Medical Science

To Cite:
Urbańska J, Borowska D, Nieczaj K, Sztubińska J, Marciniak MU, Tortyna M, Sioch N, Krotofil J, Szarek P, Samsel O, Trzciński M. Seborrheic dermatitis current information on diagnosis and treatment: A literature review. *Medical Science* 2025; 29: e158ms3624
doi: https://doi.org/10.54905/disssi.v29i163.e158ms3624

Authors' Affiliation:
[1]Faculty of Medicine, Medical University of Gdańsk, M. Skłodowskiej-Curie 3a street, 80-210 Gdańsk, Poland
[2]Faculty of Medicine and Health Sciences, Collegium Medicum of the University of Zielona Góra, 28 Zyta Street, 65-046 Zielona Góra, Poland

***Corresponding author:**
Julia Urbańska,
Faculty of Medicine, Medical University of Gdańsk, M. Skłodowskiej-Curie 3a street, 80-210 Gdańsk, Poland, E-mail: juula522@gamil.com

Contact list

Julia Urbańska	juula522@gmail.com
Danuta Borowska	d_borowska@gumed.edu.pl
Kamil Nieczaj	kamilnieczaj22@gmail.com
Julia Sztubińska	juliasztubińska@wp.pl
Marta Urszula Marciniak	marta.marciniak2707@gmail.com
Marta Tortyna	marta.tortyna@gmail.com
Natalia Sioch	sioch.natalia@gmail.com
Julia Krotofil	krotofil.julia@gmail.com
Paula Szarek	paula.szarek.1@gmail.com
Olga Samsel	olga.samsel.13@gmail.com
Maciej Trzciński	maciejt99@interia.pl

ORCID list

Julia Urbańska	0009-0007-8897-7877
Danuta Borowska	0009-0002-0136-0182
Kamil Nieczaj	0009-0001-3812-8523
Julia Sztubińska	0009-0005-2503-1158
Marta Urszula Marciniak	0009-0000-2409-4935
Marta Tortyna	0009-0002-8230-6929
Natalia Sioch	0009-0001-4812-6206
Julia Krotofil	0009-0009-8309-4615
Paula Szarek	0009-0002-1102-5783
Olga Samsel	0009-0008-5291-7570
Maciej Trzciński	0009-0008-2514-3493

Peer-Review History
Received: 05 June 2025
Reviewed & Revised: 18/June/2025 to 23/August/2025
Accepted: 30 August 2025
Published: 09 September 2025

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

DISCOVERY SCIENTIFIC SOCIETY

Seborrheic dermatitis current information on diagnosis and treatment: A literature review

Julia Urbańska[1]*, Danuta Borowska[1], Kamil Nieczaj[1], Julia Sztubińska[1], Marta Urszula Marciniak[1], Marta Tortyna[1], Natalia Sioch[1], Julia Krotofil[1], Paula Szarek[2], Olga Samsel[1], Maciej Trzciński[1]

ABSTRACT

Seborrheic dermatitis (SD) is inflammatory disease which is chronic and relapsing. It primarily affects areas with abundant sebaceous glands. It most often occurs on the scalp, face, and upper trunk. SD most commonly occurs in infants and adults aged 30 to 60. Pathogenesis encompasses various aspects. The disease is of multifactorial etiology, with Malassezia proliferation, immunologic response of the host, sebaceous gland activity, and environmental factors playing a role. A review of the current state of knowledge in diagnosis and treatment of SD aimed to appraise related studies. Clinical evaluation is what the diagnosis depends on, and occasionally dermatoscopic examination as well as histopathological analysis does also. Treatment involves topical antifungal medications, corticosteroids, and other medications. In some cases, phototherapy is used. In cases of resistance to recurrent symptoms, recent studies indicate the potential usefulness of microbiome-modulating therapies and systemic pharmacologic interventions. A comprehensive understanding of the basic mechanisms of the disease about to available treatment methods is crucial for effective and individualised patient management.

Keywords: seborrheic dermatitis, Malassezia, antifungal therapy, topical treatments, skin inflammation

1. INTRODUCTION

SD is a chronic inflammatory skin disease characterised by erythematous patches with oily, yellowish scales, mainly affecting areas rich in sebaceous glands, such as the scalp, face, and upper body (Plewig et al., 2008). The condition is common, affecting 4,38% (Polaskey et al., 2024) of the population, and may occur in up to 70% of infants during the first three months of life. In the adult population, seborrheic dermatitis most often appears in the third and fourth decades of life (Berk & Scheinfeld, 2010). The condition is strongly associated with colonisation by Malassezia yeasts, which play a key role in pathogenic mechanisms (Gaitanis et al., 2012). Although the exact cause of the condition is not fully understood, its be associated with a combination of genetic predisposition, environmental influences

and microbial activity (Tucker & Masood, 2024). Seborrheic dermatitis is clinically meaningful not only because of its chronic and relapsing course, but also due to the psychosocial burden it may impose. In addition, SD is associated with various systemic diseases, in particular human immunodeficiency virus (HIV) (Perez et al., 2025), Parkinson's disease (Tomic et al., 2022), and Down syndrome (Rork et al., 2020).

Although seborrheic dermatitis is a chronic condition that cannot be cured entirely. Clinical symptoms can usually be effectively treated with appropriate therapeutic measures. Given association with systemic conditions, thorough clinical work-up is required for appropriate patient management. Closer attention is called upon to the development of further research that can better explain the pathogenic mechanisms involved and could lead to more targeted and effective treatment.

2. REVIEW METHODS

We collected this literature survey from the beginning of April 2025 to the end of May 2025. We used the PubMed and Google Scholar databases. The following keywords were used in the search strategy: seborrheic dermatitis, diagnosis, treatment, Malassezia, topical therapy, and systemic therapy. Inclusion criteria encompassed: articles published in English; studies published between January 2010 and May 2025; randomized controlled trials, systematic reviews, meta-analyses, and high-quality cohort studies; studies focusing on adult and/or paediatric population. Exclusion criteria included: non-peer-reviewed publications; case reports and editorials; studies with incomplete data or lacking full-text access.

The selection process was conducted in accordance with PRISMA guidelines (Figure 1). Initially, 186 articles were selected. After removing duplicates and reviewing abstracts and full texts, 48 articles were included in the final review. Reasons for exclusion included irrelevance of the topic, low methodological quality, or duplication of content.

