

Abstract 154 Figure 2 A concise one-page SGLT2-inhibitor patient information leaflet made available online and printable for patients in Coventry and Warwickshire region

Conclusion This quality improvement project highlighted substantial gaps in patient understanding and awareness of SGLT2i therapy. To sustain improvements in patient awareness and adherence, a concise SGLT2i information leaflet was developed through collaborative efforts with pharmacists. **Conflict of Interest** None

155 IMPACT OF SGLT2 INHIBITION ON REVERSE CARDIAC REMODELING IN PATIENTS WITH HEART FAILURE: A SYSTEMATIC REVIEW AND META-ANALYSIS

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10.1136/heartjnl-2024-BCS.152

Introduction Several landmark randomised-controlled trials (RCT)'s have demonstrated the efficacy of sodium-glucose cotransport 2 (SGLT2) inhibitors in reducing all-cause mortality, cardiovascular (CV) mortality and rates of heart failure (HF) hospitalisations (1). Much interest surrounds their mechanism of action and whether they have direct effects on reverse cardiac remodeling (2). Therefore, we conducted a meta-analysis of placebo controlled RCTs evaluating the impact of SGLT2 inhibition on cardiac remodeling in patients with HF.

Methods We performed a systematic review and meta-analysis in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) Statement and Cochrane Collaboration (3). Data interrogation of each major database including PubMed, EMBASE, MEDLINE and Cochrane Library was performed. Randomised-controlled trials evaluating patients >18 years with HF reduced ejection fraction and HF preserved ejection fraction treated with a SGLT2 inhibitor versus placebo-control were included (4-10). Outcome measures included left ventricular end diastolic volume and volume index [LVEDV (mls)/LVEDVi (mls/m2)], Left ventricular end systolic volume and volume index [LVSDV (mls) / LVSDVi (mls/m2)], Left ventricular ejection fraction (LVEF) (%), Left ventricular mass index [LVMi] (g/m2) and left ventricular global longitudinal strain (LV GLS) (%). The mean difference (MD) and standard error were extracted from each study and a random effects model utilised for analysis. Risk of bias of the included studies was assessed using a detailed framework provided by the Cochrane Handbook for Systematic Reviews of Interventions with a planned sensitivity

analysis restricting analysis to studies assessed to have low risk. Additionally, given the differing imaging modalities used, robustness of effect was effect was further assessed using the standardized mean difference (SMD). A pre-specified subgroup analysis was performed to stratify results according to imaging modality used (cardiac MRI and echocardiography) with a further analysis delineating effect by LVEF.

Results The 7 final included studies were randomised, placebocontrolled trials in patients with heart failure comprising a total population of 620 patients (75% male). Baseline LVEF ranged from 29+/-8% to 55.2+/-4.2% and follow-up ranged from 12 weeks to 1 year with studies employing cardiac MRI (n=4) and echocardiography (n=3). Pooled data demonstrated that SGLT2 inhibition, compared to placebo control, resulted in significant improvements in LVEDV (mean difference -10.92 mls [95% CI: -16.73 to -5.11; z =3.68, p= 0.002]), LVEDVi (mean difference -5.78 mls [95% CI -10.30 to -1.26; z=2.51; p=0.01]), LVESV (mean difference -12.47 mls [95% CI -19.12 to -5.82; Z=3.68; P=0.0002]) (figure 1), LVESVi (mean difference -6.02 mls [95% CI -10.34 to -1.70; z=2.73: p=0.006]), LVM (mean difference -9.77 g [95% CI: -17.65 to -1.89: z=2.43: p=0.02]) and LVEF (mean difference +2.45 mls [95% CI 1.12 to 3.78: z=3.62: p=0.0003]) (figure 2). Only three studies assessed GLS (n=327) with no significant treatment effect noted (mean difference +0.42% [95%CI -0.19 TO 1.02; P=0.18]). Significant differences between baseline LVEF <40% and >40% were evident with loss of effect noted in patients with LVEF >40% with respect to reverse remodeling of LVESV (Chi2=4.05, df=1, p=0.04, I2=75.3%) and LVMi (Chi2=4.44, df=1, p=0.04, I2=77.5%).

