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Review

Seborrheic Dermatitis: From Microbiome and Skin Barrier Involvement to Emerging Approaches in Dermocosmetic Treatment

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Abstract: Seborrheic dermatitis (SD) is a chronic inflammatory skin disease that primarily affects sebaceous-rich areas such as the scalp, face, and upper trunk. While the precise etiology remains multifactorial, the role of the skin microbiome, particularly the proliferation of *Malassezia* species, and alterations in the skin barrier function are critical in its pathogenesis. Disruption of the skin barrier, characterized by increased transepidermal water loss (TEWL) and reduced production of epidermal lipids, creates a favorable environment for microbial overgrowth and inflammation. Recent insights highlight the interplay between the impaired barrier function, immune responses, and the skin microbiome in perpetuating the disease. Additionally, novel dermocosmetic approaches are emerging that target these underlying mechanisms, offering promising therapeutic avenues. This review provides a comprehensive overview of the involvement of skin microbiome and barrier dysfunction in seborrheic dermatitis and discusses the potential of advanced dermocosmetic treatments aimed at restoring skin homeostasis and preventing disease recurrence.

Keywords: seborrheic dermatitis; *Malassezia*; barrier impairment; pharmacological treatment; dermocosmetic treatment

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1. Introduction

Seborrheic dermatitis (SD) is a chronic relapsing skin condition characterized by patches of greasy scaly skin, often appearing on the scalp, face, and chest. It is a common disorder affecting millions of people worldwide, causing significant physical, psychological, and social distress [1].

Despite its high prevalence and impact on patients' lives, progress in understanding the underlying causes of SD has lagged behind other common inflammatory skin diseases like atopic dermatitis and psoriasis. This lack of knowledge has hindered the development of effective and targeted treatments [2].

In this review, we will delve into the latest research on the etiology of SD, focusing on the crucial roles of *Malassezia* fungi and the skin barrier. We will also discuss recent advancements in both pharmaceutical and cosmetic treatments, drawing insights from the most up-to-date scientific evidence. By examining these key factors, we aim to provide a comprehensive overview of SD and potential avenues for future research and therapeutic interventions.

2. Clinical Presentation and Epidemiology

The clinical presentation of seborrheic dermatitis in adults is characterized by papules and plaques with yellow scales, affecting intertriginous areas of the face, scalp, chest, upper back, and sternum with a preference for body regions rich in sebaceous glands and where skin folds are in contact, such as the armpits, groin, and abdomen [3–9].

A biopsy can sometimes be helpful in distinguishing seborrheic dermatitis from clinical mimics such as psoriasis, discoid lupus erythematosus, hidradenitis suppurativa, and rosacea by revealing characteristic histopathologic features. These include spongiosis, parakeratosis (focal or diffuse), clusters of neutrophils in the stratum corneum, mild perivascular lymphocytic infiltrates, and irregular thinning of the granular layer [10].

In cases of psoriasis, sebaceous glands are typically atrophic, smaller in size, and show a reduction in lipidized sebocytes [11,12]. In women, SD may be misdiagnosed as scalp allergic contact dermatitis (ACD) due to overlapping symptoms [13].

Seborrheic dermatitis is frequently described as a bimodal condition with two distinct clinical presentations and differing pathophysiological mechanisms: infantile seborrheic dermatitis (ISD) and adolescent/adult seborrheic dermatitis (ASD) [14,15].

ASD typically begins around puberty with pruritic skin lesions and greasy dandruff. In immunocompetent patients, periods of remission are common, but symptoms tend to recur with increased prevalence around middle age (40–65 years) [16].

Infantile seborrheic dermatitis predominantly affects the scalp, diaper area, neck folds, axillae, and trunk. Distress and pruritus are uncommon, and it is generally asymptomatic. ISD typically presents as erythema with an overlying greasy scale, mainly impacting the face and scalp, areas with high sebaceous gland activity, though it may also involve the trunk and flexural regions [17]. Differentiating between pityriasis capitis and diaper dermatitis can sometimes be difficult, adding to the challenge of accurately estimating ISD prevalence. While few studies have explored this, ISD is thought to peak at around 3 months of age. In an Australian cohort (N = 1116), the prevalence of ISD was reported as 45% in children under 1 year old, 10% in those 0–3 months, and 72% in children 3 months of age, followed by a decline to 1–2% by ages 3–5 years [18]. In ISD, sebaceous gland maturation is upregulated by maternal sex hormones [19]. ISD in the diaper area typically begins around 1 month of age and usually resolves by the end of the first year, with an average duration of 4–6 months [17].

