Review

Diet and acne: a review of the evidence

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Introduction

Acne vulgaris is the most common dermatologic condition in the USA, affecting more than 17 million Americans of all ages, although it is especially common in adolescents.[1] Moreover, approximately 80–90% of American adolescents experience acne.[2] Severe acne is associated with low self-esteem, poor body image, social withdrawal, and depression.[3] Pharmaceutical acne treatments are costly and have potentially severe side-effects.

Adolescent acne is typically the result of clogged, infected, pilosebaceous follicles. Adults may experience fewer comedones and more inflammatory lesions.[1] Normally, sebum travels up the follicle to the skin surface. Hormones may increase sebum production and cause follicular cells to hyperproliferate and block the follicular opening, forming a comedo.[4] Complete follicle blockage results in closed comedones (i.e. "whiteheads"), whereas incomplete blockage results in open comedones ("blackheads"). Comedo formation typically occurs over the course of 2–3 weeks.

Acne may manifest in the form of noninflammatory comedones, superficial inflammatory lesions (papules, pustules), and/or deeper inflammatory lesions (nodules, cysts). Inflammatory lesion formation occurs most commonly when *Propionibacterium acnes* colonizes the pilosebaceous unit, triggering follicular rupture and a neutrophil cascade.[5] Rarely, acne may have nonbacterial causes.[6]

In studies of diverse populations, individuals with acne commonly attribute the condition[3,7–9] or its exacerbation[3,7,8,10] to diet. Chocolate and oily or fatty foods are commonly implicated;[7,10–12] however, reviews prior to 2007 have concluded that diet plays no important role in acne and that the condition is primarily attributable to genetic predisposition and hormonal influences.[13–15]

Two large twin studies[16,17] have reported on the heritability of acne. Estimated heritability (genetic variance/phenotypic variance) ratios for acne risk and severity range from 0.5 to 0.9 among adolescent[16] and adult[17] pairs of monozygous and dizygous twins. Walton *et al.*[18] reported that sebum excretion is influenced by genetic factors, but that the development of clinical disease is mediated by environmental factors. These studies suggest that genetic factors alone do not fully account for the acne risk. Despite the genetic regulation of sebum excretion and other determinants of acne, environmental influences, such as diet, may act as modifiers of gene expression. Recently, well-designed, controlled, prospective studies have supported the association between specific dietary factors and acne. We therefore critically examined the quality and strength of the published literature examining the association between diet and acne.

Methods

We conducted a review of the relationship between diet and acne using the following keywords: "acne," "acne vulgaris," "diet," "nutrition," "food," "food allergy," "vitamin," and "chocolate." The following databases and periods were included: Medline since 1949; Embase-Medicine & Embase-Psychology (EMBASE) since 1980; the Cochrane Central Register of Controlled Trials (Cochrane) since 1898; Database of Abstracts on Reviews and Effectiveness (DARE) since 1990; PsycInfo since 1967; and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) since 1982. Articles were also obtained by bibliography review. Articles published in languages other than English were reviewed if English translations were available. Observational and interventional human studies with participants of any age, sex, or health status were included. Articles were excluded if they reported only on the potential effects of topical, herbal, or vitamin

preparations, or on the diagnosis, treatment, or pathogenesis of acne. A small sample size, a lack of a control group, and unclear statistical methods were not reasons for exclusion. The primary author (EHS) reviewed the titles and abstracts of all potentially relevant articles to determine whether they met the eligibility criteria.

Results

Of the 59 abstracts and full articles retrieved, 31 articles were excluded for reporting only on the potential effects of topical, herbal, or vitamin preparations, or on the diagnosis, treatment, or pathogenesis of acne. The remaining 28 articles were included. We excluded one study that did not report on diet. Of the 27 relevant articles, 21 were observational studies and six were clinical trials (Table 1).

Observational studies

We found 15 cross-sectional,[3,7–12,19–26] two case–control,[27,28] and four prospective cohort[29–32] studies, as summarized in Table 1.

