

ARROWBIOME KNOWLEDGE HUB · ACNE SCIENCE SERIES — CHAPTER 3

Your Skin Has an Ecosystem

And acne is what happens
when it **tips**

A premium guide to the living community on your skin — the residents that keep it in balance, what happens when that balance shifts, and why the science of acne has moved from *kill the bacteria* to *restore the balance*.

● THE SKIN MICROBIOME, EXPLAINED

A garden on your face

Imagine your face as a garden. Not a sterile patch of soil, but a thriving, settled landscape — microbes instead of plants, each species suited to a particular plot. A healthy garden is not one that has been scrubbed clean. It is one that is **balanced**: the right species hold the right ground, helpful organisms crowd out invaders, and the soil itself stays fertile and protected.

WHAT THIS CHAPTER EXPLAINS

From residents to rebalancing

- › Who lives on healthy skin — and what they do
- › Good balance vs. dysbiosis — the tipping point
- › The counterintuitive truth about *C. acnes*
- › Why you actually need *C. acnes* (some of it)
- › The skin barrier — the ecosystem's foundation
- › What inflammation really is
- › From "kill it" to "balance it"

HOW TO READ IT

One big idea per page

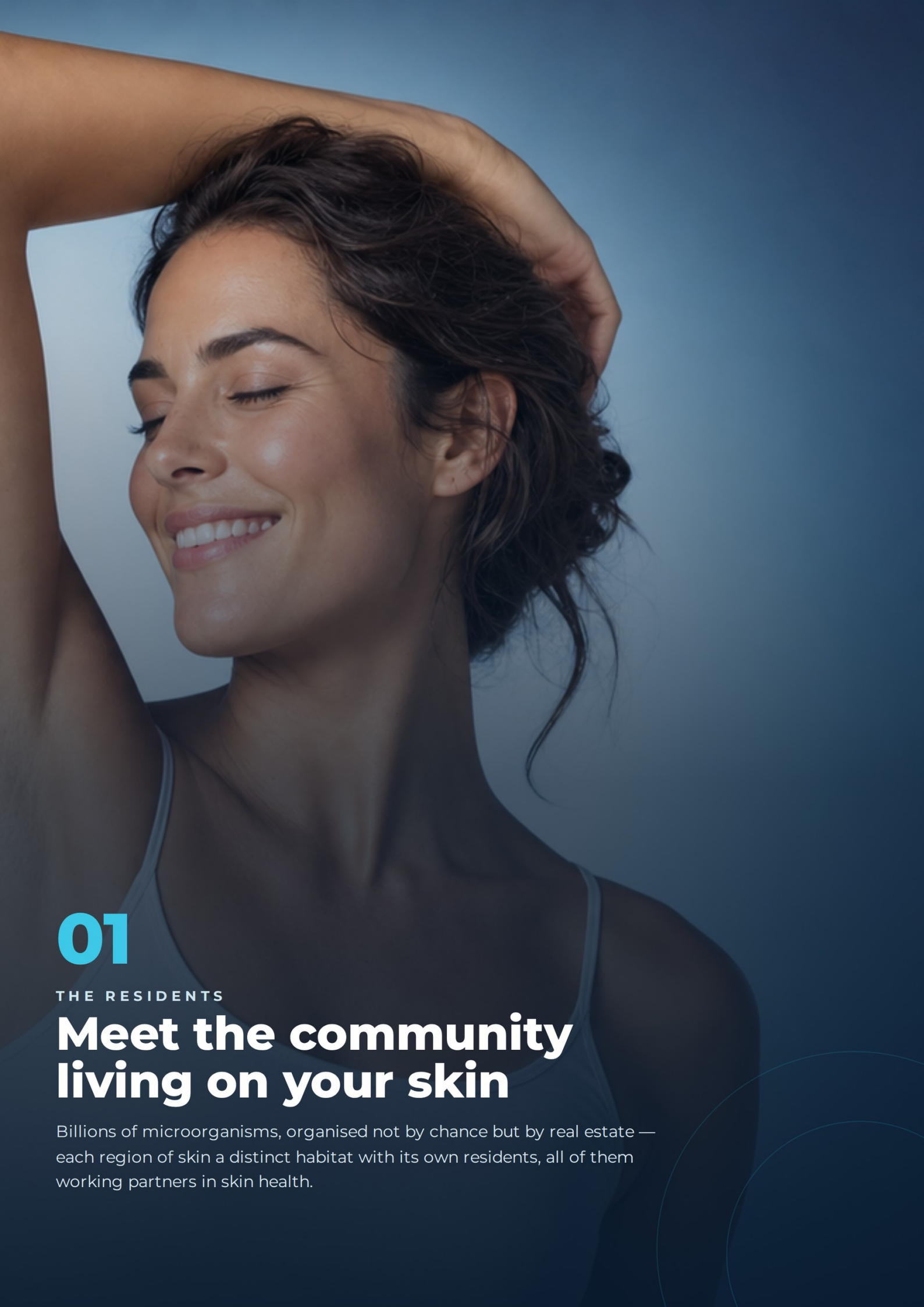
Written for a curious, non-specialist reader — cosmetic brand managers, R&D and retail teams, and anyone who wants the real science without the jargon.

