

Medical laboratory techniques

Helicobacter pylori(Lab 6)

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HELICOBACTER PYLORI

Gastritis: is a histological term that describes stomach inflammation resulting from toxic exposures, infection, idiopathic inflammation, and autoimmunity. The most common cause of gastritis is H pylori infection. Other causes include acid reflux, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDS), alcohol use, and tobacco use, all of which can irritate the lining of the stomach. Severe illness and radiation therapy can also cause gastritis

Erosive gastritis: is most commonly caused by alcohol use, tobacco use, and prolonged use of aspirin and non-steroidal anti-inflammatory drugs (NSAIDS).

The most common cause of chronic, nonerosive gastritis is a stomach infection caused by

Helicobacter pylori (H pylori).



Flagella

bacterial mobility & chemotaxis
to colonize under mucosa

Urease

neutralize gastric acid
gastric mucosal injury (by ammonia)

Lipopolysaccharides

adhere to host cells
inflammation

Outer proteins

adhere to host cells

Exotoxin(s)

- **vacuolating toxin (vacA)**
gastric mucosal injury

Secretory enzymes

- **mucinase, protease, lipase**
gastric mucosal injury

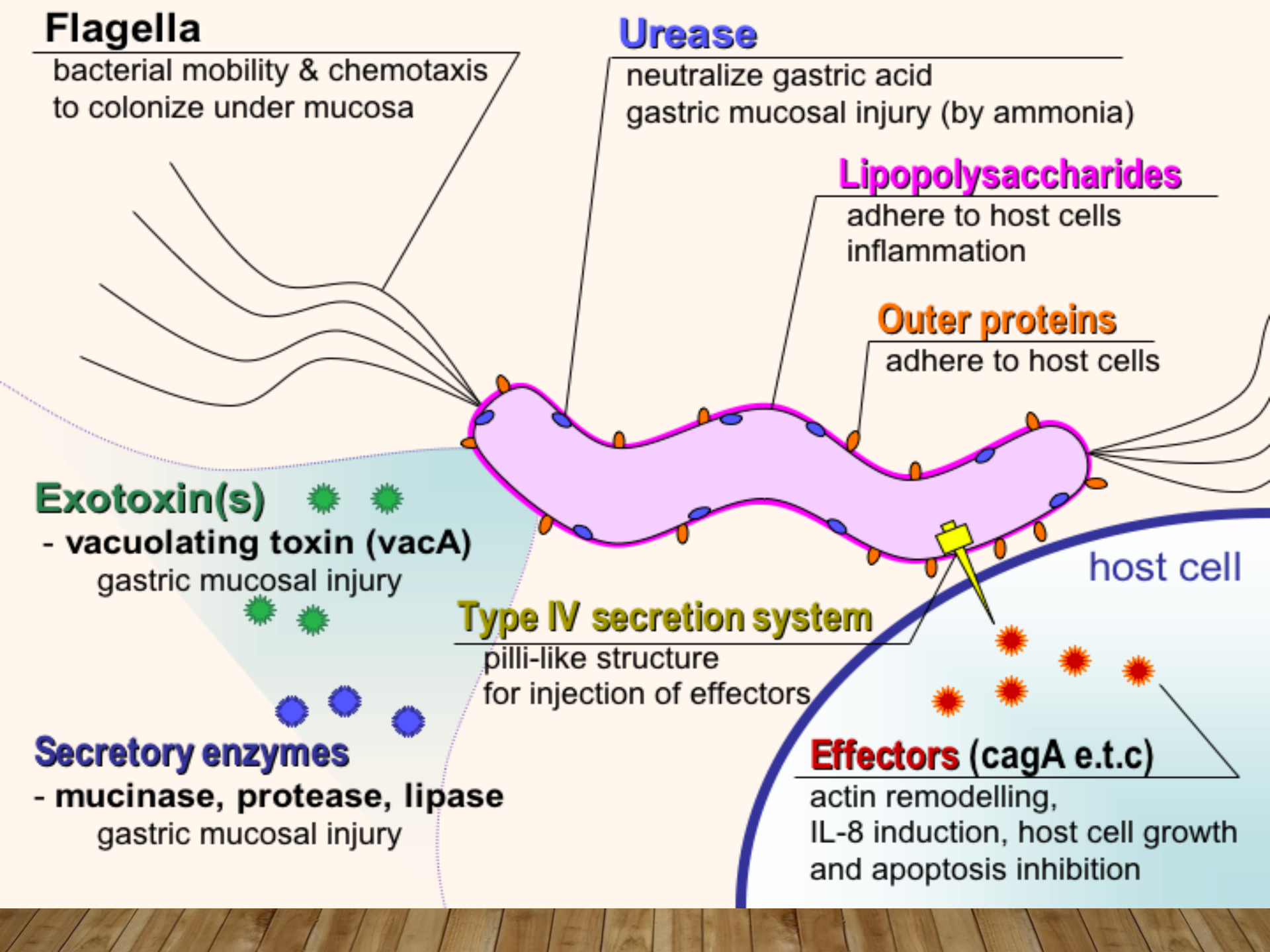
Type IV secretion system

pilli-like structure
for injection of effectors

Effectors (cagA e.t.c)

