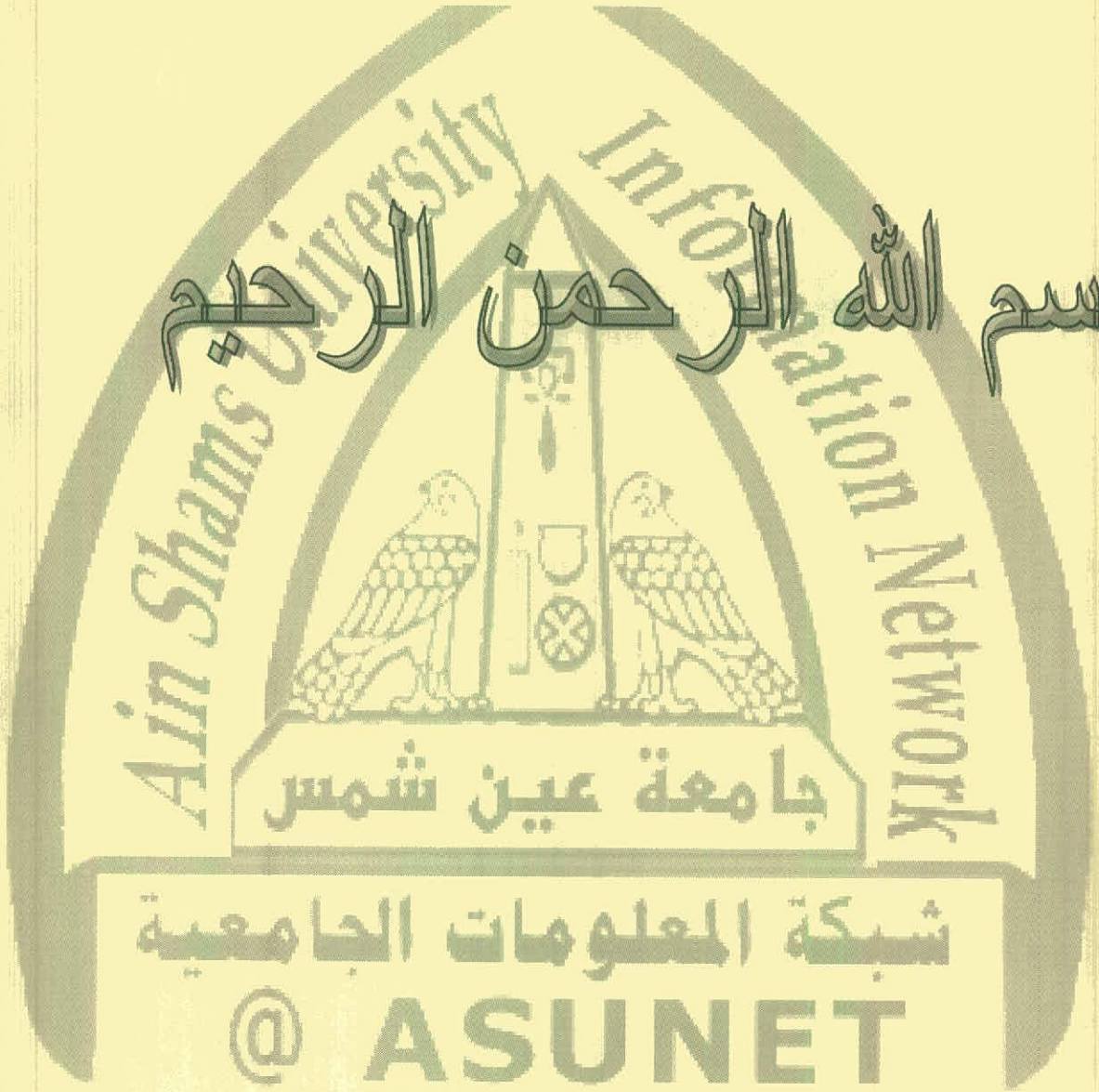
شبكة المعلومات الجامعية



بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأفلام بعيدا عن الغبار

في درجة حرارة من ١٥-٢٥ مئوية ورطوبة نسبية من ٢٠-٤٠%

To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

IMMUNOEXPRESSION OF TENASCIN AND FIBRONECTIN IN ACNE VULGARIS

Thesis

Submitted for Partial Fulfillment of Master Degree

In

Dermatology & Venereology

By

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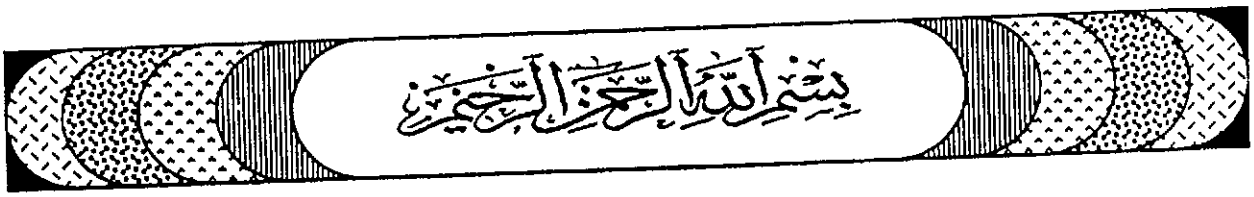

﴿ قَالُوا سُبْحَانَكَ لا عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ ﴾

صدق الله العظيم

٣٢ (البقرة)

ACKNOWLEDGEMENT

First of all, thanks to ALLAH *for helping and supporting me to accomplish this work.*

I wish to express my deep gratitude and great appreciation to Prof. Dr. ***LAYLA MANSOOR MOHAMMED*** *Professor of Dermatology & Venereology Faculty of Medicine, Tanta University, for her continuous help and guidance and kind cooperation throughout this work. she patiently guided me, provided me most readily and generously with her valuable relevant views, which kept me on the right way.*

