Results: 888 patients with RA with 3,396 follow-up visits were included in the study. 13,636 daily air pollution records were retrieved. We found an exposure-response relationship between the concentration of air pollutants and the risk of having abnormal CRP levels (Figure 1). Patients exposed to greater concentrations of air pollutants were at higher risk of having CRP levels ≥5 mg/L. Patients exposed to PM10 concentrations ≥50 µg/m3 had a 70% higher risk of having CRP levels ≥5 mg/L (OR 1.696 95% CI, 1.245-2.311). Among RA patients, 440 patients (49.5%) had at least 2 follow-up visits with a difference in DAS28-CRP of more than 1.2 points (with current DAS28-CRP ≥3.2), serving as our sample for the case-crossover study. Concentrations of CO, NO, NO2, NOx, PM10, PM2.5 and O3 were higher in the 60-day period preceding a flare (Table 1). Sensitivity analyses considering geometric mean and cumulative concentrations yielded similar results (data not shown). Remarkably, we found that the cumulative exposure to NO2 in the 60 days preceding a flare was approximately 500 µg/m3 higher than the low disease activity visit, an exposure that equates to approximately to 200 passively smoked cigarettes (3.5 cigarettes per day on a 60-day period).

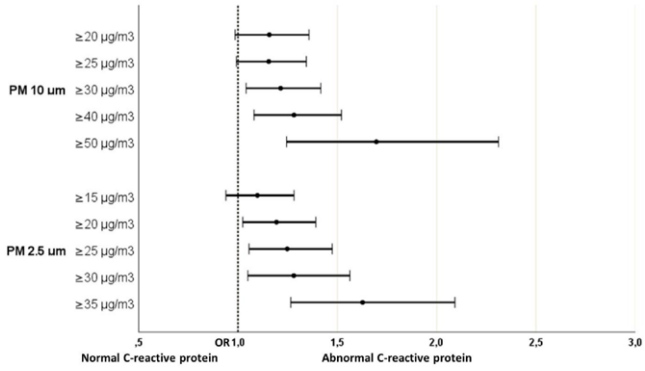

Figure 1. Odds of having abnormal CRP serum levels (≥5 mg/L) at different exposures of PM10 and PM2.5 (mean concentration in the 60 days before assessment)

Table 1. Case-crossover study. Mean concentrations (mean and Area Under the Curve) of air pollutants in the 60 days before low-disease activity visit and flare visit (DAS28-CRP difference >1.2)

Pollutant		Control period (low disease activity, n=440)	Hazard period (flare, n=440)	p value
CO µg/m^3	Mean	0.38	0.42	0.001
	AUC	22.00	24.53	0.001
NO µg/m^3	Mean	19.23	24.11	0.002
	AUC	1,120.53	1,403.88	0.002
NO_2 µg/m^3	Mean	30.91	32.44	0.042
	AUC	1,800.96	1,892.05	0.040
NO_x µg/m^3	Mean	60.34	69.35	0.004
	AUC	3,515.77	4,041.06	0.004
PM10 µg/m^3	Mean	31.21	33.65	0.011
	AUC	1,789.22	1,942.52	0.005
O_3 µg/m^3	Mean	31.08	33.79	0.002
	AUC	1,776.37	1,934.35	0.001
PM2.5 µg/m^3	Mean	23.08	24.74	0.018
	AUC	1,272.61	1,403.60	<0.001

Conclusion: We found a striking association between air pollution and RA disease severity and reactivations in a cohort of patients followed over a 5-year period. The exposure to high levels of air pollutants was associated with increased CRP levels and a higher risk of experiencing a flare of arthritis. This excessive risk was evident at very low levels of exposure, even below the proposed threshold for the protection of human health. Our study has important and direct consequences. In order to reduce the burden of RA, public and environmental health policy makers should aim to diminish gaseous and PM emissions to a larger extent as currently recommended.
Disclosure of Interests: None declared
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OP0179

DISCONTINUATION RATE OF TOFACITINIB IS SIMILAR WHEN COMPARED TO TNF INHIBITORS IN RHEUMATOID ARTHRITIS PATIENTS: POOLED DATA FROM TWO RHEUMATOID ARTHRITIS REGISTRIES IN CANADA

