Sodium-glucose co-transporter-2 inhibitors improve cardiovascular outcomes in heart failure with reduced ejection fraction regardless of ischemic etiology

D. Patoulias[1], A. Boulmpou[2], C. Tsavousoglou[2], M. Toumpourleka[2], F. Siskos[1], A. Nikolaidis[1], C.E. Papadopoulos[2], V. Vassilikos[2], M. Doumas[1]

[1]Hippokration General Hospital of Thessaloniki, Second Propedeutic Department of Internal Medicine, Thessaloniki, Greece; [2]Hippokration General Hospital of Thessaloniki, Third Department of Cardiology, Thessaloniki, Greece

Funding Acknowledgement: Type of funding sources: None.

Background: Coronary artery disease remains the main underlying cause of heart failure (HF), despite the progress in prevention, diagnosis and treatment. Sodium-glucose co-transporter-2 inhibitors have been shown to improve surrogate cardiovascular outcomes in patients with HF with reduced ejection fraction (HFrEF), regardless of diabetes status.

Purpose: We sought to determine the effect of SGLT-2 inhibitors on the primary composite endpoint (cardiovascular death or hospitalization for HF) across the two hallmark trials in the HFrEF population (EMPEROR Reduced and DAPA-HF), according to ischemic or non-ischemic etiology of HF.

Methods: We pooled data from EMPEROR reduced and DAPA-HF trials in a total of 8,474 patients with HFrEF, performing a sub-analysis according to the presence of ischemic cardiomyopathy as the underlying cause of HFrEF.

Results: Treatment with SGLT-2 inhibitors resulted in a significant decrease in the risk for the primary composite outcome in patients with HFrEF of ischemic etiology, equal to 18% (RR=0.82, 95% CI: 0.73–0.92, I^2=0%). In patients with HFrEF of non-ischemic etiology, SGLT-2 inhibitors produced a significant decrease in the risk for the primary composite outcome equal to 18% (RR=0.72, 95% CI: 0.63–0.82, I^2=0%). Despite the greater effect in patients with non-ischemic HFrEF, no subgroup difference was detected (p=0.16). Generated results are summarized in Figure 1.

Conclusions: SGLT-2 inhibitors improve surrogate cardiovascular outcomes both in patients with ischemic and non-ischemic HFrEF.

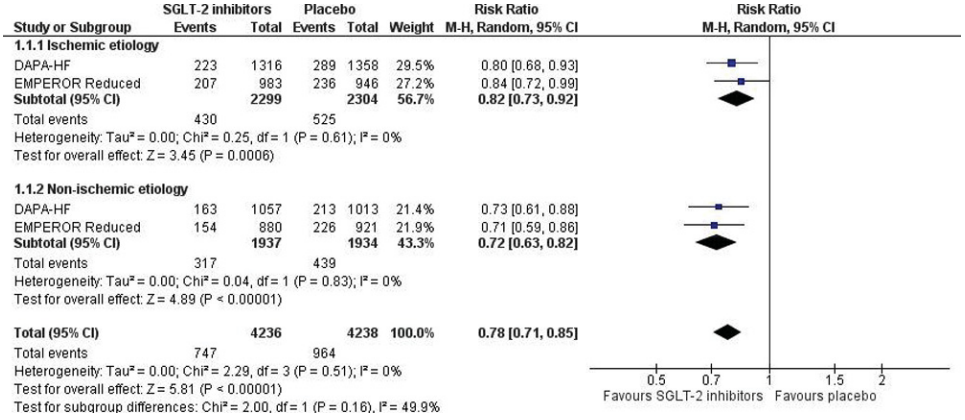

Figure 1