

RESEARCH

Open Access



Systemic inflammation response index mediates the association between relative fat mass and psoriasis risk: a population-based study

Xinyi Shao^{1†}, Jun Yu^{2†}, Qian Liu¹, Yidian Fu³, Aijun Chen^{1*}, Genlong Bai^{1*} and Jingbo Zhang^{1*}

Abstract

Background Psoriasis, a prevalent autoimmune skin condition, considerably impairs the quality of life of those who are affected by it. Several studies have demonstrated that obesity significantly contributes to both the onset and progression of psoriasis. Relative fat mass (RFM), a novel obesity index, provides a more precise measure by incorporating both height and waist circumference (WC). The aim of this study was to investigate the association between RFM and psoriasis risk, taking into account the intermediary role played by the systemic inflammation response index (SIRI).

Methods The cross-sectional study assessed data from 8,479 adults who participated in the NHANES cycles from 2003 to 2006 and 2009 to 2014. To examine the association between RFM and psoriasis, both multivariate logistic regression model and restricted cubic spline (RCS) analyses were conducted. A mediation analysis was used to clarify the role of SIRI in the association between RFM and psoriasis.

Results Higher RFM was significantly associated with a 5% higher risk of developing psoriasis (odds ratio [OR] = 1.05, 95% confidence interval [CI]: 1.02–1.08), with RFM quartiles indicating a significant trend ($P_{\text{for trend}} < 0.05$). The SIRI demonstrated a significant mediating effect on the RFM-psoriasis relationship (mediation effect ratio = 5.02%).

Conclusion Elevated RFM are associated with an increased prevalence of psoriasis. RFM has the potential to be a beneficial anthropometric measure for more accurately predicting psoriasis risk.

Keywords Relative fat mass, Psoriasis, Cross-sectional study, NHANES, Systemic inflammation response index

[†]Xinyi Shao and Jun Yu contributed equally to this work.

*Correspondence:

Aijun Chen
chenaijun@hospital.cqmu.edu.cn
Genlong Bai
934147726@qq.com
Jingbo Zhang
49554556smael@gmail.com

¹ Department of Dermatology, The First Affiliated Hospital of Chongqing Medical University, Chongqing, China

² Department of Dermatology, The Second Affiliated Hospital of Chongqing Medical University, Chongqing, China

³ Graduate School of Hebei Medical University, Shijiazhuang 050017, Hebei, China

Background

Psoriasis is an inflammatory condition that affects the entire body and is primarily driven by immune system activity, distinguished by the presence of erythematous plaques, scaling, and pruritus. Epidemiological studies report a global prevalence of 0.09%–11.43%, with evidence suggesting a gradual increase over time [1, 2]. The etiology of psoriasis involves genetic susceptibility, environmental triggers, and immune dysregulation [2]. And excessive activation of the IL-23/IL-17 axis also plays a crucial role in the pathogenesis of psoriasis. IL-23 produced by dendritic cells and macrophages promotes the differentiation of Th17 cells, most notably



IL-17. This cytokine cascade fuels inflammation, sustaining chronic inflammation and epidermal hyperplasia [3, 4]. In present, psoriasis has been considered a systemic inflammatory disorder that affects multiple organs and is associated with various complications, including metabolic disorders, cardiovascular disease (CVD), and mental health status [5, 6]. This condition can give rise to a significant decline in life quality and imposes substantial societal costs. Given its significant impact on global health, it is essential to comprehend the fundamental factors and mechanisms involved to create effective intervention and management strategies.

The occurrence of metabolic comorbidities in psoriasis has recently attracted increasing attention. Numerous studies suggested that obesity may increase the risk of psoriasis and exacerbate its symptoms [7, 8]. This relationship is thought to be mediated by the secretion and release of inflammatory factors caused by obesity [9, 10]. In clinical practice, the body mass index (BMI) and WC are established and commonly employed measures for assessing obesity. Metrics prove inadequate for a thorough evaluation of fat distribution within obese individuals, given that people sharing identical BMI values may exhibit varying patterns of fat distribution. Furthermore, WC serves only as a rudimentary gauge for estimating the degree of abdominal obesity. Therefore, more comprehensive obesity assessment indicators are required in clinical practice.

RFM, a newly anthropometric index proposed by Orison in 2018, is considered a more accurate indicator for predicting the total fat percentage of men and women than BMI [11]. Recent studies indicate that RFM is associated with several conditions, such as depression, periodontitis, stroke, and CVD [12–15]. Nonetheless, the association between RFM and psoriasis remains unclear, with limited research investigating this association in the broader US population.

Furthermore, systemic inflammation is a crucial factor in both obesity and psoriasis [16, 17]. A prior research indicated that patients with psoriasis exhibit an elevated release of cytokines from adipose tissue, particularly tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6) [17]. SIRS, an integrated inflammatory biomarker, integrates neutrophil, monocyte, and lymphocyte counts, offering a composite measure of systemic inflammation that has been validated in conditions such as cardiovascular disease and metabolic syndrome [18]. Its potential relevance to psoriasis stems from the inflammatory milieu shared by these disorders. However, its role in modulating obesity-induced psoriasis remains unclear.

