# **The Role of Diet in Acne and Rosacea**

## by JONETTE E. KERI, MD, PHD, and ADENA E. ROSENBLATT

Department of Dermatology, University of Miami Miller School of Medicine, Miami, Florida

### ABSTRACT

Diet plays an important role in the pathogenesis of a variety of diseases. Recently, there has been an increasing interest in the role of diet in the development of common skin conditions such as acne and rosacea. The association of diet and acne has been controversial and unclear. Historically, it was thought that diet did not cause acne; however, recent studies reveal a potential role of diet in the pathogenesis of acne. Individuals that have a diet with a high glycemic load or increased milk consumption are reported to have a greater likelihood of having acne. This effect may be mediated by insulin-like growth factor-1 (IGF-1) since IGF-1 levels increase with milk consumption and with a high glycemic load and are known to contribute to the formation of acne. In contrast to acne, the association of diet and rosacea is much more accepted and established. There are a variety of foods that aggravate rosacea, including spicy foods, alcoholic beverages, and hot, caffeinated drinks. Patients are advised to avoid these triggers. Interestingly, omega-3 and omega-6 fatty acids may be beneficial in the treatment of rosacea, although further investigation is necessary. Understanding the etiologic role of diet in acne and rosacea may help in the prevention and treatment of these conditions. (J Clin Aesthetic Derm. 2008;1(3):22-26.)

ur society in recent years has become increasingly interested in what we ingest. Consumers know more today than ever before about what ingredients are in the food they eat. Therefore, it was only natural to connect diet with skin diseases, particularly acne and rosacea since they are so common in the general population.

### THE ROLE OF DIET IN ACNE

When discussing the treatment options for acne, patients' beliefs must be considered. Rigopoulos et al, in a study of Greek teenagers (aged 13-18 years), found diet to be the most commonly associated causal or exacerbating factor in relation to acne, with hormones coming in second. An important take-home message from this study was that the Greek adolescents' beliefs about acne were similar to the beliefs of others in developing countries. Similar surveys show similar results, suggesting patients think diet plays a role in acne. A quick review of previous studies is noted in the article.<sup>1</sup> Entire books, such as the *Dietary Cure for Acne*, are devoted to dietary intervention (with a role for a low glycemic load diet) for the treatment of acne, and some practitioners recommend these books to their patients.<sup>2</sup> Therefore, it is imperative that dermatologists know what patients think when it comes to acne.

For more than 30 years, most dermatologists have told patients that acne is not caused by what they eat. These concepts came from two studies evaluating the role of chocolate, chocolate bars, milk, peanuts, and Coca-Cola<sup>™</sup> in acne.<sup>3-4</sup> The study involving chocolate is frequently misinterpreted for a few reasons. First, the difference between the "imitation" chocolate and "real" chocolate bars was cacao solids (cacao paste and cocoa butter). There was also no difference in sugar and fat content in the bars and there was no milk in the bars. Regardless, this study has been held as a standard for many years, although critiques are understandable as nutritional research advances.5 Of note, most studies on acne and diet are hampered by various confounders including genetics, ethnicity, and sex of the patient, and some by lack of controlled dietary groups. The practitioner reading such studies should be aware of possible biases.

### **GLYCEMIC LOAD AND ACNE**

A diet with a high glycemic load may play a role in the pathogenesis of acne. Glycemic load assesses the potential of a food to increase blood glucose and is defined as glycemic index multiplied by carbohydrate content. The glycemic index is a relative comparison of the potential of various foods to increase blood glucose given that there is an equal amount of carbohydrate in the food.<sup>6</sup> Glycemic index is a measure of carbohydrate quality (i.e., nature or source), but not quantity.7 Glycemic load takes into consideration carbohydrate quality and quantity.7

DISCLOSURE: The authors identified no conflicts of interest. ADDRESS CORRESPONDENCE TO: Jonette E. Keri, PO Box 016250 (r-250), Miami, FL 33101; jkeri@med.miami.edu



