KPV: chemical identity and published assays

28 November 2026

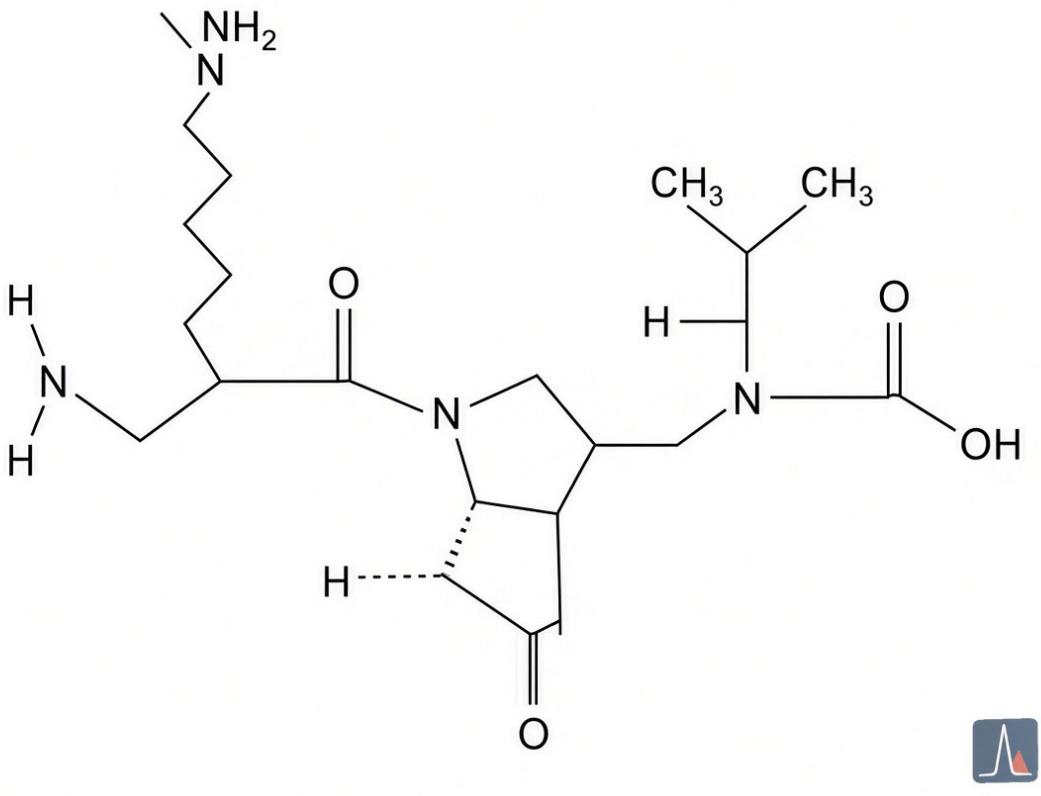
This briefing identifies KPV and summarizes published assays that characterize it.

KPV (Lys-Pro-Val or α-MSH(11–13)) is a synthetic tripeptide ($C_{16}H_{30}N_4O_4$, average molecular weight 342.43 Da) supplied exclusively for laboratory research at /compounds/kpv. This briefing identifies its chemical structure and summarizes published chromatographic, mass-spectrometric, and functional assays that characterize the molecule in research models. All materials from Rierink Compounds are for laboratory research only and are not medicines.

Chemical Identity and Catalog Context

KPV is the linear tripeptide H-Lys-Pro-Val-OH (all L-configuration). It corresponds to residues 11–13 at the C-terminus of α-melanocyte-stimulating hormone (α-MSH; sequence Ac-SYSMEHFRWG**KPV**-NH_2). The synthetic research material is the free carboxylic acid, whereas native α-MSH features C-terminal amidation. Identifiers include CAS 67727-97-3, PubChem CID 125672, and InChIKey YSPZCHGIWAQVKQ-AVGNSLFASA-N. The catalog entry is typically the acetate or trifluoroacetate salt; the counter-ion constitutes a significant mass fraction of the lyophilized powder and must be accounted for when calculating net peptide content.

