Acta Dermatovenerol APA

Acta Dermatovenerologica
Alpina, Pannonica et Adriatica

2025;34:57-62
doi: 10.15570/actaapa.2025.15

Evaluation of clinical and lifestyle factors associated with disease severity in adult patients with scalp seborrheic dermatitis: a retrospective analysis

Nazlı Caf[1] ✉, Mustafa Tümtürk[1], Defne Özkoca[2]

[1]Department of Dermatology, Faculty of Medicine, Istanbul Atlas University, Istanbul, Turkey. [2]Department of Dermatology, Faculty of Medicine, Koç University, Istanbul, Turkey.

Abstract

Introduction: Scalp seborrheic dermatitis (SSD) is a prevalent and chronic dermatological condition. Although various mechanisms have been proposed, its precise etiology and exacerbating factors remain unclear. This study aimed to identify the factors influencing clinical severity in individuals with SSD; to assess the associations between severity and variables such as gender, age at disease onset, treatment history, and the presence of comorbid systemic or dermatological conditions; and to determine potential triggering factors.

Methods: A total of 198 adult patients diagnosed with SSD were included in this retrospective study. Collected data included demographic characteristics, smoking and alcohol use, Fitzpatrick skin type, age at disease onset, duration of the most recent flare, frequency of hair washing, and the season during which the patient presented. Clinical severity of SSD was assessed by a dermatologist. In addition, the presence of systemic and dermatological comorbidities, as well as patient-reported triggering factors, was documented.

Results: A statistically significant difference in SSD severity was observed between sexes, with higher severity noted in male patients ($p = 0.006$). No significant associations were found between SSD severity and age, Fitzpatrick skin type, smoking or alcohol use, season of presentation/flare up, or the presence of systemic diseases. However, SSD severity differed significantly based on the presence of onychomycosis ($p = 0.001$).

Conclusions: The significant association between the frequency of onychomycosis and the severity of SSD highlights a potential link involving shared immunologic, microbial, and skin barrier dysfunctions underlying both conditions.

Keywords: comorbid dermatological conditions, disease severity, onychomycosis, scalp seborrheic dermatitis

Received: 23 March 2025 | **Returned for modification:** 1 May 2025 | **Accepted:** 25 May 2025

Introduction

Scalp seborrheic dermatitis (SSD), also known as seborrhea capitis, is a chronic and common condition with a relapsing–remitting course in the general population. Many mechanisms have been proposed for the etiology of the disease. Fungal components such as *Malassezia*, some bacterial agents, increased sebum production, immune factors, skin barrier disorders, and their relationships with each other have been blamed in its etiology (1–3). The overall incidence of seborrheic dermatitis (SD) tends to have two peaks, one in the first 3 months of life and one between ages 30 and 60 (1). The global prevalence of SSD was determined as 4.38% according to a recent meta-analysis (4). Dandruff may be considered a mild form of SSD, and it affects 17% to 50% of the general population (5). Men have SSD almost twice as often as women, and no ethnic predisposition to this disease has been reported (2). SSD may present with erythema, pruritus, oily yellowish scales, and/or inflammation on the scalp. It may solely present with dandruff (1, 2).

The relationship between SSD and some systemic diseases, especially neurological diseases, is well known (2). In addition, various triggering factors for the disease have been reported. Nutrition style, seasonal changes, smoking, alcohol use, certain medications, and emotional stress may be considered among these triggering factors (6). SD also follows a seasonal rhythm, flaring more often in the colder months of winter while typically easing during the summer months. Sleep deprivation and psychogenic stress are also known to contribute to the exacerbation or aggravation of SD (2). In the treatment of patients with SSD, shampoos containing ketoconazole, selenium sulfide, zinc pyrithione, tea tree oil, and ciclopirox olamine are generally used, although recurrences are common despite treatment (6). In addition, topical treatments such as glucocorticosteroids, salicylic acid, calcineurin inhibitors, antifungals, and oral antifungals are frequently used agents in the conventional treatment of SSD (7).

This study aimed to assess the factors influencing clinical severity in adult patients diagnosed with SSD. Specifically, it investigated the associations between disease severity and a range of variables, including demographic characteristics, clinical history, dermatological and systemic comorbidities, lifestyle and behavioral parameters, seasonal flaring patterns, and self-reported triggering factors. Furthermore, the study employed ordinal logistic regression analysis to identify independent predictors of increased SSD severity.

