



EUROPEAN ACADEMY OF
DERMATOLOGY AND VENEREOLOGY

Information Leaflet for Patients

ACNE: PRODUCTION, EVOLUTION AND DIAGNOSIS

The aim of this leaflet

This leaflet is designed to help you understand more about acne vulgaris: it tells you what this condition is, how it develops, what it looks like, and what factors influence it.

ACNE: PRODUCTION, EVOLUTION AND DIAGNOSIS

What is acne vulgaris?

Acne vulgaris, or acne, is an inflammatory dermatological disorder of the *pilosebaceous unit* (sebaceous glands and hair follicle) that most frequently affects male and female adolescents on the face. It is characterized by the presence of non-inflammatory and inflammatory lesions. The former, which consist of open and closed , also called “blackheads” and “whiteheads,” always precede the development of inflammatory lesions (Figure 1).



Fig. 1 Open Comedones (“blackheads”)



Closed Comedones (“whiteheads”)

The inflammatory lesions may be superficial, like *papules* and *pustules*, or deep like *nodules*.

Papules are red, raised with respect to the surrounding skin, and less than 5 mm in diameter. *Pustules* look like *papules*, but

are pus-filled (covered by a whitish-yellow collection of *purulent* material). Their popular term is “pimples.” When the lesions become larger than 5 mm, they are defined as *nodules* (Figure 2).



Fig. 2 Papule



Pustule



Nodule



Fig. 3 Erythematous macule

Post-inflammatory hyperpigmentation

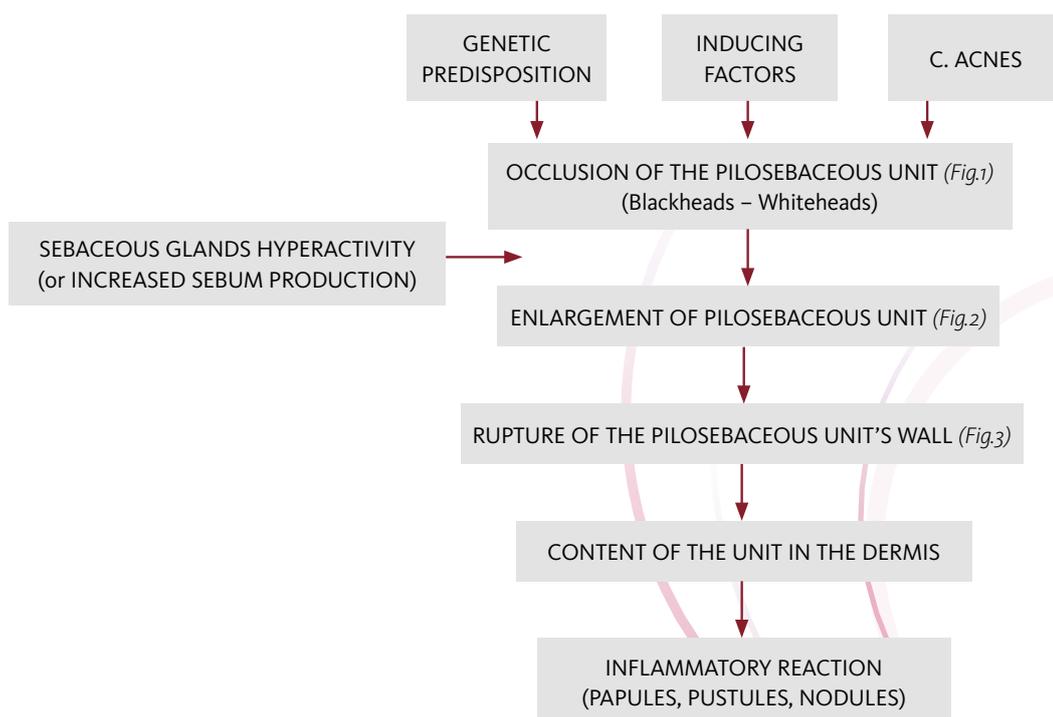
Postacne atrophic scars

When active acne lesions subside, they leave room for *erythematous macules* (a reddened flat lesion) or *post-inflammatory hyperpigmentation* (skin darkening often found in patients with skin of colour), and sometimes lead to *atrophic scars* (depressions or pits in the skin) (Figure 3).

If you have acne, your quality of life may be negatively impacted. Patients frequently suffer from low self-esteem and anxiety/depression. In adult acne patients, a higher unemployment rate in comparison with adults without acne has been found.

Acne lesions are typically localized on the face (99%), back (65%), and chest (18%) of patients.

How does acne develop?



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Who is affected by acne and how common is it?

The prevalence of adolescent acne is approximately 80%, with a peak around the age of 14 in females

and 16 in males. In adulthood, after the age of 25, around 3% of males and 12% of females are affected.

There are different events involved in the development of acne, and they are dominated by a genetic predisposition of patients. They start with the *occlusion*, or blockage, of the *pilosebaceous unit* (hair follicle and sebaceous gland) as a consequence of the increased production of the cells of the external part of the pilosebaceous unit's duct, close to the skin and their reduced tendency to detach. It clinically corresponds to open *comedones* that act like a stopper, entrapping the content of the follicle so that it can't get out. Sebum, bacteria (like *C. acnes*) and cells of the pilosebaceous unit wall, accumulated inside the follicle, result in its progressive enlargement.

