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.

REFERENCES

- [1] Klareskog L, Padyukov L, Alfredsson L. Smoking as a trigger for inflammatory rheumatic diseases. *Curr Opin Rheumatol* 2007;19(1):49–54.
- [2] Soy M, Erken E, Konca K, et al. Smoking and Behcet's Disease. *Clinical Rheumatology* 2000;19(6):508–509.
- [3] Lee SS, Choi CB, Lee EK, et al. The association between smoking and clinical manifestations in patients with Behcet's disease. *Korean J Med* 2008;75(2):202–209.
- [4] Tuna S, Alan S, Turkoglu EB. Effects of smoking and HLA-B51 on clinical manifestations in Behcet's disease: retrospective analysis of 209 patients in a Turkish population. *Arch Rheumatol* 2015;30(x):i-vii.

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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 RANDOMIZATION STUDY

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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