DIET AND ACNE

Essay

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CD2BP1 : *CD2 antigen binding protein.*

DHT : *Dihydrotestesterone.*

EGFR : *Epidermal growth factor receptor.*

EPA : *Eicosapentaenoic acid.*

ESR : *Erythrocyte sedimentation rate.*

FSH : *Follicle stimulating hormone.*

GI : *Glycemic index.*

GL : *Glycemic load.*

GLA : *Gamma - linolenic acid.*

GP : *General practitioner.*

IGF : *Insulin growth factor.*

IGF BPs : *Insulin growth factor binding proteins.*

IL : *Interleukin.*

LH : *Lutenizing hormone.*

LTB4 : Leukotriene B4.

NEFAs : Non esterified free fatty acids.

P acne : Propionibacterium acne.

PBMs : Peripheral blood monocytes.

PCOS : Polycystic ovary syndrome.

PSTPIP1 : Proline-serine-threonine phosphate interacting protein 1.

PUFAs : Polyunsaturated fatty acids.

PUVA : Long wave ultraviolet radiation.

SHBG : Sex hormone binding globulin

TNF : Tumour necrosis factor.

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INTRODUCTION

Acne is a chronic inflammatory disease of the pilosebaceous unit,characterized by formation of comedones,erythematous papules and pustules,less frequently nodules or cysts and in some cases scarring **(Degitz et al.,2007).**

Although all age groups may be affected by its many variants,it is primarily a disorder of adolescence.The economic as well as psychological impact of acne is undeniable.Although the pathogenesis of acne is precisely unknown,most recent epidemiological studies of non westernized populations suggest that dietary factors including the glycemic load may be involved**(Zaenglein and Thiboutot,2008).**

In Western nations, a majority of the population has signs and symptoms of acne at least during puberty. Epidemiological data suggests up to 80% of individuals are affected. Men and women develop acne about equally. The disease has its onset at age 10–14 years and regresses by age 20–25 years. In some patients acne may persist into the fourth or fifth decade of life **(Degitz et al.,2007).**

Although familial and ethnic factors are implicated in acne prevalence,this observation is complicated by the finding that incidence of acne has increased with the adoption of western lifestyles **(Smith et al.,2007).**

Triggering of acne by food has long been held insignificant,However, new epidemiological observations suggest that diet can play a role in acne; populations with a natural lifestyle do not develop acne.Various hypotheses

correlate acne with western diet e.g. with dairy products and with the promotion of hyperglycemia, they are increasing production of insulin and other growth factors ,thus, producing seborrhea and follicular hyperkeratosis (**Degitz et al.,2007**).

Molecular mechanisms postulated as responsible for the stimulation of the pilosebaceous unit are logical and conform to known science.Recent clinical trials have established in a small group that dietary manipulation changes the biochemical and endocrine parameters that are associated with acne metabolism in expected ways, and dietary changes are accompanied by clinical improvement **(Smith et al.,2007).**

The postulated mechanism of action consists of an elevated supply of dihydrotestosterone (DHT) acting at the intranuclear androgen receptor of the germinative cell layer of various components (sebaceous, hair, and ductal lining) of genetically predisposed pilosebaceous units **(Bataille et al.,2002).**

Epidemiologic studies show that communities that maintain a diet high in omega-3 fatty acids have low rates of acne. Indeed, a large study that refuted the acne-iodine connection found the lowest rates of acne were among adolescents consuming the greatest amounts of fish and seafood **(Logan,2003).**

Investigators reported that inhibition of leukotriene B4 reduces sebum production and improves inflammatory acne. Fish oil, rich in omega-3 fatty acids, is well documented to have leukotriene B4 inhibiting properties in human beings. Polyunsaturated fats, and omega-3 from fish oil in particular, have been inversely

correlated with androgen levels.Although large population studies have not found an association between dairy-based saturated fats and acne,they are well documented to be in association with inflammation and poor glycemic control **(Adebamowo et al .,2006).**

AIM OF THE ESSAY:

The aim of the essay is to clarify the relation between diet and acne and whether it plays an important role as a predisposing factor and its role in the help of treatment of acne.

