The association between weight-adjusted waist index and psoriasis in adults: results from NHANES 2009-2014

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Research Article

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Abstract

Background

Evidence has indicated that the correlation between obesity and psoriasis is likely multifactorial in nature. Previously, no reports have been made regarding the correlation between weight-adjusted waist index (WWI) and psoriasis.

Methods

In this cross-sectional research, we examined a cohort of adult participants who provided comprehensive data related to WWI and psoriasis, gathered during the 2009–2014 National Health and Nutrition Examination Survey (NHANES). The calculation for WWI involved dividing waist circumference (WC) by the square root of body weight. We explored the association between WWI and psoriasis through multivariate linear regression modeling and subgroup analyses. The smoothing curve fitting was also applied.

Results

The study included 15,920 participants aged over 19 years, of whom 438 had a history of psoriasis. It revealed a notable positive correlation between WWI and psoriasis (OR = 1.226; 95% CI, 1.071–1.404, P < 0.05). The quartile with the highest WWI value (OR = 1.551; 95% CI, 1.127–2.135, P < 0.05) had a significantly increased risk of developing psoriasis by 55.1% compared with the quartile with the lowest WWI value. A positive nonlinear connection between WWI and psoriasis was observed in the smoothed curve fitting results.

Conclusion

Among U.S. adults, levels of WWI are positively associated with increased odds of developing psoriasis. We propose that adopting obesity management strategies based on the WWI could be beneficial in lowering the risk of psoriasis.

Introduction

Psoriasis, most notably affecting the skin and joints, is a systemic inflammatory disease characterized by adaptive and innate immune alterations[1]. The pathogenesis of this condition is significantly influenced by tumor necrosis factor alpha, T cells, and dendritic cells.[2]. The global burden of psoriasis is a key WHO research mandate, affecting more than 60 million adults and children worldwide[3]. In psoriasis, circulating pro-inflammatory mediators lead to a widespread inflammatory response, often accompanied by comorbidities such as cardiovascular disease (CVD) and metabolic syndrome [4].
Obesity, a serious condition worldwide, is generally defined as excess body fat that is detrimental to health. The Global Burden of Disease organization reports that global obesity affected a significant population with 107.7 million children and 603.7 million adults in 2015[5]. The most commonly reported subtypes of obesity include metabolically healthy obesity, metabolically abnormal obesity, normal weight obesity, and muscle-sparing obesity[6]. Multiple cardiometabolic imaging studies indicate that normal weight or overweight individuals are often burdened with ectopic fat accumulation in normal lean tissues (liver, heart, skeletal muscle, and other areas).

Evidence is mounting that weight gain and obesity were risk factors for the onset of psoriasis[8]. The sequence of causation between psoriasis and obesity remains elusive. The notable factors contributing to this complex relationship may include pronounced social isolation, unhealthy dietary patterns, depressive tendencies, increased alcohol intake, and reduced physical activity among individuals with psoriasis[9]. Higher body mass index (BMI) is linked to an increased likelihood of psoriasis occurrence[10–12]. Furthermore, objective measures of psoriasis severity demonstrate a positive dose-dependent relationship with obesity[13, 14]. There is an association between obesity and low efficacy of anti-psoriasis medications[15]. Indeed, reduction of body mass through dietary modifications and physical activity contributes positively to the severity and incidence of psoriasis[16].

Park et al. introduced a novel ergometric method, the weight-adjusted waist index (WWI), as a means of overcoming the limitations of BMI or waist circumference (WC)[17]. High WWI values appear to correlate with adverse body composition outcomes, signifying high fat mass, low muscle mass, and low bone mass [18]. With the potential to be a comprehensive and validated index of obesity, the WWI has merit in predicting the risk of certain diseases (e.g. cardiovascular-metabolic disease, diabetes mellitus, osteoarthritis, and erectile dysfunction)[19–22]. And yet, there are no studies examining the effects of WWI on people with psoriasis.

Thereby, this study’s intention was to examine the correlation between WWI and psoriasis among US adults through the 2009–2014 NHANES.

