NEET PG Biochemistry Sample Paper-4

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 9-month-old infant is brought to the metabolic clinic due to persistent hepatomegaly, severe fasting hypoglycemia, and profound lactic acidosis. A liver biopsy reveals marked accumulation of glycogen with normal structured outer branches, alongside significant lipid droplets. Administration of epinephrine or glucagon fails to induce an increase in blood glucose levels but causes a sharp rise in blood lactate. Which of the following regulatory enzymes is most likely deficient in this patient?

(A) Glycogen phosphorylase

(B) Glucose-6-phosphatase

(C) Alpha-1,4-glucosidase

(D) Phosphofructokinase-1

Q2. A 23-year-old male is undergoing an experimental prolonged starvation protocol under strict medical observation. By day 4 of complete caloric deprivation, his brain has adapted to utilize ketone bodies for a significant portion of its energy requirements. Which of the following mitochondrial enzymes, uniquely absent in hepatocytes, is mandatory for the activation and oxidation of acetoacetate in extrahepatic tissues like the cerebral cortex?

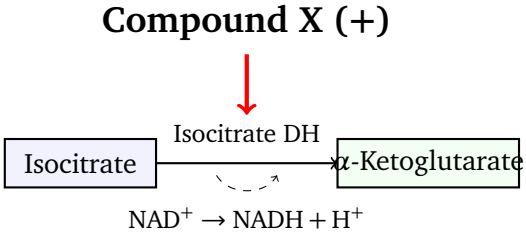
(A) Thiolase

(B) β-hydroxybutyrate dehydrogenase

(C) Succinyl-CoA-acetoacetate-CoA transferase

(D) HMG-CoA synthase

Q3. A laboratory scientist isolates hepatic mitochondria to measure metabolic flux through the Citric Acid Cycle under varying ATP/ADP ratios. Analyze the key multi-step regulatory checkpoint illustrated below. Identify the molecule or state labeled **Compound X** that acts as a potent allosteric activator to drive this critical rate-limiting oxidative decarboxylation reaction forward:



(A) ATP

(B) NADH

(C) ADP

(D) Succinyl-CoA

Q4. A 3-year-old boy presents with cognitive impairment, developmental delay, and a history of recurrent seizures. Physical exam reveals lens subluxation and marfanoid habitus. Plasma amino acid analysis displays significantly elevated levels of methionine and homocystine. The physician suspects a deficiency in cystathionine β-synthase. To optimize therapeutic intervention, which co-factor supplementation should be attempted first to salvage remaining enzyme activity?

(A) Thiamine pyrophosphate (B_1)

(B) Pyridoxal phosphate (B_6)

(C) Cobalamin (B_{12})

(D) Riboflavin (B_2)

Q5. A 34-year-old female experiences severe muscle cramping and myoglobinuria exclusively following high-intensity, short-duration sprinting exercises. Her aerobic endurance performance remains completely normal. A muscle biopsy shows normal glycogen structure but completely lacks the ability to break down glycogen down to glucose-1-phosphate during anaerobic work. Which metabolic pathway is disrupted in this patient?

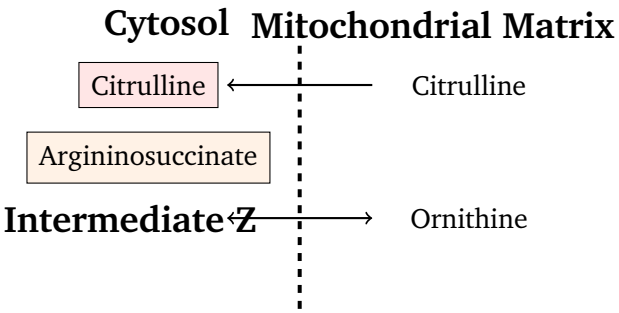
(A) Hepatic glycogenolysis (Von Gierke)

(B) Muscle glycogenolysis (McArdle)

(C) Lysosomal glycogen degradation (Pompe)

(D) Fatty acid elongation pathways

Q6. The biochemical layout of the urea cycle relies on precise spatial segregation between the mitochondrial matrix and the cytosol. Observe the biochemical transport scheme illustrated below. Identify the structural metabolic intermediate labeled **Intermediate Z** that must cross the inner mitochondrial membrane via a specific anti-port carrier system to combine with carbamoyl phosphate in the matrix:



(A) Fumarate

(B) Ornithine

(C) Arginine

(D) Aspartate

Q7. A research group isolates a mutant strain of *Escherichia coli* that synthesizes abnormally short DNA fragments containing ribonucleotide primers during replication of the lagging strand. The fragments fail to seal smoothly into a

continuous double helix. Which specific enzyme activity of DNA Polymerase I is most likely defective or missing in this mutated prokaryotic strain?

(A) $5' \rightarrow 3'$ polymerase activity

(B) $3' \rightarrow 5'$ exonuclease activity

(C) $5' \rightarrow 3'$ exonuclease activity

(D) $3' \rightarrow 5'$ polymerase activity

Q8. An oncology profile explores the mechanism of action of alpha-amanitin, a toxic cyclic peptide found in the *Amanita phalloides* death cap mushroom. If a patient accidentally ingests this toxin, which specific eukaryotic cellular process is completely blocked due to the highly selective inhibition of RNA Polymerase II?

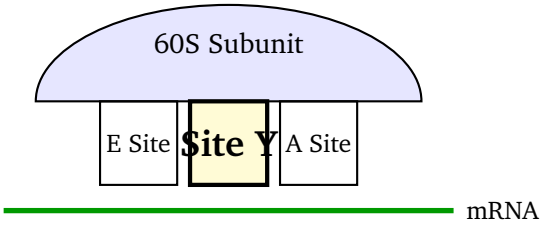
(A) Synthesis of mature 28S and 18S ribosomal RNA

(B) Synthesis of messenger RNA (mRNA) precursors

(C) Synthesis of transfer RNA (tRNA) and 5S rRNA

(D) Synthesis of short mitochondrial RNA primers

Q9. A molecular biologist designs an in-vitro assay to track the assembly steps of eukaryotic translation initiation complexes. Examine the functional step diagram provided below. Identify the exact ribosomal structural site marked **Site Y** where the initiator tRNA (Met-tRNA$_i^{Met}$) binds directly during the formation of the finalized 80S initiation complex before elongation cycles begin:



(A) Aminoacyl (A) Site

(B) Peptidyl (P) Site

(C) Exit (E) Site

(D) Shine-Dalgarno Core Site

Q10. A 42-year-old male with xeroderma pigmentosum presents with multiple aggressive cutaneous squamous cell carcinomas on his face and forearms. The underlying molecular pathology of this autosomal recessive disorder is a severe impairment in which specific DNA repair pathway responsible for removing bulky pyrimidine dimers caused by UV exposure?

(A) Base Excision Repair (BER)

(B) Mismatch Repair (MMR)

(C) Nucleotide Excision Repair (NER)

(D) Non-Homologous End Joining (NHEJ)

Q11. An experimental enzyme-catalyzed reaction is conducted in the presence of an unknown pharmacological inhibitor. Lineweaver-Burk plot analysis reveals that the lines representing the inhibited and uninhibited reactions intersect exactly on the vertical y-axis ($1/V_0$). Which statement correctly describes the kinetic parameter changes (K_m and V_{max}) and the specific type of inhibition occurring?

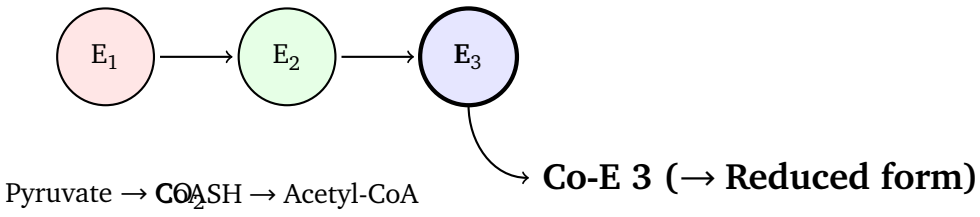
(A) K_m increases, V_{max} decreases; Non-competitive inhibition

(B) K_m increases, V_{max} remains unchanged; Competitive inhibition

(C) K_m remains unchanged, V_{max} decreases; Pure uncompetitive inhibition

(D) K_m decreases, V_{max} decreases; Mixed inhibition

Q12. A systemic biochemistry panel evaluates metabolic blocks within the pyruvate dehydrogenase (PDH) multienzyme complex. Study the precise cyclical cascade of the three catalytic subunits (E_1, E_2, and E_3) outlined below. Identify the vitamin-derived structural co-enzyme marked **Co-E 3** that acts as the final electron acceptor within the E_3 (dihydrolipooyl dehydrogenase) domain to regenerate oxidized lipoamide:



(A) Thiamine Pyrophosphate (TPP)

(B) Flavin Adenine Dinucleotide (FAD)

(C) Pyridoxal Phosphate (PLP)

(D) Biocytin

Q13. A 65-year-old chronic alcoholic male presents with severe ataxia, global confusion, and horizontal nystagmus (Wernicke-Korsakoff syndrome). Red blood cell transketolase activity is measured and found to be profoundly low, but it normalizes instantly upon addition of an exogenous vitamin cofactor. This vital vitamin acts as a crucial coenzyme for which of the following vital metabolic enzymes?

