damage. However, it is not yet well known whether hyperuricemia is independently associated with renal involvement even in patients without known predisposing factors for hyperuricemia such as decreased renal function, old age, male gender, menopause, and drugs.

Objectives: The aim of this study was to evaluate the association of baseline serum uric acid level and development of nephritis in patients diagnosed with SLE.

Methods: We retrospectively reviewed electronic medical records of 101 female SLE patients whose ages at the time of diagnosis (baseline) were 45 years old or below in a tertiary medical center from January 2000 to March 2015. SLE with renal involvement was diagnosed when patients met the renal disorder of the 1997 ACR criteria for SLE. We compared baseline serum uric acid levels of the SLE patients who had nephritis at the time of diagnosis or later (nephritis group) with the patients who had not developed nephritis (non-nephritis group) during follow-up period.

Results: Among 101 patients, 22 (22%) had hyperuricemia (serum uric acid ≥ 6 mg/dL) at baseline and 45 (45%) had developed nephritis during follow-up period (median 6.1 years). There were significant differences in baseline serum uric acid level, glomerular filtration rate (GFR), anti-dsDNA antibody and complement level between non-nephritis group and nephritis group (Table). Interestingly, among 59 patients with normal renal function (baseline GFR ≥ 90 mL/min/1.73m²), baseline serum uric acid level was also significantly higher in the nephritis group than non-nephritis group (Table). Moreover, the patients with hyperuricemia at baseline were more likely to develop nephritis than those without hyperuricemia during follow-up period (64% vs. 39%, $p=0.042$) (Figure).

Table 1. Baseline Characteristics of Study Patients with Systemic Lupus Erythematosus

	Non-nephritis group	Lupus nephritis group	<i>p</i>
All patients (%)	56 (55)	45 (45)	
Sex, Female (%)	56 (100)	45 (100)	
Age, years	28.2 $\pm$ 8.3	28.9 $\pm$ 7.6	0.680
Body Mass Index, kg/m ²	21.2 $\pm$ 3.8	22.0 $\pm$ 3.0	0.105
Follow-up, years	6.0 $\pm$ 4.3	7.5 $\pm$ 4.3	0.068
Serum Uric Acid, mg/dL	4.5 $\pm$ 1.6	5.5 $\pm$ 1.5	0.003
GFR, mL/min/1.73m ²	103.1 $\pm$ 24.9	85.6 $\pm$ 27.0	0.001
Anti-dsDNA, IU/mL	56.4 $\pm$ 107.9	141.8 $\pm$ 163.4	0.001
C3, mg/dL	80.1 $\pm$ 32.3	48.6 $\pm$ 30.2	0.000
C4, mg/dL	14.0 $\pm$ 8.3	9.8 $\pm$ 8.2	0.005
Drugs that may increase serum uric acid level ¹ (%)	5 (8.9)	2 (4.4)	0.626
Drugs that may decrease serum uric acid level ² (%)	1 (1.8)	0 (0.0)	1.000
Patients with normal renal function (baseline GFR ≥ 90) (%)	39 (66)	20 (34)	
Serum Uric Acid, mg/dL	4.3 $\pm$ 1.4	5.1 $\pm$ 1.4	0.047
GFR, mL/min/1.73m ²	113.0 $\pm$ 15.6	106.2 $\pm$ 11.7	0.095
Anti-dsDNA, IU/mL	56.2 $\pm$ 111.0	135.0 $\pm$ 185.2	0.060
C3, mg/dL	76.3 $\pm$ 35.0	47.3 $\pm$ 30.6	0.003
C4, mg/dL	13.3 $\pm$ 7.8	8.8 $\pm$ 7.2	0.028

Values are mean $\pm$ SD or number (%). ¹Diuretics, aspirin, pyrazinamide, or cyclosporine.

²Allopurinol, febuxostat, benzbromarone, losartan, fenofibrate, or atorvastatin.

Figure. Proportion of patients who had developed nephritis according to serum uric acid levels at the time of diagnosis with SLE

Conclusions: These findings suggest that high serum uric acid level at the time of diagnosis in premenopausal women with SLE may be independently associated with nephritis. More careful evaluation would be required for development of nephritis in such patients.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.4751

AB0492 NUCLEIC ACIDS OXIDATIVE STRESS STATUS IN SJÖGREN'S SYNDROME

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Background: Although there are many defence systems that protect cellular macromolecules against oxidation, oxidative DNA and RNA damage still occurs.

