Yang *et al.*
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Association of systemic immune-inflammation index (SII) with risk of psoriasis: a cross-sectional analysis of National Health and Nutrition Examination Survey 2011–2014

Xuan Yang[1,2†], Yuxin Pan[1†], Yang Zhang[1], Yang Meng[1], Tang Tong[3] and Mingyi Zhao[1*]

Abstract

Background The systemic immune-inflammation index (SII) is an emerging marker of inflammation, and the onset of psoriasis is associated with inflammation. The aim of our study was to investigate the potential impact of SII on the incidence rate of adult psoriasis.

Methods We conducted a cross-sectional study based on the National Health and Nutrition Examination Survey (NHANES) 2011–2014 data sets. Multiple logistic regression analyses with appropriate covariates adjustment were the major methods in this study. Subgroup analyses were conducted by age, gender, race, smoking status, alcohol consumption, history of heart attack, stroke, coronary heart disease and diabetes. Interactions among these variables were also detected. We further utilized smooth curve fitting to explore potential nonlinear associations between SII and psoriasis across different subgroups. The receiver operating characteristic curve analysis was used to assess the diagnostic value of SII for psoriasis in the general population and diabetic individuals. Multiple imputation was adopted as sensitivity analysis to address potential bias due to missing data.

Results 9314 participants (≥ 20 years) were included. A significant positive association was observed between SII and psoriasis (OR = 1.56; $P = 0.0069$). Subgroup analysis revealed significant positive association in males (OR = 1.52; $P = 0.0288$), females (OR = 1.61; $P = 0.0322$), Non-Hispanic Whites (OR = 1.55; $P = 0.0190$), people aged 40–59 years (OR = 1.98; $P = 0.0386$), diabetics (OR = 3.40; $P = 0.0088$), and overweight participants (OR = 1.80; $P = 0.0034$). SII had a higher predictive value for psoriasis in diabetic patients (AUC = 0.62; 95% CI [0.55, 0.70]). In stroke patients, SII was negatively correlated with the occurrence of psoriasis, and interaction test suggested the effect of SII on psoriasis was significantly modified by stroke ($P = 0.0003$). Nonlinear relationships between SII and psoriasis were observed in participants aged 20 to 39, former smokers, current drinkers, individuals with or without heart attack, those without coronary heart disease, and overweight participants.

†Xuan Yang and Yuxin Pan have contributed to the work equally and should be regarded as co-first authors.

*Correspondence:
Mingyi Zhao
zhao_mingyi@csu.edu.cn
Full list of author information is available at the end of the article

Conclusions SII was positively associated with psoriasis. Testing for SII levels may help to identify the onset of psoriasis early.

Keywords Systemic immune-inflammation index, Psoriasis, Cross-sectional study, National Health and Nutrition Examination Survey

Introduction

Psoriasis is an immune-mediated chronic, inflammatory, and systemic disease. The typical manifestation of psoriasis is scaly erythema of the skin all over the body [1]. The incidence of psoriasis varies from 0.33 to 0.6% among ethnic groups and affects an estimated 7.4 million adults in the United States [2]. The cause of psoriasis is not fully understood; current studies suggest that psoriasis is caused by both environmental and genetic factors, which ultimately leads to the occurrence or aggravation of the disease [3]. Existing studies have shown that psoriasis is caused by abnormal activation of T cells, which is related to inflammatory factors, such as tumor necrosis factor-α, interleukin (IL)-23, and IL-17 [4]. Psoriasis may not only be associated with other auto-inflammatory diseases, but its worsening can also lead to systemic inflammation [5, 6].

Systemic immune-inflammation index (SII) is a comprehensive inflammatory index determined by lymphocyte, neutrophil, and platelet counts [7]. It can be used to assess the patient's inflammatory and immune status [8, 9]. In past studies, SII has been shown to be useful for the assessment of disease severity and prognostic prediction of various cancers, cardiovascular diseases, immune diseases, among others [10–15]. There are currently blood markers to assess psoriasis, such as C-reactive protein and lymphocyte count [16]. Compared with C-reactive protein, SII is more likely to reflect the changes in lymphocytes. Compared with neutrophil to lymphocyte ratio, SII offers a more comprehensive and intuitive assessment.

There have been studies that have applied SII to the evaluation of psoriasis [17, 18].

Some studies have indicated a positive association between the SII and the risk of psoriasis, with this association being stronger among females, individuals aged 40 to 59, patients with Type 2 diabetes, those without hypercholesterolemia, and individuals who are obese or have metabolic syndrome [19, 20]. However, to date, no studies have explored the potentially complex and nonlinear associations between SII and psoriasis risk across diverse population groups with different demographic characteristics, lifestyles, and medical conditions. This, to some extent, hinders the comprehensive understanding of the association between systemic inflammation and psoriasis.

The National Health and Nutrition Examination Survey (NHANES) is a well-known public database that obtains information about the health and nutritional status of the American people. In our study, we used data from the NHANES population aged 20 years and older from 2011 to 2014 to explore the use of SII as an indicator of inflammation in psoriasis.

Materials and methods

Study design and study population

The Centers for Disease Control and Prevention's National Center for Health Statistics conducted the NHANES, a cross-sectional study, to collect health data from noninstitutionalized civilian population in the United States. The survey consists of a questionnaire investigated at home and a standardized health examination conducted by a mobile examination center. All data and details are publicly available through NHANES website (www.cdc.gov/nchs/nhanes/) [21, 22]. We used data from two consecutive NHANES cycles (2011–2012 and 2013–2014) for our analysis. Participants < 20 years (N = 8602) and pregnant females (N = 111) were first excluded, and then we excluded the rest having missing SII data (N = 918), missing psoriasis data (N = 11), and missing covariate data (N = 976). Finally, we included 9313 participants in this study (Fig. 1).

