RonanRx
PERSONALIZED MEDICINE

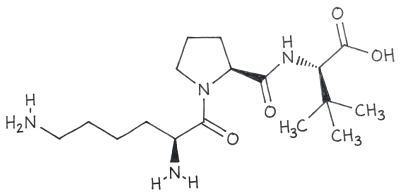
CLINICAL MONOGRAPH · TISSUE REPAIR (UNDER FDA REVIEW)

KPV

Tripeptide research ingredient with physician-request review

KPV is a short peptide made of three amino acids, lysine, proline, and valine. It is the tail end of a longer hormone called alpha-melanocyte-stimulating hormone (α-MSH), which the body produces from the same protein precursor as ACTH and beta-endorphin [brzoska2008]. Researchers became interested in KPV after observing that the full α-MSH hormone had anti-inflammatory effects and that the short C-terminal tripeptide retained much of that activity in laboratory studies.

KPV has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.



EVIDENCE POSTURE PRECLINICAL

REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

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FOR CLINICIANS

KPV (lysine-proline-valine) is the C-terminal tripeptide of alpha-melanocyte-stimulating hormone (α-MSH 11, 13), an endogenous 13-residue POMC-derived neuropeptide [brzoska2008, luger2007] [getting2003]. The tripeptide retains a substantial fraction of α-MSH's anti-inflammatory activity in vitro and in vivo despite lacking the full melanocortin receptor binding sequence; mechanistic dissection studies attribute KPV activity to inhibition of NF-κB nuclear translocation and downstream proinflammatory cytokine signaling, with contributions from both melanocortin-receptor-dependent and -independent pathways [kelly2006; bohm2019; gravina2023]. Intracellular uptake of KPV in intestinal epithelial cells is mediated by the H+-coupled di- and tripeptide transporter PepT1 [dalmasso2008] [land2012].

Preclinical efficacy has been reported in murine DSS- and TNBS-colitis models [kannengiesser2008, dalmasso2008, sun2021], in murine models of colitis-associated colorectal cancer [viennois2016], in colon-targeted oral nanoparticle and hydrogel delivery systems [laroui2010, xiao2017, zhao2022], in rodent oral mucositis [shao2021], and in keratinocyte and corneal wound-healing assays [elliott2004, sung2025, adnan2025]. Reviews summarize a coherent class of α-MSH-derived peptides with anti-inflammatory, antimicrobial, and cytoprotective activity [brzoska2010]. No published randomized clinical efficacy trials in humans are indexed in PubMed at the time of review; the evidence base is mechanistic and preclinical.

KPV has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.

Why Personalized KPV

The evidence base for KPV is still preclinical. Published work centers on cell-culture and animal models of mucosal inflammation, skin injury, oral mucositis, and host-defense biology, without large randomized human efficacy trials.

Physicians may submit patient-specific prescription requests for KPV for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, supported by patient-specific documentation, and approved by the dispensing pharmacy. Availability is determined case by case. This is not a consumer access promise; it is a clinical, sourcing, formulation, and regulatory review process. FDA has scheduled KPV-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting.

A physician-submitted pharmacy review is the regulated contrast to KPV sold through research-chemical channels, where identity, sterility, potency, clinical rationale, and patient follow-up are not tied to a licensed pharmacist.

Quick Facts About KPV

Category: Tripeptide (α-MSH C-terminal fragment, Lys-Pro-Val)

Active ingredient: Lysine-proline-valine, the three C-terminal residues (α-MSH 11, 13) of alpha-melanocyte-stimulating hormone, a 13-residue neuropeptide derived from POMC processing

FDA-approved branded forms: None. KPV is not an FDA-approved drug substance and has no approved branded product.

Evidence posture: Preclinical only, anti-inflammatory and antimicrobial activity in cell culture and rodent models of inflammatory bowel disease, wound healing, oral mucositis, and skin inflammation. No published randomized human efficacy trials.