Acne

Acne is a term derived from the greek “acme” or prime of life.The young girl who is desperately aware of the smallest comedo and the young man with his face or back a battle field of acne cysts and scars which is familiar.Both need treatment and help in coming to terms with their condition **(Buxton , 1999).**

It was in the sixth century AD that the term “acne” was first used by the emperor Justinian’s physician, Aetius Amidenus.It was later translated from Greek into Latin,and through these translations confusion arose regarding its original meaning **(Goolamali and Andison , 1977).**

The debate continues as to whether its origin is from the greek acme,meaning peak,or whether acne was actually the original term.Its use became obsolete until the 1800s,when “acne” regained a place in medical dictionaries.In 1842, Erasmus Wilson separated acne simplex(acne vulgaris) from acne rosacea **(Waisman , 1983).**

Epidemiology:

Acne is one of the most prevalent skin conditions, affecting more than 85% of teenagers. It typically starts at puberty and resolves slowly as the person reaches 20 , although some people continue to have acne till 40 and 50 years. Men and women develop acne about equally. It is seldom

life threatening and is often dismissed as a self limiting condition **(Ayer and Burrows , 2009).**

Although it is typically thought of as a a disease of youth,12% of women and 3% of men will continue to have clinical acne until 44 years of age.Caucasian boys and men have been shown to have more severe nodulocystic disease than do their black counterparts **(Goulden et al ., 1999).**

Acne is an easily treated cause of disfigurement and psychological morbidity. It affects more than 80% of people at some point in their life, up to 14% of whom consult their general practitioner (GP) and 0.3% a dermatologist. About 3.5 million consultations with GPs occur in the United Kingdom annually for acne.Morbidity can be high and associated with disfigure- ment, pain, loss of confidence, and impairment of normal social and workplace function, with documented effects on quality of life including depression, dysmorphobia, and even suicide **(Purdy and De Berker , 2009).**

Individuals at increased risk for the development of acne include those with an XYY chromosomal genotype or endocrine disorders such as polycystic ovarian syndrome,hyperandrogenism,hypercortisolism and precocious puberty.Patients with these conditions tend to have more severe acne that is unresponsive to standard therapy **(Zaenglein and Thiboutot , 2008).**

Clinical picture of acne

In most cases it is not difficult to diagnose acne but history and physical examination are key to know if there is a cause of acne and to organize an appropriate and maximally effective treatment plan.The physician should ask about history of previous medications used for acne or other conditions.A review of cosmetics and sunscreens is also helpful.In female patients,a menstrual and oral contraceptive history is important in determining hormonal influences on acne **(Zaenglein and Thiboutot,2008).**

It is important to take history since some factors and medications can cause acne.Treatment with systemic corticosteroids can cause steroid induced acne and the use of anabolic steroids can cause body-builders acne.Potent topical steroid treatment can cause perioral or periorbital dermatitis with papules and pustules.Pustular drug eruptions and bacterial and fungal folliculitis can also resemble acne but can be distinguished by the absence of comedones **(Purdy and De Berker , 2009).**

The inflammatory lesions of acne originate with comedo formation but then expand to form papules,pustules,nodules and cysts of varying severity.Erythematous papules range from 1 to 5 mm in diameter.Pustules tend to be approximately equal in size and are filled with sterile,white pus.As the severity of lesions progresses,nodules form and become markedly inflamed,indurated and tender.The cysts

of acne are deeper and filled with a combination of pus and serosanginous fluid.In patients with severe nodulo cystic acne,these lesions frequently coalesce to form massively inflamed complex plaques that can include sinus tracts (Figure 1) **(Zaenglein and Thiboutot,2008).**

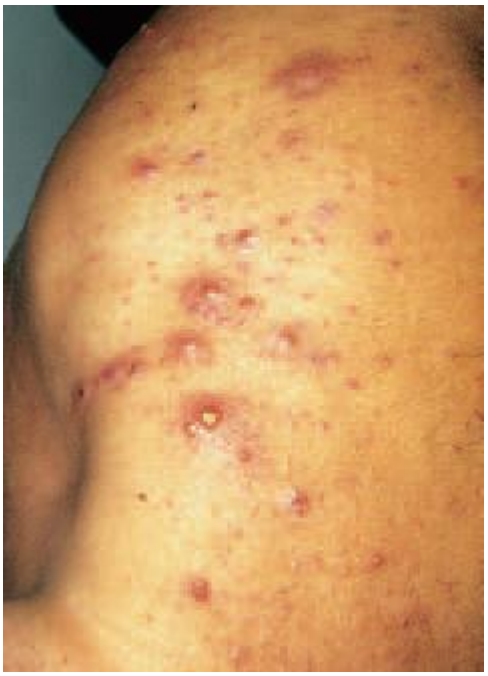

Figure 1.Papulopustular lesions (Hunter et al ., 2002).

Nodules can be more painful, more unsightly, and carry a greater risk of scarring than more superficial disease. In acne conglobata, nodules are widespread with interconnecting channels containing