PRO-INFLAMMATORY DIET AND RISK OF INCIDENT GOUT: 3 PROSPECTIVE COHORT STUDIES OF US MEN AND WOMEN

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Background: Emerging evidence suggests inflammation may drive progression from hyperuricemia to clinical gout, but the role of extrinsic, modifiable sources of chronic inflammation, such as diet, on gout risk is unknown. Notably, greater dietary inflammatory potential has been independently associated with increased risk of incident cardiovascular disease (CVD)1 and type 2 diabetes (T2D).2,3

Objectives: Prospectively examine the relation between dietary inflammatory potential and risk of gout in three large cohorts of US women and men over 30 years.

Methods: Ascertainment: The ACR survey criteria for gout for several decades,3 we studied gout risk among 164,090 women from Nurses Health Study I (1986-2004) and II (1989-2017) and 40,802 men from Health Professionals Follow-up Study (1986-2016), free of gout at baseline. Diet intake and covariates were assessed by validated questionnaires every 4 years. Inflammatory potential of diet was evaluated using a food-based empirical dietary index of inflammatory potential score (EDIP) pre-defined based on circulating levels of IL-6, C-reactive protein, adiponectin, and TNFα.4,5

We assigned an EDIP score for each participant, adjusted for total energy intake, as alcohol has anti-inflammatory properties4, but is associated with a higher gout risk, particularly beer.5

Results: We documented 2,874 incident gout cases over 5,124,940 person-years of follow-up. In pooled multi-variable-adjusted analyses, those in the highest quintile of EDIP had 59% higher gout risk (multivariable RR 1.59; 95% CI 1.41–1.79), compared with the lowest (Table 1). This remained positive with further adjustment for BMI, a likely causal intermediate (RR 1.72, 11 to 1.42), and was stronger among non-drinkers (RR 2.37, 1.58 to 2.56) than drinkers (RR 1.57, 1.38 to 1.78) (Table 1).

| G1: | Q1: lowest | Q2: | Q3: | Q4: | Q5: highest | P for trend |
|-----|------------|-----|-----|-----|------------|------------|
| Overall | 473 | 493 | 530 | 623 | 755 | 0.015 |
| N cases | 1024,571 | 1025,618 | 1025,284 | 1024,779 | 1024,688 | 0.003 |
| Person-years | 100 (Ref) | 105 (0.92, 1.13 (0.39, 1.33 (1.18, 1.64 (1.46, 0.001 |
| Adjusted RR | 1.00 (0.92, | 1.12 (0.98, | 1.31 (1.16, | 1.59 (1.14, | 0.001 |
| MV-Adjusted*** | 1.19) | 1.27) | 1.31) | 1.14) | 1.16) | 0.001 |
| MV + BMI | | | | | | 0.001 |
| No Alcohol Use | | | | | | 0.001 |
| N cases | 256 | 58 | 84 | 143 | 251 | 0.009 |
| Person-years | 118,301 | 189,938 | 249,389 | 313,511 | 396,080 | 0.001 |
| MV-Adjusted*** | 1.10 (0.88, 1.16 (1.01, 1.12) | 1.13 (1.12) | 1.14 (1.12) | 0.001 |
| Alcohol Use | | | | | | 0.001 |
| N cases | 447 | 435 | 446 | 480 | 504 | 0.001 |
| Person-years | 965,271 | 835,680 | 775,895 | 711,267 | 628,609 | 0.001 |
| MV-Adjusted*** | 1.19 (1.13) | 1.29 (1.25) | 1.50 (1.50) | 0.001 |

*Multivariable (MV) models adjusted for age (months), White race, smoking, menopause (women only), hormone use (women only), physical activity, history of hypertension, and diuretic use. **MV + BMI models additionally adjusted for BMI (a likely causal intermediate)
Conclusion: Habitual pro-inflammatory dietary pattern was independently associated with higher risk of incident gout in these prospective cohorts, even beyond the pathway through adiposity. Our findings support a role for chronic inflammation in development of gout, similar to CVD and T2D. Adhering to a diet with lower inflammatory potential may modulate systemic inflammation, potentially reducing gout risk and these life-threatening comorbidities.

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THE EFFECT OF UV-B RADIATION EXPOSURE ON THE RISK OF DEVELOPING RHEUMATOID ARTHRITIS
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Background: UV-B radiation has known immunomodulatory properties, but to what extent UV-B radiation exposure might affect the occurrence of rheumatoid arthritis (RA) has been relatively little studied, and with partially contradictory results.

Objectives: To investigate the association between sun- and travel habits, as proxy markers for UV-B radiation exposure, and risk of incident RA, overall and by RA subtype.

