

# How do we stomach our food?

## Part 2 – Control of gastric secretion

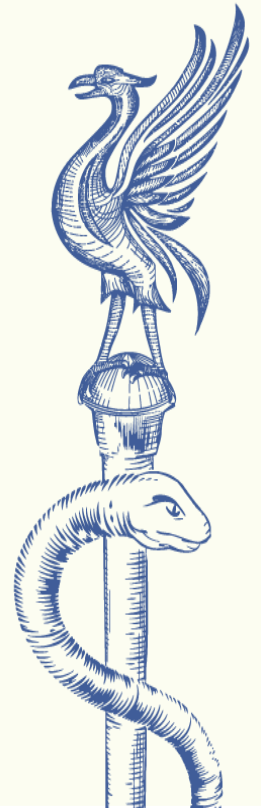
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<http://pcwww.liv.ac.uk/~bjcampbl/Indigestion%201.htm>



# **Part 2 – Control of gastric secretion**

# Learning Outcomes:

- LO1 - Differentiate between the motility of proximal and distal stomach (receptive relaxation, gastric accommodation, reservoir function, propulsion, grinding and retropulsion)
- LO2 - Define and describe the composition and function of gastric secretions (acid, pepsinogens/pepsin, intrinsic factor, mucus, gastric lipase)
- LO3 - Define the cellular mechanisms of gastric acid secretion (i.e. gastric parietal cells and the proton pump)
- **LO4** - Differentiate between the three phases in gastric secretion in response to ingestion of a meal
- **LO5** - Explain what is hyper-acid secretion, introduce the role/importance of the *Helicobacter pylori* as a cause of gastric disease and mechanisms of gastric acid blockade.

