OPINION ARTICLE

Impact of Human Pancreas in Immunity and in Diabetes

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ABSTRACT

Autoimmune Pancreatitis (AIP) is a persistent inflammation that responds to steroid medication and is thought to be caused by the body's immune system attacking the pancreas. Pancreas-activated neutrophils generate more cytokines, enhancing the inflammatory response by recruiting extra neutrophils, macrophages, and innate immune cells. Both acute and chronic pancreatitis might cause your pancreas to produce fewer enzymes required to break down and utilize nutrients from food. Even if you eat the same meals or the same amount of food, this can result in malnutrition, diarrhoea, and weight loss.

INTRODUCTION

Salt and fluid absorption and secretion are two processes that are critical to epithelial function and whole-body fluid homeostasis, and are thus closely regulated in epithelial tissues. The CFTR anion channel regulates secretion and absorption in a variety of epithelial tissues, including the lungs, GI and reproductive tracts, sweat and salivary glands. It is not unexpected, then, that CFTR function deficiencies are connected to disease, including life-threatening secretory diarrhoea like cholera, as well as the inherited disease Cystic Fibrosis (CF), one of the most common life-limiting genetic diseases in many cultures. More recently, CFTR failure has been linked to the aetiology of acute pancreatitis, chronic obstructive pulmonary disease (COPD), and asthma hyperresponsiveness, highlighting its critical role in overall health and disease. CFTR modulates several pathways in epithelial physiology, including epithelial surface hydration and luminal pH regulation. Indeed, recent research has implicated luminal pH as a critical regulator of epithelial barrier function and innate defence, notably in the airways and gastrointestinal tract. In this chapter, we will describe the properties of CFTR in three different tissues to demonstrate its various operational functions in epithelial function: the airways, the pancreas, and the sweat gland [1].

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T1DR is Typically Characterized by

- Variable degree of insulitis and loss of insulin staining on pancreas transplant biopsy (with most commonly absent), minimal to moderate, and rarely severe pancreas and/or kidney transplant rejection;
- The conversion of T1D-associated autoantibodies (to the autoantigens GAD65, IA-2, and ZnT8), preceding hyperglycemia by a variable length of time; and
- The presence of autoreactive T cells in peripheral blood, pancreas transplant, and/or peripancreatic transplant lymph nodes.

There is no therapeutic regimen that has so far controlled the progression of islet autoimmunity, even when additional immunosuppression was added to the ongoing chronic regimens; we hope that future research, particularly in-depth analysis of pancreas transplant biopsies with recurrent diabetes, will aid in the identification of more effective therapeutic approaches. [2].

The identification of innate immune receptors such as the Toll-like receptor and the Nod-like receptor has highlighted the relevance of innate immunity in host defence. The innate immune system is relevant in a variety of clinical circumstances, including autoimmune disorders. The role of innate immunity in the aetiology of metabolic disorders such as type 2 diabetes, metabolic syndrome, or atherosclerosis, which were traditionally thought to be inflammatory disorders, is also being recognized [3].

Most clinical trials have focused on only one piece of the issue, immunological suppression, with little success. The gastrointestinal tract is a critical area where our genes interact with the environment. Before we can develop long-term methods of preventing, treating, or curing T1D, we must first understand the influence of all of its primary

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contents, including microorganisms, nutrition, and immune system. Based on recent discoveries in the field, we expand our prior gut-centric model in this study [4].

Recurrence of insulin-dependent diabetic mellitus following pancreatic transplantation has previously been described relatively infrequently. However, newer data show that recurrence rates are substantial. Diabetes recurs after identical-twin pancreatic transplants in the absence of immunosuppressive medication, indicating that it is an autoimmune disease. Immunologic markers (autoantibodies, autoreactive T cells) predict recurrence after deceased donor pancreatic transplantation. Selective cell destruction, which is still uncommon, is not limited to MHC compatibility. Diabetes in living pancreatic donors is uncommon; it can be avoided in large part by rigorous metabolic examination prior to donation and control of obesity [5].

CONCLUSION

This effort uncovered a slew of studies that call numerous T1D dogmas into question, as well as data indicating the disease's pathology's remarkable heterogeneity. Improved understanding and respect for pancreatic pathology in T1D may lead to better disease classification, a better explanation of why the problem arises, and better therapeutics for disease prevention and management.

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