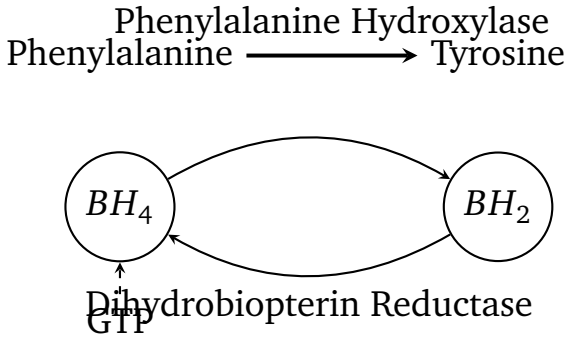
NEET PG Biochemistry Sample Paper-2

Duration: 15 Minutes **Maximum Marks: 64**

Instructions

- This paper contains **16** Multiple Choice Questions.
- Each correct answer carries **+4** mark. Incorrect answer: **−1** marks. Only **one** correct option.
- Unattempted questions carry **0** marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

Q1. A 4-year-old child presents with mild intellectual disability, recurrent seizures, and a "mousy" body odor. Laboratory evaluation reveals elevated levels of serum phenylalanine and phenylpyruvate. The clinician suspects classic phenylketonuria (PKU). Which of the following cofactors is essential for the enzyme deficient in this condition, and what is its immediate precursor?



(A) Tetrahydrobiopterin (BH_4); Derived from Guanosine triphosphate (GTP)

(B) Thiamine pyrophosphate (TPP); Derived from Vitamin B_1

(C) Pyridoxal phosphate (PLP); Derived from Vitamin B_6

(D) Flavin adenine dinucleotide (FAD); Derived from Vitamin B_2

Q2. During a seminar on molecular genetics, a post-graduate student explains the replication mechanism in prokaryotes. She correctly notes that DNA Polymerase III requires a free hydroxyl group to initiate elongation. Which

of the following statements accurately characterizes the enzymatic synthesis of the RNA primer required for this process?

(A) It is synthesized by RNA Polymerase II moving in a $3' \rightarrow 5'$ direction.

(B) It is synthesized by DnaG primase, a specialized RNA polymerase that does not require a free $3'$-OH to initiate synthesis.

(C) It is generated by the exonuclease action of DNA Polymerase I during the proofreading phase.

(D) It is synthesized by Topoisomerase I to release torsional strain during replication initiation.

Q3. An emergency room physician evaluates an unresponsive 45-year-old chronic alcoholic brought in by his friends. He is severely hypoglycemic. Laboratory evaluation reveals a high NADH/NAD+ ratio in hepatocytes, resulting from rapid ethanol clearance by alcohol dehydrogenase. The biochemical basis for hypoglycemia in this patient is directly related to the inhibition of which of the following processes?

(A) Glycogenolysis due to the non-competitive inhibition of glycogen phosphorylase by acetaldehyde.

(B) Fatty acid oxidation caused by the exhaustion of the available intracellular pool of Coenzyme A.

(C) Gluconeogenesis due to the shift in equilibrium turning pyruvate into lactate and oxaloacetate into malate.

(D) The Pentose Phosphate Pathway due to the feedback inhibition of glucose-6-phosphate dehydrogenase by NADPH.

Q4. A 28-year-old female presents with severe dermatitis, diarrhea, and cognitive decline (dementia). A detailed history reveals a strict dietary regimen composed exclusively of corn-based products. The patient is diagnosed with Pellagra. This condition results from a deficiency in Nicotinic acid, which can also be endogenously synthesized from which of the following essential amino acids?

