Dermatology Reports
https://www.pagepress.org/journals/index.php/dr/index

eISSN 2036-7406

Please cite this article as:

Pratama AI, Legiawati L, Fitri EM, et al. Comparison of scalp interleukin-17 levels in hijab-wearing women with and without seborrheic dermatitis and the correlation with disease severity. *Dermatol Rep 2025 [Epub Ahead of Print] doi: 10.4081/dr.2025.10623*

Submitted 23/09/25 - Accepted 08/11/25

Comparison of scalp interleukin-17 levels in hijab-wearing women with and without seborrheic dermatitis and the correlation with disease severity

Aditya Indra Pratama,[1,2] Lili Legiawati,[1,2] Eyleny Meisyah Fitri,[1,2] Syougie Syougie,[3] Sri Linuwih S. W. Menaldi[1,2]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia, Jakarta; [2]Cipto Mangunkusumo Hospital, Jakarta; [3]Department of Community Medicine, Faculty of Medicine, Universitas Indonesia, Indonesia

Correspondence: Lili Legiawati, Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia; Cipto Mangunkusumo Hospital, Jl. Salemba Raya No. 6, 10430 Central Jakarta, Indonesia.
E-mail: lililegiawati@yahoo.com

Key words: hijab-wearing women; IL-17; seborrheic dermatitis; tape stripping.

Contributions: AIP, conceptualization, study design, data collection, statistical analysis, interpretation of results, and manuscript drafting; LL, supervision, conceptualization, study design, methodology guidance, critical revision of the manuscript, and final approval; EMF, supervision, conceptualization, study design, methodology guidance, critical revision of the manuscript, and final approval; SS, supervision, study design, interpretation of data, critical revision of the manuscript, and final approval; SLSWM, conceptualization, critical revision of the manuscript, and final approval.
All authors meet the ICMJE criteria for authorship, have read and approved the final manuscript, and agree to be accountable for all aspects of the work.

Conflict of interest: the authors declare that they have no competing interests.

Ethics approval and consent to participate: this study was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Universitas Indonesia (No. KET-459/UN2.FI/ETIK/PPM.00.02/2025) and Cipto Mangunkusumo Hospital (No. YR.02.01/D.IX.2.3/838/2025). Written informed consent was obtained from all participants.

Availability of data and materials: the datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Funding: this research was funded by the Directorate of Research and Development, Universitas Indonesia, under Hibah PUTI 2024 (Grant No. NKB-73/UN2.RST/HKP.05.00/2024).

Acknowledgments: the authors gratefully acknowledge the support of the Directorate of Research and Development, Universitas Indonesia, our research assistant (Victoria Theresia Dame, MD and Erika Pratista Hermawan, MD), and all study participants who made this research possible.

Abstract

Seborrheic dermatitis (SD) is linked with *Malassezia* colonization, which increases in hijab-wearing women. The growing number of hijab wearers in Indonesia makes this issue relevant. Interleukin (IL)-17 plays a role in inflammation and antifungal immunity, including against *Malassezia*. However, studies on the role of IL-17 in SD, particularly in hijab-wearing women, are currently lacking. Our study purpose was to evaluate differences in IL-17 levels between hijab-wearing women with SD and without SD, and their correlation with SD severity. This cross-sectional study included 66 hijab-wearing participants aged 18-59 years, with 34 in the SD group and 32 in the non-SD group. IL-17 expression was assessed using Enzyme-Linked Immunosorbent Assay (ELISA) on scalp skin samples obtained through tape stripping (Sebutape®). The SD group had significantly lower IL-17 levels (0.064 [0.006-0.354] pg/μg protein) compared to the non-SD group (0.105 [0.030-0.531] pg/μg protein) (p=0.002), with a strong negative correlation between IL-17 levels and SD severity scores (r= –0.728, p=0.000). The result suggests a potential protective role of IL-17 against SD.

Introduction

Seborrheic dermatitis (SD) is a chronic and recalcitrant inflammatory skin disorder that primarily affects the sebaceous gland-bearing areas. Globally, the prevalence of SD has been reported to be approximately 5%.[1] In Indonesia, outpatient visits at Cipto Mangunkusumo Hospital accounted for about 11% in 2023,[2] and the prevalence of adolescent SD significantly increased (26.54%) compared to subtropical countries.[3] Female gender, scalp predilection, and younger age were shown as negative factors for quality of life in SD patients.[4,5] The incidence of SD is also linked with the prominent *Malassezia restricta*[6] and disruption of the skin barrier[7] in hijab-wearing women. In Indonesia, where Muslims are the majority, wearing the hijab is a common practice (72% in 2018), making this issue significant.

