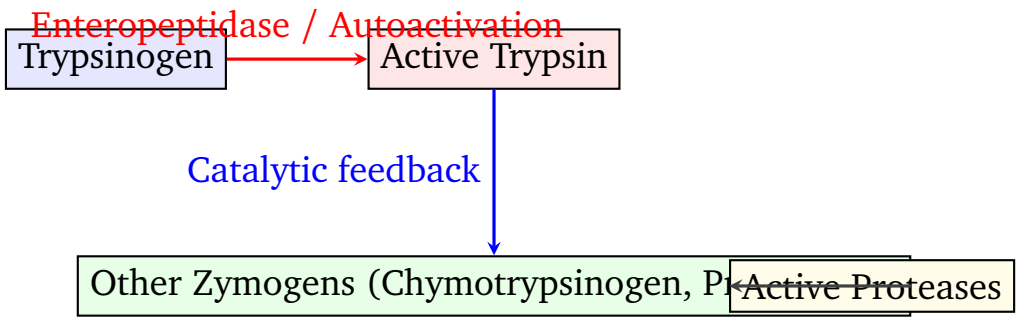
NEET PG Biochemistry Sample Paper-3

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 45-year-old chronic alcoholic presents with severe epigastric pain radiating to the back, accompanied by nausea and vomiting. Laboratory evaluation reveals significantly elevated serum amylase and lipase levels. A diagnosis of acute pancreatitis is made. The premature activation of zymogens within pancreatic acinar cells is a critical event in the pathogenesis of this condition. Consider the following cascade of enzyme activation:



Which of the following physiological protective mechanisms is most crucial in preventing this cascade from occurring prematurely within normal pancreatic acinar cells?

(A) Secretion of bicarbonate ions by pancreatic ductal cells to maintain an alkaline lumen

(B) Synthesis and co-packaging of Pancreatic Secretory Trypsin Inhibitor (PSTI / SPINK1) within zymogen granules

(C) Strict segregation of enteropeptidase exclusively within the brush border of the duodenal mucosa

(D) Maintenance of an acidic intramembranous pH within the trans-Golgi network and mature granules

Q2. A 3-year-old child is brought to the pediatrics clinic due to developmental delay, poor muscle tone, and a tendency to self-mutilate by biting his lips and fingers. Laboratory studies demonstrate marked hyperuricemia. The patient is diagnosed with Lesch-Nyhan syndrome, an X-linked recessive disorder caused by a complete deficiency of hypoxanthine-guanine phosphoribosyltransferase (HGPRT). Which of the following biochemical changes best describes the metabolic consequence of this enzyme deficiency on the purine nucleotide synthesis pathway?

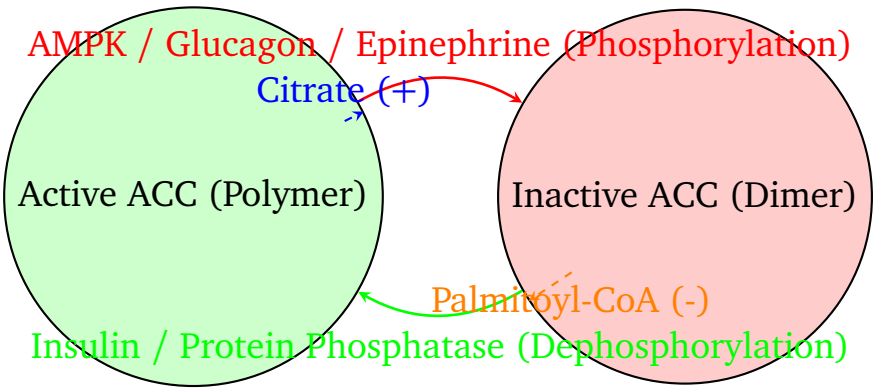
(A) Decreased intracellular levels of PRPP (5-phosphoribosyl-1-pyrophosphate) due to lack of consumption in the salvage pathway

(B) Impaired feedback inhibition of PRPP glutamyl amidotransferase leading to accelerated de novo purine synthesis

(C) Accumulation of adenosine deaminase intermediates resulting in T-cell toxic dATP buildup

(D) Upregulation of xanthine oxidase activity due to a direct allosteric activation by hypoxanthine

Q3. An investigator is studying the regulatory mechanisms of fatty acid synthesis in human hepatocytes. The rate-limiting step of this pathway is catalyzed by Acetyl-CoA Carboxylase (ACC), which converts acetyl-CoA to malonyl-CoA. The activity of ACC is tightly regulated by both allosteric modulators and reversible covalent modification as outlined below:



Based on this regulatory network, which of the following metabolic states will result in the highest rate of de novo lipogenesis?

