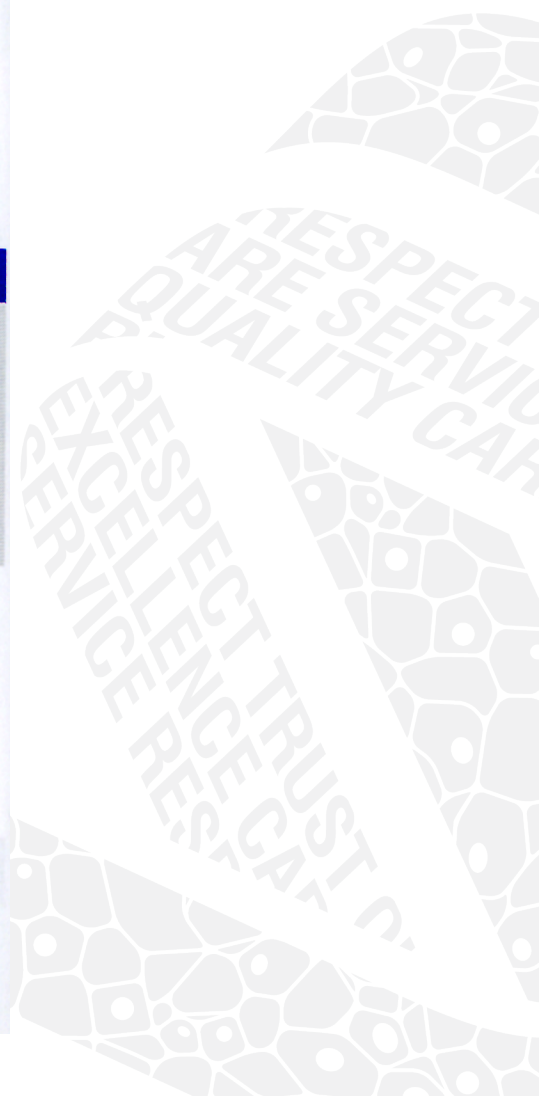
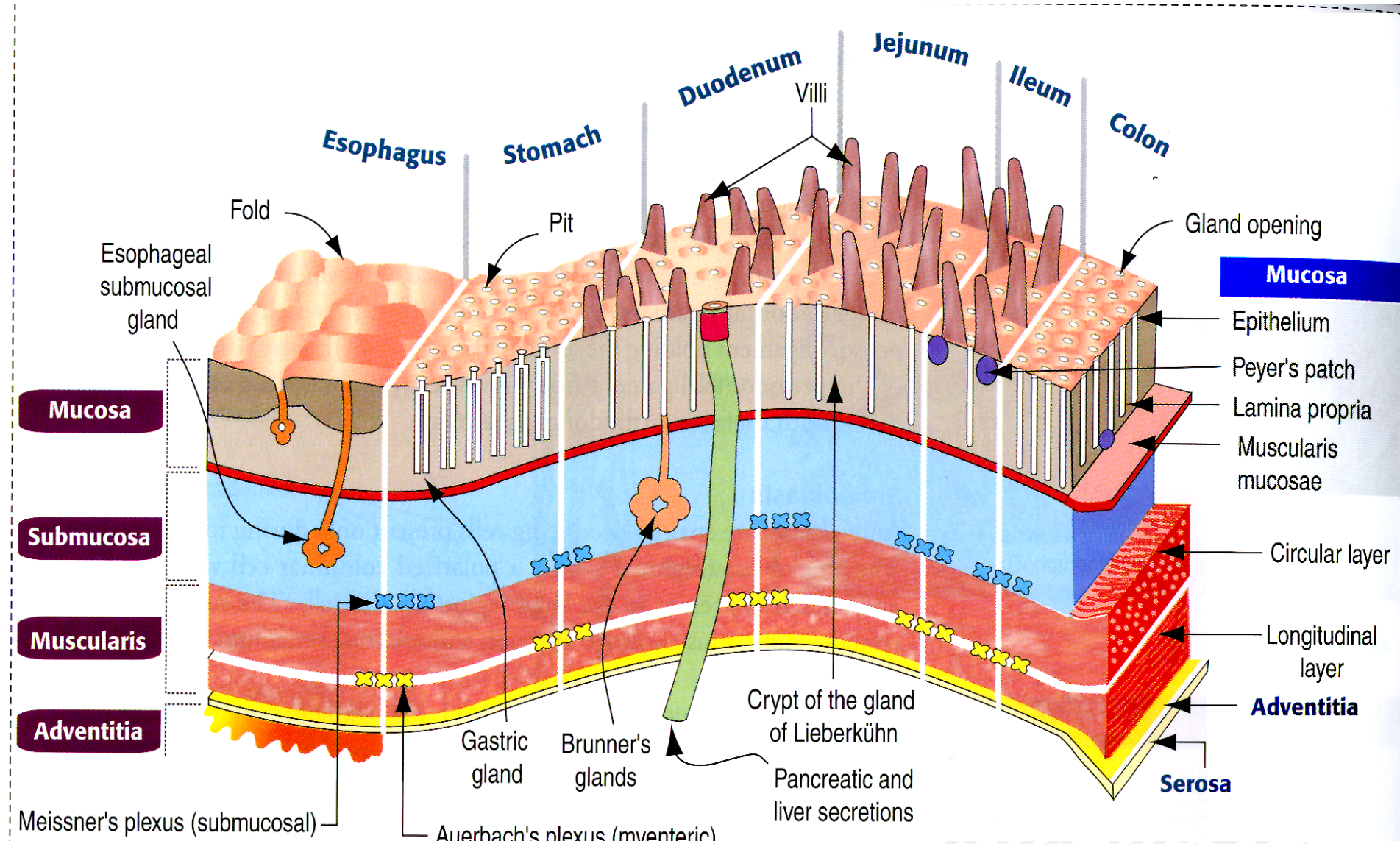


MVZ DÜREN
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Molekulare Diagnostik

