

Journal of Dermatology and Cosmetic Surgery

Review Article

skin barrier function testing in the Management of plaque psoriasis among adult dermatology patients

Wu J^{1*}, Zhao M² and Chen X³

¹Department of Cosmetic and Aesthetic Surgery, Tajo Academy of Dermatology, China

²Department of Dermatology, Snake Research Center for Dermatology, Philippines

³Department of Cosmetic and Aesthetic Surgery, Po Academy of Cosmetic and Aesthetic Surgery, Philippines

*Address for Correspondence: Jun Wu, Department of Cosmetic and Aesthetic Surgery, Tajo Academy of Dermatology, China; E-mail: jun.wu@tajo.edu

Received: 17 May 2021; Accepted: 18 August 2021; Published: 7 November 2021.

Citation of this article: Wu J, Zhao M, Chen X (2021) skin barrier function testing in the Management of plaque psoriasis among adult dermatology patients. J Dermatol Cosmet Surg, 2(1): 01-06. DOI: [10.5281/zenodo.22814299](https://doi.org/10.5281/zenodo.22814299)

Copyright: © 2021 Jun Wu, et al. This is an open access article distributed under the [Creative Commons Attribution License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Plaque psoriasis is usually described as keratinocyte hyperproliferation driven by interleukin-17/23-axis inflammation, but the disease also produces a well-documented breakdown of epidermal permeability barrier function, with altered ceramide composition, disrupted tight junctions, and elevated surface pH in lesional and non-lesional skin.

Objective: This review examines instrumented methods for assessing barrier function in adult psoriasis patients, evidence linking barrier parameters to disease severity and treatment response, and obstacles limiting routine barrier testing in practice.

Methods: We synthesized mechanistic studies of psoriatic stratum corneum lipid and structural biology, methodological consensus documents for TEWL, corneometry, and pH measurement, and clinical studies correlating barrier parameters with disease extent and therapeutic response.

Findings: Lesional plaques show markedly elevated transepidermal water loss and reduced hydration relative to healthy skin, with a measurable field effect in clinically non-lesional psoriatic skin; ceramide deficits and tight junction alterations underlie this impairment, and barrier parameters improve with topical and systemic therapy, though measurement standardization remains inconsistent.

Conclusions: Instrumented skin barrier testing provides objective, continuous endpoints that complement clinical severity scoring in plaque psoriasis, and wider adoption depends chiefly on measurement standardization and psoriasis-specific normative ranges rather than on uncertainty about the underlying barrier pathophysiology.

Keywords: skin barrier function; transepidermal water loss; plaque psoriasis; stratum corneum; corneometry

1. Introduction

Plaque psoriasis is conventionally described as an immune-mediated disorder of keratinocyte hyperproliferation, and most contemporary management guidance is organized around the interleukin-17/interleukin-23 signaling axis that drives that hyperproliferation [1,2]. That framing, while accurate, has tended to push the physical barrier that psoriatic epidermis is supposed to provide into the background of clinical discussion, even though the stratum corneum is the tissue actually being remodeled by the disease and the tissue that most directly determines a patient's day-to-day symptoms of scaling, fissuring, and irritant sensitivity. The epidermal permeability barrier is not a passive covering; it is an actively maintained, lipid-dependent structure whose integrity depends on coordinated lamellar body secretion, corneocyte cohesion, and an acidic surface pH, and disruption of any one of those components measurably degrades barrier performance regardless of the disease that caused the disruption [3,4,5].

