Abstracts

PLCE1 rs61866305 10 95902053 C 0.825 4.2E-20 33447 2.94 0.32
LINCO00424 rs12868493 13 22872349 C 0.784 3.1E-11 33413 −1.91 0.29
FBN5 rs8014161 14 92391398 T 0.639 4.3E-22 33088 −2.38 0.25
SRL rs11864324 16 4281391 T 0.779 3.0E-08 33636 −1.58 0.29
CFDP1 rs3851734 16 75371920 T 0.406 1.1E-08 33636 2.94 0.24
SMG6 rs1532292 17 2097483 T 0.618 2.9E-13 33768 1.81 0.25

Locus indicates the name of the gene in closest proximity to the lead variant.
sID = reference single nucleotide polymorphism cluster ID; CHR = chromosome; EA = effect allele; EAF = effect allele frequency; P = P-value (standard infinitesimal mixed model); N = effective number of participants; β = effect-size estimate expressed as a percentage; SE = standard error.

Disease traits. We observed no significant difference in odds ratio for MACE between the top and bottom quintiles for each PRS.

Conclusion We identified 30 genetic loci providing new candidate genes for exploration of biological mechanism of AoDs.

REFERENCES

THE ROLE OF IMPAIRED MYOCARDIAL MICROVASCULAR FUNCTION DYSGLYCEMIC PATIENTS WITH HEART FAILURE


Background Dysglycaemic (either diabetic or prediabetic) heart failure patients have worse outcomes than normoglycaemic heart failure patients. It is possible to quantify occult ischae- mic heart disease (IHD, either ischaemia on stress perfusion or infarction on late gadolinium enhancement) and myocardial microvascular function (by quantitative perfusion). We aimed to investigate whether excess risk in dysglycaemic patients with heart failure is mediated by occult ischaemic heart dis- ease or myocardial microvascular dysfunction.

Methods We recruited outpatients with a recent diagnosis of heart failure (LVEF < 50% on echocardiogram). Exclusion cri- teria included known previous myocardial infarction, revascu- larisation or angina. Patients were defined as dysglycaemic if criteria included known previous myocardial infarction, revascularisation or angina. Patients were defined as dysglycaemic if they had a previous diagnosis of diabetes or HbA1c >42
mmol/mol. Patients were followed up for major adverse cardiovascular events (MACE) including cardiovascular death, heart failure hospitalisation, non-fatal MI and non-fatal stroke. CMR studies were performed on a Siemens Prisma 3T scanner (Siemens Healthineers, Erlangen, Germany).

**Results** Of 343 patients, 176 were normoglycaemic and 167 dysglycaemic. During follow up (median 623 days) there were 35 MACE events in 30 patients, including 23 heart failure hospitalisations (6.7%), 4 strokes (1.1%), 7 cardiovascular deaths (2.0%) and 1 (0.3%) acute coronary syndrome. Univariate Cox regression analysis showed left ventricular ejection fraction (LVEF), right ventricular ejection fraction (RVEF), native T1, extracellular volume fraction, myocardial perfusion reserve (MPR) and the presence of occult IHD all to have significant association with MACE. However MPR was only associated with MACE in dysglycaemic patients (hazard ratio (HR) 0.19, 95% confidence interval (CI) 0.08–0.46, P<0.001) and occult IHD was only associated with MACE in normoglycaemic patients (HR 3.45, 95% CI 1.23–9.71, P=0.02) (figure 1). The relationship between MPR and MACE in dysglycaemic patients was still significant even after correction for LVEF, RVEF and Hba1c (HR 0.553, 95% CI 0.318–0.962, P=0.036).

**Conclusions** In patients with a recent diagnosis of heart failure, impairment of myocardial microvascular function is associated with adverse outcomes in dysglycaemic but not normoglycaemic patients, possibly explaining the excess risk in these patients. Further studies are needed to confirm these findings and establish if impaired microvascular function or associated outcomes can be altered by medical therapy.

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### Abstract 11

**A MEDICAL DEVICE-GRADE T2 PHANTOM FOR QUALITY ASSURANCE OF INFLAMMATION IMAGING BY CMR**

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**Introduction** Cardiovascular magnetic resonance (CMR) T2 mapping is key to quantifying myocardial inflammation. Use of T2 mapping in clinical studies is burgeoning but in the absence of a quality control system, single-center findings are not generalizable and longitudinal studies cannot reliably track alterations in T2 times reflecting the inflammatory state of the myocardium.

**Aim** We used our expertise gained from the development of the T1 Mapping and Extracellular Volume (T1MES) phantom, to develop a dedicated T2 mapping CMR phantom to medical device standards.

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**Abstract 11 Figure 1** (i) Schematic (not to scale) showing the internal and external phantom structure. (ii) Phantom front view showing isocentre line and liquid crystal display thermometer. (iii) T1 and T2 times in the T2 phantom as measured at 3T and 1.5T. Slow scan reference data for T1 times shown in green were obtained using a 5(3) 256-matrix RR = 900 ms variant of MOLLI adapted for native T1 mapping; T2 times in blue are obtained using a T2 mapping sequence (SSFP); T2 times in red were obtained by the manufacturer in Australia using a 1.4T Bruker minispec relaxometer at 22°C. Tube arrangement is such that the more temperature-dependent and therefore unstable long-T1 tubes are away from the corners and towards the middle of the 3X3 array. (iv) Exemplar T2 and T1 maps on a Siemens 3T Prisma clinical CMR scanner. (v) The 9 relaxometry scopes per tube explained. FA = Flip angle; GBCA = Gadolinium-based contrast agent; HDPE = high-density polyethylene; IRSE = inversion recovery spin echo; MOLLI = modified Look-Locker inversion recovery; myo = myocardium; PVC = polycarbonate; RR = inter-beat interval; SE = spin echo; SSFP = steady-state free precession; T = Tesla.