Inflammatory dietary pattern and incident psoriasis, psoriatic arthritis, and atopic dermatitis in women: A cohort study

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Background: Diet is a modulator of inflammation that might impact inflammatory skin diseases.

Objective: To assess the relationship between pro-inflammatory dietary patterns and incident psoriasis, psoriatic arthritis (PsA), and atopic dermatitis (AD).

Methods: We conducted cohort studies among women in the Nurses' Health Study II. The Empirical Dietary Inflammatory Pattern (EDIP) score was calculated at baseline and every 4 years. Incident psoriasis, PsA, and AD were assessed by validated self-report. We used multivariable-adjusted Cox proportional hazards models to calculate hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between EDIP quintiles and risk for psoriasis, PsA, and AD.

Results: We had 85,185 participants in the psoriasis analysis and 63,443 in the AD analysis. There were 1432 cases of psoriasis, 262 cases of PsA, and 403 cases of AD. Pro-inflammatory dietary patterns were not associated with the risk for outcomes in multivariable models (all *P* values for trend >.05). HRs comparing the highest to the lowest EDIP quintile were 0.99 (95% CI 0.83-1.18) for psoriasis, 1.22 (95% CI 0.81-1.83) for PsA, and 0.96 (95% CI 0.69-1.34) for AD.

Limitations: Recall and self-report.

Conclusion: Our findings do not support dietary inflammatory potential as a risk factor for psoriasis, PsA, or AD. (J Am Acad Dermatol 2019;80:1682-90.)

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CAPSULE SUMMARY

- Diet is an important modulator of systemic inflammation and might play a role in the development and progression of inflammatory skin diseases.
- Our findings do not support that decreasing the inflammatory potential of the diet can be used as a preventive measure in patients at risk for psoriasis, psoriatic arthritis, or atopic dermatitis.

Psoriasis and atopic dermatitis are chronic immune-mediated, inflammatory skin conditions affecting 3%[1,2] and 5%-7%,[3,4] respectively, of the US adult population. Psoriatic arthritis, a seronegative spondyloarthropathy, is present in up to 30% of patients with psoriasis.[5] Although intrinsic immunologic processes play a role in the pathogenesis of these conditions,[6-8] there are well-recognized factors, such as obesity,[9-11] smoking,[12-15] and stress,[16,17] that could modulate risk, severity, and prognosis of these inflammatory conditions.

Diet is a modifiable factor implicated in chronic systemic inflammation[18-20] and in the development and progression of several chronic diseases.[21-24] Recent meta-analyses highlight the anti-inflammatory benefit of the Mediterranean diet; this diet may reduce circulating C-reactive protein (CRP), interleukin 6 (IL-6), and adiponectin concentrations[25] and may lower the risk for diabetes[26] and cancer mortality,[27,28] both of which have known pro-inflammatory pathologies.[29-33] The Empirical Dietary Inflammatory Pattern (EDIP) is a recently developed and validated measure[34]; dietary patterns with high EDIP scores are associated with higher levels of tumor necrosis factor α (TNF-α), TNF-α receptor 1, TNF-α receptor 2, CRP, IL-6, and adiponectin.[34-36] Psoriasis and psoriatic arthritis are T-cell helper 1 (T_H1)—mediated and T_H17-mediated diseases; there are increased serum levels of IL-6, CRP, and TNF-α,[37] biomarkers of systemic inflammation that are also elevated in other chronic inflammatory diseases, such as diabetes,[38] cardiovascular disease,[39,40] and inflammatory bowel disease.[41] In contrast with psoriasis and psoriatic arthritis, atopic dermatitis is primarily a T_H2-mediated skin disease, with lesser T_H1 and T_H17 inflammatory cytokine involvement.[42]

Although many patients wonder whether an anti-inflammatory diet can decrease their risk for inflammatory skin disease, patients and clinicians have limited evidence on which to base ensuing discussions. The objective of our study was to assess whether pro-inflammatory dietary patterns increase the risk for incident psoriasis, psoriatic arthritis, and atopic dermatitis in a large cohort of US women. We hypothesized that consumption of a pro-inflammatory diet, as measured by higher EDIP scores, would increase the risk of developing psoriasis and psoriatic arthritis. Given the divergent inflammatory mechanisms underlying psoriatic disease and atopic dermatitis, we expected the relationship between EDIP score and atopic dermatitis to be null.