Seborrheic dermatitis is more common in males across all ethnicities, though a recent global analysis found that men outpace women in prevalence only in age groups 65 years and older [20]. The non-inflammatory form, which is not limited to the scalp and often involves erythema along with pruritus, flaking, and scaling, is estimated to affect 50% of individuals affected by SD [21]. Xue Y et al. [22], after analyzing the 2019 Global Burden of Disease data from medical records, disease registries, surveillance systems, and the literature, estimated an age-standardized global incidence of adolescent/adult SD at 1850 cases per 100,000 person-years (PY).

The global prevalence of SD is 4% (95% CI, 3.58–5.17%), with significant variability (I^2 = 99.94%). Subgroup analyses showed a higher prevalence in adults (5.64% [95% CI, 4.01–7.27%]) compared to children (3.70% [95% CI, 2.69–4.80%]) and neonates (0.23% [95% CI, 0.04–0.43%]). Geographic analysis revealed differences, with the highest prevalence in South Africa (8.82% [95% CI, 3.00–14.64%]) and the lowest in India (2.62% [95% CI, 1.33–3.92%]) [23–25].

The Rotterdam Study, a prospective population-based study in the Netherlands, found that the prevalence of ASD among middle-aged and elderly adults (median age 68 years) was approximately 14% [21].

The global prevalence of seborrheic dermatitis, estimated at 3–5%, is likely underestimated due to diagnostic variability across regions.

Adolescent/adult SD affects multiple racial and ethnic groups worldwide. However, standardized comparisons across regions show the highest age-standardized prevalence of ASD in sub-Saharan Africa (416 per 100,000 person-years) and the United States and Canada (359 per 100,000 person-years), with the lowest prevalence in Central Asia (142 per 100,000 person-years) and Eastern Europe (143 per 100,000 person-years). In Italy, data from 2007 to 2017 show that in 2017, approximately 343 individuals per 100,000 were affected by SD [26].

Studies on patients with adolescent/adult seborrheic dermatitis (ASD) have demonstrated a statistically significant association between the severity of SD and elevated levels of stress and depression, highlighting the bidirectional relationship between psychological factors and skin conditions [27,28]. Stress, often acting as both a trigger and an exacerbating factor, can alter immune responses and disrupt the skin barrier, potentially worsening SD symptoms. Conversely, the visible and chronic nature of SD can lead to social embarrassment, low self-esteem, and heightened psychological distress, further perpetuating the cycle.

The negative impact of SD on quality of life (QoL) is increasingly recognized as a major concern, extending beyond the physical symptoms of itch and flaking to include emotional and social well-being. In a European survey of dermatological patients suffering from itch, participants reported significantly higher levels of stress and depression, underscoring how persistent dermatological symptoms can exacerbate psychological burdens and diminish overall life satisfaction [29]. This emphasizes the importance of addressing mental health in conjunction with physical treatment in SD management.

Further insights into the impact of SD on QoL come from assessments using the Dermatologic Life Quality Index (DLQI), which revealed demographic and disease-specific factors influencing the extent of this burden. For instance, women, younger individuals, and those with higher educational levels experienced a more pronounced negative impact. This may reflect differing societal expectations, aesthetic concerns, or heightened health awareness among these groups. Interestingly, patients with dandruff alone reported significantly better QoL than those with SD or SD combined with dandruff ($p < 0.001$ for both comparisons) [30]. This suggests that the broader clinical manifestations of SD, including erythema, scaling, and inflammation, contribute more significantly to distress and functional impairment than dandruff in isolation.

