Abstract

Type 1 diabetes (T1D) is an organ specific autoimmune disorder characterised by the immune-mediated destruction of insulin-producing pancreatic beta cells. Beta-cell destruction is mediated primarily by cellular components of the immune system, especially auto-reactive T cells. Nowadays, a goal of many studies is built up the best system for identification of individuals in prediabetes stage and to treat them to preserve sufficient amount of insulin producing beta cells.

We identified several candidate pathways and proteins which could be important in pathology of T1D, like an antiviral responses and differentiation of Th17 pathways. We observed differences in dendritic cells count and in their cytokines production. Our data support the notion that the establishment of proinflammatory environment in genetically predisposed individuals along with the involvement of non-specific immune mechanisms is critical for the initiation of autoimmune, destructive insulitis. Nonetheless, patient's autoantibody profile reflects the type of cellular immune response and should be take in a count as well. This finding may be useful in design of immunointervention studies to prevent T1D.

Considering the heterogeneity of the clinical course of this disease and perhaps different mechanisms of molecular pathology, immune interventions should be more personalised.

Key words: Type 1 diabetes, islets of langerhans, insulitis, autoimmunity, autoreactive T cells, Th17, DC, cytokines, autoantibodies, microarray