METABOLIC PEELS

A Paradigm Shift in Chemical Peeling

From injury-based exfoliation to biological skin regulation

I'm here to **challenge a paradigm,** not to sell products.
This lecture is about **mechanism** and **strategy**.

Alain Tenenbaum, MD, PhD, DSc
Facial Plastic & Aesthetic Surgery | Chemical Peel Specialist | International Peelings Society

chemicalpeeling.com

The Classic Peel Model Has a Conceptual Flaw

We designed peels around injury — not around the skin's biology

INTERNATIONAL PEELINGS SOCIETY

THE STANDARD MENTAL MODEL

⚠ **BARRIER DISRUPTION:**
is not a side effect — it IS the mechanism.
We normalize a biological insult.

⚠ **FORMULATION VARIABILITY:**
means two "35% TCA" peels can behave completely differently.
Concentration ≠ behavior

⚠ **VISIBLE FROSTING/PEELING:**
is treated as *proof of efficacy*.
It is actually a **depth surrogate** — a crude one.

⚠ **PATIENT VARIABILITY:**
is ignored.
Phototype IV and II get the same protocol !
One forgives it. The other *does not*.

DIFFERENT CONCEPT. DIFFERENT MECHANISMS. DIFFERENT OUTCOMES.

PEELINGS SOCIETY

What “Metabolic Peel” Actually Means

Redefining the category from chemistry to strategy

CONVENTIONAL PEEL

- Primary objective: controlled exfoliation
- Success = depth of injury achieved
- Visible peeling = proof of action
- Recovery = repair after damage
- Downtime is accepted as necessary cost
- Biology follows chemistry

NOT by the depth or frost you want to achieve

METABOLIC PEEL

- Primary objective: biological regulation
- Success = functional epidermal change
- Visible peeling is NOT required
- Recovery = continuation of guided renewal
- Downtime is minimized by design
- Chemistry serves the biological target

The **CHOICE** of agent is driven by the **biological target.**

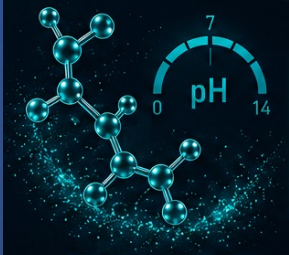
*“Metabolic” describes the **STRATEGY** and **TARGET** — not a new acid category.*

THE KEY TO UNDERSTANDING ACIDS IN COSMETOLOGY

THE HENDERSON–HASSELBALCH EQUATION

INTERNATIONAL PEELINGS SOCIETY

THE FOUNDATION FOR UNDERSTANDING ACID BEHAVIOR IN SKIN



$$pH = pK_a + \log \left(\frac{[A^-]}{[HA]} \right)$$

pH	pK_a	$[A^-]$	[HA]	DIRECT INTERPRETATION
MEASURED ACIDITY	ACID DISSOCIATION CONSTANT	CONJUGATE BASE	ACID	[HA]
Indicates the acidity of a solution.	Intrinsic property of the acid. At pH = pK_a, the acid and its conjugate base are in a 50/50 ratio.			Only the non-ionized (uncharged) form penetrates the lipid barrier. A lower pH → more non-ionized fraction → deeper penetration.

IN REAL SKIN, THIS EQUATION IS **THEORETICAL.** Why?

- ✕ Dynamic skin buffering capacity
- ✕ Non-homogeneous diffusion
- ✕ Interaction with lipids and proteins
- ✕ Variable micro-environment

IN PRACTICE:

“ THE pH YOU APPLY IS NOT THE pH THAT ACTS.”

Dr. Alain Tenenbaum

SCIENCE • EXPERIENCE • RESULTS

TENENBAUM–TIZIANI DYNAMIC ACID INTERACTION MODEL

A functional classification of peeling acids based on protonic profile and biological interaction

ACID CATEGORY	ACID / SUBCATEGORY	PROTONIC PROFILE (pKa)						DOMINANT INTERACTION PROFILE	NUMBER OF REACTIONS
		pKa > 3 rising	pKa = 3	pKa < 3	pKa1	pKa2	pKa3		
ALIPHATIC HYDROXY ACIDS	Alpha Hydroxy (Aliphatic)		Tartaric acid (Wine A)		3.04	4.37	–	Metabolism (cellular bio-regulation)	2 (Diprotic)
ORGANIC ACIDS (POLYHYDROXY)	Citric acid (Citric A)				3.15	4.77	6.40	Progressive metabolic modulation	3 (Triprotic)
	Malic acid (Apples A)				3.40	5.13	–	Metabolism (gentle cellular support)	2 (Diprotic)
	Glycolic acid (Glycol A)				3.83	–	–	Rapid keratolytic-modulatory	1 (Monoprotic)
	Lactic acid (Milk A)				3.86	–	–	Metabolism + hydration support	1 (Monoprotic)
AROMATIC ACIDS	Mandelic acid (Almond A)		Mandelic acid (Almond A)		3.37	–	–	Metabolism (antimicrobial + comedolytic)	1 (Monoprotic)
KETO ACID	Pyruvic acid (Grapes A)			Pyruvic acid (Grapes A)	2.49	–	–	Unstable metabolic profile (highly reactive)	1 (Monoprotic)
DICARBOXYLIC ACIDS	Azelaic acid				4.55	5.59	–	Metabolic modulation (anti-inflammatory)	2 (Diprotic)
SPECIAL FUNCTION ACIDS	Salicylic acid (Beta-Hydroxy)				2.97	–	–	Lipophilic follicular keratolysis	1 (Monoprotic)
	Kojic acid				7.49			Enzymatic melanocyte modulation	1 (Monoprotic)
	Alpha-lipoic acid				4.70			Mitochondrial / penetration-enhancing	1 (Monoprotic-like)
STRONG ACID	TCA			TCA	0.54	–	–	Coagulative injury-driven	1 (Monoprotic)
PHENOLIC COMPOUND	Phenol (Benzene ring)				9.95			High toxicity potential (protein denaturation)	Alcohol-Base (Non-acid)

NOTES

- pKa values indicate the acid's dissociation potential.
- The closer the pKa to skin pH (~5.0), the greater the non-ionized fraction and interaction potential.
- Biological response depends on concentration, vehicle, contact time and skin condition.

DOMINANT INTERACTION PROFILE – LEGEND

- Rapid keratolytic / destructive
- Progressive metabolic modulation
- Metabolic / bio-regulatory
- Special function (enzymatic / antioxidant / penetration)
- Follicular / lipophilic
- Injury-driven / coagulative
- Toxic / protein denaturation

KEY TAKEAWAY

Acids are not intrechangeable.
Their behavior is defined not by concentration alone, but by their protonic profile and dominant biological interaction with the skin.

chemicalpeelling.com

THE 5 PILLARS OF THE

TENENBAUM–TIZIANI DYNAMIC ACID INTERACTION MODEL

A functional classification of peeling acids based on protonic profile and biological interaction

1. PENETRATION EASE

(Passive Diffusion Potential)

Depends on non-ionized fraction, molecular size, lipophilicity and vehicle.

RATING	ACIDS (examples)
VERY HIGH	TCA
HIGH	Glycolic acid Lactic acid Salicylic acid Pyruvic acid
MODERATE	Citric acid Malic acid
LOW	Kojic acid
VERY LOW	Azelaic acid Alpha-lipoic acid

The greater the non-ionized fraction at skin pH (~5.0), the easier the acid penetrates.

2. KERATOLYTIC POTENTIAL

(Stratum Corneum Disruption)

Depends on acidity, penetration and protein interaction / denaturation.

RATING	ACIDS (examples)
VERY HIGH	TCA
HIGH	Glycolic acid Lactic acid
MODERATE–HIGH	Salicylic acid Pyruvic acid
MODERATE	Citric acid Malic acid
LOW–MODERATE	Azelaic acid
VERY LOW	Kojic acid Alpha-lipoic acid

Keratolysis results from both chemical activity and the ability to reach and act within the corneocyte.

3. MOISTURIZING / HUMECTANT POTENTIAL

Ability to retain water in the stratum corneum.

RATING	ACIDS (examples)
VERY HIGH	Lactic acid
HIGH	Glycolic acid
MODERATE–HIGH	Malic acid
MODERATE	Citric acid
LOW–MODERATE	Pyruvic acid
LOW	Salicylic acid Azelaic acid
VERY LOW	Kojic acid Alpha-lipoic acid
NONE	TCA

Humectant potential improves comfort, reduces downtime and supports barrier quality when properly balanced.

4. METABOLIC (BIOREGULATORY) POTENTIAL

Ability to modulate cellular processes without triggering injury.

RATING	ACIDS (examples)
VERY HIGH	Alpha-lipoic acid
HIGH	Azelaic acid Kojic acid
MODERATE	Citric acid Malic acid Lactic acid
LOW–MODERATE	Glycolic acid Salicylic acid
LOW	Pyruvic acid
NONE	TCA

Bioregulatory acid behavior optimizes results while respecting cellular homeostasis.

5. BUFFERING RECOMMENDATIONS

For better control, tolerance and predictability.

STRATEGY	ACIDS (examples)
YES	Glycolic acid Lactic acid Salicylic acid Pyruvic acid
YES (often)	Citric acid Malic acid
NO (usually)	Azelaic acid Kojic acid
NO	Alpha-lipoic acid
NO (rarely)	TCA

Buffering modulates pH delivery, reduces irritation risk and increases treatment reproducibility.

NOTES

(1) pKa values indicate the acid's dissociation potential.
(2) Passive diffusion only. Does not account for formulation, vehicle, or residence time.
(3) Triprotic acid: pKa1 = 3.15 / pKa2 = 4.77 / pKa3 = 6.40
(4) Alpha-lipoic acid acts as a penetration catalyst: it fluidizes lipid domains and enhances delivery of co-applied actives in a controlled manner.
(5) Suggested working pH range for topical formulations (general guidance).

DOMINANT INTERACTION PROFILE – LEGEND

- Rapid keratolytic / destructive
- Progressive metabolic modulation
- Metabolic / bio-regulatory
- Special function (enzymatic / antioxidant / penetration)
- Follicular / lipophilic
- Injury-driven / coagulative
- Toxic / protein denaturation

KEY MESSAGE

The Tenenbaum–Tiziani Model classifies acids by how they behave in the skin — not by concentration.

Different acids. Different functions.
Different strategies. Better outcomes.

TENENBAUM–TIZIANI
SCIENCE • EXPERIENCE • RESULTS

chemicalpeeling.com

THE SKIN DOESN'T RESPOND TO ACIDS
IT RESPONDS TO SIGNALS

Understanding diffusion, modulation, and biological interaction

DIFFUSION

Acid state & pKa

At physiological skin pH (~5.5), most acids exist in equilibrium between ionized and non-ionized forms.

Only the non-ionized (uncharged) form penetrates the lipid barrier.

A lower pH → more non-ionized fraction → deeper penetration.

Ionization depends on the relationship between pKa and pH.

MODULATION

Vehicle controls resistance time & spread
Skin preparation modifies barrier property

The vehicle determines how long the active form stays at the stratum corneum interface.

Aqueous vehicles = rapid spread, difficult to control.
(Ex Salicylic Acid in ethanol/glycerol and TCA liq/gel)

Gel or cream vehicles = slower diffusion, better clinical control.

Skin preparation (degreasing) dramatically alters penetration kinetics.

BIOLOGICAL EFFECT

Target & modulation

Here is where metabolic peels diverge from standard logic: the metabolic approach targets the **VIABLE** epidermis — the living cells.

We're not trying to destroy and replace.
We're trying to regulate and support.

You either regulate cells — or destroy tissue !

