NEET PG Biochemistry Sample Paper-1

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 ataxia, confusion, and nystagmus. Laboratory evaluation reveals a state of severe thiamine deficiency. Which of the following biochemical conversions will be most directly impaired in this patient?

(A) Succinate to fumarate

(B) Pyruvate to acetyl-CoA

(C) Malate to oxaloacetate

(D) Isocitrate to α-ketoglutarate

Q2. A researcher is analyzing the replication of an exotic viral genome. She observes that DNA synthesis is initiated normally, but elongation is rapidly halted due to the presence of an aberrant, highly stable RNA-DNA hybrid structure that the replication machinery fails to resolve. Defect or inhibition of which of the following prokaryotic enzyme functions mimics this phenotype?

(A) DNA polymerase I $5' \rightarrow 3'$ exonuclease activity

(B) DNA polymerase I $3' \rightarrow 5'$ exonuclease activity

(C) DNA polymerase III holoenzyme assembly

(D) DNA topoisomerase II subunit cleavage activity

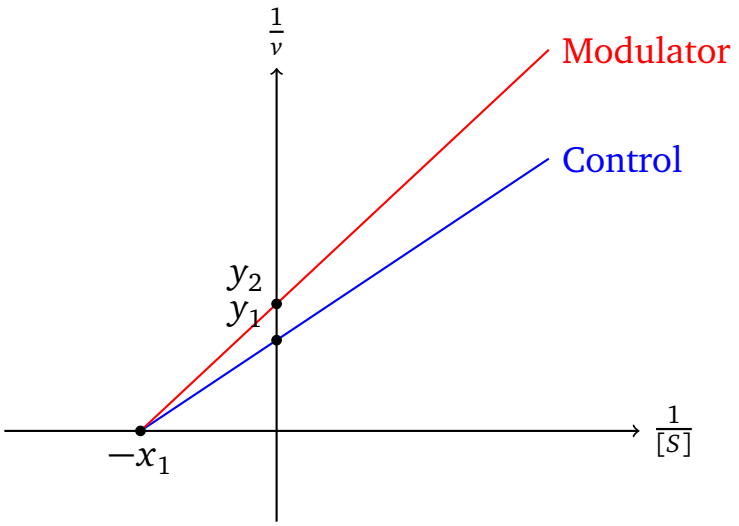
Q3. An infant presenting with severe mental retardation, macroorchidism, and large prominent ears is diagnosed with Fragile X syndrome. The molecular pathology involves trinucleotide repeat expansion in the FMR1 gene. What is the precise mechanism by which this genomic alteration leads to a loss of function of the FMR1 protein?

(A) Alternative splicing leading to an unstable frame-shifted protein transcript ypermethylation of CpG islands within the promoter region causing transcriptional silencing

(B) Premature stop codon formation resulting in nonsense-mediated decay of mRNA

(C) Defective nuclear export of the processed mRNA transcript due to abnormal polyadenylation

Q4. Consider the given double-reciprocal (Lineweaver-Burk) plot illustrating the kinetics of a key regulatory enzyme of fatty acid synthesis in the presence and absence of a specific metabolic modulator.



Based on this kinetic profile, which of the following statements accurately characterizes the mechanism of action of the modulator?

(A) It acts as a competitive inhibitor, increasing K_m while leaving V_{max} unaltered

(B) It acts as a non-competitive inhibitor, decreasing V_{max} while leaving K_m unaltered

(C) It acts as an uncompetitive inhibitor, decreasing both V_{max} and K_m

(D) It acts as an allosteric activator, increasing enzyme affinity for its primary substrate

Q5. A 50-year-old male is evaluated for extreme fatigue, dark urine, and scleral icterus. A peripheral blood smear shows numerous schistocytes and bite cells. He was recently prescribed a course of primaquine for malaria chemoprophylaxis. This clinical presentation is fundamentally driven by a metabolic block in which of the following pathways?

