Written Micro Questions PPPM

WRITTEN MICRO-QUESTIONS PPPM

Lectures 1 - 7

Compare prokaryotes & eukaryotes or animal cell or human cell.

	Prokaryotes	Eukaryotes
Membrane bound organelles	Absent	Present
Mitosis	Absent	Present
Nuclear membrane	Absent	Present
Chromosomal no.	One copy (Haploid)	Two copies (Diploid)
Histones	No histones	Histones with DNA
Ribosome	70S "50S large subunit + a 30S small subunit"	80S "40S small subunit + a 60S large subunit"
Peptidoglycan	Present	Absent

Mention 3 different shapes of bacterial cell

1) Cocci or spherical. e.g. Staphylococci
2) Bacilli or cylindrical e.g. Diphtheria bacilli.
3) Spiral:
   - One curve as vibrio
   - Multiple as spirochetes

Mention the components of bacterial envelope.

1) Capsule
2) Cell wall
3) Cytoplasmic membrane

Enumerate the secondary lymphoid organs & its functions.

- Trap & concentrate antigens.
- Lymphocytes developed in primary lymphoid organs reach the secondary lymphoid organs via blood & lymphatic vessels; where they can meet, & recognize antigen, and initiate the immune response.
- Include the lymph nodes , the spleen & MALT
- Mucosa-Associated Lymphoid Tissues include:
  a) Tonsils in the nasopharynx
  b) Peyer`s patches in the sub-mucosal surfaces of small intestine
  c) also act as secondary lymphoid aggregates.

Mention the different types of selection process & its site.

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- Occur in thymus gland
- Positive selection
- Cells that are able to recognize & not bind to self Ag are selected to grow.
- Negative selection
- Cells that can not recognize and efficiently bind self Ags are autoreactive cells & undergo apoptotic cell death.

Mention two different mechanisms to innate immunity

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- **Physical barriers:**
  Skin → epidermis (keratinocytes)
  Mucous membranes epithelium
- **Chemical & environmental barriers:**
  PH:
  Microcidal molecules: (α- & β- defensins)

About soluble defense mechansims of innate immunity, mention two.

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1) The complement system.
2) Cytokines:
   - Mannose-binding lectin (**MBL**).
   - C-reactive protein (**CRP**)

Mention 2 characters of adaptive immunity

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1) **Specificity**:
   The immune response is specific for a certain antigen Specificity ensures that different antigens elicit specific responses.
2) **Memory:**
   The ability to "remember" & respond more strongly to repeated exposure to the same microbe.

Compare between innate immunity and adaptive immunity

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	Innate immunity	Adaptive immunity
Protection	1st line of defense Immediate (hours)	2nd line of defense Delayed (days)
Specificity	Non-specific	Specific
Memory	None	Yes
Non-reactivity to self	Yes	Yes
Receptors	Pattern recognition receptors	↑ B cell receptor ; BCR ↑ T cell receptor ; TCR
Cells	1. Phagocytes: macrophages & neutrophils 2. NK cells	1. B lymphocytes 2. T lymphocytes
Complement activation	Alternative & MBL pathways	Classical pathway
Cytokines	↑ IL-1, IL-12, ↑ IFN-a, IFN-ß, IFN-γ, ↑ TNF & chemokines	IL-2, IL-4, IL-5, IL-13, IL-17, IFN- γ

Mention the steps for T dependent activation of B cells

1) BCRs bind antigens.
2) Ag are endocytosed.
3) The antigen is digested into fragments
4) Ag are displayed at the cell surface inside a class II MHC to be presented to Th cells
5) Th1 cells bind the B cell & secrete IL4 that:
   a) Stimulate the B cell to proliferate into a clone of cells with identical BCRs.
   b) Differentiate into plasma cells that secrete antibodies.

Mention different subsets of T lymphocytes

a) Helper T cells (CD 4 ++) TH
b) Cytotoxic T cells (CD 8 +) Tc
c) Regulatory T cells (Treg)

About natural killer cells
Mention its functions, receptors.

1) Killing tumor cells.
2) Killing virus-infected cells.
3) Antibody-dependent cellular cytotoxicity (ADCC)
   ☐ Ab coat target cells then NKs kill them.
3) Produce IFN-γ which activate macrophages.