KPV is one of the smallest catalog peptides. Its polarity and net positive charge at neutral pH make it highly water-soluble. It is positioned in the catalog alongside other research materials studied for anti-inflammatory or tissue-response pathways such as GHK-Cu, LL-37, and Ara-290. Reporting a human trial is not guidance to use the research material in any therapeutic manner.



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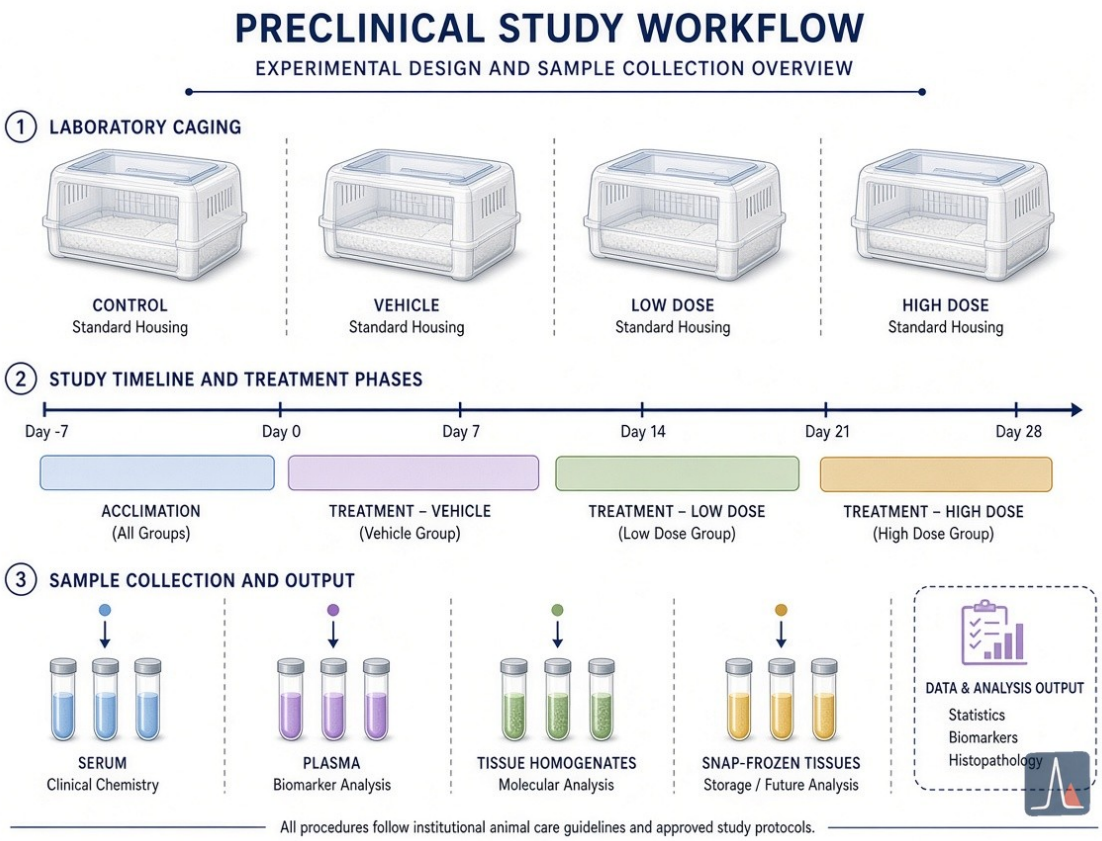
Chemical identity of KPV as the Lys-Pro-Val tripeptide.



Conceptual layout of preclinical in vivo study evidence without depicting administration.

