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Diabetes, β cell apoptosis and the Role of SIRT2 and Rel/A NFkB subunit

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Abstract

Introdcution: Streptozotocin is a chemical compound once used as an antibiotic, but its toxicity for the β cells of the islets of Langerhans has dramatically limited its use. This molecule is currently used in an experimental context for the study of type 1 diabetes, due to the latter adverse effects on β cells. In fact, the experimental model of STZ-induced diabetes helps to understand the mechanisms involved in β-cell death.

We performed this murine study to examine new mechanisms by which STZ induces β-cells apoptosis. Two cellular factors SIRT2 and NF-kB were specifically targeted to unveil their potential roles in β-cell survival.

Material & Methods: A culture of a NOD mouse in β-cell line named “NIT1” was exposed to STZ. In the cell lysate, we determined the inhibition of the two factors studied: SIRT2 and NF-kB.

Results: STZ did induce β-cell death with an IC 50 value = 4 µg/mL. In addition, we confirmed that STZ inhibits the phosphorylation of the RelA p65, a NF-kB subunit, leading to the apoptotic process. Also, STZ does significantly inhibit SIRT2.

Conclusion: STZ induces the death of β-cell trough the inhibition of SIRT2 and NF-KB-p65. It is not yet clear whether the two inhibitions are related or not. Further studies are needed to reveal this. These results would also contribute to better understanding of the early stages of β-cell damages and its role in the involving type 1 diabetes.