Pseudomonas aeruginosa

Presented by

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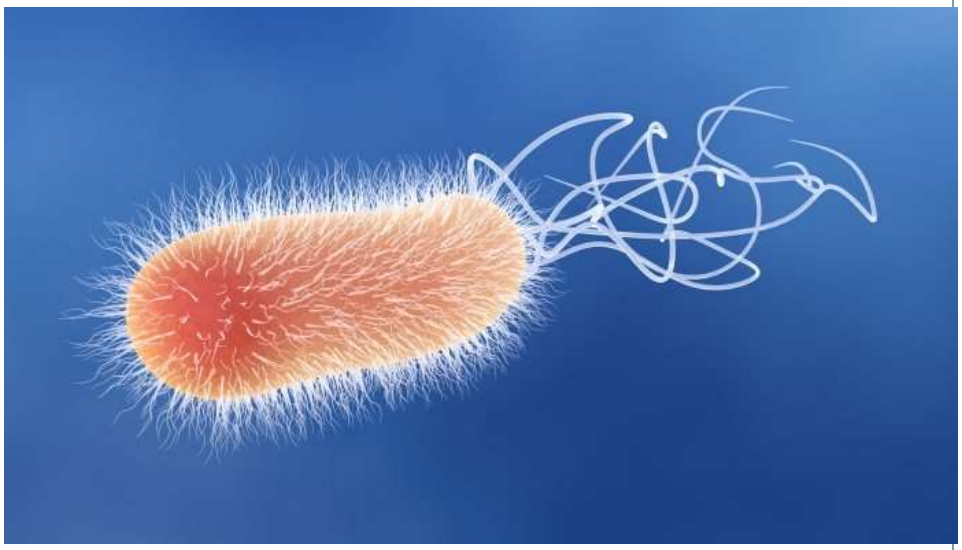
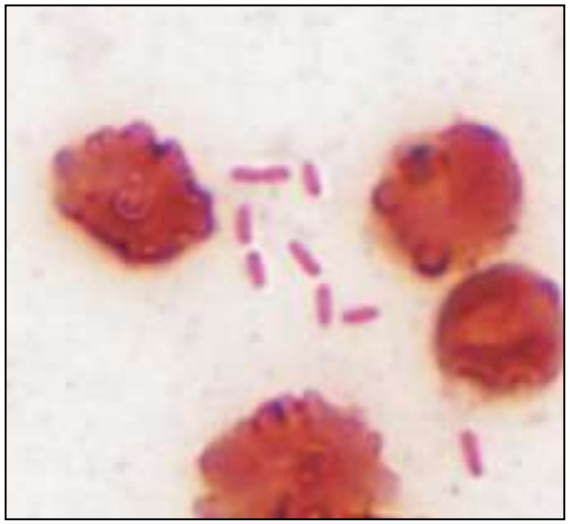
Pseudomonas aeruginosa

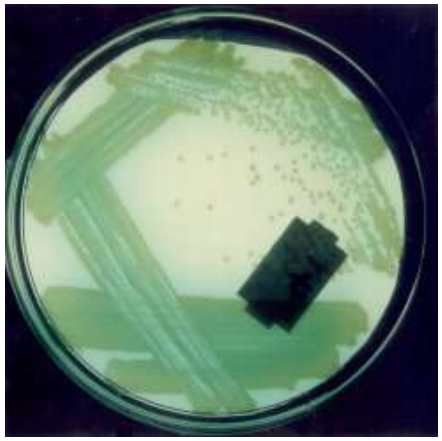
Morphology

- Gram-negative rods.
- Motile with polar flagella.
- Some strains may be capsulated.
- Non-sporing.





