

¹³Department of Medicine, Division of Clinical Immunology and Rheumatology, University of Alabama at Birmingham, Birmingham, AL, USA; ¹⁴Department of Rheumatology, Johns Hopkins University School of Medicine, Baltimore, MD, USA; ¹⁵Arthritis Research UK Centre for Epidemiology, Centre for Musculoskeletal Research, Institute of; ¹⁶Inflammation and Repair, Manchester Academic Health Sciences Centre, The University of Manchester, Manchester, UK and NIHR Manchester Musculoskeletal Biomedical Research Unit, Central Manchester University Hospitals NHS Foundation Trust, Manchester Academic Health Science Centre; ¹⁷Lupus Research Unit, The Rayne Institute, St Thomas' Hospital, King's College London School of Medicine, London, UK; ¹⁸Feinstein Institute for Medical Research, Manhasset, NY, USA; ¹⁹Division of Rheumatology and Immunology, Department of Medicine, University of North Carolina, Chapel Hill, NC, USA; ²⁰Department of Medicine, West Penn Allegheny, Pittsburgh, PA, USA; ²¹Northwestern University and Feinberg School of Medicine, Chicago, IL, USA; ²²Department of Rheumatology, University Hospital Lund, Lund, Sweden; ²³Department of Rheumatology and Centre for Rheumatology Research Fossvogur Landspítali University Hospital, Reykjavik, Iceland; ²⁴Lanarkshire Centre for Rheumatology, Hairmyres Hospital, East Kilbride, Scotland UK; Autoimmune Disease Unit, Department of Internal Medicine, Hospital Universitario Cruces, University of the Basque Country, Barakaldo, Spain; ²⁵Emory University, Atlanta, Georgia, USA; ²⁶UCSD School of Medicine, La Jolla, CA, USA; ²⁷Division of Rheumatology, Department of Internal Medicine, Istanbul Medical Faculty, Istanbul University, Istanbul, Turkey; ²⁸Unit for Clinical Therapy Research (ClinTRID), The Karolinska Institute, Stockholm, Sweden; ²⁹Josep Font Autoimmune Diseases Laboratory, IDIBAPS, Department of Autoimmune Diseases, Hospital Clinic, Barcelona, Spain; ³⁰Medical University of South Carolina, Charleston, South Carolina, USA; ³¹Department of Rheumatology Rigshospitalet, Copenhagen University Hospital, Copenhagen, Denmark; ³²University of Manitoba, Winnipeg, Manitoba, Canada; ³³Columbia University Medical Centre, New York, USA; ³⁴Dept. of Rheumatology, Kantonsspital, Schaffhausen, Switzerland

10.1136/lupus-2016-000179.89

Background The objective of this study was to describe the frequency of myocardial infarction (MI) prior to the diagnosis of SLE and within the first 2 years of follow-up.

Materials and methods The SLICC atherosclerosis inception cohort enters patients within 15 months of SLE diagnosis. MIs were reported and attributed to a specialised vascular event form. MIs were confirmed by one or more of the following: abnormal EKG, typical or atypical symptoms with EKG abnormalities and elevated enzymes (≥ 2 times ULN), or abnormal stress test, echocardiogram, nuclear scan or angiogram. Descriptive statistics were used.

Results 31 of 1848 patients that entered the cohort had an MI. Of those, 23 patients had an MI prior to SLE diagnosis or within the first 2 years of disease. Of the 23 patients studied 60.9% were female, 78.3% were Caucasian, 8.7% Black, 8.7% Hispanic and 4.3% other. The mean age at SLE diagnosis was 52.5 ± 15.0 years. Of the 23 MIs that occurred, 16 MIs occurred at a mean of 6.1 ± 7.0 years prior to diagnosis and 7 occurred within the first 2 years of follow-up. Risk factors associated with early MI in univariate analysis are male sex, Caucasian, older age at diagnosis, hypertension, hypercholesterolemia, family history of MI and smoking. In multivariate analysis only age (OR = 1.06 95% CI: (1.03, 1.09)), hypertension (OR = 5.01, 95% CI: (1.38, 18.23)), hypercholesterolemia (OR = 4.43, 95% CI: (1.51, 12.99)) and smoking (OR = 7.50, 95% CI: (2.38, 23.57)) remained significant risk factors.

Conclusions In some lupus patients MI may develop even before the diagnosis of SLE or shortly thereafter, suggesting that there may be a link between autoimmune inflammation and atherosclerosis.

Acknowledgements This abstract is being submitted on behalf of the Systemic Lupus International Collaborating Clinics (SLICC) group.

