Pseudomonas sp

The Pseudomonads and Acinetobacters are widely distributed in soil and water as saprophytic . *Pseudomonas aeruginosa* sometimes colonizes humans (intestine and skin) and is the major human pathogen of the group.
***P. aeruginosa* is invasive and toxigenic, produces infections in patients with abnormal host defenses, and is an important nosocomial pathogen (multi-drug resistant and even grow in disinfectants used in hospitals. They are gram-negative, motile, aerobic rods some of which produce water-soluble pigments.**

PSEUDOMONAS AERUGINOSA

A. Typical Organisms

P aeruginosa is motile and slender rod shaped Gram negative bacilli (Figure 1).

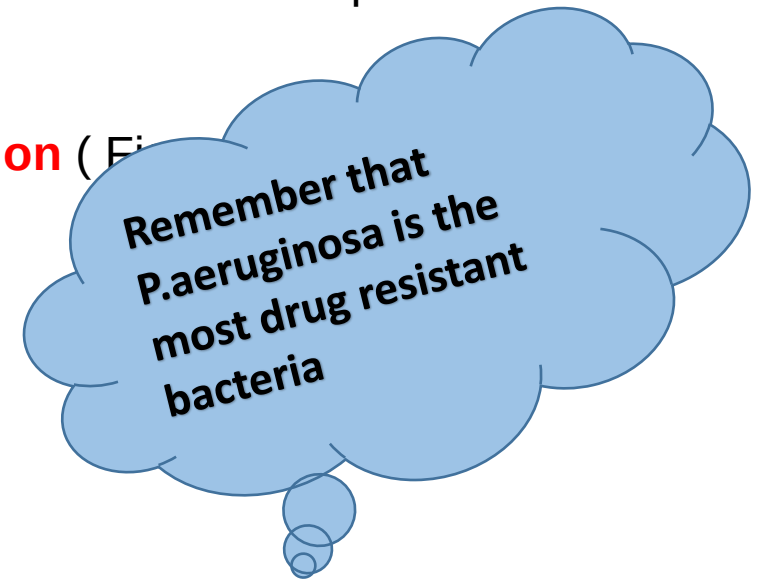
B. Culture

- *P aeruginosa* is an **obligate aerobe** that grows on any media (very simple bacteria even in tap water)
- sometimes producing **a sweet or grapelike odor**.
- Some strains **hemolyze blood** or produce darkness due to the **hemodigestion** (Fi
- It produces large, opaque, irregular colonies of butyrous consistency (Figure 3)
- It produces water soluble pigment pyocyanin which diffuses in medium (Figure 4).

Pigments are of following types:

a. Pyocyanin (bluish green).
b. Fluorescein (yellowish green).
c. Proverdin (green (fluorescent)). (Figure 5)
d. Pyorubin (red).
e. Pyomelanin (black).

- Some strain may be **non-pigmented**
- Cultures from patients with cystic fibrosis (CF) often yield *P aeruginosa* organisms that form mucoid colonies as a result of overproduction of alginate, an exopolysaccharide (Figure 6)



C. Growth Characteristics

- *P aeruginosa* grows well at 37–42°C; its growth at 42°C helps differentiate it from other *Pseudomonas* species in the fluorescent group.
- It is **oxidase positive.**
- It does not ferment carbohydrates, but many strains oxidize glucose (TSI= K/K) (Figure 7).
- Identification is usually based on colonial morphology, oxidase positivity, the presence of characteristic pigments, and growth at 42°C.

Cetrimide agar is the selective media for P.aeruginosa which inhibit other bacteria including other species of Pseudomans and Colonies exhibit a blue-green to green pigment (Figure 6) and fluoresce under short wavelength (254 nm) ultraviolet light (Figure 8).

Antigenic Structure and Toxins

Pili (fimbriae) extend from the cell surface and promote attachment to host epithelial cells. The exopolysaccharide capsule is responsible for the mucoid colonies seen in cultures from patients with CF. The lipopolysaccharide, which exists in multiple immunotypes, is responsible for many of the endotoxic properties of the organism. ***P aeruginosa*** **can be typed by lipopolysaccharide immunotype and by pyocin (bacteriocin) susceptibility or by molecular methods using RE**. Most *P aeruginosa* isolates from clinical infections produce extracellular enzymes, including elastases, proteases, and two hemolysins (a heat-labile phospholipase C and a heat-stable glycolipid).

