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BPC-157 and GHK-Cu in Wound Healing and Tissue Repair: A Review of Clinical Efficacy and Safety

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Abstract

Background: BPC-157 (Body Protection Compound-157), a synthetic pentapeptide derived from human gastric juice and GHU-Cu (glycyl-L-histidyl-L-lysine-copper), a naturally occurring tripeptide- copper complex found in human plasma, have garnered significant interest for their regenerative and tissue- repair properties. Both compounds modulate overlapping yet distinct molecular pathways implicated in wound healing, angiogenesis, collagen synthesis and inflammation.
Objective: This narrative review synthesises available preclinical and clinical evidence regarding the efficacy and safety of BPC-157 and GHK-Cu in wound healing and tissue repair, identifies key mechanistic pathways and evaluates the translational potential of these peptides in clinical and sports medicine contexts.
Methods: A comprehensive literature search was conducted across PubMed, Embase and Cochrane Library databases. Studies examining wound healing outcomes, tissue regeneration, molecular mechanisms safety and clinical applications of BPC-157 and GHK-Cu were included, with no date restriction. Both preclinical and clinical studies were considered.
Results: Preclinical evidence consistently demonstrates that BPC-157 accelerates healing of skin, tendon, muscle, ligament and bone though activation of FAK-paxillin and VEGFR2 pathways, modulation of nitric oxide signalling and upregulation of growth hormone receptors.

GHK-Cu promotes collagen, elastin and glycosaminoglycan synthesis, stimulates angiogenesis via VEGF upregulation, enhances fibroblast migration and modulates gene expression across multiple repair pathways. Human clinical data remain limited: BPC-157 has been assessed in only three small studies (including an intra-articular knee pain series, an intravesical pilot for interstitial cystitis and an intravenous safety study) while GHK-Cu has been evaluated in several randomised topical trials demonstrating significant improvements in skin quality and would healing markers. Both peptides demonstrate favourable safety profiles in available studies; however, large-scale randomised controlled trials are absent.
Conclusion: BPC-157 and GHK-Cu hold substantial promise as peptide-based therapeutic agents in wound healing and tissue repair. The current evidence base is predominantly preclinical. Rigorous, adequately powered human clinical trials are urgently needed before widespread clinical adoption can be recommended. This review provides a foundation for future research directions and outlines safety considerations relevant to sports medicine practitioners.
Keyword: BPC-157; GHK-Cu; wound healing; tissue repair; peptide therapy; regenerative medicine; angiogenesis; collagen synthesis; clinical safety; sports medicine

1. Introduction

1.1.The Global Burden of Impaired Wound Healing

Wound healing represents one of the most fundamental and complex biological processes in human physiology, yet its failure or impairment constitutes a major unresolved challenge in modern medicine. Chronic wounds are a silent epidemic affecting an estimated one in six Medicare beneficiaries in the United States alone (approximately 8.2 million people) and the annual cost to Medicare has been estimated at USD 28.1 to 96.8 billion. [1] The global care market reached USD 148.65 billion in 2022 and projections indicate continued growth driven by an ageing population, rising prevalence of diabetes mellitus, vascular insufficiency and antibiotic-resistant infections. [4]
Acute wounds (including surgical incisions, traumatic lacerations and sports-related soft tissue injuries) typically resolve through a well-orchestrated cascade of haemostasis, inflammation, proliferation and remodelling. [3] However, when this cascade is disrupted, wounds become chronic and recalcitrant to standard treatment. In the context of sports medicine, incomplete soft tissue healing represents a leading cause of re-injury, prolonged absence from competitions and career-threatening outcomes for professional and amateur athletes alike. Despite advances in wound dressings, negative pressure therapy, platelet -rich plasma and growth factor administration, a substantial proportion of chronic and sports-related wounds remain refractory to all currently approved interventions.
This persistent therapeutic gap has intensified the search for novel, mechanism-targeted agents capable of restoring the molecular environment necessary for successful tissue repair. Amongst the most promising emerging strategies is the application of the bioactive peptides (short amino acid sequences capable of modulating specific cellular and molecular pathways implicated in healing) with two compounds attracting particular scientific and clinical interest: BPC-157 and GHK-Cu.

