Smoking, but not alcohol, is associated with risk of psoriasis in a Taiwanese population-based cohort study

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Background: Alcohol consumption and smoking have long been suspected of increasing the risk of developing psoriasis. Most evidence to date has derived from cross-sectional or case-control studies.

Objective: We sought to investigate the effects of alcohol and smoking on incident psoriasis.

Methods: Alcohol consumption, smoking status, and other covariates were collected from four rounds (2001, 2005, 2009, and 2013) of the Taiwan National Health Interview Survey. Incident psoriasis was identified from the National Health Insurance database. Cox regression model was used for the analysis.

Results: Of 60,136 subjects, 242 (0.40%) developed psoriasis. After controlling for demographics and comorbidities, alcohol consumption was not significantly associated with psoriasis risk. Conversely, psoriasis risk was higher for current smokers than never smokers (adjusted hazard ratio 1.47 [95% confidence interval 1.04-2.07]). The risks were higher among subjects who smoked >25 cigarettes per day and for >20 pack-years. In subgroup analysis, current smoking was significantly associated with risk of psoriasis without psoriatic arthritis but not psoriatic arthritis alone.

Limitations: Alcohol consumption was not assessed based on the number of drinks consumed.

Conclusion: Current smoking increased the risk of psoriasis, particularly augmented for individuals who smoked >25 cigarettes per day and for >20 pack-years, while alcohol consumption was not significantly associated with psoriasis development. (J Am Acad Dermatol 2019;80:727-34.)

Key words: alcohol; cohort study; psoriasis; smoking.

P soriasis is a common skin disease with multiple etiologies comprising genetic and nongenetic risk factors, including trauma, ultraviolet light exposure, medications, diet, obesity, infection, and mental stress.[1,2] The prevalence of psoriasis varies globally, probably because of differences in genetic and environmental factors.[3-5] The most strongly associated susceptibility gene of psoriasis is the human leukocyte antigen−Cw6 locus. The prevalence of human leukocyte antigen−Cw6 differs widely and is much lower in patients with

psoriasis who are of Chinese descent compared with white patients with psoriasis.[6] It is a longstanding suspicion that alcohol consumption and smoking are both associated with an increased risk of psoriasis onset and disease worsening.[7] Understanding the effects of alcohol and smoking on psoriasis development would provide important information regarding its optimal management. However, most evidence regarding the effects of alcohol and smoking on psoriasis development derives from cross-sectional or case-control studies.[7-11] A single cohort

study from the United States examined the association between risk of incident psoriasis and alcohol consumption.[12] This cohort consisted entirely of American female nurses, which limits the generalizability of the findings. Other cohort studies examining the effects of smoking on incident psoriasis have been conducted in Western populations (the Nurses' Health Study [NHS], NHS II, and Health Professionals' Follow-Up Study).[13-15] The effects of alcohol and smoking in Asian and Western populations might differ because of the genetic differences in the metabolism of these factors.[16] We conducted a nationwide population-based cohort study to investigate the impact of alcohol consumption and smoking on incident psoriasis in the Taiwanese population.

METHODS
Data sources
A cohort was assembled from 4 rounds (2001, 2005, 2009, and 2013) of the National Health Interview Survey (NHIS), a nationwide survey in Taiwan. The NHIS is a cross-sectional survey jointly initiated by the National Health Research Institute and the Health Promotion Administration. The survey, conducted by an experienced survey team once every 4 years, used a multistage stratified systematic sampling scheme to obtain a nationally representative sample of the Taiwanese population. In-person interviews collected information on demographics, socioeconomic status, height, weight, health status, health behavior, and medical care use in survey participants. Details of the design and sampling scheme have been reported previously.[17]

The National Health Insurance (NHI) is a mandatory national health insurance program with an enrollment rate of 99%. The NHI Research Database (NHIRD) provides comprehensive information about the insured subjects, including details of demographics (date of birth, sex, and residential location) and claims data (outpatient and inpatient care, medical diagnoses, prescriptions, and operations). The NHIRD has been widely used in epidemiologic studies of psoriasis.[18-22] Permission was sought from the NHIS participants to link to their NHIRD data for research purposes. Only participants who consented to link their NHIRD data were eligible for the present study. In our study, there were 84,249 subjects selected from the 2001, 2005,

2009, and 2013 NHIS databases, and 86.9% (n = 73,218) consented to link their NHIS data to NHIRD records. This method for linking the NHIS data to the NHIRD has been used in several published studies.[23-25] The dataset linkage process was performed at the Data Center of the Ministry of Health and Welfare. All identification numbers of subjects were encrypted to ensure that no person or medical care facility was identifiable.

