TIFR GS Biology

Sample Paper – 4

Duration: 180 Minutes **Maximum Marks: 80**

Instructions

- This paper contains **60 Multiple Choice Questions** (single correct answer) modelled on the **JGEEBILS** (TIFR Graduate School Biology) entrance examination.
- Questions are in **4 sections**: General, Physics, Chemistry, and Biology. Each section has **Sub-section A** (10 questions, 1 mark each) and **Sub-section B** (5 questions, 2 marks each).
- **Sub-section A:** $+1$ for correct answer; -0.5 for incorrect answer; 0 for unattempted.
- **Sub-section B:** $+2$ for correct answer; **no negative marking**; 0 for unattempted.
- Only **one** option is correct per question. Read all options before answering.
- Syllabus: UG-level Biology, Physics, Chemistry, and General Science/Mathematics. No fixed topic list — questions test conceptual understanding, data reasoning, and experimental interpretation.
- Personal calculators, mobile phones, and electronic gadgets are strictly prohibited. Rough sheets will be provided.

GENERAL — Sub-section A (Q1–Q10: $+1$ mark each; -0.5 per wrong)

Q1. Consider the dataset: $\{4,\ 7,\ 7,\ 9,\ 11,\ 13\}$. What is the **mode** of this dataset?

(A) 4

(B) 9

(C) 8.5

(D) 7

Q2. A shopkeeper buys an article for Rs 800, marks it up by 50%, and then offers a 20% discount on the marked price. What is the shopkeeper's profit percentage?

(A) 20%

(B) 15%

(C) 25%

(D) 30%

Q3. Five friends A, B, C, D, and E stand in a row. The following constraints hold: B is immediately to the left of C; D is at one end; E is between A and B. If D is at the *leftmost* position, what is C's position from the left?

(A) 2nd

(B) 3rd

(C) 4th

(D) 5th

Q4. In the 3×3 grid below, find the missing number (marked $?$):

3	5	7
4	6	8
5	7	?

(A) 6

(B) 9

(C) 8

(D) 10

Q5. What is the angle (in degrees) between the hour hand and the minute hand of a clock at **3:30**?

(A) 90°

(B) 60°

(C) 85°

(D) 75°

Q6. In a group of 500 people, 40% are males. Of the males, 30% are graduates. How many male graduates are there in the group?

(A) 100

(B) 150

(C) 60

(D) 200

Q7. Identify the next term in the series: $2,\ 3,\ 5,\ 7,\ 11,\ 13,\ \_\_$

(A) 17

(B) 15

(C) 14

(D) 19

Q8. Complete the analogy: *Catabolism : Degradation* **::** *Anabolism :* _____

(A) Respiration

(B) Biosynthesis

(C) Fermentation

(D) Excretion

Q9. You have 9 coins that look identical; one is slightly lighter (fake). Using a balance scale (not a spring balance), what is the *minimum* number of weighings guaranteed to identify the fake coin?

(A) 4

(B) 1

(C) 3

(D) 2

Q10. Today is Wednesday. What day of the week will it be **100 days** from today?

(A) Monday

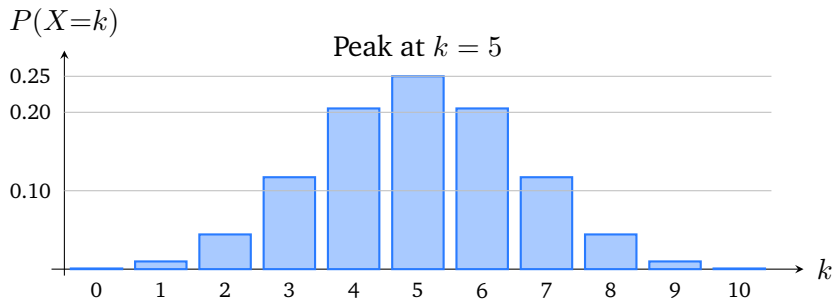
(B) Tuesday

(C) Friday

(D) Saturday

GENERAL — Sub-section B (Q11–Q15: $+2$ marks each; no negative marking)

Q11. The bar chart below shows the probability distribution $P(X{=}k)$ for the binomial random variable $X \sim B(n{=}10,\ p{=}0.5)$, where $P(X{=}k) = \binom{10}{k}(0.5)^{10}$.



For $X \sim B(10,\ 0.5)$, what is the **mean** $\mu = np$?

(A) 2

(B) 5

(C) 7

(D) 10

Q12. In simple linear regression $\hat{y} = a{+}bx$, the slope is given by $b = r\dfrac{s_y}{s_x}$, where r is the Pearson correlation coefficient, s_y is the standard deviation of y, and s_x is the standard deviation of x. If $r = 0.8$, $s_y = 15$, and $s_x = 6$, what is the value of b?

(A) 2.0

(B) 0.32

(C) 3.0

(D) 0.5

Q13. The standard error of the mean (SEM) quantifies how precisely the sample mean estimates the population mean. It is defined as $\text{SEM} = \frac{s}{\sqrt{n}}$, where s is the sample standard deviation and n is the sample size. If $s = 20$ and $n = 100$, what is the SEM?

(A) 20

(B) 10

(C) 0.20

(D) 2

Q14. A researcher compares the mean response times of two independent groups (Group 1 and Group 2) using a two-sample independent t-test. Which of the following correctly states the **null hypothesis** (H_0) for this test?

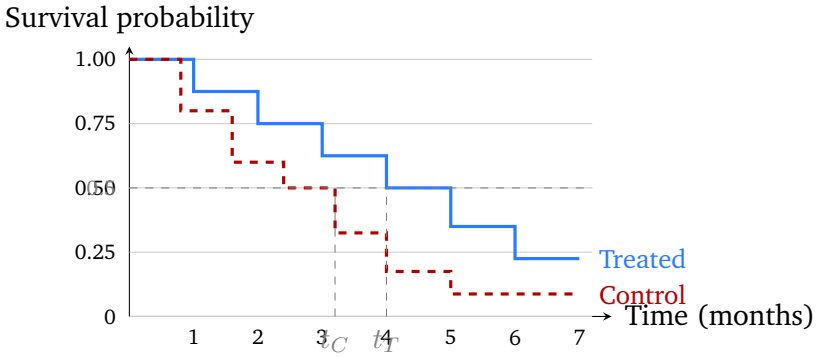
(A) $H_0 : \mu_1 > \mu_2$

(B) $H_0 : \mu_1 \neq \mu_2$

(C) $H_0 : \mu_1 = \mu_2$

(D) $H_0 : \sigma_1^2 = \sigma_2^2$

Q15. The figure shows Kaplan–Meier (KM) survival curves for a *treated* group (solid line) and a *control* group (dashed line) in a clinical study.



At the time where each survival curve crosses the horizontal line at $S(t) = 0.5$, what quantity is being directly estimated for each group?

(A) Mean survival time

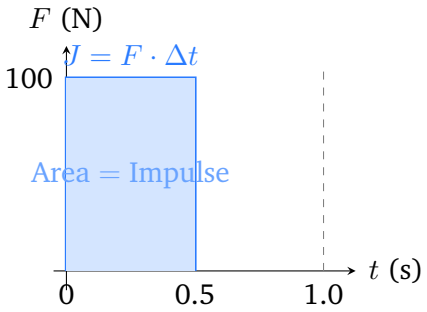
(B) Median survival time

(C) Hazard ratio between groups

(D) 95% confidence interval for survival

PHYSICS — Sub-section A (Q16–Q25: $+1$ mark each; -0.5 per wrong)

Q16. The F–t (force vs. time) graph below shows a constant force $F = 100\,\text{N}$ acting on a 2 kg ball that starts from rest, for a duration of 0.5 s. The area under the curve equals the impulse $J = \Delta p$. What is the speed of the ball at $t = 0.5\,\text{s}$?



(A) 25 m/s

(B) 50 m/s

(C) 10 m/s

(D) 5 m/s

Q17. A solid sphere of mass $m = 2\,\text{kg}$ rolls without slipping along a flat surface with a centre-of-mass speed $v = 5\,\text{m/s}$. For a solid sphere, the moment of inertia is $I = \frac{2}{5}mr^2$ and the rolling condition gives $\omega = v/r$. What is the **total kinetic energy** (translational + rotational)?

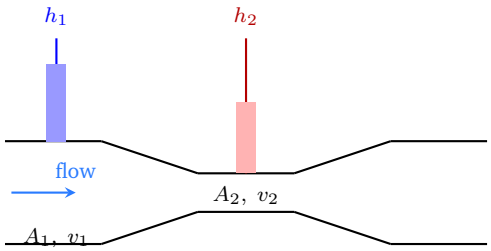
(A) 25 J

(B) 17.5 J

(C) 50 J

(D) 35 J

Q18. The figure shows a horizontal Venturi meter: a wide pipe of cross-sectional area $A_1 = 4\,\text{cm}^2$ narrows to a throat of area $A_2 = 1\,\text{cm}^2$. The water speed at the wide section is $v_1 = 1\,\text{m/s}$. Using the continuity equation $A_1 v_1 = A_2 v_2$, what is the water speed v_2 at the throat?



(A) 1 m/s (unchanged)

(B) 2 m/s

(C) 4 m/s

(D) 16 m/s

Q19. An electric dipole with dipole moment $p = 1.0 \times 10^{-30}\,\text{C m}$ is placed in a uniform electric field $E = 1.0 \times 10^{5}\,\text{N/C}$. The angle between the dipole moment vector and the field is $\theta = 30^\circ$. What is the magnitude of the torque $\tau = pE\sin\theta$ acting on the dipole?

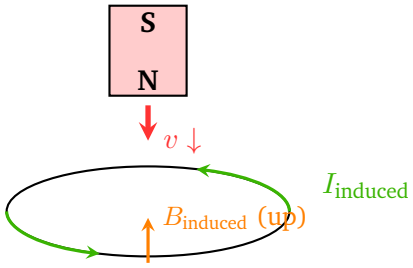
(A) $1.0 \times 10^{-25}\,\text{N m}$

(B) $5.0 \times 10^{-26}\,\text{N m}$

(C) $\sqrt{3} \times 10^{-25}\,\text{N m}$

(D) $1.0 \times 10^{-26}\,\text{N m}$

Q20. The figure shows a bar magnet (north pole downward) moving *toward* and *into* a conducting coil. The induced current direction is shown (counterclockwise when viewed from above), creating a magnetic field that opposes the increasing flux through the coil.



Coil (viewed from above)

Which law determines the **direction** of the induced current, stating that it must oppose the change in magnetic flux?

(A) Lenz's law

(B) Biot–Savart law

(C) Ampere's circuital law

(D) Coulomb's law

Q21. A light ray travels from glass (refractive index $n_1 = 1.5$) to air (refractive index $n_2 = 1.0$). Using Snell's law, the critical angle θ_c for total internal reflection satisfies $\sin\theta_c = n_2/n_1$. What is the critical angle for this glass–air interface?

(A) 30°

(B) 60°

(C) 90°

(D) $\approx 41.8^\circ \quad [\arcsin(2/3)]$

Q22. In a Wheatstone bridge, four resistors P, Q, R, and S are connected in a diamond arrangement. The galvanometer shows zero deflection (null condition) when $P/Q = R/S$. If $P = 4\,\Omega$, $Q = 8\,\Omega$, and $R = 6\,\Omega$, find the value of S for bridge balance.

(A) $3\,\Omega$

(B) $6\,\Omega$

(C) $12\,\Omega$

(D) $24\,\Omega$

Q23. Bragg's law for X-ray diffraction states $2d\sin\theta = m\lambda$. For crystal planes with interplanar spacing $d = 0.20\,\text{nm}$ and X-rays of wavelength $\lambda = 0.10\,\text{nm}$, calculate the angle θ for the *first-order* maximum ($m = 1$).

(A) $\approx 14.5^\circ$ $\quad[\sin\theta = 0.25]$

(B) $\approx 30^\circ$

(C) $\approx 45^\circ$

(D) $\approx 90^\circ$

Q24. The nucleus ${}^4_2\text{He}$ contains 2 protons and 2 neutrons. Using the atomic mass data: proton mass $= 1.0073\,\text{u}$, neutron mass $= 1.0087\,\text{u}$, and measured ${}^4\text{He}$ mass $= 4.0026\,\text{u}$ (where $1\,\text{u} \approx 931.5\,\text{MeV}/c^2$), calculate the nuclear **binding energy** of ${}^4\text{He}$.

(A) $\approx 7.1\,\text{MeV}$

(B) $\approx 27.4\,\text{MeV}$

(C) $\approx 56.5\,\text{MeV}$

(D) $\approx 2.2\,\text{MeV}$

Q25. A proton ($m_p = 1.67 \times 10^{-27}\,\text{kg}$) has kinetic energy $E_k = 1\,\text{MeV} = 1.6 \times 10^{-13}\,\text{J}$. Using the de Broglie relation $\lambda = h/p$ with $p = \sqrt{2m_pE_k}$ and $h = 6.63 \times 10^{-34}\,\text{J s}$, what is the de Broglie wavelength of this proton?