MATERIALS AND METHODS

Study population

The Nurses' Health Study II (NHS-II) is a large prospective cohort study started in 1989 involving 116,430 US female nurses aged 25-42 years. Data on risk factors and diseases are collected by questionnaire biennially.

Assessment of diet and calculation of EDIP scores

In the NHS-II, food frequency questionnaires were started in 1991 and are administered every 4 years. The development of the EDIP score in a sample of 5230 women in the Nurses' Health Study has been described elsewhere.[34] In brief, the EDIP is an empirical score of the overall inflammatory potential of diets that is based on the consumption of food groups most predictive of 3 plasma markers of inflammation (IL-6, CRP, and TNF-α receptor 2). The EDIP score is the weighted sum of 18 food groups, with lower scores indicating diets that are maximally anti-inflammatory and higher scores indicating diets that are maximally pro-inflammatory. The EDIP score has been validated[34] and has a higher ability to predict concentrations of CRP, TNF-α, and adiponectin than nutrient-based dietary inflammatory indices.[35]

The following food groups comprising the EDIP score are positively correlated with inflammatory marker concentrations (ie, pro-inflammatory): processed meat, red meat, organ meat, white fish, vegetables other than green leafy vegetables and dark yellow vegetables, refined grains, high-energy beverages (cola and other carbonated beverages with sugar and fruit drinks), low-energy beverages (low-energy cola and other low-energy carbonated beverages), and tomatoes. The following food

Abbreviations used:

BMI:	body mass index
CI:	confidence interval
CRP:	C-reactive protein
EDIP:	Empirical Dietary Inflammatory Pattern
HR:	hazard ratio
IL-6:	interleukin-6
NHS-II:	Nurses' Health Study II
T_H1:	T-cell helper 1
TNF-α:	tumor necrosis factor α

groups are inversely correlated with inflammatory marker concentrations (ie, anti-inflammatory): beer, wine, tea, coffee, dark yellow vegetables, green leafy vegetables, snacks (ie, popcorn, crackers), fruit juice, and pizza.[34] Some of these groupings are counterintuitive and apparently contradictory, but the groupings can be explained by the differential nutritional content in the related foods. For example, fresh tomatoes have a low content of bioavailable lycopene (a major anti-inflammatory nutrient)[43] compared with 2-5 times higher concentrations in cooked tomato paste.[44] Furthermore, pizza typically contains large amounts of high-fat dairy, which has anti-inflammatory and low insulinemic properties.[45] This explains why pizza is grouped as anti-inflammatory while tomatoes are not. As the goal of the EDIP score is to account for the overall effect of dietary patterns, only food groups that explain maximal variation in the 3 noted inflammatory biomarkers are retained for analysis.

Diagnosis of psoriasis and psoriatic arthritis

Participants were asked whether they had received a psoriasis diagnosis by a clinician periodically throughout cohort follow-up. Participants were asked the year of diagnosis in intervals when assessed in 2005 (before 1991, 1991-1994, 1995-1998, 1999-2002, and 2003+), 2009 (before 1995, 1995-1999, 2000-2004, 2005-2006, and 2007+), and 2013 (before 1995, 1995-2002, 2003-2008, 2009-2010, and 2011+). Psoriasis reports were confirmed with the Psoriasis Screening Tool questionnaire, which confirms cases with a 94% specificity.[46] Participants completing the Psoriasis Screening Tool were also asked the specific year in which their psoriasis was diagnosed. Participants with psoriasis were asked whether they had also received psoriatic arthritis diagnoses, and their reports were validated using the Psoriatic Arthritis Screening and Evaluation questionnaire, which has a 73%-80% specificity.[47] Only validated cases of psoriasis and psoriatic arthritis were counted as incident cases in our analyses.