(A) High intracellular energy charge, elevated circulating insulin levels, and high cytosolic citrate concentrations

(B) Low intracellular energy charge, elevated circulating glucagon levels, and high palmitoyl-CoA concentrations

(C) High intracellular cyclic AMP (cAMP) levels, activated protein phosphatase 2A, and low citrate concentrations

(D) Elevated AMP-activated protein kinase (AMPK) activity, low insulin-to-glucagon ratio, and high citrate concentrations

Q4. A 28-year-old woman is evaluated for generalized fatigue and mild icterus. A peripheral blood smear reveals spherocytes, and a direct antiglobulin test is negative, suggesting a hereditary red cell membrane or metabolic defect. Further biochemical assay confirms a significant deficiency in Pyruvate Kinase (PK) activity within her erythrocytes. Which of the following changes in the erythrocyte metabolic profile is uniquely observed in PK deficiency compared to other proximal glycolytic enzyme defects?

(A) Depletion of 2,3-bisphosphoglycerate (2,3-BPG) leading to an increased affinity of hemoglobin for oxygen

(B) Accumulation of 2,3-bisphosphoglycerate (2,3-BPG) leading to a rightward shift in the oxygen-hemoglobin dissociation curve

(C) Complete cessation of the Hexose Monophosphate (HMP) shunt due to feedback inhibition by fructose-1,6-bisphosphate

(D) Inability to maintain the red cell membrane lipid bilayer due to a severe deficiency of cytosolic NADPH

Q5. During a laboratory session, medical students are analyzing DNA replication kinetics using E. coli extracts. They introduce a targeted mutation into the gene encoding DNA Polymerase I, specifically disrupting its 5-to-3 exonuclease activity while fully preserving its polymerase and 3-to-5 exonuclease functions. Which of the following molecular phenotypes will be demonstrated by this mutant bacterial strain during replication?

(A) Inability to synthesize the leading strand continuously from the origin of replication

(B) Accumulation of Okazaki fragments containing intact RNA primers at their 5-ends

(C) A significantly increased mutation rate due to the loss of replication proofreading capability

(D) Failure of DNA topoisomerase to resolve positive supercoils ahead of the advancing replication fork

Q6. A 30-year-old male is undergoing chemotherapy for acute myeloid leukemia. To prevent the development of tumor lysis syndrome, he is started on Allopurinol. This drug effectively reduces the formation of uric acid by acting on Xanthine Oxidase. What is the precise mechanism of inhibition exerted by allopurinol and its active metabolite, oxypurinol, on xanthine oxidase?

(A) Oxypurinol acts as a suicide inhibitor (mechanism-based inactivator) by coordinating tightly to the molybdenum-iron center of the enzyme

(B) Allopurinol acts as a non-competitive inhibitor, binding to an allosteric site and altering the enzyme conformation

(C) Oxypurinol functions as a reversible competitive inhibitor that competes exclusively with hypoxanthine without binding the active site covalently

(D) Allopurinol causes irreversible uncompetitive inhibition by trapping the enzyme-substrate complex after xanthine binds

Q7. A newborn screening panel detects highly elevated levels of immunoreactive trypsinogen, and subsequent genetic testing confirms that the infant is homozygous for the Delta-F508 mutation in the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) gene. This specific mutation involves the deletion of three base pairs encoding a phenylalanine residue. Which of the following intracellular sorting errors represents the primary pathogenic mechanism of this molecular defect?

