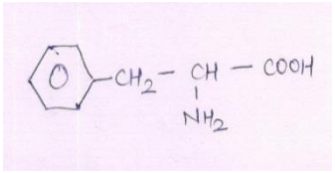
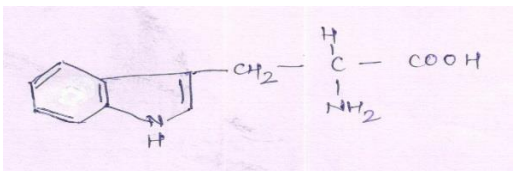
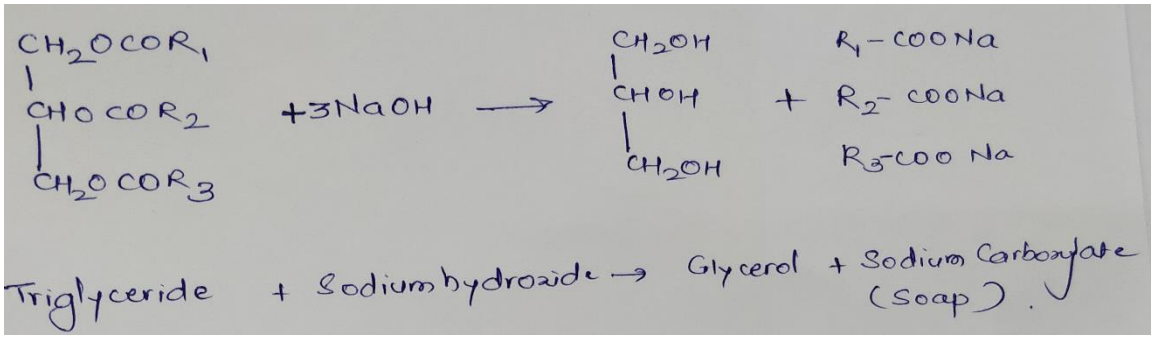
Subject Title: Biochemistry & Clinical Pathology **Subject Code:** 0808

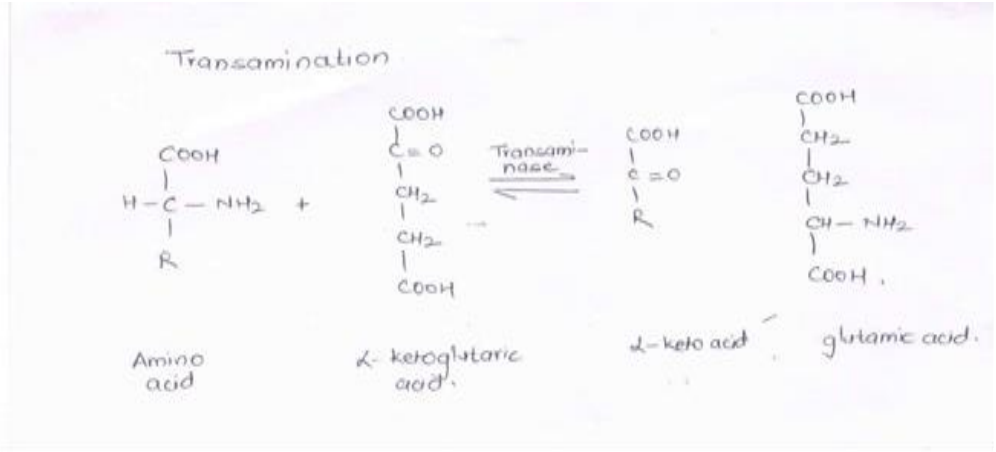
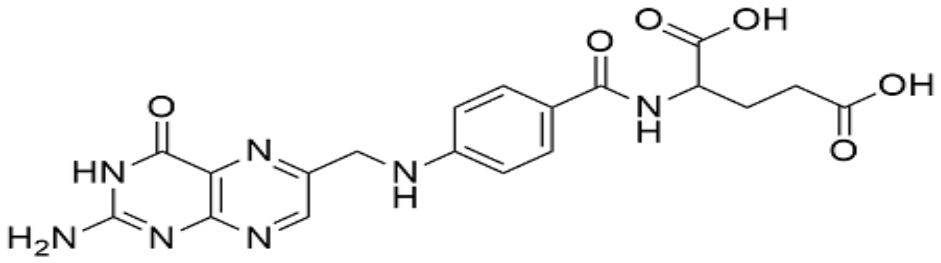
Important Instructions to examiners:

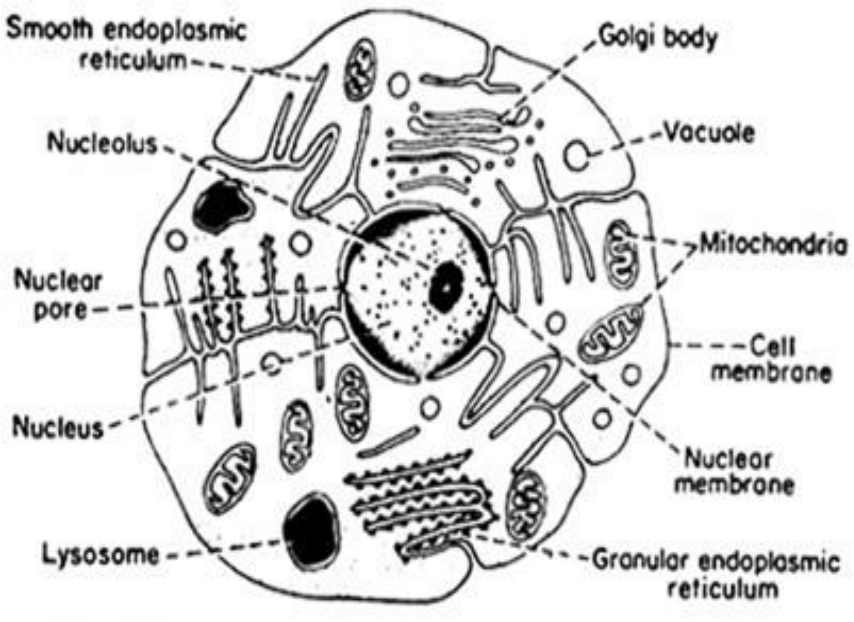
1) The answers should be examined by key words and not as word-to-word as given in the model answer scheme.
2) The model answer and the answer written by candidate may vary but the examiner may try to assess the understanding level of the candidate.
3) The language errors such as grammatical, spelling errors should not be given more Importance (Not applicable for subject English and Communication Skills.
4) While assessing figures, examiner may give credit for principal components indicated in the figure. The figures drawn by candidate and model answer may vary. The examiner may give credit for anyequivalent figure drawn.
5) Credits may be given step wise for numerical problems. In some cases, the assumed constant values may vary and there may be some difference in the candidate's answers and model answer.
6) In case of some questions credit may be given by judgement on part of examiner of relevant answer based on candidate's understanding.
7) For programming language papers, credit may be given to any other program based on equivalent concept.

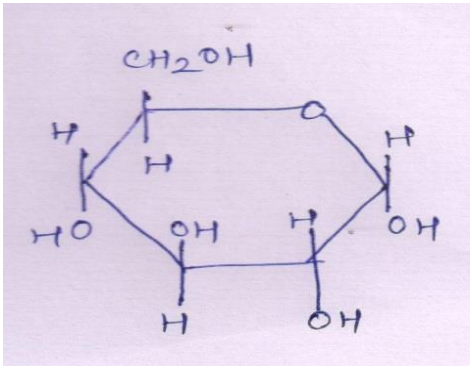
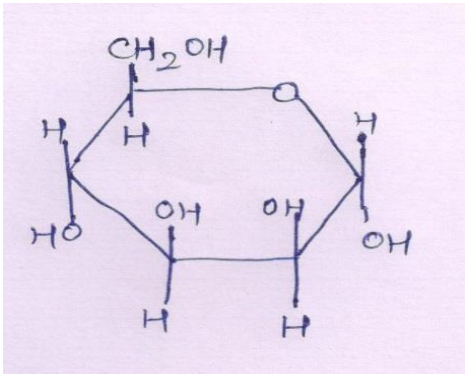
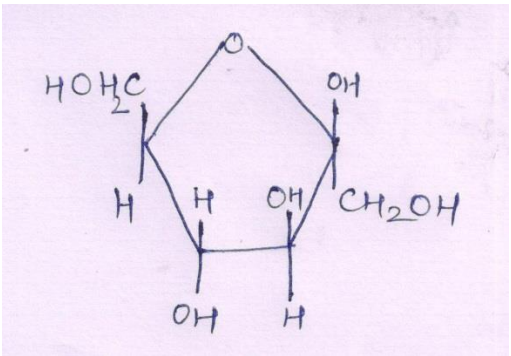
Q. No.	Sub Q. N.	Answer	Marking Scheme
1		**Solve any Eight of the followings:**	**16M**
1	**a)**	**Define & explain metabolism.** All biochemical changes that occur in biological system are grouped together as metabolism. Metabolism is divided into two categories: Catabolism: It's a degradative process concerned with the breakdown of complex molecules to simpler ones along with release of energy. Anabolism : Includes the biosynthetic reactions of formation of complex molecules from simple ones and requiring energy.	**1Mdefn** **1M Expln.**
	b)	**What is enediol reaction of carbohydrate? Give its biological importance.** The process of shifting a hydrogen atom from one carbon atom to another to produce enediols is known as tautomerization. Sugars possessing anomeric carbon atom undergo such reaction in alkaline medium. **Importance**: The enediols are highly reactive; hence sugars in alkaline solution are powerful reducing agents.	**1M expln** **1M importanc e**
	c)	**What are essential amino acids? Give structure of any one of them.** Essential amino acids: Amino acids which cannot be synthesized by the body but which are required for normal functioning of body and supplied through diet. Examples: Isoleucine,Histidine,Leucine,Methionine,Lysine,Phenylalanine,Tryptophan, Threonine. Phenylalanine  Tryptophan 	**1M Defn** **1M Any correct structure**

