



Role of Diet in Acne

Essay

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List of Abbreviations

AR	Androgen receptor
BTC	Betacellulin
13-cis-RA	13-cis-retinoic acid
DHEA	Dehydroepiandrosterone
DHEA-S	Dehydroepiandrosterone sulphate
DHT	Dihydrotestosterone
EGCG	Epigallocatechin-3-gallate
EGF	Epidermal growth factor
EGFR	Epidermal growth factor receptor
EPA	Eicosapentaenoic acid
FoxO1	Forkhead box protein O1
GH	Growth hormone
GI	Glycemic index
HB-EGF	Heparin binding epidermal growth factor
IGF-1	Insulinlike growth factor 1
IGF1R	Insulin like growth factor-1 receptor
IGF-2	Insulinlike growth factor 2
IGFBP	Insulin like growth factor binding protein
IGFBP-3	Insulin like growth factor binding protein-3
IR-A	Insulin receptor-A
IR-B	Insulin receptor-B
L.acidophilus	Lactobacillus acidophilus
LGL	Low glycemic load
LPS	lipopolysaccharide
LTB4	Leukotriene B4

MUFAs	Monounsaturated fatty acids
NADPH	Nicotinamide Adenine Dinucleotide Phosphate Hydrogen
P. acnes	Propionibacterium acnes
PI3K	Phosphoinositide-3 kinase
PS	Phytosphingosine
SFAs	Saturated fatty acids
SHBG	Sex hormone-binding globulin
SIBO	Small intestinal bacterial over growth
TGF-beta1	Transforming growth factor-beta 1
TGF-α	Transforming growth factor alpha

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INTRODUCTION

Acne vulgaris is a common skin disease, affecting more than 85% of adolescents, women being affected more frequently than men (*Collier et al., 2007*). It is seen in nearly 100% of individuals at some time during their lives (*Sandra et al., 2008*). For some, it is temporary and resolves by the mid-20s; however, more severe cases often take longer to resolve, and it can persist into adult years in as many as 50% of individuals (*Sandra et al., 2008; Thiboutot et al., 2009*). Although it does not affect overall health, its impact on emotional well-being and function can be critical, especially active acne and its sequel, like permanent scarring, leaves psychological stress that do not always correlate with the clinician's assessment of severity at one point in time (*Katsambas et al., 2004; Munavalli and Weiss, 2008*). The psychological fallout in acne patients includes much higher rates of clinical depression, anxiety, anger, suicidal thoughts, and even suicide itself (*Katzman and Logan, 2007*). Increasing age of affliction with acne can proportionately affect the quality of life in various ways, including employment, social behavior, and body dissatisfaction (*Munavalli and Weiss, 2008*). The more severe the acne, the greater the negative impact on quality of life (*Sandra et al., 2008*).

Acne is often a chronic disease and not just a self-limiting disorder of teenagers. The following characteristics have been used to define its chronic state: a prolonged course, a pattern of recurrence or relapse, manifestation as acute outbreaks or slow onset, and a psychological and social impact that affects the individual's quality of life. Factors that have been linked to a chronic course include stress-related production of

adrenal androgens, *Propionibacterium acnes* (*P. acnes*) colonization, and familial background (*Thiboutot et al., 2009*).

The clinical features of acne include seborrhoea (excess grease), non-inflammatory lesions (open and closed comedones), inflammatory lesions (papules and pustules), and various degrees of scarring (Fig .1). The distribution of acne corresponds to the highest density of pilosebaceous units (face, neck, upper chest, shoulders, and back). Nodules and cysts comprise severe nodulocystic acne (*Jacob et al., 2001; Meixner et al., 2008; Shalita, 2004*).