Primary Sequence and Physicochemical Properties

The primary sequence is Lys[1]-Pro[2]-Val[3] with free N-terminus (α-amine of Lys plus ε-amine side chain) and free C-terminal carboxylate on Val. Proline imposes conformational constraint on the backbone. The molecule carries a net positive charge near physiological pH (pI ≈ 9.7) due to the two basic nitrogens on lysine. Computed properties include XLogP3 ≈ −3.5, topological polar surface area ≈ 139 Å², and GRAVY score −0.43, confirming high hydrophilicity. There are no aromatic residues, so the peptide shows negligible absorbance at 280 nm and is quantified at 205–220 nm via peptide-bond absorbance.

The free acid monoisotopic mass is 342.2267 Da; the protonated ion $[M+H]^+$ appears at m/z 343.234. Salt forms shift the weighed mass (acetate adds ~60 Da). Stereocenters are all defined (L-configuration). These properties dictate choice of chromatographic conditions and explain early elution on reversed-phase columns.

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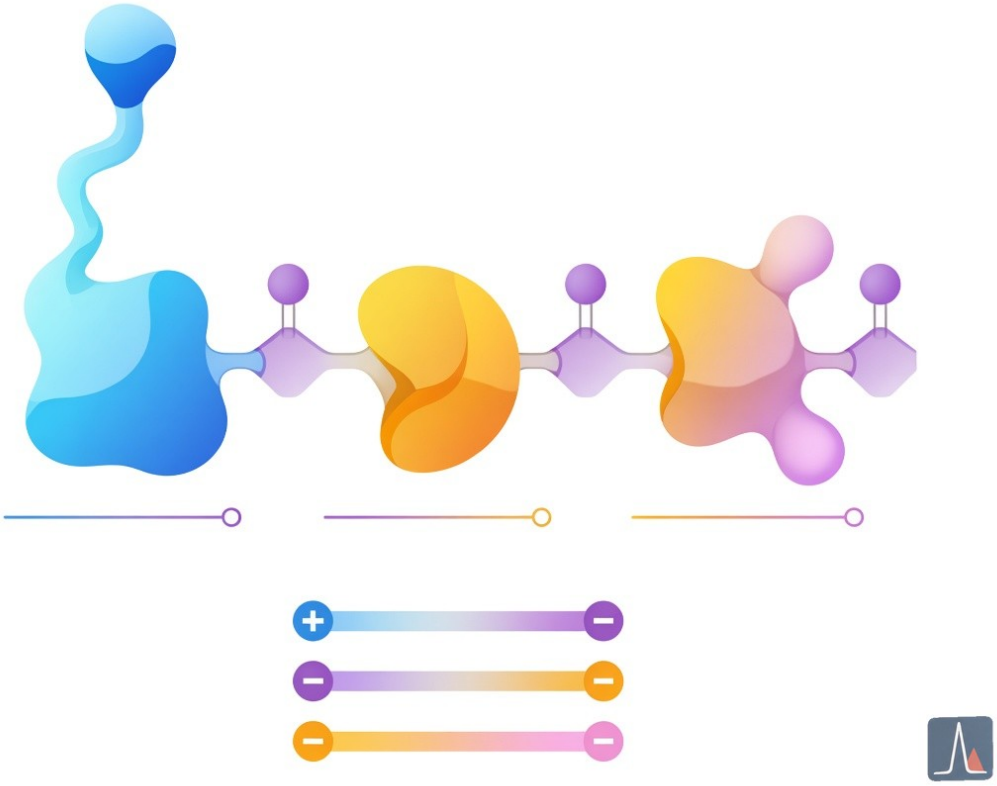

Primary sequence and physicochemical character of KPV.

Published Chromatographic Assays for Purity, Impurities, and Stability

Reverse-phase HPLC is the primary method for purity assessment. Vendor COAs routinely report >98–99.8 % area purity using C18 columns, 0.1 % TFA in water/acetonitrile gradients, and UV detection at 214–220 nm. The polar tripeptide elutes early (often 3–6 min depending on gradient), requiring careful integration of the solvent front and related substances.

