RESEARCH COMPOUNDS GUIDE

PEPTIDES ARE THE FUTURE OF SCIENCE

BREAKTHROUGH SCIENCE IN PEPTIDE RESEARCH

METABOLISM RESEARCH

METABOLISM RESEARCH INTRODUCTION

WHAT IS METABOLISM?

Metabolism refers to the full set of chemical reactions that convert nutrients into usable energy within cells. Every cell in the body depends on these reactions to function.

Efficient metabolic function supports cellular repair, energy production, and the maintenance of biological systems. When metabolic processes become less efficient, published research has linked the decline to increased adipose tissue accumulation, reduced energy output, and elevated risk markers for metabolic conditions.

THE RESEARCH QUESTION: AGE-RELATED METABOLIC DECLINE

Metabolic rate declines measurably with age. Published data indicate that basal energy expenditure decreases progressively beginning around age 20, with significant cumulative effects by age 60–70.

This decline correlates with reduced cellular repair capacity and diminished organ function. A central question in metabolism research is whether these changes are modifiable—and if so, through what mechanisms.

MITOCHONDRIA: CELLULAR ENERGY PRODUCTION

Mitochondria are organelles found in nearly all eukaryotic cells. Their primary function is oxidative phosphorylation—the conversion of nutrients into adenosine triphosphate (ATP), the cell's energy currency.

A single cell may contain hundreds to thousands of mitochondria. High-energy-demand tissues—cardiac muscle, skeletal muscle, hepatic tissue—contain the highest mitochondrial density.

Age-related mitochondrial dysfunction is well-documented in the literature. As organisms age, mitochondrial efficiency decreases, ATP output declines, and reactive oxygen species (ROS) production increases. Published research has identified mitochondrial function as a key variable in age-related cellular decline.

Several of the compounds in this section have been studied for their effects on mitochondrial signaling pathways and cellular energy regulation.

COMPOUNDS IN METABOLISM RESEARCH

MOTS-C

TYPE: A MITOCHONDRIAL-DERIVED PEPTIDE

LEARN MORE →

WHAT IT IS

MOTS-c is a 16-amino acid peptide first identified in 2015 at the University of Southern California (Lee et al., Cell Metabolism). It is encoded within the mitochondrial genome—making it one of the first peptides discovered to originate from mitochondrial DNA rather than nuclear DNA.

This discovery expanded the scientific understanding of mitochondria from energy-producing organelles to active signaling participants in cellular regulation.

HOW IT WORKS

MOTS-c activates the AMPK signaling pathway, a central regulator of cellular energy homeostasis. Under conditions of metabolic stress, MOTS-c translocates to the nucleus, where it influences the expression of genes involved in fatty acid oxidation and glucose metabolism.

Researchers classify MOTS-c as part of a newly identified category of mitochondrial-derived peptides (MDPs) that function as retrograde signaling molecules between mitochondria and the nuclear genome.

WHAT RESEARCH SHOWS

In murine models, MOTS-c administration was associated with reduced weight gain under high-fat diet conditions and improved markers of glucose regulation (Lee et al., 2015).

A notable finding: aged mice administered MOTS-c demonstrated approximately double the running endurance of age-matched controls in treadmill testing. This led researchers to describe MOTS-c as a potential “exercise mimetic” in published literature.

Circulating MOTS-c levels have been measured as declining with age in both animal models and human plasma samples. Whether this decline is causally related to age-associated metabolic changes remains an active area of investigation.

WHY IT MATTERS

MOTS-c expanded the field’s understanding of mitochondrial communication. The identification of mitochondrial-derived peptides as active signaling molecules has opened new research directions in cellular energy regulation and age-related metabolic function.

NEXT UP: TESAMORELIN

TESAMORELIN

TYPE: A SYNTHETIC ANALOG OF GROWTH HORMONE-RELEASING HORMONE (GHRH)

LEARN MORE →

WHAT IT IS

Tesamorelin is a synthetic peptide analog of growth hormone-releasing hormone (GHRH), the endogenous signaling molecule that stimulates growth hormone (GH) secretion from the anterior pituitary.

