

Lecture # 1

Welcome 4th stage students

What do you planning for your future?

Do you want to apply for working in the diagnostic medical laboratory?

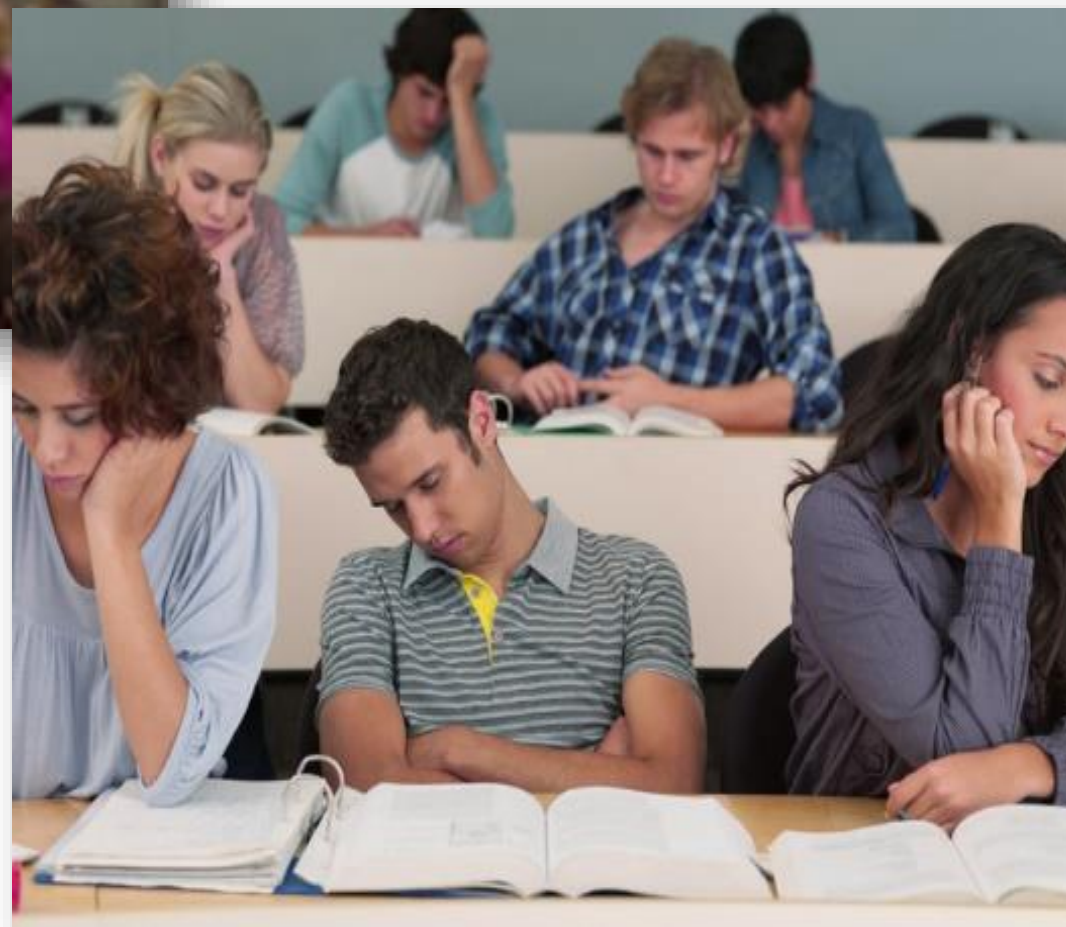
If Yes, You need course of

Medical Microbiology

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Do you like to be active learners or passive learners?



We want to make an agreement that insure active learning

What I want from you?

1- Regular attendance

2- Active participation

3- Ask ask ask ask questions to embarrass me

4- Criticize my teaching method and propose the best teaching method.

5- Respect the opinion of other

6- Respect each other because all of us belong to one species and the life requires cooperation

What do you want from me?

Remember that you will be graduated as soon as possible and take responsibility so be an active member of the society and be leader for other in order to build up a shiny future for the next generations.



Try to be the first or second or third or within 10th ranks in order to join higher studies like Master of science but remember that your success may be in other fields not your specialty like business (it is ur life ur option)

Bacterial pathogenesis

The style of this lecture is in the form of discussion because you should have information from 2nd year study through microbiology subject.

The style of the rest of lectures will be in the form of cases. Each case related to one pathogen and there will be a lot of questions about that pathogen, so you should study the subject thoroughly before answering the case. In each case, there will be general information that pave the way for diagnosis, so read carefully and then answer the question. If your diagnosis is wrong, other subsequent questions will be wrong.

Why bacterial pathogenesis is important?

What are the outcomes of understanding bacterial pathogenesis ?