# TABLE 1. Glycemic indices (low = 1–55, moderate = 56–69, high = 70–100) and glycemic loads (low = 1–10, moderate = 11-19, high = 20+) for various foods

TYPE OF FOOD	GLYCEMIC INDEX	GLYCEMIX LOAD	TYPE OF FOOD	GLYCEMIC INDEX	GLYCEMIX LOAD
Coca-Cola <sup>1</sup>	63	16	Doughnut	76	17
Multigrain bread	43	6	Gatorade <sup>2</sup>	78	12
All–Bran cereal <sup>3</sup>	38	9	White bread	70	10
Sweet corn	60	20	Cornflakes cereal <sup>3</sup>	92	24
Brown rice	50	16	Chickpeas	31	9
Skim milk	32	4	White rice	56	23
lce cream	62	8	Whole milk	40	3
Apple	40	6	Low-fat yogurt	14	2
Grapes	43	7	Banana	51	13
Watermelon	72	4	Mango	41	8
Macaroni and cheese⁴	64	32	Peach	28	4
Corn chips⁵	42	11	Spaghetti	32	15
M&M's peanut <sup>7</sup>	33	6	Kudos bar⁵	62	20
Popcorn <sup>9</sup>	55	6	Cashew nuts <sup>®</sup>	22	3
Carrots	47	3	Pretzels <sup>10</sup>	83	16
Sweet potato	48	16	Baked potato	60	18

Glycemic reference is glucose with a glycemic index of 100. Adapted from Foster-Powell et al.<sup>7</sup>

<sup>1</sup>Atlanta, GA <sup>2</sup>Spring Valley Beverages Pty Ltd, Cheltenham, Australia <sup>3</sup>Kellogg's, Battle Creek, MI <sup>4</sup>Kraft General Foods Canada Inc, Don Mills, Canada <sup>5</sup>Smith's Snack Food Co., Australia <sup>6</sup>M&M/Mars, Hackettstown, NJ <sup>7</sup>Mars Confectionery, Australia <sup>8</sup>Coles Supermarkets, Australia <sup>9</sup>Green's Foods, Australia <sup>10</sup>Parker's, Smith's Snack Food Co., Australia

Clinical and Aesthetic

23

Furthermore, there is some variability in the values assigned to foods depending on geographic location (i.e., brown rice from Canada compared to brown rice from the United States) as well as the particular preparation of the food (i.e., fresh corn compared to frozen corn).<sup>7</sup> Low glycemic index foods have values between 1 and 55, moderate glycemic index foods have values between 56 and 69, and high glycemic index foods have values between 70 and 100. Glycemic load values between 1 and 10 are low, glycemic load values between 11 and 19 are moderate, and a glycemic load value of 20 or more is considered high. Table 1 lists the glycemic indices and loads for a variety of foods, and shows that processed foods, such as white bread and doughnuts, have much higher glycemic loads than unprocessed fruits and vegetables. For a complete database on the glycemic indices and loads for a large number of foods as well as information on the glycemic index, practitioners can refer their patients to www.glycemicindex.com. Cordain et al proposed that there may be an association between acne and glycemic load, reporting the prevalence of acne in two non-Westernized populations.<sup>8</sup> These populations, the Ache hunter-gatherers of Paraguay and the Kitavan Islanders of Papua New Guinea, had no evidence of acne. The authors hypothesized that these people had low glycemic loads. Their diets differ from Westernized diets, which often create a high glycemic load. However, controversy with this study stems from debate as to how much of the condition is related to genetics versus environment. To truly elucidate the role of diet in acne, there must be a mechanism by which diet leads to the development of acne.

