hospitals between 1st January 2000 and 31st July 2014. This data was amalgamated according to the Algorithm for Comorbidities, Associations, Length of stay and Mortality (ACALM) study protocol using ICD-10 and OPCS-4 coding. Long term mortality analysis was performed using cox regression adjusting for demographics, comorbidities and Charlson Comorbidity Index.

Results Of 1,816,230 patients, there were 28,699 ACS, 36,719 HF and 52,812 AF patients with marital status. ACS was more common in males (64%) but an even split was seen in HF and AF. Males with ACS, HF and HF were significantly younger than females. Cox regression showed that married (OR0.77) or widowed (OR0.76) patients had lower long term mortality than single or divorced patients with ACS. Among those with HF and AF, married (OR0.70 HF; OR0.68 AF) and widowed patients (OR0.71 HF;OR0.69 AF) had significantly better mortality compared to singles. Divorcees had the worst mortality (OR1.31 HF;OR1.26 AF). As shown in table 1 and figure 1, widowed females had lower mortality compared with widowed males in the ACS, HF and AF groups. Interestingly, single males with HF had lower mortality compared to single females. In the AF group, married and divorced females had lower mortality than married and divorced males respectively.

Conclusion Understanding gender differences in marital status will further our understanding of the complex role of the support mechanisms that are required to best manage patients with ACS, HF and AF. Targeting patients with the worst outcomes with a greater focus on holistic care could lead to improved prognosis.

Conflict of Interest Nil

103 REAL WORLD HIGH-SENSITIVITY TROPONIN LEVELS IN AN ENTIRE HOSPITAL POPULATION: INSIGHTS FROM THE CHARIOT STUDY

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Background The use of high-sensitivity troponin (hs-cTn) is established in guideline-directed clinical practice to facilitate the diagnosis or exclusion of myocardial infarction (MI). The manufacturer provided 99th percentile for this assay is derived from a small population of relatively healthy individuals but is used as an "upper limit of normal" (ULN) in routine hospital practice. This raises the question: does this ULN actually represent the 99th centile of the distribution of the assay in an all comers hospital population, in most of whom there is no clinical suspicion of an MI?

Methods Twenty thousand consecutive patients attending this large University Teaching Trust who had biochemistry tests for any clinical reason, either as in- or outpatient had an hs-cTn assay as part of this study. In those patients in whom hs-cTn was not requested by their supervising clinician, the result of the assay was nested, and never revealed to doctor or patient. Ethical approval for this method required special permission from the national Confidentiality Advisory Group following support from the British Cardiac Patients Association. The Beckman Coulter Access AccuTnI+3 assay was used

Abstract 103 Table 1 Percentage of inpatients with hs-cTn above the ULN (specialities with more than 100 patients)

Source	Number of patients	Percentage with hs-cTn above ULN
Acute medical admissions	610	11.9%
Medicine, Elderly care & Rehab	358	11.7%
Day case	721	1.4%
Cardiovascular & Thoracic	426	16.7%
Neurosciences	338	6.8%
Haematology & Oncology	862	4.5%
Surgery & obstetrics	1263	3.9%
Critical care	123	39.0%

Abstract 103 Table 2 Frequency of hs-cTn elevation in outpatients with a coded co-morbidity

Co-morbidity	Number of patients	Percentage with hs-cTn above ULN
Heart failure	408	9.8%
Atrial fibrillation	481	7.0%
Ischaemic heart disease / angina	883	5.4%
Diabetes mellitus (any type)	938	4.1%
Hypertension	2284	3.8%
Dyslipidaemia	769	5.4%
Chronic obstructive pulmonary disease	523	3.6%
Rheumatoid arthritis	290	2.1%
Obesity	1153	2.2%

throughout. These data were combined with the source location for the blood test, as well as the presence of coded co-morbidity since the year 2000.

Results There were 18171 patients left once patients discharged with a diagnosis of MI (122) and those suspected of MI (1707) by the clinical team were excluded. There were 4759 inpatients, 9280 outpatients and 4132 patients in the emergency department with 7.3%, 2.0% and 7.4% of these having an hs-cTn level above the manufacturer-provided ULN respectively. Table 1 demonstrates the prevalence of hs-cTn levels above the ULN in hospitalised patients by speciality. In terms of outpatients; cardiothoracic, renal and oncology/haematology patients were the most likely to have an hs-cTn above the ULN with 7.4%, 4.9% and 2.6% respectively above this level. There were 4265 (46%) outpatients in whom there was at least one coded co-morbidity present. These patients were more likely to have an hs-cTn above the ULN than those without a coded co-morbidity (2.8% versus 1.2%, P<0.0001). Table 2 demonstrates the prevalence of outpatients with an hs-cTn above the ULN by coded co-morbidity.