(A) Gluconeogenesis

(B) Pentose Phosphate Pathway

(C) Glycogenolysis

(D) Citric Acid Cycle

Q6. A 3-year-old boy is brought to the clinic due to global developmental delay and compulsive self-mutilating behaviors, including biting his lips and fingers. Laboratory studies demonstrate highly elevated serum uric acid levels. The primary enzyme defect in this patient involves which type of metabolic reaction?

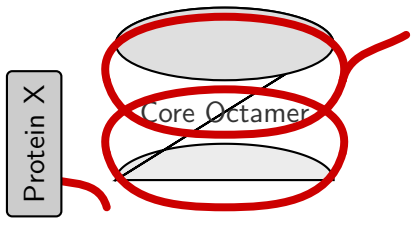
(A) De novo purine nucleotide biosynthesis

(B) Purine nucleotide salvage synthesis

(C) Pyrimidine nucleotide degradation

(D) De novo pyrimidine nucleotide biosynthesis

Q7. A laboratory investigator is studying the structural organization of eukaryotic chromatin. He constructs a representation of the fundamental repeating structural subunit of chromatin as shown below.



Which amino acid residue configuration would you expect to be most densely enriched within the structural core complex depicted above to facilitate its tight interaction with genomic DNA?

(A) Glutamate and Aspartate

(B) Lysine and Arginine

(C) Leucine and Valine

(D) Tryptophan and Phenylalanine

Q8. An infant born to consanguineous parents presents with severe hypotonia, psychomotor retardation, and a distinctive musty body odor. A screening test indicates markedly elevated plasma phenylalanine. However, subsequent biochemical assay reveals that the activity of the enzyme phenylalanine hydroxylase itself is completely within the normal reference range. A deficiency in the synthesis or recycling of which of the following cofactors best explains these findings?

(A) Tetrahydrobiopterin (BH_4)

(B) Pyridoxal phosphate (PLP)

(C) Flavin adenine dinucleotide (FAD)

(D) Thiamine pyrophosphate (TPP)

Q9. A newborn is evaluated for profound fasting hypoglycemia, hepatomegaly, and severe lactic acidosis. Intravenous administration of epinephrine or glucagon fails to induce an increase in blood glucose levels, but causes a prompt, marked increase in blood lactate. A liver biopsy shows significantly elevated levels of glycogen possessing abnormally short outer branches. Which of the following enzymes is most likely deficient?

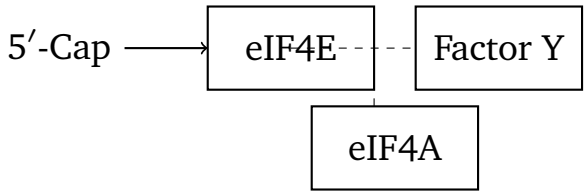
(A) Glucose-6-phosphatase

(B) Glycogen phosphorylase

(C) Amylo-α-(1,6)-glucosidase

(D) Phosphofructokinase-1

Q10. The diagrammatic scheme below captures the eukaryotic mRNA translation initiation apparatus assembling around the 5′ cap structure.



The component labeled as "Factor Y" serves as a critical scaffolding bridge that directly coordinates the binding of eIF4E, eIF4A, and the poly(A) binding protein (PABP). Which of the following is Factor Y?

(A) eIF4G

(B) eIF3

(C) eIF2

(D) eIF4B

Q11. A 2-week-old female infant is evaluated for failure to thrive, persistent vomiting after milk ingestion, and early cataract formation. Urinalysis reveals the presence of reducing sugars, but a glucose oxidase dipstick test is negative. What is the primary intracellular metabolic trap that leads to the pathophysiological manifestations observed in this condition?