Seborrheic dermatitis is caused by the hydrolysis of free fatty acids by *Malassezia*, which then activates the dendritic cells through pattern recognition receptors (PRRs). Dendritic cells stimulate both T helper (Th)2 and Th17, which then results in the production of the cytokines interleukin (IL)-2, IL-6, IL-10, IL-12, and IL-17.[8,9]

Currently, the role of IL-17 in SD remains poorly understood; however, IL-17 is strongly associated with other autoinflammatory diseases.[8] Experimental studies of *Mpzl3* mice with the SD phenotype found increased IL-17 levels accompanied by an increase in γδ T cells as the primary IL-17-producing cells.[9] In the epicutaneous infection model, the colonization of *Malassezia pachydermatis* increased IL-17-mediated inflammation, and the knockout IL-17 model showed uncontrollable *Malassezia* growth, suggesting a relationship between *Malassezia* and the degree of inflammatory response.[10]

The persistence of the *Malassezia* colony is shown to induce chronic inflammation.[11] It was also reported that *Malassezia* infection[12] and SD[13] occurred in IL-17 inhibitor treatment.

There are no studies on IL-17 levels in SD, especially among hijab-wearing women, or their connection to the extent of inflammation caused. This study explored differences in IL-17 levels between hijab-wearing women with SD and non-SD, and the correlation with SD scalp severity.

Materials and Methods

Design overview

This study was an analytical cross-sectional investigation aimed at measuring the mean difference of IL-17 levels in the scalps of hijab-wearing women with SD compared to hijab-wearing women without SD. This study also analysed the correlation between IL-17 value and the severity score of SD.

Setting and participants

The study was conducted at the Dermatology and Venereology Outpatient Clinic, Cipto Mangunkusumo Hospital (Indonesia), from May to June 2025. The ethical approval for this study was granted by the Health Research Ethics Committee, Faculty of Medicine, Universitas Indonesia, and Cipto Mangunkusumo Hospital. Participants were divided into two groups: hijab-wearing women with SD (SD group) and hijab-wearing women without SD (non-SD group). Inclusion criteria for both groups consisted of women with scalp SD lesions aged between 18 and 59 years, who have worn hijab for at least 5 years for a minimum of 8 hours daily, and who willingly agreed to participate in the study. Exclusion criteria for both groups included having any scalp disorder (such as alopecia, trichotillomania, malignancy, inflammation other than SD, or infection), use of any topical or therapeutic product (including ingredients like antifungal, anti-inflammatory, or corticosteroids) within the past 2 weeks, use of systemic medication (including antibiotics, immunosuppressants, or nonsteroidal anti-inflammatory drugs) within the past 4 weeks, and pregnancy or lactation.

From the time of enrolment, there were 34 participants in the SD group and 32 participants in the non-SD group. The minimum required sample size was calculated using the formula for detecting a mean difference in IL-17 levels and for assessing the correlation between IL-17 levels and the SD severity score.

SD severity score

The severity of SD scalp was measured with the 16-point scale described by Zhang *et al*.[14] The SD severity was divided into three categories: mild, moderate, and severe. The points measured included

adherent scalp flaking (0-10), area of erythema (0-3), and pruritus (0-3). A total accumulation of 0-5 points showed mild, 6-9 points showed moderate, 10-16 points showed severe SD.[14]

Skin stripping

IL-17 was measured from the stratum corneum and sebum collected using the tape absorption method with Sebutape®. We adopted the sample collection method described by Perkins *et al*.[15] Sebutape® was divided horizontally into three linear strips and applied to the scalp for one minute each. In contrast to the previous study, which used two strips, we used three strips (nine linear sections).
Collected samples were processed into skin homogenates and analyzed for IL-17 using an Enzyme-Linked Immunosorbent Assay (ELISA) kit from Cloud-Clone Corp (Houston, TX, USA).

