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 (**T**hiosulfate **C**itrate **B**ile salts **S**ucrose) agar
- ***V. cholerae* grow without salt**
  - Most **other vibrios are halophilic**

Vibrio spp. (Family Vibrionaceae) *Associated with Human Disease*

Species	Source of Infection	Clinical Disease
V. cholerae	Water, food	Gastroenteritis
V. parahaemolyticus	Shellfish, seawater	Gastroenteritis, wound infection, bacteremia
V. vulnificus	Shellfish, seawater	Bacteremia, wound infection, cellulitis
V. alginolyticus	Seawater	Wound infection, external otitis
V. hollisae	Shellfish	Gastroenteritis, wound infection, bacteremia
V. fluvialis	Seafood	Gastroenteritis, wound infection, bacteremia
V. damsela	Seawater	Wound infection
V. metschnikovii	Unknown	Bacteremia
V. mimicus	Fresh water	Gastroenteritis, wound infection, bacteremia
*V. furnissii**	Seawater	Gastroenteritis
*V. cincinnatiensis**	Unknown	Bacteremia, meningitis
*V. carchariae**	Seawater	Wound (shark bite)

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 10-30ºC**
- 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

Virulence Factor	Biologic Effect
Cholera toxin	Hypersecretion of electrolytes and water
Coregulated pilus	Adherence to mucosal cells
Accessory colonization	Adhesin factor
Hemagglutination-protease (mucinase)	Induces intestinal inflammation and degradation of tight junctions
Siderophores	Iron sequestration
Neuraminidase	Increase toxin receptors

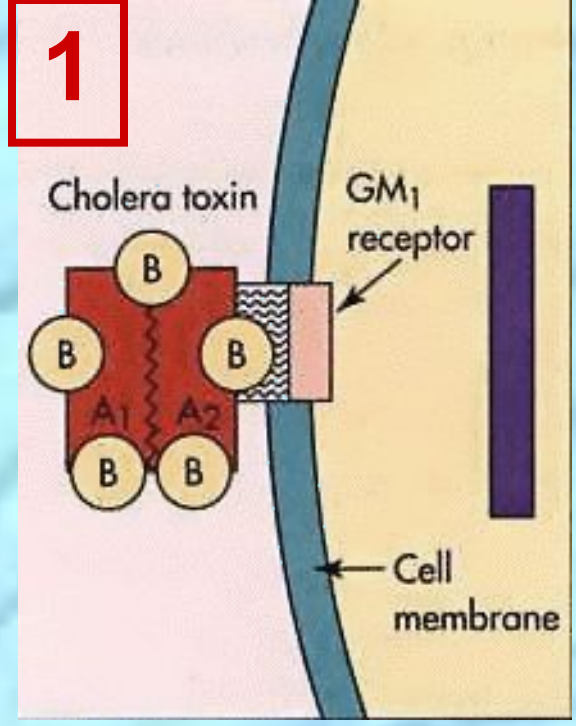
Two Broad Classes of Bacterial Exotoxins

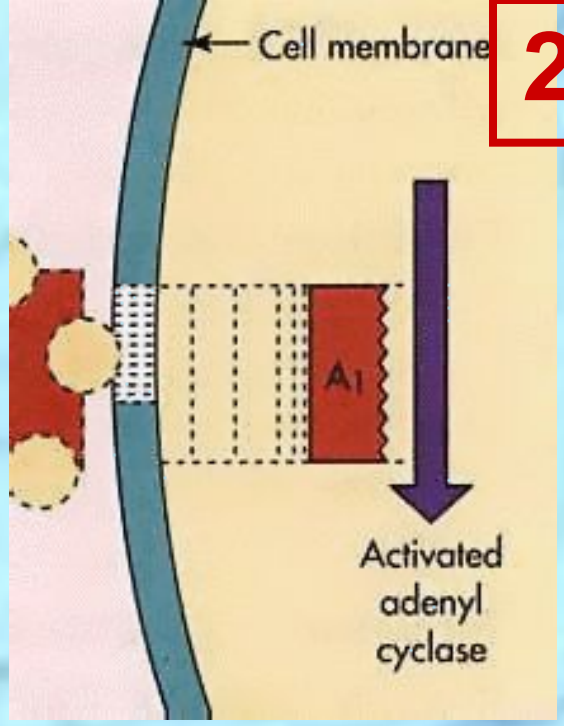
- **Intracellular Targets: A-B dimeric (two domain) exotoxins:** (prototype is diphtheria toxin of *Corynebacterium diphtheriae*):
  - **Bipartite structure:** Binding domain (B) associated with absorption to target cell surface and transfer of active component (A) across cell membrane; once internalized, 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 **GM_1 ganglioside receptors** in small intestine
- Reduction of disulfide bond in A-subunit activates A_1 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**