Conclusion This meta-analysis of seven placebo-controlled, randomised trials, provides an additional data and insight into the effects of SGLT2 inhibition on reverse cardiac remodeling in patients with heart failure. Compared to placebo control, we found that treatment with a SGLT2 inhibitor produced significant improvements in several markers of reverse cardiac

Figure 1a: Left ventricular end-systolic volume index [LVESVi (mls/m²)]





			SGLT2i	Placebo		Mean Difference	Mean Difference
Study or Subgroup	Mean Difference	SE	Total	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI
9.2.1 Cardiac MRI							
EMPATROPISM	-11.34	2.9713	42	42	22.4%	-11.34 [-17.16, -5.52]	
EMPIRE-HF	-5.5	2.5637	89	87	24.7%	-5.50 [-10.52, -0.48]	
REFORM	-8.6	2.7635	28	28	23.5%	-8.60 [-14.02, -3.18]	
SUGAR-DM	9.1	6.636	42	50	9.0%	9.10 [-3.91, 22.11]	
Subtotal (95% CI)			201	207	79.6%	-6.22 [-11.73, -0.70]	-
Heteropeneity: Tau*:	: 19.48; Chi ² = 8.67,	df = 3 (P	= 0.03; (°=65%			
Test for overall effect	Z = 2.21 (P = 0.03)						
9.2.2 Echocardiogra	phy						
DAPA-VO2	-3.36	3.3378	45	45	20.4%	-3.36 [-9.90, 3.18]	
Subtotal (95% CI)			45	45	20.4%	-3.36 [-9.90, 3.18]	
Heterogeneity: Not ap	plicable						
Test for overall effect	Z = 1.01 (P = 0.31)						
Total (95% CI)			246	252	100.0%	-5.78 [-10.30, -1.26]	•
Heteropeneity: Tau ^a	: 14.94; Cihi ^a = 9.79,	-20 -10 0 10 20					
Test for overall effect	Z = 2.51 (P = 0.01)	Eavours SCI T2i Eavours control					
Test for subgroup dif	ferences: ChiP = 0.4	rervera overen Parouia coneor					

Abstract 155 Figure 1 Five studies assessed LVEDVi (mls) (n=498) and LVESVi (mls) (n=498) with pooled data demonstrating that SGLT2 inhibition, compared to controls, significantly decreased *a*). LVESVi (mean difference -6.02 mls [95% CI -10.34 to -1.70; Z=2.73; P=0.0006]) and *b*). LVEDVi (mean difference -5.78 mls [95% CI: -10.30 to -1.26; z =2.52, p= 0.01)]. There were no significant differences between imaging modality used for LVEDVi (CMR versus echocardiography; Chi²=0.22, df=1, p=0.64, I²=0%) or LVESVi (CMR versus echocardiography; Chi²=0.43, df=1, p=0.51, I²=0%)

Figure 2a: Left ventricular mass [LVM (g)]

			Experimental	Control		Mean Difference	Mean Difference	
Study or Subgroup	Mean Difference	SE	Total	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI	
6.3.1 Cardiac MRI								
EMPATROPISM	-21.9	5.3389	42	42	19.4%	-21.90 [-32.36, -11.44]		
EMPA-VISION (HFreF)	-9.65	3.83	17	18	23.3%	-9.65 [-17.16, -2.14]		
SUGAR-DM	-2.6	2.8672	42	50	25.7%	-2.60 [-8.22, 3.02]		
EMPA-VISION (HFpEF)	-0.41	5.3317	13	11	19.4%	-0.41 [-10.86, 10.04]		
Subtotal (95% CI)			114	121	87.8%	-8.24 [-16.48, 0.01]	-	
Heterogeneity: Tau# = 51.95; Chi# = 12.13, df = 3 (P = 0.007); I# = 75%								
Test for overall effect: Z =	1.96 (P = 0.05)							
6.3.2 Echocardiography								
EMPIRE-HF	-20.7	8.8267	89	86	12.2%	-20.70 [-38.00, -3.40]		
Subtotal (95% CI)			89	86	12.2%	-20.70 [-38.00, -3.40]		
Heterogeneity: Not applic	able							
Test for overall effect Z =	2.35 (P = 0.02)							
Total (95% CI)			203	207	100.0%	-9.77 [-17.65, -1.89]	-	
Heterogeneity: Tau* = 54.	.75; Chi ^a = 14.51, df	= 4 (P =	0.006); I* = 72%				50 .25 0 25 50	
Test for overall effect: Z =						Favours SGLT2i Favours control		
Test for subgroup differen	nces: Chi# = 1.62, d	f=1 (P=	0.20), #= 38.44	%				

