ArrowBiome®

ARROWBIOME KNOWLEDGE HUB · ACNE SCIENCE SERIES · CHAPTER 1

What Is Acne, Really?

The science behind every breakout

Why pores clog, what the bacteria really do, and why balance — not eradication — is the goal. A premium explainer grounded in current peer-reviewed science.

KNOWLEDGE HUB · PUBLIC EDITION

Prepared with Aiden · June 2026

arrowbiome.com
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A NOTE BEFORE YOU READ

What this chapter explains

Almost everyone has felt the small, private dread of a new breakout — yet acne remains one of the most misunderstood conditions in medicine. This chapter sets out what is genuinely known, and genuinely myth, about why breakouts happen and who gets them. It is pure education: no products, no claims — just the science of the skin.

INSIDE THIS CHAPTER

From pore to person

- Inside the pore — the pilosebaceous unit
- How a pimple forms — the four-factor cycle
- The lesion types, and why they differ
- Meet *C. acnes* — friend, not foe
- The skin microbiome & dysbiosis
- Triggers, genetics & myths debunked
- The emotional reality of acne

HOW TO READ IT

Evidence you can trust

Every scientific claim is cited in-text by a superscript number[00] and listed in full at the back, with a direct link to the peer-reviewed source.

The science draws on a literature synthesis of more than fifty studies (2005–2026), spanning dermatology, microbiology and genetics.

This is the public edition of an ArrowBiome Knowledge Hub explainer — written for brand, R&D, retail and investor audiences alike.

THE SHORT VERSION

Acne is not a hygiene problem — it is biology.

Acne vulgaris is a chronic inflammatory condition of the skin's oil-producing units — the most common skin disease on Earth, affecting an estimated **9.4% of all people** and more than **85% of adolescents** at some point in life.[1,2] Modern science has retired the old "too much bacteria" story for a richer picture: **four interacting forces** — oil, blocked pores, microbes and inflammation — that reinforce one another in a self-perpetuating cycle.[7,14] At its centre sits *Cutibacterium acnes*, overwhelmingly a **helpful, lifelong resident** of healthy skin rather than an enemy to be eliminated.[29,32] The goal of modern skin science is therefore balance, not eradication.

SECTION 1 · INSIDE THE PORE

The pilosebaceous unit

To understand acne, you first have to meet the structure where it lives: the **pilosebaceous unit**. Despite the formidable name, it is simply the working assembly of a hair follicle and its attached oil gland.[3,4] Three parts matter here — the hair follicle, the sebaceous (oil) gland, and the **infundibulum**, the funnel-shaped upper channel through which oil travels to the skin surface.

The sebaceous gland is the engine. It is packed with cells called **sebocytes**, which fill with lipids until they burst completely, releasing their contents as sebum — a process called holocrine secretion.[5,6] That sebum is not waste oil: it lubricates skin, helps it retain water, carries antimicrobial molecules, and takes part in innate immune defence.[4,7] In a healthy unit, sebum flows up through the infundibulum and onto the surface without incident.

