BIOMOD LABS · RESEARCH PEPTIDE CO.

RESEARCH COMPOUND MONOGRAPH · SERIES 1 · NO. 010

BPC-157

Body Protection Compound — a pentadecapeptide derived from a fragment of a gastric juice protein, characterised in three decades of preclinical animal and in-vitro studies of tissue repair, angiogenesis, and neuroprotection.

CAS REGISTRY	CATALOG REFERENCE	CLASS	DATE OF ISSUE
137525-51-0	BM-LYO-010	Synthetic peptide · 15 a.a.	May 2026

BPC-157, also designated Body Protection Compound 157 or pentadecapeptide BPC 157, is a synthetic 15-residue sequence (GEPPPGKPADDAGLV) reported to correspond to a stable partial sequence of a larger gastric juice protein. Across approximately three decades of preclinical investigation, predominantly originating from research groups associated with Sikiric and colleagues at the University of Zagreb School of Medicine and replicated by additional independent groups, BPC-157 has been examined in rodent models of cutaneous wound repair, tendon and ligament injury, gastrointestinal ulceration, traumatic brain injury, vascular occlusion, and dopamine-modulated behaviour. Reported observations in these animal and in-vitro systems include accelerated granulation tissue formation, modulation of vascular endothelial growth factor signalling, activation of the focal adhesion kinase / paxillin pathway, and attenuation of injury markers across several organ systems. ***This monograph summarises published preclinical findings for laboratory research reference only.***

01 Compound Profile

COMMON DESIGNATION	BPC-157 · Body Protection Compound 157 · Pentadecapeptide BPC 157
PRIMARY SEQUENCE	Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val
ONE-LETTER SEQUENCE	GEPPPGKPADDAGLV
CAS REGISTRY	137525-51-0
MOLECULAR FORMULA	$C_{62}H_{98}N_{16}O_{22}$
AVERAGE MOLECULAR MASS	1419.55 g · mol^{-1}
SYNONYMS (DEVELOPMENT CODES)	PL 14736 · Bepecin
PHYSICAL FORM	White / off-white lyophilised solid
SOLUBILITY (LAB RECONSTITUTION)	Reported soluble in bacteriostatic or sterile water; stable across a wide pH range in published in-vitro reports[5]
STORAGE (RESEARCH HANDLING)	Lyophilised solid: 2–8 °C, light-protected, desiccated. Reconstituted solution: refrigerated, per laboratory protocol.
ANALYTICAL SPECIFICATION	≥ 99 % purity by HPLC (BIOMOD Labs internal release specification)

02 Origin and Chemistry

BPC-157 originated from work begun in the early 1990s at the University of Zagreb School of Medicine, where Sikiric and collaborators were investigating a larger native protein recovered from gastric juice, designated "body protection compound" (BPC). The 15-residue fragment now designated BPC-157 was synthesised as a stable analogue believed by its discoverers to retain the cytoprotective behaviour attributed to the parent protein.[8] Subsequent published work has appeared under several development aliases, including the code PL 14736, used during early industrial-sponsored research programmes targeting inflammatory bowel research models.[13]

Chemically, BPC-157 is notable among bioactive peptides for the unusually high frequency of proline residues — four of fifteen positions — which contributes to a constrained conformation and confers resistance to several common peptidases. Published stability work reports retention of the parent sequence under conditions that rapidly degrade many comparator peptides.[5] IN VITRO

03 Proposed Mechanisms in Preclinical Models

The published literature attributes BPC-157's reported effects in animal and cell-culture systems to a constellation of pathways rather than a single receptor target. Investigators have variously described roles in growth-factor mimicry, modulation of focal adhesion signalling, nitric-oxide-dependent vascular tone, and intersection with monoaminergic systems. The unifying observation across these proposals is that BPC-157 appears, in preclinical models, to facilitate the assembly of repair-supporting cellular machinery at sites of experimental injury. PRECLINICAL · ANIMAL MODEL