Sebaceous glands of acne patients produce sebum in a greater amount compared to patients without acne. In addition, their sebum contains substances that promote inflammation. If the duct remains closed, progressive enlargement of the follicle ends up rupturing its wall, and the outflow of contents in the surrounding dermis induces inflammation.

Another factor in the development of acne is a bacterium named *C. acnes*, present on the skin of all human beings (known as normal skin flora). In acne patients, *C. acnes* produces chemical substances that impact the immune system and adjust and trigger inflammation. It may also be a co-factor in blocking the *pilosebaceous unit*. *C. acnes* doesn't work in acne as an agent of infection; acne is known to be a non-infectious disease.

All the above-mentioned factors result in the development of inflammatory lesions that characterise acne: *papules*, *pustules* and *nodules*.

Are there external factors that may influence the evolution of your acne?

Hormones

The most important hormonal factors in acne are *androgens*, male hormones that women also produce, although in a lower amount in comparison to men. In patients with acne, for genetic reasons, androgens are more effective in stimulating *sebaceous gland* activity and blocking the pilosebaceous unit.

On the practical side, combined oral contraceptives, commonly called "the pill", and antiandrogens, are effective in female acne patients. Other combined contraceptives (pills or devices) contain progestins that may induce acne or worsen already existing acne.

The consumption of anabolic steroids by athletes, like testosterone and its derivatives, are known triggers of mild to severe forms of acne. Prolonged use of systemic corticosteroids may be the cause of a cutaneous eruption characterized by numerous, identical, small *papules*, similar to but not actually those of acne, which are usually distributed on the upper chest.

Stress

Stressful events, through the release of chemical molecules produced by neural activation, are known as potential triggering factors of acne.

Diet

Controversial data are available in the literature about the relationship between acne and diet. A possible correlation between acne and dairy products may exist, but it is neither confirmed nor widely accepted. In practical terms, it is advisable to follow a balanced diet, meaning all types of food in the right portions.

Nutritional supplements

The consumption of a large amount of whey proteins is suspected to exacerbate acne lesions, although the data supporting that are weak.

Pollution and drugs

Dioxins and other similar chemical compounds are involved in environmental pollution and are known to induce an *acneiform reaction* (a skin condition that is not truly acne but looks similar to it). Also, a high level of environmental pollution may potentially increase the inflammatory aspects of acne.

Drugs like lithium, isoniazid, high doses of vitamin B12 and some anticancer agents (EGFRi, BRAFi, MEKi) can also lead to *acneiform eruptions*.

Sun exposure

An in-depth, “systematic” review of 7 studies found no convincing evidence that natural sunlight exposure improves acne. In some patients, a moderate level of sun exposure may induce a temporary improvement. The short-term effect of sun tanning is largely related to camouflage. Long-term, strong sun exposure, in particular if associated with erythema, may actually lead to worsening of acne.

Weather conditions

Living in very hot and humid conditions may induce an *acneiform reaction* known as *acne aestivalis*, characterised by similar, small, red papules, localized on the upper chest.

Smoking

Smoking may worsen acne, particularly in adult females, but this is not fully agreed upon.

Cosmetics

Oily, occlusive, and *comedogenic* cosmetic products may induce or worsen acne, in particular the type with open *comedones* (“blackheads”). You need to look for the term “*non comedogenic*” on the package of the right cosmetic to use.

Pregnancy

Pregnant patients over 25 years old with acne have more acne lesions during the second trimester. The highest number of acne lesions has been found in patients who are pregnant for the first time, and in female or low birthweight newborns.

Acne Severity



Fig. 4 Mild



Moderate



Severe

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What are the different clinical characteristics of acne?

Characteristics of acne are related to severity (mild, moderate, severe; see Figure 4), the age of appearance (neonatal, infantile, mid-childhood, preadolescence, adolescence, adult), and/or the skin colour (dark skin).

In most patients with acne, both inflammatory and non-inflammatory lesions continuously appear and disappear, changing position, with each lesion persisting for an average of 3 to 6 weeks. It often leaves an erythematous macule, *post-inflammatory hyperpigmentation* (skin darkening), and sometimes a scar.

Severity

In mild acne, a few lesions cover less than half of the face, while in moderate acne, lesions are more numerous and cover over half of the face. In severe acne, lesions are all over the face and often on the chest, with nodules and sometimes cysts.

Age of onset

Neonatal acne is present at birth and/or during the first 6 weeks of life. When it is present between 6 weeks and 12 months old, it is defined as *infantile acne*. Between 1 and 7 years old, it is called *mid-childhood acne*. *Preadolescent acne* occurs between 7 and 12 years old, and *adolescent acne* between 12 and 19 years. *Adult acne*, persistent or with a late onset, is diagnosed after the age of 25.

Acne may be similar to other conditions, but not the same

Acne must be differentiated by the clinician from skin eruptions that mimic its clinical features. For example, *folliculitis* is a bacterial infection characterized by isolated pustules, growing around the exit of a hair from the follicle, and no comedones. *Rosacea* is an inflammatory eruption of the face that appears in adulthood and but does not include *comedones*. *Acneiform eruptions* are usually due to drugs or weather conditions, also without *comedones* (see section “Are there external factors that may influence the evolution of your acne?”). ■