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.

REFERENCES:
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Disclosure of Interests: J. Hagman: None declared, B. Delcoigne: None declared, L. Klarekskog: None declared, L. Alfredsson: None declared, J. Asking: None declared.

Background: UV-B radiation has known immunomodulatory properties, but to what extent UV-B radiation exposure might affect the occurrence of rheumatoid arthritis (RA) is 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 Epidemiological Investigation of Rheumatoid Arthritis (EIRA) study, recruited between 2006 and 2017. The association between sunbathing frequency, solarium use, and frequency of travel 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) serostatus.

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 those in the general population: 15% vs. 20%.

Table 1. RA cases and controls with adjusted odds ratios and confidence intervals for overall risk of RA and by RA serostatus.

<table>
<thead>
<tr>
<th>Exposure variable</th>
<th>RA cases</th>
<th>Controls</th>
<th>RF+</th>
<th>RF-</th>
<th>All RA</th>
<th>RF+</th>
<th>RF-</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunbathinga Never</td>
<td>249</td>
<td>495</td>
<td>161</td>
<td>85</td>
<td>ref</td>
<td>ref</td>
<td>ref</td>
</tr>
<tr>
<td>At least once a month</td>
<td>398</td>
<td>844</td>
<td>265</td>
<td>124</td>
<td>(0.86-1.29)</td>
<td>1.07</td>
<td>0.98</td>
</tr>
<tr>
<td>At least once a week</td>
<td>376</td>
<td>751</td>
<td>239</td>
<td>130</td>
<td>(0.90-1.38)</td>
<td>1.07</td>
<td>1.21</td>
</tr>
<tr>
<td>Daily</td>
<td>120</td>
<td>278</td>
<td>75</td>
<td>43</td>
<td>(0.69-1.20)</td>
<td>0.91</td>
<td>0.96</td>
</tr>
<tr>
<td>Travelb Never</td>
<td>314</td>
<td>537</td>
<td>208</td>
<td>103</td>
<td>ref</td>
<td>ref</td>
<td>ref</td>
</tr>
<tr>
<td>Seldom</td>
<td>294</td>
<td>568</td>
<td>193</td>
<td>97</td>
<td>0.98</td>
<td>0.98</td>
<td>0.98</td>
</tr>
<tr>
<td>Once a year</td>
<td>359</td>
<td>805</td>
<td>227</td>
<td>121</td>
<td>(0.79-1.21)</td>
<td>1.07</td>
<td>1.10</td>
</tr>
<tr>
<td>More than once a year</td>
<td>176</td>
<td>462</td>
<td>112</td>
<td>61</td>
<td>(0.54-0.87)</td>
<td>0.68</td>
<td>0.70</td>
</tr>
<tr>
<td>Never</td>
<td>991</td>
<td>2083</td>
<td>634</td>
<td>336</td>
<td>ref</td>
<td>ref</td>
<td>ref</td>
</tr>
<tr>
<td>Once per year or more</td>
<td>153</td>
<td>290</td>
<td>107</td>
<td>46</td>
<td>(0.85-1.35)</td>
<td>1.07</td>
<td>1.11</td>
</tr>
</tbody>
</table>

OR = adjusted odds ratio, CI = confidence interval, N = number of participants, RA = rheumatoid arthritis, RF = reference, RF+ = rheumatoid factor. A frequency of sunbathing if the weather invites it (e.g. monthly) and frequency of travel to sunnier countries than Sweden in the last 5 years. CI: Confidence intervals.

Disclosure of Interests: K. S. K. Ma1, L. T. Wang2, 3National Taiwan University, Taipei, Taiwan, ROC, Department of Life Science, T aipei, Taiwan, Republic of China; 4National Taiwan University Hospital, Department of Obstetrics & Gynecology, Taipei, Taiwan, Republic of China.

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 have a higher risk of developing pSS.

Methods: We performed a prospective 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 airway 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 occurred during chronic inflammation.


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