These findings collectively highlight the need for a holistic approach to SD management. Addressing psychological comorbidities, improving patient education, and implementing targeted treatments for symptom control are essential to enhancing patients' overall quality of life. Integrating QoL assessments into clinical practice can provide valuable insights into the broader impact of SD and help tailor interventions to individual patient needs.

3. Etiopathogenesis

The precise pathophysiology of seborrheic dermatitis remains unclear due to its multifaceted and complex etiology. However, three key interrelated factors contribute to its development: individual susceptibility caused by an imbalanced immune response leading to inflammation, cutaneous microbial dysbiosis characterized by notable colonization of *Malassezia* species, and a compromised epidermal barrier [Figure 1].

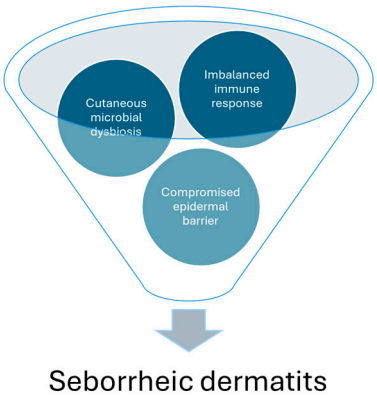

Figure 1. Main pathogenic mechanisms involved in the development of seborrheic dermatitis.

Of these factors, the role of *Malassezia* has been the most thoroughly studied, largely due to its presence in lesional skin and the positive clinical response of SD to antifungal treatments [31].

3.1. The Role of Malassezia

The human skin acts as a protective physical barrier and is also a complex microenvironmental ecosystem. Its surface is inhabited by a diverse range of microorganisms, including bacteria, archaea, viruses, and fungi, collectively referred to as the skin microbiome. The unique ecosystem of each individual is shaped by various skin niches, which vary from dry areas like the heel and volar forearm to moist areas such as the antecubital fossa and axilla, and from dry and oily regions like the face and upper back to moist and oily sites like the scalp. These variations in skin niches lead to significant microbial diversity between different body sites. The eukaryotic component of the skin microbiome is primarily dominated by *Malassezia* species, which are most abundant in sebaceous areas such as the scalp, face, chest, and upper back, while their presence is lower on the trunk and arms [32].

Malassezia fungi predominantly colonize seborrheic areas of the skin, where they utilize both saturated and unsaturated fatty acids for their growth [33]. Because they lack the genes needed to produce fatty acids themselves, *Malassezia* species rely on fatty acids supplied by the host. These fungi release enzymes that break down the lipids found on the skin's surface, producing unsaturated free fatty acids that can trigger skin inflammation.

Specifically, *Malassezia* fungi produce enzymes like lipases and phospholipases that hydrolyze triglycerides in sebum, releasing free fatty acids that can further break down into inflammatory substances, such as oleic acid and arachidonic acid.

Oleic acid harms keratinocytes provoking skin desquamation. The breakdown products of arachidonic acid can damage the outer layer of the skin, disrupt the skin barrier, and lead to abnormal skin keratinization, ultimately contributing to inflammatory skin conditions [34–39].

The strong correlation between yeast count and disease severity, as well as the observed improvement in affected skin after antifungal treatment, points to *Malassezia*'s significant role in the pathogenesis of SD. However, emerging evidence indicates that immune dysregulation and skin barrier function are likely central to SD pathogenesis, with *Malassezia* playing a secondary associated role. As suggested by Chang C.H. and Chovatiya R. [40] we should reframe our view of SD to focus primarily on the host immune system and skin epidermal barrier, similar to other types of eczema.

Malassezia overgrowth may be significant only in individuals predisposed by variations in sebaceous gland function, immune response, and lipid composition. Host factors play a key role in this model, as *Malassezia* species are commonly present on healthy skin without causing disease. In conclusion, it is well understood that while *Malassezia* may contribute to the development of seborrheic dermatitis, its presence alone is not enough to trigger the condition [41–46].