Antigen receptor (KARs & KIRs)

Enumerate steps of phagocytosis

Steps of phagocytosis:

1. Delivery of phagocytes to site of infection:
   - Diapedisis
   - Chemotaxis
2. Recognition of microbes: phagocytes recognize microbes in blood and tissues
   - by surface receptors specific for microbes e.g. Toll-like receptors "TLRs"
3. Phagocytic adherence to microbes
   - Opsonization : coating of microbe by opsonin {antibody or complement C3b, C4b}
   - Binding of microbe to receptors on phagocytic cells
4. Ingestion (engulfment) of microbe into cytoplasm of the cell within a membrane vesicle → phagosome
5. Fusion of phagosome with lysosome of cell → Phagolysosome (digestive vesicle)
6. Intracellular killing:

Oxygen independent	Oxygen dependent
1) lysosomal granules, 2) lactoferrin, 3) low pH 4) lysosomal enzymes	1) toxic oxygen derived products 2) toxic nitrogen oxides

Mention the action & function of antigen presenting cells	▪ **Action:** 1) Capture, transport, process protein antigens into peptides and present the peptides to T cells. 2) they are rich in class II MHC molecules **Function:** 1. Capture & transport antigens to peripheral lymphoid tissues 2. Process antigens into pieces (peptides) 3. Present peptides to T lymphocytes → adaptive immunity
Mention types & 2 function of stem cells	1) Embryonic stem cells: Adult stem cells:. 2) Leukemia: Bone marrow transplants 3) Cardiovascular disease: repairing damaged heart cells after a heart attack
Define haptens & its role in immunity.	▪ A small Ag with epitopes capable of binding with immune receptor ▪ Without inducing immune response ▪ BUT can produce immune response only when conjugated with large carrier molecule (as a protein) → immune response against epitopes of hapten & carrier.
Enumerate factors affecting & influence immunogenicity	1) **Size** : Proteins > 10 KDs are more immunogenic 2) **Complexity**: Complex proteins with numerous, diverse epitopes are more to induce an immune response than are simple peptides that contain only one or few epitopes. 3) **Conformation & accessibility :** Epitopes must be "seen by" & be accessibile to the immune system. 4) **Chemical properties**: a. A protein is good immunogens. b. Many charbohydrates, steroids, & lipids are poor immunogens. c. Amino acids and haptens are, by themselves, not immunogenic.

Mention forms of antibodies	1) Secreted Ig 2) Membrane bound Ig in plasma & mucosa & interstitial fluids of tissues Expressed on B cell surface (IgM & IgD) as BCR for Ag " If bind with Ag, initiate B cell response"
Enumerate regions of antibodies acc to proteolytic activity	Hinge region Fc " fragment crystalline" Fab " fragment Ag Binding" وظف مم مم مم جدا
Define monoclonal antibodies , polyclonal	▪ Identical monospecific antibodies that are produced by one type of immune cell that are all clones of a single parent cell. ▪ In contrast, antibodies obtained from the blood of an immunized host are called polyclonal antibodies.
Mention 2 applications for monoclonal antibodies	1) **Identification of phenotypic markers** : They have been used to define clusters of differentiation (CD markers) on lymphocytes. 2) **Immunodiagnosis**: The diagnosis of many infectious & systemic diseases relies on the detection of specific antigens or antibodies in the circulation or tissues by use of mAbs.
How antibodies adapt its function in ADCC	**IgG** Bind to infected cells by the Fab regions & bind by Fc to Fc receptors on NK cells. The NK cells are activated and kill the cells. **IgE** Bind to helminthic parasites by the Fab regions, and bind by Fc to Fc receptors on eosinophils The eosinophils are activated to release their granule contents, which kill the parasites.

Mention the function of antibody in mucosal immunity

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1) IgA is the major class that is produced by the (MALT) in the GIT & RT and transported to the lumens of organs.
2) In mucosal secretions, IgA binds to microbes & toxins present in the lumen & neutralize them by blocking their entry.

Compare between primary & secondary immune response

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	Primary response	Secondary response
Def	When we are exposed to an antigen for the first time	If at a later date we are re-exposed to the same antigen
Onset	There is a lag of several days (10 days) before specific antibody becomes detectable. "slow onset"	More rapid appearance of antibody,
Antibody	IgM.	IgG (Or IgA, or IgE)
Duration	After a short time, the antibody level declines." short lived "	Remains detectable for months or years. "long lived"

Mention the 2 signals for activation of T Cells

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1) The 1st signal : Peptide + MHC on the surface of APCs recognized by TCR- CD3.
2) The 2nd co-stimulatory signal :
   - The interaction of B7 molecule on APCs with CD28 on T cells.
   - In absence of 2nd signal, exposure of T cells to antigen → anergy (unresponsiveness).

