

THE COMPARISON OF THE OCCURRENCE OF BETA CELLS AUTOANTIBODY AND REGULATORY T CELLS (CD4+CD25+FOXP3+) IN PATIENTS WITH TYPE 1 DIABETES, THEIR SIBLINGS AND CONTROL GROUP.



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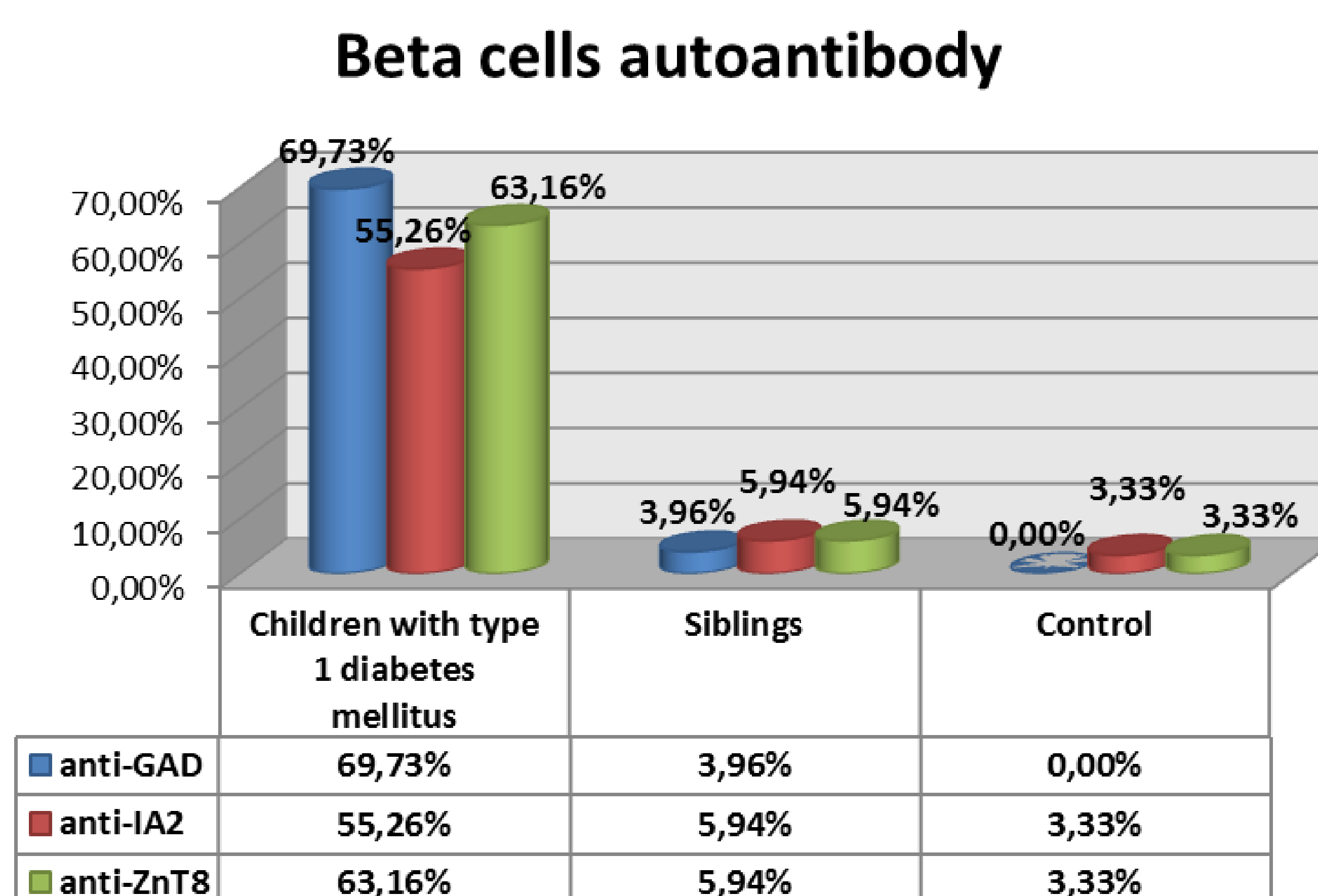
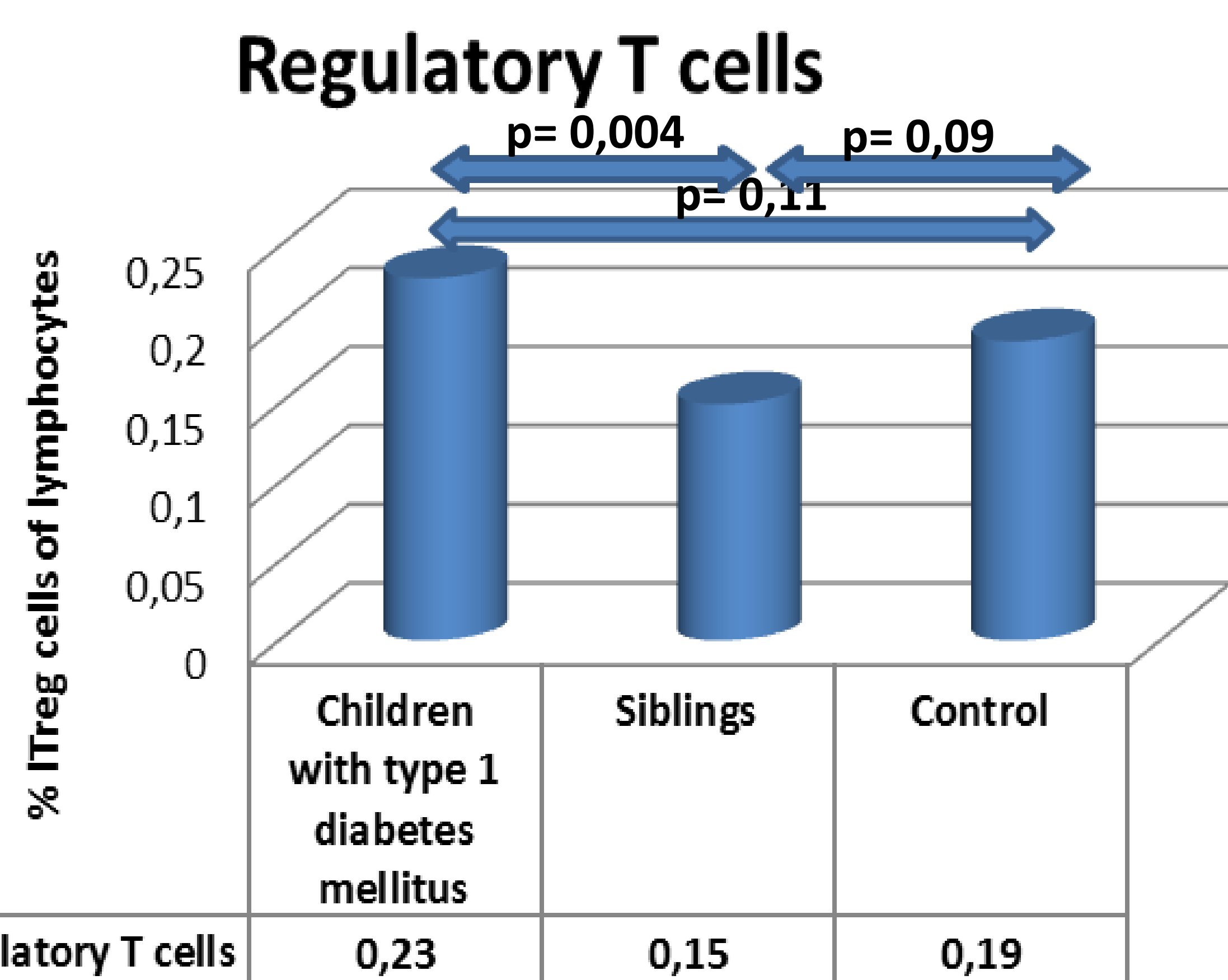
Background

