GATE Biotechnology

Sample Paper – 7

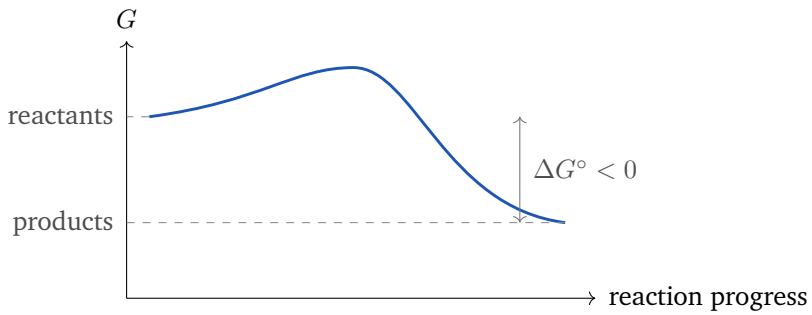
Duration: 128 Minutes **Maximum Marks: 72**

Instructions

- This paper contains **46 questions** covering the core **Biotechnology** subject of the GATE paper conducted by the IITs and IISc (General Aptitude and Engineering Mathematics are provided as separate papers).
- **Q1–Q20 carry 1 mark each** and **Q21–Q46 carry 2 marks each**, for a total of **72 marks**.
- Each question carries **negative marking of one-third of its allotted marks**: a wrong 1-mark question costs **0.33** and a wrong 2-mark question costs **0.67**. Unattempted questions carry no penalty.
- Every question here is a single-correct **MCQ**. The real GATE paper also has Multiple Select (MSQ) and Numerical Answer Type (NAT) questions, and **those carry no negative marking**.
- Attempt this paper in one timed sitting of about **128 minutes**, the share this core subject earns at GATE's overall pace of 180 minutes for 65 questions. Use $R = 8.314$ J mol^{-1}K^{-1} and take $\ln 2 = 0.693$ where required, unless stated otherwise.

Biochemistry

Q1. A biochemical reaction reaches equilibrium with an equilibrium constant $K_{eq} = 100$ at 298 K, its free-energy profile sketched below. Using $\Delta G^{\circ} = -RT \ln K_{eq}$ with $R = 8.314$ J mol^{-1}K^{-1} and $\ln 10 = 2.303$, the standard free-energy change is closest to: **[1 mark]**



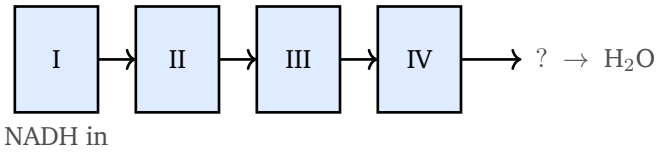
(A) -11.4 kJ mol^{-1}

(B) $+11.4$ kJ mol^{-1}

(C) -22.8 kJ mol^{-1}

(D) -5.7 kJ mol^{-1}

Q2. The mitochondrial electron transport chain passes electrons through a series of carriers, as shown, coupling the flow to proton pumping. The terminal electron acceptor at the end of the chain, which is reduced to water, is: **[1 mark]**



(A) NAD^+

(B) cytochrome c

(C) molecular oxygen (O_2)

(D) FAD

Q3. The regularly repeating α-helix and β-pleated sheet arrangements of a polypeptide backbone, stabilised mainly by hydrogen bonds, are examples of the protein's: **[1 mark]**

(A) primary structure

(B) secondary structure

(C) tertiary structure

(D) quaternary structure

Q4. A reaction at constant temperature $T = 300$ K has an enthalpy change $\Delta H = -40$ kJ mol^{-1} and an entropy change $\Delta S = +50$ J mol^{-1}K^{-1}. Using $\Delta G = \Delta H - T\Delta S$, the Gibbs free-energy change is: **[1 mark]**

(A) -25 kJ mol^{-1}

(B) $+55$ kJ mol^{-1}

(C) -40 kJ mol^{-1}

(D) -55 kJ mol^{-1}

Q5. One turn of the citric acid (Krebs) cycle reduces coenzymes that then feed electrons into the respiratory chain. The principal electron-carrying coenzyme that is reduced at three separate dehydrogenase steps in a single turn of the cycle is: **[1 mark]**

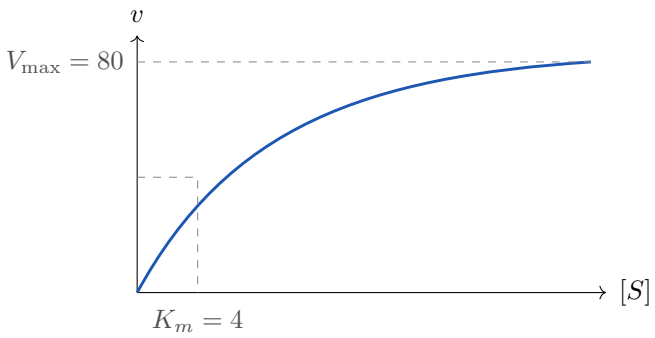
(A) NADH

(B) $FADH_2$

(C) ATP

(D) coenzyme A

Q6. An enzyme obeying Michaelis–Menten kinetics has $V_{\max} = 80$ μmol min^{-1} and $K_m = 4$ mM, as sketched. The initial velocity when the substrate concentration $[S] = 12$ mM is: **[1 mark]**



(A) 80 μmol min^{-1}

(B) 20 μmol min^{-1}

(C) $40\ \mu\text{mol min}^{-1}$

(D) $60\ \mu\text{mol min}^{-1}$

Microbiology

Q7. Penicillin, the first antibiotic to be produced industrially on a large scale, is obtained by submerged fermentation of the filamentous fungus: [1 mark]

(A) *Streptomyces griseus*

(B) *Escherichia coli*

(C) *Saccharomyces cerevisiae*

(D) *Penicillium chrysogenum*

Q8. The aminoglycoside antibiotic streptomycin, historically the first effective drug against tuberculosis, is produced industrially by the actinomycete: [1 mark]

(A) *Penicillium notatum*

(B) *Streptomyces griseus*

(C) *Bacillus subtilis*

(D) *Aspergillus niger*

Q9. Penicillin exerts its antibacterial action by inhibiting the transpeptidase (penicillin-binding protein) that cross-links the growing bacterial: [1 mark]

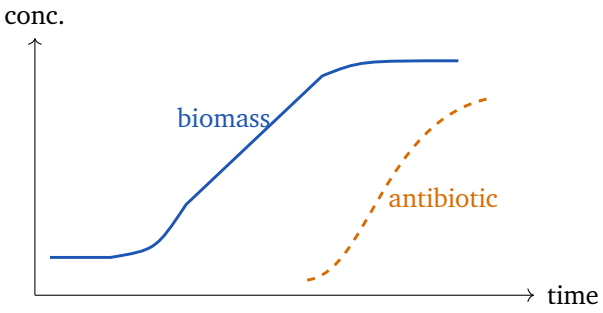
(A) cytoplasmic membrane

(B) DNA during replication

(C) ribosome during translation

(D) peptidoglycan cell wall

Q10. In a batch antibiotic fermentation the growth and product curves are shown. Antibiotics are secondary metabolites, so their maximum production rate coincides with the: [1 mark]



(A) lag phase

(B) exponential (trophophase) growth phase

(C) stationary phase (idiophase)

(D) death phase

Q11. Streptomycin, gentamicin and kanamycin all kill bacteria by binding the 30S ribosomal subunit and causing misreading of mRNA. On this basis they belong to the antibiotic class of: [1 mark]