(A) $\approx 2.9\,\text{nm}$

(B) $\approx 0.29\,\text{pm}$

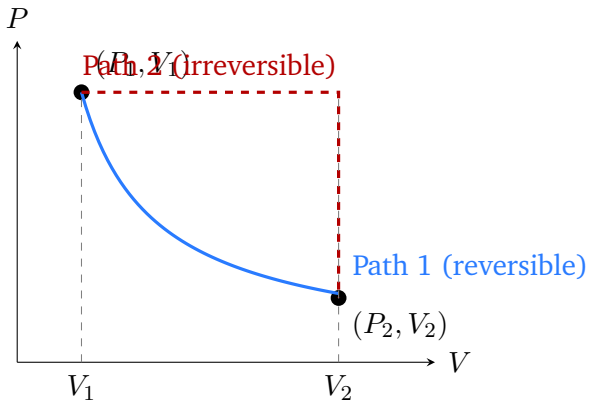
(C) $\approx 2.9\,\text{pm}$

(D) $\approx 2.9 \times 10^{-14}\,\text{m}$ $\quad(\approx 29\,\text{fm})$

PHYSICS — Sub-section B (Q26–Q30: +2 marks each; no negative marking)

Q26. The P–V diagram below shows two paths connecting the *same* initial state (P_1, V_1) to the *same* final state (P_2, V_2): Path 1 is a smooth reversible curve; Path 2 is an irreversible step-function (isobaric then isochoric step). The entropy change $\Delta S = S_{\text{final}} - S_{\text{initial}}$ is a *state function*

(path-independent), but entropy *generated* ($\sigma_{\text{gen}} \geq 0$) depends on the process.



For which path is entropy *generated* ($\sigma_{\text{gen}} > 0$)?

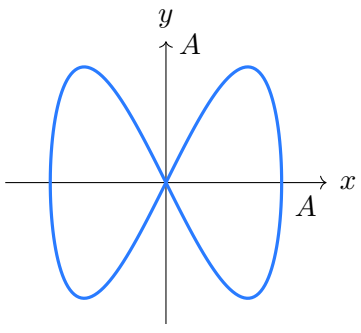
(A) Path 1 only (reversible processes generate entropy)

(B) Both paths generate equal entropy because ΔS is the same

(C) Path 2 only (irreversible processes generate entropy; reversible processes have $\sigma_{\text{gen}} = 0$)

(D) Neither path generates entropy; all entropy change comes from heat exchange

Q27. A Lissajous figure is formed on an oscilloscope by applying $x(t) = A\sin(\omega t)$ to the x-channel and $y(t) = A\sin(2\omega t)$ to the y-channel (frequency ratio $\omega_x : \omega_y = 1 : 2$, i.e., the y-component oscillates twice as fast). The parametric curve is shown below.



ω_x:ω_y = 1:2

If both x and y components have the same amplitude and the frequency ratio is $\omega_x : \omega_y = 1 : 2$, what shape does the Lissajous figure trace?

(A) A straight diagonal line (ratio 1:1, zero phase difference)

(B) A figure-eight (two loops, one above and one below the x-axis)

(C) A circle (ratio 1:1, 90° phase difference)

(D) An ellipse (ratio 1:1, intermediate phase difference)

Q28. In a Newton's rings experiment, a plano-convex lens is placed on a flat glass plate. The air film between them has zero thickness at the centre (point of contact). When illuminated from above with monochromatic light, the central spot of the ring pattern is *dark*. What is the primary reason for this central dark spot?

(A) A phase shift of π (half-wavelength) occurs upon reflection at the denser glass surface (lower surface), causing destructive interference even at zero path difference

(B) The central point is dark because the lens absorbs all incident light at the point of contact

(C) Maximum constructive interference occurs at the centre due to equal path lengths of both reflected rays

(D) The air gap refractive index ($n = 1$) causes total internal reflection, blocking light at the centre

Q29. Two coils share magnetic flux. When the current in coil 1 changes at a rate $\dfrac{dI_1}{dt} = 5.0\,\text{A/s}$, the induced EMF in coil 2 is measured to be $\mathcal{E}_2 = 0.50\,\text{V}$. Using the relation $\mathcal{E}_2 = M\dfrac{dI_1}{dt}$, what is the mutual inductance M?

(A) 2.5 H

(B) 1.0 H

(C) 0.5 H

(D) 0.1 H

Q30. The quantum-mechanical transmission probability T for a particle tunneling through a rectangular potential barrier of height V_0 (with $V_0 > E$) and width d is approximately $T \propto e^{-2\kappa d}$, where $\kappa = \sqrt{2m(V_0 - E)}/\hbar$.

What happens to T if the barrier width is **doubled** (while V_0, E, and m are kept constant)?

(A) T is halved (decreases by factor 2)

(B) T is reduced to zero (no transmission through a doubled barrier)

(C) T decreases exponentially: the new $T' \approx T^2$ (since $e^{-2\kappa(2d)} = (e^{-2\kappa d})^2$)

(D) T is unchanged because κ adjusts to compensate

CHEMISTRY — Sub-section A (Q31–Q40: $+1$ mark each; -0.5 per wrong)

Q31. Cyclohexanone reacts with *meta*-chloroperoxybenzoic acid (m-CPBA) under mild conditions. This reaction (Baeyer–Villiger oxidation) inserts an oxygen atom adjacent to the carbonyl group. What is the *product*?

(A) Cyclohexanol (by reduction of the carbonyl)

(B) ε-Caprolactone (7-membered ring lactone; oxygen inserted between C1 and C6)

(C) Cyclohexane-1,2-diol (by epoxidation of the ring double bond)

(D) Cyclohex-2-enone (by α,β-dehydrogenation)

Q32. When an alkyl or aryl halide (R-X) reacts with magnesium metal in dry diethyl ether, it forms a Grignard reagent (R-MgX). In what capacity does the organic fragment (R) act in subsequent reactions?

(A) As a carbanion equivalent (strong nucleophile), attacking electrophilic carbonyl carbons

(B) As an electrophile (carbocation), seeking electron-rich centres

(C) As a radical species, initiating chain reactions

(D) As a Lewis acid, coordinating to lone pair donors

Q33. According to Pearson's Hard-Soft Acid-Base (HSAB) principle, hard Lewis acids preferentially bind hard bases, and soft acids prefer soft bases. Cr^{3+} is classified as a *hard* Lewis acid. Which of the following ligands, all of which could potentially coordinate to Cr^{3+}, is a **hard** Lewis base that Cr^{3+} would preferentially choose over I^-?

(A) I^- (iodide; soft base — large, polarisable)

(B) SCN^- binding via S (thiocyanate-S; borderline soft)

(C) CN^- (cyanide; soft π-acid ligand)

(D) F^- (fluoride; small, non-polarisable, hard base)

Q34. The osmotic pressure π of a dilute solution is given by the van 't Hoff equation: $\pi = iMRT$, where i is the van 't Hoff factor, M is molarity (mol L^{-1}), $R = 0.0821\,\text{L atm mol}^{-1}\,\text{K}^{-1}$, and T is temperature in Kelvin. Calculate π for a 0.10 M glucose solution (non-electrolyte; $i = 1$) at $T = 300\,\text{K}$.

(A) 0.246 atm

(B) 24.6 atm

(C) 2.46 atm

(D) 0.082 atm

Q35. The Tyndall effect is the visible scattering of a beam of light by colloidal particles (diameter 1–100 nm) in a dispersion medium. Which of the following solutions or dispersions would *most clearly* exhibit the Tyndall effect when a laser beam is directed through it?

(A) A clear aqueous NaCl solution (ionic, particle size $< 1\,\text{nm}$; true solution)

(B) A freshly prepared starch-in-water suspension (colloidal dispersion, particle $\sim 10\,\text{nm}$)

(C) Distilled water (no dissolved species to scatter light)

(D) A dilute HCl solution (strong acid, completely dissociated; true solution)

Q36. The complex $[Co(NH_3)_6]^{3+}$ has Co^{3+} (d^6) with NH_3 as a strong-field ligand. In an octahedral crystal field, the d-orbitals split into t_{2g} (lower, $-4\,Dq$) and e_g (upper, $+6\,Dq$). For a d^6 **low-spin** complex, what is the electron configuration and the **crystal field stabilisation energy (CFSE)**?

(A) $t_{2g}^6\, e_g^0$; CFSE$= -24\,Dq$ (6 electrons each at $-4\,Dq$)

(B) $t_{2g}^4\, e_g^2$; CFSE$= -8\,Dq$ (high-spin weak-field)

(C) $t_{2g}^3\, e_g^3$; CFSE$= 0$ (no stabilisation)

(D) $t_{2g}^5\, e_g^1$; CFSE$= -16\,Dq$

Q37. In an electroplating cell used to deposit a layer of copper onto an iron object, the iron object is connected to the negative terminal of the power supply. Which role does the iron object play in the electrolytic cell?

(A) Anode — iron is oxidised, losing electrons to deposit copper

(B) Electrolyte — iron conducts ions between anode and cathode

(C) Salt bridge — iron maintains electrical neutrality during electrolysis

(D) Cathode — copper ions (Cu^{2+}) from solution are reduced and deposited onto iron

Q38. In isoelectric focusing (IEF), proteins migrate through an immobilised pH gradient (IPG) gel under an electric field until each protein reaches the pH at which it carries **zero net charge**. This pH is called the isoelectric point (pI). At the pI, a protein

(A) carries maximum positive charge and migrates toward the cathode at highest speed

(B) carries maximum negative charge and migrates toward the anode at highest speed

(C) carries zero net charge, stops migrating, and is focused into a sharp band

(D) precipitates irreversibly and cannot be recovered for further analysis

Q39. Sucrose (specific rotation $[\alpha]_D^{25} = +66.5^\circ$, dextrorotatory) is hydrolysed in aqueous acid to yield an equimolar mixture of glucose ($[\alpha]_D^{25} = +52.5^\circ$) and fructose ($[\alpha]_D^{25} = -92.4^\circ$). The resulting mixture is *levorotatory*. What is this glucose–fructose mixture called?

(A) Reducing sugar

(B) Invert sugar

(C) Maillard product

(D) Lactulose

Q40. Cyclopropane has internal C–C–C bond angles of approximately 60°, significantly smaller than the ideal tetrahedral angle of 109.5° for sp^3 carbon. This angle strain destabilises the molecule. Compared to cyclohexane (strain-free chair conformation), cyclopropane is

(A) More reactive, undergoing ring-opening reactions with Br_2, HBr, and H_2 that cyclohexane does not

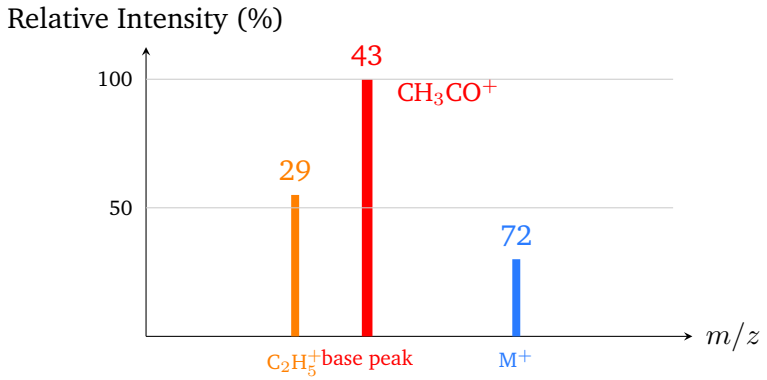
(B) Less reactive because the small ring is geometrically rigid and protected from attack

(C) More stable because eclipsing strain is minimised in a three-membered ring

(D) Unreactive toward electrophiles since it lacks π electrons

CHEMISTRY — Sub-section B (Q41–Q45: +2 marks each; no negative marking)

Q41. The schematic mass spectrum below shows the key peaks observed for 2-butanone (methyl ethyl ketone, $CH_3COCH_2CH_3$, MW = 72 Da). The molecule undergoes α-cleavage on either side of the carbonyl group.



Which fragment gives the **base peak** in the mass spectrum of 2-butanone, and what is its structure?

(A) $m/z = 72$: molecular ion M^+ (intact molecule minus one electron)

(B) $m/z = 29$: ethyl carbocation ($C_2H_5^+$) from loss of CH_3CO

(C) $m/z = 57$: loss of CH_3 from M^+, giving $[M - 15]^+$

(D) $m/z = 43$: acylium ion (CH_3CO^+) formed by α-cleavage losing the ethyl group

Q42. In quantum chemistry, solving the full Schrödinger equation for a molecule is enormously simplified by one key approximation: because electrons ($m_e \approx 9.1 \times 10^{-31}$ kg) are far lighter than nuclei (proton $m_p \approx 1.67 \times 10^{-27}$ kg, i.e., $m_p/m_e \approx 1836$), electrons adjust almost instantaneously to any displacement of the nuclei. Therefore, nuclear and electronic motions can be treated as *separable*. This assumption is called the

(A) Hartree–Fock approximation

(B) Franck–Condon principle

(C) Born–Oppenheimer approximation

(D) Variational principle

Q43. The Nernst equation at 25°C is $E = E^\circ - \dfrac{0.0592}{n} \log_{10} Q$. For the zinc half-cell $Zn^{2+}(aq) + 2e^- \rightarrow Zn(s)$ with $E^\circ = -0.76\,\text{V}$ and $[Zn^{2+}] = 0.010\,\text{M}$ (so $Q = [Zn^{2+}] = 0.010$ and $n = 2$), what is the electrode potential E?

