Review

Cigarette smoking as environmental factor influencing psoriasis: a review of clinical, immunological and therapeutic data

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ABSTRACT

Psoriasis is a common chronic immune-mediated disease that affects millions of people worldwide and is characterised by scaly plaques on the skin. Although the true aetiology is complex, including genetic and environmental factors, cigarette smoking itself is known as a significant modifiable environmental variable and risk factor for the susceptibility and the severity of psoriasis. Scientific evidence has demonstrated that smoking, in addition to increasing the likelihood of suffering from the disease, also significantly aggravates its clinical course, therefore resulting in less effective treatments and the induction of systemic comorbidities. In this review, we critically consider, based on the most relevant epidemiological findings, the main molecular and immunological mechanisms by which smoking can impact psoriasis and elucidate the clinical consequences of smoking withdrawal as an essential aspect of the therapeutic approach to patients with psoriasis.

Introduction

Psoriasis is classified as a chronic inflammatory immune-mediated disease. Its symptoms manifest in the skin and the joints, in addition to compromising the entire immune-metabolic system. It is one of the most prevalent skin diseases in the world. Its global prevalence rate is estimated at 2% to 4%. It is generally recognised that environmental factors participate greatly in the genesis of diseases and their clinical management, while genetic factors continue to play a role in susceptibility on a person-by-person basis (1, 2).

Cigarette smoking has been identified as an important environmental factor influencing the severity and distribution of psoriatic lesions. A large quantity of epidemiological research and meta-analyses reveals that there is a very strong relationship between cigarette smoking and psoriasis: smokers have about twice the likelihood of contracting this disease compared with those who do not smoke. This correlation reveals a dose-effect relationship: the risk increases in proportion to daily cigarette consumption exposure period, and total consumption (pack-years). Findings show that women have a higher risk generally, a result interpreted as likely due to hormonal and epigenetic changes unique to each sex (3).

As demonstrated in intermittent studies, heavy smokers have exhibited psoriasis, thereby indicating a robust correlation between smoking and specific forms of psoriasis. It has been demonstrated that smokers are predisposed to the development of psoriasis, with an elevated risk of palmoplantar psoriasis and pustular psoriasis (3, 4). Individuals afflicted with psoriasis already possess a heightened risk of developing psoriatic arthritis (PsA) in comparison to those not affected by the condition. Moreover, the act of smoking has been demonstrated to further amplify this risk. It has been demonstrated that smokers are more prone to the development of PsA, and that this occurs at an earlier age (5).

Patients who smoke have a worse overall clinical condition, including a worse quality of life, bigger lesions and higher PASI scores (6). Smoking not only plays a major role in the predisposition of the disease, but also contributes to the overall inflammatory response systemically. This modulates the subsequent oxidation stress, activation of immune cells and overproduction of pro-inflammatory cytokines such as

TNF-α, IL-6 and IL-17. These factors result in greater systemic comorbidities, resistance to therapy and the systemic chronicity of an inflammatory reaction (2, 6). Smoking makes many biological and conventional treatments ineffective, which raises concerns about adapting treatment protocols for smokers. Despite the numerous correlations observed, there is a paucity of specific studies examining the relationship between cigarette smoking and greater resistance to conventional systemic treatments.

Understanding how smoking interacts with the pathogenesis of psoriasis is crucial for an understanding of the pathophysiology of the disorder and for developing more effective therapies and preventive measures.

Epidemiology

In a plethora of epidemiological studies, it has been found that cigarette smoking is a major risk factor in the development and progression of psoriasis. The year 2000's have seen a number of observational and case-control studies confirm that the occurrence of this disease is connected with cigarette smoking. Large-scale meta-analyses have continued to prove this high-risk association between psoriasis and tobacco smoke exposure as well as provide new insights into the ways genes combine with other environmental factors, such as active and passive smoking exposure for example, to give us chronic skin faults (7).

