Vibrio, Aeromonas, Campylobacter, and Helicobacter

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0) Big Picture: Where they live + what they cause

Shared opening idea

• All are Gram-negative rods widely distributed in nature.
• They are studied together because many are water/animal-associated GI pathogens.

Reservoirs / natural habitat

• Vibrios: marine and surface waters.
• Aeromonads: fresh and brackish waters.
• Campylobacters: animals, especially domesticated animals.
• Helicobacters: GI and hepatobiliary tracts of humans and mammals; also chickens and wild birds.

Major clinical anchors

• V. cholerae: cholera - profuse watery diarrhea -> dehydration/death.
• C. jejuni: common cause of enteritis in humans.
• H. pylori: gastritis and duodenal ulcer disease.

Master map: organism -> habitat -> most tested clue

Organism group	Most important habitat/source	High-yield lab/clinical clue
Vibrio	Marine/surface water; contaminated water/food; seafood	Comma-shaped, oxidase+, polar flagella, halophilic; V. cholerae yellow on TCBS; cholera toxin -> cAMP watery diarrhea
Aeromonas	Fresh/brackish water; food/water; traumatic wound exposure	Oxidase+ Gram-negative rods resembling Enterobacteriaceae; often beta-hemolytic on blood agar
Campylobacter	Poultry, cattle, sheep, pigs, birds, pet dogs	Microaerophilic, grows best at 42 C, gull-wing rods, darting motility; C. jejuni hippurate+
Helicobacter pylori	Humans mainly; gastric mucous layer	Spiral Gram-negative rod, catalase+, oxidase+, urease+; causes chronic gastritis/ulcers/cancer risk

1) The Vibrios + Vibrio cholerae: morphology and identification

A) The Vibrios - general properties

Shape + basic tests

• Comma-shaped, facultatively anaerobic rods.
• Catalase positive and oxidase positive.
• Most species are motile by polar flagella.

Growth requirements

• Broad temperature range: 14-40 C.
• High pH range: 8.5-9.5.
• Require sodium chloride for growth - halophilic, meaning salt-loving.

Important species

• V. cholerae O1 and O139 cause cholera in humans.
• V. parahaemolyticus and V. vulnificus can cause skin/soft tissue infections, sepsis, or gastroenteritis.
• Cholera is associated with poor sanitation and contaminated water/food.

B) V. cholerae - morphology and identification

Culture and appearance

• Comma-shaped, curved, motile rod.
• Grows well at 37 C on routine media and selective TCBS agar.
• V. cholerae produces yellow colonies on TCBS because it ferments sucrose.

TCBS color contrast

• V. cholerae: sucrose fermenter -> yellow colonies.
• V. parahaemolyticus and V. vulnificus: non-sucrose fermenters -> green colonies.

Specimen timing

• Stool specimens should be collected early in diarrheal illness.
• In endemic areas, stool culture on TCBS plus enrichment broth culture are appropriate.

Biochemical pattern

• Regularly ferments sucrose and mannose but not arabinose.
• Oxidase positive.
• Can grow on most agar media without additional salt.

O antigen serogroups

• V. cholerae has O LPS antigen with about 200 serogroups.
• O1 and O139 cause epidemic and pandemic cholera.
• Non-O1/non-O139 can cause cholera-like diarrheal disease.

O1 serotypes and biotypes

• O1 serotypes: Ogawa, Inaba, Hikojima.
• Two epidemic O1 biotypes: classic and El Tor.
• El Tor produces hemolysin and is resistant to polymyxin B.

Capsules

• O139, non-O1 V. cholerae strains, and V. vulnificus produce acidic polysaccharide capsules.
• V. cholerae O1 does not produce this capsule.

2) Vibrio cholerae: toxin, disease, diagnosis, treatment, epidemiology

A) Cholera toxin + pathogenesis

Enterotoxin structure/action

• Heat-labile enterotoxin made of A and B subunits.
• B subunits bind GM1-ganglioside receptor on enterocyte membrane.
• A1 subunit increases intracellular cAMP.

Mechanism of diarrhea

• A1 ADP-ribosylates the stimulatory G protein and locks it active.
• Persistent adenylate cyclase activation -> increased cAMP.
• cAMP drives active secretion of Na, Cl, K, bicarbonate, and water into intestinal lumen.

Pathologic result

• Prolonged hypersecretion of water and electrolytes.
• Electrolyte-rich diarrhea can reach 20-30 L/day.
• Can cause dehydration, shock, acidosis, and death.

Attachment and toxins

• Organisms attach to epithelial microvilli.
• They multiply and liberate cholera toxin.
• They may also liberate mucinases and endotoxin.

Host/source/risk

• Pathogenic only for humans.
• Can grow with copepods and zooplankton.
• Contaminated food and water are the more likely source of infection.
• Achlorhydria/hypochlorhydria or PPI use lowers infectious dose: about 10^3 instead of 10^10.