Methods

Adults that were clinically diagnosed with SSD at the Dermatology Outpatient Clinic of the Medical Faculty, Atlas University, Istanbul, Turkey between August 1st, 2024, and January 31st, 2025, were included in this retrospective study. Patients under age 18, pregnant and lactating women, and patients with missing data were excluded. Patients with only dandruff were excluded;

✉ Corresponding author: naslicaf@hotmail.com

inclusion required at least one erythematous and yellow-scaly SSD plaque.

Demographic data of the patients, smoking/alcohol use status, Fitzpatrick skin types, age of disease onset, duration of the last attack, frequency of hair washing per week, season of admission to the outpatient clinic or flare, SSD severity clinically assessed by a single dermatologist (clinically classified as mild, moderate, or severe), presence of systemic disease, and additional dermatological disease in addition to triggering factors stated by the patient were recorded. It was also noted whether the participants had received treatment previously and what was prescribed to the patient in his or her current application.

Approval from the local ethics committee was obtained before initiation of the study (approval number: E-22686390-050.99-58773, Istanbul Atlas University Ethics Committee). The study was conducted according to the principles of the Declaration of Helsinki.

Statistical analysis

Analysis of the data included in the research was performed with the SPSS® 27.0 (IBM, New York, USA). Numbers and percentage values were given for categorical variables.

Pearson's chi-squared test was used to test the relationship between categorical data. The Shapiro–Wilk test was used to test the conformity of continuous variables in the study to normal distribution. Descriptive statistics including mean, standard deviation, and minimum and maximum values were calculated for continuous variables. Intergroup comparisons and group comparisons regarding the characteristics included in the demographic information form were made with the Mann–Whitney *U* and Kruskal–Wallis *H* tests because the data set did not show a normal distribution. A value of $p < 0.05$ was considered statistically significant.

Results

Demographic data

The study enrolled 198 patients diagnosed with SSD. Fifty-seven percent of the participants were male, and 43% were female. The mean age was 34.85 ± 14.89 years (Table 1). Smoking and alcohol use were reported by 43% and 7% of the patients, respectively. Fitzpatrick skin type III was the most common type, with a frequency of 44% (Table 1).

Among all patients, 72% had no systemic disease. Among those with systemic diseases, the most common were hypothyroidism (23%), hypertension (20%), and type II diabetes mellitus (11%; Table 1).

Regarding dermatologic comorbidities, 24% had androgenetic alopecia, 23% had onychomycosis, 23% had xerosis cutis, 21% had tinea pedis, 17% had acne vulgaris, 14% had telogen effluvium, 7% had rosacea, 3% had vitiligo, and 2% had folliculitis. The frequency of patients washing their hair was an average of 4.08 ± 1.70 days per week and ranged from 1 to 7 days.

Prior to their presentation at our institution, 66% of patients had received treatment. The therapeutic agents initiated by our outpatient clinic during the patients' visits were as follows: 37% were prescribed ketoconazole, 24% ciclopirox olamine, 35% zinc pyrithione, 21% shampoos containing selenium sulfide, 30% topical corticosteroids, 31% a topical combination of corticosteroid and salicylic acid, 12% topical calcineurin inhibitors, and 7% oral itraconazole. Patients with previous treatments had a higher frequency of moderate and severe disease ($p = 0.012$).

Disease triggers and seasonality

Of the patients included in the study, 58% reported stress, 10% smoking, 5% alcohol use, 49% seasonal change, 35% fatty diet, and 42% physical fatigue and insomnia as self-reported triggers of their diseases. Fifty-five percent of the patients were admitted in winter ($n = 109$), 22% in autumn ($n = 43$), 17% in spring ($n = 34$), and 6% in summer, based on either the timing of admission or the patient-reported onset of symptom flares ($n = 12$; Table 1).