The meta-analysis by Armstrong *et al*. (8) is probably one of the most robust and widely cited meta-analyses here. The results offer solid and statistically significant evidence that there is an association between smoking and onset of psoriasis. Active smokers are nearly twice as likely to get the disease compared with non-smokers. Even people who have stopped smoking or ex-smokers also show significantly higher risk than those who never smoked. This might indicate a long-term effect of smoking on the immune system and skin homeostasis. This association is particularly pronounced in patients with moderate or severe psoriasis, for whom smoking serves as a significant driver of pathology. And not only is it significant, one review of incidence studies even showed a clear dose-response relationship, with risk rising proportionately to the number of cigarettes smoked daily and total duration of exposure (8).

One of the most recent meta-analyses is by Zhou *et al*. The study results confirmed that current smokers had a 63% greater risk of developing psoriasis than non-smokers. Past smokers also had a significantly higher risk, which indicates that this effect is persistent and cumulative for skin immunobiology over time (9).

From a therapeutic point of view, smoking has been associated with the reduced efficacy of certain biological treatments, mainly anti-TNF and anti-IL-17 agents. But patients who were ever smokers were significantly less likely to achieve a clinical response after 6 months than never smokers. This finding is of practical relevance to doctors, suggesting that smoking exacerbates the disease and reduces both more advanced treatments' effectiveness (9).

Pathogenic mechanisms

Research has identified cigarette smoke as a key environmental factor in the development and progression of psoriasis. The exact mechanisms behind this relationship are not yet fully understood. A series of mechanisms mediate the action of this involvement, innate and adaptive immunity, promoting the release of pro-inflammatory cytokines, cellular redox balance, exacerbating keratinocyte dysfunction and amplifying inflammatory signalling pathway, skin barrier function, epigenetic regulation and nicotine's role (1, 3, 6).

Oxidative stress and chronic inflammation

Cigarette smoke (CS) introduces a large number of free radicals and reactive oxygen species (ROS) into the body, exceeding the body's own antioxidant capacity. This imbalance can damage lipids, proteins and DNA, leading to cellular malfunction and persistent inflammation. The result is oxidative stress.

Oxidative stress (OS) is a pathophysiological mechanism that characterises numerous disease states. The process of lipid oxidation results in the formation of compounds that exhibit characteristics such as cytotoxic and mutagenic properties that influence on the ageing process and the inflammatory response. It has been accepted that lipid metabolism disorders play an important role in the etiopathogenesis of psoriasis (10, 11). OS is increased in psoriasis patients regardless of their smoking status (11).

Paraoxonase (PON)1 represents one of the most significant antioxidant defence systems against lipid oxidation. The enzyme under consideration is capable of both paraoxonase and arylesterase activity. It has been demonstrated to protect against lipid peroxidation, a process instigated by OS, and to counteract the deleterious effects of OS. There are only a few studies that have compared psoriasis patients with healthy individuals in terms of serum PON1⁄arylesterase activity (12). It is evident that smoking has a strong correlation with psoriasis, with the hypothesis that it plays a significant role in the initiation of the condition, as well as in determining its severity and resistance to treatment. It has been hypothesised that the deleterious effects of smoking on the skin are a consequence of increased ROS and decreased antioxidant gene expression. The decreased arylesterase activity observed in smoker psoriasis patients suggests that smoking may be a significant risk factor, potentially exacerbating the severity of psoriasis by increasing oxidative stress in these patients.

Malondialdehyde is a reliable indicator of the state of lipid peroxidation that occurs *in vivo*. Elevated levels of malondialdehyde (MDA) and diminished activity of superoxide dismutase (SOD), a pivotal antioxidant enzyme in the human body, have been identified as potentially contributing to the exacerbation of psoriasis in smokers. In addition to studies which show that MDA levels in patients with psoriasis are significantly higher compared with healthy controls, there are contrasting studies which show that there is no difference (13).