Conclusion These data suggest that application of a manufacturer-derived 99th centile for hs-cTn assay to a hospital population may be flawed, particularly if any assumption is made that a result above this level may represent acute MI in patients without a classical history. Caution and a good understanding of the assay is imperative for accurate diagnosis and management of hospital patients with an apparently elevated level, but these results also raise interesting questions about what the levels actually indicate in patients in whom the suspicion f acute Mi is low. More data are required.

Conflict of Interest Educational support from BAYER

104 HERITABILITY AND FAMILY-BASED GWAS ANALYSES OF THE CIRCULATING CERAMIDE, ENDOCANNABINOID, AND N-ACYL ETHANOLAMIDE LIPIDOME

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Introduction Lipids of the endocannabinoid (eCB), N-acyl ethanolamine (NAE), and ceramide (CER) classes are potential novel biomarkers of coronary artery disease and type-2 diabetes. Major-gene effects have been discovered for certain lipid species, notably lipoprotein(a). We sought to establish the heritability of eCB, NAE, and CER species, and identify DNA variants influencing their concentrations in plasma.

Methods We undertook heritability (QTDT, GCTA) and GWAS analyses (FaST-LMM) of 11 eCBs and NAEs, and 37 CERs in 1,016 plasma samples from 196 British Caucasian families ascertained through a hypertensive proband, using targeted lipidomics by mass spectrometry, and Illumina 660W-Quad genotyping.

Results Anandamide (AEA), a potent eCB, was found heritable (h2AEA = 32–35%; P<5.80 × 10–11), however other less studied NAE species, presented higher estimates of heritability (h2NAEs = 41–79%; P<3.89 × 10–13). 24–46% of the variation in potential biomarker CER is due to genetic factors (P<1.00 × 10–7). GWAS identified associations with eQTLs of proteins involved in the metabolism of eCB and NAE (e.g. FAAH; PNAEDHEA <6.33 × 10–12,) as well as CER (SPTLC3; PCERN(24)S(18) <8.99 × 10–19) and novel loci implicated in cancer risk and non-alcoholic fatty liver disease (e.g. FBXO28; PCERN(24)S(19)ratio <1.95 × 10–8, SULT1C4; PCERN(24)S(19) <8.99 × 10–19).

Two-sample Mendelian randomisation suggests that a variant in FAAH (rs324420) influencing the level of plasma NAEs (e.g. PNAEDHEA<6.33 × 10–12) is causally associated with obesity, drug addiction, and anxiety. As an example, participants with the rs324420 AA genotype had a mean DHEA plasma concentration of 518 pg/ml, which decreased by 27% in those with the AC genotype, and a further 11% in those carrying the CC genotype.

Conclusion We demonstrate for the first time estimates of heritability for this extended array of bioactive lipids, identify GWAS-significant SNPs associating with their levels in circulation, and implicate the lipid species studied here in cardiovascular disease, cancer, and drug addition. The results shown here can be used for prioritisation of lipid mediators for large-scale Mendelian Randomisation studies, and in the identification of causal metabolic pathways, novel diagnostics and drug targets for disease intervention.

Conflict of Interest None

105 UNREPORTED CORONARY ARTERY CALCIFICATION ON UNGATED CT CHEST IMAGING: AN OPPORTUNITY MISSED?

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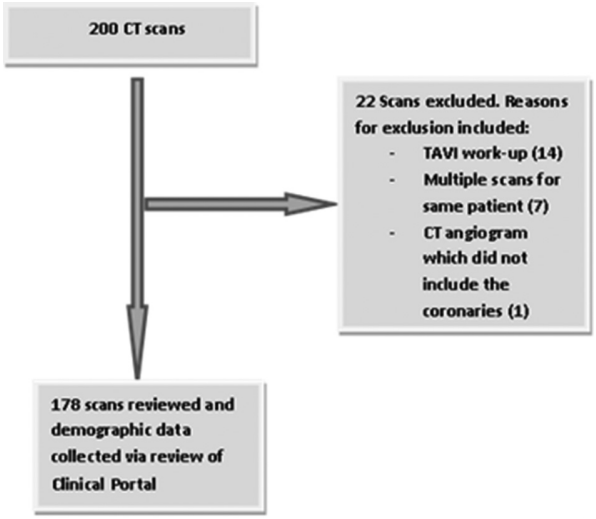
Introduction Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide1. Disability-adjusted life years (DALY) are high in part due to the years of life lost and early onset of disease. Multiple therapeutic interventions including lipid modifying drugs, aggressive blood pressure control, detection and treatment of diabetes and additional lifestyle measures reduce the risk of cardiovascular events in patients with established vascular disease2. Coronary artery calcification (CAC) is initiated and propagated by endothelial dysfunction and vascular inflammation. Thus, the



Abstract 105 Figure 1