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

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

Pathogenesis of V.cholerae Incubation period: 2-3 days High infectious dose: >108 CFU • 10^{3-10⁵} 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 \triangleright 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)

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
 - V. cholerae grow as yellow colonies
- Biochemical and serological tests

Treatment & Prevention of V. cholerae

- Untreated: 60% fatality
- Treated: <1% fatality</p>
- 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

Virulence Factor

Biologic Effect

Cholera toxin

Coregulated pilus Accessory colonization Hemagglutinationprotease (mucinase) Siderophores Neuraminidase

Hypersecretion of electrolytes and water Adherence to mucosal cells Adhesin factor

Induces intestinal inflammation and degradation of tight junctions Iron sequestration Increase toxin receptors

Two Broad Classes of Bacterial Exotoxins Intracellular Targets: A-B dimeric (two domain) exotoxins: 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 (G_s) 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



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.

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*
- 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; eneterotoxin; 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