### Methods

#### Survey population

The National Center for Health Statistics (NCHS) undertakes a broad survey—the National Health and Nutrition Examination Survey (NHANES), which is updated every two years to assess the nutritional and healthy status of the U.S. ambulatory population. The study adopted a stratified multi-stage probability strategy and recruited a relatively large number of participants. The Ethics Review Board of NCHS endorsed the work and informed consent was signed by all participants. Baseline information for this research was gathered from the 2009–2014 NHANES data. 30,468 participants took part in this round of surveys, with a final total of 15,920 subjects enrolled in the study after excluding individuals with a
history of psoriasis, missing statistical information on the WWI, and individuals younger than or equal to 19 years of age. The detailed participant inclusion process for the study is depicted in Fig. 1.

**Evaluation of weight-adjusted-waist index**

The WWI serves as a metric for assessing obesity. Health technicians trained in body measurements measured waist circumference and weight at a mobile examination center.

The following equation was used to identify the weight-adjusted waist index: 

\[
\frac{\text{waist circumference cm}}{\sqrt{\text{weight kg}}}
\]

In our analysis, we regarded WWI as a continuous variable and grouped participants according to WWI quartiles for subsequent investigation. WWI served as an exposure indicator in our research.

**Covariates of interest**

We conducted subgroup analyses employing stratified multivariate logistic regression models, categorizing factors including sex (male/female), age (tertile), race (Mexican American/other Hispanic/non-Hispanic white/non-Hispanic black/other race), educational attainment (less than high school/high school/high school or more/other), smoking (yes/no), alcohol consumption (yes/no), body mass index (< 25 kg/m<sup>2</sup>, normal weight/25-29.9 kg/m<sup>2</sup>, overweight/≥ 30 kg/m<sup>2</sup> obese), and the following five categories of comorbidities (Yes/No): diabetes, congestive heart failure, coronary heart disease, stroke, and cancer. Furthermore, these stratification factors were considered as potential effect modifiers, and we incorporated an interaction term via likelihood ratio test to assess variations in associations among different subgroups. Absent values for continuous variables were entered as medians, and missing values for categorical variables were entered as patterns of existing cases. Detailed measurement protocols for these variables can be accessed on the website [www.cdc.gov/nchs/nhanes/](http://www.cdc.gov/nchs/nhanes/).

**Statistical analysis**

NHANES, designed to yield data representative of the noninstitutionalized civilian population in the United States, guided our statistical analysis. We adhered to CDC guidelines, incorporating NHANES sampling weights and addressing the complexities of multistage cluster surveys. Weighted multiple linear regression analyses were used to explore the linear association between WWI and psoriasis incidence. Similarly, we utilized smoothed curve fitting to detect nonlinear effects of WWI on psoriasis. Our study included three models: Model 1, without covariate adjustments; Model 2, adjusted for sex, race, and age; and Model 3, adjusted for all covariates. Subgroup analyses were also conducted. Significance was determined at \( p < 0.05 \). For statistical analysis, we utilized R ([http://www.r-project.org](http://www.r-project.org)) and EmpowerStats ([http://www.empowerstats.com](http://www.empowerstats.com)).

**Results**

**Baseline characteristics**
This study consisted of 15,920 participants. Table 1 shows data on the clinical and demographic profile of the study population with or without psoriasis. The mean age of patients with psoriasis was $(55.18 \pm 16.43)$ years, 49.32% were male, and the mean WWI was $(11.207 \pm 0.843)$. On the other hand, the mean age of patients without psoriasis was $(48.59 \pm 17.51)$ years, 48.78% were males and the mean WWI was $(11.029 \pm 0.841)$. It can be seen that WWI differed significantly between the two groups ($p < 0.05$).

Compared to the group without a history of psoriasis, the group with a history of psoriasis was significantly older, had a higher percentage of non-Hispanic whites and those with a high school education or higher, a higher percentage of comorbidities including stroke, coronary artery disease, diabetes mellitus, congestive heart failure, and cancer, a higher prevalence of cigarette smoking and alcohol abuse, and higher levels of BMI. However, there were no significant differences in WWI with and without psoriasis by gender, educational level, and presence of stroke (all $p > 0.05$).

