STUDY

Impact of Obesity and Smoking on Psoriasis Presentation and Management

Mark D. Herron, MD; Michael Hinckley, BA; Matthew S. Hoffman, BS; Jason Papenfuss, MD; Christopher B. Hansen, MD; Kristina P. Callis, MD; Gerald G. Krueger, MD

Objective: To study the impact of obesity and smoking on psoriasis.

Design: Cross-sectional study.

Setting: University of Utah Department of Dermatology clinics.

Patients: A case series of patients with psoriasis enrolled in the prospective Utah Psoriasis Initiative (UPI) (which carefully performs phenotyping of patients with psoriasis) was compared with 3 population databases: the Behavioral Risk Factor Surveillance System of the Utah population, the 1998 patient-member survey from the National Psoriasis Foundation, and 500 adult patients who attend our clinics and do not have psoriasis (nonpsoriatic population).

Results: The prevalence of obesity in patients within the UPI population was higher than that in the general Utah population (34% vs 18%; $P<.001$) and higher than that in the nonpsoriatic population attending our clinics. Assessment of body image perception with a standardized diagram in the UPI group resulted in the median body image score of normal weight at 18 years of age and the onset of psoriasis, but it transitioned to overweight at the time of enrollment in the UPI. Thus, obesity appears to be the consequence of psoriasis and not a risk factor for onset of disease. We did not observe an increased risk for psoriatic arthritis in patients with obesity; furthermore, obesity did not positively or negatively affect the response or the adverse effects of topical corticosteroids, light-based treatments, and systemic medications. The prevalence of smoking in the UPI population was higher than in the general Utah population (37% vs 13%; $P<.001$) and higher than in the nonpsoriatic population (37% vs 25%; $P<.001$). We found a higher prevalence of smokers in the obese population within the UPI than in the obese population within the Utah population (25% vs 9%; $P<.001$).

Conclusions: Patients with psoriasis attending the University of Utah Dermatology Clinics were more likely to be obese and to smoke compared with nonpsoriatic patients and more likely to be obese compared with other large cohorts with psoriasis. Smoking appears to have a role in the onset of psoriasis, but obesity does not. The high prevalence of obesity and smoking in a psoriasis cohort has not been previously noted; if confirmed, it supports the prediction that a significant portion of patients with psoriasis will have the comorbid conditions and public health issues of those with obesity and smoke.

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Author Affiliations:
Departments of Dermatology, University of Utah School of Medicine, Salt Lake City (Messrs Hinckley and Hoffman and Drs Callis and Krueger), Medical University of South Carolina, Charleston (Dr Papenfuss), and The University of Texas Southwestern Medical Center, Dallas (Dr Hansen). Dr Herron is in private practice in Montgomery, Ala.

OBESITY AND SMOKING have emerged as factors that appear to have an impact on many aspects of psoriasis, from natural history and management to possible public health issues. To study this further, we used information being gathered by the Utah Psoriasis Initiative (UPI). The UPI is a prospective study to characterize phenotypic features of patients with psoriasis (hereafter referred to as psoriasis patients) and their family members. The hypothesis underlying the UPI is that genotype predicts phenotype in psoriasis and that stratifying for phenotype will provide more informative genotyping. Patients enrolled in the study provide history on their psoriasis and response to therapy and undergo an examination by a research fellow, who is a physician trained by two of us (K.P.C. and G.G.K). This report focuses on the clinical features of those UPI subjects who are obese and/or smoke.

See also pages 1537, 1542, 1549, 1556, 1580, and 1589

During the past decade, obesity has become epidemic within the United States.[1-7] The prevalence of obesity has increased steadily during the past 2 decades. Obesity is a hazard that contributes to distinct diseases such as hypertension, type 2 diabetes mellitus, and obstructive sleep apnea. Abnormal eating behavior and a sedentary lifestyle have contributed to the epidemic of obesity on a global scale.[8-15] In the Utah population, the prevalence of obesity among adults has increased dur-

ing the past decade.[1,4,6,7] Although Utah has not been protected against the national epidemic of obesity, the prevalence rates of obesity are among the lowest in the nation.[1] From a study on Utah health, behavior, and risk, the percentage of students in grades 9 through 12 who were overweight was 6.2% compared with a 10.5% national average.[16]

