

CME/CE/MOC Offering



Marielle Jamgochian, MBS,
Mahin Alamgir, MD, and
Babar Rao, MD

Diet in Dermatology: Review of Diet's Influence on the Conditions of Rosacea, Hidradenitis Suppurativa, Herpes Labialis, and Vitiligo

Abstract: *The influence of dietary patterns on cutaneous disease has been an oft-posed question to dermatologists by patients in a clinical setting. Similarly, the popularity of nutritional supplementation with vitamins, minerals, and nutraceutical blends has been increasing. Dermatologists, primary care physicians, and other providers should be familiar with dietary interventions that are evidence-based and those that are more marketable than efficacious. In this review, the modification of diet, including dietary exclusion and dietary supplementation for the treatment of rosacea, hidradenitis suppurativa (HS), herpes labialis, and vitiligo was investigated. Despite abundant anecdotal evidence, the literature search found no high-quality evidence that an elimination diet for rosacea “trigger foods” improved rosacea symptoms though these elimination diets (of hot, spicy, alcohol-containing, or cinnamaldehyde-containing foods) had low risk of harm. There is*

evidence that zinc supplementation and vitamin D supplementation in deficient patients is helpful for treating HS. For herpes labialis, L-lysine supplementation was found to be

Polypodium leucotomos in conjunction with phototherapy was found to increase repigmentation, as well as vitamin D supplementation in deficient patients.

 Diet in Dermatology: Review of Diet's Influence on the Conditions of Rosacea, Hidradenitis Suppurativa, Herpes Labialis, and Vitiligo Giving more attention to diseases that are common and may significantly affect quality of life will allow physicians to be informed about dietary impact on the full spectrum of cutaneous disease 

effective for prophylaxis but not for decreasing duration of active lesions. For vitiligo, the use of the herb

Keywords: integrative dermatology; vitiligo; hidradenitis suppurativa; rosacea; herpes

DOI: 10.1177/15598276211026592. From the Rutgers Robert Wood Johnson Medical School, Piscataway, NJ, USA (MJ) and Department of Dermatology, Rutgers Robert Wood Johnson Medical School, Piscataway, NJ, USA (MA, BR). Address correspondence to: Marielle Jamgochian, MBS, Rutgers Robert Wood Johnson Medical School, 675 Hoes Lane West, Piscataway, NJ 08854, USA; e-mail: mjamgoch@rvjms.rutgers.edu.

For reprints and permissions queries, please visit SAGE's Web site at <http://www.sagepub.com/journalsPermissions.nav>.

Copyright © 2021 The Author(s)

Background/Introduction

Dietary influence on systemic disease is a concept nearly as old as medicine itself. The oft-quoted “Let food be thy medicine and medicine be thy food,” attributed to Hippocrates, has been repeatedly cited in the literature and used to legitimize “nutraceutical” formulas and attribute pharmaceutical properties to foods.¹ Medicine has long since moved past the “Four Humors” concept, popular in ancient times. Providers have rightfully transitioned to managing patients based on evidence-based recommendations.

With the rising popularity of nutritional supplements, diet- and lifestyle-based interventions, and increased availability of information and misinformation online, it is essential that physicians stay up-to-date on the existing literature. 77% of adults in the United States take dietary supplements, most commonly a multivitamin, vitamin D, or vitamin C supplement.² Additionally, the percentage of adults who report taking herbal and botanical supplements has increased, in part due to the increasing popularity of green tea supplements, turmeric, and cannabidiol products.² Because of the booming popularity of supplements that parallels the multi-billion dollar “wellness” industry, it is important to have a solid understanding of underlying disease pathophysiology to determine the plausibility of supplements that may in fact be more marketable than truly efficacious.

Physicians should also be informed about the use of herbal supplements in the United States. Under the Dietary Supplement Health and Education Act of 1994, the burden of proof does not lie with the producer to prove the efficacy of their product.³ Most products are brought to the market under the assumption that the ingredients within are “safe”

as they are not placed under the same rigorous scrutiny with which the FDA regulates drugs. Tests of purity of herbal supplements showed DNA from plants not listed on the labels, and they also contained contaminants that could be of concern to consumers’ health.⁴ Thus, providers must be careful in their recommendations, especially those of herbal products that are only regulated in a post-consumer fashion and may be more difficult to test for purity than singular vitamins and minerals.

Finally, though the link between diet and dermatologic disease has been examined through the lens of diseases such as acne, psoriasis, and atopic dermatitis before, it is also imperative that we review the literature for conditions that are less commonly discussed but that still have an impact on patients’ quality of life. Giving more attention to diseases that are common and may significantly affect quality of life will allow physicians to be informed about dietary impact on the full spectrum of cutaneous disease.

