Chapter 2

Carbohydrates Metabolism

Q.1. What are the various pathways by which glucose is utilised?

Ans.
- Glycolysis followed by tricarboxylic acid cycle
- Hexose monophosphate shunt pathway
- Glycogenesis (synthesis of glycogen from glucose)
- Conversion to galactose and then to lactose
- Conversion to fat
- Gives rise to non-essential amino acids

Q.2. Which is the main sugar of the blood?

Ans. Glucose

Q.3. Is glucose easily permeable through the cellular membrane?

Ans. Yes

Q.4. Is liver or muscle glycogen easily available for metabolism?

Ans. Liver glycogen

Q.5. What is glycolysis?

Ans. **Glycolysis** is the metabolic pathway that converts glucose $C_6H_{12}O_6$, into pyruvic acid, $CH_3COCOOH$. The free energy released in this process is used to form the high-energy molecules, i.e. adenosine triphosphate and reduced nicotinamide adenine nucleotide.

Glycolysis is a sequence of ten reactions catalyzed by various enzymes.

Q.6. What are the key (regulatory) enzymes of glycolytic pathway?

Ans. Hexokinase, phosphofructokinase (PFK) and pyruvate kinase.

Q.7 Name the most important regulatory enzyme of glycolysis.

Ans. PFK-1

Q.8. What is the difference between PFK-1 and PFK-2?

Ans. The key difference between PFK-1 and PFK-2 is that **PFK-1** catalyzes the conversion of fructose 6-phosphate and ATP to fructose 1,6-bisphosphate and ADP while **PFK-2** catalyzes the synthesis of fructose 2, 6-bisphosphate from fructose 6-phosphate.

Q.9. What is the functional role of PFK-2?

Ans. The role of PFK-2 is in the **metabolic reprogramming of cancer cells.**

Q.10. What is the functional role of fructose 2, 6-bisphosphate?

Ans. Frctose-2, 6-bisphosphate is a stimulator of PFK-1 by its ability to increase the affinity of PFK-1 for fructose-6-phosphate.

Q.11. What is the outcome of glycolysis?

Ans. Glycolysis **produces 2 ATP, 2 NADH, and 2 pyruvate molecules: Glycolysis, or the aerobic catabolic breakdown of glucose,** produces energy in the form of ATP, NADH, and pyruvate, which itself enters the citric acid cycle to produce more energy.

Q.12. What is the range of fasting blood sugar?

Ans. 70–100 mg/dl. (4.2–6.1 mmol/L)

Q.13. What is the normal blood sugar level?

Ans. 80–120 mg/dl.

Q.14. What is the range of postprandial (PP) blood sugar level?

Ans. Less than 140 mg/dl.

Q.15. What is the normal range of renal threshold value for glucose?

Ans. 160–180 mg/dl.

Q.16. What is the ATP yield per molecule of glucose via glycolysis alone *under anaerobic conditions*?

Ans. Two ATP (4 – 2 = 2) per molecule of glucose metabolized.

Q.17. What is the ATP yield per molecule (one molecule) of glucose if oxidised completely via glycolysis and TCA cycle under aerobic conditions?

Ans. 32 ATP per molecule of glucose oxidised, i.e. via glycolysis 12 ATP and via TCA cycle 20 ATP.

Q.18. How many molecules of ATP are formed in TCA cycle from (i) one molecule of acetyl CoA, and (ii) one molecule of pyruvate?

Ans. (i) 10 ATP
(ii) 12.5 ATP

Q.19. What is the generation of ATP through pyruvate dehydrogenase reaction alone by 2 moles of pyruvic acid?

Ans. 5

Q.20. What is the generation of ATP in TCA cycle alone?

Ans. 10 + 10 = 20 ATP.

Q.21. How many molecules of ATP are generated upon oxidation of one molecule of NADH via electron transport chain (ETC)?

Ans. 2.5

Q.22. How many molecules of ATP are generated upon oxidation of one molecule of FADH via electron transport chain (ETC)?

Ans. 1.5

Q.23. What is the full form of TCA cycle?

Ans. Tricarboxylic acid cycle.

Q.24. What are other names of TCA cycle?

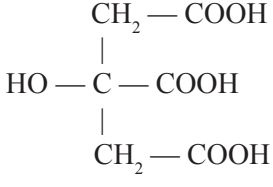
Ans. Citric acid cycle and Krebs cycle.

Q.25. Who invented TCA cycle?

Ans. Sir Hans Adolf Krebs (Nobel Prize in 1953)

Q.26. What is the chemical structure of tricarboxylic acid?

Ans.



Q.27. What do you know about the amphibolic nature (biological significance) of TCA cycle?

Ans.
- As TCA cycle functions in both **catabolism** and **anabolism** hence called an '**amphibolic'** pathway.
- It plays a pivotal role in cellular repiration.