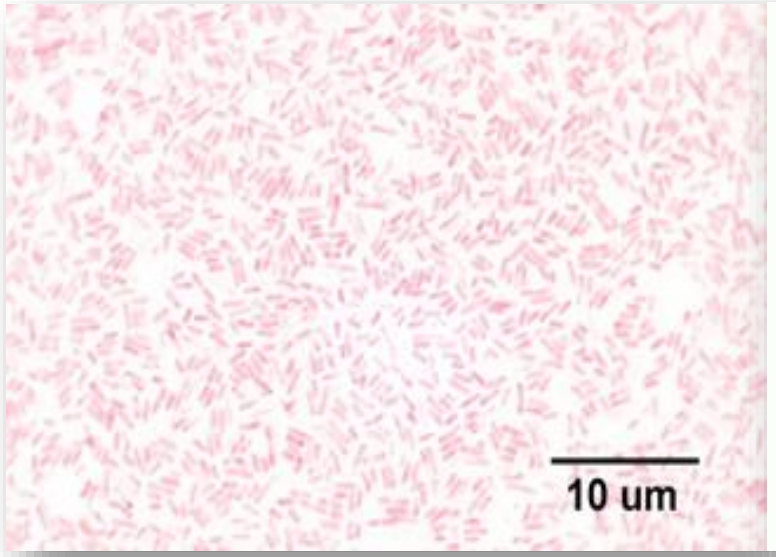


Figure 1. Gram reaction of P.aeruginosa

Figure 2. Hemodigestion caused by P.aeruginosa

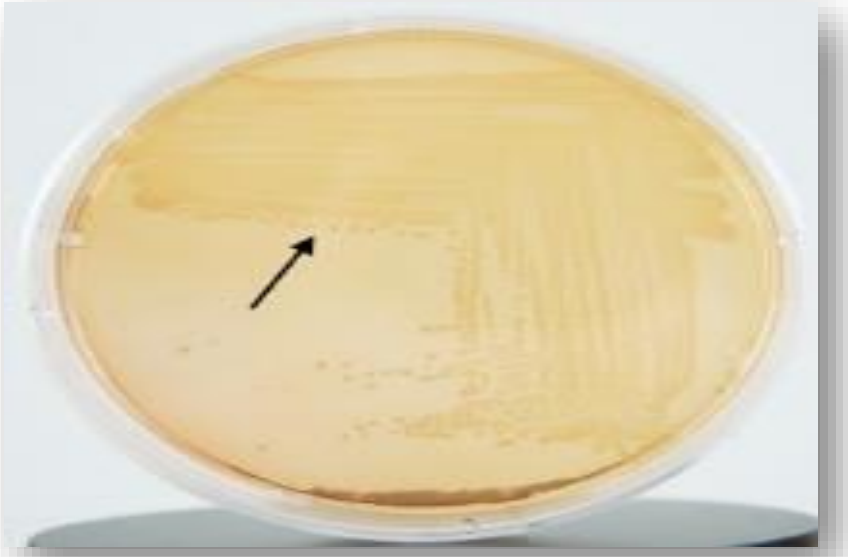


Figure 3. Colonies of P.aeruginosa

Figure 4. Bluish coloration of nutrient agar by P.aeruginosa

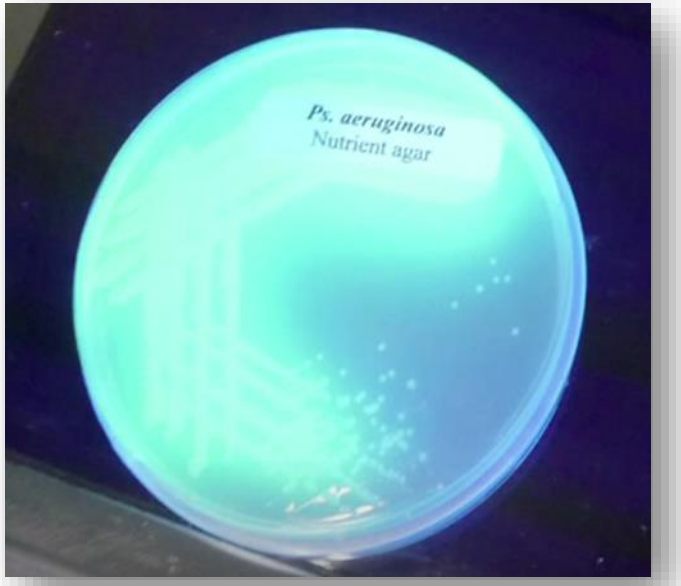


Figure 5. Fluorescent colonies of P.aeruginosa

Figure 6. large mucoid colonies of P.aeruginosa

Figure 7. TSI (K/K)