The OS resulting from cigarette smoke triggers various intracellular signalling pathways which are involved in the pathogenesis of psoriasis. One significant pathway is nuclear factor kappa-light-chain-enhancer of activated B cells (NF-ϰB), additionally mitogen-activated protein kinases (MAPK) and the Janus kinase/signal transducer and activator of transcription (JAK/STAT) pathway (5, 6).

This provides a theoretical basis for the hypothesis that chronic inflammation may underlie the early onset and progression of the disease, as well as its reduced response to therapy (10, 11).

Activation of the Th1/Th17 immune system

The inflammatory response represents a vital component in the initiation and evolution of psoriasis.

Psoriasis is a T cell-dependent autoimmune disease. Disease manifestation is orchestrated by proinflammatory CD4-positive T helper cells producing either interferon-γ (Th1) or interleukin (IL)-17 (Th17). These Th1 and Th17 cells interact with dermal dendritic cells and cause an inflammation that mainly involves interferon-γ, tumour necrosis factor, IL-8, IL-12, IL-17, IL-19, and IL-23.

A number of studies have indicated that smoke cigarette (CS) results in an increase in the peripheral blood leukocyte count of approximately 20% to 25%. *In vivo*, CS has been demonstrated to be associated with an increased level of multiple inflammatory markers, including C-reactive protein, interleukin-6, and tumour necrosis factor alpha, in both male and female smokers (14).

Substantial evidence from several studies indicates that smoking might perturb the inflammation of Th17 cells (15). One study showed that the percentage of circulating Th17 (CD3+ cells) in peripheral blood differs significantly from healthy people, smokers with psoriasis and nonsmokers. Importantly, smokers had lower total white cell counts than non-smokers but higher monocytes and eosinophil, similar numbers of lymphocytes and granulocytes (16).

In addition, *in vitro* exposure of resting central memory T cells to tobacco smoke extract (TSE) can induce Th17 production. This exposure also results in increased expression of IL-17 and IL-22. These results provide insights into the Th17 cell generation in the human body and explain the high levels of IL-17 and IL-22 in the psoriatic lesions on the skin of smokers, which fails to control these cytokines efficiently (16). IL-22 is increased in psoriatic lesions and is implicated in keratinocyte hyperplasia (2, 17).

Serum concentrations of IL-22 in patients with psoriasis are well correlated with the Psoriasis Area and Severity Index Score, and therefore, IL-22 is closely related to psoriasis severity. One study demonstrates the relation between tobacco smoke and IL-17 and IL-22, which exacerbate psoriasis. Furthermore, smokers show a marked elevation of dermal dendritic cells and also plasmacytoid cells that mediate IL-23 production. IL-23 is essential for the survival and proliferation of T helper 17 (Th17) cells, which in turn secrete IL-17 and IL-22. This cascade leads to the production of cytokines such as CXCL1 and CXCL8 by keratinocytes, which induces neutrophil infiltration and the formation of psoriatic plaques (15, 18). Activation of Th17 cells through higher levels of smoking could, therefore, potentially lead to higher psoriasis severity. Additionally, increased serum levels of IL-17 and IL-22 could possibly account for the non-responsiveness to conventional treatments as well as the higher incidence of comorbid conditions in patients with psoriasis who are smokers (2-4).

Compromised skin barrier

One critical component in the pathophysiology of psoriasis is the epidermal skin barrier, that conferring biochemical properties that enable resilience against environmental threats and maintain homeostasis. The skin barrier may be conceptualised as common thread of complex interactions between genetics, host immunity, the cutaneous microbiome, and environmental exposures. There is evidence that skin barrier integrity is altered in psoriasis, with a decrease in ceramides (required for cell-cell adhesion and skin barrier function) and structural proteins (filaggrin, loricrin, involucrin). These modifications are conducive to skin permeability and penetration of the skin by pro-inflammatory mediators (7, 19, 20).

Chronic exposure to cigarette smoke can further alter the lipid profile of the stratum corneum (20).

