Biochemistry Cell biology Immunology

Most Important Portion of All Msc Entrance Exams

BHU JNU CUCET GAT-B(JNU CEEB) IIT JAM TIFR

Sanjoy Das

M.Sc ,School of Life Sciences

Jawaharlal Nehru University(JNU), New Delhi

Ditam Chakraborty

M.Sc ,Department of Biotechnology

Jadavpur University(JU),Jadavpur,Kolkata,West Bengal

Sourav Mondal

M.Sc ,Department Of Zoology

University of Kalyani, West Bengal

Contents

A. Biochemistry

1.Aminoacids and their properties

2.Titration of aminoacis

3.Isoelectric points, Pka

4.Absorption of aminoacids by UV Rays

5.Peptide and Ramachandran plots

6.Structure of aminoacids(primary , secondary and tertiary structure)

7.Collagen

8.Protein Sequencing

9.Protein Assay

10.Beer Lambert Equation

11.Carbohydrates and properties

12.Monosaccharides

13.Disaccharides

14.Heteropolysaccharides

15.Lipids and Their properties

16.Types of lipids

17.Lipid derivatives

18.Enzymes

19.Classification of Enzymes

20.Cofactors and coenzymes

21.Enzymes kinetics

22.Enzymes Inhibition

23.Enzymes Regulation

24.Isoenzymes

25.Zymogens

26.Ribozymes

27.Vitamins

28.Bioenergetics

29.Carbohydrate Metabolism

30.Amino acid Metabolism

31.Lipid Metabolism

32.Nucleic acid Metabolism

B. Cell biology

1.Plasma membrane

2.Types of phospholipids and their properties

3.ABO blood group

4.Membrane Transport

5.Endoplasmic reticulum(ER)

6.Golgi Apparatus

7.All sorting signals

8.All Cell organelle and their Specific Enzymes

9.Cytoskeletons and their all inhibitors

10.Cell cycle

11.Cell Division

12.Cell Signalling

13.Cell Junction

C.Immunology

1.Types of immunity

2.Formation and Maturation of Lymphocytes

3.Pathway for antigen Prossesing and Presentation

4.Cells and Organs of immune system

5.Antigen

6.Immunoglobulins

7.Types of immunoglobulin and their functions

8.Antigen Antibody Interactions

9.ABO Blood group

10.MHC molecules(Major Histocompatibility Complex)

11.Abzymes,Toxoids, and Vaccine

12.Hypersensitivity

13.Histamine receptors

14.Cytokines and their functions

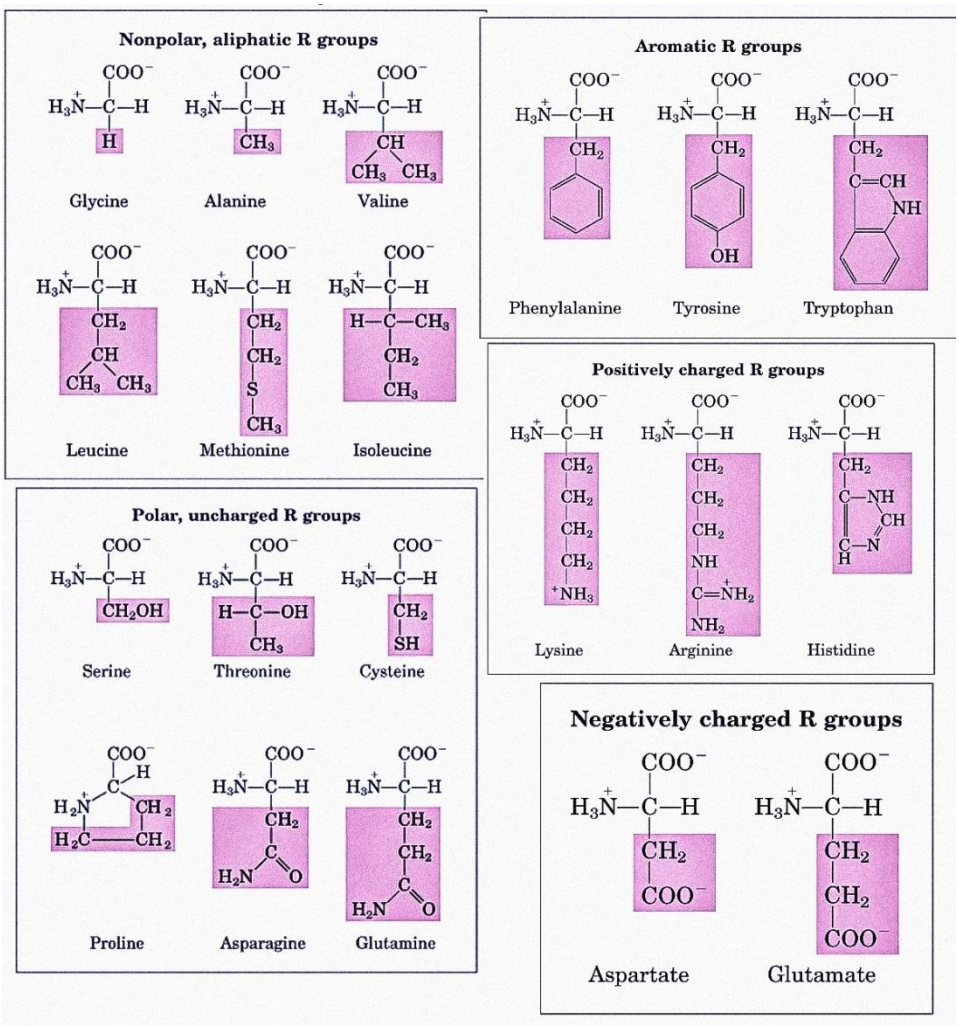
15.Guidance For All Msc Entrance Examination In India and relevant books

Biochemistry

Aminoacids and proteins

Properties of aminoacids

How many standard amioacids are found in biological systems and side chains properties of the each aminoacids, you should remember name of the all aminoacids specially glycine,proline,charged and all aromatic aminoacids. Remember the below list clearly.



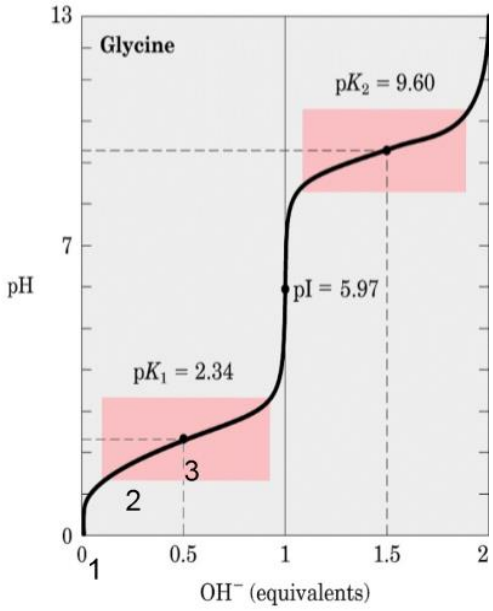
Amino acids consist of:

- a basic amino group (—NH2)
- an acidic carboxyl group (—COOH)
- a hydrogen atom (—H)
- a distinctive side chain (—R).

20 Standard aminoacids are present in the biological system.

Titration of the aminoacids:(remember Pka value of these two aminoacids)

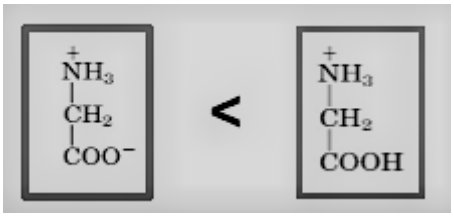
Titration curve of amino acid (glycine)



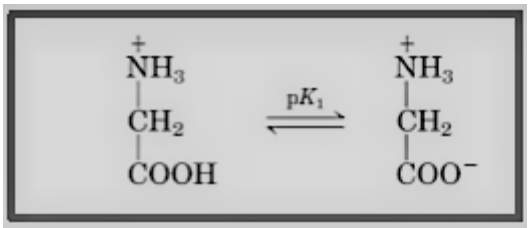
1. At a very low pH (acidic) both groups are fully protonated where the solution predominantly contains



2. When the pH is raised, the –COOH group start to be deprotonated and the proportion will be:



3. pH= pKa1, where it will act as a buffer and the solution will contain an equal amount of :



What is Pka?what is isoelectric point?

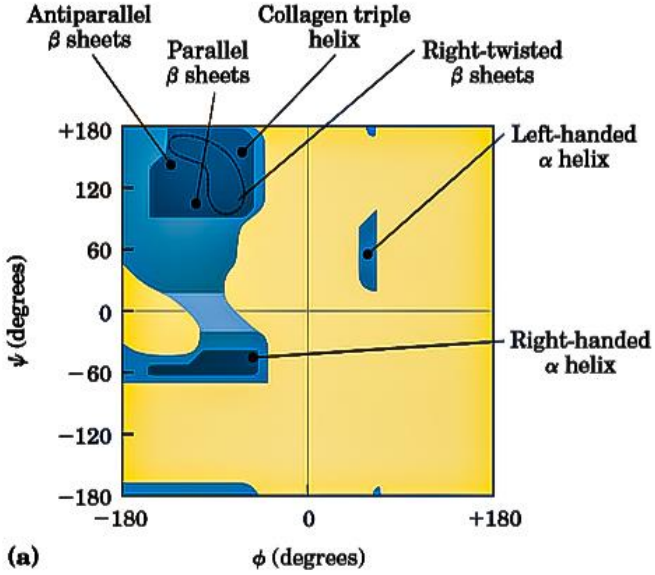
The **pKa value** is one method used to indicate the strength of an acid. **pKa** is the negative log of the acid dissociation constant or Ka **value**. A lower **pKa value** indicates a stronger acid.

The **isoelectric point** (**pI**), is the pH at which a molecule carries no net electric charge or is electrically neutral.

Absorption of UV light by aminoacids

Aminoacids	Absorbance(nm)
Phenylalanine	257.4
Tyrosine	274.6
Tryptophan	279.8

Peptide bond and Ramachandran plot



SECONDARY STRUCTURE	BONDS	
	PHI	SHI
Right handed Alpha helix	-57	-47
Antiparallel Beta sheet	-139	+135
Parallet beta sheet	-119	+113

Secondary stucture of the proteins(alpha helix):

How many acids present per turn of the aminoacids-**3.6**

Pitch length-**0.54nm**

Parameters of Beta sheets-

Diastance between the adjacent aminoacids of the Beta sheet-**3.5A**

What is the force that stabilizes the Tertiary and quarternary structure of the aminoacids?

The major force is Hydrophobic interaction, and the others are

1.Hydrogen bonds

2.Electrostatic interaction

3.Van daar waal interaction

How to determined the molecular weight of the proteins?

Average Molecular weight of the aminoacid is appproximatly 110Da [1000Da=1Kda]

What is the molecular weight of the protein which containing 200 aminoacids?

Answer=110*200=22000Da

Collagen:

Collagen is a Extracellular Matrix proteins that is present abundantly in the connective tissue.The basic unit of the collagen protein is Tropocollagen.Tropocollagen is made up of Three left handed alpha helix polypeptides.Amino acids sequence of the alpha chain is Gly-X-Y,where X is Proline and Y is 3 or 4 Hydroproline and 5hydrolysine.Post translational modification(hydroxilation) is occur in the golgi apparatus by two main important enzyme-

1.Prolyl hydroxylase

2.Lysyl hydroxylase

Vitamin C is the Cofactor for the these enzyme.Deficiency of the vitamin C Scurvy disease occur.

Protein sequencing:

N-terminal analysis of aminoacids-1-Fluoro-2,4-Dinitrobenzene(FDBN) anf dansyl chloride are used for N-terminal analysis of the aminoacids.

Edmund degradation: It is the process by which proteins are sequence from N-terminal of the aminoacids.

Cleavege of site of different proteases

PROTEASE	SITE OF CLEAVAGE
Pepsin	Amino side of the Aromatic aminoacids(tryptophan,tyrosine, and Phenylalanine)
Trypsin	Carboxyl side of the Arginine and Lysine

Chymotrypsin	Carboxyl side of the aromatic aminoacids(tryptophan,tyrosine, and Phenylalanine)
Elastase	Carboxyl side of the Gly,Ala and ser.
Carboxypeptidase A	Carboxyl side of the Long hydrophobic aminoacids
Aminopeptidase	Amino terminal of the any aminoacids

PROTEIN ASSAY:Biuret assay,Folin method, and Bradford method

BEER LAMBERT EQUATION: Equation you must read because lots of questions are coming from that area also.

$$A = \varepsilon c l$$

A	Absorbance	
ε	Molar absorption coefficient	$M^{-1}cm^{-1}$
c	Molar concentration	M
l	optical path length	cm

Purification of the proteins by different techniques-

1.ELECTROPHORESIS

2.CHROMATOGARPHY

3.CENTRIFUGALTION

Just you have to know the function of those purification techniques.

Carbohydrates

What are carbohydrates?

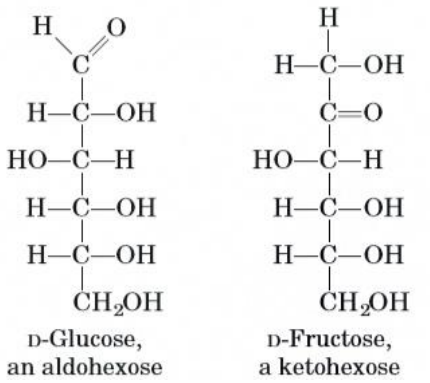
Carbohydrates are polyhydroxy aldehydes or ketones, or substances that yield such compounds on hydrolysis.

Types of the carbohydrates?

Monosaccharides,oligosaccides and polysaccarides

Monosaccharides

Characteristics of the Monosaccarides:specially read characteristics of the glucoses and fructoses.



What do you mean Aldolases and ketoses?

If the carbonyl group is at an end of the carbon chain (that is, in an aldehyde group) the monosaccharide is an aldose; if the carbonyl group is at any other position (in a ketone group) the monosaccharide is a ketose.

What do you mean by chiral molecules?

Chiral carbon is **carbon** atom which is bonded with four different compounds(groups) at the same time..

What do you mean by enantiomers?

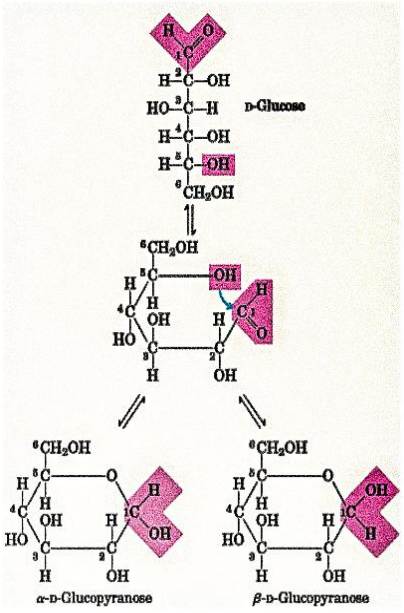
Enantiomers are chiral molecules that are *mirror images* of one another. Furthermore, the molecules are *non-superimposable* on one another. This means that the molecules cannot be placed on top of one another and give the same molecule

What is epimer?(remember the example of the mannose glucose and glucose galactose)

Two sugars that differ only in the configuration around one carbon atom are called epimers; D-glucose and D-mannose, which differ only in the stereochemistry at C-2, are epimers, as are D-glucose and Dgalactose

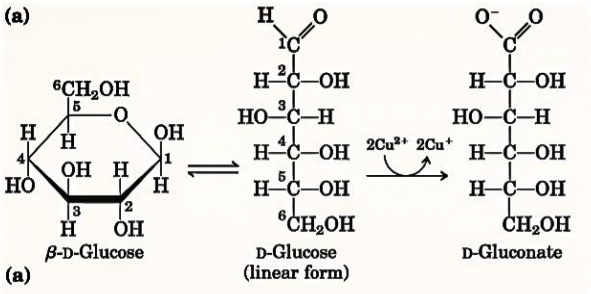
Anomer: Isomeric forms of monosaccharides that differ only in their configuration about the hemiacetal or hemiketal carbon atom are called anomers

Mutarotation: The alpha and Beta anomers of D-glucose interconvert in aqueous solution by a process called mutarotation.



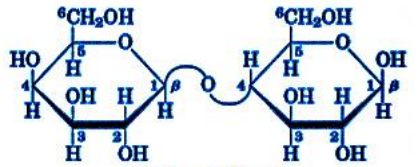
Monosaccharides Are Reducing Agents

Monosaccharides can be oxidized by relatively mild oxidizing agents such as ferric (Fe3—————————) or cupric (Cu2+) ion.

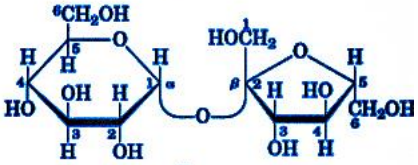


Disaccharides:

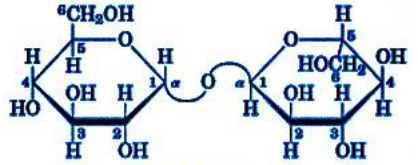
Sucrose ,maltose and lactose(what is the basic units of these disaccarides, what type of bonds are present between them and and their physiological role)



Lactose (β form)
β-D-galactopyranosyl-(1→4)-β-D-glucopyranose
Gal(β1→4)Glc



Sucrose
α-D-glucopyranosyl β-D-fructofuranoside
Glc(α1↔2β)Fru



Trehalose
α-D-glucopyranosyl α-D-glucopyranoside
Glc(α1↔1α)Glc

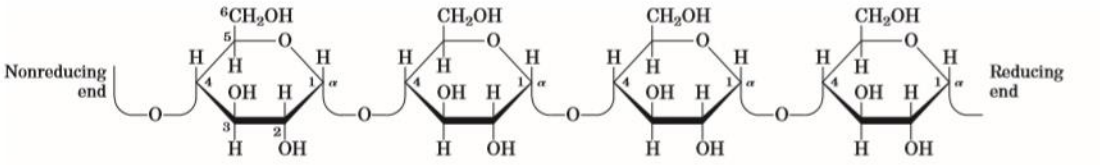
Polysaccharides:

Homopolysaccharides

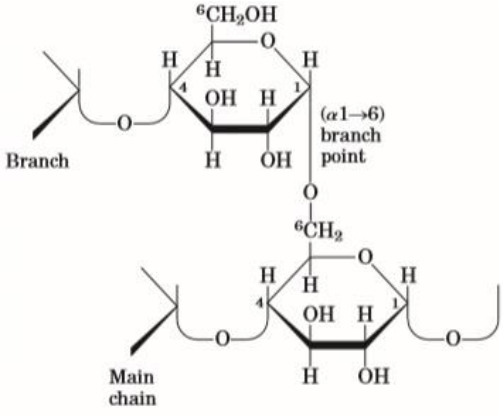
Starch: Starch contains two types of glucose polymer, amylose and amylopectin. The former consists of long, unbranched chains of D-glucose residues connected by (alpha1-4) linkages.

What is amylose and amylopectin? What is the difference between them?

Amylose consists of a linear, helical chains of roughly 500 to 20,000 alpha-D-glucose monomers linked together through alpha (1-4) glycosidic bonds.



Amylopectin molecules are huge, branched polymers of glucose, each containing between one and two million residues. In contract to amylose, amylopectin is branched.



Glycogen:basic units and where it is present?

Glycogen is the main storage polysaccharide of animal cells. Like amylopectin, glycogen is a polymer of (alpha1-4)-linked subunits of glucose, with (alpha1-6)-linked branches, but glycogen is more extensively branched (on average, every 8 to 12 residues) and more compact than starch. Glycogen is especially abundant in the liver.

Cellulose:what is the basic unit of the cellulose?What kind of bond is present in celluose? Where it is present and physiological roles?

Cellulose molecule is a linear, unbranched homopolysaccharide, consisting of 10,000 to 15,000 D-glucose units. But there is a very important difference: in cellulose the glucose residues have beta configuration.

Chitin:basic units,bonds between them and where it is present?

Chitin is a linear homopolysaccharide composed of N-acetylglucosamine residues in beta linkage. . Chitin is the principal component of the hard exoskeletons of nearly a million species of arthropods—insects, lobsters, and crabs.

Polymer	Type*	Repeating unit†	Size (number of monosaccharide units)	Roles/significance
Starch				Energy storage: in plants
Amylose	Homo-	(α1→4)Glc, linear	50–5,000	
Amylopectin	Homo-	(α1→4)Glc, with (α1→6)Glc branches every 24–30 residues	Up to 10^6	
Glycogen	Homo-	(α1→4)Glc, with (α1→6)Glc branches every 8–12 residues	Up to 50,000	Energy storage: in bacteria and animal cells
Cellulose	Homo-	(β1→4)Glc	Up to 15,000	Structural: in plants, gives rigidity and strength to cell walls
Chitin	Homo-	(β1→4)GlcNAc	Very large	Structural: in insects, spiders, crustaceans, gives rigidity and strength to exoskeletons
Dextran	Homo-	(α1→6)Glc, with (α1→3) branches	Wide range	Structural: in bacteria, extracellular adhesive
Peptidoglycan	Hetero-; peptides attached	4)Mur2Ac(β1→4) GlcNAc(β1	Very large	Structural: in bacteria, gives rigidity and strength to cell envelope
Agarose	Hetero-	3)D-Gal(β1→4)3,6-anhydro-L-Gal(α1	1,000	Structural: in algae, cell wall material
Hyaluronate (a glycosamino-glycan)	Hetero-; acidic	4)GlcA(β1→3) GlcNAc(β1	Up to 100,000	Structural: in vertebrates, extracellular matrix of skin and connective tissue; viscosity and lubrication in joints

Heteropolysaccharides

Glycosaminogycans

Dermatan sulphate

Keratin sulphate

Heparin

Heparan sulphate

Hyaluronic acids and proteoglycans

Lipids

What are lipids and their function?