Statistical analysis

Data were stored in a database and analyzed using SPSS version 21 (IBM Corp., Armonk, NY, USA). The chi-square test was applied to categorical variables, with Fisher's exact test used when chi-square assumptions were not met. Due to non-normal distribution, the Mann-Whitney test was employed to compare IL-17 levels between groups. Spearman's correlation test was performed to evaluate the relationship between IL-17 levels and disease severity scores. A p-value of <0.05 was considered statistically significant.

Results

Sociodemographic characteristics

The sociodemographic characteristics of the study participants are presented in Table 1. A total of 66 individuals were enrolled, comprising 34 in the SD group and 32 in the non-SD group. The mean age in the SD group was 37.94±9.19 years, while in the non-SD group it was 35.78±10.18 years (p=0.368). Most participants in the SD group had a medium level of education (61.8%), whereas in the non-SD group, the majority had higher education (65.6%), suggesting that both groups generally had moderate-to-high education levels. In terms of occupation, the majority of participants in both groups worked as civil servants or private employees: 82.4% in the SD group and 68.8% in the non-SD group. The average duration of hijab use was significantly longer in the SD group than in the non-SD group (17.4±6.3 *vs.* 14.8±7.3 years; p=0.043).
No participants reported a history of HIV/AIDS or cardiovascular disease; meanwhile, the prevalence of hypertension and diabetes mellitus was relatively low in both. The prevalence of dyslipidaemia was higher in the SD group compared to the non-SD group (26.5% *vs.* 3.1%). However, comorbidity

assessment in this study was based solely on medical history interviews and did not use standardized diagnostic procedures.

More than half of the SD group (52.6%) reported a family history of SD, compared to the non-SD group (40.6%), though this difference was not statistically significant (p=0.45). No participants in either group reported being active smokers. Most participants in both groups were categorized as overweight or obese, classified as high risk for metabolic syndrome. This proportion was higher in the SD group (82.4%) than in the non-SD group (68.8%), although the difference was not statistically significant (p=0.317).

Clinical characteristics

The clinical characteristics of participants are shown in Table 2. Among SD participants, 11 (32.4%) had mild SD, 15 (44.1%) had moderate SD, and 8 (23.5%) had severe SD. There was a trend toward increased topical treatment use with increasing severity of SD: mild SD (18.2%), moderate SD (20%), and severe SD (50%), although the difference was not statistically significant (p=0.251). The mean duration of SD (in weeks) was longer in the severe group (19.75±7.44 weeks) than in the mild SD (18.18±6.27 weeks) and moderate SD (15.53±5.55 weeks); however, the difference was not statistically significant (p=0.256). Itch intensity, measured with the Visual Analog Scale (VAS), was higher in the severe SD group (median 8, range 3-9), compared with the mild (median 6, range 3-8) and moderate groups (median 5, range 1-8). These differences were not statistically significant (p=0.067). According to the Dermatology Life Quality Index (DLQI), 50% of participants in the severe SD group reported a severe impact on quality of life, compared with 27.3% in the mild SD and 11.8% in the moderate SD groups. Although there was a trend toward greater impact in the severe group, the difference was not statistically significant (p=0.76).

Comparison of IL-17 in the SD and non-SD groups

The measurement of IL-17 in the 66 participants was 0.086 (0.006-0.531) pg/µg protein. The median level in the SD group was 0.064 pg/µg protein (range: 0.006-0.354 pg/µg), whereas in the non-SD group it was 0.105 pg/µg protein (range: 0.030-0.531 pg/µg). This difference yielded a statistically significant p-value of 0.002. These results indicate that scalp IL-17 levels in hijab-wearing women with SD are lower compared to those without SD.

Correlation between scalp IL-17 levels in hijab-wearing women with SD and severity scores

Based on the correlation analysis between IL-17 levels and SD severity scores on the 16-point scale described by Zhang *et al*.,[14] strong negative correlation was observed ($r = -0.728$; $p<0.001$), indicating that lower IL-17 levels were associated with higher severity scores. This statistically significant

correlation reinforces the observed trend of lower IL-17 levels in the SD group compared to the non-SD group.