(A) aminoglycosides

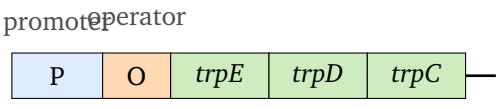
(B) β-lactams

(C) tetracyclines

(D) macrolides

Cell Biology and Molecular Biology

Q12. The *trp* operon of *E. coli*, shown below, encodes enzymes for tryptophan biosynthesis. Tryptophan acts as a co-repressor, so when it is abundant inside the cell the transcription of the operon is: [1 mark]



(A) strongly activated

(B) unaffected by tryptophan

(C) repressed (switched off)

(D) made constitutive

Q13. In the *lac* operon, the enzyme β-galactosidase converts a little lactose into the true physiological inducer, a small molecule that binds the repressor and inactivates it. This inducer is: [1 mark]

(A) lactose acting directly

(B) allolactose

(C) glucose

(D) cyclic AMP

Q14. Catabolite (glucose) repression of the *lac* operon works through the catabolite activator protein (CAP), which can bind DNA and boost transcription only after it forms a complex with a signalling molecule that accumulates when glucose is scarce. That molecule is: [1 mark]

(A) cyclic GMP

(B) ATP

(C) allolactose

(D) cyclic AMP (cAMP)

Q15. Besides repression, the *trp* operon is fine-tuned by a mechanism in which the ribosome's progress over a short leader peptide determines whether an RNA hairpin forms and prematurely terminates transcription. This second control mechanism is called: [1 mark]

(A) induction

(B) attenuation

(C) catabolite repression

(D) recombination

Q16. In eukaryotes, the large ribonucleoprotein machine that removes introns from a pre-mRNA and ligates the flanking exons together is the: **[1 mark]**

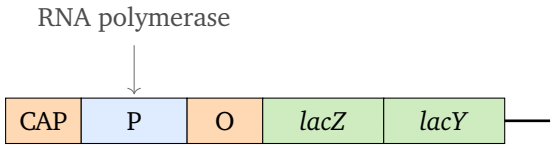
(A) spliceosome

(B) ribosome

(C) proteasome

(D) RNA polymerase II acting alone

Q17. The upstream region of the *lac* operon is drawn below. The DNA element to which RNA polymerase binds to begin transcription of the structural genes is the region marked: **[1 mark]**



(A) the operator (O)

(B) the terminator

(C) the promoter (P)

(D) a structural gene

Q18. An operon whose structural genes are transcribed only when a specific inducer molecule is present, so that the encoded enzymes appear only when their substrate is available, is described as an: **[1 mark]**

(A) repressible operon

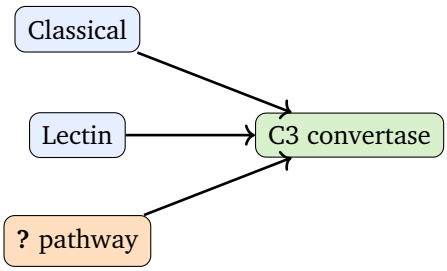
(B) constitutive operon

(C) inducible operon

(D) attenuated operon

Genetics, Immunology and Evolution

Q19. The complement system can be triggered by three converging routes, two of which are the classical and the lectin pathways, as sketched. The third route, which is antibody-independent and is initiated directly on microbial surfaces, is the: **[1 mark]**



(A) adaptive pathway

(B) alternative pathway

(C) humoral pathway

(D) cellular pathway

Q20. All three complement activation pathways converge on the cleavage of a single central component into an anaphylatoxin fragment and an opsonising fragment. This pivotal complement protein is: [1 mark]

(A) C3

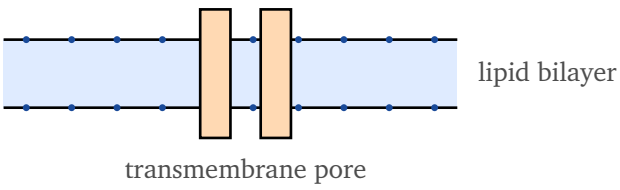
(B) C9

(C) C1q

(D) IgG

Genetics, Immunology and Evolution (continued)

Q21. The terminal steps of the complement cascade assemble several late components into a ring-shaped structure that punches a pore through the target-cell membrane, as shown, causing osmotic lysis. This pore-forming assembly is the: [2 marks]



(A) inflammasome

(B) opsonin

(C) interferon

(D) membrane attack complex (MAC, C5b–9)

Q22. Cells of the innate immune system recognise conserved pathogen-associated molecular patterns (PAMPs), such as bacterial lipopolysaccharide, using germline-encoded pattern-recognition receptors. A major family of such membrane-bound receptors is the: [2 marks]

(A) T-cell receptors (TCRs)

(B) B-cell receptors (BCRs)

(C) MHC class I molecules

(D) Toll-like receptors (TLRs)

Q23. In a large randomly mating population at Hardy–Weinberg equilibrium for a gene with two alleles, the recessive homozygous phenotype (genotype aa) occurs at a frequency of 0.09. The frequency of the dominant allele is: [2 marks]

(A) 0.30

(B) 0.70

(C) 0.09

(D) 0.49

Q24. In a test cross of a dihybrid $AaBb$ (two linked genes) with a double-recessive $aabb$ tester, 300 progeny are scored and 45 of them are recombinant types. The genetic map distance between the two loci is: [2 marks]

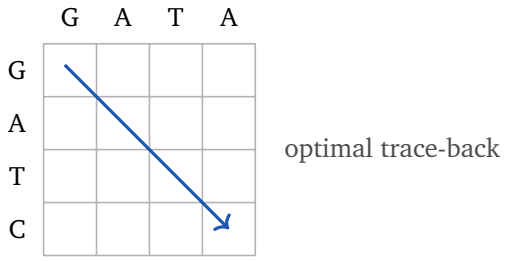
(A) 45 centimorgan

(B) 30 centimorgan

(C) 6.7 centimorgan

(D) 15 centimorgan

Q25. Two protein sequences are aligned end to end over their full lengths using a dynamic-programming matrix, filled as shown, with the best-scoring trace-back giving the optimal *global* alignment. The classical algorithm for this global alignment is: [2 marks]



(A) the Smith–Waterman algorithm

(B) the Needleman–Wunsch algorithm

(C) BLAST

(D) ClustalW

Q26. To find the best-matching *local* region shared between two sequences (for example, a common domain embedded in otherwise dissimilar proteins), the appropriate dynamic-programming algorithm, which allows the alignment to start and end anywhere, is: [2 marks]

(A) the Needleman–Wunsch algorithm

(B) the FASTA heuristic filter

(C) the Smith–Waterman algorithm

(D) a hidden Markov model

Q27. In a BLAST search report, the statistic that estimates the number of alignments scoring at least as high as the observed hit that would be expected purely by chance in a database of that size is the: [2 marks]

(A) Expect value (E-value)

(B) sum of bit scores

(C) percentage identity

(D) gap-opening penalty

Q28. When aligning two protein sequences, each aligned residue pair is scored using a substitution matrix derived from conserved blocks of related proteins, reflecting how often one amino acid is replaced by another during evolution. A widely used matrix of this type is: **[2 marks]**

(A) BLOSUM62

(B) a restriction map

(C) a phylogenetic tree

(D) the standard codon table

Q29. The restriction endonuclease *Eco*RI cleaves its palindromic recognition sequence $5'$-G$\downarrow$AATTC-$3'$ between the guanine and the adjacent adenine on each strand, leaving identical single-stranded $5'$ overhangs (sticky ends). The four-base overhang produced is: **[2 marks]**

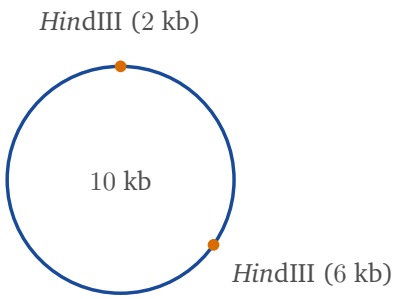
(A) $5'$-TTAA

(B) $5'$-AATT

(C) a blunt end (no overhang)

