Genus Campylobacter

C. jejuni & C. coli ← emerged as common human pathogens.

General characteristics

- Gram negative comma, S or gall-wing shape rods, motile with single polar flagellum & non-spore forming

- They are thermophilic (37°C – 42°C), grow on reduced O₂ (0.5%) and 20% CO₂

- The selective media is "Skirrows media" incorporated with vancomycin, polymyxin B & Trimethoprim to inhibit growth of normal flora.

- They are oxidase positive, catalase positive, urease negative, reduce nitrate and produce H₂S, they do not ferment CHO.

- Hippurate Hydrolysis test is used to differentiate between spp.

The antigens structure:

1- Heat stable lipopolysaccharide O-Ag with endotoxic activity.

2- Heat labile flagellar H-Ag.
**Pathogenesis**

Domestic animals (cattle, chickens & dogs) serve as source of the pathogens for humans. Transmission is usually feco-oral, large infective doses are needed ($\geq 10^4\text{m.o.}$) because They are sensitive To gastric acidity. The bacteria multiply in the small intestine → invading the epithelium & producing inflammation which result in appearance of RBC & WBC. So, localized invasion and toxic effect are responsible for development of enteritis.

**Clinical findings:**

After incubation period (1-7) days, acute onset begins as watery foul-smelling diarrhea followed by bloody stool accompanied by fever and sever abdominal pain. This enterocolitis usually self-limited (5-8) days, but are susceptible to erythromycin that shortens the duration of fecal shedding of bacteria.

**Laboratory diagnosis:**

A-specimen → diarrheal stool.  
Blood

B-culture on skirrow's media → small, gray, circular and glistening colonies ← detected by Gram’s stain for typical morphology.
Helicobacter pylori

This micro-organism emerged in 1990s, previously classified as Compylobacters because they have common characters.

General characteristics

1-Gram negative, spiral shaped rods. actively motile with multiple polar flagella.

2-Culture on skirrow’s media (3-6 days/37°C)

3-Oxidase positive, catalase positive and urease positive (more active than that of Proteus).
Pathogenesis of H. pylori

The natural habitat of H. pylori is the human stomach, it is acquired by ingestion. H. pylori use to live on gastric mucosa but not in the lumen where the pH only (1-2), while it is actively motile when attach to epithelial surface where the pH (6-7), also produce protease that modifies the gastric mucosa reducing the ability of acid to diffuse through mucus.

H. pylori produce potent urease activity that cause accumulation of large ammonia which buffer the acidity. Toxin and LPS may damage the mucosal cells.

Gastritis is characterized by chronic and active inflammation, even epithelial destruction and glandular atrophy may occur, thus H. pylori may be a major risk factor for gastric cancer.

Clinical findings

After short incubation period, patient will develop acute gastritis (abdominal pain, nausea, flatulence and bad smell breath) for 2 weeks but hypochlorhydria may persist leading to chronic gastritis, peptic ulcer, deudenal ulcer or high risk of gastric cancer. Those present with recurrent upper abdominal pain frequently accompanied by GIT bleeding. No bacteremia or disaminated disease.
Complete cure observed after elimination of the organism, although some people can harbor the pathogens for years without ill effect.

**Laboratory diagnosis of H. pylori**

*Speciment* → - Gastric biopsy

- Blood (serological Ab)

1-Histological smear: Gastritis and peptic ulcer usually diagnosed by gastroscopy, the taken biopsy can be stained by Giemsa or special silver stain, those can show the curved or spiral organisms.

2-Culture of biopsy: usually use Skirrow's media, it take 3-6 days in microaeropilic environment.

3-Urease test either:

- **In vitro urease** test done by inoculating the biopsy material on urea agar media, urease enzyme will split urea within 2hr shifting the pH to alkaline changing the color of indicator (phenol red) from yellow to pink.

- **Invivo (urea breath test)** done when $\text{C}^{13}$ or $\text{C}^{14}$ labeled urea ingested by the patient $\rightarrow$ urease activity $\rightarrow$ labeled $\text{CO}_2$ will be generated and can be detected in patients exhaled breath.

4-Serological test: the presence of serum IgG as anti-H.pylori Ab will reflect the infection but is of limited use in follow up.
**Treatment**

Triple therapy of Amoxicillin or tetracycline with metronidazol and bismuth sulfate for 14 days, can eradicate H.pylori in 70-95% of patients.