Conclusions: Individuals at risk of RA who have developed systemic autoimmunity associated with RA and/or symptoms, have enrichment of *Prevotella spp* in comparison with healthy controls. Our findings support the hypothesis of a causal role of *Prevotella spp* in the development of RA, which could lead to future attempts to interfere with its intestinal colonisation during the preclinical stages of disease.

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SAT0678 SEASONALITY OF NEW EARLY INFLAMMATORY ARTHRITIS CASES: PRELIMINARY RESULTS FROM THE CANADIAN EARLY ARTHRITIS COHORT

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Background: Disease clustering suggests a possible environmental cause. However, confirming the causes of time-place clustering is challenging. Rheumatoid arthritis (RA) is an autoimmune disease that may be triggered by environmental factors: viral or bacterial infections can lead to RA via molecular mimicry, epitope spreading, B-cell-mediated pathway, bystander activation and via superantigens. Putative infections may also cluster.

Objectives: To study the seasonal distribution of early inflammatory arthritis (IA) onsets in a Canadian sample, and potential relationships with influenza (flu) outbreaks.

Methods: Data from an incidence cohort of adults (>18 years) with early IA with <13 months symptom duration from the Canadian Early Arthritis Cohort (CATCH) enrolled between January 2007 – January 2017. Patient-reported date of symptom onset and physician-reported date of IA onset were used to estimate the monthly frequency of early inflammatory arthritis onsets. Monthly flu case frequencies from September 2010 – December 2016 were retrieved from the Public Health Agency of Canada's FluWatch national surveillance system of confirmed influenza A and B cases. Time-series Poisson regression analyses were performed to assess the non-random covariance of IA and flu with different hypothesised time lags.

Results: A total of 2262 with early IA were included. Patient-reported IA onset was more frequent in winter months compared to other seasons ($p=0.002$), with peaks in January (figure 1). The ten-year aggregated data also showed January peaks of IA onset. There was a positive linear correlation between the number of IA and influenza A onsets ($r=0.23$, $p=0.045$), and almost no relationship between the number of IA and influenza B cases ($p=0.87$) (figure 2).

Abstract SAT0678 – Figure 1

Abstract SAT0678 – Figure 2

Conclusions: Our study provides novel information on the increased onset of IA in relation to seasonal influenza patterns in a Canadian population and adds to the existing literature supporting viral infections as a possible trigger for RA onset.

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SAT0679 CIGARETTE SMOKING AND RISK OF BEHCET'S DISEASE: A CASE-CONTROL STUDY

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Background: Behcet's disease (BD) is a chronic inflammatory multisystem disease characterised by recurrent oral and genital aphthous ulcers, uveitis and skin lesions. Smoking play an important role in the development of inflammatory diseases.¹

Objectives: BD is an inflammatory disease and smoking may have a role in its triggering. Although few studies reported association between smoking and clinical manifestations of BD, to the best of our knowledge, there is no research on the risk of developing BD in smokers.²⁻⁴ This case-control study was conducted to investigate the association between smoking and the risk of BD.

Methods: We included 192 patients with BD and 822 healthy siblings of patient with BD and 373 age and sex matched healthy unrelated persons as control groups (table 1). Written informed consent was obtained from all the participants. Demographic data and smoking history of patients and their siblings were obtained by direct and in some cases by telephone interview with the participants. Demographic data and smoking history of healthy controls were obtained by direct interview. Current and former smokers were classified together as ever smokers for the purposes of this analysis. We carried out multivariate analyses with a logistic regression model with BD as the main outcome variable and smoking history as the main predictor variable. Models were adjusted for age, gender, marital status, educational status and pack-years.

Results: The patients with BD were more often ever smokers than healthy siblings of patients and healthy unrelated controls (table 1). However, pack-years of smoking in control groups were significantly more than the BD group. By multivariate logistic regression and after adjustment for age, sex, marital status and educational status (model 1), ever smoking was significantly associated with an increased risk of BD compared with never smoking. In comparison with healthy siblings and healthy unrelated persons who never smoked, the relative risk (RR) of developing BD was 1.43 and 1.51, respectively. After including pack year in multivariate analysis (model 2), the RR of developing BD in ever smokers increased to 2.23 and 2.01, respectively.

Abstract SAT0679 – Table 1. Demographic characteristics of participants

Variables	BD group (n=192)	Healthy sibling group (n=822)	P-value	Healthy unrelated control group (n=373)	P-value
Age (years)	38.9±10.6	39.3±11.3	NS	37.9±9.7	NS
Gender:			0.001		NS
Male	118 (61.5)	399 (48.5)		228 (61.1)	
Female	74 (38.5)	423 (51.5)		145 (38.9)	
Smoke status			0.01		0.025
Never-smoker	141 (73.4)	673 (81.9)		302 (81)	
Current	43 (22.4)	138 (16.8)		58 (15.5)	
smokers	8 (4.2)	11 (1.3)		13 (3.5)	
Past smokers	51 (26.6)	149 (18.1)		71 (19)	
Ever smokers			0.003		0.007
Pack-years of smoking	11.19±8.8	17.79±11.4		14.19±10.7	

Qualitative and quantitative variables were displayed as numbers (percentages) and means ±SD, respectively.
BD: Behcet's disease; NS: non-significant

No significant differences were observed in the clinical manifestations of BD patients in ever smokers and never smokers. However, disease activity, at disease presentation and last visit in ever smokers was significantly more than never smokers.

