NEET PG Biochemistry Sample Paper-6

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 3-year-old boy presents with profound fasting hypoglycemia, hepatomegaly, and severe lactic acidosis. Laboratory evaluation reveals marked hyperuricemia and hyperlipidemia. A liver biopsy shows significantly elevated glycogen content with normal structure. The patient fails to exhibit a rise in blood glucose following an intravenous administration of galactose or fructose. Defect in which of the following enzymes is the primary driver of this presentation?

(A) Glucose-6-phosphatase

(B) Glycogen phosphorylase

(C) Amylo-α-(1,6)-glucosidase

(D) Phosphofructokinase-1

Q2. A molecular biologist isolates a mutant strain of *Escherichia coli* that exhibits remarkably high rates of spontaneous mutations. Biochemical assay of DNA Polymerase III isolated from this strain reveals a completely abolished $3' \rightarrow 5'$ exonuclease activity, while its $5' \rightarrow 3'$ polymerase activity remains entirely intact. Which structural component or subunit of the DNA Polymerase III holoenzyme complex is most likely defective in this mutant strain?

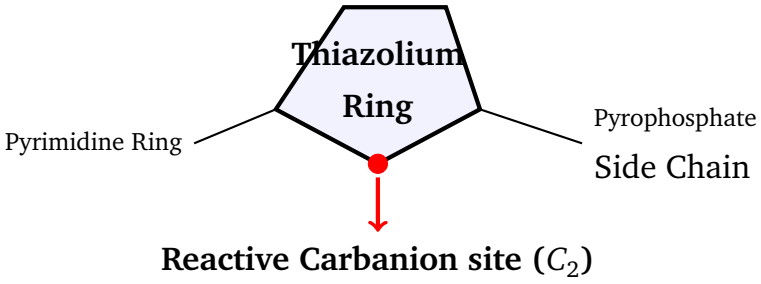
(A) α subunit

(B) ϵ subunit

(C) θ subunit

(D) β_2 clamp

Q3. A 45-year-old chronic alcoholic presents to the emergency department with confusion, horizontal nystagmus, and severe ataxia. The attending physician suspects a classic nutritional deficiency. The critical enzyme system affected requires a specific vitamin cofactor whose active diagnostic parameters are mapped on the reaction mechanism profile shown below. Identify the target coenzyme synthesized from this vitamin that acts directly at the indicated catalytic center:



(A) Thiamine pyrophosphate (TPP)

(B) Pyridoxal phosphate (PLP)

(C) Flavin adenine dinucleotide (FAD)

(D) Nicotinamide adenine dinucleotide (NAD^+)

Q4. An investigator analyzes a pedigree of a rare metabolic disorder characterized by progressive neurodegeneration and lactic acidosis. The disease affects both males and females. Affected males never transmit the disease to their offspring, whereas affected females transmit the trait to all of their children, though with highly variable clinical severity among siblings. This phenotypic variability within a single kinship is best explained by which genetic mechanism?

(A) Variable expressivity due to heteroplasmy

(B) Genomic imprinting at paternal loci

(C) Gonadal mosaicism in the mother

(D) Allelic heterogeneity at nuclear loci

Q5. A 2-week-old neonate presents with poor feeding, vomiting, and progressive jaundice. Physical examination reveals bilateral cataracts and hepatomegaly. Urinalysis is strongly positive for reducing sugars but completely negative for glucose via glucose oxidase strips. The infant is immediately placed on a lactose-free formula. Accumulation of which metabolite within the crystalline lens is responsible for cataract formation in this infant?

