Bacteroides

Bacteroides is a genus of Gram-negative, bacillusbacteria. *Bacteroides* species are non-endospore-forming, anaerobes, and may be either motile or non-motile, depending on the species.[1] The DNA base composition is 40-48% GC. Unusual in bacterial organisms, *Bacteroides* membranes contain sphingolipids. They also contain meso-diaminopimelic acid in their peptidoglycan layer.

Bacteroides are normally mutualistic, making up the most substantial portion of the mammalian gastrointestinal flora,[2] where they play a fundamental role in processing of complex molecules to simpler ones in the host intestine.[3]As many as 10^{10}-10^{11} cells per gram of human feces have been reported.[4] They can use simple sugars when available; however, the main sources of energy for *Bacteroides* species in the gut are complex host-derived and plant glycans[5].

One of the most important clinically is *Bacteroidesfragilis*.

Scientific classification

Kingdom:Bacteria

Phylum: Bacteroidetes

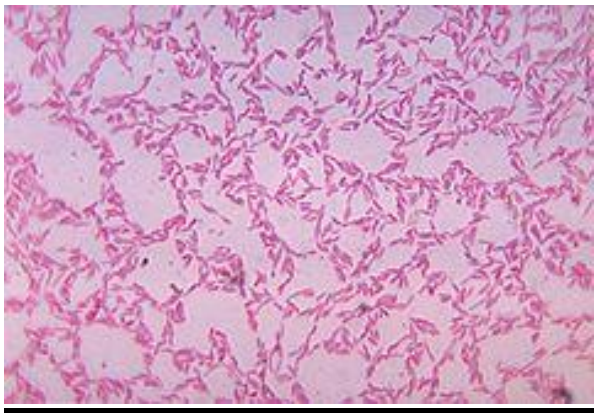
Class: Bacteroidetes

Order: Bacteroidales

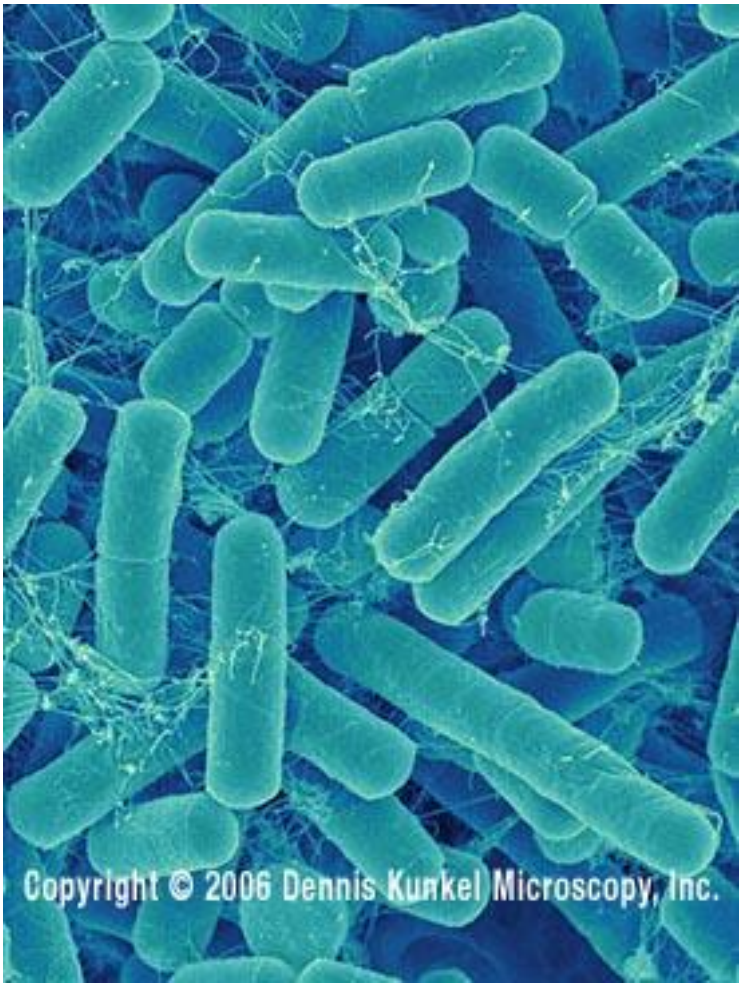
Family: Bacteroidaceae

Genus:*Bacteroides*

Species: *B. fragilis*



Bacteroides spp. anaerobically cultured in blood agar medium



*Bacteroides*spp

Pathogenesis

Bacteroides species also benefit their host by excluding potential pathogens from colonizing the gut. Some species (*B. fragilis*, for example) are opportunistic human pathogens, causing infections of the peritoneal cavity, gastrointestinal surgery, and appendicitis via abscess formation, inhibiting phagocytosis, and inactivating beta-lactam antibiotics.[6] Although

Bacteroides species are anaerobic, they are aerotolerant and thus can survive in the abdominal cavity.