actin remodelling,
IL-8 induction, host cell growth
and apoptosis inhibition

host cell



In some cases, gastritis can be life threatening, with symptoms including:

- **Bloody stool** (blood may be red, black, or tarry in texture)
- **Severe abdominal pain**
- **Vomiting blood or black material** (resembling coffee grounds)

Although acute infection can cause abdominal pain and dyspepsia, there is typically no clinical recognition of acute infection.



SYMPTOMS

The symptoms of gastritis If infection with H pylori bacteria is the cause, will **remain** as long as the infection is **untreated**. H. pylori is uniquely **adapted to the acidic environment** of the stomach through its **ability to metabolize urea to ammonia**, which provides a buffered microenvironment that allows prolonged asymptomatic colonization.

Symptoms include **burning abdominal pain, Loss of appetite, nausea with or without vomiting**

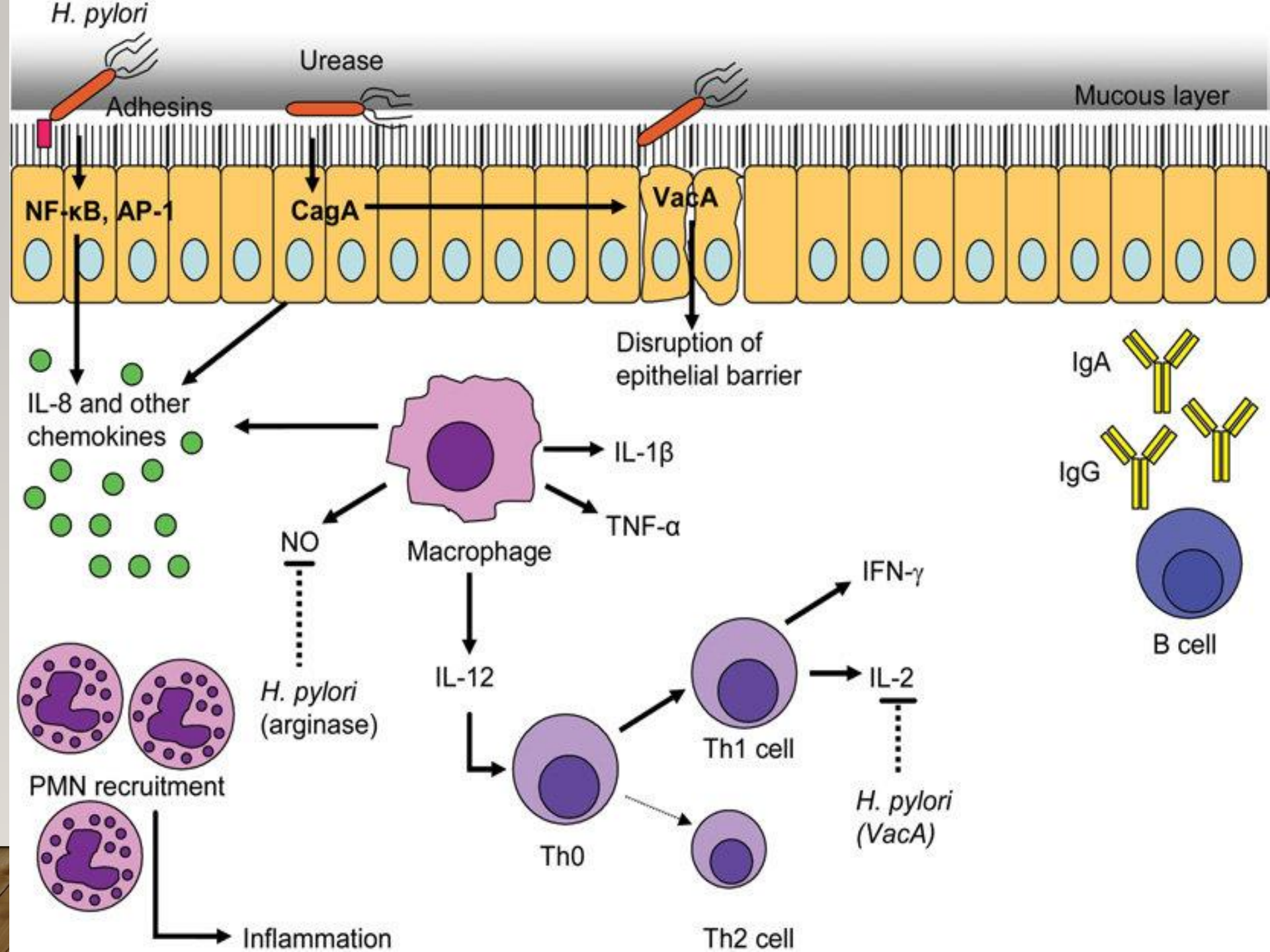
Rather, the burden of **H. pylori** results from **chronic infection of the stomach**. **The development of peptic ulcer disease and adenocarcinoma caused by chronic H. pylori infection** correlates with the anatomical distribution of inflammation. When H. pylori chronic gastritis affects the antrum predominantly, there is an **association with duodenal ulcers, increased serum gastrin levels and excess acid production**, and no gastric mucosal atrophy. However, when H. pylori affects the body and the antrum in a confluent or patchy manner, intestinal **metaplasia** develops, **oxyntic mucosa atrophies**, and **acid production decreases**.