Endogenous GHRH production decreases with age, which correlates with reduced GH output—a well-documented phenomenon in endocrinology known as somatopause. Declining GH levels are associated in the literature with changes in body composition, including increased visceral adiposity and decreased lean mass.

HOW IT WORKS

Tesamorelin stimulates pulsatile GH release from the pituitary gland, preserving the physiological secretory pattern. This mechanism is distinct from exogenous GH administration, which bypasses the hypothalamic-pituitary axis and may suppress endogenous production via negative feedback.

WHAT RESEARCH SHOWS

Tesamorelin has been studied in controlled clinical trials, most extensively in the context of HIV-associated lipodystrophy. In this population, published trial data (Falutz et al., NEJM, 2007) reported reductions in visceral adipose tissue. The compound received FDA approval for this specific indication under the trade name Egrifta.

It is important to note that the FDA-approved pharmaceutical formulation (Egrifta) is a distinct regulated product. Research-grade tesamorelin is not an FDA-approved medicine.

Additional published studies have examined tesamorelin's effects on GH-axis biomarkers including IGF-1 levels. Research continues into the broader significance of GHRH-axis modulation in age-related body composition changes.

WHY IT MATTERS

Tesamorelin is notable as one of the most extensively studied compounds in GHRH-axis research. Its clinical trial history provides a larger evidence base than most peptides in this category, making it a valuable reference point for understanding growth hormone signaling and its relationship to metabolic parameters.

NEXT UP: GLUTATHIONE

GLUTATHIONE

TYPE: AN ENDOGENOUS TRIPEPTIDE ANTIOXIDANT

LEARN MORE →

WHAT IT IS

Glutathione is a tripeptide composed of glutamate, cysteine, and glycine. It is synthesized endogenously in virtually all mammalian cells and serves as the primary intracellular thiol antioxidant.

Reactive oxygen species (ROS) are generated as a byproduct of normal oxidative metabolism and in response to environmental exposures. Glutathione's primary biochemical function is the neutralization of ROS through enzymatic and non-enzymatic pathways.

HOW IT WORKS

Glutathione functions through its reduced thiol group, which donates electrons to neutralize reactive oxygen species. It also serves as a cofactor for glutathione peroxidase and glutathione S-transferase enzyme families, and participates in the regeneration of other antioxidants including ascorbic acid (vitamin C) and α-tocopherol (vitamin E).

Glutathione also plays a documented role in Phase II hepatic conjugation reactions—the process by which the liver renders lipophilic compounds water-soluble for excretion.

WHAT RESEARCH SHOWS

Published research documents age-related decline in intracellular glutathione levels across multiple tissue types. This decline has been correlated in observational studies with increased oxidative stress markers and reduced antioxidant capacity.

The relationship between glutathione status and age-related conditions is an active area of investigation. Researchers are studying whether measured declines in glutathione are a contributing factor to age-associated oxidative damage or primarily a downstream marker.

Within mitochondrial biology, glutathione is recognized as essential for maintaining organelle function. Mitochondrial oxidative phosphorylation generates substantial ROS, and glutathione is the primary intramitochondrial defense against oxidative damage.

WHY IT MATTERS

Glutathione research highlights the role of antioxidant defense systems in cellular function. Its ubiquity across tissues, its documented age-related decline, and its central position in both mitochondrial protection and hepatic function make it a reference compound for understanding oxidative stress biology.

WHAT WE'VE LEARNED

COMMON THEMES

Looking at these compounds, several patterns emerge:

1. **Mitochondrial function is a central research target.** All three compounds—MOTS-c, Tesamorelin (via the GH-IGF-1 axis), and Glutathione—converge on mitochondrial biology. Whether through signaling (MOTS-c), hormonal support for energy metabolism (Tesamorelin), or direct organelle protection (Glutathione), mitochondria emerge as a consistent focal point in published metabolism research.
2. **Measurable age-related declines.** MOTS-c, GH-axis hormones, and glutathione all show documented decreases with age in published studies. Whether these declines are causally linked to metabolic changes—or are correlative markers of broader aging processes—remains a key question in the field.
3. **Endogenous signaling pathways as research targets.** Rather than introducing novel exogenous agents, these compounds engage pathways that already exist within the organism. This research approach—studying modulation of endogenous systems—represents an active direction in the field.

