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THE GENUS CLOSTRIDIUM
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- **Anaerobic** spore forming Gram-positive bacilli.
- Found in soil & in normal intestinal flora of man and animals.
- Exotoxin(s) play an important role in disease pathogenesis.
- Form spores, its position is useful in species identification.
- Shows optimum growth when plated on blood agar at 37°C.
- Most are saprophytes but a few are pathogenic for humans.
- **C. tetani** and **C. botulinum** produce the most potent human biological toxins.
Species

- *C. tetani*: Tetanus.
- *C. perfringens*: Gas gangrene.
- *C. botulinum*: Botulism.
- *C. difficile*: Pseudomembranous colitis.
Clostridium tetani

- Cl tetani is the causative agent of tetanus.
- Found in soil, intestinal tracts and feces of humans and various animals.
- Carrier rates in humans vary from 0 to 25%.
Clostridium tetani, *morphology*

- Gram-positive bacilli motile with peritrichate flagellae
- Non capsulated
- Forms round terminal spores, (drumstick appearance)
- By Gram-stain the spore remains unstained.
- There are 11 strains of C. tetani differed on the basis of flagellar antigens.
C. tetani produces two toxins

a. Tetanolysin:
   • A hemolysin that has no role in Pathogenesis.

b. Tetanospasmin:
   • A neurotoxin responsible for the symptoms of tetanus
   • All C. tetani strains produce a toxin with identical immunological and pharmacological properties.
   • The toxin is heat labile, destroyed at 56 °C in 5 minutes, and is oxygen labile.
   • The toxin rapidly converts to toxoid in the presence of formalin.
   • Toxin action: It blocks the release of inhibitory neurotransmitters, so it produces the generalized muscular spasms characteristic of tetanus.
Clostridium tetani, *pathogenesis*

- Injury to host tissue (wounds, umbilical stump, surgical suture), contaminated with C. tetani spores that germinate and produce tetanospasmin.
- C. tetani is not an invasive organism.
- Infection remains localized and toxin reaches the central nervous system along neural axons or by bloodstream.
- The clinical pattern of tetanus is spasms, rigidity of the voluntary muscles and convulsions (trismus, lock jaw).
- Death occurs due to respiratory failure.
Clostridium tetani, *diagnosis*

- Patient should be treated on a clinical basis.
- Anaerobic culture of tissues from wounds: culture on blood agar at 37°C.
- C tetani produces colonies with complete haemolysis on blood agar and swarming.
- Has terminal spherical spores.
- Produces indole, but does not produce acid from glucose.
- Isolation of C.tetani must be confirmed by production of toxin and its neutralization by specific antitoxin.
Clostridium tetani, *control*

- Tetanus toxoid is used for immunization, as part of DPT vaccine.
- Three injections are given in the first year of life, and a booster is given about a year later, and again on the entrance into elementary school.
- Booster doses are recommended only every 10 years.
1. Antitoxin:
   - Tetanus antitoxin prepared in animals or humans (human antitoxin is preferred), can neutralize the toxin before it becomes fixed into nervous tissue.
   - Dose ranges from 500 IU to 3000-6000 IU injected intramuscularly in a single injection.

2. Surgical measures: Removal of necrotic tissue that is essential for proliferation of the organism.

3. Antibiotics: Penicillin or metronidazole is used to kill bacteria and stops further toxin production.

4. Tetanus toxoid:
   - In cases of clean, minor wounds, tetanus toxoid should be given if the patient has not had a booster dose within the past 10 years.
   - For more serious wounds, toxoid should be given if patient has not had a booster dose within the past 5 years.
C. perfringens

• C. perfringens causes
  – wound infections that lead to gas gangrene,
  – anaerobic cellulitis,
  – severe uterine infections,
  – food poisoning.

• Spores are highly resistant to disinfectants and boiling for long period.

• C. perfringens, has five types, designated A, B, C, D, and E.

• Each of these types produces specific protein toxins.
Toxins and enzymes

- C. perfringens produces a large variety of enzymes and exotoxins.

1. **Alpha toxin** (lecithinase):
   - It is lethal and necrotizing; it lyses cell membrane lecithins, causing cell death. Can be detected by Nagler’s reaction.

2. **Theta-toxin** (oxygen-labile cytolysin) are important in the disease pathology, causes tissue destruction by several mechanisms.

