

**Abstract 93 Table 1** Baseline lupus factors influencing damage accrual in 99 lupus patients. Regression analysis

	OR	95% confidence limits	p value
<b>New damage accrual</b>			
Intercept	0.085	-	0.024
Cytotoxics use (yes)	12.64	0.88–182.47	0.062
SLEDAI-2K	1.130	0.996–1.28	0.057
Anti-NSPA positivity	17.604	1.13–273.52	0.040
<b>New damage accrual excluding anti-NSPA</b>			
Intercept	0.20	-	<0.001
Cytotoxics use (yes)	11.866	1.39–101.53	0.024
SLEDAI-2K	1.149	1.03–1.29	0.016

Candidate variables considered were: age, education, disease duration, SLEDAI-2K, SDI, cognitive deficit by Cambridge Neuropsychological Test Automated Battery (CANTAB), depression score of the Hospital Anxiety and Depression Scale (HAD), presence of antiphospholipid syndrome, prednisone use, cytotoxics use, anti-dsDNA positivity, anti-ribosomal P (anti-P) and anti-neuronal surface P antigen (anti-NSPA) antibodies positivity.

with final damage whereas SLEDAI-2K and cytotoxics associated with accrual damage. Models including anti-NSPA showed its impact on final and accrual damage. Cognitive deficit, depression and other autoantibodies were not damage predictors.

**Conclusions** Our findings support that disease activity and cytotoxic use are relevant lupus damage predictors. Cognitive dysfunction and depression do not contribute to damage accrual as may fluctuate in lupus patients. A potential influence of anti-NSPA antibodies on damage accrual is proposed.

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#### 94 DEVELOPMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS SYMPTOMS IS ASSOCIATED WITH CIGARETTE SMOKING

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**Background** Smoking is associated with the development of aCCP positive rheumatoid arthritis. The association between smoking and SLE is more uncertain. 1) We wanted to compare SLE patients smoking habits before symptom development with population controls.

**Methods** 306 SLE patients fulfilling ACR classification criteria from Oslo area were sent a questionnaire in 2010 regarding smoking. Antibody profiles were collected from medical records.

Year of first symptom was defined by the patient, or if the doctors time of the first SLE symptom was earlier, this year was used. Juvenile SLE was defined by start of symptom before 16 years of age. One pack year was 20 cigarettes daily in one year.

The proportion smokers in the Norwegian population were found online 2) and every SLE patient was matched with population controls for the year of the first symptom, age (number not available for every years of age, but 10 year groups) and gender.



**Abstract 94 Figure 1** The proportion of adult SLE patient smoking at the time of their first SLE symptom

**Results** 255/306 (80%) SLE patients responded to the questionnaire. The mean age in 2010 was 48 years old (SD 15, min 18 max 98) There were 39 patients with juvenile SLE whereof 64% were never smokers and 4/39 (10%) were smoking at the time of the 1. symptom.

Among adult SLE, 116 (42%) were never smokers and 83 (35%) were previous smokers. In year 2010 53 (22%) SLE patients were still smoking vs 20% in the control population (ns).

109/216 (50%) adult SLE patients were smoking at the time of the first SLE symptom vs 35% in the matched control population ( $p < 0,05$ ), figure 1. SLE patients had been smoking for a mean of 14 years when first symptom appeared and the mean pack year before first symptom were 10 (SD 11). The mean pack year after first symptom were 1 (SD 5).

Ever smokers did not have more dsDNA, aSm or aRNP antibodies, but they had a tendency to more discoid lupus (17% vs 8%).

**Conclusions** Even though prevalent SLE patients do not smoke more than their population controls, SLE patients are more often smokers at the time of the first symptom, especially in the higher age groups. This might indicate smoking as an environmental risk factor for developing SLE. However, smoking is tightly related to several other socioeconomic and cultural factors which must be explored in a prospective manner.

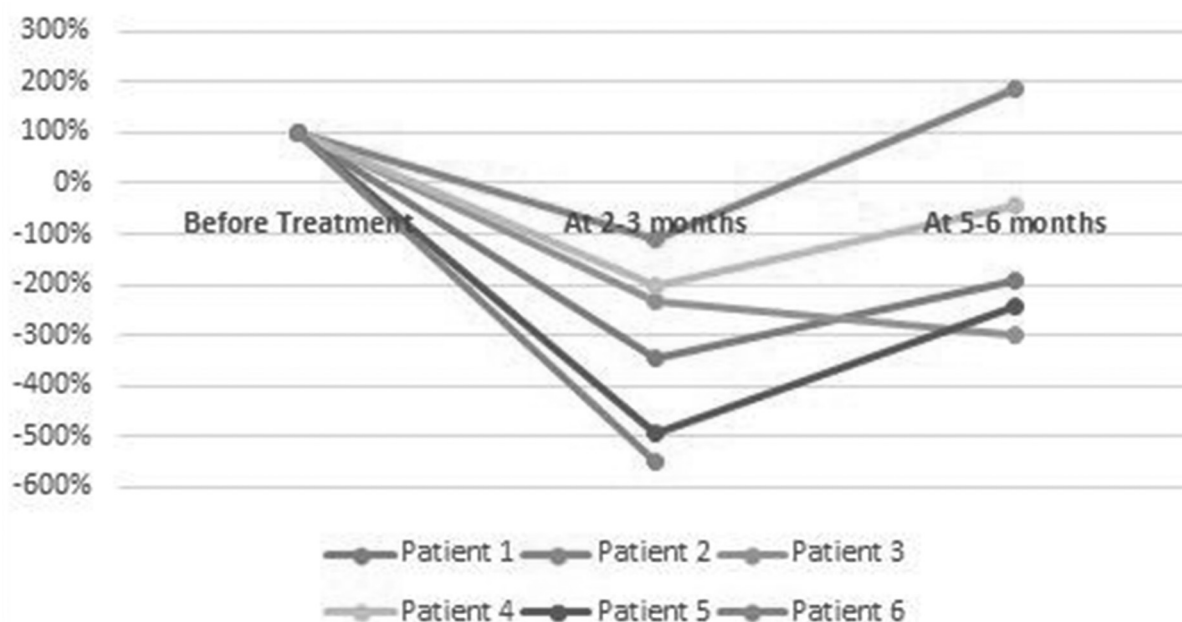
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#### 95 THE PERFORMANCE OF A RENAL ACTIVITY INDEX IN LUPUS NEPHRITIS IN INDUCTION THERAPY

