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SEBORRHEIC DERMATITIS, INSULIN RESISTANCE, AND METABOLIC SYNDROME: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS - A NARRATIVE REVIEW

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ABSTRACT

Background: Seborrheic dermatitis (SD) is a common, chronic, and recurrent inflammatory skin condition. Available data increasingly suggest a possible association between SD and metabolic disorders, particularly insulin resistance (IR) and metabolic syndrome (MetS).

Objective: The purpose of this study was to evaluate the current evidence regarding the association between SD and IR and MetS, the potential biological mechanisms, and the clinical significance of these associations.

Methods: A narrative review was conducted of English-language literature published between 2005 and 2026, retrieved from the PubMed/MEDLINE database between July and September 2026.

Key findings: The most consistent findings concern insulin resistance (IR). Patients with SD were found to have higher fasting insulin levels and higher HOMA-IR values, as well as a higher prevalence of insulin resistance. Findings regarding metabolic syndrome are less consistent, although some studies report a higher prevalence of metabolic syndrome and abnormalities in lipid profile, waist circumference, and blood pressure. Potential mechanisms linking SD to metabolic disorders include chronic inflammation, oxidative stress, and abnormalities in insulin signaling and lipid metabolism. However, their direct role in the pathogenesis of SD has not yet been confirmed.

Conclusions: An analysis of the available data suggests a possible association between SD, IR and metabolic disorders. However, it does not allow conclusions to be drawn regarding causality. The results obtained underscore the importance of awareness of potential metabolic risk, but they do not justify routine screening. Therefore, larger, prospective, and methodologically standardized studies are needed.

KEYWORDS

Seborrheic Dermatitis, Insulin Resistance, Metabolic Syndrome, HOMA-IR, Metabolic Dysfunction

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1. Introduction

1.1. Seborrheic dermatitis: clinical and epidemiological background

Seborrheic dermatitis (SD) is a common, chronic, and recurrent inflammatory skin condition. Its characteristic symptoms include erythematous lesions covered with white or yellowish, often greasy scales [1,2]. They commonly occur in areas with high sebaceous gland activity, particularly the scalp and face - including the areas around the eyebrows and the nasolabial folds - as well as around the ears, chest, and other seborrheic areas [1]. The lesions may be accompanied by itching. The clinical presentation, including the morphology and severity of the lesions, depends on age and individual skin characteristics [1,2]. Data from a recent meta-analysis, which includes more than 1.26 million people, indicate that the global prevalence of SD is approximately 4.38%, and among adults, it is estimated at 5.64% [3]. The prevalence of SD varies depending on age, sex, and geographic region. The condition is more common in men and older adults [2,3]. Although SD is not a life-threatening condition, its chronic and recurrent nature makes it clinically significant. Patients report that their symptoms affect their daily functioning and impair their psychological and social well-being [2,4]. A recent meta-analysis indicated a moderately severe decline in quality of life on average, with the severity of this decline increasing with the severity of the disease [4].

1.2. Current understanding of seborrheic dermatitis pathophysiology

The pathophysiology of seborrheic dermatitis depends on many factors and results from the interaction between the skin microbiome, sebaceous gland activity, epidermal barrier dysfunction, and an abnormal immune response [2,5,6]. Lipophilic yeasts of the genus *Malassezia*, particularly *M. restricta* and *M. globosa*, which metabolize lipids present in sebum, are considered to play a key role. They produce lipases that result in the formation of free fatty acids, including oleic acid. These products may exacerbate irritation, scaling, and

the inflammatory response in susceptible individuals [5,6,7]. The mere presence of *Malassezia* does not determine the development of SD, as these yeasts are part of the normal skin microbiome. The relationship between their abundance and the severity of the disease remains unclear [7,8]. Changes in the composition of the bacterial and fungal microbiota are also observed in SD, indicating a broader dysbiosis of the microbiome [5,6,7]. The function of the sebaceous glands and the lipid composition of the skin surface also play a significant role in the pathophysiology of SD. The integrity of the epidermal barrier may be compromised due to an abnormal lipid organization in the stratum corneum, including ceramides. As a result, its permeability to microbial metabolites increases [2,5]. Furthermore, the activation of innate and adaptive immune mechanisms leads to increased expression of pro-inflammatory mediators and activation of the Th1, Th17, and Th22 pathways [5,6,9]. The course of these processes also depends on individual and environmental factors, including, among others, genetic predisposition, androgenic activity, stress, and climatic conditions [6,8]. According to current data, SD should be viewed as a complex inflammatory dermatosis in which microbiological factors interact with immunological, lipid, and barrier disorders [2,7,9].

1.3. Insulin resistance and metabolic syndrome: biological and clinical context

In insulin resistance (IR), there is a reduced response to insulin in tissues such as skeletal muscle, the liver and adipose tissue. This leads to disturbances in the regulation of glucose uptake and production, which, in the early stages of the condition, are compensated for by increased insulin secretion and hyperinsulinemia, thereby maintaining glucose homeostasis for a certain period [10]. In clinical and epidemiological studies, insulin resistance is usually estimated using the HOMA-IR (Homeostatic Model Assessment of Insulin Resistance) index, which is calculated from fasting glucose and insulin concentrations [10]. Metabolic syndrome (MetS) is a group of metabolic disorders that increase the risk of type 2 diabetes and cardiovascular disease. Harmonised clinical criteria identify five main disorders: central obesity, impaired fasting glucose, hypertriglyceridemia, low HDL cholesterol (HDL-C) and high blood pressure. To diagnose metabolic syndrome, at least three of these conditions must be present [11]. An important component of the pathophysiological mechanism of MetS is visceral adipose tissue dysfunction, which contributes to lipid metabolism disorders, insulin resistance and low-grade chronic inflammation [12]. Alongside these disturbances, oxidative stress also increases, which further exacerbates metabolic dysfunction through excessive production of reactive oxygen species, lipid peroxidation and disturbances in redox signalling [13].

1.4. Emerging evidence linking seborrheic dermatitis with insulin resistance and metabolic syndrome

Evidence emerging in recent years suggests that seborrheic dermatitis may co-occur with metabolic disorders, particularly insulin resistance and metabolic syndrome. Case-control studies have shown that patients with seborrheic dermatitis have, among other things, higher insulin levels and HOMA-IR values, a higher prevalence of insulin resistance, and abnormalities in selected components of metabolic syndrome, such as triglyceride and HDL cholesterol levels, waist circumference, and blood pressure [14,15,16]. The findings regarding metabolic syndrome itself remain inconclusive. Some studies show that it occurs more frequently in patients with SD, while others do not indicate a statistically significant difference compared to control groups [14,15,16]. There is a broader meta-analysis that demonstrates an association between SD and metabolic syndrome. However, the data were derived from a small number of studies [17]. Observations to date therefore suggest a possible association between SD and metabolic disorders. However, they do not allow us to determine the nature or direction of this relationship or to establish a causal link.

1.5. Rationale and objective

Despite growing interest in the systemic determinants of seborrheic dermatitis, the role of metabolic disorders in this disease remains poorly understood. Review articles published to date have focused primarily on a broader spectrum of systemic comorbidities, dietary factors, and obesity in seborrheic dermatitis, whereas data on insulin resistance and metabolic syndrome have not yet been described as a single, interrelated area [17,18,19]. An important issue is determining the relationship between SD and insulin resistance and metabolic syndrome, as well as its potential impact on clinical management. This narrative review aims to provide a critical synthesis of the current evidence regarding these relationships, analyze the potential biological mechanisms underlying them, and assess the possible clinical implications.

