NIPER JEE Pharmacy Subjects

Sample Paper – 6

M.S.(Pharm) / M.Pharm Joint Entrance Examination

Duration: 96 Minutes **Maximum Marks: 80**

Instructions

- This paper contains **160 single-correct Multiple Choice Questions** drawn from the pharmaceutical-sciences syllabus of the **NIPER Joint Entrance Examination** (M.S.(Pharm) / M.Pharm).
- Each correct answer carries **+0.5 marks**. **0.125 mark is deducted** for every wrong answer, and an unattempted question gets **0 marks**. Maximum marks: **80**.
- The paper runs continuously from **Q1 to Q160** across six parts: Pharmaceutics; Pharmacology & Toxicology; Pharmaceutical & Medicinal Chemistry; Pharmaceutical Analysis & QA; Pharmacognosy; and Pharmaceutical Biotechnology & Microbiology.
- Only **one** option is correct. Personal calculators, mobile phones, and other electronic gadgets are strictly prohibited.

Part A: Pharmaceutics

Q1. Lead(II) iodide PbI_2 dissociates as $PbI_2 \rightleftharpoons Pb^{2+} + 2\,I^-$. If its molar solubility in water is s, the correct expression for its solubility product K_{sp} is:

(A) s^2

(B) $4s^3$

(C) $27s^4$

(D) s^3

Q2. A drug with an ether/water partition coefficient $P = 4$ is present as 200 mg in 100 mL water and is extracted once with 100 mL ether (equal

volumes). The amount of drug (mg) remaining in the aqueous phase at equilibrium is:

(A) 160

(B) 100

(C) 80

(D) 40

Q3. A blend is made of 30% Surfactant X (HLB $= 15$) and 70% Surfactant Y (HLB $= 5$). The HLB of the blend (algebraic, weight-fraction average) is:

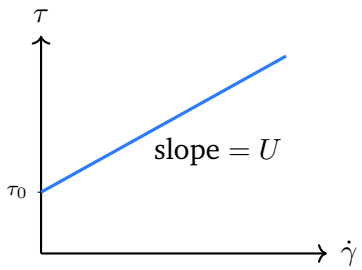
(A) 10.0

(B) 6.0

(C) 8.0

(D) 12.0

Q4. The flow curve below is a straight line that does *not* pass through the origin but intercepts the shear-stress axis at a finite value τ_0 before becoming linear. This behaviour is:



(A) Bingham plastic flow (has a yield value)

(B) Newtonian flow

(C) pseudoplastic flow

(D) dilatant flow

Q5. According to the Stokes–Einstein relation, the diffusion coefficient D of a spherical solute molecule in a liquid is:

(A) directly proportional to the particle radius

(B) inversely proportional to the medium viscosity

(C) independent of absolute temperature

(D) directly proportional to the square of the radius

Q6. The capillary rise of a liquid in a glass tube of radius r (perfect wetting) is given by $h = 2\gamma/(r\rho g)$. If the capillary radius is halved while all other factors stay constant, the height of rise will:

(A) halve

(B) stay the same

(C) double

(D) become one-quarter

Q7. A powder shows a bulk density of 0.45 g/mL and a tapped density of 0.60 g/mL. Carr's compressibility index is:

(A) 15%

(B) 33%

(C) 30%

(D) 25%

Q8. For the same powder (bulk density 0.45 g/mL, tapped density 0.60 g/mL), the Hausner ratio is approximately:

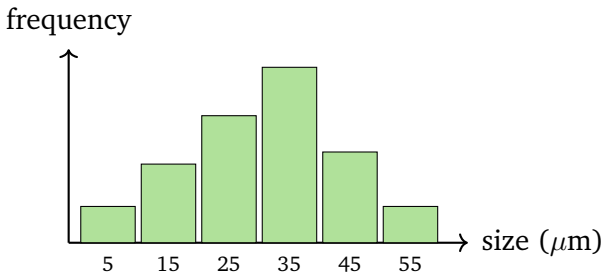
(A) 1.33

(B) 0.75

(C) 1.15

(D) 1.50

Q9. The frequency histogram below shows a particle-size distribution. The modal size class (in μm) is:



(A) 5 – 15

(B) 15 – 25

(C) 35 – 45

(D) 45 – 55

Q10. A buffer contains a weak acid ($pK_a = 4.20$) and its salt in a salt:acid molar ratio of 10:1. Using the Henderson–Hasselbalch equation, the pH is (log 10 = 1):

(A) 4.20

(B) 5.20

(C) 3.20

(D) 6.20

Q11. The sodium chloride equivalent (E) of a drug is 0.20. To render 30 mL of a 1.0% w/v solution of this drug isotonic, the amount of drug-equivalent NaCl already contributed (in grams) is:

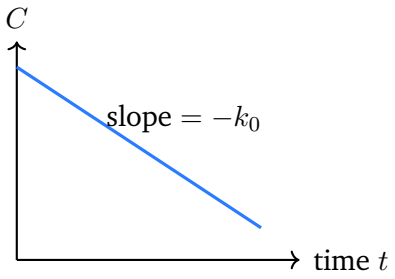
(A) 0.30

(B) 0.20

(C) 0.090

(D) 0.060

Q12. For a drug suspension, a plot of concentration C remaining versus time t is a straight line with a negative slope (figure). The decomposition follows:



(A) zero-order kinetics

(B) first-order kinetics

(C) second-order kinetics

(D) pseudo-first-order kinetics

Q13. A drug degrades by first-order kinetics with a half-life ($t_{1/2}$) of 200 days. The first-order rate constant k (per day) is approximately ($\ln 2 = 0.693$):

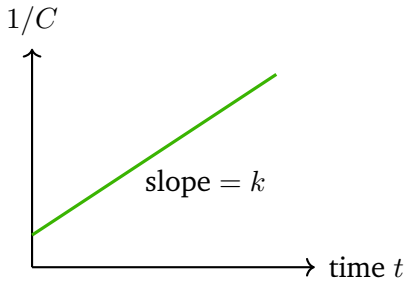
(A) 0.693

(B) 0.0693

(C) 3.47×10^{-3}

(D) 1.39×10^{-2}

Q14. For a decomposition reaction, a plot of $1/C$ (reciprocal of concentration) against time gives a straight line with positive slope (figure). The reaction order is:



(A) first order

(B) second order

(C) zero order

(D) third order

Q15. In accelerated stability testing, the rate of a degradation reaction increases with temperature according to the Arrhenius equation $k = A\,e^{-E_a/RT}$. A plot used to obtain the activation energy E_a is:

(A) k versus T

(B) $\ln k$ versus T

(C) k versus $1/T$

(D) $\ln k$ versus $1/T$

Q16. The Freundlich adsorption isotherm is written as $x/m = k\,C^{1/n}$. The linear (logarithmic) form used to determine the constants k and n is a plot of:

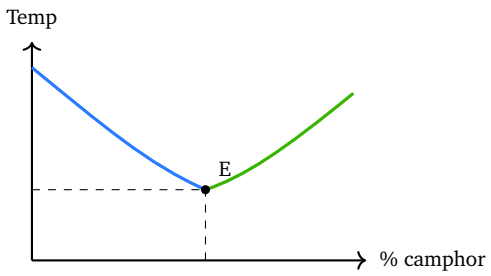
(A) $\log(x/m)$ versus $\log C$

(B) x/m versus C

(C) $C/(x/m)$ versus C

(D) $1/(x/m)$ versus $1/C$

Q17. The temperature–composition diagram below shows two liquidus curves meeting at the lowest point E. A mixture of menthol and camphor at composition E melts at a single, minimum temperature without leaving any solid. Point E is the:



(A) peritectic point

(B) triple point

(C) eutectic point

(D) inversion temperature

Q18. According to the Noyes–Whitney equation $dC/dt = \dfrac{DA}{h}(C_s - C)$, increasing the stirring (agitation) rate of the dissolution medium chiefly increases the dissolution rate by:

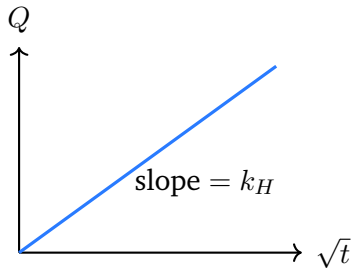
(A) raising the saturation solubility C_s

(B) raising the diffusion coefficient D directly

(C) increasing the surface area A

(D) decreasing the diffusion-layer thickness h

Q19. For a homogeneous (non-porous) ointment matrix, the cumulative amount of drug released Q plotted against $\sqrt{t}$ gives a straight line through the origin (figure). The release follows the:



(A) Hixson–Crowell model

(B) Higuchi (matrix-diffusion) model

(C) first-order model

(D) zero-order model

Q20. A drug shows *high* aqueous solubility and *high* intestinal permeability, and its dissolution is rapid. According to the Biopharmaceutics Classification System (BCS), it belongs to:

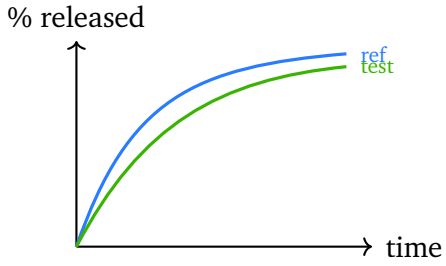
(A) Class I

(B) Class II

(C) Class III

(D) Class IV

Q21. In comparing the dissolution profiles below of a test and a reference product, the similarity factor f_2 is computed (figure). For the two profiles to be judged *similar*, the value of f_2 must lie between:



(A) 0 and 15

(B) 15 and 50

(C) 50 and 100

(D) 100 and 150

Q22. Talc is incorporated into a tablet granulation mainly to act as a:

(A) binder

(B) disintegrant

(C) diluent

(D) glidant / lubricant

Q23. Hydroxypropyl methylcellulose (HPMC) is most commonly used in tablet film coating as the:

(A) plasticiser

(B) film-forming polymer

(C) opacifier

(D) anti-tack agent

Q24. For uncoated tablets of average weight 250 mg, the IP/USP weight-variation limit is $\pm 5\%$. The acceptable weight range (mg) for an individual tablet is therefore:

(A) 245 – 255

(B) 240 – 260

(C) 237.5 – 262.5

(D) 225 – 275

Q25. Among the standard hard-gelatin capsule sizes for human use, the size with the *largest* fill volume (capacity) is:

(A) 000

(B) 0

(C) 3

(D) 5

Q26. The displacement value of a drug in cocoa butter is 2. To prepare 10 suppositories each containing 0.2 g of the drug using a 1 g mould (nominal cocoa-butter weight 1 g each), the mass of cocoa butter (g) required is:

(A) 8.0

(B) 9.0

(C) 10.0

(D) 11.0

Q27. In large-volume parenteral fluids, dextrose is frequently added primarily to:

(A) act as a preservative

(B) raise the pH to neutrality

(C) adjust the tonicity (and supply calories)

(D) chelate trace metal ions

Q28. An emulsion conducts electricity well when tested with a conductivity (electrical) test. This indicates that the continuous (external) phase is:

(A) oil, i.e. a w/o emulsion

(B) a non-conducting micro-emulsion

(C) a multiple w/o/w system

(D) water, i.e. an o/w emulsion

Q29. In a suspension, the sediment occupies 18 mL of an original 24 mL volume after settling. The sedimentation volume F is:

(A) 0.75

(B) 1.33

(C) 0.25

(D) 0.18

Q30. White petrolatum (soft paraffin) is classified as which type of ointment base?

(A) absorption base

(B) oleaginous (hydrocarbon) base

(C) water-removable (o/w) base

(D) water-soluble base

Q31. A fluid-energy (jet) mill achieves very fine particle-size reduction (down to a few micrometres) chiefly by:

(A) cutting with rotating knives

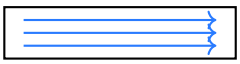
(B) compression between two rollers

(C) inter-particle attrition and impact in high-velocity fluid streams

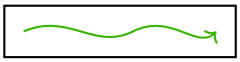
(D) crushing under a heavy reciprocating pestle

Q32. The two streamline sketches below show fluid flow in a pipe: pattern (i) has smooth parallel streamlines, pattern (ii) shows chaotic eddies. For

flow through a straight pipe, fully turbulent flow (pattern ii) is generally taken to occur when the Reynolds number exceeds:



(i) laminar



(ii) turbulent

(A) 100

(B) 500

(C) 2100

(D) 4000

Q33. In lyophilisation (freeze-drying), the *secondary* drying stage chiefly removes:

(A) residual bound (adsorbed) moisture by desorption

(B) all of the free ice by sublimation

(C) the eutectic solute crystals

(D) the inert nitrogen blanket

Q34. Biodegradable polymeric microspheres prepared from poly(lactic-co-glycolic acid) (PLGA) provide prolonged (controlled) drug release primarily because the drug is released as the polymer undergoes:

(A) instantaneous dissolution on contact with water

(B) slow hydrolytic degradation / erosion of the matrix

(C) magnetic targeting to the liver

(D) photochemical bleaching in tissue

Part B: Pharmacology & Toxicology

Q35. A drug is eliminated entirely by the kidney. At a steady plasma concentration of 4 mg/L its urinary excretion rate is measured as 8 mg/min. The renal clearance of the drug is:

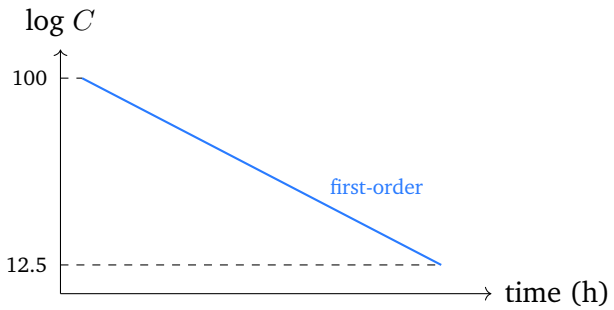
(A) 0.5 L/min

(B) 2 L/min

(C) 32 L/min

(D) 4 L/min

Q36. The semi-log plot below shows the plasma concentration of a drug after an IV bolus falling along a straight line from 100 mg/L to 12.5 mg/L over 6 hours (first-order elimination). The elimination half-life and rate constant are approximately:



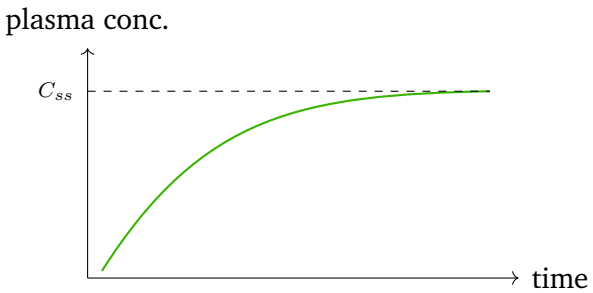
(A) $t_{1/2} = 6$ h, $k_e = 0.116$ h^{-1}

(B) $t_{1/2} = 1$ h, $k_e = 0.693$ h^{-1}

(C) $t_{1/2} = 3$ h, $k_e = 0.231$ h^{-1}

(D) $t_{1/2} = 2$ h, $k_e = 0.347$ h^{-1}

Q37. During a constant-rate IV infusion the plasma concentration approaches a plateau (steady state) as shown. For a drug with clearance 3 L/h, the infusion rate needed to maintain a target steady-state concentration C_{ss} of 5 mg/L is:



(A) 15 mg/h ($R_0 = CL \times C_{ss}$)

(B) 0.6 mg/h

(C) 1.67 mg/h

(D) 8 mg/h

Q38. Genetic polymorphism of N-acetyltransferase-2 divides patients into "slow" and "fast" acetylators. Compared with fast acetylators, slow acetylators receiving a standard dose of an acetylated drug (e.g. an antitubercular such as isoniazid or the vasodilator hydralazine) tend to have:

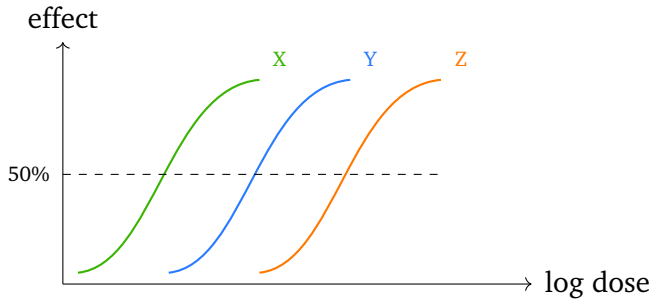
(A) Faster clearance and sub-therapeutic plasma levels

(B) No difference in plasma concentration

(C) Higher plasma drug concentrations and greater risk of concentration-dependent toxicity (e.g. peripheral neuropathy, drug-induced lupus)

(D) Reduced absorption from the gut

Q39. Three full agonists X, Y and Z acting at the same receptor give the graded log dose-response curves shown (same E_{max}). Ranking them from most to least potent gives:



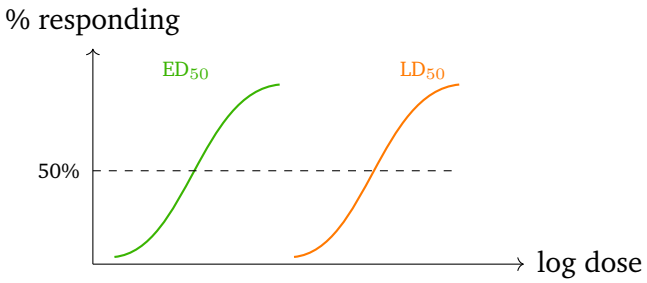
(A) Z > Y > X

(B) Y > X > Z

(C) All equally potent

(D) X > Y > Z (the leftmost curve has the lowest ED_{50} and is most potent)

Q40. The quantal curves below give an ED_{50} of 10 mg and an LD_{50} of 200 mg for a drug. Its therapeutic index (TI) is:



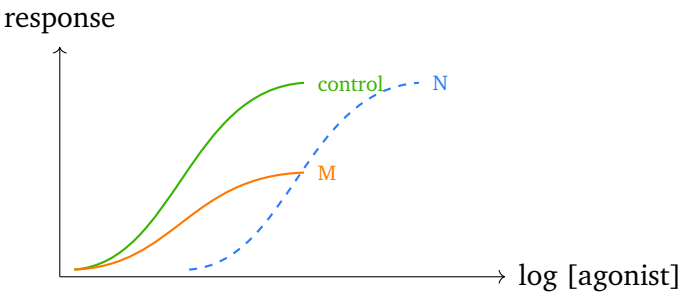
(A) 20 (TI = LD_{50}/ED_{50} = 200/10)

(B) 0.05

(C) 190

(D) 2000

Q41. The two curve changes below are produced by two antagonists added to a control agonist curve. Curve M (maximum depressed, not restored by more agonist) and curve N (parallel rightward shift, same maximum) respectively represent:



