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KNOWLEDGE HUB · ACNE SCIENCE SERIES — CHAPTER 5

Not All *C. acnes* Are the Same

The subtype story that changes everything

Healthy skin and acne-prone skin carry the same bacterium — in the same amount. The difference is not how much lives on your skin, but which kind.

PUBLIC EDITION · FREE TO SHARE

A NOTE BEFORE YOU READ

What this explainer covers

For decades, acne was blamed on one bacterium running wild. The modern science tells a subtler, more useful story — about a **family of subtypes** living in balance, and what happens when one of them takes over. This chapter explains who that family is, which member causes trouble, why the others are worth protecting, and what it means for treating skin at its root.

INSIDE THIS CHAPTER

From species to subtype

- Meet the six phylotypes of *C. acnes*
- What makes phylotype IA1 the troublemaker
- Why dominance — not quantity — drives acne
- The protective subtypes worth keeping
- The hidden cost of wiping them all out
- Why targeted removal also clears blackheads

HOW TO READ IT

Intelligent, not technical

Written for a curious, commercially-minded reader. Analogies do the heavy lifting; every claim is grounded in current peer-reviewed science (2013–2026).

Key terms are bolded. Each visual makes one idea land at a glance. The full reference list is on the final page.

THE SHORT VERSION

Acne is a problem of balance, not numbers.

Healthy and acne-prone skin carry similar total amounts of *C. acnes*. Acne appears when one pro-inflammatory subtype — **phylotype IA1** — comes to dominate and the protective subtypes are lost. The implication is profound: the goal is not to sterilise the skin, but to **rebalance** it.

INTRODUCTION

One species, many faces

Cutibacterium acnes is the most abundant bacterium on human skin — a permanent resident of the oil-rich pores of the face, chest and back. Its very name has cast it for decades as the villain of acne. Yet the same organism thrives, in equal abundance, on flawless skin.

The paradox dissolves the moment we stop treating *C. acnes* as a single entity. Like a large family, the species contains members with very different temperaments. Some are aggressive, biofilm-building, inflammation-provoking strains. Others are quiet commensals that keep the skin acidic, defended and in balance.

Over the past decade, high-resolution genetic typing has resolved *C. acnes* into distinct **phylotypes** — and shown that acne is less a story of bacterial overgrowth than of a shift in the balance of power among these subtypes. This chapter unpacks who the family members are, which one causes trouble, why the others are worth protecting, and what it all means for skin health.

6
PHYLOTYPES WITHIN ONE SPECIES

~96%
IA1 SHARE OF C. ACNES IN SEVERE BACK ACNE

37×
C. ACNES LOST WHEN BROADLY SUPPRESSED

01

MEET THE FAMILY

The phylotype classification

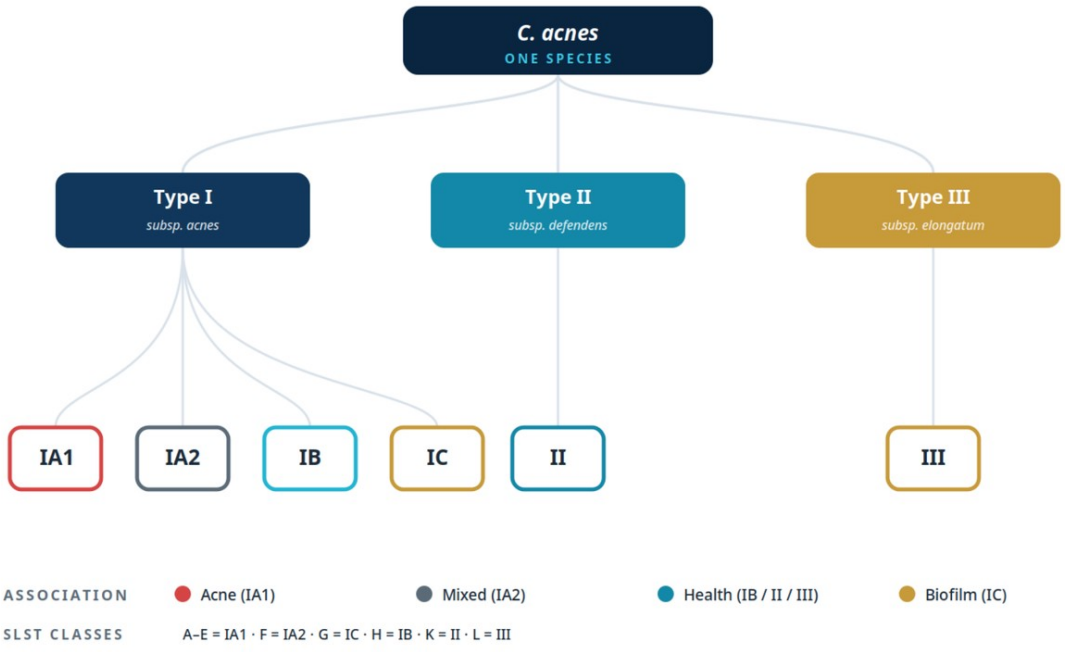
Three subspecies, six phylotypes, one shared name. How scientists learned to tell the members of the *C. acnes* family apart.

