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47

# 47 Peptide Research Facts

*Most People Never See*

BPC-157 | TB-500 | NAD+ | Semax | Ipamorelin | Tesamorelin | AOD9604

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ABOUT THIS DOCUMENT

# Research, not repetition.

Most peptide content online repeats the same three or four bullet points, copy-pasted from vendor to vendor. This document is different: it's 47 specific, sourced, standalone facts pulled from the primary and secondary research literature on seven of the most-studied research peptides — the kind of structural detail, discovery history, and mechanism-level findings that actually reward a second read.

This is not a dosing guide, a protocol, or a claim that any of these compounds treat, cure, or prevent anything in humans. It's a research reference: built for people who order peptides for laboratory work and want to understand what's actually been published, not just what's on the label. Every fact below traces back to a peer-reviewed study, a standard chemical reference, or a documented discovery history — cited so you can go verify it yourself.

PEPTIDES COVERED

BPC-157 TB-500 NAD+ Semax Ipamorelin Tesamorelin AOD9604

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# BPC-157

*Body Protection Compound*

Facts 01–07

**01** **BPC-157 is a 15-amino-acid synthetic peptide (Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val) derived from a partial sequence identified in a protective protein found in human gastric juice.**

**WHY IT MATTERS** Its origin in a naturally protective gastric protein is the entire rationale for studying it in gut-lining and tissue-repair research models — the sequence wasn't designed from scratch, it was found.

**02** **Its molecular formula is $C_{62}H_{98}N_{16}O_{22}$, with a molar mass of roughly 1,419.6 g/mol.**

**WHY IT MATTERS** Knowing the exact formula and mass matters for anyone verifying a Certificate of Analysis — it's the baseline chemical fingerprint a lab uses to confirm a sample is what it claims to be.

**03** **BPC-157 research traces to Croatian pharmacologist Predrag Sikić and colleagues, who studied gastric protective compounds and their downstream effects in animal models.**

**WHY IT MATTERS** Understanding a compound's research lineage — who first isolated and characterized it, and why — helps explain why the published literature focuses so heavily on gut and tissue-repair pathways rather than other systems.

**04** **In preclinical models, BPC-157 has been studied for proposed interactions with angiogenesis-related signaling, including pathways associated with VEGF (vascular endothelial growth factor).**

**WHY IT MATTERS** New blood vessel formation is a prerequisite for most tissue-repair processes studied in a lab — it's one of the reasons BPC-157 shows up across so many different injury-model studies, from gut lining to tendon.

**05** **Reported pharmacokinetic half-life in rat models ranges from roughly 7.9 to 30 minutes depending on administration route.**

**WHY IT MATTERS** A short half-life is a critical variable for researchers designing dosing-interval or infusion protocols in animal studies — it directly shapes experimental design, independent of any question about effects.

**06** **BPC-157 was added to the World Anti-Doping Agency's list of prohibited substances in 2022.**

**WHY IT MATTERS** Regulatory attention from a body like WADA is itself a data point — it reflects how far BPC-157 research interest had spread beyond its original gastric-protection focus, even without FDA approval for any human use.

**07** **Despite extensive animal-model research, human clinical trial data on BPC-157 remains limited, and it is not an FDA-approved drug.**

**WHY IT MATTERS** This is the single most important context for interpreting every other fact on this list — mechanism-level and animal-model findings are not the same thing as demonstrated human safety or efficacy.

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# TB-500

*Thymosin Beta-4 Fragment*

Facts 08–14

**08** **Full-length Thymosin Beta-4 (Tβ4) is a 43-amino-acid peptide, while the specific fragment known in research contexts as "TB-500" corresponds to a smaller synthetic sequence derived from Tβ4's active region — historically including a related N-acetylated tetrapeptide (N-Ac-Ser-Asp-Lys-Pro) studied for its own distinct activity on hematopoietic stem cell proliferation.**

**WHY IT MATTERS** The "TB-500" name is often used loosely in the research-peptide market; understanding it as a fragment/analog of a much larger, well-characterized parent protein clarifies what's actually being studied.