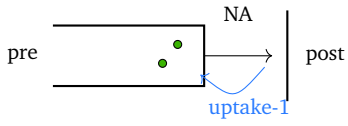
(A) M = competitive antagonist; N = non-competitive antagonist

(B) M = non-competitive (insurmountable) antagonist; N = competitive (surmountable) antagonist

(C) Both are competitive antagonists

(D) M = partial agonist; N = full agonist

Q42. At the adrenergic varicosity shown, the principal mechanism that terminates the action of released noradrenaline is:



(A) Hydrolysis by acetylcholinesterase in the synaptic cleft

(B) Diffusion alone, with no active transport involved

(C) Reuptake into the nerve terminal by the noradrenaline transporter (uptake-1/NET), followed by intraneuronal MAO metabolism

(D) Conversion to acetylcholine within the cleft

Q43. Among β-adrenoceptor antagonists, pindolol is distinguished by possessing intrinsic sympathomimetic activity (ISA). This property means that pindolol:

(A) Irreversibly alkylates the β receptor

(B) Is a pure (silent) antagonist with no agonist effect

(C) Blocks α_1 receptors in addition to β receptors

(D) Acts as a partial agonist, producing weak β stimulation at rest while blocking the receptor against high sympathetic drive, causing less resting bradycardia

Q44. Bethanechol is used to relieve post-operative non-obstructive urinary retention because it:

(A) Is a direct muscarinic agonist resistant to acetylcholinesterase that stimulates detrusor (bladder) smooth muscle contraction

(B) Blocks muscarinic receptors on the bladder

(C) Is a nicotinic receptor antagonist

(D) Acts as a β_3-adrenergic agonist relaxing the detrusor

Q45. Tamsulosin is preferred over non-selective α-blockers for benign prostatic hyperplasia because it is comparatively selective for the:

(A) β_2 receptor of bronchial muscle

(B) α_{1A}-adrenoceptor subtype of prostatic and bladder-neck smooth muscle, relaxing it with relatively little effect on vascular α_{1B} receptors (less postural hypotension)

(C) Muscarinic M_3 receptor of the detrusor

(D) α_2-adrenoceptor in the CNS

Q46. Ephedrine raises blood pressure partly by an indirect mechanism. Indirectly-acting sympathomimetic amines act mainly by:

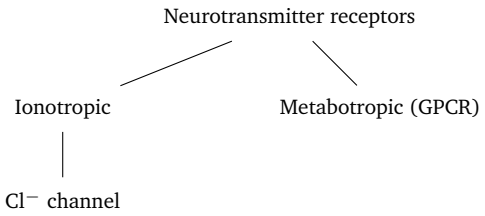
(A) Directly activating α and β receptors only, with no transmitter release

(B) Inhibiting monoamine oxidase exclusively

(C) Displacing and releasing stored noradrenaline from sympathetic nerve terminals (hence liable to tachyphylaxis as stores deplete)

(D) Blocking the noradrenaline transporter without releasing transmitter

Q47. In the neurotransmitter-receptor tree shown, the branch ending in "ionotropic $\rightarrow$ Cl^- channel" corresponds to which receptor, the target of benzodiazepines and barbiturates?



(A) β-adrenergic receptor (G_s-coupled)

(B) Muscarinic M_2 receptor (G_i-coupled)

(C) Dopamine D_1 receptor

(D) GABA-A receptor, a ligand-gated chloride channel whose opening hyperpolarises the neuron

Q48. Sertraline must not be combined with a monoamine oxidase inhibitor because the combination can precipitate serotonin syndrome. Sertraline relieves depression by:

(A) Selectively inhibiting the serotonin (5-HT) reuptake transporter, raising synaptic serotonin

(B) Blocking dopamine D_2 receptors

(C) Irreversibly inhibiting monoamine oxidase-A

(D) Activating GABA-A chloride channels

Q49. Carbamazepine is used for partial seizures, trigeminal neuralgia and bipolar disorder. Its principal anticonvulsant mechanism, and a notable pharmacokinetic property, are:

(A) Enhancement of GABA synthesis; it is a potent enzyme inhibitor

(B) T-type calcium channel blockade; no effect on hepatic enzymes

(C) Use-dependent blockade of voltage-gated sodium channels; it is a hepatic enzyme inducer (including of its own metabolism, autoinduction)

(D) NMDA receptor antagonism; it inhibits CYP enzymes

Q50. Dextromethorphan is widely used as a non-analgesic antitussive. It suppresses cough by:

(A) Strong μ-opioid agonism producing analgesia and euphoria like morphine

(B) A central action on the medullary cough centre (the d-isomer of an opioid with little analgesic or addictive activity at antitussive doses)

(C) Local anaesthesia of bronchial mucosa

(D) Histamine H_1 receptor blockade

Q51. Pregabalin and gabapentin are effective in neuropathic pain and partial seizures. Despite their names they do not act on GABA receptors; instead they:

(A) Enhance GABA reuptake into neurons

(B) Block voltage-gated sodium channels like phenytoin

(C) Inhibit GABA transaminase

(D) Bind the $\alpha_2\delta$ subunit of voltage-gated calcium channels, reducing presynaptic calcium influx and excitatory neurotransmitter release

Q52. High-potency typical antipsychotics such as fluphenazine cause more extrapyramidal symptoms than atypical agents because they:

(A) Produce strong blockade of nigrostriatal dopamine D_2 receptors with relatively weak 5-HT_{2A} antagonism

(B) Block only histamine H_1 receptors

(C) Are selective serotonin reuptake inhibitors

(D) Have no affinity for dopamine receptors at all

Q53. Propofol is a widely used intravenous induction agent producing rapid onset and rapid recovery. Its main molecular action is:

(A) NMDA glutamate receptor antagonism (the ketamine mechanism)

(B) μ-opioid receptor agonism

(C) Potentiation of GABA-A receptor chloride currents, enhancing inhibitory neurotransmission

(D) Voltage-gated sodium channel blockade

Q54. Isosorbide mononitrate is preferred over glyceryl trinitrate for chronic prophylaxis of angina mainly because it:

(A) Acts by blocking cardiac calcium channels

(B) Has high oral bioavailability (no significant first-pass metabolism) and a longer duration suitable for prophylaxis; nitrate tolerance is managed with a daily nitrate-free interval

(C) Increases myocardial oxygen demand to relieve pain

(D) Is reversed by protamine

Q55. Intravenous lidocaine used for ventricular arrhythmias is a Vaughan-Williams Class Ib agent. Its electrophysiological action is to:

(A) Block potassium channels and markedly prolong the action potential

(B) Block β-adrenergic receptors

(C) Block L-type calcium channels in the AV node

(D) Block sodium channels with fast onset/offset kinetics, shortening the action-potential duration and acting preferentially on depolarised (ischaemic) ventricular tissue

Q56. Amiloride is a potassium-sparing diuretic that, unlike spironolactone, is not an aldosterone antagonist. It acts by:

(A) Directly blocking the epithelial sodium channel (ENaC) in the late distal tubule/collecting duct, reducing Na^+ reabsorption and K^+ loss

(B) Inhibiting the $Na^+/K^+/2Cl^-$ cotransporter in the loop of Henle

(C) Antagonising the mineralocorticoid receptor

(D) Inhibiting carbonic anhydrase in the proximal tubule

Q57. Enoxaparin, a low-molecular-weight heparin, differs from unfractionated heparin in that it:

(A) Is taken orally and monitored by INR

(B) Directly inhibits thrombin without antithrombin

(C) Acts mainly through antithrombin to inhibit factor Xa (greater anti-Xa than anti-IIa activity), has a more predictable response and usually needs no routine aPTT monitoring

(D) Is reversed completely and reliably by vitamin K

Q58. Cetirizine causes far less sedation than the older antihistamine promethazine because cetirizine:

(A) Is an H_2-receptor antagonist

(B) Is a second-generation H_1 antagonist that penetrates the blood-brain barrier poorly, so central H_1 blockade and drowsiness are minimal

(C) Blocks muscarinic receptors strongly

(D) Is a mast-cell stabiliser with no receptor activity

Q59. Celecoxib was developed to reduce the gastrointestinal toxicity of traditional NSAIDs. Compared with non-selective NSAIDs it:

(A) Irreversibly acetylates platelet COX-1 like aspirin

(B) Inhibits lipoxygenase rather than cyclooxygenase

(C) Has no anti-inflammatory action

(D) Selectively inhibits COX-2 (sparing gastric COX-1 and platelet thromboxane), giving fewer GI ulcers but a recognised increase in cardiovascular thrombotic risk

Q60. Zileuton is used in asthma. Unlike the leukotriene-receptor antagonist montelukast, zileuton acts by:

(A) Inhibiting 5-lipoxygenase, thereby blocking the synthesis of all leukotrienes from arachidonic acid

(B) Blocking the cysteinyl-leukotriene $CysLT_1$ receptor

(C) Inhibiting cyclooxygenase-2

(D) Stabilising mast cells to prevent histamine release

Q61. Misoprostol, a synthetic prostaglandin E_1 analogue, is co-prescribed with NSAIDs to prevent gastric ulcers because it:

(A) Blocks histamine H_2 receptors on parietal cells

(B) Neutralises gastric acid chemically as an antacid

(C) Replaces the protective gastric prostaglandins that NSAIDs suppress, inhibiting acid secretion and promoting mucus/bicarbonate and mucosal blood flow

(D) Irreversibly inhibits the H^+/K^+-ATPase proton pump

Q62. Ceftriaxone, a third-generation cephalosporin, is used for serious infections including bacterial meningitis. As a β-lactam it acts by:

(A) Inhibiting the 50S ribosomal subunit

(B) Binding penicillin-binding proteins and inhibiting peptidoglycan cross-linking (cell-wall synthesis); its good CSF penetration suits meningitis

(C) Inhibiting DNA gyrase

(D) Disrupting folate synthesis

Q63. Ciprofloxacin, a fluoroquinolone, exerts its bactericidal effect by:

(A) Binding the 30S ribosomal subunit and causing mRNA misreading

(B) Inhibiting cell-wall transpeptidation

(C) Inhibiting dihydrofolate reductase

(D) Inhibiting bacterial DNA gyrase (topoisomerase II) and topoisomerase IV, blocking DNA supercoiling and replication

Q64. Metronidazole is effective against anaerobic bacteria and protozoa such as *Giardia* and *Entamoeba*. Its selective toxicity arises because:

(A) Its nitro group is reduced inside anaerobic/microaerophilic organisms to reactive cytotoxic intermediates that damage microbial DNA, a reduction that does not occur appreciably in aerobic host cells

(B) It inhibits the host ribosome selectively

(C) It blocks the bacterial cell wall like penicillin

(D) It chelates iron required by aerobic bacteria

Q65. Levothyroxine is the standard replacement therapy for hypothyroidism. Pharmacologically it:

(A) Inhibits thyroid peroxidase to lower hormone synthesis

(B) Is synthetic T_4 that is converted peripherally to the more active T_3 and acts on nuclear thyroid-hormone receptors to restore the euthyroid state; it has a long half-life allowing once-daily dosing

(C) Blocks peripheral conversion of T_4 to T_3

(D) Destroys thyroid tissue by emitting radiation

Q66. A child with acute iron overdose is treated with a specific chelating antidote. Which antidote-poison pairing is the CORRECT match for iron poisoning?

(A) Iron poisoning $\rightarrow$ dimercaprol (BAL)

(B) Iron poisoning $\rightarrow$ D-penicillamine

(C) Iron poisoning $\rightarrow$ deferoxamine (desferrioxamine), which chelates ferric iron for renal excretion

(D) Iron poisoning $\rightarrow$ atropine

Q67. Co-administering a potent CYP3A4 inhibitor (for example ketoconazole or grapefruit-juice constituents) with a drug metabolised by CYP3A4 typically results in:

(A) Faster metabolism and lower plasma levels of the substrate

(B) Induction of the enzyme over several days

(C) No pharmacokinetic change

(D) Reduced metabolism of the substrate, raising its plasma concentration and the risk of dose-related toxicity

Q68. When assessing whether a drug crosses the placenta to reach the fetus, the property that most favours transplacental passage by passive diffusion is the drug being:

(A) Lipid-soluble, of low molecular weight, un-ionised and poorly protein-bound

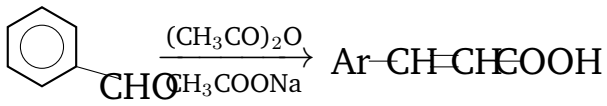
(B) Highly ionised and water-soluble

(C) Of very high molecular weight (e.g. heparin)

(D) Extensively bound to maternal plasma proteins

Part C: Pharmaceutical & Medicinal Chemistry

Q69. The scheme below shows an aromatic aldehyde condensing with an aliphatic acid anhydride in the presence of the sodium salt of the acid, giving an α,β-unsaturated (cinnamic-type) acid after work-up.



This synthesis of cinnamic acid from benzaldehyde and acetic anhydride is the:

(A) Claisen–Schmidt condensation

(B) Cannizzaro reaction

(C) Perkin reaction

(D) Wittig reaction

Q70. An aryl ketone is heated with amalgamated zinc (Zn–Hg) and concentrated hydrochloric acid, which reduces the carbonyl group entirely to a methylene (CH_2) under **acidic** conditions. This deoxygenation of a C=O group is the:

(A) Clemmensen reduction

(B) Wolff–Kishner reduction

(C) Rosenmund reduction

(D) Bouveault–Blanc reduction

Q71. An acyl azide ($RCON_3$), on gentle heating, loses nitrogen and rearranges to an isocyanate (R–N=C=O), which on hydrolysis gives a primary amine with one fewer carbon. This thermal degradation is the:

(A) Hofmann bromamide reaction

(B) Schmidt reaction

(C) Lossen rearrangement

(D) Curtius rearrangement

Q72. A ketone is first converted to an enamine with a secondary amine (e.g. pyrrolidine); the nucleophilic enamine is then alkylated or acylated, and hydrolysis regenerates the carbonyl bearing the new α-substituent. This route to clean monoalkylation at the α-carbon is the:

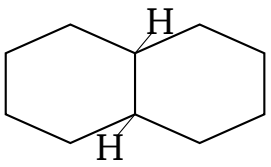
(A) Mannich reaction

(B) Stork enamine reaction

(C) Michael addition

(D) Reformatsky reaction

Q73. The decalin skeleton drawn below consists of two fused cyclohexane chairs sharing an edge. In the **trans** isomer the two ring-fusion hydrogens are on opposite faces and both rings are locked in the rigid chair–chair arrangement.



Compared with *cis*-decalin, *trans*-decalin is more stable mainly because:

(A) all ring-fusion bonds are equatorial–equatorial, minimising 1,3-diaxial strain

(B) it is aromatic whereas the cis form is not

(C) it can freely ring-flip while the cis form cannot

(D) it contains an additional double bond

Q74. A secondary alcohol (e.g. a steroidal hydroxyl) is selectively oxidised to the corresponding ketone using excess acetone (the hydride acceptor) and an aluminium alkoxide catalyst, without touching sensitive C=C bonds. This mild oxidation is the:

(A) Rosenmund reduction

(B) Meerwein–Ponndorf–Verley reduction

(C) Oppenauer oxidation

(D) Swern oxidation

Q75. Arrange the following nitrogen compounds in order of **decreasing** basicity (in water): (i) ethylamine, (ii) aniline, (iii) acetamide.

(A) (iii) $>$ (ii) $>$ (i)

(B) (ii) $>$ (i) $>$ (iii)

(C) (i) $>$ (iii) $>$ (ii)

(D) (i) $>$ (ii) $>$ (iii)

Q76. The transformation below cleaves the carbon–carbon double bond of an alkene into two carbonyl fragments, using ozone followed by a reductive work-up (Zn / H_2O).

$$R_2C{=}CR'_2 \xrightarrow[\text{(ii) Zn, } H_2O]{\text{(i) } O_3} R_2C{=}O \;+\; O{=}CR'_2$$

This oxidative double-bond cleavage to two carbonyl compounds is called:

(A) hydroboration–oxidation

(B) Wacker oxidation

(C) dihydroxylation

(D) ozonolysis

Q77. Two molecules of an alkyl halide react with metallic sodium in dry ether to give a symmetrical alkane containing twice the carbons, e.g. 2 CH_3CH_2Br $\rightarrow$ $CH_3CH_2CH_2CH_3$. This coupling is the:

(A) Wurtz reaction

(B) Kolbe electrolysis

(C) Frankland reaction

(D) Corey–House synthesis

Q78. For both S_N1 and S_N2 reactions, a better leaving group is one whose conjugate base is more stable (weaker base). Which of the following is the **best** leaving group?

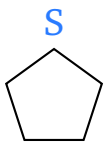
(A) –OH (hydroxide)

(B) –OTs (tosylate / *p*-toluenesulfonate)

(C) $–NH_2$ (amide)

(D) $–OCH_3$ (methoxide)

Q79. The aromatic five-membered heterocycle drawn below contains one sulfur atom contributing a lone pair to the aromatic sextet (6 π electrons). It is found in the antibiotic ticarcillin and the antiplatelet drug clopidogrel.



Identify this heterocycle.

(A) furan

(B) pyrrole

(C) imidazole

(D) thiophene

Q80. Addition of HBr to propene in the **presence of organic peroxides** gives 1-bromopropane (anti-Markovnikov) instead of 2-bromopropane, because the reaction now proceeds by a free-radical chain in which the more stable secondary radical forms. This peroxide effect is the:

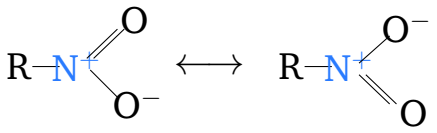
(A) Markovnikov addition

(B) Kharasch (peroxide) effect

(C) Saytzeff orientation

(D) Hofmann orientation

Q81. The two equivalent resonance structures of the nitro group are shown below; the negative charge is shared equally between the two oxygens and the nitrogen carries a formal positive charge.



The principal consequence of this delocalisation is that the nitro group is:

(A) a strong electron donor by resonance

(B) non-polar, with the two N–O bonds of different length

(C) a powerful electron-withdrawing group, with both N–O bonds equal and the ring strongly deactivated

(D) basic, readily protonated at nitrogen

Q82. An alkyl chloride or bromide is converted to the corresponding alkyl **fluoride** by heating with an inorganic fluoride such as AgF, Hg_2F_2 or SbF_3. This halogen-exchange route to organofluorine compounds is the:

(A) Swarts reaction

(B) Finkelstein reaction

(C) Hunsdiecker reaction

(D) Wurtz–Fittig reaction

Q83. D-Glucose and D-mannose differ in configuration at only **one** of their several stereocentres (C2). Such a pair of diastereomeric sugars that differ at a single stereocentre is termed a pair of:

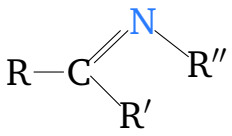
(A) epimers

(B) enantiomers

(C) anomers

(D) conformers

Q84. When a primary amine condenses with an aldehyde or ketone (with loss of water), the carbon–nitrogen double-bonded product drawn below is formed.