(A) Accumulation of Galactose-1-phosphate in tissues

(B) Accumulation of Sorbitol in the lens and liver

(C) Depletion of Fructose-1-phosphate in hepatocytes

(D) Deficient synthesis of UDP-glucose

Q12. A patient presents with extreme sensitivity to ultraviolet radiation, demonstrating multiple skin carcinomas at a very early age. The biochemical defect involves a breakdown in the nucleotide excision repair pathway. Which of the following enzymatic events occurs first during the normal operation of this specific DNA repair pathway in healthy human cells?

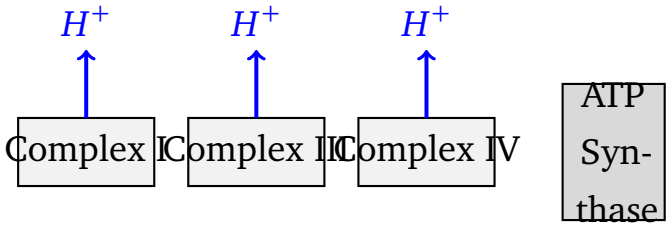
(A) Removal of an altered single base by a specific DNA glycosylase

(B) Recognition of helical distortion followed by endonucleolytic cleavage of the damaged strand

(C) $3' \rightarrow 5'$ exonucleolytic degradation of the entire damaged template strand

(D) Direct photochemical reversal of pyrimidine dimers by a light-activated lyase

Q13. A patient is diagnosed with a rare neurodegenerative disorder characterized by defective mitochondrial protein import. Biochemical analysis shows that proteins destined for the mitochondrial matrix are synthesized normally in the cytosol but fail to translocate across the inner mitochondrial membrane. This translocation step relies heavily on a proton motive force. The diagram below shows the structural layout of the inner mitochondrial membrane respiratory assemblies.



If a specific chemical compound completely blocks the translocation of protons through Complex IV without altering the rest of the electron transport chain, what will be the primary direct impact on the generation of the mitochondrial matrix proton gradient?

(A) Total abolition of the proton gradient across the inner membrane

(B) Reduction, but not complete loss, of the proton gradient due to continued pumping by Complexes I and III

(C) An immediate increase in the proton gradient due to feedback activation of Complex I

(D) Reversal of the proton gradient direction through ATP synthase operating in reverse

Q14. A 55-year-old corporate executive presents with sudden onset of severe, agonizing pain and swelling in his left first metatarsophalangeal joint. Synovial

fluid analysis under polarized light microscopy confirms the diagnosis of acute gouty arthritis. Colchicine is prescribed. What is the exact mechanism by which colchicine treats this acute metabolic presentation?

(A) Competitive inhibition of xanthine oxidase, lowering uric acid production

(B) Inhibition of tubulin polymerization, preventing neutrophil migration and degranulation

(C) Uricosuric activation of renal organic anion transporters to enhance excretion

(D) Allosteric inhibition of PRPP synthetase within the purine biosynthetic pathway

Q15. A molecular biologist isolates a mutant strain of E. coli that expresses the enzymes of the lac operon constitutively, regardless of the presence or absence of lactose in the medium. Sequencing reveals a mutation in the lacI gene. Which of the following molecular descriptions best characterizes the defect in the protein encoded by this mutated lacI gene?

(A) An altered repressor protein that cannot bind to the operator locus

(B) An altered repressor protein that binds irreversibly to the operator locus

(C) An altered repressor protein that cannot bind the inducer molecule (allolactose)

(D) An altered CAP protein that cannot bind cyclic AMP (cAMP)

Q16. A 6-month-old child presents with progressive psychomotor regression, macrocephaly, and an exaggerated startle response to loud noises. Ophthalmoscopic exam reveals a striking cherry-red spot on the macula. No hepatosplenomegaly is detected. This clinical presentation is a direct consequence of an inability to degrade which of the following lipid classes?

(A) Sphingomyelin

(B) GM_2 ganglioside

(C) Glucocerebroside

(D) Galactocerebroside

Detailed Solutions

Q1.

