THE PIVOTAL PROTOCOL - EDUCATIONAL REFERENCE SERIES

Complete Compound Reference Guide

A comprehensive educational reference for The Pivotal Protocol curriculum. Covers mechanism, dosing context, cycle parameters, use cases, research status, monitoring, cautions, and stack compatibility for each major compound class.

Educational purposes only. This document is for research literacy and educational context only. Dosing ranges cited are drawn from published research literature and represent what has been studied - not clinical prescriptions. Nothing here constitutes medical advice, diagnosis, or a treatment recommendation. The Pivotal Protocol is an education and teaching operation. All compound use should occur under the guidance of a licensed physician.

Compounds Covered in This Guide

Recovery Peptides

BPC-157

TB-500 (Thymosin Beta-4 fragment)

GH Secretagogues — GHRPs

Ipamorelin

GHRP-2

GHRP-6

GH SECRETAGOGUES + GHRH ANALOGS

CJC-1295 (no DAC)

CJC-1295 (with DAC)

Sermorelin

Tesamorelin

FAT-SELECTIVE

AOD-9604

LONGEVITY / MITOCHONDRIAL	NEUROPEPTIDES
Epithalon	Semax
SS-31 (Elamipretide)	Selank
MOTS-c	Dihexa
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CLASS 01

Recovery Peptides

BPC-157

Body Protection Compound-157 | 15-amino acid stable gastric pentadecapeptide fragment

CLASS	Cytoprotective / Regenerative Peptide
PRIMARY MECHANISM	Upregulates growth hormone receptor expression; promotes angiogenesis via VEGFR2 pathway; modulates nitric oxide signaling; accelerates tendon-to-bone healing via COX-2 and FAK-paxillin pathways
TYPICAL DOSING RANGE (EDUCATIONAL)	200–500 mcg per administration
ADMINISTRATION ROUTE	SubQ injection near site of injury; systemic SubQ also studied; oral form (less bioavailable) in some research
TYPICAL CYCLE LENGTH	4–12 weeks (injury-specific may be shorter)
TYPICAL OFF-PERIOD	4–8 weeks

Primary Use Cases

- Tendon and ligament injury recovery
- Muscle tear healing support
- Gut lining repair (gastric ulcer models in research)
- Post-surgical recovery (animal models)
- Inflammatory joint conditions (research context)

Key Research Notes

Primarily Animal Data The overwhelming majority of BPC-157 research is rodent and small-animal studies. Results are striking across a range of injury models. Human trials are limited. The translation from animal to human data remains scientifically uncertain but is a subject of active educational interest in the longevity and performance community.

What to Monitor

- CRP and inflammatory markers (may decrease with use)
- Subjective pain and function scores for the target injury
- Liver enzymes (ALT/AST) at baseline and end of cycle

Notable Cautions: Some concerns exist in oncology-adjacent research that the pro-angiogenic and growth-promoting properties of BPC-157 could theoretically accelerate tumor growth in individuals with existing malignancy. This is an active area of scientific discussion. Individuals with personal or family history of cancer should discuss this context with their physician before any protocol consideration.

Stack Compatibility: Commonly paired with TB-500 (complementary mechanisms - BPC-157 drives local healing; TB-500 promotes systemic cell migration to injury site). Compatible with GH secretagogues in the same protocol. No known pharmacokinetic conflicts with other peptides in this guide.

TB-500

Thymosin Beta-4 fragment | Synthetic analog of the naturally occurring Thymosin Beta-4 protein

CLASS	Regenerative / Anti-inflammatory Peptide
PRIMARY MECHANISM	Promotes actin polymerization and cell migration; upregulates stem cell chemotaxis to injury sites; anti-inflammatory via NF-kB downregulation; promotes new blood vessel formation
TYPICAL DOSING RANGE (EDUCATIONAL)	2-2.5 mg per administration; loading phase vs. maintenance phase used in some protocols
ADMINISTRATION ROUTE	SubQ or IM injection; systemic rather than site-specific
TYPICAL CYCLE LENGTH	4-12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Systemic tissue repair and recovery
- Muscle injury and strain recovery
- Cardiac tissue research (human trials in progress for cardiac conditions)
- Neurological repair (early animal research)
- Chronic inflammation reduction

