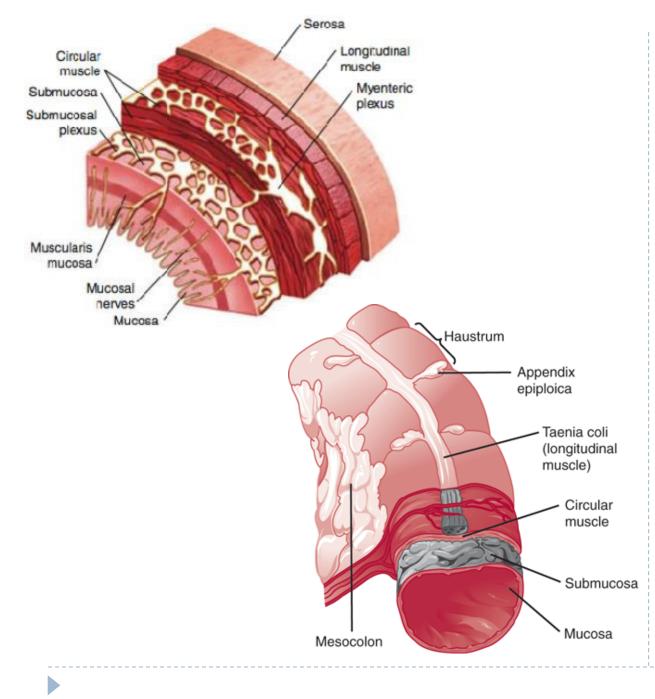


DESIGNED BY: TAMER ALTAMIMI "SMILE"



Sigmoid Diverticulitis

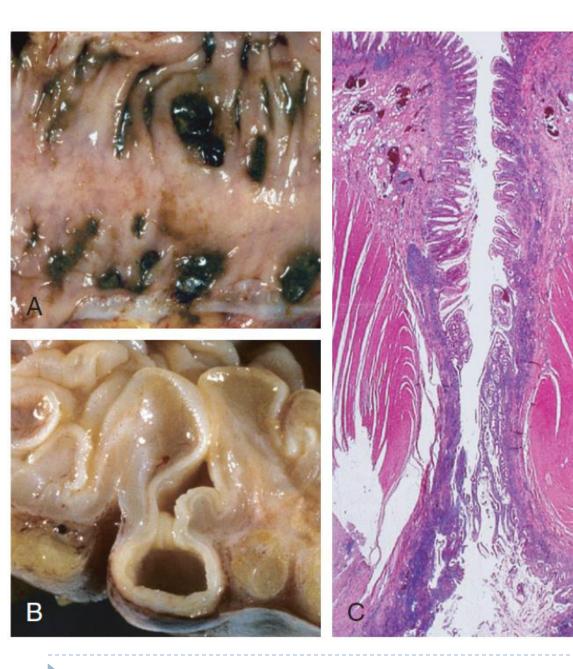
Elevated intraluminal pressure

Unique structure of the colonic muscularis propria (taeniae coli)

More common in elderly especially in western society (dietary fiber)

Diverticula mostly asymptomatic

Diverticulitis usually resolves spontaneously or after ABx



Sigmoid Diverticulitis

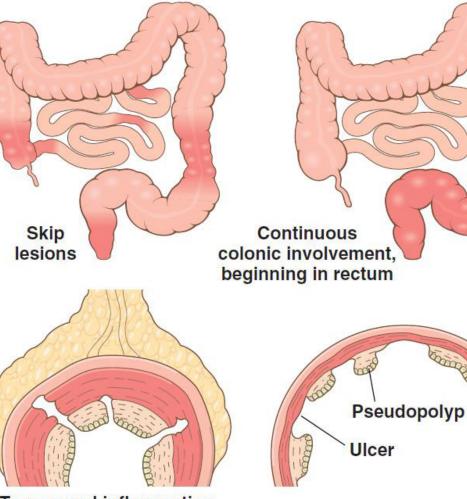
Regular distribution

Most common in the sigmoid colon

Mucosa and submucosa outpouching

- Flattened or atrophic mucosa
- Compressed submucosa
- Attenuated muscularis propria (often absent)

