Vibrio, Aeromonas & Plesiomonas
General Characteristics of Vibrio, Aeromonas and Plesiomonas

➢ Similarities to Enterobacteriaceae
  • Gram-negative
  • Facultative anaerobes
  • Fermentative bacilli

➢ Differences from Enterobacteriaceae
  • Polar flagella
  • Oxidase positive

➢ Formerly classified together as Vibrionaceae
  • Primarily found in water sources
  • Cause gastrointestinal disease
  • Shown not closely related by molecular methods
Morphology & Physiology of Vibrio

- Comma-shaped (vibrioid) bacilli
- *V. cholerae, V. parahaemolyticus, V. vulnificus* are most significant human pathogens
- Broad temperature & pH range for growth on media
  - 18-37°C
  - pH 7.0 - 9.0 (useful for enrichment)
- Grow on variety of simple media including:
  - MacConkey’s agar
  - TCBS (Thiosulfate Citrate Bile salts Sucrose) agar
- *V. cholerae* grow without salt
  - Most other vibrios are halophilic
<table>
<thead>
<tr>
<th>Species</th>
<th>Source of Infection</th>
<th>Clinical Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>V. cholerae</em></td>
<td>Water, food</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td><em>V. parahaemolyticus</em></td>
<td>Shellfish, seawater</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. vulnificus</em></td>
<td>Shellfish, seawater</td>
<td>Bacteremia, wound infection, cellulitis</td>
</tr>
<tr>
<td><em>V. alginolyticus</em></td>
<td>Seawater</td>
<td>Wound infection, external otitis</td>
</tr>
<tr>
<td><em>V. hollisae</em></td>
<td>Shellfish</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. fluvialis</em></td>
<td>Seafood</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. damsela</em></td>
<td>Seawater</td>
<td>Wound infection</td>
</tr>
<tr>
<td><em>V. metschnikovii</em></td>
<td>Unknown</td>
<td>Bacteremia</td>
</tr>
<tr>
<td><em>V. mimicus</em></td>
<td>Fresh water</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. furnissii</em></td>
<td>Seawater</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td><em>V. cincinnatiensis</em></td>
<td>Unknown</td>
<td>Bacteremia, meningitis</td>
</tr>
<tr>
<td><em>V. carchariae</em></td>
<td>Seawater</td>
<td>Wound (shark bite)</td>
</tr>
</tbody>
</table>
Epidemiology of Vibrio spp.

- *Vibrio* spp. (including *V. cholerae*) grow in estuarine and marine environments worldwide
- All *Vibrio* spp. can survive and replicate in contaminated waters with increased salinity and at temperatures of 10-30°C
- Pathogenic *Vibrio* spp. appear to form symbiotic (?) associations with chitinous shellfish which serve as an important and only recently recognized reservoir
- Asymptomatically infected humans also serve as an important *reservoir* in regions where cholera is endemic
Taxonomy of Vibrio cholerae

➢ >200 serogroups based on somatic O-antigen

➢ O1 and O139 serogroups are responsible for classic epidemic cholera

➢ O1 serogroup subdivided into
  • Two biotypes: El Tor and classical (or cholerae)
  • Three serotypes: ogawa, inaba, hikojima

➢ Some O1 strains do not produce cholera enterotoxin (atypical or nontoxigenic O1 V. cholerae)

➢ Other strains are identical to O1 strains but do not agglutinate in O1 antiserum (non-cholera (NCV) or non-agglutinating (NAG) vibrios) (non-O1 V. cholerae)

➢ Several phage types
Epidemiology of Vibrio cholerae

- Cholera recognized for more than two millennia with sporadic disease and epidemics
- **Endemic** in regions of Southern and Southeastern Asia; origin of pandemic cholera outbreaks
- Generally in communities with poor sanitation
- Seven pandemics (possible beginning of 8th) since 1817 attributable to increased world travel
- Cholera spread by contaminated water and food
- Human carriers and environmental reservoirs
Recent Cholera Pandemics

7th pandemic:
- *V. cholerae* O1 biotype El Tor
- Began in Asia in 1961
- Spread to other continents in 1970s and 1980s
- Spread to Peru in 1991 and then to most of South & Central America and to U.S. & Canada
- By 1995 in the Americas, >10^6 cases; 10^4 dead

