Vibrioni

Bacilli G-, forma a virgola, mobili con 1 flagello polare, asporigeni, acapsulati, aerobi facoltativi, metabolismo fermentativo.

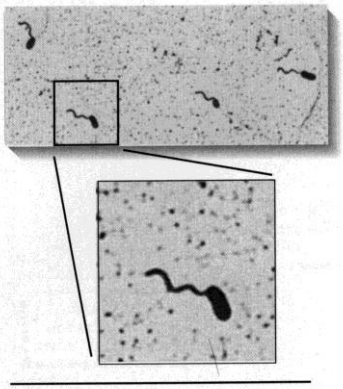


Figure 15.17
Light micrograph showing the curved, rod-shaped cells characteristic of vibrio species.

TABELLA 30–1. Specie di *Vibrio* associate a malattie nell'uomo

SPECIE	FONTE DI INFEZIONE	MALATTIA CLINICA
V. cholerae	Acqua, alimenti	Gastroenterite
V. parahaemolyticus	Crostacei, acqua di mare	Gastroenterite, infezione di ferite, batteriemia
V. vulnificus	Crostacei, acqua di mare	Batteriemia, infezione di ferite, cellulite
V. alginolyticus	Acqua di mare	Infezione di ferite, otite esterna
V. hollisae	Crostacei	Gastroenterite, infezione di ferite, batteriemia
V. fluvialis	Pesci	Gastroenterite, infezione di ferite, batteriemia
V. damsela	Acqua di mare	Infezione di ferite
V. metschnikovii	Sconosciuta	Batteriemia
V. mimicus	Acqua dolce	Gastroenterite, infezioni di ferite, batteremia
*V. furnissii**	Acqua di mare	Gastroenterite
*V. cincinnatiensis**	Sconosciuta	Batteriemia, meningite
*V. carchariae**	Acqua di mare	Ferita (morso di squalo)

* Ceppi isolati raramente associati a infezioni umane.

Diverse specie patogene per l'uomo

Comprendono molti gruppi sierologici definiti in base all'antigene O. I sierotipi O1 possono avere due diversi biotipi ElTor produce emolisine, viene escreto più a lungo, sopravvive di più in acqua.
V. cresce in natura in ambienti marini, e può replicare in acqua in condizioni particolari, oltre che in molluschi e crostacei.
L'infezione si diffonde mediante cibi e acqua contaminati.

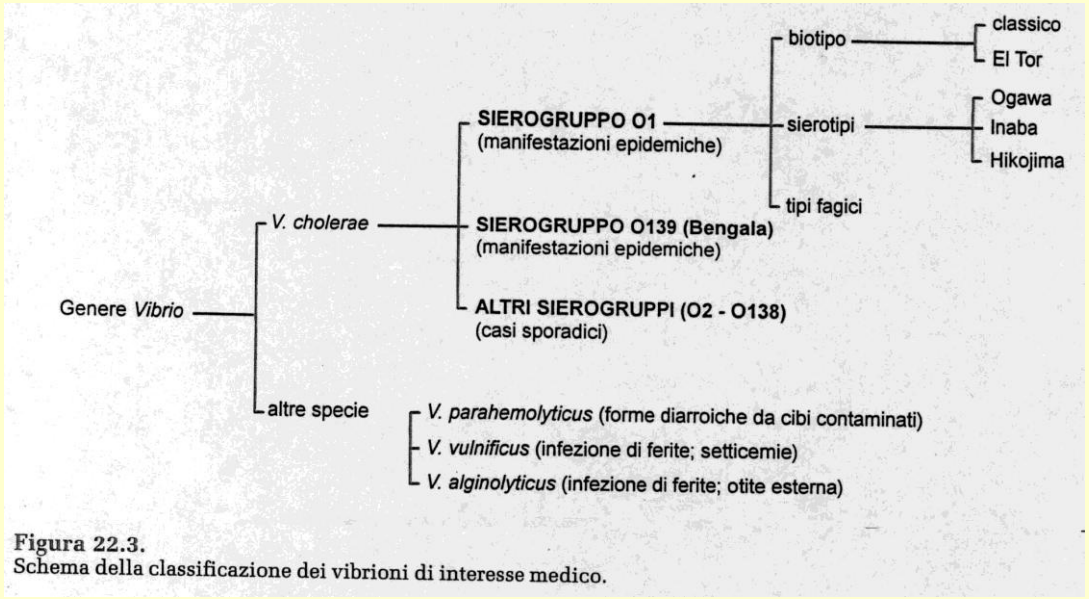


Figura 22.3.
Schema della classificazione dei vibrioni di interesse medico.

