ESSENTIALS OF COSMETIC DERMATOLOGY IN AESTHETIC MEDICINE

CBAM Aesthetic Mastery Series

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ABOUT AUTHORS

Dr. Reza Akef is a highly experienced and respected cosmetic physician, with over 15 years of experience in teaching and practicing cosmetic medicine. He is currently the Assistant Professor at McMaster University and the Chairman of the Canadian Board of Aesthetic Medicine (CBAM), where he shares his knowledge and expertise with other healthcare professionals to provide the best care for their patients.

Dr. Akef is board certified by the College of Family Physicians of Canada in both Family and Emergency Medicine. Additionally, he is deeply involved in interventional pain practice cooperation for the treatment of Migraines, chronic pain, TMJ, Myofascial pain, and Fibromyalgia. He is a leading cosmetic physician and an internationally recognized specialist in non-surgical cosmetic procedures.

Dr. Akef is dedicated to understanding his clients' concerns and creating personalized treatment plans that meet their needs and goals. He is committed to thoughtfully and strategically creating individualized combinations of leading, safe treatments that exceed client expectations. He is also the founder of Angel Gloss spa in Toronto, an established, leading destination for non-surgical cosmetic procedures for individuals seeking leading physicians with proven experience, the latest technology, and cutting-edge techniques.

Dr. Akef is actively involved in multiple training courses, webinars, and conferences all around the world, including prestigious international conferences such as IMCAS, AMWC, and Dubai Derma. He participates as a speaker at these events and is always looking to stay up to date with the latest developments in the field of cosmetic medicine.

Mohsen Talani is the Director of CBAM. He has over 18 years of experience teaching anatomy in Canada and internationally to students and healthcare practitioners in different fields of medicine such as physicians, dentists, nurses, chiropractors, and physiotherapists.

After graduating from medical school at a young age, he became a medical doctor in his home country, Iran, and practiced in the field of emergency medicine. While pursuing his Master's thesis in neuroscience at McGill University, he discovered his passion for teaching. Since then, he has been working in the field of medical science education, with a focus on anatomy.

He strongly believes that with the rapidly growing aesthetic industry, all practitioners should have a profound knowledge of skin basics, fat compartments, muscles, blood vessels, and nerves, in order to provide safe and effective care to clients and prevent or minimize post-treatment complications linked to lack of anatomy knowledge.

He is currently training healthcare practitioners who seek to have a deep knowledge of anatomy. He incorporates fun, innovative, and easy-to-understand methods of teaching in all of his classes, guest lectures, and presentations. Additionally, he supervises the cadaver lab dissections and hands-on practicals.

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Preface

Cosmetic dermatology has evolved from a discipline once concerned narrowly with the management of skin disease into a vast and increasingly sophisticated field that now sits at the intersection of medicine, technology, and the human desire for self-confidence. The modern practitioner is expected not only to understand the structure and function of the skin, but to be conversant in laser physics, regenerative medicine, pharmacology, and the careful art of patient assessment — and to know, in any given clinical encounter, which of these tools is the right one to reach for.

This handbook has been written to serve as a compact and clinically useful reference across the six core domains that make up the foundation of contemporary aesthetic practice: acne, scarring, cellulite and adipose disorders, pigmentation, vascular lesions, and hair loss.

Each Part follows the same logical arc that experienced clinicians follow at the bedside — understanding the underlying biology, accurately classifying what is seen, and selecting a treatment plan suited to the individual patient.

The text is written for a mixed audience: medical aestheticians refining their existing knowledge, registered nurses moving deeper into clinical procedures, and physicians who want a structured, single-volume overview of the field. The depth is set deliberately at a level where every chapter is detailed enough to be clinically useful, but compact enough that the whole work can be read in a focused weekend and returned to whenever a particular condition arrives in the clinic.

What this book is not is a substitute for hands-on training, supervised practice, or the manufacturer-specific guidance attached to any device or product.

Aesthetic medicine rewards practitioners who couple a strong theoretical foundation with disciplined practical learning. This volume is intended to be the first half of that pairing.

The material here is based on the curriculum of the Canadian Board of Aesthetic Medicine (CBAM) Cosmetic Dermatology Interactive Online Course, restructured and re-presented as a textbook for ease of reference.

The original lessons have been organised into clinical themes rather than weekly modules, so the reader may use the book either sequentially as a structured learning path or selectively as a clinical reference.

It is the author's hope that this handbook will sit on the desks of practitioners as a trusted companion: open often, marked freely, and returned to again and again as the field — and the practitioner — continue to grow.

How to Use This Book

This handbook is organised into six clinical Parts, each devoted to one of the principal domains of cosmetic dermatology practice. Within each Part, the chapters progress from underlying biology and pathophysiology, through clinical assessment, to treatment selection and execution — mirroring the workflow of a real consultation.

For practitioners new to the field, read the book sequentially. Each Part has been written to stand on the shoulders of the one before it, and the cumulative effect is a complete foundation in cosmetic dermatology.

For experienced practitioners, the book is designed for selective use. The Table of Contents and the Appendices have been built so that any single chapter, table, or scale can be retrieved in seconds during clinical hours.

For trainers and supervisors, each chapter closes with a Key Points summary that may be used as the basis for case discussion, oral examination, or self-review.

The following conventions are used throughout:

- **Key Points boxes** at the end of each chapter distill the most clinically important takeaways.
- **Clinical Pearls** highlight practice-tested observations that experienced practitioners learn through experience.
- **Cautions flag** situations in which a treatment choice or technique carries a heightened risk profile.
- **Figure and table** placeholders marked [FIGURE X.Y] and [TABLE X.Y] indicate where diagrams, clinical photographs, or reference tables are to be inserted at the design stage.
- **Cross-references of the form** (see Chapter X) point the reader to related material elsewhere in the book.

- **The appendices** at the end of the book contain forms, scales, and reference tools that have been formatted for practical use — they may be photocopied or adapted for use in the practitioner's own clinic.

A note on safety: every clinical recommendation in this book represents general practice as currently taught in the source curriculum. The practitioner is, at all times, responsible for applying their own clinical judgement, deferring to the instructions for use (IFU) of any specific product or device, and complying with the regulatory framework of their own jurisdiction.

Part I. ACNE AND ITS MANAGEMENT

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WHY ACNE HAPPENS

Acne is the most common skin condition you will see. It affects about 85% of people between their teens and late twenties, and roughly 1 in 12 is left with permanent scars. For many patients it is not just a skin problem — it damages confidence and mood long after the spots fade.

Acne is a chronic inflammatory disease, and four things have to go wrong together for it to develop.

Once you understand those four things, every treatment in Chapter 3 makes sense — because each one targets one or more of them.

1.1 The pilosebaceous unit

Acne always starts in the pilosebaceous unit: the hair follicle, its oil (sebaceous) glands, and a tiny muscle. These open onto the skin through a pore. They cover the whole body except the palms and soles — which is why those areas never get acne.

