Review Article

# Weight Loss and Dietary Interventions for Hidradenitis Suppurativa: A Systematic Review

Journal of Cutaneous Medicine and Surgery
2020, Vol. 24(1) 64–72

DOI: 10.1177/1203475419874412
jcms.sagepub.com

Canadian Dermatology Association
Association canadienne de dermatologie

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**Abstract**

Hidradenitis suppurativa (HS) is a common inflammatory disorder characterized by recurrent, painful, and malodorous abscesses and nodules predominantly in skin folds. HS is associated with substantial morbidity and poor quality of life. There are no curative therapies, and the only approved biologic drug has variable efficacy and requires high doses, making adjunct treatments crucial. An important risk factor for disease severity is obesity. Our primary objective was to conduct a systematic review examining weight loss and dietary interventions, in HS. Our secondary objective was to examine nutritional supplements in HS.A systematic literature search was conducted using Medline, EMBASE, and the Cochrane Database. We included all study types in adults (>18 years), with a minimum sample size of 5, examining the effects of any dietary or weight loss intervention on HS severity. Two authors screened $n = 1279$ articles of which 9 met inclusion criteria. All included studies were observational and all interventions were associated with various measures of decreased HS severity. Patient-controlled weight loss and bariatric surgery were associated with HS regression, though a subset of patients with significant increase in panniculi experienced exacerbations and required excision of excess skin. Diets demonstrating benefit eliminated dairy and brewer's yeast. Nutritional supplements including zinc gluconate, vitamin D, and riboflavin had a suppressive, rather than curative, effect on HS lesions in single studies. Overall, the reviewed interventions show promise as potential adjunct treatments in a HS management plan. Prospective randomized controlled trials should validate these findings.

**Keywords**

hidradenitis suppurativa, obesity, weight loss, diet, lifestyle

## Introduction

Hidradenitis suppurativa (HS) is a chronic, inflammatory skin condition characterized by recurrent abscesses, nodules, and fistulating sinus tracts that commonly present in intertriginous regions.[1-6] Symptoms of HS include malodorous discharge and painful eruptions that often lead to social isolation and stigma.[2,7-10] Studies demonstrate that patients with HS experience a poorer quality of life compared to other dermatologic conditions such as psoriasis.[9]

Estimated HS prevalence ranges from 0.3% to 4%.[11] Females are affected up to 3 times more often.[12] Risk factors include cigarette smoking,[1] obesity,[1,13] high androgen levels,[1] and autosomal inheritance.[1] The *2019 North American clinical management guidelines for hidradenitis suppurativa* stratifies treatments based on Hurley stage and recommends a tailored approach to each patient. Treatments include topical or intralesional therapies, systemic antibiotics, hormonal therapies, retinoids, systemic immunomodulators, biologic drugs, and surgical therapies.[14] Most recently, adalimumab, a TNF inhibitor, has been approved to treat moderate-to-severe HS; however it has variable efficacy.[4]

The association between HS and obesity has been well established, with numerous studies demonstrating greater disease severity with increasing body mass index (BMI).[15-20] A retrospective study of 249 patients with obesity due to undergo bariatric surgery showed a point prevalence for HS

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of 18.1%.[21] Several mechanisms may explain the association of obesity with HS including larger skin folds contributing to increased mechanical friction, humid microenvironments encouraging bacterial proliferation, and obesity contributing a systemic inflammatory state.[22-24]

Lifestyle changes such as dietary modification to promote weight loss have demonstrated HS symptom reduction.[25] Weight loss through bariatric surgery and medical therapies such as liraglutide have also shown promise.[21,26,27] Additionally, diets excluding potential HS triggers such as dairy products, high glycemic index foods, brewer's yeast, and high fat foods have also been reported to reduce HS symptoms.[28-30] Studies suggest that insulin resistance may be one mechanism contributing to these observations. HS has been associated with metabolic syndrome, and the prevalence of insulin resistance is significantly higher among these patients compared to the rest of the population.[31] Intake of dairy, high fat foods, and high glycemic diets have been shown to induce insulin resistance,[32,33] hence these could exacerbate the baseline endocrine dysregulation associated with HS.

Considering the prevalence of HS, its significant morbidity, and the potential impact of lifestyle interventions, an examination of the effectiveness of these strategies is warranted. To date, no review has presented a synthesis on dietary modifications and weight reduction to reduce symptoms of HS. The primary objective of our systematic review is to determine the role of dietary modifications and weight loss on HS in adults (>18 years of age). Our secondary objective is to examine the effect of nutritional supplements on HS severity.

## Materials and Methods

The Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement[34] was used to guide this study (see Supplementary File S1 for checklist). A review protocol following PRISMA-P was created a priori but not registered in a central database (Supplementary File S2). Ethics review is not required for this type of research at our institutions.

### *Eligibility Criteria*

Studies were selected based on the study design, participants, interventions, and outcomes. We included randomized controlled trials (RCTs), cohort studies, case-control studies, and case-series with more than 5 subjects. We excluded cross-sectional studies, case reports, and review papers. Studies had to be conducted on adult (>18 years) subjects. Eligible interventions were any weight reduction strategies and any dietary modifications. Dietary modifications included changes to promote weight loss and the elimination of potential triggering foods. Weight reduction interventions included, but were not limited to, diet or exercise and bariatric surgery. All forms of dietary intervention were included, including but not limited to low-calorie, low-fat, low-carbohydrate, and the Mediterranean diet. All comparators were considered including placebo interventions and usual care. We excluded studies where lifestyle interventions were used concurrently with other medical or surgical therapies. We included studies where any measure of HS severity was reported as an outcome.