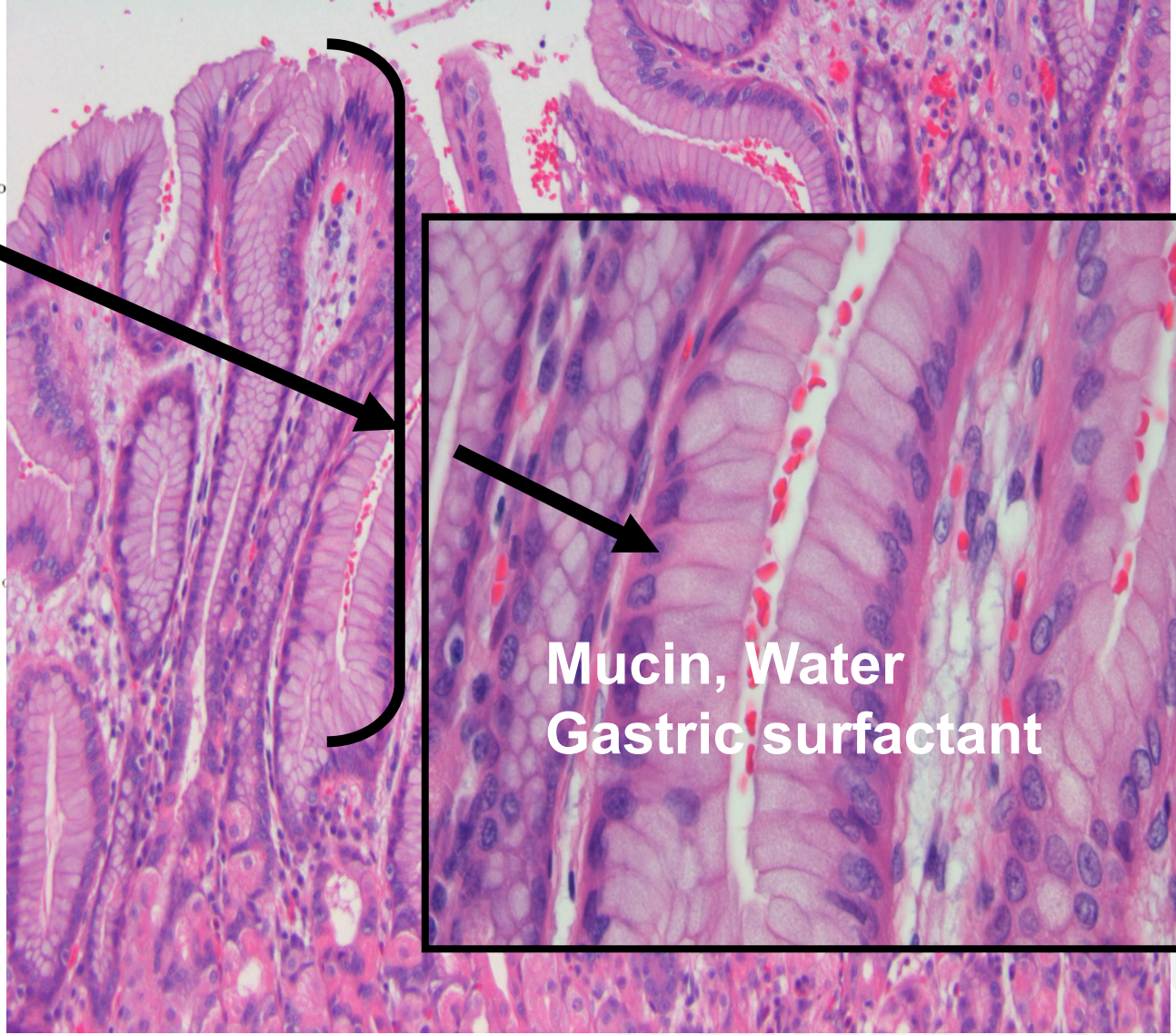
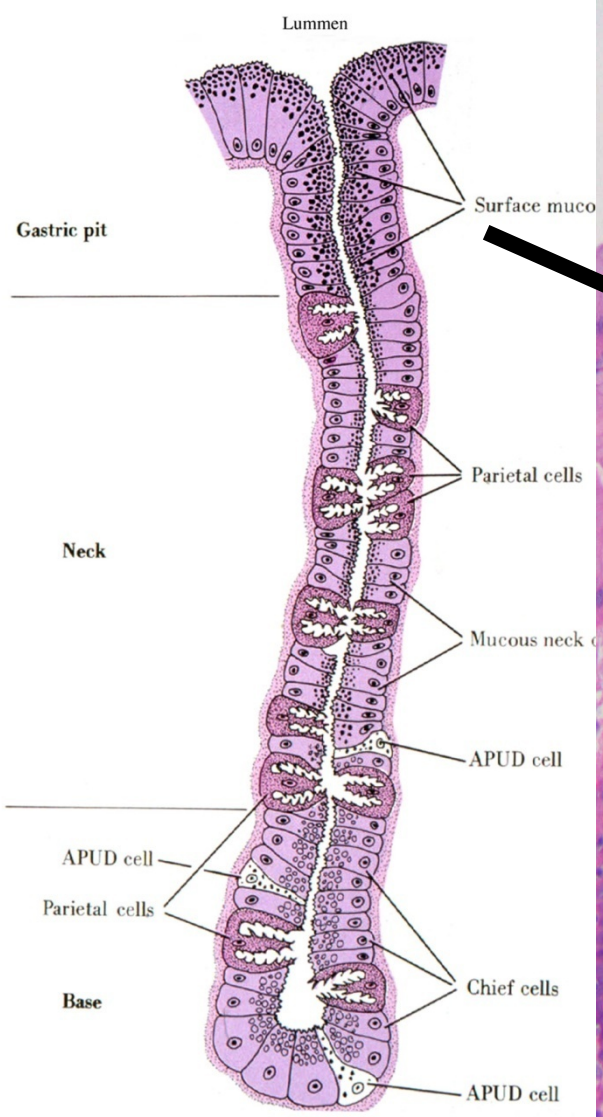
Magen und HP



