



**Slow growing mycobacterial species**

**Disease caused**

<u><i>M.kansaii</i></u>	Infection of previously damaged lungs or infection in HIV patients
<u><i>M.malmoense</i></u>	Infection of previously damaged lungs and cervical adenitis
<u><i>M.marinum</i></u>	Fish-tank <u>granuloma</u> and swimming pool <u>granuloma</u> Resistant to INH and PZA
<u><i>M.ulcerans</i></u>	Progressive ulcerating skin lesions
<u><i>M.gordona</i></u>	Resistant to INH and PZA

**Rapid growing mycobacterial species**

<u><i>M.fortuitum</i></u> ( <i>saprophytes</i> )	Soil organism that causes prosthetic joint abscesses, injection site abscess, and secondary pulmonary infection
<u><i>M.chelon</i></u> <i>ei</i> ( <i>saprophyte</i> )	Wound infections
<u><i>M.flavescens</i></u>	Rarely pathogenic
<u><i>M.smegmatis-phlei</i></u>	Found in urine and <u>smegma</u> but have no clinical significance



Notes



Comments

**Slow growing mycobacterial species**

**Species name**

**Genus**

**Species**

**Characteristics**

**Pathogenesis**

**Diagnosis**

**Treatment**

**Prevention**

**MYCOBACTERIUM LEPRAE**

*The organism causes leprosy (Hansen's disease)*

**MYCOBACTERIUM LEPRAE**

- M. leprae has not been grown in the laboratory, either on artificial media or in cell culture. It can be grown in experimental animals, such as mice and armadillos. Humans are the natural hosts.

**MYCOBACTERIUM LEPRAE**

- The optimal temperature for growth (30°C) is lower than body temperature; it therefore grows preferentially in the skin and superficial nerves. It grows very slowly, with a doubling time of 34 days. This makes it the slowest-growing human bacterial pathogen. One consequence of this is that antibiotic therapy must be continued for a long time, usually several years.

- Infection is a **slow** process; discharge of bacteria is **prolonged** because it is **slowly** cleared from the body.

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## Differences Between Primary and Secondary TB

<u>Primary tuberculosis</u>	<u>Secondary tuberculosis</u>
Lesion in lower and middle lung lobes (base of lung)	Lesion in apex of lung lobes
Involvement of regional lymph nodes with caseation	Less marked regional lymphadenopathy
Cavitation rare	Cavitation prominent
Immune reaction not prompt	Prompt immune reaction is mounted
Occurs in persons with normal immunity	Occurs in patients with waning immunity



Notes



Comments

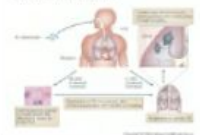
te of primary tuberculosis

fibrosis → calcification  
see primary tuberculosis  
• primary tuberculosis

Secondary tuberculosis

- Definition: the infection of an individual who has been previously infected or sensitized
- The infection may be acquired from
  - Endogenous source: reactivation of dormant primary complex
  - Exogenous source

Spread of Tuberculosis



- Primary lesions usually occur in the lower lobes, whereas reactivation lesions usually occur in the apices.
- Reactivation lesions also occur in other well-oxygenated sites such as the kidneys, brain, and bone.
- Reactivation is seen primarily in immune compromised or debilitated patients.

Differences Between Primary and Secondary Tuberculosis

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## Clinical Findings

- **Oropharyngeal tuberculosis** typically presents as a painless ulcer accompanied by local adenopathy.
- ✓ **Erythema nodosum**, characterized by tender nodules along the extensor surfaces of the tibia and ulna, is a manifestation of primary infection seen in patients who are controlling the infection with a potent cell-mediated response

nca



Notes



Comments

### Lesions Formed

Lesions of tuberculosis usually form in the lungs. The parenchymal lesions and the draining lymph nodes are called Ghon complex. Primary infection usually occurs in the lower lobes, reactivation lesion usually occurs in the upper lobes.

### Symptoms and Signs of Tuberculosis

- Most (approximately 90%) infections with *M. Tuberculosis* are asymptomatic. Asymptomatic infections, also known as latent infections, can reactivate and cause symptomatic tuberculosis.

### Symptoms and Signs of Tuberculosis



### Clinical Findings

- Pulmonary tuberculosis causes cough and hemoptysis.
- Scrofula is mycobacterial cervical lymphadenitis that presents as swollen, nontender lymph nodes, usually unilaterally. Both *M. tuberculosis* and *Mycobacterium scrofulaceum* cause scrofula. Lymphadenitis is the most common extrapulmonary manifestation of tuberculosis.

### Clinical Findings

- Oropharyngeal tuberculosis presents as a painless ulcer accompanied by adenopathy.
- Erythema nodosum, characterized by tender nodules along the extensor surfaces of the tibia and ulna, is a manifestation of primary infection seen in patients who are controlling the infection with a potent cell-mediated response.