Key Research Notes

Primarily Animal Data | Some Human Trials

Full-length Thymosin Beta-4 has been studied in human clinical trials for dry eye disease and cardiac conditions. TB-500 (a fragment) has extrapolated research. The peptide is naturally produced in the body and concentrated at sites of injury. TB-500 is a banned substance in athletic competition under WADA rules.

What to Monitor

- Inflammatory markers (CRP, ESR)
- Subjective recovery metrics
- General CBC at baseline and end of cycle

Notable Cautions: Same pro-angiogenic theoretical concern as BPC-157 in oncology-adjacent contexts. WADA-banned for competitive athletes. No human long-term safety data exists.

Stack Compatibility: Pairs well with BPC-157 (see BPC-157 stack notes). Compatible with GH secretagogues. No known conflicts with neuropeptides or mitochondrial compounds.

CLASS 02

GH Secretagogues: GHRPs

Ipamorelin

Ipamorelin | Selective GH secretagogue / Ghrelin receptor agonist

CLASS	GHRP (Growth Hormone Releasing Peptide) / Ghrelin Mimetic
PRIMARY MECHANISM	Selective agonist at the ghrelin receptor (GHS-R1a) in the pituitary; stimulates GH pulse release without meaningful cortisol, prolactin, or ACTH co-stimulation
TYPICAL DOSING RANGE (EDUCATIONAL)	100–300 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	8–12 weeks
TYPICAL OFF-PERIOD	4–8 weeks

Primary Use Cases

- GH pulse amplification
- Body composition (supporting lean mass, reducing adiposity via GH axis)
- Sleep quality improvement (GH pulse at delta sleep window)
- Recovery and anti-aging protocols

Key Research Notes

Human Data Available

Ipamorelin is among the most studied GHRPs. Clinical trials have evaluated it for post-operative GI motility in addition to GH secretagogue research. Its selectivity – notably the absence of cortisol and prolactin co-stimulation seen in GHRP-2 and GHRP-6 – makes it the most commonly referenced GHRP in educational peptide literature.

What to Monitor

- IGF-1 (primary efficacy marker)
- Fasted glucose and HbA1c
- Sleep quality (subjective)

- Water retention (transient fluid retention is a common early effect)

Notable Cautions: Fasted administration required for meaningful GH pulse. Carbohydrate ingestion within 2-3 hours blunts response significantly. Mild water retention common in first 2-3 weeks; generally resolves.

Stack Compatibility: Synergistic with GHRH analogs (CJC-1295 no DAC, Sermorelin, Tesamorelin). The GHRP + GHRH combination produces a substantially larger GH pulse than either alone - a well-documented synergy in GH secretagogue research. Compatible with recovery peptides and neuropeptides in same protocol.

GHRP-2

Growth Hormone Releasing Peptide-2 | Synthetic hexapeptide ghrelin receptor agonist

CLASS	CHRP / Ghrelin Mimetic (non-selective)
PRIMARY MECHANISM	Potent ghrelin receptor agonist; stimulates strong GH pulse; also stimulates cortisol and prolactin release via secondary pathways
TYPICAL DOSING RANGE (EDUCATIONAL)	100-300 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	8-12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Potent GH pulse stimulation
- Muscle building protocols (higher GH amplitude than Ipamorelin)
- GH deficiency research models

Key Research Notes

Human Data Available

GHRP-2 produces a larger GH pulse than Ipamorelin but carries the disadvantage of co-stimulating cortisol and prolactin. This hormonal profile makes baseline prolactin measurement particularly important before use. Research has confirmed its potent GH-releasing effect in human volunteers.