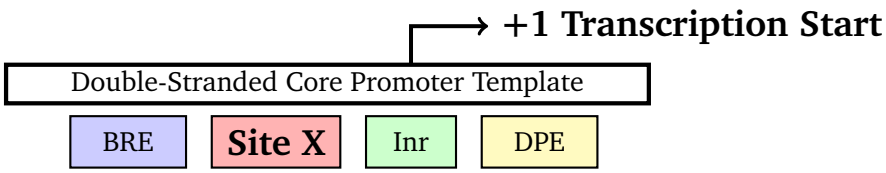
(A) Galactose-1-phosphate

(B) Galactitol

(C) Sorbitol

(D) Fructose-1-phosphate

Q6. An experimental oncology trial evaluates a new small-molecule inhibitor targeting eukaryotic transcription initiation. Investigators perform a high-resolution footprinting assay to pinpoint where the primary transcription factors assemble on the core promoter elements of a target oncogene, as mapped schematically below. Identify the exact site block where the TFIID complex initiates assembly via structural recognition of its TATA-binding protein (TBP) subunit:



(A) BRE (TFIIB Recognition Element)

(B) Site X (TATA Box)

(C) Inr (Initiator Element)

(D) DPE (Downstream Promoter Element)

Q7. Lineweaver-Burk plots are utilized to analyze the inhibition profiles of a novel therapeutic agent designed against a key bacterial metabolic pathway.

In the presence of a constant concentration of the inhibitor, the vertical intercept ($1/V_{max}$) of the reciprocal plot remains completely unchanged, whereas the horizontal intercept ($-1/K_m$) shifts significantly closer to the origin. This mathematical transformation is characteristic of which type of enzyme inhibition?

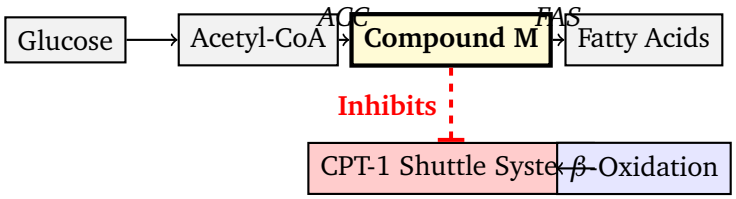
(A) Competitive inhibition

(B) Non-competitive inhibition

(C) Uncompetitive inhibition

(D) Suicide irreversible inhibition

Q8. A translational research group investigates the regulatory nodes controlling the flux of hepatic fatty acid synthesis versus β-oxidation. The biochemical cross-talk between these opposing pathways is managed via reciprocal feedback pathways mediated by an allosteric regulator. Based on the regulatory network scheme provided below, identify the correct molecular identity of intermediate **Compound M** which blocks carnitine palmitoyltransferase-1 (CPT-1):



(A) Malonyl-CoA

(B) Acetoacetyl-CoA

(C) Citrate

(D) Propionyl-CoA

Q9. A 5-year-old child presents with global developmental delay, self-mutilating behavior (lip and finger biting), hyperuricemia, and choreoathetosis. Urinalysis demonstrates a massive excretion of orange-colored uric acid crystals. This clinical picture arises from a deep defect in the purine salvage pathway. The absent enzyme normally utilizes which of the following activated sugar complexes as a substrate donor?

(A) 5-Phosphoribosyl-1-pyrophosphate (PRPP)

(B) Ribose-5-phosphate

(C) Uridine diphosphate glucose (UDP-Glucose)

(D) Cytidine diphosphate choline (CDP-Choline)

Q10. A medical student undergoing intense physical training completes a prolonged 30-kilometer marathon. During the final legs of the race, muscle glycogen stores are entirely depleted, and the liver maintains systemic euglycemia primarily via de novo gluconeogenesis. Which of the following pairs of substrates serve as purely gluconeogenic precursors requiring mitochondrial transport loops for processing into glucose?