Regulatory T cells (Treg) of phenotype CD4+CD25+FoxP3+ involves active suppression of excessive immune response. The population of Treg cells from patients with type 1 diabetes (DM1) have numeric and functional abnormalities. Although there are many reports of investigations on human and animal populations, the role of regulatory T cells in the development of type 1 diabetes is still unclear. The aim of the study is to compare the population of regulatory T cells and the correlation between Treg cells and beta cells autoantibody in healthy siblings of children with DM1 to healthy children from non-diabetic families and to children with DM1.

Methods

Peripheral blood mononuclear blood cells were obtained from 76 children with DM1, their siblings - 101, and 30 healthy children. Treg cells were characterized by flow cytometry FACSCalibur (Becton Dickinson, USA).

The auto-antibodies were determined by ELISA. The results were analyzed with STATISTICA 10 PL.



Results

The number of regulatory T cells from diabetic patients was higher (average percentage $0,23 \pm 0,20$) than that in the siblings ($0,15 \pm 0,14$) ($p=0,004$). There was no significant difference in the number of Treg cells between children with DM1 and the control group ($0,19 \pm 0,15$; $p=0,11$) and between siblings and the control group ($p=0,09$).

The levels of anti IA2 and anti ZnT8 antibodies were statistically significant higher in siblings in comparison to the control group (anti IA2 Ab $p=0,0000001$; anti ZnT8 Ab $p=0,00001$). The level of anti-GAD in siblings was similar to that in the control group. There was no correlation between the number of Treg cells and the co-occurrence of beta cells auto-antibody.

Conclusion

The results suggest that regulatory T cells probably provide protection from development of disease and the dysfunction of Treg cells contributes to the autoimmune pathogenesis of type 1 diabetes.

