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REVIEW ARTICLE

Smoking and the Skin

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Abstract. Smoking is the main modifiable cause of disease and death in the developed world. Tobacco consumption is directly linked to cardiovascular disease, chronic bronchitis, and many malignant diseases. Tobacco also has many cutaneous effects, most of which are harmful. Smoking is closely associated with several dermatologic diseases such as psoriasis, pustulosis palmoplantaris, hidrosadenitis suppurativa, and systemic and discoid lupus erythematosus, as well as cancers such as those of the lip, oral cavity, and anogenital region. A more debatable relationship exists with melanoma, squamous cell carcinoma of the skin, basal cell carcinoma, and acne. In contrast, smoking seems to protect against mouth sores, rosacea, labial herpes simplex, pemphigus vulgaris, and dermatitis herpetiformis. In addition to the influence of smoking on dermatologic diseases, tobacco consumption is also directly responsible for certain dermatoses such as nicotine stomatitis, black hairy tongue, periodontal disease, and some types of urticaria and contact dermatitis. Furthermore, we should not forget that smoking has cosmetic repercussions such as yellow fingers and fingernails, changes in tooth color, taste and smell disorders, halitosis and hypersalivation, and early development of facial wrinkles.

Key words: tobacco, smoking, skin, aging.

HÁBITO DE FUMAR Y PIEL

Resumen. El hábito de fumar constituye la principal causa evitable de enfermedad y muerte en el mundo occidental. Su consumo se relaciona directamente con enfermedades cardiovasculares, bronquitis crónica y numerosos procesos neoplásicos. El tabaco ejerce asimismo múltiples y diversos efectos sobre la piel, la mayor parte de ellos nocivos. En este sentido, el hábito de fumar se halla estrechamente relacionado con numerosas enfermedades dermatológicas como la psoriasis, la pustulosis palmoplantar, la hidrosadenitis supurativa, el lupus eritematoso sistémico y discoide, así como con procesos neoplásicos del labio, de la cavidad oral y de la región anogenital, entre otros. Existe una relación controvertida con el melanoma, el carcinoma escamoso de la piel, el carcinoma basocelular y el acné. Por otra parte, este hábito parece que ejercería un efecto protector frente al desarrollo de las aftas orales, de la rosácea, del herpes simple labial, del pénfigo vulgar y de la dermatitis herpetiforme. Aparte de la influencia que el hábito de fumar ejerce sobre las enfermedades dermatológicas, el consumo de tabaco es también responsable directo de distintas dermatosis como la estomatitis nicotínica, la lengua negra vellosa, la enfermedad periodontal, así como de algunos tipos de urticaria y de dermatitis de contacto. Además, no hay que olvidar la repercusión cosmética de este hábito, que conlleva una pigmentación amarilla de los dedos y de las uñas, una alteración del color normal de los dientes, una disminución del gusto y del olfato, halitosis e hipersalivación y un desarrollo precoz de arrugas faciales.

Palabras clave: tabaco, fumar, piel, envejecimiento.

Introduction

Smoking, which was an uncommon habit in 1900, reached epidemic levels during the last century and peaked in 1964, the year in which 40% of adults in the United States of America were smokers. Since then, tobacco use has declined gradually, although 28% of adults in developed countries are still smokers. Over the last decade, the number of smokers in Spain has declined slightly to around 30% of the adult population. This decline represents a reduction

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Table 1. Toxic Constituents of Tobacco

Solid Phase (Particles)	*Gas phase*
Nicotine	Carbon dioxide
Phenol	Carbon monoxide
Catechol	Hydrogen cyanide
Quinoline	Nitrogen oxides
Aniline	Acetone
Toluidine	Formaldehyde
Nickel	Acrolein
N-Nitrosodimethylamine	Ammonium
Benzopyrenes	Pyridine
Benzanthracene	3-Vinylpyridine
2-Naphthylamine	N-Nitrosodimethylamine
	N-Nitrosopyrrolidine

in the number of male smokers, while the number of women who smoke has increased during the same period.