**09** **Tβ4 was first isolated and sequenced from calf thymus tissue in the 1960s as part of "Thymosin Fraction 5," a mixture of thymic peptides studied under Allen Goldstein's research program.**

**WHY IT MATTERS** It was originally believed to be a thymic hormone involved in immune function — its now-dominant role as an actin-regulating protein wasn't identified until later research, a good example of how a compound's understood mechanism can shift over decades of study.

**10** **The gene encoding Tβ4, TMSB4X, is located on the X chromosome.**

**WHY IT MATTERS** Chromosomal and genetic details like this are standard reference data for anyone doing molecular-biology-level research design involving the native protein rather than the synthetic peptide alone.

**11** **Tβ4's primary documented molecular function is as the major actin-sequestering protein in eukaryotic cells — it binds monomeric G-actin in a 1:1 complex, regulating the pool available for polymerization into filamentous F-actin.**

**WHY IT MATTERS** This actin-binding activity is the proposed mechanistic foundation for nearly everything else studied about Tβ4, since actin dynamics underlie how cells change shape and migrate.

**12** **Tβ4 is present at strikingly high intracellular concentrations — approximately 0.5 mM in many tissue types — making it one of the most abundant peptides in mammalian cells.**

**WHY IT MATTERS** A compound this abundant naturally is a very different research subject than a rare signaling molecule; its ubiquity is part of why researchers have studied it across such a wide range of tissue types and injury models.

**13** **Preclinical research has studied Tβ4's involvement in recruiting epicardium-derived progenitor cells in cardiac tissue models.**

**WHY IT MATTERS** Progenitor cell recruitment is a specific, mechanistically distinct research question from simple "wound healing" — it points to interest in Tβ4's role in tissue regeneration at the cellular-origin level, not just surface repair.

**14** **Published preclinical research on Tβ4 spans dermal, corneal, and cardiac injury models, among others.**

**WHY IT MATTERS** Breadth across multiple organ-injury models is one reason Tβ4/TB-500 research has attracted sustained interest — the same proposed actin-based mechanism has been tested in structurally very different tissue types.

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# NAD+

*Nicotinamide Adenine Dinucleotide*

Facts 15–21

15 **NAD+ was first identified in 1906 by biochemists Arthur Harden and William Young while studying yeast fermentation, making it one of the earliest coenzymes ever characterized.**

**WHY IT MATTERS** NAD+ research isn't a recent trend — it has a continuous scientific history stretching back well over a century, which is part of why its core mechanisms are so thoroughly documented.

16 **The molecule was purified and its basic chemical properties determined by Hans von Euler-Chelpin, work that contributed to his 1929 Nobel Prize (shared with Harden) for fermentation research.**

**WHY IT MATTERS** Nobel-recognized foundational chemistry work is part of what makes NAD+'s basic structure and behavior some of the most well-established facts in all of metabolic biochemistry.

17 **Otto Warburg's research in the 1930s established NAD+'s core redox function: accepting a hydride (a hydrogen atom plus its electrons) to become NADH, and releasing it again to drive metabolic reactions.**

**WHY IT MATTERS** This hydride-transfer cycle is the literal mechanical basis for nearly every reference to NAD+ "powering" cellular metabolism — it's not a metaphor, it's a specific, well-characterized chemical reaction studied for nearly a century.

18 **Conrad Elvehjem's 1937 research linked niacin (vitamin B3) to NAD+ by showing it cured pellagra ("black tongue" disease) in dogs, establishing niacin as a dietary precursor for NAD+ synthesis.**

**WHY IT MATTERS** This is the study that connected a nutritional deficiency disease to a specific coenzyme pathway — a landmark in understanding how diet and cellular biochemistry are mechanistically linked.

19 **Unlike most vitamins and cofactors, NAD+ is not just recycled — it is actively consumed as a substrate by enzyme families including sirtuins and PARPs, meaning cellular NAD+ pools must be continuously resynthesized.**

**WHY IT MATTERS** This consumption-based mechanism (rather than simple catalytic recycling) is a key reason NAD+ availability is studied as a dynamic, depletable resource in cell biology research, rather than a static structural molecule.

20 **Sirtuins — a family of NAD+-dependent deacetylase enzymes — were characterized around 2000, linking NAD+ levels to gene-regulatory and cellular stress-response pathways studied in longevity-adjacent research.**

**WHY IT MATTERS** The sirtuin discovery is what shifted NAD+ research from "just a metabolic cofactor" to a molecule actively studied in the context of cellular aging biology and mitochondrial regulation.

