Barrier-First Derm: Integrative Care of Skin Conditions

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About Me

& Disclosures

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- Compensated by BRB CE for this presentation

Agenda & Learning Outcomes

- Describe the structure and function of the epidermal barrier and its role in inflammatory skin conditions
- Explain how barrier dysfunction contributes to acne, rosacea, and perioral dermatitis
- Differentiate between common inflammatory dermatoses using clinical presentation and barrier status
- Identify key signs of impaired barrier function, including increased sensitivity and TEWL (trans epidermal water loss)
- Apply a phased, barrier-first approach to treatment planning
- Determine when patients are ready to progress to active therapies
- Select appropriate internal, topical, and procedural interventions based on barrier integrity
- Recognize and avoid common iatrogenic factors that exacerbate skin conditions
- Integrate microbiome and immune considerations into dermatological care
- Utilize clinical frameworks to improve treatment tolerance, adherence, and outcomes

The Shift in Skin Disease:
Why Our Patients Are Getting Worse

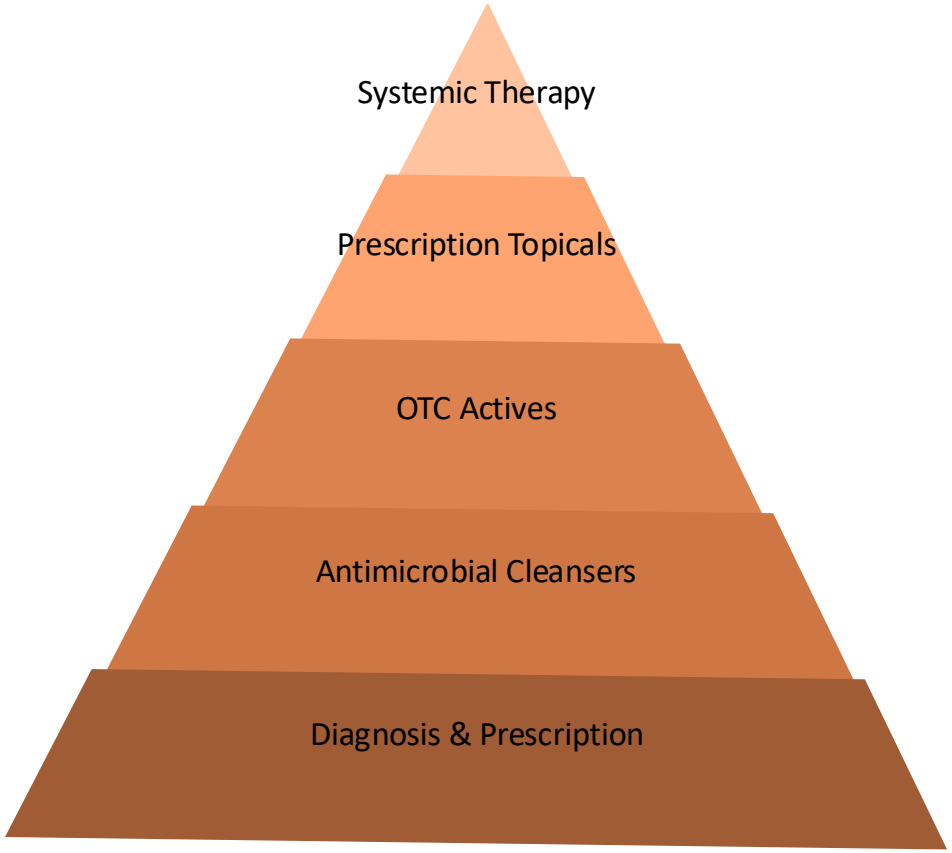
Why Standard Protocols Fail

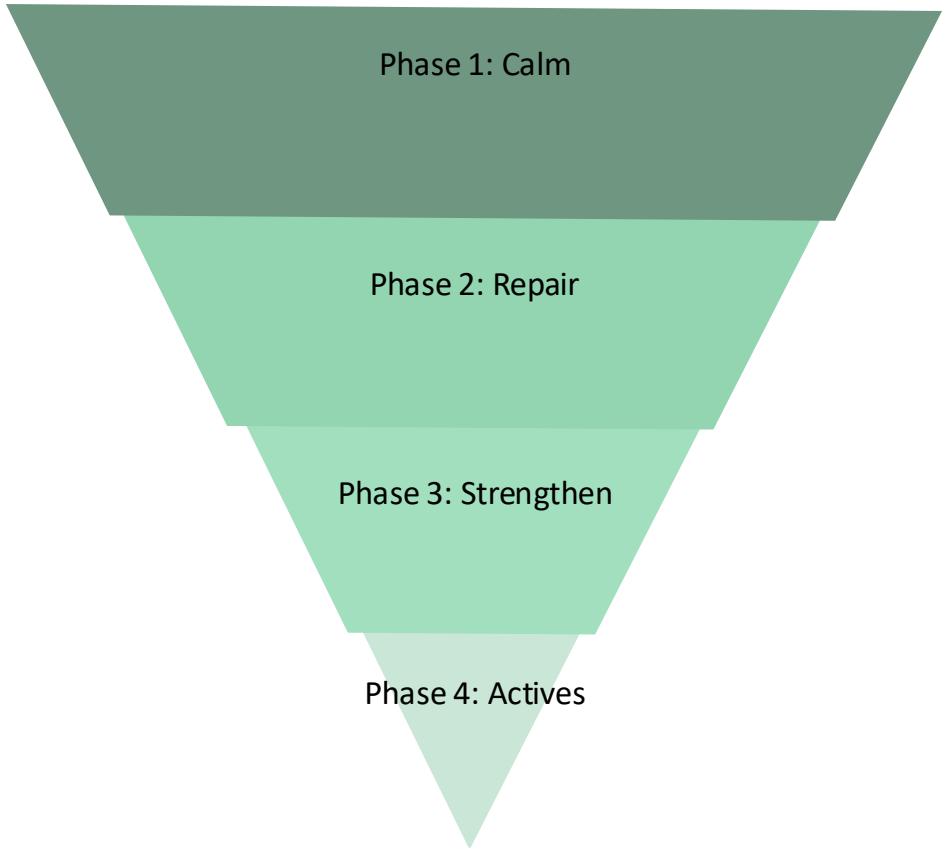
Most conventional protocols begin with antimicrobials or actives.

Barrier dysfunction remains untreated, creating a cycle:
flare → treat → temporary improvement → worse flare

The Treatment Ladder vs The Barrier Funnel

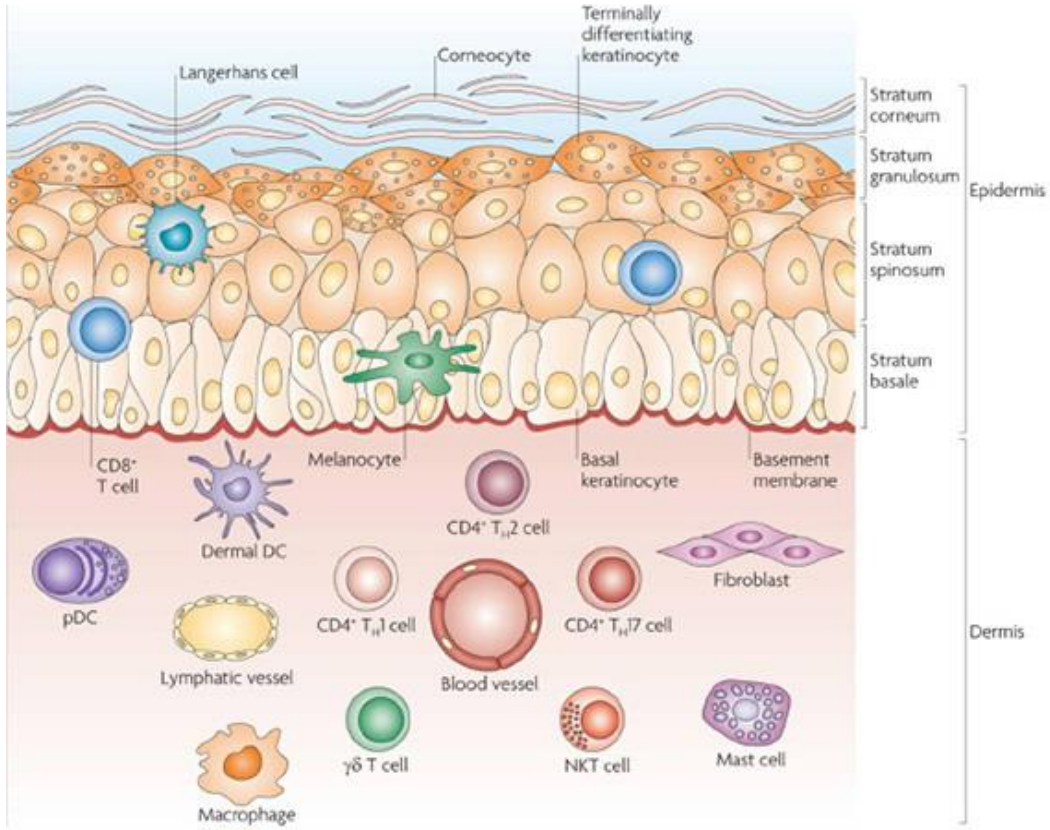
Result: flare → treat → worse flare
Barrier unaddressed throughout





