

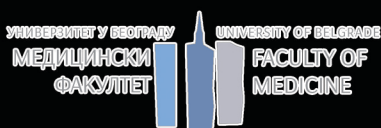


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Streptozotocin, an FDA approved drug, affects the oxidative stress parameters and purinergic signaling components in primary rat astrocyte cultures

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The antibiotic streptozotocin (STZ) is an FDA approved for pancreatic neuroendocrine tumors. It has also been used for rat model of diabetes induction, where it causes a progressive increase in BBB permeability, and activates glial cells. In intracerebroventricular injected STZ-induced AD model, the abnormal mitochondrial morphology, decrease ATP biosynthesis, accumulation of reactive oxygen species (ROS), disrupted homeostasis of brain insulin signaling and defect in cerebral glucose metabolism were observed. Streptozotocin has been used to induce mitochondrial, endoplasmic and in general oxidative stress in neuronal cells and in astrocytoma C6 cell line *in vitro*.

Our study aimed to analyze the STZ effects on primary rat astrocyte cultures. The testing of STZ concentration range (1, 5, 10 and 20 mM) in MTT assay, excluded the 20 mM STZ which evoked a significant decrease in mitochondrial activity in astrocytes. As ROS are the most pronounced parameters elevated in STZ disease modeling, we analyzed GSH, SH groups and MDA 24 h after the STZ application. The 10 mM STZ lowered GSH levels, while SH groups showed a STZ dose dependent decrease. On the other hand, MDA showed a slight, but not significant increase following STZ concentration increase. Moreover, we investigated changes in the purinergic signaling system. Our results show the drop of CD73 activity 24 h after the 10 mM STZ treatment, accompanied by CD73 immunofluorescence decrease on the astrocyte membranes. Similarly, nucleoside triphosphate diphosphohydrolase 2 (NT2) was downregulated on astrocyte membranes. These results encourage further analysis of the P1 and P2 purinergic receptors.