CONCLUSION: In our study, patients treated with Tafamidis had a lower deterioration in ventricular function at 12 months follow-up compared with an untreated group. Although all measures of cardiac function demonstrated a difference in our cohort, indexing GLS to $\sqrt{(RR)}$ may allow assessment in myocardial function in more patients especially in the context of variable RR interval when LVEF measurements can be time consuming and less reliable.

Variable	Without Tafamidis (n=24)	With Tafamidis (n=24)	p-value
Age:	65.67±11.28	72.71±9.64	0.04
Sex:			0.09
Male	15 (62.5%)	21 (87.5%)	
Female	9 (37.5%)	3 (12.5%)	
Comorbidities:			0.96
Smoking	6 (25.0%)	6 (25.0%)	
Alcohol	9 (37.5%)	10 (41.7%)	
Dyslipicemia	10 (41.7%)	14 (58.3%)	
Diabetes	5 (20.8%)	5 (20.8%)	
Hypertension	11 (45.8%)	15 (62.5%)	
Dialysis	1 (4.2%)	0 (0.0%)	
Implanted Device:			0.08
Pacemaker	3 (12.5%)	2 (8.3%)	
ICD	5 (20.8%)	0 (0.0%)	
CRT-D	1 (4.2%)	1 (4.2%)	
None	15 (62.5%)	21 (87.5%)	
Treatmerts:			0.54
Diuretics	10 (41.7%)	15 (62.5%)	
Beta Blocker	9 (37.5%)	11 (45.8%)	
ACE Inhibitor	5 (20.8%)	8 (33.3%)	
ARB	3 (12.5%)	7 (29.2%)	
Digoxin	1 (4.2%)	0 (0.0%)	
MCRA	7 (29.2%)	8 (33.3%)	
Diflunisal	2 (8.3%)	4 (16.7%)	
Other medications	13 (54.2%)	7 (29.2%)	
NYHA Class:			0.07
Asymptomatic	4 (16.7%)	1 (4.2%)	
Class 1	3 (12.5%)	6 (25.0%)	
Class 2	7 (29.2%)	14 (58.3%)	
Class 3	7 (29.2%)	3 (12.5%)	
Class 4	2 (8.3%)	0 (0.0%)	
unknown	1 (4.2%)	0 (0.0%)	
BNP:			
Presenting BNP (pg/mL)	239.31±295.95	195.25±281.20	0.59
BNP Peak (pg/mL)	430.76±593.39	283.00±563.85	0.67
hATTR variants:			0.00011
No variant/wildtype	5 (20.8%)	19 (79.2%)	
Val142Ie	9 (37.5%)	4 (16.7%)	
Thr80Ala	4 (16.7%)	0 (0.0%)	
Val50Met	4 (16.7%)	0 (0.0%)	
Ala120Ser	1 (4.2%)	0 (0.0%)	
Val50Leu	0 (0.0%)	1 (4.2%)	
unknown	1 (4.2%)	0 (0.0%)	

TABLE 1: Descriptive characteristics of 24 patients treated without Tafamidis and 24 patients treated with Tafamidis. P-values were determined by using the Wilcoxon rank-sum test and Fisher's exact test.

P035
EVALUATION OF THE REAL-WORLD PLACE OF THE ICOSAPENT ETHYL (IPE) FOR THE TREATMENT OF PATIENTS WITH HYPERTRIGLYCERIDEMIA REQUIRING INVASIVE CORONARY ANGIOGRAM

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BACKGROUND: Although the REDUCE-IT trial recently demonstrated the benefits of Icosapent ethyl (IPE) on reducing ischemic events in a broad population of primary and secondary prevention patients with hypertriglyceridemia, its generalizability to patients referred for a coronary angiogram is not known yet. This study aims to determine the proportion of patients referred for a coronary angiogram that would be eligible for IPE treatment.

