Acne: The Role of Medical Nutrition Therapy

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ABSTRACT

Acne is a common disease in Westernized nations, particularly among adolescents and young adults. Acne has substantial effects on quality of life, making treatment essential. Medical nutrition therapy as a potential treatment for acne is not new, although the literature examining diet and acne during the past 100 years is mixed. During the late 1800s and early 1900s, diet was commonly used as an adjunct treatment for acne. During the 1960s, however, the diet–acne connection fell out of favor. In recent years, dermatologists and registered dietitians have revisited the idea and become increasingly interested in the role of medical nutrition therapy in acne treatment. This article reviews the history and existing literature examining the association between diet and acne. Although the total number of studies conducted within the past 40 years is relatively small, the growing body of epidemiologic and experimental evidence suggests a relationship between diet and acne. Compared with other dietary factors, more research examines dietary glycemic load. The evidence is more convincing for high glycemic load diets, compared with other dietary factors. To date there are no randomized controlled trials investigating the relationship between frequent dairy or milk consumption and acne. Similarly, the number of research studies examining the relationship between dietary fat and/or n-3 fatty acids is sparse and the evidence is less robust. Taken together, several methodologic limitations need to be addressed, and additional research, preferably randomized controlled trials, is warranted before comprehensive evidence-based guidelines can be established. While dermatologists and registered dietitians continue to debate and research the potential relationship between diet and acne, the best dietary approach is to address each acne patient individually, carefully considering the possibility of dietary counseling.
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ACNE IS RELATIVELY COMMON DISEASE, AFFECTING more than 17 million Americans and approximately 80% to 90% of American adolescents.[1] Although acne incidence peaks during adolescence, the condition frequently continues into adulthood with the mean age of treatment approximately 24 years of age.[2] Although stereotypes suggest acne is a trivial, self-limiting, cosmetic disorder, acne is not an insignificant problem.[3] The social, psychological, and emotional effects are reported to be similar to patients diagnosed with asthma, arthritis, epilepsy, and diabetes.[4] Acne has substantial effects on quality of life, including social withdrawal, anxiety, and depression, making treatment important.[5,6]

The role of medical nutrition therapy (MNT) to help manage acne is not new. Early research reported an association between diet and acne, particularly chocolate, sugar, and fat.[7,8] Consequently, health care providers routinely restricted these foods as a part of acne treatment.[9] However, during the latter half of the 20th century, diet was not believed to play a role in acne development. This change occurred because of the results of two important research studies that are repeatedly cited in the literature and popular culture as evidence to refute the association between diet and acne.[10,11] More recently, dermatologists and registered dietitians have revisited the diet–acne relationship and become increasingly interested in the role of MNT in acne treatment. This reversal is based on several thought-provoking studies examining the role of diet and acne, and on relatively new evidence elucidating the influence of diet on the endocrine and immune responses involved in acne pathogenesis. Thus, the history of diet and acne can be categorized in three distinct phases: early history, the rise of the diet–acne myth, and recent research. The purpose of this review is to review these phases to evaluate the evidence for diet and acne.

We conducted a literature search between January 1, 2012, and July 1, 2012, using PubMed MeSH terms *Acne Vulgaris* and *Diet* or *Nutrition Therapy* or *Glycemic Index* or *Dairy Products* or *Milk* or *Fatty Acids, Omega-3* or *Dietary Fats*. The search was limited to human research published in the English language. Articles were excluded if the primary intervention included a vitamin, mineral, or herbal supplement. Research design was not a reason for exclusion, due to the high number of studies with methodologic limitations, small sample sizes, unclear or

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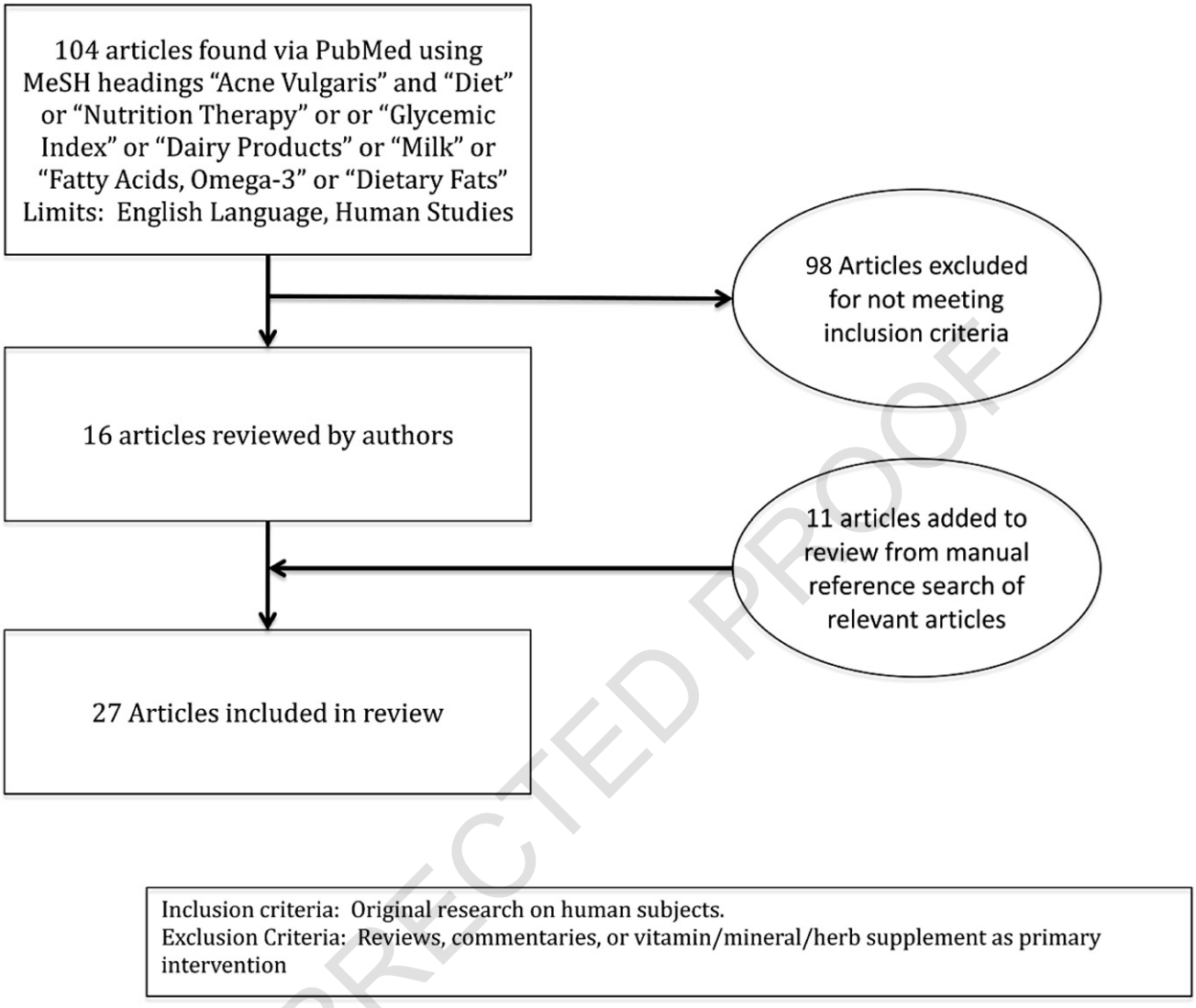

Figure 1. Flow diagram of literature review processes evaluating the relationship between diet and acne.

no statistical analysis, lack of a control group, or observational or anecdotal data. Figure 1 summarizes the major elements of this search, and the Table presents a summary of the literature.