Mention the role of granules in killing by T cytotoxic cells

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1. Perforin, which form pores in the target cell membrane.
2. Granzymes enter the target cells through these pores & induce apoptosis through the activation of caspases

Compare between class I,II MHC

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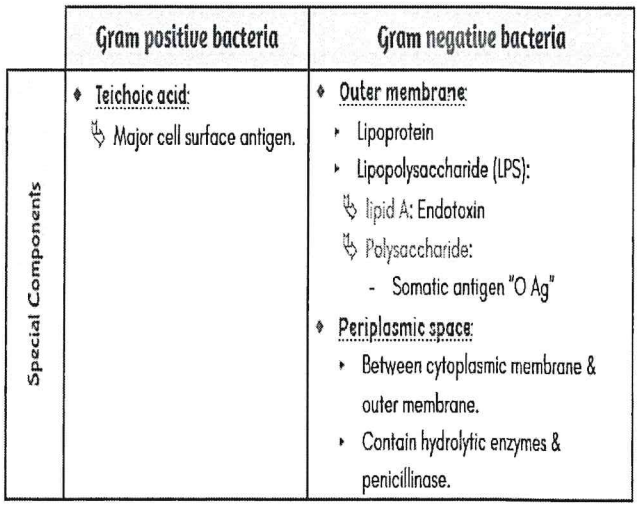
Class I MHC molecules	Class II MHC molecules
2 polypeptide chains, a chain formed of 3 domains (α 1, α 2, α 3), attached to a polypeptide chain called ß2 microglobulin encoded by a gene outside MHC.	2 polypeptide chains α chain (α 1 & α 2) and ß chain (ß1 & ß2).
α 1 & α 2 domains form the cleft or groove which bind peptide.	α 1 and ß1 domains form the peptide binding cleft.
Present antigen to **CD8+** cells.	Present antigen to **CD4+** cells.
Class I molecules are expressed on **all nucleated cells.**	Class II is expressed on **APCs only.**

Mention steps of complement activation pathways	ال 3 اى ﷺ من هم تنيجى
Enumerate 3 functions of complement system	1) Opsonization & phagocytosis 2) Complement-mediated lysis 3) Stimulation of inflammatory reactions
Enumerate 2 mediators , regulators of innate immunity , adaptive immunity.	▪ **Innate:** 1) TNF-a Activation of neutrophils & inflammation. 2) IL-1 Activation of neutrophils & inflammation. ..... ▪ **Adaptive:** 1) IL-2 Proliferation of T, NK, and B cells. 2) IL-4 B cell isotype switch to IgE and mast cell proliferation.
Define adjuvants & its mechanism	**Def** It is an agent that stimulates the immune system & ↑ the response to a vaccine, without having any specific antigenic effect by itself. **Mechanisms of action** 1) Prolong antigen retention. 2) Increase antigen size, so promote phagocytosis & presentation by macrophages. 3) Increase local immune cell influx (i.e., influx of macrophages & other immune cells to injection site). 4) Increase local cytokine production.

About immunosuppression, how induction occur, indications.	**Induced by** : Drugs - Radiation. **Indications** 1. Prevention of graft (transplant) rejection. 2. Treatment of hypersensitivity. 3. Treatment of autoimmune diseases.
Mention the mechanism of cyclosporine , azathioprine induce immunosuppression.	1) Inhibit T cell activation by blocking T cell cytokine production (IL-2) 2) The most commonly used immunosuppressive drugs for prevention of graft rejection. ..... 1) They are antiproliferative drugs, inhibit synthesis of purines that are required for cell division. 2) They inhibit lymphocyte proliferation

Lecture 8

Classify bacteria according to habitat	1) Parasitic (need host): bacterial flora (commensally) in soil, air and water 2) Saprophytic (free living): pathogenic bacteria
List structures present in bacterial envelope ?	1) Cell wall. 2) Cytoplasmic membrane 3) Capsule or slime layer.
List the differences in special components in cell wall of gram +ve , -ve	
What is the function of periplasmic space in gram -ve bacteria	▪ It is the space between cytoplasmic membrane and outer membrane ▪ contains hydrolytic enzymes and penicillinase

What is the importance of bacterial cell wall **Enumerate 3 functions of bacterial cell wall**	1) Antigenic character: in gram +ve , -ve .... 2) Cell wall is the target for the action of some Antibiotics 3) Preservation of the shape of the cell. 4) Cell wall is responsible for Gram staining reaction 5) Plays an essential role in cell division 6) Permeability of the cell , Non selectively permeable 7) Protective against high internal osmotic pressure 8) Toxicity
What are the antigenic characters of gram +ve , -ve bacterial cell wall	▪ In Gram positive: Teichoic acid. ▪ In Gram negative: somatic "O" antigen (Polysaccharide).
List the sites & function of lysozyme in resistance to bacteria	- Enzymes that attack cell walls: - Peptidoglycan is hydrolyzed by lysozyme - found in tears, saliva & nasal secretions
What are the functions of bacterial cell membrane ?	1. Electron transport & oxidative phosphorylation: for energy production (ATP). 2. Chemotactic function: contain receptors of binding and repellents 3. Excretion of hydrolytic enzymes 4. Permeability &transport: ☐ Transport nutrients into and waste products out of the cell. 5. Biosynthetic function: ☐ Carries enzymes & molecules for biosynthesis of cell wall, DNA & memb. Lipids

