**Clostridium**


Clostridium species from a stool sample.

Scientific classification

Domain: Bacteria

Phylum: firmicutes

Class: Clostridia

Order: Clostridiales

Family: Clostridiaceae

Genus: ***Clostridium***

***Species***

*C. barati*
*C. chauvoei*
*C. difficle*
*C. novyi*
*C. perfringens*
*C. septicum*
*C. sordelli*
*C. tetani*
*C. botulinum*

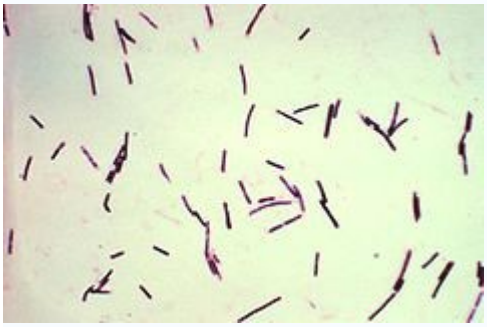
***Clostridium*** is a genus of Gram positive bacteria, belonging to the Firmicutes. They are obligate anaerobes capable of producing endospore. Individual cells are rod-shaped, which gives them their name, from the Greek *kloster* or spindle. These characteristics traditionally defined the genus, however many species originally classified as *Clostridium* have been reclassified in other genera.

**Pathology**

*Clostridium* consists of around 100 species that include common free-living bacteria as well as important pathogens. There are four main species responsible for disease in humans:

- *Clostridium botulinum*, an organism producing a toxin in food/wound that causes botulism.
- *C. difficile*, which can overgrow other bacteria during antibiotic therapy, can cause pseudomembranous colitis.
- *C. perfringens*, formerly called *C. welchii*, causes a wide range of symptoms, from food poisoning to gas gangrene. Also responsible for enterotoximia (also known as "overeating disease" or "pulpy kidney disease") in sheep and goats.[7] *C. perfringens* also takes the place of yeast in the making of salt rising bread.
- *C. tetani*, the causative organism of tetanus. ]

**Clostridium perfringens**



*Clostridium perfringens bacilli*

***Clostridium perfringens*** (formerly known as *Clostridium welchii*) is a Gram positive, rod-shaped, anaerobic,spore forming bacterium of the genus Clostridium. *C. perfringens* is ubiquitous in nature and can be found as a normal component of decaying vegetation, marine sediment, the intestinal tract of human and other vertibrates, insects, and soil.

**Infection characteristics**

*C. perfringens* is commonly encountered in infections as a benign component of the normal flora.

Infections due to *C. perfringens* show evidence of tissue necrosis,bacteremia and gas gangrene, which is also known as clostridial myonecrosis. The toxin involved in gas gangrene is known as α-toxin, which inserts into the plasma membrane of cells, producing gaps in the membrane that disrupt normal cellular function.

After ingestion bacteria multiply and lead to colic, diarrhoea and sometimes nausea.

**Food poisoning**

C. perfringens bacteria are the cause of food-borne illness, with poorly prepared meat and poultry the

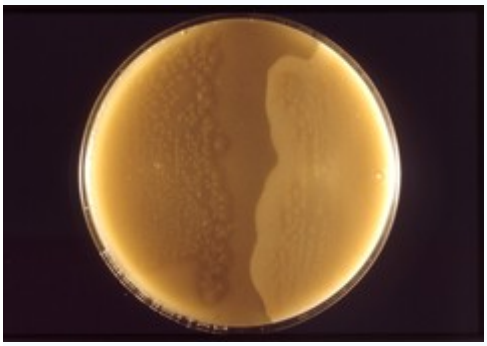
main culprits in harboring the bacterium. The Clostridium perfringens enterotoxin (CPE) mediating the disease is often heat-resistant and can be detected in contaminated food and feces.