Barrier-ready skin
Actives perform as intended

The barrier as an immune organ: Beyond "protection”



Stratum Corneum Architecture

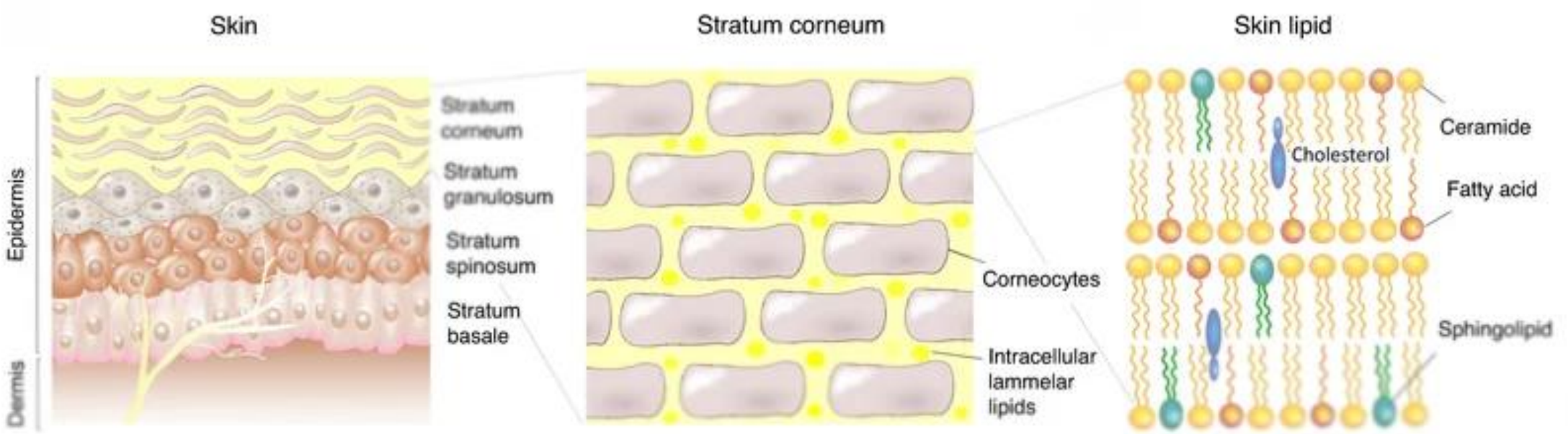
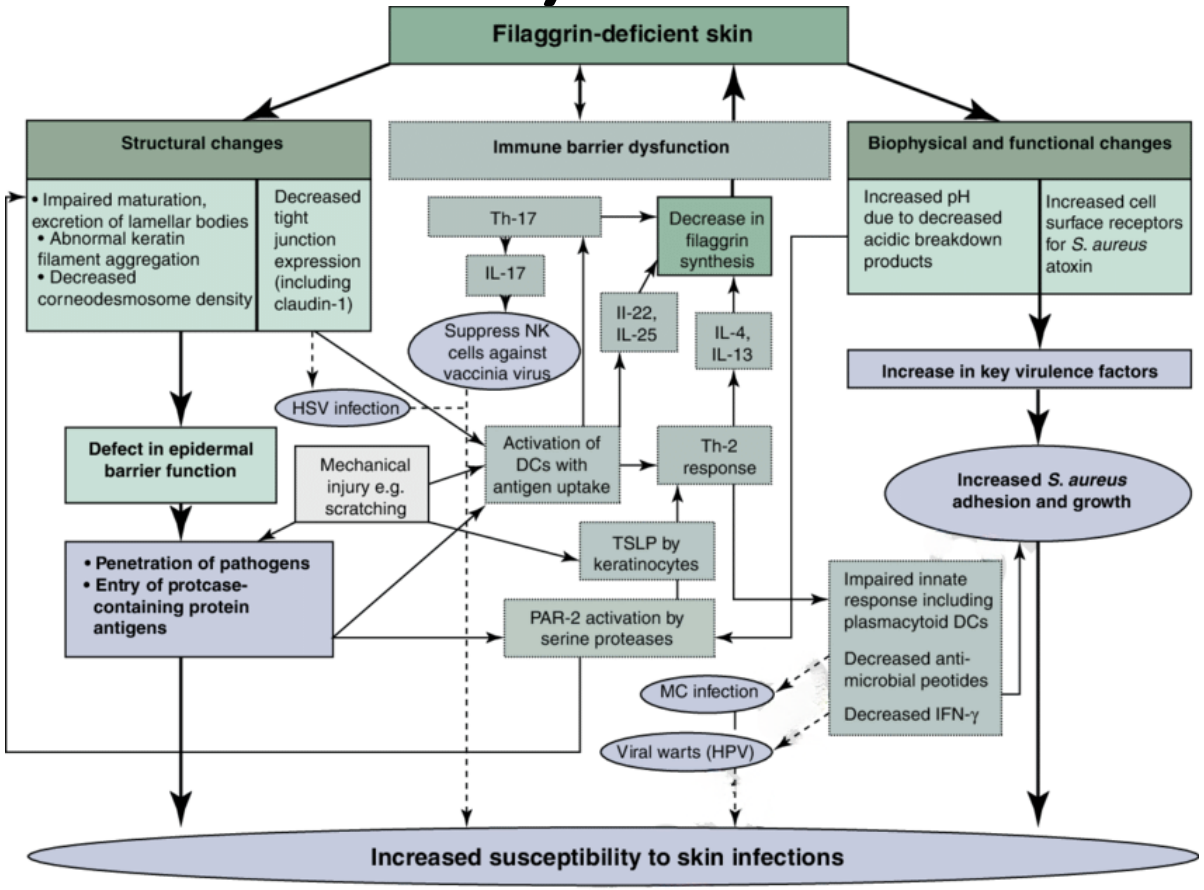


Image from: Gray, Natalie & Limberg, Maren & Bräuer, Anja & Raap, Ulrike. (2021). Novel functions of S1P in chronic itchy and inflammatory skin diseases. Journal of the European Academy of Dermatology and Venereology. 36. 365-372. 10.1111/jdv.17764.