Study cohorts
Subjects ≥12 years of age were selected from the 4 rounds of the NHIS database. We excluded subjects with a previous psoriasis diagnosis (n = 110), unknown alcohol consumption (n = 4639), unknown smoking status (n = 46), or unknown covariates (n = 8287). Subjects were followed from the time of the NHIS interview until a diagnosis of psoriasis, death, or to December 31, 2015. Deaths were confirmed by linking to the death certificate database of Taiwan. This project was approved by the Institutional Review Board of Taipei Veterans General Hospital (2017-08-005CC).

Measurement of alcohol consumption, smoking status, and other covariates
Detailed information on alcohol consumption, smoking status, body mass index (BMI), marital status, education level, and monthly household income was obtained from the NHIS database. Based on the level of alcohol consumption, subjects were classified into never, social (less than once per week), regular (once per week or more but not to the extent of being intoxicated), or heavy drinkers (once per week or more and to the extent of being intoxicated). As for smoking status, ever smokers had smoked >100 cigarettes and never smokers had smoked <100 cigarettes. Among the ever smokers, those who had smoked the month before the interview were classified as current smokers; otherwise, subjects were classified as former smokers. For current smokers, data were available regarding the number of cigarettes smoked daily in the past 30 days, years of smoking, and age when smoking began. The BMI was categorized as underweight (<18.5 kg/m^2), normal weight (18.5-22.9 kg/m^2), overweight (23.0-24.9 kg/m^2), obese class I (25.0-29.9 kg/m^2), and obese class II

CAPSULE SUMMARY

- Although alcohol and smoking have been suspected of increasing the risk of psoriasis, the evidence remains inconclusive.

- Our study shows that current smoking increased the risk of developing psoriasis, while alcohol consumption was not associated with incident psoriasis.

- Restricting smoking is needed to reduce the burden of psoriasis.

($\geq$30 kg/m^2). Marital status was categorized as unmarried, married/cohabiting, and other (eg, widowed, divorced, separated, or single parent). Educational level was classified into elementary or lower, high school, and university or higher education. Monthly household income was classified into <30,000, 30,000 to <70,000, and $\geq$70,000 New Taiwan dollars.

Comorbidities

The presence of comorbidities was identified from the NHIRD based on the *International Classification of Diseases, 9th revision, Clinical Modification* (ICD-9-CM) codes and included cancer (ICD-9-CM codes 140-208), diabetes (ICD-9-CM code 250), hyperlipidemia (ICD-9-CM code 272), hypertension (ICD-9-CM codes 401-405), coronary artery disease (ICD-9-CM codes 410-414), cerebrovascular disease (ICD-9-CM codes 430-438), chronic obstructive pulmonary disease (COPD; ICD-9-CM codes 491, 492, and 496), asthma (ICD-9-CM code 493), chronic liver disease (ICD-9-CM code 571), chronic kidney disease (ICD-9-CM codes 580-587), and connective tissue disease (ICD-9-CM codes 710, 714). A person was considered to have comorbidity only if the condition occurred in an inpatient setting or required $\geq$3 outpatient visits.

Outcomes

The primary outcome for the analysis was incident psoriasis, defined according to ICD-9-CM codes 696.0 and 696.1 from the NHIRD. Subjects were considered to have psoriasis only if the diagnosis was made by dermatologists or rheumatologists and the condition occurred in an inpatient setting or required $\geq$3 outpatient visits. The psoriasis identification algorithm has previously been validated by Lee et al.[20] To identify incident psoriasis, we excluded subjects with a previous psoriasis diagnosis, defined as the presence of psoriasis identified before NHIS enrollment.

Subgroup analysis

Etiologies of psoriasis and psoriatic arthritis (PsA) may differ, and therefore we conducted a subgroup analysis based on the presence or absence of PsA. Patients with PsA were defined as those with $\geq$3 outpatient claims or $\geq$1 inpatient claim of ICD-9-CM code 696.0 during the same period; otherwise, patients were considered to have psoriasis without PsA.

Statistical analysis

We estimated the incidence rate of psoriasis and person-time of follow-up in subjects based on different levels of alcohol consumption and smoking status. The Cochran—Armitage trend test was used to determine the trend in psoriasis incidence and the subjects' characteristics according to the levels of alcohol consumption and smoking status, respectively.