3. **Extracellular enzyme**: as hemolysins, proteases, lipases, collagenase and hyaluronidase, contribute to the invasive process.

4. **Enterotoxins**: are produced by some strains of C. perfringens.
Pathogenesis of gas gangrene

- Gas gangrene is a disease with a poor prognosis and often fatal outcome.
- Occurs after severe traumatic wound, lacerated with muscle damage.
- Spores vegetate, clostridia multiply producing toxins, causing more tissue necrosis and systemic toxemia.
- Infected muscle is discolored, edematous with a foul-smelling exudate; gas bubbles from anaerobic fermentation.
- Clostridial wound infections usually are polymicrobial.
- In gas gangrene, the primary pathogen can be any one of clostridial species including C perfringens, C novyi, C septicum, or C histolyticum.
Diagnosis of gas gangrene

• Diagnosis of clostridial wound infections is based on clinical symptoms.
• Patient should be treated without waiting for laboratory confirmation.

**Specimens:** Material from wounds, pus, tissue, exudates, which are examined by:

• A. Gram stains:
  – Gram-positive, sporulated,
  – Non motile,
  – Capsulated bacilli.
Diagnosis of gas gangrene

B. Culture of clinical specimens:
• *perfringens* is obligate anaerobe.
• grow on blood agar with complete haemolysis.
• On egg yolk-glucose agar or serum agar colonies are surrounded by opaque white precipitate due to production of lecithinase (Nagler ‘s reaction).
Prevention & treatment

- Penicillin G (to kill the organism)
- Hyperbaric oxygen (to inhibit growth of clostridia).
- Antitoxin: which should be administered systemically as early as possible.
- Sanitary: Early cleansing of any wound, the surgical removal of affected tissues.
- Prevention of wound contamination is the most important factor in controlling clostridial wound infections.
C. botulinum

- Anaerobic Gram-positive bacilli motile by peritrichous flagella with oval, subterminal spores.
- The spores of C. botulinum can survive boiling (100 degrees at 1 atm) for more than one hour although they are killed by autoclaving.
- It is widely distributed in soil, and intestinal tracts of birds, mammals and fish.
C. Botulinum, *toxin*

- Seven antigenic types of toxins exist.
- The toxins are designated A, B, C1, D, E, F, and G.
- Types A, B, E and F are the most toxic for humans.
- Type A is the most potent exotoxin known (10 ng can kill a normal adult).
- Toxin is heat-labile.
C. Botulinum, *toxin*

- Toxin binds to neuromuscular junctions of parasympathetic nerves and interferes with acetylcholine release, causing flaccid muscle paralysis.
C. Botulinum, *pathogenesis*

- Depends on neurotoxin production.
- Cause disease in 3 ways:
  
  **1. Food poisoning:**
  - The botulinum toxin is ingested with food (specially home canned) in which spores have germinated and the organism has grown.
C. Botulinum, *pathogenesis*

2. Wound botulism

- a rare disease, results from *C. botulinum* growing in the necrotic tissue of wound.
C. Botulinum, *pathogenesis*

3. **Infant botulism**: germination of spores in the GIT.
   - At its site of entry into the body, spores vegetate, cells replicate and release toxin, the toxin passes into the blood stream.
   - Toxin binds to neuromuscular junctions of parasympathetic nerves and interferes with acetylcholine release, causing flaccid muscle paralysis.
   - Clinical symptoms of botulism begin 18-36 hours after toxin ingestion.
   - Neurologic features develop: blurred vision, inability to swallow, difficulty in speech, descending weakness of skeletal muscles and respiratory paralysis.
C. Botulinum, \textit{prevention}

- Proper food handling and preparation.
- Boiling of contaminated food will inactivate the toxin, as it is heat-labile.
- Food containers that bulge may contain gas produced by C. botulinum and should not be opened or tasted.
- A multivalent toxoid produces good protective antibody response.
- Its use is unjustified due to the infrequency of the disease.
C. Botulinum, diagnosis

- Demonstrating toxin in the patient's feces, serum, or vomitus.
- Fecal samples are the best specimens for detecting toxin in food poisoning or infant botulism.
- Toxin is usually detected by its lethal effect in mice coupled with neutralization of this effect by specific antisera.
C. Botulinum, *treatment*