LOOKING AHEAD

Metabolism research intersects with other major areas of investigation. In Part 2, we examine compounds studied in the context of tissue maintenance and cellular regeneration—research areas where metabolic function plays a supporting role.

Published literature increasingly treats metabolism, tissue homeostasis, and cellular aging as interconnected systems rather than isolated fields. The compounds in this section have been studied for their relevance across multiple biological pathways.

REFERENCES

These sources provide more detail on the topics covered. Scientific papers can be found on PubMed (pubmed.gov).

Mitochondrial Peptides

Lee C, et al. The mitochondrial-derived peptide MOTS-c promotes metabolic homeostasis. Cell Metabolism. 2015;21(3):443-454.

Tesamorelin Research

Falutz J, et al. Effects of tesamorelin (TH9507), a growth hormone-releasing factor analog, in human immunodeficiency virus-infected patients with excess abdominal fat. The New England Journal of Medicine. 2007;357(23):2349-2360.

Glutathione and Aging

Richie JP Jr, et al. Randomized controlled trial of oral glutathione supplementation on body stores of glutathione. European Journal of Nutrition. 2015;54(2):251-263.

LONGEVITY & HEALTHSPAN RESEARCH

LONGEVITY & HEALTHSPAN RESEARCH INTRODUCTION

WHAT IS LONGEVITY SCIENCE?

Longevity research investigates the biological mechanisms underlying aging and seeks to identify factors that influence healthspan—the period of life spent in functional health, as distinguished from total lifespan.

The field has moved beyond asking whether aging can be slowed in model organisms (published studies have demonstrated lifespan extension in C. elegans, Drosophila, and murine models). The current research focus is on understanding which mechanisms are conserved across species and which may be relevant to mammalian biology.

HALLMARKS OF AGING

In 2013, López-Otín et al. published a landmark framework identifying nine hallmarks of aging (Cell, 2013), expanded to twelve in a 2023 update. These hallmarks provide the organizing framework for most current aging research. Key hallmarks relevant to this section include:

> **Genomic instability.** Accumulated DNA damage from endogenous and exogenous sources. Repair efficiency declines with age, allowing mutations to accumulate.

> **Telomere attrition.** Progressive shortening of telomeric sequences with each cell division, eventually triggering cellular senescence or apoptosis.

> **Mitochondrial dysfunction.** Declining efficiency of oxidative phosphorylation, increased ROS production, and reduced ATP output—discussed in detail in Part 1.

> **Impaired tissue homeostasis.** Reduced regenerative capacity and slower tissue turnover, associated with declining stem cell function and altered growth factor signaling.

> **Altered intercellular communication.** Changes in immune signaling, including chronic low-grade inflammation ("inflammaging"), which impairs normal tissue maintenance.

The compounds in this section have been studied for their relevance to tissue homeostasis, regenerative signaling, and cellular repair mechanisms—hallmarks where peptide research has generated the most published data.

COMPOUNDS IN LONGEVITY & HEALTHSPAN RESEARCH

GHK-CU (COPPER PEPTIDE)

TYPE: AN ENDOGENOUS COPPER-BINDING TRIPEPTIDE

LEARN MORE →

WHAT IT IS

GHK-Cu (glycyl-L-histidyl-L-lysine:copper(II)) is a naturally occurring tripeptide-metal complex first isolated from human plasma in the 1970s. The copper ion is essential to its biological activity—the peptide without copper does not demonstrate the same effects in published studies.

HOW IT WORKS

Gene expression studies have identified approximately 4,000 genes modulated by GHK-Cu—roughly 6% of the human genome. Published analyses of these gene expression changes show upregulation of genes associated with extracellular matrix production (including collagen synthesis) and downregulation of genes associated with inflammatory signaling.