FDA-approval status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

Compounded under: Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

> **SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY**
>
> Physicians may submit patient-specific prescription requests for KPV for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case.
>
> - **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
> - **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
> - **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
> - **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
> - **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is KPV?

KPV is a synthetic tripeptide composed of L-lysine, L-proline, and L-valine, corresponding to residues 11, 13 (the C-terminus) of alpha-melanocyte-stimulating hormone. α-MSH itself is a 13-amino-acid neuropeptide produced by post-translational processing of pro-opiomelanocortin (POMC), the same precursor that yields adrenocorticotropic hormone (ACTH), β-endorphin, and β-lipotropin. The full α-MSH molecule binds melanocortin receptors MC1R through MC5R; its central message sequence (residues 4, 10, HFRWGKP) carries the pigmentation-active pharmacophore [brzoska2008, luger2007].

Interest in the KPV C-terminal fragment originated with observations that anti-inflammatory effects of α-MSH could be reproduced by C-terminal fragments lacking the canonical melanocortin-receptor-binding message sequence [luger2007, brzoska2010]. The Getting et al. 2003 mechanistic dissection demonstrated that the anti-inflammatory effect of α-MSH could be separated from the pigmentation pharmacophore and localized to the C-terminal tripeptide [getting2003]. Subsequent work characterized cytokine-suppressive activity in keratinocytes, intestinal epithelial cells, and bronchial epithelium [elliott2004, kelly2006, land2012].

KPV is supplied as a synthetic peptide for research use and is not an FDA-approved drug substance. It has no FDA-approved branded product and no human prescribing-information label.

How KPV Works

KPV's reported mechanism of action centers on inhibition of nuclear factor-kappa B (NF-κB) signaling and downstream suppression of proinflammatory cytokine production. In keratinocytes, intestinal epithelial cells, and bronchial epithelial cells, exposure to KPV reduces TNF-α-induced NF-κB nuclear translocation and lowers transcription of IL-1β, IL-6, IL-8, and other NF-κB-driven cytokines [elliott2004].

Whether KPV acts primarily through melanocortin receptors (MC1R, MC3R, MC5R) or through receptor-independent intracellular mechanisms has been investigated in multiple model systems and is not fully resolved. Getting and colleagues (2003) dissected the anti-inflammatory effect and reported that KPV retained activity in melanocortin-receptor-blockade conditions, supporting a partially receptor-independent intracellular mechanism [getting2003]. Land (2012) characterized KPV signaling in bronchial epithelium with a proposed contribution from MC3R agonism [land2012]. Reviews acknowledge the dual mechanism and emphasize that the tripeptide retains anti-inflammatory activity in models where the parent α-MSH pharmacophore is not required [brzoska2010, luger2007] [kelly2006].

In intestinal epithelial cells, KPV is internalized by the proton-coupled oligopeptide transporter PepT1, which is upregulated in inflamed colonic mucosa [dalmasso2008, viennois2016]. PepT1-mediated uptake

concentrates KPV at the site of inflammation and may underlie the colon-targeted activity reported in DSS- and TNBS-colitis models.

Biological Role of KPV

Alpha-melanocyte-stimulating hormone is a 13-residue neuropeptide produced from POMC processing in the pituitary intermediate lobe, hypothalamus, skin keratinocytes, and immune cells. It binds the five melanocortin receptors (MC1R, MC5R) with distinct receptor-affinity and tissue-expression patterns and contributes to pigmentation (MC1R), energy balance (MC4R), exocrine function (MC5R), and immune-cell modulation (MC1R, MC3R, MC5R). Anti-inflammatory and immunomodulatory effects of α-MSH have been characterized across innate and adaptive immune cell types, with cytokine suppression and downregulation of NF-κB-driven transcription as recurring mechanisms [brzoska2008, luger2007].

The C-terminal tripeptide KPV (α-MSH 11, 13) retains a substantial fraction of the anti-inflammatory activity of the parent hormone despite lacking the central message sequence required for canonical melanocortin-receptor pharmacology [getting2003, brzoska2010]. This dissociability of anti-inflammatory activity from pigmentation pharmacology motivated the original interest in KPV as a candidate small-peptide anti-inflammatory and underlies the current preclinical evidence map.