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Background: Tofacitinib (TOFA) is an oral, small molecule drug used for rheumatoid arthritis (RA) treatment as the first or an alternative option to biologic disease- modifying antirheumatic drugs (bDMARDs), including tumor necrosis factor inhibitors (TNFi). The similarity in retention of TNFi and TOFA was previously reported separately by the Ontario Best Practices Research Initiative (OBRI) and the Quebec cohort RHUMADATA®.
Objectives: To increase the study power, we propose to evaluate the discontinuation rate (due to any reason) of TNFi compared to TOFA, using pooled data from both these registries.
Methods: RA patients enrolled in the OBRI and RHUMADATA initiating their TOFA or TNFi between 1st June 2014 (TOFA approval date in Canada) and 31st Dec 2019 were included. Time to discontinuation was assessed using adjusted Kaplan-Meier (KM) survival and Cox regression models. To deal with confounding by indication, we estimated propensity scores for covariates with a standard difference greater than 0.1. Models were then adjusted using stratification and inverse probability of treatment weight (IPTW) methods. Multiple imputation (Imputation by Chained Equation method, N=20) was used to deal with missing data for covariates at treatment initiation.
Results: A total of 1318 patients initiated TNFi (n=825) or TOFA (n=493) with mean (SD) disease duration of 8.9 (9.3) and 13.0 (10.1) years, respectively. In the TNFi group, 78.8% were female and mean age (SD) at treatment initiation was 57.6 (12.6) years. In the TOFA group, 84.6% were female and mean (SD) age at treatment initiation was 59.5 (11.5) years. The TNFi group was less likely to have prior biologic use (33.9%) than the TOFA group (66.9%). At treatment initiation, the mean (SD) CDAI was significantly ($p<0.05$) lower in the TNFi group [20.0 (11.7)] compared to the TOFA group [22.1(12.4)]. Physical function measured by HAQ-DI was also significantly lower ($p<0.05$) in the TNFi compared to the TOFA group (1.2 vs.1.3).
Over a mean follow-up of 23.2 months, discontinuation was reported in 309 (37.5%) and 182 (36.9%) of all TNFi and TOFA patients, respectively. After adjusting for propensity score deciles across 20 imputed datasets, there was no significant difference in discontinuation between treatment groups (adjusted HRs: 0.96, 95% CI: 0.78-1.18; p=0.69). The results were similar for two propensity adjustment methods. Figure 1 shows IPTW adjusted KM survival curves comparing discontinuation rates in patients treated with TNFi and TOFA.

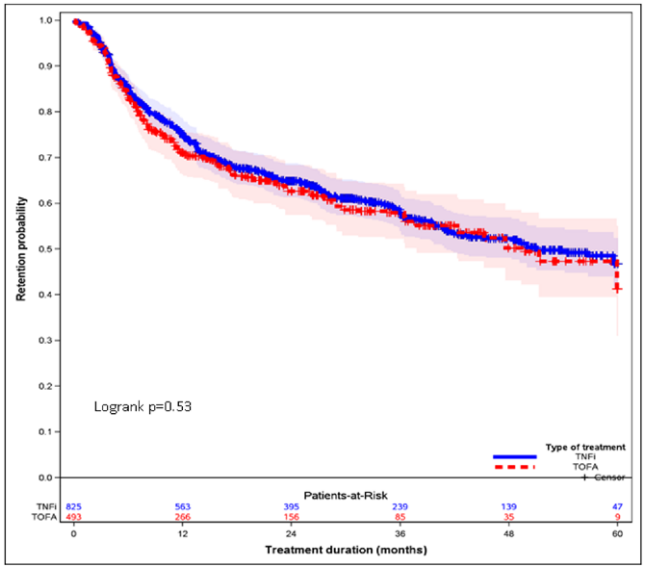

Figure 1. Note: Propensity Score Weighted (IPTW) Survival Curves was performed using one imputed dataset

Conclusion: In this pooled real -world data study, we found that TNFi and TOFA retention is similar in patients with RA. In the next step we will analysis the data for specific reasons of dicontinutaion. We will also repeat analysis comparing discontinuation in the first users versus those after one or more biologic failure.
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OP0180