Overall, this study used NHANES data to explore the association between RFM and psoriasis risk, addressing an existing knowledge gap. Additionally, we also

examined whether the SIRS mediates the association between elevated RFM and higher psoriasis risk, suggesting that systemic inflammation may act as a connecting factor. The results of this study may provide new epidemiological evidence for the association between RFM and psoriasis, as well as the potential mediating role of SIRS between the two.

Methods

Data source

The NHANES is a cross-sectional survey which organized biennially by the National Center for Health Statistics (NCHS). The NHANES intended to gather comprehensive data on health and nutrition. The NHANES dataset is made available to the public in biennial cycles and encompasses extensive information on dietary habits, nutritional health, overall health, and health-related behaviors. This study included data from the official website of the NHANES for five cycles (2003–2006 and 2009–2014).

Criteria of exclusion: (1) participants under 20 years of age ($N=23,371$); (2) missing measurements for RFM ($N=2151$); and (3) missing measurements of relevant covariates ($N=13,434$). The final sample size was 8479 adults (Fig. 1).

Assessment of psoriasis

Certified dermatologists diagnosed psoriasis through a thorough morphological assessment of distinctly outlined red plaques accompanied by silvery scales. Data were gathered using a questionnaire administered by the interviewer. The survey inquired of participants, “Have you ever been told by a doctor that you had psoriasis?” Individuals who provided a positive response were categorized as having psoriasis, whereas those who chose not to respond or indicated they were unsure were classified as not experiencing the condition.

Definition of RFM and systemic inflammatory index

RFM is an innovative obesity metric that evaluates the percentage of total body fat by measuring height (cm) and WC (cm). RFM is computed using the following formula:

$$RFM_{female} = 76 - (20 \times \frac{Height}{WC})$$

$$RFM_{male} = 64 - (20 \times \frac{Height}{WC})$$

Systemic inflammation was assessed using the SIRS. The formula for SIRS is as follows:

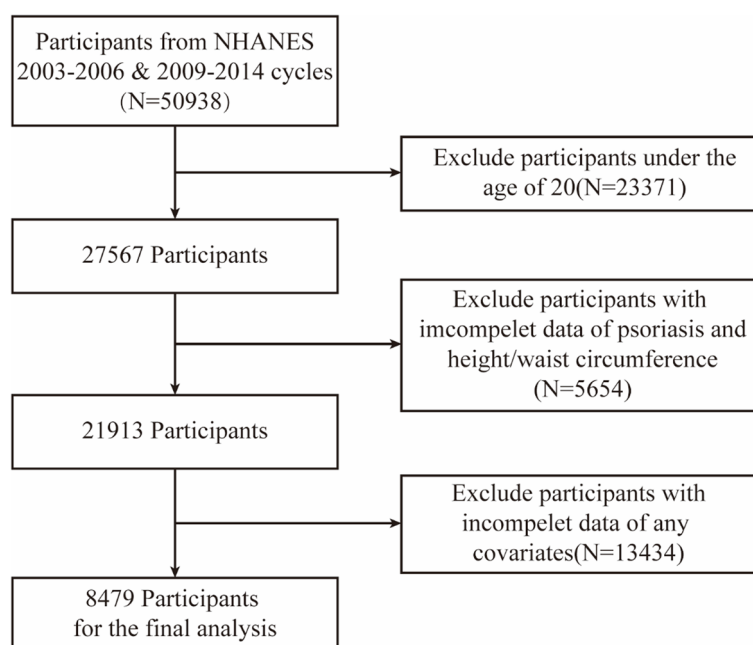


Fig. 1 Flowchart of the inclusion and exclusion process for participants in the NHANES database from 2003–2006 and 2009–2014. NHANES, National Health and Nutrition Examination Survey

$$SIRI = \text{Neutrophil Counts} \times \frac{\text{Monocyte Counts}}{\text{Lymphocyte Counts}}$$

Assessment of covariates

This study considered covariates based on relevant prior research, such as sex, age, ethnicity, poverty income ratio (PIR), marital status, education level, smoking or not, drinking alcohol or not, triglyceride (TG) level, low-density lipoprotein (LDL) level, and history or chronic disease. Detailed descriptions of the covariate assessments are provided in the Supplementary Materials.

Statistical analysis

All statistical analyses were carried out using R software (version 4.4.1) in combination with EmpowerStats (version 4.1). Continuous variables with a normal distribution were shown as means \pm standard deviation (SD), whereas categorical variables were displayed as frequencies (percentages). For continuous variables with normal distributions, an independent samples *t*-test was conducted. Categorical variables were assessed by chi-squared test, and Fisher's exact test was applied when the chi-squared test's assumptions were not met.

A multivariate logistic regression analysis was conducted to investigate the association between psoriasis risk and RFM. Model 1 represents the crude, unadjusted model. Model 2 adjusts for basic demographic factors, including age, gender, and race/ethnicity. Model 3 further

incorporates adjustments for educational level, marital status, PIR, smoking status, alcohol drinking status, history of hypertension, diabetes, CVD status, TG, and LDL levels based on the adjustments made in Model 2.