Solution

Concept:

Thiamine (Vitamin B1) serves as an essential precursor for Thiamine Pyrophosphate (TPP), a vital cofactor required for oxidative decarboxylation and transketolase reactions. Chronic alcohol consumption severely compromises thiamine status by impairing its active intestinal absorption, decreasing hepatic storage, and reducing chemical phosphorylation to its active coenzyme form, causing acute neurological breakdown.

Solution:

(a) The conversion of pyruvate to acetyl-CoA is catalyzed by the multienzyme Pyruvate Dehydrogenase Complex (PDHC) located inside the mitochondrial matrix space.

(b) PDHC requires five distinct coenzymes to function sequentially: TPP (B_1), FAD (B_2), NAD+ (B_3), Coenzyme A (B_5), and Lipoic Acid.

(c) In severe thiamine deficiency, PDHC activity drops precipitously because the E_1 pyruvate dehydrogenase subunit lacks its bound TPP catalytic activator.

(d) This enzymatic block restricts pyruvate from entering the Citric Acid Cycle, forcing its alternative conversion into lactate via anaerobic pathways.

(e) While α-ketoglutarate dehydrogenase also requires TPP, the conversion of succinate to fumarate depends solely on Succinate Dehydrogenase (FAD-dependent), and malate to oxaloacetate depends on Malate Dehydrogenase (NAD-dependent).

Final Answer: Pyruvate to acetyl-CoA

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept:

Bacterial DNA replication requires the continuous and systematic removal of transient RNA primers from lagging-strand Okazaki fragments. Failure to excise these RNA segments creates highly stable, non-functional RNA-DNA hybrid segments that warp the physical topology of the duplex, blocking further processive progression of the replication fork machinery.

Solution:

(a) E. coli DNA Polymerase I is a specialized, multifunctional repair enzyme that possesses a distinct $5' \rightarrow 3'$ exonuclease catalytic activity housed on its small structural domain.

(b) This specific $5' \rightarrow 3'$ exonuclease function is uniquely capable of degrading RNA nucleotides that are base-paired to a DNA template strand ahead of it.

(c) As DNA Polymerase I advances, it simultaneously hydrolyzes the old RNA primer in front of it and fills the resulting molecular gap with new DNA nucleotides.

(d) If this $5' \rightarrow 3'$ exonuclease mechanism is inactivated or defective, the replication machinery cannot clear the existing RNA primers from the template.

(e) The persistence of these un-cleaved RNA primers creates stubborn RNA-DNA hybrids that arrest the structural advancement of the processive DNA Polymerase III holoenzyme.

Final Answer: DNA polymerase I $5' \rightarrow 3'$ exonuclease activity

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept:

Fragile X syndrome is characterized by an abnormal expansion of CGG trinucleotide repeats within the 5′ untranslated region (5′ UTR) of the FMR1 gene on the X chromosome. This massive genetic expansion modifies local chromatin structure through specific epigenetic mechanisms, turning a transcriptionally active locus into heterochromatin and eliminating protein production.

Solution:

(a) Normal individuals typically carry fewer than 50 CGG repeats, whereas affected individuals express a full mutation containing more than 200 expanded triplet repeats.

(b) This high density of CGG repeats introduces an excessive concentration of CpG dinucleotide islands directly within the promoter and regulatory architecture of the FMR1 gene.

(c) Cellular DNA methyltransferases recognize these dense CpG clusters and systematically attach methyl groups to the cytosine bases, inducing severe hypermethylation.

(d) The hypermethylated DNA then recruits Methyl-CpG-binding domain proteins, which associate with histone deacetylases to pack the local chromatin tightly.

(e) This localized epigenetic remodeling blocks basic transcription factors from binding to the promoter, resulting in complete transcriptional silencing and zero mRNA synthesis.