- For both food poisoning and wound botulism, antitoxin therapy is most effective if administered early.
- Once the botulinum toxin has bound to nerve endings, its activity is unaffected by antitoxin.
- Individuals known to have ingested food with botulism should be treated immediately with antiserum.
- Any circulating toxin can be neutralized by intravenous injection of antitoxin.
C. difficile

- Slender, gram-positive anaerobic bacillus
- Produces large, oval sub terminal spores.
- It is non hemolytic.
- C difficile cause antibiotic associated diarrhea and
- pseudomembranous colitis
C difficile, *pathogenesis*

- C difficile disease is caused by the overgrowth of the organism in intestinal tract, primarily in the colon.
- Organism replicates and secretes two toxins. Toxin A which is an enterotoxin that causes fluid accumulation in the bowel, toxin B which is a potent cytotoxin.
- Nearly all toxigenic strains produce both toxins A and B.
- People in good health usually do not get C. difficile disease, as this species can't compete with normal intestinal flora but, when antibiotics eliminate these normal flora, C. difficile can flourish and produce disease.
- Individuals who use antibiotics for prolonged periods and elderly or who are immunocompromised, are at risk.
C. difficile, pathogenesis

Clostridium difficile spores and vegetative cells are ingested.

Most vegetative cells are killed in the stomach, but spores can survive the acid environment.

C. difficile spores germinate in the small bowel upon exposure to bile acids.

Flagella facilitate C. difficile movement; a polysaccharide capsule discourages phagocytosis.

Gut mucosa facilitates adherence to the colonic epithelium.

C. difficile vegetative cells produce toxins A and B and hydrolytic enzymes (1). Local production of toxins A and B leads to production of tumour necrosis factor-alpha and proinflammatory interleukins, increased vascular permeability, neutrophil and monocyte recruitment (2), opening of epithelial cell junctions (3) and epithelial cell apoptosis (4). Local production of hydrolytic enzymes leads to connective tissue degradation, leading to colitis, pseudomembrane formation (5) and watery diarrhea.
C difficile, diagnosis

- Diagnosis of C difficile disease includes Diarrhea associated with antibiotic therapy in the preceding 4 to 6 weeks, associated with the isolation of C difficile and the presence of toxin A and/or toxin B in the stool.

**Toxins can be detected by:**

- a. cellular cytotoxicity test: C difficile if added to cultured mammalian cells causes a cytopathic effect that is neutralized with specific antiserum.
- b. A latex agglutination test which is widely used.
- c. Enzyme-linked immunoassays: can be used to detect both toxin A and B
C difficile, *treatment*

- Vancomycin or oral metronidazole are used to treat active disease.
- In many cases, antibiotic treatment is not needed, as symptoms resolve 1-14 days after the offending antibiotic is discontinued.
C difficile, prevention

- Judicious use of antibiotics.
- Use of contact precautions with patients with known or suspected cases of disease.
- Health care workers caring for patients infected with C. difficile should wear gloves and strictly adhere to proper hand washing procedures.
- Implementation of an effective environmental and disinfection strategy.
Other G+ve bacilli
Actinomycetes

- Fungus-like bacteria that form filamentous branches.
- Obligate anaerobes
- Reside in the mouth and in the intestinal tract.
- Trauma leads to pathogenic proliferation of the organisms causing actinomycosis.
- Abscesses and swelling at the site of infection.
- Diagnosis:
  - Microscopic examination of pus.
  - The fluid will have a granular texture which is caused by sulfur granules. These sulfur granules are actually composed of the bacterium and its waste.
- The species most commonly associated with actinomycosis is *A. israelii*.
- Actinomycosis can often be treated with penicillin.
Lactobacillus

- Non-spore-forming, Gram positive bacilli
- Ferment glucose into lactic acid
- Catalase positive and can grow on tomato juice agar (pH 5).
- Some species are found as saprophytes in vegetables and animals
- Others are present in oral, gastrointestinal and vaginal flora.
- Lactobacillus is generally harmless to humans, rarely causes diseases.
- Because of their ability to derive lactic acid from glucose, they create an acidic environment which inhibits growth of many bacterial species in vagina which can lead to urogenital infections.
Bifidobacterium

- Anaerobic, Gram-positive bacilli
- Rarely associated with infection.
- The only pathogenic species is *Bifidobacterium dentium*, a normal inhabitant of the gut flora
- Under the microscope, these bacteria appear to be bone shaped,
- They require a very low oxygen tension to survive and to achieve moderate growth.
Eubacterium

- Eubacterium species are not clinically important. But, they may cause opportunistic infections because they are normal flora of the intestinal tract.

