



Pseudomonas aeruginosa

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Pseudomonas aeruginosa



Morphology

- •Gram-negative rods.
- •Motile with polar flagellae.
- •Some strains may be capsulated.
- •Non-spore forming.









Culture characters

- Strict aerobic ****
- Grow on many types of media
- Gives greenish colour to nutrient agar with sweet grape-like fruity odor, and some strains may cause βhemolysis.
- Grow at 37° to 42°
- It produces exopigments that consist of:

Pyocyanin- nonfluorescent bluish pigment

Pyoverdin- fluorescent greenish pigment











 β -hemolysis on blood agar

Greenish colour on nutrient agar





Biochemical reaction

- Do not ferment carbohydrates.
- Oxidase positive



Identification of *P. aeruginosa* is usually based on oxidase test and its colonial morphology: β -hemolysis, the presence of characteristic **pigments**, sweet odor, and growth at 42 °C.

Virulence Factors P. aeruginosa



- Pili for attachment to host cells
- **Capsule** seen in cultures from patients with cystic fibrosis.
- LPS- endotoxin, multiple immunotypes.
- **Pyocyanin:** catalyzes the production of toxic forms of oxygen that cause tissue damage.
- **Pyoverdin:** a siderophore (iron-chelating compounds).

Proteases

proteasecausestissuedamageandhelpbacteriaspread.

- Phospholipase C: a hemolysin
- Exotoxin A: causes tissue necrosis, disrupts protein synthesis) and immunosuppressive.



P. aeruginosa: Pathogenesis



- 1. Disruption of mucous membrane and skin.
- 2. Usage of intravenous or urinary catheters.
- 3. Neutropenia (as in cancer therapy).

It commonly complicates burned and cystic fibrosis patients.

P. aeruginosa is invasive and toxigenic. It attaches to and colonizes the mucous membrane or skin, invade locally, and produces systemic diseases and septicemia.

P. aeruginosa is **resistant to many antibiotics**. It becomes dominant when more susceptible bacteria of the normal flora are suppressed.







Clinical Diseases

Infection of wounds and burns

(blue-green pus). Patients with severe burns may develop into bacteremia.

Skin and nail infections

Meningitis (when introduced by lumbar puncture).

Pulmonary infection

Tracheobronchitis

Necrotizing pneumonia in CF patients: diffuse, bilateral bronchopneumonia with microabscess and necrosis.

Eye infections

Ear infections

Otitis externa: mild in swimmers; malignant (invasive) in diabetic patients. Chronic otitis media Osteochondritis of the foot. Urinary tract infection Gastrointestinal infection

Sepsis







Laboratory Diagnosis

- **Specimen:** skin lesions, pus, urine, blood, spinal fluid, sputum.
- Culture: ???
- Biochemical reaction: ???

Treatment

Combined antibiotic therapy is generally required to avoid resistance that develops rapidly when single drugs are employed. It may be sensitive to Aminoglycosides or quinolones















Bacillus anthracis



Bacillus



B. anthracis: anthrax of the animals and humans.

Morphology and Physiology

➤Large gram-positive rods, have square ends, arranged in long chains.

Spore is located in the center of the cell.

Most are saprophytic (soil, water, air, and on vegetation.)

≻Encapsulated and non-motile

Capsule consists of polypeptide (poly-D-glutamic acid)





Morphology and physiology

➤The spores can withstand dry heat and certain disinfectants for moderate periods, and persist for years in dry earth.

➤Aerobic or facultative anaerobe

➢Culture: nonhemolytic gray-white colonies with dry surface (cut glass appearance and irregular margins) on blood agar plates and grow on nutrient agar.







Pathogenesis and Immunity Virulence factors

- **Capsule** (encoded from a plasmid)
- Exotoxins (A-B toxins encoded from another plasmid)
 - Edema toxin is composed of protective antigen (B-subunit) and edema factor (EF; an adenylate cyclase). This toxin complex increases vascular permeability which leads to shock.
 - Lethal toxin is composed of protective antigen and lethal factor (LF; a metalloprotease). This toxin causes cell death and stimulates macrophages to release proinflammatory cytokines.





Pathogenesis and Immunity

Primarily a disease of herbivores (sheep, cattle, horses); humans are rarely affected. (Zoonotic).

In animals, portal of entry is mouth and GI tract. In humans, scratches in the skin (95% of infection), ingestion or inhalation lead to infection.

➤The spores germinate in the tissue at the site of entry, and growth of the vegetative forms results in gelatinous edema and congestion. *Bacillus* spread via lymphatics to the blood and other tissues.





Clinical Diseases

Progressive hemorrhagic lymphadenitis /Mediastinitis (enlargement of mediastinal lymph nodes), bloody pleural effusion, sepsis, and meningitis (50% patients). Fatal if untreated 100%

Cutaneous anthrax (malignant pustule) papule-pustule-ulcer with black eschar surrounded by marked oedema

Gastrointestinal anthrax (very rare) vomiting-pain and bloody diarrhea.







Human Cutaneous Anthrax Sampling (Suspected)



Laboratory Diagnosis

>Specimens: fluid or pus from local lesion, blood, or sputum.

Smears: long chains (a characteristic of *B. anthracis*) of large gram-positive rods with central spores can be seen.

>Immuno-fluorescence stain can be used.

Culture: nonhemolytic gray colonies with dry surface on blood agar plates.

Identification: made in a reference lab by direct fluorescent Ab test against capsular polypeptide or PCR test.

Serological tests: detection of antibodies to lethal toxin and edema toxin.

Treatment

Multi drug therapy, Ciprofloxacin, rifampin and vancomycin