Figure 2b: Left ventricular ejection fraction [LVEF (%)]

			Experimental	Control		Std. Mean Difference	Std. Mean Difference
Study or Subgroup	Std. Mean Difference	SE	Total	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI
4.3.1 Cardiac MRI							
EMPA-VISION (HFpEF)	1.47	1.5664	13	11	9.9%	1.47 [-1.60, 4.54]	
EMPA-VISION (HFreF)	1.87	1.8215	17	18	8.3%	1.87 [-1.70, 5.44]	
EMPATROPISM	6.1	0.7726	42	42	16.4%	6.10 [4.59, 7.61]	
REFORM	1.2	2.2124	28	28	6.5%	1.20 [-3.14, 5.54]	
SUGAR-DM	0.6	1.0307	42	50	14.1%	0.60 [-1.42, 2.62]	
Subtotal (95% CI)			142	149	55.1%	2.41 [-0.37, 5.19]	
Heterogeneity: Tau* = 7.8	3; Chi# = 22.80, df = 4 (F	= 0.000	1); I*= 82%				
Test for overall effect: Z =	1.70 (P = 0.09)						
4.3.2 Echocardiography							
DAPA-V02	1.66	1.3112	45	45	11.7%	1.66 [-0.91, 4.23]	
EMPIRE-HF	1.4	1.1971	89	86	12.6%	1.40 [-0.95, 3.75]	
Qianyu Fu et al	3	0.1859	30	30	20.5%	3.00 [2.64, 3.36]	
Subtotal (95% CI)			164	161	44.9%	2.62 [1.66, 3.58]	•
Heterogeneity: Tau* = 0.2	29; Chi# = 2.71, df = 2 (P	= 0.26);1	*= 26%				
Test for overall effect: Z =	5.36 (P < 0.00001)						
Total (95% CI)			306	310	100.0%	2.45 [1.12, 3.78]	
Heterogeneity: Tau# = 2.20; Chi# = 26.35, df = 7 (P = 0.0004); I# = 73%							
Test for overall effect Z =	3.62 (P = 0.0003)						Fauture control Fauture SCI T2i
Test for subgroup differe	nces: Chi# = 0.02, df = 1	(P = 0.8)	0, I [#] = 0%				return control returns out is

Abstract 155 Figure 2 *a).* Four studies assessed LVMi (n=410) with SGLT2 inhibition producing significant reductions versus placebo control (mean difference -9.77 mls [95% CI: -17.65 to -1.89: z=2.43: p=0.02]). *b).* LVEF was measured in seven studies (n=616) with a significant improvement noted with SGLT2 inhibition compared to control (mean difference +2.45 mls [95% CI 1.12 to 3.78: z=3.62: p=0.0003]). There was no significant difference between imaging modality used for LVMi (CMR versus echocardiography; Chi²=1.62, df=1, p=0.20, l²=38.4%) or LVEF (Chi²=0.02, df=1, p=0.89, l²=0%). ***EMPA-VISION** provided two cohorts within one study therefore are presented separately

remodeling, specifically, LVESV, LVESVi, LVEDV, LVEDVi, LVM, LVMi and LVEF. These effects appeared more pronounced in patients with a baseline LVEF <40%. Conflict of Interest None

156 ARE CURRENT CUT-OFFS TO IDENTIFY STAGE B HEART FAILURE (SBHF) APPROPRIATE IN THE UK?

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10.1136/heartjnl-2024-BCS.153

Introduction People with Type 2 Diabetes (T2D) are at increased risk of heart failure, and as such are classified as having Stage A Heart Failure (SAHF). Stage B Heart Failure (SBHF) encompasses patients without symptoms of heart failure that have evidence of raised filling pressures, biochemical or structural cardiac changes. Identifying patients with SBHF is believed to confer a higher risk of disease progression. The aim of this work was to assess the number of asymptomatic healthy volunteers and people with T2D characterised as having SBHF, according to American Heart Association/American College Cardiology/Heart Failure Society of America suggested criteria.

Methods A single-centre, prospectively recruited cohort of middle-aged asymptomatic adults with T2D and healthy volunteers, with no history or signs of cardiovascular disease (NCT03132129), underwent comprehensive cardiac phenotyping with transthoracic echocardiography and circulating brain