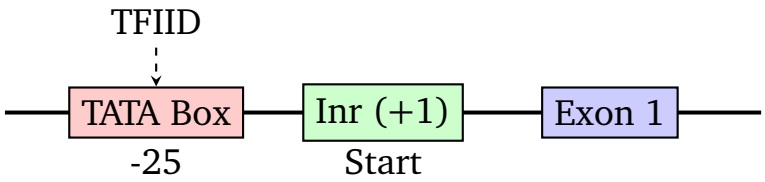
(A) Normal synthesis but absolute failure of the protein to integrate into the endoplasmic reticulum membrane

(B) Premature termination of translation resulting in a truncated, non-functional cytosolic polypeptide

(C) Recognition of the misfolded protein by endoplasmic reticulum quality control mechanisms, leading to its degradation via the ubiquitin-proteasome pathway

(D) Defective trafficking from the Golgi apparatus to the plasma membrane due to aberrant terminal sialylation

Q8. A molecular biologist is mapping transcription factor binding kinetics onto a specific eukaryotic gene. The diagram below represents a transcription transcription unit highlighting the relationship between structural and regulatory elements:



During assembly of the pre-initiation complex (PIC) at this TATA-box containing promoter, which of the following general transcription factors is responsible for recognizing and binding directly to the TATA box sequence to initiate the recruitment of RNA Polymerase II?

(A) TFIIA

(B) TFIIB

(C) TFIID via its TBP (TATA-binding protein) subunit

(D) TFIIH via its ATP-dependent helicase subunit

Q9. A 55-year-old male is evaluated for progressive peripheral neuropathy, ataxia, and mild cognitive decline. His dietary history is notable for strict veganism for the past 12 years without nutritional supplementation. Laboratory tests reveal a macrocytic anemia with hypersegmented neutrophils, alongside significantly elevated serum levels of both homocysteine and methylmalonic acid. The accumulation of methylmalonic acid in this patient is due to the impairment of an enzyme that requires which of the following coenzyme forms of vitamin B12?

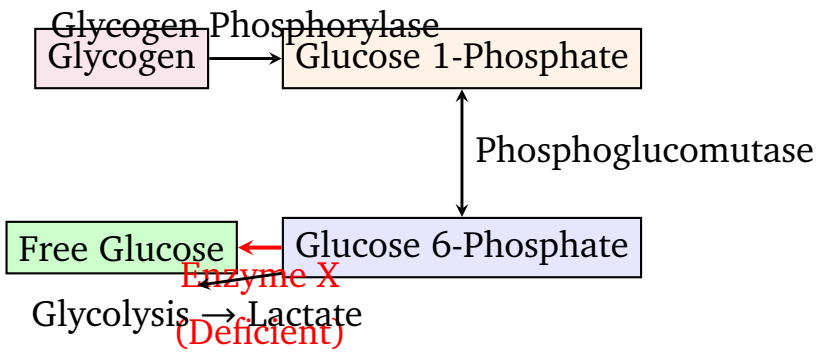
(A) Methylcobalamin

(B) 5-Deoxyadenosylcobalamin

(C) Hydroxocobalamin

(D) Cyanocobalamin

Q10. A 6-month-old infant is brought to the physician because of severe hepatomegaly, fasting hypoglycemia, and failure to thrive. Administration of glucagon or epinephrine does not lead to an increase in blood glucose levels, but instead produces a marked rise in blood lactate. A liver biopsy shows an enormous accumulation of glycogen with normal structure. The biochemical pathway of glycogenolysis and its intersection with gluconeogenesis is shown below:



Based on the clinical presentation and the metabolic layout, the deficient Enzyme X is located primarily within which of the following subcellular compartments?

(A) Cytosol

(B) Mitochondrial Matrix

(C) Lumen of the Smooth Endoplasmic Reticulum

(D) Lysosomal Matrix

Q11. A 12-year-old boy presents with severe photosensitivity, blistering, and hyperpigmentation on sun-exposed areas of his skin. The family history is positive for skin malignancies at an early age. He is diagnosed with Xeroderma Pigmentosum. This disease highlights the critical importance of repairing DNA lesions induced by ultraviolet radiation. Which of the following sequential steps correctly outlines the molecular mechanism of the repair pathway that is defective in this patient?

(A) Glycosylase cleavage of damaged base → AP endonuclease nicking → DNA Polymerase beta filling → DNA Ligase

(B) Endonuclease cleavage on both sides of the lesion → Excision of an oligonucleotide fragment → DNA Polymerase delta/epsilon synthesis → DNA Ligase

(C) Mismatch recognition by MutS homolog → MutL recruitment → Exonuclease I digestion → DNA Polymerase delta → DNA Ligase

(D) Photolyase activation by visible light → Direct monomerization of cyclobutane pyrimidine dimers without strand cleavage

Q12. An investigator is studying the structural features of human tRNA molecules involved in translation. She isolates a fraction of cytoplasmic tRNAs and analyzes their primary and tertiary conformations. Which of the following statements regarding the structural motifs of a mature, functional eukaryotic tRNA molecule is completely accurate?

