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+ Peptide Applications Quick Reference

A researcher's field guide to peptide categories, mechanisms, and therapeutic research areas. Mechanism, application domain, and clinical evidence at a glance.

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+ How to Use This Reference

This guide organizes peptides by their primary research application domain. Each entry includes the mechanism of action, sequence characteristics, key clinical evidence, and research considerations. Citations are drawn exclusively from peer-reviewed literature and major clinical trial publications.

RESEARCH-USE ONLY DISCLAIMER

All peptides discussed in this reference are intended strictly for laboratory research purposes. None are approved for human consumption, diagnosis, treatment, or prevention of disease unless explicitly stated. Researchers must comply with all applicable institutional and regulatory requirements.

Category Color Key

CATEGORY	TAG	RESEARCH FOCUS
Metabolic Research	METABOLIC	GLP-1, GIP, dual/triple agonists — glucose regulation, weight management
Recovery & Repair	RECOVERY	Tissue healing, musculoskeletal regeneration, anti-inflammatory mechanisms
Performance Research	PERFORMANCE	Growth hormone pathways, body composition, metabolic enhancement
Cognitive Research	COGNITIVE	Nootropic mechanisms, neuroprotection, synaptic plasticity
Longevity Research	LONGEVITY	Telomerase, cellular senescence, anti-aging pathways
Cosmetic Research	COSMETIC	Skin remodeling, collagen synthesis, dermatological application

+ Metabolic Research

Peptides in this category modulate glucose homeostasis, insulin secretion, appetite regulation, and energy metabolism. The GLP-1 receptor agonist class has produced landmark clinical results in diabetes and obesity, with dual and triple agonists representing the next wave of therapeutic innovation.

Semaglutide GLP-1 RA

METABOLIC

Mechanism: Long-acting GLP-1 receptor agonist; enhances glucose-dependent insulin secretion, suppresses glucagon, delays gastric emptying, reduces appetite via central mechanisms.

Key Evidence: SELECT trial (n=17,604): 20% reduction in MACE vs. placebo in patients with obesity and CVD (HR 0.80, 95% CI 0.72-0.90; P<0.001).[1] STEP 1: mean weight loss 15.2% at 68 weeks.[2]

Research Notes: Long half-life (approx. 7 days) enables once-weekly dosing. Investigate sustained vs. pulsatile receptor activation effects.

Tirzepatide GIP/GLP-1 RA

METABOLIC

Mechanism: Dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist. Combines incretin effects with adipose tissue insulin sensitization.

Key Evidence: SURPASS-2: superior HbA1c reduction vs. semaglutide 1 mg (mean difference -0.5%). SURMOUNT-1: up to 20.9% mean weight loss at 15 mg dose.[3][4]

Research Notes: Dual mechanism may offer advantages in adipose tissue remodeling. Monitor GI tolerability profile differences vs. selective GLP-1 RAs.

Liraglutide GLP-1 RA

METABOLIC

Mechanism: Acylated GLP-1 analogue with 97% sequence homology to native GLP-1. Albumin binding extends half-life to 13 hours, enabling once-daily dosing.

Key Evidence: LEADER trial: 13% reduction in MACE in T2DM with high CV risk. SCALE Obesity and Prediabetes: 8.0% mean weight loss at 3.0 mg.[5]

Research Notes: Shorter-acting than semaglutide; useful for comparing continuous vs. intermittent GLP-1 receptor occupancy effects.

Exenatide GLP-1 RA

METABOLIC

Mechanism: Synthetic exendin-4 analogue; GLP-1 receptor agonist resistant to DPP-4 degradation. First-in-class GLP-1 RA for T2DM.

Key Evidence: Isolated from *Heloderma suspectum* venom. Approved for T2DM 2005; extended-release formulation approved 2012. Weight loss 2-3 kg vs. placebo in monotherapy trials.[6]

Research Notes: Reference compound for GLP-1 RA pharmacology. Twice-daily and once-weekly formulations available for comparative study.

+ Recovery & Repair

These peptides are studied for their effects on tissue regeneration, wound healing, anti-inflammatory activity, and musculoskeletal repair. Research spans soft tissue injury models, bone healing, gastrointestinal mucosal protection, and corneal repair.

BPC-157 PLD-116 / PL 14736

RECOVERY

Mechanism: Synthetic pentadecapeptide derived from human gastric juice BPC. Modulates nitric oxide pathway, enhances angiogenesis, promotes tendon-to-bone healing, accelerates collagen reorganization.