GHK-Cu has also been studied for effects on angiogenic signaling pathways and DNA repair gene expression.

WHAT RESEARCH SHOWS

In published wound-healing models, GHK-Cu administration was associated with increased collagen deposition rates and accelerated tissue remodeling. These findings have led to its incorporation in commercial dermatological formulations.

Plasma concentration studies document significant age-related decline: from approximately 200 ng/mL in young adults to approximately 80 ng/mL by age 60 (Pickart et al., 2015).

Gene expression profiling studies (using computational analysis of the Connectivity Map database) suggest that GHK-Cu shifts gene expression patterns in aged cells toward profiles more closely resembling younger tissue. This area of research remains active and is the subject of ongoing investigation.

WHY IT MATTERS

GHK-Cu is notable for the breadth of gene expression changes associated with a single small peptide. The documented age-related decline in plasma levels, combined with the scope of its genomic effects, has made it a focus of research into age-related changes in tissue maintenance signaling.

NEXT UP: TB-500 (THYMOSIN BETA-4)

TB-500 (THYMOSIN BETA-4)

TYPE: A SYNTHETIC FRAGMENT OF THYMOSIN BETA-4, STUDIED FOR TISSUE REPAIR SIGNALING

LEARN MORE →

WHAT IT IS

TB-500 is a synthetic peptide derived from thymosin beta-4, a 43-amino acid peptide found endogenously in high concentrations in wound fluid, platelets, and other tissues. Thymosin beta-4 was first characterized for its role in actin polymerization and cell motility.

HOW IT WORKS

Published mechanistic studies describe TB-500's effects on cell migration (facilitating cellular movement toward sites of tissue damage), angiogenic signaling (promoting vascular network formation), and inflammatory pathway modulation.

The peptide has demonstrated activity across multiple tissue types in preclinical models, including muscle, tendon, ligament, dermal, and neural tissue.

WHAT RESEARCH SHOWS

In animal injury models, TB-500 administration has been associated with increased rates of tissue remodeling in musculoskeletal structures. Published preclinical studies report effects on extracellular matrix reorganization and vascular formation in damaged tissue.

Research has also examined TB-500 in the context of age-related changes in tissue regenerative capacity. Published studies in aged animal models suggest altered repair signaling as a component of tissue decline.

WHY IT MATTERS

TB-500 is studied for its activity across multiple tissue types in preclinical models—an unusual breadth for a single peptide. Its relevance to tissue homeostasis research, particularly in the context of age-related changes in regenerative signaling, has generated sustained interest in the published literature.

NEXT UP: BPC-157

BPC-157

TYPE: A SYNTHETIC PENTADECAPEPTIDE STUDIED IN PRECLINICAL TISSUE REPAIR MODELS

LEARN MORE →

WHAT IT IS

BPC-157 is a synthetic 15-amino acid peptide derived from a sequence found in human gastric juice proteins. It was first characterized by Sikiric et al. at the University of Zagreb.

Unlike most peptides, BPC-157 demonstrates stability under acidic conditions—a property documented in published studies that has generated particular research interest.

HOW IT WORKS

Published mechanistic research describes BPC-157's effects on angiogenic signaling, growth factor activation, inflammatory pathway modulation, and cytoprotective mechanisms. The compound has shown activity across multiple tissue types in preclinical models, which is unusual for a single peptide.

Published studies also report that BPC-157 crosses the blood-brain barrier in animal models, leading to investigation of its effects on central nervous system pathways.

WHAT RESEARCH SHOWS

Over 100 published preclinical studies have examined BPC-157 across multiple tissue types—musculoskeletal, gastrointestinal, dermal, and neural. The majority of this evidence base comes from animal models, primarily from the University of Zagreb research group.

In neurological research contexts, published studies report effects on neurotransmitter pathway signaling (including dopaminergic and serotonergic systems) and on markers of neuronal viability in injury models.