8th pandemic (??)
- *V. cholerae* O139 Bengal is first non-O1 strain capable of causing epidemic cholera
- Began in India in 1992 and spread to Asia, Europe and U.S.
- Disease in humans previously infected with O1 strain, thus no cross-protective immunity
Pathogenesis of V. cholerae

➢ Incubation period: 2-3 days

➢ High infectious dose: $>10^8$ CFU
  - $10^3$-$10^5$ CFU with achlorhydria or hypochlorhydria (lack of or reduced stomach acid)

➢ Abrupt onset of vomiting and life-threatening watery diarrhea (15-20 liters/day)

➢ As more fluid is lost, feces-streaked stool changes to rice-water stools:
  - Colorless
  - Odorless
  - No protein
  - Speckled with mucus
Pathogenesis of *V. cholerae* (cont.)

- Cholera toxin leads to profuse loss of **fluids and electrolytes** (sodium, potassium, bicarbonate)
  - **Hypokalemia** (low levels of K in blood)
  - Cardiac **arrhythmia** and **renal failure**
- Cholera toxin **blocks uptake of sodium & chloride** from lumen of small intestine

- **Death attributable to:**
  - **Hypovolemic shock** (due to abnormally low volume of circulating fluid (plasma) in the body)
  - **Metabolic acidosis** (pH shifts toward acid side due to loss of bicarbonate buffering capacity)
Treatment & Prevention of V. cholerae

➢ Untreated: 60% fatality
➢ Treated: <1% fatality
➢ Rehydration & supportive therapy
  • Oral
    Sodium chloride (3.5 g/L)
    + Potassium chloride (1.5 g/L)
    + Rice flour (30-80g/L)
    + Trisodium citrate (2.9 g/L)
  • Intravenous (IV)
➢ Doxycycline or tetracycline (Tet resistance may be developing) of secondary value
➢ Water purification, sanitation & sewage treatment
➢ Vaccines
## Virulence Factors Associated with Vibrio cholerae O1 and O139

<table>
<thead>
<tr>
<th>Virulence Factor</th>
<th>Biologic Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholera toxin</td>
<td>Hypersecretion of electrolytes and water</td>
</tr>
<tr>
<td>Coregulated pilus</td>
<td>Adherence to mucosal cells</td>
</tr>
<tr>
<td>Accessory colonization</td>
<td>Adhesin factor</td>
</tr>
<tr>
<td>Hemagglutination-protease (mucinase)</td>
<td>Induces intestinal inflammation and degradation of tight junctions</td>
</tr>
<tr>
<td>Siderophores</td>
<td>Iron sequestration</td>
</tr>
<tr>
<td>Neuraminidase</td>
<td>Increase toxin receptors</td>
</tr>
</tbody>
</table>
Two Broad Classes of Bacterial Exotoxins

- **Intracellular Targets**: A-B dimeric (two domain) exotoxins: (prototype is diphtheria toxin of *Corynebacterium diphtheriae*):
  - Bipartite structure: Binding domain (B) associated with absorption to target cell surface and transfer of active component (A) across cell membrane; once internalized, domain (A) enzymatically disrupts cell function
  - Receptor-mediated endocytosis (host cell uptake and internalization of exotoxin)
  - ADP-ribosylation of intracellular target host molecule

- **Cellular Targets**: Cytolytic exotoxins (usually degradative enzymes) or cytolysins: hemolysis, tissue necrosis, may be lethal when administered intravenously
Cholera Toxin (A2-5B)(Vibrio cholerae)

➢ Chromosomally-encoded; Lysogenic phage conversion; Highly conserved genetic sequence
➢ Structurally & functionally similar to ETEC LT
➢ B-subunit binds to GM₁ ganglioside receptors in small intestine
➢ Reduction of disulfide bond in A-subunit activates A₁ fragment that ADP-ribosylates guanosine triphosphate (GTP)-binding protein (Gₛ) by transferring ADP-ribose from nicotinamide adenine dinucleotide (NAD)
➢ ADP-ribosylated GTP-binding protein activates adenyl cyclase leading to an increased cyclic AMP (cAMP) level and hypersecretion of fluids and electrolytes
Mechanism of Action of Cholera Toxin

NOTE: In step #4, uptake of $\text{Na}^+$ and $\text{Cl}^-$ from the lumen is also blocked.