Discussion

There was no significant difference in age, occupancy, family history of SD, and nutritional status between the SD and non-SD groups. The proportion of participants with higher education was higher in the non-SD group; however, both groups generally had educational backgrounds ranging from senior high school to the undergraduate level, indicating a well-educated population.[16] Dyslipidaemia was higher in the SD group based on history taking; however, this study did not conduct laboratory studies necessary for the diagnosis of dyslipidaemia.[17] The duration of hijab usage was longer in the SD group (17.38±6.31 years) compared to the non-SD group (14.75±7.25 years), which was statistically significant (p=0.043). Long-term use of hijab may create a humid, poorly ventilated scalp environment, thereby impairing barrier function.[7] Scalp microbiome studies in hijab-wearing women also show a predominance of *M. restricta* and *Staphylococcus capitis*, species associated with SD severity and recurrence.[6]

This study shows a trend in which increasing SD severity is associated with greater itch intensity and a greater impact on quality of life. Severe cases were more likely to have used treatment, reported higher VAS scores, and showed greater DLQI impact, although differences were not statistically significant. Since all participants were women, who generally experience greater disruption in daily functioning, this may help explain the predominantly moderate-to-severe impairment in quality of life observed.[3,18]

The study found that scalp IL-17 levels in hijab-wearing women with SD were significantly lower than in those without SD (median 0.064 [0.006-0.354] *vs.* 0.105 [0.030-0.531] pg/µg protein; p=0.002). Reduced IL-17 levels may be associated not only with inflammatory processes but also with the broader physiological and pathological functions of this cytokine. Physiologically, IL-17 contributes to host defence and tissue repair, and several studies have demonstrated its role in immune responses against bacteria, fungi, and viruses.[19] Regarding antifungal defence, IL-17 has been implicated in superficial fungal infections, including *Candida* and *Malassezia*.[10,19]

The reduction in IL-17 may also be linked to *Malassezia*'s role in SD.[8,9] Although *Malassezia* has long been associated with inflammation in SD, the precise host response and inflammatory mechanisms remain unclear.[20,21] Nevertheless, several experimental models using epicutaneous inflammation support the role of IL-17 in epithelial defence and response to *Malassezia*. Recent studies have revealed that *Malassezia*, although a normal skin commensal, can elicit complex and

ambivalent IL-17-mediated immune responses: protective against over-colonization yet potentially exacerbating inflammation under acute conditions.[20-22]

The homeostatic response to *Malassezia* is dominated by IL-17, which is produced by T cells in normal conditions. Sparber *et al.*[10] showed that *Malassezia* triggers neutrophil infiltration and the expression of IL-17A/F, IL-22, and IL-23 in murine skin, regardless of barrier disruption. In this model, IL-17 or IL-23 deficiency resulted in failure to control *Malassezia* colonization. Jia *et al.*[11] also showed that *Malassezia globosa* induces pathogenic Th17 differentiation via keratinocyte-derived IL-23 through the TLR2/MyD88/NF-κB pathway, indicating that keratinocytes serve as a primary source of inflammatory signals in controlling *Malassezia*.

Ruchti *et al.*[22] found that $V\gamma4^+$ γδ T cells were the leading producers of IL-17A in mouse skin colonized by *Malassezia pachydermatis*. These cells exhibit memory-like behaviour, capable of recognizing *Malassezia* specifically without requiring antigen presentation by other cells, and persist long after colonization. Activation of IL-17 in these γδT17 cells depends on IL-23 and IL-1 but is independent of PRR signalling *via* toll-like receptors (TLRs) or C-type lectin receptors (CLRs). Miyachi *et al.*[23] found that *Malassezia furfur* induces IL-17-mediated skin inflammation through IL-36 receptor activation and MyD88 signalling in keratinocytes, even without barrier disruption. This suggests that *Malassezia* can directly stimulate inflammation in intact skin *via* the IL-36R/MyD88 axis, involving innate lymphoid cells type 3 (ILC3).

Ungar *et al.*,[20] using tape-stripping transcriptomic profiling, identified upregulation of the IL-17 pathway in facial SD lesions compared to normal skin. This activation included IL-17C, IL-36G, and antimicrobial peptides such as DEFB4, suggesting IL-17-driven inflammation in SD is largely Th1-mediated with minimal Th2 involvement.

Perkins *et al.*,[15] using a similar tape-stripping method as this study, reported reduced IL-1α and increased IL-2 in SD. IL-1α, an early epithelial signal for IL-17 activation, when reduced, indicates a weakened pathogen alarm system. Conversely, elevated IL-2 may reflect a shift toward regulatory immune activation or proliferation of other T cell subsets not directly involved in *Malassezia* clearance.

