Materials and methods  The 1000 Faces of Lupus is a multicenter Canadian cohort of over 2000 patients. Sociodemographics, ACR classification criteria (ACRc), autoantibodies, disease activity scores (SLEDAI), Systemic Lupus International Collaborating Clinics damage index (SDI) scores, and treatments are collected using standardised tools. Ethnicity was self-reported. Asian subgroups were divided by origin country into East Asian (EA), Southeast Asian (SEA), South Asian (SA) and Central Asian (CA). Baseline data for Asians and Caucasians were abstracted and cross-sectional univariate analyses including t-tests, one-way ANOVA, and chi-square tests were performed.

Results  There were 334 Asians (EA = 176, SEA = 78, SA = 78, CA = 2), and 1275 Caucasians. CA were excluded. Mean Asian onset age was younger (EA = 23 ± 13 years; SEA = 21 ± 10 years; SA = 20 ± 11 years, Caucasian 33 ± 15 years, p < 0.001), but this was due to very frequent childhood onset in Asians (EA = 49%; SEA = 51%; SA = 61%) compared to Caucasians (17%, p < 0.001) (Figure 1). Over 40% of Asians were immigrants, and a higher proportion were males (EA = 15%; SEA = 16%; SA = 19%) compared to Caucasians (10%, p = 0.008). More Asians (90%) completed high school compared to Caucasians (83%, p = 0.007). Income was similar between all Asian subgroups and Caucasians. ACRc and SLEDAI scores were not different, but nephritis was more frequent in all Asians: (EA = 57%; SEA = 63%; SA = 51%) compared to Caucasians (33%, p < 0.001). Asians were more frequently (ever) seropositive: (dsDNA+: EA = 62%; SEA = 63%; SA = 78%; Caucasians 52%, p < 0.001), (antiSm+: EA = 31%; SEA = 50%; SA = 30%; p = 0.01, Caucasian 19%, p < 0.001). (antiRNP+: EA = 20%; SEA = 32%; SA = 22%; p = 0.03, Caucasians 16%, p < 0.001). Treatment with prednisone (EA = 55%; SEA = 67%; SA = 65%), cyclophosphamide (EA = 13%; SEA = 21%; SA = 20%), and mycophenolate (EA = 15%; SEA = 19%; SA = 9%) was more frequent in Asians compared to Caucasians (40%, 10%, 8%, respectively, p < 0.001 for all) likely reflecting renal disease.

Mean disease duration in Asians was 8 years but most had no damage (SDI = 0, EA = 66%; SEA = 64%; SA = 79%) compared to Caucasians (47%, p < 0.001).

Conclusions  In this analysis comparing Asian ethnic subgroups, we found only subtle differences between EA, SEA, and SA with SLE; as expected disease appeared more severe than in Caucasians. However, a strikingly high proportion of all Asians had onset in childhood. Along with the high proportion who were new Canadians, this suggests the potential for a growing burden of SLE in this population. Future studies of outcomes and optimal treatments are indicated.

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Abstract CE-24 Figure 1  Mean Age at SLE Diagnosis by Site (Paediatric or Adult)
Abstracts

patients with cSLE. 2) Identify both baseline and disease course (time-varying) predictors of damage trajectory.

Methods Single centre, retrospective, inception cohort. We included 473 patients who were diagnosed and followed, from 1st January 1985 to 30th September 2011. Patients had to be <18 years at diagnosis, have satisfied the ACR classification criteria for SLE, were treated for <3 months with steroids or an immunosuppressant or any other disease, and have had at least 3 visits. Longitudinal childhood data was obtained from our database while adulthood data was obtained from either a research database or patients’ charts. Clinical information at every visit was collected: for SLE disease activity index 2000 (SLEDAI2K), the SDI, laboratory results, and medications. Predictors were identified using a weighted generalised estimating equation (WGEE). Time-varying predictors: disease activity, individual items of SLEDAI2K, corticosteroid, immunosuppressant and antimarial exposures, were lagged by 6, 12, 18 and 24 months prior to each visit.