SECTION 1 · MEET THE FAMILY

A family of six

C. acnes is divided into **six main phylotypes** — IA1, IA2, IB, IC, II and III — nested within three broader lineages now recognised as subspecies: type I (*subsp. acnes*), type II (*subsp. defendens*) and type III (*subsp. elongatum*). The names of the latter two are telling: *defendens*, the defender, and *elongatum*, a health-associated lineage almost entirely absent from acne lesions.

THE C. ACNES FAMILY TREE



This map was drawn incrementally — from single-gene typing to **multilocus sequence typing (MLST)** and, crucially for studying mixed skin communities, **single-locus sequence typing (SLST)**, which reads one variable region yet can detect several co-existing subtypes in a single sample. Comparative genomics across 255 genomes confirms the picture: an open pan-genome where a large accessory gene set drives the differences between lineages. The phylotypes occupy genuinely different ecological niches — which is exactly why the **composition** of the community, not its size, is what matters.

02

THE TROUBLEMAKER

What makes IA1 so problematic

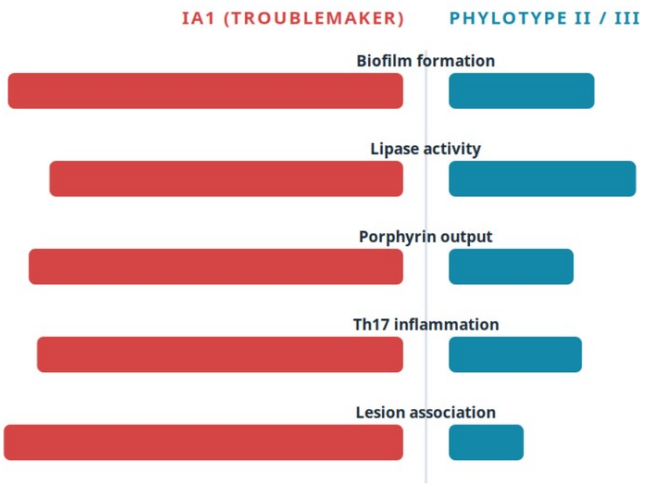
One subtype carries the cluster of traits best suited to colonising — and inflaming — a blocked pore.

SECTION 2 · THE TROUBLEMAKER

The traits that make IA1 aggressive

If any subtype deserves the historical reputation of *C. acnes*, it is **phylotype IA1** — the lineage most consistently enriched in acne lesions. It is the strongest **biofilm builder**, forming the most abundant, best-structured fortresses of self-made matrix. It produces more **porphyrins** — pigments that drive reactive oxygen and ignite inflammation via the NLRP3 inflammasome. And it activates the **Th17 inflammatory pathway** far more strongly than its health-associated cousins.

TRAIT COMPARISON · IA1 VS PROTECTIVE SUBTYPES



A CAREFUL NUANCE

IA1 carries the dangerous traits — it is not a clean villain.

