

## **Acne and smoking: results from an Italian multicentric study.**

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### **Introduction:**

Several skin diseases are known to be associated with cigarette smoking, but few contrasting data have been published about a possible correlation between acne and smoking. Of the published studies, some suggested a positive (1,2), some a negative and some no association between smoking and acne (3,4). This may be related to methodological issues such as study populations, use of different disease classifications, and adjustments for confounding variables. Moreover, published statistics have been mainly carried out on juvenile acne or on non homogenous population.

Results from an Italian observational study revealed a strong prevalence of a peculiar, non-inflammatory form of acne among adult female smokers (5). DDI (Donne Dermatologhe Italia) association, during a campaign on the general cutaneous effects of smoking, conducted a multicentric observational study to assess the frequency of this form of acne on an extended female population.

### **Methods**

Study population were women between 25 and 50 years old, recruited among the patients of 46 Italian dermatologic sites. Each site was requested to enroll at least 20 consecutive non smokers women. 20 consecutive female smokers were enrolled among dermatologic outpatients . For each subject, data were collected (by a questionnaire) about age (25-35 yrs, 36-45 yrs, 46-55 yrs), smoking habit, hormonal pathologies, age of acne onset, acne severity. A dermatological visit was performed in order to assess the presence and the severity of acne.

### **Results**

A total of 1822 subjects were enrolled (918 smokers, 904 non-smokers). Healthy subjects were 1322, vs 500 patients with acne. Subjects were homogeneously distributed into the 3 age-groups without differences between smokers and no-smokers, acne and healthy subjects. Among patients with acne, smokers were 274 ( 54.8%) while among healthy subjects they were 644 (48.7 %)(fig.1)( $p < 0,05$ )

Among acne patients, late onset acne was more frequent among smokers (47,9%) than in non-smokers (34,1%) (fig.2) ( $p < 0,05\%$ ).

Data about acne severity and associated hormonal pathologies were incomplete and were not considered for statistical analysis.

## **Discussion.**

The results seem to confirm that adult female acne is more frequent among smokers. Several experimental studies demonstrate that smoking, and nicotine in particular, can interfere with at least 3 major aspects of acne pathogenesis.

It's commonly accepted that smoking provokes significant alterations on the skin microcirculation, on keratinocytes and on the collagen and elastin synthesis. Nicotine induces vasoconstriction associated with local hyperaemia. It inhibits inflammation through effects on central and peripheral nervous system and through direct effect on immune cells (6).

A relative deficiency in antioxidants caused by smoking with consequent alterations in sebum composition (peroxidation on sebaceous lipids) has been demonstrated (5). This property could contribute to the onset and/or exacerbation of this pathology in subjects already predisposed to this disease.

Finally, smoke can contribute to acne pathogenesis by acetylcholine nicotinic receptors.

Nicotinic receptors are heterodimers composed by the combination of different subunits.

This traduces in virtually unlimited combination possibilities, and can explain the different genetic susceptibility to different cholinergic substances partly in clinical use, like nicotine.

Blocking of ACh receptors results in a complete inhibition of epidermal differentiation and proliferation to the point of epidermolysis. In contrast, stimulation with cholinergic drugs,

and nicotine in particular, traduces in an increased epithelial thickness and an improved epithelial maturation. Nicotinic receptors are expressed also on sebocytes and on

sebaceous glands. So sebocyte differentiation, sebum production or sebum composition may be altered by endogenously produced ACh acting in a paracrine manner or

exogenously by tobacco-derived nicotine. The highest concentration of both ACh, AChR and nicotine can be found at the infundibulum of the pilosebaceous unit suggesting an

important role of the cholinergic system in acne vulgaris (8).

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