$\text{HCO}_3^-$ = bicarbonate which provides buffering capacity.
Mechanism of Action of Cholera Toxin

1. *V. cholerae* 
2. Cholera toxin binds to the cell membrane via a ganglioside receptor.
3. Activation of adenylate cyclase, increasing cAMP levels.
4. Increased ion transport (Na+, H2O, Cl-, K+, HCO3-) leading to Diarrhea and Loss of cell nutrients.

Diagram:
- A1 and A2 subunits of the cholera toxin complex.
- Binding of the toxin to the ganglioside receptor.
- Activation of adenylate cyclase, indicated by an increase in cAMP levels.
### TABLE 19-3 Properties of A-B Type Bacterial Toxins

<table>
<thead>
<tr>
<th>TOXIN</th>
<th>ORGANISM</th>
<th>GENETIC CONTROL</th>
<th>SUBUNIT STRUCTURE</th>
<th>TARGET CELL RECEPTOR</th>
<th>BIOLOGICAL EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthrax toxins</td>
<td>Bacillus anthracis</td>
<td>Plasmid</td>
<td>Three separate proteins (EF, LF, PA)</td>
<td>Unknown, probably glycoprotein</td>
<td>Increase in target-cell cAMP level, localized edema; LF + PA: death of target cells and experimental animals</td>
</tr>
<tr>
<td>Bordetella adenylate cyclase toxin</td>
<td>Bordetella species</td>
<td>Chromosomally</td>
<td>A-B</td>
<td>Unknown, probably glycolipid</td>
<td>Increase in target cell cAMP level, modified cell function or cell death</td>
</tr>
<tr>
<td>Botulinum toxin</td>
<td>C. botulinum</td>
<td>Phage</td>
<td>A-B</td>
<td>Possibly ganglioside (GD₁₆)</td>
<td>Decrease in peripheral, presynaptic acetylcholine release, flaccid paralysis</td>
</tr>
<tr>
<td>Cholera toxin</td>
<td>V. cholerae</td>
<td>Chromosomally</td>
<td>A-5B</td>
<td>Ganglioside (GM₁)</td>
<td>Activation of adenylate cyclase, increase in cAMP level, secretory diarrhea</td>
</tr>
<tr>
<td>Diphtheria toxin</td>
<td>C. diphtheriae</td>
<td>Phage</td>
<td>A-B</td>
<td>Probably glycoprotein</td>
<td>Inhibition of protein synthesis, cell death</td>
</tr>
<tr>
<td>Heat-labile enterotoxins</td>
<td>E. coli</td>
<td>Plasmid</td>
<td>Similar or identical to cholera toxin</td>
<td>Probably glycoprotein</td>
<td>Block of signal transduction mediated by target G proteins</td>
</tr>
<tr>
<td>Pertussis toxin</td>
<td>B. pertussis</td>
<td>Chromosomally</td>
<td>A-5B</td>
<td>Unknown, probably glycoprotein</td>
<td>Similar or identical to diphtheria toxin</td>
</tr>
<tr>
<td><em>Pseudomonas</em> exotoxin A</td>
<td><em>P. aeruginosa</em></td>
<td>Chromosomally</td>
<td>A-B</td>
<td>Unknown, but different from diphtheria toxin</td>
<td>Inhibition of protein synthesis, cell death</td>
</tr>
<tr>
<td>Shiga toxin</td>
<td><em>Shigella dysenteriae</em></td>
<td>Chromosomally</td>
<td>A-5B</td>
<td>Glycoprotein or glycolipid</td>
<td>Decrease in neurotransmitter release from inhibitory neurons, spastic paralysis</td>
</tr>
<tr>
<td>Shiga-like toxins</td>
<td><em>Shigella</em> species, E. coli</td>
<td>Phage</td>
<td>Similar or identical to Shiga toxin</td>
<td>Ganglioside (GT₁) and/or GD₁₆</td>
<td></td>
</tr>
<tr>
<td>Tetanus toxin</td>
<td>C. tetani</td>
<td>Plasmid</td>
<td>A-B</td>
<td>Unknown, probably glycoprotein</td>
<td></td>
</tr>
</tbody>
</table>
Summary of *Vibrio parahaemolyticus* Infections

**Physiology and Structure**
Curved gram-negative bacilli.
- Facultative anaerobe.
- Fermenter.
- Simple nutritional requirements but requires salt for growth.

