two short Khavinson-class peptide bioregulators from the pineal lineage. Epitalon (AEDG, tetrapeptide) is studied mainly in telomere, pineal-ageing, and lifespan models. Pinealon (EDR, tripeptide) is investigated for neuronal antioxidant and gene-expression endpoints. Both share a Russian research origin with limited independent replication.

Full article: Epitalon vs Pinealon: A Research-Use Comparison of Two Khavinson Bioregulator Peptides · References 46–53 (#references)

Attribute	.....	.....
Chemical class	Synthetic tetrapeptide (AEDG) — Ala-Glu-Asp-Gly	Synthetic tripeptide (EDR) — Glu-Asp-Arg
Khavinson class	"Cytogen" / pineal bioregulator — telomere-focused[46,48] (#references)	"Cytogen" / pineal bioregulator — neuro-antioxidant and gene-expression focused[50,51] (#references)
Primary mechanism studied (preclinical)	Telomerase activation and telomere elongation in human somatic cells (in vitro)[46] (#references); reduced tumour incidence and extended lifespan in rodent models[48] (#references); pineal-ageing marker modulation[47,49] (#references)	Increased cell viability; suppression of free radical levels in vitro[50] (#references); gene-expression regulation in Alzheimer's-related models[51] (#references); neuroprotective endpoints alongside structurally related EDR-class peptides[52] (#references); prenatal neuroprotection in rat offspring[53] (#references)
Replication / independence note	Primarily Khavinson-group publications; limited independent replication	Primarily Khavinson-group publications; very few independent replications
Format (Condor Research)	Vial (lyophilised) · Epitalon Capsules	Vial (lyophilised) · Pinealon Capsules
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL ONLY Cell / rodent work; single-group provenance; not an approved drug; no human RCTs	PRECLINICAL ONLY Cell / rodent work; single-group provenance; not an approved drug; no human RCTs
How to choose	Telomerase / telomere-length biology; lifespan-biomarker models	Neuronal antioxidant / gene-expression models; EDR-class peptide biology

Single-group provenance (Khavinson et al.) is the primary caveat for both peptides. The mechanisms are plausible but require broader independent replication. For laboratory research use only.

PAIR 07

5-Amino-1MQ vs SLU-PP-332

Two unrelated metabolic-research tool compounds that converge on exercise-mimetic and NAD+-related biology by entirely different mechanisms: 5-Amino-1MQ inhibits NNMT (a methyltransferase regulating NAD+ and methyl donors); SLU-PP-332 agonises ERRα/β/γ nuclear receptors to activate mitochondrial gene programmes. Opposite logic — inhibitor vs agonist.

Full article: 5-Amino-1MQ vs SLU-PP-332: Inhibitor versus Agonist in Metabolic Research · References 54–63 (#references)

Attribute	.....	.....
Chemical class	Small-molecule NNMT inhibitor (quinoline derivative)	Synthetic pan-ERRα/β/γ nuclear-receptor agonist
Target	Nicotinamide N-methyltransferase (NNMT) — enzyme inhibition[54,55] (#references)	Estrogen-related receptors α, β, γ (ERR) — nuclear receptor agonism[58,59] (#references)
Mechanistic logic	Inhibiting NNMT preserves NAD+ and methyl-donor availability; modulates adipose differentiation and metabolic gene expression[55] (#references)	ERR agonism activates PGC-1α-related mitochondrial biogenesis and oxidative-metabolism gene programmes — an "exercise mimetic" approach[58,59] (#references)
Primary preclinical model contexts	High-fat-diet obese rodents; HeLa anti-proliferation (in vitro)[56] (#references); cancer-associated fibroblast models[57] (#references); pancreatic fibrosis (rodent)[63] (#references)	Aerobic exercise capacity in mice[58] (#references); metabolic syndrome models[59] (#references); orally active SLU-PP-915 analogue data[60] (#references)
Format (Condor Research)	Vial (lyophilised) · 5-Amino-1MQ Capsules	Vial (lyophilised) · SLU-PP-332 Capsules
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL ONLY Rodent metabolic models; in vitro; no human RCTs; not an approved drug	PRECLINICAL ONLY Rodent exercise-capacity models; very early tool-compound status; no human data
How to choose	NNMT biology; NAD+/methyl-donor axis; adipogenesis; cancer fibroblast metabolism	ERR nuclear-receptor biology; mitochondrial biogenesis; exercise-mimetic model design

5-Amino-1MQ inhibits (enzyme blocker, indirectly raises NAD+); SLU-PP-332 agonises (nuclear receptor activator, turns on mitochondrial gene programmes). Opposite logic, overlapping metabolic-research space.