Any of a class of organic compounds that are fatty acids or their derivatives and are insoluble in water but soluble in organic solvents. They include many natural oils, waxes, and steroids.

Cholesterol molecules are used in biological system like steroids hormone.

You have to remember the common names of fatty acids and the number of carbon molecules in the fattyacids like palmatic acid has 16 carbon molecules.

Carbon skeleton	Structure*	Systematic name†	Common name (derivation)	Melting point (°C)	Solubility at 30 °C (mg/g solvent)	
					Water	Benzene
12:0	$CH_3(CH_2)_{10}COOH$	*n*-Dodecanoic acid	Lauric acid (Latin *laurus*, "laurel plant")	44.2	0.063	2,600
14:0	$CH_3(CH_2)_{12}COOH$	*n*-Tetradecanoic acid	Myristic acid (Latin *Myristica*, nutmeg genus)	53.9	0.024	874
16:0	$CH_3(CH_2)_{14}COOH$	*n*-Hexadecanoic acid	Palmitic acid (Latin *palma*, "palm tree")	63.1	0.0083	348
18:0	$CH_3(CH_2)_{16}COOH$	*n*-Octadecanoic acid	Stearic acid (Greek *stear*, "hard fat")	69.6	0.0034	124
20:0	$CH_3(CH_2)_{18}COOH$	*n*-Eicosanoic acid	Arachidic acid (Latin *Arachis*, legume genus)	76.5		
24:0	$CH_3(CH_2)_{22}COOH$	*n*-Tetracosanoic acid	Lignoceric acid (Latin *lignum*, "wood" + *cera*, "wax")	86.0		
16:1(Δ^9)	$CH_3(CH_2)_5CH{=}CH(CH_2)_7COOH$	*cis*-9-Hexadecenoic acid	Palmitoleic acid	1–0.5		
18:1(Δ^9)	$CH_3(CH_2)_7CH{=}CH(CH_2)_7COOH$	*cis*-9-Octadecenoic acid	Oleic acid (Latin *oleum*, "oil")	13.4		
18:2($\Delta^{9,12}$)	$CH_3(CH_2)_4CH{=}CHCH_2CH{=}CH(CH_2)_7COOH$	*cis*-,*cis*-9,12-Octadecadienoic acid	Linoleic acid (Greek *linon*, "flax")	1–5		
18:3($\Delta^{9,12,15}$)	$CH_3CH_2CH{=}CHCH_2CH{=}CHCH_2CH{=}CH(CH_2)_7COOH$	*cis*-,*cis*-,*cis*-9,12,15-Octadecatrienoic acid	α-Linolenic acid	−11		
20:4($\Delta^{5,8,11,14}$)	$CH_3(CH_2)_4CH{=}CHCH_2CH{=}CHCH_2CH{=}CHCH_2CH{=}CH(CH_2)_3COOH$	*cis*-,*cis*-,*cis*-,*cis*-5,8,11,14-Icosatetraenoic acid	Arachidonic acid	−49.5		

What is saturated fattyacids and unsaturated fattyacids?

Saturated fatty acids lack double bonds between the individual carbon atoms, while in **unsaturated fatty acids** there is at least one double bond in the fatty acid chain. **Saturated** fats tend to be solid at room temperature and from animal sources, while **unsaturated** fats are usually liquid and from plant sources.

What is triglycerides and waxes?what is the difference between them?

Triacylglycerols are composed of three fatty acids each in ester linkage with a single glycerol**.**

Waxes have a different structure (esters of fatty acids with long chain alcohols) and can be found in both animals and plants.

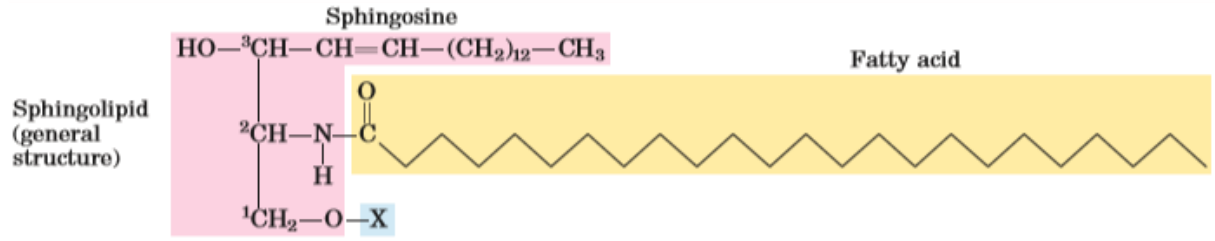
What is phospholipids and their types?

A phospholipid is made up of two fatty acid tails and a phosphate group Head. Fatty acids are long chains that are mostly made up of hydrogen and carbon, while phosphate groups consist of a

phosphorus molecule with four oxygen molecules attached. These two components of the phospholipid are connected via a third molecule, glycerol.

Types of phospholipids	Head group	Net charge at neutral PH
Phosphatidic acids	-H	-1
Phophatidylethanolamine	ethanolamine	0
Phosphatidylcholine	Choline	0
Phosphatidylserine	serine	-1
cardiolipin	phosphotidylglycerol	-2

Spingolipids basic structure: Sphingolipids are composed of one molecule of the long-chain amino alcohol sphingosine or one of its derivatives, one molecule of a long-chain fatty acid, and a polar head group that is joined by a glycosidic linkage in some cases and by a phosphodiester in others.

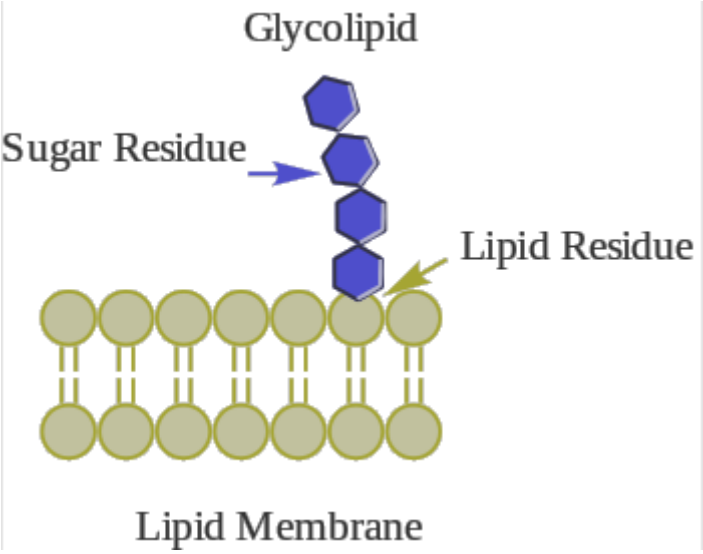


Sphingolipids

Phospholipids	Head groups
Ceramide	-H
Sphingomyelin	Phosphocholine
Ganglioside	Complex Oligosaccharides
Lactosylceramide	Di, tri, tetra, saccharide

Glycolipids:

Glycolipids are lipids with Carbohydrates attached by a glycosisdic bonds. Their role is to maintain the stability of the cell membrane and to facilitate cellular recognition, which is crucial to the immune response and in the connections that allow cells to connect to one another to form tissues.



Types of glycolipids-like globosides,Cerebrosides.

You should remember the that cholesterol is precursors of the steroids hormones.

Eicosonoids:

Prostaglandins: Prostaglandins contain a five-carbon ring originating from the chain of arachidonic acid. Their name derives from the prostate gland. Prostaglandins elevate body temperature (producing fever) and cause inflammation and pain

Thromboxenes: The thromboxaneshave a six-membered ring containing an ether. They are produced by platelets (also called thrombocytes) and act in the formation of blood clots and the reduction of blood flow to the site of a clot.

The nonsteroidal antiinflammatory drugs (NSAIDs)— aspirin, ibuprofen, and meclofenamate, for example— were shown by John Vane to inhibit the enzyme prostaglandin H2 synthase (also called cyclooxygenase or COX), which catalyzes an early step in the pathway from arachidonate to prostaglandins and thromboxanes

Leukotrienes: Leukotrienes, first found in leukocytes, contain three conjugated double bonds. They are powerful biological signals. For example, leukotriene D4, derived from leukotriene A4, induces contraction of the muscle lining the airways to the lung.

Lipoproteins like LDL,HDL

Enzymes

What is Enzyme and their function.classification of the enzyme and you should remember the names of the enzymes the the classes like all dehydrogenase enzymes belongs to oxidoreductase.

Enzyme: Proteins that speeds up the rate of a chemical reaction in a living organism. An **enzyme** acts as catalyst for specific chemical reactions, converting a specific set of reactants (called substrates) into specific products.

Enzymes, like other proteins, have molecular weights ranging from about 12,000 to more than 1 million. Some enzymes require no chemical groups for activity other than their amino acid residues. Others require an additional chemical component called a **cofactor**—either one or more inorganic ions, such as Fe2+, Mg2+, Mn2+, or Zn2+, **or a complex organic or metalloorganic molecule called a coenzyme**. Some enzymes require both a coenzyme and one or more metal ions for activity. A coenzyme or metal ion that is very tightly or even covalently bound to the enzyme protein is called **a prosthetic group**. A complete, catalytically active enzyme together with its bound **coenzyme** and/or metal ions is called **a holoenzyme**. The protein part of such an enzyme is called **the apoenzyme or apoprotein**.

Classification of enzymes

Enzymes types	Types of Reaction catalyzed
Oxidoreductases	Transfer of electrons (hydride ions or H atoms)
Transferases	Group transfer reactions
Hydrolases	Hydrolysis reactions (transfer of functional groups to water)
Lyases	Addition of groups to double bonds, or formation of double bonds by removal of groups
Isomerases	Transfer of groups within molecules to yield isomeric forms

Ligases	Formation of COC, COS, COO, and CON bonds by condensation reactions coupled to ATP cleavage

Which cofactor is involved in which enzyme reaction

Cofactors	Enzymes
Cu2+	Cytochrome oxidase
Fe2+ or Fe3+	Cytochrome oxidase, catalase, peroxidase
K+	Pyruvate kinase
Mg2+	Hexokinase, glucose 6-phosphatase, pyruvate kinase
Mn2+	Arginase, ribonucleotide reductase
Mo	Dinitrogenase
Ni2+	Urease
Se	Glutathione peroxidase
Zn2+	Carbonic anhydrase, alcohol dehydrogenase, carboxypeptidases A and B

Coenzymes That Serve as Transient Carriers of Specific Atoms or Functional Groups

Coenzyme	Chemical group transfered	Obtain from
Thiamine pyrophosphate	**Aldehydes**	**Thiamine (vitamin B1)**
Flavin adenine dinucleotide	**Electrons**	**Riboflavin (vitamin B2)**
Nicotinamide adenine dinucleotide	**Hydride ion (:H+)**	**Nicotinic acid (niacin)**
Pyridoxal phosphate	**Amino groups**	**Pyridoxine (vitamin B6)**
Coenzyme A	**Acyl groups**	**Pantothenic acid and other compounds**

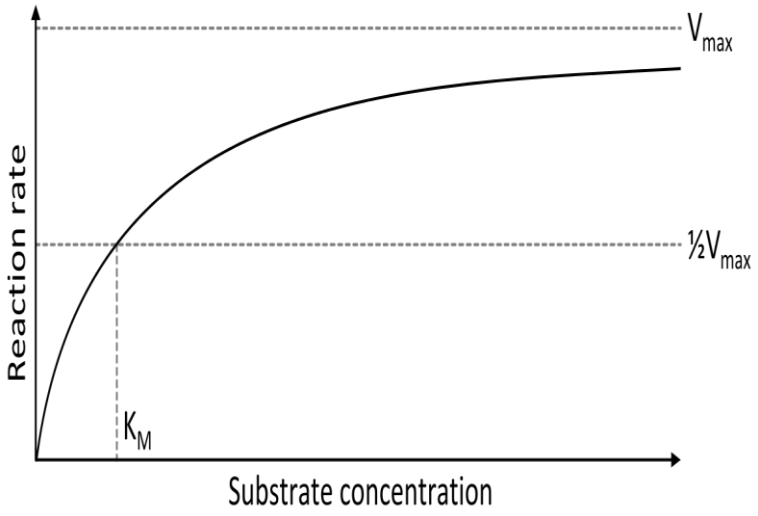
Tetrahydrofolate	One-carbon groups	Folate
Lipoate	Electrons and acyl groups	
5Deoxyadenosylcobalamin(vitamin12)	H atoms and alkyl groups	Vitamin B12
Biocytin	CO2	Biotin

Enzymes lowering the Activaton Energy of the reaction.(sometimes question are asked from this line). Catalysts enhance reaction rates by lowering activation energies.

Enzyme Kinetics: Michaelis Menten Equation(most important),Vmax,Km you must read.

$$V_0 = \frac{V_{max}[S]}{K_m + [S]}$$

This is the Michaelis-Menten equation, the rate equation for a one-substrate enzyme-catalyzed reaction. It is a statement of the quantitative relationship between the initial velocity V0, the maximum velocity Vmax, and the initial substrate concentration [S], all related through the Michaelis constant Km.



Impotance of the Km

Km does represent a measure of the affinity of the enzyme for its substrate in the ES complex. When substrate concentration increases Km also increase.

Turn over number(Kcat):

Km is the maximal number of molecules of substrate converted to product per active site per unit time when the enzyme is saturated with substrate .Kcat represents the kinetic efficiency of enzymes.

Specificity Constant:

Kcat/Km known as Specificity constant. It measure the efficiency of the enzymes.Lower value of the Km leads to high specificity of the enzymes.That means high value of the Kcat and low value of the Km leans to high specificity of the enzymes.

Lineweaver Burk plots

The **Lineweaver–Burk plot** (or **double reciprocal plot**) is a graphical representation of the Lineweaver–Burk equation of enzyme kinematics.

The plot provides a useful graphical method for analysis of the Michaelis Menten equation, as it is difficult to determine precisely the V_{max} of an enzyme-catalysed reaction:

$$V = \frac{V_{\max}[S]}{K_m + [S]}$$

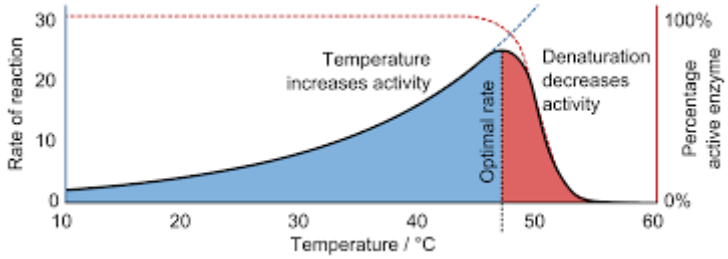
Taking the reciprocal gives

$$\frac{1}{V} = \frac{K_m + [S]}{V_{\max}[S]} = \frac{K_m}{V_{\max}}\frac{1}{[S]} + \frac{1}{V_{\max}}$$

Relation between graph and Km,Vax

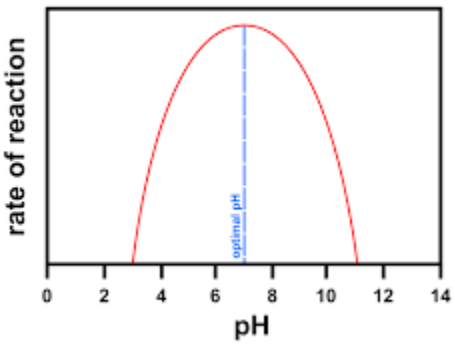
Effect of PH and temperature

Enzyme is denatured or unfolded at high temperature. Due to the denaturation of the protein catalytic property of the enzyme decreased. Every enzyme performs optimum activity at specific temperture.



Effect of PH

Every enzyme perform optimum activity at specific pH, they exhibit a bell-shaped curve. Small intestine enzymes show their optimum activity at high pH whereas pepsin shows optimum activity at low pH(2-3).



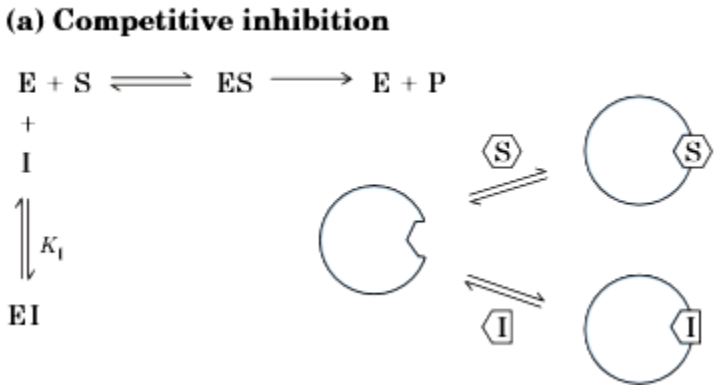
Enzyme Inhibition:

Competitive,Noncompetitive and uncompetitive Inhibition and Graph

Enzyme inhibition may be reversible or irreversible. Reversible inhibition can be done non-covalently or compete with substrate of the enzyme. In case of the irreversible enzyme inhibitor binds with covalently in the active site of the molecule and permanently inhibit the activity of the enzyme.

Competitive inhibition:

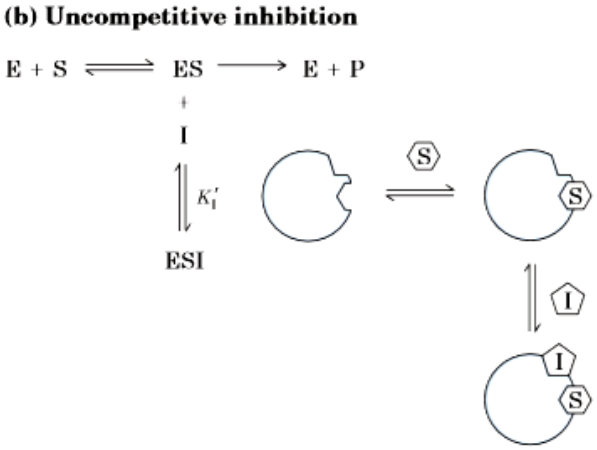
Reversible Inhibition One common type of reversible inhibition is called competitive inhibition. A competitive inhibitor competes with the substrate for the active site of an enzyme. While the inhibitor (I) occupies the active site it prevents binding of the substrate to the enzyme.In competitive inhibition,Km increase but Vmax remain constant Inhibitor can not change the turnover number of the enzyme.



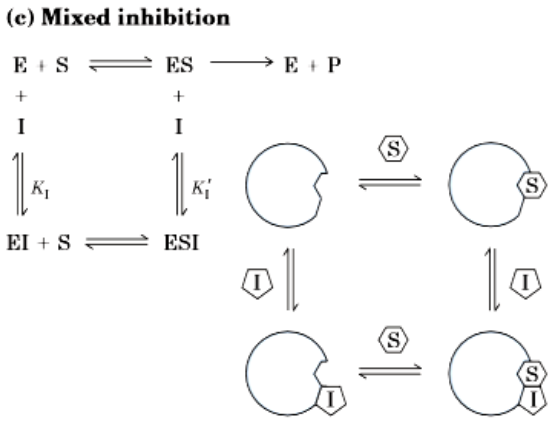
Two other types of reversible inhibition, uncompetitive and mixed.

Uncompetitive:

An uncompetitive inhibitor binds at a site distinct from the substrate active site and, unlike a competitive inhibitor, binds only to the ES complex. Apparent Vmax and Km both decrease.



Mixed Inhibitor(Non-competitive inhibition): A mixed inhibitor also binds at a site distinct from the substrate active site, but it binds to either E or E.



Types of inhibition	Vmax	Km
Competitive	No change	Increase
Non-competitive(Mixed)	Decrease	No change
Uncompetitive	Decrease	decrease

Regulation of enzymes: Specially Allosteric regulation by Non covalent modification

The activity of allosteric enzymes is adjusted by reversible binding of a specific modulator to a regulatory site. Modulators may be the substrate itself or some other metabolite, and the effect of the modulator may be inhibitory or stimulatory. The kinetic behavior of allosteric enzymes reflects cooperative interactions among enzyme subunits.

Enzymes Regulation By covalent Modification like phosphorylation.

