

# *Gastro-intestinal tract (GIT)*

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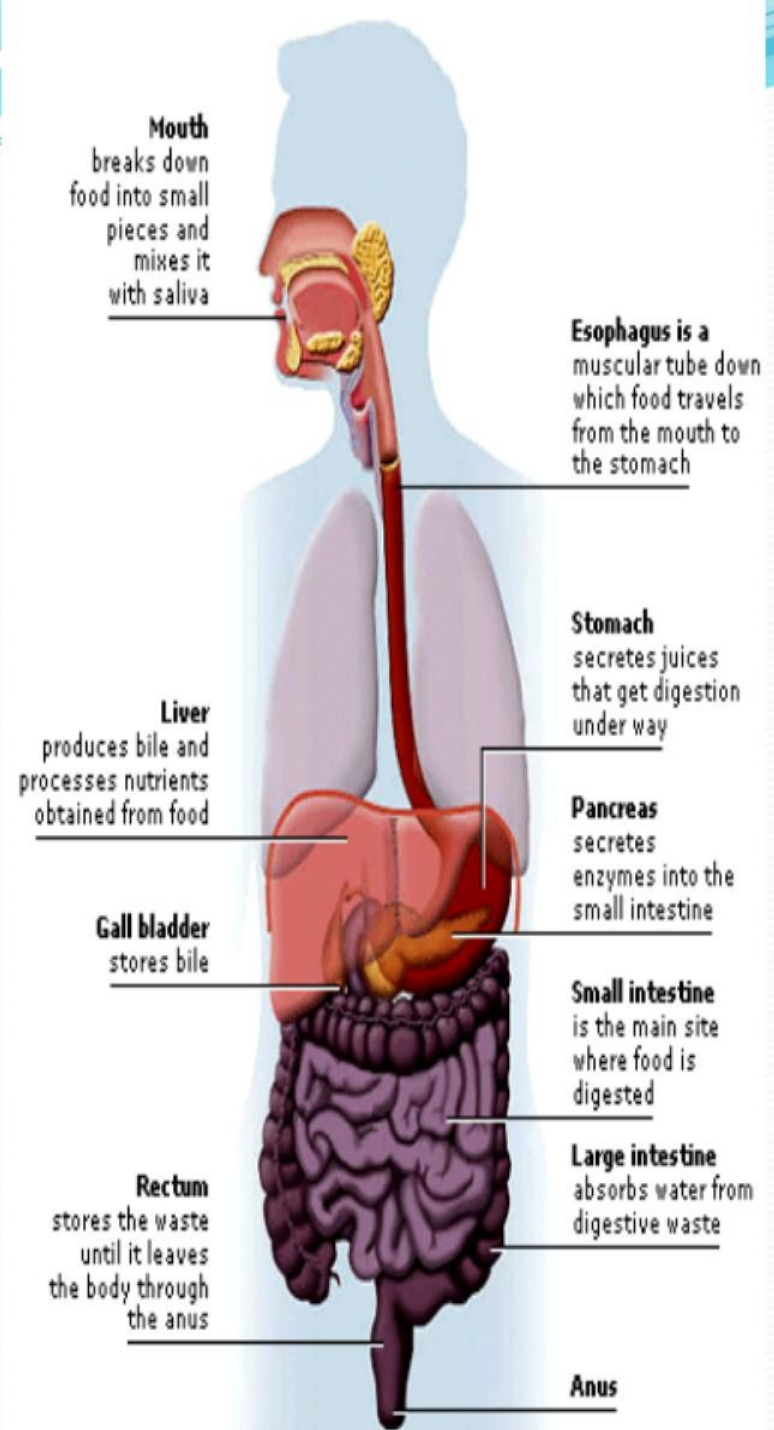
• **Digestive system is formed of:**

• **Alimentary tract (Gut – Gastrointestinal tract):**

It is a muscular tube (about 5-7 meters) that includes the mouth, pharynx, stomach, small intestine and large intestine.

• **Digestive glands:**

These glands include salivary glands, gastric glands, liver, pancreas and intestinal glands. These glands release their secretions containing digestive enzymes into gut.