- *E. lentum*, the most often isolated species, may cause endocarditis and some wound infections.
Propionibacterium

• P. acnes, is a usually harmless microbe.
• It has been linked to certain cases of
  – endocarditis,
  – wound infections, and abscesses.
  – It can infect acne sites on the skin but it does not cause them.
• In Gram-stain film they show clump or branch and show uneven staining patterns.
• Colonies grow in anaerobic or microaerophilic environment using blood agar.
Peptostreptococcus

- Very small anaerobic bacteria that grow in chains.
- Usually non harmful, present in flora of skin, urethra and urogenital tract.
- Can cause infections of bones, joints and soft tissue.
- Their increasing resistance to antibiotics as penicillin G and clindamycin makes them especially important to clinical work.
MYCOBACTERIA
Mycobacteria are obligate aerobe, rods, non spore forming, acid fast bacteria. The cell wall contains high lipid content (40-60%), which is responsible for their staining and cultural characters. Mycobacteria are not classified as either Gram-positive or Gram-negative because they do not have the chemical characteristics of either, it stains very weakly Gram-positive or not at all.
Mycobacteria are classified into:

1. Members of the Mycobacterium tuberculosis complex (M. tuberculosis, M. bovis, M. africanum, M. microti) that cause tuberculosis in different hosts.

2. Non-tuberculous species (virtually all other species), which are classified according to their growth rate and pigmentation with and without exposure to light.

3. M. lepra, the causative agent of leprosy.
Mycobacterium Tuberculosis

- **General Characteristics**
  - It is a facultative intracellular parasite, usually of macrophages.
  - The high lipids content in the cell wall causes
    - impermeability to stains and dyes,
    - resistance to many antibiotics
    - resistance to acids and alkalis
Mycobacterium TB, morphology

- Small, straight or slightly curved, non motile, non sporulated, occur single, in pairs or in masses.
- Acid and alcohol fast (resist decolourization with acid and alcohol),
- Not stained by Gram
• **Cultural characters**
  
  – Obligate aerobe, 5 to 10% CO2, enhances growth
  – No growth on ordinary media.
  – Optimum temperature is 37 °C (strict mesophile).
  – No growth below 30 °C or above 39 °C.
  – Very slow grower, may require 4 to 6 weeks to get visual colonies.

Two media are used to grow M.TB

1. An egg based medium as Lowenstein-Jensen and Dorest egg media.
2. An agar based medium as Middlebrook's medium.
Tuberculosis

- Primarily affects the lower respiratory system
- Characterized by chronic productive cough, low-grade fever, night sweats, and weight loss.

**Pathogenesis**
- TB enter the alveoli by airborne transmission.
- They resist destruction by alveolar macrophages and multiply, forming the primary lesion or tubercle;
- Spread to regional lymph nodes, enter the circulation, and reseed the lungs.
- Tissue destruction results from cell-mediated hypersensitivity.

**Host Defenses**
- Acquired resistance is mediated by T lymphocytes,
- They lyse infected macrophages directly or activate them via soluble mediators (e.g., gamma interferon) to destroy intracellular bacilli;
- Antibodies play no protective role.
Pulmonary tuberculosis, diagnosis

1. Clinical specimens:
   - sputum (3-5 morning samples),
   - bronchial or gastric washings.

2. Direct smear:
   - Smear is prepared from sputum either direct or after liquefaction by N-acetyl-L-cysteine or by other methods.
   - Can be stained by carbol fuchsin (Ziehl-Neelsen) or fluorochrome stain.
   - By Ziehl-Neelsen (Z-N) stain, acid-fast bacilli appear pink in a contrasting blue background.
Pulmonary tuberculosis, diagnosis

2. Direct smear:
- By fluorochrome stain, the bacilli appear as bright yellow fluorescent against dark background.
- Direct smear is rapid, cheap and simple method for diagnosis but it is non sensitive, the sensitivity range is 25 to 75%.
- At least 5000-10,000 bacilli per ml of sputum are needed to detect positive microscopic smear.
- Other acid-fast bacilli are similar to M.TB in stained smear and should be ruled out by other methods.
- A positive stain and negative culture may be caused by non viable organisms, which can occur in persons receiving antituberculous medication.
• **3. Culture**
  
  The sample is treated with NaOH. This kills other contaminating bacteria (decontamination) but does not kill the M.TB. present because M.TB. is resistant to alkali.
  
