Research Article

Evolution Of Anti-Hyperpigmentation Skincare: From Traditional Remedies To Modern Cosmeceutical Approaches For Adolescents

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ABSTRACT

A frequent dermatological issue, skin hyperpigmentation is marked by the darkening of the skin due to an excess of melanin production. Affected individuals experience uneven skin tone and aesthetic concerns. Treatment for hyperpigmentation is still difficult, and the available treatments are time-consuming and have inconsistent results, which reduces patient compliance. Topical medications like hydroquinone, kojic acid, and glycollic acid, which aim to decrease melanin synthesis and boost epidermal turnover, are the mainstay of first-line treatments. As supplemental treatments, oral formulations including melatonin and cysteamine hydrochloride have also showed promise. Chemical peels and laser treatments are examples of second-line therapies that are typically carried out under medical supervision and produce quicker results, but they also carry a higher risk of side effects like erythema, irritation, and post-inflammatory pigmentary changes.

Plant-based substances, herbal extracts, and natural formulations have been utilised in traditional medicine to treat pigmentation-related issues and to improve skin tone. These conventional methods have gradually evolved into evidence-based cosmeceutical formulations including phytochemicals, botanical extracts, antioxidants, tyrosinase inhibitors, and sophisticated delivery systems thanks to developments in dermatological science and cosmetic technology. Adolescents may be especially affected by hyperpigmentation and uneven skin tone because of hormonal fluctuations, inflammation from acne, sun exposure, and post-inflammatory hyperpigmentation. The creation of gentle, secure, and scientifically validated anti-hyperpigmentation cosmeceuticals for use on teenage skin is therefore becoming more and more necessary.

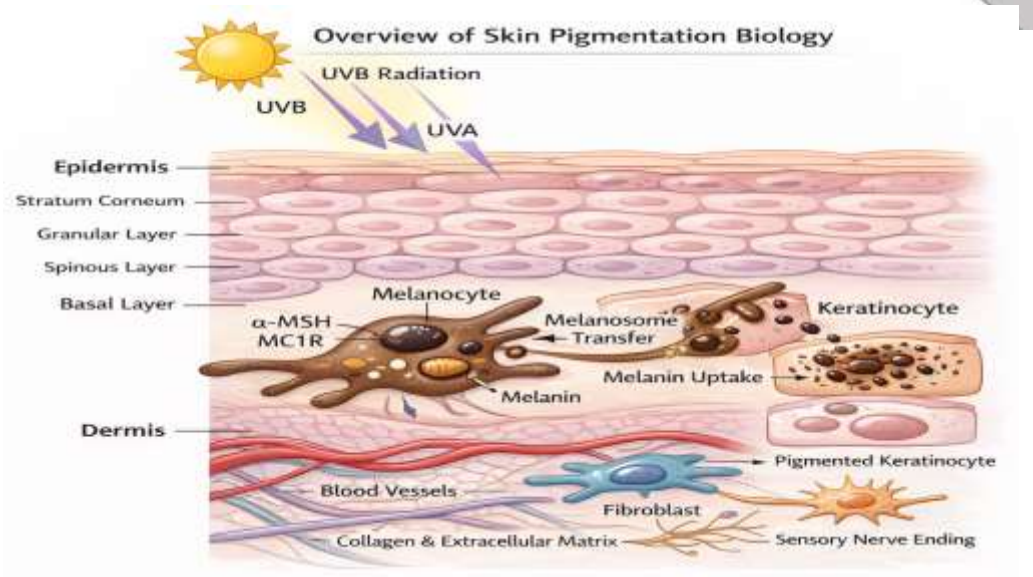
This study covers a wide range of hyperpigmentation diseases, their pathogenic causes, and the development of pharmaceutical and cosmeceutical therapies from traditional to contemporary treatments. Emerging botanical and phytochemical-based treatments, contemporary administration methods, safety concerns, effectiveness, and patient compliance are highlighted, especially with regard to teenage skin health.

Keywords: Cosmetics, Hyperpigmentation, Melanin, Cosmeceutic.

1. Introduction

Skin pigmentation is a fundamental biological trait that determines the colour of human skin and protects against ultraviolet (UV) radiation [1–3].The main factor influencing skin color is melanin, which is generated by melanocytes in the basal layer of the epidermis [4,5].A complex network of biochemical pathways, cellular interactions, and environmental factors regulate melanin synthesis, packaging, transfer, and degradation [6–8]. Melanogenesis is strictly controlled in healthy skin; however, this equilibrium can be upset by genetic predisposition, hormonal changes, inflammation, exposure to UV and visible light, medications, and environmental contaminants, which can result in focal or widespread changes in pigmentation (hypopigmentation or hyperpigmentation) [9–12].

Figure 1. Overview of Skin Pigmentation Biology



Schematic illustration of human skin depicting the epidermal and dermal layers and the epidermal–melanocyte unit. Melanocytes located in the basal layer synthesize melanin within melanosomes in response to ultraviolet radiation via α-MSH–MC1R signaling. Mature melanosomes are transferred through melanocyte dendrites to surrounding keratinocytes, where melanin accumulates above the nucleus to provide photoprotection and determine skin color. Dermal components such as fibroblasts, blood vessels, and extracellular matrix are shown to highlight dermal–epidermal crosstalk influencing pigmentation.

Melasma, lentigines, periorbital hyperpigmentation, and post-inflammatory hyperpigmentation (PIH) are examples of hyperpigmentation disorders that are prevalent dermatological complaints with substantial psychosocial and quality-of-life effects [13–15].Fitzpatrick skin types III–VI, which encompass sizable portions of the South and Southeast Asian, African, Middle Eastern, and Latin American populations, are particularly prone to these problems [16,17]. This review thoroughly examines the biology of pigmentation and the pathophysiology of common hyperpigmentary disorders, with a focus on mechanisms that are therapeutically actionable. In nations like India, cultural and social preferences for lighter skin tones have historically and currently influenced consumer demand for skin-lightening products, driving a large and rapidly growing market for cosmeceuticals and dermatologic interventions designed to reduce pigmentation [18–21]. We evaluate new delivery systems (nanocarriers, liposomes, and sustained-release formulations), talk about limitations and safety concerns, and outline the evidence for traditional treatments (topical agents, chemical peels, laser and light therapies) [22–28].We also thoroughly assess plant-derived tyrosinase inhibitors, antioxidants, and anti-inflammatory chemicals that have promise in preclinical and clinical investigations, given the enormous interest in natural and botanical agents in the Indian and larger Asia-Pacific markets [29–33]. The analysis concludes by contextualizing these clinical and scientific advancements within the Indian cosmetics and personal care business, including data-driven insights into consumer behavior, market size, regulatory considerations, and ethical concerns about colorism and fairness [34–38]. We point out gaps in the literature and suggest priority areas for further research throughout the paper, particularly translational studies that connect molecular discoveries with safe, efficient, and culturally appropriate treatments [39–41].Melanogenesis, sebaceous gland activity, inflammation, and skin-barrier function can all be impacted by the significant physiological and hormonal changes that adolescent skin experiences. Acne is a result of increased androgen activity during adolescence, and UV exposure, mechanical lesion manipulation, and recurrent inflammation can all lead to post-inflammatory hyperpigmentation (PIH). Melanocytes may react aggressively to inflammatory mediators in darker skin phototypes, leading to chronic dermal or epidermal pigment deposition. Melanocyte activity, melanin synthesis, and melanosome transfer to keratinocytes can all be enhanced by cytokines, prostaglandins, reactive oxygen species, and other inflammatory mediators [9, 12].