Final Answer: Hypermethylation of CpG islands within the promoter region causing transcriptional silencing

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept:

Enzyme inhibition kinetics can be deciphered by evaluating shifts in the intercepts of a Lineweaver-Burk double-reciprocal plot. Modulators modify the apparent affinity (K_m) or the maximal catalytic velocity (V_{max}) of an enzyme, which alters the slope and axial intersections of the plotted lines.

Solution:

(a) The Lineweaver-Burk equation plots the reciprocal of initial velocity ($1/v$) on the vertical y-axis against the reciprocal of substrate concentration ($1/[S]$) on the horizontal x-axis.

(b) The vertical y-intercept represents the exact mathematical value of $1/V_{max}$, while the horizontal x-intercept corresponds directly to the value of $-1/K_m$.

(c) In the provided TikZ diagram, the control plot and the modulated plot intersect perfectly at the exact same point on the negative horizontal x-axis ($-x_1$).

(d) Because the x-intercept remains identical in both states, the Michaelis constant (K_m) of the regulatory enzyme is entirely unaltered by the modulator.

(e) However, the y-intercept increases significantly from y_1 to y_2 under the influence of the modulator, indicating that the true V_{max} has decreased ($V_{max2} < V_{max1}$), a classic hallmark of pure non-competitive inhibition.

Final Answer: It acts as a non-competitive inhibitor, decreasing V_{max} while leaving K_m unaltered

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept:

Erythrocytes depend entirely on the Pentose Phosphate Pathway (PPP) to generate the reducing equivalents of NADPH. NADPH is crucial for maintaining glutathione in its reduced state, which neutralizes dangerous reactive oxygen species and safeguards red blood cell membranes against destructive lipid peroxidation.

Solution:

(a) Glucose-6-Phosphate Dehydrogenase (G6PD) catalyzes the first rate-limiting step of the Pentose Phosphate Pathway, yielding NADPH.

(b) Reduced glutathione uses electrons from NADPH to convert toxic hydrogen peroxide into harmless water molecules within the erythrocyte cytoplasm.

(c) Antimalarial medications like primaquine generate significant oxidative stress by inducing free radical formation within circulating red blood cells.

(d) Patients with an underlying genetic block in the PPP cannot generate adequate levels of NADPH to restore reduced glutathione reserves during oxidative challenges.

(e) This molecular failure leads to hemoglobin denaturation (forming Heinz bodies) and severe hemolysis, presenting as schistocytes, bite cells, and scleral icterus.

Final Answer: Pentose Phosphate Pathway

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

Lesch-Nyhan syndrome is an X-linked recessive disorder characterized by a severe deficiency in the purine salvage enzyme Hypoxanthine-Guanine Phosphoribosyltransferase (HGPRT). This single enzymatic failure prevents cells from recycling free purine bases, causing a severe accumulation of uric acid and critical neurological disruption.

Solution:

(a) The HGPRT enzyme normally catalyzes the purine nucleotide salvage synthesis pathway by condensing free hypoxanthine or guanine bases with PRPP to form IMP or GMP.

(b) When purine salvage synthesis is blocked, the intracellular concentration of hypoxanthine and guanine rises rapidly as recycling becomes impossible.

(c) These excess un-salvaged purine bases are immediately directed into the downstream purine degradation pathway, where xanthine oxidase converts them into uric acid.

(d) Furthermore, the loss of purine salvage causes intracellular PRPP levels to skyrocket, which accelerates de novo purine biosynthesis via feed-forward activation.

(e) The combined effect of increased de novo synthesis and elevated degradation generates extreme hyperuricemia, driving the classic presentation of cognitive delay and self-mutilation.

Final Answer: Purine nucleotide salvage synthesis

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept:

Eukaryotic genomic DNA is organized into nucleosome repeating subunits to achieve high levels of spatial compaction within the nucleus. The structural core octamer relies entirely on stable electrostatic interactions with the phosphodiester backbone of DNA, requiring a unique and specific amino acid composition.

Solution:

(a) The double-stranded DNA helix possesses a highly negative electrical charge due to the repeating phosphate groups that form its structural backbone.

