IMMUNOLOGICAL MECHANISMS SHAPING THE DEVELOPMENT OF TYPE 1 DIABETES MELLITUS

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The aim of this study was to review the immunological mechanisms shaping the development of type 1 diabetes mellitus. A bibliographic review of the narrative type was performed from the databases SciELO, PubMed and Google Scholar, using the keywords: type 1 diabetes mellitus, immune response, and pathogenesis. It has been observed that the type 1A Diabetes Mellitus (T1D) is an autoimmune disease that typically manifests in children and adolescents, resulting in the destruction of insulin-producing cells in the pancreas. The incidence of T1D has been on the rise in children under 15 years of age over the years. This condition is triggered by the activity of TCD8+ immune cells, which mistakenly attack insulin-producing cells. Furthermore, research indicates that children recently diagnosed with T1D exhibit altered genetic patterns in regulatory T cells (Tregs), suggesting the possibility of an immunoregulatory biomarker. The imbalance in insulin production leads to fluctuations in glucose levels and short- and long-term complications, including severe hypoglycemia and cardiovascular diseases. This integrative review aimed to synthesize the immunological mechanisms involved in T1D. Studies highlighting the significance of the major histocompatibility complex (MHC) and variations in Treg and TCD8+ cells in T1D pathogenesis were analyzed. The findings indicate that MHC class II shows a strong correlation with the disease, especially specific genes, while Treg and TCD8+ cells play crucial roles in the autoimmune response. Understanding these mechanisms is essential for early detection and improving the quality of life of T1D patients, as well as providing valuable insights for future treatments. Given the profound impact of T1D on individuals' lives and healthcare systems, comprehending the immunological and metabolic mechanisms involved is paramount for early detection and enhancing the quality of life of those affected. Introduction to T1D: T1D is an autoimmune disease characterized by the selective destruction of pancreatic beta cells responsible for insulin production. Typically, this condition manifests in children or adolescents, with an increased incidence in individuals under 15 years of age. Humoral and Cellular Autoimmunity: T1D involves an autoimmune response that includes the presence of autoantibodies, such as anti-pancreatic islet, anti-insulin, and anti-glutamic acid decarboxylase (GAD-65) antibodies. These autoantibodies are associated with the process of beta cell destruction. Cellular Immunity: The immune system plays a fundamental role in beta cell destruction. This involves cells such as CD4+ and CD8+ T lymphocytes, macrophages, and dendritic cells. These cells act in insulitis, which is the inflammatory process in pancreatic islets. Genetic Susceptibility: Susceptibility to T1D is inherited and strongly linked to certain HLA (human leukocyte antigen) system genotypes. Specific alleles increase the risk of developing the disease. Conclusions, T1D is a complex disease resulting from the interplay of genetic and environmental factors, leading to the destruction of insulin-producing beta cells. Understanding these factors is essential for research and the development of more effective therapeutic approaches Environmental Factors: Environmental factors, such as viral infections, childhood diet, and toxins, also play a significant role in T1D development. They can act as triggers to initiate the disease in genetically predisposed individuals.

Keywords: Type 1 Diabetes Mellitus; Immune System Diseases; Immunological Mechanisms.

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