Ceramide Subclasses

Fatty Acid / Sphingoid	Non-hydroxy fatty acid [N]	α-hydroxy fatty acid [A]	Esterified ω-hydroxy fatty acid [EO]
Dihydrosphingosine [DS]	Ceramide NDS (aka Ceramide 10)	Ceramide ADS (aka Ceramide 11)	Ceramide EODS (aka Ceramide 12)
Sphingosine [S]	Ceramide NS (aka Ceramide 2)	Ceramide AS (aka Ceramide 5)	Ceramide EOS (aka Ceramide 1)
Phytosphingosine [P]	Ceramide NP (aka Ceramide 3)	Ceramide AP (aka Ceramide 6)	Ceramide EOP (aka Ceramide 9)
6-hydroxy sphingosine [H]	Ceramide NH (aka Ceramide 8)	Ceramide AH (aka Ceramide 7)	Ceramide EOH (aka Ceramide 4)

Filaggrin, NMF (Natural Moisturizing Factor) & TEWL



Gan, E.Y., Cai, S.C.S., Tang, M.B.Y. (2014). Filaggrin and Skin Infections. In: Thyssen, J., Maibach, H. (eds) Filaggrin. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-642-54379-1_27

The skin microbiome ecosystem - commensals, opportunists & quorum sensing

Healthy skin is a balanced ecosystem, not a sterile surface

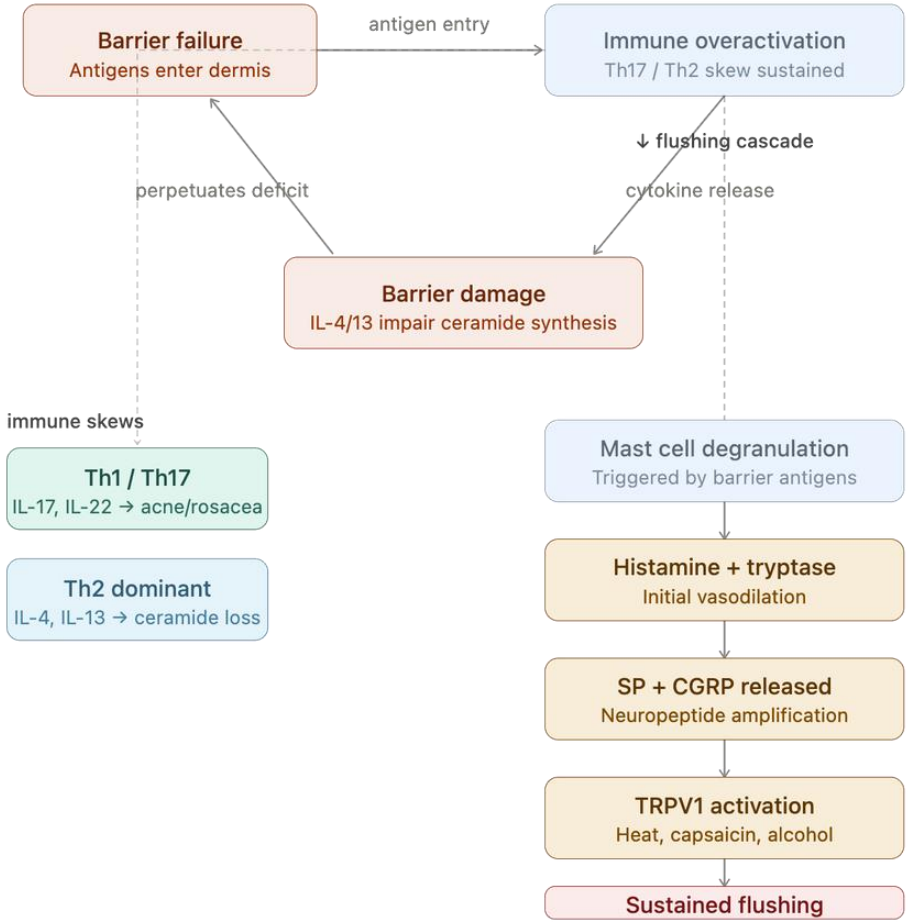
The dominant commensal bacteria on human skin are primarily represented by three major genera:

- Staphylococcus (Staphylococcus epidermidis)
- Cutibacterium (Cutibacterium acnes)
- Corynebacterium

