NEET PG Biochemistry Sample Paper-9

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

Instructions

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

Q1. A 4-month-old infant presents with profound fasting hypoglycemia, lactic acidosis, and hepatomegaly. A liver biopsy shows markedly elevated glycogen content with normal structure, but functional assays demonstrate a complete absence of the glucose-6-phosphatase catalytic subunit activity. Which of the following transport processes or metabolic shifts is most directly compromised as a primary downstream consequence in this patient's hepatocytes?

(A) Export of free glucose via GLUT2

(B) Import of galactose via GLUT5

(C) Mitochondrial export of malate

(D) Cytosolic condensation of acetyl-CoA

Q2. An experimental drug blocks the allosteric activation of Acetyl-CoA Carboxylase 1 (ACC1) by citrate while leaving its basal unactivated state untouched. Cultured hepatocytes treated with this compound are supplied with radiolabeled [1 —^{14} C]acetate. Which of the following lipid fractions will show the most immediate and significant reduction in radioactive tracer incorporation?

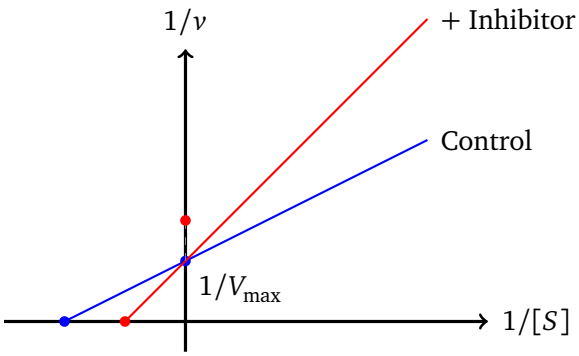
(A) Palmitate

(B) Cholesteryl esters

(C) Mevalonate

(D) Squalene

Q3. An enzyme-catalyzed reaction following classic Michaelis-Menten kinetics is evaluated in the presence of a novel synthetic inhibitor. The investigator plots the kinetic data using a Lineweaver-Burk presentation. Analyze the diagnostic double-reciprocal profile provided below:



Based on the shared or shifted intercepts displayed along the axes, identify the definitive mechanism of inhibition and its specific effect on K_m.

(A) Competitive inhibition; K_m increases

(B) Non-competitive inhibition; K_m remains unchanged

(C) Uncompetitive inhibition; K_m decreases

(D) Mixed inhibition; K_m decreases

Q4. A neonate with suspected hyperammonemia due to an inborn error of the urea cycle exhibits massively elevated levels of plasma citrulline, moderate orotic aciduria, and undetectable levels of argininosuccinate. Which of the following enzyme systems is defective in this infant?

(A) Argininosuccinate synthetase

(B) Argininosuccinate lyase

(C) Carbamoyl phosphate synthetase I

(D) Ornithine transcarbamylase

Q5. During a high-resolution prokaryotic DNA replication assay, a mutant strain of *Escherichia coli* is isolated that synthesizes Okazaki fragments normally,

but fails to join them together into a continuous lagging strand. Biochemical fractionation reveals that the mutant enzyme cannot form the essential enzyme-AMP intermediate required to activate the 5′-phosphate group of the nicked DNA. Which enzyme is mutated?

(A) DNA Ligase

(B) DNA Polymerase I

(C) DNA Polymerase III

(D) Topoisomerase I

Q6. A 14-year-old girl is evaluated for short stature, a webbed neck, and primary amenorrhea. Karyotype analysis reveals a mosaicism pattern: $45, X/46, XX$. Which of the following mechanical events occurring during early post-zygotic mitotic divisions is the most likely cellular etiology of this mosaic presentation?

