Methods: Urine samples (n=86) were obtained from LN patients and classified in two groups: patients with eGFR >60 (GFRhigh, n=68, 62F/6M, age: 34.07±13.24) and patients with eGFR ≤60 (GFRlow, n=18, 14F/4M, age: 35.22±13.76). RNA from urine samples was isolated using TRIzol-Chloroform technique and then reverse-transcribed using random primers. Levels of BLyS expression were evaluated using Quantitative Real Time PCR (QPCR). All amplifications were carried out in duplicate and threshold cycle (C_t) scores were averaged for calculations of relative expression values. The C_t scores were normalized against C_t scores by subtracting the corresponding β2Microglobuline (β2M) control, or $DCt=C_{t,gene}- C_{t,B2M}$.To test for differential gene expression between groups, a two sample t-test was performed to compare the DCt in the two groups.
Results: DCt is inversely proportional to BLyS's expression. We evaluated data from ΔCt analysis observing that mRNA levels of BLyS in eGFRlow (6.193±1.787) were higher than those from eGFRhigh (7.564±2.326), with a statistically significant difference between groups (p=0.0288).

	eGFRlow	eGFRhigh	
BLyS (ΔCt)	6.193±1.787	7.564±2.326	p=0.0288

Conclusions: In the present cross-sectional study, increased levels of BLyS were observed in patients with eGFR ≤60. These gene expression results might be linked to B cell activation and proliferation in kidney and thus in urine samples. Combination of eGFR and BLyS appears to be a good biomarker.
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THU0018 ANGIOTENSINOGEN AS A MARKER OF INJURY IN LUPUS NEPHRITIS

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Background: Lupus Nephritis (LN) is one of the most severe forms of systemic lupus erythematosus (SLE) (1). Angiotensinogen (AGT) gene encodes the only glycoprotein known to be a precursor of the vasopresor angiotensin II (Ang II). Ang II is also a growth factor and a profibrogenic cytokine (2). In kidney transplantation AGT has been founded down expressed in biopsies with chronic allograft dysfunction (3). In LN, AGT deserves evaluation.
Objectives: To investigate AGT expression in biopsies and urines from LN patients.
Methods: 32 biopsies/urines paired from 32 LN patients was included. Kidney biopsies were evaluated according to the ISN/RPS classification system. Levels of AGT were evaluated using Quantitative Real Time PCR. Threshold cycle (C_t) scores were averaged for calculations of relative expression values. The C_t scores were normalized against C_t scores by subtracting β2Microglobuline control, or $\Delta Ct=C_{t,gene}- C_{t,B2M}$. Data expressed as ΔCt are inversely proportional to gene expression level. Nonparametric Mann Whitney test analysis and Anova with Bonferroni test were performed.
Results: 26 (81.3%) patients were female with a mean age at biopsy time of 31.9±29 years. The SLEDAI at the time of biopsy was 10.5 (IQR 0–15.7) and SLICC ≥1 in 13 (32.5%), hypocomplementemia 13/31 (41.9%) and positive DNA in 11/29 (37.9%) patients. Biopsies from patients with proteinuria ≥0.5 and renal failure (n=23), proteinuria isolated (n=14), LN remission (n=9), renal failure (n=7) and nephrotic syndrome (n=2) were performed. The mean value of ΔCt AGT gene expression in renal biopsy was 4.50 (IQR 3.51 – 5.67) and AGT in urine samples was 13.94 (IQR 11.66 – 17.89).
Conclusions: In the present study we found a potential utility of AGT mRNA levels in samples of active vs remission LN patients. Prospective studies are needed for confirming these results.

Table 1. AGT gene expression in biopsies and urines samples

	Class I/ Normal Biopsies n=3	Class II Biopsies n=6	Class IV Biopsies n=12	Class V/VI Biopsies n=10	p p
ΔCt AGT Biopsies*	5,57 (3,60–5,57)	3,67 (2,19–5,37)	5,34 (4,75–10,93)	4,35 (3,45–4,57)	0,02
ΔCt AGT Urines**	14,11 (12,44–14,11)	11,19 (9,53–11,59)	16,77 (12,88–18,25)	15,06 (12,16–17,62)	0,01

*p<0,05 class IV *vs* II; **p<0,05 Class IV *vs* II.

Table 2. AGT gene expression in biopsies according proteinuria levels

	Proteinuria ≤0,5	Proteinuria >0,5	p
ΔCt AGT Biopsies	3,60 (3,34–4,41)	4,79 (3,73–6,39)	0,04

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THU0019 FEATURES OF TELOMERE LENGTH DISTRIBUTION ON INDIVIDUAL CHROMOSOMES IN RHEUMATOID ARTHRITIS

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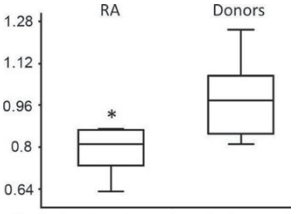
Background: Telomeres are nucleoprotein structures,that protect the ends of chromosomes during cell divisions [1,2,3]. Previously it was found, that average telomere length in immune cells is reduced in atopic and autoimmunity disorders.This fact indicates an early immune aging in immune-mediated diseases [4,5]. The distribution of telomere repeats on different chromosomes has an individual telomere profile in humans [6] and may be a congenital feature, that accelerates immunosenescence.
Objectives: The purpose of this study was to evaluate the length of telomeres in the arms of individual chromosomes in patients with RA and healthy donors.
Methods: The study included 6 patients with RA and 6 healthy donors (the median age 51.5 (50–54) and 51.5 (49–53) years respectively). Metaphase spreads obtained from PBMCs were used in this study. Written informed consent was obtained from each person enrolled in the study. At the time of sampling, RA inpatients characterized with acute exacerbation of the disease received treatment at the Clinic of Immunopathology,Novosibirsk. RA was diagnosed by clinicians according to ACR/EULAR 2010. For measurement of the telomere length on individual chromosome arms we used Q-FISH with $(C_3TA_2)_3$ PNA-probe. Inverted DAPI banding was used for chromosome identification according to ISCN 2013. The new MeTeLen software was developed to estimate the telomere repeats relative quantity (http://www.bionet.nsc.ru/en/development/application-development/development-of-a-computer/metelen.html) in metaphase images.
Results: When comparing the telomere length, it was found, that telomere on chromosome 16 p are shorter in patients with RA than in donors. Since each person has an individual telomere profile, we also analyzed the presence of shortened telomere sequences on individual chromosome arms relative to the average length of telomeres for each subject separately. As a result, patients with RA have a larger number of significantly shortened telomeres than donors (see Table).

Comparison group	Chromosome arms with shortened telomere repeats
Healthy volunteers	12p, 19p, 2q, 20q
Patients with RA	7p, 12p,16p, 17p, 19p, 2q, 20q, 21q



Telomere length on chromosome 16 p (expressed in relative units) in patients with RA and healthy donors. Data are presented as median and interquartile range, *-significant difference (p< 0.05, Mann-Whitney U test).

Conclusions: The revealed features of telomeric profiles of patients with RA may be an indication of a proliferative stress, that occurs as a result of the mass immune cell proliferation in the immunopathology. It can be assumed, that the presence of a great number of shortened telomeres can promote cell death through apoptosis. The observed shortening of the telomere length on chromosome 16 p in RA may be relevant in its pathogenesis. It is known, that telomere shortening can lead to increased gene expression near the telomere DNA region. Thus, in 16 p 13 a number of genes is localized, that are associated with RA or may be involved in its development.