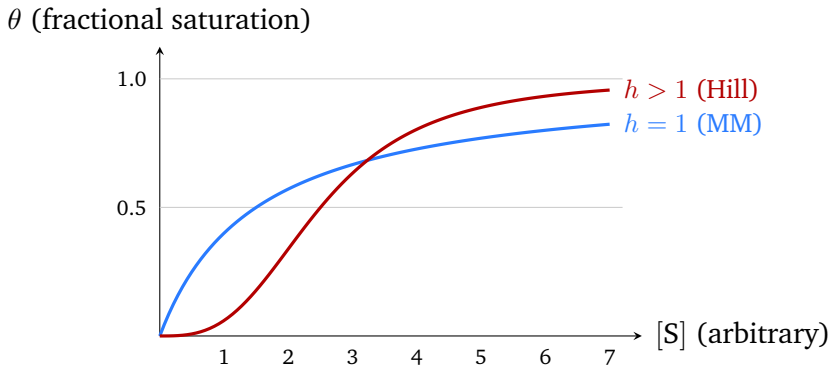
(A) $-0.82\,\text{V}$

(B) $-0.70\,\text{V}$

(C) $-0.76\,\text{V}$ (unchanged from standard)

(D) $-0.64\,\text{V}$

Q44. The figure compares two binding curves: a *hyperbolic* Michaelis–Menten curve (Hill coefficient $h = 1$, non-cooperative) and a *sigmoidal* Hill curve ($h > 1$, cooperative binding), both plotted as fractional saturation θ vs. substrate concentration $[S]$.



Which curve shows **cooperative binding**, and what does a Hill coefficient $h > 1$ indicate?

(A) The sigmoidal (red) curve; $h > 1$ indicates positive cooperativity — binding of one ligand increases the affinity of remaining sites

(B) The hyperbolic (blue) curve; $h > 1$ indicates multiple independent binding sites

(C) Both curves; $h > 1$ indicates the substrate is at saturating concentration

(D) Neither curve; cooperative binding always produces a straight line on a Hill plot

Q45. In affinity chromatography, a ligand with specific affinity for the target protein is covalently immobilised on a resin. A mixture of proteins is passed through the column; only proteins with structural affinity for the immobilised ligand are retained, while all others pass through (flow-through). The bound protein is then eluted by adding excess free ligand or altering buffer conditions. What *property* of the target protein does affinity chromatography fundamentally exploit?

(A) Molecular size (MW) — larger proteins elute first from gel-filtration resin

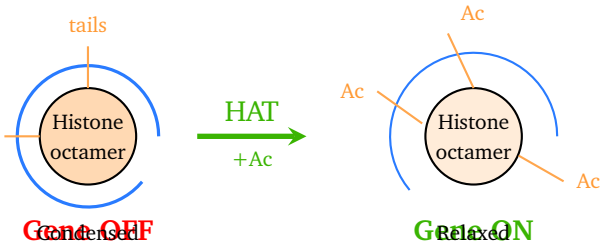
(B) Net charge — proteins bind or elute based on the isoelectric point and buffer pH

(C) Hydrophobicity — proteins retained by hydrophobic interaction with non-polar resin

(D) Specific and reversible binding affinity for a particular ligand (biochemical recognition)

BIOLOGY — Sub-section A (Q46–Q55: $+1$ mark each; -0.5 per wrong)

Q46. The schematic below illustrates chromatin remodeling by histone acetyltransferase (HAT). HAT transfers acetyl groups ($-COCH_3$) from acetyl-CoA to lysine residues on histone N-terminal tails.



What is the direct effect of histone acetylation by HAT on **chromatin structure** and **gene expression**?

(A) Acetylation increases positive charge on histones, tightening DNA binding and silencing genes

(B) Acetylation promotes DNA methylation at CpG islands, causing heterochromatin formation

(C) Acetylation neutralises positive charges on histone tails, relaxing chromatin and facilitating transcription

(D) Acetylation recruits histone deacetylases (HDACs), condensing chromatin and repressing transcription

Q47. In the RNA interference (RNAi) pathway, exogenous or endogenous double-stranded RNA (dsRNA) is processed by the endonuclease **Dicer** into short interfering RNA (siRNA, ≈ 21 nt duplexes). The siRNA duplex is then loaded into the RNA-Induced Silencing Complex (RISC); one strand (guide strand) directs RISC to complementary mRNA sequences. What is the specific role of the **Argonaute** protein within RISC?

(A) Argonaute unwinds the siRNA duplex and exports it from the nucleus

(B) Argonaute is the catalytic "Slicer" component that endonucleolytically cleaves the target mRNA complementary to the guide strand

(C) Argonaute methylates the 5' cap of target mRNAs, preventing their translation

(D) Argonaute recruits Dicer to degrade dsRNA into progressively smaller fragments

Q48. During the *elongation* phase of translation, the ribosome contains three tRNA-binding sites: the A-site (aminoacyl), P-site (peptidyl), and E-site (exit). Immediately after peptide bond formation (catalysed by peptidyl transferase activity of the large ribosomal subunit), what occupies the **P-site**?

(A) The peptidyl-tRNA bearing the growing polypeptide chain attached to the tRNA at the P-site codon

(B) The incoming aminoacyl-tRNA (aa-tRNA) delivered by EF-Tu $\cdot$ GTP

(C) A deacylated (empty) tRNA just before it exits to the E-site

(D) The release factor RF1 or RF3 recognising a stop codon

Q49. During meiosis II in a cell that had undergone normal meiosis I, the sister chromatids of *one* chromosome fail to separate (non-disjunction in meiosis II). Considering all four products from that meiosis, what are the types of gametes produced?

(A) All four gametes are aneuploid $(n+1)$

(B) Two disomic $(n+1)$ and two nullisomic $(n-1)$ gametes — consistent with meiosis I non-disjunction

(C) All four gametes are normal (n)

(D) Two normal (n) gametes, one disomic $(n+1)$, and one nullisomic $(n-1)$ gamete

Q50. Intracellular vesicle trafficking requires precise fusion of vesicle membranes with target membranes. SNARE (**S**oluble **N**SF **A**ttachment protein **RE**ceptor) proteins play an essential role in this process. Which of

the following **correctly** describes the mechanism?

(A) v-SNAREs on the vesicle form a homotypic complex with other v-SNAREs to generate the fusion force

(B) SNAREs act as calcium sensors that trigger exocytosis only in neuronal synapses

(C) v-SNAREs on the vesicle pair with complementary t-SNAREs on the target membrane to form a tight trans-SNARE complex, pulling the membranes close enough to fuse

(D) NSF (ATPase) directly catalyses membrane fusion by forming a pore between vesicle and target

Q51. In *Escherichia coli*, the SOS response is a global DNA-damage response that upregulates > 40 genes involved in DNA repair, mutagenesis, and cell cycle arrest. A key sensor is the RecA protein. What molecular signal **activates** RecA to initiate the SOS response?

(A) Binding of the LexA repressor to RecA's SOS box promotes its co-protease activity

(B) Accumulation of single-stranded DNA (ssDNA) at stalled replication forks, which RecA coats to form a nucleoprotein filament — the activated co-protease form

(C) UV-induced thymine dimers directly bind the RecA active site and activate its kinase domain

(D) Double-strand breaks activate the MRN complex, which in turn phosphorylates RecA at Ser^{345}

Q52. In mice, the agouti yellow allele (A^y) is dominant for yellow coat colour but causes embryonic lethality when homozygous (A^y/A^y). A cross between two yellow heterozygous mice ($A^y/a \times A^y/a$) is performed. Among the *surviving* offspring, why do we observe a **2:1 ratio** of yellow:agouti mice instead of the expected 3:1 Mendelian ratio?

(A) The A^y allele has incomplete penetrance in the heterozygous state, causing some yellow mice to appear agouti

(B) The a/a offspring die perinatally due to a linked lethal mutation, leaving fewer agouti survivors

(C) The cross violates the law of segregation because A^y is a semi-dominant allele

(D) The A^y/A^y homozygous class dies in utero (embryonic lethal), removing 1/4 of expected offspring from the surviving pool

Q53. ATM (Ataxia Telangiectasia Mutated) is a serine/threonine protein kinase that acts as a master sensor and signal transducer in the DNA damage response (DDR). What specific type of DNA lesion primarily and directly activates ATM kinase?

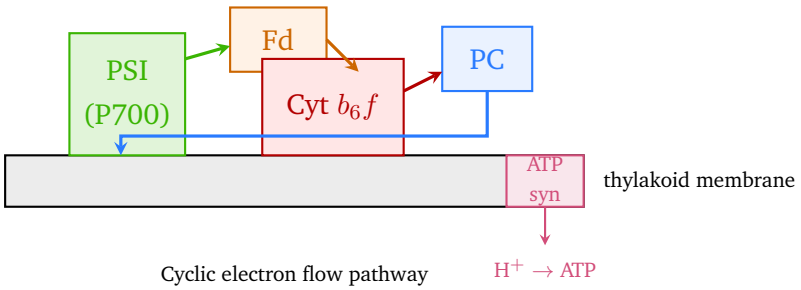
(A) DNA double-strand breaks (DSBs), sensed via the MRN complex (MRE11–RAD50–NBS1) that recruits and activates ATM at the break site

(B) UV-induced cyclobutane pyrimidine dimers (CPDs) recognised by the XPC-RAD23B complex

(C) Alkylation damage (e.g., O^6-methylguanine) repaired by MGMT methyltransferase

(D) Mismatched base pairs during replication, sensed by MutSα (MSH2-MSH6)

Q54. The diagram below shows **cyclic electron flow** in the chloroplast thylakoid membrane. Unlike linear (non-cyclic) electron flow, *no* PSII is involved, *no* water is oxidised, and *no* NADPH is produced.



What is the **net product** of cyclic electron flow?

(A) NADPH only (electrons reduce Fd→FNR→$NADP^+$)

(B) Both ATP and NADPH (same products as linear electron flow, but no O_2 evolution)

(C) ATP only (electrons cycle back to P700 via Cyt b_6f without reducing $NADP^+$)

(D) O_2 and ATP (water is oxidised as the electron donor)

Q55. During *primary succession* on a newly exposed bare rock surface (e.g., after a volcanic lava flow), ecological communities develop progressively from an abiotic substrate. Which organisms are typically the **pioneer** species that initiate this process?

(A) Grasses and annual herbs, which require minimal soil to establish

(B) Lichens (mutualism of fungi and photosynthetic algae/cyanobacteria) and/or mosses, which can colonise bare rock and begin soil formation

(C) Shrubs and perennial woody plants that rapidly invade open habitats

(D) Deciduous trees that form the eventual climax community on the bare rock substrate

BIOLOGY — Sub-section B (Q56–Q60: +2 marks each; no negative marking)

Q56. Single-cell RNA sequencing (scRNA-seq) enables transcriptome profiling at single-cell resolution. In droplet-based platforms (e.g., 10x Genomics Chromium), each cell is co-encapsulated with a bead carrying oligonucleotide tags composed of (i) a cell barcode (identifying the cell) and (ii) a **Unique Molecular Identifier (UMI)** — a short random nucleotide sequence. After reverse transcription and PCR amplification, what is the primary purpose of the **UMI**?

(A) The UMI identifies which cell the RNA originated from (cell demultiplexing)

(B) The UMI serves as a primer for reverse transcription of poly-A RNA into cDNA

(C) The UMI marks the 5' end of each cDNA for strand-specific sequencing library construction

(D) The UMI allows computational deduplication of PCR amplicons: reads sharing the same UMI from the same cell originate from a single original mRNA molecule, enabling accurate transcript counting

Q57. A somatic cell is heterozygous (Aa) at a tumour suppressor locus, where the wild-type allele is A (functional) and a is a loss-of-function mutation. During mitosis, a reciprocal recombination event occurs between the locus and the centromere (mitotic crossing over). After segregation, one daughter cell can become homozygous aa — losing the remaining wild-type allele. This phenomenon is called **loss of heterozygosity (LOH)**. By what mechanism does LOH occur in this scenario?

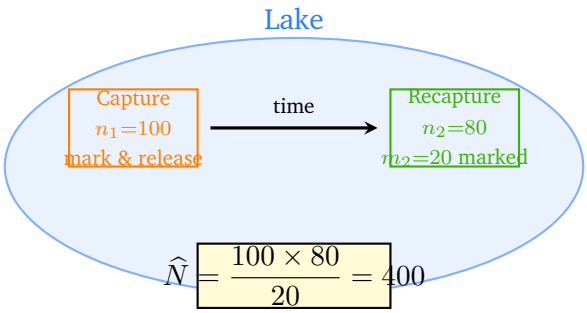
(A) Mitotic recombination (somatic crossing over) between the locus and the centromere generates aa daughter cells

(B) Meiotic non-disjunction producing aneuploid cells with only the a-bearing chromosome

(C) Transcriptional silencing of A by promoter CpG methylation (epigenetic LOH)

(D) Frameshift mutation in A introduced by error-prone DNA polymerase during replication stress

Q58. A wildlife biologist uses the Lincoln–Petersen mark-recapture method to estimate the population size of fish in a lake. In the **first** sampling event, $n_1 = 100$ fish are captured, individually marked (e.g., with a fin clip), and released. In the **second** sampling event (after sufficient time for mixing), $n_2 = 80$ fish are captured, of which $m_2 = 20$ are found to bear marks from the first event. The population estimate is $\widehat{N} = n_1 n_2 / m_2$.



Using the Lincoln–Petersen method, what is the estimated fish population $\widehat{N}$?