In model 3, a regression analysis using RCS was employed to further assess the dose–response relationship. Stratified analyses were conducted to assess potential moderating effects of age, sex, race, education status, marital status, PIR, smoke status, alcohol consumption, hypertension, diabetes and CVD. Furthermore, we conducted a mediation analysis to investigate whether RFM (X) impact on the occurrence of psoriasis (Y) is mediated by SIRI (M). The "Mediation" methodology was utilized to estimate various quantities of causal mediation analysis, including indirect effects (IE), direct effects (DE), total effects (TE), and proportional mediation effects. And interaction *P* values were determined using likelihood ratio tests. Statistical significance is indicated by a *P*-value less than 0.05 on a two-tailed test.

Results

Baseline characteristics

This research involved 8479 adults aged 46.0 ± 16.9 years. Data for the excluded participants is provided in Supplementary Material Table 1. The sex distribution was nearly balanced, with 49.1% men and 50.9% women. Among them, 234 (2.76%) had psoriasis. The mean RFM value among the patients was 35.2 ± 8.70 , which was significantly elevated in individuals diagnosed with psoriasis

Table 1 Characteristics of participants in the NHANES datasets 2003–2006 & 2009–2014

Characteristic	Participants ^a			P value
	Total (N = 8479)	Without psoriasis (N = 234)	With psoriasis (N = 8245)	
Age, mean ± SD	46.0 ± 16.9	49.7 ± 16.0	45.9 ± 17.0	< 0.001
Faily PIR, mean ± SD	2.6 ± 1.6	2.8 ± 1.7	2.6 ± 1.6	0.070
TG, mean ± SD	121.5 ± 67.6	129.7 ± 69.5	121.2 ± 67.5	0.057
LDL, mean ± SD	114.3 ± 35.5	115.5 ± 35.6	114.3 ± 35.3	0.611
RFM, mean ± SD	35.2 ± 8.7	36.9 ± 8.4	35.2 ± 8.7	0.004
Year cycles, N (%)				0.4592
2003–2004	1091 (15.6)	31 (18.4)	1060 (15.5)	
2005–2006	1277 (17.0)	36 (20.4)	1241 (16.9)	
2009–2010	2096 (21.2)	60 (19.5)	2096 (22.2)	
2011–2012	1882 (22.8)	58 (24.1)	1824 (22.3)	
2013–2014	2070 (23.4)	49 (17.6)	2021 (23.6)	
Gender, N (%)				0.813
Male	4112 (49.1)	112 (48.2)	4000 (49.1)	
Female	4367 (50.9)	122 (51.8)	4245 (50.9)	
Age, N (%)				0.1582
20 to < 45	4224 (50.6)	92 (41.7)	4132 (50.9)	
45 to < 60	2260 (31.3)	77 (37.8)	2183 (31.1)	
≥ 60	1995 (18.1)	65 (20.5)	1930 (18.0)	
Race and ethnicity^b, N (%)				0.0001
Hispanic	1988 (13.0)	37 (7.2)	1951 (13.2)	
White	4011 (70.5)	148 (82.4)	3863 (70.1)	
Black	1703 (10.3)	32 (6.5)	1671 (10.4)	
Other	777 (6.2)	17 (3.9)	760 (6.3)	
Educational level, N (%)				0.2505
< high school	1945 (15.8)	47 (14.4)	1898 (15.9)	
High school / equivalent	1880 (22.0)	50 (17.9)	1830 (22.2)	
> high school	4654 (62.1)	137 (67.8)	4517 (62.0)	
Marital status, N (%)				0.781
Married	4455 (57.0)	126 (56.7)	4329 (57.0)	
Never married	1645 (18.3)	39 (16.6)	1613 (18.3)	
Living with partner	756 (8.2)	20 (7.4)	736 (8.2)	
Others ^c	1623 (16.5)	56 (19.3)	1567 (16.4)	
Family PIR, N (%)				0.4307
< 1.3	2637 (21.0)	75 (20.1)	2562 (21.1)	
1.3 to < 3.5	3091 (35.5)	66 (31.8)	3025 (35.6)	
≥ 3.5	2751 (43.5)	93 (48.1)	2658 (43.3)	
Smoking status, N (%)				0.0247
No	4703 (54.7)	111 (46.3)	4592 (55.0)	
Yes	3776 (45.3)	123 (53.7)	3653 (45.0)	
Alcohol consumption, N (%)				0.4474
No	2259 (21.6)	54 (18.8)	2205 (21.7)	
Yes	6220 (78.4)	180 (81.2)	6040 (78.3)	
Hypertension, N (%)				0.0001
No	5333 (65.6)	115 (49.3)	5218 (66.1)	
Yes	3146 (34.4)	119 (50.7)	3027 (33.9)	
Diabetes, N (%)				0.9555
No	7440 (90.7)	201 (90.8)	7239 (90.7)	
Yes	1039 (9.3)	33 (9.2)	1006 (9.3)	

Table 1 (continued)

