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Empagliflozin attenuates renal fibrosis through the DsbA-L- CAS-STING pathway

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Objectives: Chronic kidney disease (CKD) is associated with an increase in morbidity and mortality. Renal fibrosis is a common pathway leading to the progression of CKD. Recent studies have reported an improvement of CKD with use of sodium glucose cotransport 2 inhibitors (SGLT2i) in both patients with DM and those without it. However, the mechanism of SGLT2i's effect upon CKD has not been elucidated. In current study, we examined the effect of SGLT2i on renal fibrosis in rats caused by unilateral ureteral obstruction (UUO).

Methods: Rats were randomly divided into two groups (each n=8). One group was treated by Empagliflozin after UUO induction while the other group was left untreated. Kidneys were harvested two weeks after UUO. We evaluated the mitochondrial damage pathway presumed to contribute to the inflammation.

Results: UUO has resulted in marked renal fibrosis and triggered the activation of the cGAS-STING pathway. Empagliflozin has been shown to attenuate renal fibrosis and decrease the activation of the cCAS-STING pathway. The expression of disulfide-bond A oxidoreductase-like proteins (DsbA-L) was increased in the SGLT2i treated group (Figure 1).

Conclusions: These findings suggest that SGLT2i attenuates the development of renal fibrosis via inhibition of mitochondrial damage.

figure1.The expression of disulfide-bond A oxidoreductase-like prtoeins was increased in ther SGLT2i treated group.