- **Function of the digestive system:**

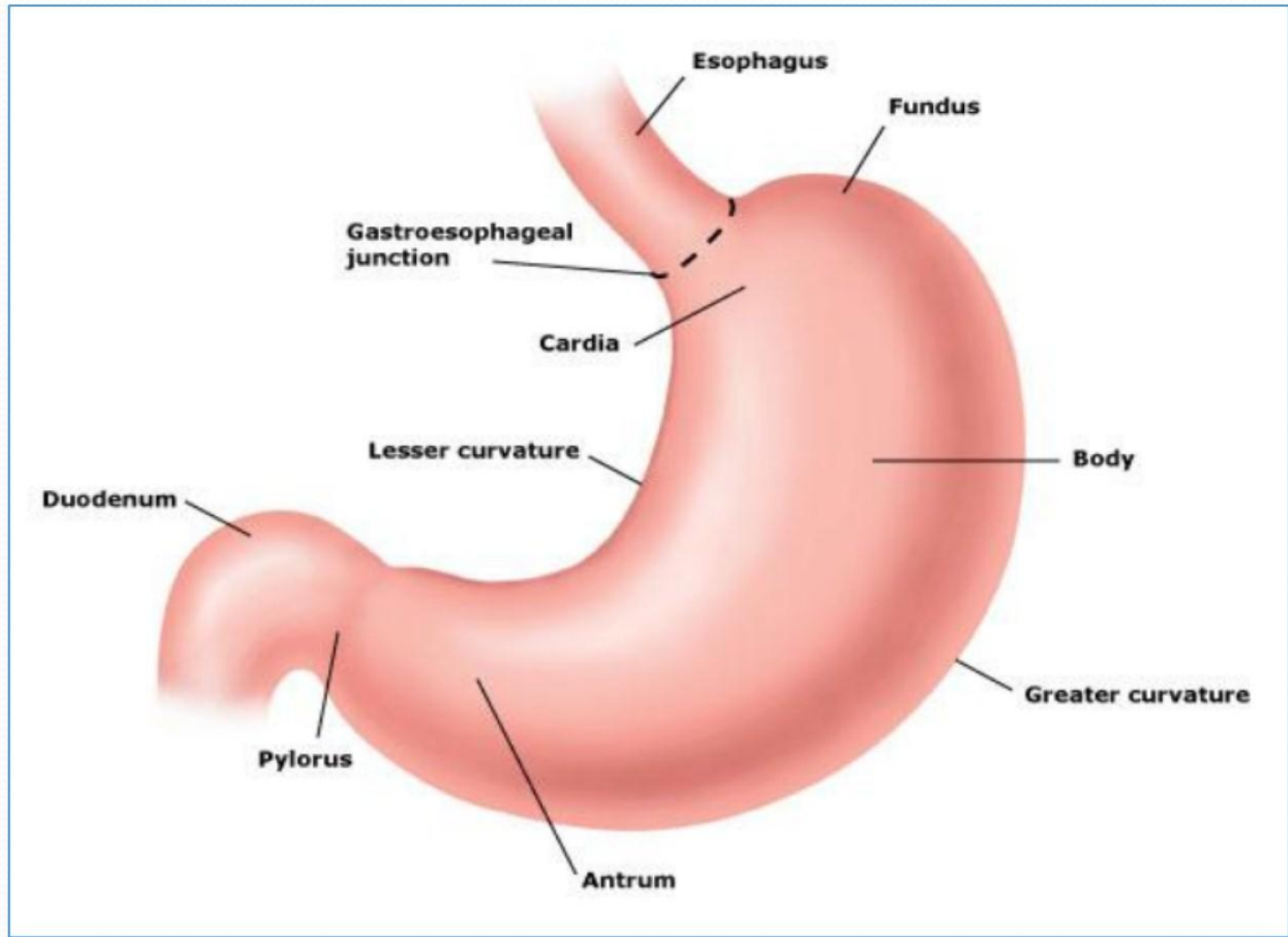
1. ***Motility.***

2. ***Secretion.***

3. ***Digestion and absorption***

## ○ ***Gastric secretion***

The stomach secretes 3 liters/day of gastric juice, the gastric juice is acidic secretion composed of about 99% water and electrolytes ( $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Ca}^{++}$ ),  $\text{HCl}$  and organic constituents (Mucus, enzymes, intrinsic factor).



## ➤ Pepsin:

**Pepsin** is an enzyme whose zymogen (pro-enzyme), pepsinogen, is released by the chief cells in the stomach and that degrades food proteins into peptides.

➤ Gastric lipase: Weak lipolytic enzyme.

## ➤ Intrinsic factor:

It is glycoprotein secreted by parietal cells of gastric glands in the body and fundus. It combines with vitamin B<sub>12</sub> → intrinsic factor - vitamin B<sub>12</sub> complex which is absorbed in the lower ileum. Its absence leads to vitamin B<sub>12</sub> deficiency (pernicious anemia).