Exposure factor and outcome

SII was the exposure in this study. Lymphocyte, segmented neutrophil, and platelet counts were performed using the Coulter® method, which accurately counts cells by detecting changes in electrical resistance as they pass through small holes in conductive liquids. These counts were expressed as 1000 cells/ml. SII was calculated using the formula: platelet counts multiplied by segmented neutrophil counts, then divided by lymphocyte counts [23]. The outcome of interest in this study was psoriasis. Trained interviewers used the Computer-Assisted Personal Interviewing system to ask participants "Ever been told by a doctor or other health care professional that you have psoriasis?", and collected the results. As shown in Fig. 2, SII was positively skewed in participants. To address this, we performed a logarithmic conversion of SII in subsequent analyses.

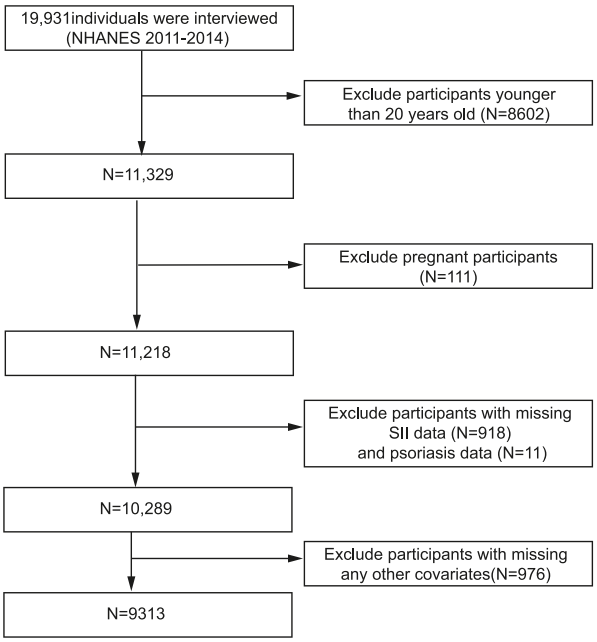

Fig. 1 Flow chart of study participants

Covariates

Based on previous studies, we adopted gender, age, race, education level, ratio of family income to poverty, body mass index (BMI), smoke, alcohol, physical activity, heart attack, coronary heart disease, stroke, and diabetes as covariate [8, 9, 17, 24]. We treated gender, race, education level, smoke (1. Never: participants had not smoked as many as 100 cigarettes in lifetime; 2. Smoking former: participants had smoked up to 100 cigarettes but were not smoking now; 3. Smoking now: participants currently smoked every day or some days.) [25], alcohol (1. Never: participants had fewer than 12 drinks; 2. Drinking former: participants had more than 12 drinks in lifetime, but had not drunk in the last year at the time of the survey; 3. Drinking now: participants had more than 12 drinks in lifetime and had drunk alcohol in the last year; 4. Missing.), physical activity (1. <600; 2. 600–1199; 3. ≥ 1200 MET min per week; 4. Missing) [26], heart attack (1. Yes; 2. No), coronary heart disease (1. Yes; 2. No), stroke (1. Yes; 2. No), and diabetes (1. Yes; 2. No; 3. Borderline) as categorical variables. Ratio of family income to poverty, BMI and age were treated as continuous variables. In the subgroup analysis, participants were classified into underweight (BMI <18.5 kg/m^2), normal (18.5 kg/m^2 $\leq$ BMI <25 kg/m^2) overweight (25 kg/m^2 $\leq$ BMI <30 kg/m^2), and obesity (BMI ≤ 30 kg/m^2) groups based on BMI.