Characteristic	Participants ^a			P value
	Total (N = 8479)	Without psoriasis (N = 234)	With psoriasis (N = 8245)	
CVD, N (%)				0.0269
No	8216 (97.3)	218 (94.7)	7998 (97.3)	
Yes	263 (2.7)	16 (5.3)	247 (2.7)	

BMI Body mass index, *LDL* Low-density lipoprotein, *PIR* Poverty impact ratio, *RFM* Relative fat mass, *SD* Standard deviation, *CVD* Cardiovascular disease, *TG* Triglycerides

^a Data are reported as unweighted counts (weighted percentages)

^b Participants provided self-reported data regarding their race and ethnicity

^c Including widowed, separated, or divorced

(mean RFM: 36.9 ± 8.40) compared to those without psoriasis (mean RFM: 35.2 ± 8.70). Compared with those who had never had psoriasis, participants who had psoriasis were often older, non-Hispanic White, smokers, and had a higher hypertension/CVD history ($P < 0.05$) (Table 1).

Associations between RFM and psoriasis risk

In this section, we conducted a multiple regression analysis, constructing models 1–3 by adjusting for covariates, with the first quartile of RFM serving as the reference group (Table 2). A notable positive association was found in Model 1, suggesting that as RFM increased, the risk of psoriasis also significantly increased ($P < 0.005$). And this association was remained in the other 2 adjusted models. The results from Model 3 revealed that each per unit increment in RFM was associated with a 5% elevation in the risk of developing psoriasis (OR = 1.05, 1.02–1.08). After fully adjusting for covariates, significant differences were observed in the second (Q2, OR = 1.57, 0.97–2.55) and the third quartiles (Q3, OR = 1.78, 1.08–2.95) compared to the reference group Q1, with Q4 showing a more pronounced difference (OR = 2.17, 1.24–3.78). Within the population studied, an elevation in RFM associated significantly with a heightened risk of psoriasis. In all 3 models, the trend-test indicated that the prevalence

of psoriasis increased significantly with an increase in RFM ($P_{\text{for trend}} < 0.05$).

RCS analyses

An RCS analysis was conducted after fully adjusting for covariates in Model 3 to investigate the potential dose–response relationship (Fig. 2). The results demonstrate that there is no significant non-linear relationship between RFM and the risk of developing psoriasis ($P_{\text{for nonlinear}} = 0.536$).

Subgroup analyses

The robustness of the association between RFM and psoriasis was evaluated through subgroup analyses and interaction tests, which also aimed to identify potential differences in populations. The analysis revealed a consistent association in most subgroups (Fig. 3). The PIR notably altered the association between RFM and psoriasis, demonstrating unique associations among various PIR categories. Specifically, a significantly positive association was observed between RFM and psoriasis among adults with middle- and lower-income levels (OR = 1.07, 1.03–1.10; OR = 1.09, 1.05–1.13). In contrast, in individuals with a high-income level, RFM showed no significant association with psoriasis (OR = 1.01, 0.97–1.05). Analyses performed on subgroups categorized by

Table 2 Associations of relative fat mass with psoriasis (N = 8479)

	Prevalence (95% CI)	Model 1		Model 2		Model 3	
		OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Per 1 unit increase	3.09 (2.55, 3.64)	1.02 (1.01, 1.04)	0.0079	1.05 (1.02, 1.08)	0.0011	1.05 (1.02, 1.08)	0.0019
Quartiles							
Q1 (9.4 to < 28.7)	2.16 (1.45, 2.87)	1(Ref.)		1(Ref.)		1(Ref.)	
Q2 (28.7 to < 34.8)	3.41 (2.43, 4.40)	1.60 (0.98, 2.62)	0.0631	1.68 (1.03, 2.73)	0.0396	1.57 (0.97, 2.55)	0.0746
Q3 (34.8 to < 42.4)	3.27 (2.13, 4.41)	1.53 (0.96, 2.46)	0.0796	1.99 (1.20, 3.33)	0.0101	1.78 (1.08, 2.95)	0.0282
Q4 (42.4 to < 58.4)	3.59 (2.58, 4.61)	1.69 (1.07, 2.67)	0.0269	2.49 (1.38, 4.47)	0.0033	2.17 (1.24, 3.78)	0.0086
P for trend		0.0286		0.0031		0.0082	