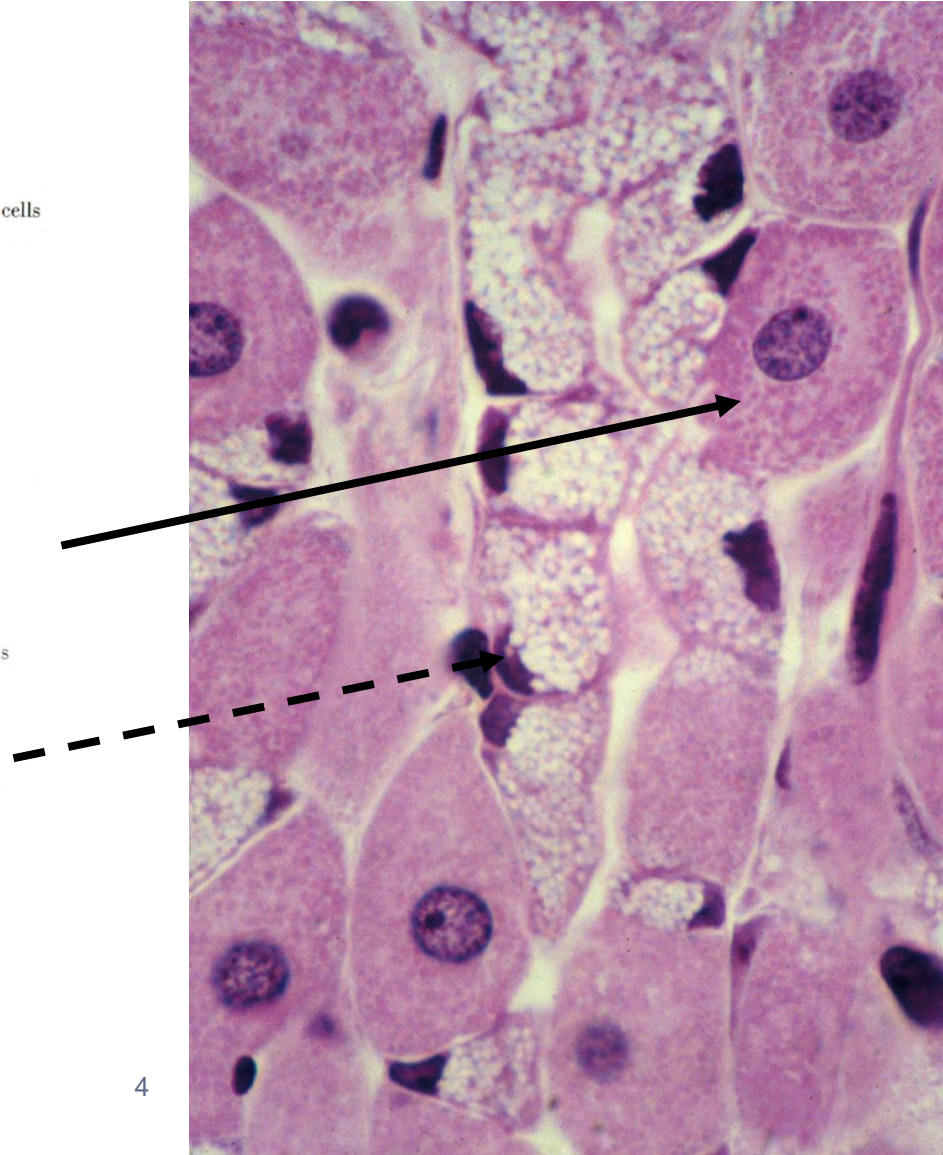
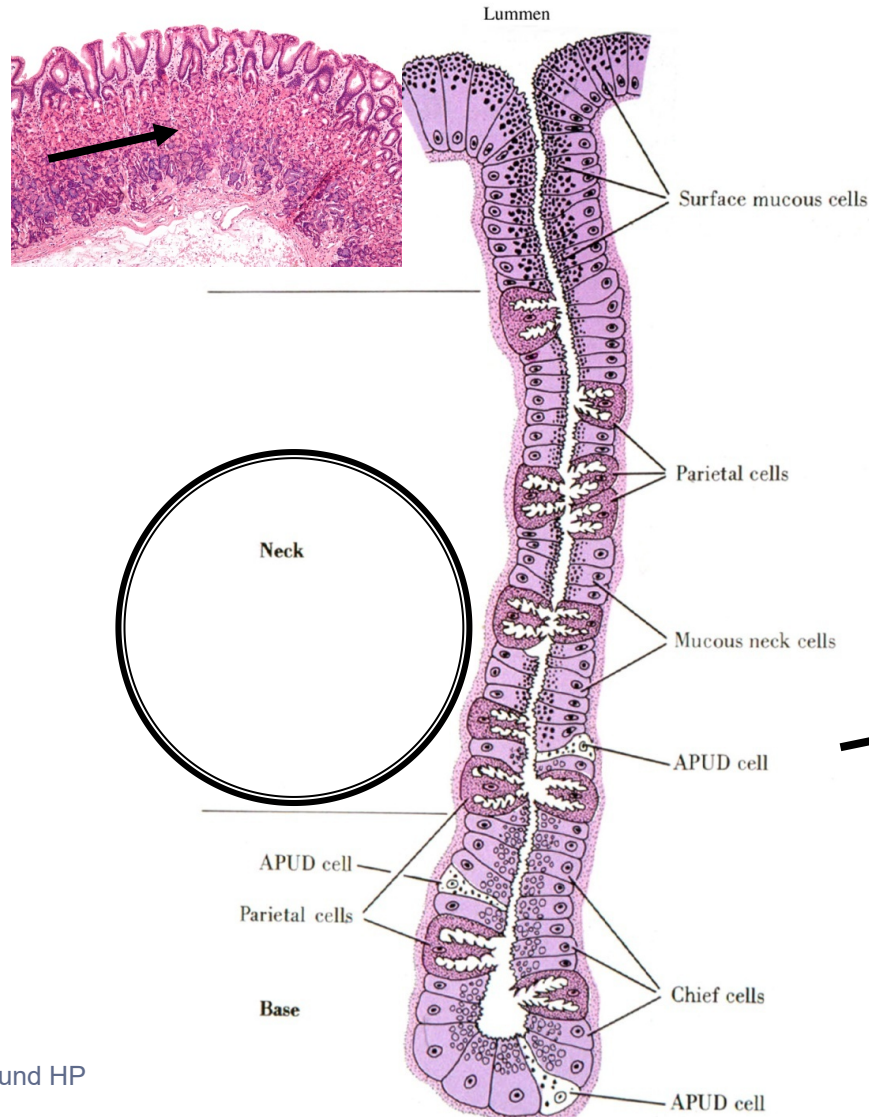
Architecture of the tractus