Every claim is grounded in peer-reviewed evidence (2015–2026); full sources are listed at the back. No products are mentioned — this is science, not a sales pitch.

THE SHORT VERSION

Acne is a story of balance, not invasion.

When a garden tips out of balance, you rarely get a new species arriving. More often the residents that were always there shift in proportion — one strain overtakes its neighbours, diversity collapses, and the ground beneath becomes inhospitable. Acne, the contemporary literature increasingly agrees, is a story of exactly this kind: **not an infestation, but a garden that has tipped.**



01

THE RESIDENTS

Meet the community living on your skin

Billions of microorganisms, organised not by chance but by real estate — each region of skin a distinct habitat with its own residents, all of them working partners in skin health.

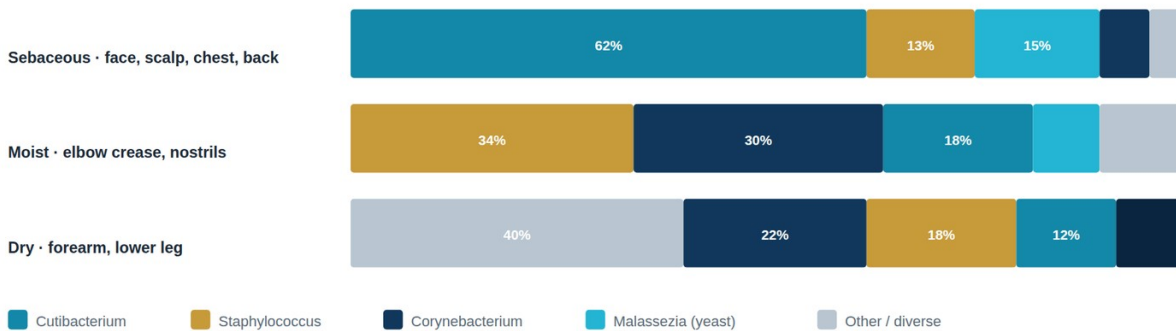
Different neighbourhoods, different residents

Each region of skin is a microhabitat defined by its oiliness, moisture and exposure — and different microbes settle accordingly. This site dependence is one of the most reliably reproduced findings in skin microbiome research.

WHERE THE RESIDENTS LIVE

WHO LIVES WHERE — COMMUNITIES SHIFT BY SKIN REGION

Relative share of the dominant residents at each site type (illustrative, after Grice and Segre; Byrd et al.)



OILY ZONES

Cutibacterium country

Sebaceous sites — face, scalp, chest, back — are dominated by *Cutibacterium*, especially *C. acnes*, which thrives in the lipid-rich, low-oxygen follicle. The yeast *Malassezia* is the most abundant fungal resident.

NOT PASSIVE LODGERS

A working ecosystem

C. acnes makes acidifying fatty acids; *S. epidermidis* acts as a sentinel, releasing succinic acid and lipoteichoic acid that suppress pathogens and calm inflammation. Far from dirt — a working neighbourhood.

Sources: O'Neill & Gallo, *Microbiome* 2018; Grice & Segre, *Nat Rev Microbiol* 2011; Dagnelie et al., *Exp Dermatol* 2021. Shares illustrative.