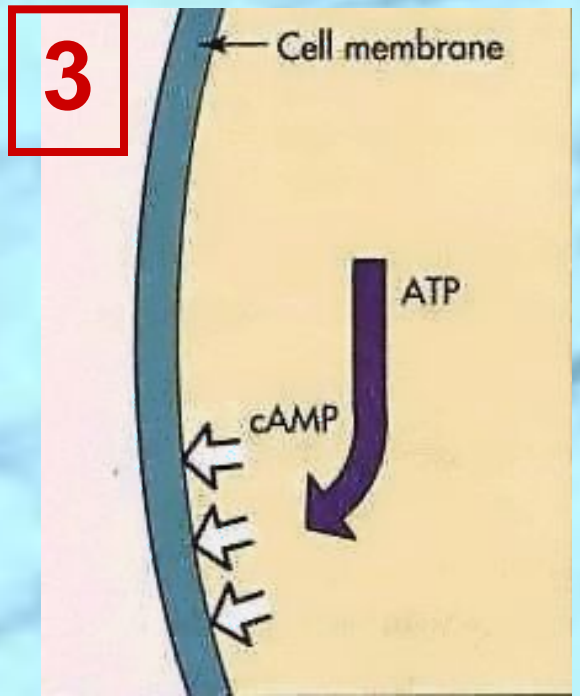
Mechanism of Action of Cholera Toxin

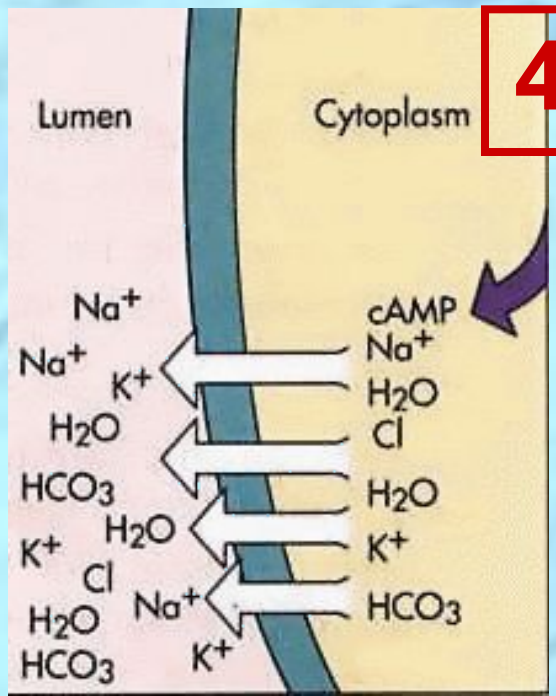




NOTE: In step #4, uptake of Na^+ and Cl^- from the lumen is also blocked.

HCO_3^- = bicarbonate which provides buffering capacity.





