Discussion

Name of Student

Institutional Affiliation

1-1 Discussion

Type 1 diabetes is characterized by the immune system mistakenly attacking and destroying insulin-producing beta cells in the pancreas. Genetics plays a critical role in this disease. Specific genes increase the risk of developing Type 1 diabetes, particularly those linked to the human leukocyte antigen (HLA) complex, which helps regulate the immune system (Klak et al., 2020). These genetic markers influence how the body differentiates between its cells and potentially harmful invaders. A person inheriting these specific genes from both parents may have a higher risk of developing this condition.

Patients with Type 1 diabetes present with symptoms such as increased thirst, frequent urination, hunger, fatigue, and blurred vision. These symptoms arise because the body can no longer produce insulin, which is necessary for cells to absorb glucose from the bloodstream and convert it into energy. Without insulin, sugar builds up in the blood, leading to hyperglycemia. The kidneys respond by trying to eliminate excess glucose through urine, which explains the frequent urination and consequent dehydration and increased thirst.

The physiological response to the lack of insulin is the body's attempt to find alternative energy sources. Cells, deprived of their primary energy source, glucose, begin to break down fats and proteins, leading to weight loss and fatigue. The process also leads to the production of ketones, which can accumulate in the blood and lead to diabetic ketoacidosis, a potentially life-threatening condition.

The primary cells involved in this process are the pancreatic beta cells, immune cells that mediate the autoimmune response, and various cells in the kidneys, eyes, and nervous system that are affected by high blood sugar levels. Gender and genetic variations can influence the presentation and management of Type 1 diabetes (Giandalia et al., 2021). For instance, males and females might experience different onset ages and rates of progression, and genetic differences can affect the susceptibility to and severity of the disease. These

variations necessitate personalized approaches to treatment and management, emphasizing the importance of understanding individual characteristics in the context of autoimmune diseases like Type 1 diabetes.

References

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Klak, M., Gomółka, M., Kowalska, P., Cichoń, J., Ambrożkiewicz, F., Serwańska-Świętek, M., Berman, A., & Wszoła, M. (2020). Type 1 diabetes: Genes associated with disease development. *Central European Journal of Immunology*, *45*(4), 439–453.

Responses

Hello Sarah,

I agree with your assessment regarding the impact of genetics on the progression of Type 1 Diabetes Mellitus. Your explanation of how genetic predispositions contribute to the disease aligns with recent studies highlighting the role of HLA genes in autoimmune responses. However, I would like to add a perspective on the environmental triggers that might interact with these genetic factors. Research indicates that environmental factors, such as viral infections, might act as a catalyst for the onset of Type 1 diabetes in genetically predisposed individuals. This interaction between genes and the environment could explain variations in disease onset and severity, adding another layer to our understanding of its pathophysiology. Your focus on the genetic aspects is crucial, but incorporating environmental influences could provide a more holistic view of the disease's etiology.

Hi Richard,

Your analysis of the physiological responses in Type 1 diabetes, particularly the role of insulin deficiency leading to hyperglycemia, is well-articulated. However, I have a different viewpoint on the symptom of increased hunger (polyphagia) you described. While it is accurate that insulin deficiency leads to poor glucose uptake by cells, causing energy deprivation that triggers hunger, it is also important to consider that this symptom can be exacerbated by the inefficiency of the signaling pathways involved in satiety and hunger in the hypothalamus. This brain region plays a critical role in regulating energy homeostasis, which is disrupted in the context of diabetes due to fluctuating blood glucose levels. Therefore, the symptom of hunger not only arises from cellular glucose deprivation but also from a disrupted central regulatory mechanism, which you might consider discussing in further depth to enhance understanding of the systemic impacts of this disease.