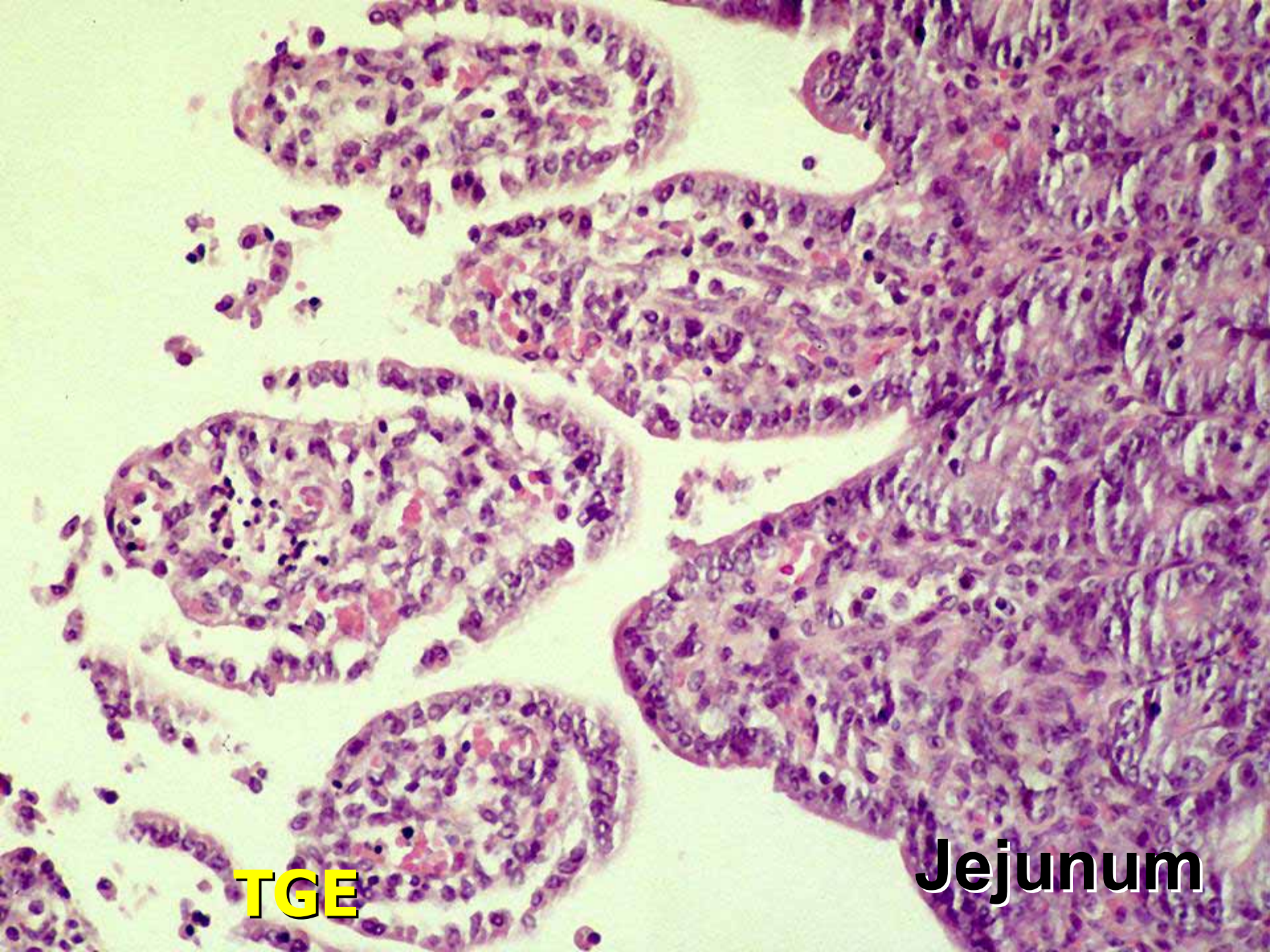
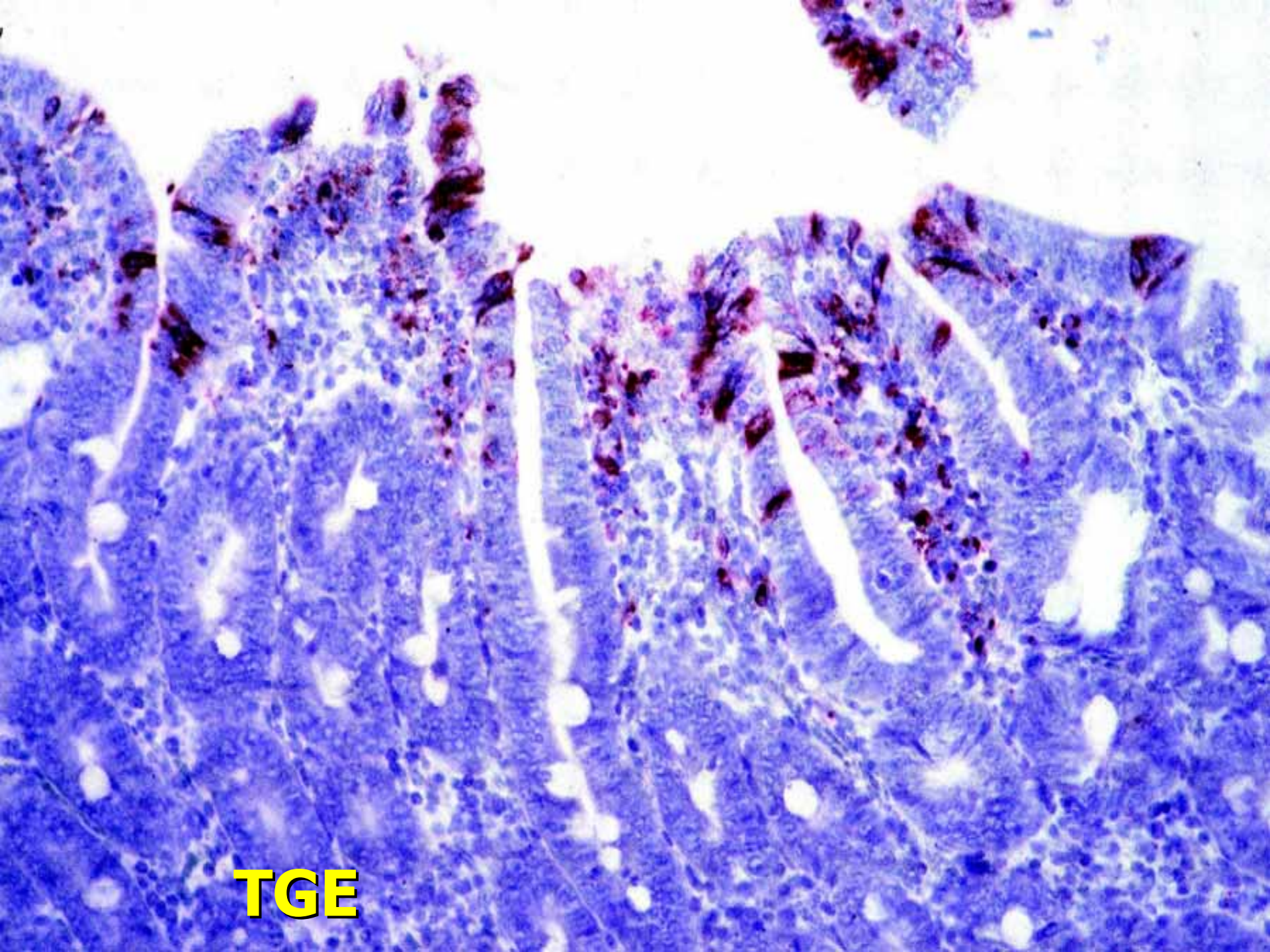

TGE
Jejunum


TGE
Jejunum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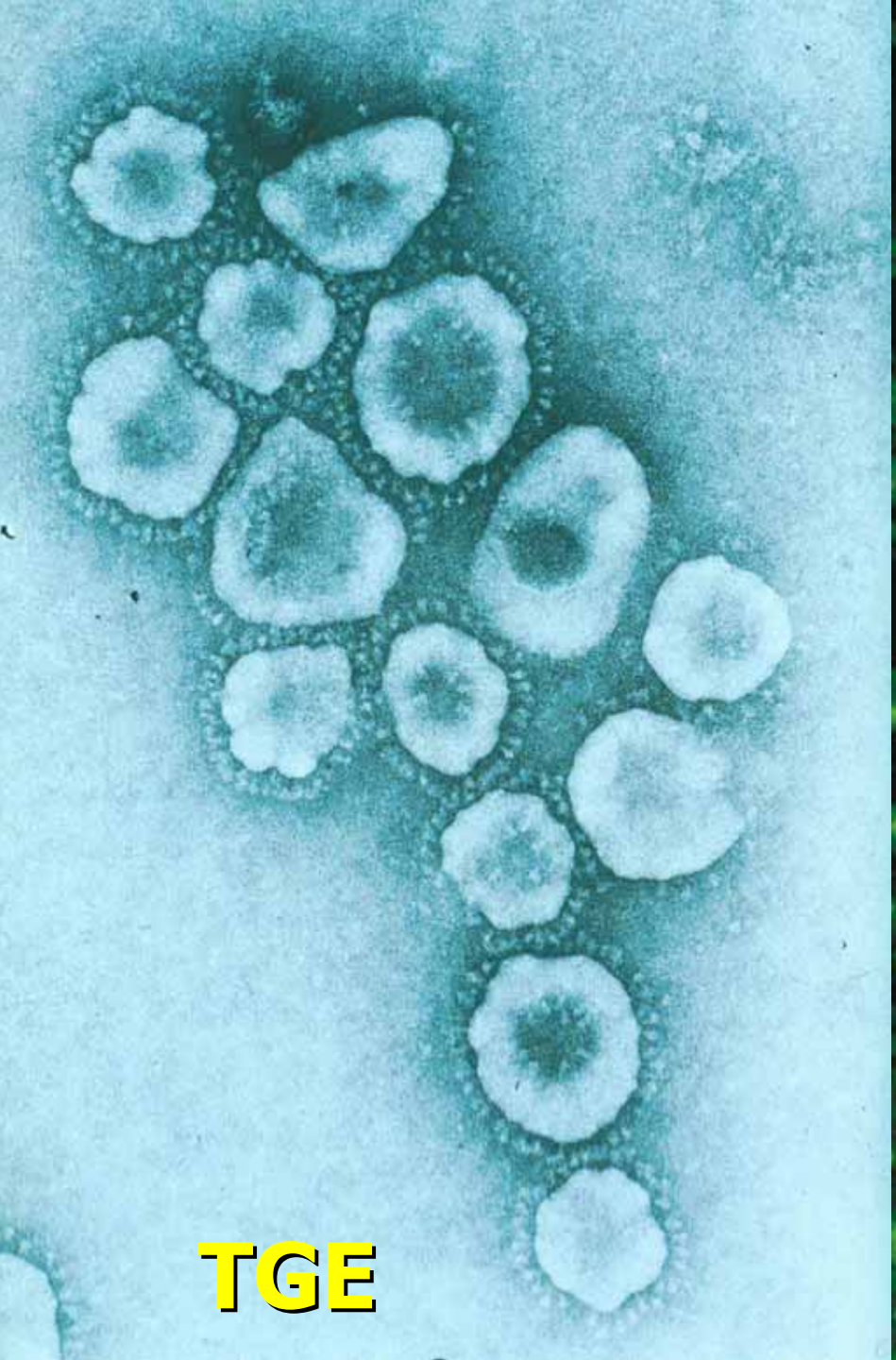
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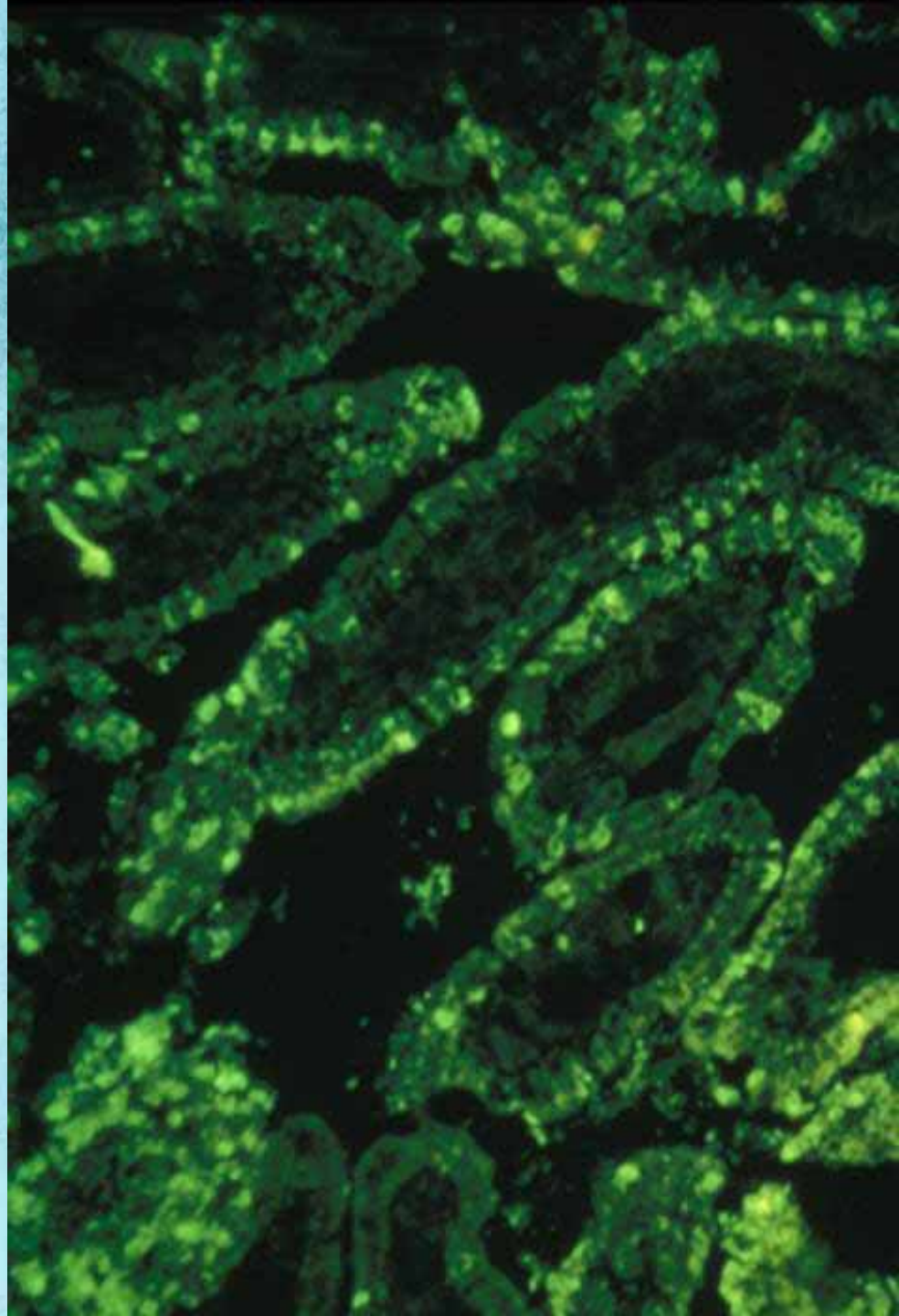