It is important to note that while the preclinical literature is extensive, controlled human clinical trial data for BPC-157 remains limited at the time of publication.

WHY IT MATTERS

BPC-157's unusually broad tissue activity profile and its stability in acidic conditions have made it one of the most-published peptides in preclinical tissue research. Its cross-tissue effects—spanning musculoskeletal, gastrointestinal, and neurological systems—continue to generate research interest in the context of tissue maintenance biology.

SYNERGISTIC FORMULATIONS

Many researchers are exploring combinations of peptides to enhance regenerative effects. When peptides work together, they can create synergistic benefits greater than any single peptide alone. Three notable research formulations combine these compounds:

GLOW RESEARCH FORMULATION

Composition:
GHK-Cu + BPC-157 + TB-500

LEARN MORE →

RESEARCH FOCUS

This formulation combines three compounds that have been individually studied for effects on tissue maintenance signaling. GHK-Cu has been studied for gene expression modulation and extracellular matrix signaling. BPC-157 has been studied for its broad tissue activity profile. TB-500 has been studied for cell migration and vascular signaling.

The research rationale for combining these compounds is based on published observations that tissue homeostasis involves multiple simultaneous signaling pathways. Whether multi-compound approaches produce effects that differ from single-compound administration is an active area of preclinical investigation.

KLOW RESEARCH FORMULATION

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

LEARN MORE →

RESEARCH FOCUS

KLOW adds KPV—a tripeptide derived from alpha-melanocyte stimulating hormone (α-MSH)—to the GLOW compound set. KPV has been studied in published research for its effects on inflammatory signaling pathways, particularly NF-κB pathway modulation.

The inclusion of KPV introduces an immunomodulatory research dimension to the tissue-focused compounds in GLOW. Published research on the intersection of inflammatory signaling and tissue maintenance has generated interest in multi-pathway approaches.

TISSUE REPAIR RESEARCH FORMULATION

Composition:
BPC-157 + TB-500

LEARN MORE →

RESEARCH FOCUS

This two-compound formulation pairs the two peptides in this section with the most published data on tissue-related signaling. BPC-157 and TB-500 have been studied individually across overlapping tissue types, and research interest exists in whether co-administration produces additive or synergistic effects in preclinical models.

Published literature on multi-peptide research approaches remains early-stage. The mechanistic rationale for combination studies is based on the observation that biological tissue repair involves concurrent signaling across multiple pathways.

WHAT WE'VE LEARNED

COMMON THEMES

1. **Tissue homeostasis as a central research theme.** All compounds in this section have been studied for effects related to tissue maintenance, repair signaling, or regenerative capacity. This reflects the published consensus that maintaining tissue function is a key variable in biological aging.
2. **Documented age-related declines.** GHK-Cu plasma levels decline measurably with age. Age-related changes in regenerative factor levels have been documented across multiple compound classes. Whether supplementing declining endogenous signals modifies biological outcomes is a central question in aging research.
3. **Multi-pathway research approaches.** The formulations in this section reflect an emerging research direction: studying multiple signaling pathways simultaneously rather than isolating single targets. Published literature on this approach is early-stage but growing.
4. **Interconnected biological systems.** Published research increasingly describes tissue damage, inflammatory signaling, and regenerative capacity as interconnected systems rather than independent variables. This interconnection is a recurring theme across aging research disciplines.

LOOKING AHEAD

Part 3 examines compounds studied in the context of neurological function and age-related changes in the central nervous system. Published literature connects the hallmarks of aging discussed in this section—tissue homeostasis, inflammatory signaling, mitochondrial function—to CNS biology as well.

REFERENCES

These sources provide more detail on the topics covered. Scientific papers can be found on PubMed (pubmed.gov).

Hallmarks of Aging

López-Otín C, et al. The hallmarks of aging. Cell. 2013;153(6):1194-1217.

López-Otín C, et al. Hallmarks of aging: An expanding universe. Cell. 2023;186(2):243-278.