### *Information Sources and Search Strategy*

We searched Medline (OVID interface, 1946 onwards), EMBASE (OVID interface, 1947 onwards), and the Cochrane Central Register of Controlled Trials (Wiley interface, current issue) with no limits imposed on study design, date, or language. The initial search conducted on September 3, 2017, was updated on January 15, 2018. Search strategies were created by one reviewer (AS) in consultation with an academic librarian. Examples of search terms include "hidradenitis suppurativa," "body weight," and "diet" (see Supplementary File S3). To ensure comprehensiveness, we examined reference lists of included studies for additional articles. Literature search results were exported to Covidence,[35] an article management software program by the Cochrane Collaboration (Veritas Health Innovation, Melbourne, Australia).

### *Study Selection*

Two authors (AS and CJ) screened the titles and abstracts of articles for the removal of duplicates and for inclusion using our predetermined criteria. The full texts of the selected studies were assessed for eligibility independently in an unblinded manner by AS and CJ.

### *Data Extraction*

Two reviewers (AS. and CJ) used a standard form to extract data independently. Uncertainties in data extraction were resolved by discussion, with a third reviewer available in case of disagreement (PF). Data was extracted on the study type, setting of the intervention, sample size, inclusion criteria, subject characteristics (age in years, sex, BMI in $kg/m^2$, Hurley stage), intervention type, control, follow-up period, and outcomes. Study authors were contacted when the reviewers required additional clarification.

### *Outcomes*

The primary outcome assessed was HS severity as defined by Hurley stage and Hidradenitis Suppurativa Clinical Response. Secondary outcomes included other measures of HS severity such as total abscess and inflammatory-nodule count, number of locations of lesions, and the modified Sartorius score. Various measures of patient quality of life,

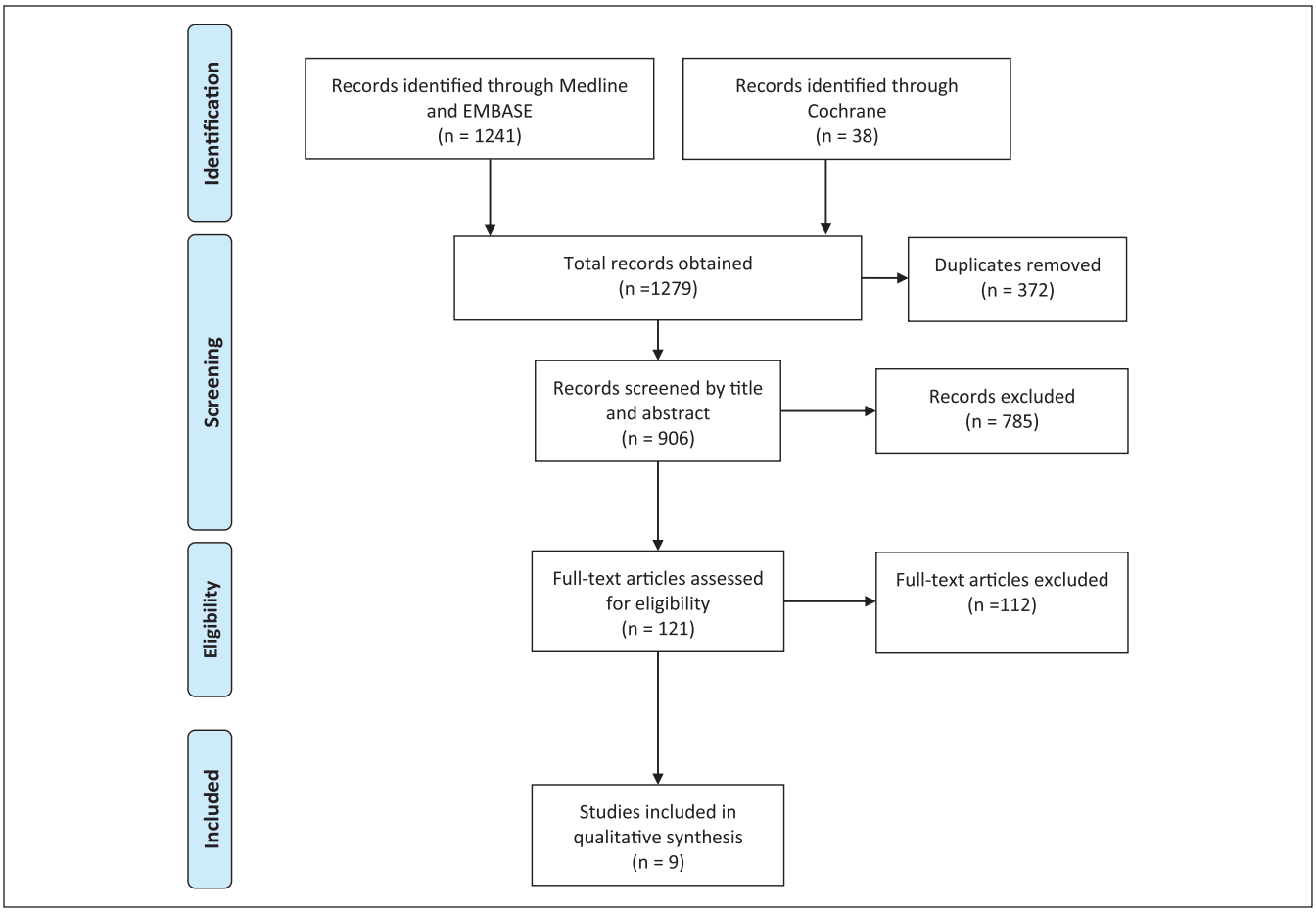


**Figure 1.** PRISMA[33] flowchart illustrating the number of studies obtained through the systematic search, screening, and inclusion stages. PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