What to Monitor

- IGF-1
- Prolactin (baseline essential; retest at week 6)
- Cortisol (morning cortisol trend)
- Fasted glucose, HbA1c

Notable Cautions: Prolactin co-stimulation can cause libido changes, mood effects, and in rare cases gynecomastia. Cortisol co-stimulation adds to chronic stress

burden. Individuals with anxiety disorders or adrenal dysfunction should evaluate this context carefully with their physician.

Stack Compatibility: Synergistic with GHRH analogs. Some educational frameworks prefer pairing GHRP-2 with a GHRH analog for shorter, higher-amplitude pulse protocols. Not commonly stacked with Ipamorelin (redundant mechanism class; GHRP-2 dominant effect would predominate).

GHRP-6

Growth Hormone Releasing Peptide-6 | Hexapeptide ghrelin receptor agonist

CLASS	GHRP / Ghrelin Mimetic (non-selective)
PRIMARY MECHANISM	Ghrelin receptor agonist; strong GH pulse stimulation; pronounced ghrelin-mediated appetite stimulation; cortisol and prolactin co-stimulation similar to GHRP-2
TYPICAL DOSING RANGE (EDUCATIONAL)	100–300 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	8–12 weeks
TYPICAL OFF-PERIOD	4–8 weeks

Primary Use Cases

- Potent GH stimulation
- Appetite stimulation (intentional, for individuals in a caloric deficit or with appetite suppression)
- Muscle building with aggressive eating strategy

Key Research Notes

Human Data Available

GHRP-6 was one of the first synthetic GHRPs studied and has a substantial human research literature. Its hunger-stimulating effect is its most predictable and prominent side effect — ghrelin is the primary hunger hormone, and GHRP-6 behaves as a strong ghrelin mimetic. This makes it less suitable for individuals using protocols in conjunction with caloric restriction.

What to Monitor

- IGF-1
- Prolactin, cortisol
- Caloric intake and body composition (hunger drive can undermine caloric targets)

- Fasted glucose, HbA1c

Notable Cautions: Strong hunger drive can be counterproductive on caloric restriction protocols. Same cortisol and prolactin concerns as GHRP-2. Fasted window requirement applies - the hunger signal shortly after injection is a known and expected effect.

Stack Compatibility: Same synergy with GHRH analogs as other GHRPs. Appetite stimulation makes this a niche choice - typically selected specifically for its hunger-driving effect, not as a default GHRP choice.

CLASS 03

GH Secretagogues: GHRH Analogs

CJC-1295 (no DAC)

Modified GRF 1-29 | Short-acting GHRH analog

CLASS	GHRH Analog (short-acting)
PRIMARY MECHANISM	Acts on GHRH receptors in the pituitary to amplify the amplitude of GH pulses; does not itself initiate a pulse but amplifies the somatotroph response when combined with a GHRP
TYPICAL DOSING RANGE (EDUCATIONAL)	100-300 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	8-12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- GH pulse amplification when co-administered with a GHRP (primary use case)
- Physiologic pulsatility preservation (preserves natural GH pulse pattern unlike continuous GH administration)

Key Research Notes

Human Data Available

CJC-1295 without DAC (also known as Modified GRF 1-29) has a half-life of approximately 30 minutes, making it a clean, pulse-preserving GHRH analog. It is almost always used in combination with a GHRP - the synergy between GHRH + GHRP receptor stimulation produces a substantially larger GH pulse than either compound alone.

What to Monitor

- IGF-1 (with and without GHRP partner)
- Fasted glucose, HbA1c
- Water retention (fluid retention common in first weeks)

Notable Cautions: Almost never used as a standalone - its value is in GHRP co-administration. Using it alone produces minimal effect. Timing precision matters: co-administer with GHRP in same fasted window.

Stack Compatibility: Best combined with Ipamorelin (most common pairing in educational literature). Also stacks with GHRP-2 or GHRP-6. Not typically combined with CJC-1295 with DAC (redundant mechanisms). Compatible with recovery and neuropeptide classes.