What are the functions of bacterial capsule	1) Virulence factor: as it protects bacterial cell from phagocytosis. 2) Protects cell wall: against bacteriophage, complement & lysozymes 3) - Antigenic (K-antigen): used in serodiagnosis or vaccine preparation
Enumerate 4 types of arrangement of flagellae	a) Monotrichous: single polar flagellum. b) Lophotrichous: multiple polar flagellae. c) Amphitrichous: One flagellum in each pole of the cell. d) Peritrichous: flagella distributed over the entire cell. e.g. E.coli.
why motility is important function to bacteria ?	1. Movement to nutrients. 2. Movement to optimal oxygen concentration in aerobic bacteria. 3. Choosing site for colonization. 4. penetration through a viscid mucous secretion.
What are the differences between flagellae and pili	1. Occur in motile as well as non-motile strains but flagellae in motile organisms 2. More numerous "100-500" per cell. 3. Much shorter and thinner but flagellae is longer and thicker 4. more or less straight, flagellae are spiral. 5. Originate from cytoplasmic membrane Not originated from cytoplasm like flagellae.
List the different antigenic parts types in bacteria	1- Flagellae (H-antigens). 2- Capsule K-antigen 3- In gram negative bacteria Polysaccharide somatic Ag (O Ag)

Lecture 9	
Define Bacterial endospores (give examples, function)	• Some bacteria are capable of forming resistant endospores under certain unfavorable in vitro (outside the body) conditions as: starvation, desiccation, heat and chemical agents. • **Examples:** The genus Clostridium (anaerobic) and genus Bacillus (aerobic) • **Function:** It is a resting cell, highly resistant to desiccation, heat and chemical agents.
What is the structure of bacterial endospore	1) Bacterial DNA 2) Small amount of cytoplasm & Very little water 3) Cell membrane 4) Peptidoglycan 5) Thick keratin coat with + Ca
What is meant by germination of bacteria	• The spore has no metabolic activity and can remain dormant for many years. • Upon exposure to water and appropriate nutrients: • specific enzymes degrade the coat, water and nutrients enter, and germination into a metabolizing, reproducing bacterial cell occurs
What are the steps for bacterial sporulation ?	1. The nuclear material in the cell moves to one pole. 2. Cytoplasmic membrane: invaginates to form the forespore engulfs forespore within a second membrane. 3. Cortex formation by deposition of Dipicolinic acid + ca. 4. Formation of spore coat. 5. Release of endospores. 6. Rest of bacterial cells undergoes autolysis.

Enumerate 3 basic elements for bacterial growth	• Major : Carbon & Nitrogen • Minor : Sulphur, Phosphorous , Ca, K & Mg
What are the bacteria that need high concentration of Co2	• Neisseria & Brucella abortus stimulation of growth (capnophilic) • ☐ Bacillus anthrax & Pasteurella.formation of capsule • ☐ Staphylococcus aureus enterotoxin production

What is meant by the following & give example

Capnophilic	organisms require higher concentrations of CO2 (5-10%) to be provided in the culture media	**Neisseria & Brucella abortus**
Obligatory aerobes	can grow only in presence of free O2.	**T.B**
Facultative anaerobes	can grow well in presence or absence of O2.	**Most pathogenic bacteria**
Obligatory anaerobes	✓ Grow in absence of O2 and cannot grow in the presence of oxygen, due to lack of peroxidase enzyme or catalase enzyme so in presence of O2, peroxides will be formed which is toxic to the organism.	**Clostridium Group**
Microaerophilic	grow best in presence of a minimal amount of O2.	**Propionibacterium acne**
Autotrophic	assimilate inorganic chemicals (CO2) as the only source of carbon.	**saprophytic bacteria.**
Heterotrophic	Require organic sources of carbon	**Pathogenic bacteria**

List examples of bacterial enzymes	1) Proteolytic enzymes → act on protein 2) Saccharolytic enzymes → act on CHO 3) Lipolytic enzymes → act on lipids 4) Respiratory enzymes → dehydrogenases and oxidases

what are the characters of endopigment, exopigments & give examples

	Endopigment	Exopigment
Character	▸ Remains bound to the body of the organism and do not diffuse into the surrounding medium. ▸ They are demonstrated on a solid media.	▸ Diffuses into the surrounding medium.
Bacterial colony	Colored	Colored
Surrounding medium	Not Colored	Colored
Function	▸ **Play a role in:** 1) Bacterial respiration & 2) Have antibacterial action.	
Examples	▸ **Red pigment:** Serratia marcescens. ▸ Golden yellow: S. aureus.	▸ **Pseudomonas aeruginosa** produces: ↳ **Blue pigment** called pyocyanin. ↳ Yellow pigment called fluorescens.

