

The Impact of Family History, Dietary Factors, Body Mass Index, and Menstrual History on Severity of Acne Vulgaris Among Females

Prof. Dr. Fadia Abdelhakim Sorour¹, Prof. Dr. Hamed Mohamed Abdo¹, Prof. Dr. Amal Mohamed Mahmoud Aldinary² and Yosra Gamal Hasanain¹

¹Department of Dermatology and Venereology, Faculty of Medicine - Al-Azhar University, Egypt.

²Department of Community Medicine, Faculty of Medicine, Al-Azhar University, Egypt.

yosralgamal@gmail.com

Abstract: Acne is one of the most common skin disorders worldwide and occurs mostly at puberty with a prevalence of almost 95%. Although acne is principally a disorder of adolescence, the prevalence of adult patients with acne is increasing. The impact of acne to a patient's social and psychological state can be major and leads to a reduced quality of life. The prevalence of acne varies in different countries and over time, and it has been postulated that different lifestyles may influence acne prevalence. Along this line, a few studies reported that acne was virtually absent in non-Westernized countries in those living and eating in their traditional manner, whereas acne began to appear when these populations changed their eating habits to those more similar to Western populations. So we conducted case-control study to assess the impact of family history, body mass index, dietary factors, and menstrual history on severity of acne vulgaris in females aged 15 to 30 years. Our study confirms the important role of a family history on the risk of moderate to severe acne, and suggests that lower BMI values, may have a protective effect. Diet rich in sugar or whole milk influenced the risk of moderate to severe acne irrespective of family history and BMI. Finally, our study points to a diet rich in fish and proteins as being protective against acne. The influence of dietetic and environmental factors in acne should be further explored.

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1. Introduction

Acne vulgaris is ranked as the 8th most common disease worldwide, with an estimated prevalence of 9.4% globally (Vos *et al.*, 2012). It affects individuals of most ages; however, the prevalence shows a significant peak in adolescence (Schafer *et al.*, 2001). Research estimates that between 35% and 90% of adolescents have acne to some extent (Stathakis *et al.*, 1997), and moderate to severe acne is found in 10–20% of young people (Bhate *et al.*, 2013).

The impact of acne to a patient's life can be major. It leads to a reduced quality of life comparable to chronic conditions such as asthma, diabetes, back pain and arthritis (Mallon *et al.*, 1999), and is associated with social and psychological problems (Halvorsen *et al.*, 2011). As with other major chronic conditions, acne constitutes a great economic burden to both the patient and to the society (Bickers *et al.*, 2006).

Considering the extensiveness of acne, a great interest exists to reveal and comprehend its possible causative factors. Much is known about the pathophysiology of acne that eventually leads to a chronic inflammation in pilosebaceous units (Webster 2005). What provokes these events to happen, however, is not fully understood.

During the last decade, the acne-diet connection has been brought back to credibility, after being considered a myth for a long time. This hypothesis suggests that consumption of different foods influence the occurrence of acne. In 2002, Cordain *et al.* directed the focus to ethnical differences in acne prevalence and the possible impact of the Westernized diet. Several studies published since then have demonstrated links between dietary elements and acne, and the research supporting an association between milk and acne show some of the more consisting and convincing results (Burriss *et al.*, 2013).

To analyze the role played by different factors, including family history, dietary factors, body mass index, and menstrual history in acne development, we conducted a case-control study in young female patients with a diagnosis of mild to severe acne versus control subjects with no or minimal acne at several dermatologic outpatient clinics in Egypt.

2. Patients and Methods

The present study included 300 female, their ages ranged from 15 to 30 years divided into two groups 150 patients with acne vulgaris mild, moderate or severe, and 150 control subject with no acne. Patients were collected from the outpatient Dermatology clinic

at ABO-ALNOMROS General Hospital during the period from September 2014 to May 2016. Patients were the cases of acne vulgaris either mild, moderate or severe without any other dermatological diseases.

Methods:

All patients were subjected to *a*) Standardized questionnaire to collect information about general socio-demographic characteristics (regarding the age, marital status and education), family History of acne among first degree relatives, food frequency to record information about the intake of selected food items including the usual number of portions per week, and menstrual history (regarding the age at menarche, use of oral contraceptive pills, average length of menstrual cycles and regularity of menstrual cycles), *b*) Anthropometric measures, including height and weight. Body mass index (BMI) was calculated as weight (kg) / height (m²) (WHO, 1995), *c*) Examination of acne lesions in patients with grading of severity, acne severity was defined according to a global score. Four categories were considered as follows: (1) no or minimal acne lesions, where only a few comedones are present; (2) mild acne, where

lesions include several non-inflammatory comedones with less than 10 inflammatory lesions; (3) moderate acne with many comedones, papules, pustules, but no nodules; and (4) severe acne with inflammatory nodules in addition to papules and pustules (Burton *et al.*, 1971).