Incubation time is between 6 and 24 (commonly 10-12) hours after ingestion of contaminated food. Often times meat is well prepared but too far in advance of consumption. Since C. perfringens forms spores that can withstand cooking temperatures, if let stand for long enough germination ensues and infective bacterial colonies develop. Symptoms typically include abdominal cramping and diarrhea - vomiting and fever are unusual. The whole course usually resolves within 24 hours. Very rare, fatal cases of clostridial necrotizing enteritis (also known as Pig-Bel) have been known to involve "Type C" strains of the organism, which produce a potently ulcerative β-toxin. This strain is most frequently encountered in Papua New Guinea.

**Gas Gangrene**

Cl.perfringens is the most common bacterial agent for Gas gangrene.

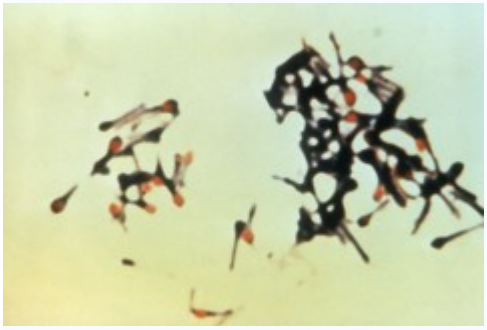
**Colony characteristics**



*C. perfringens* colonies on an egg yolk agar plate showing a white precipitate

On blod agar plates, *C. perfringens* grown anaerobically produces β-haemolytic, flat, spreading, rough, translucent colonies with irregular margins. A Nagler agar plate, containing 5-10% egg yolk, is used to presumptively identify strains which produce α-toxin, a diffusible lecithinase which interacts with the lipids in egg yolk to produce a characteristic precipitate around the colonies. One half of the plate is inoculated with antitoxin to act as a control in the identification.

**Clostridium tetani**



*Clostridium tetani bacilli*

***Clostridium tetani*** is a rod-shaped, anaerobic bacterium of the genus Clostridium. Like other

*Clostridium* species, it is Gram positive, and its appearance on a gram stain resembles tetanus rackets or drumsticks. *C. tetani* is found as spores in soil or as parasites in the gastrointestinal tract of animals. *C. tetani* produces a potent biological toxin, tetanospamin, and is the causative agent of tetanus.

**Characteristics**

*C. tetani* is a rod-shaped, obligate anaerobe which stains Gram positive in fresh cultures; established cultures may stain Gram negative. During vegetative growth, the organism cannot survive in the presence of oxygen, is sensitive to heat and has flagella which provide limited mobility. As the bacterium matures, it develops a terminal spore, which gives the organism its characteristic appearance. *C. tetani* spores are extremely hardy, and are resistant to heat and most antiseptics.The spores are distributed widely in manure- -treated soils.

**Toxicity**

*C. tetani* usually enters a host through a wound to the skin and then it replicates. Once an infection is established, *C. tetani* produces two exotoxins, tetanolysin and tetanospasmin. Eleven strains of *C. tetani* have been identified, which differ primarily in flagellar antigens and in their ability to produce tetanospasmin. The genes that produce toxin are encoded on a plasmid which is present in all toxigenic strains, and all strains that are capable of producing toxin produce identical toxins.

Tetanolysin serves no known function to *C. tetani*, and the reason the bacteria produce it is not known with certainty. Tetanospasmin is a neurotoxin and causes the clinical manifestations of tetanus. Tetanus toxin is generated in living bacteria, and is released when the bacteria lyses, such as during spore germination or during vegetative growth. A minimal amount of spore germination and vegetative cell growth are required for toxin production.

On the basis of weight, tetanospasmin is one of the most potent toxins known. The estimated minimum human lethal dose is 2.5 nanograms per kilogram of body weight, or 175 nanograms in a 70 kg (154 lb) human. The only toxins more lethal to humans are botulism toxin, produced by close relative Clostridium botulinum and the enterotoxin produced by Corynaebacterium diphtheriae, the causative agent of diphtheria.

