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CLINICAL REPORT

Excessive Body Weight and Smoking Associates with a High Risk of Onset of Plaque Psoriasis

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Accumulating evidence indicates that body weight, alcohol and smoking are associated with psoriasis. However, these factors have scarcely been investigated in relation to onset and disease activity at onset of psoriasis. A population-based case-control study was performed including 373 cases with onset of first-time plaque psoriasis within 12 months and matched healthy controls. Psoriasis activity was measured using the Psoriasis Area and Severity Index (PASI). Analyses were performed using conditional logistic regression. In multivariable analyses for each unit increment in body mass index, there was statistically significant 9% increased risk for psoriasis onset and 7% higher risk for increased PASI. Obesity (body mass index ≥30) compared with normal body weight was associated with a two-fold increased risk for psoriasis onset. Smoking was associated with a 70% increased risk for onset, but was not related to PASI. A positive association with alcohol drinking was observed among men, but not among women. No associations were observed for weight gain and use of smokeless tobacco. Our results indicate that excessive body weight and smoking are risk factors for onset of psoriasis and that higher body mass index increases the PASI of plaque psoriasis at onset. ***Key words: body mass index; body weight; smoking; plaque psoriasis; psoriasis onset, severity.***

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Psoriasis is one of the most prevalent inflammatory diseases in Western society, affecting all age groups in about 3% of the population (1–3). There is substantial disease heterogeneity with distinct clinical phenotypes (4), dominated by chronic plaque psoriasis, as well as variability in disease severity (5).

Even though the genetic background is of importance for disease manifestations (6), modifiable lifestyle factors, such as body weight (7–10), alcohol consumption (10–14) and smoking (7, 9, 14–18), have also been associated with established psoriasis (19, 20). However, there is only limited data on the association of these factors with the first-time onset of psoriasis (21–23) and there is no data on the association of these factors with disease activity at onset.

In the present population-based case-control study we focused on plaque psoriasis because current tools for assessing disease activity are most reliable in this phenotype (24). The aim was to investigate relative body weight, alcohol consumption and smoking in relation to onset and disease activity in individuals with first-time psoriasis. We also studied such biologically plausible risk factors as recent weight gain and smokeless tobacco, which have been associated with inflammatory response (25).

METHODS

Study population

We performed a population-based case-control study of the first onset of psoriasis. Cases were recruited from the Stockholm area between January 2001 and January 2006. Patients were eligible for this study if they were over 15 years of age and presented with psoriasis lesions for the first time on non-hairy skin within 12 months prior to inclusion. They were recruited from a variety of sources described previously (family doctors, advertisements in freely available daily newspapers, Swedish Psoriasis Association's internet site and referral from dermatologists in Stockholm). The information in the advertisement was short and general. The included cases constitute part of the Stockholm Psoriasis Cohort (SPC), cases still at school that filled in a slightly different questionnaire and were not analysed in the present study (27). All cases underwent a medical examination at the Dermatology Clinic at Karolinska University Hospital. The study physician asked the patient in detail when the first ever lesion occurred. The psoriasis "onset" point was set by the physician based on information from the patient. Controls were matched for sex, age in one year intervals, postal code number and randomly selected from the Swedish Population Registry. Six healthy equivalent controls were chosen for each case and after further random selection only one of them was invited to participate. If the person actively declined to participate in the study or failed to respond to a postal questionnaire after two reminders, a new equivalent control was invited.

Of the 565 patients initially included in the SPC, we excluded 7 misclassified patients, whose diagnosis was not confirmed at a follow-up examination. The remaining 558 psoriasis cases were identified, and 88% of these could be matched with healthy controls, yielding 493 matched pairs. Furthermore, we excluded 108 patients with phenotypes other than plaque psoriasis and 12 patients who were already on systemic treatment for psoriasis at inclusion. After these restrictions 373 matched pairs were available for analysis in the present study.

The study was approved by the Regional Committee of Ethics in Stockholm and carried out according to the principles of the Declaration of Helsinki.

