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Alcohol intake and risk of incident psoriasis in US women: A prospective study

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Abstract

Objective—To evaluate the independent association between alcohol consumption and risk of developing psoriasis and to determine if this risk is associated with different types of alcoholic beverages.

Design—A prospective study of female nurses who were followed up from 1991 to 2005.

Setting—Nurses' Health Study II, a cohort of 116 671 US women aged 27 to 44 years in 1991.

Participants—The study included 82 869 women who reported amount and type of alcohol intake on biennial questionnaires. We excluded participants with a history of psoriasis prior to 1991.

Main Outcome Measure—The main outcome measure consisted of a self-report of incident physician-diagnosed psoriasis. For a sensitivity analysis, we had a subset of confirmed psoriasis cases.

Results—There were 1,150 cases of incident psoriasis. Compared with women who did not drink alcohol, the multivariate relative risk (RR) of psoriasis was 1.73 (95% confidence interval [CI], 1.16–2.58) for alcohol consumption ≥ 2.3 drinks/week. When examined by type of alcoholic beverage, the association with alcohol intake was due to non-light beer (multivariate RR for ≥5 drinks/wk 1.83; 95% CI, 1.16–2.86); light beer, red wine, white wine, and liquor were not

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significantly associated with psoriasis risk. The association with non-light beer intake became stronger in a subset of confirmed psoriasis cases (multivariate RR for 2.29; 95% CI, 1.36 – 3.85).

Conclusions—Non-light beer intake is associated with an increased risk of developing psoriasis among women. Other alcoholic beverages did not increase the risk of psoriasis in this study.

INTRODUCTION

Psoriasis is a common immune-mediated skin disease [1, 2]. The association between alcohol consumption and increased risk of psoriasis onset and psoriasis worsening has long been suspected. For example, individuals with psoriasis drink more alcohol than individuals without psoriasis [3], and alcohol intake may exacerbate psoriasis severity [4]. Case-control studies reported a significant association between alcohol consumption and psoriasis, but these studies retrospectively ascertained alcohol consumption. Furthermore, these associations showed a marked gender difference in men [3, 5], and did not show a significant association between alcohol and psoriasis in women [6]. One case-control study of alcohol intake and newly diagnosed psoriasis and showed a dose-response, but the findings were not significant after adjusting for cofactors [7].

Alcohol may induce psoriasis via multiple mechanisms, including immunological changes such as keratinocyte proliferation [8] and up-regulation of pro-inflammatory cytokines [9]. Other potential mechanisms include an increased risk of infection and mechanical trauma, which are well known to trigger psoriasis [10].

Risk of psoriasis may vary depending on type of alcoholic beverage (ie, beer, wine, and spirits), as does the risk of other diseases, but no data are available on psoriasis. One prospective study confirmed, for example, that beer confers a larger risk of gout than spirits and wine [11]. Risk for psoriasis may vary by type of alcoholic beverage given previous evidence that different types of alcoholic beverages have conferred deleterious effects in other inflammatory diseases. If certain types of alcoholic beverages have different effects on risk of psoriasis, then this fact would have practical implications for psoriasis prevention and management.

We prospectively evaluated the association between total alcohol consumption and risk of incident psoriasis in a cohort of women in the United States with no history of psoriasis. In this study, we also explored the association between type of alcoholic beverage and risk for incident psoriasis.

METHODS

Study population

The Nurses' Health Study II (NHS II) is an ongoing longitudinal study of 116,430 female registered nurses from 15 states in the United States who were between the ages of 25 and 42 when they completed and returned a baseline questionnaire in 1989 [12, 13]. The cohort is followed with biennial questionnaires and the follow-up rate exceeds 90%.