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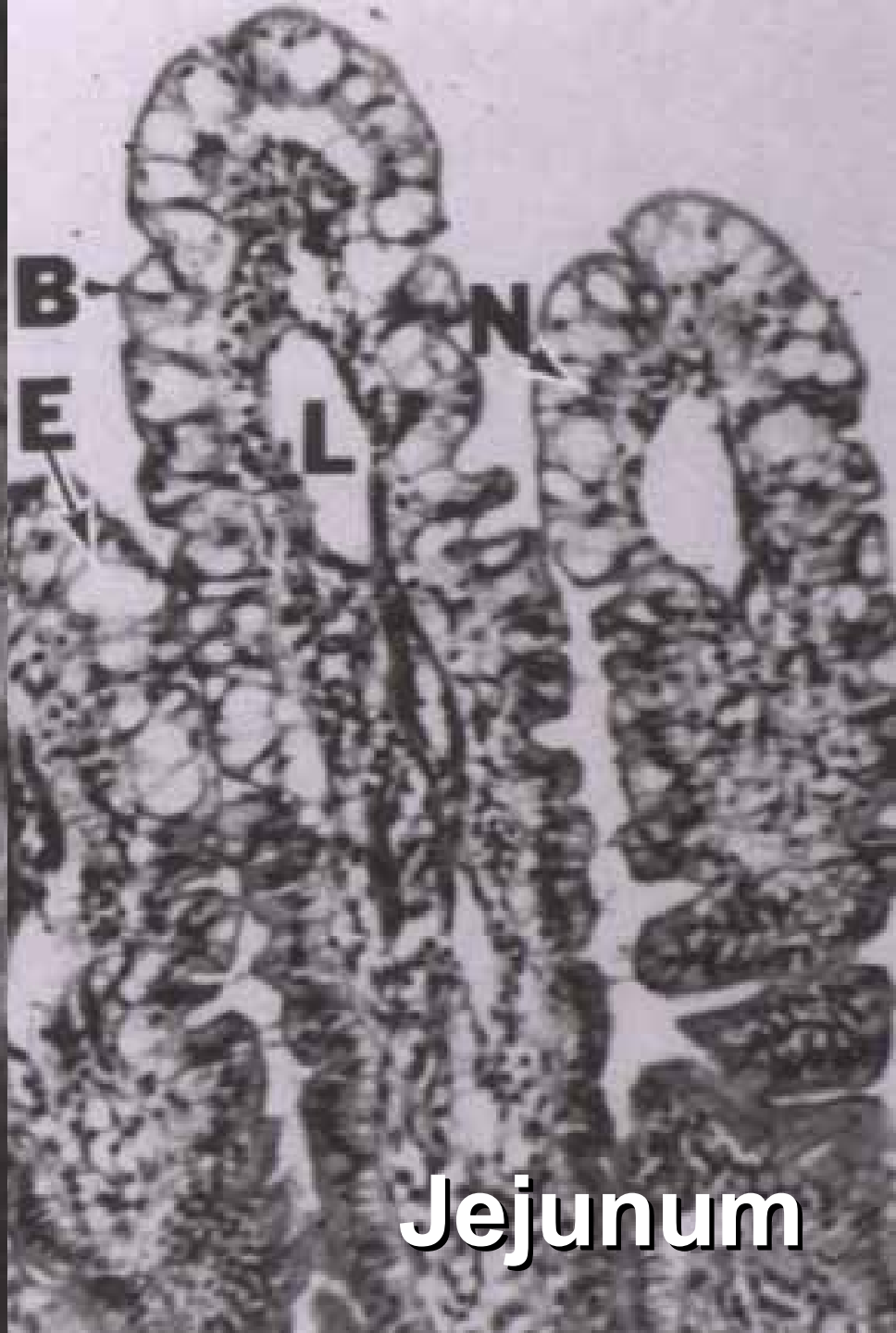
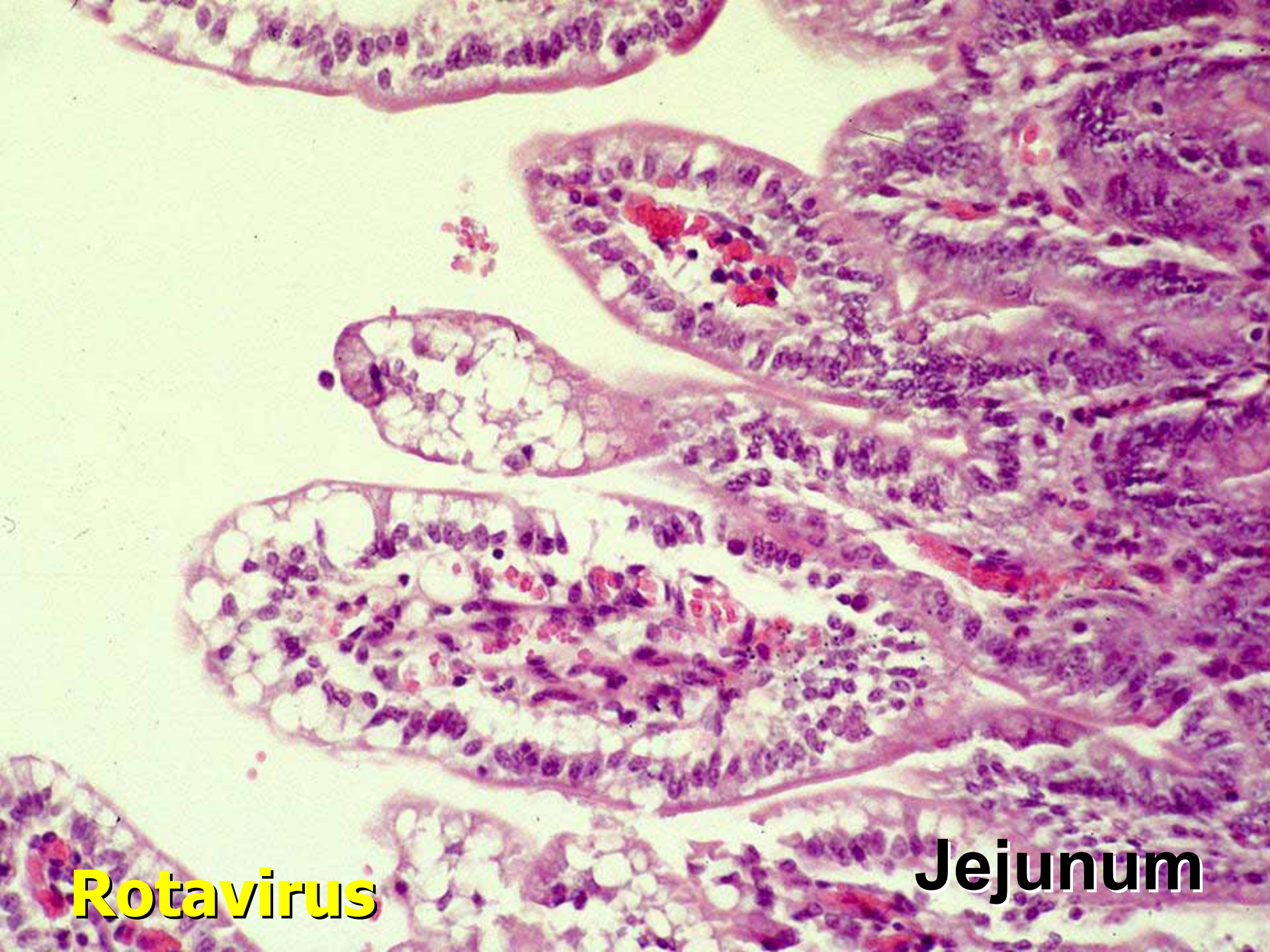
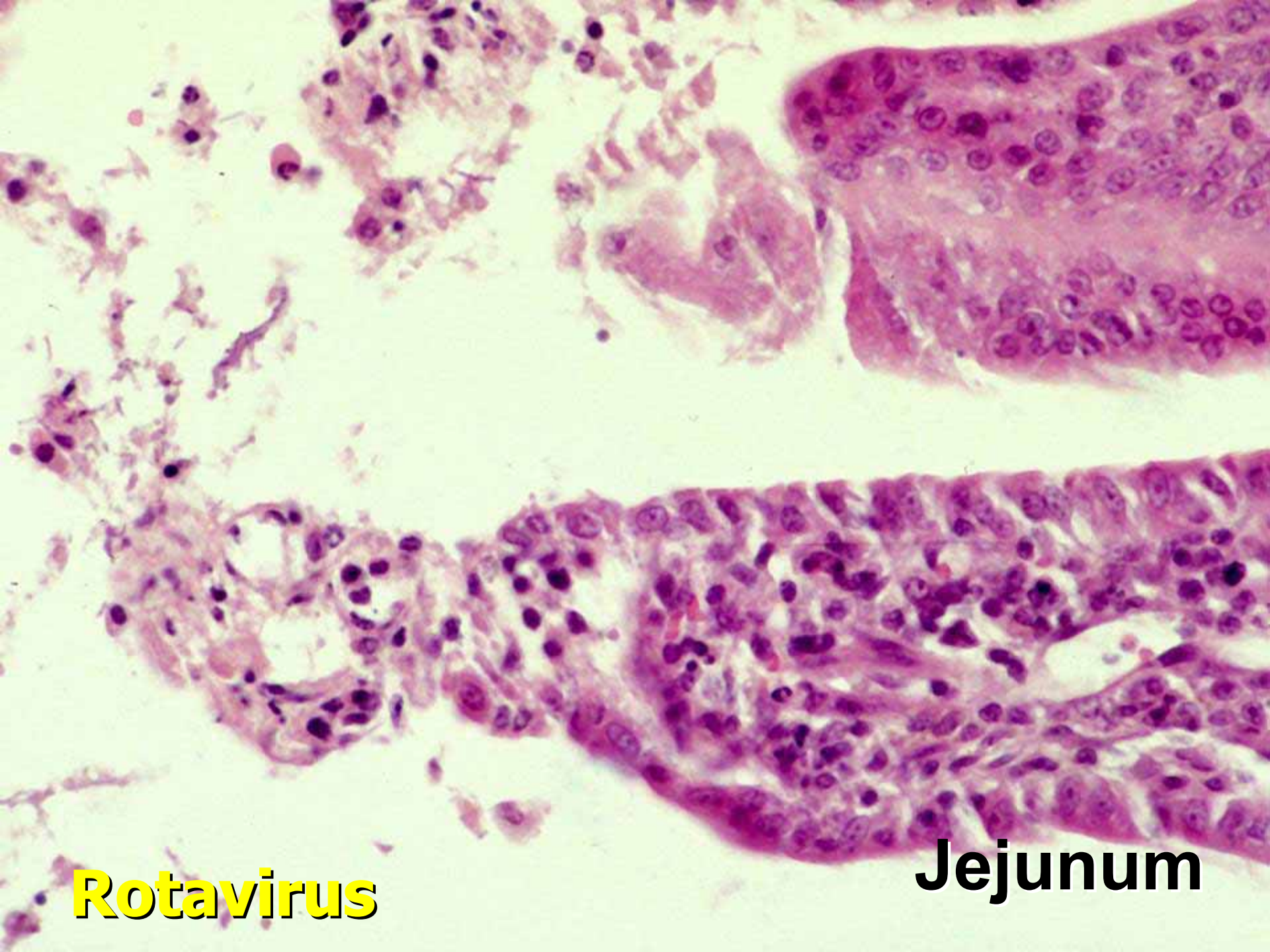


