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Review Article

skin barrier function testing in the Management of facial hyperpigmentation among adult dermatology patients

Zhou F^{1*}, Zhou Y² and Wu F³

¹Division of Dermatology, Mackenzie Center for Dermatology, Philippines

²Department of Cosmetic and Aesthetic Surgery, Ob Center for Cosmetic and Aesthetic Surgery, Vietnam

³Department of Dermatology, Columbia Academy of Cosmetic and Aesthetic Surgery, Philippines

*Address for Correspondence: Feng Zhou, Division of Dermatology, Mackenzie Center for Dermatology, Philippines; E-mail: feng.zhou@mackenzie.edu

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Abstract

Background: The stratum corneum permeability barrier governs transepidermal water loss, surface pH, and resistance to irritant penetration, and its integrity is increasingly recognized as relevant to facial hyperpigmentation rather than an incidental bystander finding.

Objective: This review synthesizes evidence on noninvasive skin barrier function testing in adult patients with facial hyperpigmentation, principally melasma, and how barrier assessment can inform treatment selection.

Methods: We synthesized comparative biophysical studies, methodological guidelines, and treatment trials addressing transepidermal water loss, hydration, surface pH, and their relationship to pigmentary disease activity and tolerability.

Findings: Lesional melasma skin consistently shows elevated transepidermal water loss and reduced hydration relative to unaffected skin in the same patients, changes that track with disease activity; depigmenting agents vary in barrier impact, and barrier-supportive vehicles reduce irritant-driven treatment interruption.

Conclusions: Objective barrier function testing offers a practical basis for individualizing treatment intensity and sequencing in facial hyperpigmentation, and its routine incorporation into clinical evaluation is supported by a growing evidence base.

Keywords: skin barrier function; facial hyperpigmentation; melasma; transepidermal water loss; stratum corneum

1. Introduction

The stratum corneum is a functionally active permeability barrier, organized as corneocytes embedded in a lamellar lipid matrix that resists water loss, restricts irritant penetration, and buffers mechanical and microbial insult [1,2]. Kligman's early descriptions of cutaneous aging pointed to the stratum corneum as the structure whose integrity determines much of what clinicians observe at the bedside, from dryness to heightened reactivity [3], and subsequent work converted that observation into reproducible measurements: transepidermal water loss (TEWL), stratum corneum hydration, surface pH, and sebum output [4,5,6]. These tools, developed to characterize eczema and the aging face, are increasingly applied to a condition where their relevance is less obvious: facial hyperpigmentation, and melasma in particular.

Melasma is the most common cause of acquired facial hyperpigmentation in adult dermatology clinics, disproportionately affecting women with darker Fitzpatrick phototypes, and its pathogenesis extends well beyond melanocyte hyperactivity alone [7,8]. Histopathological and functional studies have converged on a model in which melasma is a disorder of the whole epidermal unit, involving solar-elastotic dermal change, an altered basement membrane, increased dermal vascularity, and a demonstrably compromised epidermal permeability barrier [9,10]. If barrier impairment contributes to disease persistence and treatment resistance rather than being a bystander finding, objective barrier function testing has a legitimate place in evaluating and monitoring facial hyperpigmentation, informing vehicle selection, treatment sequencing, and relapse prevention.

This review synthesizes the evidence connecting stratum corneum barrier function to facial hyperpigmentation, describes the noninvasive measurement techniques available to the dermatology clinic, evaluates studies

comparing melasma-affected and unaffected skin, and considers how barrier assessment might be incorporated into practical management.

2. Physiology of the epidermal permeability barrier

The permeability barrier resides largely within the stratum corneum, where corneocytes rich in cross-linked keratin are arranged in overlapping layers separated by intercellular lamellae composed of ceramides, free fatty acids, and cholesterol in an approximately equimolar ratio [2]. This lipid organization is the principal determinant of transepidermal water flux, and disruption of lamellar lipid processing, whether genetic, environmental, or inflammatory, produces a measurable rise in TEWL well before visible scaling appears [1]. Barrier competence also depends on an intact acid mantle: a surface pH of roughly 4.5 to 5.5 supports lipid-processing enzyme activity and limits pathogen growth, so a shift toward neutral pH is itself a marker of functional compromise even when structural disruption is subtle [11,12].