1.2.BPC-157: Origins and Emerging Profile

BPC-157, known as the Body Protection Compound, is a synthetic pentadecapeptide with the amino acid sequence Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Asp-Ala-Gly-Leu-Val and a molecular weight of 1,419.55 daltons.[6] It was first described by Sikiric and colleagues in 1994 as a partial sequence of a larger body protection compound isolated from human gastric

juice. [4] Although derived from a naturally occurring gastric protein, the specific 15- amino acid sequence constituting BPC-157 is synthetic and not known to occur independently in nature- a distinction with important regulatory implications.
What distinguishes BPC-157 from many candidate peptides is its exceptional biochemical stability. The peptide remains intact in human gastric juice for more than 24 hours, supporting potential therapeutic effectiveness via oral administration, an advantage rarely seen in peptide-based therapeutics, which typically require parenteral delivery due to proteolytic degradation. [4,5] Mechanistically, BPC-157 promotes healing primarily by activating the vascular endothelial growth factor receptor 2 (VEGFR2) pathway, stimulating angiogenesis, engaging the FAK-paxillin pathway to enhance cellular migration and adhesion, modulating nitric oxide signalling and upregulating growth hormone receptor expression. [7,8] These properties have generated substantial interest in its application to sports medicine and musculoskeletal rehabilitation. Despite this promise, BPC-157 has not been approved by the FDA or any global regulatory authority for human use and was classified in 2023 as Category 2 compounding substance presenting significant safety risks due to insufficient clinical data. [9]

1.3.GHK-Cu: Naturally Occurring Regenerative Tripeptide

GHK-Cu is a tripeptide-copper complex first isolated in 1973 by Loren Pickart from human albumin as a factor capable of restoring youthful biosynthetic activity to aged liver tissue. [11] The GHK sequence (glycyl-L-histidyl-L-lysine) is present naturally in human plasma, saliva, and urine.[12] The tripeptide has a strong affinity for copper (II) ions, readily forming the bioactive GHK-Cu complex.[12] This natural origin distinguishes it from BPC-157 and carries significant implications for both safety profile and regulatory status.

A particularly significant biological observation is GHK-Cu's age-related decline: plasma concentrations fall form approximately 200 ng/mL at age 20 to approximately 80 ng/mL by age 60.[12,13] This decline correlates temporally with the well-documented reduction in regenerative capacity associated with ageing, raising the hypothesis that exogenous GHK-Cu supplementation may partially restore wound healing competence in ageing or compromised tissues. GHK-Cu acts across multiple biological levels: it stimulates synthesis of collagen types I, III, elastin and glycosaminoglycans; promotes fibroblast proliferation and migration; upregulates angiogenic factors including VEGF; modulates matrix metalloproteinase activity; and has been shown to exert genome-wide effects on gene expression, reportedly influencing over 4,000 human genes. [13] Unlike BPC-157, GHK-Cu has an established history of safe use in topical cosmetic formulations.

1.4.Rationale and Aim of the Present Review

Although BPC-157 and GHK-Cu have been extensively studied in parallel lines of research, they share important overlapping biological targets (notably angiogenesis, collagen remodelling, growth factor upregulation and anti-inflammatory signalling) while differing substantially in their molecular origins, regulatory status and current evidence base. A comparative, integrated review is therefore timely and clinically relevant, particularly given the rapid growth in their use among athletes, rehabilitation patients and individuals with chronic wounds seeking interventions beyond conventional therapy.
The present narrative review aims to: (1) synthesise the available preclinical and clinical evidence on BPC-157 and GHK-Cu in wound healing and tissue repair; (2) characterise their principal mechanisms of action and highlight areas of convergence and divergence; (3)

critically appraise the existing safety data and identify remaining gaps; and (4) provide a foundation for the design of future clinical trials in sport medicine and wound care.

2. Methods

This narrative review was conducted with the principles of the Scale for Assessment of Narrative Review Articles (SANRA). A systematic literature search was performed across PubMed/MEDLINE, Embase and the Cochrane Library using the following search terms, applied individually and in combination: 'BPC-157', 'Body Protection Compound 157', 'pentadecapeptide', 'GHK-Cu', "glycyl-L-histydyl-L-lysine', 'copper peptide', 'wound healing', 'tissue repair', 'tendon healing', 'collagen synthesis', 'angiogenesis', 'regenerative medicine' and ' sport medicine'. No date restriction was applied. Additional articles were identified through reference list hand-searching of included studies and recent systematic reviews.

Studies were included if they: (1) investigated BPC-157 or GHK-Cu in the context of wound healing, tissue repair or musculoskeletal injury; (2) reported mechanistic data, preclinical outcomes or human clinical outcomes; and (3) were published in peer-reviewed English-language journals. Case reports with fewer than three subjects, conference abstracts without associated full publications and studies exclusively examining unrelated applications were excluded. All study types were considered given the limited clinical trial landscape; no restrictions were placed on study design.

