

*Evaluation of the therapeutic diet for enhancement of acne status*  
*Tarek M. Afifi, Aml A. El-Ashmawy Fakharanyand Nadia E. El-*

---

## **Evaluation of the therapeutic diet for enhancement of acne status**

**Tarek M. Afifi\*, Aml A. El-Ashmawy\*\* and Nadia E. El-Fakharany\***

\*Department of Nutrition and Food Sciences, Faculty of Home Economics, Menoufia University, Shubin El- Kom, Egypt.

\*\* Department of Dermatology & Venerology, Faculty of Medicine, Tanta University, Tanta, Egypt.

### **ABSTRACT**

**M**edical nutrition therapy was as a potential treatment for acne because it play very important role in control on condition. The objective of this study was to identify the role of diet on pathogenesis and enhancement of acne cases. The study included 30 patients with mean age (12-24 y) with two degrees of acne (mild and sever) .they were divided into three groups every group included 10 patients (5 males and 5 females) The first group treated by drug, second group treated by experimental diet and third group treated by both of drug and experimental diet .The study began in the period from April 2015 to December 2016. Our results showed that correlation coefficient food intake with biochemical analysis in the studied groups .There relationship between deficiency vitamins with deficiency GSH and increased androgens hormones .also, there was correlated between deficiency zinc, calcium, magnesium and vitamins with increased cholesterol, triglycerides, and androgen hormones .also, there was high significant correlation positive between deficiency ca, mg zinc intake with decrease GST. Conclusion, macronutrients (carbohydrates, proteins, fiber and fats) and micronutrients (vitamins and minerals essential from a nutritional point of view) preserve the skin from worsening acne and improve its appearance.

**Key words:** *acne, nutrition therapy, diet, dairy, carbohydrate glycemic*

## **INTRODUCTION**

Acne is the most common disease of the skin that affects individuals in all ages (Wolf et al., 2004). Teenagers are the most common sufferers of acne, purely because of the hormonal shifts that are associated with puberty. Current figures indicate nearly 85% of people will develop acne at some point between the ages of 12 and 25 years (Torrelo, et al., 2005). A large body of evidence now exists showing how diet may directly or indirectly influence the following 5 proximate causes of acne:

Increased proliferation of basal keratinocytes within the pilosebaceous duct, incomplete separation of ductal corneocytes from one another via impairment of apoptosis and subsequent obstruction of the pilosebaceous duct, androgen-mediated increases in sebum production, colonization of the comedo by *Propionibacterium acnes* and inflammation both within and adjacent to the comedo (Cordain 2005).

A low glycemic load (LGL) diet improved symptoms and insulin sensitivity in acne patients (Stear, S., 2001). Convincing data exist supporting the role of dairy products and high-glycemic index (GI) food in influencing hormonal factors, which can increase acne prevalence and severity (Adebamowo et al., 2005).

Current research determining the association between dietary modification and acne severity is relatively inconclusive. Although several studies investigating the relationship between acne severity and dairy products, carbohydrates, glycemic index, and high glycemic load exist, data supporting these relationships is inconsistent (Ismail et al., 2012). This lack of clarity is primarily due to the lack of randomized clinical trials with adequate power and applicability.

This study proposal would like to provide a more conclusive, well-designed trial to help tease out the evidence from the convoluted data.

## **Subject & Methods**

### **Subject:**

Total number of 30 patient(15 male and 15 female ) with acne were from moderate and sever degree randomly selected from attending the university hospital in Tanta .The study was conducted in the period from April 2015 to December 2016.Each group of study groups received experimental diet for 120 continuous days. Each group of study groups included 10 patient with two degrees of acne(mild and sever) were divided into 5 males and 5 females .These groups were as follows:

-First group as treated by oral doxycycline dose (antibiotic) and Clindamycin in some cases or Benzyl peroxide In other cases by topical.

-Second group was treated by experimental diet that was the recommended LGL diet consisted of 25% energy from protein, 45% from low-GI carbohydrates and 30% energy from fats according to (Smith et al., 2007), as

well as nutritional recommendations for vitamins and minerals.

-Third group was treated by both of drug and experimental diet.

Informed consent was obtained from all patients and controls. The Medical Ethics Committee of the Faculty of Medicine, Tanta University approved the study protocol.

The examination diagnosis and follow-up were performed by a dermatologist

### **Method design:**

Three formulated tools were used in the study:

**First tool:** Interviewing questionnaires that included three parts:

- Questions related to food habits
- Food intake
- Diet history according to (Abdelkader.,2001).