- It has multiple interconnections with other pathways.
- It provides ground for interconversion of numerous metabolites.
- Functions in both the ways: produces and consumes intermediates relevant to a most of other pathways.
- **Examples:** Most amino acids enter the cycle via intermediates and get oxidized (catabolism).
- Side-by-side, the intermediates of TCA cycle are useful for biosynthetic reactions (anabolism), e.g. succinyl CoA in the synthesis of heme.
- Oxaloacetate and α-ketoglutarate for the formation of aspartate and glutamate respectively.

Q.28. Where does substrate level phosphorylation occur in TCA cycle?

Ans. In the **conversion of succinyl CoA to succinate**, the reaction is catalysed by the enzyme succinate thiokinase, GTP is formed which, in turn, gets converted to ATP.

Q.29. Name dicarboxylic acids.

Ans. Succinic acid, oxalic acid, etc.

Q.30. What is the chemical structure of succinic acid?

Ans. CH_2COOH
|
CH_2COOH

Q.31. Name two keto acids involved in carbohydrate metabolism?

Ans.
- Pyruvic acid
- α-ketoglutaric acid

Q.32. What is oxidative decarboxylation?

Ans. Oxidation accompanied by decarboxylation is called oxidative decarboxylation.

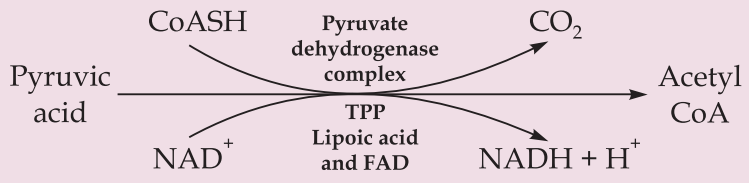
Q.33. Why TCA cycle is considered to be the common pathway for carbohydrate, lipid and protein metabolism?

Ans. It is considered to be the common pathway for the metabolism of carbohydrates, lipids and proteins because it brings about complete oxidation of acetyl CoA to carbon dioxide and water.
Acetyl CoA is produced from all the three main metabolism:

1. Carbohydrate metabolism	: via	glycolysis
2. Lipid metabolism	: via	β-oxidation
3. Protein metabolism	: via	transamination

Q.34. What is the oxidative decarboxylation product of pyruvic acid?

Ans. Acetyl CoA



Q.35. Name the enzyme of the above oxidative decarboxylation reaction.

Ans. **Pyruvate dehydrogenase multienzyme complex**. This is an enzyme complex which consists of the following three enzymes:

1. Pyruvate dehydrogenase
2. Dihydrolipoyl acetyltransferase
3. Dihydrolipoyl dehydrogenase

Q.36. Which enzyme catalyzes the conversion of acetyl CoA to citric acid?

Ans. Citrate synthase (condensing enzyme).

Q.37. Name the coenzymes and cofactors involved in the oxidative decarboxylation of pyruvic acid.

Ans.
- Thiamine pyrophosphate (TPP)
- Lipoic acid
- Flavin adenine dinucleotide (FAD)
- Nicotinamide adenine dinucleotide (NAD^+)
- Coenzyme A (CoA-SH)
- Mg^{++} ions

Q.38. Name the vitamin involved in the oxidative decarboxylation of pyruvic acid.

Ans. Lipoic acid

Q.39. What is the other name of lipoic acid?

Ans. Thioctic acid

Q.40. Which is the main site of carbohydrate metabolism?

Ans. Liver

Q.41. Where does TCA cycle occur in the cell?

Ans. Mitochondria.

Q.42. Why it is called citric acid cycle or TCA cycle or Krebs cycle?

Ans. Because citric acid is formed first of all in this pathway. TCA– because the citric acid consists of three carboxylic groups.

Q.43. What is the importance/metabolic significance of hexose monophosphate shunt (HMP shunt) pathway?

Ans.
- **Provides NADPH** which is used for the biosynthesis of fatty acids, steroids and other important substances of the body.
- **Provides pentose sugars** which are used in the biosynthesis of nucleic acids.

Q.44. Name the sites of HMP shunt pathway.

Ans. Liver, adipose tissue, lactating mammary glands, adrenal cortex, testis, etc.

Q.45. What do you understand by branching and debranching enzymes?

Ans. Branching enzymes produce a branching in the molecule by establishing (l, 6) glycosidic linkages.
Debranching enzymes produce cleavage of α (l, 6) linkages.

Q.46. What is glycogenolysis?

Ans. It is the breakdown of polysaccharide glycogen into glucose in liver and kidney tissues and glucose-6-PO_4 in the muscles.

Q.47. Why is glucose-6-PO_4 end product of glycogenolysis in muscles?

