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SKIN MICROBIOME IN SEBORRHEIC DERMATITIS: PATHOGENESIS, THERAPEUTIC IMPLICATIONS, AND ENVIRONMENTAL MODULATION OF DYSBIOSIS

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ABSTRACT

Seborrheic dermatitis (SD) is one of the most commonly diagnosed inflammatory dermatoses. In recent years, the approach to the pathogenesis of SD has been expanded beyond the model describing the proliferation of fungi of the genus Malassezia as the main initiating factor. More recent studies have indicated that one of the key mechanisms is the loss of diversity of the skin microbiota, which enables the dominance of microorganisms promoting the exacerbation of disease lesions. Current reports emphasize the impact of disturbances in the composition of the skin microbiota, impairment of the epidermal barrier, and the host immune response as a complex pathomechanism. In parallel, the literature provides numerous lines of evidence indicating that environmental factors, such as pollution and lifestyle, may actively affect the skin microbiome, promoting its dysbiosis. Consequently, the environment may be considered as one of the factors actively modulating the disease and an important element in the context of personalized therapy aimed at maintaining microbiological balance.

The aim of this study is to present current research describing the impact of environmental factors on the development of skin dysbiosis and to discuss a perspective focused on restoring the balance of the skin microbiota and its protective mechanisms as a potential element positively influencing the clinical presentation in patients with seborrheic dermatitis. Disruption of the skin microbiome balance constitutes a key element in the pathogenesis of seborrheic dermatitis, which justifies a therapeutic approach aimed at restoring microbiological homeostasis in order to achieve and maintain disease remission.

KEYWORDS

Seborrheic Dermatitis, Skin Microbiome, Malassezia, Dysbiosis, Epidermal Barrier, Environmental Factors

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I. Introduction

Seborrheic dermatitis is a chronic disease occurring in approximately 4.38% of the population (Polaskey et al., 2024).

The highest number of cases is recorded among infants, adolescents, and young adults, and the next increase in incidence appears after the age of 50. The condition affects men more often than women (Dessinioti & Katsambas, 2013). The disease lesions concern areas of the skin characterized by a high density of sebaceous glands – the scalp, face, and upper part of the trunk. The affected skin is covered with white or yellowish crusts, often accompanied by itching (Dall'Oglio et al., 2022). Seborrheic dermatitis still negatively affects the quality of life of many patients, interfering with their psychosocial comfort and daily functioning (Ozcan et al., 2023). Available therapies do not always guarantee a lasting remission of the disease (Navarro Triviño et al., 2025), which encourages further search for the sources of the condition and more effective treatment methods.

The previous approach explained the pathogenesis of seborrheic dermatitis by the classical model, which assumed that the main mechanism is the overproduction of sebum, stimulating the growth of Malassezia spp. Then these microorganisms caused increased lipid metabolism, during which substances stimulating the formation of disease lesions were produced (Cheng et al., 2025). Although the significance of the classical pathogenic model has been established, it does not fully explain the mechanisms responsible for disease development.

Currently, the etiopathogenesis of seborrheic dermatitis is perceived more broadly as an effect of imbalance in the skin microbiome, impairment of the epidermal barrier, and the host immune response (Navarro Triviño et al., 2025). Moreover, the literature suggests that environmental and lifestyle-related factors may actively modulate these interactions, influencing the composition and stability of the skin microbiome and thereby contributing to disease development, exacerbation, and recurrence. This perspective supports

viewing seborrheic dermatitis as a disorder of the skin ecosystem rather than excessive proliferation of a single species. Despite growing interest, this issue remains poorly described. The aim of this study is to present the main models of seborrheic dermatitis pathogenesis, discuss current treatment approaches, and broaden the perspective by integrating environmental factors as clinically relevant modulators of skin microbiome homeostasis and disease course.

II. Methodology

A narrative literature review was conducted to examine the role of the skin microbiome in the pathogenesis of seborrheic dermatitis, with particular emphasis on microbial dysbiosis and environmental modulation. The review also explored emerging evidence on the impact of environmental and lifestyle factors, including pollution, hygiene practices, and dietary patterns, on skin microbiome composition. To prepare this narrative review, the PubMed and Google Scholar databases were searched, including studies from 2002 to 2025. The literature search was performed using combinations of terms related to: *microbiome, seborrheic dermatitis, malassezia, antifungal, climate, diet, air pollution, detergents, environment.* The review included original research, narrative review, meta-analysis and clinical studies on human and animal models.

III. Results:

1. Classical and emerging evidence – Malassezia as a dominant but not exclusive pathogenic factor

For many years, Malassezia yeasts have been considered a key microbial component in the pathogenesis of seborrheic dermatitis, primarily due to their preferential colonization of sebaceous skin regions. Both culture-based investigations and molecular approaches consistently demonstrate an altered relative abundance and species distribution of Malassezia spp. in seborrheic dermatitis lesions compared with non-lesional or healthy skin, particularly on the scalp and facial areas (M. Park et al., 2021 Xu et al., 2016). High-throughput sequencing studies published in recent years confirm that while Malassezia constitutes part of the normal cutaneous microbiota, patients with seborrheic dermatitis exhibit distinct species distributions, most notably an increased predominance of Malassezia restricta and Malassezia globosa in affected skin sites (Clavaud et al., 2013; Wu et al., 2015).