I would like to express my great thanks to Prof. Dr. ***HUSSEIN MOHAMMED GHORABA*** *Professor of Pathology, Faculty of Medicine Tanta University for his great help, meticulous supervision, continuous guidance and encouragement during the entire work.*

I would like to express my sincere thanks to Dr. ***AMANY AHMAD KOLKAILA,*** *Assistant professor of Dermatology & Venereology , Faculty of Medicine, Tanta University for her kind supervision, guidance, and encouragement.*

CONTENT

INTRODUCTION

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Acne vulgaris is one of the most common dermatological diseases. The prevalence may be as high as 83-95% at age 16, but decreases at age 20, and is very low at the age of 35 [1]. Acne vulgaris is the clinical expression of inflammation of the pilosebaceous unit [2]. The aetiology and pathogenesis are not completely known, but the following factors are involved:

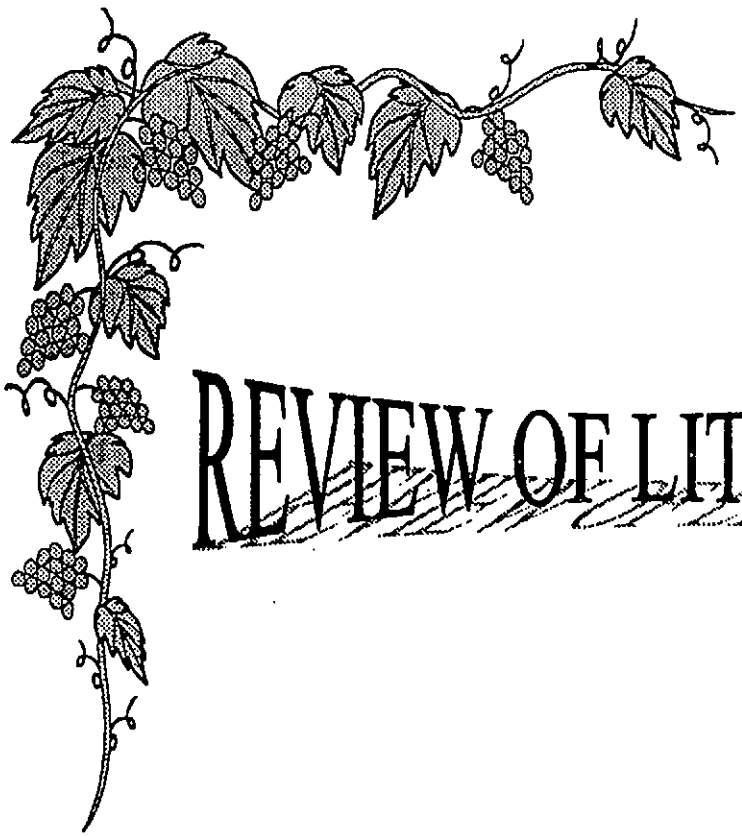
1] Increased sebum production.

