Review Article

Clitoria ternatea (Butterfly Pea Flower): A Multifunctional Phytocosmeceutical for Collagen Stimulation, Anti-Photoaging, and Skin Barrier Enhancement – A Comprehensive Review

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ABSTRACT

The processes leading to skin aging are cumulative, intrinsic, and extrinsic processes, which culminate in collagen degradation, the decline of fibroblast activity, and barrier dysfunction. The activation of MAPK, AP-1, and NF-KB signaling pathways caused by the presence of reactive oxygen species causes an increase in the activity of matrix metalloproteinases and the inhibition of TGF- 0 signaling, which causes progressive dermal degeneration. The review assesses the phytochemical composition and dermatological properties of *Clitoria ternatea* L. with a focus on collagen prevention, anti-photoaging, and barrier improvement. *Clitoria ternatea* has anthocyanins, flavonoids, phenolic acid, triterpenoids, and saponins, which exhibit antioxidant, anti-inflammatory, anti-collagenase, and fibroblast-stimulating properties. Preclinical evidence indicates that there are dramatic increases in collagen density, fibroblast proliferation, and TGF-B expression and simultaneous decreases in MMP activity and measures of oxidative stress. The plant has high potential as a multifunctional cosmetic giving ingredient. Nonetheless, the absence of human clinical trials is also one of the key limitations. The next step in the working direction should be standardisation, pharmacokinetics, and randomised controlled studies.

Keywords: *Clitoria ternatea*; butterfly pea flower; anthocyanins; collagen; photoaging; matrix metalloproteinases; TGF-β; antioxidant; cosmeceutical; skin barrier.

1. INTRODUCTION

Ageing of skin is a complicated biological process that is achieved by genetic programming and environmental exposure. Anti-ageing products are becoming more and more popular across the world, with the population of those preferring botanical actives tested scientifically over synthetic agents[1].

Dermal collagen is the structural bone of the skin, and it represents about 70-80 per cent of the dermal dry mass[2]. The breakdown of collagen starts at an early age in adulthood, with a rate of 1 per cent per year, which is estimated to increase with ultraviolet exposure[3]. This destruction is triggered mostly by matrix metalloproteinases that are triggered by oxidative stress and inflammatory signal pathways[4].

Telomere reduction, dysfunction of the mitochondrion, and decreasing activity of fibroblasts are typical of intrinsic ageing[5]. Ultraviolet radiation is the primary cause of extrinsic ageing, or photoaging, which is promoted by pollution and lifestyle characteristics that elevate reactive oxygen species[6]. Both of these pathways lead to the same end, but the transcription factors (AP-1 and NF-KB) are upregulated to increase the expression of MMP and inhibit the process of collagen synthesis[7].

The actives of cosmeceuticals like retinoids, vitamin C, and niacinamide are partial solutions and limited by their ability to be stable, irritate, or limited in scope of action[8]. This has led to a need for multifunctional botanical compounds that can act on a number of ageing pathways at the same time.

One of the new promising candidates is *Clitoria ternatea*, which is a tropical plant that is commonly used in traditional medicine. It has a high phytochemical profile that allows it to interact with various molecular targets that play a role in skin ageing[9]. The review presents a critical assessment of its use in dermatology in accordance with the existing scientific findings.

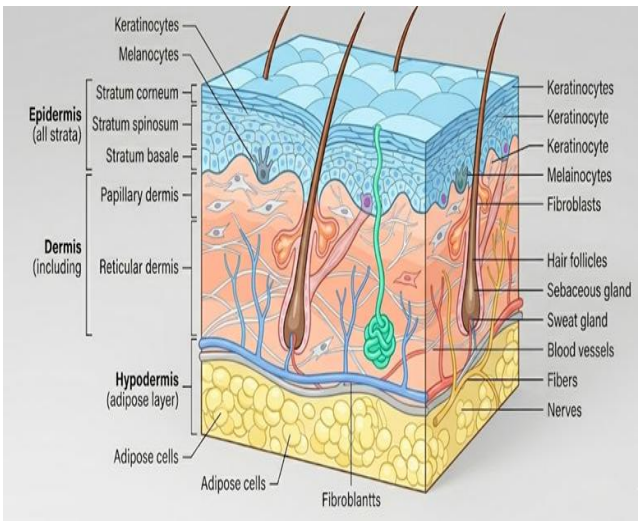

Figure 1: Structure of human skin showing epidermis, dermis, and hypodermis with major cellular components and extracellular matrix elements.

