

Normal bile secretion			
Bile Salt Metabolism and Kinetics	<ul style="list-style-type: none"> <li>• synthesized from cholesterol</li> <li>• major excretory pathway for cholesterol</li> <li>• bile salt distribution <ul style="list-style-type: none"> <li>-40% cholate</li> <li>-40% chenodeoxycholate</li> <li>-20% deoxycholate</li> <li>-13% lithocholate</li> </ul> </li> </ul>	<p><b>1° bile salts: cholate &amp; chenodeoxycholate</b></p> <ul style="list-style-type: none"> <li>-formed directly from cholesterol</li> <li>-synthesis regulated thru feedback inhibition <ul style="list-style-type: none"> <li>-bile salts extracted from portal venous blood during enterohepatic circ. <ul style="list-style-type: none"> <li>→ ↓ <b>7α-hydroxylase</b> (rate limiting for cholesterol → bile)</li> <li>→ ↓ <b>HMG-CoA reductase</b> (rate limiting for cholesterol synthesis)</li> </ul> </li> </ul> </li> <li>• w/in hepatocyte bile salt is conjugated (strong bond—only bacteria can cleave it) → ↓pKa of bile acid → ↑solubility in duodenal-jejunal pH (6.5)</li> <li>• bile salts efficiently reabsorbed by Na<sup>+</sup>-coupled transport in ileum</li> <li>• after one enterhepatic circulation, 10-20% of bile salts no longer conjugated <ul style="list-style-type: none"> <li>→ re-conjugation after uptake → re-excretion</li> </ul> </li> </ul> <p><b>2° bile salts: deoxycholate &amp; lithocholate</b></p> <ul style="list-style-type: none"> <li>-anaerobic bacteria attack bile salts</li> <li>• significant amt. of deoxycholate reabsorbed</li> </ul>	<ul style="list-style-type: none"> <li>• FXR nuclear receptor <ul style="list-style-type: none"> <li>-activated by bile salts at physiologic conc.</li> <li>-acts w/RXR → ↑transcription of promoters in bile salt metabolism</li> </ul> </li> <li>-activated FXR <ul style="list-style-type: none"> <li>→ ↑bile salt transporters, ↑ileal bile acid binding protein</li> <li>→ ↓7α-hydroxylase</li> </ul> </li> <li>• LXR coordinates cholesterol transport to the liver <ul style="list-style-type: none"> <li>-activated LXR → ↑phospholipid/cholesterol transporter in periph. cells → ↑free cholesterol efflux → ↑HDL → ↑hepatic cholesterol uptake</li> </ul> </li> </ul>
Physiochemical properties	<ul style="list-style-type: none"> <li>• unassociated molecules in dilute solution</li> <li>• form micelles at high concentration <ul style="list-style-type: none"> <li>-must exceed critical micellar concentration (2-3mM)</li> <li>-normal conc. 50-150mM</li> </ul> </li> <li>• ileal disease/resection → ↓bile reabsorption → steatorrhea unless bile salt synthesis compensates (max. production is ~3g/day)</li> </ul>		
Bile Secretion	<ul style="list-style-type: none"> <li>• active transport in the canaliculus <ul style="list-style-type: none"> <li>-each bile molecule obligates a volume of water and electrolytes</li> </ul> </li> <li>• <b>secretin</b> → release of an alkaline solution</li> <li>• cholesterol and lecithin secretion occur at the level of the hepatocyte</li> </ul>		
Gallbladder fuction	<ul style="list-style-type: none"> <li>• mucosa <ul style="list-style-type: none"> <li>-can concentrate bile salts <ul style="list-style-type: none"> <li>-extracts electrolytes and water</li> </ul> </li> <li>-active transport of Na<sup>+</sup>, Cl<sup>-</sup>, HCO<sub>3</sub><sup>-</sup> obligates a volume of water → ↑lipid conc. w/o change in osmolality</li> </ul> </li> <li>• bile secretion is continuous <ul style="list-style-type: none"> <li>-in fasting state bile is diverted to the gallbladder</li> <li>-feeding → ↑fatty acids and ↑essential A.A.s → CCK release from small intestine → sphinchter of Oddi relaxation and gallbladder contraction</li> </ul> </li> </ul>		