**Second tool:** Biochemical analysis including lipid profile{ total cholesterol and triglycerides according to

**Manzoor et al (2016)**}; androgen hormones{free testosterone for males according to **Smith, et al. (2007)**. and progesterone for females (**Arora ,et al .2011**)},glutathione according to **Michaelsson & Edqvist (1984)**, blood glucose according to **Smith, (2008)** and CBC according to **David and Dugdale (2012)**.

Samples were taken before and after the experiment, so as to compare and analyze the results statistically.

Three tool: Drug and dietary intervention

#### ***Statistical analysis:***

Results were collected, tabulated and statistically analyzed by SPSS version 20. Data were described in terms of mean  $\pm$  SD, frequencies. For comparison by t-test and p.Pearson to measure correlation coefficient were used  $P < 0.05$  was considered to be statistically significant.

#### **RESULTS & DISCUSSION:**

The results showed there was no significant differences in the medicine group between before and after test for macronutrients while in diet and mixture groups, there was significant difference at ( $p < 0.001$ ) between before and after test in energy, protein, fat, fiber and carbohydrates, these results were in agreement with (**Smith, et al .2007**).

The results in table (2), (3), showed that there was no significant in medicine group for food intakes from micronutrients between before and after test, while there was significant difference at ( $p < 0.05$ ) between before and after in diet and mixture group for sodium, calcium, iron, zinc, vitamin .A, vitamin B1, vitamin B2, and magnesium these results were in agreement with (**Obikoya.2010**).

In table (4), (5) Current study showed increase in levels of Triglycerides and total Cholesterol before test in experimental groups

because increased the consumption of dietary fat led to increased sebum production. Deduced on that increased serum total cholesterol level may affect the development of acne vulgaris by increasing androgens are synthesized from plasma cholesterol, according to **Manzoor, et al (2016)**.

In the same table, results showed that testosterone level was higher in medicine, diet and mixture groups before test than after the experience of the observed low levels of testosterone in groups received experimental food compared with before the experiment, these results agreed with **(Smith et al.,2007)**.

Also, shows a higher serum level of progesterone was found in patients with acne vulgaris. this result agreed with **Arora et al., (2011)**. The influence of progesterone seems to be more complicated in acne, because premenstrual changes correlate with peak levels of progesterone, but

natural and artificial progesterone display both androgenic activity and androgenic activity according to **Zouboulis (2003)**.

The study showed that the level of glutathione was lower in studied groups before test this agree with **Ikeno et al., (2011)** who mention that a decline in antioxidative activity led by a decrease in GSH quantity may play an important role in pathogenesis of acne vulgaris.

Food intake before the experiment was lacking vitamin B6, riboflavin, and selenium which were required in the manufacture of glutathione, and adequate dietary consumption of food rich in these vitamins and minerals can help the body to optimize glutathione production **(Jones et al 1992)**.

There was no difference between the groups before the experiment in level of glucose blood because high-GI

carbohydrates may be a significant cause of acne, these results agreed with **Cordain et al ., (2001)** The evidence suggests that high glycemic load (HGL) diets may trigger acne by inducing hyperinsulinemia while the differences was after test in both the diet group because Low glycemic load (LGL) diets may play a dual role in the prevention of hyperinsulinemia by lowering the postprandial insulin according to **Willett et al. (2002)**.

In addition, **Cordain et al (2001)** proposed that diet induced hyperinsulinemia may elicit an endocrine response that simultaneously promotes follicular epithelial growth and enhanced sebaceous gland activity two factors responsible for acne proliferation. Therefore, it is possible that a high dietary GL may account for higher prevalence of acne in Western societies.

The results in table (6a), (6b) and (6c) were

showed correlation coefficient of food intake ingredient with biochemical analysis in the studied groups.

There was high positive significant correlation ( $p < 0.01$ ) among calories intake with cholesterol, triglycerides and free testosterone; while, it correlated negative significantly higher ( $p > 0.01$ ) with GST. This results harmony with **Roopam et al (2014)** who reported that increased fat content leads to increased lipid peroxidation and hence the generation of Reaction Oxygen Species (ROS) which cause oxidative damage in acne patients.

There was high negative significant correlation ( $p < 0.01$ ) among dietary fiber with total cholesterol this agreed with **Brown et al, (1999)** who found that various soluble fibers reduce total cholesterol and increasing soluble fiber can make only a small contribution to dietary therapy to lower

cholesterol. Also, the present results showed negative correlation between dietary fibers with blood glucose. The high intake of dietary fiber is associated with enhanced insulin sensitivity according to **Katrina et al., (2003)**.