Methods: We performed a matched case-control study of 1151 incident cases with new-onset RA and 2374 population controls from the Swedish Epidemiology Investigation of Rheumatoid Arthritis (EIRA) study, recruited between 2006 and 2017. The association between sunbathing frequency, solarium use, and frequency of travels to sunnier countries than Sweden (exposures) and risk of RA (outcome) were assessed as odds ratios (OR) with 95% confidence intervals (CI) through logistic regression, and adjusted for age, sex, residential region, year of study entry, body mass index, education, income, smoking and alcohol consumption. We further assessed effect modification by self-reported skin type, income and education, and by rheumatoid factor (RF) status.

Results: Overall, the frequency of sunbathing, and solarium use, were similar among RA cases and controls: ‘never doing sunbathing’ amongst RA cases vs. controls: 22% vs. 21%, ‘sunbathing daily when possible’: 10% vs. 12%, and solarium use 13% vs. 12%. The proportion of ‘not travelled abroad to a sunnier country than Sweden during the past 5 years’ was higher for RA cases than controls: 27% vs. 23%, and ‘travelling abroad more than once a year’ was less common among RA cases: 15% vs. 20%.

Sunbathing was not linked to risk of RA (OR 0.91, 95% CI 0.69-1.20), nor was solarium (OR 0.89, 95% CI 0.65-1.35). Stratification by skin type revealed no major effect modification, nor did stratification by RF status. In contrast, frequency of travel to sunnier countries than Sweden was inversely associated with RA risk comparing the most to least frequent travelers (OR 0.68, 95% CI 0.54-0.87). When stratified by educational level, this association was confined to individuals with medium (OR 0.69, 95% CI 0.46-0.98) or high (OR 0.60, 95% CI 0.40-0.89) and absent among subjects with low education (OR 1.10, 95% CI 0.56-1.99). No such interaction was observed between travel habits and income.

Conclusion: Proxy markers for UV-B exposure (sunbathing frequency and solarium use within the past five years) do not seem to be strong risk factors for RA. Frequency of travels abroad was inversely associated to RA risk. The nature behind this association remains unclear.

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EXPOSURE TO ENVIRONMENTAL AIR POLLUTANTS AS A RISK FACTOR FOR PRIMARY SJÖGREN’S SYNDROME: A POPULATION-BASED COHORT STUDY
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Background: Recent studies suggest that air pollution may play a role in autoimmune diseases. However, few of them report the correlation between air pollution and primary Sjögren’s syndrome (pSS).

Objectives: We sought to determine whether people exposed to environmental fine particulate air pollution of air pollution have a higher risk of developing pSS.

Methods: We performed a matched population-based cohort study from the National Health Insurance Research Database (NHIRD) of Taiwan’s population, using the international Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) to categorize each disease diagnosis. Air pollution data on Nitric oxide (NO), methane (CH4), and carbon monoxide (CO) were obtained from the Taiwan Air Quality-Monitoring Database (TAQMD), where daily air pollution data from community-based monitoring sites (78 sites since 1993) was available on a real-time basis. We followed up from January 1st, 1998 to the endpoint of SS diagnosis or to December 31, 2011. The daily average air pollutant concentrations were divided into 4 quartile-based groups (Q1-Q4). The incidence rate, hazard ratios (HRs), as well as 95% confidence intervals for pSS, were stratified by the quartiles of air pollutant concentration, and calculated with a Cox proportional regression model. Finally, Ingenuity Systems Pathway Analysis (IPA) was conducted to identify activated pathways among air way epithelial cells exposed to airborne coarse, fine, and ultrafine particles, and parotid gland tissues from pSS patients using Z-score visualization.

Results: A total of 200 patients were diagnosed with SS. The mean age of patients with pSS was 53.1 years. The incidence of pSS was 0.11%. With the increase in exposure concentrations of nitrogen dioxide, methane, and carbon monoxide (from Q1 to Q4), the incidence rate for pSS of per 1000 person-years increased from 0.7 to 1.19, from 0.93 to 2.14, and from 0.57 to 1.06, respectively. Moreover, compared with Q1, the adjusted HR in Q4 after adjusting for age, gender, monthly income and urbanization levels increased to 1.86, 2.21 and 2.04, respectively. IPA analyses suggested that the underlying cellular mechanisms involved up-regulation of chronic inflammatory pathways including fibrosis signaling pathway.

Conclusion: Exposure to air pollutants, specifically NO, CH4, and CO, was associated with SS development, mostly driven by fibrotic signaling cascades occurring during chronic inflammation.

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DRUG COST FOR BIOLOGIC AND TARGETED SYNTHETIC DMARDs FOR RHEUMATOID ARTHRITIS PATIENTS IN NORWAY FROM 2010 TO 2019 - A COUNTRY WITH A NATIONAL TENDER SYSTEM FOR DRUG PRESCRIPTION
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