1- I will know the source of infection whether from animals (zoonotic infections) or from environment or from human.

2- I will know the route/s of infection or transmission so I will protect myself or other from getting infection (prevention is better than treatment). Increase the public awareness to create healthy society.

3- I will know the mechanisms by which pathogenic bacteria produce infection.

Take two examples of Diarrhea caused by *Vibrio cholerae* and *Campylobacter jejuni*. The first causes disturbance of the function of gut mucosal cells without inflammation so watery diarrhea but without inflammatory cells (GSE=normal), while the second invade mucosal cells and causes inflammation(GSE= pus cells). So treatment will be rehydration for the first and antibiotics for the second.

4- I will know the complications of untreated infection and their dangerous on the life of infected individuals.

In typhoid fever if untreated may lead to intestinal perforation and death. Untreated cases of Gonorrhea in women may lead to infertility.

5- I will know whether the infection can becomes carrier and where in the body and their consequences on the society.

6- I will know how to collect appropriate samples for diagnosis

7- I will know the main clinical signs and symptoms

What are the types of relationship between host (me) and bacteria? Count or enumerate

What are infection and infestation? Or Define infection and infestation.- write on-----

Is any infection can lead to infection? Answer by yes or not then give explanation

What are the types of infections?

Types of relationship between host (me) and bacteria

A- Saprophytic (not requires living organisms) live on dead animals or in the environment (air, water and soil)

B- Parasitic relationship (on the cells =extracellular or in the cells (extracellular) or both=facultative intracellular

1- Commensals (mutualistic relationship) usually doesn't cause diseases e.g normal flora. These N.flora found in some body area in high quantities like colon, upper R.T, distal urogenital tract or in low number like esophagus , stomach (contaminated body area. whereas, absent in other body sites like blood circulation, CNS upper genitourinary tract and lungs (sterile body area)

2- Opportunistic (change in the natural niche like E.coli and UTI) or immunosuppression. Viridians Streptococci that cause subacute bacterial endocarditis (patients with prosthetic heart valves)

3- Pathogenic bacteria =able to cause infections and armed by virulence factors (should be killed)

A- Low pathogenic bacteria like Staph.aureus (BSL 1,2)

B- High pathogenic bacteria like T.B (BSL 3)

Stages of infectious process

1- Source of infection

Humans (carrier or case)

Animals (zoonotic diseases)

Environment (water, soil and air)

2- Modes of transmission

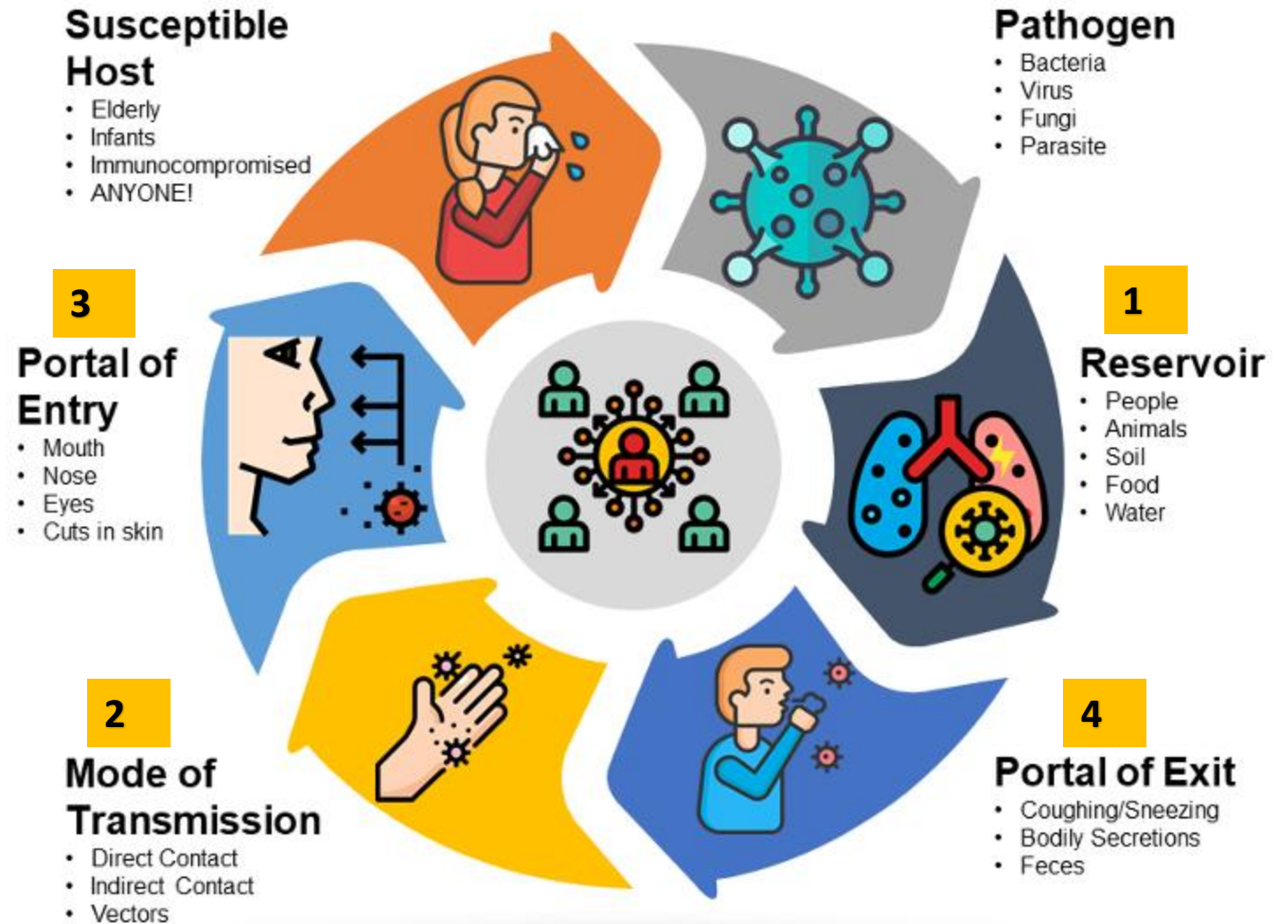
- Inhalation
- Ingestion
- Blood transfusion
- Sexual contact
- Injection
- bites