Table 1. Baseline Characteristics of Patients With and Without Psoriasis
<table>
<thead>
<tr>
<th>Variable</th>
<th>History of Psoriasis (n = 438 [2.8%])</th>
<th>No History of Psoriasis (n = 15482 [97.2%])</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>52.183 ± 16.426</td>
<td>48.586 ± 17.511</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gender (%)</td>
<td></td>
<td></td>
<td>0.825</td>
</tr>
<tr>
<td>Men</td>
<td>49.315</td>
<td>48.779</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>50.685</td>
<td>51.221</td>
<td></td>
</tr>
<tr>
<td>Race (%)</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mexican American</td>
<td>8.904</td>
<td>14.346</td>
<td></td>
</tr>
<tr>
<td>Other Hispanic</td>
<td>9.817</td>
<td>9.850</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic White</td>
<td>57.991</td>
<td>42.404</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic Black</td>
<td>11.187</td>
<td>21.360</td>
<td></td>
</tr>
<tr>
<td>Other Races</td>
<td>12.100</td>
<td>12.040</td>
<td></td>
</tr>
<tr>
<td>Education level (%)</td>
<td></td>
<td></td>
<td>0.492</td>
</tr>
<tr>
<td>Less than high school</td>
<td>21.461</td>
<td>24.338</td>
<td></td>
</tr>
<tr>
<td>High school or GED</td>
<td>22.831</td>
<td>22.058</td>
<td></td>
</tr>
<tr>
<td>Above high school</td>
<td>55.708</td>
<td>53.501</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>0.000</td>
<td>0.103</td>
<td></td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>57.991</td>
<td>43.815</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Alcohol use (%)</td>
<td>24.586</td>
<td>17.142</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.782 ± 6.789</td>
<td>28.904 ± 6.776</td>
<td>0.003</td>
</tr>
<tr>
<td>Comorbidities (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>16.895</td>
<td>11.685</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Congestive heart failure.</td>
<td>6.164</td>
<td>2.694</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>7.763</td>
<td>3.611</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke</td>
<td>3.269</td>
<td>3.881</td>
<td>0.478</td>
</tr>
<tr>
<td>Cancer</td>
<td>15.753</td>
<td>9.005</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WWI</td>
<td>11.207 ± 0.843</td>
<td>11.029 ± 0.841</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Mean ± SD for continuous variables: the p-value was calculated by weighted linear regression model. % for categorical variables: the p-value was calculated by a weighted chi-square test. BMI, body mass index;
WWI, weight-adjusted-waist index.

**Association between WWI and psoriasis.**

As depicted in Table 2, a favorable correlation between WWI and the risk of psoriasis was evident in this study. This connection remained consistent even in the unadjusted model 1. (OR=1.285; 95% CI, 1.148-1.439, p<0.05) and the fine-tuned model 2 (OR=1.226; 95% CI, 1.071-1.404, p<0.05). In the fully adjusted model (Model 3), which accounted for all confounders, the association between WWI and psoriasis became nonsignificant (OR=1.011; 95% CI, 0.840-1.216, p>0.05). In our sensitivity analysis, we converted WWI from a continuous variable to a categorical variable (quartiles). In Model 2, participants in the quartile with the highest WWI value (OR=1.551; 95% CI, 1.127-2.135, p<0.05) had a significantly increased risk of developing psoriasis by 55.1% compared to the WWI value in the smallest quartile. Likewise, individuals in the middle quartile exhibited a heightened risk of psoriasis, although this association did not reach statistical significance.