pain, depression, and anxiety were also examined. Surrogate measures such as BMI, percent body fat, waist circumference, and hip to waist ratio were also recorded if available.

### *Risk of Bias in Individual Studies*

Risk of bias was assessed by 1 reviewer (AS) using the Newcastle-Ottawa Scale for case-control and cohort studies.[36] Adaptations of the scale were made for case-series studies. RCTs were assessed using the Cochrane Risk of Bias tool.[37] Uncertainties were resolved by consultation with a second reviewer.

### *Data Synthesis*

We planned a narrative synthesis as the anticipated clinical and statistical heterogeneity of the data precluded meta-analyses.

### *Risk of Bias Across Studies*

The Grading of Recommendations Assessment, Development and Evaluation (GRADE) criteria[38] was used by 1 reviewer (AS) to assess the quality of evidence for outcomes across studies for each intervention. Uncertainties were resolved by consultation with a second reviewer. The quality of evidence was reported as "High," "Moderate," "Low," or "Very low."

## Results

Our literature search yielded 1279 results, of which 9 studies[21,28,29,39-44] were ultimately included in the review (Figure 1). All studies (Table 1) were observational, specifically case-series/case-control ($n = 4$),[28,39,43,44] prospective single cohort ($n = 3$),[29,40,42] and retrospective single cohort ($n = 2$).[21,41] Qualitative data synthesis is presented in Table 2.

Most studies were conducted on outpatients at hospital-based dermatology departments, all located in Europe or North America. The combined sample size across studies was 263. Most studies were small, with the largest being a case-series on 78 patients.[28] Demographic data on patients were not reported for all studies. Based on available information, patients were derived from various age groups (range 19-71 years), predominantly female (range 50%-100%), and varied BMI (range 19.1-70.9 kg/m$^2$). Of the 2

**Table 1.** Characteristics of Studies Included in Review.

| Study (author and year) | Study design | Setting | Sample size | Population/relevant inclusion criteria | Mean age (range) | Sex (% male) | Mean BMI (range) | Hurley stage | Intervention vs comparison | Follow-up period | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Weight loss | | | | | | | | | | | |
| Boer (2017)[39] | Case-series | Deventer Hospital, Netherlands | 14 | HS patients with obesity who developed abdominal lesions due to Koebner phenomenon | 37.9 (23-52) | 50% | 35.8 (30.1-45) | I ($n = 8$)<br>II ($n = 6$) | Weight loss vs no weight loss/weight gain | | Extent of abdominal lesions |
| Javorsky et al (2013)[43] | Case-series | Online postings | 35 | HS patients who had bariatric surgery | | | | | Bariatric surgery (no comparison) | | Self-reported HS severity |
| Kromann et al (2014)[45] | Retrospective single cohort | Hvidovre Hospital, Denmark | 35 | HS patients with obesity (BMI > 30) undergoing bariatric surgery (gastric bypass or banding) | 46 (21-67) | 26% | Presurgery: 41.9 (30.9-70.9) | | Bariatric surgery leading to weight loss of at least 15% (no comparison) | 2 y | Mean no. of eruption sites<br>Mean no. of scarring sites |
| Dietary modification | | | | | | | | | | | |
| Cannistra et al (2013)[29] | Prospective single cohort | Hopital Bichat C.B. Paris, France. Catholic University of the Sacred Heart, Rome, Italy. University La Sapienza, Rome, Italy | 12 | All patients with HS followed at center. Patients had incision and debridement or surgical excision, followed by brewer's yeast-free diet | | 42% | 32.25 (19-52) | | Brewer's yeast-free diet for 12 mo (no comparison) | 4 y | Extent of lesions<br>Quality of life |
| Danby (2013)[41] | Retrospective single cohort | | 78 | HS patients on dairy-restricted diet | | | | | Dairy-restricted diet (no comparison) | | Self-reported change in unspecified symptoms |
| Danby (2015)[28] | Case-series with patients acting as own controls | United States | 47 | HS patients on dairy-free diet | | | | | Dairy-free diet (no comparison) | | Self-reported change in unspecified symptoms |
| Supplements | | | | | | | | | | | |
| Mackenna et al (1960)[44] | Case-series | St. Bartholomew's Hospital, London | 6 | HS patients given riboflavin supplement | Range 19-52 | 0% (100% female) | | | Riboflavine 3 mg TID (no comparison) | 3 y | Disappearance of lesions |
| Guillet et al (2015)[42] | Prospective single cohort, within larger case-control study | Nantes Hospital, France | 14 | Patients with HS for at least 1 y, untreated | In sample of 22 (prior to subject dropout)<br>Mean 35 | In sample of 22 (prior to subject dropout)<br>36% | In sample of 22 (prior to subject dropout)<br>Mean 25.95 | | Vitamin D, dosed according to deficiency (no comparison) | 6 mo | Number of nodules<br>Number of flare-ups |
| Brocard et al (2007)[40] | Prospective single cohort | CHU Hôtel-Dieu, Nantes, France | 22 | HS patients who had previously undergone treatment (systemic antibiotic, isotretinoin, surgery, or antiandrogens) that was ineffective | 38.3 (18-71) | 32% | | I: 11<br>II: 10<br>III: 1 | Zinc gluconate 90 mg initially, then reduced dose if achieved partial or complete remission (vs no comparison) | 6 mo | Complete remission (disappearance of lesions or no new lesions for a minimum of 6 mo)<br>Partial remission (minimum 50% reduction of nodules and/or shorter cycle of each inflammatory lesion) |