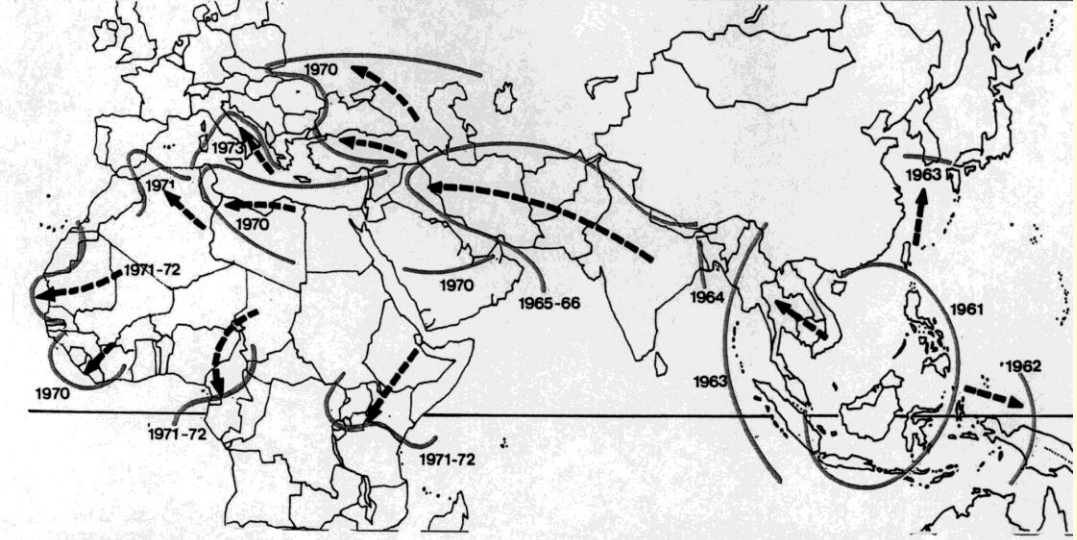
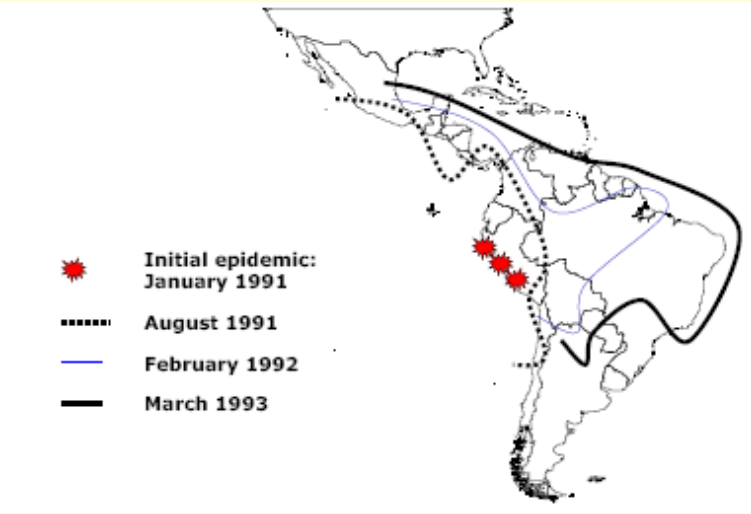
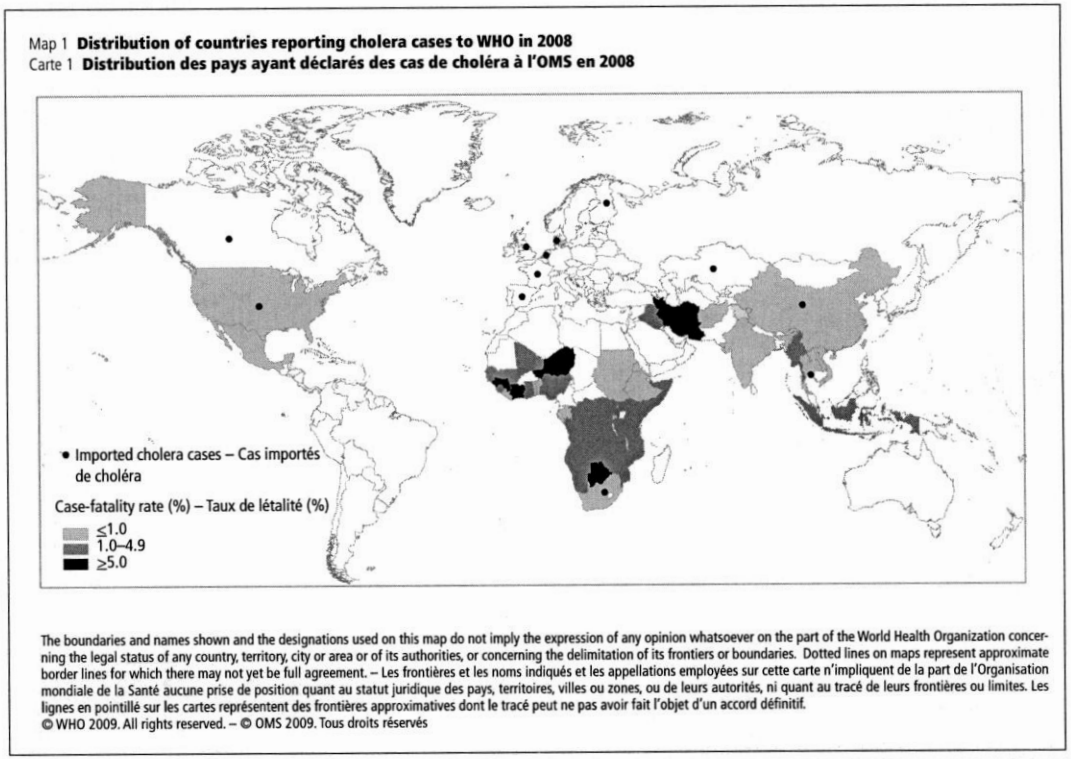