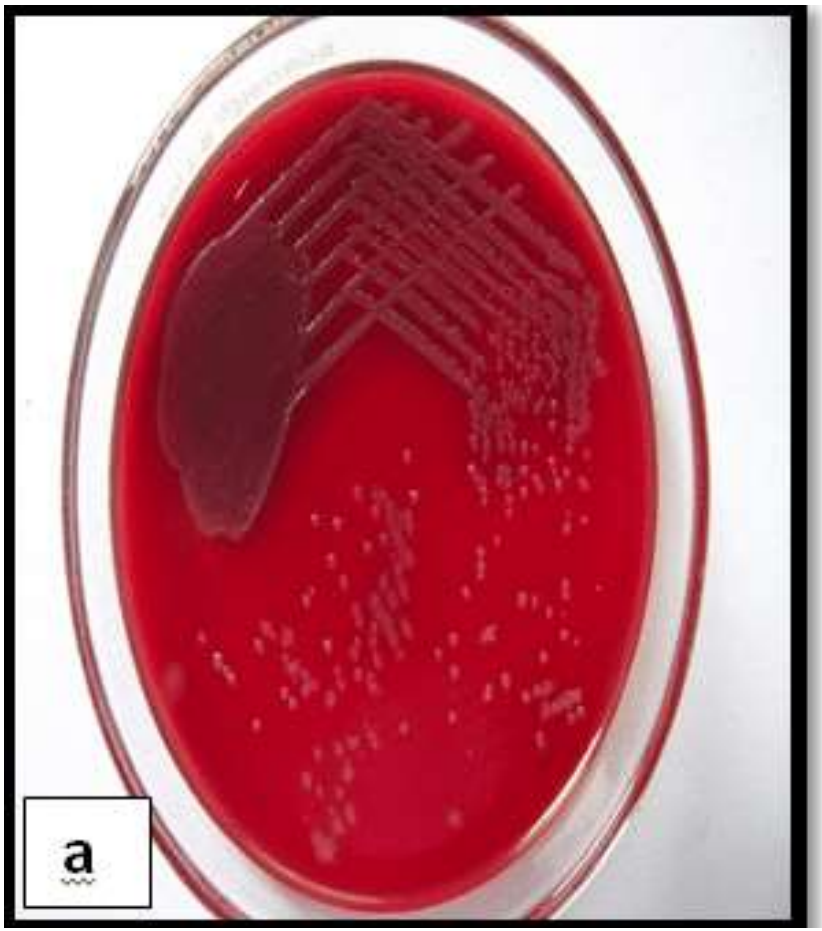
Culture characters



- 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 β-hemolysis.
- Grow at 37° to 42°
- It produces exopigments that consist of:

Pyocyanin- nonfluorescent bluish pigment

Pyoverdin- fluorescent greenish pigment

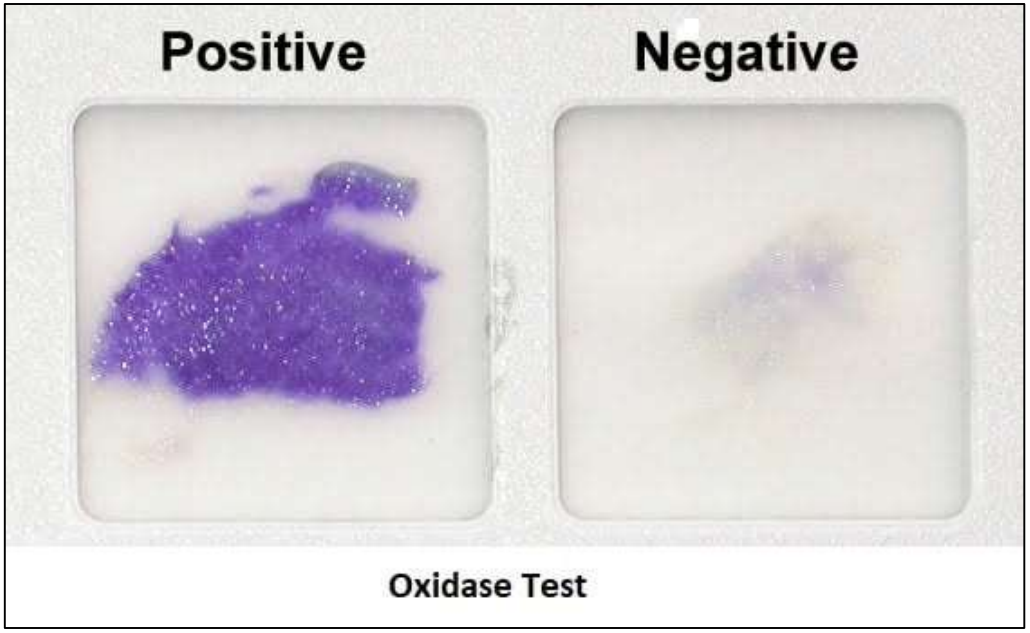


β-hemolysis on blood agar

Greenish colour on nutrient agar

Biochemical reaction

- Do not ferment carbohydrates.
- Oxidase positive



Oxidase Test

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 ºC.**

Virulence Factors

P. aeruginosa

Antigenic structure, enzymes, and toxins

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

- **Proteases**

  protease cause tissue damage and help bacteria spread.
- **Phospholipase C**: a hemolysin
- **Exotoxin A:** causes tissue necrosis, disrupts protein synthesis) and immunosuppressive.

P. aeruginosa: Pathogenesis

This organism is widely distributed in nature and is commonly present in **moist environments** in hospitals. It is pathogenic only when introduced into areas devoid of normal defenses, e.g.,

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.