2. Methodology

2.1. Study design and scope

The following paper is a narrative review of the literature on the association between seborrheic dermatitis (SD) and insulin resistance (IR) and metabolic syndrome (MetS). The review included data on the prevalence of IR and MetS in individuals with SD, the individual components of metabolic syndrome, factors that may modify the observed associations, and their potential clinical significance. The analysis also considered possible biological mechanisms linking SD to metabolic disorders. The review was based on human studies, while experimental data were included selectively to elucidate potential pathophysiological mechanisms.

2.2. Literature search strategy

The literature review was conducted in the PubMed/MEDLINE database from July to September 2026. The following search terms were used: “seborrheic dermatitis” AND “insulin resistance” and “seborrheic dermatitis” AND “metabolic syndrome.” To identify publications addressing specific aspects of the topic under analysis, the search was expanded to include terms related to carbohydrate and lipid metabolism disorders, obesity, and potential biological mechanisms, including “HOMA-IR,” “hyperinsulinemia,” “obesity,” “lipid metabolism,” “inflammation,” “oxidative stress,” “IGF-1,” “sebaceous glands,” and “skin barrier.” The publications included in the review were published between 2005 and 2026.

2.3. Eligibility criteria

This review included peer-reviewed English-language publications on the association between SD and IR and MetS, or their components, including observational studies, systematic reviews, meta-analyses, and relevant narrative reviews. Studies examining factors that may influence the relationships under analysis, such as obesity, anthropometric parameters, lifestyle, and dietary factors, were also included in this review. Experimental studies were used selectively to illustrate potential mechanisms linking metabolic disorders to the pathophysiology of SD. Publications not directly related to the scope of the review, duplicates, and non-peer-reviewed materials were excluded.

2.4. Study selection and data synthesis

Eligible publications were analyzed in terms of study design, characteristics of the study population, methods for diagnosing and assessing the severity of SD, methods for assessing IR and MetS and their individual components, as well as potential confounding factors and key clinical outcomes. Data on potential biological mechanisms that could explain the observed associations were also evaluated. Given the heterogeneity of the available studies, the results were synthesized narratively, without conducting a meta-analysis or a formal risk-of-bias assessment.

3. Results

3.1. Characteristics of the available evidence

The available data describing the association between seborrheic dermatitis and insulin resistance and metabolic syndrome come mainly from observational studies published between 2014 and 2025. Most of these are small, single-center case-control studies that were conducted primarily in adult populations [14,15,16]. Analyses conducted in subsequent years included a more detailed assessment of carbohydrate metabolism, body composition, adipokines, oxidative stress, and dietary factors [20,21]. A large cross-sectional population study based on electronic health records was also published, involving more than 18,000 individuals with or without a diagnosis of SD [22]. Case-control studies involved relatively small groups, typically consisting of several dozen people with SD and several dozen people in the control group [14,16,20]. For example, Erdoğan et al. described a group of 53 patients with SD and 50 control subjects [14], while Semiz et al. included 49 and 40 participants, respectively [20]. The diagnosis of SD in most studies was based on clinical assessment, and the severity of skin lesions was described using standardized scales, primarily the Seborrheic Dermatitis Area and Severity Index (SDASI) [14,20,21]. In a population-based study, the identification of patients with SD was based on a dermatological diagnosis recorded in medical records [22]. The methods used to assess metabolic disorders in the available literature vary. Parameters such as BMI and waist circumference, blood

pressure, fasting blood glucose, and lipid profile were analyzed [14,15,16]. In studies focusing on insulin resistance, insulin levels were also measured and the HOMA-IR index was calculated [14,20]. Sometimes, HbA1c levels and an oral glucose tolerance test were additionally included to assess carbohydrate metabolism [14]. The diagnostic criteria for metabolic syndrome varied across studies, with some using the criteria of the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) [16,20]. The scope of metabolic assessment also varied and, in selected studies, additionally included body composition analysis [21] and measurements of adiponectin, leptin, and markers of oxidative stress [20].

3.2. Seborrheic dermatitis and insulin resistance

A case-control study by Erdoğan et al., which evaluated 53 adult patients with seborrheic dermatitis and 50 healthy individuals matched for age, sex, and BMI, provides the most accurate data on insulin resistance in patients with seborrheic dermatitis. The analysis included fasting blood glucose and insulin levels, HbA1c, HOMA-IR, and glucose concentration two hours after administration of 75 g of glucose in an oral glucose tolerance test (OGTT). A HOMA-IR value > 2.5 was established as indicative of insulin resistance [14]. The study found higher fasting insulin levels in patients with SD compared to the control group. A similar trend was observed for HOMA-IR, indicating reduced insulin sensitivity in the SD group [14]. Furthermore, it should be noted that these differences were also reflected in the prevalence of insulin resistance: 31 of 53 patients with SD (58.49%) met the HOMA-IR criterion of > 2.5 , compared with 11 of 50 control subjects (22%; $p=0.0001$) [14]. This result is one of the most clear-cut indications that impaired insulin sensitivity may be more common in individuals with SD. At the same time, not all indicators of carbohydrate metabolism differed between the groups. Although fasting blood glucose was higher in the SD group, this difference was not statistically significant [14]. The opposite pattern was observed after a glucose tolerance test: glucose concentration at 120 minutes during the OGTT was significantly higher in patients with SD. When considered alongside elevated fasting insulin levels and HOMA-IR values, these results suggest that insulin resistance may be present despite the absence of significant differences in fasting glucose levels [14]. A more recent study by Semiz and Aktaş yielded results consistent with this line of observation. In a group of 49 patients with SD, the fasting insulin concentration averaged 14.2 ± 10.5 mU/L, whereas in 40 healthy individuals it was 7.7 ± 3.1 mU/L ($p<0.001$). Additionally, the mean HOMA-IR value was nearly twice as high in the SD group: 3.29 ± 3.01 versus 1.74 ± 0.73 in the control group ($p=0.001$). Fasting glucose levels, however, did not differ significantly between the groups (89.7 ± 10.2 vs. 87.2 ± 10.1 mg/dL; $p=0.100$) [20]. In the analyzed study, a slightly higher threshold value for insulin resistance was adopted, defined as a HOMA-IR ≥ 2.9 , which indicates methodological differences between individual studies. Another important aspect is the potential association between impaired insulin sensitivity and the severity of SD. Erdoğan et al. reported a positive correlation between SDASI scores and both fasting insulin levels ($r = 0.337$; $p = 0.014$) and HOMA-IR ($r = 0.300$; $p=0.029$). As disease severity increased, higher glucose levels were observed at the 2-hour mark of the OGTT ($r=0.223$; $p=0.024$). However, this association was not observed for fasting glucose levels or HbA1c levels. It should be emphasized that no similar associations were found for the subjective assessment of disease severity [14]. The association between the severity of SD and insulin resistance parameters was not confirmed in the study by Semiz and Aktaş. After comparing groups of patients with mild and moderate-to-severe SD, no significant differences were found in fasting insulin levels ($p=0.615$), HOMA-IR values ($p=0.665$), or fasting blood glucose ($p=0.763$). However, the authors observed that a greater severity of skin lesions was associated with a higher glycemic index and glycemic load of the diet, rather than with the assessed markers of insulin resistance [20]. Data on the duration of SD are even more incomplete. In a study by Erdoğan et al., no significant associations were found between disease duration and fasting insulin levels ($p=0.57$), HOMA-IR ($p = 0.40$), fasting blood glucose ($p = 0.30$), and HbA1c ($p = 0.071$). The only statistically significant association was observed for glucose levels measured at 120 minutes during the OGTT, which showed a weak positive correlation with disease duration ($r = 0.217$; $p = 0.028$) [14]. In summary, the available data suggest a higher prevalence of insulin resistance in patients with SD, which manifests primarily as elevated fasting insulin levels and higher HOMA-IR values. At the same time, fasting glucose levels may not differ significantly from those observed in individuals without SD [14,20]. Similar results have also been reported in broader studies on metabolic disorders associated with skin diseases [17,23]. It should be emphasized, however, that the small number of studies directly assessing this relationship, the small sample sizes of the analyzed groups, and the inconclusive results regarding the association between insulin resistance and the severity and duration of SD limit the possibility of an unambiguous interpretation of these relationships.