Outcome assessment

Structured clinical examination was performed for diagnosis and inclusion of the cases by one of three dermatologists involved in the study. Each clinical examination resulted in phenotypic classification and assessment of disease activity using the Psoriasis Area and Severity Index (PASI) (28). PASI is a validated instrument with high inter-observer reliability (29). Psoriasis was classified into phenotypic subsets, such as plaque, guttate or other phenotypes representing palmoplantar, pustular, erythrodermic and inverse psoriasis (27). Diagnosis of psoriatic arthritis was completed by a rheumatologist following established criteria (33).

Exposure assessment

All patients and controls answered an identical self-administered questionnaire. Body mass index (BMI) was calculated by dividing self-reported weight (in kg) by height (in m^2). A validation of self-reported weight compared with measured weight at inclusion was performed in the first 100 consecutive cases (Pearson's correlation coefficient = 0.95). Economic status was assessed by self-reports on a 5-point scale (very good = 1, good = 2, either good or bad = 3, bad = 4 and very bad = 5) and dichotomized into "high" (1–3) and "low" (4–5) status. Weight gain was assessed by self-reported weight gain during the last 12 months (yes/no) and an open question about actual weight gain in kg. There were also questions about current and recent (last 24 months) pregnancies. Questions on present alcohol consumption included predefined frequency categories (never, ≤once a month, 2–4 times a month, 2–3 times a week, and ≥4 times a week) and predefined number of standard drinks (one standard drink defined as 15 cl wine, 4 cl liquor, or 33 cl beer) at one occasion (1–2, 3–4, 5–6, 7–9, or ≥10 drinks). Participants were asked about present smoking status (regular current, occasional current, stopped within the last 12 months, stopped more than 12 months ago, or never smoker). The same questions were asked about smokeless tobacco use.

Statistical analysis

Odds ratios (OR) were calculated with 95% confidence intervals (CI) using conditional logistic regression analysis with 1:1 matching of cases and controls.

BMI was classified according to the World Health Organization (WHO) (30): normal body weight (BMI < 25 kg/m^2), overweight (BMI 25–29.9 kg/m^2) and obesity (BMI ≥ 30 kg/m^2). BMI was also analysed as a continuous variable. Weight gain during the last 12 months was classified into two categories (< 5 kg or ≥ 5 kg) after exclusion of woman with current pregnancy or pregnancy during the last 24 months. Weight gain was also analysed as a continuous variable in kg and as BMI increment in %. The alcohol variable was created by multiplying the frequency of alcohol drinking by the number of drinks on one occasion. Alcohol consumption was analysed as total monthly consumption of drinks. Three categories of alcohol consumption were based on the tertile distribution among the controls. "Non-smokers" category included those who never smoked or quitted more than 12 months ago; "smokers" were defined as regular current, occasional current or those who quitted during the last 12 months. "Smokeless tobacco non-users" or "smokeless tobacco users" categories were defined as for smoking. Distributions among cases and controls were compared using a χ^2 test.

Univariate analyses were performed first and variables that were statistically significant or considered as confounders were included as covariates in the final multivariable model. To test for the statistical significance for interactions between the variables included in the final model, likelihood-ratio tests were performed in a conditional logistic regression model. The effect modification of the studied associations by sex was tested using χ^2 test statistics for ratio of discordant matched pairs (31).

Our patients presented mostly mild psoriasis and we constructed equal PASI subgroups based on tertile distribution according to the distribution in the sample. Ordinal logistic regression was used to calculate the odds for being in the two higher PASI subgroups at onset compared with the lowest PASI group (32). Trend across ordered groups was assessed by using non-parametric Kruskal-Wallis test.

All analyses were conducted using the SPSS 14.0 for Windows (SPSS Inc, Chicago, IL, USA, 2005) and STATA 8 (StataCorp LP, College Station, TX, USA, 2003) software packages.

RESULTS

Among 373 patients with first-time onset psoriasis included in the present study, psoriasis arthritis and entesitis were diagnosed in 27% of the patients and family history of psoriasis in the first-degree relatives was reported by 29% of patients. The vast majority of patients presented mild psoriasis, moderate psoriasis (PASI 10–20) was present in 4.5% and severe psoriasis (PASI > 20) in 0.5% of the patients. The age of participants at inclusion ranged from 17 to 84 years.

There was no statistically significant difference in economic status between cases and controls (χ^2, $p = 0.7$); "high" compared with "low" status (OR = 0.9; 95% CI 0.6–1.6). The distribution of the studied variables is presented in Table I.

