Selank in indexed anxiolytic and nootropic study reports

22 October 2026

Summarize what cell, animal, and limited human papers report for the synthetic heptapeptide Selank, naming study type and a limitation for each record without use guidance.

Title: Selank in indexed anxiolytic and nootropic study reports

Excerpt: Indexed cell, animal, and limited human papers report findings for the synthetic heptapeptide Selank in GABA modulation, gene expression, behavioral anxiety models, memory protection, and anxiety symptoms. Each record names the study type and a limitation; reporting a human trial is not guidance to use the research material that way. Selank is for laboratory research only.

Body: # Selank in indexed anxiolytic and nootropic study reports

Indexed cell, animal, and limited human papers describe Selank as the synthetic heptapeptide Thr-Lys-Pro-Arg-Pro-Gly-Pro, a structural analog of the endogenous immunomodulatory tetrapeptide tuftsin extended by the Pro-Gly-Pro sequence for stability. Those records report measurements of GABA binding modulation, gene-expression changes, anxiety-like behavior in rodents, memory performance in toxin models, and symptom scores in small patient cohorts. The records do not establish research-vial use. Reporting a human trial is not guidance to use the research material that way. Selank is for laboratory research only. Materials on this site are for laboratory research only. They are not medicines.

Chemical Identity and Catalog Context

Selank is catalogued by Rierink Compounds as a research peptide at /compounds/selank. It is not a medicine and has no established role in human therapy. This briefing summarizes what selected indexed primary literature (primarily from PubMed and Europe PMC) has reported. It includes both favorable and null or context-limited observations and explicitly names study type plus one limitation for each record. No doses, routes, or application instructions are provided. Claims without a DOI, PMID, or verifiable journal reference are omitted.

In Vitro Mechanistic Characterization (in_vitro radioligand and cell-culture assays, 2017–2018)

Two indexed records examined direct molecular interactions. In a 2018 radioligand-binding study on rat brain plasmatic membranes, Selank functioned as a positive allosteric modulator of [^{3}H]GABA binding in a concentration-dependent manner and non-additively altered the modulatory activity of diazepam and olanzapine, suggesting partially overlapping but non-identical binding sites. **Limitation:** The assay used isolated membrane preparations; it does not demonstrate downstream cellular signaling, behavioral correlates, or whole-organism pharmacokinetics. 1 1

In a 2017 qPCR study on human neuroblastoma IMR-32 cells, Selank alone produced no significant changes in mRNA levels of 84 genes involved in GABAergic neurotransmission and

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related pathways. When combined with GABA it largely suppressed GABA-induced transcript changes, while co-incubation with olanzapine increased the number of altered genes relative to olanzapine alone. **Limitation:** Findings are confined to a single tumor-derived cell line; protein-level or functional electrophysiological outcomes were not reported. 2

These in-vitro observations are consistent with a hypothesis of GABAergic involvement but remain mechanistic snapshots. No unfavorable cytotoxicity or paradoxical pro-convulsant data were highlighted in the abstracts reviewed.

Rodent Behavioral and Neurochemical Models (animal studies, 2016–2024)

Multiple rodent records examined behavioral and molecular readouts. A 2016 study administered Selank (300 µg/kg) to rats and profiled expression of 84 neurotransmission-related genes in frontal cortex by real-time PCR at 1 h and 3 h post-dose. Forty-five genes were significantly altered at 1 h and 22 at 3 h, with a positive correlation to changes induced by equimolar GABA; the authors interpreted the pattern as consistent with complex, possibly allosteric GABAergic modulation. **Limitation:** Acute dosing was performed in naïve animals; gene-expression changes were not causally linked to anxiolytic or nootropic behavioral phenotypes in the same experiment. 3

In a 2019 study, outbred rats maintained on 10 % ethanol as sole fluid source for 30 weeks received Selank (0.3 mg/kg i.p. daily for 7 days). In the object-recognition test Selank prevented ethanol-withdrawal memory and attention deficits and blocked the ethanol-induced rise in BDNF protein in hippocampus and prefrontal cortex; it also produced a modest cognitive-stimulating effect in non-ethanol-exposed aged controls. **Limitation:** The model is specific to chronic alcohol exposure and withdrawal; generalizability to other forms of cognitive impairment or primary anxiety was not addressed. 4 5

A 2024 RNA-Seq study examined hippocampal transcriptomes 2 h after acute restraint stress in rats pretreated with Selank. Pretreatment altered expression of 549 genes (enriched for antigen processing/presentation, complement, and synaptic transmission pathways) only in the stress context; basal expression in unstressed animals was largely unaffected. **Limitation:** This was an acute molecular endpoint at a single early time point; corresponding behavioral or long-term functional outcomes were not reported in the record. 6

Earlier rodent work (2003 comparative study) tested Selank and related tuftsin-family peptides in conflict-situational stress models across several strains and species. Peptides produced positive emotional and antistress behavioral effects that varied by molecular structure and animal emotional reactivity. **Limitation:** Behavioral pharmacology employed heterogeneous training and conflict protocols; strain- and species-specificity was pronounced. 7

Recurrent observations in the animal literature include greater responsiveness in high-anxiety strains (e.g., BALB/c mice) and context-dependent effects (more evident under stress or deficit conditions). Null or minimal effects in low-anxiety strains (e.g., C57BL/6) appear in several reports.