The pathogenic relevance of Malassezia spp. in seborrheic dermatitis is more closely associated with their lipid-dependent metabolism and interactions with the epidermal barrier than with simple fungal overgrowth. Malassezia species are obligate lipophilic yeasts that lack fatty acid synthase genes necessary for de novo synthesis of fatty acids and therefore depend on host sebum as their primary lipid source, preferentially consuming saturated fatty acids while releasing unsaturated ones, such as oleic acid (Ambaw et al., 2021, M. Park et al., 2021). These yeasts secrete lipases and phospholipases that hydrolyze sebaceous triglycerides into free fatty acids, directly altering the cutaneous lipid milieu (M. Park et al., 2021). Experimental evidence demonstrated that free fatty acids, particularly oleic acid, disrupt the structural organization of the stratum corneum lipids, fluidize the lipid matrix, increase transepidermal water loss, and enhance the permeation of compounds through the skin barrier (Kováčik et al., 2023; Mack Correa et al., 2014). These metabolic effects can promote abnormal keratinocyte desquamation and facilitate deeper penetration of irritants and microbial metabolites, amplifying innate immune activation and contributing to the chronic inflammation characteristic of seborrheic dermatitis (Ambaw et al., 2021; Kováčik et al., 2023). This model underscores the integrated role of fungal lipid metabolism, host barrier integrity, and immune responses in disease pathogenesis rather than attributing pathology solely to fungal overgrowth.

In addition to epidermal barrier alterations, increasing evidence indicates that Malassezia spp. and their metabolic products can modulate cutaneous immune responses. Experimental studies employing human keratinocytes and mice models have demonstrated that selected Malassezia species are capable of activating innate immune pathways, including pattern-recognition receptors such as Toll-like receptor 2, leading to downstream activation of NF-κB signaling and the release of proinflammatory cytokines (Jia et al., 2024). Recent mechanistic investigations further suggest that Malassezia-induced inflammation may involve activation of inflammasome-associated pathways in keratinocytes, providing an additional link between fungal colonization and epidermal immune dysregulation (H. R. Park et al., 2021). Nevertheless, it is essential to note that most available mechanistic data originate from in vitro or ex vivo experimental systems. Current evidence indicates that the relevance of these immune pathways in vivo is likely modulated by host-specific factors, including epidermal barrier integrity, sebaceous gland activity, and interactions with bacterial members of the skin microbiome.

2. Seborrheic Dermatitis as a Disease Associated with Microbial Imbalance

The development of microorganism sequencing methods in recent years has shown that the pathogenesis of seborrheic dermatitis is more complex than previously assumed. Increasingly, it is emphasized that in patients with seborrheic dermatitis (SD), the observed changes are not limited solely to the excessive proliferation of *Malassezia* yeasts. More recent studies of the skin microbiome suggest that these changes also involve other fungal species as well as bacteria (Lin et al., 2021; Sanders et al., 2021). Such observations indicate the presence of dysbiosis, that is, a disturbed balance of microorganisms, as one of the important elements of the disease process. Consequently, the pathogenesis of SD is becoming interpreted more broadly—as a result of dysregulation of the skin microbiological ecosystem rather than the action of a single microorganism.

Although *Malassezia* is still considered the most important genus of yeasts associated with SD, DNA sequencing–based studies increasingly show that many other fungal species, which were previously not mentioned in the context of the pathogenesis of this disease, are also present in lesional skin. In the study by Tanaka et al., pyrosequencing was used to analyze the skin microbiome of patients with SD. Thirty fungal taxa were identified, with *Malassezia* remaining the dominant genus in both lesional and non-lesional skin. However, the microbiota within the lesions showed greater diversity, which further supports the hypothesis of the key role of dysbiosis in the development of SD (Tanaka et al., 2014).

In a separate study, the team of Mahmoudi (Mahmoudi & Rezaie, 2020) focused on the analysis of the microbiome present within lesional skin. In the course of the study, several fungal species other than *Malassezia* were identified that were present in noticeable amounts, including *Candida parapsilosis*, *Cryptococcus albidus*, *Rhodotorula mucilaginosa*, and *Penicillium polonicum*. Importantly, these microorganisms may also be present on the skin of healthy individuals, which leads to interpreting the obtained results in terms of a disturbance of microbiological balance rather than a classical fungal infection. It is also worth noting that some of these fungi—especially *Candida parapsilosis*—may coexist with *Malassezia* and together form a biofilm. Such an association may hinder treatment and sustain skin inflammation. However, it should be remembered that the conclusions were based on a relatively small study group and should not be fully extrapolated to the entire patient population.

Healthy skin is characterized by the dominance of Actinobacteria—including *Cutibacterium* (formerly *Propionibacterium*)—particularly in sebum-rich areas. In contrast, moist areas are more readily colonized by *Staphylococcus* and *Corynebacterium* (Grice & Segre, 2011; Byrd et al., 2018).

In the study by Tao et al., the bacterial skin microbiome of healthy individuals was compared with that of patients with seborrheic dermatitis. Analysis of the samples revealed clear differences in the dominance of individual bacterial groups between the compared populations. In patients with seborrheic dermatitis, bacteria of the genus *Staphylococcus* were more frequently detected (35.9% vs. 8.4%). In contrast, the proportion of *Cutibacterium*, which dominated the microbiome of healthy skin, was clearly reduced in these patients (37.8% vs. 63.5%) (Tao et al., 2023).