CE-11 EPIDEMIOLOGIC EVIDENCE FOR THE EFFECT OF POVERTY ON SLE DAMAGE: A LONGITUDINAL STUDY

Edward Yelin*, Jinoos Yazdany, Laura Trupin. University of California, San Francisco, USA

10.1136/lupus-2016-000179.90

Background The relationship between poverty and SLE damage has been observed in several cross-sectional studies, but it remains unclear whether the loss of work due to SLE caused poverty or the reverse. Use of longitudinal data reduces the risk of reverse causation. The aim of the present study was to examine the effects of poverty at one point on subsequent damage, to assess whether the “dose” of poverty affects the extent of damage, and to evaluate the impact on damage of permanently exiting poverty.

Materials and methods Data are from the Lupus Outcomes Study (LOS). LOS participants were recruited from diverse sources in 2003 and followed through 2015 through annual structured surveys. In each year we characterised the respondents' poverty status based on household income and family size. Beginning in 2007, the survey included a validated measure of disease damage, the Brief Index of Lupus Damage. We used ordinary least squares regression to estimate the impact of 1) poverty in 2009, 2) the “dose” of poverty defined as the percentage of years in poverty between 2003 and 2009, and 3) the effect of permanently leaving poverty by 2009 on change in damage between 2009 and 2015, with and without adjustment for potential confounding variables (demographics, education, SLE duration, characteristics of health care, and health behaviours). To account for attrition and missing variables, multiple imputation was used.

Results In 2009, there were 783 respondents to the LOS annual survey, of whom 94% were female, 35% non-white, and 15% were in poverty. They were 49.8 (SD12.3) years of age and had had SLE for 16.9 (SD8.3) years. BILD damage scores averaged 1.9 (SD2.0, range 0–12). Table 1 shows the effect of poverty in 2007, “dose of poverty” between 2009 and 2015, and exiting poverty on change in damage, with and without adjustment. Those in poverty had greater increases in damage as did those continuously poor vs. poor some years vs. never poor. Exiting poverty was associated with change in damage scores closer to that among those who were never poor with the passage of as little as a year and smaller than those who remained poor. In all

Abstract CE-11 Table 1 Effect of poverty, percent of years in poverty, and exiting poverty on change in BILD damage scores, 2009–2015

	Poverty status		Percent of years in poverty			
	Poor	Not Poor	All Years	$\geq 50\%$ of Yrs.	$< 50\%$ of Yrs.	Never Poor
Unadjusted	2.02	1.33	2.52	1.59	1.54	1.32
Adjusted	1.97	1.34	2.45	1.45	1.49	1.34
	Exited poverty permanently					
	Stayed Poor	1 Yr. Ago	2-3 Yrs. Ago	5-11 Yrs. Ago	Total	Never Poor
Unadjusted	2.08	1.47	1.43	1.17	1.40	1.33
Adjusted	1.98	1.24	1.44	1.08	1.30	1.36

Cells are change in damage scores.

Adjusted models include demographics, duration, health care characteristics and health behaviours. Change in damage scores differs significantly by poverty status, percent of years in poverty, and exiting poverty, with and without adjustment ($p < .05$).

analyses, adjustment had minimal effect on results, indicating that the effect of confounding variables was minimal.

Conclusions The present study improves the certainty that poverty is etiologically related to damage and not an artefact of study design.

Acknowledgements Robert Wood Johnson Investigator in Health Policy Award, NIAMS P60 AR-053308

CE-12 COMPARISON OF CLASSIFICATION CRITERIA, SELF-ASSESSMENTS AND IMMUNOLOGIC PROFILES IN PATIENTS WITH INCOMPLETE AND SYSTEMIC LUPUS ERYTHEMATOSUS

¹Nancy J Olsen*, ¹Carl McAloose, ¹Jamie Carter, ²Bobby Kwanghoon Han, ³Indu Raman, ³Quan-Zhen Li, ⁴Duanping Liao. ¹Division of Rheumatology, Department of Medicine, Penn State MS Hershey Medical Centre, Hershey PA 17033; ²Division of Rheumatology, Department of Medicine, Cooper Medical School of Rowan University, Voorhees NJ; ³Department of Immunology, University of Texas Southwestern Medical Centre, Dallas TX USA; ⁴Department of Public Health Sciences, Penn State University, College of Medicine, Hershey PA 17033