3. Results

Table I shows the distribution of patients according to severity of acne. It shows that 40% of patients have mild acne, 42.7% of patients have moderate acne and 17.3% of patients have severe acne.

Table I: Severity of Acne among the Studied Patients.

	No(150)	%
Severity of Acne:		
Mild	60	40.0%
Moderate	64	42.7%
Severe	26	17.3%

Table II: Family History of Acne among the Studied Sample.

Groups	Cases (150)	Controls (150)	Significant Test	P value
Family history				
Family history of acne:			Chi square= 27.58	0.000*
Yes	116(77.3%)	72(48.0%)		
No	34(22.7%)	78(52.0%)		
Number of first degree relatives	6.92± 2.30	7.41± 2.57	Student t test t= 1.72	0.086
Affected members in first degree relatives:	116	72		
No affection				
o One	26(22.4%)	42(58.3%)	Chi square= 26.34	0.000*
o Two	47(40.5%)	20(27.8%)		
o Three+	43(37.1%)	10(9.13%)		

*significant difference (p value <0.05).

Table III: Odds Ratio of Family History of Acne as Factors affecting Presence of Acne by Comparing Cases to Control.

	Odds Ratio	Confidence Interval	P value
Family history of acne			
Yes/No	3.70	2.18- 6.28	0.000*

*significant (p value <0.05).

Table II, and table III show the distribution of patients and control subjects according to family history of acne in first degree relatives. Family history of acne in first-degree relatives has significant role in increasing the risk of acne as compared with the control subjects (OR 3.7 and CI 2.18- 6.28).

Table IV shows the distribution of patients and control subjects according to food consumption. We noticed that increased consumption of macaroni (OR

5.98, CI 2.56-14.00), milk (OR 1.76, CI 1.08- 2.86) and potato chips (OR 2.27, CI 1.35- 3.84) have significant role in increasing the risk of acne, and increased consumption of fish (OR 0.61, CI 1.08- 2.86), fresh vegetables (OR 0.37, CI 0.35- 0.97), red meat (OR 0.47, CI 0.24- 0.93), legumes (OR 0.49, CI 0.29- 0.82) and nuts (OR 0.53, CI 0.31- 0.90) have significant role in decreasing the risk of acne. We noticed that food has minimal effect on severity of

acne. No marked association between acne and other dietary items emerged.

Table IV: Odds Ratio of Food Items as Factors Affecting Presence of Acne by Comparing Cases to Control.

Groups Food Items	Odds Ratio	Confidence interval	P value
Macaroni and Bread: (>3 times / ≤ 3 times and rarely)	5.98	2.56-14.00	0.000*
Fresh vegetables and fruits: (>3 times / ≤ 3 times and rarely)	0.37	0.35- 0.97	0.015*
Legumes: (>3 times / ≤ 3 times and rarely)	0.49	0.29- 0.82	0.003*
Milk: (>3 times / ≤ 3 times and rarely)	1.76	1.08- 2.86	0.015*
Yogurt: (>3 times / ≤ 3 times and rarely)	0.60	0.29- 1.23	0.130
Fish: (>3 times / ≤ 3 times and rarely)	0.61	0.37- 1.03	0.048*
Chicken: (>3 times / ≤ 3 times and rarely)	0.60	0.35- 1.04	0.051
Red meat: (>3 times / ≤ 3 times and rarely)	0.47	0.24- 0.93	0.019*
Processed meat: (>3 times / ≤ 3 times and rarely)	1.65	0.84- 3.25	0.115
Cake/ oriental sweets: (>3 times / ≤ 3 times and rarely)	1.13	0.63- 2.02	0.674
Chocolate: (>3 times / ≤ 3 times and rarely)	1.56	0.96- 2.56	0.059
Potato Chips/snacks: (>3 times / ≤ 3 times and rarely)	2.27	1.35- 3.84	0.000*
Nuts/peanuts: (>3 times / ≤ 3 times and rarely)	0.53	0.31- 0.90	0.012*

*significant difference (p value <0.05).

Table V: Menstrual History and BMI and Severity of Acne among the Studied Cases

Groups BMI and Menstrual history	Mild(60)	Moderate (64)	Severe (26)	Significant Test ANOVA test	P value
Body mass index (mean ± SD)	24.75±3.83	26.97±6.41	27.33±3.18	ANOVA testF= 3.88	0.023*
Age at menarche	13.00±1.36	12.87±1.17	12.4615± 1.30	ANOVA testF=1.63	0.199
Menstrual period (mean ± SD)	5.53±1.40	5.56±1.28	6.53±1.67	ANOVA testF= 5.32	0.006*
Regular Menstrual Cycle:					
yes	52(86.7%)	42(65.6%)	16(61.5%)	Chi square=9.24	0.010*
no	8(13.3%)	22(34.4%)	10(38.5%)		
OCPs:				Chi square=	
yes	14(23.3%)	14(21.9%)	5(19.2%)	0.18	0.914
no	46(76.7%)	50(78.1%)	21(80.8%)		

*significant difference (p value <0.05).