3.2. The Role of Skin Barrier

The skin barrier acts as the first line of defense against environmental insults, and its impairment is a critical factor in the pathogenesis of various forms of dermatitis. The stratum corneum, the outermost layer of the skin, serves as the primary barrier to environmental insults and water loss. It is organized in a 'brick-and-mortar' structure, where corneocytes act as 'bricks' embedded in a lipid matrix ('mortar'), composed predominantly of ceramides, cholesterol, and free fatty acids. This lipid matrix forms lamellar bilayers for maintaining barrier integrity. Beneath the stratum corneum, the stratum granulosum plays a key role in the synthesis and secretion of lipid precursors via lamellar bodies, ensuring proper lipid organization [47]. Disruptions in the composition or organization of these lipids are associated with barrier dysfunction, as observed in conditions like atopic dermatitis and psoriasis.

In atopic dermatitis, the dysfunction of the skin barrier is well documented. Studies have shown that patients with AD exhibit a significant reduction in ceramide levels and alterations in lipid composition within the SC, which correlate with increased skin permeability and susceptibility to irritants and allergens [48–50]. The role of ceramides is

particularly noteworthy, as they constitute approximately 50% of the total lipid mass in the SC and are crucial for barrier recovery following disruption [51,52]. Furthermore, the presence of inflammatory cytokines, such as interleukin-1α, is elevated in the context of barrier impairment, further exacerbating the inflammatory vicious cycle associated with dermatitis [53,54].

The role of host factors, such as barrier impairment, has been less thoroughly investigated compared to the study of *Malassezia*'s role in seborrheic dermatitis. But, as observed in other dermatological conditions like atopic dermatitis, alterations in the skin barrier also play a crucial role in the etiopathogenesis of seborrheic dermatitis.

Research has demonstrated a correlation between skin barrier dysfunction and the prevalence of seborrheic dermatitis. A study by Sanders et al. [21] highlighted that xerosis cutis, a condition indicative of compromised skin barrier function, was associated with an increased incidence of seborrheic dermatitis in a middle-aged and elderly population. This suggests that the integrity of the skin barrier is vital in preventing the onset of SD, as compromised barrier function may allow for increased irritant penetration and microbial colonization.

Individuals with seborrheic dermatitis exhibit changes in abundance and types of ceramides that compose the stratum corneum, which may compromise the structural integrity of the skin barrier, leading to hyperproliferation, abnormal keratinization, and flaking. Regarding the altered composition of ceramides, seborrheic skin appears to have an increase in Cer[NS] and Cer[AS], accompanied by a significant reduction in Cer[NdS], Cer[EOS], Cer[NP], Cer[NH], and Cer[AP] [31].

Transepidermal water loss, also observed in seborrheic dermatitis, reflects a weakened skin barrier. A chronically impaired stratum corneum should not only be viewed as a consequence of inflammation in seborrheic dermatitis but also as a key contributor to the inflammatory process itself, much like our current understanding of atopic dermatitis. In seborrheic dermatitis, an inherent disruption of the epidermal barrier may trigger the activation of the immune system and the production of cytokines. Additionally, the elevated sebum levels, which are a hallmark of seborrheic dermatitis, may result from a compromised skin barrier and excessive shedding of skin cells [55,56].

In conclusion, skin barrier impairment plays a crucial role in the development and exacerbation of seborrheic dermatitis.

The interplay between barrier dysfunction, microbial colonization, and inflammatory responses highlights the need for therapeutic strategies that not only target inflammation but also focus on restoring and maintaining skin barrier integrity [57–59]. Understanding these mechanisms is essential for developing effective treatments for seborrheic dermatitis and improving patient outcomes.

3.3. Other Factors

Many predisposing factors, beyond *Malassezia* and barrier impairment, have been linked to ASD, including (1) neurological diseases such as Alzheimer's, Parkinson's, major depression, a wide range of neurological dysfunctions, brain injuries, or spinal cord damage; (2) increased sebaceous gland activity; and (3) primary and acquired immunodeficiency, such as lymphomas, HIV/AIDS infection, or immunosuppressant treatments [60–64]. The prevalence of ASD in patients with Parkinson's disease ranges from 52% to 59%, and in HIV/AIDS patients from 34% to 83%, although a decline has been noted in the latter group since the introduction of highly active antiretroviral therapy [60]. In cases of chronic immunosuppression, as seen in organ transplant recipients or patients with HIV/AIDS, hepatitis C, alcoholic pancreatitis, and certain malignancies, the increased incidence of SD confirms the key role played by the immune system plays in the ASD etiopathogenesis. Immunohistochemical analyses of SD lesions show a shift toward a pro-inflammatory signaling environment [65].

