Masahiko Hirota, Pancreat Disord Ther 2018, Volume 8
DOI: 10.4172/2165-7092-C1-010

3rd International Conference on
HEPATOBILIARY & PANCREATIC DISORDERS
September 17-18, 2018 | Philadelphia, USA

Masahiko Hirota

Kumamoto Regional Medical Center, Japan

Role of autophagy in the development of acute pancreatitis

Introduction: Autophagy is an intracellular bulk degradation process within cells. We previously showed that excessive autophagy was induced in the pancreas of *Psti* (pancreatic secretory trypsin inhibitor)-knockout mice (Gastroenterology 129: 696.). We also confirmed that excessive autophagy is induced in the pancreas of cerulein-induced acute pancreatitis. However, the role of autophagy in acute pancreatitis (whether autophagy activates trypsinogen to trypsin within the acinar cells or autophagy is induced in response to trypsin activation to dissolve harmful trypsin) is not well elucidated.

Objectives: The objectives are to elucidate the role of autophagy in acute pancreatitis.

Masahiko Hirota We developed and analyzed two experimental systems: 1) *Psti* and *Atg5* (autophagy-related gene 5) double-deficient mice and 2) acinar cell-specific (conditional) *Atg5* deficient mice. *Atg5* is one of the key proteins to precede the autophagic process and *Atg5* deficient mice could not induce autophagy.

Results: 1) Both intraacinar trypsin activity and acinar cell destruction were not induced in *Psti* and *Atg5* double-deficient mice. 2) Loss of autophagy caused resistance to activation of trypsinogen to trypsin within the acinar cells in conditional *Atg5* deficient mice. The severity of acute pancreatitis induced by cerulein was also greatly reduced in conditional *Atg5* deficient mice. These results indicate that autophagy is essential for trypsinogen activation to trypsin in the pancreatic acinar cells and exerts acinar cell destruction.

Conclusion: Autophagy activates trypsinogen and triggers the development of acute pancreatitis.

Biography

Masahiko Hirota has completed his PhD from Kumamoto Unversity. He is the director of Kumamoto Regional Medical Center, Japan and the clinical professor of Department of Gastroenterological Surgery, KumamoTo University Medical School, Japan. He has published more than 250 papers in reputed journals.

mhirota@krmc.or.jp

Notes: