

Semax — Research Summary

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1. What It Is

Semax is a synthetic heptapeptide (Met-Glu-His-Phe-Pro-Gly-Pro) derived from the ACTH(4-10) fragment of adrenocorticotrophic hormone, with the Pro-Gly-Pro tail added for enzymatic stability. Developed at the Institute of Molecular Genetics (Russian Academy of Sciences), it has been approved in Russia since the 1990s/2011 (sources vary on exact year) as a 0.1% nasal spray for ischemic stroke, cognitive disorders, and optic nerve diseases, requiring a prescription there. It is not FDA- or EMA-approved.

Mechanism: Activates melanocortin receptors (particularly MC4R) without the HPA-axis/glucocorticoid activation of full ACTH, and robustly upregulates BDNF and NGF expression in hippocampus and cortex — the mechanistic through-line most connected to its studied cognitive and neuroprotective effects.

2. Reported Effects in Research Literature

Semax shares the same evidence-base limitation as Selank: extensive Russian preclinical and clinical research, with limited independent Western replication.

- Clinical stroke-rehabilitation trial: A 2018 non-randomized, open-label study (Gusev, Martynov, et al., Zhurnal Nevrologii i Psikiatrii) in 110 post-stroke patients (43 men, 67 women, mean age 58.0±9.7) found intranasal Semax (6,000 mcg/day, two 10-day courses 20 days apart) elevated plasma BDNF and improved Barthel index and MRC motor-scale scores over ~5 months in both early- and late-rehabilitation subgroups. Effect sizes, confidence intervals, and p-values were not reported in the published abstract, and the absence of randomization/placebo control limits causal inference.
- Preclinical BDNF/NGF data: Cell-culture work reported increased BDNF and NGF mRNA following Semax exposure; separate rodent studies measured increased hippocampal BDNF protein and a 3-fold increase in BDNF mRNA (exon III) with a 2-fold increase in trkB mRNA after a single dose.
- Ischemic stroke models (animal): Semax reduced infarct volume, improved neurological recovery, and modulated inflammation- and cell-survival-related gene expression in rodent cerebral ischemia models.
- Foundational development summary: A 15-year development review (PMID 9173745) documents the compound's pharmacology history; historical human authorization in Russia does not establish approval elsewhere or validate current research-labeled supplier products.
- Research limitations: Small clinical studies have examined ischemic stroke endpoints, but independent international replication is limited. Cognitive-enhancement claims in healthy (non-stroke) populations are not established by the existing BDNF/NGF preclinical data, which does not by itself demonstrate cognitive improvement in humans.

3. Clinical & Preclinical Study References

Gusev EI, Martynov MY, et al. Semax stroke rehabilitation study (110 patients). Zh Nevrol Psikhiatr Im S S Korsakova. 2018.

Dolotov OV, et al. Semax and hippocampal BDNF/trkB mRNA changes in rats. PMID: 16996037

Eremin KO, et al. PMID: 16362768

Ashmarin IP, et al. 15-year Semax development summary. PMID: 9173745

Cell-culture BDNF/NGF mRNA study. PMID: 11457573

Rodent hippocampal BDNF/neurotrophin expression study. PMID: 19662538

Regulatory/Safety Note: Semax is approved in Russia for stroke recovery and cognitive/optic-nerve indications, but is not approved by the FDA or EMA. Historical Russian regulatory authorization does not establish safety or efficacy standards recognized in the United States, and does not validate research-labeled material sold outside that approved Russian pharmaceutical product.

This compound has not been evaluated by the FDA and is not intended to diagnose, treat, cure, or prevent any disease in humans or animals.
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