Ans. Because muscles lack the enzyme glucose-6-phosphatase.

$$\text{Glucose-6-PO}_4 \xrightarrow[\times]{\text{Glucose-6-phosphatase}} \text{Glucose}$$

Q.48. Name the sites of glycogenolysis.

Ans. Main sites are liver and muscles.

Q.49. What is the role of cyclic AMP in glycogenolysis?

Ans. Cyclic AMP in the presence of dephosphorylase kinase brings about the conversion of dephosp-horylase (inactive form) into phosphorylase (active form).

Q.50. From where cyclic AMP comes in 'glycogenolysis'?

Ans. From ATP.

$$\text{ATP} \xrightarrow[Mg^{++}]{\text{adenyl cyclase}} \text{Cyclic AMP}$$

Q.51. What is the first breakdown product of glycogen metabolism?

Ans. Glucose-1-phosphate

Q.52. What is gluconeogenesis? Name some gluconeogenic substances.

Ans. It is the formation of **glucose** or **glycogen** from non-carbohydrate sources like glycerol, lactic acid, propionic acid, pyruvic acid and amino acids.

Q.53. What is the difference between gluconeogenesis and neoglucogenesis?

Ans. No difference

Q.54. Name the main sites of gluconeogenesis.

Ans. Liver and kidneys

Q.55. Name some gluconeogenic amino acids.

Ans. Glycine, valine, methionine, cysteine, aspartic acid and glutamic acid.

Q.56. What is the significance of 'gluconeogenesis pathway'?

Ans.
- It is a useful pathway especially when an individual is consuming less carbohydrates.
- It helps in the maintenance of blood sugar level within its normal range.
- It brings about proper disposal of glycerol and lactic acid, which are formed during muscular activity/exercise.

Q.57. Are there other alternative pathways of carbohydrate metabolism than the glycolysis and TCA cycle?

Ans. Following are the other two alternative pathways of carbohydrate metabolism.
- Pentose phosphate pathway (HMP shunt or phosphogluconate oxidative pathway or Warburg-Dickens-Lipmann pathway).
- Glucuronic acid cycle

Q.58. What happens if carbohydrates are taken in excess?

Ans. Excess intake of carbohydrates is harmful because in the animal body excess carbohydrates are converted to fats which are eventually stored in various tissues of the body causing **obesity**.

Q.59. Are there found other monosaccharides than glucose in the blood?

Ans. **Ordinarily** other monosaccharides like galactose, fructose and mannose do not remain present in the blood because all these have a tendency to be converted to glucose by their metabolism respectively. They may be found to be present in the blood only if their metabolism gets defected.

Q.60. Are there present oligosaccharides or polysaccharides in the blood; if no, why?

Ans. No, because all oligosaccharides and polysaccharides after digestion and metabolism are converted into glucose.

Q.61. How blood sugar level is controlled within its normal limits?

Ans. It is kept controlled within its normal range **by the liver mainly** by **balance between glycogenesis and glycogenolysis-cum-gluconeogenesis**. Besides these, several other factors like hormones, kidneys, etc. do also play role.

Q.62. Name the regulatory enzymes of gluconeogenesis pathway.

Ans.
- Pyruvate carboxylase
- Phosphoenolpyruvate carboxykinase (key regulatory enzyme)
- Fructose 1,6-bisphosphatase
- Glucose-6-phosphatase

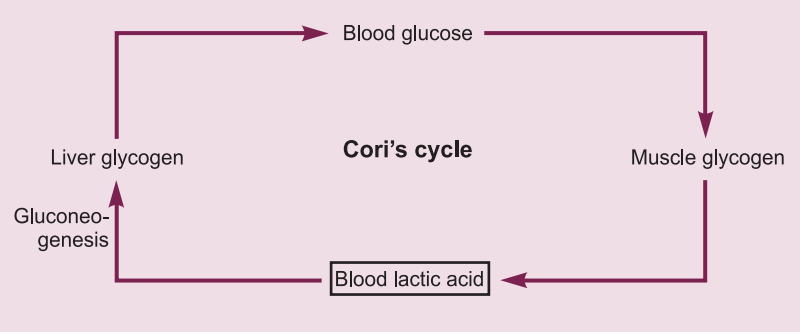
Q.63. Name the enzymes responsible for the reversal of gluconeogenesis.

Ans. The same enzymes that are used in glycolysis between PEP and fructose-1-6-bisphosphate, as they are virtually reversible, namely:
- Phosphoglycerate mutase
- Phosphoglycerokinase
- Triosephosphate isomerase
- Aldolase.

Q.64. What is Cori's cycle?

Ans. The Cori's cycle, named after its discoverer, **Carl Ferdinand Cori** and **Gerty Cori** is a metabolic pathway in which lactate produced by anaerobic glycolysis in muscles is transported to the liver and converted to glucose, which then returns to

the muscles and is cyclically metabolized back to lactate as shown below in diagram.



