

The male excess in good EULAR response was more pronounced in seronegative compared with seropositive RA (1.98 (CI 1.4–2.8) compared to 1.21 (0.96–1.53)).  
**Conclusions:** The association of male gender with improved outcomes in early RA has not been shown before in a national cohort of this scope. Previous work suggests seronegative individuals achieve greater clinical response (2), here we present this effect amplified in men. To the authors' knowledge this is a new finding. This is likely multifactorial, with biological effect of gender, greater diagnostic uncertainty and higher reporting of global scores in women all potentially playing a role.

**References:**

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**SAT0677 THE IMPACT OF DISEASE ACTIVITY DURING PREGNANCY IN WOMEN WITH SLE ON THE OCCURRENCE OF PREECLAMPSIA AND PREMATURE BIRTH**

A.C.G. Skorpen<sup>1,2</sup>, S. Lydersen<sup>3</sup>, I.-M. Gilboe<sup>4</sup>, K.Å. Salvesen<sup>5</sup>, M. Wallenius<sup>1,2</sup>. <sup>1</sup>National advisory unit on Pregnancy and Rheumatic Diseases, Trondheim University Hospital; <sup>2</sup>Department of Neuromedicine and Movement Science; <sup>3</sup>Regional Center for Child and Youth Mental Health and Child Welfare, Norwegian University of Science and Technology, Trondheim; <sup>4</sup>Department of Rheumatology, Oslo University Hospital Rikshospitalet, Oslo; <sup>5</sup>Department of Laboratory Medicine, Children's and Women's Health, Norwegian University of Science and Technology, Trondheim, Norway

**Background:** Pregnancy in women with systemic lupus erythematosus (SLE) is associated with an increased risk of complications such as preeclampsia and premature birth. Active disease is considered one of the risk factors.

**Objectives:** The aim of this study was to explore the impact of disease activity in pregnant women with SLE on preeclampsia and premature birth, and compare the occurrence with pregnancies from the general obstetric population.

**Methods:** We linked data from RevNatus with data from the Medical Birth Registry of Norway (MBRN). RevNatus is a Norwegian nationwide prospective observational register including women with an inflammatory rheumatic disease when planning pregnancy or after conception. The register was established in 2006 and is administered by the National advisory unit on pregnancy and rheumatic diseases. Women 18 years or older are recruited and followed-up in each trimester of pregnancy and at 6 weeks, 6 months and 12 months after birth. MBRN is a national birth registry. The population constituted all singleton live births recorded in MBRN in the period 2006 – 2014. The births in women diagnosed with SLE in MBRN and included in RevNatus formed the patient group (n=180). The references were all other births (n=498849). We performed logistic regression, and calculated OR for preeclampsia and premature birth in the patient population compared to the references from the general obstetric population. The target population was then split in two groups according to disease activity assessed in the 2nd trimester, and compared to references.

**Results:** Women with SLE had a significantly higher risk of preeclampsia and premature birth than references after adjustment for maternal age, parity and smoking (Table 1).

Concerning preeclampsia, there was no significant difference in women with no disease activity compared to references, whereas active disease resulted in a significantly higher odds. We observed a two-fold and eight-fold increase in risk of premature birth in women with quiescent and active disease, respectively (Table 2).

Table 1. Preeclampsia and premature birth in references and in women with SLE

Adverse event, n (%)	References n=498,849	SLE n=180	Adj OR* (95% CI)	p
Preeclampsia	15132 (3.0)	14 (7.8)	2.64 (1.53, 4.58)	0.001
Premature birth	27063 (5.5)	34 (18.9)	4.25 (2.92, 6.19)	<0.001

\*Adjusted for maternal age, parity and smoking in pregnancy.

Table 2. Preeclampsia and premature birth in references and in women with SLE according to disease activity status

Adverse event, n (%)	References n=498,849	No disease activity† n=85	Adj OR* (95% CI)	p
Preeclampsia	15132 (3.0)	4 (4.7)	1.60 (0.58, 4.38)	0.36
Premature birth	27063 (5.4)	11 (12.9)	2.78 (1.47, 5.24)	0.002

Adverse event, n (%)	References n=498,849	Disease activity‡ n=63	Adj OR* (95% CI)	p
Preeclampsia	15132 (3.0)	9 (14.3)	5.05 (2.48, 10.30)	<0.001
Premature birth	27063 (5.4)	21 (33.3)	8.63 (5.10, 14.60)	<0.001

†LAI-P=0. ‡LAI-P>0. \*Adjusted for maternal age, parity and smoking in pregnancy.

**Conclusions:** Women with SLE have a higher risk of preeclampsia and premature

birth than references from the general obstetric population. Women with active disease in pregnancy are most prone to these complications.