CROHN DISEASE



Transmural inflammation Ulcerations Fissures

mucosa and submucosa

ULCERATIVE COLITIS

Inflammatory Bowel Disease

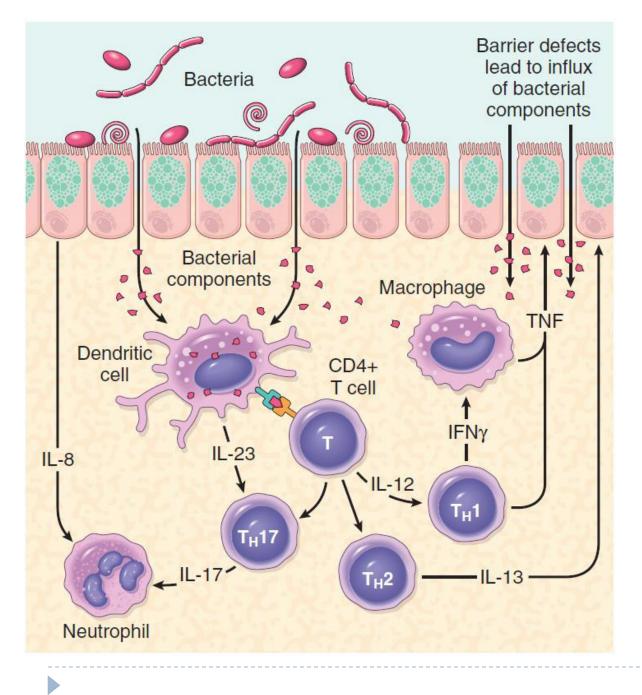
Chronic inappropriate mucosal immune activation

 \bigcirc predisposition (young)

"Hygeine hypothesis"

Increased malignant potential (Colon involvement)

