Abstract Submission Form

The Women's Heart Center Program Committee is accepting abstract submission forms through **August 15, 2025**. Completed forms should be emailed to WHC@TheChristHospital.com.

Abstract submissions should be gender- and sex-specific research pertaining to one of the program topics outlined below.

The Program Committee wishes to encourage young scientific investigators and will reward up to 4 abstracts/posters submitted by presenters considered early career (definition provided below). First place will receive $1000, second place will receive $500, and two honorable mentions will each receive $250.

The presenting author will be sent an email with the status of the submission by **August 22, 2025**. If your abstract is accepted, your notification will contain complete presentation information. However, please note the following:

- All human subject research must conform to the principles of the Declaration of Helsinki of the World Medical Association.
- The presenting author should be able to provide documentation of IRB approval if requested.
- The Program Committee is unable to reimburse presenters for travel, hotel, or per diem expenses.
- Submission of an abstract constitutes a commitment by the presenting author (or designee) to present in-person at the symposium on October 3, 2025, during the following times:
    - Registration & Networking: 7:00 – 8:00 am
    - Networking Lunch: 12:00 – 1:30 pm
    - Poster Session Award Announcement: 4:50 – 5:10 pm
- All accepted abstract presenters must register for the symposium via Eventbrite and pay the applicable registration fees (trainees and invited speakers will have the registration fee waived).
- If an author wishes to withdraw an abstract, please email WHC@TheChristHospital.com.

Presenting Author Information

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Early Career (Defined as physicians, scientists, medical students, and other healthcare providers currently in residency or fellowship programs or within three years of training)? Yes ☒ No ☐

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Disclosures: Please list any relevant financial disclosures.
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Abstract Topic (must be gender- or sex-specific)

- ☐ Preventative cardiology
- ☐ General cardiology
- ☐ Interventional cardiology
- ☐ Heart failure
- ☐ Cardio-oncology
- ☐ Cardio-obstetrics
- ☐ Electrophysiology
- ☐ Cardiovascular Imaging
- ☒ Coronary Microvasculature
- ☐ Social Determinants of Health
- ☐ Mental Health
- ☐ Precision Medicine

Title: Empagliflozin Significantly Reduces Circulating Markers of Endothelial Dysfunction in Ischemia with Non-Obstructive Coronary Arteries (INOCA) and Coronary Microvascular Dysfunction: EMbArk Phase II Pilot Trial

Background: Up to 70% of patients with angina undergoing invasive coronary angiography have Ischemia with Non-Obstructive Coronary Arteries (INOCA). Patients with endothelial dysfunction [EnD] and coronary microvascular dysfunction [CMD] have impaired myocardial blood flow and ischemia despite the absence of obstructive CAD, which is partially attributed to inflammation. Sodium Glucose Co-Transporter 2 inhibitors (SGLT2i's) have remarkable cardiovascular benefits and reduce EnD. However, these effects have not been examined in an INOCA population with CMD.

Methods: Phase II open label pilot trial was conducted in INOCA patients (n = 7) with CMD treated with the SGLT2i Empagliflozin (EMPA, 10mg/daily) for 90 days. CMD was diagnosed with the invasive thermodilution method using adenosine and acetylcholine, excluding patients with diabetes or heart failure. We investigated change in circulating levels of inflammatory cytokines, ROS markers (plasma 8-isoprostane, myeloperoxidase – MPO and total MPO activity), antioxidants (total antioxidant capacity) and circulating levels of $CD34^{+}$ repair endothelial microvesicles (eEVs) and inflammatory $VCAM^{+}$ eEVs at baseline and 45-days following treatment. We also compared baseline samples to INOCA with no-CMD controls (n = 4) from stored samples.

Results: In an all-female cohort, mean age was 60 ± 12 years (Table 1). Through the 45-day visit, there were two reported UTI's, one vaginal yeast infection and one non-clinically significant decrease in GFR. INOCA patients with CMD had significantly upregulated levels of 9 proinflammatory cytokines (CRP, TNFα, IL1β, IL-6, ICAM-1, VCAM-1, E-Selectin, Thrombomodulin, VEGF) compared to no-CMD controls; and these levels were reduced following EMPA

treatment (Fig 1A – 1D; 1F – 1J); and anti-inflammatory IL-10 was downregulated compared to non-CMD controls and increased with treatment (Fig. 1E). INOCA patients with CMD had elevated ROS markers and reduced antioxidant capacity compared to no-CMD controls, and these improved with treatment (Fig 1K – 1N). INOCA patients with CMD had significantly elevated total concentration of inflammatory eEVs ($VCAM^+$) and reduced repair eEVs ($CD34^+$) at baseline that improved with EMPA treatment (Fig. 1O – 1P).

Conclusions: In a cohort of INOCA patients, as compared to patients with no-CMD, patients with CMD had elevated circulating markers associated with EnD. This pilot study indicates that SGLT2i treatment is well-tolerated and has the potential to reduce inflammation, ROS, and eEVs in INOCA patients with CMD.