

**10 YEAR'S QUESTION PAPER FOR 1ST PROF MBBS EXAMINATION-
BIOCHEMISTRY**

CARBOHYDRATE CHEMISTRY- DIGESTION & ABSORPTION

GROUP-B (7 MARKS)

1. Indicate in details the chemical composition of glycosaminoglycans and proteoglycans. Name the carbohydrates present in glycoproteins and glycolipids. [5+2][2013][2015]
2. Describe the bonds present in glycosaminoglycans. Indicate the chemical difference b/w the proteoglycans & glycoproteins. [3+4][2006]
3. Define stereoisomerism. Describe different types of stereoisomerism of glucose. [1+6][2005]
4. Describe the various forms of isomerism exhibited by carbohydrates. Name the carbohydrates present in glycoproteins. [5+2][2017]

GROUP-C (3 MARKS) SHORT NOTES

1. Blood group antigens. [2010][2016]
2. Proteoglycans. [2009]
3. Glycosaminoglycans. [2004]
4. Glycemic index of carbohydrates. [2017]
5. Invert sugar. [2017]
6. Glucose transporters. [2017]

GROUP-D(3 MARKS) EXPLAIN WHY

1. Glucose and fructose form similar osazone crystals. [2011]
2. Sucrose has no anomers. [2006]
3. Sucrose is a non-reducing sugar. [2005]
4. Defective lactose digestion may lead to a clinical condition. [2015]

CARBOHYDRATE METABOLISM

GROUP-A(12 MARKS)

1. Describe in a flow diagram the metabolic pathways of glycogen formation and degradation in the body. Describe in separate charts how cyclic AMP regulates this process by enzyme modification.[4+8][2013]
2. In a flow diagram, describe the metabolic steps of glycogenesis and glycogenolysis in muscle and show how cAMP integrates their regulation.[6+6][2010]
3. Describe the metabolic steps of citric acid cycle in a flow diagram indicating the enzymes and co-enzymes involved and highlighting the steps where the energy is produced. Mention the steps in the cycle which are irreversible in nature. Indicate how propionate is converted to one of the intermediates of this cycle.[8+2+2][2013]
4. With a suitable flow chart, describe the TCA cycle. Justify TCA cycle as the final common metabolic pathway.[7+5][2005]
5. In a flow diagram, indicate the metabolic steps by which propionate can be converted to glucose and show how key enzymes of gluconeogenesis are controlled.[6+6][2010]
6. How fructose and galactose are metabolized in the body? Mention the inherited disorders related with their metabolism.[8+4][2006]
7. On complete oxidation, glucose leads to production of carbon dioxide and water. Mention those metabolic steps where carbon dioxides are evolved. Give a detailed account of enzymes, co-enzymes and control mechanisms involved in these steps. Mention three examples of metabolic reactions where carbon dioxide is utilized in this process.[6+3+3][2016]

GROUP-B (7 MARKS)

1. Give a brief account of glycogen storage disease.[7][2014]
2. Describe in details how pyruvate is converted to Acetyl-CoA in the body.[7][2013]
3. Describe multi-enzyme complex and various reactions involved in the oxidation of pyruvic acid to acetyl-CoA. [7][2011]
4. Describe the HMP shunt pathway in a flow diagram.[7][2007]
5. Explain with a flow diagram how glycolysis and gluconeogenesis in the liver are controlled by fructose 2,6 bisphosphate & the bifunctional enzyme 6-phosphofructo-2-kinase.[7][2017]

GROUP-C (3 MARKS) SHORT NOTES

1. Rapoport Leubering cycle. [2011][2017]
2. Essential pentosuria.[2010][2016]
3. Glycogen Storage disease. [2009]
4. Role of vitamins in TCA cycle[2009]
5. Metabolic role of Glucuronic acid.[2008]
6. Regulation of pyruvate dehydrogenase complex. [2007]
7. Key glycolytic enzymes. [2006]

