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Review

Evaluating dietary considerations in hidradenitis suppurativa: a critical examination of existing knowledge

Seçil Vural,[1] Defne Baskurt,[2] Şebnem Yıldırıcı,[2] Gunel Rasulova,[1] Senem Danacı,[3] and Aysenur Botsalı[4]

[1]Department of Dermatology and Venereology, Koç University School of Medicine, Istanbul, Turkey, [2]School of Medicine, Koç University, Istanbul, Turkey, [3]Department of Medical Pharmacology, Istanbul Cerrahpasa University, Istanbul, Turkey; and [4]Department of Dermatology and Venereology, University of Health Sciences, Gülhane Training and Research Hospital, Ankara, Turkey

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Correspondence
Seçil Vural
Department of Dermatology and Venereology
Koç University
School of Medicine
Davutpaşa Cad, No: 4, Topkapı
Istanbul 34010
Turkey
E-mail: sevural@ku.edu.tr

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Abstract

Hidradenitis suppurativa (HS) is a chronic condition that can overwhelm patients, and the effectiveness of supplementary dietary treatments remains uncertain. The primary aim of this review is to explore the connection between diet and HS progression. However, it is imperative to note that the evidence supporting a substantial role of the diet in HS remains weak. Dietary alterations alone should not be considered independent solutions for managing HS. Medical therapy continues to be indispensable for adequate treatment. Research indicates that the Mediterranean lifestyle and diet may provide cost-effective and beneficial adjustments when combined with traditional therapies. Conversely, foods with a high glycemic index and dairy could worsen HS symptoms, conceivably through mechanisms linked to insulin resistance and inflammation. Zinc, known for its antioxidant properties, shows promise as an adjunct therapy. Moreover, evidence suggests a connection between vitamin D deficiency and HS severity, although the findings are inconclusive. Brewer's yeast-free diet, B12 supplementation, intermittent fasting, and reducing the intake of refined sugar and dairy merit further investigation. In conclusion, this review highlights the need for additional research because of the lack of standardized reporting of clinical effects in the studies under scrutiny. A deeper exploration of the pathophysiology focusing on dietary modifications and their potential associations with HS severity is essential. Furthermore, it is crucial to recognize that patients' willingness to experiment with new diets makes them vulnerable to fraudulent interventions, highlighting the importance of evidence-based dietary guidance.

Introduction

Hidradenitis suppurativa (HS) is a chronic, devastating disease characterized by inflammatory nodules, sinus tract formation, and scarring, especially of the axillary, inguinal, and inframammary areas and buttocks. HS is a multifactorial disease, and over the last decade, substantial efforts have been made to identify the contribution of genetics and aberrant inflammatory response. Consequently, the cytokine milieu has been identified for targeted treatment approaches.[1–3] On the other hand, lifestyle is considered a vital confounder, and HS has well-documented associations with smoking, obesity, diabetes mellitus, and metabolic syndrome.[4–7]

In dermatological practice, physicians commonly encounter patients seeking answers to what alleviates or aggravates their disease. While dietary modifications may benefit symptom

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control, they should not be considered the only treatment option. Adequate medical treatment is needed to manage HS, and dietary interventions should be employed as adjuncts to medical therapy rather than as a replacement. Nevertheless, the investigation into the influence of diet on HS is confronted with limitations, primarily because of the inherent complexity of designing well-structured randomized dietary studies of sufficient duration. There are numerous challenges in designing these controlled trials, the most important ones being poor patient adherence to the diet list in the long term and the difficulty of excluding other potential contributing factors that may influence the disease. Moreover, the absence of a standardized diet duration and the heterogeneous designs of the present studies also hinder the analysis of the available data. Here, we summarize the current literature investigating the relationship between dietary habits and HS.

Materials and methods

The review was conducted according to PRISMA guidelines, with inclusion criteria focusing on articles written in English containing original results with objective or subjective measurement of HS course and severity. To identify eligible records from 1943 until September 2023, we conducted a systematic literature search in PubMed, Scopus, Google Scholar, and Medline without any restrictions on publication time, patient age, gender, or ethnicity, using the keywords "hidradenitis suppurativa AND diet, vitamin D, alcohol, Saccharomyces cerevisiae, brewer's yeast, dairy, smoking, zinc, myoinositol, riboflavin OR niacinamide." EndNote 20 was utilized to collect references and remove duplicates. Exclusion criteria included articles that were not reported in English or did not report original results related to diet and HS association. The selection of studies involved a two-phase process. DB and SY performed the literature review and screened the articles based on their titles and abstracts to select those that met the inclusion criteria. After this initial screening, full texts of potentially eligible articles were obtained and assessed for eligibility independently by DB and SY. In cases of disagreement, a consensus was reached after discussion with a third reviewer (SD). Data were extracted from each study, including study design, sample size, patient characteristics, dietary assessments, and outcomes.

Results

The review process was initiated by a literature search, which retrieved a total of 8,259 articles. After removing duplicates, 735 articles were assessed for eligibility, and 46 were ultimately included in the study.[7–52] A flowchart depicting the selection process can be found in Figure 1.