Jejunum


Rotavirus
Jejunum


Rotavirus
Jejunum

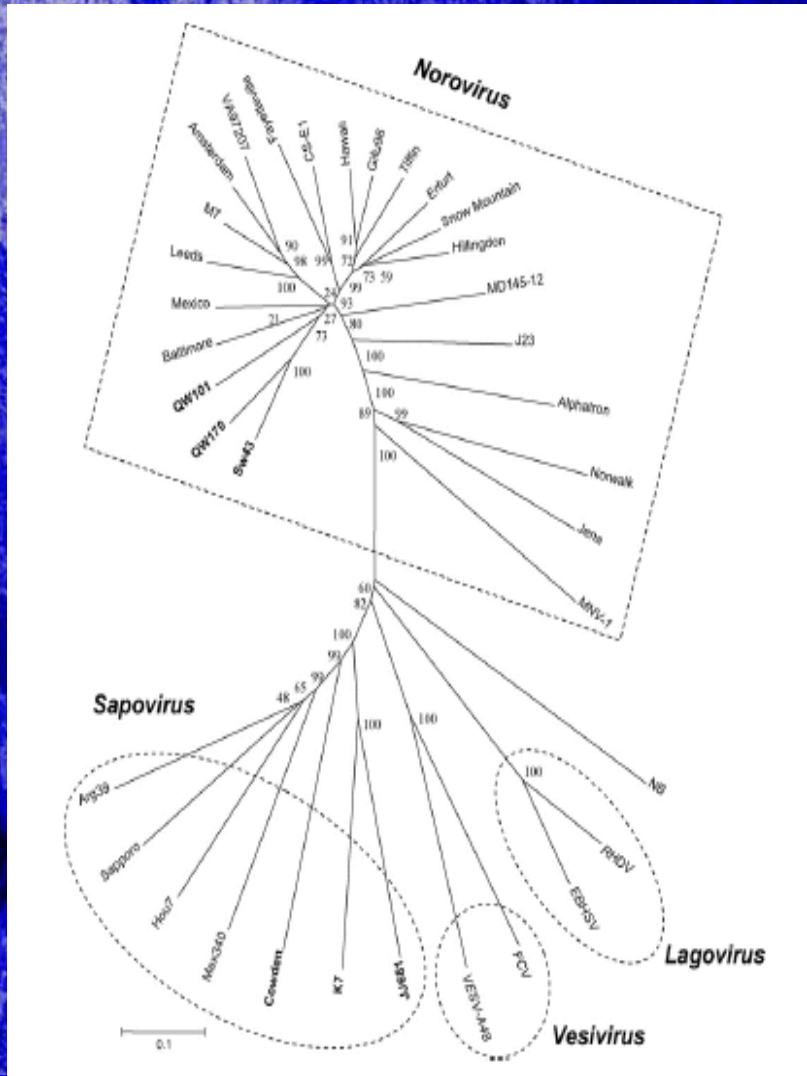
Porcine Norovirus and Sapovirus

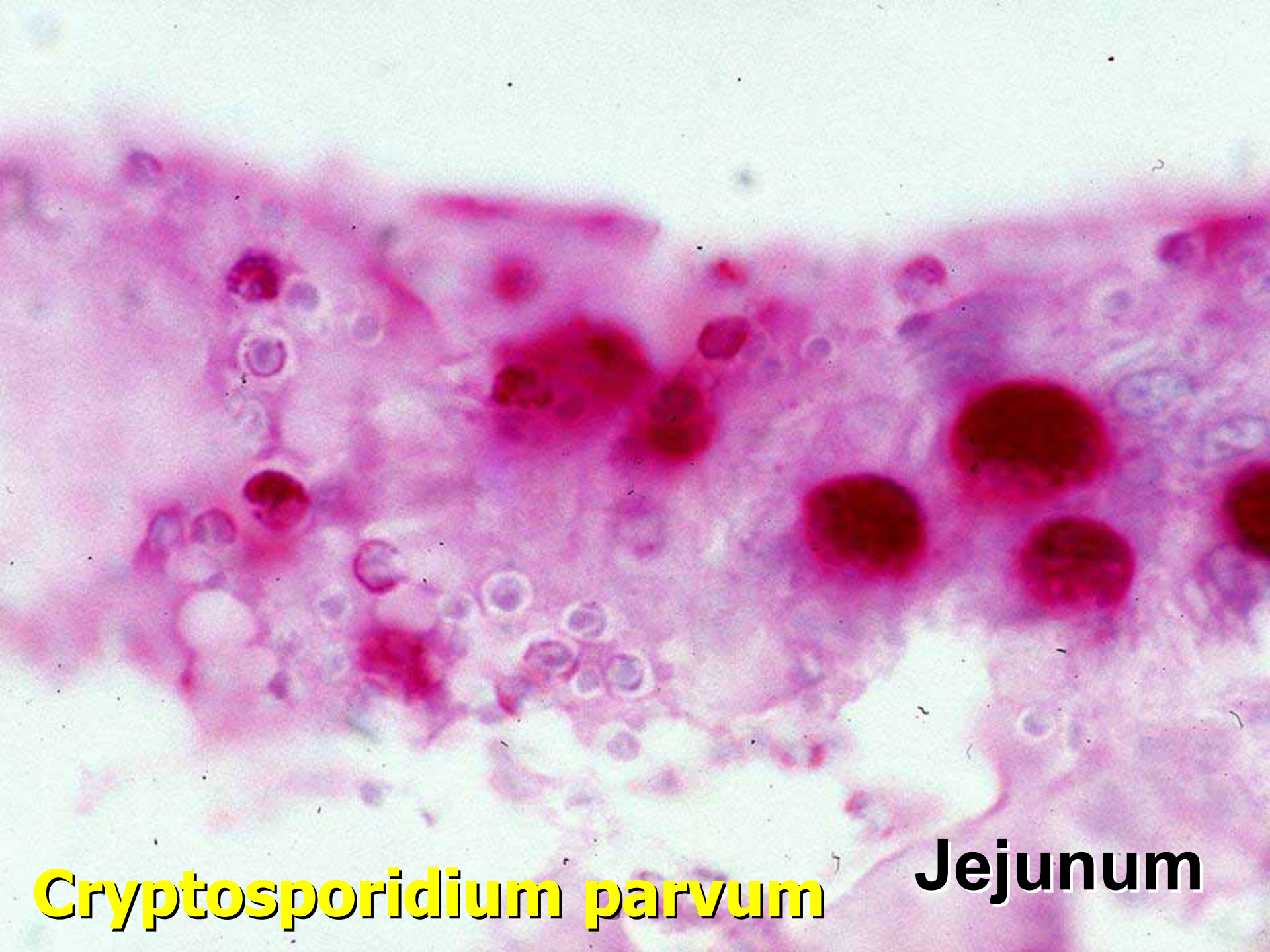
Porcine Norovirus

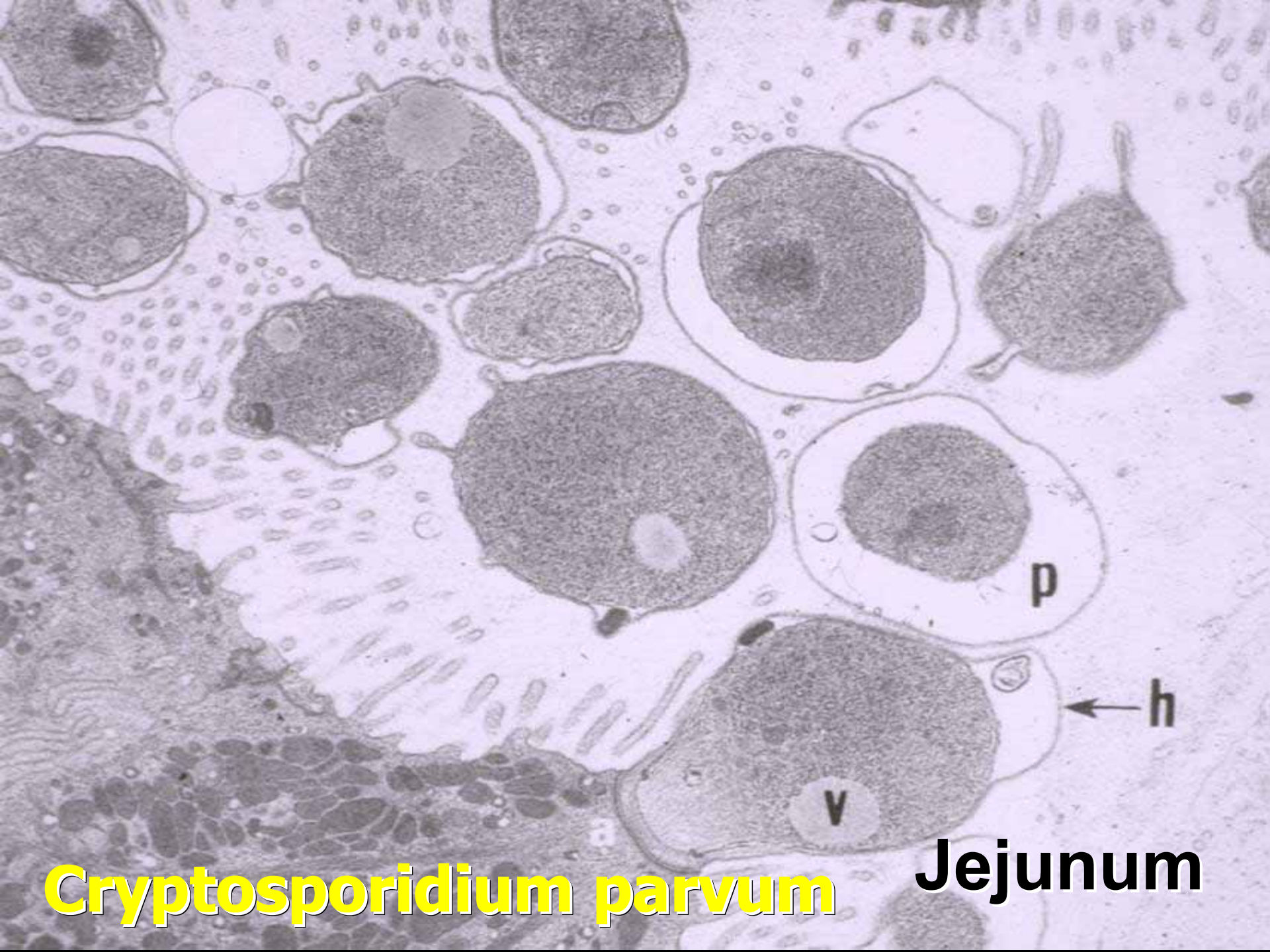
- Detected in finisher pigs (20-24 wks) only, with an overall prevalence of 20%

Porcine Sapovirus

- Cowden-like porcine Sapovirus most prevalent
- Overall prevalence of 62%
- Most prevalent in post-weaning pigs (83%)
- Least prevalent in nursing pigs (21%)