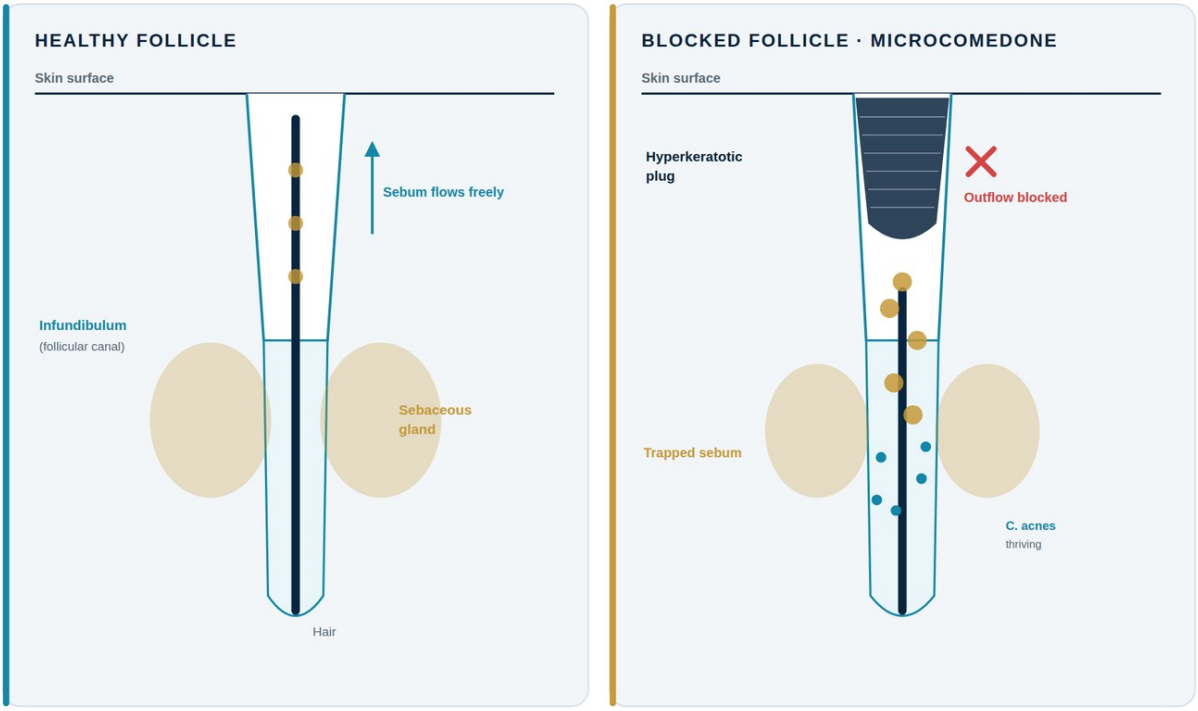


FIGURE 1 · The pilosebaceous unit. In a healthy follicle (left) sebum flows freely to the surface; when the infundibulum is plugged by hyperkeratinisation (right), sebum is trapped and *C. acnes* thrives in the resulting low-oxygen pocket.

WHY ACNE LANDS WHERE IT DOES

It follows the oil

The face, chest and upper back carry the highest density of large, active sebaceous glands — the forehead and nose alone hold roughly 400–900 glands per cm^2.[8] This is why acne concentrates in these "sebaceous-rich" zones rather than appearing everywhere.[9]

WHAT HAPPENS WHEN A PORE BLOCKS

The microcomedone

Sebum rises and the channel's lining cells multiply and stick together instead of shedding — **follicular hyperkeratinisation**.[10,3] Together they form a microscopic plug: the **microcomedone**, the invisible seed of nearly every acne lesion.[12,11,13]

SECTION 2 · HOW A PIMPLE FORMS

The four-factor cycle

For decades, acne was explained as a linear story: oily skin, plus bacteria, equals pimples. The modern model is a system, not a line. Four factors interact and feed back on one another — and the consensus across the literature is that all four are essential to the disease.[7,14,15]