Table 1 | **Patients' sociodemographic characteristics and disease information.**

	n	%
Sex		
Female	86	43.4
Male	112	56.6
Age		
Mean ± *SD*	34.85 ± 14.89	
Median (min–max)	30 (18–79)	
Age group (years)		
18–29	98	49.5
30–43	46	23.2
44–55	31	15.7
56–79	23	11.6
Smoking		
Nonsmoker	112	56.6
Smoker	86	43.4
Alcohol use		
No	184	92.9
Yes	14	7.1
Age at disease onset (years)		
Mean ± *SD*	25.52 ± 13.40	
Median (min–max)	20 (9–79)	
Last attack duration (days)		
Mean ± *SD*	98.26 ± 110.6	
Median (min–max)	60 (3–700)	
Hair washing frequency/week		
Mean ± *SD*	4.08 ± 1.70	
Median (min–max)	4 (1–7)	
Fitzpatrick skin type		
I	7	3.5
II	57	28.8
III	87	43.9
IV	46	23.2
V	1	0.5
Season of admission/flare		
Winter	109	55.1
Spring	34	17.2
Summer	12	6.1
Autumn	43	21.7
SSD Severity		
Mild	101	51.0
Moderate	73	36.9
Severe	24	12.1
Systemic disease		
No	142	71.7
Yes	56	28.3
Hypothyroidism	13	23.2
Hyperthyroidism	3	5.4
Hypertension	11	19.6
Diabetes mellitus	6	10.7
Coronary artery disease	4	7.1
Parkinson's disease	2	3.6
Rheumatoid arthritis	3	5.4
Asthma	5	8.9
Dyslipidemia	5	8.9
Iron deficiency anemia	4	7.1

SD = standard deviation, min = minimum, max = maximum, SSD = scalp seborrheic dermatitis.

Clinical data and their relationship with disease severity

The mean age of onset of the disease was 25.52 ± 13.40 years. The mean SSD last attack duration was 98.26 ± 110.60 days. Clinically, 51% of patients had mild SSD, 37% had moderate SSD, and 12% had severe SSD (Table 1).

A statistically significant relationship was found between SSD severity and sex (p = 0.006), with males having more severe SSD (Table 2). No statistically significant associations were found between SSD severity and age group, Fitzpatrick skin type, smoking, alcohol use, season of admission, or the presence of systemic diseases (p > 0.05 for all; Tables 2, 3).

SSD severity was also significantly related to prior treatment history (p = 0.012) because patients that had received treatment previously had higher SSD severity (Table 2). There was no statistically significant difference in SSD severity according to whether the patients had acne, tinea pedis, vitiligo, folliculitis, rosacea, androgenetic alopecia, telogen effluvium, or xerosis (p > 0.05; Table 3).

A significant association was observed between the presence of onychomycosis and SSD severity (p = 0.001), with more severe cases seen in patients with onychomycosis (Table 3). No triggering factor, including stress, seasonal change, or dietary habits, exhibited a significant effect on disease severity (p > 0.05; Table 4). There was no significant relationship between SSD severity and age of onset, disease duration, duration of last attack, or hair washing frequency (p > 0.05; Table 5). Ordinal logistic regression analysis revealed that sex and treatment history significantly influenced SSD severity. Male gender (p = 0.007) and prior treatment (p = 0.007) were independently associated with higher SSD severity (Table 6).

Table 2 | **Relationship between demographic data and scalp seborrheic dermatitis severity.**

	Mild *n* (%)	Moderate *n* (%)	Severe *n* (%)	x^2	*p*
Sex				10.362	0.006*
Male	47 (42.0)	46 (41.1)	19 (17.0)		
Female	54 (62.8)	27 (31.4)	5 (5.8)		
Age group (years)				1.870	0.931
18–29	51 (52.0)	36 (36.7)	11 (11.2)		
30–43	20 (43.5)	20 (43.5)	6 (13.0)		
44–55	17 (54.8)	10 (32.3)	4 (12.9)		
56–79	13 (56.5)	7 (30.4)	3 (13.0)		
Smoking				4.912	0.086
Non-smoker	58 (51.8)	36 (32.1)	18 (16.1)		
Smoker	43 (50.0)	37 (43.0)	6 (7.0)		
Alcohol use				1.361	0.506
No	94 (51.1)	69 (37.5)	21 (11.4)		
Yes	7 (50.0)	4 (28.6)	3 (21.4)		
Fitzpatrick skin type				10.908	0.207
I	6 (85.7)	1 (14.3)	0 (0.0)		
II	30 (52.6)	17 (29.8)	10 (17.5)		
III	41 (47.1)	39 (44.8)	7 (8.0)		
IV	24 (52.2)	15 (32.6)	7 (15.2)		
V	0 (0.0)	1 (100.0)	0 (0.0)		
Season of admission/flare				6.385	0.381
Winter	54 (49.5)	40 (36.7)	15 (13.8)		
Spring	17 (50.0)	14 (41.2)	3 (8.8)		
Summer	10 (83.3)	2 (16.7)	0 (0.0)		
Autumn	20 (46.5)	17 (39.5)	6 (14.0)		
Systemic disease				0.293	0.864
No	71 (50.0)	54 (38.0)	17 (12.0)		
Yes	30 (53.6)	19 (33.9)	7 (12.5)		
Previous treatment				8.829	0.012*
No	44 (65.7)	18 (26.9)	5 (7.5)		
Yes	57 (43.5)	55 (42.0)	19 (14.5)		

*p < 0.05 was considered statistically significant.