GROUP-D (3 MARKS) EXPLAIN WHY

1. Phosphofructokinase I is known as pacemaker of glycolysis.[2012]
2. Von-Gierke's disease is associated with hyperuricemia.[2012][2017]
3. Impairment of pentose phosphate pathway (PPP) leads to erythrocytic hemolysis.[2011]
4. G6PD deficiency leads to hemolytic anemia.[2009,'07]
5. Sodium fluoride is added to blood samples drawn for blood sugar estimation.[2008]
6. Long chain fatty acids can not be converted to glucose in human body though the reverse is possible.[2010]
7. Fat can be synthesized from glucose but glucose can't be synthesized from fat.[2005][2016]
8. Fructose leads to formation of more VLDL.[2008]
9. Galactosemic patients are often associated with congenital cataract.[2016]

LIPID CHEMISTRY- DIGESTION & ABSORPTION**GROUP – A (12 MARKS)**

1. Classify phospholipids with examples. Mention their specific role in maintaining the fluidity of plasma membrane.[10+2][2013]
2. Classify different phospholipids of physiological importance with their functions.[8+4][2004]

GROUP-B (7 MARKS)

1. Tabulate a detailed account of chemical composition of plasma lipoproteins. [7][2010]
2. Describe amphiphatic lipids with examples. Describe their behavior in aqueous medium. State the importance of liposomes in clinical practice.[2+3+2][2009]
3. Classify phospholipids. Indicate their specific role in maintaining plasma membrane fluidity.[3+4][2007]
4. Classify the fatty acids in details & indicate their physical properties.[5+2][2017]

GROUP-C (3 MARKS) SHORT NOTES

1. Separation and identification of lipids by thin layer chromatography. [2013]
2. Eicosanoids.[2008]
3. Sphingolipids.[2008]
4. Gangliosides.[2006]
5. Omega-3 fatty acids.[2015]
6. Glycosphingolipids[2017]

GROUP- D (3 MARKS) EXPLAIN WHY

1. Lecithin is amphipathic as well as amphoteric in nature.[2014]
2. Arachidonic acid may not be considered as an essential fatty acid.[2010]
3. Intake of fish oils are beneficial for cardiac patients in contrast to animal fat.[2009]
4. Acid number helps in the identification of rancidity in fats and oils.[2016]

LIPID METABOLISM**GROUP-A (12 MARKS)**

1. Give an account of fatty acid synthase complex. Describe the metabolic pathway for de-novo synthesis of palmitate in the body.[3+9][2014]
2. Describe the metabolic steps of biosynthesis of cholesterol. Discuss the control metabolism associated with HMG CoA reductase. Explain reverse cholesterol transport.[8+2+2][2017]

GROUP-B(7 MARKS)

- 1._ Give the exact chemical composition of very low density lipoprotein. Explain their formation and fate inside the body.[2+5][2013]
- 2._ Describe how ketone bodies are formed & subsequently degraded in the body.[3+4][2015]

GROUP-C (3 MARK) SHORT NOTES

- 1._ Control of HMG-CoA reductase.[2013]
- 2._ Role of carnitine in fatty acid metabolism.[2015]
- 3._ Fatty acid synthase complex. [2017]

GROUP-D (3 MARKS) EXPLAIN WHY

- 1._ Both uncontrolled diabetes mellitus and prolonged fasting produce ketosis but its magnitude is less in the case of prolonged fasting.[2014]
- 2._ Citric acid cycle operating in the mitochondria can take part in the extra mitochondrial fatty acid synthesis.[2004]
- 3._ Ketone bodies are degraded in the extrahepatic tissues only.[2013]
- 4._ HDL is involved in reverse cholesterol transport.[2015]
- 5._ Lipoprotein lipase deficiency may lead to hyperglyceridemia.[2015]
- 6._ Citrate plays an important role in fatty acid synthesis.[2017]

AMINO ACID , PROTIEN CHEMISTRY, DIGESTION AND ABSORPTION & TISSUE PROTEINS AND PROTEIN PURIFICATION

GROUP-A (12 MARKS)