(A) 160

(B) 200

(C) 400

(D) 800

Q59. mTORC1 (mechanistic Target of Rapamycin Complex 1) is a master regulator of cell anabolism and growth. When activated (e.g., by amino acids, growth factors via PI3K–Akt, and high energy status), mTORC1 directly phosphorylates two key effector proteins to stimulate mRNA translation and ribosome biogenesis. Which pair of substrates is **directly** phosphorylated by active mTORC1?

(A) Akt (Ser^{473}) and AMPK (Thr^{172}) — activating the entire PI3K pathway

(B) S6K1 (p70 ribosomal S6 kinase) and 4E-BP1 (eIF4E-binding protein 1) — promoting cap-dependent translation

(C) eIF2α and eIF4G — inducing integrated stress response (ISR) and selective translation

(D) p53 (Ser^{15}) and MDM2 — initiating cell cycle arrest and apoptosis

Q60. During an adaptive immune response, activated B cells in germinal centres undergo **somatic hypermutation** — the introduction of point mutations at a rate $\sim 10^6$-fold above background specifically in the variable (V) regions of immunoglobulin heavy and light chain genes. This gener-

ates B cell clones with diverse antigen-binding affinity, enabling **affinity maturation**. Which enzyme **catalyses** somatic hypermutation?

(A) RAG1/RAG2 recombinase — the endonuclease that cuts at recombination signal sequences (RSS) during V(D)J recombination

(B) Terminal deoxynucleotidyl transferase (TdT) — adds random nucleotides (N-additions) at V(D)J junctions

(C) Poly-η (DNA polymerase eta) — error-prone polymerase that directly introduces the mutations during replication

(D) Activation-Induced Cytidine Deaminase (AID) — deaminates cytosine to uracil in ssDNA regions of V genes, initiating the mutagenic cascade

Detailed Solutions

Solution

Concept — Mode vs. Mean:
The *mode* is the value occurring most frequently. The mean ($\bar{x}$) is the arithmetic average.

Step 1 — Tally frequencies:
Dataset: $\{4,\ 7,\ 7,\ 9,\ 11,\ 13\}$. Value 7 appears **twice**; all others appear once. Mode $= 7$.

Step 2 — Compute mean to distinguish (A)–(C):
$\bar{x} = (4+7+7+9+11+13)/6 = 51/6 = 8.5$.

Why other options are wrong:

Q1.

- **(A)** 4 — appears once; not the mode.
- **(B)** 9 — appears once; not the mode.
- **(C)** 8.5 — this is the arithmetic *mean*, not the mode.

Final Answer: Mode $= 7 \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 1

Solution

Concept — Successive Percentage Changes:
Markup applies to cost price; discount applies to the marked price. They operate on different bases, so net profit $\neq 50\% - 20\%$.

Step 1 — Marked price:
MP $= 800 \times 1.50 =$ Rs 1200.

Step 2 — Selling price after 20% discount:
SP $= 1200 \times 0.80 =$ Rs 960.

Step 3 — Profit percentage on cost:
$$\text{Profit\%} = \frac{960 - 800}{800} \times 100 = \frac{160}{800} \times 100 = \mathbf{20\%}.$$

Why other options are wrong:

Q2.

- **(B)** 15% — incorrect; SP would need to be Rs 920.
- **(C)** 25% — incorrect; SP would need to be Rs 1000.
- **(D)** 30% — naïve subtraction of percentages ($50\% - 20\%$); ignores different base prices.

Final Answer: Profit% $= 20\% \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 2

Solution

Concept — Constraint-Based Linear Arrangement:
Apply constraints iteratively to find the unique valid arrangement.

Step 1 — Fix D at position 1 (leftmost).

Step 2 — Use "E is between A and B" and "B is to the left of C":
Try D(1), B(2), E(3), C(4), A(5). Check constraints:

Q3.

- B(pos 2) is to the left of C(pos 4) ✓
- E(pos 3) lies between B(pos 2) and A(pos 5): $2 < 3 < 5$ ✓
- D at leftmost ✓

All three constraints are satisfied. **C is at position 4.**

Why other options are wrong:

- **(A)** 2nd — position 2 is occupied by B in the valid arrangement.
- **(B)** 3rd — position 3 is occupied by E.
- **(D)** 5th — position 5 is occupied by A.

Final Answer: C is at the 4th position $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 3

Solution

Concept — Pattern Recognition in a Number Grid:
Identify the rule governing rows and columns independently.

Step 1 — Column pattern: Each column increases by 1 going down. Column 1: 3, 4, 5 $(+1)$. Column 2: 5, 6, 7 $(+1)$. Column 3: 7, 8, ? (should be $8 + 1 = 9$).

Step 2 — Row pattern (cross-check): Each row increases by 2 moving right. Row 3: 5, 7, ? $\Rightarrow ? = 7 + 2 = 9$. Both rules agree.

Why other options are wrong:

Q4.

- **(A)** 6 — only holds if the column decreases, which contradicts the ascending pattern.
- **(C)** 8 — off by one; misidentifies the column increment as 0.
- **(D)** 10 — correct for row increment of 3, not 2.

Final Answer: Missing number $= 9 \Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 4

Solution

Concept — Clock Angle Formula:
At time $H{:}M$, the angle between hands $= |30H - 5.5M|$ degrees (take the smaller

of the angle and $360° - \text{angle}$).

Step 1 — Apply formula for 3:30:
$\theta = |30 \times 3 - 5.5 \times 30| = |90 - 165| = 75°$.

Step 2 — Verify: hour hand at 3:30: Hour hand moves $0.5°$/min. At 3:30, it is at $90 + 15 = 105°$ from 12. Minute hand is at $180°$. Angle $= 180 - 105 = 75°$ ✓.

Why other options are wrong:

Q5.

- **(A)** $90°$ — angle at exactly 3:00, not 3:30 (hour hand has moved).
- **(B)** $60°$ — ignores the $15°$ advance of the hour hand.
- **(C)** $85°$ — arithmetic error.

Final Answer: Angle $= 75° \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 5

Solution

Concept — Sequential Percentage Calculation:
Apply percentages step by step.

Step 1 — Number of males: $500 \times 0.40 = 200$ males.

Step 2 — Male graduates: $200 \times 0.30 = 60$ male graduates.

Why other options are wrong:

Q6.

- **(A)** 100 — takes 30% of 500 instead of 30% of males only.
- **(B)** 150 — takes 30% of 500 and adds an erroneous correction.
- **(D)** 200 — is the total number of males, not graduates.

Final Answer: Male graduates $= 60 \Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 6

Solution

Concept — Prime Number Sequence:
The series lists consecutive prime numbers: numbers divisible only by 1 and themselves.

Step 1 — List primes after 13: 14 is divisible by 2; 15 by 3 and 5; 16 by 2; **17** is prime ($17/2, 17/3, 17/5, 17/7$ — none exact).

Sequence: 2, 3, 5, 7, 11, 13, **17**.

Why other options are wrong:

Q7.

- **(B)** 15 — divisible by 3 and 5; composite.
- **(C)** 14 — divisible by 2; composite.
- **(D)** 19 — prime, but is the *next* prime after 17, not the immediate next in

the sequence.

Final Answer: Next prime $= 17 \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 7

Solution

Concept — Scientific Analogy (Metabolism):
Catabolism breaks down complex molecules (degradation) to release energy. Anabolism does the opposite: it builds complex molecules from simpler ones (biosynthesis), consuming energy.

Analogy: Catabolism $\rightarrow$ Degradation; therefore Anabolism $\rightarrow$ **Biosynthesis**.

Why other options are wrong:

Q8.

- **(A)** Respiration — a catabolic process, not the opposite of degradation.
- **(C)** Fermentation — an anaerobic catabolic pathway; breaks down sugars.
- **(D)** Excretion — elimination of waste products; unrelated to biosynthesis.

Final Answer: Anabolism : Biosynthesis $\Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 8

Solution

Concept — Ternary Search / Balance Scale Divide-and-Conquer:
Each balance weighing has 3 outcomes (left side heavier, right side heavier, balanced), so n weighings can distinguish among 3^n possibilities.

Step 1 — Calculate: With 9 coins, one lighter: $3^1 = 3$ (insufficient for 9 coins with 1 weighing alone if coins were distinguishable), but $3^2 = 9$ possibilities exactly. With **2 weighings**: divide 9 into groups of 3. Weigh group 1 vs group 2; if balanced, the fake is in group 3. Then weigh 2 coins from the identified group; if balanced, the remaining unweighed coin is fake. **2 weighings suffice and are necessary.**

Why other options are wrong:

Q9.

- **(A)** 4 — far more than needed.
- **(B)** 1 — one weighing gives only 3 outcomes; insufficient to pinpoint among 9 coins.
- **(C)** 3 — would be needed for 27 coins; excessive for 9.

Final Answer: Minimum weighings $= 2 \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 9

Solution

Concept — Modular Arithmetic (Days of the Week):
Days repeat every 7 days. Find the remainder when 100 is divided by 7, then advance from Wednesday.

Step 1 — Compute $100 \mod 7$**:**
$100 = 14 \times 7 + 2$, so $100 \mod 7 = 2$.

Step 2 — Advance 2 days from Wednesday:
Wednesday $\xrightarrow{+1}$ Thursday $\xrightarrow{+1}$ **Friday**.

Why other options are wrong:

Q10.
- **(A)** Monday — would require remainder 5 ($100 \mod 7 = 5$; incorrect).
- **(B)** Tuesday — would require remainder 6.
- **(D)** Saturday — would require remainder 3.

Final Answer: Day after 100 days = Friday $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 10

Solution

Concept — Binomial Distribution Mean:
For $X \sim B(n, p)$, the mean is $\mu = np$ and the variance is $\sigma^2 = np(1-p)$. The distribution is symmetric about μ when $p = 0.5$.

Step 1 — Apply formula:
$\mu = np = 10 \times 0.5 = \mathbf{5}$.

Step 2 — Confirm from chart: The bar chart shows the peak at $k = 5$, consistent with the mean and mode coinciding for a symmetric binomial with $p = 0.5$.

Why other options are wrong:

Q11.
- **(A)** 2 — corresponds to np if $p = 0.2$.
- **(C)** 7 — corresponds to np if $p = 0.7$.
- **(D)** 10 — equals n, not np; would require $p = 1$.

Final Answer: $\mu = np = 5 \Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 11

Solution

Concept — Regression Slope:
In simple linear regression, $b = r \cdot \frac{s_y}{s_x}$ relates slope to correlation and standard deviations.

Step 1 — Substitute values:

$b = 0.8 \times \dfrac{15}{6} = 0.8 \times 2.5 = \mathbf{2.0}$.

Why other options are wrong:

Q12.

- **(B)** 0.32 — computes $r \times (s_x/s_y)$ (inverted ratio): $0.8 \times 6/15 = 0.32$.
- **(C)** 3.0 — uses r as multiplied by s_y alone: $15/5 = 3$; incorrect formula.
- **(D)** 0.5 — $s_x/s_y = 6/15 = 0.4$; does not match any plausible substitution.

Final Answer: $b = 2.0 \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 12

Solution

Concept — Standard Error of the Mean (SEM):
SEM $= s/\sqrt{n}$ decreases with larger samples, reflecting improved precision of the mean estimate.

Step 1 — Substitute:
$\text{SEM} = \dfrac{20}{\sqrt{100}} = \dfrac{20}{10} = \mathbf{2}$.

Why other options are wrong:

Q13.

- **(A)** 20 — is just s; forgets to divide by $\sqrt{n}$.
- **(B)** 10 — uses $\sqrt{n}$ in denominator but takes $\sqrt{n} = \sqrt{10}$ (wrong n).
- **(C)** 0.20 — divides by n rather than $\sqrt{n}$.

Final Answer: SEM $= 2 \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 13

Solution

Concept — Null Hypothesis for Two-Sample t-Test:
The two-sample independent t-test tests whether two population means are equal. The null hypothesis represents "no effect" — no difference between groups.

Step 1 — State H_0:
$H_0 : \ \mu_1 = \mu_2$ (there is no difference between the two population means).

Step 2 — State H_1:
$H_1 : \ \mu_1 \neq \mu_2$ (two-tailed) or directional (one-tailed).

Why other options are wrong:

Q14.

- **(A)** $\mu_1 > \mu_2$ — this is a one-tailed *alternative* hypothesis, not the null.
- **(B)** $\mu_1 \neq \mu_2$ — this is the alternative hypothesis H_1 for a two-tailed test.
- **(D)** $\sigma_1^2 = \sigma_2^2$ — this is the assumption of the Levene test of equal variances, not the t-test null.

Final Answer: $H_0 : \mu_1 = \mu_2 \Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 14

Solution

Concept — Kaplan-Meier Survival Analysis:
A KM survival curve is a step function estimating $S(t) = P(\text{survival} > t)$. The curve starts at $S(0) = 1$ and drops at each observed event (death/failure) time.

Step 1 — Identify the crossing of $S(t) = 0.5$:
When the KM curve crosses $S(t) = 0.5$ (the 50% survival level), the corresponding time t is the **median survival time** — the time by which half the subjects have experienced the event.

Step 2 — Biological meaning:
The treated group crosses $S = 0.5$ at a later time (t_T) than the control (t_C), meaning the treated group has a longer median survival.

Why other options are wrong:

Q15.