Table 4 | **Relationship between disease triggering factors and scalp seborrheic dermatitis severity.**

Trigger		Mild *n* (%)	Moderate *n* (%)	Severe *n* (%)	x^2	*p*
Stress	No	41 (49.4)	31 (37.3)	11 (13.3)	0.233	0.890
	Yes	60 (52.2)	42 (36.5)	13 (11.3)		
Smoking	No	90 (50.3)	67 (37.4)	22 (12.3)	0.399	0.819
	Yes	11 (57.9)	6 (31.6)	2 (10.5)		
Alcohol	No	95 (50.3)	71 (37.6)	23 (12.2)	1.010	0.604
	Yes	6 (66.7)	2 (22.2)	1 (11.1)		
Seasonal change	No	58 (56.9)	34 (33.3)	10 (9.8)	3.058	0.217
	Yes	43 (44.8)	39 (40.6)	14 (14.6)		
Fatty diet	No	67 (52.3)	45 (35.2)	16 (12.5)	0.457	0.796
	Yes	34 (48.6)	28 (40.0)	8 (11.4)		
Fatigue, sleep deprivation	No	61 (53.5)	42 (36.8)	11 (9.6)	1.684	0.431
	Yes	40 (47.6)	31 (36.9)	13 (15.5)		

*p < 0.05 was considered statistically significant.

Table 5 | **Relationship between age, age at disease onset, disease duration, and scalp seborrheic dermatitis severity.**

		Mild	Moderate	Severe	*p*
Age (years)	Mean ± *SD*	35.16 ± 5.32	33.88 ± 14.65	36.54 ± 14.18	0.587
	Median	29.0	30.0	33.5	
Age at disease onset (years)	Mean ± *SD*	26.58 ± 3.47	24.05 ± 13.50	25.54 ± 12.95	0.214
	Median	22.0	19.0	20.5	
Disease duration (years)	Mean ± *SD*	8.57 ± 8.57	9.96 ± 9.34	11.0 ± 9.32	0.284
	Median	5.0	9.0	9.5	
Last attack duration (days)	Mean ± *SD*	90.88±100.90	108.78±124.30	97.29 ± 107.13	0.346
	Median	50.0	60.0	55.0	
Hair washing frequency/week	Mean ± *SD*	4.17 ± 1.60	4.04 ± 1.80	3.83 ± 1.80	0.499
	Median	4.0	3.0	3.5	

*$p < 0.05$ was considered statistically significant.
SD = standard deviation.

Table 6 | **Relationship between regression analysis of factors determining scalp seborrheic dermatitis severity.**

	OR	95% CI	*p*
Age	0.981	0.954/1.008	0.154
Sex (male)	2.519	1.845/3.193	0.007*
Fitzpatrick skin type			
I	0.516	−2.211/2.524	0.124
II	1.066	0.249/1.883	0.878
III	0.981	0.247/1.715	0.959
Season of admission			
Winter	0.900	0.181/1.619	0.774
Spring	0.923	−0.013/1.860	0.868
Summer	0.218	−1.528/1.965	0.087
Previous treatment	0.400	−0.262/1.062	0.007*
Smoking	0.727	0.116/1.337	0.305
Alcohol	0.670	−0.451/1.791	0.484
Acne vulgaris	1.709	0.884/2.533	0.203
Tinea pedis	1.342	0.582/2.102	0.448
Onychomycosis	1.725	0.933/2.518	0.177
Rosacea	0.634	−0.635/1.904	0.482
Androgenetic alopecia	1.264	0.486/2.042	0.555
Xerosis cutis	1.844	1.007/2.682	0.152
(Trigger) stress	0.919	0.310/1.527	0.785
(Trigger) seasonal change	1.797	1.194/2.400	0.057
(Trigger) fatigue, sleep deprivation	1.414	0.824/2.005	0.250

*$p < 0.05$ was considered statistically significant. x^2 (20) = 40.678; $p = 0.004$; $R^2 = 0.217$.
OR = odds ratio, CI = confidence interval, x^2 = Chi squared, R^2 = R squared.