1. Describe the salient features of alpha helix and beta pleated sheet structure of proteins. Mention the non-covalent interactions which stabilize protein confirmation. Briefly discuss the role of peripheral & integral proteins in the network of plasma proteins.[4+3+5][2014]
2. Compare and explain the oxygen binding curves of hemoglobin and myoglobin. Indicate the conformational changes that occur in hemoglobin on oxygenation. Mention the basic variations in the chemical structures of HbS and HbM as compared to the adult hemoglobin.[6+3+3][2014][2016]
3. Describe how the amino acid composition, N-terminal & C-terminal residues of a protein are determined & identified. Describe the bonds responsible for the four structures of proteins . Briefly indicate how a molecular weight of a protein is determined.[7+3+2][2013]
4. Describe the peptide bond. What are the different forces that stabilize the protein structure at the different levels of organization ? Give an example to explain the primary structure that determines the functional state of proteins.[4+5+3][2011]
5. Discuss the four orders of protein structures. Describe the alpha helical form of a globular protein. State briefly how the amino acid sequence in a polypeptide chain can be determined.[6+2+4][2010]
6. Discuss briefly how the chemical structures of myoglobin and hemoglobin influence their biological activities. Describe the changes that take place in hemoglobin on oxygenation. [6+6][2010]
7. Describe in detail how the number, kind and sequence of amino acids in a polypeptide chain are determined. [4+4+4]
8. Using hemoglobin and myoglobin as models , justify the statement, "Chemical structure of a protein decides its biochemical functions". [12][2007]

GROUP-B(7 MARKS)

1. Describe the principles of electrophoresis. Illustrate with diagram the electrophoretic separation of the serum proteins indicating the significance of each separated band. Explain the importance of acute phase reactants. [3+2+2][2014]
2. Describe the mechanism of absorption of amino acids from the gut. What is Hartnup's disease?[5+2][2009]
3. Describe the primary, secondary and tertiary structures of the proteins mentioning the forces that stabilize them.[7][2008]
4. Describe the chemical structure of collagen and the chemical reason for its toughness. [5+2][2007]

5. Describe briefly the chemical structures of hemoglobin. Compare the chemical structures of hemoglobin and myoglobin. Explain how oxyhemoglobin and deoxyhemoglobin act as buffers in the maintenance of acid base balance in our body.[2+1+4][2006]
6. Give an outline of procedure for determination of primary structure of a protein having single polypeptide chain. [7] [2005]
7. Name the different immunoglobulins. Give the structure and functions of IgG. [2+5][2004]
8. Classify L-amino acids present in the proteins. Explain how amino acids are separated and identified from a mixture of amino acids . [2+5][2015]

GROUP-C (3 MARKS) SHORT NOTES

1. Glycosylated Hemoglobin.[2011]
2. Prions.[2011]
3. Protein folding.[2007][2015]
4. Bonds maintaining the tertiary structure of protein. [2006]
5. 2,3 BPG on Hb-Oxygen interaction.[2004]
6. Selenocystine. [2015]
7. Electrophoresis. [2016]
8. Beta pleated sheet. [2017]

GROUP -D (3 MARKS) EXPLAIN WHY

1. Patient with Hb-S often suffers from anemia.[2013][2017]
2. Collagen has quarter staggered triple helical structure.[2012]
3. Glycine solution can not rotate the plane of plain polarized light.[2012]
4. 2,3 BPG helps in delivery of Oxygen to the tissues.[2011]
5. 2,3 BPG helps in decreasing the affinity of hemoglobin towards oxygen. [2007]
6. Both protein and urea give positive biuret test. [2005]
7. Chaperons play a very significant role in protein folding.[2016]
8. Hb-A1c provides valuable information for management of diabetes mellitus.[2016]
9. Myoglobin does not exhibit Bohr effect.[2015]

AMINO ACID AND PROTEIN METABOLISM

GROUP-A (12 MARKS)

1. Describe how catabolism of haem produces bilirubin. Indicate in details the process of uptake, conjugation and secretion involved in transfer of bilirubin from blood to bile.[6+6][2014]
2. Describe the formation and degradation of epinephrine in the body. [7+5][2007]

GROUP-B (7 MARKS)

1. Describe the process of transamination and oxidative deamination in the body.[4+3][2014]
2. Write the synthesis, transport and degradation of catecholamines.[7][2011]
3. Describe how catecholamines are synthesized and degraded inside the human body.[3+4][2010]
4. What are the metabolic products of tyrosine? Describe with suitable flow chart the biosynthesis of catecholamines.[2+5][2008]
5. Describe the formation and fate of ammonia inside the body.[2+5][2009]/[3+4][2006]
6. Give the reaction intermediates and bioenergetics of urea cycle .[3+3+1][2004]
7. Write down with flow chart the steps of catabolism of carbon skeleton phenylalanine & mention the steps which are blocked in phenylketonuria and alkaptonuria.[5+2][2005]