In addition, there was negative correlation between cholesterol and protein intake this data agree with results don by **Pang et al., (2017)**.

There was high significant negative correlation ( $p < 0.01$ ) among calcium and magnesium with cholesterol, this results harmony with **Ditscheid et al , (2005)** who explained it that calcium work by binding to bile acids and cholesterol in the small intestine, similar to the way fiber and bile acid resins work. By binding to cholesterol in the small intestine, cholesterol is not absorbed into the blood and is instead excreted out of the body in the feces.

**Amber et al (1949)** mention to that deficiency of calcium and magnesium from the diet markedly increases the free fatty acid.

In the same tables results could be noticed that there was high significant negative correlation ( $p < 0.01$ ) among zinc with cholesterol, triglycerides, free testosterone and progesterone. Also, there was high significant positive correlation ( $p < 0.01$ ) with GST.

#### **CONCLUSION:**

The present study suggested that nutrition-related lifestyle factors play a role of acne pathogenesis. It's should be guided to avoid high glycemic load diets (ie, processed foods, refined sugars). Nutrients (carbohydrates, proteins, fats) and micronutrients (vitamins and minerals essential from a nutritional point of view) preserve the skin from worsening acne and improve its appearance. Changes in the nutritional status that alter the structure



and function of the skin can directly affect the appearance of the skin and worsen the condition.

## REFERENCES

**Abdel kader MK (2001):**

Evaluation of Nutrition status , Faculty of Home Economics , Helwan University ,*first edition, Arab Nile group, , Cairo, Egypt .*

**Adebamowo CA; Spiegelman D; Danby FW; Frazier AL; Willett WC and Holmes MD (2005):**

High school dietary dairy intake and teenage acne. *J, Am Acad Dermatol.*;52 (2): 201-207.

**Amber L S; Cheng- Margaret G; Morehouse Harry J and Deuel JR (1949):**

The Effect of the Level of Dietary Calcium and Magnesium on the Digestibility of Fatty Acids, Simple Triglycerides, and Some Natural and Hydrogenated Fats: One Figure, *the Journal of Nutrition*, Volume 37, Issue 2, 1

February 1949, Pages 237–250

**Arora MK; Yadav A and Saini V (2011):**

Role of hormones in acne vulgaris. *Clin Biochem.*; 44:1035-1040.

**Brown L; Rosner B; Willett WW and Sacks FM (1999):**

Cholesterol-lowering effects of dietary fiber: a meta-analysis. *Am J Clin Nutr.* ;69 (1):30-42.

**Cordain L; Watkins BA and Mann N(2001):**

Fatty acid composition and energy density of foods available to African hominids. *World Rev. Nutri. Diet*, go, 144-161

**Cordain L (2005):**

Implications for the Role of Diet in Acne ,Article literature review in seminars in cutaneous medicine and surgery, Department of Health and Exercise Science, Colorado State University, Fort

Collins,24:84-91.  
*Elsevier Inc.* All rights reserved.

**David C and Dugdale A (2012):**

CBC: MedlinePlus Medical Encyclopedia .*MedlinePlus.C.F.* (*Wekibedia*).org.1-10-2014.02:47. AM.

**Ditscheid B ; Keller S and Jahreis G (2005):**

Cholesterol metabolism is affected by calcium phosphate supplementation in humans. *J Nutr.* Jul; 135(7):1678-82.

**Ikeno H; Tochio T; Tanaka H and Nakata S (2011):**

Decrease in glutathion may be involved in pathogenesis of acne vulgaris. *J, Cosmet Dermatol*; 10(3):240-4.

**Ismail NH; Manaf ZA and Azizan NZ (2012):**

High glycemic load diet, milk and ice cream consumption are related to acne vulgarism in Malaysian young adults: a case

control study." *BMC Dermatology*; 12:13.

**Jones DP; Coates RJ and Flagg EW (1992):**

Glutathion in Food Listed in the Nathional Cancer Institute 's Hhealth Habits and History Food Frequency Questionnaire .*Nutr Cancer* 17:13-26.

**Katrina Y Ionen; Carola Saloranta; Carina kronbergkippla; Leif Groop; Antti Aro and Suvi Virtanen (2003):**

Association of dietary fiber with glucose metabolism non diabetic reltives of subjectWith type 2 diabetic.*J ,Diabetic Care* ,volume 26 number7.

**Manzoor S; Suhail R; Syed S; Farah S; Samia A and Shazia J(2016):**

The relationship between blood lipid profile and acne in non-obese, non-pros patient *J. International journal of contemporary*

medical; 3(4):1096-1099.