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S E Q

## Lesions Formed

- The primary lesion of tuberculosis usually occurs in the lungs. The parenchymal exudative lesion and the draining lymph nodes together are called **Ghon complex**. Primary lesions usually occurs in the lower lobes, where as reactivation lesion usually occurs in the apices.



Notes



Comments

### Types of tuberculosis

**Primary tuberculosis** is a form of disease that is a previously unexposed and immunized person.

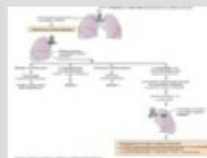
**Reactivation tuberculosis** is the pattern of fat areas in previously sensitized or host.

### Lesions Formed

- Lesions are dependent on the presence of the organism and the host response. There are two types of lesions.
- (1) **Exudative lesions**, which consist of an acute inflammatory response and occur chiefly in the lungs at the initial site of infection.
- (2) **Granuloma lesions**, which consist of a central area of giant cells (Langhans type) containing bacilli surrounded by a zone of epithelioid cells.

### Lesions Formed

- A **tubercle** is a granuloma surrounded by fibrous tissue that has undergone central caseation necrosis. Tubercles heal by fibrosis and calcification.
- A tubercle can erode into bronchus, empty its caseous contents and thereby spread the organism to other parts of the lung and other persons if expectorated.



### Lesions Form

- The primary lesion of tuberculosis occurs in the lungs. The parenchymal exudative lesion and the draining lymph nodes together are called **Ghon complex**. Primary lesions usually occurs in the lower lobes, where as reactivation lesions usually occurs in the apices.



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Question

## Types of tuberculosis

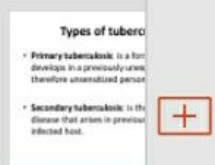
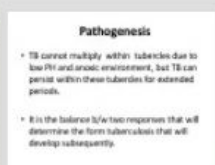
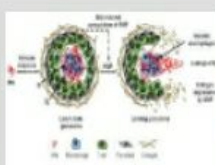
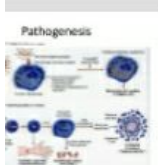
- **Primary tuberculosis:** is a form of disease that develops in a previously unexposed and therefore unsensitized person.
- **Secondary tuberculosis:** is the pattern of disease that arises in previously sensitized or infected host.



Notes



Comments



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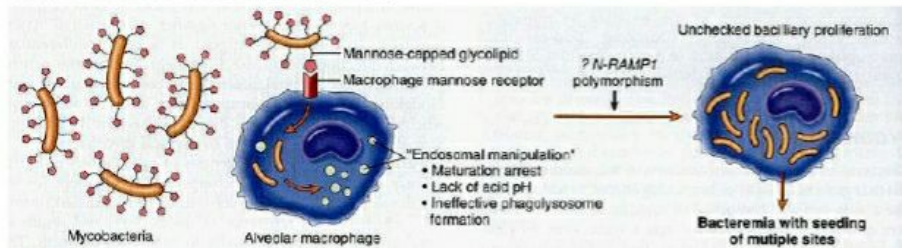
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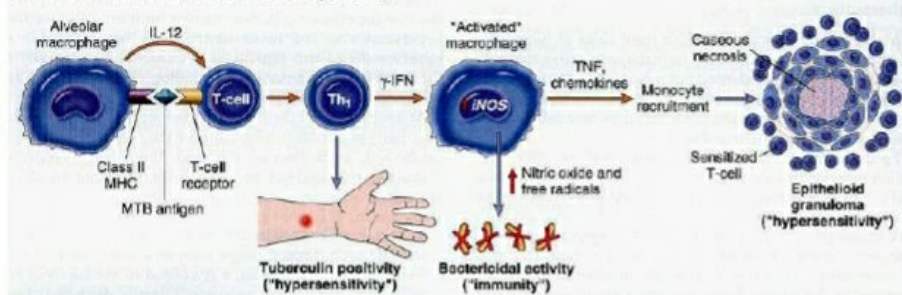
SEY  
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# Pathogenesis

A. PRIMARY PULMONARY TUBERCULOSIS (0-3 weeks)



B. PRIMARY PULMONARY TUBERCULOSIS (>3 weeks)



TB



Notes



Comments

**Pathogenesis**

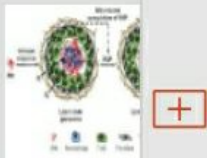
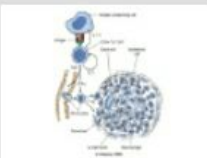
weeks after infection, host response results in caseous necrosis.

high self-healing TB response resulting in low of macrophages that are capable of digesting tubercle bacilli.

-alarming response is the result of a hyper hypersensitivity (1914).