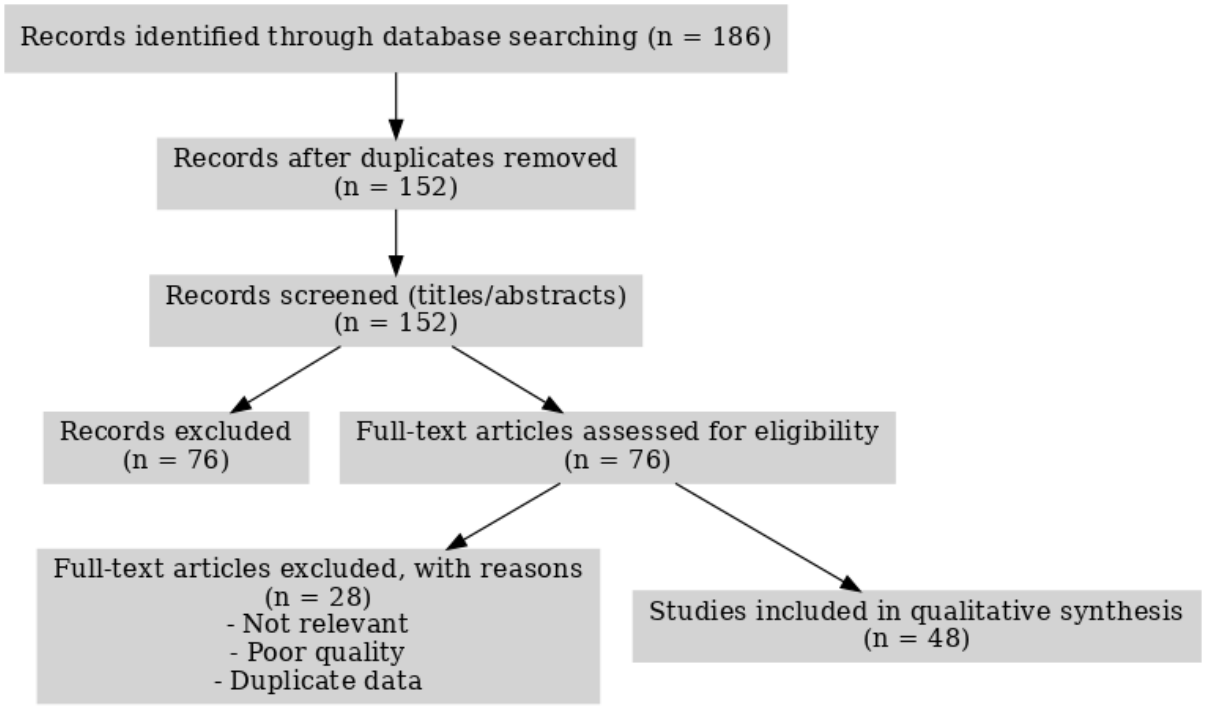

Figure 1. PRISMA diagram showing the article selection process.

3. RESULTS AND DISCUSSION

The most important features and results of selected studies on SD treatment are summarised in Table 1.

Table 1. Summary of selected studies included in the review.

Author (Year)	Country	Study Type	Sample Size	Main Findings
Roussel et al., 2023	Netherlands	RCT	90	Ketoconazole effective for facial SD vs placebo
Goldust et al., 2013	Iran	RCT	70	Sertaconazole 2% comparable to tacrolimus and pimecrolimus
Azizzadeh et al., 2022	Iran	RCT	80	Sertaconazole 2% = pimecrolimus 1% in reducing SD symptoms
Braza et al., 2003	USA	Open-label study	20	Tacrolimus 0.1% effective and well-tolerated
Papp et al., 2012	Canada	RCT	66	Tacrolimus 0.1% vs hydrocortisone 1% equally effective
Kim et al., 2013	Korea	Double-blind trial	80	Proactive tacrolimus reduced SD relapses
Wananukul et al., 2012	Thailand	RCT	54	Hydrocortisone 1% effective in infants; comparable to licochalcone
Emtestam et al., 2012	Sweden	RCT	90	Urea + lactic acid + propylene glycol effective vs placebo
Zirwas et al., 2023	USA	RCT	226	Roflumilast foam 0.3% highly effective in SD treatment
Miller et al., 2025	USA	Review	-	Roflumilast offers rapid symptom relief with low side effects
Barbosa et al., 2024	Brazil	RCT	100	1% selenium disulfide comparable to 2% ketoconazole shampoo
Miri et al., 2025	Iran	RCT	60	5% atorvastatin shampoo more effective than placebo
Kose et al., 2005	Turkey	Open-label trial	30	Oral itraconazole effective in SD management
Ghodsi et al., 2015	Iran	RCT	60	Itraconazole effective for control and relapse prevention
Zisova (2006)	Bulgaria	RCT	40	Fluconazole 150–300 mg/week improved moderate-severe SD
de Souza Leão Kamamoto et al., 2017	Brazil	RCT	52	Low-dose isotretinoin effective for moderate-severe SD
Demirbaş et al., 2025	Turkey	RCT	70	20 mg isotretinoin had lower recurrence than 10 mg
Lax et al., 2024	Global	Meta-analysis	-	Topical ruxolitinib effective, especially in sensitive areas
Sahoo et al., 2025	India	Case series	6	Oral tofacitinib improved severe SD, well tolerated
Perez et al., 2025	USA	Review	-	Botulinum toxin may reduce scalp inflammation in SD

Wang et al., 2024	China	Split-face RCT	40	IPL-PDT reduced sebum secretion and erythema
Liu et al., 2025	China	Review	-	Chitosan shows antifungal and moisturizing properties in SD

Pathogenesis and Risk Factors

The development of SD is caused by a mix of factors, like genetic susceptibility, immune system involvement, and colonization by Malassezia yeasts. The interaction between these elements accelerates epidermal renewal and inflammatory reactions, which contribute to the clinical symptoms observed in people affected by this disease. (Trznadel-Grodzka et al., 2012).

Because they are lipophilic and respond to the body's immune system, and Malassezia species include a large variety of types of yeast that exist in different environments, they can provide not only infection but also headaches and fever when they become predominant in one part of a host's flora. The yeast strains themselves also cause or exacerbate inflammation, partly because their metabolic byproducts are inflammatory agents and partly through direct interaction with the skin barrier. Although Malassezia species are a natural component of the skin microbiota, they can become pathogenic under certain conditions, contributing to inflammation and the development of lesions characteristic of seborrheic dermatitis. Malassezia spp. trigger an inflammatory response, which is central to the pathogenesis of SD (Nakabayashi et al., 2000). Malassezia spp. stimulate both innate and adaptive immune responses. The strength and type of this response depend on the yeast's virulence and the sensitivity of the host's immune system (Dessinioti and Katsambas, 2013). Recent studies show that hyphal forms of Malassezia are associated with a greater severity of clinical symptoms of SD (Li et al., 2022). Malassezia globosa and Malassezia restricta are most frequently isolated from patients with SD, with M. globosa having a particularly high prevalence (Dawson, 2007). These species tend to colonize sebaceous gland-rich regions, such as the scalp and facial skin, consistent with the typical distribution of seborrheic dermatitis lesions (Wikramanayake et al., 2019). The therapeutic efficacy of antifungal agents, in in particular ketoconazole, further confirms the involvement of Malassezia in the disease process, as clinical improvement is commonly observed after treatment (Zani et al., 2016). However, the just presence of Malassezia isn't enough cause disease, as because healthy people often have it without any symptoms, which shows that we need to think about other stuff relates to the host and environment in how the disease happens. Other factors may also influence the development of the disease, including the composition of lipids on the skin surface and individual characteristics of the immune system (Picardo and Cameli, 2008).