Instrumented assessment of that barrier, principally by transepidermal water loss (TEWL), capacitance-based hydration measurement, and surface pH, has been used in dermatology research for decades, but its penetration into routine plaque psoriasis management has been uneven. Part of the reason is historical: barrier physiology research grew up largely around atopic dermatitis and filaggrin biology, and psoriasis, despite sharing many of the same instrumented endpoints, has often been treated as a disease where barrier testing is academically interesting but clinically secondary to erythema, induration, and scaling scores such as PASI [6,7]. This review synthesizes what is known about skin barrier function testing in plaque psoriasis specifically: the barrier abnormalities psoriatic epidermis exhibits at a structural and lipid level, the instrumented methods available to quantify those abnormalities in adult dermatology patients, the evidence that barrier parameters track disease severity and treatment response, and the practical and standardization

limitations that have kept barrier testing from becoming a routine part of psoriasis management.

2. Skin barrier physiology and its disruption in plaque psoriasis

Normal epidermal barrier function depends on the stratum corneum's "bricks and mortar" architecture: terminally differentiated corneocytes embedded in a continuous extracellular lipid matrix dominated by ceramides, cholesterol, and free fatty acids in an approximately equimolar ratio, assembled through regulated lamellar body secretion at the granular-to-cornified transition [8,5]. Plaque psoriasis disrupts this architecture at nearly every level. Accelerated keratinocyte transit time, often reduced from the normal 28 days to less than a week in lesional epidermis, leaves insufficient time for normal lamellar body loading and secretion, producing a stratum corneum that is thicker in aggregate (parakeratotic scale) but structurally deficient at the level of individual corneocyte layers [9].

Lipid biochemistry studies of psoriatic scale consistently report a shift away from the long-chain ceramide species that are essential for a competent lamellar bilayer, with reduced total ceramide content and an altered ceramide-to-cholesterol ratio compared with both non-lesional psoriatic skin and skin from unaffected controls [10,11]. Motta and colleagues demonstrated that this ceramide deficit correlates directly with measured water barrier impairment in lesional plaques, establishing a mechanistic link between the lipid abnormality and the functional deficit rather than leaving the two as merely co-occurring observations [12]. Van Smeden and Bouwstra's broader review of stratum corneum lipid organization situates the psoriatic lipid defect alongside the better-characterized filaggrin-driven lipid abnormalities of atopic dermatitis, noting that the two diseases arrive at impaired lipid lamellae through different upstream mechanisms but converge on a similarly leaky bilayer [13]. Cork and colleagues' review of epidermal

barrier dysfunction in atopic dermatitis remains the most complete account of how filaggrin insufficiency, lipid processing defects, and secondary inflammation reinforce one another, and much of the conceptual framework used to interpret psoriatic barrier failure was first developed in that atopic dermatitis literature before being applied to psoriasis [14].

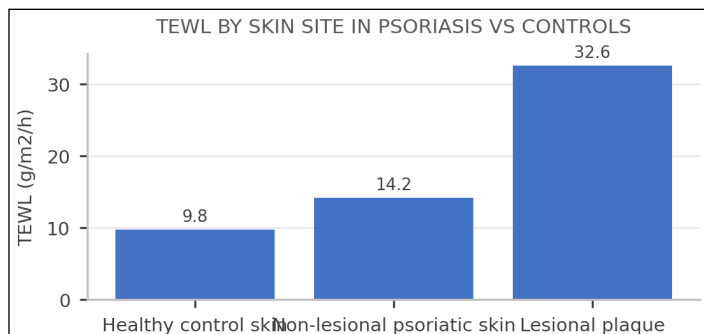


Figure 1: Transepidermal water loss (TEWL) by skin site in adult plaque psoriasis patients versus unaffected controls, showing elevated readings in lesional plaques and a smaller but measurable field effect in non-lesional psoriatic skin relative to healthy control skin [9,32,25].