**Table 2.** Summary of Study Results.

| Study | Intervention | Outcome data | Quality of study according to Newcastle-Ottawa Scale (maximum 9 stars) | Level of evidence for outcome |
|---|---|---|---|---|
| Weight loss | | | | |
| Boer (2017)[39] | Patient-controlled weight loss (degree of weight loss unspecified) | Abdominal lesions regressed first before lesions in other locations in all cases | * | Abdominal lesions regressed first: Very low |
| Javorsky et al (2013)[43] | Bariatric surgery (degree of weight loss unspecified) | 69% had worsening symptoms postoperatively, 26% of which had improvement after removal of excess skin | None | Improvement in self-reported HS severity: Very low |
| Kromann et al (2014)[21] | Bariatric surgery leading to substantial weight loss (at least 15% of BMI) | 17/35 (48.6%) had no HS symptoms, 7/35 (20%) had fewer active eruption sites, 7/35 (20%) had no changes, and 4/35 had more active eruption sites.<br>Statistically significant decrease in mean eruption sites from 1.93 to 1.22 with ES clinically crucial ($P = .003$, ES = 2.92). Mean scarring sites after eruptions reduced from 1.31 to 0.84 ($P = .002$, ES = 0.33) | **** | Decrease in mean number of eruption sites: Very low<br>Decrease in mean number of scarring sites: Very low |
| Dietary modification | | | | |
| Cannistra et al (2013)[29] | Brewer's yeast-free diet | All patients had immediate stabilization of clinical symptoms and complete regression of skin lesions over 12-mo treatment period.<br>All patients had return of quality of life including reestablishment of sexual activity in patients with perigenital lesions | **** | Regression of lesions: Very low<br>Improvement in quality of life: Very low |
| Danby (2014)[41] | Dairy-restricted diet | 65% improved despite partial compliance | * | Self-reported improvement in unspecified symptoms (combined across studies): Very low |
| Danby (2015)[28] | Dairy-free diet | 83% improved, none worsened | *** | |
| Supplements | | | | |
| Mackenna et al (1960)[44] | Riboflavine 3 mg thrice daily | In all cases, lesions disappeared. After several months, could stop supplementation without relapse | None | Disappearance of lesions: Very low |
| Guillet et al (2015)[42] | 25-Hydroxy-vitamin D3 level (ng/mL) vs number of Uvedose ampoules received<br>Vitamin D $\geq$ 30—0<br>25 $\leq$ vitamin D $<$ 30—1<br>20 $\leq$ vitamin D $<$ 25—2<br>15 $\leq$ vitamin D $<$ 20—3<br>10 $\leq$ vitamin D $<$ 15—4<br>5 $\leq$ vitamin D $<$ 10—5<br>Vitamin D $<$ 5—6 | Statistically significant decrease in the number of nodules at 6 mo (M6) with a mean of 51% and a median of 75% ($P = .01133$). Therapeutic success (improvement of at least 20%) at M6 for the number of nodules achieved in 11 (79%) patients ($Chi^2$ test, $P = .08415$)<br>The mean decrease in the frequency of flare-ups was 18% at M6, which was not statistically significant ($P = .2041$). The therapeutic success (improvement of at least 20%) at M6 for the frequency of flare-ups was achieved in 6 (43%) patients (chi-squared test, $P = .982$) | ***** | Decrease in no. of nodules: Very low<br>Decrease in no. of flare-ups: Very low |
| Brocard et al (2007)[40] | Initially 90 mg of zinc gluconate (Rubozinc) daily. When complete remission (disappearance of lesions or no new lesions) or partial remission (minimum 50% reduction in number of nodules or shorter cycle of inflammatory lesion) was maintained for 4 mo, dose was decreased by 15 mg every 2 mo | 8/22 (36%) experienced complete remission. When treatment was reduced, relapses occurred at 30-60 mg daily doses, and recurrence disappeared with dose increase. Partial remission observed in 14/22 (63.6%) patients. None experienced deterioration of HS | *** | Complete remission: Very low<br>Partial remission: Very low |

studies[39,40] that reported Hurley stage, almost all patients were stage I or II.