P. aeruginosa

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

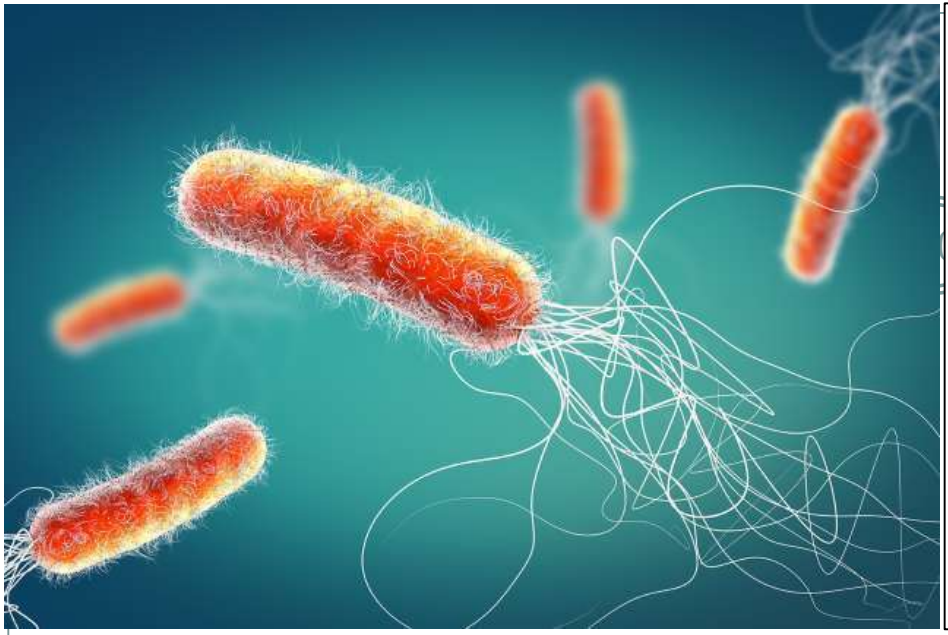
P. aeruginosa

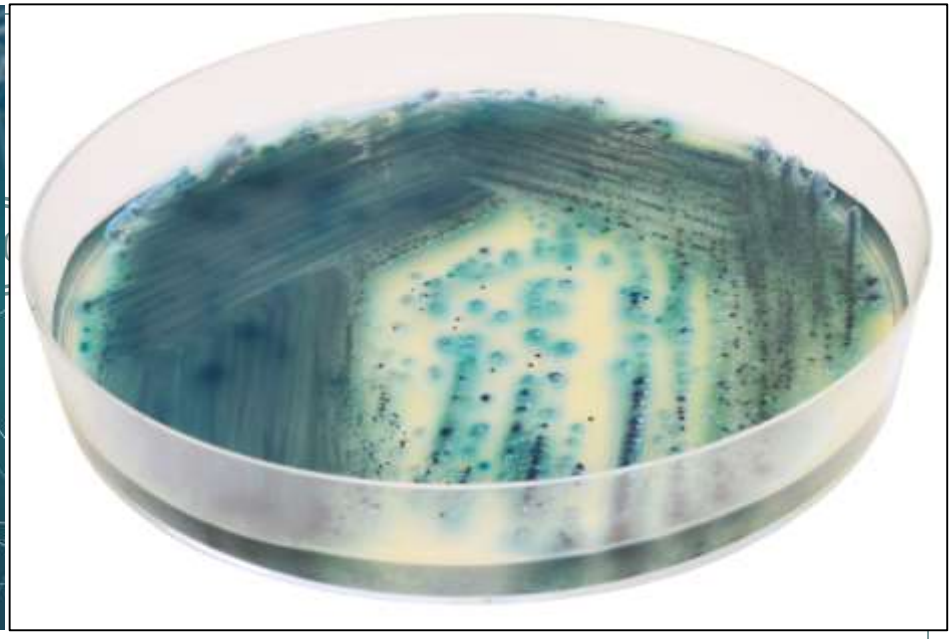
Laboratory Diagnosis

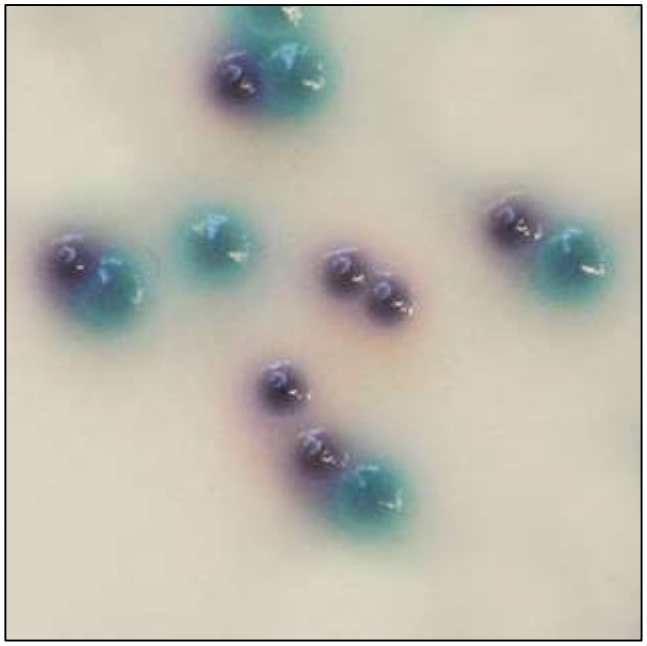
- **Specimen:** skin lesions, pus, urine, blood, spinal fluid, sputum.
- **Culture:???**
- **Biochemical reaction: ???**