It is important for physicians and other providers to be able to answer inquiries from patients concerning the relationship between diet and specific cutaneous diseases within an evidence-based framework. This article will review studies and systematic reviews that examine the relationship between diet, including dietary supplements, and the conditions of rosacea, hidradenitis suppurativa, herpes labialis, and vitiligo. For the conditions listed, relevant studies will be summarized and graded for quality of evidence (Table 1). Then, evidence-based recommendations for dietary changes will be made, providing clinicians a framework by which to recommend or dissuade interventions for patients with dietary concerns.

Rosacea

Rosacea is a chronic inflammatory skin disease with greatest

prominence in light-skinned people of northern European heritage. Clinical presentations are varied, the exact pathophysiology is unknown, and etiology is likely multifactorial due to a dysregulated immune response and vascular and neurogenic factors.^{5,6} Patient-reported triggers for flares include foods, heat, and sun exposure.

Vitamin D. Case-control studies have shown mixed results concerning the relationship between serum vitamin D levels and presence of rosacea. One study from Ekiz et al. found higher mean serum levels of 25-OH-D compared to controls.⁷ However, in this study, the prevalence of vitamin D deficiency in the rosacea group was higher than that in the control group, calling into question whether the study was adequately controlled for confounders. However, increased vitamin D levels are also associated with sun exposure, a factor implicated in rosacea exacerbation; thus, sun exposure may be a confounder. Conversely, Park et al. found that the mean serum level of vitamin D in rosacea patients was significantly lower than controls.⁸ Additionally, cathelicidin, an antimicrobial peptide, is upregulated by vitamin D and implicated in pathogenesis of rosacea.⁸ These mixed results show that larger epidemiological studies are needed to clarify the association between serum vitamin D levels and rosacea presence and severity.

Zinc. Zinc, in its role as a free-radical scavenger, has a wide range of antimicrobial activity.⁹ It is possible that zinc plays a role in reducing Demodex mites or cutaneous bacteria, which are postulated to play a role in rosacea pathogenesis.^{9,10} Oral zinc sulfate (220 mg) did not improve rosacea severity scores compared to placebo.¹¹ Conversely, a small placebo-controlled randomized controlled trial(RCT)

Table 1.Grades of Recommendation.⁵²

Grade of Recommendation	Level of Evidence	Type of Study
A	1a	Systematic review of (homogeneous) randomized controlled trials
A	1b	Individual randomized controlled trials (with narrow confidence intervals)
B	2a	Systematic review of (homogeneous) cohort studies of “exposed” and “unexposed” subjects
B	2b	Individual cohort study/low-quality randomized control studies
B	3a	Systematic review of (homogeneous) case-control studies
B	3b	Individual case-control studies
C	4	Case series, low-quality cohort or case-control studies
D	5	Expert opinions based on non-systematic reviews of results or mechanistic studies

with 25 participants and a high dropout rate showed statistically significant improvement with 300 mg zinc supplementation compared to placebo.¹²

Elimination Diets

Anecdotally, elimination diets have been reported to reduce rosacea flares.¹³ In a 2005 survey of over 400 patients by the National Rosacea Society, 78% of respondents reported altering their diet due to rosacea, with 95% of them reporting reduced flares after dietary changes.¹⁴ Foods that reportedly cause flares fall into four general categories: hot foods, spicy foods, cinnamaldehyde-containing foods, and alcohol.¹³ Though there is mechanistic evidence to support elimination diets for control of rosacea symptoms, RCT evidence is lacking due to difficulties in designing such studies. However, avoidance of the aforementioned foods is an intervention unlikely to cause harm and may be used as an adjunct to traditional treatments.

Summary

Case-control studies seeking to quantify serum levels of vitamin D in rosacea patients have shown mixed results. Despite the possible role of vitamin D in rosacea pathogenesis, there is inadequate evidence to suggest that vitamin D supplementation can relieve or exacerbate rosacea symptoms, so more research is needed. RCTs examining zinc supplementation for rosacea have similarly yielded mixed results, with insufficient evidence to support its use. Diets eliminating foods implicated in rosacea, such as spicy, hot, alcohol, and cinnamaldehyde-containing foods, are supported by anecdotal and mechanistic evidence and unlikely to cause harm. A summary of recommendations for rosacea can be found in [Table 2](#).

Hidradenitis Suppurativa

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease consisting of painful scarring nodules and abscesses leading to

draining sinus tracts. It is located in areas with abundant apocrine glands: in the groin, axilla, and surrounding areas. The pathogenesis of HS is multifactorial. Immune dysregulation in hair follicles, and subsequent keratin plugging, and bacterial colonization may play a role. In addition, inflammatory environmental factors strongly associated with HS include smoking, obesity, and insulin resistance. Along with obesity, micronutrient deficiency may play a role.¹⁵ Based on a recent meta-analysis, HS patients are four times more likely to be obese than the general population.¹⁵ HS patients are also more likely to be insulin resistant or develop insulin resistance than controls.¹⁶ Further, HS patients often have higher levels of pro-inflammatory cytokines including TNF-alpha, IL-1B, and IL-17, lending evidence that HS is correlated to a pro-inflammatory state.^{17,18} The skin-gut axis has also been postulated to play a role.⁶

Table 2.

