BIOMOD LABS · RESEARCH PEPTIDE CO.

RESEARCH COMPOUND MONOGRAPH · SERIES 1 · NO. SPR-005

Selank (Nasal Spray Preparation)

A single-component aqueous nasal spray preparation of the Russian Academy of Sciences tuftsin-Pro-Gly-Pro heptapeptide Selank — formulated for intranasal mucosal delivery via a 200-actuation metered-dose bottle at 0.25 mg active per spray.

CATALOG REFERENCE	FORM FACTOR	STRENGTH	DATE OF ISSUE
BM-SPR-005	Nasal spray · 50 mg / bottle · 200 sprays · 0.25 mg per spray	50 mg active per bottle	May 2026

Selank is a synthetic heptapeptide of sequence Thr-Lys-Pro-Arg-Pro-Gly-Pro (TKPRPGP), developed at the Institute of Molecular Genetics of the Russian Academy of Sciences under N. F. Myasoedov as a metabolically stabilised analogue of the endogenous immunomodulatory tetrapeptide tuftsin (TKPR). The native tuftsin sequence — produced by enzymatic cleavage of the IgG immunoglobulin heavy chain — is rapidly degraded by peptidases. Appending a C-terminal Pro-Gly-Pro tail (a "glyproline" motif) substantially extended metabolic stability and yielded a compound investigated in rodent models of anxiety-related behaviour, BDNF expression in hippocampus and prefrontal cortex, and GABA-system gene expression. ***This monograph summarises published preclinical findings for laboratory research reference only.***

01 Compound Profile

COMMON DESIGNATION	Selank
PRIMARY SEQUENCE	Thr-Lys-Pro-Arg-Pro-Gly-Pro
ONE-LETTER SEQUENCE	TKPRPGP
CAS REGISTRY	129954-34-3
MOLECULAR FORMULA	$C_{33}H_{57}N_{11}O_9$
AVERAGE MOLECULAR MASS	751.87 g · mol^{-1}
PARENT SEQUENCE	Tuftsin (TKPR) + C-terminal Pro-Gly-Pro stabilising tail
PHYSICAL FORM	White lyophilised solid
SOLUBILITY (LAB RECONSTITUTION)	Soluble in sterile water, bacteriostatic water, and physiological saline; intranasal preparations in published rodent work use saline-based vehicles[1]
STORAGE (RESEARCH HANDLING)	Lyophilised solid: −18 °C, desiccated; reconstituted solution refrigerated short-term per laboratory protocol
ANALYTICAL SPECIFICATION	≥ 99 % purity by HPLC (BIOMOD Labs internal release specification)

02 Origin and Chemistry

Tuftsin is a naturally occurring tetrapeptide (Thr-Lys-Pro-Arg) liberated by enzymatic cleavage of the IgG immunoglobulin heavy chain — historically characterised for immunomodulatory and phagocytosis-promoting activities in macrophage preparations. The peptide is unstable in plasma owing to rapid peptidase action at its termini. The Selank design addresses this liability by attaching a Pro-Gly-Pro "glyproline" tail to the C-terminus of the parent tuftsin sequence: proline-rich termini are notably resistant to peptidase cleavage, and the resulting heptapeptide retains the tuftsin pharmacophore while exhibiting markedly extended metabolic stability in published reports.[2]

The terminal PGP motif is itself a known endogenous bioactive sequence with documented intrinsic activity, and the construct combines tuftsin-derived activity with PGP-related effects. Selank does not appear to bind classical GABA-A benzodiazepine sites in radioligand displacement studies; the proposed mechanism instead invokes modulation of GABA-A subunit gene expression and engagement of upstream neurotrophic and enkephalinase-related pathways.[3] IN VITRO