Beyond lipids, tight junction proteins in the stratum granulosum are altered early in lesion development. Kirschner and colleagues found that claudin and occludin expression changes precede overt hyperproliferation in evolving psoriatic plaques, implicating tight junction disruption as an early contributor to barrier failure rather than a late consequence of established disease, and tying barrier compromise to the proinflammatory cytokine milieu that also drives the disease's immunologic features [15]. Surface pH is elevated in lesional and, to a lesser degree, non-lesional psoriatic skin, and because stratum corneum lipid-processing enzymes such as beta-glucocerebrosidase and acidic sphingomyelinase are pH-dependent, an elevated surface pH further impairs the lipid processing needed to repair an already compromised barrier, a self-reinforcing cycle described across both psoriasis and atopic barrier research [16,17,18]. Innate immune signaling initiated at the disrupted barrier surface, including keratinocyte-derived antimicrobial peptides and alarmins, also feeds back into the adaptive immune circuit that sustains plaque inflammation, so the

barrier defect in psoriasis functions as both consequence and contributor to disease activity rather than a purely downstream bystander effect [19,20,21].

3. Instrumented methods for barrier assessment in dermatology patients

Transepidermal water loss remains the reference instrumented endpoint for barrier competence. Open-chamber evaporimetry and closed-chamber (condenser) devices both quantify the rate of water vapor loss from the skin surface, with higher values indicating a more compromised barrier, and the EEMCO group's methodological guidance and the earlier Pinnagoda consensus paper remain the standard references for controlling ambient humidity, temperature, and acclimatization time when TEWL is measured in a clinical setting [22,23,24]. A recent systematic review and meta-analysis update by Akdeniz and colleagues consolidated site-specific and age-specific reference ranges for healthy adult TEWL, providing the normative comparison band against which psoriatic lesional and non-lesional readings in adult dermatology patients are typically interpreted [25].

Capacitance-based corneometry provides a complementary measure of stratum corneum hydration rather than barrier flux, and the EEMCO guidance for electrical methods of hydration assessment describes the calibration and probe-contact standards needed for reproducible readings [26]. Corneometry and TEWL are frequently paired in the same assessment because they capture distinct, only loosely correlated dimensions of barrier status: a plaque can be simultaneously dehydrated at the surface and highly permeable to water loss from below, and reporting only one of the two measures risks an incomplete picture of barrier competence [27,28]. Surface pH measurement by flat glass electrode is the third routine instrument, and because pH governs the enzymatic lipid processing described above, pH mapping across lesional, perilesional, and non-lesional skin has

been used to characterize the spatial gradient of barrier recovery around a resolving plaque [17,16].

Table 1: Instrumented methods used to assess skin barrier function in plaque psoriasis, the barrier parameter each captures, and the governing methodological standard [22,23,26,29].			
Method	Parameter measured	Typical clinical use	Standardization reference
Open/closed-chamber evaporimetry	Transepidermal water loss (TEWL)	Barrier permeability at lesional, perilesional and non-lesional sites	Pinnagoda consensus; EEMCO TEWL guidance
Capacitance corneometry	Stratum corneum hydration	Paired with TEWL to separate hydration from permeability status	EEMCO electrical-methods guidance
Flat glass surface electrode	Surface pH	Spatial pH mapping across resolving plaques	EEMCO/dermatologic pH literature
Confocal Raman spectroscopy	Natural moisturizing factor, lipid profile	Distinguishing filaggrin- from lipid-driven barrier deficits	Kezic filaggrin-genotype biomarker studies

Natural moisturizing factor content, assessed either indirectly through corneometry-correlated biomarkers or directly by confocal Raman spectroscopy, offers a further layer of resolution, particularly useful for distinguishing barrier deficits driven by filaggrin-pathway abnormalities from those driven primarily by lipid or proliferative abnormalities, a distinction increasingly relevant now that psoriasis and atopic dermatitis are understood to share some, but not all, barrier mechanisms [29,30,31]. In practice, adult dermatology patients being followed for plaque psoriasis are most commonly assessed with paired TEWL and corneometry readings taken at standardized lesional, perilesional, and contralateral non-lesional sites, a protocol design that traces directly back to the methodological standardization work summarized above [23,26].