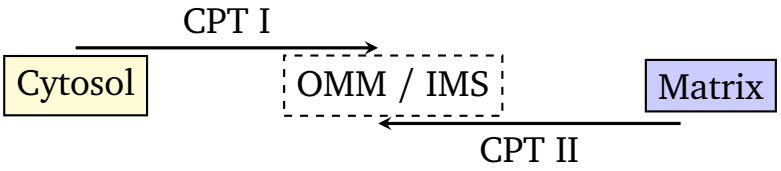
(A) The amino acid is covalently attached via an ester bond to the 5-phosphorylated end of the tRNA molecule

(B) The CCA sequence at the 3-hydroxyl terminus is added post-transcriptionally in the nucleus by a specific nucleotidyltransferase

(C) The anticodon loop contains the highly conserved T-psi-C motif responsible for binding to the small ribosomal subunit

(D) The D-loop is rich in pseudouridine residues and determines the specific charging fidelity of aminoacyl-tRNA synthetase

Q13. A 2-month-old infant is evaluated for persistent lethargy and hypoketotic hypoglycemia after an overnight fast. Plasma acylcarnitine profiling demonstrates a significant accumulation of long-chain fatty acylcarnitines, but free carnitine levels are reduced. A deficiency in the Carnitine Palmitoyltransferase I (CPT I) enzyme is suspected. Consider the carnitine shuttle pathway diagrammed below:



Under physiological conditions, which of the following metabolic intermediates acts as a powerful allosteric inhibitor of CPT I, preventing the simultaneous oxidation of fatty acids during active fatty acid synthesis?

(A) Acetyl-CoA

(B) Malonyl-CoA

(C) Propionyl-CoA

(D) Acetoacetyl-CoA

Q14. A 35-year-old woman is evaluated for a suspected endocrine disorder. Genomic analysis of her peripheral blood leukocytes reveals a mutation in a gene encoding a specific G-protein coupled receptor kinase. Further investigation focuses on the molecular mechanisms regulating her cellular response to hormones. Which of the following statements correctly identifies the exact biochemical event that directly terminates the signaling activity of the alpha-subunit of a heterotrimeric G-protein complex?

(A) Dissociation of the G-alpha subunit from the G-beta-gamma dimeric complex

(B) Intrinsic GTPase activity of the G-alpha subunit hydrolyzing bound GTP to GDP

(C) Phosphorylation of the G-alpha subunit by an activated protein kinase A (PKA)

(D) Receptor-mediated exchange of bound GDP for a molecule of cytosolic GTP

Q15. A medical genetics team is analyzing a pedigree of a family affected by a rare metabolic bone phenotype. The condition appears to affect both males and females, and affected fathers transmit the condition to all of their daughters but to none of their sons. Affected mothers transmit the condition to approximately half of their sons and half of their daughters. Which of the following modes of inheritance is completely consistent with this transmission pattern?

(A) Autosomal Dominant

(B) X-linked Recessive

(C) X-linked Dominant

(D) Mitochondrial (Maternal) Inheritance

Q16. A 20-year-old male athlete consumes a raw egg diet consisting of 8 to 10 raw egg whites daily as part of a extreme bodybuilding regimen. After several months, he develops severe dermatitis, glossitis, alopecia, and mild paresthesias. A deficiency of biotin (Vitamin B7) is diagnosed, induced by the binding of raw egg white avidin to biotin. This vitamin serves as an essential coenzyme for which of the following groups of enzymes?

(A) Carboxylases

(B) Aminotransferases (Transaminases)

(C) Dehydrogenases

(D) Decarboxylases

Detailed Solutions

Q1.

Solution

Concept:

Acute pancreatitis involves auto-digestion of pancreatic tissue due to premature intrapancreatic activation of proteolytic zymogens. Trypsinogen conversion to active trypsin represents the central trigger event because active trypsin subsequently processes and activates all other digestive zymogens within the cascade.

