The effect of gluten-free diet on β-cell residual capacity, immune function and gut microbiome in children with newly diagnosed type 1. diabetes

Abstract

The pathophysiology of the onset and progression of type 1 diabetes (T1D) is not fully understood. Gluten has a proinflammatory effect on the immune system and is therefore considered as one of the factors affecting the onset and progression of T1D.

The aim of the thesis is to allow a complex insight into the role of the GFD on the residual β-cell capacity, T1D control, gut microbiome, gut permeability, subtypes of immune cells and the effect of gut microbiome transfer into germ-free non-obese diabetic (NOD) mice on the incidence of diabetes.

On the group of 45 children with T1D (26 intervention group, 19 control group) we proved the association of the GFD with slower decrease of β-cell residual capacity (the difference in the trend of C-peptide decrease 409 pmol/l/year; $p = 0,04$) and lower HbA_1c (by 7,8 mmol/mol; $p=0,02$). We also described the changes in the gut bacteria that were differentially abundant after the administration of the GFD and the changes in abundance of the regulatory and effector immune cells. We showed there was no change in the gut permeability with respect to the study group. We also proved that the transfer of human gut microbiota to germ-free non-obese diabetic mice leads to the decrease in the incidence of diabetes in these mice.

Altogether, we offered a complex insight into a number of changes that are associated with the initiation of the GFD in children with T1D and thus extended the knowledge of the effects of gluten on the human body.