Assessment of Psoriasis

In 2005, NHS II participants were asked if they had ever received a physician diagnosis of psoriasis and if so, the date of diagnosis. Of the 82,869 participants who responded to the psoriasis question in 2005, a total of 2,430 women reported being diagnosed with psoriasis; 1,280 of these were prevalent cases at baseline in 1991 and 1,150 incident cases occurred between 1991 and 2005. We started follow-up in 1991 because it is the first year for which

we have information regarding alcohol intake. There were 1,069 women with incident psoriasis included in this analysis.

We confirmed a subset of self-reports using the Psoriasis Screening Tool (PST) questionnaire14. The PST is a one page self-administered questionnaire that assigns a diagnosis of psoriasis based on responses to seven questions. The questions on the PST were derived from expert opinion and the National Psoriasis Foundation's survey on psoriasis. Three questions inquire about being diagnosed with psoriasis by some type of medical provider, such as a dermatologist, a primary care doctor, or a nurse practitioner. Four questions inquire about having morphological skin changes of psoriasis, three of which are accompanied by pictures. Pictures were chosen to represent each of three common psoriasis phenotypes, namely plaques, nail changes, and scalp psoriasis. One non-pictorial question inquires about inverse psoriasis. Scoring algorithms were developed to assign a diagnosis of psoriasis depending on a participant's response to the questions. These scoring algorithms were based on multiple a priori hypotheses. A pilot study showed that the PST was able to screen for psoriasis with 99% sensitivity and 94% specificity14 in a population of adults attending an outpatient dermatology clinic of a tertiary care referral center. The PST has yet to be tested in a population with an expected low prevalence of psoriasis. However using Bayes' theorem, the calculated positive predictive value of the PST is 25%. In other words, a positive test result on the PST increases the pre-test probability of having psoriasis from 2% (assumed prevalence of psoriasis in the United States population15, 16) to 25%. We mailed the PST to all NHS II participants who self-reported a physician diagnosis of psoriasis with an overall response rate of 87%.

Assessment of Alcohol Intake

Information on alcohol intake was updated in 1995, 1999, and 2003. The 1991 questionnaire included questions on the average intake of alcoholic beverages (regular beer, light beer, red wine, white wine, and liquor) during the past year. Nurses responded to the following question: "For each food listed, fill in the circle indicating how often on average you have used the amount specified during the past year." Intake of each beverage was ascertained in nine categories (number of drinks): none or less than once per month, 1 to 3 per month, 1 per week, 2 to 4 per week, 5 to 6 per week, 1 per day, 2 to 3 per day, 4 to 5 per day, and 6 or more per day. Total amount of alcohol consumed was estimated at 12.8 g for a glass, bottle, or can of beer (12 oz), 11 g for a glass of wine (4 oz), and 14 g for a shot of liquor (1.5 oz). Total alcohol intake was computed as the sum of the intake from beer, wine, and liquor. One drink was defined as 12.8g of alcohol (median amount of alcohol in beer, wine, and liquor). Beverage-specific consumption was also calculated and analyzed separately. The reproducibility and validity of this questionnaire for alcohol intake have been previously documented in the NHS I cohort [17].

Assessment of Covariates

Date of birth and height were reported on the 1989 questionnaire. Participants reported their current weight 13, smoking status [12], dietary intake, and physical activity on the biennial mailed questionnaires. Quintiles of dietary folate equivalents (DFE) were calculated from a validated semi-quantitative food frequency questionnaire and quintiles of metabolic equivalents (METS) were calculated from questions about various type of physical activity. Body mass index was calculated by dividing updated weight in kilograms by the square of height in meters. Previously, the accuracy of self-reported anthropometric measures was validated among 140 NHS I participants. Self-reported and measured weights were highly correlated (0.97) [18].

Statistical analysis

We computed person-time of follow-up for each participant from the return date of the 1991 questionnaire to the date of diagnosis of psoriasis or the end of the study period. We used Cox proportional hazards models to estimate the age-adjusted and multivariate relative risk of incident psoriasis in women who reported alcohol intake compared to those who did not. We categorized alcohol intake into six categories-none, 1 to 4 g per week, 5 to 9 g per week, 10 to14 g per week, 15 to 29 g per week, and 30 g or more per week. We categorized type of alcoholic beverage (regular beer, light beer, red wine, white wine, and liquor) into four categories-none, 1 to 3 per month, 1 per week, 2 to 4 per week, and 5 or greater per week. We categorized body mass index at baseline and at each questionnaire cycle into six categories: <21kg/m^2, 21–22.9, 23–24.9, 25–27.4, 27.5–29.9, 30–34.9 and ≥35. We categorized DFE as low and high based on the median for DFE. Median DFE was available only in 1999 and 2003. The median DEF was 976 mcg in 1999 and 1000 mcg in 2003. We applied the 1999 median DFE to three time periods: 1991–1994, 1995–1998, and 1999–2002. We applied the 2003 median DFE to the 2003–2006 time period. We explored potential interactions by DFE (high and low according to the period specific median) by testing the significance of interaction terms added to our final multivariate models. For all relative risks, we calculated 95% confidence intervals and all p-values were two-sided. Statistical analyses were performed using SAS software, version 9.1 (SAS Institute Inc, Car, NC). The Partners Health Care System Institutional Review Board approved this study.

RESULTS

Over the 14-year follow-up period, 1069 incident cases of psoriasis occurred. There was no material difference in mean age between alcohol drinkers and abstainers (Table 1). Alcohol abstainers had a higher BMI and were less physically active. Proportion of current and past smokers was higher in alcohol drinkers and dietary folate intake was higher in abstainers.

We observed an increased age-adjusted relative risk of psoriasis in women who consumed greater than 2.3 alcoholic beverages per week compared to nondrinkers (Table 2). This risk remained significant after adjusting for age, BMI (both continuous and categorical), smoking, physical activity, and DFE. Although the relative risks did not reach statistical significance in women who consumed less than 2.3 drinks per week, there was a significant trend towards increased risk for psoriasis with greater alcohol intake (p for trend 0.002).

We also found that the risk of psoriasis varied by type of alcoholic beverage. After adjusting for age, women who drank ≥ 5 beers per week had a greater relative risk of psoriasis (Table 3). The excess risk of psoriasis associated with consumption of at least 5 non-light beers per week for all NHS2 participants was 1.53%. We calculated the excess risk, which was 106 per 100K person-years (PY) − 46 per 100K PY = 60 per 100K PY. This gave a relative risk increase of 1.3. This risk remained significant after simultaneously adjusting for age, BMI, smoking, physical activity, and DFE. Women who drank any amount of light beer, white wine, red wine, or liquor were not at increased risk of incident psoriasis. We performed analyses with a common reference group that excluded any women who drank any type of alcohol and found no difference in the effect estimates. We also evaluated the multivariate effect of beer on psoriasis risk in a model that included all types of alcoholic beverages and there was no material change in the association.

We also evaluated the influence of DFE intake on the association between beer and risk of psoriasis. Although the risk for psoriasis was higher in the low DFE group (multivariate RR ≥ 5 drinks/wk 2.11; 95% CI 1.15–3.89), the interaction was not statistically significant (p=0.18).

The PST was sent to 2430 who self-reported a physician diagnosis of psoriasis. Of these, 1150 were incident cases and 1280 were prevalent cases. Of the 1150 incident cases, only 1069 were used for analysis. The response rate to the PST among the incident psoriasis cases was 73%, and the response rate among the prevalent psoriasis cases was 82%. Of the incident cases who responded with a completed PST questionnaire, 86% were actually confirmed to have psoriasis as determined by the PST. Among these confirmed cases, we observed a materially elevated and statistically significant relative risk of psoriasis in women who consumed ≥ 2.3 alcoholic beverages per week and in women who drank ≥ 5 non-light beers per week (Table 4).