Smoking is the leading preventable cause of disease and death in the Western world, and accounts for some 20% of deaths in these countries. Worldwide, approximately 2 million people die every year because of smoking, half of them under 70 years of age. The tissues and organs directly exposed to the smoke, such as the airways, bear the brunt of the primary effects of tobacco use. Furthermore, numerous components of tobacco smoke and its active metabolites can have specific toxic and carcinogenic effects on a number of different organs. For example, the association between smoking and the following diseases is well known and has been studied in depth: chronic bronchitis, pulmonary emphysema, ischemic heart disease, and cancers of various organs including the lungs, mouth, pharynx, larynx, esophagus, uterine cervix, kidney, and bladder. Approximately 30% of all cancer deaths in the Western world are due to smoking. Although the epidemiological association between cigarette smoking and these diseases has been clearly established, the underlying mechanisms are as yet poorly understood.

Tobacco smoke comprises a volatile or gas phase and a solid or particulate phase (Table 1). The gas phase, with around 500 components, represents 95% of the weight. The particulate phase, which represents 5% of the weight, is composed of approximately 3500 constituents, the most important of which is nicotine alkaloid. These are the substances responsible for the broad range of toxic effects tobacco smoke has on the tissues and organs of the human body. The skin is an organ exposed to cigarette smoke both directly through contact with environmental smoke and indirectly in the form of the toxic substances that pass into the bloodstream from the inhaled smoke. It is not surprising, therefore, that smoking affects the skin in many different ways, most of which are harmful.

The Effects of Smoking on the Skin

In order to provide a clearer analysis of the repercussions of smoking on the skin, this review is divided into 3 sections: the first of these deals with the influence of smoking on the development and course of a series of skin diseases, the second reviews the skin diseases caused by smoking, and the third is a discussion of the cosmetic effects of tobacco use.

Smoking and Skin Disease

This section is a review of the literature dealing with the effects of smoking on the incidence and course of various skin diseases (Table 2).

Psoriasis

In recent years, a large number of studies have shown a relationship between smoking and psoriasis.[1-8] From an analysis of this research we have been able to draw the following conclusions: smoking is associated with an increased risk of developing psoriasis, particularly the pustular forms, and this association is particularly significant in women; smoking reduces the response to psoriasis treatment; smokers and ex-smokers have a higher risk of developing severe psoriasis than nonsmokers, and this risk is directly related to the intensity of the habit (number of cigarettes smoked per day) and cumulative consumption (pack-years, calculated by multiplying the number of packs smoked per day by the number of years the person has smoked).

However, the pathogenic mechanisms of this association remain unclear. When Sonnex et al[9] analyzed the in vivo response of polymorphonuclear leukocytes (PMN) to a standard chemotaxin, they observed that the PMNs of the psoriatic smokers responded to a greater degree than the PMNs of nonsmoking psoriatic patients, control smokers, and control nonsmokers. This finding would suggest that smoking had an effect on the PMNs of the psoriatic patients, although other, as yet unidentified, factors are probably also involved.

Table 2. The Effects of Smoking on Certain Skin Diseases

Increased Incidence and/or Exacerbation	*Decreased Incidence and/or Improvement*	*Relationship Unclear*	*No Relationship*
Psoriasis	Rosacea	Melanoma	Atopic dermatitis
Palmoplantar pustulosis	Oral aphthous ulcers	SCC of the skin	Mucosal pemphigoid
Scar formation	Herpes simplex labialis	Basal cell carcinoma	Oral lichen planus
SCC of the lip	Pemphigus vulgaris	Acne	
SCC of the oral cavity	Dermatitis herpetiformis		
Anal and genital SCC			
Hidradenitis			
Allergic contact dermatitis			
Favre-Racouchot syndrome			
Systemic lupus erythematosus			
Discoid lupus erythematosus			

Abbreviation: SCC, squamous cell carcinoma.