The included studies varied in study design, patient population, and sample size. Tables 1–5 and Tables S1–S4 comprehensively summarize the studies, including the number of patients, study design, results, and critical observations categorized by diet types. Reports found during the literature search mainly consist of case series, case reports, cross-sectional studies, and cohorts rather than randomized controlled trials. Therefore, this review was conducted as a comprehensive rather than a systematic review, with a detailed overview of included studies. The studies included 5,555 patients, with the most significant sample consisting of 1,776 patients.

Patient perceptions on the influence of diet on HS

Given the considerable challenges in managing HS, patients are deeply invested in exploring dietary modifications, which has led them to focus on identifying specific foods as triggers. Patients have been experimenting with various dietary and nutritional modifications to alleviate HS symptoms, reflecting their search for a personalized solution based on individual experiences. Unfortunately, the evidence supporting the relationship between diet and HS is not yet strong.

The patients' perception of diet is examined in several questionnaire studies to provide clinicians with insights into patient perspectives on dietary influences. Three survey studies and one questionnaire on patient perceptions were identified, summarized in Table 1. It is important to note that these questionnaire studies did not specify concurrent medical or surgical treatments for HS patients. Fernandez et al.[9] found that 32.6% of 777 participants identified specific exacerbating foods, including sweets, bread and pasta, dairy, and high-fat foods. Similarly, a study with 242 HS patients showed that 75.8% had made dietary alterations, primarily excluding gluten, dairy, refined sugars, tomatoes, and alcohol, with 30.9% reporting significant symptom improvement.[10]

A user-friendly StuffThatWorks® questionnaire, designed by physicians, allows patients to share their experiences with chronic conditions such as HS, enabling data analysis of treatment effectiveness. Interestingly, among Hurley I patients, dietary changes and turmeric (curcumin) were rated as more effective than tetracyclines for symptom management. Although turmeric's specific impact on HS patients has not been studied, it is known for its anti-inflammatory properties and has demonstrated safety in six human trials.[53,54]

According to the survey, various beneficial dietary changes were reported, including a ketogenic diet, autoimmune protocol diet, sugar reduction or elimination, dairy reduction or elimination, carbohydrate reduction or elimination, nightshade reduction or elimination, gluten-free diet, chocolate avoidance, red meat reduction or elimination, unspecified dietary changes, weight loss and smoking cessation were cited as beneficial for symptom relief. Dairy emerged as the most likely food item to exacerbate HS symptoms.

A cross-sectional survey study by Macklis et al.[11] reported that the most significant improvements occurred following the paleo and anti-inflammatory diet categories, with the detox and

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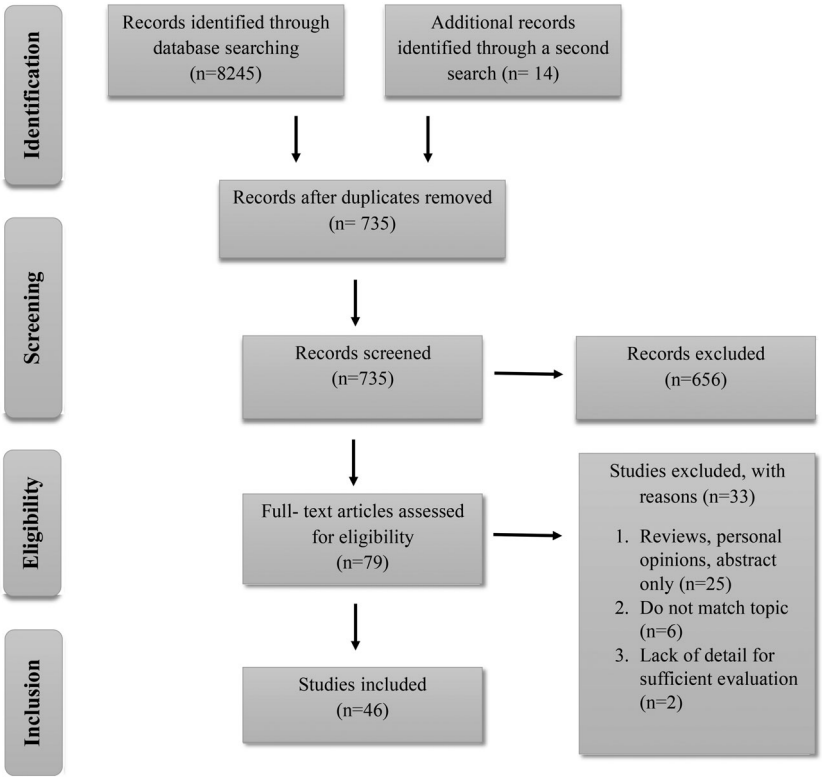

Figure 1 Article selection flowchart according to PRISMA guidelines

Mediterranean diets also showing positive effects. It is essential to acknowledge, however, that this study presents several notable limitations, including the utilization of the Hurley staging system to gauge improvements, as this system offers a static evaluation of the extent of the disease, the absence of examinations by dermatologists, as well as the lack of information regarding concomitant therapeutic interventions.