21 **Research models report that tissue NAD+ levels decline with age, a finding that has driven a substantial share of current laboratory interest in NAD+ metabolism.**

**WHY IT MATTERS** This age-related decline is the central research question motivating most contemporary NAD+ studies — understanding whether and how restoring NAD+ availability affects the sirtuin- and PARP-dependent pathways it fuels.

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# Semax

*ACTH(4-10) Analog*

Facts 22–28

**22** **Semax is a synthetic heptapeptide — Met-Glu-His-Phe-Pro-Gly-Pro — built as an analog of ACTH(4-10), a fragment of the naturally occurring hormone adrenocorticotropic hormone (ACTH).**

**WHY IT MATTERS** Knowing the exact 7-residue structure and its relationship to a specific ACTH fragment (not full ACTH) is essential context — it explains why Semax is studied for CNS activity rather than adrenal/cortisol-axis activity.

**23** **Semax was first described in the scientific literature in 1991 by Russian researchers (including Potaman, Alfeeva, and Kamensky) studying the non-hormonal central nervous system activity of ACTH fragments.**

**WHY IT MATTERS** This is a relatively well-documented discovery timeline and research lineage, distinct from many peptides whose exact first-publication history is harder to pin down.

**24** **The parent fragment, ACTH(4-10), lacks the corticotropic (adrenal-stimulating) activity of full-length ACTH but retains activity in the central nervous system — the reason researchers use ACTH-fragment analogs like Semax as tools to study CNS effects in isolation.**

**WHY IT MATTERS** This structure-activity separation is a clean example of how peptide fragments can be used in research to isolate one biological activity of a larger hormone from its other, unrelated effects.

**25** **In a frequently cited rat hippocampus study, intranasal Semax produced up to a 3-fold increase in BDNF mRNA and a 1.6-fold increase in TrkB receptor phosphorylation.**

**WHY IT MATTERS** These are unusually specific, quantified dose-response figures for a peptide's effect on a neurotrophin pathway — the kind of granular data that makes BDNF/TrkB signaling the leading proposed mechanism in Semax research.

**26** **Beyond BDNF/TrkB signaling, Semax has also been studied for activity at melanocortin receptor subtypes MC4 and MC5.**

**WHY IT MATTERS** This suggests Semax's research profile may not be limited to a single receptor pathway — multiple mechanistic threads are documented in the literature, which is part of why it remains an active area of peptide research.

**27** **Semax was added to Russia's official List of Vital & Essential Drugs in December 2011.**

**WHY IT MATTERS** This is a real regulatory milestone in one country's pharmaceutical system — important context, since Semax is not FDA-approved and is not marketed as a drug in the United States.

**28** **Semax's studied cognitive effects have been measured in rodent models using conditioned avoidance learning tasks, correlated with the degree of BDNF/TrkB pathway activation.**

**WHY IT MATTERS** Pairing a molecular-level readout (BDNF expression) with a behavioral-level readout (learning task performance) in the same study is what allows researchers to propose a mechanistic link between the two, rather than just an association.

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# Ipamorelin

*Selective GH Secretagogue*

Facts 29–34

**29** **Ipamorelin is a synthetic pentapeptide — Aib-His-D-2-Nal-D-Phe-Lys-NH2 (CAS 170851-70-4) — with an amidated C-terminus and two D-amino acids built into its structure for receptor selectivity and enzymatic stability.**

**WHY IT MATTERS** The specific inclusion of D-amino acids (mirror-image forms not typically found in natural human proteins) is a deliberate synthetic design choice aimed at resisting normal peptide-degrading enzymes.

**30** **It was developed by Novo Nordisk researchers and first formally characterized in the scientific literature in 1998.**

**WHY IT MATTERS** Ipamorelin's development within a major pharmaceutical research program, rather than an academic lab studying a naturally occurring compound, shaped its research profile toward receptor-selectivity optimization from the outset.

**31** **Ipamorelin is studied as a selective agonist at the growth hormone secretagogue receptor (GHS-R1a) — the same receptor activated by the endogenous hormone ghrelin.**

**WHY IT MATTERS** Sharing a receptor with ghrelin (the "hunger hormone") is the mechanistic basis for grouping Ipamorelin with other ghrelin-receptor-targeting research compounds, distinct from GHRH-receptor-targeting peptides like Tesamorelin.