Body weight and weight gain

The risk of the first plaque psoriasis onset was statistically significantly positively associated with relative body weight. Obesity compared with normal body weight was associated with two-fold increased risk (p for trend < 0.001) (Table II). One unit increment in BMI was associated with a 9% higher risk (OR = 1.09; 95% CI 1.04–1.16; p for trend = 0.001). When BMI was adjusted for weight gain in kg or % increase in BMI, the results were similar (OR = 1.10; 95% CI 1.04–1.16; p for trend = 0.001 and OR = 1.09; 95% CI 1.04–1.15; p for trend = 0.001, respectively). There was no statistically significant association of the weight gain (≥ 5 kg) during the last 12 months with plaque psoriasis onset.

The disease activity of plaque psoriasis at the first onset measured by PASI was statistically significantly positively associated with increased body weight compared with normal weight (Table III). One unit increment in BMI was associated with a 13% higher risk (OR = 1.13; 95% CI 1.03–1.25; p for trend = 0.01) in the subgroup with PASI ≥ 3.5 and a 23% higher risk

Table I. *Distribution of relative body weight and lifestyle factors in first plaque psoriasis onset cases and matched controls[a] and in the cases divided by sex and disease activity*

	Controls	Cases			Disease activity (PASI		
		All	Women	Men	0–1.9	2.0–3.4	≥3.5
	n = 373	*n* = 373	*n* = 212	*n* = 161	*n* = 123	*n* = 103	*n* = 124
Women %	57	57	100	0	61	58	53
Age, years, mean ± SD	46 ± 16	46 ± 16	48 ± 16	45 ± 15	47 ± 15	48 ± 15	46 ± 16
PASI, mean ± SD	–	3.5 ± 3.3	3.2 ± 2.9	3.9 ± 3.7	1.1 ± 0.5	2.6 ± 0.4	6.8 ± 3.8
Time to inclusion, months, mean ± SD	–	6.9 ± 3.8	6.9 ± 3.8	6.8 ± 3.8	7.4 ± 3.7	6.9 ± 4.0	6.2 ± 3.8
BMI, %, mean ± SD	25.0 ± 3.7	25.7 ± 4.4	25.4 ± 4.8	26.2 ± 3.8	25.3 ± 4.0	25.7 ± 4.6	26.3 ± 4.7
<25 kg/m²	57	48	51	43	51	46	44
25–29.9 kg/m²	34	37	35	40	37	39	37
≥30 kg/m²	9	15	13	17	11	16	19
Weight gain, ≥5 kg, %[b]	15	17	22	11	23	11	14
Alcohol, %, mean ± SD	18 ± 21	19 ± 23	13 ± 14	27 ± 29	18 ± 18	22 ± 28	19 ± 23
0–4 drinks/month	30	27	34	17	24	28	28
5–19 drinks/month	38	39	40	37	43	34	39
≥20 drinks/month	32	34	26	46	33	39	33
Smokers, %[c]	22	37	39	34	42	29	39
Smokeless tobacco users, %[c]	16	15	5	27	11	22	9

[a]Matched for age, sex and postal code number. Matched pairs for cases and controls: body mass index (BMI), *n* = 369; weight gain *n* = 316; alcohol, *n* = 354; smoking, *n* = 289 and snuff use; *n* = 254.
[b]Weight gain during last the 12 months excluding pregnant women or pregnancies during the last 24 months.
[c]Smokers/smokeless tobacco users = daily, occasional and former ≤ 12 months; non-smokers/ non-snuffers = never and former > 12 months.
PASI: Psoriasis Area and Severity Index; SD: standard deviation.

(OR = 1.23; 95% CI 1.10–1.36; *p* for trend < 0.001) in the subgroup with PASI 2.0–3.4. There was no statistically significant association in the group with the lowest PASI 0–1.9.

Comparing three groups with increasing plaque psoriasis activity by using the multivariable ordinal logistic regression analysis (adjusted for age, sex, socioeconomic factors, time to the inclusion, weight gain, alcohol intake and smoking), we observed that one unit increment in BMI was statistically significantly associated with a 7% higher risk of increased psoriasis activity measured by PASI at the onset (OR = 1.07: 95% CI 1.01–1.13); five BMI units increment was associated with a 38% increased risk (OR = 1.38: 95% CI 1.04–1.83).