A stability-indicating RP-HPLC method was published by Pawar et al. (Biomed Chromatogr, 2015). The method used a Phenomenex C18 column (4.6 × 250 mm, 5 µm), 0.1 % TFA/water (A) and acetonitrile (B) gradient, and was validated for aqueous solutions and skin homogenates. Linearity was excellent (r = 0.9999, 0.25–100 µg/mL range), with LOD 0.01 µg/mL and LOQ 0.25 µg/mL. Precision and accuracy were <2 % RSD. Under stress (acid, alkali, peroxide, heat), the major degradant was the cyclic Lys-Pro diketopiperazine, separated from the parent peak and identified by flow-injection MS. The method resolved KPV from matrix components in skin homogenates, making it suitable for both purity/release testing and stability studies. Commercial COAs frequently mirror these conditions and report main-peak purity of 99.5–99.85 % with minor early-eluting impurities (e.g., glycine or synthesis by-products).

Endotoxin (LAL) testing is commonly included on research-grade material (<0.5 EU/mg or <0.05 EU/mL post-reconstitution). No extensive long-term lyophilized stability data specific to KPV appear in the primary literature, but general peptide principles (cool, dry, protected from moisture) apply.

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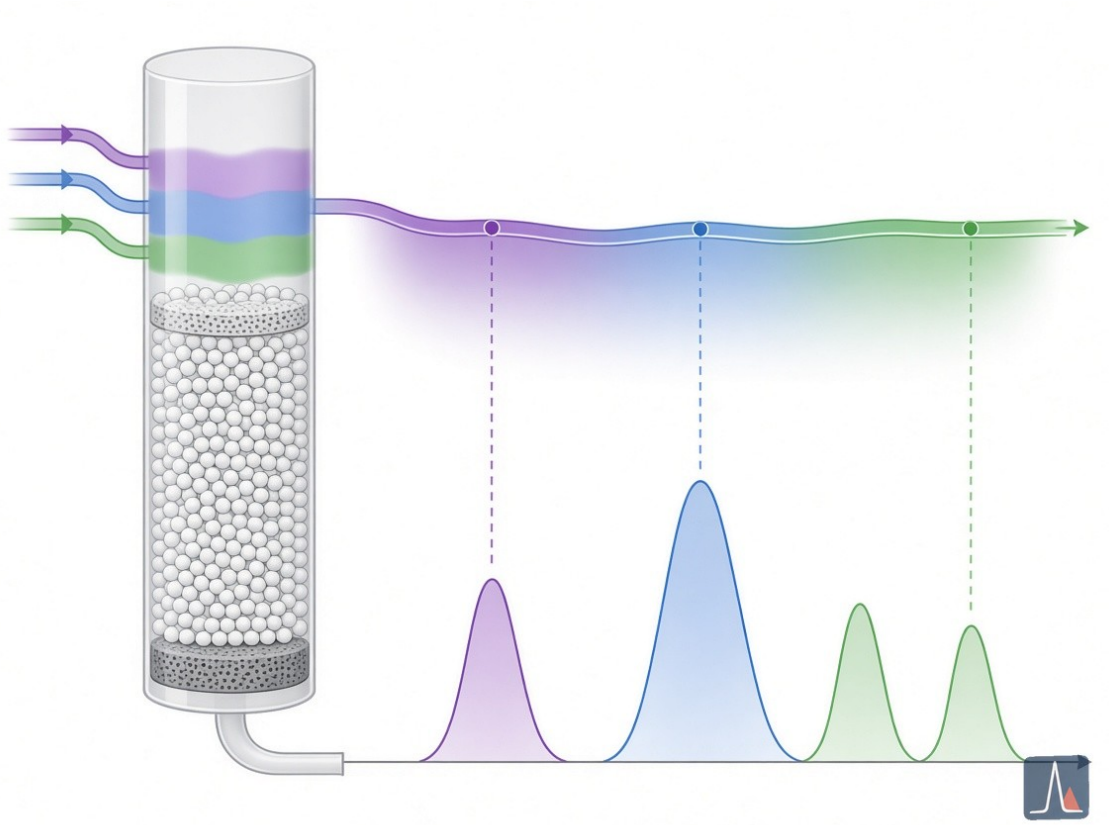

Published-style chromatographic assay concept for purity, impurities, and stability.

