to those in membranous LN Type V (Figure 3). It outperformed conventional biomarkers (anti-dsDNA antibody, C3, C4) in discriminating the two different types of LN. Urinary ALCAM levels correlated positively with activity index (AI) in renal histopathology, indicating a role in guiding clinical management of LN.

Conclusions Urinary ALCAM is a promising biomarker for predicting renal histopathological changes in LN, which may serve as a valuable surrogate of renal biopsy. These findings should be confirmed in a larger cohort to further validate the utility of urinary ALCAM as a biomarker.

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IDENTIFICATION OF SERUM BIOMARKERS FOR SYSTEMIC LUPUS ERYTHEMATOSUS USING A LIBRARY OF PHAGE DISPLAYED RANDOM PEPTIDES AND DEEP SEQUENCING

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Abstract 165 Figure 1 Schematic diagram and workflow. A. The schematic for phage display screening and next generation sequencing. B. This study is composed of three major phases, i.e., screening phase, validation phase I, and validation phase II

Abstract 165 Figure 2 The candidate peptide biomarkers were discovered in the screening phase. A. The peptides which could differentiate SLE patients from healthy controls were discovered using next generation sequencing. B. Reactivity of the candidate peptides with SLE patients and health controls. C. Receiver operating characteristic curve analysis of the candidate peptides between SLE patients and health controls

Abstract 165 Figure 3 Four candidates of SLE serum biomarker were confirmed in validation phase. A. Reactivity of the serum biomarkers with sera of SLE patients and healthy controls in the ELISA validation. B. Receiver operating characteristic curve analysis of the four candidate peptides with SLE patients and health controls

Abstract 165 Figure 4 Assessment of the specificity of the four biomarkers with other related auto-immune diseases. Comparison of the SLE patients, healthy controls and other auto-immune patients, i.e., RA, ADM, BD and AS. Asterisks indicate statistical difference as compared to the SLE group (p

Abstract 165 Figure 5 Receiver operating characteristic curve analysis using the combinational panel of the four biomarkers. The best model and the classifier for SLE against healthy controls. Based on the cross validation, a best model [(3.479 expression level of SLE2018Val001) + (13.131 expression level of SLE2018Val002) (0.051 expression level of SLE2018Val006) + (7.517 expression level of SLE2018Val008) - 10.263] was generated with the final 4 peptide panel using generalized linear model from the R language

Abstract 165 Table 1 Characteristics of study participants in the screening phase

Variable	SLE count (n=50)			Health count (n=50)		
	No.	Mean	%	No.	Mean	%
Age, years						
Mean		37.8			38.1	
Interquartile		30-45			23-53	
Sex						
Male	5		10	5		10
Female	45		90	45		90
Disease Duration						
Median		4.00				
Interquartile		1.00-8.96				
SLEDAI						
Median		7				
Interquartile		5-13				
Hypocomplementemia			77.5			
Anti-dsDNA positive			38.7			
Antinuclear antibody positive			44.0			
Clinical Manifestation						
Fever			41.9			
Malar Rash			34.9			
Arthralgia			27.9			
Pulmonary Artery						
Hypertension			7.0			
Lupus Nephritis			67.4			
Leukocytopenia			27.9			
Thrombocytopenia			11.6			
Sjogren Syndrome			7.0			
Concurrent Medicine Use						
Glucocorticoids			88.4			
Hydrochloriquine			39.5			
Mycophenolate mofetil			11.6			
Cyclosporin			0.0			
Tacrolimus			4.7			
Cyclophosphamide			2.3			
Aspirin			18.6			
Calcitrol			76.7			

SLEDAI : Systemic Lupus Erythematosus Disease Activity Index.

Abstract 165 Table 2 Characteristics of study participants in the validation phase-I

Variable	SLE count (n=60)			Health count (n=60)		
	No.	Mean	%	No.	Mean	%
Age, years						
Mean		41.4			41.2	
Interquartile		30-51			23-57	
Sex						
Male	9		15	10		16
Female	51		85	50		84
Disease Duration						
Median		0.26				
Interquartile		0.08-1.03				
SLEDAI						
Median		7				
Interquartile		4-10				
Hypocomplementemia			78.0			
Anti-dsDNA positive			50.0			
Antinuclear antibody positive			16.0			
Clinical Manifestation						
Fever			10.0			
Malar Rash			15.0			
Arthralgia			21.7			
Pulmonary Artery			1.7			
Hypertension						
Lupus Nephritis			65.0			
Leukocytopenia			38.3			
Thrombocytopenia			13.3			
Sjogren Syndrome			5.0			
Concurrent Medicine Use						
Glucocorticoids			84.3			
Hydrochloriquine			52.9			
Mycophenolate mofetil			7.8			
Cyclosporin			3.9			
Tacrolimus			2.0			
Cyclophosphamide			5.9			
Aspirin			23.5			
Calcitrol			86.3			

SLEDAI : Systemic Lupus Erythematosus Disease Activity Index.