4. Barrier parameters as biomarkers of disease severity and course

A substantial body of evidence links instrumented barrier readings to conventional clinical severity measures in plaque psoriasis, though the relationship is not always linear. Lesional TEWL rises with increasing plaque

thickness and scale, and several cohort assessments have reported that non-lesional skin in patients with more extensive disease also runs higher TEWL than non-lesional skin in patients with limited disease, suggesting a field effect that extends barrier compromise beyond visibly involved plaques [9,32]. This field effect is clinically relevant because it implies that barrier testing at an apparently uninvolved site can still carry information about overall disease activity, a property not shared by visual severity indices, which by definition can only be scored where lesions are visible.

Table 2: Reported associations between skin barrier parameters and plaque psoriasis disease characteristics across the reviewed literature [9,12,33,15].			
Barrier parameter	Site	Reported association	Source
TEWL	Lesional plaque	Rises with plaque thickness and scale severity	[9]
Ceramide content	Lesional scale	Reduced content correlates with measured water-barrier impairment	[12]
Serum IL-17A/IL-22/IL-6	Systemic	Elevated with more extensive disease; IL-22 alters keratinocyte lipid genes	[33]
Tight junction proteins	Stratum granulosum	Claudin/occludin changes precede overt hyperproliferation	[15]

Cytokine correlates of barrier disruption reinforce the biomarker argument. Elevated serum interleukin-17A, interleukin-22, and interleukin-6 have each been reported in patients with more extensive plaque disease, and interleukin-22 in particular has direct effects on keratinocyte differentiation and lipid-processing gene expression that plausibly link systemic inflammatory burden to the local barrier phenotype measured at the skin surface [33,1]. Because TEWL and corneometry are continuous, instrument-derived values rather than ordinal clinician scores, they offer a level of measurement precision that makes them attractive as secondary endpoints in interventional psoriasis studies, particularly

for detecting early barrier recovery that precedes visible improvement in erythema or induration.

5. Effect of topical and systemic therapies on barrier recovery

Topical corticosteroids, still the most widely used first-line therapy for localized plaque psoriasis, illustrate the double-edged relationship between treatment and barrier status. Kao and colleagues showed in a mechanistic model that short-term glucocorticoid application, while suppressing the inflammatory signal driving hyperproliferation, simultaneously compromises permeability barrier homeostasis and reduces stratum corneum integrity through direct inhibition of lipid synthesis, meaning that corticosteroid-treated plaques can show improved erythema scores while barrier instrumentation continues to register elevated TEWL [34]. This finding underlies the now-common practice of co-prescribing barrier-repair emollients alongside topical corticosteroid regimens, a strategy supported by Lodén's review of emollient mechanisms and by ceramide-specific formulation data from Draelos showing accelerated barrier-parameter normalization when a ceramide-dominant moisturizer is added to standard therapy [35,36].

Systemic and biologic therapies, by resolving the hyperproliferative and inflammatory drive at its source rather than only suppressing the surface inflammatory readout, have generally been associated with more durable barrier-parameter normalization once lesions clear, consistent with the mechanistic model in which lamellar body secretion and lipid processing recover once keratinocyte transit time returns toward normal [8,37]. The contrasting immunologic architecture of psoriasis and atopic dermatitis described by Guttman-Yassky and colleagues is a useful frame here: because interleukin-17/23-axis-directed biologics act on a different inflammatory circuit than the type 2 pathway targeted in atopic dermatitis, barrier normalization in

psoriasis under biologic therapy follows a somewhat different trajectory and timeline than the barrier improvement reported after dupilumab or similar agents in atopic dermatitis trials, a distinction relevant when barrier endpoints from the atopic dermatitis literature are extrapolated to psoriasis without adjustment [31,30].