**Virulence**
Refer to Table 30-3 for complete listing.
- Hemolysin.
- Adhesin.

**Epidemiology**
Organism found in estuarine and marine environments worldwide.
- Associated with consumption of contaminated shellfish.
- Not commonly isolated in the United States but is a major pathogen in countries where raw fish is eaten.

**Diseases**
Diarrhea ranging from mild disease to a cholera-like illness.
- Typical presentation is an explosive, watery diarrhea.
- Less commonly associated with wound infections and bacteremia.

**Diagnosis**
Culture should be performed as with *V. cholerae*.

**Treatment, Prevention, and Control**
Self-limited disease, although antibiotics can shorten symptoms and fluid loss.
- Disease prevented by proper cooking of shellfish.
- No vaccines are available.
Summary of Vibrio vulnificus Infections

**Diseases**
- Wound infections that can progress rapidly to formation of bullae and tissue necrosis.
- Septicemia following ingestion of contaminated shellfish.
- Infection associated with exposure of a wound to contaminated salt water or ingestion of improperly prepared shellfish.

**Epidemiology**
- High mortality rate in immunocompromised patients.
- Septicemia following ingestion of contaminated shellfish.

**Virulence**
- Resistant to complement- and antibody-mediated serum killing (thus systemic infections).
- Production of hydrolytic enzymes (collagenase, proteases).

**Physiology and Structure**
- Simple nutritional requirements but requires salt for growth.
- Simple nutritional requirements but requires salt for growth.
- Curved gram-negative bacilli. Facultative anaerobe.

**Fermenter**
- Simple nutritional requirements but requires salt for growth.
- Simple nutritional requirements but requires salt for growth.
- Curved gram-negative bacilli. Facultative anaerobe.
### Virulence Factors Associated with Non-cholerae Vibrios

<table>
<thead>
<tr>
<th>Organism</th>
<th>Virulence Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>V. parahaemolyticus</em></td>
<td>Thermostable direct hemolysin</td>
</tr>
<tr>
<td><em>V. vulnificus</em></td>
<td>Serum resistance, antiphagocytic polysaccharides, cytolysins, collagenase, protease, siderophore</td>
</tr>
<tr>
<td><em>V. alginolyticus</em></td>
<td>Collagenase</td>
</tr>
<tr>
<td><em>V. bollisae</em></td>
<td>Heat-stable and heat-labile enterotoxin, hemolysin</td>
</tr>
<tr>
<td><em>V. damsela</em></td>
<td>Cytolysin</td>
</tr>
</tbody>
</table>

(Kanagawa positive)
Laboratory Identification of Vibrios

➢ Transport medium - Cary-Blair semi-solid agar
➢ Enrichment medium - alkaline peptone broth
  • Vibrios survive and replicate at high pH
  • Other organisms are killed or do not multiply
➢ Selective/differential culture medium - TCBS agar
  • \textit{V. cholerae} grow as yellow colonies
➢ Biochemical and serological tests
Characteristics and Epidemiology of Aeromonas (Family Aeromonadaceae)

- Gram-negative facultatively anaerobic bacillus resembling members of the Enterobacteriaceae
- Motile species have **single polar flagellum** (nonmotile species apparently not associated with human disease)
- 16 phenospecies: Most significant human pathogens *A. hydrophila*, *A. caviae*, *A. veronii* biovar *sobria*
- Ubiquitous in **fresh and brackish water**
- Acquired by **ingestion of or exposure to contaminated water or food**
Clinical Syndromes of Aeromonas

- Associated with gastrointestinal disease
  - Chronic diarrhea in adults
  - Self-limited acute, severe disease in children resembling shigellosis with blood and leukocytes in the stool
  - 3% carriage rate

- Wound infections

- Opportunistic systemic disease in immunocompromised

- Putative virulence factors include: endotoxin; hemolysins; enterotoxin; proteases; siderophores; adhesins
Afimbriated Aeromonas hydrophila

Nonadherent Afimbriated Bacterial Cells and Buccal Cells
Adherent Fimbriated Bacterial Cells and Buccal Cells

