The Effect of Deferoxamine and Ferrostatin-1 Treatments on Total ROS in HK-2 Cells

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Investigations indicate that diabetic patients with myocardial infarctions (MI) have an increased risk of acute kidney injury (AKI) post-MI. In diabetic MI, blood hemoglobin (Hb) rises, which induces the production of reactive oxygen species (ROS) that cause AKI through ferroptosis. Deferoxamine (DFO) and ferrostatin-1 (Fer-1) are treatments that reduce ROS through iron-chelation or the catalysis of radical reduction, respectively. This study investigates how ROS levels in Proximal Tubular Cells (HK-2) change with concentration adjustments of Fer-1 and DFO, as well as whether a mixture of DFO and Fer-1 at 1μM causes a synergic reduction of ROS. The results indicate that DFO, treated at 1μM, 10μM, and 100μM, had a dose-dependent reduction of ROS but did not achieve a significant reduction of ROS ($p>0.05$) at 1μM. Conversely, DFO 1μM significantly increased ROS in the second experiment, indicating possible prooxidant interactions of DFO at low concentrations. Fer-1, treated at 1μM, 5μM, and 10μM, indicated a dose-dependent response until 5μM. However, there was no significant reduction in ROS between 5μM and 10μM due to the unsaturation of Fer-1 past 5μM. When Fer-1 and DFO were mixed at 1μM, the ROS was not significantly different from Fer-1 1μM, so there was no synergic effect ($p>0.05$). Hence, future treatments of Fer-1 and DFO should be done independently, DFO should be treated at concentrations of at least 10μM, where there is a significant reductive effect, and Fer-1 should be treated up to 5μM, where there is maximal ROS reduction.

Awards Won:

Third Award of $1,000