GROUP- C (3 MARKS) SHORT NOTES

1. Acute intermittent porphyria.[2014]
2. S-Adenosyl Methionine.[2013]
3. Maple Syrup Urine Disease. [2010]
4. Polyamines. [2010]
5. Phenylketonuria.[2004]

GROUP- D(3MARKS) EXPLAIN WHY

1. Patient with carcinoid syndrome may exhibit pellagra.[2013]
2. Alkaptonuria is often associated with generalized pigmentation of connective tissue (Ochronosis). [2013]
3. Ammonia is toxic to Central Nervous System. [2012]
4. Phototherapy (exposure to blue light) helps in treatment of neonatal physiological jaundice.[2011]
5. Urine turns black on standing in Alkaptonuria.[2010]
6. Urinary urobilinogen is increased in hemolytic jaundice . [2009]
7. Proteinuria. [2004]

CHEMISTRY OF NUCLEOTIDES AND NUCLEIC ACIDS

GROUP- B (7 MARKS)

1. With the help of a diagram describe the chemical structure of a tRNA and mention the function of its different arms.[3+4][2007]
2. Give the structure of a deoxyribonucleotide. Write down with the help of a diagram and mention its different functions. [2+5][2005]

GROUP- C (3 MARKS)

1. t-RNA.[2011]
2. Synthetic nucleotide analogues. [2009]
3. Pseudo nucleotides. [2007]
4. Structural features of A, B and Z-DNA.[2007]
5. Bonds in polynucleotides.[2017]

GROUP-D (3 MARKS) EXPLAIN WHY

1. DNA with higher GC content have relatively higher T_m . [2014]
2. RNA is alkali labile while DNA is alkali resistant.[2012]
3. DNA can occur in different 3D models.[2004]
4. Synthetic nucleotides are used as drugs.[2015]
5. DNA is more stable than RNA.[2016]
6. Adenine nucleotides have various functions beside making nucleic acids. [2017]

NUCLEOTIDE METABOLISM

GROUP-B (7 MARKS)

1. Name the endpoint of purine catabolism and process of breakdown of purine. [2+5][2008]
2. Indicate the source of nitrogen and carbon atoms of the purine ring in a diagram.
Describe how purines are catabolised.[7][2006]

GROUP-C (3 MARKS) SHORT NOTES

1. Gout.[2013]
2. Source of nitrogen and carbon atoms of the purine ring.[2013]
3. Purine salvage pathway. [2005]

GROUP-D (3 MARKS) EXPLAIN WHY

1. Dietary purines are not essential.[2008]
2. Intake of alcohol may aggravate the symptoms of gout.[2016]
3. Synthetic nucleotides are used as drugs.[2015]

BIOLOGICAL OXIDATION

GROUP – A (12 MARKS)

1. Explain oxidative level & substrate level phosphorylation. Give two examples of substrate level phosphorylation. Explain the mitochondrial electron transport chain.[2+2+8][2008]
2. Describe with diagram the respiratory chain complexes that span the inner mitochondrial membrane indicating the specific sites of energy production. What are its mobile components? Mention the role of inhibitors of respiratory chain. What purpose is served by their use in vitro. What happens when complex I is deficient? [5+2+5+1][2006]

GROUP-B (7 MARKS)

1. Describe the operation and significance of glycerophosphate shuttle and malate shuttle.[3+4][2014]
2. What is oxidative phosphorylation? Differentiate it from substrate level phosphorylation. Illustrate with a diagram how ATP is synthesized in mitochondria?[2+2+3][2014]
3. Describe the mitochondrial electron transport chain. How the inhibitors of ETC differ from uncouplers of oxidative phosphorylation?[5+2][2011]
4. Describe the chemiosmotic coupling hypothesis of oxidative phosphorylation. [7][2010]