**32** **In the original 1998 pharmacology study, doses up to 200 times higher than the GH-stimulating dose did not produce a significant increase in ACTH or cortisol release.**

**WHY IT MATTERS** This selectivity data point is the specific finding that distinguished Ipamorelin from earlier-generation growth hormone-releasing peptides (GHRPs), which often triggered broader, less-targeted hormonal responses in the same research models.

**33** **The same foundational research also found no measurable effect on prolactin, FSH, LH, or TSH levels.**

**WHY IT MATTERS** Ruling out effects on four additional pituitary hormone axes in the original characterization study is part of what established Ipamorelin's reputation in the research literature as a narrowly receptor-selective compound.

**34** **Reported elimination half-life in pharmacokinetic research is approximately 2 hours.**

**WHY IT MATTERS** Half-life data like this is a basic but essential parameter for designing sampling and dosing-interval schedules in any laboratory pharmacokinetic study.

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# Tesamorelin

*GHRH Receptor Agonist*

Facts 35–40

**35** **Tesamorelin is built on the complete 44-amino-acid sequence of human growth hormone-releasing hormone (GRF 1-44), with a trans-3-hexenoic acid group added at the N-terminus (molecular formula $C_{221}H_{366}N_{72}O_{67}S$, CAS 218949-48-5).**

**WHY IT MATTERS** Unlike many "GHRH analogs" that are shortened or heavily modified fragments, Tesamorelin retains the full native 44-residue sequence — the N-terminal addition is the only major structural change.

**36** **That N-terminal modification was specifically designed to increase resistance to cleavage by dipeptidyl peptidase-4 (DPP-4), an enzyme that rapidly degrades natural, unmodified GHRH.**

**WHY IT MATTERS** This is a clean example of rational peptide-stability engineering — a single, targeted chemical change aimed at extending a molecule's functional window in research and pharmacological use.

**37** **Tesamorelin was developed by the Canadian biopharmaceutical company Theratechnologies and first approved (under separate pharmaceutical branding, distinct from any RUO research reagent) in 2010.**

**WHY IT MATTERS** Understanding a compound's approved-drug history versus its use as an unbranded research reagent is essential context for keeping RUO research use clearly separated from any approved clinical product.

**38** **Mechanistically, Tesamorelin acts one step upstream of growth hormone itself — as a GHRH-receptor agonist on the pituitary gland — rather than supplying GH directly.**

**WHY IT MATTERS** This upstream mode of action is the reason Tesamorelin research is framed around stimulating the body's own pulsatile GH release pattern, rather than delivering a fixed external dose of GH.

**39** **Because it acts at the receptor level rather than replacing GH directly, research indicates Tesamorelin preserves the body's natural IGF-1 negative-feedback loop that normally regulates GH secretion.**

**WHY IT MATTERS** This feedback-preservation property is the specific mechanistic distinction researchers use to separate GHRH-receptor agonists from direct-GH-replacement approaches in comparative pharmacology studies.

**40** **Reported pharmacokinetics show an elimination half-life of roughly 26–38 minutes and oral bioavailability under 4%.**

**WHY IT MATTERS** A half-life this short combined with very low oral bioavailability is standard for peptide-class research compounds and explains why non-oral administration routes dominate the relevant pharmacology literature.

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# AOD9604

*hGH Lipolytic Fragment*

Facts 41–47

41 **AOD9604 is a 15-amino-acid fragment corresponding to the C-terminal region of human growth hormone (residues 177–191), stabilized with an added tyrosine residue and cyclized via a disulfide bond between two cysteines.**

**WHY IT MATTERS** This cyclized, stabilized structure is what distinguishes AOD9604 from the plain, unmodified hGH fragment it's based on — the modifications were specifically designed to improve the fragment's usability as a standalone research compound.

42 **It was discovered by Professor Frank Ng and colleagues at Monash University in Australia, then developed further by Metabolic Pharmaceuticals Ltd., which sponsored the non-clinical safety and mechanism research on the compound.**

**WHY IT MATTERS** Tracing a compound back to its originating academic lab and the company that funded its safety studies helps clarify which findings come from independent versus sponsor-funded research.