# The vagus nerve, appetite & acid



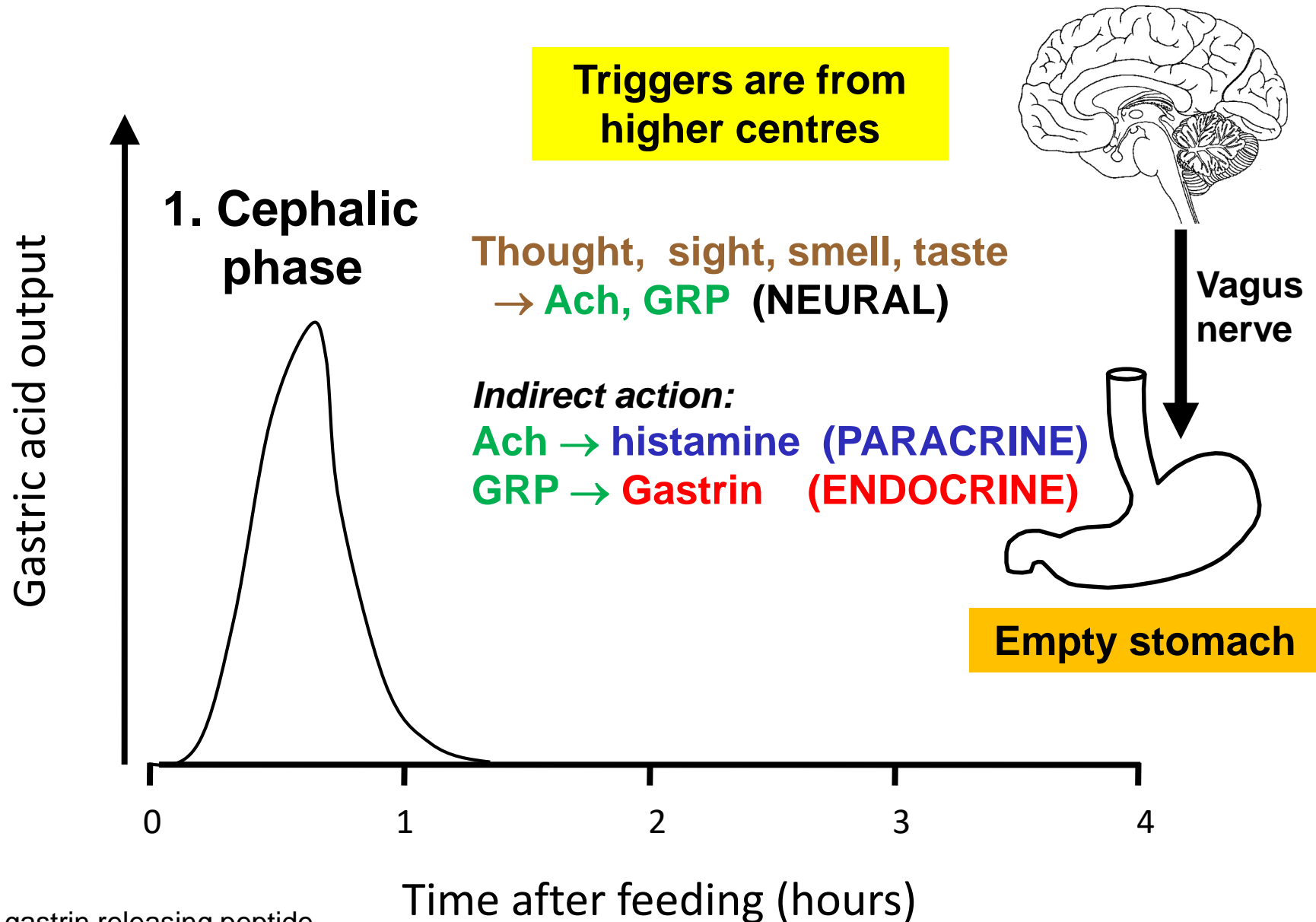
I P Pavlov

Nobel Prize, 1904 ...  
*in recognition of his work on  
the physiology of digestion*

*"Appetite spells gastric  
juice" .....*

gastric juice marketed  
for the stimulation of  