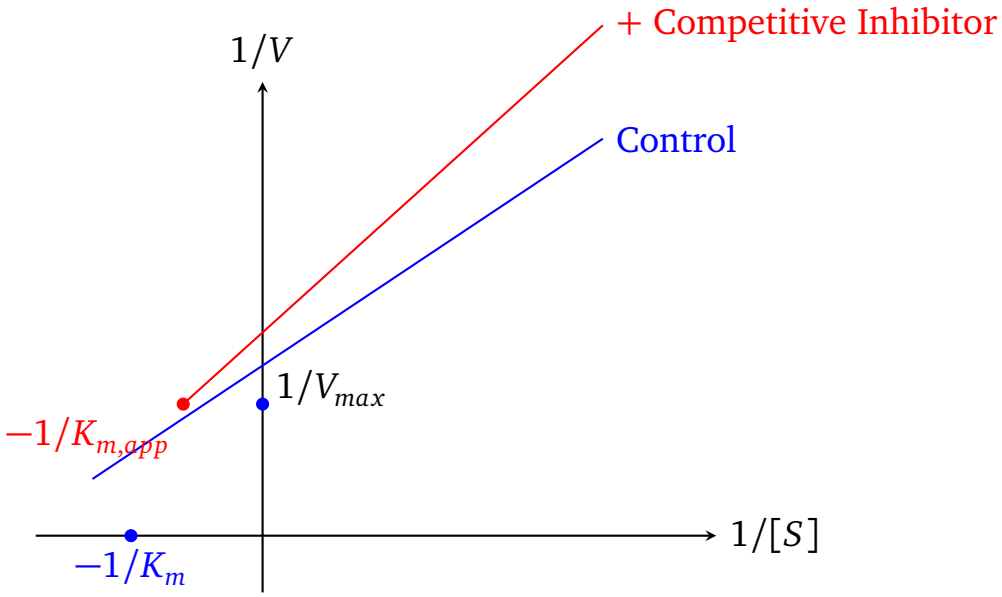
(A) Tyrosine

(B) Phenylalanine

(C) Methionine

(D) Tryptophan

Q5. An investigator studying enzyme kinetics sets up an in vitro assay to evaluate a novel competitive inhibitor for an essential bacterial transpeptidase. When plotting the data using a Lineweaver-Burk double-reciprocal format, which of the following shifts is expected when comparing the inhibited reaction to the uninhibited control?



(A) The plot retains the same x-intercept but exhibits a significantly higher y-intercept.

(B) The plot retains the same y-intercept ($1/V_{max}$) but shifts its x-intercept ($-1/K_m$) closer to the origin.

(C) Both the x-intercept and the y-intercept shift proportionately away from the origin.

(D) The plot becomes a completely horizontal line parallel to the 1/[S] axis due to total enzyme inactivation.

Q6. A newborn is evaluated for poor feeding, severe hypotonia, and macroglossia. An echocardiogram confirms massive cardiomegaly. Muscle biopsy shows a striking accumulation of glycogen within lysosomal structures, while cyto-

plasmic glycogen levels remain structurally normal. Which of the following enzymes is completely deficient in this patient?

(A) α-1,4-glucosidase (Acid maltase)

(B) Glucose-6-phosphatase

(C) Debranching enzyme (α-1,6-glucosidase)

(D) Glycogen phosphorylase

Q7. A laboratory scientist performs a southern blot analysis to confirm a genomic rearrangement. Before applying the labeled single-stranded probe to the membrane, the double-stranded genomic DNA must be fully denatured. Which of the following structural parameters directly increases the melting temperature (T_m) of a double-stranded DNA molecule?

(A) An increased concentration of Adenine-Thymine (A-T) base pairs.

(B) A decreased ionic strength or lower salt concentration in the surrounding buffer.

(C) An increased percentage of Guanine-Cytosine (G-C) base pairs due to triple hydrogen bonding.

(D) The absolute length of the DNA fragment, independent of nucleotide sequences.

Q8. A 6-month-old infant is brought to the clinic due to worsening developmental delay, unprovoked spasticity, and a tendency to compulsively bite his own lips and fingers. Laboratory studies demonstrate significant hyperuricemia. The defective enzyme in this patient is key to purine salvage. What is the normal metabolic fate of the immediate substrate that accumulates inside cells due to this metabolic block?

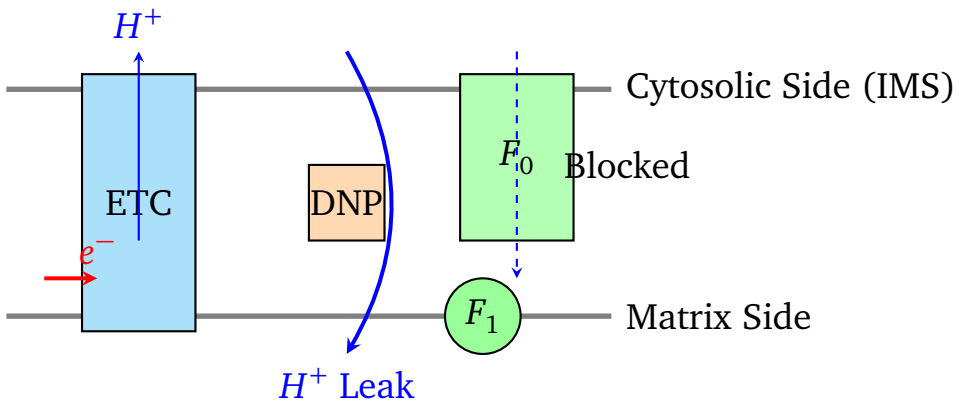
(A) PRPP (Phosphoribosyl pyrophosphate), which accelerates the de novo purine synthesis pathway.