Mechanism of Action of Cholera Toxin

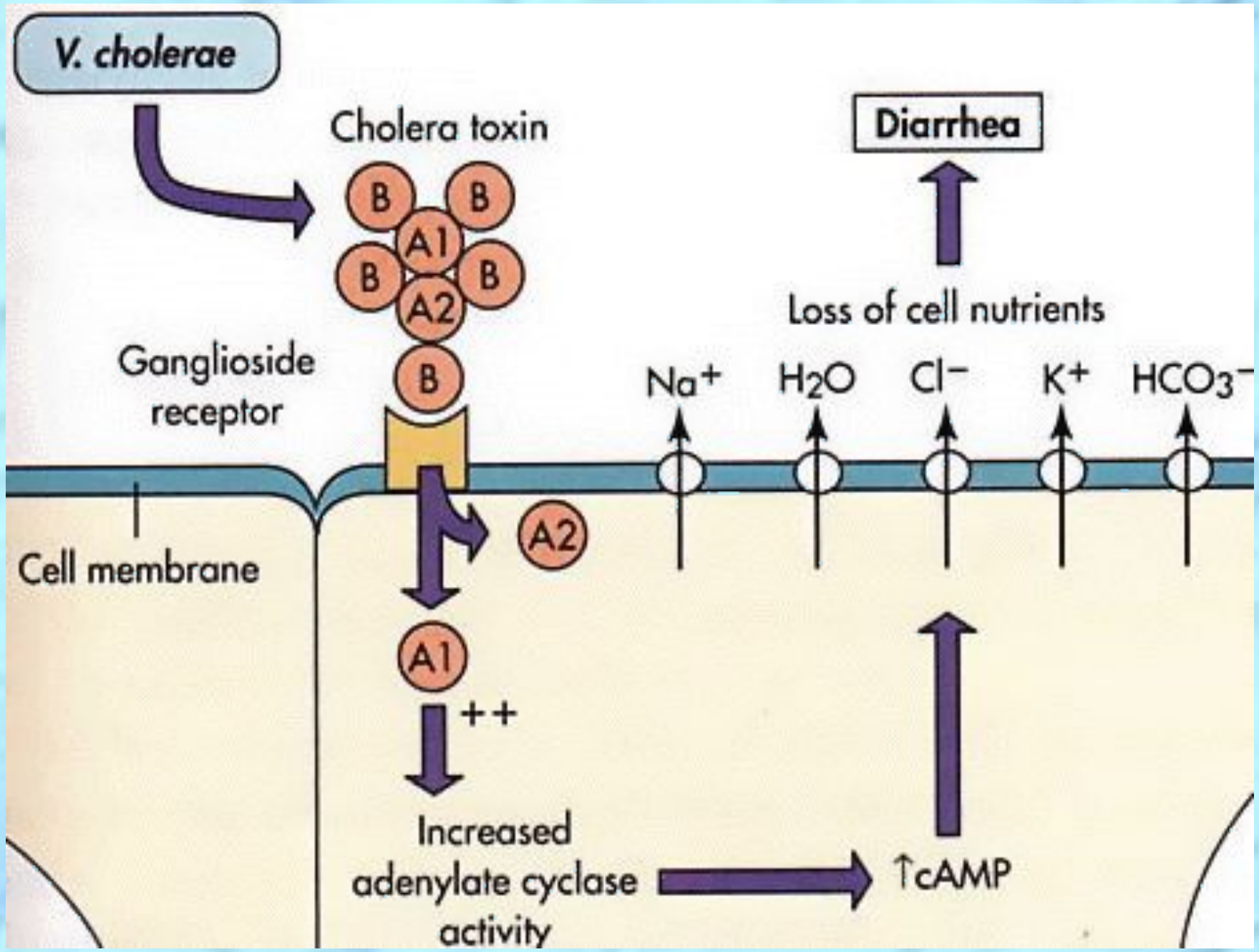


TABLE 19-3 Properties of A-B Type Bacterial Toxins

TOXIN	ORGANISM	GENETIC CONTROL	SUBUNIT STRUCTURE	TARGET CELL RECEPTOR	BIOLOGICAL EFFECTS
Anthrax toxins	*Bacillus anthracis*	Plasmid	Three separate proteins (EF, LF, PA)	Unknown, probably glycoprotein	EF + PA: increase in target-cell cAMP level, localized edema; LF + PA: death of target cells and experimental animals
Bordetella adenylate cyclase toxin	*Bordetella* species	Chromosomal	A-B	Unknown, probably glycolipid	Increase in target cell cAMP level, modified cell function or cell death
Botulinum toxin	*C. botulinum*	Phage	A-B	Possibly ganglioside (GD_{1b})	Decrease in peripheral, presynaptic acetylcholine release, flaccid paralysis
Cholera toxin	*V. cholerae*	Chromosomal	A-5B	Ganglioside (GM_1)	Activation of adenylate cyclase, increase in cAMP level, secretory diarrhea
Diphtheria toxin	*C. diphtheriae*	Phage	A-B	Probably glycoprotein	Inhibition of protein synthesis, cell death
Heat-labile enterotoxins	*E. coli*	Plasmid	Similar or identical to cholera toxin		
Pertussis toxin	*B. pertussis*	Chromosomal	A-5B	Unknown, probably glycoprotein	Block of signal transduction mediated by target G proteins
Pseudomonas exotoxin A	*P. aeruginosa*	Chromosomal	A-B	Unknown, but different from diphtheria toxin	Similar or identical to diphtheria toxin
Shiga toxin	*Shigella dysenteriae*	Chromosomal	A-5B	Glycoprotein or glycolipid	Inhibition of protein synthesis, cell death
Shiga-like toxins	*Shigella* species, *E. coli*	Phage	Similar or identical to Shiga toxin		
Tetanus toxin	*C. tetani*	Plasmid	A-B	Ganglioside (GT_1) and/or GD_{1b}	Decrease in neurotransmitter release from inhibitory neurons, spastic paralysis

