Background: Despite the decline in their incidence rates, the recurrent myocardial infarction (MI) events are associated with significant morbidity, short- and long-term mortality. Relative to our understanding of risk for first events, the aetiology of recurrent MI is poorly understood.

Methods: We used UK-Biobank, a large prospective cohort of 500,000 individuals, to investigate the genetic predisposition of recurrent MI. We performed a GWAS in 3386 UK-Biobank participants admitted to hospital due to MI at least twice within a period of 28 days - 1.5 years and 8567 controls with one unique hospital record with MI diagnosis or MI hospital admissions, which occurred outside the aforementioned period.

Results: In total, 215 variants representing 27 loci reached a suggestive significance level of $10^{-5}$. Among these, 17 loci have been implicated in coronary artery disease (CAD) and other cardiovascular phenotypes (e.g., KCNN2, KLF4, CACNB2, ADIPOR2, KLF5, PKD1L3), known CAD risk factors (blood pressure, CACNB2; lipid levels, ABHD4), cardiac remodelling (MAP3K5, SEMA3A), and abnormalities in platelets and coagulation (GRM7, KALRN, P2RY1).

Five of the identified genes (CHD7, IST1, KIAA1958, MAP3K5, UBFD1) were also found to be differentially expressed six months after a MI in 39 MI survivors (Greek Recurrent Myocardial Infarction Cohort) that had not experienced any recurrent event during that period (p-adj $\leq 10^{-5}$).

Conclusions: We identified 27 loci associated with increased risk of recurrent MI. We aim to identify independent datasets to replicate our findings, aiming to a greater understanding of recurrent MI determinants.

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