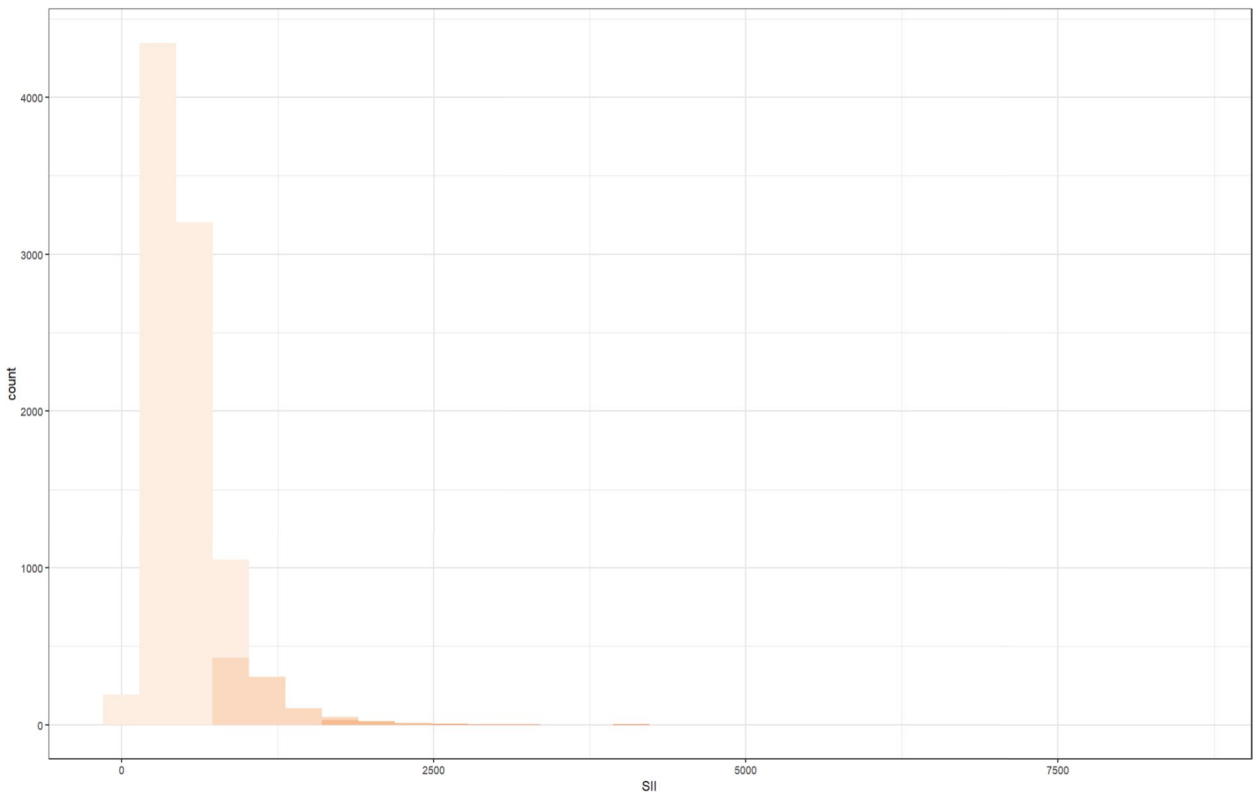

Fig. 2 Histogram of SII in participants

Statistical analysis

The NHANES survey adopted a complex, multistage, probability sampling design to ensure nationally representative health estimates. Therefore, we used CDC-recommended weights in all statistical analyses [27, 28]. We conducted multivariate logistic regression analyses and constructed three models. The non-adjusted model did not include any covariates for adjustment. Model 1 adjusted for gender, age, and race. Model 2 was fully adjusted for all covariates (gender, age, race, education level, ratio of family income to poverty, BMI, smoke, alcohol, physical activity, heart attack, coronary heart disease, stroke, and diabetes were adjusted). We grouped participants according to age (Low: 20–39 years; Middle: 40–59 years; High: 60–80 years), gender, race, smoking status, alcohol consumption, and whether they had experienced a heart attack, stroke, coronary heart disease, or been diagnosed with diabetes. Subgroup analyses were performed based on the above groupings, and interactions among them were examined. To explore the diagnostic and predictive value of SII in terms of psoriasis, the receiver operating characteristic (ROC) curve analysis was conducted. Continuous and categorical variables were represented as survey-weighted mean (95% CI) and survey-weighted percentage (95% CI), respectively. The alpha-level was set at 0.05(two sided) in our study. Sensitivity analyses were performed using the data set after multiple imputation to assess the reliability of the results. All statistical work was done with R 4.2.3, and EmpowerRCH 4.1.

Results

Baseline characteristics of participants

9313 participants were included in our study, including 4554 males and 4759 females. We divided the participants into non-psoriasis group and psoriasis group and described their basic characteristics separately in Table 1. Age, SII, race, stroke, diabetes, and smoking status differed significantly between the two groups ($P<0.05$). The mean value of SII was higher in participants with psoriasis. The box and violin plots demonstrated the distribution of SII in the two groups and conveyed the fact that psoriasis patients have higher values of SII than the normal population (Fig. 3).

The association between SII and psoriasis

In weighted multivariate logistic regression analyses, SII.LOG was positively associated with the incidence of psoriasis, as shown in Table 2. This result was significant in all three models. Each unit increase in SII.LOG would increase the risk by approximately 75% in the non-adjusted model (OR = 1.75; 95% CI [1.37, 2.24]; $P<0.0001$), 62% in the adjust I model (OR = 1.62; 95% CI [1.26, 2.08]; $P=0.0009$), and 56% in the adjust II model (OR = 1.56; 95% CI [1.17, 2.09]; $P=0.0069$). We conducted the sensitivity analysis after transforming SII.LOG into categorical variables to ensure the stability of the results (SII.LOG was equally divided into four groups, and the groups gradually increased from Q1 to Q4). The risk of developing psoriasis in Q4 was approximately 2.51 times higher in the non-adjusted model (OR = 2.51; 95% CI [1.55, 4.08]; $P=0.0008$); 2.25 times higher in the adjust I model (OR = 2.25; 95% CI [1.37, 3.70]; $P=0.0038$), and 2.16 times higher in the adjust II model (OR = 2.16; 95% CI [1.18, 3.95]; $P=0.0171$) than in Q1. As shown in Additional file 2: Figure S1a, the results of ROC curve analysis suggested that SII may have a moderate diagnostic and predictive value for psoriasis (AUC = 0.59; 95% CI [0.56, 0.63]).

Subgroup analysis

We presented the results of the subgroup analyses as forest plots in Fig. 4. We found that the positive association of SII with the incidence of psoriasis was significant in both genders (Male: OR = 1.52; 95% CI [1.05, 2.20]; $P=0.0288$; Female: OR = 1.61; 95% CI [1.04, 2.48]; $P=0.0322$). In other subgroups, the positive association was significant in participants aged 40–59 years (OR = 1.98; 95% CI [1.03, 3.78]; $P=0.0386$), non-Hispanic whites (OR = 1.55; 95% CI [1.13, 2.12]; $P=0.0190$), non-smokers (OR = 2.51; 95% CI [1.69, 3.73]; $P=0.0011$), non-drinkers (OR = 2.86; 95% CI [1.56, 5.25]; $P=0.0027$), those without heart attack (OR = 1.56; 95% CI [1.12, 2.16]; $P=0.0122$), those without stroke (OR = 1.87; 95% CI [1.42, 2.46]; $P=0.0005$), those without coronary heart disease (OR = 1.49; 95% CI [1.09, 2.03]; $P=0.0154$), those with diabetes (excluding those with borderline diabetes) (OR = 3.40; 95% CI [1.62, 7.12]; $P=0.0088$), those without diabetes (OR = 1.57; 95% CI [1.18, 2.08]; $P=0.0114$), and those who were overweight (OR = 1.80; 95% CI [1.21, 2.66]; $P=0.0034$).