The Western diet, characterized by high-glycemic index foods, red meat, dairy, and egg protein, has been associated with acne vulgaris, suggesting a potential negative impact on HS.[55] Patient experiences support this link, with dairy as the most reported exacerbating factor, followed by high-glycemic index foods, and red meat consumption. It is essential to maintain a critical perspective, acknowledging that the findings predominantly stem from personal experiences, rendering them inherently subjective. Still, this patient-centered approach holds promise for guiding future HS research.

Dietary factors exacerbating hidradenitis suppurativa

Dairy, high glycemic load, and hyperinsulinemia cycle

The extensive research connecting diet to acne vulgaris sheds light on the mechanisms underpinning HS. Both conditions target the terminal hair follicle.[56,57] High-glycemic index foods, including junk food, beverages, pizza, potatoes, and white bread, lead to rapid blood glucose spikes. Dairy and simple carbohydrates in the Western diet elevate insulin and insulin-like growth factor-1 (IGF-1), downregulating forkhead box protein O1 (FOXO1). FOXO1 suppression results in heightened androgen receptor activity, increased sebaceous function, and keratinocyte proliferation in acne while also activating the mammalian kinase target of rapamycin complex 1 (mTORC1) signaling, promoting cellular hyperproliferation in the folliculosebaceous unit. This shared pathway contributes to HS and acne vulgaris pathology, resulting in occlusion, inflammation, and scarring in high-friction areas like the axilla, groin, and inframammary regions.[55,58,59]

Median Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) levels are significantly higher in HS patients compared to controls, supporting the role of the vicious cycle of glucose-hyperinsulinemia in HS.[20] The role of high-glycemic index foods is further supported by the recognition of anti-carboxy-ethyl-lysine (anti-CEL) antibodies in HS patients' sera.[60] These antibodies are linked with advanced glycation end products (AGEs), produced when high glucose levels react with proteins or lipids in a non-enzymatic manner.[60,61] AGEs bind to their specific receptors and stimulate the production of proinflammatory cytokines such as interleukin-1 beta (IL-1β) and tumor necrosis factor-alpha (TNF-α), promoting follicular hyperkeratosis, inflammation, and fibrosis. AGEs play a significant

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Table 1 Studies evaluating patient perceptions of dietary modifications on disease activity

Article	Study type	P (*n*)	C (*n*)	Type of diet/lifestyle	ToA	Score	Sys MTx	Results & comments
Fernandez et al. (2020)[9]	Report of cross-sectional web-based survey	744	No	Sweets, dairy, bread/pasta/rice, vegetables, fruit, chicken, fish	BL	NR	NR	• Sweets, bread/pasta/rice, dairy identified as triggers by 67.9, 51.1, 50.6, and 42.2% respectively. • Vegetables, fruits, chicken, fish identified as alleviating foods. • HS patients may benefit from receiving dietary counseling.
Dempsey et al. (2020)[10]	Cross-sectional survey	242	No	Elimination of gluten, dairy, refined sugars, tomatoes, alcohol	BL	Self- reported survey	NR	• 182 patients tried elimination of at least one food item. • 154 patients tried elimination of more than one food item. • 30.9% reported elimination of foods had improved HS
Macklis et al. (2022)[11]	Cross-sectional survey	591	No	Diets (Paleo, keto, gluten-free, anti-inflammatory, MD, detox) Personal products	>2 months	Self-reported questionnaire	NR	• Various diet categories and changing harsh soaps and detergents were associated with +1–2 subjective point improvement. • Most significant improvement with paleo and anti-inflammatory diets. • Limited by lack of information on concurrent systemic therapies and self-evaluations rather than dermatologists

role in the pathogenesis of chronic diseases, including diabetes mellitus.[62]

In a recent animal study, a high-fat diet (HFD) induced follicular hyperkeratosis, a distinct histological feature linked to neutrophilic folliculitis.[13] The authors proposed that HFD may contribute to neutrophilic folliculitis by increasing neutrophil infiltration around hair follicles via CXC chemokine receptor 2 (CXCR2) signaling. Moreover, HFD-induced pathological changes in the gut microbiome have been linked to heightened inflammation, matrix remodeling, and skin tract formation in HS patients.[63]

Various dietary modifications, like dairy elimination, low-glycemic load diets, and weight loss, have been explored as adjuvant HS prevention or treatment strategies. These studies are summarized in Table 2. However, existing data do not offer clear evidence of their benefits.[64] While Danby et al. reported clinical improvement in 83% of 47 HS patients on a dairy-free diet, these findings were based on personal communication rather than an objective clinical assessment.[14]

In a retrospective study of 40 HS patients using a questionnaire-based approach, 35% reported digestive issues and experimented with different diets.[12] Two patients were diagnosed with gluten-sensitive enteropathy, noting HS improvement on a gluten-free diet. Seven patients experienced symptom relief with a low-dairy/low-carbohydrate diet, excluding milk, lactose, sugar, nightshades, wheatmeal products, and grains.[12] The study suggested incorporating gluten-sensitive enteropathy assessment into routine HS patient examinations.

Saccharomyces cerevisiae and yeast-containing food

Brewer's yeast, also known as *Saccharomyces cerevisiae*, is a single-cell fungus involved in alcohol fermentation and commonly found in baked goods (e.g., pizza, cookies, bread, cakes), vinegar, black tea, soy sauces, fermented cheese (e.g., gorgonzola, bleu d'Auvergne), mushrooms, wine, and beer. Its potential role as a contributing factor in HS pathogenesis has been debated.