Recommendations for Rosacea.

Dietary Modification	Recommendation	Grade and Level of Evidence
Vitamin D supplementation	Insufficient evidence for recommendation	Grade D, level 5
Zinc supplementation	Insufficient evidence for recommendation	Grade B, level 2b
Elimination diet	Insufficient evidence for recommendation, with low risk of harm	Grade D, level 5

Dairy. Dairy products have been postulated to play a role in the development of HS as dairy components such as casein, whey, insulin-like growth factor 1 (IGF1), and other androgenic factors can stimulate the pilosebaceous unit, possibly exacerbating HS.¹⁹⁻²¹

Mechanistic studies have shown that dairy and other foods containing high proportions of leucine can increase activity in the mTOR pathway, leading to increased lipogenesis in sebaceous glands.⁶ Some small studies have found increased mTOR activity in HS patients.²² In a pilot cohort study of 47 patients following a dairy-free diet, 83% were reported to have a degree of improvement in disease.²¹ No high-quality RCTs have been performed, as difficulties in allocation and blinding are present when trying to test elimination diets.

Low GI/GL Diet. Patients with HS have higher levels of insulin resistance than age- and sex-matched controls.¹⁶ This relationship holds true even when controlling for BMI.^{16,23} Thus, some have suggested that a low GI/GL diet could be beneficial in HS. However, no controlled trials have been performed.

“Brewer’s Yeast Free”. Patients in one prospective case series were found to be positive for anti-*Saccharomyces cerevisiae* antibodies and were placed on yeast-exclusion diets.²⁴ 12 patients showed improvement and lesion stabilization over 12 months. In the other series, 20 patients were evaluated after a yeast-

exclusion diet for 3 months, and a mean improvement in pain and “number of impaired days” was reported.²⁵ This was seen independent of weight loss. Since yeast-free diets also tend to be lower in processed foods such as baked goods, the results could be confounded by patients adhering to an overall “healthier” diet with fruits and vegetables and fewer processed foods.

Weight Loss Interventions. Since obesity is correlated to the presence of HS, weight loss can help the course of disease. Mechanistically, weight loss can minimize friction in skin folds which contributes to pilosebaceous unit plugging and tract formation.¹⁵ In addition, patients who are obese tend to have higher incidence of insulin resistance as well as a greater all-round inflammatory milieu.²⁶ Micronutrient deficiencies could play a role, as many with obesity may also be malnourished.¹⁵ As one of the most effective long-term weight loss strategies available for patients appears to be bariatric surgery, some investigations have addressed this procedure. In a retrospective cohort study, of 45 patients with HS who underwent bariatric surgery, 49% (N = 17) had resolution of disease. Despite some success, 11% (N = 4) had worsening HS severity, possibly due to the micronutrient malabsorption associated with such procedures.²⁷

Zinc. Micronutrient and mineral deficiencies have been postulated to play a role in the pathogenesis of HS.

For example, a case-control study by Proveda et al. (N = 244) demonstrated an OR of 6.7 for zinc deficiency when comparing HS patients to age-matched healthy controls.²⁸ In a retrospective cohort study, Hessam et al. investigated 66 patients taking 90 mg/day of oral zinc supplementation and found a significant difference in Dermatology life quality index, number of inflammatory nodules, and erythema scores compared to controls, though other subjective measures like fistula count and visual analogue scale scores did not differ significantly.²⁹ In a pilot study (N = 22), patients experienced at least partial remission with 8 achieving full remission on 90 mg/day of zinc gluconate.³⁰ Side effects include abdominal pain and GI disturbance (22%) but for patients who can tolerate it, zinc supplementation seems like a safe adjunct.^{29,30}

Vitamin D. HS patients may also be deficient in vitamin D. Vitamin D deficiency is common overall, but case-control and cross-sectional studies have found that HS patients are 5.47 times more likely to be deficient.^{31,32} In a pilot cohort study by Guillet et al., authors supplemented vitamin D-deficient HS patients per their baseline needs, showing statistically significant decreases in nodules and disease flares at 6 months.³¹ Despite promising results, large-scale randomized trials have not been performed.