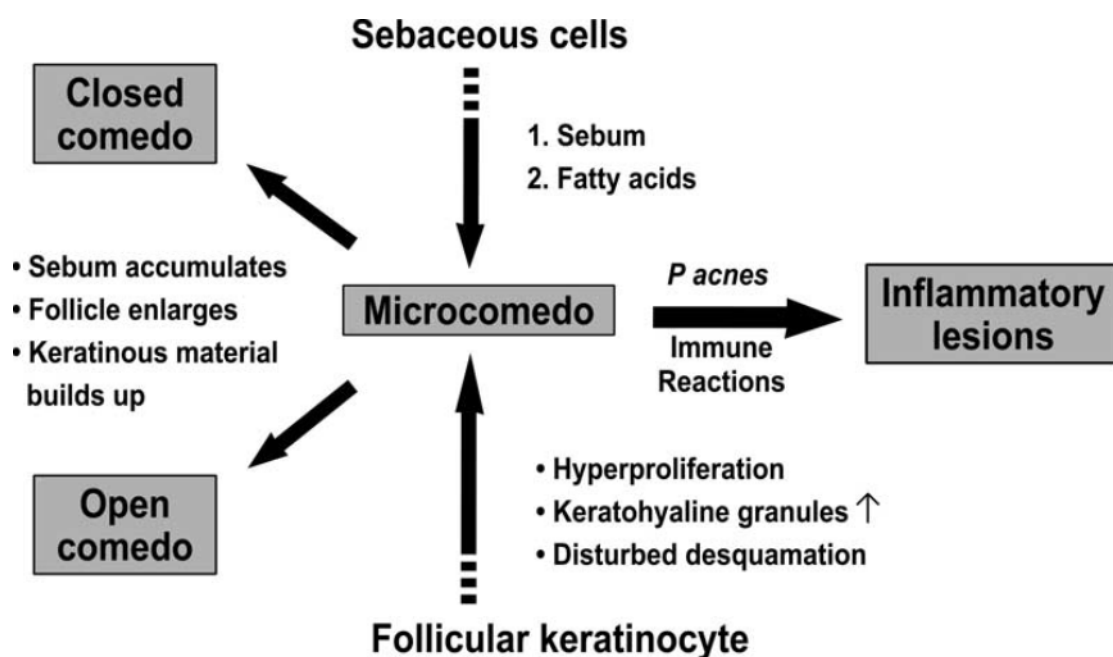


Fig. (1): Lesion progression in acne (*Gollnick et al., 2003*)

Acne vulgaris is a chronic dermatosis. It is a disease of the pilosebaceous follicle with four fundamental etiopathogenic factors: sebaceous hyperproduction, follicular hyperkeratinization, increase of

P. acnes colonization, and periglandular dermal inflammation. It affects all races, although it is less intense in Asians and Blacks (**Steiner, 2002; Steiner et al., 2003**). Its overall prevalence varies between 35% and 90% in adolescents; In the West, it affects 79 to 95 % of adolescents (**Cordain et al., 2002**). Acne is a disease that is highly influenced by genetics since follicular hyperkeratinization and sebaceous secretion are under hormonal control (**Sobral Filho et al., 1997**).

For many decades, researchers have tried to prove an old theory, surrounded by myths and popular beliefs: the association between acne and diet (**Anderson, 1971; Cordain, 2005**). Dietary factors have long been implicated in the pathogenesis of acne. It is well known that increased sebum production plays a fundamental role in acne, and evidence suggests that dietary manipulation alters sebaceous gland output. Extreme caloric restriction dramatically decreases the sebum excretion rate, and these changes can be reversed when a normal diet is resumed; also, increased consumption of dietary fat or carbohydrate increases sebum production, and modifications in the type of carbohydrate intake in diet can also alter sebum composition (**Rigopoulos et al., 2007**).

Aim of the essay

The aim of the essay is to clarify the possible relation between diet and acne.

Diet and Acne

Before the 1960s, dietary advice was a standard part of acne therapy. Data published in 1931 suggested that impaired glucose tolerance existed in patients with acne (*Campbell, 1931*). Abnormal carbohydrate metabolism was implicated in acne and patients were recommended to avoid consuming excessive carbohydrates and high-sugar foods (*Belisario, 1951*).

Nutrition and diet are affecting overall health; that statement needs no particular citation as every nutritional textbook advocates for this. But can diet affect acne? (*Thiboutot, 2008*). The current status of the relationship of diet and acne is not clear and under debate. On the one hand, the American Academy of Dermatology published recommendations in 2007 suggesting that caloric restriction has no benefit in the treatment of acne and that there is insufficient evidence to link the consumption of certain “food enemies” to acne (*Strauss et al., 2007*). On the other hand, recent studies have suggested a rather close relationship between diet and acne (*Smith et al., 2007a; Smith et al., 2008b*).

Although controversial, certain dietary factors have been implicated as causative agents in disease progression. It is a common perception among acne patients that foods such as sweets, chocolate, and nuts, as well as fried and oily foods, will exacerbate the disease (*Green and Sinclair, 2001; El-Akawi et al., 2006b*). Many studies have been performed to determine the true effect of diet on acne; however, the results have been largely inconclusive (Table 1) (*Veith and Silverberg, 2011*). Although the majority of evidence supporting a link between diet and acne falls under dairy products category and carbohydrates category,

a brief discussion of other dietary factors implicated in acne including omega-3 fatty acids, antioxidants, zinc, vitamins, and iodine follows (*Bowe et al., 2010*).

Bett et al., (1967) provided evidence that sugar consumption did not significantly increase the incidence of acne in 1967. However, more recent studies demonstrated that high glycemic index and/or glycemic load foods (e.g., refined sugars, white carbohydrates) specifically increased the risk for acne (*Cordain et al., 2002; Smith et al., 2007a; Smith et al., 2007b; Smith et al., 2008a; Smith et al., 2008b; Jung et al., 2010*), which was strengthened by the results of the study published by *Cordain et al., (2002)* that showed the absence of acne in individuals from Papua New Guinea and Paraguay; individuals residing in these areas are devoid of a high glycemic load in their diet.