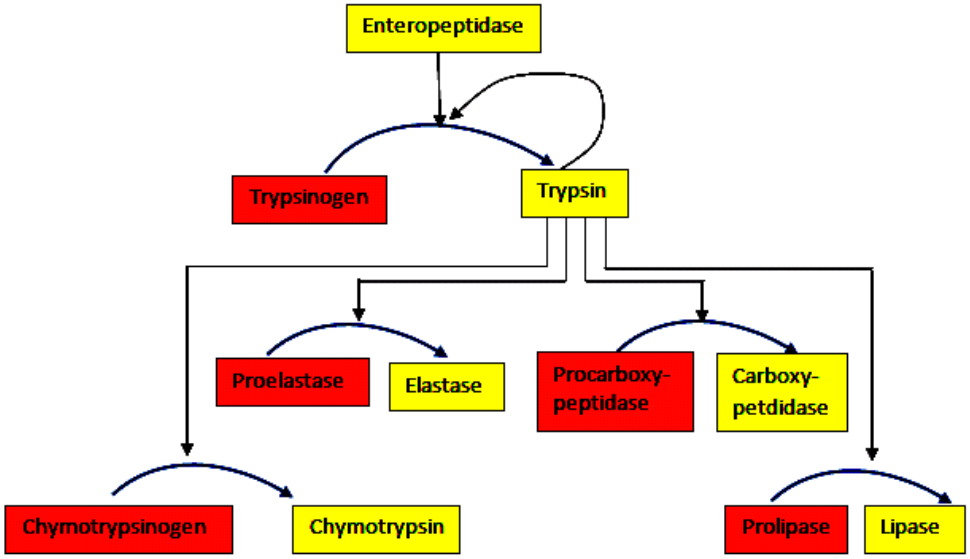
What are Isoenzymes and Zymogens? And their examples?

Isoenzymes

Isoenzymes (also called isozymes) are alternative forms of the same enzyme activity that exist in different proportions in different tissues. Isoenzymes differ in amino acid composition and sequence. Example:LDH(Lactose Dehydogenase)(LDH1,LDH2,LDH3,LDH4,LDH5) and Hexokinase, Glucokinase.

ZYMOGENS

A **zymogen**(**proenzyme)** is an inactive precursor of an enzyme. It will be functionally active when it is cleaved by different enzymes (proteases).



What is Ribozymes?

Ribozymes (**ribo**nucleic acid en**zyme**s) are RNA molecules that have the ability to catalyze specific biochemical reactions, including RNA splicing in gene expression.Ribozymes require Mg2+ ions as cofactor.

Function of the ribozymes

1.It can break phosphodiester bonds.(RNaseP)

2.It can create peptide bonds.(in the ribosome-Peptidyl transferase)

Vitamins

Coenzymes That Serve as Transient Carriers of Specific Atoms or Functional Groups

Coenzyme	Chemical group transfered	Obtain from
Thiamine pyrophosphate	Aldehydes	Thiamine (vitamin B1)
Flavin adenine dinucleotide	Electrons	Riboflavin (vitamin B2)
Nicotinamide adenine dinucleotide	Hydride ion (:H+)	Nicotinic acid (niacin)
Pyridoxal phosphate	Amino groups	Pyridoxine (vitamin B6)
Coenzyme A	Acyl groups	Pantothenic acid and other compounds
Tetrahydrofolate	One-carbon groups	Folate
Lipoate	Electrons and acyl groups	
5Deoxyadenosylcobalamin(vitamin12)	H atoms and alkyl groups	Vitamin B12
Biocytin	CO2	Biotin

Vitamins and deficiency disease

Vitamins	Deficiency disease
Vitamin A	**Night blindness**
Vitamin C	**Scurvy**
Vitamin D	**Rickets,Osteomalacia**
Vitamin B1	**Beriberi**
Vitamin B2	**Dermatitis**
Vitamin B3	**Pellagra**
Vitamin B6	**Megaloblastic Anemia**
Vitamin B12	**Pernecious Anemia**
Vitamin K	**Bleeding Diathesis**

What are the water soluble and fat soluble vitamins?

Vitamin A, Vitamin D, Vitamin E, And Vitamin K are fat-soluble Vitamins

Vitamin B complex and vitamin C are Wter-soluble Vitamins.

Respiration

Energetics

Relationships among K

,Delta G, and the Direction of Chemical Reactions under Standard Conditions

Delta G	Reaction
Negative	Proceed Forword reaction(spontaneously)
Zero	At equilibrium

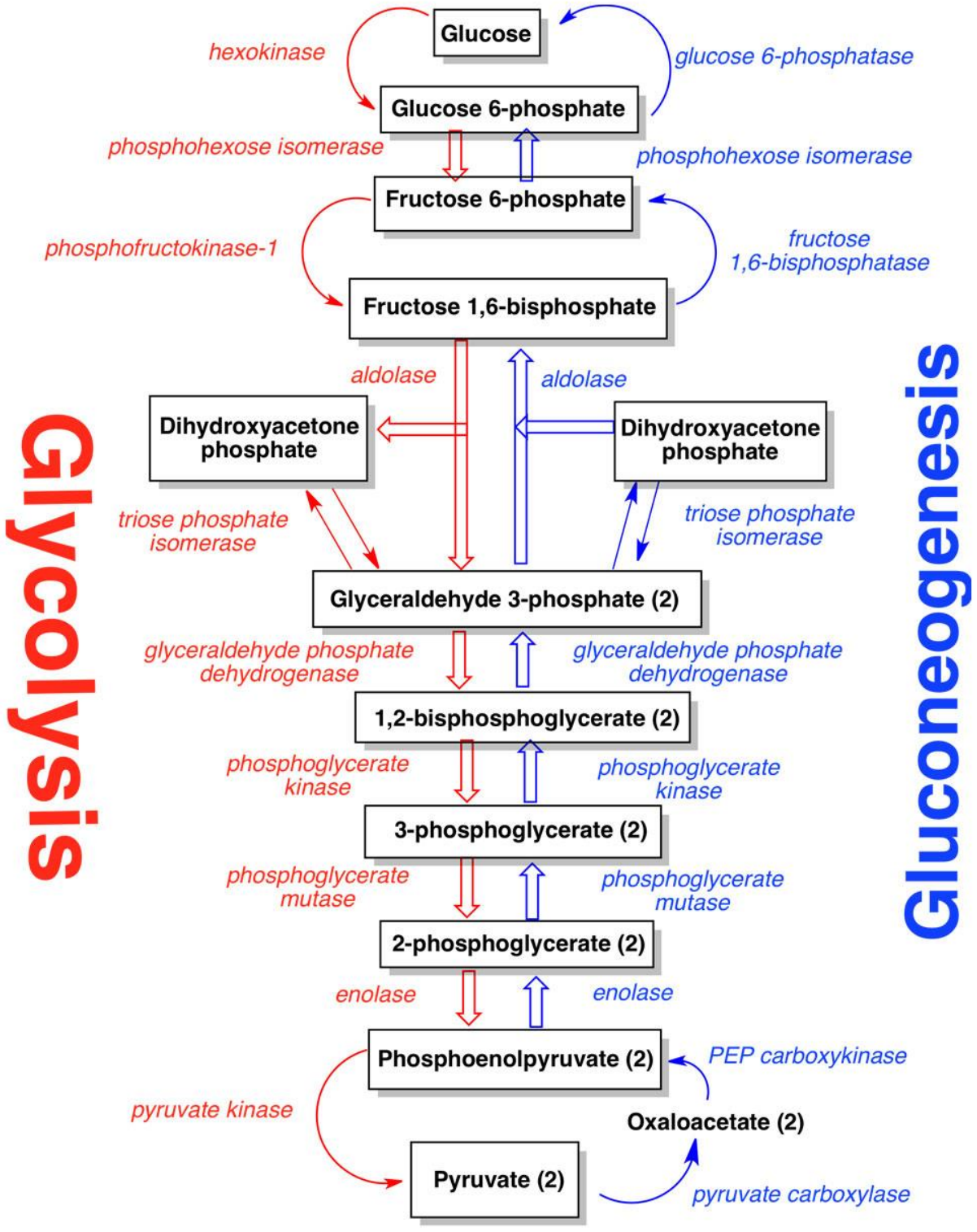
Positive	Proceed reverse

Relationhip Standard Free Energies and metabolites

Metabolites	KJ/mol(delta G)	Kcal/mol(delta G)
Phosphoenolpyruvate	**-61.9**	**-14.8**
1,3-Bisphosphoglycerate	**-49.3**	**-11.8**
ATP(ATP ►AMP+ppi)	**-45.6**	**-10.9**
Phosphocreatine	**-43.0**	**-10.3**
ADP(ADP → AMP+Pi)	**-32.8**	**-7.8**
ATP(ATP →ADP+Pi)	**-30.5**	**-7.3**

Carbohydrates Metabolism

Glycolysis (Embden-Meyerhoff-Parnas Pathway)



You should remember the Whole cycle because it is very important.

ATP calculation of the Glycolysis is most important topicc for all the biological entrance examniation.

How many ATP are produced from single glucose molecule:

2 NADH produced in glycolysis (2.5 ATP each) + 8 NADH produced in Krebs cycle (2.5ATP each) + 2 FADH2 produced(1.5 ATP Each) + 2 ATP produced in the Krebs cycle + 2 ATP produced in glycolysis = 5 + 20 + 3 + 2 + 2 = 32 ATP

And What is Substrate level Phosphorylation?In which step of the glycolysis pathway substrate level phosphorylation occurs.

Substrate-level phosphorylation is a metabolic reaction that results in the formation of ATP or GTP by conversion of a higher energy substrate (whether phosphate group attached or not) into lower energy product and a using some of the released chemical energy, the Gibbs free energy, to transfer a phosphoryl (PO_3) group to ADP or GDP from another phosphorylated compound.

1,3-bisphosphoglycerate is then dephosphorylated via phosphoglycerate kinase, producing 3-phosphoglycerate and ATP through a substrate-level phosphorylation.

The second substrate-level phosphorylation occurs by dephosphorylating phosphoenolpyruvate, catalyzed by pyruvate kinase, producing pyruvate and ATP.

Fates of the glycolysis.

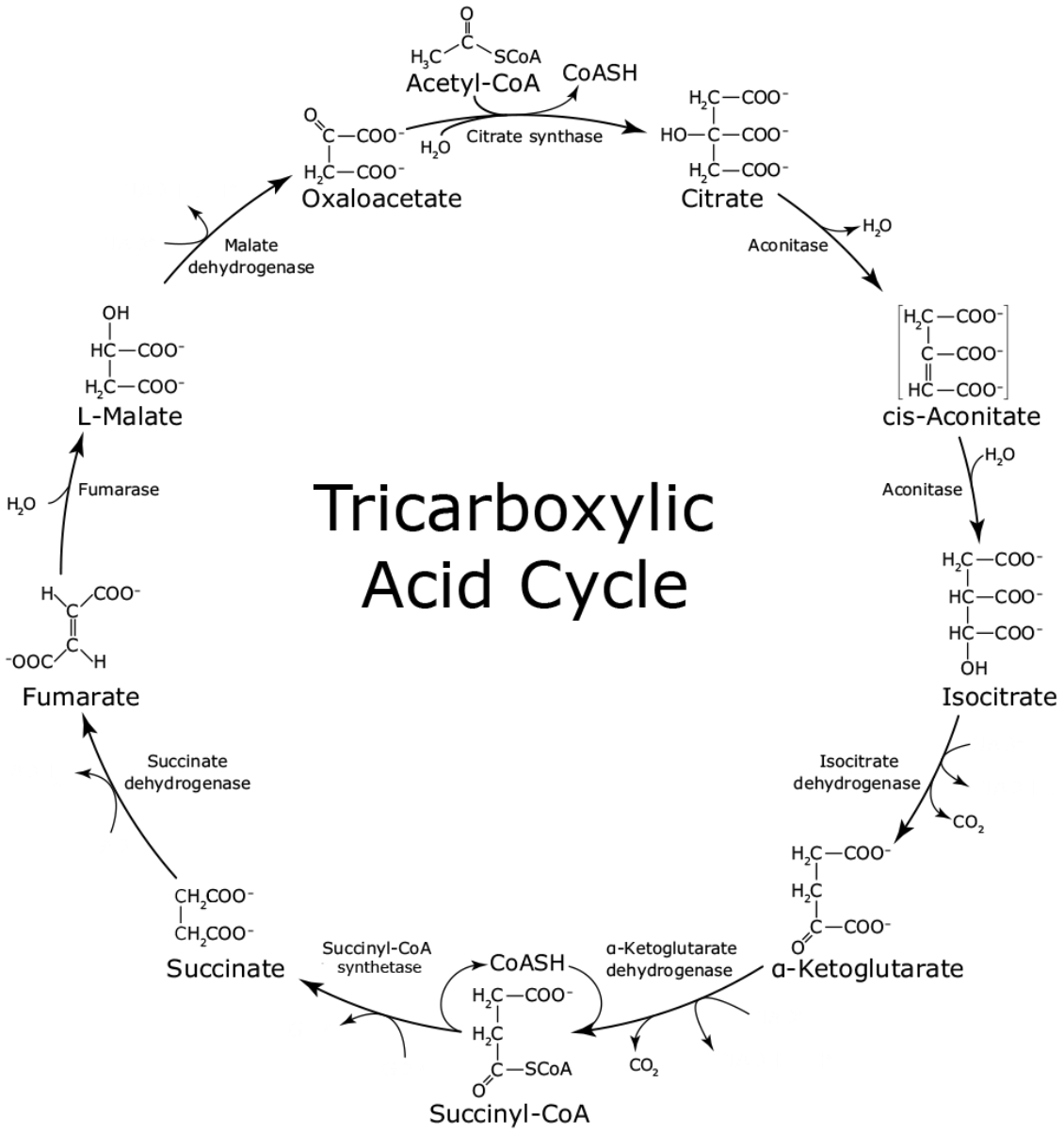
Remember the name of the irreversible enzymes like phosphofructokinase

Fermentation:

Alcoholic and Lactic acids fermentation and their enzymes,ATP calculation

Pyruvate oxidation(must read all the things)

TCA cycle(krebs cycle)



Must remember the whole krebs cycle

Succine dehydrogenase is a inner mitrochondrial transmembrane protein

ATP Calculation is the most important portion of the TCA cycle (most important one)

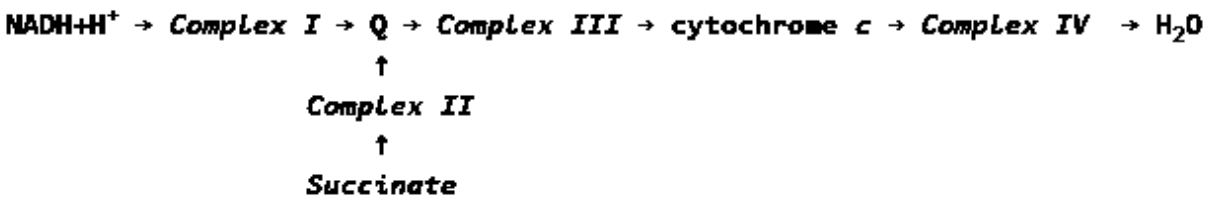
Oxidative Phosphorylation

The Protein Components of the Mitochondrial Electron-Transfer Chain

Protein complexes	Number of subunits	Prosthetic group
I NADH Dehydrogenase	14	FMN,Fe-S
II Succinase Dehydrogenase	4	FAD,Fe-S
III Cytochrome c oxidoreductase	12	Hemes,Fe-S
IV Cytochrome Oxidase	13	Hemes,Cua,Cub

All the complexes and their function how much H+ pump into the intermembrane space

Remember the Series of the Electron transfer in ETC(Electron Transport System)



Inhibitors of the all complexes(must)

Inhibitors	Complexes
Rotenone,Amobarbitol(Amytol),Piecidin A	Complex I
Malonate	Complex II(Succinate dehydrogenase)
Antimycin A	Complex III
Cyanide,Azide, and Carbon monoxide	Complexe IV
Oligomycin	ATP synthase (block F0 particle)
Atractyloside	ATP-ADP Translocator in the mitrochondria

Structure of the ATP Synthase complexes

Uncouplers(Remember just)

Pentose Phosphate Pathway

1.It provides Ribose sugars and NADPH to the cells.

2.NADPH is produced in Oxidative pathway

Just you have to remmber the above two things because most of the questions will be asked from that area.

GLUCONEOGENESIS

It is the biochemical pathway that convert non carbohydrates precursors into the Glucose.

It is takes place in the liver.

Above two things you must remember

Remember the cycle and specially irreversible enzymes.(go to above glycolysis picture)

ATP Calculation(must)

Regulation of the Gluconeogenesis.

Glycogenolysis

Where it occurs and what is the function of the Glycogenolysis?

Liver and Muscles.

You should remember two main enzymes names and their function-

1.Glycogen phosphorylase

2.Debranching Enzyme

If you have enough time then you must read all the cycle.

Glycogenesis

You have to name of the two enzyme and their function-

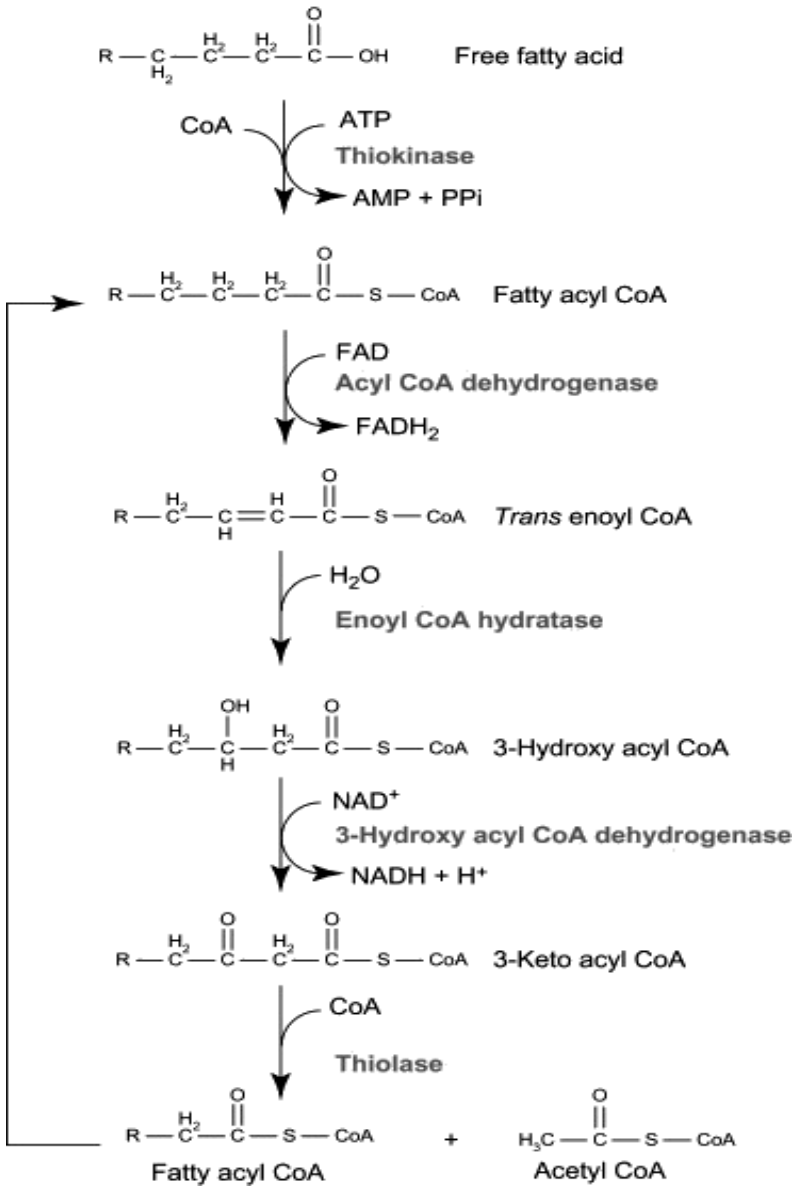
1.Glycogen Synthase

2.Branching Enzyme

For synthesis of glycogen primers are required like **existing glycogen or Glycogenin.**

Lipid metabolism

You have to just remember two pathways in fattyacids metabolism-



1.Fattyacids degradation(beta oxidation).Acyl-carnitine shuttle.

Whole pathway is very important specially ATP calculation.(most important).

How many ATPs are produced from one molecules of Palmatic acids?

Oxidation of of palmitic acid yields 7 NADH + 7 FADH2 + 8 acetyl-CoA in 7 cycles of mitochondrial beta oxidation. Every acetyl-CoA yields 3 NADH + 1 FADH2 + 1 GTP (=ATP) during Krebs cycle. Considering an average production of 3 ATP/NADH and 2 ATP/FADH2 using the respiratory chain, you have 131 ATP molecules. However you have to used 2 ATP molecules for the initial activation of every fatty acid that is going to be oxidized in the mitochondria.