Diagnosis of atopic dermatitis

Atopic dermatitis was assessed by self-report in 2013. Patients were asked if they ever received a diagnosis of eczema (atopic dermatitis) by a clinician and what year this occurred in intervals (before 1995, 1995-2002, 2003-2008, 2009-2010, and after 2010). In 2017, women who had reported a diagnosis of atopic dermatitis were sent a supplemental questionnaire asking them to reaffirm their self-report and answer related questions.[48] Questions from that supplemental questionnaire are used in 2 separate algorithms to confirm a diagnosis of atopic dermatitis with a ≥84% specificity.[49] Our primary atopic dermatitis case definition (definition 1) was women who reaffirmed their self-report on the supplemental questionnaire. In sensitivity analyses, we applied the 2 validated algorithms to enhance the specificity of the diagnosis (definitions 2 and 3).

Assessment of covariates

At cohort baseline (1989), participants were asked their height and race/ethnicity. Biennially, participants were asked about their weight; smoking status; physical activity; and diagnoses of hypercholesterolemia, type 2 diabetes, cardiovascular disease, and asthma.

Statistical analysis

We excluded participants with unknown birth years. For the psoriasis and psoriatic arthritis analysis, participants were excluded if they had died before 1991, had prevalent psoriasis at baseline in 1991, or reported having psoriasis but without confirmation during validation. For the atopic dermatitis analysis, participants were excluded if they had died before 1995, had prevalent atopic dermatitis at baseline in 1995, or reported atopic dermatitis on the main questionnaire but did not have their report confirmed during validation. In 2009, participants were also asked about a history of atopic dermatitis without a year of diagnosis. Patients who reported having a history of atopic dermatitis in 2009 but not in 2013 were excluded; 3589 with self-reported atopic dermatitis and 352 from the validation study were removed from the study. In total, 85,185 patients were included in the psoriasis and psoriatic arthritis analysis, and 63,443 patients were included in the atopic dermatitis analysis.

We calculated person-years of follow-up from the date of return of the baseline questionnaire for each analysis until the date of death; diagnosis of psoriasis, psoriatic arthritis, or atopic dermatitis; or end of follow-up (2013), whichever was earliest. The date of diagnosis of skin disease was considered the

Table I. Characteristics of Nurses' Health Study II participants by baseline Empirical Dietary Inflammatory Pattern score in quintiles

	Total Empirical Dietary Inflammatory Pattern score quintile				
Characteristic by cohort	**1**	**2**	**3**	**4**	**5**
Psoriatic disease cohort (1991)					
No. participants	17,037	17,037	17,037	17,037	17,037
Age, years,* in 1991	37 ± 4	37 ± 5	36 ± 5	36 ± 5	35 ± 5
Empirical Dietary Inflammatory Pattern score	−477 ± 214	−174 ± 52	−18 ± 42	140 ± 53	465 ± 217
White race	98	97	96	95	94
Body mass index, kg/m^2	24 ± 4	24 ± 5	24 ± 5	25 ± 5	26 ± 6
Physical activity level, metabolic-equivalents h/week	25 ± 32	22 ± 27	20 ± 26	19 ± 25	18 ± 25
Total energy intake, kcal/d	1802 ± 54	1693 ± 508	1694 ± 509	1771 ± 528	1999 ± 581
Alcohol intake, g/d	6 ± 9	3 ± 5	3 ± 5	2 ± 4	2 ± 5
Current smoking	15	11	10	9	12
Comorbid cardiovascular disease	<1	<1	<1	<1	<1
Comorbid type 2 diabetes	<1	<1	<1	<1	<1
Comorbid hypertension	5	5	6	7	9
Comorbid hypercholesterolemia	13	13	14	15	18
Atopic dermatitis cohort (1995)					
No. participants	12,688	12,689	12,689	12,689	12,688
Age, years,* in 1995	42 ± 4	41 ± 5	40 ± 5	40 ± 5	40 ± 5
Empirical Dietary Inflammatory Pattern score	−406 ± 174	−149 ± 45	−9 ± 38	135 ± 48	429 ± 193
White race	98	97	96	95	94
Body mass index, kg/m^2	24 ± 5	25 ± 5	25 ± 6	26 ± 6	28 ± 7
Physical activity level, metabolic-equivalents h/week	25 ± 30	22 ± 29	21 ± 25	19 ± 24	18 ± 25
Total energy intake, kcal/d	1780 ± 532	1721 ± 519	1741 ± 524	1811 ± 547	2006 ± 586
Alcohol intake, g/d	7 ± 10	4 ± 6	3 ± 5	2 ± 5	2 ± 4
Current smoking	13	10	8	8	10
Comorbid cardiovascular disease	<1	<1	<1	<1	<1
Comorbid type 2 diabetes	<1	<1	<1	1	1
Comorbid hypertension	7	7	8	10	13
Comorbid hypercholesterolemia	17	18	20	23	26
Comorbid asthma	8	8	9	9	10