Abstract 165 Table 3 Characteristics of study participants in the validation phase-II

Variable	No.	Mean	%
SLE	15		
Age, years		35.9	
Male	2		13
Female	13		87
RA	7		
Age, years		53.2	
Male	2		28
Female	5		72
DM	9		
Age, years		51.0	
Male	3		33
Female	6		66
BD	20		
Age, years		34.3	
Male	9		21
Female	11		79
AS	15		
Age, years		36.1	
Male	11		73
Female	4		27
Health	20		
Age, years		40.8	
Male	0		0
Female	20		100

The abbreviations used are as follow: RA, rheumatoid arthritis ; DM, dermatomyositis; BD, behcet disease; AS, ankylosingspondylitis.

Background Systemic lupus erythematosus (SLE) is a chronic, complex autoimmune disorder characterized by the production of autoantibodies and heterogeneous clinical presentation. Biomarkers are in urgent need for the accurate diagnosis of the disease.

Methods SLE serum autoantibodies were discovered and validated using serum samples from independent sample cohorts encompassing 306 participants divided into three groups, i.e., healthy, SLE patients, and other autoimmune diseases. To discover biomarkers for SLE, a phage displayed random peptide library (Ph.D. 12) and deep sequencing were applied to screen specific autoantibodies in a total of 100 serum samples from 50 SLE patients and 50 healthy controls. A statistical analysis protocol was setup for the identification of peptides as potential biomarkers. For validation, ten peptides were analyzed using enzyme linked immunosorbent assays (ELISA) in two independent cohorts.

Results For the screening phase, a total of 116 peptides were highly enriched by the sera of SLE patients as compared to that of the health controls. Further validation showed that using a set of four peptides panel could achieve an AUC of 0.86. Among the four peptides, two of them were further confirmed in an independent group of patients with SLE and other autoimmune diseases.

Conclusions We demonstrate that a M13 phage displayed random peptide library in combination with deep sequencing can be used to identify peptides that could be specifically recognized by IgG in the sera of SLE patients.

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THE INCREASING PREVALENCE OF SLE, 2001–2011: UNITED STATES NATIONWIDE POPULATION-BASED ESTIMATES

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Background The prevalence rate of SLE in previous reports has widely varied by as much as 12-fold over the years, which is likely due to variabilities in study populations, case definitions, and case ascertainment sources. Previous studies covered relatively small populations such as city, county, state, or Medicaid. To our knowledge, there are no reports of SLE prevalence covering the entire US population. Although, elegant CDC registries covering several counties in 5 states provide valuable information on SLE prevalence, there is an important unmet need to estimate the national prevalence of SLE.

Methods We used ambulatory physician visits derived from the National Ambulatory Medical Care Survey and the National Hospital Ambulatory Medical Care Survey databases based on a multistage probability design to enumerate encounters that represent SLE using ICD-9 code 710.0. We then estimated the number of SLE patients from these encounters. SAS was used to determine the mean and 95% CI of SLE prevalence, overall and by sex, race/ethnicity, age, and geographic region. We calculated the cumulative percent change in prevalence between 2001 and 2011 (Chi-square test), modeled trends, and calculated the annual percent change using Joinpoint regression analysis.

Results The overall prevalence rate of SLE during 2001–2011 is 86.4 per 1 00 000 persons (95% CI, 69.6–103.3). The annual SLE prevalence increased 6% (95% CI, 2.5–9.5) each year from 2001 through 2011, with cumulative increase of 59.4% during this period (72.2 in 2001 to 115.1 in 2011, $p < 0.0001$). The period prevalence of SLE during 2001–2011 is 9-fold higher in females (137.5 [95% CI, 109.2–165.9]) than males (15.2 [95% CI, 8.0–22.4]). Non-Hispanic black persons had the highest prevalence (200.8 [95% CI, 125.9–275.8]). There were substantial variations in SLE prevalence by geographic regions: persons living in the West had the highest prevalence (108.9), followed by South (89.5), Northeast (79.4), and Midwest (65.7).

Conclusions Analysis of national survey data reveals an increasing trend in SLE prevalence across the US during 2001–2011, which may reflect an increased recognition of SLE, changes in physicians coding practices, decreasing mortality, or actual increases in the prevalence. Lack of verification of SLE diagnosis by rheumatologists is a major limitation of this study, especially since there is discordance even among rheumatologists as to what constitutes a diagnosis of lupus. Nevertheless, our data show demographic and regional differences in SLE prevalence across the US, with the highest prevalence in black persons and females, as reported previously, and in the West.

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