A prospective cohort study showed no difference overall in glycemic load, dietary glycemic index, leptin levels, serum fasting glucose, or insulinlike growth factor 1 (IGF-1) (a marker of acute insulinemia) between acne patients and controls (*Kaymak et al., 2007*). However, between 2007 and 2008, Smith et al., conducted 4 studies that clearly correlated the consumption of a high glycemic load diet to the prevalence of acne (*Smith et al., 2007a; Smith et al., 2007b; Smith et al., 2008a; Smith et al., 2008b*). Most notably, *Smith et al., (2007a)* showed that patients in a 12-week interventional trial of low glycemic index diet had fewer acne lesions. Additional studies with broader foci also have shown a correlation in the consumption of sweets and acne (*Halvorsen et al., 2009; Law et al., 2010*).

Table (1): Studies Addressing Diet and Acne Vulgaris (a) (Veith and Silverberg, 2011)

Reference	Year	Participants, n	Study Design(b)	Age Range (Average) of Evaluated Patients	Acne Contributing Factor(s) Studied	Conclusions
Jung et al	2010	1285	Prospective	N/A	Dairy, high-fat diet, high glycemic load	Junk food, carbonated drinks, fried chicken, roast pork, and nuts aggravate acne
Yang et al	2010	627	Prospective	14–40 y (27.8 y)	Obesity	Obese women have higher serum testosterone levels but less acne
Kim et al	2010	36	Interventional with placebo control	18–30 y (22.7 y)	Dairy	Lactoferrin-enriched milk may decrease acne inflammation vs fermented milk
Wei et al	2010	5696	Retrospective	17–25 y (21.56 y)	High-fat diet	Fried food is correlated with an increase in acne
Law et al	2010	322	Retrospective	17.4–20.8 y (19.1 y)	Dairy avoidance; high glycemic load	For a subset of patients, desserts and fruit juices are associated with acne, and milk is protective; overall, no specific dietary factor is associated with acne risk
Ghodsi et al	2009	1002	Retrospective	12–20 y (16 y)	Oily foods, sweet foods	Strong correlation exists between consumption of sweets, oily foods, and acne
Halvorsen et al	2009	3775	Retrospective	18–19 y (N/A)	Chocolate, oily foods, sweet foods	In adolescent boys, increased consumption of potato chips, chocolate, and sweets is associated with acne; in adolescent girls, the only dietary association is a lack of vegetables
Abulnaja	2009	60	Retrospective	16–22 y (18 y for obese with acne; 20 y for obese without acne; 17 y for not obese with acne; 18 y for not obese without acne)	Obesity (BMI, more than 27)	Higher serum triglycerides were found in obese patients with acne
Smith et al	2008a	12	Pilot	15–20 y (16.6 y for low glycemic load diet; 17.6 y for high glycemic load diet)	Glycemic load	High glycemic load diets are associated with a reduced serum hormone-binding globulin and increased IGF

Table (1): Studies Addressing Diet and Acne Vulgaris (a) (Veith and Silverberg, 2011)

Reference	Year	Participants, n	Study Design(b)	Age Range (Average) of Evaluated Patients	Acne Contributing Factor(s) Studied	Conclusions
Adebamowo et al	2008	4273	Retrospective	9–15 y (144 mo without acne; 141 mo with acne)	Dairy	Skim milk is associated with acne in teenaged boys
Smith et al	2008b	43	Interventional	15–25 y (N/A)	Glycemic load	Low glycemic load diet increased the ratio of saturated to monosaturated fatty acids on the skin, which was correlated with fewer acne lesions
Kaymak et al	2007	91	Prospective	19–34 y (21.71 y for acne; 22.14 y for controls)	Glycemic load, overall glycemic index	Dietary glycemic index, glycemic load, serum testing, leptin, glucose, and IGF-1 did not differ significantly between acne patients and controls
Smith et al	2007b	31	Interventional	15–25 y (18.2 y for low glycemic load; 18.5 y for controls)	Glycemic load	Low glycemic load and high protein diet causes further clinical improvement of acne than a high glycemic load diet
Smith et al	2007a	43	Interventional	15–25 y (18.2 y for low glycemic index; 18.5 y for controls)	Glycemic load	Low glycemic index diet associated with clinical improvement in acne and increased insulin sensitivity
Adebamowo et al	2006	6094	Prospective	9–15 y	Dairy	Greater levels of milk consumption are associated with acne in adolescent girls
Adebamowo et al	2005	47.355	Interventional	25–42 y (N/A)	Dairy	Total milk and skim milk consumption is associated with an increase in acne in women
Cordain et al	2002	300	Prospective	15–25 y (N/A)	Glycemic load	Low glycemic load diet of Kitavan Islanders of Papua New Guinea and Aché hunter-gatherers of Paraguay is correlated to the absence of acne in all ages
Yeh	1975	568	Retrospective	14–16 y (N/A)	Fatty food, fried food	Peanuts and fried food are associated with acne risk
Fulton et al	1969	65	Interventional	N/A	Chocolate	No correlation was found between consumption of chocolate and acne development