Malabsorption more with Crohn



Inflammatory Bowel Disease

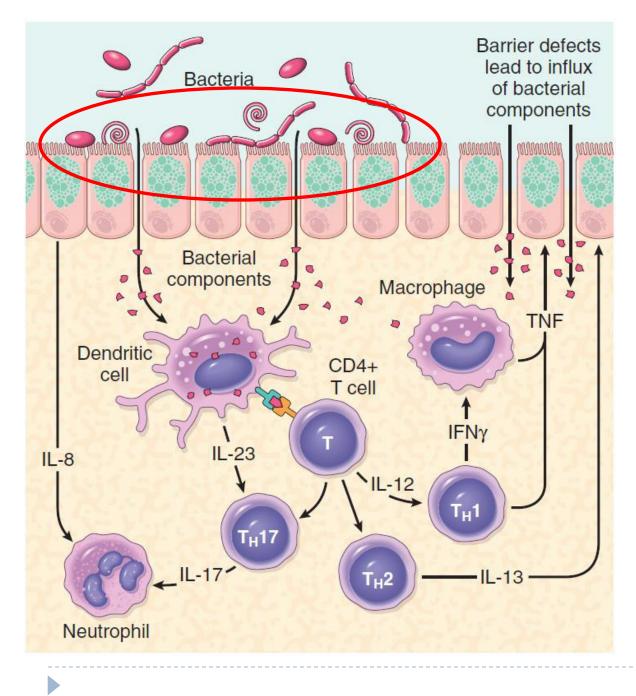
Pathogenesis

Aberrant host interactions with intestinal microbiota

Intestinal epithelial dysfunction

Aberrant mucosal immune responses

Tx: Immune suppression modulation



Inflammatory Bowel Disease

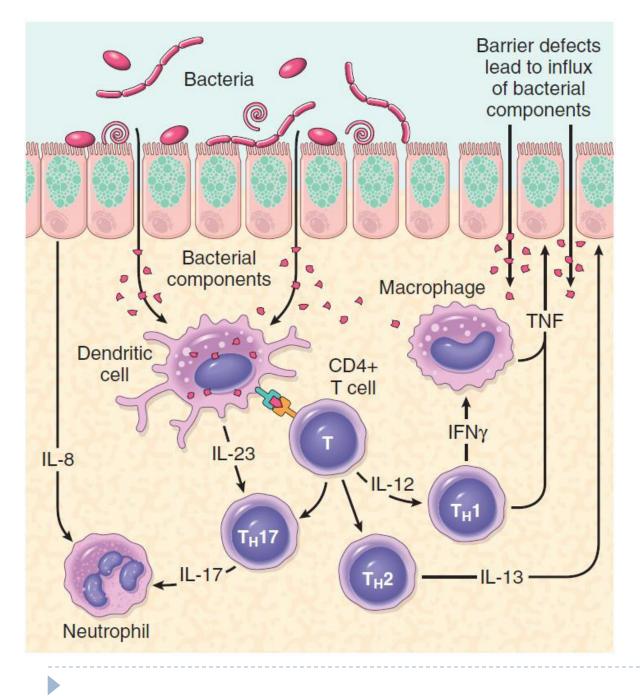
Genetics

Increased risk with an affected family member

Concordence rates higher in CD compared to UC

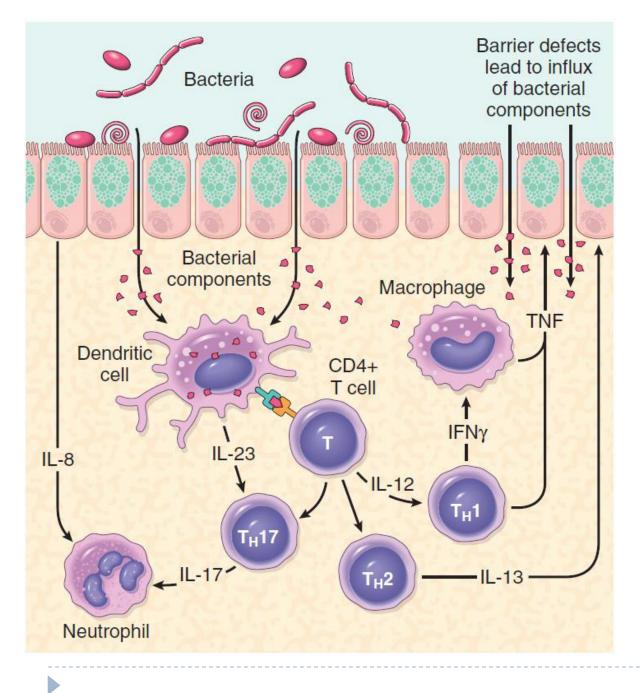
NOD2 binds to intracellular bacterial cell wall products (less effective polymorphism in CD)

ATG16L1 & IRGM (CD)



Inflammatory Bowel Disease **Mucosal** immune responses Development of T_H1 (CD) & T_H17 IL-23R polymorphisims protective (CD & UC) T_H2 development (UC) with increased IL-13 Polymorphisms of *IL-10* &

IL-10R (UC)

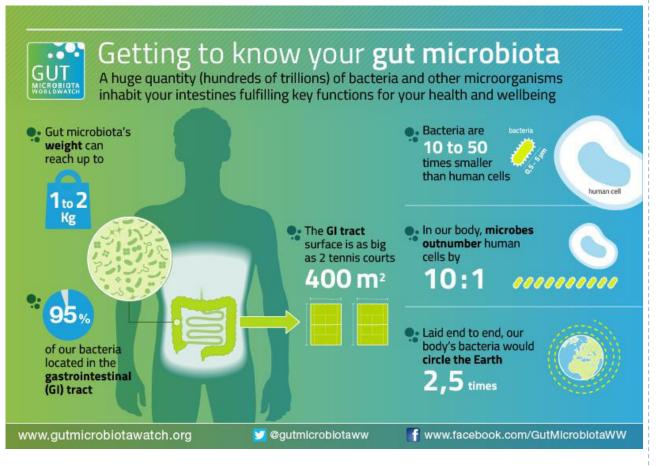


Inflammatory Bowel Disease

Epithelial defects

Defects in intestinal epithelial tight junction (CD)

Abnormal paneth cell granules affect composition of the luminal microbiota (CD)