2. SKIN STRUCTURE AND PHYSIOLOGY

The skin of a human being is composed of three major layers, which include the epidermis, the dermis, and the hypodermis. Each of the layers uses structural integrity, functional protection, and each has its particular age-related changes (Fig.1)[10].

2.1. Epidermis

Epidermis is a stratified layer of epithelial cells that is made of keratinocytes. It serves as the major barrier to environmental damage and averts transepidermal loss of water[11].

The topmost layer is the stratum corneum, which is made up of corneocytes enclosed by a lipid matrix made out of ceramides, cholesterol, and fatty acids. This framework keeps the structure hydrated and intact with barriers[12].

Keratinocyte turnover reduces by up to 50 per cent in old age, resulting in a loss of renewal and barrier dysfunction[13]. The epidermal thickness reduces slowly, and the melanocyte density decreases, leading to a lack of homogenous pigmentation and ultraviolet radiation sensitivity[14].

2.2. Dermis

Mechanical strength and elasticity are given by the dermis. It is filled with fibroblasts, collagen fibres, elastin, as well as ground materials like hyaluronic acid[15].

Dermal matrix is dominated by collagen types I and III. The production and remodelling of these structural proteins is controlled by fibroblasts[16].

The results of ageing include fragmentation of collagen fibres, a decrease in the integrity of elastin, and loss of hyaluronic acid[17]. The effects of these changes are wrinkles, loss of elasticity, and loss of hydration[18].

2.3. Hypodermis

Hypodermis is primarily composed of adipose tissue and connective structures, which offer cushions and thermal protection[19].

As one ages, he/she experiences redistribution of fats and loss of volume, leading to the appearance of sags and hollows on the face[20]. Topical treatments do not affect this layer much, but preserving the dermal integrity can slow the appearance of changes related to the hypodermal atrophy.

3. MOLECULAR MECHANISMS OF SKIN AGING

Skin ageing is a multi-factorial event that is caused by oxidative stress, enzyme activation, and cellular signalling impairment (Fig.2)[21].

3.1. Intrinsic Aging

Biological processes are intrinsic to the ageing process. Cell division and senescence in fibroblasts are limited by the process of telomere shortening[22].

The senescence cells secrete inflammatory cytokines and matrix-destroying enzymes, which cause a microenvironment that speeds up the destruction of tissues[23].

The consequences of mitochondrial dysfunction are the enhancement of the production of reactive oxygen species, which subsequently damages the cellular components and triggers ageing-related signalling pathways[24].