ASD development or flare-ups have been associated with certain drugs, such as Auranofin, Fluorouracil, Griseofulvin, Haloperidol, Lithium, and Psoralen. However, it's important to note that the underlying conditions these drugs are prescribed for—rheumatoid

arthritis, various cancers, fungal infections, neurological or psychiatric disorders, bipolar disorder, and psoriasis—are themselves predisposing factors for ASD [66].

The peaks in SD incidence among three age groups (infancy between 2 weeks and 12 months, adolescence, and ages 30 to 60) and its occurrence in seborrheic areas have led to the suspicion of a pathogenic influence tied to specific environmental, microbial, and/or hormonal changes in the skin that are age-related.

Several studies have investigated and found a significant correlation between ASD incidence and severity with modifiable lifestyle factors, such as obesity, hypercholesterolemia, alcohol consumption, and metabolic diseases like hypertension and diabetes [67–69]. Notably, some of these factors (e.g., diabetes, alcohol intake) are also linked to immunosuppressive environments [70,71].

In contrast, Ozgul A. et al. [72] found no significant differences in weight ($p = 0.309$), body mass index ($p = 0.762$), fat mass ($p = 0.092$), metabolic age ($p = 0.916$), body density ($p = 0.180$), minerals ($p = 0.699$), visceral adiposity ($p = 0.401$), or protein levels ($p = 0.665$) between 39 SD patients aged 18–39 and 39 matched controls. However, the small sample size does not rule out a significant effect in a larger population.

The relationship between diet and ASD remains even more controversial. Sanders M.G.H. et al. [73] studied 636 ASD patients (14.5% of 4379 participants) and found that a dietary pattern high in fruit was associated with lower odds of SD after adjusting for confounders, while a Western dietary pattern—characterized by meat, potatoes, and alcohol consumption—was linked to higher odds of SD, but only in females. The authors suggest that the protective effect of fruit may be due to the presence of nutrients acting as methyl donors, which prevent the expression of inflammatory genes, or to the high psoralen content in citrus fruits, which increases UV sensitivity and may protect against SD.

In a study comparing 59 patients with 208 controls, daily consumption of certain foods—such as white bread ($p = 0.002$), rice or pasta ($p < 0.001$), non-acidic fruits ($p = 0.014$), leafy green vegetables ($p = 0.007$), other vegetables ($p = 0.001$), roasted or fried nuts ($p = 0.047$), raw nuts ($p = 0.022$), and coffee ($p = 0.041$)—was associated with higher rates of SD. Foods that frequently exacerbated SD included spicy foods (16.9%), sweets (16.9%), fried foods (13.5%), dairy products (11.9%), and citrus fruits (10.2%). Conversely, citrus fruits leafy green vegetables (8.5% for each) and other vegetables (6.8%) were observed to improve SD [74].

A systematic review of 13 studies—including 8 case-control, 3 cross-sectional, and 2 randomized controlled trials, involving 13,906 patients—arrived at similar conclusions: a Western diet, alcohol consumption, and obesity have negative effects, while fruit intake appears to be beneficial. Furthermore, SD was associated with significantly higher levels of copper, manganese, iron, calcium, and magnesium and lower levels of zinc and vitamins D and E, though the benefit of supplementation requires further investigation in interventional studies [70].

4. Pharmacological Treatment

Seborrheic dermatitis is a chronic skin condition with no definitive cure. The primary goal of treatment is to improve the patient's quality of life by managing symptoms and flare-ups while minimizing unnecessary and long-term side effects. Erythema, scaling, pruritus, and sebaceous gland overactivity can be alleviated by controlling inflammation and reducing the proliferation of *Malassezia* yeast. Clinical outcomes are more favorable when acute-phase treatment is followed by maintenance therapy [75–82].