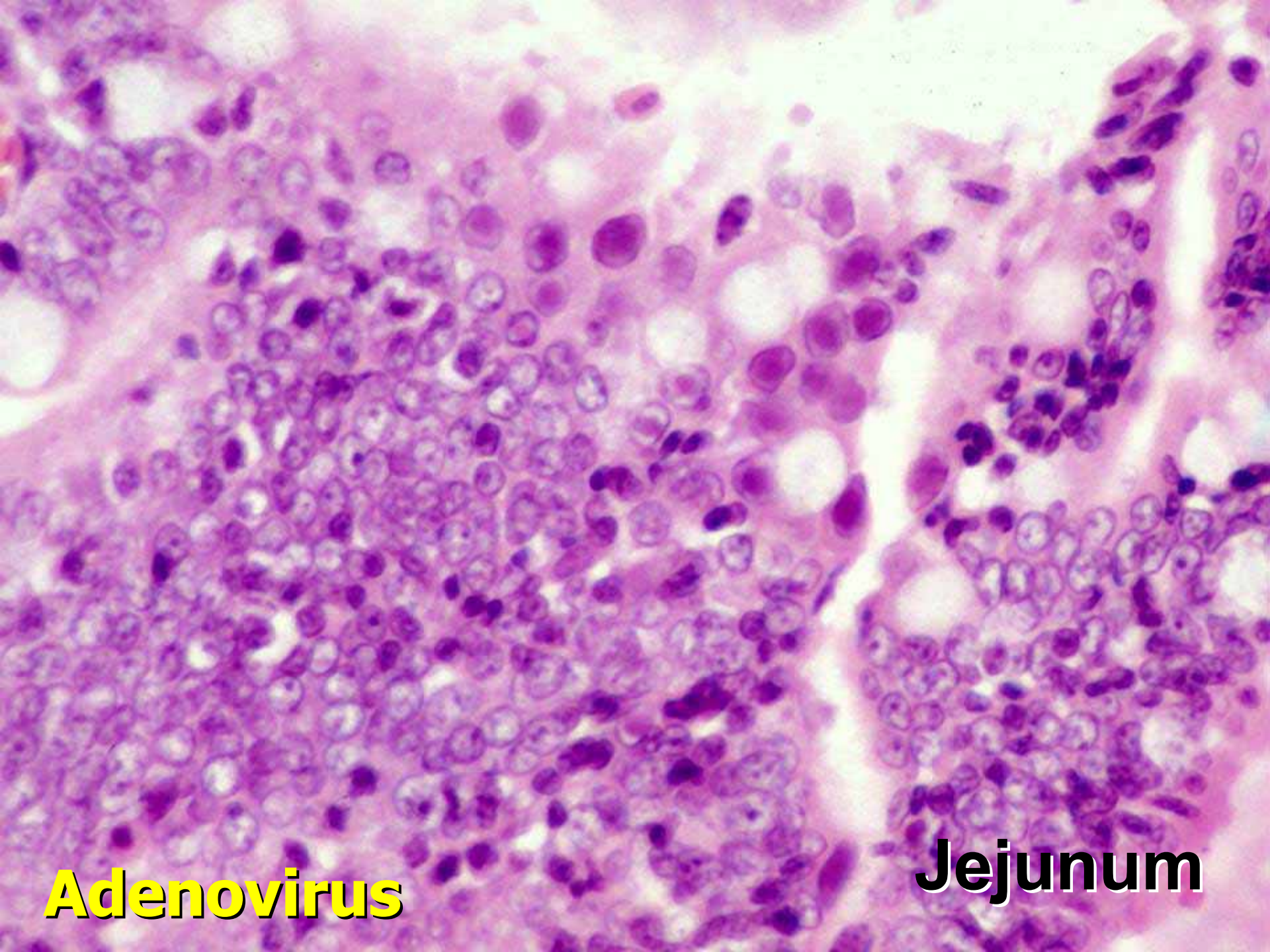


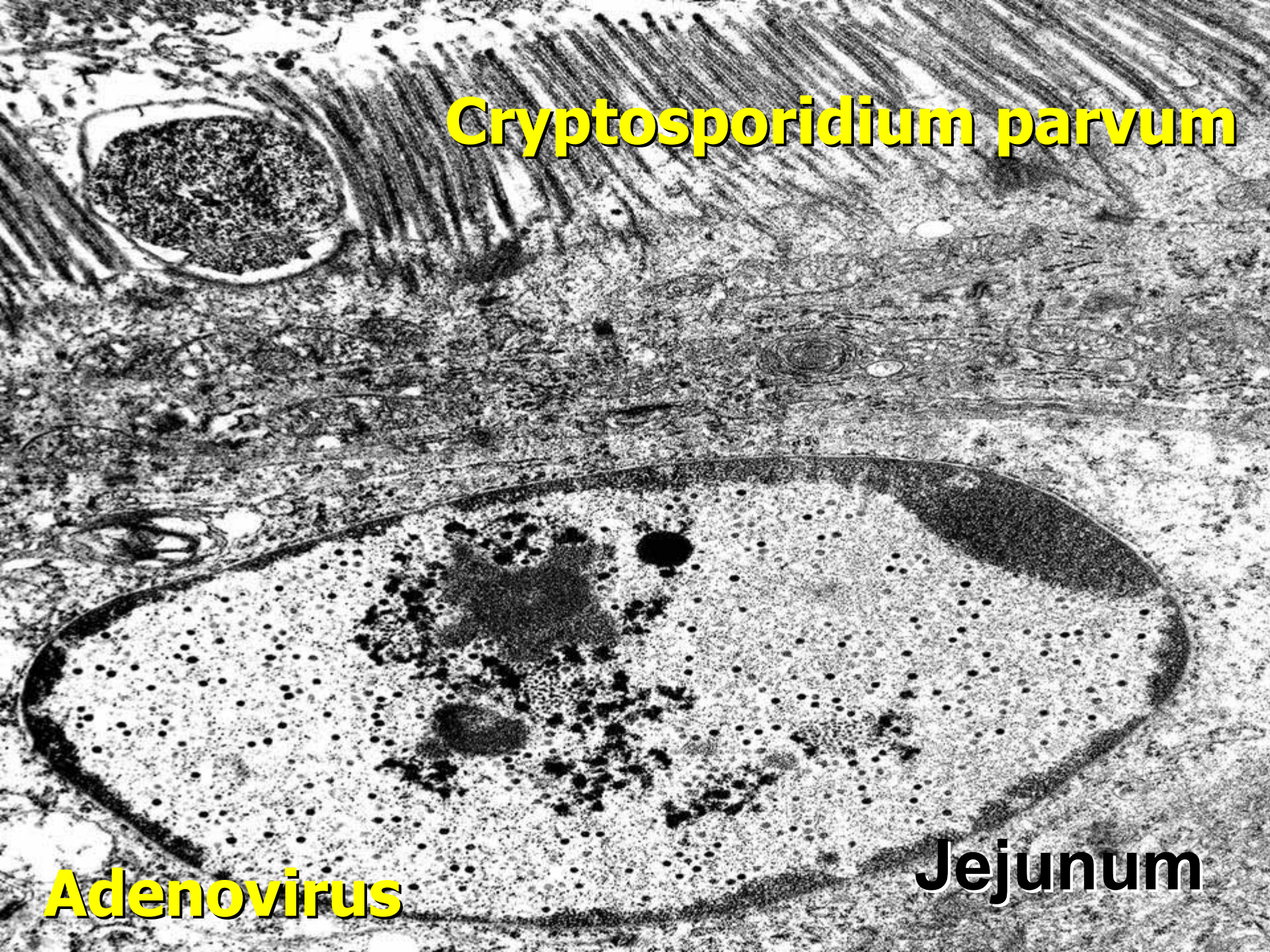

Cryptosporidium parvum
Jejunum

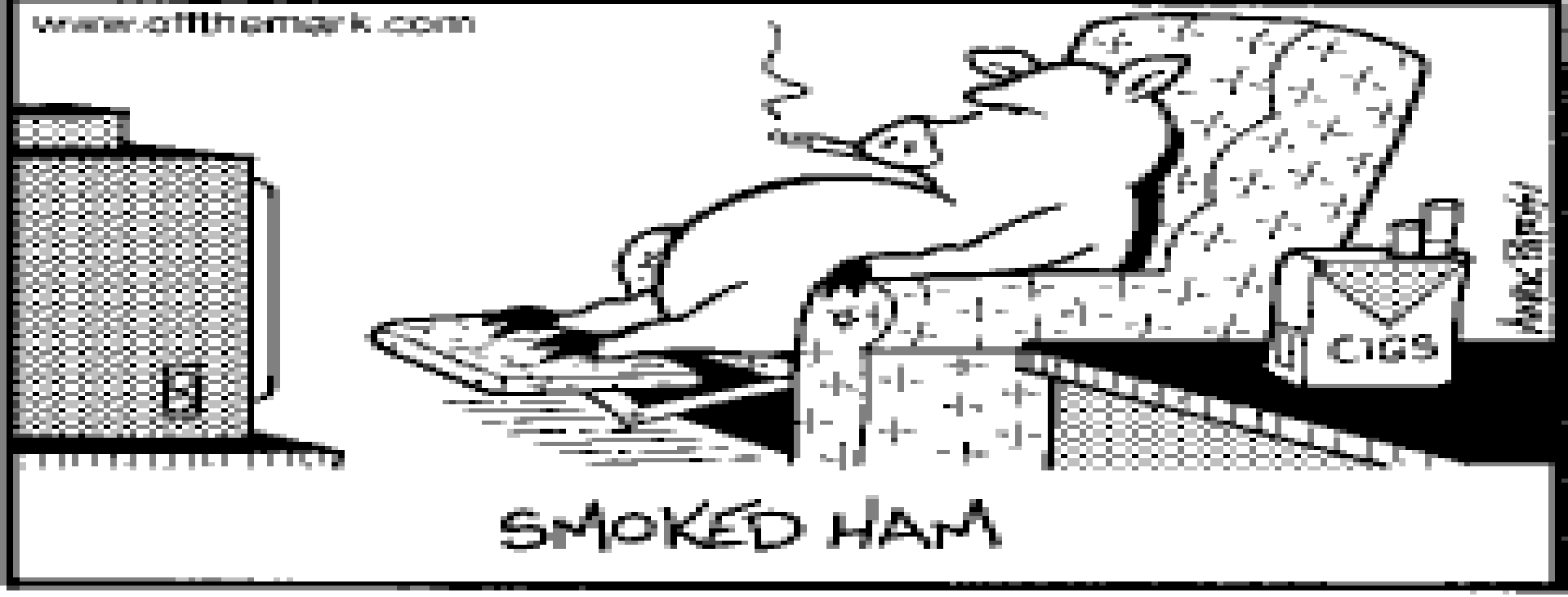


Cryptosporidium parvum

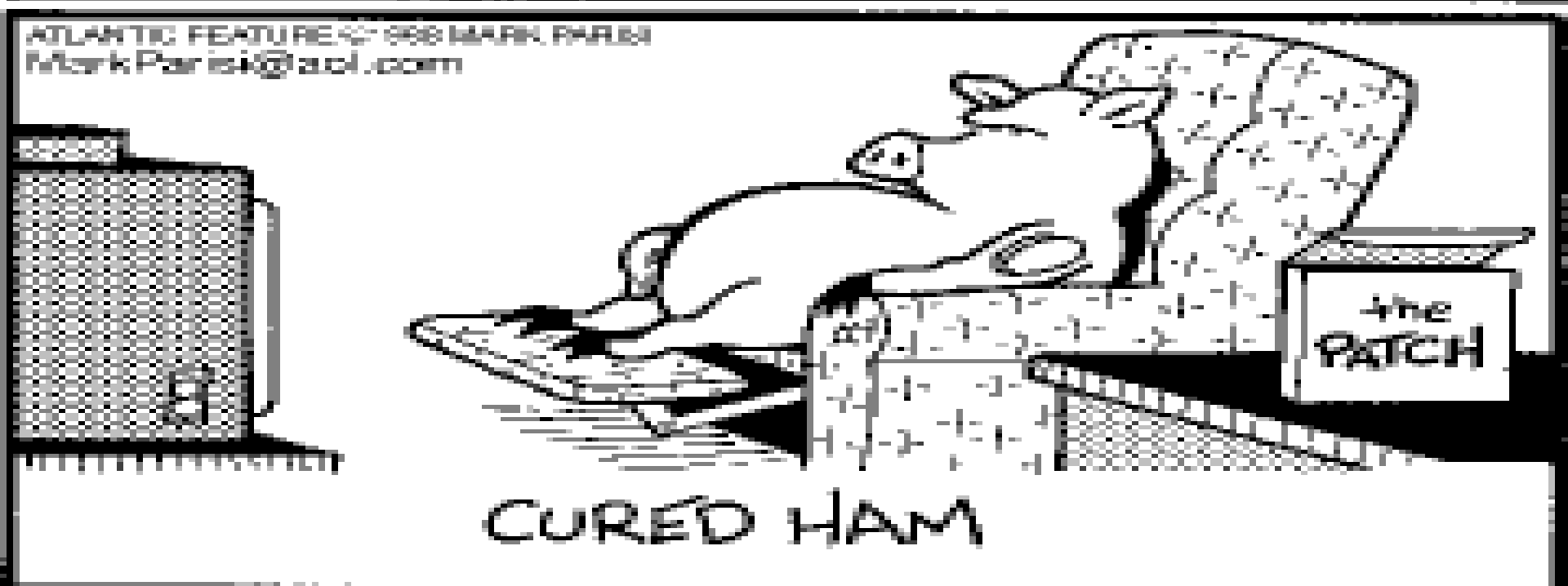
Jejunum


Adenovirus
Jejunum


Cryptosporidium parvum
Adenovirus
Jejunum



SMOKED HAM



CURED HAM

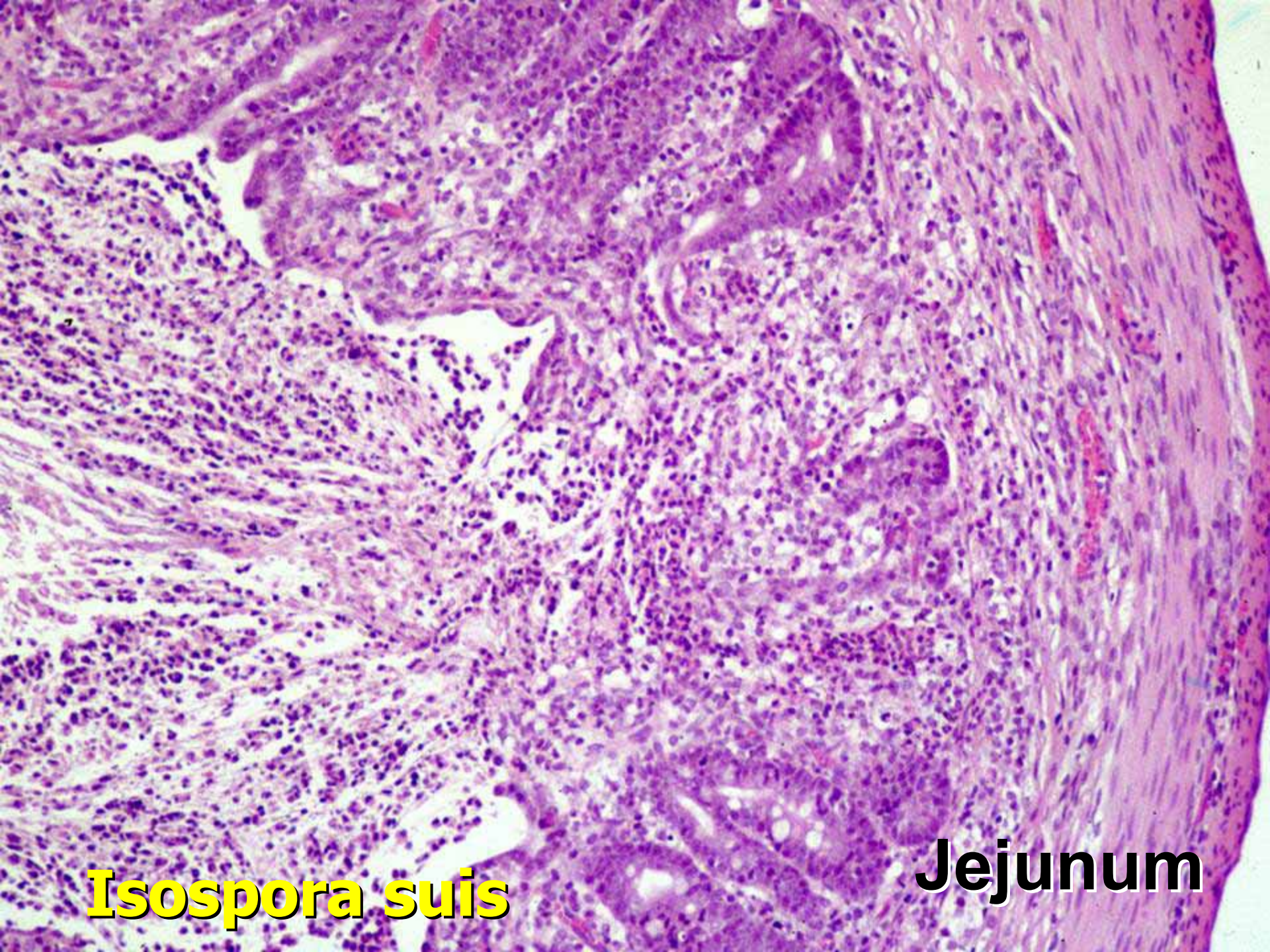
Isospora suis

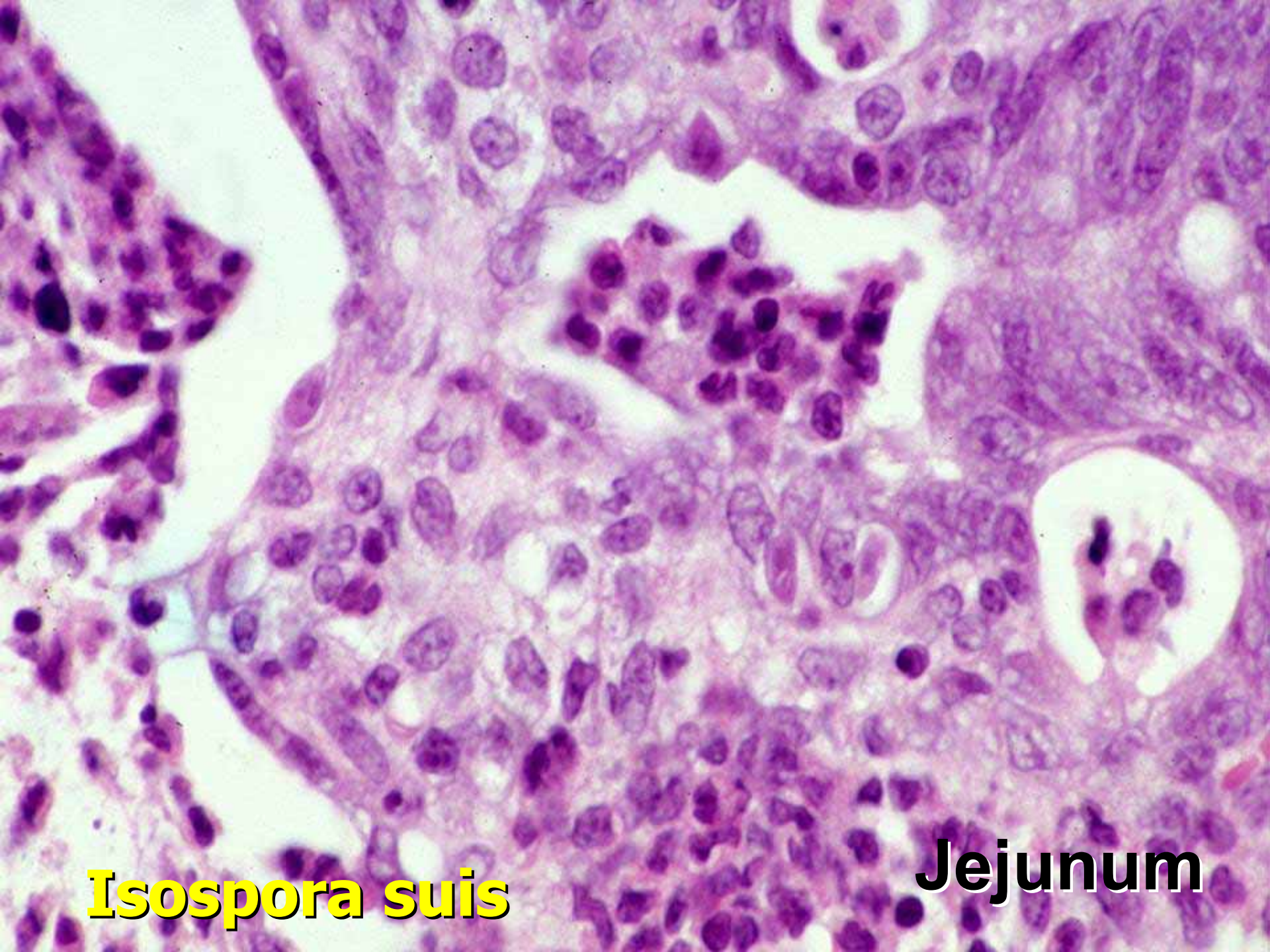
- most common in pigs from 5 days to 4 weeks-of-age
- rarely occurs in older pigs (Eimeria debliecki)
- I. suis replicates through 2 sequential asexual cycles (schizogeny) and 1 sexual cycle (gametogeny) in the cytoplasm of the epithelial cells in the small intestine
- moderate to severe atrophic enteritis
- bright yellow fibrinous mucosal pseudomembrane, can be removed with gentle scraping to reveal a glistening mucosa beneath

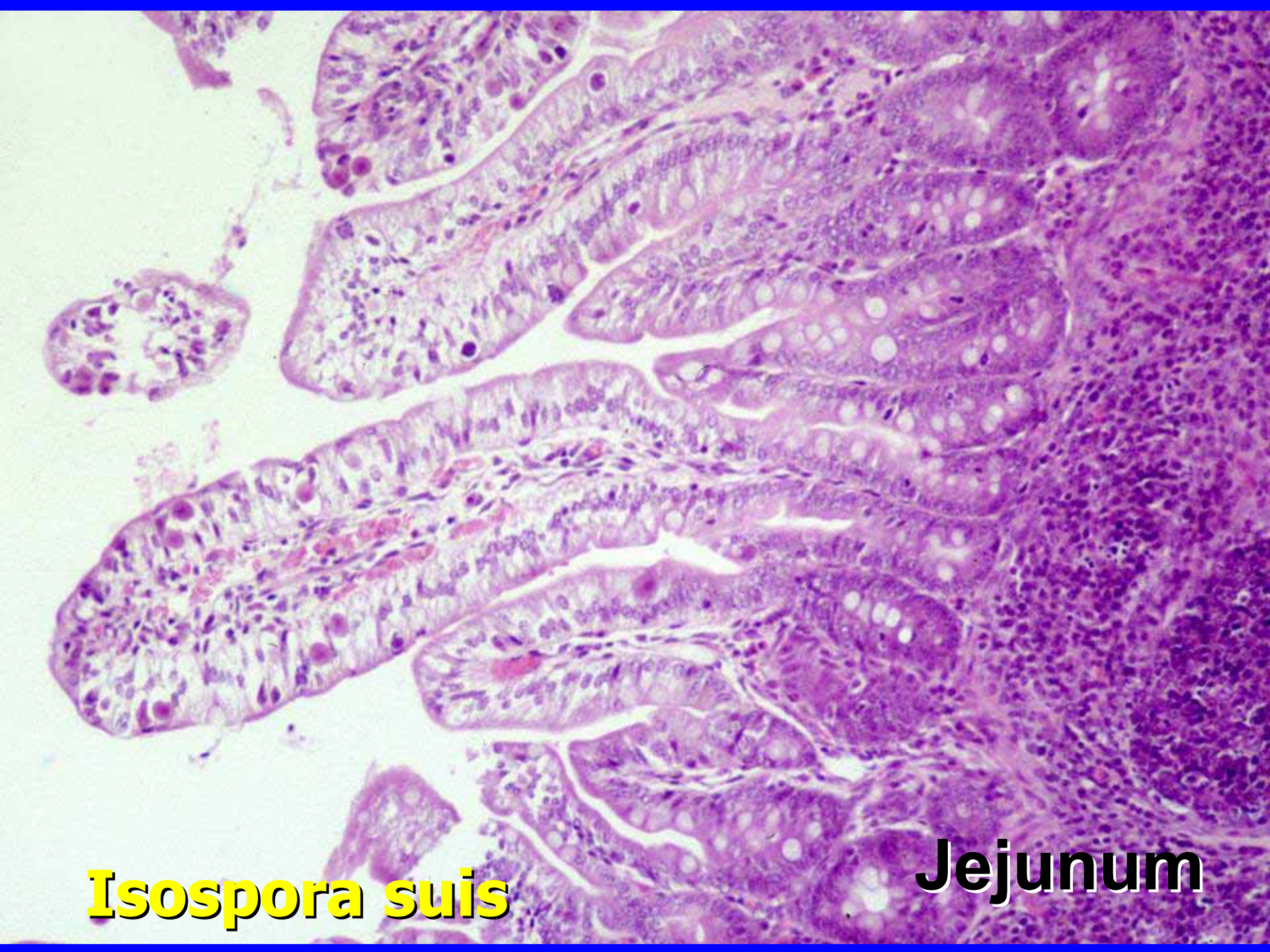
Isospora suis


Isospora suis

Isospora suis


Isospora suis
Jejunum


Isospora suis
Jejunum


Isospora suis
Jejunum

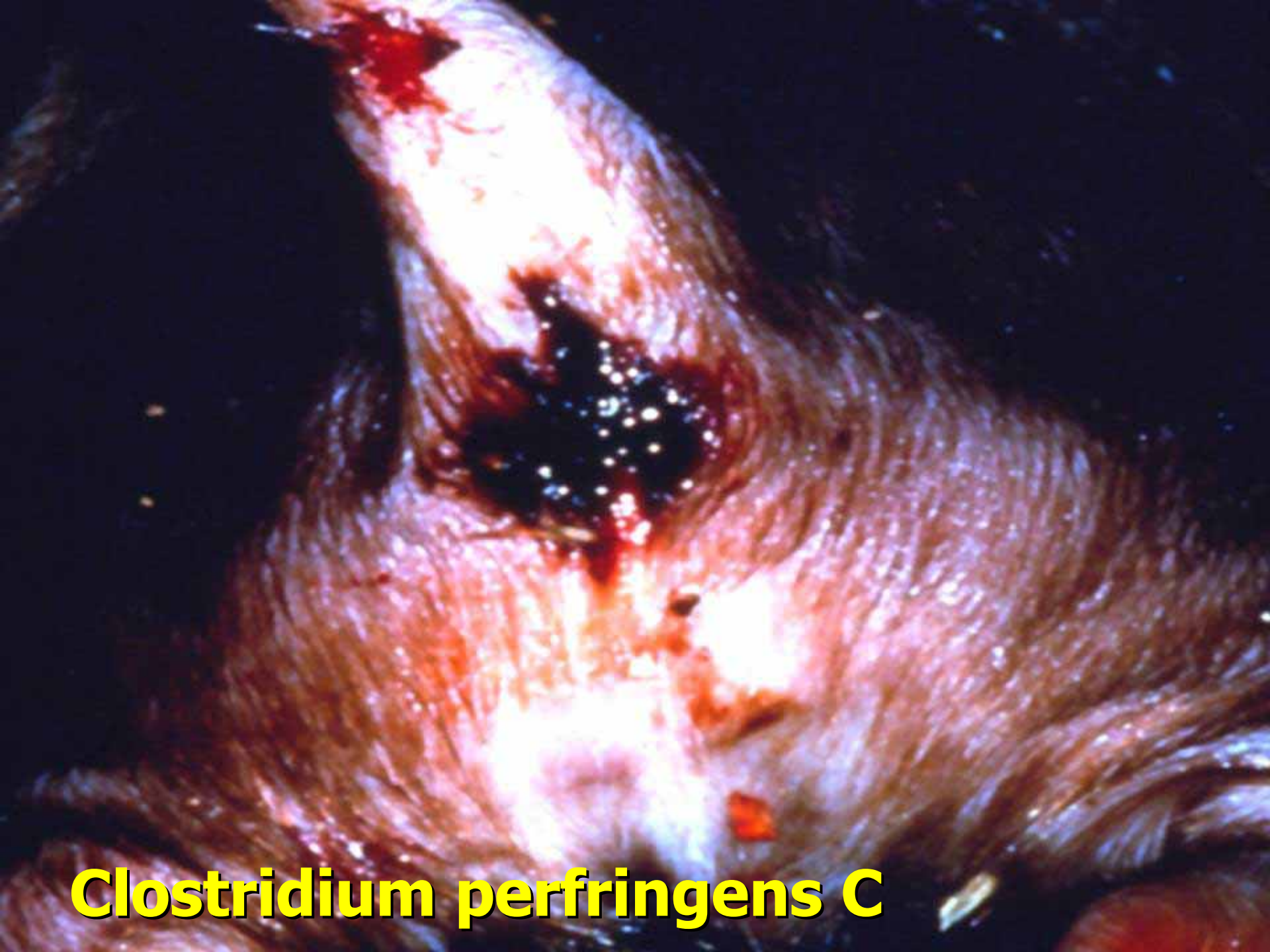

Isospora suis
Jejunum

Clostridium perfringens type C

- less than 1 week of age
- some pigs may survive initially, but tend to grow poorly and die by 2 – 3 weeks-of-age
- present in small numbers in sow feces
- out-compete “normal flora” C. perfringens strains in gut
- segmental transmural necrohemorrhagic enteritis with subserosal and intramural emphysema

