



FORMULATION AND EVALUATION OF HERBAL ANTI-MELASMA CREAM CONTAINING MULBERRY EXTRACT

Ms. Amruta Nilesh Shinde
Bachelor of Pharmacy (B.Pharm)

Ms. Sonali Sanjay Shirke
(M.pharm in Quality assurance)

Department of Pharmacy,
College of Pharmacy, Medha, Satara, Maharashtra, India

ABSTRACT :-

Melasma is a common dermatological condition characterized by hyperpigmentation, primarily affecting the facial region. The present study focused on the formulation and evaluation of a herbal anti-melasma cream containing mulberry extract as the main active ingredient because of its tyrosinase inhibitory potential. Green tea extract was also incorporated for its antioxidant and photoprotective properties, along with suitable excipients to develop a stable and cosmetically acceptable formulation.

The cream was prepared using the emulsification method and evaluated for various physicochemical parameters such as appearance, pH, spreadability, homogeneity, washability, and skin irritation. The formulated cream exhibited a smooth texture, good consistency, and a white to off-white appearance. The pH of the formulation was found to be within the range of 6.2–6.4, making it suitable for topical application. In addition, the formulation showed satisfactory spreadability, easy washability, and no signs of skin irritation.

The findings suggest that the developed herbal cream is stable, safe, and potentially effective for the management of melasma and other hyperpigmentation disorders. The formulation may serve as a promising natural alternative for topical skin care applications.

INTRODUCTION :-

Melasma is a common acquired skin pigmentation disorder characterized by symmetrical brown to grayish-brown patches that mainly appear on the facial region. The condition is more frequently observed in women and individuals with darker skin tones, particularly in areas exposed to intense sunlight. The development of melasma is influenced by multiple factors, including ultraviolet (UV) radiation, hormonal changes, genetic susceptibility, and increased melanocyte activity. Enhanced activity of the enzyme tyrosinase leads to excessive melanin production, which plays a major role in the pathogenesis of the disorder^[1].

Several conventional therapies, such as hydroquinone, corticosteroids, and retinoids, are commonly used for the treatment of melasma. While these agents may provide beneficial effects, their long-term use is often associated with side effects including skin irritation, redness, dryness, and safety-related concerns. As a result, attention has increasingly shifted toward herbal and naturally derived alternatives that are considered comparatively safer and more suitable for prolonged topical use^[1].

Mulberry (*Morus alba*) has gained considerable interest as a natural depigmenting agent because it contains various bioactive constituents, including flavonoids and phenolic compounds. These phytochemicals possess notable tyrosinase inhibitory activity, which helps reduce melanin synthesis and may assist in the management of hyperpigmentation disorders such as melasma^[2,3]. Furthermore, mulberry extract demonstrates antioxidant activity that can help protect the skin against oxidative damage induced by UV exposure.

Green tea (*Camellia sinensis*) is another important herbal ingredient commonly incorporated into dermatological and cosmetic formulations. It is rich in polyphenolic compounds, especially epigallocatechin gallate (EGCG), which exhibit potent antioxidant and anti-inflammatory properties. Studies have shown that green tea can suppress tyrosinase activity and decrease melanin formation, thereby supporting skin brightening and protection against UV-mediated skin damage. The inclusion of green tea extract in topical preparations may therefore improve the therapeutic potential of anti-melasma formulations^[4].

In view of these properties, the present study was undertaken to formulate and evaluate a herbal anti-melasma cream containing mulberry extract, along with green tea extract as a supportive herbal component, with the objective of developing a safe, stable, and effective topical formulation for the management of skin hyperpigmentation.



Fig.: Melasma Skin

DISORDER :-

MELASMA :

Melasma is a common acquired hyperpigmentation disorder characterized by symmetrical brown to grayish macules and patches that mainly appear on sun-exposed areas of the face, including the cheeks, forehead, nose, and upper lip. The condition is more commonly observed in women and individuals with darker skin types. The development of melasma is multifactorial and involves ultraviolet (UV) radiation, hormonal influences, genetic susceptibility, and increased melanocyte activity, resulting in excessive melanin production^[1,5].