(A) Malate dehydrogenase

(B) Pyruvate carboxylase

(C) α-Ketoglutarate dehydrogenase

(D) Isocitrate dehydrogenase

Q14. A 14-year-old girl is evaluated for short stature, a wide webbed neck, a low posterior hairline, and primary amenorrhea. Cytogenetic analysis reveals a mosaic karyotype containing $45, X/46, XX$ cellular populations. Which of the following classic meiotic or mitotic phenomena is the primary mechanical cause underlying this mosaic clinical presentation?

(A) Meiotic non-disjunction during maternal oogenesis

(B) Mitotic non-disjunction or chromosome lag during early embryonic cleavage

(C) Reciprocal balanced translocation during paternal gametogenesis

(D) Robertsonian translocation involving acrocentric chromosomes

Q15. Pedigree analysis of a multi-generational family tracking a rare neuromuscular degenerative disease reveals that an affected female transmits the clinical phenotype to all of her biological children (both sons and daughters) with absolute penetrance. However, affected males never pass the condition to any of their offspring. This strict inheritance pattern represents which genetic phenomenon?

(A) X-linked dominant inheritance with male lethality

(B) Mitochondrial (maternal) inheritance

(C) Autosomal dominant inheritance with variable expressivity

(D) Genomic imprinting with paternal activation

Q16. A newborn infant with profound hypotonia, feeding difficulties, and a weak, high-pitched mewing cry is diagnosed with Cri-du-chat syndrome. High-resolution chromosomal micro-array analysis confirms a structural aberration. This genetic disorder is caused by which specific structural abnormality of chromosome 5?

(A) Deletion of the short arm (5p)

(B) Duplication of the long arm (5q)

(C) Pericentric inversion

(D) Isochromosome formation

Detailed Solutions

Q1.

Solution

Concept: Glycogen storage diseases (GSD) are caused by defects in enzymes that regulate glycogen synthesis or breakdown. Von Gierke disease (GSD type I) stems from a deficiency in glucose-6-phosphatase, the enzyme responsible for removing the phosphate group from glucose-6-phosphate to release free glucose into the blood during glycogenolysis and gluconeogenesis.

Solution:

Let's analyze the clinical presentation and biochemical markers described:

(a) The infant presents with hepatomegaly, fasting hypoglycemia, and lactic acidosis. The liver biopsy reveals an abundance of structurally normal glycogen (normal outer branches), ruling out debranching or branching enzyme deficiencies.

(b) Administering epinephrine or glucagon fails to raise blood glucose levels, confirming that glycogenolysis is blocked at its final common step.

(c) Instead of glucose, hormonal stimulation causes a sharp rise in blood lactate. This occurs because the accumulated glucose-6-phosphate cannot leave the hepatocyte; it is shunted into glycolysis, converting into pyruvate and then excess lactic acid. This clinical constellation identifies a missing **glucose-6-phosphatase** enzyme.

Final Answer: Glucose-6-phosphatase

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept: Ketone bodies (acetoacetate and β-hydroxybutyrate) are synthesized in the liver during prolonged fasting or starvation but cannot be utilized there. For extrahepatic tissues to harvest energy from acetoacetate, it must be converted back into acetyl-CoA via a specific mitochondrial activation step.

Solution:

Let's trace the enzymatic pathway required for ketone body utilization:

(a) Acetoacetate is activated in extrahepatic mitochondria by receiving a coenzyme A group from succinyl-CoA.

(b) This critical transfer is catalyzed by the enzyme **succinyl-CoA-acetoacetate-CoA transferase** (also known as thiophorase):

$$\text{Acetoacetate} + \text{Succinyl-CoA} \rightarrow \text{Acetoacetyl-CoA} + \text{Succinate}$$

(c) Thiophorase is uniquely absent in hepatocytes, which prevents the liver from consuming the ketone bodies it synthesizes, ensuring they are preserved for peripheral tissues like the brain and skeletal muscle.