Compare between Exotoxin & Endotoxin

	Exotoxins	Endotoxins
Def	These are diffusible toxins, which diffuse into the surrounding medium.	Remain bound to the body of the organism & released only when the organism disintegrates.
Action	On specific organ according to its types	On Interlukin 1 & Tumor necrosis factor (TNF)
Antigenicity & toxicity	Highly	Less
Gene code it	On plasmid or transferred by bacteriophage	On chromosome
Diffusibility	Diffusible outside bacteria	Non diffusible
Preparation	▸ Growing the organism in fluid media. ▸ Then filtrated through a bacterial filter. ▸ The filtrate contain the toxin.	Disintegration of the organism
Source	G + ve mainly & G-ve	G - ve
Structure	Protein	Lipopolysaccharides
Specificity	Specific in action	Non specific
Effect of heat (60-80C)	Thermolabile Destroyed	Thermostable
Effect of 0.3%formaline	Detoxicated	Not detoxicated

What is net result of bacterial growth in fluid media

- uniform turbidity: facultative anaerobic bacteria
- surface pellicle aerobic bacteria
- sediment leaving relatively clear medium anaerobic bacteria

what is the cause of varying duration of lag phase

1. type of organism: 1 hr in Ecoli , few days in TB
2. Type of media: more suitable → more shorter
3. Size of inoculum: bigger → shorter lag phase
4. Stage from which the bacteria is taken: if log phase → lag phase is shorter

What is the cause of appearance of bacterial death in stationary phase	1. Exhaustion of nutrients. 2. O2 Starvation. 3. Accumulation of toxic materials
Enumerate the phases of growth curve & their correlation clinically in vivo	1) The lag phase :Incubation period in vivo. 2) The logarithmic phase (exponential) : invasive period 3) The stationary phase : symptoms and signs in vivo. 4) The decline phase : convalescent period in vivo.

Lecture 10 - 11

What are the most famous extrachromosomal elements

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- Plasmids
- Transposons
- Bacteriophage (Virus infecting bacteria)

Define plasmid

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Piece of DNA that exist separated from the chromosome, containing origin of replication, so independently replicate from the chromosome

Classify plasmid according to shape

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	Covalently closed circular (CCC)	Semicircular	Linear
Incidence	✓ Most common	✓ Transient form	✓ In some bacteria
Description	✓ Double strand, ✓ completely closed circular forms ✓ as E.coli	✓ One strand, ✓ completely Closed & the other is opened	✓ Double strand, ✓ Unstable as it's attacked by exonucleases

Classifiy plasmids according to (copy number, Compatibility of plasmids)

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copy number	Compatibility of plasmids
Stringent plasmids: 1- 2 copies/cell E.g.: F-plasmid & phage plasmid hybrid (P2) ◆ **Low copy number plasmids:** 10 - 15 copies/cell E.g.: pSC 101 ◆ **High copy number plasmids:** up to 50 copies/cell E.g.: Col E plasmid ❖ **Extremely high copy number plasmids:** up to 100 - 200 copies/cell	Compatible plasmids ✓ cell can maintain more than one plasmid in the same cell ✓ If they carry different origins of replication Incompatible plasmids ✓ inability of two plasmids with the same origin of replication to be maintained in the same cell ✓ If they carry same origins of replication

Define: ((you must define general def of plasmid firstly)) ☺

Stringent plasmids:

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Shuttle vector

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- **Stringent plasmids:**

1- 2 copies/cell E.g.: F-plasmid & phage plasmid hybrid (P2)

- **Shuttle vector:**

Can mobilized itself & another plasmid from cell to cell

May be natural or artificial plasmid

Compare between natural & artificial plasmid	Natural plasmids: ▪ All the above plasmids are present naturally in bacterial & some yeast cells. ▪ like F-plasmid (F-pili during conjugation) Artificial plasmids: ▪ They are naturally present plasmid but designed artificially by adding antibiotic resistant markers or DNA sequence to be target of restriction endouncleases. ▪ Used in genetic engineering as vectors
Define plasmid and mention 2 beneficil uses for bacteria	**Def**: as before **Functions** : 1) Resistance: Antibiotic resistance ,, Heavy metal → metal reductase ,,UV → DNA repair enzymes 2) Conjugation. 3) Production: Toxins, enzymes & bacteriocin 4) Biochemical Reactions: sugar fermentation.
How plasmid act as vector ?	• It can be used in Molecular biology (as a vector): Cloning vectors. • Gene therapy: Plasmids used for insertion of therapeutic genes at pre-selected chromosomal target sites.
Define transposons and mention the simplest structure	▪ Def: Extra-chromosomal small pieces of DNA that are capable of moving itself from one location in DNA to another (Movable elements or Jumping genes) ▪ They can transpose into any DNA molecules Except themselves ▪ Insertion sequence is the simplest form ▪ Their ends have the same repeats either direct or inverted repeats (15 - 25bp) ▪ Encode only ptn needed for own transpositions