CJC-1295 (with DAC)

CJC-1295 with Drug Affinity Complex | Long-acting GHRH analog

CLASS	GHRH Analog (long-acting)
PRIMARY MECHANISM	DAC modification allows covalent binding to serum albumin, dramatically extending half-life; creates sustained background GHRH elevation rather than discrete pulses
TYPICAL DOSING RANGE (EDUCATIONAL)	1-2 mg per administration; weekly or twice-weekly dosing
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	8-12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Sustained GH elevation pattern (distinct from pulsatile model)
- Convenience of once-weekly dosing
- GH-axis support in individuals who prefer low-frequency injection schedules

Key Research Notes

Human Data Available

The DAC modification enables a half-life of 6-8 days, allowing weekly dosing. This creates a fundamentally different pharmacokinetic profile than short-acting GHRH analogs. Whether sustained background GHRH elevation is superior or inferior to pulsatile patterns is an open educational discussion. Human trials confirmed IGF-1 elevation with weekly dosing.

What to Monitor

- IGF-1 (draw 3-4 days post-injection for plateau-phase reading)
- Fasted glucose, HbA1c (sustained GH elevation has more chronic insulin resistance potential)
- Water retention (more pronounced with long-acting pattern)

Notable Cautions: The non-pulsatile nature of this compound raises theoretical concerns about chronic somatotroph suppression and desensitization over time - a debated point in peptide research community. Higher monitoring frequency for glucose is appropriate due to sustained GH elevation pattern.

Stack Compatibility: May be used with GHRPs (weekly CJC-DAC + daily Ipamorelin is a protocol found in educational literature). Not recommended to stack with CJC-1295 no DAC (redundant). Compatible with recovery and neuropeptide classes.

Sermorelin

Sermorelin Acetate | First 29 amino acids of endogenous GHRH

CLASS	GHRH Analog (natural sequence fragment)
PRIMARY MECHANISM	Binds GHRH receptor on pituitary somatotrophs; stimulates GH release via natural ligand sequence; preserves feedback regulation (less supraphysiologic ceiling)
TYPICAL DOSING RANGE (EDUCATIONAL)	200–500 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	12–24 weeks (longer cycles more common than other GHRH analogs)
TYPICAL OFF-PERIOD	4–8 weeks

Primary Use Cases

- GH deficiency (adult-onset) research and treatment context
- Gentler GH axis support with preserved natural feedback
- Anti-aging and longevity protocols
- Physician-supervised GH optimization programs

Key Research Notes

Substantial Human Data

Sermorelin was FDA-approved for pediatric GH deficiency (though approval was withdrawn when synthetic GH became more economical). It has among the most favorable safety profiles of any GHRH analog due to its natural sequence and preserved ceiling effect from pituitary feedback mechanisms.

What to Monitor

- IGF-1 at baseline, week 6, end of cycle
- Fasted glucose, HbA1c
- Antibody formation (longer cycles - rare, but monitored in clinical use)

Notable Cautions: Injection site reactions are the most common adverse effect. Headache and flushing reported in early use. Very long cycles (24 weeks+) in clinical contexts have prompted antibody testing - discussed with physician for extended protocols.

Stack Compatibility: Combines with all GHRPs. Often the GHRH analog of choice for longer, gentler protocols due to its natural sequence and safety profile.

Tesamorelin

Tesamorelin | GHRH analog with trans-2-hexenoic acid modification for stability

CLASS	GHRH Analog (stabilized)
PRIMARY MECHANISM	GHRH receptor agonist; modification improves stability vs. native GHRH; stimulates pituitary GH release with preserved pulsatility; clinically documented visceral fat reduction
TYPICAL DOSING RANGE (EDUCATIONAL)	1-2 mg per administration; once daily
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	12-26 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Visceral adiposity reduction (FDA-approved for HIV lipodystrophy)
- GH axis optimization in longevity protocols
- Metabolic health improvement
- Body recomposition (visceral fat reduction + lean mass support)

Key Research Notes

Substantial Human Data - FDA Approved Drug

Tesamorelin (Egrifta) holds FDA approval for HIV-associated lipodystrophy - making it the GHRH analog with the most robust human clinical data. Randomized controlled trials have confirmed statistically significant visceral fat reduction and IGF-1 elevation. It is the reference standard for GHRH analog educational comparison.