Skin dryness and scaling are features of psoriasis. This can be further aggravated by smoking, which increases oxidative stress and inflammation, and decreases filaggrin and other structural proteins expression (1).

Recent studies have identified nicotinic cholinergic receptors (nAChRs) in human keratinocytes. Prolonged exposure to nicotine has been demonstrated to induce a state of perpetual receptor activity, resulting in alterations to the regulation of intracellular calcium. This has the capacity to exert influence on a range of cellular processes, including differentiation, migration and adhesion. Smokers have significantly shorter ceramide acyl chain length and more unsaturated ceramides, leading to perturbation of the lamellar organisation of lipids and the barrier function. Smoking also promotes upregulation of degradative enzymes (matrix metalloproteinases [MMPs]) with a catalytic activity against epidermal structural proteins and pro-inflammatory cytokine production thus causing inflammation and barrier damage (20).

The issue is complicated by the fact that there are numerous toxic substances in cigarettes that have not yet been identified. Nicotine has been identified as a significant contributor to the excitation of normal signal forms in the nervous system. It is important to note that the cells in proximity to these receptors are keratinocytes, which have the potential to modulate their functioning pathways through transduction. Furthermore, a plethora of cell types have been identified, including immune system cells, such as T cells and B cells, as well as lines of cells from leukaemias or thymocytes. These cells have the capacity, through nicotine-activating pathways, can increase the secretion of cytokines, such as IL-12, IL-2, TNF, INF-α, and the granulocyte monocyte colony-stim-

ulating factor (19, 21).

Epigenetic alterations

Dysregulation of multiple epigenetic mechanisms, including aberrant DNA methylation, altered histone modifications, and miRNA expression has been shown to play a crucial role in the pathogenetic scenarios of psoriasis. Studies have shown that different stages of the disease can manifest unique epigenetic profiles. Epigenomic analyses have discerned thousands of differentially methylated CpG sites in smokers versus non-smokers, suggesting a widespread influence of smoking on the epigenome. Hypermethylation of genes and the modify the activity of histone deacetylases (HDACs) causing immune system dysfunction and these modifications being associated with overexpression of pro-inflammatory genes and hence the chronic inflammation characteristic of psoriasis (9, 22). The relationship between the development of psoriasis and smoking, and the role of epigenetic changes in gene expression despite sharing mutations, needs additional exploration. Understanding how smoking affects the epigenome and how this might impact psoriasis may offer new perspectives for therapeutic options (23).

Effects on clinical outcome and therapeutic response

Psoriasis is a chronic, immune-mediated inflammatory disease characterised by a wide range of phenotypes. The main clinical forms are plaque psoriasis (the most common form, accounting for around 85-90% of cases), guttate psoriasis, pustular psoriasis, erythrodermic psoriasis and inverse psoriasis. The disease can also affect the nails, the scalps and the joints (psoriatic arthritis).

There is now a wealth of evidence suggesting that smoking exacerbates the localisation, refractoriness and disabling manifestations of psoriasis, resulting in clinical forms that are less responsive to conventional and biological treatments (17, 18).

There are no studies in the literature correlating smoking with the onset of a particular variant of psoriasis, but it can be noted that smoking, through oxidative stress, Th1/Th17 activation, and stimulation of nicotinic receptors, may contribute to the onset of the disease and make treatment more difficult, especially in so-called difficult sites. There are several case reports that identify a particular involvement of cigarette smoking in the onset of pustular, palmoplantar, nail, joint, and scalp psoriasis.

Palmoplantar and pustular psoriasis

Pustular psoriasis is characterised by superficial pustulation of the lesions. Lesions are frequently localised to the palms and soles but may be generalised. Localised pustular psoriasis has little systemic effect on the body, but the lesions can be hard to treat and recur frequently. Generalised pustular psoriasis is often associated with fever and malaise, and there may be fluid and electrolyte disturbances and infection. This form of psoriasis has a high rate of relapse and may lead to death. Many patients with generalised pustular psoriasis are treated on an inpatient basis. While palmoplantar manifestations can occasionally occur in plaque psoriasis, it is becoming increasingly evident that these localisations constitute a distinct subgroup in terms of both clinical presentation and pathogenesis, with a strong environmental link, particularly to cigarette smoking (19).

