evaluate the relative likelihood of death among those with SLE compared to those without SLE at the same hospital.

Results We included 4001 hospitalizations of patients with SLE and sepsis, and 2 02 888 hospitalizations of patients with sepsis without SLE. The number of SLE hospitalizations ranged from 5 to 66 per hospital. Across all hospitals, 11.5% of patients with SLE and 13.1% of patients without SLE died during the hospitalization. The RR was greater than 1.0 in 44% of hospitals, and greater than 2.0 in 16% of hospitals, indicating increased risk of mortality by a factor of two or more among patients with SLE compared to those without SLE at the same hospital. Based on classification tree analysis, large urban hospitals were less likely to have RR >2 than smaller hospitals (13% vs 22%), particularly those in the mid-west or northeast (35%).

Conclusions Risks of mortality due to sepsis among patients with SLE varied widely among U.S. hospitals. Risks were higher for patients with SLE at smaller hospitals.

Acknowledgements Supported by the Intramural Research Program, NIAMS/NIH.

CS-28 VALIDATION OF REMISSION AND LUPUS LOW DISEASE ACTIVITY STATE AS PREDICTORS OF ORGAN DAMAGE IN SLE

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10.1136/lupus-2018-lsm.63

Background Outcome measures that combine control of SLE activity and prednisone reduction are clinically relevant. A clinical goal in SLE is to reduce risk of long-term organ damage. We assessed whether two recently proposed disease activity outcomes were predictive of future damage.

Methods For each month of follow-up in a large SLE cohort, we determined whether the patient was in Clinical Remission (as defined by the DORIS working group) or lupus low disease activity state (LLDAS) (as defined by Franklyn et al). Clinical Remission was defined as a PGA<0.5, clinical SLEDAI=0 and no prednisone or immunosuppressants. Clinical Remission on Treatment allowed for prednisone ≤5 mg/day and immunosuppressant use. LLDAS was defined as a SLEDAI ≤4, PGA≤1.0, no major organ activity, and no new activity. LLDAS on treatment allowed for prednisone use ≤7.5 mg/d and immunosuppressants. Damage was defined using the SLICC/ACR Damage index.

Results There were 81 118 person-months observed among 2026 patients (92% female, 53% Caucasian, 39% African-American). Table 1 shows the rates of damage, per person month, in subgroups defined by Remission or LLDAS.

Damage rates were relatively low when LLDAS was achieved at least 50% of the time. These rates were similar to those experienced by patients who met a more stringent treatment restriction with Remission on Treatment at least 50% of the time.

Conclusions Percent time in LLDAS had a clear dose response for rate ratios of organ damage. Even 25% of time in

Abstract CS-28 Table 1 Rates of new damage, in subgroups defined by past levels of disease activity

Percentage of Prior Months in:	Number of person-months observed	Number of months with an increase in SLICC/ACR Damage Index	Rate of damage per 100 person months	Rate Ratios	P-values
Clinical Remission	—	—	—	—	—
None	35 772	406	1.13	1.0 (Ref)	—
Not none, but<25%	14 358	102	0.71	0.60 (0.48, 0.75)	<0.0001
25% to 50%	6573	50	0.76	0.66 (0.46, 0.94)	0.023
50% to 75%	3845	27	0.70	0.63 (0.42, 0.97)	0.035
75%+	1641	10	0.61	0.58 (0.30, 1.15)	0.12
Clinical Remission on Treatment	—	—	—	—	—
None	16 491	250	1.52	1.0 (Ref)	—
Not none, but<25%	20 169	170	0.84	0.54 (0.44, 0.67)	<0.0001
25% to 50%	14 344	103	0.72	0.46 (0.36, 0.60)	<0.0001
50% to 75%	8396	54	0.64	0.43 (0.30, 0.60)	<0.0001
75%+	2789	18	0.65	0.45 (0.27, 0.75)	0.0019
LLDAS	—	—	—	—	—
None	30 366	343	1.13	1.0 (Ref)	—
Not none, but<25%	10 880	106	0.97	0.86 (0.69, 1.07)	0.18
25% to 50%	5012	40	0.80	0.70 (0.51, 0.98)	0.037
50% to 75%	8494	60	0.71	0.63 (0.48, 0.83)	0.0010
75%+	7527	46	0.61	0.54 (0.40, 0.73)	<0.0001
LLDAS on Treatment	—	—	—	—	—
None	7656	117	1.53	1.0 (Ref)	—
Not none, but<25%	10 555	134	1.27	0.83 (0.65, 1.06)	0.14
25% to 50%	12 686	129	1.02	0.66 (0.51, 0.85)	0.0013
50% to 75%	18 151	133	0.73	0.48 (0.37, 0.61)	0.0010
75%+	13 141	82	0.62	0.40 (0.30, 0.54)	<0.0001

remission reduced organ damage. LLDAS on treatment >50% of the time, which led to a 50% reduction in organ damage, is an easier goal to achieve (3 times more person-months observed in our cohort) and more realistic as a clinical trial outcome.

Acknowledgements The Hopkins Lupus Cohort is funded by NIH AR 43727 and NIH AR 69572.