SUPP.

Tesofensine vs Enclomiphene

An atypical pair: different target classes, different research questions, included on the site as "metabolic / hormonal axis" comparators. Tesofensine is a triple monoamine reuptake inhibitor studied in CNS and energy-balance models; Enclomiphene is the trans-isomer of clomiphene, a SERM studied in HPG-axis and gonadotropin models.

Full article: Tesofensine vs Enclomiphene: A Research-Use Comparison · References 64–71 (#references)

Attribute		
Chemical class	Triple monoamine reuptake inhibitor (5-HT, DA, NE)	Selective estrogen receptor modulator (SERM) — trans-isomer of clomiphene
Target	SERT, DAT, NET (serotonin, dopamine, norepinephrine transporters)[64] (#references)	Estrogen receptor (ER antagonist) → HPG axis, LH/FSH release[68] (#references)
Primary mechanism studied (preclinical)	Appetite suppression via α1-adrenoceptor and D1R pathways in DIO rats[65] (#references); improved glycaemic control vs sibutramine/rimonabant[66] (#references); hypothalamic GABAergic neuron silencing[67] (#references)	ER antagonism → increased LH/FSH → endogenous testosterone in hypogonadal models[68,69] (#references); preserves sperm counts vs topical testosterone (clinical observation)[70] (#references)
Format (Condor Research)	Capsules	Capsules
Purity / QC	≥99% HPLC; third-party COA (CZ lab)	≥99% HPLC; third-party COA (CZ lab)
Evidence quality	PRECLINICAL + PHASE II Reached Phase II obesity trial; not approved; no EMA/FDA marketing authorisation	EARLY HUMAN DATA Small clinical studies in secondary hypogonadism; not approved by EMA; IND-exempt in some jurisdictions — supplied here strictly as RUO reference material
How to choose	CNS monoamine reuptake biology; energy-balance and obesity-model research	HPG-axis biology; gonadotropin / testosterone-axis research models

Different targets, different axes. Paired on the site as metabolic/hormonal comparators for researchers navigating body-composition adjacent compound selection. Not therapeutic guidance.

How to Use These Tables

Each table row consolidates specification data already published on condorresearch.com/research/. No new claims are made. The "Evidence quality" row is the most important: every compound listed here is at the preclinical stage or early human-data stage in the research context in which it is supplied. No approved-drug status applies to any compound in the RUO context supplied here.

Click the article link in each pair header to read the full analysis, including mechanism sections, evidence-quality discussion, and the complete references list with PMIDs and DOIs. COA documents are accessible from individual product pages at condorresearch.com.

Frequently Asked Questions

What does 'research use only' mean for these compounds?

Research Use Only (RUO) means the compounds are supplied exclusively for qualified in vitro, cell-culture, and animal-model laboratory work. They are not approved drugs, cosmetics, supplements, or food additives. No therapeutic, diagnostic, or veterinary use is implied or permitted. See the full Research Use Only Disclaimer (https://condorresearch.com/research-use-only-disclaimer/).

Are any of these compounds approved for human use?

No compound listed in this document is approved for human administration in the context it is sold here. Some parent molecules have approved pharmaceutical forms in other jurisdictions (e.g., afamelanotide, tesamorelin, semax in Russia), but those approvals are entirely separate from the RUO reference materials supplied by Condor Research.

How is purity verified across all compounds?

All Condor Research compounds are characterised at ≥99% purity by HPLC and/or mass spectrometry. Third-party, independent batch testing is conducted in the Czech Republic. Lot-specific Certificates of Analysis (COA) are linked from each product page. For the testing framework overview, see Quality & Third-Party Testing (https://condorresearch.com/quality-and-third-party-testing/).