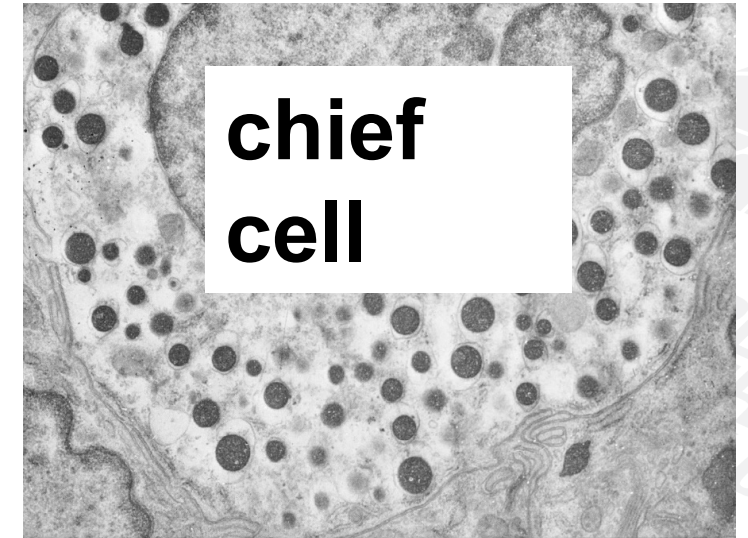
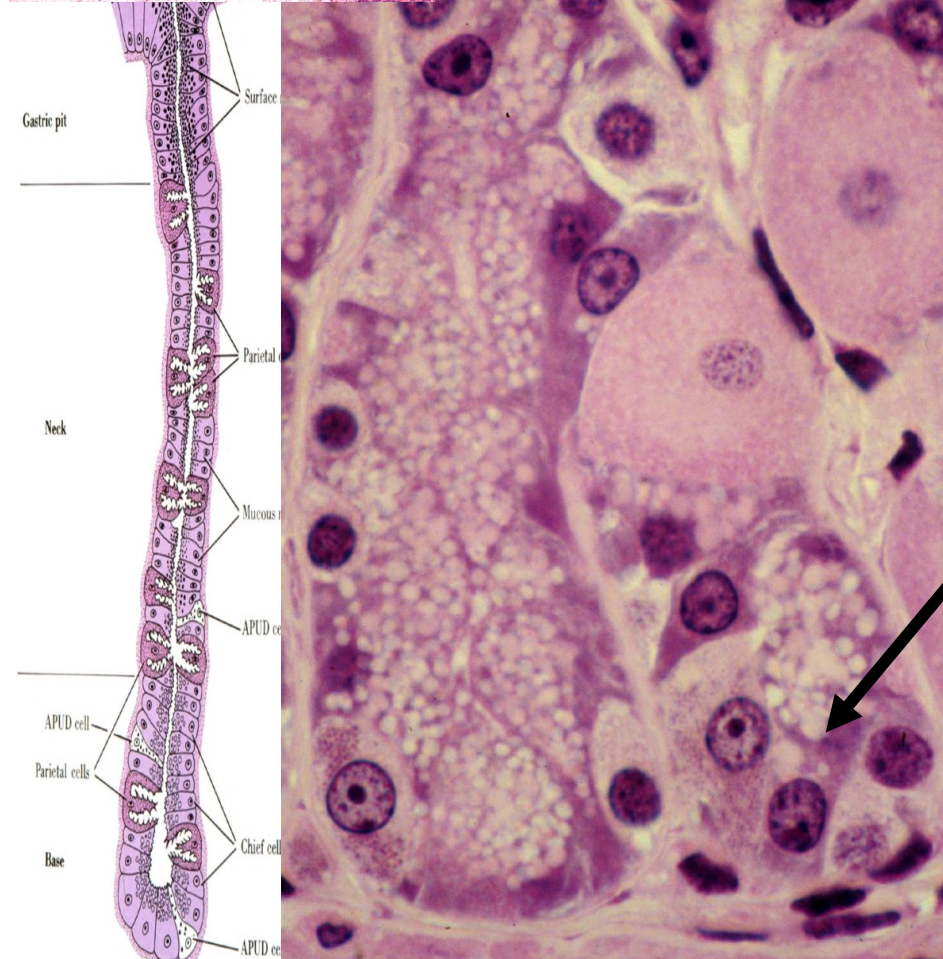
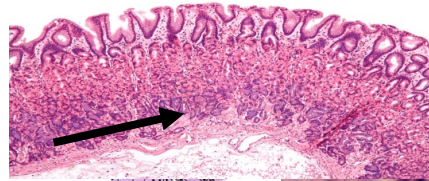
Surface mucous cells



Parietal (oxyntic) cells and mucous neck cells



Base of the glands: chief cells (hoofdcellen)



argentaffine cell

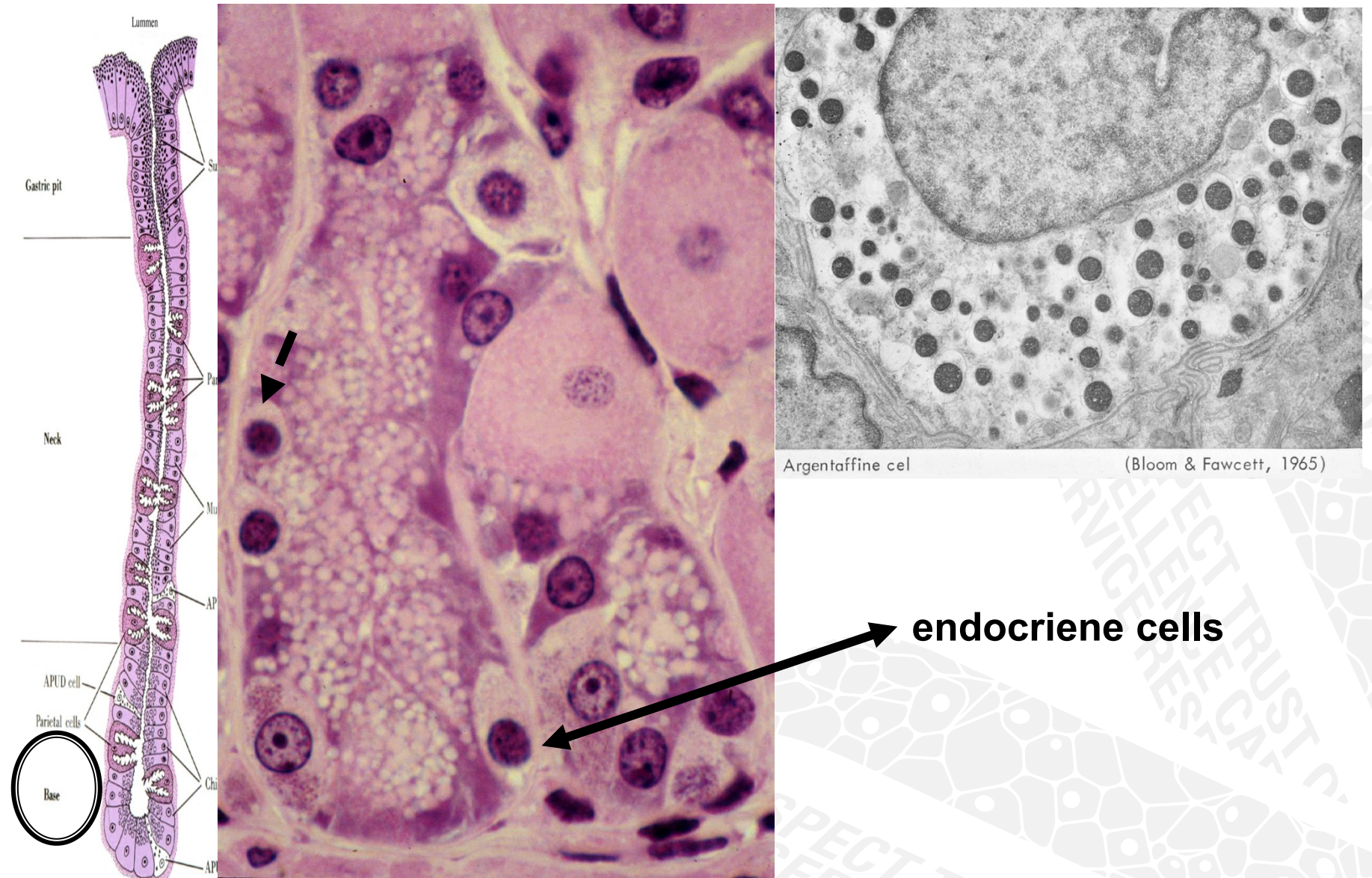
(Bloom & Fawcett, 1965)