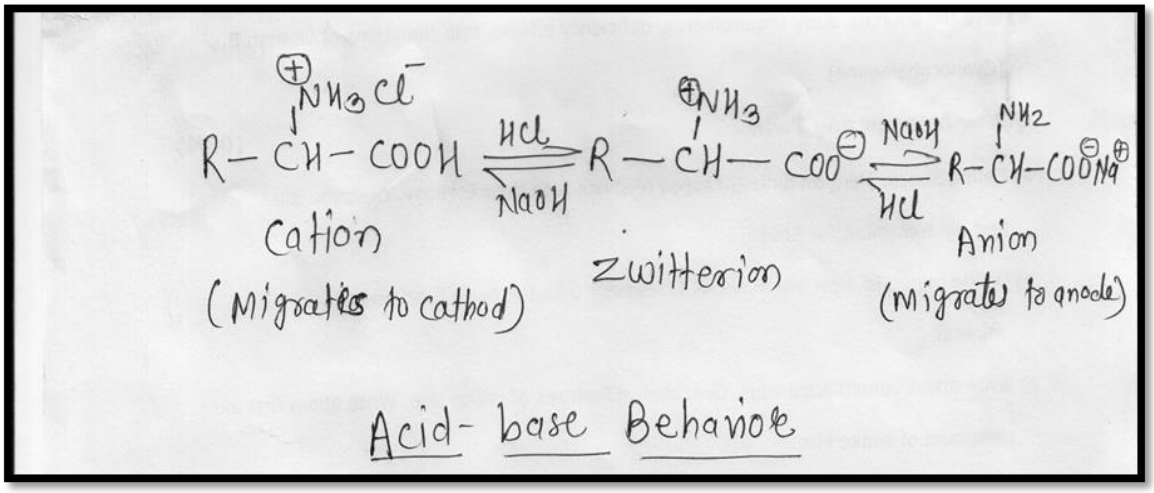
	d)	**Explain with chemical reaction, saponification reaction of simple fats.** When triglycerides in fat/oil react with aqueous NaOH or KOH, they are converted into soap and glycerol. This is called alkaline hydrolysis of esters. Since this reaction leads to the formation of soap, it is called the Saponification process.  	**1M Expln& Rn each**
	e)	**What is egg white injury? Give its symptoms.** It's a deficiency disease of biotin which is rare and generally observed when large quantities of raw eggs are consumed. Egg white contains large amount of protein avidin which binds to biotin very tightly and prevents its absorption in the intestine. The avidin in egg white may be a defence mechanism inhibiting growth of bacteria. When eggs are cooked, avidin gets denatured along with other egg white proteins. **Symptoms:** Anaemia Loss of appetite, Nausea, Dermatitis, Glossitis	**1 M expln 1 m symptoms**
	f)	**Define pathology. Name any one pathological condition in human being.** **Definition**: It's a significant field in medical diagnosis and medical research, concerned mainly with the causal study of disease, whether caused by pathogens or non-infectious physiological disorder. Pathological condition: Diabetis ,Anaemia , Pyuria, Haematuria, Proteinuria, ,Jaundice etc. **Any other correct pathological condition be considered.**	**1M defn 1M any one correct condition**

	g)	**What do you mean by s-GOT in enzymes? What is its significance?** Serum glutamic-oxaloacetic transaminase, or SGOT/AST. It is an enzyme made by liver cells. When liver cells are damaged, it leaks out into the bloodstream and the level in the blood becomes higher than normal. **Significance:** It may be elevated in liver damage, in diseases affecting other organs, such as myocardial infarction, acute pancreatitis, acute haemolytic anemia, severe burns, acute renal disease, musculoskeletal diseases, and trauma.	**1M each**
	h)	**What is the importance of electron transport & oxidative phosphorylation in carbohydrate metabolism?** Most of the free energy released during the oxidation of glucose to CO_2 is retained in the reduced coenzymes NADH and $FADH_2$ generated during glycolysis and the citric acid cycle. During respiration, electrons are released from NADH and $FADH_2$ and eventually are transferred to O_2, forming H_2O according to the following overall reactions: $NADH + H^+ + ½\ O2 = NAD^+ + H_2O$ $FADH_2 + ½\ O_2 = FAD + H_2O$ Importance : 1. To transfer electrons from NADH and $FADH_2$ to the oxygen so as to form water. 2 .These electrons are used to power ATP production.	**2M**
	i)	**Explain the process of transamination in protein catabolism.** In transamination, the NH_2 group on one molecule is exchanged with the C =O group on the other molecule. The amino acid becomes a keto acid, and the keto acid becomes an amino acid.In this example alpha ketoglutaric acid becomes glutamic acid, amino acid becomes keto acid. This reaction is reversible. It is important for redistribution of amino group and production of non-essential amino acid as per the requirement of the cell.	**2M**

		It diverts excess amino acids towards energy generation. 	
	j)	**What are ketone bodies? What is ketogenesis?** Ketone bodies are acetone, acetoacetic acid and beta hydroxyl butyric acid, their synthesis can occur in response to an unavailability of blood glucose. Ketogenesis is the biochemical process by which organisms produce a group of substances collectively known as ketone bodies by the breakdown of fatty acids and ketogenic amino acids. This process supplies energy to certain organs (particularly the brain) under circumstances such as fasting.	**1M each**
1	**k)**	**Give only structure of folic acid.**  Folic acid	**2M**
1	**l)**	**How water is distributed in the different compartments in the body of human being?** Body fluid refers to body water & dissolved substances. Fluid comprises of about 60% of total body weight.	**2M**

		Intracellular Fluid (ICF): The amount of water that's inside our cells accounts for 2/3rds of our Total body water. Extracellular Fluid (ECF): The amount of water that surrounds our cells accounts for 1/3 of our Total body water. About 80% of ECF is interstitial fluid & 20% is blood plasma fluid.	
2		**Attempt any FOUR of the followings**	**12M**
2	**a)**	**Define cell. Draw neat labelled diagram of atypical animal cell & give two functions of mitochondrion.** **Cell :** Cell is the basic structural, functional, and biological unit of all living organisms. is the basic structural, functional, and biological unit of all known living organisms. **Diagram:**  **Functions of Mitochondria (any two)** Mitochondria are engaged in oxidative metabolism. Are responsible for the transportation of chemical energy into biological energy, in the form of ATP . All enzymes involved in Kreb's cycle are present in mitochondria.	**0.5M Defn** **1.5M Diagram** **1M functions**

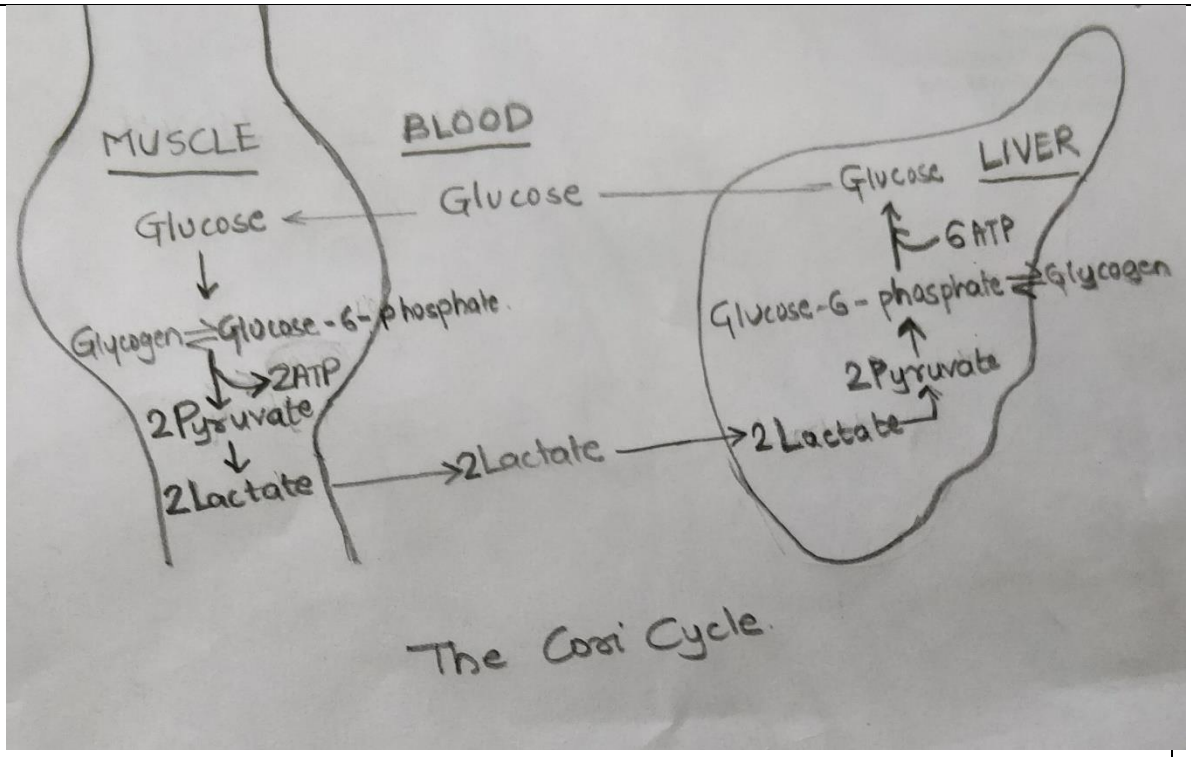
2	**b)**	**Give structures of the following:** i) Alpha D-glucose.  ii) Alpha-D mannose  iii) Beta-D fructose 	**3M** **1M each**

2	**c)**	**Discuss acid base nature of amino acids & explain isoelectric point of an amino acid.** **i.** Amino acids are amphoteric in nature ii. The amino group (NH2) can accept proton (H+) and form cation (NH3+). iii. The carboxyl group can donate H+ and for anion (COO-). iv. At acidic pH the amino acids are positively charged. V. At basic pH they are negatively charged. vi. At intermediate pH, the charge is zero, it carries both positive and negative charges. vii. This pH is called **Isoelectric pH.** At the isoelectric pH, the amino acid exists as Zwitter ion which carries equal number of positive and negative charges and net charge becomes zero . viii. At the **Isoelectric point** that amino acid becomes insoluble and precipitates out.  	**3M**
2	**d)**	**Define lipids. Classify lipids with examples.** **Lipids** are organic substances relatively insoluble in water, soluble in organic solvents related to fatty acids & utilised by living cells. **Classification:** Simple lipids: Esters of fatty acids with alcohol. • Fats & oils : Castor oil • Waxes : Bees wax	**1Mdefn** **2M** **Classifn**