Notably, the risk of psoriasis development associated with SII.LOG was particularly pronounced in the diabetic population. Each unit increases in SII.LOG was associated with an approximately 2.4-fold increased risk of psoriasis development in the diabetic population (OR = 3.40). We further performed ROC curve analysis for the diabetic population to investigate the diagnostic or predictive value of SII for psoriasis in this population. As shown in Additional file 2: Figure S1b, SII had a higher predictive value for psoriasis in the diabetic population (AUC = 0.62; 95% CI [0.55, 0.70]) compared to the general population. Another valuable result was that in participants who had uffered a stroke, SII.LOG was negatively associated with the risk of psoriasis (OR = 0.04; 95% CI [0.003, 0.57]; $P=0.0211$). In our sensitivity analysis, we

Table 1 Description of participants characteristics

	Non-psoriasis	Psoriasis	*P*-value
Age (year)	47.35 (46.45, 48.24)	51.57 (49.16, 53.99)	0.0009
Ratio of family income to poverty	2.91 (2.77, 3.06)	3.05 (2.80, 3.31)	0.1619
BMI (kg/m^2)	28.95 (28.68, 29.22)	29.97 (28.74, 31.20)	0.0975
SII	526.43 (512.66, 540.20)	614.23 (564.63, 663.82)	0.0005
Gender			0.4582
Male	48.74 (47.69, 49.79)	46.39 (40.03, 52.86)	
Female	51.26 (50.21, 52.31)	53.61 (47.14, 59.97)	
Race			< 0.0001
Mexican American	8.10 (5.94, 10.94)	4.35 (2.20, 8.41)	
Other Hispanic	5.88 (4.28, 8.02)	4.70 (2.61, 8.32)	
Non-Hispanic White	67.54 (62.23, 72.43)	79.31 (72.64, 84.70)	
Non-Hispanic Black	10.78 (8.38, 13.75)	5.17 (3.22, 8.22)	
Other Race	7.71 (6.46, 9.17)	6.46 (4.30, 9.61)	
Education level			0.4584
Less than high school	15.05 (12.84, 17.55)	12.68 (7.74, 20.08)	
High school	20.94 (18.98, 23.04)	18.82 (12.94, 26.55)	
Higher than high school	64.02 (60.56, 67.34)	68.51 (58.30, 77.19)	
Coronary heart disease			0.3434
Yes	3.23 (2.63, 3.96)	4.39 (2.47, 7.65)	
No	96.77 (96.04, 97.37)	95.61 (92.35, 97.53)	
Heart attack			0.1053
Yes	3.11 (2.69, 3.60)	5.12 (2.93, 8.82)	
No	96.89 (96.40, 97.31)	94.88 (91.18, 97.07)	
Stroke			0.0268
Yes	2.70 (2.36, 3.09)	5.28 (2.89, 9.45)	
No	97.30 (96.91, 97.64)	94.72 (90.55, 97.11)	
Physical activity			0.4773
< 600 MET min per week	37.48 (36.13, 38.85)	41.56 (33.42, 50.18)	
600–1199 MET min per week	17.04 (15.71, 18.47)	14.92 (10.76, 20.33)	
≥ 1200 MET min per week	23.18 (21.84, 24.58)	19.03 (13.59, 25.99)	
Missing	22.30 (20.74, 23.94)	24.49 (17.03, 33.89)	
Drink			0.9280
Never	10.15 (8.22, 12.47)	9.92 (6.33, 15.24)	
Drinking former	4.32 (3.68, 5.06)	4.13 (1.49, 10.91)	
Drinking now	7.12 (6.40, 7.91)	8.56 (4.92, 14.49)	
Missing	78.41 (75.78, 80.84)	77.38 (70.72, 82.90)	
Diabetes			0.0097
Yes	9.56 (8.67, 10.52)	15.81 (11.39, 21.54)	
No	88.00 (86.92, 89.00)	81.23 (75.38, 85.96)	
Borderline	2.44 (2.05, 2.91)	2.95 (1.28, 6.68)	
Smoke			< 0.0001
Never	56.24 (54.19, 58.27)	47.76 (40.12, 55.51)	
Smoking former	23.67 (22.00, 25.42)	40.65 (33.91, 47.75)	
Smoking now	20.09 (18.47, 21.82)	11.59 (8.31, 15.94)	

Continuous variables were represented as survey-weighted mean (95% CI). Categorical variables were represented as survey-weighted percentage (95% CI)

transformed SII.LOG into a categorical variable and performed an interaction test. After comprehensively considering and adjusting for all relevant covariates, we observed a significant interaction between stroke and the association between SII.LOG and the risk of developing psoriasis ($P = 0.0003$) (Additional file 1: Table S1).

