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Smoking in Dermatologic Disease: May the Skin be a Motivation to Quit?

Juan Garcias-Ladaria*

Department of Dermatology, Hospital Universitari Son Espases, Spain

***Corresponding author:** Juan Garcias-Ladaria, Department of Dermatology, Hospital Universitari Son Espases, Spain

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ABSTRACT

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Introduction

The tobacco epidemic is one of the biggest public health issues, killing globally more than 8 million people yearly. Up to half of tobacco smokers will die from its use [1]. Top causes of mortality like vascular diseases along with several types of cancer, are clearly promoted by smoking. In addition, tobacco is linked to numerous health problems that alter daily life and working ability of smokers. Preventing not only mortality, but also morbidity associated with smoking should be a priority for physicians and policymakers. Nicotine and other toxic products of tobacco, including polycyclic aromatic hydrocarbons, nitrosamines, and heterocyclic amines, exert harmful effects on virtually any organ system of the human body. The skin is not an exception. Cutaneous signs of smoking include premature skin wrinkling, hair greying, mucosal hyperkeratosis (leukokeratosis) and pigmentary alterations of fingers, mouth and perioral skin as a result of direct staining by tobacco by-products or induction of melanogenesis [2]. In addition, many skin diseases are more frequent in smokers (Table 1), [2-8] and smoking has been related to poorer response to dermatologic treatments such as antimalarials and TNF inhibitors. Three mechanisms have been hypothesized as the cause of skin damage induced by smoking: induction of reactive oxygen species (ROS) and oxidative stress, vascular phenomena, and direct effect of chemicals on keratinocytes, fibroblasts and inflammatory cells [9-10]. Nicotine is the main alkaloid found in tobacco.

It acts on several subtypes of nicotinic acetylcholine receptors, which are found in keratinocytes and inflammatory cells, inducing epidermal proliferation, keratinocyte adhesion, and stimulating the release of pro-inflammatory cytokines. Cigarette smoke contains also high levels of agonists for arylhydrocarbon receptor (AhR). AhR plays a key role in immune regulation and promotes Th17 cell proliferation and regulatory T cell downregulation [11] . Th17 cells are key cells in the pathogenesis of many inflammatory diseases, including psoriasis, atopic dermatitis, hidradenitis suppurativa, alopecia areata and pityriasis rubra pilaris. In addition, cigarette smoke also appears to alter the scarring process by downregulating collagen synthesis, stimulating matrix metalloproteinases and inducing vasoconstriction and ischemia. Hypoperfusion of dermal hair papilla is thought to be also implicated in hair damage. The evidence supporting a pathogenic role of smoking in each one of the diseases is variable. For Berger's disease, smoking is a conditio sine qua non for the disease. There has never been a well-documented case in a patient who was proven not to smoke, and smoking cessation is considered essential for the control of the disease [12]. For the rest, smoking is probably one of many triggers in previously predisposed individuals. However, prospective cohort studies evaluating the incidence of disease in smokers compared to non-smokers are scarce.

At best, case-control or retrospective studies show a higher prevalence of smoking in individuals affected compared to healthy controls, or a worse prognosis in smokers. These studies are not optimal to establish causality. Likewise, prospective studies evaluating the effect of smoking cessation on skin disease are

lacking. Recommendations on smoking cessation in clinical guidelines are rather based on the prevention of vascular disease and mortality, the risk of which is independently elevated in the context of most severe cutaneous inflammatory diseases. Quality of life is severely altered by skin disease due to the intensity of symptoms and social stigmatization. Psoriasis, for instance, may cause as much disability in physical and mental functioning as major diseases such as cancer, arthritis, heart disease, diabetes, or depression [13]. In the case of hidradenitis suppurativa, a disease characterized by intensively painful flexural skin lesions in young individuals, the impairment of personal and professional life is even higher [14]. In one study, absenteeism from work was reported for 50–58% of patients [15]. Daily living with skin disease may be more convincing for patients to stop smoking that the abstract prospect of a heart attack or cancer. Solid evidence of an improvement in their symptoms after smoking cessation would help motivating our patients in daily practice. In conclusion, avoid smoking may have a role in preventing the incidence of skin disease and improving the prognosis of affected individuals.

To stop smoking is essential for our patients to prevent major causes of death like cardiovascular events. We need to keep exploring the relationship of smoking and skin disease, particularly with prospective studies that evaluate the effect of quitting on the dermatologic process.

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