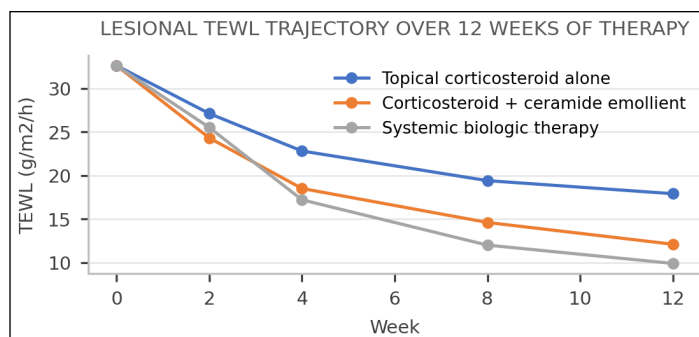


Figure 2: Lesional TEWL trajectory over a 12-week course of therapy, comparing topical corticosteroid alone, topical corticosteroid plus ceramide-dominant emollient, and systemic biologic therapy, illustrating the more durable barrier normalization reported with biologic and combination emollient regimens [34,36,35,8].

6. Standardization challenges and practical limitations

Despite the mechanistic and biomarker evidence reviewed above, several practical obstacles have limited routine barrier testing in plaque psoriasis clinics. Ambient humidity and temperature materially affect TEWL readings, and clinics without a controlled acclimatization room struggle to generate values comparable across visits, a problem the EEMCO and Pinnagoda guidance documents were explicitly written to address but that many clinical settings cannot fully implement [22,23]. Probe placement on an irregular, scaling plaque surface introduces variance that is not present when the same instruments are used on smooth non-lesional skin, and repeated measurement at precisely the same anatomic point over a treatment course, needed to track individual-patient trends, requires photographic or grid-based site marking that adds time to a standard clinic visit.

Instrument cost and the absence of a validated,

universally adopted normal range specific to psoriatic non-lesional skin, as distinct from the general adult population ranges summarized by Akdeniz and colleagues, further limit interpretability at the individual-patient level [25]. Corneometry and pH readings are similarly sensitive to recent bathing, occlusion, and topical product application, and consensus protocols recommend a washout period before measurement that is not always feasible to enforce in a busy dermatology practice. Finally, because barrier parameters correlate with but do not fully substitute for clinician-scored severity indices such as PASI, and because the strength of that correlation varies across the studies reviewed here depending on measurement site and disease extent, barrier instrumentation is best positioned as an adjunct to, rather than a replacement for, standard clinical scoring in current plaque psoriasis management [32,27].

7. Conclusion

Plaque psoriasis is, at the level of the stratum corneum, a disease of barrier failure as much as it is a disease of keratinocyte hyperproliferation and cytokine-driven inflammation, with well-characterized deficits in ceramide content, lamellar lipid organization, tight junction integrity, and surface pH regulation that are directly measurable with standard dermatologic instrumentation [3,10,15]. Transepidermal water loss, capacitance-based hydration, and surface pH, applied with the methodological rigor the EEMCO and Pinnagoda consensus documents describe, provide continuous, objective readouts that correlate with disease extent, respond measurably to both topical and systemic therapy, and can detect field-level barrier compromise in skin that appears clinically uninvolved [23,26,9]. Wider adoption in routine adult dermatology practice will depend on resolving the standardization and normative-range gaps discussed above, but the existing evidence base already supports incorporating instrumented barrier assessment as a complementary, mechanistically grounded measure

alongside conventional severity scoring in the management of plaque psoriasis [25,7].