Abbreviations: *CI* Confidence interval, *OR* Odds ratio, *Q* Quartile

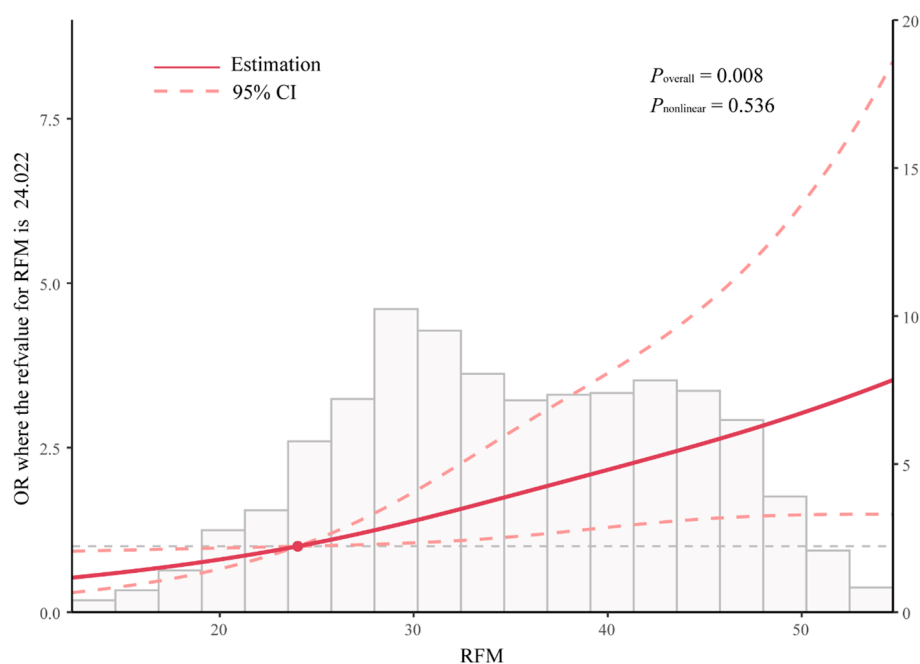


Fig. 2 The dose–response relationship between the RFM and psoriasis. The solid red line illustrates the estimated association, and the dashed lines represent the 95% CI. RFM, relative fat mass; CI, confidence interval

sex demonstrated a noteworthy association in females (OR=1.05, 1.01–1.08, $P<0.05$), whereas the association for males did not show significance.

Mediation analyses

The potential mediating impact of the SIRI on the association between RFM and psoriasis risk was assessed through parallel mediation analyses. The results indicated that the SIRI had significant mediating effects on this association, which accounting for 5.02% of the total effect ($P<0.001$) (Fig. 4).

Discussion

Using the NHANES database, we conducted a cross-sectional study to explore the association between RFM and psoriasis risk among adults in the US. The findings suggest that individuals with heightened RFM possess a greater risk of developing psoriasis, even after thorough consideration of various confounding factors. Additionally, the SIRI was recognized as a potential mediator in this association. The association remained consistent across subgroups of participants stratified by sex, age, race, education, smoking, alcohol drinking, hypertension, diabetes, and CVD. Notably, the association was stronger among female participants, middle- to low-income participants, smokers, and participants without CVD or diabetes. Research suggested that smoking is an independent risk factor for psoriasis, either inducing the

onset of the disease or worsening the existing condition [19]. The result also may be related to sex hormones, differences in daily health management caused by income. Previous study suggested that most immune cells express estrogen receptors and may respond to estrogen stimulation. Androgen regulate inflammation as well by targeting immune cells to attenuate inflammation [20, 21]. And the association between low to medium PIR and abdominal fat accumulation also has been demonstrated by previous studies, poverty leads to cheap food choices, resulting in overconsumption and obesity among households and consumers [22]. Our study also suggested that the association appears to be more pronounced in participants without CVD or diabetes, which indicted multiple variables may lead to disease development simultaneously. The results imply that RFM could serve as an important marker for early identification of psoriasis in individuals at high risk. Further prospective investigations are required to validate the observations made in this research.

Recently, the link between obesity and psoriasis has garnered significant attention from researchers. A mendelian randomization analysis revealed a causal association between BMI and psoriasis [23]. In a mouse model induced by imiquimod, a substantial relationship between obesity and the magnitude of psoriasis severity was also observed, wherein expanded adipose tissue released elevated quantities of cytokines, including IL-6

