

Fundamentals of Hyperpigmentation Disorders: Causes, Concerns, and Treatment Strategies, Particularly in Skin of Color Patients

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ABSTRACT

Background: Pigmentary disorders, including hyperpigmentation, are among the most common dermatologic diagnoses in individuals with skin of color (SOC), particularly individuals with African, Asian, or Hispanic heritage, and often carry a significant quality-of-life burden. Although the evidence base has grown, gaps persist in epidemiology, in pathophysiology-driven treatment selection, and in the appropriate representation of Fitzpatrick skin types III to VI in clinical studies.

Methods: A comprehensive review of PubMed and Medline-indexed hyperpigmentation literature published between 2000 and 2026 was reviewed, with priority given to randomized controlled trials, meta-analyses, large epidemiologic series, and consensus statements.

Results: Our current understanding of hyperpigmentation has extended well beyond melanocyte hyperactivity alone. Across the major hyperpigmentation disorders, visible light acting through opsin-3 (OPN3), inflammatory and hormonal signaling, and, in melasma, basement-membrane disruption with a senescent dermal microenvironment contribute to pigment formation and treatment resistance, findings that highlight the importance of both UV radiation and visible-light photoprotection. Clinical and meta-analytic evidence supports a multimodal therapeutic approach, consistent with recent international consensus recommendations. Quality-of-life impairment is consistent across populations, a pattern that should inform counseling, while commonly used diagnostic tools and machine-learning algorithms continue to underperform in skin of color.

Discussion: Treatment of hyperpigmentation remains a multimodal therapy with the need for routine assessment of patient-reported quality of life impact.

Conclusion: Closing the evidence gap for skin of color is the next priority in treatment of hyperpigmentation and will require inclusive trial design, validated diagnostic tools, and standardized outcome measures.

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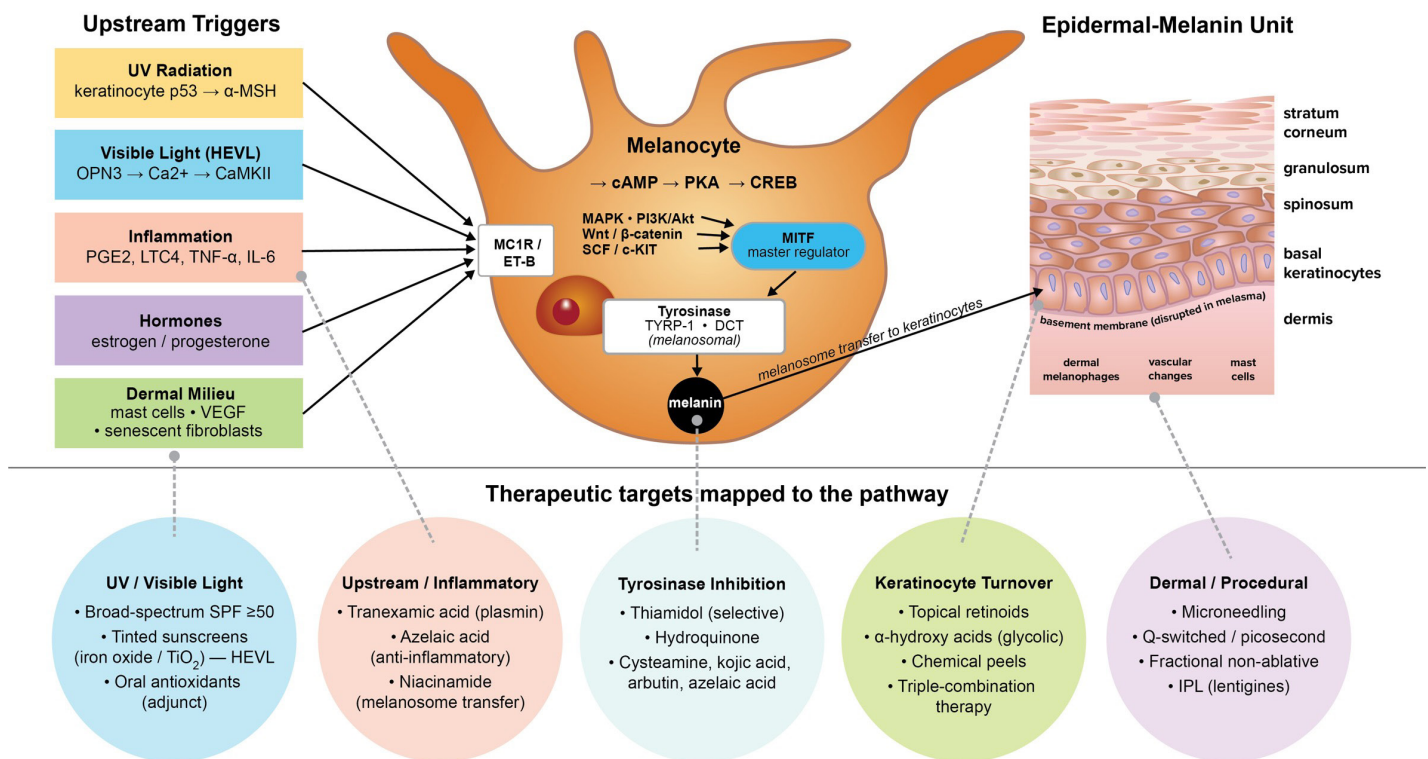
INTRODUCTION