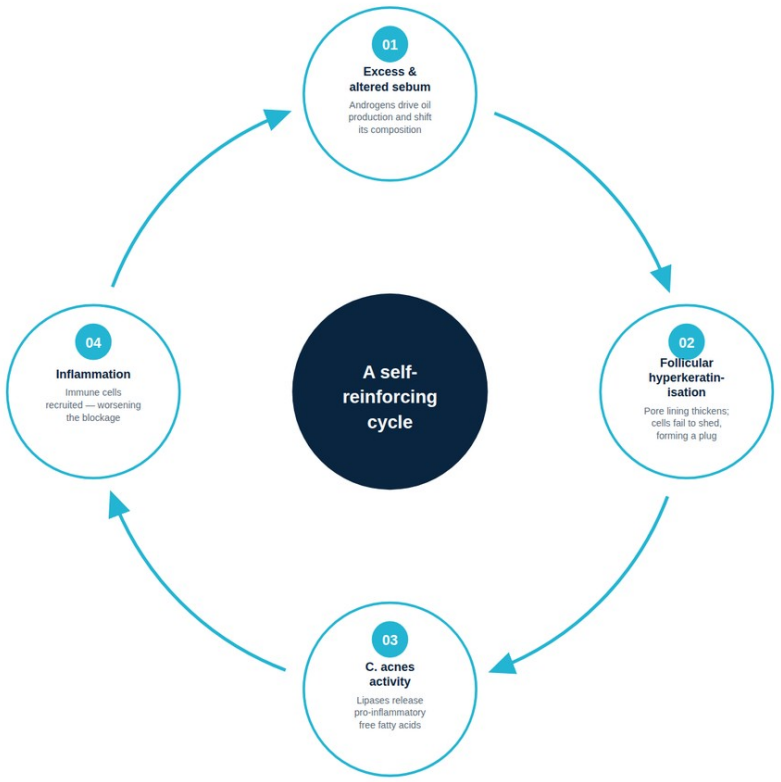


FIGURE 2 · The four canonical drivers of acne do not act in isolation — each one feeds the next, closing a self-reinforcing loop that sustains the disease.

THE SELF-REINFORCING LOOP

Each factor feeds the next

Androgens enlarge the glands and increase oil flow, creating a lipid-rich, oxygen-poor niche that favours *C. acnes*.[17,7] The bacteria use lipases to break sebum into **free fatty acids** — molecules that are themselves both pore-clogging and inflammatory.[19,29] Those acids recruit immune cells and worsen the very hyperkeratinisation that began the blockage.[3,16] As the plug grows, the follicle turns hypoxic, favouring the bacteria still further.[3,20] Remarkably, **inflammation begins before any pimple is visible** — immune cells gather around normal-looking follicles, helping to *create* the clog rather than merely cleaning up after it.[21,22]

SECTION 3 · NOT ALL ACNE IS THE SAME

A guide to lesion types

"Acne" is an umbrella term for several distinct lesions, and the difference between them is not cosmetic — it reflects what is happening biologically and guides how each should be treated. Clinicians divide them into two families: non-inflammatory comedones and inflammatory lesions. Most people have a mix of both.[23,24]

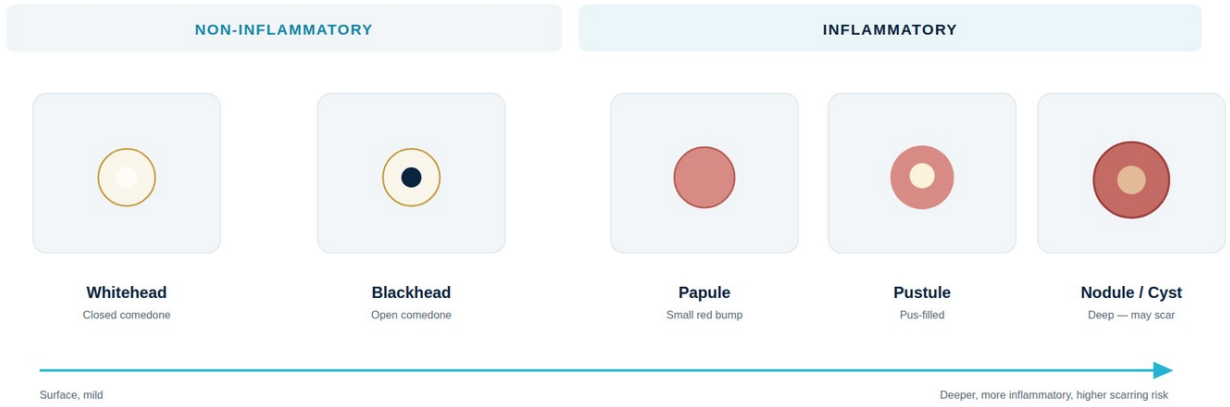


FIGURE 3 · The acne lesion spectrum, from non-inflammatory comedones to deep inflammatory lesions. Severity and scarring risk rise from left to right.