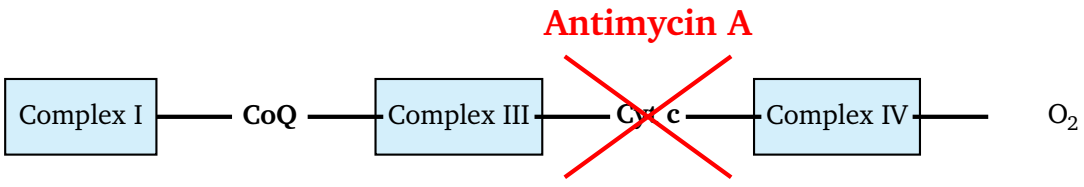
(A) Mitotic nondisjunction

(B) Meiotic anaphase lag

(C) Balanced reciprocal translocation

(D) Homologous recombination error

Q7. The terminal electron transport chain (ETC) within the inner mitochondrial membrane is susceptible to inhibition by specific toxins. The simplified pathway below illustrates electron flow through the respiratory complexes. Inhibitor X represents Antimycin A. Following exposure to a lethal dose of this inhibitor, which redox component would accumulate predominantly in the reduced state?



(A) Cytochrome *b*

(B) Cytochrome *c*

(C) Cytochrome a

(D) Cytochrome a_3

Q8. A patient presents with severe neurological deficits, symmetric dermatitis on sun-exposed skin areas, and persistent diarrhea. Urinalysis shows highly elevated levels of neutral amino acids (tryptophanuria). This clinical syndrome is identical to Pellegra because the renal and intestinal transport defect deprives the liver of the essential substrate needed for the endogenous synthesis of which vitamin coenzyme?

(A) NAD^+

(B) FAD

(C) Thiamine pyrophosphate

(D) Pyridoxal phosphate

Q9. A molecular biologist isolates a mutant eukaryotic cell line where the TATA-binding protein (TBP) exhibits an abnormally high, irreversible affinity for the minor groove of the gold-standard consensus TATA sequence box. What will be the direct operational effect of this mutation on transcription?

(A) Failure to clear the promoter site

(B) Loss of $5'$-capping efficiency

(C) Premature polyadenylation termination

(D) Accelerated elongation rate by RNA Pol II

Q10. A 5-year-old boy shows severe developmental delays, a cheerful demeanor, unprovoked paroxysms of laughter, and an ataxic wide-based gait. Genetic mapping uncovers a microdeletion limited to chromosome 15q11-q13. Further diagnostic testing confirms that this microdeletion is located strictly on the maternally inherited chromosome. Which epigenetic phenomenon explains why the paternal copy cannot rescue this phenotype?

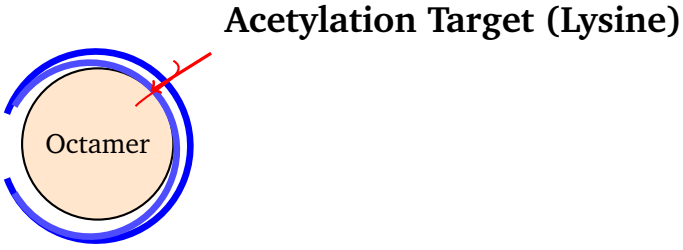
(A) Genomic imprinting

(B) X-inactivation skewing

(C) Telomeric position effect

(D) Histone acetylation silence

Q11. The structural arrangement of eukaryotic nucleosomes regulates the physical access of transcriptional machinery to genomic DNA templates. Review the conformational core assembly illustrated below:



When a pioneer transcription factor recruits a histone acetyltransferase (HAT) complex to acetylate specific lysine residues on these exposed core tails, what physical change occurs within this micro-domain?

(A) Loss of positive charge, loosening DNA binding

(B) Gain of positive charge, tightening DNA binding

(C) Loss of negative charge, inducing DNA cleavage

(D) Hyper-methylation of the adjacent cytosine bases

Q12. A 6-month-old infant of Ashkenazi Jewish descent experiences progressive psychomotor regression, macrocephaly, a prominent startle response to sound, and a bilateral "cherry-red spot" observed on fundoscopic examination. Hepatosplenomegaly is notably absent. Amniotic cell screening reveals a severe deficiency of the Hexosaminidase A enzyme. Which specific lipid intermediate accumulates inside the lysosomes of this patient's neurons?

(A) GM_2 ganglioside

(B) Glucocerebroside

(C) Sphingomyelin

(D) Galactocerebroside

Q13. A 3-year-old boy presents with severe self-mutilating behaviors, including lip and finger biting, gouty arthritis, and cognitive impairment. Laboratory investigations indicate hyperuricemia and an elevated urinary uric acid-to-creatinine ratio. This condition is caused by a complete loss-of-function mutation in an enzyme of the purine salvage pathway. What is the immediate metabolic consequence of this enzyme's deficiency on the *de novo* purine synthesis pathway?

