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PROCEEDINGS



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# Microscopic study of ferroptotic death of $\beta$ -cells in diabetogenic conditions *in vitro*

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Ferroptosis is a recently described, programmed form of cell death. It is iron-dependant and characterized by the accumulation of lipid peroxides and reactive species. The main pathological hallmark of diabetes is  $\beta$ -cell loss, and so far, several types of  $\beta$ -cell death have been described. However, involvement of ferroptosis in  $\beta$ -cell loss induced by diabetogenic factors is still unexplored.

The aim of this study was to investigate potential involvement of ferroptosis in the regulation of  $\beta$ -cell loss in diabetes. For that purpose Rin-5F pancreatic  $\beta$ -cells were treated with well-known diabetes-mimicking agents: high glucose (HG; 25 mM), hydrogen peroxide ( $H_2O_2$ ; 75  $\mu$ M) and streptozotocin (STZ; 10 mM) for 12h in the absence or presence of ferroptosis inhibitor, ferrostatin-1 (Fer-1, 1.5  $\mu$ M). As a positive control, an inducer of ferroptosis RSL3 (3  $\mu$ M) was used. Cells were prepared for flow cytometry (death cell assay – propidium iodide (PI) staining; dihydrorhodamine 123 (DHR) reactive oxygen species (ROS) detection) and microscopic analyses (phase contrast analysis of cells viability, morphology and cell confluence; Sudan III detection of neutral lipids and lipofuscin; Prussian blue detection of intracellular iron accumulation; C11-BODIPY detection of lipid peroxides and immunofluorescence detection of phospho-NFE2-related factor 2 (pNrf2)).

Our results demonstrated that mimicking diabetic microenvironment by HG, STZ and  $H_2O_2$  induced ferroptosis of  $\beta$ -cells *in vitro* (Fig. 1), since observed alterations were similar to those induced by RSL3. As we observed microscopically, total cell number decreased, percentage of dead PI+ cells increased and cell changed their morphology from typical to spherical and detached. In addition, increased accumulation of lipid peroxides, ROS, lipids and/or lipofuscin and iron were observed after these treatments. Fer-1 rescued cells from death after all treatments, along with abolishing the effects of those treatments on ROS, lipid peroxides and iron content. Further, Fer-1-induced activation of Nrf2, which is well known as an antioxidant transcriptional factor that regulates level of many of the ferroptosis-related molecules including those involved in metabolism of GSH, iron and lipids. Taken together, our results demonstrated ferroptosis involvement in the  $\beta$ -cell loss under diabetogenic conditions, thus proposing it as a new potential target in the diabetes therapy approach.

Figure 1. Demonstration of ferroptotic-related alterations of Rin-5F pancreatic  $\beta$ -cells treated with ferroptosis inducer RSL3, high glucose (HG), hydrogen peroxide ( $H_2O_2$ ) or streptozotocin (STZ) in the absence or presence of ferroptosis inhibitor, ferrostatin-1 (Fer-1)

**Figure 1**

