CSIR NET Life Science Questions Answers With Solutions

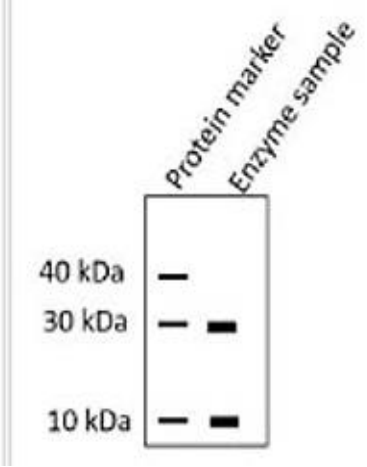
Q1. The central rod domain of keratin protein is 300 amino acids in length. What is the approximate length (in Å) of the rod domain if the peptide consists of:
i) Distorted α-helix
ii) True α-helix
iii) Anti-parallel β-sheet
(a) (i) 425 Å; (ii) 450 Å; (iii) 1041 Å
(b) (i) 450 Å; (ii) 450 Å; (iii) 840 Å
(c) (i) 425 Å; (ii) 425 Å; (iii) 1004 Å
(d) (i) 340 Å; (ii) 425 Å; (iii) 104 Å

Q2. Which one of the following nucleic acids, with the same concentrations in water, will form a stable stem-loop structure upon annealing by heating and flash cooling on ice?
(a) 5' – GGCUUAUUUUCUUCGG – 3'
(b) 5' – CCGAACUUUUAUUCGG – 3'
(c) 5' – AUGCCAUUUUCGGCUU – 3'
(d) 5' – AGAGCGUUUUAUUCGG – 3'

Q3. The amino acid sequence of tetrapeptides (P, Q, R) is shown below:
P) Asp-Gly-Asp-Ser
Q) Gly-Ser-Arg-Gly
R) Gly-Lys-Arg-Ala
i. Calculate the net charge on the above tetrapeptides at pH 7.0
ii. If the mixture of the above tetrapeptides is separated on a cation-exchange column at pH 7.0, which tetrapeptide will elute last?
Choose the correct answer:
(a) P, -2; Q, +1; R, +2 and (ii) R
(b) P, -3; Q, +2; R, +3 and (ii) Q
(c) P, -1; Q, +1; R, +1 and (ii) P
(d) P, -1; Q, 0; R, +1 and (ii) R

Q4. In a mammalian cell undergoing active glycolysis, what would be the effect of a sudden increase in the concentration of metabolites, AMP, citrate, ATP, and glucose-6-phosphate?
(A) Increased ATP inhibits glycolysis.
(B) Increased AMP stimulates glycolysis.
(C) Increased citrate and glucose-6-phosphate stimulate glycolysis.
(D) Increased AMP and citrate inhibit glycolysis.
Which one of the following represents the combination of all correct statements?
(a) C and D
(b) B and D
(c) A and B
(d) A and C

Q5. You have purified an enzyme with a total molecular weight of 60 kDa and analyzed it using SDS-PAGE. The gel results show two distinct bands at 30 kDa and 10 kDa.



Based on this data, which of the following statements is most likely correct about the quaternary structure of this enzyme?
(a) Two subunits of 30 kDa
(b) One subunit of 30 kDa and three subunits of 10 kDa
(c) Six subunits of 10 kDa
(d) Three subunits of 30 kDa and one subunit of 10 kDa

Q6. What is the V0/Vmax ratio for an enzymatic reaction when [S] = 3 Km and 9 Km, respectively?
(a) 0.65 and 0.83
(b) 0.55 and 1.0
(c) 0.93 and 1.2
(d) 0.75 and 0.9

Q7. *Pseudomonas aeruginosa* produces a blue-green pigment called pyocyanin, which is essential for its survival and pathogenicity. To study the pyocyanin biosynthetic pathway, six mutants that cannot produce pyocyanin were isolated. These mutants were crossed with each other, and their ability to restore pyocyanin production was recorded in the following table:

Mutant	1	2	3	4	5	6
1	-	+	+	+	+	+
2	+	-	+	+	+	+
3	+	+	-	+	+	+
4	+	+	+	-	+	+
5	+	+	+	+	-	+
6	+	+	+	+	+	-

is pyocyanin negative,+pyocyanin positive
Based on this data,can you predict how many genes are responsible for pyocyanin production?

(a) One
(b) Two
(c) Five
(d) Six

Q8. Iron (Fe) is taken up by cells via receptor-mediated endocytosis utilizing transferrin and transferrin receptor. In a cell line with a mutation in the transferrin receptor that is unable to interact with transferrin at pH 4-6, which one of the following steps will be first affected in this pathway?
(a) Binding of transferrin to iron in plasma.
(b) Association of iron-bound transferrin with transferrin receptor on the plasma membrane.
(c) Release of iron in the endosomes.
(d) Recycling of transferrin to the plasma membrane.

Q9. In yeast, temperature-sensitive mutants of cell cycle regulators, cdc2 (the key cyclin-dependent kinase), cdc13 (required for telomeric DNA replication), and cdc13 rad9 (which carries an additional mutation in the DNA damage sensor) were isolated. When grown in non-permissive temperatures for a few hours, different phenotypes were observed in these mutants. Choose the option that correctly describes the most likely phenotype for all of these mutants.
(a) The cdc2 mutants will arrest in G1/S, cdc13 in M, and cdc13 rad9 in S-phase.
(b) The cdc2 mutants will arrest after a few divisions in G1, cdc13 in M, and cdc13 rad9 in S-phase.
(c) The cdc2 mutants will arrest in G1/S, G2, and M, cdc13 in G2, and cdc13 rad9 will continue to divide for a few generations.
(d) The cdc2 mutants will arrest in G1/S, G2, and M, cdc13 in G1 and M, and cdc13 rad9 in all phases of the cell cycle.

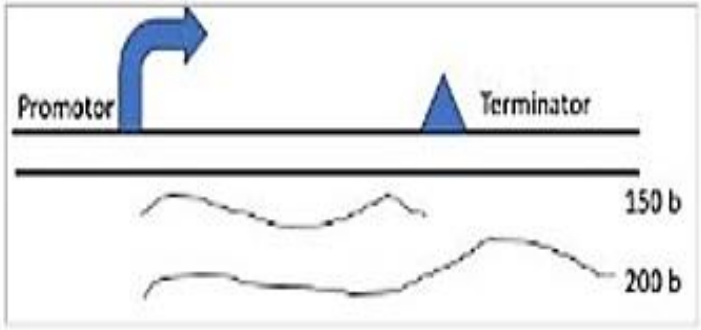
Q10. The statements below attempt to describe a few characteristics of Alu repeats found in the human genome:
A. Alu elements are a class of short interspersed elements (SINEs).
B. SINEs are autonomous transposons.
C. Alu repeat originated from cDNA copies of 7SL RNA.
D. Alu repeats have a relatively high AT content.
E. They are preferentially located in the gene-poor G chromosome bands.
Which one of the following options shows the combination of all correct statements?
(a) A, B, and E
(b) B, C, and D
(c) A and C only
(d) C and E only

Q11. In *C. elegans*, PAR proteins segregate at the cell cortex in the zygote to establish cell polarity. This is dependent on the regulation of the cortical actin cytoskeleton by RhoA. An investigator sought to directly inhibit actin polymerization to analyze the impact of this inhibition on PAR protein localization. Which one of the following chemicals would be the most suitable?
(a)Taxol
(b)Colchicine
(c)Latrunculin
(d) LY294002

Q12. A researcher plans to study protein trafficking into the endoplasmic reticulum (ER). For this purpose, they plan different experimental conditions shown below:
A. The cytosol is mixed with mRNA that codes for a secreted protein, followed by western blotting with antibodies against the secreted protein.
B. The cytosol is mixed with mRNA that codes for a secreted protein and rough microsomes, followed by western blotting with antibodies against the secreted protein.
C. The cytosol is mixed with mRNA that codes for a secreted protein and rough microsomes followed by protease treatment. Subsequently, western blotting with antibodies against the secreted protein is done.
Consider that mRNA that codes for a secreted protein is added in an abundant amount. Which experimental control(s) would be the best to confirm the polypeptide entry into the ER?
(a) A and B only
(b) B only
(c) C only
(d) A, B, and C

Q13. Following statements were made about mRNA splicing:
(A) Involvement of a cis-acting branchpoint site (or branchpoint sequence) present near the 3' end of each exon is essential for splicing.
(B) In the first step of the splicing reaction, 2'-OH of the conserved U at the branch-site acts as a nucleophile to attack the phosphoryl group of the conserved G in the 5' splice site.
(C) The newly liberated intron adapts the shape of a lariat due to the joining of the 5' end of the intron to the branchpoint.
(D) During the splicing process, there is no net gain in the number of chemical bonds.
(E) Prp22 (a DEAD-box helicase) is required for stripping the spliced mRNA from the spliceosome.
Which one of the following options shows the correct combination of all true statements?
(a) B, C, D
(b) C, D, E
(c) A and E only
(d) B and D only

Q14. Given below is the structure of a gene whose transcription is terminated in a Rho-independent manner. When the terminator is operational, the short transcript of 150 bases is formed. When it is not operational, a longer transcript of 200 bases is produced. A researcher generated mutations in the terminator region and examined the transcript lengths.



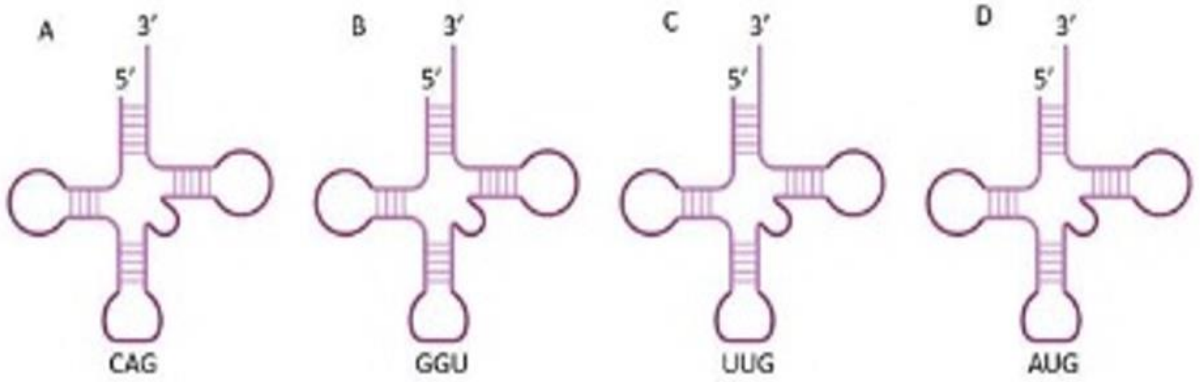
The manipulations performed were:

(A) Three nucleotides of the string of 8Ts were replaced by GCC.
(B) The 8T sequence was transferred to the template strand.
(C) The sequences generating the paired stem were altered to disrupt pairing.
(D) The sequences generating the paired stem were altered to disrupt pairing, but compensatory mutations restored pairing.
Which option correctly predicts the transcript sizes in each case?
(a)Short transcript in A and D; long in B and C
(b)Long transcript in A, B, and D; short in C
(c)Short transcript in B and D; long in A and C
(d) Long transcript in A, B, and C; short in D

Q15. The following statements were made about the initiation of translation in eukaryotes:
(A) eIF2 facilitates correct recognition and binding of ribosomal subunits.
(B) eIF2B activates eIF2 by replacing its GDP with GTP.
(C) eIF3 binds to the 60S ribosomal subunit and inhibits its reassociation with the 40S subunit.
(D) eIF5 promotes association between the 60S ribosomal subunit and the 48S complex.
(E) eIF6 binds to the 60S ribosomal subunit and blocks reassociation with the 40S subunit.
Which one of the following options shows the correct combination of all correct statements?
(a) A, B, D
(b) B, D, E
(c) B, C, E
(d) A, C, D

Q16. The following statements were made about the rolling circle mechanism of replication:
(A) It occurs unidirectionally, with only one replicating fork.
(B) The E. coli ΦX174 phage uses this mechanism to replicate its double-stranded circular genome.
(C) E. coli utilizes this mechanism to replicate its double-stranded DNA genome.
(D) In λ phage, the progeny DNA may range several genomes long before it is packaged.
(E) The lagging strand is not formed in the rolling circle mechanism of replication.
Which one of the following options shows the correct combination of all correct statements?
(a)A and C
(b)A and D
(c)B and C
(d)D and E

Q17. A portion of an mRNA encoding a protein is shown below, with the start codon underlined.
5' .....CCUCAAACAGACACCAUGUUGCACCUGACUCCU....3'
Which one of the following tRNAs is most likely used for translating the second codon in the open reading frame of the protein?



(a) A
(b) B
(c) C
(d) D

Q18. The table provides protein modifications (Column X) and their functions (Column Y)

Column X (Protein Modification)	Column Y (Function)
a. Palmitoylation	ii. Membrane anchoring
b. polySUMOylation	i. Protein degradation
c. Glycosylphosphatidylinositol (GPI)	ii. Membrane anchoring
d. Mannose-6-phosphate	iii. Lysosomal targeting

Which one of the following options represents all correct matches between Column X and Column Y?
(a) a-ii; b-i; c-iv; d-iii
(b) a-i; b-iv; c-ii; d-iii
(c) a-ii; b-i; c-ii; d-iii
(d) a-iv; b-iv; c-ii; d-i

Q19. Bacteria employ various mechanisms to invade or enter host cells, which can be either phagocytic or non-phagocytic in nature. Given below are mechanisms commonly used for bacterial entry into non-phagocytic cells.
Statements:
(A) Some bacteria express a protein called invasin that is recognized by host-cell β1 integrins.
(B) Actin polymerization along with assembly of the clathrin coat results in the internalization of bacteria by the zipper mechanism.
(C) Some bacteria, including Salmonella enterica, use the trigger mechanism to inject a set of effector molecules into the cytosol through the type III secretion system.
(D) Some bacteria attach to host cell surface receptors inducing local elevation of Ca^{2+} in the cytosol, leading to the fusion of lysosomes with bacteria-containing plasma membrane vesicles.
Which one of the following options represents the combination of correct mechanisms for invading non-phagocytic cells?
(a) A, B, and C
(b) B, C, and D
(c) A, B, and D
(d) A, C, and D

Q20. The cAMP-PKA-CREB pathway is crucial in biological processes such as hormone synthesis, metabolism, and memory formation. The statements describe the effects of mutations in this pathway on gene transcription by CREB.
(A) Loss of function mutation in a cAMP binding site of the PKA regulatory subunit leads to the inactivation of gene expression.
(B) Activating mutation in the GTP-binding domain of the α subunit of Gs leads to the activation of gene expression.
(C) Inactivating mutation that prevents the regulatory subunit of PKA from binding the catalytic subunit leads to the activation of gene expression.

(D) Inactivating mutation in the PKA phosphorylation site of CREB leads to the activation of gene expression.

Which one of the following statement is Incorrect?

(a) Only A
(b) A and D
(c) Only D
(d) B and D

Solutions

S1. Ans.(a)
Sol. (i) 425 Å; (ii) 450 Å; (iii) 1041 Å
Explanation:

Keratin, a structural protein, has a rod domain that can adopt different secondary structures, each having a different extension per amino acid. The three conformations considered in this question are the **distorted α-helix, true α-helix, and anti-parallel β-sheet**.

1. **True α-helix:**
   - In an α-helix, each amino acid extends approximately **1.5 Å per residue** along the helical axis.
   - Given **300 amino acids**, the total length is: $300 \times 1.5 = 450 A^{\circ}$
2. **Distorted α-helix:**
   - The distorted α-helix has slightly different hydrogen bonding, leading to a reduced extension of **1.4 Å per residue**.
   - For **300 amino acids**, the approximate length is: $300 \times 1.4 = 425 A^{\circ}$
3. **Anti-parallel β-sheet:**
   - In a β-sheet conformation, each amino acid contributes **3.4 Å per residue** to the length.
   - So, for **300 amino acids**, the total length is: $300 \times 3.4 = 1020 A^{\circ} \approx 1041 A^{\circ}$

Thus, the correct answer is **(a) 425 Å, 450 Å, and 1041 Å**.

Information Booster:

- **Keratin proteins** play a key role in providing mechanical strength to cells and tissues, especially in hair, nails, and skin.
- **α-helices** are the predominant structure in fibrous proteins like keratin, with strong hydrogen bonding contributing to stability.
- **Distorted α-helices** often arise due to amino acid variations or environmental factors like pH and temperature.
- **Anti-parallel β-sheets** are common in structural proteins but are more extended compared to α-helices.
- The **length of a protein domain** is crucial for its function, influencing interactions with other cellular components.

Additional Knowledge:

(i) Distorted α-helix (425 Å)

- A **distorted α-helix** has irregular hydrogen bonding, affecting its helical turn.
- The helical pitch varies slightly, leading to a shorter total length than a regular α-helix.
- Found in **mutant keratin proteins** or in keratin regions interacting with other molecules.

(ii) True α-helix (450 Å)

- The **classic α-helix** has **3.6 residues per turn** and a pitch of **5.4 Å per turn**.
- It is **right-handed**, stabilized by **intramolecular hydrogen bonds**.
- Common in **fibrous proteins**, such as α-keratin, **myosin**, and **collagen-related proteins**.

(iii) Anti-parallel β-sheet (1041 Å)

- The **β-sheet** structure is more extended than an α-helix.
- Each residue contributes **3.4 Å**, giving a total of **~1041 Å for 300 amino acids**.
- Found in **structural and globular proteins**, including silk fibroin.

S2. Ans.(b)

Sol. 5' – CCGAACUUUUAUUCGG – 3'

Explanation:

A **stem-loop structure** (also called a **hairpin loop**) is a secondary structure that forms when a single-stranded nucleic acid **folds back on itself**, creating a complementary **double-stranded stem region** and an **unpaired loop region**.

Key factors influencing **stem-loop stability** include:

1. **Complementary Base Pairing**: Strong base-pairing between regions forms a **stable stem**.
2. **GC Content**: **G≡C pairs form three hydrogen bonds**, making them more stable than A=U pairs in RNA.
3. **Loop Length**: Too short or too long loops reduce stability.
   - **(a) 5' – GGCUUAUUUUCUUCGG – 3'**
     - The potential **stem**: GGCU.....UCGG
     - The loop is **too long** due to excess U's, reducing stability.
   - **(b) 5' – CCGAACUUUUAUUCGG – 3'** (**Correct Answer**)
     - The potential **stem**: CCGA.....UCGG
     - A **strong GC-rich stem** with a **balanced loop** makes this structure stable.
   - **(c) 5' – AUGCCAUUUUCGGCUU – 3'**
     - The potential **stem**: AUGCC.....CGCUU
     - The stem is **less GC-rich**, making it **weaker** compared to (b).
   - **(d) 5' – AGAGCGUUUUAUUCGG – 3'**
     - The potential **stem**: AGAGC.....CGG
     - The **loop is large**, leading to an unstable structure.

Thus, **option (b) forms the most stable stem-loop structure due to a well-balanced GC-rich stem and an optimal loop size**.

Information Booster:

- **Stem-loop structures** are critical in **RNA folding**, affecting stability and function.
- **RNA hairpins** are found in tRNA, ribosomal RNA, and regulatory non-coding RNAs.
- **GC-rich stems** contribute to **high thermal stability** due to three hydrogen bonds per pair.
- **Optimal loop size** (4–10 nucleotides) enhances stability; very short or long loops destabilize the structure.
- **Flash cooling** after heating helps stabilize the correct conformation by rapidly locking in base-pairing interactions.

Additional Knowledge:

(a) 5' – GGCUUAUUUUCUUCGG – 3'

- The presence of **too many uracils (U's) in the loop** increases flexibility but reduces stem stability.
- **GGCU** at the start can form a partial **GC-paired stem**, but the rest is weak.

(b) 5' – CCGAACUUUUAUUCGG – 3' (Most Stable)

- The **stem** (CCGA...UCGG) contains **strong GC pairing**, ensuring high stability.
- The loop contains **UUUUUAUU**, which is an ideal size for stable formation.

(c) 5' – AUGCCAUUUUCGGCUU – 3'

- The **stem** contains **AUGCC...GGCUU**, but **weaker AU pairs** reduce stability.
- Loop formation is possible but **less stable** than (b).

(d) 5' – AGAGCGUUUUAUUCGG – 3'

- The **AGAGC...CGG stem** is possible, but the loop is **longer**, making it unstable.
- The presence of **multiple purines (G, A) in the loop** affects folding efficiency.

S3. Ans.(a)

Sol. P = -2; Q = +1; R = +2 and (ii) R

Explanation:

To determine the net charge of each **tetrapeptide at pH 7.0**, we need to analyze the charge contributions of the amino acid **side chains** and **terminal groups**:

Step 1: Assigning pKa Values and Charge Contributions

- **Aspartic Acid (Asp, D):** pKa ~3.9 → **Negative (-1) at pH 7**
- **Glutamic Acid (Glu, E):** pKa ~4.2 → **Negative (-1) at pH 7**
- **Arginine (Arg, R):** pKa ~12.5 → **Positive (+1) at pH 7**
- **Lysine (Lys, K):** pKa ~10.5 → **Positive (+1) at pH 7**
- **N-terminal (NH_3^+ group):** pKa ~9 → **Remains +1 at pH 7**
- **C-terminal (COO^- group):** pKa ~2 → **Remains -1 at pH 7**

Step 2: Net Charge Calculation for Each Tetrapeptide

P) Asp-Gly-Asp-Ser

- **Asp (D) contributes -1 each** → (-1) + (-1) = **-2**
- **N-terminal (+1) and C-terminal (-1) cancel out**
- **Total net charge: -2**

Q) Gly-Ser-Arg-Gly

- **Arg (R) contributes +1**
- **N-terminal (+1) and C-terminal (-1) mostly cancel out**
- **Total net charge: +1**

R) Gly-Lys-Arg-Ala

- **Lys (K) contributes +1, Arg (R) contributes +1**
- **N-terminal (+1) and C-terminal (-1) mostly cancel out**
- **Total net charge: +2**

Step 3: Elution Order in Cation-Exchange Chromatography

- **Cation-exchange chromatography retains positively charged peptides** (binds strongest to those with higher net positive charge).
- **Negatively charged peptides elute first**, while **highly positive peptides elute last**.

Elution order (first to last):

1. **P (-2) → Elutes first (weakly retained).**
2. **Q (+1) → Moderately retained.**
3. **R (+2) → Strongly retained, elutes last.**

Information Booster:

- **Cation-exchange chromatography** retains **positively charged molecules**, while **anion-exchange chromatography** retains **negatively charged molecules**.
- **pH significantly affects peptide charge** due to ionizable groups.
- **Aspartic acid & Glutamic acid** lower the pI (more negative charge at neutral pH).
- **Arginine & Lysine** increase the pI (more positive charge at neutral pH).
- **Proteins with a high pI bind more strongly to cation-exchange columns** and require higher salt concentrations to elute.