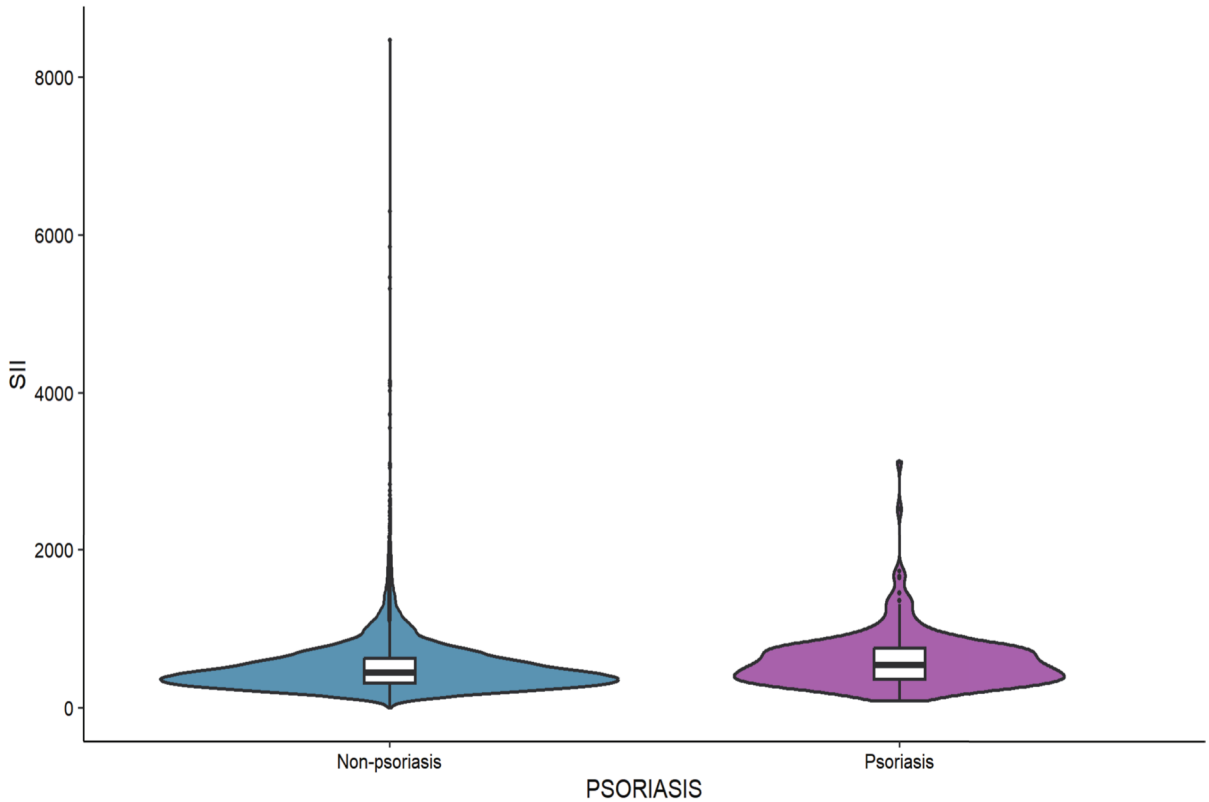

Fig. 3 Distribution of SII in non-psoriasis and psoriasis participants

Table 2 Weighted multivariate logistic analysis SII.LOG and psoriasis

	Non-Adjusted OR (95% CI), *P*-value	**Adjust I OR (95% CI), *P*-value**	**Adjust II OR (95% CI), *P*-value**
SII.LOG	1.75 (1.37, 2.24), < 0.0001	1.62 (1.26, 2.08), 0.0009	1.56 (1.17, 2.09), 0.0069
Stratified by SII.LOG quartiles			
Q1	ref	ref	ref
Q2	1.25 (0.74, 2.10), 0.4082	1.17 (0.69, 2.00), 0.5620	1.15 (0.59, 2.27), 0.6073
Q3	1.33 (0.78, 2.25), 0.2993	1.23 (0.72, 2.12), 0.4526	1.18 (0.59,2.37), 0.5645
Q4	2.51 (1.55, 4.08), 0.0008	2.25 (1.37, 3.70), 0.0038	2.16 (1.18,3.95), 0.0171

Non-adjusted: no adjust for covariates. Adjust I: gender, age, and race were adjusted. Adjust II: gender, age, race, education level, ratio of family income to poverty, BMI, smoke, alcohol, physical activity, heart attack, coronary heart disease, stroke, and diabetes were adjusted

The results of smooth curve fitting across different subgroups (Fig. 5) indicate that this nonlinear relationship is predominantly significant in the following subgroups: participants aged 20 to 39, former smokers, current drinkers, individuals regardless of whether they have heart attack, those without coronary heart disease, and those who are overweight.

Sensitivity analysis

To test the reliability and stability of the results, we performed multiple imputation on the missing values of continuous variables in the covariates five times. The newly generated data sets were analyzed separately, and their combined effects were calculated. In the sensitivity analyses, the positive association between SII.LOG and the risk of psoriasis remained significant ($P < 0.05$) (Additional file 1: Table S2), The results of the subgroup analyses were also consistent with the original analyses (Additional file 3: Figure S2). In addition, we transformed SII.LOG into categorical