GHK-Cu

Pickart L, et al. GHK peptide as a natural modulator of multiple cellular pathways in skin regeneration. Biomed Res Int. 2015;2015:648108.

TB-500 (Thymosin Beta-4)

Crockford D, et al. Thymosin beta4: structure, function and biological properties supporting current and future clinical applications. Ann N Y Acad Sci. 2010;1194:179-189.

BPC-157

Sikiric P, et al. Brain-gut axis and pentadecapeptide BPC 157. Curr Neuropharmacol. 2016;14(8):857-865.

Galperin D, et al. Pentadecapeptide BPC 157 and its effects on the central and peripheral nervous system. Front Neurol. 2018;9:991.

BRAIN HEALTH & AGE-RELATED DECLINE RESEARCH

BRAIN HEALTH & AGE-RELATED DECLINE RESEARCH INTRODUCTION

THE CNS RESEARCH CHALLENGE

The human brain contains approximately 86 billion neurons, each forming thousands of synaptic connections. This complexity—estimated at over 100 trillion synapses—makes the central nervous system among the most challenging areas in biological research.

Age-related neurological conditions represent a major focus of current biomedical research. Published literature documents well-characterized pathological processes, yet effective interventions remain limited. Many compounds that demonstrated efficacy in preclinical models have failed to translate to clinical success.

This translational gap has driven research interest in alternative approaches, including peptide-based compounds that may engage multiple signaling pathways simultaneously.

AGE-RELATED CHANGES IN CNS BIOLOGY

Published research has identified several well-characterized age-related changes in CNS biology:

> **Protein aggregation.** Misfolded protein accumulation is a hallmark of multiple age-related neurological conditions. Different conditions involve different protein species, but the aggregation mechanism shares common features across pathologies.

> **Neuroinflammation.** Microglial activation and chronic neuroinflammatory signaling have been documented as features of the aging CNS. Published research describes a shift from neuroprotective to neurotoxic microglial phenotypes with age.

> **Synaptic loss.** Age-related decline in synaptic density and plasticity is well-documented. The balance between synaptogenesis and synaptic pruning shifts with age, which correlates with published measures of cognitive function.

> **Mitochondrial dysfunction.** Neurons are among the highest energy-demand cells in the body. Age-related mitochondrial decline disproportionately affects CNS function, as discussed in Part 1.

NEUROTROPHIC FACTOR RESEARCH

Neurotrophic factors—including brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and glial cell line-derived neurotrophic factor (GDNF)—are endogenous proteins that support neuronal survival, synaptic plasticity, and axonal growth.

Published research documents age-related declines in neurotrophic factor expression. However, direct administration of neurotrophic factors as therapeutic agents is limited by their molecular size and inability to cross the blood-brain barrier.

Several peptides in this section have been studied for their effects on neurotrophic factor expression through indirect mechanisms—an approach that avoids the blood-brain barrier limitation.

COMPOUNDS IN BRAIN HEALTH & AGE-RELATED DECLINE RESEARCH

BPC-157

TYPE: A SYNTHETIC PENTADECAPEPTIDE STUDIED IN PRECLINICAL TISSUE REPAIR AND NEUROLOGICAL MODELS

LEARN MORE →

WHAT IT IS

BPC-157 was introduced in Part 2 for its broad tissue activity profile. This section examines published research on its effects in neurological models specifically.

HOW IT WORKS — CNS CONTEXT

Published studies report that BPC-157 crosses the blood-brain barrier in animal models. Mechanistic research describes effects on multiple neurotransmitter systems, including dopaminergic and serotonergic pathways, as well as modulation of nitric oxide (NO) signaling.

WHAT RESEARCH SHOWS

In preclinical CNS injury models, published studies (primarily from the Sikiric research group) report effects on neuronal viability markers and functional recovery measures.

Research has also examined BPC-157's effects on neurotransmitter pathway signaling. Published studies describe modulation of dopamine and serotonin system activity in animal models, though the significance of these findings for broader neurological research remains under investigation.