Palmoplantar Pustulosis

Several authors have found a significant association between smoking and the development of palmoplantar pustulosis (PPP). In a large multicenter study, O'Doherty and Macintyre[10] found that 80% of patients with PPP were current smokers at the time of disease onset, compared to only 36% of the controls. The relative risk of PPP in smokers compared to nonsmokers was 7.2. Moreover, 90% of these patients are women, and women smokers are 74 times more likely to develop PPP than nonsmoking women of the same age.[11] Smoking cessation appears to be associated with an improvement in PPP.[12]

As occurs in the case of psoriasis, many factors other than smoking—such as stress, personality, and genotype—probably intervene in the etiology and pathogenesis of this disease.[10] Based on the results of a study undertaken to determine the presence of serum antibodies to nicotinic acetylcholine receptors in patients with PPP, Hagforsen et al[13] advanced the hypothesis that PPP may be an autoimmune disease partly induced by tobacco smoke. Evidence of a high prevalence of thyroid dysfunction in patients with PPP would support this hypothesis.[14]

Autoimmune Blistering Diseases

Although bullous pemphigoid is the most common autoimmune blistering disease, no research has been published on its relationship with smoking. Only 2 studies analyzing the relationship between mucosal pemphigoid and smoking have been published, neither of which found any significant association.[15,16]

The authors who have studied the relationship between tobacco use and pemphigus vulgaris reported that smokers and ex-smokers are less likely to develop this disease than nonsmokers.[17,18] Mehta and Martin[19] reported the case of a patient with pemphigus vulgaris resistant to combined therapy with systemic corticosteroids, cyclophosphamide, and sulfones whose improvement after he started smoking again was sufficient to warrant withdrawal of treatment 2 months later.

Similarly, studies that analyzed tobacco use in patients with celiac disease[20-22] and dermatitis herpetiformis[23,24] found an inverse association between smoking and these entities. Prasad et al,[25] who investigated possible etiologic and pathogenic mechanisms, reported that the incidence of endomysial-antibody-positive cases among adults recently diagnosed with celiac disease was lower among smokers than nonsmokers. By contrast, McMillan et al[26] found no relationship between smoking and the presence of anti-gliadin antibodies.

Postsurgical Wound Healing

The toxic components of cigarette smoke, in particular nicotine, carbon monoxide, and hydrogen cyanide, interfere with the processes involved in wound repair.[27] Smokers develop more skin-related postsurgical complications than nonsmokers, including the

development of more unsightly scars,[28] a higher frequency of suture failure,[29] hair loss in the area of surgical intervention,[30] and poorer outcomes with skin flaps and full-thickness grafts. In one study, the risk of total or partial loss of flaps and grafts was 37% among active smokers compared to 17% among nonsmokers and patients who had not smoked for at least a year prior to surgery.[31] This risk has been shown to be dependent on the number of cigarettes smoked per day.[32] In light of this evidence, patients should be advised of the need to stop smoking before and after surgery. Although the minimum period during which smoking should be avoided is not well established, it appears that abstinence for as little as 4 weeks before surgery is associated with improved scarring and healing, and abstinence for between 5 days and 4 weeks is recommended after surgery.[33,34]

Skin Cancer

Tobacco smoke contains more than 40 mutagens and carcinogens, notably polycyclic aromatic hydrocarbons, various nitrosamines, and heterocyclic amines. It is the presence of these substances and the immunosuppressive effect of nicotine that makes tobacco the causative agent of cancer in numerous organs. The organs that come into direct contact with the smoke—including the oral cavity, esophagus, lungs, and bronchus—are at greatest risk, but smokers also have a greater risk of developing malignant disease at other sites, such as the cervix, pancreas, bladder, kidney, stomach, and hematopoietic system.[35] The skin is an organ highly exposed to tobacco smoke and its carcinogens, both through direct contact and by way of systemic circulation, so that it would not be surprising if smokers were found to be at higher risk for developing skin cancer than nonsmokers. However, the relationship between tobacco and malignant diseases of the skin has not been confirmed in all kinds of skin cancers.