Three studies investigating the relationship between brewer's yeast and HS were identified: one questionnaire-based cohort, one clinical trial, and one prospective case-control study[31–33] (Table 3). All studies reported improved HS lesions, symptoms, and fewer flare-ups following a yeast-free diet for 3–12 months. Aboud et al. and Cannistra et al. reported frequent inflammatory lesion flares within 24–48 hours after accidental yeast consumption (87 and 100%, respectively).[31,33]

Immunization with *Saccharomyces cerevisiae* can induce anti-*Saccharomyces cerevisiae* antibodies (ASCAs).[33] Cannistra et al.[31] found ASCA IgG positivity in 100% of their study population, suggesting a potential link between HS and ASCA IgG, with yeast-free diets as a potential intervention.

In a recent study of syndromic forms of HS, 87.5% of patients tested positive for ASCA IgG antibodies.[65] Five patients with asthenia, anemia, and elevated serum C-reactive protein (CRP) levels underwent gastrointestinal (GI) endoscopy; three showed pathology results suggesting various stages of GI tract

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Table 2 Studies evaluating the effects of dairy, high glycemic load, and hyperinsulinemia cycle on disease activity

Article	Study type	P (*n*)	C (*n*)	Type of diet/lifestyle	ToA	Score	Sys MTx	Results & comments
Sutton and Marks (1943)[8]	CR	1	No	Low-fat diet and 0.065 g thyroxine hormone twice daily	2 months	NR	NR	• Symptoms resolved completely. • Relapse occurred when thyroxine doses were missed. • Low-fat diet can be beneficial
Kurzen et al. (2019)[12]	Questionnaire-based retrospective study	40	No	Dairy- and carbohydrate-free diet	NR	NR	NR	• Low-dairy and low-carbohydrate diet resulted in improvement in seven patients. • In two patients with celiac disease HS improved with gluten-free diet. • Incorporating gluten-sensitive enteropathy assessment in routine HS examination may be beneficial
Nakamizo et al. (2021)[13]	Animal study	NR[a]	NR	High-fat diet	NR	NR	NR	• Dietary FA promotes neutrophilic folliculitis and hyperkeratosis of follicular epidermis.
Danby et al. (2015)[14]	Report of self-observation in a review	47	No	Dairy-free diet	NR	NR	NR	• Improvement in 83% with none worsening.

[a]Study conducted with mice.

Table 3 Studies evaluating the effects of *Saccharomyces cerevisiae* and yeast-containing food on disease activity

Article	Study type	P (*n*)	C (*n*)	Type of diet/ lifestyle	ToA	Score	Sys MTx	Results & comments
Cannistra et al. (2013)[31]	Clinical trial	12	No	Brewer's yeast- free diet	12 –24 months	NR	NR	• Immediate stabilization of HS lesions and improved QoL • Relationship between HS and brewer's yeast need to be analyzed in multicentric studies
Colboc et al. (2016)[32]	Prospective multicenter study	20	No	Brewer's yeast- free diet	3 months	Scale 0–10 (self & dermatologist)	80%	• Improvement in pain ($P = 0.006$), inflammation ($P = 0.018$) discharge intensity ($P = 0.005$) • Yeast-free diet can be adjunct to therapy. • Larger sample size studies are needed
Aboud et al. (2020)[33]	Questionnaire-based cohort study	37	No	Brewer's yeast- free diet	6 months and 6 years	Self- reported survey	NR	• 70% described improvement in HS. 81% report improvement in <6 months. • Yeast-free diet leads to lesion stabilization and possibly a decrease in the need for more invasive surgeries

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Table 4 Studies evaluating the effects of alcohol consumption on disease activity

Article	Study type	P (*n*)	C (*n*)	Type of diet/ lifestyle	ToA	Score	Sys MTx	Results & comments
Shlyankevich et al. (2014)[28]	Retrospective case-control	1,776	1,730	Alcohol	NR	NR	NR	• Substantially smaller OR with alcohol dependence was observed after controlling for psychiatric comorbidities. • Instead of a cause, alcohol use seemed an outcome of psychiatric disease burden
Walter et al. (2018)[29]	Retrospective cohort study	48	No	Alcohol	12 months	NR	NR	• Increased alcohol consumption had significant effect on HS relapse after surgery ($P = 0.027$)
Peterson et al. (2020)[30]	Retrospective cohort study	149	No	Alcohol	NR	NR	100%	• The alcohol consumption rate was 56.8%. • No statistically significant relationship observed between alcohol and HS.
Shlyankevich et al. (2014)[28]	Retrospective case-control study	1,776	1,730	Alcohol	NR	NR	NR	• Substantially smaller OR with alcohol dependence was observed after controlling for psychiatric comorbidities. • Instead of a cause, alcohol use seemed an outcome of psychiatric disease burden
Walter et al. (2018)[29]	Retrospective cohort study	48	No	Alcohol	12 months	NR	NR	• Increased alcohol consumption had significant effect on HS relapse after surgery ($P = 0.027$)
Peterson et al. (2020)[30]	Retrospective cohort study	149	No	Alcohol	NR	NR	100%	• Alcohol consumption rate was 56.8%. • No statistically significant relationship observed between alcohol and HS.

inflammation, suggestive of subclinical inflammatory bowel disease (IBD).[31,65] The presence of ASCA IgG and GI inflammation are considered indicators of subclinical digestive tract inflammation. Notably, the study observed sterile pus in HS lesions, suggesting a potential link between dietary intolerance and inflammatory responses.[33] These findings hint at the possible involvement of gut dysbiosis and mycobiome in HS pathogenesis. The response to dietary yeast restriction and the presence of ASCA IgG in a notable number of HS patients suggest a potential link to this hypothesis.[33] However, further research is needed to establish a significant association and confirm the efficacy of yeast-free diets.