**Michaëlsson G and Edqvist LE (1984):**

Erythrocyte glutathione peroxidase activity in acne vulgaris and the effect of selenium and vitamin E treatment, *Acta Derm Venereol.*; 64(1):9-14

**Obikoya G (2010):**

Vitamines for Acne. The vitamins & nutrition center, *Seacra Enterprises Inc*

**Rubin MG; Kim K and Logan AC (2008):**

Acne vulgaris, mental health and omega-3 fatty acids: a report of cases. *Lipids Health Dis.* ;7:36.

**Roopam Bassi; Saurabh Sharma; Manjeet Kaur and Aditi Sharma (2014):**

A study of changes in lipid profile in obese and non-obese females with acne vulgaris, *National Journal of Physiology, Pharmacy & Pharmacology Vol 4* | Issue 2 | 125 – 127

**Pang SJ; Jia SS; Man QQ; Song S; Li YQ; Song PK; Zhao H and Zhang J (2017):**

Dietry cholesterol in the elderly Chinese population: An analysis of CNHS 2010-2012, *J. nutrient* 9:943

**Smith RN; Neil J Mann; Anna Braue; Henna Ma`kela`inen and George A Varigos (2007):**

A low-glycemic-load diet improves symptoms in acne vulgaris patients: a randomized controlled trial. *Am.J,clin Nutr*;86:107-15.

**Smith RN (2008):**

The Role of Diet in Clinical and Endocrine Manifestations of Acne Vulgaris. A thesis submitted in complete fulfilment of the requirements for the degree of Doctor of Philosophy. School of Applied Sciences Science, Engineering and Technology Portfolio RMIT

University Melbourne,  
Australia

"A new concept for  
acne therapy: a pilot  
study with zileuton, an  
oral 5-lipoxygenase  
inhibitor." *Archives of  
Dermatology*, 139 (5):  
668-70.

**Stear S (2001):**

Carbohydrates in  
diabetes: Optimizing  
glycaemic control.  
*Community Nurse*; 15-  
16.

**Torrelo A; Pastor A and  
Zambrano A(2005):**

Severe acne in  
fantumsuccesfully  
treated with  
isotretinoin, *Pediatric  
Dermatology*, vol .22  
No.4 357-359

**Willett W; Manson J and Liu  
S (2002):**

Glycemic index,  
glycemic load, and risk  
of type 2 diabetes. *AM,  
J. Clin Nutr*; 76 (1):  
274s- 80s

**Wolf R; Matz H and Orion  
E(2004):**

Acne and diet. *Clin  
Dermatol.*; 22 (5): 387-  
93 .

**Zouboulis CC; Nestoris S;  
Adler YD; Orth M; Orfanos  
CE; Picardo M; Camera E  
and Cunliffe WJ (2003):**

**Table (1): (mean±SD) and (%) from Recommended Dietary Allowances (RDA) for Macronutrients (g) among experimental groups before and after test.**

Category	Medicine		Diet		Mixture	
Variable	Before	after	before	after	Before	after
Energy(k cal)						
Mean±SD	2755.2± 257	2683.8± 332	2770.9± 249	1718.5± 149	2758.2± 275	1799.8± 319
%from RDA	140%	141%	145%	90%	145%	94%
P.value	0.598		0.000*		0.000*	
Protein(g)						
Mean±SD	28± 6.1	30± 5.4	31± 5.8	51.5± 12.1	29± 5.5	52.7± 5.5
%from RDA	56%	60%	62%	102%	58%	104%
P.value	0.956		0.000*		0.000*	
Fat(g)						
Mean±SD	113± 18.9	112.8± 17.6	117.7± 24.7	71.9± 6.04	113± 32.9	67.9± 6.4
%from RDA	158%	156.6%	164.6%	100%	158%	94.9%
P.value	0.982		0.000*		0.000*	
Fiber(g)						
Mean±SD	12.2± 0.56	11.8± 0.52	13.8± 0.56	26.8± 5.9	12.24± 0.56	26.05± 6.2
%from RDA	46%	45%	52%	100	46.1	100
P.value	0.119		0.000*		0.000*	
Carbohydrates(g)						
Mean±SD	384.7± 45.2	354.8± 123.5	416.2± 60.1	288.6± 14.5	443.8± 49.09	280.8± 12.7
%from RDA	128%	118%	138%	96.2%	147.9%	93.6%
P.value	0.481		0.000*		0.000*	

