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Deep research on
GHK-Cu

Executive summary

GHK-Cu (commonly marketed as **Copper Tripeptide-1**) is a copper(II) complex of the endogenous tripeptide **glycyl-L-histidyl-L-lysine (GHK)** that is present in human biofluids and declines with age. [1] Its biological rationale centers on (i) **high-affinity copper binding and “buffered” copper delivery**, (ii) **extracellular-matrix (ECM) remodeling** (collagen, elastin, glycosaminoglycans; MMP/TIMP balance), and (iii) **anti-inflammatory/antioxidant signaling** (e.g., NF-κB/MAPK suppression; Nrf2/HO-1 activation in disease models). [2]

The evidence base is **mechanistically rich but clinically thin**. The strongest human anti-wrinkle data located in primary sources includes (a) an **8-week randomized, double-blind facial trial (n=40 women)** using a **lipid nano-carrier GHK-Cu serum**, reporting statistically significant reductions in wrinkle volume and depth versus controls; however, formulation concentration details and independent replication are limited. [3] A separate **randomized trial in CO₂-laser resurfaced skin (n=13 completers)** found **no objective improvement** in erythema, wrinkles, or overall skin quality compared with control regimens, but **higher patient-reported improvement** in overall skin quality (P=.04). [4]

Pharmacokinetics (PK) are best described as **route- and formulation-dependent topical exposure with uncertain systemic relevance**. A key quantitative ex vivo human skin study applying **0.68% aqueous copper as a GHK tripeptide complex** reported measurable copper flux and substantial retention (“depot”) across skin strata over 48 hours, suggesting the complex can deliver copper through and into skin under diffusion-cell conditions. [5] Still, **robust human systemic PK (plasma GHK/GHK-Cu levels post-topical, half-life, distribution)** is largely absent in the open primary literature reviewed here, and claims about crossing the blood–brain barrier remain speculative. [6]

Regulatory reality: GHK-Cu is widely used as a **cosmetic ingredient**, while related copper-peptide forms have also appeared as components in **wound-care medical devices**. For example, a wound dressing device listing shows a **510(k) “substantially equivalent” decision** for a hydrogel dressing that includes **prezotide copper acetate** (a

copper-peptide salt form) among components; the device documentation also includes explicit labeling/claim limitations. [7]

Key gaps: independent replication of clinical anti-wrinkle effects; dose–response and comparative effectiveness versus standard actives; long-term safety; systemic exposure in real-world topical use; and validated PK/PD biomarkers tying copper-peptide delivery to measurable ECM remodeling and inflammatory/oxidative endpoints. [8]

Chemical identity and structure

Core identities and nomenclature

GHK-Cu is most commonly encountered in consumer labeling as **Copper Tripeptide-1** (INCI). Supplier and regulatory/registry contexts may also use names like **prezotide copper** (or salts such as **prezotide copper acetate**) and “copper peptide.” [9]

A critical analytical nuance is that “GHK-Cu” may refer to **different salt/counterion forms** (e.g., acetate/diacetate), which changes the reported **molecular formula and molecular weight** without changing the underlying **GHK:Cu coordination motif**. This matters for identity testing, dosing calculations, and cross-study comparability. [10]

Coordination chemistry and redox implications

Multiple structural studies and reviews converge on a coordination description in which **Cu(II)** binds via the **histidine imidazole nitrogen**, the **N-terminus amine**, and the **deprotonated amide** of the Gly–His bond; additional coordination can occur through neighboring carboxylates in solution. [11] A recurring mechanistic claim is that copper’s **redox activity is “silenced” (attenuated)** when complexed with GHK, supporting the concept of **non-toxic copper delivery** relative to unliganded copper ions. [11]