(B) Inosine monophosphate (IMP), which is directly converted into xanthine without forming hypoxanthine.

(C) Adenosine triphosphate (ATP), which acts as an allosteric activator of ribonucleotide reductase.

(D) Xanthine, which precipitates within the lysosomal compartment of neurons.

Q9. During a research experiment, cells are incubated with an uncoupling agent called 2,4-dinitrophenol (DNP). Which of the following combinations correctly details the immediate effects of DNP on the proton-motive force, inner mitochondrial membrane oxygen consumption, and ATP synthesis rates?



(A) Proton gradient increases; Oxygen consumption drops to zero; ATP synthesis speeds up

(B) Proton gradient collapses; Oxygen consumption increases; ATP synthesis drops precipitously

(C) Proton gradient remains unchanged; Oxygen consumption drops; ATP synthesis stalls

(D) Proton gradient collapses; Oxygen consumption drops; ATP synthesis remains unaffected

Q10. A 55-year-old male is evaluated for sudden, intense pain, erythema, and swelling in his right first metatarsophalangeal joint. Polarized light microscopy of synovial fluid reveals negatively birefringent, needle-shaped crystals. The clinician initiates treatment with Allopurinol. What is the precise mechanism of action of Allopurinol in lowering serum uric acid?

(A) It acts as a suicide, mechanism-based competitive inhibitor of Xanthine Oxidase after conversion to oxypurinol.

(B) It blocks the renal reabsorption of uric acid at the level of the proximal convoluted tubule via URAT1.

(C) It directly cleaves circulating uric acid into the highly soluble metabolite allantoin.

(D) It downregulates the transcription of the PRPP synthetase gene through nuclear receptors.

Q11. A multi-disciplinary research group investigates the structural modifications of eukaryotic messenger RNA before nucleocytoplasmic export. They focus on the $5'$-7-methylguanosine cap. Which of the following choices accurately reflects the exact linkage type that joins the cap structure to the terminal $5'$ nucleotide of the nascent pre-mRNA molecule?

(A) $3' \rightarrow 5'$ phosphodiester linkage

(B) $5' \rightarrow 5'$ triphosphate bridge

(C) $2' \rightarrow 5'$ phosphodiester bond

(D) $3' \rightarrow 3'$ pyrophosphate linkage

Q12. A 3-year-old child presents with hepatomegaly, severe fasting hypoglycemia, and marked hyperlipidemia. Physical exam reveals subcutaneous eruptive xanthomas on the extensor surfaces. Blood draw shows milky-white lipemic plasma. A complete lack of glucose-6-phosphatase catalytic activity is confirmed. Which of the following pathways accounts for the intense hyperlipidemia seen in this patient?

(A) Diversion of accumulated Glucose-6-phosphate into glycolysis, generating excess Acetyl-CoA and NADPH for de novo lipogenesis.

(B) Accelerated activation of hormone-sensitive lipase due to high circulating levels of insulin.

(C) Impaired clearance of chylomicrons caused by an inherited co-existing mutation in apolipoprotein C-II.

(D) Reduced mitochondrial transport of free fatty acids mediated by an overproduction of malonyl-CoA.

Q13. An investigator analyzes eukaryotic transcription initiation. She isolates a mutant cell line where RNA Polymerase II cannot successfully transition from transcription initiation to transcription elongation, resulting in abortive transcripts. This defect is most likely tied to a loss of kinase activity in which of the following basal transcription factors?

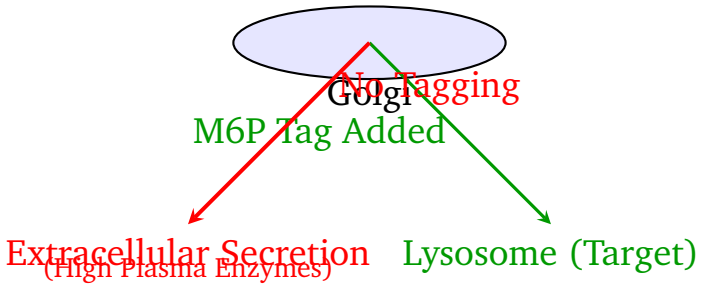
(A) TFIIA

(B) TFIIB

(C) TFIID

(D) TFIIH

Q14. A pediatric geneticist evaluates an 11-month-old boy with coarse facial features, severe corneal clouding, restricted joint mobility, and marked psychomotor delays. Plasma lysosomal enzyme assays demonstrate massive elevations of multiple acid hydrolases, while fibroblast cultures show dense intracellular inclusions and low internal enzyme levels. This condition is due to a structural sorting defect involving which of the following modifications?



(A) Failure to attach a Mannose-6-phosphate recognition tag in the Cis-Golgi network.