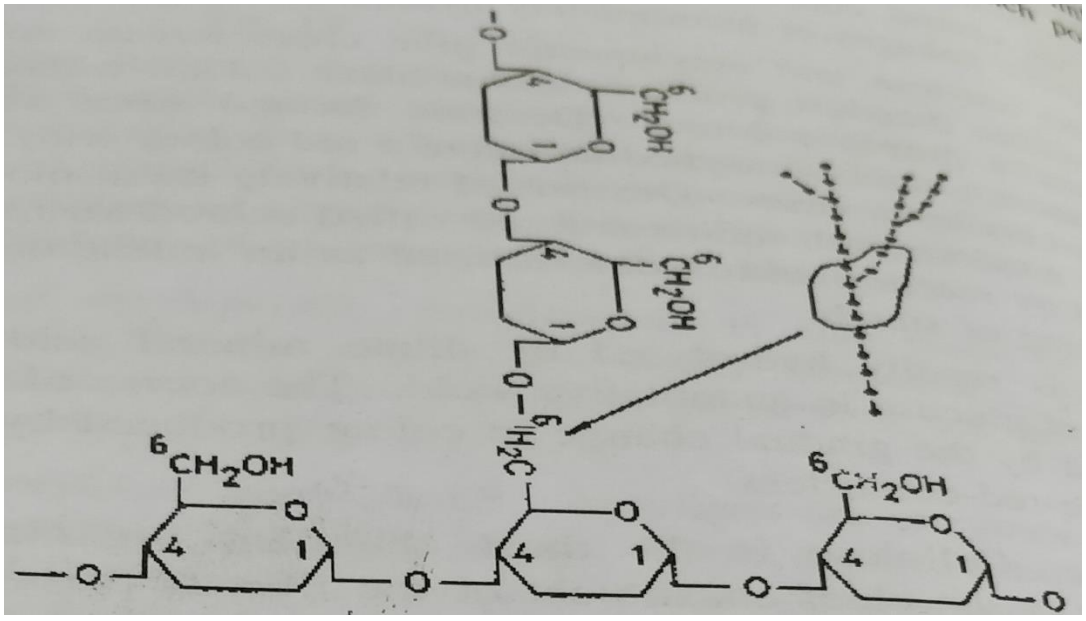
		Compound Lipid • Glycerophospholipids.,Sphingophospholipids, Glycolipids:. • Lipoprotiens: Contain protiens • Sulpholipids • Aminolipids • Lipoprotiens: Contain protiens • Sulpholipids: • Aminolipids: Derived Lipids: • Eg: Alcohols, Glycerol, Fatty acids etc Miscellaneous Lipids: • Eg : Carotenoids, Squalene. Neutral Lipids: • They are mono, di, triacylglycerols, cholesterol, cholesteryl esters. **Schematic representation can be considered**	
2	**e)**	**Explain any six biological functions of Calcium.** **Calcium is involved in:** • Formation & development of bones &teeth • Muscle contraction • Blood clotting • Growth of children • Transmission of nerve impulse • Activation of enzymes • Regulation of permeability of membranes • Release of hormones • Cell to cell contact & adhesion of cells in a tissue • Calcium acts on myocardium & prolongs systole.	**3M for any six functions**

<table>
<tr><td>2</td><td>f)</td><td>Give significance of abnormal constituents of urine (any six)
<table>
<tr><th>Abnormal constituents</th><th>Significance in Associated ailment</th></tr>
<tr><td>Sugar (glucose)</td><td>Glycosuria- Diabetes mellitus</td></tr>
<tr><td>Ketone bodies</td><td>Ketonuria- Diabetes mellitus, Pregnancy, Carbohydrate starvation</td></tr>
<tr><td>Albumin</td><td>Proteinuria- Pregnancy, severe exercise, high protein meal, Nephritis</td></tr>
<tr><td>Bile pigments</td><td>Jaundice /Hepatitis</td></tr>
<tr><td>Blood</td><td>Haematuria- Acute inflammation of urinary organs, T.B., Cancer, Haemolytic jaundice etc.</td></tr>
<tr><td>Pus</td><td>Pyuria- Inflammation of urinary bladder, urethra, kidney</td></tr>
</table></td><td>3M</td></tr>
<tr><td>3</td><td></td><td>Attempt any FOUR of the followings</td><td>12M</td></tr>
<tr><td>3</td><td>a)</td><td>Give pharmaceutical & therapeutic use of enzymes.
Pharmaceutical use of enzymes-
• Rennin is used for cheese preparation
• Glucose isomerase is used for production of syrup.
• Alpha amylase is used in food industry to covert starch to glucose
• Penicillin acylase is used for production of 6- amino pencilanic acid
• Papain, pepsin and trypsin are used in preparation of digestants.
Therapeutic use of enzymes-
• Trypsin: Purified enzyme is used orally or parenterally or intramuscularly in treatment of acute thrombophlebitis
• Streptokinase: Bacterial enzyme causes fibrinolysis & dissolution of clot.</td><td>3M
(Any 3 points can be considered in each use -1.5 M)</td></tr>
</table>

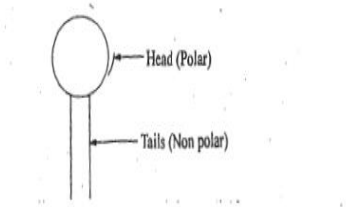
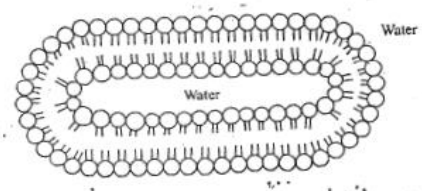
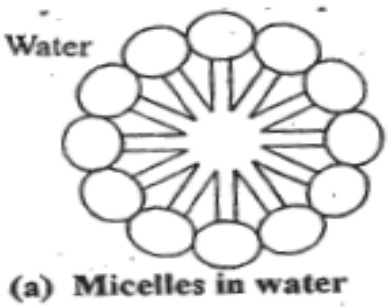
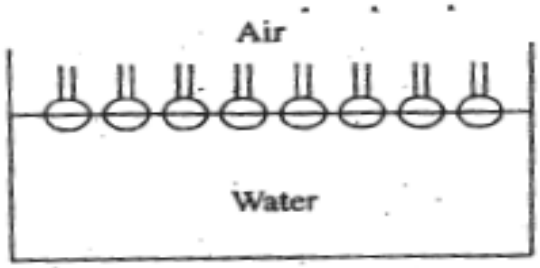
		• Pepsin is used in treatment of gastric achylia • Lysoenzyme useful in treatment of eye infection • Galactosidase useful in treatment of lactose intolerance. • Sulphanilamide because of its similarity with PABA competes with it & inhibits enzyme folic acid synthatase & selectively kills pathogenic organisms. • Allopurinol acts as competitive inhibitor of xanthin &reduces its conversion to uric acid .So it is useful in treatment of gout. **Other correct related examples can be considered**	
3	**b)**	**Explain 'Coris' cycle and give its biological importance**. The cycle involving the synthesis of glucose in liver from the skeletal muscle lactate and the reuse of glucose thus synthesized by the muscle for energy purpose is known as Cori cycle. Lactate produced by the active skeletal muscle is a major precursor for the process of gluconeogenesis. Under anaerobic conditions, pyruvate is reduced to lactate by lactate dehydrogenase (LDH) $\text{Pyruvate} + \text{NADH} + H^+ \xleftrightarrow{\text{LDH}} \text{Lactate} + NAD^+$ Lactate is a dead end in glycolysis, since it must be reconverted to pyruvate for its further metabolism. The plasma membrane is freely permeable to lactate. Lactate is carried from the skeletal muscle through blood and handed over to liver, where it is oxidized to pyruvate. Pyruvate, so produced, is converted to glucose by gluconeogenesis, which is then transported to the skeletal muscle. **Biological importance-** The cycle's importance is based on the prevention of lactic acidosis in the muscle under anaerobic conditions. However, normally before this happens the lactic acid is moved out of the muscles and into the liver. The cycle is also important in producing ATP, an energy source, during muscle activity.	**3M (Explanation and diagram-2M, biological importance-1M)**

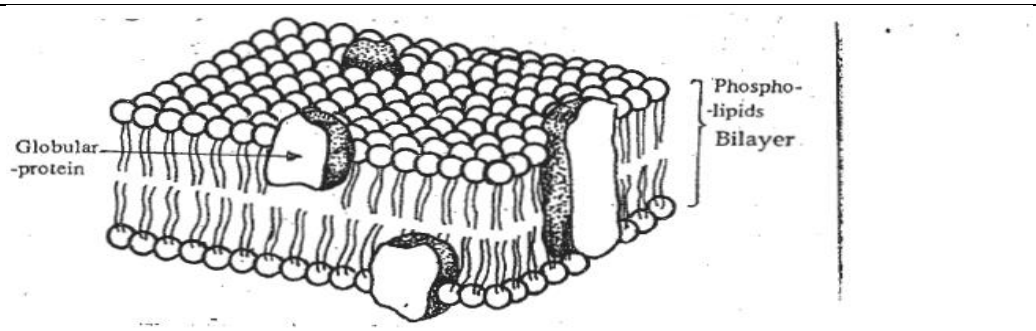
			
3	**c)**	**How ammonia is produced in the body? Enlist different ways of disposal of ammonia from the body.** Ammonia is produced in the body by the metabolism of amino acids and other nitrogenous compounds. At physiological pH, ammonia exists as ammonium ion ($NH4^+$). Ammonia is produced from the amino acids by transamination and Deamination, from biogenic amines, amino group of purines and pyrimidines and by the action of intestinal bacteria (urease) on urea. Disposal of ammonia: The organisms, during the course of evolution have developed different mechanisms for the disposal of ammonia from the body. The animals in this regard are of three different types: 1. Ammoniotelic- The aquatic animals dispose of ammonia in to the surrounding water. 2. Uricotelic- Ammonia is converted mostly to uric acid. Eg: reptiles and birds. 3. Ureotelic- The mammals including man convert ammonia into urea. Urea is non-toxic and soluble compound, hence easily excreted.	**3M (Each explanation- 1.5M)**

