RonanRx
PERSONALIZED MEDICINE

CLINICAL MONOGRAPH · NEURO & COGNITIVE (UNDER FDA REVIEW)

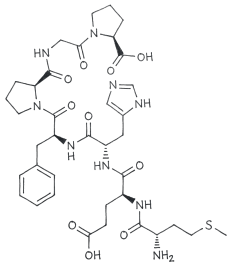
Semax

ACTH-fragment peptide with physician-request review

Semax is a small synthetic peptide developed in Russia in the late 1980s. It is a chemical analog of a fragment of adrenocorticotropic hormone (ACTH), specifically the part of ACTH numbered 4 through 10, with an extra three-amino-acid tail (Pro-Gly-Pro) that makes it resistant to rapid breakdown in the body. It is given as nasal drops.

In Russia, semax is approved as a prescription medicine for ischemic stroke, transient ischemic attack, cerebrovascular insufficiency, and (in children) attention and cognitive disorders [gusev1997; gusev2005]. Most of the human clinical trials supporting these uses were conducted in the Russian Federation and published in Russian-language journals; Western regulators have not reviewed or approved semax.

Semax has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.



EVIDENCE POSTURE EMERGING PRECLINICAL

REVIEWED **2026-05-11**


State-licensed 503A


Pharmacist reviewed


Doctor led


Cold-chain ready


Patient choice preserved

Contents

Click any section to jump there. Page numbers update on render.

FOR CLINICIANS

Semax (Met-Glu-His-Phe-Pro-Gly-Pro) is a synthetic ACTH(4-10) analog developed in the late 1980s at the Institute of Molecular Genetics, Russian Academy of Sciences, by the laboratory of Myasoedov and colleagues [medvedeva2014; medvedeva2017; dergunova2021]. The C-terminal Pro-Gly-Pro extension confers proteolytic stability sufficient for intranasal absorption with measurable CNS exposure [shevchenko2006] [dmitrieva2010]. Mechanism is multimodal: upregulation of BDNF and NGF and their cognate receptors (trkB, trkA) in hippocampus, basal forebrain cholinergic nuclei, and ischemic penumbra; modulation of immune-response and pro-inflammatory gene expression after focal cerebral ischemia; mild mu-opioid receptor activity contributing to behavioral and analgesic profile [maslova2003, grivennikov2008]; and possible transthyretin binding as a peripheral neuroprotective mechanism [vyunova2016].

Semax has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.

Why Personalized Semax

The evidence base for Semax is concentrated in Russian clinical and pharmacology literature, with use described for neurologic indications abroad. That record has not been converted into an FDA-approved US product or a modern US safety and dosing program.

Physicians may submit patient-specific prescription requests for Semax for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, supported by patient-specific documentation, and approved by the dispensing pharmacy. Availability is determined case by case. This is not a consumer access promise; it is a clinical, sourcing, formulation, and regulatory review process. FDA has scheduled Semax-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting.

The legitimate US pathway is not to treat foreign approval or online availability as a substitute for pharmacy review. A patient-specific prescription request must be reviewed against US compounding requirements and the actual evidence record.

Quick Facts About Semax

Category: ACTH(4-10) analog heptapeptide, research peptide in the U.S.

Active ingredient: Semax, Met-Glu-His-Phe-Pro-Gly-Pro, a synthetic heptapeptide combining ACTH(4-7) (Met-Glu-His-Phe) with a C-terminal Pro-Gly-Pro extension that confers proteolytic stability and an extended half-life relative to native ACTH fragments

Origin: Developed in the late 1980s at the Institute of Molecular Genetics, Russian Academy of Sciences, Moscow

Russian/EAEU regulatory status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

U.S. FDA status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

WADA status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

RonanRx position: Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

Physicians may submit patient-specific prescription requests for Semax for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case.

- **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
- **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
- **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
- **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
- **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is Semax?

Semax is a synthetic heptapeptide with the amino acid sequence Met-Glu-His-Phe-Pro-Gly-Pro (single-letter MEHFPGP). The first four residues correspond to positions 4 through 7 of adrenocorticotropic hormone (ACTH); the C-terminal Pro-Gly-Pro tripeptide is appended to confer resistance to

aminopeptidase and carboxypeptidase degradation and to extend functional half-life relative to native ACTH(4-10), which is rapidly cleaved in plasma and tissue.

Semax was synthesized and characterized in the late 1980s by Ashmarin, Myasoedov, and colleagues at the Institute of Molecular Genetics of the Russian Academy of Sciences in Moscow, with the design hypothesis that ACTH(4-10) carries behaviorally active, melanocortin-related but glucocorticoid-independent CNS activity, and that proteolytic stabilization would yield a clinically tractable nootropic and neuroprotective agent [dolotov2003; shevchenko2006]. The pharmaceutical product is manufactured by Innovative Research Production Center 'Peptogen' (Moscow) and marketed in the Russian Federation and Eurasian Economic Union as Semax-MV 0.1% and 1% intranasal drops.

Outside the Russian Federation and EAEU, semax is not an approved medicinal product. In the United States it is not FDA-approved in any U.S. indication, is not a generally recognized as safe (GRAS) substance, and is not a listed dietary ingredient. FDA has placed semax under 503A bulk substance evaluation in Category 2, indicating identified safety concerns or insufficient evidence to support a positive determination; 503A patient-specific compounding from bulk semax is not permitted while the substance is in Category 2 [fda503a; fda_category2].