Table II. *Multivariable odds ratios[a] (OR) with 95% confidence intervals (95% CI) for plaque psoriasis onset and for all and for women and men separately in relation to relative body weight, weight gain, alcohol consumption and smoking*

	All	Women	Men
	OR (95% CI)[a]	OR (95% CI)[a]	OR (95% CI)[a]
BMI			
<25 kg/m²	1 (ref)	1 (ref)	1 (ref)
25–29.9 kg/m²	1.6 (1.0–2.4)	2.0 (1.0–4.0)	1.2 (0.7–2.1)
≥30 kg/m²	2.0 (1.1–3.6)	1.4 (0.6–3.4)	2.3 (0.9–5.9)
Weight gain last 12 months[b]			
<5 kg			
≥5 kg	0.9 (0.5–1.5)	0.9 (0.4–1.9)	0.9 (0.4–2.3)
Alcohol			
0–4 drinks/month	1 (ref)	1 (ref)	1 (ref)
5–19 drinks/month	1.5 (0.9–2.6)	0.7 (0.3–1.5)	3.4 (1.4–8.1)
≥20 drinks/month	1.7 (1.0–3.0)	1.0 (0.4–2.3)	3.1 (1.4–7.2)
Smoking[c]			
Non-smokers	1 (ref)	1 (ref)	1 (ref)
Smokers	1.7 (1.1–2.6)	3.0 (1.5–5.7)	1.3 (0.7–2.4)

[a]OR from conditional logistic regression model (adjusted for age, sex, post code) including body mass index (BMI), weight gain, alcohol and smoking.
[b]Weight gain during last 12 months excluding pregnant women or with pregnancies during the last 24 months.
[c]Smokers = daily, occasional and former ≤ 12 months, non-smokers = never and former smokers > 12 months.

Alcohol consumption

We observed a statistically significant positive association between alcohol consumption and psoriasis onset in men, but not in women. We did not observe a clear

Table III. *Multivariable odds ratios[a] (OR) with 95% confidence intervals (95% CI) of the disease activity at plaque psoriasis onset in relation to relative body weight, alcohol consumption and smoking*

	Plaque psoriasis activity at onset		
	PASI 0–1.9	PASI 2.0–3.4	PASI ≥3.5
	OR (95% CI)[a]	OR (95% CI)[a]	OR (95% CI)[a]
BMI			
< 25 kg/m²	1 (ref)	1 (ref)	1 (ref)
25–29.9 kg/m²	1.8 (0.9–3.9)	2.5 (1.0–6.3)	2.0 (0.8–4.8)
≥ 30 kg/m²	1.6 (0.5–5.5)	7.5 (1.7–33.9)	3.3 (1.0–10.6)
Alcohol			
0–4 drinks/month	1 (ref)	1 (ref)	1 (ref)
5–19 drinks/month	4.5 (1.4–14.5)	1.3 (0.4–3.6)	0.8 (0.3–2.0)
≥ 20 drinks/month	2.8 (0.9–8.9)	1.7 (0.6–4.7)	1.2 (0.4–3.4)
Smoking[b]			
Non-smokers	1 (ref)	1 (ref)	1 (ref)
Smokers	1.7 (0.8–3.8)	1.7 (0.7–4.1)	2.4 (1.9–5.3)

[a]OR from conditional logistic regression model adjusted for matched variables (age, sex, post code) and body mass index (BMI), weight gain, alcohol and smoking.
[b]Smokers = daily, occasional and former ≤ 12 months, non-smokers = never and former smokers > 12 months.

dose-response association between total amount of alcoholic drinks consumed during a month and risk of disease onset, (p for trend = 0.4) (Table II). Heavy episodic drinking (≥ 5 drinks on one occasion) compared with 4 drinks or less was not statistically significantly associated with the plaque psoriasis onset (OR = 1.4; 95% CI 0.8–2.4).

Disease activity at onset was not associated with alcohol consumption in the multivariable ordinal logistic regression analysis; for drinking ≥ 20 drinks compared with ≤ 4 drinks per month (OR = 0.8; 95% CI 0.5–1.5, p for trend = 0.6).