Disorders of bile secretion	<ul style="list-style-type: none"> <li>• ↓bile secretion/cholestasis → ↑plasma conc. of normal bile constituents</li> <li>• hepatitis and alcoholic cirrhosis → greater defects in bilirubin transport than those of bile salts</li> <li>• chronic cholangitides (biliary cirrhosis, chronic drug rxns., sclerosing cholangitis) → greater interference of bile salt secretion than pigment excretion</li> <li>• common bile duct obstruction → failure of bile secretion</li> </ul>		
Gallstone Formation	<ul style="list-style-type: none"> <li>• most prevalent disease of biliary system</li> <li>• 75% are cholesterol stones</li> <li>-pure cholesterol or mixed</li> <li>• remainder are pigment stones</li> <li>-mostly Ca<sup>2+</sup> bilirubinate</li> </ul>	<ul style="list-style-type: none"> <li>• form when cholesterol or bilirubin conc. exceeds solubilizing capacity of bile</li> <li>• cholesterol solubility</li> <li>-only soluble in solutions at least 40% bile salts</li> <li>-restricted range of solubility</li> <li>• <b>virtually all pts. w/ gallstones have bile saturated w/ cholesterol</b></li> <li>-not all pts w/persistently saturate bile will develop gallstones</li> <li>• <b>nucleation</b> in important factor</li> <li>-lithogenic bile rapidly nucleates</li> <li>-normal bile contains promoters (glycoproteins) and inhibitors (apolipoproteins) of nucleation</li> <li>-imbalance → crystal precipitation</li> <li>• <b>stasis of gallbladder</b> bile promotes gallstone formation and occurs in: <ul style="list-style-type: none"> <li>-prolonged parenteral nutrition</li> <li>-pregnancy</li> </ul> </li> <li>• Ca<sup>2+</sup> salts interact w/promoters and may be nidus of stones</li> <li>• <b>only defects in bile acid or cholesterol secretion</b> have been identified as <b>causes</b></li> <li>-NOT defects in lecithin</li> <li>• many pts. have diminished bile salt pool → suggests defective feedback reg. of 7α-hydroxylase</li> <li>• estrogens (2° to ↑hepatic LDL receptors) → ↑cholesterol secretion into bile</li> <li>• <b>obese pts.</b> <ul style="list-style-type: none"> <li>-over-secrete cholesterol into bile</li> <li>-during weight-loss cholesterol saturation in bile ↑ further</li> <li>-obese pts. who lose weight by starvation diets have a very high incidence of gallstone formation</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• pharmacologic <ul style="list-style-type: none"> <li>-oral feedings of chenodeoxycholic acid</li> <li>-prolonged trials showed efficacy</li> <li>-mechanism may be ↓HMG CoA red.</li> </ul> </li> <li>-ursodeoxycholic acid</li> <li>• cholecystectomy</li> <li>-Tx of choice</li> <li>-low rate of stone formation after surgery</li> <li>-mechanism may be more rapid and continuous circulation of bile salt pool</li> </ul>

Pancreatitis	•	•	•	•
Acute	<ul style="list-style-type: none"> <li>• 45% due to gallstones</li> <li>• 35% due to alcohol</li> <li>• 10% are idiopathic</li> <li>-2/3 are this are due to “biliary sludge” (microlithiasis)</li> <li>• high incidence in AIDS pts.</li> <li>• rare in children</li> <li>-trauma most common cause</li> <li>-Cystic fibrosis also a cause</li> </ul>	<ul style="list-style-type: none"> <li>• ↑pancreatic intraductal pressure is sufficient for development</li> <li>• earliest abnormality is coalescence of zymogen granules w/ lysosomes</li> <li>-<b>trypsinogen</b> → <b>trypsin</b> → activation of pancreatic proteases, most importantly <b>phospholipase A2</b> and <b>elastase</b> → digestion of acinar cells and surrounding tissues and blood vessels</li> <li>-release of <b>lipase</b> → fat saponification → free fatty acids → bind <math>Ca^{2+}</math> → hypocalcemia</li> <li>-<b>phospholipase A2</b> also degrades surfactant</li> <li>-<b>trypsin</b> → complement activation and ↑vascular permeability → vascular collapse</li> </ul>	<ul style="list-style-type: none"> <li>• onset sudden</li> <li>• course unpredictable</li> <li>• <b>Early complications</b> (1-2wks)</li> <li>-<b>pancreatic necrosis</b> <ul style="list-style-type: none"> <li>- &gt;50% necrosis → poor prognosis</li> <li>-necrotic tissue becomes