METABOLIC ✓ | CONVENTIONAL ✗

Dr. Alain Tenenbaum
SCIENCE • EXPERIENCE • RESULTS

Tenenbaum–Tiziani Dynamic Acid Interaction Model
A functional classification of peeling acids based on protonic profile and biological interaction

chemicalpeeling.com

Two Different Targets — Two Different Clinical Logics

INTERNATIONAL PEELINGS SOCIETY

CONVENTIONAL TARGET

Stratum corneum
Physical removal → keratolysis, desquamation

Viable epidermis
Chemical injury → inflammatory trigger

Dermal layer
Coagulative injury → secondary inflammation

Injury-driven, destructive approach

VS

METABOLIC TARGET

Keratinocyte function
- Mitochondrial function support (cell viability)
- ATP-dependent cellular activity
- Controlled differentiation signaling

Melanocyte regulation
Tyrosinase inhibition (kojic, azelaic), transfer blockade

Basal layer renewal
- Support of normal cell cycle — not forced replacement

Regulation-driven, restorative approach

Dr. Alain Tenenbaum
SCIENCE • EXPERIENCE • RESULTS

Tenenbaum-Tiziani Dynamic Acid Interaction Model
A functional classification of peeling acids based on protonic profile and biological interaction

THE TWO ESSENTIAL BARRIERS IN SKIN HOMEOSTASIS

Different structures. Different functions. Both must be respected.

INTERNATIONAL PEELINGS SOCIETY

1. EPIDERMAL LIPID BARRIER (STRATUM CORNEUM)

The gatekeeper: prevents excess entry

STRUCTURE
Corneocytes embedded in a lipid matrix (ceramides, cholesterol, fatty acids)

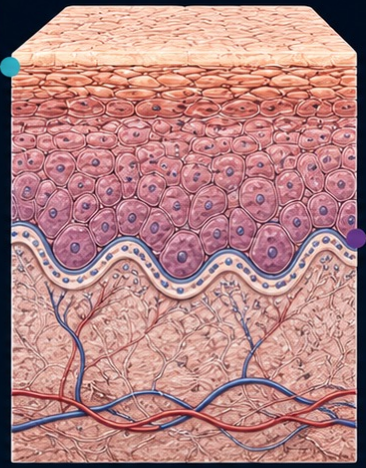
LOCATION
Top of the epidermis

PRIMARY FUNCTION
Limits transepidermal water loss and blocks uncontrolled penetration of exogenous and endogenous agents

WHAT IT CONTROLS
Passive diffusion of molecules

WHAT DISRUPTS IT[1]
Surfactants, high pH, solvents, over-exfoliation, inflammation

CLINICAL IMPACT IF DAMAGED
Dryness, irritation, increased sensitivity, inflammation, barrier dysfunction



2. DERMAL–EPIDERMAL JUNCTION BARRIER (BASEMENT MEMBRANE COMPLEX)

The anchor: maintains integrity and communication

STRUCTURE[1]
Basement membrane + hemidesmosomes + anchoring fibrils + lamina densa

LOCATION
At the base of the epidermis, between epidermis and dermis

PRIMARY FUNCTION
Anchors epidermis to dermis and regulates cell communication, migration, and structural integrity

WHAT IT CONTROLS
Cell signaling, melanocyte behavior, immune surveillance, repair processes

WHAT DISRUPTS IT[1]
Over-aggressive peels, deep injury, chronic inflammation, enzymes (MMPs), UV damage

CLINICAL IMPACT IF DAMAGED
PIH, scarring, delayed healing, loss of structural support, chronic inflammation

KEY DIFFERENCES

EPIDERMAL LIPID BARRIER	ASPECT	DERMAL–EPIDERMAL JUNCTION BARRIER
Prevents entry	Role	Maintains integrity & communication
Stratum corneum (lipid matrix)	Location	Basement membrane (DE junction)
Passive diffusion (molecules)	Control	Cell behavior & structural stability
Surface disruption	Common injury	Deep injury / over-aggression
Dryness, irritation, sensitivity	Clinical consequence	PIH, scarring, chronic inflammation

CLINICAL TAKEAWAY

Respect the lipid barrier to control penetration.

Respect the DEJ barrier to prevent long-term damage.

Smart protocols protect both.

PROTOCOL IMPLICATION

Effective peeling is not about destroying barriers, but about modulating them intelligently for safe, predictable, and sustainable results.

THE GOAL IS NOT PENETRATION. THE GOAL IS BIOLOGICAL OPTIMIZATION.

Tenenbaum–Tiziani Dynamic Acid Interaction Model

EPIDERMAL LIPID BARRIER (STRATUM CORNEUM)

The gatekeeper: prevents excess entry and controls water balance

COMPOSITION

CERAMIDES
~50%
Main structural lipids, create ordered lamellae

CHOLESTEROL
~25%
Regulates fluidity and barrier stability

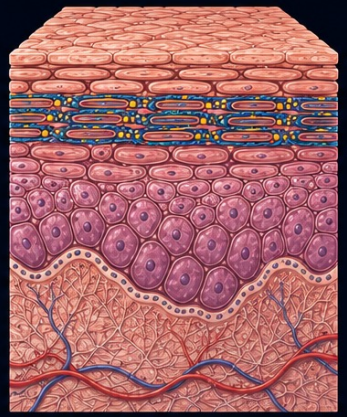
FREE FATTY ACIDS
~15%
Fills gaps between ceramides, maintains acid mantle (pH)

LIPID MATRIX ORGANIZATION

"Brick and mortar" model

Corneocytes = bricks

Lipids = mortar



PRIMARY FUNCTIONS

BARRIER TO WATER LOSS
Limits transepidermal water loss (TEWL) and maintains hydration

BARRIER TO EXOGENOUS AGENTS
Controls penetration of irritants, allergens, microbes and chemicals

MICROBIOME SUPPORT
Maintains acidic pH (4.5–5.5) and supports beneficial microbiome balance

SENSORY PROTECTION
Reduces sensitivity by preventing irritation and neurogenic inflammation

RESILIENCE & RECOVERY
Ensures rapid recovery after mild stress and daily aggressions

WHEN THE LIPID BARRIER IS INJURED

Functional consequences – usually superficial, but clinically significant

- Increase in transepidermal water loss (TEWL) → Sensitive skin / hypersensitivity
- Dryness, tightness, rough texture → Eczema-like reactions / dermatitis
- Erythema, irritation, stinging, burning → Acne flares (barrier-impaired skin)
- Enhanced penetration of irritants and allergens → Microbiome dysbiosis
- Reactive inflammation & barrier vulnerability → Delayed healing & prolonged recovery
- → Chronic sensitivity over time

CLINICAL CONSEQUENCES (examples)

DRYNESS DEHYDRATION · IRRITATION ERYTHEMA · BURNING STINGING · REACTIVE INFLAMMATION · BARRIER DYSFUNCTION · MICROBIOME INSTABILITY

CLINICAL TAKEAWAY

The lipid barrier controls what enters and what escapes. Protect it to preserve comfort, tolerance and results.

