Methods: Wrist outcome groups (Groups) 1–4 were identified: 1=S0 T0 (not swollen, not tender); 2=S0 T1 (not swollen but tender); 3=S1 T0 (swollen but not tender); 4=S1 T1 (swollen and tender). Power Doppler (PD) and grey-scale (GS) US were used to grade (a) synovitis semi-quantitatively (0–3) at the following joint recesses: dorsal radiocarpal and intercarpal, dorsal ulnocarpal and volar radiocarpal, and (b) tenosynovitis dichotomously (0=no, 1=yes) at the following tendon sites: extensor digitorum, extensor carpi ulnaris, and flexor digitorum. Scores on each wrist consisted of a PD score, a GS score and a combined (PD +GS) US (CUS) score. Positivity (+ve) for PD, GS and CUS scores was analysed using a generalised linear repeated measures mixed model with binary distribution and logit link. Scores were analysed using a general linear repeated measures mixed model assuming Gaussian errors. In both analyses, patients were modelled as random effects, and wrist (R/L) and follow up visit (baseline, 3 months) as fixed effects. Pairwise comparisons on +ve and scores were carried out among the 4 groups in the context of the models. P-values were not adjusted for multiple comparisons.

Results: 122 wrist assessments resulted from 32 RA subjects (87.5% female; 78.1% Chinese; mean (SD) disease duration of 42.8 (52.9) months) who either started or escalated on systemic corticosteroids and DMARDs. All subjects were assessed at baseline and 29 at 3 months. Significant differences among Group scores were: 4 vs 1 (PD, $p=0.0031$; GS, $p=0.0159$; CUS, $p=0.0045$), 4 vs 2 (PD, $p=0.0176$; GS, $p=0.0160$; CUS, $p=0.0074$), and 4 vs 3 (CUS, $p=0.0374$). Significant differences among +ve were: 4 vs 1 (PD, $p=0.0007$), 4 vs 2 (PD, $p=0.0234$), and 3 vs 1 (PD, $p=0.0202$). No significant Group differences were found for 2 vs 1 (for +ve and scores) and when comparing the 4 groups for GS +ve and CUS +ve. Table 2 shows the frequency distribution of patients by wrist and follow up. There were no significant effects attributable to differences in wrists or follow up visit (p -values all >0.05).

Abstract SAT0676 – Table 1. Analysis summary of ultrasound scores and positivity in the wrist.

Wrist Outcome Group ¹	LS Mean ²	p-value comparison with S0T1			LS Mean Estimated Probability ³	p-value comparison with S0T1		
		S1T0	S1T1			S1T0	S1T1	
PD score					PD positivity			
1=S0T0	0.56	0.7545	0.0712	0.0031**	0.21	0.3151	0.0202*	0.0007***
2=S0T1	0.68		0.2518	0.0176*	0.38		0.3378	0.0234*
3=S1T0	1.12			0.1003	0.56			0.0552
4=S1T1	1.55				0.79			
GS score					GS positivity			
1=S0T0	3.61	0.8195	0.3803	0.0159*	0.83	0.4937	0.3008	0.9983
2=S0T1	3.45		0.3544	0.0160*	0.91		0.9707	0.9984
3=S1T0	4.11			0.0549	0.90			0.9984
4=S1T1	5.05				1.00			
Combined US (PD+GS) score					Combined US (PD + GS) positivity			
1=S0T0	4.22	0.9145	0.2063	0.0045**	0.83	0.4937	0.3008	0.9983
2=S0T1	4.12		0.2641	0.0074**	0.91		0.9707	0.9984
3=S1T0	5.21			0.0374*	0.90			0.9984
4=S1T1	6.60				1.00			

¹ Wrist Outcome Groups: 1=S0T0 (not swollen, not tender), 2=S0T1 (not swollen but tender), 3=S1T0 (swollen but not tender), 4=S1T1 (swollen and tender).

² Least squares mean from general linear repeated measures mixed model with Gaussian error; patients modelled as random effects, wrist (R/L) and follow up visits (BL, 3mo) modelled as fixed effects. No significant differences between wrists ($p=0.1886$) or follow up visits ($p=0.4106$).

³ Least squares mean estimated probability using a generalized linear repeated measures mixed model with binary probability distribution and logit link; patients modelled as random effects, wrist (R/L) and follow up visits (BL, 3mo) modelled as fixed effects. No significant differences between wrists ($p=0.3720$) or follow up visits ($p=0.5052$).