Fimbriated *Aeromonas hydrophila*
Characteristics of Plesiomonas

➢ Formerly Plesiomonadaceae
➢ Closely related to *Proteus* & now classified as Enterobacteriaceae despite differences:
  • Oxidase positive
  • Multiple polar flagella (*lophotrichous*)
➢ Single species: *Plesiomonas shigelloides*
➢ Isolated from *aquatic environment* (fresh or estuarine)
➢ Acquired by *ingestion of or exposure to contaminated water or seafood* or by exposure to amphibians or reptiles
➢ Self-limited gastroenteritis: secretory, colitis or chronic forms
➢ Variety of uncommon *extra-intestinal infections*
### Characteristics of Aeromonas and Plesiomonas Gastroenteritis

<table>
<thead>
<tr>
<th>Epidemiological Features</th>
<th>Aeromonas</th>
<th>Plesiomonas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Natural Habitat Source of Infection</strong></td>
<td>Fresh or brackish water Contaminated water or food</td>
<td>Fresh or brackish water Contaminated water or food</td>
</tr>
<tr>
<td><strong>Clinical Features</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Abdominal Cramps</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Fever</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Blood/WBCs in Stool</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Pathogenesis</strong></td>
<td>Enterotoxin (???)</td>
<td>Invasiveness</td>
</tr>
</tbody>
</table>
REVIEW
**Vibrio spp. (Family Vibrionaceae) Associated with Human Disease**

<table>
<thead>
<tr>
<th>Species</th>
<th>Source of Infection</th>
<th>Clinical Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>V. cholerae</em></td>
<td>Water, food</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td><em>V. parahaemolyticus</em></td>
<td>Shellfish, seawater</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. vulnificus</em></td>
<td>Shellfish, seawater</td>
<td>Bacteremia, wound infection, cellulitis</td>
</tr>
<tr>
<td><em>V. alginolyticus</em></td>
<td>Seawater</td>
<td>Wound infection, external otitis</td>
</tr>
<tr>
<td><em>V. bollisae</em></td>
<td>Shellfish</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. fluvialis</em></td>
<td>Seafood</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. damsela</em></td>
<td>Seawater</td>
<td>Wound infection</td>
</tr>
<tr>
<td><em>V. metschnikovii</em></td>
<td>Unknown</td>
<td>Bacteremia</td>
</tr>
<tr>
<td><em>V. mimicus</em></td>
<td>Fresh water</td>
<td>Gastroenteritis, wound infection, bacteremia</td>
</tr>
<tr>
<td><em>V. furnissii</em></td>
<td>Seawater</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td><em>V. cincinnatiensis</em></td>
<td>Unknown</td>
<td>Bacteremia, meningitis</td>
</tr>
<tr>
<td><em>V. carchariae</em></td>
<td>Seawater</td>
<td>Wound (shark bite)</td>
</tr>
</tbody>
</table>
Epidemiology of Vibrio spp.

➢ *Vibrio* spp. (including *V. cholerae*) grow in estuarine and marine environments worldwide.

➢ All *Vibrio* spp. can survive and replicate in contaminated waters with increased salinity and at temperatures of 10-30°C.

➢ Pathogenic *Vibrio* spp. appear to form symbiotic (?) associations with chitinous shellfish which serve as an important and only recently recognized reservoir.

➢ Asymptomatically infected humans also serve as an important reservoir in regions where cholera is endemic.
Taxonomy of Vibrio cholerae

- >200 serogroups based on somatic O-antigen
- **O1** and **O139 serogroups** are responsible for classic epidemic cholera
- O1 serogroup subdivided into
  - **Two biotypes**: El Tor and **classical** (or cholerae)
  - **Three serotypes**: ogawa, inaba, hikojima
- Some O1 strains do not produce cholera enterotoxin (atypical or nontoxigenic O1 *V. cholerae*)
- Other strains are identical to O1 strains but do not agglutinate in O1 antiserum (non-cholera (NCV) or non-agglutinating (NAG) vibrios) (non-O1 *V. cholerae*)
- Several phage types
Epidemiology of Vibrio cholerae

- Cholera recognized for more than two millennia with sporadic disease and epidemics
- **Endemic** in regions of Southern and Southeastern Asia; origin of pandemic cholera outbreaks
- Generally in communities with **poor sanitation**
- Seven pandemics (possible beginning of 8\(^{th}\)) since 1817 attributable to increased world travel
- Cholera spread by **contaminated water and food**
- Human carriers and environmental reservoirs
Vibrio cholerae Infections

Summary of

Organism can multiply freely in water. Organisms found in estuarine and marine environments worldwide (including along the coast of the United States) associated with chitinous shellfish.