3	**d)**	**What are lipid storage diseases? Explain arteriosclerosis.** Definition- Lipid storage diseases, or the lipidosis, are a group of inherited metabolic disorders in which harmful amounts of fatty materials (lipids) accumulate in various cells and tissues in the body. Examples: Obesity, Arteriosclerosis, Niemann-pick disease, Farber's disease, Gaucher's disease etc. **Arteriosclerosis** is a complex disease characterised by thickening or hardening of arteries due to accumulation of lipids (particularly cholesterol, free and esterified) collagen, fibrous tissue, proteoglycans, calcium deposit etc in the inner arterial wall. Arteriosclerosis is a progressive disorder that narrows and ultimately blocks the arteries. Coronary arteries-the arteries supplying blood to the heart are the most commonly affected leading to myocardial infarction or heart attacks. The development of arteriosclerosis & risk of coronary heart disease (CHD) is directly correlated with plasma cholesterol and LDL (bad cholesterol).On the other hand, plasma HDL is inversely correlated with CHD. Certain diseases which are associated with arteriosclerosis include diabetes mellitus, hypothyroidism, hyperlipoproteinaemia. Causes of arteriosclerosis: Obesity, excessive smoking, lack of exercise, hypertension, stress and high consumption of saturated fats etc.	**3M (Definition -1M and explanation -2M)**
3	**e)**	**Explain biological role of carbohydrates.** 1) They are the most abundant dietary source of energy (4 Cal/gm) for all organisms. 2) Carbohydrates are precursors for the synthesis of organic compounds like nucleic acids and amino acids. 3) Carbohydrates (as glycoproteins and glycolipids) participate in the structure of cell membrane and cellular functions such as cell growth, adhesion and fertilization. 4) Carbohydrates also serve as the storage form of energy (glycogen) to meet the immediate energy demands of the body. 5) Carbohydrates are structural components of many organisms like exoskeleton of some	**3M (any 6 points - 1/2M each)**

		insects. 6) Help in breakdown of fatty acids and prevents ketosis. 7) Carbohydrates are the raw materials used for several industries like paper, plastics, textiles, alcohol etc. 8) Provide dietary fibre.	
3	**f)**	**Define polysaccharides. Explain the structure of glycogen.** **Definition:-** Carbohydrates which yield more than ten molecules of monosaccharides on hydrolysis are generally termed as polysaccharides. General formula is $(C_6H_{10}O_5)_n$. **Structure of Glycogen**: - Glycogen is the reserved carbohydrates in the animals and is found in significant amount in liver and muscle. Glycogen is made up of D-glucose residues. Upon hydrolysis it yields D-glucose as the product. Glycogen is a highly branched polysaccharide and resembles amylopectin in structure. Glycogen is a branched biopolymer consisting of linear chains of glucose residues with an average chain length of approximately 8–12 glucose units. Glucose units are linked together linearly by α (1→4) glycosidic bonds from one glucose to the next. Branches are linked to the chains from which they are branching off by α (1→6) glycosidic bonds between the first glucose of the new branch and a glucose on the stem chain. The structure is given below. 	**3M (Define- 1M and structure of glycogen with explanation- 2M)**

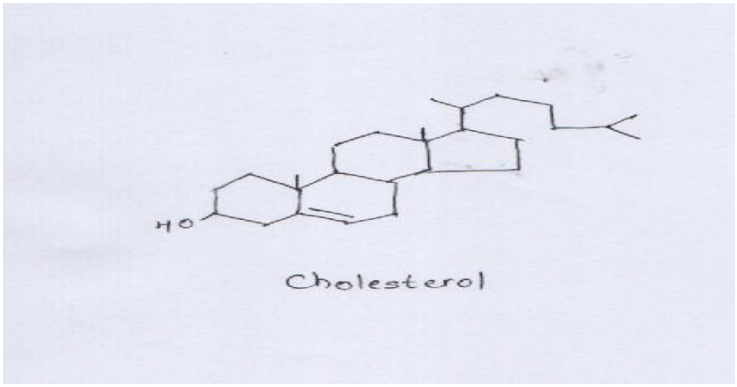
4		**Attempt any FOUR of the followings**	**12M**
4	**a)**	**Explain any one protein deficiency disease.** The protein deficiency diseases are:- Kwashiorkar Marasmus Nutritional edema **Kwashiorkar-**It is predominantly found in children between 1-5 yrs. It is due to insufficient intake of proteins as the diet of a weaning child consists of carbohydrate. Symptoms: Stunted growth, Edema on legs & hands, Diarrhoea , Discoloration of hair skin, Anemia , Apathy, Moon face, Decreased plasma albumin concentration. Treatment: Protein rich food. **Marasmus-** Occurs in children below 1 yr age. Symptoms: Growth retardation, Muscle wasting, Anaemia , Weakness, No edema ,No decreased concentration of plasma albumin Treatment: Mother's milk. **Nutritional Edema-** Results from long continued deprivation of proteins & usually occurs in famine areas. This Protein deficiency in adults is very rare. Symptoms: Weight loss, General lethargy, Frequent loose stools, Delay in wound healing, Edema Treatment: Food items like soyabean, milk, eggs.	**3M (Types-1M.Any one disease-2M)**
4	**b)**	**Define the following:** i) Polensky value: It is the number of milliliter of 0.1 N KOH required to neutralize the insoluble fatty acids from 5gm of fat or oil. It is an indicator of how much volatile and insoluble fatty acids present in total fats and oil. ii) Iodine value: It is the number of grams of iodine required to saturate or absorbed by 100gms of fat or oil. It helps to determine level of unsaturated fatty acids present in total fat or oil.	**3M (Each definition-1M)**

		iii) Sap value: It is the number of milligrams of KOH required to saponify free or combined fatty acids present in 1 gram of fat or oil. It is a measure of mean molecular weight of all fatty acids present in fat or oil.	
4	**c)**	**Explain the role of lipids in biological membrane with the help of models.** The major component of biological membrane is phospholipid. Phospholipid has 2 long chains of hydrocarbon of fatty acids. The chains are hydrophobic and have strong polar group i.e. phosphate at 3rd carbon of glycerol. When phospholipids are added to aqueous medium they form micelles, monolayer & bilayer, depending on the concentration of Phospholipids. The hydrophilic & hydrophobic interaction of phospholipids is forming bilayer in water. Hydrophobic tails are hidden from aqueous environment and form an internal hydrophobic phase whereas hydrophilic heads are exposed to the surface. Bilayer system of this type is extensively studied as model of natural membrane.  Fig. A molecule of phospholipid.  Fig. Phospholipid bilayer.  (a) Micelles in water  (b) Monolayer at air-water interface	**3M (Explanation -1M & Diagrams - 2M for all)**

		 Fig. Fluid mosaic model of plasma membrane Biomembranes are made up phospholipids lipoproteins, glycoproteins and proteins all these components are assembled together by non covalent interactions.	
4	**d)**	**Define dehydration. Explain causes, symptoms & treatment of dehydration.** **Definition**- It is a condition characterized by water depletion in the body. It may be due to loss of water alone or due to deprivation of water & electrolytes. **Causes** :Diarrhoea, vomiting, excessive sweating , fluid loss in burns, adrenocortical dysfunction, Kidney diseases, Cholera **Symptoms**: Increased pulse rate, low blood pressure, sunken eyeballs, decreased skin elasticity, lethargy, confusion & ultimately coma. **Treatment :** i) Intake of plenty of water . ii) If a person can't take orally water be given I.V.ly in an isotonic solution (5%glucose) iii) If dehydration is due to loss of electrolytes, then electrolytes can be given orally or intravenously.	**3M (Definition -1/2M causes 1/2M, sym. 1M, treat.1M)**
4	**e)**	**What are co-enzymes? Name co-enzymes of the following vitamins:** Co-enzymes are the organic molecules often derived from vitamin B complex group that participate directly in enzymatic reaction. Many enzymes catalyze the reactions only in presence of specific non protein organic molecules called the co-enzyme. i) Thiamin TPP (Thiamine pyrophosphate)	**3M Definition- 1M and co-enzymes 1/2M each)**

		ii) Pyridoxine PLP (Pyridoxal phosphate) ,PP (Pyridoxine phosphate) iii) Riboflavin FMN (Flavin mononucleotide) and FAD (Flavin adenine dinucleotide) iv) Nicotinamide NAD^+ (Nicotinamide adenine dinucleotide) and $NADP^+$ (Nicotinamide adenine dinucleotide phosphate)	
4	**f)**	**Explain causes, symptoms & treatment of the following diseases:** **i) Scurvy** Causes: Deficiency of vitamin C Symptoms: Weakness, pain in bones and joints , loosening of teeth, poor healing of wound, internal haemorrhage, swelling of long bone, Easy factorability of bones. Scurvy leads to the formation of spots on the skin, spongy gums, and bleeding from all mucous membranes. The spots are most abundant on the thighs and legs, and a person with the ailment looks pale, feels depressed, and is partially immobilized. In advanced scurvy there are open, suppurating wounds and loss of teeth, sluggish hormonal function of adrenal cortex, swollen joints, osteoporosis. Treatment: Treatment involves taking vitamin C supplements and eating citrus fruits, potatoes, broccoli and strawberries. **ii) Pellagra** Causes: Deficiency of niacin (B3) and protein especially proteins containing the essential amino acid tryptophan. Because tryptophan can be converted into niacin, foods with tryptophan but without niacin, such as milk, prevent pellagra. Symptoms: High sensitivity to sunlight , dermatitis, alopecia, oedema, red skin lesions ,mental confusion, diarrhoea, eventually dementia Treatment: Pellagra can be treated with niacin (usually as niacinamide). The frequency and amount of niacinamide administered depends on the degree to which the condition has progressed.	**3M (Each disease 1.5M each)**

5		**Solve any FOUR of the followings**	**12M**
5	**a)**	**Explain functions and pathology of lymphocytes and platelets.** Functions of lymphocytes.(any two) 1.These produce antitoxins and antibodies 2. They help in healing of wounds. 3. Play a key role in immunity. Pathology of lymphocytes: (any 1) Lymphocytosis: Increase in number of lymphocytes count above normal range in blood & is observed in viral infection like Hepatitis A, Bordetella pertusis. Lymphopenia : Decrease in number of Lymphocytes below the normal value & is observed in CHF and temporary conditions of administration of adrenocorticosteriod hormones. Functions of platelets (any two) 1.Initiate blood clotting 2.Repair capillary endothelium 3.Involved in haemostatic mechanism Pathology of platelets (any 1) Thrombocytopenia. Decrease in numbers of platelets below the normal range and is observed Leukaemia, aplastic anaemia, megaloblastic anaemia, dengue , malaria etc. Thrombocythemia : Increase in numbers of platelets below the normal range and	**3M (1.5 M each)**

		is observed in conditions like acute haemorrhage, leukaemia etc	
5	**b)**	**Give structure and two colour reactions of cholesterol** **(Any two reactions)** 1) Liebermann-Burchard test: When chloroform solution of cholesterol is treated with acetic anhydride & concentrated sulphuric acid, green colour is formed. 2) Salkowaski Test: When chloroform solution of cholesterol is treated with concentrated sulphuric acid, upper layer gives red colour and H_2SO_4 layer gives green colour. 3) Formaldehyde-H2SO4 Test: To a solution of cholesterol in chloroform in dry test tube, if 2ml of formaldehyde-sulphuric acid solution is added, cherry colour develops.  	**3M** **1Mstr.** **2M for reactions**
5	**c)**	**Define Compound Lipids. Explain any two important biological functions of Phospholipids.** **Compound Lipid:** These are esters of fatty acids containing groups such as phosphate nitrogenous base, carbohydrates, proteins etc in addition to an alcohol and a fatty acids. Examples : Phospholipids (lecithin, cephalin), Glycolipids. Lipoproteins etc. **Functions: (any two)** 1. Phospholipids form structural components of membrane& regulate membrane permeability. 2.Phospholipids are responsible for maintaining conformation of electron transport chain	**3M** **(1M def.** **2M fun.)**

components & so cellular respiration

3.Phospholipids participate in absorption of fat from intestine & also transport of lipids