- **(A)** Mean survival time — requires integrating the area under the KM curve; not read directly from the $S = 0.5$ crossing.
- **(C)** Hazard ratio — computed separately (e.g., log-rank test or Cox model), not read from the curve crossing.
- **(D)** 95% confidence interval — shown as bands around the KM curve, not the central estimate.

Final Answer: The quantity estimated is the *median* survival time $\Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 15

Solution

Concept — Impulse-Momentum Theorem:
Impulse $J = F \cdot \Delta t =$ change in momentum $\Delta p = m \cdot \Delta v$. For a constant force, the impulse equals the area of the rectangular F–t graph.

Step 1 — Compute impulse:
$J = F \cdot \Delta t = 100\,\text{N} \times 0.5\,\text{s} = 50\,\text{N s}$.

Step 2 — Find final velocity (starting from rest):
$\Delta p = m\,v_f - m\,(0) \Rightarrow v_f = J/m = 50/2 = \mathbf{25\,m/s}$.

Why other options are wrong:

Q16.

- **(B)** 50 m/s — forgets to divide by m; takes $J = v_f$ directly.
- **(C)** 10 m/s — uses F/m (acceleration) as speed, ignoring Δt.
- **(D)** 5 m/s — divides acceleration ($50\,\text{m/s}^2$) by Δt again unnecessarily.

Final Answer: $v_f = 25\,\text{m/s} \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 16

Solution

Concept — Rolling Kinetic Energy:
For rolling without slipping, total KE $= \frac{1}{2}mv^2 + \frac{1}{2}I\omega^2$. With $I = \frac{2}{5}mr^2$ and $\omega = v/r$:

$$KE_{\text{rot}} = \tfrac{1}{2} \cdot \tfrac{2}{5}mr^2 \cdot \tfrac{v^2}{r^2} = \tfrac{1}{5}mv^2$$

$$KE_{\text{total}} = \tfrac{1}{2}mv^2 + \tfrac{1}{5}mv^2 = \tfrac{7}{10}mv^2$$

Step 1 — Substitute $m = 2\,\text{kg}$, $v = 5\,\text{m/s}$:
$KE_{\text{total}} = \frac{7}{10} \times 2 \times 25 = \frac{7}{10} \times 50 = \mathbf{35\,J}$.

Why other options are wrong:

Q17.

- **(A)** 25 J — only translational KE ($\frac{1}{2}mv^2$); omits rotation.
- **(B)** 17.5 J — only rotational KE ($\frac{1}{5}mv^2$).
- **(C)** 50 J — uses mv^2 (missing factor $\frac{1}{2}$) for translational part only.

Final Answer: $KE_{\text{total}} = 35\,\text{J} \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 17

Solution

Concept — Continuity Equation (Fluid Mechanics):
For an incompressible fluid, $A_1v_1 = A_2v_2$ (conservation of mass flow rate).

Step 1 — Solve for v_2:
$$v_2 = \frac{A_1}{A_2}\,v_1 = \frac{4\,\text{cm}^2}{1\,\text{cm}^2} \times 1\,\text{m/s} = \mathbf{4\,m/s}.$$

Step 2 — Physical check: Narrowing the pipe by a factor of 4 speeds the fluid by a factor of 4; pressure at the throat is lower (Bernoulli).

Why other options are wrong:

Q18.

- **(A)** 1 m/s — ignores the area change; speed unchanged only if $A_1 = A_2$.
- **(B)** 2 m/s — uses ratio $A_1/A_2 = 2$ (wrong; $4/1 = 4$, not 2).
- **(D)** 16 m/s — squares the area ratio instead of using it linearly.

Final Answer: $v_2 = 4\,\text{m/s} \Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 18

Solution

Concept — Torque on an Electric Dipole:
$\tau = pE\sin\theta$, where p is the dipole moment, E the electric field, and θ the angle between them.

Step 1 — Substitute:
$\tau = (1.0 \times 10^{-30})(1.0 \times 10^{5})\sin 30^\circ = 10^{-25} \times 0.5 = \mathbf{5.0 \times 10^{-26}}\,\mathrm{N\,m}$.

Why other options are wrong:

Q19.

- **(A)** $1.0 \times 10^{-25}\,\mathrm{N\,m}$ — uses $\sin 90^\circ = 1$ (maximum torque, wrong angle).
- **(C)** $\sqrt{3} \times 10^{-25}\,\mathrm{N\,m}$ — uses $\sin 60^\circ = \sqrt{3}/2$ (wrong angle); also magnitude off.
- **(D)** $1.0 \times 10^{-26}\,\mathrm{N\,m}$ — off by a factor of 5; arithmetic error.

Final Answer: $\tau = 5.0 \times 10^{-26}\,\mathrm{N\,m} \Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 19

Solution

Concept — Lenz's Law:
Lenz's law states that the direction of an induced current is such that the magnetic field it creates *opposes* the change in the magnetic flux that caused it (a consequence of energy conservation).

Step 1 — Apply to the figure: The N-pole moves toward the coil (downward), increasing upward flux linkage through the coil. The induced current flows counterclockwise (viewed from above), producing an upward magnetic field that opposes the increasing flux.

Step 2 — Identify the law: This direction-determining rule is **Lenz's law**. The magnitude of the induced EMF is given by Faraday's law; Lenz's law gives the *direction*.

Why other options are wrong:

Q20.

- **(B)** Biot–Savart law — gives the magnetic field produced by a current element; does not determine direction of induced current.
- **(C)** Ampere's circuital law — relates magnetic field circulation to enclosed current; does not predict induced current direction from flux change.
- **(D)** Coulomb's law — governs electrostatic force between charges; unrelated to induction.

Final Answer: Lenz's law $\Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 20

Solution

Concept — Critical Angle for Total Internal Reflection:
At the critical angle, the refracted ray grazes the interface ($\theta_r = 90°$). Snell's law gives: $n_1 \sin\theta_c = n_2 \sin 90° \Rightarrow \sin\theta_c = n_2/n_1$.

Step 1 — Calculate:
$\sin\theta_c = \dfrac{1.0}{1.5} = \dfrac{2}{3} \approx 0.667 \Rightarrow \theta_c = \arcsin(0.667) \approx \mathbf{41.8°}$.

Why other options are wrong:

Q21.
- **(A)** 30° — corresponds to $\sin\theta_c = 0.5$, i.e., $n_1 = 2.0$; not the case here.
- **(B)** 60° — corresponds to $\sin\theta_c = \sqrt{3}/2 \approx 0.866$; incorrect for $n = 1.5$.
- **(C)** 90° — would mean no total internal reflection ever occurs ($n_1 = n_2$).

Final Answer: $\theta_c = \arcsin(2/3) \approx 41.8° \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 21

Solution

Concept — Wheatstone Bridge Null Condition:
At balance: $P/Q = R/S$, so $S = QR/P$.

Step 1 — Substitute:
$S = \dfrac{8 \times 6}{4} = \dfrac{48}{4} = \mathbf{12\,\Omega}$.

Why other options are wrong:

Q22.
- **(A)** 3 Ω — uses $S = P \cdot R/Q = 4 \times 6/8 = 3$ (inverted ratio).
- **(B)** 6 Ω — takes $S = R$ (ignores P and Q ratio).
- **(D)** 24 Ω — computes $S = Q \cdot R = 8 \times 6/2$ (wrong formula).

Final Answer: $S = 12\,\Omega \Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 22

Solution

Concept — Bragg's Law:
$2d\sin\theta = m\lambda$. For $m = 1$ (first order), $\sin\theta = \lambda/(2d)$.

Step 1 — Substitute:
$\sin\theta = \dfrac{0.10\,\text{nm}}{2 \times 0.20\,\text{nm}} = \dfrac{0.10}{0.40} = 0.25$.

Step 2 — Find θ:
$\theta = \arcsin(0.25) \approx \mathbf{14.5°}$.

Why other options are wrong:

Q23.
- **(B)** 30° — would require $\sin\theta = 0.50 = \lambda/(2d)$, implying $\lambda = 0.20\,\text{nm}$.
- **(C)** 45° — would require $\sin\theta = 0.707$; inconsistent with given values.

- **(D)** 90° — physically impossible for a first-order maximum with these parameters.

Final Answer: $\theta \approx 14.5^\circ \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 23

Solution

Concept — Nuclear Binding Energy from Mass Defect:
$\Delta m = (Zm_p + Nm_n) - m_{\text{nucleus}}$; $\quad BE = \Delta m \times 931.5\,\text{MeV/u}$.

Step 1 — Compute total constituent mass:
$2m_p + 2m_n = 2(1.0073) + 2(1.0087) = 2.0146 + 2.0174 = 4.0320\,\text{u}$.

Step 2 — Mass defect:
$\Delta m = 4.0320 - 4.0026 = 0.0294\,\text{u}$.

Step 3 — Binding energy:
$BE = 0.0294 \times 931.5 \approx \mathbf{27.4}\,\text{MeV}$.

Why other options are wrong:

Q24.

- **(A)** 7.1 MeV — is the *binding energy per nucleon* ($27.4/4 \approx 6.85\,\text{MeV}$ per nucleon); not the total.
- **(C)** 56.5 MeV — overestimates by using an incorrect mass defect.
- **(D)** 2.2 MeV — is the deuteron binding energy; not ^{4}He.

Final Answer: $BE(^4\text{He}) \approx 27.4\,\text{MeV} \Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 24

Solution

Concept — de Broglie Wavelength for a Proton:
$\lambda = h/p$, where $p = \sqrt{2m_pE_k}$ for a non-relativistic proton with kinetic energy E_k.

Step 1 — Compute momentum:
$p = \sqrt{2 \times 1.67 \times 10^{-27} \times 1.6 \times 10^{-13}} = \sqrt{5.344 \times 10^{-40}} \approx 2.31 \times 10^{-20}\,\text{kg m/s}$.

Step 2 — Wavelength:
$$\lambda = \frac{6.63 \times 10^{-34}}{2.31 \times 10^{-20}} \approx 2.87 \times 10^{-14}\,\text{m} \approx 29\,\text{fm}.$$

Step 3 — Physical sense: A 1 MeV proton has a de Broglie wavelength of order femtometers — nuclear-scale, consistent with proton scattering experiments.

Why other options are wrong:

Q25.

- **(A)** 2.9 nm — wavelength of a low-energy electron (∼eV); proton at 1 MeV is far smaller.
- **(B)** 0.29 pm — $\sim 10^{-13}$ m; off by an order of magnitude.

- **(C)** 2.9 pm — $\sim 10^{-12}$ m; two orders of magnitude too large.

Final Answer: $\lambda \approx 2.87 \times 10^{-14}$ m $\Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 25

Solution

Concept — Entropy Generation vs. Entropy Change:
$\Delta S = S_{\text{final}} - S_{\text{initial}}$ is path-independent (state function). Entropy *generation* $\sigma_{\text{gen}} \geq 0$: zero for reversible, positive for irreversible processes (second law).

Step 1 — Same ΔS for both paths: Since initial and final states are identical, ΔS is the same for Path 1 and Path 2.

Step 2 — Entropy generated:

Q26.

- Path 1 (reversible): $\sigma_{\text{gen}} = 0$ (ideal, no irreversibilities).
- Path 2 (irreversible step-function): finite friction/dissipation in the non-equilibrium steps; $\sigma_{\text{gen}} > 0$.

Why other options are wrong:

- **(A)** Path 1 generates entropy — false; reversible processes have $\sigma_{\text{gen}} = 0$ by definition.
- **(B)** Both generate equal entropy — false; σ_{gen} is path-dependent even though ΔS is not.
- **(D)** Neither generates entropy — false; any real irreversible process generates entropy.

Final Answer: Path 2 (irreversible) generates entropy $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 26

Solution

Concept — Lissajous Figures and Frequency Ratios:
When two perpendicular sinusoidal oscillations of frequency ratio $\omega_x : \omega_y = 1 : 2$ are combined, the parametric curve $x = A\sin(\omega t)$, $y = A\sin(2\omega t)$ traces a figure-eight.

Step 1 — Algebraic verification: $y = \sin(2\omega t) = 2\sin(\omega t)\cos(\omega t) = 2x\sqrt{1 - x^2}$. This describes two symmetric loops (one above, one below the x-axis), forming a **figure-eight**.

Step 2 — General rule: The number of loops in x and y directions equals ω_y and ω_x respectively (for 1:2 ratio: 2 loops along y, 1 along x).

Why other options are wrong:

- **(A)** Straight line — occurs for ratio 1:1 with zero phase difference.
- **(C)** Circle — occurs for ratio 1:1 with 90° phase difference.
- **(D)** Ellipse — occurs for ratio 1:1 with intermediate phase difference.

Final Answer: Figure-eight (two loops) $\Rightarrow$ **(B)**

Q27. **Answer: (B)** ↑ Go back to Q 27

Solution

Concept — Newton's Rings and Phase Changes on Reflection:
Two reflected rays interfere: ray 1 reflects off the lower curved surface of the lens (going from glass into air: no phase shift), and ray 2 reflects off the upper flat surface of the plate (going from air into glass: phase shift of π, equivalent to path difference $\lambda/2$).

Step 1 — At the centre (air gap $= 0$): Path difference due to gap $= 2 \times 0 = 0$. But the phase shift of π on reflection from the denser glass plate adds an effective path difference of $\lambda/2$. Total path difference $= \lambda/2 \Rightarrow$ **destructive interference** $\Rightarrow$ **dark centre**.