10.1136/lupus-2016-000179.91

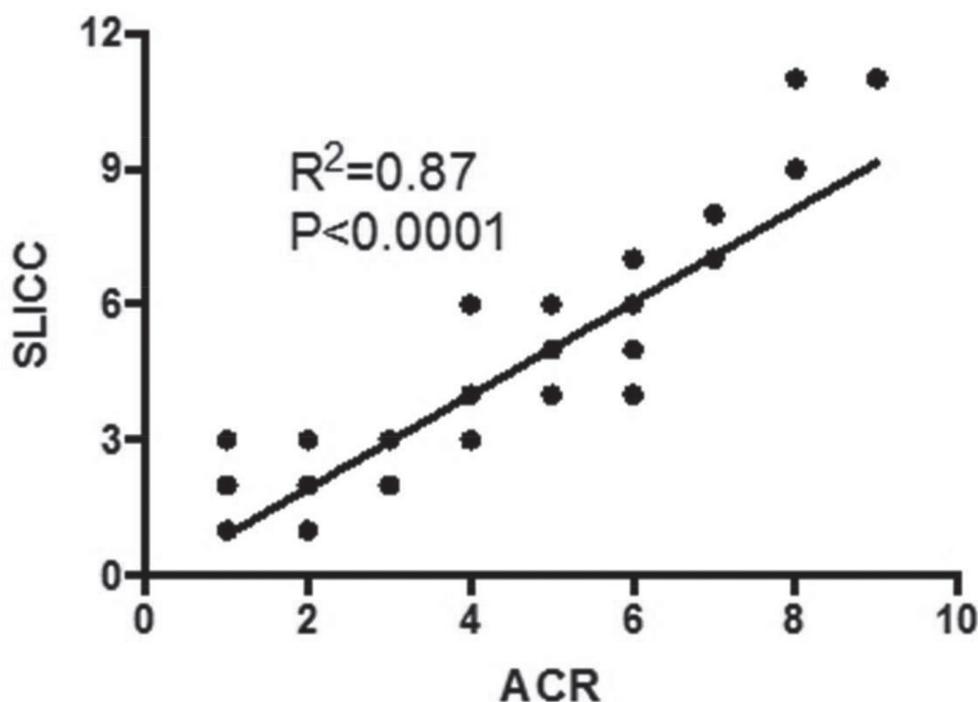
Background The syndrome of incomplete lupus erythematosus (ILE) likely includes individuals at risk for development of systemic lupus erythematosus (SLE). Studies of interventions to lower risk or prevent further disease in ILE are of interest. Design of such trials will require methods to classify patients and to assess risk. The goals of the present study were to evaluate performance of updated SLE classification criteria to define ILE and to probe for other features in these patients that might be useful as indicators of disease status. A long term goal is to develop prognostic multifaceted risk profiles that would have clinical applications.

Materials and methods Patients with ILE (N = 70) and SLE (N = 32) defined by the 1997 American College of Rheumatology (ACR) criteria were then reclassified using the 2012 Systemic Lupus International Collaborating Clinics (SLICC) criteria. Disease activity, patient self-assessments and levels of autoantibodies, soluble mediators and expressed Type I interferon (IFN) genes also were measured in the ILE and SLE patients and compared to healthy control (HC) individuals.

Results The two sets of classification criteria were highly correlated (Figure 1; $R^2 = 0.87$). ILE patients were older ($P = 0.0043$), with lower SLEDAI scores ($P = 0.023$) and greater dissatisfaction with their health status ($P = 0.034$) than SLE patients. Anti-C1q and sCD27 levels were correlated with ACR criteria and SLE Disease Activity Index (SLEDAI) scores ($P \leq 0.0004$). Three cytokines, IL-7, IL12p70 and IL-13, were lower in both ILE and SLE than in HCs. Two IFN-related cytokines IP = 10 and MCP-1, were higher in SLE than in ILE. Of three IFN genes measured, IFI27 showed the greatest difference between ILE and SLE.

Conclusions The 2012 SLICC SLE classification criteria likely can be used to define ILE in future research trials. Patients with ILE are somewhat dissatisfied with their condition, possibly related to anxiety about the lack of a clear diagnosis. Further patient-reported outcome studies in this population would be of interest. Reliable assessment of lupus risk will likely include demographic, clinical and immunologic features. Some of the latter may suggest novel approaches to early treatment.

Acknowledgements This project was funded, in part, with a grant from the Pennsylvania Department of Health using Tobacco CURE Funds. The Department specifically disclaims responsibility for any analyses, interpretations or conclusions. It was also supported in part by the National Institutes of Health, NIAMS U34 AR067392. The data entry assistance of Fan He is appreciated.



Abstract CE-12 Figure 1 Correlation between two SLE classification criteria, the 1997 ACR and 2012 SLICC sets, in 102 patients with either ILE or SLE. Values on each axis correspond to numbers of criteria in each of the sets. Significance determined using Pearson's R.