(A) Accelerated PRPP amidotransferase activity

(B) Feedback inhibition of PRPP synthetase

(C) Accumulation of adenosine deaminase intermediates

(D) Reduced availability of ribose-5-phosphate

Q14. A chronic alcoholic presents with confusion, ophthalmoplegia, and an unstable, ataxic gait. The emergency department physician suspects Wernicke encephalopathy and orders an intravenous infusion. Which of the following parameters should be measured or addressed before administering glucose to avoid worsening the neurological decline?

(A) Erythrocyte transketolase activity

(B) Serum methylmalonic acid level

(C) Plasma homocysteine concentration

(D) Leoflorin cleavage rate

Q15. An in vitro translation system is constructed using a synthetic polyribonucleotide template containing alternating uracil and guanine residues (5′-UGUGUGUGU
Assuming translation initiates randomly without a fixed start codon, which of the following peptide products will be synthesized by this experimental cell-free system?

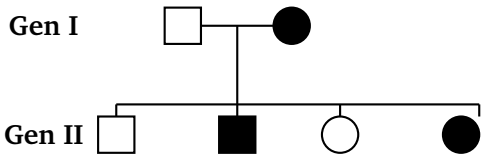
(A) A homopolymer of Cysteine

(B) A homopolymer of Valine

(C) A heteropolymer of Cysteine and Valine

(D) A heteropolymer of Leucine and Glycine

Q16. Pedigree charts track hereditary transmission modes across multiple clinical generations. Analyze the multi-generation disease distribution layout mapped below:



Given that affected fathers pass this condition to all of their daughters but none of their sons, what is the definitive mode of inheritance demonstrated here?

(A) Autosomal recessive

(B) X-linked dominant

(C) X-linked recessive

(D) Mitochondrial maternal

Detailed Solutions

Q1.

Solution

Concept: Glucose-6-phosphatase catalyzes the final shared chemical step of both hepatic glycogenolysis and gluconeogenesis, hydrolyzing glucose-6-phosphate into free glucose and inorganic phosphate within the lumen of the endoplasmic reticulum.

Solution:

Let's trace the biochemical adjustments and transport logic in these hepatocytes:

(a) The absence of catalytic glucose-6-phosphatase activity leaves the hepatocyte unable to generate free, dephosphorylated glucose from its inner metabolic pathways during periods of fasting.

(b) Under normal conditions, the generated internal free glucose would exit the hepatocyte down its concentration gradient into the bloodstream via the high-capacity, bidirectional glucose transporter 2 (GLUT2).

(c) Therefore, the primary, direct downstream consequence of losing this enzyme is the absolute compromise of the **Export of free glucose via GLUT2**.

(d) Galactose enters cells through GLUT2, while GLUT5 is a specialized fructose transporter. Mitochondrial export of malate and cytosolic acetyl-CoA processing are upstream or collateral pathways that are not the direct mechanical exit step blocked here.

Final Answer: Export of free glucose via GLUT2

Answer: (A)

Go Back to Question 1

Q2.

Solution

Concept: Acetyl-CoA Carboxylase (ACC) is the rate-limiting and committed enzyme of de novo fatty acid synthesis, converting acetyl-CoA into malonyl-CoA. Citrate acts as a critical forward allosteric activator of ACC, inducing its polymerization from an inactive monomeric state into its highly active polymeric filament structure.

Solution:

Let's map out the metabolic downstream fates of the radiolabeled acetate tracer:

(a) Cells process acetate into acetyl-CoA. Acetyl-CoA sits at a fork in lipid architecture: it can either enter the de novo fatty acid synthesis pathway or the cytosolic mevalonate pathway for cholesterogenesis.

(b) By introducing a drug that completely prevents the allosteric activation of ACC1 by citrate, the production of malonyl-CoA is sharply diminished.

(c) Since malonyl-CoA is the obligate substrate used by Fatty Acid Synthase to build long-chain saturated fatty acids, this block will cause an immediate and profound reduction in the synthesis of **Palmitate** ($C_{16:0}$).