Q.65. How pyruvate is converted to glucose?

Ans. Pyruvate gets converted into glucose by the pathway of gluconeogenesis as shown below:

Pyruvate ⟶ Oxaloacetate
Oxaloacetate ⟶ Phosphoenolpyruvate
Phosphoenolpyruvate ⟶ Glucose

Q.66. How will you estimate true glucose (tell the best method)?

Ans.
- GOD/POD method (glucose oxidase peroxidase method).
- Nelson-Somogyi method (it is as accurate as enzymic method but time consuming).
- *o*-toluidine method

Q.67. Name the conditions/diseases in which blood sugar level gets increased (elevated).

Ans.
- Diabetes mellitus
- Hyperpituitarism
- Hyperthyroidism
- Hyperadrenalism
- Thyrotoxicosis

Q.68. Name the conditions/diseases in which blood sugar level is found to be low.

Ans.
- Hypothyroidism (myxoedema, cretinism)
- Hypopituitarism (Simmonds' disease)
- Hypoadrenalism (Addison's disease)
- Overdosage of insulin in the treatment of diabetes
- Hyperinsulinism, etc.

Q.69. What is TM_G?

Ans. The maximum rate at which glucose can be reabsorbed from the renal tubules is known as TM_G.

Q.70. Name the hormones responsible for raising the blood sugar level, i.e. hyperglycemic hormones.

Ans.
- Glucagon
- Epinephrine
- Thyroid hormones
- Adrenal cortex hormones
- Growth hormone, etc.

Q.71. What is diabetes mellitus?

Ans. It is a state of persistent/chronic hyperglycemia is known as diabetes mellitus which takes place due to the deficiency of insulin hormone.

Q.72. What is the literal meaning of dia, betes and mellitus?

Ans. Diabetes comes from Greek word which means:
Dia = through, betes = passing, mellitus = honey (sweet)
मधुमेह (मधु + मेह या मधु + बारिश अर्थात मूत्र के द्वारा शहद अर्थात शक्कर की बारिश); ऐसे रोगी जिन्हें मधुमेह होती है, वे यदि पक्के फर्श पर मूत्र त्याग करते हैं तो थोड़ी ही देर में उस स्थान पर चीटियाँ आ जाते हैं जो कि इस प्रकार से मूत्र के माध्यम से त्यागी गई शक्कर को ग्रहण करते हैं। यह शक्कर उन्हें ऊर्जा प्रदान करती है।

Q.73. What is hypoglycemia?

Ans. A state in which blood sugar level gets lowered than the normal range.

Q.74. What is hyperglycemia?

Ans. A state in which blood sugar level gets raised than the normal range.

Q.75. What is insulin?

Ans. It is a hypoglycaemic hormone.

Q.76. Who discovered the existence of insulin?

Ans. Banting, Best and Macleod in the year 1921.

Q.77. Who discovered the structure of insulin?

Ans. **Sanger,** for which he was awarded **Nobel Prize** in the year **1958**. (Sanger won the **Nobel Prize** twice, i.e. in **1958** for discovering the **structure of protein insulin** and in **1980** for the **base sequence of nucleic acids**).

Q.78. From where insulin is secreted in the human body?

Ans. By the β-cells of the islets of Langerhans of the pancreas.

Q.79. What do you know about the structure of insulin hormone?

Ans. It is a polypeptide and consists of **51** amino acids. It consists of two chains i.e. 'A' and 'B'. 'A' chain contains 21 whereas 'B' 30 amino acids. Both these chains remain linked to each other by two interchain disulfide bridges that connect A7 to B7 and A20 to B19. A third interchain disulfide bridge connects residues 6 and 11 of the A chain.

Q.80. Why insulin cannot be given orally to the diabetic patients?

Ans. Insulin (proteinous in nature) is a polypeptide which may be easily digested by the proteolytic enzymes of the digestive system into amino acids before it reaches the blood, hence it is not given orally (by mouth).

Q.81. What is diabetes insipidus?

Ans. Lack of antidiuretic hormone (**ADH**) gives rise to a condition known as diabetes insipidus. Level of sugar in blood of such individuals is found within normal range **but such individuals may excrete urine up to 30 L per day**.

Q.82. What is the abnormality in the urine samples of severe diabetic patient versus starving individual?

Ans. In severe diabetic patient, urine will exhibit the presence of sugar and ketone bodies whereas in case of starving individuals only ketone bodies will be found and no sugar.

Q.83. Which different reducing sugars appear in urine and under what conditions?

Ans.