Oxidative damage to nucleic acids has been found to be associated with etiopathogenesis and disease activity of inflammatory disorders. Oxidised guanine species have been recognized as a biomarker of oxidative DNA and RNA damage by endogenously generated oxygen radicals. Sjögren's syndrome (SS) is an autoimmune disorder and associated with overexpression of proinflammatory cytokines. Related to excess expression of proinflammatory cytokines, a prooxidant state could be postulated in SS.

Objectives: We aimed to evaluate levels of nucleic acids oxidative stress products in patients with SS.

Methods: 11 patients with SS diagnosed according to 2012 American College of Rheumatology (ACR) Classification Criteria for Sjögren's Syndrome, 19 patients with psoriatic arthritis (PsA), diagnosed according to Classification Criteria for Psoriatic Arthritis (PSARC), 9 patients with rheumatoid arthritis (RA), diagnosed according to 2010 Rheumatoid Arthritis Classification Criteria, and 12 healthy controls were included. All SS patients were on hydroxychloroquine sulphate 400mg/day. All PsA and RA patients were on methotrexate 15–20mg/week and folic acid 5 mg/week. The serum samples were collected from participants and stored at -30 °C until assayed. Three oxidised guanine species, 8-hydroxy-2'-deoxyguanosine as a DNA oxidation marker, 8-hydroxyguanosine as a RNA oxidation marker, and 8-hydroxyguanine as a DNA and RNA oxidation marker, were measured using DNA/RNA Oxidative Damage ELISA Kit (Cayman Chemicals, USA).

Statistics: All data are presented as mean and standard deviation (SD). Differences between groups were examined using Kruskal-Wallis tests. The Mann-Whitney U test was performed to test the significance of pairwise differences using Bonferroni correction to adjust for multiple comparisons. A p-value less than 0.05 were considered as statistically significant. Statistical analysis of correlation was performed by using the Spearman rank test.

Results: There was no statistically significant difference between the groups in terms of age and gender. The average level of serum oxidised guanine species in the PsA, RA, SS and healthy control groups were 2871.77 $\pm$ 336.20, 2672.20 $\pm$ 292.04, 3375.57 $\pm$ 344.21, 2777.55 $\pm$ 237.05 pg/mL respectively. Oxidised guanine species levels were significantly higher in patients with SS and positively correlated with CRP levels ($p=0.011$, $r=0.726$) (table 2).

Table 1. Demographic characteristics of individuals

	Psoriatic arthritis	Rheumatoid arthritis	Sjögren's syndrome	Healthy controls	<i>p</i> value
Age	41.05 $\pm$ 11.61	51.56 $\pm$ 11.17	48.25 $\pm$ 10.31	41.17 $\pm$ 6.50	0.076
Female/male	8/11	8/1	11/1	5/7	0.096

Table 2. Correlation between oxidised guanine species level and ESR, CRP, IgG, IgA, IgM, C3, C4

		ESR	CRP	IgG	IgA	IgM	C3	C4
oxidised guanine species level	<i>p</i>	0.428	0.01	0.721	0.556	0.04	0.745	0.704
	<i>r</i>	0.253	0.726	0.226	0.189	-0.595	0.105	0.123

ESR: erythrocyte sedimentation rate, CRP: C-reactive protein, Ig: Immunoglobulin, C: Complement

Conclusions: A marked increase in DNA damage leading to oxidative stress may contribute to tissue damage in SS.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3286

AB0493 BLYS UPREGULATION IS RELATED TO LYMPHOPENIA IN SYSTEMIC ERYTHEMATOUS LUPUS PATIENTS

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Background: B lymphocyte stimulator factor (BlyS) is produced by wide range of cells of the immune system, and has proven to be a key factor in the selection and survival of B cells. BlyS is an important factor in the pathology of Systemic Lupus Erythematosus; elevated serum levels (≥ 20 ng/mL) of soluble BlyS are at increased risk of flare.

Objectives: Analyze the association among BlyS levels and clinical manifestations, as well as with SLE clinical activity.

Methods: A cross-sectional and observational study was performed in patients diagnosed of SLE according to SLICC 2012 criteria and healthy controls. The study included a complete blood-test and clinical data collected by personal interview. Disease activity assessment was made by SLEDAI index and for the evaluation of chronic damage we used the validated SLICC damage index. Serum concentration of BlyS was analyzed by colorimetric methods. Lupus patients were dichotomized as high and low BlyS levels based on BlyS levels above 2 SD of the mean in healthy controls. Biostatistical analysis with R (3.3.2.) was performed.