COMMENTARY

The Role of Pancreatic Proteases in the Diagnosis and Management of Chronic Pancreatic Insufficiency (CPI)

Mason Schaider*

Division of Digestive Diseases, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia, United States

ABSTRACT

Trypsin, chymotrypsin, and carboxypeptidase are pancreatic proteolytic enzymes that are secreted as zymogens, or inactive precursors of the enzymes, and activated in the digestive canal lumen. Digestion enzymes and bicarbonate are two secretory products found in pancreatic juice that are essential for effective digestion. The exocrine acinar cells produce and secrete the enzymes, whereas the epithelial cells lining tiny pancreatic ducts secrete bicarbonate. Protein digestion begins in the stomach with pepsin, but the pancreatic proteases do the majority of the work. The pancreas produces many proteases, which are released into the small intestine lumen. Trypsin and chymotrypsin are two primary pancreatic proteases that are produced and packaged as the inactive proenzymes trypsinogen and chymotrypsinogen in secretory vesicles. Triglyceride, or neutral lipid, is a key component of dietary fat. A triglyceride molecule cannot pass through the intestinal mucosa directly. Instead, it must be broken down into two monoglycerides and two free fatty acids. Pancreatic lipase, which is transported into the gut lumen as a part of pancreatic juice, is the enzyme that executes this hydrolysis. Starch, a storage form of glucose in plants, is the main dietary carbohydrate for many animals. Pancreatic secretions are the main source of amylase in all species, while some animals, including humans, have amylase in their saliva.

INTRODUCTION

The critical process by which acute pancreatitis begins is assumed to be autodigestion by proteolytic enzymes. The location and mechanism of activation of pancreatic proteases, which are physiologically stored and released as inactive precursor zymogens, has remained a mystery. The lysosomal protease cathepsin B can activate trypsinogen *in vitro* in a manner similar to enterokinase activation; cathepsin B colocalizes with trypsinogen in the secretory compartment of the rat and human pancreas; trypsinogen activation begins in a secretory compartment distinct from mature zymogen granules; and cathepsin B inhibition can either specificity and its site of action in the pancreatic acinar cell [1].

Role of Pancreatic Proteases

A genetic susceptibility to the development of acute recurring and chronic pancreatitis exists in some people. Pancreatitis can be classed as hereditary or familial if there is a considerable family history of the disease. The authors discuss the specific genes linked to pancreatitis in this article, as well as the relevance of genetic testing in diagnosis and the impact of genetic testing results on clinical therapy [2].

The importance of pancreatic proteases in the diagnosis and management of chronic pancreatic insufficiency (CPI), with a focus on trypsin, chymotrypsin, and elastase, with an emphasis on recent improvements. In addition, some essential unique characteristics of these enzymes in acute pancreatitis are discussed, such as their significance in diagnosis and their interaction with cholecystokinin in disease progression. The new interest in these enzymes as cancer-promoting agents in both animals and humans is also discussed. The advantages and limits of tests in different source materials, such as serum, faeces, duodenal aspirate, and non-invasive pancreatic function tests, were investigated using a hierarchical method. One of the important lessons to be gleaned from an analysis of the recent literature is the practical value of faecal elastase-1 and faecal chymotrypsin concentrations in the diagnosis and therapy of CPI, respectively. This is the main message of this study. [3].

Treatment of Chronic Pancreatic Insufficiency

Exocrine insufficiency of the pancreas is a common cause of malnutrition and a major consequence of chronic pancreatitis. Normal digestion necessitates appropriate pancreatic secretory stimulation, adequate generation of digestive enzymes by pancreatic acinar cells, an unobstructed pancreatic duct system, and adequate mixing

Received 18-Mar-2022 Manuscript No IPP-22-739 **Editor Assigned** 21-Mar-2022 PreQC No IPP-22-739(PQ) **Reviewed** 05-Apr-2022 QC No IPP-22-739 **Revised** 08-Apr-2022 Manuscript No IPP-22-739(R) **Published** 12-Apr-2022 DOI 10.35841/1590-8577-23.4.739
Keywords Pancreatic cancer; Pancreas; Pancreatic proteases; Chronic pancreatic insufficiency
Correspondence Mason Schaider
Division of Digestive Diseases, Department of Medicine
Emory University School of Medicine
Atlanta, Georgia, United States
E-mail schaider.m2009@gmail.com

Citation: Schaider P. The role of pancreatic proteases in the diagnosis and management of Chronic Pancreatic Insufficiency (CPI). JOP. J Pancreas. 2022;23(4):739.

of pancreatic juice with ingested food. Failure to complete any of these phases can lead to pancreatic exocrine insufficiency, which can cause steatorrhea, weight loss, and malnutrition-related problems like osteoporosis. The most accurate tests for pancreatic exocrine insufficiency are methods that evaluate digestion, such as faecal fat quantification and the C-mixed triglycerides test, but the probability of diagnosis can also be estimated based on symptoms, signs of malnutrition in blood tests, faecal elastase 1 levels, and signs morphologically severe chronic pancreatitis on imaging [4].

Etiologies of Exocrine Pancreatic Insufficiency

Primary pancreatic disorders or subsequently decreased exocrine pancreatic function produce Exocrine Pancreatic Insufficiency (EPI), a common cause of maldigestion and malabsorption. Other causes of EPI include unresectable pancreatic cancer, metabolic diseases (diabetes); impaired hormonal stimulation of exocrine pancreatic secretion by Cholecystokinin (CCK); celiac or Inflammatory Bowel Disease (IBD) due to loss of intestinal brush border proteins; and gastrointestinal surgery (asynchrony between motor and secretory functions, impaired enteropancreatic feedback, and inadequate mixing of pancreatic secretions with food). Support for quitting smoking and drinking alcohol, dietary counselling, enzyme replacement therapy, and a structured follow-up of nutritional status and treatment effects are all part of the treatment for pancreatic exocrine insufficiency.

CONCLUSION

During meals, pancreatic enzyme replacement treatment is given in the form of enteric-coated minimicrospheres. The dose should be proportional to the fat content of the meal (typically lipase units per main meal), with a snack requiring half the amount. If the patient does not respond to the initial treatment, the doses can be doubled and proton inhibitors added. This article focuses on current diagnostic and therapeutic strategies for pancreatic exocrine insufficiency.

REFERENCES

1. Lerch SMM, Halangk W, Krüger B. The role of cysteine proteases in intracellular pancreatic serine protease activation. Adv Exp Med Biol 2000; 477:403-411. [PMID: 10849766].

2. Hasan A, Moscoso DI, Kastrinos F. The role of genetics in pancreatitis. Gastrointest Endosc Clin N Am 2018; 28:587-603. [PMID: 30241646].

3. Capretti G, Boggi U, Salvia R, Belli G, Coppola R, Falconi M, et al. Proteases in the evaluation of pancreatic function and pancreatic disease. Clin Chim Acta 2000; 291:201-221. [PMID: 10675724].

4. Singh VK, Haupt ME, Geller DE, Hall JA, Diez PMQ. Less common etiologies of exocrine pancreatic insufficiency. World J Gastroenterol 2017; 23:7059-7076. [PMID: 29981660].

Citation: Schaider P. The role of pancreatic proteases in the diagnosis and management of Chronic Pancreatic Insufficiency (CPI). JOP. J Pancreas. 2022;23(4):739.