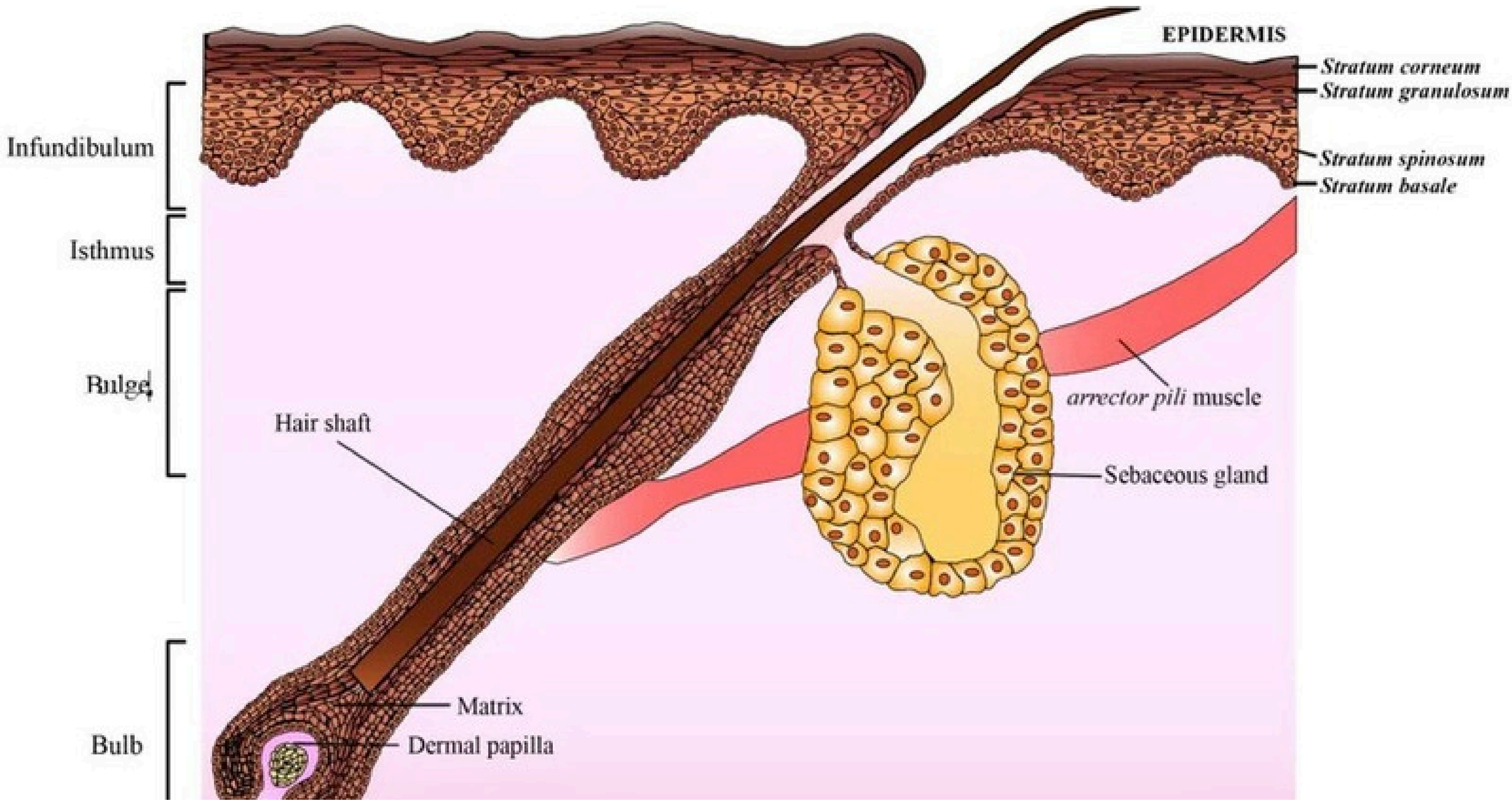


FIGURE 1.1 — Cross-section of the pilosebaceous unit

The oil gland makes sebum, which keeps skin and hair from drying out — about 90% of your skin's oil comes from these glands. Oil production is driven by hormones, especially androgens, which is why acne starts at puberty.

Three things about this anatomy explain almost everything that goes wrong:

- The pore can get blocked when the lining cells get sticky and pile up.
- The inside of the follicle — no oxygen, lots of oil — is the perfect home for one bacterium, C. acnes.
- The oil gland isn't passive — it's an active hormone- and inflammation-producing organ.

1.2 The four pillars of acne

For acne to develop, four things go wrong at once — and they feed each other, which is why acne is so stubborn and why one treatment is rarely enough.

#	What goes wrong	What you see	What targets it
1	Too much oil — and the wrong kind	Shiny, oily T-zone	Retinoids, isotretinoin, hormonal therapy, low-GI diet
2	Pore lining gets sticky and clogs	Whiteheads, blackheads, rough texture	Retinoids, salicylic acid, peels
3	*C. acnes* shifts to a bad strain, forms biofilms	Red, tender spots	Benzoyl peroxide, short antibiotics, light/laser
4	Immune system overreacts (Th17)	Redness, swelling, dark marks	Anti-inflammatories, dapsone, isotretinoin

Table 1.1 — The four pillars at a glance

Pillar 1 — too much oil (and the wrong kind)

At puberty, hormones turn up oil production. In acne, two things happen: there's more oil, and its chemical makeup changes (some fats oxidize into inflammatory chemicals). So it isn't just how much oil — it's what kind. That's why controlling oiliness alone often doesn't fix acne.

Pillar 2 — the pore gets blocked

The lining cells become sticky and form a plug. The first lesion is invisible — a microcomedone — forming weeks before any spot appears. Every acne lesion starts here, which is why treatment must continue even when the skin looks clear.