DISCUSSION

In this prospective study of US women, we found the risk for psoriasis varied by the amount and type of alcoholic beverage. Although overall women who drank more than 2.3 alcoholic beverages per week were at greater risk for psoriasis, non-light beer was the only alcoholic beverage that conferred an increased risk for psoriasis. The risk for psoriasis was 1.8 times higher among women who consumed ≥ 5 non-light beers per week per day compared to those who abstained from alcohol. When we used a more precise definition for psoriasis, the risk was 2.3 times higher among women who consumed ≥ 5 non-light beers per week. These associations were independent of other potential risk factors for psoriasis such as age, smoking, BMI, physical activity, and DFE. The excess risk of psoriasis associated with consumption of at least 5 non-light beers per week for all NHS2 participants was 1.53%; however this excess risk would likely be more pronounced in the general population where the prevalence of non-light beer consumption is higher than the prevalence of beer consumption in this cohort. For example, this excess risk increases to 15.3% given the prevalence of drinking in the general US population[19].

Non-light beer was the only alcoholic beverage that increased the risk for psoriasis, suggesting that certain non-alcoholic components of beer, which are not found in wine or liquor, may play an important role in new onset psoriasis. One of these components may be the starch-source used in making beer. Beer is one of the few non-distilled alcoholic beverages that use a starch-source for fermentation, which is commonly barley. This differs from wine that uses a fruit-source (grapes) for fermentation. Some types of liquors such as vodka may use a starch-source for fermentation; however these starches are physically separated from the liquor during distillation. Starch sources such as barley contain gluten, which has been shown to be associated with psoriasis. For example, individuals with psoriasis have elevated levels of anti-gliadin antibodies (AGA) and may have a so called ‘latent-gluten sensitivity’ compared to individuals without psoriasis [20]. Several studies have shown that a gluten-free diet may improve psoriasis severity in patients with gluten sensitivity 20–23. One case report describes a patient with Celiac disease and psoriasis whose skin lesions improved shortly after starting a gluten-free diet 24. Light beer also contains gluten, however, this study did not show an association between light beer intake and incident psoriasis. This may be due possibly to the lower amounts of grain used to make light beer compared to non-light beer (personal communication, W. Kerr (wkerr@arg.org)). Although a gluten free diet helps clear psoriasis [22, 24], it remains unknown whether gluten contributes to new onset psoriasis and whether this only occurs in predisposed individuals, such as those with latent gluten sensitivity.

Limitations of our study deserve comment. The retrospective recall of psoriasis onset may have led to misclassification of psoriasis onset. We were also unable to examine whether the risk for psoriasis was different for early-onset psoriasis versus later-onset psoriasis given the age of the NHS II cohort at baseline (25 to 42 years). This well-educated female cohort provides high quality data with little loss to follow-up but does not represent a random

sample of US women. Although the absolute rates of psoriasis and distribution of alcohol intake may not be representative of a random sample of US women, the biological effects of alcohol intake on psoriasis should be similar. Our study was observational; thus, we cannot rule out the possibility that statistical error and unmeasured factors, such as socioeconomic status and stress, might contribute to the observed associations, however we included as many known risk factors (obesity, smoking, dietary intake, and physical activity) that met the definition of possible confounders into our analyses. As in other epidemiological studies on psoriasis [25–29], our diagnosis of psoriasis relied on self-reports of physician-diagnosed psoriasis and we did not require an examination by a dermatologist. Previous validation studies in the Nurses' Health Study for another skin condition, basal cell carcinoma, found self-reports to be greater than 90% accurate [30, 31]. Furthermore, in the subset of confirmed psoriasis cases, we found a stronger association between risk of incident psoriasis and overall alcohol intake as well as non-light beer intake.