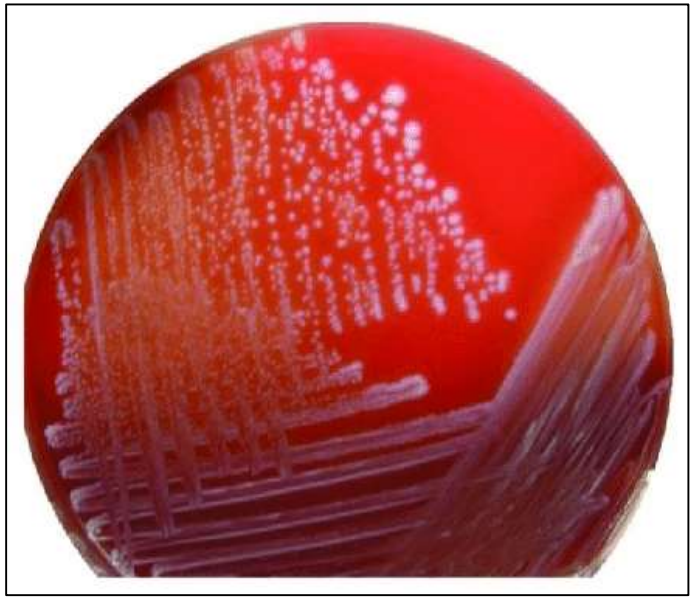
Treatment

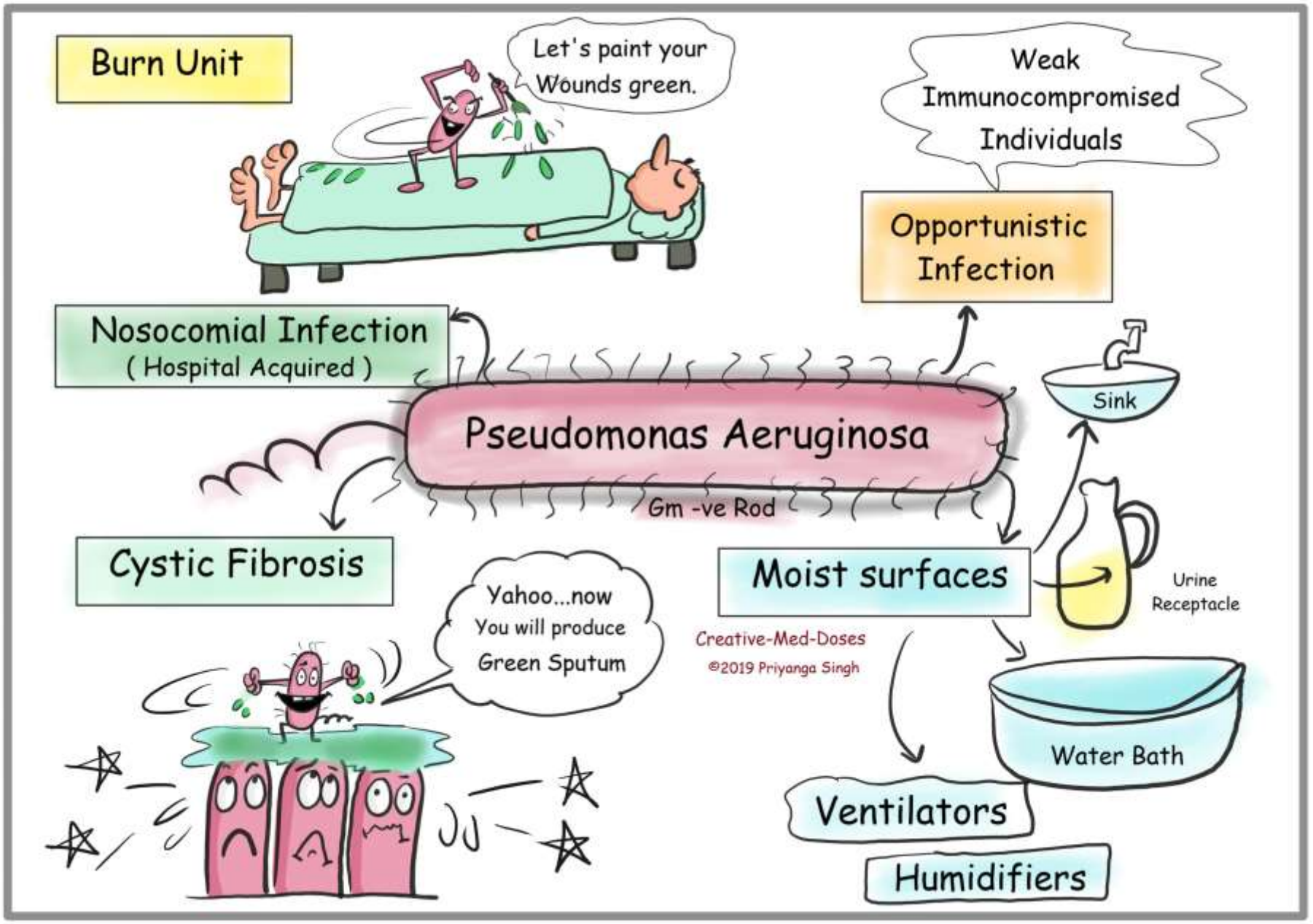
Combined antibiotic therapy is generally required to avoid resistance that develops rapidly when single drugs are employed. Aminoglycoside, antipseudomonal B-lactam or a quinolone