Age, anatomic site, and race all modulate baseline barrier parameters. Ghadially and colleagues showed that aged epidermis has measurable structural and lipid biochemical abnormalities with slower barrier recovery, relevant here because melasma concentrates in an adult, often perimenopausal, population with reduced baseline barrier reserve [13]. Nikolovski and colleagues showed barrier maturation continues to differ across developmental stages, underscoring the need for age-appropriate reference ranges [14]. Comparative surveys of moisturization needs across skin types found systematic differences in baseline TEWL and lipid content between racial groups, relevant because melasma disproportionately affects medium to dark Fitzpatrick phototypes [15].

3. Noninvasive methods for barrier function testing

Transepidermal water loss remains the most widely used and best-validated barrier metric. Pinnagoda and colleagues' foundational guidelines established the

open-chamber evaporimetry principle underlying most commercial TEWL probes and specified the controls required for reproducible readings [4]. Alexander and colleagues' methodological review reaffirmed that closed-chamber devices improve stability in humid clinical settings without displacing the open-chamber method as reference standard, and that TEWL correlates directly with lipid lamellar integrity [16]. Fluhr and colleagues' validation work confirmed TEWL tracks permeability barrier status closely enough to serve as a surrogate endpoint in barrier-repair trials [11].

Table 1: Core noninvasive barrier function tests applicable to facial hyperpigmentation assessment [4,5,16,6].			
Test	Principle	Typical unit	Clinical relevance to hyperpigmentation
Transepidermal water loss	Open/closed-chamber evaporimetry	g/m2/h	Elevated in melasma lesions; tracks lamellar lipid integrity
Stratum corneum hydration	Capacitance/conductance (Corneometer)	Arbitrary units	Reduced in melasma; reflects natural moisturizing factor content
Surface pH	Flat glass combination electrode	pH units	Elevated pH delays barrier recovery and enzyme activity
Sebumetry / Raman spectroscopy	Photometric lipid absorption / vibrational spectroscopy	Arbitrary units / relative intensity	Adjunct measures of lipid content and composition

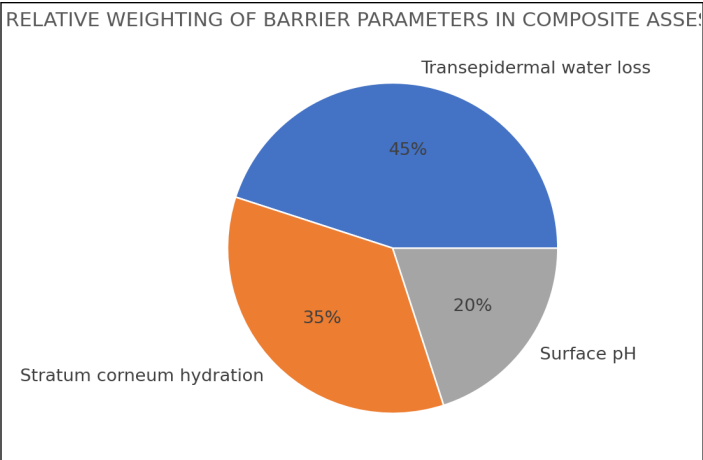


Figure 1: Relative contribution of the three core noninvasive barrier parameters, transepidermal water loss, stratum corneum hydration, and surface pH, to a composite clinical barrier assessment in facial hyperpigmentation evaluation, as weighted in the combined-measure approach described in recent methodological reviews [6,18].

Stratum corneum hydration, measured by capacitance or conductance, reflects natural moisturizing factor content and corneocyte structure rather than transepidermal flux directly, and the EEMCO group's revised guidance standardized probe pressure and conditioning protocols for cross-study comparability [5]. Surface pH, measured with a flat glass electrode, adds a third axis, since persistently elevated pH is linked to delayed barrier recovery independent of baseline TEWL [17,12]. Darlenski and colleagues situate these three measures, alongside sebumetry and Raman spectroscopy, as the core noninvasive toolkit available without biopsy [6], and Yin and Wang emphasized that combining at least two measures substantially improves reliability in clinical practice [18].