2. Historical Perspectives on Skin Lightening

Thousands of years have been spent improving complexion and changing skin tone, a technique that has its roots in sociocultural, religious, and aesthetic views from many civilizations. Skin lightening practices have changed over time, moving from traditional plant- and mineral-based preparations to contemporary dermatological and cosmeceutical treatments, as evidenced by archeological, anthropological, and linguistic data. Many of the societal standards of identity, class hierarchy, and beauty that still exist in modern nations are reflected in these rituals.

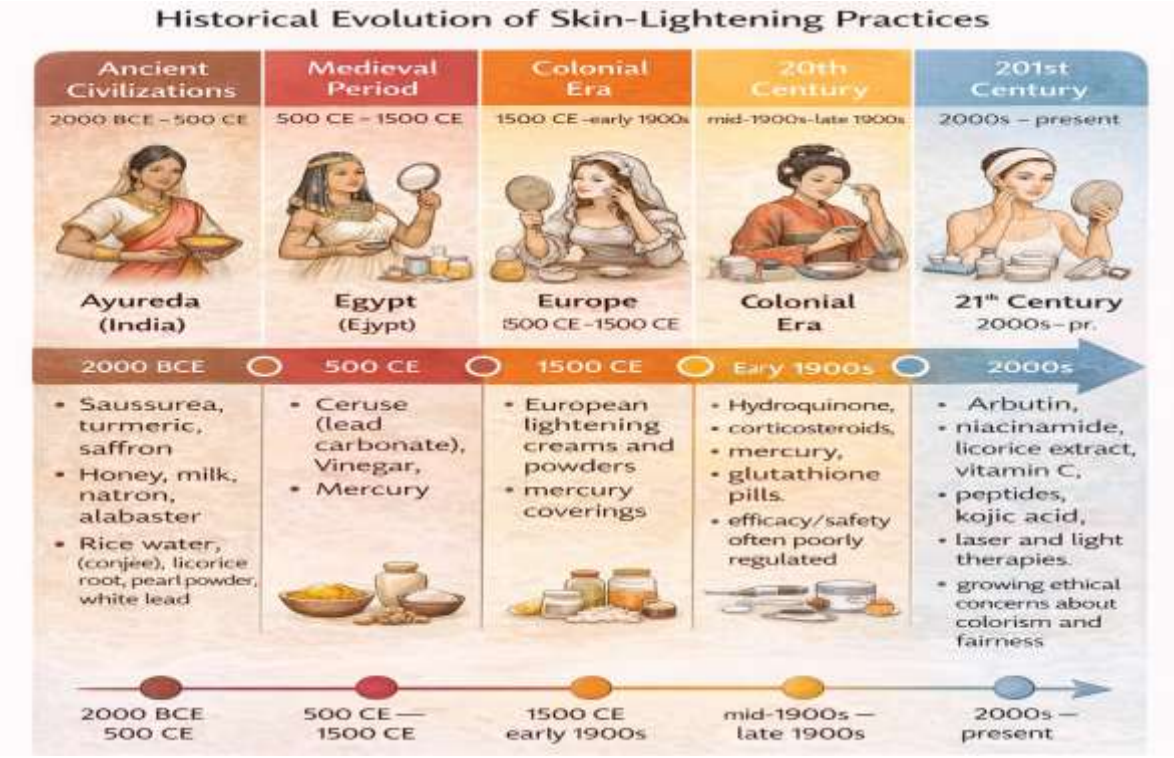


Figure 2. Historical Evaluation of Skin -lightening Practices

2.1 Ancient Civilizations and Early Pigment Modulation Practices

Ancient civilizations like Egypt, India, China, Greece, Rome, and Mesopotamia all used skin-enhancing practices. Complexion enhancement was essential to everyday grooming and spiritual protection, according to evidence from Egyptian tombs, medical papyri, and cosmetic objects [42–44]. To get lighter and smoother skin tones, Egyptians utilized formulas containing honey, milk, natron, lead carbonate, chalk, and herbal extracts [45,46]. Malachite-derived eye pigments and lead-based face paints (ceruse) were both decorative and antibacterial. Numerous herbal combinations intended to improve skin luster and reduce pigmentation are described in ancient Indian Ayurvedic and traditional medicinal books as the Charaka Samhita and Sushruta Samhita [47–49]. For the purpose of depigmenting and improving the complexion, preparations comprising Saussurea lappa, Berberis aristata, Cedrus deodara, Pongamia pinnata, Sesamum indicum, and turmeric were frequently employed [50–52]. Long-standing cultural preferences for lighter skin tones are further shown in the ritualistic usage of saffron (Crocus sativus) for expectant mothers and cosmetic masks made of lentils, honey, and milk [53,54]. Herbal whitening treatments, such as the use of rice water, mulberry root, licorice extract, and pearl powder, which are all recognized for their tyrosinase-inhibitory or brightening properties, were also mentioned in ancient Chinese and Korean medical literature [55–57]. The cultural relationship between lighter skin and beauty, nobility, and purity was reinforced by the Chinese ideal of "jade-like skin" and the Korean concept of "baekji" (clear, white skin). In the past, teenagers and young adults were exposed to traditional skincare techniques employing domestic and botanical products such as rice water, mulberry-derived preparations, turmeric, sandalwood, neem, gram flour, and liquorice. These treatments are a significant source of bioactive substances taken from nature. However, there may be significant differences in their therapeutic efficacy, stability, concentration, and purity. Therefore, either standardised botanical extracts

or scientifically recognised active ingredients like niacinamide, vitamin C derivatives, arbutin, kojic acid, azelaic acid, antioxidants, and specific peptides have been used to create modern cosmeceuticals [47, 49, 52].