The mean weight of patients with psoriasis in recent clinical trials was more than 90 kg.[17,18] This finding plus a review of the literature that revealed that study patients using systemic agents had excess body weight led us to hypothesize that obese patients are more likely to have severe psoriasis.[17-20] We also hypothesized that obesity increases the pressure on the patient carrying a genotype for psoriasis to develop psoriasis, and thus we would observe that obesity preceded the onset of psoriasis in those enrolling in the UPI. Furthermore, it was hypothesized that obesity would alter treatment choice and response. Obesity may increase the likelihood of adverse effects of systemic agents such as methotrexate and cyclosporine.[21-26]

Although smoking has not been studied in a large cohort of psoriasis patients in the United States, it has been characterized as a comorbidity of psoriasis in Europe.[27-29] Thus, we analyzed the data of the UPI cohort to gain insight into the role smoking may have in the development and presentation of psoriasis.

METHODS

UTAH PSORIASIS INITIATIVE

The UPI is a prospective collection of phenotypic data of individuals with psoriasis to test the hypothesis that genotype dictates phenotype. In 2002, patients began enrolling in the UPI. Men and women enrolled in the UPI have undergone phenotyping by responses to structured questionnaires and physical examination by 2 trained clinical research fellows J.P. and C.B.H.). All subjects carry an unequivocal diagnosis of psoriasis and all were nonselectively recruited from the population of patients older than 18 years who attend or have attended Department of Dermatology clinics at the University of Utah, Salt Lake City. All have been examined by one or two of us (K.P.C. and G.G.K.). More than 500 patients have been enrolled.

Before physical examination and interview, each subject was asked to sign an informed consent and complete a questionnaire. The questionnaire provided investigators with a detailed account of a variety of phenotypic traits, including but not limited to age at onset, site of initial lesions, diagnosis and/or symptoms of psoriatic arthritis, comorbid medical conditions, social habits (tobacco and alcohol use), distribution of psoriatic lesions, exacerbating and ameliorating conditions, therapeutic history, and global assessment of psoriasis at present and at its worst. The questionnaire also queried the family history (known first-, second-, and third-degree relatives with psoriasis).

Subjects then underwent a physical examination and interview. The interview by the research fellow (J.P. or C.B.H.) reviewed current and past existence of other forms of psoriasis (guttate, palmar/plantar, inverse, pustular, or erythrodermic) and generated additional information on the patient's response to various treatment modalities. Specifically, the interviewer queried whether treatment led to clearing or partial clearing or had no to minimal effect, which also discerned agents that were no longer effective or no longer tolerable. The patient was guided through a phenotyping exercise of his or her disease in the untreated state using a composite photograph. The photograph depicted a profile of presentation of psoriasis (from thin lesions with minimal scale to very thick lesions with severe scale). Patients were also asked to generate an estimate of body surface area (BSA) covered with psoriasis when the disease was the worst it has ever been, using 1 palm to represent 1% of their BSA. The examiner also assessed the BSA and the presence of nail disease, reviewed the arthritis or arthralgia symptoms, and examined for signs of psoriatic arthritis.

ANTHROPOMETRIC MEASUREMENTS

We determined the body mass index (BMI) of patients in the UPI and in the 1998 patient-member survey from the National Psoriasis Foundation (NPF) on the basis of self-reported height and weight. At enrollment into the UPI, subjects were asked to report their current height (in feet and inches), current weight (in pounds), and their weight (in pounds) at 18 years of age. For analysis, all values were then converted into BMI using the following equations:

$$\text{BMI} = (\text{Weight in Pounds} \times 703)/(\text{Square of Height in Inches})$$
$$= (\text{Weight in Kilograms})/(\text{Square of Height in Meters})$$

Clinically accepted guidelines note that a BMI from 25.0 to 29.9 is overweight; a BMI of greater than or equal to 30.0 is obese; and a BMI of greater than or equal to 35.0 is morbidly obese.[30-33]

COMPARATIVE/CONTROL POPULATION

Prevalence data on obesity, smoking, and smokers in the obese population within the state of Utah was obtained from Behavioral Risk Factor Surveillance System (BRFSS) Utah Prevalence Data. Further assessment of body weight and BMI came from 2 other sources. The first source was the 1998 NPF patient-membership survey, which was completed by more than 17 000 subjects; in this self-reported survey, height, weight, age at disease onset, family history of psoriasis, presence of inverse psoriasis, and BSA affected at the time of the survey were recorded. The second source was a survey of the prevalence of obesity and smoking in 500 patients older than 18 years who attended our dermatology clinics for reasons other than psoriasis (nonpsoriatic population or cohort).