Key Evidence: Transected quadriceps model: effective muscle healing in rats. [7] Achilles tendon detachment: promoted tendon-to-bone healing and opposed corticosteroid aggravation. [8] Review: BPC 157 enhances growth hormone receptor expression in tendon fibroblasts. [9]

Research Notes: Systemic (subcutaneous) and local (topical) administration both effective in preclinical models. Oral bioavailability demonstrated in animal studies.

TB-500 THYMOSIN BETA-4

RECOVERY

Mechanism: Synthetic fragment of thymosin beta-4 (residues 1-43). Promotes actin polymerization, cell migration, angiogenesis, and wound healing via integrin-linked kinase pathway.

Key Evidence: TB-4 accelerates wound healing in dermal and corneal models. Cardioprotective effects demonstrated in ischemia-reperfusion injury models. Promotes hair follicle stem cell migration and differentiation. [10]

Research Notes: Key research area: actin cytoskeleton modulation in cell migration. Widely studied in cardiac, dermal, and ophthalmic repair contexts.

COMPARATIVE ATTRIBUTE	BPC-157	TB-500 (THYMOSIN BETA-4)
Origin	Gastric juice BPC	Thymus gland
Primary Pathway	NO system, VEGF	Actin polymerization
Key Tissue Models	Muscle, tendon, GI	Cardiac, corneal, dermal
Route Evidence	Oral, SC, topical	SC, topical
Clinical Stage	Preclinical/I BD trials	Phase II (wound healing)

+ Performance Research

Growth hormone secretagogues (GHS) stimulate endogenous GH release via the ghrelin receptor (GHS-R1a). These compounds are investigated for effects on body composition, bone density, metabolic rate, and recovery kinetics.

Ipamorelin GH SECRETAGOGUE

PERFORMANCE

Mechanism: Pentapeptide (Aib-His-D-2-Nal-D-Phe-Lys-NH₂). Selective ghrelin receptor agonist that stimulates GH release without significantly elevating ACTH, cortisol, or prolactin — a selectivity profile unique among GHRPs.

Key Evidence: First selective GH secretagogue described (Raun et al., 1998). In swine: GH release ED₅₀ 2.3 nmol/kg, no ACTH or cortisol elevation at >200x ED₅₀ dose.[11] Preserved natural pulsatile GH secretion pattern.

Research Notes: Ideal for studying isolated GH effects without confounding from other pituitary hormones. Short half-life produces discrete GH pulses.

CJC-1295 GHRH ANALOGUE

PERFORMANCE

Mechanism: Modified GHRH (1-29) analogue with DAC (drug affinity complex) for albumin binding. Extends half-life from minutes to days, providing sustained GH and IGF-1 elevation.

Key Evidence: DAC conjugation increases in vivo half-life to 6-8 days. Stimulates GH pulse amplitude and frequency. IGF-1 elevation sustained over multiple days vs. native GHRH minutes.[12]

Research Notes: Without DAC (CJC-1295 no DAC / Mod GRF 1-29), half-life is 30 min. Compare pulsatile vs. sustained GH stimulation paradigms.

GHRP-6 GH SECRETAGOGUE

PERFORMANCE

Mechanism: Hexapeptide GHRP that stimulates GH release via hypothalamic/pituitary GHS-R1a. Also increases appetite through orexigenic pathways and elevates cortisol and prolactin.

Key Evidence: Potent GH release in vivo; ED₅₀ approximately 3.9 nmol/kg in swine. Increases food intake acutely. Less selective than ipamorelin — releases ACTH and cortisol.[11]

Research Notes: Useful as a comparative non-selective GHS control. Appetite stimulation complicates metabolic studies requiring caloric control.

Tesamorelin GHRH ANALOGUE

PERFORMANCE

Mechanism: Synthetic analogue of human GHRH (1-44) with trans-3-hexenoic acid modification for enhanced stability. FDA-approved for HIV-associated lipodystrophy.

Key Evidence: EGF037 trial: significant reduction in visceral adipose tissue (approx. 15%) over 26 weeks in HIV lipodystrophy patients. GH and IGF-1 levels increased within 2 weeks.[13]

Research Notes: FDA-approved status provides reference safety and efficacy data. Research focus: visceral vs. subcutaneous adipose selectivity mechanisms.

+ Cognitive Research

Cognitive peptides are studied for effects on memory formation, synaptic plasticity, neuroprotection, and modulation of neurotransmitter systems. Research spans cholinergic, dopaminergic, and BDNF-related pathways.

Semax ACTH-DERIVED

COGNITIVE

Mechanism: Synthetic heptapeptide (Met-Glu-His-Phe-Pro-Gly-Pro) derived from ACTH 4-10. BDNF stimulator; modulates brain-derived neurotrophic factor expression and serotonergic neurotransmission.