Host immune response and genetic predisposition

Genetic predisposition and host immune response shape the pathogenesis of SD. Recent studies have aimed to explain the immunogenetic basis of SD using advanced methods of statistical genetics. It was decided to investigate the causal relationship between different immune cell phenotypes and the risk of developing SD (Xian et al., 2025). The findings identified several immune traits with either protective or pathogenic associations. Five immune phenotypes were significantly associated with a reduced risk of SD, indicating a potentially immunomodulatory or protective function:

-CD24 transitional,
- CD28 − DN(CD4 − CD8−)%T cell,
- CD45RA − CD28 − CD8br AC,
- Resting Treg%CD4,
- SSC − A on NKT.

Conversely, six immune phenotypes demonstrated a positive association with increased SD risk, suggesting a possible pro-inflammatory or pathogenic role:
- CD127 − CD8br AC,
- CD20on CD24 + CD27+,
- CD27on IgD − CD38br,
- CD27on unsw mem,
- CD28 + DN(CD4 − CD8−)%T cell,
- HLA DR on CD33br HLA DR + CD14dim.

The results of this study highlight the immunological complexity underlying SD and draw attention to the key role of different immune cell subsets in increasing susceptibility to the disease or providing protection against it. These findings may aid future research into the development of targeted therapeutic strategies based on immunomodulation.

Studies have identified mutations, such as in the ZNF750 gene, that correlate with SD, suggesting a hereditary component to the disease (Birnbaum et al., 2006). Experimental studies using mouse models with the Mpzl3 gene knocked out have provided valuable information on the genetic basis of dermatological changes very similar to SD, thus confirming the influence of hereditary factors on the pathogenesis of the disease (Wikramanayake et al., 2017). At the same time, non-genetic influences such as psychological stress, temperature fluctuations, and hormonal shifts are also known to aggravate the condition, further supporting its multifactorial etiology (Dessinioti & Katsambas, 2013).

A diet high in simple carbohydrates (white bread, rice, pasta) was significantly associated with seborrheic dermatitis (SD). Daily consumption of leafy green vegetables, non-acidic fruits, nuts, and coffee also correlated with a higher prevalence of SD. The use of butter for frying and the consumption of meat with visible fat increased the likelihood of having SD. Individuals with SD had a higher intake of vitamin D and a lower intake of iron. Many individuals reported that spicy foods, sweets, fried foods, dairy products, and citrus fruits worsened their symptoms (Alshaebi et al., 2023).

In a study (Sanders et al., 2019), high fruit consumption was inversely proportional to the risk of SD, while a Western diet rich in meat, potatoes and alcohol was associated with a higher risk. No significant associations were found at all for diets high in vegetables, fats, or overall antioxidant capacity (FRAP score). SD is associated with various systemic and metabolic conditions. According to research (Kulakli et al., 2024), patients with SD are more prone to early osteoarthritis, experience greater knee pain and show increased thickness of the femoral cartilage. In Parkinson's disease, seborrheic dermatitis affects 52–59% of patients and is more strongly associated with the akinetic-rigid phenotype than with the tremor-predominant form (Wilkowski et al., 2025). Although seborrheic dermatitis patients are also common sufferers of diabetes, hypertension, and obesity, there have been no consistent relationships demonstrated in studies. There are no significant body composition differences. An exception is the visceral protein level is positively and height negatively associated with SD severity (Kluglein et al., 2024).

Clinical Presentation and Diagnosis

Seborrheic dermatitis (SD) is a chronic, recurrent inflammatory skin disease that can affect anyone at any age. It is characterized by red, scaly patches covered with yellowish, oily scales. It most often appears in areas with a high number of sebaceous glands – on the scalp, face, behind the ears, on the front of the chest, and on the upper back. Symptoms are age-dependent. Babies often suffer from "cradle cap," presenting with yellow, sticky scales on the scalp. Adolescent and adult patients typically have erythematous, greasy plaques. Additionally, fine yellow scales and itching may occur.

Skin manifestations of SD are common, and scalp involvement is one of the most frequent. Adults who have scalp involvement are likely to suffer from what is known as Pityriasis simplex; this condition is called cradle cap in infants. The face is another frequent location for SD attacks. Lesions around the ears can lead to complications such as otitis. The condition may also extend to the chest and back. SD can be challenging to differentiate from other skin conditions such as psoriasis, atopic dermatitis, tinea capitis, candidiasis, contact dermatitis, erythrasma, impetigo, lichen simplex chronicus, nummular dermatitis, pityriasis rosea, rosacea, secondary syphilis, systemic lupus erythematosus, and tinea corporis (Clark et al., 2015).

Skin diseases may appear different both in appearance and on histopathologic examination. It's important to be able to diagnose them skillfully. The primary investigation for SD is clinical, with an emphasis on the lesion's morphology and its anatomical location. Physical examination is the cornerstone of diagnosis since no markers are available and no laboratory tests are performed as routine that can confirm the diagnosis. A thorough patient history, including past skin diseases such as dermatitis, and also data from family members that suggest susceptibility to such conditions, provides additional confirmation of diagnosis and enables the clinician to refine their initial impression. In cases where the diagnosis is unclear, dermatoscopy can be used for diagnosis. This can reveal branching blood vessels, unusual red vascular structures, structureless areas, and honeycomb-like discoloration (Xu et al., 2014). When non-invasive methods are inconclusive, histopathological examination can be used. In seborrheic dermatitis, standard features include spongiosis, inflammatory infiltrates, follicular plugging, shoulder parakeratosis, and prominent lymphocytic exocytosis (Park et al., 2016).

Although biopsy can be helpful in atypical or refractory cases, most diagnoses can be confidently made based on clinical presentation alone, avoiding the need for invasive procedures. The Seborrheic Dermatitis Area and Severity Index (SDASI) measures

the severity of the condition. By utilizing a standardized method, it assesses both the extent and intensity of skin lesions, which can direct therapy decisions and keep track over time of how diseased tissue is progressing. This index has been frequently used in clinical trials to objectively assess the severity of the disease and to examine its association with various clinical and psychosocial features (Cömert et al., 2007; Emre et al., 2012). Studies show that a higher SDASI score is associated with higher stress levels, suggesting the influence of psychological factors on the development and exacerbation of SD (Sarac and Kocatürk, 2022).

The SDASI scale assesses disease severity in nine common locations: the forehead, scalp, nasolabial folds, eyebrows, behind the ears, on the auricles, between the breasts, on the upper back, and on the cheeks or chin. For each location, it measures redness, scaling, and extent of lesions, allowing for a more accurate assessment of the disease. In each region, the three key symptoms of scaling, erythema, and pruritus are separately evaluated on a scale from none (0) to severe (3). To reflect the clinical relevance of each region, multiply the total score for each area by a specific weighting factor: forehead (0.1), scalp (0.4), nasolabial folds (0.1), eyebrows (0.1), postauricular area (0.1), auricles (0.1), intermammary region (0.2), back (0.2), and cheek or chin (0.1). Add the weighted values for the entire body together, and you can calculate your total SDASI Score. SDASI allows for a more nuanced and easy-to-use manner of categorizing the extent and severity of disease. It is also helpful for assessing the patient's condition and the effectiveness of treatment in clinical and research trials (Sarac and Kocatürk, 2022).