  Sputum is inoculated in selective medium containing antimicrobial agents as (Lowenstein-Jensen) as it is contaminated with normal bacterial flora.
  
  Cultures are incubated at 35°C to 37°C in an atmosphere of 5 to 10% CO2.
  
  All cultures should be examined weekly for 4-8 weeks.
  
  Colonies on L-J medium are, raised, rough, confluent, grayish, and dry (eugenic growth).
  
  Culture is highly sensitive, specific and it allows visualization of colony morphology and pigmentation, which is useful for distinguishing colonies of M.TB from those of some nontuberculous mycobacteria.
Pulmonary tuberculosis, diagnosis

- **Rapid culture method (BACTEC System):**
  - The more rapid broth systems.
  - The media used in the BACTEC system are broth media containing radio-labeled palmitic acid as the sole carbon source.
  - As M.TB. multiplies, it utilizes the palmitic acid and release radio-labeled CO2. Using the BACTEC system, M.TB. growth can be detected in 9-16 days.
4-Intradermal skin test (Tuberculin test)

- It is a cell-mediated hypersensitivity skin test

- Based on that individual previously infected with tubercle bacilli will develop hypersensitivity to proteins of M.TB. which develops 6-8 weeks after infection.

- Intradermal injection of PPD (purified protein derivative) into a previously infected, hypersensitive person results in the delayed (48-72 hr) appearance of an indurated reaction.
4-Intradermal skin test (Tuberculin test)

- Skin testing is performed by using old tuberulin or PPD,
- injected either intradermal or by multipuncture.
- The Mantoux test is the standard tuberculin test,
- It requires the intradermal injection of (0.1 ml) containing (5 tuberculin units of PPD).
- The injection is made into the anterior aspect of the forearm.
4-Intradermal skin test (Tuberculin test)

- The transverse diameter of induration is measured after 48 to 72 hours.
- The test is considered positive if the diameter of the induration is 10 mm or greater. (erythema, swelling and induration).
- Positive reactions mean that an individual has been previously exposed to M.TB.
- Negative reaction, induration less than 5 mm. An area of surrounding erythema alone without induration, is ignored and considered negative.
- In negative individuals, the test may be repeated with higher doses of PPD e.g. 100 or 200 units
- It is impossible to distinguish between active disease and past infection by a positive tuberculin test.
- Recent conversion of the reaction from negative to positive needs clinical attention.
4-Intradermal skin test (Tuberculin test)

- False positive tests means positive reaction in absence of previous exposure to M.TB which may be due to prior exposure or infection with other mycobacteria or vaccination with BCG.

- False negatives means negative reaction in spite of previous exposure to M.TB which occurs in persons with impaired CMI response and other conditions such as late stages of tuberculosis malnutrition, steroid therapy.

- In areas where T.B. is endemis, most adult populations are tuberculin positive. For example, in Egypt up to 80% of adults give a positive Mantoux test due to previous sub clinical infection or vaccination. Therefore, the value of tuberculin reaction as a diagnostic test for active infection is restricted to children under 5 years of age, but in adults it is of value only if negative to exclude a suspected infection.
5. Recent methods for diagnosis

A. DNA probes and gas-liquid chromatography are rapid, specific and sensitive methods for identification of mycobacteria after sufficient growth is present on medium.

B. PCR: Is useful for direct detection of mycobacteria in clinical specimen within 24 hours or less. It is rapid, specific and sensitive.
Diagnosis of renal tuberculosis

- Sample: early morning urine of at least 3 successive days.
- Centrifugation of urine and preparation of smear from the deposit.
- Staining of the smear by Z-N stain,
  - should be decolourized by both acid and alcohol to differentiate M.TB from *M.smegmatis* which is found normally in the genitalia and which is acid fast but non alcohol fast.
- Culture of deposit after decontamination on L-J medium.
Diagnosis of tuberculous meningitis

- C.S.F is collected by lumbar puncture.
- C.S.F is less turbid than that in meningococcal meningitis and contains excess of lymphocytes.
- Direct film is prepared from the deposit after centrifugation.
- The deposit is cultured directly on L-J medium or Dorest egg medium without decontamination.
Mycobacterium bovis