poor appetite

# Classical Phases of Gastric Acid Secretion



# Classical Phases of Gastric Acid Secretion

Distension of stomach

Ach (NEURAL – intrinsic nerves)

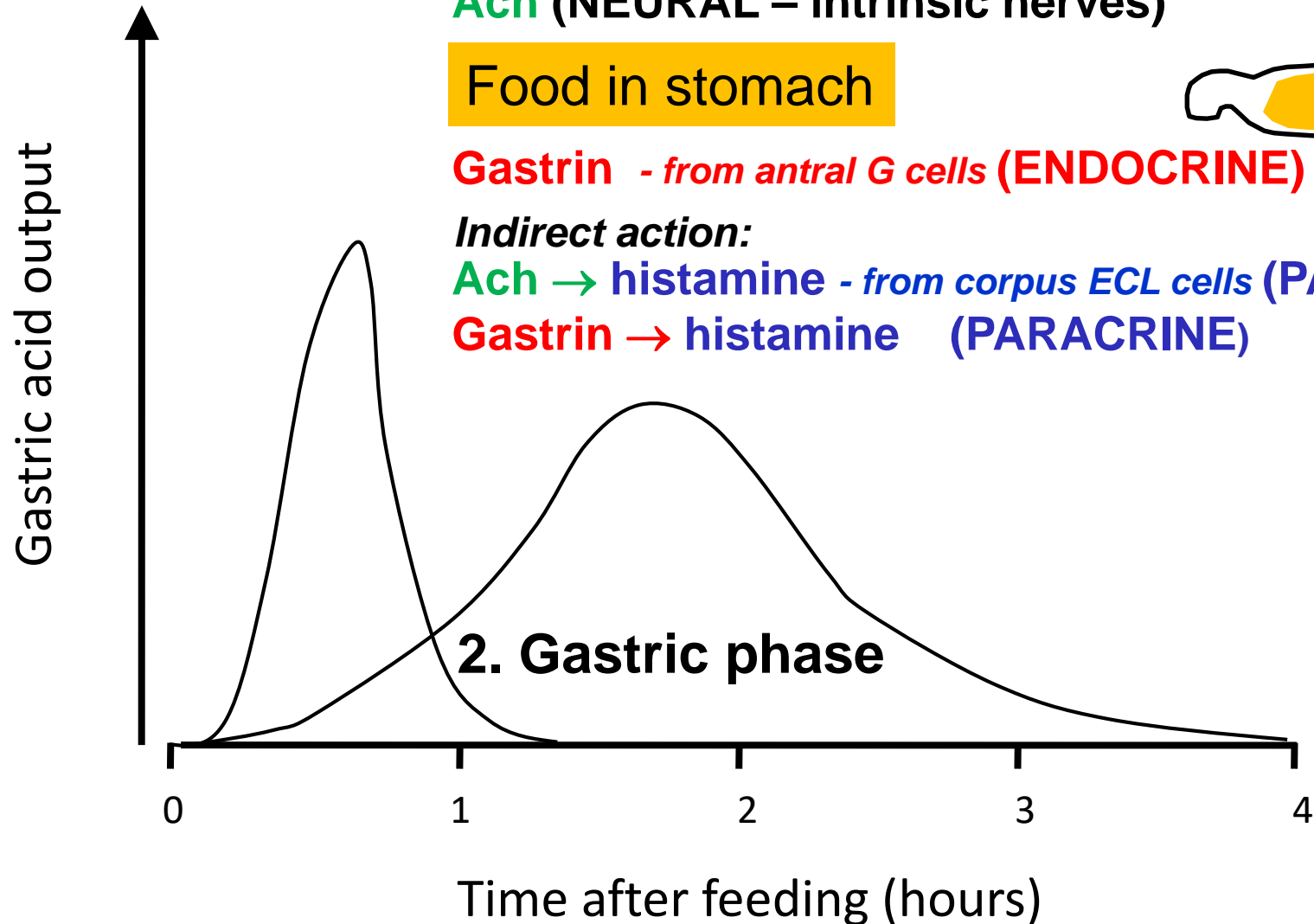
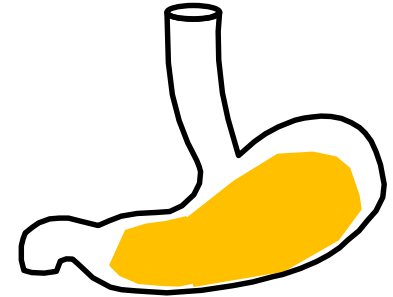
Food in stomach