KPV Research History

Interest in α-MSH-derived peptides as anti-inflammatory agents grew through the 1990s and 2000s. Cutuli et al. (2000) characterized antimicrobial activity of α-MSH peptides including KPV-containing fragments against Staphylococcus aureus and Candida albicans, contributing to the dual anti-inflammatory and antimicrobial framing [cutuli2000]. Getting, Schiöth, and Perretti (2003) published the foundational mechanistic dissection showing that anti-inflammatory activity localized to the C-terminal tripeptide and could be separated from the pigmentation pharmacophore [getting2003]. Elliott et al. (2004) demonstrated cytokine-modulating activity of KPV in human keratinocyte cell lines, supporting a dermatologic rationale [elliott2004], and Kelly et al. (2006) reported that immobilized GKPV (α-MSH 10, 13, a closely related fragment) inhibits TNF-α-stimulated NF-κB activation [kelly2006].

The intestinal application emerged in the late 2000s. Dalmasso et al. (2008) reported that KPV is taken up by intestinal epithelial cells via PepT1 and that oral KPV reduced DSS-colitis severity in mice [dalmasso2008]. Kannengiesser et al. (2008), working in the Luger group, extended the finding across murine DSS and CD4+CD45RB-high transfer colitis models, characterizing KPV as a candidate anti-inflammatory in inflammatory bowel disease [kannengiesser2008]. Laroui et al. (2010) translated the work into a colon-targeted nanoparticle delivery system that reduced colitis severity in mice at lower drug exposures than free KPV [laroui2010]. Viennois et al. (2016) extended the framework to colitis-associated colorectal cancer in a mouse model and characterized the therapeutic benefit of PepT1-mediated KPV

delivery [viennois2016]. Xiao et al. (2017) developed a hyaluronic acid-functionalized nanoparticle system for oral KPV delivery that further improved efficacy in murine ulcerative colitis [xiao2017].

Concurrent dermatologic and ophthalmic work continued. Brzoska et al. (2008) published a comprehensive Endocrine Reviews summary of α-MSH and related tripeptides covering biochemistry, anti-inflammatory and protective effects in vitro and in vivo, and proposed therapeutic perspectives [brzoska2008]. Brzoska et al. (2010) extended this with the 'terminal signal' framework arguing that anti-inflammatory effects of α-MSH-related peptides operate beyond the canonical pharmacophore [brzoska2010]. Böhm and Luger (2019) reviewed the case for melanocortin peptides, including KPV, as future therapeutics in cutaneous wound healing [bohm2019]. Recent hydrogel and nanoparticle delivery work [sun2021] has continued to refine preclinical formulations for colitis and oral mucositis [sun2021, zhao2022, shao2021]. Reviews of the broader melanocortin system in IBD [gravina2023] and of tripeptides in wound healing [adnan2025] integrate KPV into the current preclinical evidence map; no randomized human efficacy trials are indexed.