This **latter type** of *H. pylori* chronic gastritis is associated with **gastric ulcerations and increased risk for adenocarcinoma and mucosa-associated lymphoreticular tissue (MALT) B-cell lymphoma.** Although eradication of *H. pylori* can reverse the **mucosal atrophy** and **restore acid production** in this setting, mucosal restoration occurs only in a minority of patients and does **not necessarily reverse the intestinal metaplasia.**




IMMUNE PATHOPHYSIOLOGY

The immune mechanisms for the persistence of HP infection in the stomach: suggest that pro-regulatory effects of **H. pylori infection**, including local **IL-10** production, increases in **regulatory T cells (Tregs)** in the gastric mucosa and increased **antigen-presenting cell (APC)** phagocytosis of apoptotic cells all contribute to persistence of chronic H. pylori gastritis.



DIAGNOSIS

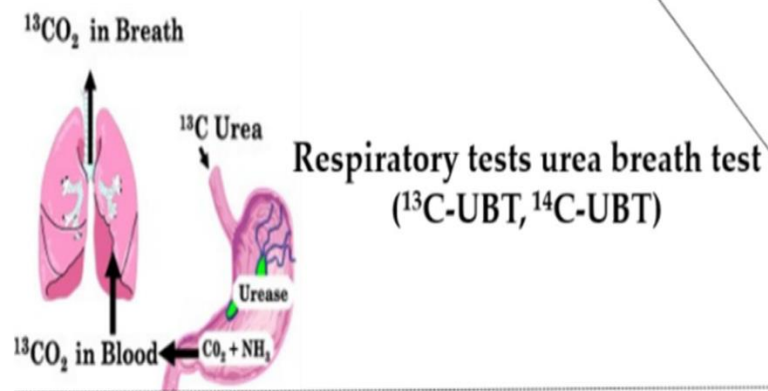
Active disease can be diagnosed with endoscopic **biopsy**, **which has high sensitivity and specificity**, while simultaneously assessing peptic and **malignant complications**.

- **Noninvasive testing** : includes **serum antibody detection** (best used in highly endemic areas to predict active infection),
 - **urea breath testing** (**limited** by **expense** & possible **false-positive** results), -
 - **fecal antigen testing** (which has potential advantages in the setting of intestinal **metaplasia** and after antibiotic treatment).
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Non-Invasive methods

Invasive methods

Current diagnostic tests for *H. pylori*



Normal

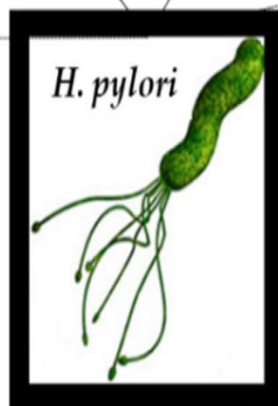


H. pylori gastritis

Endoscopy

Culture

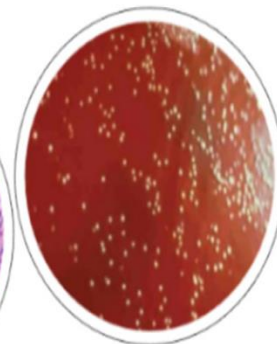
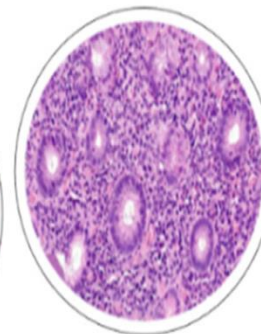
Stool antigen (SAT)