(D) $5'$-GC

Q30. The circular plasmid shown is 10 kb long and carries two *Hin*dIII recognition sites, located at the 2 kb and 6 kb positions measured around the circle. Complete digestion with *Hin*dIII yields DNA fragments of: **[2 marks]**



(A) 2 kb and 6 kb

(B) 4 kb and 6 kb

(C) 5 kb and 5 kb

(D) 10 kb only (single linearised piece)

Bioprocess Engineering and Process Biotechnology

Q31. To reuse a costly enzyme over many batches, it is confined inside the lattice of a calcium-alginate gel bead so that the enzyme is physically caged while substrate and product diffuse freely in and out. This method of immobilisation is called: **[2 marks]**

(A) covalent binding to a support

(B) adsorption onto a carrier

(C) cross-linking with glutaraldehyde

(D) entrapment in a gel matrix

Q32. Compared with simple physical adsorption, immobilising an enzyme by forming direct covalent bonds between enzyme residues and an activated solid support offers the main advantage of: **[2 marks]**

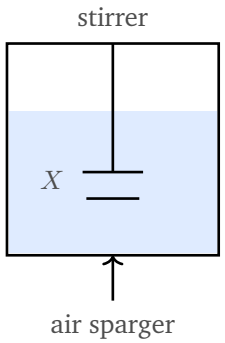
(A) leaving the enzyme's stability unchanged

(B) allowing the enzyme to leak off easily

(C) reducing how often the catalyst can be reused

(D) firm attachment with very little enzyme leakage during operation

Q33. In the batch stirred-tank bioreactor shown, an organism grows exponentially and the biomass concentration rises from 2 g L^{-1} to 16 g L^{-1} over 9 hours. The specific growth rate μ during this period is nearest to: **[2 marks]**



(A) $0.12\ \mathrm{h}^{-1}$

(B) $0.23\ \mathrm{h}^{-1}$

(C) $0.35\ \mathrm{h}^{-1}$

(D) $0.69\ \mathrm{h}^{-1}$

Q34. In a continuous chemostat, the dilution rate D is progressively increased. When D is raised above the organism's maximum specific growth rate $\mu_{\max}$, the cells can no longer replace themselves fast enough and the culture undergoes: **[2 marks]**

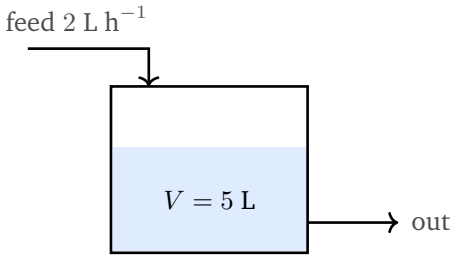
(A) sporulation

(B) washout

(C) diauxic growth

(D) flocculation

Q35. The chemostat shown is held at a constant working volume of $5\ \mathrm{L}$ and fed with sterile medium at a volumetric flow rate of $2\ \mathrm{L\,h}^{-1}$. Its dilution rate is: **[2 marks]**



(A) $2.5\ \mathrm{h}^{-1}$

(B) $0.2\ \mathrm{h}^{-1}$

(C) $0.4\ \mathrm{h}^{-1}$

(D) $10\ \mathrm{h}^{-1}$

Q36. For an immobilised-enzyme bead, the observed reaction rate can fall below the intrinsic kinetic rate when substrate cannot reach the catalyst fast enough. When the overall rate is controlled by how quickly substrate is transported to the biocatalyst rather than by the chemistry itself, the reactor is said to be: **[2 marks]**

(A) diffusion (external mass-transfer) limited

(B) reaction (kinetically) limited

(C) thermodynamically limited

(D) light limited

Q37. In a batch fermentation, the product concentration climbs to a maximum of 60 g L^{-1} at the end of 30 hours of operation. The overall volumetric productivity of the product for this batch is: **[2 marks]**

(A) 0.5 g $L^{-1}h^{-1}$

(B) 1800 g $L^{-1}h^{-1}$

(C) 2 g $L^{-1}h^{-1}$

(D) 30 g $L^{-1}h^{-1}$

Q38. For a spherical pellet of immobilised biocatalyst in which internal diffusion may limit the reaction, the effectiveness factor η is defined as the ratio of: **[2 marks]**

(A) substrate concentration to product concentration

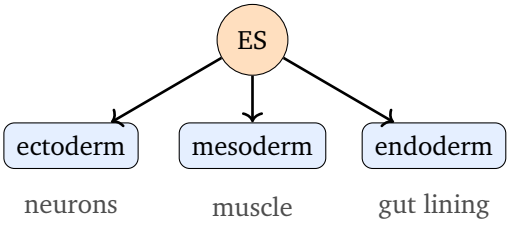
(B) K_m to $V_{\max}$

(C) the actual observed reaction rate to the rate that would occur with no diffusion limitation

(D) cell mass to substrate consumed

Plant and Animal Biotechnology

Q39. Embryonic stem cells isolated from the inner cell mass of the blastocyst can give rise to derivatives of all three germ layers, as sketched, but not to the extra-embryonic tissues. Cells with this developmental capacity are described as: **[2 marks]**



(A) pluripotent

(B) totipotent

(C) unipotent

(D) multipotent

Q40. Adult somatic cells can be reprogrammed into induced pluripotent stem cells (iPSCs) by the forced expression of a defined set of four transcription factors identified by Takahashi and Yamanaka: Oct4, Sox2, Klf4 and: [2 marks]

(A) p53

(B) Ras

(C) c-Myc

(D) β-catenin

Q41. Bone marrow contains a population of adult stem cells that continuously replenish all the circulating blood cell lineages, from erythrocytes to lymphocytes. These are the: [2 marks]

(A) hematopoietic stem cells

(B) neural stem cells

(C) cardiac progenitor cells

(D) epidermal stem cells

Q42. The first mammal successfully cloned from an adult (fully differentiated) body cell by somatic cell nuclear transfer, and thus a landmark of animal biotechnology, was: [2 marks]

(A) a laboratory mouse

(B) Dolly the sheep

(C) an African clawed frog

(D) a dairy cow

Q43. In somatic cell nuclear transfer (SCNT), reproductive cloning is achieved by transplanting the nucleus of a donor somatic cell into a recipient cell that has had its own nucleus removed. That recipient cell is a(n): [2 marks]

(A) enucleated oocyte (egg cell)

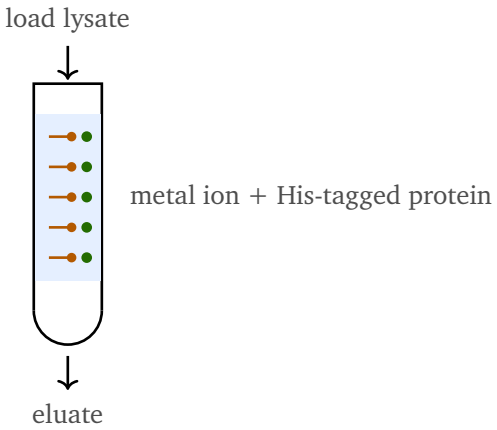
(B) mature sperm cell

(C) another intact somatic cell

(D) bacterial host cell

Analytical Techniques

Q44. A recombinant protein engineered with a tail of six histidine residues (a His_6 tag) is purified by immobilised-metal affinity chromatography on the column sketched below. The His-tag binds specifically to divalent metal ions chelated on the resin, namely: [2 marks]