Table 2. The association between WWI and psoriasis.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR(95% CI) P value</td>
<td>OR(95% CI) P value</td>
<td>OR(95% CI) P value</td>
</tr>
<tr>
<td>WWI</td>
<td>1.285 (1.148, 1.439)</td>
<td>1.226 (1.071, 1.404)</td>
<td>1.011 (0.840, 1.216)</td>
</tr>
<tr>
<td></td>
<td>0.00001</td>
<td>0.00310</td>
<td>0.91141</td>
</tr>
<tr>
<td>WWI quartiles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile 1</td>
<td>Reference</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>Quartile 2</td>
<td>1.343 (1.003, 1.797)</td>
<td>1.271 (0.942, 1.714)</td>
<td>1.201 (0.860, 1.679)</td>
</tr>
<tr>
<td></td>
<td>0.04758</td>
<td>0.11647</td>
<td>0.28243</td>
</tr>
<tr>
<td>Quartile 3</td>
<td>1.343 (1.003, 1.797)</td>
<td>1.239 (0.906, 1.694)</td>
<td>1.028 (0.710, 1.487)</td>
</tr>
<tr>
<td></td>
<td>0.04758</td>
<td>0.17992</td>
<td>0.88519</td>
</tr>
<tr>
<td>Quartile 4</td>
<td>1.768 (1.340, 2.332)</td>
<td>1.551 (1.127, 2.135)</td>
<td>1.061 (0.701, 1.606)</td>
</tr>
<tr>
<td></td>
<td>0.00002</td>
<td>0.00711</td>
<td>0.77970</td>
</tr>
<tr>
<td>P for trend</td>
<td>0.00008</td>
<td>0.01046</td>
<td>0.98707</td>
</tr>
</tbody>
</table>

Model 1, no covariates were adjusted. Model 2, age, sex, and race were adjusted. Model 3, age, sex, race, education level, smoking status, drinking status, BMI, diabetes, congestive heart failure, coronary artery disease, stroke and cancer were adjusted. 95% CI, 95% confidence interval; OR, odds ratio; WWI, weight-adjusted-waist index. p < 0.05 was considered statistically significant.

As shown in Table 3, further subgroup analyses and interaction tests showed that the association between WWI and psoriasis was not consistent in the overall population. In subgroup analyses stratified by sex, there was an independent and significant positive association between WWI and psoriasis in men.
(OR = 1.031; 95% CI, 1.002-1.061, p<0.05), but the association was not statistically significant in women in all models. In the age (tertile) subgroups, only the oldest group, WII, was positively associated with psoriasis. When subgroups were examined by BMI, a substantial positive association between WWI and psoriasis was detected in both the overweight group and obese group within the unadjusted model. WWI remained positively associated with psoriasis risk in smokers, non-drinkers, non-coronary heart disease and non-diabetic populations.