The most recent attempt to make the connection between diet and acne comes from a study of male patients (aged 15–25 years) who were placed on a low glycemic load experimental diet versus controls.<sup>9</sup> The patients' skin surface lipids were analyzed after tape stripping collection with the results suggesting a change in fatty acid composition of sebum. When evaluating the study, it must first be noted that sebum is not the sole cause of acne; acne is a multifactorial disease. Secondly, the change in sebum composition could have been related to the weight loss the experimental subjects experienced. Finally, the study included only males, and it did not take into account genetics.

A high glycemic load diet leads to an increase in insulin and a subsequent increase in free insulin-like growth factor-1 (IGF-1), which is known to be involved in the pathogenesis of acne.<sup>10</sup> IGF-1 is a hormone that has a structure similar to insulin and is involved in cell growth and proliferation. IGF-1 promotes acne by inducing hyperkeratosis and epidermal hyperplasia, a first step in the follicular plug.<sup>11</sup> IGF-1 also stimulates sebaceous gland lipogenesis and androgens which are known to cause an increase in sebum.<sup>12-13</sup> It appears that IGF-1 and androgens can act synergistically in the pathogenesis of acne. This carries over to the clinic setting, where post-adolescent women (20–25 years) with acne had higher levels of IGF-

24

1 versus those women without acne.<sup>14</sup> Young men with acne have also been studied with respect to their diet. Smith et al recently studied 43 men (15-25 years) with acne who were given instructions to follow a high carbohydrate diet similar to their current diet (control group) compared to a group given instructions to follow a low glycemic load diet for 12 weeks.<sup>15</sup> There was a significant decrease in the number of acne lesions following diet modification in the low glycemic load group compared to the control group.<sup>15</sup> The low glycemic load group also noted weight loss, reduced free androgen index and dehydroepiandrosterone sulfate (DHEAS), and increased insulin-like growth factor binding protein-1 (IGFBP-1), which is known to be inversely correlated with insulin and free IGF-1 levels.<sup>15-17</sup> Another prospective cohort study of 49 university students (men and women) were also evaluated with respect to their diets, presence of acne, and specific laboratory values. In this study, no differences were observed between patients with acne and controls in serum glucose, insulin, leptin, overall glycemic index, or glycemic load.<sup>17</sup> The one result that can be viewed with interest was that the glycemic index was significantly higher in patients with acne for longer than two years compared to those with acne for less than two years.<sup>17</sup> This may be relevant to the recent increase in prevalence of adult acne noted by many physicians, and should be further explored. Limitations to this study include small sample size and patient recall bias.

In clinical practice how can we connect IGF-1 and acne? Some of our best evidence for a role of diet comes from patients with polycystic ovarian disease (PCOS). PCOS is characterized by acne, irregular menses, alopecia, hirsutism, infertility, insulin resistance, and obesity. However, a patient can have PCOS and not have all of its characteristics. Laboratory evaluations of such patients reveal increased levels of insulin, free IGF-1, and IGFBP-1 levels.18-20 androgens and decreased Interestingly, acne improves with medications that improve insulin metabolism, such as metformin, tolbutamide, and pioglitazone, indicating the insulin metabolism plays a role in the pathogenesis of acne in PCOS patients.<sup>21</sup> Insulin-sensitizing agents also increase IGFBP-1 levels and decrease IGF-1 levels.<sup>22–24</sup> Unfortunately, there has been no study of the effects of a low glycemic diet on acne and IGF-1 levels in PCOS patients. Further investigation of the relationship between diet, acne, and IGF-1 levels is necessary, particularly in PCOS patients.