How Semax Works

Semax is a multimodal neuroactive peptide. Its principal CNS effects in preclinical studies are upregulation of brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF), together with their tyrosine kinase receptors trkB and trkA, in hippocampus, basal forebrain cholinergic nuclei, and ischemic cortical penumbra [dolotov2006_brainres; dolotov2006_jneuro; shadrina2010]. Through this neurotrophin-axis activation, semax is hypothesized to support cholinergic neuron survival, synaptic plasticity, and post-ischemic functional recovery.

After focal cerebral ischemia-reperfusion in rat models, semax administration alters expression of large gene-expression panels related to inflammation, immune response, vascular remodeling, and apoptosis [dolotov2003; medvedeva2014; medvedeva2017]. The pro-inflammatory mediator suppression and vascular-gene modulation profiles are mechanistically aligned with the clinical neuroprotection claim in acute ischemic stroke [filippenkov2024].

Semax exhibits mild mu-opioid receptor activity, contributing to its behavioral profile (anxiolytic-like and antinociceptive effects in rodent models) without producing classical opioid sedation, respiratory depression, or dependence at studied doses [maslova2003, grivennikov2008]. Peripheral binding to transthyretin has been proposed as an additional vector for neuroprotective signaling [vyunova2016]. Semax does not display glucocorticoid-axis effects characteristic of full-length ACTH (ACTH 1-39) because it lacks the melanocortin receptor-2 (MC2R) binding determinants required for adrenocortical steroidogenesis [agapova2007; filippenkov2020; dergunova2021].

Biological Role of Semax

ACTH and its proteolytic fragments have been recognized since the 1970s as carrying behaviorally active, melanocortin-mediated CNS effects that are distinct from the steroidogenic action of full-length ACTH on the adrenal cortex. ACTH(4-10) was identified as a key behaviorally active fragment, but its therapeutic application was limited by rapid proteolytic degradation. The semax program represented an early-1990s peptide-engineering response: append a stabilizing tripeptide tail (Pro-Gly-Pro) to a behaviorally active ACTH fragment to yield a clinically tractable nootropic and neuroprotective agent.

Semax sits within a family of melanocortin-system-derived peptides studied for neuroprotection. The C-terminal Pro-Gly-Pro tripeptide is itself bioactive, activating neurotrophin transcription independently of the full heptapeptide [dmitrieva2010], suggesting that semax acts in part through Pro-Gly-Pro-mediated mechanisms that may overlap with other glyprolines studied for cytoprotection.

Detailed Mechanism of Semax

Neurotrophin axis. Dolotov and colleagues (2003, Dokl Biol Sci) reported that intranasal semax stimulates BDNF expression across multiple rat brain areas in vivo [dolotov2003]. Follow-on work in J Neurochem (2006) demonstrated specific semax binding sites in rat basal forebrain and a corresponding increase in BDNF protein in basal forebrain cholinergic nuclei [dolotov2006_jneuro]; a parallel Brain Research report demonstrated regulation of BDNF and trkB receptor expression in the hippocampus [dolotov2006_brainres]. Agapova et al. (2007, Neurosci Lett) and Shadrina et al. (2010, J Mol Neurosci) extended the analysis to NGF gene expression and characterized the temporal dynamics of NGF and BDNF transcript induction after semax administration [agapova2007, shadrina2010]. Dmitrieva et al. (2010, Cell Mol Neurobiol) showed that both semax and its C-terminal Pro-Gly-Pro fragment activate transcription of neurotrophins and their receptor genes in the rat brain after cerebral ischemia, indicating that the Pro-Gly-Pro extension is itself bioactive [dmitrieva2010].

Post-ischemic transcriptomics. Medvedeva and colleagues (2014, BMC Genomics) reported genome-wide expression profiling in the rat focal-ischemia model and demonstrated that semax preferentially affects genes related to the immune and vascular systems [medvedeva2014]. A 2017 follow-up in Mol Genet Genomics narrowed the focus to immune-response gene regulation after ischemic brain injury [medvedeva2017]. Dergunova et al. (2021) demonstrated that semax suppresses mRNA transcripts encoding pro-inflammatory mediators induced by reversible cerebral ischemia [dergunova2021]. Filippenkov et al. (2020 and 2024) reported broad transcriptome compensation by ACTH-like peptides after ischemia-reperfusion, with the 2024 Biomedicines paper documenting that semax-class peptides restore ischemia-disrupted gene expression profiles by 24 hours post-stroke [filippenkov2020, filippenkov2024].

Opioidergic and behavioral pharmacology. Maslova et al. (2003, Neurosci Behav Physiol) reported that semax corrects post-hypoxic hyperactivity and learning deficits in a developmental rat model, consistent with a behavioral-stabilization profile relevant to ADHD-like phenotypes [maslova2003]. Grivennikov et al. (2008, Restor Neurol Neurosci) characterized semax effects on rat basal forebrain cholinergic neurons and reported behavioral activity without the dependence liability associated with full opioid agonists [grivennikov2008]. Tsai (2007, Med Hypotheses) proposed semax as a candidate ADHD and Rett syndrome therapeutic on mechanistic grounds [tsai2007].

Pharmacokinetics. Shevchenko et al. (2006, Bioorg Khim) characterized the kinetics of semax penetration into rat brain and blood after intranasal administration using tritium-labeled peptide, providing the principal published PK substrate for the clinical intranasal-drop dosing regimen [shevchenko2006]. The Pro-Gly-Pro C-terminal extension is critical: native ACTH(4-10) is degraded within minutes in plasma, whereas semax retains measurable CNS bioactivity over hours after intranasal dosing.