Summary. A dairy elimination diet has shown promising results in case series, but the evidence at this time is not adequate to recommend dairy elimination. Similarly, while mechanistic data and animal studies support a role of high GI/GL diets in the pathogenesis of HS, there is not enough high-quality evidence to recommend these diets for treatment of HS. Additionally, more data are needed before the clinical significance of ASCA levels is determined, and higher quality studies with more clinically significant endpoints are needed before a yeast-free diet could be recommended to patients. While weight loss does seem to improve HS outcomes in many patients, bariatric surgeries can lead to increased incidence of micronutrient deficiencies and recommendations to undergo surgery should be based on surgical society guidelines. Finally, zinc and vitamin D supplementation should be considered in HS patients, especially if deficient. A summary of recommendations for HS can be found in [Table 3](#).

Herpes Labialis

Herpes labialis occurs as a result of recurrent exacerbation of an infection with the human herpes simplex virus, a DNA virus with 2 subtypes, HSV1 and HSV2. HSV1 is the strain more likely to infect the oral mucosa and is common, with up to 90% of US adults estimated to have had previous exposure.³³ Despite this, the majority of those infected do not have recurrent outbreaks; an estimated one-third have recurrent episodes.³⁴ Commonly cited triggers of recurrent outbreaks include stresses such as illness or pregnancy, as well as increased UV exposure.³⁴

L-lysine. Based on studies in tissue culture, HSV uses arginine-rich proteins in order to facilitate reproduction, and lysine inhibits viral reproduction.³³ A 2017 review

article detailed 12 studies, ten of which investigated the prophylactic use of lysine for prevention of cold sores and mouth ulcers and 2 of which investigated lysine supplementation for treatment of active lesions. The 2 studies on active lesions showed no statistically significant differences in lesion duration.³³ The studies on prevention showed mixed results, with no controlled studies showing statistically significant reduction in lesion frequency at doses less than 1 gram/day. At higher doses (1.2–1.26 grams per day), studies found mixed results, with 2 showing significant reductions in frequency and 2 showing no significant difference. The highest dose tested was 3 grams per day, which demonstrated statistically significant reductions in lesion recurrence³³ and has been demonstrated to be a safe dose.³⁵ **Conversely, foods high in arginine are proposed to precipitate lesion recurrence. Some studies have investigated the use of L-lysine supplementation in conjunction with a low arginine diet, but none investigating a low arginine diet alone.³³ Use of a low arginine diet in conjunction with L-lysine supplementation may be associated with reduction in lesion frequency.³³ However, more research needs to be done in order to recommend use of a low arginine diet.**

Zinc. Case-control studies have described lower salivary zinc levels in patients with recurrent herpes labialis.^{10,36} Zinc salts have been shown to damage HSV in vitro by inhibiting viral protein production and DNA replication as well as deactivating free viruses.³⁷ Oral zinc supplementation trials have not been randomized or placebo controlled, and thus there is insufficient evidence to suggest zinc supplementation for prophylaxis or treatment of active lesions. Long-term zinc supplementation can lead to copper

deficiency, and thus concurrent copper supplementation should be recommended if a patient chooses to take oral zinc supplements.³⁸

Summary. L-lysine supplementation can be effective in reducing recurrence of herpes labialis lesions in patients with recurrent outbreaks. Evidence supports use at 3 g/day, as lower levels of supplementation show mixed evidence of effectiveness. L-lysine supplementation has not proven effective for reduction in duration or symptoms of active lesions. Oral zinc supplementation has insufficient evidence to recommend its use at this time. A summary of recommendations for herpes labialis can be found in [Table 4](#).

Vitiligo

Oxidative stress is thought to play an important role in the pathogenesis of vitiligo. The literature supports that vitiligo patients have a decreased total antioxidant activity as well as increased markers of oxidative stress.³⁹

Vitamin C and B12. In a study on vitamin C, vitamin B12, and broadband UVB, 9 patients were treated, and they experienced adequate repigmentation, achieved over 6–8 weeks.⁴⁰ However, this was an observational study with no control group. Similarly, a study showed that combining potent topical corticosteroids with oral vitamin B12 and vitamin C supplementation was effective for vitiligo.⁴¹ This was also observational with no control groups; thus, more research needs to be done to determine the effect of vitamin supplementation over potent topical corticosteroid use alone.

Vitamin D. Cross-sectional data on vitamin D status in vitiligo patients show mixed results. A pilot study on 16 vitamin D–deficient vitiligo patients examined efficacy of high-dose oral vitamin D supplementation on vitiligo

Table 3.

Recommendations for Hidradenitis Suppurativa.

Dietary Modification	Recommendation	Grade and Level of Evidence
Dairy elimination	Insufficient evidence for recommendation	Grade D, level 5
Low glycemic index/Glycemic load diet	Insufficient evidence for recommendation	Grade D, level 5
Brewer's yeast free	Insufficient evidence for recommendation for patients without yeast sensitivity	Grade C, level 4
Weight loss—Bariatric surgery	Insufficient evidence for recommendation	Grade B, level 2b
Zinc supplementation	Yes	Grade B, level 2a
Vitamin D supplementation	Yes, if vitamin D deficient	Grade B, level 2b

Table 4.