Inflammatory Bowel Disease

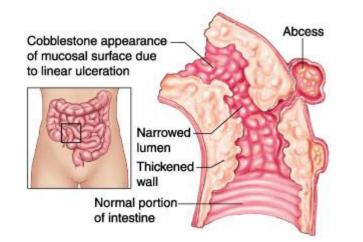
Microbiota

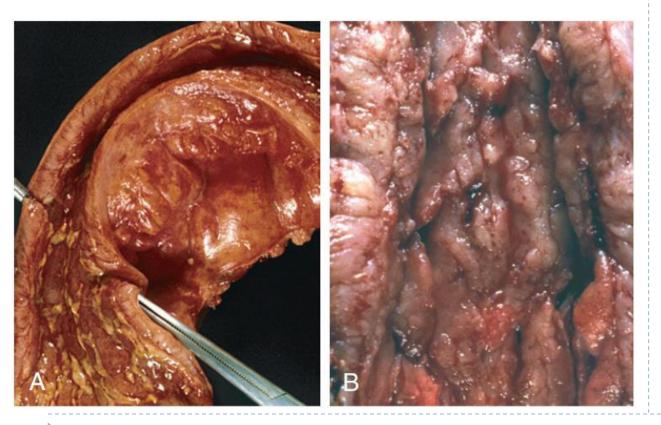
10¹² organisms/mL of fecal material in the colon (50%)

We're only 10% human!

Significant variation

Metronidazole used in maintaining remission (CD)





Crohn Disease

Most common sites at presentation are the terminal ileum, ileocecal valve, and cecum

Remember skip lesions

Strictures are common (A)

Cobblestone appearance with thickened wall (B)

Fissures - perforation - fistula

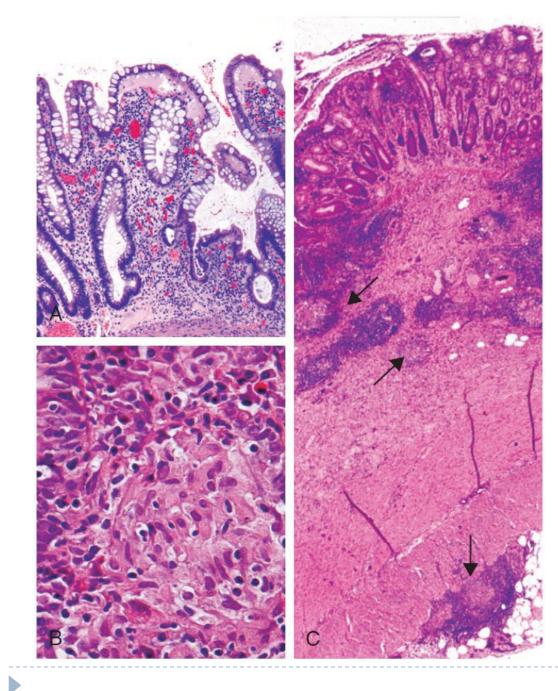


Crohn Disease

Wall thickening from

- Transmural edema
- Inflammation
- Submucosal fibrosis
- Hypertrophy of the muscularis propria

With extensive transmural disease, creeping fat (C)



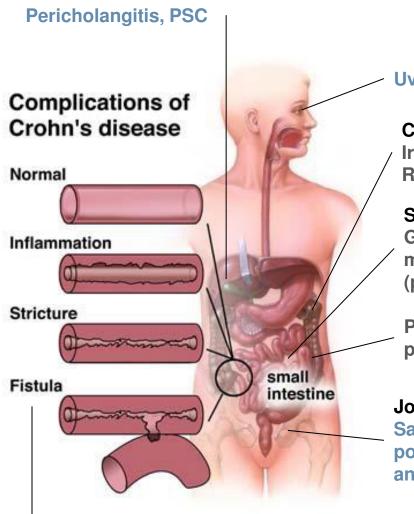
Crohn Disease

Abundant neutrophils \rightarrow crypt abscesses

Cycles of destruction & regeneration lead to abnormal crypt shapes/branching (A)

Noncaseating granulomas are a hallmark of CD (B & C)

The absence of granulomas does not exclude CD as a Dx



May also involve the urinary bladder, vagina, and abdominal or perianal skin

Uveitis Colon Iron deficiency anemia Risk of adenocarcinoma Small intestine Generalized nutrient

malabsorption (protein, B₁₂)