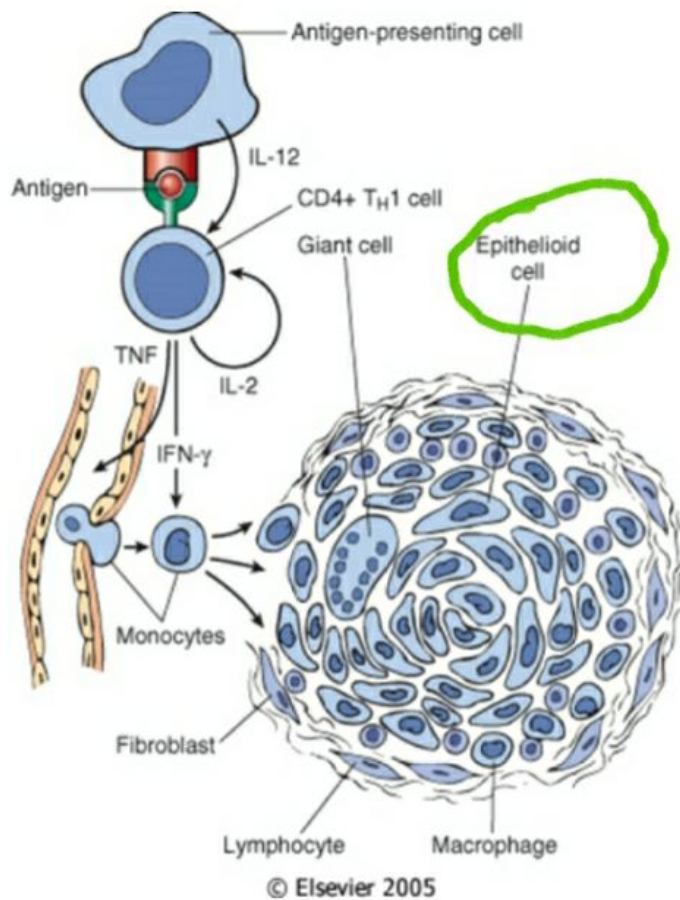
**Pathogenesis**

- For persons with intact cell-mediated immunity, the host defense may be formation of granulomas around the TB tuberculous organisms.
- These tubercle type bacilli form from an accumulation of activated T lymphocytes and macrophages, which creates a micro environment that limits replication and the spread of the Mycobacteria.
- This environment of macrophages and produces early solid necrosis at the center of the lesion. However, the bacilli are able to adapt to this.





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Notes



Comments

**Pathogenesis**

- Intracellular pathogen
- its phagosome lysosome fusion
- is lysosomal enzymes
- them produce a protein called
- “repertoire protein” that prevents
- enzyme from fusing with the
- phagosome

**Pathogenesis**

- T<sub>H</sub>1 multiplies within the macrophage and
- macrophage burst.
- Other macrophages diffuse from peripheral
- blood, phagocytose TB and are recruited, but
- are unable to destroy TB.
- These macrophages loaded with TB migrate
- through lymphatics to the hilar lymph nodes.
- These initial stages are asymptomatic and any
- specific defense mechanism is avoided.

**Pathogenesis**

- About 3-6 weeks after infection, low host response
- to TB, tuberculous meningitis
- Macrophage activation CD8 response resulting in
- the activation of macrophages that are capable of
- killing and digesting tubercle bacilli.
- The tissue damaging response is the result of a
- delayed type hypersensitivity (DTH).

**Pathogenesis**

- For persons with intact cell-mediated immunity, the
- best defense step is formation of granulomas
- around the TB tuberculous organisms.
- These nodular type lesions form from an
- accumulation of activated T lymphocytes and
- macrophages, which creates a micro-environment
- that limits replication and the spread of the
- mycobacteria.
- The environment destroys macrophages and
- produces early solid necrosis at the center of the
- lesion. However, the bacilli are able to adapt to
- survive.





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TB  
SEQ

## Pathogenesis

- **facultative intracellular pathogen**
  - inhibits phagosome-lysosome fusion
  - resists lysosomal enzymes
- The organism produces a protein called “exported repetitive protein” that prevents the phagosome from fusing with the lysosome.



