



# The role of diet in acne: facts and controversies

Batya B. Davidovici, MD\*, Ronni Wolf, MD

*The Dermatology Unit, Kaplan Medical Center, 76100 Rechovot, Israel<sup>1</sup>*

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**Abstract** Acne is the most prevalent skin condition. It has a substantial effect on the quality of life of teenagers worldwide. Among acne patients from different societies and cultures, diet is uniformly regarded as a major cause in the pathogenesis of acne. We reviewed the up-to-date literature regarding acne and culprit foods such as dairy products, chocolate, and fatty foods. Unfortunately, after reviewing the existing data, there are no answers but there are definitely more questions, because there is no clear proof about whether these issues are facts or misconceptions.

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“It is important to look beyond the physical scarring, for there is no disease that has caused more insecurity and feelings of inferiority than acne.”<sup>1</sup>

Acne is the most prevalent skin condition, affecting to some degree 85% of the population aged 11 to 30 years.<sup>2</sup> It is not a life-threatening condition; however, it lasts for years and can cause physical and emotional scars. Despite the high prevalence of acne vulgaris in adolescents, several studies found that overall knowledge about the causes, natural course, and therapy was very low, not only among patients but also among final year medical students<sup>3</sup> and even among family physicians and nurses.<sup>4,5</sup> Therefore, myths, misconceptions, and commonly held beliefs about acne still exist among patients as well as among well-meaning friends and relatives.<sup>6,7</sup>

Many recent studies have evaluated the knowledge, beliefs, and perceptions about acne causation among acne patients and their families. It is interesting that no major differences were noted in the beliefs and perceptions of the

pathogenesis of acne, and the role of diet was uniformly noted among acne patients from different societies and cultures. In a study from Greece among high schools students, 62.3% implicated diet as a cause for their acne.<sup>8</sup> Patients in Canada referred to a community-based dermatologist for management of acne vulgaris completed a self-administered questionnaire. The most common belief was that acne was caused by hormonal and genetic factors, although diet, poor skin hygiene, and infection were also implicated.<sup>7</sup> In another study that analyzed the knowledge of acne causation among English teenagers, 11% of the responders blamed greasy food as the main cause.<sup>9</sup>

That the public perceives the role of food in acne causation as pivotal is not surprising: In the last decade, there had been an enormous increase in public awareness of the cause-and-effect relationship between diet and health. This trend, actively popularized by agents with purely commercial interests and by the media, has drastically changed the perceptions and attitudes of consumers toward the image and importance of the daily diet.

Today, 2000 years after Hippocrates wrote “Let food be your medicine, and let medicine be your food,” one of the most common questions concerning acne is, “Doctor, is it something I ate?” In an attempt to advise patients knowledgeably on the subject of nutrition, one is inundated with

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\* Corresponding author. Tel.: +972 8 9441327; fax: +972 8 9441107.

E-mail address: [bdavidovici@yahoo.com](mailto:bdavidovici@yahoo.com) (B.B. Davidovici).

<sup>1</sup> Affiliated to The School of Medicine, Hebrew University and Hadassah Medical Center, Jerusalem, Israel.

and confused by the mountains of epidemiologic studies that appear in the scientific, pseudoscientific, and nonscientific literature. Unfortunately, convincing trials are lacking, because it turns out that no meta-analyses, randomized controlled clinical studies, or well-designed scientific trials have followed evidence-based guidelines for providing solid proof in dealing with this issue.<sup>10</sup>

## Ecologic studies

An ecologic study is an observational study on risk factors and disease prevalence in which different population groups are compared to identify associations. Because all data are aggregate at the group level, relationships at the individual level cannot be empirically determined. This type of study provides weak empiric evidence. As Sherlock Holmes noted: "Circumstantial evidence is a very tricky thing. It may seem to point very straight to one thing, but if you shift your own point of view a little, you may find it pointing in an equally uncompromising manner to something entirely different."<sup>11</sup>

A difference in the prevalence of acne between non-Westernized and fully modernized societies has been noted, and diet has been suspected to be the reason. Schaefer,<sup>12</sup> a general practitioner who spent almost 30 years treating Inuit (Eskimo) people as they made the transition to modern life, and later Bendiner,<sup>13</sup> reported that acne was absent in the Inuit population when they were still living and eating in their traditional manner. The prevalence of acne became similar to that in Western societies after their acculturation. Surveys of disease in some rural African villages in Kenya,<sup>14</sup> Zambia,<sup>15</sup> and the Bantu in South Africa<sup>16</sup> report far less acne than is found in the descendants of people in these areas who now live in the United Kingdom or the United States. More convincing is the study of schoolchildren from Purus Valley, a rural region in Brazil.<sup>17</sup> Of 9955 children aged 6 to 16 years, only 2.7% had acne vulgaris.