Glucose **(glucosuria)** :	Appears in urine in **renal glycosuria** and diabetes mellitus.
Lactose **(lactosuria)** :	During later stage of pregnancy and lactation.
Galactose **(galactosuria)** :	In galactosaemia due to the deficiency of enzyme galactose–1–PO_4 uridyl transferase. This condition is found in infants.
Fructose **(fructosuria)** :	Due to consumption of large amount of fruits containing fructose such as plums, cherry, grapes, etc.
Pentoses **(pentosuria)** :	Due to consumption of large amount of fruits containing pentoses. Also, in congenital abnormality characterized by inability to metabolize L-xylulose.

Q.84. How the collection of blood specimen is done for estimating blood glucose?

Ans. Blood sample is collected in potassium oxalate-sodium fluoride vial, popularly known as **'fluoride vial'**.

Q.85. What is the function of potassium oxalate and NaF in the vial?

Ans.
- Potassium oxalate: Acts as an anticoagulant.
- NaF: Prevents glycolysis by inhibiting the enzyme **enolase** of the **glycolytic pathway**.

Q.86. What is diabetic acidosis?

Ans. A **serious diabetes complication** where the body produces excess blood acids (ketones).
This condition occurs when there is not enough insulin in the body. It can be triggered by infection or other illness.

Q.87. What is the treatment of Favism?

Ans. Diet devoid of fava-beans

Q.88. What is the treatment of diabetes mellitus?

Ans.
- Oral administration of antidiabetic drugs.
- Subcutaneous injection of soluble insulin.
- Carbohydrates restricted diet and intake of fenugreek (methi) seeds powder (1–4 teaspoons per day depending upon the severity) is also helpful in controlling it.
- **Regular physical exercises** like morning walk, and **'yoga'** are also helpful **(lifestyle changes)**.

Q.89. Of which pathway glucose-6-phosphate dehydrogenase is an important enzyme?

Ans. Pentose phosphate pathway

Q.90. What harm does the genetic deficiency of glucose-6-phosphate dehydrogenase cause?

Ans. **Hemolysis** and **jaundice**, particularly after the administration of **vitamin K**, **sulpha drugs** and **quinine** and after eating **fava-beans**.

Q.91. Which Indian races are susceptible to the genetic deficiency of glucose-6-phosphate dehydrogenase?

Ans. Parsis of Western India, Punjabis and Sindhis (P, P, S).

Q.92. What is Favism?

Ans. It is caused after eating fava-beans (contains toxic substances)

in which there occurs hemolysis as in such patients there is found genetic **deficiency of enzyme glucose-6-phosphate dehydrogenase**.

Q.93. What does the symbol ~P~ denote?

Ans. High energy phosphate group or bond.

Q.94. Who introduced the ~P~ symbol?

Ans. Scientist **Lipmann** (Nobel prize in 1953)

Q.95. What is renal diabetes?

Ans. This is not the true diabetes. It is a very rare condition in which renal absorption of glucose gets so diminished that significant amount of glucose escapes into the urine. Such a condition is known as **renal diabetes.**

Q.96. How 'carbohydrate metabolism' is controlled in the human body?

Ans. It is controlled by a battery of hormones which are secreted from several endocrine glands, e.g. the pancreas, the anterior pituitary gland, and the adrenal cortex.

Q.97. What is insulin shock?

Ans. Administration of insulin to a normal animal leads to a profound hypoglycemia resulting into convulsions or unconsciousness, such a state is termed insulin shock.

Q.98. What do you mean by 'Antibodies' to insulin?

Ans. **The repeated injection of insulin** results in the production of **antibodies to insulin** in all the subjects, whether diabetic or not, after 2 or 3 months of treatment. At times, high concentrations of the antibodies may be found in such subjects, who are said to be ***'resistant to insulin'*** as insulin has got almost negligible effect in them.

Q.99. What is the effect of carbohydrate free diet in human beings?

Ans. When the diet is carbohydrate free, ketosis takes place which is due to the fact that in the absence of carbohydrates, more fats (reserved in various tissues) burn (i.e. get oxidized) giving rise to more ketone bodies which are easily detectable in the urine (**ketonuria**).

Q.100. What is the normal level of ketone bodies in the blood?

Ans. Around 1 mg/dL

Q.101. What is the level of ketone bodies in the blood of uncontrolled diabetics?

Ans. It may reach 300–400 mg/dl or even higher.

Q.102. What is the normal level of ketone bodies (as acetone) in the urine per day?

Ans. Up to 50 mg.

Q.103. What is the level of ketone bodies (as acetone) in the urine of uncontrolled diabetics per day?

Ans. 10–50 g/L

Q.104. What is diabetes insipidus?

Ans. The posterior pituitary secretes an antidiuretic hormone **(ADH)** named pitressin, which is responsible to regulate the absorption of water by the renal tubules. **Deficiency of this hormone** leads to the condition known as **"diabetes insipidus"** in which enormous volume of urine is excreted **(up to 30 L per 24 hrs.)** having a **specific gravity as low 1.001 to 1.003.**

Q.105. What is juvenile onset type diabetes?