(b) To wrap around the core octamer, the histone proteins (H2A, H2B, H3, and H4) must express a net positive surface charge to facilitate binding.

(c) The basic amino acids, Lysine and Arginine, carry positively charged side chains at physiological pH values due to their specialized amino and guanidino groups.

(d) Structural analysis shows that the histone core octamer is heavily enriched with these basic Lysine and Arginine residues, stabilizing the entire assembly.

(e) Acidic amino acids like glutamate would repel the DNA, while hydrophobic residues like leucine are buried internally, making basic residues the primary choice.

Final Answer: Lysine and Arginine

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept:

Hyperphenylalaninemia can stem from a structural defect in the Phenylalanine Hydroxylase (PAH) apoenzyme or from a deficiency in its mandatory non-protein coenzyme, Tetrahydrobiopterin (BH4). When BH4 is missing, multiple monooxygenase enzyme systems fail simultaneously, resulting in a complex neurological presentation.

Solution:

(a) Phenylalanine Hydroxylase requires molecular oxygen and BH4 to successfully convert phenylalanine into the amino acid tyrosine.

(b) If BH4 synthesis or its recycling by Dihydrobiopterin Reductase is impaired, PAH cannot function, even if the PAH enzyme structure is normal.

(c) BH4 is also an absolute requirement for Tyrosine Hydroxylase and Tryptophan Hydroxylase, which govern the synthesis of dopamine, epinephrine, and serotonin.

(d) Consequently, a cofactor deficiency causes a drop in central neurotransmitter levels, leading to severe hypotonia and psychomotor regression.

(e) Accumulation of alternative phenylalanine metabolites like phenylacetate generates the classic musty body odor, indicating a clear defect in the BH4 system.

Final Answer: Tetrahydrobiopterin (BH_4)

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept:

Cori disease (Glycogen Storage Disease Type III) is caused by a genetic deficiency in the glycogen debranching enzyme, Amylo-α-(1,6)-glucosidase. This deficiency prevents the complete breakdown of glycogen polymer branches, producing a distinct structural abnormality within stored glycogen reserves.

Solution:

(a) Glycogen phosphorylase can cleave linear α-(1,4)-glucosidic bonds but stops working four glucosyl residues away from a branch point, leaving a limit dextrin.

(b) The glycogen debranching enzyme possesses two catalytic tasks: a transferase function and an amylo-α-(1,6)-glucosidase function that removes the remaining branch residue.

(c) Without this debranching activity, glycogen breakdown is halted, leaving behind an altered glycogen structure with short, stubby outer branches.

(d) When fasting, glucagon and epinephrine stimulate glycogen degradation, but the structural block prevents glucose release, resulting in severe fasting hypoglycemia.

(e) The liver shifts to gluconeogenesis to generate glucose, which increases the accumulation of lactic acid, triggering a profound systemic lactic acidosis.

Final Answer: Amylo-α-(1,6)-glucosidase

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept:

Eukaryotic translation initiation requires the assembly of the multi-subunit eIF4F cap-binding complex on the 5′ end of processed mRNA transcripts. This biochemical complex acts as a physical bridge that brings the mRNA to the 40S ribosomal subunit to start protein synthesis.

Solution:

(a) The functional eIF4F complex is made up of three distinct initiation factors: eIF4E, eIF4A, and the large scaffolding factor labeled as Factor Y.

(b) The eIF4E subunit directly recognizes and binds to the 7-methylguanosine cap structure located at the extreme 5′ end of the mRNA molecule.

(c) The eIF4A subunit serves as an ATP-dependent RNA helicase that unwinds local secondary hairpin structures within the 5′ untranslated region.

(d) Factor Y represents eIF4G, a large scaffolding protein that contains specific binding sites for eIF4E, eIF4A, and the poly(A) binding protein (PABP).

