Pancreas - Structure and Function

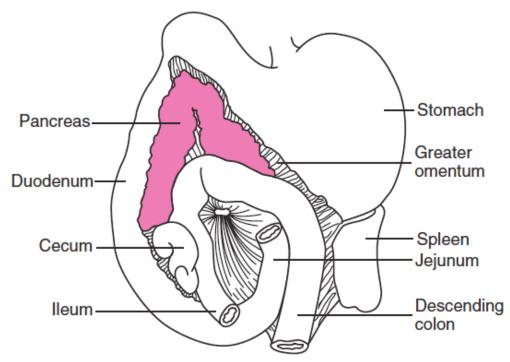
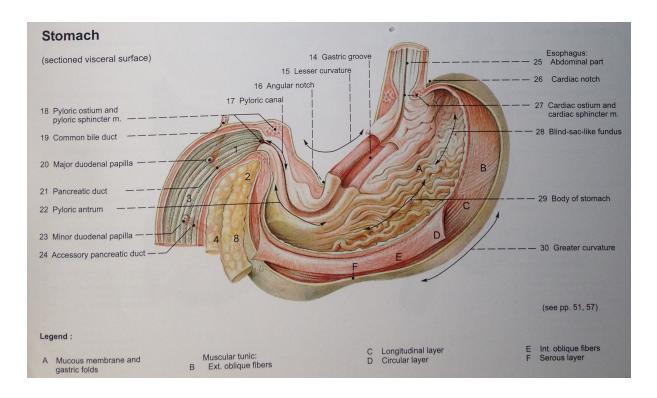
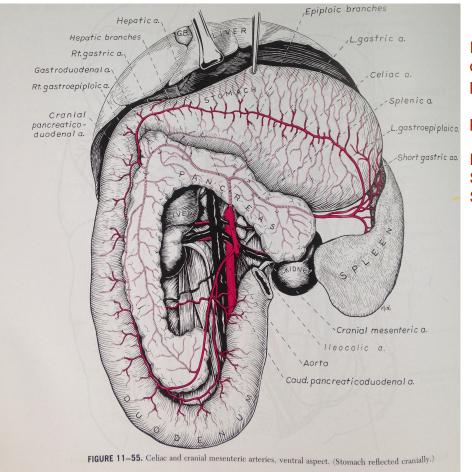


Figure 60-1 Anatomic relationship of the pancreas to other abdominal viscera. (Reprinted with permission from Johnson LR: *Gastrointestinal Physiology*, 2nd ed, Philadelphia, Elsevier, 2007.)





Right Lobe: celiac a. > cr. & cd. pancreaticoduodenal aa.

pancreaticoduodenal v.

Left Lobe: Splenic a. (pancreatic br.) Splenic v. (2 vv. converge)

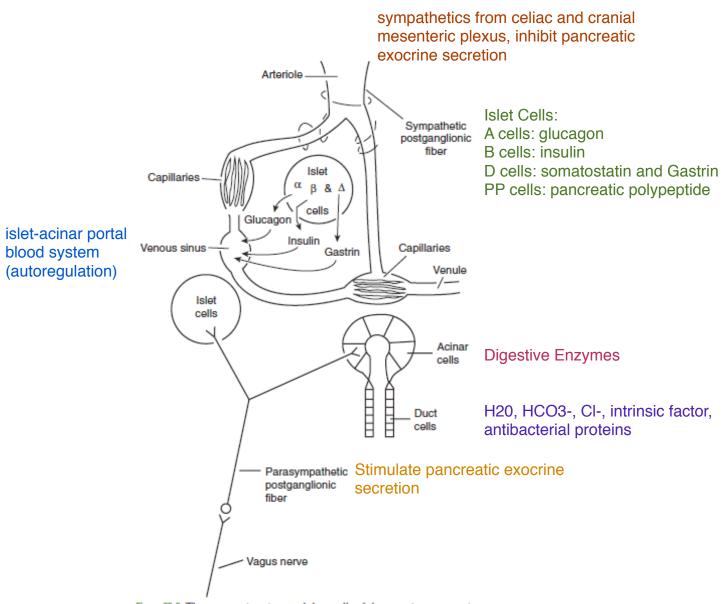
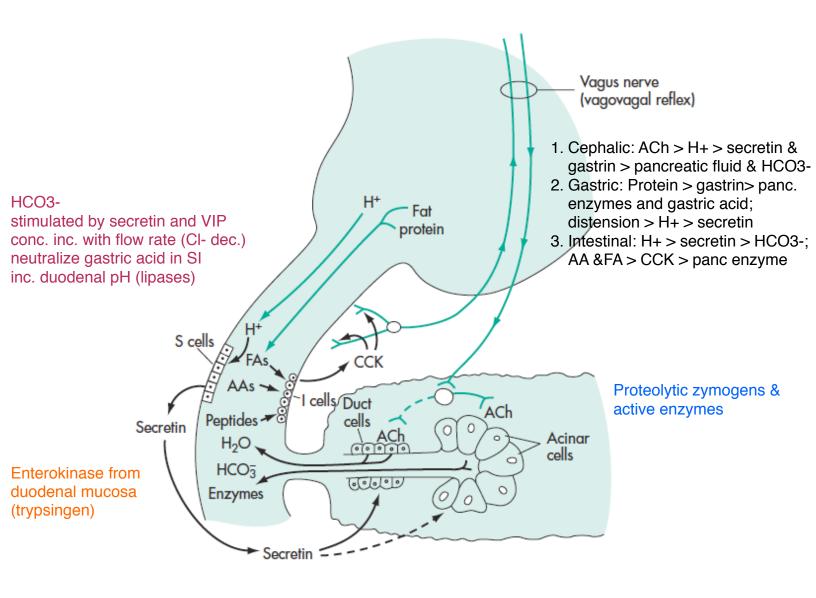