4.Phospholipids act as surfactants

5. They are involved in signal transmission across membranes.

6. Cephalins participate in blood clotting.

5 d) Explain the following colour reactions. **3M (1M each)**

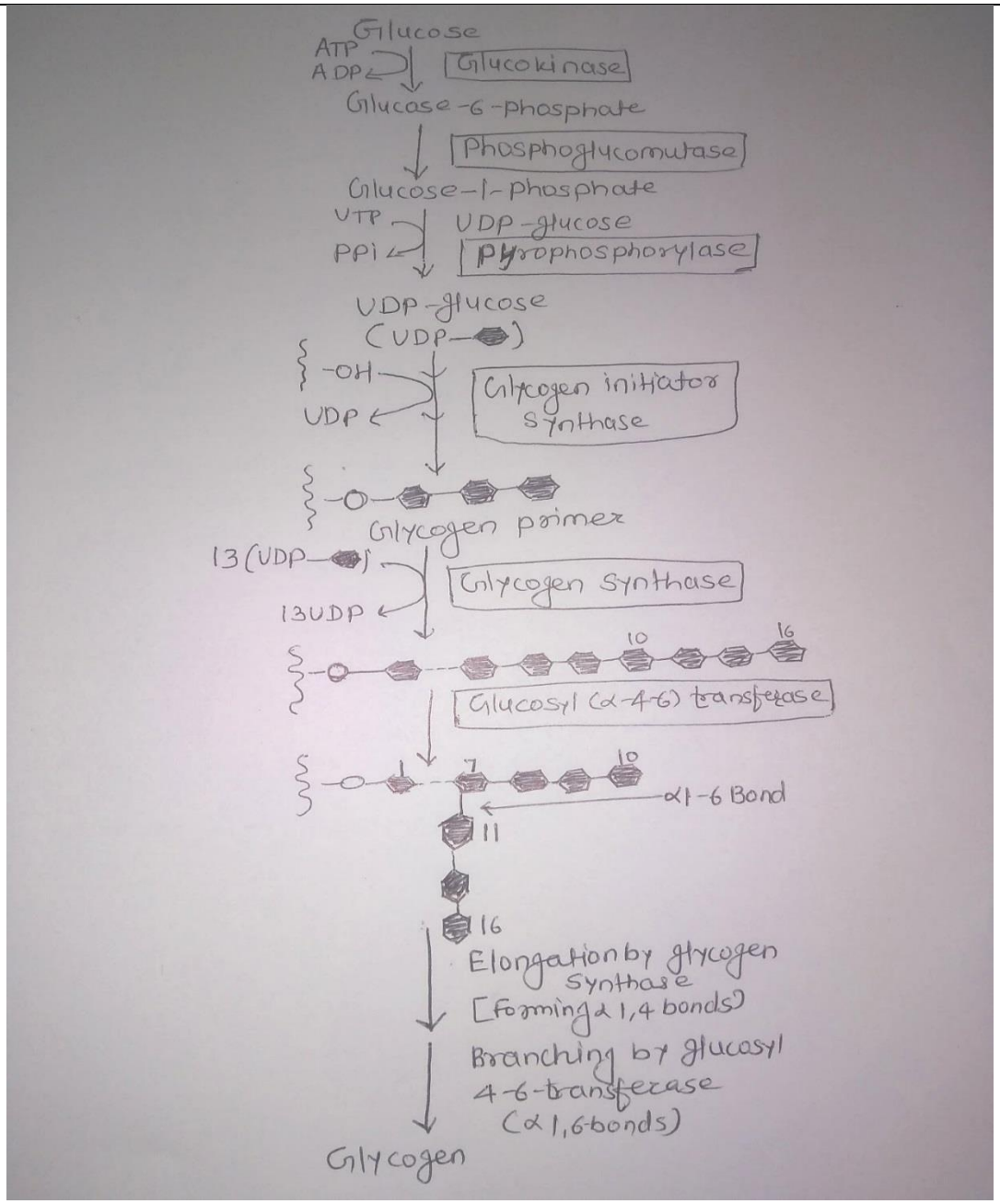
Sr. No.	**Test**	**Observation**	**Inference**
(i)Seliwanoff's Test	Seliwanoff reagent + sugar solution, boil for two mins.	Red color or ppt	Ketones present like fructose or sucrose.
(ii) Ninhydrin reaction	Protein solution + Ninhydrin solution. Boil for two mins. And cool.	Blue colour	Amino acids or proteins present.
(iii)Newman's test	Protein solution + 40% NaOH . Heat for 1 min. And cool. Then add conc. HNO_3 + ammonium molybdate solution and heat.	Cannery yellow colour	Casein confirmed

5 e) **Discuss-** **3M (1.5 M each)**

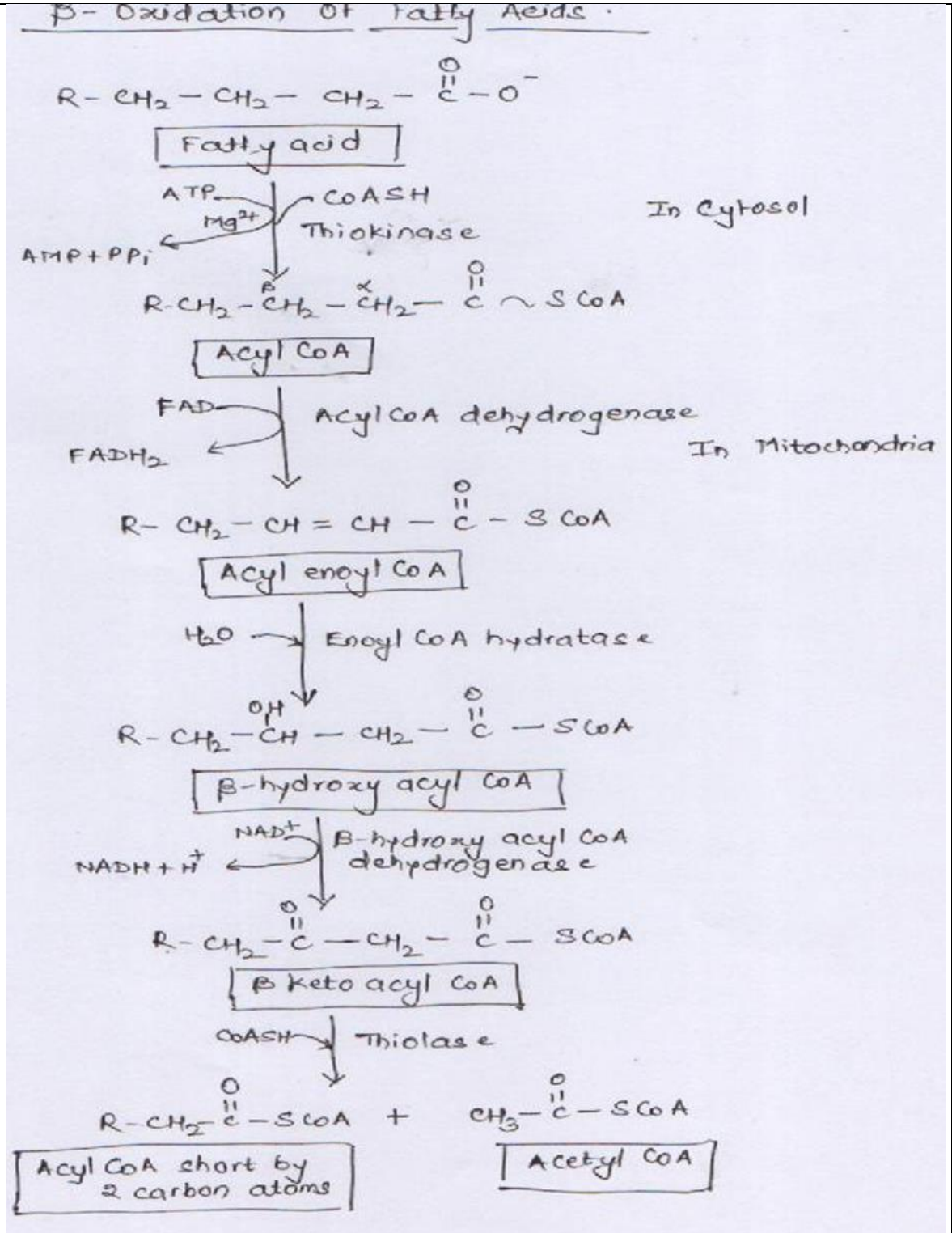
(i) Pernicious anaemia:

In this type of anaemia essential factors are absent which are required for the formation of RBC. So RBC count is decreased i.e. intrinsic factors responsible for absorption of