What tipping the scale looks like

A healthy skin community has two signatures: it is **diverse** and it is **even**. Microbiologists call this balanced state *eubiosis*. Its opposite — the tipped garden — is *dysbiosis*: a disturbance of the community's structure, not necessarily its size.

THE BALANCE, NOT THE SIZE

EUBIOSIS VS DYSBIOSIS — THE SCALE, NOT THE SIZE



Acne is a disturbance of community structure — not a population explosion. The same residents shift in proportion.

REMEMBER THIS

Dysbiosis runs both ways with the barrier.

Acne-prone skin shows higher water loss, a higher (less acidic) pH and more sebum — and these barrier changes track closely with **lower microbial diversity**. A weakened barrier makes the ground less hospitable; an unbalanced community further weakens the barrier. The soil and the garden fall together.

Sources: Dréno et al., *Am J Clin Dermatol* 2020; Cavallo et al., *Sci Rep* 2022; Zhou et al., *CCID* 2022.

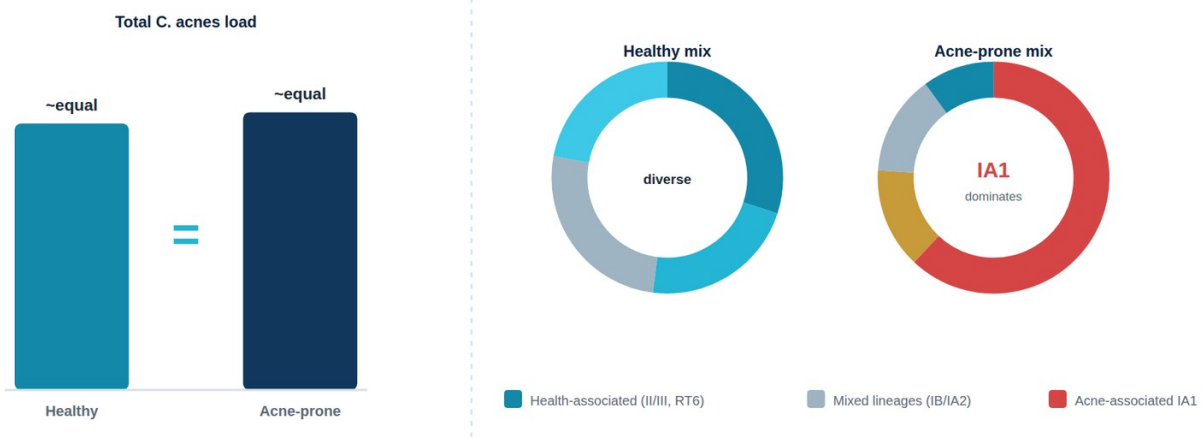
It's not how many — it's which ones

If acne were simply an infection, you would expect far more *C. acnes* on spotty skin. It is not so. The total amount is broadly similar between healthy and acne-affected skin — so blaming the bacterium's presence explains nothing.

SAME QUANTITY, DIFFERENT FAMILY

SAME QUANTITY, DIFFERENT FAMILY

Total *C. acnes* is broadly similar on clear and acne-prone skin — the phylotype mix is what differs.



AN EXTENDED FAMILY

Cousins, not clones

Within the species sit genetically distinct lineages — **phylotypes**. On healthy skin the family is mixed, including health-associated members like phylotype II and ribotype RT6.

THE TILT

When IA1 takes over

On acne-prone skin the family narrows: phylotype **IA1** (carrying virulence-linked ribotypes RT4/RT5) comes to dominate, while the peaceable cousins recede.

Sources: Rozas et al., *Microorganisms* 2021; Mias et al., *JEADV* 2023; Cavallo et al., *Sci Rep* 2022. Composition illustrative of reported patterns.



02

The good guys & the ground beneath

Why you need **C. acnes** — and why the barrier holds it all up

The same species can protect or provoke. And none of it works without an intact skin barrier — the ecosystem's living soil.

Why you need *C. acnes* (some of it)

If *C. acnes* were simply a villain, healthy skin would be better off without it. The opposite is true: a balanced population is one of the skin's most valuable assets. The species is often called "**Janus-faced**" — protective or provocative depending on which strains dominate.