(A) immobilised protein A

(B) immobilised glutathione

(C) immobilised streptavidin

(D) chelated nickel (Ni^{2+}) ions

Q45. After the His-tagged protein has bound to a nickel affinity column and the contaminants have been washed away, the pure target protein is recovered by: [2 marks]

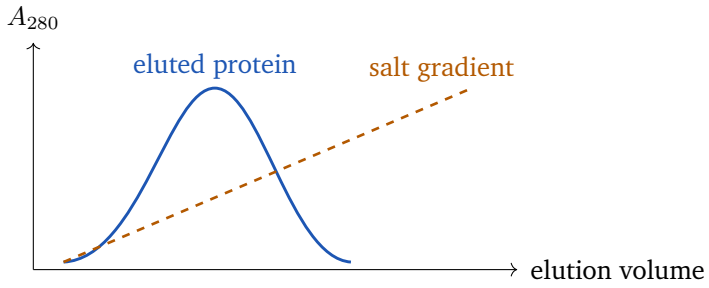
(A) adding a competing ligand such as imidazole that displaces the bound protein from the metal

(B) making the column longer to increase resolution

(C) lowering the flow rate to zero and waiting

(D) heating the packed column to $95\ ^{\circ}\text{C}$

Q46. Fast protein liquid chromatography (FPLC), whose gradient elution trace is shown, is preferred over conventional HPLC for purifying native proteins mainly because it uses: **[2 marks]**



(A) very high pressures that denature the proteins

(B) only gaseous mobile phases

(C) mild, biocompatible conditions and lower operating pressures that preserve protein activity

(D) no stationary phase at all

Detailed Solutions

Q1.

Solution

Concept – free energy and the equilibrium constant: At equilibrium the standard free-energy change is $\Delta G^\circ = -RT \ln K_{eq}$.

Step 1 – list the data: $K_{eq} = 100$, $T = 298$ K, $R = 8.314$ J mol^{-1}K^{-1}.

Step 2 – express the logarithm: $\ln 100 = \ln(10^2) = 2 \ln 10 = 2 \times 2.303 = 4.605$.

Step 3 – form the product RT: $RT = 8.314 \times 298 = 2477.6$ J mol^{-1}.

Step 4 – substitute into the equation: $\Delta G^\circ = -\,2477.6 \times 4.605$.

Step 5 – multiply: $\Delta G^\circ = -11409$ J mol^{-1}.

Step 6 – convert to kilojoules: $\Delta G^\circ \approx -11.4$ kJ mol^{-1}.

Step 7 – check the sign: $K_{eq} > 1$ means products are favoured, so ΔG° must be negative, consistent with the exergonic profile drawn.

Final Answer: $\Delta G^\circ \approx -11.4$ kJ mol^{-1} $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – the respiratory electron transport chain: Electrons from NADH and $FADH_2$ flow "downhill" through complexes I–IV toward the carrier with the most positive reduction potential.

Step 1 – trace the electron flow: NADH $\rightarrow$ Complex I $\rightarrow$ ubiquinone $\rightarrow$ Complex III $\rightarrow$ cytochrome c $\rightarrow$ Complex IV.

Step 2 – identify the last step: At Complex IV (cytochrome c oxidase) the electrons are handed to the terminal acceptor.

Step 3 – name the terminal acceptor: That acceptor is molecular oxygen, O_2, which is reduced to water: $O_2 + 4e^- + 4H^+ \rightarrow 2H_2O$.

Why the other options are wrong:

- A, NAD^+: is the *initial* electron donor's oxidised form, not the terminal acceptor.
- B, cytochrome c: is an intermediate mobile carrier between Complexes III and IV.
- D, FAD: is an early carrier (in Complex II), not the final one.

Final Answer: molecular oxygen (O_2) $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – the four levels of protein structure: Primary is the residue sequence; secondary is local hydrogen-bonded backbone folding; tertiary is the overall 3-D fold of one chain; quaternary is the assembly of several chains.

Step 1 – identify what stabilises α-helices and β-sheets: Both are held together by regular hydrogen bonds between backbone amide and carbonyl groups.

Step 2 – match to a structural level: Such locally repeating, hydrogen-bonded backbone patterns define the **secondary** structure.

Step 3 – confirm: They are neither the mere sequence (primary) nor the full 3-D fold (tertiary), so secondary structure is correct.

Why the other options are wrong:

- A, primary: is only the linear order of amino acids.
- C, tertiary: is the complete folded shape of the whole chain.
- D, quaternary: needs two or more polypeptide chains together.

Final Answer: secondary structure $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – the Gibbs free-energy relation: $\Delta G = \Delta H - T\Delta S$ combines the enthalpy and entropy contributions at a fixed temperature.

Step 1 – list the data in consistent units: $\Delta H = -40$ kJ mol^{-1} $= -40000$ J mol^{-1}, $\Delta S = +50$ J mol^{-1}K^{-1}, $T = 300$ K.

Step 2 – compute the entropy term: $T\Delta S = 300 \times 50 = 15000$ J mol^{-1}.

Step 3 – substitute into the equation: $\Delta G = -40000 - 15000$.

Step 4 – carry out the subtraction: $\Delta G = -55000$ J mol^{-1}.

Step 5 – convert to kilojoules: $\Delta G = -55$ kJ mol^{-1}.

Step 6 – interpret: Both a negative ΔH and a positive ΔS favour the process, so

ΔG is strongly negative and the reaction is spontaneous at all temperatures.

Final Answer: $\Delta G = -55$ kJ mol$^{-1} \Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – reducing power from the citric acid cycle: Each turn of the cycle strips electrons from intermediates and stores them on coenzymes that later drive oxidative phosphorylation.

Step 1 – count the reduction steps per turn: Three NAD^+ molecules are reduced (at isocitrate dehydrogenase, α-ketoglutarate dehydrogenase and malate dehydrogenase).

Step 2 – compare with the other carriers: Only one $FADH_2$ is made (at succinate dehydrogenase) and one GTP/ATP by substrate-level phosphorylation.

Step 3 – identify the principal carrier: The coenzyme reduced at three points, and the main electron carrier to the chain, is therefore **NADH**.

Why the other options are wrong:

- B, $FADH_2$: is reduced only once per turn.
- C, ATP: is an energy currency, not the electron-carrying coenzyme asked for.
- D, coenzyme A: carries acyl groups, not electrons.

Final Answer: NADH $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – the Michaelis–Menten rate law: $v = \dfrac{V_{\max}[S]}{K_m + [S]}$.

Step 1 – list the data: $V_{\max} = 80$ μmol min^{-1}, $K_m = 4$ mM, $[S] = 12$ mM.

Step 2 – substitute the values: $v = \dfrac{80 \times 12}{4 + 12}$.

Step 3 – evaluate the numerator: $80 \times 12 = 960$.

Step 4 – evaluate the denominator: $4 + 12 = 16$.

Step 5 – divide: $v = \dfrac{960}{16} = 60$ μmol min^{-1}.

Step 6 – sanity check: Since $[S] = 3K_m$, the velocity should be $\frac{3}{4}V_{\max} = 0.75 \times 80 = 60$, which agrees.

Final Answer: $v = 60\ \mu\text{mol min}^{-1} \Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – industrial production of penicillin: The original antibiotic is a fungal secondary metabolite made in large aerated fermenters.

Step 1 – recall the production organism: High-yielding industrial strains of the mould *Penicillium chrysogenum* (formerly *P. notatum*) are used.

Step 2 – confirm the class of organism: It is a filamentous fungus, matching the "filamentous fungus" wording of the question.

Why the other options are wrong:

- A, *Streptomyces griseus*: a bacterium that makes streptomycin, not penicillin.
- B, *E. coli*: a gut bacterium, not a penicillin producer.
- C, *S. cerevisiae*: baker's/brewer's yeast, used for ethanol, not penicillin.