3.3. Seborrheic dermatitis and metabolic syndrome

In contrast to the relatively consistent findings regarding insulin resistance described in the previous section, the results of studies analysing the presence of full-blown metabolic syndrome in patients with seborrheic dermatitis are more varied. In a study by Imamoğlu et al., which included 47 patients with seborrheic dermatitis (SD) and 36 healthy individuals, MetS was diagnosed according to the NCEP ATP III criteria. Metabolic syndrome was diagnosed in 9 patients with SD (19.1 per cent) and 3 individuals from the control group (8.3 per cent). The difference was not statistically significant ($p=0.216$), even though the prevalence of MetS in the SD group was more than twice as high [16]. In the study by Erdoğan et al., which also used the NCEP ATP III criteria, the findings were similar. Metabolic syndrome was diagnosed in 12 out of 53 patients with SD (22.64%) and in 6 out of 50 healthy individuals (12%). In this study, too, the difference was not statistically significant ($p=0.155$), despite the higher prevalence of metabolic syndrome in the SD group [14]. A study by Sobhan et al. yielded similar results, with MetS diagnosed in 20.5% of patients with SD and 7.7% of the control group, although, once again, the difference was not statistically significant ($p=0.104$) [17]. Thus, in all three studies, the prevalence of MetS was higher in individuals with SD. However, it was not possible to demonstrate a significant difference between the groups, due to limitations associated with small sample sizes. Akbaş et al. reported conflicting study results. The study involved 54 people with SD and 47 healthy control subjects. MetS was diagnosed in 35.2% of those with SD and 10.6% of the healthy subjects, and the results were statistically significant ($p=0.004$) [15]. When interpreting the differences between the studies, however, attention should be paid to the definitions of MetS used. Older studies [14,16] used the NCEP ATP III criteria, whereas Akbaş et al. applied the NCEP ATP III/AHA-NHLBI (The American Heart Association - National Heart, Lung, and Blood Institute) criteria, which included a lower fasting blood glucose threshold and corresponding waist circumference values. The discrepancy between the studies can therefore be partly explained by differences in the diagnostic protocols, which may have influenced the number of participants meeting at least three MetS criteria. A meta-analysis by Sodagar et al. provides a new perspective, in which the two case-control studies available at the time were included in the analysis of SD. A total of 181 participants took part in the studies, and the individual studies did not show a significant association between SD and MetS. However, the authors found an OR of 2.45 for the occurrence of MetS in conjunction with SD, following the pooling of data, which suggests that people with SD are more likely to develop metabolic syndrome [17]. When interpreting the results, however, attention should be paid to the small number of studies on SD included in the analysis. Analysing the individual components of MetS helps to identify more precisely which disorders lead to the observed metabolic signal. Lipid abnormalities are one of the most frequently observed findings. Imamoğlu et al. observed low HDL-C levels in 31 out of 47 patients with SD compared with 15 out of 36 individuals in the control group ($p=0.044$), whilst the prevalence of hypertriglyceridaemia did not show any significant differences between the groups. Furthermore, HDL-C levels showed a negative correlation with the severity of SD as assessed by the SDASI ($r = -0.312$; $p = 0.033$), indicating that lower HDL-C levels were associated with more severe skin lesions [16]. Lipid abnormalities were also observed in other studies. Erdoğan et al. reported higher triglyceride concentrations in patients with SD. Moreover, triglyceride levels correlated positively with the SDASI ($r = 0.355$; $p = 0.009$). At the same time, no association was observed between TG levels and disease duration [14]. In a study by Akbaş et al., elevated triglycerides were found significantly more frequently in patients with SD ($p = 0.015$), whilst HDL-C levels were lower than in the control group ($p = 0.050$). The authors also noted a higher prevalence of abdominal obesity in the SD group [15]. However, the findings regarding central obesity are not entirely consistent. In the study by Imamoğlu et al., abdominal obesity was observed in 10 out of 47 patients with SD and in 3 out of 36 individuals in the control group. However, the difference was not statistically significant ($p=0.135$) [16]. Erdoğan et al., on the other hand, found a larger waist circumference in the group of patients with SD, and BMI correlated positively with the SDASI ($r = 0.298$; $p=0.030$), although this relationship was not associated with disease duration [14]. These findings suggest that obesity parameters are not consistently abnormal across all studies, but may contribute to the metabolic profile of SD. The next component is blood pressure. In a small study by Imamoğlu et al., no significant difference in the prevalence of hypertension was found between the groups [16]. However, Akbaş et al. reported higher systolic and diastolic blood pressure readings in patients with SD ($p=0.016$ and $p=0.029$, respectively) [15]. Significant support for this finding is provided by a large population-based study by Linder et al., which included 9,255 patients with SD and 9,246 individuals without SD. Hypertension was observed in 27.1% and 24.7% of participants respectively (OR = 1.13; 95% CI: 1.05–1.21), and after adjusting for factors including age, sex, smoking, obesity and diabetes, the association remained significant (adjusted OR = 1.23; 95% CI: 1.12–1.35; $p < 0.001$) [22]. When measuring

fasting blood glucose, however, the distinction between patients with SD and healthy individuals is less clear-cut. In the studies by Erdoğan et al. and Imamoğlu et al., no significant difference was found in this parameter [14,16]. In the study by Imamoğlu et al., hyperglycaemia was additionally observed in 6 out of 47 patients with SD and 2 out of 36 individuals in the control group ($p=0.456$) [16]. In the context of the findings regarding insulin and HOMA-IR, described in section 6.2, this may suggest that insulin resistance can be detected more rapidly and more definitively than abnormal fasting blood glucose levels. The available data also do not show a clear association between MetS as a whole and the severity or duration of SD. Imamoğlu et al. found no association between disease duration and MetS, nor any significant differences in metabolic syndrome parameters between mild and moderate-to-severe SD [16]. Similarly, Akbaş et al. found comparable SDASI scores and disease duration in patients with and without MetS [15]. In summary, the results suggest that MetS may be more common in patients with SD. However, the evidence from individual studies is inconclusive. A pattern comprising selected components of the syndrome, mainly increased waist circumference or obesity, dyslipidaemia and elevated blood pressure, is more conclusive than a diagnosis of the full syndrome [14,15,16,22]. When considered alongside the higher prevalence of insulin resistance described earlier, these findings suggest a broader pattern of metabolic abnormalities in patients with SD. However, their significance and correlations require cautious interpretation.