KPV Timeline

- **2000** • Cutuli et al. (J Leukoc Biol) characterize antimicrobial activity of α-MSH peptides including KPV-containing fragments against S [cutuli2000]. aureus and C. albicans
- **2003** • Getting, Schiöth, and Perretti (J Pharmacol Exp Ther) dissect anti-inflammatory activity of α-MSH and localize it to the C-terminal KPV tripeptide [getting2003]
- **2004** • Elliott et al [elliott2004]. (J Invest Dermatol) demonstrate KPV (α-MSH 11, 13) signaling in human keratinocyte cell lines
- **2006** • Kelly et al [kelly2006]. (Peptides) report that immobilized GKPV (α-MSH 10, 13) inhibits TNF-α-stimulated NF-κB activation
- **2007** • Luger and Brzoska (Ann Rheum Dis) review α-MSH-related peptides as a class of anti-inflammatory and immunomodulating drug leads [luger2007]
- **2008** • Dalmasso et al [dalmasso2008]. (Gastroenterology) report that PepT1-mediated KPV uptake reduces intestinal inflammation in murine DSS colitis
- **2008** • Kannengiesser et al [kannengiesser2008]. (Inflamm Bowel Dis) extend the IBD finding to multiple murine colitis models including the CD4+CD45RB-high transfer model
- **2008** • Brzoska et al [brzoska2008]. (Endocr Rev) publish a comprehensive review of α-MSH and related tripeptides covering biochemistry, anti-inflammatory effects, and therapeutic perspectives
- **2010** • Laroui et al [laroui2010]. (Gastroenterology) describe a polysaccharide-hydrogel colon-targeted KPV nanoparticle delivery system that reduces murine colitis severity

2010 • Brzoska et al [brzoska2010]. (Adv Exp Med Biol) advance the 'terminal signal' framework, anti-inflammatory effects of α-MSH-related peptides beyond the canonical pharmacophore

2012 • Land (Int J Physiol Pathophysiol Pharmacol) characterizes KPV mechanism in human bronchial epithelial cells and proposes an MC3R-agonist contribution [land2012]

2016 • Viennois et al [viennois2016]. (Cell Mol Gastroenterol Hepatol) extend the framework to colitis-associated colorectal cancer in mice; therapeutic benefit of PepT1-mediated KPV

2017 • Xiao et al [xiao2017]. (Mol Ther) develop a hyaluronic acid-functionalized oral nanoparticle delivery system for KPV in murine ulcerative colitis

2019 • Böhm and Luger (Exp Dermatol) review melanocortin peptides including KPV as candidate cutaneous wound-healing therapeutics [bohm2019]

2021 • Sun et al [sun2021]. (ACS Biomater Sci Eng) report a self-cross-linked γ-polyglutamic acid hydrogel KPV formulation in rat TNBS ulcerative colitis

2021 • Shao et al [shao2021]. (Biomater Sci) describe an in situ mucoadhesive hydrogel capturing KPV with anti-inflammatory and antibacterial effects in chemotherapy-induced oral mucositis

2022 • Zhao et al [zhao2022]. (Acta Biomater) characterize a KPV-binding double-network hydrogel that restores gut mucosal barrier in an inflamed mouse colon

2023 • Gravina et al [gravina2023]. (Cells) review the melanocortin system in inflammatory bowel diseases including KPV mechanism and therapeutic potential

2025 • Sung et al [sung2025]. (Tissue Cell) report KPV mitigation of fine-dust-induced keratinocyte apoptosis and inflammation via MAPK/NF-κB modulation

2025 • Adnan et al [adnan2025]. (Int J Med Sci) review tripeptides, including KPV, in wound healing and skin regeneration

Compounded KPV (503A)

Physicians may submit patient-specific prescription requests for pharmacy review. For KPV, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for KPV is still preclinical. Published work centers on cell-culture and animal models of mucosal inflammation, skin injury, oral mucositis, and host-defense biology, without large randomized human efficacy trials.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. FDA has scheduled KPV-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For KPV, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for KPV are reviewed before any preparation is made or released. A physician-submitted pharmacy review is the regulated contrast to KPV sold through research-chemical channels, where identity, sterility, potency, clinical rationale, and patient follow-up are not tied to a licensed pharmacist.

KPV Formulations and Routes

Form	Concentration	Description
Research-grade synthetic tripeptide	—	KPV is available from peptide chemistry suppliers as a synthetic lysine-proline-valine tripeptide for research use. It is not an FDA-approved drug substance and is not available as a manufactured pharmaceutical product.
Preclinical experimental formulations (not commercial products)	—	Published preclinical studies have used colon-targeted polysaccharide-hydrogel nanoparticles [laroui2010], hyaluronic acid-functionalized oral nanoparticles [xiao2017], γ-polyglutamic acid self-cross-linked hydrogels [sun2021], KPV-binding double-network hydrogels [zhao2022], and in situ mucoadhesive hydrogels [shao2021]. These are research formulations characterized in animal models, not commercial drug products.