GROUP-C (3 MARKS) SHORT NOTES

1. Uncouplers.[2004]
2. Malate shuttle.[2005]

GROUP-D(3 MARKS) EXPLAIN WHY

1. G6PD is responsible for erythrocyte membrane rigidity. [2014]
2. Brown adipose tissue promotes thermogenesis. [2011, '10]

CLINICAL FUNCTION TEST, MEMBRANE TRANSPORT AND ENZYMES

GROUP-A (12 MARKS)

1. Explain the Michaelis Menten equation and explain the role of substrate concentration on the rate of enzyme catalyzed reaction with the help of graphs. Illustrate how V_{max} and K_m are affected by competitive and non competitive inhibition of enzymes. “ The K_m value for glucokinase is

much higher than that for hexokinase though both act on glucose"- explain the statement.[6+4+2][2017][2013]

2. Name 5 enzymes whose catalytic activities are altered by covalent phosphorylation-dephosphorylation and indicate their functions. According to International Union of Biochemists, enzymes are classified into six major groups. Indicate in which groups the following enzymes belong:
 - A) Adenylate cyclase,
 - B) DNA dependant RNA polymerase.
 - C) Aldolase,
 - D) Chymotrypsin,
 - E) Reverse Transcriptase,
 - F) Enolase
 - G) Acetyl CoA carboxylase. [5+7][2017][2015]

GROUP-B(7 MARKS)

1. Describe the renal mechanism for regulation of acid base balance. What is the biomedical importance of anion gap?[4+3][2014]
2. Define jaundice. With the help of liver function test and urine test, show it can be differentiated b/w hepatocellular and obstructive jaundice. [2+5][2008]
3. Explain the mechanism of allosteric regulation of enzyme activity using PFK as an example. Mention the other mechanisms by which the enzyme action is regulated.[4+3][2016]
4. Describe the methods of determining the chemical structure of any unknown biomolecule.[7][2017]

GROUP- C (3 MARKS) SHORT NOTES

1. Receptor mediated endocytosis.[2014]
2. Ionophores.[2014]
3. Respiratory acidosis. [2013]
4. Non functional plasma enzymes.[2016]

GROUP-D(3 MARKS) EXPLAIN WHY

1. Renal clearance study is an early predictor of impending renal failure.[2014]
2. Isoenzymes of Alkaline Phosphatase are of diagnostic significance.[2014]
3. Non function plasma enzymes are important only for clinical purposes.[2014]
4. Levels of hepatic enzymes can differentiate b/w hemolytic, hepatocellular and obstructive jaundice.[2013]
5. Colloids are biologically important having clinical significance.[2013]
6. The mode of action of metallo-enzymes and metal activated enzymes are different.[2013]
7. The concentration of creatinine in blood predicts renal function of an individual.[2008]
8. Isoenzyme assay is helpful in the diagnosis of MI.[2015]

FREE RADICALS AND ANTIOXIDANTS

GROUP-B(7 MARKS)

1. What are free radicals? How do they damage the biological systems? Name the various antioxidants protecting the organisms. [1+3+3][2009]
2. Describe the reactions catalyzed by the enzyme superoxide-dismutase. Describe the Cyt-P450 dependant microsomal hydroxylation reaction. Mention two examples of hydroxylation reactions where ascorbic acid is involved.[1+5+1][2006]

GROUP- C (3 MARKS) SHORT NOTES

1. Biochemical functions of peroxisomes.[2014]
2. Antioxidant enzymes. [2014]
3. Role of Cyt-P450 in hydroxylation reaction.[2013]
4. Super-oxide dismutase.[2013]
5. Glutathion.[2008]

GROUP-D(3 MARKS) EXPLAIN WHY

1. Superoxide dismutase protects aerobic organisms against oxygen toxicity.[2008]
2. Lipid peroxidation is a source of free radicals.[2007]
3. Defence mechanisms of the body to fight the toxicity of free oxygen species.[2004]
4. ROS damages cellular architecture.[2006]

CELL CYCLE AND CANCER, VITAMINS AND MINERALS

GROUP-C (3 MARKS) SHORT NOTES

1. Tumor markers.[2014]
2. Ceruloplasmin. [2014]
3. Cell cycle regulators.[2010]
4. Proto-oncogenes. [2009]
5. Activation of Proto-oncogenes.[2006]
6. Marker Enzymes.[2006]