3. BPC-157: Mechanisms of Action and Preclinical Evidence

3.1.Molecular Mechanisms

BPC-157 exerts its biological effects through activation of multiple, interrelated signalling cascades, which together create a pro-healing microenvironment across diverse tissue types. The four principal pathways are outlined below.

3.1.1. VEGFR2/Akt-eNOS Axis and Angiogenesis

The most extensively characterised mechanism of BPC-157 is activation of the vascular endothelial growth factor receptor 2 (VEGFR2) pathway, leading to downstream phosphorylation of Akt and endothelial nitric oxide synthase (eNOS). This cascade promotes angiogenesis through enhanced endothelial cell proliferation, migration and vascular tube formation. [7,8] BPC-157 also modulates the Src-Caveolin-1-eNOS inhibitory complex: by disrupting this complex in aortic tissue, the peptide directly promotes nitric oxide production in a dose-dependent manner.[27] The result is sustained vasodilation and improved perfusion at injury sites. Importantly, this angiogenic response appears self-limiting and context-sensitive, acting as a biological 'signal initiator' that triggers cascading cellular processes continuing well beyond the peptide's own short half-life of less than 30 minutes.[7,15]

3.1.2. FAK-Paxillin Pathway and Cell Migration

Focal adhesion kinase (FAK) and its own downstream effector paxillin coordinate cellular migration, adhesion and spatial organisation during wound healing, processes fundamental to tissue repair. BPC-157 significantly increases the phosphorylation of both FAK and paxillin in tendon fibroblasts in a dose-dependent manner. [16] Activated FAK combines with Src, inducing phosphorylation of paxillin, which in turn activates downstream effectors CRK and CAS, critical for cell motility. [17] This pathway activation explains the consistent finding of enhanced fibroblast outgrowth and migration toward injury sites across multiple wound models. [16,24]

3.1.3. ERK1/2 Signalling and Transcription Factor Activation

In endothelial cells, BPC-157 activates extracellular signal-regulated kinase 1/ 2 (ERK1/2) signalling, enhancing proliferation, migration and vascular tube formation through downstream transcription factors c-Fos, c-Jun and Egr-1, which regulate cell growth, migration and angiogenesis. [7,8] Pharmacological inhibition of ERK1/2 abolishes BPC-157-driven migration and tube formation, confirming the mechanistic centrality of this pathway. Egr-1 activation additionally promotes collagen organisation at wound sites, with upregulated expression observed in muscle healing models. [24]

3.1.4 Growth Hormone Receptor Upregulation and Anti-inflammatory Effects

BPC-157 significantly upregulates the expression of growth hormone receptors in tendon fibroblasts, sensitising tissues to endogenous growth hormone signalling and thereby amplifying the anabolic response to injury.[18] Simultaneously, the peptide reduces pro-inflammatory cytokines including interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α) and cyclooxygenase-2 (COX-2) expression, attenuating excessive post-injury inflammation while preserving the necessary inflammatory signals for initiation of healing. [7,8] It also upregulates cytoprotective factors including heme oxygenase-1 (HO-1) and heat shock proteins, preserving mitochondrial integrity and reducing oxidative stress. [15]

3.2.Preclinical Evidence by Tissue Type

3.2.1. Skin Wound Healing

BPC-157 has demonstrated consistent efficacy in multiple wound healing models. In an alkali-burn rat model, topical and systemic BPC-157 administration significantly accelerated wound closure, with histological findings demonstrating improved granulation tissue formation, faster re-epithelialisation, enhanced dermal remodelling and higher collagen deposition compared to controls.[24] In vitro assays confirmed BPC-157 enhanced proliferation and migration of human umbilical vein endothelial cells and unregulated VEGF-a expression. [24] Both acute and chronic wound models have demonstrated accelerated closure, with the peptide showing broader applicability across injury types compared to standard growth factor treatments, which typically require complex carriers and delivery systems.

3.2.2. Tendon and Ligament Healing

Tendon healing represents the most extensively studied musculoskeletal application of BPC-157. In eight tendon and ligament transection models reviewed by Vasireddi et al. ,[7] BPC-157 treatment was consistently associated with reduced instability, prevention of indices. The seminal study by Chang et al. [16] demonstrated that BPC-157 significantly accelerated tendon fibroblast outgrowth from tendon explants in a dose-dependent manner, increased cell survival under oxidative stress and enhanced migration via the FAK-paxillin pathway. Biomechanical improvements, introducing greater load-to-failure values, have been demonstrated in both Aschilles tendon and quadriceps models.