Recommendations for Herpes Labialis.

Dietary Modification	Recommendation	Grade and Level of Evidence
L-Lysine for prophylaxis	Yes, 3 g/day	Grade B, level 2b
L-lysine for active lesions	No	Grade B, level 2b
Zinc supplementation	Insufficient evidence for recommendation	Grade C, level 4

Table 5.

Recommendations for Vitiligo.

Dietary Modification	Recommendation	Grade and Level of Evidence
Vitamin B12/Vitamin C supplementation	Insufficient evidence for recommendation	Grade C, level 4
Vitamin D	Yes, if deficient	Grade C, level 4
Vitamin E	Insufficient evidence for recommendation	Grade B, level 3b
"Antioxidant blend"	Insufficient evidence for recommendation	Grade B, level 2b
<i>Polypodium leucotomos</i>	Yes, in conjunction with phototherapy	Grade B, level 2b
Gingko	Insufficient evidence for recommendation	Grade B, level 2b

repigmentation.⁴² Ten of 16 patients in the study had 26–75% repigmentation with ingestion of 35,000 IU per day. Patients had significantly increased serum vitamin D levels after supplementation. Despite this high

level of supplementation, this level caused minimal adverse effects, as patients were kept on a lower calcium diet in order to prevent hypervitaminosis D and hypercalcemic effects.⁴² Per current guidelines based on musculoskeletal

outcomes, patients with vitiligo and concurrent vitamin D deficiency should be supplemented; however, ideal doses for vitiligo are not established. In addition, screening criteria for vitamin D levels are inconsistent and there is no universal

consensus on which individuals should be screened.⁴³

Vitamin E. Vitamin E, or α -tocopherol, is a vitamin that acts as an antioxidant. Vitamin E supplementation has been investigated as an adjunct to phototherapy in vitiligo. One study compared products of lipid peroxidation in patients taking vitamin E + NB-UVB to those taking non-broadband ultraviolet B (NB-UVB) alone and found products of lipid peroxidation were reduced in the active group compared to controls.⁴⁴ The active group achieved higher levels of repigmentation compared to the control group though this did not undergo statistical analysis.

Antioxidant Blend. A randomized, placebo-controlled study (N = 28) sought to compare an antioxidant blend “antioxidant pool (AP)” containing α -lipoic acid (50 mg), vitamin C (50 mg) and vitamin E (20 mg), poly-unsaturated fatty acids (PUFA) (12%), and cystine monohydrate (50 mg) plus NB-UVB to NB-UVB alone.⁴⁵ The patients taking AP plus NB-UVB achieved greater therapeutic success compared to controls. In addition, markers of antioxidant activity were increased with decreased levels of reactive oxygen species in controls. **Though these results are promising, the clinical significance of these results is unclear and further studies should be done to**

further evaluate the effectiveness of this particular antioxidant blend.

Polypodium Leucotomos.

Polypodium leucotomos is a plant in the fern family that has both antioxidant and immunomodulating properties.⁴⁶ Various reviews contend the plant can be effective for pigmentation disorders when used in conjunction with NB-UVB.^{46,47} No studies have been published with *Polypodium* alone, without concurrent phototherapy.

Middelkamp et al. authored a placebo-controlled, double-blind study comparing the use of *P. leucotomos* in conjunction with NB-UVB therapy.⁴⁸ The authors showed that patients taking the supplement had significantly more repigmentation in the head and neck area compared to placebo. No significant differences were seen in other body areas. In addition, the effects were most pronounced in those with types II and III skin; the study was not adequately powered to detect an effect in skin types IV–V. The most common reported adverse effect was GI upset.

In Reyes et al., researchers investigated the impact of adding *P. leucotomos* to Psoralen and ultraviolet A (PUVA) therapy.⁴⁹ It was a randomized double-blind placebo-controlled study which found that the subjects allocated to the PUVA + *P. leucotomos* group had a higher percentage of subjects with 50% or greater repigmentation.

Ginkgo biloba. In a pilot clinical trial by Szczurco et al., 12 patients were administered 60 mg of *Ginkgo biloba* twice daily.⁵⁰ The subjects reported improvement in vitiligo measures without adverse effects, but better-controlled, larger RCTs will be needed. A double-blind placebo-controlled RCT studying *Ginkgo biloba* extract with folic acid showed statistically significant improvement in skin repigmentation and slowing in spread of disease compared to placebo.⁵¹ The cited studies do not report serious adverse events.^{50,51} However, *Ginkgo biloba* has theoretical risks of clotting abnormalities or medication interactions. As such, the risks of using ginkgo may outweigh the known benefits of this supplement.

