Disclosure of Interests: Atul Deodhar Speakers bureau: Novartis and Pfizer, Consultant of: Novartis, Pfizer, AbbVie, Eli Lilly, UCB Pharma, GlaxoSmithKline, Galapagos, Janssen, Boehringer Ingelheim and Celgene, Grant/research support from: Novartis, Pfizer, AbbVie, Eli Lilly, UCB Pharma, GlaxoSmithKline, R Ranza Speakers bureau: AbbVie, Janssen, Lilly, Novartis, and Pfizer, Consultant of: AbbVie, Janssen, Lilly, Novartis, and Pfizer, Grant/research support from: AbbVie, Janssen, Fabiana Ganz Shareholder of: AbbVie, Employee of: AbbVie, Tianming Gao Shareholder of: AbbVie, Employee of: AbbVie, Jaclyn Anderson Shareholder of: AbbVie, Employee of: AbbVie, Andrew Ostor Consultant of: AbbVie, BMS, Roche, Janssen, Lilly, Novartis, Pfizer, UCB, Gilead, and Paradigm
DOI: 10.1136/annrheumdis-2021-eular.439

Epidemiology and public health

OP0234 RISK OF ACUTE MYOCARDIAL INFARCTION AMONG NEW USERS OF CHONDROITIN SULPHATE: A NESTED CASE-CONTROL STUDY

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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 aged 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.

Results: A total of 23,585 incident cases of AMI and 117,405 controls were included. The mean age was 67.0 (SD 13.4) years and 71.75% were male, in both groups. 558 (2.37%) 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: 0.46-0.72) and disappeared after discontinuation (recent and past users). The reduced risk among current users was observed in both short-term (<365 days AOR 0.58; 95%CI: 0.45-0.75) and long-term users (>364 days AOR 0.56; 95%CI 0.36-0.87), in both sexes (men, AOR=0.52; 95%CI:0.38-0.70; women, AOR=0.65; 95%CI: 0.46-0.91), in individuals over or under 70 years of age (AOR=0.54; 95%CI:0.38-0.77, and AOR=0.61; 95%CI:0.45-0.82, respectively) and in individuals at intermediate (AOR=0.65; 95%CI:0.48-0.91) 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 either 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.

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Disclosure of Interests: Ramón Mazzucchelli Speakers bureau: UCB, Lilly, Grant/research support from: Pfizer, Roche, Amgen, Sara Rodríguez-Martin: None declared, Alberto García-Vadillo: None declared, Miguel Gil: None declared, Antonio Rodríguez-Miguel: None declared, Diana Barreira-Hernández: None declared, Alberto García-Lledó: None declared, Francisco de Abajo: None declared

DOI: 10.1136/annrheumdis-2021-eular.123

OP0235 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

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: Ascertaining the ACR survey criteria for gout for several decades,³ we studied gout risk among 164,090 women from Nurses Health Study I (1986-2016) and II (1989-2017) and 40,598 men from Health Professionals Follow-up Study (1986-2016), free of gout at baseline. Dietary 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 TNF α .⁴

We assigned an EDIP score for each participant, adjusted for total energy take, and prospectively examined the association between quintiles of EDIP score and incident gout, adjusting for potential confounders. We also stratified by alcohol intake, as alcohol has anti-inflammatory properties,⁴ 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 multivariable-adjusted analyses, those in the highest EDIP quintile 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 1.27, 1.12 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).

Table 1. Risk Ratio (95% CI) of Gout According to Quintiles of Inflammatory Diet Score, Overall and by Alcohol Use