Figura 22.2.
Mappa della diffusione del colera da vibrioni «El Tor» nel periodo 1961-1973.



Epidemia Sud America,
1991: 400.000 casi
2002: 22 casi



HAITI Ott 2010: epidemia post terremoto, 231.070 casi totali, con 4.5492.591 morti (2%)
MA in zone rurali mortalità anche al 10%
(dati a Feb 2011)

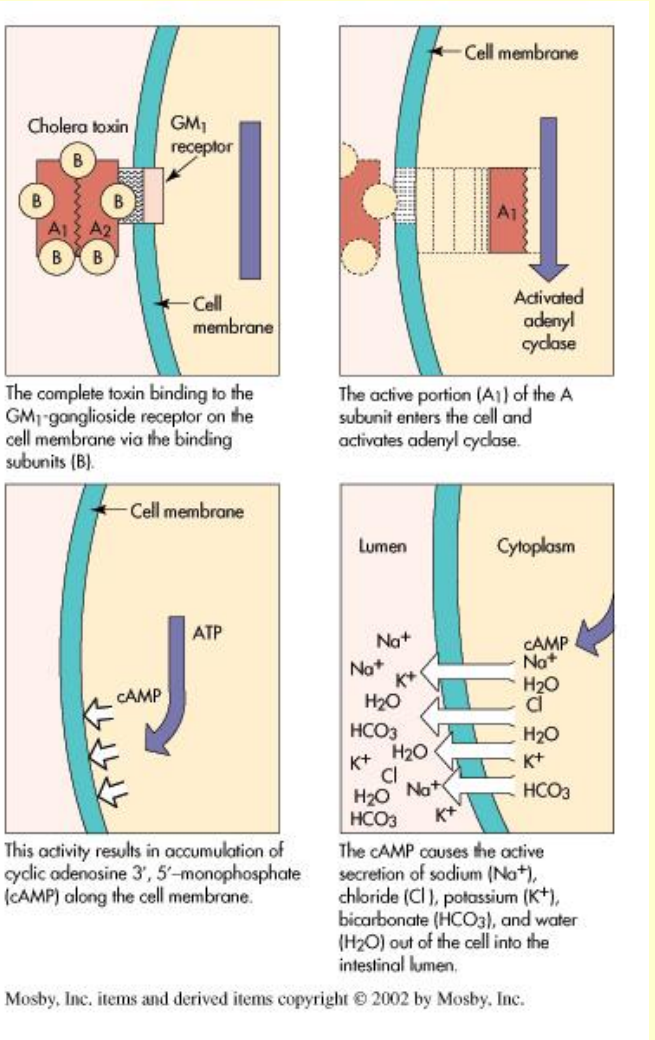
Dopo l'ingestione, il vibrione infetta l'intestino. Le adesine sono importanti per colonizzazione e virulenza. Il batterio non è invasivo, e causa malattia attraverso una enterotossina, che si lega a recettori sulle cellule intestinali e causa secrezione di elettroliti e acqua, anche 1 litro all'ora.