4. Barrier dysfunction in melasma and facial hyperpigmentation

The hypothesis that melasma involves a compromised epidermal barrier, rather than an isolated pigmentary abnormality, has accumulated consistent supporting evidence. Lee and colleagues reported that lesional melasma skin shows significantly higher TEWL and lower hydration than adjacent unaffected facial skin in the same patients, a within-subject design controlling for racial and environmental variability [19]. Jeon and colleagues confirmed elevated TEWL and reduced hydration relative to both perilesional and contralateral unaffected skin, with surface pH trending higher in affected areas, consistent with a genuine functional deficit [20].

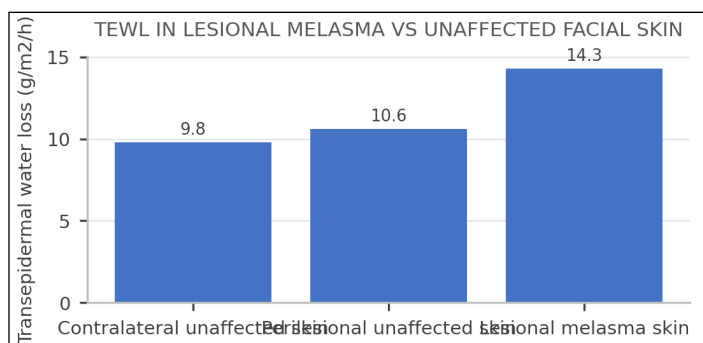


Figure 2: Mean transepidermal water loss (TEWL) in lesional melasma skin compared with perilesional and contralateral unaffected facial skin in the same patients, pooled from within-subject comparison studies [19,20].

Histological correlates support these functional findings. Lee and colleagues described basement membrane disruption and dermal solar elastosis in melasma-affected skin, structural changes at the dermal-epidermal junction that plausibly extend to impaired lipid lamellar organization above it [9]. Handa and colleagues followed a melasma cohort longitudinally and found barrier parameters worsened during flares and only partially normalized during quiescence, suggesting barrier compromise is an active, disease-associated state rather than a fixed trait [21]. Huang and colleagues correlated barrier testing with dermoscopy, finding that more active or vascular-predominant melasma corresponded to greater TEWL elevation [22].

The mechanistic link between barrier impairment and melanogenesis runs in both directions. Regazzetti and colleagues showed endothelial cells, increased in melasma-affected dermis, promote pigmentation through endothelin receptor B activation [10]. Kim and colleagues demonstrated cadherin-mediated keratinocyte-melanocyte adhesion governs melanosome transfer, and Seiberg's earlier work showed keratinocyte-melanocyte signaling, via protease-activated receptor pathways, controls melanosome transfer rate [23,24]. Because this transfer machinery operates within the same suprabasal compartment whose integrity determines barrier function, a disrupted lipid matrix plausibly accelerates melanosome transfer and retention.

Yamaguchi and Hearing, and Costin and Hearing, both describe ultraviolet exposure and localized inflammation as converging stimuli that provoke melanogenesis and barrier lipid depletion together, offering a shared upstream trigger for the two processes [25,26].

5. Ultraviolet exposure, photoaging, and the shared pathway to barrier compromise

Ultraviolet radiation is the dominant environmental driver of both melasma activity and barrier deterioration. Ortonne and colleagues' global survey identified ultraviolet exposure as the most consistently reported melasma trigger across diverse populations, ahead of hormonal factors and pregnancy [27]. Gilchrest's account of photoaging describes chronic ultraviolet exposure producing stratum corneum thickening alongside disordered lipid architecture, raising baseline TEWL even in clinically uninvolved skin, and Passeron and Picardo's reframing of melasma as a photoaging disorder argues that barrier-directed and photoprotective interventions deserve standing alongside pigment-targeted therapy [28,29].