The CAMP factors once blamed on IA1 are in fact present across *all* phylotypes, and a mobile **linear plasmid** predicts a strain's inflammatory potency better than its subtype label does. IA1 is best understood as the lineage that most often **carries** the trouble — which is why the smarter target is its biofilm and behaviour, not the label.

Source: Kuehnast et al. 2018, Int. J. Med. Microbiol.; Spittaels et al. 2021, iScience; O'Neill et al. 2024, J. Invest. Dermatol.; Mias et al. 2023, JEADV.

03

THE PROOF

Dominance, not total bacteria

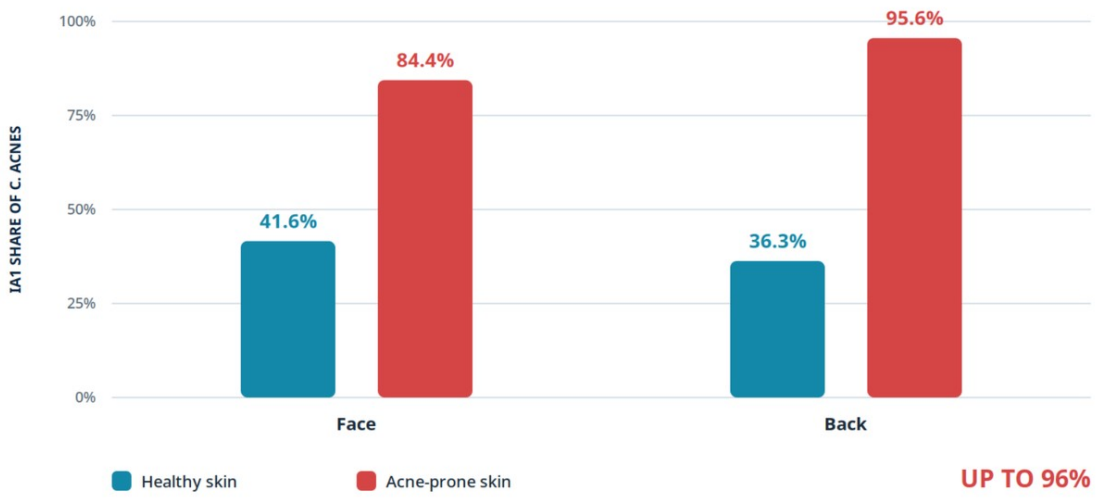
The single most consequential finding in modern acne microbiology: it is the mix that changes, not the headcount.