- Similar to M.TB in morphology and culture characters.
- Differs in being shorter and thick, on L-J medium, gives poor growth as glycerol does not favour its growth but pyruvic acid favours its growth.
- It causes TB in cows and rarely in humans. Both cows and humans can serve as reservoirs.
- Humans can also be infected by the consumption of non pasteurized milk. This may lead to the development of extra pulmonary TB.
Treatment

• Tuberculosis is usually treated with four different antimicrobial agents.
• The course of drug therapy usually lasts from 6-9 months.
• The most commonly used drugs are
  – rifampin
  – isoniazid
  – pyrazinamide
  – ethambutol or streptomycin.
Prevention

- **BCG vaccine:**
- Living attenuated vaccine prepared from bovine strain (bacillus of Calmette and Guerin)
- Prepared by repeated subculture of bovine strain for 250 times on glycerol-potato-bile medium.
- Given by intradermal injection.
- Given to all infants in the first year of life and to tuberculin negative adults.
- BCG vaccinated person will be converted from negative tuberculin reactor to positive reactors (tuberculin conversion).
- Should not be given to tuberculin positive persons.
Nontuberculous Mycobacteria

- Previously "atypical" mycobacteria.
- Widely distributed in natural sources (air, soil, and water).
- Are non virulent to guinea pigs.
- Not transmitted from human to human.
- Are important opportunist in immunocompromised patients.
- Produce a wide range of clinical conditions as
  - pulmonary disease caused by *M. kanssasii* and *M. avium-intracellulare*,
  - disseminated infection in immunocompromised patients, particularly HIV-infected individuals, caused by the *M. avium-intracellulare* complex,
  - cervical lymphadenitis caused by *M. scrofulaceum*
  - granulomatous skin lesions and soft tissue infections caused by *M. marinum* (swimming pool granuloma) or *M. ulcerans*.
- Are resistant to the usual anti-tuberculosis drugs.
- No vaccine is available.
- Diagnosis requires culture and identification.
Nontuberculous Mycobacteria, classification

- Non tuberculous mycobacteria are classified by pigmentation in the light or dark and by growth rate into 4 groups (The Runyon groups).
- Species in groups I to III are slow growers further characterized by pigment production into:
  - Group I: Photochromogens are pigmented only when exposed to light.
  - Group II: Scotochromogens form pigment in the dark.
  - Group III: Nonchromogens are non pigmented.
  - Group IV: are rapid growers.
Mycobacterium leprae

- *M. lepra*, the causative agent of leprosy.
- Humans are the only natural host for *M. lepra*
- Structure *M. lepra* is similar to other mycobacteria

**Morphology:**
- Acid fast bacilli, found in large masses or single, straight or slightly curved.
- Stained by modified Ziehl-neelsen stain which is modified by using 5% H2SO4 for decolourization, as it is less acid fast than M.TB.
- It cannot be cultivated *in vitro* and it multiplies very slowly *in vivo.*
Mycobacterium leprae, clinical picture

Two clinical forms

1. lepromatous:
   - With granulomatous nodules in skin, peripheral nerves, mucous membranes and internal organs, with loss of specific cell-mediated immunity.

2. Tuberculoid
   - With skin and nerve lesions with intact cell-mediated immunity.

Diagnosis

- **Specimens**: nerve and skin lesions biopsies and nasal scrapings.
- **Film**: stained by modified Z-N stain to show acid fast bacilli in masses or single.
- **Culture**: *M leprae* cannot be cultured.
- **The lepramin skin test**: A heat-killed suspension of *M leprae* is injected into skin of the patient; it has little diagnostic value but will provide information of prognostic importance about the immune status of the individual.
- **The PCR technique**: By which very small amounts of *M leprae* DNA can be detected directly in clinical specimens, may be useful in diagnosis.
## Differences between M. tuberculosis & Non-tuberculous mycobacteria

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<th>Non tuberculous mycobacteria</th>
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<td>Acid fast</td>
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<td>Growth on L-J medium</td>
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<td>Temperature</td>
<td>Strict mesophile</td>
<td>Grow at 22,37 &amp; 45 °C</td>
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<td>Growth</td>
<td>Slow</td>
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<td>P-nitrobenzoic acid</td>
<td>Inhibit growth</td>
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