8. References

1. Lowes, M.A., Suárez-Fariñas, M., Krueger, J.G. (2014) Immunology of psoriasis. *Annual Review of Immunology*, 32: 227-255. DOI: [10.1146/annurev-immunol-032713-120225](https://doi.org/10.1146/annurev-immunol-032713-120225)
2. Griffiths, C.E.M., Barker, J.N.W.N. (2007) Pathogenesis and clinical features of psoriasis. *Lancet*, 370(9583): 263-271. DOI: [10.1016/S0140-6736\(07\)61128-3](https://doi.org/10.1016/S0140-6736(07)61128-3)
3. Elias, P.M. (2008) Skin barrier function. *Current Allergy and Asthma Reports*, 8(4): 299-305. DOI: [10.1007/s11882-008-0048-0](https://doi.org/10.1007/s11882-008-0048-0)
4. Proksch, E., Brandner, J.M., Jensen, J.M. (2008) The skin: an indispensable barrier. *Experimental Dermatology*, 17(12): 1063-1072. DOI: [10.1111/j.1600-0625.2008.00786.x](https://doi.org/10.1111/j.1600-0625.2008.00786.x)
5. Madison, K.C. (2003) Barrier function of the skin: "la raison d'être" of the epidermis. *Journal of Investigative Dermatology*, 121(2): 231-241. DOI: [10.1046/j.1523-1747.2003.12359.x](https://doi.org/10.1046/j.1523-1747.2003.12359.x)
6. Boehncke, W.H., Schön, M.P. (2015) Psoriasis. *Lancet*, 386(9997): 983-994. DOI: [10.1016/S0140-6736\(14\)61909-7](https://doi.org/10.1016/S0140-6736(14)61909-7)
7. Armstrong, A.W., Read, C. (2020) Pathophysiology, clinical presentation, and treatment of psoriasis: a review. *JAMA*, 323(19): 1945-1960. DOI: [10.1001/jama.2020.4006](https://doi.org/10.1001/jama.2020.4006)
8. Menon, G.K., Feingold, K.R., Elias, P.M. (1992) Lamellar body secretory response to barrier disruption. *Journal of Investigative Dermatology*, 98(3): 279-289. DOI: [10.1111/1523-1747.ep12497866](https://doi.org/10.1111/1523-1747.ep12497866)
9. Ghadially, R., Reed, J.T., Elias, P.M. (1996) Stratum corneum structure and function correlates with phenotype in psoriasis. *Journal of Investigative Dermatology*, 107(4): 558-564. DOI: [10.1111/1523-1747.ep12582813](https://doi.org/10.1111/1523-1747.ep12582813)
10. Motta, S., Monti, M., Sesana, S., Caputo, R., Carelli, S., Ghidoni, R. (1993) Ceramide composition of the psoriatic scale.