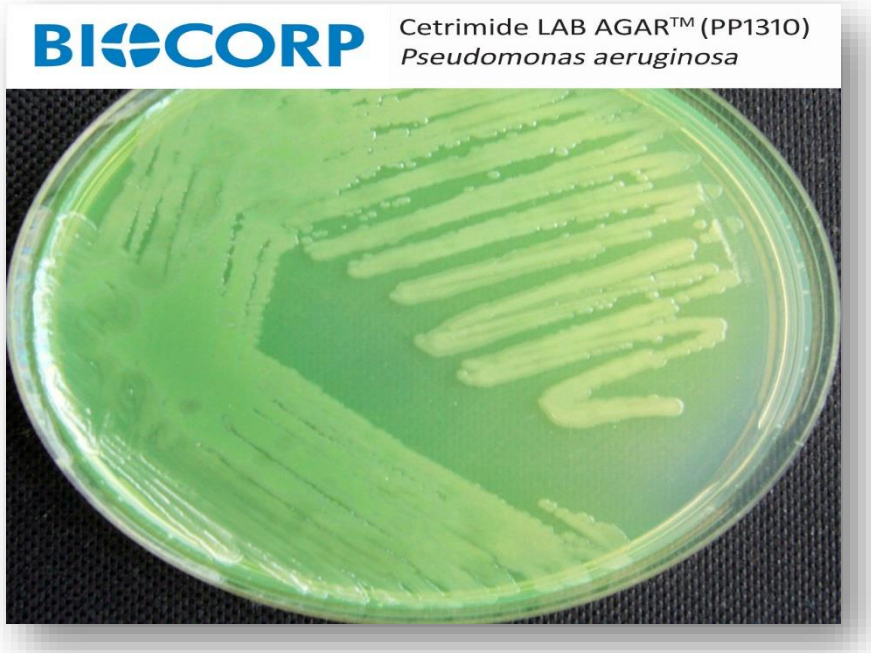


Figure 8. Growth of P.aeruginosa on cetrimide agar

Many strains of *P aeruginosa* produce **exotoxin A**, which causes tissue necrosis and is lethal for animals when injected in purified form. The toxin blocks protein synthesis by a mechanism of action identical to that of **diphtheria toxin**, although the structures of the two toxins are not identical.

Pathogenesis: *Pseudomonas aeruginosa* is one of the **most troublesome agents** causing **nosocomial infections**. It is commonly encountered in secondary infection of **wound, burns and chronic ulcers of skin**. The bacterium attaches to and colonizes the mucous membranes of skin, invades locally and produces systemic infection. These processes are promoted by **pili, enzymes and toxins**. Besides these, **lipopolysaccharide** plays a direct role in causing **fever, shock, oliguria, leukocytosis, leukopenia, disseminated intravascular coagulation and adult respiratory distress syndrome**.

Clinical findings

P. aeruginosa cause infections in immunocopromised person (Cancer like leukemia and lymphoma) or presence of predisposing factors. Causes nosocomial infection and multiple drug resistant.

1- Wound and burn infections (Blue green pus)

2- Meningitis during contaminated lumber puncture

3- UTI in indwelling catheterized patients

4- Necrotizing pneumonia through contaminated respirators

5- Otitis in among swimmers and diabetics

6- Eye infection due to trauma or contaminated surgical operation

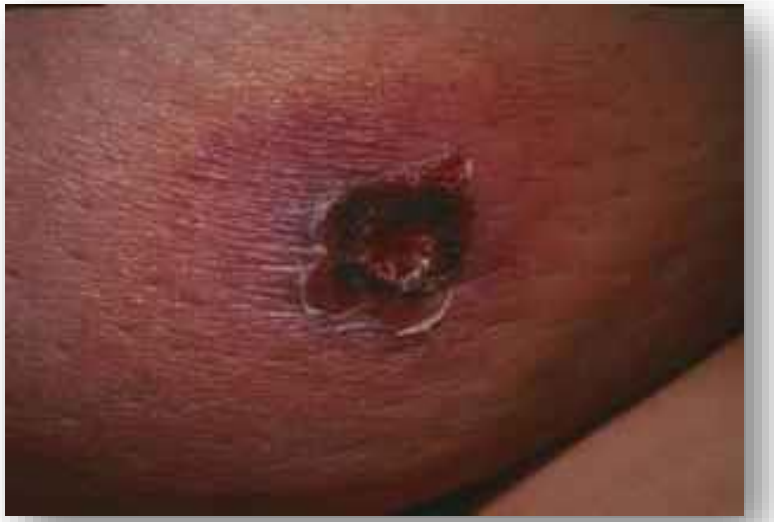


Figure 9. Ecthyma gangrenosum caused *P.aeruginosa*

7- Septicemia in cancer patients

8- Hemorrhagic necrosis of the skin (ecthyma gangrenosum) surrounded by erythema and often don not contain pus (Figure 9)