Cross-sectional studies

Population-based studies suggest that acne prevalence is lower in rural societies than in industrialized populations. Cordain *et al.*[19] studied the Kitavan islanders of Papua New Guinea (n = 1200) and the Aché hunter-gatherers of Paraguay (n = 115). The islanders subsisted mainly on root vegetables, fruit, fish, and coconut. Their intake of dairy products, coffee, alcohol, cereals, oils, sugar, and salt was minimal. An estimated two-thirds of the Aché hunter-gatherer diet consisted of sweet manioc, peanuts, maize, and rice. Approximately one-quarter of their diet consisted of flour, sugar, and meat. No cases of acne were detected in either population. The authors suggested that the low fat intake and the absence of high-glycemic-index foods may explain the low prevalence of acne in these populations.

Freyre *et al.*[20] compared acne prevalence in three Peruvian populations, including indigenous and white populations (n = 2214). Among 12–18-year-olds, the indigenous population showed a significantly ($P < 0.001$) lower acne prevalence (28%) than the white population (45%) or those of mixed ancestry (43%). Each adolescent group had a lower prevalence of acne than that reported in 12–18-year-old Americans.[20]

Bechelli *et al.*[21] assessed the prevalence of acne in 9955 Brazilian schoolchildren: 8980 were impoverished urban children, whereas 975 were from rural areas. Less than 3% of the combined population (2.7%) demonstrated evidence of acne.

Two reports have suggested that acne prevalence increases as populations adopt a Western diet through migration or cultural change. Reports of northern Canadian Inuits made no mention of acne until acculturation with their southern neighbors and subsequent increases in soda, beef, dairy products, and processed foods, after which the acne prevalence increased.[22] Pre-World War II Okinawans, who traditionally followed a diet of sweet potatoes, rice, and vegetables, together with some soybeans, but little meat, reported an increase in acne prevalence after adopting a diet high in animal products.[23]

Seven studies have assessed the perceptions of factors believed to affect acne.[3,7–12] In a 2007 study, Rigopoulos *et al.*[7] assessed the beliefs about acne among 13–18-year-old Greek students with and without acne. Self-reported acne was present among 59% of students. Among 316 students with and without acne, 62% cited diet as a causal factor, and 66% believed chocolate was an exacerbating factor. In 2006, El-Akawi *et al.*[10] reported that, of 166 Jordanian male and female untreated clinic patients with acne, participants believed that their acne was aggravated by nuts (89%), chocolate (85%), cakes/biscuits (57%), oily food (53%), fried food (52%), eggs (42%), or milk, yogurt, and cheese (23%). Nearly one-fifth (19%) believed that consuming fruits and vegetables improved their acne. A survey administered by Ikaraoha *et al.*[11] to 174 Nigerian students aged 18–32 years demonstrated that 75% of participants believed that an oily or fatty diet contributed to their acne. Of 130 male and female Saudi Arabian patients attending an acne clinic, more than one-quarter of participants believed that diet caused (26%) or exacerbated (32%) their acne.[8] A 2003 study[9] of Saudi Arabian high school and college students (n = 517), aged 15–29 years, demonstrated that 72% of students with and without acne (and 79% of 217 students with acne) believed that diet contributed to acne. In a 2001 study in the *Journal of the American Academy of Dermatology*, Tan *et al.*[3] reported that acne was believed to be caused by diet less frequently than by hormonal or genetic factors. Although 32% of acne patients believed that diet caused their acne, 64% believed that hormones and 38% believed that genetics were responsible. In addition, 44% of participants believed that diet aggravated their acne. In 2001, Green and Sinclair[12] reported that almost half of 215 sixth-year Australian medical students believed that diet aggravated their patients' acne, citing chocolate and oily or fatty foods as the most common dietary factors.

Kaymak *et al.*[24] examined the association between acne and the glycemic index and glycemic load of the daily diet, insulin sensitivity, and insulin-like growth factor (IGF) levels in 91 university students (n = 49 acne patients, n = 42 control patients). Participants completed a food frequency questionnaire from which the authors calculated the glycemic index using published reports. Physicians assessed acne as well as insulin resistance through the calculation of the homeostatic model assessment (HOMA) index: [fasting insulin (microU/mL) × fasting blood glucose (mmol/L)/22.5]. There were no significant differences in fasting glucose or insulin levels, and none of the participants had insulin resistance. In patients with

Table 1 Observational and interventional studies of the association between diet and acne