Summary. Antioxidant mixes and vitamin blends purported to improve repigmentation in vitiligo do not have robust evidence supporting their use alone, as the majority of studies have sought to clarify their role as an adjunct to high-potency topical steroids, non-steroidal topicals, and phototherapy. Due to mixed results and low-quality study design, there is insufficient evidence to recommend their use. Vitamin D should be supplemented if deficient, but its role in improving vitiligo repigmentation is unclear. There is sufficient evidence to recommend supplementation with *P. leucotomos* as an adjunct to phototherapy. A summary of recommendations for vitiligo can be found in [Table 5](#).

CME/CE Article Quiz

American College of Lifestyle Medicine (ACLM) members can earn FREE CME/CE credit by reading this approved CME/CE article and successfully completing the online CME/CE activity. Non-members can earn CME/CE for \$40 per article. Visit lifestylemedicine.org to join the ACLM.

Instructions.

1. AJLM CME/CE Articles and Quizzes are offered online only through the American College of Lifestyle Medicine and are accessible at lifestylemedicine.org/store. ACLM Members can enroll in the activity, complete the quiz, and earn this CME/CE for free. Non-members will be charged \$40 per article.
2. A Passing score of 80% or higher is required in order to be awarded the CME/CE credit.

Conclusion

Research on the effect of diet on cutaneous disease is in its infancy. There is an abundance of mechanistic rationale for supplementation or elimination of certain foods, vitamins, and minerals, but not much evidence for clinical use in the form of randomized controlled trials, both due to difficulty in designing such trials and the lack of funding for nutrition-based interventions. Relaxed regulations on vitamins and supplements within the United States make contamination, especially of herbal products, a danger, and providers should keep this in mind when recommending supplements to their patients. There exists a need for further research into the role of dietary supplementation or elimination in the treatment or management of dermatologic disease.

Summary

- Previous investigations have examined the relationship between diet and disease in rosacea, hidradenitis suppurativa, herpes labialis, and vitiligo.
- We sought to summarize evidence from published literature in order to provide recommendations for or against different dietary supplements/modifications.
- Concise recommendations will help providers provide evidence-based, up-to-date care and recommendations for patients.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

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

Ethical Approval

Not applicable, because this article does not contain any studies with human or animal subjects.

ORCID iD

Marielle Jamgochian  <https://orcid.org/0000-0002-0009-2131> 

References

1. Cardenas D. Let not thy food be confused with thy medicine: The hippocratic misquotation. *E-SPEN J*. 2013;8(6):e260-e262. doi:10.1016/j.clnme.2013.10.002.
2. CRN consumer survey on dietary supplements-council for responsible nutrition. 2019. <https://www.crnusa.org/2019survey/Topline-Infographic#more>. Accessed October 1, 2020.
3. Dietary supplement health and education Act of 1994. 2020. https://ods.od.nih.gov/About/DSHEA_Wording.aspx. Accessed October 1, 2020.
4. Newmaster SG, Grguric M, Shanmughanandhan D, Ramalingam S, Ragupathy S. DNA barcoding detects contamination and substitution in North American herbal products. *BMC Med*. 2013;11(1):222. doi:10.1186/1741-7015-11-222.
5. Rainer BM, Kang S, Chien AL. Rosacea: Epidemiology, pathogenesis, and treatment. *Derm Endocrinol*. 2017;9(1):e1361574. doi:10.1080/19381980.2017.1361574.
6. Maarouf M, Platto JF, Shi VY. The role of nutrition in inflammatory pilosebaceous disorders: Implication of the skin-gut axis. *Australas J Dermatol*. 2019;60(2):e90-e98. doi:10.1111/ajd.12909.
7. Ekiz Ö, Balta İ, Şen BB, Dikilitaş MC, Özüğüz P, Rifaioğlu EN. Vitamin D status in patients with rosacea. *Cutan Ocul Toxicol*. 2014;33(1):60-62. doi:10.3109/15569527.2013.797907.
8. Park BW, Ha JM, Cho EB, et al. A study on vitamin D and cathelicidin status in patients with rosacea: Serum level and tissue expression. *Ann Dermatol*. 2018;30(2):136-142. doi:10.5021/ad.2018.30.2.136.
9. Gupta M, Mahajan VK, Mehta KS, Chauhan PS. Zinc therapy in dermatology: A review. *Dermatol Res Pract*. 2014;2014:1-11. doi:10.1155/2014/709152.
10. Read SA, Obeid S, Ahlenstiel C, Ahlenstiel G. The role of zinc in antiviral immunity. *Adv Nutr*. 2019;10(4):696-710. doi:10.1093/advances/nmz013.
11. Bamford JTM, Gessert CE, Haller IV, Kruger K, Johnson BP. Randomized, double-blind trial of 220 mg zinc sulfate twice daily in the treatment of rosacea. *Int J Dermatol*. 2012;51(4):459-462. doi:10.1111/j.1365-4632.2011.05353.x.
12. Sharquie KE, Najim RA, Al-Salman HN. Oral zinc sulfate in the treatment of rosacea: A double-blind, placebo-controlled study. *Int J Dermatol*. 2006;45(7):857-861. doi:10.1111/j.1365-4632.2006.02944.x.
13. Weiss E, Katta R. Diet and rosacea: The role of dietary change in the management of rosacea. *Dermatol Pract Concept*. 2017;7(4):31-37. doi:10.5826/dpc.0704a08.
14. Drake L. Hot sauce, wine and tomatoes cause flare-ups, survey finds. Rosacea.org. <https://www.rosacea.org/rosacea-review/2005/fall/hot-sauce-wine-and-tomatoes-cause-flare-ups-survey-finds>. Accessed January 22, 2021
15. Choi F, Lehmer L, Ekelem C, Mesinkovska NA. Dietary and metabolic factors in the pathogenesis of hidradenitis suppurativa: A systematic review. *Int J Dermatol*. 2020;59(2):143-153. doi:10.1111/ijd.14691.
16. 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.
17. Mozeika E, Pilmane M, Nürnberg B, Jemec G. Tumour necrosis factor-alpha and matrix metalloproteinase-2 are expressed strongly in hidradenitis suppurativa. *Acta Derm Venereol*. 2013;93(3):301-304. doi:10.2340/00015555-1492.
18. Sabat R, Jemec GBE, Matusiak Ł, Kimball AB, Prens E, Wolk K. Hidradenitis suppurativa. *Nat Rev Dis Primers*. 2020;6(1):1-20. doi:10.1038/s41572-020-0149-1.
19. Kucharska A, Szmurlo A, Sińska B. Significance of diet in treated and untreated acne vulgaris. *Adv Dermatol Allergol*. 2016;2(2):81-86. doi:10.5114/ada.2016.59146.