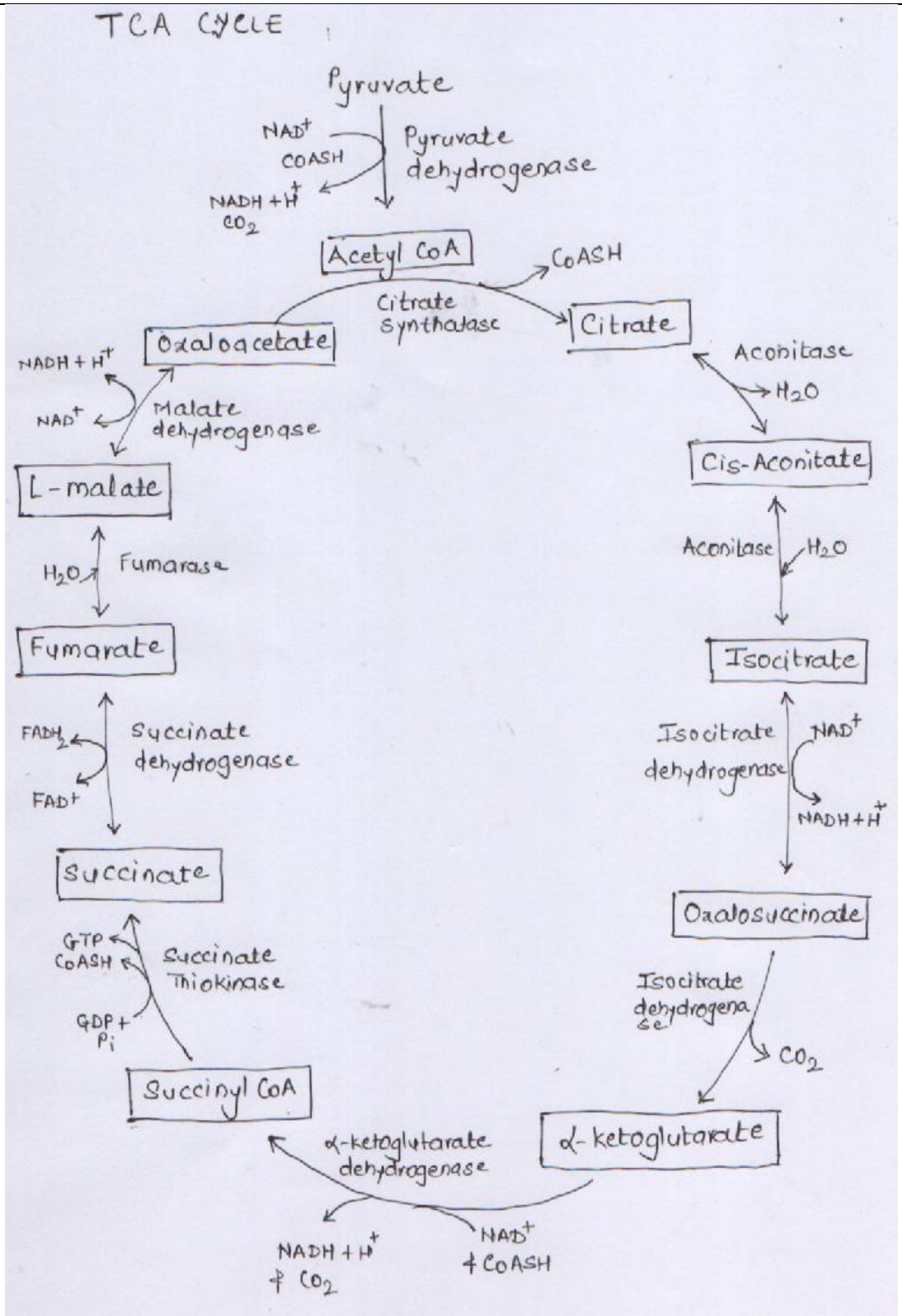
		vitamin B_{12} from gastric acid are absent. Symptoms: Shortness of breath, Tired feeling, Numbness, tingling of fingers, neuronal degeneration, confusion etc. Treatment : Vitamin B_{12} IM **iii) Sickle cell anaemia:** It is genetic disorder. Bone marrow produces abnormal type of cells. The shape of large number of red cells is like sickle cell or crescentric and the life span is completely shortened. Patients with sickle cell show marked susceptibility to infection and there is blockage of blood supply to vital organs as sickle cells don't pass through small blood capillaries. These patients should avoid places with low oxygen supply. Symptoms : Susceptibility to infection ,improper blood supply to vital organs etc. Treatment : 1 .Avoid going to higher altitude where oxygen supply is less. 2 .Blood transfusion in severe cases.	
5	**f)**	**Define & explain Glycogenesis. Give in brief, importance of the process.** **Definition** Of Glycogenesis: It is the process of conversion of glucose into glycogen in the liver. It takes place in the cytosol , requires ATP and UTP, besides glucose. **Importance of glycogenesis**: 1. Excess of glucose is utilised to form glycogen which is stored in liver and muscles reserved for muscular activities. 2. Helps to maintain blood glucose level. 3. In case of carbohydrate starvation stored glycogen is converted to glucose to give energy. **Explanation: Diagrammatic presentation can also be considered** 1. Synthesis on UDP – Glucose 2. Requirement of primer to initiate glycogenesis. 3. Glycogen synthesis by Glycogen synthase. 4. Formation of branches in glycogen.	**3M** **(1M each)**


Glucose
ATP
ADP
Glucokinase
Glucose-6-phosphate
Phosphoglucomutase
Glucose-1-phosphate
UTP
PPi
UDP-glucose
Pyrophosphorylase
UDP-glucose
(UDP—)
-OH
UDP
Glycogen initiator synthase
Glycogen primer
13(UDP—)
13UDP
Glycogen Synthase
10
16
Glucosyl (α-4-6) transferase
1
7
10
α1-6 Bond
11
16
Elongation by glycogen synthase
[Forming α 1,4 bonds]
Branching by glucosyl 4-6-transferase
(α 1,6-bonds)
Glycogen

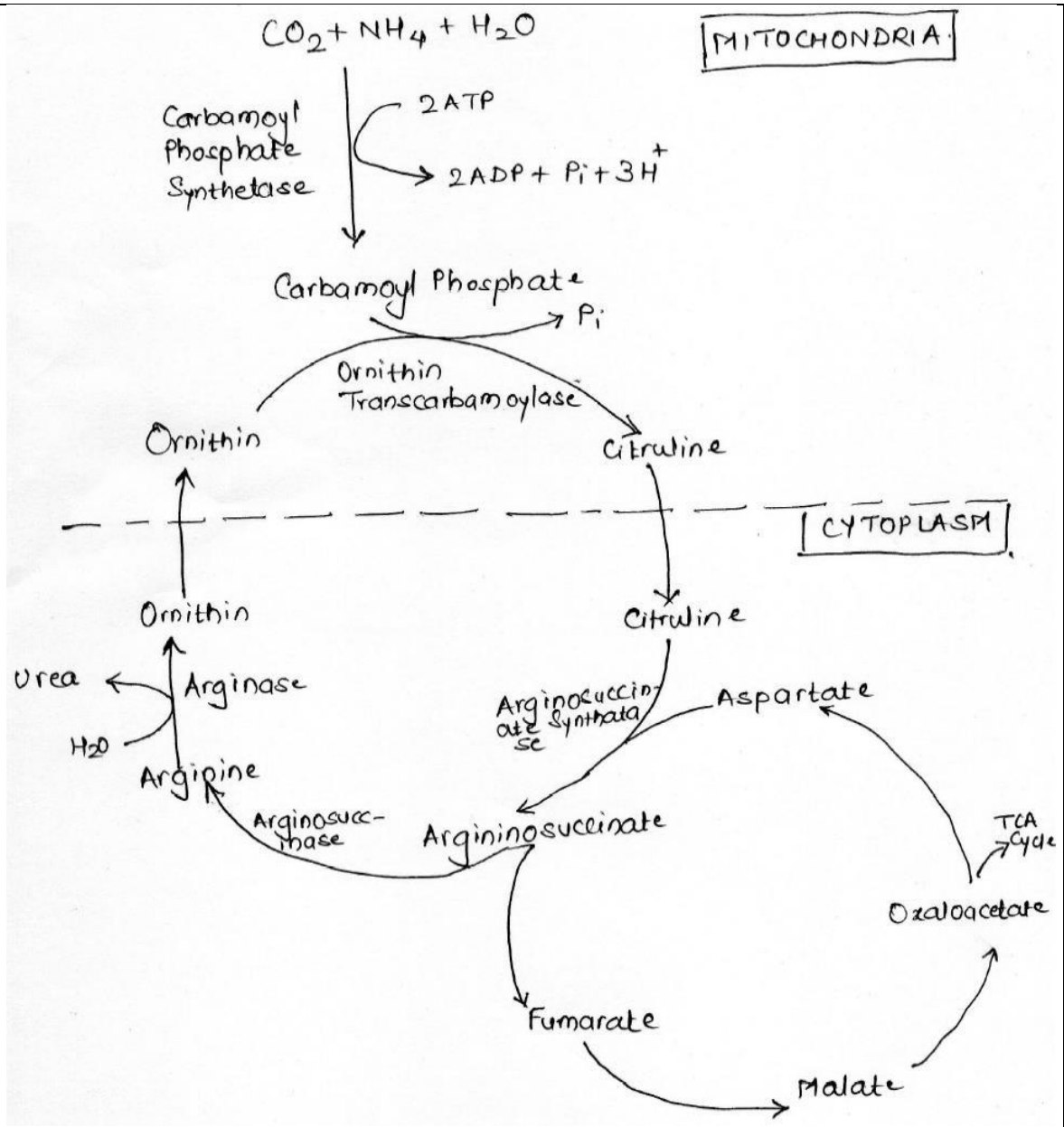
6		**Solve any FOUR of the followings**	**16M**
6	**a)**	**Explain reactions of beta oxidation of fatty acids.** **(Detailed diagrammatic representation can be considered for full marks)** Beta oxidation is the main pathway used to liberate energy by oxidation of fatty acid It takes place in the beta carbon of fatty acid with removal of 2 carbons at a time from the carboxyl end of the molecule. The process repeats itself until the fatty acid with even number of carbon is completely converted to acetate molecules. Fatty acid containing even & odd number of carbon atoms as well as unsaturated fatty acids are oxidised by beta oxidation. It takes place in 5 steps in mitochondria of liver. 1. Activation of fatty acid. Long chain fatty acid gets activated to fatty acyl CoA in presence of CoASH, thiokinase&ATP 2. Fatty acylCoA undergoes dehydrogenation in presence of acyl CoA dehydrogenase &FAD to give alpha,beta unsaturated fatty acyl CoA 3. Addition of water molecule across the double bond results into formation of Beta hydroxy acyl CoA in presence of Enoyl CoA dehydratase 4. Hydroxyl group of Beta hydroxy acyl CoA gets oxidised to keto group forming Beta keto acyl CoA in presence of Beta hydroxy acyl CoA dehydrogenase & NAD+ 5. Thiolytic cleavage of acyl CoA takes place in presence of Beta keto acyl CoA Thiolase&CoASH. Acyl CoA thus formed contains 2 Carbons less than original acyl CoA which undergoes further oxidation by Beta-oxidation. Acetyl CoA is also formed which enters TCA cycle.	**4M**


β- Oxidation of Fatty Acids :
R- CH2 - CH2 - CH2 - C(=O)-O⁻
Fatty acid
ATP
Mg2+
CoASH
Thiokinase
In Cytosol
AMP + PPi
R-CH2-CH2-CH2-C(=O)~SCoA
Acyl CoA
FAD
Acyl CoA dehydrogenase
FADH2
In Mitochondria
R-CH2-CH=CH-C(=O)-SCoA
Acyl enoyl CoA
H2O
Enoyl CoA hydratase
R-CH2-CH(OH)-CH2-C(=O)-SCoA
β-hydroxy acyl CoA
NAD+
NADH + H+
β-hydroxy acyl CoA dehydrogenase
R-CH2-C(=O)-CH2-C(=O)-SCoA
β Keto acyl CoA
CoASH
Thiolase
R-CH2-C(=O)-SCoA + CH3-C(=O)-SCoA
Acyl CoA short by 2 carbon atoms
Acetyl CoA