Table 3. Subgroup analysis for the association between WWI and psoriasis.
<table>
<thead>
<tr>
<th>WWI</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI) P value</td>
<td>OR (95% CI) P value</td>
<td>OR (95% CI) P value</td>
</tr>
<tr>
<td>gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>1.496 (1.262, 1.773) &lt;0.0001</td>
<td>1.355 (1.098, 1.671) 0.0046</td>
<td>1.165 (0.866, 1.568) 0.3131</td>
</tr>
<tr>
<td>Women</td>
<td>1.162 (0.990, 1.363) 0.0655</td>
<td>1.136 (0.953, 1.355) 0.1546</td>
<td>0.925 (0.726, 1.180) 0.5308</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tertile 1</td>
<td>1.043 (0.821, 1.325) 0.7314</td>
<td>1.118 (0.860, 1.454) 0.4052</td>
<td>1.003 (0.680, 1.480) 0.9866</td>
</tr>
<tr>
<td>Tertile 2</td>
<td>1.139 (0.918, 1.413) 0.2384</td>
<td>1.114 (0.892, 1.392) 0.3401</td>
<td>1.148 (0.842, 1.566) 0.3820</td>
</tr>
<tr>
<td>Tertile 3</td>
<td>1.410 (1.149, 1.731) 0.0010</td>
<td>1.429 (1.151, 1.775) 0.0012</td>
<td>1.007 (0.757, 1.340) 0.9606</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25</td>
<td>1.213 (0.949, 1.551) 0.1225</td>
<td>1.007 (0.722, 1.404) 0.9686</td>
<td>0.993 (0.671, 1.469) 0.9702</td>
</tr>
<tr>
<td>&gt;=25, &lt;30</td>
<td>1.264 (1.020, 1.566) 0.0323</td>
<td>1.210 (0.915, 1.598) 0.1808</td>
<td>1.268 (0.922, 1.744) 0.1447</td>
</tr>
<tr>
<td>&gt;=30</td>
<td>1.243 (1.015, 1.523) 0.0353</td>
<td>1.118 (0.887, 1.408) 0.3441</td>
<td>0.934 (0.717, 1.217) 0.6137</td>
</tr>
<tr>
<td>Smoke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>YES</td>
<td>1.317 (1.132, 1.532) 0.0004</td>
<td>1.205 (1.009, 1.439) 0.0398</td>
<td>0.953 (0.752, 1.208) 0.6896</td>
</tr>
<tr>
<td>NO</td>
<td>1.212 (1.021, 1.438) 0.0276</td>
<td>1.196 (0.979, 1.461) 0.0802</td>
<td>1.082 (0.806, 1.453) 0.6004</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>YES</td>
<td>1.361 (1.045, 1.773) 0.0224</td>
<td>1.139 (0.835, 1.554) 0.4104</td>
<td>0.924 (0.624, 1.368) 0.6921</td>
</tr>
<tr>
<td>NO</td>
<td>1.268 (1.098, 1.463) 0.0012</td>
<td>1.203 (1.015, 1.427) 0.0329</td>
<td>1.028 (0.834, 1.268) 0.7953</td>
</tr>
<tr>
<td>Coranary heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>YES</td>
<td>1.002 (0.631, 1.590) 0.9943</td>
<td>0.965 (0.578, 1.611) 0.8922</td>
<td>0.917 (0.536, 1.569) 0.7521</td>
</tr>
<tr>
<td>NO</td>
<td>1.269 (1.129, 1.428) &lt;0.0001</td>
<td>1.237 (1.075, 1.423) 0.0030</td>
<td>1.024 (0.846, 1.240) 0.8075</td>
</tr>
</tbody>
</table>
Model 1: no covariates were adjusted. Model 2: age, gender, and race were adjusted. Model 3: age, sex, race, education level, smoking status, drinking status, BMI, diabetes, congestive heart failure, coronary artery disease, stroke and cancer were adjusted.

In the subgroup analysis stratified by gender and age, the model is not adjusted for sex and age, respectively.

95% CI, 95% confidence interval; OR, odds ratio; WWI, weight-adjusted-waist index. p < 0.05 was considered statistically significant.

Figure 2 shows the smoothed curve fit with adjustments based on the covariates (gender, age, and race). The results show a positive nonlinear relationship across the entire range of the WWI.

**Discussion**

This finding investigated the correlation between WWI and psoriasis in non-hospitalized Americans. In our cross-sectional analysis of 15920 participants, we found that individuals with higher WWI exhibited an elevated likelihood of psoriasis. Subgroup analyses showed that none of the stratification variables had a discernible impact on the consistency of the relationship between WWI and psoriasis, and the positive association persisted. As far as we know, this study is the first attempt to focus on the relationship between WWI and psoriasis.