FOUR JOBS A BALANCED *C. ACNES* DOES

FOUR JOBS A BALANCED *C. ACNES* DOES FOR YOU

RoxP

Antioxidant shield

Secretes RoxP — its most abundant protein — neutralising UV- and pollution-driven free radicals.

pH 4.5–5.5

Keeps skin acidic

Turns sebum into fatty acids that hold the acid mantle, the pH beneficial microbes prefer.

Guard

Excludes pathogens

An acidic, lipid-rich surface plus antimicrobials make it hard for *S. aureus* to take hold.

Lipids

Tunes the oil film

A diverse population helps modulate sebum and lipids, keeping the niche stable.

AN HONEST NOTE ON THE EVIDENCE

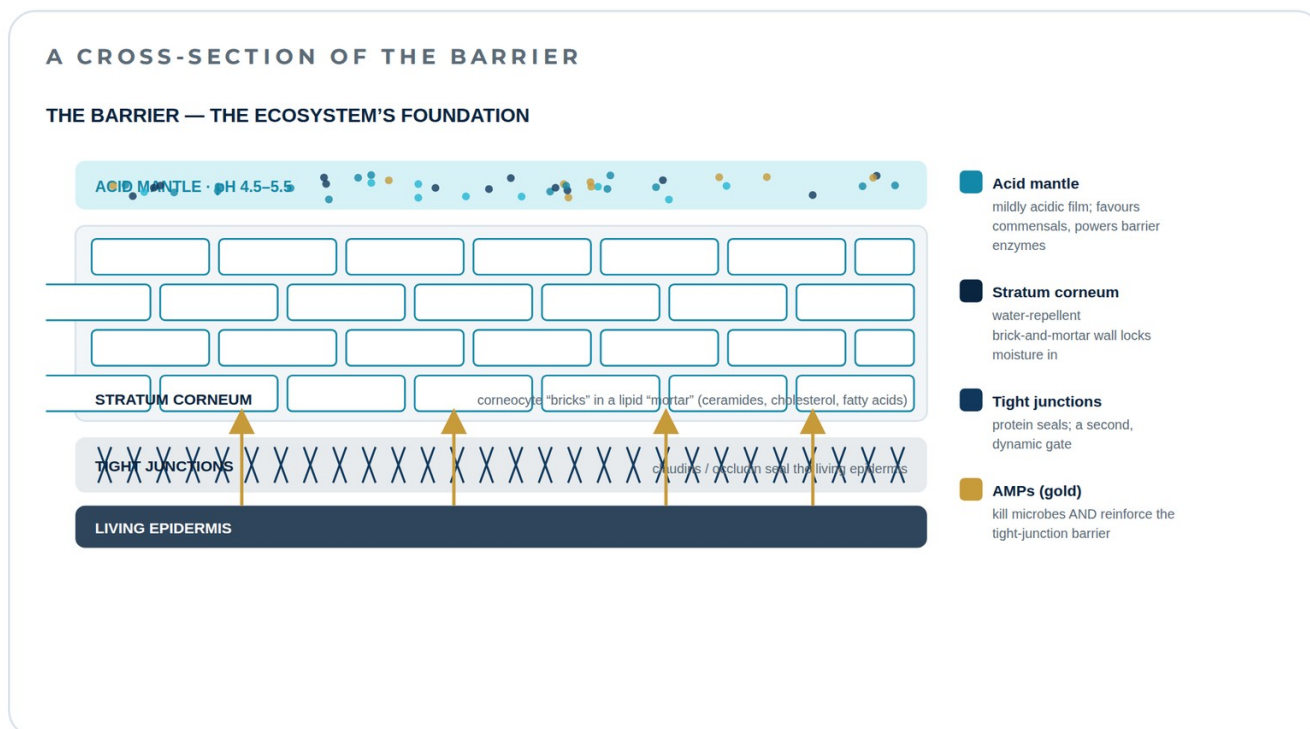
The principle is robust; the strain-level detail is a frontier.

That commensal *C. acnes* provides antioxidant protection, pH maintenance and colonisation resistance is **well supported**. The more specific claim that these benefits belong uniquely to phylotypes II and III — rather than to balanced populations broadly — remains more inferential, with limited strain-resolved human evidence so far.