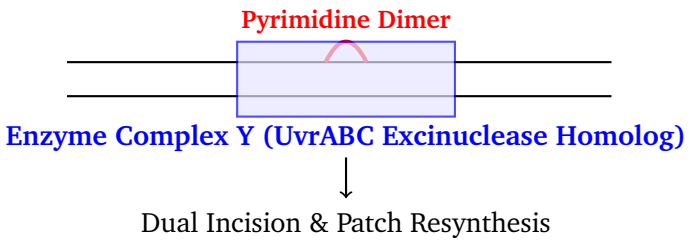
(A) Lactate and Pyruvate

(B) Glycerol and Alanine

(C) Acetyl-CoA and Acetoacetate

(D) Leucine and Lysine

Q11. A 34-year-old woman is evaluated for a suspicious skin lesion on her forearm. Molecular testing reveals a defect in a specialized DNA repair pathway that fixes damage induced by environmental radiation ultraviolet exposure. The specific biochemical cascade involves recognizing and excising oligonucleotide segments around the distortion. According to the repair mechanism structural blueprint below, name the pathognomonic clinical phenotype that directly results from a deficiency in **Enzyme Complex Y**:



(A) Xeroderma Pigmentosum

(B) Hereditary Nonpolyposis Colorectal Cancer (HNPCC)

(C) Ataxia Telangiectasia

(D) Li-Fraumeni Syndrome

Q12. A full-term infant develops severe hyperammonemia, tachypnea, and lethargy within 36 hours of birth. Plasma amino acid analysis reveals markedly elevated concentrations of citrulline, reaching levels more than 50 times the upper limit of normal. Orotic acid concentrations in the urine are within normal physiological parameters. Which of the following enzymes is structurally or functionally compromised in this newborn patient?

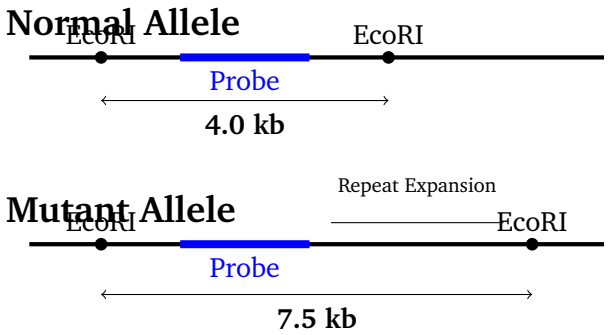
(A) Argininosuccinate synthetase

(B) Carbamoyl phosphate synthetase I

(C) Ornithine transcarbamylase

(D) Argininosuccinate lyase

Q13. A clinical geneticist uses restriction enzyme digestion followed by Southern blotting to identify carriers of a trinucleotide repeat expansion disorder. The normal and mutant alleles are shown below. A labeled probe hybridizes to the highlighted genomic region. Which fragment size would be detected on the Southern blot in a homozygous mutant individual?



(A) 4.0 kb

(B) 7.5 kb

(C) 11.5 kb

(D) 3.5 kb

Q14. A newborn is evaluated for hypotonia, developmental delay, and severe seizure activity. Laboratory workup reveals elevated levels of methylmalonic

acid in the blood and urine. Further cellular assays confirm that the total levels of cobalamin (Vitamin B_{12}) inside the cells are normal, but there is a complete inability to synthesize adenosylcobalamin. Methylmalonyl-CoA mutase requires this cofactor to convert its substrate into which Krebs cycle intermediate?

(A) Succinyl-CoA

(B) Oxaloacetate

(C) α-Ketoglutarate

(D) Fumarate

Q15. During eukaryotic translation initiation, the scan mechanism precisely positions the 40S ribosomal subunit at the correct initiation codon embedded within a consensus flanking sequence. If the sequence elements surrounding the start codon are mutated away from the classical Kozak consensus sequence framework, which of the following molecular outcomes is most likely to be observed?