Solution:

(a) Under normal physiological conditions, pancreatic acinar cells synthesize and package proteolytic enzymes as inactive zymogens within membrane-bound secretory granules.

(b) To safeguard against accidental intracellular activation caused by trace amounts of trypsinogen auto-activation, acinar cells co-package Pancreatic Secretory Trypsin Inhibitor (PSTI), encoded by the SPINK1 gene.

(c) PSTI acts as a highly specific, immediate physical antagonist by binding directly to the active site of prematurely formed trypsin, rendering it catalytically inert.

(d) If this protective inhibitor system is overwhelmed or genetically defective, active trypsin accumulates, initiates downstream zymogen activation, and causes acute parenchymal necrosis.

(e) Other factors like keeping enteropeptidase isolated in the duodenum are crucial external safeguards, but intrinsic PSTI synthesis provides the absolute primary intra-acinar defense.

Final Answer: Synthesis and co-packaging of Pancreatic Secretory Trypsin Inhibitor (PSTI / SPINK1) within zymogen granules.

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept:

Lesch-Nyhan syndrome is caused by a complete absence of the purine salvage enzyme hypoxanthine-guanine phosphoribosyltransferase (HGPRT). This enzymatic block leads to a severe accumulation of substrate intermediates alongside excessive de novo purine nucleotide overproduction.

Solution:

(a) HGPRT normally consumes 5-phosphoribosyl-1-pyrophosphate (PRPP) to convert hypoxanthine and guanine into IMP and GMP. When HGPRT is absent, PRPP usage drops drastically, leading to high intracellular PRPP accumulation.

(b) PRPP acts as an essential substrate and a powerful allosteric feed-forward activator for PRPP glutamyl amidotransferase, the rate-limiting enzyme of the de novo purine pathway.

(c) Concurrently, the failure of the salvage pathway lowers intracellular levels of IMP and GMP, eliminating normal feedback inhibition on PRPP glutamyl amidotransferase.

(d) The dual impact of elevated PRPP stimulation and lost purine nucleotide feedback inhibition dramatically accelerates de novo purine synthesis.

(e) Unsalvaged hypoxanthine and purine degradation products are shifted entirely into the xanthine oxidase pathway, generating extreme hyperuricemia and clinical self-mutilation phenotypes.

Final Answer: Impaired feedback inhibition of PRPP glutamyl amidotransferase leading to accelerated de novo purine synthesis.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept:

Acetyl-CoA Carboxylase (ACC) is the key rate-limiting enzyme governing de novo fatty acid biogenesis. It transitions between an active polymeric form and an inactive dimeric form, dictated by cellular energy status and hormonal cues.

Solution:

(a) ACC activity is stimulated via dephosphorylation by protein phosphatases, which are directly upregulated by insulin signaling in well-fed states.

(b) High ATP levels signify a high energy charge, shutting down the energy-sensing kinase AMPK, which would otherwise phosphorylate and inactivate ACC.

(c) Citrate functions as a highly potent local allosteric activator that physically drives the polymerization and activation of ACC dimers.

(d) High cytosolic citrate confirms an abundant supply of carbon precursors moving out of the mitochondria for lipogenic processing.

(e) Conversely, glucagon, epinephrine, and palmitoyl-CoA act as inhibitory inputs that push the enzyme toward its inactive dimeric state.

Final Answer: High intracellular energy charge, elevated circulating insulin levels, and high cytosolic citrate concentrations.

Answer: (A)

Go Back to Question 3

Q4.

Solution

Concept:

Pyruvate kinase (PK) catalyzes the final step of anaerobic glycolysis, converting phosphoenolpyruvate into pyruvate with the generation of ATP. Erythrocytes lack mitochondria and rely exclusively on this pathway for energy production.

Solution:

(a) A metabolic block at pyruvate kinase causes a significant upstream backup of glycolytic intermediates within the erythrocyte cytoplasm.

(b) This systemic accumulation forces a marked increase in flux through the Luebering-Rapoport shunt, directly generating 2,3-bisphosphoglycerate (2,3-BPG).

(c) Elevated internal levels of 2,3-BPG bind tightly to deoxyhemoglobin, stabilizing its low-affinity T-state conformation.

(d) This structural stabilization causes a rightward shift in the oxygen-hemoglobin dissociation curve, facilitating enhanced oxygen unloading to tissues.