Pigmentary disorders, including cutaneous hyperpigmentation, are among the most common dermatologic diagnoses in individuals with SOC, particularly individuals with African, Asian, or Hispanic heritage.^{1,2} The principal hyperpigmentation conditions include melasma, post-inflammatory hyperpigmentation (PIH), solar lentigines, drug-induced and infectious pigmentary changes, maturational dyschromia, and acquired dermal macular hyperpigmentation (ADMH). In a cross-sectional analysis of 41,283 patients, melasma was significantly more prevalent in

Asian and Hispanic populations, and was strongly comorbid with rosacea and atopic dermatitis.³

For the clinician, the central difficulty is rarely diagnosis; it is the management of a chronic and relapsing condition, the disproportionate burden carried by patients with deeply pigmented skin, and the gap between the populations enrolled in clinical trials and those encountered in practice. This review summarizes the state-of-the-science on hyperpigmentation, with a particular focus on SOC patients, that should help guide treatment approaches.

FIGURE 1. Melanogenesis and therapeutic targets. Upstream triggers, including UV, visible light acting through OPN3, inflammation, hormones, and the dermal milieu, converge on the melanocyte to activate MITF through the α -melanocyte-stimulating hormone (α -MSH)/melanocortin-1 receptor (MC1R)/cyclic AMP, Wnt/ β -catenin, SCF/c-KIT, MAPK, and PI3K/Akt cascades. MITF in turn drives expression of tyrosinase, TYRP-1, and DCT. The lower panel maps the major therapeutic classes to their points of intervention: 1) photoprotection; 2) upstream and inflammatory modulators (TXA, azelaic acid, niacinamide); 3) tyrosinase inhibitors (thiamidol, hydroquinone, cysteamine, kojic acid, arbutin); 4) keratinocyte-turnover agents (retinoids, α -hydroxy acids, triple combination); and 5) procedural and dermal interventions.^{4,5,32,35,42-45}



α -MSH, α -melanocyte-stimulating hormone; ATK, protein kinase B; CaMKII, Ca²⁺/calmodulin-dependent protein kinase II; cAMP, cyclic adenosine monophosphate; SCF, Skp, Cullin, F-box containing complex; c-KIT, CD117 KIT proto-oncogene receptor tyrosine kinase; CREB, cAMP-response-element-binding protein; DCT, dopachrome tautomerase; ET-B, endothelin receptor type B; HEVL, high-energy visible light; IL-6, interleukin-6; IPL, intense pulsed light; LTC₄, leukotriene C₄; MAPK, mitogen-activated protein kinase; MC1R, melanocortin-1 receptor; MITF, microphthalmia-associated transcription factor; OPN3, Opsin-3; PGE₂, prostaglandin E₂; PKA — protein kinase A; PI3K, phosphoinositide 3-kinase; SPF, sun protection factor; TNF- α , tumor necrosis factor alpha; TXA, tranexamic acid; TYRP-1, tyrosinase-related protein 1; VEGF, vascular endothelial growth factor.

MATERIALS AND METHODS

The authors conducted a comprehensive review of the literature, searching PubMed and MEDLINE for peer-reviewed, English-language articles published between 2000 and 2026, with an emphasis on those appearing from 2015 onward. Search terms combined Medical Subject Headings (MeSH) and free-text descriptors for the hyperpigmentation subtypes, melanogenesis, skin of color, validated outcome instruments, and therapeutics. Randomized controlled trials (RCTs), systematic reviews and meta-analyses, consensus statements, and large epidemiologic studies were prioritized.