Values are means ± standard deviation or percentages and are standardized to the age distribution of the study population.
*Value is not age-adjusted.

specific year of diagnosis, if available, or the middle value of the given range of diagnosis years.

EDIP scores were calculated as the cumulative average score from all reports up to the start of each 2-year follow-up interval to best represent habitual long-term dietary intake and reduced within-person variation. Due to the high within-person correlations in EDIP scores between adjacent data cycles, we carried forward nonmissing dietary intake data from the previous data cycle to replace any missing data in the next cycle. Covariate data were treated similarly.

We used Cox proportional hazards models to calculate age-adjusted and multivariable-adjusted hazard ratios (HRs) with 95% confidence intervals (CIs) for the association of EDIP quintiles with the risk of developing incident psoriasis, psoriatic arthritis, and atopic dermatitis. We tested the proportional hazards assumption and found it was not violated for any of the outcomes (all $P > .05$). Covariates adjusted for in the main multivariable model (multivariable model 1) included age, race (white vs nonwhite), body mass index (BMI, kg/m^2), smoking (never smoker, past smoker, current smoker [1-14, 15-25, and >25 cigarettes per day]), alcohol intake (0 g/d, 0.1-4.9 g/d, 5.0-9.9 g/d, and ≥10 g/d), calorie consumption (kcal/day in quintiles), and exercise (metabolic equivalents/week in quintiles). The second multivariable model (multivariable model 2) included all of the above covariates in addition to comorbid cardiovascular disease, hypertension, hypercholesterolemia, and type 2 diabetes. For atopic dermatitis, we also used a third multivariable model (multivariable model 3), additionally adjusting for a history of asthma. Further, for atopic dermatitis analyses, we conducted stratified analyses of multivariable models 1 and 2 by comorbid asthma. For each of the

Table II. Risk for psoriasis and psoriatic arthritis according to quintiles of EDIP score in Nurses' Health Study II

EDIP score quintile by disease	No. person-years	No. cases	Age-adjusted HR (95% CI)	MV-adjusted HR1 (95% CI)*	MV-adjusted HR2 (95% CI)*†
Psoriasis					
1	402,913	298	Reference	Reference	Reference
2	403,114	276	0.94 (0.80-1.11)	0.96 (0.81-1.13)	0.96 (0.81-1.13)
3	402,684	269	0.93 (0.78-1.09)	0.95 (0.80-1.12)	0.94 (0.79-1.12)
4	402,999	283	0.98 (0.83-1.15)	0.98 (0.82-1.16)	0.97 (0.81-1.15)
5	402,776	306	1.07 (0.91-1.26)	0.99 (0.83-1.18)	0.97 (0.82-1.16)
P value for trend			.31	.85	.67
Psoriatic arthritis					
1	406,087	44	Reference	Reference	Reference
2	406,250	51	1.20 (0.80 -1.80)	1.23 (0.82-1.84)	1.22 (0.81-1.83)
3	405,855	47	1.13 (0.75-1.71)	1.11 (0.73-1.69)	1.09 (0.71-1.66)
4	406,133	53	1.30 (0.87-1.94)	1.16 (0.76-1.76)	1.13 (0.74-1.71)
5	405,909	67	1.67 (1.14-2.45)	1.22 (0.81-1.83)	1.17 (0.77-1.76)
P value for trend			.007	.54	.70