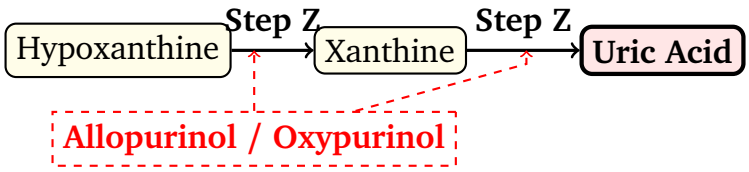
(A) Total failure of 60S subunit recruitment

(B) Reduced efficiency of translation initiation with alternative start-site selection

(C) Complete block of peptidyltransferase activity during elongation

(D) Accelerated degradation of mRNA via the nonsense-mediated decay pathway

Q16. A 58-year-old male presenting with acute, excruciating pain, redness, and swelling in the first metatarsophalangeal joint is diagnosed with an acute gouty flare. Joint aspirate confirms the presence of negatively birefringent, needle-shaped crystals. The pharmacodynamic intervention focuses on halting the purine degradation loop. Based on the enzymatic logic provided below, which enzyme is directly inhibited by the first-line maintenance agent Allopurinol at **Step Z**?



(A) Hypoxanthine-guanine phosphoribosyltransferase

(B) Xanthine oxidase

(C) Adenosine deaminase

(D) PRPP synthetase

Detailed Solutions

Q1.

Solution

Concept: Von Gierke disease (Glycogen Storage Disease Type I) is caused by a metabolic deficiency in glucose-6-phosphatase. This enzyme resides on the luminal surface of the endoplasmic reticulum and catalyzes the terminal common step of both glycogenolysis and gluconeogenesis: cleaving the phosphate group from glucose-6-phosphate to produce free glucose.

Solution:

Let's analyze the physical signs, laboratory results, and dynamic metabolic behaviors described:

(a) The 3-year-old child presents with a combination of fasting hypoglycemia, hepatomegaly, and severe lactic acidosis, accompanied by hyperuricemia and hyperlipidemia.

(b) A liver biopsy reveals elevated concentrations of structurally normal glycogen (ruling out debranching or branching enzyme mutations like GSD III or IV).

(c) The diagnostic test shows that blood glucose fails to rise following the administration of fructose or galactose. These monosaccharides enter glycolysis downstream, where they are metabolized into glucose-6-phosphate:

$$\text{Galactose/Fructose} \rightarrow \cdots \rightarrow \text{Glucose-6-Phosphate} \xrightarrow{\text{G6Pase}} \text{Glucose}$$

Because the terminal **glucose-6-phosphatase** enzyme is defective, the cell cannot convert this accumulated pool into free glucose, and the substrates are instead shunted into lactic acid, uric acid, and lipid synthesis pathways.

Final Answer: Glucose-6-phosphatase

Answer: (A)

Go Back to Question 1

Q2.

Solution

Concept: Prokaryotic DNA Polymerase III is a complex holoenzyme that serves as the primary engine for replicative genomic synthesis. High fidelity replication depends on a specialized multi-subunit core that contains distinct locations for nucleotide polymerization and immediate proofreading exonuclease repair.

Solution:

Let's assign the specific functional tasks to the structural subunits of the DNA Polymerase III catalytic core:

(a) α **subunit (dnaE):** Responsible for the standard $5' \rightarrow 3'$ polymerase catalytic elongation activity along the primed template.

(b) ϵ **subunit (dnaQ):** Houses the primary **$3' \rightarrow 5'$ exonuclease proofreading activity**. This domain detects and hydrolyzes mispaired nucleotides at the immediate $3'$ end of the growing strand before elongation continues.

(c) When the ϵ subunit is functionally compromised or mutated, the proofreading activity is abolished while the $5' \rightarrow 3'$ polymerase action remains intact, causing a dramatic increase in uncorrected replication mismatches and spontaneous mutations.

(d) The θ subunit acts as a structural stabilizer for ϵ, and the β_2 homodimer clamp forms the sliding ring that maintains high processivity.