Alcohol consumption

Saccharomyces cerevisiae plays a vital role in alcoholic beverage production by fermenting glucose to ethanol. The prevalence of ASCA IgG antibodies in patients experiencing flares after alcohol intake has not been investigated. Epidemiological studies have noted a high rate of alcohol consumption among HS patients, but the association between alcohol and HS etiology remains controversial. Three studies investigating the effects of alcohol on HS were identified, one retrospective case-control and two retrospective cohort studies,[28–30] with mixed results (Table 4). One study found a higher rate of local HS recurrences following wide-excision surgery in patients who consumed alcohol, though the significance was borderline.[29]

Another study with 149 HS patients reported a 56.8% alcohol consumption rate.[30] An online survey of 242 HS patients showed that 88 participants reduced their alcohol intake, with 48 avoiding it altogether, yet no significant improvement was observed.[10] A study by Shlyankevich et al.[28] demonstrated that although alcohol dependence exhibited a higher odds ratio (OR) in univariate analysis, after stratifying for other comorbidities, alcohol intake might, in fact, be a consequence of the psychiatric disease burden associated with HS rather than a cause.

According to a population-based study by Garg et al.,[66] HS patients are more prone to anxiety disorders, major depressive disorder, and substance use disorders (SUD). Substance abuse is often used as a coping mechanism for the physical and psychological burden of the disease. In this study investigating alcohol, cannabis, and opioid use, 4% of the 32,625 HS patients have SUD, a statistically significant ratio compared to the 2% SUD ratio observed in the non-HS population. Nearly half of the SUD cases were alcohol abuse. Thus, the authors suggested that pain and the impact of HS on quality of life might have a more significant effect on SUD than co-existent psychiatric disorders. Overall, the current evidence regarding the association between alcohol consumption and HS is conflicting.

Beneficial diets and strategies

Mediterranean diet

The Mediterranean diet (MD) is globally recognized as one of the healthiest eating patterns, renowned for its positive impact on overall well-being and cardiovascular health. While variations exist across countries, MD generally consists of a diet abundant

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Table 5 Studies evaluating effects of Mediterranean diet and intermittent fasting on disease activity

Article	Study type	P (*n*)	C (*n*)	Type of diet/lifestyle	ToA	Score	Sys MTx	Results & comments
Barrea et al. (2018)[15]	Cross-sectional study	41	41	MD	NR	Sartorius score HS-PGA	No	• Controls consumed more fish and less red meat than HS patients. • HS patients had worse body composition and lower adherence to MD. • Commitment to MD may decrease disease severity.
Lorite-Fuentes et al. (2022)[16]	Cross-sectional study	221	No	MD Exercise	NR	Hurley stage, IHS4, NRS	>50%	• MD adherence negatively correlated with IHS4 ($P < 0.001$). • Exercise is related to higher MD adherence. • Virgin olive oil was the main dietary element associated with lower HS activity ($P < 0.05$).
Velluzzi et al. (2021)[17]	Case-control study	35	35	MD	NR	Sartorius score, MDS	NR	• Low adherence to MD in HS patients was reported compared to controls.
Damiani et al. (2019)[27]	Observational crossover pilot study	55	No	Intermittent fasting	1 month	IHS4	85%	• Improvement in draining fistulas and abscesses in 70.9% • Future studies are necessary to confirm findings since most patients also had systemic therapies.

BL, baseline; C, control group; CDLQI, Children's Dermatology Life Quality Index; CR, case report; CS, case series; DLQI, Dermatology Life Quality Index; FA, fatty acids; Hi-SCR, Hidradenitis Suppurativa Clinical Response; HOMA-IR, Homeostasis model assessment-estimated insulin resistance; HS, hidradenitis suppurativa; HS-PGA, HS physician global assessment; ID, iron deficiency; IHS4, International Hidradenitis Suppurativa Severity Score System; MD, Mediterranean diet; MDS, med diet score; mHSS, modified HS score; NR, not reported; NRS, numeric rating system; OR, odds ratio; PS, patient group; PS, pilot study; QoL, quality of life; ToA, timepoint of assessment; VAS, Visual Analog Scale.

in vegetables, fruits, legumes, nuts, beans, cereals, grains, fish, and healthy fats, primarily olive oil, with limited consumption of meat and dairy products. Three studies looking specifically into the HS and MD relationship were identified, two cross-sectional studies and one case-control study,[15–17] summarized in Table 5. There is agreement among all studies that HS patients exhibited lower adherence to MD, indicated by lower Mediterranean diet scores (MDS)[17] and a negative correlation of MD adherence with the International Hidradenitis Suppurativa Severity Score System (IHS4).[16]

One study implies that adherence to MD could potentially be a supportive factor in managing HS and reducing its clinical severity.[15] Embracing MD might help enhance body composition and potentially alleviate HS symptoms.