Compare between composite Tn and non composite Tn & give examples for its functions

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COMPOSITE	NON COMPOSITE
2 IS at both ends ✓ Central piece encode for: • Antibiotic resistance • Virulence factor	No IS at their ends but encode for transposition protein. ✓ Central piece encode for: • Antibiotic resistance • Virulence factor • Catabolic enzymes
Tn 5: for kanamycin resistance ✓ Tn 10: for tetracycline resistance	Tn 3: carry ampicillin resistance gene ✓ Tn 7: carry streptomycin & trimethoprim resistance gene

Enumerate types of gene transfer

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1) Vertical gene transfer Transfer of genetic material from parental organism to progeny Include Conjugation. Transduction Transformation

2) Horizontal (lateral) gene transfer organism transfers genetic material to another organism that is not its offspring.

Define conjugation and what are the requirements for it to occur

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Definition:

It is a form of gene transfer in which two cells come in direct contact and DNA is transferred from one cell (donor) to the other (recipient).

Requirements:

1) Donor cell should contain F-plasmid, which encodes for F pili needed for conjugation.
2) Donor plasmid should contain tra gene (conjugative type) to mobilize the plasmid.
3) Conjugation most frequently occurs in Gram negative bacilli but Gram-positive organisms can make conjugation.

How conjugation occur in gram positive bacteria

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Mediated by signaling molecules called pheromones.

List the steps of conjugation.	▪ Donor Gram negative bacilli containing F plasmid come in contact with other F- Gram negative bacilli. ▪ The F pili of the donor cell (F +) attach to specific receptor on the recipient cell (F -). ▪ The F pili contract the recipient cell to be in close contact and then canalization occurs through the F pili between the two cells. ▪ Once canal is formed, F plasmid start to mobilize one strand of its ds DNA to the recipient cell. ▪ New ds DNA is formed from the single strand in both donor and recipient cells so the recipient now contain F plasmid and changed to F+ cell which will act as a donor cell

Lecture 12	
Enumerate criteria of ideal antibiotic	1) Selective toxicity i.e. to act at a target site in the infecting organism causing its inhibition without injuring host cells. 2) Bactericidal rather than bacteriostatic. 3) Do not develop antibacterial resistance. 4) Broad spectrum of activity against wide range of microorganisms. 5) Not allergic. 6) Long plasma half-life, 7) Water soluble & stable. 8) Good tissue distribution including C.S.F.
Mention different types of antibiotic combination	1) Synergism : A bactericidal drug when combined with another bactericidal drug may produce a synergistic effect. 2) Antagonism: A bacteriostatic drug combined with a bactericidal drug is likely to produce antagonistic effect. 3) Addition: A bacteriostatic drug combined with another bracteriostatic drug is usually merely additive.
Mention the advantages and disadvantages of antibiotic combinations	**Advantages** 1) Promote treatment in patients suspected to have serious microbial infections. 2) Prevent or delay drug resistance. 3) Effective in mixed or unknown infection. **Disadvantages** 1) ↑ the chance for drug reaction. 2) High cost. 3) Drug antagonism possibility.
Mention 2 mechanisms for antibiotic drugs	1) ↓ intracellular accumulation: - by ↓ permeability &/or ↑ active efflux of the antibiotic (efflux pump). 2) Enzymatic inactivation - ß-lactamases, that inactivate ß-lactams. - Acetyl transferases that inactivate Tetracyclins.

Mention the role of plasmid in antibiotic resistance

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- Bacteria often contain extrachromosomal genetic elements called plasmids (R factors), are class of plasmids that carry genes for resistance to one or more antimicrobial drugs.
- Plasmids frequently carry genes that code for production of enzymes that inactivate or destroy antimicrobial agents e.g.
  1) β- lactamase, which is effective against penicillins & cephalosporins
  2) Acetyl transferase, which destroys chloramphenicol).

Compare between chromosomal and plasmid resistance

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Chromosomal Resistance (Drug Resistant Mutants)	Plasmid Resistance (R Factor) & Transposons
Spontaneous mutation → Altered target.	Presence of R factor (Plasmid that carry the genes of resistance).
Need the presence of antibiotic → selective pressure factor.	Not need for selective pressure factor.
Low frequency of transmission.	High frequency of transmission.
Less common	More common.
e.g. mutation in P12 of 30S ribosomal subunit → Streptomycin resistance.	e.g. RP1 plasmid that encodes resistance to ampicillin.

Mention 2 methods for evaluation of antibiotic activity.

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1) Disc diffusion method (Kirby - Bauer method)
2) Dilution method

Lecture 13

What are the general characters of viruses (enumerate & mention)

1. They do not have membrane-bound nucleus = prokaryotes.
2. They are not cells, they do not have cell membrane = A-cellular. 3. They are obligate intracellular parasites (They do not have ribosomes) & contain one kind of nucleic acid (RNA or DNA)
4. They are the smallest infectious agents (20 to 300 nm in diameter).