Final Answer: ϵ subunit

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept: Wernicke-Korsakoff syndrome is a neurological disorder caused by severe nutritional deficiency of thiamine (Vitamin B_1), which is common in chronic alcohol use disorders due to poor dietary intake and impaired GI absorption. Thiamine is the direct precursor for a vital organic coenzyme that participates in central carbohydrate oxidative decarboxylation pathways.

Solution:

Let's map the structural coenzyme mechanism illustrated in the diagram:

(a) Vitamin B_1 is converted inside cells into its active coenzyme form, **thiamine pyrophosphate (TPP)**.

(b) As detailed in the topology map, the functional heart of TPP is its central thiazolium ring. The acidic carbon atom situated between the nitrogen and sulfur atoms (C_2) readily deprotonates to yield a highly reactive, stabilized **reactive carbanion site**.

(c) This carbanion attacks carbonyl carbons on substrates like pyruvate and α-ketoglutarate, stabilizing the decarboxylation intermediates. A structural deficiency in **thiamine pyrophosphate (TPP)** restricts the activity of pyruvate dehydrogenase and α-ketoglutarate dehydrogenase, depriving brain tissue of required ATP.

Final Answer: Thiamine pyrophosphate (TPP)

Answer: (A)

Go Back to Question 3

Q4.

Solution

Concept: Mitochondrial inheritance (maternal transmission) describes traits encoded by genes within the circular mitochondrial DNA (mtDNA). Because mature spermatozoa do not contribute functional cytoplasm or organelles to the zygote at fertilization, mitochondrial mutations are transmitted exclusively along the maternal line.

Solution:

Let's evaluate the inheritance behavior and phenotypic variations across generations:

(a) The pedigree reveals that affected males never pass the condition to their children, whereas affected females transmit the trait to all of their offspring. This matches a maternal, non-Mendelian mitochondrial inheritance profile.

(b) During early embryonic cleavage divisions, mitochondria are randomly distributed into daughter blastomeres, a process known as replicative segregation.

(c) If a mother carries a mixture of wild-type and mutant mitochondrial genomes, this state is called **heteroplasmy**. Random segregation causes sibling tissues to inherit vastly different ratios of mutant vs normal mtDNA. This variation in mutation load leads to **variable expressivity**, altering the clinical severity of the neurodegenerative phenotype among siblings.

Final Answer: Variable expressivity due to heteroplasmy

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: Classic galactosemia is an autosomal recessive disorder most frequently caused by a deficiency in galactose-1-phosphate uridyltransferase (GALT). When milk containing lactose is ingested, it is cleaved into glucose and galactose, causing galactose and its metabolic intermediates to pool within tissues.

Solution:

Let's track the alternative metabolic conversion that takes place within the lens tissue:

(a) Because the primary pathway is blocked, excess circulating free galactose enters peripheral tissues like the lens of the eye.

(b) In the lens, the enzyme aldose reductase reduces the aldehyde group of galactose into its corresponding polyol sugar alcohol form, **galactitol**:

$$\text{Galactose} + \text{NADPH} + \text{H}^+ \xrightarrow{\text{Aldose Reductase}} \text{Galactitol} + \text{NADP}^+$$

(c) **Galactitol** is osmotically active and cannot diffuse out of the cells. Its accumulation draws water into the lens fibers, causing cellular swelling, protein denaturation, and early-onset bilateral cataract formation. Sorbitol accumulation causes a similar cataract mechanism in diabetic patients, but is derived from glucose rather than galactose.

Final Answer: Galactitol

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: Transcription initiation by eukaryotic RNA Polymerase II requires the sequential assembly of general transcription factors (GTFs) onto core promoter consensus sequence elements positioned upstream of the transcription start site (+1).

Solution:

Let's analyze the organization of elements within the core promoter blueprint:

(a) The assembly cascade begins when the multiprotein complex **TFIID** recognizes and binds the promoter region.

(b) Specifically, the TATA-binding protein (TBP) subunit of TFIID directly attaches to the **TATA box (Site X)**, a highly conserved consensus sequence rich in adenine and thymine base pairs located approximately 25 to 30 base pairs upstream of the initiation locus.