Ans. This type is caused by **genes** and **environmenal factors**, such as viruses which destroy β-cells of the pancreas, as a result of which almost no secretion of insulin hormone takes place. It generally takes place in adolescence. Urgent treatment in this disease with insulin is wanted, otherwise **ketoacidosis and diabetic coma** take place.

Q.106. What is adult or maturity-onset type diabetes mellitus?

Ans. It usually develops in middle-aged or elderly subjects over the age of 40 years who are often obese. This is a mild or moderate type of diabetes which can be **controlled by diet and oral hypoglycemic drugs**. In such cases, plasma insulin levels are low than the normal. Those having high **sugar level**, are treated with insulin.

Q.107. If you get blue colour in the test tube while performing Benedict's qualitative test in an urine sample, then, how much concentration of sugar does it denote?

Ans. Almost nil, i.e. negative test

Q.108. If you get greenish colour in the above test, then, how much concentration of sugar does it denote?

Ans. 0.5%

Q.109. If you get yellowish colour in the above test, then, how much concentration of sugar does it denote?

Ans. 1%

Q.110. If you get orangish colour in the above test, then, how much concentration of sugar does it denote?

Ans. 1.5%

Q.111. If you get reddish colour in the above test, then, how much concentration of sugar does it denote?

Ans. 2% and above

Q.112. By using which drug experimental diabetes mellitus may be induced in animals?

Ans. Alloxan or streptozotocin or phlorhizin.

Q.113. How much glucose may be excreted in the urine of a normal person in 24 hours?

Ans. Usually < 100 mg

Q.114. How much glucose may be excreted in the urine of a severe diabetic person in 24 hours?

Ans. 5–6% or even more

Q.115. Which enzyme is supposed to play role in the reabsorption of sugar in the renal tubules?

Ans. Phosphorylase

Q.116. Name the conditions of endocrine hyperactivity in which one can find glucose in ones urine.

Ans.
- Hyperthyroidism
- Hyperpituitarism
- Hyperadrenalism

Q.117. What is the best mean (technique) of showing the presence of the minimum quantity of galactose in the urine of an individual?

Ans. Thin layer chromatography (TLC)

Q.118. Does anaesthesia cause raise in blood sugar level?

Ans. Yes

Q.119. Name the eatables which are supposed to be rich in fructose.

Ans. Fruits, honey, syrup and jams

Q.120. Which enzyme is found to be lacking/absent in essential fructosuria and what is its repurcussion?

Ans. **Hepatic Fructokinase**. It is a benign condition characterized by the incomplete metabolism of fructose in the liver leading to its excretion in urine.

Q.121. What is the function of enzyme fructokinase?

Ans. It catalyses the conversion of fructose to fructose-1-phosphate.

Q.122. What are the renal threshold values for galactose, fructose and pentoses?

Ans. **They have no renal threshold**, therefore, they are excreted in the urine as soon as their level is high in the blood.

Q.123. How many people in India are sufferers of diabetes mellitus today?

Ans. **Nearly 8 crores** (**second largest in the world**).

Q.124. What are the main outside contributing factors of diabetes mellitus?

Ans.
- Changed lifestyle
- Eating habits
- Added stress
- Reduced physical exercises

Q.125. What are the major types of diabetes?

Ans.
- Type I diabetes: Due to β-cell destruction, usually leading to **absolute insulin deficiency**.
- Type II diabetes: Due to a progressive **insulin secretory defect**
- Gestational diabetes mellitus (GDM): **Diabetes which is caused during 2nd or 3rd trimester of pregnancy.** It is a temporary phase till the delivery of the baby.
- Specific types of diabetes: It is due to other causes, e.g., monogenic diabetes syndromes (such as neonatal diabetes and maturity—onset diabetes of the young [MODY]), diseases of the exocrine pancreas (such as cystic fibrosis) and **drug**-or **chemical-induced diabetes** (such as upon treatment of HIV/AIDS or after organ transplantation).

Q.126. How diabetes is diagnosed and managed?

Ans. It is diagnosed and managed by checking glucose level in blood as under:
- Fasting blood glucose
- Random glucose test
- HbA1c (glycosylated hemoglobin) test

Q.127. What are the main common causes of diabetes mellitus?

Ans.
- Obesity
- Inactive lifestyle: Both are responsible for about 90–95% of diabetics.
- Next cause is **insufficient production of insulin**.

Q.128. How is diabetes controlled?

Ans. This is achieved by the following three major ways:

1. Following a strict diet protocol
2. Adhering to routine and uniform physical exercises
3. Taking medicines and/or injections regularly

Q.129. What is the importance of HbA1c test?