Treatment Options – Current Evidence

Seborrheic dermatitis is a chronic, relapsing inflammatory skin condition that requires long-term management. Although seborrheic dermatitis is a chronic and long-term disease for which there is no effective cure, current therapeutic strategies focus on relieving symptoms and reducing the frequency and severity of flare-ups. The first line of treatment usually involves the use of topical antifungal agents, such as ketoconazole or ciclopirox, which combat Malassezia spp. colonisation, together with topical corticosteroids or calcineurin inhibitors to suppress skin inflammation, especially in anatomically sensitive areas such as the face.

Keratolytic agents, including salicylic acid and selenium sulfide, are often used to reduce excessive keratinisation and improve the clinical appearance of lesions. In cases characterised by moderate to severe disease or disease resistant to treatment, systemic antifungal therapy, such as oral itraconazole, may be indicated. Treatment is selected individually, taking into account the severity of the disease, the location of the lesions, response to previous therapies and other patient-related factors.

Topical Therapies

The main goals of topical therapy include reducing inflammation, limiting the spread of fungi, and alleviating symptoms of itching and skin peeling. The following sections summarise the main types of local treatment methods used in SD therapy.

Antifungals

Malassezia spp., which plays an important role in the progression of the disease, is not eliminated by topical antifungals at all. In a randomized, controlled trial, ketoconazole in 2% cream successfully treated mild to moderate facial seborrheic dermatitis. After two weeks of twice-daily treatments, it changed 50% of subjects' scores for erythema and scaling from high to low—half what had been achieved by placebo (Roussel et al., 2023). Thus, ketoconazole has become the first-line treatment on these grounds of both safety and efficacy.

Ciclopirox olamine has been confirmed in a number of clinical trials, that it is effective in treating fungal infections and alleviating symptoms caused by them. Therefore, it proves to be a reliable therapy as well. Miconazole is another commonly used for moderate infections. Sertaconazole 2% cream has demonstrated high efficacy and safety in the treatment of facial seborrheic dermatitis. Randomized clinical trials have shown that sertaconazole cream 2% has comparable efficacy to tacrolimus 0.03% and pimecrolimus 1% in reducing the main clinical symptoms of SD, including erythema, scaling, and itching, with a favourable safety and tolerability profile (Goldust et al., 2013). Similarly, in a double-blind study in which the evaluators were unaware of the results, sertaconazole was found to be as effective as 1% pimecrolimus, and both agents produced statistically significant clinical improvement (Azizzadeh et al., 2022). These results confirm that sertaconazole can be used not only as an effective topical antifungal agent but also as a compound with anti-inflammatory properties, producing therapeutic results comparable to those obtained with topical calcineurin inhibitors, especially in the case of facial skin.

Corticosteroids

The standard treatment for SD is mild-to-moderate topical corticosteroids (e.g., hydrocortisone 1 percent), which decrease itching and inflammation. A short course of hydrocortisone 1% reduces redness and scaling quickly, especially during flares; its effectiveness has been confirmed by clinical studies in adult populations (Goldust et al., 2013; Papp et al., 2012). However, due to potential adverse effects associated with long-term use of corticosteroids, particularly in delicate areas of the face, alternative agents such as sertaconazole and tacrolimus may offer more favourable safety and efficacy profile in long term. In infants, researchers found that hydrocortisone was as effective as a moisturizer containing licochalcone, supporting its use even in paediatric cases. However, clinicians may opt for milder alternatives when treating sensitive skin (Wananukul et al., 2012).

Calcineurin inhibitors

Topical calcineurin inhibitors, such as pimecrolimus, are effective in managing inflammation and offer a lower risk of side effects compared to corticosteroids. Tacrolimus 0.1% ointment has proven effective and well-tolerated in treating facial seborrheic dermatitis. An open-label study (Braza et al., 2003) showed rapid symptom improvement with reasonable safety. A randomized trial (Papp et al., 2012) found tacrolimus to be effective as hydrocortisone 1%, but without the risk of skin atrophy. A double-masked study demonstrated that the proactive use of topical treatment twice weekly significantly reduced relapse rates in patients with seborrheic dermatitis (Kim et al., 2013). Studies confirm that tacrolimus is safe and reduces the need for steroids, both at the beginning of treatment and later in the course. A 1% pimecrolimus cream has also been shown to be effective and well-tolerated in the treatment of facial SD – it significantly reduced redness, scaling, and itching, while also being safe (Goldust et al., 2013; Azizzadeh et al., 2022). Because it is non-steroidal and carries a low risk of thinning the skin, it is particularly suitable for long-term use on delicate areas such as the face.

Salicylic acid and propylene glycol

Salicylic acid reduces desquamation, and propylene glycol helps moisturize the skin; together, they improve treatment outcomes. Salicylic acid helps exfoliate the skin and facilitates the absorption of corticosteroids. This approach works best at the beginning of treatment, before moving on to maintenance therapy (Massiot et al., 2023). A topical solution of urea, lactic acid, and propylene glycol (K301) significantly reduces skin scaling, erythema, and itching compared to placebo (Emtestam et al., 2012). The treatment was well tolerated and effective, providing keratolytic and moisturizing benefits as a non-steroidal option for symptom relief.

PDE-4 inhibitors

Roflumilast 0.3% foam, a selective phosphodiesterase-4 (PDE-4) inhibitor, represents the first novel therapeutic class approved for the treatment of seborrheic dermatitis in over two decades (Zirwas et al., 2023; Wang et al., 2025). It offers potent anti-inflammatory effects in a non-steroidal, alcohol- and fragrance-free, water-based formulation, suitable even for sensitive areas like the eyelids and genital region. The non-greasy foam formulation exhibits favourable tolerability and is particularly suitable for application on the scalp. Clinical studies show that roflumilast acts rapidly and maintains its effectiveness over time. Investigator's Global Assessment (IGA) scores improved in 80% of patients, and itching significantly decreased after just 48 hours. As a drug administered once a day, roflumilast has few side effects and is well-tolerated. For patients who have not responded well to first-line treatment, it is a safe and efficacious option (Miller et al., 2025).

Medicinal shampoos

Recent studies have shown that shampoos containing selenium sulphide or ketoconazole are effective in treating scalp dandruff. As these preparations have antifungal properties and cause peeling of the scalp skin, they then also help the condition of that area to improve. Shampoos with 1% selenium disulfide and 2% ketoconazole have similar effects, although ketoconazole has a faster onset (Barbosa et al., 2024). A combination of selenium disulfide and salicylic acid was particularly effective in patients with many scaly scalps (Wang et al., 2025). 5% Atorvastatin shampoo was more superior than a placebo in improving symptoms, probably due to its anti-inflammatory effects (Miri et al., 2025). While most patients benefit from topical treatment, some have persistent, widespread, or refractory symptoms and require systemic therapy or other interventions.