3.4. Factors potentially modifying the associations

Current data suggest that the association between seborrheic dermatitis and metabolic disorders is influenced by factors such as demographic characteristics, anthropometric parameters, disease severity, and lifestyle. In their study, Erdoğan et al. matched groups by age, sex, and BMI. Among the group of patients with seborrheic dermatitis (SD), a higher SDASI score correlated positively with age ($r = 0.393$; $p=0.004$) and BMI ($r=0.298$; $p=0.030$). At the same time, the mean BMI did not differ significantly between the group of patients with SD and the control group, whereas body weight and waist circumference were significantly higher in the group of individuals with SD [14]. However, the findings regarding the association between adiposity and SD were not consistent. Akbaş et al. reported a higher BMI in patients with SD compared to the control group (26.9 vs. 23.5 kg/m²; $p=0.002$) [15]. Özgül et al. obtained different results, finding no significant differences in BMI or body composition parameters [21]. Furthermore, in the latter study, body composition parameters did not correlate significantly with the SDASI [21]. Age and the course of SD may also be relevant to the observed associations with metabolic disorders. A study by Akbaş et al. showed that individuals with SD and comorbid metabolic syndrome were older, and the disease onset occurred at a later age in this group compared to those with SD without metabolic syndrome. However, no significant differences were found between these groups in terms of gender, disease duration, or SD severity as assessed by the SDASI [15]. Erdoğan et al. observed that the duration of SD did not correlate with most of the analyzed variables. Significant associations were found only for the 2-h PG (2-h plasma glucose) value from the OGTT and for smoking [14]. Additionally, Imamoğlu et al. found no significant difference in the duration of SD between individuals with and without metabolic syndrome. However, they observed a negative correlation between SD severity and HDL-C levels, meaning that higher SDASI scores were associated with lower HDL-C levels [16]. Family history and lifestyle factors may also play an important role. Erdoğan et al. demonstrated that patients with SD were more likely than those in the control group to have a positive family history of metabolic disorders (66.0% vs. 38.0%; $p=0.004$) and to be smokers (49.1% vs. 30.0%; $p=0.048$) [14]. Similarly, Imamoğlu et al. observed a higher prevalence of a family history of metabolic diseases in patients with SD (78.7% vs. 55.6%; $p=0.032$) [16]. Current data suggest that dietary habits may also be a potential modifier of the observed associations. Semiz et al. found that individuals with SD had a higher glycemic load and lower total dietary antioxidant capacity compared to those in the control group. Furthermore, a higher glycemic index and glycemic load were associated with more severe seborrheic dermatitis. Some of these associations remained significant even after adjusting for BMI, components of metabolic syndrome, and HOMA-IR [20]. A synthesis of these results suggests that age, central obesity and BMI, family history of metabolic disorders, smoking, and diet are factors that may partially modify the observed associations between seborrheic dermatitis and insulin resistance and metabolic syndrome. In contrast, the significance of gender and the duration of SD remains less clear. The results continue to be heterogeneous due to differences in the characteristics of the study populations and in the exclusion criteria used, including comorbidities and the use of medications that affect metabolic parameters [14,15,20].

3.5. Potential pathophysiological links between SD, IR, and MetS

3.5.1. Chronic low-grade inflammation

One possible mechanism linking SD to IR and MetS is low-grade chronic inflammation. In the course of SD, local activation of keratinocytes and immune cells is accompanied by increased expression of pro-inflammatory mediators and the involvement of Th1- and Th17-dependent immune responses [8,9]. Elevated cytokine production has been observed in the skin of individuals with SD, and increased serum concentrations of IL-2 and IFN- γ have also been noted, suggesting that the inflammatory process may not be limited solely to the skin [8]. Chronic low-grade inflammation is also one of the components of the pathophysiology of insulin resistance and metabolic syndrome, and the release of pro-inflammatory cytokines may affect insulin signaling pathways, contributing to the persistence of metabolic disorders [23]. The coexistence of immune activation within the skin in SD and the systemic inflammation accompanying IR and MetS may therefore suggest the involvement of partially shared pathophysiological mechanisms. It should be noted, however, that these associations do not currently allow for the determination of a relationship or the confirmation of its causal nature [8,23].

3.5.2. Insulin and IGF-related signaling

Another potential link between IR, MetS and SD is impaired insulin and IGF-1 signalling. Compensatory hyperinsulinaemia, which often accompanies insulin resistance, may affect insulin- and IGF-1-dependent pathways [24]. This may affect SD, given that sebaceous glands are metabolically active structures, and insulin and IGF-1 can alter the function of sebocytes and lipid synthesis [25,26]. Experimental studies have shown that IGF-1 increases the expression of SREBP-1, which is a key regulator of lipogenic genes, by activating the PI3K/Akt pathway, leading to increased lipogenesis in sebocytes [25]. Furthermore, exposure of cultured sebocytes to IGF-1 resulted in increased expression of IGF1R, SREBP and PI3KCA, as well as increased sebum production [26]. As the function of the sebaceous glands and the composition of sebum play a role in the pathophysiology of SD, increased IGF-1 signalling and hyperinsulinaemia may represent a plausible mechanism linking metabolic disorders to SD. However, conclusive evidence of this relationship in SD remains incomplete [24,25,26].

3.5.3. Oxidative stress

Oxidative stress may be one of the pathophysiological mechanisms linking seborrheic dermatitis to metabolic disorders. Studies have shown that patients with seborrheic dermatitis exhibited reduced total antioxidant capacity as well as elevated total oxidative status and oxidative stress indices. These findings suggest a redox imbalance characterized by a predominance of pro-oxidative processes [16,20]. The role of oxidative mechanisms in the pathophysiology of SD is further supported by disturbances in thiol-disulfide homeostasis [27]. At the same time, excessive production of reactive oxygen species and impaired antioxidant mechanisms are key components of insulin resistance and metabolic syndrome. These disturbances may contribute to abnormal insulin signaling, lipid peroxidation, endothelial dysfunction, and chronic inflammation [13]. It should be emphasized that increased oxidative stress in SD was also observed after adjusting for BMI, components of metabolic syndrome, and HOMA-IR [20]. The abnormalities in the oxidative-antioxidant balance observed in both SD and IR as well as MetS point to a potential role for oxidative stress as a common biological mechanism [13,20,28]. However, the available literature lacks sufficient evidence to determine the direction of this association or establish its causal nature [20,27].

3.5.4. Lipid metabolism and sebaceous activity

Abnormalities in lipid metabolism may be one of the additional mechanisms common to SD, IR, and MetS. Lipid metabolism disorders often coexist with insulin resistance, while the sebaceous glands remain metabolically active, and their function is regulated by hormonal factors [24,25]. Insulin and IGF-1 may enhance lipogenesis in sebocytes, in part by activating the PI3K/Akt pathway and SREBP-1, leading to increased lipid synthesis [29,30]. However, in the pathogenesis of SD, it is not only the quantity of sebum produced that may be significant, but also its qualitative composition. Yeasts of the genus *Malassezia* utilize the triglycerides present in sebum, leading to the release of free fatty acids, particularly unsaturated fatty acids. These compounds may affect the integrity of the epidermal barrier and contribute to the exacerbation of the inflammatory response [26,31]. Furthermore, the results of recent lipidomic studies indicate changes in the lipid profile of sebum in patients with SD [29].

3.5.5. Skin barrier and microbial environment

Disorders of the microbiome and the skin barrier may be the factors linking the metabolic mechanisms described earlier to the pathophysiology of SD. In diseased skin, increased transepidermal water loss and abnormalities in ceramide composition have been observed, indicating impairments in barrier integrity [2,32]. At the same time, changes in the bacterial microbiome are observed in SD, such as, amongst others, a reduced proportion of *Cutibacterium* and an increased proportion of *Staphylococcus* [33,34]. The role of *Malassezia* is more complex. Although yeast counts do not show a consistent correlation with the presence or severity of SD, its lipid metabolites may contribute to barrier dysfunction and exacerbate inflammatory processes [2,32,34]. Although a direct role in the association between SD, IR and MetS has not yet been confirmed, the body of available evidence points to the existence of interrelationships between the lipid environment, the inflammatory process, the microbiome and the skin barrier [9,32].