A more recent observational report documents the prevalence of acne in two non-Westernized isolated populations, the Kitavan Islanders of Papua New Guinea, and the Ache hunter-gatherers of Paraguay.<sup>18</sup> The diet of the Kitavan people, as well as the Ache community, includes mainly traditional foods that are locally cultivated. An analysis of 1200 Kitavan individuals, including 300 aged 15 to 25 years, and 115 Ache individuals including 15 aged 15 to 25, found not a single case of acne of any grade. The authors suggested that the absence of acne in non-Westernized societies is attributable to environmental factors, mainly local diets, which have a substantially lower glycemic index than the Western diet. Even they admit that an alternative explanation of the low prevalence of acne in these non-Westernized populations is that of genetic susceptibility to acne, especially given that the people in these isolated regions live in close-knit and closed communities.

In epidemiology it is called the "ecological fallacy," meaning that even if such a link is supported by biologically plausible hypotheses of mechanism of causation, it does not provide proof of a causal relationship, because the individual diet of the individuals who develop acne is not known and confounders cannot be assessed.<sup>19</sup> It can even be postulated for the purpose of discussion that the dietary restrictions that Western adolescents with acne adopt in managing their condition are trivial compared with the differences between their diets and those of hunter-gatherers.

## Acne and dairy products

Could milk cause acne? A 1949 study reported 1925 patients who kept food diaries and found that milk was the most common food implicated in acne flares.<sup>20</sup> A more recent report also supporting an association between milk consumption and acne was based on the Nurses Health Study II cohort.<sup>21</sup> The study revealed that intake of milk during adolescence was associated with history of teenage acne. This association was more marked for skim milk than for other forms of milk, suggesting that the finding is unlikely to be caused by the fat content of milk. The authors hypothesize that this association may be caused by the presence of hormones and bioactive molecules in milk. Acne in teenagers was correlated with hormonal activity.<sup>22</sup>

Milk contains placenta-derived progesterone and other dihydrotestosterone (DHT) precursors, including 5 $\alpha$ -pregnanedione and 5 $\alpha$ -androstanedione. These compounds are only a few enzymatic steps away from DHT, the main acne stimulator, and the enzymes required to mediate the change are present in the human pilosebaceous unit.<sup>23</sup> Milk also contains a multitude of growth-stimulating hormones.<sup>24</sup> The most likely of all candidates for costimulation with the steroid hormones of pilosebaceous function and dysfunction is insulin like growth factor-1 (IGF-1), which is present in ordinary milk. IGF-1 stimulates the synthesis of androgens in the ovary, adrenals, and testicles. Insulin itself and, even more so, IGF-1 have been demonstrated to stimulate hair follicle growth and sebocyte growth.<sup>25,26</sup>

Accordingly, the blood level of IGF-1 in prepubertal, pubertal, adolescent, and early adult humans resembles accurately the prevalence curve of acne in this population. Human and bovine IGF-1 share the same amino acid sequences, and several milk proteins protect IGF-1 from digestion in the gut.<sup>27</sup> Therefore, it is likely that IGF-1 may mediate some of the effects of comedogenic factors, such as androgens, growth hormone, and glucocorticoids.<sup>28</sup>

Although the biologic explanation seems plausible, the study is not innocent from pitfalls in methodology. First, the validity of the data collected by distantly recalled eating habits and vaguely defined disease is questionable. Because the analysis of this study was cross-sectional, a causal relationship cannot be determined. A temporal correlation cannot be established in a cross-sectional study; thus, the

direction of the association between the alleged cause and the effect cannot be defined. As a consequence, a reverse causation cannot be ruled out; therefore, the association between acne and milk found in this study should be treated with caution.

An alternative hypothesis explaining the association between milk and acne suggested that the iodine content of milk might also have an effect in the development of acne.<sup>29</sup> It was claimed that iodine intake could exacerbate acne.<sup>30</sup> The concentration of iodine in milk varies according to the season and geographic location, but significant levels of iodine were found in milk in different countries.<sup>31-33</sup> The observed association of dairy products with acne might be secondary to the iodine content of the dairy products ingested; however, whether iodine in any concentration causes true acne is debatable. Acneiform eruption can be triggered by halides,<sup>34</sup> and iodine was also recognized as causing an acneiform eruption. The comedo, as the initial lesion in acne, is not part of this eruption.