SECTION 3 · THE PROOF

It is the mix that changes

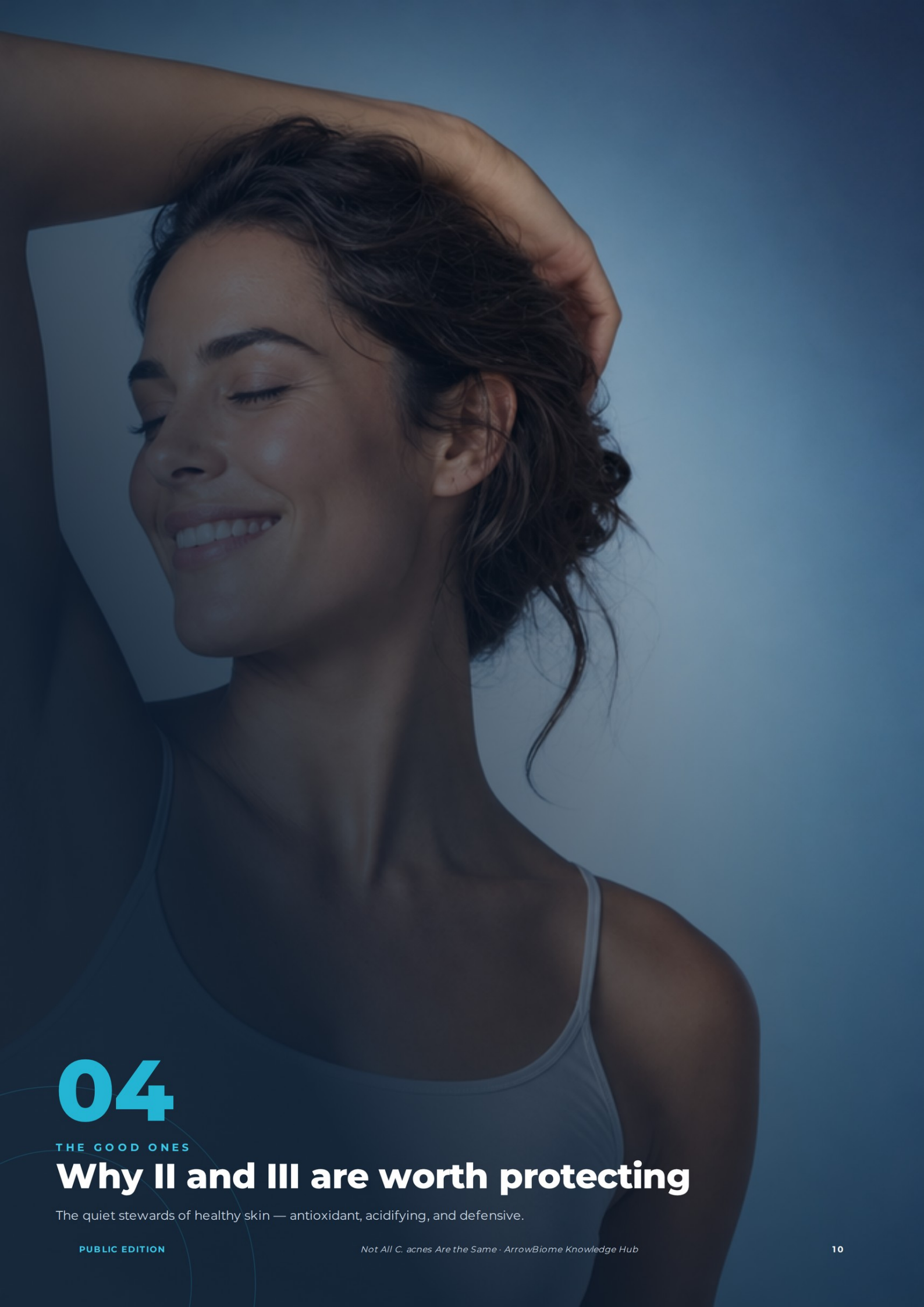
In the foundational study of 49 acne patients and 52 healthy controls, *C. acnes* made up about **87% of bacteria in both groups** — yet the strain populations differed sharply. The shift is stark at the clinical level: on the back of patients with severe acne, IA1 climbs to roughly **96%** of isolates, against about 39% in healthy skin.

IA1 DOMINANCE · HEALTHY VS ACNE-PRONE SKIN



At the level of the individual pore, acne follicles are dominated almost exclusively by a single IA1 clonal complex, whereas healthy follicles hold a heterogeneous mix of subtypes. Acne, then, is a story of **monoculture replacing diversity** — and that matters because the immune system does not see all subtypes equally. In skin-explant studies, IA1 alone provoked far stronger inflammation than the **same quantity** of bacteria supplied as a balanced mixture. The other subtypes actively dampen the response; lose them, and the brakes come off.

Source: Fitz-Gibbon et al. 2013, J. Invest. Dermatol.; Dagnelie et al. 2018, Acta Derm. Venereol.; Dagnelie et al. 2019, JEADV; Cheung et al. 2024, Exp. Dermatol.