(B) Defective *O*-linked glycosylation within the rough endoplasmic reticulum lumen.

(C) Abnormally high rates of ubiquitination leading to premature proteasomal degradation.

(D) Absence of a hydrophobic *N*-terminal signal peptide sequencing tag.

Q15. A 30-year-old G1P0 female at 12 weeks' gestation undergoes genetic screening. The results indicate that she carries a balanced reciprocal translocation. During clinical counseling, the medical geneticist explains that while the patient is phenotypically normal, her germ cells could undergo unequal segregation during meiosis. Which of the following statements best describes the primary structural consequence of a balanced reciprocal translocation?

(A) An exchange of chromosomal material between non-homologous chromosomes with no net loss or gain of genetic material.

(B) Fusion of the long arms of two acrocentric chromosomes with the loss of both short arms.

(C) A 180-degree rotation of a single chromosomal segment that contains the central centromere.

(D) The amplification of a localized trinucleotide repeat sequence during lagging-strand synthesis.

Q16. An infant presents with profound lethargy, persistent vomiting, and metabolic acidosis. Urinalysis demonstrates high concentrations of methylmalonic acid. Further diagnostic workup points to a severe metabolic block. A deficiency in which of the following vitamins would directly phenocopy this metabolic presentation despite having a wild-type methylmalonyl-CoA mutase enzyme?

(A) Vitamin B_6 (Pyridoxine)

(B) Vitamin B_{12} (Cobalamin)

(C) Vitamin B_9 (Folate)

(D) Vitamin B_7 (Biotin)

Detailed Solutions

Q1.

Solution

Concept: The conversion of phenylalanine to tyrosine is catalyzed by phenylalanine hydroxylase. This enzyme is a monooxygenase that requires molecular oxygen and the specific electron-donating coenzyme tetrahydrobiopterin (BH_4). A complete or partial deficiency of this enzyme results in classic phenylketonuria (PKU), marked by an accumulation of systemic phenylalanine and alternative neurotoxic metabolites like phenylpyruvate, which are excreted in the urine and impart a characteristic mousy or musty body odor.

Solution:

(a) Phenylalanine hydroxylase utilizes tetrahydrobiopterin (BH_4) to insert one atom of oxygen into the phenyl ring of phenylalanine to form the hydroxyl group of tyrosine, while the other oxygen atom is reduced to water.

(b) During this hydroxylation reaction, BH_4 is oxidized to dihydrobiopterin (BH_2). To maintain active hydroxylation, BH_2 must be continuously regenerated back to BH_4 via the action of the enzyme dihydrobiopterin reductase using NADPH.

(c) Tetrahydrobiopterin is not a vitamin-derived cofactor; instead, it is synthesized endogenously by the human body through a specialized de novo metabolic pathway.

(d) The initial, rate-limiting step in the endogenous synthesis of tetrahydrobiopterin involves the purine nucleotide guanosine triphosphate (GTP), which is converted via GTP cyclohydrolase I.

(e) Therefore, a deficiency in phenylalanine hydroxylase requires BH_4 for its catalytic mechanism, a cofactor that is directly derived from a GTP precursor.

Final Answer: Tetrahydrobiopterin (BH_4); Derived from Guanosine triphosphate (GTP)

Answer: (A)

Go Back to Question 1

Q2.

Solution

Concept: DNA replication requires a pre-existing primer because replicative DNA polymerases can only add deoxyribonucleotides to an existing 3′-OH group. In prokaryotes like Escherichia coli, initiation of both leading and lagging strand synthesis depends on the creation of short RNA oligonucleotides, which provides the necessary terminus for DNA Polymerase III to begin elongation.

Solution:

(a) The synthesis of the mandatory short RNA primer sequence is executed by a specific, dedicated enzyme known as DnaG primase.

(b) Unlike replicative DNA polymerases, DnaG primase belongs to the class of RNA polymerases and possesses the biochemical capacity to initiate polynucleotide synthesis de novo without requiring an existing primer template.

(c) DnaG primase recognizes specific trinucleotide sequences on single-stranded template DNA and synthesizes a primer of approximately 10 to 12 nucleotides in length in the $5' \rightarrow 3'$ direction.

(d) RNA Polymerase II is a eukaryotic enzyme responsible for transcribing protein-coding messenger RNAs and is completely absent in prokaryotes.

(e) DNA Polymerase I processes and removes these RNA primers later in replication via its unique $5' \rightarrow 3'$ exonuclease activity rather than generating them during proofreading.

