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Possible Modulators of Smoking-Associated Psoriasis Risk in Postmenopausal Women Include Alcohol and Smoking Cessation

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Introduction

A persistent inflammatory skin condition, psoriasis has a prevalence of 0.9 to 8.5% [1]. Psoriasis has a complex pathogenesis that includes both hereditary and non-genetic variables, such as trauma, exposure to UV light, medications, nutrition, obesity, infections, and psychological stress [2,3]. Inflammation and more unhealthy living choices have been independently linked to diabetes, hypertension, and cardiovascular disease in psoriasis, a T-cell-mediated inflammatory illness that has been classified as a systemic inflammatory disease. However, there is disagreement regarding the connection between drinking and eczema. Comparatively, smoking was more frequently linked to a higher chance of developing psoriasis, as well as the progression of the condition and a poor reaction to therapy (Table 1 and Figure 1) [4-7]. Smoking is a major confounder because it has been linked to both drinking alcohol and dermatitis [8-11]. We sought to ascertain whether drinking patterns or quitting smoking affected postmenopausal women taking part in the Women's Health Initiative (WHI) chance of getting smoking-induced psoriasis. Even though psoriasis and psoriatic arthritis are two distinct conditions, psoriasis typically comes first. Additionally, some doctors designate arthritis and psoriasis with the number 696-0. In the WHI Dietary Modification Study, hazard ratios and 95% confidence intervals were calculated using Cox proportional hazards models divided by age and allocation status [12]. Previous cross-sectional and case-control studies have shown a link among smoking and psoriasis risk, as well as a parenteral partnership smoking and psoriasis. However, the precise impacts on women and men are still unknown, and it is also impossible to determine the risk of psoriasis in elderly women and men. Curiously, increasing oestrogen levels correspond with improvement in psoriasis, whereas smoking is known to have an estrogen-blocking effect [13-15]. Moreover, type 1 psoriasis is more common in younger women, and smoking may affect psoriasis in different ways depending on the kind [16]. Psoriasis has been linked to fluctuations in oestrogen levels [1], but nothing is known about the connection between menopausal hormone therapy and the risk of developing psoriasis.

Table 1: Effect of smoking cessation on psoriasis risk [7].

Cigarettes per day	Current smoker	Past smoker
< 5	Reference	0.98 (0.64 - 1.49)
5 - 4	Reference	0.67 (0.51 - 0.88)[a]
15 - 24	Reference	1.08 (0.80 - 1.45)
> 25	Reference	0.93 (0.62 - 1.37)
Years of smoking	**Current smoker**	**Past smoker**
< 5	Reference	0.96 (0.39 - 2.38)
5 - 24	Reference	0.65 (0.46 - 0.90)[a]
> 25	Reference	1.06 0.86 - 1.32)

Note: Smoking cessation effect on psoriasis was tested by comparing past vs current smokers at baseline. The analysis was stratified across number of cigarettes/day and total years of smoking; [a]Statistically significant.

Methods

The service charge Medicare International Classification of Diseases was utilized to identify patients who had been diagnosed with psoriasis, and self-administered surveys were used to gather data on demographics, medical history, and smoking and drinking habits. In the WHI study group, hazard ratios in Cox regression models were stratified by age and randomization, and ethnicity, income, body mass index, and a history of nonmelanoma skin cancer were considered.

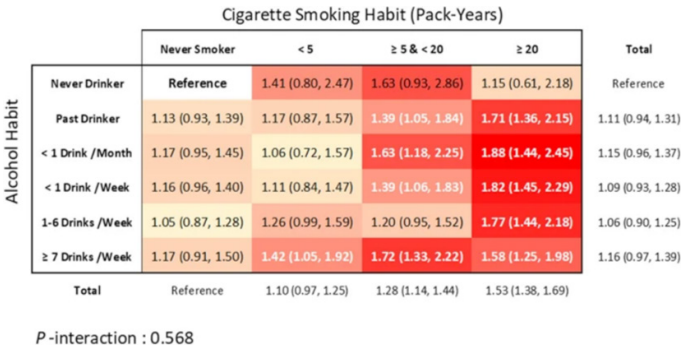

Figure 1: Psoriasis risk based on alcohol consumption habits stratified by cigarette smoking habits. Alcohol (rows) and cigarette smoking (columns) habits on psoriasis risk using "Non-Drinker" and "Never Smoker" as the reference group. Hazard ratios were acquired using a Cox regression model adjusted for ethnicity, income, body mass index, and a history of non-melanoma skin cancer. The model was also stratified by age group, and by Women's Health Initiative trial groups. Text bolded in white are statistically significant. The table cell colors yellow to red indicate increasing hazard ratios [7].