The univariate Cox regression model assessed the crude association of alcohol consumption, smoking status, and other covariates with incident psoriasis by computing the hazard ratios (HRs) and the corresponding 95% confidence intervals (CIs). A multivariable Cox regression model estimated the independent association between different levels of alcohol consumption, smoking status, and incident psoriasis after adjusting for potential confounders. Other smoking variables that were analyzed included smoking intensity, duration, and pack-years. Sensitivity analysis was performed to assess the robustness of results by using different classification of smoking status according to pack-years of smoking. Adjusted HRs (aHRs) with 95% CIs were calculated to indicate the strength and direction of these associations. Two-sided *P* values < .05 were considered statistically significant. Data management and analyses were performed with SAS software (version 9.4; SAS Institute, Cary, NC).

RESULTS
Characteristics of the study subjects

A total of 60,136 participants of NHIS were included in this cohort study. The overall mean (standard deviation [SD]) age was 40.64 (17.81) years, 50.2% were males, and the mean (SD) follow-up time was 8.92 (4.45) years (Table I). Most study subjects (61.4%) were classified as never drinkers, while only 1.6% were heavy drinkers. Compared with never drinkers, heavy and regular drinkers were more likely to be male, overweight or obese, less educated, current smokers, and to have low household income and comorbidities, including diabetes, hyperlipidemia, hypertension, COPD, and chronic liver disease. Among the 60,136

Table I. Baseline characteristics of the study population according to the level of alcohol consumption

Characteristics	Alcohol consumption				
	Never, n = 36,930	Social, n = 15,640	Regular, n = 6592	Heavy, n = 974	P_{trend}
Age in years, mean (SD)	40.81 (19.53)	38.26 (14.64)	45.15 (13.91)	41.90 (11.91)	<.0001
Gender, n (%)					
Female	22,834 (61.83)	5908 (37.77)	1086 (16.47)	110 (11.29)	<.0001
Male	14,096 (38.17)	9732 (62.23)	5506 (83.53)	864 (88.71)	
BMI (kg/m^2), n (%)					
Underweight (<18.5)	4417 (11.96)	1017 (6.50)	255 (3.87)	30 (3.08)	<.0001
Normal weight (18.5-22.9)	16,286 (44.10)	6531 (41.76)	2295 (34.81)	357 (36.65)	
Overweight (23.0-24.9)	6573 (17.80)	3235 (20.68)	1460 (22.15)	207 (21.25)	
Obese class I (25.0-29.9)	7596 (20.57)	3891 (24.88)	2133 (32.36)	308 (31.62)	
Obese class II (≥30)	2058 (5.57)	966 (6.18)	449 (6.81)	72 (7.39)	
Smoking status, n (%)					
Never	32,009 (86.67)	9992 (63.89)	2131 (32.33)	203 (20.84)	<.0001
Current	3917 (10.61)	4521 (28.91)	3810 (57.80)	737 (75.67)	
Former	1004 (2.72)	1127 (7.21)	651 (9.88)	34 (3.49)	
Comorbidity, n (%)					
Cancer	2497 (6.76)	647 (4.14)	441 (6.69)	83 (8.52)	.0003
Diabetes	5542 (15.01)	1464 (9.36)	1085 (16.46)	203 (20.84)	.0332
Hyperlipidemia	8387 (22.71)	2832 (18.11)	1722 (26.12)	302 (31.01)	.0125
Hypertension	10,436 (28.26)	3024 (19.34)	2238 (33.95)	394 (40.45)	.0220
Coronary artery disease	4797 (12.99)	1226 (7.84)	830 (12.59)	104 (10.68)	<.0001
Cerebrovascular disease	3366 (9.11)	670 (4.28)	543 (8.24)	87 (8.93)	<.0001
COPD	3436 (9.30)	904 (5.78)	659 (10.00)	104 (10.68)	.0019
Asthma	3594 (9.73)	1094 (6.99)	471 (7.15)	84 (8.62)	<.0001
Chronic liver disease	5604 (15.17)	2071 (13.24)	1332 (20.21)	279 (28.64)	<.0001
Chronic kidney disease	2113 (5.72)	509 (3.25)	300 (4.55)	52 (5.34)	<.0001
Connective tissue disease	1422 (3.85)	369 (2.36)	168 (2.55)	12 (1.23)	<.0001

BMI, Body mass index; *COPD,* chronic obstructive pulmonary disease; *SD,* standard deviation.

subjects, 21.6% were current smokers, 4.7% were former smokers, and 73.7% had never smoked (Table II).

