

**Cross Sectional Study Evaluating the Prevalence
and Risk Factors of Acne Vulgaris among Ain
Shams University Medical Students**

Thesis

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List of Contents

<i>Subject</i>	<i>Page No.</i>
List of Tables.....	i
List of Figures	iii
List of abbreviations	iv
Introduction.....	1
Aim of the Work.....	3
Review of Literature	
Acne Vulgaris	4
Risk Factors of Acne Vulgaris.....	24
Results	37
Discussion.....	56
Conclusion and Recommendations	67
Summary.....	68
References	70
Appendix	I
Arabic Summary	—

List of Tables

Table No.	Title	Page No.
Table (1):	Personal characteristics and family history among study participants	37
Table (2):	Relation between gender and age of onset of acne	38
Table (3):	Acne characteristics among study participants	40
Table (4):	Weight, height and body mass index among study participates.....	41
Table (5):	Risk factors of acne among participants	42
Table (6):	Relation between gender and grade of acne	43
Table (7):	Sites of acne predilection	44
Table (8):	Acne lesions among study participants	45
Table (9):	Treatment received among study participants	46
Table (10):	Factors increasing acne among study participants	47
Table (11):	Comparison between cases with and without acne as regard personal characteristics	48
Table (12):	Comparison between cases with and without acne as regard anthropometric data	49
Table (13):	Comparison between cases with and without acne as regard risk factors of acne	51
Table (14):	Comparison between cases with different grades of acne as regard personal characteristics.....	52

List of Tables *(Cont...)*

Table No.	Title	Page No.
Table (15):	Comparison between cases with different grades of acne as regard body mass index.....	53
Table (16):	Comparison between cases with different grades of acne as regard risk factors of acne	54
Table (17):	Comparison between cases with different grades of acne as regard exacerbating factors of acne	55

List of Figures

Figuer No.	Title	Page No.
Figure (1):	How acne develops	6
Figure (2):	A schematic diagram of comedogenesis	7
Figure (3):	A–D. Acne pathogenesis	7
Figure (4):	Lesion progression in acne	8
Figure (5):	A proposed overall theory for acne pathogenesis.....	9
Figure (6):	Stages of acne.	18
Figure (7):	Skin type of participants	38
Figure (8):	Age of onset of acne	39
Figure (9):	Prevalence of acne vulgaris	40
Figure (10):	Relation between gender and acne severity.....	43
Figure (11):	Sites of acne predilection.....	44
Figure (12):	Comparison between cases with and without acne as regard anthropometric measures	49

List of abbreviations

ANOVA test	: Analysis of Variance test
APCs	: Antigen Presenting Cells
BMI	: Body Mass Index
DAMPs	: Danger-associated molecular patterns
DHEA-S	: Dehydroepiandrosterone sulfate
DHT	: Dihydrotestosterone
HS	: Highly Significant
IGF-I	: Insulin-like Growth Factor 1
IL	: Interleukin
MAPK	: Mitogen-Activated Protein Kinase
Max	: Maximum
MDA	: Malondialdehyde
Min	: Minimum
N	: Not Significant
N	: Number
NF-kB	: Nuclear Factor kappabeta
NO	: Nitric Oxide
P.acne	: Propriobacterium Acne
PAMs	: Pathogen--associated molecular patterns
PMNLs	: polymorphonuclear leukocytes
PUFA	: Polyunsaturated Fatty Acid
PUVA	: Psoralens and UltraViolat A
S	: Significant
SD	: Standard Deviation
Sig	: Significance

List of abbreviations *(Cont...)*

SOD	: Superoxide Dismutase
SPSS	: Statistical Package for Social Science
TGF-β	: Transforming Growth Factor Beta
TLR2	: Toll-like Receptor 2
TNF	: Tumour Necrotic Factor
XD	: Xanthine Dehydrogenase
XO	: Xanthine Oxidase

Introduction

Acne is a chronic inflammatory disease of the pilosebaceous units. It is characterized by formation of comedones, erythematous papules and pustules, less frequently by nodules or pseudocysts and in some cases is accompanied by scarring (*Simpson and Cunliffe, 2004*).

Acne vulgaris is a multifactorial, spontaneously resolving pleomorphic skin disease, characterized by a variety of non-inflamed and inflamed skin lesions (*Wilcox et al., 2007*).

The precise mechanisms of the acne process are not completely understood, however it is known to be characterized by sebum overproduction, follicular hyperkeratinization and inflammation (*Zaenglein and Thiboutot, 2008*).

Acne is a disease of high prevalence worldwide. Community-based studies in the UK, Australia, New Zealand, Singapore, and Nigeria have found prevalence rates ranging from 27% to 93% in adolescents. The wide range resulted from the much higher prevalence during late than early adolescence (*Tan et al., 2007*). Although acne occurs in all races there is much difference in prevalence among these groups (*Halder et al., 2003*).