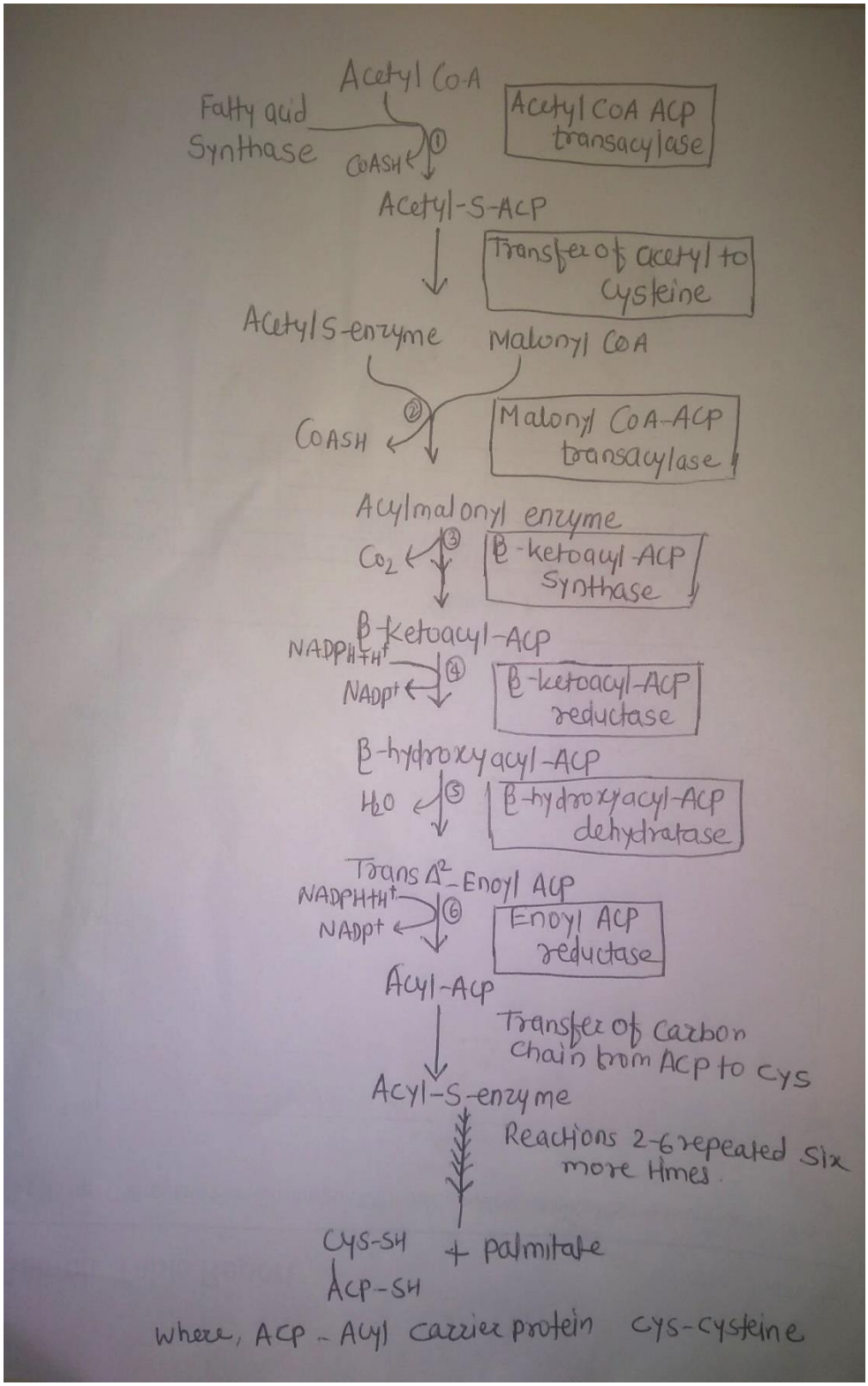
6	**b)**	**Explain reactions of Kreb's cycle.** **(Detailed diagrammatic representation can be considered for full marks)** Kreb's cycle: It's a central pathway for release of energy from acetyl CoA whch is produced from glycolysis, catabolism of fatty acids or amino acids 1. Condensation of acetylCoA obtained from pyruvic acid with oxaloacetate to form citric acid in presence of citrate synthatase 2. Conversion of citric acid to cis aconitate in presence of aconitase&fe2+ 3. Cis acotinic acid accepts water to give isocitric acid in presence of acotinase& Fe2 4. Isocitric acid undergoes oxidation in presence of isocitric dehydrogenase & NAD+ to give Oxalosuccinic acid 5. Decarboxylation of oxalosucccinic acid to alpha ketoglutaric acid in presence of isocitri dehydrogenase, Mg/ Mn 6. Oxidative decarboxylation of alpha ketoglutaric acid to succinyl CoA in presence of alpha ketoglutarate dehydrogenase, CoA-SH, NAD+, Mg 7. SuccinylCoa gets converted to succinic acid in presence of succinate thiokinase, GDP, Mg 8. Succinic acid undergoes dehydrogenation in presence of succcinate dehydrogenase, FAD+ to form fumaric acid 9. Fumaric acid takes up water molecule in presence of fumarase to form maleic acid 10. Maleic acid undergoes oxidation in presence of malate dehydrogenase, NAD+ to form oxaloacetic acid. 11. Cycle gets repeated again by entrance of another molecule of Acetyl CoA	**4M**


TCA CYCLE
Pyruvate
NAD+
COASH
Pyruvate dehydrogenase
NADH + H+
CO2
Acetyl CoA
CoASH
Citrate synthatase
Oxaloacetate
Citrate
NADH + H+
NAD+
Malate dehydrogenase
Aconitase
H2O
L-malate
Cis-Aconitate
H2O
Fumarase
Aconitase
H2O
Fumarate
Isocitrate
FADH2
FAD+
Succinate dehydrogenase
Isocitrate dehydrogenase
NAD+
NADH + H+
Succinate
Oxalosuccinate
GTP
CoASH
GDP + Pi
Succinate Thiokinase
Isocitrate dehydrogenase
CO2
Succinyl CoA
α-ketoglutarate
α-ketoglutarate dehydrogenase
NADH + H+
+ CO2
NAD+
+ CoASH

6	**c)**	**Explain urea cycle in detail.** **(Detailed diagrammatic representation can be considered for full marks)** 1) Molecule of ammonia, CO2 & phosphate are condensed to form 'Carbamoyl phosphate' in presence of enzyme 'carbamoyl-phosphate synthetase.) Carbamoyl phosphate transferred to ornithine forms citrulline in presence of an enzyme ornithine transcarboxylase. This reaction takes place in mitochondria. The citrulline formed in this reaction enters in cytoplasm & the next reactions take place in cytoplasm 3) Citrulline condenses with Aspartate to form argininosuccinate. The reaction is catalysed by an enzyme Arginosuccinatesynthetase. 4) Arginosuccinate is now cleaved into 'arginine' & 'fumarate' by the enzyme 'arginosuccinase'. Fumarate formed may be converted to oxaloacetate via the actions of enzymes 'fumerase'& malate dehydrogenase & then transmitted to regenerate aspartate. 5) Finally arginine is cleaved into ornithine & urea by the enzyme arginase. With this reaction cycle is completed & ornithine molecule accepts molecule of carbamoyl phosphate to repeat the cycle. the overall equation of the urea cycle is: NH3 + CO2 + aspartate + 3 ATP + 2 H2O → urea + fumarate + 2 ADP + 2 Pi + AMP + PPi	**4M**

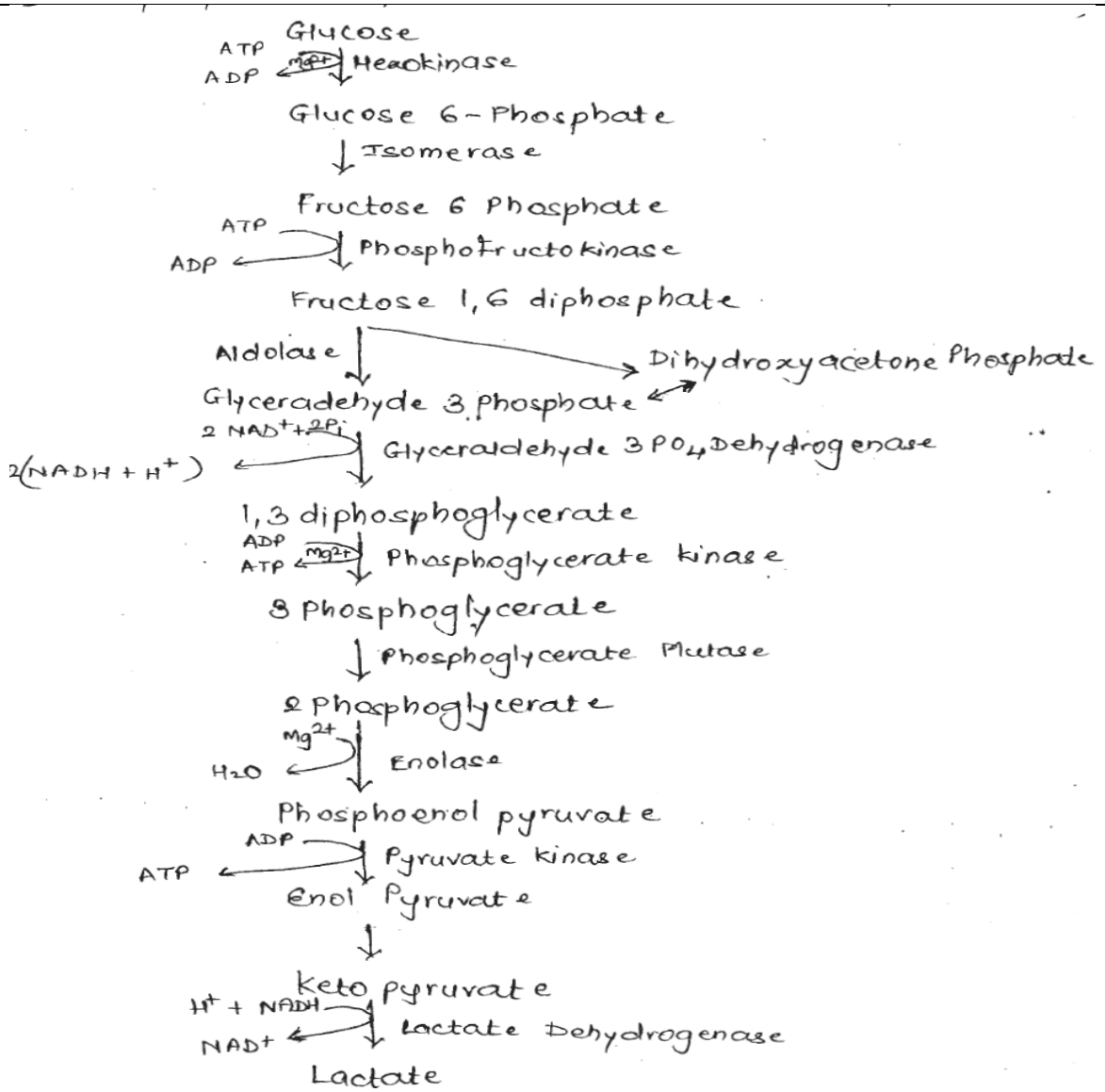

$CO_2 + NH_4 + H_2O$
MITOCHONDRIA
Carbamoyl Phosphate Synthetase
2ATP
2ADP + Pi + 3H+
Carbamoyl Phosphate
Pi
Ornithin Transcarbamoylase
Ornithin
Citrulline
CYTOPLASM
Ornithin
Citrulline
Urea
Arginase
H2O
Arginine
Argininosuccinate Synthetase
Aspartate
Arginosuccinase
Argininosuccinate
TCA Cycle
Oxaloacetate
Fumarate
Malate