Systemic therapies

Systemic treatment is usually reserved for severe, extensive or resistant forms of SD, and its therapeutic goal is to combat both inflammatory pathways and excessive growth Malassezia yeasts.

Oral antifungals

Systemic antifungal medications such as itraconazole and fluconazole can effectively treat moderate, severe, or refractory SD. They are particularly helpful for those patients who do not respond to topical therapy. Treatment regimens are tailored to patients' needs and require careful monitoring during long-term use because of the risk of liver damage, etc.

In an open-label trial, significant improvement was reported using itraconazole 100 mg twice daily for 1 week, followed by 100 mg once daily for 1 day per week over the next 2 months (Kose et al., 2005). A randomized, placebo-controlled study confirmed the efficacy of itraconazole in both symptom control and relapse prevention, using a regimen of 100 mg/day for 2 weeks, followed by 100 mg/day for two consecutive days each month over a 3-month maintenance period (Ghodsi et al., 2015). Oral fluconazole has shown promise as a systemic treatment for moderate to severe or treatment-resistant seborrheic dermatitis. Fluconazole at a dose of 300 mg once weekly for 2–4 weeks brought clinical improvement, emphasizing its dual antifungal and anti-inflammatory effects (Zisova, 2006). A randomised placebo-controlled study demonstrated the efficacy of fluconazole administered at a dose of 150 mg once a week for two weeks, resulting in a significant reductions in erythema, skin peeling and pruritus compared to placebo.

These results support the role of fluconazole as a practical, short-term systemic treatment option in selected patients with SD, particularly in cases refractory to topical therapy. Further studies are essential to determine the optimal dosing regimen and assess long-term safety and tolerability.

Isotretinoin

Oral isotretinoin has shown much promise in the treatment of moderate to severe acne vulgaris. Moreover, low doses of isotretinoin were associated with significant clinical improvement in a randomized clinical study (de Souza Leão Kamamoto et al., 2017). A recent study tested 10 and 20 mg daily doses for efficacy and safety (Demirbaş et al., 2025). Both doses worked well and were well tolerated. The 20 mg dose reduced the number of relapses, suggesting possibly less or perhaps greater protection against disease recurrence.

Upcoming Therapies and Innovations

Current SD treatment strategies seek to both eliminate the cause of the disease and restore the dermis's ability to support life.

Development of New Topical Therapies

The topical treatment ruxolitinib, a Janus kinase (JAK) inhibitor, is one of the most promising. It dramatically reduces inflammation, erythema, and itching in SD patients after several weeks (Lax et al., 2024). A recent network meta-analysis placed ruxolitinib at the top of systemic anti-inflammatory drugs. It also has a good safety profile and is easy to take, making it a viable option for people who may benefit from nonsteroidal medications in the long run, especially those with sensitive skin or those who request long-term treatment.

Development of New Systemic Therapies

Another JAK inhibitor, oral tofacitinib, was administered to adults with severe and very severe SD in a recent case series (Sahoo et al., 2025). Patients' erythema, scaling, and itching noticeably improved. The treatment was well tolerated and produced no serious side effects. These preliminary results suggest that tofacitinib may be a good systemic treatment option for patients with hard-to-treat disease. But larger, controlled studies are needed to confirm its effectiveness and safety.

Botulinum Toxin

Botulinum toxin is becoming more popular as an adjunct therapy for SD, especially when the scalp is affected. This drug probably reduces skin inflammation and sebum production. Studies indicate that botulinum toxin injections might suppress the function of the sebaceous glands, and thereby reduce redness and scaling (Perez et al., 2025). Although evidence is still sketchy, its effect on sebum production suggests potential application to patients manifesting hard-to-treat symptoms. But further research is needed to establish the optimal dosage and effectiveness.

Light-based and Photodynamic Therapy

A clinical study examined the effect of combining intense pulsed light with topical photodynamic therapy (IPL-PDT) on the treatment of seborrhea (Wang et al., 2024). This treatment significantly lowered sebum output and reduced redness of the skin. Patients tolerated it well, and no serious side effects were noted. The results suggest that IPL-PDT may be a helpful adjunct therapy, especially for patients who do not respond to standard treatment.

Microbiome modulation

Chitosan, a biocompatible polysaccharide, shows potential in the treatment of SD due to its anti-inflammatory, antibacterial, and moisturizing properties. It appears to inhibit the growth of Malassezia spp., a key pathogen responsible for this disease, while improving skin barrier repair and hydration (Liu et al., 2025). Chitosan is used as an active ingredient in topical medications to help with inflammation, flaking, and itching. It's very safe to use. However, whether it is actually effective requires more clinical trials to prove.

Additional Investigational Approaches

Some new treatments for SD, such as cannabinoids and various Vitamin D derivatives, are currently being explored to see if they are beneficial. Although in severe cases systemic therapy may be life-saving, careful patient selection is essential, especially for therapy; possible adverse reactions must be carefully monitored. Many patients successfully control their disease just by keeping it local, underlining the need for an individual path to treatment that takes the disease's severity into account.

Long-term management and prevention of recurrence

SD is a chronic and recurrent inflammatory skin disease that most often affects areas with a large number of sebaceous glands. Effective long-term treatment requires not only suppression of clinical symptoms and reduction in the frequency of relapses, but also constant control of key pathogenic factors, including Malassezia yeast proliferation and skin inflammation. A comprehensive treatment strategy typically combines pharmacological therapy, lifestyle changes, and ongoing patient education.

Lifestyle factors can play a helpful role in controlling the disease. Changes in eating habits and the use of stress reduction are associated with improved symptom stability and reduced recurrence rates, highlighting the importance of a holistic and individualised approach to treatment. Although overall nutritional patterns such as vegetarian, meat-based, or mixed diets did not significantly differ between patients with seborrheic dermatitis and healthy controls, certain specific eating behaviours were associated with more severe disease manifestations (Batan et al., 2025). Patients with SD had significantly lower AFHC (Attitude to Healthy Food Choices) scores compared to healthy individuals, suggesting generally less healthy eating behaviours. Among dietary components, higher bread consumption was significantly more common in SD patients than in the control group ($p = 0.001$).

Among people with SD, those with moderate or severe disease were more likely to eat animal fats and margarine, while those with milder symptoms were more likely to choose vegetable oils ($p = 0.008$). In addition, a statistically insignificant trend towards higher consumption of refined sugars was observed in the group of people with moderate to severe disease compared to the group of people with mild disease ($p = 0.050$). These results suggest that dietary patterns characterised by high consumption of refined carbohydrates, saturated fats, margarine, and sugars may be associated with greater disease severity. On the other hand, consumption of unsaturated fats, such as those found in vegetable oils, may correlate with less severe clinical symptoms.

Although patients with seborrheic dermatitis showed higher mean scores for depression, anxiety, and stress, as well as total scores on the Depression, Anxiety, and Stress Scales (DASS-21) compared to healthy individuals in the control group, these differences did not reach statistical significance (Batan et al., 2025). However, among SD patients, those with moderate to severe symptoms had significantly higher anxiety subscale scores ($p = 0.035$) and total DASS-21 scores ($p = 0.049$) than those with mild disease. Although the results for depression and stress also showed an upward trend in the group of patients with a more severe course of the disease, these differences were not statistically significant.