Reference	Year	Study design	Population	Total *n*	Acne assessment	Intervention (if applicable)	Study length (if applicable)	Results
Observational studies								
Cordain *et al.*[19]	2002	Cross-sectional; population-based	Male, female ≥ 10 years	1315	General practitioner	None	N/A	No acne (0%) in two populations reporting non-Western diets No other analyses reported
Freyre *et al.*[20]	1998	Cross-sectional; population-based	Male, female	2214	One "professional"	None	N/A	Indians had lower prevalence of acne (28%) than white (45%) or mixed-race (43%) populations ($P < 0.001$)
Bechelli *et al.*[21]	1981	Cross-sectional; population-based	Schoolchildren; age not reported	9955	2–4 dermatologists	None	N/A	Acne prevalence = 2.7% in entire population
Bendiner[22]	1974	Cross-sectional; population-based	Male, female All ages	Not reported	Practitioner; type not reported	None	N/A	Acne was first reported after adopting a high-glycemic diet (i.e. containing candy, soda)
Steiner[23]	1946	Cross-sectional; population-based	Male, female All ages	150	Practitioner; type not reported	None	N/A	Acne prevalence increased on an animal product-rich diet
Rigopoulos *et al.*[7]	2007	Cross-sectional	Male, female 13–18 years	316	Dermatologist	None	N/A	66% of students believed that chocolate caused acne
El-Akawi *et al.*[10]	2006	Cross-sectional	Male, female 13–42 years	166	Dermatologist	None	N/A	Acne patients believed acne was aggravated by nuts (89%), chocolate (85%), cake (57%), and fried foods (52%), and improved by fruits, vegetables (19%)
Ikaraoha *et al.*[11]	2005	Cross-sectional	Male, female 18–32 years	174	Self-reported	None	N/A	75% of students with acne believed a fatty diet caused acne
Tallab[8]	2004	Cross-sectional	Male, female Mean age 21 years	130	Dermatologist	None	N/A	Acne patients (≥ 26%) believed diet caused or aggravated acne
Al-Hoqail[9]	2003	Cross-sectional	Male, female 15–29 years	517	Self-reported	None	N/A	72% of students believed diet caused acne
Tan *et al.*[3]	2001	Cross-sectional	Male, female Mean age 22 years	78	Dermatologist	None	N/A	Acne patients (≥ 32%) believed diet caused or aggravated acne
Green and Sinclair[12]	2001	Cross-sectional	Gender, age not reported; 6th-year university students	215	No acne assessment	None	N/A	41% ($n = 88$) of medical students believed diet aggravated acne; 12% of these 88 blamed chocolate
Kaymak *et al.*[24]	2007	Cross-sectional	Male, female Mean age 22 years	91	Practitioner; type not reported	None	N/A	Positive association with IGF-1, negative association with IGF-BP-3 levels ($P < 0.05$) Longer acne duration positively associated with higher glycemic index levels ($P < 0.05$)
Khanna *et al.*[25]	1991	Cross-sectional	Male, female; age not reported Students	200	Self-reported	None	N/A	No association with calories, protein, carbohydrates, or fat ($P > 0.05$)
Bourne[26]	1956	Cross-sectional	Male 15–40 years	2720	Army physician	None	N/A	Adolescents: no association with body weight ($P > 0.05$) Adults: positive association with body weight ($P = 0.013$)
Adebamowo *et al.*[27]	2005	Case–control	Female 25–42 years	47,355	Recall of "physician-diagnosed severe acne"	None	N/A	Negative association with saturated fat (PR = 0.9; 95% CI, 0.80–0.94) Positive associations with total milk intake (PR = 1.2; 95% CI, 1.03–1.44) and vitamin D Inverse dose–response between milk fat and acne