What to Monitor

- IGF-1 (primary efficacy and safety marker)
- Fasted glucose, HbA1c (clinical trials showed slight glucose increase; clinically relevant in pre-diabetic individuals)
- Waist circumference (primary outcome in clinical trials)

Notable Cautions: Clinical trial data showed modest HbA1c increases. Contraindicated in active malignancy per FDA labeling. Injection site reactions in some subjects. Arthralgia (joint pain) reported in clinical trial cohorts.

Stack Compatibility: Combines with GHRPs for amplified effect. Given its visceral fat focus, it is often paired with Ipamorelin in body composition protocols. Compatible with recovery peptide classes.

CLASS 04

Fat-Selective Peptides

AOD-9604

Anti-Obesity Drug 9604 | C-terminal fragment of human GH (amino acids 176-191)

CLASS	GH Fragment / Lipolytic Peptide
PRIMARY MECHANISM	Mimics the lipolytic (fat-burning) domain of GH without engaging the growth-promoting IGF-1 pathway; stimulates lipolysis and inhibits lipogenesis via beta-3 adrenergic receptor pathway
TYPICAL DOSING RANGE (EDUCATIONAL)	300-500 mcg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Targeted fat loss (particularly visceral and subcutaneous adipose)
- Body composition without IGF-1 elevation (individuals who want lipolytic effects without GH axis stimulation)

- Combination with GLP-1 class agents for metabolic protocols

Key Research Notes

Limited Human Data AOD-9604 received FDA GRAS (Generally Recognized as Safe) status as a food additive - notable for its safety assessment, though this does not validate its lipolytic efficacy claims in humans. Phase II trials for obesity showed modest results. The mechanistic case for its lipolytic effect is well-supported in animal research.

What to Monitor

- Body composition (key outcome marker)
- Fasted glucose (unlike GH, AOD-9604 does not appear to affect insulin sensitivity significantly - but monitoring remains prudent)
- IGF-1 (should not increase with AOD-9604 use - confirms the compound is operating as intended)

Notable Cautions: Limited long-term human safety data. Should be dosed in fasted window for lipolytic effect alignment. Not a substitute for caloric management.

Stack Compatibility: Compatible with GH secretagogue protocols (different mechanism - can be used alongside GHRP/GHRH stacks without conflict). No known pharmacokinetic conflicts with other compounds in this guide.

CLASS 05

Longevity and Mitochondrial Peptides

Epithalon

Epithalon (Epitalon) | Tetrapeptide Ala-Glu-Asp-Gly derived from pineal gland extract Epithalamin

CLASS	Telomere / Longevity Peptide
PRIMARY MECHANISM	Stimulates telomerase activity; proposed role in telomere elongation; pineal gland regulation; antioxidant properties; circadian rhythm normalization via melatonin pathway interaction
TYPICAL DOSING RANGE (EDUCATIONAL)	5-10 mg per administration; typically given as a course
ADMINISTRATION ROUTE	SubQ injection or IV (IV in research settings)
TYPICAL CYCLE LENGTH	10-20 day courses
TYPICAL OFF-PERIOD	4-6 months between courses

Primary Use Cases

- Longevity and anti-aging protocols
- Sleep quality improvement (melatonin pathway)
- Circadian rhythm support
- General antioxidant and cellular health support

Key Research Notes

Limited Human Data

Most Epithalon research originates from Soviet and Russian research groups, with some human observational data showing longevity-associated biomarker improvements. The telomerase stimulation mechanism is biologically plausible and well-documented in cell culture. Translation to human longevity outcomes is speculative at this time.