The results suggest that higher levels of anxiety may be associated with more severe SD symptoms, meaning that stress, particularly anxiety, may exacerbate the disease.

Future directions in pathogenesis and diagnosis

Current scientific research increasingly emphasizes the importance of dividing SD's pathogenesis into different factors. While Malassezia species have been regarded as the main etiological agents for many years, there is now evidence that other factors—including host genetic susceptibility, dysregulation of innate and adaptive immune responses, and environmental influences—may also play a critical role in disease development and progression. A better understanding of the mechanisms that drive SD is helping to develop more effective and tailored treatments, improving long-term outcomes.

Although Malassezia yeast has long been considered the primary cause of SD, new research shows that it is only one of many factors. Overactivity of the sebaceous glands and a weakened skin barrier also play a significant role. Growing evidence suggests that an imbalance of skin bacteria may also play a role in the development of SD (Adalsteinsson et al., 2020). People with SD have been found to have changes in the composition of their skin microflora, including a significantly higher prevalence of Staphylococcus aureus colonization compared to healthy individuals (Park et al., 2016). These findings suggest that, in addition to fungal overgrowth, imbalances in bacterial communities may influence the inflammatory milieu and clinical expression of the disease.

Additionally, Staphylococcus and Propionibacterium, the two dominant but antagonistic bacterial genera on the scalp, appear to influence the development of SD and dandruff, depending on their relative abundance (Xu et al., 2016). Disruption of the normal skin microbiota may initiate inflammatory responses that contribute to the development of SD. More clinical trials are needed to assess the effectiveness of emerging treatments and to optimize current therapeutic protocols. Additionally, genetic research has revealed links between seborrheic dermatitis and mutations such as those in the ZNF750 gene, suggesting that host immune mechanisms play a significant role and warrant further genetic investigation into disease susceptibility (Cohen et al., 2012).

Dermatology in artificial intelligence (AI) has appeared as a valuable tool. To distinguish scalp psoriasis from local SD, Yu et al., (2022) created a deep learning model based on dermatoscopic images. According to the report, this model achieved high accuracy, with a sensitivity of 96.1% and a specificity of 88.2%. Its AUC was 0.922; it was better than the dermatoscopist dermatologists trained in work with.

4. CONCLUSION

Seborrheic dermatitis (SD) has been explained as a common, chronic skin disorder whose diagnosis emerges mainly from clinical examination. It includes dermatoscopy and, in cases that are indicated, a histopathological study. Topical antifungal agents like azoles, as well as anti-inflammatory agents such as corticosteroids or calcineurin inhibitors, are the primary focus in treatment. Recently, new treatments including nonsteroidal antifungals, microbiome-targeted therapies, isotretinoin, botulinum toxin, chitosan, and topical JAK inhibitors have been introduced. Although these treatments show potential, further evidence based on long-term studies is required to establish their efficacy and safety. Once we have identified the exact nature of these interactions, treatments can be designed more effectively and safely to inhibit the growth of Malassezia or to understand how the immune system and sebaceous apparatus operate in conjunction with Malassezia. Standardized diagnosis and disease staging will be important to develop better patient care and further research in SD.

Acknowledgments

The authors have no acknowledgments to disclose.

Author's Contribution

All authors contributed significantly to the preparation of this manuscript. Julia Urbańska conceived and designed the review topic and supervised the project. Kamil Nieczaj, Julia Sztubińska, Marta Tortyna, Marta Marciniak conducted the literature search and data extraction. Authors Paula Szarek, Olga Samsel, Natalia Sioch performed critical analysis and interpretation of the collected data. Authors Kamil Nieczaj and Maciej Trzciński contributed to the writing of the manuscript draft. Author Danuta Borowska coordinated the final editing and formatting of the manuscript. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Informed consent

Not applicable.

Ethical approval
Not applicable.

Funding
This study has not received any external funding.

Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES

1. Adalsteinsson JA, Dawes SE, Leslie KS. An update on the microbiology, immunology and genetics of seborrheic dermatitis. Exp Dermatol 2020;29:481-9. doi:10.1111/exd.14091
2. Alshaebi M, Zahed L, Osaylan M, Sulaimani S, Albahlool A, Abduljabbar MH, Hariri J. Association between diet and seborrheic dermatitis: a case-control study. Cureus 2023;15: e48782. doi:10.7759/cureus.48782
3. Azizzadeh M, Gholami S, Jafari M, Dadkhahfar S. The efficacy and safety of pimecrolimus 1% cream vs sertaconazole 2% cream in the treatment of patients with facial seborrhoeic dermatitis: a randomized blinded trial. Clin Exp Dermatol 2022;47:926-31. doi:10.1111/ced.15067
4. Barbosa V, Silva T, Gonçalves A, Almeida L, Silva A, Lima A, Ferreira J. A comparative randomized clinical study assessing the efficacy of a 1% selenium disulfide-based shampoo versus 2% ketoconazole shampoo in subjects with moderate to severe scalp seborrheic dermatitis. Skin Appendage Disord 2024;10:497-504. doi:10.1159/000539209
5. Batan T, Acer E, Kaya Erdoğan H, Ağaoğlu E, Bilgin M, Saraçoğlu ZN. The relationship between nutrition habits, BMI, anxiety, and seborrheic dermatitis. J Cosmet Dermatol 2025; 24:e16737. doi:10.1111/jocd.16737
6. Berk T, Scheinfeld N. Seborrheic dermatitis. P T. 2010;35(6) :348–52.
7. Birnbaum RY, Zvulunov A, Hallel-Halevy D, Cagnano E, Finer G, Ofir R, Geiger D, Silberstein E, Feferman Y, Birk OS. Seborrhea-like dermatitis with psoriasiform elements caused by a mutation in ZNF750, encoding a putative C2H2 zinc finger protein. Nat Genet 2006;38:749-51. doi:10.1038/ng1813
8. Braza TJ, DiCarlo JB, Soon SL, Cohen JL. Tacrolimus 0.1% ointment for seborrhoeic dermatitis: an open-label pilot study. Br J Dermatol 2003;148:1242-4.
9. Clark GW, Pope SM, Jaboori KA. Diagnosis and treatment of seborrheic dermatitis. Am Fam Physician 2015;91:185-90.
10. Cohen I, Scott E, Marmur ES. ZNF750 is expressed in differentiated keratinocytes and regulates epidermal late differentiation genes. PLoS One 2012;7:e42628. doi:10.1371 /journal.pone.0042628
11. Cömert A, Sener S, Yesilova Y, Gokoz O, Yagci R, Ozturk P. Efficacy of oral fluconazole in the treatment of seborrheic dermatitis: a placebo-controlled study. Am J Clin Dermatol 2007;8:235-8. doi:10.2165/00128071-200708040-00005
12. Dawson TL Jr. Malassezia globosa and restricta: breakthrough understanding of the etiology and treatment of dandruff and seborrheic dermatitis through whole-genome analysis. J Invest Dermatol Symp Proc 2007;12:15-9. doi:10.1016/j.jisp. 2007.06.005
13. de Souza Leão Kamamoto C, Rodrigues Júnior A, Pereira RMR, Pereira MG, Ribeiro MC, da Silva RM. Low-dose oral isotretinoin for moderate to severe seborrhea and seborrheic dermatitis: a randomized comparative trial. Int J Dermatol 2017;56:80-5. doi:10.1111/ijd.13408
14. Demirbas A, Toktay O, Yildiz H, Kaya A, Unal S, Celik Y. Efficacy, safety, and recurrence in seborrheic dermatitis: a dose-dependent analysis of oral isotretinoin (10 mg vs 20 mg). J Am Acad Dermatol 2025;S0190-9622(25)02114-0. doi:10.1016 /j.jaad.2025.05.1376
15. Dessinioti C, Katsambas A. Seborrheic dermatitis: etiology, risk factors, and treatments: facts and controversies. Clin Dermatol 2013;31:343-51. doi:10.1016/j.clindermatol.2013.01.0 01
16. Emre S, Atakan N, Erdal ME, Topcuoglu C, Kiliç A, Bilgiç A. The association of oxidative stress and disease activity in seborrheic dermatitis. Arch Dermatol Res 2012;304:683-7. doi:10.1007/s00403-012-1254-0
17. Emtestam L, Berne B, Sundblad L, Ros AM, Rorsman F, Bäckström M. Treatment of seborrhoeic dermatitis of the scalp with a topical solution of urea, lactic acid, and propylene glycol (K301): results of two double-blind, randomised,