Notes



Comments



### Antigenic Determinants

- **Cell wall:**
  - Act as virulence factors
  - Cause antibody production
  - Initiate tuberculous reaction
- **Subunit vaccines:**
  - Antigenic proteins & surface elements
  - Evolve immediate type of hypersensitivity

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### Other Virulence Mechanisms

- **Slow growth rate**
  - Structure contains cord factor (trehalose 6,6) or cannot be triggered to enter state TB
  - High lipid concentration in cell wall
  - Accounts for impermeability and resistance to antimicrobial agents
  - 8% of the cell wall membrane: pathogen and host unable to remove lipids very effectively
  - Inside the macrophage it can inhibit phagosome-lysosome fusion by secretion of a protein that modifies the phagosome membrane

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### Pathogenesis

- Disrupt nuclei are cultured, that are prevented by killing, smearing, and staining
- Once nuclei are released, the bacteria are non-specifically taken up by phagocytic macrophages
- The large disrupt nuclei reaches upper respiratory tract, and the small disrupt nuclei reaches air sacs of the lung (primary) where infection begins

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### Pathogenesis

- Facultative intracellular pathogen
- Inhibits phagosome-lysosome fusion
- Resists lysosomal enzymes
- The organism produces a protein “exported repetitive protein” that prevents the phagosome from fusing with lysosome.

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## MYCOBACTERIUM

- *M. tuberculosis* is resistant to dehydration and therefore survives in dried expectorated sputum.
- Humans are the natural reservoir of *M. tuberculosis*



Notes



Comments

### MYCOBACTERIUM

**"KOH-fast bacilli"** because of their cell walls, which are relatively able to resist basic dyes unless the combined with phenol.

ing is required for decolorization because of the high content of the cell wall lipids, arabinoside.

to the color granules and bacilli in the sputum and in the sputum, it is used for concentration and staining.

### MYCOBACTERIUM

- *M. tuberculosis* grows slowly (i.e., it has a doubling time of 18 hours, in contrast to most bacteria, which can double in number in 1 hour or less).
- The growth is so slow, cultures of clinical specimens must be held for 6 to 8 weeks before being recorded as negative.

### MYCOBACTERIUM

• *M. tuberculosis* can be cultured on bacteriologic media, where as *M. leprae* cannot. Media used for its growth contain complex nutrients and dyes (malachite green) known as Löwenstein-Jensen media. The dyes inhibit the unwanted normal flora present in sputum samples.

### MYCOBACTERIUM

• *M. tuberculosis* is relatively resistant to acids and alkalies. NaOH is used to concentrate clinical specimens, it destroys unwanted bacteria.

### MYCOBACTERIUM

• *M. tuberculosis* is resistant to dehydration and therefore survives in dried expectorated sputum.

• Humans are the natural reservoir of *M. tuberculosis*



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safe and hygienic.

- **Specimens should not be collected from bed pans.**
- Vomitus not advised.

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## Enrichment Medium

- Enrichment of the fecal specimens are done on Alkaline peptone water
- Venkataraman Ramakrishan Meidum is simple medium can be used as transport medium



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17

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## Selective Medium - TCBS

- V.cholrae grows well on Thiosulphate citrate bile sucrose (TCBS ) agar, on which it produces yellow colonies that are readily visible against the dark green background of the agar.



Dr.T.V.Rao MD

33



- Diarrhea occurs as much as 20 – 30 Liters/Day fluids are lost.
- Results in dehydration
- Shock
- Acidosis
- Can lead to death.
- About 60% of infections are caused with classic V.cholrae and are asymptomatic, about 75% of infections are caused by El Tor biotype



Dr.T.V.Rao MD

## Clinical features

- The incubation period is 1 – 4 days for person who develop symptoms, depends on the size of the inoculums ingested
- Manifest with  
Nausea , vomiting,  
profuse diarrhea, and abdominal cramps  
Rice water stool characteristic of cholera  
loss of fluid leads to profound dehydration  
Circulatory collapse and anuria.

Dr.T.V.Rao MD



## Clinical Findings

- Watery diarrhea in large volumes is the hallmark of cholera. There are no red blood cells or white blood cells in the stool. **Rice-water stool is the term often applied to the non-bloody effluent.** There is no abdominal pain, and subsequent symptoms are referable to the marked dehydration



colonization, is related to secretion of the bacterial enzyme mucinase, which dissolves the protective glycoprotein coating over the intestinal cells.

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## Pathogenesis

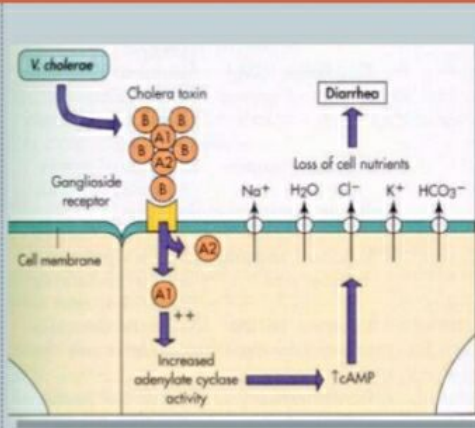
- After adhering, the organism multiplies and secretes an **enterotoxin called cholera toxin** ✓ (cholera toxin). This exotoxin can reproduce the symptoms of cholera even in the absence of the *Vibrio organisms*.