Why other options are wrong:

- **(B)** Lens absorbs light — false; the lens is transparent glass.
- **(C)** Constructive interference at centre — false; the phase shift makes it destructive.
- **(D)** Total internal reflection — not applicable; the air gap does not cause TIR in this geometry.

Final Answer: Phase shift of π at the glass plate causes destructive interference $\Rightarrow$ **(A)**

Q28. **Answer: (A)** ↑ Go back to Q 28

Solution

Concept — Mutual Inductance:
The induced EMF in coil 2 due to changing current in coil 1: $\mathcal{E}_2 = M\,\dfrac{dI_1}{dt}$. Solving: $M = \mathcal{E}_2 / \dfrac{dI_1}{dt}$.

Step 1 — Substitute:
$M = \dfrac{0.50\,\text{V}}{5.0\,\text{A/s}} = \mathbf{0.10\,H}.$

Why other options are wrong:

- **(A)** 2.5 H — inverts the formula: $M = (dI/dt)/\mathcal{E} = 5/0.5 = 10$; then divides by 4 arbitrarily.

Q29.

- **(B)** 1.0 H — off by factor of 10; arithmetic error.
- **(C)** 0.5 H — confuses $\mathcal{E}$ with M directly.

Final Answer: $M = 0.10\,\text{H} \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 29

Solution

Concept — Quantum Tunneling Exponential Barrier Dependence:
$T \approx e^{-2\kappa d}$, where $\kappa = \sqrt{2m(V_0 - E)}/\hbar$ is independent of d.

Step 1 — Double the barrier width:
$T' \approx e^{-2\kappa(2d)} = e^{-4\kappa d} = \left(e^{-2\kappa d}\right)^2 = T^2$.

Step 2 — Implication: If $T = 10^{-3}$, then $T' = 10^{-6}$ — an exponential (not linear) decrease. Quantum tunneling is exquisitely sensitive to barrier width.

Why other options are wrong:

Q30.

- **(A)** T halved — linear reduction; wrong functional form. Doubling d doubles the exponent.
- **(B)** $T = 0$ — transmission never goes exactly to zero for a finite barrier; it is exponentially small but non-zero.
- **(D)** T unchanged — κ is independent of d; the barrier width directly enters the exponent.

Final Answer: $T' \approx T^2$ (exponential decrease) $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 30

Solution

Concept — Baeyer-Villiger Oxidation:
Peroxyacids (e.g., m-CPBA) oxidise ketones by inserting an oxygen atom between the carbonyl carbon and the more-substituted (or better-migrating) adjacent carbon, converting a ketone to an ester (or lactone for cyclic ketones).

Step 1 — Apply to cyclohexanone: Oxygen is inserted between the carbonyl carbon (C1) and the adjacent ring carbon, opening the ring and forming a 7-membered ring lactone: ε-**caprolactone** (ε-caprolactone, *oxepan-2-one*).

Step 2 — Migration aptitude: In cyclic ketones, both adjacent carbons are equivalent; oxygen insertion gives the corresponding lactone with the same ring size $+1$ atom.

Why other options are wrong:

Q31.

- **(A)** Cyclohexanol — would require reduction; m-CPBA is an oxidant, not reductant.

- **(C)** Cyclohexane-1,2-diol — m-CPBA can epoxidise alkenes, but cyclohexanone has no alkene.
- **(D)** Cyclohex-2-enone — α,β-dehydrogenation requires DDQ or SeO_2; not a Baeyer-Villiger product.

Final Answer: ε-Caprolactone $\Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 31

Solution

Concept — Grignard Reagent Nucleophilicity:
In R-MgX, the C-Mg bond is highly polarised ($C^{\delta-}$-$Mg^{\delta+}$). The carbon bears a partial negative charge, making it a **carbanion equivalent** — a powerful nucleophile and strong base.

Step 1 — Reactivity: Grignard reagents attack electrophilic centres: carbonyl carbons (aldehydes, ketones, esters, CO_2), producing alcohols and carboxylic acid derivatives after workup.

Why other options are wrong:

Q32.

- **(B)** Electrophile — the carbon in RMgX is nucleophilic, not electrophilic. Electrophiles are electron-deficient.
- **(C)** Radical species — Grignard formation is ionic, not radical (no peroxide or light required).
- **(D)** Lewis acid — Mg is a Lewis acid, but the organic carbon (carbanion) acts as a Lewis base/nucleophile.

Final Answer: R acts as a carbanion equivalent (strong nucleophile) $\Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 32

Solution

Concept — HSAB (Pearson) Principle:
Hard acids bind preferentially to hard bases; soft acids prefer soft bases. "Hardness" correlates with small size, high charge, and low polarisability.

Step 1 — Classify Cr^{3+}: Small, highly charged $(3+)$, non-polarisable $\Rightarrow$ **hard Lewis acid**.

Step 2 — Classify ligands:

Q33.

- I^-: large, highly polarisable $\Rightarrow$ soft base.
- SCN^- (S-bound): soft end.
- CN^-: soft π-acid ligand.
- F^-: small, non-polarisable, high charge density $\Rightarrow$ **hard base**.

Hard Cr^{3+} prefers hard F^- over soft I^-, consistent with Cr^{3+} being commonly found in fluoride and oxide complexes.

Why other options are wrong:

- **(A)** I^- — soft; not preferred by the hard Cr^{3+}.
- **(B)** SCN^-(S) — borderline/soft; less preferred than F^-.
- **(C)** CN^- — soft π-acceptor; preferred by soft metal centres (Pt^{2+}, Pd^{2+}).

Final Answer: F^- (hard base) $\Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 33

Solution

Concept — van 't Hoff Osmotic Pressure:
$\pi = iMRT$; for a non-electrolyte (glucose), $i = 1$.

Step 1 — Substitute:
$\pi = 1 \times 0.10 \times 0.0821 \times 300 = 0.10 \times 24.63 = \mathbf{2.46}$ atm.

Why other options are wrong:

Q34.

- **(A)** 0.246 atm — off by factor 10; uses $M = 0.01$ instead of 0.10.
- **(B)** 24.6 atm — off by factor 10 in the other direction; uses $M = 1.0$.
- **(D)** 0.082 atm — uses only R without M or T contributions.

Final Answer: $\pi \approx 2.46$ atm $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 34

Solution

Concept — Tyndall Effect in Colloids:
Colloidal particles (diameter 1–100 nm) scatter visible light (Tyndall effect). True solution particles (< 1 nm) are too small to scatter light; the beam passes invisibly.

Step 1 — Identify the colloid: A starch-in-water suspension contains macromolecular aggregates (∼5–100 nm) $\Rightarrow$ colloidal dispersion $\Rightarrow$ exhibits **Tyndall effect**.

Why other options are wrong:

Q35.

- **(A)** NaCl(aq) — true solution; Na^+ and Cl^- ions (< 0.5 nm) do not scatter light.
- **(C)** Distilled water — no dispersed particles; no scattering.
- **(D)** HCl(aq) — fully dissociated ionic true solution; no colloidal particles.

Final Answer: Starch suspension (colloidal) $\Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 35

Solution

Concept — CFSE for d^6 Low-Spin Octahedral Complex:
In a strong-field octahedral ligand field, d-orbitals split into t_{2g} ($-4\,Dq$) and e_g ($+6\,Dq$). For d^6 low-spin: all 6 electrons fill t_{2g} ($t_{2g}^6\, e_g^0$).

Step 1 — CFSE:
CFSE $= 6 \times (-4\,Dq) + 0 \times (6\,Dq) = -24\,Dq$.

Step 2 — NH_3 is a strong-field ligand (spectrochemical series: $NH_3 > H_2O \gg F^-$), confirming low-spin configuration for Co^{3+} (d^6).

Why other options are wrong:

Q36.

- **(B)** $t_{2g}^4\, e_g^2$, CFSE$= -8Dq$ — high-spin configuration, appropriate for weak-field ligands (e.g., F^-).
- **(C)** $t_{2g}^3\, e_g^3$, CFSE$= 0$ — incorrect electron count and no stabilisation.
- **(D)** $t_{2g}^5\, e_g^1$ — incorrect for d^6; this would be 6 electrons total but distributed wrongly.

Final Answer: $t_{2g}^6\, e_g^0$; CFSE $= -24\,Dq \Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 36

Solution

Concept — Electroplating and Electrode Roles:
In an electrolytic cell (electroplating), the object to be coated is connected to the *negative* terminal (cathode). Metal cations from solution are *reduced* and deposit as metal on the cathode.

Step 1 — At the cathode (iron object):
$Cu^{2+}(aq) + 2e^- \rightarrow Cu(s)$ — copper deposits on iron.

Step 2 — At the anode (copper plate):
$Cu(s) \rightarrow Cu^{2+}(aq) + 2e^-$ — copper dissolves, maintaining $[Cu^{2+}]$.

Why other options are wrong:

Q37.

- **(A)** Anode — the anode is the copper plate (positive terminal), not the iron object.
- **(B)** Electrolyte — the electrolyte is the $CuSO_4$ solution; iron is the electrode.
- **(C)** Salt bridge — a salt bridge appears in galvanic cells; irrelevant here.

Final Answer: Iron object is the cathode $\Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 37

Solution

Concept — Isoelectric Focusing and pI:
The isoelectric point (pI) is the pH at which a protein's net charge is zero. At pI, the protein has equal numbers of positive and negative charges, giving no net movement in an electric field.

Step 1 — At pI: The electrophoretic force is zero (no net charge). The protein *stops migrating* and is concentrated (focused) into a sharp band at the position in the IPG gel corresponding to its pI.

Step 2 — Application in 2D-PAGE: IEF (first dimension) separates proteins by pI; SDS-PAGE (second dimension) separates by mass. The combination (2D-PAGE) gives high resolution.

Why other options are wrong:

Q38.

- **(A)** Maximum positive charge — occurs at pH $\ll$ pI; protein still migrates toward cathode.
- **(B)** Maximum negative charge — occurs at pH $\gg$ pI; protein migrates toward anode.
- **(D)** Irreversible precipitation — proteins may precipitate at pI but can usually be resolubilised; precipitation is a practical problem, not the definition.

Final Answer: At pI, zero net charge; protein stops migrating $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 38

Solution

Concept — Invert Sugar (Sucrose Hydrolysis):
Sucrose ($[\alpha]_D^{25} = +66.5°$) is hydrolysed to glucose ($[\alpha] = +52.5°$) and fructose ($[\alpha] = -92.4°$) in equimolar amounts. The mixture's specific rotation: $(52.5 + (-92.4))/2 = -19.95°$ (levorotatory). The sign has *inverted* from $+$ to $-$, hence the name **invert sugar**.

Step 1 — Etymology: "Inversion" refers to the change in optical rotation from dextrorotatory (sucrose) to levorotatory (glucose + fructose mixture).

Why other options are wrong:

Q39.

- **(A)** Reducing sugar — sucrose itself is non-reducing; glucose and fructose are reducing sugars. But the mixture is called invert sugar, not just "reducing sugar."
- **(C)** Maillard product — the Maillard reaction is a non-enzymatic browning between amino acids and reducing sugars (dry heat); unrelated.
- **(D)** Lactulose — a synthetic disaccharide (galactose + fructose) used as a laxative; not the product of sucrose hydrolysis.

Final Answer: The equimolar glucose + fructose mixture is called invert sugar ⇒ **(B)**

Answer: (B) ↑ Go back to Q 39

Solution

Concept — Ring Strain and Reactivity of Cyclopropane:
The 60° C-C-C angles in cyclopropane deviate severely from the ideal 109.5° tetrahedral angle, creating Baeyer (angle) strain and significant p-orbital character in the C-C bonds (bent bonds, "banana bonds"). This makes cyclopropane much more reactive than cyclohexane.

Step 1 — Ring-opening reactions cyclopropane undergoes but cyclohexane does not:

Q40.

- Catalytic hydrogenation: $+H_2$ / Pt $\rightarrow$ propane.
- Addition of HBr: $\rightarrow$ 1-bromopropane.
- Addition of Br_2: $\rightarrow$ 1,3-dibromopropane.

These reactions are *electrophilic additions*, analogous to alkene reactivity.

Why other options are wrong:

- **(B)** Less reactive because geometrically rigid — ring strain actually *increases* reactivity.
- **(C)** More stable due to minimised eclipsing strain — eclipsing strain is indeed absent in cyclopropane, but angle strain dominates and destabilises it overall.
- **(D)** Unreactive toward electrophiles — false; cyclopropane reacts with electrophiles via ring opening.

Final Answer: Cyclopropane is more reactive (ring-opening with Br_2, HBr, H_2) ⇒ **(A)**

Answer: (A) ↑ Go back to Q 40

Solution

Concept — Mass Spectrometry α-Cleavage of Ketones:
In EI mass spectrometry, ketones undergo α-cleavage (bond between the carbonyl carbon and an adjacent carbon). 2-Butanone (CH_3-CO-CH_2CH_3) can cleave on either side of C=O.

Step 1 — α-Cleavages:

Q41.

- Cleavage of C(carbonyl)-CH_2CH_3: gives CH_3CO^+ ($m/z = 43$, acylium) + $CH_2CH_3\cdot$ (neutral radical lost).

- Cleavage of C(carbonyl)-CH_3: gives $C_2H_5CO^+$ ($m/z = 57$) + $CH_3\cdot$; or loss of CH_3 gives $m/z = 57$.
- $C_2H_5^+$ fragment ($m/z = 29$) from C-C bond remote from carbonyl.