> 🔑 **Pearl**
>
> why you keep treating after clearing. Microcomedones form 4–8 weeks before you can see them. Stop the moment skin clears and relapse is guaranteed. Keep a topical retinoid going 2–3 nights a week as maintenance.

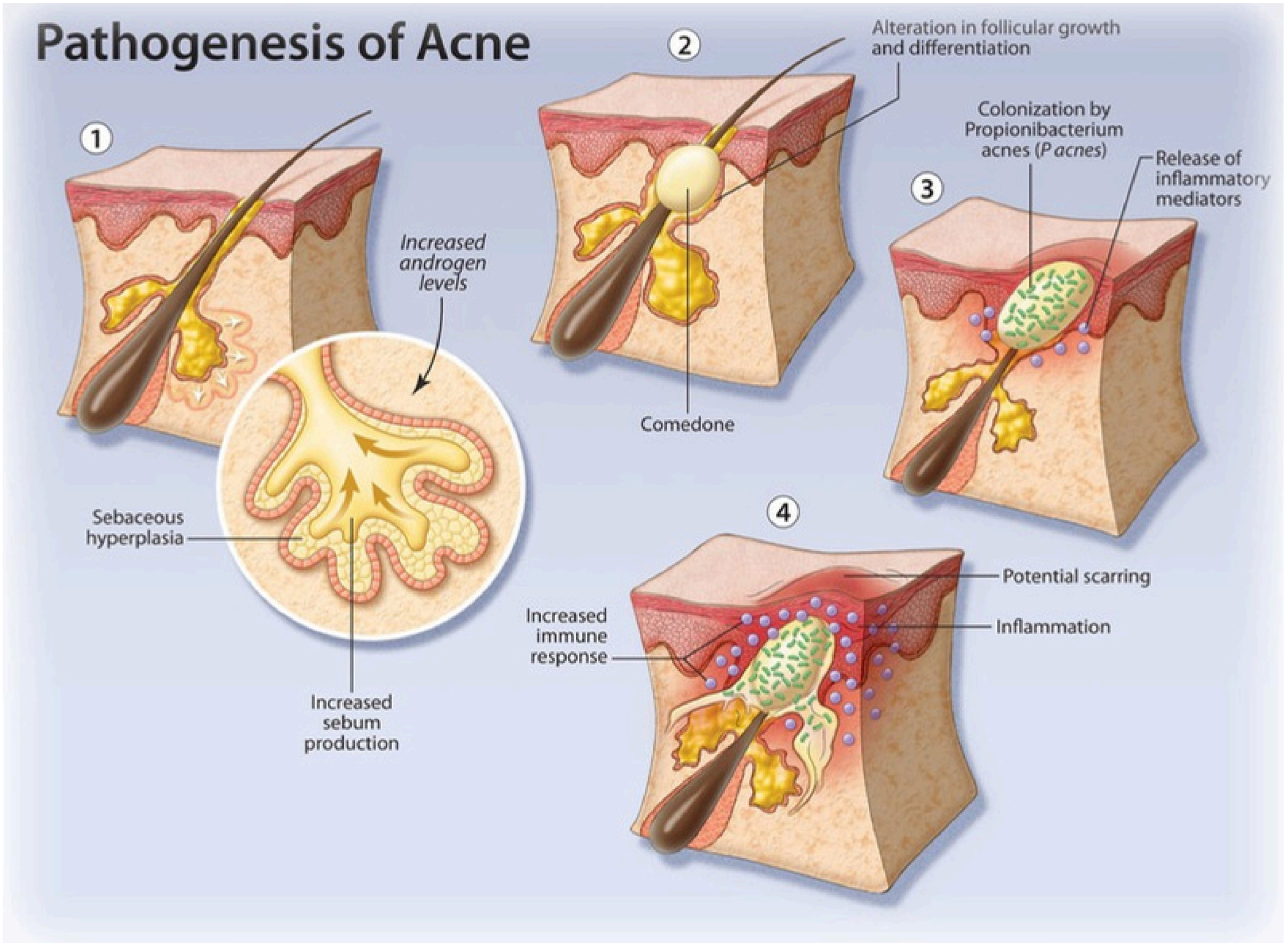
Pillar 3 — the bacteria shift

C. acnes lives on everyone's skin — even clear faces — making up about 87% of the bacteria in these units. So the problem isn't more bacteria. What changes is: one bad strain takes over, the bacteria form protective biofilms (much harder to kill), and the skin's microbe balance is lost (dysbiosis). This is why long-term antibiotics are discouraged — they worsen the imbalance — and why benzoyl peroxide is the go-to: bacteria can't become resistant to it.