B) Clinical findings

Disease spectrum

• Ranges from asymptomatic intestinal colonization to severe diarrhea.
• Incubation period: 12 hours to 3 days depending on inoculum size.

Classic symptoms

• Sudden nausea and vomiting.
• Then profuse diarrhea with abdominal cramps.
• Rice-water stool.

Severe cholera

• Fluid loss can reach 1 L/hour.
• Dehydration can lead to shock and collapse.
• Mortality 25-50% untreated; reduced to about 1% with early fluid replacement.

Biotype and immunity

• V. cholerae O1 El Tor tends to cause milder disease than classic biotype.
• Attack of cholera is followed by immunity to reinfection, but duration/degree are not known.
• Serum vibriocidal antibodies titer >= 1:20 are associated with protection; antitoxin antibodies are not protective.

3) Vibrio cholerae: laboratory tests, treatment, prevention + other Vibrios

A) Diagnostic laboratory tests

Specimens and transport

• Stool specimens collected early in diarrheal illness.
• Inoculate within 2-4 hours of collection.
• If delayed, mix in Cary-Blair transport medium.

Microscopy clue

• Dark-field or phase-contrast microscopy shows motility.
• Shooting star motility after mixing with polyvalent O1 antisera suggests V. cholerae O1.

Culture

• Grows well on most agar media.
• Rapid growth in alkaline peptone broth/water containing 1% NaCl at pH 8.5.
• Also grows on TCBS agar.

Confirmation

• Slide agglutination using anti-O group 1 or group 139 antisera.
• Biochemical reaction patterns complete identification.

B) Treatment, prevention, and epidemiology

Treatment priority

• Water and electrolyte replacement is the most important step.
• It corrects severe dehydration and salt depletion.

Antimicrobials

• Appropriate antimicrobial therapy reduces duration and stool shedding.
• Effective: tetracycline, TMP-SMX, fluoroquinolones, doxycycline.
• Children/pregnancy: erythromycin and/or azithromycin are appropriate choices.

Pandemics and spread

• Seven pandemics since 1817 have been recorded.
• Caused by classic O1 biotype except the seventh, which is O1 El Tor.
• Spread by mildly/early ill individuals, water, food, and flies.
• Only 1-5% of exposed susceptible persons develop disease.

Carriage and control

• Infected people shed only during first few days of illness.
• No long-term carriage in humans; seldom exceeds 3-4 weeks.
• Prevention: education, improved sanitation, patient isolation, disinfection.
• Antimicrobial treatment reduces symptoms and transmission; chemoprophylaxis for household contacts can limit spread.
• Cholera vaccines are available and advised for travel to endemic areas.

C) Non-O1 / non-O139 V. cholerae, V. parahaemolyticus, V. vulnificus

Non-O1 and non-O139 V. cholerae

• Generally not associated with classic epidemic cholera.
• Can cause mild diarrhea, cholera-like diarrhea, wound infection, or septicemia.
• Associated with contaminated water, seafood, or environmental exposure.
• Some strains produce toxins/virulence factors but usually lack classic O1/O139 + cholera toxin epidemic combination.
• Important in patients with comorbidities or aquatic exposure.

V. parahaemolyticus

• Halophilic Vibrio associated with marine environments.
• Usually follows raw/undercooked seafood, especially shellfish.
• Acute gastroenteritis: watery diarrhea, cramps, nausea, vomiting, sometimes fever.
• Some cases have dysentery-like features.
• Diagnosis by stool culture with appropriate selective methods; illness usually self-limited.

V. vulnificus

• Halophilic Vibrio associated with seawater and shellfish, especially oysters.
• Infection via contaminated raw oysters or wound exposure to seawater.
• GI symptoms may be followed by rapidly progressive septicemia.
• Wound infection can progress to cellulitis, bullae, necrotizing fasciitis, and septic shock.
• Severe disease risk: liver disease, alcoholism, hemochromatosis, immunosuppression, chronic illness.

4) Aeromonas and Campylobacter

A) Aeromonas

Basic features and habitat

• Ubiquitous inhabitants of fresh and brackish water.
• Gram-negative, facultative anaerobic rods.
• Ferment carbohydrates and may resemble Enterobacteriaceae.
• Key difference: Aeromonas are oxidase-positive.

Culture and species

• Grow on blood agar and enteric differential/selective agars.
• On blood agar they are usually beta-hemolytic.
• Main human species: A. hydrophila, A. caviae, A. veronii biovar sobria.

Gastroenteritis

• A. caviae is most frequently associated with gastroenteritis.
• Ranges from watery diarrhea to dysentery-like illness.
• Associated symptoms: abdominal pain, fever, nausea, vomiting.
• Follows ingestion of contaminated food or water, especially warm summer months.
• Usually self-limited; severe diarrhea/dehydration or children may require hospitalization.