This C=N containing condensation product is a(n):

(A) enamine

(B) imine (Schiff base)

(C) amide

(D) nitrile

Q85. Erythromycin, azithromycin and clarithromycin share a large macrocyclic lactone ring bearing sugar residues and inhibit bacterial protein synthesis at the 50S ribosomal subunit. As a chemical class they are:

(A) tetracyclines

(B) aminoglycosides

(C) β-lactams

(D) macrolides

Q86. Saquinavir, ritonavir and lopinavir (the "-navir" drugs) are antiretroviral agents that act by:

(A) inhibiting HIV aspartic protease, blocking maturation of viral polyproteins

(B) inhibiting reverse transcriptase as nucleoside analogues

(C) blocking the influenza neuraminidase

(D) alkylating viral DNA

Q87. Synthetic glucocorticoids such as dexamethasone and betamethasone are far more potent anti-inflammatories than cortisol. A key structural

modification responsible for the increased potency and longer duration is:

(A) removal of the steroid nucleus entirely

(B) replacement of the four rings by a single benzene ring

(C) introduction of a 9α-fluoro substituent and a 16-methyl group on the steroid skeleton

(D) conversion of the molecule into a quaternary ammonium salt

Q88. In enzyme-inhibition assays, the concentration of an inhibitor that reduces the enzyme (or receptor) activity to half its maximum is reported as the:

(A) partition coefficient (log$P$)

(B) IC_{50} (half-maximal inhibitory concentration)

(C) Hammett σ constant

(D) molar refractivity

Q89. Among the non-covalent forces that hold a drug at its receptor, the weakest and most distance-dependent (varying as $1/r^6$), arising from transient induced dipoles, are the:

(A) van der Waals (London dispersion) forces

(B) ionic (electrostatic) bonds

(C) covalent bonds

(D) hydrogen bonds

Q90. Thalidomide is the classic cautionary example in chiral drug design because:

(A) it has no stereocentre at all

(B) both enantiomers are completely inactive

(C) it is achiral and cannot be resolved

(D) one enantiomer is sedative while the other is teratogenic, and the two interconvert (racemise) *in vivo*

Q91. Chloroquine and related 4-aminoquinoline antimalarials are thought to kill the malaria parasite chiefly by:

(A) inhibiting fungal CYP51

(B) preventing the detoxifying polymerisation of toxic haem into inert haemozoin in the parasite food vacuole

(C) blocking the bacterial 30S ribosome

(D) inhibiting human HMG-CoA reductase

Q92. Tetracyclines inhibit bacterial protein synthesis at the 30S ribosome, but their oral absorption is markedly reduced when taken with milk or antacids. The chemical reason is that tetracyclines:

(A) are destroyed by gastric acid only

(B) are too lipophilic to dissolve

(C) chelate di- and trivalent metal cations (Ca^{2+}, Mg^{2+}, Al^{3+}, Fe^{2+}) to form poorly absorbed complexes

(D) are inactivated by light alone

Q93. Many ester and amide drugs degrade by hydrolysis that is catalysed by both H^+ and OH^-. A plot of the observed degradation rate constant against pH (a pH–rate profile) for such a drug is typically:

(A) a straight horizontal line independent of pH

(B) monotonically increasing with pH only

(C) monotonically decreasing with pH only

(D) V-shaped (or U-shaped), with a pH of maximum stability at the minimum

Q94. In collision theory of reaction rates, not every collision having energy

$\geq E_a$ leads to reaction. The fraction that does, accounting for the requirement that molecules be correctly aligned, is expressed by the:

(A) steric (orientation) factor, P

(B) partition coefficient

(C) rate-determining step alone

(D) ionic strength

Q95. Which pharmaceutical inorganic compound is used (with sodium nitrite) as an **antidote in cyanide poisoning**, acting as a sulfur donor that converts cyanide to the far less toxic thiocyanate?

(A) calcium carbonate

(B) sodium thiosulfate

(C) magnesium sulfate

(D) potassium iodide

Q96. A diester with a long enough chain (e.g. a diethyl adipate) undergoes an **intramolecular** base-promoted ester condensation to give a cyclic β-keto ester. This intramolecular Claisen condensation is the:

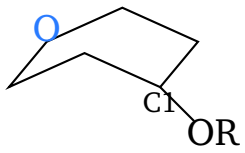
(A) Reformatsky reaction

(B) Stobbe condensation

(C) Dieckmann condensation

(D) Darzens reaction

Q97. In the pyranose chair drawn below, an electronegative substituent (such as –OR or a halogen) at the anomeric carbon (C1, next to the ring oxygen) shows an unexpected preference for the **axial** rather than the equatorial position.



This stereoelectronic preference for the axial orientation at the anomeric centre is known as the:

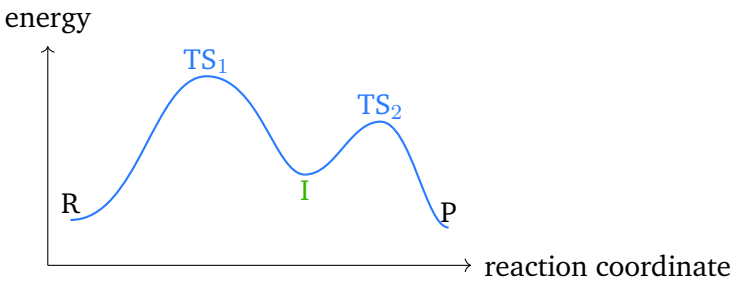
(A) 1,3-diaxial strain

(B) gauche effect

(C) Hofmann effect

(D) anomeric effect

Q98. The reaction-coordinate diagram below shows a two-step reaction passing through an intermediate (I). The **first** barrier (TS_1) is higher than the second (TS_2).



For this profile, which statement is correct?

(A) The reaction is concerted with no intermediate.

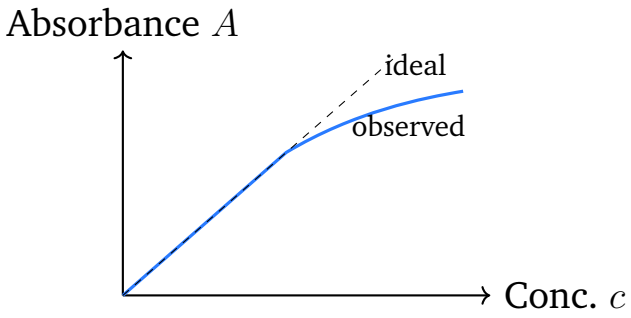
(B) The second step is rate-determining because TS_2 comes later.

(C) The intermediate I is a transition state and cannot be isolated.

(D) The first step (over TS_1, the higher barrier) is the rate-determining step.

Part D: Pharmaceutical Analysis & Quality Assurance

Q99. The plot below shows absorbance against concentration for a coloured drug measured at its λ_{max}. The line is straight at low concentration but bends *towards the concentration axis* at higher concentration, as drawn. This negative deviation from the Beer–Lambert law at high concentration is most commonly attributed to:



(A) The path length increasing with concentration

(B) The detector becoming more sensitive at high signal

(C) A rise in the molar absorptivity with dilution

(D) Solute–solute interactions and changes in refractive index at high concentration

Q100. In diazotization titrimetry the titrant is sodium nitrite, in which one NO_2^- reacts with one primary aromatic amine (a $1:1$ reaction). The normality of a 0.10 M sodium nitrite solution used for such assays is therefore:

(A) 0.10 N

(B) 0.20 N

(C) 0.050 N

(D) 0.010 N

Q101. During the EDTA titration of calcium in a sample that also contains traces of interfering heavy-metal ions, a small amount of *potassium cyanide* is added before titration. The function of the cyanide here is to act as a:

(A) Redox indicator that signals the end point

(B) Buffer that fixes the solution pH at 10

(C) Masking agent that complexes the interfering metals so they are not titrated

(D) Primary standard for the EDTA

Q102. Affinity chromatography, widely used to purify enzymes and antibodies, achieves separation because the stationary phase carries an immobilised ligand that:

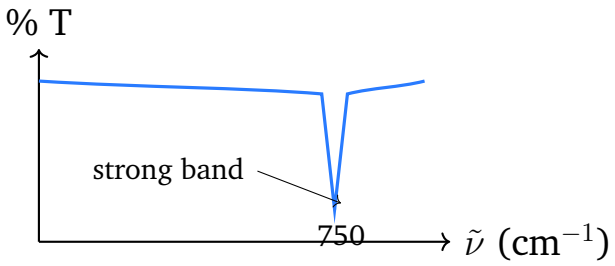
(A) Sieves molecules strictly by their size

(B) Binds the target molecule through a specific, reversible biological interaction

(C) Separates analytes only by their net electrical charge

(D) Volatilises the analytes in a heated injector

Q103. The schematic IR spectrum below highlights a strong band near **750 cm^{-1}** in the low-wavenumber (fingerprint) region. Strong absorption in the 690–900 cm^{-1} range of this type is most diagnostic of:



(A) Aromatic C–H out-of-plane bending (ring-substitution pattern)

(B) O–H stretch of a free alcohol

(C) C=O stretch of an aldehyde

(D) N–H stretch of an amine

Q104. A redox indicator is suitable for a particular titration only when its standard transition (redox) potential lies:

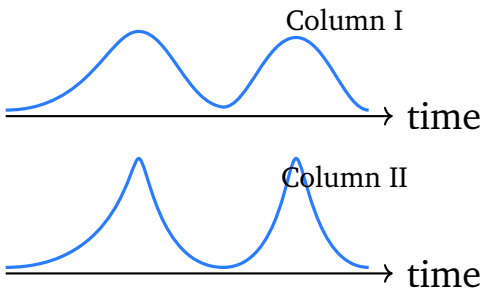
(A) Far below the potential of the analyte at all points

(B) Within the steep potential break that occurs near the equivalence point

(C) Exactly at zero volts regardless of the system

(D) Above the potential of the strongest oxidant present

Q105. The two chromatograms below were obtained for the same pair of analytes on two columns. Column II (lower trace) gives sharper, narrower peaks than Column I while the retention times are essentially unchanged. The improved resolution on Column II is chiefly due to:



(A) A larger void volume in Column II

(B) A higher mobile-phase flow rate causing band broadening

(C) A decrease in the selectivity factor α

(D) A greater number of theoretical plates (higher efficiency), narrowing the peaks

Q106. In fluorimetry, the fluorescence intensity rises with analyte concentration at low levels but then *falls off* at high concentration, so the calibration curve bends back. This loss of linearity at high concentration is mainly due to the:

(A) Stokes shift increasing with concentration

(B) Excitation lamp drifting in intensity

(C) Inner-filter effect (self-absorption / reabsorption of emitted light)

(D) Monochromator slit widening automatically

Q107. In the standardisation of permanganate against oxalate the balanced reaction is $2\mathrm{MnO_4^-} + 5\mathrm{C_2O_4^{2-}} + 16\mathrm{H^+} \rightarrow 2\mathrm{Mn^{2+}} + 10\mathrm{CO_2} + 8\mathrm{H_2O}$. How many moles of oxalate are oxidised by 1 mole of permanganate?

(A) 2.0 mol

(B) 2.5 mol

(C) 5.0 mol

(D) 0.4 mol

Q108. A short *guard column* packed with the same material is placed just ahead of the analytical HPLC column. Its main purpose is to:

(A) Trap particulates and strongly retained contaminants, protecting and prolonging the analytical column

(B) Increase the flow rate delivered to the detector

(C) Act as the main separation column

(D) Serve as the sample injection loop

Q109. For *para*-xylene (1,4-dimethylbenzene), in which the molecule's symmetry makes all four aromatic protons equivalent and both methyl groups equivalent, how many distinct ^{1}H NMR signals are expected?

(A) 4

(B) 3

(C) 6

(D) 2

Q110. For the titration of a weak acid with a strong base, the equivalence-point solution is mildly *alkaline*. The most appropriate visual indicator, whose colour-change interval brackets that basic equivalence pH, is:

(A) Methyl orange (pH 3.1–4.4)

(B) Methyl red (pH 4.4–6.2)

(C) Phenolphthalein (pH 8.3–10.0)

(D) Bromophenol blue (pH 3.0–4.6)

Q111. In the mass spectrum of an alcohol, a prominent fragment ion appears 18 mass units below the molecular ion (at $m/z = \mathrm{M}-18$). This loss of 18 is most commonly attributed to the elimination of:

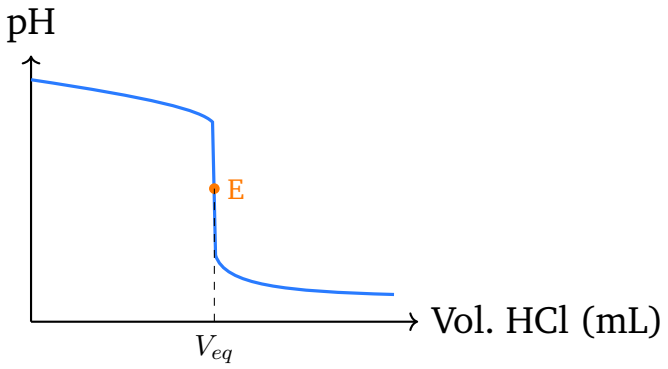
(A) A methyl radical (CH_3)

(B) A molecule of water (H_2O)

(C) A carbon monoxide molecule (CO)

(D) A hydroxyl radical only as OH^-

Q112. The curve below shows pH versus volume of *acid* added when a strong base is titrated with a strong acid. The pH starts high and falls, with a steep drop at the point marked **E**. Point **E** corresponds to the:



(A) Equivalence point, where moles of acid added equal moles of base initially present

(B) Point of maximum buffer capacity

(C) Half-neutralisation point

(D) Point where the indicator is fully oxidised

Q113. In flame photometry, when two alkali metals are present together, the emission reading for one element may be falsely raised because radiation from the other element overlaps. This kind of error is best classified as:

(A) A path-length error

(B) A change in the analyte's molar mass

(C) A spectral (radiation) interference between the elements

(D) A purely random noise fluctuation

Q114. For the trace determination of halogenated pesticide residues, the gas-chromatographic detector of choice, owing to its very high sensitivity to electronegative (halogen-containing) compounds, is the:

(A) Flame ionization detector (FID)

(B) Thermal conductivity detector (TCD)

(C) Refractive-index detector (RID)

(D) Electron-capture detector (ECD)

Q115. In the AAS determination of calcium, phosphate in the sample lowers the signal by forming a refractory calcium phosphate that does not atomise fully. Adding excess *lanthanum chloride* corrects this by:

(A) Increasing the wavelength of the hollow-cathode lamp

(B) Acting as a releasing agent that preferentially binds phosphate, freeing calcium to atomise

(C) Cooling the flame to reduce ionisation

(D) Serving as the internal standard for emission

Q116. In ion-exchange chromatography of a protein mixture, bound analytes are commonly eluted by gradually *increasing the salt (ionic-strength) concentration* of the mobile phase. The increasing salt elutes the proteins because the added counter-ions:

(A) Raise the column temperature to volatilise the proteins

(B) Reduce the path length of the detector cell

(C) Compete with the bound proteins for the charged sites, displacing them in order of binding strength

(D) Increase the molecular size of the proteins

Q117. To assay an antacid containing insoluble *magnesium hydroxide*, a measured excess of standard HCl is added to dissolve and react with it, and the unreacted acid is then titrated with standard NaOH. This indirect approach is used because:

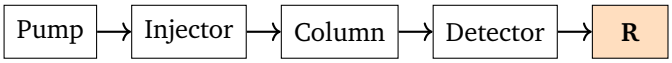
(A) The solid reacts slowly/incompletely with titrant in a direct titration, so reacting with excess acid first ensures completion

(B) NaOH cannot react with HCl

(C) Magnesium hydroxide is a primary standard

(D) Direct titration would need no indicator at all

Q118. In the liquid-chromatograph block diagram below, identify the unit labelled **R**, which is placed *after* the detector and converts the detector output into the printed/stored chromatogram:



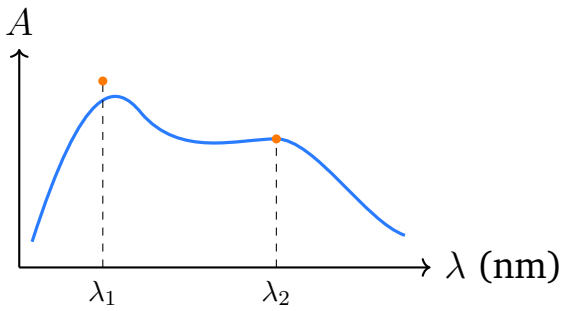
(A) Mobile-phase reservoir

(B) Recorder / data-acquisition system

(C) High-pressure pump

(D) Guard column

Q119. The UV absorption curve below shows a drug with two maxima, at λ_1 and λ_2. Some pharmacopoeial identity tests specify the *ratio* of absorbances $A_{\lambda_1}/A_{\lambda_2}$. From the figure, where A at λ_1 is about 0.80 and A at λ_2 is about 0.40, this absorbance ratio is approximately:



(A) 0.50

(B) 1.0

(C) 0.40

(D) 2.0

Q120. After developing a TLC plate of colourless lipophilic compounds, the analyst places the plate in a closed tank containing *iodine crystals* and brownish spots appear. Iodine vapour works here as a:

(A) Mobile phase that re-develops the plate

(B) Fluorescent coating built into the silica

(C) Non-destructive visualising (detection) reagent for organic spots

(D) Primary standard for calibrating R_f

Q121. The Clark electrode, used to measure dissolved oxygen, is an *amperometric* sensor. It quantifies oxygen by measuring the:

(A) Current produced by the electrochemical reduction of O_2 at a fixed applied potential

(B) Mass-to-charge ratio of oxygen ions

(C) pH change caused by dissolved oxygen

(D) Light emitted when oxygen is excited in a flame

Q122. During routine QC testing a batch assay value falls *outside* the registered acceptance limits. Under GMP/QA practice, the first formal step the laboratory must take is to:

(A) Immediately release the batch and ignore the value

(B) Initiate a documented out-of-specification (OOS) investigation before any decision on the batch

(C) Average the failing result with passing results to bring it into range

(D) Discard the sample and report no result

Part E: Pharmacognosy & Natural Products

Q123. A pharmacognosist arranges crude drugs strictly according to the botanical position of the source plant, placing all drugs from the family Apocynaceae together and all from the Solanaceae together, regardless of the plant part or active constituent. This basis of grouping is called:

(A) Morphological classification

(B) Taxonomical (botanical) classification

(C) Chemical classification

(D) Pharmacological classification

Q124. In the morphological classification of crude drugs, "organized" drugs are direct plant organs with a definite cellular structure. Which of the following sets contains **only organized** drugs?