Between 1993 and 1998, women were attracted from 40 US healthcare facilities by mass mailing a recruiting leaflet. Participants in the study took part in four distinct studies: an estrogen-only study, an oestrogen plus gestin study, a diet study, and a calcium and vitamin D study.

Women who had psoriasis prior to joining the WHI as well as those not followed for two years were eliminated from the remaining 112,184 women. The 106,844 postmenopausal women who made up the final study cohort were not included because their smoking or alcohol use history was lacking (n = 5340). Surveys were utilized to gather data on baseline characteristics, health information, and lifestyle habits including smoking status, smoking age. Based on replies to a biannual survey, smoking duration, pack years, and years since stopping were estimated. The gap between the age at which a person first started smoking and the aged at which they stopped for present smokers and past smokers, respectively, was used to determine the duration of smoking. By deducting the age of the last smoking quit from the current age, years since the last cessation were computed. We multiplied the daily cigarette consumption by the amount of smoking years to approximate the number of pack-years.

While enrolled in Medicare Part A and B, psoriasis was classified by a single International Classification of Diseases, Ninth Revision diagnostic code of 6960 (psoriatic arthritis) or 696.1 (other psoriasis) [17,18]. At the conclusion of the two-year monitoring period, participants were placed in the risk group. To ensure that no individuals had psoriasis at the beginning of the research, delayed entry was used to eliminate all systemic psoriasis/psoriatic arthritis cases. If you were no longer registered in the paid service at the time of the event, it was censored; Following the illness, psoriasis was highly connected with alcohol intake, smoking, and bad life events. All statistical analyses were carried out with SAS, all testing was two-tailed, and P and 0.05 were used to define the level of significance.

Conclusion

These results imply that drinking alcohol does not change the probability of psoriasis linked to smoking. In postmenopausal women, smoking, but not drinking, is a risk factor for developing psoriasis. Emphasizing smoking cessation and smoking cessation counselling may be helpful for patients who already have other risk factors for psoriasis since greater pack-years were linked to a greater risk of psoriasis and smoking cessation, and conversely, a decreased risk of psoriasis in moderate smokers Many factors may be in play, while it is hard to determine what makes smoking raise the chance of developing psoriasis. Prolonged smoking exposure has been linked to altered inflammatory and immune functions, especially both adaptive and innate immune responses, which might also worsen psoriasis. Smoking has been linked to the generation of pro-inflammatory cytokines related to psoriasis and has been shown to increase T-cell proliferation through nicotine-induced improved ability of antigen-presenting cells [19, 20]. Moreover, smoking modifies polymorphonuclear cells' shape and functionality, which are responsible for the primary inflammation invades in psoriasis. Smoking can contribute to the development of psoriasis by directly producing oxidants, inducing oxidative stress, and lowering antioxidant levels. Another potential method involves the nicotinic cholinergic receptors found in keratinocytes, which can promote calcium influx and quicken cell differentiation. Moreover, psoriasis patients, particularly some with stopping smoking is clinically relevant even in individuals with existing psoriasis since it has been linked to illness and a lack of response in addition to being a risk factor for psoriasis. The dose-response connection among lymphoid tissue mass and cells is decreased by long-term exposure to the chemical benzo[a]pyrene, which is present in cigarettes. onset of the disease, have a particular psychological profile with heightened anxiety and sadness and may smoke as a kind of self-medication [21]. Smoking cigarettes, which have a higher tar and nicotine content, might hasten immunological alterations. As a result of the findings of this investigation, smoking has been identified as a separate health risk for psoriasis. We saw a steady rise in the rate of psoriasis that was correlated with an increase in the length or severity of current smoking.

