Epidemiology and public health

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Background: There is some evidence from epidemiological studies suggesting that CS and glucosamine could play a role in cardiovascular disease (CVD) prevention (1-4). Studies to date have included prevalent users, therefore a bias that overestimates protection cannot be excluded.

Objectives: To test the hypothesis that chondroitin sulphate (CS) or glucosamine reduce the risk of acute myocardial infarction (AMI).

Methods: Case-control study nested in a primary cohort composed of patients included 40 to 99 years, with at least one year of follow-up in the BIFAP database during the 2002-2015 study period. From this cohort of patients, we identified incident cases of AMI and randomly selected five controls per case, matched by exact age, gender, and index date. Adjusted odds ratios (AOR) and their corresponding 95% confidence interval (CI) were calculated through a conditional logistic regression. Only new users of CS or glucosamine were considered. The mean age was 67.0 (SD 13.4) years and 71.75% were male, in this group.

Results: A total of 23,585 incident cases of AMI and 117,405 controls were included. The mean age was 670 (SD 13.4) years and 71.7% were male, in both groups. 558 (23.7%) cases and 3,082 (2.62%) controls used or had used CS. The current use of CS was associated with a lower risk of AMI (AOR 0.57; 95% CI 0.48-0.70) and high cardiovascular risk (AOR 0.48, 95% CI 0.27-0.83), but not in those at low risk (AOR=1.11; 95% CI 0.48-2.56). In contrast, the current use of glucosamine was not associated with increased or decreased risk of AMI (AOR=0.86; CI95% 0.66-1.08).

Conclusion: Our results support a cardioprotective effect of CS, while no effect was observed with glucosamine. The highest protection was found among subgroups at higher cardiovascular risk.

REFERENCES:
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:
[1] Li et al. J Amer Coll Cardiology (2020) PMID 33153576
[4] Tabung et al. PMID 27358416
[5] Choi et al. PMID 15094272

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OP0236
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 Epidemiological 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) 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 among RA cases and controls: ‘never doing sunbathing’ amongst RA cases 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 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>OR† for RA (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunbathing‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>249</td>
<td>495</td>
<td>161</td>
<td>85</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>1.05</td>
</tr>
<tr>
<td>At least once a week</td>
<td>376</td>
<td>751</td>
<td>239</td>
<td>130</td>
<td>(0.85-1.25)</td>
</tr>
<tr>
<td>Daily</td>
<td>120</td>
<td>278</td>
<td>75</td>
<td>43</td>
<td>(0.69-1.20)</td>
</tr>
<tr>
<td>Travel§</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never Seldom</td>
<td>314</td>
<td>537</td>
<td>208</td>
<td>103</td>
<td>ref</td>
</tr>
<tr>
<td>Once a year</td>
<td>359</td>
<td>805</td>
<td>227</td>
<td>121</td>
<td>0.82</td>
</tr>
<tr>
<td>More than once a year</td>
<td>176</td>
<td>463</td>
<td>112</td>
<td>61</td>
<td>(0.63-1.02)</td>
</tr>
<tr>
<td>Never</td>
<td>991</td>
<td>2083</td>
<td>634</td>
<td>336</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>1.07</td>
</tr>
</tbody>
</table>

OR = adjusted odds ratio, CI = confidence interval, N = number of participants, RA = rheumatoid arthritis, ref = reference, RF = rheumatoid factor. A frequency of sunbathing if the weather invites to spend time outdoors and the sun is shining. A frequency of travels to sunnier countries than Sweden in the last 5 years. * Adjusting for age, sex, region, index year, BMI, smoking, alcohol consumption, education level and income. ‡ < 1% of data was missing for all variables.

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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OP0237
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 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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OP0238
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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