Acne vulgaris is one of the most frequently encountered conditions in all of dermatology and primary care practice. Even in its mild form, acne can have impacts on mental health

(e.g anxiety and depression), as well as on social interactions, self-confidence, self-esteem, and employment opportunities (*Callender, 2004*).

Acne, being an important cosmetic problem, has important medical, psychological, and social impact on quality of life. Such impact is the trigger for acne patients to seek medical help (*Goodman, 2006*).

Studies proved that acne patients are in great need for health care, which can lead to significant costs for the health care system due to the frequency and chronic course of acne. The degree of inflammation in acne determines the risk and amount of post-acne scarring. Acne scarring originates from a deep inflammatory reaction, and involves the destruction or loss of connective tissue, with dermal atrophy and fibrosis. Even though atrophic scars are the most common type of post-acne scarring, some patients may also present with hypertrophic scars and keloids (*Choi et al, 2006*).

Although acne is easy to diagnose, the polymorphic nature of acne vulgaris and its varied extent of involvement do not permit simple evaluation of its severity. Because the acne lesions may vary in number during the natural course of the disease, various measurements have been developed, based on clinical examination and photographic documentation, to assess the clinical severity of acne vulgaris (*Witkowski and Parish, 2004*).

Aim of the Work

The aim of this study was to evaluate the prevalence of acne vulgaris among Ain Shams University medical students and to find out the possible risk factors associated with acne vulgaris.

Acne Vulgaris

Acne vulgaris is a chronic inflammatory condition of the pilosebaceous unit of the skin. It is one of the most frequent chronic skin diseases and the commonest dermatologic disorder of adolescents (*Uslu et al., 2008*). Acne word is derived from the Greek word acme, which means “prime of life (*Cooley et al., 1998*).

Epidemiology of Acne Vulgaris

➤ **Incidence**

Acne vulgaris is virtually universal in adolescence. An individual is more likely to develop acne than any other disease (*Simpson and Cunliffe., 2004*). Hospital-based studies done on acne vulgaris in Asian population have shown that the incidence of acne vulgaris ranged from 11.2% to 19.6% of the total new patients attending hospitals (*Al-Ameer and Al-Akloby 2002; Tan et al., 2007*). A survey of Australian private dermatology practices reported that 10% of new attending patients were for acne (*Stathakis et al., 1997*).

Regarding Egyptian studies, acne vulgaris was found to affect 5.37% among the surveyed population in rural areas of Assuit Governorate, Upper Egypt (*Abdel-Hafez et al., 2003*). The difference in acne incidence rates between different societies cannot be solely attributed to genetic differences among populations but likely results from different environmental factors

and identification of these factors may be useful in the treatment of acne (*Cordain et al., 2002*).

➤ **Prevalence**

There are many factors which may influence the prevalence of acne vulgaris (*Daniel et al., 2000*).

Community-based study of Tehran high school pupils confirms that acne is a very common disorder in adolescents and mirrors the global significance of the disease in particular, moderate to severe acne, which requires systemic treatment (*Gollnick et al., 2003*) and may have a considerable emotional impact, exhibits a high prevalence seen in Iranian pupils which was less commonly (14%) compared with other populations reported (*Ghodsi et al., 2009*).

Regarding Egyptian studies, epidemiologic study of acne vulgaris among secondary schools in Ismailia Governorate, Egypt shows that the prevalence of acne vulgaris in the studied group was found to be 54.2% with no statistical significant difference in acne distribution between urban and rural areas.

Age group and prevalence of acne shows that there is a high statistical significant difference between different age groups where acne was more prevalent in older age > 16 years than age of 16 years or younger. Sex prevalence showed that 51.5% of the males and 56% of the females in the studied group had acne (*Mohamed, 2011*).

Aetiopathogenesis of Acne Vulgaris

The aetiopathogenesis of acne vulgaris is multifactorial and is related to the consequences of abnormal follicular epithelial proliferation and keratinization, excess sebum production, intrafollicular *Propriobacterium acne* (*P.acne*) colonization, and inflammation (**Figure 1**) (*Bhambri et al., 2009*).