Heparin-binding epidermal growth factor on heart & nerve surfaces

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

Organism	Virulence Factors
V. parahaemolyticus	Thermostable direct hemolysin (Kanagawa positive)
V. vulnificus	Serum resistance, antiphagocytic polysaccharides, cytolysins, collagenase, protease, siderophore
V. alginolyticus	Collagenase
V. hollisae	Heat-stabile and heat-labile enterotoxin, hemolysin
V. damsela	Cytolysin

Laboratory Identification of Vibrios

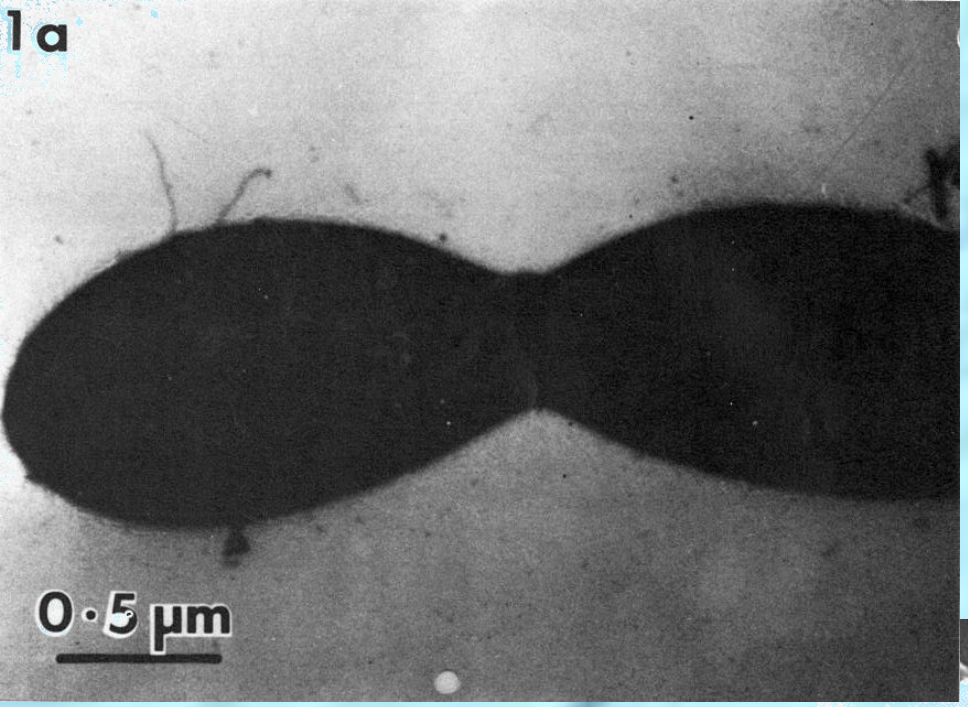
- Transport medium - Cary-Blair semi-solid agar
- Enrichment medium - **alkaline peptone broth**
  - Vibrios **survive and replicate at high pH**
  - Other organisms are killed or do not multiply
- **Selective/differential** culture medium - **TCBS agar**
  - *V. cholerae* grow as **yellow colonies**
- Biochemical and serological tests