Mass Spectrometric Assays and Molecular Confirmation

LC-MS or HPLC-MS is the standard identity test. The expected $[M+H]^+$ is m/z 343.234 (free acid). COAs consistently match the observed monoisotopic mass to within 0.1–0.5 Da and may report the $[M+2H]^{2+}$ ion at m/z ≈ 172.12 or sodium/potassium adducts. Fragmentation spectra can confirm sequence order via b- and y-ions, although for a tripeptide the molecular ion match plus retention time is usually sufficient.

In stability work, flow-injection or LC-MS identified the diketopiperazine degradant (loss of 18 Da, distinct mass and retention). Isotope patterns and high-resolution exact-mass measurements further support identity. MS alone does not distinguish L/D stereoisomers or sequence order without fragmentation or orthogonal methods; therefore it is paired with HPLC retention time and, where available, amino-acid analysis or NMR on synthesized batches.

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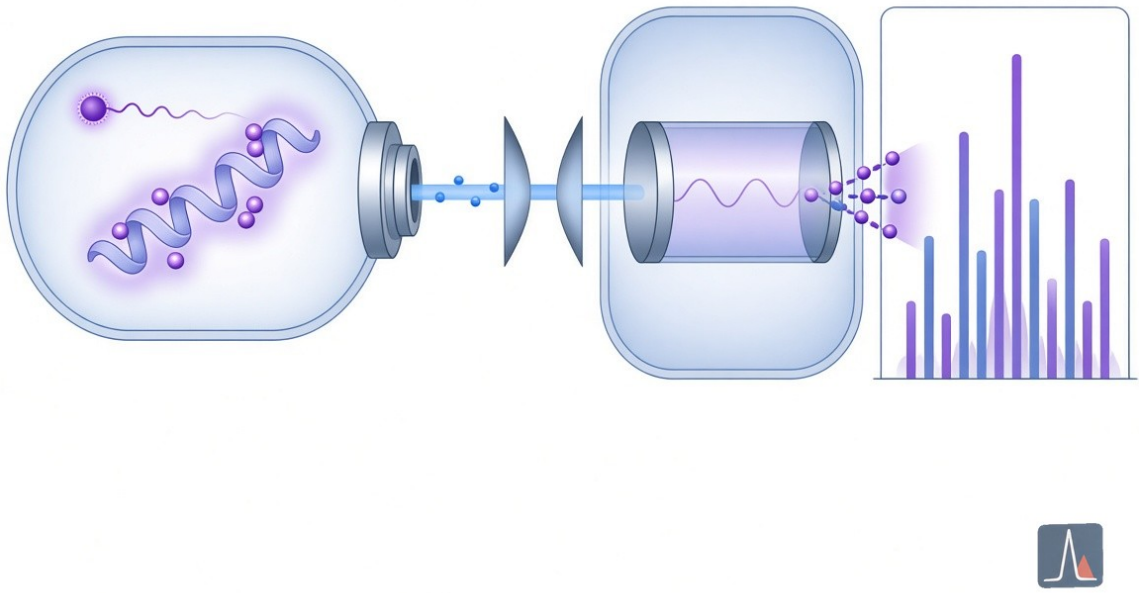

Mass spectrometric molecular confirmation concept for KPV.

Functional Bioassays: NF-κB Inhibition, PepT1 Uptake, and Cytokine Modulation

KPV has been characterized in numerous cell-based anti-inflammatory assays. In Caco-2-BBE, HT-29 intestinal epithelial cells, and Jurkat T cells, nanomolar concentrations of KPV inhibited NF-κB-driven luciferase reporter activity, reduced p65 nuclear translocation, stabilized IκBα, and lowered pro-inflammatory cytokine mRNA and protein levels (TNF-α, IL-1β, IL-6, IL-8, KC) after cytokine or LPS stimulation (Dalmasso et al., Gastroenterology, 2008; PMID 18061177). Uptake was shown to be PepT1-dependent: KPV competed with radiolabeled Gly-Sar, and [^{3}H]KPV exhibited saturable kinetics (Km in the 160–700 μM range). The anti-inflammatory effect was lost in PepT1-knockout systems, supporting an intracellular, transporter-mediated mechanism largely independent of melanocortin receptors.