(A) Agar, mastic, isabgol husk

(B) Tragacanth, kino, catechu

(C) Dill fruit, ginger rhizome, quassia wood

(D) Gum acacia, aloe, opium

Q125. Modern systems of arranging crude drugs use the distribution of chemical constituents (and serological/genetic data) as markers of natural relationship among plants. The classification that groups plants on the basis of their characteristic secondary-metabolite chemistry is termed:

(A) Alphabetical classification

(B) Morphological classification

(C) Pharmacological classification

(D) Chemotaxonomic classification

Q126. Caffeine, a purine alkaloid used as a central-nervous-system stimulant, is obtained chiefly from the dried ripe seeds of which plant?

(A) *Coffea arabica*

(B) *Papaver somniferum*

(C) *Atropa belladonna*

(D) *Rauwolfia serpentina*

Q127. The non-depolarising skeletal-muscle relaxant tubocurarine, a bis-benzylisoquinoli alkaloid and the active principle of the arrow-poison "curare", is obtained from the dried bark and stem of which plant?

(A) *Cinchona officinalis*

(B) *Chondrodendron tomentosum*

(C) *Ephedra gerardiana*

(D) *Lobelia inflata*

Q128. Aconitine, the highly toxic constituent of the dried roots of *Aconitum napellus*, is a diterpenoid (terpenoid-derived) alkaloid in which the nitrogen is not contributed by an amino-acid skeleton. On this basis aconitine is best classified as a:

(A) True alkaloid with amino-acid-derived ring nitrogen

(B) Protoalkaloid

(C) Pseudo-alkaloid (terpenoid alkaloid)

(D) Glycoalkaloid

Q129. During preliminary phytochemical screening, an aqueous-acidic plant extract gives a **reddish-brown precipitate** on addition of a reagent prepared from iodine and potassium iodide. This positive precipitation reaction for alkaloids is the:

(A) Bornträger's test

(B) Molisch's test

(C) Legal's test

(D) Wagner's test

Q130. Oleandrin is a cardiac (cardenolide) glycoside whose steroidal aglycone carries a five-membered α,β-unsaturated butenolide ring. Its source is the dried leaf of which plant?

(A) *Nerium oleander*

(B) *Glycyrrhiza glabra*

(C) *Cassia angustifolia*

(D) *Dioscorea deltoidea*

Q131. Cascara sagrada (the dried bark of *Rhamnus purshiana*) is a purgative whose action is due to anthraquinone (cascaroside) glycosides. The presence of these anthraquinone derivatives is confirmed by a pink-to-red colour in the ammoniacal layer in which reaction?

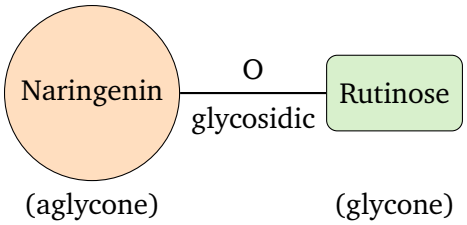
(A) Keller–Kiliani test

(B) Bornträger's test

(C) Baljet test

(D) Liebermann–Burchard test

Q132. The schematic below represents naringin, the bitter flavanone glycoside of grapefruit peel (*Citrus paradisi*), drawn as its aglycone joined to a disaccharide through an O-glycosidic bond.



The non-sugar half "naringenin" and the bitter taste it imparts make naringin chemically a:

(A) Cardiac (steroidal) glycoside

(B) Anthraquinone glycoside

(C) Flavonoid (flavanone) glycoside

(D) Cyanogenetic glycoside

Q133. Frangula bark (*Rhamnus frangula*) is used as a mild laxative. Its purgative glycosides, glucofrangulins, on hydrolysis liberate which class of aglycone, accounting for the laxative effect that is shared with senna and rhubarb?

(A) Steroidal cardenolide aglycones

(B) Triterpenoid saponin aglycones

(C) Cyanogenetic (cyanohydrin) aglycones

(D) Anthraquinone (emodin-type) aglycones

Q134. Dill (the dried ripe fruit of *Anethum graveolens*) yields a volatile oil used as a carminative. Its characteristic odour is due chiefly to which monoterpene ketone, also abundant in caraway?

(A) Carvone

(B) Eugenol

(C) Cinnamaldehyde

(D) Menthol

Q135. Ginger (the dried rhizome of *Zingiber officinale*) owes its pungency to non-volatile constituents present in its oleoresin. These pungent principles are:

(A) Zingiberene and bisabolene (the volatile sesquiterpenes)

(B) Gingerols and shogaols

(C) Curcuminoids

(D) Piperine and chavicine

Q136. Mastic, a pale-yellow oleoresin used as a varnish base and dental material, is obtained as a pathological exudate from incisions in the stem of which plant?

(A) *Commiphora molmol*

(B) *Boswellia serrata*

(C) *Pistacia lentiscus*

(D) *Ferula asafoetida*

Q137. Agar, obtained from the cell walls of certain red seaweeds (*Gelidium* and *Gracilaria* species), is valued in microbiology because a 1.5% solution sets to a firm gel that is not liquefied by most bacteria. Agar is chemically a:

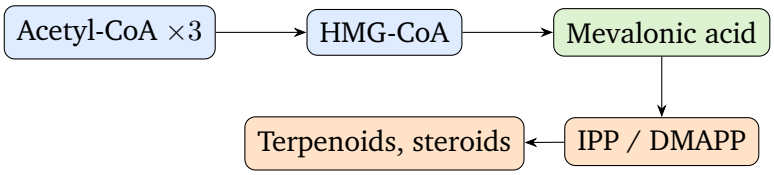
(A) Fructan (inulin-type) polysaccharide

(B) Gum exudate of glucuronic-acid units

(C) Tannin-rich glycosidic resin

(D) Galactan (sulphated galactose polysaccharide)

Q138. The biosynthetic scheme below outlines the formation of the universal C5 isoprene building block in plants. Identify the pathway depicted.



(A) Acetate–mevalonate pathway giving terpenoids and steroids

(B) Shikimic acid pathway giving aromatic amino acids

(C) Acetate–malonate pathway giving fatty acids

(D) Pentose phosphate pathway giving sugars

Q139. Gallic acid and the hydrolysable gallotannins of crude drugs such as myrobalan are aromatic compounds built from a C_6-C_1 unit. Through which biosynthetic pathway is this aromatic ring of gallic acid generated in the plant?

(A) Acetate–mevalonate pathway

(B) Shikimic acid pathway

(C) Glycolytic pathway

(D) Citric acid cycle

Q140. For drugs such as quassia and gentian, the standardisation parameter expressed as "the reciprocal of the highest dilution (in g) of the drug that

still has a recognisable bitter taste, relative to a quinine hydrochloride standard" is the:

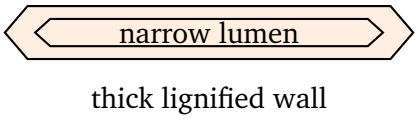
(A) Swelling index

(B) Foaming (afrosimetric) index

(C) Bitterness value

(D) Acid value

Q141. The microscopical sketch below shows an element commonly seen in powdered barks and woods: an elongated, thick-walled, pointed cell with a narrow lumen whose walls turn pink-red with phloroglucinol/HCl. This diagnostic element is a:



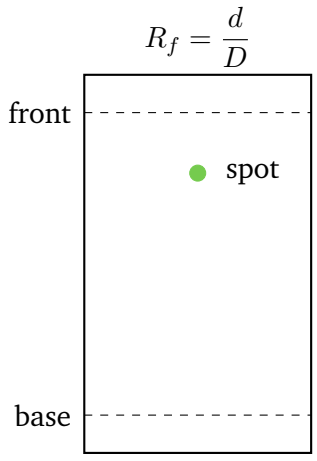
(A) A starch granule

(B) A non-lignified cotton (unicellular) trichome

(C) A pitted parenchyma cell

(D) A lignified sclerenchymatous fibre

Q142. The TLC plate below is a fingerprint of a herbal extract, marked with the spotting line (base), one resolved spot, and the solvent front.



If the spot travels $d = 3.2$ cm while the solvent front D advances 4.0 cm from the baseline, the R_f value of the spot is:

(A) 0.80

(B) 1.25

(C) 0.32

(D) 0.50

Part F: Pharmaceutical Biotechnology & Microbiology

Q143. A type II restriction endonuclease recognises the palindrome AAGCTT and cleaves each strand between the two A residues (A|AGCTT). The product of this enzyme is:

(A) Blunt-ended fragments, because the cut is at the centre of the site

(B) Fragments with 5′ AGCT cohesive overhangs that can re-anneal

(C) Fragments with 3′ recessed overhangs lacking any phosphate

(D) Single-stranded DNA circles with no free ends

Q144. To clone a eukaryotic gene so that it can be expressed in a bacterial host without introns, one first prepares cDNA from the corresponding mature mRNA. The enzyme that synthesises the first DNA strand on this RNA template is:

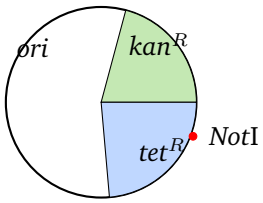
(A) Terminal deoxynucleotidyl transferase

(B) Polynucleotide kinase

(C) Reverse transcriptase (RNA-dependent DNA polymerase)

(D) RNase H acting alone

Q145. The circular expression vector shown carries an origin of replication (*ori*), a kanamycin-resistance marker (kan^R), a tetracycline-resistance gene (tet^R), and a unique *Not*I site lying inside tet^R. A gene of interest is ligated into the *Not*I site. Recombinant clones are most simply distinguished from non-recombinants because the recombinants become:



(A) Tetracycline-sensitive while remaining kanamycin-resistant

(B) Kanamycin-sensitive while remaining tetracycline-resistant

(C) Resistant to both antibiotics simultaneously

(D) Unable to replicate because the *ori* is interrupted

Q146. Before ligating a foreign insert, a cloning vector that has been linearised with a single restriction cut is often treated with **calf intestinal alkaline phosphatase**. The purpose of this dephosphorylation step is to:

(A) Add 5′ phosphates so the vector ligates more efficiently

(B) Methylate the vector to protect it from the restriction enzyme

(C) Degrade contaminating RNA in the preparation

(D) Remove 5′ phosphates so the vector cannot re-circularise on itself

Q147. Human growth hormone (somatropin) was among the early recombinant proteins. When such a non-glycosylated polypeptide is produced in *Escherichia coli*, it frequently accumulates intracellularly as dense insoluble aggregates that must be solubilised and refolded. These aggregates are called:

(A) Inclusion bodies

(B) Plasmids

(C) Endospores

(D) Periplasmic vesicles

Q148. In an industrial fermentation, nutrient (often the carbon source) is added incrementally during the run to avoid substrate inhibition and the Crabtree-type overflow effect, while no broth is withdrawn until the end. This mode of operation is termed:

(A) Pure batch culture

(B) Fed-batch culture

(C) Continuous (chemostat) culture

(D) Solid-state surface culture

Q149. Industrial production of vitamin B_{12} (cyanocobalamin) for pharmaceutical use is carried out by fermentation using bacteria such as *Pseudomonas denitrificans* or *Propionibacterium*. Vitamin B_{12} is required in the diet because:

(A) Plants synthesise it abundantly and animals get it from leaves

(B) It is a simple amino acid made by all cells

(C) Only certain microorganisms can synthesise this cobalt-containing corrinoid; animals cannot make it

(D) It is a structural carbohydrate of the bacterial wall

Q150. After a fermentation that yields an **extracellular** antibiotic in the broth, the **first** downstream-processing operation is normally the removal of cells and solids from the liquid. The unit operation used for this primary separation is:

(A) Lyophilisation of the whole broth

(B) Crystallisation of the pure product

(C) Chromatographic polishing on a resin column

(D) Cell separation by filtration or centrifugation

Q151. Glucose oxidase, widely used in biosensors and in the assay of blood glucose, catalyses the transfer of electrons from glucose to molecular oxygen. According to the EC classification, such an enzyme belongs to the class of:

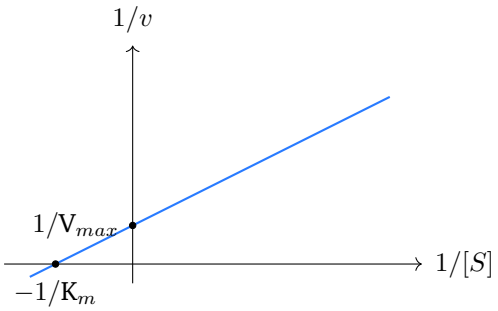
(A) Oxidoreductases (EC 1)

(B) Transferases (EC 2)

(C) Hydrolases (EC 3)

(D) Ligases (EC 6)

Q152. The double-reciprocal (Lineweaver–Burk) plot of $1/v$ against $1/[S]$ is shown. For this linear transform of the Michaelis–Menten equation, the intercept on the **vertical ($1/v$) axis** corresponds to:



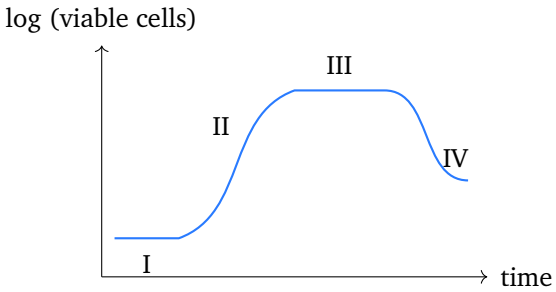
(A) $-1/\mathrm{K}_m$

(B) $1/\mathrm{V}_{max}$

(C) The slope $\mathrm{K}_m/\mathrm{V}_{max}$

(D) The turnover number k_{cat}

Q153. The bacterial batch-growth curve below plots log viable count against time. In the labelled phase III, the rate of new cell formation is balanced by the rate of cell death so that the net change in viable count is essentially zero. This phase is the:



(A) Lag phase, where cells adapt to the medium

(B) Log (exponential) phase, with maximal growth rate

(C) Stationary phase, where growth and death are balanced

(D) Death (decline) phase, where viable count falls sharply

Q154. The biological indicator most widely used to validate moist-heat (steam) sterilisation cycles consists of the highly heat-resistant endospores of:

(A) *Escherichia coli*

(B) *Staphylococcus aureus*

(C) *Aspergillus niger*

(D) *Geobacillus stearothermophilus*

Q155. A patient with seasonal allergic rhinitis shows mast-cell degranulation triggered when allergen cross-links antibodies bound to high-affinity Fc receptors on the mast-cell surface. The immunoglobulin class responsible for this type I hypersensitivity is:

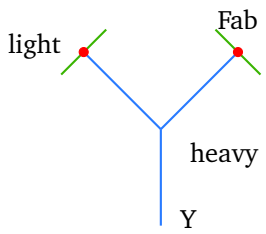
(A) IgE

(B) IgG

(C) IgM

(D) IgA

Q156. The Y-shaped IgG monomer below is cleaved by the protease **papain** above the disulphide bonds of the hinge. The region marked Y, which carries the effector site for complement and Fc-receptor binding but does **not** bind antigen, is the:



(A) Variable antigen-binding paratope

(B) Fc region (constant stem of the heavy chains)

(C) Complementarity-determining region

(D) Light-chain variable domain

Q157. In a sandwich enzyme-linked immunosorbent assay (ELISA) for a protein antigen, the bound analyte is finally detected by an enzyme-conjugated antibody that converts a colourless substrate to a coloured product. A common reporter enzyme used for this colorimetric read-out is:

(A) DNA ligase

(B) *Taq* DNA polymerase

(C) Horseradish peroxidase

(D) Reverse transcriptase

Q158. The diphtheria component of the DPT vaccine is prepared by treating the bacterial exotoxin with formaldehyde so that it loses toxicity but keeps its antigenic determinants, thereby inducing protective antibodies. This kind of immunogen is classified as a:

(A) Live attenuated vaccine

(B) Whole inactivated (killed) vaccine

(C) Conjugate polysaccharide vaccine

(D) Toxoid (inactivated toxin) vaccine

Q159. Sterile pharmaceutical products are compounded in a laminar-air-flow cabinet that delivers a unidirectional stream of air essentially free of microorganisms and particulates. The filter that renders the supplied air sterile by removing at least 99.97% of 0.3 μm particles is a:

(A) HEPA filter

(B) Sintered-glass (G3) filter

(C) Standard paper pre-filter

(D) Activated-charcoal filter

Q160. In antimicrobial susceptibility testing, the **lowest concentration** of an antibiotic that visibly **inhibits the growth** of a test organism after stan-

dard incubation (with no requirement that the organism be killed) is defined as the:

(A) Minimum bactericidal concentration (MBC)

(B) Minimum inhibitory concentration (MIC)

(C) Lethal dose 50 (LD_{50})

(D) Zone of equivalence

Detailed Solutions

Q1.

Solution

Concept — Solubility product of a 1:2 salt: For $PbI_2 \rightleftharpoons Pb^{2+} + 2\,I^-$, if molar solubility $= s$ then $[Pb^{2+}] = s$ and $[I^-] = 2s$. **Reasoning:** $K_{sp} = [Pb^{2+}][I^-]^2 = (s)(2s)^2 = 4s^3$. **Why the other options are wrong:**

- (A) s^2 is for a 1:1 salt such as AgCl.
- (C) $27s^4$ is for a 1:3 salt (AB_3).
- (D) s^3 ignores the stoichiometric factor 4 from the $(2s)^2$ term.

Final Answer: $K_{sp} = (s)(2s)^2 = 4s^3 \Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept — Single extraction: With equal volumes, $P = C_{ether}/C_{water}$ equals the mass ratio. Fraction remaining in water $= \dfrac{V_w}{V_w + P\,V_o}$. **Reasoning:** $= \dfrac{100}{100 + 4(100)} = \dfrac{100}{500} = 0.20$. Remaining $= 0.20 \times 200 = 40$ mg (160 mg extracted into ether). **Why the other options are wrong:**

- (A) 160 mg is the amount extracted, not remaining.
- (B) 100 mg assumes no extraction.
- (C) 80 mg uses $P = 1.5$ instead of 4.

Final Answer: remaining $= 200/(1+4) = 40$ mg $\Rightarrow$ D

Answer: (D) Go Back to Q2

Q3.