THE BLACKHEAD MYTH, SETTLED

It is chemistry, not dirt

The dark tip of an open comedone is a blend of **melanin pigment** in shed skin cells and the **oxidation** of sebum exposed to air — not grime.[17,25] This is why no amount of scrubbing clears a blackhead: the material sits inside the follicle, and the colour forms there.

WHY THE DISTINCTION MATTERS

Different biology, different risk

Comedonal lesions are dominated by abnormal skin-cell build-up; inflammatory lesions show far stronger immune and microbial activation.[26,27] Deeper lesions — nodules and cysts — reach the dermis and trigger enzymes that degrade its structure, carrying the highest risk of **permanent scarring**.[28]

04

SECTION 4 · THE MICROBE

Meet *C. acnes*

Your skin's most misunderstood resident — overwhelmingly a helpful, lifelong partner, not the villain of the story.

SECTION 4 · FRIEND, NOT FOE

A misunderstood commensal

If acne has a villain in the popular imagination, it is *Cutibacterium acnes*. The reputation is largely undeserved. *C. acnes* is the single most abundant microbe on healthy human skin, making up more than **90% of the bacterial community** in oil-rich areas like the face and back.[29] It lives on virtually everyone, almost always without harm — a lifelong partner that helps keep the skin's ecosystem stable.

A HELPFUL RESIDENT

It earns its keep

By fermenting sebum, *C. acnes* helps maintain the skin's slightly acidic surface — a pH that discourages genuine pathogens such as *Staphylococcus aureus*.[29] It makes antimicrobial compounds and secretes RoxP, an antioxidant that protects skin from oxidative stress.[30]

STRAINS, NOT NUMBERS

Which lineage dominates

Virulence is **strain-dependent, not load-dependent**.[31,32] Certain lineages — notably phylotype IA1 — are enriched in acne-prone skin and carry more aggressive traits; other lineages are tied to clear skin.[33,34] The same person can carry both.

THE CLINICAL IMPLICATION

Balance, not eradication

Wiping out *C. acnes* is the wrong goal. When isotretinoin dramatically depletes it, the door can open for less friendly bacteria like *S. aureus* to expand.[35] Acne is less about *how much C. acnes* is present, and more about **which strains dominate** — so the aim is to restore balance, not to sterilise the skin.

SECTION 5 · YOUR SKIN'S ECOSYSTEM

The microbiome balance

Your skin is not so much a barrier as a thriving habitat. Roughly ten billion microorganisms live across its surface, organised into communities that vary by region.[29] This is the **skin microbiome** — a living ecosystem whose balance is increasingly understood to be central to skin health.

>90%

OF BACTERIA IN OILY ZONES ARE *C. ACNES* ON HEALTHY SKIN[29]

10bn

MICROORGANISMS LIVING ACROSS THE SKIN SURFACE[29]

3

BACTERIAL GROUPS DOMINATE, DISTRIBUTED BY SKIN REGION[9]

On healthy skin, oil-rich zones are ruled by *Cutibacterium*; moist areas favour *Staphylococcus*; drier regions host a mix including *Corynebacterium*.[9] In the sebaceous zones where acne forms, *C. acnes* and *Staphylococcus epidermidis* act as co-guardians, holding the community in equilibrium through antimicrobial molecules and competition for space.[18,36] A healthy microbiome is, above all, a **diverse and balanced** one.

HOW DYSBIOSIS ARISES

Balance lost, not bacteria gained

The modern view of acne centres on **dysbiosis** — a loss of healthy balance — rather than simple overgrowth.[34,12] Inflammatory lesions show reduced microbial diversity and a relative overabundance of virulent *C. acnes* strains.[33,37] Crucially, it is the **loss of strain diversity** — the tipping of the community toward aggressive lineages — that appears to trigger trouble; experimentally reducing diversity increases inflammatory signalling (IL-17, IL-8).[38] Some neighbours are actively protective: a strain of *Staphylococcus capitis* produces peptides that selectively target *C. acnes* without harming other residents.[39,40,41] The field now thinks about treatment not as warfare, but as **gardening**.