What to Monitor

- Sleep quality (often the most reliably noticed subjective effect)
- General CBC at baseline

- No established biomarker specific to Epithalon efficacy in routine clinical use

Notable Cautions: The theoretical concern that telomerase activation could promote oncogenesis is an active scientific debate. This context should be discussed with a physician, particularly for individuals with cancer history or strong family history of cancer.

Stack Compatibility: Short course nature means it is generally run independently or between longer GH-axis cycles. No known pharmacokinetic conflicts.

SS-31

SS-31 (Elamipretide) | Szeto-Schiller peptide; mitochondria-targeted antioxidant tetrapeptide

CLASS	Mitochondrial Targeting Peptide
PRIMARY MECHANISM	Selectively concentrates in the inner mitochondrial membrane; binds cardiolipin; stabilizes cristae architecture; reduces mitochondrial ROS; improves ATP production efficiency
TYPICAL DOSING RANGE (EDUCATIONAL)	1-10 mg per administration (wide range across research studies)
ADMINISTRATION ROUTE	SubQ injection; IV in clinical trials
TYPICAL CYCLE LENGTH	4-12 weeks
TYPICAL OFF-PERIOD	4-8 weeks

Primary Use Cases

- Mitochondrial function optimization
- Age-related mitochondrial decline research
- Heart failure (active clinical trials - Elamipretide)
- Skeletal muscle mitochondrial efficiency

Key Research Notes

Strong Animal Data | Human Trials Ongoing

SS-31 (branded as Elamipretide in clinical development) is in Phase II/III trials for heart failure with preserved ejection fraction and Barth syndrome. Animal data across aging, cardiac, renal, and neurological models is substantial. The mitochondrial targeting mechanism is among the most compelling in longevity peptide research.

What to Monitor

- Subjective energy and endurance (most commonly reported effect)
- No established routine biomarker for SS-31 efficacy in clinical monitoring

- Injection site reactions (noted in clinical trials)

Notable Cautions: Injection site reactions are the most common adverse effect in clinical trials. Long-term SubQ safety data limited. Human dose-finding is not complete.

Stack Compatibility: No mechanistic conflicts with any compound in this guide. Often included in advanced longevity stacks alongside MOTS-c, recovery peptides, or GH-axis compounds.

MOTS-c

Mitochondrial Open Reading Frame of the 12S rRNA-c | Mitochondrial-derived peptide

CLASS	Mitochondrial-Derived Peptide (MDP)
PRIMARY MECHANISM	Encoded in mitochondrial 12S rRNA; regulates nuclear gene expression; activates AMPK pathway; improves insulin sensitivity; promotes metabolic flexibility; exercise mimetic properties in animal models
TYPICAL DOSING RANGE (EDUCATIONAL)	5-10 mg per administration
ADMINISTRATION ROUTE	SubQ injection
TYPICAL CYCLE LENGTH	4-8 weeks
TYPICAL OFF-PERIOD	4 weeks minimum

Primary Use Cases

- Metabolic health and insulin sensitivity
- Exercise performance and recovery
- Longevity and healthspan protocols
- Age-related metabolic decline (animal models)

Key Research Notes

Strong Animal Data | Very Limited Human Data

MOTS-c is a fascinating discovery in mitochondrial biology. First described in 2015, it represents a new class of hormone-like signaling molecules encoded in mitochondrial DNA. Mouse studies show dramatic effects on insulin sensitivity, exercise performance, and longevity. First-in-human safety studies are just beginning. This is among the most scientifically novel compounds in this guide.

What to Monitor

- Fasted glucose and insulin sensitivity markers (HOMA-IR)
- Subjective energy and performance
- Injection site reactions

Notable Cautions: Minimal human safety data. Reconstituted peptide requires strict cold-chain storage. This compound sits at the frontier of peptide research - educational context appropriate, clinical application not established.

Stack Compatibility: No known conflicts with any compound in this guide. Metabolic and mitochondrial mechanisms are complementary to GH-axis and recovery peptide protocols.