Among these factors, ultraviolet radiation is considered one of the primary triggers, as it stimulates melanogenesis through activation of tyrosinase and other melanogenic enzymes. Hormonal influences, particularly estrogen and progesterone, also contribute significantly to the condition, which explains the increased incidence of melasma during pregnancy and among individuals using oral contraceptives^[5,6]. Furthermore, oxidative stress and inflammatory mediators are known to enhance melanocyte stimulation and promote melanin synthesis, thereby contributing to disease progression.

Melasma is regarded as a chronic and recurrent dermatological condition that is often challenging to manage because of its complex etiology and high recurrence rate. Conventional treatment approaches include topical depigmenting agents, chemical peels, and laser-based therapies. Although these treatments may show clinical improvement, they are frequently associated with adverse effects and inconsistent therapeutic outcomes^[6,7]. Consequently, there has been growing interest in the use of herbal and naturally derived therapies, which are considered safer and potentially more suitable for the long-term management of melasma and related hyperpigmentation disorders^[7,8].

ETIOLOGY:

Melasma is a multifactorial skin disorder that develops due to the combined influence of environmental, hormonal, and genetic factors. Among these, ultraviolet (UV) radiation is considered one of the major contributing factors, as it stimulates melanocytes to produce excess melanin through the activation of melanogenic enzymes, particularly tyrosinase^[9].

Hormonal factors also play an important role in the pathogenesis of melasma. Increased levels of estrogen and progesterone, especially during pregnancy or following the use of oral contraceptives, can enhance melanocyte activity and promote hyperpigmentation^[10].

Genetic predisposition is another significant factor associated with the condition. Individuals with a family history of melasma are more likely to develop the disorder, suggesting a hereditary component in its occurrence. In addition, the use of certain medications and photosensitizing drugs may trigger or worsen pigmentation by increasing the skin's sensitivity to sunlight^[11].

Cosmetic products and skin irritation may further contribute to the development of melasma by inducing inflammatory reactions that stimulate melanogenesis. Moreover, oxidative stress caused by reactive oxygen species (ROS) has been reported to enhance melanocyte activation and melanin synthesis, thereby contributing to disease progression^[12].

Overall, the etiology of melasma involves the interaction of several factors, including UV exposure, hormonal imbalance, genetic susceptibility, medications, cosmetic usage, and oxidative stress, all of which contribute to abnormal melanin deposition in the skin.

EPIDEMIOLOGY :-

Melasma is a common acquired pigmentary disorder that occurs more frequently in women and individuals with darker skin tones. Epidemiological studies have reported that nearly 90–93% of melasma cases are observed in females, indicating a strong association between the condition and hormonal influences^[13]. The disorder is most commonly seen during the reproductive age group, particularly between 20 and 50 years of age, further supporting the role of hormonal factors in its pathogenesis^[14].

The prevalence of melasma differs according to geographic region, ethnicity, and the degree of sun exposure. The condition is especially common in tropical and subtropical areas where exposure to ultraviolet (UV) radiation is high. Individuals with Fitzpatrick skin types III–V, including Asian, Hispanic, Middle Eastern, and African populations, are more susceptible to melasma because of their naturally higher melanin content^[15].

Several epidemiological reports have shown that melasma accounts for a considerable number of dermatological consultations worldwide, emphasizing its clinical and cosmetic significance. The disorder is generally chronic and recurrent in nature, and its severity often increases with prolonged or repeated exposure to sunlight^[16].

Overall, melasma is a widely prevalent dermatological condition influenced by multiple factors such as gender, age, ethnicity, skin type, and environmental exposure, particularly ultraviolet radiation.

PATHOPHYSIOLOGY :-

Melasma is a complex and multifactorial hyperpigmentation disorder characterized by dysregulation of melanogenesis along with structural and functional alterations within the epidermis and dermis. Current research suggests that melasma is not merely a disorder of melanocytes but involves complex interactions among keratinocytes, fibroblasts, endothelial cells, and inflammatory mediators within the skin microenvironment^[15].

1. Role of Ultraviolet (UV) Radiation and Light Exposure

Ultraviolet radiation, particularly UV-A and UV-B, is considered the major triggering factor in the development of melasma. Exposure to UV radiation stimulates melanocytes through activation of signaling pathways that increase the expression of melanogenic enzymes such as tyrosinase, tyrosinase-related protein-1 (Tyrp1), and Tyrp2, ultimately leading to enhanced melanin synthesis^[17,18].

Recent studies have also demonstrated the contribution of visible light, especially blue light, in promoting persistent pigmentation, particularly in individuals with darker skin types. This finding highlights the broader role of photic exposure in the pathogenesis of melasma^[15].