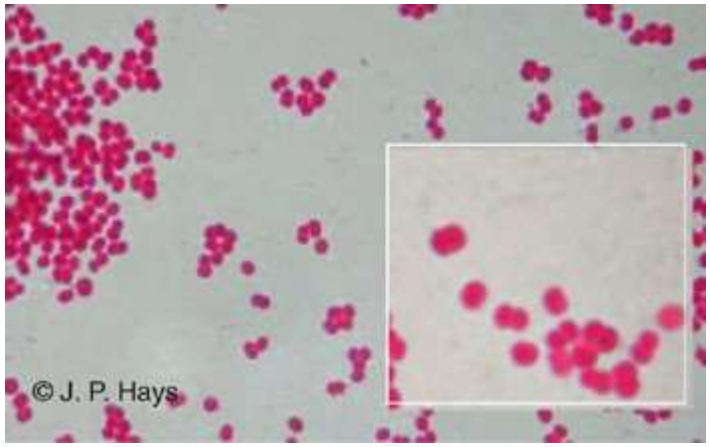




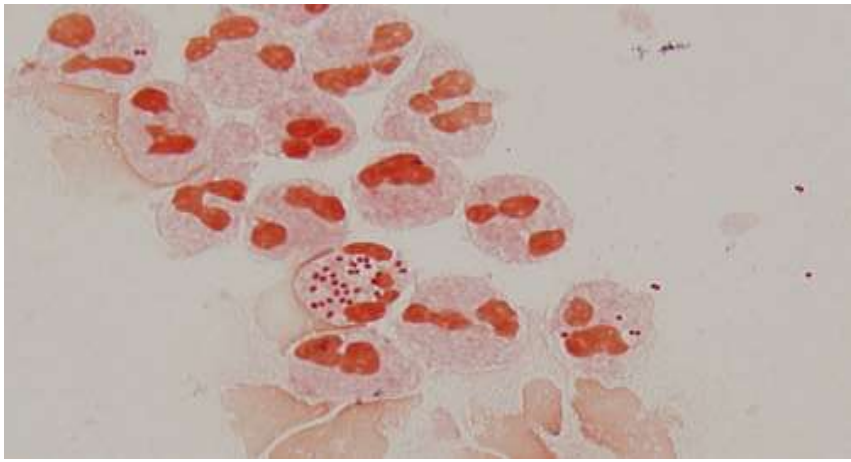


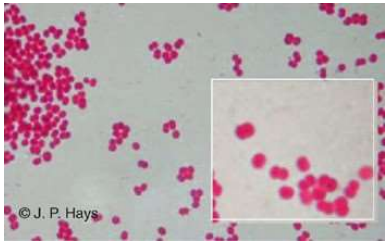



Burn Unit
Let's paint your Wounds green.
Weak Immunocompromised Individuals
Opportunistic Infection
Nosocomial Infection
(Hospital Acquired)
Pseudomonas Aeruginosa
Gm -ve Rod
Sink
Cystic Fibrosis
Moist surfaces
Urine Receptacle
Yahoo...now You will produce Green Sputum
Creative-Med-Doses
©2019 Priyanga Singh
Water Bath
Ventilators
Humidifiers



Moraxella catarrhalis

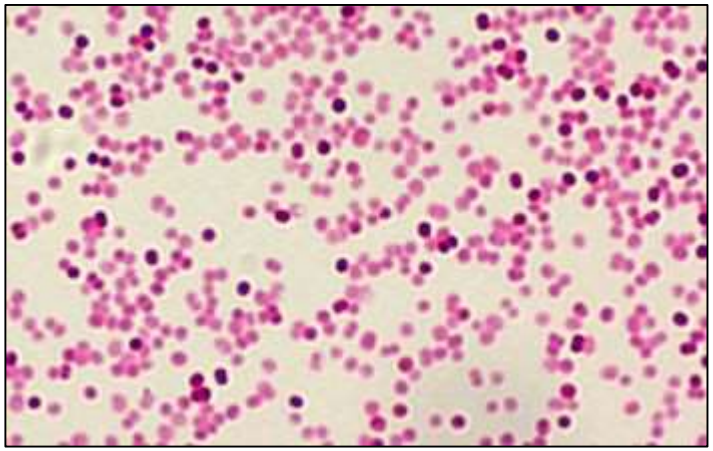




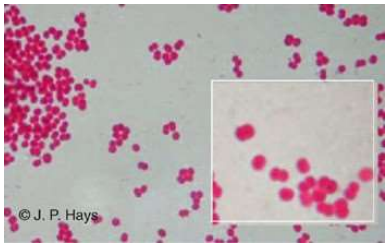
Moraxella catarrhalis

Morphology and culture characters

- Gram-negative cocci or diplococci
- Non motile
- Aerobic
- Grayish-white colonies which scoot across the agar without disruption when pushed by loop **(Hockey puck sign)**