CI, Confidence interval; *CPD*, cigarettes per day; *EDIP*, Empirical Dietary Inflammatory Pattern; *HR*, hazard ratio; *MV*, multivariable.
*Adjusted for age, race (white vs nonwhite), body mass index (kg/m^2), smoking (never smoker, past smoker, current smoker [1-14 CPD, 15-25 CPD, and >25 CPD]), alcohol intake (0 g/d, 0.1-4.9 g/d, 5.0-9.9 g/d, and ≥10 g/d), calorie consumption (kcal/day in quintiles), and exercise (metabolic equivalents/week in quintiles).
†Also adjusted for comorbid cardiovascular disease, hypertension, hypercholesterolemia, and type 2 diabetes.

analyses, the *P* value for trend was calculated by using the median EDIP score within each quintile.

All statistical analyses were performed with SAS version 9.2 (SAS Institute Inc, Cary, NC). A 2-tailed *P* value <.05 was considered statistically significant.

RESULTS

We had 85,185 participants in the psoriatic disease analysis and 63,443 participants in the atopic dermatitis analysis. Baseline characteristics of participants in the psoriasis analysis according to EDIP quintile are presented in Table I. Participants with higher EDIP scores generally had higher BMI, lower physical activity and alcohol consumption, and higher rates of hypercholesterolemia and hypertension. Similar patterns were seen in the baseline characteristics of participants included in the atopic dermatitis analysis, with the addition that asthma was somewhat more common with higher EDIP scores.

There were 1425 cases of psoriasis during 2,014,487 person-years and 262 cases of psoriatic arthritis during 2,030,235 person-years. There was no association between pro-inflammatory diet and incident psoriasis in the age-adjusted or multivariable-adjusted models (all *P* values for trend >.05; Table II). For psoriatic arthritis, a direct association was seen between high EDIP scores (quintile 5 vs quintile 1, 1.67, 95% CI 1.14-2.45, *P* value for trend = .007) in the age-adjusted analysis, but this association was not significant in multivariable model 1 (quintile 5 vs quintile 1, 1.22, 95% CI 0.81-1.83, *P* value for trend = .54) or multivariable model 2 (quintile 5 vs quintile 1, 1.17, 95% CI 0.77-1.76, *P* value for trend = .70). To assess reasons for attenuation of the association with psoriatic arthritis in the multivariable models, we conducted analyses with EDIP as the exposure, psoriatic arthritis as the outcome, and age plus each individual covariate individually in separate models. We found that BMI was largely responsible for the attenuation. To assess possible effect modification by BMI, we then conducted age- and multivariable-adjusted analyses stratified by BMI (<25 and ≥25 kg/m^2), with further adjustment for BMI as a continuous variable within each stratum. There was no significant difference between the BMI strata (*P* value for interaction = .94); results are shown in Table III.

There were 403 cases of atopic dermatitis during 1,130,810 person-years. EDIP scores were not associated with incident atopic dermatitis in the age- or multivariable-adjusted models (all *P* value for trend >.05; Table IV). Sensitivity analyses with more stringent atopic dermatitis definitions were similar to our primary analysis, and stratified analyses indicated no differential results according to history of asthma (data not shown).