Final Answer: Succinyl-CoA-acetoacetate-CoA transferase

Answer: (C)

Go Back to Question 2

Q3.

Solution

Concept: Isocitrate dehydrogenase catalyzes the oxidative decarboxylation of isocitrate to α-ketoglutarate within the mitochondrial matrix. This step serves as a major rate-limiting checkpoint of the Citric Acid Cycle and is tightly regulated by the energetic status of the cell.

Solution:

Let's analyze the allosteric regulators acting on the isocitrate dehydrogenase checkpoint:

(a) High cellular energy indicators, such as ATP and NADH, reflect a fully charged state and act as negative allosteric inhibitors to slow down the cycle.

(b) Conversely, a low energy state is signaled by an accumulation of **ADP (Compound X)**. ADP binds to the enzyme, increasing its affinity for isocitrate and acting as a potent **allosteric activator** (+) to accelerate the production of reducing equivalents ($NADH + H^+$) for the electron transport chain.

Final Answer: ADP

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept: Classical homocystinuria is an autosomal recessive metabolic disorder most commonly caused by a genetic deficiency in cystathionine β-synthase (CBS). This enzyme converts homocysteine and serine into cystathionine along the methionine metabolic pathway.

Solution:

Let's determine the co-factor requirement for this structural pathway:

(a) The enzyme cystathionine β-synthase requires **pyridoxal phosphate (PLP, Vitamin B_6)** as an essential catalytic co-factor.

(b) In many patients, the mutation results in a reduced binding affinity between the apoenzyme and its co-factor rather than a complete loss of protein expression.

(c) Administering high doses of **vitamin B_6 (pyridoxal phosphate)** can saturate the mutant enzyme, stabilizing its tertiary structure, saving residual catalytic activity, and significantly reducing toxic plasma homocysteine levels.

Final Answer: Pyridoxal phosphate (B_6)

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept: Glycogen storage disease type V (McArdle disease) is caused by an inherited deficiency of muscle glycogen phosphorylase (myophosphorylase). This enzyme breaks down internal glycogen stores into glucose-1-phosphate to supply energy during muscle contraction.

Solution:

Let's evaluate the tissue-specific metabolic disruption presented:

(a) The patient experiences severe muscle cramping and myoglobinuria exclusively during high-intensity, short-duration exercise (anaerobic glycolysis), while aerobic endurance is normal.

(b) The muscle biopsy reveals that glycogen cannot be cleaved into glucose-1-phosphate, a step catalyzed exclusively by glycogen phosphorylase.

(c) Because hepatic glycogenolysis functions normally (blood glucose levels are stable), this defect is limited to the skeletal muscle isoform of the enzyme. This presentation matches **muscle glycogenolysis (McArdle disease)**.

Final Answer: Muscle glycogenolysis (McArdle)

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: The reactions of the urea cycle are compartmentalized between the mitochondrial matrix and the cytosol. This spatial segregation requires specialized carrier transport proteins to move intermediates back and forth across the inner mitochondrial membrane.

Solution:

Let's track the molecular cycle across the compartmental borders:

(a) Inside the mitochondrial matrix, carbamoyl phosphate combines with ornithine to produce citrulline. Citrulline is then transported out of the matrix into the cytosol.

(b) In the cytosol, citrulline is converted into argininosuccinate, then arginine, and finally cleaved to yield urea and regenerate **ornithine (Intermediate Z)**.

(c) To complete the loop, this newly regenerated cytosolic **ornithine** must cross back into the inner mitochondrial membrane via an ornithine/citrulline antiporter to serve as the substrate for the next round of synthesis.

Final Answer: Ornithine

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: Lagging strand synthesis during DNA replication occurs discontinuously, producing short segments known as Okazaki fragments. Each fragment begins with a temporary RNA primer that must be excised and replaced with DNA nucleotides before DNA ligase can seal the backbone.

Solution:

Let's examine the multi-functional catalytic properties of prokaryotic DNA Polymerase I:

(a) Prokaryotic DNA Polymerase I possesses three distinct catalytic activities: a $5' \to 3'$ polymerase, a $3' \to 5'$ proofreading exonuclease, and a unique $5' \to 3'$ exonuclease.

(b) The **$5' \to 3'$ exonuclease activity** is uniquely responsible for identifying, nicking, and removing the ribonucleotide RNA primers from the $5'$ ends of Okazaki fragments.

(c) If this specific $5' \to 3'$ exonuclease function is defective, the RNA primers remain attached to the DNA segments, preventing proper gap filling and blocking DNA ligase from creating a continuous double helix.