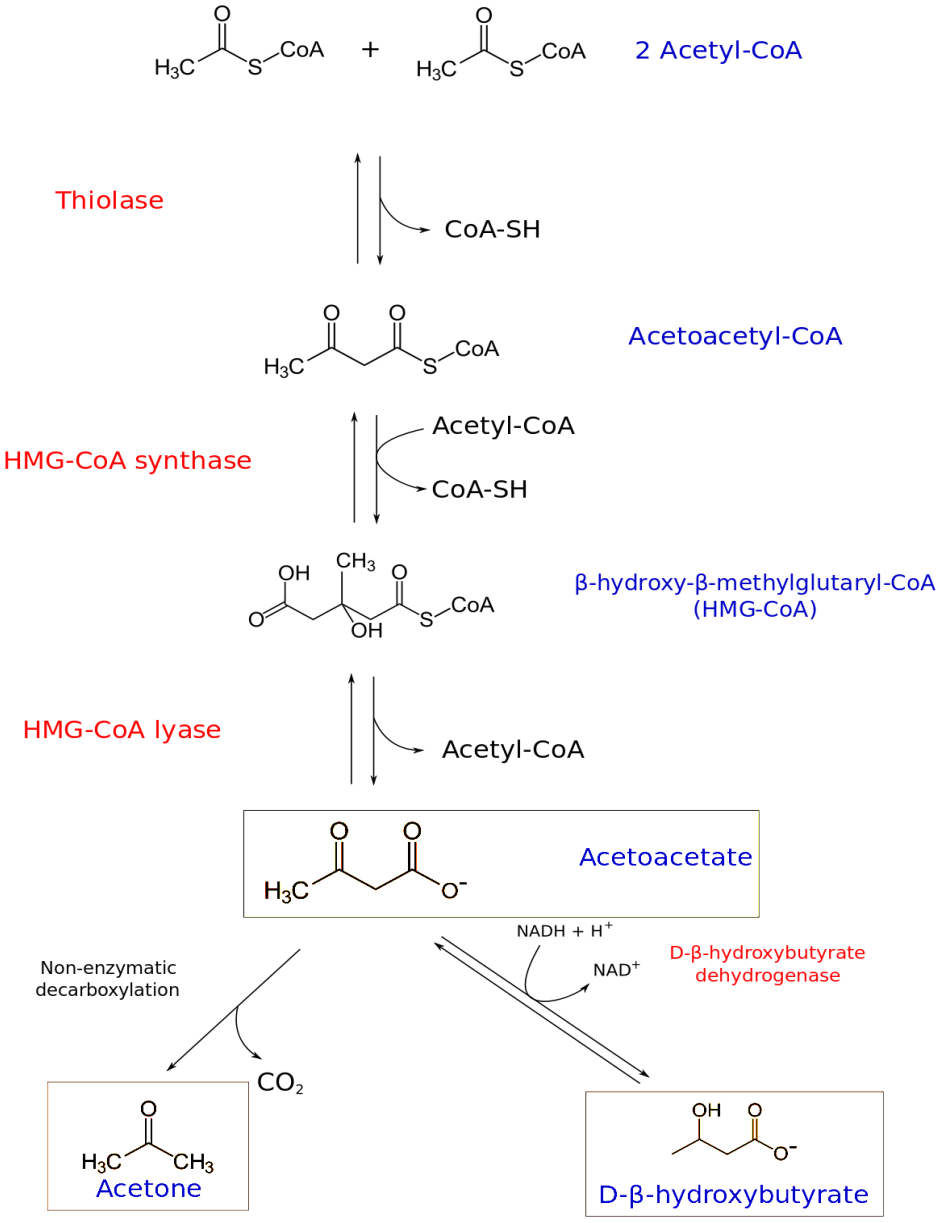
Reaction name	NADH	$FADH_2$	Total ATP
Beta Oxidation	7NADH=7*2.5=17.5 NADH=2.5ATP	7FADH=7*1.5=10.5 $FADH_2$=1.5ATP	28
Krebs Cycle	24NADH=24*2.5=60	8$FADH_2$=8*1.5=12	72
1 GTP produced each krebs cycle			8
Total ATP			108

You have follow the same formula for every fattyacids.

2.Fattyacids synthesis.

Read whole pathways and here lots of calculated question will be asked.

And the least important portion is ketogenesis.just you have to know the process.



Aminoacids Metabolism

Transamination reaction

You have to read the process of the transamination reaction and enzymes cofactor pyrodoxal phosphate.

Deamination process(read only deamination process-not so much deep)

And Urea cycle(ornithine cycle)

Nucleic acid Metabolism

Just you have to remember two pathway(not so much details) for synthesis for nucleic acids-

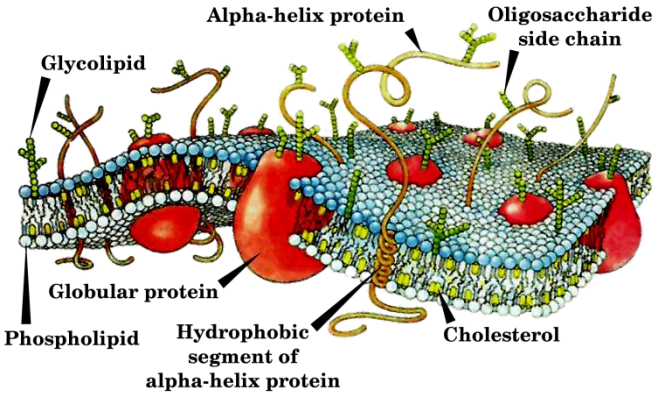
1.De-novo pathway

2.Salvage Pathway

Cell biology

Plasma membrane

Plasma membrane is made of lipids (amphiphatic molecules contains both hydrophobic and hydrophilic part) and proteins.Other components are also found like **glycolipids,glycoproteins and cholesterols**.



Plasma membrane is made up of phospholipid bilayer. Two types of phospholipids are present-

1**. Glycerophospholipid**:fattyacids joined to the glycerol molecule.

2**.Sphingophospholipids**.

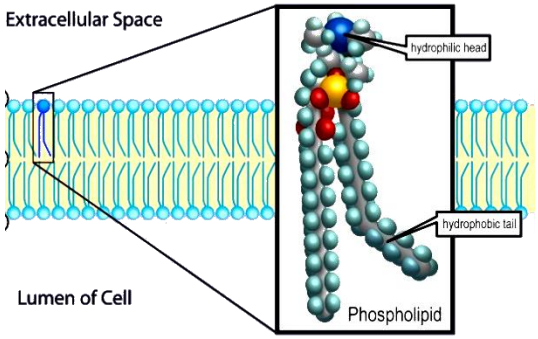
Four types of phospholipids are found-

(a)Phosphotidylcholine:Mostly abundant phospholipids.at neutral ph it has net charge is zero.

(b)Phosphotidylethanolamine:net charge zero at neutral ph.

(c)Phosphotidylserine:present inner leaflets of the of the plasma membrane and it contains negative charge.

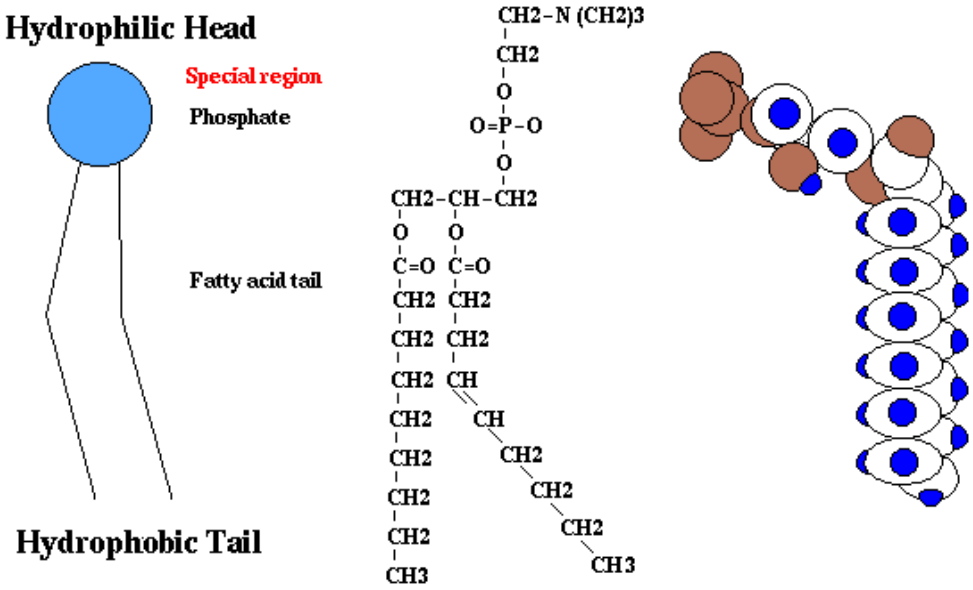
(d)Sphingomyelin:



Flippase: it is an enzyme that transport glycerophospholipids from outer leaflets to inner leaflets

Floppase: Transport glycerophospholipids from inner leaflets to outer leaflets.

Both of them work as a ATP dependent manner.



Fluidity of the plasmamembrane

Temperature,chain length and degree of saturation determine the fluidity of the membrane.

Temperature↑=fluidity↑

Chain length of the fattyacids↑ =fluidity of the plasmamembrane↓

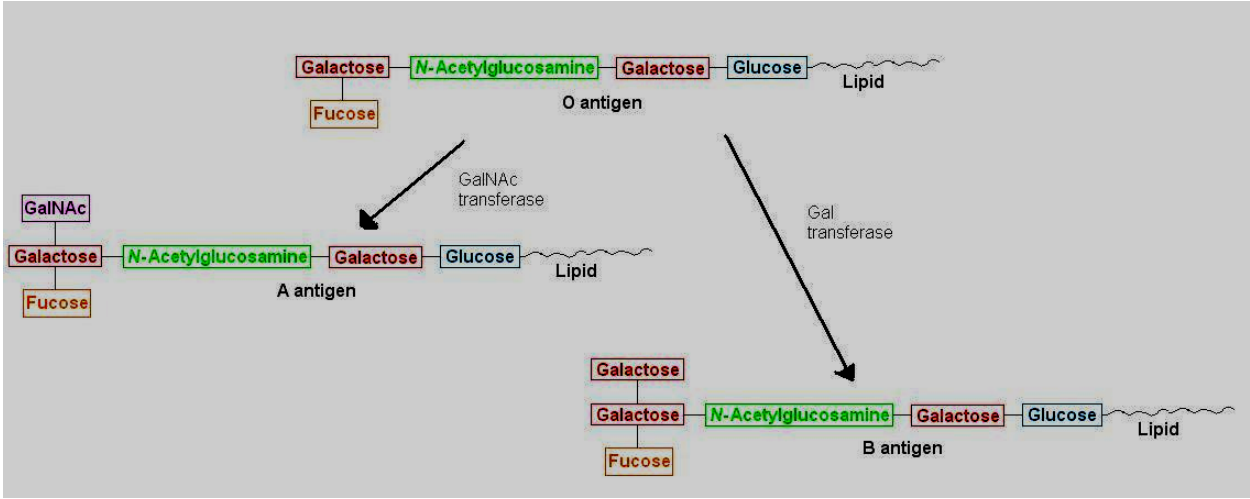
Unsaturation of the plasmamembrane↑ = fluidity of the plasmamembrane↑

ImportantTransmembrane proteins

Glycophorins:131 aminoacids single pass transmembrane RBC proteins.

Band 3 proteins present in the RBC that exchange the choride-bicarbonate ions in the plasmamembrane. 1

ABO blood groups



Permeability of the molecules across the plamsamembrane

Small gaseous molecules(O2,CO2,N2)>H2O,glycerol,urea>Glucose,sucrose>H,Na,K,Cl ions(actually Charge ions are not permeable)

Passive transport:It is occur without involement of the energy and depends on the concentration gradient of the molecules.

Remember that Band III and Glycophorin Proteins present in the

(i)simple diffusion

(ii)Faciliated diffusion:it contains all the carrier mediated transporter.the Most importants transporters are-

GLUT-glucose transporters:

It is a 12 transmembrane proteins and transports glucose molecules across the plasmamembrane.

GLUT 1	Erytrocytes
GLUT2	Liver

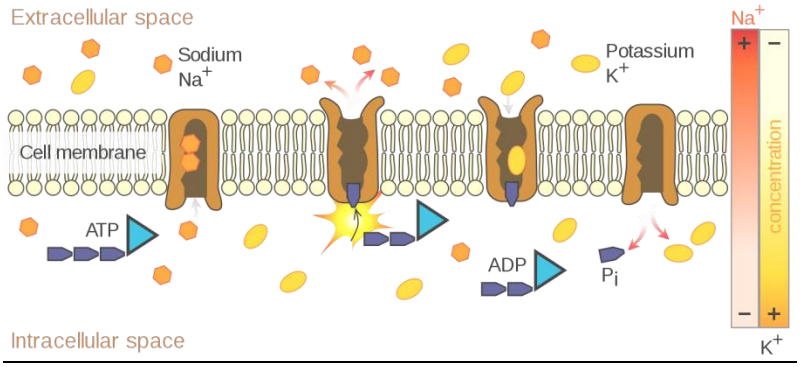
GLUT 3	Brain
GLUT 4	Metabolically active tissue like muscles it helps to transport glucose molecules in response to insulin.
GLUT 5	Small intestine

Active transport

Transport the molecules against concentration gradient and it utilize the energy like ATP,GTP,etc,.

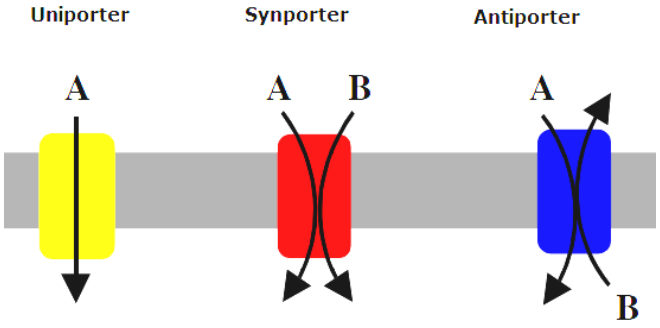
The most important transporters are

(i)Na-K pump:efflux 3 sodium ions and influx 2 potassium.



(ii)CFTR(Cystic Fibrosis Transmembrance Conductance Regulator):it is transport Cl ions when it is nonfunction cystic fibrosis is occur.

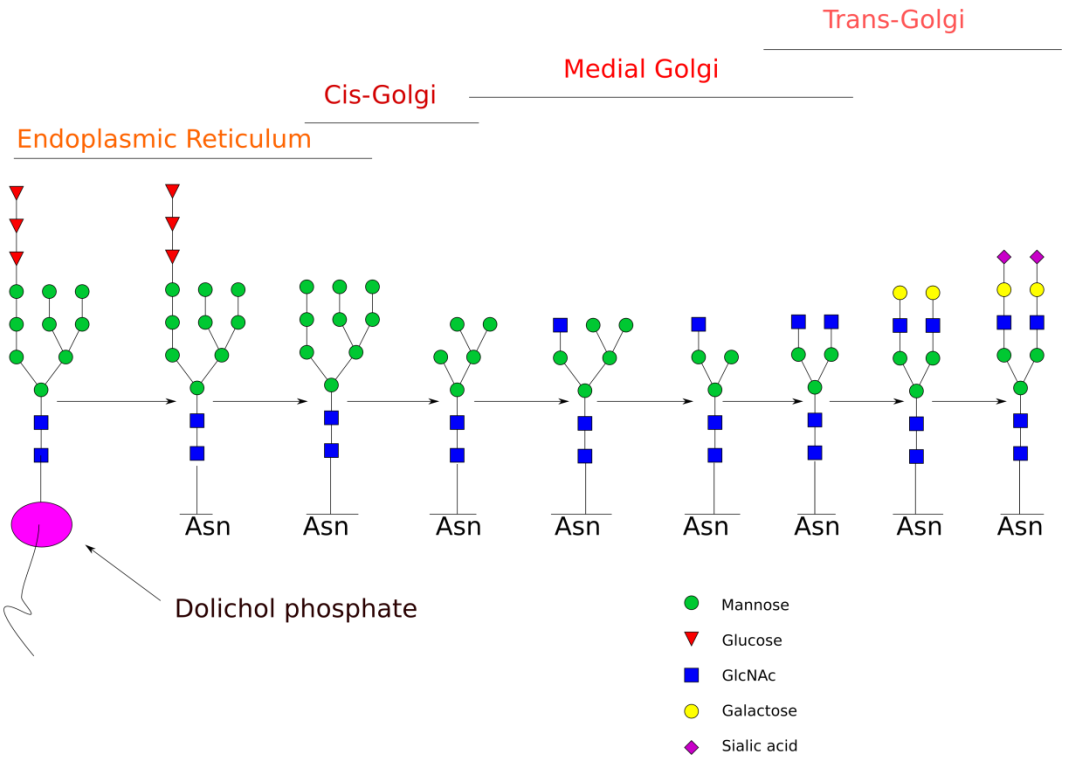
Secondary active transport



N-linked gycosylation

The oligosaccaride chain=Dolicol phosphate-2 N-acetylglucosamine-9 Mannose-3 Glucose

The oligosaccaride chain is transfered to **the Asn-x-Ser/Thr region** of the protein by oligosaccaride protein transferase.where x is any aminoacids except proline.



Remember one thing O-linked glycosylation occur in the golgi apparatus and N-linked gycosylation occur in the Endoplasmic reticulam.

Name of the chaperone(protein folding machinery) of the ER(endoplasmic reticulam).

Bip,calnexin and calreticulam.

Disulphide bonds can be formed in the ER by PDI(protein Disulphide Isomerase)

You should remember the two things which are

1.RER(rough endoplasmic reticulam):protein is synthesize here.

2.SER(smooth endoplamic reticulam):which is resonsible for the detoxification it is known as xenobiotics.

Golgi apparatus

It is a part of the endomembrane system.it processes the majority of the proteins and add lots of carbohydrades residue to the protein at specific site.**Brefeldin A (BFA)** is a fungal metabolite used experimentally to disrupt the secretion pathway as a method of testing Golgi function.BFA blocks the activation of some ADP-ribosylation factors (ARFs).ARFs are small GTPases which regulate vesicular trafficking through the binding of COPs to endosomes and the Golgi.BFA inhibits the function of several guanine nucleotide exchange factors (GEFs) that mediate GTP-binding of ARFs

Vesicles Coat proteins:it is a special type protein which helps to target the appropriate protein to the accurate destination in the intracellular enviornment.

Coated vesicle	Coated proteins	Transport proteins
Clathrin	Clathrin,Ap1	Trans golgi to endosome
Clathrin	Clathrin,AP2	Plamamembrane to endosome
COPI	COPI	Golgi to golgi and golgi to ER
COPII	COPII	From ER to Golgi

Sorting signals for organelles

Signal sequence	Type of proteins	Transport steps
Lys-Asp-Glu-leu(KDEL)	Secreted proteins	Signal for transport of proteins from golgi to ER
Lys-Lys-X-X(KKXX) where x is any amino acids	Membrane proteins	Golgi to ER
Diacidic	Membrane proteins	ER to golgi
Mannose 6-Phosphate	Secreted proteins	Signal for lysosomal proteins
PTS 1(Peroxisomal Transport Proteins)-SKL(Ser-Lys-leu) signal is present in the C-terminal of the protein.	Resident proteins	Peroxisomal resident protein
MTS(mitrochondrian Target sequence) at the N-terminal of	---------- It is depends on the sequence of	Mitrochondrial proteins

the proteins.	the signal	
Thylakoid Targeting Sequence	It is depend on the proteins	Chloroplast proteins

Organelles and their important enzymes

Every organelles have special type of enzymes that perform a specific function.This kind of question also asked in all entrance examination.

Organelle	Specific enzyme	Function of the enzyme
Endoplasmic reticulum	PDI(Protein Disulphide Isomerase)	Add disulphide bonds to the proteins
Golgi apparatus	Glycotransferase	Add monosaccarides to the proteins
Lysosome	Hydrolases enzyme like cathepsin	Degradation of majority of the molecules
Mitrochondria	All kreb cycle enzmes and cytrochrome	Convert acetyl CoA into the H20 and Co2
Chloroplast	RuBisco	Co2 +H20 is converted into glucose
Peroxisome	Catalase	$2H_2O_2 \longrightarrow 2H_2O + O_2$

Cytoskeletons

Three types of cytoskeletons are present in the eukaryotic cells-

1.Microtubules

2.Actin filament

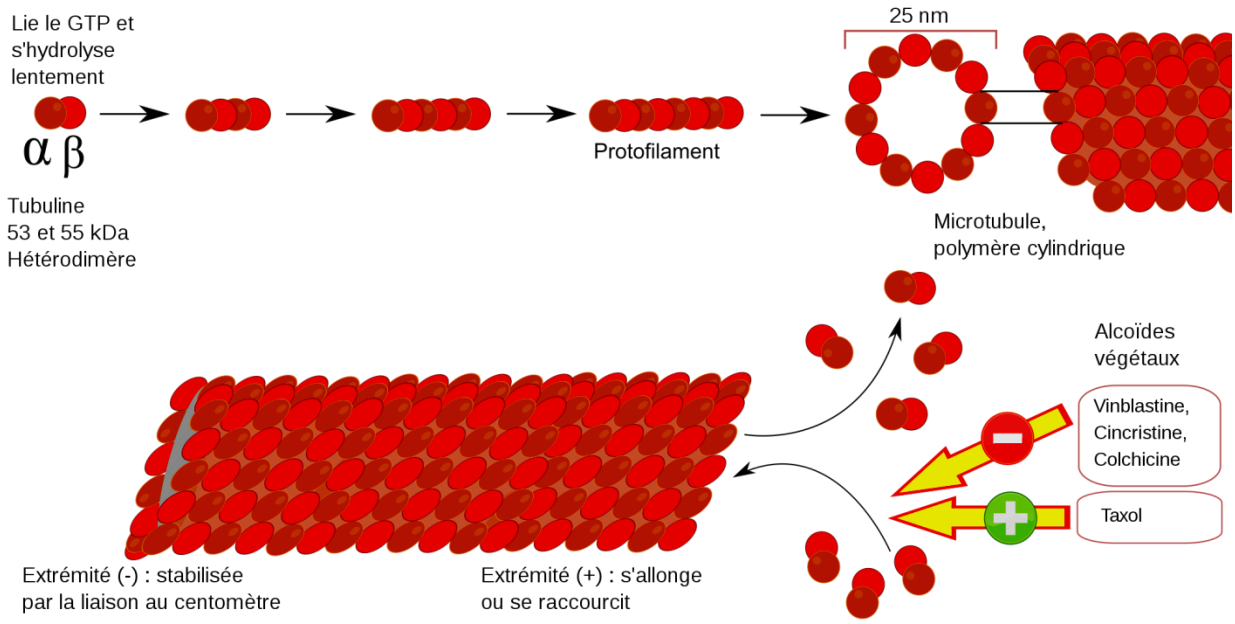
3.Intermediate filament

Microtubules

It is a hollow,cylindrical structure,25nm in diameter. It is made up of alpha and beta tubulin dimer. Microtubules use GTP for polymerization. **MAP(Microtubules Associated Proteins)** helps for polymerization of microtubules. It is dynamic in nature.