KPV Safety

No published human safety data are indexed for KPV at the time of this review. Animal-model studies in mice and rats report short-term tolerability at the experimental doses used, with no consistent organ-toxicity signal across the colitis, oral mucositis, and wound-healing literature [13][15][16]. Absence of acute toxicity in short-duration rodent studies does not establish human safety; longer-term toxicology, immunogenicity, reproductive toxicity, carcinogenicity, and pharmacokinetic data have not been published.

Because KPV is on FDA's Category 2 bulk-substance list for 503A compounding [21], FDA has identified either safety concerns or an information gap that must be evaluated. Clinicians considering KPV-containing preparations from non-503A sources should be aware that such products are not subject to FDA bulk-

substance review, USP <797> sterility standards, or pharmacist verification of identity and potency [7617]. Availability through RonanRx is determined case by case after pharmacy review.

Published reviews of α-MSH-derived peptides as a class describe a generally favorable preclinical safety profile relative to small-molecule anti-inflammatories, attributed to the short peptide structure and rapid catabolism [8510]; these reviews are not equivalent to human safety data for KPV specifically.

Contraindications

Honest gap. No published human contraindications for KPV are indexed. KPV is not an FDA-approved drug substance and has no prescribing-information label. FDA Category 2 bulk-substance status precludes 503A compounding pending reclassification.

Searched: PubMed, FDA bulk-substance review documents on 2026-05-11 · terms *KPV tripeptide AND (contraindication OR contraindicated OR hypersensitivity)*.

Drug interactions

Honest gap. No published human drug-interaction studies for KPV are indexed at the time of review. PepT1 transporter-mediated uptake in intestinal epithelium [dalmasso2008] suggests theoretical co-substrate or inhibitor interactions with other PepT1 substrates (e.g., β-lactam antibiotics, ACE inhibitors with peptidomimetic structure) but no clinical interaction studies have been published.

Searched: PubMed, DailyMed on 2026-05-11 · terms *KPV tripeptide AND (drug interaction OR pharmacokinetic interaction)*.

Adverse events

Honest gap. No published human adverse-event series are indexed for KPV. Preclinical rodent studies in colitis, oral mucositis, and wound-healing models report short-term tolerability without consistent organ-toxicity signals at experimental doses, but human adverse-event data are absent.

Searched: PubMed, FDA Adverse Event Reporting System (FAERS) on 2026-05-11 · terms *KPV tripeptide AND (adverse event OR adverse effect OR side effect)*.

Monitoring KPV Therapy

No RonanRx-specific monitoring protocol has been established for KPV. If a patient-specific prescription is submitted, supporting clinical rationale may be requested, and monitoring expectations would be reviewed case by case against the published evidence, route, sterile or nonsterile status, concomitant therapies, and patient risk factors.

KPV in Special Populations

KPV Evidence Quality

The KPV evidence base is preclinical. Indexed PubMed literature consists of mechanistic in vitro studies in keratinocyte, intestinal epithelial, and bronchial epithelial cell lines, rodent models of inflammatory bowel disease using DSS and TNBS chemical colitis induction or CD4+CD45RB-high transfer protocols, colon-targeted nanoparticle and hydrogel oral delivery systems characterized in murine colitis [laroui2010, xiao2017, zhao2022], rodent oral mucositis models [shao2021], keratinocyte and corneal wound-healing assays [elliott2004, sung2025], and narrative and systematic reviews summarizing the class. Cutuli et al [sun2021; kelly2006; getting2003]. (2000) and related work characterized antimicrobial activity against S [brzoska2008; luger2007; brzoska2010]. aureus and C [kannengiesser2008; dalmasso2008; viennois2016]. albicans [cutuli2000] [gravina2023; adnan2025].