2. Epidermal–Dermal Interactions

The interaction between epidermal and dermal cells plays a crucial role in maintaining melanogenesis in melasma. Keratinocytes, fibroblasts, and endothelial cells release several paracrine mediators, including stem cell factor (SCF), endothelin-1, and α -melanocyte-stimulating hormone (α -MSH), which activate melanocytes and enhance melanin production^[18].

In addition, fibroblast senescence and alterations in the dermal microenvironment further contribute to chronic pigmentation and disease persistence.

3. Hormonal Influence

Hormonal factors, particularly estrogen and progesterone, are strongly associated with the development of melasma. These hormones increase melanocyte sensitivity and stimulate melanogenesis by enhancing tyrosinase activity and regulating melanocyte receptors. This mechanism explains the higher prevalence of melasma among women, especially during pregnancy and hormonal therapy^[10].

4. Oxidative Stress and Inflammation

Oxidative stress is considered an important factor in melasma pathogenesis. Ultraviolet radiation induces the formation of reactive oxygen species (ROS), which stimulate melanocyte activity and promote melanin synthesis. Furthermore, inflammatory mediators and mast cell activation contribute to persistent pigmentation and skin damage [17,19].

5. Dermal Changes and Structural Alterations

Melasma is associated with several dermal abnormalities that resemble changes seen in photoaged skin. These include:

- Disruption of the basement membrane
- Solar elastosis
- Increased vascularization (angiogenesis)
- Accumulation of mast cells

Such structural alterations facilitate the deposition of melanin into the dermis and are believed to contribute to the chronicity and recurrence of the condition [15,19].

6. Genetic and Molecular Factors

Genetic predisposition is another important contributor to melasma. Altered expression of genes associated with melanogenesis, inflammation, and skin barrier function has been observed in affected individuals. Recent evidence also suggests that microRNAs and epigenetic mechanisms may regulate melanocyte activity and influence disease progression [17,19].

HISTOPATHOLOGY :-

Melasma is associated with characteristic histopathological alterations involving both the epidermal and dermal layers of the skin, reflecting its complex and multifactorial pathogenesis.

1. Epidermal Changes

One of the major histopathological features of melasma is excessive melanin deposition within the epidermis, particularly in the basal and suprabasal layers. Melanin is distributed throughout different layers of the epidermis, while melanocytes often exhibit increased dendricity and enhanced functional activity. However, the total number of melanocytes generally remains normal or only slightly elevated, suggesting that melanocyte hyperactivity rather than cellular proliferation is primarily responsible for the increased pigmentation [15,20].

In addition, keratinocytes in melasma lesions contain increased numbers of melanosomes, which are more extensively dispersed when compared with normal skin.

2. Dermal Changes

Dermal involvement is considered a consistent and important feature of melasma. Histopathological examination commonly demonstrates:

- Melanin incontinence with the presence of dermal melanophages
- Solar elastosis indicating chronic ultraviolet-induced skin damage
- Disruption of the basement membrane
- Increased vascularization and mast cell infiltration

These dermal abnormalities are believed to contribute to the chronicity, persistence, and therapeutic resistance commonly observed in melasma patients [21,22].

3. Basement Membrane and Structural Alterations

Basement membrane disruption is frequently reported in melasma lesions. Damage to this structure facilitates the transfer of melanin or melanocytes into the dermis, resulting in deeper and more persistent pigmentation. Histological studies have also described the presence of “pendulous melanocytes” extending into the dermis, which may occur due to structural alterations induced by prolonged ultraviolet exposure [22].

4. Inflammatory and Vascular Changes

Melasma lesions often exhibit features of subclinical inflammation, including increased mast cell numbers and enhanced vascularity. These inflammatory and vascular changes stimulate melanocytes through the release of cytokines, inflammatory mediators, and growth factors, thereby promoting melanogenesis and sustaining pigmentation [15,21].

5. Histological Types of Melasma

Based on the location of pigment deposition, melasma is histologically classified into three major types:

- **Epidermal type:** Characterized by increased melanin deposition within the epidermis
- **Dermal type:** Characterized by the presence of melanophages within the dermis
- **Mixed type:** Shows features of both epidermal and dermal pigmentation

Recent studies, however, indicate that most melasma cases demonstrate mixed histological features, highlighting the complex nature of the disorder ^[20].