Note: significant differences was  $p < 0.05$

**Table (2): mean±SD and (%) from Recommended Dietary Allowances (RDA) for Micronutrients "meniral" among experimental groups before and after test.**

Category	Medicine		Diet		Mixture	
Variable	before	after	before	after	Before	after
<b>Sodium(mg)</b>						
Mean±SD	2751.5± 214.9	2680.5± 288.1	2609.7± 184.5	2339± 146.5	2751.5± 214.9	2311± 111
% from RDA	117%	116%	111%	99%	117%	98.5%
P.value	0.429		0.001*		0.001*	
<b>Calcium(mg)</b>						
Mean±SD	398.9± 64.4	381.6± 67.4	458.3± 24.1	1068.2± 139.7	398.9± 64.4	955.7± 351.6
% from RDA	39.9%	38%	45%	97%	39.9%	96.8%
P.value	0.550		0.000*		0.000*	
<b>Magnesium(mg)</b>						
Mean±SD	132.2± 24.6	137.5± 48.3	114.4± 18.7	390.9± 15	132.2± 24.6	384± 24.8
% from RDA	33%	34.3%	28.6	97.7%	33%	96%
P.value	0.761		0.001*		0.004*	
<b>Iron(mg)</b>						
Mean±SD	12.7± 2.4	12.7± 1.6	12.42± 2.8	17.1± 5.19	12.7± 2.4	18.9± 5.3
% from RDA	70.5%	70.5%	70.4%	95%	70.2%	100%
P.value	.307		0.000*		0.000*	
<b>Zinc(mg)</b>						
Mean±SD	8.19± 1.7	8.37± 1.4	9.16± 1.5	15.82± 2.3	7.87± 1.2	14.7± 1.5
% from RDA	58.5%	59%	60%	100%	57%	99.9%
P.value	.806		0.000*		0.000*	

Note: significant differences was  $p < 0.05$

**Table (3): mean±SD and (%)from Recommended Dietary Allowances(RDA) for Micronutrients "vitamin"among experimental groups before and after test.**

Category	medicine		Diet		Mixture	
Variable	before	after	before	after	before	after
<b>V.A(ug)</b>						
Mean±SD	317.1± 31.9	314.8± 30.3	326.6± 37.5	931.5± 121.3	315.1± 31.9	991.5± 125
%from RDA	31.7%	31.5%	32.4%	93.15	31%	95.8%
P.value	0.811		0.000*		0.000*	
<b>V.C(mg)</b>						
Mean±SD	29.6± 2.8	32± 1.9	26.9± 2.8	56.8± 7.8	31.6± 2.8	55.8± 7.4
%from RDA	53.8	58%	48.9%	100%	57.9%	100%
P.value	0.993		0.000*		0.000*	
<b>V.B1(mg)</b>						
Mean±SD	0. 74±0.12	0.56 ±0.12	0.48± 0.13	1.4± 0.9	0.74± 0.11	1.3± 0.9
%from RDA	49.3%	37.3%	32%	99.5%	49.3%	99.1%
P.value	0.455		0.000*		0.000*	
<b>V.B2(mg)</b>						
Mean±SD	0.37± 0.11	0.74± 0.12	0.25± 0.14	1.5± 0.2	0.57± 0.11	1.7± .07
%from RDA	21.7%	49.3%	14.7%	88.2%	33.5%	100%
P.value	0.532		0.000*		0.000*	