Malassezia is a commensal organism, evidenced by its ability to stimulate various PRRs, not only pro-inflammatory (*e.g.*, *via* TLR2) but also regulatory and tolerogenic pathways (*e.g.*, *via* Mincle, Dectin-1, Dectin-2, and Langerin), which are associated with transforming growth factor (TGF)-β and IL-10. The thick lipid layer of the *Malassezia* cell wall has been shown to increase intracellular IL-10 expression in keratinocyte cultures.[24]

Malassezia requires saturated fatty acids for survival; thus, human sebum serves as a source of saturated fatty acids, which are metabolized by *Malassezia* lipase (Mlip). The metabolism yields free

fatty acids, including oleic acid, which has irritant effects in SD by disrupting ceramides and increasing skin permeability. However, oleic acid also exhibits anti-inflammatory properties. These anti-inflammatory effects act *via* major histocompatibility complex (MHC) expression modulation, suppressing neutrophil proliferation[25] and enhancing regulatory T cell responses.[26]

El Mahi *et al.*[27] found that skin areas with high oleic acid in resolved psoriatic lesions showed lower expression of IL-17 transcription factors (SPRR2A, SPRR2D, DEFB4A) upon T cell activation in skin explants. A comparable IL-17-suppressing mechanism mediated by fungal lipase products was identified in Candida albicans, wherein Basso *et al.*[28] demonstrated that *C. albicans*-derived palmitic acid inhibits IL-17 production through dendritic cell pathways modulation.

Several studies on IL-17 inhibitors report an increased risk of superficial fungal infections, including *Malassezia*. The safety profile of secukinumab in pustular psoriasis showed a 1.05% incidence of superficial fungal infections.[29] Alam *et al.*[12] described a case of generalized tinea versicolor one month after ixekizumab treatment. SD itself has also been reported as a potential side effect of IL-17 inhibitors, though the incidence is low, as reported by Saeki *et al.* (8/91; 8.8%).[13]

This study supports the understanding that SD is a complex chronic inflammatory skin condition. IL-17 plays a crucial role in limiting *Malassezia* colonization through acute inflammatory responses *via* TLR2 and direct activation, while also being modulated by regulatory and tolerogenic responses *via* Mincle, Dectin-1/2, and Langerin. The scalp milieu in hijab-wearing women, characterized by increased transepidermal water loss (TEWL) and higher prevalence of *M. restricta*, may necessitate IL-17-driven homeostatic mechanisms. The observed reduction in IL-17 among hijab-wearing women may reflect a disrupted homeostatic response, contributing to the development of SD. This IL-17 reduction may be mediated by oleic acid, a Malassezia-derived metabolic product that functions as an IL-17 inhibitor. The proposed model of scalp immune homeostasis in hijab-wearing women, based on this study, is illustrated in Figure 1.

Correlation analysis demonstrated a strong negative correlation between IL-17 levels (pg/µg protein) and SD severity scores based on the 16-point scale ($r = -0.728$; $p = 0.000$). This indicates that IL-17 levels decrease as SD severity increases, and vice versa.

The severity score on the 16-point scale reflects increasing levels of scaling, erythema, and pruritus in SD, which are clinical signs of inflammation.[14] An increase in *M. restricta* and *M. globosa* has been associated with higher SD severity.[8] *Malassezia* secretes enzymes that hydrolyse triglycerides and phospholipids in sebum (lipases and phospholipases). This process generates large amounts of free fatty acids (*e.g.*, oleic acid), which directly disrupt cutaneous lipid homeostasis. These free fatty acids also exacerbate skin barrier dysfunction through mechanisms such as pH dysregulation, imbalance in the ceramide/cholesterol ratio, induction of parakeratosis, impaired stratum corneum

integrity, and activation of immune responses.[30] The immune response triggered by *Malassezia* and its lipid byproducts may involve dendritic cell activation. High-virulence *Malassezia* species such as *M. restricta* and *M. globosa* have been shown to upregulate caspase-1 and IL-1β expression in human keratinocytes.[8]

Studies have also shown that the absence of IL-17A/F or MyD88 signalling in keratinocytes leads to delayed *Malassezia* clearance due to a prolonged neutrophil-mediated inflammatory phase. This compensatory mechanism is less effective than IL-17-mediated clearance, resulting in persistent *Malassezia* colonization and chronic inflammation.[11,22]

Figure 2 illustrates the proposed model linking IL-17 levels to SD severity. IL-17 plays a crucial antifungal role in regulating *Malassezia* colonization. However, increased *Malassezia* growth and oleic acid production can suppress IL-17 responses. In parallel, *Malassezia* overgrowth disrupts the skin barrier through oleic acid-induced keratinocyte responses. These responses, including dendritic cell activation, increased IL-1β, and inflammasome signalling, lead to localized inflammation. Reduced IL-17 may shift the host's antifungal defence from IL-17-driven pathways to less effective neutrophil-dominant mechanisms, thereby promoting persistent colonization and chronic inflammation. This chain of events may explain the observed increase in SD severity.