Causa diarrea acquosa (feci ad acqua di riso), con mortalità elevata in mancanza di trattamento (60% vs >1%)

Sono disponibili un paio di vaccini, che hanno però dimostrato utilità limitata, con immunità breve ed incompleta

TABLE 32–2. Virulence Factors of *Vibrio cholerae* O1 and O139

Virulence Factor	Biologic Effect
Cholera toxin	Hypersecretion of electrolytes and water
Toxin co-regulated pilus	Adherence to intestinal mucosal cells; binding site for CTXϕ
Accessory cholera enterotoxin	Increases intestinal fluid secretion
Zonnula occludens toxin	Increases intestinal permeability
Colonization factor	Adhesin factor
Neuraminidase	Modifies cell surface to increase GM_1 binding sites for cholera toxin



The complete toxin binding to the GM_1-ganglioside receptor on the cell membrane via the binding subunits (B).

The active portion (A_1) of the A subunit enters the cell and activates adenyl cyclase.

This activity results in accumulation of cyclic adenosine 3', 5'–monophosphate (cAMP) along the cell membrane.

The cAMP causes the active secretion of sodium (Na^+), chloride (Cl^-), potassium (K^+), bicarbonate (HCO_3), and water (H_2O) out of the cell into the intestinal lumen.

Key facts

- Cholera is an acute diarrhoeal disease that can kill within hours if left untreated.
- There are an estimated 3–5 million cholera cases and 100 000–120 000 deaths due to cholera every year.
- Up to 80% of cases can be successfully treated with oral rehydration salts.
- Effective control measures rely on prevention, preparedness and response.
- Provision of safe water and sanitation is critical in reducing the impact of cholera and other waterborne diseases.
- Oral cholera vaccines are considered an additional means to control cholera, but should not replace conventional control measures.

WHO

Risk for Travelers

- Travelers who follow usual tourist itineraries and who observe food safety recommendations while in countries reporting cholera have virtually no risk. The risk is increased for those who drink untreated water or eat poorly cooked or raw seafood in disease-endemic areas.
- From 1996 through 2006, only 40 confirmed cases of cholera in the United States were acquired abroad.

CDC

Cholera

Disease Listing | General Information | **Technical Information** | Additional Information

Clinical Features	Profuse watery diarrhea, vomiting, circulatory collapse and shock. Many infections are milder diarrhea or asymptomatic.
Etiologic Agent	*Vibrio cholerae* serogroup O1 or O139 that produces cholera toxin.
Incidence	0-5 cases per year in the United States. A major cause of epidemic diarrhea throughout the developing world. Ongoing global pandemic in Asia, Africa and Latin America for the last four decades.
Sequelae	25-50% of typical cases are fatal if untreated.
Transmission	Contaminated drinking water or food. Large epidemics often related to fecal contamination of water supplies or street vended foods. Occasionally transmitted through eating raw or undercooked shellfish that are naturally contaminated.
Risk Groups	Virtually none in the United States. Risk extremely low (1 per million) even in travelers. Persons living in poverty in the developing world.

BOX 32–3. Clinical Summaries

Vibrio cholerae

Cholera: Begins with an abrupt onset of watery diarrhea and vomiting and can progress to severe dehydration, metabolic acidosis and hypokalemia, and hypovolemic shock

Gastroenteritis: Milder forms of diarrheal disease can occur in toxin-negative strains of *V. cholerae* O1 and in non-O1 serotypes

Vibrio parahaemolyticus

Gastroenteritis: Generally self-limited with an explosive onset of watery diarrhea and nausea, vomiting, abdominal cramps, headache, and low-grade fever

Wound infection: Associated with exposure to contaminated water

Vibrio vulnificus

Wound infection: Severe, potentially fatal infections characterized by erythema, pain, bullae formation, tissue necrosis, and septicemia

BOX 32–2. Summary of *Vibrio cholerae* Infections

Physiology and Structure

Curved gram-negative rods; facultative anaerobe; fermenter
Simple nutritional requirements; do not require salt for growth but can tolerate it
Strains subdivided into more than 140 serogroups (O cell wall antigens)
V. cholerae serogroup O1 is further subdivided into serotypes (Inaba, Ogawa, Hikojima) and biotypes (classical, el tor)

Virulence

Refer to Table 32–2

Epidemiology

Serotype O1 is responsible for major pandemics (worldwide epidemics) with significant mortality in developing countries; 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
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

Refer to Box 32–3

Diagnosis

Microscopic examination of stool generally nonproductive because the organism is diluted in the large volume of watery diarrhea
Culture should be performed early in course of disease with fresh stool specimens

Treatment, Prevention, and Control

Fluid and electrolyte replacement are crucial
Antibiotics reduce 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
Combination inactivated whole cell and cholera toxin B subunit vaccines provide limited protection; other vaccines are in development