*Note: significant differences was  $p < 0.05$*

**Table (4): Comparison mean  $\pm$ SD for biochemical analysis in serum among experimental groups**

Category	Medicine		diet		mixture	
	Before	After	Before	after	Before	after
<b>Total Lipids</b>						
<b>Total cholesterol(mg/dl)</b>						
mean $\pm$ SD	248 $\pm$ 51	254 $\pm$ 37.4	227 $\pm$ 22.3	162 $\pm$ 20.78	258 $\pm$ 51.7	139.9 $\pm$ 29.9
P.value	0.744		0.000*		0.000*	
<b>Triglycerides(mg/dl)</b>						
mean $\pm$ SD	237.9 $\pm$ 5.6	240.4 $\pm$ 46.1	244.1 $\pm$ 52.9	101.4 $\pm$ 16.5	233.7 $\pm$ 97.5	96.9 $\pm$ 21.3
P.value	0.916		0.000*		0.000*	
<b>Androgen Hormones</b>						
<b>Free. Testosterone (pg/ml) n=5 (male)</b>						
mean $\pm$ SD	252 $\pm$ 23.16	250.6 $\pm$ 10.64	256.8 $\pm$ 13.88	184.6 $\pm$ 24.7	270.6 $\pm$ 18.8	166.6 $\pm$ 32.01
P.value	0.917		0.000*		0.000*	
<b>Progesterone(ng/ml) n=5 (female)</b>						
mean $\pm$ SD	3.16 $\pm$ 1.4	2.52 $\pm$ 0.49	3.2 $\pm$ 0.36	1.7 $\pm$ 0.882	3.58 $\pm$ 0.38	1.6 $\pm$ 0.497
P.value	0.362		0.009*		0.000*	
<b>Blood glucose</b>						
<b>Fasting blood glucose(mg/dl)</b>						
mean $\pm$ SD	87.9 $\pm$ 9.5	93.2 $\pm$ 12.09	90.8 $\pm$ 16.7	88.6 $\pm$ 10.4	89.8 $\pm$ 10.4	86.3 $\pm$ 6.7
P.value	0.291		0.041*		0.036*	
<b>Post perindial .blood glucose(mg/dl)</b>						
mean $\pm$ SD	107.7 $\pm$ 51.8	109.9 $\pm$ 37.4	107 $\pm$ 19.5	106 $\pm$ 12.9	108.9 $\pm$ 12.04	105.7 $\pm$ 8.57
P.value	0.686		0.035*		0.050*	

Note: significant differences was  $p < 0.05$



**Table (5): Comparison mean  $\pm$ SD for biochemical analysis in whole blood among experimental groups**

Category	Medicine		diet		mixture	
	Before	After	Before	after	Before	after
<b>Glutathione (GPX)</b>						
mean $\pm$ SD	31.8 $\pm$ 6.7	34.9 $\pm$ 6.3	31.4 $\pm$ 5.5	173.9 $\pm$ 54.36	32.8 $\pm$ 8.35	255.1 $\pm$ 91.06
P. value	0.306		0.000*		0.000*	
<b>CBC</b>						
<b>Hemoglobin(g/ml)</b>						
mean $\pm$ SD	12 $\pm$ 1.3	12.8 $\pm$ 0.95	11.9 $\pm$ 1.4	13.6 $\pm$ 0.7	11.7 $\pm$ 0.58	13.5 $\pm$ 0.71
P. value	0.010*		0.005*		0.000*	
<b>White blood cells count(mm<sup>6</sup>)</b>						
mean $\pm$ SD	10661 $\pm$ 254.9	6883 $\pm$ 1323.6	11354 $\pm$ 2785.5	6458 $\pm$ 1158.2	7629 $\pm$ 2856.3	5732 $\pm$ 702.7
P. value	0.000*		0.000*		0.000*	
<b>Lymphocytes(%)</b>						
mean $\pm$ SD	22.30 $\pm$ 6.10	34 $\pm$ 4.90	23 $\pm$ 7.30	37.9 $\pm$ 2.4	21 $\pm$ 5.90	31 $\pm$ 4.25
P. value	0.000*		0.000*		0.000*	
<b>Neutrophils(%)</b>						
mean $\pm$ SD	74.6 $\pm$ 7.37	59.7 $\pm$ 6.14	71.9 $\pm$ 7.9	59 $\pm$ 5.6	76 $\pm$ 5.98	58.9 $\pm$ 4.04
P. value	0.000*		0.001*		0.000*	

*Note: significant differences was  $p < 0.05$*

**Table (6a): Correlation coefficient between biochemical analysis and food intake**

Variable	Cho		TG		Free. Test		progesterone		GST		F.B.G		pp.B.G	
	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>
Calories(1 <sup>st</sup> )	-.126	-.146	.105	-.026	.461	.550	.210	-.463	.211	-.017	-.177	.007	.152	.171
Calories(2 <sup>nd</sup> )	.149	.707**	-.178	.714**	-.236	.832**	-.089	.463	.007	-.816**	-.150	.436*	.046	.219
Protein(1 <sup>st</sup> )	.416	.452*	.026	.230	-.413	.433	-.208	-.114	.179	-.219	.168	-.130	.120	-.151
Protein(2 <sup>nd</sup> )	-.090	.712**	.007	-.838**	.162	-.885**	-.016	-.533	-.011	.831**	.116	-.414	.011	-.292
Fat(1 <sup>st</sup> )	-.381	-.591*	.360	-.297	.493	-.227	.319	-.244	.428	.395	.112	-.004	-.011	.261
Fat(2 <sup>nd</sup> )	.316	.834*	-.078	.758**	-.620	.674*	-.453	.338	.127	-.808**	-.096	.219	.039	-.028
Carbohydrate(1 <sup>st</sup> )	-.454	-.450*	-.140	-.365	.592	-.036	-.016	-.003	.009	.353	-.401	-.239	-.478*	.111
Carbohydrate(2 <sup>nd</sup> )	.282	.836**	.099	.896**	-.546	.623	-.031	.680*	-.024	-.802**	-.039	.169	.011	.000

\*correlation is significant at the 0.05 level (2-tailed).