Conclusions

This study showed IL-17 levels in women wearing hijab with SD were significantly lower compared to those without SD, with a significant negative correlation with SD clinical severity. This study was the first to explore IL-17 levels in the SD of hijab-wearing women using a non-invasive tape-stripping method. However, it had several limitations: it did not assess the dynamic changes of IL-17 due to its cross-sectional design; it did not measure other relevant cytokines, including the pro-inflammatory IL-1, IL-23, and IL-36, as well as the anti-inflammatory IL-10; it did not include a microbiome analysis; and it limited the correlation analysis to the SD severity score without ensuring a minimum number of participants in each severity category, as the study was designed as a preliminary investigation. Further research, based on this study's limitations, is advised to investigate the role of IL-17 in SD.

References

1. Jackson JM, Alexis A, Zirwas M, et al. Unmet needs for patients with seborrheic dermatitis. J Am Acad Dermatol 2022:1-8.
2. Dermatology and Venereology Department, Faculty of Medicine, Universitas Indonesia–Cipto Mangunkusumo Hospital (FKUI-RSCM). General Dermatology Outpatient Visits 2022–2023. Jakarta: Department of Dermatology & Venereology FKUI-RSCM; 2024.
3. Xuan M, Lu C, He Z. Clinical characteristics and quality of life in seborrheic dermatitis patients: a cross-sectional study in China. Health Qual Life Outcomes 2020;18:308-16.
4. Szepietowski JC, Reich A, Wesołowska-Szepietowska E, et al. Quality of life in patients suffering from seborrheic dermatitis: Influence of age, gender and education level. Mycoses 2009;52:357-63.
5. Araya M, Kulthanan K, Jiamton S. Clinical characteristics and quality of life of seborrheic dermatitis patients in a tropical country. Indian J Dermatol 2015;60:519-25.
6. Widaty S, Surachmiati L, Legiawati L, et al. Scalp microbiome of healthy women wearing hijab compared to those not wearing hijab: A cross-sectional study. Sci Rep 2023;13:1-9.
7. Etnawati K, Siswati, AS, Pudjiati SR, et al. The role of Malassezia sp, sebum level and Trans Epidermal Water Loss (TEWL) toward the dandruff severity between hijab and non hijab wearing subjects. J Med Sci 2018;50:351-6.
8. Adalsteinsson JA, Kaushik S, Muzumdar S, et al. An update on the microbiology, immunology and genetics of seborrheic dermatitis. Exp Dermatol 2020;29:481-89.
9. Wikramanayake TC, Borda LJ, Miteva M, et al. Seborrheic dermatitis-Looking beyond Malassezia. Exp Dermatol 2019;28:991-1001.
10. Sparber F, De Gregorio C, Steckholzer S, et al. The skin commensal yeast Malassezia triggers a type 17 response that coordinates anti-fungal immunity and exacerbates skin inflammation. Cell Host Microbe 2019;25:389-403.
11. Jia Q, Hu J, Wang X, et al. *Malassezia globosa* induces differentiation of pathogenic Th17 cells by inducing IL-23 secretion by keratinocytes. Mycopathologia 2024;189:85.
12. Alam HS, Ward JM, Davis LS. Generalized tinea versicolor following initiation of ixekizumab therapy. JAAD Case Rep 2021;18:54-6.
13. Saeki H, Nakagawa H, Nakajo K, et al. Japanese Ixekizumab Study Group. Efficacy and safety of ixekizumab treatment for Japanese patients with moderate to severe plaque psoriasis, erythrodermic psoriasis and generalized pustular psoriasis: Results from a 52-week, open-label, phase 3 study (UNCOVER-J). J Dermatol 2017;44:355-62.
14. Zhang F, Li Y, Ren W, et al. Establishment of clinical evaluation criteria for scalp seborrheic dermatitis. J Cosmet Dermatol 2023;22:3042-46.