The treatment of symptomatic adult SD typically involves the topical application of anti-inflammatory medications. For acute episodes, topical corticosteroids (such as betamethasone valerate: 0.12% foam, clobetasol propionate: 0.05% shampoo), systemic corticosteroids, or topical calcineurin inhibitors (pimecrolimus: 1% cream, tacrolimus: 0.1% ointment) are preferred and applied once or twice daily until symptoms resolve.

A Cochrane systematic review (36 RCTs, N = 2706) comparing topical therapies for SD in patients over 16 years old found that both potent and mild steroids were more effective

than placebo in achieving total clearance of skin lesions in short-term (relative rate of clearance = 3.8; 95% confidence interval [CI], 1.2 to 11.6) and long-term treatment (relative rate of clearance = 2.2; 95% CI, 1.1 to 4.6). Potent vs. mild steroids, steroids vs. azoles, and steroids vs. calcineurin inhibitors were equally effective at achieving total clearance, with steroids being 78% less likely to cause adverse effects than calcineurin inhibitors during short-term use (8 weeks) [83]. Despite their efficacy, the long-term use of steroids is limited by adverse effects such as skin atrophy, telangiectasia, and rebound [84].

Topical calcineurin inhibitors (CNIs) offer an alternative as they block the inflammatory cascade involved in the disease without the risk of skin atrophy. Additionally, topical phosphodiesterase 4 inhibitors, which reduce the production of pro-inflammatory mediators by decreasing cyclic adenosine monophosphate degradation, have also proven effective [85].

Systemic oral treatments for SD include itraconazole, terbinafine, fluconazole, ketoconazole, pramiconazole, prednisone, and isotretinoin. Ketoconazole (200 mg daily for 4 weeks) has been associated with more relapses compared to itraconazole (200 mg/day for the first week, followed by 200 mg/day for the first 2 days of each month from month 2 to 11) and fluconazole (50 mg/day for 2 weeks or 200–300 mg weekly for 2–4 weeks) [86].

Calcineurin inhibitors (CNIs) like cyclosporine, tacrolimus, pimecrolimus, and voclosporin are also effective in adult SD. These immunosuppressant drugs are used in various autoimmune disorders, including lupus nephritis, idiopathic inflammatory myositis, interstitial lung disease, and atopic dermatitis. CNIs bind with high affinity to specific cytoplasmic receptors (immunophilins), including cyclophilin and FK-binding proteins. By inhibiting calcineurin, these drugs prevent the transcription of interleukin-2 and other cytokines in T lymphocytes, thereby disrupting the activation, proliferation, and differentiation of T cells. Their primary effect is on T-helper cells, but they also inhibit T-suppressor and T-cytotoxic cells [87]. However, the use of CNIs is not approved in Italy for the treatment of seborrheic dermatitis due to the lack of large-scale clinical trials specifically addressing this condition and the availability of other well-established treatment options.

A comprehensive literature review and international expert consensus on the management of scalp SD in adults was recently published by S. Vano-Galvan et al. The treatment algorithm [Table 1] proposed in the study aims to help prescribers manage SD more efficiently, particularly in more severe cases where approved therapies are limited [88].

Table 1. Treatment algorithm proposed by Vano-Galvan S.; Reygagne P.; Melo D.F.; Barbosa V.; Wu W.Y.; Moneib H.; and Piraccini B.M. A comprehensive literature review and an international expert consensus on the management of scalp seborrheic dermatitis in adults [88].

Agent	Dose/Formulation	Schedule	Comments
Ketoconazole	1–2% shampoo	Twice weekly for 4 weeks	Ketoconazole 2% shampoo once weekly for 6 months has been shown to be effective in preventing relapse.
	2% foam	Twice daily for 4 weeks	Twice daily continuative use [for up to 12 months] has demonstrated high safety profile.
	2% gel	Twice weekly for 4 weeks	Fast efficacy and low rate of recurrences after discontinuation.
	2% foaming gel	Twice weekly for 1 month → once weekly for 3 months	Significant reduction in erythema and *P. orbicular* count by microbiological evaluation vs. %0.005 betamethasone dipropionate lotion.
Ciclopirox	1–1.5% shampoo 0.77% gel	3 times a week for 4 weeks Twice daily for 4 weeks	No statistically significant difference in clinical response for higher vs. lower concentrations.
Miconazole	2% solution	Once daily for 3 weeks	Miconazole 2% solution + 1% hydrocortisone solution more effective than 2% miconazole as monotherapy.