Respect it: use the right pH, appropriate vehicles, and gentle exfoliation strategies.

Injure it: expect sensitivity, inflammation and poor outcomes.

DIFFERENT BARRIER. DIFFERENT RISK. DIFFERENT CONSEQUENCES. Superficial barrier injury ≠ deep structural damage.

Dr. Alain Tenenbaum
SCIENCE • EXPERIENCE • RESULTS

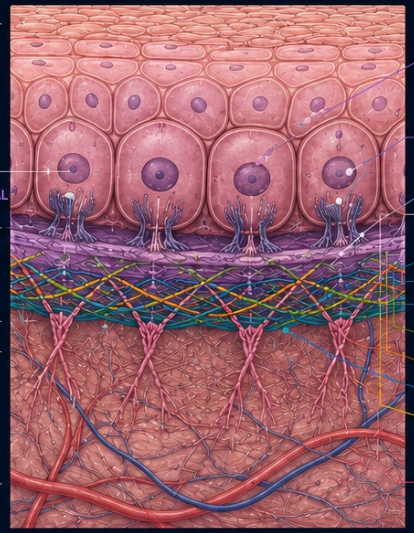
Tenenbaum–Tiziani Dynamic Acid Interaction Model
A functional classification of peeling acids based on protonic profile and biological interaction

chemicalpeeling.com

DERMAL–EPIDERMAL JUNCTION BARRIER

(DEJ / BASEMENT MEMBRANE COMPLEX)

The foundation: maintains integrity, communication and long-term stability



KEY COMPONENTS

- **Hemidesmosomes** — Anchor basal cells to the basement membrane
- **Lamina lucida** — Interface zone
- **Lamina densa** — Protein-rich layer (protective scaffold)
- **Anchoring fibrils (Type VII collagen)** — Secure membrane to dermis
- **Collagen IV**
- **Laminin**
- **Nidogen (Entactin)**
- **Integrins** — Cell–matrix communication
- **Junction proteins (occludin, claudins, cadherins)** — Regulate adhesion and signaling

PRIMARY FUNCTIONS

MECHANICAL ANCHORING
Maintains attachment of epidermis to dermis

BIOLOGICAL COMMUNICATION
Regulates signaling between keratinocytes, melanocytes and fibroblasts

MELANOCYTE STABILITY
Essential for pigment transfer and phototype stability

TISSUE REPAIR ORCHESTRATION
Controls cell migration, proliferation and matrix remodeling

BARRIER OF INTEGRITY
Prevents aberrant penetration and maintains structural organization

WHEN THE DEJ IS INJURED

Deeper consequences beyond visible irritation

Disruption of anchoring structures	→ Epidermal instability
Damage to laminin / collagen IV network	→ Loss of adhesion
Altered signaling	→ Melanocyte dysregulation
Chronic inflammation at the interface	→ Persistent cytokine activation
Abnormal repair activation	→ Fibrosis / scarring
Matrix remodeling dysregulation	→ Structural aging
Vascular and immune activation	→ PIH and erythema

CLINICAL CONSEQUENCES OF DEJ INJURY

- PIH (POST-INFLAMMATORY HYPERPIGMENTATION)
- CHRONIC INFLAMMATION
- SCARRING & FIBROSIS
- ACCELERATED AGING
- PIGMENT HEAULITY POOR RECOVERY

CLINICAL TAKEAWAY

The DEJ is not a diffusion barrier.
It is an integrity barrier.

Preserve it, and the skin recovers, rejuvenates and stays stable.
Damage it, and complications become difficult to manage.

Dr. Alain Tenenbaum
SCIENCE • EXPERIENCE • RESULTS

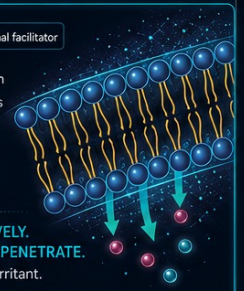
Tenenbaum–Tiziani Dynamic Acid Interaction Model
A functional classification of peeling acids based on protonic profile and biological interaction

chemicalpeeling.com

ACID PENETRATION — BEYOND CONCENTRATION

Penetration depends on dynamic buffering and biological context — not on labeled concentration.

ACID	pKa	ESTIMATED NON-IONIZED FRACTION UNDER ACIDIC CONDITIONS	KEY PENETRATION CHARACTERISTICS (BIOLOGICAL CONTEXT MATTERS)
Glycolic acid	3.8	~50%	High diffusivity due to small molecular size Penetration varies with pH gradient & vehicle
Lactic acid	3.9	~50%	More hydrophilic Behavior highly context-dependent
Salicylic acid	3.0	~17%	Lipophilic. Preferential follicular penetration
TCA	0.54	~100%	Barrier injury mechanism Controlled depth — dominated by protein coagulation
Alpha-lipoic acid (ALA) NEW	4.7	PENETRATION FACILITATOR / DELIVERY MODULATOR ★ Functional facilitator ✓ **Modulates** membrane fluidity and lipid organization ✓ Enhances controlled delivery of co-applied actives ✓ Optimizes the skin microenvironment for deeper functional interaction ✓ Supports deeper action without aggressive keratolysis or barrier damage ALA DOES NOT PENETRATE AGGRESSIVELY. IT CHANGES HOW OTHER MOLECULES PENETRATE. It is a smart penetration enhancer, not an irritant.	
Kojic acid	7.9	Barely at all	Acts primarily via enzymatic modulation



THE pH YOU APPLY IS NOT THE pH THAT ACTS.
THE SKIN CONTINUOUSLY MODIFIES IT.

SOME MOLECULES ACT AS ACIDS. OTHERS ACT AS BIOLOGICAL PENETRATION MODULATORS.