Similar observations were presented in a study conducted by Sanders et al., in which the composition of the bacterial skin microbiome of patients with seborrheic dermatitis was analyzed and compared with that observed in healthy participants. The authors observed clear differences between the two groups, and lesional skin was more frequently colonized by bacteria of the genus *Staphylococcus* (Sanders et al., 2021). A comparable pattern of changes within the skin microbiome was presented by Lin et al., who demonstrated that the scalp microbiome of patients with seborrheic dermatitis differs from that observed in healthy individuals. It was also emphasized that the observed disturbances do not concern a single microorganism—significant presence was observed by both fungi of the genera *Malassezia* and *Aspergillus*, as well as bacteria, including *Staphylococcus* and *Pseudomonas* (Lin et al., 2021). This supports a newer approach to viewing seborrheic dermatitis as a condition resulting from a disturbance of the skin's microbiological balance rather than from the action of a single pathogenic factor.

In dermatological literature, dandruff is often considered alongside seborrheic dermatitis and described as a clinically milder, limited form of the same disease spectrum (Adalsteinsson et al., 2020; Dessinioti & Katsambas, 2013). Studies conducted in patients with dandruff have shown that an increased presence of bacteria of the genus *Staphylococcus* correlated with greater transepidermal water loss and increased pruritus intensity (Saxena et al., 2018). In contrast, the presence of *Cutibacterium* showed a positive correlation with water content and sebum levels in the skin (Xu et al., 2016). The collected data indicate that the microbiological changes correlated with seborrheic dermatitis are of a dysbiotic nature rather than the result of domination by a single microorganism. Increasingly, it is emphasized that disturbances in the skin's microbiological balance, along with impairment of epidermal barrier function, may lead to the intensification of local inflammatory responses and the persistence of disease symptoms.

3. Clinical evidence – modulation of the microbiome improves symptoms of seborrheic dermatitis

Many recent studies suggest that a disrupted skin microbiome is involved in the pathogenesis of seborrheic dermatitis (SD). As mentioned before, clinical observations and microbiological analyses indicate that patients with seborrheic dermatitis show an increased presence of Malassezia yeasts compared to healthy individuals (Navarro Triviño et al., 2025; Tajima et al., 2008). These clinical data are consistent with the hypothesis that SD is associated with disturbance in the skin microbiome, in which the balance between commensal and potentially proinflammatory microorganisms is shifted.

Fundamental evidence that the skin microbiome is a therapeutic target and can be modified comes from the results of antifungal therapies that focus on reducing Malassezia colonization. Standard treatment for seborrheic dermatitis includes topical azoles (such as ketoconazole) and antifungal compounds such as selenium sulfide, which have been shown in numerous studies to significantly improve the clinical symptoms of the disease by reducing yeast load on the skin (Massiot et al., 2022; Tynes et al., 2024). Maitre et al. found that antifungal treatment leads to a reduction in skin flaking and redness, accompanied by a decrease in the Malassezia population, clearly indicating a link between the microbiome and the clinical course of the disease (Maître et al., 2025). Furthermore, therapeutic reduction in yeast levels often correlates with remission of symptoms, while their re-growth is associated with disease recurrence, highlighting the dynamic between microbiome composition and patient clinical status (Gupta et al., 2003).

In recent years, there has been a significant increase in interest not only in antifungal treatment but also in strategies that modulate the skin microbiome, such as probiotics, prebiotics, and postbiotics. Although there are still relatively few direct clinical studies on these interventions in SD, recent work provides promising evidence of their potential benefits. As a key example, Truglio et al. used a topical probiotic-enriched oily suspension containing Lactobacillus crispatus and Lacticaseibacillus paracasei in patients with seborrheic dermatitis; after treatment, a reduction in clinical symptoms and a reduction in Malassezia counts were observed, along with changes in the proportions of skin bacteria (including a higher proportion of Lactobacillus), suggesting that targeted modulation of the mycobiome and bacteriome may modify the course of the disease (Truglio et al., 2024). These data are important because they show the clinical results of microbiome modulation, not only the elimination of fungi, but also the promotion of beneficial microorganisms, which improves skin condition.

Literature reviews on the skin microbiome and postbiotic products also emphasize that postbiotics, inactive microbial cells and their metabolites, are a new class of interventions with therapeutic potential that affect skin barrier functions and immunomodulation without the risk of infection associated with live organisms (Prajapati et al., 2025). Although their use in seborrheic dermatitis requires confirmation in large clinical trials, data support their general role in skin health and microbiome modulation in other dermatoses, making them a promising direction for translational research (Rušanac et al., 2025).

In this context, the growing interest in probiotics, prebiotics, and postbiotics stems from their multidirectional action: probiotics can inhibit potentially pathogenic microorganisms by competing for resources and producing antimicrobials, prebiotics support the growth of beneficial microorganisms, and postbiotics have immunomodulatory and anti-inflammatory effects (Prajapati et al., 2025; Rušanac et al., 2025)

This approach differs from classic therapies, which are mainly antiseptic or anti-inflammatory in nature; it is pro-ecosystemic.