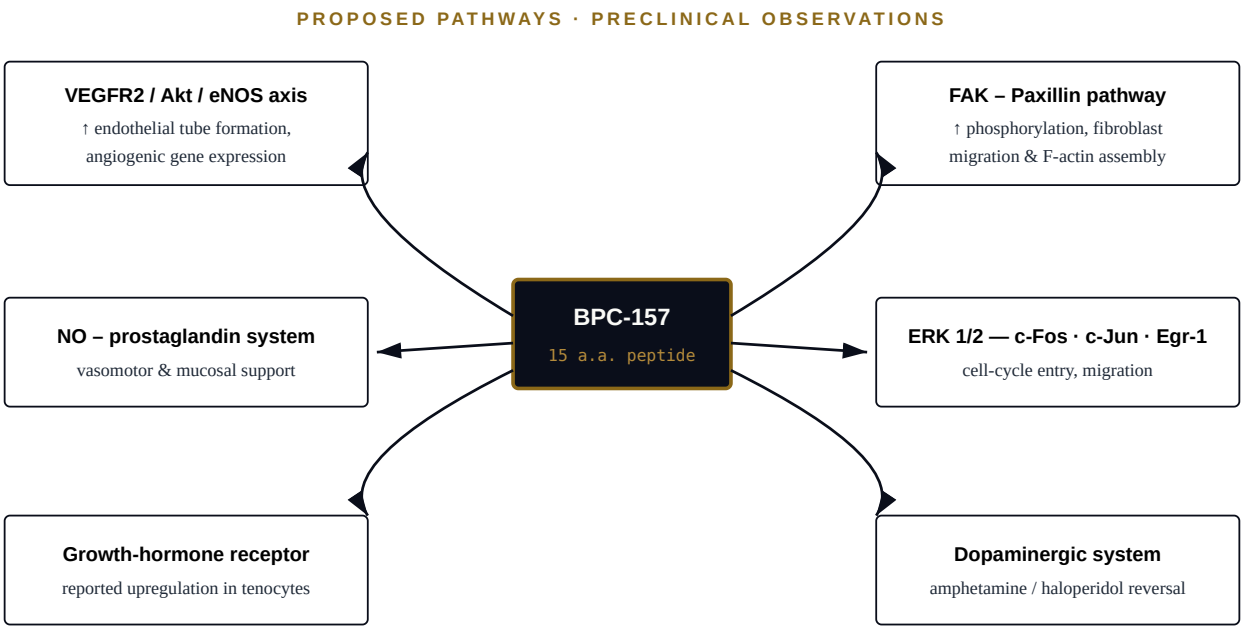

FIGURE 1 Schematic representation of pathways reported in preclinical animal and cell-culture investigations of BPC-157. Arrows indicate proposed influence as described in the cited literature; mechanistic claims remain hypotheses supported by rodent and in-vitro evidence. Compiled from refs 1, 3, 6, 8, 10, 15.

3.1 Angiogenic and Growth-Factor Mimicry

In a 2015 study using an alkali-burn cutaneous wound model in rats and human umbilical vein endothelial cells (HUVECs) in vitro, Huang and colleagues reported that BPC-157 exposure correlated with elevated expression of VEGF-A and accelerated formation of vascular tubes in Matrigel assays.[6] The investigators further reported that downstream ERK 1/2 phosphorylation and the immediate-early genes c-Fos, c-Jun, and Egr-1 were upregulated in BPC-157-exposed cultures, suggesting that the peptide may engage proliferation- and migration-associated transcriptional programmes. IN VITRO

A 2017 study by Hsieh and colleagues using rat aortic ring assays and HUVEC cultures reported that BPC-157 promoted angiogenic outgrowth, with the effect linked to activation of VEGFR2 and downstream Akt/eNOS signalling — providing a mechanistic bridge between the angiogenic observations and the nitric-oxide-related phenomena reported elsewhere in the corpus.[15]

3.2 Focal Adhesion and Cytoskeletal Remodelling

Chang and colleagues examined tendon fibroblasts cultured from Achilles tendons of rats. They reported that, in cultures exposed to BPC-157, phosphorylated forms of focal adhesion kinase (FAK) and paxillin increased without a corresponding change in total protein levels, suggesting modulation of pre-existing pools rather than de-novo synthesis.[1] Western blot data

in the same report were consistent with an enhanced F-actin formation pattern, a marker associated with directed cell migration. The authors proposed activation of the FAK–paxillin pathway as one route by which the peptide may influence tendon fibroblast behaviour. The same group's subsequent work reported transient upregulation of growth-hormone receptor expression on tenocytes following BPC-157 exposure.[12]

3.3 MONOAMINERGIC MODULATION

A line of work originating with Jelovac and colleagues and continued in follow-up studies has reported that BPC-157 administration in rats attenuated stereotypic behaviour induced by amphetamine and reduced haloperidol-induced supersensitivity to amphetamine in chronic dosing paradigms.[10] These observations have been used by the originating group to argue for a "peptidergic defence" interaction with the dopamine system in rodents, although the precise binding site or transporter target has not been characterised in the published literature reviewed here. PRECLINICAL · RODENT