Dr. Alain Tenenbaum
SCIENCE • EXPERIENCE • RESULTS

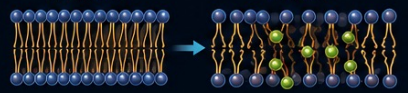
Tenenbaum—Tiziani Dynamic Acid Interaction Model
A functional classification of peeling acids based on protonic profile and biological interaction

MECHANISM OF ACTION OF LIPOIC ACID

A PENETRATION ENHANCER AND BIOLOGICAL MODULATOR

1. MODULATES LIPID MEMBRANE FLUIDITY

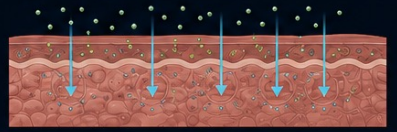
ALA inserts into lipid bilayers and increases fluidity by disrupting tight packing of lipid chains.



RESULT
Increased fluidity = easier movement of molecules across the membrane

2. ENHANCES DELIVERY OF CO-APPLIED ACTIVES

By improving diffusion pathways, ALA facilitates deeper and more uniform penetration of other active molecules.



RESULT
Better delivery, deeper action, more predictable clinical results

3. OPTIMIZES THE SKIN MICROENVIRONMENT

ALA has antioxidant and chelating properties.

- **Antioxidant** — Reduces oxidative stress
- **Chelating** — Binds metal ions that promote inflammation
- **Anti-inflammatory** — **Modulates** redox signaling pathways

RESULT
Healthier tissue response and improved tolerance

4. SUPPORTS FUNCTIONAL, NOT DESTRUCTIVE, INTERACTION

ALA does not cause protein coagulation or barrier injury.

It is not an exfoliating acid.
It is a biological facilitator.

→ Goal: controlled penetration with preserved barrier and optimal outcomes.

ALA DOES NOT WORK BY FORCE. IT WORKS BY INTELLIGENCE.
It changes the environment so that the right molecules can reach the right targets.

chemicalpeeling.com

CHEMICAL PENETRATION POTENTIAL

Why Acids Behave Differently Before Skin Buffering

INTERNATIONAL PEELINGS SOCIETY

HIGHER NON-IONIZED FRACTION → GREATER PASSIVE DIFFUSION POTENTIAL

Chemical potential is only the starting point. ***Real skin behavior remains dynamic.***

Tenenbaum–Tiziani Dynamic Acid Interaction Model

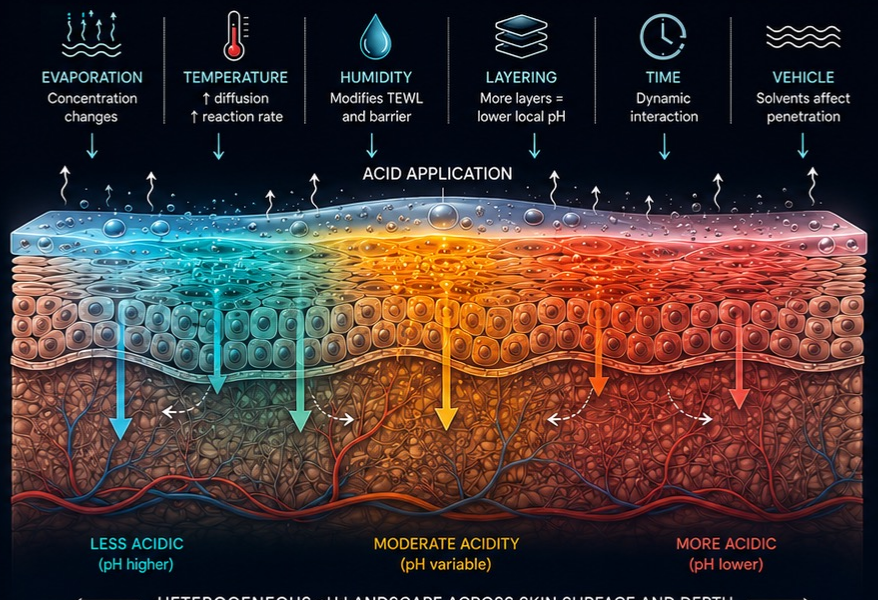
DYNAMIC SKIN BUFFERING MODEL

Why the "Applied pH" Is Never the Real Biological pH

INTERNATIONAL PEELINGS SOCIETY

FACTORS MODIFYING REAL SKIN pH INTERACTION

- **Vehicle evaporation** — Concentration and pH change over time
- **Temperature** — Influences diffusion, kinetics and evaporation
- **Ambient humidity** — Low humidity ↑ TEWL and alters barrier behavior
- **TEWL status** — High TEWL = faster water and acid flux
- **Barrier integrity** — Altered barrier = modified buffering capacity
- **Sebum composition** — Lipids interact with acids and modify penetration
- **Number of layers applied** — More layers = greater acid load and lower local pH potential
- **Contact time** — The longer the contact, the greater the interaction
- **Skin hydration** — Well-hydrated skin buffers differently than dry skin



CLINICAL CONSEQUENCES

Same acid ≠ same behavior
Biological response depends on dynamic conditions, not the bottle.

Same pH ≠ same penetration
Real penetration depends on skin state and microenvironment.

Layering changes biological response
Each additional layer increases acid load and modifies pH locally.

Skin buffering continuously modifies diffusion
The skin is a dynamic, intelligent biological system.

Real skin is not a laboratory beaker
It actively transforms and regulates the chemical environment.

THE pH WRITTEN ON THE BOTTLE IS ONLY THE STARTING CONDITION.
THE SKIN IMMEDIATELY TRANSFORMS THE CHEMICAL ENVIRONMENT.

6 CLINICAL SITUATIONS WHERE METABOLIC-LOGIC PEELS OUTPERFORM CONVENTIONAL APPROACHES

Clinical logic changes outcomes.