Final Answer: It is synthesized by DnaG primase, a specialized RNA polymerase that does not require a free 3′-OH to initiate synthesis.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept: The hepatic metabolism of ethanol generates massive quantities of reducing equivalents in the form of cytosolic and mitochondrial NADH. This rapid shift in the cellular redox state significantly alters the normal metabolic pathways of the liver, leading to an inability to maintain blood glucose levels during fasting periods and resulting in profound hypoglycemia.
Solution:

(a) Ethanol is oxidized to acetaldehyde by cytosolic alcohol dehydrogenase, which reduces NAD^+ to NADH. Acetaldehyde is further oxidized to acetate in the mitochondria by aldehyde dehydrogenase, producing another molecule of NADH.

(b) The resulting high ratio of NADH to NAD^+ in hepatocytes drives reversible dehydrogenase reactions backward to clear the excess NADH pool.

(c) Pyruvate is preferentially reduced to lactate by lactate dehydrogenase, which simultaneously depletes the intracellular pool of pyruvate available for gluconeogenesis.

(d) Similarly, oxaloacetate is converted to malate by malate dehydrogenase, draining another essential structural intermediate required for glucose synthesis.

(e) Because the key substrates pyruvate and oxaloacetate are effectively sequestered, hepatic gluconeogenesis is profoundly inhibited, leading to severe fasting hypoglycemia in depleted chronic alcoholics.

Final Answer: Gluconeogenesis due to the shift in equilibrium turning pyruvate into lactate and oxaloacetate into malate.

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept: Pellagra is a classic clinical deficiency disease characterized by the triad of dermatitis, diarrhea, and dementia. It arises due to a systemic shortage of nicotinic acid (niacin or Vitamin B_3), which serves as an essential component of the coenzymes NAD^+ and $NADP^+$. Humans can satisfy their niacin requirements either through direct dietary intake or via endogenous biosynthetic conversion.

Solution:

(a) The human liver possesses a metabolic pathway capable of synthesizing nicotinic acid nucleotides endogenously from the essential amino acid tryptophan.

(b) This complex kynurenine pathway requires several enzymatic steps, including structural intermediates that depend on vitamin cofactors like pyridoxal phosphate (B_6) and riboflavin (B_2).

(c) Approximately 60 mg of dietary tryptophan is required to synthesize 1 mg of nicotinic acid equivalents, making it an inefficient but vital alternative source.

(d) Corn (maize) is exceptionally low in usable tryptophan and contains niacin in a bound, unabsorbable form known as niacytin.

(e) A strict corn-based diet induces a concurrent deficiency of both preformed dietary niacin and the tryptophan precursor required for its endogenous synthesis, directly causing Pellagra.

Final Answer: Tryptophan

Answer: (D)

Go Back to Question 4

Q5.

Solution

Concept: Enzyme inhibitors alter the kinetic parameters of an enzyme-catalyzed reaction. In competitive inhibition, the inhibitor structurally resembles the substrate and binds exclusively to the free enzyme at the active site, directly competing with the natural substrate for binding. This mechanism can be mathematically evaluated using the double-reciprocal Lineweaver-Burk plot.

Solution:

(a) Because a competitive inhibitor binds reversibly to the active site, its inhibitory effect can be completely overcome by increasing the substrate concentration to saturating levels.

(b) As a result, the maximum velocity (V_{max}) of the enzyme-catalyzed reaction remains entirely unchanged in the presence of a competitive inhibitor.

(c) On a Lineweaver-Burk plot, the y-intercept represents $1/V_{max}$; thus, the inhibited and uninhibited plots intersect precisely at the same point on the vertical axis.

(d) However, the presence of the inhibitor increases the apparent Michaelis constant ($K_{m,app}$), meaning a higher concentration of substrate is required to achieve half-maximal velocity.

(e) The x-intercept represents $-1/K_m$. An increase in K_m shifts the x-intercept closer to the origin, producing a steeper slope.

Final Answer: The plot retains the same y-intercept ($1/V_{max}$) but shifts its x-intercept ($-1/K_m$) closer to the origin.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: Glycogen storage diseases are inherited disorders caused by deficiencies in enzymes regulating glycogen synthesis or degradation. While most glycogen degradation occurs in the cytoplasm via glycogen phosphorylase and debranching enzyme, a small fraction of cellular glycogen is continuously trafficked into lysosomes for degradation by an acid hydrolase.

Solution:

(a) Pompe disease (Glycogen Storage Disease Type II) is caused by a complete deficiency of the lysosomal enzyme α-1,4-glucosidase, also known as acid maltase.

(b) This enzyme is responsible for breaking down the glycogen molecules that enter the lysosomal vacuole during normal cellular component turnover.

(c) When α-1,4-glucosidase is missing, glycogen accumulates within membrane-bound lysosomes, disrupting normal architecture and function in cardiac and skeletal muscle tissue.