### **MILK CONSUMPTION AND ACNE**

Milk consumption has also been associated with increased IGF-1 levels and implicated in the pathogenesis of acne.<sup>25</sup> In addition to IGF-1, it is important to note that milk contains a variety of other hormones including estrogens; progesterone, which can act as an androgen receptor agonist; and dihydrotestosterone (DHT) precursors such as 5-pregnanedione and 5-androstenedione.<sup>26</sup> While progesterone and DHT

precursors may be important in promoting acne in milk consumers, estrogen, in the form of birth control, has been used to treat acne. Therefore, it is unclear whether hormones in milk promote acne or protect against it. The association between milk consumption and acne has been known for many years and has gained more support from recent studies.<sup>27</sup> One study of 47,355 women from the Nurses Health Study II retrospectively assessed dietary intake and whether participants had acne during their teenage vears.27 The participants completed questionnaires regarding diet and teenage acne. This study found a positive association between milk and teenage acne.<sup>28</sup> Limitations of this study include its retrospective design and possible recall bias. More recently, the offspring of the women in the Nurses Health Study II listed above were evaluated in the Growing Up Today Study (GUTS) for the presence of acne. A positive association was found between milk and acne in this population as well.<sup>29</sup> Although there was also a positive association in this population, it is important to note that since the participants of GUTS are the offspring of the Nurse Health Study II subjects, the genetic component needs to be taken into consideration and it was not. In addition, this study only looked at young men and may again involve recall bias.

Counseling of patients on diet and acne can be done quickly with an emphasis on a few salient points. It is important to educate not only the patient, but accompanying family members as well, if present. The multifactorial pathogenesis of acne should be reviewed and patients should be told that the role of diet and acne has not been defined clearly. Women with possible PCOS should be considered as a population of patients where diet may play a more significant role. Identifying these patients can lead to long-term health benefits since identification of insulin resistance and tendency toward hyperlipidemia at an earlier age leads to treatment that can decrease long-term morbidity. Also, early identification can help with infertility, which may go unnoticed until a later time. All PCOS patients are advised to follow a low glycemic index diet.<sup>30-31</sup> This diet may not only reduce acne in these patients, but it will also improve their insulin resistance. Finally, it is important not to dismiss a patient who swears by a perceived trigger of acne. If they indeed have such a trigger, it should be avoided as long as good dietary practice is followed. For example, if a patient abstains from milk products, he or she should take oral calcium supplements.

### THE ROLE OF DIET IN ROSACEA

Unlike acne, the belief that food aggravates rosacea has been accepted. Patients are counseled to avoid rosacea triggers and, thus, refrain from such items as spicy foods; alcoholic beverages; and hot, caffeinated drinks. The list of triggers can be more extensive and include fruits, marinated meats, and cheeses. A good reference for patients concerning such foods is the National Rosacea Society (www.rosacea.org). A 1999 survey by the National Rosacea Society of 3,151 rosacea patients determined different food triggers. With regard to alcohol ingestion in rosacea patients, this survey found red wine as the most likely culprit, followed by hard liquor, then beer as the least likely to cause symptoms in patients. With regard to spices, cayenne pepper aggravated rosacea 36 percent of the time, red pepper 34 percent of the time, black pepper 18 percent of the time, white pepper 9 percent of the time, and paprika 9 percent of the time. In the fruit/vegetable category, citrus items and tomatoes were identified most often as triggers. For more information on this survey, visit the National Rosacea Society Website at www.rosacea.org.

In addition to particular food types being triggers for rosacea, the temperature of food and beverages can also aggravate rosacea. Guzman-Sanchez et al quantitatively evaluated heat sensitivity of the skin of rosacea patients.<sup>32</sup> They found that the heat-pain threshold was lower in the skin affected by rosacea, and that it was specifically more prominent in the papulopustular rosacea subtype compared to unaffected areas in the same patient. This provides good evidence that it is the heat of food that can exacerbate rosacea. For years, patients have been told to avoid hot, caffeinated beverages such as coffee and tea, and although caffeine may aggravate rosacea, there is now quantitative evidence that heat is a true culprit. Therefore, for many patients, an iced coffee or tea may be less of a trigger than originally thought.