CS-29

CREATION OF A WEIGHTED SLICC SLE CLASSIFICATION CRITERIA AND COMPARISON WITH OTHER SLE CLASSIFICATION CRITERIA

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10.1136/lupus-2018-lsm.64

Background In previous validation work, the SLICC 2012 SLE classification criteria were more sensitive than the revised ACR-11 criteria, while both criteria had similar agreement with physician diagnoses. Both of these classification rules count each SLE manifestation equally. Our objective was to derive and test a classification rule which differentially weights the variable used in the SLICC classification rule. We also compared this rule to a recently proposed EULAR/ACR classification rule that also uses a weighted approach. [Costenbader KH, Johnson S, Aringer M. EULAR/ACR Classification Criteria Update for SLE. Presented at the 2017 ACR/ARHP Annual Meeting, San Diego CA, November 4–8, 2017].

Methods The physician-rated patient scenarios used to develop the 2012 SLICC classification criteria were re-employed to devise a weighted classification rule. A multiple linear regression model was constructed with the 2012 SLICC criteria variables as predictors and the binary outcome (physician classification of SLE) as the outcome. Weights for each criteria were generated by multiplying each criteria's coefficient by 100 and rounding to the nearest integer. The 'Direct Coombs' criteria was deleted for simplicity. Weights for remaining manifestations were: acute cutaneous (26), chronic cutaneous (12), oral ulcers (16), arthritis (9), serositis (16), renal without biopsy (9), neurologic (9), hemolytic anemia (1), leukopenia or lymphopenia (14), thrombocytopenia (15), alopecia (9), ANA (17), anti-dsDNA (19), anti-Sm (16), antiphospholipid antibodies (8), low complement (11). Classification cutoff was the score that maximized overall agreement (i.e., the sum of sensitivity and specificity) of the new weighted criteria with physician diagnosis. Patients with lupus nephritis or the new weighted classification rule of 56 or more with at least one clinical component and one immunologic component were classified as SLE. We evaluated the performance of this revised SLICC criteria on an independent set of patient scenarios and compared this to the performance of the older revised ACR criteria, the previous SLICC 2012 criteria, and the newly proposed EULAR/ACR criteria.

Results Table 1 shows the performance of the four classification rules. There was no statistically significant difference between any pair of rules with respect to overall agreement with the physician diagnosis.

Conclusions The two newly derived weighted classification rules did not perform better than the existing list-based rules in terms of over-all agreement. Since the list-based rules are easy to calculate, they may be preferred in most clinical settings.

Abstract CS-29 Table 1 Sensitivity and specificity of four different SLE classification rules based on physician diagnoses of patient scenarios

Classification Rule	Sensitivity (n=349)	Specificity (n=341)	Overall Agreement (n=690)
Revised ACR-11	290 (83%)	326 (96%)	616 (89%)
SLICC 2012	340 (97%)	288 (84%)	628 (91%)
Proposed EULAR/ACR	317 (89%)	302 (90%)	619 (90%)
Weighted SLICC 2012 criteria	310 (88%)	304 (89%)	614 (89%)

Acknowledgements Presented on behalf of SLICC. The Hopkins Lupus Cohort is funded by NIH AR 43727 and NIH AR 69572.

CS-30

SLEEP DISTURBANCES AMONG WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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10.1136/lupus-2018-lsm.65

Background Sleep disturbances (SD) are reported to be common in SLE, but relatively few studies have addressed the issue. We examined the frequency and severity of self-reported SD among individuals with SLE and predictors of SD.

Methods Data were from the National Data Bank for Rheumatic Diseases (NDB), for which participants complete questionnaires every 6 months. In one questionnaire, items about the presence of physician-diagnosed obstructive sleep apnea (OSA) and restless-leg syndrome (RLS), symptoms of OSA and RLS, and the Medical Outcomes Study Sleep Scale (MOS-S) were included. The MOS-S yields 5 subscales; results are shown here only for one (Sleep Problems Index I, SPI-I). Frequencies of reports of OSA, RLS, and RLS symptoms were tabulated. Multivariate regression analyses identified independent predictors of OSA and RLS (logistic regression) and SPI-I scores (linear regression). Potential predictors included age, race, education, smoking, Rheumatic Disease Comorbidity Index (RDCI),¹ asthma, obesity (BMI ≥ 30 kg/m²), disease duration, pain, prednisone and other medication use, and disease activity (Systemic Lupus Activity Questionnaire, SLAQ²) and damage (Brief Index of Lupus Damage, BILD³).

Results Subjects (n=362) were mean age 61 $\pm$ 13 years and had SLE duration of 26 $\pm$ 13 years. 23% reported physician-diagnosed OSA and 20% RLS, compared to ~2%–4% and ~10%, respectively, in the general population. 18% and 34% had symptoms of OSA and RLS, respectively. Mean SPI-I was 39.6 ($\pm$ 20.2), >0.5 standard deviation higher (worse) than a population mean. Independent predictors of diagnosed OSA were greater age, obesity, asthma, RDCI, and disease activity (table 1). Predictors of RLS symptoms were RDCI and disease activity (table 1). Worse scores on SPI-I were associated with younger age, low education, higher RDCI, smoking, and greater pain and disease activity (table 1).

Conclusion Both OSA and RLS were more common in SLE than in the population; SPI-I scores were also worse. Some predictors of SDs were similar to predictors in the population (age, obesity), but disease activity was also associated with SD.