Semax Research History

The semax program originated in the late 1980s in the laboratory of N.F. Myasoedov and I.P. Ashmarin at the Institute of Molecular Genetics, Russian Academy of Sciences, Moscow. The design rationale was to stabilize a behaviorally active ACTH(4-7) sequence via a proteolytically resistant Pro-Gly-Pro extension. Russian preclinical work through the 1990s established a neuroprotective profile in focal cerebral ischemia models and a behavioral-stabilization profile in post-hypoxic developmental injury, leading to Russian Ministry of Health approval of Semax-MV intranasal drops for ischemic stroke and cerebrovascular insufficiency [filippenkov2020; filippenkov2024].

The first Russian clinical report of semax in acute hemispheric ischemic stroke was published by Gusev and colleagues in 1997 (Zh Nevrol Psikhiatr) and described clinical and electrophysiological benefit during the acute period [gusev1997] [medvedeva2017]. A 2005 Russian-language report (Gusev et al.) described semax in prevention of disease progression and exacerbations in patients with cerebrovascular insufficiency [gusev2005] [agapova2007]. A 2018 Russian-language efficacy paper from the same group described semax across different stages of ischemic stroke [gusev2018] [dolotov2003; dolotov2006_jneuro]. Russian preclinical mechanistic work in the 2000s established the BDNF/NGF axis as the principal pharmacological target, and Russian transcriptomic work from the 2010s and early 2020s extended the mechanistic picture to large-scale immune and inflammatory gene-expression modulation after focal ischemia [medvedeva2014; dolotov2006_brainres; shadrina2010].

Western engagement with semax has been limited. The principal Western-journal hypothesis paper is Tsai (2007, Med Hypotheses), proposing semax for ADHD and Rett syndrome on mechanistic grounds [tsai2007]. No FDA-registration phase 2 or phase 3 trial of semax has been reported in any indication. No Cochrane systematic review or large Western-led independent replication of the Russian stroke data exists. FDA has placed semax in Category 2 of its 503A bulk drug substance evaluation, indicating that the agency

considers the available evidence insufficient or the safety profile insufficiently characterized to support 503A compounding [fda503a, fda_category2] [dergunova2021].

Semax Timeline

Late 1980s Semax (Met-Glu-His-Phe-Pro-Gly-Pro) synthesized and characterized at the Institute of Molecular Genetics, Russian Academy of Sciences, Moscow, in the laboratory of Myasoedov and Ashmarin

1997 • Gusev et al [gusev1997]. publish the first Russian clinical and electrophysiological report of semax in acute hemispheric ischemic stroke (Zh Nevrol Psikhiatr)

2003 • Dolotov et al [dolotov2003]. (Dokl Biol Sci) report that intranasal semax stimulates BDNF expression across multiple rat brain regions in vivo

2003 • Maslova et al [maslova2003]. (Neurosci Behav Physiol) demonstrate that semax corrects post-hypoxic hyperactivity and learning deficits in a developmental rat ADHD-like model

2004 • Sheremet et al [sheremet2004]. (Vestn Oftalmol) report experimental rationale for using semax as a neuroprotector in optic-nerve diseases

2005 • Gusev et al [gusev2005]. (Zh Nevrol Psikhiatr) report semax in prevention of disease progress and exacerbations in patients with cerebrovascular insufficiency

2006 • Dolotov et al [dolotov2006_jneuro]. (J Neurochem) report specific semax binding sites in rat basal forebrain and increased BDNF protein in cholinergic nuclei

2006 • Dolotov et al [dolotov2006_brainres]. (Brain Res) demonstrate semax regulation of BDNF and trkB receptor expression in the rat hippocampus

2006 • Shevchenko et al [shevchenko2006]. (Bioorg Khim) characterize kinetics of semax penetration into brain and blood after intranasal administration using tritium-labeled peptide

2007 • Tsai (Med Hypotheses) publishes the principal Western-journal hypothesis paper proposing semax as a candidate ADHD and Rett syndrome therapeutic on mechanistic grounds [tsai2007]

2007 • Agapova et al [agapova2007]. (Neurosci Lett) characterize neurotrophin gene expression in rat brain under semax action

2008 • Grivennikov et al [grivennikov2008]. (Restor Neurol Neurosci) characterize semax effects on rat basal forebrain cholinergic neurons

2010 • Dmitrieva et al [dmitrieva2010]. (Cell Mol Neurobiol) show that semax and its C-terminal Pro-Gly-Pro fragment activate transcription of neurotrophins and their receptor genes after cerebral ischemia

- **2010** • Shadrina et al [shadrina2010]. (J Mol Neurosci) compare temporal dynamics of NGF and BDNF gene expression under semax action
- **2014** • Medvedeva et al [medvedeva2014]. (BMC Genomics) report genome-wide transcriptional analysis demonstrating that semax preferentially affects immune- and vascular-system genes in rat brain focal ischemia
- **2016** • Vyunova et al [vyunova2016]. (Mol Gen Mikrobiol Virusol) propose a role for transthyretin in the biological mechanism of semax neuroprotection
- **2017** • Medvedeva et al [medvedeva2017]. (Mol Genet Genomics) report that semax regulates expression of immune response genes during ischemic brain injury in rats
- **2018** • Gusev et al [gusev2018]. (Zh Nevrol Psikhiatr) report on the efficacy of semax in the treatment of patients at different stages of ischemic stroke
- **2020** • Filippenkov et al [filippenkov2020]. (Genes) publish transcriptome-level analysis of the protective properties of semax following cerebral ischemia-reperfusion in rats
- **2021** • Dergunova et al [dergunova2021]. (Mol Biol Mosk) demonstrate that semax suppresses mRNA transcripts encoding pro-inflammatory mediators induced by reversible cerebral ischemia
- **2024** • Filippenkov et al [filippenkov2024]. (Biomedicines) demonstrate that ACTH-like peptides including semax compensate ischemia-disrupted rat brain gene expression profiles 24 hours after experimental stroke