Final Answer: *Penicillium chrysogenum* $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – source organism of streptomycin: Many clinically important antibiotics come from soil actinomycetes of the genus *Streptomyces*.

Step 1 – recall the specific producer: Streptomycin was isolated by Waksman's group from *Streptomyces griseus*.

Step 2 – confirm the organism type: *Streptomyces* are filamentous, Gram-positive actinobacteria, matching "actinomycete".

Why the other options are wrong:

- A, *Penicillium notatum*: a mould that makes penicillin.
- C, *Bacillus subtilis*: makes bacitracin/subtilin, not streptomycin.
- D, *Aspergillus niger*: an industrial source of citric acid.

Final Answer: *Streptomyces griseus* $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – mechanism of β-lactam antibiotics: Penicillin is a β-lactam that mimics the D-Ala–D-Ala terminus of peptidoglycan precursors.

Step 1 – identify the target enzyme: It irreversibly acylates the transpeptidase (a penicillin-binding protein) that cross-links glycan strands.

Step 2 – consequence for the cell: Without cross-linking, the **peptidoglycan cell wall** cannot be completed, so growing cells lyse under turgor pressure.

Step 3 – why it is selective: Human cells have no peptidoglycan wall, which is why penicillin is relatively non-toxic to us.

Why the other options are wrong:

- A, membrane: is targeted by polymyxins, not penicillin.
- B, DNA replication: is targeted by quinolones.
- C, ribosome: is the target of aminoglycosides, tetracyclines and macrolides.

Final Answer: the peptidoglycan cell wall $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – primary versus secondary metabolites: Secondary metabolites are not needed for growth and are typically produced after active division slows.

Step 1 – classify antibiotics: Antibiotics are secondary metabolites (idiolites).

Step 2 – link production to a growth phase: Their synthesis peaks when the culture leaves rapid growth and enters the **stationary phase**, historically called the idiophase.

Step 3 – read the figure: The dashed antibiotic curve rises just as the biomass curve flattens, exactly at the stationary phase.

Why the other options are wrong:

- A, lag phase: cells are only adapting; no product yet.

- B, exponential phase (trophophase): energy goes into biomass, not secondary metabolites.
- D, death phase: cells are lysing and production has ceased.

Final Answer: the stationary phase (idiophase) $\Rightarrow$ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – antibiotic classification by target and structure: Antibiotics are grouped by chemistry and mechanism (e.g. β-lactams, aminoglycosides, tetracyclines, macrolides).

Step 1 – note the shared features: Streptomycin, gentamicin and kanamycin are amino-sugars linked by glycosidic bonds and all bind the 30S ribosomal subunit, causing mRNA misreading.

Step 2 – assign the class: Amino-sugar antibiotics acting on the 30S subunit are the **aminoglycosides**.

Why the other options are wrong:

- B, β-lactams: (penicillins, cephalosporins) attack the cell wall, not the ribosome.
- C, tetracyclines: also bind 30S but are a chemically distinct class and are bacteriostatic.
- D, macrolides: (e.g. erythromycin) bind the 50S subunit.

Final Answer: aminoglycosides $\Rightarrow$ A

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – the *trp* operon is a repressible system: Its genes are normally ON and are switched OFF when the end product accumulates.

Step 1 – role of tryptophan: Tryptophan is a co-repressor; it binds the inactive *trp* aporepressor and activates it.

Step 2 – fate of the activated repressor: The tryptophan–repressor complex binds the operator and blocks RNA polymerase.

Step 3 – outcome when tryptophan is abundant: Transcription of the biosynthetic genes is therefore **repressed (switched off)**, because the cell no longer needs to make its own tryptophan.

Why the other options are wrong:

- A, activated: is the opposite of what a co-repressor does.
- B, unaffected: ignores the feedback control.
- D, constitutive: would mean expression regardless of tryptophan, which is not the case.

Final Answer: repressed (switched off) $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – induction of the *lac* operon: The operon turns on only when a specific small-molecule inducer inactivates the repressor.

Step 1 – the true inducer: A basal amount of β-galactosidase isomerises lactose into **allolactose**.

Step 2 – action of allolactose: Allolactose binds the *lac* repressor, changing its shape so it can no longer hold the operator.

Step 3 – result: The freed operator lets RNA polymerase transcribe *lacZ*, *lacY* and *lacA*.

Why the other options are wrong:

- A, lactose directly: lactose itself is a poor inducer; it must first be converted to allolactose.
- C, glucose: actually keeps the operon down through catabolite repression.
- D, cAMP: acts via CAP to boost transcription but is not the molecule that inactivates the repressor.

Final Answer: allolactose $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – catabolite repression and CAP: Positive control of the *lac* operon needs an activator that responds to the glucose level.

Step 1 – link glucose to the signal: When glucose is scarce, adenylate cyclase raises the level of **cyclic AMP (cAMP)**.

Step 2 – action of cAMP: cAMP binds CAP; the cAMP–CAP complex then binds the CAP site and helps RNA polymerase load onto the promoter.

Step 3 – outcome: Strong transcription of *lac* occurs only when glucose is low (high cAMP) and lactose is present.

Why the other options are wrong:

- A, cGMP: is not the signalling molecule for CAP in *E. coli*.
- B, ATP: is the precursor of cAMP, not the effector itself.
- C, allolactose: acts on the repressor, not on CAP.

Final Answer: cyclic AMP (cAMP) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q14

Q15.

Solution

Concept – attenuation of the *trp* operon: A second layer of control acts *after* transcription has begun, at a leader region ahead of the structural genes.

Step 1 – the leader peptide: The leader mRNA encodes a short peptide rich in tryptophan codons; a ribosome translates it as transcription proceeds.

Step 2 – coupling to RNA structure: When tryptophan (hence charged $tRNA^{Trp}$) is plentiful, the ribosome moves quickly and lets a terminator hairpin form, stopping transcription before the genes.

Step 3 – name the mechanism: This premature transcription termination controlled by translation is called **attenuation**.

Why the other options are wrong:

- A, induction: describes turning an operon on, as in *lac*.
- C, catabolite repression: is glucose-mediated CAP control.
- D, recombination: is DNA rearrangement, unrelated here.

Final Answer: attenuation $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Q16.

Solution

Concept – pre-mRNA splicing in eukaryotes: Eukaryotic genes contain introns that must be excised before translation.

Step 1 – name the machine: Splicing is done by the **spliceosome**, a complex of small nuclear ribonucleoproteins (snRNPs: U1, U2, U4, U5, U6) plus proteins.

Step 2 – what it does: It recognises the $5'$ and $3'$ splice sites and the branch point, removes each intron as a lariat, and joins the neighbouring exons.

Why the other options are wrong:

- B, ribosome: translates mRNA into protein; it does not splice.
- C, proteasome: degrades proteins.
- D, RNA polymerase II alone: transcribes the pre-mRNA but does not carry out splicing by itself.

Final Answer: the spliceosome $\Rightarrow$ A

Answer: (A) Go Back to Q16

Q17.

Solution

Concept – functional elements of an operon: Promoter, operator and structural genes each have a distinct job.

Step 1 – define the promoter: The **promoter** is the DNA sequence that RNA polymerase recognises and binds to start transcription; in the figure it is the region marked P where the polymerase is shown loading.

Step 2 – contrast with the operator: The operator (O) is a control switch bound by the repressor, not by the polymerase.

Why the other options are wrong:

- A, operator: binds the repressor, controlling access; polymerase does not initiate there.
- B, terminator: lies at the far end and stops transcription.
- D, structural gene: is transcribed, not the polymerase-binding start site.

Final Answer: the promoter (P) ⇒ C

Answer: (C) Go Back to Q17

Q18.