Incidence of psoriasis according to alcohol consumption and smoking status

During the 536,711 person-years of follow-up, 242 subjects developed psoriasis, including 140 never drinkers, 61 social drinkers, 37 regular drinkers, and 4 heavy drinkers. Incidence rates per 100,000 person-years in never, social, regular, and heavy drinkers were 39.96, 51.42, 65.38, and 35.94, respectively. After controlling for demographics and comorbidities, no significant association was observed between alcohol consumption and psoriasis risk (Table III).

Incident psoriasis occurred in 145 never smokers, 80 current smokers, and 17 former smokers. Incidence rates per 100,000 person-years in never, current, and former smokers were 36.57, 66.88, and 82.48, respectively. In univariate analyses, current and former smokers had a significantly increased risk of psoriasis. After adjusting for potential

confounders, current smokers still had a higher risk of incident psoriasis compared with never smokers (aHR 1.47 [95% CI 1.04-2.07]). Other independent risk factors for incident psoriasis were age (per 10-year increase), male sex, obesity, cancer, chronic liver disease, and connective tissue disease.

Compared with never smokers, the aHRs (95% CIs) for incident psoriasis were 1.70 (1.06-2.72) for subjects who smoked >25 cigarettes per day and 2.38 (1.54-3.67) for those with >20 pack-years of smoking (Table IV). However, no statistically significant trend in psoriasis risk was observed across categories of cigarettes per day, years of smoking, and pack-years of smoking. In the sensitivity analysis that reclassified subjects according to pack-years of smoking, the association between alcohol consumption and psoriasis risk remained insignificant (data not shown).

In subgroup analysis, current smoking significantly increased the risk of psoriasis without PsA (aHR 1.49 [95% CI 1.07-2.10]), while we did not observe a significant association between smoking and PsA after adjustment for potential confounders.

Table II. Baseline characteristics of the study population according to smoking status

Characteristics	Never, n = 44,335	Current, n = 12,985	Former, n = 2816	P_{trend}
		Smoking status		
Age in years, mean (SD)	39.62 (18.48)	41.41 (14.37)	53.06 (16.53)	<.0001
Gender, n (%)				
Female	28,299 (63.83)	1414 (10.89)	225 (7.99)	<.0001
Male	16,036 (36.17)	11,571 (89.11)	2591 (92.01)	
BMI (kg/m^2), n (%)				
Underweight (<18.5)	4956 (11.18)	669 (5.15)	94 (3.34)	<.0001
Normal weight (18.5-22.9)	19,628 (44.27)	4980 (38.35)	861 (30.58)	
Overweight (23.0-24.9)	8085 (18.24)	2716 (20.92)	674 (23.93)	
Obese class I (25.0-29.9)	9255 (20.88)	3709 (28.56)	964 (34.23)	
Obese class II (≥30)	2411 (5.44)	911 (7.02)	223 (7.92)	
Alcohol consumption, n (%)				
Never	32,009 (72.20)	3917 (30.17)	1004 (35.65)	<.0001
Social	9992 (22.54)	4521 (34.82)	1127 (40.02)	
Regular	2131 (4.81)	3810 (29.34)	651 (23.12)	
Heavy	203 (0.46)	737 (5.68)	34 (1.21)	
Comorbidity, n (%)				
Cancer	2554 (5.76)	770 (5.93)	344 (12.22)	<.0001
Diabetes	5788 (13.06)	1868 (14.39)	638 (22.66)	<.0001
Hyperlipidemia	9452 (21.32)	2842 (21.89)	949 (33.70)	<.0001
Hypertension	11,270 (25.42)	3499 (26.95)	1323 (46.98)	<.0001
Coronary artery disease	4924 (11.11)	1385 (10.67)	648 (23.01)	<.0001
Cerebrovascular disease	3230 (7.29)	984 (7.58)	452 (16.05)	<.0001
COPD	3298 (7.44)	1266 (9.75)	539 (19.14)	<.0001
Asthma	3936 (8.88)	953 (7.34)	354 (12.57)	.3236
Chronic liver disease	6355 (14.33)	2247 (17.30)	684 (24.29)	<.0001
Chronic kidney disease	2100 (4.74)	586 (4.51)	288 (10.23)	<.0001
Connective tissue disease	1617 (3.65)	260 (2.00)	94 (3.34)	<.0001

BMI, Body mass index; *COPD*, chronic obstructive pulmonary disease; *SD*, standard deviation.