Discussion

This study aimed to identify the factors influencing clinical severity in patients with SSD. Specifically, we examined how variables such as sex, age at disease onset, prior treatment history, and the presence of systemic or dermatological comorbidities relate to disease severity, as well as potential triggering factors.

Sebaceous gland activity is known to play a key role in the etiopathogenesis of SSD, with androgens significantly influencing gland function. This relationship is commonly cited as the reason for the higher prevalence of SSD in men (3). While some studies suggest that increased cosmetic product use may lead to higher rates of SSD in women, the prevailing view is that the condition is more frequent in men (3, 8). In our study, SSD was both more common and more severe in male patients, with a statistically significant difference in severity based on sex. The number of studies specifically assessing SSD in relation to disease severity is limited. Notably, our findings are confined to scalp involvement, unlike broader studies that include other forms of SD, which may enhance their clinical relevance in understanding SSD-specific patterns. One study examining SD in general reported no significant association between sex and disease severity (9). However, by focusing solely on SSD and identifying greater severity in male patients, our results may offer a valuable contribution to the limited body of literature addressing SSD severity specifically.

Findings in the literature regarding the relationship between smoking, alcohol use, and the severity of SD are inconsistent; while some studies suggest a potential association, others report no clear effect (9). However, there is a lack of studies specifically investigating these factors in relation to SSD.

A study conducted on adolescent males with SSD reported that there was no relationship between smoking and SSD (10). Similarly, in our study, no statistical difference was found between alcohol or smoking and SSD disease severity. Although our sample size is limited, our results may suggest that smoking and alcohol may not increase SSD severity. Given potential differences in usage between adolescents and adults, more data are needed in adult populations.

It is well established that SSD exhibits seasonal variation, with higher incidence during the winter months and lower prevalence in the summer (11, 12). In line with this, our study also recorded the highest number of SSD cases in winter and the lowest in summer. Notably, the seasonal variation observed was more pronounced than previously reported, with a sharper decline during the summer months. This may be influenced by factors such as healthcare-seeking behavior, increased cosmetic awareness, social media influence, or regional differences in sun exposure. However, as these variables were not directly assessed in our study, this interpretation remains speculative and warrants further investigation. While seasonal changes in SSD prevalence were evident, no statistically significant differences in disease severity were found across seasons. Given the limited number of studies exploring seasonal variation in SSD severity, this finding may represent a meaningful contribution to the current literature.

Certain systemic diseases, particularly neurological conditions, are known to increase the prevalence of SD, including its scalp-specific form, SSD (2, 12). However, there is a lack of data in the literature regarding the impact of these systemic conditions on SSD severity. One study examining patients with Parkinson's disease reported a positive correlation between SD severity and motor symptom severity, though it did not focus specifically on SSD (13). In our study, no statistically significant difference in SSD severity was observed between patients with and without systemic diseases. However, this finding should be interpreted with caution due to the small number of participants with neurological disorders and the fact that the study population was not composed exclusively of neurologically affected individuals. Therefore, while systemic diseases may contribute to SSD prevalence, our results suggest they do not necessarily influence disease severity. It is also important to note that immunosuppressive conditions such as HIV, which may affect disease severity, were not present among the systemic diseases assessed in this study.

A statistically significant difference in SSD severity was ob-

served based on prior treatment history, with greater severity found in patients who had previously received treatment. This may suggest that individuals with more severe baseline disease are more likely to seek or require treatment. Alternatively, treatment-related alterations in scalp physiology could also contribute to increased severity. However, due to the retrospective nature of this study and the absence of longitudinal data, these interpretations remain speculative. Further research—particularly studies incorporating microbiological, immunological, or pharmacogenetic analyses—is needed to clarify the underlying mechanisms.