Extraintestinal disease

• A. hydrophila causes wound infections after traumatic injury exposed to fresh/brackish water.
• Cellulitis usually develops within 48 hours.
• Fasciitis, myonecrosis, osteomyelitis, and systemic symptoms may occur.
• Soft tissue infection may rarely follow medicinal leech use.
• Sepsis mostly due to A. hydrophila, mainly in hematologic malignancy and/or liver disease.

Diagnosis and treatment

• Suggested by Gram-negative rods, oxidase positivity, growth on blood/enteric agars, and usually beta-hemolysis.
• Gastroenteritis usually needs no antimicrobials; severe cases may improve faster with treatment.
• Clinically significant aeromonads resist penicillin and ampicillin; often resist cefazolin and ticarcillin.
• Active options: 3rd-gen cephalosporins, aztreonam, carbapenems, aminoglycosides, especially fluoroquinolones.
• Empiric therapy may use two or more agents until susceptibility results are available.

B) Campylobacter - culture, diagnosis, disease, treatment, epidemiology

Culture requirements

• Requires microaerobic atmosphere: reduced O2 5-7% and increased CO2 10% for optimal growth.
• Most grow best at 42 C; growth can be seen between 36 C and 42 C.
• Primary plates for C. jejuni isolation should be incubated at 42 C.

Selective medium and colonies

• Skirrow medium contains vancomycin, polymyxin B, and trimethoprim to inhibit other bacteria.
• Colonies are generally colorless or gray.
• May be watery/spreading or round/convex.
• No hemolysis on blood-containing agar.

Biochemical/microscopy clues

• C. jejuni and C. coli are oxidase-positive and catalase-positive.
• Campylobacters do not oxidize or ferment carbohydrates.
• Gram stain: gull-wing-shaped rods.
• Dark-field/phase-contrast: darting motility.
• Culture on selective media is definitive for C. jejuni enteritis.
• Positive hippurate hydrolysis distinguishes C. jejuni from other Campylobacter species.

Disease

• Acquired orally from contaminated food, drink, poultry, or contact with infected animals.
• Multiply in small intestine, invade epithelium, cause inflammation with RBCs and WBCs in stool.
• C. jejuni and C. coli: most commonly gastroenteritis.
• C. fetus: bacteremia and extraintestinal infections in pregnant women and immunocompromised patients.
• C. jejuni gastroenteritis: crampy abdominal pain, profuse diarrhea that may be bloody, headache, malaise, fever.

Treatment and complications

• Illness is usually self-limited for 5-8 days.
• Recurrence occurs in about 5-10% of patients.
• Most cases resolve without antimicrobials.
• Macrolides, especially erythromycin, may be used and shorten fecal shedding.
• Postdiarrheal complications: Guillain-Barre syndrome, reactive arthritis, Reiter syndrome.

Reservoirs

• Reservoirs include poultry, cattle, sheep, pigs, birds, and pet dogs depending on species.

5) Helicobacter pylori

A) Features, identification, and pathogenesis

Basic features

• Spiral-shaped Gram-negative rod.
• Catalase-positive, oxidase-positive, and urease-positive.
• Motile with single and/or multiple monopolar, typically sheathed flagella.
• Humans are the primary host-reservoir.

Associated diseases

• Antral gastritis.
• Duodenal peptic ulcer disease.
• Gastric ulcers.
• Gastric adenocarcinoma.
• Gastric MALT lymphoma.

Culture identification

• Can be isolated from gastric biopsy specimens.
• Requires 37 C, microaerophilic humid atmosphere, usually 3-6 days incubation.
• If culture is negative, incubate up to 14 days.
• Primary isolation uses enriched media with blood/blood products such as chocolate agar, or antibiotic-containing media such as Skirrow medium.
• Colonies on blood agar range from gray to translucent.

Urease function

• Urease breaks urea into ammonia and CO2.
• This helps neutralize gastric acid and supports survival in the stomach.

Colonization and movement

• Colonizes gastric-type epithelial cells.
• Found deep in the mucous layer near epithelial surface.
• Flagella-mediated motility allows movement through gastric mucus toward epithelium.

Virulence factors and inflammation

• Tissue damage relates to mucinase, phospholipase, neutrophil-activating protein A, heat shock protein 60, CagA, and VacA.
• CagA induces IL-8 -> neutrophil attraction.
• VacA affects cell death/proliferation and activates IL-8-mediated acute inflammation.
• Histology: acute and chronic inflammation with polymorphonuclear and mononuclear infiltrates.
• Antimicrobial therapy clears H. pylori and improves gastritis and duodenal ulcer disease.