In conclusion, since the skin microbiome can be systematically modified both with conventional antifungal therapies and innovative microbiological interventions, it is crucial to understand which external and environmental factors naturally disrupt or promote the balance of the skin microbiome. In the context of seborrheic, such factors may include climate, hygiene, cosmetics, diet, stress, and exposure to allergens or pro-inflammatory compounds, which can affect both the direct proportions of microorganisms and the host's immune response (Navarro Triviño et al., 2025). For this reason, future research should focus not only on modulating the microbiome therapeutically but also on identifying the natural determinants of its stability and dysbiosis, which is crucial for preventing recurrence and developing personalized treatment strategies.

4. Environment and lifestyle as factors modulating the skin microbiome – a new field of research in SD

Contemporary dermatology is undergoing a paradigmatic shift in understanding the pathogenesis of seborrheic dermatitis (SD). For decades, this condition was viewed mainly through the lens of endogenous sebaceous gland dysregulation and the presence of *Malassezia* yeasts. However, in light of recent molecular and epidemiological studies, this picture is significantly expanding. SD currently appears as a multifactorial disorder in which the skin "exposome"—the sum of all environmental and lifestyle factors an organism is exposed to from conception—plays a key role. This section provides a comprehensive analysis of the mechanisms by which exogenous factors—from urban pollution, through dietary modulation of lipid metabolism, to daily hygiene rituals—affect the delicate microbiological balance of the skin, leading to dysbiosis and inflammation.

An analysis of available scientific literature indicates that the skin microbiome is not a static shield but a dynamic ecosystem that reacts to environmental stimuli in real time. Understanding these interactions is crucial for developing new therapeutic strategies that go beyond traditional antifungal treatment to include holistic lifestyle and environmental modifications for the patient.

4.1 Urbanization, smog, and environmental exposure

The process of rapid urbanization and industrialization observed over recent decades correlates with a significant increase in the prevalence of chronic inflammatory skin diseases, including seborrheic dermatitis. The urban environment, characterized by a specific microclimate, air pollution, and limited contact with natural biodiversity, exerts selective pressure on the skin microbiome, promoting pro-inflammatory species at the expense of protective commensals.

The role of air pollution and particulate matter (PM)

Air pollution, particularly particulate matter with an aerodynamic diameter of less than 2.5 micrometers (PM2.5), has been identified as one of the most significant environmental risk factors for skin health. Breakthrough cohort studies, such as the analysis of UK Biobank data, have provided strong epidemiological evidence of a direct link between exposure to air pollution and the incidence of late-onset seborrheic dermatitis (Chen et al., 2024). These results are fundamental as they shift the weight of the discussion on SD etiology from genetic predisposition to modifiable external factors.

The pathophysiological mechanism by which particulate matter induces or exacerbates SD is multi-stage and involves both direct damage to the epidermal barrier and modulation of the microbiome. As demonstrated by researchers, exposure to high concentrations of PM2.5 leads to a measurable reduction in skin microbiome diversity. This decline affects both alpha-diversity indices (species richness within a sample) and changes in beta-diversity (differences between samples). Loss of biodiversity is an adverse phenomenon because a diverse microbiome acts as a buffer, protecting against the dominance of individual pathobionts. Under smog exposure conditions, there is an overgrowth of pathogenic microorganisms accompanied by a decline in beneficial commensal bacteria, resulting in dysbiosis (Tai et al., 2025). Additionally, PM2.5 exerts a direct effect on skin physiology that favors the growth of *Malassezia*. Studies have observed that these particles damage tight junction proteins in the epidermis, leading to increased transepidermal water loss (TEWL) and elevated skin pH (Boonpethkaew et al., 2024; Kim et al., 2021). The physiological acidic pH of the skin (approx. 4.7–5.5) is crucial for inhibiting the growth of pathogens such as *Staphylococcus aureus* and for maintaining the proper lipid structure of the epidermis. Alkalization of the skin environment resulting from pollution creates an ecological niche favoring colonization by unwanted bacteria and may modify the virulence of lipophilic fungi.

Polycyclic Aromatic Hydrocarbons (PAHs) and the "Cutotype" Concept

A particularly toxic component of urban smog involves polycyclic aromatic hydrocarbons (PAHs), which adsorb onto the surface of PM particles. Research on the impact of PAHs on the skin microbiome has yielded fascinating discoveries regarding the existence of so-called "cutotypes"—specific microbiological profiles determined by the level of exposure to pollutants. Using advanced metagenomic sequencing, it was demonstrated that chronic exposure to PAHs is strongly correlated with changes in the composition and function of the skin microbiome. In individuals living in areas with high PAH concentrations, a significant decrease in the integrity and stability of microbial networks was noted (Leung et al., 2020).

A key discovery in the context of SD is the observation that PAH exposure levels correlate with the abundance of fungi of the genus *Malassezia* and bacteria of the genus *Propionibacterium* (currently *Cutibacterium*) (Leung et al., 2023). This suggests that organic pollutants may act as "fuel" or a selective factor for these microorganisms. This mechanism is likely linked to the aryl hydrocarbon receptor (AhR) pathway.

PAHs are potent ligands for AhR, the activation of which in keratinocytes and sebocytes can lead to oxidative stress and changes in lipid synthesis. An altered sebum lipid profile, richer in lipid peroxides (formed by the attack of reactive oxygen species generated by PAHs), may stimulate *Malassezia* growth or alter their metabolism to a more aggressive, pro-inflammatory phenotype.