Solution

Concept — HLB of a blend: The HLB of a surfactant mixture is the weight-fraction-weighted average of the component HLB values. **Reasoning:** HLB $= 0.30(15) + 0.70(5) = 4.5 + 3.5 = 8.0$. **Why the other options are wrong:**

- (A) 10.0 is the simple arithmetic mean of 15 and 5, ignoring the weights.

- (B) 6.0 over-weights the low-HLB surfactant.
- (D) 12.0 over-weights the high-HLB surfactant.

Final Answer: $0.30(15) + 0.70(5) = 8.0 \Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept — Bingham plastic flow: A material that does not flow until a finite yield stress τ_0 is exceeded, after which τ rises linearly with $\dot{\gamma}$, is a Bingham plastic. **Reasoning:** The non-zero stress-axis intercept τ_0 is the yield value; the slope is the plastic mobility U. Concentrated suspensions and some pastes behave this way. **Why the other options are wrong:**

- (B) Newtonian curves pass through the origin with no yield value.
- (C) Pseudoplastic curves are nonlinear and bow toward the stress axis.
- (D) Dilatant curves bow toward the shear-rate axis.

Final Answer: finite yield intercept $\tau_0 \Rightarrow$ Bingham plastic $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept — Stokes–Einstein relation: $D = \dfrac{k_B T}{6\pi\eta r}$, where η is medium viscosity and r the solute radius. **Reasoning:** D is inversely proportional to viscosity η and to radius r, and directly proportional to absolute temperature T. **Why the other options are wrong:**

- (A) D is inversely (not directly) proportional to radius.
- (C) D rises with temperature; it is not independent of T.
- (D) The dependence on radius is first power in the denominator, not a square.

Final Answer: $D \propto 1/\eta \Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept — Capillary rise: $h = \dfrac{2\gamma}{r\rho g}$, so $h \propto 1/r$ at constant γ, ρ, g. **Reasoning:** Halving r doubles h (inverse proportionality). **Why the other options are wrong:**

- (A) Halving would require $h \propto r$, the opposite relation.
- (B) "Stay the same" ignores the $1/r$ dependence.
- (D) One-quarter would need $h \propto 1/r^2$.

Final Answer: $h \propto 1/r$; halving r doubles $h \Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept — Carr's compressibility index: Carr's % $= \dfrac{\rho_{tapped} - \rho_{bulk}}{\rho_{tapped}} \times 100$. **Reasoning:** $= \dfrac{0.60 - 0.45}{0.60} \times 100 = \dfrac{0.15}{0.60} \times 100 = 25\%$, indicating passable-to-poor flow. **Why the other options are wrong:**

- (A) 15% uses the wrong denominator difference.
- (B) 33% wrongly divides 0.15 by bulk density 0.45.
- (C) 30% over-estimates the difference.

Final Answer: $(0.15/0.60) \times 100 = 25\% \Rightarrow$ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept — Hausner ratio: HR $= \rho_{tapped}/\rho_{bulk}$. **Reasoning:** HR $= 0.60/0.45 = 1.33$, consistent with the 25% Carr's index of the previous question (poor/passable flow). **Why the other options are wrong:**

- (B) 0.75 is the inverted ratio; HR is always ≥ 1.
- (C) 1.15 corresponds to good flow, contradicting the 25% Carr's index.
- (D) 1.50 over-estimates the ratio.

Final Answer: HR $= 0.60/0.45 = 1.33 \Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept — Mode of a frequency distribution: The mode is the size class with the greatest frequency (tallest bar). **Reasoning:** The bars peak at the 35–45 μm class (height 2.9), which is taller than its neighbours. **Why the other options are wrong:**

- (A) 5–15 has the lowest frequency.
- (B) 15–25 is below the peak.
- (D) 45–55 lies on the descending tail.

Final Answer: tallest bar is the 35–45 μm class $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept — Henderson–Hasselbalch: $\text{pH} = \text{pK}_a + \log \dfrac{[\text{salt}]}{[\text{acid}]}$. **Reasoning:** pH $= 4.20 + \log(10/1) = 4.20 + 1 = 5.20$. **Why the other options are wrong:**

- (A) 4.20 assumes equal salt and acid ($\log 1 = 0$).
- (C) 3.20 subtracts instead of adds the log term.
- (D) 6.20 uses a ratio of 100:1.

Final Answer: $\text{pH} = 4.20 + 1 = 5.20 \Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept — Sodium chloride equivalent: The NaCl equivalent contributed by a drug = (grams of drug) $\times E$. **Reasoning:** A 1.0% w/v solution in 30 mL contains 0.30 g drug. NaCl equivalent $= 0.30 \times 0.20 = 0.060$ g. **Why the other options are wrong:**

- (A) 0.30 g is the mass of drug, not its NaCl equivalent.
- (B) 0.20 confuses E with the equivalent mass.
- (C) 0.090 uses 0.45 g of drug.

Final Answer: $0.30 \times 0.20 = 0.060$ g $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept — Zero-order kinetics: When the rate is independent of concentration, $C = C_0 - k_0 t$, a straight line of C versus t with slope $-k_0$. **Reasoning:** A linear decline of concentration with time (constant amount lost per unit time) is the hallmark of zero-order decomposition, typical of drug suspensions where the solution stays saturated. **Why the other options are wrong:**

- (B) First-order gives a straight line only for $\ln C$ versus t, not C versus t.
- (C) Second-order is linear for $1/C$ versus t.
- (D) Pseudo-first-order also yields a linear $\ln C$ plot, not a linear C plot.

Final Answer: linear C versus $t \Rightarrow$ zero order $\Rightarrow$ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept — First-order half-life: $t_{1/2} = 0.693/k$, so $k = 0.693/t_{1/2}$. **Reasoning:** $k = 0.693/200 = 3.465 \times 10^{-3}$ per day $\approx 3.47 \times 10^{-3}$ day^{-1}. **Why the other options are wrong:**

- (A) 0.693 forgets to divide by 200.
- (B) 0.0693 divides by 10 instead of 200.
- (D) 1.39×10^{-2} divides by 50.

Final Answer: $k = 0.693/200 = 3.47 \times 10^{-3}$ day^{-1} $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept — Order from linear plot: Second-order kinetics give $\frac{1}{C} = \frac{1}{C_0} + kt$, a straight line of $1/C$ versus t with positive slope k. **Reasoning:** The linear reciprocal-concentration plot uniquely identifies second-order behaviour. **Why the other options are wrong:**

- (A) First order is linear for $\ln C$ versus t.
- (C) Zero order is linear for C versus t.
- (D) Third order would be linear for $1/C^2$ versus t.

Final Answer: linear $1/C$ versus $t \Rightarrow$ second order $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept — Arrhenius plot: Taking logarithms of $k = A\,e^{-E_a/RT}$ gives $\ln k = \ln A - \frac{E_a}{R} \cdot \frac{1}{T}$. **Reasoning:** A plot of $\ln k$ versus $1/T$ is a straight line of slope $-E_a/R$, from which E_a is obtained. **Why the other options are wrong:**

- (A) k versus T is curved (exponential), not linear.
- (B) $\ln k$ versus T is not linear in T.
- (C) k versus $1/T$ is also non-linear.

Final Answer: $\ln k$ versus $1/T$ (slope $= -E_a/R$) $\Rightarrow$ D

Answer: (D) Go Back to Q15

Q16.

Solution

Concept — Freundlich isotherm: $x/m = k\,C^{1/n}$; taking logs gives $\log(x/m) = \log k + \frac{1}{n}\log C$. **Reasoning:** A plot of $\log(x/m)$ versus $\log C$ is linear with slope $1/n$ and intercept $\log k$. **Why the other options are wrong:**

- (B) x/m versus C is the curved isotherm itself, not linear.
- (C) $C/(x/m)$ versus C is the linear form of the Langmuir (not Freundlich) isotherm.

- (D) $1/(x/m)$ versus $1/C$ is another Langmuir double-reciprocal form.

Final Answer: $\log(x/m)$ versus $\log C \Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q16

Q17.

Solution

Concept — Eutectic point: The lowest point where two liquidus curves intersect; the eutectic mixture melts (or freezes) at a single minimum temperature as if it were a pure compound. **Reasoning:** Menthol and camphor form a classic eutectic system; at composition E the mixture liquefies at the minimum temperature, which is why such mixtures can become moist/liquid on trituration. **Why the other options are wrong:**

- (A) A peritectic involves a solid reacting with liquid to form a new solid, not a simple minimum.
- (B) The triple point refers to a single pure substance, not a binary mixture.
- (D) Inversion temperature relates to emulsion phase inversion, unrelated here.

Final Answer: lowest meeting point of liquidus curves $\Rightarrow$ eutectic point $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q17

Q18.

Solution

Concept — Noyes–Whitney equation: $dC/dt = \dfrac{DA}{h}(C_s - C)$, where h is the stagnant diffusion-layer (boundary-layer) thickness. **Reasoning:** Increasing agitation thins the hydrodynamic boundary layer, decreasing h and thereby increasing the dissolution rate. It does not change C_s, D, or A. **Why the other options are wrong:**

- (A) C_s is a thermodynamic property set by temperature and solvent, not stirring.
- (B) D depends on temperature and viscosity, not on stirring.
- (C) Surface area A is a property of the particles, unchanged by stirring.

Final Answer: agitation thins $h \Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q18

Q19.

Solution

Concept — Higuchi model: For matrix-controlled release, $Q = k_H\sqrt{t}$, so Q versus $\sqrt{t}$ is linear through the origin. **Reasoning:** The straight line through the origin on a $\sqrt{t}$ axis is the diagnostic signature of Higuchi (matrix-diffusion) release, as seen for drugs dispersed in ointment or polymer matrices. **Why the other options are wrong:**

- (A) Hixson–Crowell gives a linear cube-root-of-amount plot.
- (C) First order is linear for $\log(\%\,\text{remaining})$ versus t.
- (D) Zero order is linear for amount versus t (not $\sqrt{t}$).

Final Answer: $Q \propto \sqrt{t}$ through origin $\Rightarrow$ Higuchi $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q19

Q20.

Solution

Concept — BCS classification: Class I = high solubility + high permeability; Class II = low solubility, high permeability; Class III = high solubility, low permeability; Class IV = low/low. **Reasoning:** High solubility plus high permeability with rapid dissolution defines BCS Class I, for which dissolution is rarely rate-limiting. **Why the other options are wrong:**

- (B) Class II has low solubility (dissolution-limited).
- (C) Class III has low permeability.
- (D) Class IV is poor in both solubility and permeability.

Final Answer: high solubility + high permeability $\Rightarrow$ Class I $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q20

Q21.

Solution

Concept — Similarity factor f_2: A logarithmic transformation of the mean squared difference between test and reference percentages released; f_2 ranges from 0 to 100. **Reasoning:** Profiles are considered similar when f_2 lies between 50 and 100 (with $f_2 = 100$ for identical profiles). Values below 50 indicate dissimilarity. **Why the other options are wrong:**

- (A) 0–15 and (B) 15–50 both fall in the dissimilar region.
- (D) f_2 cannot exceed 100, so 100–150 is impossible.

Final Answer: similar when $50 \leq f_2 \leq 100 \Rightarrow$ C

Answer: (C) Go Back to Q21

Q22.

Solution

Concept — Tablet excipient functions: Talc is a hydrophobic, plate-like powder that reduces inter-particle friction and improves powder flow while easing tablet ejection. **Reasoning:** It therefore functions chiefly as a glidant and lubricant in the granulation. **Why the other options are wrong:**

- (A) Binders (e.g. starch paste, PVP) impart cohesion; talc does not.
- (B) Disintegrants promote tablet break-up; talc is not a disintegrant.
- (C) Diluents (e.g. lactose) add bulk; talc is used in much smaller amounts.

Final Answer: talc acts as a glidant / lubricant $\Rightarrow$ D

Answer: (D) Go Back to Q22

Q23.

Solution

Concept — Film coating components: A film coat needs a film-forming polymer, a plasticiser, a colourant/opacifier and a solvent. **Reasoning:** HPMC (hypromellose) is the principal water-soluble film-forming polymer that lays down the continuous coat. **Why the other options are wrong:**

- (A) Plasticisers are agents such as PEG or triacetin, not HPMC.
- (C) Opacifiers are pigments such as titanium dioxide.

- (D) Anti-tack agents are typically talc; HPMC is the polymer itself.

Final Answer: HPMC is the film-forming polymer $\Rightarrow$ B

Answer: (B) Go Back to Q23

Q24.

Solution

Concept — Weight-variation limit: The acceptable individual weight is the average $\pm$ the stated percentage. **Reasoning:** 5% of 250 mg $= 12.5$ mg, giving a range of $250 \pm 12.5 = 237.5$ to 262.5 mg. **Why the other options are wrong:**

- (A) 245–255 corresponds to $\pm 2\%$.
- (B) 240–260 corresponds to $\pm 4\%$.
- (D) 225–275 corresponds to $\pm 10\%$.

Final Answer: $250 \pm 12.5 = 237.5$–262.5 mg $\Rightarrow$ C

Answer: (C) Go Back to Q24

Q25.

Solution

Concept — Hard-gelatin capsule sizing: Human capsule sizes run 000, 00, 0, 1, 2, 3, 4, 5 from largest to smallest fill volume. **Reasoning:** Size 000 has the largest capacity (about 1.37 mL), while size 5 is the smallest (about 0.13 mL). **Why the other options are wrong:**

- (B) Size 0 is intermediate, smaller than 000.
- (C) Size 3 is small.
- (D) Size 5 is the smallest of all.

Final Answer: largest capacity is size 000 $\Rightarrow$ A

Answer: (A) Go Back to Q25

Q26.

Solution

Concept — Displacement value (DV): Cocoa butter displaced by drug = (mass of drug)/DV. Cocoa butter needed = (nominal base mass) − (drug/DV) per unit, summed over all units. **Reasoning:** Total drug = $10 \times 0.2 = 2.0$ g; cocoa butter displaced = $2.0/2 = 1.0$ g; nominal base for 10 moulds = $10 \times 1 = 10$ g; cocoa butter required = $10 - 1.0 = 9.0$ g. **Why the other options are wrong:**

- (A) 8.0 g over-subtracts (assumes DV = 1).
- (C) 10.0 g ignores the drug displacement entirely.
- (D) 11.0 g adds instead of subtracting the displaced base.

Final Answer: $10 - (2.0/2) = 9.0$ g $\Rightarrow$ B

Answer: (B) Go Back to Q26

Q27.

Solution

Concept — Tonicity adjustment in parenterals: Isotonic fluids must approximate the osmotic pressure of blood plasma; dextrose is a common, physiologically inert tonicity agent. **Reasoning:** A 5% w/v dextrose solution is isotonic with plasma and additionally provides calories, which is why dextrose is widely used to adjust tonicity of intravenous fluids. **Why the other options are wrong:**

- (A) Dextrose has no preservative action.
- (B) It does not act as a pH buffer.
- (D) Chelation is the role of agents such as EDTA, not dextrose.

Final Answer: dextrose adjusts tonicity (and adds calories) $\Rightarrow$ C

Answer: (C) Go Back to Q27

Q28.

Solution

Concept — Emulsion type by conductivity: Electrical conductivity is governed by the continuous phase. Water (with electrolytes) conducts; oil does not. **Reasoning:** Good conductivity means the external (continuous) phase is aqueous, i.e. an oil-in-water (o/w) emulsion. **Why the other options are wrong:**

- (A) A w/o emulsion has an oil continuous phase and conducts poorly.
- (B) Conductivity here is high, so it is not a non-conducting system.
- (C) A w/o/w multiple emulsion has an oil intermediate phase and conducts poorly.

Final Answer: high conductivity $\Rightarrow$ aqueous external phase $\Rightarrow$ o/w $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q28

Q29.

Solution

Concept — Sedimentation volume: $F = V_u/V_0$, the ratio of the final sediment volume to the original suspension volume. **Reasoning:** $F = 18/24 = 0.75$. A high F (close to 1) indicates a desirable, easily redispersible (flocculated) suspension. **Why the other options are wrong:**

- (B) 1.33 is the inverted ratio; $F \leq 1$ for normal flocculation.
- (C) 0.25 is the fraction of supernatant, not sediment.
- (D) 0.18 misreads the volumes as a fraction of 100.

Final Answer: $F = 18/24 = 0.75 \Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q29

Q30.

Solution

Concept — Ointment base classes: Oleaginous (hydrocarbon) bases, absorption bases, water-removable (emulsion) bases and water-soluble bases. **Reasoning:** White petrolatum is a purified semisolid hydrocarbon mixture, the prototype oleaginous (hydrocarbon) base; it is occlusive and emollient and contains no water. **Why the other options are wrong:**

- (A) Absorption bases (e.g. wool fat) take up water; petrolatum itself does not.
- (C) Water-removable bases are o/w creams (e.g. hydrophilic ointment).
- (D) Water-soluble bases are PEG-type bases.

Final Answer: white petrolatum is an oleaginous (hydrocarbon) base $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q30

Q31.

Solution

Concept — Fluid-energy (jet) mill: Particles are entrained in high-velocity gas/air streams and reduced by violent inter-particle collisions and attrition. **Reasoning:** The dominant mechanism is impact and attrition between particles themselves, producing micron-sized powders (micronisation) with no moving grinding surface. **Why the other options are wrong:**

- (A) Cutting with knives describes a cutter mill.
- (B) Compression between rollers describes a roller mill.
- (D) Crushing under a pestle describes an end-runner/edge-runner mill.

Final Answer: inter-particle attrition and impact in fluid streams $\Rightarrow$ C

Answer: (C) Go Back to Q31

Q32.

Solution

Concept — Reynolds number regimes: For pipe flow, $Re < 2100$ is laminar, $2100 < Re < 4000$ is transitional, and $Re > 4000$ is fully turbulent. **Reasoning:** The chaotic-eddy pattern (ii) corresponds to fully turbulent flow, generally taken above $Re \approx 4000$. **Why the other options are wrong:**

- (A) 100 and (B) 500 are well within the laminar region.
- (C) 2100 is the upper limit of laminar flow, the start of the transition (not fully turbulent).

Final Answer: fully turbulent for $Re > 4000 \Rightarrow$ D

Answer: (D) Go Back to Q32

Q33.

Solution

Concept — Stages of freeze-drying: Freezing, then primary drying (sublimation of free ice), then secondary drying (desorption of bound water). **Reasoning:** Secondary drying raises the shelf temperature to remove residual adsorbed (bound) moisture by desorption, lowering the final water content to a few percent for long-term stability. **Why the other options are wrong:**

- (B) Sublimation of free ice is the *primary* drying stage.
- (C) Eutectic crystals form during freezing, not removed in secondary drying.
- (D) Nitrogen blanketing is part of final stoppering, not a drying step.