TREATMENT & MANAGEMENT :-

The management of melasma remains challenging because of its chronic, relapsing, and treatment-resistant nature. Effective treatment generally requires a multimodal approach that includes photoprotection, topical medications, systemic therapies, and various procedural interventions.

1. Photoprotection

Photoprotection is considered the foundation of melasma management. Regular use of broad-spectrum sunscreens that protect against UV-A, UV-B, and visible light is essential to minimize melanocyte stimulation and prevent recurrence of pigmentation. Physical sunscreens containing zinc oxide or titanium dioxide are particularly effective because they provide broader protection against ultraviolet and visible light exposure ^[23,24].

2. Topical Therapies

Topical depigmenting agents are regarded as the first-line treatment for melasma. Commonly used agents include:

- **Hydroquinone:** Considered the gold standard treatment due to its ability to inhibit tyrosinase activity and reduce melanin synthesis.
- **Retinoids (e.g., tretinoin):** Promote epidermal cell turnover and improve the penetration of other topical agents.
- **Corticosteroids:** Help reduce inflammation and suppress melanocyte activity.

Triple combination therapy consisting of hydroquinone, tretinoin, and a corticosteroid has demonstrated greater clinical efficacy compared with monotherapy.

Other topical agents such as azelaic acid, kojic acid, niacinamide, and arbutin also exhibit depigmenting properties and are widely used in melasma treatment ^[24,25].

3. Systemic Therapy

Systemic treatment options, particularly oral tranexamic acid, have shown promising outcomes in moderate to severe melasma. Tranexamic acid is believed to reduce melanocyte activation and vascular involvement associated with the disorder. However, its use requires careful monitoring because of the possibility of adverse effects and contraindications ^[25,26].

4. Chemical Peels

Superficial chemical peels using agents such as glycolic acid, salicylic acid, and lactic acid help remove pigmented epidermal layers and stimulate skin renewal. Chemical peels are commonly used as adjunctive therapy in combination with topical depigmenting agents to improve treatment outcomes ^[24].

5. Laser and Light-Based Therapies

Laser and light-based procedures, including Q-switched Nd:YAG lasers and intense pulsed light (IPL), specifically target melanin pigments within the skin. Although these procedures may provide clinical improvement, they are associated with a risk of post-inflammatory hyperpigmentation, especially in individuals with darker skin types ^[26].

6. Herbal and Emerging Therapies

Recently, there has been growing interest in herbal and naturally derived therapies for melasma management. Natural ingredients such as mulberry extract, green tea, licorice, and other plant-derived compounds possess tyrosinase inhibitory, antioxidant, and anti-inflammatory properties. These herbal therapies are considered safer alternatives with fewer adverse effects and may be more suitable for long-term use in the management of hyperpigmentation disorders ^[2].

NEED FOR INVESTIGATION :-

Melasma is a chronic and recurrent hyperpigmentation disorder that remains difficult to manage effectively. Although several therapeutic approaches are available, recurrence of pigmentation is common, particularly following sun exposure or hormonal stimulation. This recurring nature of the disease highlights the necessity for safer and more effective long-term treatment strategies.

Conventional treatment options such as hydroquinone, tretinoin, and corticosteroids are widely used in the management of melasma. However, these therapies may not produce satisfactory results in all patients, and some individuals exhibit only partial or delayed clinical improvement. Furthermore, prolonged use of these agents is often associated with adverse effects including skin irritation, erythema, contact dermatitis, and exogenous ochronosis, especially with long-term hydroquinone therapy. These limitations have increased the demand for safer alternatives with improved patient tolerability.

The pathophysiology of melasma is highly complex and involves multiple contributing factors such as ultraviolet radiation, hormonal imbalance, genetic predisposition, oxidative stress, and excessive melanin synthesis. Because of this multifactorial nature, therapies targeting a single pathway may not provide complete therapeutic benefit. Therefore, the development of multi-targeted treatment approaches has become increasingly important.

In addition to its clinical manifestations, melasma has a significant psychological and cosmetic impact on affected individuals because it predominantly involves the facial region. The visible pigmentation may lead to reduced self-confidence, emotional distress, and impaired quality of life, thereby increasing the need for effective and cosmetically acceptable therapeutic formulations.