- Biochimica et Biophysica Acta, 1182(2): 147-151. DOI: [10.1016/0925-4439\(93\)90135-N](https://doi.org/10.1016/0925-4439(93)90135-N)
11. Schäfer, L., Kragballe, K. (1991) Abnormalities in epidermal lipid metabolism in psoriasis. *Journal of Investigative Dermatology*, 96(1): 10-15. DOI: [10.1111/1523-1747.ep12514653](https://doi.org/10.1111/1523-1747.ep12514653)
 12. Motta, S., Sesana, S., Monti, M., Giuliani, A., Caputo, R. (1994) Abnormality of water barrier function in psoriasis. Role of ceramide fractions. *Archives of Dermatology*, 130(4): 452-456.
 13. van Smeden, J., Bouwstra, J.A. (2016) Stratum corneum lipids: their role for the skin barrier function in healthy subjects and atopic dermatitis patients. *Current Problems in Dermatology*, 49: 8-26. DOI: [10.1159/000441540](https://doi.org/10.1159/000441540)
 14. Cork, M.J., Danby, S.G., Vasilopoulos, Y., Hadgraft, J., Lane, M.E., Moustafa, M., et al. (2009) Epidermal barrier dysfunction in atopic dermatitis. *Journal of Investigative Dermatology*, 129(8): 1892-1908. DOI: [10.1038/jid.2009.133](https://doi.org/10.1038/jid.2009.133)
 15. Kirschner, N., Poetzel, C., von den Driesch, P., Wladykowski, E., Moll, I., Behne, M.J., et al. (2009) Alteration of tight junction proteins is an early event in psoriasis: putative involvement of proinflammatory cytokines. *American Journal of Pathology*, 175(3): 1095-1106. DOI: [10.2353/ajpath.2009.080973](https://doi.org/10.2353/ajpath.2009.080973)
 16. Danby, S.G., Cork, M.J. (2016) pH in atopic dermatitis. *Current Problems in Dermatology*, 49: 95-107. DOI: [10.1159/000441550](https://doi.org/10.1159/000441550)
 17. Levin, J., Maibach, H. (2008) Human skin buffering capacity: an overview. *Skin Research and Technology*, 14(2): 121-126. DOI: [10.1111/j.1600-0846.2007.00271.x](https://doi.org/10.1111/j.1600-0846.2007.00271.x)
 18. Jensen, J.M., Fölster-Holst, R., Baranowsky, A., Schunck, M., Winoto-Morbach, S., Neumann, C., et al. (2004) Impaired sphingomyelinase activity and epidermal differentiation in atopic dermatitis. *Journal of Investigative Dermatology*, 122(6): 1423-1431. DOI: [10.1111/j.0022-202X.2004.22621.x](https://doi.org/10.1111/j.0022-202X.2004.22621.x)
 19. Nickoloff, B.J. (1999) Skin innate immune system in psoriasis: friend or foe? *Journal of Clinical Investigation*, 104(9): 1161-1164. DOI: [10.1172/JCI8633](https://doi.org/10.1172/JCI8633)
 20. Pasparakis, M., Haase, I., Nestle, F.O. (2014) Mechanisms regulating skin immunity and inflammation. *Nature Reviews Immunology*, 14(5): 289-301. DOI: [10.1038/nri3646](https://doi.org/10.1038/nri3646)
 21. Gutowska-Owsiak, D., Ogg, G.S. (2012) The epidermis as an adjuvant. *Journal of Investigative Dermatology*, 132(3 Pt 2): 940-948. DOI: [10.1038/jid.2011.398](https://doi.org/10.1038/jid.2011.398)
 22. Pinnagoda, J., Tupker, R.A., Agner, T., Serup, J. (1990) Guidelines for transepidermal water loss (TEWL) measurement. *Contact Dermatitis*, 22(3): 164-178. DOI: [10.1111/j.1600-0536.1990.tb01553.x](https://doi.org/10.1111/j.1600-0536.1990.tb01553.x)
 23. Rogiers, V. (2001) EEMCO guidance for the assessment of transepidermal water loss in cosmetic sciences. *Skin Pharmacology and Applied Skin Physiology*, 14(2): 117-128. DOI: [10.1159/000056341](https://doi.org/10.1159/000056341)
 24. Fluhr, J.W., Feingold, K.R., Elias, P.M. (2006) Transepidermal water loss reflects permeability barrier status: validation in human and rodent in vivo and ex vivo models. *Experimental Dermatology*, 15(7): 483-492. DOI: [10.1111/j.1600-0625.2006.00437.x](https://doi.org/10.1111/j.1600-0625.2006.00437.x)
 25. Akdeniz, M., Gabriel, S., Lichterfeld-Kottner, A., Blume-Peytavi, U., Kottner, J. (2018) Transepidermal water loss in healthy adults: a systematic review and meta-analysis update. *British Journal of Dermatology*, 179(5): 1049-1055. DOI: [10.1111/bjd.17025](https://doi.org/10.1111/bjd.17025)
 26. Berardesca, E. (1997) EEMCO guidance for the assessment of stratum corneum hydration: electrical methods. *Skin Research and Technology*, 3(2): 126-132. DOI: [10.1111/j.1600-0846.1997.tb00174.x](https://doi.org/10.1111/j.1600-0846.1997.tb00174.x)
 27. Sator, P.G., Schmidt, J.B., Hönigsmann, H. (2003) Comparison of epidermal hydration and skin surface lipids in healthy individuals and in patients with atopic dermatitis. *Journal of the American Academy of Dermatology*, 48(3): 352-358. DOI: [10.1067/mjd.2003.105](https://doi.org/10.1067/mjd.2003.105)