INTERNATIONAL PEELINGS SOCIETY

1. HYPERPIGMENTATION & MELASMA

- Melanocyte regulation
- Less inflammatory signaling
- Lower PIH risk in darker phototypes

2. FATIGUED OR STRESSED SKIN

- Mitochondrial support
- Improved cellular energy
- Luminosity without aggressive exfoliation

3. POST-INFLAMMATORY RECOVERY

- Barrier already compromised
- Supports recovery phases
- Lower inflammatory burden

4. LONG-TERM MAINTENANCE

- Repeatable protocols
- Less cumulative barrier injury
- Progressive improvement over time

5. ACNEIC SKIN WITH PIGMENT RISK

- Sebaceous normalization
- Pigment-conscious strategy
- Reduced post-inflammatory risk

6. SENSITIVE / ROSACEA-PRONE SKIN

- Barrier-supportive approach
- Reduced inflammatory signaling
- Compatible with fragile skin states

THE GOAL IS NOT STRONGER INJURY. THE GOAL IS SMARTER BIOLOGICAL REGULATION.

Where Most Complications Occur — and Why Protocol Intelligence Matters

Complications are not random. They follow a predictable biological logic.

WHERE MOST COMPLICATIONS ACTUALLY OCCUR

Not the acid. The barrier. And the response that follows.

PRIMARY SITE OF INJURY	TYPICAL COMPLICATIONS	WHY IT HAPPENS
Epidermal Lipid Barrier (Stratum Corneum)	• Dryness, burning, irritation • Erythema, stinging, tightness • Reactive inflammation • Barrier dysfunction, sensitivity • Microbiome imbalance	Over-aggression, high pH, inadequate hydration, repeated stripping, lack of barrier support.
Dermal–Epidermal Junction (DEJ) Basement Membrane	• PIH (post-inflammatory hyperpigmentation) • Chronic inflammation • Scarring, fibrosis • Delayed healing, persistent erythema • Pigment instability, aging acceleration	Deep injury, uncontrolled inflammation, disruption of the DEJ, melanocyte instability, impaired repair.
Contextual Factors & Patient Variables	• Exacerbations in melasma/acne/rosacea • Prolonged downtime • Poor tolerance, recurrence	Ignoring phototype, history, barrier status, disease activity, or lifestyle factors.

PROTOCOL INTELLIGENCE: SEQUENCING OVER AGGRESSION

Control the interaction. Guide the response. Protect the outcome.

1. PREPARE & PROTECT

- Assess barrier status and disease activity
- Optimize skin environment (pH, hydration, lipids)
- Choose the right acid(s) for the target
- Set realistic depth objectives

2. TREAT & MODULATE

- Use the minimum effective concentration
- Control pH and exposure time
- Layer strategically (if needed)
- Observe the skin's real-time response

3. REPAIR & STABILIZE

- Reinforce the lipid barrier immediately
- Control inflammation proactively
- Support repair and DEJ integrity
- Educate and follow-up consistently

The best protocols don't push the skin harder. They work with its biology.

KEY MESSAGE

Complications arise when we ignore one of three truths:

1. The wrong barrier is injured.
2. The skin's response is underestimated.
3. The protocol is not adapted to the patient.

Intelligence is not about stronger acids.
It is about better decisions.

AVOID FROSTING MINDSET: Whitening or "frosting" is not a goal. It is a warning sign of loss of control and risk of barrier + DEJ injury, especially in darker phototypes.

Higher risk of PIH, inflammation and barrier damage in darker phototypes and compromised skins.

FUSION SLIDE FOR 24–25

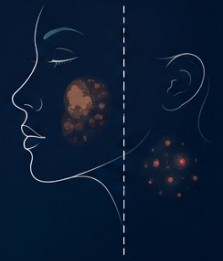
Clinical Case Logic: Melasma & Acne

Two conditions. Different targets. Different protocols. Different risks.

MELASMA IN PHOTOTYPE IV

WHAT WE MUST DO	WHAT HAPPENS WHEN WE DON'T
✓ Stabilize before stimulating	✗ Post-inflammatory hyperpigmentation (PIH)
✓ Protect the barrier and the DEJ	✗ Melanocyte hyperactivation
✓ Use low-inflammatory, controlled protocols	✗ Chronic inflammation
✓ Focus on modulation, not destruction	✗ Patchy, resistant melasma
✓ Prevent heat, inflammation and rebound	✗ Longer downtime and patient frustration

CORE PRINCIPLE: In melasma, aggression fuels the disease.
Control, modulation and barrier protection create stability and results.



ACNE / POST-INFLAMMATORY PIGMENTATION

WHAT WE MUST DO	WHAT HAPPENS WHEN WE DON'T
✓ Normalize the follicular environment	✗ Post-inflammatory hyperpigmentation (PIH)
✓ Reduce inflammation and sebum signaling	✗ Prolonged inflammation
✓ Use antibacterial + anti-inflammatory strategy	✗ Sebaceous gland overstimulation
✓ Controlled exfoliation, not deep injury	✗ Acne flare or worsening
✓ Support barrier repair and sebum balance	✗ Scarring risk and uneven texture

CORE PRINCIPLE: In acne, the goal is not surface stripping.
It is follicular normalization and inflammation control.

PROTECT THE BARRIERS.
Lipid Barrier + DEJ Integrity

CHOOSE THE RIGHT TARGET.
Melanocyte modulation vs. Follicular normalization

ADAPT THE PROTOCOL.
Patient, phototype, history, and disease activity

THE GOAL IS NOT AGGRESSION. THE GOAL IS PREDICTABLE, SAFE, AND SUSTAINABLE RESULTS.

PHOTOTYPE-SPECIFIC RISK: THE MOST IGNORED VARIABLE IN PEELING

INTERNATIONAL PEELINGS SOCIETY

> *PIH is not an accident. It is a completely predictable biological response.* When you injure skin in a patient with high melanocyte reactivity, you trigger IL-1, TNF-alpha, prostaglandin E2, and these cytokines directly stimulate tyrosinase. The melanocyte deposits pigment as a photoprotective response. You treated the skin aggressively. *The skin defended itself with pigment.*

FITZPATRICK TYPE I–II

LOW PIH RISK

- Standard and metabolic both suitable
- Barrier recovery fast
- Lower melanocyte reactivity

FITZPATRICK TYPE III

MODERATE PIH RISK

- Careful patient selection
- Avoid inflammatory triggers
- Metabolic approach preferred for maintenance

FITZPATRICK TYPE IV

HIGH PIH RISK

- Standard peels require careful depth control
- Metabolic logic strongly preferred
- Pre-conditioning with tyrosinase inhibitors essential

FITZPATRICK TYPE V–VI

VERY HIGH PIH RISK

- Conventional exfoliative peeling HIGH RISK
- Metabolic regulatory approach strongly indicated
- No injury-based mechanism without pre/post protocol

PIH RISK IS PREDICTABLE, NOT RANDOM. ANY PRACTTIONER WHO HAS HAD PIH COMPLICATIONS "THEY DIDN'T EXPECT" WAS NOT APPLYING PHOTOTYPE-SPECIFIC PROTOCOL LOGIC.