Epidemiology and Pathophysiology

Three mechanistic developments over the past decade have reshaped how health care providers should approach hyperpigmentation (Figure 1). The first concerns visible light. It is now clear that visible light, and in particular the high-energy short-wavelength (blue) band, drives sustained pigmentation in Fitzpatrick skin types (FSTs) III to VI by acting on opsin-3 (OPN3)

on melanocytes, triggering a calcium-dependent CAMKII/CREB/MAPK cascade that phosphorylates microphthalmia-associated transcription factor (MITF) and produces a stable tyrosinase/TYRP-1 complex.⁴ Because broad-spectrum sunscreens protect only against ultraviolet (UVA/UVB) radiation alone, use of high sun protection factor (SPF) sunscreen alone is insufficient in darker skin types (FST III-VI); thus, counseling should explicitly address visible-light exposure particularly for SOC patients.

The second development is the recognition that the pathology of hyperpigmentation, and of melasma in particular, extends well beyond melanocyte hyperactivity. Basement-membrane disruption with pendulous melanocytes and melanin incontinence, solar elastosis, mast-cell accumulation, senescent fibroblasts, and elevated vascular endothelial growth factor (VEGF) expression are now recognized as contributing features.^{5,6} These findings help to explain why dermal and mixed-pattern disease tends to be chronic and why rebound is common after procedures, and they provide the rationale for combining several treatment modalities to achieve success.

The third development is a clearer understanding of how inflammatory and hormonal triggers operate. Keratinocyte-derived prostaglandins, leukotrienes, and cytokines, including interleukin-1, interleukin-6, tumor necrosis factor- α , stem cell factor (SCF), and interleukin-33, drive PIH, whereas estrogen and progesterone receptors on melanocytes mediate the hormonal susceptibility characteristic of melasma.⁶ In addition, epigenetic regulation of tyrosinase, TYRP1, dopachrome tautomerase (DCT), and MITF through DNA methylation, histone acetylation, and non-coding RNAs offers a plausible explanation for treatment resistance.⁷ Taken together, these advances support a layered approach that addresses upstream signaling, the melanogenic enzymatic apparatus, and the dermal contributors that topical therapy alone may not reach.

Clinical Types: Focus on Diagnostic and Recognition Gaps

Hyperpigmentation disorders can be organized according to etiology, distribution, depth (epidermal, dermal, or mixed), Fitzpatrick predilection, and characteristic associations (Table 1). Of these features, depth is the most consequential, because dermal and mixed pigmentation are predictably refractory to topical therapy and account for most treatment failures when they are overlooked.⁵ The centrofacial pattern accounts for approximately 95% of melasma cases;⁸ whereas the malar and mandibular variants are easily mistaken for poikiloderma of Civatte or ADMH and are consequently undertreated. Two categories deserve particular emphasis because they remain under-recognized, and each is readily mistaken for melasma.

Maturational dyschromia

This entity presents as ill-defined hyperpigmented patches on the lateral cheeks, temples, and forehead of middle-aged to older patients with skin of color, and it reflects the effect of cumulative ultraviolet (UV) and visible-light exposure on age-related melanocyte changes.^{9,10} It is commonly misdiagnosed as melasma, which can lead to inappropriate hormonal counseling and the overuse of hydroquinone-containing regimens. Appropriate recognition and diagnosis could lead to appropriate management centered on sufficient UV radiation and visible light photoprotection.

Infectious causes of hyperpigmentation

These causes are often under-taught despite their global relevance. Visceral leishmaniasis (kala-azar) classically presents with diffuse skin darkening and may be the presenting sign in immunocompromised patients, including those with human immunodeficiency virus (HIV) coinfection.^{11,12} Chikungunya infection produces characteristic centrofacial and nasal hyperpigmentation (the "chik sign"), reported in 37.5% of patients in a tertiary-care series.^{13,14} Dengue fever can produce post-rash macular pigmentation.¹⁵ Tinea versicolor, caused by *Malassezia* species, produces hyperpigmented or hypopigmented scaly trunk patches that are identifiable by yellow Wood-lamp fluorescence and potassium hydroxide examination.¹⁶ In HIV and AIDS, generalized cutaneous hyperpigmentation may reflect antiretroviral therapy (notably zidovudine), opportunistic infection, adrenal insufficiency, or HIV itself.^{11,12} In these settings,

TABLE 1.