Recently, growing attention has been directed toward herbal and naturally derived therapies due to their potential safety, efficacy, and suitability for long-term use. Plant-based agents such as mulberry and green tea extracts possess tyrosinase inhibitory, antioxidant, and anti-inflammatory properties that may be beneficial in the management of hyperpigmentation disorders. Furthermore, advances in topical drug delivery systems aim to improve drug penetration, formulation stability, and patient compliance.

Despite the availability of multiple treatment modalities, there is still no permanent cure for melasma. Therefore, continuous research is essential to develop safer, more effective, and long-lasting therapeutic approaches for the prevention and management of this condition.

AIM & OBJECTIVE :-

AIM:

“Formulation and Evaluation of Herbal Anti-Melasma Cream Containing *Morus alba* (Mulberry) and *Camellia sinensis* (Green Tea)”

OBJECTIVE:

To formulate and develop a stable and effective herbal anti-melasma cream using mulberry extract as the active pharmaceutical ingredient.

- To utilize *Morus alba* leaf extract as a natural tyrosinase inhibitor for reducing melanin synthesis and managing hyperpigmentation.
- To incorporate *Camellia sinensis* (green tea) extract for its antioxidant, anti-inflammatory, and skin-protective properties.
- To evaluate the physicochemical properties of the formulated cream, including:
 1. Appearance
 2. pH
 3. Viscosity
 4. Spreadability
 5. Homogeneity
- To assess the skin compatibility and safety of the formulation through skin irritation testing.

- To develop a formulation that is safe, cost-effective, and suitable for topical application in the management of melasma and related hyperpigmentation disorders.

PLAN OF WORK :-

Collection of relevant information from research articles, review papers, and scientific journals related to melasma and herbal therapies.



Selection and finalization of the research topic.



Selection of plant materials, including *Morus alba* (mulberry) as the primary active ingredient along with supportive herbal ingredients such as *Camellia sinensis* (green tea), aloe vera, and other suitable herbal agents.



Selection of appropriate excipients required for the formulation of the herbal cream.



Methodology

- Preparation of mulberry extract
- Preparation of green tea extract



Experimental Work

- Formulation of herbal anti-melasma cream
- Evaluation of the formulated cream based on the following parameters:

- Physical appearance
- pH determination
- Spreadability test
- Washability test
- Skin irritation test
- Homogeneity
- Stability studies



Analysis and interpretation of the obtained results.



Conclusion based on the stability, safety, and effectiveness of the formulated herbal anti-melasma cream.

EXCIPIENT PROFILE :-

EXCIPIENT	ROLE
1. Aloe Vera Gel	Soothing Agent
2. Vitamin E	Antioxidant
3. Stearic acid	Emulsifier, Thickener
4. Cetyl alcohol	Stabilizer
5. Liquid Paraffin	Emollient
6. Glycerin	Humectant
7. Triethanolamine	pH Adjuster
8. Methyl paraben	Preservative
9. Lavender Essence	Perfume
10. Distilled Water	Vehicle

Table : Excipients

METHODOLOGY :-

- ❖ Preparation of Mulberry Extract :

Method: Maceration Technique

Step 1: Collection and Drying

- Fresh mulberry (*Morus alba*) leaves were collected and thoroughly washed with water to remove dirt and impurities.
- The cleaned leaves were dried under shade to prevent degradation of active phytoconstituents.

Step 2: Size Reduction

- The dried leaves were ground into a coarse powder using a mechanical grinder or mixer.

Step 3: Weighing

- Approximately 5 g of the powdered drug was accurately weighed using a digital balance.

Step 4: Maceration (Soaking)

- The weighed powder was transferred into a clean container.
- About 50 mL of ethanol was added in a drug-to-solvent ratio of 1:10.
- The container was tightly closed with a lid and covered with aluminium foil to minimize solvent evaporation and light exposure.
- The mixture was kept undisturbed for 48 hours with occasional stirring to ensure proper extraction of phytoconstituents.

Step 5: Filtration

- After completion of maceration, the mixture was filtered using filter paper to separate the liquid extract from the solid residue.

Step 6: Concentration

- The filtrate was concentrated by evaporating the solvent on a water bath until a concentrated extract was obtained.

Step 7: Storage

- The concentrated extract was stored in an amber-colored airtight container and kept in a cool place until further use in formulation development.