Microtubules are made up of 13 protofilament and it is nucleating from MTOC(Microtubule Organizer Centres) like centrosome.

It is transport the protein molecules(proteins) in the intracellular environment act like a highway.



Microtuble organizing and associated proteins :-

Proteins	Site of binding	Features
Gama tubulin ring complex	Minus end	Nucleates the microtuble from centrosome .
MAPs	Lateral sides.	Stabilizes the filament.
XAMP215	Plus end	Stabilizes the filament .
Kinesin 13	Plus end	Induces catastrophe, disassembly .
MAP2	Lateral side	Crosslinks the filament ad

		stabilizes.
Tau	Lateral side	Also croslinks and stabilizes the filament .

Actin filament(microfilament)

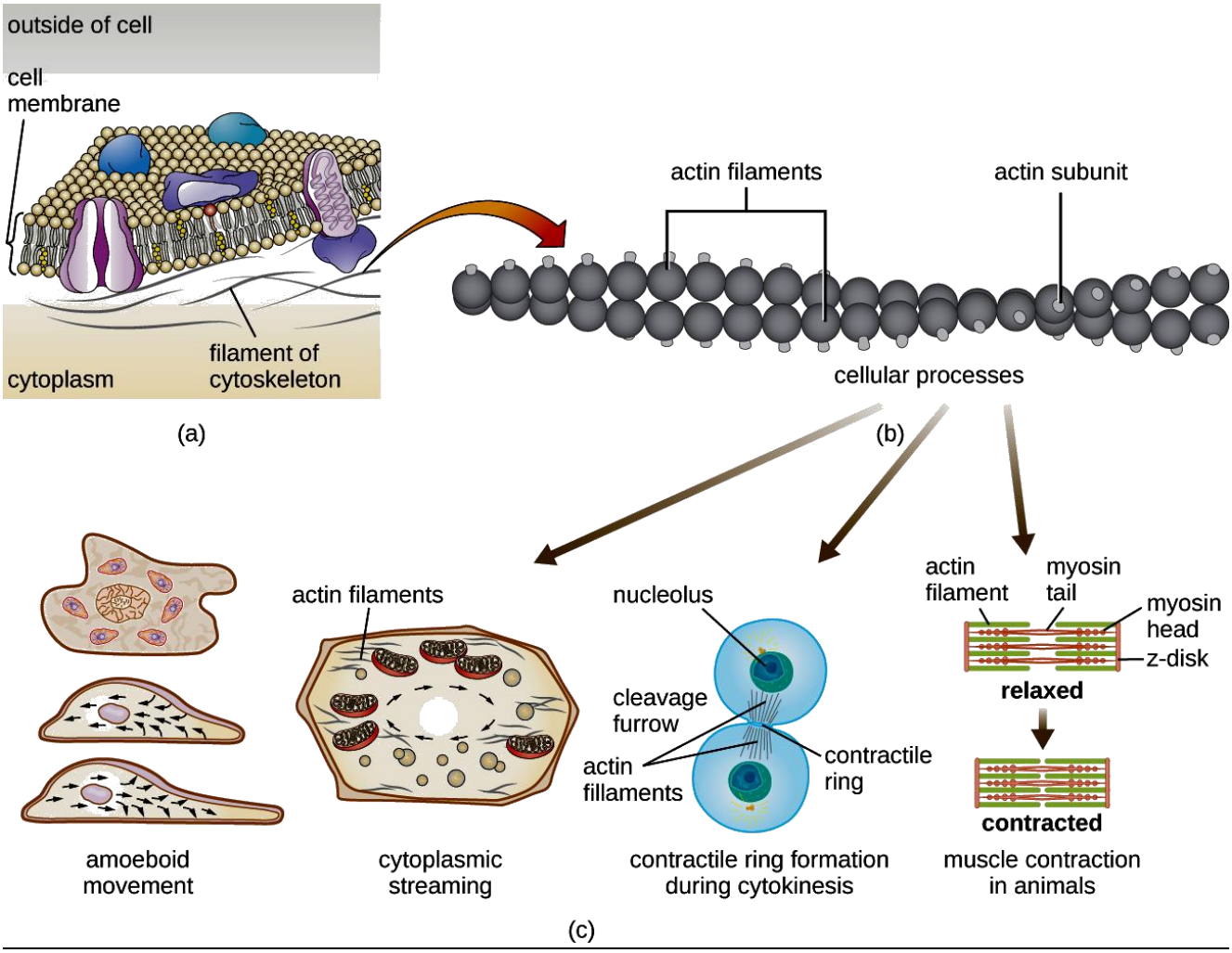
Actin filament is made up of **G-actin**. G-protein is a monomeric globular protein and it is act as a ATPase. It uses ATP instead of GTP for polimerization.G-actin polymerize and form F-actin, two actin twisted to each other to form actin filament. It is 7nm in diameter.

Both microtubules and actin filament have polarity means plus end and minus end, but intermediate filament does't have polarity.

Actin filamentorganizing and associated proteins :-

Proteins	Site of binding	Features
Formin	Plus end	Nucleates the aactin filament.
profilin	Actin subunits	Elongates the filament .
Thymosin	Actin subunits	Prevents assembly.
cofilin	ADP containing filament subunits	Accelerates the disassembly .
Arp2/3	Miunus end of filament	Form actin web by nucleating branch .
Spectrin	Filament	Crosslinks and attaches the filament to the plasma membrane .
Fimbrin	Filament	Stabilizes and crosslinks.
Alpha actinin	Filament	Croslinks and stabilizes.

Filamin	Lateral side of filament	Crosslinks and stabilizes the filament.



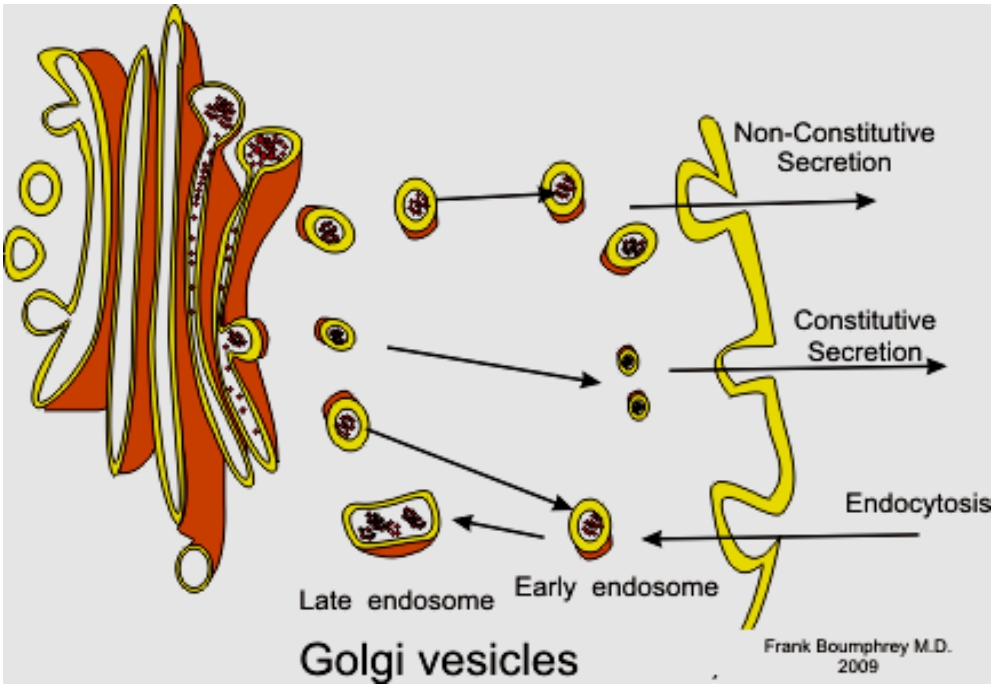
Inhibitor of the cytoskeletons(it is very important for all exams)

Actin filament

Cytochalasin D	Binds to the plus end of the filament and prevent elongation.
Latrunculin	Bind G-actin monomers and prevent them from polymerization.
Phalloidin	Binds with the actin filament and prevent from depolymerization.

Microtubules inhibitors

Colchicine	**Binds with tubulin subunits and prevent polymerization.**
colcemid	Binds with tubulin subunits and prevent polymerization
Nocodazole	Binds with microtubules and depolymerize them
Taxol	Binds with microtubules and stabilize them.



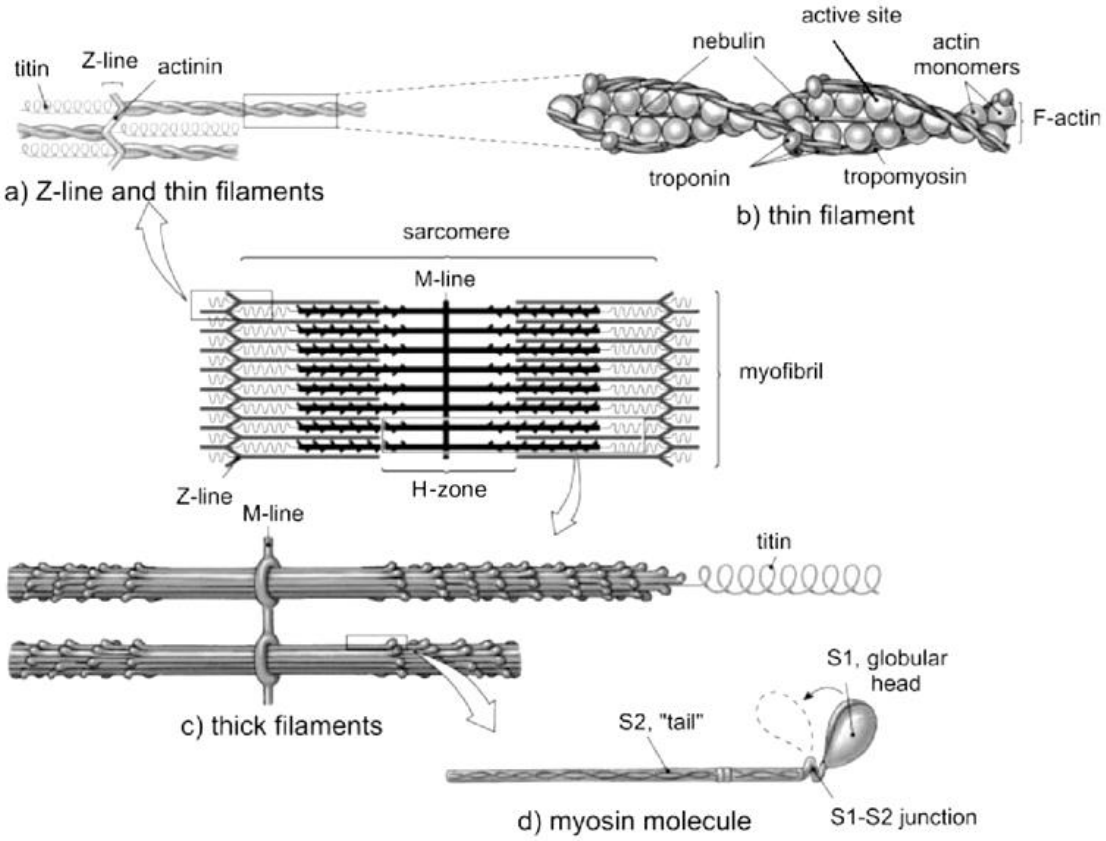
Major Muscle Protein

Muscle protein	Function
Actin	Major components of thin filament
Myosin	Major components of thik filament
Tropomyosin	Binds along with length of the thin filament
Troponin	Regulate calcium contraction of muscle fibre
Titin	Links thik filament to the Z line
Nebulin	Links thin filament to the Z line and stabilize the thin filament
Myomesin	Myosin binding protein pesent at the M line of the thick filament
Alpha Actinin	Bundles actin filament and attach them to Z-line
CapZ	Attach actin filaments to the Z line;caps actin
Tropomodulin	Maintain thin filament length and stability

MIcrotuble motor proteins :-

Kinesin -14	**minus end directed , contian single motor domain.**
Kinesin-5	Plus end directed , contain two motor domain.
Kinesin-4,10(chromokinesin)	Plus end directed , contain single motor domain.
Kinesin-2	Plus end directed , containn single motor domain.
Dyenin	Minus end directed , single motor domain.

Diagrams of sarcomere



Intermediate Filament:

Intermediate filament is a alpha helical rod shaped filament that provide mechanical streangth to the cells.Four types of Intermediate filament are found-

Class	Intermediate proteins(IF)	In which cells it is found
I	Type I, Acidic keratins	Epithelia
II	Type II, Basic keratins	Epithelia
III	Vimentin	Mesenchyma
	Desmin	Muscle

IV	Neurofilament proteins L,M,H	Central nervous system
V	Lamin A,B,C	Nucleus
VI	Nestin	Neuroepithelial cells

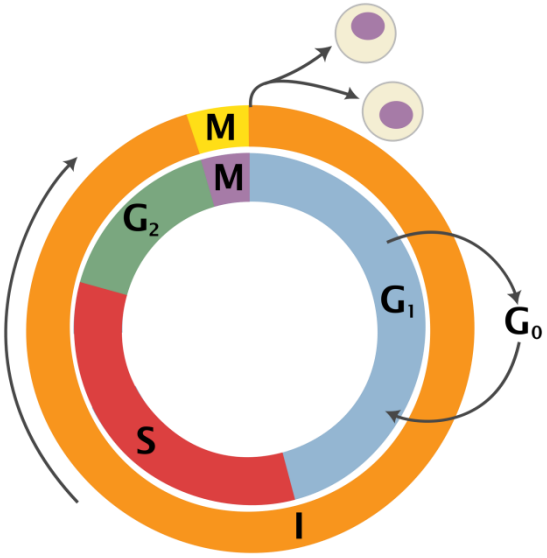
Intermediate filament associated protein :-

protein	Binding site	Features
Plectin	Crosslinks the filament	Stabilizes the filament

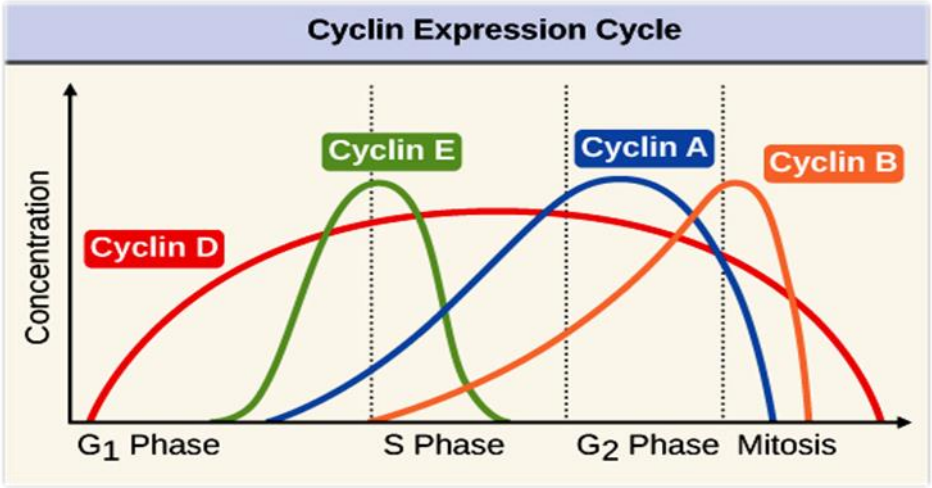
Severing proteins :-

Microuble	Katasin
Microfilament	Gelsolin

Cell Cycle

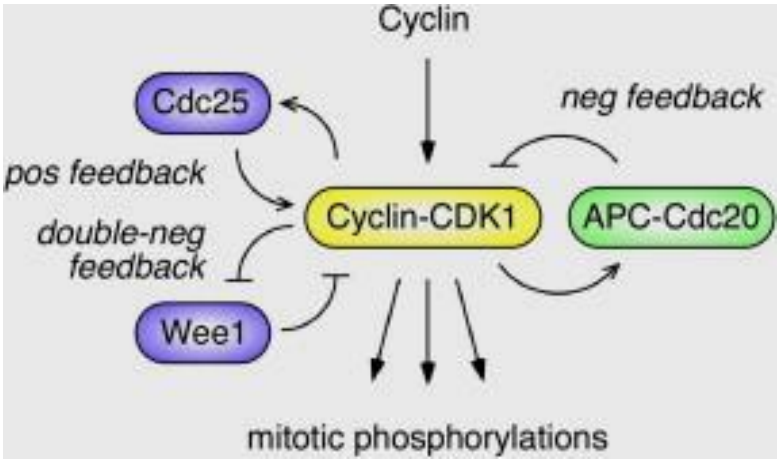


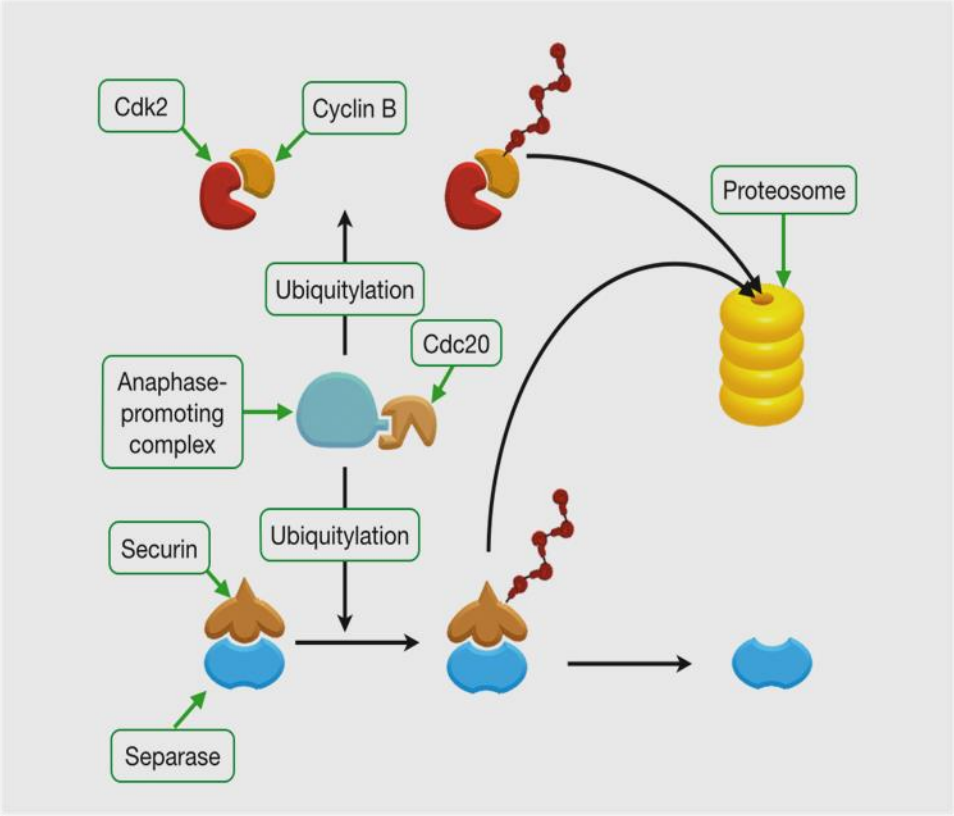
Phases	Vertebrate Cyclin	Vertebrate CDK	Yeast Cyclin	Yeast CDK
G1CDK	**Cyclin D**	**CDK4,6**	**Cln 3**	**CDK1**
G1/S CDK	**Cyclin E**	**CDK2**	**Cln1,2**	**CDK1**
S CDK	**Cyclin A**	**CDK2**	**Clb5,6**	**CDK1**
M CDK	**Cyclin B**	**CDK1**	**Clb2,3,4**	**CDK1**



Regulators	Function
CAK kinase	Activates CDKs
Cdc25 phosphatase	Activates CDKs
Wee1 kinase	Inhibits CDKs
Cdc14 phosphatase	Activates Cdh1 to degrade mitotic Cyclins
Sic 1	Binds and inhibits S phase CDKs
CKIs (P^27 , P^21)	Binds and inhibits CDKs
INK4	Binds G1 CDKs and inhibts
Rb	Binds E2F transcription activator and inhibits .
SCF	Degradation of Sic 1 and CKIs
APC/C^Cdc20	Degradation of securin , initiate Anaphase
APC/C^Cdh1	Degradation of M cyclin and Geminin and allow helicase loading on replication origins .