DIET AND ACNE: EARLY HISTORY

Dermatology textbooks during the late 1800s and early 1900s frequently recommended dietary restriction as an adjunct treatment to existing dermatology therapy.[7,8] At this time, however, dermatologists did not fully understand the mechanisms underlying acne pathogenesis. Therefore, the diet–acne hypothesis and subsequent dietary recommendations were predominantly based on observation, anecdotal evidence, and speculation. In 1921, researchers observed chocolate increased blood lipid concentrations, and they surmised that chocolate similarly increased oil production by the sebaceous glands, augmenting acne severity.[12] In 1931, researchers reported patients with acne to have impaired glucose tolerance.[13] As a result, the researchers recommended patients avoid excessive carbohydrate consumption, including chocolate and sugar. This suggestion was further supported by later research demonstrating an improvement in acne severity among patients following a restricted carbohydrate diet.[14] This group of researchers speculated a disorder of carbohydrate metabolism increased acne. In 1949, an observational study reported an association between frequent milk consumption and acne severity and recommended patients with acne restrict high-fat dairy products.[15] In 1959, a small case study[16] demonstrated a decrease in acne severity among patients following a low-saturated–fat and low-total–fat diet. Taken together, these early studies generally reported an association between diet and acne.

Early Studies that Effectively Disassociated Diet and Acne

Although the diet–acne association was well established by the 1960s, many researchers disputed the association, reporting a lack of convincing evidence. In an attempt to fill the gap

Table. Studies investigating diet and acne, January 1, 1960 through July 1, 2012

Reference	Design	Participants	Intervention	Primary outcome	Results/conclusions	Covariates considered	Limitations
Abedamowo and colleagues (2005)[29]	Retrospective cohort	47,355 females, ages 25-42 y	FFQ[a]	Acne prevalence	Acne was positively associated with frequent consumption of total milk (PR[b] 1.22, *P* trend=0.002), whole milk (PR 1.12; *P* trend=0.56), low-fat milk (PR 1.16; *P* trend=0.25) and skim milk (PR 1.44; *P* trend=0.003) and negatively associated with consumption of saturated fat (PR 0.88; *P* trend=0.04)	Age at baseline and menarche, body mass index at age 18 y, energy intake	Self-reported acne, retrospective data collection, low clinical significance, and not adjusted for potential confounders such as heredity
Abedamowo and colleagues (2006)[30]	Prospective cohort	6,094 females, aged 9-15 y	FFQ	Acne prevalence	Acne was positively associated with frequent consumption of total milk (PR 1.2; *P* trend <0.001), whole milk (PR 1.19; *P* trend <0.001), low-fat milk (PR 1.17; *P* trend=0.002) and skim milk (PR 1.19; *P* trend <0.001)	Age, height, and energy intake	Self-reported acne, low clinical significance, and authors unable to distinguish trend between low-fat and whole milk
Abedamowo and colleagues (2008)[31]	Prospective cohort	4,273 males, ages 9-15 y	FFQ	Acne prevalence	Acne was positively associated with frequent consumption of total milk (PR 1.16; *P* trend=0.77) and skim milk (PR 1.19; *P* trend=0.02)	Age, height, and energy intake	Self-reported acne, low clinical relevance, and authors unable to distinguish trend between low-fat and whole milk
Anderson (1971)[11]	Case series	27 participants, age not reported	Consumption of chocolate, milk, peanuts, or cola	New acne lesions	No increases in acne lesions	NA[c]	Small sample size, unrandomized, unblinded, no baseline diet analysis, no statistical analysis, self-reported data collection, no control group, age not reported
Bett (1967)[20]	Cross-sectional	16 participants with acne, 16 healthy controls, age 15-27 y	Questionnaire on sugar consumption	Acne prevalence	No association between acne and sugar consumption	NA	Small sample size, questionnaire not validated, acne-scoring tool not disclosed
Cordain and colleagues (2002)[43]	Cross-sectional	1,200 Kitavan participants 115 Aché participants age 15-25 y	7-wk skin investigation	Acne prevalence	No acne observed in either population consuming non-Western diets	NA	Unable to account for confounders such as genetics and environmental factors
Cornbleet and Gigli (1960)[17]	Non-randomized clinical trial	(1) 15 patients with acne, age- and sex-matched controls (2) 52 participants with acne	(1) Oral glucose tolerance test (2) Sugar-restricted diet	(1) Glucose tolerance (2) Changes in acne	(1) No association between glucose tolerance and acne (2) No changes in acne on a sugar-restricted diet	NA	Short duration, small sample size, unrandomized, no baseline diet analysis, quantitative analysis not presented

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Table. Studies investigating diet and acne, January 1, 1960 through July 1, 2012 *(continued)*

Reference	Design	Participants	Intervention	Primary outcome	Results/conclusions	Covariates considered	Limitations
Di Landro and colleagues (2012)[32]	Case-control	205 participants with acne, 358 controls with no or mild acne, ages 10-24 y	FFQ	Acne prevalence	Acne was positively associated with frequent consumption of total milk (OR[d] 1.78) and skim milk (OR 2.2). Acne was negatively associated with frequent consumption of fish (OR 0.68)	Age, BMI[e], family history of acne	Inclusion of mild acne in control group, retrospective data collection, possible participant recall bias, limited generalizability to adults aged >24 y, FFQ not validated, type of fish not specified
Fulton and colleagues (1969)[10]	Crossover, subject-blinded, intervention	30 adolescents and 35 young male prisoners with acne, no ages specified	Chocolate bar or control bar	Acne severity	No change in acne severity during chocolate bar or placebo consumption	NA	Short duration, small sample size, no baseline diet analysis, quantitative analysis not presented, treatment and placebo were similar in caloric value, GI[f] and nutrient composition, inappropriate grouping (not age- or sex-matched)
Gaul (1965)[19]	Case report	4 participants with acne, ages 14-24 y	Low-sodium diet	Total acne lesions	Low-sodium diet was correlated with decrease in acne lesions	NA	Small sample size, unrandomized, no baseline diet analysis, quantitative analysis not presented, unblinded, confounding variables not considered
Ghodsi and colleagues (2009)[57]	Cross-sectional	1,002 participants, 793 participants with mild acne, 140 participants with moderate-severe acne, 68 participants without acne, ages 12-20 y	Questionnaire	Acne prevalence	Acne was positively associated with sweets ($P<0.0005$), nuts ($P<0.0005$), chocolate ($P=0.03$), and oily foods ($P=0.02$)	NA	Small sample size for control group, questionnaire not validated or published with manuscript, analysis not adjusted for possible confounders such as weight and energy intake; may not be generalizable to populations aged >20 y
Grant and Anderson (1965)[18]	Intervention	8 participants with acne	Milk chocolate bar in addition to usual dietary habits, half of participants consumed antacid before chocolate bar	Acne severity, total acne lesions	Chocolate did not aggravate acne. No significant effects with antacid consumption.	NA	Short duration, small sample size, unrandomized, no baseline diet analysis, quantitative analysis not presented unblinded, no control group
Halvorsen and colleagues (2009)[54]	Cross-sectional	3,775 participants, ages 18-19 y	Questionnaire on dietary habits	Acne prevalence	Acne was negatively associated with frequent vegetable consumption among females (OR 1.38)	Diet, lifestyle, and socioeconomic factors	Self-reported acne, FFQ not validated, results not significant after multivariate adjustment, may not be generalizable to populations outside the age range of 18-19 y