Final Answer: $5' \to 3'$ exonuclease activity

Answer: (C)

Go Back to Question 7

Q8.

Solution

Concept: Eukaryotic transcription utilizes three distinct nuclear RNA polymerases, each dedicated to synthesizing specific classes of cellular RNA. Alpha-amanitin acts as a potent inhibitor by blocking the structural translocation mechanism of specific polymerases.

Solution:

Let's distinguish between the transcription profiles of the three major eukaryotic RNA polymerases:

(a) **RNA Polymerase I:** Synthesizes the major ribosomal RNA precursors (28S, 18S, and 5.8S rRNA) in the nucleolus; it is insensitive to alpha-amanitin.

(b) **RNA Polymerase II:** Synthesizes **messenger RNA (mRNA) precursors** (hnRNA) and most small nuclear RNAs (snRNA). It is **highly sensitive to inhibition by alpha-amanitin**.

(c) **RNA Polymerase III:** Synthesizes transfer RNAs (tRNA) and the small 5S rRNA subunit; it is sensitive only at high toxin concentrations.

Ingesting alpha-amanitin arrests the synthesis of new mRNA precursors, halting global cellular protein translation and causing severe hepatotoxicity.

Final Answer: Synthesis of messenger RNA (mRNA) precursors

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: The functional ribosome contains three internal structural cavities that orchestrate translation elongation cycles: the Aminoacyl (A) site, the Peptidyl (P) site, and the Exit (E) site.

Solution:

Let's map the binding sequence during translation initiation:

(a) During elongation, all incoming aminoacyl-tRNAs must first enter and bind to the active Aminoacyl (A) site.

(b) However, the translation initiation complex is unique. The specialized initiator tRNA (Met-tRNA$_i^{Met}$) completely bypasses the open A site and binds directly to the **Peptidyl (P) Site (Site Y)**.

(c) This specific placement positions the initial methionine residue to form the first peptide bond once the matching 60S subunit joins the complex to begin elongation.

Final Answer: Peptidyl (P) Site

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: Ultraviolet (UV) radiation damages DNA by inducing covalent bonds between adjacent pyrimidine bases, forming bulky pyrimidine-pyrimidone (6-4) photoproducts or cyclobutane pyrimidine dimers. These lesions distort the double helix structure and stall transcription machinery.

Solution:

Let's evaluate the repair pathway that corrects these bulky helical distortions:

(a) Bulky helical distortions are recognized and corrected exclusively by the **Nucleotide Excision Repair (NER)** pathway.

(b) The NER mechanism employs an endonuclease complex to excise a multi-nucleotide fragment containing the dimer, followed by gap filling by DNA polymerase and sealing by DNA ligase.

(c) Xeroderma pigmentosum is caused by an inherited autosomal recessive mutation in genes encoding the NER repair proteins (such as XPA through XPG), leaving the patient unable to repair UV-induced damage and drastically increasing their risk for cutaneous malignancies.

Final Answer: Nucleotide Excision Repair (NER)

Answer: (C)

Go Back to Question 10

Q11.

Solution

Concept: Lineweaver-Burk plots transform the non-linear Michaelis-Menten equation into a linear form ($1/V_0$ vs $1/[S]$). On these axes, the y-intercept represents $1/V_{max}$ and the x-intercept represents $-1/K_m$.

Solution:

Let's deduce the kinetic parameters from the described geometric intersection point:

(a) The problem states that the lines for the inhibited and uninhibited reactions intersect exactly on the vertical y-axis. This means that the y-intercept ($1/V_{max}$) is identical for both conditions, proving that **V_{max} remains unchanged**.

(b) As the line for the inhibited reaction becomes steeper, its x-intercept shifts closer to the origin (a less negative value), which means that the apparent **K_m increases**.

(c) An unchanged V_{max} combined with an increased K_m is the classic kinetic signature of **competitive inhibition**, where the inhibitor competes directly with the substrate for the active site. This inhibition can be overcome by adding excess substrate.

Final Answer: K_m increases, V_{max} remains unchanged; Competitive inhibition

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: The pyruvate dehydrogenase (PDH) multienzyme complex catalyzes the irreversible conversion of pyruvate into acetyl-CoA. It requires five distinct co-enzymes to complete its coordinated catalytic sequence across three subunits: thiamine pyrophosphate (E_1), lipoic acid (E_2), and coenzyme A, FAD, and NAD^+ (E_3).