Can I combine compounds from different comparison pairs in a single study design?

Combination questions follow the experimental design — researchers define readouts, controls, and statistical power independently. Condor Research supplies co-formulated blends (BPC-157 + TB-500, CJC-1295 no DAC + Ipamorelin, GLOW, KLOW) for convenience, but any combination study should be designed and powered as its own experiment. No therapeutic rationale is stated or implied.

Where can I find lot-specific COA data?

Each product page on condorresearch.com links to its current COA. The Quality & Third-Party Testing (https://condorresearch.com/quality-and-third-party-testing/) page provides a centralised overview of the testing framework used for all compounds.

How current are these comparison tables?

Data were consolidated from content published on condorresearch.com in 2025–2026 and reflect the peer-reviewed literature cited on each comparison page. Individual pages carry the date of last review. This document was compiled June 2026.

References

1. Chang CH et al. The promoting effect of pentadecapeptide BPC 157 on tendon healing involves tendon outgrowth, cell survival, and cell migration. *J Appl Physiol.* 2011;110(3):774–780. PMID 21030672 (https://pubmed.ncbi.nlm.nih.gov/21030672/)
2. Cerovecki T et al. Pentadecapeptide BPC 157 (PL 14736) improves ligament healing in the rat. *J Orthop Res.* 2010;28(9):1155–1161. PMID 20225319 (https://pubmed.ncbi.nlm.nih.gov/20225319/)
3. Novinscak T et al. Gastric pentadecapeptide BPC 157 as an effective therapy for muscle crush injury in the rat. *Surg Today.* 2008;38(8):716–725. PMID 18668315 (https://pubmed.ncbi.nlm.nih.gov/18668315/)
4. Chang CH et al. Pentadecapeptide BPC 157 enhances the growth hormone receptor expression in tendon fibroblasts. *Molecules.* 2014;19(11):19066–19077. PMID 25415472 (https://pubmed.ncbi.nlm.nih.gov/25415472/)
5. Sikiric P et al. Cytoprotective gastric pentadecapeptide BPC 157 resolves major vessel occlusion disturbances. *World J Gastroenterol.* 2022;28(1):23–46. PMID 35125818 (https://pubmed.ncbi.nlm.nih.gov/35125818/)
6. Sikiric P et al. The Stable Gastric Pentadecapeptide BPC 157 Pleiotropic Beneficial Activity. *Pharmaceuticals.* 2024;17(4):461. PMID 38675421 (https://pubmed.ncbi.nlm.nih.gov/38675421/)
7. Goldstein AL et al. Thymosin beta4: actin-sequestering protein moonlights to repair injured tissues. *Trends Mol Med.* 2005;11(9):421–429. PMID 16099219 (https://pubmed.ncbi.nlm.nih.gov/16099219/)
8. Philp D, Kleinman HK. Animal studies with thymosin beta, a multifunctional tissue repair and regeneration peptide. *Ann N Y Acad Sci.* 2010;1194:81–86. PMID 20536453 (https://pubmed.ncbi.nlm.nih.gov/20536453/)
9. Philp D et al. The actin binding site on thymosin beta4 promotes angiogenesis. *FASEB J.* 2003;17(14):2103–2105. PMID 14500546 (https://pubmed.ncbi.nlm.nih.gov/14500546/)
10. Schlesinger DH et al. Growth-modulating serum tripeptide is glycyl-histidyl-lysine. *Experientia.* 1977;33(3):324–325. PMID 858356 (https://pubmed.ncbi.nlm.nih.gov/858356/)
11. Pickart L et al. GHK Peptide as a Natural Modulator of Multiple Cellular Pathways in Skin Regeneration. *Biomed Res Int.* 2015;2015:648108. PMID 26236730 (https://pubmed.ncbi.nlm.nih.gov/26236730/)
12. Pickart L, Margolina A. Regenerative and Protective Actions of the GHK-Cu Peptide in the Light of the New Gene Data. *Int J Mol Sci.* 2018;19(7):1987. PMID 29986520 (https://pubmed.ncbi.nlm.nih.gov/29986520/)
13. Trapaidze A et al. Thermodynamic study of Cu2+ binding to the DAHK and GHK peptides by isothermal titration calorimetry. *J Biol Inorg Chem.* 2012;17(1):37–47. PMID 21898044 (https://pubmed.ncbi.nlm.nih.gov/21898044/)
14. Pyo HK et al. The effect of tripeptide-copper complex on human hair growth in vitro. *Arch Pharm Res.* 2007;30(7):834–839. PMID 17703734 (https://pubmed.ncbi.nlm.nih.gov/17703734/)
15. Jung JI et al. Vitamin C-linker-conjugated tripeptide AHK stimulates BMP-2-induced osteogenic differentiation. *Differentiation.* 2018;101:1–7. PMID 29567599 (https://pubmed.ncbi.nlm.nih.gov/29567599/)