1. *Melanoma*. Until a few years ago, the many studies undertaken to evaluate the relationship between smoking and the development of melanoma had not provided sufficient evidence to establish the influence of smoking on the incidence of this cancer. Prognostic implications had, however, been demonstrated by a number of studies.[36-41] The authors of these studies reported that smokers more often presented metastasis at diagnosis, and that the disease-free interval after diagnosis was shorter, and mortality from melanoma greater in smokers than in nonsmokers. More recently, however, 2 studies with very significant results have been published. In 2003, Freedman et al[42] found that long-term smokers had a lower risk of developing melanoma than nonsmokers, although they found no association between this effect and the number of packs smoked per day. These findings were confirmed in 2007 when Odenbro et al,[43] who undertook a large, well-controlled cohort study, found that smoking was inversely associated with cutaneous melanoma. In that study, nonsmokers were 35%-50% more likely to develop cutaneous melanoma than smokers and 25% more likely than ex-smokers. The risk of developing melanoma was found to be inversely associated with the number of years of smoking and cumulative tobacco consumption (pack-years). The authors of that study suggested that the mechanism of action might be the immunosuppressive effect of tobacco, which might protect the melanocytes from inflammatory reactions caused by UV radiation. Another possible explanation is that smokers tend to be less physically active than nonsmokers and therefore spend less time outdoors.
2. *Squamous cell carcinoma of the skin*. Most of the studies carried out to determine the role of smoking on the development of squamous cell carcinoma of the skin demonstrated a statistically significant association between smoking and this malignant process.[44-48] However, some other studies did not find any relationship.[49,50] Grodstein et al[46] prospectively analyzed the development of squamous cell skin cancer in relation to a variety of factors, including phenotype, sun exposure, and cigarette smoking, over an 8-year period in a cohort of 107 900 individuals, predominantly white women. The results of that study showed that current smokers were 50% more likely to develop squamous cell skin cancer than people who have never smoked. Karagas et al[47] and De Hertog et al [48] also found an association between smoking and squamous cell carcinoma of the skin, with current smokers having the highest risk, followed by former smokers, and finally nonsmokers. This risk was related to the number of years as a smoker and the number of cigarettes or pipes smoked.
3. *Squamous cell carcinoma of the lip*. Most studies on the relationship between tobacco use and lip cancer have found smoking to be a risk factor for the development of dysplastic and malignant lip lesions.[51-53] As the population of smokers is large and only a few of these people develop lip cancer, other factors are probably involved, including phenotype, sun exposure (both exposure at an early age and cumulative exposure in individuals who work outdoors), and alcohol consumption, among others.[54,55] Nevertheless, approximately 80% of the patients who develop lip cancer are smokers.[51]
4. *Squamous cell carcinoma of the oral cavity*. Squamous cell carcinoma accounts for approximately 90% of

oral cancers, and the association of this entity with smoking is well documented.[56-59] Tobacco use is responsible for approximately 91% of oral cancer in men and 59% in women.[60] All types of tobacco and the various ways of consuming it, including holding the smoke in the mouth without active inhalation, increase the risk of oral cancer. This risk is greater in women[61] and in smokers of nonfilter cigarettes compared to smokers of filter tip cigarettes,[62] and it is closely related to the number of pack-years reported.[63,64] Consequently, tobacco cessation brings about a substantial decrease in the incidence of leukoplakia and carcinoma of the oral mucosa.[65] Moreover, heavy alcohol consumption has a synergistic effect when combined with tobacco use, significantly increasing the risk of developing oral carcinomas.[66] A review of this topic cites many studies that found associations between smoking and numerous genetic and nongenetic changes in cells of the oral mucosa, including DNA polymorphisms, micronuclei, chromosomal abnormalities, and increased adherence of cancer-associated bacteria.[67] The same review reports that a number of studies have found correlations between such buccal cell changes and the development of malignant tumors.