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Pathogenesis

### Vibrio Cholerae

- *Vibrio Cholerae* enterotoxin activates the stimulatory Gs protein via ADP-ribosylation. This stimulates secretion of chloride ions and water from enterocytes into the small intestines, and causing watery diarrhea.



The diagram illustrates the mechanism of cholera toxin. It shows a cholera toxin molecule (composed of B, A1, and A2 subunits) binding to a ganglioside receptor on the cell membrane. This binding leads to increased adenylate cyclase activity, which results in the production of cAMP (TcAMP). The cAMP then stimulates the secretion of Na<sup>+</sup>, H<sub>2</sub>O, Cl<sup>-</sup>, K<sup>+</sup>, and HCO<sub>3</sub><sup>-</sup> ions from the cell, leading to diarrhea. The loss of these cell nutrients is explicitly labeled as the cause of diarrhea.

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## Pathogenesis ✓

- *V. cholerae* is transmitted by **fecal contamination of water** and food, primarily from human sources. Human carriers are frequently asymptomatic and include individuals who are either in the incubation period or convalescing. The main animal reservoirs are marine shellfish, such as shrimp and oysters. Ingestion of these without adequate cooking can transmit the disease.

4

## Pathogenesis

- The pathogenesis of cholera is dependent on colonization of the small intestine by the organism and secretion of enterotoxin. For colonization to occur, large numbers of bacteria must be ingested because the organism is particularly sensitive to stomach acid. Persons with little or no stomach acid, such as those taking antacids or those who







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epidemic disease, whereas non-O1 organisms either cause sporadic disease or are nonpathogens.

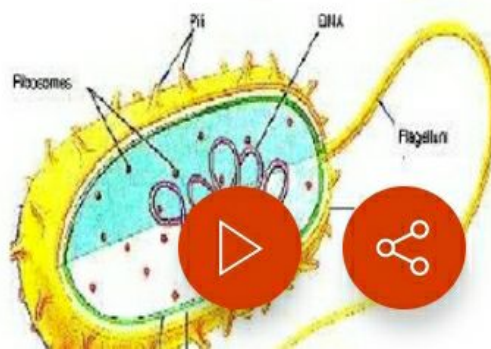
## Important Properties

- The O1 organisms have two biotypes, called El Tor and Classic, and three serotypes, called Ogawa, Inaba, and Hikojima. (Biotypes are based on differences in biochemical reactions, whereas serotypes are based on antigenic differences). Sero-group O139 organisms, which caused a major epidemic in 1992, are identified by their reaction to antisera to the O139 polysaccharide antigens

## V.cholrae - typing

- V.cholrae O1 has determinants that make possible further typing
- Serotypes are Ogawa, Inaba and Hikojima.

Epidemic V.cholerae is



Widal test

## Serological diagnosis

- *Salmonella* isolate can be identified and grouped by the slide agglutination test into serogroup A, B, C, D based on its O antigen.
- Diagnosis can be made serologically by detecting a rise in antibody titer in the paired patient's serum (Widal test).

MCO

- “Fish-eye” growth (lactose non-fermenter with sulfur reduction) on DCA agar



Thyphoid



mld

## Laboratory Diagnosis

### • Samples

- 1. Enterocolitis ----- stool
- 2. Typhoid fever----- first week ----- blood
- second week-----stool
- third week ----- urine
- chronic carriers---stool



Typhoid  
matters?

## How is it spread?

- Ingestion of food and water contaminated by human and animal wastes.
- *S. typhi*, the cause of typhoid fever, is transmitted only by humans, but all other species have a significant animal as well as human reservoir.
- The most frequent animal source is poultry and eggs, but meat products that are inadequately cooked are also the cause.

Viva  
Ques



SCIENCE

#### 4) Enterohemorrhagic *E. coli* (EHEC)

- Represented by a single strain (serotype O157:H7) produces shiga like toxin.
- Copious bloody discharge (hemorrhagic colitis), intense inflammatory response, may be complicated by hemolytic uremic syndrome.

S E Q  
M L Q

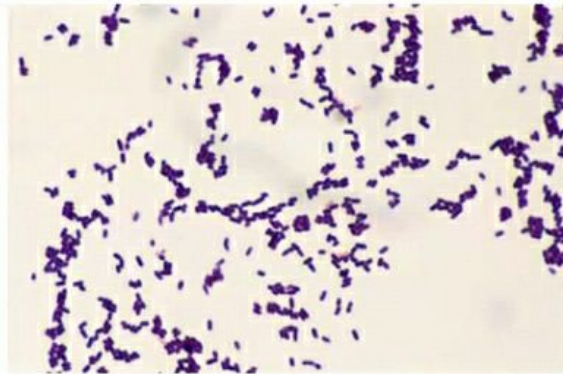
## Introduction

- *E. coli* is a most abundant facultative anaerobe in colon and feces.
- It is the most common cause of urinary tract infection.
- It is most important cause of neonatal meningitis.
- The agent most frequently associated with "travelers diarrhea"
- Some strains cause bloody diarrhea.



