Abstract 8 Figure 1

obtained from the full scans. 200 scans were used as a testing set.

Results Task 1: The network successfully learned the 7 key points on the transaxial stack, and the scan planes it proposed closely matched those chosen by radiographers using dedicated localisers, with a median out-of-plane distance at the key points of only 3.4 mm (IQR 1.6–6.3) - less than half the slice thickness (8 mm) of the gold standard cines.

Task 2: The system was successful in segmenting the transaxial slices (Dice 0.91). Diagnostic accuracy was high for abnormalities including left and right ventricular dilatation (90.5%, 85.5%), LVH (85%) and ascending aorta dilatation (94.4%). The ROC AUC for diagnosing pleural effusions was 0.91.

Conclusion A neural network can use the standard transaxial stack of images to identify important pathology and accurately predict scan planes close to those of expert radiographers using dedicated localisers. This system could speed up the delivery of CMR and lead to faster and more dynamic scanning protocols.

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IDENTIFICATION OF THIRTY NOVEL LOCI FOR CARDIOVASCULAR MAGNETIC RESONANCE DERIVED AORTIC DISTENSIBILITY IN THE UK BIOBANK

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Introduction Cardiovascular magnetic resonance (CMR)-derived aortic distensibility (AoD) is a validated tool to measure arterial stiffness¹. This local stiffness marker is an independent predictor of cardiovascular events and all-cause mortality². Our knowledge of genes modulating aortic stiffness remains limited. We conducted genome-wide association studies (GWASs) for AoD in the ascending aorta (AA) and proximal descending aorta (PDA).

Methods 34,039 UK Biobank (UKB) participants of European ancestry with transverse cine images of pulmonary trunk and right pulmonary artery acquired using a 1.5T CMR scanner were included. AoD is defined as the relative change in cross-sectional aorta area for a given change in central pulse pressure, they were either manually derived or processed through a developed automated algorithm³. GWASs were performed using linear mixed models⁴ using ~7 million imputed variants adjusted for age, sex, height, weight, systolic blood pressure, aortic area segmentation method (manual/automated), imaging centre, genotype array, smoking, diabetes mellitus and top 10 principal components. Bioinformatic analyses were performed including investigation of trait pleiotropy. Polygenic risk scores (PRS) for AA and PDA were calculated using PRSice⁵ and tested for association with major adverse cardiovascular events (MACE) in the UKB cohort without imaging data (n=98,559).

Results and discussion Variants at 18 independent loci for AA and 16 loci for PDA AoD were genome-wide significant, $P < 5 \times 10^{-8}$ (table 1 and figure 1) with four shared loci between the two phenotypes. The heritability for both AA and PDA AoD was ~27%, much higher than arterial stiffness index (6%)⁶ but comparable to pulse wave velocity (36–40%)^{7,8}. We captured a modest percentage of the genetic variance for each trait (2.7% for AA; 2.1% for PDA). Several candidate genes are highlighted including *ELN* for both traits, with *ISL1* and *PCSK1* both involved in insulin regulation that may explain the interplay with diabetes^{9,10}. Variants at 13 loci were significant with blood pressure and/or coronary artery

Abstract 9 Figure 1 Miami plots of genome-wide association test statistics for aortic distensibility in the ascending (AA AoD) and descending aorta (PDA AoD). The red line indicates the P -value threshold for genome-wide significance (5×10^{-8}) while the blue line indicates P -value threshold for suggestive significance (1×10^{-5})

Abstract 9 Table 1 Genome-wide significant loci for aortic distensibility in the ascending aorta ($n = 33,346$) and proximal descending aorta ($n = 33,800$)