Mainly lower regions of the glands

Rich in endoplasmic reticulum
Storage of **pepsinogen**
HCL converts the inactive pepsinogen to **pepsin**: digestion of proteins

Lipase and hormones (leptin)

Base of the glands: endocrine cells (APUD)



Gastric acid secretion: proton pump on parietal cells

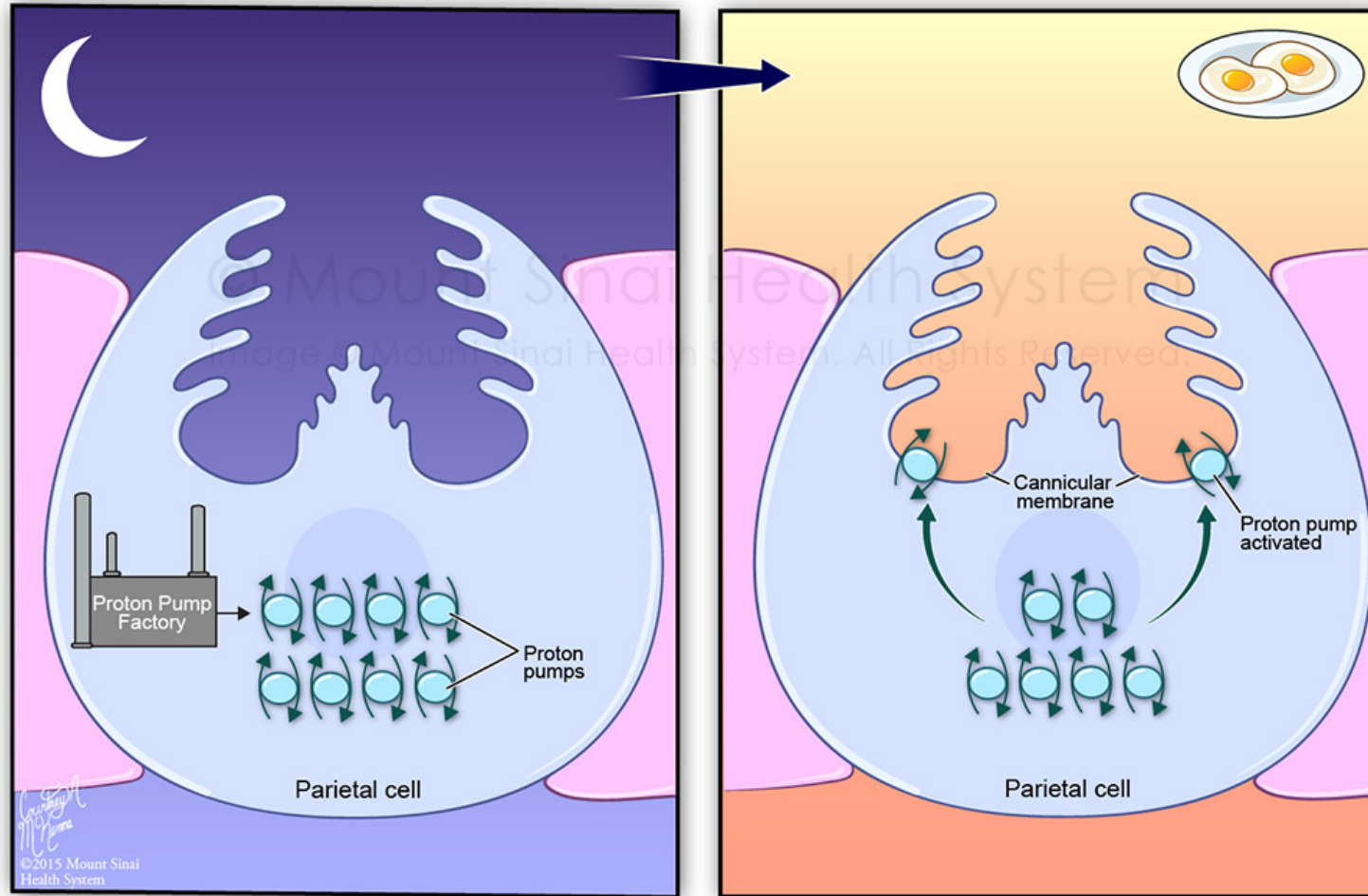
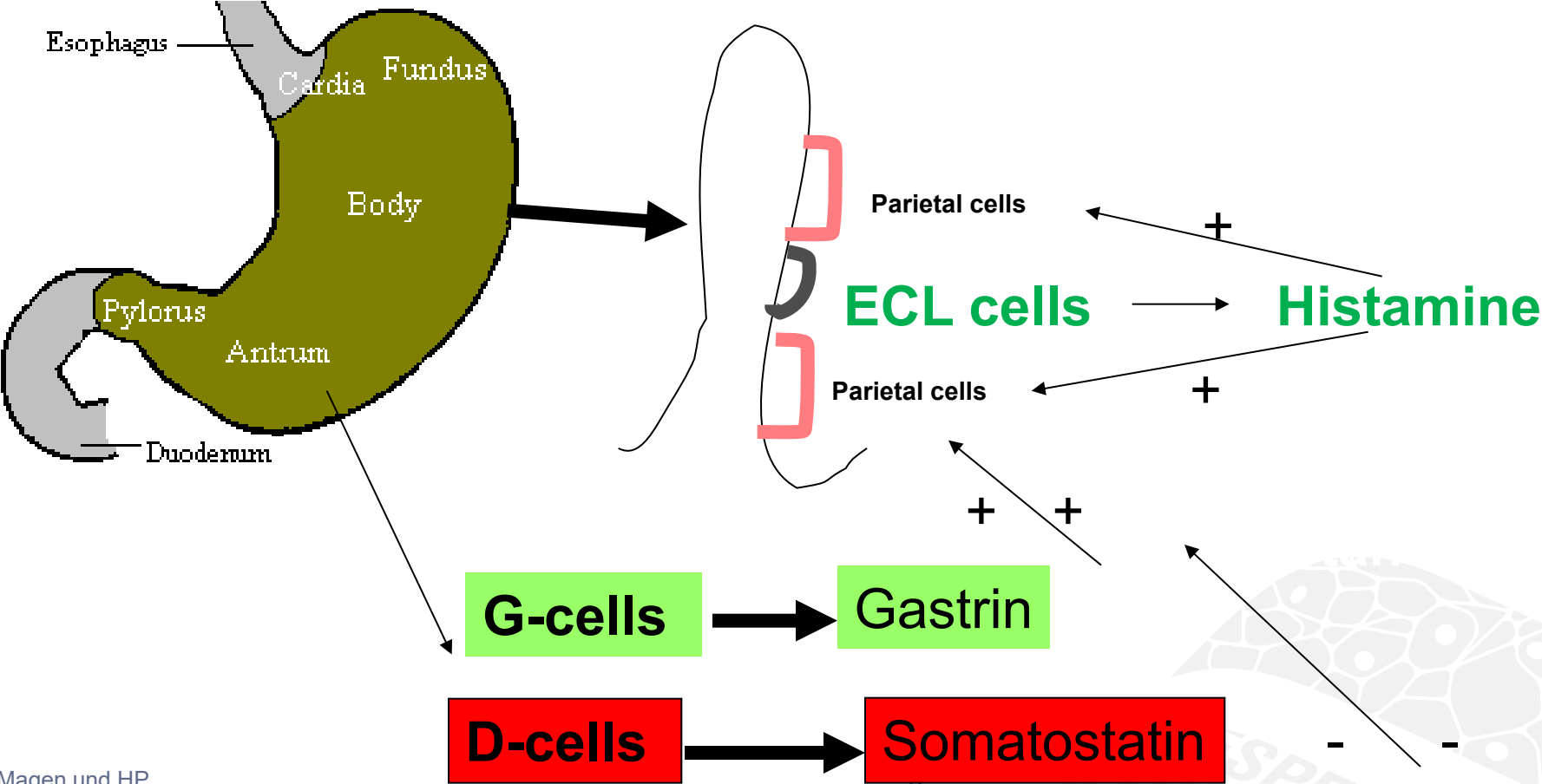