Final Answer: secondary drying removes bound moisture by desorption $\Rightarrow$ A

Answer: (A) Go Back to Q33

Q34.

Solution

Concept — PLGA microspheres: PLGA is a biodegradable polyester that hydrolyses in aqueous body fluids to lactic and glycolic acids. **Reasoning:** As the polymer matrix slowly hydrolyses and erodes, entrapped drug is released gradually over days to weeks, giving controlled (sustained) release from the microspheres. **Why the other options are wrong:**

- (A) PLGA does not dissolve instantly; that would give a burst, not controlled release.
- (C) Magnetic targeting requires magnetite, not inherent to PLGA.
- (D) Photochemical bleaching is irrelevant to PLGA drug release.

Final Answer: slow hydrolytic degradation/erosion of PLGA matrix $\Rightarrow$ B

Answer: (B) Go Back to Q34

Q35.

Solution

Concept — Renal clearance: Renal clearance equals the urinary excretion rate divided by the plasma concentration, $CL_R = (\text{rate of urinary excretion})/C_p$. **Reasoning:** Here $CL_R = 8\,\text{mg/min}/4\,\text{mg/L} = 2$ L/min. Clearance is the volume of plasma cleared of drug per unit time, so dividing an amount-per-time by a concentration gives a volume-per-time. **Why the other options are wrong:**

- (A) 0.5 L/min inverts the ratio.
- (C) 32 L/min multiplies instead of dividing.
- (D) 4 L/min uses the plasma value directly without the excretion rate.

Final Answer: $CL_R = 8/4 = 2$ L/min $\Rightarrow$ B

Answer: (B) Go Back to Q35

Q36.

Solution

Concept — First-order elimination: A constant fraction is lost per unit time; each half-life halves the concentration, and $k_e = 0.693/t_{1/2}$. **Reasoning:** 100 $\rightarrow$ 50 $\rightarrow$ 25 $\rightarrow$ 12.5 mg/L is three halvings over 6 h, so $t_{1/2} = 6/3 = 2$ h and $k_e = 0.693/2 = 0.347\ \text{h}^{-1}$. **Why the other options are wrong:**

- (A) 6 h is the whole interval, not one half-life.
- (B) 1 h would need 6 halvings (factor 64).
- (C) 3 h gives only two halvings (factor 4), reaching 25 mg/L.

Final Answer: Three halvings over 6 h $\Rightarrow t_{1/2} = 2$ h, $k_e = 0.347\ \text{h}^{-1} \Rightarrow$ D

Answer: (D) Go Back to Q36

Q37.

Solution

Concept — Maintenance dosing rate: At steady state the rate in equals the rate of elimination, so the infusion rate $R_0 = CL \times C_{ss}$. **Reasoning:** $R_0 = 3\,\text{L/h} \times 5\,\text{mg/L} = 15$ mg/h. Clearance and the desired steady-state concentration alone fix the maintenance rate; the volume of distribution affects only how fast the plateau is reached, not its height. **Why the other options are wrong:**

- (B), (C) Divide rather than multiply the two quantities.
- (D) 8 mg/h uses an incorrect clearance value.

Final Answer: $R_0 = CL \times C_{ss} = 3 \times 5 = 15$ mg/h $\Rightarrow$ A

Answer: (A) Go Back to Q37

Q38.

Solution

Concept — Acetylator polymorphism: N-acetyltransferase-2 activity is genetically bimodal; slow acetylators metabolise acetylated drugs more slowly. **Reasoning:** With reduced acetylation, the parent drug is cleared more slowly, so plasma levels are higher and concentration-dependent adverse effects are more likely (isoniazid peripheral neuropathy, hydralazine/procainamide drug-induced lupus). **Why the other options are wrong:**

- (A) Slow acetylators clear the drug more slowly, not faster.
- (B) There is a clear pharmacokinetic difference between phenotypes.
- (D) The polymorphism affects metabolism, not gut absorption.

Final Answer: Slow acetylators have higher levels and more toxicity $\Rightarrow$ C

Answer: (C) Go Back to Q38

Q39.

Solution

Concept — Potency on a graded curve: For equally efficacious agonists, the curve lying furthest to the left (lowest ED_{50}) is the most potent. **Reasoning:** All three reach the same E_{max}, so they differ only in the dose needed for half-maximal effect. X is leftmost (lowest ED_{50}), then Y, then Z, giving potency order X $>$ Y $>$ Z. **Why the other options are wrong:**

- (A) Reverses the order; Z is the least potent.
- (B) Misorders the middle and end curves.
- (C) Equal potency would require overlapping curves.

Final Answer: Leftmost curve most potent $\Rightarrow$ X $>$ Y $>$ Z $\Rightarrow$ D

Answer: (D) Go Back to Q39

Q40.

Solution

Concept — Therapeutic index: TI is the ratio of the median lethal dose to the median effective dose, TI $= LD_{50}/ED_{50}$. **Reasoning:** With ED_{50} = 10 mg and LD_{50} = 200 mg, TI $= 200/10 = 20$. A larger TI indicates a wider margin of safety between the effective and toxic dose ranges. **Why the other options are wrong:**

- (B) 0.05 inverts the ratio.
- (C) 190 subtracts instead of dividing.
- (D) 2000 multiplies the two doses.

Final Answer: TI $= 200/10 = 20 \Rightarrow$ A

Answer: (A) Go Back to Q40

Q41.

Solution

Concept — Antagonist curve signatures: A competitive antagonist gives a parallel rightward shift with unchanged E_{max} (surmountable); a non-competitive antagonist depresses E_{max} (insurmountable). **Reasoning:** Curve M has a lowered maximum that extra agonist cannot restore, so it is non-competitive (insurmountable). Curve N is shifted right but reaches the same maximum, so it is competitive (surmountable). **Why the other options are wrong:**

- (A) Swaps the two interpretations.
- (C) Only N behaves competitively.
- (D) These are antagonist curves, not agonist curves.

Final Answer: M = non-competitive, N = competitive $\Rightarrow$ B

Answer: (B) Go Back to Q41

Q42.

Solution

Concept — Termination of adrenergic transmission: Released noradrenaline is removed chiefly by neuronal reuptake (uptake-1), not by enzymatic hydrolysis in the cleft. **Reasoning:** The noradrenaline transporter (NET, uptake-1) carries transmitter back into the terminal, where intraneuronal monoamine oxidase metabolises it; some is also taken up extraneuronally (uptake-2) and methylated by COMT. This is why reuptake blockers and indirect sympathomimetics modify adrenergic signalling. **Why the other options are wrong:**

- (A) AChE hydrolyses acetylcholine at cholinergic, not adrenergic, synapses.
- (B) Diffusion contributes little; active reuptake dominates.
- (D) Noradrenaline is not converted to acetylcholine.

Final Answer: Reuptake by uptake-1/NET then MAO $\Rightarrow$ C

Answer: (C) Go Back to Q42

Q43.

Solution

Concept — Intrinsic sympathomimetic activity: Some β-blockers are partial agonists that produce weak stimulation while still blocking against strong agonist drive. **Reasoning:** Pindolol weakly stimulates β receptors at rest (low sympathetic tone) yet blocks them when sympathetic activity is high, so it causes less resting bradycardia and may less affect lipids than pure antagonists. **Why the other options are wrong:**

- (A) It is not an irreversible alkylating agent.
- (B) A silent antagonist has no agonist effect; pindolol does, by definition of ISA.
- (C) Combined α/β blockade describes labetalol/carvedilol, not ISA.

Final Answer: Partial β agonist (ISA), less resting bradycardia $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q43

Q44.

Solution

Concept — Direct muscarinic agonist: Bethanechol is a choline ester selective for muscarinic receptors and resistant to acetylcholinesterase. **Reasoning:** Because it is not hydrolysed by AChE it acts for a useful duration, stimulating M_3 receptors on detrusor smooth muscle to contract the bladder and on gut smooth muscle to increase motility, relieving non-obstructive urinary retention and post-operative ileus. **Why the other options are wrong:**

- (B) It is an agonist, not an antagonist, at the bladder.
- (C) It acts on muscarinic, not nicotinic, receptors.
- (D) A β_3 agonist relaxes the detrusor (used for overactive bladder), the opposite action.

Final Answer: AChE-resistant muscarinic agonist contracting detrusor $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q44

Q45.

Solution

Concept — α_1-subtype selectivity: Tamsulosin preferentially blocks the α_{1A} subtype concentrated in prostatic and bladder-neck smooth muscle. **Reasoning:** Relaxing α_{1A}-rich prostatic smooth muscle improves urinary flow in BPH, while sparing vascular α_{1B} receptors limits the postural hypotension seen with non-selective α-blockers. **Why the other options are wrong:**

- (A) It is not a β_2 agent.
- (C) M_3 antimuscarinics treat overactive bladder, a different target.
- (D) Central α_2 agonism describes clonidine/methyldopa.

Final Answer: α_{1A}-selective blockade of prostatic smooth muscle $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q45

Q46.

Solution

Concept — Indirect sympathomimetics: These amines act mainly by releasing stored noradrenaline rather than by directly occupying adrenoceptors. **Reasoning:** They are taken up into nerve terminals and displace noradrenaline from vesicles into the synapse. As stores deplete with repeated dosing the response wanes (tachyphylaxis). Ephedrine has both direct and indirect components. **Why the other options are wrong:**

- (A) Purely direct action describes agonists like phenylephrine, not indirect amines.
- (B) MAO inhibition is not the principal mechanism (though it can potentiate them).
- (D) Pure transporter blockade without release describes cocaine/reuptake inhibitors.

Final Answer: Release of stored noradrenaline, prone to tachyphylaxis $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q46

Q47.

Solution

Concept — Ionotropic chloride channel: The GABA-A receptor is a ligand-gated (ionotropic) chloride channel, the major inhibitory receptor in the CNS. **Reasoning:** GABA opens the integral Cl^- channel, hyperpolarising the neuron. Benzodiazepines increase the frequency, and barbiturates the duration, of channel opening, enhancing inhibition. This matches the "ionotropic $\rightarrow$ Cl^- channel" branch. **Why the other options are wrong:**

- (A) The β-adrenergic receptor is a metabotropic GPCR.
- (B) M_2 is a G_i-coupled GPCR.
- (C) D_1 is a G_s-coupled GPCR.

Final Answer: GABA-A ligand-gated Cl^- channel $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q47

Q48.

Solution

Concept — SSRI mechanism: Sertraline selectively inhibits the serotonin reuptake transporter (SERT). **Reasoning:** Blocking SERT raises synaptic 5-HT with little effect on noradrenaline or dopamine transporters. Combining it with an MAOI causes excessive serotonin and the serotonin syndrome (agitation, hyperthermia, clonus, autonomic instability), so the two classes must not overlap. **Why the other options are wrong:**

- (B) D_2 blockade is an antipsychotic action.
- (C) Irreversible MAO-A inhibition describes the MAOI it should not be combined with.
- (D) It does not act on GABA-A channels.

Final Answer: Selective serotonin reuptake inhibition $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q48

Q49.

Solution

Concept — Carbamazepine: It blocks voltage-gated sodium channels and is a strong hepatic enzyme inducer. **Reasoning:** Use-dependent Na^+ channel blockade stabilises neuronal membranes for partial/tonic-clonic seizures and trigeminal neuralgia. It induces CYP enzymes, including those metabolising itself (autoinduction), so its clearance rises over the first weeks and many drug interactions follow. **Why the other options are wrong:**

- (A) Its main action is Na^+ channel block, and it induces rather than inhibits enzymes.
- (B) T-type Ca^{2+} block is the ethosuximide mechanism.
- (D) It is not an NMDA antagonist and is an inducer, not an inhibitor.

Final Answer: Na^+ channel blockade plus enzyme (auto)induction $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q49

Q50.

Solution

Concept — Central antitussive: Dextromethorphan, the d-isomer of a codeine analogue, suppresses cough centrally with negligible analgesia or dependence at usual doses. **Reasoning:** It raises the cough threshold by acting on the medullary cough centre. Lacking meaningful μ-opioid analgesic activity, it is a non-narcotic antitussive (though high doses have NMDA-antagonist/dissociative effects). **Why the other options are wrong:**

- (A) It is not a strong μ agonist like morphine.
- (C) It does not act by local anaesthesia of the mucosa.
- (D) H_1 blockade is not its antitussive mechanism.

Final Answer: Central cough-centre suppression, non-narcotic $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q50

Q51.

Solution

Concept — Gabapentinoids: Pregabalin and gabapentin bind the $\alpha_2\delta$ subunit of voltage-gated calcium channels. **Reasoning:** Binding $\alpha_2\delta$ reduces calcium influx at presynaptic terminals and lowers release of excitatory transmitters (glutamate, noradrenaline, substance P), which underlies their action in neuropathic pain and as add-on antiepileptics. Despite the name, they do not act on GABA receptors. **Why the other options are wrong:**

- (A) They do not enhance GABA reuptake (that would be the opposite of useful).
- (B) Na^+ channel block is the phenytoin/carbamazepine mechanism.
- (C) GABA-transaminase inhibition is the vigabatrin mechanism.

Final Answer: $\alpha_2\delta$ calcium-channel binding $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q51

Q52.

Solution

Concept — Typical vs atypical antipsychotics: High-potency typicals strongly block D_2 with little 5-HT_{2A} antagonism, predisposing to extrapyramidal symptoms. **Reasoning:** Heavy nigrostriatal D_2 blockade by fluphenazine disrupts the dopamine-acetylcholine balance, causing parkinsonism, dystonia and akathisia. Atypicals add 5-HT_{2A} blockade, which partly restores striatal dopamine release and reduces EPS. **Why the other options are wrong:**

- (B) H_1 blockade causes sedation, not EPS.
- (C) It is not an SSRI.
- (D) It has high D_2 affinity, which is the cause of EPS.

Final Answer: Strong nigrostriatal D_2 blockade with weak 5-HT_{2A} block $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q52

Q53.

Solution

Concept — Propofol mechanism: Propofol potentiates GABA-A receptor chloride currents. **Reasoning:** By enhancing GABA-mediated inhibition it produces rapid loss of consciousness; its high lipid solubility and rapid redistribution give quick onset and recovery, making it a popular induction and maintenance agent. It can cause dose-dependent hypotension and respiratory depression. **Why the other options are wrong:**

- (A) NMDA antagonism is the ketamine mechanism.
- (B) It is not an opioid agonist.
- (D) Na^+ channel block describes local anaesthetics, not propofol.

Final Answer: Potentiation of GABA-A chloride currents $\Rightarrow$ C

Answer: (C) Go Back to Q53

Q54.

Solution

Concept — Isosorbide mononitrate: It is the active metabolite of the dinitrate, with essentially complete oral bioavailability and a long action suited to prophylaxis. **Reasoning:** Unlike glyceryl trinitrate (which has extensive first-pass metabolism and a short action), mononitrate escapes first-pass and acts for hours, releasing nitric oxide to venodilate and reduce preload. A nitrate-free interval each day prevents tolerance. **Why the other options are wrong:**

- (A) Nitrates act via NO/cGMP, not calcium-channel block.
- (C) They reduce, not increase, myocardial oxygen demand.
- (D) Protamine reverses heparin, not nitrates.

Final Answer: High oral bioavailability, long action, tolerance avoided by nitrate-free interval $\Rightarrow$ B

Answer: (B) Go Back to Q54

Q55.

Solution

Concept — Class Ib antiarrhythmic: Lidocaine blocks sodium channels with fast onset/offset and shortens the action-potential duration. **Reasoning:** Its rapid binding kinetics make it selective for rapidly firing or depolarised (ischaemic) ventricular tissue, suppressing ventricular arrhythmias after myocardial infarction while having little effect on normal atrial tissue. It shortens, rather than prolongs, repolarisation. **Why the other options are wrong:**

- (A) K^+ block prolonging the action potential is Class III (amiodarone).
- (B) β-blockade is Class II.
- (C) AV-node calcium block is Class IV (verapamil).

Final Answer: Fast Na^+ block, shortens APD, targets ischaemic ventricle $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q55

Q56.

Solution

Concept — Amiloride mechanism: Amiloride directly blocks the epithelial sodium channel (ENaC) in the late distal tubule and collecting duct. **Reasoning:** Blocking ENaC reduces sodium reabsorption and the lumen-negative potential that drives potassium secretion, so it produces a mild diuresis while sparing potassium. It works independently of aldosterone, unlike spironolactone. **Why the other options are wrong:**

- (B) $Na^+/K^+/2Cl^-$ inhibition is the loop-diuretic action.
- (C) Mineralocorticoid-receptor antagonism describes spironolactone.
- (D) Carbonic-anhydrase inhibition describes acetazolamide.

Final Answer: Direct ENaC blockade, potassium-sparing $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q56

Q57.

Solution

Concept — Low-molecular-weight heparin: Enoxaparin acts through antithrombin but inhibits factor Xa more than thrombin. **Reasoning:** Its shorter chains accelerate antithrombin-mediated inhibition of factor Xa while being too short to bridge antithrombin to thrombin efficiently, giving a higher anti-Xa:anti-IIa ratio. This yields a more predictable dose response, subcutaneous once/twice-daily dosing and no routine aPTT monitoring. **Why the other options are wrong:**

- (A) It is injected, not oral, and not monitored by INR.
- (B) Direct thrombin inhibition describes dabigatran/argatroban.
- (D) Vitamin K reverses warfarin; protamine only partially reverses LMWH.

Final Answer: Antithrombin-mediated, predominantly anti-Xa, predictable, little monitoring $\Rightarrow$ C

Answer: (C) Go Back to Q57

Q58.

Solution

Concept — Second-generation H_1 antihistamines: Cetirizine is poorly CNS-penetrant, so it causes little sedation. **Reasoning:** Being more polar and a P-glycoprotein substrate, cetirizine crosses the blood-brain barrier poorly, so central H_1 blockade and drowsiness are minimal compared with the lipophilic first-generation agent promethazine, which readily enters the brain. **Why the other options are wrong:**

- (A) It is an H_1, not H_2, antagonist.
- (C) Strong antimuscarinic action characterises older antihistamines, not cetirizine.
- (D) It blocks the H_1 receptor rather than merely stabilising mast cells.

Final Answer: Second-generation H_1 antagonist with poor CNS entry $\Rightarrow$ B

Answer: (B) Go Back to Q58

Q59.

Solution

Concept — Selective COX-2 inhibition: Celecoxib spares COX-1 (gastric mucosa, platelets) while inhibiting inducible COX-2 at inflammation sites. **Reasoning:**

Sparing platelet COX-1 means it does not inhibit thromboxane and gives fewer peptic ulcers, but it leaves vascular prostacyclin (COX-2-derived) suppressed without the antiplatelet balance, contributing to an increased cardiovascular thrombotic risk. **Why the other options are wrong:**

- (A) It does not irreversibly acetylate platelet COX-1; aspirin does.
- (B) It inhibits cyclooxygenase, not lipoxygenase.
- (C) It is a genuine anti-inflammatory.