2] Abnormal follicular Keratinization.

3] Proliferation of propionibacterium acnes in the sebum

4] Inflammation [3].

Tenascin and fibronectin are extracellular matrix glycoproteins which can interact with cells and alter their capacity to adhere, migrate and proliferate.In contrast with fibronectin, tenascin has a restricted distribution in normal skin, but is induced during epidermal proliferation, and wound healing. Because acne involves hyperproliferation of ductal keratinocytes, and rupture of the duct may occur during inflammation, the distribution of tenascin and fibronectin may be changed in acne lesions [4].



REVIEW OF LITERATURE

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ACNE VULGARIS

Definition:

Acne, as defined by Kligman[5], is a polymorphic disorder which exhibits a series of diverse lesions, comedones, papules, pustules, nodules, cysts and scars.

It is a chronic, self limited, inflammatory disease of the pilosebaceous units[6], seen primarily in adolescents [7]. If left untreated, it can leave physical and emotional scars that may be devastating [8].

Epidemiology:

Acne vulgaris is sufficiently common that it has often been termed physiologic[9]. It affects nearly 85% of adolescents and young adults [10].

It usually starts in adolescence and resolves by the mid-twenties[11]. Acne develops earlier in females than in males [12], which may reflect the earlier onset of puberty [6].

The incidence of acne is highest during the middle to late teenage period, then it decreases. However, acne may persist, particularly in women, through the third decade or even later. On the other hand, the severest forms of acne most often occur in teenage boys [13].

Pathogenesis:

Acne is a disorder of the sebaceous follicles, which are special pilosebaceous units located on the face , chest , and back , the sites of primary involvement. They consist of sebaceous glands associated with small hair follicles. Several factors contribute to the pathogenesis of acne:

I- increased sebum production

II- abnormal follicular keratinization.

III- Proliferation of Propionibacterium acnes in the sebum, and

IV- Inflammation[3] .

I) Increased sebum production :

In attempt to throw light on the role of increased sebum production in the pathogenesis of acne, the following aspects have been explored :

a- Role of sebum and seborrhoea.

b- Role of androgens.

c- Role of squalene and oleic acid.

a- Role of sebum and seborrhoea:-

Sebum is the product of the holocrine sebaceous glands [14] . It is a mixture of different lipid classes, the principal constituents of which are triglycerides, in addition to wax esters, squalene and minor amounts of cholesterol esters and cholesterol [15,16]. Analysis of lipids collected from the skin surface showed that they had lower levels of triglycerides[17] and varying amounts of free fatty acids (FFA) as well as cholesterol but no

squalene .FFA of human skin are derived primarily from triglycerides of sebum by the action of bacterial lipase [18,19].

The role of sebaceous glands in the pathogenesis of acne has long been recognized, so much so that the disease is standardly classified as a sebaceous gland disorder. However, such a designation is an over simplification as the disease is of multifactorial aetiology [20].

A high rate of sebum excretion was considered as an essential factor in comedogenesis [21]. All acne patients exhibit high sebum production [22]. Pochi and Strauss [23]. found that acne patients excreted more sebum than did normal individuals. Moreover, the levels of sebum excretion correlated well with the severity of acne [24]. The severity of seborrhoea in acne was positively correlated with the mean sebum excretion of individual follicles but not with the number of active follicles[25]. Though the relation between acne and seborrhoea has been well documented, sebum seems to have an indirect role in the development of acne lesions. Sebum contains materials serving as nutrients for the microflora inhabiting the pilosebaceous follicles [26]. Therefore, increased rates of sebum excretion should probably be associated with increased number of bacteria and higher level of bacterial activity within the follicle [17]. Hence, it could be likely that bacteria, rather than sebum itself are the direct triggers.

Examination of the lipid retained in comedones and in large sebaceous follicles showed lipolysis to FFA amounting to 100% [17]. Strauss and pochi [27] and Kligman et al, [28] expressed the view that FFA were the irritants primarily responsible for the initial stages of the