Epidemiology

Organism responsible for major pandemics (worldwide pandemics), with significant mortality in underdeveloped countries. All pandemics of cholera caused by serotype O1, although O139 can cause similar diseases and may cause a pandemic.

Epidemiology

Cholera toxin is primarily responsible for the watery diarrhea characteristic of this species. Adherence factors are important for establishing the initial colonization in the intestines, permitting the toxin to function.

Virulence

Refer to Table 30–2 for complete listing.

Two biotypes of V. cholera O1 strains—El tor and classical (this is important for epidemiologic classification of isolates).

Simple nutritional requirements; do not require salt for growth but can tolerate it. Strains subdivided by their O cell wall antigens.

Facultative anaerobe. Curved gram-negative bacilli.
Bacterial levels increase in contaminated waters during the warm months.
Spread by consumption of contaminated food or water.
Direct person-to-person spread is rare because the infectious dose is high.
The infectious dose is high because most organisms are killed by stomach acids.

**Disease**
Cholera.
Presentation can range from mild disease to severe life-threatening disease.
Disease is characterized by profuse watery diarrhea.
Death is caused by electrolyte abnormalities and massive fluid loss.

**Diagnosis**
Culture should be performed early in course of disease with fresh stool specimens.

**Treatment, Prevention, and Control**
Fluid and electrolyte replacement are crucial.
Antibiotic therapy reduces the bacterial burden and exotoxin production, as well as duration of diarrhea.
Doxycycline (adults), trimethoprim-sulfamethoxazole (children), or furazolidone (pregnant women) is administered.
Improved hygiene is critical for control.
The killed parenteral vaccine is of no value, but the newer oral vaccine has some protective value.
Pathogenesis of V.cholerae (cont.)

➢ Cholera toxin leads to profuse loss of fluids and electrolytes (sodium, potassium, bicarbonate)
  • Hypokalemia (low levels of K in blood)
  • Cardiac arrhythmia and renal failure

➢ Cholera toxin blocks uptake of sodium & chloride from lumen of small intestine

➢ Death attributable to:
  • Hypovolemic shock (due to abnormally low volume of circulating fluid (plasma) in the body)
  • Metabolic acidosis (pH shifts toward acid side due to loss of bicarbonate buffering capacity)
Virulence Factors Associated with Vibrio cholerae O1 and O139

<table>
<thead>
<tr>
<th>Virulence Factor</th>
<th>Biologic Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholera toxin</td>
<td>Hypersecretion of electrolytes and water</td>
</tr>
<tr>
<td>Coregulated pilus</td>
<td>Adherence to mucosal cells</td>
</tr>
<tr>
<td>Accessory colonization</td>
<td>Adhesin factor</td>
</tr>
<tr>
<td>Hemagglutination-protease (mucinase)</td>
<td>Induces intestinal inflammation and degradation of tight junctions</td>
</tr>
<tr>
<td>Siderophores</td>
<td>Iron sequestration</td>
</tr>
<tr>
<td>Neuraminidase</td>
<td>Increase toxin receptors</td>
</tr>
</tbody>
</table>
Mechanism of Action of Cholera Toxin

- **V. cholerae** binds to ganglioside receptor.
- Cholera toxin binds to receptor and enters cell.
- A1 catalyzes the conversion of GTP to GDP, increasing adenylate cyclase activity.
- Increased cAMP leads to increased fluid and electrolyte secretion and diarrhea.

**Diarrhea**
- Loss of cell nutrients

**Cell membrane**
- Sodium ($\text{Na}^+$), water ($\text{H}_2\text{O}$), chloride ($\text{Cl}^-$), potassium ($\text{K}^+$), bicarbonate ($\text{HCO}_3^-$) transport.
Summary of Vibrio parahaemolyticus Infections

Physiology and Structure
Curved gram-negative bacilli.
  Facultative anaerobe.
  Fermenter.
  Simple nutritional requirements but requires salt for growth.

Virulence
Refer to Table 30-3 for complete listing.
  Hemolysin.
  Adhesin.

Epidemiology
Organism found in estuarine and marine environments worldwide.
  Associated with consumption of contaminated shellfish.
  Not commonly isolated in the United States but is a major pathogen in countries where raw fish is eaten.

Diseases
Diarrhea ranging from mild disease to a cholera-like illness.
  Typical presentation is an explosive, watery diarrhea.
  Less commonly associated with wound infections and bacteremia.