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Table. Studies investigating diet and acne, January 1, 1960 through July 1, 2012 *(continued)*

Reference	Design	Participants	Intervention	Primary outcome	Results/conclusions	Covariates considered	Limitations
Jung and colleagues (2010)[58]	Cross-sectional	1,285 participants, 783 participants with acne, 502 age-matched controls, mean age 24 y	Questionnaire on dietary habits	Acne prevalence	Acne was positively associated with instant noodles ($P=0.01$), junk food ($P=0.002$), carbonated drinks ($P=0.005$), processed cheese ($P=0.04$), braised pork ($P=0.02$), roasted pork ($P<0.001$), fried chicken ($P=0.001$), stewed chicken ($P=0.001$), nuts ($P=0.002$), and seaweed consumption ($P=0.003$). Among a subset of acne patients, acne was positively associated with roast pork ($P=0.02$), fried chicken ($P<0.02$), and nuts consumption ($P=0.03$).	NA	Short duration, results not controlled for confounding factors, FFQ not published with article, results may not be generalizable to Westernized diets
Kaymak and colleagues (2007)[47]	Prospective cohort	49 participants with acne, 42 healthy controls, ages 19-34 y	FFQ, blood biochemical markers	GI/GL[g], serum glucose, insulin sensitivity, IGF-1[h], IGFBP-3[i]	No significant differences in GI/GL, serum glucose concentrations, insulin sensitivity or IGF-1 among participants. Participants with acne had higher levels of IGFBP-3 concentrations ($P<0.001$) compared with controls	NA	Retrospective data collection, FFQ not validated nor included with article, some foods not included in the calculation of GL (dairy, fruits, vegetables, meats, fish), may not be generalizable to adults or obese population, different data in tables compared to text
Kim and colleagues (2010)[33]	Randomized, placebo-controlled intervention	36 participants with acne, ages 18-30 y	Fermented milk with 200 mg lactoferrin or placebo (fermented milk only)	Inflammatory and total lesion count	Participants consuming fermented milk with lactoferrin decreased total inflammatory lesion count ($P=0.019$) and total lesion count ($P=0.033$) compared with controls	NA	May not be applicable to obese population, not adjusted for possible confounding factors such as age of menarche
Kwon and colleagues (2012)[60]	Randomized controlled trial with blinded investigator	32 participants with mild to moderate acne, ages 20-27 y	Low GL diet or control diet	Number of inflammatory and noninflammatory acne lesions, histopathologic changes in acne lesions	Participants following a low GL diet decreased noninflammatory acne lesions ($P=0.02$), size of sebaceous glands ($P=0.03$), SREBP-1[j] ($P=0.03$) and IL-8[k] concentrations ($P=0.03$) after 10 wk. Participants following a low-GL diet decreased inflammatory acne lesions ($P=0.03$) after 5 wk	NA	Did not account for possible confounding factors, including consumption of dairy products, saturated fat, *trans* fat or fiber, self-reported dietary intake, failure to measure key hormonal factors, including IGF-1, IGFBP-3, and SHBG[l]
Law and colleagues (2009)[55]	Cross-sectional	322 participants, 82 participants with acne, 240 healthy controls, ages 17.4-20.8 y, mean age 19.1±1.7 y	Questionnaire on dietary habits	Acne prevalence	Acne was positively associated with dessert ($P=0.04$) and fruit juice ($P=0.02$) and negatively associated dairy and soy ($P=0.04$) among a subset of participants	NA	Short duration, small sample size for acne participants, FFQ was not extensive, may not be generalizable to Westernized diet or populations outside the age range of 17.4-20.8 y

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Table. Studies investigating diet and acne, January 1, 1960 through July 1, 2012 *(continued)*

Reference	Design	Participants	Intervention	Primary outcome	Results/conclusions	Covariates considered	Limitations
Reynolds and colleagues (2010)[59]	Randomized controlled trial	58 males with acne, mean age 16.5±1.0 y	Low-GL diet or high GL control	Acne severity, insulin sensitivity, androgen, SHBG, IGF-1, and IGFBP-3 concentrations	No significant differences in acne severity, insulin sensitivity, androgen, SHBG, IGF-1, or IGFBP-3 concentrations	Adjusted for change in dermatologist and photo/in-person acne grading method and differences in baseline diets	Short duration, high drop out rate, lack of randomization, nonvalidated acne grading methods, baseline diets not assessed, food diaries only completed on weekends, differences between GI/GL of groups may not have been large enough to affect glycemic and insulin responses in adolescents, may not be generalizable to women, non–boarding school students, or adults
Rouhani and colleagues (2009)[56]	Cross-sectional	2,528 participants following the South Beach diet, 90.4% female, 75.3% with acne	Internet South Beach Diet FFQ	Self-observed acne improvement	More than 86.7% of respondents reported an improvement in acne. Approximately 91% of respondents decreased the dose or number of acne medications	NA	Self-reported acne improvement, diet intake not assessed, non-randomized trial, FFQ not provided, total energy or weight loss not assessed, may not be generalizable to males, probable participant section and recall bias.
Rubin and colleagues (2008)[63]	Case study	5 participants with acne, 3 male and 2 female, ages 18-23 y	4 n-3 fatty acid supplements per day. Each supplement contains 250 mg eicosapentaenoic acid (EPA), 3.75 mg zinc gluconate, 50 μg selenium, 50 μg chromium, and 50 mg epigallocatechin gallate (EGCG) from green tea extract	Total and inflammatory lesion counts	4 participants had a decrease in total lesion counts All subjects had some reduction in inflammatory lesion counts	NA	Very small sample size, quantitative analysis not presented, dietary analysis not published with article
Smith and colleagues (2007)[50]	Randomized controlled trial with blinded investigator	43 males, ages 15-25 y	Low-GL diet or high GL control diet	Total inflammatory acne lesions, total lesion counts, insulin sensitivity	Participants following a low-GL diet had decreased inflammatory acne lesions ($P=0.02$), total acne lesions ($P=0.03$) and BMI ($P=0.001$), and increased insulin sensitivity ($P=0.026$) compared with controls	NA	Analysis not controlled for confounding factors such as fiber, fat or protein intake, dairy or weight loss, may not be generalizable to women, obese or populations outside the age range of 15-25 y
Smith and colleagues (2007)[51]	Randomized controlled trial with blinded investigator	43 males with acne, ages 15-25 y	Low-GL diet or high GL control diet	Total inflammatory acne lesions, total lesion counts, androgen concentration	Participants following a low-GL diet had decreased inflammatory acne lesions ($P=0.02$), total acne lesions ($P=0.01$), BMI ($P=0.002$), and androgen concentrations ($P=0.04$) compared with controls	Adjusted for differences in baseline values, age, ethnicity	Analysis not controlled for confounding factors such as fiber, fat, or protein intake, dairy or weight loss, may not be generalizable to women, obese, or populations outside the age range of 15-25 y