\*\*correlation is significant at the 0.01 level (2-tailed).

(1) is before test      (2) is after test.

**Table (6b): Correlation coefficient between biochemical analysis and food intake**

Variable	Cho		TG		Free. Test		progesterone		GST		F.B.G		pp.B.G	
	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>
Fiber(1 <sup>st</sup> )	.247	-.017	.286	-.085	-.039	-.495	-.147	-.390	-.147	.056	.656**	-.041	.543*	.136
Fiber(2 <sup>nd</sup> )	-.193	-.772**	-.028	-.857**	.326	-.877**	.017	-.648*	-.031	.892**	.090	-.239	.001	-.039
Sodium(1)	-.009	.278	-.052	.189	-.383	-.437	.054	.108	.151	-.086	-.238	.151	.084	.261
Sodium(2)	.256	.885**	.885**	-.036	-.509	.733*	-.202	.578	.044	-.848**	-.069	.179	.086	.063
Calcium(1)	.069	.490*	-.222	.280	-.653	.038	-.776**	.065	.056	-.375	.063	.205	-.112	.041
Calcium(2)	-.238	-.826**	.113	-.857**	.370	-.787**	.046	-.633*	.032	.857**	.167	-.035	.026	-.061
Magnesium(1 <sup>st</sup> )	-.885	.268	-.240	.173	-.228	.636*	.486	.382	.150	-.313	-.215	.096	.101	.051
Magnesium(2 <sup>nd</sup> )	-.213	-.563**	.087	-.658**	.224	-.709*	.221	-.324	.081	.878**	.026	-.245	-.039	-.445*
Iron(1 <sup>st</sup> )	-.025	-.157	.263	.126	.121	.051	.414	.373	.212	-.123	.152	.290	.178	.164
Iron(2 <sup>nd</sup> )	-.280	-.778**	.200	-.799**	.355	-.627	.030	-.708*	.063	.821**	.164	-.238	.013	-.110
Zinc(1 <sup>st</sup> )	.185	.060	.046	-.056	-.043	.254	.106	-.365	-.077	-.038	-.163	-.068	-.127	-.376
Zinc(2 <sup>nd</sup> )	-.243	-.823**	.090	-.869**	.308	-.808**	-.060	-.761*	-.005	.816**	.153	-.217	.018	-.051

\*correlation is significant at the 0.05 level (2-tailed).

\*\*correlation is significant at the 0.01 level (2-tailed).

(1) is before test (2) is after test.



**Table (6c): Correlation coefficient between biochemical analysis and food intake**

Variable	Cho		TG		Free. Test		Progesterone		GST		F.B.G		pp.B.G	
	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>	1 <sup>st</sup>	2 <sup>nd</sup>
V.A(1 <sup>st</sup> )	-.245	-.291	-.180	-.068	-.302	-.288	.157	.168	-.299	.068	.093	.215	-.072	.067
V.A(2 <sup>nd</sup> )	-.206	-.807**	.057	-.861**	.375	-.821**	-.010	-.666*	-.024	.853**	.192	-.256	.483	.007
V.C(1 <sup>st</sup> )	-.179	-.308	.149	-.049	-.206	-.513	.239	-.071	-.062	.084	-.062	.024	.108	-.261
V.C(2 <sup>nd</sup> )	-.250	-.856**	.073	-.891*	.358	-.819**	.023	-.648*	-.020	.883*	.169	-.254	.031	-.087
V.B1(1 <sup>st</sup> )	-.036	-.365	-.551*	-.422	.224	-.040	.088	-.245	-.082	.344	-.137	.028	.100	.041
V.B1(2 <sup>nd</sup> )	-.252	-.841**	.036	-.895**	.310	-.847**	.013	-.667*	-.057	.858**	.133	-.239	.010	-.056
V.B2(1 <sup>st</sup> )	-.035	-.881	-.118	-.094	.250	-.345	-.621	-.460	-.101	.053	.081	.277	.054	.298
V.B2(2 <sup>nd</sup> )	-.252	-.841**	.037	-.894**	.310	-.847**	.011	-.668*	-.057	.858**	.133	-.239	.010	-.056

\*correlation is significant at the 0.05 level (2-tailed).