Final Answer: Selective COX-2 inhibition, fewer GI effects, higher CV risk $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q59

Q60.

Solution

Concept — Leukotriene synthesis inhibition: Zileuton inhibits 5-lipoxygenase, the enzyme that begins leukotriene synthesis from arachidonic acid. **Reasoning:** By blocking 5-LOX it reduces formation of both the cysteinyl-leukotrienes ($LTC_4/D_4/E_4$) and LTB_4, lessening bronchoconstriction and inflammation. This contrasts with montelukast, which leaves synthesis intact but blocks the $CysLT_1$ receptor. **Why the other options are wrong:**

- (B) Receptor blockade is the montelukast mechanism.
- (C) COX-2 inhibition affects prostaglandins, not leukotrienes.
- (D) Mast-cell stabilisation describes cromoglicate.

Final Answer: 5-lipoxygenase inhibition blocking leukotriene synthesis $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q60

Q61.

Solution

Concept — Misoprostol: It is a PGE_1 analogue that replaces the gastroprotective prostaglandins suppressed by NSAIDs. **Reasoning:** Acting on parietal-cell EP receptors it inhibits acid secretion and, via mucosal cells, promotes mucus and bicarbonate secretion and mucosal blood flow, protecting against NSAID-induced ulcers. (It is uterotonic, so it is contraindicated in pregnancy.) **Why the other options are wrong:**

- (A) H_2 blockade describes ranitidine/famotidine.
- (B) It is not a chemical antacid.
- (D) Irreversible proton-pump inhibition describes omeprazole.

Final Answer: Prostaglandin replacement, cytoprotective and acid-suppressing $\Rightarrow$ C

Answer: (C) Go Back to Q61

Q62.

Solution

Concept — Cephalosporin (β-lactam): Ceftriaxone binds penicillin-binding proteins and blocks peptidoglycan cross-linking. **Reasoning:** As a β-lactam it inhibits transpeptidase enzymes, halting cell-wall synthesis and killing dividing bacteria. Its broad Gram-negative cover and good cerebrospinal-fluid penetration make it a standard choice for bacterial meningitis. **Why the other options are wrong:**

- (A) 50S inhibition describes macrolides/chloramphenicol.
- (C) DNA gyrase inhibition describes fluoroquinolones.
- (D) Folate-synthesis inhibition describes sulfonamides.

Final Answer: PBP binding/cell-wall inhibition with good CSF entry $\Rightarrow$ B

Answer: (B) Go Back to Q62

Q63.

Solution

Concept — Fluoroquinolone target: Ciprofloxacin inhibits bacterial DNA gyrase and topoisomerase IV. **Reasoning:** By inhibiting DNA gyrase (topoisomerase II) it prevents the negative supercoiling needed for DNA replication and transcription; topoisomerase IV inhibition impairs chromosome separation. The result is bactericidal interruption of DNA synthesis. **Why the other options are wrong:**

- (A) 30S binding/misreading describes aminoglycosides.
- (B) Transpeptidation inhibition describes β-lactams.
- (C) DHFR inhibition describes trimethoprim.

Final Answer: Inhibition of DNA gyrase/topoisomerase IV $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q63

Q64.

Solution

Concept — Metronidazole selectivity: It is a prodrug activated by reduction in anaerobic and microaerophilic organisms. **Reasoning:** Inside anaerobes/protozoa the low-redox environment reduces its nitro group to reactive intermediates that fragment microbial DNA. Aerobic host cells lack this reductive activation, giving selective toxicity against anaerobic bacteria and protozoa such as *Giardia* and *Entamoeba*. **Why the other options are wrong:**

- (B) It does not act on the host ribosome.
- (C) It does not inhibit cell-wall synthesis.
- (D) Iron chelation is not its mechanism.

Final Answer: Reductive activation to DNA-damaging intermediates in anaerobes $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q64

Q65.

Solution

Concept — Levothyroxine: It is synthetic T_4 used to replace deficient thyroid hormone. **Reasoning:** After absorption, T_4 is deiodinated peripherally to the more active T_3, which binds nuclear thyroid-hormone receptors to regulate metabolism and restore the euthyroid state. Its long half-life (about a week) permits stable once-daily dosing. **Why the other options are wrong:**

- (A) Thyroid-peroxidase inhibition is the antithyroid (carbimazole) action.
- (C) Blocking T_4-to-T_3 conversion is the opposite of replacement (e.g. propylthiouracil/propranolol at high dose).
- (D) It is not a radioactive agent; radioiodine ablation is different.

Final Answer: Synthetic T_4 acting via nuclear receptors, long half-life $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q65

Q66.

Solution

Concept — Iron chelation: Deferoxamine is the specific parenteral chelator for acute iron poisoning. **Reasoning:** Deferoxamine binds ferric iron to form ferrioxamine, which is excreted renally (classically giving urine a reddish "vin rose" colour), lowering the free iron that causes mitochondrial and hepatic toxicity. So the iron $\rightarrow$ deferoxamine pairing is correct. **Why the other options are wrong:**

- (A) Dimercaprol is used for arsenic/mercury/lead, not iron.
- (B) D-penicillamine is used for copper (Wilson's) and some heavy metals.
- (D) Atropine treats cholinergic/organophosphate toxicity, not iron.

Final Answer: Iron poisoning $\rightarrow$ deferoxamine $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q66

Q67.

Solution

Concept — Enzyme inhibition interaction: A CYP3A4 inhibitor slows the metabolism of CYP3A4 substrates, raising their plasma levels. **Reasoning:** Ketoconazole and grapefruit-juice furanocoumarins inhibit CYP3A4 (the gut/liver

enzyme handling many drugs), so substrate clearance falls, plasma concentrations rise and dose-related toxicity becomes more likely. Unlike induction, inhibition is rapid in onset. **Why the other options are wrong:**

- (A) Inhibition slows metabolism, raising not lowering levels.
- (B) Induction (slower, increasing metabolism) is the opposite effect, e.g. rifampicin.
- (C) A clinically important interaction does occur.

Final Answer: Reduced metabolism, higher substrate levels and toxicity $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q67

Q68.

Solution

Concept — Placental drug transfer: The placenta behaves like a lipid membrane; passive diffusion favours small, lipophilic, un-ionised, unbound molecules. **Reasoning:** Lipid-soluble drugs of low molecular weight that are largely un-ionised at physiological pH and not tightly protein-bound cross most readily to the fetus. Large or highly ionised, water-soluble or highly bound drugs cross poorly (heparin, a large molecule, does not reach the fetus). **Why the other options are wrong:**

- (B) Ionised, water-soluble drugs diffuse poorly across lipid membranes.
- (C) High molecular weight (e.g. heparin) hinders passage.
- (D) Extensive protein binding keeps drug in maternal plasma.

Final Answer: Lipid-soluble, low MW, un-ionised, poorly bound crosses best $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q68

Q69.

Solution

Concept — Perkin reaction: An aromatic aldehyde condenses with an aliphatic acid anhydride in the presence of the sodium (or potassium) salt of the acid to give an α,β-unsaturated acid. **Reasoning:** The carboxylate base generates the anhydride enolate, which adds to benzaldehyde; subsequent dehydration and hydrolysis give cinnamic acid (Ar–CH=CH–COOH). This is the Perkin reaction. **Why**

the other options are wrong:

- (A) Claisen–Schmidt is a crossed aldol between an aromatic aldehyde and a ketone, not an anhydride.
- (B) Cannizzaro is a base-induced disproportionation, giving no C=C.
- (D) Wittig needs a phosphorus ylide, not an anhydride.

Final Answer: It is the Perkin reaction $\Rightarrow$ C

Answer: (C) Go Back to Q69

Q70.

Solution

Concept — Clemmensen reduction: Reduction of a carbonyl (C=O) all the way to a methylene (CH_2) under **acidic** conditions. **Reasoning:** Amalgamated zinc and concentrated HCl convert aldehydes/ketones to the corresponding alkane. It is the acidic complement of the basic Wolff–Kishner reduction and is used for acid-stable substrates. **Why the other options are wrong:**

- (B) Wolff–Kishner uses hydrazine/base, not Zn–Hg/acid.
- (C) Rosenmund reduces an acid chloride to an aldehyde, not C=O to CH_2.
- (D) Bouveault–Blanc reduces esters to alcohols (Na/ethanol).

Final Answer: It is the Clemmensen reduction $\Rightarrow$ A

Answer: (A) Go Back to Q70

Q71.

Solution

Concept — Curtius rearrangement: Thermolysis of an acyl azide gives an isocyanate by migration of R from carbon to electron-deficient nitrogen, with loss of N_2. **Reasoning:** $RCON_3 \xrightarrow{\Delta} R\text{–}N{=}C{=}O$ $(+N_2)$; aqueous hydrolysis of the isocyanate then yields RNH_2 (with loss of CO_2), one carbon shorter. This is the Curtius rearrangement. **Why the other options are wrong:**

- (A) Hofmann bromamide starts from an amide with Br_2/NaOH, not an azide.
- (B) Schmidt uses HN_3 with a carboxylic acid/ketone under acid.
- (C) Lossen rearrangement starts from a hydroxamic acid derivative.

Final Answer: It is the Curtius rearrangement $\Rightarrow$ D

Answer: (D) Go Back to Q71

Q72.

Solution

Concept — Stork enamine reaction: An enamine, formed from a ketone and a secondary amine, is a neutral nucleophile at its α-carbon for clean monoalkylation/acylation. **Reasoning:** Ketone + pyrrolidine $\rightarrow$ enamine; the enamine reacts with an alkyl/acyl halide; hydrolysis regenerates the α-substituted ketone. It avoids the over-alkylation common with enolates. **Why the other options are wrong:**

- (A) Mannich uses an amine + formaldehyde + an enolisable carbonyl to give a β-amino ketone.
- (C) Michael is conjugate addition to an enone, a different bond-forming event.
- (D) Reformatsky uses an α-halo ester with zinc.

Final Answer: It is the Stork enamine reaction $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q72

Q73.

Solution

Concept — Decalin stereochemistry: Decalin is two fused cyclohexane chairs; the *trans* fusion places the ring-junction bonds equatorial–equatorial. **Reasoning:** In trans-decalin both rings are rigid chairs joined equatorial–equatorial, so 1,3-diaxial interactions are minimised; it cannot ring-flip and is more stable (by about 11 kJ/mol) than cis-decalin, which has one axial junction bond. **Why the other options are wrong:**

- (B) Neither decalin is aromatic; both are saturated.
- (C) It is the *cis* isomer that is flexible; trans is locked.
- (D) Decalin contains no double bond.

Final Answer: Equatorial–equatorial fusion minimises strain $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q73

Q74.

Solution

Concept — Oppenauer oxidation: A secondary alcohol is oxidised to a ketone using a ketone (acetone) as the hydride acceptor and an aluminium alkoxide catalyst. **Reasoning:** It is the reverse of the Meerwein–Ponndorf–Verley reduction.

The mild, base-like conditions leave C=C double bonds untouched, which makes it valuable in steroid chemistry. **Why the other options are wrong:**

- (A) Rosenmund is a reduction (acid chloride $\rightarrow$ aldehyde).
- (B) MPV is the reduction direction (carbonyl $\rightarrow$ alcohol).
- (D) Swern oxidation uses DMSO/oxalyl chloride, not Al alkoxide/acetone.

Final Answer: It is the Oppenauer oxidation $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q74

Q75.

Solution

Concept — Amine basicity: Basicity depends on availability of the nitrogen lone pair. Alkyl groups (+I) increase it; resonance delocalisation decreases it. **Reasoning:** Ethylamine (+I, fully available lone pair) is the strongest base. In aniline the lone pair is delocalised into the ring, weakening it. In acetamide the lone pair is delocalised into the carbonyl, making it essentially non-basic. So (i) $>$ (ii) $>$ (iii). **Why the other options are wrong:**

- (A) Reverses the order; acetamide is the weakest, not the strongest.
- (B) Places aniline above ethylamine, but aniline's lone pair is delocalised.
- (C) Ranks acetamide above aniline, but the amide lone pair is the least available.

Final Answer: ethylamine $>$ aniline $>$ acetamide $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q75

Q76.

Solution

Concept — Ozonolysis: Ozone adds across a C=C bond to form an ozonide, which on reductive work-up (Zn/H_2O or Me_2S) cleaves into two carbonyl fragments. **Reasoning:** Each doubly bonded carbon becomes a carbonyl carbon. Reductive work-up stops at the aldehyde/ketone stage (oxidative work-up with H_2O_2 would give acids). This is ozonolysis. **Why the other options are wrong:**

- (A) Hydroboration–oxidation gives an anti-Markovnikov alcohol, no C=C cleavage.
- (B) Wacker oxidation converts a terminal alkene to a methyl ketone, not cleavage.
- (C) Dihydroxylation (OsO_4) gives a 1,2-diol without breaking the C–C bond.

Final Answer: It is ozonolysis $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q76

Q77.

Solution

Concept — Wurtz reaction: Two alkyl halide molecules couple with sodium metal in dry ether to give a symmetrical alkane. **Reasoning:** 2 R–X + 2 Na $\rightarrow$ R–R + 2 NaX. The method is best for symmetrical alkanes with an even number of carbons; mixed halides give product mixtures. **Why the other options are wrong:**

- (B) Kolbe electrolysis couples carboxylate salts electrolytically, not halides with Na.
- (C) Frankland reaction makes organozinc compounds.
- (D) Corey–House uses a lithium dialkylcuprate with an alkyl halide (allows unsymmetrical alkanes).

Final Answer: It is the Wurtz reaction $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q77

Q78.

Solution

Concept — Leaving-group ability: A good leaving group departs with the bonding electrons and is stabilised as a weak base (the conjugate base of a strong acid). **Reasoning:** Tosylate (–OTs) is the conjugate base of a strong sulfonic acid; the negative charge is spread over three oxygens, so it is a very weak base and an excellent leaving group, far better than hydroxide, methoxide or amide. **Why the other options are wrong:**

- (A) Hydroxide is a strong base, a poor leaving group.
- (C) Amide ($^{-}NH_2$) is a very strong base, an extremely poor leaving group.
- (D) Methoxide is also a strong base and poor leaving group.

Final Answer: Tosylate is the best leaving group $\Rightarrow$ B

Answer: (B) Go Back to Q78

Q79.

Solution

Concept — Thiophene: A five-membered aromatic heterocycle with one sulfur whose lone pair completes the aromatic sextet (6 π electrons). **Reasoning:** The figure shows a five-membered ring with a single S heteroatom. Thiophene is the sulfur analogue of furan/pyrrole and is the core of ticarcillin and clopidogrel. **Why the other options are wrong:**

- (A) Furan has oxygen, not sulfur.
- (B) Pyrrole has an N–H, not sulfur.
- (C) Imidazole is a five-membered ring with two nitrogens.

Final Answer: The heterocycle is thiophene $\Rightarrow$ D

Answer: (D) Go Back to Q79

Q80.

Solution

Concept — Peroxide (Kharasch) effect: In the presence of peroxides, HBr adds to alkenes by a free-radical chain, giving the anti-Markovnikov product. **Reasoning:** A bromine radical adds to the terminal carbon, generating the more stable

secondary carbon radical; abstraction of H then places Br on C1. The net result is 1-bromopropane, the anti-Markovnikov product. This reversal is the Kharasch peroxide effect (only HBr behaves so). **Why the other options are wrong:**

- (A) Markovnikov addition (ionic, no peroxide) gives 2-bromopropane.
- (C) Saytzeff orientation concerns elimination, not addition.
- (D) Hofmann orientation also concerns elimination regiochemistry.

Final Answer: It is the Kharasch (peroxide) effect $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q80

Q81.

Solution

Concept — Resonance in the nitro group: The two equivalent N–O resonance forms delocalise the negative charge over both oxygens. **Reasoning:** Because the contributors are identical, the two N–O bonds are equal (bond order ≈ 1.5). The group withdraws electrons strongly by both resonance and induction, so it is a powerful deactivator and meta-director on a benzene ring. **Why the other options are wrong:**

- (A) The nitro group withdraws, never donates, electrons by resonance.
- (B) It is highly polar, and the two N–O bonds are equal, not different.
- (D) The nitrogen bears a formal $+$ charge; the group is not basic.

Final Answer: Strong electron-withdrawing group, equal N–O bonds $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q81

Q82.

Solution

Concept — Swarts reaction: Conversion of an alkyl chloride/bromide to an alkyl fluoride using a metallic fluoride. **Reasoning:** Heating R–Cl with AgF, Hg_2F_2 or SbF_3 exchanges the halogen for fluorine. It is the classical laboratory route to alkyl fluorides (and to early CFC refrigerants). **Why the other options are wrong:**

- (B) Finkelstein exchanges to *iodide* using NaI/acetone.
- (C) Hunsdiecker is a decarboxylative bromination of a silver carboxylate.
- (D) Wurtz–Fittig couples an aryl with an alkyl halide using Na.

Final Answer: It is the Swarts reaction $\Rightarrow$ A

Answer: (A) Go Back to Q82

Q83.

Solution

Concept — Epimers: Diastereomers that differ in configuration at only one of several stereocentres. **Reasoning:** D-Glucose and D-mannose are identical at every stereocentre except C2; differing at a single centre makes them C2-epimers. **Why the other options are wrong:**

- (B) Enantiomers differ at *every* stereocentre (mirror images).
- (C) Anomers differ specifically at the new anomeric (C1) centre formed on ring closure.
- (D) Conformers differ only by bond rotation, not configuration.

Final Answer: They are epimers $\Rightarrow$ A

Answer: (A) Go Back to Q83

Q84.

Solution

Concept — Imine (Schiff base): The C=N condensation product of a primary amine with an aldehyde or ketone, formed with loss of water. **Reasoning:** The structure shows a carbon doubly bonded to nitrogen that bears an R″ group, i.e. $R_2C{=}N{-}R''$, the defining feature of an imine/Schiff base. **Why the other options are wrong:**

- (A) An enamine has C=C–N (nitrogen on an sp^2 carbon adjacent to the double bond), formed from a *secondary* amine.
- (C) An amide has C(=O)–N, a carbonyl not a C=N.
- (D) A nitrile is C≡N, a triple bond.

Final Answer: It is an imine (Schiff base) $\Rightarrow$ B

Answer: (B) Go Back to Q84

Q85.