Identifier table

Item	Free tripeptide (GHK / Tripeptide-1)	Copper complex used in cosmetics	
		(Copper Tripeptide-1 / GHK-Cu)	Copper-peptide salt form used in a penetration study (example)
Typical naming	“GHK”, “glycyl-L-histidyl-L-lysine” [12]	“Copper Tripeptide-1”, “GHK-Cu” [13]	“GHK-Cu (Ac) ₂ ” / glycyl-L-histidyl-L-lysine cuprate diacetate [5]
Molecular weight (illustrative)	~340.38 g/mol [14]	commonly reported ~401.9 g/mol (varies with salt/registry conventions) [15]	salt form includes acetate; formula reported explicitly in-study [5]

		Copper complex used in cosmetics	
Item	Free tripeptide (GHK / Tripeptide-1)	(Copper Tripeptide-1 / GHK-Cu)	Copper-peptide salt form used in a penetration study (example)
Key distinction	no copper	copper coordinated to GHK	same coordination motif but explicit counterions in tested material

Synthesis, formulation, and stability

Synthesis route

Publicly described manufacturing is consistent with standard peptide production:

1. **Peptide synthesis** of GHK (typically via solid-phase peptide synthesis in industrial practice), followed by purification (commonly HPLC-based in research-grade contexts).
2. **Chelation/complexation** with Cu(II) under controlled pH/stoichiometry to achieve a defined **GHK:Cu molar ratio**.
3. **Conversion to a desired formulation form** (e.g., lyophilized powder; aqueous/glycerin concentrate; encapsulated delivery system). [16]

In supplier specifications, the **GHK:Cu ratio** is sometimes explicitly controlled near ~1:1. [17]

Formulation patterns and “use levels”

GHK-Cu is **hydrophilic**, creating delivery challenges through the stratum corneum; this drives the use of **nanocarriers/lipid systems**, liposomes/niosomes, and “cool-down phase” processing in cosmetics manufacturing. [18]

Commercial and supplier documentation shows **two parallel concentration paradigms**:

- **Consumer-facing “1%” branding** used by some high-profile products.
- **Formulator use-level guidance** (often far lower in final formulations) in ppm-to-0.1% ranges, with the caveat that concentrates may be much higher and then diluted. [19]

Stability and compatibility

A preformulation study (abstract-accessible) reports that GHK-Cu is susceptible to **hydrolytic cleavage under basic and oxidative stress**, more modestly under acidic stress, while remaining **stable in water and pH 4.5–7.4 buffers for at least two weeks at 60 °C**; it also reports very negative logD values (high hydrophilicity) and compatibility differences across carrier components (e.g., Span 60 systems). [20]

A product-level example reports a **pH 6–7 range** for a copper-peptide serum and explicitly advises avoiding combination with several “direct actives,” reflecting practical compatibility concerns (whether due to irritation risk, copper complex stability, or ingredient interactions). [21]

Formulation and storage specifications from technical sheets

Source/form	Concentration format	Key specs (selected)	Storage / shelf life
A supplier TDS “Copper Tripeptide-1 – 5000 ppm” (aqueous/glycerin system)	5000 ppm = 0.5% active peptide in the supplied liquid [22]	Described as blue liquid; includes multi-ingredient base and states typical formulation concentrations on the order of 0.001%–0.1% in finished cosmetics (guidance). [23]	Noted in TDS context; typical storage guidance is cool/dry and protected from heat/light. [22]
ELEMENTAL SRL[24] “GHK Copper Tripeptide-1 concentrate solution”	“50000 ppm” concentrate; composition indicates 4–6% Copper Tripeptide-1 in a glycerin/water base (batch/spec dependent) [17]	pH 5.0–7.0; Cu ²⁺ content 10–16% and GHK content 64–80% per dry matter; micro limits (TAMC/TYMC; specified pathogens absent). [17]	Shelf life 24 months; store closed, cool, dry, protected from humidity (per TDS). [17]

Pharmacodynamics and molecular mechanisms

ECM remodeling and dermal biology

Animal wound model evidence (primary): In a rat wound-chamber model, sequential injections of GHK-Cu produced a **concentration-dependent increase** in dry weight, DNA, total protein, collagen, and glycosaminoglycans in wound chambers; collagen stimulation was proportionally greater than non-collagen proteins; **type I and III collagen mRNAs increased while TGF- β mRNA did not**, and a control tripeptide lacked significant effects. [25]