Practical Classification of Cutaneous Hyperpigmentation Disorders				
Subtype	Distribution	Depth	Fitzpatrick	Clinical Pearls / Associations
Melasma	Centrofacial (~95%); malar; mandibular face	Epidermal / dermal / mixed	III–V	Female predominance; pregnancy, oral contraceptives; UV + visible light; comorbid rosacea, atopic dermatitis
PIH	Sites of prior inflammation or injury	Epidermal > dermal	IV–VI	Acne, atopic dermatitis, procedures; persistent in deeper phototypes
Solar lentigines	Dorsal hands, face, forearms, upper chest	Epidermal	Any (rises with age)	Cumulative UV; differentiate from early lentigo maligna
Drug-induced	Diffuse, photoexposed, or mucosal	Variable	Any	Minocycline, antimalarials, amiodarone, chemotherapy; polypharmacy a risk factor
Endocrine / systemic	Diffuse or flexural	Variable	Any	Addison's disease, insulin resistance, hemochromatosis
ADMH (lichen planus pigmentosus, erythema dyschromicum perstans, Riehl's)	Face, neck, trunk	Dermal	III–VI	Lichenoid interface inflammation; concentrated in skin of color
Maturational dyschromia	Lateral cheeks, temples, forehead	Epidermal	IV–VI	Cumulative UV + visible light; age-related; often misdiagnosed as melasma
Infectious (kala-azar, chikungunya, dengue, tinea versicolor, HIV)	Diffuse (kala-azar, HIV); centrofacial / nose (chikungunya); trunk (tinea versicolor)	Epidermal $\pm$ dermal	Any (severe in skin of color)	Visceral leishmaniasis; arboviral; <i>Malassezia</i> ; ART, opportunistic infection

ADMH, acquired dermal macular hyperpigmentation; ART, antiretroviral therapy; HIV, human immunodeficiency virus; PIH, post-inflammatory hyperpigmentation; UV, ultraviolet light.

identifying the underlying cause early, may guide overall treatment approach.

Acquired dermal macular hyperpigmentation

Acquired dermal macular hyperpigmentation (ADMH), formalized by global consensus in 2018 and reaffirmed in 2025, encompasses lichen planus pigmentosus, erythema dyschromicum perstans, idiopathic eruptive macular pigmentation, and Riehl melanosis.^{17,18} These entities are concentrated in skin of color and share overlapping clinical and histopathologic features.

Impact on Quality of Life

While hyperpigmentation disorders are common, the extent and location of involvement can significantly affect quality of life (QoL), including a significant psychological burden for patients.⁹ A 2022 meta-analysis of 14 studies (n = 1,398) demonstrated consistent QoL impairment across diverse melasma populations, with emotional distress and impaired social functioning predominating.¹⁹ In a Greek case-control study, Melasma Area and Severity Index (MASI) severity correlated with both depression and Melasma Quality of Life (MELASQoL) scores, and the prevalence of anxiety was significantly elevated.²⁰ Furthermore, in a South African cohort of Fitzpatrick IV to VI patients, menopausal status, cheek involvement, severity, and educational level each independently predicted MELASQoL impairment, indicating that deeper pigmentation does not lessen the burden.²¹

Two findings should shape how dermatology HCPs structure their conversations with patients. First, clinical improvement in pigmentation does not reliably track patient-reported burden because MASI and MELASQoL correlate inconsistently, which indicates that objective severity and lived experience capture related but distinct dimensions.¹⁹ A patient who reports persistent psychosocial impact despite a documented improvement in MASI is not reporting in error, and dismissing this response may lead to the lack of continued treatment adherence. Second, PIH compounds the burden of the underlying dermatosis. Acne in particular is associated with elevated anxiety, depression, and suicidal ideation,²² and in skin of color the residual pigmentation often outlasts the primary lesions.²³ Assessing patient-reported burden at baseline and at follow-up therefore identifies the patients whose pigmentation has responded adequately but whose psychosocial burden warrants additional support.