(d) Because cytoplasmic glycogen degradation mechanisms remain completely intact, blood glucose levels and cytoplasmic glycogen structure appear entirely normal in these patients.

(e) The classic infant presentation includes profound muscle weakness (hypotonia), macroglossia, and massive cardiomegaly due to glycogen deposition in cardiomyocytes, leading to early cardiac failure.

Final Answer: α-1,4-glucosidase (Acid maltase)

Answer: (A)

Go Back to Question 6

Q7.

Solution

Concept: The melting temperature (T_m) of DNA is defined as the temperature at which 50% of the double-stranded DNA molecules dissociate into single strands. This denaturation process requires breaking the non-covalent hydrogen bonds between complementary base pairs and overcoming the hydrophobic stacking interactions between adjacent bases.

Solution:

(a) Adenine-Thymine (A-T) base pairs are held together by two hydrogen bonds, whereas Guanine-Cytosine (G-C) base pairs are joined by three hydrogen bonds.

(b) The additional hydrogen bond, combined with stronger base-stacking interactions between adjacent G-C pairs, gives G-C pairs higher thermal stability than A-T pairs.

(c) Consequently, a higher percentage of G-C base pairs within a DNA fragment increases the total energy required to disrupt the double helix, raising its T_m.

(d) Decreasing the ionic strength or salt concentration of the surrounding buffer lowers T_m because fewer cations are available to shield the negatively charged phosphate backbones from mutual electrostatic repulsion.

(e) While the total sequence length influences melting behavior in small oligonucleotides, the nucleotide base composition remains the dominant factor for genomic fragments.

Final Answer: An increased percentage of Guanine-Cytosine (G-C) base pairs due to triple hydrogen bonding.

Answer: (C)

Go Back to Question 7

Q8.

Solution

Concept: Lesch-Nyhan syndrome is an X-linked recessive disorder caused by a severe deficiency of hypoxanthine-guanine phosphoribosyltransferase (HGPRT). This enzyme is a critical component of the purine salvage pathway, which recycles free purine bases back into nucleotide monophosphates, saving cellular energy.

Solution:

(a) HGPRT catalyzes the conversion of hypoxanthine to inosine monophosphate (IMP) and guanine to guanosine monophosphate (GMP), using 5-phosphoribosyl-1-pyrophosphate (PRPP) as the ribose donor.

(b) In the absence of functional HGPRT, hypoxanthine and guanine cannot be salvaged. This causes a massive accumulation of intracellular PRPP because it is not consumed by the salvage pathway.

(c) Excess PRPP acts as a potent allosteric activator of amidophosphoribosyltransferase, the rate-limiting enzyme of the de novo purine synthesis pathway.

(d) This activation accelerates de novo purine synthesis far beyond cellular needs, leading to the overproduction and degradation of excess purines into uric acid.

(e) The resulting severe hyperuricemia causes gouty arthritis, kidney damage, and the hallmark neurological symptoms of the syndrome, including choreoathetosis and compulsive self-mutilation.

Final Answer: PRPP (Phosphoribosyl pyrophosphate), which accelerates the de novo purine synthesis pathway.

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Oxidative phosphorylation relies on an intact electrochemical proton gradient across the inner mitochondrial membrane. The electron transport chain (ETC) pumps protons from the matrix into the intermembrane space, generating a proton-motive force that drives ATP synthesis via F_0F_1-ATP synthase. Uncoupling agents uncouple electron transport from ATP synthesis.

Solution:

(a) 2,4-Dinitrophenol (DNP) is a lipophilic weak acid that dissolves in the inner mitochondrial membrane and acts as a proton ionophore.

(b) DNP binds protons in the intermembrane space where the concentration is high, diffuses across the membrane, and releases them into the alkaline matrix.

(c) This proton leak bypasses the ATP synthase channel, collapsing the electrochemical proton gradient and eliminating the driving force for ATP synthesis, causing ATP production to drop.

(d) To compensate for the loss of ATP, the electron transport chain operates at a maximal rate, consuming oxygen rapidly to re-establish the gradient, though it continuously dissipates as heat.

Final Answer: Proton gradient collapses; Oxygen consumption increases; ATP synthesis drops precipitously

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: Gout is an inflammatory arthritis caused by the crystallization of uric acid as monosodium urate inside joints, often presenting as severe pain in the first metatarsophalangeal joint. Uric acid is the final breakdown product of purine metabolism in humans, synthesized via successive oxidations of hypoxanthine and xanthine.

Solution:

(a) The enzyme xanthine oxidase catalyzes the conversion of hypoxanthine to xanthine, and subsequently xanthine to uric acid.