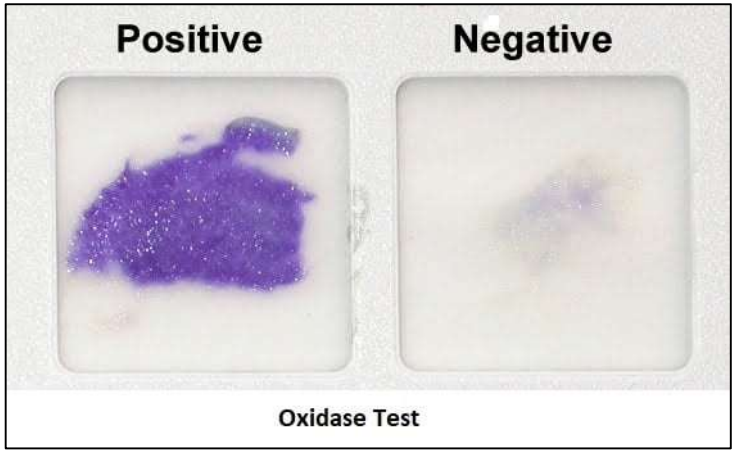


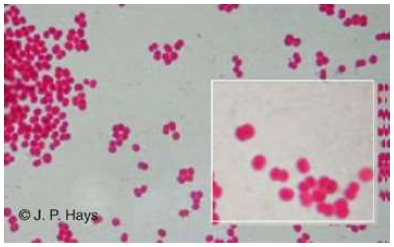
(Hockey puck sign)



Moraxella catarrhalis

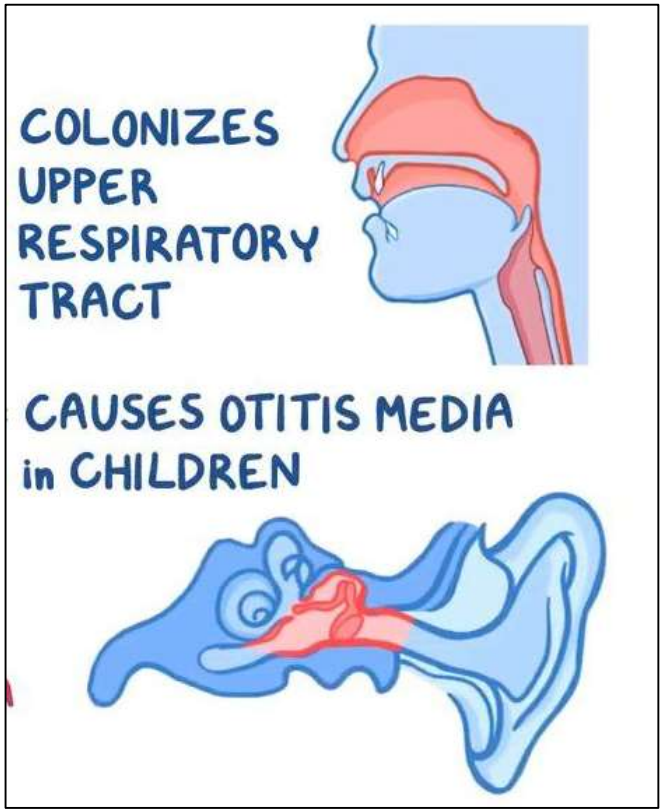
- **Biochemical reaction**
- Oxidase positive
- Catalase positive
- Non sugar fermenter
- Produce beta- lactamase
- DNase positive



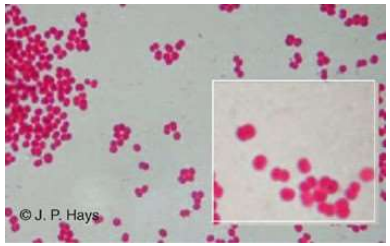


Moraxella catarrhalis

- **Clinical infections**
  - Pneumonia
  - Sinusitis
  - Otitis media (3rd most common cause)
  - Eye, CNS, Joints infection
- **Predisposing factors**
  - Advanced age
  - Immunodeficiency
  - Neutropenia
  - Other debilitating diseases