04

THE GOOD ONES

Why II and III are worth protecting

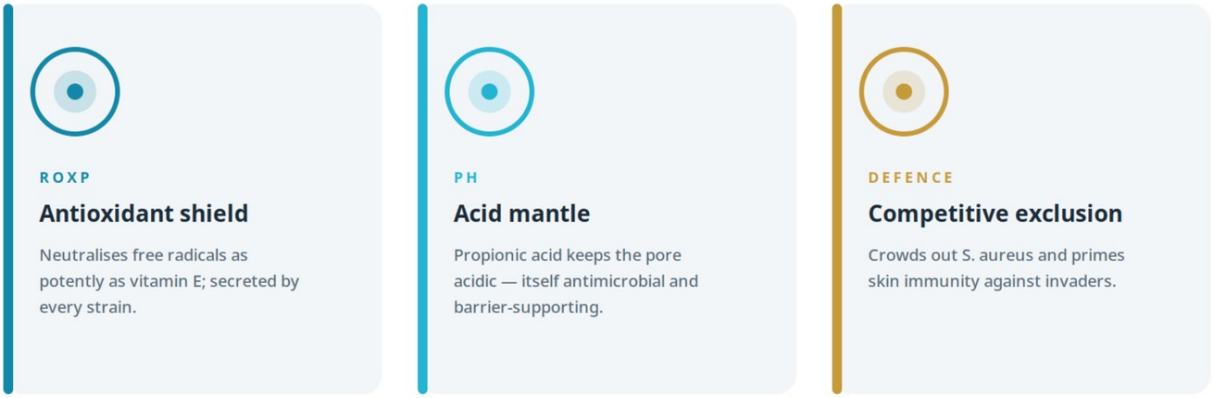
The quiet stewards of healthy skin — antioxidant, acidifying, and defensive.

SECTION 4 · THE GOOD ONES

The protective subtypes

If IA1 is the family troublemaker, phylotypes **II and III** are its quiet stewards. Type II is literally named *defendens* — the defender — and the evidence for its protective role, while more mechanistic than interventional, is substantial and consistent.

THE PROTECTIVE TOOLKIT OF HEALTHY C. ACNES



The whole species secretes **RoxP**, an antioxidant as potent as vitamin E and essential for healthy colonisation. By fermenting sebum, *C. acnes* produces **propionic acid** that keeps the pore acidic — itself antimicrobial — and instructs skin cells to build barrier lipids. And a balanced community provides **colonisation resistance**: it crowds out *Staphylococcus aureus* and primes skin immunity against invaders. Type II and III are reliably enriched on healthy skin and depleted in lesions. They are not bystanders but active contributors to equilibrium — which makes preserving them a therapeutic objective in its own right.

Source: Allhorn et al. 2016, Sci. Rep.; Andersson et al. 2019, Sci. Rep.; Almoughrabie et al. 2023, Sci. Adv.; Tsuru et al. 2021, Microbiol. Spectr.; Brüggemann et al. 2021, Front. Microbiol.