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Table. Studies investigating diet and acne, January 1, 1960 through July 1, 2012 *(continued)*

Reference	Design	Participants	Intervention	Primary outcome	Results/conclusions	Covariates considered	Limitations
Smith and colleagues (2008)[52]	Non-randomized clinical trial	12 males with acne, ages 15-20 y	7-d feeding trial in research facility, low-GL diet or high GL control diet	Insulin sensitivity, androgen, IGF-1, SHBG, and IGFBP-3 concentrations	Participants following a low-GL diet had increased insulin sensitivity ($P=0.03$) and IGFBP-3 ($P=0.03$), and decreased SHBG concentrations ($P=0.03$) compared with controls. No significant differences in IGF-1 or androgen concentrations	NA	Nonrandomized trial, self-reported acne history, changes in acne severity not reported due to short duration of trial, results may not be applicable to women, obese or populations outside the age range of 15-20 y
Smith and colleagues (2008)[53]	Randomized controlled trial with blinded investigator	31 males with acne, ages 15-25 y	Low-GL diet or high GL control diet	Ratio of saturated to monounsaturated fatty acids of skin surface triglycerides, total lesion counts, inflammatory acne lesions, sebum outflow	Participants following a low-GL diet had increased ratio of saturated to monounsaturated fatty acids of skin surface triglycerides ($P=0.007$) and decreased total lesion counts ($P=0.046$), and BMI ($P<0.001$) compared with controls. The increase in saturated/monounsaturated ratio correlated with total acne lesion counts ($P=0.03$). No significant difference in inflammatory acne lesions or sebum outflow	NA	Analysis not controlled for fiber, fat, or protein intake, or weight loss, may not be applicable to females, obese or populations outside the age range of 15-25 years.
Wei and colleagues (2010)[65]	Cross-sectional	5,696 participants, 2,920 participants with acne, ages 17-25 y	Questionnaire on dietary habits	Acne prevalence	Acne was positively associated with a high-fat diet (OR 1.439), fried food (OR 1.174), and spicy food (OR 1.146). Acne was negatively associated with frequent fruit consumption (OR 0.865)	NA	*P* value not provided for dietary analysis, nonvalidated questionnaire, results may not be generalizable to populations outside the age range of 17-25 y
Wu and colleagues (2007)[64]	Cross-sectional	3,163 children and adolescents, ages 10-18 y, 1,691 participants with acne	Questionnaire on dietary habits	Acne prevalence	No association between diet and acne	NA	FFQ not validated, results not adjusted for confounding factors, retrospective data collection, results may not be generalizable to populations aged >19 y

[a]FFQ=food frequency questionnaire.
[b]PR=prevalence ratio.
[c]NA=not applicable.
[d]OR=odds ratio.
[e]BMI=body mass index.
[f]GI=Glycemic Index.
[g]GL=glycemic load.
[h]IGF-1=insulin-like growth factor-1.
[i]IGFBP-3=insulin-like growth factor binding protein-3.
[j]SREBP=sterol regulatory element binding protein.
[k]IL-8=interleukin 8.
[l]SHBG=sex hormone binding globulin.

in the literature, researchers began to conduct actual intervention studies. The total number of studies conducted in the 1960-1970s was small; however, these studies are notable as they marked a turning point in the diet–acne history. In 1961, researchers examined the association between carbohydrate metabolism and acne.[17] The researchers reported normal glucose tolerance among acne patients and a restricted carbohydrate diet did not improve acne severity. Similarly, in 1965, researchers reported no changes in acne among participants consuming chocolate, in addition to usual dietary intake, after 1 week.[18] Conversely, in 1965, a different group of researchers demonstrated improvements in acne among participants consuming a low-sodium diet.[19] The researchers suggested sodium promotes inflammation, increasing acne severity. In 1967, other researchers found no differences in sugar consumption between patients with acne and healthy controls.[20]

In 1969, Fulton and colleagues[10] examined the relationship between chocolate and acne in a double-blind crossover study. This study is worthy of discussion, as it is one of the most frequently referenced experiments dispelling the association between diet and acne. Participants (n=65) with mild-to-moderate acne consumed a milk chocolate bar or placebo, daily, for 4 weeks. After a 3-week washout period, participants consumed the alternate treatment for an additional 4 weeks. Improvements in acne severity were based on decreases in total acne lesion counts. Based on this criterion, the researchers determined chocolate did not affect acne development. Although this group of researchers utilized a control group and conducted quantitative statistical analyses, this study was flawed for several reasons. For example, the intervention and placebo treatment were nearly identical in total energy, fat, and sugar. Furthermore, the methods used to score changes in acne severity lacked precision. The researchers categorized participants based on acne severity, which was determined by total lesion count. At the conclusion of the study, acne changes were based on increases or decreases in total lesion count of at least 30%. Using this methodology, a decrease in total lesion count of 29% would be considered unaffected by food, regardless of changes in acne severity and/or inflammatory lesions.[21]

In 1971, Anderson and colleagues[11] further researched the diet–acne relationship. Similar to the research presented by Fulton and colleagues[10] this research study is important to discuss in detail because it is repeatedly cited as evidence to refute the diet–acne hypothesis. Researchers examined the dietary habits of university students (n=27) with self-reported dietary acne triggers and self-perceived acne. Participants were subdivided into small groups of unknown size and instructed to consume a large portion of chocolate, milk, roasted peanuts, or carbonated beverage, daily, in addition to usual dietary patterns. The researchers mapped acne lesions onto a sheet of paper before and after the study treatment. After only 1 week, the participants did not exhibit any new flares of acne, leading to the conclusion that diet does not influence acne development. Similar to the research presented by Fulton and colleagues,[10] this study had several methodologic errors. The sample size was small (n=27) and participants were subdivided into smaller groups of unknown sizes, making it unlikely that the study had adequate statistical power. In addition, the researchers did not utilize a control group, describe the methods for grading acne, use quantitative statistical analyses, randomize participants, specify the age range of participants, or analyze baseline dietary habits. Consequently, the treatment may not have varied from the participants' usual diet. Furthermore, the researchers were not blinded; therefore, the study results were subject to possible bias.

Although the studies conducted in the 1960s and 1970s primarily investigated the effects of chocolate on acne, the results erroneously led to the general consensus that diet was not associated with acne. Unfortunately, this statement is problematic. These studies had critical flaws in research design, making debatable the conclusion that diet and acne are unrelated. More importantly, these studies were designed before the establishment of the Glycemic Index (GI) and glycemic load (GL) and before dermatologists fully understood the role of endocrine mechanisms in acne pathogenesis or the duration of time necessary for a treatment to influence acne development.[22,23] Despite flaws, diet and acne were not further investigated for nearly 40 years. The rediscovery of the diet–acne association was due to a variety of factors, including advances in our understanding of acne pathogenesis, new epidemiologic evidence supporting diet and acne, and a thorough critical analysis of early studies.