MEASUREMENT OF BODY IMAGE PERCEPTION

At the time the UPI was initiated, we recorded the subject's self-reported weight and height at 18 years of age and at enrollment. Later, as we determined that many enrolling in the UPI were overweight, we began questioning subjects about their body image at the following time points: 18 years of age, the onset of psoriasis, and enrollment. This information was requested from enrollees in response to a mailed questionnaire for those who enrolled early in the initiative or at enrollment for those who entered later in the study. For this, participants were asked to choose an image that corresponded to the choices represented in the **Figure**. Stunkard et al[34] originally generated this diagram for use in studies of obesity and eating disorders. The body image diagram has subsequently been validated as a tool to assess body image perception.[35]

STATISTICAL ANALYSIS

All analyses and statistical comparisons were conducted with Stata (StataCorp LP, College Station, Tex) and SPSS (SPSS Inc, Chicago, Ill) statistical software. We compared the proportion of obese subjects and smokers in the UPI with the proportion

of smokers in the obese population in the other 2 groups, controlling for sex and age, by means of the Mantel-Haenszel χ^2 test. It was not possible to stratify by sex and age simultaneously in the Utah control population data because these data were not available from the BRFSS prevalence data. The status of the sex and age of individuals in the BRFSS was not known, and thus we reported a stratified analysis rather than a logistic regression model. For stratum-specific comparisons, an ordinary χ^2 test or, if the data were sparse, a Fisher exact test was used. We adjusted the stratum-specific *P* values for multiple comparisons using the multiple comparison procedure of Finner.[36] Effects such as the association between obesity and smoking and psoriasis were reported as prevalence odds ratios (ORs) with 95% confidence intervals (CIs).

Testing for associations between 2 categorical variables was performed with an ordinary χ^2 test or, if data were sparse, Fisher exact test. Testing for differences between groups on continuous variables was performed using an independent-groups *t* test. We tested for a linear relationship between BMIs at 18 years of age and age at onset using the Pearson correlation coefficient. The self-reported body image scores at 18 years of age, onset of psoriasis, and enrollment were treated as ordered categorical variables. To test for a difference in the ordered categorical body image across these 3 periods, we used the Friedman test, a nonparametric equivalent of a 1-sample repeated-measures analysis of variance.

RESULTS

The subject demographic data are summarized in **Table 1**. The mean age at onset of psoriasis in this cohort was in the third decade of life (Table 1). The mean age at enrollment was in the fifth decade of life. Most patients in the UPI had a normal BMI at 18 years of age, but 71% developed an overweight or obese BMI by enrollment. Sex and ethnic similarities and age differences between the UPI and Utah populations are given in Table 1. At enrollment, the mean age of the UPI cohort was 49.8 years; of the nonpsoriatic cohort, 44.7 years; of the general Utah population, 45.7 years; and of the NPF cohort, 54.6 years. The UPI population had significantly more obesity than the Utah population, when we controlled for sex and age (Mantel-Haenszel χ^2 test, $P<.001$) (**Table 2**). Results of the χ^2 analysis showed that subjects in the UPI had increased BMIs compared with the nonpsoriatic population.

In 1998, the NPF asked their members to participate in a membership survey, and 17 388 responded, for a response rate of more than 50%. Self-reported height and weight permitted a comparison of body weight between the UPI and NPF populations (Table 1). Sex, age, and ethnic differences between the UPI and NPF populations are also given in Table 1. The mean BMIs were in the overweight category for both the UPI population and NPF survey respondents. Both groups had a median age at onset in the third decade of life. The UPI population had significantly more obese subjects than did the NPF population when we controlled for age at enrollment and sex (Mantel-Haenszel χ^2 test, $P<.001$) (Table 2).