15. Perkins MA, Cardin CW, Osterhues MA, et al. A non-invasive tape absorption method for recovery of inflammatory mediators to differentiate normal from compromised scalp conditions. Skin Res Technol 2002;8:187-93.
16. Arikunto S. Dasar-dasar Evaluasi Pendidikan [In Bahasa Indonesia]. Jakarta: Rineka Cipta; 2010, p. 173.
17. Aman AMA, Soewondo P, Soelistijo SA, et al. Panduan Pengelolaan Dislipidemia di Indonesia [In Bahasa Indonesia]. Jakarta: PB PERKENI; 2021.
18. Padovan M, Benincasa BB, Panduri S, et al. Assessing the burden of dermatological diseases on work life from a gender perspective. Sci Rep 2025;15:24014.
19. Huangfu L, Li R, Huang Y, et al. The IL-17 family in diseases: from bench to bedside. Signal Transduct Target Ther 2023;8:402.
20. Ungar B, Manson M, Kim M, et al. Tape-strip profiling identifies unique immune and lipid dysregulation in patients with seborrheic dermatitis. J Am Acad Dermatol 2025;92:1277-87.
21. Sparber F, LeibundGut-Landmann S. Host responses to *Malassezia* spp. in the Mammalian Skin. Front Immunol 2017;8:1614.
22. Ruchti F, Tuor M, Mathew L, et al. γδ T cells respond directly and selectively to the skin commensal yeast Malassezia for IL-17-dependent fungal control. PLoS Pathog 2024;20:e1011668.
23. Miyachi H, Wakabayashi S, Sugihira T, et al. Keratinocyte IL-36 Receptor/MyD88 signaling mediates Malassezia-Induced IL-17-dependent skin inflammation. J Infect Dis 2021;223:1753-65.
24. Thomas DS, Ingham E, Bojar RA, et al. In vitro modulation of human keratinocyte pro- and anti-inflammatory cytokine production by the capsule of Malassezia species. FEMS Immunol Med Microbiol 2008;54:203-14.
25. Chen CY, Lee YH, Chang SH, et al. Oleic acid-loaded nanostructured lipid carrier inhibits neutrophil activities in the presence of albumin and alleviates skin inflammation. Int J Nanomedicine 2019;14:6539e53.
26. Pompura SL, Wagner A, Kitz A, et al. Oleic acid restores suppressive defects in tissue-resident FOXP3 Tregs from patients with multiple sclerosis. J Clin Invest 2021;131:e138519.
27. El Mahi Y, Varin A, Vetter M, et al. Resolved psoriasis with abundant Oleic acid in stratum corneum exhibits lower T-cell-driven IL-17 signature. J Invest Dermatol 2023;143:2145-52.e6.
28. Basso P, Dang EV, Urisman A, et al. Deep tissue infection by an invasive human fungal pathogen requires lipid-based suppression of the IL-17 response. Cell Host Microbe 9;30:1589-1601.e5.
29. Fujishige A, Seko N. Safety and effectiveness of secukinumab in Japanese patients with generalized pustular psoriasis: A post-marketing surveillance. J Dermatol 2025;52:773-86.
30. Cheng Y, Cong J, Xu J, et al. Research progress on the exacerbation of lipid metabolism by Malassezia and its impact on the skin barrier function. Cosmetics 2025;12:67-77.

Table 1. Sociodemographic characteristics of subjects comparing scalp IL-17 levels in hijab wearing women with SD and without SD (n=66).

Characteristic	**Non-SD (n=32)**	**SD (n=34)**	**p-value**
Age (years), median	35.78±10.18	37.94±9.19	0.368[a]
Education, n (%)			
Lower level education	0 (0)	0 (0)	0.048[b]
Middle level education	11 (34.4)	21 (61.8)	
Higher level education	21 (65.6)	13 (38.5)	
Occupancy, n (%)			
Resident	4 (12.5)	1 (2.6)	0.317[b]
Civil servant/private employee	22 (68.8)	28 (82.4)	
Other	6 (18.8)	5 (14.7)	
Duration of hijab use (years)	13 (5-35)[*]	17.38±6.32[**]	0.043[c]
History of diseases other than SD, n (%)			
HIV/AIDS	0 (0)	0 (0)	NA
Dyslipidemia	1 (3.1)	9 (26.5)	0.013[d]
Hypertension	2 (6.3)	2 (5.9)	1.000[d]
Diabetes mellitus	1 (2.9)	1 (3.1)	1.000[d]
Heart disease	0 (0)	0 (0)	NA
Family history of SD, n (%)	13 (40.6)	18 (52.6)	0.45[b]
History of active smoking habits, n (%)	0 (0)	0 (0)	NA
Nutritional status, n (%)			
Low risk of metabolic syndrome (underweight and normal)	10 (31.3)	6 (17.6)	0.317[b]
High risk of metabolic syndrome (overweight and obese)	22 (68.8)	28 (82.4)	

