

Tranexamic Acid

Tranexamic Acid (abbreviated as TXA, also known as trans-4-(aminomethyl)cyclohexanecarboxylic acid) is a synthetic lysine derivative. Originally developed as a medical antifibrinolytic hemostatic drug, it was later confirmed by dermatological research to deliver outstanding spot-fading, anti-inflammatory and redness-relieving benefits. Unlike brightening actives such as vitamin C and arbutin that directly inhibit tyrosinase, tranexamic acid blocks upstream melanin-activating signals induced by inflammation and ultraviolet radiation. It integrates spot fading, anti-inflammation, redness alleviation and skin repair. Characterized by mildness and low irritation, it serves as a core functional ingredient for melasma, post-inflammatory hyperpigmentation (PIH) and erythematous acne marks, and is widely incorporated into brightening formulations for sensitive-skin and post-aesthetic-procedure repair.

Product Information

Trade Name	Tranexamic Acid
INCI Name	TRANEXAMIC ACID
CAS Number	1197-18-8
EC Number	214-818-2
Appearance	White crystalline powder
Purity	≥99%
Solubility	Freely soluble in water and glacial acetic acid; practically insoluble in acetone and 96% ethanol
Recommended Dosage & Formulation pH	Serums / spot-fading stock solutions: 2%-5%; Lotions / creams / masks: 0.5%-2%; Sunscreens / after-sun repair products: 0.5%-1.5% pH range: 4.0-7.0
Storage	Keep in hermetically sealed original containers; store in cool, dry, dark ambient environment (10-25 °C) with moisture protection. Shelf-life of unopened original packaging: 36 months
Regulatory Compliance	Listed in China Catalogue of Used Cosmetic Raw Materials; included in EU CosIng database; globally compliant raw material for cosmetic markets

Human Skin Safety and Tolerance

Occlusive patch test: 48- hour occlusive patch test performed on 30 healthy subjects. No irritant reactions including erythema, edema or stinging were observed at concentrations of 2%, 3% and 5%. No delayed adverse responses in the 72- hour follow- up.

Abraded- skin irritation test: In damaged- skin model assays, the irritation potential of tranexamic acid shows no significant difference from the normal saline control group. It can be gently applied to abraded skin after aesthetic medical procedures.

Photopatch test: No photosensitization or phototoxicity upon UVA/UVB irradiation. No risk of rebound hyperpigmentation when used in daytime with sunscreen.

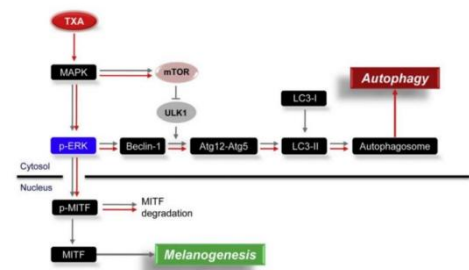
Repeated Open Application Test (ROAT) for sensitization: No contact sensitization potential with extremely low sensitization probability. Well- tolerated for sensitive skin and barrier- compromised skin.

Note: Extremely rare adverse cases are only reported for high- dose oral medical administration. No irritation or allergic events have been documented for topical cosmetic use at routine concentrations. It is safe for long- term application across all skin types.

Upstream Blocking of Melanin- Activating Signals (Distinct from Vitamin C Derivatives)

Ultraviolet radiation and inflammation activate conversion of keratinocyte plasminogen into plasmin, which releases arachidonic acid and prostaglandins to stimulate excessive melanin production in melanocytes. Tranexamic acid competitively binds to the lysine- binding site of plasmin, cutting off the pro- melanogenic signalling cascade at the source.

