Seborrheic Dermatitis: New Pathophysiology Evidence and Therapeutic Implications

AMJ American Medical Journal

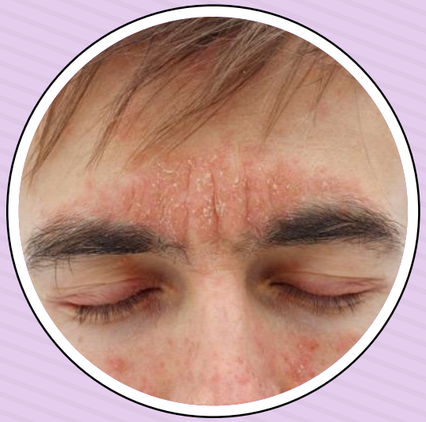
This infographic publication has been supported by a Medical Education Grant from Arcutis Biotherapeutics

Dermatol AMJ. 2026; https://doi.org/10.33590/dermatolamj/PHOO3308

Background

~4.4%

SebDerm is a **chronic, inflammatory** skin disease that **waxes and wanes over time** and affects **~4.4%** of the global population.[1]



Erythema is a clinical manifestation **in all skin types**; it just **may look different** in **different skin types** (for example as hyper- or hypopigmentation).[2]

Perioral | Hairline (forehead) | Hairline (neck) | Scalp

~45% of patients report that SebDerm has a **significant negative impact** on their QoL.[2]

Conventional treatment strategies based on topical **antifungals** and **corticosteroids** generally achieve only **partial symptom control** and their **long-term** use may be limited by **adverse effects.**[3,4]

Pathophysiology

Recent research indicates that the pathophysiology of SebDerm is driven by the **interplay** between **immune activation, skin barrier dysfunction, and skin microbiome dysbiosis.**[5-7]

Skin barrier dysfunction

Downregulation of **barrier-related genes** and attenuation of **lipid metabolism pathways.**[5,6]

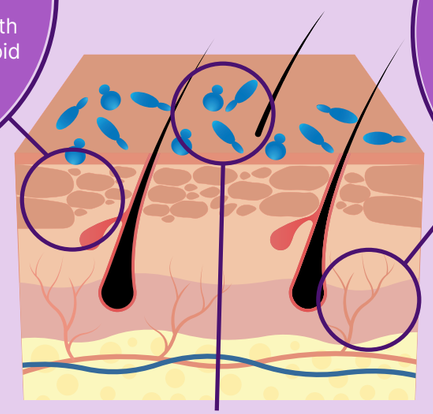
Higher **disease activity correlates significantly** with decreased expression of lipid metabolism genes.[5,6]

Immune activation

Upregulation of genes in the **IL-23/Th17 and Th22 pathways**, and some Th1-related.[5,6]

Upregulation of IL-17 signaling, innate immunity, and **inflammatory pathways**.[5,6]

Higher **disease activity correlates significantly** with increased expression of Th17/Th22 cytokines.[5,6]



Skin microbiome dysbiosis

The skin barrier dysfunction and immune dysregulation in SebDerm is associated with the **overgrowth** of ***Malassezia* spp.** of lipophilic yeasts.[8]

Malassezia spp. produce enzymes that hydrolyze lipids and can **promote inflammation** and **exacerbate skin flaking**.[8]

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Abbreviations:

IL: interleukin; JAK: Janus kinase; PDE4: phosphodiesterase 4; QoL: quality of life; SebDerm: seborrheic dermatitis; Th: T-helper cell.

It's Not All About *Malassezia*

While SebDerm is associated with an **overgrowth of the *Malassezia* spp.**,[8] the **link with disease activity** remains **incompletely defined**.[5]

Assumptions about the role of *Malassezia* in SebDerm are more **clinically driven** than **evidence driven**. However, research suggests that *Malassezia* are **more likely** to be **commensal microbes** rather than a core etiological factor.[7,9]

Immune reactivity to commensal microbes such as *Malassezia* may **exacerbate inflammation**.[5]

Chronic antifungal use in SebDerm may drive drug **resistance**, and appropriate **antifungal stewardship** should be considered in treatment decisions.[10]

Emerging Therapeutic Options

Emerging treatments **target** the **underlying pathophysiology** of SebDerm to achieve more **durable** disease control, particularly in patients with severe, recurrent, or treatment-resistant disease. For example, **biologic therapies, JAK inhibitors, and PDE4 inhibitors**.[11]