Additional Knowledge:

(a) P = -2

- Contains **two Asp residues**, making it highly negative at pH 7.
- **Least retained in cation-exchange chromatography**.

(b) Q = +1
- Contains **Arg** which makes it slightly positive.
- Retained **moderately** in the column.

(c) R = +2 (Most positive, elutes last)
- Contains **both Lys and Arg**, giving it the highest positive charge.
- **Strongly retained**, requiring **higher salt concentration** to elute.

S4. Ans.(c)
Sol. A and B
Explanation:
Glycolysis is regulated by several **key metabolites** that act as activators or inhibitors of **rate-limiting enzymes** such as **phosphofructokinase-1 (PFK-1)** and **pyruvate kinase**.

1. **ATP and Glycolysis (A is Correct)**
   - **ATP is an allosteric inhibitor of glycolysis**, particularly of **phosphofructokinase-1 (PFK-1)**.
   - When ATP levels are high, **cells do not need more energy**, so glycolysis is **slowed down**.
2. **AMP and Glycolysis (B is Correct)**
   - **AMP is a key activator of glycolysis**.
   - High AMP levels indicate **low energy (ATP is depleted)**, leading to **activation of PFK-1** and **stimulation of glycolysis**.
3. **Citrate and Glucose-6-Phosphate (C is Incorrect)**
   - **Citrate inhibits glycolysis** (not stimulates it), acting as a feedback inhibitor of **PFK-1**.
   - **Glucose-6-phosphate regulates glycolysis indirectly**, but it mainly regulates **glycogen synthesis**.
4. **AMP and Citrate (D is Incorrect)**
   - **AMP stimulates** glycolysis, **not inhibits** it.
   - **Citrate inhibits glycolysis**, but this statement is incorrect because AMP and citrate **do not both inhibit glycolysis**.

Thus, the correct answer is **(c) A and B**.
Information Booster:
- **Phosphofructokinase-1 (PFK-1)** is the **rate-limiting enzyme of glycolysis**.
- **High ATP inhibits PFK-1**, slowing glycolysis to prevent excess ATP production.
- **AMP is a strong activator of PFK-1**, ensuring glycolysis continues when ATP is low.
- **Citrate is an indicator of the TCA cycle's activity** and inhibits glycolysis when the cell has sufficient energy.
- **Glucose-6-phosphate inhibits hexokinase**, preventing unnecessary glucose breakdown when sufficient glucose is available.

Additional Knowledge:
(A) ATP Inhibits Glycolysis (Correct)
- ATP binds to **PFK-1**, reducing its activity.
- When ATP is high, glycolysis is downregulated to prevent excessive ATP production.
- **Example:** Resting muscle has high ATP, leading to glycolysis inhibition.

(B) AMP Stimulates Glycolysis (Correct)
- **AMP activates PFK-1**, promoting glycolysis.
- High AMP signals **low cellular energy**.
- **Example:** Intense exercise increases AMP, activating glycolysis.

(C) Citrate and Glucose-6-Phosphate Stimulate Glycolysis (Incorrect)
- **Citrate inhibits** glycolysis by allosterically inhibiting **PFK-1**.
- **Glucose-6-phosphate primarily regulates glycogen metabolism** rather than glycolysis.

(D) AMP and Citrate Inhibit Glycolysis (Incorrect)
- AMP **stimulates** glycolysis, while citrate **inhibits** it.
- This statement is incorrect as both do not inhibit glycolysis.

S5. Ans.(b)
Sol. One subunit of 30 kDa and three subunits of 10 kDa
Explanation:
The enzyme has a total molecular weight of **60 kDa**, but the **SDS-PAGE results show two bands at 30 kDa and 10 kDa**, suggesting that it consists of multiple subunits.

- **Step 1: Understanding SDS-PAGE**
  SDS-PAGE separates proteins based on size, breaking them down into individual subunits. A single polypeptide chain appears as a distinct band.
- **Step 2: Interpreting the Gel Bands**
  - The presence of a **30 kDa band** suggests that a **subunit of this weight is part of the enzyme**.
  - The **10 kDa band** indicates additional smaller subunits.
- **Step 3: Evaluating Possible Subunit Combinations**
  - **(a) Two subunits of 30 kDa** → (30 × 2) = 60 kDa (*Possible but unlikely based on intensity of bands*)
  - **(b) One subunit of 30 kDa and three subunits of 10 kDa** → (30 + 10×3) = 60 kDa(*Most reasonable choice*)
  - **(c) Six subunits of 10 kDa** → (10 × 6) = 60 kDa (*Possible but does not fit well with band intensities*)
  - **(d) Three subunits of 30 kDa and one subunit of 10 kDa** → (30×3 + 10×1) = 100 kDa (*Incorrect, exceeds total weight of enzyme*)

Thus, the **best answer is (b): One subunit of 30 kDa and three subunits of 10 kDa.**

Information Booster

1. **SDS-PAGE helps determine subunit composition** by separating proteins based on molecular weight.
2. **Multisubunit proteins can be homomeric (same subunits) or heteromeric (different subunits).**
3. **Quaternary structure is determined by the number and arrangement of subunits in a protein.**
4. **Enzymes often have regulatory subunits** that assist in function, stability, or activity modulation.
5. **Band intensity in SDS-PAGE provides clues about subunit quantity and relative abundance.**
6. **Examples of heteromeric proteins:** Hemoglobin ($\alpha_2\beta_2$), RNA polymerase, and ATP synthase.

Additional Knowledge

(A) Two subunits of 30 kDa

- If the enzyme had **only two subunits of 30 kDa**, SDS-PAGE should show **one band at 30 kDa**.
- Since an additional **10 kDa band** is visible, this is **unlikely to be the correct answer**.

(B) One subunit of 30 kDa and three subunits of 10 kDa (Correct Answer)

- This suggests the enzyme has **one large subunit (30 kDa) and three smaller ones (10 kDa each)**.
- Many biological enzymes follow this pattern, where the larger subunit has **catalytic activity**, and smaller ones play **regulatory or structural roles**.
- **Example:** Pyruvate Dehydrogenase, which has different subunit sizes for various functions.

(C) Six subunits of 10 kDa

- If the enzyme consisted of **six identical 10 kDa subunits**, SDS-PAGE would show **only a single band at 10 kDa**.
- Since **both 30 kDa and 10 kDa bands are present**, this option is **less likely**.

(D) Three subunits of 30 kDa and one subunit of 10 kDa (Incorrect)

- The total weight would be **(30×3 + 10×1) = 100 kDa**, which **exceeds the given molecular weight (60 kDa)**.
- Since the enzyme is **not 100 kDa**, this option is **incorrect**.

S6. Ans.(d)
Sol. 0.75 and 0.9
Explanation:

To determine the V_0/V_{max} ratio, we use the **Michaelis-Menten equation**:

$$V_0 = \frac{V_{max}[S]}{K_m + [S]}$$

Step 1: Calculate V_0/V_{max} for [S] = 3 Km

Substituting **[S] = 3 Km** into the equation:

$$V_0 = \frac{V_{max}(3K_m)}{K_m + 3K_m} = \frac{3V_{max}K_m}{4K_m} = \frac{3}{4}V_{max} = 0.75V_{max}$$

Thus,

$$V_0/V_{max} = 0.75$$

Step 2: Calculate V_0/V_{max} for [S] = 9 Km

Substituting **[S] = 9 Km** into the equation:

$$V_0 = \frac{V_{max}(9K_m)}{K_m + 9K_m} = \frac{9V_{max}K_m}{10K_m} = \frac{9}{10}V_{max} = 0.9V_{max}$$

Thus,

$$V_0/V_{max} = 0.9$$

Step 3: Choose the Correct Answer
From the given options, the correct answer is:
Option (d): 0.75 and 0.9
Information Booster :

1. **Michaelis-Menten equation** is fundamental in enzyme kinetics, explaining how reaction velocity depends on substrate concentration.
2. **Km (Michaelis constant)** represents the substrate concentration at which the reaction velocity is **half of Vmax**.
3. **When [S] = Km, V0=0.5Vmax**
4. **When [S] = 10 Km, V0 approaches 0.91Vmax** showing near saturation.
5. **Higher [S] values lead to saturation**, meaning V0 asymptotically approaches Vmax but never exceeds it.
6. **Enzyme inhibitors (competitive, non-competitive, uncompetitive)** affect Km and Vmax differently.