From a clinical perspective, palmoplantar psoriasis is one of the most debilitating forms of the disease. It affects areas essential for locomotion and manipulation, severely limiting quality of life even when the disease does not spread systemically. Patients report pain, deep fissures, bleeding, and difficulty performing everyday tasks, which often has an underestimated psychological impact (17).

Epidemiological studies conducted in Europe and Asia have shown that over 70-90% of patients with palmoplantar pustulosis are active smokers and that smoking often precedes clinical onset. This finding is highly significant as it suggests an etiological role rather than merely an aggravating factor. While the meta-analysis by Armstrong *et al*. (8) highlighted an increased prevalence of smoking in patients with severe psoriasis, it is the study by Benzian-Olsson *et al*. (24) that has clarified the biological mechanisms linking smoking and pustulosis. The study also suggests that PPP symptoms are particularly severe in patients with early-onset disease, women, and current smokers.

Studies, have been conducted in literature, showed that exposure to smoke increases the expression of IL-36γ in tonsillar epithelial cells, a process enhanced by the presence of IL-17A. This synergy between the IL-17/IL-36 axis suggests an inflammatory mechanism amplified by smoking, which could explain the acral localisation and chronic nature of pustulosis (4).

Finally palmoplantar pustulosis has traditionally been classified separately from generalised pustular psoriasis. However, genetic and immunological data support a unified view in which smoking may act as a trigger for localised expression of a systemic process, particularly in genetically predisposed individuals (24, 25).

Nail psoriasis

Nail psoriasis is one of the most common and debilitating manifestations of psoriatic disease. Nail psoriasis is a significant morbidity of the disease with its long duration, and its difficulty of treatment. Common alterations include pitting, onycholysis, subungual hyperkeratosis, splinter haemorrhages, and dyschromia (leukonychia and oil spots) (26).

In the literature, nail psoriasis is seen in 10% to 80% of patients with psoriasis (27). Many studies indicate that quality of life is more affected by nail involvement than by other regions. Nail involvement is significantly higher in patients with psoriasis with cigarette smoking. In the systemic effect, it is already known that smoking increases oxidative stress and cytokines involved in psoriasis pathogenesis. Local angiopathic effects of smoking have been reported to increase nail diseases (10). This marked increase in nail psoriasis in smokers may be due to local angiopathic effects in addition to systemic effects. Smoking reduces blood flow to the nail matrix, compromising its keratinocytes and causing local inflam-

mation. ROS (reactive oxygen species) and the Th17/IL-23 pathway may contribute to nail plate disorganisation and immune cell dysfunction (26, 27).

Nail involvement is linked to a worse disease state, as measured by the Nail Psoriasis Severity Index (NAPSI). Smokers have higher NAPSI scores on average, with larger lesions, more subungual thickening and a higher chance of chronic disease (28).

Another reason for this increase may be that cigarette rituals (lighting, gripping cigarettes) lead to koebnerization. Again, heavy metals and heat in cigarette ash may contribute to the koebnerization effect.

Observational studies have shown that the incidence of nail involvement is significantly higher in psoriatic patients who smoke than in non-smokers, and that the number of cigarettes smoked per day is directly correlated with the number of nails affected. This association remained significant even after correction for the extent of cutaneous psoriasis, suggesting a specific effect of smoking on the nails rather than simply being a consequence of systemic disease severity (26).

Another important factor is therapeutic resistance. Nail psoriasis is notoriously difficult to treat and often requires systemic or biological drugs. This suggests that smoking not only promotes the expression of nail phenotypes but also reduces the effectiveness of treatments by interfering with target immunological mechanisms (28).