16. Gonzalez P et al. Why the Ala-His-His Peptide Is an Appropriate Scaffold to Remove and Redox Silence Copper Ions. *Biomolecules.* 2022;12(10):1327. [Related scaffold; family context for AHK-Cu.] PMID 36291536 (https://pubmed.ncbi.nlm.nih.gov/36291536/)

17. Mehr A et al. The copper(II)-binding tripeptide GHK, a valuable crystallization and phasing tag. *Acta Crystallogr D.* 2020;76(Pt 12):1222–1232. PMID 33263328 (https://pubmed.ncbi.nlm.nih.gov/33263328/)

18. Raun K et al. Ipamorelin, the first selective growth hormone secretagogue. *Eur J Endocrinol.* 1998;139(5):552–561. PMID 9849822 (https://pubmed.ncbi.nlm.nih.gov/9849822/)

19. Johansen PB et al. Pharmacokinetic evaluation of ipamorelin and other peptidyl growth hormone secretagogues. *Xenobiotica.* 1998;28(11):1083–1092. PMID 9879640 (https://pubmed.ncbi.nlm.nih.gov/9879640/)

20. Venkova K et al. Efficacy of ipamorelin, a novel ghrelin mimetic, in a rodent model of postoperative ileus. *J Pharmacol Exp Ther.* 2009;329(3):1110–1116. PMID 19289567 (https://pubmed.ncbi.nlm.nih.gov/19289567/)

21. Jetté L et al. Human GRF1-29-albumin bioconjugates activate the GRF receptor; identification of CJC-1295 as a long-lasting GRF analog. *Endocrinology.* 2005;146(7):3052–3058. PMID 15817669 (https://pubmed.ncbi.nlm.nih.gov/15817669/)

22. Teichman SL et al. Prolonged stimulation of GH and IGF-I secretion by CJC-1295 in healthy adults. *J Clin Endocrinol Metab.* 2006;91(3):799–805. PMID 16352683 (https://pubmed.ncbi.nlm.nih.gov/16352683/)

23. Ionescu M, Frohman LA. Pulsatile secretion of GH persists during continuous stimulation by CJC-1295. *J Clin Endocrinol Metab.* 2006;91(12):4792–4797. PMID 17018654 (https://pubmed.ncbi.nlm.nih.gov/17018654/)

24. Sackmann-Sala L et al. Activation of the GH/IGF-1 axis by CJC-1295 results in serum protein profile changes. *Growth Horm IGF Res.* 2009;19(6):471–477. PMID 19386527 (https://pubmed.ncbi.nlm.nih.gov/19386527/)

25. Alba M et al. Once-daily administration of CJC-1295 normalizes growth in the GHRH knockout mouse. *Am J Physiol Endocrinol Metab.* 2006;291(6):E1290–E1294. PMID 16822960 (https://pubmed.ncbi.nlm.nih.gov/16822960/)

26. Vyunova TV et al. Peptide-based Anxiolytics: The Molecular Aspects of Heptapeptide Selank Biological Activity. *Protein Pept Lett.* 2018;25(10):914–923. PMID 30255741 (https://pubmed.ncbi.nlm.nih.gov/30255741/)