Smoking and smokeless tobacco

In the multivariable analysis we observed that smokers had a statistically significantly 70% higher risk of disease onset compared with non-smokers (Table II). In the multivariable model analysing smoking status as three categories, we observed that current (regular or occasional) smokers had OR = 1.6 (95% CI 1.0–1.4) and ex-smokers (quitted more than 12 months ago or quitted during the last 12 months) had OR = 0.9 (95% CI 0.5–1.4) compared with never smokers. No association was observed between current smokeless tobacco use and the onset of plaque psoriasis (OR = 1.0; 95% CI 0.6–1.9).

Regarding disease activity, smoking was statistically significantly associated with the disease in the subgroup of psoriasis cases with the highest PASI ≥ 3.5 (Table III). In the multivariable ordinal logistic regression analysis smoking was not associated with the activity of plaque psoriasis at onset (OR = 1.0; 95% CI 0.6–1.6).

There was no statistically significant effect modification by sex of the observed associations for overweight (χ^2 p = 0.6), obesity (χ^2 p = 0.8), alcohol consumption ≥ 20 drinks a month (χ^2 p = 0.4) and smoking (χ^2 p = 0.2).

DISCUSSION

In the present population-based case-control study we observed a statistically significant positive association of relative body weight with onset of plaque psoriasis. Furthermore, our data suggested that higher BMI may be associated with higher psoriasis activity (PASI) at onset. Smokers had a statistically significantly increased risk of developing plaque psoriasis, but we did not observe association of smoking with higher disease activity at onset. Overall no clear associations were observed between alcohol drinking and disease onset or activity. However, our data indicated that men drinking more alcohol may have higher risk for psoriasis onset. Recent weight gain and smokeless tobacco were not associated with plaque psoriasis.

Our results showing that overweight and obesity may increase the risk to develop psoriasis are in agreement with a prospective cohort study in the general UK population (23), a prospective cohort of American women (Nurses' Health Study II) (21), and an Italian case-control study (22). To the best of our knowledge there is no previous study investigating increased BMI as risk factor for increased disease activity at onset of psoriasis. Our results indicated a possible positive association between increased BMI and disease activity, but there was no clear dose-response association. PASI might be affected by increased body surface area in very obese individuals; however, in our study we had only 3 cases with BMI > 40, one in each PASI group. Previous studies of established chronic psoriasis have shown positive association between increased relative body weight and disease activity; risk estimates varied from OR = 3.0 (95% CI 1.5–6.0) to OR = 1.7 (95% CI 1.6–1.9) when comparing obesity with normal weight (7, 9, 33).

The positive association of smoking with risk for onset of psoriasis observed in our study is in agreement with previous studies (15, 22, 23). However, also in line with previous results for the established disease, smoking was not associated with disease activity at onset in our study (9, 14). Lack of an overall and dose-response association between alcohol consumption and psoriasis onset in women in our study is in agreement with previous studies (13, 17). Our results for men, showing that alcohol consumption might be a risk factor for psoriasis onset, even though we have not observed a dose-response or association between binge drinking and psoriasis onset, are in agreement with two previous studies (13, 18). In the Italian study men drinking more than 2 drinks/day compared with non-drinkers had OR = 1.9 (95% CI 1.0–3.3) (13). In the Finnish study on heavy drinking men, consumption of 8 drinks/day compared with non-drinking was associated with two-fold increased risk for psoriasis, OR = 2.2 (95% CI 1.3–3.9) (18).

Psoriasis is an inflammatory disease. Excessive adipose tissue in overweight and obesity has been shown to produce pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-α) (34), which is involved in psoriasis (35). The constituents of cigarette smoke may also have pro-inflammatory effects (26, 36). These pro-inflammatory effects of excessive adiposity and cigarette smoke may, at least partly, reflect biological mechanisms underlying the positive associations with psoriasis observed in our study.