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Gastric acid secretion: endocrine cells



- Acid: PH 1-3
- Pepsin: Protease

Highly aggressive

Protectieve mechanismen

Regulation of acid production:
G- and D-cells

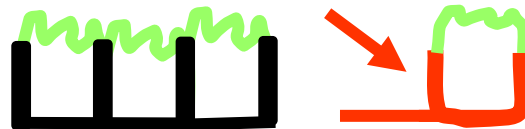


Feedback to the parietal cells



Mucosal barrier

Epithelial surface cells
Apical membrane resistance

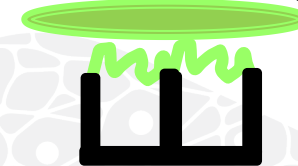


Acute repair



Stem cells
Regeneration

Mucoid cap



Protective mechanisms: cardia and antrum glands

Cardia:

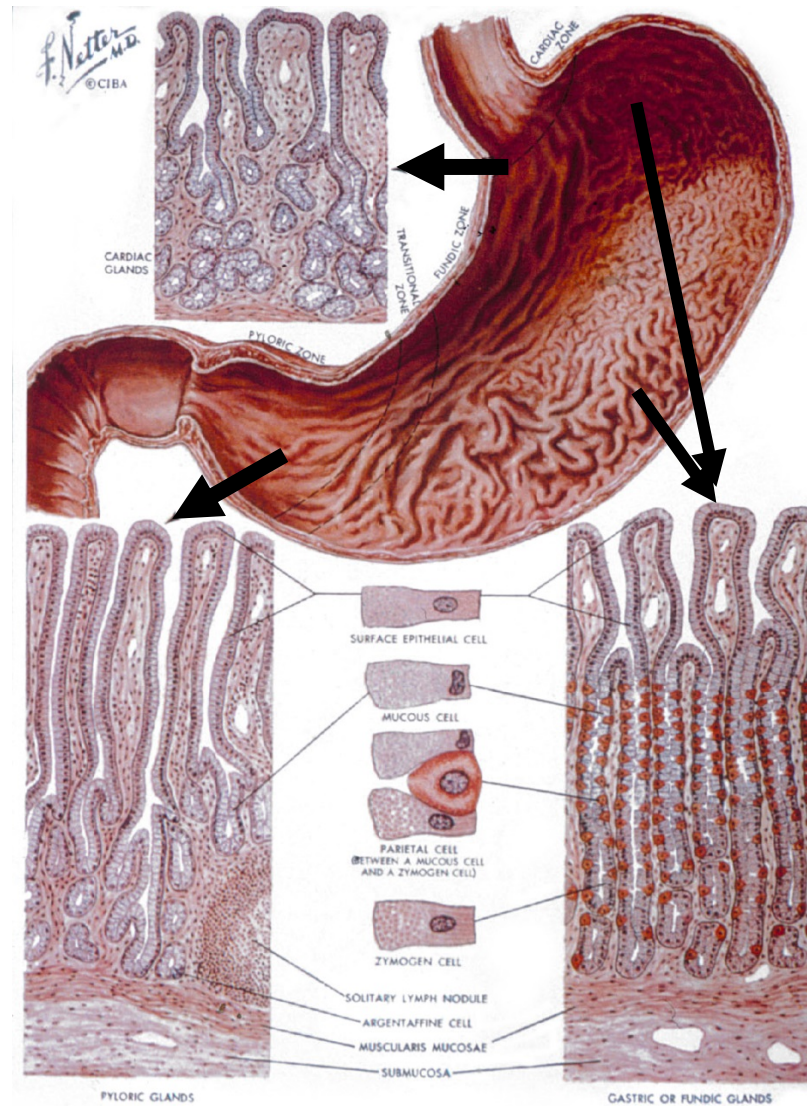
mucus

HCL neutralization

Antrum/Pylorus:

mucus

HCL neutralization



Fundus/body:

acid & pepsin

Digestion

Gastritis: Helicobacter pylori

maagzuurresistente bacterie
overleefd goed in de zure omgeving
in tegenstelling tot andere bacteriën