Additional Knowledge:
(A) 0.65 and 0.83

- Incorrect because at **[S] = 3K_m**, V_0/Vmax should be **0.75**, not **0.65**.
- At **[S] = 9K_m**, the correct value is **0.9**, not **0.83**.

(B) 0.55 and 1.0

- At **[S] = 3K_m**, V_0/Vmax = 0.75, which is **greater than 0.55**.
- At **[S] = 9K_m**, V_0/Vmax = 0.9, but **not 1.0** (which happens only at infinite [S]).

(C) 0.93 and 1.2

- These values are incorrect because V_0 cannot **exceed Vmax (1.2 is not possible)**.

S7. Ans.(c)

Sol. Five

A complementation group consists of mutants that fail to complement each other, meaning they are defective in the same gene.

1. Identify Patterns in the Table:
   - If a mutant does not complement another, they belong to the same gene.
   - If a mutant complements another ("+" result), they belong to different genes.
2. Analyzing the Table:
   - Each mutant complements all other mutants, except itself.
   - This suggests that each mutant represents a separate gene.
   - However, if there were six genes, we would expect at least one case where a mutant does not complement another.
   - Since one pair of mutants shares the same gene, we conclude there are five distinct genes.
3. The six mutants fall into five complementation groups **(**meaning five genes are involved in pyocyanin biosynthesis).
4. The correct answer is: **(c) Five Genes**

Information booster :

1. Complementation tests help identify the number of genes involved in a biosynthetic pathway.
2. If two mutants do not complement each other (- result), they belong to the same gene.
3. If two mutants complement each other (+ result), they belong to different genes.
4. Since five unique complementation groups exist, five genes control pyocyanin production.
5. Understanding genetic pathways through complementation analysis is crucial for metabolic and molecular biology studies.

Additional Knowledge:

- **Complementation analysis** is widely used in **genetics and molecular biology** to identify the number of genes involved in a process.
- This type of experiment is commonly used for studying **biosynthetic pathways**, **genetic disorders**, and **functional genomics**.

S8. Ans.(d)

Sol. Recycling of transferrin to the plasma membrane

The transferrin receptor mutation that prevents interaction with transferrin at pH 4-6 affects the recycling of transferrin to the plasma membrane. Normally, iron-bound transferrin (holo-transferrin) binds to the transferrin receptor (TfR) at the cell surface and is internalized via receptor-mediated endocytosis. In the acidic environment of the endosome (pH ~5.5), transferrin undergoes a conformational change that reduces its affinity for iron, facilitating its release into the cytoplasm.

Information Booster:

1. **Transferrin is a glycoprotein** responsible for iron transport in the blood.
2. **Iron uptake occurs via receptor-mediated endocytosis**, where transferrin binds to transferrin receptor (TfR) on the cell surface.

3. **Endosomal acidification (pH ~5.5) induces iron release**, allowing free iron to enter the cytoplasm.
4. **The transferrin receptor then recycles to the plasma membrane**, returning apo-transferrin (iron-free transferrin) to the extracellular space.
5. **Mutations in TfR can cause iron-related disorders**, such as anemia due to defective iron uptake.
6. **Iron is essential for cellular functions**, including DNA synthesis, respiration, and metabolism.

Additional Knowledge:

(a) Binding of transferrin to iron in plasma:

- Iron exists in two forms in plasma: **Fe^{2+} (ferrous) and Fe^{3+} (ferric)**.
- Transferrin binds Fe^{3+} with high affinity at physiological pH (~7.4).
- This step occurs **independently of the transferrin receptor** and is not affected by receptor mutations.

(b) Association of iron-bound transferrin with transferrin receptor on the plasma membrane:

- The transferrin receptor (TfR) is a **type II transmembrane glycoprotein** that binds transferrin.

(c) Release of iron in the endosomes:

- Once internalized, the transferrin-TfR complex enters early endosomes.
- Acidification of the endosome (pH 5-6) triggers **conformational changes in transferrin**, reducing its affinity for Fe^{3+}.

(d) Recycling of transferrin to the plasma membrane (correct answer):

- After iron release, apo-transferrin (iron-free transferrin) remains bound to TfR and recycles to the plasma membrane.
- In a cell line with a mutation in the transferrin receptor that is unable to interact with transferrin at pH 4-6, transferrin will not bind with transferrin receptor again and the receptor will recycle to plasma membrane alone without transferrin.
- At neutral pH (extracellular), apo-transferrin dissociates from TfR and reenters circulation.

S9. Ans.(c)

Sol. The cdc2 mutants will arrest in G1/S, G2, and M, cdc13 in G2, and cdc13 rad9 will continue to divide for a few generations.

1. **cdc2 Mutants:**
   - **Cdc2 (Cyclin-dependent kinase) is essential for cell cycle progression.**
   - It regulates **G1/S transition**, **G2/M transition**, and **mitosis**.
   - When mutated, cells **fail to progress past these checkpoints**, leading to **arrest at G1/S, G2, and M phases**.
2. **cdc13 Mutants:**
   - **Cdc13 is required for telomere maintenance and DNA replication.**
   - Its loss causes **telomere damage**, triggering **a G2 DNA damage checkpoint arrest**.
3. **cdc13 rad9 Mutants:**
   - **Rad9 is a DNA damage checkpoint protein** required for stopping the cell cycle upon DNA damage.
   - If **rad9 is mutated in a cdc13 background**, the telomere defects **fail to trigger a checkpoint arrest**, allowing the cells to **continue dividing for a few generations** until lethal damage accumulates.

Thus, the most accurate description of the phenotypes of these mutants is **option (c)**.

Information Booster:

1. **Cdc2 (CDK1 in higher eukaryotes) is a master regulator of the cell cycle.**
2. **Cdc13 is a telomere-binding protein** essential for protecting telomeres from degradation.
3. **Rad9 is a checkpoint protein** that signals DNA damage and induces cell cycle arrest.
4. **Loss of Rad9 in cdc13 mutants leads to unchecked telomere loss**, delaying cell cycle arrest.
5. **Yeast temperature-sensitive mutants are commonly used** to study essential genes.
6. **CDK1 (human homolog of cdc2) is a key target in cancer therapy**, as its dysregulation leads to uncontrolled cell proliferation.

S10. Ans.(c)

Sol. A and C only.

1. Statement A - Correct:
   - Alu elements are classified as short interspersed elements (SINEs).
   - SINEs are a class of retrotransposons that do not encode their own reverse transcriptase but rely on other mobile elements (e.g., LINEs) for transposition.
2. Statement B - Incorrect:
   - SINEs are not autonomous transposons because they lack the necessary enzymatic machinery for their own mobilization.
   - Unlike LINEs, which encode reverse transcriptase, SINEs require the LINE-1 (L1) retrotransposon machinery for transposition.
3. Statement C - Correct:
   - Alu repeats originated from cDNA copies of 7SL RNA, a component of the signal recognition particle (SRP).
   - Alu elements are derived from processed RNA sequences and subsequently amplified in the genome.
4. Statement D - Incorrect:
   - Alu elements are GC-rich, not AT-rich.
   - They are predominantly found in gene-rich, GC-rich regions of the genome.
5. Statement E - Incorrect:
   - Alu elements preferentially localize in GC-rich regions, which are typically gene-rich regions.
   - G-bands on chromosomes are gene-poor and AT-rich, where Alu elements are less frequent.