27. Filatova E et al. GABA, Selank, and Olanzapine Affect the Expression of Genes Involved in GABAergic Neurotransmission in IMR-32 Cells. *Front Pharmacol.* 2017;8:89. PMID 28293190 (https://pubmed.ncbi.nlm.nih.gov/28293190/)

28. Kolik LG et al. Selank, Peptide Analogue of Tuftsin, Protects Against Ethanol-Induced Memory Impairment by Regulating BDNF Content. *Bull Exp Biol Med.* 2019;167(5):641–644. PMID 31625062 (https://pubmed.ncbi.nlm.nih.gov/31625062/)

29. Kasian A et al. Peptide Selank Enhances the Effect of Diazepam in Reducing Anxiety in Unpredictable Chronic Mild Stress Conditions in Rats. *Behav Neurol.* 2017;2017:5091027. PMID 28280289 (https://pubmed.ncbi.nlm.nih.gov/28280289/)

30. Zozulya AA et al. Efficacy and possible mechanisms of action of a new peptide anxiolytic selank in the therapy of generalized anxiety disorders. *Zh Nevrol Psikhiatr.* 2008;108(4):38–48. PMID 18454096 (https://pubmed.ncbi.nlm.nih.gov/18454096/)

31. Sciacca MFM et al. Semax, a Synthetic Regulatory Peptide, Affects Copper-Induced Aβ Aggregation and Amyloid Formation. *ACS Chem Neurosci.* 2022;13(4):486–496. PMID 35080861 (https://pubmed.ncbi.nlm.nih.gov/35080861/)

32. Stavchansky VV et al. Insight into Glyproline Peptides' Activity through the Modulation of Inflammatory and Neurosignaling Genetic Response Following Cerebral Ischemia-Reperfusion. *Genes (Basel).* 2022;13(12):2380. PMID 36553646 (https://pubmed.ncbi.nlm.nih.gov/36553646/)

33. Inozemtseva LS et al. Antidepressant-like and antistress effects of the ACTH(4-10) synthetic analogs Semax and Melanotan II on male rats. *Eur J Pharmacol.* 2024;984:177068. PMID 39442746 (https://pubmed.ncbi.nlm.nih.gov/39442746/)

34. Panikratova YR et al. Functional Connectomic Approach to Studying Selank and Semax Effects. *Dokl Biol Sci.* 2020;490(1):9–11. PMID 32342318 (https://pubmed.ncbi.nlm.nih.gov/32342318/)

35. Gusev EI et al. The efficacy of semax in the treatment of patients at different stages of ischemic stroke. *Zh Nevrol Psikhiatr.* 2018;118(3 Pt 2):61–68. PMID 29798983 (https://pubmed.ncbi.nlm.nih.gov/29798983/)

36. Covarrubias AJ et al. NAD+ metabolism and its roles in cellular processes during ageing. *Nat Rev Mol Cell Biol.* 2021;22(2):119–141. PMID 33353981 (https://pubmed.ncbi.nlm.nih.gov/33353981/)

37. Verdin E. NAD+ in aging, metabolism, and neurodegeneration. *Science.* 2015;350(6265):1208–1213. PMID 26785480 (https://pubmed.ncbi.nlm.nih.gov/26785480/)

38. Mills KF et al. Long-Term Administration of Nicotinamide Mononucleotide Mitigates Age-Associated Physiological Decline in Mice. *Cell Metab.* 2016;24(6):795–806. PMID 28068222 (https://pubmed.ncbi.nlm.nih.gov/28068222/)

39. Rajman L et al. Therapeutic Potential of NAD-Boosting Molecules: The In Vivo Evidence. *Cell Metab.* 2018;27(3):529–547. PMID 29514064 (https://pubmed.ncbi.nlm.nih.gov/29514064/)

40. Grozio A et al. Slc12a8 is a nicotinamide mononucleotide transporter. *Nat Metab.* 2019;1(1):47–57. PMID 31131364 (https://pubmed.ncbi.nlm.nih.gov/31131364/)