5. *Squamous cell carcinoma of the anogenital region*. Smoking is associated with an increased risk of developing carcinomas of the penis, vulva, cervix, and anus, but not of the vagina.[68-72] This increase is dose-related, and tobacco cessation results in a reduction of the associated risk. In addition to increasing the risk of squamous cell carcinoma of the vulva,[73,74] smoking also reduces survival in these patients,[75] and is associated with a greater frequency of high-grade lesions.[69] Smoking also interacts with genital warts, and women with both these risk factors have 35 times the risk of developing vulvar cancer than those with neither.[76]
6. *Basal cell carcinoma*. The results of studies on the relationship between smoking and basal cell carcinoma are inconsistent. Boyd et al[77] found a higher percentage of smokers in a group of young women with basal cell carcinoma as compared to a control group. Similarly, Wojno[78] and Milan et al[79] both found a statistically significant association between smoking and the risk of developing basal cell carcinoma in women, but not in men. Erbagci and Erkilic[80] reported a higher frequency of sclerodermiform than solid basal cell carcinomas among smokers, and suggested that smoking may induce the differentiation of basal cell carcinoma towards morpheaform forms. However, other authors found no association between tobacco use and basal cell carcinoma.[48,81-83]

Other Skin Diseases

1. *Aphthous ulcers*. The results of various studies suggest that nicotine may exercise a protective effect against aphthous ulcers, since a higher incidence of aphthous ulcers was observed among nonsmokers than smokers.[84-86] Furthermore, tobacco cessation has been shown to result in a worsening of the ulcers, which improve after resumption of the habit.[87] The hypothesis that the mucosal hyperkeratinization associated with smoking affords protection against aphthous ulcers has been advanced as a possible explanation for this phenomenon.
2. *Acne*. The results of studies concerning the effects of smoking on acne are inconsistent. Several studies have found an inverse association between tobacco use and the development of acne lesions in both men and women,[88] only in men,[89] and only in women.[90] However, Firooz et al[91] found no association between smoking and acne. By contrast, Shäfer et al[92] and Chuh et al[93] found a higher prevalence of acne among smokers than among nonsmokers, and a dose-dependent relationship between acne severity and the number of cigarettes smoked per day.
3. *Rosacea*. The percentage of smokers among patients with rosacea is lower than in the population in general.[94]
4. *Hidradenitis suppurativa*. König et al[95] found a higher proportion of smokers among patients with hidradenitis suppurativa than among healthy controls (88.9% and 46.0%, respectively; odds ratio, 9.4).
5. *Contact dermatitis*. Smoking appears to be a risk factor for the development of allergic contact dermatitis.[96-98] Linneberg et al[98] found smoking to be significantly associated with positive patch test results (True test), positive patch test reactions to nickel, and allergic nickel contact dermatitis. These associations showed a dose-dependent relationship with the quantity of tobacco consumed and were independent of age, sex, and prior exposure to nickel. Moreover, smoking itself was a cause of contact dermatitis, as we will discuss in the following section.
6. *Atopic dermatitis*. Mills et al[99] found no differences in smoking prevalence between patients with atopic dermatitis and a control group.
7. *Lupus erythematosus*. Most studies on the subject show that smokers are at greater risk for both systemic and discoid lupus erythematosus than nonsmokers, and that this risk is significantly associated with the number of cigarettes smoked per day.[100-105]
8. *Favre-Racouchot syndrome*. According to a study by Keoug et al,[106] smoking appears to play an important role in the pathogenesis of Favre-Racouchot syndrome. In that study, the incidence of the syndrome was higher among smokers than nonsmokers, and in the

population of smokers studied, tobacco consumption was greater in those who presented these lesions than in the unaffected individuals.