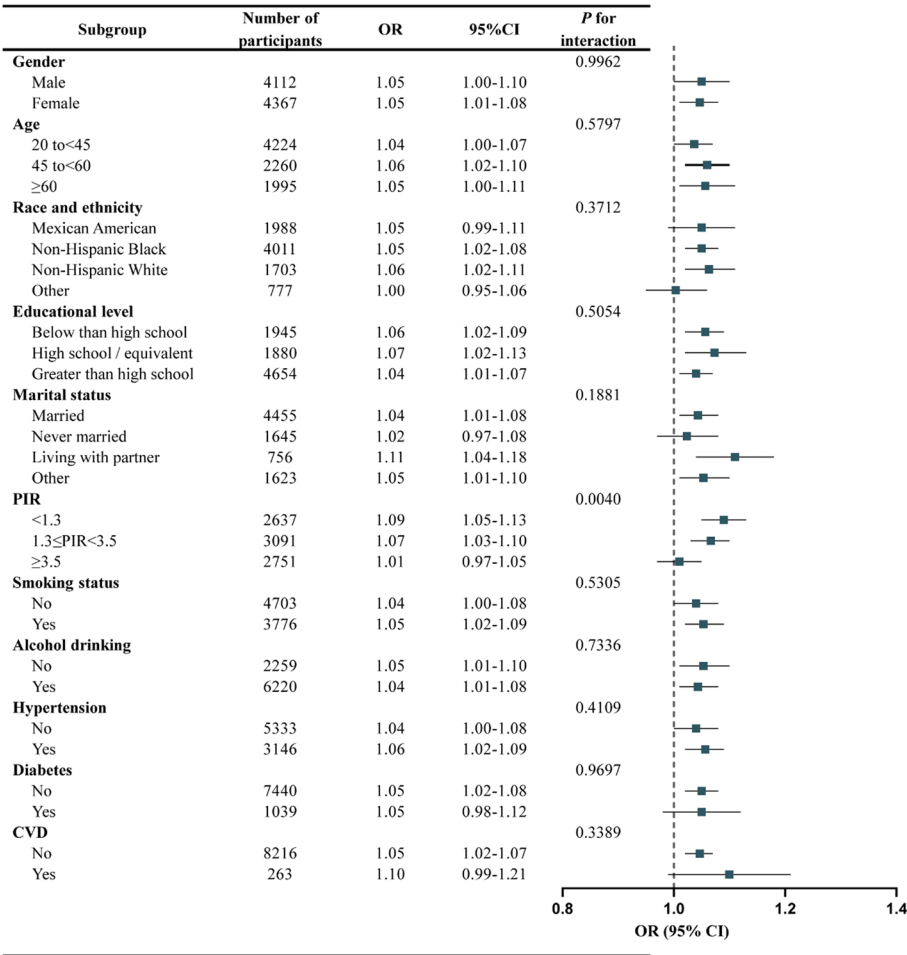


Fig. 3 Stratified analysis of the association between RFM and psoriasis in adults in the NHANES 2003–2006 and 2009–2014. RFM, relative fat mass

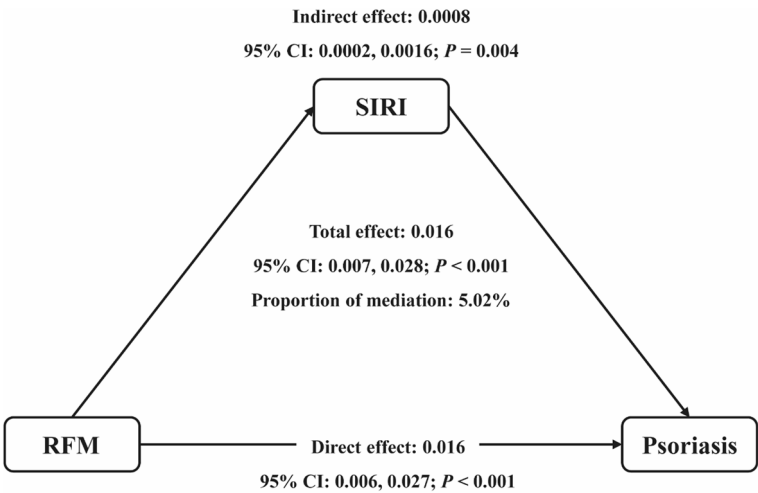


Fig. 4 Path diagram of the mediation analysis of SIRC on the relationship between RFM and psoriasis. SIRC, systemic inflammation response index; RFM, relative fat mass

and TNF- α . There is a closer association between abdominal obesity (excess visceral fat) and psoriasis, possibly because visceral fat releases inflammatory cytokines such as leptin and TNF- α . Therefore, it is crucial to identify indicators of central obesity that can be easily calculated.

RFM is a newly devised index by American researchers, which offers a more precise assessment of body size and fat distribution compared to BMI and WC. It is easy to calculate, cost-effective, and easy to use. A prior investigation revealed that, in comparison to BMI, RFM exhibits a stronger association with obesity assessments derived from dual-energy X-ray absorptiometry (DXA), which are considered more accurate [24]. Besides, according to Wang et al., RFM serves as a reliable predictor for a range of metabolic markers, including glucose and lipid metabolism and inflammation, as demonstrated in a prospective cohort study with 26,754 subjects [12]. Another study also found in a cohort study that RFM predicts dyslipidemia and metabolic syndrome more effectively than BMI [11]. Hence, RFM offers a more thorough and accurate evaluation of obesity-related metabolic disorders.

The findings imply that SIRI mediates the association between RFM and psoriasis, suggesting that systemic inflammation bridges the gap between obesity and psoriasis. Although the exact association between RFM and psoriasis remains unclear, there are some possible explanations. The immune mechanisms underlying obesity and psoriasis share considerable overlap, with inflammatory responses playing a pivotal role in the progression of both conditions. Several studies have suggested that the adipose tissue serves as an active endocrine organ, releasing multiple pro-inflammatory cytokines and adipokines [25–27]. Among the adipokines, leptin, resistin, and adiponectin are the most frequently studied. Leptin and resistin are recognized as pro-inflammatory adipokines due to their ability to stimulate the inflammatory process, whereas adiponectin mitigates the occurrence of inflammatory responses by suppressing TNF- α . Studies have demonstrated that individuals suffering from psoriasis exhibit increased levels of resistin and leptin, whereas the expression of adiponectin is diminished. The secretion of T helper (Th) 1 cells and IL-17A is stimulated by leptin, which might contribute to psoriasis pathogenesis [27]. Resistin has the capacity to stimulate the release of various cytokines, particularly TNF- α , IL-6, and IL-12. The observed decrease in adiponectin levels in psoriasis patients may be explained by the inhibitory relationship that exists between adiponectin and TNF- α . In mice, a deficiency of adiponectin leads to excessive presence of IL-17-producing dermal $\gamma\delta$ T cells, exacerbating inflammation of the skin [28]. The results of this research provide additional indirect support to the findings of the present study, implying that systemic inflammation may

serve as a mediator in the connection between obesity and psoriasis.