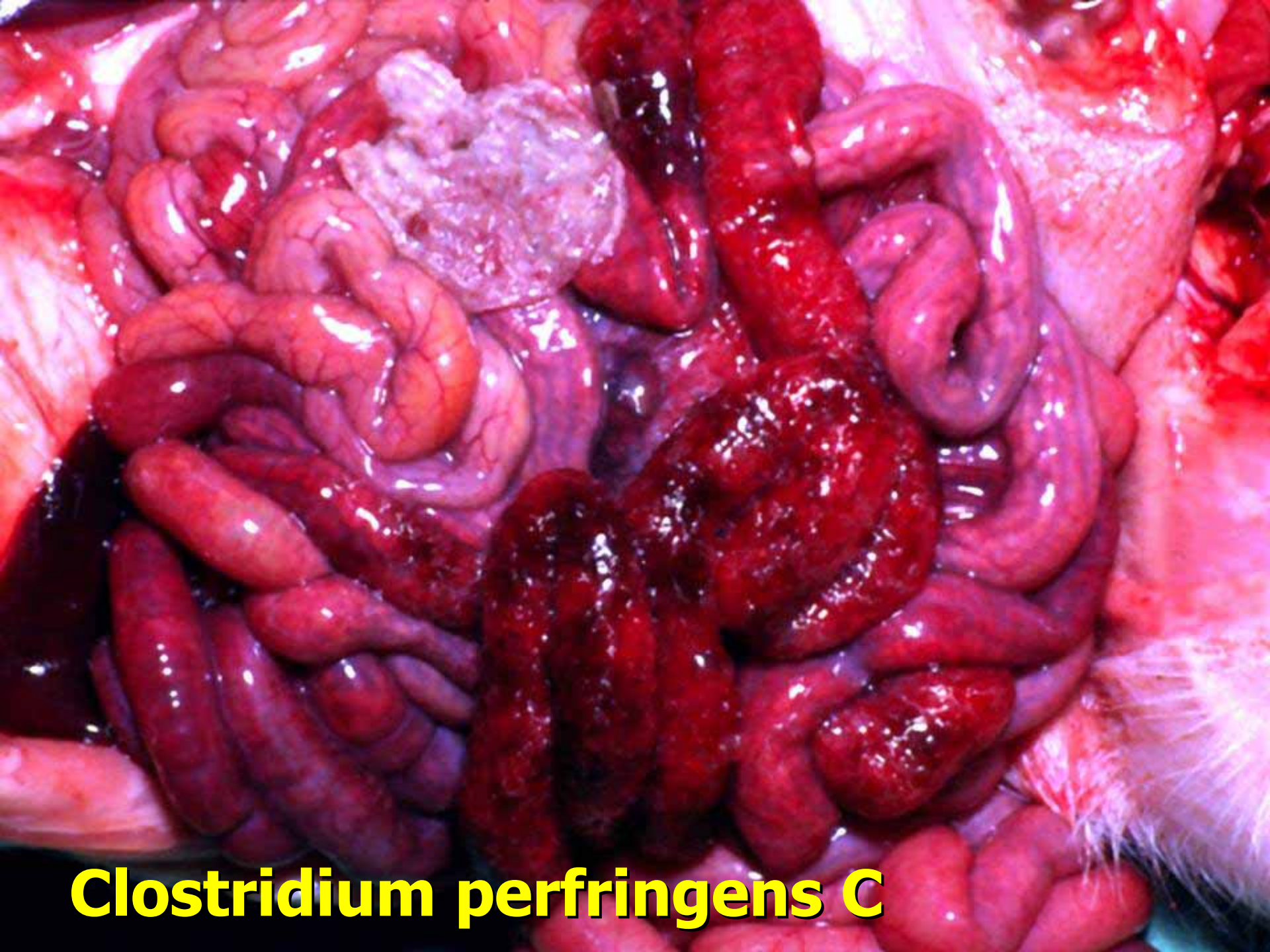
Clostridium perfringens type C

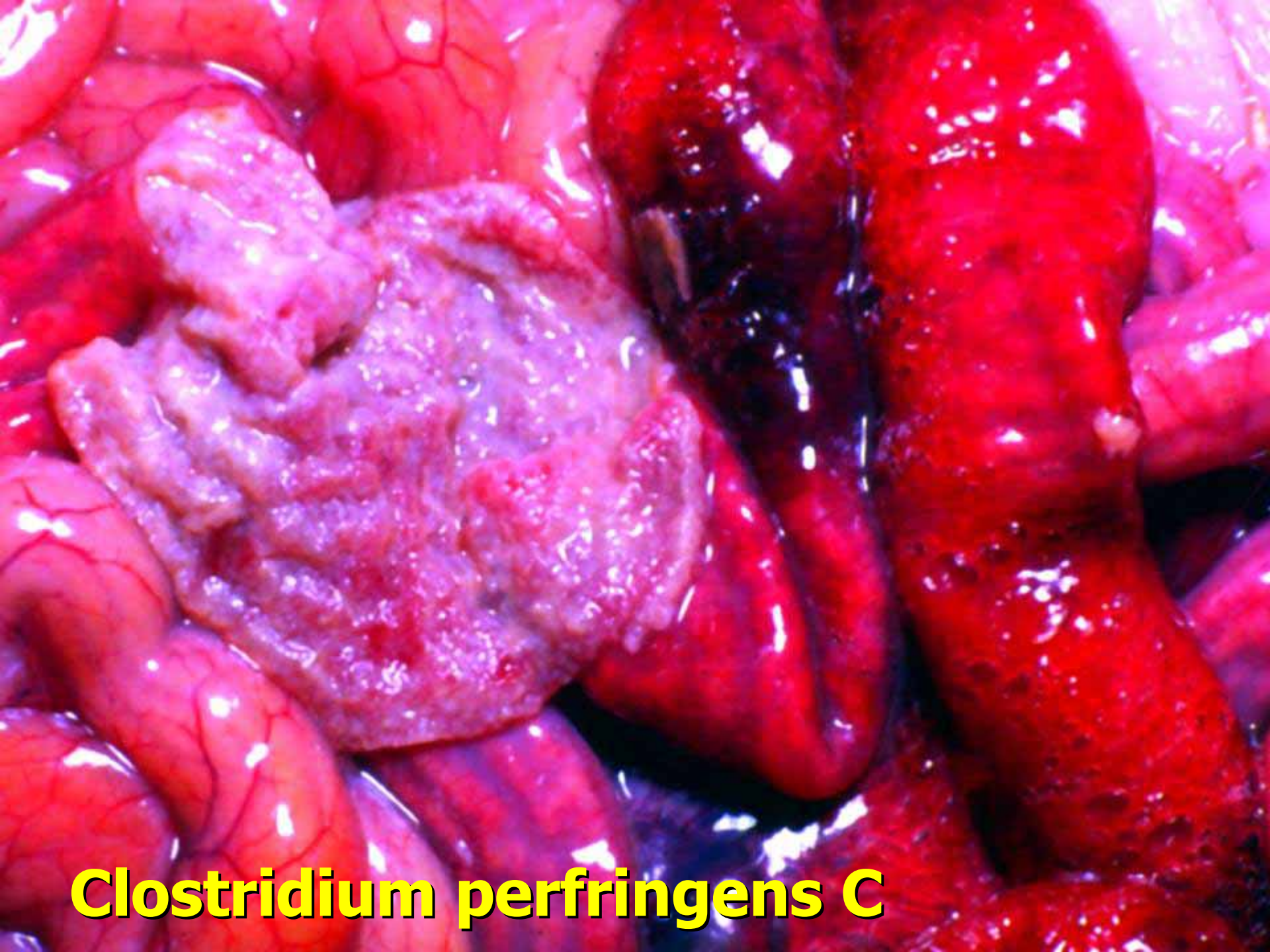
- necrosis begins before bacterial contact with enterocytes in jejunum
  - damage to microvilli, mitochondria, terminal capillaries
  - likely due to effects of beta toxin (CPB)
  - acts in absence of normal protease activity
- adheres, colonizes (often after necrosis) by unknown mechanisms, does not actively invade
- dramatic epithelial necrosis, emphysema
- increased capillary permeability may facilitate uptake to circulation, promote systemic effects
- ultimate cause of death: toxemia