Step 2 — Base peak: $m/z = 43$ (CH_3CO^+, acylium ion) is the most stable fragment (resonance stabilisation by the C=O) and is therefore the **base peak** (100% relative intensity), as shown in the spectrum.

Why other options are wrong:

- **(A)** $m/z = 72$ (M^+) — present but not the base peak; intensity is lower.
- **(B)** $m/z = 29$ ($C_2H_5^+$) — present but less stable; not the base peak.
- **(C)** $m/z = 57$ — loss of CH_3 (-15); minor fragment in 2-butanone.

Final Answer: Base peak at $m/z = 43$ (CH_3CO^+, acylium ion) ⇒ **(D)**

Answer: (D) ↑ Go back to Q 41

Solution

Concept — Born-Oppenheimer Approximation:
The key insight: $m_e/m_p \approx 1/1836$. Electrons move $\sim 10^3$ times faster than nuclei. Therefore, for any nuclear configuration, electrons instantaneously reach their ground state. Nuclear and electronic wavefunctions can be *separated*: $\Psi_{\text{total}} \approx \psi_e(\mathbf{r};\mathbf{R}) \cdot \chi_n(\mathbf{R})$.

Step 1 — Practical consequence: Potential energy surfaces (PES) can be computed for fixed nuclear positions — the foundation of computational chemistry, molecular mechanics, and understanding chemical reactions.

Why other options are wrong:

Q42.

- **(A)** Hartree-Fock — an approximation for treating electron-electron repulsion (mean-field); a separate concept from nuclear-electronic separation.
- **(B)** Franck-Condon principle — uses Born-Oppenheimer but specifically governs vibrational overlap integrals in electronic transitions (spectroscopy).
- **(D)** Variational principle — states the trial wavefunction energy is always $\geq$ true ground state energy; a mathematical theorem, not about nuclear motion.

Final Answer: Born-Oppenheimer approximation ⇒ **(C)**

Answer: (C) ↑ Go back to Q 42

Solution

Concept — Nernst Equation for a Half-Cell:
$E = E^\circ - \dfrac{0.0592}{n} \log_{10}[Zn^{2+}]$ at 25°C (for the reduction half-reaction with $n = 2$).

Step 1 — Substitute:
$E = -0.76 - \frac{0.0592}{2}\log_{10}(0.010) = -0.76 - (0.0296) \times (-2) = -0.76 + 0.0592 \approx -\mathbf{0.70}\,\text{V}$.

Step 2 — Physical sense: Reducing $[Zn^{2+}]$ makes the Zn electrode slightly less negative (closer to zero) than standard; a more negative electrolyte concentration would drive more dissolution.

Why other options are wrong:

Q43.

- **(A)** $-0.82\,\text{V}$ — would result from increasing $[Zn^{2+}]$ above 1 M, not decreasing it.
- **(C)** $-0.76\,\text{V}$ — is E° (standard, $[Zn^{2+}]= 1\,\text{M}$); non-standard conditions must use Nernst.
- **(D)** $-0.64\,\text{V}$ — arithmetic error; off by $\sim 0.12\,\text{V}$.

Final Answer: $E \approx -0.70\,\text{V} \Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 43

Solution

Concept — Cooperative Binding and the Hill Equation:
Hill equation: $\theta = \frac{[S]^n}{K_d^n + [S]^n}$. For $n = 1$: hyperbolic (Michaelis-Menten). For $n > 1$: sigmoidal (cooperative). For $n < 1$: negative cooperativity.

Step 1 — Identify curves from the figure:

Q44.

- Blue hyperbolic curve: $n = 1$, non-cooperative (Michaelis-Menten).
- Red sigmoidal curve: $n > 1$, positive cooperativity — the curve is initially flat (low affinity at low $[S]$) then rises steeply. Classic example: haemoglobin-O_2 binding ($n \approx 2.8$).

Step 2 — Biological meaning of $n > 1$: Binding of the first ligand molecule increases the affinity of remaining sites (conformational change, e.g., T-state $\rightarrow$ R-state in haemoglobin). This gives an all-or-none switch-like response.

Why other options are wrong:

- **(B)** Hyperbolic curve; multiple independent sites — independent sites give $n = 1$ exactly.
- **(C)** Both curves; $n > 1$ means saturating $[S]$ — n is not about saturation; it is about cooperativity.
- **(D)** Cooperative binding gives a straight line on a Hill plot — a Hill plot linearises the sigmoidal curve; the curve itself is not straight.

Final Answer: Sigmoidal (red) curve; $n > 1$ indicates positive cooperativity $\Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 44

Solution

Concept — Affinity Chromatography Principle:
Affinity chromatography exploits the specific, high-affinity, reversible binding between a target molecule and an immobilised ligand — the same biochemical recognition used in biological function (enzyme-substrate, antibody-antigen, receptor-ligand).

Step 1 — Mechanism:

Q45. (a) Load: all proteins pass through; only the target binds to the immobilised ligand.
(b) Wash: non-specific proteins are removed.
(c) Elute: target released by competing ligand, salt, or pH change.

Result: very high purity in one step (thousands-fold enrichment possible).

Why other options are wrong:

- **(A)** Molecular size — exploited by size-exclusion (gel-filtration) chromatography; not affinity.
- **(B)** Net charge / pI — exploited by ion-exchange chromatography or IEF; not affinity.
- **(C)** Hydrophobicity — exploited by hydrophobic interaction chromatography (HIC); not affinity.

Final Answer: Specific and reversible binding affinity for a particular ligand $\Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 45

Solution

Concept — Histone Acetylation and Chromatin Remodeling:
Lysine residues on histone N-terminal tails carry $+$ charges (protonated $-NH_3^+$) that interact electrostatically with negatively charged DNA phosphate backbone, compacting chromatin.

Step 1 — Effect of acetylation by HAT: The acetyl group ($-COCH_3$) neutralises the positive charge on lysine (ε-$NH_3^+ \rightarrow \varepsilon$-$NHCOCH_3$; no charge). This **reduces** the electrostatic attraction between histones and DNA, causing the nucleosome to loosen, chromatin to **relax** (euchromatin), and transcription factors/RNA Pol II to access the promoter $\Rightarrow$ **gene activation**.

Step 2 — Enzyme classification: HAT (histone acetyltransferase) adds acetyl; HDAC (histone deacetylase) removes it, reversing the effect.

Why other options are wrong:

- **(A)** Increases positive charge — incorrect; acetylation *neutralises* positive charge.
- **(B)** Promotes DNA methylation — DNA methylation is a separate epigenetic mark catalysed by DNMTs; not a direct consequence of HAT activity.
- **(D)** Recruits HDACs — HDACs do the *opposite* (deacetylate and condense chromatin).

Final Answer: Acetylation neutralises histone + charge → relaxes chromatin → activates transcription ⇒ **(C)**

Q46. **Answer: (C)** ↑ Go back to Q 46

Solution

Concept — RNAi Pathway and Argonaute "Slicer" Activity:
In the RNAi pathway: dsRNA $\xrightarrow{\text{Dicer}}$ siRNA duplex → loaded into RISC → guide strand directs RISC to complementary mRNA → Argonaute cleaves mRNA.

Step 1 — Argonaute (AGO2 in mammals): The PIWI domain of AGO2 has endonuclease (RNase H-like) "Slicer" activity. When the guide strand is perfectly complementary to the target mRNA, AGO2 cleaves the mRNA at the phosphodiester bond between positions 10 and 11 of the guide strand (counting from the 5' end). This produces mRNA fragments that are rapidly degraded.

Why other options are wrong:

- **(A)** Unwinds siRNA and exports from nucleus — the RNA-helicase that unwinds the siRNA duplex acts before RISC assembly; Argonaute's role is cleavage, not export.
- **(C)** Methylates 5' cap — mRNA cap methylation is a co-transcriptional modification; Argonaute has no methyltransferase activity.
- **(D)** Recruits Dicer to degrade dsRNA — Dicer acts *upstream* of RISC/Argonaute; they are sequential, not Argonaute recruiting Dicer.

Final Answer: Argonaute is the "Slicer" endonuclease that cleaves target mRNA ⇒ **(B)**

Q47. **Answer: (B)** ↑ Go back to Q 47

Solution

Concept — Ribosomal tRNA Sites During Elongation:
Three tRNA-binding sites in the ribosome: A-site (aminoacyl, incoming aa-tRNA), P-site (peptidyl, growing chain), E-site (exit, deacylated tRNA about to leave).

Step 1 — State before peptide bond formation (pre-translocational):

- P-site: peptidyl-tRNA (tRNA carrying the growing polypeptide chain).
- A-site: incoming aminoacyl-tRNA (delivered by EF-Tu·GTP in prokaryotes).

Step 2 — After peptide bond formation: The ribosome catalyses transpeptidation (peptidyl transferase). The new peptide bond is made between the carboxyl of the peptidyl-tRNA in P and the amino group of aa-tRNA in A. **Immediately after**, the P-site tRNA is deacylated (empty) and the A-site carries the elongated peptidyl-tRNA. Translocation then moves peptidyl-tRNA from A to P.

Step 3 — During elongation (pre-translocational state, P-site): The **peptidyl-tRNA** (polypeptide still attached) occupies the P-site.

Why other options are wrong:

- **(B)** Incoming aa-tRNA — this is the *A-site* occupant.
- **(C)** Deacylated tRNA — occupies the E-site (prior to exiting), not the P-site during active elongation.
- **(D)** Release factor — binds the A-site only upon encountering a stop codon; not present during normal elongation.

Final Answer: Peptidyl-tRNA (polypeptide chain) occupies the P-site $\Rightarrow$ **(A)**

Q48. **Answer: (A)** ↑ Go back to Q 48

Solution

Concept — Non-disjunction in Meiosis II:
Meiosis II is essentially a mitotic division separating sister chromatids. If non-disjunction occurs in one secondary oocyte/spermatocyte (say cell 1), sister chromatids of one chromosome fail to separate.

Step 1 — Products from cell 1 (non-disjunction):

- One gamete receives *both* sister chromatids of the affected chromosome: $n+1$ (disomic).
- The other gamete receives *none*: $n-1$ (nullisomic).

Step 2 — Products from cell 2 (normal meiosis II):

- Two normal n gametes.

Step 3 — Total from one meiosis: Two normal (n), one disomic $(n+1)$, one nullisomic $(n-1)$ — 4 gametes total.

Why other options are wrong:

- **(A)** All four aneuploid — only cell 1 is affected; cell 2 divides normally.
- **(B)** Two $n+1$ and two $n-1$ — this is the pattern for meiosis I non-disjunction (primary non-disjunction).
- **(C)** All four normal — this is the result of a normal meiosis.

Q49.

Final Answer: Two n, one $n+1$, one $n-1 \Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 49

Solution

Concept — SNARE-Mediated Membrane Fusion:
Intracellular vesicle fusion requires physical proximity of two membranes and subsequent bilayer merger. SNAREs provide both the specificity and the mechanical force.

Step 1 — Trans-SNARE complex formation:

Q50.

- v-SNARE (on vesicle, e.g., VAMP/synaptobrevin) pairs with t-SNARE complex on target membrane (e.g., syntaxin + SNAP-25).
- The four SNARE helices (1 from v-SNARE, 3 from t-SNARE) zipper together from N- to C-terminus, forming a tight, stable *trans*-SNARE complex.
- This zippering pulls the two membranes into close apposition (< 5 nm), overcoming electrostatic repulsion and enabling lipid bilayer mixing (fusion).

Why other options are wrong:

- **(A)** v-v homotypic complexes — v-SNAREs pair with t-SNAREs (*hetero*typic); v-v pairing does not drive productive fusion.
- **(B)** SNAREs as calcium sensors only in neurons — the core SNARE machinery is universal; calcium sensing is provided by synaptotagmin, not SNAREs per se.
- **(D)** NSF directly catalyses fusion — NSF (ATPase) *disassembles* cis-SNARE complexes (post-fusion recycling); it does not catalyse fusion directly.

Final Answer: v-SNARE + t-SNARE form a trans-SNARE complex that drives membrane apposition and fusion $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 50

Solution

Concept — SOS Response Activation in *E. coli*:
Under normal conditions, the LexA repressor binds SOS box operator sequences and represses > 40 SOS genes. DNA damage triggers the SOS response through RecA.

Step 1 — Signal: single-stranded DNA: DNA damage (UV, alkylation, replication fork stalling) generates ssDNA gaps. RecA protein binds ssDNA cooperatively, forming a right-handed nucleoprotein filament (RecA-ssDNA-ATP complex) — the activated form.

Step 2 — LexA autocleavage: The activated RecA filament stimulates the latent auto-proteolytic activity of LexA (and other repressors, e.g., UmuD). LexA cleaves itself at Ala84-Gly85, inactivating the repressor and derepressing SOS genes (including *recA*, *umuCD*, *sulA*, *lexA* itself).

Why other options are wrong:

Q51.

- **(A)** LexA binding promotes RecA activity — inverted; RecA promotes LexA *autocleavage*.
- **(C)** Thymine dimers directly bind RecA — the direct signal is ssDNA (from replication stalling at dimers), not the dimers themselves.
- **(D)** MRN complex phosphorylates RecA — MRN is the eukaryotic DSB sensor (activates ATM); RecA activation in prokaryotes is not by phosphorylation.