DISCUSSION

In this study, no significant association was observed between alcohol consumption and incidence of psoriasis. Evidence of a positive correlation between alcohol consumption and the risk of psoriasis has been reported; however, most data are derived from cross-sectional or case-control studies, which are subject to many sources of bias.[26] In the Qureshi et al[12] study involving 82,869 US female nurses with a 14-year follow-up, the multivariate relative risk of psoriasis was 1.72 (95% CI 1.15-2.57) for women having ≥2.3 drinks per week compared with never drinkers. The risk of psoriasis varied by the type of alcoholic beverage with nonlight beer being the only alcoholic beverage that increased psoriasis risk. However, the validity of the study results was diminished by self-reported psoriasis diagnosis and uncontrolled confounding factors, especially comorbidities. Therefore, whether alcohol represents an indisputable risk factor for psoriasis remains inconclusive.

Our study suggests that alcohol consumption is not an independent risk factor for psoriasis in the Taiwanese population. Several studies have found the presence of ethnic differences in alcohol metabolism. Variant alleles of the aldehyde dehydrogenase-2 (*ALDH2*2*) and alcohol dehydrogenase-1B genes (*ADH1B*2*) are harbored by 73% and 50% of the Taiwanese population, respectively, but are rarely found in whites.[27,28] Individuals with heterozygote and homozygote of *ALDH2*2* have respectively 5- and 18-fold higher blood acetaldehyde levels after drinking.[16,28] Some studies have supported the role of alcohol metabolites, acetaldehyde, and acetone in the pathogenesis of psoriasis.[29] The ethnic disparities in alcohol metabolism may influence the effects of alcohol on psoriasis development. However, further studies are needed to elucidate the mechanisms underlying the ethnic differences in the association between alcohol and psoriasis.

Smoking is more consistently associated with psoriasis compared with alcohol.[15,30,31] Herein, we found that current smokers had a significantly higher risk of developing psoriasis. In a pooled analysis of the NHS, NHS II, and the Health Professionals' Follow-Up Study, current smokers were nearly twice

Table III. Univariate and multivariate associations among alcohol consumption, smoking status, and incident psoriasis

	Univariable	Multivariable
Characteristics	**HR (95% CI)**	**aHR* (95 % CI)**
Alcohol consumption		
Never	1	1
Social	1.42 (1.05-1.92)[†]	1.20 (0.87-1.66)
Regular	1.71 (1.19-2.46)[‡]	1.06 (0.72-1.58)
Heavy	0.88 (0.33-2.38)	0.51 (0.19-1.41)
Smoking status		
Never	1	1
Current	1.83 (1.40-2.41)[§]	1.47 (1.04-2.07)[†]
Former	2.48 (1.50-4.11)[§]	1.16 (0.68-1.99)
Age (per 10-year increase)	1.36 (1.27-1.46)[§]	1.43 (1.26-1.61)[§]
Gender		
Female	1	1
Male	2.04 (1.56-2.67)[§]	1.72 (1.24-2.38)[‡]
BMI, kg/m^2		
Normal weight (18.5-22.9)	1	1
Underweight (<18.5)	0.51 (0.25-1.01)	0.70 (0.35-1.40)
Overweight (23.0-24.9)	1.13 (0.77-1.66)	0.89 (0.60-1.31)
Obese class I (25.0-29.9)	2.12 (1.56-2.87)[§]	1.57 (1.14-2.17)[‡]
Obese class II (≥30)	3.22 (2.10-4.93)[§]	2.57 (1.65-4.02)[§]
Comorbidity		
Cancer	2.51 (1.72-3.66)[§]	1.52 (1.02-2.25)[†]
Diabetes	2.00 (1.49-2.68)[§]	1.03 (0.72-1.46)
Hyperlipidemia	1.66 (1.27-2.17)[§]	0.92 (0.67-1.28)
Hypertension	2.26 (1.76-2.92)[§]	1.17 (0.84-1.64)
Coronary artery disease	1.40 (0.99-1.99)	0.58 (0.39-0.87)[‡]
Cerebrovascular disease	1.47 (0.97-2.22)	0.65 (0.42-1.03)
COPD	2.17 (1.54-3.05)[§]	1.08 (0.72-1.60)
Asthma	1.51 (1.03-2.23)[†]	1.04 (0.69-1.59)
Chronic liver disease	2.13 (1.61-2.81)[§]	1.54 (1.14-2.08)[‡]
Chronic kidney disease	2.38 (1.59-3.58)[§]	1.22 (0.79-1.90)
Connective tissue disease	2.87 (1.85-4.44)[§]	2.41 (1.53-3.80)[§]

aHR, Adjusted hazard ratio; *BMI*, body mass index; *CI*, confidence interval; *COPD*, chronic obstructive pulmonary disease; *HR*, hazard ratio.
*Cox model adjusted for age, sex, BMI level, marital status, educational level, monthly household income, alcohol consumption, smoking status, and comorbidities.
[†]$P < .05$.
[‡]$P < .01$.
[§]$P < .001$.