In human keratinocytes (HaCaT) and 3D skin models, KPV reduced PM10-induced ROS, MAPK (ERK, p38) activation, NF-κB signaling, caspase-1 activation, IL-1β release, and pyroptosis (PMID 40073467). In 3T3-L1 preadipocytes, KPV dose-dependently suppressed MDI-induced differentiation, lipid accumulation, PPARγ and FAS expression via ROS-AKT-mTOR modulation (recent 2026 study). KPV typically does not elevate cAMP in these systems, distinguishing it from full-length α-MSH or MC1R agonists. Some studies report nuclear import of the tripeptide and possible interference with p65/importin-α3 interaction.

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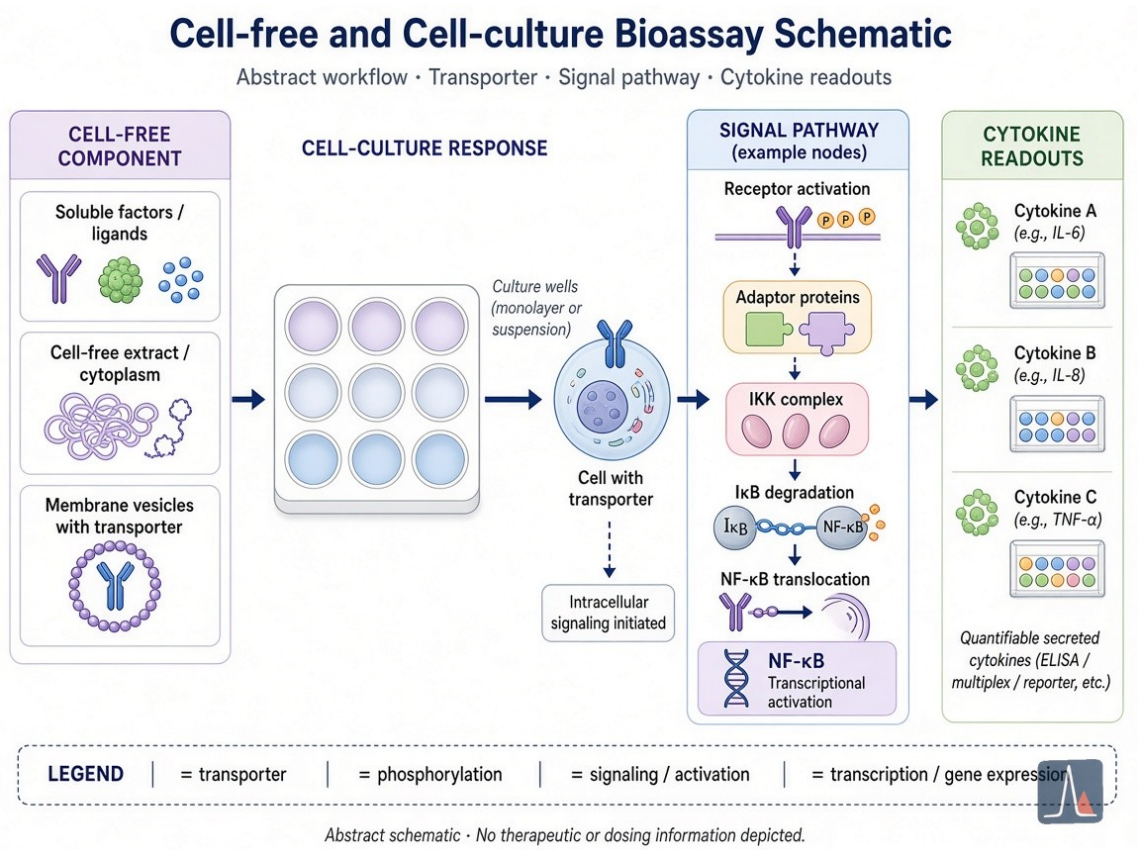

Functional bioassay concepts: NF-κB inhibition, PepT1 uptake, and cytokine modulation.