Additionally, the use of extra virgin olive oil emerged as the most effective component of MD ($P < 0.05$). However, it is important to note that over half of the patients in this study were concurrently undergoing systemic medical therapy with antibiotics and/or biologics.[16]

Recommending MD for HS management could be a promising and cost-effective adjunct to medical management but further prospective studies are necessary to explore the potential benefits of MD on HS management in larger and more diverse patient populations.

Intermittent fasting

One study aimed to investigate the impact of intermittent fasting on 55 HS patients during fasting in Ramadan of 2018[27] (Table 5). Notably, these patients did not report any significant changes in their weight over the fasting period. However, a substantial decrease from 11.00 ± 5.88 to 10.15 ± 6.45 ($P < 0.0001$) was observed in IHS4, indicating an improvement in the severity of HS.[27] While these findings suggest a potentially beneficial impact of intermittent fasting, it is crucial to recognize that these patients were already undergoing various systemic therapies, including a range of antibiotics and biologics.

Weight loss, bariatric surgery, and adjunctive drugs for weight loss and insulin resistance

Obesity is a recognized risk factor for HS inflammatory lesions, likely because of chronic low-grade systemic inflammation. Obesity can also trigger follicular hyperkeratosis, causing occlusion that is exacerbated by skinfold humidity, leading to bacterial overgrowth and infections. Additionally, obesity and insulin

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resistance complicate endocrine changes and HS skin manifestations.[25] Elevated insulin levels may promote androgen production, increasing sebaceous activity and keratinocyte proliferation.[52] Although weight loss is expected to benefit HS, outcomes following bariatric surgery (BS) remain controversial, likely because of HS's multifactorial nature. Seven studies on weight loss's effects on HS were found (Table S1). The designs were three case series, two cohorts, and one cross-sectional study.[7,21–26] Studies agree on significant improvement in HS severity and a higher likelihood of remission in patients with substantial weight loss. However, reports are mixed regarding the effects of BS on HS. In particular, in a case series by Garcovich et al.,[26] significant weight loss, exceeding 20% of total body weight with more than 50% expected loss, was observed in post-BS patients with 10 patients who had biliopancreatic diversion procedure developing HS post-surgery. One HS patient experienced worsened symptoms as a result of malabsorption from BS. Excess skin, a common result of post-BS weight loss, could increase skin friction and worsen HS.[7,26] Moreover, rapid post-BS weight loss might alter dermal extracellular matrix composition, causing mild inflammation, collagen resorption, and elastic fiber degeneration, potentially affecting HS progression.[67]

Oral antidiabetic drugs have also been employed as adjunctive therapies as a result of their association with significant weight loss and anti-inflammatory effects (S1). One case report and one case series have reported the benefits of metformin in improving HS severity by reducing insulin resistance.[18,19] Recently, GLP-1 analogs, liraglutide and semaglutide, have emerged as therapeutic choices. Two case reports and one case series with 14 patients have reported the use of liraglutide in HS patients.[50–52] In these studies, patients showed significant weight loss and improvement in Hurley and Dermatology Life Quality Index (DLQI) scores. Notably, most patients mentioned in these studies (12/16) were on concomitant systemic therapies. There were no reports of HS patients on semaglutide. Long-term experience with these agents in HS patients is limited, and further studies, as well as a multidisciplinary approach to management, are required to determine whether these agents can be used as therapeutic modalities in HS patients.

Zinc

Zinc is an essential micronutrient in innate and adaptive immune system regulation, besides its crucial role in metabolic and hormonal pathways. It acts as an inhibitor of neutrophil chemotaxis, phagocytosis of granulocytes, and activation of natural killer cells. Zinc regulates the production of pro-inflammatory cytokines such as TNF-α and interleukin-6. It also modulates the antioxidant enzyme Zn-Cu superoxide dismutase and the androgenic enzyme 5-alpha reductase type I. Given its crucial role in immune regulation and wound healing, zinc has been suggested to play a role in the pathogenesis of various inflammatory skin diseases, such as inflammatory acne, folliculitis decalvans, and HS caused by immune dysregulation.[68,69]

Six studies on the relationship between zinc and HS were identified, including one pilot study, one retrospective cohort, one case-control, one case report, one case series, and one clinical trial[40–45] (Table S2). Studies have shown that serum zinc levels are lower in patients with HS compared to the healthy population, and as such, zinc supplementation has been considered an adjunctive therapy.[42] The study by Garcovich et al.[70] revealed that zinc deficiency was more prevalent among HS patients who had undergone BS before the onset of HS, suggesting zinc deficiency may be a trigger for facilitating disease onset. While the benefits of zinc supplementation for HS have been debated, research has indicated that zinc's in vitro anti-inflammatory effects may play an important role in symptom improvement. Moreover, a 3-month duration of zinc supplementation boosted the expression of innate immunity markers.[71] The studies also show that daily 90 mg zinc supplementation may help reduce symptom severity and induce remission in Hurley grade I-II HS patients.[40,41]

Zinc supplementation has also been explored in the pediatric population. A case series by Offidani et al.[43] included eight prepubertal HS patients and demonstrated that short-term oral azithromycin in conjunction with routine oral zinc and topical clindamycin proved to be an effective and safe approach.