Metabolites produced by the microbiome in response to PAHs also change. Metagenomic studies indicate an enrichment of aromatic compound degradation pathways in the microbiomes of individuals exposed to smog, indicating bacterial adaptation to a toxic environment; however, the cost of this adaptation may be borne by the host in the form of chronic inflammation (Leung et al., 2020).

Climate, UV radiation, and seasonality

The environment comprises not only anthropogenic pollution but also natural climatic factors, which are undergoing rapid fluctuations in the era of climate change. Seborrheic dermatitis shows strong seasonality, with a tendency to improve in summer months and exacerbate in the autumn-winter period. This phenomenon is closely linked to the physiology of *Malassezia* and the function of the epidermal barrier.

Research confirms that temperature, humidity, and ultraviolet (UV) radiation are key determinants of *Malassezia* growth on the skin.

● **UV Radiation:** Although moderate doses of UV exhibit immunosuppressive effects and can inhibit the growth of certain fungi (explaining summer remissions), the interaction of UV with air pollution (phototoxicity) can lead to intensified oxidative stress (Fadadu et al., 2023). Future research should focus on the synergistic action of UV and smog, which may destabilize skin homeostasis more strongly than either factor alone.

● **Humidity and Temperature:** Low humidity and low temperature (typical of winter and air-conditioned rooms) lead to a decrease in stratum corneum hydration and weakening of the epidermal barrier. A compromised barrier is more permeable to *Malassezia* metabolites and antigens, facilitating the initiation of an inflammatory reaction even with a stable fungal population (Akbulut et al., 2022).

The influence of heavy metals is also noteworthy. Studies conducted in Hong Kong showed that although blood levels of heavy metals in patients with eczema and SD were within normal ranges, a correlation was observed between lead levels and the severity of itching and quality of life, as well as reduced levels of zinc, which is crucial for epidermal integrity and antimicrobial immunity (Hon et al., 2010).

The Biodiversity Loss Hypothesis and the "Western" Microbiome

The contemporary view on the environmental impact on SD would be incomplete without referencing the "biodiversity hypothesis." It posits that contact with a microbiologically rich natural environment (soil, plants) enriches the human microbiome and trains the immune system. Urbanization, by replacing green spaces with "grey infrastructure," cuts humans off from these sources (Prescott et al., 2017).

As urbanization progresses and the transition to a Western lifestyle occurs, bacterial diversity of the skin decreases. Ancestral species (e.g., *Lactobacillus* spp., *Burkholderia* spp.) are lost, and their place is taken by dominant genera such as *Staphylococcus*, *Corynebacterium*, and *Cutibacterium*. Crucially for SD pathogenesis, the depletion of bacterial flora is accompanied by an increase in the relative abundance of fungi, including potentially pathogenic genera *Malassezia*, *Aspergillus*, and *Candida* (Skowron et al., 2021). This suggests that the natural bacterial flora acts as a "guardian" controlling the fungal population. In a sterile, urban environment, this mechanism fails, which, combined with barrier damage by smog, creates ideal conditions for the development of seborrheic dermatitis.

Table 1. Summary of the impact of key environmental factors on the pathomechanism of SD.

Environmental Factor	Mechanism of Action on Skin	Impact on Microbiome and SD Course	Source
Particulate Matter (PM2.5)	Damage to tight junctions, increased pH, oxidative stress.	Reduction of alpha-diversity, promotion of pathogen growth, barrier damage facilitating penetration of *Malassezia* antigens.	Chen et al., 2024, Tai et al., 2025
PAHs (Polycyclic Aromatic Hydrocarbons)	Activation of AhR receptor, squalene peroxidation.	Selection of "cutotypes" rich in *Malassezia* and anaerobic bacteria, disruption of microbial networks.	(Leung et al., 2020)
Urbanization (Lifestyle)	Isolation from natural microbiota, "hygiene hypothesis".	Decreased bacterial diversity, increased abundance of skin fungi (fungal dysbiosis).	Leung et al., 2020
Climate (Winter/Low Humidity)	Reduction of stratum corneum hydration, decrease in barrier lipid production.	Weakening of the physical barrier, exacerbation of clinical symptoms despite lack of changes in *Malassezia* abundance.	Akbulut et al., 2022

4.2 Diet, Lipid Balance, and the Mycobiome

The role of diet in the etiopathogenesis of skin diseases, including seborrheic dermatitis, has been a subject of debate for years, often marginalized in favor of pharmacotherapy. However, contemporary studies utilizing advanced metabolomic and microbiological analysis provide undeniable evidence for the existence of a strong "gut-skin axis." In the case of SD, this axis functions primarily through dietary modulation of sebum composition—which is the exclusive food source for lipophilic *Malassezia* fungi—and through systemic regulation of inflammation and oxidative stress.

Lipid Metabolism as a Key to *Malassezia* Pathogenesis

The basis for understanding the impact of diet on SD is the unique biology of the etiological agent—fungi of the genus Malassezia. As shown in genomic studies, these yeasts (e.g., M. globosa, M. restricta) have evolutionarily lost the fatty acid synthase gene, making them obligate lipophiles (De Pessemier et al., 2021). This means they are completely dependent on exogenous lipids supplied by the host in the form of sebum.