03 Proposed Mechanisms in Preclinical Models

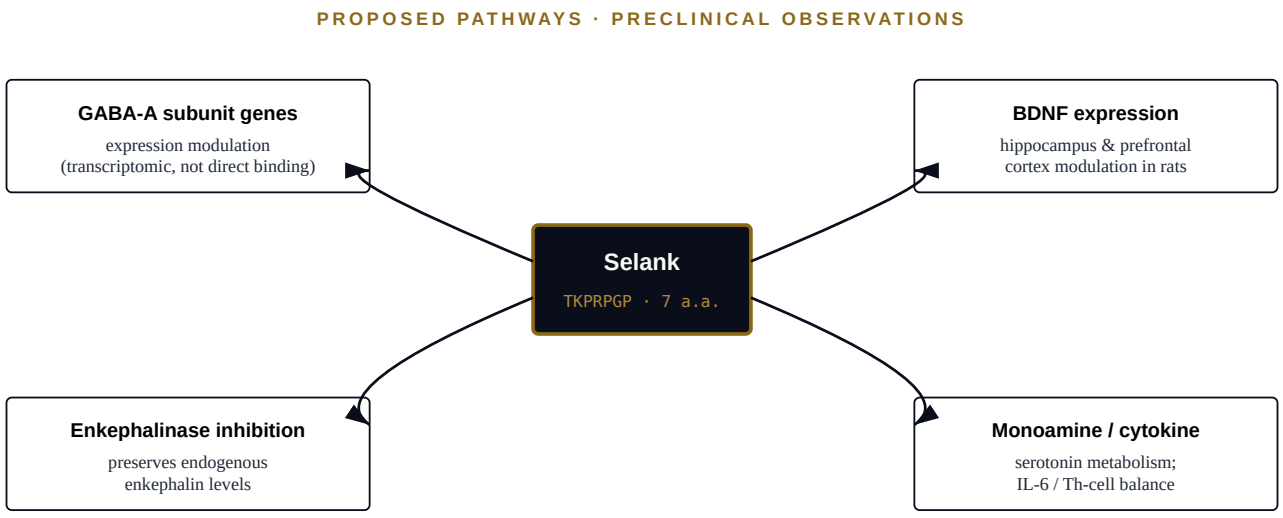

FIGURE 1 Schematic of proposed pathways reported in preclinical animal investigations of Selank. Compiled from refs 1, 3, 4, 5.

3.1 BDNF EXPRESSION IN HIPPOCAMPUS AND PREFRONTAL CORTEX

Inozemtseva and colleagues, in a 2008 study published in *Doklady Biological Sciences*, reported that intranasal Selank administration in rats modulated brain-derived neurotrophic factor (BDNF) expression in the hippocampus.[1] A 2019 study by Kolik, Nadorova, Antipova and colleagues extended this work, examining outbred rats receiving chronic ethanol exposure and reporting that Selank administration over a 7-day window during alcohol withdrawal prevented ethanol-induced disturbances in object recognition memory and prevented ethanol-associated changes in BDNF content in both hippocampus and prefrontal cortex.[4] PRECLINICAL · RAT

3.2 GABA-A RECEPTOR SUBUNIT GENE EXPRESSION

Kasian, Kolomin, Andreeva and colleagues, in a 2017 study published in *Frontiers in Pharmacology*, examined Selank administration in rats and reported changes in expression of multiple GABAergic-transmission genes in brain tissue, providing a transcriptomic basis for the anxiolytic-like profile reported in earlier behavioural work — distinct from the direct receptor occupancy that characterises benzodiazepines.[5]

3.3 IMMUNOMODULATORY ACTIVITY (TUFTSIN PHARMACOPHORE)

Consistent with its origin as a tuftsin analogue, Selank has been examined for effects on immune-cell preparations. Published work reports modulation of interleukin-6 (IL-6) expression and shifts in T-helper cell cytokine balance in cell-culture and rodent preparations exposed to the peptide — observations that link Selank's mechanism to its parent tuftsin lineage while expanding the pharmacological profile relative to the unmodified tetrapeptide.[6]