infected w/gram negative bacteria from intestine</li> </ul> </li> <li>-<b>shock</b></li> <li>-<b>hypoxia</b> <ul style="list-style-type: none"> <li>-systemically released toxic factors → ↑capillary perm. in lung</li> <li>-if severe → ARDS</li> </ul> </li> <li>-<b>hypocalcemia</b></li> <li>-<b>hyperglycemia</b> <ul style="list-style-type: none"> <li>-glucagon release, ↓insulin release</li> </ul> </li> <li>-<b>acute renal failure</b> <ul style="list-style-type: none"> <li>-ATN 2° to hypotension</li> </ul> </li> <li>-<b>jaundice</b> <ul style="list-style-type: none"> <li>-when inflammation in pancreas head</li> </ul> </li> <li>• <b>Late complications</b> (&gt;2wks)</li> <li>-<b>pancreatic pseudocysts</b> <ul style="list-style-type: none"> <li>-fluid released into surrounding area</li> <li>-can occur anywhere in abdominal cavity</li> </ul> </li> <li>-<b>pancreatic ascites</b> <ul style="list-style-type: none"> <li>-if disrupted duct communicates w/peritoneal cavity</li> </ul> </li> <li>-<b>pancreatic abscesses</b> <ul style="list-style-type: none"> <li>-necrotic tissue 2° infected → walled-off pus collections</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• <b>Diagnosis</b></li> <li>-<u>clinical presentation</u> <ul style="list-style-type: none"> <li>-excruciating epigastric pain radiating to the back</li> <li>-vomiting</li> <li>-low grade fever</li> <li>-in severe cases: shock, resp. failure, psychosis, delirium, coma</li> </ul> </li> <li>-<u>lab tests</u> <ul style="list-style-type: none"> <li>-↑<b>serum amylase</b>, w/this clinical setting, <b>is the hallmark of pancreatitis</b></li> <li>-if pt. delays going to hospital ↑amylase may be missed</li> <li>-in this case use serum lipase</li> <li>-<b>magnitude of ↑amylase does NOT predict severity</b></li> </ul> </li> <li>-<u>images studies</u> <ul style="list-style-type: none"> <li>-<b>abdominal CT</b> is best</li> <li>-<b>abdominal sonogram</b> poor for assessing pancreas, good for gallstones</li> </ul> </li> <li>• <b>Treatment</b></li> <li>-mainly supportive</li> <li>-stop oral intake</li> <li>-IV hydration</li> <li>-analgesics</li> <li>-nasogastric suctioning for vomiting</li> </ul>
Chronic	<ul style="list-style-type: none"> <li>• gallstone virtually <b>never</b> a cause</li> <li>• 70-80% due to <b>alcoholism</b></li> <li>• tropical (nutritional)</li> <li>-esp. S. India, Indonesia, Africa</li> <li>• Hereditary</li> <li>-familial pancreatitis, hyperlipidemia, cystic fibrosis</li> </ul>	<ul style="list-style-type: none"> <li>• Alcohol</li> <li>-<b>earliest detectable change in small ductules</b></li> <li>-alcohol → ↑digestive enzyme secretion &amp; potentiates CCK effects on enzyme secretion → protein-rich fluid accumulation → <b>local small duct obstruction</b></li> <li>-↓conc. of <b>lithostatin</b> (normally prevents <math>Ca^{2+}</math> deposition)</li> <li>-calcified <b>stones</b>, if found, are a <b>late</b> complication</li> <li>• Tropical (Nutritional)</li> <li>-unclear whether nutrient-deficient diet or tropical rootstock cassava → impaired free radical scavengers</li> <li>-<b>diffuse calcification invariably seen on X-ray</b></li> </ul>	<ul style="list-style-type: none"> <li>• ↑intraductal P and/or neural inflammation → <b>abdominal pain</b></li> <li>-occurs immediately after eating</li> <li>-weight loss due more to avoidance of pain than malabsorption</li> <li>• does not occur until &gt;90% of pancreas destroyed</li> <li>-<b>malabsorption</b></li> <li>-<b>diabetes</b></li> <li>• diffuse pancreatic calcifications found on 30-40% of X-rays</li> <li>-diagnostic</li> <li>• complications</li> <li>-pseudocysts</li> <li>-pancreatic ascites</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Treatment</b></li> <li>-<b>analgesics</b> <ul style="list-style-type: none"> <li>-often requires narcotics</li> <li>-<b>pancreaticojejunostomy</b> provides pain relief in 80% of cases, if duct is dilated</li> <li>-<b>partial pancreatectomy</b> relief in 50% of cases</li> </ul> </li> <li>-<b>pancreatic enzyme replacement</b></li> </ul>