2.2 Medieval to Pre-Modern Evolution of Skin-Lightening Practices

Skin whitening remained a social status symbol during the Middle Ages, especially in Europe and Asia. Despite serious health risks, European women used hazardous mixes comprising lead white, vinegar, and mercury salts to get lighter skin during the Middle Ages [58–60]. Similarly, oshiroi, or white face powders made from rice flour and later cosmetics containing lead and mercury, were used by Japanese ladies throughout the Heian period. For centuries, these formulas were in use. Depigmenting procedures became entwined with changing sociocultural norms in South Asia. The link between a fair complexion and a better social status was strengthened by the advent of Aryans and the caste system that followed [61–63]. The use of herbal pastes, oils, and washes in Ayurvedic beauty rituals has increased. Common home techniques included the use of neem-based formulations, sandalwood paste, gram flour combinations, and face masks based on turmeric [64–66].

2.3 Colonial Influence and the Institutionalization of Colourism

Colourism's sociocultural roots were strengthened by colonial rule, especially British dominance in India and American and European presence throughout Asia and Africa [67–69]. Colonial myths contributed to enduring psychological and societal biases by portraying people with lighter skin as superior, intelligent, and civilized. During the 19th and early 20th centuries, these factors influenced the early commercial cosmetic sector, which saw a rise in the popularity of imported lotions and powders that made claims of "skin lightening,""blemish removal," and "complexion cleansing" [70,71].

A multigenerational desire for lighter skin was fostered in India by colonial-era occupational choices, social mobility views, and marriage conventions tied to fairness [72–74]. The fairness cream boom in post-independence India was made possible by these ideas.

2.4 20th Century: Industrialization and Commercial Skin-Lightening Products

Traditional formulas gave way to chemically manufactured substances in the worldwide cosmetics sector over the 20th century. Skin-lightening products were transformed with the advent of hydroquinone, mercury salts, corticosteroids, and resorcinol derivatives [75–77]. Asia, Africa, and the Middle East saw intense marketing of early commercial goods from Europe and America. Brands that promoted fairness as the cultural standard of success and beauty, such Afghan Snow, Vicco, and Fair & Lovely (1975), dominated the Indian market [78–80]. Colorism was further institutionalized by the marketing narrative, which frequently presented lighter skin as a means of achieving better job and marriage possibilities.

2.5 Contemporary Era: Ethical Awareness and the Shift Toward “Brightening”

Significant changes have occurred in the 21st century as a result of growing ethical concerns about colorism, consumer awareness of chemical safety, desire for natural and herbal products, tighter regulations, and international movements for representation and inclusivity. Marketing messaging has changed from "whitening" to "brightening,""glow enhancement," and "improved skin clarity" as a result of these modifications [81–83]. Older hazardous substances are rapidly being replaced with plant-derived antioxidants, tyrosinase inhibitors, peptides, niacinamide, vitamin C derivatives, and actives based on nanocarriers in modern formulations [84–88]. By emphasizing healthy, even-toned skin rather than fair complexion, the Korean beauty (K beauty) and Japanese beauty (J beauty) businesses are now at the forefront of innovation. The demand for pigmentation-modulating goods is growing worldwide, particularly in East Asia, Africa, and India, despite shifting terminology [89–92]. The necessity for safe, scientifically supported, and culturally sensitive methods of managing pigmentation is highlighted by this ongoing need.

3. Hyperpigmentation Disorders

A collection of disorders known as hyperpigmentation are defined by a concentrated, patchy, or diffuse darkening of the skin brought on by an excess of melanin deposition. Changes in melanocyte activity, disturbed melanin transfer, post-inflammatory processes, photodamage, or systemic effects are the causes of these illnesses. People with darker skin phototypes (Fitzpatrick III–VI) are disproportionately affected, which frequently leads to severe psychosocial burden and therapeutic difficulties [93–96].

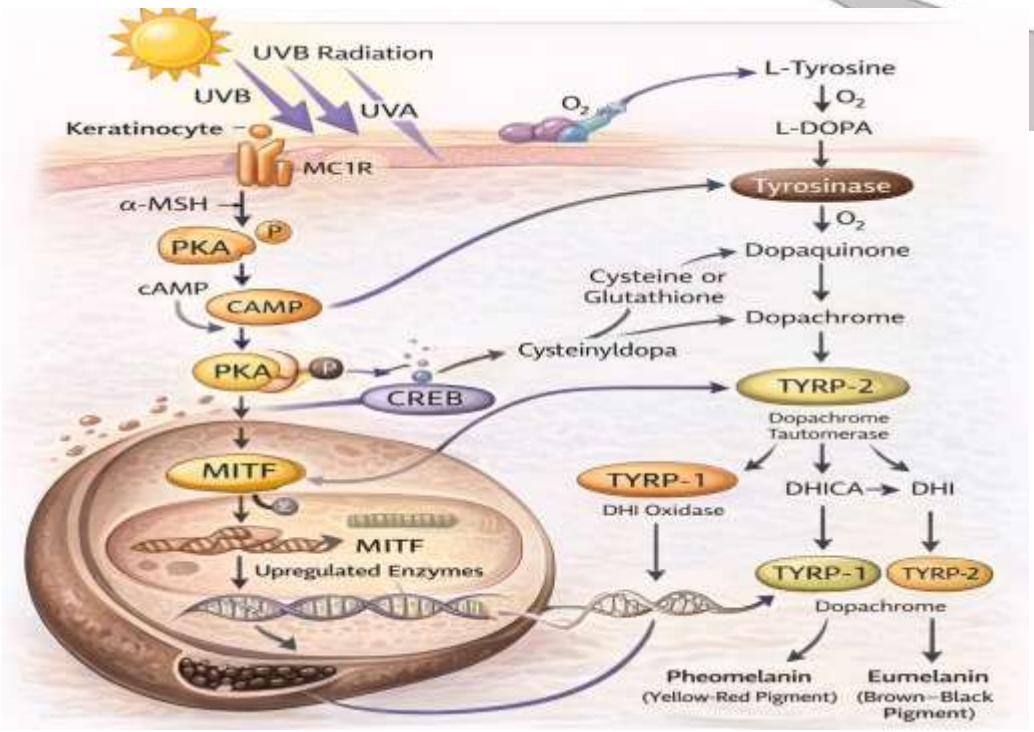


Figure 3. Melanogenesis Pathway and Key Regulatory Enzymes Biochemical pathway illustrating tyrosinase. TYRP-1. TYRP-2, MITF regulation, and the influence of UV-induced signaling (a-MSH-MC1R pathway).