Clinical Contexts for Semax

Acute ischemic stroke and cerebrovascular insufficiency (Russian Federation indication)

EMERGING

Evidence should be interpreted in context for Semax. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Evidence should be interpreted in context for Semax. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Attention-deficit/hyperactivity disorder and cognitive deficit in children (Russian Federation pediatric label) EMERGING

Evidence should be interpreted in context for Semax. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Evidence should be interpreted in context for Semax. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Optic-nerve disease and other neuroprotective applications PRECLINICAL

Experimental and preclinical only; not an approved indication anywhere.

Evidence should be interpreted in context for Semax. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Compounded Semax (503A)

Physicians may submit patient-specific prescription requests for pharmacy review. For Semax, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for Semax is concentrated in Russian clinical and pharmacology literature, with use described for neurologic indications abroad. That record has not been converted into an FDA-approved US product or a modern US safety and dosing program.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. FDA has scheduled Semax-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For Semax, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for Semax are reviewed before any preparation is made or released. The legitimate US pathway is not to treat foreign approval or online availability as a substitute for pharmacy review. A patient-specific prescription request must be reviewed against US compounding requirements and the actual evidence record.

Semax Formulations and Routes

Form	Concentration	Description
Intranasal drops (Russian Federation approved product)	0.1% (1 mg/mL) and 1% (10 mg/mL) intranasal solution	Semax-MV is manufactured by Innovative Research Production Center 'Peptogen' (Moscow) and marketed in the Russian Federation and EAEU as a prescription intranasal drop preparation. The 0.1% strength is used for outpatient and pediatric indications; the 1% strength is used for acute neurological emergencies including ischemic stroke. The product is not FDA-approved and is not legally imported into the United States for prescription use.

Routes used in published literature: intranasal.

Semax Dosing

Route	Population	Range	Duration	Study type
Intranasal	Adults with ischemic stroke (Russian Federation Semax-MV labeled regimen)	Russian-language label: 1% solution administered as intranasal drops totaling 12, 18 mg/day in divided doses during the acute period of ischemic stroke. Dosing ranges and durations vary across Russian clinical reports.	10 days typical in published Russian trials	Russian Federation approved labeling and published Russian clinical reports
Intranasal	Adults with chronic cerebrovascular insufficiency (Russian Federation Semax-MV labeled regimen)	Russian-language label: 0.1% solution administered as intranasal drops totaling typically 600, 900 mcg/day in divided doses	10, 14 day courses repeated per Russian clinical practice	Russian Federation approved labeling and published Russian clinical reports

RonanRx does not publish a consumer dosing schedule for Semax. Any request requires a valid patient-specific prescription, supporting clinical rationale, and pharmacist review. Route, strength, dosing interval, monitoring expectations, and dispensing quantity would be determined case by case from the prescriber's documentation and pharmacy feasibility review.

Patients who travel abroad and have received semax in jurisdictions where it is approved should disclose this to U.S. prescribers, particularly in the context of stroke or cerebrovascular care, so that the prescriber can document the exposure history. Continuation of semax obtained outside the United States is not facilitated by RonanRx [gusev1997; gusev2005; gusev2018].

Semax Safety

Published Russian clinical data describe a generally favorable tolerability profile for intranasal semax in adult stroke and cerebrovascular insufficiency populations, with adverse events typically limited to mild local nasal irritation and infrequent reports of headache or transient changes in blood pressure [123]. The evidence base is small, concentrated in Russian-language journals, and not subject to the post-marketing pharmacovigilance infrastructure that supports U.S. or European regulatory decisions.

Long-term safety in pediatric populations, the population for which Semax-MV carries a Russian-Federation cognitive-deficit indication, is not well characterized by Western-standard controlled studies. Effects on hypothalamic-pituitary-adrenal axis development, neurotrophin-signaling-related off-target effects, and long-term cognitive trajectories have not been studied at a level that would support FDA approval. FDA's placement of semax in Category 2 of the 503A bulk drug substance evaluation reflects this evidentiary gap and the agency's safety-concern threshold [2122].

Patients who obtain semax from non-pharmaceutical sources (research-chemical suppliers, internet vendors) face additional safety risks unrelated to the molecule itself: unverified identity, potency, sterility, and endotoxin content. Such material is not subject to USP <797> sterile-compounding controls and may carry contamination, mis-dosing, and counterfeit risks. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Contraindications

Russian Federation labeling for Semax-MV lists contraindications including pregnancy, lactation, acute psychotic states, severe endocrine disorders, history of convulsive seizures, and known hypersensitivity to the peptide. These contraindications derive from the Russian regulatory record and have not been independently evaluated by FDA or EMA.

Because semax is not FDA-approved, is not available through legitimate U.S. pharmaceutical channels, and is in FDA Category 2 for 503A compounding evaluation, the practical U.S. contraindication is that semax should not be used outside an authorized investigational protocol [2122].