Another novel dietary influence in rosacea patients may be the omega-3 and omega-6 fatty acids. Recently, there is evidence in the ophthalmology literature that supplements or foods that contain such fatty acids may help with dry eyes.<sup>33</sup> Some dermatologists have been using flax seed oil that contains high levels of omega-3 fatty acid to help combat ocular rosacea.

Clearly, the impact of diet on rosacea is far more accepted and documented than acne. Thus, patients with this condition should be encouraged to keep a food diary to help them elucidate possible triggers. With little effort, physicians can encourage such diaries and avoidance of triggers.

### **CONCLUSION**

In conclusion, practitioners may ask: "Should changes be made in clinical practice?" "What should we be telling our patients?" One answer to these questions may be that we still do not know the role of diet in acne. However, we can encourage our patients to eat well-balanced diets for overall better health. Additionally, dermatologists should be on the look out for patients with PCOS. Finally, if patients feel they have true triggers of acne or rosacea, they should be encouraged to avoid such foods while still eating a balanced diet.

#### REFERENCES

- Rigopoulos D, Gregoriou S, Ifandi A, et al. Coping with acne: beliefs and perceptions in a sample of secondary school Greek pupils. *J Eur Acad Dermatol Venereol.* 2007;21:806–810.
- 2. Loren Cordain, Phd. The Dietary Cure for Acne. Fort



Collins, CO: Paleo Diet Enterprises; 2006.

- Fulton JE, Plewig G, Kligman AM. Effect of chocolate on acne vulgaris. JAMA. 1969;210:2071–2074.
- 4. Anderson PC. Foods as the cause of acne. *Am Fam Physician*. 1971;3:102–103.
- 5. Cordain L. Implications for the role of diet in acne. Semin Cutan Med Surg. 2005;24:84–91.
- 6. Jenkins DJ, Wolever TM, Taylor RH, et al. Glycemic index of foods a physiological basis for carbohydrate exchange. *Am J Clin Nutr.* 1981;34(3):362–366.
- Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr.* 2002;76:5–56.
- Cordain L, Lindeberg S, Hurtado M, et al. Acne vulgaris: a disease of Western civilization. Arch Dermatol. 2002;138(12):1584-1590.
- Smith RN, Braue A, Varigos GA, Mann NJ. The effect of a low glycemic load diet on acne vulgaris and the fatty acid composition of skin surface triglycerides. J Dermatol Sci. 2008;50:41–52.
- Holt SH, Miller JC, Petocz P. An insulin index of foods the insulin demand generated by 1000-kJ portions of common foods. *Am J Clin Nutr.* 1997;66:1264–1276.
- 11. Edmondson SR, Thumiger SP, Werther GA, et al. Epidermal homeostasis the role of the growth hormone and insulin-like growth factor systems. *Endocr Rev.* 2003;24:737–764.
- 12. Deplewski D, Rosenfield RL. Growth hormone and insulin-like growth factor have different effects on sebaceous cell growth and differentiation. *Endocrinology*. 1999;140:4089–4094.
- Bol KK, Kiguchi K, Gimenez-Conti I, et al. Overexpression of insulin-like growth factor-1 induces hyperplasia, dermal abnormalities, and spontaneous tumor formation in transgenic mice. *Oncogene*. 1997;14:1725–1734.
- 14. Cappel M, Mauger D, Thiboutot D. Correlation between serum levels of insulin-like growth factor 1, dehydroepiandrosterone sulfate, and dihydrotestosterone and acne lesion counts in adult women. *Arch Dermatol.* 2005;141(3):333–338.
- 15. Smith RN, Mann NJ, Braue A, et al. The effect of a highprotein, low glycemic-load diet versus a conventional, high glycemic-load diet on biochemical parameters associated with acne vulgaris: a randomized, investigator-masked, controlled trial. J Am Acad Dermatol. 2007;57(2):247–256.
- 16. Smith RN, Mann N, Roper J, et al. A pilot study to determine the short-term effects of a low glycemic load diet on hormonal markers of acne: a nonrandomized, parallel, controlled feeding trial. *Mol Nutr Food Res.* 2008;52(6):718–726.
- 17. Kamayak Y, Adisen E, Ilter N, et al. Dietary glycemic index and glucose, insulin, insulin-like growth factor-I, insulin-like growth factor binding protein 3, and leptin levels in patients with acne. J Am Acad Dermatol. 2007;57(5):819–823.
- 18. Thierry van Dessel HJ, Lee PD, Faessen G, et al.