Ans.
- HbA1c test is an important indicator of long-term **glycemic control**
- It reflects the cumulative glycemic history of the preceding 2–3 months
- A reliable measure of chronic hyperglycemia
- It correlates well with the risk of long-term diabetes complications

Q.130. What are the complications (metabolic abnormalities) of uncontrolled diabetes mellitus?

Ans. **Major** complications include:

(a) **Cardiovascular (heart) complications** including atherosclerosis
(b) **Retinopathy (irreversible eye diseases)**
(c) Nephropathy (**severe damages to kidneys**)
(d) **Neuropathy (damage to the nervous system)** and many other severe complications

More the hyperglycemia, more the severity of complications.

Q.131. What is diabetic nephropathy?

Ans. **Diabetic nephropathy** is a common complication of type 1 and type 2 diabetes. Overtime, poorly controlled diabetes can cause damage to the blood vessel clusters in kidneys that filter waste from blood. This can lead to kidney damage and high blood pressure.

Q.132. What is microalbuminuria and its significance?

Ans. **Microalbuminuria** (micro—very little, albuminuria—albumin in urine). It indicates the **presence of an abnormal urinary excretion of albumin, signifying endothelial dysfunction and** an increased risk for cardiovascular morbidity and mortality.

Q.133. What is Connecting peptide and its significance?

Ans. Connecting peptide is an **indicator** of how much insulin is being produced by an individual. A low level (or no

C-peptide) indicates that one's pancreas is producing **little** or no insulin. A low level may be normal if one has not eaten recently.

Q.134. What is the most common endocrine disease of the day?

Ans. Diabetes mellitus

Q.135. On which chromosome, the gene for the synthesis of insulin is found to be located?

Ans. Insulin gene is located on the short arm of chromosome No. 11 in humans

Q.136. How many amino acids are found in human preproinsulin and what is its molecular weight?

Ans. 110 amino acids, M.W. is 12,000 daltons

Q.137. How many amino acids are found in human proinsulin and what is its molecular weight?

Ans. 86 amino acids, molecular weight is 9,390 daltons.

Q.138. How many amino acids are found in human insulin and what is its molecular weight?

Ans. 51 (21 in A chain and 30 B chain) amino acids, molecular weight is 5808 Da (daltons)

Q.139. How much sulphur does insulin contain?

Ans. 3.4%

Q.140. What is the normal fasting insulin concentration in plasma?

Ans. < 25 mIU/L (<174 pmol/L)

Q.141. What is the half-life of insulin receptor?

Ans. 6–12 hours

Q.142. What is the approximate number of receptors per cell in the mammals?

Ans. The surface of a typical cell bears 10,000–20,000 receptors for a particular hormone

Q.143. How many amino acids are in C-peptide?

Ans. C-peptide is a peptide composed of **31 amino acids.** It is released from the pancreatic β-cells during cleavage of insulin from proinsulin chain.

Q.144. Name the enzymes catalyzing irreversible reactions of glycolysis.

Ans.
- Hexokinase (glucokinase)
- Phosphofructokinase
- Pyruvate kinase

Q.145. What are the major differences between the enzymes hexokinase and glucokinase?

Ans. Differences between hexokinase and glucokinase

Sl. No.		**Hexokinase**	**Glucokinase**
1.	Occurrence	Ubiquitous, (found in all cells)	Found mainly in liver
2.	Affinity to substrate	High	Low
3.	Km value	Low km (about 0.01 mM) for substrates	High km (10 mM)for glucose
4.	V_{max}	Low	High
5.	Feedback inhibition	Feedback inhibited by glucose-6-phosphate	No direct feedback inhibition
6.	Induction by insulin	Not induced by insulin	Induced by insulin
7.	Specificity	Acts on all hexoses, i.e. glucose, fructose and mannose (catalyses the phosphorylation of glucose only).	Acts on glucose only (catalyses the phosphorylation of glucose only)
8.	Capacity	Low	High

9.	Function, i.e. phosphorylation	Acts even when blood sugar level is low, glucose is phosphorylated, vis-a-vis utilised by the cells of the body	Acts only when blood sugar level is high (> 100 mg/dl), then, glucose is taken up for phosphorylation by the cells of the liver for the synthesis of glycogen **(glycogenesis)**

Q.146. What is Rapport-Leubering cycle and its importance?

Ans. It is a supplementary pathway to glycolysis which operates in RBCs of men and other mammals. This cycle is mainly concerned **with the synthesis of 2,3-bisphosphoglycerate in the RBCs** which facilitates the release of oxygen from oxyhaemoglobin in the tissues.

Q.147. What do you know about glycogenin?

Ans. **Glycogenin** in an **enzyme** involved **in converting glucose to glycogen**. It acts as a primer, by polymerizing the first few glucose molecules, after which other enzymes take over. It is classified as **a glucosyltransferase**.

Q.148. What is the importance of uronic acid pathway?