Human fibroblast mechanistic evidence (primary): GHK-Cu has been shown to accelerate growth of normal and irradiated fibroblasts in vitro and to increase early production of growth factors like **bFGF** and **VEGF** in a serum-free model using fibroblasts derived from radiated tissue. [26]

MMP/TIMP balance and matrix biomarkers (primary): In human adult dermal fibroblasts, GHK-Cu exposure altered expression patterns of MMPs and TIMPs, and increased

collagen and elastin production; the associated clinical component used wrinkle depth/volume endpoints to translate these matrix effects to facial appearance. [3]

Antioxidant and inflammatory signaling

NF- κ B / MAPK suppression (primary disease model): In a murine LPS-induced acute lung injury model and macrophages, GHK-Cu reduced ROS, increased SOD activity, and reduced TNF- α /IL-6 with suppression of **NF- κ B p65** activity and **p38 MAPK** signaling. [27]

Nrf2/HO-1 axis and cigarette-smoke emphysema model (primary): In a cigarette-smoke emphysema model and A549 cells, GHK-Cu is reported to attenuate oxidative stress markers (e.g., GSH/T-AOC/MDA patterns), with mechanistic data emphasizing **Nrf2 nuclear translocation** and **HO-1** induction, alongside reduced inflammatory signaling (including NF- κ B-related markers). [28]

Copper homeostasis and “buffered” copper delivery

The mechanistic premise that GHK binds copper with high affinity, can exchange copper with albumin and related carriers, and may reduce copper redox cycling underpins many of its proposed tissue-protective effects. [11] This may matter specifically in contexts where **free metal-driven oxidative stress** is implicated (wounds, inflammatory tissue damage, possibly neurodegeneration). [29]

Mechanism map (conceptual)

flowchart TD

A[Topical or local application] --> B[Delivery through/into skin depends on carrier]

B --> C[GHK-Cu reaches epidermis/dermis microenvironment]

C --> D[Copper binding + exchange with local ligands]

D --> E[Fibroblast activation + growth factor modulation]

E --> F[ECM remodeling: collagen, elastin, GAGs; MMP/TIMP balance]

D --> G[Redox/inflammation modulation]

G --> H[NF- κ B & MAPK downshift; Nrf2/HO-1 upshift (model-dependent)]

F --> I[Clinical correlates: wrinkle metrics, skin density/elasticity]

H --> I

Pharmacokinetics and biodistribution

Topical absorption and skin retention: quantitative ex vivo human data

A pivotal quantitative study evaluated copper delivery when applied as a copper–GHK tripeptide complex (reported as a diacetate cuprate form) to ex vivo human skin using flow-through diffusion cells under “infinite dose” conditions. [5] Key details:

- **Dose/formulation:** 0.68% aqueous copper as the tripeptide complex. [5]
- **Tissues:** isolated stratum corneum, heat-separated epidermis, and dermatomed skin. [5]

- **Quantification:** copper measured via ICP-MS in receptor fluid and tissues over 48 hours. [5]

Results (dermatomed skin): - Permeability coefficient $K_p \approx 2.43 \pm 0.51 \times 10^{-4} \text{ cm/h}$, with $\sim 136.2 \pm 17.5 \text{ } \mu\text{g/cm}^2$ copper permeated into receptor fluid over 48 hours and $\sim 82 \pm 8.1 \text{ } \mu\text{g/cm}^2$ retained in tissue as a depot (values reported in the abstract/table set). [5]

Interpretation caveats: - These are **ex vivo diffusion-cell** results and may over- or under-estimate real-world exposure due to differences in occlusion, metabolism, desquamation, and formulation. The authors discuss reservoir formation and possible systemic availability from stratum corneum retention depending on lag time vs turnover assumptions. [5]

Formulation dependence and dermal delivery challenges

A randomized clinical trial and supporting discussion emphasize that GHK-Cu is **highly hydrophilic**, making efficient trans-epidermal delivery challenging, motivating the use of **lipid-based nanocarriers** to improve dermal delivery. [30]