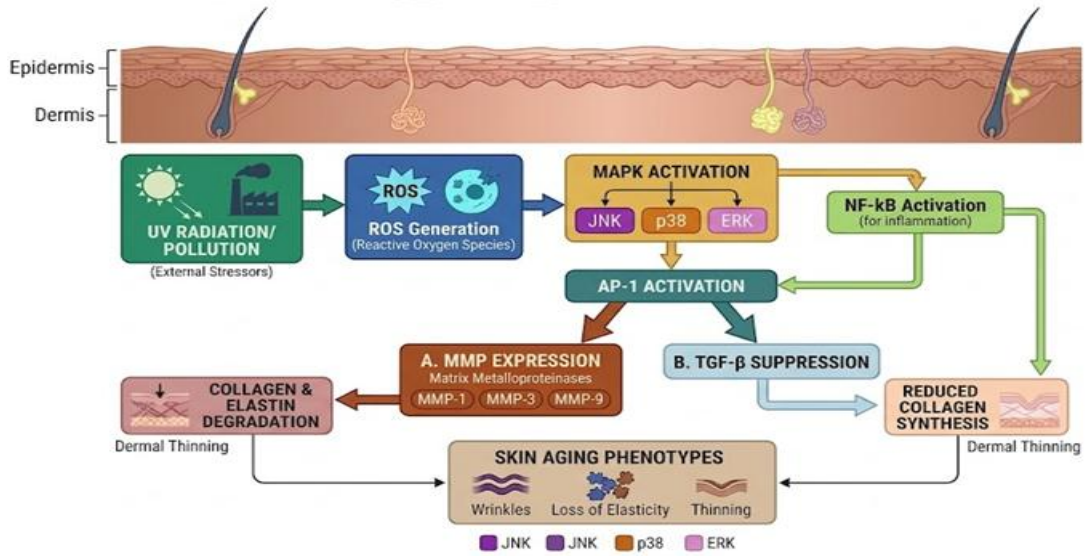

Figure 2: Molecular pathways of skin ageing showing ROS generation, MAPK activation, MMP expression, and TGF-β suppression.

3.2 Extrinsic Aging and Photoaging

Ultraviolet radiation is mostly the cause of extrinsic ageing. UV-B leads to the damage of DNA, and UV-A goes deeper into the dermis and produces reactive oxygen species[25].

These receptors result in the activation of MAPK pathways and transcription factors that enhance MMPs synthesis and collagen production, which is inhibited[26].

Oxidative stress is increased by environmental pollutants and lifestyle habits and leads to chronic inflammation in the skin[27].

3.3 Role of ROS, MMPS, and TGF-β

ROS serve as the leading precipitators of the ageing process in the skin by stimulating intracellular signal transduction[28].

MMPs destroy collagen and other components of the extracellular matrix. MMP-1 is an activator of collagen degradation, and MMP-9 and MMP-3 result in additional breakdown of matrixes[29].

The TGF-β controls the production of collagen. Old age leads to a decrease in TGF-B receptor activity and downstream signaling, which causes a decrease in collagen production[30].

This is because of the imbalance between augmented MMP action and depleted TGF-β signaling, resulting in progressive dermal atrophy (Table 1)[31].

Table 1: Key Molecular Contributors to Skin Ageing

Component	Biological Role	Effect During Ageing
Reactive Oxygen Species (ROS)	Oxidative signaling molecules	Increase MMP activation and cellular damage
Matrix Metalloproteinases (MMP-1, MMP-3, MMP-9)	Collagen and ECM degradation	Elevated expression leads to collagen breakdown
TGF-β (Transforming Growth Factor Beta)	Collagen synthesis regulation	Decreased signaling reduces collagen production
NF-κB	Inflammatory transcription factor	Chronic activation causes inflammaging
AP-1	Regulates MMP gene expression	Upregulated under UV exposure

4. *Clitoria ternatea*: BOTANICAL AND ETHNOMEDICINAL OVERVIEW

Clitoria ternatea L. is a plant of the family Fabaceae and is very abundant in tropical and subtropical areas such as India, Thailand, and Malaysia[32]. It is a perennial climber that grows peculiar blue or white flowers, and is generally referred to as butterfly pea.

Flowers are most pharmacologically useful in dermatological use because they contain a high concentration of anthocyanins and flavonoids[33].

Ayurvedically, the plant is considered a rejuvenating herb and is applied in skin diseases, wound healing, and inflammation[34]. The Southeast Asian cultures also utilise it as a natural colourant and herbal remedy for the health of the skin[35].

It is ethnopharmacological evidence that encourages its utilisation in the healing of wounds, anti-ageing, and brightening of skin, which coincides with the current experimental results[36].