Regulation of the Wee,cdc25 and cyclin-CDK1





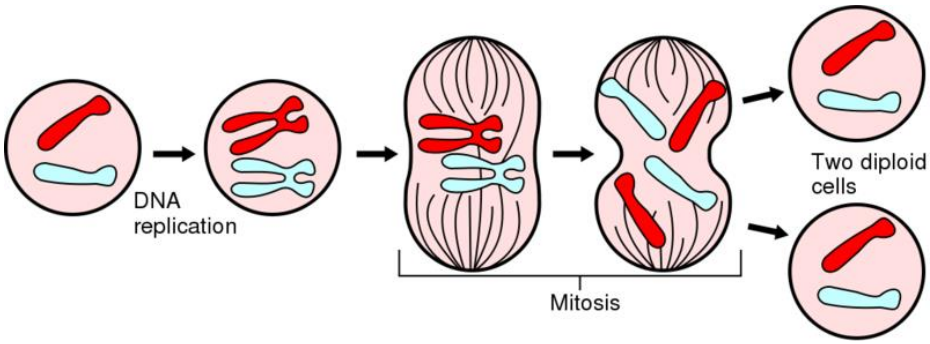
Characteristics of the all cell cycle phases

Interphase	Features
G0	Restriction point
G1	Cell growth
S	DNA synthesis
G2	Cell gowth before entering Mitosis .

Mitosis cell division phases and their special features

Mitosis	Features
Prophase	**centrioles divide , asters formation kicks in , nuclear envelope disintegration starts, chromosomes condense.**
Metaphase	**chromosome acttached with microtubles and line up to form metaohase plate.**
Anaphase	**chromosome separation takes place and pole ward movement take place.**
Telophase	**nuclear envelope reformation start, chromosome dissolve again .**

Simple diagram of the cell division



All check points(it is very important for all entrance examinations)

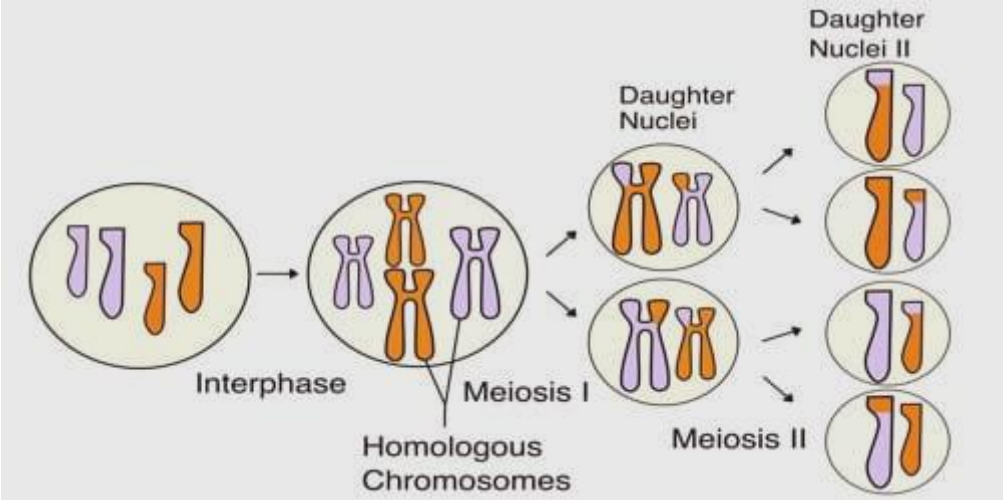
Checkpoints	Features
G1 checkpoint	Checks proper growth of cell , if not then cell goes to G0 phase .
S Checkpoint	Checks proper DNA synthesis in order to progress in cell cycle .
G2/M Checkpoint	Checks proper growth of cell , if it ready to enter or not .
M checkpoint	Checks cell proper spindle attachment with chromosome also Known as spindle checkpoint .

All cell divisions and their Special characteristics(a lots of question will be asked from this section so read carefully)

Mieosis	*Features*
Prophase 1	
Leptotene	The appearance of the chromosomes at this stage is like ‘‘a string with beads.’’
Zygotene	synaptonemal complex formation occur .
Pachaytene	crossing over takes place . Here synaptonemal complex formation complete.
Diplotene	chromosome migrate from the synaptonemal complex , chiasma formation takes place .
Diakinesis	Nucleolus disappears , Nuclear envelope disintegrates and centrosome move to the equator . Chiasma separation takes place .
Metaphase 1	bivalents align at the equatorial plate
Anaphase 1	two chromosomes of each bivalent separate and move to the opposite ends of the cells
Telophase 1	nuclear membrane reappears
Cytokinesis	Cytoplasmic divison takes place , gives rise to two cells (2n) each .
Prophase 2	Nuclear membrane disintrigation starts .
Metaphase 2	Metaphase plate of chromosome occur.

Anaphase 2	Pole ward movement of chromosome occur .
Cytokinesis 2	Again cytoplasmic division takes place giving 4 haploid (n) cells .

What happened if aneuploidy occurs at meiosis I and meiosis II



Chromosome numerical aberration :-

Non Disjunction at Anaphase 1 Chromosomes	
Euploidy	2n-n
polyploidy	2n+n , 2n+2n
Atopolyploidy(same species chr.) Allopolyploidy (different species chr)	
Non Disjuction at Anaphase 2	
Monosomy	2n-1
Trisomy	2n+1
Nulisomy	2n-2
Tetrasomy	2n+2

Questions:

Suppose a organism contains 46 chromosome in its cells .How many chromosome should be present in mitosis and meiosis stages ?

Ans :-

Mitosis	Chromosome no.	Chromatids
Prophase	46	92
Metaphase	46	92
Anaphase	92	92
Telophase	92	92
End of mitosis , Separated cells	46	46

Meiosis	Chromosome no.	Chromatids
Prophase 1	46	92
Metaphase 1	46	92
Anaphase 1	46	92
Telophase 1	46	92

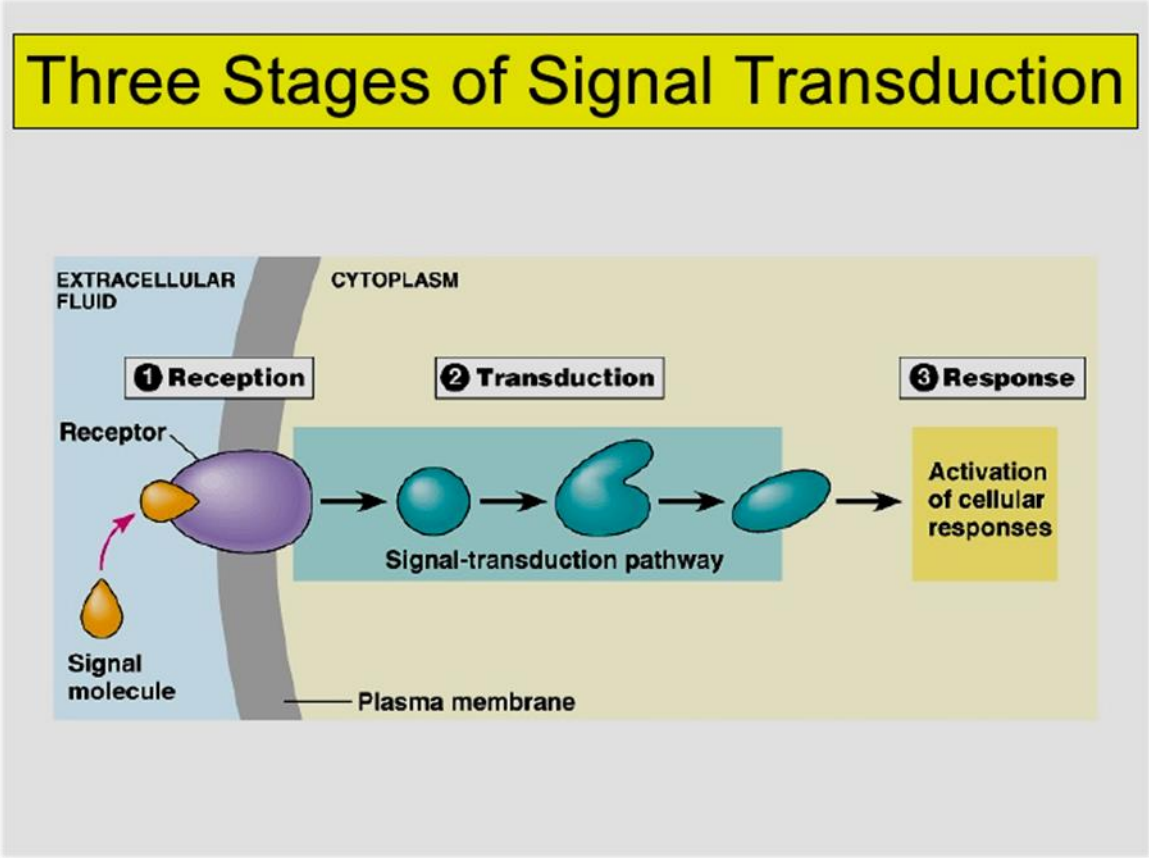
End of meiosis 1 **Separated cells**	**23**	**46**
Prophase 2	**23**	**46**
Metaphase 2	**23**	**46**
Anaphse 2	**46**	**46**
Telophase 2	**46**	**46**
End of meiosis2, **Separated cells**	**23**	**23**

Important Points:- The number of chromosome depends on the number of centromere ,

However, in meiosis I anaphase 1 the chiasma separation occur between non sister chromatids, no centromere separation occur thats why centromere number does not get doubled for this reason chromosome number remains the same in anaphase 1 .

In Anaphase 2 the centromere separation takes place leading to form double number of chromosome 46 . at the end of meiosis each cell give rise to 2 cells containing 23 chromosomes forming 4 haploid cells .

Cell signalling



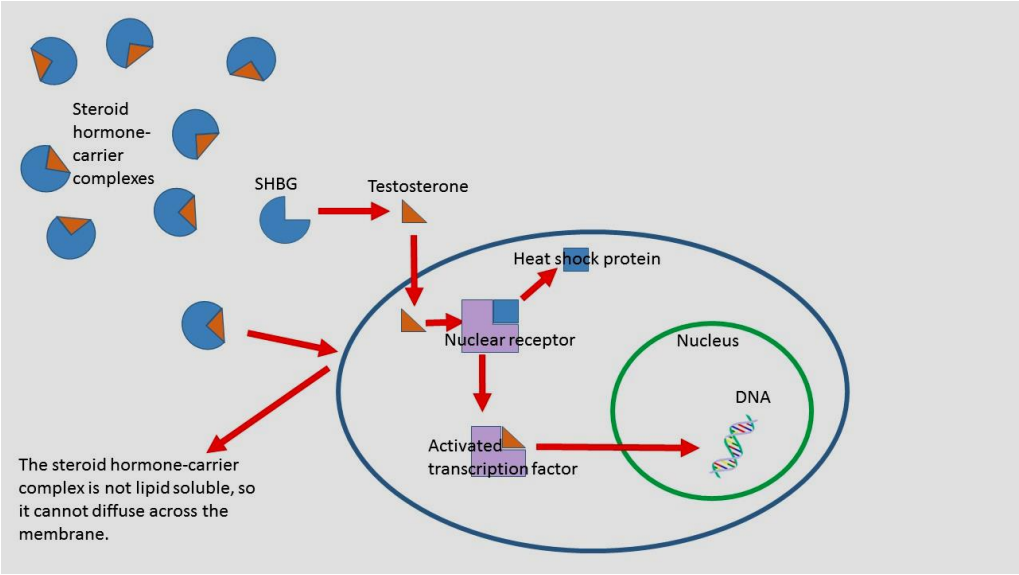
Cell signallig process:-

Recognizing the signaling molecule (ligand)hence known as reception.	**Transducing the signal by amplifying, hence transduction .**	**Finally activation of certain genes, hence response.**

The way of transmitting signalling

Autocrine	**Cell secrete a chemical messenger that binds to autocrine receptor of that particular cell.**
Paracrine	**A cell secrete a hormone that binds to the receptor of nearby cell .**
Endocrine	**A cell secrete a hormone that circulate in blood and binds to receptor of a far away cell .**
Juxtacrine	**Here , cell interact with receptor proteins of adjacent cell , this is a contact specific signalling .**
synaptic	**Neurotansmitter released at synaptic terminal and transmit the signal to the next neuron .**
Gap juction	**Here a gap present between two cells** **(connexin formed the gap) and signal can transfer through it .**

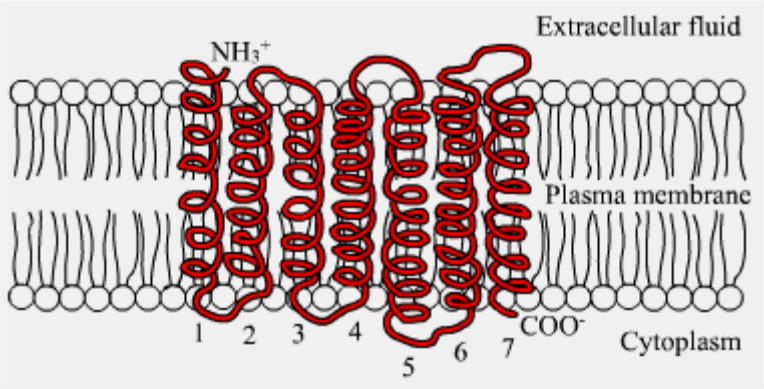
Steroid hormone Nuclear receptor signaling :- steroid hormone hydrophoic in nature can pass through the plasma membrane and gets bind with nuclear receptor.



Steps of signaling :-

1. **Steroid hormone passes the membrane .**
2. **Replaces the heat shock protein from the nuclear receptor .**
3. **Binds with the receptor , and act as a trnascription factor .**
4. **Active the certain genes .**

GPCR signaling:- is heptahelical receptor with an attached G protein , also known as serpentine receptor for water soluble hormones (adrenaline , vassopressin , etc.) and drugs .



cAMP signal pathway	Phosphatidylinositol signal pathway
1. Ligand binds to the receptor .	1. Ligand binds to the receptor .
2. G-alpha subunit of G protein ,dissociate from G-beta and G-gamma subunit of GPCR .	3. Alpha subunit of g protein dissoviate from beta and gamma subunit of G protein .
4. G alpha activates adenylate cyclase .	2. G alpha activate PLC.
5. Adenylate cyclase convert ATP into cAMP .	3. Phospholipase cleaves the phosphotidylinositol into IP3 and DAG .
6. cAMP activate protein kinase C .	6 . IP3 binds Ca+ channel of ER release the Ca+ into the cytosol .
7. PKC ultimately activate the CREB .	8. DAG binds with the membrane .
9. This act as a transcription activator .	9 . both DAG and Ca2+ activates PKC. Leading to active CREB .
10. Gene expression induced .	10 .Finally gene expression occur.

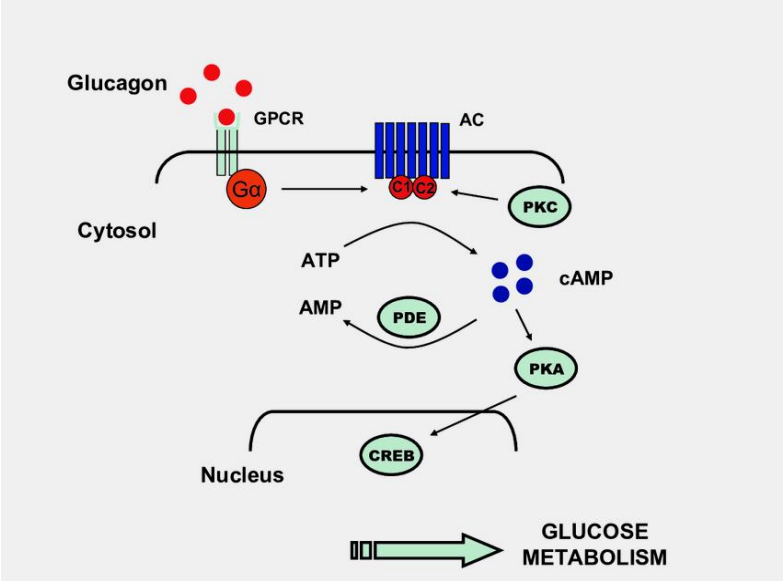
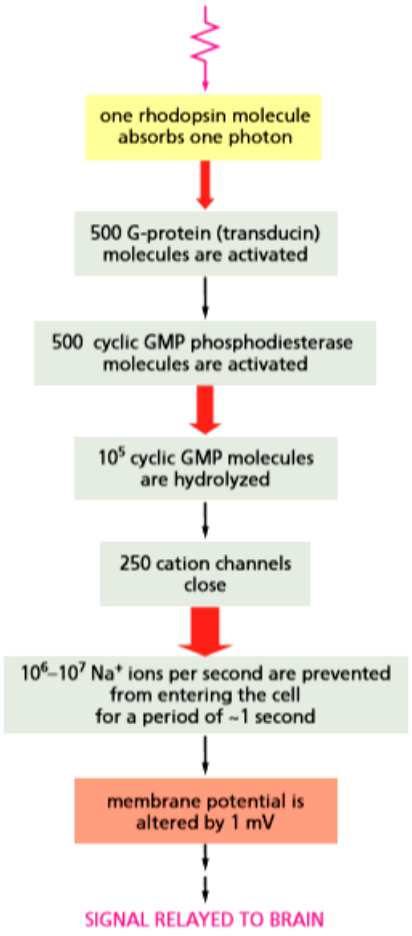


Fig :- GPCR cAMP signaling pathway

Family members	Action mediated by	Function
G_s	alpha	Activate adenylyl cyclase and Ca channel
G_{olf}	alpha	Activate adenylyl cyclase in olfactory sensory neurons
G_i	alpha	Inhibit adenylyl cyclase
G_o	Beta and gamma	Activate potassium channel
G_t	Alpha	Activate cyclic GMP phosphodiestarase
G_q	Alpha	Activate phospholipase C-beta

Rhodopsin Singnaling:

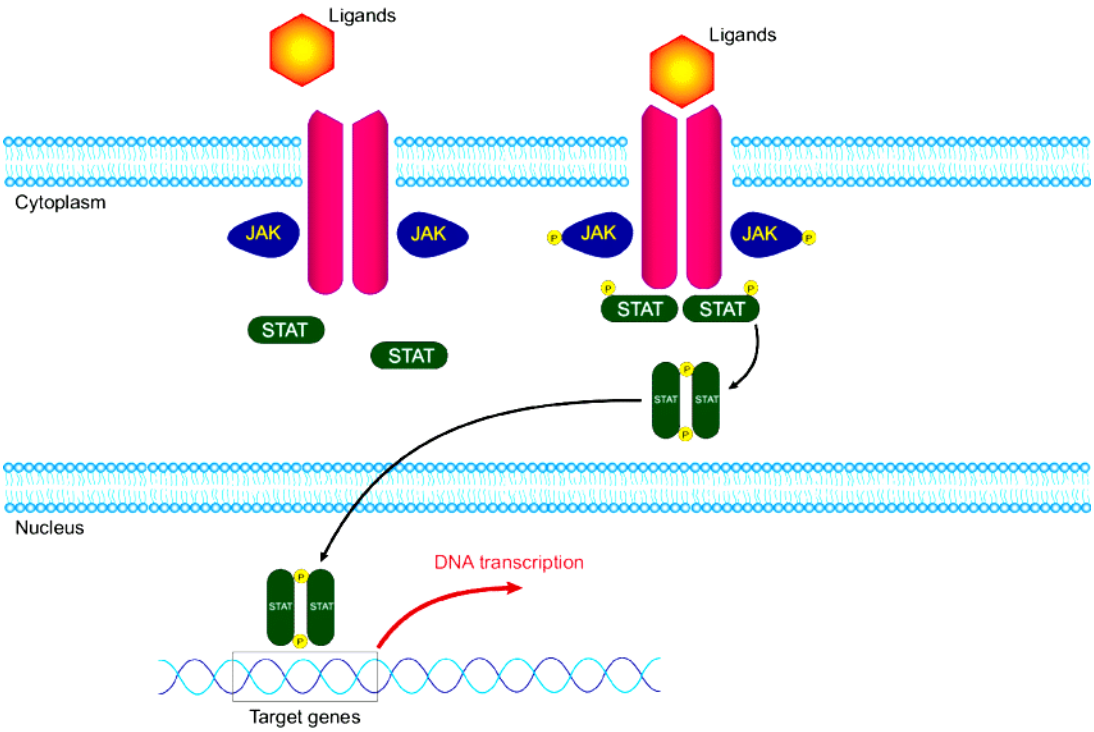


Cholera Toxin: Cholera Toxin is protein which is secreted from *Vibrio cholerae* bacteria. A subunit of the Cholera Toxin Add ADP-Ribose to the G protein. For that reason G_{GTP} Proteins no longer Hydrolysed and constantly High amount of the Na+ effluxes going on to the intestine lumen.(read it carefully from the books).

