66 months (weighted average 40 months). Mean age was 53.9 years with average ejection fraction of 41.3%. Overall incidence of appropriate therapy, reported in all studies, was 38.1% during the follow-up period. Left ventricular systolic dysfunction (LVSD) with ejection fraction < 40% was a predictor of appropriate therapy in the majority of studies, as were sustained VA during electrophysiological testing (EP) in one study. All-cause mortality was reported in six studies, with incidence of 6.0% over a median follow-up period of 42 months; only two mortality events were linked to a primary arrhythmic cause.

Conclusions Appropriate ICD therapies in patients with CS is commonly associated with LVSD, which may act as a surrogate for scar burden. The utility of EP testing in this setting remains unclear.

Conflict of Interest No

Heart failure

106 UNCOVERING MECHANISMS OF OBESITY-RELATED HEART FAILURE USING CARDIOVASCULAR MAGNETIC RESONANCE IMAGING IN THE UK BIOBANK

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Introduction Obesity is a rising public health crisis and a major risk factor for heart failure (HF). However, underlying mechanisms are incompletely understood. In this study, we investigate longitudinal associations of obesity with incident HF and cardiovascular imaging phenotypes in the UK Biobank (UKB). Importantly, we use cardiovascular magnetic resonance (CMR) to investigate potential mechanisms driving the obesity-HF relationships.

Methods The UKB cohort comprises over half a million individuals recruited from across the UK between 2006–2010. The UKB imaging study, which includes CMR, commenced in 2015 with plan to scan a random 20% subset of the original cohort. We defined obesity using body mass index (BMI) and waist-to-hip-ratio (WHR) measured at baseline recruitment. Incident HF events were identified through linked Hospital Episode Statistics data (censor date December 2021). CMR scans were analysed using an automated pipeline. We used Cox proportional hazard regression models adjusted for potential confounders to estimate the associations of BMI and WHR with incident HF in the whole sample. We used linear regression to characterise obesity (BMI, WHR) associations with CMR phenotypes. Finally, we used multiple mediation analysis to define the role of obesity-related cardiac remodelling in driving its associations with incident HF, independent of cardiometabolic diseases (diabetes, hypertension, high cholesterol).

Results In 491,606 UK Biobank participants (mean age 56.6 years, 54.3% women) over 12.2±0.9 years of prospective follow-up, higher BMI [HR 1.34 (1.32, 1.37)] and WHR [HR 1.30 (1.30, 1.36)] were associated with a greater hazard of HF. In the subset of participants with CMR (n=31,107), greater obesity was associated with adverse left ventricular (LV) structure (higher LV mass, greater concentricity), poorer LV global functional index, and lower myocardial native T1. In multiple mediation analysis, hypertension had an important role in mediating the associations of obesity with incident HF. Adverse LV remodelling (higher LV mass, greater concentricity) were also major mediators of the obesity-HF associations, independent of cardiometabolic disease. Notably, higher native T1 (indicated greater myocardial fibrosis) mediated a significant fraction of the relationship between obesity and incident HF. Given that in the whole cohort greater obesity was related to lower native T1, this observation may indicate different stages of progression in obesity-related cardiac remodelling.

Conclusion Our findings demonstrate the association of obesity with a greater risk of HF and adverse alterations of LV structure, function, and myocardial character. Importantly, we highlight the role of specific adverse cardiovascular remodelling patterns in driving obesity-HF associations. Thus, our findings highlight the growing public health importance of obesity as a driver of HF and propose novel channels for dedicated mechanistic research.

Conflict of Interest None

107 EMPAGLIFLOZIN IN HEART FAILURE WITH A PRESERVED EJECTION FRACTION ≥50%: RESULTS FROM THE EMPEROR-PRESERVED CLINICAL TRIAL

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Background In EMPEROR-Preserved, empagliflozin reduced the composite primary endpoint of cardiovascular (CV) death/hospitalisation for heart failure (HHF) in patients with heart failure (HF) with left ventricular ejection fraction (LVEF) >40%. We assessed the effect of empagliflozin in patients with a preserved LVEF ≥50% (considered 'true HFpEF' by many clinicians) and contrasted it with HF patients with mildly-reduced LVEF of 41–49% (i.e., <50%).

Methods Of 5,988 randomised patients, 1,983 had LVEF <50% and 4,005 had LVEF ≥50%. The outcomes included (1) the primary endpoint, (2) first and total HHF, (3) change in KCCQ-Clinical Summary Score (CSS), and (4) NYHA class at Week 52.

Results Patients with LVEF ≥50% (vs LVEF <50%) were more frequently women and were older; they had median NT-proBNP 946 pg/mL, and mean eGFR 59 mL/min; approximately half had atrial fibrillation or diabetes at baseline. In patients with LVEF ≥50%, empagliflozin reduced the risk of CV death/HHF by 17% (p=0.024), driven by a reduction in HHF (see table). Time-to-first-event of HHF was reduced by 22% (p=0.013) and total HHF by 17% (p=0.113). Empagliflozin produced meaningful improvements in KCCQ-CSS and NYHA class at Week 52. Compared with placebo, empagliflozin increased KCCQ-CSS in patients with LVEF ≥50% by 1.46 points (0.42–2.51; p=0.006); these empagliflozin-treated patients were 34% more likely to be in a lower NYHA class (p<0.001).