placebo-controlled studies. Mycoses 2012;55:393-403. doi: 10.1111/j.1439-0507.2011.02126.x
18. Gaitanis G, Magiatis P, Hantschke M, Bassukas ID, Velegraki A. The Malassezia genus in skin and systemic diseases. Clin Microbiol Rev 2012;25:106-41. doi:10.1128/CMR.00021-11
19. Ghodsi SZ, Barikbin B, Marzban A, Khezri F, Shamsadini L. Efficacy of oral itraconazole in the treatment and relapse prevention of moderate to severe seborrheic dermatitis: a randomized, placebo-controlled trial. Am J Clin Dermatol 2015;16:431-7. doi:10.1007/s40257-015-0133-9
20. Goldust M, Rezaee E, Raghifar R, Farshchian M. Treatment of seborrheic dermatitis: the efficiency of sertaconazole 2% cream vs. tacrolimus 0.03% cream. Ann Parasitol 2013;59:73-7.
21. Goldust M, Rezaee E, Raghifar R. Clinical study of sertaconazole 2% cream vs. hydrocortisone 1% cream in the treatment of seborrheic dermatitis. Ann Parasitol 2013;59:119-23.
22. Goldust M, Rezaee E, Raghifar R. Treatment of seborrheic dermatitis: comparison of sertaconazole 2% cream versus pimecrolimus 1% cream. Ir J Med Sci 2013;182:703-6. doi: 10.1007/s11845-013-0960-8
23. Kim TW, Lee JY, Park YW, Lee DY, Han KH, Kim MB, Kim JH. Proactive treatment of adult facial seborrhoeic dermatitis with 0.1% tacrolimus ointment: randomized, double-blind, vehicle-controlled, multi-centre trial. Acta Derm Venereol 2013;93:557-61. doi:10.2340/00015555-1532
24. Kluglein KA, Smith JT, Miller K, Johnson R, Lee S. Exploring systemic comorbidities and lifestyle factors associated with seborrheic dermatitis: a scoping review. Cureus 2024;16: e73555. doi:10.7759/cureus.73555
25. Kose O, Erbil H, Gur AR. Oral itraconazole for the treatment of seborrhoeic dermatitis: an open, noncomparative trial. J Eur Acad Dermatol Venereol 2005;19:172-5.
26. Kulakli S, Yalcin B, Erdogan M, Kaya A, Ozkan S. Is seborrheic dermatitis associated with early-stage osteoarthritis? Medicine (Baltimore) 2024;103:e37217. doi: 10.1097/MD.0000000000037217
27. Lax SJ, Kalic T, Zotos PC, Tzanetakou V, van der Valk PGM, Navaratnam R, Hollis S, Wong S, Banks A, Chakravarty K. Topical anti-inflammatory treatments for eczema: a Cochrane systematic review and network meta-analysis. Clin Exp Allergy 2024;54:960-72. doi:10.1111/cea.14556
28. Liu Y, Zhang L, Chen X, Wang J, Li H, Zhou Y. Potential applications of chitosan in seborrheic dermatitis and other skin diseases: a comprehensive review. Clin Cosmet Investig Dermatol 2025;18:533-42. doi:10.2147/CCID.S504778
29. Massiot P, Lecha M, de Lacharrière O, Boukris N, Rochette S. Maintenance effect of a once-weekly regimen of a selenium disulfide-based shampoo in moderate-to-severe scalp seborrheic dermatitis after initial treatment with topical corticosteroid/salicylic acid. Eur J Dermatol 2023;33(S1):13-18. doi:10.1684/ejd.2023.4401
30. Miller AC, Johnson M, Harris L, Thompson R. Roflumilast for the treatment of seborrheic dermatitis: a review. Skin Ther Lett 2025;30:1-4.
31. Miri F, Ghodsi SZ, Niazy S, Barikbin B, Marzban A. The additional effect of 5% atorvastatin shampoo in the treatment of adult patients with mild to moderate seborrheic dermatitis of the scalp: a prospective, randomised, double-blind trial. Indian J Dermatol Venereol Leprol 2025;91:222-5. doi:10.25259 /IJDVL_245_2024
32. Nakabayashi A, Sugita T, Nishikawa A, Tsuboi R, Aoki T, Hiruma M. Identification of Malassezia species isolated from patients with seborrhoeic dermatitis, atopic dermatitis, pityriasis versicolor and normal subjects. Med Mycol 2000;38:337-41. doi:10.1080/mmy.38.5.337.341
33. Papp KA, Werbel T, Rosoph L, Del Rosso JQ, Kerrouche N. Single-blind, randomized controlled trial evaluating the treatment of facial seborrheic dermatitis with hydrocortisone 1% ointment compared with tacrolimus 0.1% ointment in adults. J Am Acad Dermatol 2012;67:e11-5. doi: 10.1016/j.jaad .2011.02.032
34. Park JH, Kim DW, Kim JW, Lee JH, Kim YC. Histopathological differential diagnosis of psoriasis and seborrheic dermatitis of the scalp. Ann Dermatol 2016;28:427-32. doi:10.5021/ad.2016.28.4.427
35. Perez D, Johnson M, Smith R, Lee A. An evaluation of the ambulatory diagnosis and treatment of seborrheic dermatitis in PWH in a regional healthcare system. HIV Res Clin Pract 2025;26:2491891. doi:10.1080/25787489.2025.2491891
36. Perez SM, Ho AS, Leavitt M, Chen C, Black J. Botulinum toxin in the treatment of hair and scalp disorders: current evidence and clinical applications. Toxins (Basel) 2025;17:163. doi: 10.3390/toxins17040163
37. Picardo M, Cameli N. Seborrheic dermatitis. In: Williams H, Bigby M, Diepgen T, Herxheimer A, Naldi L, Rzany B, editors. Evidence-based dermatology. 2nd ed. Malden, MA: Blackwell Publishing; 2008; 164-70.
38. Plewig G, Janssen T, Wolff K, Goldsmith LA, Katz SI. Fitzpatrick's dermatology in general medicine. New York: McGraw-Hill; 2008.
39. Polaskey MT, Chang CH, Daftary K, Fakhraie S, Miller CH, Chovatiya R. The global prevalence of seborrheic dermatitis: A systematic review and meta-analysis. JAMA Dermatol 2024;160(8):846–55. doi:10.1001/jamadermatol.2024.1987