## Laboratory diagnosis

- **Gram stain:** Small **Gram-positive rods** arranged in V- or L-shaped forms similar to *Corynebacteria* & diphtheroids.



- **Motility Test:** **Tumbling** movement distinguishes it from *Corynebacteria* (non-motile).

mcp

## Pathogenesis

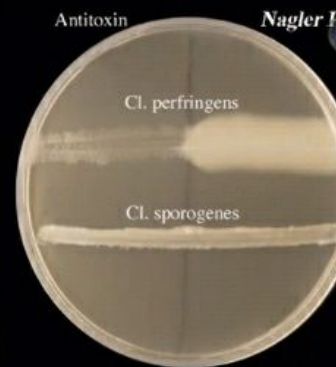
### Disease is exotoxin mediated

- Diphtheria toxin inhibits protein synthesis by **ADP-ribosylation of elongation factor 2. (EF-2)**.
- Toxin affects all eukaryotic cells.
- Toxin has two functional domains.
- First domain (B subunit) mediates binding of toxin to glycoprotein receptors on cell membrane.
- Second domain (A subunit) possesses enzymatic activity that cleaves nicotinamide from nicotinamide adenine dinucleotide (NAD) & transfers remaining ADP-ribose to EF-2, thereby inactivating it.

# OJPE

## Nagler's Reaction:

- Egg yolk agar used to demonstrate the presence of lecithinase.
- Nagler's reaction:
- *C. perfringens* produces an opacity in medium containing lecithin due to lecithinase C activity (alpha toxin).
- This opacity inhibited by applying specific antitoxic serum to medium which inactivates lecithinase.
- Technique referred as Nagler reaction

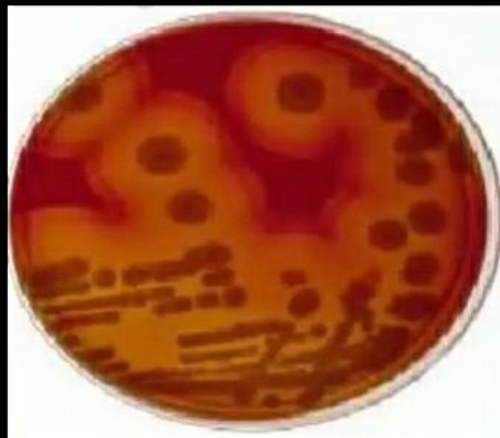




# OSPE

## Culture & Sensitivity:

- Blood agar: Large beta-haemolytic colonies produced (most food-poisoning strains are non-haemolytic).
- Some strains produce a double zone of haemolysis.



Dipe  
nca

## *Clostridium perfringens: cause*

Gas gangrene & Food poisoning

1. **Gas Gangrene: (myonecrosis, necrotizing fasciitis)**
  - Associated with war wounds, automobile, motorcycle accidents & septic abortions (endometritis).
2. **Food Poisoning:** Spores located in **soil**, contaminate **food**. The heat-resistant spores survive cooking & germinate.

viva



## Special forms

- **Wound botulism:**
- Spores contaminate wound, germinate & produce toxin at the site. Wound botulism associated with drug abuse (skin-popping with black tar heroin).
- **Infant botulism:**
- Organisms grow in gut & produce toxin there.
- Ingestion of honey containing organism implicated in transmission of infant botulism.
- Affected infants develop weakness or paralysis & need respiratory support.



From Ali Ihtashaam F17-136 to Everyone  
F17-136





Zoom

Leave

## Clinical findings

- Descending weakness & paralysis (flaccid paralysis), including diplopia, dysphagia, and respiratory muscle failure, are seen.
- No fever is present.
- In contrast, Guillain-Barré syndrome is an ascending paralysis

### Botulism Symptoms

If you have recently developed the following symptoms, go to the hospital now:

- Double Vision
- Difficulty Swallowing
- Blurred Vision
- Dry Mouth
- Droopy Eyelids
- Muscle Weakness (Starts in shoulders and descends through body)
- Slurred Speech



Unmute



Start Video



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Participants



More



## Clinical Findings

- ❑ **Cutaneous anthrax:** Painless ulcer with a black eschar (crust, scab), local edema (**malignant pustule**). Untreated cases progress to bacteremia and death.
- ❑ **Pulmonary anthrax** (wool sorter's disease): influenza like symptoms, dry cough & substernal pressure, progressing to hemorrhagic mediastinitis, bloody pleural effusions, septic shock & death.
- ❑ **Gastrointestinal anthrax:** Vomiting, abdominal pain & bloody diarrhea.