Table 1 *Continued*

Reference	Year	Study design	Population	Total *n*	Acne assessment	Intervention (if applicable)	Study length (if applicable)	Results
Bett *et al.*[28]	1967	Case–control	Male, female Mean age 20 years	48	Dermatologist	None	N/A	No association with sugar intake ($P > 0.05$)
Adebamowo *et al.*[29]	2008	Prospective cohort	Male 9–15 years	4273	Self-reported	None	Three years	Positive association with total milk intake (PR = 1.16; 95% CI, 1.01–1.34)
Adebamowo *et al.*[30]	2006	Prospective cohort	Female 9–15 years	6094	Self-reported	None	Three years	Positive association with all categories of milk intake (PR = 1.20; 95% CI, 1.06–1.32 for total milk intake)
Chiu *et al.*[31]	2003	Prospective cohort	Male, female Mean age 22 years	22	Self-reported	None	One semester	Perceived diet quality negatively associated with acne exacerbation and acne severity ($r = -0.5$, $P = 0.02$)
Robinson[32]	1949	Prospective cohort	Male, female All ages *n* = 1583 aged 16–25 years	2083	Dermatologist	None	Not reported	Milk products positively associated in diet records with acne Dairy-free, low-fat diet prescribed, but no follow-up
Clinical trials								
Smith *et al.*[33]	2008	Interventional, control group (single-blind)	Male 15–25 years	31	Dermatologist	Low-glycemic-load diet	12 weeks	Positive association with ratio of saturated/monounsaturated fatty acids in skin surface ($P = 0.007$)
Smith *et al.*[34,39]	2007	Interventional, control group (single-blind)	Male 15–25 years	43	Dermatologist	Low-glycemic-load diet	12 weeks	Negative associations with acne lesions ($P \leq 0.03$) and insulin sensitivity test ($P = 0.03$) Positive association with IGF-BP-1, negative association with SHBG levels ($P \leq 0.03$)
Anderson[35]	1971	Interventional	Male, female Age not reported University students	≥ 27	Dermatologist	Self-selected food (peanuts, chocolate bars, milk, or cola)	One week	27 cases of food sensitivity No association with any test food overall Quantitative analyses not reported
Fulton *et al.*[36]	1969	Interventional, control group (crossover single-blind)	Male, female Adolescents and adults Age not reported	65	Dermatologist	Non-milk chocolate bars (placebo or 10-fold cocoa-enhanced)	Four-week intervention and three-week washout	No association with cocoa-enhanced chocolate Quantitative analyses not reported
Grant and Anderson[38]	1965	Interventional	Male, female Age not reported University students	8	Senior medical student and dermatologist	Milk chocolate bars	Two days of chocolate bars; five days of observation	No association with chocolate overall Quantitative analyses not reported
Gaul[37]	1965	Interventional	Male, female 14–24 years	30	Dermatologist	Low-sodium diet	Three months	Fewer and less active acne lesions on test diet Quantitative analyses not reported

IGF, insulin-like growth factor; IGF-BP, IGF-binding protein; N/A, not applicable; PR, prevalence ratio; SHBG, sex hormone-binding globulin.

acne, levels of IGF-1 were higher and levels of IGF-binding protein-3 were significantly lower than those of controls. Participants with acne of more than 2 years' duration ate a diet with a significantly higher glycemic index than did participants with acne of less than 2 years' duration.

Khanna *et al.*[25] studied energy, carbohydrate, protein, and fat intake among 200 students in India with and without acne. They compared the diets of those with severe, moderate, mild, and no acne, and reported no dietary differences ($P > 0.05$) among the four groups; however, the researchers used a *t*-test, intended for the comparison of two groups, rather than the appropriate analysis of variance statistical test to compare the four groups. It is unclear whether the results from appropriate statistical tests would have differed from those reported.

In a 1956 study published in the *British Medical Journal*, Bourne[26] found that adult British soldiers, aged 20–40 years, with acne were significantly heavier than those without acne (73.2 kg vs. 67.5 kg; $P = 0.013$). Adolescent British soldiers aged 15–19 years with acne weighed more than those without acne (62.7 kg vs. 60.5 kg), although the differences were not significant.

Case–control studies

In a 2005 study, Adebamowo *et al.*[27] tested the hypothesis that milk (whole, powdered, low-fat, and skimmed) intake was associated with a risk of teenage acne. More than 47,000 nurses were questioned about their adolescent diets and whether they had experienced "physician-diagnosed severe acne" during their teenage years. Prevalence ratios (PRs), comparing acne prevalence at the highest (more than three servings per day) to lowest (one serving or less per week) intake categories, were computed. In multivariate models adjusted for energy, present age, age of menarche, and body mass index, total milk intake was associated with severe acne [PR = 1.22; 95% confidence interval (CI), 1.03–1.44]. Severe acne prevalence increased as the milk fat content decreased: PR = 1.12 (CI, 1.00–1.25) for whole milk; PR = 1.16 (CI, 1.01–1.34) for low-fat milk; PR = 1.44 (CI, 1.21–1.72) for skimmed milk. Trend tests were significant for total milk [P(trend) = 0.002] and skimmed milk [P(trend) = 0.003]. The only nutrients significantly and positively associated with acne were vitamin D supplementation and total energy intake. High saturated fat intake was inversely associated with acne risk (PR = 0.88; CI, 0.80–0.94). There were no associations between acne and soda, French fries, pizza, or chocolate. The authors hypothesized that the hormones found in milk products were responsible for milk's association with acne.

