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Alguayn A.

THE ROLE OF VIRUSES AND AUTOIMMUNE PROCESSES IN THE DAMAGE OF BETA CELLS IN TYPE 1 DIABETES MELLITUS

Tutor: PhD, associate professor Chepelev S.N.
Department of Pathological Physiology
Belarusian State Medical University, Minsk

Diabetes Mellitus (DM) is a syndrome of disturbed metabolism involving carbohydrate, protein, and fat which results from the degree of insulin deficiency (absolute or relative) and tissue sensitivity to its actions.

Absolute insulin deficiency (Type 1 DM) occurs with autoimmune destruction of insulin secreting β-cells (Type 1A DM) and other congenital (genetic defects in the formation or function of the endocrine pancreas), or acquired (relapsing pancreatitis and pancreatectomy) conditions.

Here we review the Pathogenesis of T1DM is different from that of type 2 diabetes mellitus, where both insulin resistance and reduced secretion of insulin by the β cells play a synergistic role. We will present genetic, environmental and immunologic factors that destroy β cells of the endocrine pancreas and lead to insulin deficiency.

Type 1 diabetes is usually present in individuals without a family history. The main genes predisposing to T1DM are within the major histocompatibility complex (MHC) region, often called HLA a closely related genetic loci encoding molecules expressed on the surface of immune or other cells and limiting the specificity of T lymphocytes to recognize antigens. HLA gene loci are distinguished into two classes. Class I HLA molecules are associated with antigens that are recognized by the receptor of cytotoxic T lymphocytes (CD8+), which eventually destroy the antigenic target and are expressed by most cells. Class II HLA molecules are essential for the recognition of antigens by T helper lymphocytes (CD4+), which initiate the immune response and promote cellular co-operation and are expressed only by immune cells.

Insulin-VNTR, Insulin-VNTR polymorphisms near the insulin gene contribute to 10% of T1DM genetic predisposition. This region on chromosome 11 consists of variable tandem repeats, with two common classes: small and large. Type III is protective against T1DM by promoting insulin presentation in the thymus, eliminating autoreactive T cells, and safeguarding pancreatic beta cells from autoimmune damage.

CTLA-4, The CTLA-4 gene on chromosome 2 regulates T cell activation and immune response. It provides negative signals to activated T cells, inducing tolerance and preventing autoimmunity. By competing with CD28 for binding to B7 molecules, CTLA-4 limits T cell activation and proliferation. Polymorphisms in CTLA-4 are associated with autoimmune disorders like T1DM, disrupting immune regulation and potentially leading to autoimmune responses.

Environmental factors also play an important role in the pathogenesis of T1DM. The environmental factors involved include viruses (rubella, coxsackievirus B or enteroviruses), toxins and nutrients (cow’s milk, cereals).

Understanding the intricate interplay between viruses, autoimmune processes. Viral infections can trigger autoimmune responses that lead to the destruction of beta cells in the pancreas. This process, known as molecular mimicry or direct damage, can cause autoimmune diseases like Type 1 Diabetes. The interplay between viruses and autoimmune processes impacts insulin production and blood sugar regulation by targeting and damaging beta cells.

In conclusion Type 1 Diabetes is caused by the immune system mistakenly attacking insulin-producing cells due to a combination of genetic and environmental factors, leading to a lack of insulin production and high blood sugar levels. Viruses may trigger autoimmune responses in susceptible individuals, but they do not directly cause Type 1 Diabetes.