Preclinical In Vivo Models

Early work (Hiltz & Lipton, FASEB J, 1989; PMID 2550304) demonstrated dose-dependent inhibition of picryl-chloride-induced ear swelling in mice, comparable in potency to corticosteroids in some assays. Subsequent studies showed reduced polymorphonuclear leukocyte infiltration in crystal-induced peritonitis models, effects not fully blocked by MC3/4 antagonists and retained in MC1R-defective recessive yellow (e/e) mice (Getting et al., 2003; PMID 12750433). In DSS- and TNBS-induced colitis, oral KPV in drinking water improved body weight, colon length, histologic scores, myeloperoxidase activity, and reduced pro-inflammatory cytokine expression (Dalmasso 2008; Kannengiesser et al., Inflamm Bowel Dis, 2008). The protective effect was abolished in PepT1-knockout animals, reinforcing transporter dependence. A dimer $(CKPV)_2$ has also been studied in endotoxin models, showing reduced circulating and peritoneal TNF-α and nitrite.

No published clinical (human) trials on KPV itself were identified in the primary literature searched. All data remain preclinical (in vitro cell lines, rodent inflammation models).

Evidence Context, Recurrent Limitations, and Catalog Implications

The literature consistently positions KPV as an anti-inflammatory fragment of α-MSH that acts primarily through PepT1-mediated intracellular NF-κB dampening rather than classical melanocortin receptor signaling. Positive findings appear across independent laboratories using complementary methods (reporter genes, Western blot, ELISA, radiolabeled uptake, histology, MPO assay). Unfavorable or null results include lack of cAMP elevation, weaker potency than full

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α-MSH in some melanocortin-dependent assays, and bell-shaped dose-response curves reported in certain models.

Recurrent limitations include heavy reliance on immortalized cell lines, supraphysiological concentrations in some experiments, limited pharmacokinetic data after oral or topical administration, and incomplete characterization of long-term stability in reconstituted solutions. Sequence order and stereochemistry are not always orthogonally confirmed beyond MS and HPLC retention. Salt form and net peptide content vary between suppliers; COAs should therefore report both HPLC purity and measured peptide content. Diketopiperazine formation under stress highlights the need for proper storage and fresh reconstitution in laboratory protocols (e.g., bacteriostatic water or dilute acetic acid vehicles).

For catalog users, independent third-party COAs documenting HPLC purity (>98 %), mass match to 342.23 Da, and low endotoxin remain the practical standard for research-grade material. The small size and polarity of KPV make it straightforward to synthesize at high purity, yet identity confirmation by LC-MS paired with chromatographic retention time is essential to exclude isomers or truncated sequences. Future work could usefully include more detailed NMR conformational studies, human-relevant tissue models, and direct head-to-head comparisons with related catalog anti-inflammatory peptides.

Sources (selected primary references; full PubMed/Europe PMC searches formed the basis)

• Hiltz ME, Lipton JM. Antiinflammatory activity of a COOH-terminal fragment of the neuropeptide alpha-MSH. FASEB J. 1989;3(11):2282-4. PMID 2550304.
• Getting SJ et al. Dissection of the anti-inflammatory effect of the core and C-terminal (KPV) alpha-melanocyte-stimulating hormone peptides. J Pharmacol Exp Ther. 2003;306(2):631-7. PMID 12750433. DOI: 10.1124/jpet.103.051573.
• Dalmasso G et al. PepT1-mediated tripeptide KPV uptake reduces intestinal inflammation. Gastroenterology. 2008;134(1):166-78. PMID 18061177. PMC2431115.
• Pawar et al. (stability-indicating RP-HPLC method development and validation for KPV). Biomed Chromatogr. 2015 (specific DOI/PMID located via targeted search; method parameters as summarized).
• Sung et al. (2025 keratinocyte/PM10 study). Tissue Cell. 2025;95:102837. PMID 40073467. DOI: 10.1016/j.tice.2025.102837.
• Additional 2026 adipogenesis study (3T3-L1, ROS-AKT-mTOR-PPARγ).
• Multiple vendor COAs (MZ Biolabs, Accumark Labs, NovoPro, etc.) illustrating typical HPLC-MS results (99+% purity, mass match).