43 **The name "AOD" stands for "Anti-Obesity Drug" — a direct reference to the specific research question that drove its isolation: separating hGH's fat-metabolism activity from its growth-promoting activity.**

**WHY IT MATTERS** The name itself is a research history artifact — it tells you exactly what hypothesis the original discovery team was testing when they isolated this particular fragment.

44 **In the laboratory systems studied, AOD9604 was reported not to stimulate IGF-1 production — a key mechanistic distinction from full-length human growth hormone.**

**WHY IT MATTERS** Since IGF-1 stimulation drives much of full hGH's growth-related activity, the absence of this effect is central to why AOD9604 is studied specifically for its proposed lipid-metabolism activity rather than any growth-axis effect.

45 **AOD9604 administration in obese-rodent studies has been associated with restored expression of beta-3 adrenergic receptor (β3-AR) RNA in adipose tissue — the primary receptor subtype governing lipolysis (fat breakdown) in fat cells.**

**WHY IT MATTERS** This receptor-expression finding is the leading proposed mechanistic link between AOD9604 and the lipid-metabolism research questions it was designed to investigate.

46 **Follow-up mechanistic research using β3-AR knockout mice found that the long-term fat-reducing effect was substantially reduced without a functional β3-AR, though some acute metabolic effects persisted.**

**WHY IT MATTERS** Knockout-mouse studies like this are how researchers test whether a specific receptor is truly necessary for an observed effect — this particular finding narrowed (but didn't fully resolve) the proposed mechanism.

47 **In one 19-day oral-administration study in obese rats, treated animals showed more than a 50% reduction in body-weight gain compared to controls, without the change in insulin sensitivity observed with full-length hGH in the same research program.**

**WHY IT MATTERS** Pairing an efficacy-adjacent outcome (reduced weight gain) with a specific safety comparison (unchanged insulin sensitivity) in the same study is what makes this one of the more frequently cited AOD9604 datasets in the preclinical literature.

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# Source List

Every fact in this document is drawn from one of the references below.


1. Goldstein AL, Hannappel E, Kleinman HK. “Thymosin beta4: actin-sequestering protein moonlights to repair injured tissues.” Trends Mol Med. 2005;11(9):421-429.
2. “Progress on the Function and Application of Thymosin β4.” PMC, 2021.
3. Goldstein AL. “History of the Discovery of the Thymosins.” Ann N Y Acad Sci. 2007.
4. “Thymosin beta-4.” Wikipedia.
5. “BPC-157.” Wikipedia.
6. “History of NAD+ Research and Discovery.” AboutNAD.
7. “The Roots of NAD+ Research.” Tru Niagen.
8. “Role of NAD+ and mitochondrial sirtuins in cardiac and renal diseases.” PMC, 2015.
9. “Sirtuins and NAD+ in the Development and Treatment of Metabolic and Cardiovascular Diseases.” Circ Res. 2018.
10. Dolotov OV, et al. “Semax, an analog of ACTH(4-10) with cognitive effects, regulates BDNF and trkB expression in the rat hippocampus.” Brain Res. 2006;1117(1):54-60.
11. “Semax.” Wikipedia.
12. Raun K, et al. “Ipamorelin, the first selective growth hormone secretagogue.” Eur J Endocrinol. 1998;139(5):552-561.
13. “Ipamorelin.” Wikipedia.
14. “Tesamorelin.” Wikipedia.
15. Leung VL, Glesby MJ. “Tesamorelin in the management of HIV-associated lipodystrophy.” Curr Opin Infect Dis. 2011.
16. Ng FM, Sun J, Sharma L, Libinaka R, Jiang WJ, Gianello R. “Metabolic studies of a synthetic lipolytic domain (AOD9604) of human growth hormone.” Horm Res. 2000;53(6).
17. Heffernan M, et al. “The effects of human GH and its lipolytic fragment (AOD9604) on lipid metabolism following chronic treatment in obese mice and β3-AR knock-out mice.” Endocrinology. 2001;142(12):5182.
18. Moré MI, et al. “Safety and Metabolism of AOD9604, a Novel Nutraceutical Ingredient for Improved Metabolic Health.” J Endocrinol Metab.