3.6. Summary of the evidence

Given the above findings, it can be surmised that seborrheic dermatitis is associated with metabolic disorders, although the evidence regarding insulin resistance appears more consistent than that regarding metabolic syndrome as a whole. Patients with SD had higher fasting insulin levels, higher HOMA-IR values, and a higher prevalence of insulin resistance, despite the absence of significant differences in fasting glucose levels [14,20]. Data on the association between SD and metabolic syndrome show greater variability. Some studies have demonstrated a significantly higher prevalence of MetS in patients with SD [15,20], while other studies have reported only a numerically higher prevalence that did not reach statistical significance [14,16]. Additionally, a meta-analysis reported a positive association between SD and MetS. However, this analysis was based on a small number of studies on SD [17]. In various studies, patients with SD exhibited abnormalities such as increased waist circumference, hypertriglyceridemia, and reduced HDL-C levels [14,15,16]. Furthermore, they also had higher blood pressure readings and a higher prevalence of hypertension [15,22]. However, the results regarding BMI and body composition were inconclusive. Data from one study did not indicate significant differences in BMI, body fat percentage, or visceral adiposity between patients with SD and those in the control group [21]. The available data therefore suggest that seborrheic dermatitis may co-occur with certain features of metabolic dysfunction. However, these findings are not entirely consistent [14,15,20]. The interpretation of these associations is limited by small sample sizes, differences in the MetS criteria used and population characteristics, and the observational nature of the available studies. These factors also make it difficult to determine the direction of the observed associations and do not allow for conclusions regarding a causal relationship [14,15,16].

4. Discussion

4.1. Principal findings

This review indicates that the associations between seborrheic dermatitis and metabolic disorders are not uniform and are not equally well documented. The available evidence is more consistent with regard to insulin resistance than to metabolic syndrome. Patients with SD described by Erdoğan et al. had higher fasting insulin levels, HOMA-IR values, and blood glucose levels at the two-hour mark of the OGTT, and the prevalence of insulin resistance was more than twice as high as in the control group [14]. Similar observations were made by Semiz et al., who reported higher HOMA-IR values in patients with SD [20]. Therefore, it can be concluded that the findings to date suggest a link between SD and insulin resistance, although the nature of this relationship remains not fully understood. The limited number of studies, small sample sizes, and their observational nature make it difficult to assess the direction of this relationship and do not allow for conclusions regarding causality. The relationship between SD and MetS remains less clear. Several studies have shown that metabolic syndrome was more common in patients with SD, although the difference was not always statistically significant. Imamoğlu et al. and Erdoğan et al. reported a higher prevalence of MetS in groups with SD than in control groups. However, in both studies, these differences were not statistically significant [14,16]. Akbaş et al. obtained different results, demonstrating a significantly higher prevalence of MetS in individuals with SD (35.2% vs. 10.6%) [15]. A similar association was found by Semiz et al., who identified metabolic syndrome in 14 of 49 patients with SD, whereas no cases were reported in the 40-person control group [20]. A meta-analysis of case-control studies supports this association. However, the conclusion regarding seborrheic dermatitis was based on a small number of studies [17]. An analysis of the individual

components of metabolic syndrome does not point to a single dominant disorder that could account for the observed association between SD and MetS. However, studies have relatively frequently reported abnormalities in the lipid profile, particularly reduced HDL-C levels and elevated triglyceride levels [14,15,16]. Another factor that may play a key role in this correlation is blood pressure. A large population-based study demonstrated an independent association between SD and hypertension after statistically adjusting for potential confounding factors such as age, sex, smoking, obesity, and diabetes [22]. At the same time, Özgül et al. found no significant differences in BMI, total body fat mass, or visceral fat mass, suggesting that greater body fat does not fully explain the observed association between SD and metabolic disorders [21]. Given the above findings, the association between seborrheic dermatitis and insulin resistance appears to be more consistent than that with metabolic syndrome as a whole. The observed association between seborrheic dermatitis and metabolic syndrome, on the other hand, may reflect the coexistence of several metabolic abnormalities, such as carbohydrate and lipid metabolism disorders and elevated blood pressure. The evidence currently available in the literature supports the hypothesis of a link between SD and metabolic disorders. However, it does not allow for establishing a causal relationship between them or for recognizing MetS as a confirmed comorbidity of SD.

4.2. Interpretation of the relationship between SD, IR, and MetS

There are several non-mutually exclusive models through which the relationships between SD, IR and MetS can be interpreted. The first of these posits that metabolic disorders create an environment that contributes to the development or exacerbation of SD. Changes in adipokine activity, lipid metabolism disorders and low-grade chronic inflammation associated with IR and MetS may potentially affect the skin [17,23]. From this perspective, the higher insulin levels and HOMA-IR values observed in patients with SD, along with accompanying lipid abnormalities, may suggest that metabolic dysregulation is not merely a coincidental comorbidity [14,20]. In a study by Semiz and Aktaş, patients with SD were also found to have lower adiponectin concentrations and higher leptin levels, whilst the association between adiponectin concentration and SD persisted after adjusting for BMI, MetS components and HOMA-IR [20]. A second possible explanation lies in shared pathophysiological mechanisms, without implying a direct causal link. SD is a chronic, inflammatory dermatosis. However, IR and MetS are also characterised by disturbances in the regulation of metabolic and immunological processes. This co-occurrence could be partly explained by their sharing of pathways associated with oxidative stress, chronic inflammation, insulin signalling and lipid metabolism [17,20,23]. A meta-analysis by Sodagar et al. indicates an epidemiological association between SD and MetS. However, the certainty of this estimate is significantly limited by the fact that only two studies were available for SD [17]. Another interpretation suggests that some of the observed associations may be due to the co-occurrence of confounding factors. In the studies analysed, the groups differed in terms of body weight, waist circumference, smoking status, family history and dietary parameters [14,20]. At the same time, not all results confirm higher levels of obesity in people with SD. Özgül et al. found no differences between patients and control subjects in terms of BMI, body fat mass and visceral fat [21]. Furthermore, the inconclusive findings regarding MetS - such as the absence of a significant difference in some studies and a markedly higher prevalence in others - do not support a simple, unidirectional model [14,15, 16]. It therefore seems reasonable to interpret that SD and metabolic dysfunction are interrelated, although this relationship is likely to be multifactorial. At present, the findings do not allow us to determine whether IR and MetS increase the risk of SD, or whether all these conditions are partly underpinned by common biological mechanisms and exposures. It is therefore not possible to establish a causal relationship in this association. This relationship requires confirmation in prospective studies.

