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Empagliflozin Attenuates Inflammation-induced Endothelial Glycocalyx Disruption within the Isolated Murine Aorta

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Empagliflozin, a sodium glucose co-transporter 2 inhibitor (SGLT2-I), was initially developed as an anti-diabetic drug but also exhibits cardioprotective properties. This study primarily investigates its role in attenuating endothelial glycocalyx (eGC) disruption, one of the key consequences of vascular inflammation. Additionally, we claim that empagliflozin enhances the endothelial-dependent vasodilation of aorta during inflammation through eGC protection. Empagliflozin (1 μM) preserved eGC integrity in TNF (10 ng/ml)-stimulated aorta, as demonstrated by fluorescence microscopy and further confirmed using atomic force microscopy. Notably, we also showed that empagliflozin mitigates TNF-induced overexpression of intracellular adhesion molecule 1 indicating its anti-inflammatory properties. Furthermore, our findings indicate that inhibition of sodium-hydrogen exchanger 1 with cariporide (10 μM) and endothelial sodium channel with amiloride (0.1 μM) also mitigated eGC disruption. This suggests that empagliflozin's protective effect on endothelial dysfunction is sodium-dependent yet SGLT2-independent. These findings identify empagliflozin as a promising eGC protectant, however, the precise mechanisms underlying its action require further investigation.

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