3.4 VASCULAR OCCLUSION AND COLLATERAL CIRCULATION

A 2022 review and series of experimental reports from Sikiric and colleagues, published in *World Journal of Gastroenterology*, summarises preclinical work in rat models of major vessel occlusion — including ischaemia-reperfusion injury following the Pringle manoeuvre, and induced Budd-Chiari syndrome. The authors describe attenuation of portal and caval venous pressure rises and apparent promotion of collateral vessel formation in BPC-157-treated animals relative to vehicle controls.[16]

04 Preclinical Findings by Tissue System

SYSTEM	ANIMAL MODEL / PREPARATION	REPORTED OBSERVATION	REF.
Cutaneous wound	Rat alkali burn; HUVEC Matrigel assay	↑ granulation tissue, VEGF-A, ERK 1/2 phosphorylation	6
Tendon (Achilles)	Rat transection model; tendon fibroblast culture	↑ FAK / paxillin phosphorylation; ↑ outgrowth & migration	1, 4
Gastrointestinal mucosa	Rat gastric, oesophageal, and colonic lesion models	Apparent acceleration of mucosal repair across acute & chronic lesions	7, 8
Skeletal muscle	Rat gastrocnemius crush + corticosteroid challenge	Partial reversal of methylprednisolone-induced muscle damage	9
Central nervous system	Rat closed-head trauma model	Reduced 24-hour mortality; lower hemorrhage and edema scores	11
Vascular / hepatic	Rat ischaemia-reperfusion; induced Budd-Chiari	Attenuation of portal/caval pressure rises; collateral vessel formation	16
Dopamine-related behaviour	Rat amphetamine / haloperidol paradigm	Attenuation of stereotypy; reversal of supersensitivity	10
Heart / cardiovascular	Multiple rat cardiac challenge models	Reported cytoprotective observations across infarction, arrhythmia, pulmonary hypertension preparations	17

SYSTEM	ANIMAL MODEL / PREPARATION	REPORTED OBSERVATION	REF.
Inflammatory bowel	Rat colitis (PL 14736 development programme)	Mucosal protection; advanced to early-phase industrial development	13

4.1 CUTANEOUS WOUND AND BURN MODELS

Huang and colleagues conducted both in-vivo (rat alkali burn) and in-vitro (HUVEC) experiments and reported that BPC-157-exposed animals exhibited what the authors described as accelerated re-epithelialisation, more abundant collagen deposition, and enhanced VEGF-A expression at injured sites compared with vehicle controls.[6] Earlier work by Seiwerth and colleagues observed similar patterns across skin, colon anastomosis, and subcutaneous sponge-implantation models in rats, with histological examination showing greater numbers of collagen fibres, reticulin, and microvessels in treated animals.[5]

4.2 TENDON, LIGAMENT, AND MUSCULOSKELETAL REPAIR

Krivic and colleagues examined functional recovery of the Achilles tendon-to-bone unit following transection in rats and compared BPC-157 to methylprednisolone administration. Functional indices in the corticosteroid arm were reported as worsened relative to control, while BPC-157 animals showed earlier recovery on the indices used.[4] A 2019 review by Gwyer and colleagues synthesises the broader musculoskeletal soft-tissue literature and notes that consistent histological observations across independent groups, while encouraging, derive in substantial part from a single network of laboratories.[12] REVIEW

4.3 GASTROINTESTINAL REPAIR

BPC-157 was originally characterised for cytoprotective behaviour in the gastric mucosa of rodents, and the gastrointestinal-focused literature remains the largest single body of work.[3] Seiwerth and colleagues compared BPC-157 to canonical angiogenic growth factors (EGF, FGF, VEGF) across multiple ulceration models in the oesophagus, stomach, duodenum, and lower GI tract of rats, and reported that BPC-157 produced consistent observations across both acute and chronic lesion types, whereas the comparators were more model-specific.[7]

4.4 CENTRAL NERVOUS SYSTEM

Tudor and colleagues administered BPC-157 in a rat closed-head impact model of traumatic brain injury and reported that, relative to vehicle controls, treated animals exhibited a lower 24-hour mortality rate and reduced severity of subarachnoid and intraventricular haemorrhage on histology, with apparently attenuated cerebral oedema.[11] The authors also tested administration prior to impact and reported a more favourable conscious-to-unconscious ratio in pre-treated animals. PRECLINICAL · RODENT

4.5 CARDIOVASCULAR AND VASCULAR SYSTEM

A 2022 review article in *Biomedicines* by Sikiric and colleagues compiles preclinical work in rat models of myocardial infarction, heart failure, pulmonary hypertension, arrhythmia, and thrombosis. The authors describe consistent cytoprotective observations across these animal preparations, with proposed mechanisms invoking the nitric-oxide system and rapid activation of collateral circulation in response to vascular occlusion.[17]