METHODS AND RESULTS: In this prospective study, all patients referred to the catheterization laboratory in a tertiary academic center between January 1st, 2022 and February 15th, 2022 were enrolled. Patients meeting the inclusion criteria of the REDUCE-IT trial were included. A total of 350 patients were screened and included in our study. The average age was 68±11 and 64% were males. Hypertension and diabetes were present in 76% and 40% of patients, respectively. A total of 253 patients (72.3%) had dyslipidemia. The mean left ventricular ejection fraction (LVEF) was 52±14%. A lipid profile was available in 73% of cases in the 6 months preceding the procedure. A total of 38 patients (16.2%) referred to the catheterization laboratory currently on statin therapy would be eligible for IPE, according to REDUCE-IT criteria. No difference was found between patients with acute coronary syndrome and chronic coronary syndrome in terms of elibility for IPE treatment (13.5% vs 21.8%, p=0.132). No eligible patient was yet started on IPE.

CONCLUSION: These analyses would support IPE usage as an adjunct to secondary prevention therapies in eligible patients referred for a coronary angiogram. Findings of this study need to be confirmed in larger scale cohorts.

P036
HOMOZYGOUS FAMILIAL HYPERCHOLESTEROLEMIA IN CANADA

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BACKGROUND: Homozygous familial hypercholesterolemia (HoFH) is life-threatening orphan disease characterized by high levels of low-density lipoprotein cholesterol (LDL-C). Untreated patients often present with extensive xanthomas and marked premature atherosclerotic cardiovascular disease (ASCVD) before the age of 20. Prior to the advent of statins and extracorporeal LDL filtration techniques, survival beyond 30 years of age was unusual. Treatment with lipid-lowering therapy is highly efficacious and has dramatically increased life expectancy, reducing the risk of ASCVD to background population rates. Canada is known to have several founder effect regions for HoFH, including Québec. Clinical outcomes in HoFH patients, especially ASCVD events such as myocardial infarctions or stroke are difficult to capture, in part because of the rarity of the disorder and the lack of registry focusing on this disease.

METHODS AND RESULTS: The objective of our project is to obtain a comprehensive registry of HoFH in Canada, estimate the cost to society caused by HoFH burden of disease in Canada, and implement changes to advocate access to specialized care for these patients. A standardized

questionnaire was sent to the 19 academic sites across Canada participating in the FH Canada network. We previously identified 79 cases across the country, and have captured 46 of these cases. Here we describe their medical history, lipid levels, treatments and clinical outcomes. At the time of entry in the Registry, the mean age was 44 +/- 19 years, with a majority of females (54.4%), representing cases across 5 provinces. The average age of diagnosis was 16 4 years. with 67.4% having untreated LDL > 10 mmol/L. Presence of physical markers, such as xanthomas or corneal arcus, were also found in 80.4% and 26.1% of patients, respectively. For lipid-lowering therapy, 52.2% were undergoing LDL-apheresis, 91% were on statins and 41% on PCSK9 inhibitors. 54.3% displayed ASCVD, with 43.5% having aortic stenosis, 15.2% having experienced a myocardial infarction, and 36.9% having undergone one or more coronary artery bypass graft procedures.

CONCLUSION: We plan to use this data at provincial and national levels, helped by the Canadian Organization for Rare Diseases (CORD) and the Réseau Québecois des maladies orphelines (RQMO), to provide HoFH patients access to care, including orphan drugs such as evinacumab, and treatment techniques such as LDL apheresis. This work will provide important new health-related knowledge about the determinants of ASCVD risk and phenotypic manifestations of HoFH in Canada, and examine the quality of life and burden to the healthcare system.