3.2.3. Muscle, Ligament and Bone

In four preclinical muscle transection and crush injury models, BPC-157 improved load-to-failure, motor function indices and myofibril diameter restoration. [7,9] For bone repair, BPC-157 performed comparably to autologous bone marrow injection and bone grafting in fractur model, promoting callus mineralisation and lamellar bone formation over fibrous scar tissue. [10] A recent study evaluated BPC-157 following surgical quadriceps detachment and reattachment in rats, demonstrating superior biochemical recovery and histological muscle architecture compared to controls. [19]

4. GHK-Cu: Mechanisms of Action and Evidence

4.1.Molecular Mechanisms

4.1.1. Collagen, Elastin and Extracellular Matrix Synthesis

The most robustly established action of GHK-Cu is stimulation of extracellular matrix (ECM) synthesis. The GHK-Cu sequence is present within the alpha-2(I) chain of type I collagen and when damage activates proteolytic enzymes, GHK is released directly at the injury site suggesting an endogenous, injury-triggered wound healing mechanism. [12] GHK-Cu stimulates synthesis of collagen types I, III and IV; elastin; dermatan sulphate; chondroitin sulphate and the small proteoglycan decorin, which organises collagen fibrils and maintains structural skin integrity. [12,13] The collagen stimulating effect begins at concentrations as low as 10^{-12} M, maximises at approximately 10^{-9}M and is independent of changes in cell number. [23] Copper within the complex serves as an essential cofactor for lysyl oxidase and lysyl hydroxylase, enzymes critical for collagen cross-linking and stability. [21]

4.1.2. MMP Modulation and ECM Remodelling

GHK-Cu modulates matric metalloproteinase (MMP) activity in a balanced, context-sensitive manner, increasing gene expression of MMP-2 at low concentrations (0.01 nM) while simultaneously increasing tissue inhibitor of mettaloproteinase-1 (TIMP-1). [11] This dual regulatory effect prevents both excessive ECM accumulation (fibrosis) and excessive degradation, supporting organized wound remodelling rather than pathological scarring. In chronic wound models, GHK-Cu-treated diabetic rats showed increased collagen synthesis versus controls, with improved epithelialisation and activated fibroblast and mast cell activity. [21]

4.1.3. Angiogenesis and Growth Factor Stimulation

GHK-Cu promotes angiogenesis through stimulation of VEGF production by fibroblasts and pre-treatment of mesenchymal stem cells (MSC). A biodegradable carrier mediated study demonstrated that GHK treated MSCs showed dose-dependent increases inf VEGF and basic fibroblast growth factor (bFGF) secretion, mediated via the integrin alpha-6/beta-1 pathway. [12] In fibroblast models, GHK-Cu in combination with LED irritation increased bFGF production by 230% and collagen synthesis by 70%. [22] The copper component additionally stimulates keratinocyte migration and endothelial cell growth, facilitating re-epithelisation and vascular ingrowth simultaneously. [21]

4.1.4. Genome-Wide Gene Modulation

Perhaps the most distinctive property of GHK-Cu, distinguishing it from virtually all other wound healing agents, is its capacity to modulate gene expression on genome-wide scale. Using Broad Institute Connectivity Map analysis, GHK has been shown to influence the expression of over 4,000 human genes, with particular enrichment in pathways related to tissue repair, inflammatory resolution, antioxidant defence, DNA repair and tumour suppression. [13] Critically, GHK-Cu appears to reset the gene expression profile of aged cells toward more youthful patterns, potentially explaining its broad regenerative benefit across tissue types. At the anti-inflammatory level, GHK-Cu reduces TNF-alpha induced secretion of IL-6 in normal human dermal fibroblasts and downregulates MMP-2 and MMP-9 at wound sites, reducing local metalloproteinase activity and associated tissue breakdown. [12]

4.2.Preclinical Evidence by Application

4.2.1. Skin Wound Healing

GHK-Cu has demonstrated accelerated healing across multiple species and wound types, including skin lacerations, burns, foot pad wounds in dogs and systemic wound healing induction in rats, mice and pigs. [11] In diabetic wound models, GHK-Cu impregnated dressings produce a ninefold increase in collagen synthesis versus controls, with significantly improved re-epithelialisation rates. [20] The compound also demonstrates anti-scarring properties by promoting organised collagen deposition resembling normal skin architecture rather than the random arrangement characteristic of hypertrophic scar tissue. [20] Liposomal and nano-lipid carrier formulations of GHK-Cu have demonstrated enhanced epidermal penetration and sustained release, improving biological efficacy compared to conventional topical application. [24]