(e) By binding to PABP on the 3′ tail and eIF4E on the 5′ cap, eIF4G circularizes the mRNA transcript, maximizing translation efficiency.

Final Answer: eIF4G

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept:

Classic galactosemia is an autosomal recessive metabolic disorder caused by a congenital deficiency of the enzyme Galactose-1-phosphate uridylyltransferase. The inability to process galactose leads to the rapid cellular entrapment of intermediate metabolites within highly vulnerable metabolic tissues, initiating cytotoxic injury, osmotic imbalances, and early organ damage.

Solution:

(a) Ingested lactose is hydrolyzed into glucose and galactose, after which galactose is phosphorylated into galactose-1-phosphate by the action of hepatic galactokinase.

(b) The absolute deficiency of galactose-1-phosphate uridylyltransferase completely blocks the subsequent conversion of galactose-1-phosphate into UDP-galactose.

(c) This enzymatic disruption leads to a severe intracellular accumulation of galactose-1-phosphate in the liver, brain, kidneys, and ocular lenses.

(d) High levels of intracellular galactose-1-phosphate act as a metabolic toxin, depleting inorganic phosphate pools, damaging cellular structures, and impairing liver function.

(e) Excess free galactose enters alternative pathways where aldose reductase reduces it into galactitol, an osmotically active sugar alcohol that accumulates in the lens.

(f) The accumulation of galactitol draws fluid into the lens fibers, creating physical opacities that cause early cataracts, while un-metabolized reducing sugars spill into the urine.

Final Answer: Accumulation of Galactose-1-phosphate in tissues

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept:

Nucleotide Excision Repair (NER) is a highly conserved genomic maintenance pathway designed to identify and remove bulky, helix-distorting DNA lesions. These structural distortions are typically caused by ultraviolet radiation, chemical carcinogens, or large adducts that interfere with normal transcription and replication processes.

Solution:

(a) The nucleotide excision repair process initiates with a specialized scanning mechanism where specific multi-subunit protein complexes monitor the genome.

(b) In humans, the XPC complex continuously surveys the DNA duplex to recognize structural abnormalities and helical distortions caused by pyrimidine dimers.

(c) Once a bulky lesion is identified, transcription factor IIH (TFIIH) is recruited to utilize its helicase subunits to unwind the surrounding DNA strands.

(d) Following structural stabilization, dual endonucleases (XPF and XPG) make specific incisions on both the $5'$ and $3'$ sides of the damaged strand.

(e) A short oligonucleotide fragment containing the damaged bases is excised and released from the duplex, leaving a single-stranded gap.

(f) DNA polymerase fills the newly created gap using the intact complementary strand as a template, and DNA ligase seals the remaining nick.

Final Answer: Recognition of helical distortion followed by endonucleolytic cleavage of the damaged strand

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept:

The mitochondrial electron transport chain (ETC) uses the free energy released by sequential electron transfers to pump protons from the matrix into the intermembrane space. This directional pumping generates an electrochemical proton motive force across the inner membrane, which fuels chemical ATP synthesis and drives protein translocation.

Solution:

(a) The mitochondrial respiratory assembly features three distinct proton pumps: NADH-coenzyme Q oxidoreductase, Coenzyme Q-cytochrome c reductase, and Cytochrome c oxidase.

(b) Complex I transfers electrons from NADH to ubiquinone, capturing sufficient free energy to translocate four protons across the inner mitochondrial membrane.

(c) Complex III processes electrons from ubiquinone and transfers them to cytochrome c, pumping an additional four protons into the intermembrane space.

(d) Complex IV receives electrons from reduced cytochrome c molecules to reduce oxygen to water, pumping two additional protons across the inner membrane bilayer.

(e) Blocking the proton translocation function of Complex IV selectively prevents it from adding its normal contribution to the intermembrane proton pool.

(f) Because Complexes I and III continue to transfer electrons and pump protons normally, a significant, functional proton gradient is maintained across the membrane.