Key Evidence: Increases BDNF expression in rat hippocampus. Improves memory consolidation in animal models of cognitive impairment. Neuroprotective in hypoxia and ischemia models. Approved in Russia for cognitive and cerebrovascular disorders.[14]

Research Notes: Intranasal administration route provides direct CNS access. Compare BDNF-mediated vs. direct receptor-mediated mechanisms.

Selank ACTH-DERIVED

COGNITIVE

Mechanism: Synthetic heptapeptide (Thr-Lys-Pro-Arg-Pro-Gly-Pro) with anxiolytic and nootropic properties. Modulates GABAergic and enkephalin systems without sedation.

Key Evidence: Increases expression of GABA(A) receptor subunits. Anxiolytic effects demonstrated in rodent anxiety models at low doses. Synergistic with benzodiazepines in preclinical studies.[15]

Research Notes: Non-sedating anxiolytic profile is unique. Research applications in anxiety-cognition interaction studies.

Dihexa ANGIOGENIN

COGNITIVE

Mechanism: Oral small molecule peptide derivative of angiogenin. Potent hepatocyte growth factor (HGF) binding peptide that promotes synaptogenesis and dendritic arborization.

Key Evidence: Demonstrated 7x greater potency than BDNF in promoting synapse formation in vitro. Reverses cognitive impairment in scopolamine-induced amnesia models. Orally bioavailable — crosses BBB.[16]

Research Notes: Oral bioavailability is rare among cognitive peptides. HGF/c-Met pathway activation is the proposed mechanism of synaptogenic action.

GHK-Cu COPPER PEPTIDE

COGNITIVE

Mechanism: Tripeptide glycyl-L-histidyl-L-lysine copper complex. Modulates gene expression of approximately 4,000 human genes, resetting expression from aged to youthful profiles. Anti-inflammatory and antioxidant properties.

Key Evidence: C57BL/6 mice (28 months): improved spatial navigation learning after 3 weeks of treatment. Decreased brain inflammation; increased HDAC2 expression suggesting epigenetic modulation.[17]

Research Notes: Also categorized under cosmetic research. Gene expression profiling approach reveals broad regulatory effects.

+ Longevity Research

Longevity peptides target mechanisms of cellular aging: telomerase activity, cellular senescence, oxidative stress, epigenetic dysregulation, and mitochondrial function. These compounds represent some of the most actively investigated research peptides in geroscience.

Epitalon AEDG / EPITHALAMIN

LONGEVITY

Mechanism: Synthetic tetrapeptide (Ala-Glu-Asp-Gly) corresponding to epithalamin from pineal gland. Activates telomerase, elongates telomeres, regulates melatonin production, and modulates gene expression.

Key Evidence: Human fetal fibroblasts: extended proliferative capacity beyond 44th passage (control ceased at 34th).[18] CBA mice: 4-fold increase in mice reaching 23 months; maximum lifespan extended to 34 months.[19] SHR mice: 13.3% extension in lifespan of last 10% survivors.[20]

Research Notes: Multiple mechanisms likely contribute: telomerase activation, antioxidant effects, and pineal gland regulation. Human clinical data limited but promising.

Thymalin THYMUS PEPTIDE

LONGEVITY

Mechanism: Synthetic thymus-derived peptide that modulates T-cell differentiation and immune function. Restores thymic-dependent immunity in aging models.

Key Evidence: Combined with Epitalon in human studies: improved immune parameters and reduced acute respiratory infections in elderly subjects. Synergistic effects with epithalamin peptides on biomarkers of aging.[21]

Research Notes: Key research area: immunosenescence and thymic involution. Often studied in combination with pineal peptides.

FOXO4-DRI SENOLYTIC

LONGEVITY

Mechanism: Cell-penetrating peptide that disrupts FOXO4-p53 interaction, selectively inducing apoptosis in senescent cells (senolysis) while sparing healthy cells.

Key Evidence: Restored fitness, hair density, and kidney function in aged mice. Selectively cleared senescent cells without apparent toxicity. First senolytic peptide demonstrated effective in vivo.[22]

Research Notes: Senolytic mechanism distinct from senomorphics. Dose-response relationship for senescent cell clearance vs. safety is key research parameter.

SS-31 (Elamipretide) MITOCHONDRIAL

LONGEVITY

Mechanism: Cell-permeable peptide that targets cardiolipin in the inner mitochondrial membrane. Stabilizes mitochondrial cristae structure, reduces ROS production, and improves ATP synthesis efficiency.