Solution:

Let's analyze the oxidation-reduction events within the E_3 (dihydrolipooyl dehydrogenase) domain:

(a) After the E_2 stage completes the transfer of the acetyl group to CoASH, the lipoamide prosthetic group is left in a reduced, inactive sulfhydryl state.

(b) To restore the complex for the next catalytic cycle, the E_3 domain uses an enzyme-bound **Flavin Adenine Dinucleotide (FAD / Co-E 3)** molecule to accept electrons from the reduced lipoamide, regenerating its oxidized form.

(c) The resulting reduced $FADH_2$ then transfers its electrons to a soluble NAD^+ molecule, yielding $NADH + H^+$ as the final product.

Final Answer: Flavin Adenine Dinucleotide (FAD)

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept: Wernicke-Korsakoff syndrome is a severe neurological manifestation of thiamine (Vitamin B_1) deficiency, a condition frequently seen in chronic alcoholism due to poor nutrition and impaired intestinal absorption. Thiamine pyrophosphate (TPP) serves as an essential co-enzyme for several key carbohydrate metabolic pathways.

Solution:

Let's identify the specific enzymes that depend on thiamine pyrophosphate (TPP):

(a) TPP is required by transketolase in the pentose phosphate pathway, pyruvate dehydrogenase, and **α-ketoglutarate dehydrogenase** in the Citric Acid Cycle.

(b) Measuring baseline erythrocyte transketolase activity and observing its immediate normalization upon adding exogenous TPP confirms a functional thiamine deficiency.

(c) Among the choices provided, **α-ketoglutarate dehydrogenase** is the only Citric Acid Cycle enzyme that requires TPP to perform its oxidative decarboxylation sequence. A deficiency in this step impairs ATP production in high-metabolic tissues like the brain.

Final Answer: α-Ketoglutarate dehydrogenase

Answer: (C)

Go Back to Question 13

Q14.

Solution

Concept: Turner syndrome can present with mosaicism, where an individual possesses multiple distinct cell lines derived from a single zygote. This differs from non-mosaic cases, which are typically caused by meiotic errors during gametogenesis.

Solution:

Let's analyze the mechanical divergence between meiotic and mitotic errors:

(a) Meiotic non-disjunction occurs during gamete formation, resulting in a zygote where *every* cell carries the abnormal chromosome count (e.g., non-mosaic $45, X$).

(b) Chromosomal mosaicism ($45, X/46, XX$) develops *after* fertilization has occurred normally to produce a $46, XX$ zygote.

(c) During subsequent early embryonic cleavage divisions, a post-zygotic **mitotic non-disjunction or chromosome lag** event occurs. A chromosome fails to separate properly or is left behind during anaphase, creating two distinct cell lineages that persist throughout development.

Final Answer: Mitotic non-disjunction or chromosome lag during early embryonic cleavage

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept: Mitochondrial (maternal) inheritance describes the transmission of genetic traits encoded within the mitochondrial genome (mtDNA). Because the zygote inherits virtually all of its cytoplasm and organelles from the oocyte rather than the sperm, mitochondrial traits follow a strict maternal pattern.

Solution:

Let's analyze the transmission lines across the generations:

(a) An affected female transmits the phenotype to **all of her biological offspring** (both sons and daughters) with absolute penetrance because her oocytes provide the mitochondrial matrix for the zygote.

(b) Conversely, **affected males never transmit the phenotype** to any of their offspring. The mature spermatozoa contain mitochondria only within the midpiece, which is shed or selectively destroyed upon fertilization. This presentation matches a classic **mitochondrial inheritance** profile.

Final Answer: Mitochondrial (maternal) inheritance

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept: Cri-du-chat (cat's cry) syndrome is a classic congenital structural chromosomal disorder named after the characteristic high-pitched, mewing cry of affected infants, which is caused by abnormal laryngeal development.

Solution:

Let's identify the specific structural chromosomal abnormality that causes this syndrome:

(a) Cri-du-chat syndrome is caused by a partial terminal or interstitial **deletion of the short arm of chromosome 5** (5p).

(b) The severity of symptoms (including profound hypotonia, cognitive impairment, microcephaly, and distinct facial dysmorphism) directly correlates with the size of the deleted segment on the 5p region.

(c) Alternative modifications like duplications, inversions, or isochromosomes do not produce this specific phenotype.

Final Answer: Deletion of the short arm (5p)

Answer: (A)

Go Back to Question 16

Answer Key

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