(e) This elevated 2,3-BPG profile is unique to PK deficiency; defects in more proximal enzymes diminish downstream intermediates, lowering 2,3-BPG.

Final Answer: Accumulation of 2,3-bisphosphoglycerate (2,3-BPG) leading to a rightward shift in the oxygen-hemoglobin dissociation curve.

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept:

Bacterial DNA Polymerase I is a multi-functional enzyme that plays a vital role in processing Okazaki fragments on the lagging strand during genomic replication. It possesses independent polymerase, proofreading, and primer-removal domains.

Solution:

(a) Okazaki fragment maturation requires the complete removal of RNA primers positioned at the 5-end of newly synthesized lagging strand segments.

(b) The 5-to-3 exonuclease activity of DNA Polymerase I is uniquely designed to recognize these nicks and hydrolyze the RNA primer ahead of it.

(c) Simultaneously, its polymerase activity synthesizes DNA to replace the excised primer track via nick translation.

(d) Selectively mutating the 5-to-3 exonuclease domain eliminates the cell's ability to remove these temporary ribonucleotide sequences.

(e) Consequently, lagging strand synthesis stalls with Okazaki fragments attached to intact RNA primers, preventing proper DNA ligation.

Final Answer: Accumulation of Okazaki fragments containing intact RNA primers at their 5-ends.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

Xanthine oxidase handles the sequential oxidation of hypoxanthine to xanthine, and xanthine to uric acid. Allopurinol is an analog substrate that undergoes processing by the enzyme to yield an exceptionally high-affinity inhibitor.

Solution:

(a) Xanthine oxidase initially converts allopurinol into its active metabolite, oxypurinol (alloxanthine), through its standard catalytic cycle.

(b) Oxypurinol remains bound within the active site and coordinates directly with the reduced molybdenum-iron cofactor center.

(c) This chemical coordination freezes the enzyme in an inactive state, preventing further substrate turnover.

(d) This phenomenon, where an enzyme converts an innocuous substrate into an irreversible inactivator, defines suicide or mechanism-based inhibition.

(e) It differs from simple competitive inhibition because it renders the specific enzyme molecule completely inactive via stable, long-term coordination.

Final Answer: Oxypurinol acts as a suicide inhibitor (mechanism-based inactivator) by coordinating tightly to the molybdenum-iron center of the enzyme.

Answer: (A)

Go Back to Question 6

Q7.

Solution

Concept:

The Delta-F508 mutation in the cystic fibrosis transmembrane conductance regulator (CFTR) gene results in the deletion of a single phenylalanine residue, causing significant changes in protein tertiary structure.

Solution:

(a) The missing phenylalanine residue prevents the nascent CFTR peptide chain from folding into its native tertiary conformation within the endoplasmic reticulum.

(b) The endoplasmic reticulum quality control machinery recognizes the mutant CFTR variant as an aberrantly folded protein.

(c) Chaperone networks halt its forward transport to the Golgi apparatus and target it for retrotranslocation into the cytosol.

(d) Once inside the cytosol, the misfolded protein undergoes polyubiquitination and complete degradation by the 26S proteasome.

(e) Because the channel protein never reaches the apical membrane, epithelial chloride secretion is lost, inducing cystic fibrosis pathology.

Final Answer: Recognition of the misfolded protein by endoplasmic reticulum quality control mechanisms, leading to its degradation via the ubiquitin-proteasome pathway.

Answer: (C)

Go Back to Question 7

Q8.

Solution

Concept:

Eukaryotic transcription initiation requires the orderly assembly of general transcription factors (GTFs) along with RNA Polymerase II at the core promoter region to establish a functional pre-initiation complex.

Solution:

(a) The TATA box is a highly conserved, adenylate- and thymidylate-rich regulatory sequence situated approximately 25 base pairs upstream of the transcription start site.

(b) General transcription factor IID (TFIID) acts as the initial scaffolding component that recognizes and docks at this core region.

(c) TFIID achieves this locating step through its specialized TATA-binding protein (TBP) subunit, which bends the DNA double helix.

(d) The physical bending induced by TBP creates a structural platform that enables the sequential recruitment of TFIIA, TFIIB, and RNA Polymerase II.

(e) Other factors like TFIIH possess enzymatic functions like helicase activity but do not perform the primary sequence recognition step.