Studied interventions for HS included weight loss interventions, dietary modifications independent of weight loss, and nutritional supplements. All interventions were associated with an improvement in HS symptoms, though a subset of patients undergoing bariatric surgery experienced exacerbations. In these cases the excess skin resulting from substantial weight loss led to an exacerbation of symptoms that was found in some cases to resolve with removal of excess skin.[43] Various assessments of HS severity were used, with most referring to total number or the extent of, lesions. Several studies assessed HS severity based on patient-reported improvement in unspecified symptoms. No studies reported the modified Sartorius score or the Hidradenitis Suppurativa Clinical Response. All studies were moderate to low quality when assessed by the Newcastle-Ottawa Scale (star range 0-5, out of 9). The level of evidence according to GRADE for all outcomes was "Very low."

Weight loss interventions included unspecified, patient-controlled methods in a case-series ($n = 14$) that led to regression of abdominal lesions before other locations on the body.[39] Two studies examining bariatric surgery showed mixed results. In a case-series ($n = 35$) that did not report the reduction in BMI, 69% of patients worsened postoperatively, with 29% improving once excess skin was removed.[43] In contrast, a retrospective single cohort ($n = 35$) that achieved a minimum 15% reduction in BMI showed statistically significant decreases in mean eruption sites from 1.93 to 1.22 ($P = .003$, ES = 2.92) and scarring sites from 1.31 to 0.84 ($P = .002$, ES = 0.33) post bariatric surgery. Only 11.4% of patients in this study worsened.[45]

Dietary modifications independent of weight loss comprised elimination diets of a potential triggering food or ingredient. A prospective single cohort study ($n = 12$) examining a brewer's yeast (primarily containing the fungus *Saccharomyces cerevisiae*, used in bakery products and fermented foods) free diet for 12 months following surgical excision demonstrated regression of skin lesions over the year and improvement of quality of life in all patients.[29] Two other studies examined dairy restriction. In a retrospective single cohort study ($n = 78$) 65% of patients reported a subjective improvement in symptoms despite partial compliance with a dairy-restricted diet.[41] A case-series with patients acting as their own controls ($n = 47$) found that 83% subjectively reported symptom improvement after eliminating diary.[46]

Nutritional supplementation in HS was investigated in single studies. A case-series ($n = 6$) found that riboflavin (vitamin B2) 3 mg orally 3 times daily over several months was associated with a disappearance of lesions in all subjects.[44] A prospective single cohort study ($n = 14$) found that patients supplemented with vitamin D according to their assessed deficiency (Table 2) experienced a statistically significant decrease after 6 months in the mean number of nodules of 51% ($P = .01$), though the mean decrease in frequency of flare-ups of 18% was not statistically significant ($P = .98$).[42] A prospective single cohort study ($n = 22$) examining zinc gluconate 90 mg (with subsequent dose reduction based on positive response) in HS refractory to previous treatments found that 99.6% experienced a partial or complete remission of symptoms.[40]

## Discussion

The effects of patient-directed weight loss, bariatric surgery, dietary exclusion of brewer's yeast, and dairy restriction on HS severity were examined. Nutritional supplementation with riboflavin, vitamin D, and zinc gluconate was also studied in this review. All interventions were associated with improvement in HS symptoms though 1 study showed mixed results with a majority of patients worsening after bariatric surgery which was partially relieved by excision of excess skin.[43] All studies were observational and of moderate to low quality. All outcomes had a very low evidence, mainly as a result of poor study validity; however, results within studies were consistent with moderate effect sizes. Our review suggests a role for these lifestyle interventions in the treatment of HS.

Of note, the *2019 North American clinical management guidelines for hidradenitis suppurativa*[47] recommends screening for obesity and counseling for weight loss and states that practitioners may recommend oral zinc supplements but notes weak evidence. It states insufficient evidence to recommend avoidance of dairy or brewer's yeast, or to supplement with vitamin D. The guidelines are based on a subset of studies included in this article, hence we present a more comprehensive review here.

The role of weight loss in alleviating HS symptoms has several proposed mechanisms. There is an association of weight loss with fewer affected HS sites. Mechanical stress, such as friction and pressure, is a risk factor in HS.[39] It can cause tears in the acroinfundibulum and initiate a cascade of inflammation that leads to the Koebner phenomenon.[39] Obesity predisposes patients to the effects of mechanical stress[48] by increasing contact with skin or between clothing skin folds. One study found that weight loss causes the regression of abdominal lesions first as contact with tight clothing in this region is relieved.[39] A separate case study showed similar findings with weight loss by dietary measures leading to HS resolution, particularly in frictional areas.[25] Following bariatric surgery, a subset of HS patients and non-HS patients reporting a worsening of skin problems[21,43]and the emergence of new lesions[21] was attributed to excess skin resulting from dramatic weight loss.

Dietary restriction of HS-triggering foods was also investigated. In one included study, patients were thought to have severe reactions to brewer's yeast as lesions reappeared within 24-48 hours following accidental consumption.[29] All patients had detectable IgG antibodies to anti-*S. cerevisiae*

antigen, suggesting that an antibody-mediated reaction to this food antigen activates mechanisms leading to tissue damage.[31] Furthermore, the role of dairy was investigated in another study. Dairy raises insulin and insulin-like growth-factor 1 (IGF-1) levels.[41] These open the androgen receptor, which, when stimulated by dairy androgens, increases production of intraductal keratinocytes.[28] Hyperkeratinization causes follicular occlusion, rupture, inflammation, and eventual disintegration.[41] This is followed by exposure to commensal bacteria, sinus tract formation, and scarring.[41]