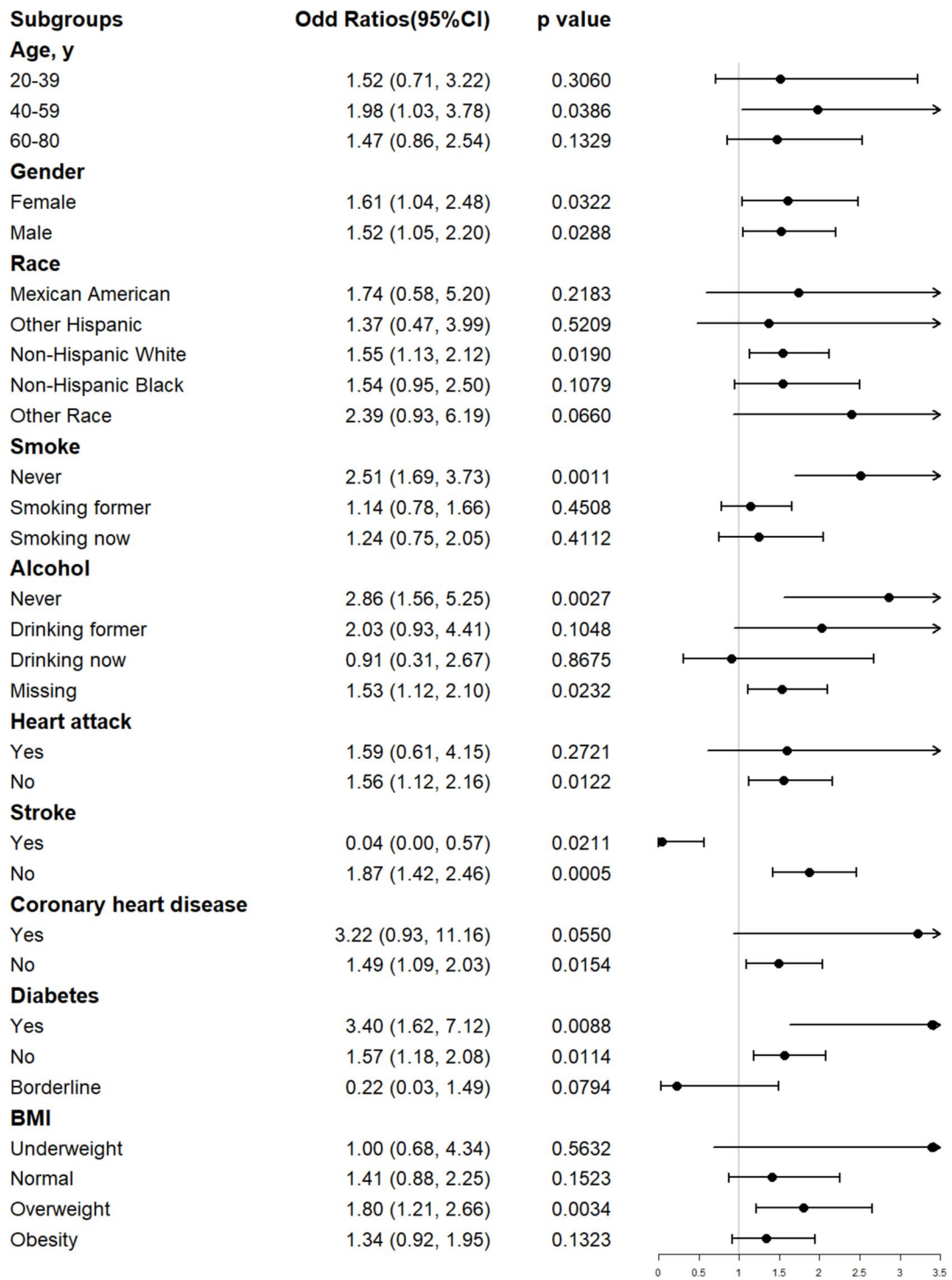

Fig. 4 Forest plot of subgroup analysis. Gender, age, race, education level, ratio of family income to poverty, BMI, smoke, alcohol, physical activity, heart attack, coronary heart disease, stroke, and diabetes were adjusted in all subgroup analyses

variables to conduct multivariate logistic regression and subgroup analyses. The results were in agreement with the original results (Table 2, Additional file 1: Table S1).