(d) Because the acetyl-CoA pool is no longer being drawn into fatty acid creation, it becomes increasingly available to enter the alternative pathway, preserving or even increasing the synthesis of cholesterol precursors like mevalonate, squalene, and cholesteryl esters.

Final Answer: Palmitate

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: Lineweaver-Burk double-reciprocal transformations help differentiate enzyme inhibition mechanisms. The vertical y-intercept represents $1/V_{\text{max}}$, the horizontal x-intercept represents $-1/K_m$, and the slope is equal to K_m/V_{max}.

Solution:

Let's read the visual data from the provided graph:

(a) Both the uninhibited Control line (blue) and the Inhibitor line (red) intersect at the exact same position on the negative horizontal axis. This means the value of $-1/K_m$ is identical for both conditions, proving that **K_m remains completely unchanged**.

(b) In contrast, the vertical intercept ($1/v$) shifts upward in the presence of the inhibitor, demonstrating that $1/V_{\text{max}}$ has increased, which means the maximum operational velocity (V_{max}) has decreased.

(c) An unchanged K_m value combined with a decreased V_{max} value is the absolute signature of **Non-competitive inhibition**. In this mechanism, the inhibitor binds with equal affinity to both the free enzyme and the enzyme-substrate complex at an allosteric site, decreasing catalytic output without altering substrate binding.

Final Answer: Non-competitive inhibition; K_m remains unchanged

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept: The urea cycle converts neurotoxic ammonia into excretable urea. Inherited blocks at different steps of this cycle result in distinct patterns of biochemical intermediate accumulation in the plasma and urine.

Solution:

Let's analyze the biochemical clues to locate the exact enzymatic lesion:

(a) The neonate displays massive plasma citrulline accumulation combined with an absolute absence of downstream argininosuccinate. This indicates that citrulline is successfully formed but cannot be condensed with aspartate to advance through the cycle.

(b) This specific transformation step is catalyzed by the cytosolic enzyme **Argininosuccinate synthetase**:

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

A total failure of this step leads directly to Citrullinemia Type I.

(c) The accumulated upstream carbamoyl phosphate leaks from the mitochondria into the cytosol, where it enters the pyrimidine synthesis pathway, leading to the observed moderate orotic aciduria. A defect in argininosuccinate lyase would instead cause elevated levels of argininosuccinate.

Final Answer: Argininosuccinate synthetase

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: DNA replication requires the coordination of several enzymes to manage both the leading and lagging strands. During lagging strand replication, discontinuous synthesis yields short Okazaki fragments that must eventually be covalently sealed together to ensure genomic continuity.

Solution:

Let's look closely at the chemical activation mechanism described:

(a) The isolated mutant strain synthesizes Okazaki fragments and removes RNA primers normally, but fails to complete the final phosphodiester bond ligation step.

(b) Mechanistically, prokaryotic **DNA Ligase** utilizes NAD^+ as an energy source to drive this reaction. The enzyme must first react with NAD^+ to covalently attach an AMP molecule to a conserved active-site lysine residue, forming an essential enzyme-AMP intermediate:

$$\text{Enzyme} + NAD^+ \rightarrow \text{Enzyme-AMP} + \text{NMN}$$

(c) This activated AMP group is then transferred to the $5'$-phosphate group at the nick site, preparing it for nucleophilic attack by the adjacent $3'$-hydroxyl group.

(d) Because the mutant enzyme cannot form this **enzyme-AMP intermediate**, the activation step fails, identifying **DNA Ligase** as the defective enzyme.

Final Answer: DNA Ligase

Answer: (A)

Go Back to Question 5

Q6.

Solution

Concept: Chromosomal mosaicism refers to the presence of two or more genetically distinct cell lines within a single individual. This develops when a genetic error occurs during mitotic cell divisions in the embryo after fertilization.

Solution:

Let's analyze the cellular mechanisms causing a mosaic Turner syndrome pattern $(45, X/46, XX)$:

(a) The zygote begins as a normal diploid cell with a $46, XX$ karyotype following standard fertilization.

