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PREDICTORS OF EROSION AND JOINT SPACE NARROWING PROGRESSION IN PATIENTS WITH EARLY RHEUMATOID ARTHRITIS

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Background: Joint damage in rheumatoid arthritis (RA) includes erosions and joint space narrowing (JSN). Predictors of these processes, and the underlying mechanisms, require further study¹.

Objectives: To investigate the relation between patient characteristics at RA diagnosis and progression of erosions and JSN, over 5 years.

Methods: Consecutive early RA patients (symptom duration <12 months), recruited 1995-2005 from a defined area, managed according to usual care with no pre-specified treatment protocol, were followed through 5 years. Radiographs of hands and feet were scored in chronological order by a trained reader according to the modified Sharp-van der Heijde score (SHS), including separate erosion- and JSN scores. The relations between baseline variables and progression of erosion- and JSN scores over 5 years (log transformed), were assessed using linear regression.

Results: 233 early RA patients were included. Radiographs at baseline and 5 years were available for 162 patients. Results on predictors of rapid radiographic progression of the total SHS have been reported previously². The median (interquartile) progression of erosion and JSN scores were 4 (0-8) and 8 (1-16), respectively. RF and anti-CCP predicted progression of erosions and JSN over 5 years, with stronger associations for erosions (Table 1). Baseline erosion- and JSN scores each predicted progression of JSN, while baseline JSN did not predict progression of erosions. In crude analyses, higher disease activity, CRP and ESR were associated with progression of both erosions and JSN, while statistically more robust for the former. Smoking and high levels of Cartilage Oligomeric Matrix Protein (COMP) (>12 U/L) were both associated with progression of erosions (B= 0.12, p= 0.016) and (B= 0.12, p= 0.02) respectively) but not JSN, in adjusted analyses (Table 1). Overweight or obesity (BMI >25kg/m²) was associated with less progression of JSN (B= -0.14, p= 0.018, adjusted for RF, age and baseline JSN score).

Table 1. Baseline predictors of progression of erosion and JSN scores (log transformed) from baseline to 5 years in linear regression

Baseline characteristics	Erosion score				JSN score			
	Crude		Adjusted for RF and baseline erosion score		Crude		Adjusted for RF, age and baseline JSN score	
	B	p	B	p	B	p	B	p
Age (per SD)	0.02	0.39	0.02	0.45	0.06	0.051	NA	NA
Overweight or obese vs. normal BMI ^a	-0.05	0.34	-0.03	0.62	-0.14	0.03	-0.14	0.018
Ever vs. never smokers	0.16	0.004	0.12	0.016	0.05	0.49	0.06	0.33
RF positivity	0.23	<0.001	NA	NA	0.18	0.004	NA	NA
Anti-CCP positivity	0.23	<0.001	0.13	0.02	0.17	0.008	0.09	0.18
COMP >12 U/L	0.10	0.08	0.12	0.02	0.08	0.24	0.08	0.23
Erosion score (per SD)	0.07	0.006	NA	NA	0.12	<0.001	0.08	0.02
JSN score (per SD)	0.04	0.12	-0.01	0.69	0.11	<0.001	NA	NA
High vs. moderate DAS28 ^b	0.17	0.003	0.12	0.03	0.14	0.04	0.12	0.054
ESR (per SD)	0.11	<0.001	0.08	0.002	0.09	0.003	0.06	0.04
CRP >9 mg/l (median) ^c	0.15	0.003	0.09	0.08	0.13	0.03	0.07	0.26

NA, not applicable; vs, versus.

^a Overweight or obese >25 kg/m²; normal 18.5 – 24.99 kg/m².

^b High >5.1; moderate >3.2 to <5.1.

Conclusion: RF, anti-CCP and markers of inflammation and disease activity predicted progression of erosions and JSN, in particular erosions. Development of erosions may predate cartilage damage leading to JSN. Smoking and high baseline levels of COMP predicted progression of erosions, but not JSN. Overweight and obesity may be associated with mechanisms that protect from JSN.

REFERENCES:

[1] Smolen et al, Ann Rheum Dis. 2009;68:1535-40.2 Rydell et al, Arthritis Res Ther. 2018;20:82.

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EFFECT OF AIR POLLUTION EXPOSURE ON DISEASE ACTIVITY IN RHEUMATOID ARTHRITIS

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Background: Environmental exposure (e.g. tobacco smoking, pollution) has been accepted to contribute in autoimmune diseases. Air pollution has been described to be a risk factor for developing rheumatoid arthritis (RA). Currently, RA disease remission is considered an achievable target in a significant proportion of patients. Nevertheless, diseases flares, that significantly contributes to damage progression and disability, remain unpredictable. Thus, factors able to potentially interfere on disease activity should be considered and assessed.

Objectives: The aim of our study is to evaluate the influence of particulate matter (PM) on disease activity and general health (GH) in patients with RA.

Methods: Consecutive patients with RA (ACR/EULAR Criteria 2010) resident in Lombardy (Italy) were enrolled. In each patient Disease Activity Score on 28 joints (DAS28), Simple Disease Activity Index (SDAI) and patients' GH were assessed. Daily PM concentrations, estimated by Regional Environmental Protection Agency at municipality resolution, were used to assign short-term exposure from day of visit back to 14 days. Continuous variables are expressed as mean±SD. Categorical variables are presented as absolute numbers and frequencies. Multivariable linear regression models were performed to identify the day of PM10 and PM2.5 independently associated with DAS28, GH and SDAI indices, adjusting for the variables significant at the univariate analysis. GH was log transformed to achieve normality of residuals. β coefficients were reported for 10 µg/m³ increments of PM concentrations. All statistical analyses were performed using SAS 9.4.

Results: 258 patients (age at visit 57.4±13.5 years, disease duration 16.2±12.1, female 80.2%, rheumatoid factor and/or anti-citrullinated peptide antibodies positivity 60.5%, smokers 15.5%, radiographic damage 41.5%) were enrolled in the study. Multivariable linear regression models were adjusted for radiographic damage, disease duration and age. Increases of PM2.5 and PM10 exposure (9 day before the visit) were significantly associated with worsening of DAS28, SDAI and GH (table below). PM concentrations in days other than 9 days before were significantly associated with disease activity and GH.

	PM Exposure	β x 10	SE	95% CI		P-value
DAS28	PM ₁₀ Day -9	0,10	0,05	0,00	0,19	0,041
	PM _{2.5} Day -9	0,11	0,06	0,00	0,23	0,048
SDAI	PM ₁₀ Day -9	0,80	0,39	0,02	1,57	0,043
	PM _{2.5} Day -9	0,95	0,48	0,00	1,90	0,049
GH	PM ₁₀ Day -9	0,11	0,04	0,03	0,18	0,004
	PM _{2.5} Day -9	0,14	0,05	0,06	0,23	0,002

Conclusion: In our cohort, PM10 and PM2.5 exposure seems to influence RA disease activity with a lag period that can last until 9 days before the visit. Nevertheless, the association between day-to-day PM changes and disease activity do not confirm causation. Further studies are required to evaluate the influence of air pollution on RA activity.

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