Systemic PK and metabolism: what is (and is not) known

- **Systemic PK in humans (half-life, clearance, bioavailability after topical):** not well-characterized in openly accessible primary sources reviewed here. [31]
- **Tissue distribution data:** a related open-access paper on GHK biology includes a mouse tissue “uptake” figure for GHK (not necessarily copper-complexed) and discusses copper exchange and binding in vivo contexts; however, these do not provide a modern human PK profile. [32]
- **Blood–brain barrier penetration:** proposed as possible but not established; available reviews frame this as uncertain. [6]

Clinical and commercial evidence

Key human studies and what they do (and don't) show

Study	Design and population	Intervention (publicly described)	Outcomes	Key results	Limitations
Badenhorst et al. 2016 [3]	Randomized, double-blind facial trial; n=40 women , age 40–65; 8 weeks, twice daily [3]	GHK-Cu in lipid nano-carrier serum vs vehicle serum and a commercial comparato	Wrinkle depth & volume; plus in vitro MMP/TIMP and collagen/elastin data [3]	Wrinkle volume ↓ 31.6% vs Matrixyl 3000 (p=0.004); volume ↓ 55.8% vs vehicle (p<0.001);	Concentration of GHK-Cu in the final serum not clearly specified in the abstracted sections; potential sponsor involvement; needs

Study	Design and population	Intervention (publicly described)	Outcomes	Key results	Limitations
		containing Matrixyl 3000 [3]		depth ↓ 32.8% vs vehicle (p=0.012). [3]	independent replication and standardized metrology reporting. [3]
Miller et al. 2006 [4]	Randomized trial in circumoral CO ₂ laser resurfacing; n=13 completers [4]	Post-treatment regimen with vs without GHK-Cu-containing skincare [4]	Erythema resolution; wrinkles; overall skin appearance; patient questionnaire [4]	No statistically significant objective differences; patient questionnaire showed higher improvement in overall skin quality with GHK-Cu regimen (P=.04). [4]	Very small sample; post-procedure setting; product composition/dose not fully described in the abstract; subjective endpoint signal without objective corroboration. [4]

Evidence for dermal exposure as a plausibility bridge

A quantitative ex vivo diffusion-cell study provides a mechanistic bridge showing that copper delivered as a GHK tripeptide complex can penetrate human skin layers and form tissue depots under laboratory conditions. [5] This does not prove clinical efficacy, but it supports the plausibility of “more than surface-only” exposure depending on formulation and usage conditions.

Commercial concentration claims and real-world regimens

A practical reality is that most cosmetics do **not disclose** copper peptide concentration on-label. Two prominent exceptions are products that embed concentrations in product positioning:

- The Ordinary[33] describes “Multi-Peptide + Copper Peptides 1%” as applied **morning and evening** (“a few drops”), with warnings to use only on unbroken skin and to avoid combination with several direct actives (e.g., direct acids, direct vitamin C, retinoids, strong antioxidants). [34]

- NIOD[35] describes “Copper Amino Isolate Serum 3 1:1 (CAIS3)” as containing **1% GHK-Cu** and **1% GHK**, used **daily in AM and PM** as the first step after cleansing, with similar “do not use with” restrictions. [36]
- A brand blog describing NIOD development reports a **clinical study of 32 subjects** applying the formulation **2×/day for 8 weeks**, claiming significant improvement in tone evenness (4 and 8 weeks) and elasticity (4 weeks), though full protocols/endpoints are not provided in that page. [37]

Cosmetic “use level” table (formulator-facing)

Context	Concentration basis	Notes
Supplier guidance (example)	Typical finished-formula use levels often cited around 0.001%–0.1% , with higher concentrations positioned for professional serums [38]	“Concentrate” raw materials may be far higher and diluted during formulation; compare concentrate composition/specs versus finished product marketing. [39]
Consumer “high concentration” products	1% copper peptides (and sometimes separate 1% GHK) [40]	Concentration naming doesn’t guarantee bioavailability; delivery system and skin tolerance matter. [8]