Additional preclinical research has examined BPC-157 in the context of neuroinflammatory signaling pathways.

WHY IT MATTERS

BPC-157's documented blood-brain barrier permeability, combined with its cross-tissue activity profile, has positioned it as a compound of interest in CNS research. The preclinical evidence base is substantial, though controlled human clinical data remains limited.

NEXT UP: SEMAX

SEMAX

TYPE: A SYNTHETIC HEPTAPEPTIDE DERIVED FROM THE ACTH(4–10) FRAGMENT

LEARN MORE →

WHAT IT IS

Semax is a synthetic heptapeptide developed at the Institute of Molecular Genetics, Russian Academy of Sciences. It is based on the ACTH(4–10) fragment—a portion of adrenocorticotropic hormone (ACTH) that was identified in published research as influencing neurotrophic factor expression.

The structural modification was designed to retain neurotrophic activity while eliminating the steroidogenic effects of full-length ACTH.

HOW IT WORKS

Published mechanistic studies report that Semax administration increases BDNF and NGF expression in animal models (Agapova et al., 2008). The compound also modulates dopaminergic and serotonergic neurotransmitter systems in preclinical research.

WHAT RESEARCH SHOWS

Semax has regulatory status in Russia for specific clinical indications. It has been the subject of published clinical studies conducted primarily in Russian research institutions, examining effects on cognitive function biomarkers and neurological recovery measures.

In preclinical models, published studies report neuroprotective effects in ischemia models and modulation of learning and memory performance in behavioral testing paradigms.

Published research has also reported anxiolytic effects in animal behavioral models without the sedative effects typically associated with GABAergic compounds—a finding that has generated interest in the pharmacological profile.

WHY IT MATTERS

Semax is notable for its derivation from an endogenous hormone fragment and its documented effects on neurotrophic factor expression. The published research base—primarily from Russian institutions—represents one of the more extensive peptide-CNS research programs in the literature.

NEXT UP: SELANK

SELANK

TYPE: A SYNTHETIC HEPTAPEPTIDE DERIVED FROM THE IMMUNOMODULATORY PEPTIDE TUFTSIN

LEARN MORE →

WHAT IT IS

Selank is a synthetic heptapeptide developed at the same Russian Academy of Sciences institute that produced Semax. It is based on tuftsin, a naturally occurring tetrapeptide with documented immunomodulatory properties, modified for increased stability.

HOW IT WORKS

Published mechanistic studies describe Selank's effects on GABAergic neurotransmission, specifically through modulation of GABA receptor gene expression (Filatova et al., Front Pharmacol, 2017). This mechanism is distinct from direct GABA receptor agonism.

Selank also retains immunomodulatory properties from its tuftsin-derived structure. Published research describes effects on BDNF expression and on interferon signaling pathways.

WHAT RESEARCH SHOWS

Selank has regulatory status in Russia for specific clinical indications. Published clinical studies conducted at Russian institutions have reported anxiolytic effects as measured by standard behavioral assessment instruments.

In preclinical models, published research reports effects on stress hormone regulation and behavioral measures of anxiety. Studies have also documented modulation of interferon expression—an immunological effect that connects to published research on neuroimmune interactions in age-related CNS changes.

WHY IT MATTERS

Selank is notable for its dual activity across GABAergic and immunomodulatory pathways. Published research on neuroimmune interactions—the bidirectional communication between the CNS and immune systems—has identified compounds with cross-system activity as a distinct area of investigation.

WHAT WE'VE LEARNED

COMMON THEMES

1. **Neurotrophic factor modulation as a research focus.** All three compounds in this section have been studied for effects on neurotrophic factor expression or signaling. The role of BDNF, NGF, and related factors in CNS maintenance is well-established in published literature, making this a high-interest research area.
2. **CNS-body interconnections in published research.** The themes from Parts 1 and 2—mitochondrial function, inflammatory signaling, and tissue maintenance—appear again in CNS biology. Published research increasingly treats these as interconnected systems rather than organ-specific phenomena.
3. **Age-related decline in signaling molecules.** Neurotrophic factors, neuroprotective peptides, and neuromodulatory compounds show documented age-related decreases in published studies. Whether modulating these pathways influences biological outcomes in aging is a central question in the field.
4. **Multi-pathway approaches in CNS research.** Published literature on age-related CNS changes describes complex, multi-system pathology. Compounds with activity across multiple signaling pathways have generated research interest precisely because single-target approaches have shown limited success in clinical translation.