In conclusion, our prospective study indicates that non-light beer intake is associated with an increased risk of psoriasis, whereas light beer, wine, and liquor did not increase the risk among women. Specifically, women who drank at least five non-light beers per week were 1.8 times more likely to develop psoriasis compared to women who abstained from alcohol. Lower intake of non-light beer and intake of other types of alcoholic beverages do not appear to influence the risk of developing psoriasis. Women with a high risk of psoriasis may consider avoiding higher intake of non-light beer. We suggest further investigation into the potential mechanisms of non-light beer inducing new-onset psoriasis.

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REFERENCES

1. Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet 2007 Jul 21;370(9583):263–271. [PubMed: 17658397]
2. Lowes MA, Bowcock AM, Krueger JG. Pathogenesis and therapy of psoriasis. Nature 2007 Feb 22;445(7130):866–873. [PubMed: 17314973]
3. Higgins E. Alcohol, smoking and psoriasis. Clinical & Experimental Dermatology 2000;25(2):107–110. [PubMed: 10733631]
4. Poikolainen K, Reunala T, Karvonen J, Lauharanta J, Karkkainen P. Alcohol intake: a risk factor for psoriasis in young and middle aged men? BMJ 1990;300(6727):780–783. [PubMed: 1969757]
5. Naldi L, Peli L, Parazzini F. Association of early-stage psoriasis with smoking and male alcohol consumption: evidence from an Italian case-control study. Archives of Dermatology 1999;135(12): 1479–1484. [PubMed: 10606053]
6. Poikolainen K, Reunala T, Karvonen J. Smoking, alcohol and life events related to psoriasis among women. British Journal of Dermatology 1994;130(4):473–477. [PubMed: 8186112]
7. Naldi L, Parazzini F, Brevi A, et al. Family history, smoking habits, alcohol consumption and risk of psoriasis. British Journal of Dermatology 1992;127(3):212–217. [PubMed: 1390163]