Diagnostic Gaps in Skin of Color

The diagnostic literature for pigmentary disorders shows a consistent pattern, in that the tools dermatology HCPs rely on were validated predominantly in lighter phototypes and lose accuracy in SOC patients. Wood-lamp examination, for example, shows progressively attenuated contrast as constitutive epidermal melanin increases, which limits subtype discrimination in Fitzpatrick V to VI patients, precisely where the distinction would most influence therapy.^{5,8} Dermoscopic atlases

for inflammatory and pigmentary dermatoses likewise remain skewed toward lighter phototypes, with few published studies explicitly reporting Fitzpatrick IV to VI representation;²⁴ and for the ADMH entities, the clinical and histopathologic overlap was substantial enough to require international consensus statements to harmonize nomenclature.^{17,18} Additionally, machine-learning algorithms tend to amplify these gaps.

A 2022 review documented widespread underreporting and underrepresentation of diverse skin types in machine-learning research.²⁵ In the absence of phototype-stratified validation, claims of equivalent algorithmic performance across skin types are unsupported, and uncorrected bias risks encoding historical underrepresentation into artificial intelligent (AI)-driven algorithms.

Therapeutic Approaches

Across the hyperpigmentation evidence base, the multimodal approach remains the most consistent and effective approach. Expert consensus statements developed over the past decade,^{17,18,26-28} 2 systematic reviews,^{29,30} and a substantial body of randomized controlled trial (RCT) and meta-analytic data converge on the same core principles, namely foundational photoprotection that includes visible light, multimodal topical therapy as first line, escalation to oral and procedural options for refractory disease, and structured maintenance. The 2025 global modified-Delphi consensus offers a contemporary synthesis;¹⁸ but the same principles are supported by parallel regional and international expert opinion and by the primary literature.

Photoprotection

Photoprotection is foundational across all melanin hyperpigmentation disorders. The visible-light pathway mediated by opsin-3 (OPN3) provides the mechanistic rationale,⁴ and tinted sunscreens containing iron oxides or pigmentary titanium dioxide are the only formulations supported by randomized data showing prevention of hyperpigmentation induced by visible light.³¹ Because visible light drives pigmentation in part through the generation of reactive oxygen and nitrogen species, sunscreens that combine visible-light-attenuating pigments with topical antioxidants offer a complementary mechanism, and early clinical studies indicate that antioxidant-containing formulations reduce visible-light-induced erythema and pigmentation and may limit worsening of existing pigmentary disorders.³² Patients with skin of color therefore require formulations that both absorb visible light and counter the effects of reactive species, because a non-tinted broad-spectrum sunscreen alone, however high its SPF, is mechanistically insufficient.

Topical therapy

Topical therapy is itself multimodal because no single agent addresses the multiple pathways that contribute to hyperpigmentation. Two systematic reviews concluded that

TABLE 2.**Principal Topical and Oral Therapies for Hyperpigmentation**

Agent	Mechanism	Indication	Evidence
Hydroquinone	Tyrosinase inhibition	Melasma, PIH, lentigines	Grade A; ochronosis risk; not for maintenance
Triple combination (hydroquinone + tretinoin + fluocinolone)	Multi-pathway	Moderate-to-severe melasma, first line	Grade A; rebound risk limits maintenance
Non-hydroquinone triple combination	Multi-pathway	Mild melasma, first line	Consensus ¹⁸
Azelaic acid	Tyrosinase inhibition, anti-inflammatory	Melasma, PIH; pregnancy	Grade A; pregnancy-safe
Kojic acid	Tyrosinase inhibition	Melasma, PIH; adjunct	Grade A adjunct
Niacinamide	Inhibits melanosome transfer	Melasma, PIH; maintenance	Adjunctive evidence; maintenance role
Retinoids (tretinoin, adapalene)	Keratinocyte turnover	Epidermal melasma, PIH, photodamage	First-line + maintenance for photodamage
Cysteamine	Antioxidant, tyrosinase inhibition	Melasma second-line, maintenance	Consensus ¹
Thiamidol	Selective human tyrosinase inhibition	Melasma, acne-PIH, periorbital, flexural; maintenance	Non-inferior to hydroquinone 4% (Lima); ³³ 24-wk RCT (Roggenkamp) ³⁴
Topical TXA	Plasmin inhibition	Melasma first-line; PIH	Consensus first-line mild melasma
Oral TXA	Plasmin inhibition	Moderate-to-severe / refractory melasma	Grade A meta-analysis ³⁵
Intradermal TXA / microneedling-delivered TXA	Plasmin inhibition	Moderate-to-severe melasma	Superior MASI vs. oral/topical; meta-analysis ³⁶
Arbutin	Tyrosinase inhibition	Mild melasma first-line; periorbital	Consensus ¹⁸
Polypodium leucotomos (oral)	Antioxidant, photoprotective adjunct	Melasma adjunct; HEVL protection	Adjunctive evidence ⁴³