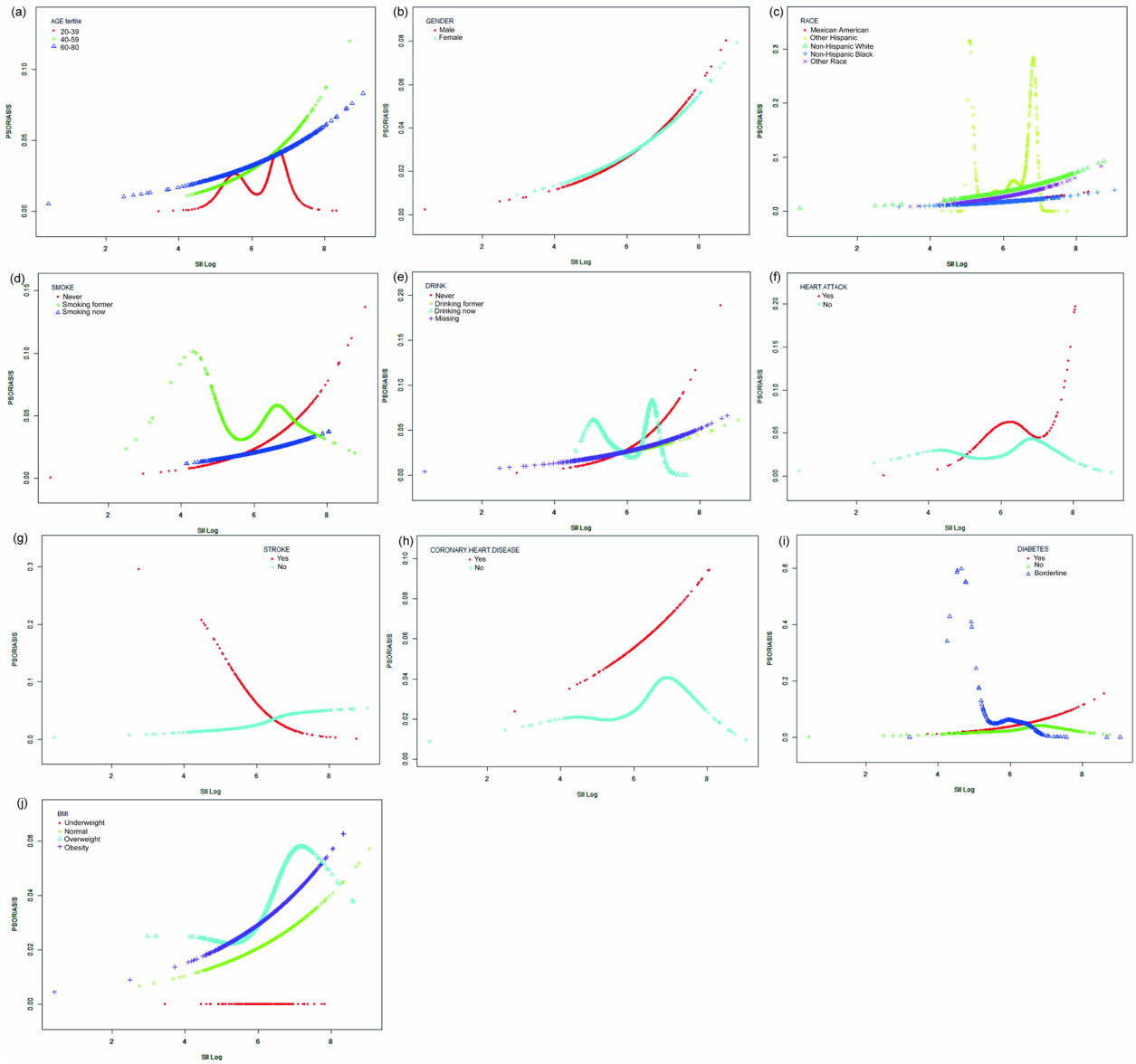

Fig. 5 Nonlinear relationship between SII and psoriasis risk across various demographics, including age (**a**), gender (**b**), and race (**c**), lifestyle factors such as smoking (**d**) and drinking (**e**), medical conditions like heart attack (**f**), stroke (**g**), coronary heart disease (**h**), and diabetes (**i**), as well as varying BMI levels (**j**)

Discussion

We used the 2011–2014 study sample from the NHANES database as a representative of U.S. adults. Our results show that the higher the level of SII, the higher the incidence of psoriasis. The results remained significant after adjusting for some and all covariates.

Previous studies have shown that in the pathogenesis of psoriasis, trauma or infection can induce host cell-derived nucleotides to form complexes with keratinocyte derived antimicrobial peptides, which causes the expansion of T cells in the skin and lymph nodes [29]. Plasma cell dendritic cells produce type I interferons and activate myeloid dendritic cells to secrete IL-23 and TNF [30]. These factors enhance the production of IL-17 and IL-1 by Th22 cells, which in turn activates TNF, CCL20, and antimicrobial peptides [31, 32]. Existing studies have shown that IL-17-driven inflammation is usually controlled by regulatory T cells and anti-inflammatory cytokines, which produce protective effects of inflammation by promoting neutrophil recruitment and antimicrobial peptide production [33,

34]. Therefore, it can be inferred that the elevation of neutrophils is related to the onset of psoriasis.

Platelets are important components of hemostasis, coagulation, thrombosis and inflammation. On one hand, platelets play a direct role as immune effector cells, on the other hand, they trigger the response of immune effector cells and indirectly cause the immune response of the body [35]. Existing studies have begun to shed light on the relationship between platelets and psoriasis. Platelet activating circulatory markers are elevated in psoriasis patients [36], at the same time, clinical research and animal studies also demonstrate that platelets are preferentially present in psoriatic skin [16]. Platelets play an initiating and regulating role in the inflammatory response. Platelet dysfunction is closely related to the pathogenesis of psoriasis [37]. In psoriasis, platelets promote the secretion of IL-4 by CD17 cells and induce endothelial injury and apoptosis through neutrophil cells in vitro, which may be one of the mechanisms of platelet activation inducing psoriasis [38, 39]. In addition, platelet surface antigen is the main feature of psoriatic polymorphonuclears [16]. If we can confirm that platelets are one of the first cells recruited to the skin, we can also prove that the early inflammatory response in psoriasis may be related to platelet activation, that is, platelets are a potential driver of skin inflammation in psoriasis [35].

There is increasing evidence for the pathogenic role of T lymphocytes in psoriasis. Driven primarily by pathogenic T cells, these T cells produce high levels of IL-23 in response to IL-17. The IL-23/T17 cell axis is the core axis in the development of psoriasis [40]. Interestingly, our results show a strong positive association between psoriasis onset and SII, which means that psoriasis onset and lymphocytes may be negatively or not associated. On one hand, acc retrospective study by Ahu Yorulmaz et al. showed that patients with psoriasis had lower lymphocyte counts compared to controls [18]. On the other hand, Sokolova et al. questioned the assessment of systemic inflammation in psoriasis, and their study found that CRP was normal in most patients with psoriatic arthritis [41]. However, determining systemic inflammation cannot be done with just one or a few markers of inflammation. In contrast, the number of single lymphocytes does not reflect the inflammatory state of the body, and we speculate that it may not be directly related to the onset of psoriasis. As a whole index, SII may be more suitable to reflect the inflammatory state and pathogenesis of psoriasis.

Based on the results of our study, SII may be one of the indicators to evaluate the onset of psoriasis and predict the onset of psoriasis. SII is obtained by simple calculation of three simple indicators: platelets, neutrophils and lymphocytes [42]. It has been shown that SII can not only help predict the onset of psoriasis, but also play a role in predicting and assessing the severity of psoriatic arthritis [17, 18]. In addition, SII can be used as an early screening indicator for psoriatic arthritis [43] and as a marker of disease activity [44]. Traditional blood tests for psoriasis usually only measure white blood cell related values. Compared with the traditional blood test for psoriasis patients, SII can be used as a new indicator to assist in the early assessment and diagnosis of psoriasis. Early detection and treatment can undoubtedly effectively improve the quality of life of patients with psoriasis.