References

1. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM (2013) Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol 133: 377-385. https://doi.org/10.1038/jid.2012.339.
2. Fry L, Baker BS (2007) Triggering psoriasis: the role of infections and medications. Clin Dermatol 25: 606-615. https://doi.org/10.1016/j.clindermatol.2007.08.015.
3. Zeng J, Luo S, Huang Y, Lu Q (2017) Critical role of environmental factors in the pathogenesis of psoriasis. J Dermatol 44: 863-872. https://doi.org/10.1111/1346-8138.13806
4. Richer V, Roubille C, Fleming P, Starnino T, McCourt C, et al. (2016) Psoriasis and smoking: a systematic literature review and meta-analysis with qualitative analysis of effect of smoking on psoriasis severity. J Cutan Med Surg 20: 221-227. https://doi.org/10.1177/1203475415616073
5. Fortes C, Mastroeni S, Leffondré K, Sampogna F, Melchi F, et al. (2005) Relationship between smoking and the clinical severity of psoriasis. Arch Dermatol 141: 1580-1584. https://doi.org/10.1001/archderm.141.12.1580
6. Di Lernia V, Ricci C, Lallas A, Ficarelli E (2014) Clinical predictors of non-response to any tumor necrosis factor (TNF) blockers: a retrospective study. J Dermatolog Treat 25: 73-74. https://doi.org/10.3109/09546634.2013.800184
7. Li W, Chan AA, Han J, Chlebowski RT, Lee DJ (2023) Alcohol and smoking cessation as potential modulators for smoking-associated psoriasis risk in postmenopausal women: the women's health initiative. Am J Clin Dermatol 24: 325-332. https://doi.org/10.1007/s40257-022-00750-8
8. Huerta C, Rivero E, Rodríguez LAG (2007) Incidence and risk factors for psoriasis in the general population. Arch Dermatol 143: 1559-1565. https://doi.org/10.1001/archderm.143.12.1559
9. Dai YX, Wang SC, Chou YJ, Chang YT, Chen TJ, et al. (2019) Smoking, but not alcohol, is associated with risk of psoriasis in a Taiwanese population-based cohort study. J Am Acad Dermatol 80: 727-734. https://doi.org/10.1016/j.jaad.2018.11.015
10. Higgins H (2000) Alcohol, smoking and psoriasis. Clin Exp Dermatol 25: 107-110. https://doi.org/10.1046/j.1365-2230.2000.00588.x
11. Li W, Han J, Choi HK, Qureshi AA (2012) Smoking and risk of incident psoriasis among women and men in the United States: a combined analysis. Am J Epidemiol 175: 402-413. https://doi.org/10.1093/aje/kwr325
12. Manson JE, Chlebowski RT, Stefanick ML, Aragaki AK, Rossouw JE, et al. (2013) Menopausal hormone therapy and health outcomes during the intervention and extended poststopping phases of the Women's Health Initiative randomized trials. JAMA 310: 1353-1368. https://doi.org/10.1001/jama.2013.278040
13. Baron JA (1984) Smoking and estrogen-related disease. Am J Epidemiol 119: 9-22. https://doi.org/10.1093/oxfordjournals.aje.a113730
14. Murase JE, Chan KK, Garite TJ, Cooper DM, Weinstein GD (2005) Hormonal effect on psoriasis in pregnancy and post partum. Arch Dermatol 141: 601-606. https://doi.org/10.1001/archderm.141.5.601
15. Hall G, Phillips TJ (2005) Estrogen and skin: the effects of estrogen, menopause, and hormone replacement therapy on the skin. J Am Acad Dermatol 53: 555-568. https://doi.org/10.1016/j.jaad.2004.08.039
16. Asgari MM, Wu JJ, Gelfand JM, Salman C, Curtis JR, et al. (2013) Validity of diagnostic codes and prevalence of psoriasis and psoriatic arthritis in a managed care population, 1996–2009. Pharmacoepidemiol Drug Saf 22: 842-849. https://doi.org/10.1002/pds.3447
17. Feldman SR, Zhao Y, Shi L, Tran MH (2015) Economic and comorbidity burden among patients with moderate-to-severe psoriasis. J Manag Care Spec Pharm 21: 874-888. https://doi.org/10.18553/jmcp.2015.21.10.874

18. Chen KL, Chiu HY, Lin JH, Ye JD, Cho YH, et al. (2019) Prevalence, clinical features and treatment pattern of patients with concurrent diagnoses of rheumatoid arthritis and psoriatic disease: results of a 14-year retrospective study in a tertiary referral center. Ther Adv Chronic Dis 10: 2040622319847900. https://doi.org/10.1177/2040622319847900

19. Aicher A, Heeschen C, Mohaupt M, Cooke JP, Zeiher AM, et al. (2003) Nicotine strongly activates dendritic cell–mediated adaptive immunity: potential role for progression of atherosclerotic lesions. Circulation 107: 604-611. https://doi.org/10.1161/01.cir.0000047279.42427.6d

20. Arican O, Aral M, Sasmaz S, Ciragil P (2005) Serum levels of TNF-α, IFN-γ, IL-6, IL-8, IL-12, IL-17, and IL-18 in patients with active psoriasis and correlation with disease severity. Mediators Inflamm 2005: 273-279. https://doi.org/10.1155/MI.2005.273

21. Kotrulja L, Tadinac M, Joki-Begi NA, Gregurek R (2010) A multivariate analysis of clinical severity, psychological distress and psychopathological traits in psoriatic patients. Acta Derm Venereol 90: 251-256. https://doi.org/10.2340/00015555-0838