ALLEVIATION OF MAO-RELATED OXIDATIVE STRESS BY ANTIDIABETIC DRUGS: OF MICE AND MEN

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The global burden of cardiometabolic diseases is expected to increase in the near future with most of related deaths occurring in low and middle-income countries. The major pathomechanisms that underlie these pathologies are chronic oxidative stress and low-grade inflammation that promote each other in a vicious circle leading to both disease progression and the occurrence of complications. Monoamine oxidase (MAO) with two isoforms, A and B, are flavoenzymes located at the outer mitochondrial membrane that have emerged as important sources of cardiovascular oxidative stress. Inflammation is responsible for age-independent increase in MAO expression in the cardiovascular system. A huge body of research demonstrated the role of original antidiabetics in improving the outcome of non-diabetic patients with cardiovascular diseases yet the underlying pathomechanisms remain elusive. Metformin, the central pillar of therapy in type 2 diabetes and Empagliflozin, a largely prescribed SGLT-2 inhibitor, alleviated MAO expression and ROS production in vascular and cardiac preparations from both murine models and humans. Here we provide the evidence for a novel and direct protective effect of antidiabetics in the cardiovascular system, independent of glucose management.