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 each 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-inflammatory 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. Catastrophic damage (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 the course of disease 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.
Antimalarials (AMs) have shown to exert a thromboprotective effect in SLE patients, but studies thus far have been limited to North American and European patients. This study was conducted to assess whether a similar effect is observed in Latin American SLE patients.

Materials and methods SLE patients with a recent diagnosis (<2 years) recruited and followed longitudinally as part of the GLADEL cohort were examined to establish risk factors for thrombotic events (TEs) and the possible preventive role of AMs. The end-point of this study was thrombosis defined as either arterial or venous occurring after entry into the cohort.

Independent variables included were socio-demographic characteristics, clinical manifestations as measured by the ACR classification criteria, laboratory, history of previous TEs and hospitalisation. For descriptive purposes, patients were divided according to use or non-use of an AMs agent (chloroquine and/or hydroxychloroquine) based on each patient’s entire follow-up period during the study. Patients were classified as “users” if they had received AMs for at least 6 months, whereas “non-users” comprised patients who had received AMs for less than 6 months or who had never received them.

Treatment with AMs, glucocorticoids and anticoagulants along with hospitalizations were considered as time-dependent covariates. The effect of AM use on thrombosis after adjustment for potential confounders (variables known to affect thrombosis and the use of AMs) was examined using a multivariable Cox regression model. A backward selection algorithm was used to select the variables retained in the model with α-level to stay in the model set to 0.05.

Results Of the 1,480 patients included in the GLADEL cohort, 1,208 (82%) were considered AMs users with median exposure time of 42.1 months (Q1–Q3: 19.1–62.3). TEs occurred in 103 (7%) of the patients during a median follow up time since enrolment of 15.4 months (Q1–Q3: 4.6–38.2). The rate of thrombosis for AM users was 1.44 per 100 patient/years of follow-up vs. 3.01 for non-AM users (HR 0.55, 95% CI: 0.37–0.82).

After adjusting for potential confounders in the Cox proportional hazards regression model, the use of AMs was associated with a 43% reduction in the thrombosis rate (HR 0.57, 95% CI: 0.38–0.85).