Strengths and limitations

This research possesses multiple advantages. It is the initial study to establish a connection between RFM and psoriasis in literature. Multivariate regression and the relationship between RFM and psoriasis risk was explored through the utilization of RCS curve analyses, thereby enhancing our understanding of their association. The use of data from the NHANES offers the advantage of a multi-ethnic representative sample, and its large sample size which could enhance the robustness and generalizability of the findings. Therefore, RFM as a simple and easy-to-use tool for assessing abdominal obesity, may be a potential indicator of psoriasis prevention or management in people. Besides, the study demonstrates that SIRI mediates the relationship between obesity and psoriasis, providing a simple and affordable biomarker for inflammation levels from whole blood cell counts.

While the study has produced significant findings, it is not devoid of limitations. Specifically, the cross-sectional design hinders the establishment of a causal relationship between RFM and psoriasis. Furthermore, self-reported evaluations of specific variables may lead to reporting bias, potentially affecting the observed associations. Besides, accurate estimates of RFM depend on reliable measurements of waist circumference. Although the intra-observer variability between anthropometric measurements is very high, inter-observer variability could be a problem. Measurement error on waist circumference (and height) can be effectively reduced with proper training of healthcare providers. Finally, the study sample exclusively comprised adults residing in the United States, potentially restricting the applicability of the findings to diverse geographical regions. Consequently, it may be necessary to employ additional fundamental research methodologies to confirm the relevance of these findings.

Conclusions

This observational study offers significant insights into the connection between RFM and psoriasis, emphasizing the potential role of the SIRI as a mediator in this relationship. The results indicate that among Americans, RFM is positively associated with the risk of developing psoriasis, underscoring the potential benefits of psoriasis prevention through the management of RFM and SIRI values.

Abbreviations

BMI	Body mass index
CDC	Centers for disease control and prevention
Th	T helper

CVD	Cardiovascular disease
TG	Triglyceride
DXA	Dual energy X-ray absorptiometry
IL	Interleukin
LDL	Low-density lipoprotein
NCHS	National center for health statistics
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
PASI	Psoriasis area and severity index
PIR	Poverty income ratio
RCS	Restricted cubic spline
RFM	Relative fat mass
SIRI	Systemic inflammation response index
SD	Standard deviation
TNF- α	Tumor necrosis factor-alpha
WC	Waist circumference

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12944-025-02528-3>.

Supplementary Material 1.

Acknowledgements

All the authors show their gratitude to the personnel who contributed to the NHANES database.

Authors' contributions

Conceptualization: ZJB, SXY, and BGL; Methodology: ZJB and LQ; Data visualization: BGL, and FYD; Original draft preparation: SXY and YJ; Draft review and editing: ZJB and BGL; Funding acquisition: ZJB and CAJ. All authors reviewed the final version of the manuscript.

Funding

Natural Science Foundation Project of Chongqing (grant number: 2024NSCQ-LZX0086); China Postdoctoral Science Foundation (grant number: 2024M763893).

Data availability

The data employed in this research is available at this website: <https://www.cdc.gov/nchs/nhanes/>.

Declarations

Ethics approval and consent to participate

The NHANES protocol received approval from the NCHS and the Ethics Review Board, and all participants provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 9 December 2024 Accepted: 12 March 2025