HEVL, high energy visible light; MASI, Melasma Area and Severity Index; PIH, post-inflammatory hyperpigmentation; RCT, randomized controlled trial; TXA, tranexamic acid.

combinations of tyrosinase inhibitors (thiamidol, hydroquinone, kojic acid, arbutin), retinoids, azelaic acid, antioxidants, niacinamide, and topical tranexamic acid (TXA) outperform monotherapy.^{29,30} The principal topical and oral agents, with their indications and supporting evidence, are summarized in Table 2. Head-to-head trial data support particular agents, in that a 90-day evaluator-blinded RCT demonstrated non-inferior MASI reduction with thiamidol compared with hydroquinone 4% (43% vs 33%),³³ and a 24-week vehicle-controlled RCT confirmed the efficacy of thiamidol in moderate-to-severe Fitzpatrick III to V melasma.³⁴ Use of any topical agent(s) as first line therapy should be monitored for adverse events, and adjusted accordingly or by patient preference with appropriate consultation.

Tranexamic acid

Tranexamic acid (TXA) is a valuable adjunct that can be delivered via several routes. A 2021 meta-analysis of 24 RCTs found that oral TXA 250 mg twice daily produced significant reductions in MASI and modified MASI (mMASI) without serious adverse events,³⁵ and a 2024 meta-analysis of 17 RCTs found that intradermal injection and microneedling-delivered TXA outperformed the oral and topical routes on

MASI endpoints, although oral administration yielded greater patient satisfaction.³⁶ A 2024 Delphi consensus from the Indian Pigmentary Disorders Society describes prescribing practices for oral TXA, including pretreatment screening for thromboembolic risk and quarterly coagulation monitoring.²⁶ Together, these data position TXA as a valuable treatment option with appropriate monitoring.

Procedural therapy

Procedural therapy occupies a defined role in refractory disease and in mixed or dermal disease, with phototype-specific considerations that guide selection (Table 3). An open-label RCT of intense pulsed light added to a stable fixed-dose triple combination produced approximately 49% MASI reduction at month 6 and 45% at month 12 compared with triple combination alone.³⁷ For solar lentigines, a 2025 systematic review identified pigment-specific lasers (Q-switched and picosecond) and intense pulsed light as first-line, and noted that picosecond systems produce less rebound PIH than Q-switched platforms in darker phototypes.³⁸ For acne-associated PIH, priming the skin with a lightening agent and avoiding perioperative retinoids reduces iatrogenic pigmentation in Fitzpatrick V to VI patients.²³

TABLE 3.

Procedural Interventions by Indication and Fitzpatrick Considerations			
Procedure	Strongest Indication	Evidence	Cautions in Fitzpatrick IV–VI
Superficial peels (glycolic, salicylic, mandelic)	Melasma, PIH	Moderate; adjunct to topicals	Start low concentration; PIH risk with overuse
Jessner / low-concentration TCA peel	Melasma, PIH	Moderate	Experienced operators only
Microneedling ± topical TXA	Dermal/mixed melasma	Strong for dermal variant ³⁶	Generally safe across phototypes
Q-switched Nd:YAG (low fluence)	Refractory melasma; non-acne PIH	Variable; recurrence common	Multiple passes increase PIH and rebound risk
Picosecond laser	Solar lentigines; refractory melasma	Strong for lentigines ³⁸	Lower PIH than Q-switched in lentigines
Intense pulsed light	Solar lentigines; freckles; refractory melasma (with triple combo)	Strong (74.6–90%); ³⁸ ~49% MASI reduction at 6 mo combined with triple combo ³⁶	Caution in deeper phototypes
Cryotherapy	Solar lentigines	Moderate	Avoid in Fitzpatrick IV–VI (high PIH risk)

MASI, Melasma Area and Severity Index; PIH, post-inflammatory hyperpigmentation; TCA, trichloroacetic acid; TXA, tranexamic acid; YAG, yttrium aluminum garnet.