Solution

Concept – inducible versus repressible operons: The two categories differ in their default state and how the effector acts.

Step 1 – define an inducible operon: An inducible operon is normally OFF and is switched ON when an inducer (often the substrate) appears; the *lac* operon is the textbook example.

Step 2 – match the description: "Enzymes appear only when their substrate is available" is exactly the behaviour of an **inducible** operon.

Why the other options are wrong:

- A, repressible: is normally ON and switched OFF by a co-repressor (as in *trp*).
- B, constitutive: is expressed at a steady level regardless of the effector.
- D, attenuated: refers to a fine-tuning mechanism, not this on/off category.

Final Answer: an inducible operon ⇒ C

Answer: (C) Go Back to Q18

Q19.

Solution

Concept – the three complement activation pathways: Complement is triggered by the classical, lectin and alternative pathways, all converging on C3.

Step 1 – recall the antibody-independent route: The **alternative pathway** starts when spontaneously hydrolysed C3 deposits directly on microbial surfaces, needing no antibody.

Step 2 – match to the figure: The two labelled inputs are classical and lectin, so the unlabelled "?" input is the alternative pathway.

Why the other options are wrong:

- A, adaptive: is a branch of immunity, not a named complement pathway.
- C, humoral / D, cellular: describe arms of the immune response overall, not

complement activation routes.

Final Answer: the alternative pathway $\Rightarrow$ B

Answer: (B) Go Back to Q19

Q20.

Solution

Concept – the central component of complement: All three pathways build a C3 convertase that cleaves one pivotal protein.

Step 1 – identify the shared substrate: That protein is **C3**, cleaved into C3a (an anaphylatoxin) and C3b (an opsonin).

Step 2 – why it is central: C3b amplifies the cascade and seeds the C5 convertase, linking the early steps to the terminal pathway.

Why the other options are wrong:

- B, C9: acts late, polymerising to form the pore, not at the convergence point.
- C, C1q: is specific to the classical pathway's start.
- D, IgG: is an antibody that can trigger the classical pathway but is not a complement component.

Final Answer: C3 $\Rightarrow$ A

Answer: (A) Go Back to Q20

Q21.

Solution

Concept – the terminal (lytic) pathway of complement: The late components assemble into a membrane-spanning pore.

Step 1 – name the assembly: Components C5b, C6, C7, C8 and multiple C9 molecules form the **membrane attack complex (MAC, C5b–9)**.

Step 2 – its action: Polymerised C9 creates a transmembrane channel (the pore in the figure); water and ions rush in and the target cell lyses.

Why the other options are wrong:

- A, inflammasome: is a cytosolic complex that activates IL-1β, unrelated to the membrane pore.
- B, opsonin: (e.g. C3b) tags cells for phagocytosis but does not lyse them.

- C, interferon: is an antiviral cytokine, not a lytic complex.

Final Answer: the membrane attack complex (MAC, C5b–9) $\Rightarrow$ D

Answer: (D) Go Back to Q21

Q22.

Solution

Concept – innate recognition of pathogens: Innate immunity uses a limited set of germline-encoded receptors that read conserved microbial signatures (PAMPs).

Step 1 – name the receptor family: The best-known membrane pattern-recognition receptors are the **Toll-like receptors (TLRs)**.

Step 2 – give an example: TLR4, for instance, recognises bacterial lipopolysaccharide and triggers inflammatory signalling.

Why the other options are wrong:

- A and B, TCR / BCR: are the highly variable, somatically rearranged receptors of *adaptive* immunity.
- C, MHC class I: presents peptides to T cells; it is not a PAMP sensor.

Final Answer: Toll-like receptors (TLRs) $\Rightarrow$ D

Answer: (D) Go Back to Q22

Q23.

Solution

Concept – the Hardy–Weinberg principle: Genotype frequencies are $p^2 + 2pq + q^2 = 1$, where p is the dominant and q the recessive allele frequency.

Step 1 – link the recessive phenotype to q: The recessive homozygote aa has frequency $q^2 = 0.09$.

Step 2 – solve for q: $q = \sqrt{0.09} = 0.3$.

Step 3 – use $p + q = 1$: $p = 1 - q = 1 - 0.3$.

Step 4 – evaluate: $p = 0.7$.

Step 5 – (check): Then $p^2 = 0.49$ and $2pq = 2(0.7)(0.3) = 0.42$; the three frequencies $0.49 + 0.42 + 0.09 = 1.00$, confirming the answer.

Final Answer: $p = 0.7 \Rightarrow$ B

Answer: (B) Go Back to Q23

Q24.

Solution

Concept – recombination frequency and map distance: The recombination frequency (RF) between two linked loci, expressed as a percentage, equals the map distance in centimorgan (cM).

Step 1 – count the recombinants: Recombinant progeny $= 45$ out of a total of 300.

Step 2 – compute the recombination frequency: $\text{RF} = \dfrac{45}{300}$.

Step 3 – evaluate as a fraction: $\dfrac{45}{300} = 0.15$.

Step 4 – express as a percentage: $0.15 \times 100 = 15\%$.

Step 5 – convert to map units: 1% recombination $= 1$ cM, so the distance is 15 cM.

Final Answer: 15 centimorgan $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q24

Q25.

Solution

Concept – global versus local alignment: A *global* alignment matches two sequences over their entire lengths.

Step 1 – identify the algorithm: The dynamic-programming method that guarantees the optimal end-to-end alignment is the **Needleman–Wunsch** algorithm.

Step 2 – how it works: It fills a scoring matrix from a fixed corner using match/mismatch and gap scores, then traces back from the final cell, exactly as sketched.

Why the other options are wrong:

- A, Smith–Waterman: gives *local*, not global, alignments.
- C, BLAST: is a fast heuristic database search, not a guaranteed-optimal global aligner.
- D, ClustalW: performs *multiple* sequence alignment, not a pairwise global one.

Final Answer: the Needleman–Wunsch algorithm $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q25

Q26.

Solution

Concept – optimal local alignment: Finding the best-matching *subregions* needs an algorithm that can start and end anywhere and never lets the score go negative.

Step 1 – identify the algorithm: The **Smith–Waterman** algorithm is the dynamic-programming method for optimal local alignment.

Step 2 – key modifications from global: Negative matrix cells are reset to zero, and the trace-back starts from the highest-scoring cell (not a corner), isolating the best local match.

Why the other options are wrong:

- A, Needleman–Wunsch: aligns full lengths (global).
- B, FASTA: is a heuristic, not a guaranteed-optimal local aligner.
- D, hidden Markov model: is a probabilistic modelling framework, not this specific local-alignment algorithm.

Final Answer: the Smith–Waterman algorithm $\Rightarrow$ C

Answer: (C) Go Back to Q26

Q27.

Solution

Concept – statistical significance in BLAST: BLAST reports how likely a hit is to arise by chance.

Step 1 – define the measure: The **Expect value (E-value)** is the number of hits with a score at least as high as the observed one expected purely by chance in a database of the given size.

Step 2 – interpret it: A very small E-value (e.g. 10^{-40}) means the match is highly unlikely to be random, so it is biologically meaningful.

Why the other options are wrong:

- B, sum of bit scores: bit score is related, but the chance-expectation statistic is the E-value.
- C, percentage identity: measures similarity but not statistical significance versus database size.

- D, gap penalty: is an input scoring parameter, not an output significance measure.

Final Answer: the Expect value (E-value) $\Rightarrow$ A

Answer: (A) Go Back to Q27

Q28.

Solution

Concept – amino-acid substitution matrices: Protein alignments score residue pairs using an empirically derived substitution matrix.

Step 1 – identify the matrix: BLOSUM (BLOcks SUbstitution Matrix), e.g. BLOSUM62, is built from conserved, ungapped blocks of related proteins.

