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Pancreatic Disorders in Cancer, Diabetes, and Obesity

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The Elusive Pancreas

The pancreas is located posterior to the stomach, and is considered secondarily retroperitoneal. Its head, neck, and body lie behind the peritoneum with its tail being the only part that is intraperitoneal. Ironically, not only is pancreas elusive anatomically, but a lot of its disorders from exocrine gland are usually not detected until they are too late. Both its anatomy and high functional reserve make the early detection difficult for pancreatic exocrine disorders.

Pancreatic Carcinoma

In 2012, pancreatic cancer, which consists mostly of pancreatic carcinoma of the exocrine gland, was estimated to have 43,920 new cases in the United States. However, its deaths were estimated to be 37,390 [1]. The projected low survival rate is, again, the result of its late detection. Unfortunately, the risk factors for pancreatic cancer remain poorly understood, and a better early diagnosis is earnestly needed. As of 2002, pancreatic cancer had the fourth highest cancer-related mortality rate in the world [2].

Diabetes

The role of pancreas in the development of diabetes certainly cannot be overemphasized. Type I Diabetes Mellitus (T1DM) is an autoimmune disorder, in which the pancreatic insulin-producing β cells are destroyed by one's own immune system. In contrast, the disease progression of Type II Diabetes Mellitus (T2DM), the most common type of diabetes, is generally affected by lifestyle factors. It remains unclear whether the development of T2DM is mainly due to reduced number of β cells, reduced function of β cells, or reduced insulin sensitivity of the target cells.

Studies have shown that the number of β cells was reduced in T2DM patients, and that the reduction in their cell number was due to β cells apoptosis [3]. However, the decrease in β cells number cannot be the sole etiology for T2DM. Mice lacking M3 muscarinic acetylcholine (ACh) receptors in pancreatic β cells displayed impaired insulin secretion and glucose tolerance, whereas mice over expressing M3 receptors displayed increased insulin secretion and glucose tolerance [4]. This study suggests that parasympathetic nervous system may affect the functionality of pancreatic β cells, and a complex crosstalk between nervous system and digestive system may be crucial in maintaining the functionality of β cells. In another interesting study, the normalization of β cell function and insulin sensitivity in T2DM was made possible by an eight-week calorie restriction [5]. These calorie-restricted T2DM participants also had reduced pancreas triglyceride, suggesting that excess lipid storage in pancreas may be detrimental to β cell function.

Obesity

The link between obesity and T2DM remains unclear. As described above, recent studies have suggested that ectopic lipid accumulation in pancreas during the development of obesity may disrupt β cell function [5,6]. Fortunately, the disruption can potentially be reversed by dietary regimen.

Besides the decrease in β cell number and function, emerging evidence has also implicated insulin resistance of the target cells in the progression of T2DM. As the amount of adipose tissue increased, the number of macrophages infiltrating the adipose tissues also increased [7,8]. These adipose tissue macrophages apparently released inflammatory cytokines, which could lead to insulin resistance in the peripheral tissues. The ectopic lipid accumulation in pancreas and the "obesity-induced low-grade chronic inflammation" [9] are thought to play significant roles in the development of T2DM in obese individuals.

Future Directions

Pancreatic disorders and therapy remain to be critical areas of research. We are faced with many challenges. Effective early diagnosis of pancreatic exocrine disorders, including early detection of pancreatic carcinoma, is urgently needed. In addition, more studies on the crosstalk between pancreatic β cells and other organ systems are needed for a better understanding of the etiology of T2DM.

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