(b) Allopurinol is a structural isomer of hypoxanthine designed to inhibit this pathway. It acts as a substrate for xanthine oxidase, which metabolizes it into its active form, oxypurinol.

(c) Oxypurinol binds tightly to the molybdenum-iron catalytic site of xanthine oxidase, locking the enzyme in an inactive state.

(d) This mechanism is classified as suicide or mechanism-based competitive inhibition, as the enzyme's own catalytic action converts the drug into an irreversible inhibitor.

(e) Inhibiting xanthine oxidase lowers plasma concentrations of poorly soluble uric acid while increasing levels of more soluble precursors like hypoxanthine and xanthine, which are readily excreted by the kidneys.

Final Answer: It acts as a suicide, mechanism-based competitive inhibitor of Xanthine Oxidase after conversion to oxypurinol.

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: The processing of eukaryotic pre-messenger RNA involves critical chemical modifications before it can be safely exported from the nucleus to the cytoplasm for translation. A key protective feature added almost immediately during transcription initiation is the synthesis of a specialized structure at the extreme leading end of the primary transcript, widely known as the $5'$-7-methylguanosine cap.

Solution:

(a) Capping begins after the transcription of approximately twenty to thirty nucleotides, when an enzyme complex interacts directly with the carboxyl-terminal domain of RNA Polymerase II.

(b) A terminal phosphate group is removed from the $5'$ end of the primary RNA transcript by an RNA $5'$ triphosphatase.

(c) The enzyme guanylyltransferase transfers a guanosine monophosphate residue from a GTP molecule to the remaining $5'$ diphosphate end of the nascent transcript.

(d) This enzymatic linkage produces an unconventional $5' \rightarrow 5'$ triphosphate bridge, which is unique because standard polynucleotide elongation occurs via $3' \rightarrow 5'$ phosphodiester bonds.

(e) Subsequently, methyltransferase enzymes add a methyl group to the nitrogen at position 7 of the terminal guanine ring and to the initial nucleotides of the transcript.

(f) This terminal linkage shields the mature messenger RNA from degraded destruction by cellular $5'$ exonucleases, stabilizes the transcript, and serves as an anchor recognized by the translation initiation complex.

Final Answer: $5' \rightarrow 5'$ triphosphate bridge

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: Von Gierke disease (Glycogen Storage Disease Type I) is an autosomal recessive metabolic disorder caused by a complete deficiency of the hepatic enzyme glucose-6-phosphatase. This terminal enzyme is essential for releasing free glucose into the circulation from both glycogenolysis and gluconeogenesis pathways. Its absence creates a profound intrahepatic accumulation of glucose-6-phosphate, completely altering secondary lipid pathways.
Solution:

(a) Because glucose-6-phosphate cannot be dephosphorylated to free glucose during fasting states, the intracellular substrate pool builds up massively inside hepatocytes.

(b) This structural accumulation forces the excess glucose-6-phosphate down the glycolytic pathway, converting it rapidly into pyruvate and subsequently into acetyl-coenzyme A.

(c) Concurrently, a significant portion of glucose-6-phosphate enters the pentose phosphate pathway, creating a vast overproduction of nicotinamide adenine dinucleotide phosphate (NADPH).

(d) The combination of abundant mitochondrial acetyl-coenzyme A and high cytosolic NADPH provides the ideal substrates for accelerated de novo fatty acid synthesis and cholesterol formation.

(e) These newly synthesized lipids are packaged into very-low-density lipoproteins (VLDL) and secreted into the blood, resulting in severe secondary hyperlipidemia, xanthomas, and lipemic plasma.

(f) Fasting conditions exacerbate this lipid synthesis loop because the body continuously attempts to mobilize glycogen stores that remain trapped within the liver tissue.

Final Answer: Diversion of accumulated Glucose-6-phosphate into glycolysis, generating excess Acetyl-CoA and NADPH for de novo lipogenesis.

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: Transcription initiation in eukaryotic cells requires the systematic assembly of general transcription factors alongside RNA Polymerase II at the core promoter region to form the pre-initiation complex. The transition from an immobilized transcription initiation complex into an actively moving transcription elongation complex is a regulated biochemical switch governed by specific covalent modifications.

Solution:

(a) The multi-subunit basal transcription factor TFIIH plays a vital dual role during the final steps of eukaryotic pre-initiation complex setup.

(b) One subunit of TFIIH functions as an ATP-dependent DNA helicase that melts the promoter DNA to form the open transcription bubble.

(c) Another distinct module of TFIIH contains a cyclin-dependent protein kinase activity that specifically target the structural carboxyl-terminal domain (CTD) of RNA Polymerase II.