Pancreatic Carcinoma	<ul style="list-style-type: none"> <li>• incidence is rising</li> <li>• by the time it causes symptoms, it is usually incurable</li> </ul>	<ul style="list-style-type: none"> <li>• 2 genes mutations identified</li> <li>- <b>K-ras</b> mutated in 75%</li> <li>-<b>DPC-4</b> deleted or mutated in 50%</li> </ul>	<ul style="list-style-type: none"> <li>• if head of the pancreas → bile duct obstruction → painless jaundice</li> <li>• can also cause diabetes</li> </ul>	<ul style="list-style-type: none"> <li>• 5-year survival &lt; 1%</li> <li>• improved survival w/tumors &lt;2cm w/radical pancreaticoduodenectomy</li> <li>• CT unreliable detecting tumors &lt;2cm</li> </ul>

Gall bladder & extra-hepatic ducts	Etiology	Pathology	Clinical/Labs	Treatment
	•		<ul style="list-style-type: none"> <li>• bacteria cannot be cultured from normal biliary tree</li> <li>-probably present in small numbers</li> <li>-enter via portal vein</li> <li>-cleared by liver via bile ducts</li> </ul>	•
Acute Cholecystitis	<ul style="list-style-type: none"> <li>• usually follows cystic duct obstruction by a stone → gall bladder distension and disrupted mucosa → conc. bile seeps into gallbladder wal → chemical irritation</li> <li>• 95% of cases stone blocking cystic duct → acute inflammatory changes</li> <li>• less common causes</li> <li>-trauma, surgery in the area, severe dehydration, polyarteritis nodosa</li> </ul>	<ul style="list-style-type: none"> <li>• grossly</li> <li>-erythema, may involve only mucosa, or extend to serosa</li> <li>-edema → thick and boggy wall</li> <li>-variable inflammation &amp; necrosis</li> </ul>	<ul style="list-style-type: none"> <li>• bacteria present</li> <li>-in wall in &gt;90% of cases</li> <li>-in lumen in &gt;75% of cases</li> <li>-typical bacteria of large bowel</li> <li>• -E.coli, Enterobacter, Strep. fecalis</li> </ul>	<ul style="list-style-type: none"> <li>• majority of pts. successfully treated w/bed rest, NPO, nasogastric suction, atropine (anti-spasmodics), narcotics, and antibiotics</li> <li>• 20% do not respond</li> <li>-pain worsens, fever &amp; leukocytosis persist</li> <li>-cholecystectomy to prevent complications</li> <li>-abscesses, gangrene, perforation, peritonitis</li> <li>-drainage if gallbladder adhesions are present</li> </ul>
Chronic Cholecystitis	<ul style="list-style-type: none"> <li>• present in most gallbladders resected for symptomatic gallstones</li> <li>• most had previous attacks of acute cholecystitis which subsided → variable degrees of damage</li> </ul>	<ul style="list-style-type: none"> <li>• wall shows variable thickening and fibrosis</li> <li>• microscopy</li> <li>-Rokitansky-Aschoff sinuses</li> <li>-hypertrophied muscularis</li> <li>-fibrosis</li> <li>-chronic inflammatory cell infiltrates</li> </ul>	•	•
Hydrops	<ul style="list-style-type: none"> <li>• persistent cystic duct obstruction w/o infection</li> <li>-presumably no bacteria present in lumen</li> </ul>	<ul style="list-style-type: none"> <li>• over months, gallbladder distends,</li> <li>• bile resorbed thru mucosa → watery fluid</li> </ul>	<ul style="list-style-type: none"> <li>• gallbladder palpable</li> <li>• no pain or tenderness</li> </ul>	•
Mucocele	• variant of hydrops	• gallbladder lumen filled w/mucus	•	•
Carcinoma	<ul style="list-style-type: none"> <li>• infrequent</li> <li>• <b>90-95% in gallbladders w/stones</b></li> <li>• most pts/ elderly w/long symptomatic h/o gallbladder disease</li> </ul>	<ul style="list-style-type: none"> <li>• diffuse thickening of wall</li> <li>-may simulate wall in chronic cholecystitis (i.e. thickened &amp; inflamed)</li> <li>• tumor ID only from sections</li> <li>• tumors are <b>adenocarcinomas</b></li> <li>-variable differentiation</li> </ul>	<ul style="list-style-type: none"> <li>• signs &amp; symptoms mimic cholelithiasis and cholecystitis</li> </ul>	<ul style="list-style-type: none"> <li>• carcinoma usually unexpected finding during surgery</li> <li>• most cases tumor not respectable b/c it has already invaded adjacent structures</li> <li>• <b>poor prognosis</b></li> <li>- &lt; 5% alive at 5yrs.</li> </ul>