\*\*correlation is significant at the 0.01 level (2-tailed).

(1)is before test      (2)is after test.

## تقييم النظام الغذائي العلاجي في تحسين حالات حب الشباب

طارق محمد عفيفي \* , أمل احمد العشماوى \*\*, نادية عيد الفخرانى \*

\* قسم التغذية وعلوم الاطعمه بكلية الاقتصاد المنزلى جامعة المنوفيه , شبين الكوم , مصر

\*\* قسم الجلديه والتناسليه بكلية الطب جامعة طنطا , طنطا , مصر

### الملخص العربي :

حب الشباب هو مرض التهابي مزمن يظهر في مرحلة المراهقه نتيجة النشاط الزائد للهرمونات في هذه الفترة , تهدف الدراسة الى التعرف على دور النظام الغذائي ف تفاقم الحاله و تحسين الحاله . الدراسة اجريت على ٣٠ حالة من المصابين بحب الشباب من الدرجة الثانيه والثالثه وتتراوح اعمارهم من ١٢ - ٢٤ عام وتم تقسيمهم الى ٣ مجموعات كل مجموعة شملت ١٠ مصابين (٥ ذكور و ٥ اناث) . بدأت الدراسة في الفترة من ابريل ٢٠١٥ الى ديسمبر ٢٠١٦ . اشتملت الدراسة على تصميم استبيان مكون من ٥ اجزاء للتعرف على الحاله الصحيه والغذائيه للمصابين والتعرف على انماط الاستهلاك الغذائي واسترجاع ٢٤ ساعه من خلال سرد ما تم تناوله من اطعمه وباستخدام جدول التحليل الغذائي تم تحويل الكميات المرادفه من العناصر الغذائيه . ايضا تم اجراء التحاليل المعملية قبل وبعد التجربه للتعرف على نسبة كل من : الكوليستيرول والدهون الثلاثيه , الهرمونات وتشمل هرمون التستوستيرون الحر في الذكور والبروجستيرون في الاناث , سكر الدم , الجلوتاثيون , صورة دم كامله للتعرف على نسبة كل من الهيموجلوبين وكرات الدم البيضاء والنتروفيل والليمفاويات وقد تلت كل مجموعه من مجموعات الدراسة العلاج المخصص لها لمدة ١٢٠ يوم متواصل . اجري التحليل الاحصائي لمقارنة النتائج قبل وبعد التجربه وكانت النتائج كالآتي : كان الماخوذ الغذائي من البروتين والمعادن الاساسيه والفيتامينات والالياف الغذائيه اقل من التوصيات الغذائيه قبل التجربه بينما كان اعلى في السعرات الكليه والكربوهيدرات والصوديوم والدهون . ايضا لوحظ ارتفاع في نسبة الكوليستيرول والدهون الثلاثيه والهرمونات وكرات الدم البيضاء في كل المجموعات بينما قلت نسبتهم بعد التجربه في المجموعات التي تلت النظام الغذائي ايضا بينت الدراسة وجود نقص في الجلوتاثيون قبل التجربه بينما ارتفعت نسبته بعد التجربه في المجموعات التي تلت النظام الغذائي المختبر . ايضا بينت الدراسة وجود علاقة طرديه بين نقص الفيتامينات ونقص نسبة الجلوتاثيون بينما وجدت علاقة عكسيه بين نقص الماخوذ الغذائي من الفيتامينات وارتفاع نسبة الهرمونات . ايضا لوحظ وجود علاقة عكسيه بين نقص الكالسيوم والمغنسيوم والزنك والفيتامينات وارتفاع نسبة الكوليستيرول وكذلك وجود علاقة طرديه بين نقص الماخوذ الغذائي من تلك العناصر ونقص نسبة الجلوتاثيون . وقد خلصت النتائج الى ان المغذيات الكبرى ( البروتين , الدهون , الكربوهيدرات , الالياف الغذائيه ) والمغذيات الصغرى ( الفيتامينات والمعادن من مصادر غذائيه ) تعمل معا على تحسين مظهر الجلد وتحسن حالات حب الشباب والسيطره على تفاقم الحاله .

**الكلمات المفتاحية** حب الشباب , التغذية العلاجي , النظام الغذائي , مؤشر سكر الدم

*Evaluation of the therapeutic diet for enhancement of acne status*

*Tarek M. Afifi, Aml A. El-Ashmawy Fakharany and Nadia E. El-*

---