8. Farkas A, Kemeny L, Szell M, Dobozy A, Bata-Csorgo Z. Ethanol and acetone stimulate the proliferation of HaCaT keratinocytes: the possible role of alcohol in exacerbating psoriasis. Archives of Dermatological Research 2003;295(2):56–62. [PubMed: 12720008]
9. Ockenfels HM, Keim-Maas C, Funk R, Nussbaum G, Goos M. Ethanol enhances the IFN-gamma, TGF-alpha and IL-6 secretion in psoriatic co-cultures. Br J Dermatol 1996 Nov;135(5):746–751. [PubMed: 8977675]
10. Higgins EM, du Vivier AW. Alcohol and the skin. Alcohol & Alcoholism 1992;27(6):595–602. [PubMed: 1292432]
11. Choi HK, Atkinson K, Karlson EW, Willett W, Curhan G. Alcohol intake and risk of incident gout in men: a prospective study. Lancet 2004 Apr 17;363(9417):1277–1281. [PubMed: 15094272]
12. Setty AR, Curhan G, Choi HK. Smoking and the risk of psoriasis in women: Nurses' Health Study II. American Journal of Medicine 2007;120(11):953–959. [PubMed: 17976422]
13. Setty AR, Curhan G, Choi HK. Obesity, waist circumference, weight change, and the risk of psoriasis in women: Nurses' Health Study II. [see comment]. Archives of Internal Medicine 2007;167(15):1670–1675. [PubMed: 17698691]
14. Dominguez PL, Assarpour A, Kuo H, Holt EW, Tyler S, Qureshi AA. Development and pilot-testing of a psoriasis screening tool. Br J Dermatol 2009 Oct;161(4):778–784. [PubMed: 19566664]
15. Koo J. Population-based epidemiologic study of psoriasis with emphasis on quality of life assessment. Dermatol Clin 1996 Jul;14(3):485–496. [PubMed: 8818558]
16. Stern RS, Nijsten T, Feldman SR, Margolis DJ, Rolstad T. Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment dissatisfaction. J Investig Dermatol Symp Proc 2004 Mar;9(2):136–139.
17. Giovannucci E, Colditz G, Stampfer MJ, et al. The assessment of alcohol consumption by a simple self-administered questionnaire. American Journal of Epidemiology 1991;133(8):810–817. [PubMed: 2021148]
18. Rimm EB, Stampfer MJ, Colditz GA, Chute CG, Litin LB, Willett WC. Validity of self-reported waist and hip circumferences in men and women. Epidemiology 1990;1(6):466–473. [PubMed: 2090285]
19. Saad, L. Drinking habits steady amid recession. [Accessed May 26, 2010]. http://www.gallup.com/poll/121277/drinking-habits-steady-amid-recession.aspx.
20. Michaelsson G, Gerden B, Ottosson M, et al. Patients with psoriasis often have increased serum levels of IgA antibodies to gliadin. British Journal of Dermatology 1993;129(6):667–673. [PubMed: 8286249]
21. Michaelsson G, Kraaz W, Gerden B, et al. Increased lymphocyte infiltration in duodenal mucosa from patients with psoriasis and serum IgA antibodies to gliadin. British Journal of Dermatology 1995;133(6):896–904. [PubMed: 8547041]
22. Michaelsson G, Gerden B, Hagforsen E, et al. Psoriasis patients with antibodies to gliadin can be improved by a gluten-free diet. [see comment]. British Journal of Dermatology 2000;142(1):44–51. [PubMed: 10651693]
23. Michaelsson G, Gerden B. How common is gluten intolerance among patients with psoriasis? Acta Dermato-Venereologica 1991;71(1):90. [PubMed: 1676232]
24. Addolorato G, Parente A, de Lorenzi G, et al. Rapid regression of psoriasis in a coeliac patient after gluten-free diet. A case report and review of the literature. Digestion 2003;68(1):9–12. [PubMed: 12949434]
25. Gelfand JM, Stern RS, Nijsten T, et al. The prevalence of psoriasis in African Americans: results from a population-based study. Journal of the American Academy of Dermatology 2005;52(1):23–26. [PubMed: 15627076]
26. Olsen AO, Grjibovski A, Magnus P, Tambs K, Harris JR. Psoriasis in Norway as observed in a population-based Norwegian twin panel. British Journal of Dermatology 2005;153(2):346–351. [PubMed: 16086747]
27. Stern RS, Nijsten T, Feldman SR, Margolis DJ, Rolstad T. Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment

dissatisfaction. Journal of Investigative Dermatology. Symposium Proceedings 2004;9(2):136–139.

28. Ferrandiz C, Bordas X, Garcia-Patos V, Puig S, Pujol R, Smandia A. Prevalence of psoriasis in Spain (Epiderma Project: phase I). [see comment]. Journal of the European Academy of Dermatology & Venereology 2001;15(1):20–23. [PubMed: 11451315]
29. Saraceno R, Mannheimer R, Chimenti S. Regional distribution of psoriasis in Italy. Journal of the European Academy of Dermatology & Venereology 2008;22(3):324–329. [PubMed: 18269600]
30. Colditz GA, Martin P, Stampfer MJ, et al. Validation of questionnaire information on risk factors and disease outcomes in a prospective cohort study of women. American Journal of Epidemiology 1986;123(5):894–900. [PubMed: 3962971]
31. Hunter DJ, Colditz GA, Stampfer MJ, Rosner B, Willett WC, Speizer FE. Diet and risk of basal cell carcinoma of the skin in a prospective cohort of women. Annals of Epidemiology 1992;2(3):231–239. [PubMed: 1342273]
32. Dominguez PL, Assarpour A, Kuo H, Holt EW, Tyler S, Qureshi AA. Development and pilot-testing of a psoriasis screening tool. Br J Dermatol. 2009 Apr 22;