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**

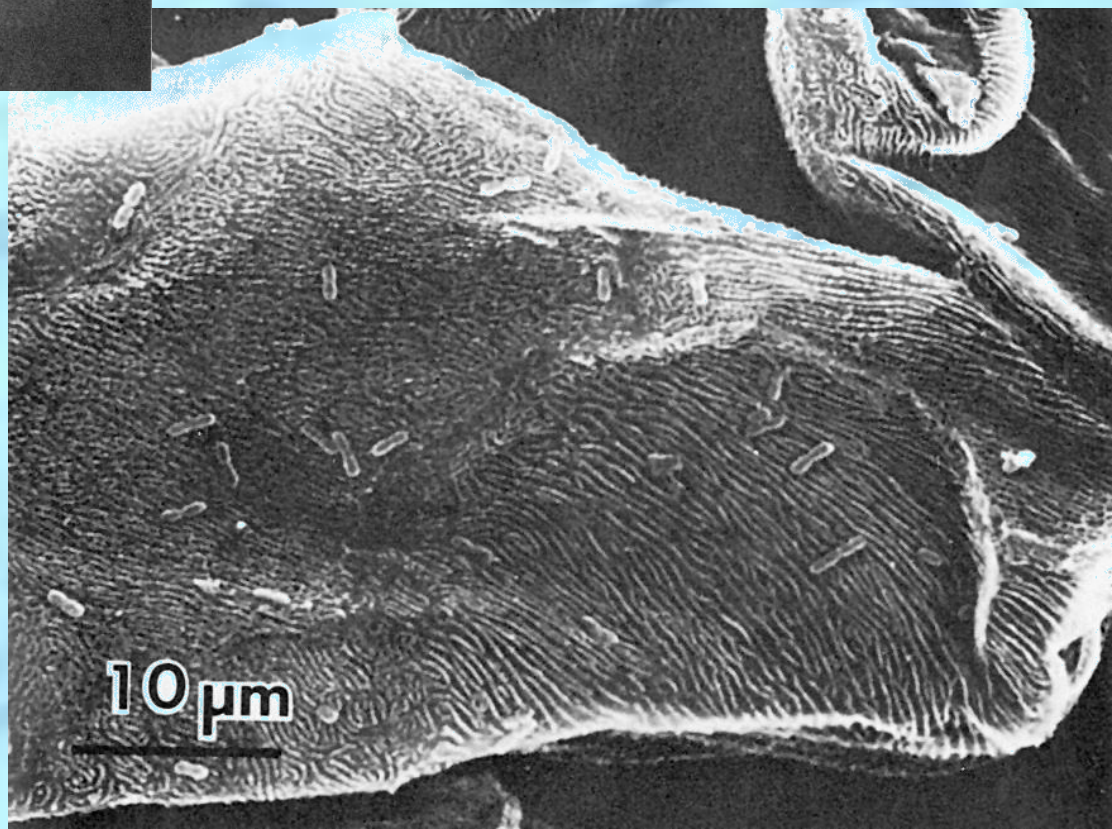
Clinical Syndromes of Aeromonas

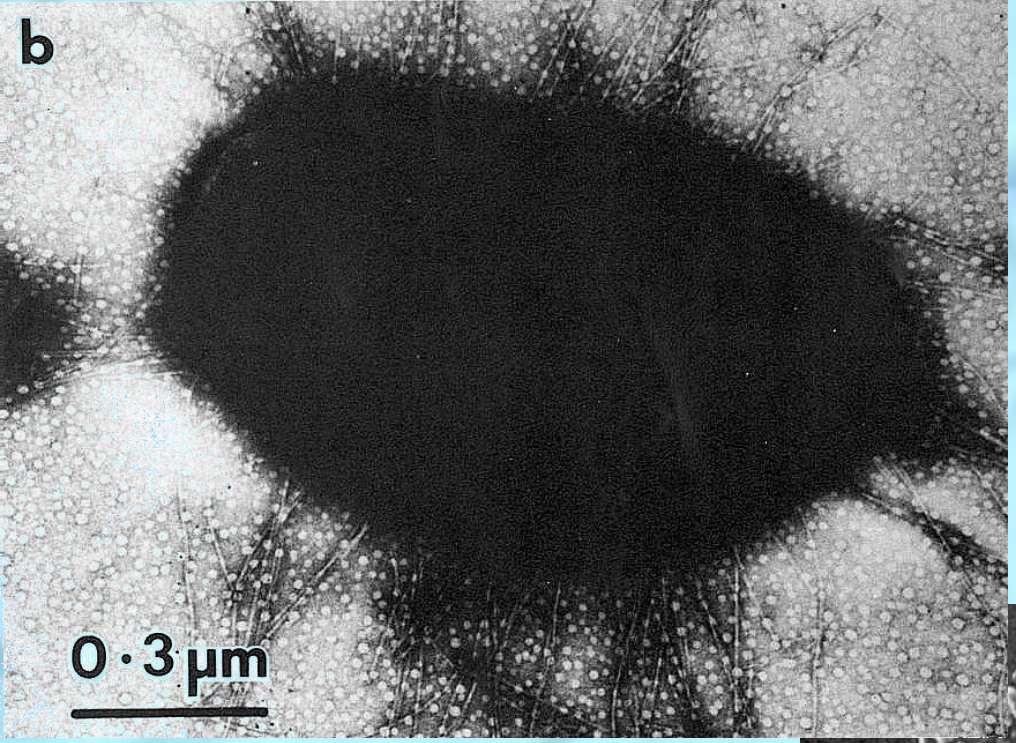
- Associated with **gastrointestinal disease**
  - **Chronic diarrhea in adults**
  - Self-limited **acute, severe disease in children** resembling shigellosis with blood and leukocytes in the stool