Fig.: Mulberry Leaf Powder Maceration

❖ Preparation of Green Tea Extract :

Method: Maceration Technique

Step 1: Collection and Drying

- Green tea (*Camellia sinensis*) leaves were collected and washed thoroughly with water to remove impurities and foreign particles.
- The leaves were then dried under shade to preserve their active constituents.

Step 2: Size Reduction

- The dried leaves were ground into a coarse powder using a mixer or mechanical grinder.

Step 3: Weighing

- About 5 g of the powdered drug was accurately weighed using a digital weighing balance.

Step 4: Maceration (Soaking)

- The weighed powder was transferred into a clean container.
- Approximately 50 mL of ethanol was added in a drug-to-solvent ratio of 1:10.
- The container was tightly closed with a lid and covered with aluminium foil to avoid solvent evaporation and exposure to light.
- The mixture was allowed to stand for 48 hours with occasional stirring to facilitate proper extraction of active phytoconstituents.

Step 5: Filtration

- After maceration, the mixture was filtered using filter paper to separate the extract from the solid residue.

Step 6: Concentration

- The filtrate was concentrated by evaporating the solvent using a water bath until a concentrated extract was obtained.

Step 7: Storage

- The concentrated extract was stored in an amber-colored airtight container and kept in a cool place until further use in formulation prepare



Fig.: Green Tea Leaf Powder Maceration

FORMULATION OF MULBERRY ANTI-MELASMA CREAM :-**Formulation Table :**

Sr.No.	Ingredient	F ₁	F ₂	F ₃
1.	Mulberry Extract	1.0gm	1.5gm	2.0gm
2.	Green Tea Extract	0.5gm	1.0gm	1.5gm
3.	Aloe Vera gel	2gm	2.0gm	2gm
4.	Vitamin E	0.50gm	0.50gm	0.50gm
5.	Stearic Acid	3gm	3gm	3gm
6.	Cetyl Alcohol	2gm	2gm	2gm
7.	Propylene Glycol	1.5ml	2.5ml	3.5ml
8.	Glycerin	2.5ml	2.5ml	2.5ml
9.	Methyl Paraben	0.10gm	0.10gm	0.10gm
10.	Triethanolamine	q.s	q.s	q.s
11.	Lavender Essence	q.s	q.s	q.s
12.	Distilled Water	q.s	q.s	q.s

Table : Formulation Table of Mulberry Anti-Melasma Cream

List of Glassware :

Sr.No.	Glassware
1.	Beaker
2.	Measuring Cylinder
3.	Glass Rod
4.	Water Bath
5.	Mortar Pestle

Table : List of Glassware

Procedure :**1. Preparation of Aqueous Phase**

The required quantity of distilled water, glycerin, and propylene glycol was accurately measured and transferred into a suitable beaker. The mixture was heated to approximately 70°C with continuous stirring until a clear and uniform solution was obtained.

2. Preparation of Oil Phase

Stearic acid and cetyl alcohol were accurately weighed and heated together separately at about 70°C until the ingredients melted completely and formed a uniform oily phase.

3. Emulsification

The oil phase was added slowly into the aqueous phase with continuous stirring using a mortar and pestle to form a stable emulsion.

4. Cooling of the Mixture

The prepared emulsion was allowed to cool gradually to approximately 40°C while maintaining continuous stirring to ensure proper consistency and uniformity.

5. Addition of Active Ingredients

Mulberry extract, green tea extract, and aloe vera gel were added to the cooled emulsion and mixed thoroughly to achieve uniform distribution of the active ingredients.

6. Addition of Other Ingredients

Methyl paraben, lavender essence, and vitamin E were incorporated into the formulation and mixed properly to ensure homogeneity of the cream.

7. pH Adjustment

Triethanolamine was added dropwise to adjust the pH of the formulation within the range of 5.5–6.5, making it suitable for topical skin application.

8. Final Mixing and Packaging

The formulation was stirred continuously until a smooth and homogeneous cream was obtained. The prepared cream was then transferred into a suitable airtight container and stored under appropriate conditions for further evaluation studies.



Fig. : Preparation of Mulberry Anti-Melasma Cream



Fig. : Formulation of Mulberry Anti-Melasma Cream

EVALUATION TESTS :-**1. Physical Properties :**

Physical Properties are the Characteristics of a Formulation that can be Observed Visually Without Changing its Chemical Composition.