Step 2 – what it captures: Each entry reflects the log-odds of substituting one amino acid for another among proteins of a given similarity level.

Why the other options are wrong:

- B, restriction map: shows enzyme cut sites on DNA, not substitution scores.
- C, phylogenetic tree: depicts evolutionary relationships, not residue-pair scores.
- D, codon table: maps codons to amino acids, not substitution likelihoods.

Final Answer: BLOSUM62 $\Rightarrow$ A

Answer: (A) Go Back to Q28

Q29.

Solution

Concept – sticky ends from *Eco*RI: *Eco*RI makes staggered cuts, generating complementary single-stranded overhangs.

Step 1 – write the site and cut points: $5'$-G↓A A T T C-$3'$ on the top strand, and $3'$-C T T A A↑G-$5'$ on the bottom strand.

Step 2 – read the exposed single strand: Each cut leaves a $5'$ protruding end reading $5'$-AATT.

Step 3 – confirm complementarity: The two AATT overhangs are complementary, so any two *Eco*RI ends can anneal and be ligated.

Why the other options are wrong:

- A, $5'$-TTAA: is the reverse reading, not the actual $5'$ overhang.
- C, blunt end: *Eco*RI makes cohesive ends, not blunt ones.
- D, $5'$-GC: is not the overhang produced by this site.

Final Answer: $5'$-AATT $\Rightarrow$ B

Answer: (B) Go Back to Q29

Q30.

Solution

Concept – fragments from a circular restriction map: Two cuts on a circle divide it into two arcs whose lengths add up to the total.

Step 1 – list the cut positions: *Hind*III sites at the 2 kb and 6 kb marks on a 10 kb circle.

Step 2 – length of the first arc: From 2 kb to 6 kb: $6 - 2 = 4$ kb.

Step 3 – length of the second arc: The remaining arc: $10 - 4 = 6$ kb.

Step 4 – state the fragments: Two fragments of 4 kb and 6 kb.

Step 5 – check: $4 + 6 = 10$ kb, the full plasmid length, confirming the split.

Final Answer: 4 kb and 6 kb $\Rightarrow$ B

Answer: (B) Go Back to Q30

Q31.

Solution

Concept – methods of enzyme immobilisation: Immobilisation methods include adsorption, covalent binding, cross-linking and entrapment.

Step 1 – describe the caging method: Trapping enzyme molecules within the pores of a polymer gel (such as calcium-alginate beads), where the enzyme cannot escape but small substrates and products diffuse in and out, is **entrapment**.

Step 2 – why the enzyme stays put: The gel mesh is smaller than the enzyme, so it is physically retained without chemical modification of its active site.

Why the other options are wrong:

- A, covalent binding: attaches the enzyme by chemical bonds to a support, not by caging.
- B, adsorption: holds the enzyme on a surface by weak forces.
- C, cross-linking: links enzyme molecules to each other with a bifunctional

reagent.

Final Answer: entrapment in a gel matrix $\Rightarrow$ D

Answer: (D) Go Back to Q31

Q32.

Solution

Concept – advantages of covalent immobilisation: Covalent attachment forms strong chemical bonds between the enzyme and the carrier.

Step 1 – effect on retention: Because the bonds are strong, the enzyme is held firmly and does not wash off during repeated operation.

Step 2 – practical benefit: This **minimal leakage** means the immobilised catalyst can be reused for many cycles and gives a cleaner, enzyme-free product stream.

Why the other options are wrong:

- A, stability unchanged: covalent binding usually *improves* operational stability.
- B, easy leakage: is a drawback of weak adsorption, not of covalent binding.
- C, reduced reusability: the opposite is true; firm binding increases reusability.

Final Answer: firm attachment with very little enzyme leakage $\Rightarrow$ D

Answer: (D) Go Back to Q32

Q33.

Solution

Concept – specific growth rate in exponential growth: For exponential growth $X = X_0 e^{\mu t}$, so $\mu = \dfrac{1}{t}\ln\dfrac{X}{X_0}$.

Step 1 – list the data: $X_0 = 2$ g L^{-1}, $X = 16$ g L^{-1}, $t = 9$ h.

Step 2 – find the fold increase: $\dfrac{X}{X_0} = \dfrac{16}{2} = 8$.

Step 3 – take the natural log: $\ln 8 = \ln(2^3) = 3\ln 2 = 3 \times 0.693 = 2.079$.

Step 4 – divide by the time: $\mu = \dfrac{2.079}{9}$.

Step 5 – evaluate: $\mu \approx 0.231$ h^{-1}.

Step 6 – round to the nearest option: $\mu \approx 0.23$ h^{-1}.

Final Answer: $\mu \approx 0.23\ \text{h}^{-1} \Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q33

Q34.

Solution

Concept – steady state and washout in a chemostat: At steady state $\mu = D$; a stable culture is possible only while the cells can grow at least as fast as they are diluted.

Step 1 – the limiting condition: The largest growth rate the organism can reach is $\mu_{\max}$.

Step 2 – what happens when $D > \mu_{\max}$: Cells are removed faster than they can be replaced, so the biomass steadily falls.

Step 3 – name the result: The culture is progressively flushed out of the vessel: this is **washout**.

Why the other options are wrong:

- A, sporulation: is a stress response, not a dilution effect.
- C, diauxic growth: is sequential use of two substrates.
- D, flocculation: is cell aggregation, unrelated to high dilution.

Final Answer: washout $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q34

Q35.

Solution

Concept – dilution rate of a chemostat: The dilution rate is $D = \dfrac{F}{V}$, the feed flow rate divided by the working volume.

Step 1 – list the data: $F = 2\ \text{L h}^{-1}$, $V = 5\ \text{L}$.

Step 2 – substitute: $D = \dfrac{2}{5}$.

Step 3 – evaluate: $D = 0.4\ \text{h}^{-1}$.

Step 4 – check the units: $\dfrac{\text{L h}^{-1}}{\text{L}} = \text{h}^{-1}$, the correct dimension for a rate constant.

Step 5 – interpret: At steady state the organism's specific growth rate would settle at $\mu = D = 0.4\ \text{h}^{-1}$.

Final Answer: $D = 0.4\ \text{h}^{-1} \Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q35

Q36.

Solution

Concept – diffusion versus reaction control in immobilised catalysts: The observed rate for a porous biocatalyst is set by whichever step is slowest: transport of substrate or the intrinsic reaction.

Step 1 – identify the slow step here: If substrate cannot reach the enzyme fast enough, transport is rate-controlling.

Step 2 – name the regime: The system is then **diffusion (external mass-transfer) limited**, and the observed rate is lower than the intrinsic kinetics would allow.

Step 3 – practical note: Increasing stirring or reducing bead size shortens the diffusion path and can move the system toward reaction control.

Why the other options are wrong:

- B, reaction limited: is the opposite case, where chemistry, not transport, sets the rate.
- C, thermodynamically limited: refers to equilibrium, not transport.
- D, light limited: applies to photobioreactors, not this enzyme bead.

Final Answer: diffusion (external mass-transfer) limited $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q36

Q37.

Solution

Concept – overall volumetric productivity: For a batch, productivity is the total product formed divided by the batch time.

Step 1 – list the data: Final product $= 60\ \text{g L}^{-1}$, batch time $= 30\ \text{h}$ (starting from zero product).

Step 2 – write the definition: Productivity $= \dfrac{\Delta P}{\Delta t} = \dfrac{60}{30}$.

Step 3 – evaluate: $= 2\ \text{g L}^{-1}\text{h}^{-1}$.

Step 4 – check the units: $\dfrac{\text{g L}^{-1}}{\text{h}} = \text{g L}^{-1}\text{h}^{-1}$, a proper volumetric productivity.

