**Results** Four SLE-risk genes were found to be associated with a decreased time until the first MI; PTPN22 (OR 1.61, p=0.041), NCF2 (OR 2.47, p=2.1×10^{-5}), STAT4 (OR 1.66, p=5.2×10^{-3}) and IL12A (OR 1.45, p=0.047) and were included in a PRS. The PRS was associated with a higher cumulative prevalence of MI in both the discovery cohort (p=1.1×10^{-3}, fig 1A) and replication cohort (p=7.7×10^{-3}, fig 1B). Exploring the PRS further in the replication cohort, patients in the high, compared to the low, PRS quartile were more often male (p=1.3×10^{-3}), and displayed higher prevalence of the ACR-1982 nephritis and immunological criteria (p=4.1×10^{-4} and p=0.036) (fig1C).

Analyzing combinations of the identified SNPs, we found the prevalence of MI to be further increased in patients homozygous for both NCF2+STAT4 (p_{discovery}=1.6×10^{-3}, p_{replication}=0.015) or STAT4+IL12A (p_{discovery}=3.0×10^{-3}, p_{replication}=0.036) (fig1D).

**Conclusion** A high polygenic risk score for MI in SLE is associated with an increased prevalence of myocardial infarction. If confirmed in prospective studies, our results suggest that genetic profiling may be useful for predicting MI in patients with SLE.