Thus, the correct statements are A and C only, making option (3) the correct answer.

Information Booster:

1. Alu elements are the most abundant transposable elements in the human genome, making up about 10% of total DNA.
2. They are non-autonomous transposons, meaning they require LINE-1 elements for mobilization.
3. Alu sequences originated from 7SL RNA, a component of the signal recognition particle (SRP), which plays a role in protein targeting.
4. They are preferentially found in GC-rich regions, often near genes and regulatory elements.
5. Alu elements are linked to genome evolution, genetic disorders, and some diseases such as Alu-mediated recombination disorders.

Additional Knowledge:

(A) Alu elements as SINEs (Correct)

- SINEs are non-autonomous retrotransposons, meaning they do not encode their own enzymes for transposition.
- Alu elements belong to the primate-specific class of SINEs and rely on LINE-1 reverse transcriptase for mobility.

(B) SINEs as Autonomous Transposons (Incorrect)
- Unlike LINEs, SINEs lack reverse transcriptase and endonuclease activity, making them non-autonomous.
- SINEs rely on LINE-1 for retrotransposition via target-primed reverse transcription (TPRT).

(C) Alu Elements Derived from 7SL RNA (Correct)
- Alu elements evolved from 7SL RNA, a component of the signal recognition particle (SRP).
- This RNA was reverse-transcribed into cDNA, inserted into the genome, and later amplified.

(D) Alu Repeats as AT-rich (Incorrect)
- Alu elements are GC-rich, especially in promoter regions of genes.
- High GC content makes Alu elements preferentially located in gene-dense regions.

(E) Alu Elements in Gene-poor G-bands (Incorrect)
- G-bands are AT-rich and gene-poor, but Alu elements prefer GC-rich, gene-dense regions.
- Thus, Alu elements are less common in G-bands.

S11. Ans.(c)
Sol. Latrunculin.
Explanation:
- **Latrunculin is the most suitable choice** because it **directly inhibits actin polymerization** by binding to actin monomers (G-actin) and preventing their incorporation into filaments (F-actin).
- Since **PAR protein localization in C. elegans depends on cortical actin dynamics**, disrupting actin polymerization with **Latrunculin** helps study its role in cell polarity.

Information Booster:
1. **Actin cytoskeleton regulates cell polarity** in various organisms, including *C. elegans*.
2. **PAR proteins (Partitioning-defective proteins)** establish asymmetric cell division in the embryo.
3. **Latrunculin binds to G-actin and prevents F-actin formation**, disrupting actin-based processes.
4. **Microtubule inhibitors (Taxol, Colchicine) do not target actin and would be ineffective.**
5. **RhoA signaling regulates actin polymerization**, affecting cortical contractility and polarity.

Additional Knowledge :
(1) Taxol (Incorrect - Targets Microtubules)
- Taxol **stabilizes microtubules by preventing depolymerization**.
- Used in **cancer therapy** to block cell division.
- **Does not affect actin polymerization**, so it is not suitable for this experiment.

(2) Colchicine (Incorrect - Targets Microtubules)
- Colchicine **inhibits microtubule polymerization** by binding to tubulin.
- Used to treat **gout and inflammation** by disrupting microtubule-dependent processes.
- **Does not target actin**, making it unsuitable for this study.

(3) Latrunculin (Correct - Inhibits Actin Polymerization)
- Directly binds **monomeric G-actin**, preventing filament formation.
- Commonly used to **study actin-dependent processes**, including cell polarity, migration, and division.
- **Best choice for inhibiting actin polymerization** in this experiment.

(4) LY294002 (Incorrect - PI3K Inhibitor)
- LY294002 is a **phosphoinositide 3-kinase (PI3K) inhibitor**, affecting signaling pathways.
- Used to study **cell growth, survival, and metabolism**, not cytoskeletal dynamics.
- **Not related to actin polymerization**, making it an incorrect choice.

S12. Ans.(d)
Sol. A,B and C
Explanation:
To confirm polypeptide entry into the ER, the experiment must distinguish between proteins that have been successfully translocated into the ER lumen and those that remain in the cytosol.
Information Booster:
1. **Secreted proteins are synthesized in the cytoplasm but require ER translocation for proper folding and secretion.**
2. **Western blotting alone cannot confirm ER entry**—it only detects protein expression.
3. **Protease treatment is a crucial control**—it degrades unprotected (cytosolic) proteins but **not ER-protected proteins**.
4. **Rough microsomes (B & C) mimic the ER environment**, allowing co-translational translocation of proteins.
5. **The signal sequence directs proteins into the ER**, where they are processed and secreted.
6. ER translocation is essential for secretion, membrane targeting, and protein processing.
7. Protease-sensitive vs. protease-protected proteins distinguish cytosolic vs. ER-localized proteins.

Additional Knowledge :
(A) Mixing Cytosol with mRNA and Performing Western Blotting
- This only confirms protein synthesis but **not translocation into the ER**.
- The protein **could be in the cytosol** rather than inside the ER.

(B) Addition of Rough Microsomes and Western Blotting
- **Microsomes provide the ER environment**, allowing translation and translocation.
- However, **this does not confirm if the protein is inside the ER or still associated with the cytosolic face** of the microsomes.
- Additional steps (e.g., protease protection assay) are needed.

(C) Protease Treatment after Microsome Addition and Western Blotting
- **Protease digests any protein that is not inside the ER.**
- If the **protein remains detectable after protease treatment**, it confirms successful ER translocation.
- This is the **gold standard method to confirm ER entry**.

S13. Ans.(b)
Sol. C, D, E
Explanation:
- Statement A (Incorrect): The branchpoint sequence is located within the intron, not at the 3' end of an exon. While the branchpoint is crucial for splicing, the positioning in the statement is incorrect.
- Statement B (Incorrect): The first step of splicing involves a nucleophilic attack by the 2'-OH of an adenine (A) at the branch-site, not a conserved uracil (U). The mention of U instead of A makes it incorrect.
- Statement C (Correct): The lariat structure is formed when the 5' end of the intron covalently joins to the branchpoint A, making it a correct statement.
- Statement D (Correct): Splicing does not create or destroy net chemical bonds but instead rearranges phosphodiester linkages, meaning no net gain occurs.
- Statement E (Correct): Prp22, a DEAD-box helicase, plays a crucial role in removing the spliced mRNA from the spliceosome.

Thus, the correct combination of true statements is C, D, and E, making Option (2) the correct answer.

Information Booster:

1. Pre-mRNA splicing removes introns and joins exons to form mature mRNA.
2. The branchpoint sequence is a conserved intron sequence, NOT at the exon-intron junction.
3. Lariat formation results from the 2'-5' linkage between the branchpoint A and the 5' end of the intron.
4. Spliceosome complex includes snRNPs (U1, U2, U4, U5, U6), which regulate splicing.
5. Prp22 helicase is essential for spliced mRNA release from the spliceosome.
6. Defective splicing can lead to Spinal Muscular Atrophy (SMA) and β-thalassemia.

Additional Knowledge:

(A) Branchpoint Sequence and Splicing

- The branchpoint site is present within the intron, NOT at the 3' end of an exon.
- It contains a conserved adenine (A), essential for initiating the first splicing reaction.
- Mutations in this sequence can cause abnormal splicing and disease.

(B) First Step of Splicing and Nucleophilic Attack

- The first step of pre-mRNA splicing involves a nucleophilic attack by the 2'-OH of an adenine (A) at the branch-site.
- This attack breaks the 5' splice site, forming a lariat intermediate.
- Since the statement incorrectly mentions U instead of A, it is false.