05

COLLATERAL DAMAGE

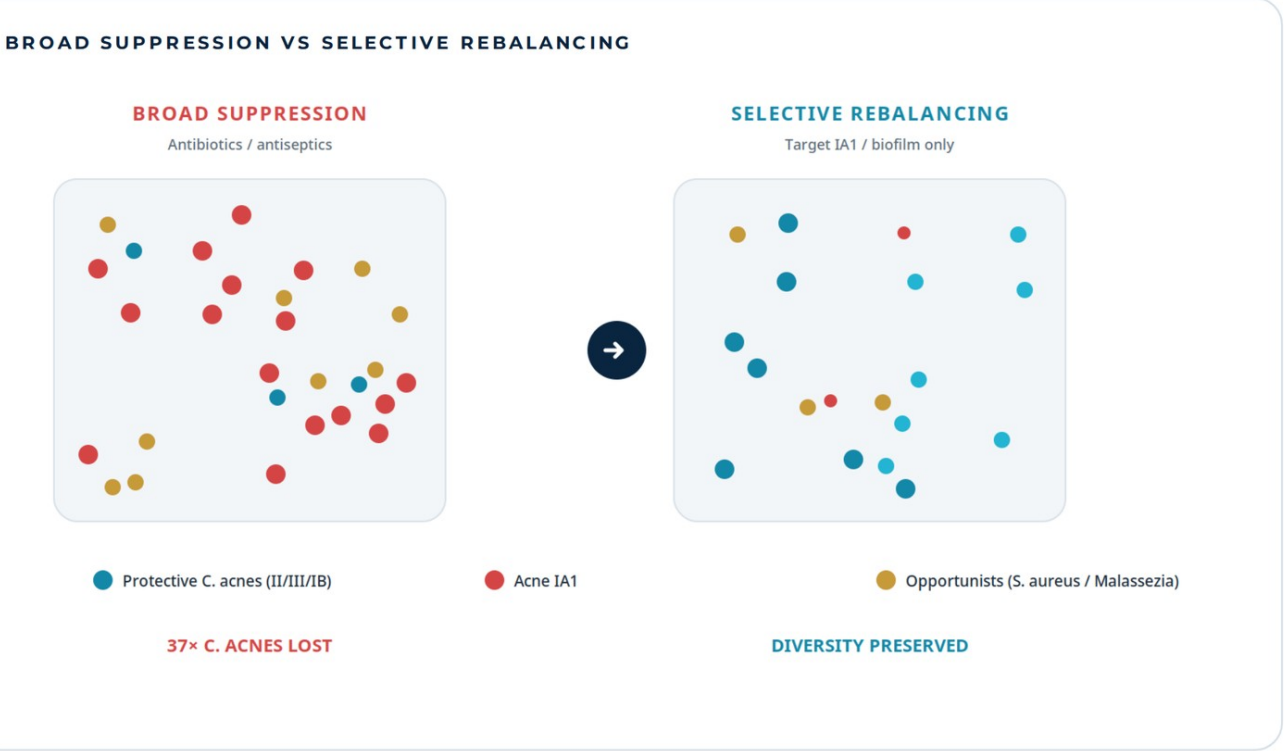
What broad suppression does

Wipe out every subtype, and you remove the guardians along with the troublemaker.

SECTION 5 · COLLATERAL DAMAGE

The cost of scorched earth

If the protective subtypes matter, then any treatment that removes *C. acnes* wholesale carries a hidden cost. This is the logic problem with broad-spectrum **antibiotics and antiseptics**: they cannot tell friend from foe.



Isotretinoin, by starving *C. acnes* of its lipid habitat, produces a roughly **37-fold depletion** — and into the vacuum, *S. aureus* expands sharply. Antibiotics add persistence and resistance: in one multi-country survey, around **66% of acne patients** carried resistant *C. acnes*. Losing the acidic, *C. acnes*-maintained pore also favours *Malassezia* yeasts and gram-negative organisms, and the barrier suffers as its lipid-inducing signals disappear. The lesson is ecological: **sterilising the skin is not the same as healing it.**

Source: Feidenhansl et al. 2024, JEADV Clin. Pract.; Dessinioti & Katsambas 2024, Dermatol. Ther.; Dréno et al. 2020, Am. J. Clin. Dermatol.; Niedźwiedzka et al. 2024, Int. J. Mol. Sci.

SECTION 6 · THE DIVERSITY QUESTION

Is balance loss itself the problem?

Two models compete to explain acne at the microbial level — and the honest answer is that they are **complementary, not rival**. The first holds that the expansion of pro-inflammatory IA1 is the proximate driver. The second holds that the **loss of subtype diversity** — the disappearance of protective IB, II and III — is itself the trigger, regardless of which lineage fills the gap.

MODEL 1 · DOMINANCE

IA1 takes over

A pro-inflammatory subtype expands — bringing biofilm, porphyrins and Th17 activation with it.

MODEL 2 · DIVERSITY LOSS

The guardians leave

Protective subtypes vanish; even an unchanged IA1 becomes more provocative without them.

The bridge between them is mechanistic: a balanced three-subtype mixture is markedly less inflammatory than IA1 alone, at identical bacterial load. Diversity is not merely a marker of health — it is functionally **protective**. The two models are one event seen from two angles: a balanced community collapsing into a pro-inflammatory monoculture.

AN HONEST LIMIT

Cause or consequence? The jury is still out.

Most evidence is cross-sectional. Few studies have followed untreated skin over time to prove diversity loss *precedes* the first lesion. The prudent reading: **restoring and protecting diversity** is a well-supported design principle — while claims that diversity loss is the sole proven first cause should be made with care.