Sources: Brüggemann et al., *Front Microbiol* 2021; Andersson et al., *Sci Rep* 2019; Stødkilde et al., *Front Cell Infect Microbiol* 2022.

The ecosystem's foundation

A garden is only as healthy as its soil. For the microbiome, that soil is the **skin barrier** — and barrier integrity and microbiome health are inseparable. The barrier is not one structure but several working in concert.



WHEN THE FOUNDATION CRACKS

The consequences cascade.

Over-washing, harsh products or inflammation breach the barrier — water is lost, surface pH rises, antimicrobial-peptide defences falter, and the now-alkaline, leaky terrain favours the unbalanced communities seen in acne. Conversely, **repairing the barrier can help restore microbial equilibrium.**

Sources: Lee & Kim, *Int J Mol Sci* 2022; Bäsler & Brandner, *J Control Release* 2016; *J Integr Dermatol* 2025 (acid mantle).



03

INFLAMMATION & THE NEW SCIENCE

Redness is a response — and the rules have changed

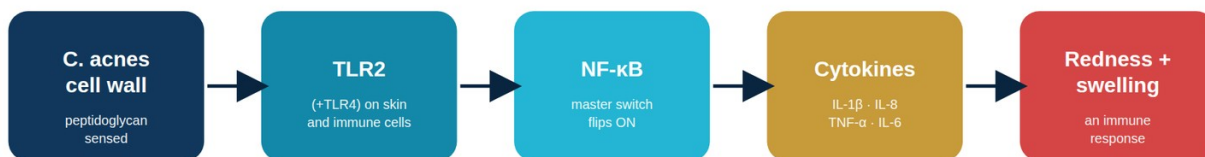
A blemish is the immune system reacting, not the bacteria doing visible damage. Understanding that has rewritten how dermatology approaches acne.

Redness is an immune response

The redness, heat and swelling of an inflamed spot are not dirt — they are your immune system responding. The skin's innate defences sense microbes through pattern-recognition receptors, and the reaction unfolds as a cascade.

FROM TRIGGER TO BLEMISH — THE CASCADE

WHAT INFLAMMATION REALLY IS — AN IMMUNE CASCADE



NOT DIRT — A REACTION.

The blemish is the immune system responding to a community that has tipped. Loss of strain diversity amplifies a Th17-biased response.

THE SENSORS

TLR2 reads the cell wall

Components of *C. acnes* — such as peptidoglycan — are detected chiefly by **TLR2** on keratinocytes, sebocytes and immune cells. When the balance tilts to IA1, this sensing becomes exaggerated.

THE AMPLIFIER

A self-reinforcing loop

Sustained signalling skews the response toward the **Th17** pathway, linked to the loss of strain diversity — which is why calming acne is about quieting an over-reaction, not sterilising skin.

Sources: Qi et al., review in *Front Immunol* 2024; Kistowska et al., *PLoS ONE* 2014; Mias et al., *JEADV* 2023.

From "kill it" to "balance it"

For decades, acne care rested on a simple premise: bacteria cause acne, so kill the bacteria. The microbiome revolution has dismantled the logic beneath it — if *C. acnes* is a normal, beneficial resident and its quantity is similar on clear and acne-prone skin, indiscriminate killing is biologically misguided.

A PARADIGM SHIFT

A PARADIGM SHIFT IN HOW WE THINK ABOUT ACNE

OLD MODEL

“Kill the bacteria”

- Acne = infection by *C. acnes*
- Broad antibiotics + harsh antimicrobials
- Aim: eliminate the microbe
- Side-effect: flattens the whole community,
- lowers diversity, can invite *S. aureus*



NEW SCIENCE

“Restore the balance”

- Acne = dysbiosis (a tipped ecosystem)
- Repair the barrier, preserve diversity
- Target inflammation, not the population
- Strain-selective, microbiome-friendly care
- Tend the garden — don't scorch it

THE HEART OF THE MATTER

Tend the garden — don't scorch it.

Broad antimicrobials flatten the whole community, lowering diversity and sometimes inviting *S. aureus* — the very dysbiosis the skin is trying to escape. The emerging philosophy is to repair the barrier, preserve diversity and target inflammation. **The most promising path is not war on a resident, but helping the community find its balance again.**

Sources: Oliveira et al., *Molecules* 2025; Dréno et al., *Am J Clin Dermatol* 2020; *Clin Exp Dermatol* 2025 (paradigm shift).