Final Answer: TFIID via its TBP (TATA-binding protein) subunit.

Answer: (C)

Go Back to Question 8

Q9.

Solution

Concept:

Vitamin B12 (cobalamin) functions as an essential cofactor for two distinct metabolic enzymes in humans: methionine synthase and methylmalonyl-CoA mutase. Deficiencies impair both pathways, causing distinct clinical biochemical markers.

Solution:

(a) Methylmalonyl-CoA mutase facilitates the structural rearrangement of methylmalonyl-CoA into succinyl-CoA within the propionate degradation pathway.

(b) This specific intramolecular isomerization requires the 5-deoxyadenosylcobalamin coenzyme form of vitamin B12 to stabilize radical intermediates.

(c) Prolonged dietary deficiency compromises this step, leading to an accumulation of upstream methylmalonic acid in tissues and serum.

(d) Elevated methylmalonic acid disrupts myelin synthesis, causing the subacute combined degeneration of the spinal cord seen in this patient.

(e) The alternate coenzyme form, methylcobalamin, is utilized exclusively by cytosolic methionine synthase to convert homocysteine to methionine.

Final Answer: 5-Deoxyadenosylcobalamin.

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept:

Von Gierke disease (Glycogen Storage Disease Type Ia) is caused by a deficiency in Glucose-6-Phosphatase, the critical final enzyme shared by both glycogenolysis and gluconeogenesis pathways.

Solution:

(a) Glucose-6-phosphate is generated in the cytosol during glycogen breakdown or de novo gluconeogenesis from substrates like lactate.

(b) To release free glucose, glucose-6-phosphate must be transported out of the cytosol into the lumen of the smooth endoplasmic reticulum.

(c) The active site of the Glucose-6-Phosphatase enzyme is uniquely sequestered on the luminal face of the smooth endoplasmic reticulum membrane.

(d) A defect in this compartmentalized system prevents the conversion of glucose-6-phosphate to free glucose, locking it inside the cell.

(e) The trapped intermediate is diverted into anaerobic glycolysis, causing lactic acidosis, severe fasting hypoglycemia, and hepatomegaly.

Final Answer: Lumen of the Smooth Endoplasmic Reticulum.

Answer: (C)

Go Back to Question 10

Q11.

Solution

Concept:

Xeroderma Pigmentosum is an autosomal recessive disorder characterized by a striking deficiency in the Nucleotide Excision Repair (NER) pathway. This particular pathway functions as the cell primary defense mechanism against bulky, helix-distorting DNA lesions induced by ultraviolet radiation.

Solution:

(a) Ultraviolet light exposure causes adjacent pyrimidine bases to form abnormal covalent linkages, producing harmful cyclobutane pyrimidine dimers or 6-4 photoproducts.

(b) In a healthy individual, the specialized multi-protein NER complex scans the genome, locates these structural distortions, and introduces dual incisions on the damaged strand.

(c) Specific endonucleases cut the damaged phosphodiester backbone at precise locations both 5-prime and 3-prime relative to the lesion site.

(d) This dual cleavage releases an oligonucleotide fragment containing the damaged bases, leaving a localized single-stranded gap in the duplex DNA.

(e) High-fidelity DNA Polymerase delta or epsilon fills the gap by synthesizing new DNA using the intact complementary strand as a template, and DNA Ligase seals the remaining nick.

Final Answer: Endonuclease cleavage on both sides of the lesion → Excision of an oligonucleotide fragment → DNA Polymerase delta/epsilon synthesis → DNA Ligase.

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept:

Transfer RNA (tRNA) molecules serve as crucial adaptors that decode messenger RNA sequences into specific polypeptide chains during translation. Each tRNA molecule undergoes highly orchestrated, multi-step post-transcriptional processing before it becomes functional.

Solution:

(a) Eukaryotic tRNA genes are initially transcribed by RNA Polymerase III inside the nucleus to produce precursor molecules containing extra flanking sequences.

(b) The 5-prime leader sequence is precisely removed by the ribozyme RNase P, while the 3-prime trailer sequence is trimmed away by specific exonucleases.

(c) Following trailer removal, the universal, non-templated 5-prime-CCA-3-prime trinucleotide sequence is attached directly to the 3-prime hydroxyl terminus.