Toxic substances

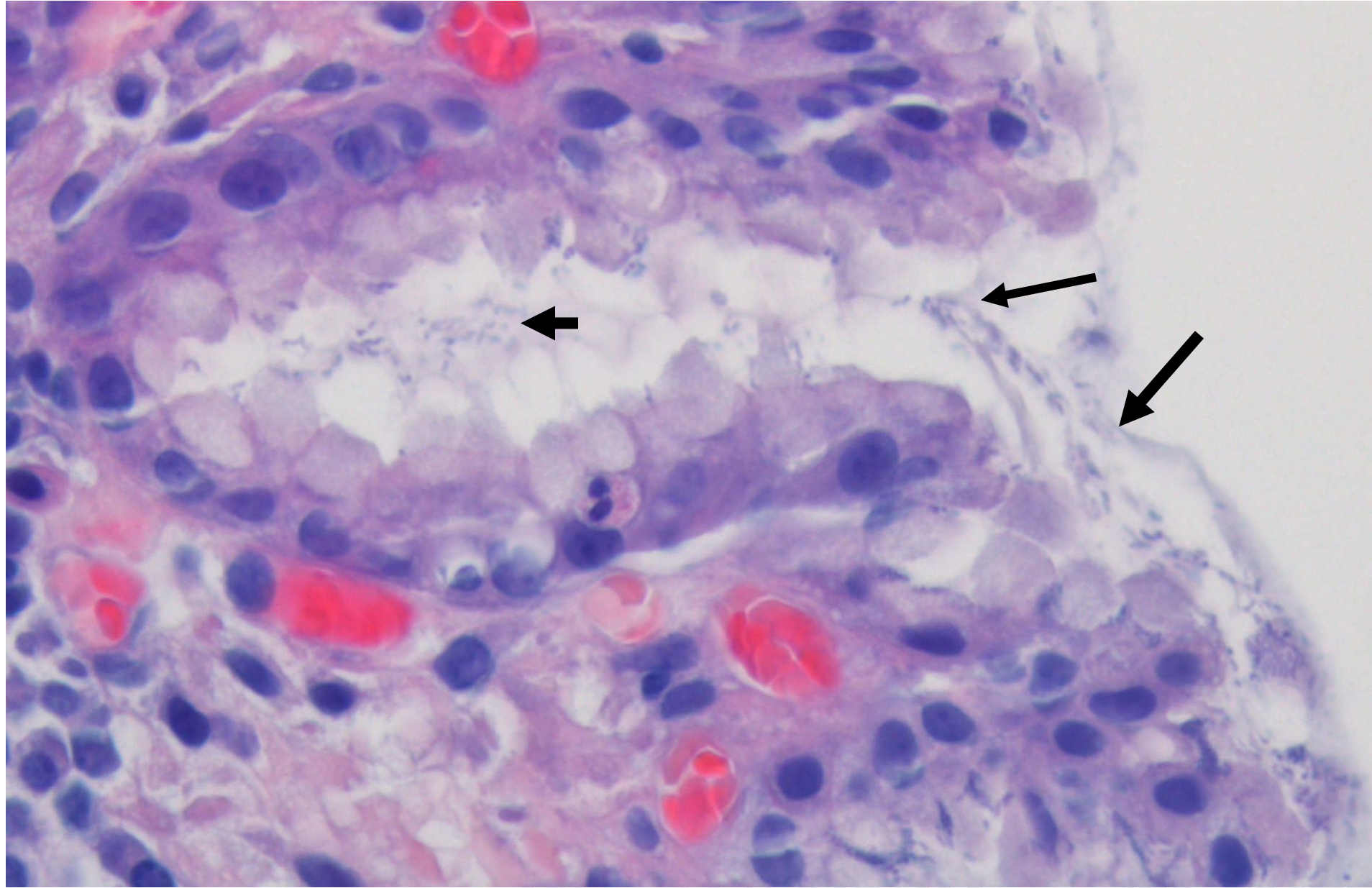
**Inflammation: acute (eosinophils/neutrophils)
chronic (lymfocytes, plasma cells)**

Gastrin ↑

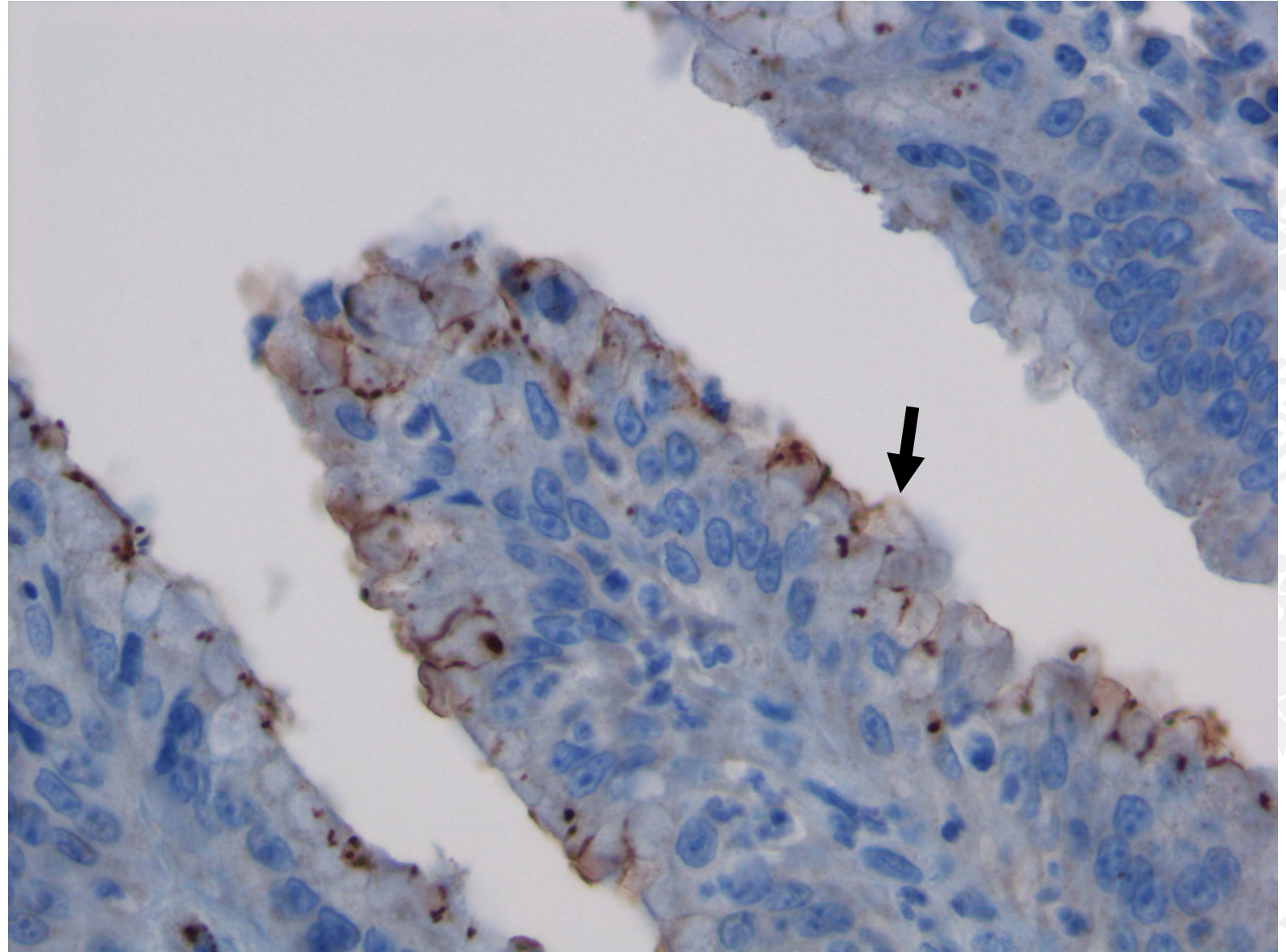
**Disruption of mucosal barrier
Epithelial damage**