CLASS 06

Neuropeptides

Semax

Semax | Synthetic ACTH 4-7 analog; heptapeptide with Pro-Gly-Pro C-terminal addition

CLASS	Nootropic Neuropeptide / BDNF Upregulator
PRIMARY MECHANISM	Increases BDNF (Brain-Derived Neurotrophic Factor) expression; upregulates NGF; modulates dopamine and serotonin systems; improves cerebral blood flow in animal models; neuroprotective effects in ischemia models
TYPICAL DOSING RANGE (EDUCATIONAL)	200-600 mcg per administration (0.1% solution common in literature)
ADMINISTRATION ROUTE	SubQ injection (primary); nasal administration studied in Russian literature (nasal references excluded per curriculum policy)
TYPICAL CYCLE LENGTH	2-4 weeks
TYPICAL OFF-PERIOD	2-4 weeks

Primary Use Cases

- Cognitive enhancement and mental clarity
- Focus and motivation during demanding cognitive work
- Neuroprotection and BDNF support
- Mood and motivation optimization
- Post-stroke and cognitive recovery (approved pharmaceutical in Russia)

Key Research Notes

Human Data Available

Semax is an approved pharmaceutical in Russia for stroke recovery and cognitive conditions. The Russian literature is extensive but not always translated or independently replicated in Western research. BDNF upregulation is a well-characterized mechanism with strong scientific backing as a longevity and cognitive health target.

What to Monitor

- Subjective cognitive function, focus, and mood (primary outcome indicators)
- Sleep quality (stimulating effect - avoid bedtime dosing)
- Blood pressure (some reports of mild elevation at higher doses)

Notable Cautions: Stimulating neuropeptide - contraindicated in bedtime window. May increase anxiety in sensitive individuals. Cycle length limitations prevent tolerance development. Not for use during acute anxiety episodes or in individuals with dopamine-sensitive conditions without physician guidance.

Stack Compatibility: Does not share mechanisms with GH-axis, recovery, or mitochondrial compounds - compatible in same protocol at different timing windows. Not stacked with Selank in the same administration window (opposing action profiles). May alternate Semax weeks with Selank weeks.

Selank

Selank | Synthetic tuftsin analog (Thr-Lys-Pro-Arg-Pro-Gly-Pro) with anxiolytic properties

CLASS	Anxiolytic Neuropeptide / Nootropic
PRIMARY MECHANISM	Modulates GABAergic and serotonergic systems; increases BDNF similarly to Semax; inhibits enkephalin-degrading enzymes; immune-modulating properties via tuftsin receptor activity
TYPICAL DOSING RANGE (EDUCATIONAL)	250–500 mcg per administration
ADMINISTRATION ROUTE	SubQ injection (primary)
TYPICAL CYCLE LENGTH	2–4 weeks
TYPICAL OFF-PERIOD	2–4 weeks

Primary Use Cases

- Anxiety reduction without sedation
- Cognitive performance under stress
- Mood stabilization
- Immune modulation (tuftsin analog properties)
- Sleep quality improvement (evening dosing compatible)

Key Research Notes

Human Data Available

Selank is also an approved pharmaceutical in Russia. Research demonstrates anxiolytic effects with a notably benign side effect profile compared to benzodiazepines — no reported dependence, withdrawal, or sedation in research literature. BDNF upregulation complements Semax; the two share a neurotrophin-promoting mechanism despite their opposing arousal profiles.

What to Monitor

- Subjective anxiety levels and mood (primary outcome)
- Sleep quality (often improved)

- Cognitive performance (nootropic effect is mild but reliable in research)

Notable Cautions: Mild sedation possible at higher doses. Not recommended before driving or operating machinery until individual response is known. May interact with anxiolytic medications - physician review required.

Stack Compatibility: Complementary to Semax in an alternating cycle pattern. Compatible with GH-axis and recovery compounds. Evening or bedtime dosing window does not conflict with any other compound in this guide.