Another approach to assess the accuracy of self-reporting was to query about body image perception at 18 years of age, at onset of psoriasis, and at enrollment into the UPI. The mean BMI at 18 years of age was 22.0 (normal weight), and the median body image score at 18 years of age was 3 (normal image of weight). At the onset of psoriasis, the median body image score was 4 (normal image of weight). This differs from the BMI and the body image perception at enrollment, when the mean BMI was 29.0 (overweight) and the median body image score was 5 (overweight). The increased trend in higher body image score across the 3 time points was statistically significant ($P<.001$). Furthermore, there was no linear association between the BMIs at 18 years of age and at the age at onset of psoriasis ($r=-0.07$; $P=.09$). The results of the self-reported weight and the body image at each of the time points are harmonious and indicate that overweight and obesity came after the onset of psoriasis. Because of the strong heritability of psoriasis in the UPI cohort and because those with the earliest age at onset had the strongest family history, we determined that obesity in early life was not linked to a family history of psoriasis (OR, 0.78 [95% CI, 0.30-2.00]; $P=.60$). These features suggest that obesity is a consequence of psoriasis, not vice versa.

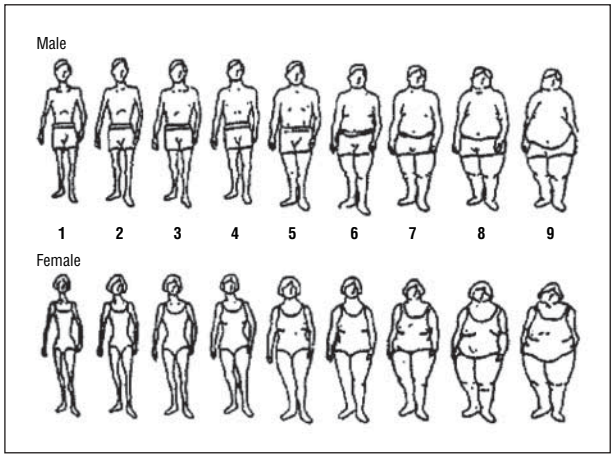

Figure. Diagram used to assess body image perception. Images 1 and 2 are considered underweight; images 3 and 4, normal weight; images 5 and 6, overweight; and images 7 through 9, obese. Adapted with permission from Stunkard et al.[34] Copyright 1983, Oxford University Press.

We analyzed several self-reported factors that might contribute to weight gain in the UPI population (n=301). Obese subjects with psoriasis were less likely than nonobese subjects to engage in physical activity at least 2 to 3 times a week for more than 30 minutes at a time; ie, 43% of obese subjects compared with 59% of nonobese subjects reported engaging in physical activity during the previous month (OR, 0.51 [95% CI, 0.32-0.82]; $P=.005$). Furthermore, 32% of obese subjects compared with 14% of nonobese subjects stated that arthritis impeded physical activity (OR, 2.82 [95% CI, 1.61-5.05]; $P=.001$). Obese subjects were more likely than nonobese subjects to have overweight and obese family members; ie, 74% of obese subjects compared with 34% of nonobese subjects stated that their parents or siblings were overweight or obese while they were growing up (OR, 5.33 [95% CI, 3.18-8.95]; $P=.001$).

Body surface area covered with disease was used to determine whether obesity is associated with more severe disease. The 1998 NPF survey was limited to 4 categories of BSA (≤2%, 3%-9%, 10%-20%, and >20%). For comparison, we scored mild disease as a BSA of 2% or

Table 1. Demographic Comparisons of the UPI, Utah, and NPF Populations*

Demographic Characteristic	UPI vs Utah Populations: UPI (n = 557)	UPI vs Utah Populations: Utah (n = 4080)	UPI vs Utah Populations: *P* Value	NPF vs UPI Populations: NPF (n = 17 378)†	NPF vs UPI Populations: *P* Value
Sex, No. (%)			.124		.009
Male	275 (49)	1873 (46)		7539 (43)	
Female	282 (51)	2207 (54)		9677 (56)	
Age at onset, y					
Mean (SD)	27.9 (17.0)			30.5 (17.4)	<.001
Median	25			27	
Age at enrollment, y					
Mean (SD)	49.8 (16.7)	45.7 (17.7)	<.001	54.6 (16.0)	<.001
Median	50	43		55	
BMI at enrollment, y					
Mean (SD)	29.1 (7.3)‡	26.2 (5.1)‡	<.001	26.9 (5.7)‡	<.001
Median	27.8	25.7		25.9	
Family history of psoriasis, No. (%)	323 (58)			10 414 (61)	.36
First-degree relative with psoriasis, No. (%)	261 (47)			7505 (44)	.09
Psoriatic arthritis, No. (%)	146 (26)			4923 (31)	.28
Race, No. (%)			.94		<.001
White non-Hispanic	520 (93)	3752 (92)		16 014 (94)	
Black non-Hispanic	1 (0.1)	13 (0.3)		123 (0.7)	
Hispanic	25 (4)	178 (4)		243 (1)	
Asian/Pacific Islander, non-Hispanic	5 (1)	43 (1)		254 (1)	
Native American, non-Hispanic	6 (1)	34 (1)		311 (2)	
BMI at enrollment, No. (%)					<.001
<25	160 (29)			6710 (40)	
25-30	205 (37)			6206 (37)	
30-35	96 (17)			2541 (15)	
>35	96 (17)			1323 (8)	