Based on the findings of our study and those of previous similar research studies, a consensus has emerged across these studies regarding the significant association between the SII and the risk of psoriasis among females, individuals aged 40–59, diabetics, those without cardiovascular disease, and those who are overweight or obese [19, 20]. Furthermore, this study uncovered a novel finding: a significant association between SII and the risk of psoriasis is also observed in individuals who neither smoke nor drink alcohol. Even more notably, a significant negative association between SII and the risk of psoriasis is evident in stroke patients. In light of these findings, we have conducted smooth curve fitting analyses for each subgroup. The results reveal potential nonlinear relationships between SII and the risk of psoriasis among participants aged 20 to 39, former smokers, current drinkers, individuals with or without heart attack, those without coronary heart disease, and overweight participants. This may suggest a complex association between inflammation and psoriasis in these populations. When assessing the relationship between SII and psoriasis in these specific groups, we need to adopt a more multidimensional and comprehensive approach.

Psoriasis has long been recognized as a disease with many complications, and due to its systemic inflammatory nature, patients with psoriasis have a higher incidence of brain and cardiovascular diseases [45]. Furthermore, psoriasis patients are at increased risk for other chronic and serious health conditions [46]. Including cardiovascular disease, metabolic syndrome, and even mental illness [47]. These diseases further increase the burden of disease and have a great impact on the morbidity and mortality of patients with psoriasis [46]. Despite this, from an epidemiological perspective, similar studies have failed to reveal a significant association between SII and psoriasis in patients with cardiovascular diseases. Only our study has suggested a complex nonlinear association between SII and the incidence of psoriasis in patients with heart attack. Although patients with cardiovascular diseases often have long-term chronic inflammatory responses, inflammation may not be the decisive factor in the concurrence of psoriasis and cardiovascular diseases.

To verify this viewpoint, more in-depth in vitro or in vivo experimental studies are needed.

In patients with diabetes, SII has the greatest impact on the onset of psoriasis. ROC curve analysis of the diabetic population also showed that SII had a higher predictive value for psoriasis in the diabetic population than in the general population. It is well known that chronic tissue inflammation has become a key feature of type 2 diabetes, SII can be used as an indicator to evaluate type 2 diabetes [48]. In addition, we already know that SII is positively associated with the incidence of psoriasis. Therefore, in the case of significant systemic inflammation, we speculate that the SII value has the greater impact on the onset of psoriasis, especially in type 2 diabetes. Another interesting finding we made was that SII was negatively associated with psoriasis in the stroke population. Numerous studies have shown systemic inflammation in stroke patients [49, 50]. Inflammation can be both harmful and beneficial in specific stages after a stroke. Although it causes the infarction to expand, it also remodels and repairs [51]. The inflammatory response expressed may also vary at different stages of the stroke course. However, the association between SII values and psoriasis in stroke patients was not elevated due to this inflammatory response, and more research may be needed to identify new pathways.

Our research has several advantages. First, our study used a large sample population from the NHANES database, which enhances the reliability and representativeness of the study. Next, we used a variety of statistical software and log-transformed SII values for greater accuracy in results. What's more, we did a more comprehensive subgroup analysis, ROC curve analysis of the whole population and special subgroups, and also used the forest map to make the results more intuitive, to clarify the difference in the effect of SII on psoriasis in different populations. Last but not least, multiple interpolation is used for sensitivity analysis to reduce the probability of false positives. However, our study also has some limitations. On one hand, cross-sectional studies are not good at describing causality. Prospective studies are required to further determine the causality between SII and psoriasis. On the other hand, although we controlled for certain confounders, the interactions between inflammation and disease are complex, and our data only represent participants from 2011 to 2014, so it cannot be ruled out that these findings are the result of a small sample size and that all confounders cannot be ruled out.

Conclusion

In summary, our study found a strong association between elevated SII and the onset of psoriasis. The association is sometimes higher when patients had other systemic diseases at the same time. In patients with diabetes, SII had the greatest effect on the onset of psoriasis, indicating that it has better predictive value in the diabetic population. The negative association between SII and psoriasis observed in the stroke population still needs further study. However, due to the limitations of this study, we look forward to conducting prospective studies to explore the causal relationship between SII and psoriasis, and to investigate the possibility of clinical application of SII in psoriasis.

Abbreviations

SII	Systemic immune-inflammation index
NHANES	National Health and Nutrition Examination Survey
BMI	Body mass index
ROC	Receiver operating characteristic

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s40001-025-02304-0.

Additional file 1.

Additional file 2: Figure S1. ROC curve in all participants (**a**) and in diabetics (**b**).

Additional file 3: Figure S2. Forest plot of subgroup analyses in sensitivity analysis. Gender, age, race, education level, ratio of family income to poverty, BMI, smoke, alcohol, physical activity, heart attack, coronary heart disease, stroke, and diabetes were adjusted in all subgroup analyses.

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Author contributions

XY and YP contributed equally to this work. XY and YP analyze the data and write the manuscript. MZ was responsible for conceptualizing the study and critically reviewing the manuscript. YZ, YM, and TT help edit ICONS and tables and revise manuscripts. All authors have read and approved the published version of the manuscript.

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Availability of data and materials

The data collected in this study was publicly available via NHANES website (www.cdc.gov/nchs/nhanes/).

Declarations

Ethics approval and consent to participate

NHANES is a free and publicly available database that has been approved by the Ethics Review Board of the National Center for Health Statistics (https://www.cdc.gov/nchs/nhanes/irba98.htm). All participants signed an informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details
[1]The Department of Pediatrics, The Third Xiangya Hospital of Central South University, Changsha, 410013, Hunan, China. [2]The Department of Dermatology, Xiangya Hospital of Central South University, Changsha 410083, Hunan, China. [3]Academician Workstation, Changsha Medical University, Changsha 410219, Hunan, China.

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