Statistically significant: * $p<0.05$; ** $p<0.01$; *** $p<0.001$.

Abstract SAT0676 – Table 2. Frequency distribution of subjects by follow-up and wrist

Wrist Outcome Group ¹	Baseline (N=32 subjects)		3-Months (N=29 subjects)	
	Left n (%)	Right n (%)	Left n (%)	Right n (%)
1:S0T0	7 (21.9)	6 (18.8)	7 (24.1)	8 (27.6)
2:S0T1	3 (9.4)	4 (12.5)	3 (10.3)	1 (3.4)
3:S1T0	9 (28.1)	9 (28.1)	11 (37.9)	10 (34.5)
4:S1T1	13 (40.6)	13 (40.6)	8 (27.6)	10 (34.5)

¹ Wrist outcome groups: 1=S0T0 (not swollen, not tender), 2=S0T1 (not swollen but tender), 3=S1T0 (swollen but not tender), 4=S1T1 (swollen and tender).

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Epidemiology, risk factors for disease or disease progression

SAT0677 PREVOTELLA COPRI IN INDIVIDUALS AT RISK FOR RHEUMATOID ARTHRITIS

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Background: *Prevotella* spp. have been identified as highly enriched in the intestinal microbiota of patients newly diagnosed with rheumatoid arthritis (RA), suggesting a role in the development of the disease.¹ Sequence homology between RA-specific autoantigens and proteins of *Prevotella copri* have been reported.² However, the role of these bacteria in the pathogenesis of the disease is not yet established.

Objectives: To determine the microbiome composition and prevalence of *Prevotella* spp. in different pre-clinical phases of RA, in a group of individuals at risk for RA, namely first degree relatives of patients with RA (RA-FDR).

Methods: In an ongoing cohort study of RA-FDR, enrolling individuals without clinical evidence of RA at inclusion, we categorised individuals in the following groups: 'healthy controls': asymptomatic RA-FDR without any autoantibodies or symptoms associated with possible RA; 'pre-clinical RA': individuals with 'systemic autoimmunity associated with RA' defined by the presence of anti-citrullinated peptide antibodies (ACPA) or rheumatoid factor (RF) and/or symptomatic individuals with clinically suspect arthralgias or unclassified arthritis. Participants provided stool samples for microbiome analysis. We excluded subjects who had undergone antibiotic therapy within the last 3 months, with known history of inflammatory bowel disease and/or gastrointestinal tract surgery ever. Stool samples processing and microbial diversity culture-independent analyses were performed. After DNA extraction, the V4 region of the 16S rRNA gene was amplified using barcoded primers and sequencing was done on an Illumina MiSeq. Statistical analyses of community structures were performed.³

Results: Of the 134 participants enrolled, 51 were categorised as 'healthy controls' and 83 as 'pre-clinical RA'. Table 1 shows the general characteristics of the two groups. The microbiota of 'pre-clinical RA' individuals was significantly altered compared to 'healthy controls', with abundance of specific bacteria, particularly an enrichment of *Prevotella* spp. (figure 1).

Abstract SAT0677 – Table 1. General characteristics of participants

Characteristics	Healthy controls n=51	Pre-clinical RA** n=83
Age, median (IQR)	55 (47–62)	58 (50–66)
Female sex, n (%)	39 (76)	74 (89)
Current Smoking, n (%)	12 (23)	16 (19)
Current Alcohol, n (%)	22 (46)	29 ⁽⁴¹⁾
Body mass index, median (IQR)	24 (22–27)	24 (22–27)
Obesity, BMI ≥ 30 , n (%)	5 (10)	9 (11)
ACPA positivity, n (%)	0 (0)	38 (46)*
RF positivity, n (%)	0 (0)	28 (34)*
Shared epitope (1 or 2 copies), n (%)	32 (65)	42 (52)

* p -value <0.05 , Kruskal-Wallis test for continuous variables and Fisher's exact test for categorical variables. **Pre-clinical RA group includes individuals with ACPA or RF positivity and/or with arthralgia or unclassified arthritis.

Abstract SAT0677 – Figure 1. Linear discriminant analysis (LDA) effect size (LEfSe) estimates the different relative abundance of bacteria in Healthy controls and Pre-clinical RA participants

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