SD, seborrheic dermatitis; NA, not applicable; [*]median (minimum-maximum); [**]mean ± standard deviation; [a]unpaired t-test; [b]continuity correction; [c]Mann-Whitney test; [d]Fisher's Exact Test.

Table 2. Clinical characteristics between mild, moderate, and severe SD (n=34).

Characteristics	Mild SD n=11 (32.4%)	Moderate SD n=15 (44.1%)	Severe SD n=8 (23.5%)	p-value
Treatment history, n (%)				
None	9 (81.8)	12 (80)	4 (50)	0.251[a]
Topical medication	2 (18.2)	3 (20)	4 (50)	
Systemic medication	0 (0)	0 (0)	0 (0)	
Duration of SD (months)*	18.18±6.27	15.53±5.55	19.75±7.44	0.256[b]
Itching complaints (VAS)	5.64±1.43*	4.93±1.75*	8 (3-9)**	0.067[c]
DLQI, n (%)				
No effect	0 (0)	0 (0)	1 (12.5)	0.76[a]
Small effect	4 (36.4)	2 (13.3)	1 (12.5)	
Moderate effect	4 (36.4)	5 (44.4)	2 (25)	
Very large effect	3 (27.3)	3 (11.8)	4 (50)	
Extremely large effect	0 (0)	0 (0)	0 (0)	

SD, seborrheic dermatitis; DLQI, Dermatology Life Quality Index; VAS, Visual Analog Scale; *mean±standard deviation, **median (minimum-maximum); [a]likelihood ratio; [b]Analysis of Variance; [c]Kruskal-Wallis test.

Figure 1. Model of IL-17 role in scalp SD among hijab-wearing women compared to non-SD controls.

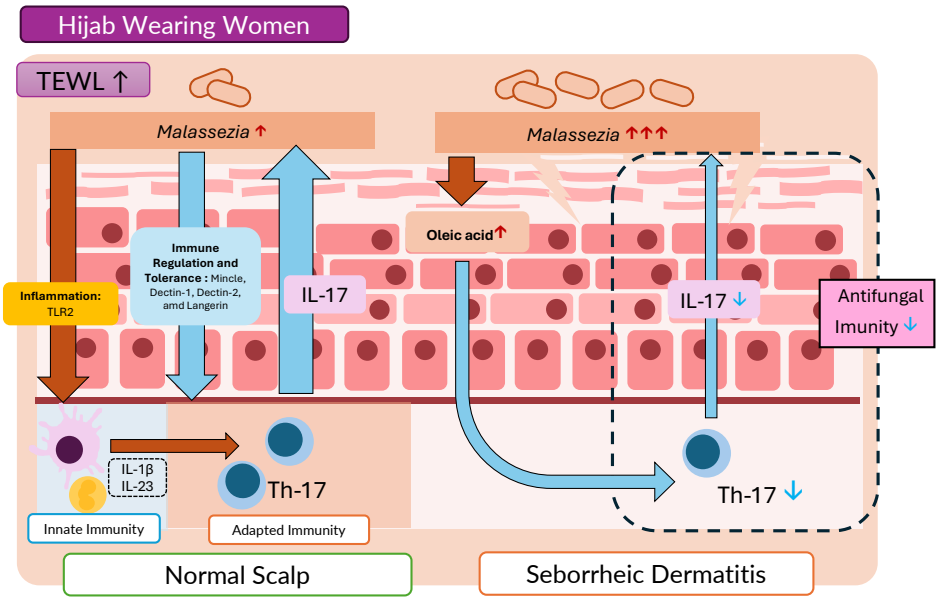

Figure 2. Model of IL-17 correlation with SD severity.