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**SAT0678 SIGNIFICANTLY INCREASED SEROPOSITIVITY, RHEUMATOID FACTOR TITRES AND RHEUMATOID NODULES IN CORNISH KAOLIN WORKERS**

D. Murphy<sup>1,2</sup>, E. Fleeming<sup>2</sup>, K. Bellis<sup>1</sup>, D. Hutchinson<sup>1,2</sup>. <sup>1</sup>Rheumatology, Royal Cornwall Hospital; <sup>2</sup>University of Exeter Medical School, Cornwall Campus, Truro, United Kingdom

**Background:** Kaolin has been mined in Cornwall, UK for over 250 years, with half of the world's production originating from the area at the turn of the 20th century. Mineral kaolinite does not react as free silica, and is used in heavy metal contamination containment due to its adsorption capabilities.<sup>1</sup>

**Objectives:** To investigate the prevalence of occupational kaolin dust exposure in male rheumatoid arthritis (RA) patients in Cornwall, UK.

**Methods:** All males diagnosed with RA under follow up at the Royal Cornwall Hospital, UK, during the study period April 2015-January 2017, were invited to complete an occupational questionnaire, detailing current occupation, last occupation (if retired) and other occupations for > 1 year.

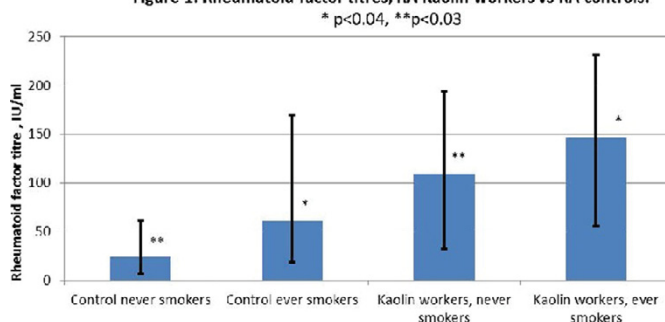
**Results:** 39/720 (5%) cases were excluded on non-return of initial questionnaires. 41/720 (6%) cases died during the study period (22 months). 54/640 (8%) remaining cases had occupational kaolin exposure, approximately 12 times higher than expected based on current census employment rates. 30/54 had long term kaolin dust exposure, living in the post code region PL22-PL26 (total male population 33693). These were matched for age ±2 years, sex and index of multiple deprivation (IMD) ±1 decile, to RA patients with no occupational dust or fume exposure. 40/110 potential controls were successfully matched.

Significantly more RA kaolin workers were seropositive for RF and ACPA than non-dust exposed RA controls (Table 1). Of RF seropositive patients, median RF titres were significantly higher in kaolin workers than unexposed controls (p<0.01). No significant differences were seen in ACPA titre. Smoking prevalence rates were not significantly different between kaolin workers and controls. Amongst ever smokers, median pack years smoked showed no difference between cases and controls.

	Kaolin workers (n=30)	Control (n=40)	Significance
RF+	28/30 (93%)	30/40 (75%)	p<0.05
ACPA+	27/30 (90%)	24/40 (60%)	p<0.01
Median seropositive RF titre (IQR)	142 (68–124)	61 (25–162)	p<0.01
Median seropositive ACPA titre (IQR)	375 (169–500)	283 (199–500)	Not sig.
Ever smokers (%)	23/30 (77%)	26/40 (65%)	Not sig.
Median pack years ever smokers (IQR)	18 (15.5–32)	17 (10.75–32.25)	Not sig.

Kaolin exposed never smokers demonstrated significantly higher RF titres than unexposed never smokers (kaolin median RF 109 (IQR 32–193.5), control median RF 24 (IQR 7–61), p<0.04), as did kaolin exposed smokers (kaolin median RF 146.7 (IQR 56–231.5), control median RF 61 (IQR 18.5–170), p<0.03, figure 1). Interestingly, 7/30 (23%) kaolin workers demonstrated nodular disease, compared to 3/40 (7.5%) matched controls, p=0.06, significantly higher than the background nodular rate 79/720 (11%) of male RA patients throughout Cornwall, p<0.04.

Figure 1. Rheumatoid factor titres, RA Kaolin workers vs RA controls.



**Conclusions:** Kaolin dust exposure in RA confers higher RF and ACPA seropositivity rates and higher RF titres. Smoking and kaolin dust exposure have an additive effect on RF titres. Higher rates of nodular disease are seen in RA patients exposed to kaolin dust, independent of smoking. We hypothesise that this interaction is due to adsorption of heavy metals from tobacco smoke or other environmental sources by kaolin particles, stimulating autoantibody production.

**References:**

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