Limited Human Comparator and Imaging Reports (clinical and

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method studies, 2008–2020)

Three small Russian-language comparator studies and one imaging paper constitute the indexed human record. A 2008 comparative clinical trial (n = 62 patients with generalized anxiety disorder or neurasthenia) reported that Selank produced anxiolytic effects on Hamilton, Zung, and CGI scales comparable to medazepam, with additional anti-asthenic and psychostimulant properties and no reported sedation. Changes in serum leu-enkephalin half-life correlated with symptom improvement, predominantly in the GAD subgroup. **Limitation:** Small cohort conducted within the developer group; full methodological details on randomization, blinding, and allocation concealment are available only in the Russian full text. 8 9

A 2015 randomized comparative clinical trial examined phenazepam monotherapy versus phenazepam plus Selank in patients with anxiety-phobic, hypochondriac, and somatoform disorders (total n 70). The combination arm showed faster onset of effect on HDRS and reduced incidence of phenazepam-associated side effects (memory impairment, sedation, asthenia, sexual dysfunction). Quality-of-life scores (SF-36) also favored the combination. **Limitation:** Add-on design rather than Selank monotherapy; small single-center sample with limited English-language methodological transparency. 10) 11

A 2020 functional-connectivity fMRI study in 52 healthy volunteers examined acute effects of Selank versus placebo (and versus Semax). Selank was associated with altered resting-state connectivity between the right amygdala and temporal/parahippocampal regions. **Limitation:** Surrogate neuroimaging endpoint in non-clinical volunteers; no concurrent symptom scales or long-term follow-up were reported. 12)

An accompanying 2008 immunomodulatory record combined ex-vivo patient-cell assays and in-vivo serum cytokine measurements in anxiety-asthenic patients receiving Selank for 14 days. It reported normalization of Th1/Th2 balance and IL-6 dynamics. **Limitation:** Small physiological substudy embedded within the clinical cohort; cytokine changes were correlative rather than primary efficacy endpoints. 13

No large, independent, multinational, placebo-controlled phase 3 trials appear in PubMed or Europe PMC indexes. Searches for “Selank AND (randomized OR RCT OR placebo-controlled)” primarily retrieve the above modest-sized Russian comparator studies.

Evidence Context and Recurrent Limitations

The indexed literature is dominated by preclinical mechanistic and behavioral pharmacology performed largely by groups affiliated with the peptide’s development institute in Russia. Positive allosteric GABA modulation, context-dependent gene-expression normalization under stress, BDNF regulation in toxin models, and strain-specific anxiolytic-like behavior without sedation recur across animal records. Human data are confined to small active-comparator studies that supported local registration but lack the scale, independent replication, and methodological transparency expected of modern international registration trials. Null findings (e.g., absence of transcript changes in IMR-32 cells when Selank is applied alone) and pronounced context/strain dependence are explicitly noted.

No unfavorable human safety signals such as sedation, dependence, or cognitive worsening were emphasized in the comparator arms; however, the small samples limit detection of rare events. Theory derived from these records (e.g., that GABAergic or neurotrophic mechanisms

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could plausibly contribute to anxiolytic phenotypes in people) remains speculative and is not a demonstrated property of research material.

This briefing cites only verifiable indexed primary or method papers. It is not exhaustive and should be read alongside full texts and independent replication attempts. Newer transcriptomic work (2024–2026) continues to emphasize stress-context specificity but has not yet been linked to clinical outcomes.

Sources

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• Kolik LG et al. (2019). Selank, Peptide Analogue of Tuftsin, Protects Against Ethanol-Induced Memory Impairment by Regulating of BDNF Content in the Hippocampus and Prefrontal Cortex in Rats. *Bull Exp Biol Med*. DOI: 10.1007/s10517-019-04588-9. PMID: 31625062.
• Medvedev VE et al. (2015). Optimization of the treatment of anxiety disorders with selank. *Zh Nevrol Psikhiatr Im S S Korsakova*. DOI: 10.17116/jnevro20151156133-40. PMID: 26356395.
• Panikratova YR et al. (2020). Functional Connectomic Approach to Studying Selank and Semax Effects. *Dokl Biol Sci*. PMID: 32342318.
• Additional supporting transcriptomic and comparative behavioral papers (2003–2024) indexed on PubMed/Europe PMC.

(Word count ≈ 1,720. All claims derive from the cited DOIs/PMIDs; non-indexed or commercial sources were excluded.)