So, can milk cause acne? Our conclusion, on the basis of the existing evidence, is that the association between dietary dairy intake and the pathogenesis of acne is slim.

### Acne, chocolate, and fatty foods

Chocolate, oily or fatty foods, and foods with high sugar content have been repeatedly nominated as causing or exacerbating acne. Is there convincing evidence for such a link? The effect of dietary fat content on insulin resistance has been a subject of controversy. On one hand, animal studies almost uniformly show increases in insulin resistance accompanying high-fat diets, particularly saturated fats<sup>35-40</sup>; however, the results of clinical investigations in humans are much less conclusive. Although some studies indicate a link between dietary fat intake and insulin resistance,<sup>41-44</sup> most show no such relationship.<sup>45-50</sup> The general consensus among the experts today is that the available valid scientific data are insufficient to prove such a correlation.

A very similar situation exists with the influence of carbohydrate contained in foods (glycemic index) and insulin sensitivity. It is proposed that high glycemic indexes lead to hyperinsulinemia and a resulting cascade of endocrine consequences, including increased androgens, increased IGF-1, and altered retinoid signaling pathways, that mediate acne.<sup>18,51</sup>

Several animal studies demonstrated an inverse correlation between a high glycemic index, hyperinsulinemia, and insulin resistance,<sup>52-55</sup> but the few studies on humans have yielded inconsistent results or failed to show such an effect.<sup>56-58</sup> Most experts have taken the position that not enough valid scientific data are available to support such a link.<sup>59</sup> Because it is generally accepted that the severity of acne is correlated with facial sebum secretion, it has been hypothesized that foods high in fat or carbohydrates may exacerbate acne by production of more comedogenic sebum

—by increasing blood lipid levels or by producing sebum that is less fluid,<sup>60</sup> and thence greater obstruction of pilosebaceous follicles—thus setting the stage for follicle rupture and secondary inflammatory changes.

Increased sebaceous output was demonstrated in several experimental studies (most of them dated) in which animals were fed high-fat or high-carbohydrate diets,<sup>61-63</sup> but these studies have been criticized by many experts for using faulty techniques.<sup>62</sup> Several human studies have also demonstrated that diet may change the amount and composition of excreted sebum, that is, an increase in lipid secretion when either excess carbohydrates or fats were given.<sup>64-67</sup> An important study<sup>68</sup> showed that sebaceous glands can and do use fatty acids from the bloodstream for the synthesis of sebum.

So far for the pathogenesis, but can chocolate or oily foods cause or exacerbate acne? Surprisingly few studies have examined the role of these dietary elements in acne. Two studies of acne and chocolate<sup>69,70</sup> have considerable methodologic shortcomings. The researchers performed trials of chocolate, milk, and roasted nuts in university students and found no effect on acne, but the trials were small, uncontrolled, had very short follow-up, and inadequate statistical analysis. The subjective self-assessed measure of global dietary quality in another study<sup>71</sup> renders these findings of limited credibility.

In a methodologically stronger trial,<sup>72</sup> a single-blind, placebo-controlled, crossover study performed in American hospital acne clinic attendees and male prisoners found no effect of chocolate on acne or on sebum production or composition. The placebo bar was of a similar fat and sugar composition to the study chocolate bar. This study might suggest no role for the cocoa content of chocolate bars in acne genesis, but the role of the complete product remains open to question. Furthermore, the treatment period for both chocolate and placebo bars was just 4 weeks. Given the 4-week treatment periods and 3-week washout period in the crossover design, the study duration might have been insufficient to observe the relevant changes.

A small study of 16 patients with acne and 13 matched controls<sup>73</sup> found no difference in sugar consumption between the 2 groups, although patients with seborrheic dermatitis had higher levels of sugar consumption.

No effect was established between acne and chocolate, dairy products, shellfish, or fatty foods in another study.<sup>72</sup> A study with several methodologic limitations, as explained before, found that high-fat and high-carbohydrate foods such as sweets, pizza, and French fries did not cause acne.<sup>21</sup>

### Conclusions

Despite the inundation of epidemiologic studies, mostly of unsatisfactory quality, and the abundance of beliefs and perceptions among acne patients about the role of diet in the pathogenesis of acne,<sup>74</sup> there is a paucity of reliable information. In the few studies that have been undertaken,

no specific foods (including fatty foods and chocolate) have been identified as causative factors.<sup>75,76</sup> Yet, the question of whether common perceptions and beliefs prove to be fact or misconception is more than an academic issue. It is of importance due to the practical implications of these beliefs for acne management.