Dihexa

Dihexa (PNB-0408) | Angiotensin IV analog; hepatocyte growth factor activator

CLASS	Nootropic Neuropeptide / HGF/MET Agonist
PRIMARY MECHANISM	Potentiates hepatocyte growth factor (HGF) activity via MET receptor; promotes synaptogenesis and hippocampal neurogenesis; dramatically enhances spatial memory and cognition in animal models
TYPICAL DOSING RANGE (EDUCATIONAL)	Not established; research dosing ranges are variable and extrapolation from animal data is highly uncertain
ADMINISTRATION ROUTE	SubQ injection or transdermal (transdermal studied in animal models)
TYPICAL CYCLE LENGTH	1–3 weeks; very short due to potency and limited data
TYPICAL OFF-PERIOD	4+ weeks minimum

Primary Use Cases

- Cognitive enhancement (the most potent nootropic compound in animal research)
- Memory and learning optimization
- Neurodegenerative disease research (Alzheimer's models)

Key Research Notes

Animal Data Only

Dihexa is consistently described in research literature as several orders of magnitude more potent than BDNF itself in synaptogenesis assays. This extraordinary potency in animal models has generated significant interest – and significant caution. There are no human pharmacokinetic, safety, or efficacy trials as

of this writing. The absence of human data combined with its potency makes this the highest-uncertainty compound in this guide.

What to Monitor

- Subjective cognitive function and mood
- Any unexpected neurological symptoms (headache, mood changes, sensory changes) - document and cease use if observed
- No established biomarker for Dihexa monitoring in humans

Notable Cautions: Extreme caution warranted due to complete absence of human safety data and extraordinary potency. The HGF/MET pathway is involved in cell growth and proliferation - the same theoretical oncological concern that applies to pro-angiogenic peptides applies here with additional uncertainty. This compound is educational context only, not a routine protocol recommendation. Physician discussion is essential before any consideration.

Stack Compatibility: Given the uncertainty, most educational frameworks recommend using Dihexa as a standalone compound - not in combination with other neurologically active compounds - when it is used at all.

DSIP

Delta Sleep-Inducing Peptide | Naturally occurring neuropeptide; first isolated from venous blood of sleeping rabbits

CLASS	Sleep-Modulating Neuropeptide
PRIMARY MECHANISM	Proposed modulation of delta (slow-wave) sleep; putative stress-reducing effects; LH pulsatility modulation; wide distribution across brain regions; exact mechanism not fully characterized
TYPICAL DOSING RANGE (EDUCATIONAL)	100–500 mcg per administration
ADMINISTRATION ROUTE	SubQ injection; 30–60 min before sleep
TYPICAL CYCLE LENGTH	1–4 weeks
TYPICAL OFF-PERIOD	Variable; situational use

Primary Use Cases

- Sleep quality improvement, particularly delta-wave sleep
- Travel and time zone disruption recovery
- Stress-associated sleep impairment
- Adjunct to GH peptide bedtime dosing protocols

Key Research Notes

Mixed Human Data

DSIP has a long research history dating to the 1970s but its mechanism and efficacy remain debated. Some human studies show improved sleep architecture; others show inconsistent results. It is a naturally occurring peptide in human plasma with varying concentrations across the sleep-wake cycle. Its bioavailability via SubQ route in humans is not fully characterized.

What to Monitor

- Sleep quality metrics (subjective scores, wearable data if available)
- Dream recall and sleep continuity

- Next-day cognitive function (should not produce morning grogginess)

Notable Cautions: Most commonly reported side effect is vivid dreams (considered a positive indicator by many). Some reports of next-morning sedation at higher doses. Not a sedative-hypnotic - mechanism is distinct from benzodiazepines or Z-drugs.

Stack Compatibility: Compatible with bedtime GHRP/GHRH dosing (can be administered in the same bedtime window). No known conflicts with recovery or mitochondrial compound classes. Not stacked with Semax in same window.

All dosing ranges cited are from published research literature. Research data tags: Animal = primarily rodent/animal studies; Human = human trial data exists; Limited = sparse or contested data. Consult a licensed physician before any compound consideration.