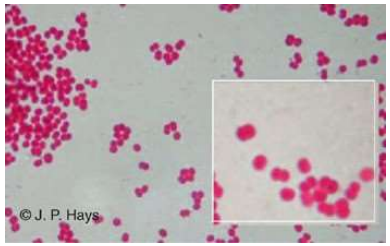
Normal commensal of the respiratory tract (humans only)
Has become an important opportunistic pathogen



Moraxella catarrhalis

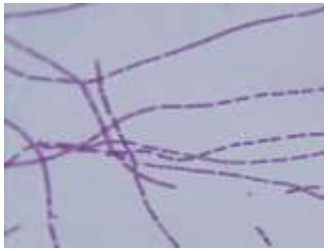
Laboratory diagnosis;

- **Sample:** pus, sputum, CSF
- **Smear:** Gram negative diplococci
- **Culture:** Smooth grayish-white colour
- **Biochemical reaction:**
- Oxidase positive
- Catalase positive
- Non sugar fermenter
- Produce beta- lactamase
- DNase positive



Moraxella catarrhalis

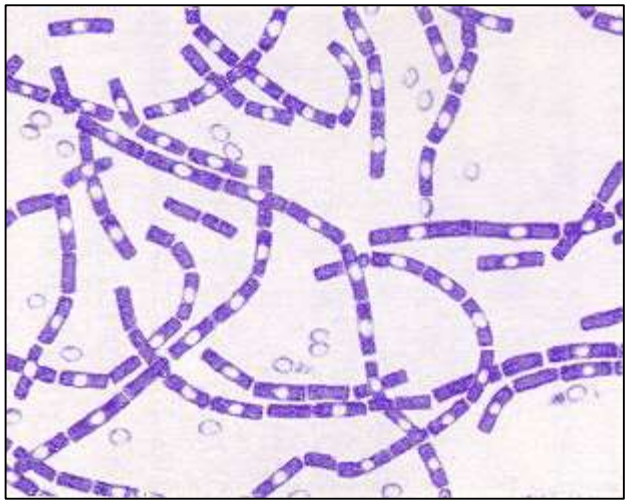
- **Treatment:** fluoroquinolones, most second and third generation cephalosporins, erythromycin, and amoxicillin-clavulanate



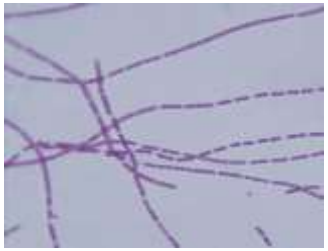
Bacillus

***B. anthracis*: anthrax of the animals and humans.**

Morphology and Physiology



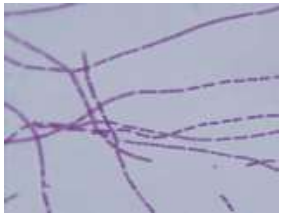
- Large gram-positive rods, have square ends, arranged in long chains.
- **Spore** is located in the center of the cell.
- Most are saprophytic (soil, water, air, and on vegetation.)
- Encapsulated and non-motile
- Capsule consists of polypeptide (poly-D-glutamic acid)



B. anthracis

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 on blood agar plates and grow on nutrient agar.

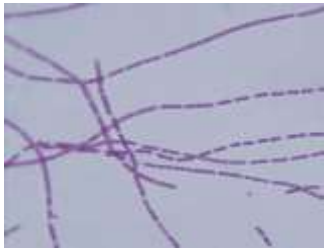


B. anthracis

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.



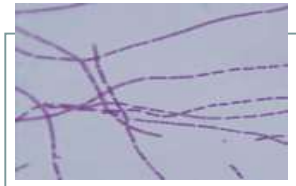
B. anthracis

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.



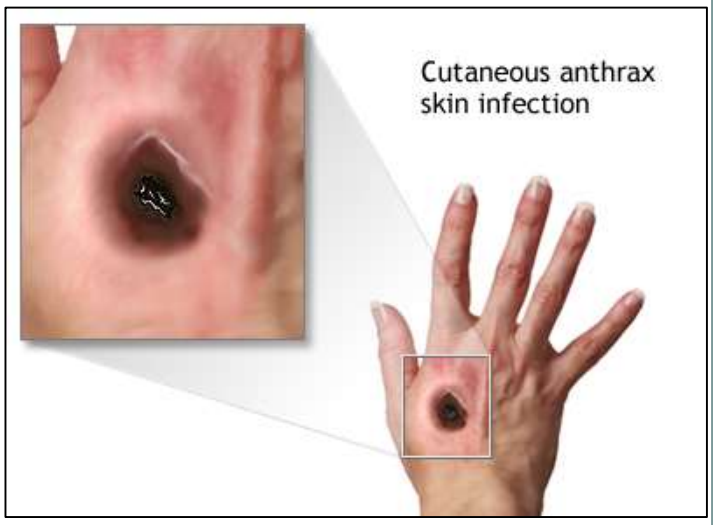
B. anthracis

Clinical Diseases

Inhalation anthrax (wool-sorters' disease): long incubation time (2 months or more).