No statistically significant difference in SSD severity was found in relation to the presence of other dermatological conditions such as acne, tinea pedis, vitiligo, folliculitis, rosacea, androgenetic alopecia, telogen effluvium, or xerosis. However, a significant association was observed between SSD severity and the presence of onychomycosis. Although the underlying mechanism remains unclear, this may suggest shared immunological or skin barrier dysfunctions associated with superficial fungal infections. Various id reactions have been reported in the literature in response to superficial fungal agents, and in some cases, SD-like erythematous scaly plaques have been described as a manifestation of such reactions (14). To date, a similar phenomenon has not been reported specifically in SSD. The increased coexistence of onychomycosis in patients with more severe SSD in this study may point toward a possible id reaction; however, in the absence of direct supporting evidence, this remains a hypothetical explanation. Given the lack of focused research on this association, further studies are needed to explore whether onychomycosis contributes to SSD severity through immunological or microbial mechanisms.

It is also recognized that various immunological mechanisms contribute to the pathogenesis of SSD. Th1 and Th17 immune responses are known to offer protection against fungal infections, while the antimicrobial peptide cathelicidin LL-37 has demonstrated defense activity against superficial fungal pathogens, including *Malassezia* species, which are implicated in SD (15). Based on the observed association between SSD and onychomycosis in this study, it can be speculated that a shared vulnerability—possibly involving impaired immunological, antimicrobial, or skin barrier mechanisms—may underlie both conditions in affected individuals. Given that both *Malassezia* and the pathogens responsible for onychomycosis are fungal in nature, further research is warranted to investigate potential overlapping immunological pathways involved in their development (2, 16, 17).

In this study, all participants reported at least one potential triggering factor—such as stress, seasonal changes, smoking, alcohol use, fatty diet, physical fatigue, or sleep deprivation—yet no statistically significant association was found between these variables and SSD severity. However, this lack of statistical significance does not necessarily rule out clinical relevance. The retrospective design, reliance on subjective self-reporting, individual variability in sensitivity, and the multifactorial nature of SSD may have contributed to these results. Previous studies on SD triggers have yielded inconsistent findings (6–8, 18, 19), and research specifically addressing SSD triggers remains limited.

Although stress, sleep disturbance, dietary habits, and seasonal changes were frequently cited by patients, their role in sustained disease severity—as opposed to triggering flare-ups—remains unclear. This distinction may require future studies to incorporate objective biomarkers, longitudinal tracking, and stratification based on stress resilience or skin barrier integrity. Moreover, the potential involvement of psychoneuroimmune mechanisms in SSD pathogenesis underscores the need for multidisciplinary research teams, including psychiatric evaluation, to explore the psychodermatological axis. Finally, larger-scale studies are essential to better understand stress-related mechanisms and their possible impact on the chronicity and severity of SSD.

The relationship between patients' weekly hair-washing frequency and SSD severity was evaluated in this study, and no statistically significant association was found. Although excessive seborrhea is recognized as a contributing factor in the pathogenesis of SSD (1), the frequency of hair washing may act more as a trigger than a determinant of disease severity. The lack of association with severity observed here suggests that other mechanisms—such as alterations in scalp microbiota or underlying immunological responses—may play a more significant role. Further research is needed to better understand these potential pathways and to clarify the role of hair hygiene habits in SSD pathophysiology.

This study specifically addresses SSD, an area that remains relatively underexplored in the literature, thereby enhancing the potential contribution of these findings. The primary limitations of the study include its retrospective and cross-sectional design, as well as the reliance on physician-based assessment rather than a validated severity scale. However, efforts were made to reduce potential bias: disease severity was evaluated by a single dermatologist, patients with incomplete data were excluded, and clinical parameters were consistently documented in patient records. To strengthen methodological rigor, future research should aim to include larger, more diverse patient populations and incorporate validated assessment tools such as the Seborrheic Dermatitis Area and Severity Index (SDASI).

Conclusions

Our study explored factors influencing the clinical severity of SSD, examining associations with demographic characteristics, disease history, comorbidities, and potential triggering factors. The findings offer new insights into determinants of SSD severity.

Although commonly cited triggers such as stress, seasonal changes, smoking, alcohol use, and dietary habits were frequently reported by patients, no statistically significant associations with disease severity were identified. This may indicate that while these factors could initiate flare-ups, the mechanisms driving sustained or more severe disease are likely multifactorial and more intricately linked to host-specific factors.

A particularly notable finding was the significant association between SSD severity and the presence of onychomycosis, suggesting the possibility of shared immunological pathways or a common defect in skin barrier function. Additionally, patients with prior treatment history demonstrated more severe disease than treatment-naive individuals, raising the hypothesis that previous interventions might alter scalp microbiota, local immunity, or barrier characteristics—though these mechanisms remain speculative and were not directly assessed in this study.

