

SEMAX — N-TERMINAL ACETYLATION OF MET-1 (AC-MET-GLU-HIS-PHE-PRO-GLY-PRO)

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- REFINED
- COGNITIVE
- N-TERMINAL ACETYLATION OF MET-1 (AC-MET-GLU-HIS-PHE-PRO-GLY-PRO)
- MELANOCORTIN-4 RECEPTOR (MC4R) / BDNF-TRKB SIGNALING AXIS

AVERAGE CONFIDENCE	PTM / IPTM	VERDICT
80.3%	0.851 / 0.941	REFINED
TARGET	UNIPROT	BINDING PROBABILITY
Melanocortin-4 receptor (MC4R) / BDNF-TrkB signaling axis	P32245	—

TLDR

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EXECUTIVE SUMMARY

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DETAILED ANALYSIS

no research brief recorded.

RESEARCH BRIEF

no research brief recorded.

SEQUENCES

NATIVE

MEHFPGP

MODIFIED

Ac-MEHFPGP

CITATIONS

1. **PMID** — (2025) — — Semax peptide targets the μ opioid receptor gene Oprm1 to promote deubiquitination and functional recovery after spinal cord injury in female mice.
2. **PMID** — (2021) — — Semax, synthetic ACTH(4-10) analogue, attenuates behavioural and neurochemical alterations following early-life fluvoxamine exposure in white rats.
3. **PMID** — (2026) — — Therapeutic Peptides in Orthopaedics: Applications, Challenges, and Future Directions.
4. **PMID** — (2022) — — Semax, a Synthetic Regulatory Peptide, Affects Copper-Induced Abeta Aggregation and Amyloid Formation in Artificial Membrane Models.
5. **PMID** — (2010) — — Semax and Pro-Gly-Pro activate the transcription of neurotrophins and their receptor genes after cerebral ischemia.
6. **PMID** — (2017) — — Semax, an analog of ACTH (transcriptome analysis study)
7. **PMID** — (2025) — — Semax, a Copper Chelator Peptide, Decreases the Cu(II)-Catalyzed ROS Production and Cytotoxicity of $a\beta$ by Metal Ion Stripping and Redox Silencing.
8. **PMID** — (2005) — — Semax, an ACTH(4-10) analogue with nootropic properties, activates dopaminergic and serotonergic brain systems in rodents.