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)




B. anthracis

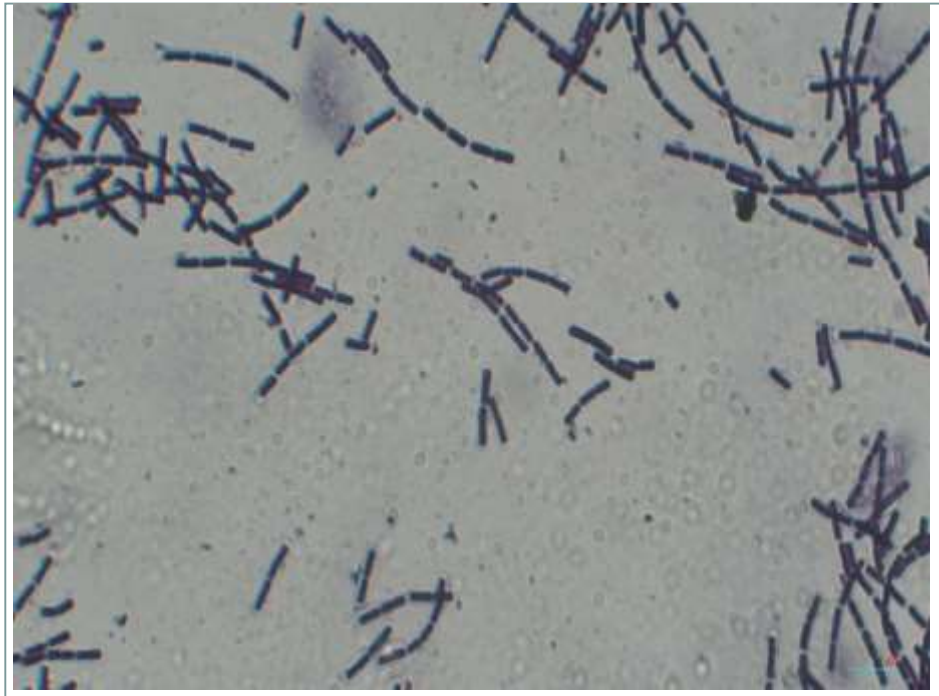
Laboratory Diagnosis

- **Specimens:** fluid or pus from local lesion, blood, or sputum.
- **Smears:** long chains (a characteristic of *B. anthracis*) of large **gram-positive rods** without 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.

B. anthracis

Treatment

Multi drug therapy, Ciprofloxacin, rifampin and vancomycin



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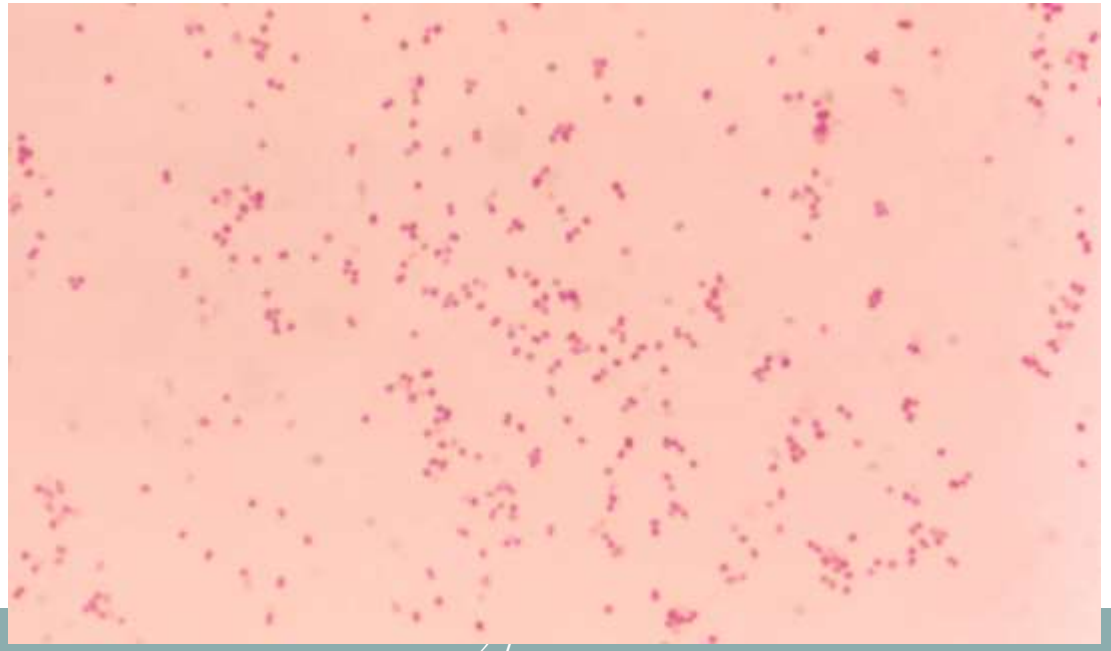


Thank
You