9. *Lichen planus*. Studies that have analyzed the effect of smoking on lichen planus, and specifically oral lichen planus, have not found any relationship between smoking and the development or course of this disease.[65,107-109]
10. *Herpes simplex virus*. Axell and Leidholm[110] found that recurrent herpes labialis was less prevalent among smokers than nonsmokers. Tobacco smoke inhibits the replication of the herpes simplex virus and reduces its cytolytic effect, thereby reducing the intensity and frequency of outbreaks of herpes labialis. However, since the oncologic activity of herpes simplex virus is inversely related to its cytolytic activity, tobacco smoke, by inhibiting cytolysis, will act as a carcinogenic factor.[111]

Skin Diseases Caused by Smoking

This second section deals with the skin diseases in which the components of cigarettes and tobacco smoke are the main, and at times the only, known causative agents, and in which tobacco cessation results in an improvement and even resolution of the process.[65,112-114]

Oral melanosis or smokers' melanosis is a benign pigmentation of the oral mucosa that takes the form of grayish-brown macular lesions caused by tar deposits and an increase in the deposition of pigment in the oral mucosal keratinocytes.[115]

Several studies have reported the development of occupational tobacco dermatitis among tobacco farm workers and workers employed in cigarette and cigar factories.[116,117] Furthermore, smoking itself can cause contact dermatitis, most often affecting the face and hands.[118-121] The characteristic presentation described involved the appearance of 2 parallel brown pigmented bands on the upper lip situated on either side of the philtrum, sometimes accompanied by brown pigmentation on the rest of the face.[118,119] In these cases, patch test results were positive for unsmoked tobacco, and doubtful or negative for smoked tobacco, nicotine, and tar, as well as smoked and unsmoked cigarette papers and filters. These results suggest that the allergen or allergens responsible are likely to be volatile substances contained in the cigarettes. The fragrances in certain scented cigarettes may be among the agents that cause contact dermatitis.[119-122] Notwithstanding the negative patch test results for nicotine reported in the cases mentioned above, there are 2 reasons why the possibility that this substance may play a role in the contact dermatitis of smokers cannot be ruled out. Firstly, cases have been reported of contact allergy to the nicotine in transdermal patches.[123] Secondly, Lee et al[124] reported the case of a smoker affected by recurrent generalized urticaria in whom the results of an intradermal test with nicotine base and a provocation test with a nicotine patch were both positive.

Smoking has also been associated with urticaria, both generalized[124] and affecting the lip.[125] Apart from the tobacco smoke itself, the triacetylglycerine found in cigarette filters and the epoxy resins used in the manufacture of certain pipes are also thought to be responsible for these allergies.

Nicotine stomatitis or leukokeratosis of the palate is characterized by the development of asymptomatic, occasionally warty, macular plaques on the rear two thirds of the hard palate. It is more common in pipe smokers and is caused more by tar than by nicotine.[112,126]

Black hairy tongue is characterized by a hyperplasia of the papillae on the dorsal surface of the tongue accompanied by the deposit of black pigment. Apart from smoking, antibiotic use, excessive bacterial growth, and tea or coffee drinking also contribute to this condition.[112,126-128]

Finally, tobacco smoke also plays a key role in the development of periodontal disease and its sequelae. According to a study by Tomar and Asma,[129] tobacco is responsible for over half of the cases of periodontal disease in adults in the United States of America. This study also demonstrated that the incidence of periodontal disease declines in former smokers in direct relation to the number of years since cessation.

The Cosmetic Effects of Smoking

In addition to the morbidity and mortality described above, tobacco smoke also has a series of other effects that give rise to various characteristics typical of smokers. These include yellow-stained fingers and fingernails, changes in tooth color, a reduced sense of taste and smell, hypersalivation, smoker's breath, dysphonia, and the smell of smoke on hair and clothes.