In table V, and table VI, we found great correlation between higher BMI and increased risk of presence of acne (OR 1.77, CI 1.09- 2.87) and severity of acne as the mean BMI of cases with mild acne was (24.75±3.83), moderate acne was (26.97±6.41) and severe acne was (27.33±3.18).

Table V, and table VI also show minimal correlation between duration of menstrual period and

risk of acne (OR 0.54, CI 0.33- 0.89) and risk of increased severity as the mean duration of menstrual period of cases with mild acne was (5.53±1.40), moderate acne was (5.56±1.28) and severe acne was (6.53 ± 1.67). Also there is correlation between irregularity of menstrual period and increased risk of presence of acne (OR 0.54, CI 0.33- 0.89) and severity of acne as percentage of cases with irregular period

and mild acne was 13.3%, moderate acne was 34.4% and severe acne was 38.5%. No marked association

was noticed between acne and other menstrual items emerged.

Table VI: Odds Ratio of BMI and Menstrual History as Factors Affecting Presence of Acne by Comparing Cases to Control.

Groups	Odds Ratio	Confidence Interval	P value
BMI ≥25/ <29.9	1.77	1.09- 2.87	0.014*
Age of menarche ≤12/≥13	1.57	0.94-2.62	0.066
Menstrual Period ≥6days/≤5days	0.54	0.33- 0.89	0.010*
Regularity of menstrual cycle Irregular/regular	2.84	1.47- 5.56	0.000*

*significant (p value <0.05).

4. Discussion

The general results of this study confirm that +ve family history increases the risk of acne and suggest that BMI and a diet rich in sugar and milk and poor in protein may influence the risk of the disease. No association emerged between intake of other food items and risk of acne. Likewise, we find minimal correlation between duration of menstrual period and risk of presence and severity of acne, also there is minimal correlation between irregularity of menstrual cycle and increased risk of presence and severity of acne.

In our study, a family history of acne was the strongest predictor of moderate to severe acne. Familial and twin studies provide supporting evidence that genetic factors play a major role in determining susceptibility to acne (Ballanger *et al.*, 2006). Genetic modeling using acne scoring in a large twin study, limited to women, conducted in the United Kingdom showed that 81% of the variance of acne was attributable to genetic effects and only the remaining 19% was attributed to unshared environmental factors (Bataille *et al.*, 2002). Results from other twin studies of acne reported heritability estimates between 50% and 90% (Kirk *et al.*, 2001).

It was postulated that obesity may be accompanied by peripheral hyperandrogenism, which may be associated with increased sebum production and the development of severe acne. At variance with these results, a study conducted in the United Kingdom within the Glasgow Alumni Cohort failed to document any association between acne and BMI (Galobard *et al.*, 2005).

In our study, data indicate a main protective effect on the development of acne from lower BMI values and a lack of a clear dose-response relationship. The same results was reached by Di Landro *et al* (2012) in case-control study of risk factors for acne in people aged 10 to 24 years.

In 2012, Di Landro *et al* concluded that diet high in milk but not in cheese was associated with the risk of acne. This result is consistent with those reported in two separate large cohort studies performed in teenagers in the United States. In these studies, Adebamowo *et al* (2005 and 2008) found a positive association between milk intake during adolescence and a history of acne. In our study, most of the cases drink whole milk, so we can't compare between the effect of skimmed milk and whole milk on aggravation of acne, but we find significant correlation between milk and increased risk of acne.

At variance with a long-lasting belief, we found no association between acne and chocolate consumption in our study. On the contrary, In 2016 Gregory *et al* made single-blind randomized crossover study and found that chocolate consumption increase in acne lesions.

In 2007 Smith *et al* showed an association between acne and a high-glycemic-load diet, with improvement of symptoms after changing to a higher protein, lower-glycemic-load diet. It was postulated that Western diets characterized by a high glycemic index could lead to hyperinsulinemia, triggering a hormonal response capable of promoting unregulated tissue growth and enhanced androgen synthesis. In our study, weekly consumption of cakes/sweets and chocolate was not associated with a higher risk of acne. These results are supported by De Landro *et al* (2012) in case-control study of risk factors for acne in people aged 10 to 24 years.

An interesting result of our study is the inverse relationship between severity of acne and the intake of fish. To our knowledge, such an association was previously reported in a case-control study from Korea (Jung *et al.*, 2010). In that study, fish intake, namely, white fish and green fish or blue tuna, was significantly higher in the control group than in the acne group. Another case-control study by De Landro *et al* (2012) supports the results we reached.

Hormonal factors have commonly been associated with acne, but only a few studies have analyzed the role of menstrual history on the risk of the condition. We found notable correlation between duration and regularity of menstrual period and acne. No significant relationship between age at menarche, or use of oral contraceptives and acne. These results are supported by De Landro *et al* (2012) in case-control study. Oral contraceptives have been linked, although not consistently, with a reduced prevalence of acne (Jemec *et al.*, 2002).

References

1. Adebamowo CA, Spiegelman D, Danby FW, et al. High school dietary dairy intake and teenage acne. *J Am Acad Dermatol*. 2005; 52:207–14.
2. Adebamowo CA, Spiegelman D, Berkey CS, et al. Milk consumption and acne in teenaged boys. *J Am Acad Dermatol* 2008;58:787-93.
3. Ballanger F, Baudry P, N'Guyen JM et al. Heredity: a prognostic factor for acne. *Dermatology* 2006; 212:145–9.
4. Bataille V, Snieder H, MacGregor AJ et al. The influence of genetics and environmental factors in the pathogenesis of acne: a twin study of acne in women. *J Invest Dermatol* 2002; 119:1317–22.
5. Bhate K, Williams HC. Epidemiology of acne vulgaris. *Br J Dermatol* 2013; 168: 474–485.
6. Bickers DR, Lim HW, Margolis D et al. The burden of skin diseases: 2004 a joint project of the American Academy of Dermatology Association and the Society for Investigative Dermatology. *J Am Acad Dermatol* 2006; 55: 490–500.
7. Burriss J, Rietkerk W, Woolf K. Acne: the role of medical nutrition therapy. *J Acad Nutr Diet* 2013; 113: 416–430.
8. Burton JL, Cunliffe WJ, Stafford I, Shuster S. The prevalence of acne in adolescence. *Br J Dermatol* 1971;85:119-26.
9. Cordain L, Lindeberg S, Hurtado M, et al. Acne vulgaris: a disease of Western civilization. *Arch Dermatol* 2002; 138: 1584–1590.
10. Di Landro A, Cazzaniga S, Parazzini F, et al. Family history, body mass index, selected dietary factors, menstrual history, and risk of moderate to severe acne in adolescents and young adults. *J Am Acad Dermatol* 2012; 67: 1129–1135.
11. Galobardes B, Davey SG, Jeffreys M, et al. Acne in adolescence and cause-specific mortality: lower coronary heart disease but higher prostate cancer mortality; the Glasgow alumni cohort study. *Am J Epidemiol* 2005;161: 1094-101.
12. Halvorsen JA, Stern RS, Dalgard F, Thoresen M, Bjertness E, Lien L. Suicidal ideation, mental health problems, and social impairment are increased in adolescents with acne: a population-based study. *J Invest Dermatol* 2011; 131: 363–370.
13. Jemec GB, Linneberg A, Nielsen NH, et al. Have oral contraceptives reduced the prevalence of acne? A population-based study of acne vulgaris, tobacco smoking and oral contraceptives. *Dermatology* 2002;204:179-84.
14. Jung JY, Yoon MY, Min SU, Hong JS, Choi YS, Suh DH. The influence of dietary patterns on acne vulgaris in Koreans. *Eur J Dermatol* 2010;20:768-72.
15. Kirk KM, Evans DM, Farthing B, Martin NG. Genetic and environmental influences on acne in adolescent twins. *Twin Res* 2001;4:190.
16. Mallon E, Newton JN, Klassen A, Stewart-Brown SL, Ryan TJ, Finlay AY. The quality of life in acne: a comparison with general medical conditions using generic questionnaires. *Br J Dermatol* 1999; 140: 672–676.
17. Schafer T, Nienhaus A, Vieluf D, Berger J, Ring J. Epidemiology of acne in the general population: the risk of smoking. *Br J Dermatol* 2001; 145: 100–104.
18. Smith RN, Mann NJ, Braue A, et al. A low-glycemic-load diet improves symptoms in acne vulgaris patients: a randomized controlled trial. *Am J Clin Nutr* 2007;86: 107-15.
19. Stathakis V, Kilkenny M, Marks R. Descriptive epidemiology of acne vulgaris in the community. *Australas J Dermatol* 1997; 38: 115–123.
20. Vos T, Flaxman AD, Naghavi M et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; 380: 2163–2196.
21. Webster GF. The pathophysiology of acne. *Cutis* 2005; 76(2 Suppl): 4–7.
22. WHO Physical status: the use and interpretation of anthropometry. Report of a WHO expert committee. Technical report series No. 854. Geneva: WHO 1995.