Final Answer: Accumulation of ssDNA at stalled forks activates RecA by forming a nucleoprotein filament ⇒ **(B)**

Answer: (B) ↑ Go back to Q 51

Solution

Concept — Dominant Lethal Allele (Agouti Yellow):
The A^y (agouti yellow) allele is dominant for yellow coat colour but *homozygous lethal*: A^y/A^y embryos die around embryonic day 6 (E6) due to ectopic expression of the *agouti* signalling protein, disrupting early development.

Step 1 — Cross: $A^y/a \times A^y/a$**. Expected Mendelian ratio:**

$$1\ A^y/A^y\ :\ 2\ A^y/a\ :\ 1\ a/a$$

The A^y/A^y class (1/4 of offspring) dies in utero.

Step 2 — Among survivors: $2\ A^y/a$ (yellow) : $1\ a/a$ (agouti) $=$ **2:1 ratio**.

Why other options are wrong:

Q52.

- **(A)** Incomplete penetrance — A^y is fully penetrant for yellow coat in heterozygotes; the deviation is due to lethality, not penetrance.
- **(B)** a/a die perinatally — false; agouti (a/a) mice are viable and fertile.
- **(C)** Violates law of segregation — segregation is normal; the distortion of phenotypic ratio is due to lethality of one genotype, not a failure of segregation.

Final Answer: A^y/A^y die in utero, removing 1/4 of offspring; survivors are 2:1 yellow:agouti ⇒ **(D)**

Answer: (D) ↑ Go back to Q 52

Solution

Concept — ATM Kinase and DNA Double-Strand Breaks:
ATM (Ataxia Telangiectasia Mutated, a PI3K-like kinase) is the master sensor and transducer of the DNA double-strand break (DSB) response.

Step 1 — Activation mechanism:

Q53. (a) DSBs are rapidly recognised by the MRN complex (MRE11-RAD50-NBS1).
(b) NBS1 directly recruits ATM (inactive dimer) to the break site.
(c) ATM undergoes autophosphorylation (Ser^{1981}) and monomerises to form active kinase.

Step 2 — ATM substrates: γH2AX (Ser^{139}) — chromatin mark for DSBs; Chk2 (Thr^{68}) — cell cycle checkpoint; p53 (Ser^{15}) — apoptosis/arrest; BRCA1.

Why other options are wrong:

- **(B)** UV-induced CPDs — activate ATR (ATR-ATRIP, not ATM), which responds to RPA-coated ssDNA at stalled replication forks.
- **(C)** Alkylation damage — repaired by direct reversal (MGMT) or base excision repair (BER); does not primarily activate ATM.
- **(D)** Mismatched bases — activate the mismatch repair (MMR) pathway via MutSα; can also activate ATR but not ATM directly.

Final Answer: DSBs activate ATM via MRN complex recruitment $\Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 53

Solution

Concept — Cyclic Electron Flow in Chloroplasts:
In linear (non-cyclic) electron flow: $H_2O \xrightarrow{PSII}$ plastoquinone $\xrightarrow{Cyt\ b_6f}$ plastocyanin $\xrightarrow{PSI}$ Fd $\xrightarrow{FNR}$ NADPH. Products: O_2, NADPH, ATP.

Cyclic electron flow: PSI (P700) $\xrightarrow{excited}$ Fd $\rightarrow$ Cyt b_6f $\rightarrow$ PC $\rightarrow$ back to P700. No PSII, no water oxidation, no O_2, no NADPH. Electrons cycle within PSI.

Step 1 — ATP production: As electrons flow through Cyt b_6f, protons are translocated across the thylakoid membrane (lumen side), building a ΔpH gradient. This drives ATP synthase: ADP + P_i $\rightarrow$ ATP. **Net product: ATP only.**

Step 2 — Physiological role: Cyclic flow fine-tunes the ATP:NADPH ratio (Calvin cycle needs $\sim$3 ATP per 2 NADPH per CO_2 fixed); it supplements ATP when NADPH supply is adequate.

Why other options are wrong:

Q54.
- **(A)** NADPH only — NADPH requires Fd to reduce $NADP^+$ via FNR; in cyclic flow, electrons return to P700 via Cyt b_6f instead.
- **(B)** Both ATP and NADPH — NADPH is produced only in linear flow (non-

cyclic).

- **(D)** O_2 and ATP — O_2 is produced by PSII water splitting; PSII is not part of cyclic flow.

Final Answer: Net product of cyclic electron flow = ATP only ⇒ **(C)**

Answer: (C) ↑ Go back to Q 54

Solution

Concept — Primary Succession and Pioneer Species:
Primary succession begins on bare, lifeless substrate (rock, lava, glacial till) with no soil or existing biological community. The pioneer species must tolerate extreme conditions: no soil, low nutrients, temperature fluctuation, and desiccation.

Step 1 — Why lichens and mosses:

Q55.

- **Lichens** (fungal-algal/cyanobacterial mutualism): can photosynthesize, fix atmospheric N_2 (if cyanobacterial partner), secrete acids that weather rock, and tolerate desiccation.
- **Mosses**: colonise small accumulations of organic matter after lichens, adding biomass.

Together they form thin soil, enabling later colonisation by herbs, shrubs, and eventually trees.

Why other options are wrong:

- **(A)** Grasses and annual herbs — require some soil to germinate; they follow lichens in the succession sequence.
- **(C)** Shrubs and perennial plants — mid-successional species; come after soil formation.
- **(D)** Deciduous trees — late successional/climax community; require well-developed soil.

Final Answer: Lichens and mosses are the primary pioneer species ⇒ **(B)**

Answer: (B) ↑ Go back to Q 55

Solution

Concept — Unique Molecular Identifiers (UMIs) in scRNA-seq:
PCR amplification in scRNA-seq introduces profound bias: some cDNA molecules are amplified many more times than others (PCR duplicates), distorting transcript counts. The UMI is a short (∼10–12 nt) random barcode ligated to each cDNA molecule *before* PCR amplification.

Step 1 — How UMIs work:

Q56. (a) During reverse transcription, each cDNA molecule acquires a unique UMI (random sequence; $4^{10} \approx 10^6$ unique tags).

(b) After PCR and sequencing, all reads with the same cell barcode + UMI + gene target originated from the *same* original mRNA molecule.

(c) Computational deduplication collapses these into a single count, regardless of how many PCR copies were generated.

Result: UMI counts reflect the number of original mRNA molecules (absolute transcript quantification).

Why other options are wrong:

- **(A)** Cell demultiplexing — this is the function of the *cell barcode*, not the UMI.
- **(B)** Primer for reverse transcription — the oligo-dT sequence primes RT; the UMI is attached to the primer but is itself not a primer.
- **(C)** Marks 5' end for strand-specific sequencing — UMIs are typically on the 3' end (poly-A capture); 5' capture is a different strategy but UMIs serve deduplication in both.

Final Answer: UMIs enable computational deduplication of PCR amplicons, giving accurate transcript counts ⇒ **(D)**

Answer: (D) ↑ Go back to Q 56

Solution

Concept — Loss of Heterozygosity (LOH) by Mitotic Recombination:
LOH is the loss of one allele at a heterozygous locus in a somatic cell, often exposing a recessive oncogenic or loss-of-function allele. One mechanism is somatic (mitotic) crossing over.

Step 1 — Mechanism:

Q57. (a) A cell heterozygous Aa undergoes mitotic recombination between the locus and the centromere.

(b) After recombination, the four chromatids carry: A-a, A-a → (after interchange) A-A and a-a configurations.

(c) If chromatids segregate such that one daughter cell receives both a chromatids: that cell becomes aa (LOH) and loses the wild-type allele A.

Frequency: Somatic recombination is rare ($\sim 10^{-6}$ per cell per division) but under selection pressure (e.g., growth advantage of aa tumour suppressor knockout), aa cells clonally expand.

Why other options are wrong:

- **(B)** Meiotic non-disjunction — occurs in germline during meiosis; not in

somatic cells during mitosis.

- **(C)** Epigenetic silencing of A — this is epigenetic LOH (promoter methylation); it silences expression but does not alter the DNA sequence. The scenario describes genetic LOH.
- **(D)** Frameshift by error-prone polymerase — point mutations could inactivate A but this is not the same as LOH (which involves loss of genetic material, not acquisition of mutation).

Final Answer: LOH by mitotic (somatic) crossing over between the locus and centromere $\Rightarrow$ **(A)**

Answer: (A) ↑ Go back to Q 57

Solution

Concept — Lincoln-Petersen Mark-Recapture:
Assumptions: closed population, equal catchability, random mixing of marked individuals before second sampling.

Formula: $\widehat{N} = \dfrac{n_1 \times n_2}{m_2}$, where n_1 = initially captured and marked, n_2 = second sample size, m_2 = marked individuals in second sample.

Step 1 — Substitute:
$\widehat{N} = \dfrac{100 \times 80}{20} = \dfrac{8000}{20} = \mathbf{400}$.

Step 2 — Intuition: If 20 out of 80 recaptured fish are marked, then marked fish ($= 100$) represent $20/80 = 25\%$ of the population. So total population $= 100/0.25 = 400$.

Why other options are wrong:

Q58.

- **(A)** 160 — computes $n_1 + n_2 = 180$; wrong formula.
- **(B)** 200 — uses $n_1 \times n_2/(2m_2)$; extra factor of 2.
- **(D)** 800 — uses $n_1 \times n_2/m_2 \times 2$; doubles the correct answer.

Final Answer: $\widehat{N} = 400$ fish $\Rightarrow$ **(C)**

Answer: (C) ↑ Go back to Q 58

Solution

Concept — mTORC1 Downstream Effectors:
mTORC1 (contains mTOR, Raptor, mLST8, PRAS40, Deptor) directly phosphorylates two critical translational regulators:

Step 1 — S6K1 (p70 S6 Kinase 1): mTORC1 phosphorylates S6K1 at Thr^{389} (in the hydrophobic motif), activating it. Active S6K1 phosphorylates S6 ribosomal protein and eIF4B, promoting ribosome biogenesis and translation initiation. Also

phosphorylates and inhibits IRS-1 (negative feedback on PI3K signalling).

Step 2 — 4E-BP1 (eIF4E-Binding Protein 1): mTORC1 phosphorylates 4E-BP1 at multiple sites ($Thr^{37/46}$, Ser^{65}, Thr^{70}), causing 4E-BP1 to release eIF4E. Free eIF4E then binds the 5' cap of mRNAs and recruits the eIF4F complex $\rightarrow$ cap-dependent translation of growth-promoting mRNAs (cyclin D1, c-Myc, HIF-1α).

Why other options are wrong:

Q59.

- **(A)** Akt and AMPK — mTORC1 is *downstream* of Akt (Akt activates mTORC1 via TSC1/2 phosphorylation); mTORC1 does not phosphorylate Akt. AMPK *inhibits* mTORC1.
- **(C)** eIF2α and eIF4G — eIF2α is phosphorylated by HRI/PKR/GCN2/PERK (ISR kinases); eIF4G is a scaffold, not a direct mTORC1 target.
- **(D)** p53 and MDM2 — regulated by ATM/CHK2 (DNA damage pathway); not mTORC1 substrates.

Final Answer: S6K1 and 4E-BP1 are the two key direct mTORC1 substrates $\Rightarrow$ **(B)**

Answer: (B) ↑ Go back to Q 59

Solution

Concept — Activation-Induced Cytidine Deaminase (AID) and Somatic Hypermutation:

AID (encoded by *AICDA*) is a B-cell-specific enzyme expressed in germinal centre B cells after antigen stimulation. It initiates both somatic hypermutation (SHM) and class-switch recombination (CSR).

Step 1 — Mechanism of AID in SHM:

Q60.

(a) AID deaminates cytosine (C) $\rightarrow$ uracil (U) in single-stranded DNA that is transiently exposed during transcription of V-region genes.

(b) U is a non-standard base: it is either replicated over (U:A $\rightarrow$ T:A transition) or processed by UNG (uracil-DNA glycosylase) and error-prone polymerases (Polη), generating a spectrum of substitutions at C:G and A:T base pairs.

(c) Mutations are confined to the complementarity-determining regions (CDRs) of heavy and light chain V regions, generating antibody diversity for affinity selection.

Why other options are wrong:

- **(A)** RAG1/RAG2 — mediates V(D)J recombination in immature B and T cells; not expressed in germinal centres; does not introduce SHM.
- **(B)** TdT — adds N-nucleotides at V(D)J junctions during recombination; not involved in SHM post-antigen stimulation.

- **(C)** Polη — an error-prone polymerase that contributes to SHM *downstream* of AID-initiated lesions, but it is not the initiating enzyme.

Final Answer: AID (Activation-Induced Cytidine Deaminase) catalyses somatic hypermutation $\Rightarrow$ **(D)**

Answer: (D) ↑ Go back to Q 60

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	D	2	A	3	C	4	B	5	D
6	C	7	A	8	B	9	D	10	C
11	B	12	A	13	D	14	C	15	B
16	A	17	D	18	C	19	B	20	A
21	D	22	C	23	A	24	B	25	D
26	C	27	B	28	A	29	D	30	C
31	B	32	A	33	D	34	C	35	B
36	A	37	D	38	C	39	B	40	A
41	D	42	C	43	B	44	A	45	D
46	C	47	B	48	A	49	D	50	C
51	B	52	D	53	A	54	C	55	B
56	D	57	A	58	C	59	B	60	D