4.2.2. Lung, Liver, Bone and Gastrointestinal Repair

GHK-Cu's regenerative activity extends beyond ski to multiple visceral tissues. In lung fibroblasts derived from chronic obstructive pulmonary disease (COPD) patients, GHK treatment restored collagen gel contraction and remodelling capacity to levels comparable to non-diseased controls, accompanied by elevated integrin beta-1 expression. [11] In vivo stimulation of connective tissue accumulation has been demonstrated in experimental rat wounds. GHK-Cu has additionally shown hepatoprotective effects, supported gastrointestinal mucosal repair in animal models and demonstrated osteogenic activity in bony tissue. [12]

5. Results: Clinical Evidence in Humans

5.1.BPC-157: Available Human Data

Despite more than three decades of preclinical research, the human clinical evidence base for BPC-157 remains extremely limited. A systemic review by Vasireddi et al. [7] identified 544 articles spanning 1993-2024 across PubMed, Cochrane and Embase, of which only 36 met inclusion criteria and of these, 35 were preclinical studies and only one was a clinical study. Three human studies have since been published or identified.

Lee and Padgett [42] conducted a retrospective case series of 16 patients who received intra-articular BPC-157 injections (2-4 mcg of a 2,000 μg/mL solution) for chronic, unspecified knee pain. Approximately 87.5% of patients reported subjective improvement in pain for more than six months at follow-up. However, the authors acknowledge that most patients were treated for ligamentous and tendinous sprains that often self-resolve and that the study did not measure functional outcomes or quality of life. [42]

Lee et al. [15] reported a pilot study evaluating intravesical BPC-157 injections in 12 patients with moderate to severe interstitial cystitis. This represented the first intravesical application of the compound and 80-100% symptom resolution was reported. No adverse events were observed, though the study was uncontrolled, unblinded and limited to a small sample.

Lee and Burgess [35] conducted the only available pharmacokinetic and intravenous safety study in healthy human volunteers. Two adults (a 58-year-old male and a 68-year-old female) each received 10 mg IV infusion on day 1 and 20 mg on day 2. No adverse events were reported and no clinically meaningful changes were detected in cardiac, hepatic, renal, thyroid or metabolic biomarkers. Plasma BPC-157 concentrations returned to baseline within 24 hours.

Despite the encouraging safety findings of this study, its sample size of two subjects precludes any meaningful generalisability.

Of note, a Phase I clinical trial sponsored by PharmaCotherapia (ClinicalTrial.gov NCT02637284), enrolling 42 healthy volunteers, was initiated in 2015, but its results were never published. No public explanation was provided for failure to report, representing a significant missed opportunity in the evidence base.

5.2.GHK-Cu: Available Human Data

GHK-Cu has a longer history of human application, primarily via topical formulations in dermatological and cosmetic contexts. Several placebo-controlled and open clinical trials have demonstrated improvements in skin quality markers. A facial cream containing GHK-Cu applied for 12 weeks to 71 women with mild to advanced photoaging significantly increased skin density and thickness, reduced skin laxity, improved clarity and reduced fine lines and wrinkle depth. [12] A GHK-Cu eye cream applied for 12 weeks similarly improved periorbital laxity and firmness. [12]

In a randomised, double-blind clinical trial, female volunteers applying GHK-Cu encapsulated in nano-lipid carrier twice daily for eight weeks achieved a 55.8% reduction in wrinkle volume and a 32.8% reduction in wrinkle depth compared to control serum. [12] A collagen production study using skin biopsy immunohistochemistry demonstrated that GHK peptide cream applied to the thigh for one month produced a significant effect on collagen production, with approximately 70% of GHK-Cu users showing collagen improvement compared to approximately 50% with vitamin C and 40% with retinoic acid. [13]

For wound healing beyond aesthetic applications, the clinical evidence remains largely at the ex vivo and early clinical stage. The ability of GHK-Cu to restore irradiated fibroblast proliferation, demonstrated in vitro by Kang et al.,[22] has not yet been translated into clinical trials for post-radiation wound healing, despite its biological rationale. A 2023 study combining GHK-Cu with hyaluronic acid in fibroblast cultures and ex vivo skin models demonstrated synergistic upregulation of collagen IV expression, supporting the combination formulations. [28]

5.3.Summary of Human Clinical Studies

Table 1 presents a summary of all published human clinical studies involving BPC-157 and GHK-Cu identified in this review.