Adenomyoma	<ul style="list-style-type: none"> <li>• most common benign tumor of gallbladder</li> <li>• more likely represent malformations</li> </ul>	<ul style="list-style-type: none"> <li>• firm, rubbery, well circumscribed nodules in fundic end of gallbladder</li> </ul>	<ul style="list-style-type: none"> <li>• usually asymptomatic</li> <li>• found incidentally on gallbladder resection</li> </ul>	•
Cholesterosis	• freq. in stone-containing gallbladder	<ul style="list-style-type: none"> <li>• thin yellow streaks in mucosa</li> <li>• foamy MΦs containing lipid in lamina propria</li> </ul>	• usually incidental finding	•
Choledocholithiasis	<ul style="list-style-type: none"> <li>• stones in common bile duct in 5-25% of pts. w/cholelithiasis</li> <li>• small stones more likely than large stones to pass into common bile ducts</li> <li>• most common extraheptic cause of obstructive jaundice</li> </ul>	• infection may extend to liver	<ul style="list-style-type: none"> <li>• no pain 20-25% of cases</li> <li>• jaundice not present &gt; 25% of cases</li> <li>• may also lead to ascending cholangitis</li> <li>-w/fever, chills, &amp; leukocytosis</li> </ul>	<ul style="list-style-type: none"> <li>• Endoscopic retrograde cholangiopancreatography (ERCP) is procedure of choice for removing stones from common duct or ampulla of Vater</li> </ul>
Gallstone Ileus	<ul style="list-style-type: none"> <li>• uncommon condition</li> <li>• one large stone gradually erodes thru gall bladder wall → gallbladder adhesions to duodenum → stone erodes into gut → propelled thru gut → excreted w/feces</li> </ul>			

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Chronic Pancreatitis	<ul style="list-style-type: none"> <li>• gallstone virtually <b>never</b> a cause</li> <li>• 70-80% due to <b>alcoholism</b></li> <li>• tropical (nutritional)</li> <li>-esp. S. India, Indonesia, Africa</li> <li>• Hereditary</li> <li>-familial pancreatitis (5-10%)</li> <li>-has acute recurrent episodes</li> <li>-cystic fibrosis: pancreatic insufficiency, but rarely pain</li> </ul>	<ul style="list-style-type: none"> <li>• <u>Alcohol</u></li> <li>-<b>earliest detectable change in small ductules</b></li> <li>-alcohol → ↑digestive enzyme secretion &amp; potentiates CCK effects on enzyme secretion → protein-rich fluid accumulation → <b>local small duct obstruction</b></li> <li>-↓conc. of <b>lithostatin</b> (normally prevents <math>Ca^{2+}</math> deposition)</li> <li>-calcified <b>stones</b>, if found, are a <b>late</b> complication</li> <li>• <u>Tropical (Nutritional)</u></li> <li>-unclear whether nutrient-deficient diet or tropical rootstock cassava → impaired free radical scavengers</li> <li>-<b>diffuse calcification invariably</b> seen on X-ray</li> </ul>	<ul style="list-style-type: none"> <li>• ↑intraductal P and/or neural inflammation → <b>abdominal pain</b></li> <li>-occurs immediately after eating</li> <li>-weight loss due more to avoidance of pain than malabsorption</li> <li>• does not occur until &gt;90% of pancreas destroyed</li> <li>-<b>malabsorption</b></li> <li>-<b>diabetes</b></li> <li>• diffuse pancreatic calcifications found on 30-40% of X-rays</li> <li>-diagnostic</li> <li>• complications</li> <li>-pseudocysts</li> <li>-pancreatic ascites</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Treatment</b></li> <li>-<b>analgesics</b></li> <li>-often requires narcotics</li> <li>-<b>pancreaticojejunostomy</b> provides pain relief in 80% of cases, if duct is dilated</li> <li>-<b>partial pancreateomy</b> relief in 50% of cases</li> <li>-<b>pancreatic enzyme replacement</b></li> </ul>