What is meant by Nucelocapsid and its function

Def; The capsid with its enclosed nucleic acid

Functions of the capsid are:

a) It protects the viral genome against inactivation by nuclease enzymes.
b) The arrangement of capsomers gives icosahedral, helical or complex symmetry.
c) Role in viral replication "attachment step".
d) Participates in adsorption of virions to susceptible cells. It determines the antigenicity.
Functions of nucleic acid: It is the infectious part of the virus (codes for viral structure and non-structural proteins).

What is the structure of viral envelope, its function ?

Structure: Lipid containing membrane that viruses acquire by budding through host cell membrane.
Lipoprotein, the lipid from the host cell membranes and protein is virus specific. Contains 'glycoproteins' which are spike-like projections on the surface of the virus, which attach to the host cell receptors during infection (attachment).
Function: Determines virus specificity and antigenicity.

Compare between non-enveloped , enveloped viruses in Penetration , release from host cells

	In nonenveloped viruses	In enveloped viruses
Penetration	crossing the plasma membrane directly or by receptor mediated endocytosis	by fusion of viral envelope with cell membrane or with membrane of endosome at the cell surface
Release	Rupture of the cell membrane and release of the mature particles	Budding through the outer cell membrane

Enumerate the steps essential for virus replication	1) Adsorption (Attachment) 2) Penetration 3) Uncoating 4) Viral gene expression 5) Viral nucleic acid synthesis (replication) 6) Assembly 7) Release
1) What are the methods of viral isolation **2) What is the indirect method for viral diagnosis** **3) Discuss how to diagnose viral infection indirectly**	a) Tissue cultures: o Pieces of animal or human tissues are trypsinizied to get separate cells. o Cells are grown in media contain amino acids, vitamins, calf serum and antibiotics. A monolayer or (sheet) of cells is formed on the flat side of the container (flask) within few days. Viruses are inoculated on the monolayer. b) Embryonated egg. c) Animal inoculation.
Mention 2 drugs used to treat the following viruses	1) Herpes viruses → Acyclovir, Ganciclovir 2) HIV Retroviruses →Azidothymidine Squinavir & indinavir 3) influenza A → Zanamivir & oseltamivir Amantadine 4) HBV & HCV → Ribavirin, Interferon -β
Mention types of interferon, origin , their role in viral replication	There are 3 types of interferons: 1) Interferon-α (IFN-α). 2) Interferon -β (IFN-β): treatment of HBV & HCV. 3) Interferon-γ (IFN-γ) Origin: IFN-α and IFN-β are secreted by the infected cell and then bind to a common cell surface receptor, known as the interferon receptor, on both the infected cell and nearby cells. IFN-γ: which induced by activated T cells Function: Interferon induces the synthesis of several host cell proteins e.g. RNA dependent protein kinase (PKR) that contributes to the inhibition of viral replication.

Lecture 14

What are the differences between bacterial and fungal cell wall, cell membrane

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	Bacteria prokaryote	Fungi eukaryote
Cell wall	rigid layer The Peptidoglycan is a complex consists of:- Backbone of N-acetyl-glucosamine alternating with N-acetyl muramic acid. Side chains: (4 amino acids)	rigid cell wall made of chitin (a polymer of N-acetyl glucosamine), glucans, mannans & complex polysaccharide
Cytoplasmic membrane	composed of bi-phospholipids & proteins prokaryotes have no sterols in cytoplasmic membrane except for Mycoplasma	encloses the cytoplasm, vacuoles, endoplasmic reticulum, and mitochondria. It contains ergosterol.

Compare between yeast and yeast like fungi and give examples

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Yeasts	Yeast like (pseudohyphae)
round to oval unicellular fungi	round to oval multi-cellular fungi
reproduce by budding or fission	reproduce by budding
progenitor then detached from mother cell	progenitor remain attached to mother cell giving a chain of elongated yeast cell
e.g. (Cryptococcus neoformans).	e.g. (Candida).