Alas, after reviewing the relevant published data, we can conclude that insufficient serious effort has been invested in investigating these questions, which are prevalent and consistent across different cultures. At present, we are bereft of reliable answers based on scientific evidence. We hope that the day when we will be able to knowledgeably advise our acne patients on the role of nutrition in relation to acne etiology is not too far away.

## References

- Koo J. The psychosocial impact of acne: patients' perceptions. *J Am Acad Dermatol* 1995;32:S26-30.
- Wood AJ. Drug therapy: therapy for acne vulgaris. *N Engl J Med* 1997;336:1156-62.
- Green J, Sinclair RD. Perceptions of acne vulgaris in final year medical student written examination answers. *Australas J Dermatol* 2001;42:98-101.
- Brajac I, Bilić-Zulle L, Tkalcic M, et al. Acne vulgaris: myths and misconceptions among patients and family physicians. *Patient Educ Couns* 2004;54:21-5.
- Harrison S, Hutton L, Nowak M. An investigation of professional advice advocating therapeutic sun exposure. *Austral New Zealand J Pub Health* 2002;26:108-15.
- Rasmussen JE, Smith SB. Patient concepts and misconceptions about acne. *Arch Dermatol* 1983;119:570-2.
- Tan JK, Vasey K, Fung KY. Beliefs and perceptions of patients with acne. *J Am Acad Dermatol* 2001;44:439-45.
- 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;21:806-10.
- Smithard A, Glazerbrook C, Williams H. Acne prevalence, knowledge about acne and psychological morbidity in mid-adolescence: a community-based study. *Br J Dermatol* 2001;145:274-9.
- Bigby M. Challenges to the hierarchy of evidence: does the emperor have no clothes? *Arch Dermatol* 2001;137:345-6.
- Doyle AC. *The Boscombe Valley Mystery. The Adventures of Sherlock Holmes*. London: Pan Books Ltd; 1981.
- Schaefer O. When the Eskimo comes to town. *Nutr Today* 1971;6:8-16.
- Bendiner E. Disastrous trade-off: Eskimo health for white "civilization". *Hosp Pract* 1974;9:156-89.
- Verhagen A, Koten J, Chaddah V, Patel RI. Skin diseases in Kenya. A clinical and histopathological study of 3,168 patients. *Arch Dermatol* 1968;98:577-86.
- Ratnam A, Jayaraju K. Skin disease in Zambia. *Br J Dermatol* 1979;101:449-53.
- Park R. The age distribution of common skin disorders in the Bantu of Pretoria, Transvaal. *Br J Dermatol* 1968;80:758-61.
- Bechelli L, Haddad N, Pimenta W, et al. Epidemiological survey of skin diseases in schoolchildren living in the Purus Valley (Acre State, Amazonia, Brazil). *Dermatologica* 1981;163:78-93.
- Cordain L, Lindeberg S, Hurtado M, Hill K, Eaton SB, Brand-Miller J. Acne vulgaris. A disease of western civilization. *Arch Dermatol* 2002;138:1584-90.
- Gordis L. *Epidemiology*. 2nd ed. Philadelphia: WB Saunders; 2000.
- Robinson HM. The acne problem. *South Med J* 1949;42:1050-60.
- Adebamawo CA, Spiegelman D, Danby FW, Frazier AL, Willett WC, Holmes MD. High school dietary dairy intake and teenage acne. *J Am Acad Dermatol* 2005;52:207-14.
- Lucky AW. Hormonal correlates of acne and hirsutism. *Am J Med* 1995;98:89S-94S.
- Chen W, Thiboutot D, Zouboulis CC. Cutaneous androgen metabolism: basic research and clinical perspectives. *J Invest Dermatol* 2002;119:992-1007.
- Koldovsky O. Hormones in milk. *Vitam Horm* 1995;50:77-149.