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**Background** Renal involvement in systemic lupus erythematosus (SLE) is associated with high morbidity and mortality. Current standard tools to monitor lupus nephritis (LN) are suboptimal compared to the invasive renal biopsy. The renal activity index in lupus (RAIL) was developed using 6 urinary biomarkers to reflect disease activity. In children this tool was 92% accurate in identifying active LN. We aim to study the changes in this score in relation to induction treatment in LN.



**Abstract 95 Figure 1** Changes in the RAIL score from baseline, at 2–3 months and at 5–6 months in 6 patients with LN. Baseline value is assigned 100%

**Methods** Urine samples were collected from active LN patients prior to induction treatment for LN and serially afterwards, coinciding with clinical visits. Luminex Bead Multiplex Assay was used for the analyses of urine biomarkers included in the RAIL score (neutrophil gelatinase-associated lipocalin, ceruloplasmin, monocyte chemoattractant protein-1, adiponectin, hemopexin, kidney injury molecule-1). RAIL scores were calculated per the defined algorithm for each urine sample. Data collected include LN histologic classification (International Society of Nephrology (ISN)/Renal Pathology Society (RPS) classification), renal SLE disease activity index (rSLEDAI) score and type of therapy.

**Results** At the time of the analysis, data from 6 active LN patients were collected longitudinally. Patients were all females and all had class IV LN per the ISN/RPS. Renal SLEDAI scores were on the higher end ( $M=11.3$ ,  $SD=3.9$ ). All patients were started on intravenous methylprednisolone and cyclophosphamide (CYC) therapy. All but one patient completed 6 doses of monthly CYC before switching to oral mycophenolate mofetil therapy. The RAIL scores for the 6 patients ranged between  $-1.8$  and  $3.29$ . All patients had reductions in their RAIL score at 2–3 months period at an average of 322% decline from baseline (Figure 1). At the end of induction treatment or at the 5–6 months interval, 5/6 samples were available for analysis and showed that 4/5 patients maintained a decline of RAIL scores below the baseline. Of note the patient with higher RAIL score at the end of treatment had only 3 monthly doses of CYC. All rSLEDAI scores decreased between baseline and the 6 months interval. However, one patient with known medication non-adherence had a flare of LN at the 6 months point leading to increased rSLEDAI.

**Conclusions** RAIL scores show overall improvement from baseline with LN induction therapy. Lack of improvement was associated with flare of disease. Additional data points and a larger study sample are required to study the ability of the RAIL score to reflect clinical improvement of LN.

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#### CEREBRAL HEMODYNAMICS AND MICROCIRCULATORY FUNCTION IN SWEDISH PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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**Background** Systemic lupus erythematosus (SLE) is a chronic inflammatory disease characterized by multiple organ involvement. Atherosclerosis is the underlying cause for SLE-related cardiovascular disease, and reliable non-invasive methods for early detection of vascular involvement, including microcirculatory assessment, is important. The aim of this study was to detect whether macro- and microcirculation is impaired in patients with SLE.

**Methods** Fifteen patients classified with SLE according to the 2012 SLICC criteria (mean age 51.4 years) with moderate atherosclerotic ultrasound findings in common carotid artery, and 15 age- and sex-matched and plaque-free population controls, (mean age 51.7) were investigated. Intima-media thickness (IMT) was recorded with high frequency ultrasound (GE Logic E9) in carotid and central arteries. Microcirculatory oxygen saturation and endothelial function were assessed with EPOS (Enhanced Perfusion and Oxygen Saturation) (PeriFlux 6000, Perimed, Järfälla, Sweden) and EndoPAT2000 system (Itamar Medical, Israel). The EPOS system measures red blood cell tissue fraction, speed resolved perfusion and oxygen saturation in the microcirculation of the skin. EndoPAT 2000 records changes in finger arterial pulsatile volume reflecting microcirculatory endothelial function. Cerebrovascular reserve capacity was assessed by Transcranial Doppler (TCD) (Sonara TCD (Natus) by detecting mean flow velocities in middle