Enumerate methods of formation of fungal spores sexually and asexually

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I) Asexual spores:

a. Sporangiospore

b. Conidiospore:

Arthroconidia (Arthrospores)

Blastoconidia (Blastospores)

Chlamydoconidium (Chlamydospores

Phialoconidia

II) Sexual spores

Basidiospores - Ascospores - Zygospores - Oospores

What are Harmful effect of fungi

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1. Hypersensitivity reactions due to environmental exposure to fungal spores.
2. Infection result from invasion of tissue and organs.
3. Toxicosis

types of fungal toxicosis

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a) Mycotoxicosis: result from accidental consumption of food products contaminated by toxin producing fungi e.g

Ergot alkaloids toxicosis Aflatoxicosis

b) Mycetismus: ingestion of fungi containing preformed toxin e.g: Mushroom poisoning

Give short account on mycetoma	it is subcutaneous mycosis Infection involving the dermis, subcutaneous tissue, muscle and fascia. ▪ Caused by Madurell, Exophiala. ▪ Chronic subcutaneous infection ▪ progress slowly and burrows into deeper tissue ▪ producing abscess which bursts with formation of chronic sinuses discharging fluid containing granules.
Define opportunistic fungi and give example	▪ Caused by saprophytic fungi affecting immunocompromised individuals. ▪ e.g. Candidiasis, Aspergillosis, and Cryptococcosis
Enumerate different clinical types of dermatophytosis and their sites	1) Tinea capitis → scalp & hair. 2) Tinea barbae → beard hair. 3) Tinea ungium → the nails 4) Tinea pedis → between toes 5) Tinea corporis → non-hairy smooth skin. 6) Tinea cruris → groin, moist areas.
Mention the mechanism of action of the following drugs	1) Caspofungin: inhibit 1,3- β glucan synthetase enzyme → Inhibit cell wall synthesis. 2) Polyenes : bind firmly to ergosterol in the fungal cell membrane forming pores → disrupt membrane function → cell death 3) Griseofulvin: interact with microtubules → disrupt mitotic spindle function → inhibit growth→ inhibit nuclear division

Lecture 15

Mention the evidence for immune reactivity "immune surveillance" to tumors:	1) ↑↑ incidence of tumors in: Immunodeficient patients , Young & very old age. 2) Antibodies & immune T cells were detected in patients with tumors. 3) Tumors with severe lympho-reticular infiltration have better prognosis. 4) Certain tumors regress spontaneously (e.g. melanomas). 5) Animals can be specifically immunized against various tumors
Mention the role of oncofetal antigens in tumor immunology.	1) Alpha-fetoprotein (AFP) 5 or more-fold increase is used for monitoring hepatomas & testicular cancers. 2) Carcinoembryonic antigen (CEA) 4–5-fold increase than normal is used to predict recurrence of colorectal tumors.

Compare between TATA, TSTA.

TATA	TSTA
Not unique to tumor	Unique to tumor cells
Expressed on normal cells, but their expression increases in tumor.	Not expressed on normal cells
f Virus-induced tumors express cell surface antigens (distinct from antigens of the virion itself) which are shared by all tumors induced by the same virus.	f Result from mutations of genes due to carcinogenic chemicals or viral transformations. f Usually there is **no** immune response against them due to their low levels.

Mention the role of cytokines response to tumors	Tumor necrosis factor (TNF- α & β) TNF- α & β: stimulate necrosis of tumor cells. TNF- α: inhibits new blood vessel formation in tumor. Interferons alpha, beta & gamma (α, β, γ) ↑MHC-I expression by tumor cells (so, ↑CTL recognition of tumor cells →↑CTL activation →↑CTL killing of tumor cells).

Mention three Mechanisms of tumor escape (evasion) from immune surveillance	1) Tumors may induce specific immunological tolerance (as tumor antigens are self- antigens) 2) Tumors may fail to express neo-antigens (that are immunogenic) or co-stimulatory molecules for T-cell activation. 3) Tumors may show lack or poor expression of MHC antigen. 4) Tumors may stimulate regulatory T cells, which suppress T cell response to tumors. 5) Tumors may secrete immunosuppressive molecules (e.g. Transforming growth factor-β; TGF-β) which inhibits lymphocytes. 6) Tumors may shed their unique antigens, which may interact & block antibodies & T cells from targeting the tumor.
Mention the mechanisms that neo antigens used in immunotherapy.	1) Used as adjuvant to traditional treatments. 2) Using active & passive means of stimulating the non-specific and specific immune response و يعفظالجول ص م

Lecture 16	
Enumerate objectives of vaccination	1) Stimulate immunologic memory → protect recipient against future disease →subsequent infection will be subclinical. 2) Prevent transmission of infection to contacts → ↑ herd immunity & ↓ circulation of microorganism in community.
Enumerate Characteristics of an Ideal Vaccine	1) High efficacy in target populations. 2) Few or no adverse reactions. 3) Safe in immune-compromised individuals & pregnant women. 4) Easy & inexpensive to deliver to developing countries. 5) Stable during transport & storage. 6) Induces life-long immunity. 7) Stimulates both humoral & CMI responses.
What is the mechanism of action of live attenuated vaccine	▪ The vaccine virus replicates in recipient → amplifies amount of antigen (Ag) available for presentation to host immune system → host immune response (IR) resembling what occurs after natural infection.
Mention advantages & disadvantages Of any type of vaccines	راجع محمد ي ع ة التطعيمات 😊👍