METABOLIC vs STANDARD PEELS: SCIENTIFIC COMPARISON

PARAMETER	STANDARD PEELS	METABOLIC PEELS
Primary mechanism	Controlled injury → repair response	Biological regulation → functional improvement
Barrier status	Disrupted (design-required)	Preserved (design-required)
Melanocyte risk	High — inflammatory trigger	Low — regulatory target
Phototype range	I–III most suitable	I–VI with appropriate selection
Downtime	Days to weeks (depth-dependent)	Hours to 24h typical
Repeatability	Limited — cumulative damage risk	High — maintenance-ready
Intraoperative endpoint	Frosting level	None — outcome at 4–6 weeks
Combination potential	Limited (barrier compromised)	High — sequences well with other modalities
Complication profile	PIH, scarring, barrier dysfunction	Minimal if correctly indicated

SCIENCE GUIDES CHOICE. BIOLOGY DEFINES OUTCOME.

CASE 1 — MELASMA / POST-INFLAMMATORY HYPERPIGMENTATION

Why modulation before aggression makes the difference

Photos unretouched – Same lighting – Phototype IV

CLINICAL PROFILE

- Phototype IV
- Chronic melasma with inflammatory component
- History of irritation with aggressive peels

STRATEGIC APPROACH

- **Step 1:** Inflammation control & barrier support
- **Step 2:** Metabolic modulation (non-injurious acids)
- **Step 3:** Very progressive exfoliation
- Strict sun protection and maintenance protocol

ACIDS & LOGIC

Priority to metabolic modulators (lactic, mandelic, kojic)
Avoid deep or rapid keratolysis
Goal: Restore biological stability before exfoliating

KEY LESSON

Pigment control is impossible without first controlling inflammation and restoring skin homeostasis.

CASE 2 — ACNE / INFLAMMATORY SKIN

Targeted follicular approach with intelligent sequencing

Photos unretouched – Same lighting – Phototype III

CLINICAL PROFILE

- Inflammatory acne, oily skin
- Follicular hyperkeratinization
- Sensitive / reactive tendency

STRATEGIC APPROACH

- Salicylic acid for follicular penetration
- Alternating with lactic acid to reduce inflammation
- Low irritation – consistent frequency
- Sebum control + barrier support

ACIDS & LOGIC

Lipophilic penetration + anti-inflammatory modulation
Avoid over-exfoliation and barrier disruption
Goal: Normalize the follicular environment

KEY LESSON

Acne improves when we normalize the follicle, not when we aggressively strip the surface.

CASE 3 — AGING / PHOTOAGING

Controlled injury still has a place – when used with precision

Photos unretouched – Same lighting – Phototype II

CLINICAL PROFILE

- Photoaging, fine lines, rough texture
- Good tolerance, no active inflammation
- Desire for visible rejuvenation

STRATEGIC APPROACH

- Preparation with metabolic modulators
- Controlled injury with TCA (medium depth)
- Adapted intervals and post-care
- Maintenance to prolong results

ACIDS & LOGIC

Controlled coagulative injury to stimulate remodeling
Depth and downtime adapted to indication
Goal: Collagen remodeling with safety and predictability

KEY LESSON

In aging skin, controlled injury is valuable when the protocol is precise, prepared, and scientifically sequenced.

Tenenbaum-Tiziani Dynamic Acid Interaction Model

INTELLECTUAL HONESTY: WHERE STANDARD PEELS STILL LEAD

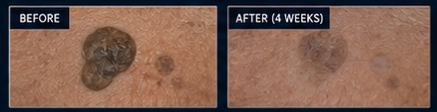
A sophisticated clinician knows which tool to use when.

Metabolic peels are not a replacement for standard chemical peeling. They are a strategic complement.

WHAT TO KNOW ABOUT TCA RESURFACING vs METABOLIC:

- TCA 20–35% remains the workhorse for photodamage, mild acne scarring, and textural improvement
- The collagen stimulation from controlled papillary dermal injury has 30+ years of evidence
- **Metabolic peels do NOT provide this — their mechanism does not reach the dermis under normal protocols**

INTERNATIONAL PEELINGS SOCIETY

		✓ TEXTURAL RESURFACING	When visible improvement in skin texture is the primary goal — acne scarring, enlarged pores, photoaging — depth-controlled TCA or medium-depth peels remain the most evidence-backed option.
		✓ ACTINIC KERATOSIS	Field treatment for AK with 5-FU-based or TCA protocols remains standard of care. Metabolic logic does not address this indication.
		✓ SEBORRHEIC, KERATOTIC SKIN	True keratolysis — the mechanical disruption of a thickened, adherent stratum corneum — often requires aggressive exfoliative chemistry. Salicylic logic applies.
		✓ IMMEDIATE VISIBLE RESULT	Some clinical contexts — pre-event, single-session treatment — require a visible endpoint the clinician can confirm intraoperatively. Frosting gives that. Metabolic peels do not.

THE BEST CLINICIANS ARE NOT LOYAL TO A MODALITY —
THEY ARE LOYAL **TO THE PATIENT AND TO THE SCIENCE.**

Tenenbaum-Tiziani Dynamic Acid Interaction Model

CLINICAL CASE LOGIC: MELASMA & ACNE

Different conditions. Different targets. Different protocols. Different risks.

MELASMA IN PHOTOTYPE IV

A disease of dysregulation and inflammation.
Our role is to stabilize, control and protect.