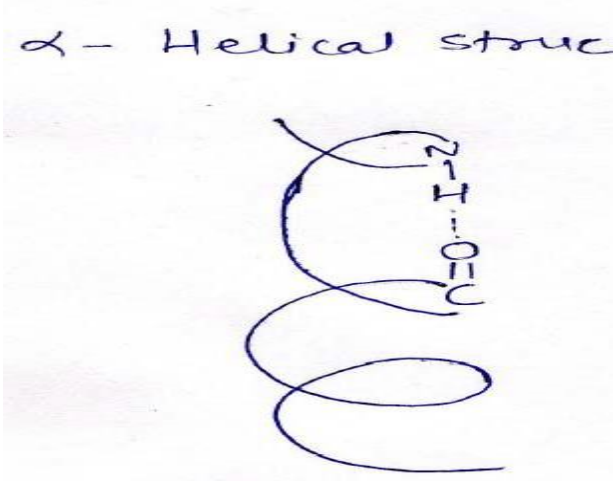
6	**d)**	**Discuss extra mitochondrial fatty acid synthesis.** **(Detailed diagrammatic representation can be considered for full marks)** The sequence of reactions for extra mitochondrial synthesis of fatty acid (palmitate) is described below. 1. The two carbon fragment of acetyl CoA is transferred to ACP of fatty acid synthase, catalysed by the enzyme acetyl CoA-ACP transacylase. The acetyl unit is then transferred from ACP to cysteine residue of the enzyme. Thus ACP site falls vacant. 2. The enzyme malonyl CoA-ACP transacylase transfers malonate from malonyl CoA to bind to ACP. 3. The acetyl unit attached to cysteine is transferred to malonyl group (bound to ACP). The malonyl moiety loses CO2 which was added by acetyl CoA carboxylase. Thus CO2 is never incorporated into fatty acid carbon chain. 4. β -Ketoacyl ACP reductase reduces ketoacyl group to hydroxyacyl group. The reducing equivalents are supplied by NADPH. (From HMP shunt). 5. β -Hydroxyacyl ACP undergoes dehydration. A molecule of water is eliminated & a double bond is introduced between α & β carbons. 6. A second NADPH-dependent reduction, catalysed by enoyl-ACP reductase occurs to produce acyl-ACP. The four-carbon unit attached to ACP is butyryl group. The carbon chain attached to ACP is transferred to cysteine residue & the reactions of malonyl CoA-ACP transacylase & enoyl-ACP reductase are repeated 6 more times. Each time, the fatty acid chain is lengthened by a two-carbon unit (obtained from malonyl CoA). At the end of 7 cycles, the fatty acid synthesis is complete & a 16-carbon fully saturated fatty acid-namely palmitate-bound to ACP is produced. 7. The enzyme palmitoyl thioesterase separates palmitate from fatty acid synthase. This completes the synthesis of palmitate.	**4M**


Acetyl Co-A
Fatty acid Synthase
CoASH
Acetyl CoA ACP transacylase
Acetyl-S-ACP
Transfer of acetyl to cysteine
Acetyl S-enzyme
Malonyl CoA
CoASH
Malonyl CoA-ACP transacylase
Acylmalonyl enzyme
Co_2
β-ketoacyl-ACP Synthase
β-ketoacyl-ACP
$NADPH+H^+$
$NADP^+$
β-ketoacyl-ACP reductase
β-hydroxyacyl-ACP
H_2O
β-hydroxyacyl-ACP dehydratase
Trans Δ^2-Enoyl ACP
$NADPH+H^+$
$NADP^+$
Enoyl ACP reductase
Acyl-ACP
Transfer of carbon chain from ACP to cys
Acyl-S-enzyme
Reactions 2-6 repeated six more times.
Cys-SH
+ palmitate
ACP-SH
Where, ACP - Acyl carrier protein Cys-cysteine

6	**e)**	**Explain reactions of Glycolysis.** **(Detailed diagrammatic representation can be considered for full marks)** It's a main pathway for glucose oxidation 1. Phosphorylation of glucose to glucose 6 phospate in preseceofenzyme hexokinase & ATP & Mg 2. Isomerisation of Glucose 6 phosphate to fructose 6 phosphate in presence of phosphohexo isomerase 3. Phosphorylation of fructose 6 phosphate to fructose 1,6 diphosphate in presence of phosphofructokinase,ATP& Mg 4. Cleavage of fructose 1,6 diphosphate to dihydroxy acetone phosphate & glyceraldehyde 3 phosphate in presence of aldolase. These 2 products are interconvertible in presence of triose phosphate isomerase 5. Glyceraldehyde 3 phosphate further undergoes oxidation to 1,3 diphosphoglycerate in presence of glyceraldehyde 3 phosphate dehydrogenase & NAD+ 6. Transformation of 1,3 diphosphoglycerate to 3- phosphoglycerate in presence of phosphoglycerate kinase, Mg & ADP 7. 3- phosphoglycerate changes to 2-phosphoglycerate in presence of phosphoglycerate mutase 8. Loss of water molecule from 2-phosphoglycerate results into formation of phosphoenol pyruvic acid in presence of enolase 9. Loss of phosphate from phosphoenol pyruvic acid results into formation of Enol pyruvic acid in presence of pyruvate kinase, Mg & ADP	**4M**

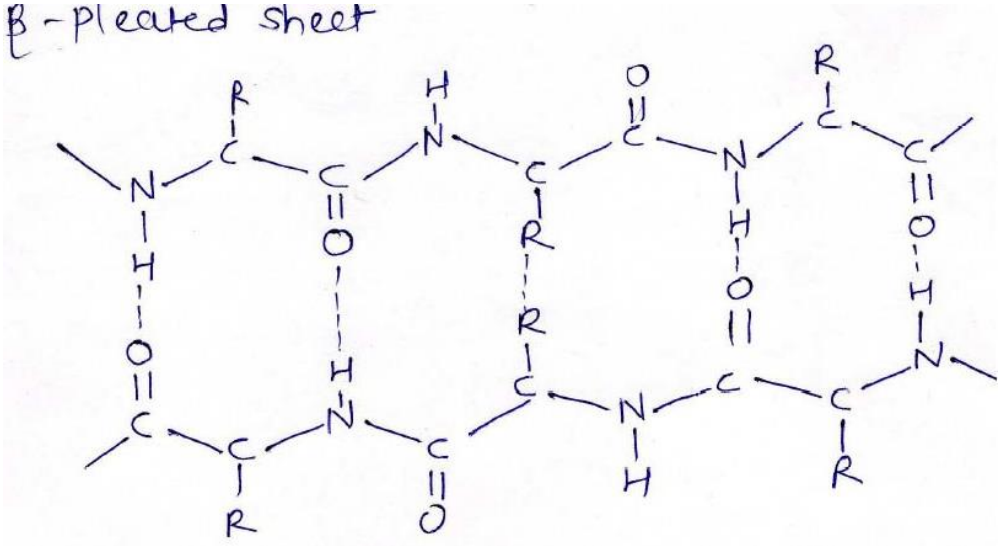
		10. Enol pyruvic acid gets converted to keto form of pyruvic acid in presence of pyruvate kinase 11. Keto pyruvic acid under aerobic conditions enter TCA cycle in mitochondria. Pyruvic acid forms main end product of glycolysis in those tissues which are supplied with sufficient Oxygen. 12. But tissues where oxygen is not supplied ,lactic acid is formed as an end product of glycolysis by reduction in presence of lactate dehydrogenase & NADH. Net reaction for glycolysis is: Glucose + 2NAD+ + 2 ADP + 2 Pi $\rightarrow$ 2 Pyruvate + 2 ATP + 2 NADH + 2 H_2O	


Glucose
ATP
ADP
Mg2+
Hexokinase
Glucose 6-Phosphate
Isomerase
Fructose 6 Phosphate
ATP
ADP
Phosphofructokinase
Fructose 1,6 diphosphate
Aldolase
Dihydroxyacetone Phosphate
Glyceradehyde 3 phosphate
2 NAD+ + 2Pi
2(NADH + H+)
Glyceraldehyde 3 PO4 Dehydrogenase
1,3 diphosphoglycerate
ADP
ATP
Mg2+
Phosphoglycerate kinase
3 Phosphoglycerate
Phosphoglycerate Mutase
2 Phosphoglycerate
Mg2+
H2O
Enolase
Phosphoenol pyruvate
ADP
ATP
Pyruvate kinase
Enol Pyruvate
Keto pyruvate
H+ + NADH
NAD+
Lactate Dehydrogenase
Lactate

6	**f)**	**Explain secondary structures of protein.** The conformation of polypeptide chain by twisting or folding is referred to as secondary structure . Two types of secondary structures are possible: i) **α- helix (α- helical)** : α helical is the most common spiral structure of protein. It has a rigid arrangement of polypeptide chain. The α - helical structure depends on the intramolecular hydrogen bonding between NH and **C=0**group of peptide bond, in the α - helix the polypeptide is folded in such a way that the C=O of each amino acid residue is hydrogen bonded to the NH of 4th amino acid residue along the chain.  (ii) **β-pleated sheet**: It is another form of secondary structure, this result from hydrogen bonding between two peptide chains. It may occur in two types a) Parallel pleated sheet: In this type of structure the polypeptide chain is side by side and in the same direction so that N-terminal residues are on the same end. This pleated sheet confirmation is stabilized by hydrogen bonding, here bonds are formed between NH group of a peptide in one chain and C=O group of a neighboring chain.	**4M**

b) anti- parallel pleated sheet-

In this type of structure the polypeptide chain lie in opposite direction so that N-terminal end of one and C- terminal of the other, face each other. In this structure the polypeptide chains are held together by hydrogen bonds, so as to give a sheet like structure and hence are called as β – pleated sheet confirmation.



Other correct representation can also be considered.