Diagnosis
Culture should be performed as with V. cholerae.

Treatment, Prevention, and Control
Self-limited disease, although antibiotics can shorten symptoms and fluid loss.
  Disease prevented by proper cooking of shellfish.
  No vaccines are available.
Summary of Vibrio vulnificus Infections

Physiology and Structure
Curved gram-negative bacilli.
Facultative anaerobe.
Fermenter.
Simple nutritional requirements but requires salt for growth.

Virulence
Refer to Table 30–3 for complete listing.
Resistant to complement- and antibody-mediated serum killing (thus, systemic infections).
Antiphagocytic capsule.
Production of hydrolytic enzymes (cytolysins, collagenase, proteases).

Epidemiology
Infection associated with exposure of a wound to contaminated salt water or ingestion of improperly prepared shellfish.

Diseases
Wound infections that can progress rapidly to formation of bullae and tissue necrosis.
Septicemia following ingestion of contaminated shellfish.
High mortality rate in immunocompromised patients.

Diagnosis
Culture wounds and blood.

Treatment, Prevention, and Control
Life-threatening illnesses that must be promptly treated with antibiotics.
Tetracyclines or aminoglycosides treatment of choice.
No vaccine is available.
## Virulence Factors Associated with Non-cholerae Vibrios

<table>
<thead>
<tr>
<th>Organism</th>
<th>Virulence Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>V. parahaemolyticus</em></td>
<td>Thermostable direct hemolysin</td>
</tr>
<tr>
<td><em>V. vulnificus</em></td>
<td>Serum resistance, antiphagocytic polysaccharides, cytolysins, collagenase, protease, siderophore</td>
</tr>
<tr>
<td><em>V. alginolyticus</em></td>
<td>Collagenase</td>
</tr>
<tr>
<td><em>V. hollisae</em></td>
<td>Heat-stable and heat-labile enterotoxin, hemolysin</td>
</tr>
<tr>
<td><em>V. damsela</em></td>
<td>Cytolysin</td>
</tr>
</tbody>
</table>

(Kanagawa positive)
Characteristics and Epidemiology of Aeromonas (Family Aeromonadaceae)

➢ Gram-negative facultatively anaerobic bacillus resembling members of the Enterobacteriaceae

➢ Motile species have single polar flagellum (nonmotile species apparently not associated with human disease)

➢ 16 phenospecies: Most significant human pathogens *A. hydrophila, A. caviae, A. veronii biovar sobria*

➢ Ubiquitous in fresh and brackish water

➢ Acquired by ingestion of or exposure to contaminated water or food
Clinical Syndromes of Aeromonas

- Associated with gastrointestinal disease
  - Chronic diarrhea in adults
  - Self-limited acute, severe disease in children resembling shigellosis with blood and leukocytes in the stool
  - 3% carriage rate

- Wound infections

- Opportunistic systemic disease in immunocompromised

- Putative virulence factors include: endotoxin; hemolysins; enterotoxin; proteases; siderophores; adhesins
Characteristics of Plesiomonas

➢ Formerly Plesiomonadaceae
➢ Closely related to *Proteus* & now classified as Enterobacteriaceae despite differences:
  • Oxidase positive
  • Multiple polar flagella (*lophotrichous*)
➢ Single species: *Plesiomonas shigelloides*
➢ Isolated from aquatic environment (fresh or estuarine)
➢ Acquired by ingestion of or exposure to contaminated water or seafood or by exposure to amphibians or reptiles
➢ Self-limited gastroenteritis: secretory, colitis or chronic forms
➢ Variety of uncommon extra-intestinal infections
# Characteristics of Aeromonas and Plesiomonas Gastroenteritis

<table>
<thead>
<tr>
<th>Epidemiologic and Clinical Features</th>
<th>Aeromonas</th>
<th>Plesiomonas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural habitat</td>
<td>Fresh or brackish water</td>
<td>Fresh or brackish water</td>
</tr>
<tr>
<td>Source of infection</td>
<td>Contaminated food or water</td>
<td>Contaminated food or water; contact with amphibians or reptiles</td>
</tr>
<tr>
<td>Clinical presentation:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Abdominal cramps</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Fever</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Blood/leukocytes in stool</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Pathogenesis</td>
<td>Enterotoxin (?)</td>
<td>Invasive</td>
</tr>
</tbody>
</table>