GROUP – D (3 MARKS) EXPLAIN WHY

1. Radio Immuno Assay techniques has got demerits also.[2013]
2. Methotrexate is used for anti-cancer therapy.[2012, '11]

MOLECULAR ENDOCRINOLOGY AND SIGNAL TRANSDUCTION

GROUP-A (12 MARKS)

1. Explain how normal blood sugar is maintained by various mechanisms.[12][2008, '04]

GROUP-B (7 MARKS) _ _ _ _ _

1. Explain the mechanisms of signal transductions by cAMP, calcium and phosphatidyl inositol system with the help of diagrams.[7][2013]
2. In a flow diagram describe how insulin and glucagon regulate the process of lipogenesis and lipolysis in adipose tissue. [7][2010]
3. Discuss the different types of G-protein coupled signal transduction processes.[7][2010]

GROUP-C (3 MARKS) SHORT NOTES

1. G-protein[2011]
2. 3'-5' cAMP. [2004]
3. Receptors in signal transductions.[2007]
4. Insulin receptors. [2006]
5. Receptor enzymes. [2005]
6. Calcium as 2nd messenger. [2005]

GROUP-D (3 MARKS) EXPLAIN WHY

1. Calcium as 2nd messenger.[2004]
2. Lipids can act as intracellular signals.[2012]
3. Receptor enzymes show intrinsic catalytic activity.[2009]

MOLECULAR BIOLOGY & GENETICS

GROUP-A (12 MARKS)

1. Describe the stage of initiation of translation process with the help of a diagram. State the mechanism of action of the following antibiotics in the inhibition of translation:
A) Streptomycin,
B) Erythromycin,
C) Chloramphenicol. [6+6][2011]
2. Write down the different types of DNA damage. Explain the mechanisms of : Mismatch DNA repair, Base excision repair, Nucleotide excision repair. [6+6][2011]
3. Describe the process of synthesis of proteins in prokaryotes. What are the roles of different antibiotics to inhibit the process of translation in prokaryotes. [8+4][2009]
4. Describe the process of replication in *E.Coli* with suitable diagram. Mention the differences among different *E.Coli* polymerases.[8+4][2008, '05]

GROUP-B(7 MARKS)

1. Describe the initiation, elongation and termination phase of transcription in eukaryotes. Name the antibiotics which specifically inhibit the microbial protein synthesis.[5+2][2015-12 marks] [2013]
2. Describe how ribonucleic acid is synthesized . Indicate the difference b/w DNA Polymerase III and RNA polymerase.[5+2][2010]
3. Indicate the different mechanisms of DNA repair.[7][2007]
4. Define mutation. Describe different types of mutations with examples.[1+6][2005]
5. Describe the operon model. Explain how it functions.[4+3][2004]
6. Give an account of negative and positive regulation of lac operon in *E.Coli*. [2015][7]
7. Enumerate the DNA damaging agents and indicate the types of damages made by them.[7][2017]

GROUP-C (3 MARKS) SHORT NOTES

1. Eukaryotic topoisomerase. [2014]
2. Polyclonal antibodies. [2014]
3. Radioisotopes. [2014]
4. Base excision repair of DNA.[2013]
5. Frame shift mutation. [2013]
6. Restriction Fragment Length Polymorphism (RFLP). [2011]
7. Monoclonal antibodies. [2011]
8. Gene therapy. [2009]
9. RNA processing. [2009]
10. Ribozyme.[2008, '07]
11. Restriction Endonuclease enzyme.[2008, '06]
12. Genome of Retrovirus. [2007]
13. PCR(Polymerase Chain Reaction). [2005]
14. Mismatch DNA repair. [2005]
15. Point mutation. [2016]
16. DNA replication in eukaryotes and prokaryotes.[2015]
17. RNA editing.[2015]

GROUP-D (3 MARKS) EXPLAIN WHY

1. RNA can act as enzyme.[2011]
2. DNA denaturation is essential for hybridization. [2007]
3. Genetic code is degenerative and unambiguous. [2005]
4. DNA is much more stable than RNA. [2016]
5. Ribosome is the ultimate ribozyme.[2017]