(C) Lariat Formation in Splicing

- The lariat structure forms due to the covalent bond between the 5' end of the intron and the branchpoint A.
- This is a temporary structure before intron degradation.

(D) Chemical Bond Rearrangement in Splicing

- Splicing does not add or remove net chemical bonds.
- It only rearranges phosphodiester linkages, ensuring correct exon joining.

(E) Role of Prp22 in mRNA Splicing

- Prp22 is a DEAD-box helicase that removes spliced mRNA from the spliceosome.
- It uses ATP hydrolysis to release the mature transcript.
- Mutations in Prp22 can trap mRNA within the spliceosome, disrupting translation.

S14. Ans.(d)

Sol. Long transcript in A,B, and C; short in D

Explanation:

Understanding Rho-independent Termination Mechanism:

- Rho-independent termination **relies on two key features**:
  1. **GC-rich stem-loop structure (hairpin)** in the mRNA.
  2. **A string of uracils (U)** following the hairpin.
- **Mechanism:**
  - The **hairpin structure** causes **RNA polymerase to pause**.
  - The **weak U-A interactions** (between the transcript and template DNA) allow **RNA to dissociate**, ending transcription.
  - **Disrupting either the hairpin or the U-rich sequence can prevent termination**, leading to a longer transcript.

Analysis of Each Mutation:

(A) Three nucleotides of the string of 8Ts were replaced by GCC.

- **Effect on termination:**
  - The **string of uracils is crucial for proper termination** because **U-A bonds are weak**.
  - **Replacing three uracils with G/C bases** strengthens base pairing, making termination inefficient.

- **Expected transcript size:**
  - **Long transcript (200 bases)** because termination is disrupted.

(B) The 8T sequence was transferred to the template strand.

- **Effect on termination:**
  - Normally, **Ts in the non-template strand result in U's in the mRNA transcript**.
  - **If Ts are moved to the template strand**, the transcript will have **A's instead of U's**.
  - **A-rich sequences do not cause termination**, as **A-U pairs are stronger** than U-A pairs.
- **Expected transcript size:**
  - **Long transcript (200 bases)** because the U-run is missing, preventing termination.

(C) Disrupting the hairpin structure.

- **Effect on termination:**
  - The **hairpin structure is necessary** to pause RNA polymerase.
  - If **hairpin formation is disrupted**, termination efficiency drops significantly.
- **Expected transcript size:**
  - **Long transcript (200 bases)** because RNA polymerase fails to pause.

(D) Disrupting the hairpin but restoring it with compensatory mutations.

- **Effect on termination:**
  - **Compensatory mutations restore base pairing** in the hairpin.
  - Since the hairpin **functions properly**, termination will occur as expected.
- **Expected transcript size:**
  - **Short transcript (150 bases)** because termination is restored.

Information Booster:

1. **Rho-independent termination** depends on the formation of a **GC-rich hairpin followed by U-rich sequences**.
2. **U-A interactions** are weaker than **G-C interactions**, making the termination site susceptible to disruption.
3. **Mutation in the U-run region** can lead to **transcription readthrough**, increasing transcript length.
4. **Disrupting the hairpin structure** prevents RNA polymerase pausing, causing **failure in termination**.
5. **Compensatory mutations** restoring the hairpin structure can recover proper termination efficiency.
6. **Mutations in bacterial terminators** can lead to **antibiotic resistance** by altering gene expression levels.

S15. Ans.(b)

Sol. B, D, E

Explanation :

Statement A: eIF2 facilitates correct recognition and binding of ribosomal subunits.

- **Incorrect.** eIF2 is **not responsible for ribosomal subunit binding**.
- Instead, **eIF2 binds GTP and Met-tRNAi (initiator tRNA), forming the ternary complex** that helps in translation initiation.
- Ribosomal subunit binding is **regulated by eIF3, eIF6, and eIF5B**.

Statement B: eIF2B activates eIF2 by replacing its GDP with GTP.

- **Correct.** eIF2 is active in its **GTP-bound form**, allowing it to deliver Met-tRNAi to the 40S ribosomal subunit.
- **eIF2B is a guanine nucleotide exchange factor (GEF)** that replaces GDP with GTP, reactivating eIF2 for another round of translation initiation.

Statement C: eIF3 binds to the 60S ribosomal subunit and inhibits its reassociation with the 40S subunit.

- **Incorrect.** eIF3 binds to the **40S ribosomal subunit**, NOT the 60S subunit.
- It **prevents premature 60S binding**, ensuring correct pre-initiation complex assembly.
- **eIF6, NOT eIF3, binds to the 60S subunit to prevent reassociation.**

Statement D: eIF5 promotes association between the 60S ribosomal subunit and the 48S complex.

- **Correct.** eIF5 functions as a **GTPase-activating protein (GAP)** that facilitates **GTP hydrolysis on eIF2**.
- This step **triggers the joining of the 60S subunit to the 48S initiation complex**, leading to the formation of the **80S ribosome**.

Statement E: eIF6 binds to the 60S ribosomal subunit and blocks reassociation with the 40S subunit.

- **Correct.** eIF6 is **a major inhibitor of ribosomal subunit joining**.
- It **binds to the 60S subunit** and prevents premature reassociation with the 40S subunit.
- eIF6 is crucial for **ribosome biogenesis** and **recycling of ribosomal subunits**.

(b) B, D, E

Information Booster:

1. **eIF2 is crucial for initiating translation** as it binds **Met-tRNAi** and delivers it to the ribosome.
2. **eIF2B functions as a nucleotide exchange factor**, converting **inactive eIF2-GDP to active eIF2-GTP**.
3. **eIF3 ensures that the 40S subunit remains free** until proper initiation factors and mRNA are recruited.
4. **eIF5 hydrolyzes GTP on eIF2**, allowing the **60S subunit to join the 48S complex**, forming the 80S ribosome.
5. **eIF6 prevents premature association** of the 40S and 60S subunits, ensuring proper ribosome recycling.

S16. Ans.(b)

Sol. A and D

Statement A: "It occurs unidirectionally, with only one replicating fork."

- Correct.
- Rolling circle replication is a unidirectional process, meaning replication proceeds in one direction around the circular DNA.
- A single replication fork is formed, and the leading strand is continuously synthesized while the other strand is displaced.

Statement B: "The E. coli ΦX174 phage uses this mechanism to replicate its double-stranded circular genome."

- Incorrect. ΦX174 phage has a single-stranded DNA genome and uses a theta replication mechanism instead of rolling circle replication for its genome synthesis.

Statement C: "E. coli utilizes this mechanism to replicate its double-stranded DNA genome."

- Incorrect.
  - E. coli uses bidirectional (theta) replication for its chromosomal DNA, not rolling circle replication.
  - The rolling circle mechanism is mainly used by bacteriophages (such as λ phage and M13), plasmids, and some viruses.

Statement D: "In λ phage, the progeny DNA may range several genomes long before it is packaged."

- Correct.
  - In bacteriophage λ, rolling circle replication produces concatemeric DNA (long continuous DNA containing multiple genomes).
  - This concatemer is then cut into genome-sized pieces before packaging into phage heads.

Statement E: "The lagging strand is not formed in the rolling circle mechanism of replication."

- Incorrect.
- In rolling circle replication, only the leading strand is continuously synthesized at the replication fork, while the displaced strand remains single-stranded temporarily.
- However, this displaced strand can later serve as a template for lagging strand synthesis
- Thus, lagging strand synthesis does occur, but it happens separately from leading strand synthesis.