4.3. Biological plausibility

The association between SD and insulin resistance and metabolic syndrome is supported by potential biological mechanisms. However, the data to date do not allow for the identification of a clear causal pathway that would directly link metabolic disorders to the development of SD. Insulin resistance and the associated compensatory hyperinsulinemia may be accompanied by chronic inflammation, lipid metabolism disorders, and increased oxidative stress, which may also affect the maintenance of normal skin homeostasis [13,35]. In this context, the role of oxidative stress remains a key issue, as patients with SD have been shown to exhibit abnormalities in oxidative-antioxidant balance parameters - including TAC, TOS, and OSI - as well as changes in thiol-disulfide homeostasis [20,27]. These findings may indicate the existence of common pathophysiological mechanisms. However, they do not allow for a definitive determination of whether

oxidative stress is a factor initiating the development of SD, a consequence of the disease, or a contributor to the severity of its symptoms. Another potential mechanism may be the effect of metabolic disorders on the function of the sebaceous glands. Insulin and IGF-1 may influence sebocyte activity and lipid metabolism, and experimental studies indicate that IGF-1 may enhance lipogenesis by activating the PI3K/Akt pathway and SREBP-1 [24,25]. In cell culture, it has been demonstrated that IGF-1 increased lipid synthesis and the expression of selected pro-inflammatory mediators, including IL-1 β , IL-6, IL-8, and TNF- α [26]. These phenomena may be relevant in the context of sebaceous gland function, as their activity and the composition of the sebum they produce are important components of the skin microenvironment in areas predisposed to SD [9]. To date, it has not been proven that hyperinsulinemia leads to the development of SD via this mechanism. The available data on the effect of the insulin/IGF-1 axis on sebocytes come largely from experimental studies and research on acne [24,26]. Potential links may also involve the skin barrier and the skin microbiome. Metabolic disorders may affect pathways related to the inflammatory response, the composition of epidermal lipids, and the functioning of the skin barrier, which in turn may influence the local environment for skin microorganisms [35]. In the case of SD, impairments in the function of the skin barrier, changes in lipid organization, the immune response, and *Malassezia* activity remain interrelated elements of pathogenesis [9]. Given this, one can consider a model in which insulin resistance and hyperinsulinemia, by influencing inflammatory processes, oxidative stress, and lipid metabolism, may modify the skin microenvironment in a way that potentially promotes the development of SD. It should be emphasized, however, that the presented model is a hypothesis based on biological premises and does not constitute a confirmed causal mechanism.

4.4. Heterogeneity and conflicting findings

The inconsistent results regarding the association between SD and IR and MetS may be due to methodological differences among the available studies. Most analyses focused on studies conducted on small groups, typically consisting of several dozen patients. This fact limits statistical power and partly explains why a higher prevalence of MetS was not always confirmed as statistically significant [14,16,21]. Furthermore, the available literature is dominated by case-control studies, which are observational in nature. While they allow for the demonstration of the co-occurrence of disorders, they do not enable the determination of their temporal sequence or the direction of the relationship [14,20]. The characteristics of the study populations also play a key role in the heterogeneity of the studies. The groups of participants differed in age, gender distribution, BMI, and waist circumference. In some studies, individuals with SD were found to have higher BMIs or larger waist circumferences than control groups [14,15,20], whereas Özgül et al. found no significant differences in BMI or body composition parameters, including total body fat mass and visceral fat mass [21]. These discrepancies pose a challenge in determining to what extent the association between SD and metabolic disorders is independent of age and obesity. The comparability of the studies is also limited due to differences in how the disorders under analysis were defined. The studies analyzed used different diagnostic criteria for MetS, and IR was assessed using various parameters and HOMA-IR cutoff values [14,15,16,20]. The method of assessing SD was also not consistent across studies, and the results regarding the association between disease severity and metabolic disorders were inconclusive. In one study, higher SDASI scores were associated with higher HOMA-IR values [14], whereas in another, no significant differences were found in HOMA-IR or the prevalence of MetS depending on the severity of SD [20]. Another problem is lifestyle factors and insufficient control for potential confounding variables. Individual studies varied in the extent to which they assessed factors such as smoking, diet, physical activity, stress, and medication use. Differences included, among other things, the approach to smoking: in one study, the control group consisted exclusively of nonsmokers [15], while in another, smokers were completely excluded from participation in the study [20]. Diet-related factors were also not systematically accounted for in all studies, which may further influence the observed associations [20]. Differences in the study populations, diagnostic criteria, and methods for assessing metabolic parameters, as well as inconsistent consideration of potential confounding factors, limit the direct comparability of the results. The methodological heterogeneity of the available studies therefore remains a significant limitation in interpreting the association between SD and IR and MetS.

4.5. Clinical implications

Despite discrepancies in the available data, the observed association between SD and IR and MetS may influence clinical practice, primarily by raising awareness of potential metabolic risks. The available data suggest that, amongst some patients with SD, abnormalities in glucose-insulin metabolism, lipid profile, blood pressure and waist circumference are more common [14,15,23]. However, this does not imply that a diagnosis of SD alone should be considered an indication for routine screening for IR or MetS. In clinical practice, it seems advisable to take basic metabolic risk factors into account as part of a comprehensive assessment of the patient. It is particularly important to consider excess weight and central obesity, given that waist circumference is a simple parameter for identifying an increased metabolic risk [36]. Where other risk factors are present - including obesity, hypertension, dyslipidemia, carbohydrate metabolism disorders or a positive family history - further assessment in accordance with general clinical management guidelines may be warranted, regardless of the presence of SD [16,36]. The course of the dermatosis may also be of significant importance. In some studies, greater severity of SD correlated with HOMA-IR or adverse lipid parameters [14,16]. However, these associations have not been unequivocally confirmed. Therefore, there is currently insufficient evidence to consider severe or recurrent SD as a separate criterion for referral for extended metabolic testing. More comprehensive reviews highlight the possibility of SD co-occurring with metabolic disorders, whilst at the same time pointing to a heterogeneous and limited evidence base [18,23]. At present, therefore, increased vigilance appears to be the most appropriate clinical approach, rather than routine metabolic screening of all patients with SD. Dermatologists may consider the presence of additional risk factors as grounds for a more comprehensive assessment of the patient's general health or for referring them to a general practitioner. However, the introduction of specific screening guidelines for SD requires confirmation of the observed associations in larger, prospective studies.

4.6. Limitations

When interpreting the results presented, a number of significant limitations of the current evidence base must be taken into account. Studies on the association between SD and IR and MetS are mainly observational in nature, and most of these studies were conducted on relatively small populations. Case-control and cross-sectional studies predominate; while these allow the co-occurrence of SD and metabolic disorders to be identified, they do not allow the temporal sequence of these conditions to be determined. There is also still a lack of longitudinal studies to assess whether IR or MetS occur prior to the development of SD, arise during the course of SD, or co-occur as independent disorders. A further complication is the significant methodological heterogeneity of the studies, including differences in population characteristics, diagnostic criteria for MetS and IR, and methods for assessing the severity of SD. Studies did not take equal account of confounding factors such as gender, BMI, central obesity, smoking, diet, lifestyle and treatment. These factors limit the possibility of directly comparing results and assessing whether the observed correlations are independent of other metabolic risk factors. Consequently, the current data do not allow conclusions to be drawn regarding a causal relationship. These limitations also apply to this review. A narrative review methodology was employed, and the search was restricted to the PubMed database. This may have resulted in some relevant publications available in other databases being overlooked. No formal assessment of the risk of systematic error or meta-analysis was carried out. Consequently, it was not possible to quantitatively estimate the strength of the correlations analysed or to formally assess heterogeneity. The conclusions of this study should therefore be interpreted as a critical synthesis of the current state of knowledge, which requires confirmation in larger, prospective and methodologically standardised studies.