Ans. Uronic acid (or glucuronic acid) pathway is an oxidative pathway of glucose which is concerned with the synthesis of so many important substances in the body, like:

- **Glucuronic** acid
- **Pentoses**
- **Ascorbic acid,** i.e. vitamin C (not in humans)

Q.149. What is essential fructosuria?

Ans. The deficiency of enzyme fructokinase causes **'essential fructosuria'** which is an asymptomatic condition associated with the excretion of fructose in urine.

Q.150. What is hereditary fructose intolerance?

Ans. Absence/deficiency of enzyme aldolase-B causes hereditary fructose intolerance that might lead to severe hypoglycemia, vomiting, jaundice, hepatic failure, etc.

Q.151. In how many hours half of the blood glucose is lost via glycolysis on standing?

Ans. 2–3 hours

Q.152. Why blood glucose is lost on standing? Which chemical reaction takes place upon standing?

Ans. Due to glycolysis, as a result of which glucose gets converted to lactic acid upon standing. The reaction takes place as follows:

$$\underset{\text{glucose}}{C_6H_{12}O_6} \longrightarrow 2\ \underset{\text{lactic acid}}{C_3H_6O_3}$$

Q.153. For how many days, the mixture of sodium fluoride and potassium oxalate can prevent glycolysis from taking place, i.e. loss of glucose in the blood sample?

Ans. 2–3 days

Q.154. Name the glucose tolerance factor which increases sugar tolerance

Ans. Chromium, (Cr^{3+})

Q.155. What do you understand by the word insulinopenia?

Ans. Inadequate secretion of insulin.

Q.156. Which type of hormone is insulin?

Ans. Peptide

Q.157. What is the normal secretion of insulin per day?

Ans. Nearly 2 mg (30–50 units).

Q.158. Nearly how much insulin does pancreas of a normal adult person contain.

Ans. 200 units

Q.159. What is lactose intolerance?

Ans. The condition when one is unable to tolerate milk sugar is called **lactose intolerance** or **hypolactasia**. This is most commonly caused due to the deficiency of enzyme **lactase** which breaks **lactose** into its constituents, i.e. glucose and galactose.

Q.160. What are the plus and minus points of consuming milk sugar (lactose)?

Ans. **Lactose** is a food source of energy for microorganisms in the colon and they ferment it to lactic acid and also generate methane and hydrogen gases as well. Such gases are harmful as they cause distension of the stomach and flatulence.

Q.161. Is there any side effect of the production of lactic acid (lactate) in the colon?

Ans. **Lactic acid,** thus, produced is osmotically active and draws water into the intestine, **causing diarrhoea**. If severe enough, the gases and diarrhoea hinder the absorption of other nutrients such as fat and protein. The simplest treatment is to avoid consumption of milk products (containing lactose).

Q.162. Name various regulatory or rate limiting enzymes of various pathways/cycles of carbohydrate metabolism.

Ans.

SI. No	Pathway/Cycle	Name of the Enzyme
1.	Glycolysis	Hexokinase (or glucokinase in the liver), PFK-1, and pyruvate kinase
2.	Glycogenesis	Glycogen synthetase
3.	Glycogenolysis	Phosphrorylase kinase and glycogen phosphorylase
4.	Gluconeogenesis	Pyruvate carboxylase, PEP carboxykinase, Fructose 1, 6-bisphosphatase, and Glucose 6-phosphatase
5.	HMP shunt	Glucose-6-phosphate dehydrogenase (G6PD) and 6-phosphogluconate dehydrogenase
6.	TCA cycle	Citrate synthase, α-ketoglutarate dehydrogenase and Isocitrate dehydrogenase

Q.163. Name the enzymes which are found deficient/missing in hereditary fructose intolerance (HFI), essential fructosuria and galactosaemia.

Ans.
- Aldolase-B
- Fructokinase
- Galactose-1-phosphate uridyl transferase, respectively

Q.164. Name some important glycogen storage diseases and their missing enzymes.

Ans.

Type	Glycogen storage disease (s)	Deficient enzyme (missing)
I	von Gierke disease	Glucose-6-phosphatase
II	Pompe's disease	Acid maltase (Lysosomal)
III	Cori's disease	Debranching enzyme, i.e. Amylo-1,6-glucosidase
IV	Andersen's disease	Branching enzyme, i.e. (Amylo-[1→ 4] → [1→ 6] transglucosidase)
V	McArdle's disease	Muscle phosphorylase
VI	Her's disease	Liver phosphorylase
VII	Tarui's disease	Phosphofructokinase

Q.165. What is the side effect of essential fructosuria?

Ans. Disease depends upon the intake of fructose and sucrose, it is asymptomatic and harmless and may go undiagnosed.

Q. 166. How many types of glycogen storage diseases are there?

Ans. At least 13 types