Table 1

Baseline characteristics according to alcohol consumption status

	Alcohol Consumption		Alcohol Intake† (drinks/wk)				
	No	Yes	0.1 – 0.3	0.4 – 0.7	0.8 – 1.1	1.2 – 2.3	≥ 2.3
Characteristics	**n = 35 058**	**n = 47 614**	**n = 32 030**	**n = 8253**	**n = 4233**	**n = 2221**	**n = 877**
Age* (years, mean)	36.3	36.1	36	36	36.6	37	37.6
Body mass index (kg/m^2, mean)	24.8	23.4	23.7	22.8	22.7	22.8	23.1
Smoking (never, %)	75	59	64	55	49	42	32
Smoking (current, %)	8	14	12	14	19	22	37
Smoking (past, %)	16	27	24	31	32	35	31
Dietary folate equivalent (mcg, mean)	913	916	921	932	909	883	794
Physical activity (metabolic equivalents per week)	18.3	22.4	21.5	24.1	25.1	24.6	22.8

*in 1991

†breakdown of the 47 614 who reported yes to alcohol consumption

Table 2

Age-adjusted and multivariate relative risks for incident psoriasis by amount of alcohol intake among women

Alcohol intake (drinks/wk)	N*	RR (95% CI) Age-adjusted	RR (95% CI) Multivariate †	RR (95% CI) Multivariate ‡
None (referent)	442	1.00	1.00	1.00
0.1 – 0.3	371	0.93 (0.80 – 1.07)	0.96 (0.83 – 1.10)	0.97 (0.82 – 1.12)
0.4 – 0.7	109	0.93 (0.75 – 1.15)	1.00 (0.80 – 1.23)	1.02 (0.82 – 1.26)
0.8 – 1.1	76	1.11 (0.87 – 1.41)	1.19 (0.93 – 1.53)	1.21 (0.94 – 1.55)
1.2 – 2.3	43	1.15 (0.84 – 1.57)	1.20 (0.87 – 1.65)	1.18 (0.85 – 1.64)
≥ 2.3	28	1.89 (1.29 – 2.77)	1.79 (1.21 – 2.64)	1.72 (1.15 – 2.57)
Total cases	1069			
P for trend		0.004	0.002	0.003

Abbreviations: RR, relative risk; CI, confidence interval, BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DFE, dietary folate equivalents; METS, metabolic equivalents

*Number of incident cases of psoriasis

†Adjusted for age, smoking (never, current, past), BMI (21–22.9, 23–24.9, 25–27.4, 27.5–29.9, 30–34.9, ≥35 kg/m^2), quintiles of DFE, and physical activity in quintiles of METS per week

‡Adjusted for age, smoking (never, current, past), BMI (continuous), quintiles of DFE and physical activity in quintiles of METS per week

Table 3

Age-adjusted and multivariate relative risks for incident psoriasis by type of alcohol beverage among women

Beer (# drinks)	N	RR (95% CI) Age-adjusted	RR (95% CI) Multivariate *
None (referent)	817	1.00	1.00
1 – 3/mo	112	1.02 (0.83 – 1.24)	1.02 (0.84 – 1.25)
1/wk	37	0.80 (0.57 – 1.10)	0.83 (0.60 – 1.16)
2 – 4/wk	21	0.80 (0.50 – 1.18)	0.80 (0.51 – 1.23)
≥ 5/wk	22	1.81 (1.18 – 2.76)	1.76 (1.15 – 2.69)
Total cases†	1009		
P for trend		0.001	0.003
Light beer (# drinks)			
None (referent)	787	1.00	1.00
1 – 3/mo	115	0.93 (0.76 – 1.13)	0.92 (0.75 – 1.12)
1/wk	45	0.91 (0.67 – 1.21)	0.91 (0.67 – 1.23)
2 – 4/wk	33	0.88 (0.62 – 1.24)	0.87 (0.61 – 1.23)
≥ 5/wk	22	1.07 (0.70 – 1.64)	0.98 (0.64 – 1.51)
Total cases†	1002		
P for trend		0.47	0.78
White wine (# drinks)			
None (referent)	585	1.00	1.00
1 – 3/mo	238	1.01 (0.87 – 1.18)	1.07 (0.92 – 1.24)
1/wk	74	0.88 (0.69 – 1.12)	0.98 (0.77 – 1.25)
2 – 4/wk	74	1.11 (0.87 – 1.41)	1.26 (0.99 – 1.62)
≥ 5/wk	38	1.03 (0.74 – 1.43)	1.17 (0.84 – 1.63)
Total cases†	1009		
P for trend		0.78	0.35
Red wine (# drinks)			
None (referent)	725	1.00	1.00
1 – 3/mo	167	1.03 (0.87 – 1.22)	1.08 (0.91 – 1.28)
1/wk	47	0.72 (0.53 – 0.97)	0.78 (0.58 – 1.05)
2 – 4/wk	47	0.83 (0.62 – 1.12)	0.93 (0.69 – 1.26)
≥ 5/wk	23	0.80 (0.52 – 1.20)	0.88 (0.58 – 1.34)
Total cases†	1009		
P for trend		0.88	0.69
Liquor (# drinks)			
None (referent)	770	1.00	1.00
1 – 3/mo	155	1.11 (0.93 – 1.31)	1.08 (0.91 – 1.29)

