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 antimalarial 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 was19.5 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.

CE-26 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.

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. SLEDAI2K 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.

CE-27 EARLY PREECLAMPSIA IN SLE PREGNANCY

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Background Early preeclampsia is a serious pregnancy complication characterised by abnormal placentation, diffuse maternal endothelial cell dysfunction, and requiring emergent delivery which may be very premature. SLE has been associated with preeclampsia, but little is known about the risks of early onset preeclampsia – a pregnancy morbidity associated with stroke, placental abruptions, and perinatal death.

Materials and methods SLE was defined as ≥2 visits in the Swedish National Patient Register (NPR, inpatient and outpatient specialist) with ≥1 SLE diagnosis from a specialist who typically treats, manages, or diagnoses SLE in Sweden (2001–2012). General population comparators (non-SLE) were sampled from the Total Population Register. We restricted to singleton births in the Medical Birth Register (MBR). Preeclampsia was defined by first ICD-coded visit during pregnancy in NPR and early-onset

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defined as <34 weeks. Obesity (BMI > 30), age, smoking, and pregestational hypertension and diabetes were defined using NPR and MBR. NPR ICD-coded visit and/or heparin dispensing during pregnancy from the Prescribed Drug Register (2006–2012) was a proxy for antiphospholipid syndrome (APS). The association between early preeclampsia and SLE was estimated by multivariable-adjusted modified Poisson models for first, subsequent, and all births. Robust standard errors and preeclampsia history were accounted for in non-first-births analyses. We investigated effect modification by pregestational hypertension, examined residual confounding by APS and misclassification of lupus nephritis as preeclampsia.

Results There were 742 births to women with SLE (343 first births) and 10484 births to women from the general population (4443 first births). Among the 32 pregnancies with early preeclampsia and SLE, 75% were first births and 34% were positive for the defined APS proxy. SLE was associated with a significantly increased risk of early preeclampsia for all, first, and subsequent births compared to non-SLE [RR = 7.3, (95% CI: = 4.5, 11.9), all births]. Although adjustment for APS proxy attenuated the association SLE remained statistically significantly associated with early preeclampsia (RR = 3.7, 95% CI: = 1.7, 7.9). Findings were similar among women with no pregestational hypertension, as well as in the absence of recent nephrology care. Risk ratios for early preeclampsia were smaller, but significant, for subsequent births compared to first and all births [RR = 2.8 (95% CI: = 1.2, 6.4) subsequent].

Conclusions Women with SLE are at increased risk of preeclampsia before 34 weeks gestation, and importantly, this increased risk may be independent of pregestational hypertension, APS, obesity, or smoking. Traditional risk factors alone may not explain the increased risk of early preeclampsia among women with SLE for first, subsequent, or any birth. Women with SLE during pregnancy should continue to be monitored carefully for early preeclampsia and future research is needed to identify what non-traditional preeclampsia factors might be causing this serious outcome.

CE-28 ANTIMALARIALS PROTECTS AGAINST THROMBOSIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE): LONGITUDINAL DATA FROM A LARGE LATIN AMERICAN COHORT

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Abstract CE-28 Table 1 Multivariable cox proportional hazard model: Time-to-thrombosis

Variable	HR	95% CI:
Antimalarials*	0.57	0.38–0.85
Gender	0.52	0.31–0.88
Previous Thrombosis	7.53	4.75–11.95
Age at enrolment (Spline)		
5 years increase at 20 years	0.92	0.81–1.04
5 years increase at 40 years	1.07	0.98–1.18
5 years increase at 50 years	1.67	1.12–2.35
Corticosteroids Dose*		
<7.5 mg/d vs. No	0.81	0.36–1.84
7.5 – 15 mg/d vs. No	1.00	0.46–2.16
≥15 – 60 mg/d vs. No	1.56	0.79–3.05
≥60 mg/d vs. No	3.15	1.43–6.94
Hospitalizations*	1.19	1.07–1.31

* Time dependent covariates.
**Variables considered as candidate for inclusion in the multivariable model but not selected in the final model were: ethnic group, medical coverage, hemolytic anaemia, renal disease, neurological disease, SLEDAI and SLICC-SDI at cohort enrolment and anticoagulant use (time-dependent).

Background 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:

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