	Q1: lowest	Q2	Q3	Q4	Q5: highest	P for trend
Overall						
N cases	473	493	530	623	755	
Person-years	1,024,571	1,025,618	1,025,284	1,024,779	1,024,688	
Age-adjusted RR	1.00 (Ref)	1.05 (0.92, 1.19)	1.13 (0.99, 1.27)	1.33 (1.18, 1.50)	1.64 (1.46, 1.84)	<0.001
MV-Adjusted* RR	1.00 (Ref)	1.04 (0.92, 1.18)	1.12 (0.98, 1.26)	1.31 (1.16, 1.48)	1.59 (1.41, 1.79)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.00 (0.88, 1.13)	1.03 (0.91, 1.17)	1.16 (1.02, 1.31)	1.27 (1.12, 1.42)	<0.001
No Alcohol Use						
N cases	26	58	84	143	251	
Person-years	118,301	189,938	249,389	313,511	396,080	
MV-Adjusted* RR	1.00 (Ref)	1.31 (0.82, 2.08)	1.37 (0.88, 2.13)	1.80 (1.18, 2.74)	2.37 (1.58, 2.56)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.28 (0.80, 2.03)	1.32 (0.85, 2.05)	1.61 (1.06, 2.45)	1.85 (1.23, 2.79)	<0.001
Alcohol Use						
N cases	447	435	446	480	504	
Person-years	906,271	835,680	775,895	711,267	628,609	
MV-Adjusted* RR	1.00 (Ref)	1.04 (0.91, 1.19)	1.13 (0.99, 1.29)	1.31 (1.15, 1.50)	1.57 (1.38, 1.78)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.00 (0.88, 1.14)	1.05 (0.92, 1.20)	1.17 (1.03, 1.33)	1.28 (1.12, 1.46)	<0.001

*Multivariable (MV) models adjusted for age (month), 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 CVD¹ and T2D.² 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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Disclosure of Interests: Natalie McCormick: None declared, Chio Yokose: None declared, Leo Lu: None declared, Amit Joshi: None declared, Hyon Choi Consultant of: Ironwood, Selecta, Horizon, Takeda, Kowa, Vaxart, Grant/research support from: Ironwood, Horizon

DOI: 10.1136/annrheumdis-2021-eular.3430

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 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

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

Exposure variable	N				OR* for RA (95% CI)		
	RA cases	Controls	RF+	RF-	All RA	RF+	RF-
Sunbathing^a							
Never	249	495	161	85	ref	ref	ref
At least once a month	398	844	265	124	1.05 (0.85-1.29)	1.07 (0.84-1.36)	0.98 (0.72-1.34)
At least once a week	376	751	239	130	1.11 (0.90-1.38)	1.07 (0.83-1.37)	1.21 (0.88-1.66)
Daily	120	278	75	43	0.91 (0.69-1.20)	0.86 (0.62-1.20)	0.96 (0.64-1.46)
Travel^b							
Never	314	537	208	103	ref	ref	Ref
Seldom	294	568	193	97	0.98 (0.79-1.21)	0.98 (0.77-1.25)	0.98 (0.71-1.35)
Once a year	359	805	227	121	0.82 (0.67-1.01)	0.80 (0.63-1.02)	0.83 (0.61-1.13)
More than once a year	176	463	112	61	0.68 (0.54-0.87)	0.68 (0.51-0.91)	0.70 (0.48-1.01)
Solarium^c							
Never	991	2083	634	336	ref	ref	Ref
Once per year or more	153	290	107	46	1.07 (0.85-1.35)	1.08 (0.83-1.40)	1.11 (0.77-1.59)

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 it? b Frequency of travels to a country sunnier than Sweden in the last 5 years? c Frequency of solarium use in the last 5 years? *Adjusting for age, sex, region, index year, BMI, smoking, alcohol consumption, education level and income. <4 % of data was missing for all variables.

controls: 27% vs. 23%, and 'travelling abroad more than once a year' was less common among RA cases: 15% vs. 20%.

Sunbathing frequency was not linked to risk of RA (OR 0.91, 95% CI 0.69-1.20), nor was solarium use (OR 1.07, 95% CI 0.85-1.35). 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 0.48-0.98) or high (OR 0.60, 95% CI 0.50-0.91) 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.

Disclosure of Interests: Johanna Hagman: None declared, Bénédicte Delcoigne: None declared, Lars Klareskog: None declared, Lars Alfredsson: None declared, Johan Askling Grant/research support from: JA acts or has acted as PI for agreements between Karolinska Institutet and the following entities, mainly in the context of the ARTIS national safety monitoring programme of immunomodulators in rheumatology: Abbvie, BMS, Eli Lilly, Merck, MSD, Pfizer, Roche, Samsung Bioepis, Sanofi, and UCB Pharma

DOI: 10.1136/annrheumdis-2021-eular.1430

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 of air pollution have a higher risk of developing pSS.

Methods: We performed a retrospective 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 occurred during chronic inflammation.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3983

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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