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 standardized 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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Background Outcomes of patients with cSLE into adulthood are poorly understood. There is no information about the longitudinal trajectory of damage in cSLE patients. We undertook this study to: 1) Determine the longitudinal damage trajectory--as measured by the ACR/SLICC SLE damage index (SDI)-- in patients with childhood-onset systemic lupus erythematosus (CSLE) from childhood to adulthood.

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.
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 for 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 anti-malarial exposures, were lagged by 6, 12, 18 and 24 months prior to each visit.

**Results** 67/473 (14%) 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. Cataract (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.