3.1 Melasma (Chloasma)

Melasma is a persistent, recurrent hypermelanosis that typically affects facial regions exposed to the sun. It is common among Asian, Hispanic, and Middle Eastern cultures and primarily affects women of reproductive age [97,98]. Based on the distribution of lesions, the disorder is clinically categorized into centrofacial, malar, and mandibular patterns.

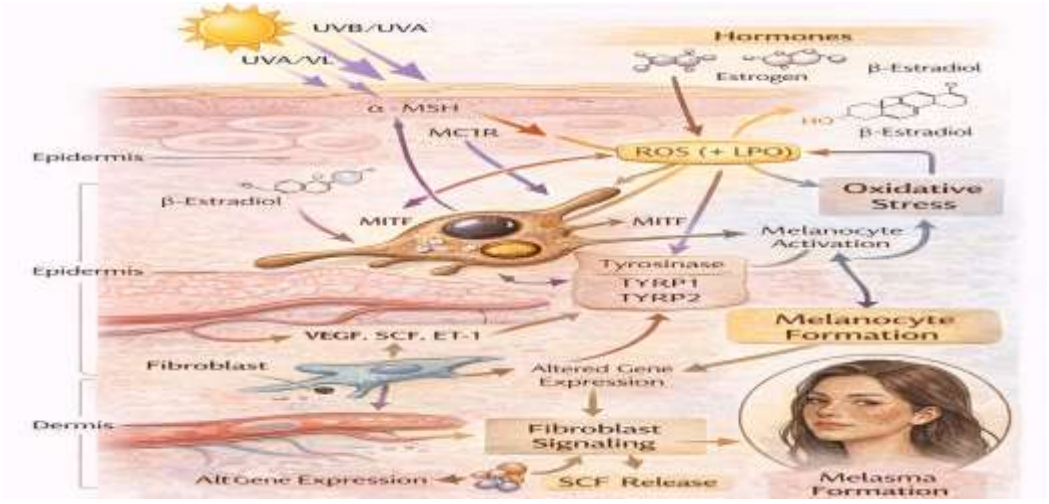


Figure 4.Pathophysiology of Melasma

Multifactorial mechanism showing roles of UV/VL radiation, hormones, vascular factors, oxidative stress, fibroblast signaling, and melanocyte activation.

3.1.1 Etiopathogenesis

Melasma is a complex condition. UV radiation (the main aggravating factor) [99], visible light (VL) and infrared radiation [100], hormonal factors like pregnancy, oral contraceptives, and hormone therapy [101,102], genetic predisposition (up to 60% of cases have a positive family history) [103], inflammatory mediators, vascular

alterations, and fibroblast-derived signals [104,105] are all recognized triggers. Histopathology shows improved vascularity, solar elastosis, epidermal thinning, and increased melanogenesis. Recent studies have demonstrated the importance of oxidative stress pathways, endothelin-1, stem cell factor (SCF), and Wnt/β-catenin signaling in the pathophysiology of disease [106–108].

3.1.2 Clinical Features and Epidemiology

The prevalence of melasma among dermatological outpatients varies from 1 to 30%, with Southeast Asia and India having some of the highest rates [109,110]. It manifests as symmetrical, hyperpigmented patches and macules with uneven boundaries and a gradual progression. Heat and prolonged sun exposure are well-known aggravating variables.

3.2 Post-Inflammatory Hyperpigmentation (PIH)

PIH is an acquired hypermelanosis that appears after irritation, injury, or inflammation of the skin. Because of the strong melanocyte response to inflammation, it is particularly prevalent in those with darker skin [111–113].