(d) The CTD consists of multiple tandem repeats of a heptapeptide sequence rich in serine, threonine, and tyrosine amino acid residues.

(e) Phosphorylation of these specific serine residues by the kinase subunit of TFIIH causes a conformational shift that releases the polymerase from the promoter.

(f) Without this crucial kinase modification, the polymerase remains locked at the initiation site, resulting in failed promoter clearance and the production of short abortive transcripts.

Final Answer: TFIIH

Answer: (D)

Go Back to Question 13

Q14.

Solution

Concept: I-cell disease (Mucolipidosis Type II) is a severe autosomal recessive lysosomal storage disorder caused by a failure to correctly target acid hydrolases to the lysosomal compartment. Lysosomal enzymes are glycoproteins synthesized in the rough endoplasmic reticulum and subsequently modified in the Golgi apparatus, where they receive a specific structural sorting signal.

Solution:

(a) Normal targeting of newly synthesized acid hydrolases depends on the addition of a mannose-6-phosphate (M6P) recognition marker within the cis-Golgi network.

(b) This modification is catalyzed by the enzyme UDP-N-acetylglucosamine-1-phosphotransferase, which attaches a phosphate group to specific mannose residues on the oligosaccharide core.

(c) In patients with I-cell disease, a genetic mutation causes a total deficiency of this phosphotransferase enzyme, leaving lysosomal proteins completely unphosphorylated.

(d) Lacking the vital mannose-6-phosphate tag, these enzymes cannot bind to M6P receptors in the trans-Golgi network for transport to lysosomes.

(e) Instead, the default constitutive secretory pathway diverts these unlabelled enzymes out of the cell, leading to massive elevations of acid hydrolases in the blood plasma.

(f) The lysosomes become depleted of functional enzymes, resulting in the intracellular accumulation of undegraded substrates that form the classic dense inclusion bodies.

Final Answer: Failure to attach a Mannose-6-phosphate recognition tag in the Cis-Golgi network.

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: Chromosomal structural abnormalities arise from mistakes that occur during DNA replication, repair, or meiotic recombination. Translocations involve the rearrangement of genetic segments between entirely non-homologous chromosomes. These alterations are categorized based on whether genetic material is conserved or lost during the breakage and reunion process.

Solution:

(a) A balanced reciprocal translocation occurs when breaks happen in two different non-homologous chromosomes, followed by a mutual swap of the detached segments.

(b) This process alters the physical positioning of the genes along the rearranged chromosomes but preserves the overall diploid count of the genome.

(c) Because there is no net loss or gain of crucial coding genetic material, carriers of a balanced translocation are typically healthy and phenotypically normal.

(d) However, significant clinical problems arise during gametogenesis when these modified chromosomes attempt to pair up and separate during meiosis I.

(e) The chromosomes form a unique quadrivalent structure during synapsis, which can segregate in an unbalanced fashion into the resulting egg or sperm cells.

(f) This segregation frequently gives rise to gametes with duplications or deletions of entire chromosomal arms, causing recurrent miscarriages or severe congenital anomalies in offspring.

Final Answer: An exchange of chromosomal material between non-homologous chromosomes with no net loss or gain of genetic material.

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept: Methylmalonic acidemia is a heterogeneous metabolic disorder characterized by an inability to convert methylmalonyl-coenzyme A into succinyl-coenzyme A. This reaction represents a vital converging step in the catabolism of odd-chain fatty acids, cholesterol side chains, and specific amino acids including valine, isoleucine, methionine, and threonine.

Solution:

(a) The conversion of methylmalonyl-CoA to succinyl-CoA is catalyzed by the mitochondrial matrix enzyme methylmalonyl-CoA mutase.

(b) This complex isomerization reaction strictly requires the active coenzyme form of cobalamin, specifically known as adenosylcobalamin (Vitamin B_{12}).

(c) If a patient suffers from a severe nutritional or functional absorption deficiency of Vitamin B_{12}, the mutase enzyme lacks its essential cofactor.

(d) This total loss of active coenzyme function mimics a structural mutation of the mutase enzyme itself, presenting with the identical biochemical phenotype.

(e) As a direct consequence, methylmalonyl-CoA accumulates upstream and is hydrolyzed into methylmalonic acid, causing severe systemic metabolic acidosis, vomiting, and lethargy.

(f) Conversely, deficiencies in pyridoxine (B_6), folate (B_9), or biotin (B_7) involve different pathways, such as transamination or carboxylation, and do not cause methylmalonic acid accumulation.

Final Answer: Vitamin B_{12} (Cobalamin)

Answer: (B)

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Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	C	4	D	5	B
6	A	7	C	8	A	9	B	10	A
11	B	12	A	13	D	14	A	15	A
16	B								