Facial skin is significantly exposed to both the sidestream smoke from the burning end of a lit cigarette and mainstream smoke inhaled and exhaled by the smoker. This direct contact between tobacco smoke and skin reduces the moisture level of the stratum corneum and contributes to the greater dryness of facial skin found among smokers.[130] Although the possible role of mechanical factors has not been well documented, it has been suggested that the pursed position of the smoker's lips while smoking and the knitted brow that may be a response to the irritative effect of smoke in the eyes could contribute to more pronounced development of lines around the mouth and the outer corner of the eyes, respectively. However, if the role played by these factors

were important, both black and white smokers should have a similar number of vertical perioral lines, and this has not been observed. Moreover, similar lines have not been reported in individuals, such as flute and trumpet players, whose occupation involves regular contraction of the facial muscles. Nonetheless, the possibility that such factors may play some role in the formation of perioral wrinkles cannot be ruled out.[131,132] On the other hand, several studies have shown that cutaneous elastosis is a marker of both actinic and thermal damage.[133] In one study, the experimental exposure of laboratory animals to isolated infrared radiation resulted in an increase in the number and thickness of elastic fibers similar to that found in solar elastosis.[134] Furthermore, an exacerbation of elastosis has been reported in individuals exposed to a heat source on a daily basis, such as bakers, firemen, and glass blowers.[133] In light of these findings, it is also possible that continuous exposure to a heat source such as a burning cigarette could contribute to the increased elastosis found in the facial skin of smokers.[135]

A number of studies published in recent years have shown that smoking is a risk factor for the development of facial wrinkles among white individuals.[136-152] This risk is independent of age and exposure to sun, and is directly related to cumulative tobacco consumption. However, only 27.5% of heavy smokers with a history of over 50 pack-years have particularly wrinkled facial skin.[142] Several studies on the effects of smoking on photoprotected skin have suggested that smoking by itself does not modify the appearance of the skin, although it may intensify damage caused by solar radiation.[138,146,153] These conclusions were based on the fact that white smokers had apparently normal skin in photoprotected areas and black smokers had normal skin in both photoexposed and photoprotected areas. However, Helfrich et al,[154] who investigated this question in greater depth in a recent study, found a correlation independent of age between the number of packs of cigarettes smoked per day and skin aging on the inner side of the arm as assessed by standardized photographs. Studies that have investigated the histopathologic substrate underlying these changes have shown an increase among smokers as compared to nonsmokers in the number and density of elastic fibers in the dermis of both photoexposed[135] and photoprotected skin.[155-157] This increase, like the development of facial wrinkles, is dose dependent and correlates with cumulative tobacco consumption.[157] Furthermore, this increase in the density of elastic fibers in the dermis of smokers appears to be due to a degenerative process rather than to the synthesis of new elastic material. This degenerative process may have a similar effect on the 2 principal components of these elastic fibers, elastin and microfibrils.[157] The similarity between these changes and the alterations found in solar elastosis[158,159] suggests that tobacco and sun exposure may interact in an additive way, thereby increasing the physiologic effects of aging on the skin. Findings obtained in recent years through research at the molecular level appear to support this hypothesis.[160,161] Like UV radiation, tobacco induces the synthesis of metalloproteinases, enzymes that degrade elastin and other structures. The synthesis of these enzymes reaches its highest level when both factors are present. The fact that tobacco smoke has phototoxic properties supports the likelihood of this relationship.[162]

All of these changes in facial skin caused by smoking are dose-dependent and tend to become evident after 35 years of age. As a result of these changes, smokers appear prematurely old and have a duller complexion and more worn and lifeless skin as well as more marked bags under the eyes and more pronounced wrinkles that appear at an earlier age.

Conclusion

Although smoking may have a certain beneficial effect on some skin diseases, it is extremely difficult to say anything in favor of this highly damaging habit. Smoking is currently the leading preventable cause of disease and death in the Western world, and its beneficial effects are insignificant when compared to the dangers of habitual tobacco use for both the smokers and those exposed to secondary smoke. It is up to us, as dermatologists, to convey this information to our patients. We are responsible for encouraging smokers to consider the value of quitting not only to improve their general health and to alleviate specific skin diseases, but also to conserve a healthier physical appearance. Advising patients to stop smoking should be a routine part of our general advice on skin care, in addition to the usual recommendations to reduce exposure to both the sun and other sources of UV radiation.[163]

Conflicts of Interest
The author declares no conflicts of interest.

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