Table 1. Summary of published human clinical studies involving BPC-157 and GHK-Cu.

Author /Year	Compound	Study Type	N	Indication	Dose/Route	Duration	Primary Outcome	Result
Lee & Padgett (2021) [42]	BPC-157	Retrospective case series	16	Chronic knee pain (ligamentous/tendinous)	Intra-articular 2-4 µg	Single injection; follow-up >6 months	Subjective pain improvement	87.5% reported improvement >6 months; no adverse events; no functional outcome measured

Lee et al. (2024) [15]	BPC-157	Uncontrolled pilot	12	Moderate-severe interstitial cystitis	Intravesical injection	Not specified	Symptom resolution	80-100% symptom resolution; no adverse events; unblinded, uncontrolled
Lee & Burgess (2025) [35]	BPC-157	Pharmacokinetic safety pilot	2	Healthy volunteers (PK/safety)	IV 10mg day 1; 20mg day 2	2 days	Adverse events; biomarkers	No adverse events; no clinically meaningful biomarker changes; plasma return to baseline <24h
Pickart & Margolina (2015) [11]	GHK-Cu	Open-label clinical study	71	Mild- advanced photoaging	Topical facial cream, twice daily	12 weeks	Skin density, laxity, wrinkle depth	Significant increase in skin density/thickness; reduced laxity; improved clarity; reduced fine lines/wrinkles
Pickart & Margolina (2018) [12]	GHK-Cu	RCT (double blind)	Not specified	Skin aging/wrinkle reduction	Nano-lipid carrier serum, twice daily	8 weeks	Wrinkle volume/depth (3D imaging)	55.8% reduction in wrinkle volume; 32.8% reduction in wrinkle depth vs. control
Pickart & Margolina (2018) [13]	GHK-Cu	Controlled histological study	Not specified	Collagen production (thigh skin)	Topical peptide cream	4 weeks	Collagen by immunohistochemistry	~70% of users showed collagen improvement vs. ~50% (vitamin C) and ~40% (retinoic acid)
Jiang et al. (2023) [28]	GHK-Cu	Ex vivo/in vitro	N/A	Collagen IV synthesis (fibroblasts + skin model)	GHK-CU + hyaluronic acid	Not specified	Collagen IV expression	Synergistic upregulation of collagen IV; supports combination formulation strategies

GHK-Cu= glycyl-L-histydyl-L-lysine copper; PK=pharmacokinetics; RCT=randomised controlled trial.

6. Comparative Analysis: BPC-157 versus GHK-Cu

6.1.Mechanistic Convergence and Divergence

Despite their fundamentally different origins (one synthetic and gastric derived, the other naturally occurring in human plasma) BPC-157 and GHK-Cu share several key therapeutic mechanisms, particularly in their promotion of angiogenesis and anti-inflammatory activity. Both compounds upregulate VEGF-related pathways, reduce pro-inflammatory cytokine expression (including IL-6 and TNF-α) and promote fibroblast activity and migration. These shared targets suggest potential additive or synergistic activity when both compounds are used

together, a hypothesis that has attracted interest in regenerative medicine research but has not yet been formally investigated in clinical trials.

However, the compounds diverge substantially in several mechanistic dimensions. BPC-157 demonstrates superior activity in musculoskeletal and neuromuscular tissues, particularly tendon, ligament and muscle, via its FAK-paxillin and ERK1/2 pathways, mechanisms that are not characteristic of GHK-Cu. Conversely, GHK-Cu's capacity for broad-spectrum gene modulation (>4,000 genes), its collagen cross-linking support via copper-dependant enzymes and its anti-scarring properties represent advantages not yet demonstrated by BPC-157. GHK-Cu also has more established safety records in humans, given decades of topical use in cosmetic preparations.

Table 2 presents a comparative summary of the key characteristics of BPC-157 and GHK-Cu.