(c) This specific structural binding distorts and bends the DNA double helix, creating a molecular platform that recruits TFIIA, TFIIB (via the BRE element), TFIIF, and RNA Polymerase II to complete the pre-initiation complex assembly.

Final Answer: Site X (TATA Box)

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: Lineweaver-Burk plots convert the hyperbolic Michaelis-Menten relationship into a linear equation by plotting $1/V_0$ versus $1/[S]$. On these axes, the vertical y-intercept corresponds to $1/V_{max}$ and the horizontal x-intercept corresponds to $-1/K_m$.

Solution:

Let's determine the kinetic parameter changes from the geometric properties described:

(a) The problem states that in the presence of the inhibitor, the vertical intercept ($1/V_{max}$) remains completely unchanged. This indicates that the maximum velocity (**V_{max}**) of the enzyme system is unaffected by the inhibitor.

(b) Meanwhile, the horizontal intercept ($-1/K_m$) shifts closer to the origin (a less negative value), which means that the apparent Michaelis constant (**K_m**) has increased.

(c) A kinetic profile where V_{max} is unchanged and K_m increases is the defining hallmark of **competitive inhibition**. In this state, the inhibitor binds reversibly to the active site, and its inhibitory effects can be overcome by adding excess substrate.

Final Answer: Competitive inhibition

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Fatty acid synthesis and β-oxidation are reciprocal metabolic pathways that are tightly coordinated to prevent a futile cycle. The primary control point is the regulation of carnitine palmitoyltransferase-1 (CPT-1), the outer mitochondrial membrane enzyme responsible for shifting long-chain fatty acyl-CoAs into the matrix.

Solution:

Let's follow the signaling intermediates outlined in the regulatory network scheme:

(a) When glucose levels are high, insulin activates acetyl-CoA carboxylase (ACC). ACC catalyzes the committed step of fatty acid synthesis by converting acetyl-CoA into **Malonyl-CoA (Compound M)**.

(b) **Malonyl-CoA** serves as the primary two-carbon donor subunit for fatty acid synthase (FAS) during elongation.

(c) Concurrently, Malonyl-CoA acts as a potent allosteric inhibitor of the **CPT-1 Shuttle System**. By blocking CPT-1, it prevents newly synthesized fatty acids from entering the mitochondria for degradation, ensuring that fatty acid synthesis proceeds without immediate oxidation.

Final Answer: Malonyl-CoA

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Lesch-Nyhan syndrome is an X-linked recessive disorder caused by a complete deficiency in hypoxanthine-guanine phosphoribosyltransferase (HGPRT), a key enzyme in the purine salvage pathway. The absence of this enzyme impairs the recycling of hypoxanthine and guanine, shunting these bases into uric acid degradation instead and triggering severe neurological dysfunction.

Solution:

Let's look at the substrate requirements of the purine salvage pathway:

(a) The missing enzyme, HGPRT, catalyzes the transfer of a ribose 5-phosphate group onto free purine bases:

$$\text{Hypoxanthine / Guanine} + \text{PRPP} \xrightarrow{\text{HGPRT}} \text{IMP / GMP} + \text{PPi}$$

(b) This reaction requires **5-phosphoribosyl-1-pyrophosphate (PRPP)** as the activated sugar complex and donor of the phosphoribosyl moiety.

(c) When HGPRT is absent, PRPP levels accumulate inside cells, allosterically accelerating de novo purine synthesis and increasing the production of toxic uric acid byproducts.

Final Answer: 5-Phosphoribosyl-1-pyrophosphate (PRPP)

Answer: (A)

Go Back to Question 9

Q10.

Solution

Concept: During prolonged exercise, de novo hepatic gluconeogenesis maintains systemic euglycemia once glycogen stores are exhausted. Gluconeogenic precursors enter the liver and are converted into oxaloacetate or dihydroxyacetone phosphate to feed the pathway, often requiring compartmental transport loops across the inner mitochondrial membrane.