3- Portal of entry

- RT
- GIT
- Genital tract
- Injured skin
- Insect bites

4- Portal of exit usually by the same route of entry

Urine, stool, blood, genital discharges, respiratory, insects



Do you have any idea about Kochs postulates?

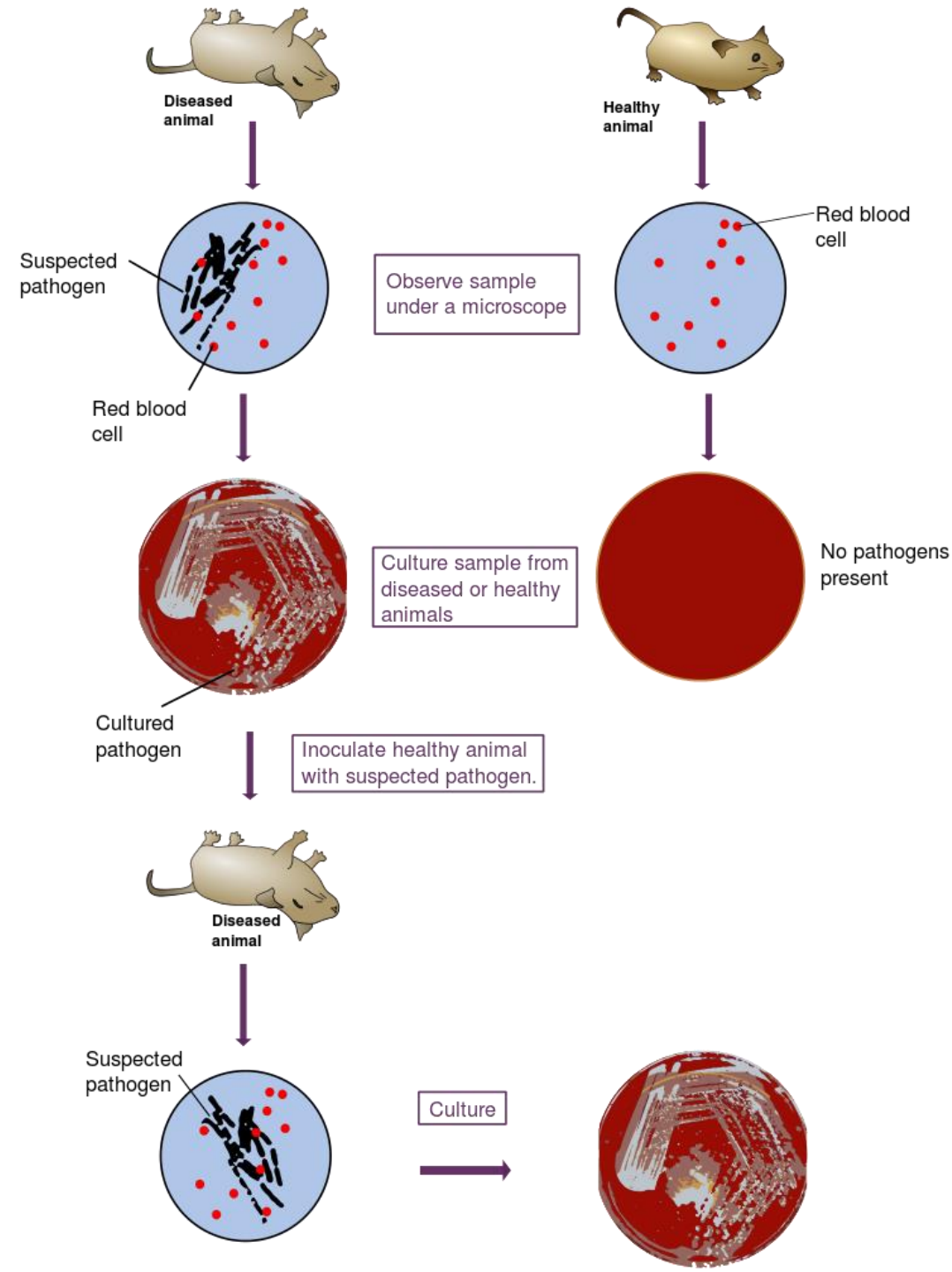
Koch's Postulates:

① The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.

② The microorganism must be isolated from a diseased organism and grown in pure culture.

③ The cultured microorganism should cause disease when introduced into a healthy organism.

④ The microorganism must be reisolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.



Do you know?

What are mechanisms by which pathogenic bacteria cause infection? i.e ()

What does mean the ability of pathogen to cause infection? ()

Degree of pathogenicity? i.e ()

() are the structures or products that enable the bacteria to cause infection

TABLE 10-6

Bacterial virulence factors

Organism	Virulence factors
<i>Staphylococcus aureus</i>	Coagulase, protein A
<i>Streptococcus pyogenes</i>	M protein
<i>Streptococcus pneumoniae</i>	Capsular polysaccharide
<i>Enterococcus faecalis</i>	Cytolysin, biofilm formation
<i>Neisseria gonorrhoeae</i>	Pili, opacity-associated proteins (Opa), IgA proteases
<i>Neisseria meningitidis</i>	Capsular polysaccharide
<i>Bacillus anthracis</i>	Capsule, edema factor, lethal factor, protective antigen
<i>Listeria monocytogenes</i>	Internalin
<i>Escherichia coli</i>	Heat-labile and heat-stable enterotoxins, pili
<i>Haemophilus influenzae</i>	Capsular polysaccharide
<i>Vibrio cholerae</i>	Cholera toxin
<i>Mycobacterium tuberculosis</i>	Mycolic acid cell wall

Adherence factors : enable bacteria to attach to the host surface like pili in E.coli in UTI and V.cholerae in GIT
-Glycocalyx of Viridans Streptococci attaches the endothelium of the heart valve to cause subacute endocarditis.
Glycocalyx of Strept.mutans to cause dental caries.

Invasion factors

1- Enzymes (degrades the host cells and tissues)

A- Spreading factors like collagenase, hyaluronidase, lecithinase all act spreading factors)

B- Leucocidin Kill phagocytic cells within fibrin coat

C- Deoxyribonuclease (Dnase) dissolve RNP (constitutes of pus and sticky prevent spreading of bacteria)

D- IgA protease inactivate mucosal IgA

E-Coagulase (fibrin clot)

F- Fibrinolysin (Staphylokinase)

2- Anti-phagocytic factors

A- Capsule

B- Cell wall M protein in Strep.pyogens

C- Protein A in Staph.aureus

D-Leukocidins

Differences between exotoxins and endotoxins

Exotoxins	Endotoxins
1. They are protein (polypeptide) and molecular weight 10,000 to 900,00.	1. They are Lipopolysaccharide in nature.
2. They are heat labile ($> 60^{\circ}\text{C}$) .	2. Heat stable
3. They are actively secreted by living cells into medium.	3. Form integral part of the cell wall and released only on disruption of bacterial cell.
4. Highly antigenic; stimulates formation of antitoxin that neutralizes toxin.	4. Weakly antigenic ; antitoxin is not formed but antibodies against polysaccharides are raised .
5. They are converted into toxoid by formaldehyde.	5. They can not be toxoided.
6. Enzymic in action.	6. No enzymic in action.
7. They have specific pharmacological effect for each toxin.	7. Non specific action of all endotoxins.
8. They have very high potency.	8. They have low potency.
9. They are highly specific for particular tissue e.g. tetanus toxin for central nervous system.	9. They are non specific in action
10. They do not produce fever in host.	10. They usually produce fever.
11. They are mainly produced by Gram positive bacteria and also by some Gram-negative bacteria.	11. They are produced by Gram-negative bacteria

12- Controlled by chromosomes, plasmids or phages

12- Controlled by bacterial chromosome only