Clostridium perfringens C

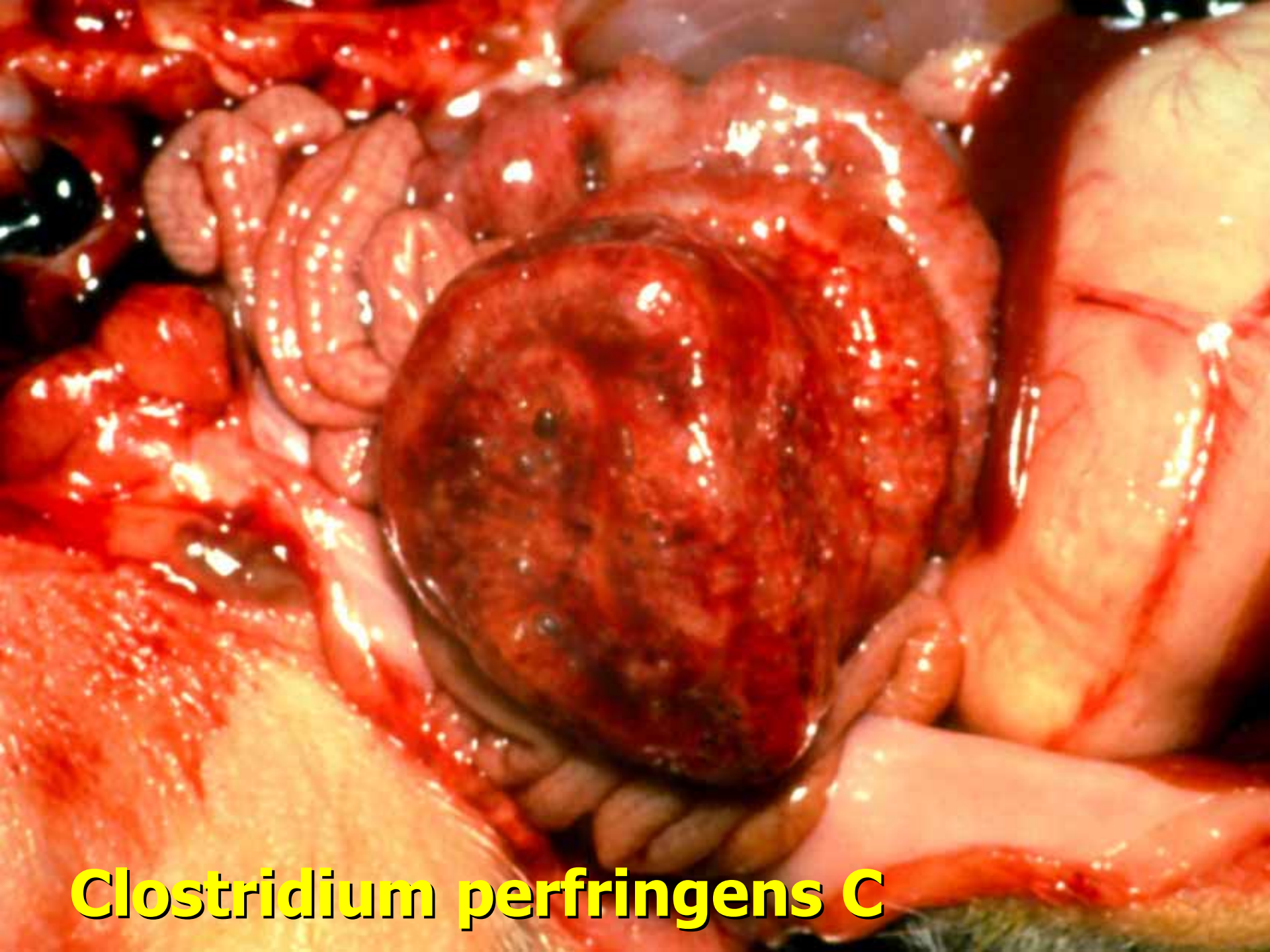
Clostridium perfringens C

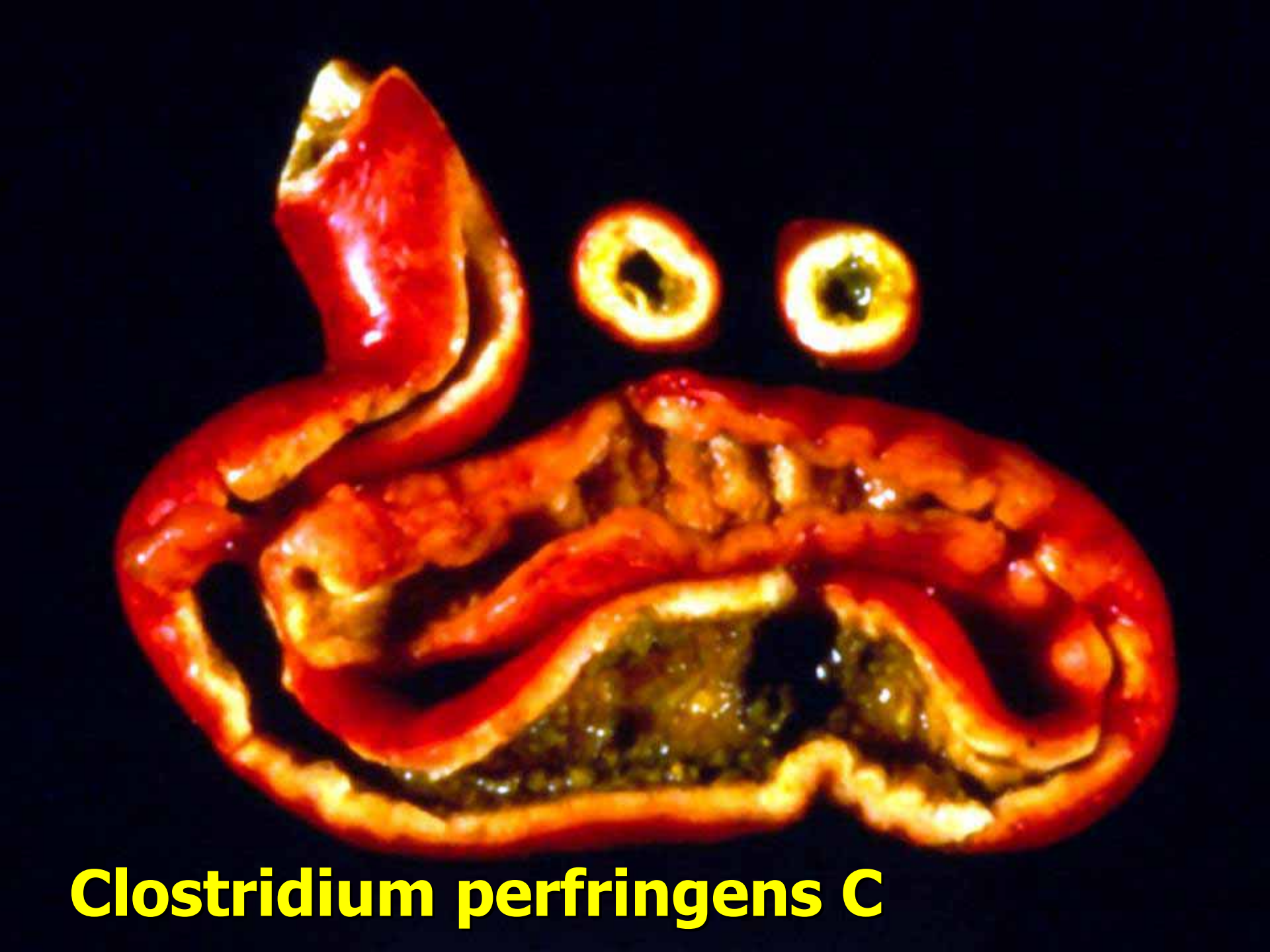

Clostridium perfringens C

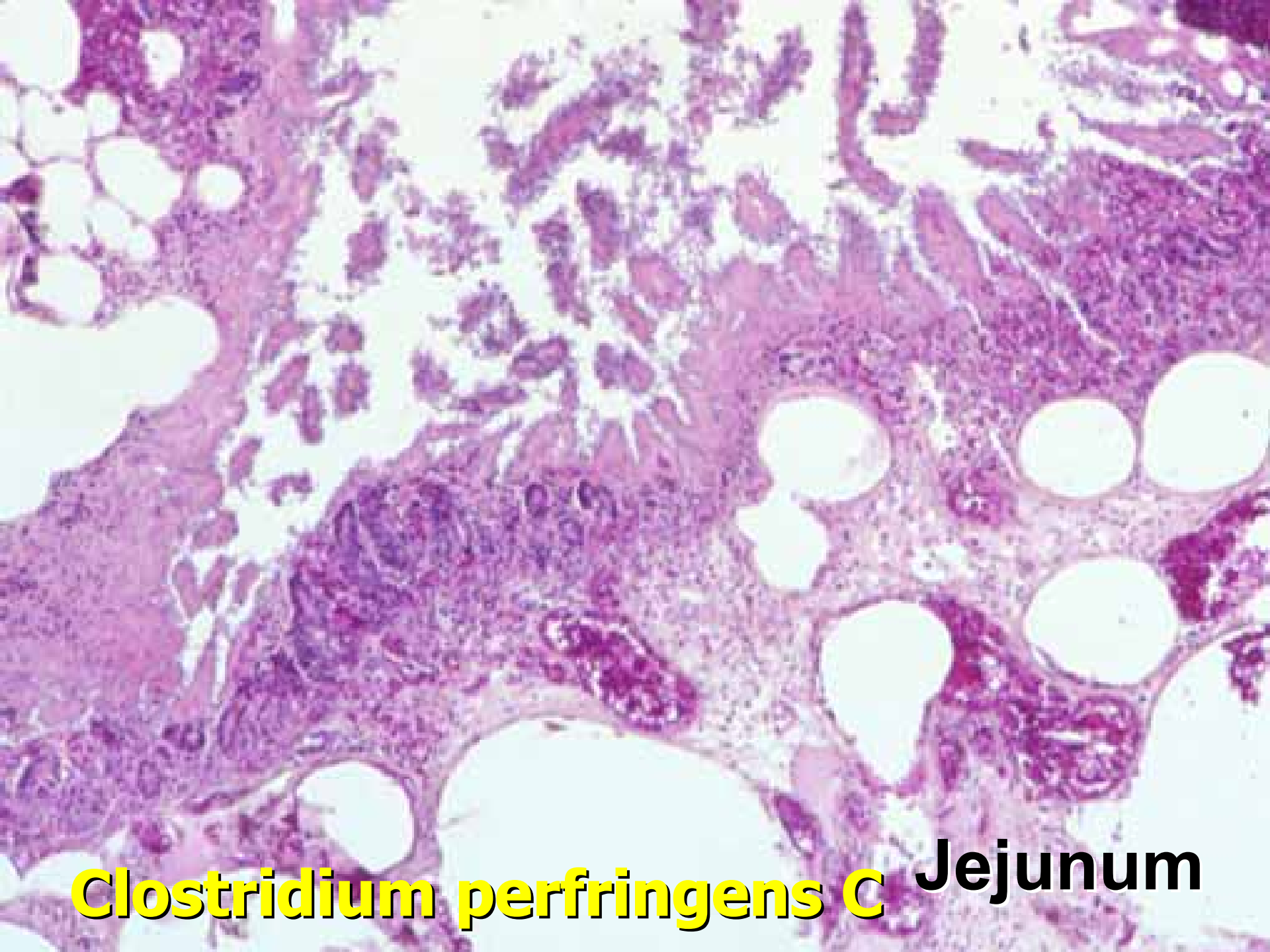

Clostridium perfringens C

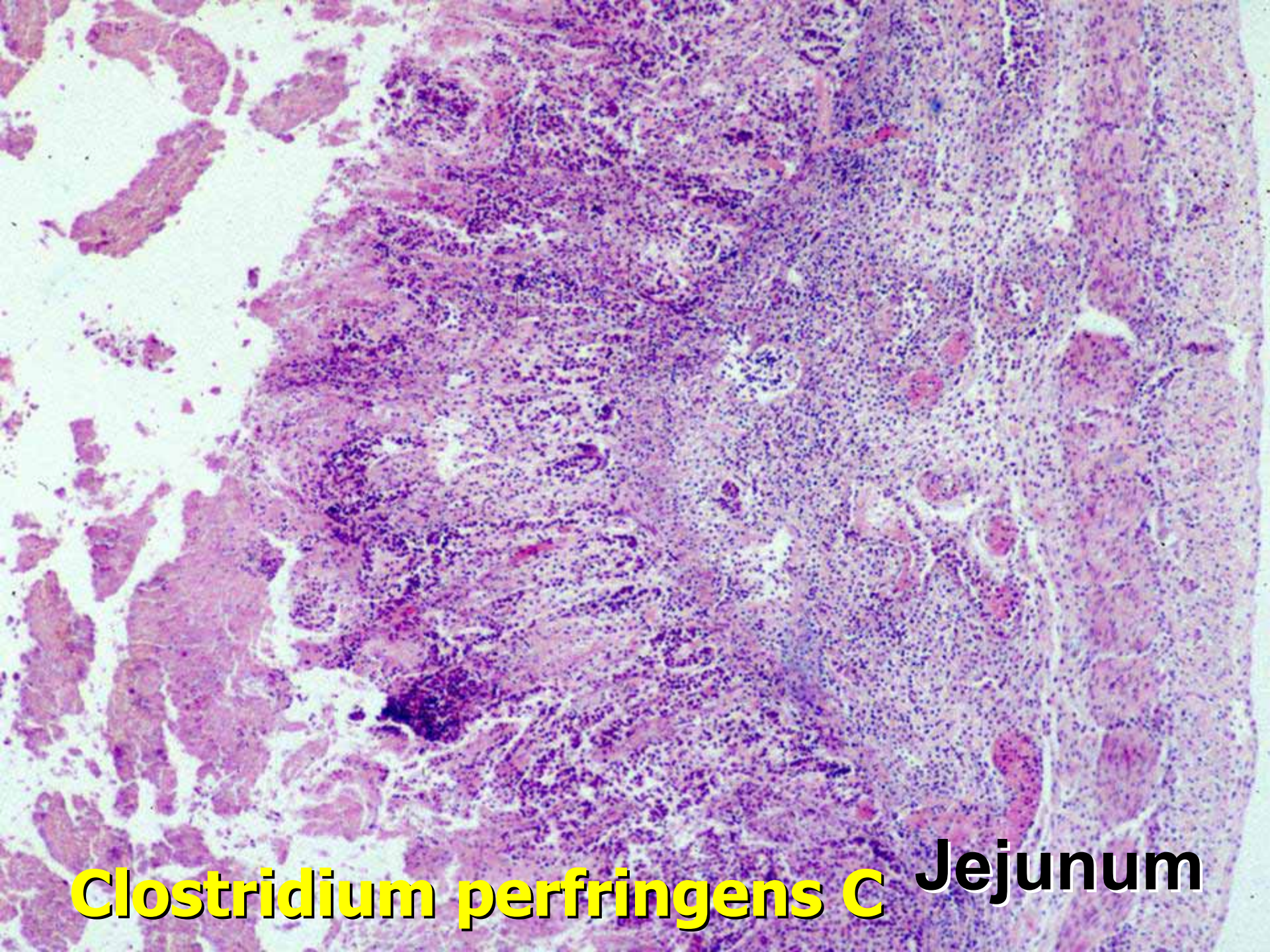

Clostridium perfringens C


Clostridium perfringens C

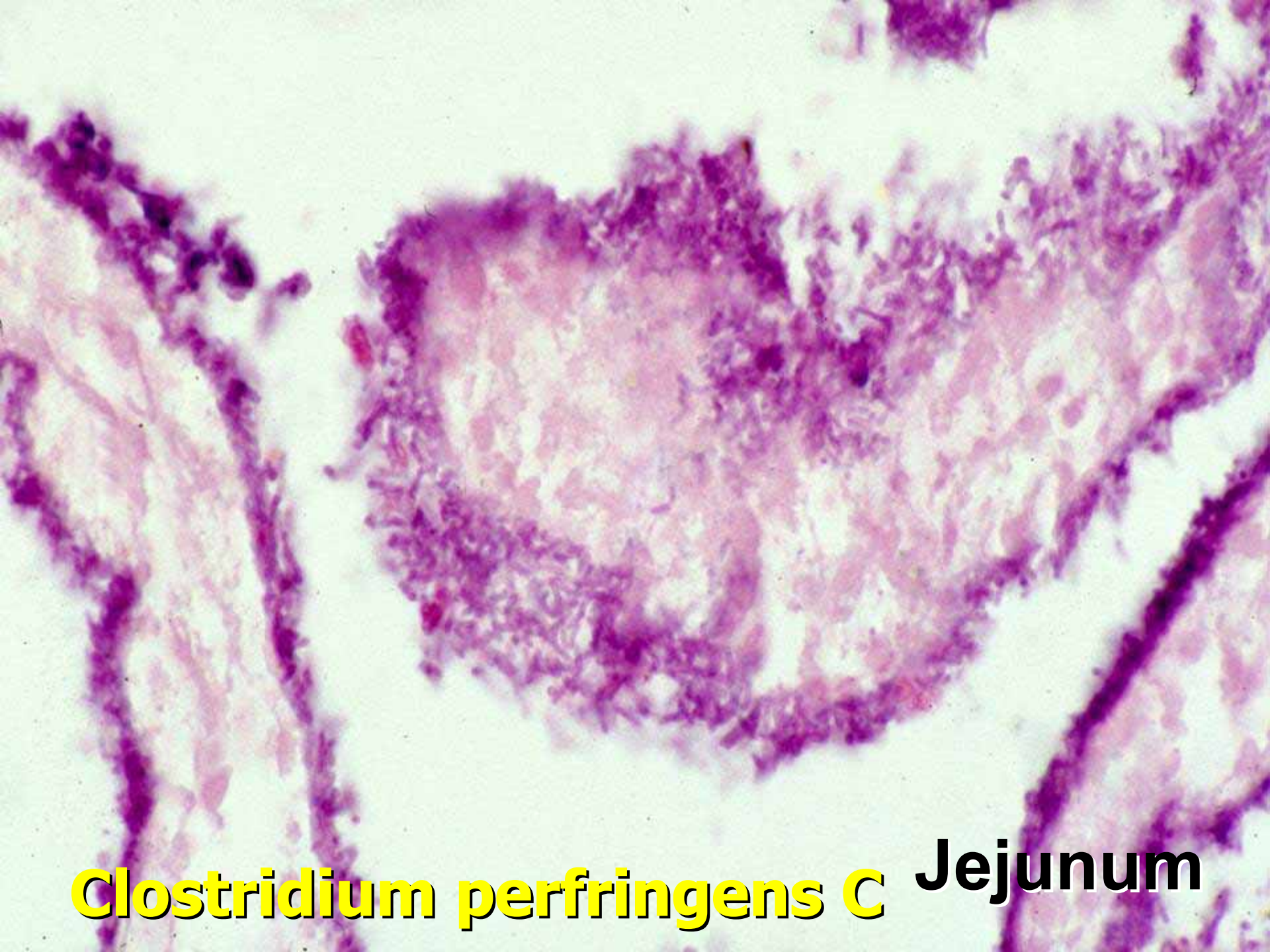

Clostridium perfringens C


Clostridium perfringens C


Clostridium perfringens C
Jejunum


Clostridium perfringens C
Jejunum

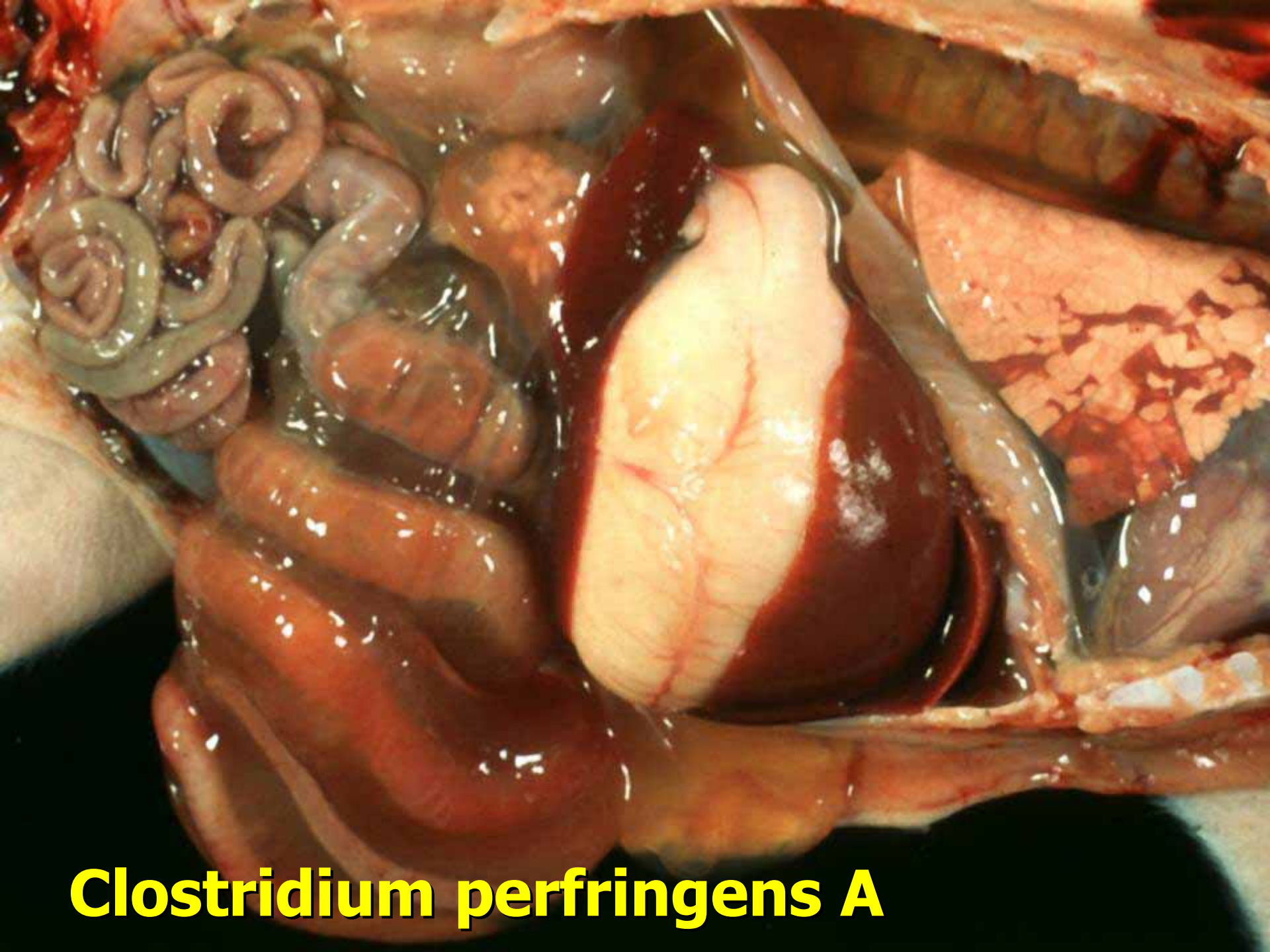

Clostridium perfringens C
Jejunum


Clostridium perfringens C
Jejunum

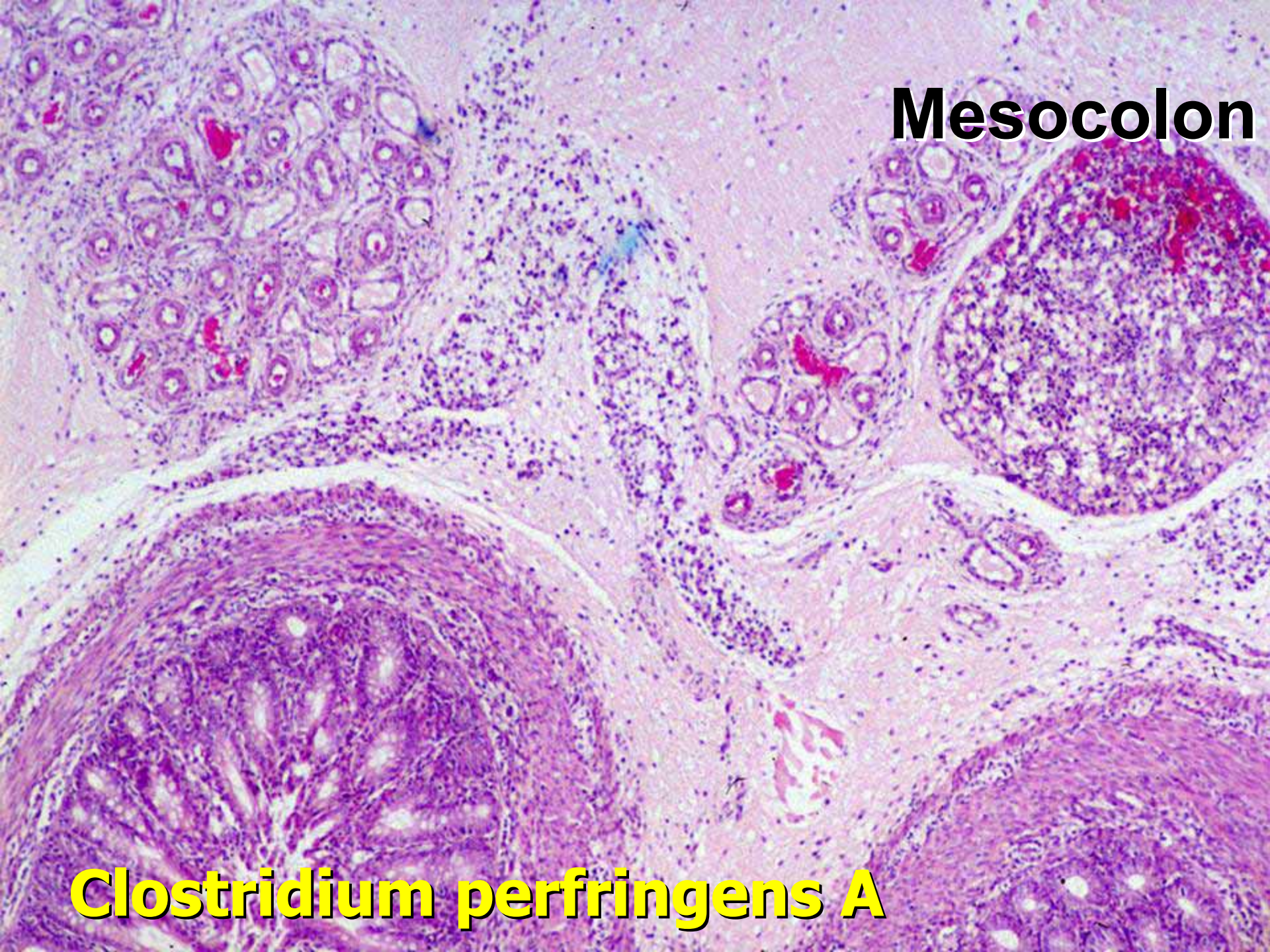

Clostridium perfringens C
Jejunum

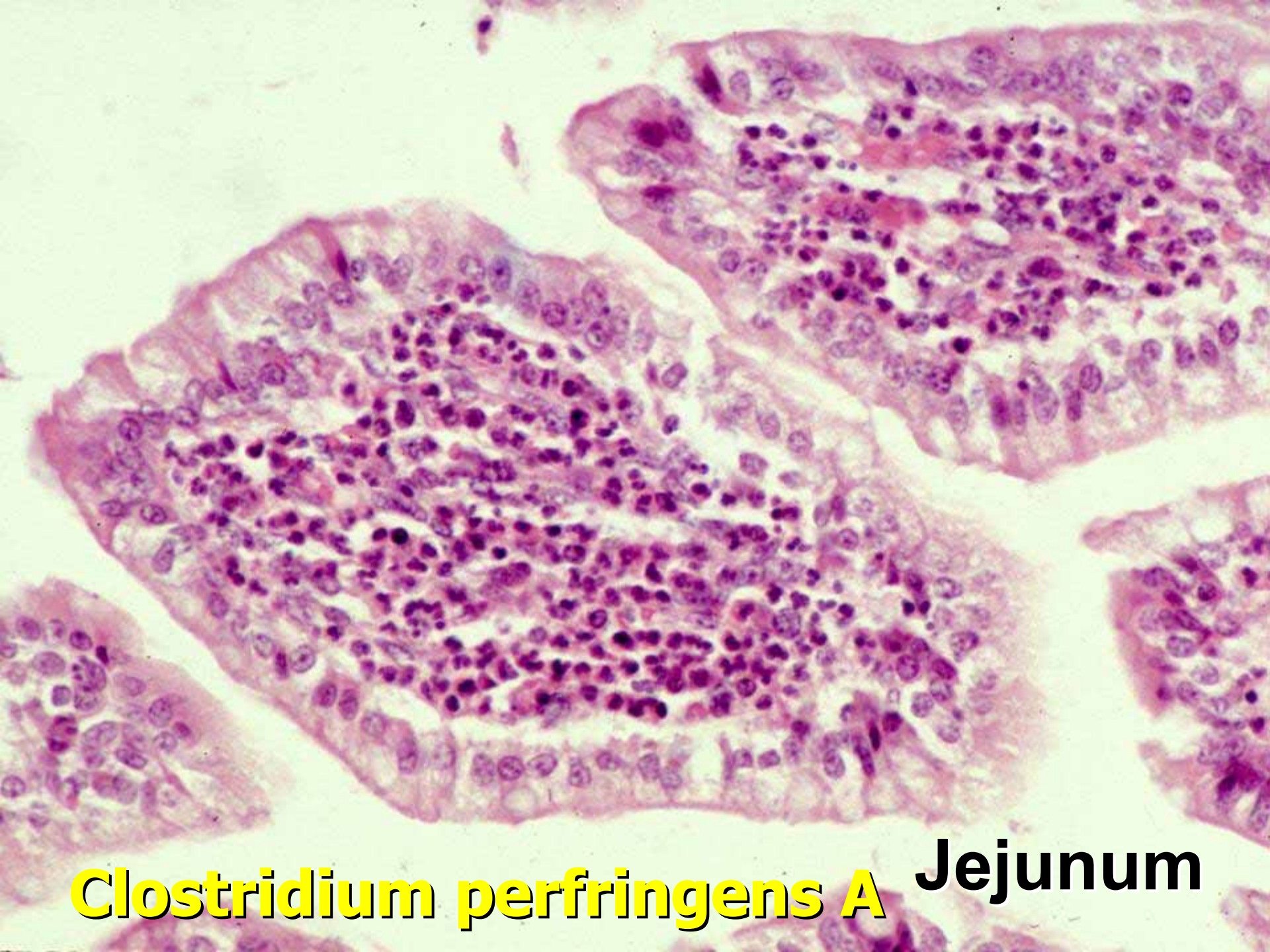
Clostridium perfringens type A

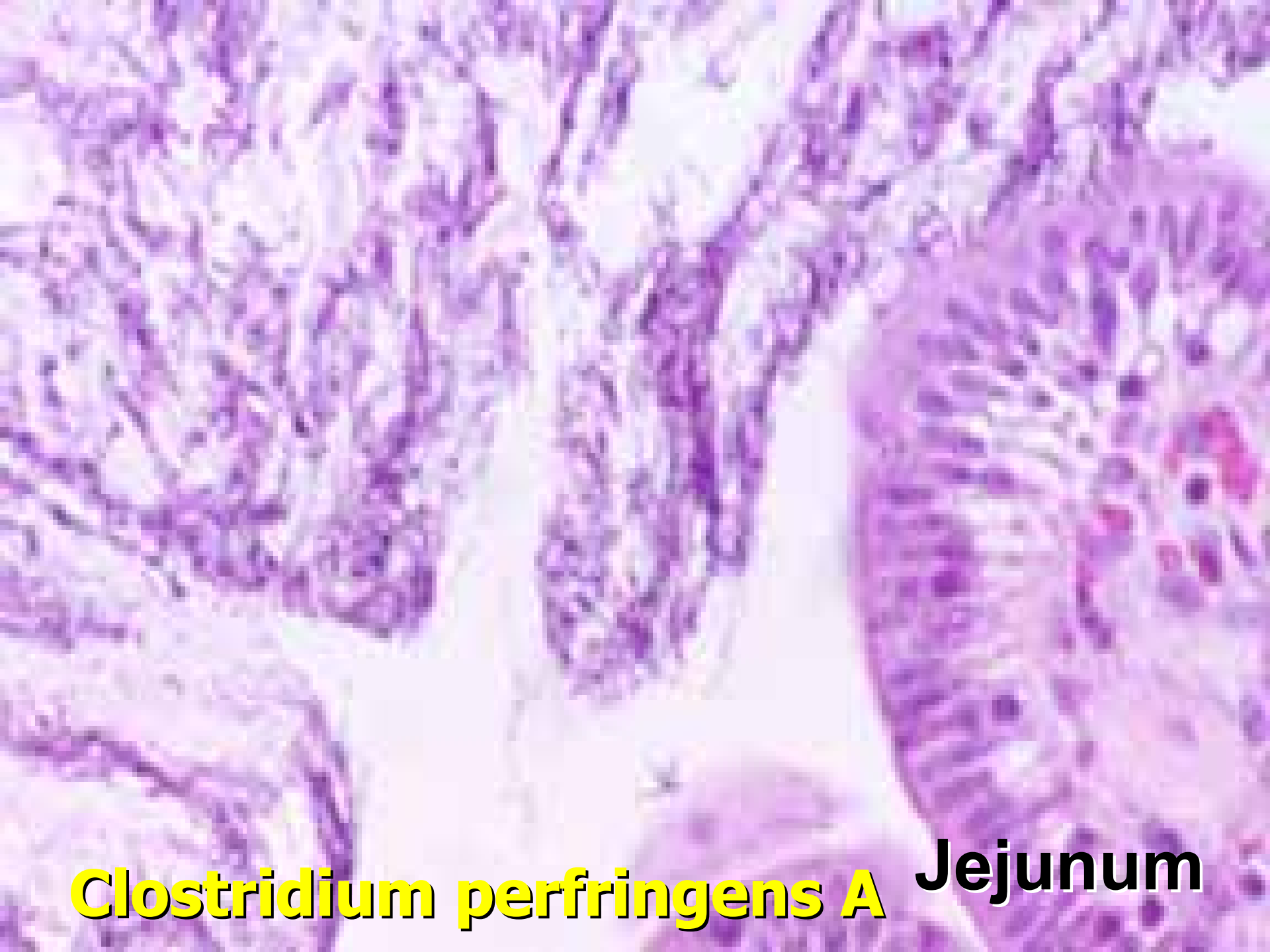
- 1 - 4 days of age
- high morbidity, low mortality
- piglets exposed orally to mixed type A population from sow feces