Drug interactions

Honest gap. No formal human drug-drug interaction studies of semax have been published. Russian-Federation labeling does not enumerate specific drug-drug interactions. Semax is a small peptide cleared by proteolytic catabolism and is not expected to participate in cytochrome P450-mediated metabolic interactions; however, this expectation is mechanistic rather than empirically demonstrated in dedicated DDI studies.

Searched: PubMed, FDA Drugs@FDA, DailyMed on 2026-05-11 · terms *semax AND (drug interaction OR pharmacokinetic interaction OR cytochrome)*.

Adverse events

Russian-language clinical reports describe mild local nasal irritation, occasional transient headache, and infrequent transient blood pressure changes as the principal observed adverse events with intranasal semax at labeled doses [1][2][3]. Serious adverse events have not been reported at clinically significant frequency in the published Russian trial literature, but these reports lack the systematic adverse-event capture, central adjudication, and pharmacovigilance follow-through expected of FDA-registration trials.

Off-label and research-chemical-channel use carries safety considerations unrelated to the molecule itself: contamination, identity errors, dose miscalculation, and counterfeit product. These risks are not captured in the published clinical literature because they are characteristic of the unregulated supply chain rather than of the peptide. RonanRx is not a source of semax under any compounding pathway.

Monitoring Semax Therapy

No RonanRx-specific monitoring protocol has been established for Semax. If a patient-specific prescription is submitted, supporting clinical rationale may be requested, and monitoring expectations would be reviewed case by case against the published evidence, route, sterile or nonsterile status, concomitant therapies, and patient risk factors.

Semax in Special Populations

Semax Evidence Quality

Evidence for semax is concentrated in Russian-language journals and in Russian preclinical mechanistic work [dolotov2003; dolotov2006_brainres; dolotov2006_jneuro]. The clinical evidence base for the principal Russian indications (acute ischemic stroke, cerebrovascular insufficiency, pediatric cognitive deficit) consists of small to moderate single-country trials reported across approximately three decades [gusev1997, gusev2005, gusev2018]; these reports have not been replicated in Western controlled trials, have not been systematically reviewed at a level supporting FDA approval, and do not meet the evidentiary standard required for FDA marketing authorization or for a positive determination on the 503A bulk drug substance list.

Preclinical mechanistic evidence is substantially stronger than the clinical evidence base [agapova2007; shadrina2010; dmitrieva2010]. Semax has a reproducible BDNF/NGF-axis activation profile, a reproducible post-ischemic gene-expression-modulation profile in rat focal-ischemia models, a characterized intranasal-PK substrate [shevchenko2006], and a documented developmental-injury behavioral phenotype [maslova2003] [filippenkov2020]. The preclinical-clinical translation gap, coherent mechanism plus modest, geographically concentrated clinical evidence, is the principal reason for the FDA

Category 2 placement in the 503A bulk substance evaluation [fda503a, fda_category2] [filippenkov2024]. RonanRx documents this evidence base so physician-submitted requests can be reviewed against the literature, the FDA process, and patient-specific rationale [medvedeva2014; medvedeva2017; dergunova2021].

Major Semax Clinical Studies

Study	Design	Participants	Duration	Finding
Gusev et al. (1997, Zh Nevrol Psikhiatr Im S S Korsakova), first clinical report of semax in acute ischemic stroke	Russian-language clinical and electrophysiological study of semax in acute hemispheric ischemic stroke	—	—	Reported clinical and electrophysiological benefit of intranasal semax during the acute period of hemispheric ischemic stroke; founding clinical citation for the Russian-Federation stroke indication [gusev1997]
Gusev et al. (2005, Zh Nevrol Psikhiatr), semax in cerebrovascular insufficiency	Russian-language clinical report of semax in prevention of disease progression and exacerbations in patients with cerebrovascular insufficiency	—	—	Reported reduction in disease progression and exacerbation frequency on intranasal semax in adults with chronic cerebrovascular insufficiency [gusev2005]
Gusev et al. (2018, Zh Nevrol Psikhiatr), semax across stages of ischemic stroke	Russian-language efficacy report across acute, subacute, and recovery stages of ischemic stroke	—	—	Reported efficacy of semax in the treatment of patients at different stages of ischemic stroke; reaffirmed the Russian-Federation acute-stroke indication [gusev2018]
Dolotov et al. (2006, J Neurochem), basal forebrain BDNF	Preclinical pharmacology study in rats using radiolabeled semax binding and BDNF protein quantitation	—	—	Semax binds specifically to sites in rat basal forebrain and increases BDNF protein levels in basal forebrain cholinergic nuclei [dolotov2006_jneuro]
Dolotov et al. (2006, Brain Res), hippocampal BDNF/ trkB	Preclinical pharmacology study in rats characterizing semax effects on BDNF	—	—	Semax regulates BDNF and trkB receptor expression in the rat hippocampus, consistent with a hippocampal-cognitive