DISCUSSION

In this large retrospective cohort study of US women, we did not find any association between a pro-inflammatory diet and increased risk for incident psoriasis, psoriatic arthritis, or atopic dermatitis. In our age-adjusted model, pro-inflammatory dietary

Table III. Psoriatic arthritis risk in quintiles of baseline EDIP score stratified by BMI in Nurses' Health Study II

EDIP score quintile by BMI	No. person-years	No. cases	Age-adjusted HR (95% CI)	MV-adjusted HR1 (95% CI)*	MV-adjusted HR2 (95% CI)*†
BMI <25 kg/m^2					
1	235,207	12	Reference	Reference	Reference
2	222,998	18	1.67 (0.80-3.46)	1.82 (0.87-3.82)	1.81 (0.86-3.80)
3	208,175	16	1.61 (0.76-3.42)	1.82 (0.84-3.93)	1.79 (0.83-3.87)
4	185,686	13	1.51 (0.68-3.32)	1.74 (0.77-3.94)	1.70 (0.75-3.86)
5	150,558	8	1.18 (0.48-2.91)	1.37 (0.54-3.52)	1.32 (0.51-3.39)
P value for trend			.74	.55	.61
BMI ≥25 kg/m^2					
1	169,242	32	Reference	Reference	Reference
2	181,568	33	0.99 (0.61-1.61)	1.04 (0.64-1.70)	1.04 (0.63-1.69)
3	196,100	31	0.87 (0.53-1.43)	0.90 (0.55-1.50)	0.89 (0.54-1.47)
4	218,890	39	0.99 (0.62-1.58)	0.97 (0.59-1.57)	0.94 (0.58-1.53)
5	253,541	58	1.28 (0.83-1.98)	1.07 (0.67-1.70)	1.02 (0.64-1.63)
P value for trend			.18	.84	.97

BMI, Body mass index; *CI*, confidence interval; *CPD*, cigarettes per day; *EDIP*, Empirical Dietary Inflammatory Pattern; *HR,* hazard ratio; *MV*, multivariable.

*Adjusted for age, race (white vs nonwhite), BMI (kg/m^2), smoking (never smoker, past smoker, current smoker [1-14 CPD, 15-25 CPD, and >25 CPD]), alcohol intake (0 g/d, 0.1-4.9 g/d, 5.0-9.9 g/d, and ≥10 g/d), calorie consumption (kcal/day in quintiles), and exercise (metabolic equivalents/week in quintiles).

†Also adjusted for comorbid cardiovascular disease, hypertension, hypercholesterolemia, and type 2 diabetes.

Table IV. Risk for atopic dermatitis according to quintiles of EDIP score in Nurses' Health Study II

EDIP score quintile	No. person-years	No. cases	Age-adjusted HR (95% CI)	MV-adjusted HR1 (95% CI)*	MV-adjusted HR2 (95% CI)*†	MV-adjusted HR3 (95% CI)*†‡
1	226,264	84	Reference	Reference	Reference	Reference
2	226,318	78	0.93 (0.68-1.27)	0.95 (0.69-1.30)	0.95 (0.69-1.29)	0.95 (0.69-1.30)
3	225,998	76	0.90 (0.66-1.22)	0.93 (0.67-1.28)	0.93 (0.68-1.28)	0.93 (0.67-1.28)
4	226,209	85	1.01 (0.75-1.37)	1.04 (0.76-1.43)	1.05 (0.76-1.44)	1.04 (0.76-1.43)
5	226,021	80	0.95 (0.69-1.29)	0.96 (0.69-1.34)	0.96 (0.69-1.35)	0.96 (0.68-1.34)
P value for trend			.94	.98	.95	.99

CI, Confidence interval; *CPD*, cigarettes per day; *EDIP*, Empirical Dietary Inflammatory Pattern; *HR,* hazard ratio; *MV*, multivariable.