Pillar 4 — the immune system overreacts

Around the blocked, oily, bacteria-filled follicle, the immune system releases inflammation chemicals — the redness, swelling, and tenderness of each spot. The pattern is called Th17 inflammation, and it explains why anti-inflammatory treatments help even when they aren't killing bacteria. If the follicle bursts, its contents spill into the skin and you get a papule, pustule, or nodule.

How they feed each other: excess oil feeds the bacteria → bacteria trigger inflammation → inflammation makes cells stickier → sticky cells trap more oil. That loop is why combining treatments that hit different pillars (e.g. retinoid + benzoyl peroxide) beats any single one.



1.3 Hormones, diet, and leptin

Acne is hormone-driven. Without androgens switching on the oil glands there is no acne — which is why it starts at puberty and tracks with conditions like PCOS.

Androgens — the original driver

Testosterone and its stronger form DHT enlarge the oil gland and increase oil. Important: most acne patients have normal blood hormone levels — their oil glands are simply unusually sensitive. So routine hormone blood tests are usually pointless, and local anti-androgens (like topical clascoterone) can work without touching body-wide hormones.

Insulin and IGF-1 — the diet link

These metabolic hormones drive acne: they make the oil gland grow and produce oil, boost androgen action, and trigger inflammation. Acne patients have higher IGF-1, and the higher it is, the worse the acne. Trials show a low-glycemic diet lowers both IGF-1 and acne severity. So “diet matters” is real, not folklore.

Factor	What it does	What to tell the patient
High-GI foods (white bread, sugary drinks, sweets)	Spike insulin → IGF-1 → more oil + inflammation	Switch to low-GI: whole grains, vegetables, legumes
Dairy (esp. skim milk, whey)	Raises insulin and IGF-1	Try 6–8 weeks dairy-free if acne is stubborn
Whey protein	Strong IGF-1 stimulant	Swap for plant-based protein
PCOS / insulin resistance	Chronic high insulin → high IGF-1	Refer for evaluation; consider metformin in selected cases
Chocolate, fried/fatty food	Weak or no consistent link	Don’t make patients feel guilty — focus on GI load and dairy

Table 1.2 — Diet factors and what to tell patients

Leptin — the newest piece

Leptin, a hormone from fat cells, also acts as an inflammation signal in the skin. The oil gland makes its own leptin, which triggers local inflammation, and the same diet pathway (mTORC1) raises it.

This helps explain why obesity, metabolic syndrome, and acne tend to cluster together.(Caveat: blood leptin isn’t consistently raised in acne — its action seems mostly local, at the oil gland itself.)

1.4 Genetics

Acne runs in families, and the science backs it: twin studies put heritability at about 78–85%. A 2022 study of over 20,000 patients found 46 genetic locations linked to acne — affecting oil-gland size, pore stickiness, immune reactivity, and follicle structure. In other words, each of the four pillars has a genetic component.

You won't do genetic testing, but this should change how you talk to patients:

- Take the blame off them. Acne is overwhelmingly genetic — not a hygiene failure or a punishment for diet. Say this out loud; they need to hear it.
- Set expectations early. A strong family history of severe acne usually means a more stubborn course. Promise effort, not miracles.
- Treat aggressively when family history is severe — including early isotretinoin if first-line fails. Once scars form, they don't go away.

Key points

- Acne starts in the pilosebaceous unit (follicle + oil glands + tiny muscle) — everywhere but palms and soles.
- Four things go wrong together: too much/wrong oil, a sticky blocked pore, C. acnes shifting to bad strains and biofilms, and Th17 immune overreaction. They feed each other.
- The problem with C. acnes is the strain shift and biofilms, not quantity — which is why long-term antibiotics are out and benzoyl peroxide is in.
- Androgens start it; insulin and IGF-1 (diet) amplify it; leptin adds local inflammation.
- Acne is ~80% genetic. Severe family history = treat aggressively early, and take the blame off the patient.

🔑 Pearl

the most useful intake question. "Did your parents or siblings have acne, and how bad was it?" The answer tells you how aggressive to be, how to set expectations, and helps lift the guilt the patient is probably carrying.