The strength of our study is the population-based design and early inclusion of psoriasis patients from a well-defined source population. Three trained dermatologists examined all patients, which enhances the homogenous evaluation of patients. The matched and randomly selected healthy controls were from the same study area and the response rate of 88% can be considered as high. However, as in any case-control design we cannot exclude a potential risk for selection bias. Individuals with social or alcohol problems might

potentially be underrepresented in the study, which could lead to attenuation of the observed associations for some risk factors. However, we tried to prevent selection bias in case sampling by broad recruitment using several approaches and by use of short and simple information about the study. Furthermore, we avoided presenting specific aims for the study in order to avoid self-selection of the study participants. Another possible weakness could be assessment of the disease onset. Even though we asked questions about the occurrence of similar lesions in the past we cannot exclude that previous minor mild psoriasis could be forgotten by some patients. Use of self-reported information may lead to misclassification of exposures and, in consequence, to attenuation of observed associations. Although the studied exposures were assessed shortly after disease onset, we cannot exclude a potential risk for recall bias in our study similarly to other studies with case-control design. The effect modification by sex of the above associations was not statistically significant, but the matched design did not allow for more detailed analyses. Therefore we cannot exclude that there may be true differences in risk factors between women and men.

In summary, we observed that increased body weight was statistically significantly positively associated with the onset of plaque psoriasis and may be associated with the risk of higher disease activity. Smoking was also associated with risk for onset of plaque psoriasis, but did not associate with disease activity. Alcohol might be associated with psoriasis onset in men, but was not associated with either plaque psoriasis onset or activity at onset in women. Recent weight gain and smokeless tobacco use were not associated with plaque psoriasis. Further studies in larger groups of patients are needed to examine these risk factors in relation to other subtypes of psoriasis.

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REFERENCES

1. Braathen LR, Botten G, Bjerkedal T. Prevalence of psoriasis in Norway. Acta Derm Venereol Suppl 1989; 142: 5–8.
2. Smith AE, Kassab JY, Rowland Payne CM, Beer WE. Bimodality in age of onset of psoriasis, in both patients and their relatives. Dermatology 1993; 186: 181–186.
3. Gelfand JM, Weinstein R, Porter SB, Neimann AL, Berlin JA, Margolis DJ. Prevalence and treatment of psoriasis in the United Kingdom: a population-based study. Arch Dermatol 2005; 141: 1537–1541.
4. Griffiths CE, Christophers E, Barker JN, Chalmers RJ, Chimenti S, Krueger GG, et al. A classification of psoriasis vulgaris according to phenotype. Br J Dermatol 2007; 156: 258–262.
5. Gelfand JM, Feldman SR, Stern RS, Thomas J, Rolstad T, Margolis DJ. Determinants of quality of life in patients with psoriasis: a study from the US population. J Am Acad Dermatol 2004; 51: 704–708.
6. Grjibovski AM, Olsen AO, Magnus P, Harris JR. Psoriasis in Norwegian twins: contribution of genetic and environmental effects. J Eur Acad Dermatol Venereol 2007; 21: 1337–1343.
7. Herron MD, Hinckley M, Hoffman MS, Papenfuss J, Hansen CB, Callis KP, et al. Impact of obesity and smoking on psoriasis presentation and management. Arch Dermatol 2005; 141: 1527–1534.
8. Hamminga EA, van der Lely AJ, Neumann HA, Thio HB. Chronic inflammation in psoriasis and obesity: implications for therapy. Med Hypotheses 2006; 67: 768–773.
9. Neimann AL, Shin DB, Wang X, Margolis DJ, Troxel AB, Gelfand JM. Prevalence of cardiovascular risk factors in patients with psoriasis. J Am Acad Dermatol 2006; 55: 829–835.
10. Sommer DM, Jenisch S, Suchan M, Christophers E, Weichenthal M. Increased prevalence of the metabolic syndrome in patients with moderate to severe psoriasis. Arch Dermatol Res 2006; 298: 321–328.
11. Braathen LR, Botten G, Bjerkedal T. Psoriatics in Norway. A questionnaire study on health status, contact with paramedical professions, and alcohol and tobacco consumption. Acta Derm Venereol Suppl 1989; 142: 9–12.
12. Monk BE, Neill SM. Alcohol consumption and psoriasis. Dermatologica 1986; 173: 57–60.
13. Naldi L, Peli L, Parazzini F. Association of early-stage psoriasis with smoking and male alcohol consumption: evidence from an Italian case-control study. Arch Dermatol 1999; 135: 1479–1484.
14. Fortes C, Mastroeni S, Leffondre K, Sampogna F, Melchi F, Mazzotti E, et al. Relationship between smoking and the clinical severity of psoriasis. Arch Dermatol 2005; 141: 1580–1584.
15. Setty AR, Curhan G, Choi HK. Smoking and the risk of psoriasis in women: Nurses' Health Study II. Am J Med 2007; 120: 953–959.
16. Mills CM, Srivastava ED, Harvey IM, Swift GL, Newcombe RG, Holt PJ, et al. Smoking habits in psoriasis: a case control study. Br J Dermatol 1992; 127: 18–21.
17. Poikolainen K, Reunala T, Karvonen J. Smoking, alcohol and life events related to psoriasis among women. Br J Dermatol 1994; 130: 473–477.
18. 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: 780–783.
19. Raychaudhuri SP, Gross J. A comparative study of pediatric onset psoriasis with adult onset psoriasis. Pediatr Dermatol 2000; 17: 174–178.
20. Marino MG, Carboni I, De Felice C, Maurici M, Maccari F, Franco E. Risk factors for psoriasis: a retrospective study on 501 outpatients clinical records. Ann Ig 2004; 16: 753–758.
21. Setty AR, Curhan G, Choi HK. Obesity, waist circumference, weight change, and the risk of psoriasis in women: Nurses' Health Study II. Arch Intern Med 2007; 167: 1670–1675.