- virulent strains become dominant in stomach
- shower remainder of gut, accumulate in lumen
- diarrheagenic effect produced without intimate mucosal association
- diarrheic effect on small intestine, jejunum
- no/minimal gross or microscopic lesions
- enterotoxin? CPA? CPB2?
- other virulence attributes?

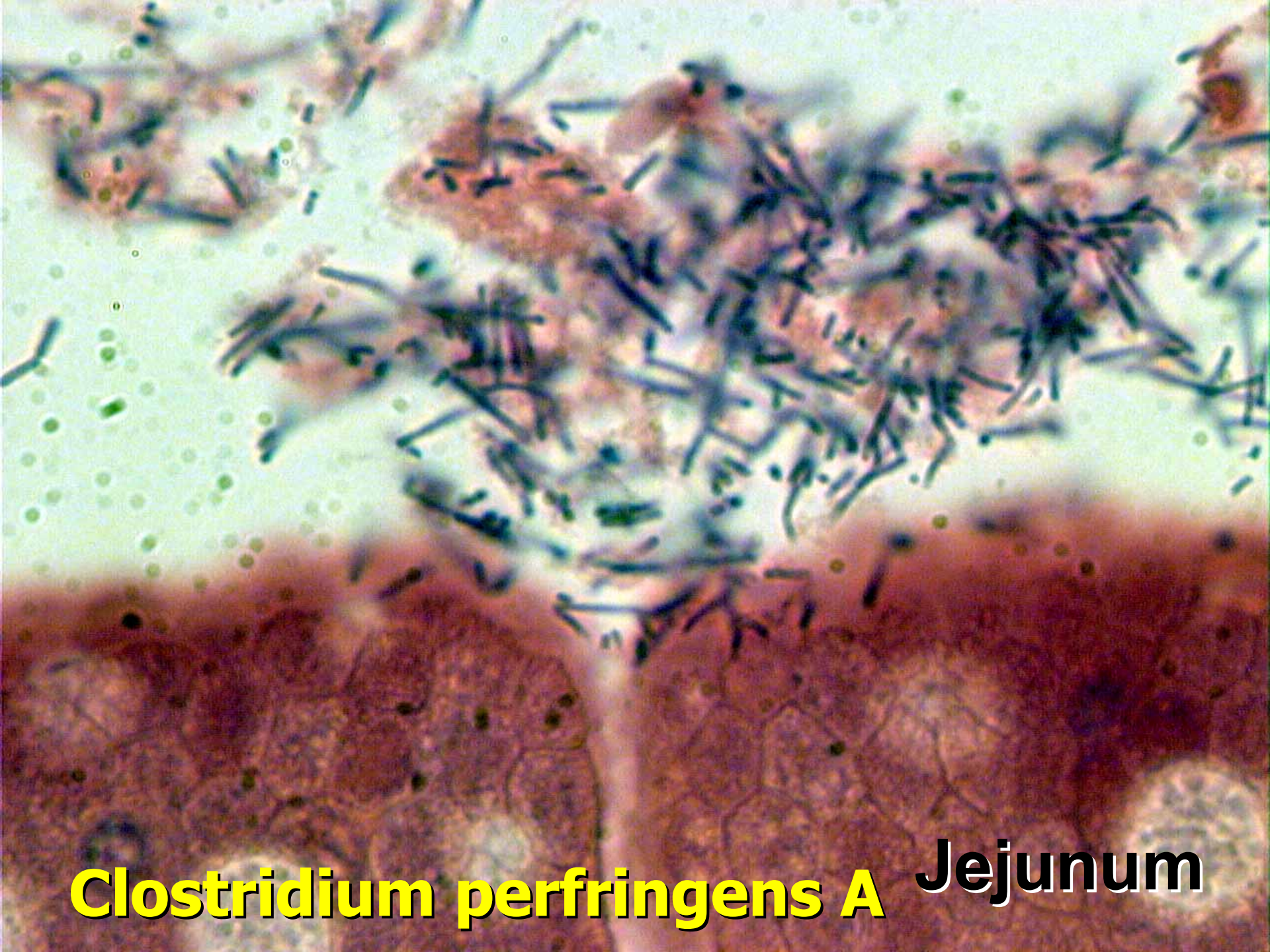

Clostridium perfringens A


Clostridium perfringens A


Mesocolon
Clostridium perfringens A


Clostridium perfringens A
Jejunum


Clostridium perfringens A
Jejunum


Clostridium perfringens A
Jejunum


Clostridium perfringens A
Jejunum

Clostridium difficile

- neonates; startup herds, low parity dams
- high mortality
- average 10% loss of condition at weaning, not recovered in grow-out period
- pasty, yellow colonic contents; constipation, obstipation
- gross lesions
  - ascities, subcutaneous edema
  - mesocolonic edema, necrotizing colitis
- microscopic lesions
  - erosive colitis w/ “volcanic” exudation