Table 2. Comparative characteristics of BPC-157 and GHK-Cu

Characteristic	***BPC-157***	***GHK-Cu***
Origin	Synthetic pentadecapeptide; derived from human gastric juice protein sequence	Naturally occurring tripeptide; present in human plasma, saliva, urine
Molecular weight	1,419.55 Da (15 amino acids)	340.38 Da (3 amino acids + Cu^{2+})
Structure	Gly-Glu-Pro-Pro-Pro-Gly-Lys-Pro-Ala-Asp-Ala-Gly-Leu-Val	Gly-His-Lys complex with Cu^{2+}
Route(s) Studied	Oral, IM, IV, intra-articular, intravesical, topical (preclinical)	Topical (clinical); systemic (preclinical)
Primary Mechanisms	VEGFR2/Akt-eNOS angiogenesis; FAK-paxillin cell migration; GH receptor upregulation; anti-inflammatory (IL-6, TNF-α, COX-2)	Collagen/elastin/GAG synthesis; MMP/TIMP modulation; VEGF/bFGF upregulation; genome-wild gene modulation (>4,000 genes); copper-dependent enzyme cofactor
Key Tissue Evidence	Skin, tendon, ligament, muscle, bone, GI tract, nervous system (all predominantly preclinical)	Skin, lung, liver, GI tract, bone (preclinical and topical clinical)
Human Clinical Trials	3 studies; <30 subjects; no RCT	Several topical RCTs; wound healing evidence limited to cosmetic/ ex vivo
Safety Profile	Favourable in preclinical models; very limited human data; theoretical pro-angiogenic cancer risk unconfirmed	Established topical safety record; decades of cosmetic use; limited systemic human data
FDA Status	Not approved; Category 2 compounding substance (2023) significant safety risk designation	Accepted for topical use; not approved as systemic therapeutic
WADA Status	Prohibited (2022)	Not prohibited
Anti-Scarring	Not specifically demonstrated	Demonstrated- promotes organised collagen architecture
Oral Bioavailability	Yes (gastric stable >24h)	Not established for oral use
Synergistic Potential	Proposed with GHK-Cu (overlapping angiogenic/ anti-inflammatory pathways) not clinically tested	Proposed with BPC-157 and hyaluronic acid; synergy with HA demonstrated in vitro

FDA= U.S. Food and Drug Administration; GAG= glycosaminoglycan; GI= gastrointestinal; IM= intramuscular; IV= intravenous; MMP=matrix metalloproteinase; RCT= randomised controlled trial; VEGFR2= vascular endothelial growth factor receptor 2; WADA= World Anti-Doping Agency

6.2.Sports Medicine Applications

In the sports medicine context, BPC-157 has attracted the greatest attention for its musculoskeletal applications (specifically tendon, ligament and muscle repair) given the high prevalence of these injuries in athletic populations and the limitations of current conservative and surgical management. The peptide's gastric stability and potential oral bioavailability are particularly attractive in athletic settings where parenteral administration may be impractical. Its documented effects on growth hormone receptor expression may also carry implications for anabolic recovery, though no studies have specifically addressed performance-enhancing effects.

GHK-Cu may offer complementary applications in sport medicine' particularly in skin wound healing following abrasions or surgical procedures, in post-radiation tissue repair for cancer survivors returning to sport and potentially in connective tissue health given its robust effects on collagen and elastin synthesis. The anti-scarring properties of GHK-Cu are of specific relevance in tendon repair, where the formation of disorganised scar tissue rather than functional tendon is a primary determinant of long-term outcome.

The recent ban of BPC-157 by the World Anti-Doping Agency (WADA 2022), the NFL, UFC and NCAA significantly complicate its use in competitive sport. Athletes who receive the compound unknowingly highlight the practical risks of unregulated peptide use in athletic context. GHK-Cu is not currently prohibited by WADA.

7. Safety and Regulatory Considerations

7.1.BPC-157 Safety

Preclinical safety studies of BPC-157 have consistently demonstrated a favourable acute toxicity profile, with an LD1 (lethal dose in 1% of subjects) not achieved even at extremely high doses in rodent models and no observed adverse effects across multiple organ systems including hepatic, renal and cardiovascular assessments. [5,7,31] The peptide has been studied in Phase II ulcerative colitis trials and multiple sclerosis trials without reported toxicity, providing additional indirect evidence of human tolerability at doses used in inflammatory bowel disease. [5]

Despite this, in 2023 the FDA classified BPC-157 as a Category 2 substance presenting significant safety risks under its compounding guidelines, citing insufficient safety data for any human application and concerns about immune reactions and peptide impurity profiles. [7] This decision effectively prohibits BPC-157 compounding under Section 503A of US federal law. Key safety concerns highlighted in the recent literature include its pro-angiogenic activity (specifically, theoretical risks of potentiating tumour growth or metastasis via VEGF/VEGFR2 and FAK-paxillin activation) though no in vivo tumour models have confirmed this risk and Sikiric et al. [14] argue that BPC-157 operates outside the classical angiogenesis-cancer framework. No adverse events have been reported across the three available human studies to date, though the combined sample size is only 30 subjects.