With regard to supplementation, several interventions showed an ability to improve HS symptoms. A study that examined riboflavin supplementation showed that when HS patients were administered 50 g of glucose, their blood sugar curves were mostly flat, signaling an inability to absorb or utilize glucose normally.[44] This phenomenon was previously observed in patients with nutritional deficiencies, hence riboflavin supplementation was proposed.[44] While there was clinical improvement, no change in the blood sugar curve was observed,[44] hence the mechanism remains unclear. After several months, riboflavin supplementation could be stopped without relapse.[44] In another study all HS patients were found to be deficient in vitamin D, at a prevalence greater than healthy patients.[42] Supplementing according to the measured vitamin D level led to rapid clinical improvement, though relapses were observed in half the patients who discontinued the supplement, suggesting a suppressive rather than curative effect.[42] With regard to the pathogenesis, several studies have demonstrated deficient skin immunity in HS that leads to an increase in organisms such as coagulase negative staphylococci, streptococci, corynebacteria, and anaerobes.[49] Vitamin D reinforces innate immunity by inducing expression of Toll-Like-Receptor (TLR)-2 and anti-microbial peptides.[50] Vitamin D also affects keratinocyte proliferation and the hair follicle cycle, hence it may prevent the follicular obstruction implicated in HS.[42] Furthermore, the role of zinc in HS has also been linked to boosting innate immunity. A prospective study of patients treated with zinc 90 mg daily for 3 months with subsequent immunohistochemical analysis of HS lesion biopsies showed a statistically significant increase in detectable innate immune markers, including TLR-2,4,7,9, β-defensin 4, intercellular adhesion molecule-1, interleukin (IL)-6, IL-10, tumor necrosis factor, α-melanocyte stimulating hormone, and IGF-1. The same study showed that prior to treatment with zinc, innate immune markers were suppressed in the skin of HS patients compared to controls, particularly in the nonlesional skin of HS patients. Hence, zinc 90 mg may exert its effects by boosting the otherwise deficient innate immunity in the skin in HS patients.[51]

### *Limitations*

Our systematic review has several limitations. At the study level, most included papers had a small sample size. Furthermore, not all studies used objective outcome measures of HS severity. Often the only information available was patient-reported improvement in unspecified HS symptoms. Additionally, none of the studies were RCTs, with the observational studies mostly being of moderate to low quality. At the review level, grey literature was not searched, hence there could be evidence that is unaccounted for in this study.

## Conclusion

There is a demonstrated association of obesity with HS, a chronic inflammatory skin condition. Weight loss has overall been shown to improve HS but may exacerbate symptoms if it results in a pronounced increase in skin folds. HS patients may benefit from restriction of dairy and brewer's yeast, as well as supplementation with zinc, vitamin D, and riboflavin. These studied interventions show promise; however, all studies in our review were observational, hence prospective RCTs are required to strengthen findings. Clinicians may consider incorporating weight loss and the elimination of potential dietary triggers, as lower risk adjunct interventions, into the management plan. Healthy eating should be discussed with HS patients as part of routine care.

### Acknowledgments

A poster based on this project was presented at the at the Canadian Dermatology Association Annual Meeting in Montreal, June 2018, The Symposium on Hidradenitis Suppurativa Advances in Toronto October 2019 and is accepted for presentation as a poster at the upcoming European Academy of Dermatology and Venereology meeting in Madrid October 2019.

### Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr Gulliver has received honoraria from AbbVie, Actelion, Amgen, Arylide, Boehringer, Celgene, Cipher, Eli Lilly, Galderma, Janssen, Leo, Novartis, Pfizer, Roche, Tribute, and Valeant, for participation on advisory boards, consultant services, and speaker engagements. He has also received grants/research support from, or been involved in clinical trials with, AbbVie, Amgen, Astellas, Celgene, Galderma, Janssen, Leo Pharma, Lilly, Novartis, Pfizer, and Regeneron. Dr Alhusayen has been an advisory board member for AbbVie and Janssen. He has also been a consultant for AbbVie. He has received honoraria from AbbVie and Eli Lilly. Dr Fleming has received honoraria and/or consulting fees for AbbVie, Cipher, Galderma, Eli Lilly, Pfizer, and Sanofi. He is an investigator for AbbVie, GlenMark, Incyte, Pfizer, and Valeant.

### Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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## Supplemental Material

Supplemental material for this article is available online.