Study	Design	Participants	Duration	Finding
	and trkB gene expression			substrate for the nootropic claim [dolotov2006_brainres]
Dmitrieva et al. (2010, Cell Mol Neurobiol), Pro-Gly-Pro bioactivity	Preclinical post-ischemia transcription study in rats comparing semax with its C-terminal Pro-Gly-Pro fragment	—	—	Both semax and its C-terminal Pro-Gly-Pro fragment activate transcription of neurotrophins and their receptor genes after cerebral ischemia, indicating that the Pro-Gly-Pro extension is itself bioactive [dmitrieva2010]
Medvedeva et al. (2014, BMC Genomics), focal-ischemia transcriptome	Genome-wide transcriptional analysis of rat brain in a focal cerebral ischemia model with and without semax	—	—	Semax preferentially affects genes related to immune and vascular systems in rat brain focal ischemia, providing a transcriptome-level substrate for the Russian neuroprotection claim [medvedeva2014]
Filippenkov et al. (2024, Biomedicines), 24-hour transcriptome compensation	Preclinical transcriptomic study of ACTH-like peptides including semax 24 hours after experimental stroke in rats	—	—	Semax-class peptides compensate the rat brain gene-expression profile disrupted by ischemia a day after experimental stroke, supporting the post-acute neuroprotection model [filippenkov2024]
Shevchenko et al. (2006, Bioorg Khim), intranasal pharmacokinetics	Tritium-labeled semax PK study characterizing penetration into rat brain and blood after intranasal administration	—	—	Established the principal published PK substrate for intranasal semax dosing; demonstrated measurable brain exposure after intranasal administration [shevchenko2006]
Maslova et al. (2003, Neurosci Behav Physiol), post-hypoxia ADHD-like model	Preclinical developmental rat model of ante- and postnatal hypoxia with hyperactivity and learning deficits	—	—	Semax corrects post-hypoxic hyperactivity and learning deficits, providing the preclinical rationale cited in the Russian-Federation pediatric ADHD/cognitive indication [maslova2003]

Study	Design	Participants	Duration	Finding
Tsai (2007, Med Hypotheses), ADHD and Rett syndrome hypothesis	Mechanism-driven hypothesis paper	—	—	Proposed semax as a candidate ADHD and Rett syndrome therapeutic on the basis of its BDNF-axis and cholinergic effects; not a controlled clinical trial [tsai2007]

Semax Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Semax is administered intranasally in the Russian-Federation approved product. Shevchenko et al. (2006) characterized intranasal-route kinetics in rats using tritium-labeled peptide, demonstrating measurable CNS exposure after intranasal administration [shevchenko2006]. The C-terminal Pro-Gly-Pro tripeptide is a critical PK feature: it confers proteolytic stability relative to native ACTH(4-10), which is degraded within minutes in plasma. Detailed human PK parameters (Cmax, AUC, t1/2, brain-to-plasma ratio) have not been published in English-language peer-reviewed sources at the level required for FDA labeling.

Semax is cleared by proteolytic catabolism. CYP-mediated metabolism is not expected to be a relevant pathway. No formal renal- or hepatic-impairment PK studies have been published. The Pro-Gly-Pro fragment is itself bioactive and may contribute to post-administration pharmacology after the parent peptide is cleaved [dmitrieva2010].

Pharmacodynamics

Principal pharmacodynamic effects characterized in preclinical and translational work are upregulation of BDNF and NGF and their receptors in hippocampus and basal forebrain, modulation of immune and vascular gene expression in post-ischemic brain, mild mu-opioid receptor activity, and possible transthyretin binding [dolotov2003; dolotov2006_brainres; dolotov2006_jneuro; medvedeva2017; filippenkov2020; grivennikov2008]. Clinical PD endpoints used in the Russian literature include neurological severity scales (NIHSS-equivalent), electrophysiological measures, and cognitive testing batteries; these endpoints have not been bridged to FDA-recognized endpoint instruments through dedicated comparative studies [agapova2007; shadrina2010; medvedeva2014; dergunova2021; filippenkov2024; vyunova2016].

Semax Storage and Handling

If a Semax preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records.

Semax Compounding & Operations

503A compounding

Physicians may submit patient-specific prescription requests for pharmacy review. For Semax, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for Semax is concentrated in Russian clinical and pharmacology literature, with use described for neurologic indications abroad. That record has not been converted into an FDA-approved US product or a modern US safety and dosing program.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. FDA has scheduled Semax-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For Semax, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for Semax are reviewed before any preparation is made or released. The legitimate US pathway is not to treat foreign approval or online availability as a substitute for pharmacy review. A patient-specific prescription request must be reviewed against US compounding requirements and the actual evidence record.

Pharmacist review

For Semax, the pharmacist review starts before any preparation is made. Valid patient-specific prescription required. Supporting clinical rationale may be requested. The pharmacist reviews ingredient status,

sourcing, formulation feasibility, state requirements, patient-specific documentation, and whether dispensing is appropriate case by case.

Quality and traceability

If a Semax preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records.

Frequently Asked Questions About Semax

Can physicians request Semax through RonanRx?

Physicians may submit patient-specific prescription requests for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case. Compounded medications are not FDA-approved, and no consumer self-ordering, office stock, or bulk dispensing is offered.

Why is semax approved in Russia but not in the United States?

Russian Federation approval of Semax-MV intranasal drops is based on a body of Russian-language clinical trials and a Russian Ministry of Health regulatory process. FDA approval requires evidence that meets U.S. statutory and regulatory standards, including controlled trials reviewed under the FDA framework [gusev2005; gusev2018]. No FDA-registration trial of semax has been reported, and FDA's current placement of semax in Category 2 under 503A bulk substance evaluation reflects the agency's assessment that the available safety or efficacy evidence is insufficient at this time [fda503a; fda_category2; gusev1997].

What does FDA Category 2 mean for 503A compounding?

FDA's 503A bulk drug substance evaluation places candidate substances into categories. Category 1 substances are acceptable for 503A compounding while FDA finalizes formal listing. Category 2 substances are not acceptable for 503A compounding because FDA has identified significant safety risk or insufficient evidence at the bulk-substance level [fda503a; fda_category2]. Semax is currently in Category 2.