ACNE PATHOGENESIS

Acne pathogenesis is related to several key factors: excess sebum production by the sebaceous glands, follicular occlusion, hyperproliferation of *Propionobacterium acnes (P. acnes)* bacteria, and inflammation.[24] Excess sebum production and hyperproliferation of follicular cells contribute to follicular occlusion and comodone formation.[25] Follicular occlusion creates a sebum rich, oxygen-poor environment, ideal for the proliferation of *P. acnes*. Immune recognition of *P. acnes*, as well as other factors, initiates an immune response causing inflammation.[25] Androgen hormones and insulin-like growth factor-1 (IGF-1) influence sebum production and are implicated in the development of acne. Additional compounds, including insulin, sex hormone binding protein (SHBP), sterol regulatory element binding protein-1 (SREBP-1), and inflammatory mediators are also associated with the development of acne.[1,26,27] These factors are also associated with diet and may provide the link between diet and acne. This hypothesis is further supported by evidence that deficiencies in hormones, such as IGF-1, are associated with decreased acne severity.[28] The understanding of these physiologic mechanisms has contributed to a renewed interest in the diet–acne hypothesis.

MODERN STUDIES INVESTIGATING DIET AND ACNE

Diet and Dairy

To date, three large studies, all conducted by Abedamowo and colleagues,[29-31] have examined the relationship between frequent dairy consumption and acne. The first study was a retrospective cohort investigating frequent dairy consumption and teenage acne. The researchers asked adult female nurses (n=47,355) to recall their usual dietary intakes during high school using a food frequency questionnaire (FFQ) and if a physician had ever diagnosed them with acne.[29] After adjusting for confounders, acne prevalence was associated with to-

tal milk consumption (prevalence ratio [PR] 1.22; *P* for trend=0.002). The association was stronger with skim milk (PR 1.44; *P* for trend=0.03) compared with low-fat milk (PR 1.16; *P* for trend=0.25) and whole milk (PR 1.12; *P* for trend=0.56). The most notable limitation of this study was the retrospective self-reported subjective data compilation. Specifically, the FFQ collected information on high school eating habits, which occurred >10 years before the study. In addition, the data were not adjusted for some important confounding variables and may not be generalizable to adults.

In 2006 and 2008 Abedamowo and colleagues[30,31] re-examined the relationship of dairy and acne in two prospective studies among adolescents aged 9 to 15 years (n=6,094 girls and 4,273 boys) using a validated FFQ. After adjusting for confounding variables among female participants, self-reported acne was positively associated with consumption of total milk (PR 1.2; *P* for trend <0.001), skim milk (PR 1.08; *P* for trend<0.001), low-fat milk (PR 1.17; *P* for trend=0.002), and whole milk (PR 1.19; *P* for trend <0.001).[30] Among male participants, self-reported acne was positively associated with consumption of total milk (PR 1.16; *P* for trend < 0.77) and skim milk (1.19; *P* for trend=0.02).[31] Limitations of these studies include self-reported acne and dietary habits. The researchers were unable to distinguish a trend between low-fat and whole milk consumption and acne prevalence. In addition, the same research group conducted all three of these large prospective studies and the PRs were close to 1, making it difficult to determine the clinical relevance of the results.

To further examine the acne-promoting role of milk, Di Landro and colleagues[32] conducted a case-control study in adolescents and young adults (aged 10 to 24 years). Participants with moderate to severe acne (n=205) were case-matched to participants with no or mild acne (n=358) and dietary habits were analyzed using a nonvalidated FFQ. After adjusting for confounding variables, acne was positively associated with frequent consumption of total milk (odds ratio [OR] 1.78) and skim milk (OR 2.2), but not whole milk or cheese. Although this research supports the results presented by Abedamowo and colleagues, several limitations warrant concern, including the use of a nonvalidated FFQ, self-reported dietary intake, inclusion of participants with mild acne into the control cases, and a retrospective study design. To date, no randomized controlled studies investigating the association between dairy and acne exist.

Recent research, conducted by a different group of investigators, presents evidence that lactoferrin-enriched fermented milk consumption may have a benefit on acne.[33] Participants (n=36, aged 18 to 30 years) were randomly assigned to consume fermented milk with 200 mg lactoferrin or placebo. After 12 weeks of consumption of lactoferrin-enriched fermented milk, participants with acne demonstrated an improvement in acne severity, total lesion count, and sebum composition. The researchers concluded lactoferrin-enriched fermented milk decreases acne severity due to the anti-inflammatory effects of lactoferrin and its ability to suppress microbial growth.[34-36] Although most evidence suggests total milk consumption as the most critical dairy component promoting acne, currently, there is insufficient evidence to recommend milk restriction as a treatment for patients with acne. However, these preliminary results may provide a potential milk alternative or adjunct therapy for a subgroup of patients with acne in which a restricted milk diet is appropriate.

GI/GL and Acne

While some researchers were investigating the association between dairy and acne, others were examining the relationship between the quality and quantity of carbohydrate consumption and acne, primarily based on epidemiologic evidence reporting low acne prevalence among populations living in rural villages.[37-39] Traditionally, Canadian Inuits, pre-World War II Okinawans, and Zulu populations do not have acne; however, acne prevalence increased among the Canadian Inuits after acculturation with neighboring countries and the adoption of processed foods, beef, and dairy.[40] Similarly, acne prevalence increased among pre-World War II Okinawans after increasing consumption of animal products.[41] Among the Zulu population, increases in acne prevalence are attributed to the migration into cities from rural villages.[42]

To explain this phenomenon, Cordain and colleagues[43] examined the relationship between carbohydrate consumption and acne. This study initiated an interest in the potential role of a high GI and/or GL diet in acne pathogenesis among dermatologists. The GI is a system of measuring the effect of carbohydrate on blood glucose[44,45] whereas GL combines the quantity and effect of the carbohydrate on blood glucose. A high-GI or -GL food increases hyperinsulinemia.[46] Therefore, researchers speculated a high-GL diet might increase acne by triggering a hormonal cascade.

Cordain and colleagues[43] examined acne prevalence among the Kitavan Islanders of Papua New Guinea (n=1,200) and the Aché hunter-gatherers of Paraguay (n=115) and did not find any cases of acne after a skin examination. The researchers speculated the absence of acne was due to diet. The Kitavan Islanders and Aché consume a substantially lower GL diet compared with Westernized nations. Unfortunately, the researchers did not utilize a control group, making it difficult to determine whether the absence of acne was due to a low-GL diet, genetics, or other environmental factors.

This hypothesis was challenged by a cross-sectional study in 2007.[47] This group of researchers investigated the role of a high-GL diet on acne severity among university students aged 19 to 34 years with (n=49) and without acne (n=42). The researchers measured fasting glucose, insulin, IGF-1, insulin-like growth factor binding protein (IGFBP)-3, and GL. There were no significant differences in glucose, insulin, IGF-1, or GL in participants with or without acne. Furthermore, none of the participants demonstrated insulin resistance. Therefore, the researchers concluded hyperinsulinemia is not involved in acne pathogenesis. Limitations of the study include a retrospective data collection and a nonvalidated FFQ, which was not published with the article. In addition, the researchers did not calculate the GL for meat, poultry, fish, vegetables, cheese, or dairy. In small quantities, these foods minimally affect the overall GL; however, large amounts may significantly influence postprandial blood glucose response. In particular, dairy causes hyperinsulinemia and has a high GL, representing an important confounder of the study design.[48,49]

In 2007-2008 Smith and colleagues[50-53] conducted three studies and published four articles examining the association between GL and acne. In the first study, the researchers randomly assigned participants (43 men ages 15 to 25 years) to a

low-GL diet or high-GL diet for 12 weeks.[50,51] The researchers used the same outcome data for two publications; however, in the second publication, the researchers adjusted the data for differences in baseline values, age, and ethnicity and analyzed additional blood biochemical markers. In the first publication, participants following a low-GL diet decreased total and inflammatory acne lesions (P=0.03 and 0.02, respectively), lost more weight (P<0.001), decreased body mass index (BMI) (P=0.001), and showed a greater improvement in insulin sensitivity (P=0.026) compared with participants following a high-GL diet.[50] In the second publication, participants following a low-GL diet decreased total and inflammatory acne lesions (P=0.01 and 0.02, respectively), lost more weight (P<0.001), decreased BMI (P=0.002), decreased androgen concentrations (P=0.04), and showed a greater improvement in insulin sensitivity (P=0.02) compared with participants following a high-GL diet.[51] On this basis, the researchers concluded that the amount and type of carbohydrate plays a role in acne pathogenesis.