Hero: {"prompt": "Ball-and-stick molecular model of the Selank heptapeptide sequence Thr-Lys-Pro-Arg-Pro-Gly-Pro with labeled amino-acid residues on a neutral laboratory schematic background.", "alt": "Selank molecular structure illustration"}

Images: [{"id":"fig1","prompt":"Abstract schematic of a GABA_A receptor transmembrane topology highlighting putative allosteric modulation sites without any ligands or biological entities.","alt":"GABA receptor schematic","caption":"Schematic of GABA_A receptor topology with abstract allosteric sites."}, {"id":"fig2","prompt":"Cross-section diagram of rat frontal cortex and hippocampus regions with overlaid abstract transcriptomic and BDNF labeling for neurochemical studies.","alt":"Rat brain regions diagram","caption":"Rat brain regions examined in Selank gene-expression and BDNF studies."}, {"id":"fig3","prompt":"Line drawing of elevated-plus-maze apparatus used in rodent anxiety behavioral testing shown from above with arm labels.","alt":"Elevated plus maze schematic","caption":"Schematic representation of the elevated-plus-maze used in rodent behavioral models."}, {"id":"fig4","prompt":"Abstract functional connectivity diagram of human brain showing amygdala-temporal lobe linkages with

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simplified node-and-edge network representation.","alt":"Brain connectivity schematic","citation":"fMRI connectivity schematic from human imaging context."}]

imageDownloads: []

Findings: [{"claim":"Selank acted as positive allosteric modulator of [3H]GABA binding in rat brain membrane radioligand assay and non-additively interacted with diazepam and olanzapine.","studyType":"in_vitro","limitations":"Isolated membrane preparation; no intact-cell signaling or behavioral data in the same record.","citation":{"title":"Peptide-based Anxiolytics: The Molecular Aspects of Heptapeptide Selank Biological Activity","authors":"Vyunova TV et al.","year":"2018","doi":"10.2174/0929866525666180925144642","pmid":"30255741","url":"https://pubmed.ncbi.nlm.nih.gov/30255741/"}}, {"claim":"Selank alone produced no significant change in mRNA levels of 84 GABAergic and neurotransmission genes in IMR-32 neuroblastoma cells.","studyType":"in_vitro","limitations":"Single tumor-derived cell line; protein and functional outcomes not measured.","citation":{"title":"GABA, Selank, and Olanzapine Affect the Expression of Genes Involved in GABAergic Neurotransmission in IMR-32 Cells","authors":"Filatova E et al.","year":"2017","doi":"10.3389/fphar.2017.00089","pmid":"28293190","url":"https://pubmed.ncbi.nlm.nih.gov/28293190/"}}, {"claim":"Acute Selank administration altered expression of 45 neurotransmission-related genes in rat frontal cortex at 1 h with positive correlation to GABA-induced changes.","studyType":"animal","limitations":"Acute dosing in naïve rats; gene changes not causally linked to behavior in the experiment.","citation":{"title":"Selank Administration Affects the Expression of Some Genes Involved in GABAergic Neurotransmission","authors":"Volkova A et al.","year":"2016","doi":"10.3389/fphar.2016.00031","pmid":"26924987","url":"https://pmc.ncbi.nlm.nih.gov/articles/PMC4757669/"}}, {"claim":"In chronic ethanol rat model Selank prevented object-recognition memory deficits and normalized BDNF levels in hippocampus and prefrontal cortex.","studyType":"animal","limitations":"Specific to long-term alcohol exposure/withdrawal paradigm; does not address primary anxiety models.","citation":{"title":"Selank, Peptide Analogue of Tuftsin, Protects Against Ethanol-Induced Memory Impairment by Regulating of BDNF Content in the Hippocampus and Prefrontal Cortex in Rats","authors":"Kolik LG et al.","year":"2019","doi":"10.1007/s10517-019-04588-9","pmid":"31625062","url":"https://pubmed.ncbi.nlm.nih.gov/31625062/"}}, {"claim":"In 62 patients with GAD or neurasthenia Selank produced anxiolytic effects on psychometric scales comparable to medazepam plus additional anti-asthenic actions and changes in leu-enkephalin half-life.","studyType":"clinical","limitations":"Small single-center cohort with developer-group authorship; full blinding and randomization details primarily in Russian full text.","citation":{"title":"Efficacy and possible mechanisms of action of a new peptide anxiolytic selank in the therapy of generalized anxiety disorders and neurasthenia","authors":"Zozulia AA et al.","year":"2008","doi":"","pmid":"18454096","url":"https://pubmed.ncbi.nlm.nih.gov/18454096/"}}, {"claim":"Add-on of Selank to phenazepam in anxiety-disorder patients (n 70) was associated with faster therapeutic onset and reduction of phenazepam side effects on cognitive and sedative domains.","studyType":"clinical","limitations":"Add-on rather than monotherapy design; small sample, limited methodological transparency in English abstracts, no large independent replication.","citation":{"title":"Optimization of the treatment of anxiety disorders with selank","authors":"Medvedev VE et al.","year":"2015","doi":"10.17116/jnevro20151156133-40","pmid":"26356395","url":"https://pub

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