Elevated serum levels of free insulin-like growth factor I in polycystic ovary syndrome. *J Clin Endocrinol Metab.* 1999;84(9):3030–3035.

- Homburg R, Pariente C, Lunenfeld B, Jacobs HS. The role of insulin-like growth factor-1 (IGF-1) and IGF binding protein-1 (IGFBP-1) in the pathogenesis of polycystic ovary syndrome. *Hum Reprod.* 1990;5(1): 32–35.
- Suikkari A, Ruutiainenen K, Erkkola R, Seppala M. Low levels of low molecular weight insulin-like growth factorbinding protein in patients with polycystic ovarian disease. *Hum Reprod.* 1989;4(2):136–139.
- De Leo V, Musacchio MC, Morgante G, et al. Metformin treatment is effective in obese teenage girls with PCOS. *Hum Reprod.* 2006;21(9):2252–2256.
- 22. Pawelczyk L, Spaczynski RZ, Banaszewska B, Duleba AJ. Metformin therapy increases insulin-like growth factor binding protein-1 in hyperinsulinemic women with polycystic ovary syndrome. Eur J Obstet Gynecol Reprod Biol. 2004;113(2):209–213.
- 23. Berker B, Emral R, Demirel C, et al. Increased insulinlike growth factor-1 levels in women with polycystic ovary syndrome, and beneficial effects of metformin therapy. *Gynecol Endocrinol.* 2004;19(3):125–133.
- De Leo V, La Marca A, Orvieto R, Morgante G. Effect of metformin on insulin-like growth factor (IGF) I and IGFbinding protein I in polycystic ovary syndrome. J Clin Endocrinol Metab. 2000;85(4):1598–1600.
- 25. Koldovsky O. Hormones in milk. Vitam Horm. 1995;50:77–149.
- Grosvenor CE, Picciano MF, Baumrucker CR. Hormones and growth factors in milk. *Endocr Rev.* 1993;14(6):710–728.
- Holmes MD, Pollak MN, Willett WC, Hankinson SE. Dietary correlates of plasma insulin-like growth factor I and insulin-like growth factor binding protein 3 concentrations. *Cancer Epidemiol Biomarkers Prev.* 2002;11(9):852–861.
- 28. Adebamowo CA, Spiegelman D, Danby FW, et al. High school dietary dairy intake and teenage acne. J Am Acad Dermatol. 2005;52(2):207–214.
- Adebamowo CA, Spiegelman D, Berkey CS, et al. Milk consumption and acne in teenaged boys. J Am Acad Dermatol. 2008;58(5):787–793.
- Marsh K, Brand-Miller J. The optimal diet for women with polycystic ovary syndrome? Br J Nutr. 2005;94(2):154–165.
- 31. Liepa GU, Sengupta A, Karsies D. Polycystic ovary syndrome (PCOS) and other androgen excess related conditions: can change in dietary intake make a difference? *Nutr Clin Pract.* 2008;23:63–71.
- Guzman-Sanchez DA, Ishiuji Y, Patel T, et al. Enhanced skin blood flow and sensitivity to noxious heat stimuli in papulopustular rosacea. J Am Acad Dermatol. 2007; 57(5):800–805.
- Rashid S, Jin Y, Ecoiffier T, et al. Topical omega-3 and omega-6 fatty acids for treatment of dry eye. Arch Ophthalmol. 2008;162(2):219-225. ●