Protective mechanisms: cardia and antrum glands

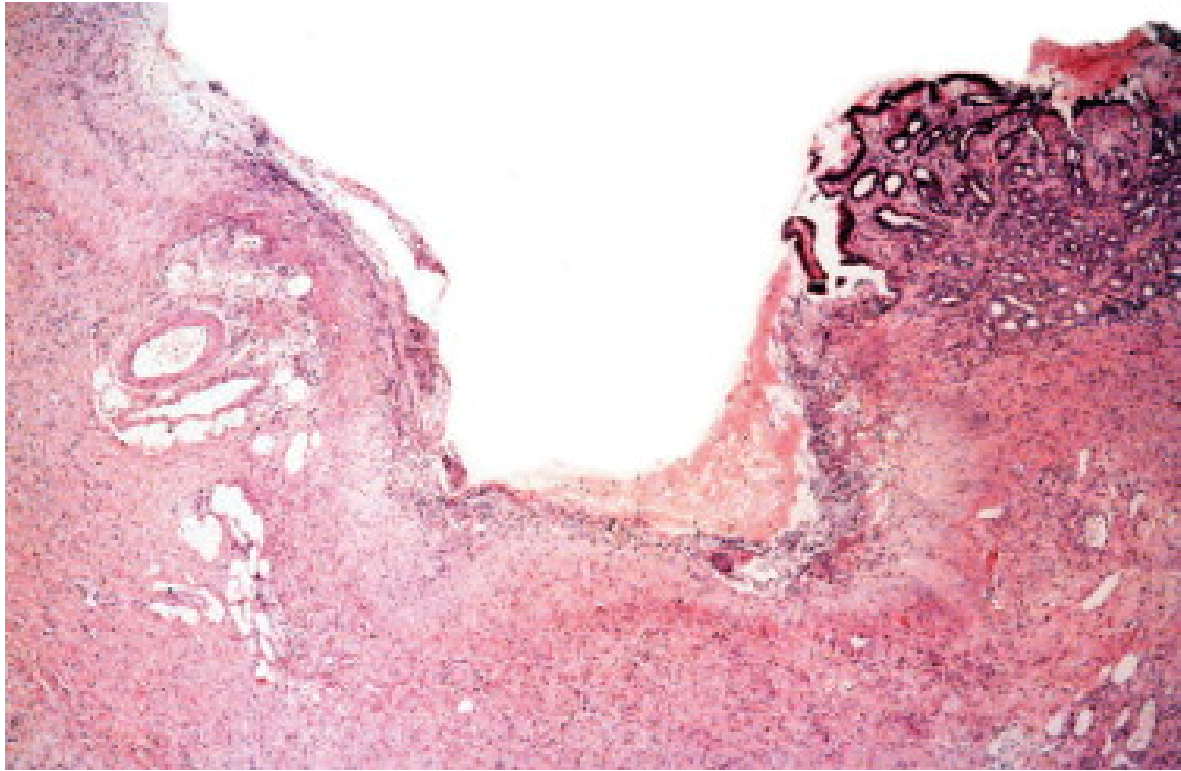


Helicobacter pylori staining



Gastric ulcer

gastric **ulcer** is defined by the loss of mucosa (including muscularis mucosae) due to inflammation



Long term effects of H pylori

Atrophic gastritis

- Gastric atrophy: loss of gastric glands
- Result of inflammation



- **Less acid**
(hypochlorhydrie leads to diarrhea)
- **less intrinsic factor**
(leads to anemia)



H pylori and its discovery

- [Ann Med.](#) 1995 Oct;27(5):565-8.
- **Helicobacter pylori in peptic ulcer: have Koch's postulates been fulfilled?**
- [Marshall BJ.](#)
- **Abstract**

- This brief review considers whether or not Koch's postulates have been fulfilled for *Helicobacter pylori* and peptic ulceration. The histological features of peptic ulcer disease in man are active chronic gastritis with antral predominance, duodenal gastric metaplasia and active duodenitis. Other features are hyperpepsinogenaemia, relative postprandial hypergastrinaemia and basal acid hypersecretion. The macroscopic features are duodenal bulb ulceration or lesser curve and antral gastric ulceration. At present, gastric colonization with *H. pylori* has been produced in small animal species (rats and mice), but the infection is difficult to establish in immunocompetent animals, and histological gastritis is unconvincing. In larger animals the germ-free pig has been the most reliable model but the gastritis tends to be chronic with little activity. **The best examples of acute infection are in three 'self-administration' experiments in humans.** In these cases acute gastritis with hypochlorhydria developed which, when it converted to active chronic gastritis, tended to be asymptomatic. Either the circumstances were incompatible with ulceration, or the experiments were not continued for the many years necessary to develop peptic ulceration. It is concluded that only one of the many steps required for the development of peptic ulceration has so far been fulfilled, i.e. the ability of *H. pylori* to produce histological gastritis in a susceptible host.



Vielen Dank

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