In the second study, the researchers conducted a nonrandomized, parallel, prospective controlled feeding study to analyze the association between GL and acne.[52] In this small study, 12 men (aged 15 to 20 years) with acne consumed either a low-GL diet or a high-GL diet. After 1 week, participants following a low-GL diet had lower androgen concentrations (P=0.04) and showed greater improvements in insulin sensitivity (P=0.03) compared with participants following a high-GL diet. In addition, participants following a low-GL diet increased IGFBP-3 concentrations from baseline (P=0.03). The researchers concluded a low-GL diet increases IGFBPs and reduces IGF activity, decreasing circulating androgen concentrations, and subsequently, acne development.

In the third study, the researchers continued to investigate the association between GL and acne; however, in this study the researchers did not examine the effects of GL on hormone levels.[53] Instead, they investigated the association between GL and sebum composition. Participants (31 men aged 15 to 20 years) were randomly assigned to a low-GL diet or a control diet (high-carbohydrate diet without reference to GL). After 12 weeks, the researchers measured facial follicular sebum outflow and the composition of skin surface triglycerides. Among participants on the low-GL diet, the ratio of saturated to monounsaturated fatty acids increased, which negatively correlated with total acne lesion counts (r=−0.39; P=0.03) and follicular sebum outflow (r=−0.49; P=0.006). These findings suggest a low-GL diet may alter factors associated with acne development, such as sebum composition and output.

Together, the four publications by Smith and colleagues[50-53] constitute the most convincing evidence to date in support of a relationship between dietary GL and acne. These results are particularly compelling because the researchers used strong study designs, including provision of staple foods, dietary counseling, regular telephone sessions, urine samples, controlled feeding environments, and food records. In addition, all but one study utilized randomized grouping. Despite their strengths, these research articles did have several limitations and the results should be interpreted with caution. The results of these publications cannot be generalized to women or anyone outside the age range of 15 to 25 years. In three of the articles, participants following the low-GL diet lost significantly more weight and consumed a diet lower in fat and higher in dietary fiber compared with participants following the high-GL diet. The researchers were unable to account for these differences; therefore, the decrease in acne cannot be solely attributed to a change in dietary GL. In both articles, after the researchers adjusted for weight loss, changes in insulin resistance and total lesion counts were no longer significant. In addition, the third publication included a small sample size, did not randomize participants, and the duration was not long enough to measure changes in acne severity. Instead, the researchers measured changes in markers of insulin resistance, speculating the changes in insulin sensitivity might inhibit factors associated with acne development in a study with a longer duration.

After the work of Smith and colleagues, five cross-sectional studies, published by various researchers, further examined the relationship between dietary GI/GL and acne between 2009-2010.[54-59] The first study primarily investigated the association between mental distress, diet, and self-reported acne among Norwegian adolescents aged 18 to 19 years (n=3,775).[54] After adjusting for confounding variables, acne was positively associated with infrequent vegetable consumption (OR 1.38) among female participants. However, the researchers did not present the quantitative analysis and the strength of the association is not known. The second cross-sectional study, published in 2009, examined the relationship between diet and acne using traditional Chinese medicine in 322 participants aged 17.4 to 20.8 years.[55] The researchers assessed diet by measuring the frequency of consumption of 11 categories of food over a 1-week period using a validated questionnaire. When participants were examined homogenously, there were no significant associations. Among a subset of participants, frequent consumption of dessert (P=0.04) and fruit juice (P=0.02) was positively associated with acne, whereas dairy and soy (P=0.04) were negatively associated with acne. Although these studies were not designed to measure the association between dietary GI/GL and acne severity, the dietary patterns in both studies are characteristic of a high-GI/GL diet, suggesting high-GI/GL foods increase acne severity among a subset of participants.

The third cross-sectional study, investigated the relationship of the South Beach Diet, which is considered a low-GI diet, and acne using an Internet-based survey.[56] Self-proclaimed active South Beach "dieters" (90.4% women, N=2,528) reported changes in self-perceived acne severity. While following the South Beach Diet, more than 85% of respondents reported an improvement in acne and approximately 90% reported decreasing the dose or number of acne medications. Although the results of this study suggest an association between diet and acne, the study suffers from several methodologic issues, making it difficult to determine conclusions. Limitations include self-reported acne improvement and participant selection and recall bias due to the retrospective study design. Moreover, the participants were actively attempting to lose weight; therefore, it is possible weight loss may have contributed to improvements in acne severity.

The final cross-sectional studies published in 2009, which had 1,002 participants aged 12 to 20 years, and 2010, which had 1,285 participants aged 12 to 20 years, investigated a variety of dietary factors and acne.[57,58] In the fourth study, the

researchers found an association between acne and select food items, including chocolate ($P=0.03$), sweet foods ($P<0.005$), and infrequent consumption of vegetables ($P=0.01$).[57] Similar to previous studies, these foods are not unusual in a high-GI/GL diet, suggesting an association between dietary GI/GL and acne. In the last cross-sectional study, the researchers measured frequent consumption of select Korean foods among Korean participants (mean age 24 years) with and without acne.[58] IGF-1, IGFBP-3 concentrations, and postprandial blood glucose were measured only in participants with acne. Among acne participants, frequent consumption of selected high-GL foods, dairy and high fat foods were significantly higher ($P<0.05$). Among a subset of acne patients, IGF-1 concentrations were significantly higher ($P<0.05$). Collectively, the researchers concluded specific dietary habits, including dietary GL and insulin resistance, might play a role in acne pathogenesis. Although both of these studies support an association between a high-GI/GL diet and acne, these study designs are far from ideal and contain limitations, including the use of a nonvalidated FFQ, self-reported acne severity, failure to assess baseline diet, probable selection bias, limited generalizability, and failure to adjust for important confounding variables. Furthermore, these studies were not originally designed to investigate the relationship of a high-GI/GL diet and acne, making it difficult to use them as evidence for MNT guidelines.

In 2010, Reynolds and colleaguues[59] continued to examine the relationship between dietary GL and acne. This randomized controlled trial was designed to limit the confounding factors presented by Smith and colleagues[50-53] Participants (58 men with a mean age 16.5±1 years) were alternately assigned to a high- or low-GL diet. After 8 weeks, facial acne improved more among participants following the low-GL diet. However, the differences did not reach statistical significance. There were no differences in insulin sensitivity between participants following a high- or low-GL diet. Although the researchers successfully maintained weight and eliminated macronutrient differences between the intervention and control group, this study also suffered from serious limitations, including a short duration, high dropout rate, limited generalizability, failure to account for baseline diet, small sample size, and a nonrandomized design. Furthermore, the researchers only required participants to complete food records on weekends, and the difference in total GL scores between participants following a high- and low-GL diet may not have been great enough to adequately affect insulin response. The researchers concluded a low-GL diet does not significantly improve acne severity; however, they acknowledged the limitations of the study make it difficult to determine conclusions.