28. Seidenari, S., Giusti, G. (1995) Objective assessment of the skin of children affected by atopic dermatitis: a study of pH, capacitance and TEWL in eczematous and clinically uninvolved skin. *Acta Dermato-Venereologica*, 75(6): 429-433. DOI: [10.2340/0001555575429433](https://doi.org/10.2340/0001555575429433)
29. Kezic, S., Kammeyer, A., Calkoen, F., Fluhr, J.W., Bos, J.D. (2009) Natural moisturizing factor components in the stratum corneum as biomarkers of filaggrin genotype. *British Journal of Dermatology*, 161(5): 1098-1104. DOI: [10.1111/j.1365-2133.2009.09342.x](https://doi.org/10.1111/j.1365-2133.2009.09342.x)
30. Kim, B.E., Leung, D.Y.M. (2018) Significance of skin barrier dysfunction in atopic dermatitis. *Allergy, Asthma & Immunology Research*, 10(3): 207-215. DOI: [10.4168/aaair.2018.10.3.207](https://doi.org/10.4168/aaair.2018.10.3.207)
31. Guttman-Yassky, E., Nograles, K.E., Krueger, J.G. (2011) Contrasting pathogenesis of atopic dermatitis and psoriasis--part I: clinical and pathologic concepts. *Journal of Allergy and Clinical Immunology*, 127(5): 1110-1118. DOI: [10.1016/j.jaci.2011.01.053](https://doi.org/10.1016/j.jaci.2011.01.053)
32. Lavrijsen, A.P.M., Bouwstra, J.A., Gooris, G.S., Weerheim, A., Boddé, H.E., Ponc, M. (1993) Barrier function parameters in various keratinization disorders: transepidermal water loss and vascular response to hexyl nicotinate. *British Journal of Dermatology*, 129(5): 547-553. DOI: [10.1111/j.1365-2133.1993.tb00485.x](https://doi.org/10.1111/j.1365-2133.1993.tb00485.x)
33. Takahashi, H., Tsuji, H., Honma, M., Ishida-Yamamoto, A., Iizuka, H. (2014) Increased serum levels of interleukin-6, interleukin-22 and interleukin-17A in patients with psoriasis. *Journal of Dermatology*, 41(4): 341-343. DOI: [10.1111/1346-8138.12496](https://doi.org/10.1111/1346-8138.12496)
34. Kao, J.S., Fluhr, J.W., Man, M.Q., Fowler, A.J., Hachem, J.P., Crumrine, D., et al. (2003) Short-term glucocorticoid treatment compromises both permeability barrier homeostasis and stratum corneum integrity. *Journal of Investigative Dermatology*, 120(3): 456-464. DOI: [10.1046/j.1523-1747.2003.12053.x](https://doi.org/10.1046/j.1523-1747.2003.12053.x)
35. Lodén, M. (2003) Role of topical emollients and moisturizers in the treatment of dry skin barrier disorders. *American Journal of Clinical Dermatology*, 4(11): 771-788. DOI: [10.2165/00128071-200304110-00005](https://doi.org/10.2165/00128071-200304110-00005)
36. Draelos, Z.D. (2008) The effect of ceramide-containing skin care products on eczema resolution duration. *Cutis*, 81(1): 87-91.
37. Choi, E.H., Man, M.Q., Wang, F., Zhang, X., Brown, B.E., Feingold, K.R., et al. (2005) Is endogenous glycerol a determinant of stratum corneum hydration in humans? *Journal of Investigative Dermatology*, 125(2): 288-293. DOI: [10.1111/j.0022-202X.2005.23799.x](https://doi.org/10.1111/j.0022-202X.2005.23799.x)