What is the published evidence base for semax?

The clinical evidence base is concentrated in Russian-language journals and consists of small to moderate single-country trials in acute ischemic stroke and chronic cerebrovascular insufficiency reported between 1997 and 2018 [gusev1997; gusev2005; gusev2018]. The preclinical mechanistic literature is substantially larger and characterizes BDNF/NGF-axis activation, post-ischemic transcriptome modulation, mild mu-

opioid receptor activity, and intranasal pharmacokinetics [dolotov2003]. Western-journal coverage is limited; the principal English-language hypothesis paper is Tsai (2007, Medical Hypotheses) on ADHD and Rett syndrome [dolotov2006_jneuro; tsai2007].

Can my doctor write a prescription for compounded semax that another pharmacy will fill?

RonanRx cannot speak for other pharmacies, but under federal law no 503A compounding pharmacy can lawfully compound from a bulk substance in FDA Category 2 of the 503A bulk drug substance evaluation [fda503a; fda_category2]. Pharmacies that compound and dispense semax from bulk are doing so contrary to the FDA framework. Patients should be cautious about any source that markets compounded semax in the United States.

Is semax banned by WADA?

Semax is not specifically named on the World Anti-Doping Agency Prohibited List as of the current revision [wada2026]. Athletes should note, however, that peptides marketed for cognitive enhancement or recovery may fall under WADA category S0 (non-approved substances), substances not currently approved by any governmental regulatory health authority for human therapeutic use, which is prohibited at all times. Athletes considering semax for any reason should consult a sports physician and the WADA framework before use.

Can physicians request Semax through RonanRx?

Physicians may submit patient-specific prescription requests for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case. Compounded medications are not FDA-approved, and no consumer self-ordering, office stock, or bulk dispensing is offered.

References

1. *[gusev1997]* Gusev EI, Skvortsova VI, Miasoedov NF, Nezavibat'ko VN, Zhuravleva EIu, Vaneeva TV. *[Effectiveness of semax in acute period of hemispheric ischemic stroke (a clinical and electrophysiological study)]*. Zhurnal Nevrologii i Psikhiatrii im. S.S. Korsakova. 1997. PMID 11517472. (accessed 2026-05-11)
2. *[gusev2005]* Gusev EI, Martynov MIu, Kostenko EV, Petrova LV, Bobyreva SN. *[Semax in prevention of disease progress and development of exacerbations in patients with cerebrovascular insufficiency]*. Zhurnal Nevrologii i Psikhiatrii im. S.S. Korsakova. 2005. PMID 15792140. (accessed 2026-05-11)
3. *[gusev2018]* Gusev EI, Martynov MY, Kostenko EV, Petrova LV, Bobyreva SN. *[The efficacy of semax in the tretament of patients at different stages of ischemic stroke]*. Zhurnal Nevrologii i Psikhiatrii im. S.S. Korsakova. 2018. PMID 29798983. (accessed 2026-05-11)
4. *[dolotov2003]* Dolotov OV, Seredenina TS, Levitskaya NG, Kamensky AA, Andreeva LA, Alfeeva LY, Nagaev II, Zolotarev YA, Grivennikov IA, Engele J, Myasoedov NF. *The heptapeptide SEMAX stimulates BDNF expression in different areas of the rat brain in vivo*. Doklady Biological Sciences. 2003. PMID 14556513. (accessed 2026-05-11)