Table 1. *Cont.*

Agent	Dose/Formulation	Schedule	Comments
Betamethasone valerate	0.12% foam	Twice daily for 4 weeks	Prolonged use not recommended, due to possible side effects.
Selenium disulphide	Shampoo	2–3 per week for 4 weeks	Decreased *M.* spp. load and changed the global bacterial distribution with a notable decrease in *Staphylococci.*

In addition to pharmacological treatments, other aspects can improve the quality of life, such as psychological support, dietary recommendations and corrections when needed, physical exercise, and humidifying living and working environments. Patients should also be mindful of the dermocosmetic products they apply to their skin. Dermocosmetic treatments are part of SD management and can help maintain the results of pharmaceutical treatment, acting as complementary therapies.

5. Dermocosmetic Treatment

Dermocosmetics have now gained recognized scientific support for their role in the treatment of various dermatological conditions, including seborrheic dermatitis.

In the acute phase of this condition, dermocosmetics can complement and support the action of pharmaceuticals. During remission, they help maintain the skin's microbiological balance and barrier function, preventing flare-ups. In mild cases, dermocosmetics can manage symptoms, improve skin appearance, and consequently enhance the individual's overall well-being.

The role of dermocosmetics has been first recognized for the treatment of atopic dermatitis. According to EADV guidelines 2020 for atopic dermatitis [89], the basic therapy must be selected and planned with a long-term perspective and special attention to long-term safety aspects. Basic therapy includes hydrating and barrier-stabilizing topical treatment, as well as avoiding specific and unspecific provocation factors. External factors also include the choice of the most appropriate detergent. When there is a dermatological pathology, the patient can tend to focus mainly on the treatment, putting cleansing in the background. However, cleansing plays a pivotal role in the management of skin disease. A key aspect of washing for skin conditions is the avoidance of harsh cleansers and alkaline soaps, which can compromise the skin barrier by stripping away ceramides and other essential epidermal lipids.

It is well-established that harsh surfactants in cleansers can harm skin proteins and lipids, resulting in post-wash tightness, dryness, barrier disruption, irritation, and even itching. For cleansers to offer skincare benefits, they must first reduce the damage caused by surfactants to skin proteins and lipids. Additionally, they should deposit and deliver beneficial ingredients, such as occlusive agents, skin lipids, and humectants, during washing to enhance skin hydration, as well as improve their mechanical and visual qualities. For individuals with skin diseases, oil-based cleansers that work by affinity are often the most suitable choice. These cleansers remove impurities by attracting and dissolving oils and dirt. Since affinity cleansers are generally less appreciated by patients than foaming ones, it would be ideal if the choice of cleanser was made considering the patient's preferences. In some cases, it is possible to use contrast cleansers if they contain a well-balanced and skin-friendly blend of surfactants. Although all surfactants generally interact with lipids to some extent, their interaction with proteins can vary widely, depending on the characteristics of their functional head group [90–93].

For dermocosmetic treatment, although some medicinal plants, such as *Myrtus communis*, *Vitis vinifera*, *Hypericum perforatum*, etc., have the potential to treat seborrheic dermatitis [94], the recommendation for skin diseases is to use the simplest formulation with the smallest number of ingredients.

The goal of dermocosmetic treatment for seborrheic dermatitis is to control *Malassezia*, mitigate inflammation, and restore the skin's barrier function [95–97]. It includes both shampoos and lotions for the scalp, and emulsions for wide skin areas.

The actives used for cosmetic purposes in seborrheic dermatitis can be divided into three main categories: antifungals, shooting agents, and restorative actives of the skin barrier.