41. Yoshino M et al. Nicotinamide mononucleotide increases muscle insulin sensitivity in prediabetic women. *Science.* 2021;372(6547):1224–1229. PMID 33888596 (https://pubmed.ncbi.nlm.nih.gov/33888596/)

42. Irie J et al. Effect of oral administration of nicotinamide mononucleotide on clinical parameters and nicotinamide metabolite levels. *Endocr J.* 2020;67(2):153–160. PMID 31685720 (https://pubmed.ncbi.nlm.nih.gov/31685720/)

43. Wen J et al. Improved Physical Performance Parameters in Patients Taking NMN: A Systematic Review of Randomized Control Trials. *Cureus.* 2024;16(8):e65961. PMID 39221308 (https://pubmed.ncbi.nlm.nih.gov/39221308/)

44. Vinten KT et al. NAD+ precursor supplementation in human ageing: clinical evidence and challenges. *Nat Metab.* 2025;7(10):1974–1990. PMID 41083806 (https://pubmed.ncbi.nlm.nih.gov/41083806/)

45. Khavinson VKh et al. Epithalon peptide induces telomerase activity and telomere elongation in human somatic cells. *Bull Exp Biol Med.*

2003;135(6):590–592. PMID 12937682 (https://pubmed.ncbi.nlm.nih.gov/12937682/)

46. Khavinson VKh. Peptides and Ageing. *Neuro Endocrinol Lett.* 2002;23 Suppl 3:11–144. PMID 12374906 (https://pubmed.ncbi.nlm.nih.gov/12374906/)

47. Anisimov VN et al. Effect of Epitalon on biomarkers of aging, life span and spontaneous tumor incidence in female Swiss-derived SHR mice. *Biogerontology.* 2003;4(4):193–202. PMID 14501183 (https://pubmed.ncbi.nlm.nih.gov/14501183/)

48. Khavinson VKh, Morozov VG. Peptides of pineal gland and thymus prolong human life. *Neuro Endocrinol Lett.* 2003;24(3-4):233–240. PMID 14523363 (https://pubmed.ncbi.nlm.nih.gov/14523363/)

49. Khavinson V et al. Pinealon increases cell viability by suppression of free radical levels and activating proliferative processes. *Rejuvenation Res.* 2011;14(5):535–541. PMID 21978084 (https://pubmed.ncbi.nlm.nih.gov/21978084/)

50. Khavinson V et al. EDR Peptide: Possible Mechanism of Gene Expression and Protein Synthesis Regulation Involved in the Pathogenesis of Alzheimer's Disease. *Molecules.* 2020;26(1):159. PMID 33396470 (https://pubmed.ncbi.nlm.nih.gov/33396470/)

51. Khavinson V et al. Neuroprotective Effects of Tripeptides-Epigenetic Regulators in Mouse Model of Alzheimer's Disease. *Pharmaceuticals (Basel).* 2021;14(6):515. PMID 34071923 (https://pubmed.ncbi.nlm.nih.gov/34071923/)

52. Arutjunyan A et al. Pinealon protects the rat offspring from prenatal hyperhomocysteinemia. *Int J Clin Exp Med.* 2012;5(2):179–185. PMID 22567179 (https://pubmed.ncbi.nlm.nih.gov/22567179/)

53. Neelakantan H et al. Selective and membrane-permeable small molecule inhibitors of nicotinamide N-methyltransferase reverse high fat diet-induced obesity in mice. *Biochem Pharmacol.* 2018;147:141–152. PMID 29155147 (https://pubmed.ncbi.nlm.nih.gov/29155147/)

54. Kraus D et al. Nicotinamide N-methyltransferase knockdown protects against diet-induced obesity. *Nature.* 2014;508(7495):258–262. PMID 24717514 (https://pubmed.ncbi.nlm.nih.gov/24717514/)

55. Akar S et al. Small molecule inhibitor of nicotinamide N-methyltransferase shows anti-proliferative activity in HeLa cells. *J Obstet Gynaecol.* 2021;41(8):1240–1245. PMID 33645410 (https://pubmed.ncbi.nlm.nih.gov/33645410/)