REC

## Pathogenesis

Bacillus anthracis has two exotoxins

### Edema factor

### lethal factor

- Each consist of two proteins having A–B subunits.
- B (binding subunit) in each of two exotoxins: **Protective antigen**: forms pores in human cell membrane, allows edema factor and lethal factor to enter cell.
- A (active subunit): enzymatic activity.

**Adenylate cyclase** (Increases intracellular concentration of cyclic AMP, causing outpouring of fluid from cell into extracellular space causing edema.

**Protease** (cleaves phosphokinase that activates MAPK signal transduction pathway, controls growth of human cells.

MCA

### Important Properties

- ▣ Large Gram-positive rod with square ends, frequently found in chains.
- ▣ Anti-phagocytic capsule (**D-glutamate**).
- ▣ Non-motile.



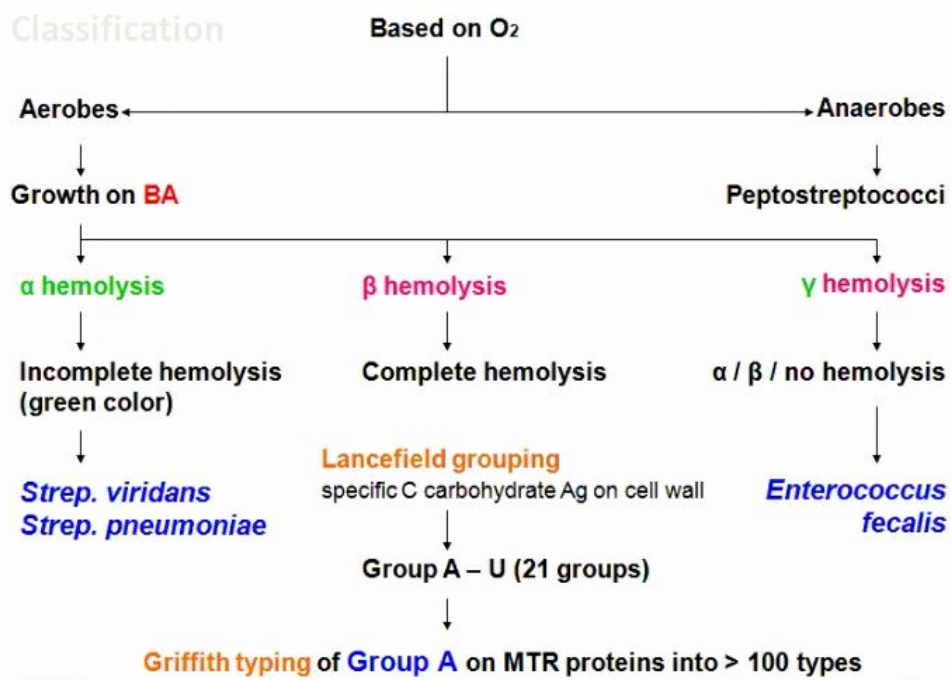
Other  
capsule is  
made up of  
peptide gly

# Biological Weapon

## *1. Bacillus anthracis cause Anthrax*

- Primarily a disease of herbivores (goat, sheep, cattle, horses).
- Humans infected incidentally by contact with infected animals, their products.
- **Bioterrorism In 2001**: outbreak of both inhalational & cutaneous anthrax in United States.
- Caused by sending spores of organism through mail.
- 18 cases, **5 died**.

MA



## Classification of Streptococci Based on Hemolysis on Blood Agar

- Hemolysis on BA
  - $\alpha$ -hemolysis
    - Partial hemolysis
    - Green discoloration around the colonies
    - e.g. non-groupable streptococci (*S. pneumoniae* & *S. viridans*)
  - $\beta$ -hemolysis
    - Complete hemolysis
    - Clear zone of hemolysis around the colonies
    - e.g. Group A & B (*S. pyogenes* & *S. agalactiae*)
  - $\gamma$ -hemolysis
    - No lysis
    - e.g. Group D (*Enterococcus spp*)





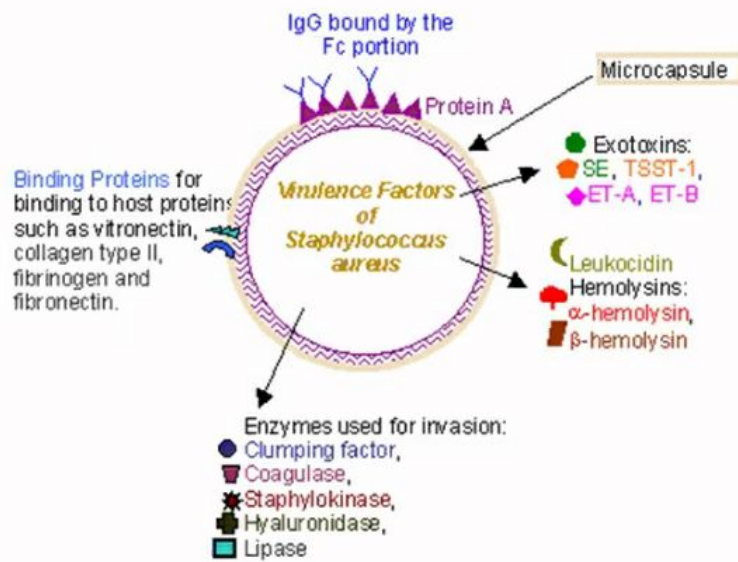
## Virulence Factors: Extracellular TOXINS