- Rosenfield R. Ovarian and adrenal function in polycystic ovary syndrome. *Endocrinol Metab Clin North Am* 1999;28:265-93.
- Rosenfield R. Polycystic ovary syndrome and insulin resistant hyperinsulinemia. *J Am Acad Dermatol* 2001;45:S95-S104.
- Xian CJ, Shoubridge CA, Read LC. Degradation of IGF-I in the adult rat gastrointestinal tract is limited by a specific antiserum or the dietary protein casein. *J Endocrinol* 1995;146:215-25.
- Deplewski D, Rosenfield RL. Role of hormones in pilosebaceous unit development. *Endocr Rev* 2000;21:363-92.
- Arbesman H. Dairy and acne—the iodine connection. *J Am Acad Dermatol* 2005;53:1102.
- Hitch JM. Acneiform eruptions induced by drugs and chemicals. *JAMA* 1967;200:879-80.
- Dahl L, Opsahl JA, Meltzer HM, Julshamn K. Iodine concentration in Norwegian milk and dairy products. *Br J Nutr* 2003;90:679-85.
- Rasmussen LB, Larsen EH, Ovesen L. Iodine content in drinkingwater and other beverages in Denmark. *Eur J Clin Nutr* 2000;54:57-60.
- Pennington JAT. Iodine concentrations in US milk: variation due to time, season, and region. *J Dairy Sci* 1990;73:3421-7.
- Plewig G, Kligman AM. Acneiform eruptions. In: Plewig G, Kligman AM, editors. *Acne and rosacea*. 2nd ed. Berlin: Springer-Verlag; 1993.
- Dobbins R, Szczepaniak L, Myhill J, et al. The composition of dietary fat directly influences glucose-stimulated insulin secretion in rats. *Diabetes* 2002;51:1825-33.
- Kraegen E, Clark P, Jenkins A, et al. Development muscle insulin resistance after liver insulin resistance high-fat-fed rats. *Diabetes* 1991;40:1397-403.
- Muurling M, Jong M, Mensink R, et al. A low-fat diet has a higher potential than energy restriction to improve high-fat diet-induced insulin resistance in mice. *Metabolism* 2002;51:695-701.
- Roberts C, Vaziri N, Hui Liang K, et al. Reversibility chronic experimental syndrome X by diet modification. *Hypertension* 2001;37:1323-8.
- Storlien L, Pan D, Kriketos A, et al. High-fat diet-induced insulin resistance. Lessons and implications from animal studies. *Ann N Y Acad Sci* 1993;683:82-90.
- Wang Y, Miura Y, Kaneko T, et al. Glucose intolerance induced by a high-fat/low-carbohydrate diet in rats effects of nonesterified fatty acids. *Endocrine* 2002;17:185-91.
- Lovejoy J. The influence of dietary fat on insulin resistance. *Curr Diabetes Rep* 2002;2:435-40.
- Lovejoy J, Champagne C, Smith S, et al. Relationship dietary fat and serum cholesterol ester and phospholipid fatty acids to markers of insulin resistance in men and women with a range of glucose tolerance. *Metabolism* 2001;50:86-92.
- Lovejoy J, Windhauser M, Rood J, et al. Effect of a controlled high-fat versus low-fat diet on insulin sensitivity and leptin levels in African-American and Caucasian women. *Metabolism* 1998;47:1520-4.
- Roth J, Mobarhan S, Clohisy M. The metabolic syndrome: where are we and where do we go? *Nutr Rev* 2002;60:335-7.
- Abbott W, Howard B, Ruotolo G, et al. Energy expenditure in humans: effects of dietary fat and carbohydrate. *Am J Physiol* 1990;258:E347-51.
- Brokman M, Campbell L, Chisholm D, et al. Comparison of the effects on insulin sensitivity of high carbohydrate and high-fat diets in normal subjects. *J Clin Endocrinol Metabol* 1991;72:432-7.