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); NPF, National Psoriasis Foundation; UPI, Utah Psoriasis Initiative.

*Blank cells indicate data not available. Percentages have been rounded and might not total 100.

†The BMI data are from 16 780 respondents; race, 16 945 respondents; age, 16 797 respondents; family history, 16 999; and arthritis, 15 806.

‡The BMI data fall within the overweight category of BMI (range, 25.0-30.0).

Table 2. Comparison of Prevalence of Obesity in the UPI, Utah, and NPF Populations

Stratification Variable	No. of Obese Subjects/Population (%): UPI	No. of Obese Subjects/Population (%): Utah	UPI vs Utah Population: Obesity Prevalence, OR (95% CI)	UPI vs Utah Population: Strata-Specific *P* Value*	UPI vs Utah Population: Summary *P* Value†	No. (%) of Obese Subjects/NPF Population	UPI vs NPF Populations: Obesity Prevalence, OR (95% CI)	UPI vs NPF Populations: Strata-Specific *P* Value*	UPI vs NPF Populations: Summary *P* Value†
Total	192/557 (34)	735/4080 (18)	2.39 (1.98-2.90)		<.001	3844/16 780 (23)	1.77 (1.48-2.12)		<.001
Sex					<.001				<.001
Male	90/275 (33)	387/1925 (20)	1.93 (1.47-2.55)	.002		1581/7539 (21)	1.83 (1.42-2.37)	.002	
Female	102/282 (36)	348/2336 (15)	3.24 (2.48-4.23)	.002		2263/9677 (23)	1.86 (1.45-2.38)	.002	
Age, y					<.001				<.001
18-34	26/120 (22)	139/1390 (10)	2.49 (1.56-3.98)	.004		309/1805 (17)	1.33 (.85-2.10)	.20	
35-50	56/161 (35)	235/1130 (21)	2.03 (1.43-2.90)	.004		1158/4766 (24)	1.66 (1.19-2.31)	.004	
51-65	72/176 (41)	233/779 (30)	1.62 (1.16-2.27)	.005		1528/5376 (28)	1.74 (1.28-2.37)	.004	
>65	38/100 (38)	127/654 (19)	2.54 (1.63-3.98)	.004		849/4833 (18)	2.88 (1.91-4.34)	.004	

Abbreviations: CI, confidence interval; NPF, National Psoriasis Foundation; OR, odds ratio; UPI, Utah Psoriasis Initiative.

*Calculated as Pearson χ^2 test with the *P* value adjusted for multiple comparisons using the procedure of Finner.[36]

†Calculated as a summary *P* value from the Mantel-Haenszel χ^2 test.

less and severe disease as a BSA of greater than 20%. Obese subjects were less likely than nonobese subjects to present with mild disease (BSA ≤2%) in the UPI population, at 38% and 52%, respectively (OR, 0.56 [95% CI, 0.38-0.81]; *P*=.002) and in the NPF survey, at 19% and 27%, respectively (OR, 0.67 [95% CI, 0.56-0.68]; *P*<.001). Therefore, obese subjects should be more likely than nonobese subjects to have severe disease (BSA >20%). This was true in the UPI population (11% and 4%, for the obese and nonobese cohorts, respectively; OR, 2.99 [95% CI,

