Abstract 59 Figure 1 Pregnancy morbidity and live births divided in the three groups

risk group (GAPSS 6–11, $n=66$) and a high risk group (GAPSS12, $n=5$).

When considering patients who ever experienced PM while treated with SoC, all patients in the high risk group experienced PM, while patients in the medium group had a significant higher rate of PM when compared to the low risk group [29 (43.9%) patients Vs. 11 (15.3%), respectively; $p<0.001$]. When analysing the number of pregnancies in the three groups, patients in the high risk group had significantly lower live birth rates, when compared to the other groups [11 (40.7%) life births Vs. 100 (62.1%) and 137 (82.5%), respectively; $p<0.05$]. Furthermore, patients with medium risk group also had significantly lower live birth rates, when compared to the lower risk group ($p<0.001$).

Figure 1 resumes the results of PM and live births divided in the three groups.

Conclusions GAPSS might be a valuable tool in identifying patients at higher risk of developing any event of PM who might need additional therapeutic approach other than SoC.

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ASSOCIATION OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) GENETIC SUSCEPTIBILITY LOCI WITH LUPUS NEPHRITIS IN CHILDHOOD-ONSET AND ADULT-ONSET WITH SLE

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Background Lupus nephritis (LN) is one of the most common and severe manifestations of systemic lupus erythematosus (SLE). We tested the association of SLE-risk loci with LN risk in childhood- (cSLE) and adult-onset SLE (aSLE).

Methods Two Toronto-based tertiary care SLE cohorts included cSLE (diagnosed <18 y) and aSLE patients (diagnosed ≥18y). Patients met ACR and/or SLICC SLE criteria and were genotyped on the Illumina MEGA or Omni1 arrays. Un-genotyped SNPs were imputed (1000 Genomes Project). HLA alleles were imputed in the Europeans only (SNP2HLA). Ancestry was inferred using principal components. We identified those with and without biopsy confirmed LN. HLA and non-HLA additive SLE risk-weighted genetic risk scores (GRSs) were tested for association with LN risk in logistic models, stratified by cSLE/aSLE and ancestry. Stratified effect estimates were meta-analyzed.

Results Of 1237 participants, 572 had cSLE (41% with LN) and 665 had aSLE (30% with LN). Increasing non-HLA GRS was significantly associated with increased LN risk (OR=1.26; 95% CI: 1.09, 1.46, $p=0.0006$) as was increasing HLA GRS in Europeans (OR=1.55; 95% CI: 1.07, 2.25; $p=0.03$). There was a trend for stronger associations between both GRSs and LN risk in Europeans with cSLE compared with aSLE.

Conclusions We observed an association between known SLE-risk loci and LN risk in children and adults with SLE, with the strongest effect observed among Europeans with cSLE. Future directions will include incorporating SLE-risk SNPs specific to non-European ancestral groups and validating findings in an independent cohort.

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DIFFERENCES IN ORGAN SYSTEM INVOLVEMENT ACROSS RACIAL/ETHNIC GROUPS: RESULTS FROM THE CALIFORNIA LUPUS EPIDEMIOLOGY STUDY

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Background SLE is a chronic disease that affects many organ systems and can cause permanent damage. We sought to determine if there are differences in patterns of SLE organ system damage among racial/ethnic groups.

Methods Data derive from the baseline visit of the California Lupus Epidemiology Study (CLUES), an ongoing cohort of patients in the San Francisco Bay Area with confirmed SLE diagnoses. Participants provided access to medical records and had a visit with a study rheumatologist. Race/ethnicity (White, African American, Hispanic of any race, and Asian) was determined by patient report. Due to the small sample size, patients from other racial groups were excluded from this analysis ($n=5$). Disease damage was measured using the SLICC/ACR Damage Index (SDI), calculated at the study visit. We examined damage at the organ system level, using items from the SDI, and defined glucocorticoid-related damage as avascular necrosis, diabetes, cataracts, and osteoporosis. Logistic regression was used to estimate the prevalence of damage in each organ system and the prevalence of glucocorticoid-related damage by race/ethnicity controlling for current age. We ranked the prevalence of damage in each organ system that comprises the SDI for each racial/ethnic group.