22. Naldi L, Chatenoud L, Linder D, Belloni Fortina A, Peserico A, Virgili AR, et al. Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: results from an Italian case-control study. J Invest Dermatol 2005; 125: 61–67.
23. Huerta C, Rivero E, Rodriguez LA. Incidence and risk factors for psoriasis in the general population. Arch Dermatol 2007; 143: 1559–1565.
24. Schmitt J, Wozel G. The psoriasis area and severity index is the adequate criterion to define severity in chronic plaque-type psoriasis. Dermatology 2005; 210: 194–199.
25. Fogarty AW, Glancy C, Jones S, Lewis SA, McKeever TM, Britton JR. A prospective study of weight change and systemic inflammation over 9 y. Am J Clin Nutr 2008; 87: 30–35.
26. Makrygiannakis D, af Klint E, Lundberg IE, Lofberg R, Ulfgren AK, Klareskog L, et al. Citrullination is an inflammation-dependent process. Ann Rheum Dis 2006; 65: 1219–1222.
27. Mallbris L, Larsson P, Bergqvist S, Vingård E, Granath F, Ståhle M. Psoriasis phenotype at disease onset: clinical characterization of 400 adult cases. J Invest Dermatol 2005; 124: 499–504.
28. Fredriksson T, Pettersson U. Severe psoriasis – oral therapy with a new retinoid. Dermatologica 1978; 157: 238–244.
29. Berth-Jones J, Grotzinger K, Rainville C, Pham B, Huang J, Daly S, et al. A study examining inter- and intrarater reliability of three scales for measuring severity of psoriasis: Psoriasis Area and Severity Index, Physician's Global Assessment and Lattice System Physician's Global Assessment. Br J Dermatol 2006; 155: 707–713.
30. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 2000; 894: i–xii, 1–253.
31. Rothman KJ. Modern Epidemiology. Boston: Little, Brown & Co; 1986; p. 279–280.
32. Brant R. Assessing proportionality in the proportional odds model for ordinal logistic regression. Biometrics 1990; 46: 1171–1178.
33. Christensen TE, Callis KP, Papenfuss J, Hoffman MS, Hansen CB, Wong B, et al. Observations of psoriasis in the absence of therapeutic intervention identifies two unappreciated morphologic variants, thin-plaque and thick-plaque psoriasis, and their associated phenotypes. J Invest Dermatol 2006; 126: 2397–2403.
34. de la Maza MP, Olivares D, Hirsch S, Sierralta W, Gattas V, Barrera G, et al. Weight increase and overweight are associated with DNA oxidative damage in skeletal muscle. Clin Nutr 2006; 25: 968–976.
35. Lowes MA, Bowcock AM, Krueger JG. Pathogenesis and therapy of psoriasis. Nature 2007; 445: 866–873.
36. Francus T, Romano PM, Manzo G, Fonacier L, Arango N, Szabo P. IL-1, IL-6, and PDGF mRNA expression in alveolar cells following stimulation with a tobacco-derived antigen. Cell Immunol 1992; 145: 156–174.