Gastrin - from antral G cells (ENDOCRINE)

*Indirect action:*

Ach → histamine - from corpus ECL cells (PARACRINE)

Gastrin → histamine (PARACRINE)



# Classical Phases of Gastric Acid Secretion

## Distension/Food in duodenum

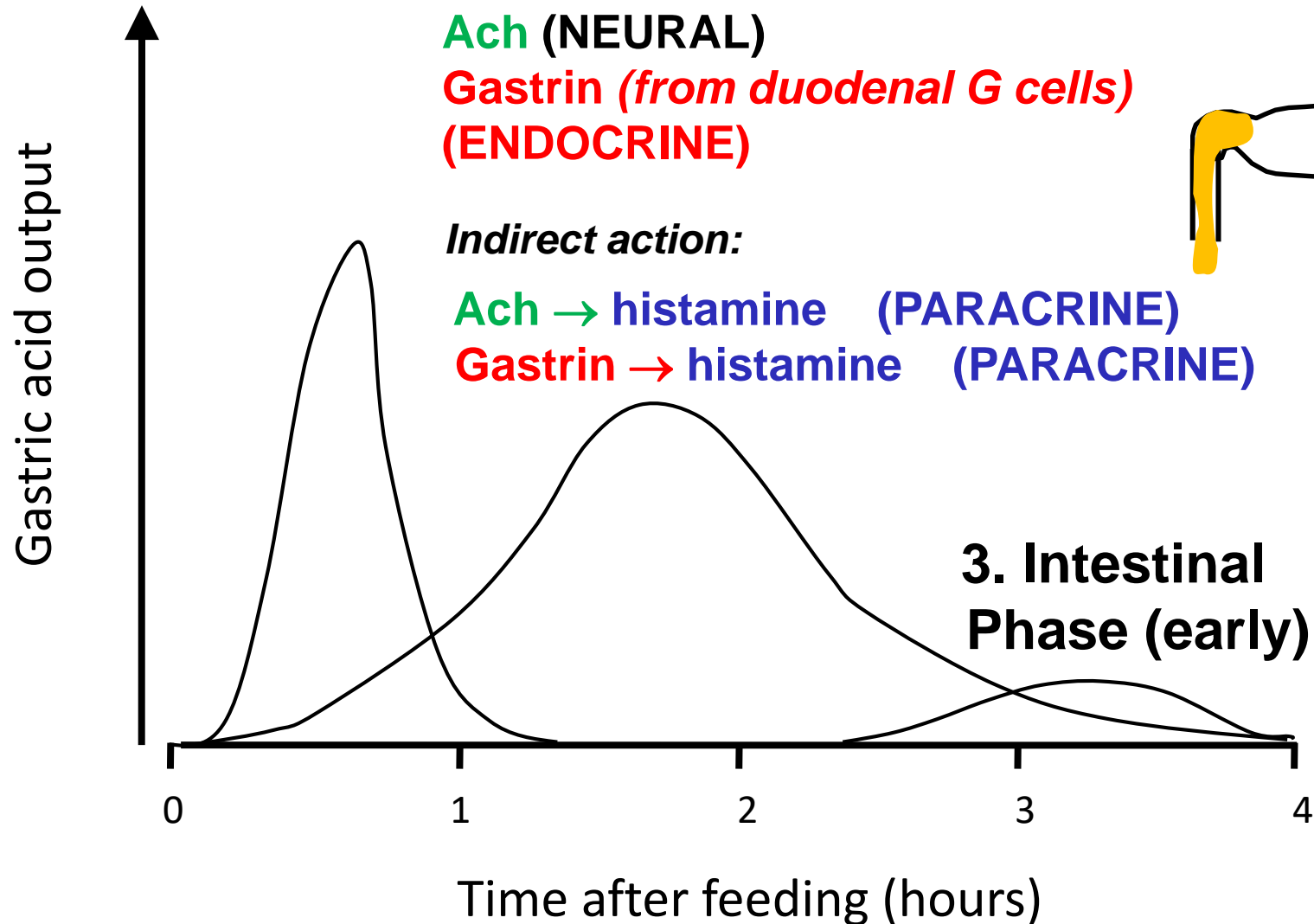
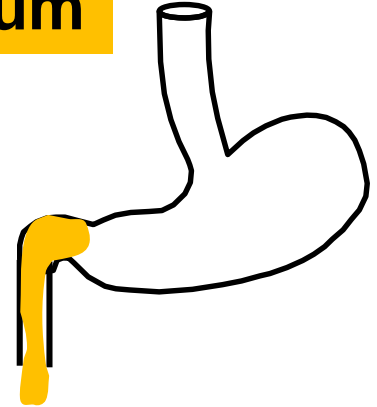
Ach (NEURAL)