Diagnosis
Clostridium perfringens/difficile

- Jejunum, ileum, colon, cecal/colonic contents
  - tissues: tied off and chilled and in 10% NBF
  - cecal/colonic contents: frozen ASAP
- Tests
  - Anaerobic culture
  - Clostridium toxin genotyping by PCR: alpha, beta, epsilon, iota, endotoxin, beta-2
  - C. difficile toxin by antigen-capture ELISA


Clostridium difficile


Clostridium difficile


Clostridium difficile

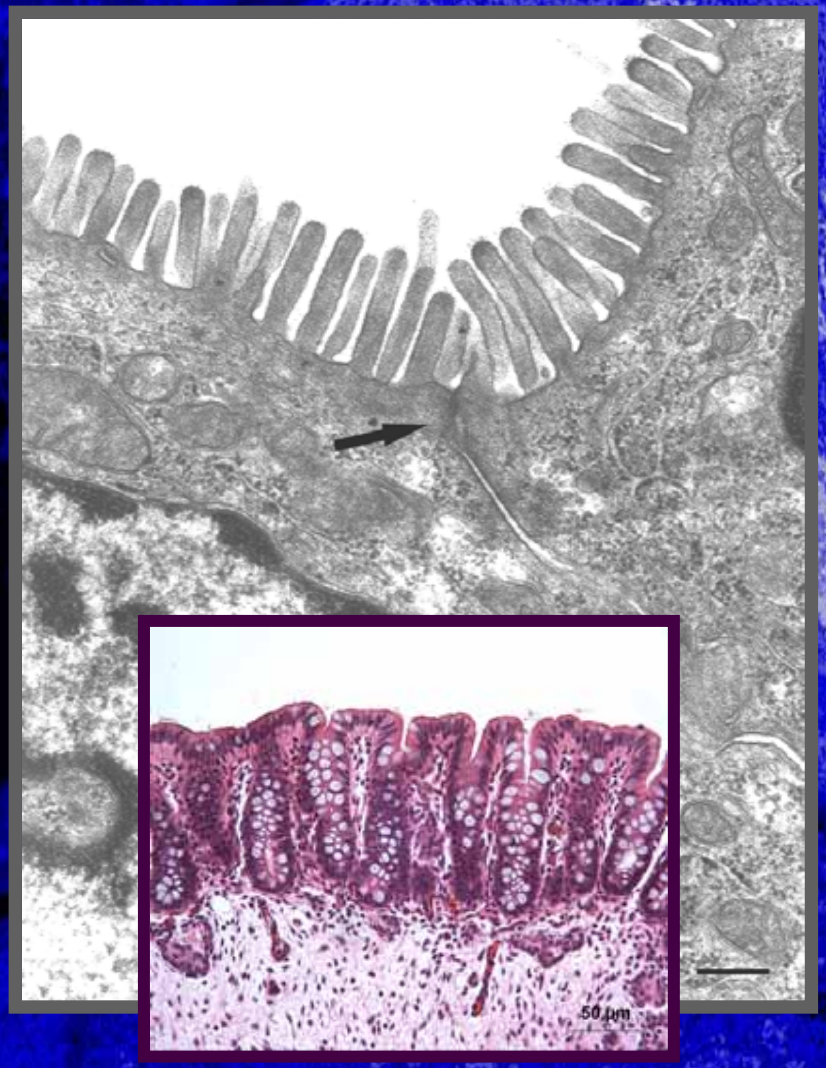

Clostridium difficile


Clostridium difficile
Jejunum

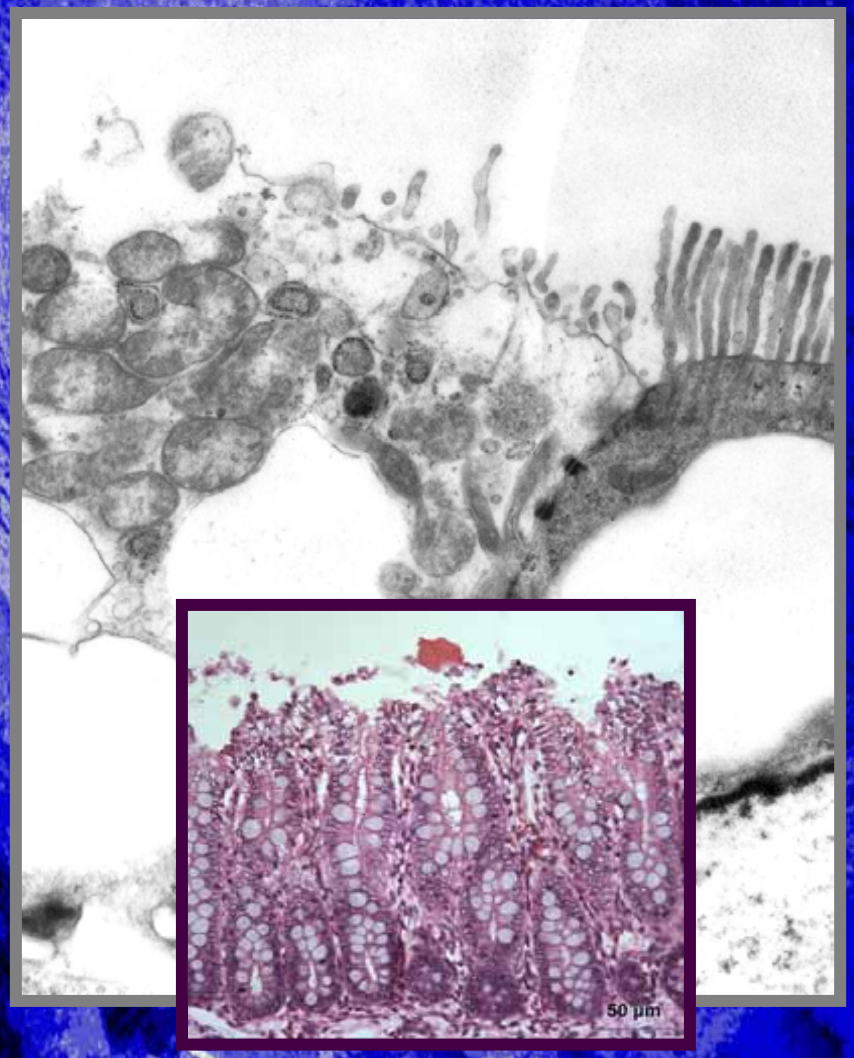

Clostridium difficile
Jejunum


Clostridium difficile
Jejunum

Lesion development is toxin-mediated

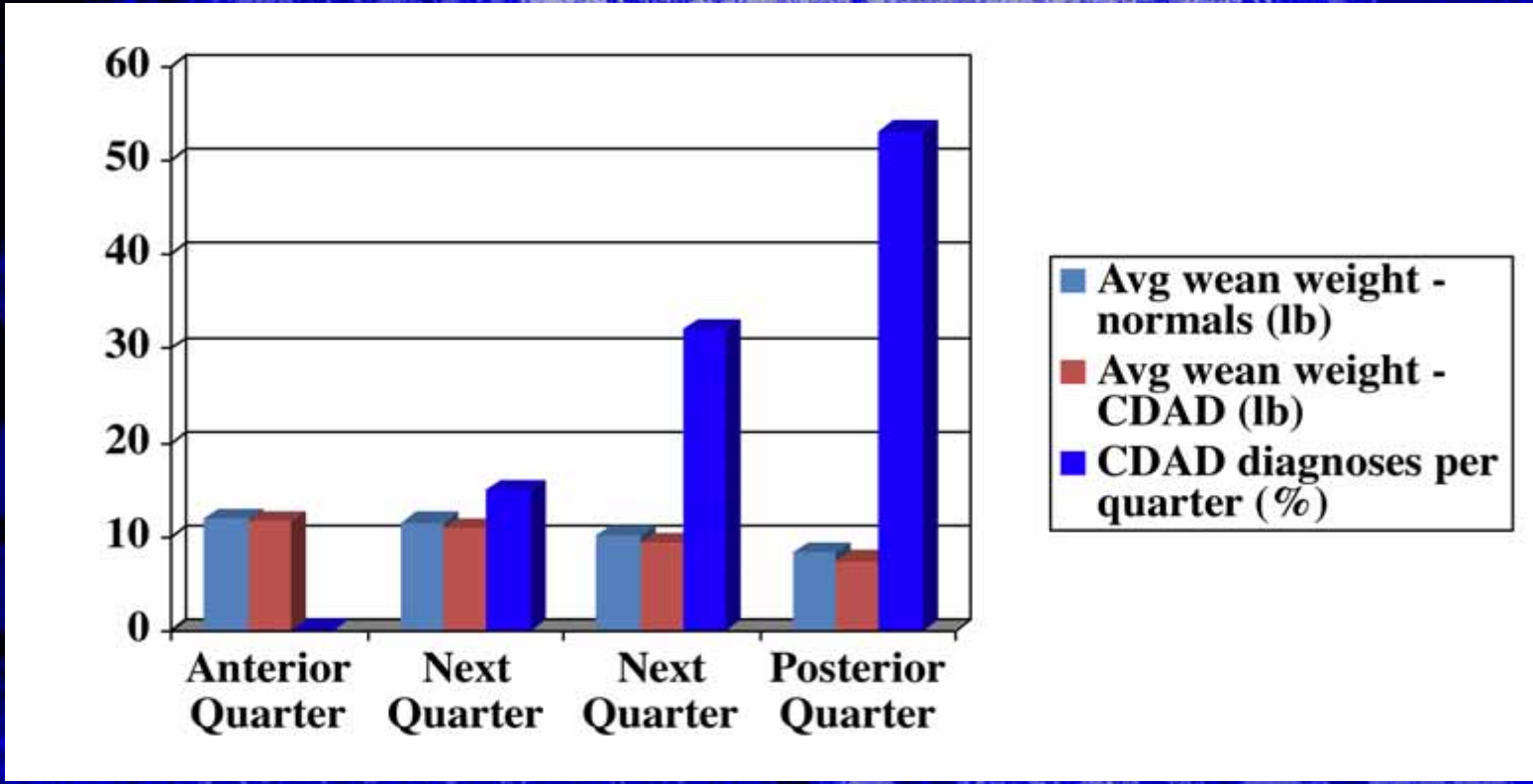


Normal



Toxin A

CDAD, Weight Gain, and Teat Position



"Meeeeowwww ... did I say it right?"

Proliferative Enteritis

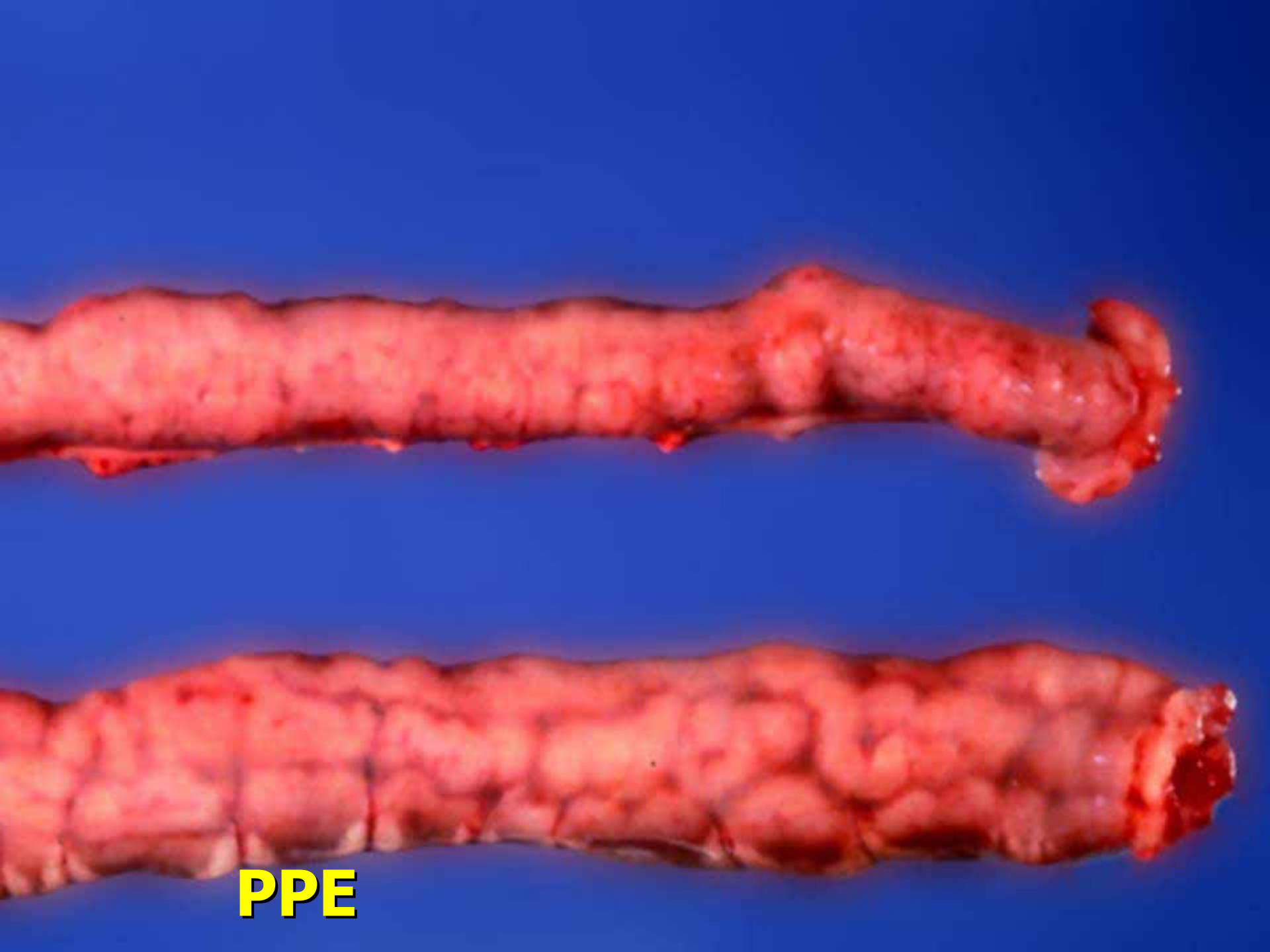
- *Lawsonia intracellularis*
- obligate intracellular bacterium
- pigs, foals, foxes, ferrets, hamsters, rabbits, guinea pigs, dogs
- more common in late finishing/new breeding stock
- brownish bloody diarrhea
- Proliferative ileitis, adenomatosis, necrotic ileitis and/or typhylocolitis, hemorrhagic enteritis
- PHE more common in high health status herds
- Necrotic enteritis +/- colitis most common 3-10 weeks after entering grower - green watery diarrhea

Proliferative Enteritis

- lesion: ileum, cecum, proximal 1/3 of colon
  intracellular proliferation in enterocytes
  crypt hyperplasia and dysplasia
- Koch's postulates fulfilled:
  - in SPF pigs
  - in gnotobiotic pigs with addition of *Bacteroides vulgaris* and *E. coli*
- experimental disease:
  - dose dependant
  - incubation: 2-3 weeks
  - intermittant shedding for at least 8 weeks

Proliferative Enteritis

- Diagnosis
  - Gross lesions
  - Microscopic lesions
    - Characteristic proliferative lesion
    - Silver stain - intracellular bacteria
    - IFA - monoclonal antibody
  - DNA probe of feces (10^7/gm)
  - PCR: 10^1 organisms/gm ileal mucosa
    10^3 organisms/gm feces
  - Serology
    - IFA - 26d p.i. 6/15 seroconverted
- Treatment and Control
  - SEW at 14d did not eliminate
  - Pigs with 100g Tylan in feed still had fecal shedding


PPE