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Sommario/riassunto

Pancreatic diseases include intractable ones including acute and chronic pancreatitis, and pancreatic cancer. In recent years, great advances have been made in the field of pancreatology, including the pathogenesis, diagnostic modalities, and development of novel therapeutic interventions. It has been established that pancreatic stellate cells play a pivotal role in the development of pancreatic fibrosis in chronic pancreatitis as well as in pancreatic cancer known as desmoplastic reaction. Although it might be still controversial, accumulating evidence has shown that interaction between pancreatic stellate cells-cancer cells contribute to the progression of pancreatic cancer through the increased proliferation and migration, and production of cytokines and extracellular matrix components. In addition, pancreatic stellate cells lead to the resistance to chemotherapy and radiation therapy. Pancreatic stellate cells attract the researchers as a novel therapeutic target of pancreatic cancer. Genetic studies have shown that mutations in the trypsin-related genes such as cationic trypsinogen (PRSS1) gene and the serine protease inhibitor, Kazal type 1 (SPINK1) gene are associated with pancreatitis. In general, each of these factors appears to limit trypsin activation or enhance inactivation, and is believed to increase intrapancreatic trypsin activity and predispose to pancreatitis when the gene is mutated. These results have supported a concept that pancreatic protease/anti-protease plays pivotal roles in the pathogenesis of pancreatitis. In addition, genetic

studies focusing on phenotypic variances would provide us with important information how genetic variants would affect the phenotypic variances. Autophagy is an intracellular bulk degradation system in which cytoplasmic components are directed to the lysosome/vacuole by a membrane-mediated process. Recent studies have highlighted a role of autophagy in acute pancreatitis. Using a conditional knockout mouse that lacks the autophagy-related (Atg) gene Atg5 in the pancreatic acinar cells, autophagy exerts a detrimental effect in pancreatic acinar cells by activation of trypsinogen to trypsin. A theory in which autophagy accelerates trypsinogen activation by lysosomal hydrolases under acidic conditions, thus triggering acute pancreatitis in its early stage. The epithelial-mesenchymal transition is a developmental process that allows a polarized epithelial cell to undergo multiple biochemical changes that enable it to assume a mesenchymal phenotype. The phenotype associated with epithelial-mesenchymal transition includes enhanced migratory capacity, invasiveness, elevated resistance to apoptosis, and greatly increased production of extracellular matrix components. In addition to its role in development, tissue regeneration, and fibrosis, epithelial-mesenchymal transition is now considered as a critical process in cancer progression. Induction of epithelial-mesenchymal transition in cancer cells results in the acquisition of invasive and metastatic properties. Epithelial-mesenchymal transition could be an important mechanism in the progression of pancreatic cancer and its poor prognosis. Autoimmune pancreatitis is a unique form of pancreatitis in which autoimmune mechanisms are suspected to be involved in the pathogenesis. There is accumulating study to deal with this new disease concept. In addition to these topics, we have selected several topics in pancreatology, focusing on recent studies increasingly deepening our knowledge in both basic and clinical researches.