## References


1. Buimer MG, Wobbes T, Klinkenbijl JH. Hidradenitis suppurativa. *Br J Surg*. 2009;96(4):350-360. doi:10.1002/bjs.6569
2. Jemec GBE. Hidradenitis Suppurativa. *N Engl J Med*. 2012;366(2):158-164. doi:10.1056/NEJMcp1014163
3. Revuz J. Hidradenitis suppurativa. *J Eur Acad Dermatol Venereol*. 2009;23(9):985-998. doi:10.1111/j.1468-3083.2009.03356.x
4. Zouboulis CC, Desai N, Emtestam L, et al. European S1 guideline for the treatment of hidradenitis suppurativa/acne inversa. *J Eur Acad Dermatol Venereol*. 2015;29(4):619-644. doi:10.1111/jdv.12966
5. Lipsker D, Severac F, Freysz M, et al. The ABC of hidradenitis suppurativa: a validated glossary on how to name lesions. *Dermatology*. 2016;232(2):137-142. doi:10.1159/000443878
6. Zouboulis CC, del Marmol V, Mrowietz U, Prens EP, Tzellos T, Jemec GBE. Hidradenitis suppurativa/acne inversa: criteria for diagnosis, severity assessment, classification and disease evaluation. *Dermatology*. 2015;231(2):184-190. doi:10.1159/000431175
7. von der Werth JM, Jemec GB. Morbidity in patients with hidradenitis suppurativa. *Br J Dermatol*. 2001;144(4):809-813.
8. Matusiak Ł, Bieniek A, Szepietowski JC. Psychophysical aspects of hidradenitis suppurativa. *Acta Derm Venereol*. 2010;90(3):264-268. doi:10.2340/00015555-0866
9. Wolkenstein P, Loundou A, Barrau K, Auquier P, Revuz J; Quality of Life Group of the French Society of Dermatology. Quality of life impairment in hidradenitis suppurativa: a study of 61 cases. *J Am Acad Dermatol*. 2007;56(4):621-623. doi:10.1016/j.jaad.2006.08.061
10. Onderdijk AJ, van der Zee HH, Esmann S, et al. Depression in patients with hidradenitis suppurativa: depression in HS patients. *J Eur Acad Dermatol Venereol*. 2013;27(4):473-478. doi:10.1111/j.1468-3083.2012.04468.x
11. Jemec GB, Heidenheim M, Nielsen NH. The prevalence of hidradenitis suppurativa and its potential precursor lesions. *J Am Acad Dermatol*. 1996;35(2 Pt 1):191-194.
12. Brown SC, Kazzazi N, Lord PH. Surgical treatment of perineal hidradenitis suppurativa with special reference to recognition of the perianal form. *Br J Surg*. 1986;73(12):978-980.
13. Edlich RF, Silloway KA, Rodeheaver GT, Cooper PH. Epidemiology, pathology, and treatment of axillary hidradenitis suppurativa. *J Emerg Med*. 1986;4(5):369-378.
14. Alikhan A, Sayed C, Alavi A, et al. North American clinical management guidelines for hidradenitis suppurativa: a publication from the United States and Canadian hidradenitis suppurativa foundations: Part II: topical, intralesional, and systemic medical management. *J Am Acad Dermatol*. 2019;81(1):91-101. doi:10.1016/j.jaad.2019.02.068
15. Sartorius K, Emtestam L, Jemec GB, Lapins J. Objective scoring of hidradenitis suppurativa reflecting the role of tobacco smoking and obesity. *Br J Dermatol*. 2009;161(4):831-839. doi:10.1111/j.1365-2133.2009.09198.x
16. Canoui-Poitrine F, Revuz JE, Wolkenstein P, et al. Clinical characteristics of a series of 302 French patients with hidradenitis suppurativa, with an analysis of factors associated with disease severity. *J Am Acad Dermatol*. 2009;61(1):51-57. doi:10.1016/j.jaad.2009.02.013
17. Crowley JJ, Mekkes JR, Zouboulis CC, et al. Association of hidradenitis suppurativa disease severity with increased risk for systemic comorbidities. *Br J Dermatol*. 2014;171(6):1561-1565. doi:10.1111/bjd.13122
18. Schrader AM, Deckers IE, van der Zee HH, Boer J, Prens EP. Hidradenitis suppurativa: a retrospective study of 846 Dutch patients to identify factors associated with disease severity. *J Am Acad Dermatol*. 2014;71(3):460-467. doi:10.1016/j.jaad.2014.04.001
19. Shalom G, Freud T, Harman-Boehm I, Polishchuk I, Cohen AD. Hidradenitis suppurativa and metabolic syndrome: a comparative cross-sectional study of 3207 patients. *Br J Dermatol*. 2015;173(2):464-470. doi:10.1111/bjd.13777
20. Bettoli V, Naldi L, Cazzaniga S, et al. Overweight, diabetes and disease duration influence clinical severity in hidradenitis suppurativa-acne inversa: evidence from the national Italian registry. *Br J Dermatol*. 2016;174(1):195-197. doi:10.1111/bjd.13864
21. Kromann CB, Ibler KS, Kristiansen VB, Jemec GB. The influence of body weight on the prevalence and severity of hidradenitis suppurativa. *Acta Derm Venereol*. 2014;94(5):553-557. doi:10.2340/00015555-1800
22. Slade DE, Powell BW, Mortimer PS. Hidradenitis suppurativa: pathogenesis and management. *Br J Plast Surg*. 2003;56(5):451-461. doi:10.1016/S0007-1226(03)00177-2
23. Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006;444(7121):860-867. doi:10.1038/nature05485
24. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. *Nat Rev Immunol*. 2011;11(2):85-97. doi:10.1038/nri2921
25. Boer J. Resolution of hidradenitis suppurativa after weight loss by dietary measures, especially on frictional locations. *J Eur Acad Dermatol Venereol*. 2016;30(5):895-896. doi:10.1111/jdv.13059
26. Thomas CL, Gordon KD, Mortimer PS. Rapid resolution of hidradenitis suppurativa after bariatric surgical intervention. *Clin Exp Dermatol*. 2014;39(3):315-318. doi:10.1111/ced.12269
27. Jennings L, Nestor L, Molloy O, Hughes R, Moriarty B, Kirby B. The treatment of hidradenitis suppurativa with the glucagon-like peptide-1 agonist liraglutide. *Br J Dermatol*. 2017;177(3):858-859. doi:10.1111/bjd.15233
28. William Danby F. Diet in the prevention of hidradenitis suppurativa (acne inversa). *J Am Acad Dermatol*. 2015;73(5):S52-S54. doi:10.1016/j.jaad.2015.07.042
29. Cannistrà C, Finocchi V, Trivisonno A, Tambasco D. New perspectives in the treatment of hidradenitis suppurativa: surgery and brewer's yeast-exclusion diet. *Surgery*. 2013;154(5):1126-1130. doi:10.1016/j.surg.2013.04.018
30. Colboc H, Fite C, Cannistra C. Interest of brewer's yeast-exclusion diet in the management of hidradenitis suppurativa. *J Clin Exp Dermatol Res*. 2016;7(5): 1-7. doi:10.4172/2155-9554.1000371
31. Vilanova I, Hernández JL, Mata C, et al. Insulin resistance in hidradenitis suppurativa: a case-control study. *J Eur Acad Dermatol Venereol*. 2018;32(5):820-824. doi:10.1111/jdv.14894
32. Tucker LA, Erickson A, LeCheminant JD, Bailey BW. Dairy consumption and insulin resistance: the role of body fat, physical activity, and energy intake. *J Diabetes Res*. 2015;2015:1-11. doi:10.1155/2015/206959