Table 3. Comparison of Prevalence of Smoking in the UPI and Utah Populations

Stratification Variable	No. of Subjects Who Smoke/Population (%)		UPI vs Utah Populations		
	UPI	Utah	Smoking Prevalence, OR (95% CI)	Strata-Specific *P* Value*	Summary *P* Value†
Total	205/557 (37)	530/4185 (13)	4.02 (3.31-4.88)	<.001	
Sex					<.001
Male	112/275 (41)	268/1887 (14)	4.15 (3.16-5.45)	.002	
Female	93/282 (33)	262/2298 (11)	3.80 (2.87-5.03)	.002	
Age, y					<.001
18-34	38/120 (32)	181/1371 (13)	3.05 (2.01-4.62)	.004	
35-50	62/161 (39)	187/1230 (15)	3.49 (2.45-4.97)	.004	
51-65	65/176 (37)	123/932 (13)	3.85 (2.69-5.52)	.004	
>65	40/100 (40)	36/750 (5)	13.22 (7.84-22.27)	.004	

Abbreviations: CI, confidence interval; NPF, National Psoriasis Foundation; OR, odds ratio; UPI, Utah Psoriasis Initiative.
*Calculated as Pearson χ^2 test with the *P* value adjusted for multiple comparisons using the procedure of Finner.[36]
†Calculated as a summary *P* value from the Mantel-Haenszel χ^2 test.

1.48-6.04]; *P*=.001) and in the NPF survey (25% and 16%, for the obese and nonobese cohorts, respectively; OR, 1.72 [95% CI, 1.58-1.87]; *P*<.001).

Patients who received a diagnosis of psoriatic arthritis from a rheumatologist or who had signs and symptoms of psoriatic arthritis at examination were judged to have psoriatic arthritis. Of the subjects enrolled in the UPI, 26% had signs and symptoms that were most compatible with a diagnosis of psoriatic arthritis. This was very similar to the NPF patient-membership survey, where 31% of respondents complained of arthritis. To determine whether obesity plays a role in the onset of psoriatic arthritis, psoriasis patients with obesity in early life were compared with psoriasis patients without obesity in early life relative to the presence of psoriatic arthritis. Obesity at 18 years of age (OR, 0.64 [95% CI, 0.23-1.76]; *P*=.381) and at enrollment (OR, 1.18 [95% CI, 0.78-1.77]; *P*=.41) did not increase the likelihood of arthritis within the UPI population. Again, obesity appears to be a consequence of and not a contributor to onset of this variant of psoriasis.

To determine whether obesity has an impact on the response to therapy in patients with plaque psoriasis, we compared the response and tolerance to systemic therapy in obese and nonobese patients in the UPI population. With respect to the clearing of psoriasis, no statistically significant difference in response to methotrexate, psoralen–UV-A, and topical corticosteroids was noted in obese vs nonobese groups. With respect to tolerating these therapies, no difference was noted in the obese and nonobese groups. However, when those treated with methotrexate underwent evaluation, we found that obese patients were more likely than nonobese patients to report that it was "no longer effective" (13% and 3%, respectively; OR, 5.21 [95% CI, 1.36-19.99]; *P*=.008).

Compared with the Utah population, there was a higher prevalence of smoking in the UPI population with psoriasis (37% vs 13%; *P*<.001) (**Table 3**). There was also a higher prevalence of smoking in the UPI population with psoriasis compared with the nonpsoriatic population attending dermatology clinics (37% vs 25%; *P*<.001). In addition, there was a higher prevalence of smokers in the obese population of psoriasis patients at enrollment (**Table 4**). In contrast to most enrollees in the UPI, the cohort that smoked at enrollment did not have the strong family history of psoriasis (OR, 0.79 [95% CI, 0.55-1.12]; *P*=.19). We also observed no relationship between a family history of psoriasis and the occurrence of obesity and smoking. Smoking is more prevalent in obese psoriasis patients compared with obese individuals who smoke in the Utah population (35% vs 9%; *P*<.001) (Table 4). When we questioned psoriasis patients who smoke about smoking and the onset of their psoriasis, 78% indicated that they began smoking before the onset of psoriasis, whereas 22% began smoking after the onset of psoriasis. We also learned that the smokers were more likely to engage in binge drinking of alcohol (defined by the BRFSS as drinking ≥5 drinks of any type of alcohol on a given day, ≥1 time in the preceding month); ie, 33% of the smokers compared with 12% of the nonsmokers had 5 or more drinks on a given day (OR, 3.71 [95% CI, 2.04-6.72]; *P*=.001). We conclude that smoking precedes the onset of psoriasis, in contrast to obesity; furthermore, subjects who smoke have a later onset of disease and a decreased probability of a family history of disease.