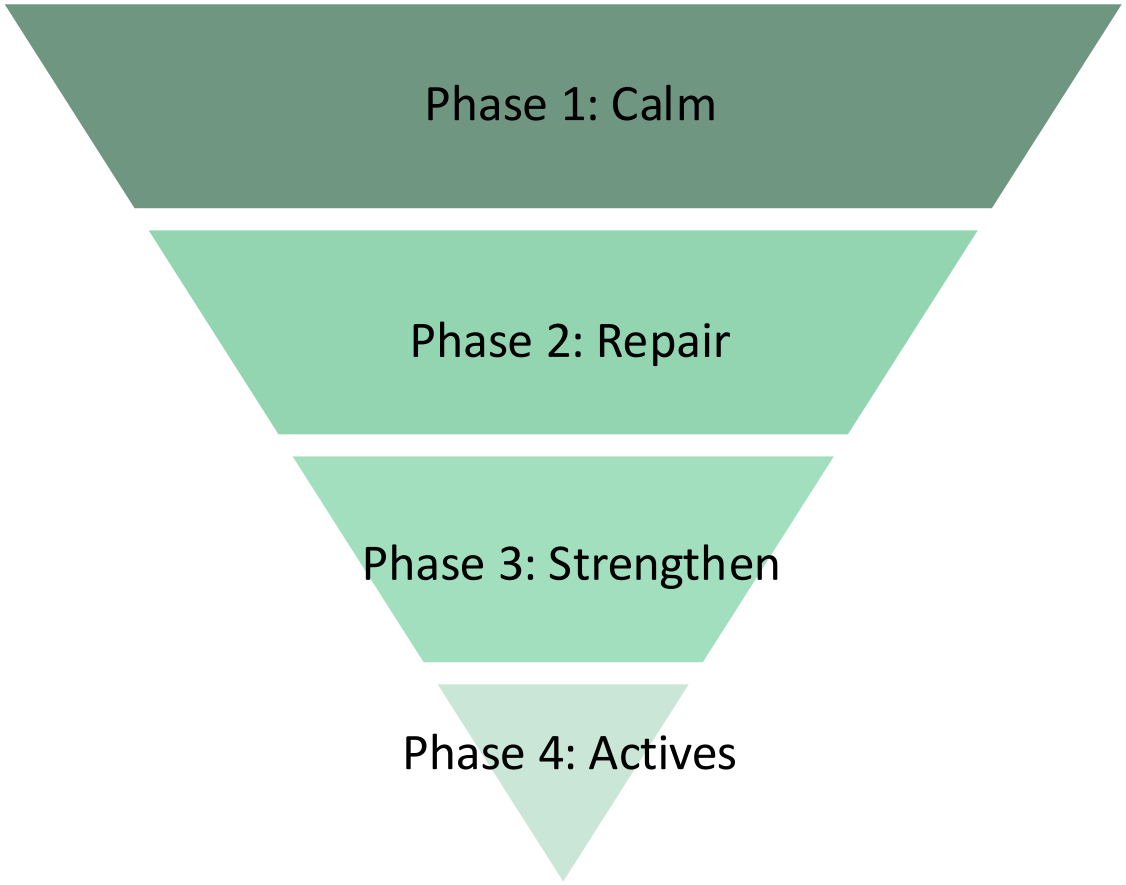
When microbes become pathogenic...

- Is the presentation follicular in distribution?
- Is there reason to suspect pH disruption - alkaline cleansers, frequent washing, high-pH actives?
- Are you looking at flushing, central face distribution, and a neurogenic quality to the reactivity?
- Is barrier disruption the primary finding?

Immune Overactivation: Th17, Mast Cells & the Flushing Cascade



The Barrier-First Method™



Phase 1: Calm

"Less is a prescription."