Diet directly influences lipogenesis in sebocytes. The intake of specific macronutrients determines the profile of fatty acids secreted onto the skin surface. Malassezia hydrolyzes sebum triglycerides, releasing free fatty acids (FFA). Some of these are consumed by the fungus, but unsaturated fatty acids (such as oleic and arachidonic acid) remain on the skin, penetrating the epidermal barrier and inducing inflammation (De Pessemier et al., 2021). Thus, any dietary modification that alters the quantity or composition of sebum directly affects mycobiome virulence.

Dietary Patterns: Fruits vs. Western Diet

One of the most important epidemiological studies in this area was conducted on a large cohort within the Rotterdam Study. Researchers demonstrated that specific dietary patterns are significantly correlated with the risk of developing SD, challenging the thesis that diet has no impact on this dermatosis (Sanders et al., 2019).

The study results indicated two opposing trends:

1. **Protective Effect of Fruits:** High fruit consumption was associated with a 25% lower risk of SD. This suggests that vitamins, polyphenols, and antioxidants contained in fruits may systemically reduce oxidative stress, protecting sebum lipids from peroxidation (a key inflammatory mechanism in SD).
2. **Harmfulness of the Western Diet:** In women, high adherence to the so-called "Western diet" (rich in meat, potatoes, processed fats, and alcohol) was associated with a 47% increase in the risk of developing SD.

Glycemic Index, Insulin, and "Sugar" Stimulation of Seborrhea

The mechanism linking carbohydrate consumption to SD exacerbation has been precisely described in the context of insulin metabolism. Researchers demonstrated in a prospective case-control study that SD patients are characterized by a significantly higher dietary glycemic load (GL) compared to healthy individuals. Moreover, patients with a severe form of the disease had a higher dietary glycemic index (GI) than those with a mild form (Semiz & Aktaş, 2025).

The pathomechanism pathway is as follows:

1. Consumption of high-GI foods causes a rapid spike in insulin and an increase in insulin-like growth factor 1 (IGF-1) (Alshaebi et al., 2023).
2. IGF-1 is a potent mitogen for sebocytes and a stimulator of lipogenesis. It activates the mTORC1 pathway, leading to increased sebum production.
3. Excess sebum provides substrates for *Malassezia*, enabling their rapid proliferation.
4. Although *Malassezia* does not ferment sugars directly (noted as early as 1939 (Ashbee & Evans, 2002)), hyperglycemia and hyperinsulinemia create a pro-inflammatory and "oily" environment ideal for the development of dysbiosis.

It has also been shown that sugar and sweets consumption is reported by patients as a factor exacerbating symptoms (Alshaebi et al., 2023).

The Role of Antioxidants and Vitamin D

- Oxidative stress is an inherent element of SD pathogenesis. Reactive oxygen species (ROS) generated by *Malassezia* metabolism and environmental factors damage keratinocytes. Studies showed that SD patients have reduced total antioxidant capacity (TAC) of plasma (Semiz & Aktaş, 2025; Akbaş et al., 2022). This suggests a systemic depletion of antioxidant reserves. A diet rich in antioxidants (vitamin C, E, carotenoids) may support ROS neutralization, preventing the oxidation of squalene into its irritating derivatives.
- Vitamin D is also a significant element. Lower levels of 25-hydroxyvitamin D are observed in patients with SD (Woolhiser et al., 2024). This vitamin is crucial for the maturation of the epidermal barrier and the production of antimicrobial peptides (AMPs), such as cathelicidin, which form the first line of defense against excessive fungal growth. Supplementation or a diet rich in vitamin D may therefore act as a microbiome regulator.

The Mediterranean Diet as a Therapeutic Model

- In contrast to the Western diet, the Mediterranean diet (MeD) appears as a nutritional model with therapeutic potential in SD. It is characterized by high consumption of anti-inflammatory omega-3 fatty acids (fish), polyphenols (olive oil), and fiber (Barber et al., 2023).

Although dedicated clinical trials strictly for SD and MeD are lacking, extrapolation of data from psoriasis and acne suggests that this diet may:

- Reduce systemic inflammation (lowering CRP).
- Improve lipid profile (increasing HDL, decreasing triglycerides) – which is significant, as metabolic syndrome is more frequently observed in SD patients (Imamoglu et al., 2016).
- Modulate gut microbiota, increasing the production of short-chain fatty acids (SCFA), which strengthen the intestinal and skin barriers (Ryczaj et al., 2025).

Table 2. Dietary components and their impact on SD pathomechanism.

Dietary Component / Pattern	Mechanism of Action	Clinical Effect in SD	Source
High GI Carbohydrates	Increase in insulin and IGF-1 -> stimulation of lipogenesis in sebocytes.	Increased sebum production, intensification of *Malassezia* colonization, exacerbation of lesions.	Semiz & Aktaş, 2025
Fruits and Vegetables (Antioxidants)	Neutralization of free radicals (ROS), protection of lipids from peroxidation.	25% reduction in SD risk (with high fruit consumption), improvement of antioxidant status.	Sanders et al., 2019; Akbaş et al., 2022
Western Diet	Pro-inflammatory, low in fiber, high content of saturated fats.	47% increase in SD risk in women, gut dysbiosis, inflammation.	Sanders et al., 2019, Woolhiser et al., 2024
Alcohol	Vasodilation, changes in sweat composition, immunosuppression.	Aggravating factor, correlation with symptom severity.	Woolhiser et al., 2024

4.3 Cosmetics, Hygiene, and the "Overcleansing" Phenomenon

In an era of growing hygiene awareness, paradoxically, excessive attention to skin cleanliness has become one of the factors driving the epidemic of skin diseases based on barrier and microbiome issues. This phenomenon, referred to as "overcleansing," is of particular importance in the pathogenesis and therapy of SD. The relationship between hygiene and the disease is extremely delicate here: on one hand, it is necessary to remove excess sebum, which acts as fodder for fungi; on the other, aggressive washing destroys the lipid barrier and eliminates beneficial bacteria, leading to a "rebound" effect and exacerbation of dysbiosis.