As antifungals, the most widely used actives in cosmetics are climbazole and piroctone olamine. Climbazole is a highly active molecule against *Malassezia*, as it can destroy their cell membranes, leading to their death. Piroctone olamine is equally effective against fungi: its action against these microorganisms is linked to its ability to penetrate the fungal cell wall and form complexes with iron ions, leading to the functional blockade of mitochondria, which are essential energy centers for cellular vitality.

In recent years, the use of climbazole in cosmetics has been restricted by European regulations to ensure consumer safety [98]. The maximum allowed concentration of climbazole is now limited to 0.5% in rinse-off products like anti-dandruff shampoos and 0.2% in leave-on products like face or body creams. These restrictions were introduced to minimize the risk of skin irritation and cumulative exposure, especially when using multiple products containing climbazole. Additionally, its use is prohibited in products intended for children due to concerns about increased skin absorption in younger individuals.

For these reasons, the cosmetic industry has started to evaluate different approaches for the containment of *Malassezia* fungi. Recently, on the market, a new approach to antifungal treatment has been proposed using the enzyme inhibition strategy [99].

For example, propanediol caprylate is an ester that, like the triglycerides found in sebum, can be metabolized by *Malassezia* lipases. Its degradation produces two byproducts: propanediol and caprylic acid. The latter is toxic to *Malassezia* itself.

Among the soothing active ingredients, glycyrrhetic acid, derived from licorice root, is one of the most used products for the treatment of seborrheic dermatitis. In addition to its anti-inflammatory-like effects, it also possesses antioxidant and antimicrobial activity. In addition to glycyrrhetic acid, bisabolol, allantoin, panthenol, and inositol are also widely used as soothing active ingredients.

Endocannabinoids are another class of substances that can be used in cosmetics for their soothing properties [100,101]. Endocannabinoids are, in fact, involved in the regulation of various biological processes, such as the growth and differentiation of skin cells and the production of mediators by these cells. In the presence of inflammatory skin diseases (such as atopic dermatitis or allergic contact dermatitis), the body increases the synthesis of endocannabinoids as endogenous protective agents. Endocannabinoids exert similar anti-inflammatory action in two ways: on the one hand, they modulate the production of cytokines by skin cells, and on the other, they act directly on the cells of the immune system present in the skin, through binding to CB1 and CB2 receptors. For this reason, it is believed that endocannabinoids and their receptors are part of the adaptive immune system involved in the regulation of skin inflammation.

Another molecule used to soothe the skin barrier is phosphatidylglycerol, which is increasingly used in cosmetic products since it has been shown to suppress skin inflammation by inhibiting toll-like receptors (TLR) activation induced by microorganisms and their metabolites in psoriatic patients [102]. By reducing irritation and inflammation, this phospholipid helps restore the skin's natural lipid layer.

Finally, the actives involved in barrier repair play a pivotal role in the dermocosmetic treatment of seborrheic dermatitis.

As previously mentioned, a common characteristic of individuals with seborrheic dermatitis is an alteration of the skin barrier and its fundamental lipids. An altered skin barrier promotes inflammation, which in turn contributes to further damage to the skin barrier. Thus, a vicious cycle is established, leading to a worsening of the disease symptoms. Therefore, in both severe and mild forms of seborrheic dermatitis, it is important to apply a product containing active ingredients that can restore the skin barrier [103–105].

Ceramides are the most effective active ingredients for repairing the skin barrier, as they represent the mortar of the skin barrier. The application of products containing ceramides has proved to be effective in the treatment of other skin conditions such as atopic dermatitis and psoriasis, for which the involvement of the skin barrier in the pathogenesis is well-established [106–108].

6. Conclusions

In conclusion, seborrheic dermatitis, like other chronic dermatitis conditions, exhibits a relapsing course that requires long-term management focused on symptom control and prevention of flare-ups. Although the central role of *Malassezia* in its pathogenesis is well established, it is crucial to recognize the contribution of individual susceptibility and skin barrier dysfunction. Treatment strategies, especially in the dermocosmetic domain, must consider these factors, emphasizing the importance of barrier repair agents. In this perspective, future therapeutic approaches should aim not only at controlling microbial colonization but also at restoring skin barrier integrity to prevent recurrences and improve patient outcomes.

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