05 Research Synthesis & Limitations

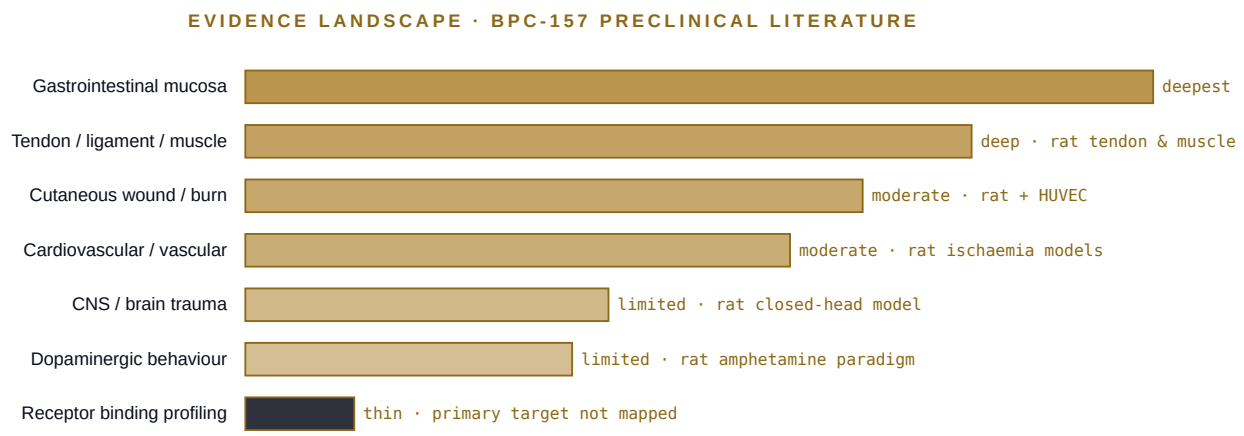

FIGURE 2 Distribution of published BPC-157 evidence across preclinical animal and in-vitro systems. Bar length approximates literature depth per system. The compound's primary receptor binding profile remains incompletely characterised.

METHODOLOGICAL NOTES

A substantial fraction of the BPC-157 corpus originates from a single research network in Zagreb, and replication outside that network, while present, is sparser. Many rodent studies do not blind histological scoring. Pharmacokinetic characterisation in any system is incomplete. The molecule's primary receptor or binding partner has not been definitively identified in the published literature. Researchers should treat consistent observations across rodent systems as hypothesis-generating rather than confirmatory, and design endpoints capable of independently testing mechanistic claims.

5.1 WHAT THE PRECLINICAL LITERATURE CONSISTENTLY REPORTS

Across independent rodent models — wound, tendon, GI mucosa, muscle, brain trauma, and vascular occlusion — investigators have repeatedly reported faster apparent histological repair in BPC-157-exposed animals than in vehicle controls. The molecular correlates most consistently observed are increased VEGF-A signal, increased phosphorylation of FAK / paxillin and ERK 1/2, modulation of nitric-oxide-dependent vascular tone, and upregulation of growth-hormone receptor expression on tenocytes.

5.2 WHERE THE PUBLISHED EVIDENCE IS THIN

Receptor-binding profiles have not been comprehensively mapped. Dose–response relationships across rodent studies vary in design and reporting. Stability in plasma versus enteral or topical exposure routes is not uniformly characterised. Independent replication of the dopamine-system observations is limited. Researchers should design experiments that can independently test the proposed mechanisms rather than assume them.

06 Laboratory Handling, Reconstitution, and Storage

BPC-157 is supplied as a lyophilised powder under research-use specifications. Published in-vitro stability data report the peptide as compatible with bacteriostatic and sterile water for injection at typical research

concentrations, and stable across a wide pH range relative to many comparator peptides — a property attributed to its proline-dense backbone.[5]

For laboratory storage, the lyophilised solid is conventionally held at 2–8 °C, desiccated and light-protected. Reconstituted solutions are conventionally held refrigerated short-term per laboratory protocol; long-term aliquoted storage at −18 °C with minimisation of freeze–thaw cycles is the standard practice in published methodology sections. Working concentrations are determined by the investigator's experimental design.