WHAT WE MUST DO

- Stabilize before stimulating
- Protect the barrier and the DEJ
- Use low-inflammatory, controlled protocols
- Focus on modulation, not destruction
- Prevent heat, inflammation and rebound

WHAT HAPPENS WHEN WE DON'T

- Post-inflammatory hyperpigmentation (PIH)
- Melanocyte hyperactivation
- Chronic inflammation
- Patchy, resistant melasma
- Longer downtime and patient frustration

CORE PRINCIPLE

In melasma, aggression fuels the disease.
Control, modulation and barrier protection create stability and results.

ACNE / POST-INFLAMMATORY PIGMENTATION

A disease of inflammation, bacterial triggers and follicular dysfunction.
Our role is to normalize and repair.

WHAT WE MUST DO

- Normalize the follicular environment
- Reduce inflammation and sebum signaling
- Use antibacterial + anti-inflammatory strategy
- Controlled exfoliation, not deep injury
- Support barrier repair and sebum balance

WHAT HAPPENS WHEN WE DON'T

- Post-inflammatory hyperpigmentation (PIH)
- Prolonged inflammation
- Sebaceous gland overstimulation
- Acne flare or worsening
- Scarring risk and uneven texture

CORE PRINCIPLE

In acne, the goal is not surface stripping.
It is follicular normalization and inflammation control.

STRATEGIC DIFFERENCES AT A GLANCE

PRIMARY DRIVER	Melanocyte dysregulation + chronic inflammation	⟷	Follicular inflammation + bacterial triggers
TARGET	Melanocytes + vascular + barrier + DEJ	⟷	Follicle + sebaceous units + inflammation
PROTOCOL APPROACH	Low inflammation, barrier-safe, modulation-first	⟷	Antibacterial + anti-inflammatory + controlled exfoliation
SUCCESS MARKER	Stable, even tone without rebound	⟷	Clear, even skin with reduced inflammation
RISK TO AVOID	Over-stimulation → PIH, patchiness, chronicity	⟷	Over-aggression → flare, PIH, scarring

KEY TAKEAWAY

Different diseases.
Different targets.
Different protocols.
Same goal: safe, predictable, sustainable results.

PROTECT THE BARRIERS

Always respect the Lipid Barrier & the DEJ.
They are the foundation of every result.

ADAPT THE PROTOCOL

To the patient.
To the phototype.
To the history.
To the disease activity.

Intelligence is not about stronger acids.
It is about the right decision for the right biology.

Tenenbaum–Tiziani Dynamic Acid Interaction Model

PROTOCOL INTELLIGENCE
MATTERS MORE THAN ACID STRENGTH

From theory to safe, predictable, and effective clinical outcomes

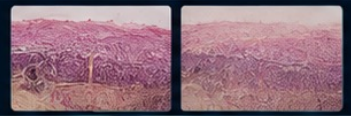
INTERNATIONAL PEELINGS SOCIETY

THE 5 PILLARS OF PROTOCOL INTELLIGENCE

1. ANALYZE
Understand the formulation
pKa, vehicle, concentration, mechanism

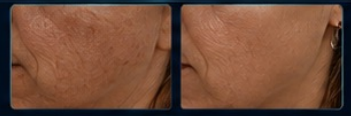
2. TARGET
Choose the right target
Indication, depth, tissue, clinical goal

3. SEQUENCE
Build the logical sequence
Order, intervals, combinations



4. PROTECT
Anticipate and prevent complications
Pre / during / post care framework

5. ADAPT
Adjust in real time
Skin response, environment, patient factors



THE RESULT OF INTELLIGENT PROTOCOLS

IT'S NOT ABOUT USING THE STRONGEST ACID.
IT'S ABOUT USING THE RIGHT ACID, IN THE RIGHT WAY, AT THE RIGHT TIME.

Tenenbaum–Tiziani Dynamic Acid Interaction Model

Advanced Training • Evidence • Logic • Mastery

CONCLUSION

5 Take-Home Messages

INTERNATIONAL PEELINGS SOCIETY

01

The mechanism defines the outcome

A peel should be selected by its biological target — not its acid name, depth, or frosting level.
Mechanism-first thinking is the foundation of safe, effective peeling.

02

Metabolic logic addresses a different biological layer

When the objective is pigment regulation, biological renewal support, or barrier-safe maintenance — metabolic formulations outperform injury-based approaches by design.

03

Most complications occur in the unsupervised window

Injury-based peeling creates a 24–72h window of vulnerability the practitioner cannot supervise.
Minimizing that window is the core safety advantage of metabolic protocols.

04

Phototype determines the risk equation

PIH is not bad luck. It is a predictable response in high-melanocyte-reactivity patients when the inflammatory mechanism is not controlled. Pre-conditioning is non-negotiable in phototype III+.

05

Protocol intelligence over aggression

The most sophisticated protocols are not the most aggressive.
They are the most intelligently sequenced — preparation, targeted treatment, and systematic post-care.

INTELLIGENT PROTOCOLS. PREDICTABLE OUTCOMES. TRUSTED RESULTS.

Stop treating the consequence.
Control the process.

Chemical peeling is not a technique. It is a strategy.
When you understand the biology — the chemistry follows
naturally, the complications diminish,
and the results speak for themselves.

Dr.
ALAIN TENENBAUM
MD; PhD; DSc
FACIAL PLASTIC SURGEON
CHEMICAL PEEL SPECIALIST
SWITZERLAND

Mr.
MAURO TIZIANI
MOLECULAR BIOLOGIST
SWITZERLAND

INTERNATIONAL PEELINGS SOCIETY
(IPSC)

THE SCIENTIFIC HOME OF
CHEMICAL PEEL EXPERTS.

CHEMICALPEELING.COM
Your global resource for science, education and professional growth.

JOIN THE INTERNATIONAL PEELINGS SOCIETY (IPSC)
Become a member.
Be part of the science.
aestheticmedicine.directory/join-directory

INTERNATIONAL-PEELINGS-SOCIETY.COM
The future home of protocols, research and global collaboration.

Private Workshops
Clinical Training
International Lectures

SCIENCE. STRATEGY. SAFETY. RESULTS.

DRPEELING@BLUEWIN.CH