56. Yang M et al. NAD+ metabolism enzyme NNMT in cancer-associated fibroblasts drives tumor progression. *J Immunother Cancer.* 2024;12(7):e009281. PMID 39067875 (https://pubmed.ncbi.nlm.nih.gov/39067875/)

57. Ren N et al. NNMT Inhibition Mitigates Cerulein-Induced Pancreatic Fibrosis. *Inflammation.* 2026;49(1):135. PMID 41904718 (https://pubmed.ncbi.nlm.nih.gov/41904718/)

58. Billon C et al. Synthetic ERRα/β/γ Agonist Induces an ERRα-Dependent Acute Aerobic Exercise Response. *ACS Chem Biol.* 2023;18(4):756–771. PMID 36988910 (https://pubmed.ncbi.nlm.nih.gov/36988910/)

59. Billon C et al. A Synthetic ERR Agonist Alleviates Metabolic Syndrome. *J Pharmacol Exp Ther.* 2024;388(2):232–240. PMID 37739806 (https://pubmed.ncbi.nlm.nih.gov/37739806/)

60. Billon C et al. An orally active estrogen receptor-related receptor agonist, SLU-PP-915, enhances aerobic exercise capacity. *J Pharmacol Exp Ther.* 2026;393(1):103787. PMID 41421047 (https://pubmed.ncbi.nlm.nih.gov/41421047/)

61. de Souza-Lima J et al. Pharmacological Activation of ERRα/β/γ as an Exercise Mimetic. *Rev Med Chil.* 2026;154(2):237–245. PMID 42024694 (https://pubmed.ncbi.nlm.nih.gov/42024694/)

62. Prajapati S et al. Nicotinamide N-Methyltransferase (NNMT): A Central Regulator in Hepatocellular Carcinoma Metabolism — A Systematic Review. *Curr Drug Metab.* 2026 Jun 4. PMID 42260786 (https://pubmed.ncbi.nlm.nih.gov/42260786/)

63. Bello NT, Zahner MR. Tesofensine, a monoamine reuptake inhibitor for the treatment of obesity. *Curr Opin Investig Drugs.* 2009;10(10):1105–1116. PMID 19777399 (https://pubmed.ncbi.nlm.nih.gov/19777399/)

64. Axel AM et al. Tesofensine, a novel triple monoamine reuptake inhibitor, induces appetite suppression by indirect stimulation of alpha1 adrenoceptor and dopamine D1 receptor pathways. *Neuropsychopharmacology.* 2010;35(7):1464–1476. PMID 20200509 (https://pubmed.ncbi.nlm.nih.gov/20200509/)

65. Hansen HH et al. The novel triple monoamine reuptake inhibitor tesofensine induces sustained weight loss and improves glycaemic control. *Eur J Pharmacol.* 2010;636(1-3):88–95. PMID 20385125 (https://pubmed.ncbi.nlm.nih.gov/20385125/)

66. Perez CI et al. Tesofensine, a novel antiobesity drug, silences GABAergic hypothalamic neurons. *PLoS One.* 2024;19(4):e0300544. PMID 38656972 (https://pubmed.ncbi.nlm.nih.gov/38656972/)

67. Hill S et al. Enclomiphene, an estrogen receptor antagonist for the treatment of testosterone deficiency in men. *IDrugs.* 2009;12(2):109–119. PMID 19204885 (https://pubmed.ncbi.nlm.nih.gov/19204885/)

68. Rodriguez KM et al. Enclomiphene citrate for the treatment of secondary male hypogonadism. *Expert Opin Pharmacother.* 2016;17(11):1561–1567. PMID 27337642 (https://pubmed.ncbi.nlm.nih.gov/27337642/)

69. Kim ED et al. Oral enclomiphene citrate raises testosterone and preserves sperm counts in obese hypogonadal men. *BJU Int.* 2016;117(4):677–685. PMID 26496621 (https://pubmed.ncbi.nlm.nih.gov/26496621/)

70. Tienforti D et al. Selective modulation of estrogen receptor in obese men with androgen deficiency: A systematic review and meta-analysis. *Andrology.* 2023. PMID 36604313 (https://pubmed.ncbi.nlm.nih.gov/36604313/)