20. Kurzen H, Kurzen M. Secondary prevention of hidradenitis suppurativa. *Dermatol Rep.* 2019;11(2):8243. doi:10.4081/dr.2019.8243.
21. Danby FW. Diet in the prevention of hidradenitis suppurativa (acne inversa). *J Am Acad Dermatol.* 2015; 73(5 suppl 1):S52-S54. doi:10.1016/j.jaad.2015.07.042.
22. Monfrecola G, Balato A, Caiazzo G, et al. Mammalian target of rapamycin, insulin resistance and hidradenitis suppurativa: A possible metabolic loop. *J Eur Acad Dermatol Venereol.* 2016;30(9): 1631-1633. doi:10.1111/jdv.13233.
23. Akdogan N, Alli N, Uysal PI, Topcuoglu C, Candar T, Turhan T. Visfatin and insulin levels and cigarette smoking are independent risk factors for hidradenitis suppurativa: A case-control study. *Arch Dermatol Res.* 2018;310(10):785-793. doi:10.1007/s00403-018-1867-z.
24. 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.
25. Colboc H, Fite C, Cannistrà C. Interest of brewer's yeast-exclusion diet in the management of hidradenitis suppurativa. *J Clin Exp Dermatol Res.* 2016;7:5. doi:10.4172/2155-9554.1000371.
26. Vinkel C, Thomsen SF. Hidradenitis suppurativa: Causes, features, and current treatments. *J Clin Aesthetic Dermatol.* 2018;11(10):17-23.
27. 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.
28. Poveda I. Serum zinc levels in hidradenitis suppurativa: A case-control study. *Am J Clin Dermatol.* 2018;19(5):771-777.
29. Hessam S, Sand M, Meier NM, Gambichler T, Scholl L, Bechara FG. Combination of oral zinc gluconate and topical triclosan: An anti-inflammatory treatment modality for initial hidradenitis suppurativa. *J Dermatol Sci.* 2016;84(2):197-202. doi:10.1016/j.jdermsci.2016.08.010.
30. Brocard A, Knol A-C, Khammari A, Dréno B. Hidradenitis suppurativa and zinc: A new therapeutic approach. *Dermatol.* 2007;214(4):325-327. doi:10.1159/000100883.
31. 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.
32. Kelly G, Sweeney CM, Fitzgerald R, et al. Vitamin D status in hidradenitis suppurativa. *Br J Dermatol.* 2014;170(6): 1379-1380. doi:10.1111/bjd.12900.
33. Mailoo VJ, Rampes S. Lysine for herpes simplex prophylaxis: A review of the evidence. *Integr Med.* 2017;16(3): 42-46.
34. Opstelten W, Neven AK, Eekhof J. Treatment and prevention of herpes labialis. *Can Fam Physician.* 2008; 54(12):1683-1687.
35. Bumpstead L. Long-term use of supplemental lysine is it safe? *J Aust Tradit-Med Soc.* 2013;19(4):228-236.
36. Jafari N, Attar A, Jafari S, Ataie M, Khozeimeh F. Comparative analysis of salivary zinc level in recurrent herpes labialis. *Dent Res J.* 2012;9(1): 19-23. doi:10.4103/1735-3327.92922.
37. Arens M, Travis S. Zinc salts inactivate clinical isolates of herpes simplex virus in vitro. *J Clin Microbiol.* 2000;38(5): 1758-1762.
38. Gaby AR. Natural Remedies for Herpes simplex. *Altern Med Rev.* 2006;11(2):9.