Chronic Relapsing Pancreatitis	<ul style="list-style-type: none"> <li>• found in chronic alcoholics w/ h/o multiple attacks of abdominal pain</li> <li>• pts. develop chronic pancreatitis &amp; pancreato-lithiasis, assoc. w/severe pain→ narcotic addiction</li> </ul>			
Pancreatic Pseudocyst	<ul style="list-style-type: none"> <li>• complication of acute pancreatitis or trauma to the pancreas</li> </ul>	<ul style="list-style-type: none"> <li>• unilocular fluid-filled cyst</li> <li>-forms in areas of extensive necrosis</li> <li>• more often in lesser sac between pancreas and stomach, colon or liver</li> </ul>	<ul style="list-style-type: none"> <li>• do not regress spontaneously</li> </ul>	<ul style="list-style-type: none"> <li>• no longer resected</li> <li>• rather, opened into adjacent structure to drain</li> </ul>

Pancreatic Carcinoma	<ul style="list-style-type: none"> <li>incidence is rising</li> <li>-5<sup>th</sup> most common cancer</li> <li>by the time it causes symptoms, it is usually incurable</li> </ul>	<ul style="list-style-type: none"> <li>2 genes mutations identified</li> <li>- <b>K-ras</b> mutated in 75%</li> <li>- <b>DPC-4</b> deleted or mutated in 50%</li> <li>70% develop in head of pancreas</li> <li><b>adenocarcinoma</b> most common</li> <li>- &gt;95% of pancreatic neoplasms</li> <li>-originate w/in pancreatic ducts</li> <li>-invasion → fibroblastic desmoplastic stromal reaction → mass which is <b>hard on palpation</b></li> <li>-infiltrate adjacent pancreas → duct obstruction → atrophy and fibrosis of adjacent pancreatic tissue</li> <li>-may have perineural invasion → pain</li> <li>-metastases often <ul style="list-style-type: none"> <li>-in LNs around pancreas &amp; abdominal organs</li> <li>-in liver and peritoneum</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>if head of the pancreas → bile duct obstruction → <b>painless jaundice</b></li> <li>-50% have obstructive jaundice</li> <li>persistent, unexplained <b>weight loss</b></li> <li>50% have abdominal pain radiating to the back</li> <li>can also cause diabetes</li> <li>metastatic deposits <ul style="list-style-type: none"> <li>→ ascites and/or enlarged nodular liver</li> </ul> </li> <li>hypercoagulability → migratory thrombophlebitis</li> </ul>	<ul style="list-style-type: none"> <li>poor prognosis</li> <li>-5-year survival 2%</li> <li>improved survival w/tumors &lt;2cm w/radical pancreaticoduodenectomy</li> <li>- &lt; 5% have respectable tumor</li> <li>CT unreliable detecting tumors &lt;2cm</li> <li>90% have unresectable tumor at diagnosis</li> <li>-50% of these cases also show metastasis</li> <li>Whipple's resection for respectable carcinomas of the head</li> </ul>
Cystadenomas	<ul style="list-style-type: none"> <li>rare benign tumors</li> </ul>	<ul style="list-style-type: none"> <li>ductal origin</li> <li>localized masses of multiple cystic structures</li> <li>-lined by benign ductal epithelium</li> <li>2 types: <ul style="list-style-type: none"> <li>-<b>mucinous</b> cystadenomas lined w/mucin producing cells</li> <li>-<b>adenocarcinomas may arise w/in these lesions</b></li> <li>-<b>serous</b> cystadenomas lined w/benign epithelial cells containing glycogen</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li></li> </ul>	<ul style="list-style-type: none"> <li></li> </ul>
Ectopic Pancreas	<ul style="list-style-type: none"> <li>ectopic islands of ductal and acinar tissue</li> <li>islets cells usually not present</li> </ul>	<ul style="list-style-type: none"> <li>may be found in stomach, duodenum</li> <li>-less often in small bowel</li> <li>freq. assoc. w/hypertrophied muscularis propria</li> <li>-produces nodule in gastric or small bowel wall</li> <li>-<b>simulates tumor on palpation</b></li> </ul>	<ul style="list-style-type: none"> <li></li> </ul>	<ul style="list-style-type: none"> <li></li> </ul>