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# O-31 - THE ISGLT2 EMPAGLIFOZIN PROMOTES ANTIOXIDANT RESPONSE IN LEUKOCYTES AND AMELIORATES THE INFLAMMATORY PROFILE OF TYPE 2 DIABETIC PATIENTS

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## Resumen

Objectives: SGLT2 inhibitors are a new class of oral anti-diabetic drugs that block renal reabsorption of glucose, decreasing hyperglycaemia in patients with type 2 diabetes. These drugs have been linked to a considerable reduction in cardiovascular risk, but the precise molecular mechanisms underlying their effects are still elusive. We aimed to evaluate the effects of the SGLT2 inhibitor empagliflozin on systemic inflammation and its potential antioxidant properties in the leukocytes of type 2 diabetic patients.

Material and methods: This is an observational, prospective follow-up study of a cohort of fifteen patients with type 2 diabetes who received 10 mg/day of empagliflozin according to standard clinical care. Measurements were taken at baseline, 12 and 24 weeks. Metabolic and anthropometric parameters were evaluated. Production of mitochondrial superoxide, glutathione content, and glutathione s-reductase and catalase mRNA levels were measured in leukocytes. Serum levels of high sensitive C-reactive protein were assessed by immunonephelometry, myeloperoxidase and interleukin-10 levels were determined by Luminex 200.

Results: In addition to a reduction in body weight and an improved metabolic profile characterized by reduced plasma glucose and HbA1c levels, we observed a reduction in superoxide production in leukocytes of diabetic patients together with increased glutathione content that was most pronounced after 24 weeks of empagliflozin treatment. Leukocyte expression of the antioxidant enzymes glutathione s-reductase and catalase and serum levels of anti-inflammatory IL-10 were enhanced after empagliflozin treatment. Concomitantly reduced hs-CRP and myeloperoxidase levels were also observed.

Conclusions: This study provides evidence of the antioxidant and anti-inflammatory properties of empagliflozin treatment in humans, which may underlie its beneficial cardiovascular effects.

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