(b) During subsequent early post-zygotic cleavage divisions, an error in chromosome segregation occurs during mitosis. This event is called **mitotic nondisjunction** (or anaphase lag during mitosis).

(c) When a sister chromatid fails to separate correctly to opposite poles during anaphase, one daughter cell inherits an extra chromosome $(47, XXX)$, while the other daughter cell loses a sex chromosome, resulting in a monosomic cell line $(45, X)$.

(d) The $47, XXX$ cell line often fails to survive or proliferate, leaving a mixture of the original normal $46, XX$ cells and the newly formed $45, X$ cells to populate the developing embryonic tissues. Meiotic errors occur before fertilization and would result in non-mosaic configurations throughout all somatic cells.

Final Answer: Mitotic nondisjunction

Answer: (A)

Go Back to Question 6

Q7.

Solution

Concept: The mitochondrial electron transport chain couples sequential redox reactions to pump protons across the inner mitochondrial membrane. Inhibiting a specific respiratory complex halts downstream electron flow while leaving upstream carriers fully reduced.

Solution:

Let's analyze the flow of electrons around the site blocked by Inhibitor X:

(a) The pathway diagram shows that **Inhibitor X (Antimycin A)** binds and blocks electron transfer through **Complex III** (Coenzyme Q : Cytochrome c Oxidoreductase).

(b) Under normal conditions, electrons from reduced Coenzyme Q ($CoQH_2$) pass through cytochrome b within Complex III, then move to cytochrome c_1, and finally to the mobile carrier cytochrome c.

(c) When Antimycin A completely blocks Complex III, electrons can still enter the chain from upstream substrates and pass through **Cytochrome b**, but they are blocked from moving further downstream.

(d) As a result, carriers located before the block, such as **Cytochrome b**, become trapped in their fully reduced states. Conversely, downstream components like Cytochrome c, Cytochrome a, and Cytochrome a_3 lose their electron supply and become fully oxidized.

Final Answer: Cytochrome b

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Hartnup disease is an autosomal recessive disorder caused by a defective neutral amino acid transporter (SLC6A19) in the apical membranes of proximal renal tubule cells and intestinal mucosal enterocytes. This impairs the absorption and retention of neutral amino acids, particularly tryptophan.

Solution:

Let's connect this transport defect to the biochemical synthesis of nicotinamide cofactors:

(a) The patient presents with pellagra-like symptoms: symmetric dermatitis, diarrhea, and severe neurological issues, along with significant neutral amino acid excretion (tryptophanuria).

(b) Under normal physiological conditions, the liver uses absorbed dietary tryptophan to synthesize nicotinic acid and its active nucleotide derivatives via the kynurenine pathway.

(c) Tryptophan acts as an organic precursor that supplies roughly half of the body's daily requirement for **NAD^+** and $NADP^+$ synthesis.

(d) Because Hartnup disease causes severe systemic wasting of tryptophan, the liver cannot produce sufficient quantities of **NAD^+**, leading to a functional niacin (Vitamin B_3) deficiency and classic pellagra symptoms.

Final Answer: NAD^+

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Eukaryotic transcription initiation requires general transcription factors to assemble sequentially at the core promoter to recruit RNA Polymerase II, open the DNA double helix, and transition from initiation to active elongation.

Solution:

Let's analyze the effect of an irreversible TBP-promoter binding dynamic:

(a) The TATA-binding protein (TBP) subunit of TFIID initiates assembly by binding to the minor groove of the TATA box, bending the DNA backbone to recruit subsequent factors like TFIIB and RNA Polymerase II.

(b) After the pre-initiation complex forms and synthesizes the initial short transcripts, the transcription complex must undergo promoter clearance. This transition requires the polymerase to break its structural ties with the promoter-bound general transcription factors so it can escape into productive elongation.

(c) If TBP exhibits an abnormally high, irreversible affinity for the TATA box sequence, the transcription complex cannot release these structural holds. This leads to a **Failure to clear the promoter site**, trapping the enzyme assembly at the initiation locus and blocking efficient transcription.

Final Answer: Failure to clear the promoter site

Answer: (A)

Go Back to Question 9

Q10.