No published randomized controlled human trials of KPV efficacy are indexed in PubMed. No published human pharmacokinetic, pharmacodynamic, or dose-response studies are indexed. No FDA-approved branded product exists. KPV is currently on FDA's Category 2 bulk-substance list for 503A compounding [fda_cat2_peptides] [bohm2019; land2012]. The mechanistic and preclinical evidence is internally consistent and biologically plausible but does not constitute clinical evidence; any clinical claim about KPV in humans extrapolates from rodent and cell-culture data and should be framed as preclinical-grade.

Major KPV Clinical Studies

Study	Design	Participants	Duration	Finding
Getting et al. (2003, J Pharmacol Exp Ther), Anti-inflammatory dissection of α-MSH	In vitro and rodent in vivo dissection of α-MSH anti-inflammatory activity, comparing the full hormone, the core message sequence, and the C-terminal KPV tripeptide	—	—	Anti-inflammatory activity localizes to the C-terminal KPV tripeptide and is at least partially independent of the canonical melanocortin-receptor-binding pharmacophore; receptor-blockade experiments support a receptor-independent intracellular mechanism contribution [getting2003]
Cutuli et al. (2000, J Leukoc Biol), Antimicrobial α-MSH peptides	In vitro antimicrobial assays of α-MSH and C-terminal fragment peptides against	—	—	α-MSH and KPV-containing C-terminal fragments exhibit antimicrobial activity against S [cutuli2000]. aureus and C.

Study	Design	Participants	Duration	Finding
	bacterial and fungal targets			albicans at micromolar concentrations
Elliott et al. (2004, J Invest Dermatol), Keratinocyte signaling	In vitro studies in human keratinocyte cell lines comparing α-MSH, MSH 11, 13 (KPV), and ACTH signaling	—	—	KPV reproduces a substantial fraction of α-MSH signaling effects in human keratinocyte cell lines, supporting a dermatologic rationale for the tripeptide fragment [elliott2004]
Kelly et al. (2006, Peptides), Immobilized GKPV inhibits NF-κB	In vitro mechanism study with immobilized α-MSH 10, 13 (GKPV) on TNF-α-stimulated NF-κB activation in cell culture	—	—	Immobilized GKPV inhibits TNF-α-stimulated NF-κB nuclear translocation and downstream cytokine transcription [kelly2006]
Dalmasso et al. (2008, Gastroenterology), PepT1-mediated KPV in DSS colitis	In vitro PepT1 transporter studies in intestinal epithelial cells and in vivo DSS-colitis model in mice receiving oral KPV	—	—	KPV is taken up by intestinal epithelial cells via PepT1; oral KPV reduces colitis severity, neutrophil infiltration, and proinflammatory cytokine expression in DSS-colitis mice [dalmasso2008]
Kannengiesser et al. (2008, Inflamm Bowel Dis), KPV in multiple murine IBD models	Murine DSS-colitis and CD4+CD45RB-high transfer colitis models with oral or systemic KPV administration	—	—	KPV reduces colitis severity, mucosal cytokine production, and histologic injury across DSS and adoptive-transfer colitis models, anti-inflammatory potential across mechanistically distinct IBD models [kannengiesser2008]
Laroui et al. (2010, Gastroenterology), Colon-targeted KPV nanoparticles	Polysaccharide-hydrogel-encapsulated KPV nanoparticles in murine DSS colitis	—	—	Colon-targeted KPV nanoparticles reduce colitis severity at substantially lower drug exposures than free KPV, supporting the local-delivery rationale [laroui2010]
Land (2012, Int J Physiol Pathophysiol Pharmacol),	In vitro mechanism study in human bronchial epithelial	—	—	KPV inhibits cellular and systemic inflammation signaling in bronchial epithelium with