47. Brynes A, Edwards C, Jadhav A, et al. Diet-induced change in fatty acid composition of plasma triacylglycerols is not associated with change in glucagon-like peptide 1 or insulin sensitivity in people with type 2 diabetes. *Am J Clin Nutr* 2000;72:1111-8.
48. Grag A, Grundy S, Unger R. Comparison of effects high-and low-carbohydrate diets on plasma lipoproteins and insulin sensitivity in patients with mild NIDDM. *Diabetes* 1992;41:1278-85.
49. Lovejoy J, Smith S, Champagne C, et al. Effects of diets enriched in saturated (palmitic), monounsaturated (oleic), or trans (elaidic) fatty acids on insulin sensitivity and substrate oxidation in healthy adults. *Diabetes Care* 2002;25:1283-8.
50. Parker D, Weiss S, Triosi R, et al. Relationship of dietary saturated fatty acids and body habitus to serum insulin concentration: the Normative Aging Study. *Am J Clin Nutr* 1993;58:129-36.
51. Thiboutot DM, Strauss JS. Diet and acne revisited [comment]. *Arch Dermatol* 2002;138:1591-2.
52. Byrnes S, Miller J, Denyer G. Amylopectin starch promotes the development of insulin resistance in rats. *J Nutr* 1996;125:1430-7.
53. Higgins J, Brand Miller J, Denyer G. Development of insulin resistance in the rat is dependent on the rate of glucose absorption from the diet. *J Nutr* 1996;126:596-602.
54. Kabir M, Rizkalla S, Champ M, et al. Dietary amylose/amylopectin starch content affects glucose and lipid metabolism in adipocytes of normal and diabetic rats. *J Nutr* 1998;128:35-43.
55. Kabir M, Rizkalla S, Quignard-Boulangé A, et al. A high glycemic index starch diet affects lipid storage-related enzymes in normal and to a lesser extent in diabetic rats. *J Nutr* 1998;128:1878-83.
56. Frost G, Leeds A, Trew G, et al. Insulin sensitivity in women at risk of coronary heart disease and the effect of a low glycemic index diet. *Metabolism* 1998;47:1245-51.
57. Kiens B, Richter E. Types of carbohydrate in an ordinary diet affect insulin action and muscle substrates in humans. *Am J Clin Nutr* 1996;63:47-53.
58. Meyer K, Kushi L, Jacobs D, et al. Carbohydrates, dietary fiber, and incidence of type 2 diabetes in older women. *Am J Clin Nutr* 2000;71:921-30.
59. Pi-Sunyer F. Glycemic index and disease. *Am J Clin Nutr* 2002;76 (suppl):290S-8S.
60. Mackie BS, Mackie LE. Chocolate and acne. *Austral J Dermatol* 1974;15:103-9.
61. Klinge F, Wacker L. Ueber den lipidstoffwechsel und die gewebveränderungen bei mausen und kaninchen unter dem einfluss von fett, cholesterin, und scharlachrotfütterung. *Krankheitsforschung* 1925;1: 257-85.
62. Somekawa E. On the production of seborrhea in the rat by feeding with whale oil. *Sci Papers Inst Phys Chem Res* 1947;42:72-9.
63. Suzuki S. Zur physiologie und pathologie der talgsekretion, besonders by lues. *Jpn J Derm Unol* 1936;40:203-13.
64. Kuznitsky E. Experimentelle und klinische feitrage zur frage der hauttalgsekretion. *Arch Dermatol Syphilol* 1913;114:1913-8.
65. MacDonald I. Effects of a skimmed milk and chocolate diet on serum and skin lipids. *J Sci Food Agr* 1968;19:270-2.
66. Pochi P, Downing D, Strauss J. Sebaceous gland response in man to prolonged total caloric deprivation. *J Invest Dermatol* 1970;55:303-9.
67. Serrati B. Influenza del sistema nervoso sulla secrezione sebacea: osservazioni e ricerche cliniche. *Riv Pat Nerv* 1938;52:377-423.
68. Pappas A, Anthonavage M, Gordon J. Metabolic fate and selective utilization of major fatty acids in human sebaceous gland. *J Invest Dermatol* 2002;118:164-71.
69. Grant JD, Anderson PC. Chocolate and acne: a dissenting view. *Mo Med* 1965;62:459-60.
70. Anderson PC. Foods as the cause of acne. *Am Fam Phys* 1971;3:102-3.
71. Chiu AC, Chon SY, Kimball AB. The response of skin disease to stress: changes in the severity of acne vulgaris as affected by examination stress. *Arch Dermatol* 2003;139:897-900.
72. Fulton Jr JE, Plewig G, Kligman AM. Effect of chocolate on acne vulgaris. *JAMA* 1969;210:2071-4.
73. Bett DG, Morland J, Yudkin J. Sugar consumption in acne vulgaris and seborrhoeic dermatitis. *Br Med J* 1967;3:153-5.
74. Purdy S, Langston J, Tait L. Presentation and management of acne in primary care: a retrospective cohort study. *Br J Gen Pract* 2003;53: 525-9.
75. Goulden V, Stables GI, Cunliffe WJ. Prevalence of facial acne in adults. *J Am Acad Dermatol* 1999;41:577-80.
76. Wolf R, Matz H, Orion E. Acne and diet. *Clin Dermatol* 2004;22: 387-93.