4.7. Future research directions

Future research should, in particular, focus on defining more precisely the nature and direction of the relationship between SD, IR and MetS. Larger, multicentre prospective studies covering diverse populations are essential, as this would enhance the representativeness of the results and minimise the influence of factors specific to individual centres. Longitudinal studies will be crucial in determining the chronology of the observed disorders and in assessing whether the development of SD is preceded by metabolic dysfunction, occurs during its course, or is an independently co-occurring phenomenon. In order to ensure the comparability of future studies, it is also advisable to increase methodological standardisation. It is appropriate to apply uniform criteria for the diagnosis and assessment of the severity of SD, consistent definitions of MetS, and clearly defined methods for assessing IR, including HOMA-IR cut-off values. Analyses should consistently take into account potential confounding factors, such as: age, sex, BMI, smoking, diet, waist circumference,

physical activity and treatment. At the same time, mechanistic studies are needed to verify the proposed link between insulin signalling, chronic inflammation, oxidative stress, lipid metabolism, sebaceous gland activity and skin barrier function. At present, the biological plausibility of these mechanisms is greater than the level of direct evidence for them in SD. Intervention studies analysing whether improvements in metabolic parameters - including insulin sensitivity, body weight or lipid profile - have an impact on the recurrence rate or severity of SD would be particularly useful. In the long term, the reverse hypothesis should also be tested: whether SD can serve as a clinical marker of increased metabolic risk. Only on the basis of such data will it be possible to determine whether the observed correlations should be taken into account in future diagnostic strategies and clinical management.

5. Conclusions

Current data suggest a potential link between seborrheic dermatitis and metabolic disorders, particularly insulin resistance and metabolic syndrome. The most consistent findings relate to insulin resistance (IR) - patients with seborrheic dermatitis have been found to have higher fasting insulin concentrations, elevated HOMA-IR values and a higher prevalence of insulin resistance. The relationship between seborrheic dermatitis and metabolic syndrome is less clear-cut. Although some studies found that metabolic syndrome was more common in patients with seborrheic dermatitis, the results were inconsistent, and the recurring abnormalities mainly involved selected components of metabolic syndrome, including the lipid profile, waist circumference and blood pressure. However, interpreting these analyses requires caution. The available studies are primarily observational, usually involve small populations, and vary in terms of diagnostic criteria and the methods used to assess metabolic disorders. They also take potential confounding factors into account to varying degrees. The lack of longitudinal studies prevents the establishment of a temporal relationship. Consequently, the current data do not allow us to determine whether IR and MetS may be associated with the development or progression of SD, nor to confirm a causal link. The suggested mechanisms, which involve chronic inflammation, oxidative stress, impaired insulin signalling and lipid metabolism, provide a biologically plausible explanation for the observed associations. However, they require direct confirmation in SD. At present, the findings do not justify routine screening for IR or MetS in all patients with SD. However, they may raise awareness of potential metabolic risk, particularly where other risk factors are present. Multicentre, prospective and longitudinal studies are required, conducted using standardised diagnostic criteria and with appropriate control for confounding factors. These will enable the clinical significance of the observed correlations to be determined and an assessment to be made as to whether SD can be considered a marker of increased metabolic risk.

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REFERENCES

1. Turchin, I., Albrecht, L., Hanna, S., Kyritsis, D., Loo, W. J., Lynde, C. W., Prajapati, V. H., Purdy, K., Rochette, L., Wiseman, M., Wong, D., Yadav, G., Yeung, J., & Gooderham, M. (2025). Current understanding of seborrheic dermatitis: Presentation, diagnosis, and special populations. *Journal of cutaneous medicine and surgery*, 29(4_suppl), 16S–23S. <https://doi.org/10.1177/12034754251368828>
2. Turchin, I., Albrecht, L., Hanna, S., Kyritsis, D., Loo, W. J., Lynde, C. W., Prajapati, V. H., Purdy, K., Rochette, L., Wiseman, M., Wong, D., Yadav, G., Yeung, J., & Gooderham, M. (2025). Current understanding of seborrheic dermatitis: Epidemiology, burden of disease, and pathophysiology. *Journal of cutaneous medicine and surgery*, 29(4_suppl), 5S–15S. <https://doi.org/10.1177/12034754251368840>
3. Polaskey, M. T., Chang, C. H., Daftary, K., Fakhraie, S., Miller, C. H., & Chovatiya, R. (2024). The global prevalence of seborrheic dermatitis: A systematic review and meta-analysis. *JAMA dermatology*, 160(8), 846–855. <https://doi.org/10.1001/jamadermatol.2024.1987>
4. Chen, J., Li, C., Polaskey, M. T., Chang, C. H., & Chovatiya, R. (2026). Impact of seborrheic dermatitis on quality of life: A systematic review and meta-analysis. *Dermatology practical & conceptual*, 16(2), 6224. <https://doi.org/10.5826/dpc.1602a6224>
5. Navarro Triviño, F. J., Velasco Amador, J. P., & Rivera Ruiz, I. (2025). Seborrheic dermatitis revisited: Pathophysiology, diagnosis, and emerging therapies-a narrative review. *Biomedicines*, 13(10), 2458. <https://doi.org/10.3390/biomedicines13102458>
6. Adalsteinsson, J. A., Kaushik, S., Muzumdar, S., Guttman-Yassky, E., & Ungar, J. (2020). An update on the microbiology, immunology and genetics of seborrheic dermatitis. *Experimental dermatology*, 29(5), 481–489. <https://doi.org/10.1111/exd.14091>
7. Chang, C. H., & Chovatiya, R. (2024). More yeast, more problems?: Reevaluating the role of *Malassezia* in seborrheic dermatitis. *Archives of dermatological research*, 316(4), 100. <https://doi.org/10.1007/s00403-024-02830-7>
8. Piacentini, F., Camera, E., Di Nardo, A., & Dell'Anna, M. L. (2025). Seborrheic dermatitis: Exploring the complex interplay with *Malassezia*. *International journal of molecular sciences*, 26(6), 2650. <https://doi.org/10.3390/ijms26062650>
9. Khimuk, S., Andrusiewicz, A., Mijas, D., & Nowicka, D. (2026). Molecular mechanisms in seborrheic dermatitis-systematic review. *International journal of molecular sciences*, 27(6), 2799. <https://doi.org/10.3390/ijms27062799>
10. Zhao, X., An, X., Yang, C., Sun, W., Ji, H., & Lian, F. (2023). The crucial role and mechanism of insulin resistance in metabolic disease. *Frontiers in endocrinology*, 14, 1149239. <https://doi.org/10.3389/fendo.2023.1149239>
11. Neeland, I. J., Lim, S., Tchernof, A., Gastaldelli, A., Rangaswami, J., Ndumele, C. E., Powell-Wiley, T. M., & Després, J. P. (2024). Metabolic syndrome. *Nature reviews. Disease primers*, 10(1), 77. <https://doi.org/10.1038/s41572-024-00563-5>
12. Islam, M. S., Wei, P., Suzaudulla, M., Nime, I., Feroz, F., Acharjee, M., & Pan, F. (2024). The interplay of factors in metabolic syndrome: Understanding its roots and complexity. *Molecular medicine (Cambridge, Mass.)*, 30(1), 279. <https://doi.org/10.1186/s10020-024-01019-y>
13. Masenga, S. K., Kabwe, L. S., Chakulya, M., & Kirabo, A. (2023). Mechanisms of oxidative stress in metabolic syndrome. *International journal of molecular sciences*, 24(9), 7898. <https://doi.org/10.3390/ijms24097898>
14. Savaş Erdoğan, S., Falay Gür, T., Özkur, E., & Doğan, B. (2022). Insulin resistance and metabolic syndrome in patients with seborrheic dermatitis: A case-control study. *Metabolic syndrome and related disorders*, 20(1), 50–56. <https://doi.org/10.1089/met.2021.0063>
15. Akbaş, A., Kılınç, F., Şener, S., & Hayran, Y. (2022). Investigation of the relationship between seborrheic dermatitis and metabolic syndrome parameters. *Journal of cosmetic dermatology*, 21(11), 6079–6085. <https://doi.org/10.1111/jocd.15121>
16. Imamoglu, B., Hayta, S. B., Guner, R., Akyol, M., & Ozelcik, S. (2016). Metabolic syndrome may be an important comorbidity in patients with seborrheic dermatitis. *Archives of medical sciences. Atherosclerotic diseases*, 1(1), e158–e161. <https://doi.org/10.5114/amsad.2016.65075>
17. Sodagar, S., Ghane, Y., Heidari, A., Heidari, N., Khodadust, E., Ahmadi, S. A. Y., Seirafianpour, F., Baradaran, H., & Goodarzi, A. (2023). Association between metabolic syndrome and prevalent skin diseases: A systematic review and meta-analysis of case-control studies. *Health science reports*, 6(9), e1576. <https://doi.org/10.1002/hsr2.1576>
18. Kluglein, K. A., South, S. C., Toska, E., Nadolny, R., Yagoda, A., & Krusz, S. J. (2024). Exploring systemic comorbidities and lifestyle factors associated with seborrheic dermatitis: A scoping review. *Cureus*, 16(11), e73555. <https://doi.org/10.7759/cureus.73555>
19. Woolhiser, E., Keime, N., Patel, A., Weber, I., Adelman, M., & Dellavalle, R. P. (2024). Nutrition, obesity, and seborrheic dermatitis: Systematic review. *JMIR dermatology*, 7, e50143. <https://doi.org/10.2196/50143>
20. Semiz, Y., & Aktaş, E. (2025). Investigating the role of dietary glycemic factors and antioxidant capacity, metabolic status, and oxidative stress in seborrheic dermatitis: A case-control study. *Journal of the American Academy of Dermatology*, 92(3), 503–510. <https://doi.org/10.1016/j.jaad.2024.10.078>