8- Infantile diarrhea- enterotoxin

Diagnostic Laboratory Tests

A. Specimens

Specimens from skin lesions, pus, urine, blood, spinal fluid, sputum, and other material should be obtained as indicated by the type of infection.

B. Smears

Gram-negative rods are often seen in smears. No specific morphologic characteristics differentiate pseudomonads in specimens from enteric or other gram-negative rods.

C. Culture

Specimens are plated on blood agar and the differential media commonly used to grow the enteric gram-negative rods. Pseudomonads grow readily on most of these media. *P aeruginosa* does not ferment lactose and is easily differentiated from the lactose-fermenting bacteria. Oxidase positive and sugar non fermenter (TSI K/K).

Treatment

An extended spectrum penicillin such as piperacillin active against *P. aeruginosa* is used in combination with an aminoglycoside, usually tobramycin. Other drugs active against *P aeruginosa* include aztreonam; carbapenems such as imipenem or meropenem; and the fluoroquinolones, including ciprofloxacin. Of the cephalosporins, ceftazidime, cefoperazone, and cefepime are active against *P aeruginosa;* ceftazidime is often used with an aminoglycoside in primary therapy of *P aeruginosa* infections, especially in patients with neutropenia.

The susceptibility patterns of *P aeruginosa* vary geographically, and susceptibility tests should be done as an adjunct to selection of antimicrobial therapy. Multidrug resistance has become a major issue in the management of hospital-acquired infections with *P aeruginosa* because of acquisition of chromosomal β-lactamases, extended-spectrum β-lactamases, porin channel mutations, and efflux pumps.

Acinetobacter baumannii

Aerobic Gram negative coccobacilli, diplococcic (resemble Neisseria) or rode shapes (Figure 10) widely distributed in the soil and water. Aerobic and non sugar fermentative and multi-drug resistant

Grow on most media (similar to P. aeruginosa but oxidase negative and non motile)

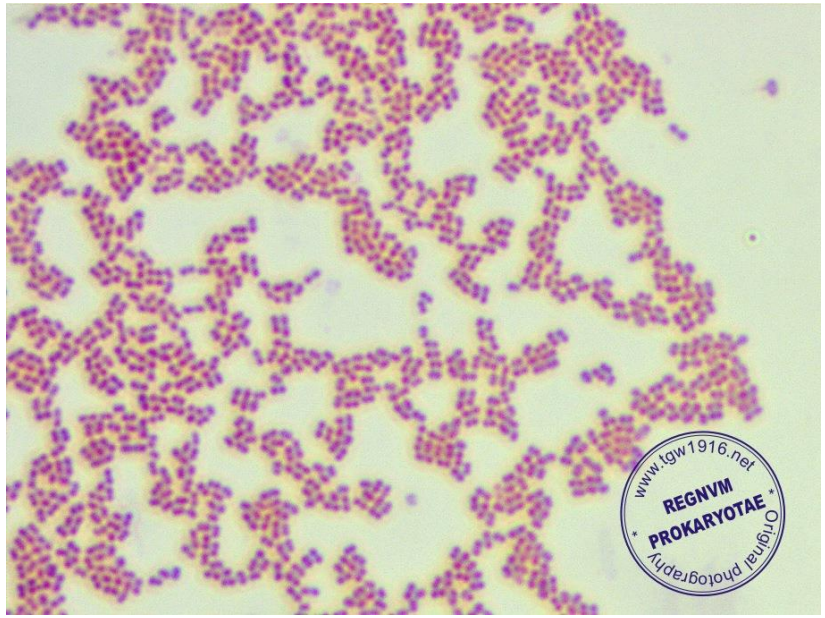


Figure 9. Acinetobacter baumannii morphology

Commensal but can cause the following infections

1- Wound infections (recorded among USA marine wounded soldiers in Iraq)
2- Meningitis
3- Pneumonia
4-UTI