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07 References

1. Chang C-H, Tsai W-C, Lin M-S, Hsu Y-H, Pang J-HS. The promoting effect of pentadecapeptide BPC 157 on tendon healing involves tendon outgrowth, cell survival, and cell migration. *J Appl Physiol.* 2011;110(3):774–780. PMID: 21030672. pubmed.ncbi.nlm.nih.gov/21030672

2. Sikiric P, Petek M, Rucman R, et al. Brain-gut Axis and Pentadecapeptide BPC 157: Theoretical and Practical Implications. *Curr Neuropharmacol.* 2016;14(8):857–865. PMC5333585. pmc.ncbi.nlm.nih.gov/PMC5333585

3. Krivic A, Majerovic M, Jelic I, Seiwerth S, Sikiric P. Modulation of early functional recovery of Achilles tendon to bone unit after transection by BPC 157 and methylprednisolone. *Inflamm Res.* 2008;57(5):205–210. doi.org/10.1007/s00011-007-7056-8

4. Seiwerth S, Sikiric P, Grabarevic Z, et al. BPC 157's effect on healing. *J Physiol Paris.* 1997;91(3-5):173–178. PMID: 9403790. pubmed.ncbi.nlm.nih.gov/9403790

5. Huang T, Zhang K, Sun L, et al. Body protective compound-157 enhances alkali-burn wound healing in vivo and promotes proliferation, migration, and angiogenesis in vitro. *Drug Des Devel Ther.* 2015;9:2485–2499. doi.org/10.2147/DDDT.S82030

6. Seiwerth S, Brcic L, Vuletic LB, et al. BPC 157 and Standard Angiogenic Growth Factors. Gastrointestinal Tract Healing, Lessons from Tendon, Ligament, Muscle and Bone Healing. *Curr Pharm Des.* 2018;24(18):1972–1989. PMID: 29998800. pubmed.ncbi.nlm.nih.gov/29998800

7. Sikiric P. The pharmacological properties of the novel peptide BPC 157 (PL-10). *Inflammopharmacology.* 1999;7(1):1–14. doi.org/10.1007/s10787-999-0022-z

8. Pevec D, Novinscak T, Brcic L, et al. Impact of pentadecapeptide BPC 157 on muscle healing impaired by systemic corticosteroid application. *Med Sci Monit.* 2010;16(3):BR81–88. PMID: 20190676. pubmed.ncbi.nlm.nih.gov/20190676

9. Jelovac N, Sikiric P, Rucman R, et al. A novel pentadecapeptide, BPC 157, blocks the stereotypy produced acutely by amphetamine and the development of haloperidol-induced supersensitivity to amphetamine. *Biol Psychiatry.* 1998;43(7):511–519. PMID: 9547930. pubmed.ncbi.nlm.nih.gov/9547930

10. Tudor M, Jandric I, Marovic A, et al. Traumatic brain injury in mice and pentadecapeptide BPC 157 effect. *Regul Pept.* 2010;160(1-3):26–32. doi.org/10.1016/j.regpep.2009.11.012

11. Gwyer D, Wragg NM, Wilson SL. Gastric pentadecapeptide body protection compound BPC 157 and its role in accelerating musculoskeletal soft tissue healing. *Cell Tissue Res.* 2019;377(2):153–159. doi.org/10.1007/s00441-019-03016-8

12. Veljaca M, Pavic-Sladoljev D, Mildner B, et al. The development of PL 14736 for treatment of inflammatory bowel disease (PL 14736 industrial development summary). *Curr Pharm Des.* 2003 archive. bib.irb.hr/192824

13. Hsieh M-J, Liu H-T, Wang C-N, et al. Therapeutic potential of pro-angiogenic BPC157 is associated with VEGFR2 activation and up-regulation. *J Mol Med.* 2017;95(3):323–333. PMID: 27847966. pubmed.ncbi.nlm.nih.gov/27847966

14. Sikiric P, Skrtic A, Gojkovic S, et al. Cytoprotective gastric pentadecapeptide BPC 157 resolves major vessel occlusion disturbances, ischemia-reperfusion injury following Pringle maneuver, and Budd-Chiari syndrome. *World J Gastroenterol.* 2022;28(1):23–46. PMID: 35125818. pubmed.ncbi.nlm.nih.gov/35125818

15. Sikiric P, Udovicic M, Barisic I, et al. Stable Gastric Pentadecapeptide BPC 157 as Useful Cytoprotective Peptide Therapy in the Heart Disturbances, Myocardial Infarction, Heart Failure, Pulmonary Hypertension, Arrhythmias, and Thrombosis Presentation. *Biomedicines.* 2022;10(11):2696. PMID: 36359218. pubmed.ncbi.nlm.nih.gov/36359218

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