Indicators: active inflammation, stinging, flushing, barrier collapse (post-retinoid, post-peel, steroid withdrawal)

Goal: reduce antigen penetration and inflammatory signaling without introducing new stimuli

Stop immediately: all actives (retinoids, AHAs, BHAs, vitamin C >5%), all exfoliants (physical or chemical), foaming/SLS cleansers, anything that stings on application

Use: a single gentle, pH-balanced cleanser (no fragrance, no surfactant cocktails); a simple occlusive or barrier-supportive moisturizer; cool water temperatures

Internal supports: anti-inflammatory diet, reduce high-histamine foods, omega-3s, consider quercetin for mast cell stabilization

Signs that you can move patient to Phase 2: stinging resolves and baseline hydration stabilizes

Phase 2: Repair

Indicators: inflammation has subsided; skin tolerates plain water without stinging; TEWL is stabilizing

Goal: restore ceramide subclasses, intercellular lipid ratios, and tight junction proteins

Avoid: humectant-only products (HA serums without lipid backing), prolonged occlusion that can dysregulate aquaporin function

Use: multi-ceramide moisturizers with cholesterol and fatty acids, peptide complexes, target moisture retention

Internal supports: linoleic acid (hemp, sunflower oil), zinc carnosine for junction integrity, collagen peptides for dermal matrix support

Signs that you can move patient to Phase 3: skin tolerates light mineral SPF without reactivity

Phase 3: Strengthen

Indicators: barrier is structurally repaired; reactivity is significantly reduced; patient tolerates SPF and single-ingredient topicals

Goal: move from structural repair to immune and microbial tolerance - prepare the skin to handle actives without reactivity

Avoid: introducing multiple products at the same time, niacinamide >4%, broad-spectrum antimicrobial topicals

Use: topical postbiotics (lactobacillus ferment lysate), centella asiatica for TRPV1 modulation

Internal supports: oral probiotics (Lactobacillus rhamnosus, L. reuteri - evidence in atopic skin); adaptogenic herbs for HPA axis normalization in stress-driven flares

Signs that you can move patient to Phase 4: exit when the "Actives Are Earned" checklist is met:

- No new reactive episodes for 2 weeks
- SPF tolerated without stinging
- At least one active ingredient tolerated without reactivity
- Patient reports skin feels stable

Phase 4: Actives

The same active ingredient produces dramatically different outcomes depending on barrier status

Indicators: all Phase 3 exit criteria met; "Actives Are Earned" checklist complete

Goal: introduce corrective and cosmetically active ingredients with minimum barrier disruption and maximum therapeutic effect, match the active to the primary driver

Avoid: stacking multiple actives simultaneously

Active Selection Principles: start at lowest available concentration, apply active for 5–10 minutes then rinse during initial introduction, use 2–3 nights per week before daily use

Internal supports: continue all Phase 2–3 internal protocols; add specific support based on driver (e.g., saw palmetto + zinc for androgenic acne; quercetin + DAO support for rosacea)

“Actives are Earned” - Readiness Checklist

- No stinging with water, cleanser, or plain moisturizer for ≥2 consecutive weeks
- Flushing/erythema at resting baseline is reduced from intake presentation
- Skin retains moisture for ≥4 hours without reapplication
- Reactions to new products are predictable (minor dryness rather than burning, stinging, or pustular flare)
- Patient rates skin as "stable" rather than "reactive" on a simple 1–10 self-assessment
- Makeup (if applicable) no longer oxidizes rapidly or causes end-of-day irritation
- At least one test product introduced in Phase 3 without reactive response

Case-Based Application

Post-Retinoid Acne Flare

Patient: 34F, 8 weeks on tretinoin 0.05%
Also using niacinamide serum, vitamin C, and an exfoliating toner nightly alongside tretinoin. Foaming cleanser twice daily.

Chief Concern: Worsening breakouts, irritation and burning sensation

Clinical Findings: Papulopustular acne worse than baseline, erythema, stinging with all products, peeling across cheeks and forehead. Patient reports "purging" but 8 weeks in with no improvement.

Steroid-Induced Perioral Dermatitis

Patient: 29F, 6-month topical steroid use

Chief Concern: Perioral rash, rebound flaring, Skin described as “on fire"

Clinical Findings: Ring of papulopustular lesions within the nasolabial grooves and chin with sparing of the vermilion border. Redness, scaling, intense burning on any product application. Had been using low-potency topical corticosteroid (prescribed for eczema) for 6 months with escalating dose.