Rapid urease test



Histology

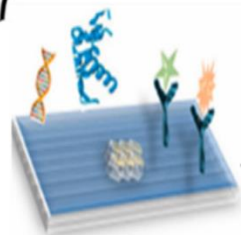


Serology



Biosensors methods

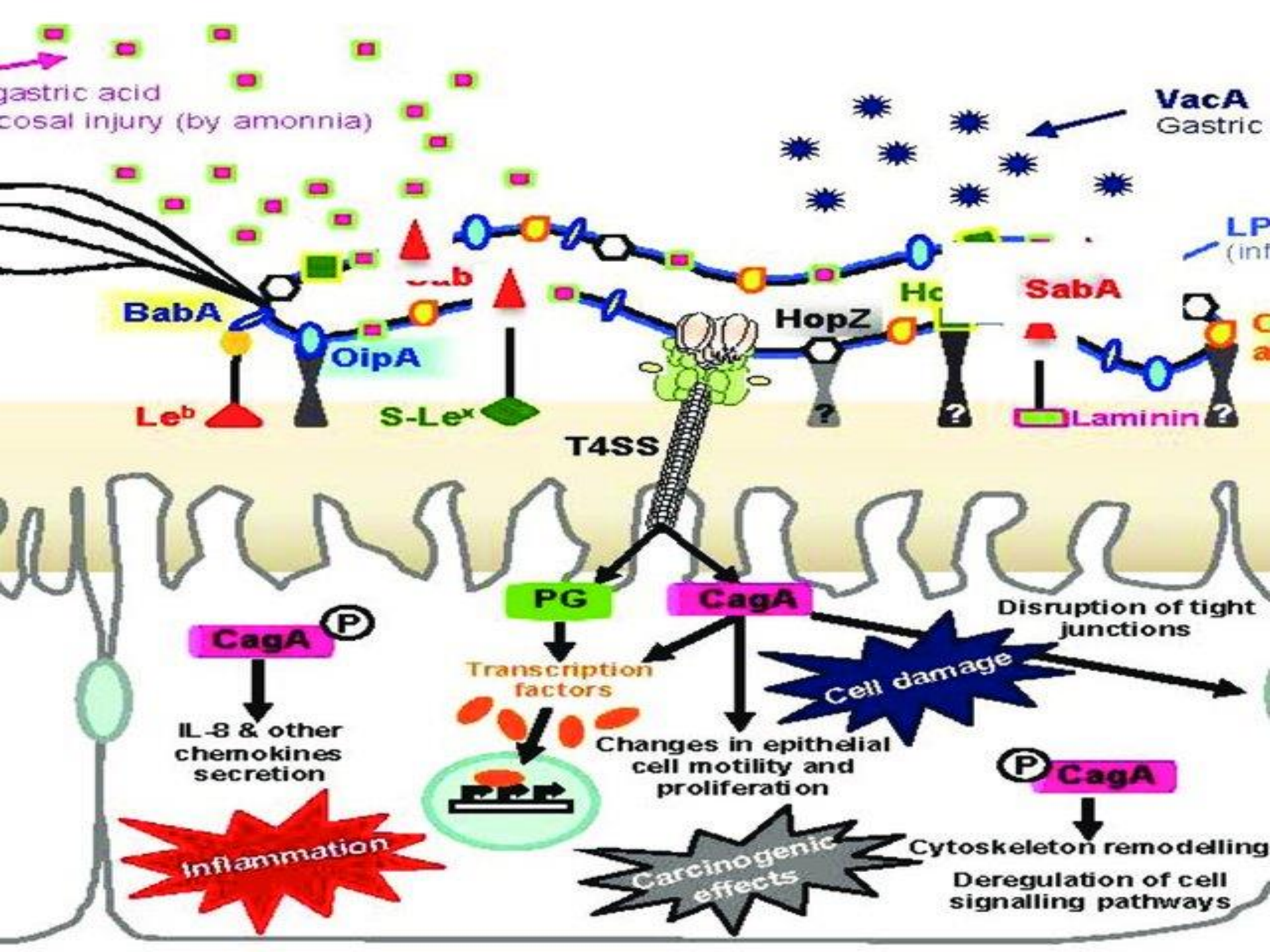
PCR Test



- Optoelectronic nano-biosensors
- Piezoelectric sensors
- Electrochemical biosensors



Detection





Thank You