SECTION 7 · TARGETED & DURABLE

The case for selectivity

If broad suppression is the problem, the logical alternative is **selectivity**: quiet the troublesome IA1 and biofilm fraction while leaving the protective subtypes — and the wider microbiome — intact. Early intervention data are promising.

DERMOCOSMETIC

−36% comedones

An extract that selectively cut the acne-specific IC subtype and raised health-associated IB reduced comedonal and inflammatory lesions without broadly suppressing *C. acnes*.

BIOFILM-TARGETING

Diversity intact

A branched lysine dendrimer engineered to disrupt IA1 biofilm specifically cut blackheads and pustules while microbiome diversity stayed measurably unchanged.

These results are encouraging but **preliminary** — small, often open-label studies. And one question remains genuinely open: **recolonisation kinetics**. No published human study yet measures how quickly IA1 returns after selective depletion, or how long a rebalanced community persists. Because the sebaceous, anaerobic pore may re-select for IA1, durable control may require pairing subtype targeting with measures that reshape the follicular habitat itself. This is the field's defining white space — and the first robust recolonisation data will be decisive.

Source: Mias et al. 2023, JEADV; Gervason et al. 2020, Cosmetics; Dashi et al. 2023, Pharmaceutics; Brüggemann et al. 2021, Front. Microbiol.

Why targeted removal clears blackheads

Here lies the most important distinction of all. **Antibiotics do not clear blackheads.** Their action is anti-inflammatory — they target the red papules and pustules of inflammatory acne, which is why guidelines reserve them for inflammatory disease and rely on retinoids for comedones. Comedonal acne has long been the blind spot of antibacterial therapy.

WHY ANTIBIOTICS MISS THE COMEDONE

Antibiotics target inflammation; only biofilm disruption addresses the retentional plug.

The reason is the **biofilm**. The microcomedone is the primary site of *C. acnes* biofilm formation, and that biofilm acts as a **biological glue**, anchoring the hyperkeratotic plug inside the pore. Within it, IA1 lipases turn sebum into comedogenic fatty acids that drive the very plugging that traps them — a self-reinforcing cycle. And biofilm-embedded bacteria are dramatically more antibiotic-tolerant. Removing the **biofilm** — rather than merely the bacteria — is what addresses comedonal as well as inflammatory acne. The target, once again, is not the species but its behaviour.

Source: Spittaels et al. 2021, Biofilm; Cavallo et al. 2022, Sci. Rep.; Mayslich et al. 2021, Microorganisms; Dashi et al. 2023, Pharmaceutics.

IN A NUTSHELL

Seven things to remember

01 · Balance, not numbers

Healthy and acne skin carry similar total *C. acnes*; acne is a shift toward IA1 and loss of diversity.

02 · Six subtypes, three subspecies

IA1 is acne-associated; II (*defendens*) and III (*elongatum*) are health-associated.

03 · IA1 carries the trouble

Superior biofilm, more porphyrins, stronger Th17 — though a plasmid, not the label, best predicts potency.

04 · The good ones earn their place

RoxP antioxidant, an acid mantle, barrier support, and defence against *S. aureus*.

05 · Broad suppression backfires

Antibiotics and isotretinoin remove guardians too, inviting *S. aureus*, *Malassezia* and resistance.

06 · Selectivity is the future

Early data show fewer lesions with diversity preserved — recolonisation timing is the open question.

07 · Biofilm removal clears blackheads

The comedonal acne antibiotics cannot reach — the clearest point of differentiation in the field.

> ***"The species is not the enemy. The lost balance is."***

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KNOWLEDGE HUB · ACNE SCIENCE SERIES — CHAPTER 5

Not All C. acnes Are the Same

A public science explainer for a curious, commercially-minded reader. Grounded in a synthesis of peer-reviewed literature (2013–2026) on *C. acnes* phylotype diversity and the skin microbiome. Free to read and share. Not medical advice; evidence strength varies by claim and is noted where the literature is associative rather than causal.

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