In 2012, Kwon and colleagues[60] published results from a blinded, randomized controlled trial examining the association between a low-GL diet and acne. The researchers hoped to clarify the conflicting evidence presented by Smith and colleagues and Reynolds and colleagues and to include histologic examination of acne lesions before and after dietary education. Participants (N=32) aged 20 to 27 years were randomized to follow either a low-GL diet or control group diet (emphasizing carbohydrate-rich foods). After 5 weeks, participants following the low-GL diet decreased inflammatory lesions ($P=0.03$). After 10 weeks, participants following the low-GL diet decreased noninflammatory lesions ($P=0.02$), size of sebaceous glands ($P=0.03$), and expression of sterol regulatory element-binding protein (SREBP)-1 ($P=0.03$). These findings are significant because it presents the first evidence that a low-GL diet may reduce the expression of SREBP-1, decreasing acne severity while also supporting the breakthrough research presented by Smith and colleagues. Unlike the research by Smith and colleagues, there were no significant changes in BMI among participants following the low-GL or control diet. However, the authors do acknowledge the lack of weight loss may be due to the short duration of the study and further research is needed to examine long-term relationship of diet and acne. Other limitations include self-reported dietary intake, small sample size, and failure to adjust for possible acne promoting confounding factors, including dairy, saturated fat, *trans* fat, and fiber consumption. In addition, the researchers did not measure key hormone factors, including IGF-1, SHBG, and IGFBP-3.

Dietary Fat and Acne Severity

As discussed previously, the work of Cordain and colleagues[43] suggested low-GL diets decrease hyperinsulinemia, thus decreasing acne severity. This hypothesis is supported by observational data demonstrating low acne prevalence among the Kitavan and Aché populations. Although these populations traditionally consume a low-GL diet, they also consume a diet low in processed foods, dairy, and total fat and high in fruits, vegetables, and fish. The estimated n-3:n-6 ratio of traditional hunter–gather diets, such as the Kitavan and Aché populations, is 1:1,[61] whereas the ratio among Westernized nations is approximately 1:20.[62] Thus, the low prevalence of acne among these populations could be due to several other dietary components, including the high consumption of n-3 fatty acids or total fat. Unfortunately, very limited data support this theory. To date, only one case study, one case-control study, and two cross-sectional studies have investigated the association between dietary fat and acne.[32,63-65]

In 2008, a case study (5 participants aged 18 to 23 years) investigated the effects of an n-3 fatty acid supplement containing eicosapentaenoic acid and antioxidants on acne development.[63] After 8 weeks, the researchers observed a decrease in acne development. Limitations of this case study include a small sample size, short duration and lack of a control group. Furthermore, the results of this case study are observational and cannot be used to determine causation.

As previously discussed, in 2012, Di Landro and colleagues[32] conducted a case-control study investigating the association between various dietary factors and acne using a FFQ. The researchers primarily investigated the association between frequent consumption of dairy and acne; however, they also examined the relationship between frequent fish consumption and acne. After adjusting for confounding factors, the researchers determined fish consumption was negatively associated with acne severity (OR 0.68), indicating frequent consumption of n-3 fatty acids have a protective effect on acne. In addition to the limitations previously discussed, the researchers did not indicate the type of fish consumed, making it difficult to determine whether the total amount of n-3 fatty acids or frequent consumption of fish decreased acne prevalence. The study does not provide information on the quantity of fish necessary to decrease acne.

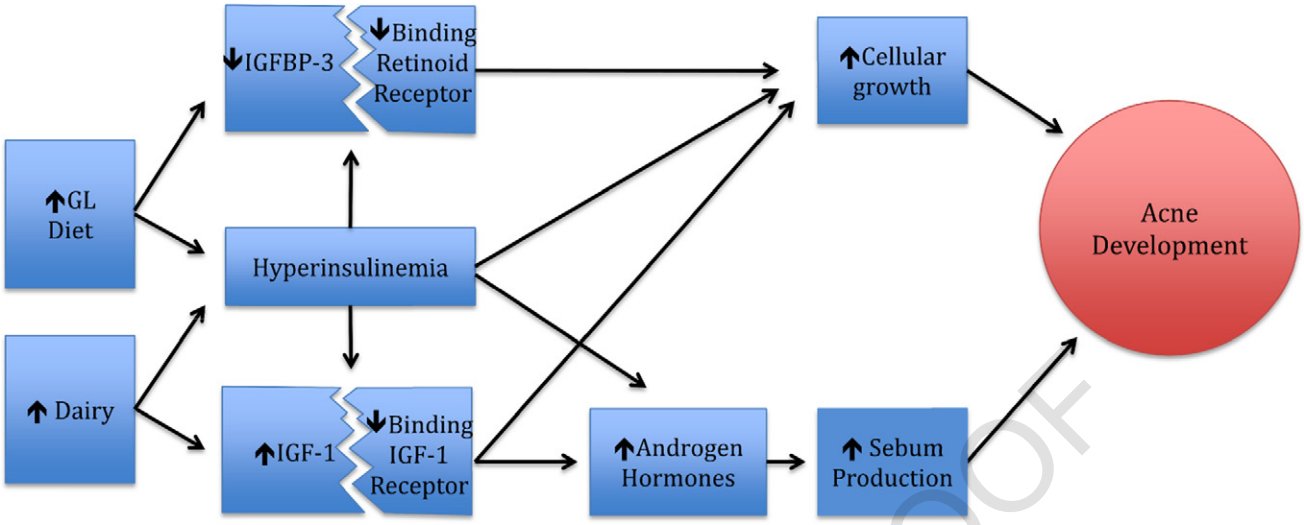

Figure 2. Current research suggests diet influences acne development. Glycemic load (GL) and Dairy ingestion lead to changes in circulating hormones, binding proteins, and receptors, leading to increased cellular growth and sebum production and influencing acne development. IGFBP-3=insulin growth factor binding protein 3. IGF-1=insulin growth factor 1. SHBG=sex hormone binding globulin.

The cross-sectional studies examining the relationship between dietary fat and acne were published in 2007 (3,163 participants aged 10 to 18 years) and 2010 (5,696 participants aged 17 to 25 years).[64,65] These studies were not specifically designed to measure the association between acne and dietary fat. Instead, they examined the relationship of acne and a variety of dietary factors, including dietary fat. In the first study, the researchers did not find an association between a high-fat diet nor frequent seafood consumption, suggesting total fat and n-3 fatty acids are not associated with acne development.[64] Conversely, the researchers of the second study found an association between acne and a high-fat diet (OR 1.439; $P<0.05$) and frequent intake of fried food (OR 1.174; $P<0.05$).[65] Limitations include the use of a nonvalidated questionnaire, limited generalizability, and failure to account for potential confounding factors.