Development timeline (selected milestones)

timeline

1973 : GHK activity identified from human albumin/plasma (early discovery narrative in later reviews)

1993 : Rat wound-chamber model shows dose-dependent ECM accumulation with GHK-Cu

2005 : In vitro irradiated fibroblast model shows growth-factor changes with 1e-9 M GHK-Cu

2006 : Small RCT in CO2 laser-resurfaced skin shows no objective benefit; subjective signal

2010 : Ex vivo human skin study quantifies copper tripeptide penetration and retention

2016 : RCT (n=40) reports wrinkle volume/depth reductions with nano-carrier GHK-Cu serum

2022 : COPD/emphysema models report Nrf2/HO-1 and NF-kB pathway modulation by GHK-Cu

Safety, regulatory status, quality control, and research priorities

Safety and adverse effects

Human trial reporting (limited but informative) suggests generally good topical tolerability:

- In the 8-week nano-carrier facial trial, the authors report the formulation was “well tolerated,” with **one participant reacting** to both the nano-carrier product and the

comparator, and they contrast this with common tretinoin-type irritant effects not observed in their participants. [3]

- In the CO₂ laser trial, the abstract does not highlight major adverse effects and emphasizes lack of objective benefit but a patient-reported improvement difference. [4]

Because GHK-Cu is a **copper-containing** complex, systemic misuse (e.g., injection/ingestion of non-pharmaceutical material) raises theoretical concerns related to copper toxicity and contamination; however, systematic long-term safety data for systemic administration in humans is not established in the sources reviewed here. [31]

Practical compatibility/sensitivity considerations show up in consumer guidance: - “Use only as directed on unbroken skin,” patch testing advice, and multiple ingredient-category incompatibilities are explicitly stated on a major product page. [21]

Regulatory status and availability

- **Cosmetics:** Supplier documentation frames Copper Tripeptide-1 as a cosmetic active ingredient and states compliance-positioning with **EU cosmetics regulation requirements** in a technical sheet context. [17]
- **United States medical device context:** U.S. Food and Drug Administration[41] device database records a **510(k)** for “LAMIN-2 HYDRATING GEL” with a “substantially equivalent” decision. [42] The associated document imagery describes the hydrogel dressing as a preserved preparation of water, glycerin, carbomer, **prezotide copper acetate**, and parabens, and lists intended-use categories (e.g., pressure ulcers and burns) while also including explicit labeling limitations (e.g., not labeled for third-degree burns; not labeled as accelerating wound healing/epithelialization). [43]

Interpretation: cosmetic availability does not imply drug-like evidentiary standards, while device clearances relate to **device intended use** and labeling constraints, not broad therapeutic claims for the ingredient per se. [44]

Recommended minimum quality-control specifications for research-grade GHK-Cu

Below is a conservative QC “floor” intended for **research materials** (especially when used in human studies or ex vivo tissues). It blends common peptide QA practice with supplier-style parameters explicitly shown in technical documentation.

Identity - Peptide identity confirmed by **LC–MS** (exact mass / isotope pattern consistent with copper complex) and/or MS/MS mapping; verify presence of copper adduct(s).

(Rationale: copper-peptide species may exist as multiple stoichiometries; confirm what you are dosing.) [45]

- Orthogonal identity: **amino acid analysis** or peptide hydrolysis profile consistent with Gly/His/Lys ratios (some suppliers specify amino acid analysis windows). [17]

- Copper content / stoichiometry by **ICP-MS** or AAS, with explicit target for **GHK:Cu molar ratio** near 1:1 where applicable. [46]

Purity and impurities - Analytical HPLC purity target (commonly $\geq 95\%$ for research peptides; higher for clinical-grade).

- Screen for free copper, residual counterions (acetate/chloride), and degradation products (stress testing is justified given reported susceptibility under oxidative/basic conditions). [47]

Microbiology - For non-sterile topical research: microbial limits similar to supplier targets (e.g., total aerobic microbial count and yeast/mold limits; absence of specified pathogens) can be adopted as baseline. [17]

- For any invasive use (not recommended without full GMP): sterility testing and endotoxin testing (LAL) become essential.