P037
INSULIN-LIKE GROWTH FACTOR BINDING PROTEIN-7 AS A MARKER OF CARDIAC REVERSE REMODELING WITH EMPAGLIFLOZIN: A SECONDARY ANALYSIS OF THE EMPA-HEART CARDIOLINK-6 RANDOMIZED CONTROLLED TRIAL

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BACKGROUND: Reverse cardiac remodeling is believed to contribute to the cardiovascular benefits offered by sodium-glucose transport protein 2 inhibitors (SGLT2i). Given the association between insulin-like growth factor binding protein 7 (IGFBP7) levels and cardiac remodeling, it has been postulated that IGFBP7 may serve as a predictor for individuals who may derive greater benefit from SGLT2i therapy. This exploratory sub-analysis of the EMPA-HEART CardioLink-6 trial examined the association between serum IGFBP7 levels and reverse cardiac remodeling in patients treated with empagliflozin.

METHODS AND RESULTS: The EMPA-HEART CardioLink-6 trial randomized 97 patients with type 2 diabetes and coronary artery disease to either empagliflozin (10 mg/d) or matching placebo for 6 months. The primary outcome was change in left ventricular mass indexed to body surface area (LVMi) from baseline to 6 months as measured by cardiac magnetic resonance imaging. Serum samples collected from patients at baseline were processed for detection of IGFBP7. A linear model adjusted for baseline characteristics was used to evaluate the relationship between baseline IGFBP7, 6-month change in LVMi, and treatment arm. An adjusted multivariable linear regression model was used to evaluate the association between baseline IGFBP7 and baseline LVMi. Patients with missing covariate data were excluded which resulted in a final analysis set of 74 patients. We observed no association between baseline IGFBP7 levels and change in LVMi, nor was there any significant difference in treatment effect between empagliflozin versus placebo patients (empagliflozin 6-month change in LVMi: 0.25g/m2 (95% CI: -0.17 g/m2 to 0.67 g/m2) per 1 ng/mL higher IGFBP7 vs. placebo 6-month change in LVMi: 0.07 g/m2 (95% CI: -0.21 g/m2 to 0.35 g/m2) per 1 ng/mL higher IGFBP7; Pinteraction = 0.49). We also did not detect any association between baseline IGFBP7 and baseline LVMi (P = 0.52). Additional sensitivity analysis assessing the association between IGFBP7 as a categorical variable and 6-month change in LVMi did not reveal any significant interaction (Pinteraction=0.86).

CONCLUSION: Our results suggest that empagliflozin-mediated cardiac reverse remodeling in patients with type 2 diabetes and coronary artery disease is independent of serum IGFBP7 levels. However, given the sample size and short study duration of the EMPA-HEART CardioLink-6 trial, further investigations evaluating whether IGFBP7 levels influence the clinical efficacy of SGLT2i are warranted.

P038
LEFT VENTRICULAR MASS PREDICTS CARDIAC REVERSE REMODELING IN PATIENTS TREATED WITH EMPAGLIFLOZIN: AN EXPLORATORY SUB-ANALYSIS OF THE EMPA-HEART CARDIOLINK-6 RANDOMIZED CONTROLLED TRIAL

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BACKGROUND: Left ventricular (LV) hypertrophy is associated with an elevated risk for cardiovascular (CV) disease events and all-cause mortality. The CV benefits of sodium-glucose transport protein 2 inhibitors (SGLT2i) have been attributed, in part, to cardiac reverse remodeling. The EMPA-HEART CardioLink-6 study reported that SGLT2 inhibition with empagliflozin was associated with a significant reduction in LV mass indexed to body surface area (LVMi) after 6 months. In this exploratory sub-analysis of the same trial, we evaluated how baseline LVMi may influence cardiac reverse remodeling with empagliflozin.

METHODS AND RESULTS: A total of 97 patients with type 2 diabetes and coronary artery disease were randomized to empagliflozin (10 mg/d) or matching placebo for 6 months. To determine if the impact of empagliflozin on the 6-month change in LVMi was dependent on baseline LVMi, the EMPA-HEART CardioLink-6 cohort was stratified into two groups − baseline LVMi ≥60 g/m2 and < 60 g/m2. Between-group comparisons were conducted using a linear regression model adjusted for