- Several important toxins are produced by *S. aureus*.
- The three clinically important are enterotoxin, toxic shock syndrome toxin and exfoliatin.

★ imp



## Virulence factors of *Staphylococcus aureus*



SEQ  
v.i.m.p

## Virulence factors:

These include

**A- Cell wall associated factors**

**B- Extracellular factors**

- Enzymes
- Toxins

V I M

STAPH LECTURE -2021 - Microsoft PowerPoint

Design Animations Slide Show Review View

Font Paragraph Drawing

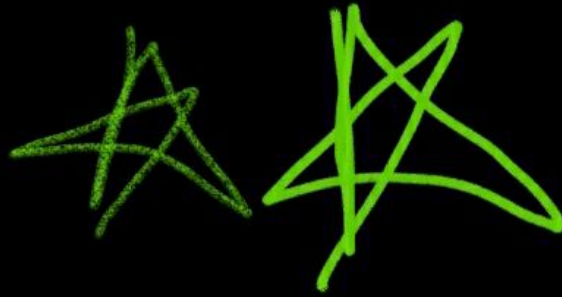
## CLASSIFICATION

- **1. Coagulase positive Staphylococci**
  - *Staphylococcus aureus*
- **2. Coagulase negative Staphylococci**
  - *Staphylococcus epidermidis*
  - *Staphylococcus saprophyticus*

to add notes

50%

✱

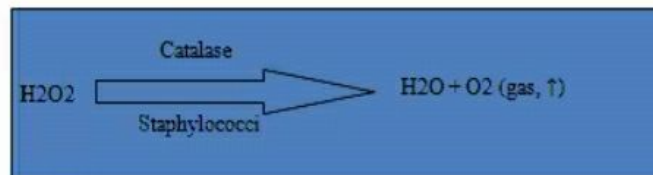


Design Animations Slide Show Review View

Font Paragraph Drawing Editing

## Catalase test

- The catalase test is use to distinguish Streptococci from Staphylococci.



to add notes



## Nomenclature of Cytokines

- Monokines – produced by mononuclear phagocytes.
- Lymphokines – produced by activated T cells, primarily helper T cell.
- Interleukins - cytokines made by one leukocyte and acting on other leukocytes.
- Chemokines- cytokines with chemotactic activities.
- Interferons - involved in antiviral responses.

# Ospe

Immunological Techniques - PowerPoint

SLIDE SHOW REVIEW VIEW



The image shows the Microsoft PowerPoint ribbon interface. The 'Paragraph' tab is active, displaying options for text alignment (left, center, right, justified), bullet points, numbering, and text direction. The 'Drawing' tab is also visible, showing options for shape fill, outline, and effects. The ribbon is partially obscured by the slide content below.

## ELISA

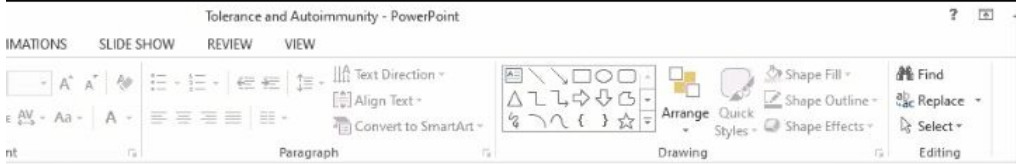
- Enzyme-Linked Immunosorbent Assay
- It is an enzymatic process which uses an enzyme to detect Ag-Ab reaction
- Advantages: specific, sensitive, rapid, inexpensive, safe, simple
- Enzymes: HRP, G6PD, Alkaline phosphatase,  $\beta$ -D-Galactosidase
- Substrates: DAB, TMB

NOTES COMMENTS



Zoom

Leave



# SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

- Chronic, multi-system inflammatory disease with protean manifestations and remitting course
- Clinical manifestations
  - Musculoskeletal (joint and muscle pain)
  - Dermatological (malar rash)
  - Renal (glomerulonephritis)
- Female to male ratio of 9:1
- Etiology is unknown
  - Genetics, race, hormones, environment



Figure 11-18 The Immune System, 2/e © Garland Science 2008

NOTES COMMENTS



HF From HassanAhmad F18-046 to Everyone F18-046

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