COMMENT

Relative to the Utah population, our cohort psoriasis patients had a higher prevalence of obesity. Our study supports previous observations on obesity in psoriasis.[37-41] One report cited that patients with psoriasis are 15% above the average body weight.[37] Another study reported that in a population of 159 200, women with psoriasis were more likely to be obese.[42] In our study, there was a greater prevalence of obesity in psoriasis patients relative to the general Utah population across all age groups. Our control survey of a nonpsoriatic population of more than 500 subjects showed that these subjects had a lower BMI than the general Utah population, validating the conclusion that our pso-

Table 4. Comparison of Prevalence of Obese Smokers in the UPI and Utah Populations

Stratification Variable	No. of Obese Subjects Who Smoke/Population (%)		UPI vs Utah Populations		
	UPI	Utah	Prevalence OR (95% CI)	Strata-Specific *P* Value*	Summary *P* Value†
Total	67/192 (35)	63/735 (9)	5.72 (3.86-8.47)	<.001	
Sex					<.001
Male	32/90 (36)	34/387 (9)	5.73 (3.28-9.99)	.002	
Female	35/100 (35)	29/348 (8)	5.92 (3.38-10.37)	.002	
Age, y					<.001
18-34	6/26 (23)	14/139 (10)	2.68 (0.92-7.78)	.094	
35-50	25/56 (45)	27/235 (11)	6.21 (3.20-12.05)	.004	
51-65	23/72 (32)	20/233 (9)	4.99 (2.54-9.82)	.004	
>65	13/38 (34)	2/127 (2)	32.50 (6.90-153.03)	.004	

Abbreviations: CI, confidence interval; NPF, National Psoriasis Foundation; OR, odds ratio; UPI, Utah Psoriasis Initiative.
*Calculated as Pearson χ^2 test with the *P* value adjusted for multiple comparisons using the procedure of Finner.[36]
†Calculated as a summary *P* value from the Mantel-Haenszel χ^2 test.

riasis patients had a significantly increased BMI. We confirm that there is an obesity-psoriasis association in patients who attend our clinic and perhaps other clinics that focus on the care and management of psoriasis, giving the perception that psoriasis patients are overweight.

Based on the body image reported by patients at 18 years of age, at onset of psoriasis, and at enrollment, body weight did not increase until after the onset of psoriasis. We conclude that there is no association between obesity in early life and age at onset of psoriasis, ie, obesity does not appear to be one of the factors known to trigger the onset of disease. Conversely, psoriasis appears to have a causal effect on obesity, likely because of the profound adverse effect of psoriasis on an individual's physical, social, and mental well-being.[43-46] Our study supports the probability that psoriasis plays a role in unhealthy behavior, eg, overeating, inactivity, and smoking. Although psoriasis is generally regarded as a non–life-threatening condition, it appears to be strongly correlated with diseases that have important public health implications, eg, diabetes and hypertension. Few would doubt the benefit of weight loss on the comorbid conditions associated with the overweight state. Although it seems likely, it is not known whether weight loss would alter the clinical features and/or the response to treatment for psoriasis patients. Prospective studies are needed to determine whether weight control would positively modify this disease.

A concern is that a large number of patients in the UPI are smokers. We observed an increase in smoking among psoriasis patients compared with the general population, while controlling for age and sex. Our data confirm the association between cigarette smoking and psoriasis recently reported in a European population.[27] Naldi et al[27] have explored the impact of smoking on psoriasis in an Italian population. In a multicenter case-control study, they reported an increased risk of psoriasis among smokers and ex-smokers compared with subjects who never smoked.[28] It has been hypothesized that excess mortality is also related to alcohol intake coupled with smoking among psoriasis patients.[29] Poikolainen et al[29] found that alcohol intake and smoking are major causes of excess mortality in psoriasis patients. In a 10-year prospective study involving psoriasis patients, Stern and Lange[47] noted no increase in cardiovascular mortality, which is contrary to reports of cerebrovascular and cardiovascular mortality being elevated in psoriasis.[48] Our data and most of the literature cause us to conclude that the prevalence of obesity, smoking, smoking by the obese population, and binge drinking in the smoking population within the UPI makes psoriasis a greater public health problem than previously thought. Studies to determine the validity of this assertion are warranted.

