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Editorial

Immunologically Tolerated Despite of Human Pancreas

Saeid Bouzari*

Associate Molecular Biology Unit, Pasteur Institute of Iran, Pasteur Ave, Iran

The human pancreas, like almost all organs in the human body, is immunologically tolerated despite the presence of innate and adaptive immune cells that promptly mediate protective immune responses against pathogens *in situ*. The PD-1/PD-L1 inhibitory pathway seems to play a key role in the maintenance of immune tolerance systemically and within the pancreatic tissue. Tissue resident memory T cells (TRM), T regulatory cells (Treg), macrophages and even β cells exhibit PD-1 or PD-L1 expression that contributes in controlling pancreatic immune homeostasis and tolerance. Dysregulation of the PD-1/PD-L1 axis as shown by animal studies and our recent experience with checkpoint inhibitory blockade in humans can lead to immune dysfunctions leading to chronic inflammatory disease and to type 1 diabetes (T1D) in genetically susceptible individuals. In this review, we discuss the role of the PD-1/PD-L1 axis in pancreatic tissue homeostasis and tolerance, speculate how genetic and environmental factors can regulate the PD-1/PD-L1 pathway, and discuss PD-1/PD-L1-based therapeutic approaches for pancreatic islet transplantation and T1D treatment.

Type 1 diabetes

We Type 1 diabetes (T1D) is a multifactorial disease of unknown aetiology. Studies focusing on environment-related prenatal changes, which might have an influence on the development of T1D, are still missing. Drugs, such as betamethasone, are used during this critical period without exploring possible effects later in life. Betamethasone can interact with the development and function of the two main players in T1D, the immune system and the pancreatic β-cells. Short-term or persistent changes in any of these two players may influence the initiation of the autoimmune reaction against β-cells. In this review, we focus on the ability of betamethasone to induce alterations in the immune system, impairing the recognition of autoantigens. At the same time, betamethasone affects β-cell gene expression and apoptosis rate, reducing the danger signals that will attract unwanted attention from the immune system. These effects may synergise to hinder the autoimmune attack. In this review, we compile scattered evidence to provide a better understanding of the basic relationship between betamethasone and T1D, laying the foundation for future studies on human cohorts that will help to fully grasp the role of betamethasone in the development of T1D.

Complications

Perinatal exposure to maternal obesity and high-fat diet (HFD) consumption not only poses metabolic risks to offspring but also impacts brain development and mental health. Using a non-human primate model, we observed a persistent increase in anxiety in juvenile offspring exposed to a maternal HFD. Post weaning HFD consumption also increased anxiety and independently increased stereotypic behaviours. These behavioural changes were associated with modified cortisol stress response and impairments in the development of the central serotonin synthesis, with altered tryptophan hydroxylase-2 mRNA expression in the dorsal and median raphe. Post weaning HFD consumption decreased serotonergic immunoreactivity in area 10 of the prefrontal cortex. These results suggest that perinatal exposure to HFD consumption programs development of the brain and endocrine system, leading to behavioural impairments associated with mental health and neurodevelopmental disorders. Also, an early nutritional intervention (consumption of the control diet at weaning) was not sufficient to ameliorate many of the behavioural changes, such as increased anxiety, that were induced by maternal HFD consumption. Given the level of dietary fat consumption and maternal obesity in developed nations these findings have important implications for the mental health of future generations.

Correspondence: Saeid Bouzari, Associate Molecular Biology Unit, Pasteur Institute of Iran, Pasteur Ave, Iran.

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