5. PHYTOCHEMICAL PROFILE

Clitoria ternatea has a wide variety of bioactive compounds, which include anthocyanins, flavonoids, phenolic acids, triterpenoids, and saponins (Table 2)[37].

Table 2: Phytochemical Profile of *Clitoria ternatea*

Class	Key Compounds	Concentration Range	Dermatological Activity
Anthocyanins	Ternatins A1–D3	0.8–3.2 g/100 g	Antioxidant, photoprotection, anti-melanogenic
Flavonoids	Quercetin, Kaempferol	150–680 mg QE/100 g	MMP inhibition, anti-inflammatory
Phenolic acids	Gallic acid, Catechin	80–320 mg GAE/100 g	ROS scavenging, antimicrobial
Triterpenoids	Taraxerol	0.1–0.4%	Collagen stimulation, enzyme inhibition
Saponins	Glycosides	1.2–4.5%	Barrier enhancement, hydration

5.1 Anthocyanins (Ternatins)

Ternatins are polyacylated anthocyanins that cause the extreme blue hue of the flower[38].

These are potent antioxidants with great free radical scavenging effects. They shield the skin cells and prevent the oxidative destruction and stress caused by ultraviolet[39].

Ternatin A1 was also shown to be an inhibitor of tyrosinase, whose activity is implicated in the skin brightening effect and pigmentation regulation[40].

5.2 Flavonoids

Quercetin and kaempferol are the other flavonoids that are available in large quantities[41].

These substances block the collagen synthesis of matrix metalloproteinases by binding to their catalytic active sites[42].

They also inhibit inflammatory pathways through the inhibition of the activation of NF-kB, inhibiting the production of cytokines and inflammation[43].

5.3. Phenolic Acids

Phenolic acids play the role of antioxidant defence via hydrogen atom transfer and electron donation[44].

Gallic acid and catechin stabilise collagen and offer antimicrobial coverage that promotes the health of the skin altogether[45].

5.4. Triterpenoids and Saponins

The first triterpenoid is taraxerol, which is involved in the preservation and stimulation of collagen [18].

It suppresses enzymes that break down collagen and promotes fibroblast production, resulting in more collagen[18].

Saponins enhance the barrier properties and hydration by increasing water retention in the stratum corneum[37].

6. MECHANISMS OF ACTION IN SKIN HEALTH

Clitoria ternatea can modify several pathways that play a crucial role in ageing the skin (Fig.3).

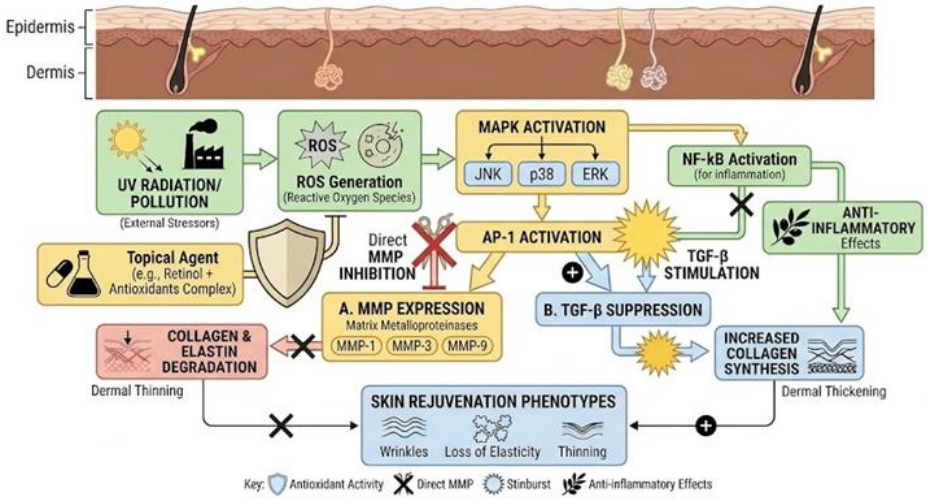

Figure 3: Mechanisms of action include antioxidant activity, MMP inhibition, TGF-β stimulation, and anti-inflammatory effects.