Gastrin (from duodenal G cells)  
(ENDOCRINE)

*Indirect action:*

Ach → histamine (PARACRINE)

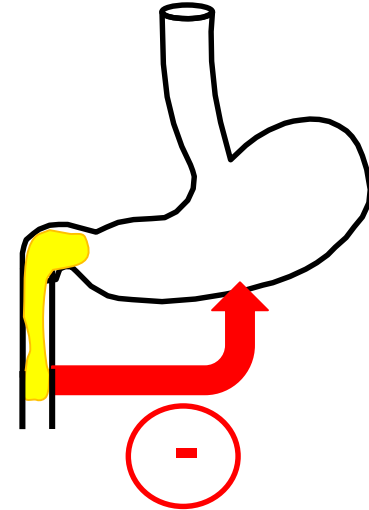
Gastrin → histamine (PARACRINE)



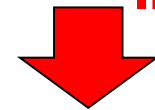
# Classical Phases of Gastric Acid Secretion

Fat-rich chyme entering the duodenum results in release of hormones to switch off gastric secretions and motility

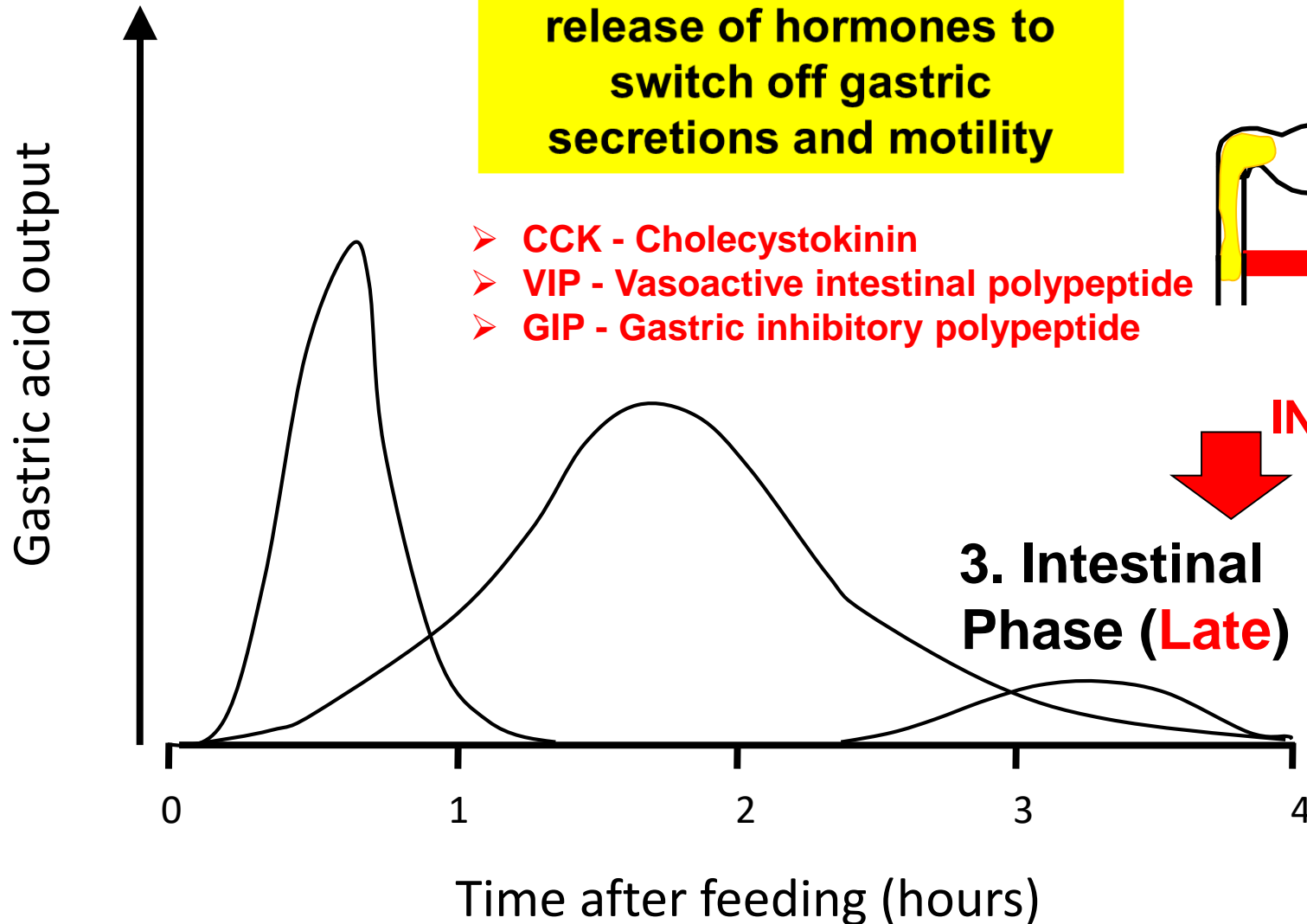
- CCK - Cholecystokinin
- VIP - Vasoactive intestinal polypeptide
- GIP - Gastric inhibitory polypeptide