Stability - Real-time and accelerated stability with monitoring of: HPLC profile, copper content, pH, and color shift (blue hue changes can indicate copper state/speciation), with attention to oxidation/basic stress sensitivity. [48]

Prioritized research gaps and questions

1. **Dose-response and exposure-response:** What dermal concentration and exposure duration are required to move ECM biomarkers (pro-collagen I, elastin, decorin; MMP/TIMP ratios) in vivo in humans? [49]
2. **Real-world dermal PK:** How much intact GHK-Cu (and/or copper transferred from it) reaches viable epidermis/dermis with modern formulations (nano-carriers, liposomes) under real consumer use patterns? [8]
3. **Long-term safety:** Does chronic daily use affect copper homeostasis locally (skin) or systemically in meaningful ways? Are there subgroups at risk (barrier impairment, concurrent actives)? [50]
4. **Comparative effectiveness:** How does GHK-Cu compare to canonical topical anti-aging agents (retinoids, vitamin C derivatives, peptides without copper) using standardized imaging endpoints and blinded assessment? [51]
5. **Mechanistic specificity:** Which effects are driven by copper delivery vs peptide signaling vs downstream gene regulation? Can biomarkers discriminate these pathways? [52]

Practical guidance for clinicians/researchers designing trials

Trial archetype: topical GHK-Cu for periorbital wrinkle reduction - **Design:** randomized, double-blind, split-face (paired) to reduce inter-subject variability; stratify by baseline wrinkle severity and age. (Split-face logic is aligned with prior facial trials.) [3]

- **Dosing arms:** choose at least two concentrations or delivery systems (e.g., standard aqueous serum vs lipid nano-carrier) because hydrophilicity makes formulation a major determinant of exposure. [30]

- **Duration:** ≥ 8 –12 weeks to match prior studies and skin remodeling timelines. [53]

- **Primary endpoints:** instrumented 3D profilometry (wrinkle depth/volume), standardized

photography with blinded grading, and patient-reported outcomes. (Wrinkle depth/volume endpoints were used in prior work.) [51]

- **Secondary endpoints:** cutometer elasticity, high-frequency ultrasound or OCT dermal thickness, transepidermal water loss, and standardized irritation scoring. [54]

- **Biomarkers (PK/PD bridge):** - Tape-strip or micro-biopsy: MMP1/MMP2, TIMP1/TIMP2, pro-collagen I markers. [3]

- Oxidative/inflammatory: Nrf2/HO-1–related proteins (if feasible), local cytokine profiles; copper quantification in stratum corneum/epidermis by ICP-MS to link delivery to response. [55]

- **Safety monitoring:** standardized dermatitis scoring, adverse-event diary, and (for higher-exposure designs like patches) basic systemic copper homeostasis panels (serum copper, ceruloplasmin) to pre-empt unintended systemic effects. [56]

Suggested sample-size calculations (illustrative, conservative assumptions)

For a split-face paired design with a continuous primary endpoint (e.g., % change in wrinkle volume by 3D profilometry), a standard approximation is:

$$\bullet \quad n \approx (Z\alpha/2 + Z\beta)^2 \times \sigma d^2 / \Delta^2$$

Where σd is the SD of within-subject differences and Δ is the mean difference you want to detect.

Example scenarios (two-sided $\alpha=0.05$, 80% power $\rightarrow Z\alpha/2=1.96$, $Z\beta=0.84$): - If $\Delta = 10$ percentage points and $\sigma d = 20$ points $\rightarrow n \approx (2.8)^2 \times 400/100 \approx 31.4 \rightarrow \sim 32$ participants. - If $\Delta = 8$ points and $\sigma d = 20$ points $\rightarrow n \approx (2.8)^2 \times 400/64 \approx 49 \rightarrow \sim 50$ participants.

These are intentionally more conservative than the large effect sizes reported in one nano-carrier trial, recognizing that early studies can overestimate effects and that formulation differences strongly influence exposure. [8]

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