Pseudomonas aeruginosa

- (Gram-) rods
- motile (polar flagella).
- Some encapsulated
- non-sporing.

- strict aerobic.
- Grow anaerobically in nitrate agar (some gram-ve rods)
- some strains → β haemolysis on blood agar
- Grow at 35 to 42°.
- produce oxidase → Pyocyanin → pyocyanin-pigmented blue pigment, Fluorescein → fluorescent yellowish green pigment.
- Oval barrel carbohydrates.
- resistant (+)

Virulence factors

- pili → attachment to host cell.
- Capsule → keep in cytosol from protease (cystic fibrosis).
- lps - endotoxin → Multiple immunogens.
- Pyocyanin → production of toxic ions of O2 that cause tissue damage (Cytotoxin).
- Pyocyanin → accumulate → Cytosol iron and pH result bacteria.
- Proteases → cause tissue damage & help bacteria spread
- phospholipase C → hemolysin.
- Exotoxin A → cause tissue necrosis & disrupt protein synthesis & immune suppression.

Pathogenesis

- widely distributed in nature (moist environment in hospitals).
- Pathogenic only when introduced into sites found at normal defenses
  - Adhesion of micro. surfaces & tissues
  - Entry of GI / urinary tract
  - Multiplication (tissue damage).
- complicates burned & cystic fibrosis patients.
- P. aeruginosa in wounds & tissues
- attaches & colonizes the mucus membrane/skin.
- invade (multiply) & produce systemic disease & septicemia.
- Resistance to many antibiotics
- Diminished when host susceptible because of the normal flora are removed

clinical diseases

- infection of wound & burns (blue-green pus)
  - patients with severe burns may develop into bacteremia
- SSTI & soft tissue infections.
- Meningitis (often introduced by lumbar puncture).
- pulmonary infection
  - infects bronchitis
  - Necrotizing pneumonia
    - in CF patients diffuse, bilateral bronchopneumonia with microabscesses & fibrosis.
- Eye infection.
- Ear infection → otitis externa and in swimmers; malignant (invasive) in diabetic patients; chronic otitis media.
- Otitis chondritis of the pinna.
- necrotizing hoof infection
- Genito-intestinal infection
- Sepsis.

Moraxella Catarrhalis

- Gram (-) cocci & diplococci.
- non motile.
- aerobic
- Grayish white colonies + without disruption when pushed by tip.
- Hockey puck sign.
- Oxidase (+)
- catalase (+)
- non sugar fermenter.
- produce β-lactamase.
- DNase (+)

Biochemical Reaction

clinical infection

- Pneumonia.
- Sinusitis.
- otitis media. (3rd most common).
- Eye, CNS, Soft tissue infection.

predisposing factor.

- Advanced age.
- Immunodeficiency.
- Neutropenia.

Bacillus anthracis

- Large gram (+) rods / square ends / arranged in long chains. (in smears → immuno-fluorescence can be used.
- Spore: in the center of the cell → spores can withstand dry heat & certain disinfectants for extended periods & persist for years in dry earth.
- Most are saprophytic (soil, water, air, on vegetation).
- Encapsulated & non-motile.
- Capsule consists of polypeptide (poly-D-glutamic acid).
- Aerobic / facultative anaerobe.
- Culture → non-hemolytic grey-white colonies with dry surface on blood agar plates & grows on nutrient agar.

Virulence Factor.

- Capsule → encoded from plasmid1
- Exotoxin (A-B toxin) encoded from another plasmid2.
  - edemic toxin → protective antigen (B-subunit) & edema factor (EF = adenylate cyclase). edema toxin complex → vascular permeability → shock
  - lethal toxin → protective Ag & lethal factor (LF = metalloproteinase) → cell death & stimulate NF to release pro-inflammatory cytokines.

Pathogenesis

- Disease of herbivores (sheep, cattle, horses) → human are rarely affected zoonotic
- In animal → portal of entry is mouth & GI tract
- In human → Scratches in the skin (95% of infections), ingestion or inhalation → skin infection
- Spores germinate in the tissue at the site of entry & growth of vegetative form results in → gelatinous edema & congestion.
- Bacillus spread via lymphatic to the blood & other tissues.

clinical diseases

- inhalation anthrax (wool-sorters disease) → high mortality (infection time (3 month or more) progressive hemorrhagic lymphadenitis/mediastinitis (enlargement of mediastinal lymph nodes). bloody pleural effusion. Sepsis & meningitis (GI patients) → failure of untreated (90%).
- Cutaneous anthrax (malignant pustule) → papule → pustule ulcer with black eschar surrounded by marked edema
- Gastrointestinal anthrax (overfeed) → vomiting pain & bloody diarrhea.

Identification

Made in reference lab by direct fluorescent Ab test against capsular polypeptide or PCR test.

Serological tests

→ Detection of Ab to lethal toxin & edema toxin

Treatment

→ Multi drug therapy