  - 3% **carriage** rate
- **Wound infections**
- Opportunistic systemic disease in immunocompromised
- **Putative virulence factors** include: endotoxin; hemolysins; eneterotoxin; proteases; siderophores; adhesins



Afimbriated
Aeromonas hydrophila

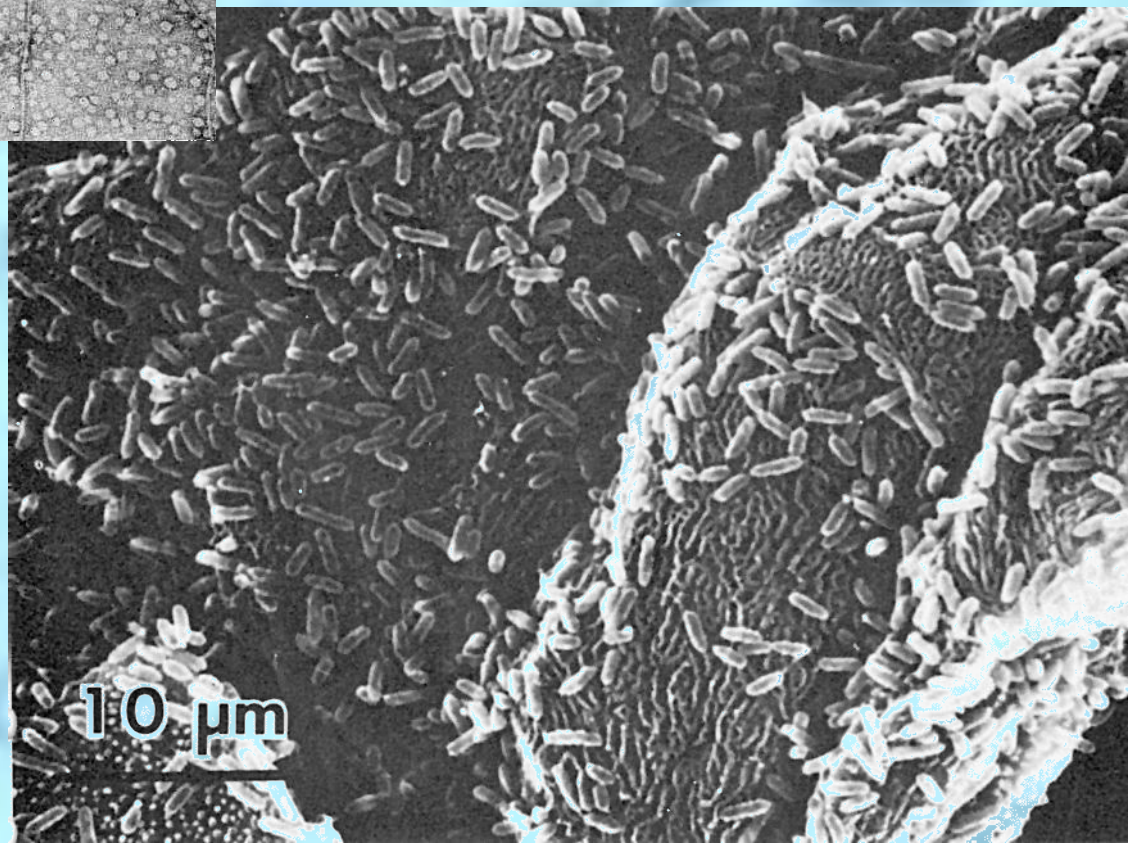
Nonadherent
Afimbriated
Bacterial Cells
and Buccal Cells