40. Rork JF, Isrow D, Mack KJ, Levy ML, Driscoll DJ, Zenlea T. Dermatologic conditions in Down syndrome: a single-center retrospective chart review. Pediatr Dermatol 2020;37:811-6. doi:10.1111/pde.14214
41. Roussel J, Kroes R, van Rijsbergen M, Spiekstra SW, Zeeuwen PLJM, Schalkwijk J, van Erp PEJ, van de Kerkhof PCM, van Rossum MM, van der Heijden E. Treatment with the topical antimicrobial peptide omiganan in mild-to-moderate facial seborrheic dermatitis versus ketoconazole and placebo: results of a randomized controlled proof-of-concept trial. Int J Mol Sci 2023;24:14315. doi:10.3390/ijms241814315
42. Sahoo CK, Sharma P, Sahu M, Chatterjee S, Raghavan R, Gupta V. The efficacy and safety of oral tofacitinib in severe to very severe adult onset seborrheic dermatitis: a case series. J Cutan Med Surg 2025; 12034754251345783. doi:10.1177/12034754251345783
43. Sanders MG, Boekhorst J, Timmerman P, van den Bogaard EH, van Oers HAM, Rodijk-Olthuis D, van Vlijmen-Willems IMJ, Schalkwijk J, Zeeuwen PLJM. Association between diet and seborrheic dermatitis: a cross-sectional study. J Invest Dermatol 2019;139:108-14. doi:10.1016/j.jid.2018.07.027
44. Sarac E, Kocatürk E. Relationship between disease severity, perceived stress, and depression in patients with seborrheic dermatitis. Marmara Med J 2022;35:362-6. doi:10.5472/marumj.1195298
45. Tomic S, Kuric I, Kuric TG, Popovic Z, Kragujevic J, Zubonja TM, Rajkovaca I, Matosa S. Seborrheic dermatitis is related to motor symptoms in Parkinson's disease. J Clin Neurol 2022;18:628-34. doi:10.3988/jcn.2022.18.6.628
46. Trznadel-Grodzka E, Silny W, Nowicki R. Investigations of seborrheic dermatitis. Part I. The role of selected cytokines in the pathogenesis of seborrheic dermatitis. Postepy Hig Med Dosw 2012;66:843-7. doi:10.5604/17322693.1019642
47. Tucker D, Masood S. Seborrheic dermatitis. StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
48. Wananukul S, Chatproedprai S, Tempark T. Randomized, double-blind, split-side comparison study of moisturizer containing licochalcone vs. 1% hydrocortisone in the treatment of infantile seborrhoeic dermatitis. J Eur Acad Dermatol Venereol 2012;26(7):894-7. doi:10.1111/j.1468-3083.2011.04187.x
49. Wang D, Chen H, Liu J, Ma X, Zhang M. A prospective, split-face, randomized controlled trial of intense pulsed light-photodynamic therapy for seborrhea. Photodiagnosis Photodyn Ther 2024;45:103973. doi:10.1016/j.pdpdt.2024.103973
50. Wang J, Smith A, Lee C, Johnson M, Davis R, Patel K, Brown S, Thompson L, Martinez N, Harris D. Chemical, biochemical, and structural similarities and differences of dermatological cAMP phosphodiesterase-IV inhibitors. J Invest Dermatol 2025;145(6):1471-1488.e1. doi:10.1016/j.jid.2024.10.597
51. Wang L, Chen Y, Zhang J, Liu H, Zhao Q, Li X, Sun Y, Wu M, Gao F. Effectiveness and tolerance of medicated shampoo containing selenium sulfide and salicylic acid in patients with seborrheic dermatitis. J Dermatolog Treat 2025;36(1):2506676. doi:10.1080/09546634.2025.2506676
52. Wikramanayake TC, Colby J, Rios E, Burdett C. Seborrheic dermatitis—looking beyond Malassezia. Exp Dermatol 2019;28:758-64. doi:10.1111/exd.14006
53. Wikramanayake TC, Rios E, Colby J, Burdett C. Loss of MPZL3 function causes seborrhoeic dermatitis-like phenotype in mice. Exp Dermatol 2017;26:736-8. doi:10.1111/exd.13150
54. Wilkowski CM, Steinhoff M, Müller S, Becker K. The association of seborrheic dermatitis and Parkinson's disease: a cross-sectional study. J Am Acad Dermatol 2025;92(1):157-9. doi:10.1016/j.jaad.2024.08.074
55. Xian Q, Zhang Y, Liu S, Wang D, Chen H. Role of immune cells in seborrheic dermatitis: a two-sample bidirectional Mendelian randomization study. Arch Dermatol Res 2025;317:482. doi:10.1007/s00403-025-04028-x
56. Xu C, Li J, Wang Y, Zhang Q. [Seborrheic dermatitis diagnosis and treatment]. Zhonghua Yi Xue Za Zhi 2014;94(44):3467–70.
57. Xu Z, Li C, Wang L, Chen Y, Zhang H, Liu F. Dandruff is associated with the conjoined interactions between host and microorganisms. Sci Rep 2016;6:24877. doi:10.1038/srep24877
58. Yu Z, Wang L, Zhang Q, Li H, Chen Y, Zhao X, Liu J, Huang M, Fang W. A deep learning-based approach toward differentiating scalp psoriasis and seborrheic dermatitis from dermoscopic images. Front Med (Lausanne) 2022;9:965423. doi:10.3389/fmed.2022.965423
59. Zani MB, Soares RC, Arruda AC, de Arruda LH, Paulino LC. Ketoconazole does not decrease fungal amount in patients with seborrheic dermatitis. Br J Dermatol 2016;175(2):417–21. doi:10.1111/bjd.14501
60. Zirwas MJ, Bhatia N, Call RS, Wilson J, Grant-Kels JM. Efficacy of Roflumilast Foam, 0.3%, in Patients With Seborrheic Dermatitis: A Double-blind, Vehicle-Controlled Phase 2a Randomized Clinical Trial. JAMA Dermatol 2023;159(6):613-620. doi:10.1001/jamadermatol.2023.0846
61. Zisova LG. Fluconazole and its place in the treatment of seborrheic dermatitis--new therapeutic possibilities. Folia Med (Plovdiv) 2006;48(1):39-45.