Perforations and peritoneal abscesses

Joints

Sacroiliitis, migratory polyarthritis, ankylosing spondylitis

Erythema nodosum, and clubbing of the fingertips

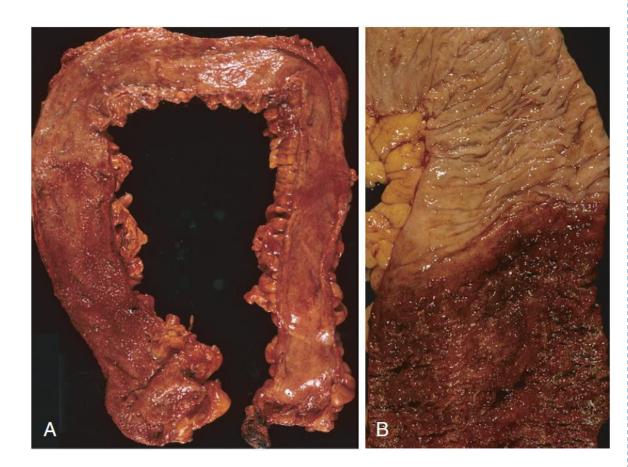
Crohn Disease

Clinical Features

Intermittent attacks of relatively mild diarrhea, fever, and abdominal pain

Right lower quadrant pain, fever, and bloody diarrhea (DDx AA, perforation)

Extraintestinal manifestations may develop before intestinal disease is recognized



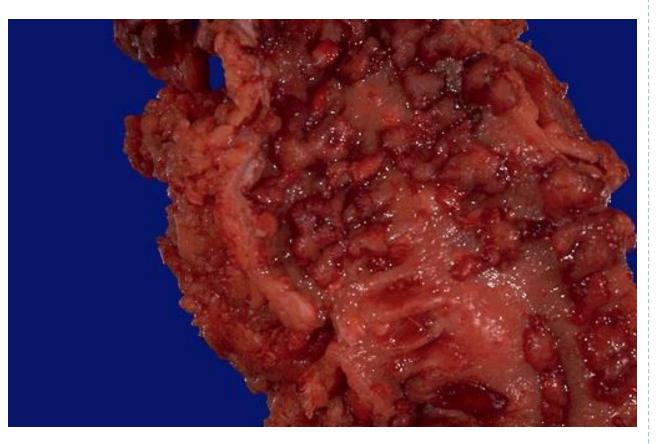
Typically limited to the colon and rectum

No skip lesions

Pancolitis (A) if severe can be associated with backwash ileitis

Abrupt transition from red and granular-appearing to normal mucosa(B)

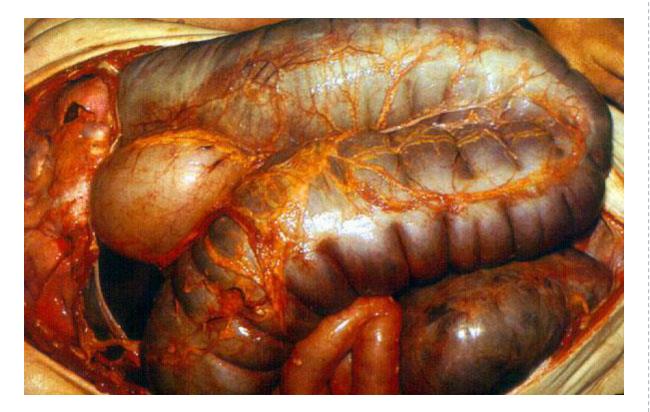
Broad-based ulcers



Pseudopolyps (islands of regenerating mucosa)

In chronic disease you may find mucosal atrophy and a flat, smooth mucosal surface lacking normal folds

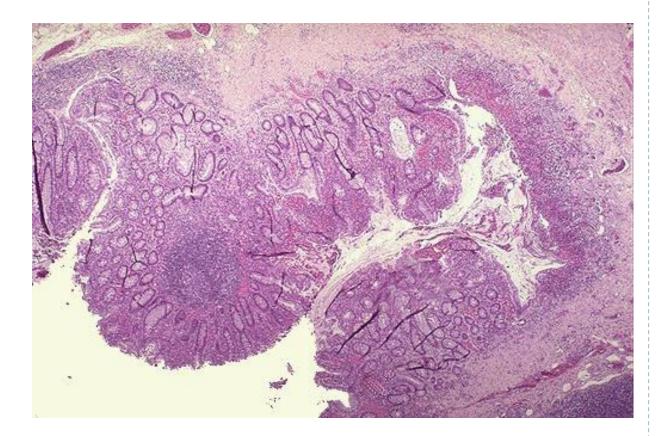
Unlike CD thickening is absent, the serosal surface is normal, and strictures do not occur



Toxic megacolon

(inflammation can damage the muscularis propria and disturb neuromuscular function)