SECTION 6 · WHY YOU?

Triggers, genetics & myths debunked

If acne is so universal, why do some people sail through adolescence clear-skinned while others struggle for years? The honest answer is a combination of forces — hormones, genes, sebum chemistry and lifestyle — most of which lie outside personal control. Androgens enlarge the glands and drive oil, which is why acne so often begins at puberty;[17,42] insulin and IGF-1 add fuel.[43,44] And genetics set the stage: twin studies estimate roughly **85% of susceptibility is heritable**.[46,47,48]

MYTH 1

"Acne is caused by dirty skin."

False. Acne is a disease of the pilosebaceous unit driven by internal biology — sebum, hormones, microbes and immunity — not surface grime.[49,50] Over-washing and harsh scrubbing can make it *worse* by stripping the barrier and disrupting the microbiome.[10]

MYTH 2

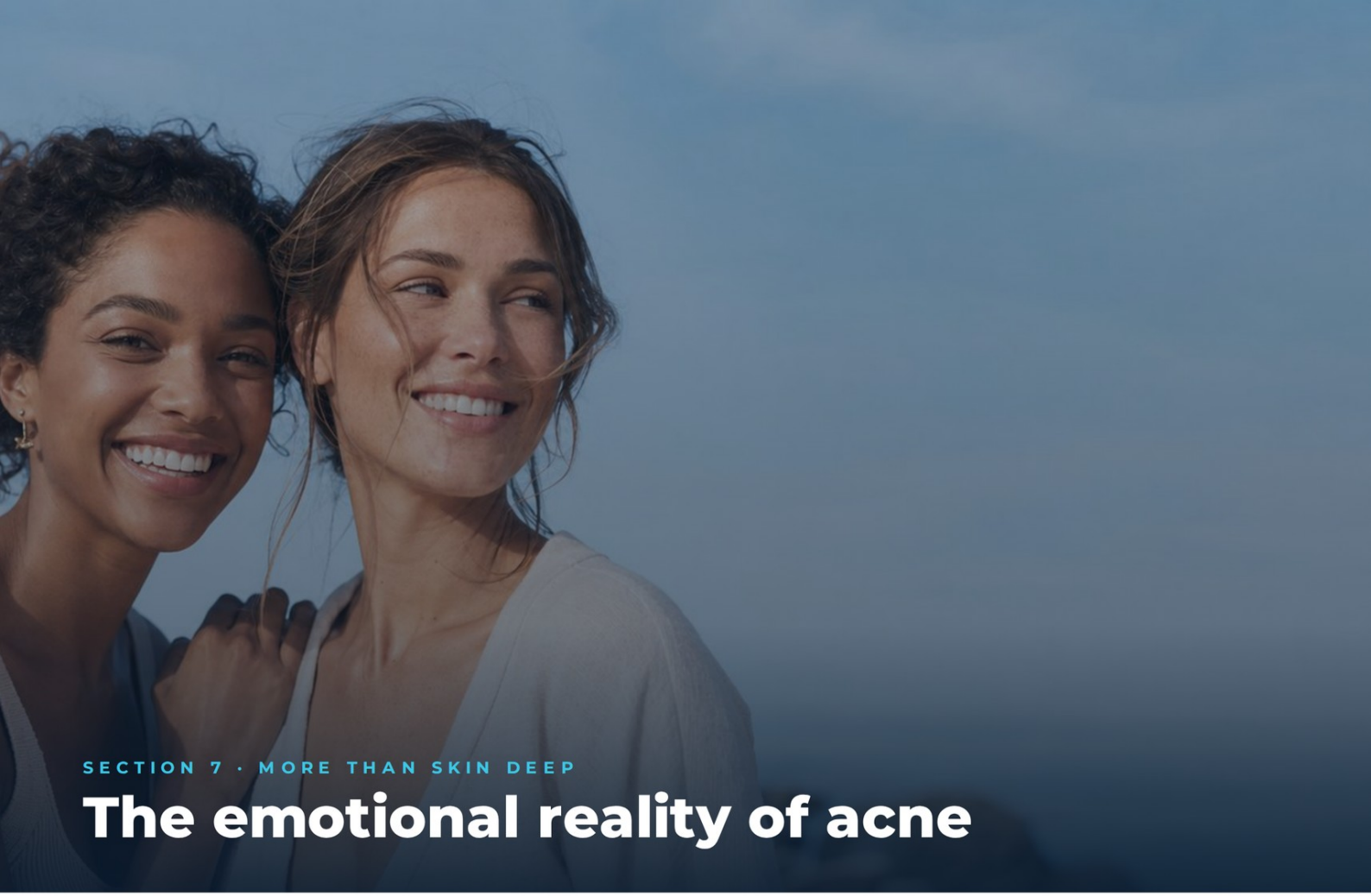
"Chocolate causes breakouts."