From a prognostic point of view, nail involvement in smokers may be an early clinical warning sign. The literature supports the hypothesis that these patients are at an increased risk of developing psoriatic arthritis, and that smoking may act as a cofactor in the development of this condition (26).

Scalp psoriasis

Scalp psoriasis is one of the most common clinical manifestations of psoriatic disease. It affects approximately 50-80% of people with psoriasis and may often be the first or only symptom of the disease. Symptoms can range from mild flaking similar to dandruff to severe inflammation, fissures, bleeding and pain. Cigarette smoking has been identified as an important environmental factor influencing the severity and distribution of psoriatic lesions. There is less evidence than for other types of psoriasis (such as pustular or palmoplantar psoriasis) linking smoking to scalp psoriasis, but clinical and epidemiological data suggest a link, albeit indirect and not direct (2).

While there are no randomised controlled studies that specifically isolate smoking as a predictor of treatment failure in the scalp, the reduced efficacy of drugs in smokers can reasonably be extended to this area (29).

Psoriatic arthritis

Psoriatic arthritis (PsA) is an inflammatory joint condition. As a pleomorphic clinical entity characterised by the development of arthritis in patients with psoriasis, PsA occurs in 6% to 42% of patients with psoriasis and the onset of joint symptoms usually does not happen until 8-10 years' duration of psoriasis skin lesions.

The unfavourable cutaneous and joint effects of smoking have been suggested from past studies due to the impact on the immune system and induction of oxidative stress (30, 31).

US scientists, coordinated by Abrar Qureshi, examined the incidence of psoriatic arthritis. The researchers found that the probability of developing this disease is proportional to the duration and intensity of a patient's smoking career. In particular, those who have smoked for less than 25 years would have a 1.7 times higher risk than non-smokers; this percentage rises to 3.12 for smokers with more than 25 years of smoking behind them. With regard to the number of cigarettes smoked, on the other hand, the statistics presented by the Harvard scholars show that the risk is 1.48 times higher for those who smoke less than 20 packs per year, 3.33 for those who smoke between 20 and 44, and 3.91 above 44 (30).

However, only sparse evidence regarding the association between smoking and PsA has been reported (31-33). One study showed that smoking may accelerate the onset of PsA in patients with psoriasis while delay the onset of PsA in healthy participants. Another case-control study did not find an increased risk of PsA among smokers with psoriasis (33).

To the best of our knowledge, no prospective data on smoking and psoriatic arthritis has been reported thus far (34).

Erythrodermic psoriasis

Erythrodermic psoriasis is a rare but potentially serious form of psoriasis, characterised by widespread skin inflammation involving more than 75-90% of the body's surface area. It is associated with severe erythema and widespread scaling, disrupted temperature regulation, high susceptibility to systemic infection, and potential haemodynamic disturbance. It is a dermatological emergency necessitating meticulous clinical assessment, hospitalisation and prompt systemic therapy.

While the pathogenesis of erythrodermic psoriasis is not completely understood, it is clear that it reflects the clinical end point of either acute or chronic dysregulation of the immune system. This is often in response to specific triggers, such as the abrupt discontinuation of systemic corticosteroids or anti-psoriatic drugs, concomitant infections or systemic diseases, the use of pro-psoriatic drugs (*e.g.*, lithium or beta-blockers), and increasingly recognised pro-inflammatory environmental factors, including cigarette smoking (19). Although the literature on the direct correlation between smoking and erythrodermic psoriasis is limited, some observational studies and cohort analyses have suggested that smoking may trigger or aggravate this clinical form.

Several studies, while not focusing exclusively on erythrodermic psoriasis, have included active smoking among environmental factors with potential systemic impact. These studies have highlighted the role of smoking in perpetuating systemic inflammation, oxidative stress damage, and dysregulation of Th1/Th17 circuits (19, 35). These circuits are also involved in erythrodermic variants. Moreover, as demonstrated in the literature, psoriatic erythroderma manifests more frequently in patients with a prolonged history

of smoking and concomitant cardiovascular and metabolic comorbidities, which are also promoted by smoking. This profile suggests that smoking may act as a destabilising cofactor, predisposing patients to progress from localised chronic forms to acute, widespread inflammatory phases (36).