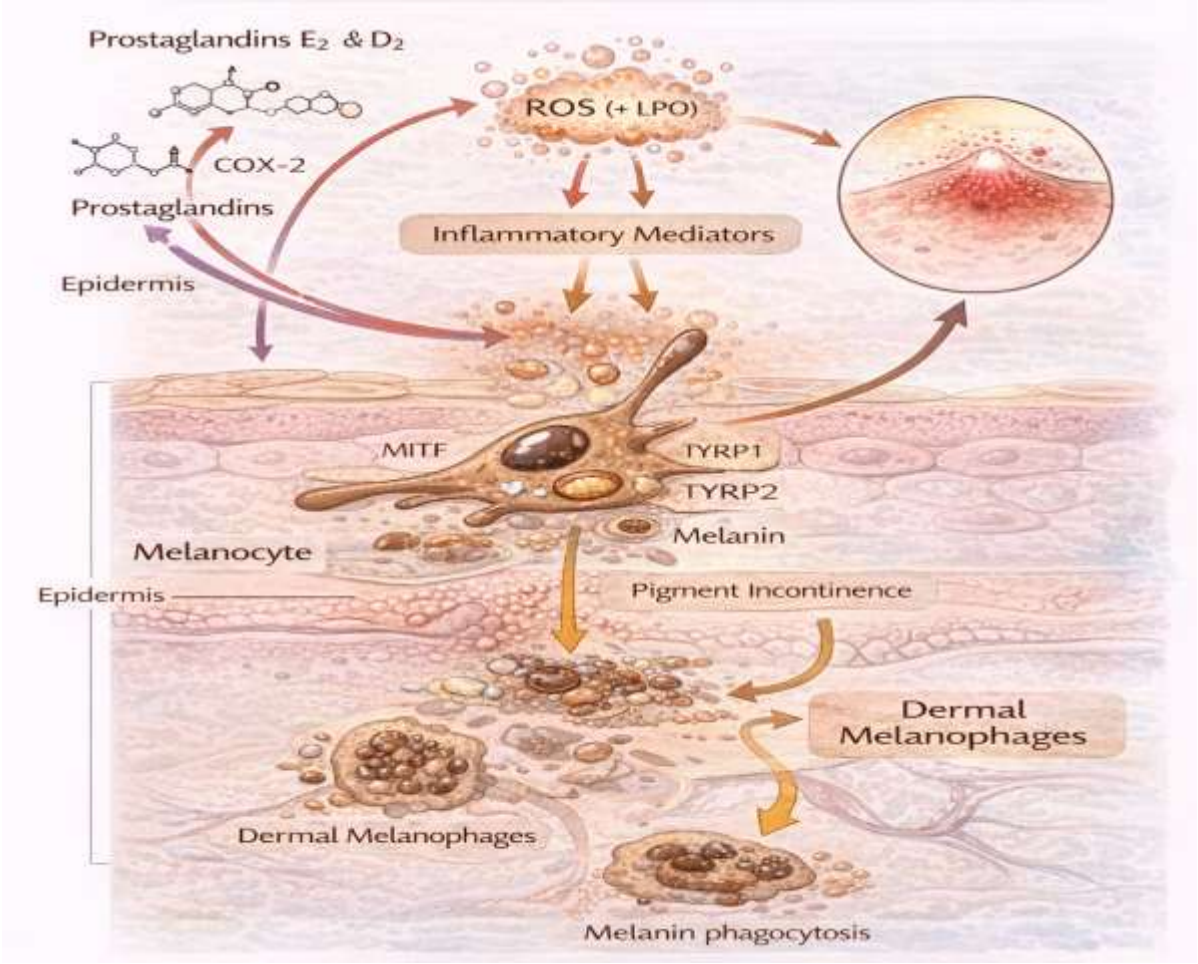


Figure 7. Mechanisms of Post-Inflammatory Hyperpigmentation (PIH)

Inflammation-driven melanogenesis illustrating cytokines, prostaglandins, ROS, pigment incontinence, and dermal melanophages.

3.2.1 Etiology and Mechanisms

Acne, psoriasis, eczema, contact dermatitis, cosmetic operations, burns, and even slight trauma are common causes. Melanocyte proliferation and melanin synthesis are stimulated during inflammation by cytokines, prostaglandins, leukotrienes, thromboxanes, and reactive oxygen species (ROS) [114,115]. There are two main patterns: Dermal PIH melanophages in the upper dermis after pigment incontinence (blue-grey) and epidermal

PIH excess melanin in the basal and suprabasal layers (dark brown). PIH is more severe and chronic in dark-skinned populations, frequently lasting months to years [116–118].

3.3 Solar Lentigines (Actinic Lentigines)

Due to significant UV exposure, solar lentigines, which are well-defined hyperpigmented macules brought on by cumulative sun exposure, are widespread in middle-aged and older people, particularly those with lighter skin types, but they are also common in India [119,120].

3.3.1 Pathogenesis

UV exposure causes overproduction of melanin, increased melanocyte density, and epidermal hyperplasia. Melanogenic regulator mutations brought on by actinic exposure increase the risk of cancer [121–123]. Lentigines have extended rete ridges, elevated basal layer melanin, and sporadic melanocyte hyperplasia on histological examination.

3.4 Periorbital Hyperpigmentation (POH)

POH manifests as patchy or diffuse brown to dark brown discoloration surrounding the eyelids. It has a complex origin and is sometimes referred to as "dark circles."

3.4.1 Causes and Mechanisms

The following are important contributing factors: Hormonal fluctuations and nutritional deficiencies [128], excessive cosmetic use and photoaging [129], post-inflammatory changes from atopic dermatitis or rubbing [126], thin periorbital skin and visible vasculature [125], genetic predisposition [124], and sleep deprivation, fatigue, and stress [127]. Due to lifestyle choices, anemia, and genetic predisposition, POH is particularly common in Indian women.

3.5 Psychosocial Impact of Hyperpigmentation

Hyperpigmentation has a substantial impact on social interactions, confidence, and emotional health in all subtypes. According to a number of studies, those who are impacted have greater anxiety levels, worse self-esteem, and a dependence on cosmetic masking [130–132]. The emotional strain is disproportionately higher in societies like India where lighter skin is prized [133–135].

4. Factors Influencing Hyperpigmentation

When melanocyte homeostasis is disturbed by internal or external stressors, increased melanin production, abnormal melanin distribution, or pigment retention result in hyperpigmentation. Choosing focused therapy approaches requires an understanding of these triggers.

4.1 Ultraviolet (UV) Radiation–Induced Hyperpigmentation

The most powerful and well-known stimulant of melanogenesis is ultraviolet light. Melanin synthesis, melanosome transfer, and melanocyte proliferation are all improved by UV exposure, which also activates keratinocyte–melanocyte signaling pathways [136–138].

4.1.1 Mechanisms

UVB (280–320 nm) causes DNA damage and p53 activation, which increases pro-opiomelanocortin (POMC) transcription. POMC is then cleaved into α-MSH, which binds to melanocytes' MC1R [139,140]. Tyrosinase is stimulated and melanogenesis is increased by UVA (320–400 nm), which penetrates deeper and produces reactive oxygen species (ROS) [141]. Melanogenic mediators, including endothelin-1, stem cell factor, nitric oxide, leukotrienes, and prostaglandins, are all upregulated by UV exposure and contribute to hyperpigmentation [142–144]. Persistent pigment darkening, photoaging, solar lentigines, and aggravation of melasma are all consequences of prolonged UV exposure.

4.2 Drug- and Chemical-Induced Hyperpigmentation

Many topical and systemic medications can deposit pigment in the dermis or epidermis or promote melanogenesis. Up to 20% of acquired pigmentation problems in dermatology are caused by drugs [145–147].

4.2.1 Common Culprit Drug Classes

Antimalarials: hydroxychloroquine and chloroquine (which cause drug-melanin complexes and dermal melanosis) [149,150], NSAIDs: naproxen-induced fixed drug eruptions that cause PIH [151], antiretrovirals: zidovudine and emtricitabine [152], cytotoxic drugs: bleomycin and cyclophosphamide [153], and antibiotics: minocycline and tetracycline [148]. Heavy metals: silver (argyria), bismuth, and arsenic [154,155].

4.2.2 Mechanisms

Levodopa is one example of a direct melanin production stimulant [156]. medication accumulation in skin tissues, alterations in pigmentation following drug eruptions, Following venous insufficiency or ecchymosis, iron deposition.

4.3 Endocrine and Hormonal Influences

Pigmentation is significantly influenced by hormone control. High levels of progesterone and estrogen raise tyrosinase activity and improve melanocyte sensitivity to UV light [157,158].