Table of Physical Properties :

Parameter	F ₁	F ₂	F ₃
Colour	Olive Green	Olive Green	Olive Green
Odour	Pleasant	Pleasant	Pleasant
Appearance	Smooth	Smooth	Smooth

Table : Physical Properties & their Observation

2.Determination of pH :

The pH of the cream was determined by dispersing 1 g of cream in 9 mL of distilled water and measuring using pH paper. The pH was found to be in the range of 5.5–6.5, which is suitable for skin application.

Table of pH :

Parameter	F ₁	F ₂	F ₃
pH	6.3	6.2	6.4

Table : pH Observation



Fig.: pH Meter Observation

3.Spreadability :

Spreadability is the ease with which a cream spreads on the skin.

- **Method**

- 1.Place cream between two glass slides
- 2.Apply weight
- 3.Measure time taken to spread

$$\text{Spreadability} : M \times L / T$$

Where,

M = weight tied to upper slide

L = length moved

T = time

4.Washability :

Washability is the ease with which the cream can be removed from the skin using water.

- **Method**

1. Apply cream on skin
2. Wash with tap water

5.Irritancy Test :

Irritancy test checks whether the cream causes irritation on skin.

- **Method**

1. Apply small amount on arm
2. Observe for 24 hours

6.Homogeneity :

Homogeneity refers to uniform distribution of all ingredients in the cream.

- **Method**

- 1.Visually inspect
- 2.Rub between fingers

RESULT :-

Sr.No.	Parameter	Observation F ₁	Observation F ₂	Obsrvation F ₃
1.	Colour	Olive Green	Olive Green	Olive Green
2.	Odour	Pleasant	Pleasant	Pleasant
3.	Appearance	Smooth	Smooth	Smooth
4.	pH	6.2	6.3	6.4
5.	Spreadability	9 g.cm/sec	10 g.cm/sec	9.4 g.cm/sec
6.	Irritancy	No	No	No

Table : Properties & Observations of F₁, F₂, F₃

In Above results, We are Observed F₂ Formulation is Safe and Effective formulation as Compared to the F₁ & F₃ Formulations.

CONCLUSION :-

The present study successfully involved the formulation and evaluation of a herbal anti-melasma cream containing mulberry (*Morus alba*) extract as the primary active ingredient along with green tea (*Camellia sinensis*) extract and other suitable excipients. The developed formulation exhibited satisfactory physicochemical characteristics, including smooth texture, good homogeneity, and an acceptable appearance.

The pH of the formulated cream was found to be within the range of 6.2–6.4, indicating its suitability for topical skin application with minimal chances of irritation. The cream also demonstrated good spreadability and easy washability, which may enhance patient convenience and compliance. Furthermore, no evidence of skin irritation, phase separation, or instability was observed during the evaluation studies.

Overall, the formulated herbal cream was found to be stable, safe, and cosmetically acceptable. The presence of mulberry extract, known for its tyrosinase inhibitory activity, may help reduce melanin synthesis and contribute to the management of melasma and other hyperpigmentation disorders. Therefore, the developed formulation may serve as a promising natural alternative for topical anti-melasma therapy.

References :-

- 1.Handog EB et al. Melasma: A clinical review. Handog EB, Galang DA, de Leon-Godinez MA, Chan GP. Melasma: a clinical review. J Am Acad Dermatol. 2011;65(4):699–714.
- 2.Chang TS. Natural melanogenesis inhibitors acting through the down-regulation of tyrosinase activity Chang TS. Natural melanogenesis inhibitors acting through the down-regulation of tyrosinase activity. Materials. 2012;5(9):1661–1685.
- 3.Lee SH et al. Mulberry extract inhibits melanin synthesis Lee SH, Choi SY, Kim H, et al. Mulberry extract inhibits melanin synthesis by suppression of tyrosinase activity. J Cosmet Sci. 2002;53(6):377–386.
- 4.Katiyar SK. Green tea and skin photoprotection Katiyar SK. Green tea prevents UV-induced oxidative stress and immune suppression in the skin. J Nutr. 2003;133(10):3248S–3254S.
- 5.Sarkar R et al. Melasma update Sarkar R, Arora P, Garg KV. Melasma update. Indian Dermatol Online J. 2014;5(4):426–435.