Ascending aortic distensibility									
Locus	rsID	CHR	Position (build 37)	EA	EA	P	N	β	SE
OTUD7B	rs67807996	1	149995265	G	0.604	4.7E-08	31109	-2.20	0.41
FIGN	rs35303331	2	164921770	A	0.767	2.4E-09	33201	2.79	0.46
ULK4	rs751328021	3	41971728	TTTCTATTTAATAGGAAATCCTATTGTG	0.870	4.0E-09	31513	-3.41	0.59
ADAMTS9-AS2/ MIR548A2	rs7638565	3	64723072	A	0.407	7.4E-11	33118	2.60	0.39
KALRN	rs4608635	3	123961496	C	0.342	3.6E-08	32019	-2.26	0.42
ARHGAP24	rs17399707	4	86716478	C	0.795	4.1E-08	32405	2.71	0.49
ANKH	rs9686327	5	15018537	G	0.819	8.2E-09	32939	-2.84	0.50
ISL1	rs540082300	5	51196504	C	0.603	3.5E-11	32939	2.66	0.40
PCSK1	rs764443335	5	95606712	AATG	0.645	1.6E-16	32824	-3.29	0.41
CEP120	rs115968765	5	122621332	C	0.732	1.1E-08	32725	2.53	0.44
LOC401324	rs342983	7	35464784	T	0.272	8.4E-10	31337	2.78	0.45
ELN	rs7795735	7	73429482	T	0.545	4.1E-40	33048	5.30	0.39
PI15	rs9721183	8	75781818	C	0.634	3.0E-12	31589	-2.83	0.41
HAS2	rs563877611	8	122649630	C	0.737	5.9E-10	30716	-2.78	0.46
SVIL	rs3006557	10	30090348	G	0.283	4.5E-08	33149	-2.33	0.43
MIR365A	rs7203968	16	14444230	G	0.833	2.3E-08	32480	2.96	0.52
CFDP1	rs4888421	16	75465339	A	0.403	1.1E-15	33082	-3.12	0.40
SRR	rs4548913	17	2209888	G	0.361	7.5E-13	32977	-2.87	0.41
Descending aortic distensibility									
Locus	rsID	CHR	Position (build 37)	EA	EA	P	N	β	SE
MASP1	3:186975537_AT_A	3	186975537	AT	0.924	1.8E-09	33222	2.77	0.46
FBXL7	rs529524362	5	15471344	G	0.664	1.2E-10	31606	1.69	0.26
PCSK1	rs10065122	5	95610520	T	0.550	1.6E-09	33468	-1.44	0.24
IGFBP3	rs1543270	7	46006323	C	0.546	4.6E-08	33560	-1.30	0.24
ELN	rs6943980	7	73424373	A	0.540	8.8E-21	33772	2.26	0.24
CCDC25	rs56228913	8	27616585	A	0.558	2.2E-09	33729	-1.42	0.24
PI15	rs9721183	8	75781818	C	0.634	1.1E-10	32019	-1.62	0.25
LRR69	rs10087555	8	92199899	T	0.312	5.0E-11	33520	1.71	0.26
KLHL38	rs7006122	8	124608614	T	0.635	2.7E-11	33333	1.67	0.25
JCAD	rs2369339	10	30168031	T	0.319	7.3E-11	33689	-1.65	0.26

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PLCE1	rs61886305	10	95902053	C	0.825	4.2E-20	33447	2.94	0.32
LINC00424	rs12869493	13	22872349	C	0.784	3.1E-11	33413	-1.91	0.29
FBLN5	rs8014161	14	92393198	T	0.639	4.3E-22	33088	-2.38	0.25
SRL	rs11864324	16	4281391	T	0.779	3.0E-08	33800	-1.58	0.29
CFDP1	rs3851734	16	75371920	T	0.406	1.1E-08	33636	-1.38	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.

rsID = 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.

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

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Background Dysglycaemic (either diabetic or prediabetic) heart failure patients have worse outcomes than normoglycaemic heart failure patients. It is possible to quantify occult ischaemic 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 disease or myocardial microvascular dysfunction.

Methods We recruited outpatients with a recent diagnosis of heart failure (LVEF < 50% on echocardiogram). Exclusion 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

Abstract 10 Figure 1 Univariate Cox regression analysis