Figure 60-2 The pancreatic acinar and duct cells of the exocrine pancreatic parenchyma with their blood supply and autonomic nervous system innervation. (Reprinted with permission from Johnson LR: Gastrointestinal Physiology, 2nd ed, Philadelphia, Elsevier, 2007.)



PSTI in zymogen granules

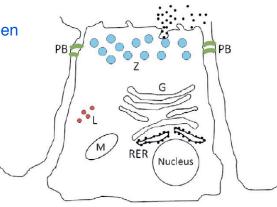


Figure 60-5 Normal pancreatic acinar cell. Zymogen granules are found in the apical region of the cell and their contents are excreted exclusively through the apical surface. Lysosomes are packaged and stored separately from zymogen granules and the paracellular barriers are intact. G, Golgi apparatus; L, lysosome; M, mitochondrion; PB, paracellular barrier; RER, rough endoplasmic reticulum; Z, zymogen granule. (Courtesy of Drs. Panagiotis Xenoulis and Joerg Steiner, Texas A & M University, College Station, Texas.)

created as precursor (RER) transported to Golgi and tagged packaged packaged with inhibitor released into ductal lumen activated in duodenum (trypsinogen)

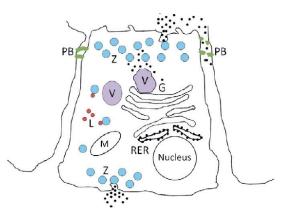


Figure 60-6 Acute pancreatitis. Secretion of the zymogen granules is redirected from the apical pole to the basolateral region of the acinar cell and into the interstitial space. Retention of zymogen granules is followed by co-localization with lysosomes and the formation of large vacuoles and premature intracellular activation of pancreatic enzymes. There is also disruption of the paracellular barrier in the pancreatic duct that allows its contents to leak into the paracellular space. G, Golgi apparatus; L, lysosome; M, mitochondrion; PB, paracellular barrier; RER, rough endoplasmic reticulum; V, vacuole; Z, zymogen granule. (Courtesy of Drs. Panagiotis Xenoulis and Joerg Steiner, Texas A & M University, College Station, Texas.)

*** ischemia, inflammation, duct obstruction***

Colocalization with lysosomal hydrolases (cathepsin B, NAG) in large vacuoles (crinophagy) >> trypsinogen activations fragile lysosomes >> release in acini

severity >> cytokines, ROS, apoptosis, altered ox/reduction

pro-/anti- inflammatory cytokines >>>>> SIRS/MODS

Questions:

- 1. What cell type (acinar/ductal) is responsible for pancreatic digestive enzyme secretion?
- 2. T/F: Transport to the Golgi complex is aberrant in pancreatitis.
- 3. What is the proper term for co-localization of zymogens with lysosomal hydrolases?
- 4. What determines progression of acute pancreatic necrosis to SIRS/MODS?
- 5. What is the primary determinant of bicarbonate secretion?

Questions:

- 1. What cell type is responsible for pancreatic digestive enzyme secretion? a. acinar cells
- 2. T/F: Transport to the Golgi complex is aberrant in pancreatitis. a. False. Synthesis and transport in tact. Co-localization contributes to disease.
- 3. Through what process does co-localization of zymogens with lysosomal hydrolases occur?

a. crinophagy

- 4. What determines progression of acute pancreatic necrosis to SIRS/MODS? a. balance of pro and anti-inflammatory cytokines
- 5. What is the primary determinant of bicarbonate secretion?
 - a. Secretin