The "Overcleansing" Phenomenon and Destruction of Beneficial Biofilm

The traditional approach to hygiene in SD relied on striving to degrease the skin and eradicate microorganisms. However, contemporary studies show that this strategy can be counterproductive. The skin is not a sterile coating but an ecosystem in which commensal bacteria, such as Staphylococcus epidermidis, perform key protective functions.

Breakthrough studies demonstrated that S. epidermidis is not a passive passenger but an active symbiont that secretes a specific sphingomyelinase. This enzyme assists the host in producing ceramides—key lipids binding the stratum corneum and preventing water loss (Zheng et al., 2022).

These bacteria form a beneficial biofilm on the skin surface, which physically and chemically protects against colonization by pathogens, including excessive Malassezia growth.

The phenomenon of overcleansing—consisting of frequent washing with strong detergents leads to the mechanical and chemical removal of this protective biofilm and the leaching of epidermal lipids. Studies showed that changes in microbiome composition are visible just one hour after washing, and aggressive cleansing leads to the depletion of commensal flora (e.g., *Paracoccus marcusii*), while promoting the growth of potentially pathogenic bacteria (Sfriso&Claypool, 2020). In the context of SD, the removal of *S. epidermidis* and ceramides weakens the barrier, making it more susceptible to penetration by irritating fatty acids released by *Malassezia*, closing the vicious cycle of inflammation.

Impact of Surfactants on Microbiome Structure: SLS vs. Mild Alternatives

The choice of cleansing agents is of key importance. Commonly used anionic surfactants in shampoos and face washes, such as sodium lauryl sulfate (SLS), are effective in removing sebum but are irritating to epidermal proteins. Even milder alternatives, such as sodium lauroyl sarcosinate, are not indifferent to the microbiota.

Recent studies showed that regular use of a sarcosinate-based cleanser for 3 weeks led to a significant decrease in facial bacterial alpha-diversity. Moreover, an unfavorable change in the prokaryotic microbiome

structure was observed: an increase in the abundance of genera Acinetobacter, Escherichia-Shigella, and Streptococcus (Zhao et al., 2024). Interestingly, the impact of the detergent on the mycobiome (fungi) was much smaller than on bacteria. This suggests that daily hygiene may selectively destroy bacterial antagonists of Malassezia, giving fungi a competitive advantage and deepening the dysbiosis characteristic of SD.

Laundry detergents may also play a role, especially in individuals with sensitive skin. Although modern "sensitive" type detergents seem safe for the microbiome (Christou et al., 2024), older in vitro studies suggested that detergent residues can damage tight junctions in the epidermis and alter the expression of genes responsible for the barrier (Rinaldi et al., 2024).

***Malassezia* Biofilms – A Therapeutic Challenge**

While the S. epidermidis biofilm is desirable, the biofilm formed by Malassezia constitutes a serious therapeutic problem. Researchers confirmed that Malassezia furfur, the main etiological factor of SD, has the ability to form treatment-resistant biofilms (Cassola et al., 2024). In this structure, fungal cells are surrounded by an extracellular matrix that hinders the penetration of antifungal drugs. Studies have shown that classic drugs, such as ketoconazole, are effective in inhibiting biofilm formation, but their ability to eradicate an already mature structure is limited (unlike, for example, chloramphenicol, which is, however, too toxic for routine use) (Cassola et al., 2024). This discovery sheds new light on the problem of SD recurrence—treatment may eliminate planktonic forms of the fungus, leaving the biofilm intact, which quickly reactivates the disease after discontinuation of drugs.

New Strategies: Postbiotics and "Rediversification"

In the face of the limitations of traditional hygiene and therapy methods, strategies aimed at restoring microbiological balance (rebalancing) rather than destroying it are gaining importance.

1. "Microbiome-friendly" Shampoos: Modern anti-dandruff preparations are designed not only to reduce the *Malassezia* population but also to support biodiversity. Clinical study of a new shampoo formula, demonstrated that effective therapy leads to the phenomenon of "rediversification" of the scalp microbiome. This means increasing species richness and restoring a balance in which the dominance of *Malassezia* and *Staphylococcus* is broken in favor of a more diverse, stable flora (Maître et al., 2025).

2. Postbiotics: A promising direction involves postbiotics—preparations containing non-living microorganisms or their metabolites. Researchers investigated the effect of a *Saccharomyces* and *Lactobacillus* fermentation complex (SLFC) on sensitive scalp (a condition often preceding or accompanying SD). It was shown that topical application of the postbiotic for 28 days alleviated symptoms (itching, burning) and led to beneficial changes in the microbiome: a decrease in *Malassezia* abundance and an increase in beneficial bacteria such as *Staphylococcus* and *Lawsonella* (Wang et al., 2023). The mechanism of action of postbiotics relies on acidifying the environment (lactic acid), providing antibacterial peptides, and modulating the immune system, without the risk of infection associated with the use of live probiotics.

In summary, hygiene in SD requires precise balancing. The goal of contemporary care is not sterility, but "microbiological harmony." This means moving away from aggressive cleansers in favor of mild preparations that support the lipid barrier and protect beneficial bacterial biofilms, while simultaneously selectively controlling the *Malassezia* population.

IV. Discussion

The findings of this narrative review highlight a significant paradigmatic shift in the understanding of seborrheic dermatitis (SD). While the classical model focused almost exclusively on the overproliferation of *Malassezia* yeasts driven by sebaceous activity, contemporary evidence suggests a far more intricate pathomechanism involving a complex interplay between microbial dysbiosis, epidermal barrier integrity, the host immune response, and the environmental exposome.

The Shift from Overgrowth to Dysbiosis

The results underscore that SD is not merely a "fungal infection" but a state of ecological imbalance within the skin microbiome. Although *Malassezia* species—particularly *M. restricta* and *M. globosa*—remain central due to their lipid-dependent metabolism, their pathogenicity is increasingly tied to their interaction with the skin barrier rather than absolute numbers. The discovery that *Malassezia* lipases hydrolyze sebum into irritating free fatty acids, such as oleic acid, provides a direct mechanistic link between fungal activity and barrier disruption.

Furthermore, the broadening of the microbiological perspective to include bacteria reveals that SD lesions are characterized by a loss of bacterial diversity. Specifically, the reduction of commensals like *Cutibacterium* and the corresponding increase in *Staphylococcus* suggest that the loss of bacterial "guardians"

may allow for the expansion of pathobionts. This suggests that therapeutic strategies should aim to restore this microbial "harmony" rather than simply eradicating specific species.

The Environmental Exposome and Modern Lifestyle

Perhaps the most significant implication for future clinical practice is the role of modifiable external factors. The correlation between urbanization, air pollution (specifically PM2.5 and PAHs), and SD incidence suggests that the modern environment exerts selective pressure on the skin's ecosystem.

Pollution and Barrier Damage: Particulate matter directly damages tight junctions and increases skin pH, creating an ecological niche that favors pathogen growth.

The "Overcleansing" Paradox: Traditional hygiene practices, often aimed at aggressive degreasing, may inadvertently exacerbate the condition by removing beneficial biofilms, such as those formed by *S. epidermidis*, which help produce protective ceramides. This highlights the need for "microbiome-friendly" skincare.

The Gut-Skin Axis and Dietary Modulation

The evidence regarding the gut-skin axis suggests that SD is also influenced by systemic metabolism. The finding that high-glycemic-index diets can stimulate lipogenesis via the insulin/IGF-1 pathway provides a clear pathway through which dietary choices can "fuel" the proliferation of lipophilic fungi. Conversely, the protective effect of antioxidant-rich fruits and potentially the Mediterranean diet suggests that systemic anti-inflammatory support can improve skin health.

Clinical Implications and Emerging Therapies

The clinical success of probiotics and postbiotics in reducing *Malassezia* counts and improving symptoms provides proof-of-concept for ecosystem-based therapies. Unlike traditional antifungal treatments, which can be followed by rapid recurrence due to resistant biofilms, these innovative approaches focus on "rediversification"—restoring a stable, diverse flora that can naturally check fungal growth.

In summary, SD should be viewed as a multifactorial disorder of the skin's microbiological and physiological ecosystem. Future research and treatment protocols must move beyond a narrow focus on antifungal agents to include barrier repair, lifestyle intervention and microbiome rebalancing.

V. Conclusions

The current state of knowledge regarding the pathogenesis of seborrheic dermatitis is undergoing dynamic changes, research on the skin microbiome and environmental factors is playing an increasingly important role in understanding the mechanisms of this disease. Currently, a growing body of evidence indicates that disturbances in microbiome balance, in combination with impairment of the epidermal barrier and the influence of environmental factors, affect the course of the disease. This implies the need to expand the diagnostic and therapeutic approach toward more personalized strategies aimed at maintaining skin microbiological homeostasis.

Conflicts of interest: No conflicts of interest to declare.

Abbreviations:

AhR - Aryl hydrocarbon receptor
AMPs - Antimicrobial peptides
CRP - C-reactive protein
FFA - Free fatty acids
GI - Glycemic index
GL - Glycemic load
HDL - high-density lipoprotein
IGF-1 - Insulin-like growth factor 1
IL-23 - Interleukin-23
MeD - Mediterranean diet
mTORC1 - Mechanistic target of rapamycin complex 1
NF-κB - Nuclear factor-kappa-light-chain-enhancer of activated B cells
PAHs - Polycyclic aromatic hydrocarbons
PM - Particulate matter
PM2.5 - Particulate matter with aerodynamic diameter ≤ 2.5μm
ROS - Reactive oxygen species

SCFA - Short-chain fatty acids
SD - Seborrheic dermatitis
SLFC - Saccharomyces-Lactobacillus fermentation complex
SLS - Sodium lauryl sulfate
TEWL - Transepidermal water loss
Th17 - T helper 17 cells
UV - Ultraviolet radiation

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