You should read **Pertussis Toxin** also from any sources because question will be asked from that area.

JAK STAT signaling :-

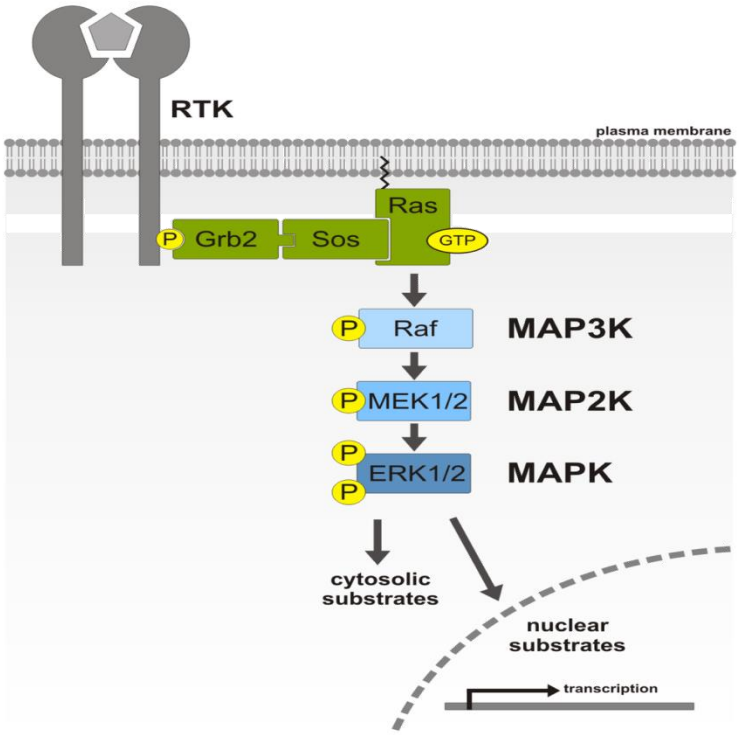
JAK - janus kinase , STAT - signal transducer and activator of transcription of proteins . cytokines (interlukins , interferons) play their roles through this pathway .



Steps of the pathway :-

1. Ligand (cytokines) binds to the receptor .
2. JAK gets phosphorylated at specific tyrosyine by trnasphosphorylation .
3. Active JAK then phosphorylate the SH2 domain of recptor .
4. Then STAT get attached with phosphorylated SH2 domain .
5. Then STAT residues gets phosphorylated and dissociates from the recptor .
6. Phosphorylated STAT form dimer and goes to the nucleus .
7 . Activate transcription of certain genes .

RTK pathway :- receptor tyrosine kinase. It also known as Ras - Raf pathway. Varous Growth factor (NGF, PDGF FGF) , Insulin hormone , insulin like growth factor use this patway .



Steps of the pathway :-

1. Ligand bind to the RTK receptor .

2. Phosphorylated receptor cyctoplasmic domain binds with adapter molecules (Grb2 , sos, Ras).

3.GTP bound Ras can relay the signal by phosphorylating the Raf also know as MAP kinase kinase kinase then several phosporayakation takes place .

4. Raf phosphorylate MEK and then activates ERK by phosphorylating it .

5. This ERK acts as a transcription activator leads to expression of certian genes .

Cell junction

Cell junction	Main Proteins	Function
Tight Junctions(zonula occludens)	Cluadins and occludin	Seal the apical surface of the Epithelial cells and prevent the free passage of the ions and water molecules. Maintain the polarity of the epithelial cells.
Adherens Junction	Cadherens proteins(calcium dependent)	Helps in cell to cell junctions
Desmosomes	Plakoglobin and desmoplakin(intracellular protein) Desmoglein and desmocollin(Extracellular proteins)	It is cell to cell junction
Hemidesmosomes	Integrins	It is provide Cell to Extracellular matrix juncion.
Gap junction	Six Connexin proteins combine to form Connexon proteins.	It helps in cell to cell communication. Help in nerve impulse conduction. Found in animal tissue. It passes ions between the cells.
Plasmodesmata	Desmotubeles are found in this cell communicating junction and found in plant cells. It is a extension of Endoplasmic Reticulum(ER)	It helps to transport small molecules and ions between the cells.

Which kind of cytoskeletons are associated with which kind of cell junction?

Cell junction	Cytoskeletons
Adherens junction	Actin filaments
Desmosome	Intermediate filament
Hemidesmosome	Intermediate filament

Cell organelle and their specific enzyme

Peroxisome

Single membrane bound organelle .

Enzyme

Catalse :- degrade H2O2 into the water and O2

Glyoxalate cycle :- here the isocitrate to succinate direct conversion occur .

Lysosome

Single membrane bound organelle . Also known as ***Suicide Bags of the cell***.

Enzymes :-

- Proteolytic (cathepsin)
- Hydrolytic (RNAse , DNAse)
- Lipolytic (lipase)

H+ ATPase pump :- maintain the low PH of lysosome .

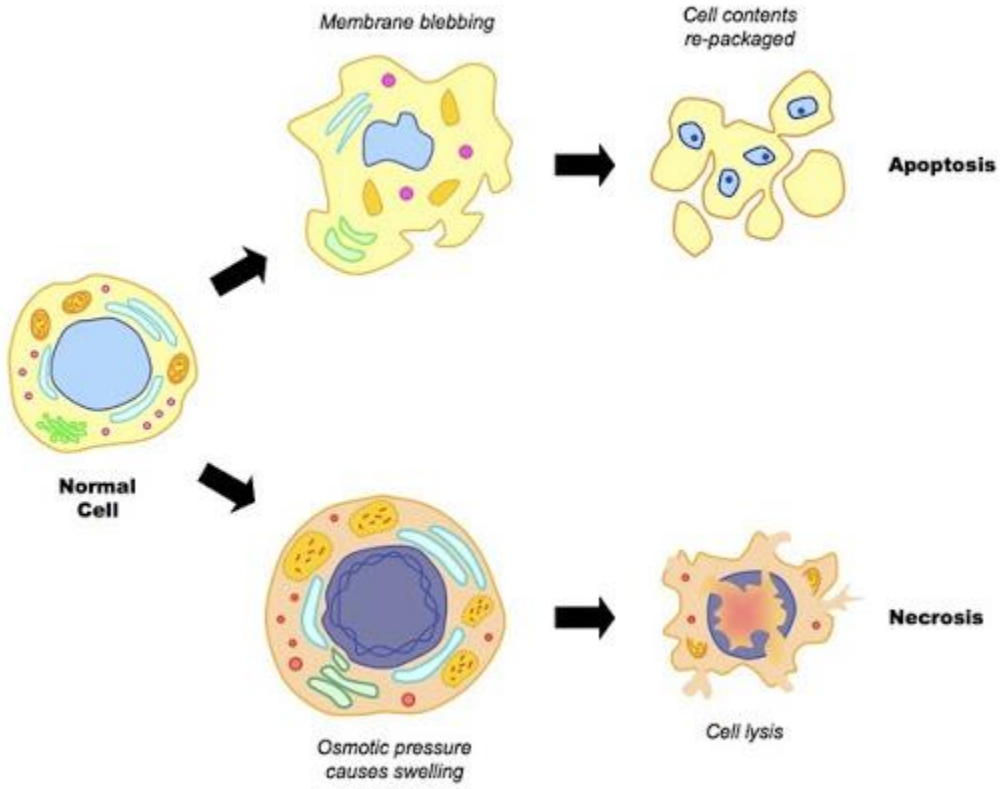
Lysosomal storage disease :- occur if the enzyme deficiency occur from gene mutation .example Tay sachs , Krebs disease etc.

Ribosome

Ribosome	Sedimentation Cofficient	rRNA Large subunit	rRNA small subunit
Bacteria	70S	5S,23S	16S
Chloroplast	70S	5S,23S	16S
Mitrochondria	55S	16S	12S

Archaebacteria	70S	5S,23S	16S
Eukaryotes	80S	5S,5.8S,28S	18S

Apotosis (Programmed Cell Death)



Functional difference :-

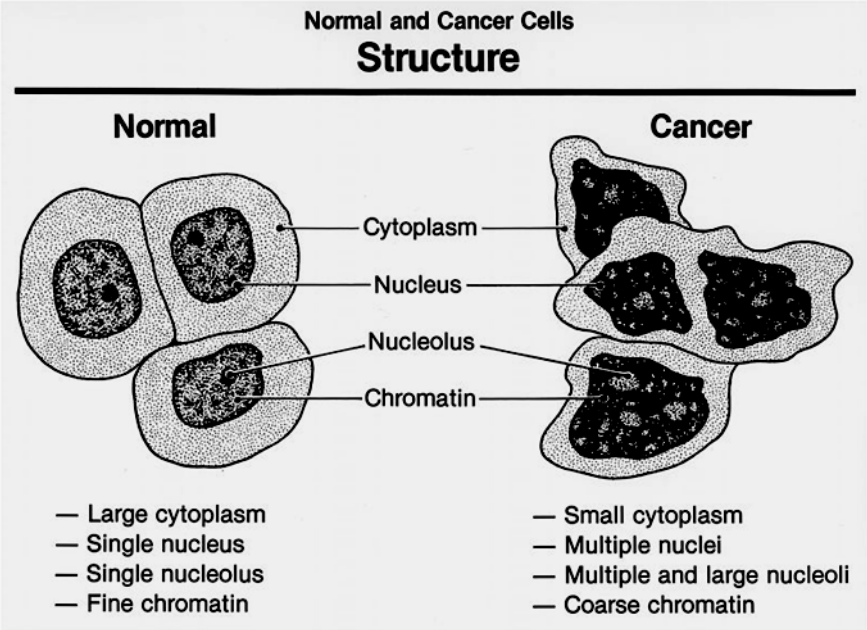
Apoptosis	Necrosis

1.cell blebbing occur .	1. cell lysis takes place .
2.Here dying cell shrinks , condense and the gets fragmented, releasing small apoptotic bodies which are engulfed . by other cells	2. here , plasma membrane gets leaked and intracellular content gets out of the cell .

Protein associated with apoptosis and their features :-

BH3 only proteins (Bid, Bim, Bad)	Promotes apoptosis
Bcl-2 , Bcl-xL	Inhibitis apoptosis
Bak, Bax	Promotes apoptosis by forming a channel in mitochondria , in order to release the Cyt c.
Apaf1	Promotes apoptosis by forming , apatosome (Apaf 1 with Cyt c) to cleaved procaspase 9 .
Procaspase 9 (Initiator caspase)	By the help of it converts into caspase 9 (initiator capase) , activates capase 3.
Caspase 3 (Effector caspase)	Kills the cell .

Cancer :-



Features of cancer:-

1.Resist apoptosis.

2.Sustain prolifertion by growth factor signaling.

3.Activate metastasis by forming invadopodium causes epithelial mesenchymal transtition (EMT).

4.Induce angeogenesis by secreting blood vessel forming growth factors.

5.Resist contact inhibition .

6.Immortal in nature .

	Normal function	Example of Gene products	Efeect of Mutation
Proto oncogenes	Promotoe cell growth and proliferation.	Anti-apoptotic proteins, transcription factors etc.	Dominant mutation occur , gain of function mutations unregulated cell divison.
Tumor supressor genes **(Rb , BRACA genes)**	Inhibits cell growth	Apoptosis promoting prtoteins, cell cycle check points proteins	Mutation usally recessive , loss of fuction of gene, allow unreguated growth.
Genome maintenance genes	Repair the DNA damage	DNA repair enzymes	Mutation usually recessive in nature, loss of function gene occur .

Chromosomal translocation leads to cancer :-

Burkitts lymphoma	Chromosomal translocation between chromosome 8 and chromosome 14 , MYC and antibody heavy chain occur respectively .
Chronic myeloid leukemia (CML)	Chromosomal translocation occur between chromosome 9 and chromosome 22 , ABL and BCR genes respectively .

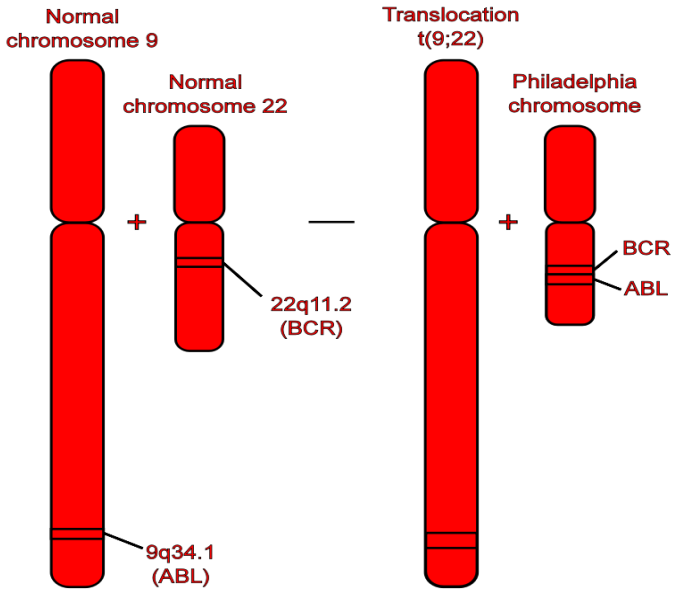


Fig:- CML disease translocation .

Disease	DNA repair system affected	Sensitivity	cancer
1. Xeroderma pigmentosa	Nucleotide Excision repair	UV radiation	Skin cancer
2. BRCA1, BRCA2	Repair Double strand breaks		Breast cancer.

Immunology

TYPES OF IMMUNITY :

On the basis of specificity

- **Innate :-** Macrophages**,** WBCs
- **Adaptive:-** B & T cells, Antibodies

On the basis of origin

- **Humoral:-** Immunoglobulins
- **Cell mediated:-** Lymphocytes

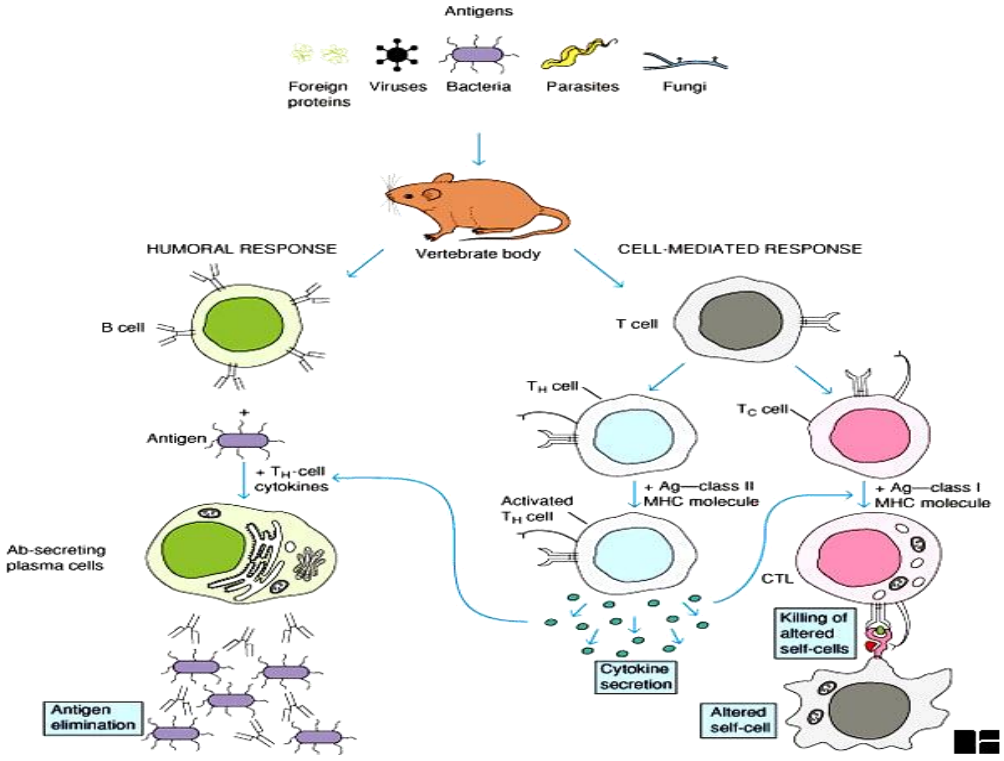


Fig 1:Overview of humoral & cell mediated immunity(credits: Kuby immunology)

Formation and maturation of lymphocytes:

	Formation	Maturation
B- cell	Bone marrow	Bone marrow
T- cell	Bone marrow	Thymus

PATHWAYS FOR ANTIGEN PROCESSING AND PRESENTATION:

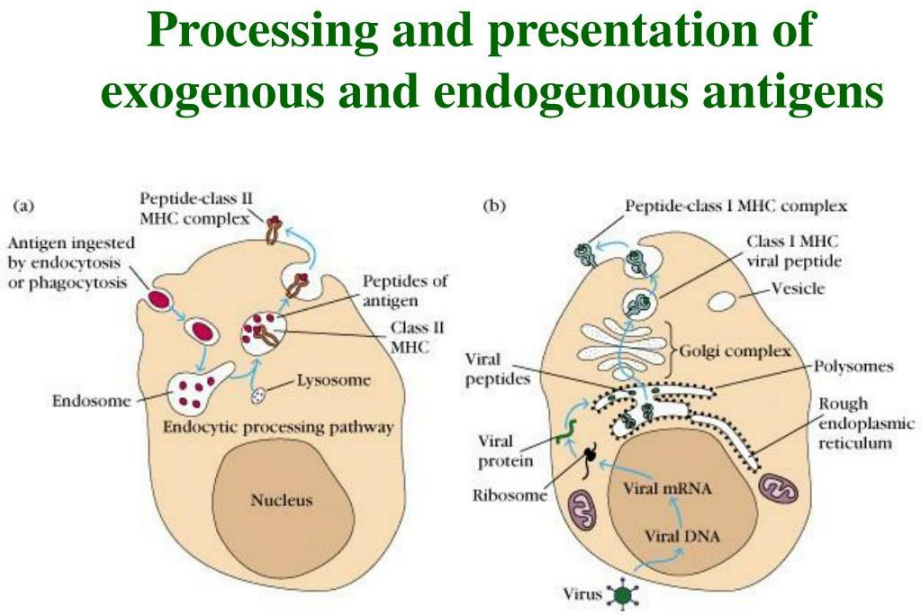


Fig 2: Antigen processing & presentation

Immunogenicity vs Antigenicity: **All immunogens are antigens but all antigens are not immunogens (with special example haptens).**

CELLS AND ORGANS OF IMMUNE SYSTEM

- **Haematopoetic stem cell lineage (myeloid and lymphoid along with sub cell lines).**
- **Apoptosis – features, difference with necrosis, genes controlling apoptosis.**
- **Graft vs Host disease.**

- **Opsonization – making antigens more susceptible to phagocytosis by macrophages.**
- **Diagrams of neutrophils,eosinophils and basophils.**

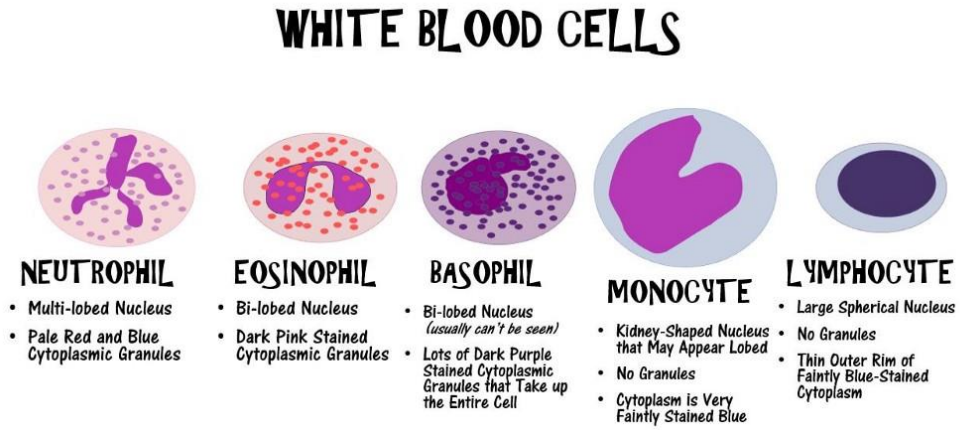


Fig 3: Source: https://mc.ai/week-5-blood-cell-classification/

ANTIGENS:

- **Properties for antigenicity**
- **Adjuvants**

TABLE 3-3 Postulated mode of action of some commonly used adjuvants

Adjuvant	POSTULATED MODE OF ACTION: Prolongs antigen persistence	Enhances co-stimulatory signal	Induces granuloma formation	Stimulates lymphocytes nonspecifically
Freund's incomplete adjuvant	+	+	+	–
Freund's complete adjuvant	+	++	++	–
Aluminum potassium sulfate (alum)	+	?	+	–
Mycobacterium tuberculosis	–	?	+	–
Bordetella pertussis	–	?	–	+
Bacterial lipopolysaccharide (LPS)	–	+	–	+
Synthetic polynucleotides (poly IC/poly AU)	–	?	–	+

Fig 4: Composition of Freund's complete & incomplete adjuvants.(Source:Kuby Immunology)

IMMUNOGLOBULINS:

- **Structure:- Two heavy chains (50,000 Kd each) and two light chains (25,000 Kd each) joined by disulfide linkages.**
- **Consists of constant and variable regions.**
- **Antibody structure by Porter & Edelmann.**

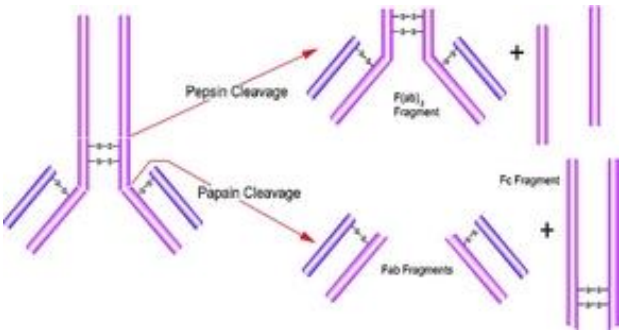


Fig 5: Digestion pf IgG by Pepsin & Papain (Source: https://www.sigmaaldrich.com)

- **Tiselieus & Kakat's experiment to prove antibodies present in Gamma globulins.**

TYPES OF ANTIBODIES AND THEIR FUNCTIONS:

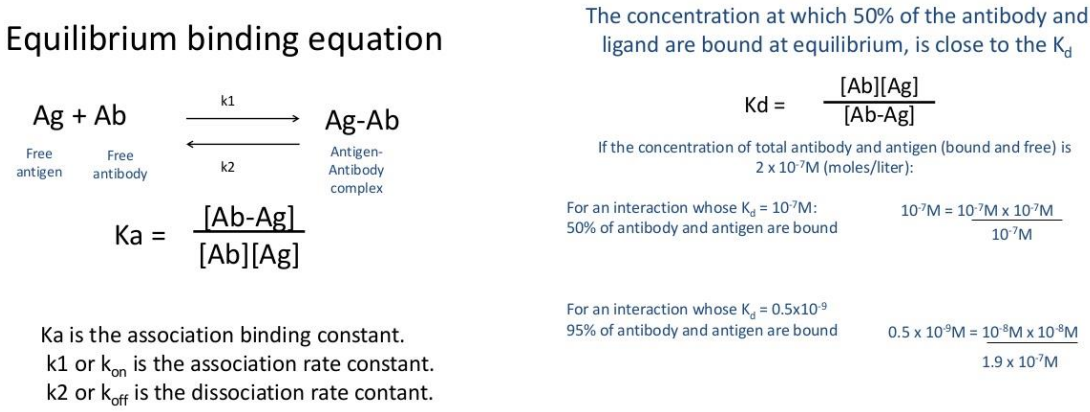
ANTIBODY CLASS	NATURE	FUNCTIONS
IgG (>80%, most abundant)	Monomer	Capable of crossing placenta.
IgA	Dimer	Secretory antibody
IgM (First Ab after infection)	Pentamer	Most effective antibody for agglutination of RBCs due to pentameric structure.
IgE	Monomer	Allergic response
IgD	Monomer	Expressed in B cells & helps to activate them by antigen presentation.