Study	Design	Participants	Duration	Finding
Bronchial epithelial KPV mechanism	cells with KPV and melanocortin-receptor-selective agonists			proposed contribution from MC3R agonism [land2012]
Viennois et al. (2016, Cell Mol Gastroenterol Hepatol), PepT1 in CAC	Murine model of colitis-associated colorectal cancer with intestinal-epithelial-specific PepT1 modulation and therapeutic KPV	—	—	PepT1 plays a critical role in promoting colitis-associated cancer; KPV delivered via PepT1 provides therapeutic benefit in the murine CAC model [viennois2016]
Xiao et al. (2017, Mol Ther), Hyaluronic-acid-functionalized KPV nanoparticles	Orally administered hyaluronic-acid-functionalized nanoparticle KPV formulation in a murine ulcerative colitis model	—	—	HA-functionalized oral nanoparticles efficiently target inflamed colon and alleviate ulcerative colitis in mice at lower systemic drug exposures than free peptide [xiao2017]
Sun et al. (2021, ACS Biomater Sci Eng), γ-PGA hydrogel KPV in TNBS colitis	Self-cross-linked cysteamine-grafted γ-polyglutamic acid hydrogel KPV formulation in rat TNBS-colitis model	—	—	Hydrogel-stabilized KPV reduces TNBS-colitis severity in rats with improved retention at the inflamed mucosa relative to free peptide [sun2021]
Shao et al. (2021, Biomater Sci), Mucoadhesive KPV hydrogel for oral mucositis	In situ mucoadhesive hydrogel capturing KPV in a chemotherapy-induced oral mucositis model in rodents	—	—	Mucoadhesive KPV hydrogel produces anti-inflammatory, antibacterial, and tissue-repair effects in chemotherapy-induced oral mucositis [shao2021]
Zhao et al. (2022, Acta Biomater), KPV-binding hydrogel restores gut barrier	KPV-binding double-network hydrogel in an inflamed murine colon model	—	—	The KPV-binding double-network hydrogel restores gut mucosal barrier function and reduces inflammation in inflamed colon [zhao2022]
Sung et al. (2025, Tissue Cell),	In vitro keratinocyte study of fine-dust-induced apoptosis and	—	—	KPV mitigates fine-dust-induced keratinocyte apoptosis and inflammation by regulating

Study	Design	Participants	Duration	Finding
Keratinocyte fine-dust injury	inflammation with KPV intervention			oxidative stress and modulating MAPK/NF-κB signaling [sung2025]
Brzoska et al. (2008, Endocrine Reviews), α-MSH and related tripeptides	Comprehensive narrative review covering biochemistry, in vitro and in vivo anti-inflammatory and protective effects of α-MSH and related tripeptides including KPV	—	—	Integrates the preclinical evidence base for α-MSH-derived peptides as anti-inflammatory and immunomodulatory candidates with proposed therapeutic perspectives in immune-mediated inflammatory disease [brzoska2008]
Böhm and Luger (2019, Exp Dermatol), Melanocortin peptides for wound healing	Narrative review of melanocortin peptides, including KPV, as candidate cutaneous wound-healing therapeutics	—	—	Reviews the preclinical case for melanocortin peptides in cutaneous wound healing and identifies KPV among the most-studied C-terminal fragments; clinical translation remains preclinical-stage [bohm2019]

KPV Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

No published human pharmacokinetic data for KPV are indexed at the time of review. The tripeptide is small (molecular weight 342.4 g/mol for the free peptide) and is expected to be rapidly catabolized by serum and tissue peptidases. In intestinal epithelial cells the H+-coupled di- and tripeptide transporter PepT1 mediates active uptake, with PepT1 expression upregulated in inflamed colonic mucosa, the molecular basis for the inflammation-targeted activity reported in murine colitis models [dalmasso2008, viennois2016].

Preclinical formulation work has focused on protecting KPV from upper-GI proteolysis and concentrating delivery at the inflamed mucosa: polysaccharide-hydrogel nanoparticles [laroui2010], hyaluronic-acid-functionalized oral nanoparticles [xiao2017], γ-polyglutamic acid hydrogels [sun2021], double-network hydrogels [zhao2022], and mucoadhesive in situ gels [shao2021]. These formulations are research tools rather than commercial products.