33. Park S, Kim YW, Kim JY, Jang EC, Doh KO, Lee SK. Effect of high fat diet on insulin resistance: dietary fat versus visceral fat mass. *J Korean Med Sci*. 2001;16(4):386-390. doi:10.3346/jkms.2001.16.4.386
34. Moher D, Shamseer L, Clarke M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Syst Rev*. 2015;4:1. doi:10.1186/2046-4053-4-1
35. Covidence. Better systematic review management. https://www.covidence.org/home. Accessed September 3, 2018.
36. Wells GA, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp. Accessed September 3, 2018.
37. Higgins JPT, Altman DG, Gotzsche PC, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ*. 2011;343:d5928. doi:10.1136/bmj.d5928
38. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*. 2008;336(7650):924-926. doi:10.1136/bmj.39489.470347.AD
39. Boer J. Should hidradenitis suppurativa be included in dermatoses showing koebnerization? Is it friction or fiction? *Dermatology*. 2017;233(1):47-52. doi:10.1159/000472252
40. Brocard A, Knol A-C, Khammari A, Dréno B. Hidradenitis suppurativa and zinc: a new therapeutic approach. *Dermatology*. 2007;214(4):325-327. doi:10.1159/000100883
41. Danby FW. ISSVD 2013 Abstracts. *J Low Genit Tract Dis*. 2013;17(6 Suppl 2):S88. doi:10.1097/LGT.0b013e3182a35c46
42. Guillet A, Brocard A, Bach Ngohou K, et al. Verneuil's disease, innate immunity and vitamin D: a pilot study. *J Eur Acad Dermatol Venereol*. 2015;29(7):1347-1353. doi:10.1111/jdv.12857
43. Javorsky E, Kimball AB. Hidradenitis suppurativa: patient experiences with bariatric surgery. *J Am Acad Dermatol*. 2013;68(4):AB89. doi:10.1016/j.jaad.2012.12.370
44. Mackenna RM, Lehmann H. The glucose tolerance curve in hidradenitis suppurativa. *Br J Dermatol*. 1960;72(4):142-144.
45. Kromann C, Ibler K, Kristiansen V, Jemec G. The Influence of body weight on the prevalence and severity of hidradenitis suppurativa. *Acta Derm Venereol*. 2014;94(5):553-557. doi:10.2340/00015555-1800
46. Danby F. Diet in the prevention of hidradenitis suppurativa (acne inversa). *J Am Acad Dermatol*. 2015;73(5 Suppl 1):S52-54. doi:10.1016/j.jaad.2015.07.042
47. Alikhan A, Sayed C, Alavi A, et al. North American clinical management guidelines for hidradenitis suppurativa: a publication from the United States and Canadian hidradenitis suppurativa foundations: Part I: diagnosis, evaluation, and the use of complementary and procedural management. *J Am Acad Dermatol*. 2019;81(1):76-90. doi:10.1016/j.jaad.2019.02.067
48. Boer J, Nazary M, Riis PT. The role of mechanical stress in hidradenitis suppurativa. *Dermatol Clin*. 2016;34(1):37-43. doi:10.1016/j.det.2015.08.011
49. Sartorius K, Killasli H, Oprica C, Sullivan A, Lapins J. Bacteriology of hidradenitis suppurativa exacerbations and deep tissue cultures obtained during carbon dioxide laser treatment: hidradenitis suppurativa exacerbation bacteriology. *Br J Dermatol*. 2012;166(4):879-883. doi:10.1111/j.1365-2133.2011.10747.x
50. Antal AS, Dombrowski Y, Koglin S, Ruzicka T, Schauber J. Impact of vitamin D3 on cutaneous immunity and antimicrobial peptide expression. *Dermatoendocrinol*. 2011;3(1):18-22. doi:10.4161/derm.3.1.14616
51. Dréno B. Hidradenitis suppurativa: the role of deficient cutaneous innate immunity. *Arch Dermatol*. 2012;148(2):182. doi:10.1001/archdermatol.2011.315