21. Ozgul, A., Altunisik, N., Turkmen, D., & Sener, S. (2023). The relationship between seborrheic dermatitis and body composition parameters. *Northern clinics of Istanbul*, 10(2), 271–276. <https://doi.org/10.14744/nci.2022.08068>
22. Linder, D., Dreier, J., Zampetti, A., Sampogna, F., & Cohen, A. D. (2014). Seborrheic dermatitis and hypertension in adults: A cross-sectional study. *Journal of the European Academy of Dermatology and Venereology : JEADV*, 28(11), 1450–1455. <https://doi.org/10.1111/jdv.12310>
23. Tripathy, K., Dulai, A. S., Min, M., & Sivamani, R. K. (2026). Insulin resistance, metabolic syndrome, and inflammatory skin disease. *Journal of clinical medicine*, 15(1), 330. <https://doi.org/10.3390/jcm15010330>
24. Okoro, O. E., Camera, E., Flori, E., & Ottaviani, M. (2023). Insulin and the sebaceous gland function. *Frontiers in physiology*, 14, 1252972. <https://doi.org/10.3389/fphys.2023.1252972>
25. Smith, T. M., Gilliland, K., Clawson, G. A., & Thiboutot, D. (2008). IGF-1 induces SREBP-1 expression and lipogenesis in SEB-1 sebocytes via activation of the phosphoinositide 3-kinase/Akt pathway. *The Journal of investigative dermatology*, 128(5), 1286–1293. <https://doi.org/10.1038/sj.jid.5701155>
26. Kim, H., Moon, S. Y., Sohn, M. Y., & Lee, W. J. (2017). Insulin-like growth Factor-1 increases the expression of inflammatory biomarkers and sebum production in cultured sebocytes. *Annals of dermatology*, 29(1), 20–25. <https://doi.org/10.5021/ad.2017.29.1.20>
27. Emre, S., Kalkan, G., Erdoğan, S., Aktaş, A., & Ergin, M. (2020). Dynamic thiol/disulfide balance in patients with seborrheic dermatitis: A case-control study. *Saudi journal of medicine & medical sciences*, 8(1), 12–16. https://doi.org/10.4103/sjmms.sjmms_50_19
28. Emre, S., Metin, A., Demirseren, D. D., Akoglu, G., Oztekin, A., Neselioglu, S., & Erel, O. (2012). The association of oxidative stress and disease activity in seborrheic dermatitis. *Archives of dermatological research*, 304(9), 683–687. <https://doi.org/10.1007/s00403-012-1254-0>
29. Darras, L., Ipinazar, C., Gravier, E., Mercadie, A., Villaret, A., Marinescu, R., Maître, M., Génies, C., Mengeaud, V., Fournier, I., & Josse, G. (2026). Lipidomic insights into seborrheic dermatitis: Clinical evaluation of sebum changes using SpiderMass. *Dermatology and therapy*, 16(5), 2465–2481. <https://doi.org/10.1007/s13555-026-01719-9>
30. DeAngelis, Y. M., Gemmer, C. M., Kaczvinsky, J. R., Kenneally, D. C., Schwartz, J. R., & Dawson, T. L., Jr (2005). Three etiologic facets of dandruff and seborrheic dermatitis: Malassezia fungi, sebaceous lipids, and individual sensitivity. *The journal of investigative dermatology. Symposium proceedings*, 10(3), 295–297. <https://doi.org/10.1111/j.1087-0024.2005.10119.x>
31. Ro, B. I., & Dawson, T. L. (2005). The role of sebaceous gland activity and scalp microfloral metabolism in the etiology of seborrheic dermatitis and dandruff. *The journal of investigative dermatology. Symposium proceedings*, 10(3), 194–197. <https://doi.org/10.1111/j.1087-0024.2005.10104.x>
32. Rousel, J., Nădăban, A., Saghari, M., Pagan, L., Zhuparris, A., Theelen, B., Gambrah, T., van der Wall, H. E. C., Vreeken, R. J., Feiss, G. L., Niemeyer-van der Kolk, T., Burggraaf, J., van Doorn, M. B. A., Bouwstra, J. A., & Rissmann, R. (2024). Lesional skin of seborrheic dermatitis patients is characterized by skin barrier dysfunction and correlating alterations in the stratum corneum ceramide composition. *Experimental dermatology*, 33(1), e14952. <https://doi.org/10.1111/exd.14952>
33. Sanders, M. G. H., Nijsten, T., Verlouw, J., Kraaij, R., & Pardo, L. M. (2021). Composition of cutaneous bacterial microbiome in seborrheic dermatitis patients: A cross-sectional study. *PloS one*, 16(5), e0251136. <https://doi.org/10.1371/journal.pone.0251136>
34. Tao, R., Li, R., & Wang, R. (2021). Skin microbiome alterations in seborrheic dermatitis and dandruff: A systematic review. *Experimental dermatology*, 30(10), 1546–1553. <https://doi.org/10.1111/exd.14450>
35. Kreouzi, M., Theodorakis, N., Nikolaou, M., Feretzakis, G., Anastasiou, A., Kalodanis, K., & Sakagianni, A. (2025). Skin microbiota: Mediator of interactions between metabolic disorders and cutaneous health and disease. *Microorganisms*, 13(1), 161. <https://doi.org/10.3390/microorganisms13010161>
36. Alberti, K. G., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., Fruchart, J. C., James, W. P., Loria, C. M., Smith, S. C., Jr, International Diabetes Federation Task Force on Epidemiology and Prevention, National Heart, Lung, and Blood Institute, American Heart Association, World Heart Federation, International Atherosclerosis Society, & International Association for the Study of Obesity (2009). Harmonizing the metabolic syndrome: A joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; american heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. *Circulation*, 120(16), 1640–1645. <https://doi.org/10.1161/CIRCULATIONAHA.109.192644>