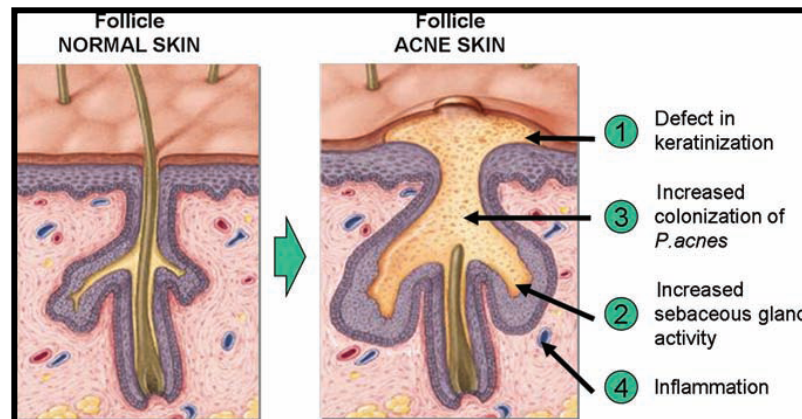


Figure (1): How acne develops (*Mayo, 2009*).

➤ **Follicular hyperkeratinization and comedone formation**

Increased sebum production and follicular hyperkeratosis result in the development of micro-comedones (*Knor, 2005*). This begins in the keratinized lining of the upper portion of the follicle. Comedone formation occurs when the corneocytes, which are normally shed into the lumen of the follicle and extruded through the follicular ostium, are retained and accumulate, leading to hyperkeratosis. As the comedone expands the sebaceous lobule undergoes regression. Because of the very narrow opening to the skin surface, there is initially an accumulation of loosely packed shed keratinocytes and sebum. With expansion of comedo, the contents become closely packed, creating whorled lamellar

concretions. As the forces increase, rupture of the comedo wall with extrusion of immunogenic keratin and sebum occurs, with inflammation (**Figures 2,3**) (*Zaenglein and Thiboutot., 2008*). Androgens also affect sebocytes and infundibular keratinocytes in a complex manner influencing cellular differentiation, proliferation, lipogenesis and comedogenesis (*Kurokawa et al., 2009*).

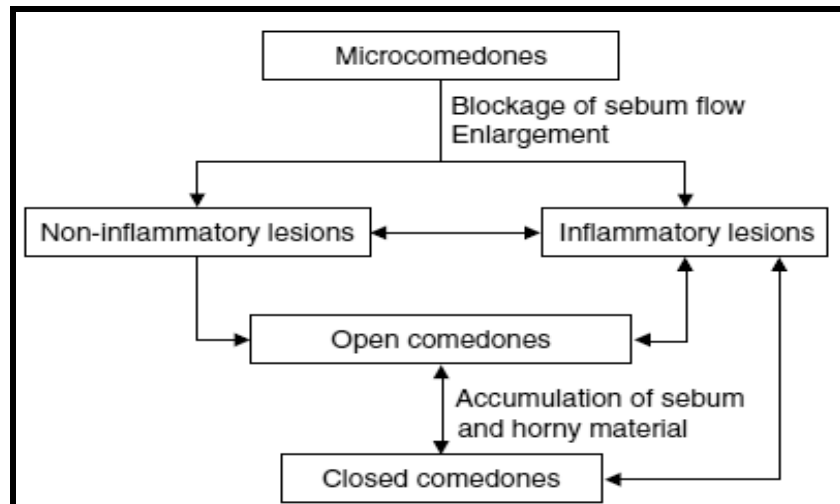


Figure (2): A schematic diagram of comedogenesis (*Gollnick, 2003*).

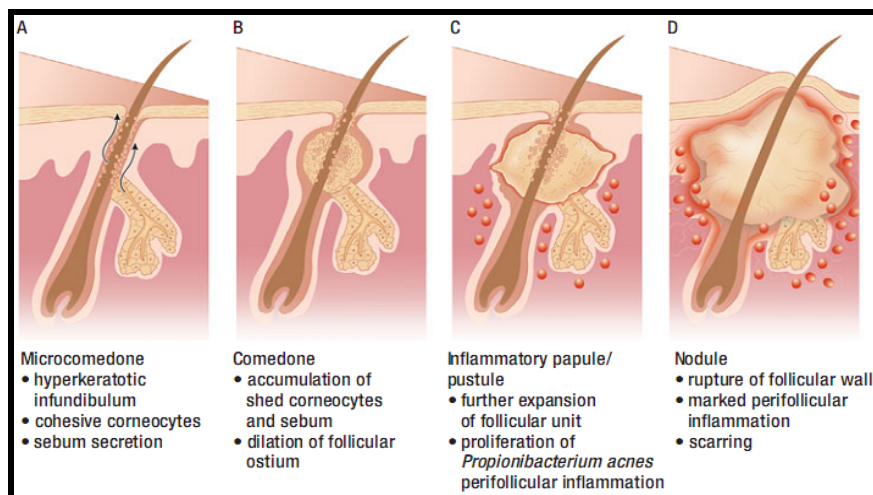


Figure (3): A–D. Acne pathogenesis (*Zaenglein et al., 2008*).