**INHIBITION**

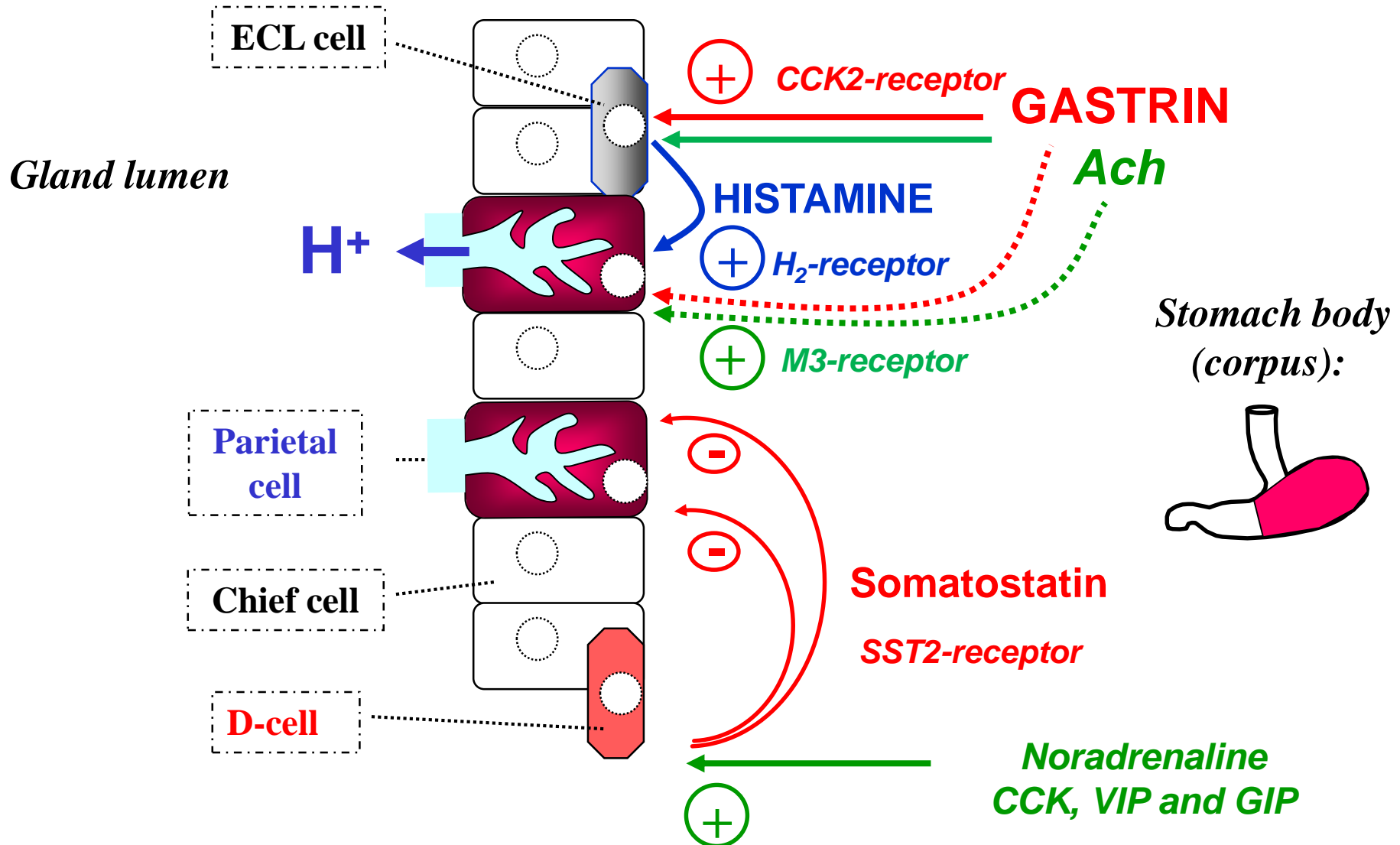


**3. Intestinal Phase (Late)**

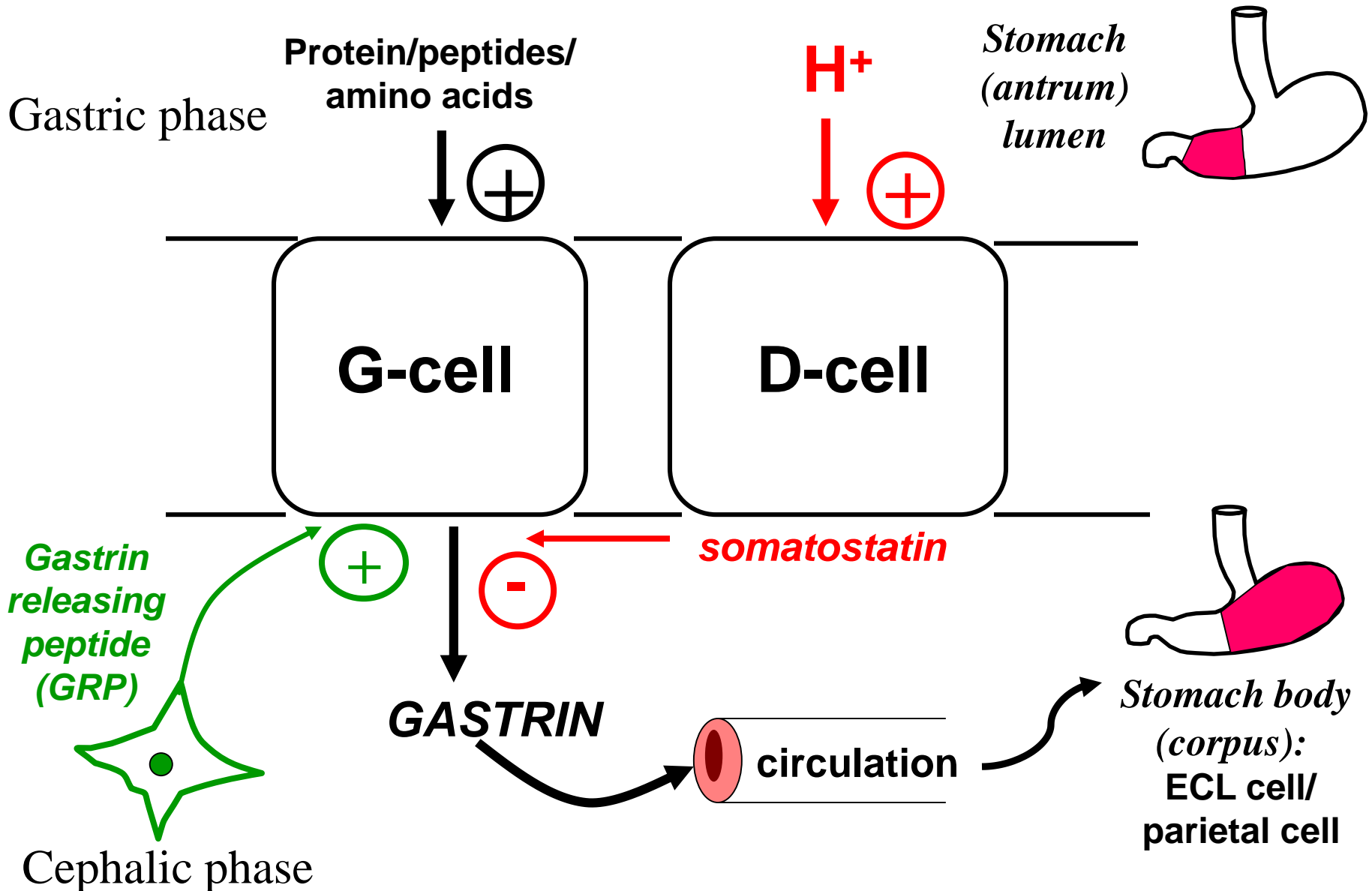




# INHIBITION OF ACID SECRETION



# Control of antral G-cell function



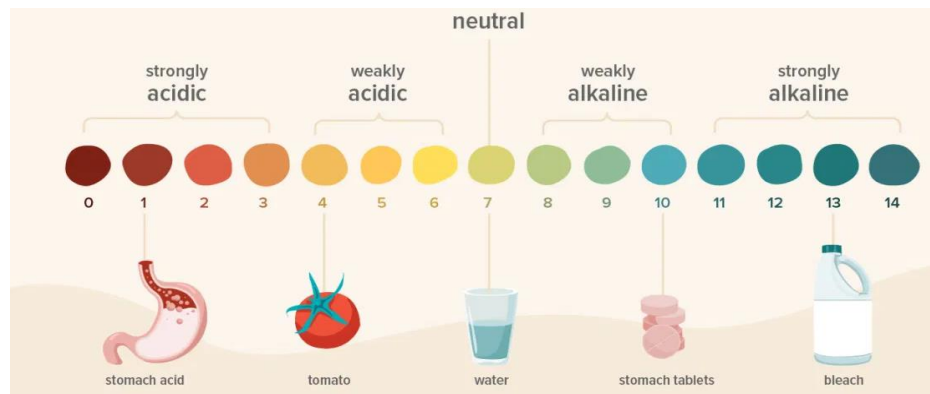
# Acid inhibitory – antacids



*reflux oesophagitis*  
“heart burn”

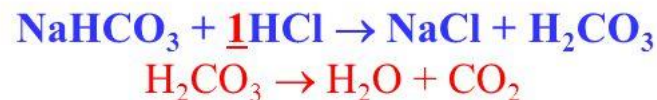


*Peptic ulcer*



## Commercial Antacids Use A Variety of Chemicals

❖ **Bicarbonate** - based antacids: Alka Seltzer



❖ **Calcium** - based antacids: Tums, Rennies



❖ **Aluminum** - based antacids: Maalox, Mylanta



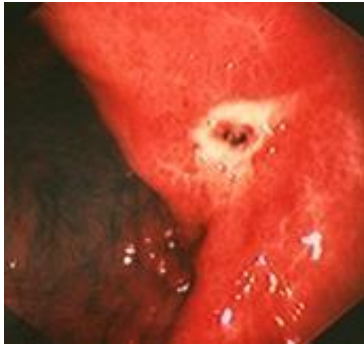
❖ **Magnesium** - based antacids: Mylanta, Milk of Magnesia