In general, *Bacteroides* are resistant to a wide variety of antibiotics — β-lactams, aminoglycosides, and recently many species have acquired resistance to erythromycin and tetracycline. This high level of antibiotic resistance has prompted concerns that *Bacteroides* species may become a reservoir for resistance in other, more highly-pathogenic bacterial strains.[7]

Bacteroidesfragilis

characteristicsBacteroidesfragilis

Bacteroidesfragilis is a Gram-negative bacillus bacterium species, and an obligate anaerobe of the gut.[8]

B. fragilis group is the most commonly isolated bacteriodaceae in anaerobic infections especially those that originate from the gastrointestinal flora. *B. fragili*s is the most prevalent organism in the *B. fragilis* group, accounting for 41% to 78% of the isolates of the group. The *B. fragilis* group is the species of Bacteroidaceaethat

is isolated with greatest frequency in clinical specimens. These organisms are resistant to penicillin by virtue of production of beta-lactamase, and by other unknown factors.[9]

This organism was formerly classified as subspecies of *B. fragilis* (i.e. *ss. fragilis, ss. distasonis*, *ss. ovatus*, *ss. thetaiotaomicron*, and ss. *vulgatus*). They have been reclassified into distinct species on the basis of DNA homology studies.[10]*B. fragilis*(formerly known as *B. fragilis ss. fragilis*, one of the subspecies of B. fragilis) is often recovered from blood, pleural fluid, peritoneal fluid, wounds and brain abscesses.

Although *B. fragilis* group is the most common species found in clinical specimens, it is the least common Bacteroides present in fecal flora, comprising only 0.5% of the bacteria present in stool. The pathogenicity of this group of organisms probably results from its ability to produce capsular material, which is protective against phagocytosis.[11]

Clinical significance

It is involved in 90% of anaerobic peritoneal infections.[12]*Bacteroidesfragilis* acts primarily at the surface of the mucosa.[13] It predominates in bacteremia[14] associated with intraabdominal infections, peritonitis and abscesses following rupture of viscus, and subcutaneous abscesses or burns near the anus.[15]

Working with lab cultures and mice, Johns Hopkins scientists have found that a strain of the common gut pathogen Bacteroidesfragilis causes colon inflammation and increases activity of a gene called spermine oxidase (SMO) in the intestine. The effect is to expose the gut to hydrogen peroxide – the caustic, germ-fighting substance found in many medicine cabinets—and cause DNA damage, contributing to the formation of colon tumors, say the scientists.[16]

Ecology

Bacteriodesfragilis lives primarily in humans and animals intestinal/colon flora. As long as *Bacteriodesfragilis* is retains within the intestinal lumen, its contribution to our body is very diverse. And once

Bacteroidesfragilis leaves the lumen and travels to adjacent areas and organs, it can be detrimental as it contributed to a variety of infections in the upper body, abdomen, skin and many others. *Bacteroidesfragilis* now act as a pathogen and invades its host by producing the enterotoxins. Due to its role as a pathogen, *Bacteroidesfragilis* can be very complex; they will be able to survive and adapt in most environments like its neighbor, *E.coli*.

MODE OF TRANSMISSION

Infection results from displacement of *Bacteroides* spp. or closely related genera from normal mucosal location as a result of trauma such as animal/human bites, burns, cuts, or penetration of foreign objects, including those involved in surgery(17). There is no evidence that organisms are invasive on their own.

virulence factor

Bacteroidesfragilis produce polysaccharide capsule high in succinic acid. Once *Bacteroidesfragilis* is released from the capsule, it paralyses the migration of

leukocytes, which is required for the site of healing. When the *Bacteroidesfragilis* kills off the leukocytes, the infections grew and if left untreated then the death rate is as high 60%. Succinic acid was used to test the neutrophil function and they have found that the succinic acid enhances the virulence factor of *Bacteroides*. Another thing they've found is that the virulence factor increases with lower pH and in microenvironment with high infections.

Bacteroidesfragilis group (~60% of intra-abdominal infections and ~70% of anaerobic bacteremias), Prevotella, Porphromonas, Fusobacterium (virulence factors: varies with genus, includes capsule, adhesins, endotoxin, enzymes [B. fragilis produces an enterotoxin]; infections: brain, oral, URT, dental, sinuses, LRT, female genital tract infections; control: drain, debride, metronidazole, clindamycin, chloramphenicol, imipenem.

Among the many virulence factors produced, B. fragilis produces an enzyme that allows the organism to survive in the presence of small amounts of oxygen. the enzymes listed below catalyzes the following reaction:

A. Beta lactamase

B. Myeloperoxidase

C. Nicotinamide adenine dinucleotide phosphate (NADPH) oxidase

D. NO synthase

E. Oxidase

F. Superoxide dismutase

Prevention

- In areas where AGNB and other anaerobes predominate, early and aggressive treatment of acute infection can prevent them from becoming chronic.
- When the risk of anaerobic infections (eg, intra-abdominal and wound infection following surgery) is high, proper antimicrobial prophylaxis may reduce the risk.
- Preventing oral florae aspiration by improving neurologic status, suctioning oral secretions, improving oral hygiene, and maintaining lower

stomach pH can reduce the risk of aspiration pneumonia and its complications.

Treatment

In general, *B. fragilis* is susceptible to metronidazole, carbapenems, tigecycline, beta-lactam/beta-lactamase inhibitor combinations (e.g., Unasyn, Zosyn), and certain antimicrobials of the cephamycin class, including cefoxitin. The bacteria have inherent high-level resistance to penicillin. Production of beta lactamase appears to be the main mechanism of antibiotic resistance in *B. fragilis*.[18]Clindamycin is no longer recommended as the first-line agent for *B. fragilis* due to emerging high-level resistance (>30% in some reports).[19]

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