*Adjusted for age, race (white vs nonwhite), body mass index (kg/m^2), smoking (never smoker, past smoker, current smoker [1-14 CPD, 15-25 CPD, and >25 CPD]), alcohol intake (0 g/d, 0.1-4.9 g/d, 5.0-9.9 g/d, and ≥10 g/d), calorie consumption (kcal/day in quintiles), and exercise (metabolic equivalents/week in quintiles).

†Also adjusted for comorbid cardiovascular disease, hypertension, hypercholesterolemia, and type 2 diabetes.

‡Also adjusted for comorbid asthma.

patterns were associated with psoriatic arthritis, but the HR was attenuated and no longer statistically significant once we adjusted for important confounders, particularly BMI. For atopic dermatitis, no significant relationship was found with pro-inflammatory diet. Though we found no convincing evidence for an association with EDIP score for any of the investigated diseases, the results followed an internal pattern consistent with our hypotheses that higher EDIP scores would have more of an association with psoriatic disease than with atopic dermatitis.

Previous studies have shown robust associations between psoriatic disease and inflammatory bowel disease, cardiovascular disease, and metabolic syndrome.[50-55] Like psoriasis and psoriatic arthritis, these diseases share elevated serum inflammatory biomarkers, such as CRP, TNF-α, and IL-6.[38-41] Recently, an association between the Mediterranean diet and psoriasis was assessed in the French NutriNet-Santé study.[56] Researchers found that low adherence to a Mediterranean diet, which has pro-inflammatory effects similar to those measured with the EDIP,[25-28] was associated with increased odds of

severe but not mild psoriasis. Those results are consistent with evidence showing that a diet with anti-inflammatory properties has beneficial health effects, such as reduced systemic inflammation,[57] metabolic syndrome,[58,59] and cardiovascular events.[60] The divergent results between that study and ours are likely related to methodologic differences, specifically that psoriasis was classified by severity in the NutriNet-Santé study, whereas we examined the risk for incident psoriasis overall.[56] The inflammatory potential of the diet might not be as crucial for psoriatic disease incidence compared with other lifestyle risks and genetic factors.[61,62] Currently, the strongest recommendation for dietary change in psoriatic disease is weight loss.[63]

We did not find evidence of a relationship between pro-inflammatory diet and incident atopic dermatitis which, like psoriasis, has a strong genetic component.[64] Furthermore, unlike psoriasis and psoriatic arthritis, atopic dermatitis is not as strongly associated with T_H1-associated inflammation,[65] though new evidence is emerging of associations between atopic dermatitis, obesity, and cardiovascular risk factors.[11,66] In addition, in 1 study, CRP levels were found to be elevated in patients with more severe atopic dermatitis.[67] Overall, though, we found no evidence that a diet with pro-inflammatory properties, in particular one associated with increased levels of IL-6, CRP, and TNF-α, increased atopic dermatitis risk. It is possible that a dietary index associated more with T_H2 inflammation would yield different results.

There are several strengths and limitations to our study. We had a large sample size and prospectively collected dietary and psoriatic disease data, and we were able to adjust for important confounders. However, given that participants were US women, our results might not be generalizable to other populations. Further, we examined adult-onset atopic dermatitis, and so, our results might not be applicable for more classic, childhood-onset disease. Questionnaire-based diagnoses, though validated, are subject to misclassification, as is the year of atopic dermatitis diagnosis. It is likely that dilution of the case pool with false-positive cases would bias our results towards the null. Most NHS-II participants with psoriasis and atopic dermatitis have mild disease[48]; longitudinal investigation of an inflammatory diet's impact on disease severity over time would be interesting but is not possible within our data set. Last, as in any observational study, there might be residual confounding by unmeasured factors.

In conclusion, a pro-inflammatory diet did not substantially modify the risk for psoriasis, psoriatic arthritis, or atopic dermatitis among women in this study. Although a pro-inflammatory diet might be associated with other health risks,[68,69] we found no reason to counsel patients about the potential impact of pro-inflammatory diets on psoriatic disease or atopic dermatitis.

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