- **Antigenic determinants on immunoglobulins – Isotype, Allotype, Idiotype**

- **Monoclonal antibodies – Method of preparation using antibody producing Plasma B cell with a Myeloma cell.**

ANTIGEN ANTIBODY INTERACTIONS

Antigen antibody interactions is mediated by four types of non-covalent interactions, i.e. HYDROGEN BONDING, HYDROPHOBIC INTERACTIONS, IONIC INTERACTIONS AND VAN DER WAALS FORCES. Below are shown associative and dissociative interactions between antigens (Ag) and antibodies (Ab).



Ka= 1/Kd

Fig 7: Source: *www.slideshare.net*

ABO BLOOD GROUPING:

Blood Group	Antigen on RBC	Serum Antibody
A	A	Anti A
B	B	Anti B
AB	Both A & B	Neither

O	None	Both Anti A & Anti B

MAJOR HISTOCOMPATIBILITY COMPLEX (MHC):

CLASS OF MHC	NATURE OF ANTIGEN	CELLS WHERE PRESENT
MHC 1	**Exogenous**	**Altered Self Cells**
MHC 2	**Endogenous**	**Antigen Presenting Cells**

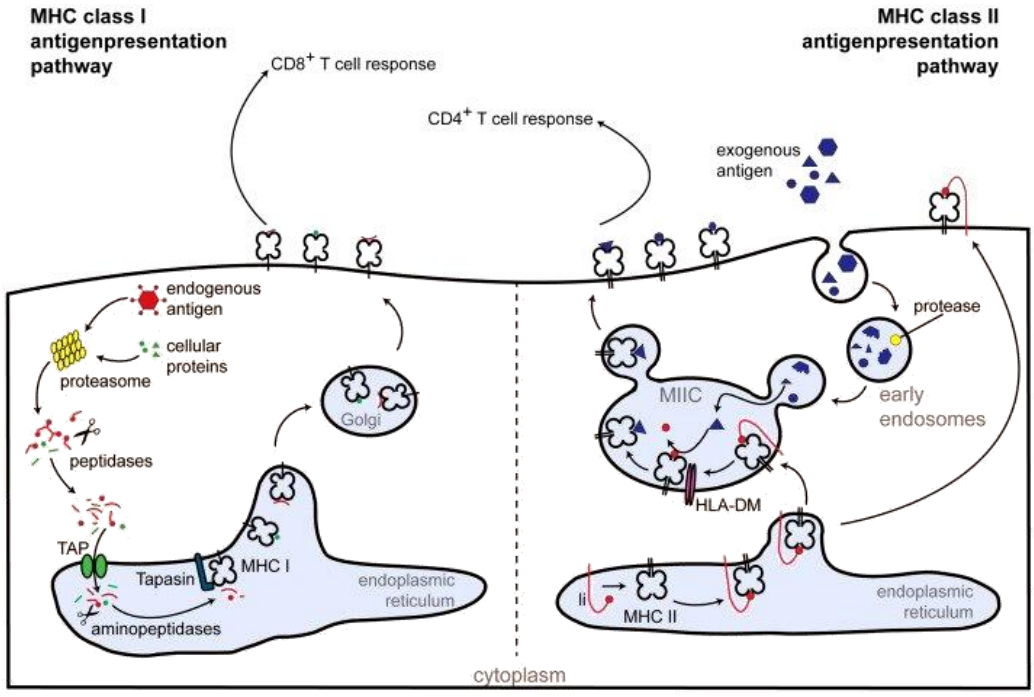


Fig 8a(left) showing endogenous antigen peptides (produced as a result of viral infection) degraded by proteosome , coupled with the MHC Class 1 inside the ER and then displayed on the surface of such Altered Self Cells. These are recognized by Tc cells which bind to the MHC 1-peptide complex by T cell receptor (TCR) and CD8+ co-receptor. Fig 8b showing exogenous bacteria/foreign peptides phagocytosed, degraded in the endosome and ultimately presented on APC surface. Helper T cells recognize and bind to such cells with their TCR and CD4+ co-receptor. [Source: www.slideshare.net]

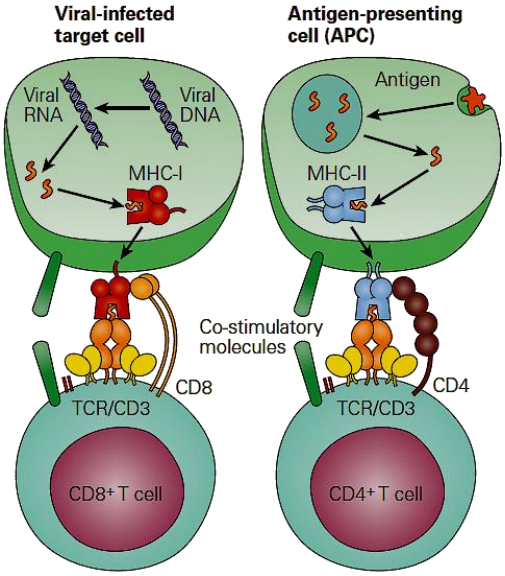
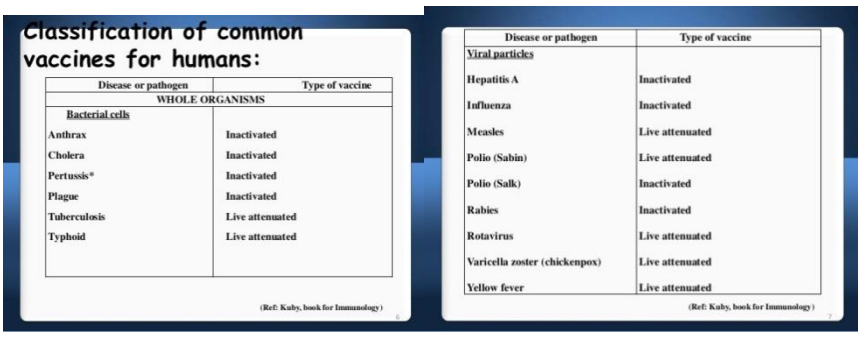


Fig 9: Recognition of Target cell-MHC 1-peptide complex by Tc cells (left)and that of APC-MHC 2-peptide complex by Th cells.(right) [Source : https://www.immunopaedia.org.za/immunology/basics/4-mhc-antigen-presentation/]

- **Abzymes – Catalytic Antibodies.**
- **Toxoids**
- **Vaccines – Classification (Live & attenuated vaccines most important).**



Classification of common vaccines for humans:

Disease or pathogen	Type of vaccine
WHOLE ORGANISMS	
Bacterial cells	
Anthrax	Inactivated
Cholera	Inactivated
Pertussis*	Inactivated
Plague	Inactivated
Tuberculosis	Live attenuated
Typhoid	Live attenuated

(Ref: Kuby, book for Immunology)

Disease or pathogen	Type of vaccine
Viral particles	
Hepatitis A	Inactivated
Influenza	Inactivated
Measles	Live attenuated
Polio (Sabin)	Live attenuated
Polio (Salk)	Inactivated
Rabies	Inactivated
Rotavirus	Live attenuated
Varicella zoster (chickenpox)	Live attenuated
Yellow fever	Live attenuated

(Ref: Kuby, book for Immunology)

Fig 10:Classification of vaccines against some common diseases and viruses affecting humans. [Source: Kuby Immunology].

HYPERSENSITIVE REACTIONS:-

There exist four types of hypersensitive reactions-

TYPE 1: IgE mediated, manifestations include systemic anaphylaxis, hay fever, asthma, eczema.

TYPE 2: IgG mediated, cell destruction via complement activation (ADCC).

TYPE 3: Immune complex mediated, causes inflammatory reactions, massive infiltration of neutrophills, causes Rheumatoid Arthritis, glomerulunephritis, serum sickness etc.

Type 4: Cell mediated, sensitized Tdth cells activate macrophages/ T cells to kill cells, causes graft rejection, contact dermitis etc.

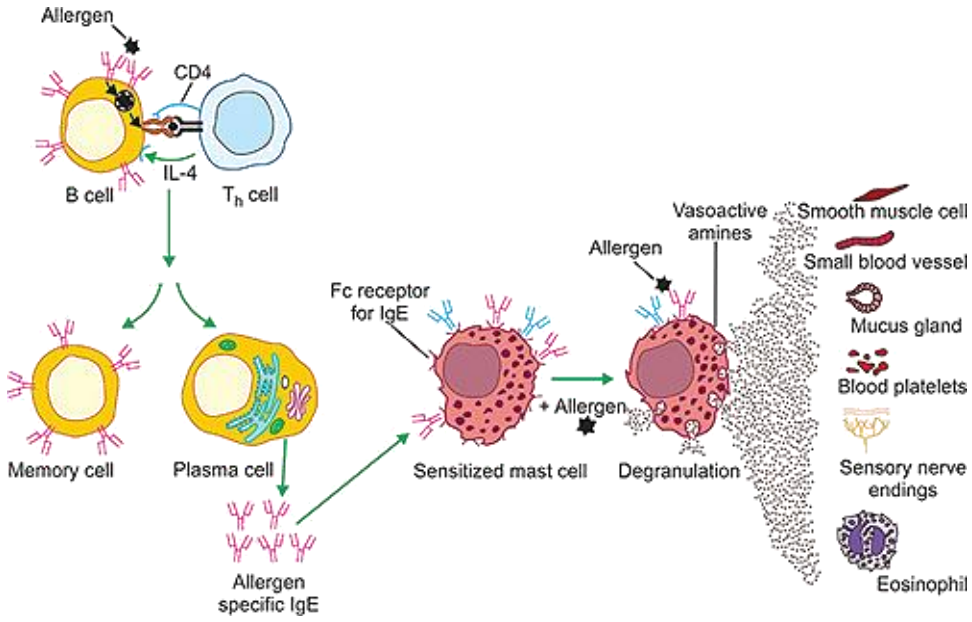


Fig 11: General mechanism of Type 1 Hypersensitive Reactions.
[Source: Kuby Immunology].

Histamine, formed by decarboxylation od amino acid Histidine, is an important component of mast cell granules.

Below is shown types of histamine receptors & their locations:

TYPES OF RECEPTORS	LOCATION	FUNCTIONS
H1	**Epithelial cells of heart & CNS.**	**Modulates internal clock, increasing brain activities.**
H2	**Heart, uterus & vascular smooth muscle cells.**	**Causes smooth muscle relaxation.**
H3	**CNS mainly.**	**Negative regulation of histamine synthesis.**
H4	**Thymus, bone marrow, spleen.**	**Regulates levels of WBC release from bone marrow.**

T- CELL HOMEOSTASIS: Homeostasis of T cells can be defined as the ability of the immune system to maintain normal T-cell counts and to restore T-cell numbers following T-cell depletion or expansion. These processes are governed by extrinsic signals, most notably cytokines. IL-7 & IL-15, two members of Gamma family of Cytokines, govern survival & proliferation of CD4+ & CD8+ T Cells. Recent evidence suggests that other cytokines, including IL-2, IL-10, IL-12, interferons and TGF-β, as well as the transcription factors T-bet and eomesodermin all play important but different roles at distinct stages of T-cell homeostasis.

MAJOR CYTOKINES, INTERLUKINS & THEIR FUNCTIONS:

NAME	BIOLOGICAL FUNCTIONS
IL-1	Co-stimulates Th activation & B cell maturation. Induces fever from hypothalamus.
IL-2	Induces Th, Tc proliferation & enhances Nk cells' activities.
IL-4	Co-stimulates activation of antigen primed B cells & induces T cell proliferation.
IFN-α	Inhibits viral replication.
IFN-β	Inhibits viral replication.
IFN-γ	Inhibits viral replication. Mediates delayed type hypersensitivity.
TGF-β	Limits inflammatory response & promotes wound healing.
TNF-α	Cytotoxic effect on tumour cells. Responsible for extensive weight loss (cachexia).
TNF-β	Cytotoxic effect on tumour cells.

Guidance For all Msc Entrance exam Aspirants

For Msc Entrance exams Should know some things

1. You should Know the Basic pattern of the exam eligibility criteria.

2. You should have previous year question paper of Msc entrance exams.

3.so question is that why the previous year question paper is very important? this question is genuine let me tell why it is important

4. Every Entrance exam follows a specific pattern of the syllabus from which most of the questions are asked so if you want to understand the basic pattern of this exam you must observe and analyze the previous year's question paper. It gives you a proper direction on how to prepare which syllabus you should follow.

5. The next important thing is that from which should you study this portion. I tell you what books you should follow for all MSC entrance exams Step by Step and I also give you the link of those books so I will be easy for you to find those books-

For Msc Zoology Entrance exams(BHU PET MSC ZOOLOGY, CUCET MSC ZOOLOGY, DU AND OTHER MSC ENTRANCE EXAMS)

(i)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(ii)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY BOOK- https://amzn.to/2AUl4nK

(iii)PARTFINDER MCQ BOOK-

(iv)ANITA SEHGAL MSC ZOOLOGY BOOK- https://amzn.to/3cQOL6n

(v)POSTGRADUATE MSC ENTRANCE BOOK(ARIHANT PUBLICATION)- https://amzn.to/2XNf8Ga

Above books for only those students who are in the final year or final semester(when you have less time for preparing Msc entrance exams).If you want to prepare for the 2nd year Then I suggest you read all the standard books.

For Msc Botany(BHU, CUCET, DU AND ALL OTHER MSC ENTRANCE EXAMS)

(i)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(ii)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY TECHNIQUES BOOK- https://amzn.to/2AUl4nK

(iii)PARTFINDER MCQ BOOK-

(iv)UNIQUE PUBLISHER MSC BOTANY BOOK- https://amzn.to/2BUEnhj

(v)POSTGRADUATE MSC BOTANY ENTRANCE BOOK(ARIHANT PUBLICATION)- https://amzn.to/2YlrpRk

Above books for only those students who are in the final year or final semester(when you have less time for preparing Msc entrance exams).If you want to prepare for the 2nd year Then I suggest you read all the standard books.

For MSc Life science(JNU DU, CUCET LIFE SCIENCE EXAM AND ALL OTHER MSC LIFE SCIENCE ENTRANCE EXAMS)

(i)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(ii)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY BOOK- https://amzn.to/2AUl4nK

(iii)PARTFINDER MCQ BOOK-

(IV)INSTANTS NOTEBOOK

1.MICROBIOLOGY- https://amzn.to/3cNzEdX

2.MOLECULAR BIOLOGY- https://amzn.to/3dS5iZc

(v)CHEMISTRY NCERT CLASS 12- https://amzn.to/2zkADVh

CHEMISTRY NCERT BOOK CLASS 11- https://amzn.to/2MM1b4T

And Chemistry handbook- https://amzn.to/2UwhJlY

(vi)PHYSICS HC VERMA- https://amzn.to/3dS5vvl

And Arihant HANDBOOK- https://amzn.to/30w3i4P

(vii)MATHEMATICS NCERT BOOK- https://amzn.to/30w5n10

And ARIHANT HANDBOOK- https://amzn.to/3feXlOa

Above books for only those students who are in the final year or final semester(when you have less time for preparing Msc entrance exams).If you want to prepare for the 2nd year Then I suggest you read all the standard books.

FOR BIOTECHNOLOGY(GAT-B JNU CEEB, IIT JAM JU, AND OTHER MSC BIOTECHNOLOGY ENTRANCE EXAMS)

(i)BD SINGH BIOTECHNOLOGY BOOK-https://amzn.to/3fcMpQO

(ii)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(iii)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY BOOK- https://amzn.to/2AUl4nK

(iv)PARTFINDER MCQ BOOK-

(V)INSTANTS NOTEBOOK

1.MICROBIOLOGY- https://amzn.to/3cNzEdX

2.MOLECULAR BIOLOGY- https://amzn.to/3dS5iZc

(vi)CHEMISTRY NCERT CLASS 12- https://amzn.to/2zkADVh

CHEMISTRY NCERT BOOK CLASS 11- https://amzn.to/2MM1b4T

And Chemistry handbook- https://amzn.to/2UwhJlY

(vii)PHYSICS HC VERMA- https://amzn.to/3dS5vvl

And Arihant HANDBOOK- https://amzn.to/30w3i4P

(viii)MATHEMATICS NCERT BOOK- https://amzn.to/30w5n10

And ARIHANT HANDBOOK- https://amzn.to/3feXlOa

Above books for only those students who are in the final year or final semester(when you have less time for preparing Msc entrance exams).If you want to prepare for the 2nd year Then I suggest you read all the standard books.

For Msc Biochemistry(AMU, BHU, CUCET, AND ALL OTHER UNIVERSITY ENTRANCE EXAMS)

(i)Lehninger Biochemistry Book(must)- https://amzn.to/2YmyYHd

(ii)CHEMISTRY NCERT CLASS 12- https://amzn.to/2zkADVh

CHEMISTRY NCERT BOOK CLASS 11- https://amzn.to/2MM1b4T

And Chemistry handbook- https://amzn.to/2UwhJlY

(iii)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(iv)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY BOOK- https://amzn.to/2AUl4nK

(v)PARTFINDER MCQ BOOK-

Above books for only those students who are in the final year or final semester(when you have less time for preparing Msc entrance exams).If you want to prepare for the 2nd year Then I suggest you read all the standard books.

For Msc Microbiology(BHU, CUCET, AND OTHER MSC ENTRANCE EXAMS)

(i)Prescott Microbiology- https://amzn.to/3cPkuF5

(ii)PARTFINDER LIFE SCIENCE BOOK COMBO- https://amzn.to/2XSCNFk

(iv)PATHFINDER BIOPHYSICS AND MOLECULAR BIOLOGY BOOK- https://amzn.to/2AUl4nK

(v)PARTFINDER MCQ BOOK-

For More Information Regarding All M.sc Entrance examination, We have a Youtube channel where A lots of videos are present already there so you can visit

SANJAY DAS

Youtubechannel-
https://www.youtube.com/watch?v=HlBNFcW8iO0&list=PLIw5aQuongCXpL1rwhKgJZF1VtkuEtEFF

Where I have made a separate Study Material Playlist form cell biology, Developmental Biology, and other subjects also. I have also a Guide for Msc entrance examination. How to prepare and how to qualify the entrance exams easily.