Rosacea with Flushing Dominance

Patient: 47F, 10-year rosacea history

Chief Concern: Uncontrolled flushing, heat & alcohol sensitivity

Clinical Findings: Persistent central facial erythema, episodic flushing (triggered by heat, wine, exercise, stress), telangiectasia, no active pustules. Has tried metronidazole gel, doxycycline, brimonidine - partial improvement only.

Adult Jawline Acne with Sensitivity

Patient: 38F, hormonally-driven jawline acne

Chief Concern: Cyclical deep cysts, skin "too sensitive" for treatment

Clinical Findings: Deep, painful nodular lesions along jaw and chin, predictably worse in luteal phase. Remainder of face is dry, reactive, and sensitive. Cyclical pattern (premenstrual), high-stress occupation, history of isotretinoin at age 22. Multiple failed retinoid attempts in past 5 years - all abandoned due to sensitivity.

"Fungal acne" Misdiagnosis

Patient: 26M, self-diagnosed via social media

Chief Concern: Forehead bumps not responding to ketoconazole

Clinical Findings: Uniform, monomorphic closed comedones across forehead and hairline. Mild oiliness. No pustules, no inflammation, no central face involvement. Has been using ketoconazole shampoo as a face wash for 6 weeks without improvement. High-protein diet, daily protein shakes with whey, gym 5x/week, occlusive sunscreen. No antibiotic history. Non-reactive skin otherwise - no stinging, no sensitivity.

Aesthetic Procedures

Modalities by Phase

Phase	Appropriate Modalities	Rationale
Phase 1 Calm	• LED phototherapy (red / near-infrared) • Gentle manual lymphatic drainage • Cool compress therapy	Non-injurious, anti-inflammatory; supports immune resolution without adding barrier stress
Phase 2 Repair	• Low-level laser therapy (LLLT) • Barrier-supportive facials (no exfoliation, no steam) • Ultrasound-assisted product penetration	Biostimulatory without trauma; supports lipid synthesis and cellular repair activity
Phase 3 Strengthen	• NeoSkin-type photothermal treatments • IPL on stable rosacea only • Gentle enzyme-based exfoliation • RF microneedling prep assessment	Barrier functional; tolerates controlled heat stress; vascular targeting appropriate when baseline erythema is stable
Phase 4 Actives	• Microneedling (select cases, no active inflammation) • Superficial chemical peels (lactic 20–30%) • Fractional laser (low fluence) • PRP	Full wound-healing capacity restored; skin can generate repair response and resolve controlled injury

What to Avoid:
High-Risk Modality & Timing Errors

- ✗ Microneedling during active inflammation
- ✗ Aggressive chemical peels on sensitized or rosacea-prone skin
- ✗ IPL on unstable rosacea
- ✗ Ablative resurfacing without documented preparation
- ✗ Multiple modalities in a single session on compromised skin

The Seven Most Common Iatrogenic Contributors to Chronic Skin Disease

1. Over-prescribing actives without barrier assessment
2. Skipping the repair phases
3. Treating the diagnosis, not the skin status
4. Stacking multiple actives simultaneously
5. Interpreting reactivity as adjustment and pushing through
6. Recommending procedures without barrier preparation
7. Addressing topical factors only (spend slightly more time)

Key Takeaways:
5 Principles to Anchor Clinical Practice

1. Barrier integrity governs all treatment outcomes: no active, procedure, or internal intervention performs as intended on a compromised barrier. Assess first, always.
2. Repair precedes correction - the sequence is non-negotiable. Calm, Repair, Strengthen, Actives. No phase is optional. No shortcut produces a durable result.
3. Less is a clinical prescription: in Phase 1, subtraction is the most powerful intervention available. Eliminating five products is a treatment plan.
4. The internal environment determines the topical ceiling - histamine load, androgen burden, gut dysbiosis, and nutritional status all limit how well the best topical protocol performs.
5. Reactivity is information, not failure — stinging, flushing, and barrier symptoms during treatment are signals to pull back and reassess, not to push through. The skin is always telling you where it is.

Questions