Maintenance and special situations

Three principles unify the therapeutic evidence and should be included in any multimodal treatment algorithm. First, maintenance differs from active treatment, and long-term use of a hydroquinone-based approach should be limited and/or heavily monitored due to cumulative tolerability and rebound concerns. Given this, use of non-hydroquinone agents, including antioxidants, kojic acid, thiamidol, retinoids, TXA, arbutin, azelaic acid, and niacinamide, should be considered for long term maintenance.^{17,18,26,28,30} Second, intravenous glutathione is not recommended at any line, because the efficacy data are insufficient and safety concerns remain.¹⁸ Third, the pregnancy-safe options are photoprotection, azelaic acid, and glycolic acid.^{18,27} For hyperpigmentation associated with infection, treatment of the underlying infection, whether anti-leishmanial, anti-fungal, or anti-retroviral, is primary, with photoprotection and topical treatment reserved for residual pigmentation, and post-chikungunya nasal pigmentation typically resolves over months without specific therapy.^{11,13,14,16}

Conclusion and Critical Gaps

Hyperpigmentation disorders fall most heavily on the patients who are least well represented in dermatologic evidence, education, and algorithm development. The literature has nonetheless advanced therapy considerably, providing head-to-head and meta-analytic support for visible-light photoprotection, multimodal topical regimens, route-stratified TXA, severity-graded escalation, and structured maintenance. The remaining gaps are concentrated in SOC, where dermoscopic atlases, machine-learning training data, validated outcome measures, and trial populations all underrepresent patients with Fitzpatrick skin types III to VI.^{3,5,21,25,38-41} Closing these gaps will require inclusive trial design, phototype-stratified validation, harmonized

outcome measures, and attention to patient-reported burden alongside objective severity. In the meantime, the responsibility falls to the dermatology community to apply the existing evidence with full awareness of the populations from whom it was derived, and to seek, document, and act on patient-reported burden in every consultation.

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ST has served as an investigator for Allergan Aesthetics, Concert Pharmaceuticals, Croma-Pharm GmbH, Lilly, and Pfizer; as a consultant for Arcutis, Armis, Avita Medical, Beiersdorf, Biorez Inc, BMS, Cara Therapeutics, Dior, EPI Health, Estée Lauder, Evolus Inc, Galderma, GloGetter, Hugel America, Incyte, Johnson & Johnson, L'Oréal, Lilly, Medscape, Pfizer, Piction Health, Sanofi, Scientis US, UCB, Vichy Laboratories; as a speaker for Beiersdorf, Catalyst Medical Education, CME Outfitters, DermSquared, HMP Global, LearnSkin, Medscape, and MJH Lifesciences; and as an editorial board member for Archives in Dermatologic Research, Cutis, and Practical Dermatology. She has also received royalties from McGraw-Hill.

SRD has served as a consultant, investigator, and/or advisor for AbbVie, Arcutis, Beiersdorf, Eli Lilly, Galderma, Incyte, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, UCB, and other companies involved in dermatologic therapeutics.

CF has served as an investigator, consultant, advisory board member, and/or speaker for AbbVie, Acclaro, Aerolase, Apogee, Beiersdorf, Benev, Bubble, Crown Aesthetics, Estée Lauder, Galderma, Incyte, Johnson & Johnson, Kenvue, L'Oréal, Novartis, Pierre Fabre, Procter & Gamble, Reframe Beauty, Revance, Sun Pharma, Veradermics, Viking Therapeutics, Xtresse, and Zenzema.

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AA has served as a consultant, advisory board member, investigator, and/or speaker for AbbVie, Aerolase, Allergan, Almirall, Alphyn, Alumis, Amgen, Apogee BioPharm, Arcutis, BauschHealth, Beiersdorf, Inc., Boehringer Ingelheim, Botanix Pharmaceuticals, Bristol Myers Squibb, Canfield Scientific, Castle Biosciences, Caudalie, Dermavant, DermSquared, Eli Lilly, Galderma, Genentech, HairDays, Incyte, Janssen Scientific Affairs, Johnson & Johnson, LEO Pharma, L'Oréal, Novartis, OrukaTherapeutics, Pfizer, RBC Consultants, Regeneron, Sanofi, Sanofi Genzyme, Sanofi/Regeneron, Scientis, Symrise, Takeda, UCB, and Veradermics Inc. He has also received royalties from Elsevier, Springer, UpToDate, Inc. (Wolters Kluwer Health), and Wiley-Blackwell; as well as equipment from Aerolase.

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