LOOKING AHEAD

Parts 1 through 3 have examined published research across three interconnected areas: cellular energy metabolism, tissue homeostasis, and CNS biology. These are not isolated domains—the hallmarks of aging framework (López-Otín et al., 2013, 2023) describes them as concurrent processes that influence each other.

The compounds covered in this publication represent a subset of an active and expanding research area. The published evidence base is strongest in preclinical models, and the translational path from animal research to clinical application remains an open and ongoing investigation.

REFERENCES

These sources provide more detail on the topics covered. Scientific papers can be found on PubMed (pubmed.gov).

Brain Health and Neurodegeneration

Cummings J, et al. Alzheimer's disease drug development pipeline. Alzheimers Dement. 2022;8(1):e12295.

Selkoe DJ, Hardy J. The amyloid hypothesis of Alzheimer's disease at 25 years. EMBO Mol Med. 2016;8(6):595-608.

BPC-157

Sikiric P, et al. Brain-gut axis and pentadecapeptide BPC 157. Curr Neuropharmacol. 2016;14(8):857-865.

Galperin D, et al. Pentadecapeptide BPC 157 and its effects on the central and peripheral nervous system. Front Neurol. 2018;9:991.

Semax and Cognitive Enhancement

Agapova TI, et al. Effect of semax on BDNF and NGF gene expression. Mol Genet Mikrobiol Virusol. 2008;(3):28-32.

Gualtieri F, et al. Semax: A novel peptide with neuroprotective and cognition-enhancing properties. CNS Drug Rev. 2002;8(4):376-388.

Selank and Anxiety Modulation

Filatova E, et al. GABA, Selank, and olanzapine affect GABAergic neurotransmission genes. Front Pharmacol. 2017;8:89.

Kozlovskaya MM, et al. Selank, a novel immunomodulatory peptide, reduces anxiety through GABA signaling. Neuropharmacology. 2014;86:65-73.

CONCLUSION

PUTTING IT ALL TOGETHER

THREE RESEARCH AREAS, ONE FRAMEWORK

This publication has examined published research across three interconnected areas: cellular energy metabolism, tissue homeostasis and regenerative signaling, and central nervous system biology. Each section covered compounds that have generated substantial preclinical research data.

INTERCONNECTED SYSTEMS

A consistent theme across all three sections: the biological systems under investigation are not independent. Published research connects them through shared mechanisms:

> **Mitochondrial function** is relevant to energy metabolism, tissue maintenance, and CNS biology—all three sections converge on this organelle.

> **Inflammatory signaling** appears in each section as a variable that influences metabolic function, tissue repair, and neurological maintenance.

> **Growth factor and signaling peptide pathways** show documented age-related declines across multiple systems—a pattern that recurs throughout published aging research.

RESEARCH COMPOUNDS AS INVESTIGATIVE TOOLS

The compounds covered in this publication are research tools. They allow investigators to probe specific biological pathways, test mechanistic hypotheses, and generate data about the systems that maintain cellular function.

Published research continues to expand. The evidence base is strongest in preclinical models, and the translational pathway from laboratory findings to clinical application involves additional stages of investigation, validation, and regulatory review.

THE RESEARCH AHEAD

The questions investigated in this publication represent active areas in the published literature: What mechanisms drive age-related changes in metabolic efficiency? What factors govern tissue regenerative capacity? What signaling pathways maintain CNS function across the lifespan?

These questions define the current frontier of peptide research. The compounds discussed here are among the tools being used to explore them.

PERFECT PEPTIDES

https://perfectpeptides.com/