Additionally, combining zinc gluconate and nicotinamide has been proposed as a beneficial maintenance strategy for mild-to-moderate HS. In a controlled trial involving 92 HS patients who had previously received systemic tetracycline treatment and achieved clinical and ultrasonographic benefits, 47 patients received a standard regimen of 90 mg of zinc gluconate and 30 mg of nicotinamide once daily for 90 days. The results, after 12–24 weeks, showed a significant reduction in the number and duration of acute flares and pain measured on the Visual Analog Scale, DLQI, and IHS4 scores in the treatment group compared to the control group. Only two patients reported mild nausea in the treatment group, with none discontinuing the treatment.[44]

Vitamin D

Vitamin D, a crucial nutrient implicated in HS pathogenesis, significantly regulates the innate immune system and keratinocyte function. It maintains skin homeostasis by controlling epidermal and hair follicle proliferation and differentiation. Vitamin D also governs the cutaneous immune response by enhancing toll-like receptor expression and antimicrobial peptide production during infections.[72]

Six studies looking at the effects of vitamin D deficiency or supplementation on HS severity were identified, including one pilot study, two case controls, two cross-sectional studies, and one retrospective cohort[34–39] (Table S3). Multiple studies have consistently reported lower vitamin D levels in HS patients

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compared to healthy age- and gender-matched controls.[34–38] There is agreement among most studies that there is a correlation between vitamin D deficiency and HS severity, measured by various scoring systems such as Hurley stage and IHS4 scores.[35–38] In addition, Guillet et al.[35] and Fabroccini et al.[36] demonstrated that 3–6 months of vitamin D supplementation can improve disease outcomes in HS patients. However, Seetan et al.[39] found no significant association between HS severity and vitamin D levels in their cross-sectional study.

Brandao et al.[69] investigated vitamin D levels in syndromic HS patients such as PASH and PAPASH and found a mean vitamin D level of 10 ng/ml. They also identified gene mutations in vitamin D metabolism in five syndromic HS patients. These mutations caused diminished or impaired protein function in the Notch signaling pathway, leading to lower vitamin D levels.

In summary, several studies have reported an association between low vitamin D levels and HS severity. While some studies have not registered a significant association, based on these findings, the evaluation of serum vitamin D levels in the initial assessment of HS can be recommended.

Other micronutrients

The investigation into the efficacy of micronutrient supplementation in managing HS represents an area of research that warrants additional scrutiny. Four studies have been identified, two case series and two case controls[46–49] (Table S4). A case series involving 10 patients with HS who received a daily regimen of myoinositol (MI) at a dosage of 2,000 mg, liposomal magnesium, and folic acid for 6 months demonstrated a reduction in HS disease activity scores, and improvement in insulin resistance, measured by Sartorius Score and HOMA-IR, respectively.[48] Similarly, a case series involving six female patients with HS reported symptom clearance following a daily supplementation of riboflavin 9mg.[46]

HS patients exhibit a notable OR of 1.59 for developing anemia, as detailed in a 2023 meta-analysis.[73] These reports highlight anemia subtypes linked to HS: microcytic anemia because of iron deficiency and anemia from chronic inflammatory diseases. Narla et al.[74] also found a notably elevated OR of 1.87 for pernicious anemia caused by B12 deficiency in this context. Vitamin B12 supplementation exhibited promise in managing HS, as reported in a case series involving three patients who concurrently suffered from IBD