Pharmacodynamics

Reported pharmacodynamic endpoints in preclinical studies include NF-κB-driven proinflammatory cytokine expression (TNF-α, IL-1β, IL-6, IL-8), histologic colitis severity scores (disease activity index, microscopic injury), neutrophil infiltration markers (myeloperoxidase activity), epithelial barrier integrity (transepithelial electrical resistance, FITC-dextran permeability), and wound-closure or re-epithelialization rates in skin and oral mucosa models [kannengiesser2008; kelly2006; xiao2017]. No validated human pharmacodynamic biomarker for KPV exists [dalmasso2008; zhao2022; shao2021].

KPV Compounding & Operations

503A compounding

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Pharmacist review

For KPV, the pharmacist review starts before any preparation is made. Valid patient-specific prescription required. Supporting clinical rationale may be requested. The pharmacist reviews ingredient status, sourcing, formulation feasibility, state requirements, patient-specific documentation, and whether dispensing is appropriate case by case.

Quality and traceability

If a KPV preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records. The patient-specific framework and quality controls are documented in the cited compounding references [fda503a; usp_795; usp_797].

Cold chain

If a KPV preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records.

Frequently Asked Questions About KPV

Can physicians request KPV through RonanRx?

Physicians may submit patient-specific prescription requests for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case. Compounded medications are not FDA-approved, and no consumer self-ordering, office stock, or bulk dispensing is offered.

What is KPV?

KPV is a synthetic tripeptide of lysine, proline, and valine, the three C-terminal residues (positions 11, 13) of alpha-melanocyte-stimulating hormone (α-MSH), a 13-residue POMC-derived neuropeptide [brzoska2008; getting2003]. The C-terminal fragment retains a substantial fraction of α-MSH's anti-inflammatory activity in preclinical models despite lacking the canonical melanocortin-receptor-binding message sequence [luger2007].

Is there human evidence for KPV?

Not in the form of published randomized human efficacy trials [shao2021; bohm2019]. The PubMed-indexed evidence base for KPV is preclinical: mechanistic in vitro studies in keratinocyte, intestinal, and bronchial epithelial cells; murine and rat models of DSS- and TNBS-colitis, colitis-associated cancer, oral mucositis, and wound healing; and class reviews of α-MSH-derived peptides [kannengiesser2008; dalmasso2008; xiao2017].

Why is KPV interesting to IBD researchers?

Kannengiesser et al. (2008) reported anti-inflammatory activity of KPV across murine DSS-colitis and CD4+CD45RB-high transfer colitis [kannengiesser2008; viennois2016]. Dalmasso et al. (2008) showed that KPV is taken up by intestinal epithelial cells via the PepT1 transporter, which is upregulated in inflamed colonic mucosa, providing a mechanistic rationale for inflammation-targeted activity [dalmasso2008]. Multiple groups have since developed colon-targeted nanoparticle and hydrogel formulations characterized in rodent colitis [xiao2017]. All of this work is preclinical.

Could KPV move from FDA Category 2 to Category 1?

Possibly, if the agency receives sufficient evidence to evaluate safety and clinical utility. The Category 2 designation reflects FDA's current assessment of the evidence base; it is not a permanent finding. RonanRx will track future bulk-substance review revisions and will reconsider compounding only if and when KPV is moved to Category 1 [fda_cat2_peptides].

Where does KPV fit in the α-MSH and melanocortin literature?

KPV is the C-terminal tripeptide of α-MSH (residues 11, 13) [land2012]. The full α-MSH binds melanocortin receptors MC1R, MC5R; the C-terminal tripeptide retains anti-inflammatory activity through partially receptor-independent intracellular pathways with proposed contributions from MC3R and MC5R, as documented in the Brzoska Endocrine Reviews summary and subsequent mechanism studies [brzoska2008; brzoska2010; getting2003].

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How to Access KPV

Compounded KPV is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed KPV, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)