Final Answer: $2\ \text{g L}^{-1}\text{h}^{-1} \Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q37

Q38.

Solution

Concept – effectiveness factor of a biocatalyst pellet: The effectiveness factor quantifies how much internal diffusion limitation lowers the rate.

Step 1 – state the definition: $\eta = \dfrac{\text{actual observed reaction rate}}{\text{rate with no diffusion limitation (surface conditions everywh}}$

Step 2 – interpret its range: $\eta \leq 1$; a value near 1 means little diffusion limitation, while a small η means the interior is starved of substrate.

Why the other options are wrong:

- A, substrate/product ratio: is a conversion measure, not the effectiveness factor.
- B, $K_m/V_{\max}$: is a kinetic parameter ratio, unrelated to the pellet effectiveness.
- D, cell mass/substrate: is the yield coefficient $Y_{X/S}$, a different quantity.

Final Answer: actual rate ÷ rate without diffusion limitation $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q38

Q39.

Solution

Concept – developmental potency of stem cells: Potency describes the range of cell types a stem cell can form.

Step 1 – describe embryonic stem cells: They come from the inner cell mass and can form all cell types of the three germ layers (ectoderm, mesoderm, endoderm), as the figure shows.

Step 2 – assign the term: Cells able to make all embryonic lineages but *not* the extra-embryonic (placental) tissues are **pluripotent**.

Why the other options are wrong:

- B, totipotent: can also form extra-embryonic tissue and a whole organism (e.g. the zygote); ES cells are one notch below this.
- C, unipotent: makes only a single cell type.

- D, multipotent: makes several related cell types within one lineage (e.g. blood cells), fewer than a pluripotent cell.

Final Answer: pluripotent $\Rightarrow$ A

Answer: (A) Go Back to Q39

Q40.

Solution

Concept – Yamanaka reprogramming factors: Four transcription factors can revert adult cells to a pluripotent state.

Step 1 – recall the set: The classic Yamanaka factors are Oct4, Sox2, Klf4 and **c-Myc**.

Step 2 – role of c-Myc: c-Myc broadly opens chromatin and boosts proliferation, raising reprogramming efficiency (though it is oncogenic, prompting safer alternatives).

Why the other options are wrong:

- A, p53: actually *blocks* reprogramming; suppressing it helps, so it is not an inducing factor.
- B, Ras: is a signalling GTPase, not one of the four factors.
- D, β-catenin: is a Wnt-pathway effector, not part of the core cocktail.

Final Answer: c-Myc $\Rightarrow$ C

Answer: (C) Go Back to Q40

Q41.

Solution

Concept – adult stem cells of the blood system: Blood cells are short-lived and must be replenished from a resident stem-cell pool.

Step 1 – name the stem cell: The bone-marrow **hematopoietic stem cells (HSCs)** give rise to all blood lineages.

Step 2 – their range: They generate both the myeloid line (red cells, platelets, granulocytes, monocytes) and the lymphoid line (B, T and NK cells); they are multipotent.

Why the other options are wrong:

- B, neural stem cells: make neurons and glia, not blood cells.
- C, cardiac progenitor cells: give rise to heart cells.
- D, epidermal stem cells: renew the skin.

Final Answer: hematopoietic stem cells $\Rightarrow$ A

Answer: (A) Go Back to Q41

Q42.

Solution

Concept – reproductive cloning by SCNT: Somatic cell nuclear transfer produces an animal genetically identical to the nucleus donor.

Step 1 – recall the landmark: Dolly the sheep (1996, Roslin Institute) was the first mammal cloned from an adult somatic (mammary gland) cell.

Step 2 – significance: Dolly proved that a differentiated nucleus can be reprogrammed by an egg's cytoplasm to make a whole organism.

Why the other options are wrong:

- A, mouse / D, cow: were cloned by SCNT only *after* Dolly, not first.
- C, frog: nuclear-transfer work in amphibians (Gurdon) preceded Dolly, but the first cloned *mammal* from an adult cell was Dolly.

Final Answer: Dolly the sheep $\Rightarrow$ B

Answer: (B) Go Back to Q42

Q43.

Solution

Concept – the recipient cell in SCNT: Nuclear transfer replaces an egg's own genetic material with a donor nucleus.

Step 1 – prepare the recipient: An oocyte (egg) has its own nucleus removed, giving an **enucleated oocyte** with intact reprogramming cytoplasm.

Step 2 – carry out the transfer: The donor somatic nucleus is inserted; the reconstructed egg is activated and begins to divide as an embryo.

Why the other options are wrong:

- B, sperm cell: is a gamete, not the enucleated recipient used in SCNT.

- C, another somatic cell: lacks the egg-cytoplasm factors needed to reprogram the nucleus.
- D, bacterial host cell: cannot support mammalian development.

Final Answer: an enucleated oocyte (egg cell) $\Rightarrow$ A

Answer: (A) Go Back to Q43

Q44.

Solution

Concept – immobilised-metal affinity chromatography (IMAC): A His-tag binds tightly to transition-metal ions held on a chelating resin.

Step 1 – identify the immobilised ligand: The resin carries a chelator (such as NTA) loaded with divalent **nickel (Ni^{2+})** ions.

Step 2 – the binding chemistry: The imidazole side chains of the six histidines coordinate the Ni^{2+}, so only the tagged protein is retained while untagged proteins flow through.

Why the other options are wrong:

- A, protein A: binds the Fc region of antibodies, not His-tags.
- B, glutathione: is the ligand for GST-tagged proteins.
- C, streptavidin: binds biotin (or biotinylated targets), not His-tags.

Final Answer: chelated nickel (Ni^{2+}) ions $\Rightarrow$ D

Answer: (D) Go Back to Q44

Q45.

Solution

Concept – specific elution in affinity chromatography: A bound protein is released by disrupting the specific interaction that holds it.

Step 1 – the elution strategy for IMAC: Adding **imidazole**, which itself coordinates Ni^{2+}, competes with the histidine residues and displaces the His-tagged protein.

Step 2 – practical execution: A rising imidazole gradient elutes the target as a sharp peak; lowering the pH is an alternative that weakens the His–Ni^{2+} bond.

Why the other options are wrong:

- B, longer column: improves resolution but does not release bound protein.
- C, zero flow: merely stops the run; the protein stays bound.
- D, heating to $95\ ^\circ$C: would denature the protein and is never used to elute it.

Final Answer: add a competing ligand (imidazole) to displace the protein $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q45

Q46.

Solution

Concept – FPLC versus HPLC for proteins: FPLC was designed specifically to purify fragile biomolecules while keeping them active.

Step 1 – the operating conditions: FPLC uses **mild, biocompatible** aqueous buffers, hydrophilic media and comparatively **low pressures**, so native proteins keep their structure and function.

Step 2 – the trade-off with HPLC: HPLC runs at much higher pressures and often uses organic solvents suited to small molecules, conditions that can denature proteins.

Why the other options are wrong:

- A, very high pressures that denature: describes HPLC, the opposite of FPLC's gentle approach.
- B, gaseous mobile phases: that is gas chromatography, not FPLC.
- D, no stationary phase: FPLC is column chromatography and does use a stationary phase.

Final Answer: mild, biocompatible, lower-pressure conditions that preserve activity $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q46

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	C	3	B	4	D	5	A
6	D	7	D	8	B	9	D	10	C
11	A	12	C	13	B	14	D	15	B
16	A	17	C	18	C	19	B	20	A
21	D	22	D	23	B	24	D	25	B
26	C	27	A	28	A	29	B	30	B
31	D	32	D	33	B	34	B	35	C
36	A	37	C	38	C	39	A	40	C
41	A	42	B	43	A	44	D	45	A
46	C								