IMPACT OF RF AND ANTI-CITRULLINATED PROTEIN ANTIBODY SEROSTATUS ON 2-YEAR RETENTION OF ABATACEPT IN PATIENTS WITH RA

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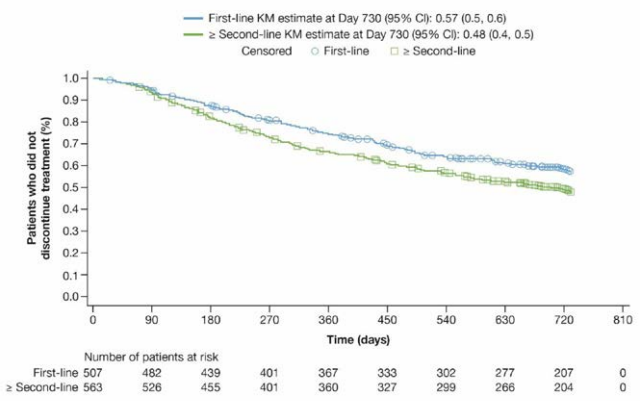
Background: Up to 50% of patients with RA discontinue DMARD treatment within 18 months.[1] However, up to 20% of patients who fail multiple treatments may have a good treatment response to another therapy.[1] Predictive biomarkers, such as RF and anti-citrullinated protein antibodies (ACPAs), may be useful to stratify patients with RA to the most appropriate treatment.[1] ASCORE (Abatacept SubCutaneOus in Routine Clinical PracticE; NCT02090556) was a 2-year, observational, prospective, multicentre study of SC abatacept for the treatment of RA in routine clinical practice.[2]
Objectives: To determine if RF/ACPA serostatus and treatment line impact abatacept retention in patients with RA in a *post hoc* analysis of ASCORE.
Methods: Eligible patients, aged ≥18 years, with active moderate-to-severe RA (ACR/EULAR 2010 criteria) who were IV abatacept-naive and initiated SC abatacept 125 mg once weekly, were enrolled into two cohorts: biologic (b) DMARD-naive patients and those with ≥1 prior bDMARD treatment failure. This *post hoc* analysis assessed abatacept retention rate at 2 years in a subset of patients with RF/ACPA serostatus data (n=1748) from the ASCORE study (N=2892; as observed). Baseline (BL) serostatus groups examined by treatment line were: RF/ACPA double positive (+/+) RA, RF/ACPA single positive (RF+/ACPA– or RF–/ACPA+) RA (data not shown) and RF/ACPA double negative (–/–) RA. Last observation carried forward (LOCF) analyses were used to assess change from BL and measures of disease remission (DAS28 [CRP] <2.6, CDAI ≤2.8, and SDAI ≤3.3) in patients with +/+ RA versus –/– RA.
Results: BL demographic and disease characteristics were similar across serostatus groups and treatment lines (Table 1). Mean age was 57.1 and 57.8 years for +/+ RA and –/– RA, respectively. Mean DAS28 (CRP) was 4.7 and 4.8 for +/+ RA and –/– RA, respectively. In patients with +/+ RA, abatacept retention was greater when given as first-line treatment (57% vs 48% when given as ≥ second-line) (Figure 1). Retention was similar in patients with –/– RA regardless of treatment line. After 2 years, mean (SE) change from BL (LOCF) in DAS28 (CRP) was –1.41 (0.06) and –0.97 (0.09) for patients with +/+ and –/– RA, respectively. For patients with +/+ RA, mean (SE) change from BL in DAS28 (CRP) was –1.62 (0.08) for those in whom abatacept was first-line and –1.19 (0.08) for those in whom abatacept was ≥ second-line. For patients with –/– RA, mean (SE) change from BL in DAS28 (CRP) was –1.03 (0.13) for those in whom abatacept was first-line and –0.93 (0.12) for those in whom abatacept was ≥ second-line. Remission rates (LOCF) were significantly (p<0.0001) higher in patients with +/+ RA vs –/– RA respectively: DAS28 (CRP) 38.4% (n=393) versus 19.3% (n=62); CDAI 50.6% (n=513) versus 33.0% (n=107); and SDAI 49.5% (n=497) versus 32.5% (n=102).
Conclusion: In this real-world analysis, patients with +/+ RA treated with first-line abatacept had higher retention than patients receiving abatacept as a ≥ second-line therapy. Remission rates on abatacept were higher in patients with +/+ RA versus –/– RA. These results support early treatment with abatacept and highlight the importance of further evaluating precision medicine approaches in RA.

Table 1. BL demographics and disease characteristics by RF/ACPA status

	+/+ RA (n=1079)		–/– RA (n=343)	
	First-line (n=511)	≥ second-line (n=568)	First-line (n=140)	≥ second-line (n=203)
Age	57.1 (13.4)	57.1 (12.2)	59.5 (14.7)	56.6 (13.2)
DAS28 (CRP)	4.7 (1.2)	4.7 (1.2)	4.8 (1.1)	4.8 (1.2)
CDAI	26.6 (12.5)	26.6 (12.4)	27.7 (12.5)	28.6 (13.8)
SDAI	28.1 (13.1)	28.1 (12.9)	29.1 (12.9)	30.2 (14.7)

Data are mean (SD). Patients with missing data for BL RF/ACPA status are excluded. ACPA=anti-citrullinated protein antibody; BL=baseline.

Figure 1. Retention rates by treatment group in patients with +/+ RA



Patients with missing data for baseline RF/ACPA status are excluded.
+/+=RF/ACPA double positive; ACPA=anti-citrullinated protein antibody; KM=Kaplan-Meier.

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