ABSTRACT

Autoimmune diabetes (type 1 diabetes, T1D) is a multifactorial disease, where the immune system reacts against pancreatic β-cells of Langerhans islets. Their progressive destruction causes insufficient function of insulin for the organism and production of autoantibodies. These antibodies serve as markers of autoimmune insulitis even before the manifestation of the disease.

T1D is triggered by environmental factors in genetically predisposed individuals. Genetic susceptibility mainly relates to HLA genes, which cause about 50 % of familiar occurences. The most risk factors are HLA-DR3, HLA-DR4 and HLA-DQ8 antigens. Their aminoacidic structure determines which peptides would bind to individual antigens and how firmly. However, these antigens do not function separately. They are in tight linkage disequilibrium and it is therefore not possible to determine the individual function of each of them in autoimmune process unambiguously.

Environmental factors can have protective effect on individuals (e.g. vitamin D), but they can have predisposition effect for autoimmunity as well. The most discussed trigger mechanisms are viral infections (specifically Coxsackie B4 virus) and premature exposure of newborns to cow-milk.