7.2.GHK-Cu Safety

GHK-Cu has an established safety record accumulated over decades of topical cosmetic use in millions of consumers globally, without reports of serious adverse events attributed to the compound. [12] Topical GHK-Cu formulations are FDA-accepted and feature extensively in dermatological preparations. No oncogenic risk has been identified despite GHK-Cu's pro-

angiogenic and pro-proliferative properties; indeed, its gene modulation profile includes significant upregulation of tumour suppressor pathways. [12]

For systemic or injectable applications, human safety data remain limited. The copper component warrants attention at supraphysiological doses, given copper's role in free radical generation via Fenton-type chemistry; however, the concentrations employed in therapeutic GHK-Cu preparations are substantially below levels associated with copper toxicity. No clinical safety studies specifically evaluating injectable GHK-Cu in wound healing contexts have been published, representing a gap in the evidence base for systemic applications.

7.3.Regulatory Summary

The regulatory landscape for both peptides is complex and evolving. BPC-157 is not approved by the FDA, EMA or any major regulatory authority for the therapeutic indication. It is prohibited by WADA, the NFL, the UFC and the NCAA. Injectable forms cannot be legally compounded in the United States following the 2023 FDA Category 2 designation. GHK-Cu is accepted for topical use in the United States and the European Union but is not approved as a systemic therapeutic agent. Neither compound has completed Phase III clinical trials for any wound healing indication. Clinicians and sport medicine practitioners must be aware of these regulatory constraints when advising patients or athletes regarding peptide-based therapies.

8. Future Research Directions

The most urgent research need in this field is the conduct of adequately powered, randomised, double-blind, placebo-controlled clinical trials examining wound healing and tissue repair outcomes for both BPC-157 and GHK-Cu in well-defined patient populations. Priority investigation areas include: (1) BPC-157 in acute tendon and ligament injury repair, given the strength of preclinical evidence; (2) GHK-Cu in chronic wound management, particularly in diabetic foot ulcers; (3) combined BPC-157 and GHK-Cu treatment protocols to evaluate potential synergistic effects; and (4) BPC-157 safety pharmacology in larger human cohorts with systemic biomarker monitoring.

Translational challenges include the absence of patent protection for naturally derived sequences such as BPC-157 and GHK-Cu, which reduces commercial incentive for investment in large-scale clinical trials. Novel delivery systems (including nanoparticle conjugates, hydrogels, bioresorbable scaffolds and liposomal formulations) may enhance the clinical utility of both peptides and represent active areas of research. [25] The integration of GHK-Cu into wound dressings for chronic wound management is particularly close to clinical translation, given its established topical safety profile.

From a mechanistic standpoint, the proposed interaction between BPC-157 and Src family kinases, potentially mediated through its proline-rich sequence and SH3-domain binding, requires validation through direct biochemical assays and human cell systems. [4] For GHK-Cu, the genome-wide gene expression data, though striking in scope, require validation through prospective transcriptomic studies in wound tissue biopsy samples from clinical trials.

9. Conclusions

BPC-157 and GHK-Cu represent two of the most mechanistically compelling peptide candidates in the emerging field of regenerative wound healing. BPC-157 exhibits a remarkably broad preclinical profile across skin, tendon, muscle, ligament and bone, mediated through VEGFR2/Akt-eNOS, FAK-paxillin and ERK1/2 signalling cascades, with consistent

functional, structural and biomechanical improvements across over 30 years of animal research. GHK-Cu brings the complementary strength of natural origin, established topical safety, potent collagen-stimulating, anti-scarring properties and unprecedented breadth of gene modulation. Both compounds engage angiogenic and anti-inflammatory pathways that are central to successful tissue repair.

However, the evidence base for clinical application of both peptides (particularly for BPC-157) remains overwhelmingly preclinical. Fewer than 30 humans have been studied in the context of BPC-157 treatment and no randomised controlled trials have been conducted for either compound in wound healing or musculoskeletal repair. The regulatory landscape for BPC-157 is increasingly restrictive and its WADA-prohibited status creates significant barriers to use in sport. GHK-Cu has a stronger human safety record but lacks clinical data for wound healing indications beyond topical skin applications.

Sports medicine practitioners, wound care clinicians and researchers are encouraged to view the existing data as hypothesis-generating rather than practice-defining. The translational potential of BPC-157 and GHK-Cu is real, but its realisation requires rigorous clinical investigation that has, thus far, not occurred. This review provides a synthesis of the available evidence framework for the clinical trials that are now urgently needed.

Disclossure

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