## ➤ Gastric hydrochloric acid (HCl):

The main constituent of gastric acid is hydrochloric acid which is produced by parietal cells (also called oxyntic cells) in the gastric glands in the stomach.

### ▪ **Functions of HCl:**

1. HCl activates pepsinogen into the enzyme pepsin, which then helps digestion of protein by breaking the bonds linking amino acids, a process known as proteolysis.
2. Antibacterial action: growth of many micro-organisms inhibited by such an acidic environment, which is helpful to prevent infection.
3. Helps in the absorption of  $\text{Ca}^{++}$  and iron.
4. Helps milk clotting.

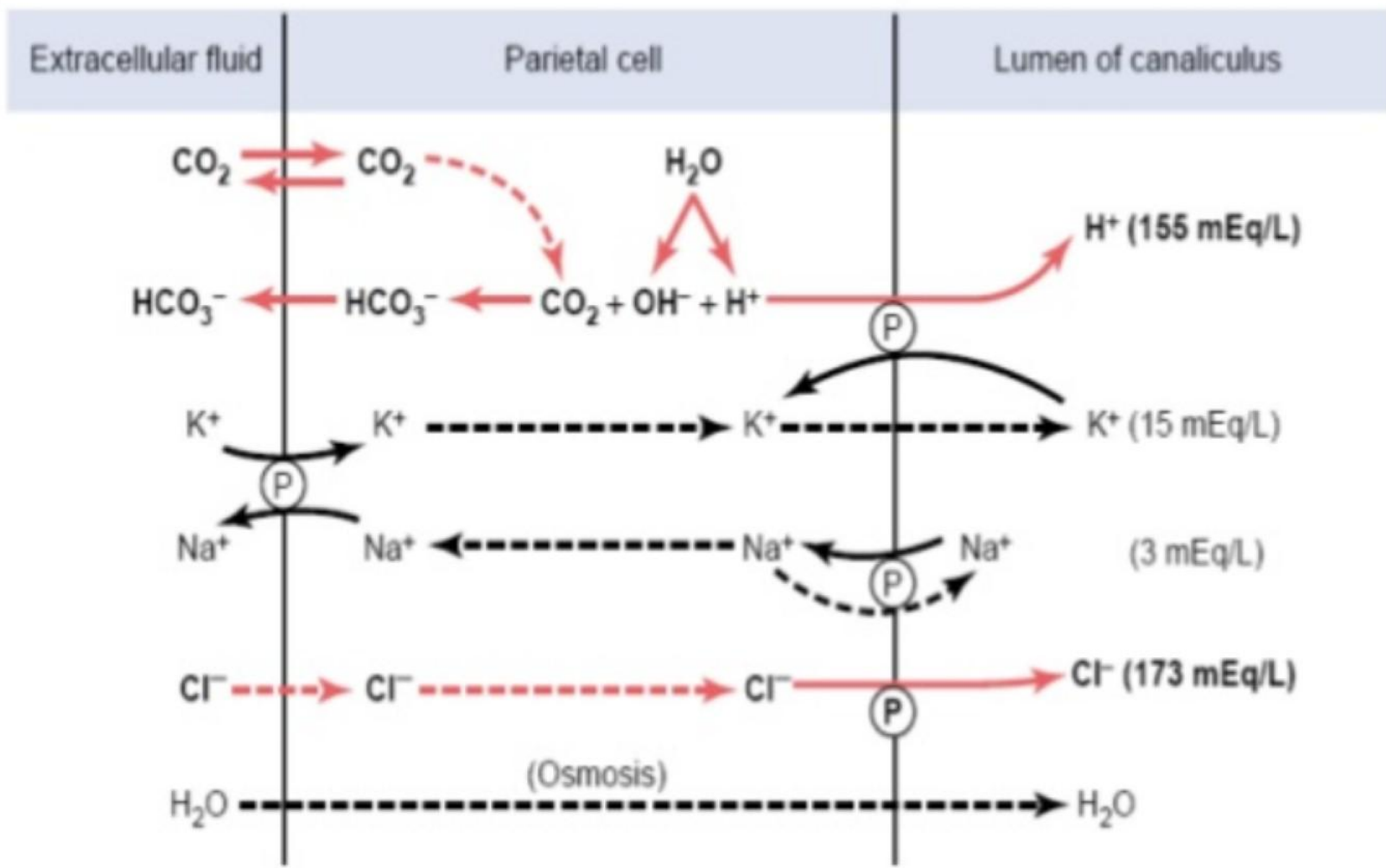
## • Formation of HCl:

Occurs in parietal cells in the following steps:

1. Ionization of intracellular water into  $H^+$  and  $OH^-$ .  $H^+$  passes to the lumen of gastric glands by  $H^+ -K^+$  pump.
2.  $CO_2$  (of blood and metabolism) will unite with  $H_2O$  that helped by carbonic anhydrase enzyme  $\rightarrow H_2CO_3 \rightarrow H^+ + HCO_3^-$ . The  $H^+$  unites with  $OH^-$  to form water again.
3.  $HCO_3^-$  passes from the cell to blood (in exchange with  $Cl^-$  ion) to unite with  $Na^+$  to form  $NaHCO_3$ .
4.  $Cl^-$  ion shifts from blood ( $NaCl \dots Na^+ + Cl^-$ ) actively into lumen of gastric glands.



# Formation of HCL



# • Control of HCl secretion:

## □ **Stimulatory factor:**

There are 3 types of receptors on the parietal cell membrane. When activated → ↑ HCl secretion:

### 1. H<sub>2</sub> receptors (Histamine receptors):

They are stimulated by histamine released from cells in the gastric mucosa that resemble mast cells. These receptors are **blocked by H<sub>2</sub> blockers, cimetidine** (blocks stimulatory effect of histamine on H<sup>+</sup> secretion and also blocks histamine's potentiation of ACh effects). Antihistaminic drugs (H<sub>1</sub>) don't interfere with the stimulatory effect of histamine on the gastric secretion.

### 2- M<sub>3</sub> receptors (Muscarinic cholinergic receptor):

They are **stimulated by acetylcholine** (released from postganglionic parasympathetic fibers). These receptors **blocked by atropine**.

### 3- Gastrin receptors:

They are **stimulated by gastrin hormone** released from cells in the antral mucosa into blood then reach the parietal cells *via* blood.

## □ **Inhibitory factor:**

These are compensatory mechanisms against excess HCl secretion:

1. Gastrointestinal hormones as secretin and Cholecystokinin hormone (CKK).
2. Enterogastric reflex; distension of duodenum → reflex (local enteric reflex) gastric inhibition to protect duodenum from excess distension and acidity.
3. Prostaglandin E.

## ❑ *How does the stomach protect itself against gastric juice?*

- ✓ The gastric enzymes are in the mucosal cells in an inactive form.
- ✓ The mucous forms a thick coat which prevents chemical and mechanical injury of the stomach.
- ✓ Low permeability of gastric mucosal cells to HCl and pepsin.
- ✓ The alkalinity of gastric cells makes unsuitable medium for pepsin activity.
- ✓ Continuous removal and regeneration of the mucosa.
- ✓ Prostaglandin:
  - Strengthen and augment the gastric mucosal barrier.
  - Stimulate mucus secretion.
  - Inhibit acid secretion.
  - Increase mucosal blood flow.

## □ Peptic ulcer:

This is a lost area in the mucosa of the stomach, first part of the duodenum (common site) or last part of the oesophagus that occurs due to auto-digestion by the gastric juice.