The clinical manifestations of tetanus are unopposed muscle contraction and spasm. Characteristic features are Risus Sardonicus (a rigid smile), Trismus (commonly known as lock-jaw), and Opisthotonus (rigid, arched back). Seizures may occur, and the autonomic nervous system may also be affected. Tetanospasmin appears to prevent the release of neurotransmitters by selectively cleaving a component of synaptic vesicles called synaptobrein.

**Treatment**

When a tetanus infection becomes established, treatment usually focuses on controlling muscle spasms, stopping toxin production, and neutralizing the effects of the toxin. Treatment includes administration of tetanus immune globulin (TIG), which comprises antibodies that inhibit tetanus toxin (also known as tetanus antitoxin), by binding to and removing unbound tetanus toxin from the body. Binding of the toxin to the nerve endings appears to be an irreversible event, and TIG is ineffective at removing bound toxin. Recovery of affected nerves requires the sprouting of a new axon terminal. Large doses of antibiotic drugs (such as metranidazile or intramuscular penicillin G) are also given once tetanus infection is suspected, to halt toxin production.

Prevention of tetanus includes vaccination, and cleaning the primary wound. Prophylaxis is effective, in the form of a tetanus toxoid vaccine, which is given with or without passive immunization with tetanus immune globulin.

**Clostridium botulinum**

***Clostridium botulinum*** is a Gram positive, rod shaped bacterium that produces the neurotoxin botulin, which causes the flaccid muscular paralysis seen in botulism. It is an anaerobic spore-former, which produces oval, subterminal endospores and is commonly found in soil.

**Group I to IV *Clostridium botulinum***
*Clostridium botulinum*, producing highly potent botulinum neurotoxin, is a diverse species consisting of four genetically and physiologically distinct groups (Groups I-IV) of organisms. Groups I and II *C. botulinum* produce A, B, E, and/or F toxins which cause human botulism. In addition, some strains of *Clostridium butyricum* and *Clostridium barati* produce type E and F toxins, respectively, and have thus been related to human

Enterotoxemia is one of the most common and costly disease problems in the U.S. sheep industry and worldwide. Preventative measures are generally recommended to prevent unnecessary losses.

**Enterotoxemia( "overeating disease" or "pulpy kidney" disease)**

Enterotoxemia, also known as "overeating disease" or "pulpy kidney" disease, is caused by the bacterium *Clostridium perfringins*, Type C and D. This bacterium is a normal inhabitant of the intestinal tract of sheep and other mammals, and normally, is not a problem. However, there are certain conditions which trigger excessive bacterial growth in which lethal amounts of toxin are produced, resulting in death of the animal.

**Enterotoxemia is usually associated with grain feeding.**

Enterotoxemia type D is most commonly associated with heavy concentrate feeding or an abrupt change in the diet, usually to a better feed. It usually affects weaned lambs that are consuming at least 3/4 of a pound of grain per day. In contrast, enterotoxemia type C most often affects nursing lambs within the first few weeks of life, causing a bloody diarrhea.

Animals with enterotoxemia are frequently found dead, without symptoms. The disease progresses rapidly and often strikes the best-doing lambs. When symptoms are observed, they are often confused with other diseases such as *e.coli* scours or polio. Affected lambs will appear depressed, may grind their teeth, twitch or convulse. Abdominal pain is common.

Treatment of affected animals is usually unrewarding. Antitoxins can be given either orally or by injection and administration of antibiotics, such as penicillin, may help, but as with other disease problems, prevention is the better course of action. Fortunately, there is an effective, inexpensive vaccine to control the disease. The vaccine is given under the skin (subcutaneously). Since lambs can be affected early in life, it is advisable to vaccinate ewes 30 days prior to lambing. This way the lambs will obtain temporary immunity from the mother's colostrum. Previously unvaccinated animals should be given two doses of the vaccine one month apart, followed by an annual booster. Lambs should receive their first vaccination when they are about four weeks of age, followed by a second injection at six weeks. Some producers vaccinate again before putting lambs into a feedlot (if the lambs are 90 days or older). Purchased feeder lambs should receive their first vaccination prior to the feeding period and be boostered three weeks later. It takes from 10 days to two weeks to develop immunity.