Overall, the results suggest that while environmental and lifestyle factors may play a role in triggering SSD episodes, the progression and severity of the disease may be more strongly influenced by individual immune responses and skin barrier integrity. Further prospective and mechanistic studies are needed to clarify these relationships.

References

1. Leroy AK, Cortez De Almeida RF, Obadia DL, Frattini S, Melo DF. Scalp seborrheic dermatitis: what we know so far. Ski Appendage Disord. 2023;9:160–4.
2. Wikramanayake TC, Borda LJ, Miteva M, Paus R. Seborrheic dermatitis—looking beyond Malassezia. Exp Dermatol. 2019;28:991–1001.
3. Adalsteinsson JA, Kaushik S, Muzumdar S, Guttman E, Ungar J. An update on the microbiology, immunology and genetics of seborrheic dermatitis. Exp Dermatol. 2020;29:481–9.
4. Polaskey MT, Chang CH, Daftary K, Fakhraie S, Miller CH. The global prevalence of seborrheic dermatitis: a systematic review and meta-analysis. JAMA Dermatol. 2024;160:846–55.
5. Tao R, Li R, Wang R. Skin microbiome alterations in seborrheic dermatitis and dandruff: a systematic review. Exp Dermatol. 2021;30:1546–53.
6. Jackson JM, Alexis A, Zirwas M, Taylor S. Unmet needs for patients with seborrheic dermatitis. J Am Acad Dermatol. 2024;90:597–604.
7. Dessinioti C, Katsambas A. Seborrheic dermatitis: etiology, risk factors, and treatments: facts and controversies. Clin Dermatol. 2013;31:343–51.
8. Woolhiser E, Keime N, Patel A, Weber I, Adelman M, Dellavalle RP. Nutrition, obesity, and seborrheic dermatitis: systematic review. JMIR Dermatol. 2024;7: e50143.
9. Etgu F. Analysis of the epidemiology, coexisting skin diseases, and variables affecting seborrheic dermatitis severity. Med Sci Int Med J. 2023;12:994.
10. de Avelar Breunig J, de Almeida HL, Duquia RP, Souza PRM, Staub HL. Scalp seborrheic dermatitis: prevalence and associated factors in male adolescents. Int J Dermatol. 2012;51:46–9.
11. Lyakhovitsky A, Tzanani I, Gilboa S, Segal O, Galili E, Baum S, et al. Changing spectrum of hair and scalp disorders over the last decade in a tertiary medical centre. J Eur Acad Dermatol Venereol. 2023;37:184–93.
12. Vano-Galvan S, Reygagne P, Melo DF, Barbosa V, Wu WY, Moneib H, et al. A comprehensive literature review and an international expert consensus on the management of scalp seborrheic dermatitis in adults. Eur J Dermatol. 2024;34: 4–16.
13. Tomic S, Kuric I, Kuric TG, Popovic Z, Kragujevic J, Zubonja TM, et al. Seborrheic dermatitis is related to motor symptoms in Parkinson's disease. J Clin Neurol. 2022;18:628–34.
14. Ilkit M, Durdu M, Karakaş M. Cutaneous id reactions: a comprehensive review of clinical manifestations, epidemiology, etiology, and management. Crit Rev Microbiol. 2012;38:191–202.
15. Gupta C, Das S, Ramachandran VG, Saha R, Bhattacharya SN, Dar SA, et al. Possible role of trichophytin antigen in inducing impaired immunological clearance of fungus in onychomycosis. Mycopathologia. 2016;181:247–51.
16. Gupta AK, Kohli Y, Summerbell RC, Faergemann J. Quantitative culture of Malassezia species from different body sites of individuals with or without dermatoses. Med Mycol. 2001;39:243–51.
17. Gupta AK, Stec N, Summerbell RC, Shear NH, Piguet V, Tosti A, et al. Onychomycosis: a review. J Eur Acad Dermatol Venereol. 2020;34:1972–90.
18. Sanders MGH, Pardo LM, Ginger RS, Kiefte-de Jong JC, Nijsten T. Association between diet and seborrheic dermatitis: a cross-sectional study. J Invest Dermatol. 2019;139:108–14.
19. Sanders MGH, Pardo LM, Franco OH, Ginger RS, Nijsten T. Prevalence and determinants of seborrhoeic dermatitis in a middle-aged and elderly population: the Rotterdam Study. Br J Dermatol. 2018;178:148–53.