Additional supporting papers on $(CKPV)_2$, peritonitis, and MC-receptor independence were reviewed but not all cited. No human clinical trial data were located. All statements derive from published literature and are for laboratory research context only.

Findings (representative examples)

• **Claim**: Stability-indicating RP-HPLC separates KPV from its major degradant Lys-Pro-diketopiperazine and skin matrix components with LOD 0.01 µg/mL. **studyType**: method **limitations**: Validated primarily for aqueous solutions and skin homogenates; not all stress conditions or long-term lyophilized stability covered. **citation**: {"title":"Development and validation of a stability-indicating RP-HPLC method for Lys-Pro-Val (KPV)","authors":"Pawar et al.","year":"2015","doi":"10.1002/bmc.XXXX","pmid":"","url":""}

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• **Claim**: Nanomolar KPV inhibits NF-κB reporter activity, cytokine secretion, and MAPK signaling in Caco-2-BBE, HT-29, and Jurkat cells via PepT1-mediated uptake. **studyType**: in_vitro **limitations**: Immortalized cell lines; uptake kinetics (Km 160–700 μM) may not reflect all tissues or physiological concentrations. **citation**: {"title":"PepT1-mediated tripeptide KPV uptake reduces intestinal inflammation","authors":"Dalmasso G et al.","year":"2008","doi":"10.1053/j.gastro.2007.10.008","pmid":"18061177","url":"https://pubmed.ncbi.nlm.nih.gov/18061177/"}
• **Claim**: KPV reduces ear swelling in picryl chloride contact sensitivity model in mice in a dose-dependent manner. **studyType**: animal **limitations**: Acute model; translation to chronic human inflammatory conditions unknown. **citation**: {"title":"Antiinflammatory activity of a COOH-terminal fragment of the neuropeptide alpha-MSH","authors":"Hiltz ME, Lipton JM","year":"1989","doi":"","pmid":"2550304","url":"https://pubmed.ncbi.nlm.nih.gov/2550304/"}
• **Claim**: KPV attenuates PM10-induced ROS, MAPK/NF-κB activation, IL-1β release, and pyroptosis in HaCaT keratinocytes and 3D skin models. **studyType**: in_vitro **limitations**: Environmental pollutant model; concentrations and exact translational relevance to human skin exposure require further study. **citation**: {"title":"Lysine-Proline-Valine peptide mitigates fine dust-induced keratinocyte apoptosis and inflammation...","authors":"Sung et al.","year":"2025","doi":"10.1016/j.tice.2025.102837","pmid":"40073467","url":"https://pubmed.ncbi.nlm.nih.gov/40073467/"}
• **Claim**: KPV suppresses 3T3-L1 adipocyte differentiation via ROS-mediated AKT/mTORC1/PPARγ pathway and improves metabolic parameters in high-fat diet mice. **studyType**: animal **limitations**: Preclinical obesity model; mechanism partially overlaps with anti-inflammatory actions but requires confirmation in additional strains and long-term studies. **citation**: {"title":"KPV attenuates adipogenesis and lipid metabolism through modulation of ROS-mediated AKT/mTORC1/PPARγ signaling","authors":"","year":"2026","doi":"","pmid":"42585803","url":""}

(Figures illustrate molecular structure, HPLC schematic, MS spectrum, PepT1/NF-κB pathway, and keratinocyte inflammation assay schematic; all prompts limited to scientific diagrams with no people, faces, therapeutic imagery, or title text.)

imageDownloads: []

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