Fimbriated
Aeromonas hydrophila

Adherent
Fimbriated
Bacterial Cells
and Buccal Cells



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

Epidemiological Features	***Aeromonas***	***Plesiomonas***
Natural Habitat	Fresh or brackish water	Fresh or brackish water
Source of Infection	Contaminated water or food	Contaminated water or food
Clinical Features		
Diarrhea	Present	Present
Vomiting	Present	Present
Abdominal Cramps	Present	Present
Fever	Absent	Absent
Blood/WBCs in Stool	Absent ⟷	Present
Pathogenesis	Enterotoxin (??) ⟷	Invasiveness

REVIEW

Vibrio spp. (Family Vibrionaceae) *Associated with Human Disease*

Species	Source of Infection	Clinical Disease
V. cholerae	Water, food	Gastroenteritis
V. parahaemolyticus	Shellfish, seawater	Gastroenteritis, wound infection, bacteremia
V. vulnificus	Shellfish, seawater	Bacteremia, wound infection, cellulitis
V. alginolyticus	Seawater	Wound infection, external otitis
V. hollisae	Shellfish	Gastroenteritis, wound infection, bacteremia
V. fluvialis	Seafood	Gastroenteritis, wound infection, bacteremia
V. damsela	Seawater	Wound infection
V. metschnikovii	Unknown	Bacteremia
V. mimicus	Fresh water	Gastroenteritis, wound infection, bacteremia
*V. furnissii**	Seawater	Gastroenteritis
*V. cincinnatiensis**	Unknown	Bacteremia, meningitis
*V. carchariae**	Seawater	Wound (shark bite)

REVIEW

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 10-30ºC**
- 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

REVIEW

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

REVIEW

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**

Summary of *Vibrio cholerae* Infections

REVIEW

Physiology and Structure

Curved gram-negative bacilli.

Facultative anaerobe.

Fermenter.

Simple nutritional requirements; do not require salt for growth but can tolerate it.

Strains subdivided by their O cell wall antigens.

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

Virulence

Refer to Table 30–2 for complete listing.

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.

Epidemiology

Organism responsible for major pandemics (worldwide epidemics), 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.

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

Organism can multiply freely in water.

Summary of *Vibrio cholerae* Infections *(cont.)*

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)