Beer (# drinks)	N	RR (95% CI) Age-adjusted	RR (95% CI) Multivariate *
1/wk	38	0.86 (0.62 – 1.19)	0.85 (0.62 – 1.18)
≥ 2/wk	49	1.13 (0.85 –1.51)	1.08 (0.81 – 1.45)
Total cases†	1012		

Abbreviations: RR, relative risk; CI, confidence interval, BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DFE, dietary folate equivalents; METS, metabolic equivalents

*Simultaneously adjusted for age, smoking (never, current, past), BMI (21–22.9, 23–24.9, 25–27.4, 27.5–29.9, 30–34.9, ≥35 kg/m^2), quintiles of DFE and physical activity in quintiles of METS per week

†Number of cases may not total 1150 due to missing data

Table 4

Age-adjusted and multivariate relative risks for confirmed* incident psoriasis by amount of alcohol intake and amount of beer intake

Alcohol intake (drinks/wk)	N	RR (95% CI) Age-adjusted	RR (95% CI) Multivariate †
None (referent)	213	1.00	1.00
0.1 – 0.3	198	1.03 (0.85 – 1.25)	1.08 (0.89 – 1.31)
0.4 – 0.7	57	1.02 (0.76 – 1.36)	1.12 (0.83 – 1.50)
0.8 – 1.1	41	1.26 (0.90 – 1.77)	0.40 (0.99 – 1.97)
1.2 – 2.3	22	1.24 (0.80 – 1.93)	1.34 (0.85 – 2.08)
≥ 2.3	19	2.68 (1.67 – 4.29)	2.54 (1.57 – 4.10)
Total cases‡	550		
P for trend		0.0005	0.0002
Beer (# drinks)			
None (referent)	418	1.00	1.00
1 – 3/mo	68	1.20 (0.92 – 1.55)	1.20 (0.93 – 1.56)
1/wk	18	0.76 (0.47 – 1.22)	0.81 (0.50 – 1.29)
2 – 4/wk	12	0.85 (0.48 – 1.50)	0.88 (0.49 – 1.57)
≥ 5/wk	15	2.40 (1.40 – 3.96)	2.29 (1.36 – 3.85)
Total cases‡	531		
P for trend		<0.0001	0.001

Abbreviations: RR, relative risk; CI, confidence interval, BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DFE, dietary folate equivalents; METS, metabolic equivalents

*Confirmed using the psoriasis screening tool [32]

†Simultaneously adjusted for age, smoking (never, current, past), BMI (21–22.9, 23–24.9, 25–27.4, 27.5–29.9, 30–34.9, ≥35 kg/m^2), and physical activity in quintiles of METS per week

‡Number of cases may not total 577 due to missing data