(d) This critical modification is executed by a template-independent nucleotidyltransferase inside the nucleus prior to nuclear export.

(e) The terminal adenosine residue of this newly added CCA sequence serves as the precise covalent attachment site for the incoming cognate amino acid.

Final Answer: The CCA sequence at the 3-hydroxyl terminus is added post-transcriptionally in the nucleus by a specific nucleotidyltransferase.

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept:

The carnitine shuttle functions as the rate-limiting regulatory gatekeeper for the mitochondrial beta-oxidation of long-chain fatty acids. This process is tightly controlled to prevent futile cycles of simultaneous lipid synthesis and degradation.

Solution:

(a) During well-fed states, active de novo fatty acid biogenesis takes place inside the cytosol, generating large quantities of Malonyl-CoA.

(b) Malonyl-CoA is produced directly from acetyl-CoA by the enzyme acetyl-CoA carboxylase, which serves as the committed step of lipogenesis.

(c) To prevent newly synthesized fatty acids from entering mitochondria for degradation, Malonyl-CoA acts as a powerful allosteric inhibitor of Carnitine Palmitoyltransferase I.

(d) This inhibition blocks the transfer of long-chain acyl groups from coenzyme A onto carnitine molecules at the outer mitochondrial membrane.

(e) When Malonyl-CoA levels decline during fasting, this allosteric blockade is lifted, allowing long-chain acylcarnitines to cross into the matrix for beta-oxidation.

Final Answer: Malonyl-CoA.

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Heterotrimeric G-proteins function as molecular switches that cycle between active and inactive states. They convert extracellular hormonal cues from G-protein coupled receptors into controlled intracellular secondary messenger signals.

Solution:

(a) In the baseline inactive state, the alpha subunit binds guanosine diphosphate (GDP) and remains associated with the regulatory beta-gamma dimeric complex.

(b) Ligand binding to the receptor triggers a conformational shift that induces the alpha subunit to exchange its bound GDP for a molecule of GTP.

(c) The binding of GTP causes the alpha subunit to dissociate from the beta-gamma complex, enabling it to modulate downstream effector enzymes.

(d) The active signaling duration of the alpha subunit is determined by its own built-in, intrinsic GTPase catalytic activity.

(e) This intrinsic enzyme activity autonomously hydrolyzes the bound GTP back into GDP, causing the subunit to reassociate with the inhibitory beta-gamma dimer.

Final Answer: Intrinsic GTPase activity of the G-alpha subunit hydrolyzing bound GTP to GDP.

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept:

Analyzing single-gene clinical pedigrees requires evaluating the specific distribution of a phenotype among male and female offspring relative to the clinical status of their parents.

Solution:

(a) An affected father carries the mutant allele on his single X chromosome and passes his Y chromosome to all of his sons.

(b) Consequently, he transmits the mutant X chromosome to all of his daughters, making them all inherit the dominant trait.

(c) The observation that all daughters but none of the sons of an affected father develop the trait strongly eliminates autosomal and recessive patterns.

(d) An affected heterozygous mother passes either her normal X or her mutant X chromosome to her children with equal probability.

(e) This matches the observed clinical distribution where approximately half of her sons and half of her daughters inherit the bone phenotype.

Final Answer: X-linked Dominant.

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept:

Biotin (Vitamin B7) functions as an essential water-soluble coenzyme involved in carbon dioxide fixation pathways. Raw egg whites contain avidin, a glycoprotein that binds biotin with extreme affinity, completely blocking its intestinal absorption.

Solution:

(a) Biotin serves as a specialized carrier of activated carboxyl groups, executing ATP-dependent carboxylation reactions across key metabolic systems.

(b) It forms a covalent amide bond with a specific lysine residue located within the active site of carboxylase enzymes.

(c) Key enzymes reliant on this coenzyme include pyruvate carboxylase for gluconeogenesis and acetyl-CoA carboxylase for de novo lipogenesis.

(d) It is also required by propionyl-CoA carboxylase, which processes odd-chain fatty acids and specific amino acids into the citric acid cycle.

(e) Deficiencies lead to impaired organic acid metabolism, culminating in the clinical presentation of dermatitis, alopecia, and neurological abnormalities.

Final Answer: Carboxylases.

Answer: (A)

Go Back to Question 16

Answer Key

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