Information Booster:

1. Rolling circle replication occurs in bacteriophages (e.g., λ phage, M13), plasmids, and some viruses.
2. Concatemers (long DNA molecules containing multiple genome copies) are formed during replication before being processed.
3. ΦX174 phage does NOT use rolling circle replication, as it has a single-stranded genome that undergoes theta replication.
4. E. coli does NOT use rolling circle replication; it employs theta replication for its chromosome.
5. The rolling circle mechanism generates only a leading strand, with the displaced strand either remaining single-stranded or replicated separately.

S17. Ans. (a)

Sol. A

Explanation:

Step 1: Identify the Open Reading Frame (ORF)

- The **start codon (AUG)** is the first codon that signals the beginning of translation.
- The **next (second) codon in the reading frame follows immediately after AUG**.
- The **second codon is UUG**, which **codes for the amino acid Leucine (Leu).**

Step 2: Determine the Complementary Anticodon

- **Codon-anticodon pairing follows complementary base pairing rules** (A-U, G-C).
- The anticodon must be **complementary and antiparallel** to the codon.
- The codon **UUG** (5' to 3') requires an anticodon **CAG** (3' to 5').

Information Booster:

1. **Codon-Anticodon Base Pairing:**
   - Codons are read **5' to 3'** direction.
   - Anticodons in tRNA are **complementary and antiparallel** (3' to 5').
2. **Start Codon (AUG) initiates translation** and recruits Methionine (Met).
3. **Leucine (Leu) is encoded by multiple codons:**
   - UUA, UUG, CUU, CUC, CUA, CUG
4. **tRNA Selection and Wobble Base Pairing:**
   - The third base in the codon can sometimes allow **alternative base pairing**, leading to degeneracy in the genetic code.

S18. Ans.(c)
Sol. a-ii ; b-I; c-ii; d-iii
Explanation :
1. **Palmitoylation → Membrane Anchoring (ii)**
   - Palmitoylation **adds a fatty acid (palmitate) to proteins**, making them **hydrophobic** and **anchoring them to the membrane**.
   - Commonly found in **membrane proteins and signaling molecules**.
2. **polySUMOylation → Protein Degradation (i)**
   - **SUMOylation (Small Ubiquitin-like Modifier attachment)** regulates **protein stability, degradation, and nuclear transport**.
   - It plays a role similar to **ubiquitination**, tagging proteins for degradation or cellular localization.
3. **Glycosylphosphatidylinositol (GPI) Anchor → Membrane Anchoring (ii)**
   - **GPI anchors tether proteins to the outer membrane of the plasma membrane**.
   - Essential for **signaling proteins and lipid raft-associated proteins**.
4. **Mannose-6-phosphate → Lysosomal Targeting (iii)**
   - Mannose-6-phosphate (M6P) tags proteins for **transport to lysosomes**.
   - Lysosomal hydrolases receive this modification in the **Golgi apparatus**, allowing recognition by **M6P receptors**

Information Booster:
1. **Post-translational modifications (PTMs) regulate protein function, localization, and degradation.**
2. **Palmitoylation and GPI anchoring** ensure proteins are **membrane-bound**.
3. **SUMOylation is involved in nuclear transport, transcription regulation, and proteasomal degradation.**
4. **Mannose-6-phosphate ensures proteins reach lysosomes**, preventing lysosomal storage disorders.
5. **Defects in lysosomal targeting (e.g., I-cell disease) result in severe metabolic dysfunctions.**

S19. Ans.(a)
Sol. A,B, and C
Explanation:
(A) Invasin and β1 Integrins → Zipper Mechanism
- Certain bacteria (e.g., **Yersinia spp.**) produce **invasins**, which bind **β1 integrins** on host cells.
- This **induces cytoskeletal rearrangement** leading to bacterial internalization.
- This process follows a **"zipper" mechanism**, where bacteria closely interact with the host cell membrane.

(B) Actin Polymerization and Clathrin Assembly → Zipper Mechanism
- Bacterial uptake via the **zipper mechanism** requires **actin polymerization** at the site of bacterial attachment.
- Example: **Listeria monocytogenes** and **Yersinia spp.** use this pathway.
- **Clathrin coating** is not typically involved in bacterial entry but is essential for **endocytosis**, which bacteria exploit.

(C) Type III Secretion System (T3SS) → Trigger Mechanism
- **Salmonella enterica, Shigella, and Pseudomonas** use the **trigger mechanism** for invasion.
- The **T3SS injects effector proteins into host cells**, inducing **membrane ruffling and cytoskeletal changes**.
- This results in **bacterial engulfment and internalization**.

(D) Ca^{2+} Elevation and Lysosomal Fusion → Not a Typical Entry Mechanism

- Ca^{2+} elevation plays a role in intracellular trafficking and **phagosome maturation**.
- However, **fusion with lysosomes is NOT a mechanism for bacterial invasion** into non-phagocytic cells.
- Instead, **bacteria avoid lysosomal fusion to prevent degradation**.

Information Booster:

1. **Zipper Mechanism (Used by Yersinia & Listeria):**
   - Involves **tight receptor-ligand interactions**.
   - Uses **β1 integrins & cadherins** to mediate actin polymerization.
2. **Trigger Mechanism (Used by Salmonella & Shigella):**
   - **Bacteria inject effectors via T3SS**, causing **membrane ruffling**.
   - Leads to **macropinocytosis-like uptake** of bacteria.
3. **Actin Cytoskeleton Remodeling:**
   - Required for **both zipper & trigger mechanisms**.
   - **Pathogens manipulate actin polymerization** to enter cells.
4. **Role of Calcium (Ca^{2+}) in Infection:**
   - **Intracellular Ca^{2+} regulates vesicular trafficking**, but it is **not a direct invasion strategy**.
   - Some bacteria manipulate **Ca^{2+} signaling** to disrupt host immune responses.

S20. Ans.(c)

Sol. Only D

(A) Loss of function mutation in a cAMP binding site of the PKA regulatory subunit leads to the inactivation of gene expression.

- **Correct.** The regulatory subunit of **PKA binds cAMP** to activate the **catalytic subunit**.
- If the cAMP-binding site is **mutated (loss of function)**, **PKA remains inactive**, preventing **CREB phosphorylation**, leading to **inactivation of gene expression**.

(B) Activating mutation in the GTP-binding domain of the α subunit of Gs leads to the activation of gene expression.

- **Correct. Gsα (stimulatory G protein)** activates **adenylyl cyclase**, increasing **cAMP levels**.
- A **constitutively active (mutated) Gsα** would keep **PKA active**, continuously **phosphorylating CREB** and **activating gene expression**.

(C) Inactivating mutation that prevents the regulatory subunit of PKA from binding the catalytic subunit leads to the activation of gene expression.

- **Correct.** Normally, the **regulatory subunit inhibits PKA** by binding to its **catalytic subunit**.
- If a **mutation prevents this binding**, the **catalytic subunit is always active**, leading to **constant CREB activation** and **gene expression**.

(D) Inactivating mutation in the PKA phosphorylation site of CREB leads to the activation of gene expression.

- **Incorrect.** CREB must be **phosphorylated by PKA** to activate gene transcription.
- If the **PKA phosphorylation site on CREB is mutated (inactivated)**, **CREB cannot be activated**, leading to **loss of gene expression**, not activation.

Information Booster:

1. **cAMP-PKA Pathway Activation:**
   - **Gsα activates adenylyl cyclase → cAMP production → PKA activation**.
   - Active **PKA phosphorylates CREB**, leading to **gene transcription activation**.

2. **PKA Structure and Function:**
   - PKA has **two regulatory subunits** and **two catalytic subunits**.
   - **cAMP binds the regulatory subunits**, releasing **active catalytic subunits**.
3. **CREB Phosphorylation:**
   - **PKA phosphorylates CREB at Ser133**, which **recruits CBP (CREB-binding protein)** to initiate transcription.
   - If the phosphorylation site is mutated (**D statement**), **gene activation fails**.