The modulation between psoriasis and obesity involves multiple factors and mechanisms. A study uncovered a noteworthy correlation between psoriasis and obesity in twins, hinting at a shared genetic underpinning for both conditions [23]. To illustrate certain variations in the Fat mass and obesity associated (FTO) gene, responsible for regulating body fat mass, have been linked to a heightened risk of obesity and an association with higher Psoriasis Area and Severity Index (PASI) scores in psoriasis patients [24, 25]. Since adipose tissue is a vital endocrine organ that releases soluble factors influencing inflammation and immunity, the expansion of adipose tissue and its release of proinflammatory agents can potentially exacerbate psoriasis [26]. Ikeda et al. discovered a synergistic exacerbation of inflammation in psoriatic skin due to obesity and dyslipidemia [27]. Moreover, obesity has a notable impact on the composition and functionality of inflammatory cells within the skin. Obese patients with psoriasis often exhibit elevated levels of resistin and leptin [28, 29]. They also observed that metabolic disorders related to leptin, inflammatory factors and palmitic acid contribute to increased activation of epidermal keratin-forming cells [27]. Christ et al. showed in a study that Western diet induced myeloid...
progenitors to undergo sustained transcriptomic and epigenomic reprogramming, leading to proliferation and an innate immune response increases [30]. The immunopathogenesis of psoriasis depends on the key role of the IL-17/IL-23 axis. Obese women appear to have higher blood levels of IL-17 and IL-23 compared to slim women [31]. Emerging data suggest that IL-17 and subpopulations of IL-17-producing cells (e.g., Th17) are determinant in the induction and development of obesity [32]. Nakamizo et al. detailed the buildup of γδ T cells that produce interleukin IL-17a in psoriatic lesions, a response triggered by the consumption of a high-fat diet, which led to worsening of psoriatic dermatitis [33]. An independent study revealed that a prolonged high-density lipoprotein (HDL) diet, continuing for more than nine months, triggered the aggregation of specific CD11c+ macrophages in the skin. This aggregation was facilitated through a mechanism dependent on epidermal fatty acid-binding protein (E-FABP) [34]. Adipose tissue triggers the release of IL-6, an important mediator influencing the polarization of CD4+ T cells to Th17 cells [35]. In addition, obesity affects the microbiome [36, 37]. Recent studies have shown that the microbiome - i.e., all microorganisms living on the internal and external surfaces of the human body - has a strong influence on human autoimmune diseases [38].

Obesity has been commonly assessed in the past with indices such as BMI and WC, which have been shown to be associated with psoriasis. In a Norwegian prospective study of 369 patients with psoriasis, the relative risks of BMI, WC, and waist-to-hip ratio were 1.22 (95% CI 1.11–1.34), 1.26 (95% CI 1.15–1.39), and 1.18 (95% CI 1.07–1.31) respectively, and the relative risk for those who gained 10 kilograms or more in weight compared with those who were weight stable was 1.72 (95% CI 1.15–2.58) [39]. In multivariate analyses, each incremental unit increase in BMI was associated with a statistically significant 9% increase in the risk of developing psoriasis and a 7% increase in the risk of an increased PASI. Compared with people of normal weight, obesity leads to a two-fold increased risk of developing psoriasis. In an analysis of the Taiwan National Health Interview Survey and the National Health Insurance database, an adjusted HR of 1.34 (95% CI 1.05–1.71) and an adjusted HR of 2.70 (95% CI 1.95–3.72) were observed for participants with a BMI of 25.0–29.0 relative to those with a BMI of 18.5–22.9 [41]. A notable correlation was observed among psoriasis patients between their BMI and WC before undergoing phototherapy, and their PASI scores [42]. The results of a 2013 study showed that weight loss through a low-energy diet (800–100 kcal/day) moderately reduces PASI and improves patients’ quality of life [43]. The WWI, the new obesity index, primarily reflects the actual situation of centrally located obesity as a result of weight adjustment. While the centrally obese group had a predominant accumulation of visceral fat, visceral fat is associated with adipocyte hypertrophy and the production of pro-inflammatory immune mediators, including adipokines (e.g. resistin and leptin) as well as cytokines (e.g. IL-6 and tumor necrosis factor-α) [44]. The strengths of the WWI in assessing psoriasis are thus demonstrated.