Mostly false — with a nuance. Chocolate itself is not the culprit, but diets high in **glycemic load** do show a consistent link. In a randomised controlled trial, a low-glycemic-load diet significantly reduced lesion counts and lowered the free androgen index versus a high-GI diet.[51,52] Dairy's role is genuinely mixed and individual.[50]

MYTH 3

"Popping pimples helps."

False, and harmful. Squeezing ruptures the follicle wall, pushing inflammatory contents deeper, intensifying inflammation, and raising the risk of scarring and post-inflammatory dark marks.[28,23] Chronic picking is recognised clinically as *acne excoriée*.



SECTION 7 · MORE THAN SKIN DEEP

The emotional reality of acne

Acne is visible, and that visibility cuts deep. Its burden extends far beyond the skin into self-image, social confidence and mental health.[54,24] A 2025 systematic review of 101 studies found broadly adverse effects on quality of life and psychological well-being, with the heaviest burden falling on adults, women, and those with more severe disease.[54]

0.22
POOLED CORRELATION BETWEEN ACNE & DEPRESSION[55]

0.25
POOLED CORRELATION BETWEEN ACNE & ANXIETY[55]

101
STUDIES CONFIRM BROAD QUALITY-OF-LIFE IMPACT[54]

WHY IT MATTERS

A genuine human need

In adult women, the quality-of-life impairment caused by acne has been described as comparable to chronic illnesses such as **asthma**.[45] This is not vanity — it is a documented impact on how people live, work and relate to others. It is precisely why solutions that are both **effective and gentle** — that respect the skin's biology rather than assaulting it — are not a cosmetic indulgence, but a real human need.

IN SUMMARY

Seven things to remember

01 **Acne is biology, not hygiene**
It is a chronic inflammatory disease of the pilosebaceous unit — the hair follicle and its oil gland — not a question of cleanliness or willpower.

02 **Four factors drive it, together**
Excess and altered sebum, follicular hyperkeratinisation, *C. acnes* activity and inflammation reinforce one another in a self-perpetuating cycle.

03 **Inflammation comes first**
Immune activity begins before any pimple is visible, reframing acne as immune-driven from the very outset.

04 **Lesion type reflects biology**
Blackheads are oxidised oil and melanin — never dirt; nodules and cysts reach the dermis and carry the highest scarring risk.

05 ***C. acnes* is mostly a friend**
It is a beneficial, lifelong skin resident. Acne is about which strains dominate, not how many bacteria are present.

06 **Balance beats eradication**
Healthy skin depends on a diverse, balanced microbiome — so the goal is to restore balance, not to sterilise the skin.

07 **The human toll is real**
Acne is strongly associated with lower self-esteem, anxiety and depression — making gentle, effective solutions a genuine human need.

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The evidence

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SCIENTIFIC FOUNDATION

Primary source: ArrowBiome / Consensus.ai literature synthesis, **Pathophysiology of Acne Vulgaris** (2026). Enriched and fact-checked against 56 peer-reviewed studies (2005–2026) across dermatology, microbiology and human genetics — each cited in full in the References. Figures are original ArrowBiome illustrations.

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