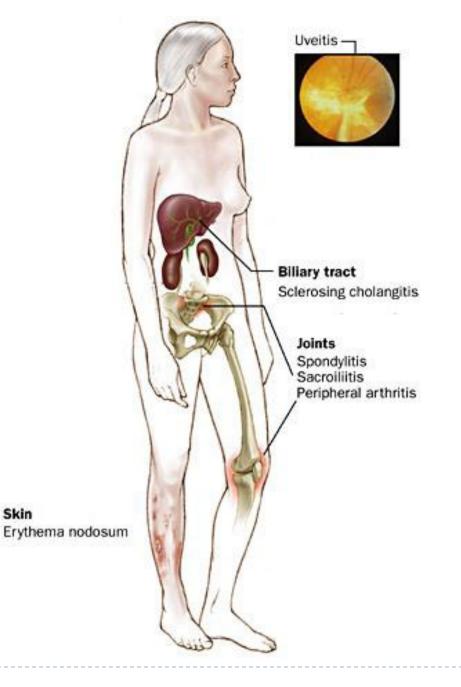
Significant risk of **perforation**



Histology similar to CD but inflammation is limited to the mucosa and superfical submucosa

Can you spot the ulcer?

Granulomas are not present



Clinical Features

Relapsing attacks of bloody diarrhea with expulsion of stringy, mucoid material, lower abdominal pain and cramps

Triggers unknown

Colectomy cures intestinal disease not extraintestinal manifestations

Major characteristics

Chrohn

- Skip lesions/cobbelstone
- Aphthous/serpentine ulcers
- Strictures
- Fissures/fistulas/perforation
- Creeping fat
- Neutrophilic infiltrate/crypt abscesses
- Paneth cell metaplasia
- Non-caseating granulomas (hallmark)

Ulcerative Colitis

- Limited to colon/rectum (BW ileitis)
- Broad based ulcers
- No thickening/no strictures
- Toxic Megacolon/perforation
- Neuromuscular function defect/dilation
- Neutrophilic infiltrate/crypt abscesses*
- Mucosal atrophy
- Regenerating mucosa (pseudopolyp)

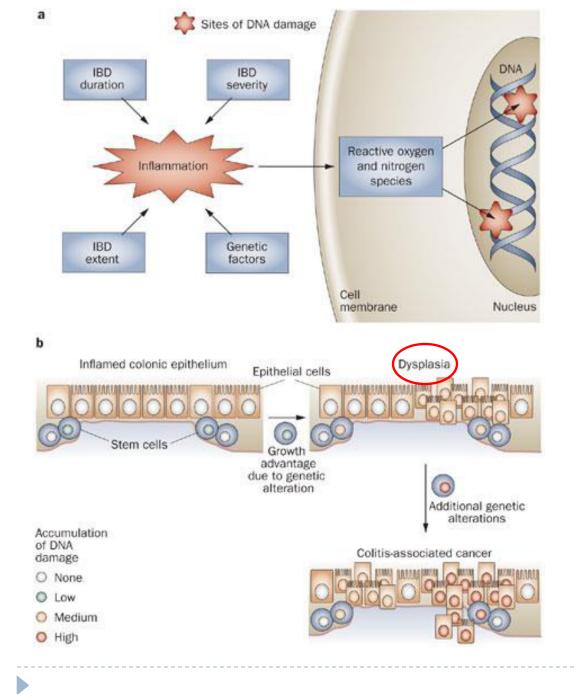
Major characteristics

Chrohn

- Intermittent attacks
- Triggers (stress, diet, smoking)
- Iron deficiency anemia (colon)
- Malabsoption (protein, B₁₂, bile salts)
- Stricture surgical resection relapse
- Extra-GI fistulas (GU, skin)
- Extraintestinal manefestations (uveitis, migratory polyarthritis, ...etc.)

Ulcerative Colitis

- Intermittent attacks
- Triggers less defined (\u00e4smoking)
- Potential blood loss anemia
- Malabsoption less of a problem
- Colectomy cures intestinal disease
- No
- Similar extraintestinal manefestations (uveitis, migratory polyarthritis, ...etc.) Colectomy does not change these.



Colitis associated neoplasia Inflammation ↓ Free radicals ↓ DNA damage and/or Protein modification

- Oncogene activation
- Tumor suppressor inactivation

Time/severity of inflammation increase risk (especially those with PSC)