Bett *et al.*[28] tested the hypothesis that acne patients consumed more sugar than age- and sex-matched controls. They compared the sugar consumption in 16 patients with acne with that in 16 patient controls with warts and 16 healthy age- and sex-matched office and factory worker controls. Diet was assessed by food frequency questionnaire. There were no significant differences in sugar consumption among the groups (121 g/day for acne patients vs. 111 g/day for wart patients and 120 g/day for healthy controls; $P > 0.05$).

Cohort studies

Four prospective cohort studies have evaluated the associations between diet and acne.[29–32] Adebamowo *et al.* followed 4273 boys[29] and 6094 girls,[30] aged 9–15 years, in 1996. Information on dietary intake was collected between 1996 and 1998, and acne prevalence and severity were assessed in 1999. The authors investigated the association between self-reported acne severity and cow's milk (whole/2%, 1%, skimmed, chocolate) intake. PRs, comparing acne prevalence at the highest (two or more servings per day) to lowest (one serving or less per week) intake categories, were computed. After adjustment for baseline age, height, and energy intake, acne severity in girls[30] was significantly associated with the intake of all categories of cow's milk: PR = 1.20 [CI, 1.09–1.31; P(trend) < 0.001] for total milk; PR = 1.19 [CI, 1.06–1.32; P(trend) < 0.001] for whole milk; PR = 1.17 [CI, 1.04–1.31; P(trend) = 0.002] for low-fat milk; PR = 1.19 [CI, 1.08–1.31; P(trend) < 0.001] for skimmed milk; PR = 1.29 [CI, 1.08–1.53; P(trend) = 0.02] for chocolate milk. In boys,[29] the association between milk intake and acne was significant for total milk (PR = 1.16; CI, 1.01–1.34) and skimmed milk (PR = 1.19; CI, 1.01–1.40) intake. The test for trend was significant only for skimmed milk intake [P(trend) = 0.02]. Among both boys and girls, there were no significant associations between acne and the intake of nonmilk dairy foods, French fries, pizza, or chocolate.

In a 2003 study, 22 university students were followed for one semester by Chiu *et al.*[31] "Dietary quality" was determined by the number of meals eaten per day and subjective self-ranking of "diet quality" on a four-point scale. Descriptive dietary data were not provided. Perceived dietary quality was inversely associated with acne exacerbation and severity ($r = -0.48$, $P = 0.02$).

During the course of a study on radiation treatment for acne in 2083 patients between 1925 and 1949, Robinson[32] collected 1–2 weeks of dietary records in a subset of patients. The number of dietary records was not reported. Milk products were the most frequently cited acne-causing food. Patients were subsequently advised to follow a low-fat, dairy-free diet; however, postintervention follow-up results were not published.

Clinical trials

Table 1 summarizes the results of six dietary intervention studies.[33–38] Three[33,34,36] of the six studies included control groups.

Smith *et al.*[34] assessed the effect of a low-glycemic-load diet (25% energy from protein and 45% energy from

low-glycemic-index carbohydrates) on acne and insulin sensitivity. Participants ($n = 43$, all male, aged 15–25 years) were randomly assigned in a parallel design to the dietary intervention or control group urged to regularly include carbohydrates without receiving information on the glycemic index. Participants were followed for 12 weeks. Blind dermatologists assessed the number of acne lesions every four weeks, starting at baseline. Relative to those on the control diet, participants on the low-glycemic-load diet experienced greater reductions in counts of all lesions (51% vs. 31%; $P = 0.03$) and inflammatory lesions (45% vs. 23%; $P = 0.02$). Participants in the intervention group experienced a significant improvement (i.e. increase) in insulin sensitivity and significant changes in androgen levels, compared with participants in the control group.[39] A positive correlation was observed between the change in total lesion counts and the change in insulin sensitivity as measured by the HOMA index ($r = 0.38$, $P = 0.01$). A change in sex hormone-binding globulin (SHBG) levels also correlated negatively with a change in lesion counts ($r = -0.38$, $P = 0.01$).

