THE EFFECT OF ESTRADIOL AGAINST TRIBUTYLTIN TOXICITY IN RAT PANCREATIC ISLETS

Introduction: Discovering of estrogen receptors in the pancreatic beta cells supported the hypothesis of protective effects of female sex steroids on pancreatic beta cells due to lower prevalence of diabetes in females. Studies show that estradiol can increase viability of beta cells, enhance insulin secretion and prevent apoptosis in vivo and in vitro.

Some metals particularly tin and cadmium have been reported to interfer with estrogen receptors and exert toxic effects through disrupting related endocrine pathways. Tributyltin is known as an endocrine disruptor which is used as a biocide against a broad range of microorganisms. Some sporadic studies indicated that tributyltin induces beta cell apoptosis and disturbs glucose homeostasis. This study was designed to assess the effects of tributyltin and estradiol on rat pancreatic islets.

Methods: Pancreatic islets of male rat were isolated, grouped (10 islets in each group) and cultured in RPMI 1640 for 24 hours at 37° C. After calculating EC_{50} of tributyltin and estradiol by using MTT assay, islets were treated with estradiol and tributyltin for 24 hours. Then viability and level of reactive oxygen species (ROS), Insulin secretion and apoptosis were measured.

Results: Tributyltin decreased cellular viability of islets along with an increase in the ROS formation, while estradiol increased viability (p value < 0.05) and decreased ROS (p value < 0.05) when added to both control group and tributyltin treated group. Tributyltin also increased insulin secretion (p value < 0.05) and induced apoptosis, while estradiol caused a decrease in insulin secretion and apoptosis induction.

Conclusion: Our results indicate that estradiol can protect beta cells of the pancreas by increasing viability and decreasing ROS formation and apoptosis against tributyltin toxicity. It can be concluded that function of estrogen receptors in the pancreas particularly beta-cells might be an important target of pharmacological and toxicological modifications in order to discover new aspects of pathophysiology and therapeutic strategies for diabetes.

Key words: Islets of Langerhans, Estrogen receptors, Estradiol, Tributyltin