Solution:

Let's evaluate the metabolic entry points of the listed precursor pairs:

(a) **Lactate and Pyruvate:** Both act as purely gluconeogenic molecules. Lactate is oxidized to pyruvate by cytosolic lactate dehydrogenase. Pyruvate must then cross into the mitochondrial matrix via the mitochondrial pyruvate carrier (MPC) to be carboxylated into oxaloacetate by the matrix enzyme pyruvate carboxylase:

$$\text{Pyruvate} + CO_2 + \text{ATP} \xrightarrow{\text{Pyruvate Carboxylase}} \text{Oxaloacetate} + \text{ADP} + \text{Pi}$$

Because oxaloacetate cannot directly cross back out, it must be converted into malate or aspartate to exit the mitochondria, utilizing a specialized transport loop to continue gluconeogenesis in the cytosol.

(b) Glycerol enters glycolysis directly in the cytosol as DHAP and bypasses mitochondrial matrix loops. Acetyl-CoA, acetoacetate, leucine, and lysine are ketogenic substrates that cannot be converted into net glucose.

Final Answer: Lactate and Pyruvate

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: Ultraviolet light causes DNA damage by inducing covalent cross-links between adjacent pyrimidine bases, forming bulky lesions like cyclobutane pyrimidine dimers. These modifications distort the double helix structure and stall transcription.

Solution:

Let's identify the repair system shown in the structural blueprint:

(a) The pathway illustrated involves recognizing helical distortions and making dual incisions to remove an oligonucleotide fragment containing the dimer. This describes **Nucleotide Excision Repair (NER)**, carried out by multi-protein complexes analogous to prokaryotic UvrABC excinucleases.

(b) An inherited, autosomal recessive mutation in genes encoding these eukaryotic repair proteins (such as XPA through XPG) prevents the repair of UV-induced lesions.

(c) This core genetic defect leads directly to **Xeroderma Pigmentosum**, a disease characterized by extreme cutaneous photosensitivity, severe sunburns, and an increased risk of developing skin malignancies.

Final Answer: Xeroderma Pigmentosum

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept: The urea cycle converts toxic free ammonia into water-soluble urea within the liver. Inherited defects in specific cycle enzymes lead to distinct accumulations of metabolic intermediates, which can be measured in plasma and urine to localize the enzymatic block.

Solution:

Let's isolate the specific enzymatic block based on the patient's metabolite profile:

(a) The infant displays severe hyperammonemia, and plasma assays show citrulline levels elevated more than 50 times the normal limit. This specific accumulation indicates that citrulline is being synthesized normally but cannot be consumed by the next step in the cycle.

(b) Citrulline is normally condensed with aspartate to form argininosuccinate via the enzyme **argininosuccinate synthetase**:

$$\text{Citrulline} + \text{Aspartate} + \text{ATP} \xrightarrow{\text{Argininosuccinate Synthetase}} \text{Argininosuccinate} + \text{AMP} + \text{PPi}$$

A functional deficiency in **argininosuccinate synthetase** causes Citrullinemia Type I, leading to an accumulation of citrulline in the blood.

(c) Defects in upstream enzymes like CPS I or OTC would prevent citrulline synthesis entirely, while a defect in argininosuccinate lyase would cause an elevation of argininosuccinate alongside milder citrulline increases.

Final Answer: Argininosuccinate synthetase

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: Southern blotting combines restriction enzyme digestion, gel electrophoresis, and complementary probe hybridization to evaluate structural changes in genomic loci, such as large insertions, deletions, or trinucleotide repeat expansions.

Solution:

Let's analyze the fragment sizes from the restriction digestion layout map:

(a) Restriction endonucleases like EcoRI cleave DNA at specific palindromic recognition sequences, cutting the genome into fragments defined by adjacent restriction sites.