Sebum sampling was completed by 31 participants ($n = 16$ intervention participants, $n = 15$ control participants).[33] At baseline and 12 weeks, follicular sebum outflow and the composition of skin triglycerides were assessed. Follicular sebum outflow and the proportions of sebum fatty acids did not differ between the groups. Compared with baseline levels ($P = 0.007$), however, participants in the intervention (but not the control) group demonstrated an increased ratio of saturated to monounsaturated sebum fatty acids. The change in ratio correlated negatively and significantly with the change in acne lesion counts ($r = -0.39$, $P = 0.03$). The study authors concluded that the desaturation of sebum fatty acids may play a role in acne development.

Anderson[35] tested the effect of four foods on acne. For 1 week, medical students were asked to consume daily servings of a self-selected test food [6 small (39-g) chocolate bars, 0.95 L of milk, 113 g of roasted (iodized) salted peanuts, or 0.71 L of cola]. Nine participants developed new acne lesions in 10 days of the tests. Nine participants developed no new lesions. The results were not grouped by test foods, and the study was neither controlled nor blind. The authors reported that the self-selected foods were not associated with acne.

Two studies specifically tested the acne-causing ability of milk chocolate. In an uncontrolled intervention in eight university students, Grant and Anderson[38] fed participants a 9.75-oz milk chocolate bar and instructed them to eat an identical second bar the next day. By day five, four participants had developed new lesions, but four had not. Statistical analyses were not presented. The investigators concluded that large amounts of chocolate did not aggravate acne.

In a single-blind crossover trial[36] designed to test the effect of chocolate on acne, 65 participants were asked to consume one 112-g dairy-free chocolate bar every day for 4 weeks. The bar was enriched 10-fold with cocoa solids and cocoa butter. During the control phase, subjects were instructed to consume a similar sized chocolate bar enriched with 28% partially hydrogenated vegetable (i.e. "trans") fat in place of the cocoa paste and cocoa butter. A 30% increase or decrease in acne lesions was considered to be clinically significant. Differences between experimental and control groups did not reach the designated clinical threshold and were not specifically reported. The authors concluded that large amounts of chocolate did not significantly affect acne, sebum composition, or sebum secretion.

In 1965, Gaul[37] prescribed a 3-month low-salt diet for 30 acne patients with baseline urinary chloride excretion of at least 12–20 g/L (the salt intake was not reported). In four cases, salt restriction reduced the number and severity of pustules and cysts. Quantitative analyses and data for the 26 other study participants were not reported.

Discussion

Population-based studies have suggested that, as diets Westernize, acne prevalence increases. Observational studies, including one case–control study[27] and two large and well-controlled prospective cohort studies,[29,30] have demonstrated an association between cow's milk intake and acne prevalence and severity (Table 2). One prospective cohort study[24] demonstrated an association between high-glycemic-index foods and longer acne duration, whereas two randomized controlled trials[33,34] demonstrated that a low-glycemic-index diet reduced acne risk. Studies have been inconclusive with respect to the association between acne and chocolate (perhaps because of methodological limitations), and have failed to demonstrate an association between acne and salt or iodine intake.

Methodologic issues may have limited the conclusions that could be drawn from the literature before 2005. Prior to that date, two prospective studies[24,32] failed to explicitly state the follow-up duration, only one intervention[36] utilized a control group, and another intervention[38] failed to clearly define the

Table 2 Summary of associations between acne and selected foods and dietary patterns

Dietary intake	Established acne causation in a specific population?
High-glycemic-load diet	Yes
Dairy (skimmed, chocolate, or total milk)	Yes
Chocolate	Inconclusive
Salt	No
Iodine	No
Saturated fatty acids	Inconclusive

changes in acne. Published studies suffered from a small sample size, lack of appropriate controls, and incomplete reporting of results. Recently published prospective studies[24,29,30,33,34] have utilized clearly defined interventions, outcomes, and appropriate statistical analyses.