Seven things to carry away from this chapter

- 1 Your skin is a living ecosystem.** Bacteria, fungi and viruses form site-specific communities that, in health, stay diverse and balanced.
- 2 Acne is dysbiosis, not overgrowth.** Total *C. acnes* is similar on clear and acne-prone skin — balance, diversity and barrier state are what change.
- 3 It's which strains, not how many.** A narrowing toward the dominant IAI phylotype is the hallmark — not a population explosion.
- 4 C. acnes is largely beneficial.** Balanced populations supply RoxP antioxidant protection, hold the acid mantle and exclude pathogens.
- 5 The barrier is the foundation.** Stratum corneum, tight junctions, acid mantle and AMPs are inseparable from microbiome health.
- 6 Inflammation is an immune response.** Redness comes from TLR2 → NF-κB signalling and cytokines (IL-1β, IL-8, TNF-α) — not dirt.
- 7 The science has moved to "balance it."** Repair the barrier and restore microbial balance rather than sterilise the skin.

Your Skin Has an Ecosystem

KNOWLEDGE HUB · ACNE SCIENCE SERIES — CHAPTER 3

SELECTED REFERENCES

- Dréno B et al. The Skin Microbiome: A New Actor in Inflammatory Acne. *Am J Clin Dermatol* 2020. doi.org/10.1007/s40257-020-00531-1
- O'Neill A, Gallo R. Host-microbiome interactions and the biology of acne vulgaris. *Microbiome* 2018. doi.org/10.1186/s40168-018-0558-5
- Rozas M et al. From Dysbiosis to Healthy Skin: Contributions of *C. acnes* to Skin Homeostasis. *Microorganisms* 2021. doi.org/10.3390/microorganisms9030628
- Mias C et al. Inflammatory acne: *C. acnes* and the Th17 pathway. *JEADV* 2023. doi.org/10.1111/jdv.18794
- Cavallo I et al. Skin dysbiosis and *C. acnes* biofilm in inflammatory acne lesions. *Sci Rep* 2022. doi.org/10.1038/s41598-022-25436-3
- Brüggemann H et al. A Janus-Faced Bacterium: roles of *C. acnes*. *Front Microbiol* 2021. doi.org/10.3389/fmicb.2021.673845
- Andersson T et al. Common skin bacteria protect via secreted antioxidant RoxP. *Sci Rep* 2019. doi.org/10.1038/s41598-019-40471-3
- Stødkilde K et al. Solution structure of RoxP and its antioxidant activity. *Front Cell Infect Microbiol* 2022. doi.org/10.3389/fcimb.2022.803004
- Lee H-J, Kim M. Skin Barrier Function and the Microbiome. *Int J Mol Sci* 2022. doi.org/10.3390/ijms232113071
- Zhou L et al. Epidermal Barrier Integrity, Microbiome Diversity & Composition in Acne. *CCID* 2022. doi.org/10.2147/ccid.s377759
- Qi X et al. A review of skin immune processes in acne. *Front Immunol* 2024. PMC10773853
- Kistowska M et al. IL-1 β & the IL-17/Th17 pathway in acne lesions. *PLoS ONE* 2014. PMC4143215
- Dagnelie M et al. *C. acnes* & *S. epidermidis*: modulators of skin inflammation. *Exp Dermatol* 2021. doi.org/10.1111/exd.14467
- Oliveira MB et al. Rebalancing the Skin: Microbiome, Pathogenesis & Future Therapies. *Molecules* 2025. doi.org/10.3390/molecules30244684
- The acid mantle in skin barrier & microbiome. *J Integr Dermatol* 2025. jintegrativederm.org
- Bäsler K, Brandner JM. Tight junctions in skin barrier function. *J Control Release* 2016. S0168365916305089

● THE SKIN MICROBIOME, EXPLAINED

Scientific foundation: a ConsensusAI synthesis of the skin microbiome in acne (50 papers, 2008–2026), enriched and fact-checked against current peer-reviewed sources (2015–2026). Educational content — not medical advice or product claims.

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