6. Guinot C et al. Melasma: epidemiology and pathophysiology Guinot C, Cheffai S, Latreille J, et al. Aggravating factors for melasma: a prospective study. *J Eur Acad Dermatol Venereol*. 2010;24(5):537–543.
7. Kang HY et al. Melasma: clinical and histological characteristics Kang HY, Ortonne JP. What should be considered in treatment of melasma. *Ann Dermatol*. 2010;22(4):373–378.
8. Briganti S et al. Chemical and instrumental approaches to treat hyperpigmentation
9. Grimes PE. Melasma: etiologic and therapeutic considerations Grimes PE. Melasma: etiologic and therapeutic considerations. *Arch Dermatol*. 1995;131(12):1453–1457.
10. Sheth VM and Pandya AG. Melasma: a comprehensive update Sheth VM, Pandya AG. Melasma: a comprehensive update. *J Am Acad Dermatol*. 2011;65(4):689–697.
11. Hexsel D et al. Melasma: epidemiology and risk factors Hexsel D, Lacerda DA, Cavalcante AS, et al. Epidemiology of melasma in Brazilian patients. *Int J Dermatol*. 2014;53(4):440–444.
12. Passeron T and Picardo M. Melasma, a photoaging disorder Passeron T, Picardo M. Melasma, a photoaging disorder. *Pigment Cell Melanoma Res*. 2018;31(4):461–465.
13. Sharma AN et al. The Burden of Melasma: Race, Ethnicity, and Comorbidities Sharma AN, Kincaid CM, Mesinkovska NA. The burden of melasma: Race, ethnicity, and comorbidities. *J Drugs Dermatol*. 2024;23(8):691–693.
14. Dias MO et al. Prevalence of facial melasma among adult women Dias MO, Minagawa FH, Abreu AF, et al. Prevalence of facial melasma among adult women in a multiracial population. *Int J Dermatol*. 2024;63(4):e89–e91.
15. StatPearls Melasma Epidemiology Review Grimes PE, et al. Melasma. StatPearls Publishing. 2024.
16. Wang LJ et al. Global research trends on melasma Wang LJ, Pang YB, Li WQ, et al. Global research trends on melasma: a bibliometric study. *Front Pharmacol*. 2024;15:1421499
17. Yang J et al. Mechanisms of ultraviolet-induced melasma formation Yang J, Zeng J, Lu J. Mechanisms of ultraviolet-induced melasma formation. *J Dermatol*. 2022;49(12):1201–1210.
18. Liu W et al. New mechanistic insights of melasma Liu W, Chen Q, Xia Y. New mechanistic insights of melasma. *Clin Cosmet Investig Dermatol*. 2023;16:429–442.
19. Espósito ACC et al. Update on Melasma—Part I Pathogenesis Espósito ACC, Cassiano DP, Silva CN, et al. Update on Melasma—Part I: Pathogenesis. *Dermatol Ther*. 2022.
20. Kang HY et al. Histologic characteristics of melasma Kang HY, Bahadoran P, Suzuki I, et al. The role of dermal changes in melasma. *J Invest Dermatol*. 2002.
21. Phansuk K et al. Dermal Pathology in Melasma: An Update Review Phansuk K, Vachiramorn V, Jurairattanaporn N, et al. Dermal pathology in melasma: An update review. *Clin Cosmet Investig Dermatol*. 2022.
22. Park GH et al. Heterogeneous Pathology of Melasma Park GH, et al. Heterogeneous pathology of melasma and its clinical implications. *Int J Mol Sci*. 2016.
23. Grimes PE. Melasma: clinical features and management Grimes PE. Melasma: clinical features and management. *Dermatol Clin*. 2019;37(2):261–268.
24. Handog EB et al. Guidelines for the management of melasma Handog EB, Datuin MS, Singal A. A global consensus recommendation on melasma management. *J Dermatol Treat*. 2020;31(8):1–10.

25.Desai SR et al. Oral tranexamic acid in melasma Desai SR, Alexis AF. Oral tranexamic acid for the treatment of melasma. J Clin Aesthet Dermatol. 2020;13(2):28–34.

26.Ogbechie-Godec OA et al. Laser therapy in melasma Ogbechie-Godec OA, Elbuluk N. Melasma: an up-to-date comprehensive review. Dermatol Ther. 2017;30(6):e12465.

27.Butt, M. S., et al. (2008). Morus alba L. nature’s functional tonic. Critical Reviews in Food Science and Nutrition, 48(10), 919–934.