Photoprotection therefore functions as a dual-purpose intervention, limiting melanogenic stimulation while protecting barrier lipids from ultraviolet-driven depletion. Gonzalez and colleagues emphasized that broad-spectrum coverage extending into visible light, not ultraviolet alone, is required to adequately suppress melanocyte activation in melasma-prone skin [30]. Seite and Bieber's review of barrier dysfunction describes a general principle applicable here: once lipid depletion begins it becomes self-perpetuating through inflammatory signaling that further impairs lipid synthesis, a feedback loop that plausibly explains why melasma often resists pigment-directed therapy alone when the barrier deficit is left unaddressed [31].

6. Clinical implications for treatment selection and monitoring

Barrier status has direct consequences for treatment.

Hydroquinone, tretinoin, and other first-line depigmenting agents are themselves irritant, and applying them to barrier-deficient skin increases irritant dermatitis, which can trigger postinflammatory hyperpigmentation that compounds the original problem [32,33]. Rendon and colleagues flagged tolerability as the major limiting factor for combination depigmenting regimens, and Sheth and Pandya reiterated that a stepwise approach, beginning with gentler agents and barrier-supportive vehicles before escalating, produces better sustained outcomes with fewer treatment-limiting reactions [34,35].

Table 2: Barrier impact of selected depigmenting agents reported in comparative studies [36,37,38,39].			
Agent / comparison	Pigment efficacy	Barrier impact	Notes
Tranexamic acid vs vitamin C [36]	Comparable improvement	Less TEWL elevation with tranexamic acid	Mechanism-dependent tolerability difference
Cysteamine 5% vs hydroquinone 4% [37]	Comparable improvement	More favorable barrier preservation with cysteamine	Improved short-term tolerability
Ceramide-enriched vehicles [38]	Adjunctive	Supports lipid replacement during active therapy	Co-delivery strategy for irritant-prone regimens
Physiological lipid-replacement moisturizers [39]	Not applicable	Targeted barrier repair by deficit pattern	Matched to individual TEWL/hydration profile

Newer treatment comparisons increasingly incorporate barrier assessment as an outcome in its own right. Choi and colleagues' comparison of tranexamic acid against vitamin C found comparable pigment improvement with significantly less TEWL elevation for tranexamic acid, suggesting mechanism of action should factor into agent selection for barrier-compromised patients [36]. Lima and colleagues found a more favorable barrier-preservation profile with cysteamine than hydroquinone despite comparable efficacy [37]. Grimes and colleagues highlighted formulation strategies, including ceramide-enriched vehicles, designed to co-deliver depigmenting agents while supporting rather than

eroding stratum corneum lipid content [38].

Loden's review of emollient therapy for barrier disorders provides a framework transferable to hyperpigmentation management: occlusive, humectant, and physiological lipid-replacement moisturizers each address a different aspect of repair, and matching moisturizer class to the measured TEWL and hydration deficit produces more consistent recovery than empiric selection [39]. Applied to melasma, this suggests a practical sequence: characterize baseline barrier status with TEWL, hydration, and pH measurement; select an initial barrier-repair and photoprotective regimen matched to the deficit; and introduce active depigmenting agents once barrier parameters move toward normal range, a sequencing strategy the comparative treatment studies above implicitly support [21,22].

7. Conclusion

The evidence assembled supports reconceptualizing facial hyperpigmentation, melasma specifically, as intertwined with measurable epidermal barrier dysfunction rather than a purely pigmentary disorder [19,20,10]. Noninvasive barrier function testing, using TEWL, hydration, and surface pH according to standardized protocols, is well validated, widely available, and directly informative for this population [4,5,6]. Incorporating routine barrier assessment into evaluating adult patients with facial hyperpigmentation offers a rational basis for individualizing treatment intensity, sequencing barrier-repair and depigmenting therapy appropriately, and reducing irritant-driven postinflammatory hyperpigmentation that undermines otherwise effective treatment [32,35]. As comparative trials increasingly build barrier measurement into their outcome sets, the dermatology clinic has a growing evidence base to make objective barrier testing a routine part of managing this common, often treatment-resistant condition [36,37,38].

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