If ewes were not vaccinated for type C enterotoxemia, lambs can be vaccinated with the type C toxoid at 2 to 3 days of age and again in 2 weeks or they can be given antiserum shortly after birth. It should be noted that colostral antibodies will interfere with vaccinations at an early age, so it is always more advisable to vaccinate the ewe than the young lamb.

Management which will aid in prevention of the disease include avoiding sudden changes to the diet. There should be a gradual transition of two to three weeks when going from a roughage to a highly concentrated ration. Feeding at regular intervals, proper ration mixing and providing adequate feeder space will also help to prevent problems. The feeding of antibiotics will help to prevent enterotoxemia in feedlot lambs.

**Blackleg (disease)**

**Blackleg**, **black quarter**, **quarter evil**, **quarter ill** (Latin: *Gangraena emphysematosa*) is an infectious bacterial disease of sheep and cattle caused by *Clostridium chauvoei* bacteria. It is found all over the world. A symptom of blackleg is characteristic swellings which make a cracking sound under pressure. **Blackleg vaccine** gives immunity against it.The use of 7-type clostridial vaccination is the most common preventative measure taken against blackleg. Burning the upper layer of soil to eradicate left-over spores is the best way to stop the spread of blackleg from diseased cattle.

**Clostridium novyi**

***Clostridium novyi*** (oedematiens) a Gram positive, endospore, forming,obligateanaerobic bacteria of the class clostridia. It is ubiquitous, being found in the soil and faeces. It is pathogenic, causing a wide variety of diseases in man and animals. It comes in three types, labelled A, B, and a non-pathogenic type C distinguished by the range of toxins they produce.

**Toxins**

The toxins are designated by Greek letters. The toxins normally produced by the various types are shown in table 1

| **Table 1** | |
|---|---|
| **C novyi type** | **Toxins** |

| | |
|---|---|
| A | alpha, gamma, delta, epsilon |
| B | alpha, beta, zeta |
| C | gamma |

The alpha-toxin of *Clostridium botulinum* types C and D, is similar to the *C novyi* beta-toxin. The A and B toxins of *Clostridium difficile* show homology with the alpha-toxin of *C novyi* as does the lethal toxin of *clostridium sordellii.*

**Alpha toxin**

The alpha-toxin is characterised as lethal and necrotizing.
The type A alpha-toxin is oedematising.

**Beta-Toxin**

The beta-toxin is characterised as haemolytic, necrotizing lecithinase.

**Gamma-Toxin**

The gamma-toxin is characterised as haemolytic, lecithinase.

**Delta-Toxin**

The delta-toxin is characterised as oxygen labile haemolysin.

**Epsilon-Toxin**

The epsilon-toxin is characterised as lecithino-vitelin and thought to be responsible for the pearly layer found in cultures.

**Zeta-Toxin**

The zeta-toxin is characterised as haemolysin.

**Animal diseases**

Gas gangrene and Infectious necrotic hepatitis.
Infectious necrotic hepatitis is a disease of large animals caused by Clostridium novyi infection. The primary infection is intestinal and transferred by the faecal-oral route. Spores of *C. novyi* escape from the gut and lodge in the liver where they remain dormant until some injury creates anaerobic conditions for them to germinate causing local necrosis and widespread damage to the microvascular system resulting in subcutaneous bleeding and blackening of the skin, hence the common name, "black disease.

**References**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2. From Wikipedia, the free encyclopedia

3. Hirsh, Dwight C.(1999): Veterinarym Microbiology, Blachwell Science, Inc.

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