

## **Abstract**

Diabetes mellitus is a chronic autoimmune disease in which the immune system attacks the insulin-secreting  $\beta$ -cells in the pancreas. It leads to an absolute deficiency of insulin.

Chronic hyperglycemia induces increased production of reactive oxygen species, which leads to a decrease of natural antioxidant level in blood, and it contributes to genesis of diabetes complications (e.g. vascular or pulmonic). Moreover, the oxidative stress results in onset of pancreas inflammations and the damage of its  $\beta$ -cells.

**Aims:** Our aim was to assess whether or not certain genotypes or their combinations occur with higher frequency among groups of patients of type 1 diabetes (T1D) and type 2 diabetes and in a control group of healthy individuals.

**Methods:** The study included groups of 40 T1D patients, 40 T2D patients and 45 healthy individuals. The polymorphisms of genes involved in the oxidative stress response were analyzed by using RFLP, PCR with TaqMan probes and allele specific PCR. The target genes involved superoxide dismutase *SOD1* and *SOD3* genes; glutathione-S-transferase *GSTM1*, *GSTT1*, *GSTP1* genes; glutathioneperoxidase gene *GPX1* and catalase gene *CAT*. The levels of plasma malondialdehyde were measured by using liquid chromatography.

**Results:** Statistically significant differences were found in the frequencies of *SOD1* (null vs. positive polymorphism in control group vs. T2D and T1D vs. T2D), *SOD3* (T1D vs. T2D), combination of *GSTT1* null and *GSTM1* wild type (T1D vs. T2D) genotypes and as well as (among the groups) in the levels of oxidative stress biomarker malondialdehyde.