B) Clinical findings and diagnosis

Clinical findings

• Acute infection causes upper GI illness with nausea and pain.
• Vomiting and fever may also occur.
• Symptoms last less than 1 week to 2 weeks.
• After acute infection, colonization may persist for years, decades, or lifelong.
• About 90% of duodenal ulcer patients and 50-80% of benign gastric ulcer patients have H. pylori infection.
• Long colonization is associated with chronic gastritis, intestinal metaplasia, atrophic gastritis, and gastric adenocarcinoma.

Specimens and histology/culture

• Gastric biopsy specimens are used for histology or culture.
• Blood is used for serum antibodies.
• Stool is used for H. pylori antigen detection.
• Histology is generally more sensitive than culture.
• Hematoxylin and eosin show acute/chronic gastritis.
• Giemsa, silver, or immunohistochemical stains show curved or spiral organisms.
• Culture is mainly for patients not responding to treatment when susceptibility testing is needed.

6) H. pylori: tests, treatment, epidemiology + final comparison

A) Serology and other diagnostic tests

Antibodies	Active infection tests	Immunity
• IgG antibodies confirm exposure, but titers do not correlate with disease severity. • IgM disappears rapidly and has little diagnostic value. • IgA and IgG persist after eradication, so antibody testing has limited value for active infection or cure.	• Histology of gastric biopsy: 95-100% sensitivity and specificity. • Rapid urease test detects urease activity within 1-2 hours by pH color change. • Urea breath test detects labeled CO2 after 13C- or 14C-labeled urea; sensitivity/specificity 94-98%. • Stool antigen ELISA diagnoses active infection and is useful as a test of cure.	• Infection induces IgM first. • Persistent IgG and IgA occur in chronic infection.

B) Treatment

Triple therapy 7-14 days	Alternative quadruple therapy 10-14 days	Important notes
• PPI twice daily. • Amoxicillin 1 g twice daily. • Clarithromycin 500 mg twice daily.	• PPI. • Metronidazole 250 mg four times daily. • Tetracycline 500 mg four times daily. • Bismuth.	• Fourteen-day regimens eradicate H. pylori in 70-95% of patients. • PPIs directly inhibit H. pylori and act as potent urease inhibitors. • Recurrent/persistent infection may require tailored therapy for antimicrobial-resistant strains.

C) Epidemiology and control

Reservoir and transmission	Prevalence	Spread evidence/control
• Humans are likely the primary, if not sole reservoir. • Transmission is mainly oral-oral and/or fecal-oral. • Infection usually acquired in early childhood and may persist lifelong without antibiotics.	• Higher in developing countries. • In Southeast Asia, adult prevalence may reach up to 90%. • In the United States: fewer than 20% in persons younger than 30 years; increases to 40-60% in those 60 years and older.	• Person-to-person spread supported by intrafamilial clustering. • Rare transmission occurs via improperly cleaned endoscopes.

Final high-yield comparison table

Organism	Key clue	Disease anchor	Diagnosis anchor	Treatment anchor
V. cholerae	Comma-shaped; oxidase+; yellow on TCBS; O1/O139 epidemic	Rice-water stool, massive watery diarrhea, dehydration	Early stool; TCBS; alkaline peptone; shooting-star motility; O1/O139 antisera	Fluids/electrolytes first; tetracycline, doxycycline, TMP-SMX, fluoroquinolone; azithro/erythro in children/pregnancy
V. parahaemolyticus	Halophilic marine Vibrio; green on TCBS as non-sucrose fermenter	Seafood gastroenteritis, sometimes dysentery-like	Stool culture with selective methods	Usually self-limited
V. vulnificus	Seawater/oysters; high risk with liver disease/iron overload/immunosuppression	Septicemia; wound cellulitis, bullae, necrotizing fasciitis, shock	Exposure + culture/clinical suspicion	Urgent antimicrobial/surgical management as severe infection
Aeromonas	Fresh/brackish water; oxidase+; beta-hemolytic	Gastroenteritis; wounds after water trauma; sepsis in liver disease/hematologic malignancy	Gram-negative rod + oxidase+ + blood/enteric growth + beta-hemolysis	Usually no antibiotics for gastroenteritis; severe: fluoroquinolones, 3rd-gen cephalosporins, etc.
Campylobacter jejuni	Microaerophilic; 42 C; gull-wing rods; darting motility; hippurate+	Bloody/profuse diarrhea, cramps, fever; GBS/reactive arthritis/Reiter later	Selective culture definitive; Skirrow medium; oxidase/catalase+	Mostly self-limited; erythromycin/macrolide may shorten shedding
H. pylori	Spiral; catalase+, oxidase+, urease+; gastric mucus	Gastritis, duodenal/gastric ulcer, gastric adenocarcinoma, MALT lymphoma	Histology, rapid urease, urea breath, stool antigen; culture mainly for treatment failure	Triple or quadruple therapy with PPI-based regimen