A limitation inherent in this study design is that we do not have prospective data to test for a causal relationship between obesity and psoriasis. Another limitation is the probability of selection bias inherent in the UPI and NPF populations. This bias is suggested, as obesity is more prevalent in psoriasis patients referred to our tertiary care center, which focuses on the care and management of psoriasis, than it was in psoriasis patients responding to the NPF survey and in our nonpsoriatic population.

Self-reported weight and height were used for analysis in the UPI, 1998 NPF patient member survey, BRFSS, and survey of the nonpsoriatic population. A high correlation between continuous measures of self-reported and measured weight and height has led investigators to infer that self-reporting of these variables is appropriate in epidemiological studies. One study reviewed data from 7455 adult participants of the Lipid Research Clinics Family Study (1975-1978) for whom self-reported and measured height and weight were available. The sensitivity of the obese category when defined with self-reported weight and height was 74%, and the specificity was 99%.[49] In addition, the Hypertension Detection and Follow-up Program evaluated the accuracy of self-reported height and weight. In that study, weight was understated by 1.6% by men and 3.1% by women, whereas height was overstated by 1.3% by men and 0.6% by women.[50] As overweight individuals tend to underreport and those who are underweight or of short stature tend to overreport, studies investigating associations of disease with height

and weight using self-reported measures will underestimate effects.[51] The BRFSS data and self-reported measurements have been assessed for reliability and validity. Specifically, measures determined to be of high reliability and validity include current smoking, blood pressure screening, height and weight (and therefore BMI), and demographic characteristics.[52] However, if patients in the UPI underreported weight, height, and smoking, then our estimates of the effects would be biased toward the null, so the true effects would be even greater.

Within the UPI, we have found that inverse psoriasis is more common in the obese population. Indeed, morbidly obese patients in the UPI trend toward inverse psoriasis. Our findings harmonize with the NPF patient survey, where inverse psoriasis was self-reported in 5% of nonobese, 11% of obese, and 13% of morbidly obese patients. We suspect that the milieu of intertriginous skin is favorable for the development of psoriasis. Regardless of mechanism, it is important to appreciate that psoriasis in the body folds of morbidly obese patients has the requisite features of inverse psoriasis that can be indistinguishable from intertrigo. For appropriate management, what appears to be psoriasis in body folds is likely not just candidiasis or intertigo. Many of our morbidly obese subjects do not have evidence of intertriginous psoriasis.

Our findings provide a significant contribution because of confirmatory data and new insights into psoriasis. We have confirmed the fact that an increased BMI is seen in psoriasis patients who have extensive disease. We have demonstrated that tobacco use is increased among psoriasis patients and that binge drinking is more likely among smokers with psoriasis. Our data have implications for the future burden of morbidity, mortality, and health care costs associated with psoriasis patients. Most concerning is the fact that the leading causes of death in the United States are tobacco use (435 000 deaths in 2000) and poor diet with physical inactivity (400 000 deaths in 2000).[53] It seems certain that the cost of providing care for psoriasis—when coupled with the increased frequency of obesity and smoking in patients attending clinics such as ours—will continue to increase. An effort to control obesity and smoking in psoriasis patients and an increased appreciation of the effects of these comorbidities are clearly needed.

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Correspondence: Gerald G. Krueger, MD, Department of Dermatology, University of Utah School of Medicine, Health Sciences Center, 30 North 1900 East, Room 4B454, Salt Lake City, UT 84132-2409 (krueger@derm.med.utah.edu).
Author Contributions: *Study concept and design:* Hansen and Krueger. *Acquisition of data:* Herron, Hinckley, Hoffman, Hansen, and Krueger. *Analysis and interpretation of data:* Herron, Papenfuss, Callis, and Krueger. *Drafting of the manuscript:* Krueger. *Critical revision of the manuscript for important intellectual content*: Herron, Hinckley, Hoffman, Hansen, Callis, and Krueger. *Statistical analysis:* Herron, Papenfuss, and Callis. *Obtained funding:* Krueger. *Administrative, technical, and material support:* Krueger. *Study supervision:* Callis and Krueger.
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Announcement

Calendar of Events: A New Web Feature

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