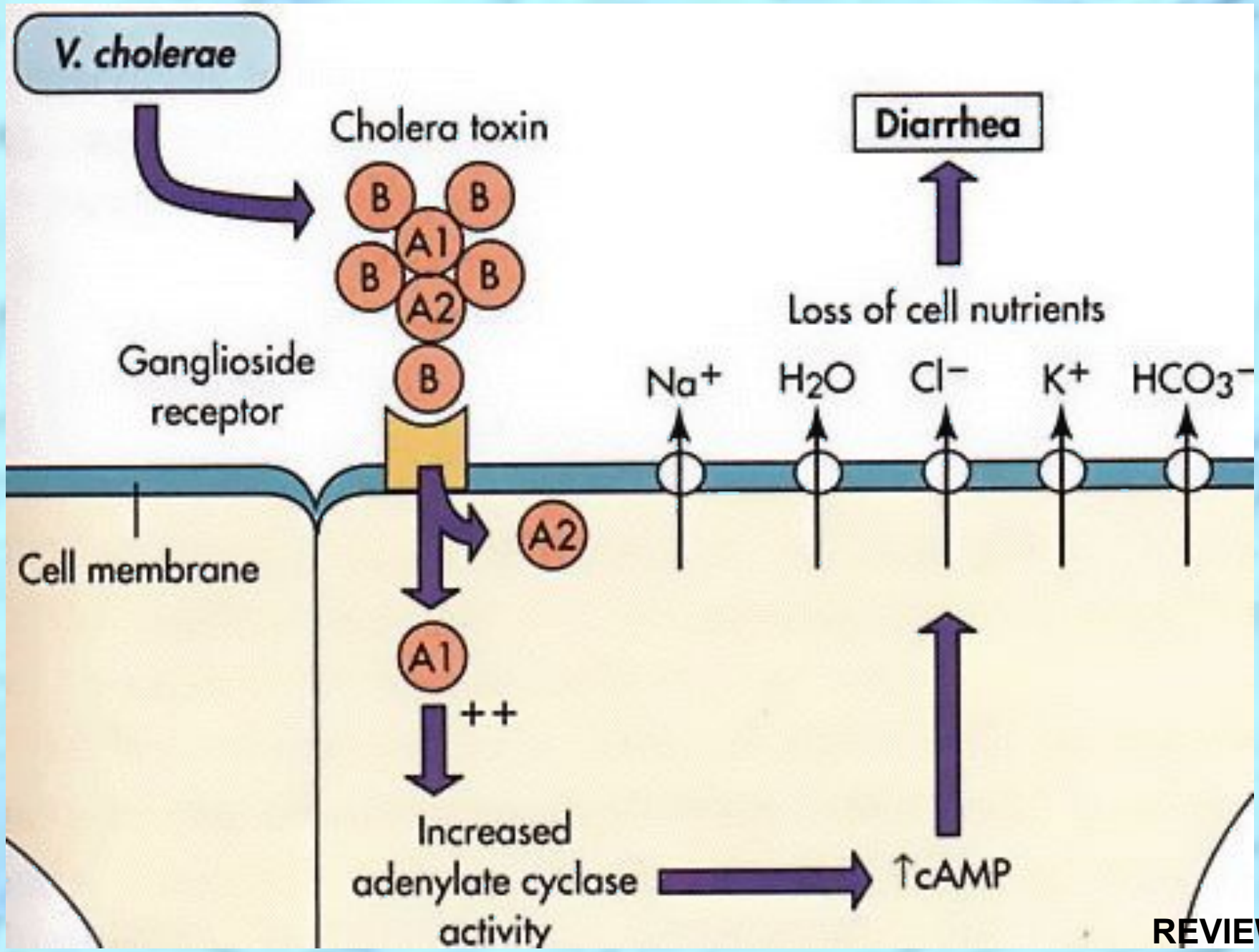
REVIEW

Virulence Factors Associated with Vibrio cholerae O1 and O139

Virulence Factor	Biologic Effect
Cholera toxin	Hypersecretion of electrolytes and water
Coregulated pilus	Adherence to mucosal cells
Accessory colonization	Adhesin factor
Hemagglutination-protease (mucinase)	Induces intestinal inflammation and degradation of tight junctions
Siderophores	Iron sequestration
Neuraminidase	Increase toxin receptors

REVIEW

Mechanism of Action of Cholera Toxin



REVIEW

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.

REVIEW

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

Organism	Virulence Factors
	(Kanagawa positive)
V. parahaemolyticus	Thermostable direct hemolysin
V. vulnificus	Serum resistance, antiphagocytic polysaccharides, cytolysins, collagenase, protease, siderophore
V. alginolyticus	Collagenase
V. hollisae	Heat-stabile and heat-labile enterotoxin, hemolysin
V. damsela	Cytolysin

REVIEW

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**

REVIEW

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

REVIEW

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**

REVIEW

Characteristics of Aeromonas and Plesiomonas Gastroenteritis

Epidemiologic and Clinical Features	*Aeromonas*	*Plesiomonas*
Natural habitat	Fresh or brackish water	Fresh or brackish water
Source of infection	Contaminated food or water	Contaminated food or water; contact v
Clinical presentation:		amphibians or reptiles
Diarrhea	Present	Present
Vomiting	Present	Present
Abdominal cramps	Present	Present
Fever	Absent	Absent
Blood/leukocytes in stool	Absent	Present
Pathogenesis	Enterotoxin (?)	Invasive

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