Published online: 27 March 2025

References

- Bai G, Peng Y, Liu Q, Shao X, Zhan Y, Chen A, et al. Association between body roundness index and psoriasis among US adults: a nationwide population-based study. *Lipids Health Dis.* 2024;23(1):373.
- Griffiths CEM, Armstrong AW, Gudjonsson JE, Barker JNWN. Psoriasis. *Lancet.* 2021;397(10281):1301–15.
- McGeachy MJ, Chen Y, Tato CM, Laurence A, Joyce-Shaikh B, Blumenschein WM, et al. The interleukin 23 receptor is essential for the terminal differentiation of interleukin 17-producing effector T helper cells in vivo. *Nat Immunol.* 2009;10(3):314–24.
- Zhou X, Chen Y, Cui L, Shi Y, Guo C. Advances in the pathogenesis of psoriasis: from keratinocyte perspective. *Cell Death Dis.* 2022;13(1):81.
- Icen M, Crowson CS, McEvoy MT, Dann FJ, Gabriel SE, Maradit KH. Trends in incidence of adult-onset psoriasis over three decades: a population-based study. *J Am Acad Dermatol.* 2009;60(3):394–401.
- Vanderpuye-Orgle J, Zhao Y, Lu J, Shrestha A, Sexton A, Seabury S, et al. Evaluating the economic burden of psoriasis in the United States. *J Am Acad Dermatol.* 2015;72(6):961–7.e5.
- Gisondi P, Fostini AC, Fossà I, Girolomoni G, Targher G. Psoriasis and the metabolic syndrome. *Clin Dermatol.* 2018;36(1):21–8.
- Ruan Z, Lu T, Chen Y, Yuan M, Yu H, Liu R, et al. Association between psoriasis and nonalcoholic fatty liver disease among outpatient US adults. *JAMA Dermatol.* 2022;158(7):745–53.
- Bavoso NC, Pinto JM, Soares MMS, Diniz MdS, Teixeira Júnior AL. Psoriasis in obesity: comparison of serum levels of leptin and adiponectin in obese subjects - cases and controls. *Anais Brasileiros de Dermatologia.* 2019;94(2):192–7.
- Hsu S, Green LJ, Lebwohl MG, Wu JJ, Blauvelt A, Jacobson AA. Comparable efficacy and safety of brodalumab in obese and nonobese patients with psoriasis: analysis of two randomized controlled trials. *Br J Dermatol.* 2019;182(4):880–8.
- Woolcott OO, Bergman RN. Relative fat mass (RFM) as a new estimator of whole-body fat percentage — A cross-sectional study in American adult individuals. *Sci Rep.* 2018;8(1):10980.
- Wang J, Guan J, Huang L, Li X, Huang B, Feng J, et al. Sex differences in the associations between relative fat mass and all-cause and cardiovascular mortality: A population-based prospective cohort study. *Nutr Metab Cardiovasc Dis.* 2024;34(3):738–54.
- Zhao L, Cao R, Zhang S. Association between relative fat mass and periodontitis: results from NHANES 2009–2014. *Sci Rep.* 2024;14(1):18251.
- Zheng Y, Huang C, Jin J, Zhao Y, Cui H, Wei C. Association between stroke and relative fat mass: a cross-sectional study based on NHANES. *Lipids Health Dis.* 2024;23(1):354.
- Zhu X, Yue Y, Li L, Zhu L, Cai Y, Shu Y. The relationship between depression and relative fat mass (RFM): A population-based study. *J Affect Disord.* 2024;356:323–8.
- Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis. *Jama.* 2020;323(19):1945–60.
- Kawai T, Autieri MV, Scalia R. Adipose tissue inflammation and metabolic dysfunction in obesity. *Am J Physiol Cell Physiol.* 2021;320(3):C375–91.
- Xia Y, Xia C, Wu L, Li Z, Li H, Zhang J. Systemic Immune Inflammation Index (SII), System Inflammation Response Index (SIRI) and risk of all-cause mortality and cardiovascular mortality: a 20-year follow-up cohort study of 42,875 US adults. *J Clin Med.* 2023;12(3):1128.
- Musumeci ML, Nasca MR, Boscaglia S, Micali G. The role of lifestyle and nutrition in psoriasis: Current status of knowledge and interventions. *Dermatol Ther.* 2022;35(9): e15685.
- Merrheim J, Villegas J, Van Wassenhove J, Khansa R, Berrih-Aknin S, le Panse R, et al. Estrogen, estrogen-like molecules and autoimmune diseases. *Autoimmun Rev.* 2020;19(3): 102468.
- Garcia-Gomez E, Vazquez-Martinez ER, Reyes-Mayoral C, Cruz-Orozco OP, Camacho-Arroyo I, Cerbon M. Regulation of inflammation pathways and inflammasome by sex steroid hormones in endometriosis. *Front Endocrinol (Lausanne).* 2019;10:935.
- Okosun IS, Annor FB, Seale JP, Eriksen MP. Abdominal adiposity and family income-to-poverty ratio in American women. *Obes Res Clin Pract.* 2014;8(3):e201–98.
- Budu-Aggrey A, Brumpton B, Tyrrell J, Watkins S, Modalsli EH, Celis-Morales C, et al. Evidence of a causal relationship between body mass index and psoriasis: a mendelian randomization study. *PLOS Med.* 2019;16(1):e1002739.
- Guzmán-León AE, Velarde AG, Vidal-Salas M, Urquijo-Ruiz LG, Caraveo-Gutiérrez LA, Valencia ME. External validation of the relative fat mass (RFM) index in adults from north-west Mexico using different reference methods. *Plos One.* 2019;14(12):e0226767.
- Gerdes S, Rostami-Yazdi M, Mrowietz U. Adipokines and psoriasis. *Exp Dermatol.* 2011;20(2):81–7.
- Sivasami P, Elkins C, Díaz-Saldana PP, Goss K, Peng A, Hamersky M, et al. Obesity-induced dysregulation of skin-resident PPAR γ + Treg

cells promotes IL-17A-mediated psoriatic inflammation. *Immunity*. 2023;56(8):1844–61.e6.

27. Słuczankowska-Glabowska S, Staniszeńska M, Marchlewicz M, Duchnik E, Łuczowska K, Safranow K, et al. Adiponectin, leptin and resistin in patients with psoriasis. *J Clin Med*. 2023;12(2):663.
28. Shibata S, Tada Y, Hau CS, Mitsui A, Kamata M, Asano Y, et al. Adiponectin regulates psoriasiform skin inflammation by suppressing IL-17 production from $\gamma\delta$ -T cells. *Nat Commun*. 2015;6(1):7687.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.