WWI proved to be correlated with various diseases. Initially reported, an observed non-linear positive correlation emerged between WWI levels and the risk of CVD and all-cause mortality [17]. It is worth noting that individuals with psoriasis face an elevated risk of CVD, a prevalent contributor to morbidity and mortality in psoriasis cases, and a recognized cardiovascular risk factor [45, 46, 47]. High serum IL-17A levels are linked to greater mortality from coronary heart disease [48]. Moreover, an intriguing finding indicated a non-linear positive relationship between WWI and hypertension [49]. Significantly, the
angiotensin-converting enzyme (ACE) gene, a pivotal component of the renin-angiotensin system, has been extensively associated with psoriasis[50–55]. Endothelin-1 might contribute to the onset of hypertension among individuals with psoriasis.[56]. Other evidence suggests that higher WWI is associated with a higher prevalence of stroke [57]. Individuals afflicted with psoriasis face an elevated susceptibility to cerebrovascular diseases(stroke), which is related to the severity of the psoriasis condition [58–61]. Atherosclerosis is the main pathologic change that precedes stroke. Notably, patients with psoriasis exhibit greater arterial stiffness than healthy controls [62, 63]. Both atherosclerosis and coronary artery disease are chronic inflammatory conditions characterized by the involvement of immune cells, including Th1 and Th17 cells, along with the infiltration of inflammatory factors at all stages of the diseases[64]. Meanwhile, research has indicated a positive correlation between WWI and the prevalence of non-alcoholic fatty liver disease (NAFLD) as well as liver fibrosis in adults in the U.S.[65]. A comprehensive systematic review and meta-analysis conducted in 2015 revealed that individuals with psoriasis face twice the risk of developing NAFLD compared to controls [66]. The inflammatory association between psoriasis and NAFLD is referred to as the "liver-skin axis" [67]. IL-17 induces hepatic stellate cell activation, which in turn produces collagen [68]. Thus, this action of IL-17 accelerates the transition from basic hepatic steatosis to steatohepatitis [67, 68]. In summary ,it is reasonable to hypothesize that WWI may be positively associated with the incidence of psoriasis.

This research possesses multiple notable strengths. First, the NHANES data contains a large sample that is representative of the U.S. population, making our results reliable. Secondly, we conducted rigorous adjustments for numerous potential confounding variables in our multivariate logistic regression analyses. Additionally, we carried out subgroup analyses to enhance the precision of the associations between risk factors and the prevalence of psoriasis. Nonetheless, it's equally crucial to acknowledge certain constraints of this study.Firstly, since this was a cross-sectional study, it was not possible to determine temporality and therefore causality. Next, the reliance on self-reported history of psoriasis may have reduced the accuracy of prevalence estimates. In addition, the sample in this analysis exclusively comprised participants from the United States, which did not consider variations in both prevalence and risk factors among different countries and ethnic groups. Therefore, it may not serve as an adequate representation of global demographics.

**Conclusion**

The study findings revealed a pronounced link between elevated WWI levels and an augmented risk of psoriasis in the U.S. adult population. Further studies are essential to validate the outcomes of this research.

**Abbreviations**

WWI Weight-adjusted waist index

NHANES National health and nutrition examination survey
WC Waist circumference
BMI Body mass index
NCHS National center for health statistics
CVD Cardiovascular disease
FTO Fat mass and obesity associated
PASI Psoriasis Area and Severity Index
HDL High-density lipoprotein
E-FABP Epidermal fatty acid-binding protein
ACE The angiotensin-converting enzyme
NAFLD Non-alcoholic fatty liver disease

Declarations

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Author Contributions.

All named authors contributed to the final paper as follows: Xiya Zhao contributed to the concept and design, statistical analysis, and drafting of the manuscript. Dr. Junqin Li contributed to the statistical analysis and drafting of the manuscript. Dr. Xinhua Li contributed to the concept and design and resolution of any discrepancies in study eligibility criteria.

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Data availability statement

Publicly available datasets were analyzed in this study. This data can be found here: www.cdc.gov/nchs/nhanes/.

Consent for publication

Not applicable.
Competing interests

All authors declare that they have no competing financial interests.

Ethics statement

This study was reviewed and approved by NCHS Ethics Review Board. The patients/participants provided their written informed consent to participate in this study. This study conformed with the Helsinki Declaration of 1964, as revised in 2013, concerning human rights.

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Figures
Figure 1

Flow chart of participants selection. NHANES, National Health and Nutrition Examination Survey.
Figure 2

The nonlinear associations between WWI and psoriasis. The solid red line represents the smooth curve fit between variables. Blue bands represent the 95% of confidence interval from the fit.