Abstract 107 Table 1

	Empagliflozin	Placebo	HR (95% CI)	*p*-value for interaction
CV death or HHF	**n/N (I/100PY)**	**n/N (I/100PY)**		
LVEF <50%	145/995 (7.2)	193/988 (10.0)	0.71 (0.57, 0.88)	0.27
LVEF ≥50%	270/2002 (6.7)	318/2003 (8.0)	0.83 (0.71, 0.98)	
HHF (first and recurrent)	**Total events/N**	**Total events/N**	**HR (95% CI)**	
LVEF <50%	122/995	209/988	0.57 (0.42, 0.79)	0.06
LVEF ≥50%	285/2002	332/2003	0.83 (0.66, 1.04)	
KCCQ CSS	**Mean adj. Δ at 52 weeks (SE)**	**Mean adj. Δ at 52 weeks (SE)**	**Mean diff vs placebo at week 52 (95% CI)**	
LVEF <50%	4.86 (0.54)	3.30 (0.55)	1.56 (0.05, 3.06) 1.46	0.92
LVEF ≥50%	4.24 (0.38)	2.78 (0.38)	(0.42, 2.51)	
NYHA class	**N analysed**	**N analysed**	**OR of lower NYHA class at week 52 (95%CI)**	
LVEF <50%	883	869	1.43 (1.14, 1.80)	0.64
LVEF ≥50%	1806	1814	1.34 (1.14, 1.58)	

Conclusion In EMPEROR-Preserved, empagliflozin significantly improved CV death/HHF in patients with LVEF ≥50% by 17%, driven by reductions in HHF. This is the first large-scale study documenting event reductions and quality-of-life benefits in patients with true HFpEF.

Conflict of Interest Received consultancy fees/lecture honoraria/research funding from the following: Boehringer Ingelheim, Eli Lilly, AstraZeneca, Novartis, BMS, Servier, Medtronic, and Novo Nordisc.

108 THE RELATION BETWEEN CHANGE IN LEFT VENTRICULAR SYSTOLIC FUNCTION AND SUBSEQUENT MORTALITY IN PATIENTS WITH CHRONIC HEART FAILURE

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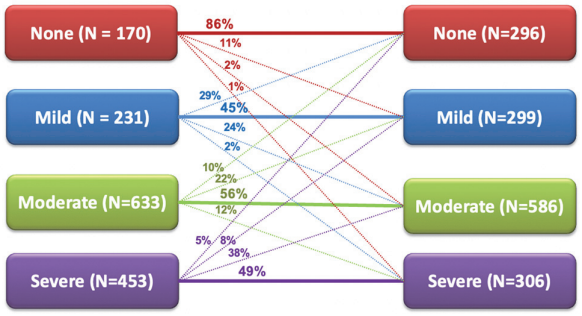
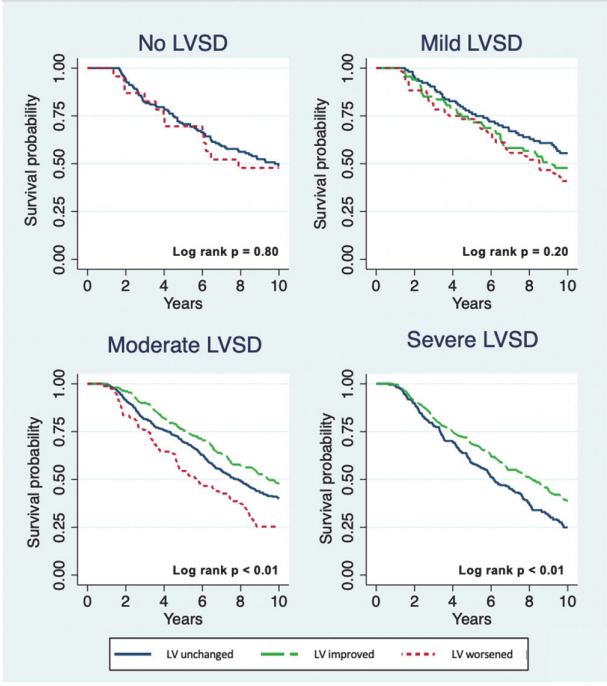
Introduction Increasing severity of impairment of left ventricular systolic dysfunction (LVSD) in patients with chronic heart failure (CHF) is associated with higher mortality. However, the relation between temporal changes in LVSD severity and long-term clinical outcome is unknown. MethodsPatients with CHF defined as the presence of compatible symptoms and either at least moderate LVSD or NTproBNP >125 ng/L were enrolled. LVSD was qualitatively assessed as: none, mild, moderate, and severe. Echocardiography was performed at baseline and 12 months. The primary endpoint was all-cause mortality. Cox proportional hazard models were used to assess the relation between changes in LVSD and outcome. Hazard ratios (HR) are reported with 95% confidence intervals (CI).Results At baseline, 170 (11%) had no, 231 (16%) mild, 633 (43%) moderate and 453 (30%) severe LVSD. Amongst patients with either moderate or severe LVSD at baseline, 40% had improvement in function at 12 months (figure 1). Amongst patients with no LVSD at baseline, only 14% had deterioration of function. During subsequent median follow up of 2773 days, 868 patients died. Worsening of LVSD was associated with increasing all-cause mortality in patients with moderate LVSD and severe LVSD at baseline (figure 2), but this was not significant after adjustment for covariables (table 1). Improvement of LVSD was independently associated with better survival in patients with moderate LVSD at baseline (HR 0.72 (95% CI: 0.53–0.98, p = 0.04).

Conclusion Greater severity of LVSD at baseline is associated with increasing likelihood of improvement. Amongst patients with moderate LVSD, improvement in LVSD is independently associated with survival.



Abstract 108 Figure 1 Stability of left ventricular systolic dysfunction (LVSD). Change of the severity of LVSD assessed by echocardiography at baseline and one year. Continuous data presented as N and percentages (%)



Abstract 108 Figure 2 Kaplan–Meier curves for all-cause mortality in patients with heart failure by change in the severity of left ventricular systolic dysfunction (LVSD) stratified by baseline LVSD. Differences between groups were evaluated using the log-rank test