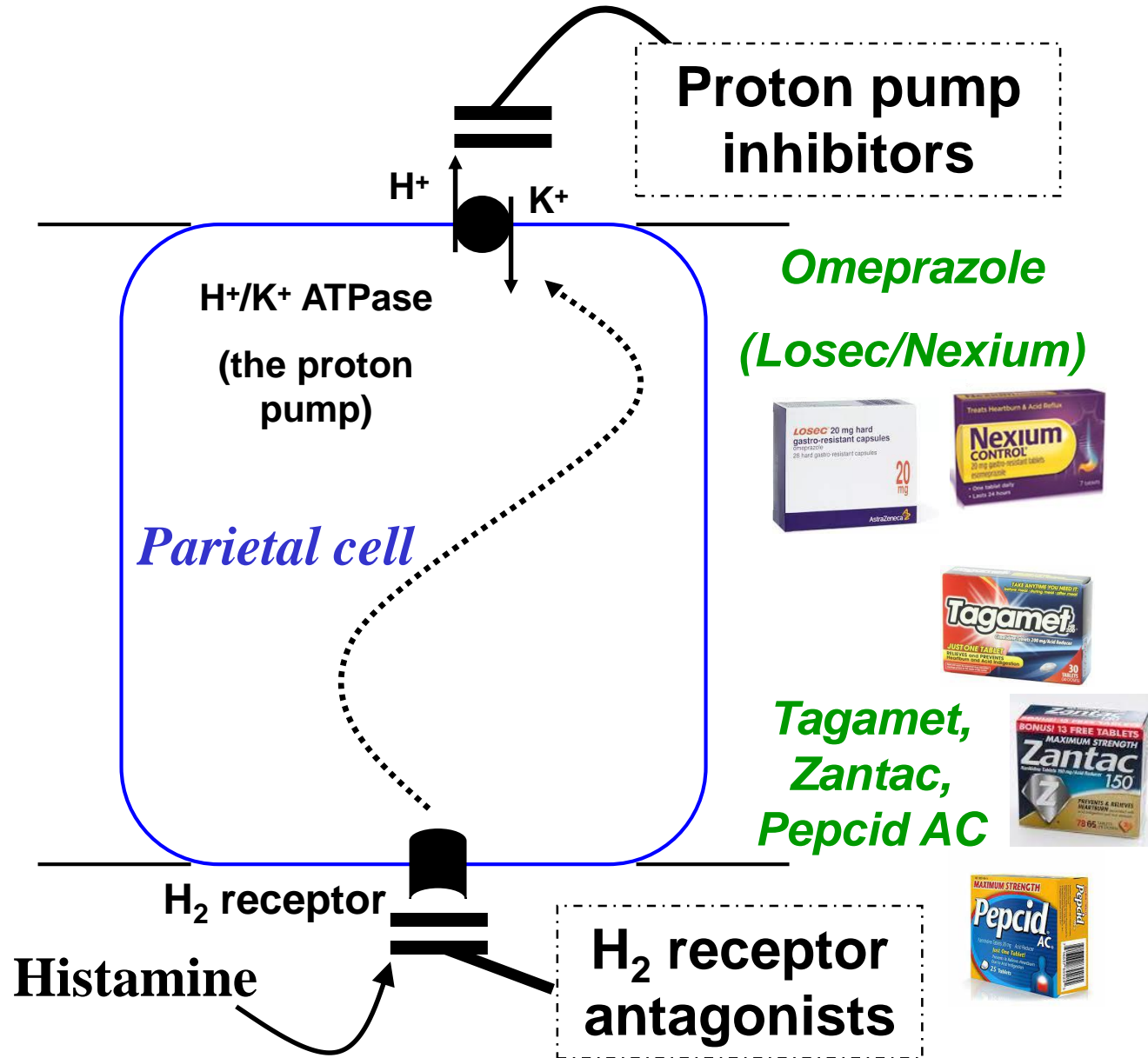
# Molecular targeted acid inhibitory therapy



reflux oesophagitis  
"heart burn"



Peptic ulcer

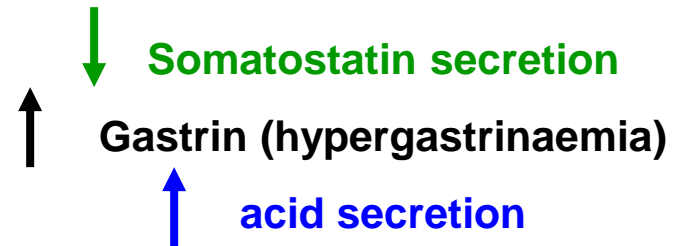


# *Helicobacter pylori*

A class 1 biological carcinogen (IARC, 1994)

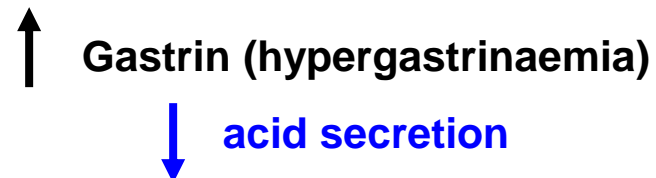


Infection in the antrum, associated with;



duodenal and peptic ulcer disease

Infection in antrum and corpus, associated with



atrophic gastritis, gastric cancer



*The Nobel Prize in Physiology or Medicine 2005*

**"for their discovery of the bacterium *Helicobacter pylori* and its role in gastritis and peptic ulcer disease"**

**3 October 2005**



**Barry J. Marshall**



**J. Robin Warren**

<http://nobelprize.org/medicine/laureates/2005/press.html>



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*Thank you for  
your attention.*

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<http://pcwww.liv.ac.uk/~bjcampbl/Indigestion%201.htm>