3.4 ENKEPHALIN SYSTEM INTERACTION

A line of work from the originating laboratory reports that Selank inhibits the activity of enkephalin-degrading peptidases in tissue homogenate preparations, with consequent elevation of intact enkephalin signal — a mechanism that has been invoked to explain the compound's reported anxiolytic-like effects in rodent behavioural paradigms without engagement of classical opioid receptor binding.[7] IN VITRO

04 Preclinical Findings Summary

SYSTEM	ANIMAL MODEL / PREPARATION	REPORTED OBSERVATION	REF.
BDNF expression	Intranasal Selank in rats, hippocampal analysis	Modulation of hippocampal BDNF expression	1
Memory under ethanol withdrawal	Outbred rats, object recognition	Prevention of withdrawal-associated BDNF changes; preserved recognition memory	4
GABA-A gene expression	Rat brain tissue, transcriptomic analysis	Modulation of multiple GABAergic transcripts	5
Immunomodulation	Cell-culture & rodent preparations	↑ IL-6 modulation; T-helper cytokine balance shifts	6
Enkephalin system	Tissue homogenate peptidase assays	Inhibition of enkephalin-degrading peptidases	7
Monoamine metabolism	Rat brain regional analysis	Serotonin metabolism modulation in defined brain regions	8

05 Research Synthesis & Limitations

METHODOLOGICAL NOTES

The Selank corpus is concentrated in Russian-language and Russian-laboratory publications, predominantly from the Institute of Molecular Genetics and affiliated groups. Independent peer-reviewed replication outside this network is sparser than for compounds in broader international circulation. Mechanistic claims around GABA-A subunit gene expression, BDNF modulation, and enkephalinase inhibition are supported by published data but would benefit from independent verification in additional model systems. The compound's pharmacophore origin in tuftsin (an immunomodulatory peptide) and its surprisingly broad neurochemical profile have attracted growing interest, and researchers designing studies should be aware of both the strengths and limitations of the existing evidence base.

06 Laboratory Handling, Reconstitution, and Storage

Selank is supplied as a lyophilised powder under research-use specifications. Published rodent methodology uses sterile saline or sterile water for injection as reconstitution vehicles; the peptide is highly water-soluble. The C-terminal proline-rich tail confers reasonable stability in aqueous solution at neutral pH, but lyophilised solid storage at −18 °C desiccated is the standard practice in cited literature, with reconstituted aliquots held at −18 °C for extended storage and freeze–thaw cycles minimised. Working concentrations are determined by the investigator's experimental design.

07 References

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F Formulation and Delivery Specifications

ACTIVE COMPOUND	Selank · CAS 129954-34-3
TOTAL MASS PER BOTTLE	50 mg active
SPRAYS PER BOTTLE	200 sprays
ACTIVE PER SPRAY	0.25 mg per spray
VOLUME PER SPRAY	0.1 mL per metered actuation
VEHICLE	Aqueous nasal-grade vehicle with preservative
ANALYTICAL SPECIFICATION	≥ 98 % purity by HPLC; content uniformity per actuation verified to ± 10 % of label claim

04 Nasal Delivery Considerations

INTRANASAL BIOAVAILABILITY AND VEHICLE CHEMISTRY

The intranasal route provides direct access to systemic circulation through the rich vascularisation of the nasal mucosa, bypassing first-pass hepatic metabolism characteristic of oral administration. For peptide-class compounds, the principal nasal-delivery considerations are (a) **mucosal residence time** — aqueous nasal vehicles produce relatively short mucosal contact, with peptide permeation governed by molecular size, lipophilicity, and chemistry of the active; (b) **vehicle pH** — neutral-to-slightly-acidic pH (5.5–7.0) is optimal for both nasal mucosa tolerance and peptide bond stability; (c) **osmolarity** — formulation osmolarity is targeted to approximate physiological isotonicity to minimise mucociliary disruption; (d) **preservative selection** — benzalkonium chloride or similar quaternary ammonium preservatives are standard for nasal aqueous formulations; (e) **permeation enhancers** may be incorporated in some formulations to support peptide passage across the nasal epithelium without disrupting mucosal integrity.