Final Answer: Reduction, but not complete loss, of the proton gradient due to continued pumping by Complexes I and III

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Acute gouty arthritis is an inflammatory condition triggered by the crystallization of monosodium urate within joint spaces due to persistent hyperuricemia. The deposition of these needles initiates a massive localized immune response characterized by leukocyte recruitment, phagocytosis, and the release of pro-inflammatory cytokines.

Solution:

(a) Monosodium urate crystals in the synovial fluid are recognized by local macrophages, which release chemotactic factors to recruit circulating neutrophils to the joint.

(b) Neutrophils migrate into the synovium, phagocytose the urate crystals, and undergo activation, releasing lysosomal enzymes and interleukins that intensify the pain.

(c) Colchicine treats this condition by binding specifically to soluble tubulin heterodimers, which prevents their assembly into functional microtubule polymers.

(d) Microtubules are essential structural components required for cellular motility, intracellular vesicle transport, and secretory degranulation pathways.

(e) Disrupting the microtubule network impairs neutrophil migration, limits chemokinesis, and blocks the release of inflammatory mediators into the joint space.

(f) Unlike allopurinol, colchicine does not alter purine metabolism or xanthine oxidase activity, working instead as an anti-inflammatory agent during acute attacks.

Final Answer: Inhibition of tubulin polymerization, preventing neutrophil migration and degranulation

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept:

The lac operon is a classic genetic model of transcriptional regulation governed by coordinated interactions between repressor proteins, operator sites, and inducer molecules. Mutations within regulatory genes alter the structural domains of these control elements, shifting the system from inducible expression to un-regulated constitutive operation.

Solution:

(a) The regulatory lacI gene encodes a homotetrameric repressor protein that maintains a high affinity for the specific nucleotide sequence of the lac operator.

(b) Under baseline conditions without lactose, the functional lacI repressor binds to the operator, physically blocking RNA polymerase from transcribing the structural genes.

(c) When lactose is available, it is converted into allolactose, an inducer that binds to the repressor and causes a conformational change that lowers its operator affinity.

(d) A loss-of-function mutation within the DNA-binding domain of the lacI gene produces an altered repressor protein that cannot bind to the operator locus.

(e) Because the operator remains unoccupied, RNA polymerase has unhindered access to the promoter, enabling continuous transcription of the lac operon enzymes.

(f) This constitutive expression proceeds independently of inducer presence, as the mutant repressor fails to interact with the regulatory DNA sequence.

Final Answer: An altered repressor protein that cannot bind to the operator locus

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept:

Tay-Sachs disease is an autosomal recessive lysosomal storage disorder caused by a genetic deficiency in the α-subunit of the lysosomal enzyme β-hexosaminidase A. This enzymatic block stops the degradation of specific sphingolipids, resulting in their progressive accumulation within central nervous system tissues.

Solution:

(a) Lysosomal enzymes systematically break down complex membrane sphingolipids through sequential hydrolytic steps to maintain normal cellular lipid homeostasis.

(b) β-Hexosaminidase A is responsible for cleaving the terminal N-acetylgalactosamine residue from polar GM2 gangliosides inside neuronal lysosomes.

(c) The complete deficiency of functional β-hexosaminidase A stops this catabolic pathway, causing GM2 gangliosides to accumulate inside neurons.

(d) This toxic accumulation forces lysosomes to swell, disrupting normal cellular physiology and triggering widespread neurodegeneration across the central nervous system.

(e) Swollen, lipid-laden ganglion cells throughout the peripheral retina create a stark visual contrast against the thin macula, presenting as a cherry-red spot.

(f) Sphingomyelin accumulation is seen in Niemann-Pick disease (associated with hepatosplenomegaly), whereas Tay-Sachs disease presents with neurodegeneration without enlargement of visceral organs.

Final Answer: GM_2 ganglioside

Answer: (B)

Go Back to Question 16

Answer Key

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