Conclusions: Our data suggest that smoking is a significant risk factor for BD and genetic factors may play an important role in the developing of BD in smokers. We also show a particularly strong relationship between smoking with BD activity and response to treatment.

Conflict of interest: We have no potential conflict of interest.

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SAT0680 THE DEVELOPMENT AND VALIDATION OF INTERSTITIAL LUNG DISEASE PREDICTION MODELS IN THREE INTERNATIONAL MIXED CONNECTIVE TISSUE DISEASE COHORTS: THE NORWEGIAN MCTD COHORT, THE HUNGARIAN MCTD COHORT AND THE MCTD COHORT FROM MINNESOTA, US

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Background: Mixed Connective Tissue Disease (MCTD) is characterised by the presence of anti-RNP antibodies with clinical features also found in SSc, SLE and IIM. There is an ongoing debate of MCTD's position as a CTD. A substantial proportion of MCTD patients develop Interstitial Lung Disease (ILD).

Objectives: This study was conducted with the aims to explore the value of MCTD diagnosis and risk assessment by developing and validating ILD prediction models.

Methods: Multivariable logistic regression analyses were performed in 3 international MCTD cohorts. ILD prediction model development from clinical and laboratory parameters was performed in the Norwegian MCTD cohort (n=119). External validation of the models were performed in the Hungarian MCTD cohort (n=196) and the MCTD cohort from Minnesota, US (n=50). ILD was diagnosed by chest CT examination.

Results: The cohort characteristics are presented in table 1. An ILD prediction model including Pulmonary Function Test (PFT) results (table 2) and excluding PFT results was developed. The Hosmer-Lemeshow goodness of fit test (HL test) was .31 and .71 and the ROC was .83 and .78 respectively. The ILD prediction model including DLCO <60% was validated in the Hungarian MCTD cohort and showed good calibration and discrimination (HL test=0.95 and ROC=0.82). The ILD prediction model excluding PFT results showed good calibration and discrimination in both the Hungarian MCTD cohort (HL test=0.72 and ROC=0.80) and the MCTD cohort from Minnesota (HL test=0.96 and ROC=0.67).

Abstract SAT0680 – Table 1. Characteristics of the three MCTD cohorts

Characteristics	Norwegian MCTD cohort (n=119)	Hungarian MCTD cohort (n=196)	Minnesota MCTD cohort (n=50)	P- value
Male sex, N (%)	32 ⁽²⁴⁾	10 (5)	8 (16)	p<0.001
Age at diagnosis, yrs	34 (10)	36 (9)	48 (16)	p<0.001
Age at CT, yrs	44 (14)	41 (11)	48 (14)	p=0.001
ILD, N(%)	52 ⁽³⁹⁾	116(59)	14 ⁽²⁸⁾	p<0.001
DLCO<60% pred	28 (23)	67 (35)	NA	p=0.025
FVC<75% pred	18 (14)	97 (49)	NA	p<0.001

Abstract SAT0680 – Table 2. ILD prediction model including PFT results

	Univariable			Multivariable		
	HR	95% CI	P value	HR	95% CI	P value
Sclerodactily	2.0	0.94–4.0	0.071			
RNP>200	3.0	1.5–7.1	0.003	4.4	1.7–11.1	0.002
SR>30	2.3	1.0–5.0	0.043			
Male sex	1.6	0.71–3.5	0.268			
Never arthritis	5.4	2.1–13.6	<0.001	4.1	1.4–11.6	0.008
Pericarditis	2.2	0.8–6.1	0.117			
Agegroup at CT						
<25 years						
26–35 years	1.0	0.2–4.5	0.953	1.8	0.3–9.8	0.504
36–45 years	2.6	0.6–	0.213	4.8	0.9–25.5	0.065
46–55 years	1.7	11.7.	0.513	2.9	0.5–14.7	0.210
56–65 years	2.3	4–7.4.	0.289	3.7	0.6–23.9	0.167
>65 years	6.0	5–10.8	0.048	13.0	1.6–	0.017
		1.0–35.4			107.3	

Conclusions: The cohorts have different characteristics. Despite these differences the ILD prediction models developed in the Norwegian MCTD cohort have shown external validity when assessed in the Hungarian MCTD cohort and the MCTD cohort from Minnesota. Risk factors of ILD in MCTD patients are high levels of anti-U1 RNP antibodies, absence of arthritis and increasing age. The successive ILD prediction across different MCTD cohorts strengthens the value of MCTD diagnosis and anti-RNP antibody detection in clinical practice.

Disclosure of Interest: None declared

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SAT0681 THE CAUSAL ASSOCIATION BETWEEN CHILDHOOD AND ADULTHOOD BODY MASS INDEX AND OSTEOARTHRITIS: A MENDELIAN RANDOMIZATIONSTUDY

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Background: Childhood obesity is a strong predictor of adult obesity. Body mass index (BMI) has been associated with a dose-response increase in risk of osteoarthritis. However, studying causality in this association is difficult due to confounding factors

Objectives: To study the causal association of childhood and adult BMI on the risk of developing osteoarthritis using Mendelian Randomization (MR) methods

Methods: A two-step MR analysis was conducted. Exposures were determined by reviewing data from two genome-wide association study (GWAS) from >47,000 children describing 15 SNPs associated with childhood BMI, and another GWAS describing 97 SNPs related to BMI within 3 39 224 adults. For the