4.3.1 Pregnancy (Melasma Gravidarum)

Elevated levels of progesterone, estrogen, and melanocyte-stimulating hormone (MSH) during pregnancy can cause or exacerbate melasma [159,160].

4.3.2 Thyroid and Adrenal Disorders

Diffuse hyperpigmentation can be brought on by hypothyroidism or hyperthyroidism [161]. Addison's illness results in global bronzing because of an excess of ACTH and MSH [162].

4.4 Inflammation-Induced Hyperpigmentation

Melanocyte dysregulation is brought on by inflammation via oxidative mediators and cytokines. Prostaglandins (PGE2, PGF2α) [163], leukotrienes (LTB4, LTC4) [164], interleukins (IL 1, IL 6), and TNF α [165], as well as ROS and nitric oxide [166], are important mediators of inflammation. PIH results from these substances' stimulation of melanocyte dendricity, elevation of tyrosinase activity, and promotion of melanin transfer to keratinocytes.

4.5 Pollution-Induced Hyperpigmentation

Pigmentary disorders are greatly exacerbated by environmental pollution, particularly in metropolitan populations. Oxidative stress, inflammation, and barrier disruption are caused by pollutants such nitrogen dioxide (NO_2), particulate matter (PM2.5), ozone (O_3), and polycyclic aromatic hydrocarbons (PAHs) [167–169].

4.5.1 Biological Effects

Melanogenesis-causing Aryl Hydrocarbon Receptor (Ahr) Pathway Upregulation [170], Matrix Metalloproteinases-causing Photoaging, Increased Lipid Peroxidation and DNA Damage [171], and Enhanced Sensitivity to UV Radiation Studies in China, India, and Korea Connect High Pollution Exposure to Worsening Melasma, Lentigines, and Overall Increased Pigmentary Irregularities [172,173].

4.6 Blue Light–Induced Hyperpigmentation

Sunlight and digital screens emit high-energy visible (HEV) blue light (390–500 nm), which has been found to be a major cause of pigmentation alterations, especially in darker skin types [174–176].

4.6.1 Mechanisms

Blue light accelerates skin aging by causing oxidative stress and ROS formation [177], stimulating the melanocyte opsin-3 (OPN3) receptor to increase melanogenesis [178], and causing lipid and protein oxidation. Frequent exposure can exacerbate melasma and PIH by causing chronic and uneven pigmentation.

5. Treatment Strategies for Hyperpigmentation

A multimodal strategy that is customized to the underlying etiology, skin type, and chronicity of the problem is necessary for the management of hyperpigmentation. The goals of treatment are to either kill melanin-rich cells, decrease melanogenesis, speed up epidermal turnover, or prevent pigment transfer. Combining treatments frequently results in better results, especially for resistant disorders like post-inflammatory hyperpigmentation (PIH) and melasma [179–181].

5.1 First-Line Topical Therapies

Because they are readily available and effective, topical depigmenting agents continue to be the cornerstone of treatment.

5.1.1 Hydroquinone (HQ)

For hyperpigmentation, hydroquinone (2–4%) is regarded as the gold standard. It works by boosting melanocyte cytotoxicity, changing the production of melanosomes, and blocking tyrosinase [182,183]. Benefits include quick start and excellent effectiveness. Limitations irritating dermatitis, rebound hyperpigmentation, exogenous ochronosis with continuous use [184].

5.1.2 Azelaic Acid

Azelaic acid (15–20%) has anti-inflammatory properties, normalizes keratinization, and lowers tyrosinase activity. beneficial for skin prone to acne, melasma, and PIH [185,186].

5.1.3 Kojic Acid

Kojic acid, which comes from Aspergillus species, chelates copper at the tyrosinase active site. 1–4% concentrations are frequently employed [187]. Long-term use is restricted by its instability and risk for sensitization.

5.1.4 Arbutin and Deoxyarbutin

Tyrosinase is safely inhibited by arbutin (α and β forms), a naturally occurring glycosylated hydroquinone derivative [188]. Deoxyarbutin is more effective and stable but may cause discomfort.

5.1.5 Retinoids

Tretinoin, adapalene, and tazarotene improve desquamation, speed up epidermal turnover, and increase other agents' penetration [189,190]. Additionally, retinoid stimulates the manufacture of collagen, which is beneficial for photoaged skin.

5.1.6 Niacinamide (Vitamin B3)

Niacinamide has anti-inflammatory and barrier-restorative properties while interfering with the transfer of melanin from melanocytes to keratinocytes [191,192]. often utilized in formulations for cosmetics.

5.1.7 Vitamin C and Derivatives

Ascorbic acid scavenges ROS, inhibits tyrosinase, and lowers oxidized dopaquinone [193]. More stability is provided by derivatives like ascorbyl glucoside and magnesium ascorbyl phosphate.

5.1.8 Botanicals and Phytochemicals

The following plant-derived ingredients are becoming more and more relevant in clinical settings: licorice extract (glabridin), which is a powerful anti-inflammatory and tyrosinase inhibitor [194], mulberry extract, which inhibits the synthesis of melanin [195], green tea polyphenols, which are antioxidants and suppress melanogenesis [196], aloesin (Aloe vera), which is a competitive tyrosinase inhibitor [197], and resveratrol, which prevents oxidative damage and melanin production [198].

5.2 Combination Therapy

Combining drugs improves treatment results and lessens side effects from monotherapy.

5.2.1 Triple Combination (TC) Cream

The best topical treatment for melasma is still the Kligman–Willis formula, which consists of hydroquinone 4% + tretinoin 0.05% + fluocinolone acetonide 0.01% [199,200]. Melanogenesis, inflammation, and epidermal turnover time are all decreased by it. Because of the potential for discomfort and side effects from steroids, long-term use should be done with caution.

5.2.2 Other Effective Combinations

Retinoids + vitamin C [203], Azelaic acid + niacinamide [204], Kojic acid + arbutin [202], HQ + glycolic acid [201].

5.3 Chemical Peels

Chemical peels promote regulated wound healing, decrease epidermal pigment, and speed up exfoliation. They are frequently used for lentigines, PIH, and melasma.

5.3.1 Alpha Hydroxy Acids (AHAs)

Peels with lactic acid (30–50%) and glycolic acid (20–70%) increase epidermal turnover and pigment dispersion [205,206]. For better effects, glycolic acid is frequently mixed with azelaic acid or HQ.

5.3.2 Beta Hydroxy Acids (BHAs)

Due to its lipid-soluble nature, salicylic acid (20–30%) is useful for PIH associated with acne [207]. It reduces inflammation and exfoliates comedones.