- TXA inhibits plasmin and blocks the p38/MAPK inflammatory pathway
- Down- regulates p- ERK and accelerates degradation of MITF, the master melanogenic transcription factor
- Inhibits the mTOR pathway and activates cellular autophagy (up- regulation of Beclin- 1/LC3- II) to degrade pre- formed melanosomes
- Synchronously down- regulates melanogenesis- related proteins including tyrosinase, TRP- 1 and TRP- 2



Assay on Inhibition of Melanogenesis- Related Protein Expression (B16F10 Melanoma Cells)

- **Test model** – Murine B16F10 melanoma cells treated with 1mg/mL TXA
- **Detection targets** – P38、ERK、MITF、Tyrosinase、TRP-1

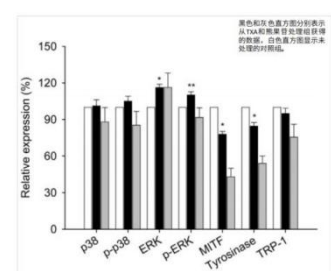
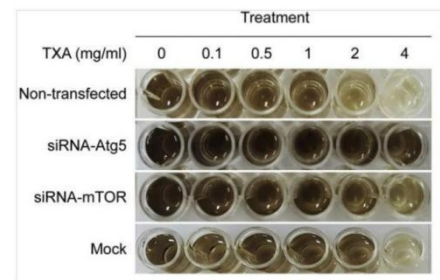


图.TXA对ERK1/2信号和黑素生成相关蛋白表达的影响

Expression levels of MITF and tyrosinase are significantly down-regulated (* $p < 0.05$, ** $p < 0.01$), directly attenuating melanin-synthesizing capacity. TXA reduces phosphorylated p-p38 expression and moderately elevates p-ERK; elevated p-ERK mediates MITF degradation.

Assay on Cellular Melanin Content Inhibition

- **Gradient concentrations** – 0/0.1/0.5/1/2/4mg/mLTXA
- **Controls** – Atg5 autophagy-gene knockdown group, mTOR-activated control group



Tranexamic Acid reduces cellular melanin content in a dose-dependent manner. The autophagy pathway constitutes a key mechanism for its depigmenting effect; brightening efficacy is substantially attenuated upon Atg5 gene knockdown.

Human Clinical Efficacy Study

- **Trial model** – 12-week clinical improvement trial for melasma (20 female subjects with moderate-to-severe melasma)

- **Test sample** – 3%

tranexamic acid brightening serum, applied full-

face. **Assessment instruments** – Mexameter melanin indexmeter, VISIA imaging system;

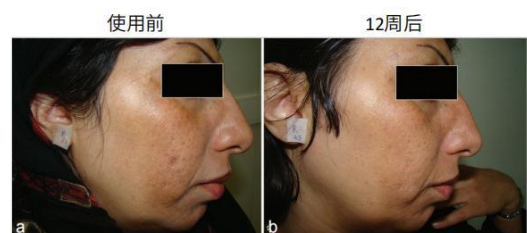


图.用氨甲环酸处理的一侧面部

Week 4: Improved brightness of pigmented spots and alleviated erythema;

Week 8: Significant reduction in melanin index ($p < 0.001$) and shrinkage of lesion area;

Week 12: 42 % decrease in MASI (Melasma Area and Severity Index), greatly enhanced skin tone evenness with 91 % subject satisfaction rate.

No adverse reactions such as irritation, dryness or rebound hyperpigmentation observed. Its efficacy is comparable to 2 % hydroquinone with markedly superior safety profile.

Complete Mechanism of Multifunctional Skincare Benefits

- **Block inflammatory pro-melanogenic pathways(core advantage)**

Inhibits plasmin generation and reduces prostaglandin and α -MSH pro-melanogenic signals. Effective for post-inflammatory hyperpigmentation, acne marks and melasma (associated with chronic low-grade inflammation).

- **Down-regulate key melanogenesis-related proteins**

Degrades MITF transcription factor, lowers expression of tyrosinase and TRP-1, and reduces total melanin synthesis within melanocytes.

- **Induce autophagy in melanocytes**

Up-regulates autophagy proteins Beclin-1 and LC3-II. Mature melanosomes are degraded via lysosomes to metabolize deposited melanin and fade established pigmented lesions.

- **Anti-inflammatory redness relief for post-inflammatory erythema (PIE)**

Inhibits mast cell degranulation and reduces release of inflammatory mediators including IL-6 and histamine. Suppresses VEGF-mediated angiogenesis, constricts dilated capillaries and ameliorates erythema, red acne marks and sensitive flushing.

- **Anti-photoaging & prevention of UV-induced hyperpigmentation**

Blocks UV-triggered plasmin activation and mitigates persistent photodamage-related pigmentation. Delivers long-term sun-spot prevention when combined with sunscreen products.