(b) In the normal allele, the distance between the two flanking EcoRI restriction sites is 4.0 kb.

(c) In the mutant allele, a trinucleotide repeat expansion has occurred between these same two flanking EcoRI cleavage locations. The map specifies that the total physical distance between these boundary sites has expanded to **7.5 kb**.

(d) Because the labeled probe matches a sequence contained within this fragment, it will hybridize to the mutant strand during Southern blotting. A homozygous mutant individual will show a single distinct band at **7.5 kb**.

Final Answer: 7.5 kb

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: Propionate metabolism converts odd-chain fatty acids, branched-chain amino acids, and cholesterol side chains into a common intermediate that can enter the Citric Acid Cycle. The final step of this pathway is mediated by the mitochondrial matrix enzyme methylmalonyl-CoA mutase.

Solution:

Let's trace the enzymatic conversion step and its cofactor requirements:

(a) Methylmalonyl-CoA mutase requires **adenosylcobalamin** (an active coenzyme form of Vitamin B_{12}) to catalyze an intramolecular rearrangement.

(b) This reaction converts L-methylmalonyl-CoA directly into the Krebs cycle intermediate **succinyl-CoA**:

$$\text{L-Methylmalonyl-CoA} \xrightarrow{\text{Methylmalonyl-CoA Mutase + Adenosylcobalamin}} \text{Succinyl-CoA}$$

(c) When cells are unable to synthesize adenosylcobalamin, this reaction is blocked, leading to an accumulation of upstream methylmalonic acid in the blood and urine, causing severe metabolic ketoacidosis.

Final Answer: Succinyl-CoA

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: In eukaryotes, translation initiation utilizes a scanning mechanism where the 43S pre-initiation complex attaches to the 5′ mRNA cap and migrates downstream along the 5′ UTR to find the initial start codon.

Solution:

Let's analyze the role of the flanking consensus sequences during start site selection:

(a) Efficiency of start codon selection depends on the surrounding nucleotide context, known as the **Kozak consensus sequence** (5′-ACCDAUGG-3', where a purine at −3 and a guanine at +4 are most critical).

(b) If mutations alter these flanking sequences away from the Kozak standard, the scanning 40S ribosomal subunit often fails to recognize the first AUG codon.

(c) This leads to leaky scanning, resulting in a **reduced efficiency of translation initiation** at that site. The ribosome will continue scanning downstream and initiate translation at alternative, suboptimal downstream start sites instead, reducing production of the native protein.

Final Answer: Reduced efficiency of translation initiation with alternative start-site selection

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept: Gout is a painful arthritic condition caused by the crystallization of excess uric acid within joint spaces. Uric acid is the final terminal byproduct of the purine nucleotide degradation pathway, synthesized from hypoxanthine and xanthine.

Solution:

Let's identify the enzymatic conversions indicated as Step Z in the logic flow diagram:

(a) The enzyme **xanthine oxidase** catalyzes a two-step oxidation within the purine degradation pathway. First, it oxidizes hypoxanthine to xanthine, and second, it oxidizes xanthine into **uric acid (Step Z)**:

$$\text{Hypoxanthine} \xrightarrow{\text{Xanthine Oxidase}} \text{Xanthine} \xrightarrow{\text{Xanthine Oxidase}} \text{Uric Acid}$$

(b) **Allopurinol** is a structural purine analog that acts as a suicide inhibitor of xanthine oxidase. It is converted by the enzyme into oxypurinol, which remains tightly bound to the active site coordination complex.

(c) This direct inhibition of **xanthine oxidase** blocks Step Z, lowering plasma uric acid levels and increasing the concentrations of more soluble upstream precursors (hypoxanthine and xanthine) to manage recurrent gouty flares.

Final Answer: Xanthine oxidase

Answer: (B)

Go Back to Question 16

Answer Key

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