6.1. Antioxidant and ROS Scavenging

The extract decreases oxidative stress as it deactivates reactive oxygen species and prevents them from triggering harmful pathways[39].

According to the cell-based research, there is a marked decrease in the intracellular ROS levels after using *Clitoria ternatea* extracts[22].

6.2. Inhibition of MMP-mediated Collagen DEGRADATION

Drugs like taraxerol and flavonoids block the activity of MMP-1 and decrease collagen degradation[18].

This assists in maintaining the skin structure and preventing the development of wrinkles[42].

6.3. Stimulation of TGF-β and Fibroblast Proliferation

The extract increases TGF-β signalling, which stimulates the production of collagen[13].

Experimental research demonstrates that there is proliferation and production of collagen by fibroblasts after treatment is done [2].

6.4. Anti-inflammatory Pathways

Flavonoids inhibit NF-KB and MAPK signatures, decreasing inflammatory processes and cytokine synthesis[43].

This helps in less skin damage and healing.

7. PRECLINICAL AND EXPERIMENTAL EVIDENCE

Several reports affirm the dermatological properties of *Clitoria ternatea* (Table 3).

These experiments reveal that there are stable increases in collagen density, fibroblast activity, and oxidative stress markers [2,5,13,17].

Antioxidant and anti-inflammatory properties are also affirmed by cell culture research with large safety margins[22].

Table 3: Preclinical Evidence Summary

Author	Year	Model	Formulation	Key Findings
Nursyafillah A	2025	UV-B rats	5–10% cream	↑ Fibroblasts, ↑ Collagen
Hutapea J	2023	UV-B rats	Gel	↑ TGF-β, ↓ MMP-1
Saritani D	2021	Rats	Cream	Improved skin texture
Nirmala S	2025	Rats	Cream	↑ Hydration, ↑ antioxidant enzymes

8. FORMULATION ASPECTS IN COSMECEUTICALS

Formulation is a very important part of the delivery of active compounds.

The anthocyanins can withstand acidic conditions and perish in alkaline environments, hence designing the formulations requires a lot of care [26].

Nanoformulations, gels, and creams have been researched as ideal delivery [33].

Nanoparticle lipid carriers enhance the penetration and stability of active compounds (Table 4) [33].

Table 4: Formulation Strategies

Formulation	pH Range	Advantage	Limitation
Serum	4.0–5.0	Fast absorption	Low stability
Cream	4.5–5.5	Stable, accepted	Limited penetration
Hydrogel	4.0–5.0	Better dermal delivery	Slight tackiness
Nanocarrier	4.0–5.0	High penetration	Expensive

9. SAFETY AND TOXICOLOGICAL CONSIDERATIONS

Clitoria ternatea demonstrates a strong safety profile based on available preclinical and traditional use data. Acute toxicity studies report no adverse effects at doses up to 2000 mg per kg in animal models, indicating a high margin of safety [32].

In vitro cytotoxicity studies using keratinocytes and fibroblasts show cell viability above 90 per cent at concentrations exceeding therapeutic levels [22]. This confirms suitability for topical applications.

Skin irritation studies using patch testing methods report no erythema or oedema at concentrations between 5 and 10 per cent [2]. Traditional use across Southeast Asia further supports its safety in long-term applications [35].

However, gaps remain. No long-term dermal toxicity studies or formal sensitisation assays, such as the local lymph node assay, have been reported. Phototoxicity of degraded anthocyanins under UV exposure also requires further evaluation [26].

The safety profile of *Clitoria ternatea* is very promising with the available preclinical and traditional use data. Studies on acute toxicity do not report adverse effects up to the 2000 mg per kg dosage in animal models, meaning that its margin of safety is very high [32].