Solution

Concept: Angelman syndrome is a neurodevelopmental disorder caused by a loss of function within the maternal copy of the *UBE3A* gene located on chromosome 15q11-q13. This gene is regulated by an epigenetic mechanism that silences alleles based on their parent of origin.

Solution:

Let's analyze how epigenetic modifications regulate expression at this locus:

(a) The patient's clinical signs—developmental delays, an ataxic wide-based gait, a cheerful demeanor, and unprovoked laughter—identify Angelman syndrome. This condition is caused here by a microdeletion on the maternally inherited chromosome 15q11-q13.

(b) This specific locus is controlled by **Genomic imprinting**, an epigenetic process where differential DNA methylation during gametogenesis selectively silences specific alleles.

(c) Within neurons of the central nervous system, the paternal copy of the *UBE3A* gene is naturally imprinted and transcriptionally silenced by a non-coding antisense transcript.

(d) Because the paternal allele is permanently silenced by **genomic imprinting**, it cannot be expressed to rescue the phenotype when the maternal copy is lost, leading to the clinical features of Angelman syndrome.

Final Answer: Genomic imprinting

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: Nucleosomes consist of genomic DNA wrapped around a core octamer of histone proteins (two copies each of H2A, H2B, H3, and H4). The tight interaction between DNA and histones is maintained by electrostatic attraction between the negatively charged phosphodiester backbone of DNA and the positively charged amino acid tails of the histone proteins.

Solution:

Let's evaluate the structural change that occurs when histone acetyltransferases (HATs) modify these tails:

(a) Histone acetyltransferases (HATs) transfer an acetyl group from acetyl-CoA onto the positively charged ϵ-amino groups of specific lysine residues located on the exposed histone tails.

(b) This covalent modification neutralizes the positive charge on the lysine residues, leading to a **Loss of positive charge, loosening DNA binding**.

(c) This charge neutralization disrupts the electrostatic attraction between the histones and the DNA backbone. As a result, the chromatin structure relaxes from a tightly condensed conformation (heterochromatin) into an open, accessible state (euchromatin), allowing transcription factors and RNA polymerase II to bind the DNA template.

Final Answer: Loss of positive charge, loosening DNA binding

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept: Tay-Sachs disease is an autosomal recessive lysosomal storage disease caused by mutations in the *HEXA* gene, which encodes the α-subunit of the enzyme β-hexosaminidase A. This enzyme is responsible for degrading specific sphingolipids within lysosomes.

Solution:

Let's identify the lipid intermediate that accumulates due to this enzymatic block:

(a) The infant displays psychomotor regression, macrocephaly, an exaggerated startle response, and a cherry-red spots on the macula without hepatosplenomegaly. This clinical presentation points to Tay-Sachs disease.

(b) Under normal conditions, functional hexosaminidase A cleaves the terminal N-acetylgalactosamine residue from complex gangliosides.

(c) A severe deficiency of this enzyme blocks this degradation step, causing **GM_2 ganglioside** to accumulate within the lysosomes of neurons:

$$GM_2 \text{ Ganglioside} \xrightarrow{\text{Hexosaminidase A}} GM_3 \text{ Ganglioside}$$

(d) This accumulation leads to progressive lysosomal distension, cellular toxicity, and widespread neuronal apoptosis. Glucocerebroside accumulates in Gaucher disease, sphingomyelin accumulates in Niemann-Pick disease, and galactocerebroside accumulates in Krabbe disease.

Final Answer: GM_2 ganglioside

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: Lesch-Nyhan syndrome is caused by an X-linked recessive, complete deficiency of hypoxanthine-guanine phosphoribosyltransferase (HGPRT), a key enzyme in the purine salvage pathway. This pathway recycles free purine bases back into nucleotide monophosphates, coordinating with the de novo purine synthesis pathway to regulate nucleotide pools.

Solution:

Let's trace the metabolic consequences of an HGPRT deficiency on the de novo synthesis pathway:

(a) HGPRT normally converts hypoxanthine and guanine into IMP and GMP using 5-phosphoribosyl-1-pyrophosphate (PRPP) as a ribose-5-phosphate donor.

(b) When HGPRT is entirely absent, free purine bases cannot be salvaged, causing intracellular concentrations of PRPP to increase significantly because it is not being consumed by the salvage pathway.