Previous studies have provided the theoretical basis for an association between diet and acne. Acne typically results from excess sebum production, which causes follicular cells to hyperproliferate and block the follicle opening. Bacteria can then hypercolonize the follicle, and an immune response results in inflammation. Sebum production may be influenced by androgens and hormonal mediators, such as SHBG and IGF-1, all of which may be influenced by dietary factors, as described below.

Dairy products

Three large studies reported a positive association between milk intake and acne. The studies of Adebamowo *et al.*[29,30] demonstrated that higher levels of milk consumption were associated with acne risk in both boys and girls. In the Nurses' Health Study II,[27] women who consumed more milk as adolescents (based on later recall) showed a greater prevalence of severe acne than those with less frequent consumption. These findings are supported by previous population-based studies,[19,22,23] in which areas with minimal consumption of dairy products had a very low acne prevalence.

In recent observational studies, skimmed milk was consistently associated with acne, suggesting that the fat content of milk does not appear to affect its acne-causing ability. Some authors have reported that the hormones in milk, such as IGF-1, 5α-reduced steroids, and α-lactalbumin, may survive milk processing and affect the pilosebaceous unit.[40] Milk consumption also increases IGF-1 production, which has been associated with ovarian androgen production in premenarchal girls and acne in adult women.[41,42]

High-glycemic-index foods

Findings from the studies of Smith *et al.*[33,34,39] have illustrated the various interactions between glycemic load, insulin sensitivity, hormonal mediators, and acne. Regular consumption of foods with a high glycemic index elevates serum insulin concentrations, which may stimulate sebocyte proliferation and sebum production,[43] suppress SHBG concentrations and raise androgen concentrations,[44] and contribute to acne. Conversely, low-glycemic-index foods have been shown to increase SHBG and reduce androgen levels; higher SHBG levels have been associated with lower acne severity.

Intervention participants in Smith's study[34] also lost weight, and the low-glycemic-load diet was higher in polyunsaturated fat and fiber and lower in saturated fat, compared with the comparison diet. These differences may be expected to influence inflammation and acne-related hormones.

Fat intake

A 1997 study of 871, 10–11-year-old girls followed prospectively for 5 years[45] found that those with severe (vs. mild or moderate) comedonal acne had significantly higher androgen levels and significantly earlier menarche. Although diets high in saturated fat increase the concentration of IGF-1, low-fat, high-fiber diets tend to decrease the concentrations of IGF-1[46] and androgens,[47] and increase the concentration of SHBG.

Fatty acid composition

The ratio of omega-6 to omega-3 fatty acids in Western diets is commonly at least 10 : 1, compared with ratios of 4 : 1 in Japan and 2 : 1 in historic, nonindustrialized populations.[48] Studies have suggested that inflammatory markers increase as this ratio increases. Omega-6 fats are precursors to proinflammatory mediators and have been associated with the development of inflammatory acne.[6] In contrast, high levels of omega-3 fatty acids have been shown to decrease inflammatory factors,[49] and may reduce acne risk by decreasing IGF-1 levels and preventing hyperkeratinization of sebaceous follicles.

Chocolate

Grant and Anderson[38] and Anderson[35] tested the acne-causing ability of milk chocolate bars. Grant and Anderson[38] did not provide a control group, and neither study reported quantitative statistical analyses. Fulton *et al.*[36] provided a control group and quantified their results. Moreover, they specifically tested cocoa rather than nonfat milk solids, milkfat, or sugar; however, the fat and sugar contents of treatment and control bars were nearly identical, minimizing the potential to detect the effects of fats or sugars on acne; the study findings did not provide conclusive evidence to establish whether cocoa solids influence acne formation.

Conclusions

Evidence suggests that components of Western diets, particularly dairy products, may be associated with acne. The hormonal effects of dietary components, such as glycemic index levels or fat or fiber intake, may mediate the effect of diet on acne risk.

Until 2005, cross-sectional, case–control, cohort, and clinical intervention studies designed to address the relationship between diet and acne typically failed to incorporate adequate controls, objective measures, and appropriate statistical analyses. Well-designed prospective studies published since 2005 have elucidated the mechanisms whereby particular foods and dietary constituents may influence acne risk and severity. In order to test the efficacy of dietary interventions, prospective, randomized trials, including controls for environmental stressors, acne medications, age, pubertal stage, and age at menarche, are essential.

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