In vitro cytotoxicity analyses with keratinocytes and fibroblasts exhibit above 90 per cent cell viability at concentrations that are higher than the therapeutic levels [22].

Patch testing is used in skin irritation studies, which show no cases of erythema and oedema in the concentrations of 5-10 per cent [2]. Its safety is also proven by its use in long-term use in Southeast Asia [35].

However, gaps remain. There are neither reported long-term dermal toxicity studies nor formal sensitisation assays, e.g., the local lymph node assay. The UV-induced anthocyanin degradation is also a phenomenon that will have to be further assessed in terms of phototoxicity [26].

10. FUTURE PERSPECTIVES

Future studies ought to be involved in clinical validation and intricate formulation strategies.

Randomised controlled trials should be done to verify the efficacy in human beings using validated outcome measures of wrinkle depth, hydration, and elasticity.

Delivery systems that have been developed using nanotechnology, like lipid carriers and ethosomes, ought to be optimised to enhance penetrability and stability.

The anthocyanin content and other extract components need to be standardised in order to be reproducible.

Studies may have an added effect with combination formulations including established actives, e.g., niacinamide and bakuchiol, via complementary mechanisms.

Metabolic engineering and other biotechnological methods could help provide scalable active compound production through the use of consistent quality.

DISCUSSION

Although there is solid preclinical data, there are numerous critical gaps to clinical translation (Table 5).

- None of the human randomised controlled trials is available.
- Absence of transdermal penetration research.
- No standard extract composition.
- Scarcity of regulatory toxicology information.
- Lack of studies on the influence.

Such loopholes limit regulatory authorisation and evidence products.

Table 5: Research Gaps

Gap	Description	Impact
Human trials	No RCT studies	No clinical validation
Pharmacokinetics	No penetration data	Unknown dermal delivery
Standardization	Variable extracts	Poor reproducibility
Safety studies	No long-term data	Regulatory limitation

Clitoria ternatea is a rare plant with multiple dermatological mechanisms in one botanical extract.

Its phytochemical diversity has allowed it to be targeted on oxidative stress, collagen degradation, inflammation, and pigmentation simultaneously. This act of multi-target makes it different from the traditional single-mechanism actives.

Preclinical evidence has always shown an increase in collagen density, fibroblast proliferation, and antioxidant status. These are the effects of ageing that come to bear directly on the structural and biochemical alterations that are related to ageing.

Nevertheless, there is a significant limitation in the absence of human clinical trials. Its use is limited to exploratory and experimental use as cosmetics without clinical evidence.

A comparison with known actives reveals that although such compounds as vitamin C and retinoids have greater clinical validation, *Clitoria ternatea* has a wider coverage in mechanistic studies.

It has a good safety profile and has conventional use, which gives it an advantage in the formulation of sensitive skin products.

CONCLUSION

Clitoria ternatea is a scientifically promising botanical with significant potential in anti-ageing and skin health applications.

Its bioactive compounds provide antioxidant protection, inhibit collagen degradation, stimulate collagen synthesis, reduce inflammation, and enhance skin barrier function.

Preclinical evidence strongly supports its efficacy in improving dermal structure and function.

However, translation into clinical practice requires human trials, standardised formulations, and comprehensive safety evaluation.

With further research and validation, *Clitoria ternatea* has the potential to become a key ingredient in next-generation cosmeceutical formulations.

Clitoria ternatea is a scientifically promising botanical that is likely to have a high application in anti-ageing and skin health.

Its bioactive compounds offer the following: protection against oxidative stress, prevention of collagen degradation, promotion of collagen synthesis, prevention of inflammation, and improvement of the skin barrier mechanism.

There is solid preclinical evidence that it is effective in enhancing dermal structure and function.

Nevertheless, the translation into clinical practice necessitates clinical trials, standardisation of formulation, and extensive safety studies.

Clitoria ternatea can replace several crucial beauty ingredient formulations in the next-generation cosmeceuticals with further research and validation.

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