DISCUSSION

Based on the current literature, a high-GI/GL diet and frequent dairy consumption are the leading factors in establishing the link between diet and acne, as shown in Figure 2. A high-GL diet increases hyperinsulinemia, which elicits an endocrine response that simultaneously stimulates IGF-1 while suppressing IGFBP-3.[66,67] IGF-1 is a powerful mediator of cellular growth, including unregulated tissue and follicular growth, and amplifies androgen bioavailability. Androgen hormones have multiple effects, including the promotion of sebum production and secretion, which is a well-established factor in acne pathogenesis.[68,69] IGF-1 additionally stimulates sebum production by increasing the expression of SREBP-1, which may stimulate additional lipogenesis in sebocytes via the activation of the phosphoinositide 3-kinase/Akt pathway.[27,70]

Normally, IGFBP-3 and SHBP function as inhibitory molecules by binding IGF-1 and androgen hormones, respectively. Insulin induced suppression of IGFBP-3 and SHBP results in an increase in available IGF-1 and androgen hormones, augmenting acne development.[71] This hypothesis is further supported by evidence that deficiencies in hormones, such as IGF-1, are associated with decreased acne severity[72] and some IGF-1 polymorphisms are associated with increased circulating IGF-1 concentrations and acne severity.[73] Furthermore, hormone management, including medications to reduce insulin secretion, has shown to be effective in treating acne.[74]

Hyperinsulinemia-mediated reduction of IGFBP-3 further induces acne development by increasing follicular growth through the nuclear retinoid-signaling pathway. Retinoids are a class of chemical compounds related to vitamin A that inhibit cellular proliferation and encourage apoptosis via binding of retinoic receptors.[75,76] Specifically, IGFBP-3 is a ligand for the retinoid X receptor-alpha and binding leads to decreased cellular growth.[77] Consequently, decreased IGFBP-3 bioavailability decreases the activity of the retinoid X receptor-alpha, increasing cellular growth and enhancing acne development.

Dairy is linked to increased acne severity through similar pathways. Dairy products contain carbohydrates and are hypothesized to increase acne severity through diet-induced hyperinsulinemia, which stimulates increased IGF-1 concentrations. Both skim and whole milk, but not cheese products, have a three- to six-fold higher GL and insulinotrophic response than predicted, based on the carbohydrate content of the milk.[78] This response suggests total milk consumption or total milk protein may have a greater influence on acne, compared with other carbohydrate foods.[79] In addition to the dairy-induced hormonal response, milk contains a magnitude of growth-stimulating hormones, including IGF-1, and concentrations remain high even after pasteurization, homogenization, and digestion.[70] Bovine IGF-1 is identical to human IGF-1 and both are able to bind to the human IGF receptor.[80,81] Interestingly, the association is stronger in skim milk, compared with high-fat milk, implying acne is unlikely to be influenced by the fat content in milk. The increased comedogenicity demonstrated in skim milk may be due to other factors

within milk, including milk proteins. Whey and casein are the main milk proteins and exhibit different growth-promoting effects. Whey protein is a potent inducer of postprandial hyperinsulinemia, whereas casein increases IGF-1 concentrations.[82,83] Although no randomized controlled trials have examined the relationship between dairy, milk, or milk protein on cellular signaling, one theory suggests leucine-rich whey protein increases acne by inducing cellular growth, androgen hormone secretion, and sebaceous lipogenesis possibly mediated by the mammalian target of rapamycin complex 1 and related pathways.[84] In addition, whey protein concentrates, commonly found in popular sports supplements, may influence acne severity.[85]

Although evidence is limited, n-3 fatty acids are hypothesized to reduce acne severity by suppressing inflammatory cytokine and leukotriene production.[86] Leukotrienes are primarily synthesized from the 5-lipooxygenase pathways and are associated with increased markers of inflammation and acne severity.[87] Suppression of leukotriene B4 concentrations after administration of a 5-lipoxygenase inhibitor decreases inflammatory acne, suggesting a therapeutic role for n-3 fatty acids among acne patients.[88] In addition, n-3 fatty acids may decrease acne by decreasing insulin[89] and IGF-1 concentrations,[90] and increasing IGFBP-3 concentrations,[91] demonstrating similar effects on acne development as GI/GL and dairy consumption.

CONCLUSIONS

The past decade has experienced tremendous growth in research investigating the acne promoting or aggravating effects of diet. Taken together, epidemiologic, observational, and experimental evidence suggests an association between diet and acne. This evidence, to date, does not demonstrate that diet causes acne, but may aggravate or influence it to some degree. However, a number of questions and concerns must be answered before the efficacy and clinical relevance of diet therapy is fully understood and evidence-based medical nutrition guidelines can be established.

The research supporting an association between dairy and acne is more consistent compared with the research presented on dietary GL, dietary fat, or n-3 fatty acids. Despite this, the same group of researchers conducted the majority of the studies on dairy and acne and the data are observational. Therefore, these studies are unable to determine causation or the quantity of milk necessary to exacerbate acne. Currently, researchers are not certain if the association between dairy and acne is independently or synergistically due to the hormones in milk, milk protein, or the effect of milk on insulin and IGF-1 concentrations.

Compared with other dietary factors, a greater number of studies examine the association between dietary GL and acne. However, limitations and methodologic issues make it difficult to compare studies, conduct meta-analyses, or establish firm conclusions. Many of the intervention studies were conducted by the same research group, had limited generalizability, and did not control for potentially important confounding variables. Other studies were not specifically designed to evaluate dietary GL and acne. In addition, research has not yet defined the cut points for a high or low GL score necessary to influence acne development. Furthermore, no research has yet examined the influence of a low-GL and low-dairy diet on acne development. The role of dietary fat and/or n-3 fatty acids also remains unknown. To date, the number of research studies examining the relationship between dietary fat and/or n-3 fatty acids is sparse and the evidence is weak.

MNT may be a reasonable option for a subset of patients with acne to consider, as an adjunct to dermatology therapy. A low-GL diet is a healthy dietary intervention, typically low in saturated fat and high in whole grains, fruit, and vegetables. The health benefits of a low-GL diet may have multiple benefits beyond acne, including weight loss and decreasing risk of obesity, cancer, and diabetes.[92-94] Similarly, n-3 fatty acids are associated with health benefits including prevention of cancer and cardiovascular disease and treatment of a variety of mental illnesses.[95] In addition, a diet lower in dairy, if sufficient in calcium and vitamin D, may be considered adequate.

These gaps in the literature should not intimidate but challenge dermatologists and registered dietitians to work collaboratively to design and conduct quality research. This research is necessary to fully elucidate preliminary results, determine the proposed underlying mechanisms linking diet and acne, and develop potential dietary interventions for acne treatment. Specifically, randomized controlled trials are necessary to investigate the therapeutic potential of nutrition education, dairy, dietary GL, and n-3 fatty acid supplementation or consumption. Furthermore, future studies should consider potential confounding variables, including various acne medications, race, sex, previous acne treatment, age, age at menarche, baseline dietary analysis, and past medical history.[96] In addition, acne quality of life should be measured before and after MNT treatment. Although these studies are necessary before comprehensive evidence-based MNT recommendations can be established, preliminary evidence regarding diet and acne is certainly worth mentioning. The medical community should not dismiss the possibility of diet therapy as an adjunct treatment for acne. At this time, the best approach is to address each acne patient individually, carefully considering the possibility of dietary counseling.

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STATEMENT OF POTENTIAL CONFLICT OF INTEREST

No potential conflict of interest was reported by the authors.

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