5.3.3 Trichloroacetic Acid (TCA)

Although they can lighten epidermal problems, medium-depth TCA peels (15–35%) can cause PIH in darker skin types [208]. Fitzpatrick IV–VI is best treated with superficial TCA mixes.

5.4 Laser and Light-Based Therapies

Laser therapy targets melanin chromophores, inducing selective photothermolysis.

5.4.1 Q-Switched (QS) Lasers

The deeper penetration and lower risk of PIH make QS Nd:YAG (1064 nm) a popular treatment for melasma and lentigines [209,210]. A series of low-fluence treatments, or "laser toning," provide incremental progress.

5.4.2 Fractional Lasers

Fractional CO_2 and Er:YAG lasers enhance pigmentation and dermal remodeling, but if not utilized carefully, they can exacerbate melasma [211]. Safer substitutes are non-ablative fractional lasers (1550 nm).

5.4.3 Intense Pulsed Light (IPL)

IPL targets hemoglobin and melanin to successfully treat lentigines and photoaging [212]. Darker skin types are more susceptible to PIH.

5.4.4 Limitations and Risks

Rebound hyperpigmentation may occur at laser-treated locations, particularly in melasma. Strict photoprotection is necessary [213].

5.5 Emerging and Advanced Therapies

5.5.1 Peptide-Based Inhibitors

Tyrosinase and melanocyte signaling pathways are inhibited by peptides like oligopeptide-68, melanostatin-5, and nonapeptide-1 [214].

5.5.2 Nanocarrier Delivery Systems

The penetration and stability of depigmenting actives are improved by liposomes, ethosomes, nanoemulsions, and solid-lipid nanoparticles [215–217].

5.5.3 Tranexamic Acid (TXA)

Topical, oral, and intradermal TXA has emerged as a promising therapy for melasma. It reduces vascularization and melanocyte activity by inhibiting plasminogen activation [218–220].

5.5.4 Antioxidant and Anti-inflammatory Agents

Anti-melanogenic action and oxidative stress reduction are supported by compounds such vitamin E, ferulic acid, niacinamide, curcumin, and cysteamine [221–223].

5.6 Sun Protection: The Cornerstone of Management

For adolescent skincare, the therapeutic objective should be restoration of an even skin tone while preserving normal melanogenesis and maintaining barrier integrity, rather than aggressive skin lightening. Because UV radiation can exacerbate PIH and promote melanogenesis, preventive measures are crucial, especially broad-spectrum photoprotection. Thus, the creation of mild, evidence-based, non-irritating and culturally sensitive cosmeceuticals, is an important means of improving adolescent skin health and reducing the long-term psychosocial impact of visible hyperpigmentation [136-144, 224].Pigmentation therapy requires the use of broad-spectrum sunscreens (SPF 30 or greater). The best filters are those that block visible light, UVB, and UVA rays [224,225]. Tinted sunscreens with iron oxide enhance protection against HEV light, which is important for melasma and PIH [226].

6. Skin Brightening vs. Lightening vs. Whitening

Although the cosmetic and dermatological industries commonly use the terms "whitening,""lightening," and "brightening" interchangeably, they refer to different processes, customer expectations, and moral considerations. Accurate product formulation, regulatory compliance, and culturally sensitive communication all depend on an understanding of these distinctions [227–229].

6.1 Skin Whitening

The word "whitening" has historically been used to describe the widespread suppression or elimination of melanin, frequently with the use of strong chemicals. Large portions of the body or face will have lighter skin tones as a result of whitening solutions' reduction of overall skin pigmentation.

6.1.1 Mechanism

Tyrosinase suppression (e.g., hydroquinone, arbutin, kojic acid) [230–232], melanocyte cytotoxicity (high-dose hydroquinone) [233], interference with melanosome production or transfer [234], and accelerated epidermal turnover [235] are the usual mechanisms by which whitening agents decrease melanogenesis.

6.1.2 Safety Concerns

High-potency steroids, uncontrolled hydroquinone, and mercury salts in particular are aggressive whitening agents that have been connected to exogenous ochronosis [236], mercury nephrotoxicity and neurotoxicity [237], skin barrier damage and atrophy [238], and rebound hyperpigmentation. Because of safety concerns, mercury and hydroquinone have been regulated in a number of nations.

6.2 Skin Lightening

Instead of a general alteration in skin color, skin whitening refers to the focused decrease of hyperpigmented lesions. The aim is to increase clarity, treat melasma or PIH, and restore uneven tone without altering the person's natural complexion.

6.2.1 Mechanism

Overactive melanocytes, post-inflammatory pigment deposition, and UV-induced melanin pathways are all addressed by lightening treatments [239–241]. Azelaic acid, niacinamide, vitamin C derivatives, retinoids, and tranexamic acid are common lightening substances [242,243]. Thus, lightening is seen as medicinal, particularly in dermatology.

6.3 Skin Brightening

Skin brightening focuses on restoring radiance and luminosity rather than altering melanin content. It improves skin reflectivity, texture, and overall glow.

6.3.1 Mechanism

Brightening products improve: hydration and barrier function [246], antioxidant protection [245], epidermal turnover [244], and dullness caused by keratinocyte accumulation [246]. AHAs, BHAs, vitamin C, vitamin E, niacinamide, and exfoliating enzymes are frequently used in brightening.

6.4 Socio-cultural and Ethical Considerations

Global cultural standards are deeply ingrained in the practice of skin tone modification. Lighter skin has long been associated with privilege, attractiveness, and socioeconomic advantage in South Asia, East Asia, Africa, and some regions of the Middle East [247–249]. Colorism, or prejudice based on skin tone within the same ethnic group, has been exacerbated by these linkages. Significant repercussions include unfair beauty standards, pressure to use dangerous products, and low self-esteem and psychological anguish [250].

6.4.1 Evolving Market Language

Global cosmetic companies have changed their names in response to growing criticism of fairness-based marketing: "Whitening" becomes "Brightening" or "Tone-evening,""Fairness cream" becomes "Spot corrector" or "Pigment-control cream" [251–253]. Nowadays, regulatory bodies in the EU, South Korea, Japan, and India support culturally neutral language.

6.5 Regulatory Framework and Safety Guidelines

Regulators emphasize consumer safety and truthful advertising.

6.5.1 Hydroquinone Regulations

In many nations, over-the-counter cosmetics are prohibited; only prescription use is permitted in the EU, USA, and India [254].