Discussion

In contemporary psoriasis management, identifying and modifying environmental risk factors is an important step towards improving both the clinical course of the disease and the therapeutic response. Cigarette smoking is one of the most significant environmental risk factors as it is associated with the onset and exacerbation of psoriasis and negatively impacts the response to systemic treatments, particularly biologics (37). However, to date, only one study has explicitly investigated the effect of smoking on the efficacy of traditional systemic therapies, such as acitretin, cyclosporine and methotrexate.

No studies have systematically evaluated the interaction between smoking and clinical response to conventional systemic treatments for psoriasis. This is an important limitation in the current literature, highlighting the need for in-depth investigations.

A greater focus on mild and moderate psoriasis would be desirable, as it is the most common form in the general population and the most frequently treated with traditional therapies. At the same time, more homogeneous studies with adequate samples and rigorous controls are needed to definitively clarify the effect of smoking on the efficacy of systemic and biological therapies.

Consequently, smoking cessation should be considered an integral component of the treatment plan. The clinical approach to cessation should begin with a comprehensive assessment of the patient's medical history. This assessment should include the addiction profile, consumption levels, motivation to change, and any psychiatric or cardiovascular comorbidities. This allows plans to be customised using a patient-centred approach (38).

Organised, repeated motivational counselling interventions are effective in helping people quit smoking, especially when combined with pharmacological support and substitution therapies. Digital resources, dedicated apps and smoke-free centres can also increase the effectiveness of the pathway (38). In this context, the dermatologist plays a strategic role. Growing awareness of the link between smoking and disease progression must lead to active promotion of tobacco cessation as an integral part of treatment, combining strong educational and motivational action with pharmacological indications.

Conclusions

Psoriasis is a long-lasting skin condition, with its prevalence influenced by the interplay of genetic and environmental factors. Clinical evidence suggests that cigarette smoking is an environmental factor that significantly worsens the disease's progression and triggers subsequent flare-ups. The detrimental effects of smoking are associated with the activation of inflammatory mediators involved in the pathological processes affecting the skin of individuals with psoriasis. Smokers are more likely to develop psoriasis compared to the general population. The widespread occurrence of smoking among those with psoriasis may be attributed to a diminished quality of life stemming from emotional challenges and complications in familial and social interactions due to this chronic condition. Therefore, at the current stage, scientific evidence more and more widely stresses that cigarette smoking is an environmental determinant of psoriasis. Smoking is not only a risk factor for developing the disease; however, it has other implications that negatively impact clinical manifestations: severity of the symptoms, distribution of lesions on the body surface area, response to systemic treatments, timeliness of onset for other diseases. In addition, smoking and tobacco are also causing devasting environmental damage.

Although most studies focus on traditional cigarette smoking, the increasing use of electronic cigarettes raises important concerns. Preliminary evidence suggests that the aerosolized components of e-cigarettes may induce oxidative stress, epithelial cell dysfunction, and the activation of inflammatory pathways similar to those triggered by conventional smoking, including the Th17/IL-22 axis. Although current data are still limited, it is plausible that e-cigarette use may contribute to the pathogenesis or exacerbation of psoriasis, warranting further investigation to clarify its immunological and clinical impact.

Dermatologists must not only prescribe drugs but be responsible for encouraging patients to quit smoking. With this in mind, smoking cessation needs to be put on the therapeutic pathway of patients with high priority interventions tailored to their specific condition. Encouraging smoking cessation increases control over the skin disease and considerably reduces overall cardiovascular risk, which can help improve quality of life and facilitate response to systemic drugs. Managing to smoke in patients with psoriasis is both practical and ethical. This approach must be structured, sustained, and interdisciplinary, offering patients not only treatment for their skin but care that better meets individual needs.

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