- **Mild barrier repair**

Mitigates stratum-corneum damage induced by inflammation and reduces transepidermal water loss (TEWL). Accelerates barrier restoration when combined with ceramides, suitable for post-aesthetic-procedure skin and seasonal sensitive phases.

Recommended Synergistic Ingredients for brightening and Skin-Tone-Evening Effects

Synergistic Ingredient	Mechanism of Action	Recommended Usage Concentration
3-O-Ethyl Ascorbic Acid	TXA blocks upstream inflammatory pro-melanogenic signals; EAA directly inhibits tyrosinase and reduces melanin. Dual-pathway potent spot-fading for melasma and stubborn hyperpigmentation	EAA 2% - 5% + TXA 2% - 3%
Niacinamide 2%-5%	TXA suppresses pro-melanogenic signalling; niacinamide blocks melanosome transfer to keratinocytes. Synchronized skin-tone-evening action for acne-mark-related dullness	Niacinamide 2% - 4% + TXA 2% - 3%
Vitamin E	Builds antioxidant recycling system, mitigates UV-triggered oxidative stress,	0.5% - 1%

	reduces photochemically induced hyperpigmentation and enhances formulation stability	
Ferulic Acid	Synergistic antioxidant effect, boosts photoprotective performance and stabilizes activity of vitamin-C derivatives	0.3% – 0.8%
Ceramide	Repairs skin barrier, mitigates irritation caused by high-concentration brightening actives and improves tolerance for	0.1% – 0.5%
Sodium Hyaluronate	Delivers long-lasting moisturization, alleviates drying effect of high-dose formulations and assists transdermal permeation	0.1% – 0.3%

Stable Anti-Inflammatory & Melanin-Suppressing Multifunctional Active Ingredient

Mild Upstream-Blocking Brightening & Repair Raw Material

- Transformed from a medical hemostatic agent into cosmetic raw material, with decades of dermatological clinical application history. Registered in cosmetic raw-material inventories worldwide. Non-cytotoxic; preferred option for subjects intolerant to hydroquinone and high-concentration vitamin C.
- Validated in multi-center clinical trials among Asian populations suffering from melasma and post-inflammatory hyperpigmentation. Dedicated ingredient for post-aesthetic-procedure repair and rebound-hyperpigmentation prevention; well-tolerated for long-term use by all skin types.

Skin Protection

- Competitively binds to the lysine-binding site of plasmin, inhibits plasmin activation, reduces release of inflammatory pro-melanogenic mediators such as prostaglandins and arachidonic acid, and blocks oxidative stress induced by UV radiation and inflammation.
- Inhibits VEGF-mediated abnormal capillary proliferation, alleviates dermal microvascular dilation and improves erythema, red acne marks and flushing.
- Suppresses mast-cell-related inflammatory pathways and lowers release of IL-6 and histamine. Soothes skin burning and sensitivity triggered by oxidation and aesthetic-procedure-related irritation.

Anti-Aging Efficacy

- Indirectly delays photo-aging-related sallowness and roughness by mitigating inflammatory destruction of dermal matrix.

Brightening & Skin-Tone-Evening

- Blocks upstream inflammatory / UV-triggered pro-melanogenic signals: inhibits keratinocyte plasminogen activation and interrupts the p38/MAPK inflammatory cascade.

- Down-regulates phosphorylated ERK to accelerate degradation of MITF, the master melanogenic transcription factor, and indirectly reduces expression of tyrosinase, TRP-1 and TRP-2; it does not bind directly to tyrosinase.
- Induces melanocyte autophagy (up-regulation of Beclin-1, LC3-II); mature melanosomes are degraded by lysosomes to metabolize pre-deposited melanin (cell-level conclusion).
- Lacks direct reductive activity towards dopaquinone, which differentiates it from vitamin-C derivatives. Combined with EAA or arbutin, it forms a dual-mode spot-fading system of “upstream signal blockade plus direct enzymatic inhibition”.
- Gentle and low-irritation. Gradually improves melasma, post-inflammatory hyperpigmentation, sun spots and uneven skin tone. Applicable for sensitive-skin and post-aesthetic-procedure use.

Key references

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