5. *[dolotov2006_jneuro]* Dolotov OV, Karpenko EA, Seredenina TS, Inozemtseva LS, Levitskaya NG, Zolotarev YA, Kamensky AA, Grivennikov IA, Engele J, Myasoedov NF. *Semax, an analogue of adrenocorticotropin (4-10), binds specifically and increases levels of brain-derived neurotrophic factor protein in rat basal forebrain*. Journal of Neurochemistry. 2006. PMID 16635254. (accessed 2026-05-11)
6. *[dolotov2006_brainres]* Dolotov OV, Karpenko EA, Inozemtseva LS, Seredenina TS, Levitskaya NG, Rozyczka J, Dubynina EV, Novosadova EV, Andreeva LA, Alfeeva LY, Kamensky AA, Grivennikov IA, Myasoedov NF, Engele J. *Semax, an analog of ACTH(4-10) with cognitive effects, regulates BDNF and trkB expression in the rat hippocampus*. Brain Research. 2006. PMID 16996037. (accessed 2026-05-11)
7. *[agapova2007]* Agapova TY, Agniullin YV, Shadrina MI, Shram SI, Slominsky PA, Lymborska SA, Myasoedov NF. *Neurotrophin gene expression in rat brain under the action of Semax, an analogue of ACTH 4-10*. Neuroscience Letters. 2007. PMID 17353092. (accessed 2026-05-11)
8. *[shadrina2010]* Shadrina M, Kolomin T, Agapova T, Agniullin Y, Shram S, Slominsky P, Lymborska S, Myasoedov N. *Comparison of the temporary dynamics of NGF and BDNF gene expression in rat hippocampus, frontal cortex, and retina under Semax action*. Journal of Molecular Neuroscience. 2010. PMID 19662538. (accessed 2026-05-11)
9. *[dmitrieva2010]* Dmitrieva VG, Povarova OV, Skvortsova VI, Limborska SA, Myasoedov NF, Dergunova LV. *Semax and Pro-Gly-Pro activate the transcription of neurotrophins and their receptor genes after cerebral ischemia*. Cellular and Molecular Neurobiology. 2010. PMID 19633950. (accessed 2026-05-11)
10. *[medvedeva2014]* Medvedeva EV, Dmitrieva VG, Povarova OV, Limborska SA, Skvortsova VI, Myasoedov NF, Dergunova LV. *The peptide semax affects the expression of genes related to the immune and vascular systems in rat brain focal ischemia: genome-wide transcriptional analysis*. BMC Genomics. 2014. PMID 24661604. (accessed 2026-05-11)
11. *[medvedeva2017]* Medvedeva EV, Dmitrieva VG, Limborska SA, Myasoedov NF, Dergunova LV. *Semax, an analog of ACTH((4-7)), regulates expression of immune response genes during ischemic brain injury in rats*. Molecular Genetics and Genomics. 2017. PMID 28255762. (accessed 2026-05-11)
12. *[dergunova2021]* Dergunova LV, Filippenkov IB, Limborska SA, Myasoedov NF. *[The Peptide Drug ACTH(4-7)PGP (Semax) Suppresses mRNA Transcripts Encoding Proinflammatory Mediators Induced by Reversible Ischemia of the Rat Brain]*. Molekuliarnaia Biologiia (Moscow). 2021. PMID 34097675. (accessed 2026-05-11)
13. *[filippenkov2020]* Filippenkov IB, Stavchansky VV, Denisova AE, Yuzhakov VV, Sevan'kaeva LE, Sudarkina OY, Dmitrieva VG, Gubsky LV, Myasoedov NF, Limborska SA, Dergunova LV. *Novel Insights into the Protective Properties of ACTH((4-7))PGP (Semax) Peptide at the Transcriptome Level Following Cerebral Ischaemia-Reperfusion in Rats*. Genes (Basel). 2020. PMID 32580520. (accessed 2026-05-11)
14. *[filippenkov2024]* Filippenkov IB, Remizova JA, Denisova AE, Stavchansky VV, Golovina KD, Gubsky LV, Andreeva LA, Myasoedov NF, Limborska SA, Dergunova LV. *ACTH-like Peptides Compensate Rat Brain Gene Expression Profile Disrupted by Ischemia a Day After Experimental Stroke*. Biomedicines. 2024. PMID 39767736. (accessed 2026-05-11)
15. *[shevchenko2006]* Shevchenko KV, Nagaev IY, Alfeeva LY, Andreeva LA, Kamenskii AA, Levitskaya NG, Shevchenko VP, Grivennikov IA, Myasoedov NF. *[Kinetics of Semax penetration into the brain and blood of rats after its intranasal administration]*. Bioorganicheskaia Khimiia. 2006. PMID 16523722. (accessed 2026-05-11)
16. *[grivennikov2008]* Grivennikov IA, Dolotov OV, Zolotarev YA, Andreeva LA, Myasoedov NF, Leacher L, Black IB, Dreyfus CF. *Effects of behaviorally active ACTH (4-10) analogue - Semax on rat basal forebrain cholinergic neurons*. Restorative Neurology and Neuroscience. 2008. PMID 18431004. (accessed 2026-05-11)
17. *[maslova2003]* Maslova MV, Maklakova AS, Sokolova NA, Ashmarin IP, Goncharenko EN, Krushinskaya YV. *The effects of ante- and postnatal hypoxia on the central nervous system and their correction with peptide hormones*. Neuroscience and Behavioral Physiology. 2003. PMID 14552554. (accessed 2026-05-11)

18. *[tsai2007]* Tsai SJ. *Semax, an analogue of adrenocorticotropin (4-10), is a potential agent for the treatment of attention-deficit hyperactivity disorder and Rett syndrome*. Medical Hypotheses. 2007. PMID 16996699. (accessed 2026-05-11)
19. *[sheremet2004]* Sheremet NL, Polunin GS, Ovchinnikova AV, Andreeva LA, Myasoedov NF, Kamenskii AA. *[An experimental substantiation for using the "Semax" neuroprotector in the treatment of optic-nerve diseases]*. Vestnik Oftalmologii. 2004. PMID 15678666. (accessed 2026-05-11)
20. *[vyunova2016]* Vyunova TV, Andreeva LA, Shevchenko KV, Myasoedov NF. *[POSSIBLE ROLE OF TRANSTHYRETIN IN THE BIOLOGICAL MECHANISM OF THE REGULATORY PEPTIDE NEUROPROTECTION.]*. Molecular Genetics, Microbiology and Virology. 2016. PMID 30383932. (accessed 2026-05-11)
21. *[fda503a]* U.S. Food and Drug Administration. *Compounding Laws and Policies — Section 503A of the Federal Food, Drug, and Cosmetic Act*. FDA Drug Compounding. 2024. https://www.fda.gov/drugs/human-drug-compounding/compounding-laws-and-policies (accessed 2026-05-11)
22. *[fda_category2]* U.S. Food and Drug Administration. *Bulk Drug Substances Used in Compounding Under Section 503A of the FD&C Act — Category 1 and Category 2 Lists*. FDA Drug Compounding — 503A Bulk Drug Substance Evaluation. 2024. https://www.fda.gov/drugs/human-drug-compounding/bulk-drug-substances-used-compounding-under-section-503a-fdc-act (accessed 2026-05-11)
23. *[wada2026]* World Anti-Doping Agency. *World Anti-Doping Agency Prohibited List*. WADA Code Annex. 2026. https://www.wada-ama.org/en/prohibited-list (accessed 2026-05-11)

How to Access Semax

Compounded Semax is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed Semax, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)