Results 67/473 (14%) patients were lost to follow-up. There were 14097 visits, 3290 patient-years. The median follow-up duration was 5.5 years, median age at diagnosis was 14.1 years and median age at last visit was 19.5 years (range 6.0–41.9 years). 67% of patients were >18 years old at last follow-up. The predicted average population damage was 0.7 at 5 years, 1.3 at 10 years, 1.9 at 15 years, 2.3 at 20 years and 2.7 at 25 years. Catastact (14%), avascular necrosis (10%) and osteoporosis (5%) were the commonest damage items. Only 2 had myocardial infarctions. Life-threatening major organ manifestations predicted higher initial damage but the accrual slowed down over time. Higher prednisone dose (12, 24 months before) and the use of cyclophosphamide (6, 12,18, 24 months before) predicted an increased damage trajectory at current visit. Antimalarial exposure (6 months before), mucosal ulcers (6, 12, 18, 24 months before) and pericarditis (6 months before) were protective against an increase in damage trajectory.

Conclusion Patients with cSLE accrue damage steadily throughout their disease course into adulthood. Baseline factors that predicted higher initial damage and influenced damage trajectory. SLE clinical features and therapeutic exposures during the course of disease, predicted a change in damage trajectory.

Methods Single-centre longitudinal inception cohort of cSLE patients (onset < 18 years) diagnosed and followed from Jan 1985 to Sep 2011. Paediatric data was obtained from our institutional cSLE database and adult data from the Toronto Lupus database or from rheumatologists’ offices. Longitudinal disease trajectory was constructed using data from every clinic visit in the 1st 10 years after diagnosis. Longitudinal SLE activity is a latent construct that is imperfectly measured with SLE disease activity index 2000 (SLEDAI2K) and prednisone exposure. SLE-DAI2K and prednisone use were then jointly modelled in a Bayesian growth mixture model (GMM). Baseline factors were tested for prediction of class membership.

Results 473 patients were included. 82% were female, median age of diagnosis was 14.1 years. There were 11992 visits, 2666 patient years. 67% of the population had transferred to adult care. Mean population SLEDAI2K and prednisone trajectories of cSLE patients showed rapid decline to low activity levels within 2 years after diagnosis. Joint GMM showed 5 latent classes in this cohort of cSLE patients. Class 1 patients (6%) have chronic moderate-high disease activity, class 2 (12%) had moderate initial disease activity and continued moderate long-term prednisone use, class 3 (17%) had initial high disease activity but achieved long-term remission, class 4 (19%) had high initial disease activity but relapsed later, class 5 (45%) had chronic low-grade disease activity. Across all classes, there was chronic use of prednisone (at least 5–10 mg/day) among cSLE patients in the first 10 years after diagnosis. Baseline major organ involvement, ethnicity, age at diagnosis and the number of baseline ACR criteria predicted probability of membership in different classes. Class 1 was associated with the most average damage accrual while class 5 was not associated with significant average damage accrual even after 10 years.

Conclusions cSLE patients could be sub-classified into 5 distinct classes of disease activity trajectories. Baseline and demographic factors predicted membership in the distinct disease classes. Different disease classes were associated with different patterns of damage trajectories.

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FROM CHILDHOOD TO ADULTHOOD: IDENTIFYING LATENT CLASSES OF DISEASE ACTIVITY TRAJECTORIES IN CHILDHOOD-ONSET SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background Although SLE patients are thought to follow different patterns of disease courses, no information is available about the longitudinal disease activity or the number of possible different disease courses. This study sought to: 1) Assess for distinguishable differences in disease activity trajectories in childhood-onset SLE (cSLE) patients; 2) Identify factors predictive of membership in different classes and 3) Assess if different disease activity trajectories are associated with different damage trajectories.

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Methods Single-centre longitudinal inception cohort of cSLE patients (onset < 18 years) diagnosed and followed from Jan 1985 to Sep 2011. Paediatric data was obtained from our institutional cSLE database and adult data from the Toronto Lupus database or from rheumatologists’ offices. Longitudinal disease trajectory was constructed using data from every clinic visit in the 1st 10 years after diagnosis. Longitudinal SLE activity is a latent construct that is imperfectly measured with SLE disease activity index 2000 (SLEDAI2K) and prednisone exposure. SLE-DAI2K and prednisone use were then jointly modelled in a Bayesian growth mixture model (GMM). Baseline factors were tested for prediction of class membership.

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Conclusions cSLE patients could be sub-classified into 5 distinct classes of disease activity trajectories. Baseline and demographic factors predicted membership in the distinct disease classes. Different disease classes were associated with different patterns of damage trajectories.