(c) Concurrently, intracellular levels of IMP and GMP fall, removing normal feedback inhibition on the rate-limiting enzyme of de novo synthesis, PRPP amidotransferase.

(d) The combination of elevated substrate levels (PRPP) and reduced feedback inhibition leads to **Accelerated PRPP amidotransferase activity**, significantly increasing de novo purine synthesis and generating excess uric acid as a degradation byproduct.

Final Answer: Accelerated PRPP amidotransferase activity

Answer: (A)

Go Back to Question 13

Q14.

Solution

Concept: Wernicke encephalopathy is an acute, life-threatening neurological condition caused by a severe deficiency of thiamine (Vitamin B_1), frequently seen in individuals with chronic alcohol use disorder. Thiamine pyrophosphate (TPP) is an essential cofactor for rate-limiting enzymes in glucose metabolism.

Solution:

Let's analyze the physiological impact of administering glucose before addressing thiamine status:

(a) Thiamine is a required cofactor for pyruvate dehydrogenase (PDH), α-ketoglutarate dehydrogenase (α-KGDH), and transketolase. These enzymes are essential for aerobic glucose oxidation and ATP generation via the Citric Acid Cycle.

(b) Infusing intravenous glucose provides a large carbohydrate load that accelerates glycolysis, rapidly consuming the body's remaining scarce stores of thiamine.

(c) This sudden depletion of thiamine stalls the PDH complex, preventing pyruvate from entering the Citric Acid Cycle and causing severe energy failure in vulnerable brain regions like the mammillary bodies.

(d) To avoid worsening this neurological decline, clinicians can assess baseline thiamine status by measuring **Erythrocyte transketolase activity** (which increases upon adding TPP), though empirical thiamine supplementation is given immediately before or alongside glucose in clinical practice.

Final Answer: Erythrocyte transketolase activity

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: The genetic code is read as continuous, non-overlapping triplets of nucleotides called codons. In an in vitro translation system lacking a fixed start codon, translation can initiate at random positions along the synthetic template, defining the reading frame based on the starting nucleotide.

Solution:

Let's analyze the reading frames generated by the alternating sequence template (5′-UGUGUGUGUGUG...-3′):

(a) Because translation can begin at any position, let's look at the possible triplet codons formed by this alternating template:

- Frame 1 begins at a 'U' residue, yielding repeating sequences of: 5′-UGU-GUG-UGU-GUG...-3′.
- Frame 2 begins at a 'G' residue, yielding repeating sequences of: 5′-GUG-UGU-GUG-UGU...-3′.

(b) According to the standard genetic code table, the codon 5′-UGU-3' codes for the amino acid **Cysteine (Cys)**, and the codon 5′-GUG-3' codes for the amino acid **Valine (Val)**.

(c) Regardless of which frame is selected, the ribosome will read alternating 5′-UGU-3' and 5′-GUG-3' codons along the continuous strand. This direct reading produces a single repeating peptide chain consisting of alternating Cysteine and Valine residues, forming a **heteropolymer of Cysteine and Valine**.

Final Answer: A heteropolymer of Cysteine and Valine

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept: Modes of inheritance describe how genetic traits are transmitted through generations. X-linked dominant inheritance is characterized by the presence of the mutant allele on the X chromosome, where a single copy is sufficient to cause the phenotype in both males and females.

Solution:

Let's evaluate the transmission rules shown in the pedigree and question text:

(a) A father passes his Y chromosome to all of his sons and his X chromosome to all of his daughters.

(b) The question states that affected fathers pass this condition to **all of their daughters but none of their sons**. This confirms that the trait is linked to the X chromosome, ruling out autosomal modes of inheritance.

(c) Because an affected father transmits his single, mutant X chromosome to every daughter, and each of those daughters develops the phenotype, a single copy of the allele is sufficient to express the trait. This pattern defines an **X-linked dominant** mode of inheritance.

(d) X-linked recessive inheritance is incorrect because female carriers typically do not express the phenotype unless they are homozygous or display skewed X-inactivation.

Final Answer: X-linked dominant

Answer: (B)

Go Back to Question 16

Answer Key

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