39. Speeckaert R, Dugardin J, Lambert J, et al. Critical appraisal of the oxidative stress pathway in vitiligo: A systematic review and meta-analysis. *J Eur Acad Dermatol Venereol.* 2018;32(7): 1089-1098. doi:10.1111/jdv.14792.
40. Don P, Iuga A, Dacko A, Hardick K. Treatment of vitiligo with broadband ultraviolet B and vitamins. *Int J Dermatol.* 2006;45(1):63-65. doi:10.1111/j.1365-4632.2005.02447.x.
41. Sendrasoa FA, Ranaivo IM, Sata M, et al. Treatment responses in patients with vitiligo to very potent topical corticosteroids combined with vitaminotherapy in Madagascar. *Int J Dermatol.* 2019;58(8):908-911. doi:10.1111/ijd.14510.
42. Finamor DC, Sinaglia-Coimbra R, Neves LCM, et al. A pilot study assessing the effect of prolonged administration of high daily doses of vitamin D on the clinical course of vitiligo and psoriasis. *Derm Endocrinol.* 2013;5(1):222-234. doi:10.4161/derm.24808.
43. Pilz S, Zittermann A, Trummer C, et al. Vitamin D testing and treatment: A narrative review of current evidence. *Endocr Connect.* 2019;8(2):R27-R43. doi:10.1530/EC-18-0432.
44. Elgoweini M, Din NNE. Response of vitiligo to narrowband ultraviolet B and oral antioxidants. *J Clin Pharmacol.* 2009;49(7):852-855. doi:10.1177/0091270009335769.
45. Dell'Anna ML, Mastrofrancesco A, Sala R, et al. Antioxidants and narrow band-UVB in the treatment of vitiligo: A double-blind placebo controlled trial. *Clin Exp Dermatol.* 2007;32(6):631-636. doi:10.1111/j.1365-2230.2007.02514.x.
46. Cohen BE, Elbuluk N, Mu EW, Orlov SJ. Alternative systemic treatments for vitiligo: A review. *Am J Clin Dermatol.* 2015;16(6):463-474. doi:10.1007/s40257-015-0153-5.
47. Nestor M, Bucay V, Callender V, Cohen JL, Sadick N, Waldorf H. Polypodium leucotomos as an adjunct treatment of pigmentary disorders. *J Clin Aesthet Dermatol.* 2014;7(3):13-17.
48. Middelkamp-Hup M, Bos J, Rius-Diaz F, Gonzalez S, Westerhof W. Treatment of vitiligo vulgaris with narrow-band UVB and oral Polypodium leucotomos extract: A randomized double-blind placebo-controlled study. *J Eur Acad Dermatol Venereol.* 2007;21(7):942-950. doi:10.1111/j.1468-3083.2006.02132.x.
49. Reyes E, Jaén P, Heras EDL, et al. Systemic immunomodulatory effects of Polypodium leucotomos as an adjuvant to PUVA therapy in generalized vitiligo: A pilot study. *J Dermatol Sci.* 2006;41(3):213-216. doi:10.1016/j.jdermsci.2005.12.006.
50. Szczurko O, Shear N, Taddio A, Boon H. Ginkgo biloba for the treatment of vitiligo vulgaris: An open label pilot clinical trial. *BMC Compl Alternative Med.* 2011;11:21. doi:10.1186/1472-6882-11-21.
51. Parsad D, Pandhi R, Juneja A. Effectiveness of oral Ginkgo biloba in treating limited, slowly spreading vitiligo. *Clin Exp Dermatol.* 2003;28(3):285-287. doi:10.1046/j.1365-2230.2003.01207.x.
52. Saragossi J. Research & subject guides: Evidence-based medicine: Levels of evidence. https://guides.library.stonybrook.edu/evidence-based-medicine/levels_of_evidence. Accessed October 16, 2020.