Key Evidence: Phase II trials in mitochondrial diseases showed improved walking distance and patient-reported outcomes. ReCLAIM trial: significant improvement in mitochondrial function biomarkers in aged skeletal muscle.[23]

Research Notes: One of the few mitochondrial-targeted peptides in clinical trials. Cardiolipin binding mechanism is well-characterized at the molecular level.

+ Cosmetic Research

Cosmetic peptides are investigated for dermatological applications including skin remodeling, collagen synthesis, barrier function, and anti-aging mechanisms. Several compounds in this category have advanced to human clinical trials for topical delivery.

GHK-Cu COPPER TRIPEPTIDE

COSMETIC

Mechanism: Tissue remodeling, wound healing, collagen/elastin synthesis stimulation, angiogenesis promotion, antioxidant and anti-inflammatory activity via copper ion chelation.

Key Evidence: Clinical studies: faster wound healing and improved skin conditions with GHK-Cu cream. At 1 nM concentration: increases bFGF and VEGF in irradiated human dermal fibroblasts. At low concentrations: potent chemoattractant for capillary cells.[\[24\]](#)

Research Notes: Clinically approved for topical application as skin rejuvenator. Carrier systems (liposomes, hydrogels) may enhance dermal penetration.

Palmitoyl Pentapeptide-4 MATRIXYL

COSMETIC

Mechanism: Lipopeptide signalling fragment of procollagen I. Stimulates extracellular matrix renewal via collagen I, III, IV synthesis and fibronectin production.

Key Evidence: Double-blind placebo-controlled trial: significant reduction in wrinkle depth (mean 18%) and roughness after 4 months of topical application. Comparable results to retinol with better tolerability.[\[25\]](#)

Research Notes: One of the most studied cosmetic peptides. Palmitoyl chain enhances skin penetration. Useful as a benchmark in topical peptide formulation studies.

Acetyl Hexapeptide-8 ARGIRELINE

COSMETIC

Mechanism: Synthetic peptide fragment of SNAP-25 that competitively inhibits SNARE complex formation, reducing acetylcholine release at the neuromuscular junction and moderating facial muscle contraction.

Key Evidence: 10% topical solution: 30% wrinkle depth reduction after 30 days in periorbital region. Botulinum toxin-like mechanism without injection requirement.[\[26\]](#)

Research Notes: Non-invasive alternative mechanism for wrinkle reduction. Compare efficacy and kinetics vs. botulinum neurotoxins in preclinical models.

Copper Peptide AHK-Cu HAIR RESEARCH

COSMETIC

Mechanism: Alanine-histidine-lysine copper complex. Promotes vascular endothelial growth factor (VEGF) expression in dermal papilla cells, stimulating hair follicle growth and extending anagen phase.

Key Evidence: Increases VEGF expression in follicle dermal papilla cells. Promotes elongation of human hair follicles ex vivo. Copper-dependence distinguishes mechanism from GHK-Cu.[\[27\]](#)

Research Notes: Mechanism distinct from GHK-Cu despite structural similarity. VEGF-mediated angiogenesis is proposed primary driver.

+ Researcher's Decision Framework

Selecting the right peptide for your research question requires matching the compound's mechanism to your experimental goals, considering assay compatibility, and accounting for practical factors like stability, solubility, and regulatory constraints.

Matching Peptides to Research Questions

RESEARCH OBJECTIVE	PRIMARY PEPTIDE CLASS	KEY MECHANISM TO MEASURE
Glucose homeostasis, weight management	GLP-1 RA, dual GIP/GLP-1	Receptor binding, cAMP signaling, gastric emptying
Muscle/tendon healing, tissue repair	BPC-157, TB-500	Collagen synthesis, angiogenesis, cell migration
Growth hormone axis, body composition	Ipamorelin, CJC-1295	GH pulse kinetics, IGF-1 response
Memory, synaptic plasticity	Semax, Dihexa	BDNF expression, dendritic spine density
Cellular aging, senescence	Epitalon, FOXO4-DR1	Telomerase activity, SA-beta-gal, p16 expression
Skin remodeling, wound healing	GHK-Cu, Matrixyl peptides	Collagen I/III, fibroblast proliferation, angiogenesis

Experimental Considerations Checklist

- **Mechanism clarity:** Does the peptide's known mechanism directly address your hypothesis?
- **Assay compatibility:** Will the peptide interfere with your detection system? Consider vehicle controls carefully.
- **Concentration range:** Have you established a dose-response relationship based on published EC50/ED50 values?
- **Time course:** Does the peptide's kinetic profile match your experimental timeline?
- **Validation:** Are you including appropriate positive and negative controls?
- **Replication:** Is your experimental design adequately powered to detect the expected effect size?

+ Clinical References

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