6.5.2 Mercury Ban

Under the Minamata Convention, WHO prohibits methylmercury and inorganic mercury salts [255].

6.5.3 Labeling and Claims

Products must refrain from making false claims like "immediate fairness." Rather, it is advised to use verified statements such "improves radiance" or "reduces dark spots" [256,257].

6.5 Modern Reformulations and Industry Trends

Safer botanical extracts (licorice, mulberry, and Centella) [258–260], non-hydroquinone brighteners (niacinamide, TXA, and resorcinol derivatives) [261], nanocarrier systems for improved penetration [262], and sustainable source of natural active ingredients are the main features of modern goods. Global trends have been influenced by the Asian beauty industries (K-beauty and J-beauty), which have popularized the idea of "healthy glow" rather than fairness [263,264].

7. Global and Indian Cosmetic Market Scenario

Over the past 20 years, the global market for skincare and pigmentation-modulating products has grown dramatically due to changes in sociocultural views of beauty, technological advancements, and growing consumer knowledge. One of the markets with the quickest rates of growth in the global beauty and personal care (BPC) sector is India [265–267].

7.1 Global Market Overview

In 2023, the worldwide skincare industry reached a valuation of over USD 150 billion, and it is still expanding at a compound annual growth rate (CAGR) of 5–6% [268]. Brightening, anti-aging, and tone-evening pigmentation-correcting cosmetics make up a significant portion of global sales.

7.1.1 Major Growth Drivers

Hyperpigmentation that is more common due to pollution, UV exposure, and hormones [269]. growth of dermocosmetics and skincare products with medical endorsements, Growing use of non-invasive dermatological treatments (IPL, laser toning, and chemical peels) [270] [271] K-beauty and J-beauty's influence on glass-skin and brightening trends [272], expanding global market for male grooming [273]. With more than 55% of global usage, Asia-Pacific continues to be the biggest market for skin-lightening and brightening products [274]. Consumer demand for pigmentation-modulating goods is dominated by countries such as South Korea, Japan, China, Thailand, the Philippines, and India.

7.2 Indian Cosmetic Industry Landscape

India’s beauty and personal care market is valued at USD 10–12 billion (2024) and is expected to grow at a CAGR of 10–12% over the next decade [275,276]. Urbanization, increased disposable income, and Western beauty influences have significantly shaped purchasing trends.

7.2.1 Market Segmentation

Facial care (34%), body care (20%), hair care (25%), hand/foot care (10%), and cosmetics (11%) make up the Indian skincare market [277]. Among these, skin-brightening and pigmentation-correcting products are among the fastest-growing subsectors.

7.3 Demand for Skin-Lightening Products in India

One of the biggest markets for skin-lightening creams worldwide is in India. Due to sociocultural conceptions that associate fairness with attractiveness, confidence, marriage prospects, and upward mobility, the industry has historically had double-digit growth rates [278,279].

7.3.1 Key Consumer Segments

Young ladies (ages 18 to 35), Professionals in the workforce and students, Future brides, populations living in cities and semi-urban areas, Male consumers are growing, particularly in the grooming sector [280].

7.4 Impact of Colourism on Market Behaviour

Consumer tastes are still heavily influenced by colorism. In the past, fairness-related ads have dominated Indian media, which has increased demand for whitening creams [281]. However, a change in nomenclature toward "brightening" and "glow-enhancing" products has resulted from regulatory action and public outcry [282].

7.5 Key Industry Players

Hindustan Unilever (Glow & Lovely) [283], L'Oréal Paris (melanin-targeting serums), Himalaya Herbals, Patanjali Ayurved (herbal formulations), Forest Essentials, Maearth, Minimalist, and Dot & Key (dermocosmetic brands) are some of the major national and international brands that dominate the Indian pigmentation-care market. Through internet channels, dermatological companies including CeraVe, La Roche-Posay, Bioderma, Sesderma, and The Ordinary are growing quickly in India.

7.6 Regulatory and Safety Landscape in India

7.6.1 BIS and CDSCO Regulations

The Central Drugs Standard Control Organization (CDSCO) and the Bureau of Indian Standards (BIS) oversee the safety of cosmetic ingredients. The following are highlighted in recent guidelines: Limitations on cosmetics containing hydroquinone, mercury, and steroids [284]; Mandatory labeling, safety testing, and prohibition of deceptive claims [285]; Penalties for advertisements that promote fairness as a social advantage or success indicator [286].

7.7 Online Retail Revolution

The cosmetics industry in India has changed as a result of e-commerce. Platforms like Nykaa, Amazon India, Flipkart, Tata 1mg, and Myntra have provided nationwide access to luxury and international skincare products [287].Online retail accounts for about 30% of skincare sales and is anticipated to reach 40–45% by 2030 [288].

7.8 Future Projections

By 2028, the Indian skin-brightening market is anticipated to reach USD 20 billion, driven by robust expansion in the following areas: dermatology, prescription-grade pigmentation therapies, clean and sustainable beauty formulations, male grooming products, and products that target pigmentation caused by pollution and blue light [289–292]. The future of pigmentation control will be shaped by cutting-edge research in peptides, stabilized vitamin C derivatives, nanotechnology, and botanical actives.

8. Conclusion

Hyperpigmentation disorders such as melasma and PIH arise from a multifactorial interplay of genetic predisposition, inflammation, photodamage, hormonal influences, and environmental stressors.Melanogenesis involves not only melanocyte-specific enzymes but also keratinocyte signaling, fibroblast–melanocyte crosstalk, oxidative stress pathways, and mechanisms driven by visible light, according to developments in molecular dermatology. The evidence currently available supports a multimodal treatment approach that includes rigorous photoprotection, such as visible-light blocking sunscreens, topical depigmenting agents (hydroquinone, azelaic acid, tranexamic acid, niacinamide, and cysteamine), and procedural therapies (chemical peels, lasers, and microneedling-assisted delivery). Recurrence and treatment resistance are nevertheless frequent despite better therapeutic results, underscoring the necessity of patient-specific regimens and long-term maintenance. Promising adjuncts for safer and more efficient pigment regulation include autophagy modulators, JAK–STAT pathway inhibitors, resorcinol derivatives, nanoformulations, and antioxidant-rich dermocosmetics. To improve treatment algorithms and provide

interventions that address the dermal and epidermal aspects of hyperpigmentation, translational research and carefully planned clinical trials are crucial.

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