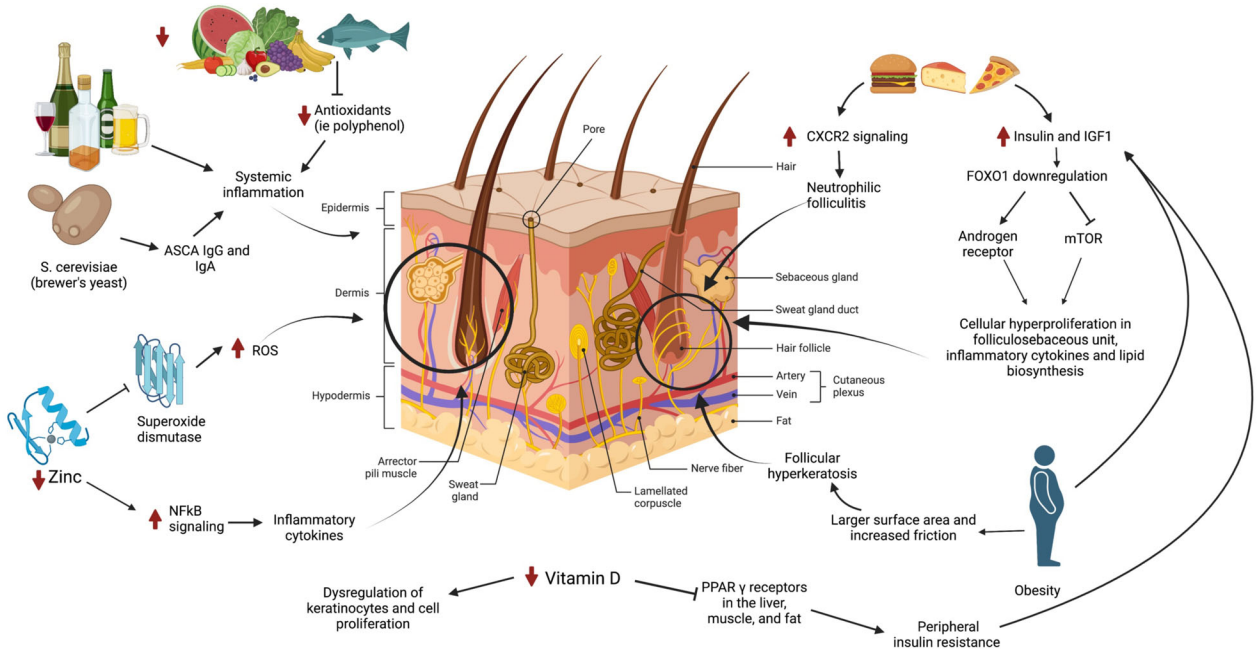

Figure 2 Theories on the contribution of dietary elements to cellular hyperproliferation and inflammation in Hidradenitis Suppurativa (HS). This illustrates the various factors that contribute to the development of cellular hyperproliferation and inflammation in HS. The diagram shows the interplay between diet, hormonal imbalances, obesity, vitamin D levels, zinc levels, and alcohol in promoting the pathogenesis of HS. Dairy and high-fat diets, characterized by increased insulin and IGF1 levels, lead to the downregulation of FOXO1 receptor. This suppresses the mTOR pathway and stimulates androgen receptors, resulting in cellular hyperproliferation and inflammatory cytokine secretion.[55,58,59] Obesity leads to insulin resistance and increases areas of friction, both of which contribute to HS lesion activation.[25] Diets low in fruits, vegetables, and fish are associated with low antioxidant intake, notably polyphenols, essential for mitigating oxidative stress and inflammation.[9] Alcohol consumption and ASCA antibodies are also linked to systemic inflammation.[35] Low vitamin D levels suppress PPAR gamma receptor activation in the liver, muscle, and fat, leading to insulin resistance. Vitamin D also plays a crucial role in regulating keratinocyte and cell proliferation.[75] Similarly, low zinc levels increase the production of ROS and inflammatory cytokines via NF-kB signaling, emphasizing the role of zinc as an antioxidant.[69] Created with BioRender.com

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and HS. This intervention led to complete resolution in two patients and partial resolution in one patient. It is noteworthy that none of these patients displayed vitamin B12 deficiency at the outset.[47] Furthermore, a separate study identified signs indicative of iron deficiency in HS patients, characterized by diminished ferritin levels, transferrin saturation, and hepcidin levels.[49]

In summary, the impact of micronutrient supplementation on HS management remains an area of limited comprehension. Future research should focus on elucidating the optimal dosages, durations, and potential adverse effects associated with these micronutrient supplements.

Conclusion

Navigating hidradenitis suppurativa management reveals uncertainty about the role of dietary interventions as a result of the absence of robust controlled clinical studies with diverse patient cohorts. It is essential to approach dietary changes with caution, recognizing that they cannot stand alone as a comprehensive solution to the multifaceted challenges posed by HS. Medical therapy remains the mainstay of treatment, highlighting the need to explore further effective interventions.

Mechanisms linking specific dietary elements and how they may influence HS activity are illustrated in Figure 2. Current literature does indicate that the Mediterranean lifestyle offers promising avenues for exploration alongside established therapies. Conversely, caution is advised regarding high-glycemic index foods and dairy products, which may worsen insulin resistance, inflammation, and potentially HS symptoms. Weight loss is associated with improved HS symptoms because of reduced friction and improved metabolic syndrome. However, bariatric surgery may lead to worsening or new-onset HS as a result of excess skin formation and micronutrient malabsorption. Zinc, with its antioxidative properties and the suspected correlation between vitamin D deficiency and HS severity, points toward potential supplements as adjuncts. Promising dietary factors, including Brewer's yeast-free diets, B12 supplementation, intermittent fasting, and reduced sugar intake, hold potential but demand deeper investigation within HS management.

Despite the intriguing findings presented here, the majority of the studies are based on observational findings and the exact mechanism underlying the effects of dietary factors on HS progression remains unknown. As a result, this review underscores the importance of further research, as the lack of standardized reporting of clinical effects in the analyzed studies calls for a deeper exploration of the pathophysiological mechanisms underlying dietary modifications and their potential associations with HS severity.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Table S1. Studies evaluating the effects of weight loss, bariatric surgery, and adjunctive drugs for weight loss on disease activity.

Table S2. Studies evaluating the effects of zinc on disease activity.

Table S3. Studies evaluating the effects of vitamin D on disease activity.

Table S4. Studies evaluating the effects of other micronutrients on disease activity.

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