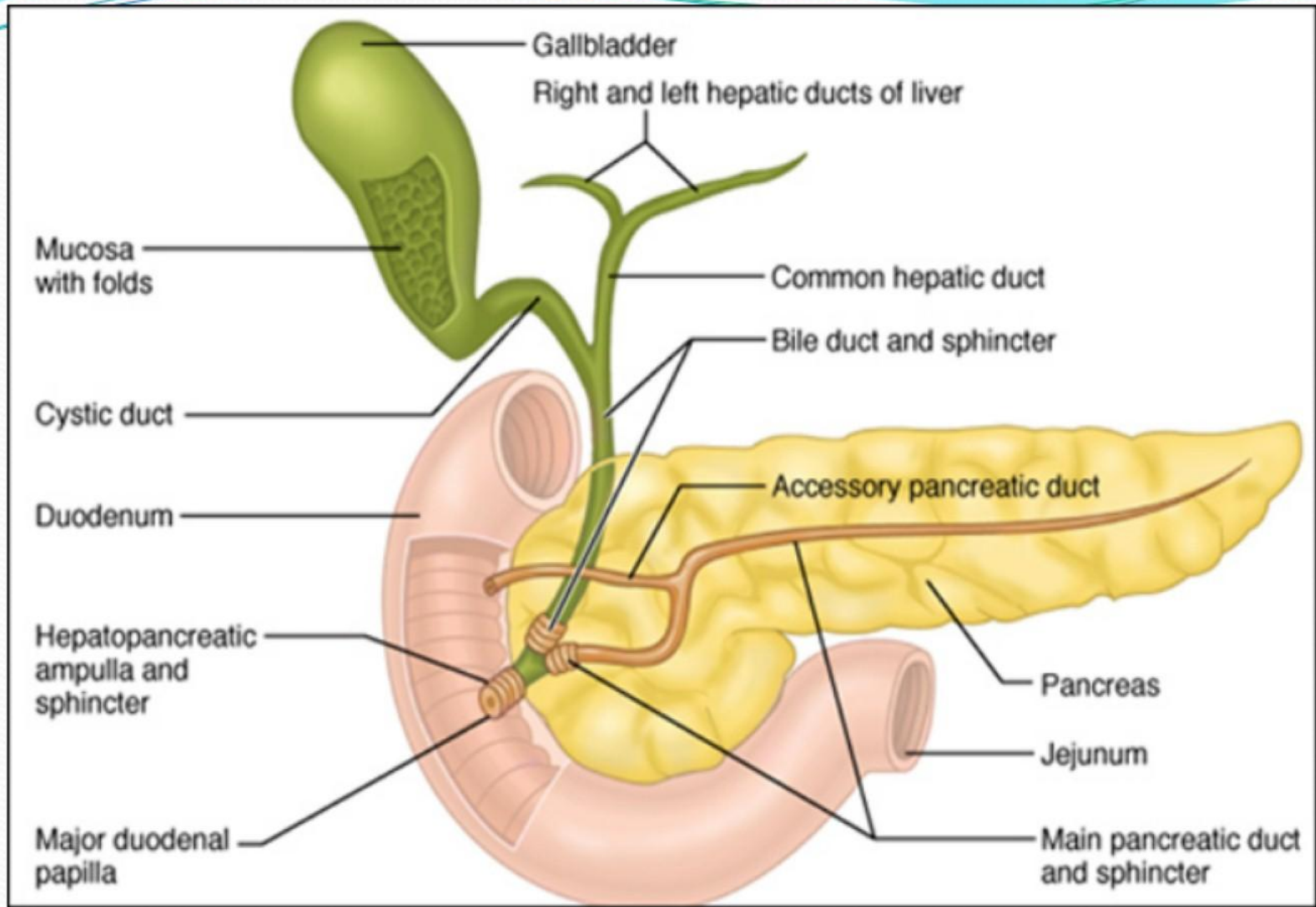
### ✓ **Causes of peptic ulcer;**

Imbalance between the rate of gastric secretion and degree of mucosal protection against autodigestion.

- Excessive gastric secretion: main cause and it may be due to;
  - Stress, anxiety or nervous tension.
  - Excessive gastrin secretion from gastric tumors (Zollinger-Ellison syndrome).
- Breakdown of the mucosal barrier: it may be due to
  - 1- Decreased mucus secretion.
  - 2- Excessive intake of gastric irritants (alcohol and spices).
  - 3- Excessive intake of aspirin that inhibit prostaglandin synthesis.
  - 4- Infection of the gastric mucosa with certain types of bacteria.
  - 5- Excessive smoking.

## □ Physiological action of Anti-peptic ulcer drugs:

1. **Atropine:** blocks  $H^+$  secretion by inhibiting cholinergic receptors on the parietal cells so inhibiting the ACh stimulation of  $H^+$  secretion.
2. **Cimetidine:** blocks  $H_2$  receptors so it inhibits histamine stimulation of  $H^+$  secretion.
3. **Omeprazole:** directly inhibits  $H^+-K^+$  ATPase and  $H^+$  secretion.



## ○ *The bile*

**Bile** is a dark green to yellowish brown fluid, produced continuously by the liver (liver bile, alkaline) then stored and concentrated in the gall bladder (gall bladder bile, acidic). At the time of digestion, both the stored bile and dilute bile coming directly from liver are discharged into the duodenum. The composition of gallbladder bile is about 97% water in addition to bile salts, bile pigments as bilirubin and fats as cholesterol.



## ❑ **Functions of bile salts:**

- Digestion, bile salts are important for complete digestion of fat.
- Absorption (of fatty acids, calcium, iron and fat soluble vitamins A, D, K and E).
- Excretion of certain drugs, toxins and minerals.
- Choleric action (bile salts stimulate bile formation by liver).
- Laxative action (bile salts stimulate intestinal mobility).
- Antiputrefactive action through helping digestion, absorption, excretion and its laxative action.
- Source of alkali.

- **Choleretics:** they are substances that increase the bile secretion from liver as bile salts and secretin hormone.
- **Cholagugues:** they are substances that evacuate the gall bladder as Cholecystokinin hormone (CKK).