Table IV. Multivariate associations between psoriasis and smoking by cigarettes per day, years of smoking, and pack-years

	aHR*(95% CI)
Cigarettes per day	
Never-smoker	1
≤5	1.42 (0.65-3.07)
>5 and ≤15	1.41 (0.91-2.19)
>15 and ≤25	1.35 (0.87-2.09)
>25	1.70 (1.06-2.72)[†]
P_{trend}	.284
Years of smoking	
Never-smoker	1
≤10	1.04 (0.59-1.83)
>10 and ≤20	1.38 (0.83-2.28)
>20	1.42 (0.99-2.06)
P_{trend}	.173
Pack-years of smoking	
Never-smoker	1
≤10	1.32 (0.86-2.04)
>10 and ≤20	1.1 (0.68-1.78)
>20	2.38 (1.54-3.67)[‡]
P_{trend}	.346

aHR, Adjusted hazard ratio; *CI*, confidence interval.
*Cox model adjusted for age, sex, BMI level, marital status, educational level, monthly household income, alcohol consumption, and comorbidities.
[†]$P < .05$.
[‡]$P < .001$.

as likely to develop psoriasis compared with non-smokers (adjusted OR 1.94 [95% CI 1.64-2.28]).[8] In a nationwide cohort study from Korea, current smokers had a higher risk of developing psoriasis (adjusted incidence ratio 1.14 [95% CI 1.13-1.15]) compared with nonsmokers.[32] These findings indicate that smoking is an independent risk factor for psoriasis both in Asian and Western populations. Previous studies have identified a graded elevation of psoriasis incidence correlating with increased smoking intensity or duration.[13] However, no dose-response association of smoking intensity, duration, or cumulative exposure with psoriasis risk was observed in our study. Previous studies have suggested that smoking increases PsA risk in Western populations.[33,34] Nevertheless, no significant association between smoking and risk of PsA was found in our study, potentially because of the small number of patients with PsA. Further studies with larger sample sizes are necessary to confirm our findings.

The strengths of this study include a cohort study design with large sample size, reliable psoriasis diagnosis made by dermatologists and

rheumatologists, and adjustment for potential confounding factors. However, several limitations remain. First, the degree of alcohol consumption was not assessed according to the number of drinks consumed, making it difficult to compare our results with those from other studies. Second, the information regarding alcohol abstinence is lacking in the NHIS database. It is not possible to distinguish subjects who quit drinking from never drinkers, therefore precluding subgroup analysis. Besides, like previous studies, self-reported measures of alcohol consumption may be biased by social desirability. Third, we measured alcohol consumption and smoking habits once and assumed that the status remained unchanged during the observation period. Fourth, the NHIS and NHIRD lack information on genetic and several environmental risk factors for psoriasis, including diet, trauma, ultraviolet light exposure, and negative life events, which were possible uncontrolled confounders. Fifth, despite the high consent rate for this record linkage study, the possibility of selection bias may not be fully eliminated. The potential source of selection bias may affect the representativeness of the data. In addition, the missing data may cause information bias. However, the observed risk was evaluated using internal comparisons; therefore, the biologic effects of alcohol consumption and smoking on psoriasis should be similar. Finally, the external validity of our findings may be a concern, because almost all of our subjects were Taiwanese.

In conclusion, no significant association was observed between alcohol consumption and psoriasis risk in the Taiwanese population. We found that the risk of psoriasis was 47% higher for current smokers than never smokers. Compared with never smokers, subjects who smoked >25 cigarettes per day and >20 pack-years had a 1.70- and 2.38-fold increased risk for psoriasis, respectively. Our study has important public health implications, adding psoriasis to the long list of diseases that may be prevented by smoking cessation.

This study was approved by the Institutional Review Board of the Taipei Veterans General Hospital (VGHIRB 2017-08-005CC).

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