Solution

Concept — Macrolides: Antibiotics built on a large macrocyclic lactone ring carrying deoxy-sugar residues; they bind the 50S ribosomal subunit. **Reasoning:** Erythromycin (14-membered lactone), azithromycin and clarithromycin are macrolides. They block the 50S exit tunnel, inhibiting bacterial protein synthesis. **Why the other options are wrong:**

- (A) Tetracyclines are four-fused-ring compounds acting at the 30S subunit.
- (B) Aminoglycosides are amino-sugars acting at the 30S subunit.
- (C) β-Lactams inhibit cell-wall synthesis, not the ribosome.

Final Answer: They are macrolides $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q85

Q86.

Solution

Concept — HIV protease inhibitors: The "-navir" drugs block the viral aspartic protease that cleaves Gag-Pol polyproteins. **Reasoning:** By inhibiting HIV protease, saquinavir and its congeners prevent maturation of viral structural proteins, so non-infectious virions are produced. They are typically peptidomimetic transition-state analogues. **Why the other options are wrong:**

- (B) Reverse-transcriptase inhibition is the action of nucleoside analogues (the "-vudine" drugs), not protease inhibitors.
- (C) Neuraminidase inhibitors (e.g. oseltamivir) target influenza.
- (D) DNA alkylation is an anticancer mechanism, not antiviral here.

Final Answer: They inhibit HIV aspartic protease $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q86

Q87.

Solution

Concept — Glucocorticoid SAR: Targeted substituents on the steroid nucleus raise anti-inflammatory potency and metabolic stability. **Reasoning:** A 9α-fluoro substituent boosts glucocorticoid activity, while 16-methylation (as in dexametha-

sone/betamethasone) reduces unwanted mineralocorticoid (salt-retaining) effects and prolongs action, giving high-potency agents. **Why the other options are wrong:**

- (A) Removing the steroid nucleus would abolish receptor binding.
- (B) Glucocorticoid activity requires the full steroid ring system, not a single benzene ring.
- (D) A quaternary ammonium salt is unrelated to glucocorticoid SAR.

Final Answer: 9α-fluoro + 16-methyl on the steroid nucleus $\Rightarrow$ C

Answer: (C) Go Back to Q87

Q88.

Solution

Concept — IC_{50}: A standard measure of inhibitor potency in a concentration–response assay. **Reasoning:** IC_{50} is the inhibitor concentration that lowers the measured activity to half its maximum under the assay conditions. A lower IC_{50} indicates a more potent inhibitor. **Why the other options are wrong:**

- (A) log$P$ describes lipophilicity, not inhibitory potency.
- (C) Hammett σ is an electronic substituent constant.
- (D) Molar refractivity is a bulk/polarisability descriptor.

Final Answer: It is the IC_{50} $\Rightarrow$ B

Answer: (B) Go Back to Q88

Q89.

Solution

Concept — Drug–receptor binding forces: Several non-covalent interactions of differing strength hold a drug at its site. **Reasoning:** Van der Waals (London dispersion) forces arise from transient induced dipoles, are the weakest, and fall off steeply as $1/r^6$, so they matter only at very close fit. Their cumulative effect over a good shape match can still be significant. **Why the other options are wrong:**

- (B) Ionic bonds are strong and longer-range ($1/r^2$).
- (C) Covalent bonds are the strongest of all, often irreversible.

- (D) Hydrogen bonds are stronger and more directional than dispersion forces.

Final Answer: Van der Waals (London) forces $\Rightarrow$ A

Answer: (A) Go Back to Q89

Q90.

Solution

Concept — Chirality and thalidomide: The two enantiomers of a chiral drug can have very different biological actions. **Reasoning:** One thalidomide enantiomer is the sedative, the other is teratogenic; because the stereocentre racemises *in vivo*, administering the "safe" enantiomer alone would not have prevented the toxicity. This is the cautionary lesson for chiral drug design. **Why the other options are wrong:**

- (A) Thalidomide does possess a stereocentre.
- (B) The enantiomers are not both inactive; they have distinct activities.
- (C) It is chiral and can be resolved (though it re-racemises).

Final Answer: Enantiomers differ in action and racemise *in vivo* $\Rightarrow$ D

Answer: (D) Go Back to Q90

Q91.

Solution

Concept — 4-Aminoquinoline antimalarials: Chloroquine acts inside the parasite's acidic food vacuole. **Reasoning:** As haemoglobin is digested, toxic free haem is normally detoxified by polymerisation to inert haemozoin. Chloroquine accumulates in the vacuole and inhibits this polymerisation, so toxic haem builds up and kills the parasite. **Why the other options are wrong:**

- (A) CYP51 inhibition is the antifungal azole mechanism.
- (C) The 30S ribosome is a bacterial target (aminoglycosides/tetracyclines).
- (D) HMG-CoA reductase is the statin target in humans.

Final Answer: It blocks haem detoxification (haemozoin formation) $\Rightarrow$ B

Answer: (B) Go Back to Q91

Q92.

Solution

Concept — Tetracycline chelation: The polar oxygen-rich tetracycline skeleton binds metal cations. **Reasoning:** Tetracyclines chelate Ca^{2+}, Mg^{2+}, Al^{3+} and Fe^{2+} (from milk, antacids, iron salts) to form insoluble, poorly absorbed complexes, lowering oral bioavailability. The same chelation explains their deposition in growing bone and teeth. **Why the other options are wrong:**

- (A) Acid lability is not the reason for the food/antacid interaction.
- (B) Lipophilicity is not the limiting factor here.
- (D) Photodegradation does not explain the metal-ion interaction.

Final Answer: They chelate di-/trivalent cations $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q92

Q93.

Solution

Concept — pH–rate profile: For specific acid- and base-catalysed hydrolysis, the rate constant depends on $[H^+]$ and $[OH^-]$. **Reasoning:** At low pH the H^+-catalysed term dominates and at high pH the OH^--catalysed term dominates, so a plot of $\log k_{obs}$ against pH is V-shaped (or U-shaped); the minimum identifies the pH of maximum stability used in formulation. **Why the other options are wrong:**

- (A) A flat line would mean no pH dependence, contrary to specific acid/base catalysis.
- (B) and (C) Pure monotonic trends ignore that *both* H^+ and OH^- catalyse the reaction.

Final Answer: A V-shaped pH–rate profile $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q93

Q94.

Solution

Concept — Collision theory: Rate depends on collision frequency, sufficient energy, and correct orientation. **Reasoning:** Even energetic collisions react only when the molecules are properly aligned. The steric (orientation) factor P (the

ratio of observed to collision-predicted rate) accounts for this geometric requirement. **Why the other options are wrong:**

- (B) The partition coefficient is a distribution property, not a kinetic factor.
- (C) The rate-determining step describes mechanism, not orientation probability.
- (D) Ionic strength affects rates of ionic reactions but is not the orientation factor.

Final Answer: It is the steric (orientation) factor $P \Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q94

Q95.

Solution

Concept — Cyanide antidote: Sodium thiosulfate supplies sulfur to the enzyme rhodanese. **Reasoning:** Sodium nitrite first generates methaemoglobin that traps cyanide; sodium thiosulfate then donates sulfur so that rhodanese converts cyanide to thiocyanate, which is far less toxic and renally excreted. **Why the other options are wrong:**

- (A) Calcium carbonate is an antacid/calcium source.
- (C) Magnesium sulfate is a saline cathartic/anticonvulsant.
- (D) Potassium iodide is an expectorant/iodine source, not a cyanide antidote.

Final Answer: Sodium thiosulfate $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q95

Q96.

Solution

Concept — Dieckmann condensation: The intramolecular version of the Claisen ester condensation. **Reasoning:** A 1,6- or 1,7-diester, treated with a base such as sodium ethoxide, cyclises so that one ester enolate attacks the other carbonyl, giving a five- or six-membered cyclic β-keto ester. Diethyl adipate thus gives ethyl 2-oxocyclopentanecarboxylate. **Why the other options are wrong:**

- (A) Reformatsky uses an α-halo ester with zinc and a carbonyl.

- (B) Stobbe is a condensation of succinate esters with a carbonyl.
- (D) Darzens forms an α, β-epoxy ester (glycidic ester).

Final Answer: It is the Dieckmann condensation $\Rightarrow$ C

Answer: (C) Go Back to Q96

Q97.

Solution

Concept — Anomeric effect: A stereoelectronic preference at the anomeric carbon for an electronegative substituent to lie axial. **Reasoning:** An axial C1 substituent allows favourable donation from a ring-oxygen lone pair into the σ^* of the C–OR bond (and avoids dipole–dipole repulsion). This stabilisation overrides the usual equatorial preference, so the axial anomer is favoured. **Why the other options are wrong:**

- (A) 1,3-Diaxial strain *opposes* the axial form; it is not the driving force.
- (B) The gauche effect is a related but distinct conformational preference about a single bond.
- (C) The "Hofmann effect" concerns elimination orientation, not anomers.

Final Answer: It is the anomeric effect $\Rightarrow$ D

Answer: (D) Go Back to Q97

Q98.

Solution

Concept — Rate-determining step: In a multistep reaction, the step with the highest transition-state energy (largest barrier from its starting point) controls the overall rate. **Reasoning:** The diagram shows two barriers with an intermediate I in the valley between them. Since TS_1 is higher than TS_2, the first step is the slowest and is rate-determining; the intermediate I is a genuine (if short-lived) species sitting in an energy minimum. **Why the other options are wrong:**

- (A) The presence of a valley (I) shows the reaction is stepwise, not concerted.
- (B) The later step is faster here (lower TS_2), so it is not rate-determining.
- (C) I sits in an energy minimum, so it is an intermediate, not a transition state.

Final Answer: The first step (higher TS_1) is rate-determining $\Rightarrow$ D

Answer: (D) Go Back to Q98

Q99.

Solution

Concept — Limitations of Beer's law: the law $A = \varepsilon cl$ assumes dilute, non-interacting absorbing species. **Reasoning:** At high concentration the average distance between solute particles shrinks, so each particle's charge distribution alters its neighbours' absorptivity, and the solution refractive index changes with concentration. These solute–solute effects make ε no longer constant, so A rises less than predicted and the curve bends towards the concentration axis (negative deviation). **Why the other options are wrong:**

- (A) Path length is fixed by the cell, not by concentration.
- (B) Detector sensitivity is not the cause of a chemical deviation from Beer's law.
- (C) ε is taken as constant in the ideal dilute limit; it does not rise on dilution.

Final Answer: Solute–solute interactions and refractive-index change at high c $\Rightarrow$ D

Answer: (D) Go Back to Q99

Q100.

Solution

Concept — Normality and equivalents: $N = M\times$ (equivalents per mole). **Reasoning:** In diazotization one mole of $NaNO_2$ reacts with one mole of primary aromatic amine, a $1:1$ reaction, so the equivalence factor is 1. Hence $N = 0.10 \times 1 = 0.10$ N; molarity and normality coincide. **Why the other options are wrong:**

- (B) 0.20 N wrongly assumes two equivalents per mole.
- (C) 0.050 N divides by 2 instead of using a $1:1$ ratio.
- (D) 0.010 N is a tenfold arithmetic error.

Final Answer: $N = 0.10 \times 1 = 0.10$ N $\Rightarrow$ A

Answer: (A) Go Back to Q100

Q101.

Solution

Concept — Masking in complexometry: a masking agent ties up interfering ions so only the target metal is titrated. **Reasoning:** Cyanide forms very stable, soluble cyanide complexes with heavy metals such as Cu, Ni, Zn and Cd, "hiding" them from EDTA. Calcium does not form a cyanide complex, so it alone reacts with EDTA, giving a selective titration. **Why the other options are wrong:**

- (A) Cyanide is not a redox indicator and signals no colour change.
- (B) The pH is fixed by a separate buffer, not by cyanide.
- (D) Cyanide is not a primary standard for EDTA.

Final Answer: Cyanide is a masking agent for interfering metals $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q101

Q102.

Solution

Concept — Affinity chromatography: separation by specific, reversible biological recognition. **Reasoning:** An immobilised ligand (e.g. a substrate analogue, antibody or metal-chelate) binds only the complementary target molecule from a mixture. Impurities pass through unretained; the captured target is later released by changing pH or adding a competing ligand. This biospecific binding gives very high selectivity. **Why the other options are wrong:**

- (A) Size-based sieving is size-exclusion chromatography, not affinity.
- (C) Charge-based separation is ion-exchange chromatography.
- (D) Volatilisation in a heated injector describes gas chromatography.

Final Answer: A specific, reversible biological interaction with the ligand $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q102

Q103.

Solution

Concept — IR fingerprint region: bands at 690–900 cm^{-1} arise from aromatic C–H out-of-plane bending. **Reasoning:** A strong absorption near 750 cm^{-1} is characteristic of aromatic C–H out-of-plane (oop) deformations. The exact pattern

of these low-wavenumber bands reveals the ring-substitution arrangement (mono-, ortho-, meta-, para-), so it is highly diagnostic. **Why the other options are wrong:**

- (B) Free O–H stretch is near 3600 cm^{-1}, not 750.
- (C) Aldehyde C=O stretch is near 1725 cm^{-1}.
- (D) N–H stretch appears around 3300–3500 cm^{-1}.

Final Answer: 750 cm^{-1} $\Rightarrow$ aromatic C–H out-of-plane bend $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q103

Q104.

Solution

Concept — Choosing a redox indicator: the indicator must change colour within the sharp potential break at the equivalence point. **Reasoning:** A redox indicator changes colour over a narrow potential window centred on its own transition potential. For a clean end point this window must fall inside the steep rise of the titration's potential–volume curve near equivalence, so the colour change coincides with completion of the reaction. **Why the other options are wrong:**

- (A) An indicator changing far below the break would flip too early.
- (C) A fixed zero-volt indicator would not match most systems.
- (D) A potential above the strongest oxidant would never be reached.

Final Answer: Within the potential break at the equivalence point $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q104

Q105.

Solution

Concept — Efficiency and resolution: for a fixed retention and selectivity, resolution improves as the plate count N rises ($R_s \propto \sqrt{N}$). **Reasoning:** Column II produces narrower peaks at the same retention times, which means each band has spread less, i.e. the column delivers more theoretical plates (higher efficiency). Narrower peaks at unchanged spacing give better baseline separation. **Why the other options are wrong:**

- (A) A larger void volume shifts, but does not sharpen, peaks.

- (B) Higher flow generally broadens peaks, worsening resolution.
- (C) A lower selectivity factor would reduce resolution, not improve it.

Final Answer: More theoretical plates (higher efficiency) narrow the peaks $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q105

Q106.

Solution

Concept — Inner-filter effect: at high concentration the sample reabsorbs its own exciting or emitted light. **Reasoning:** As concentration rises, molecules near the cell front absorb most of the excitation beam (primary inner-filter) and emitted photons are reabsorbed before leaving the cell (secondary inner-filter). The measured intensity therefore stops rising and bends downward, so fluorimetry is linear only at low concentration. **Why the other options are wrong:**

- (A) The Stokes shift is a fixed spectral property, not concentration-dependent.
- (B) Lamp drift is an instrumental fault, not the cause of this curvature.
- (D) Slit widths do not change on their own with concentration.

Final Answer: The inner-filter (self-absorption) effect $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q106

Q107.

Solution

Concept — Reaction stoichiometry: read mole ratios directly from the balanced equation. **Reasoning:** The equation has 2MnO_4^- reacting with $5\text{C}_2\text{O}_4^{2-}$, a $2:5$ ratio. Per mole of permanganate the oxalate consumed is $5/2 = 2.5$ mol. **Why the other options are wrong:**

- (A) 2.0 mol ignores the $2:5$ ratio.
- (C) 5.0 mol is the oxalate per 2 mol permanganate, not per 1.
- (D) 0.4 mol is the inverse ratio $(2/5)$.

Final Answer: $5/2 = 2.5$ mol oxalate per mole permanganate $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q107

Q108.

Solution

Concept — Guard column: a sacrificial pre-column that protects the analytical column. **Reasoning:** Placed between injector and analytical column, the guard column (same packing, short) retains particulates and strongly adsorbed sample components that would otherwise foul the main column. It is replaced cheaply and often, extending the analytical column's life without affecting the separation. **Why the other options are wrong:**

- (B) It does not raise the flow rate; the pump sets the flow.
- (C) The analytical column, not the guard, performs the separation.
- (D) The injector loop introduces sample; the guard does not.

Final Answer: It traps contaminants and protects the analytical column $\Rightarrow$ A

Answer: (A) Go Back to Q108

Q109.

Solution

Concept — NMR equivalence by symmetry: the number of signals equals the number of distinct proton environments. **Reasoning:** In *para*-xylene the two methyl groups are related by symmetry and are equivalent (one signal), and all four ring protons are equivalent by the molecular symmetry (one signal). So only **two** signals appear: aromatic H and CH_3. **Why the other options are wrong:**

- (A) 4 would require four inequivalent environments.
- (B) 3 over-counts; the para symmetry merges the ring protons.
- (C) 6 counts individual nuclei, not environments.

Final Answer: Two environments (Ar–H and CH_3) $\Rightarrow$ D

Answer: (D) Go Back to Q109

Q110.

Solution

Concept — Indicator selection: the indicator's colour-change range must straddle the equivalence-point pH. **Reasoning:** A weak acid versus strong base gives a salt of a weak acid, so the equivalence point is alkaline (pH $>$ 7). Phenolphthalein

(pH 8.3–10.0) changes colour in exactly this basic region, marking the end point sharply. **Why the other options are wrong:**

- (A) Methyl orange changes in the acidic range (3.1–4.4), too early.
- (B) Methyl red (4.4–6.2) is also acidic-side, before equivalence.
- (D) Bromophenol blue (3.0–4.6) changes far below the basic equivalence pH.

Final Answer: Phenolphthalein brackets the basic equivalence pH $\Rightarrow$ C

Answer: (C) Go Back to Q110

Q111.

Solution

Concept — Mass-spectral neutral losses: a loss of 18 corresponds to water. **Reasoning:** Alcohols readily undergo dehydration in the ion source, eliminating a neutral H_2O (mass 18) to give an M$-$18 fragment ion. This loss is a classic diagnostic clue for a hydroxyl group. **Why the other options are wrong:**

- (A) Loss of CH_3 is 15 mass units, not 18.
- (C) Loss of CO is 28 mass units.
- (D) Water is lost as a neutral molecule; the charged species is the retained fragment, not OH^-.

Final Answer: Loss of 18 = elimination of water $\Rightarrow$ B

Answer: (B) Go Back to Q111

Q112.

Solution

Concept — Titration-curve inflection: the steep drop marks the equivalence point. **Reasoning:** When a strong base is titrated with a strong acid, pH starts high and falls sharply once the added acid neutralises all the base. Point E, the steep inflection at V_{eq}, is where moles of acid added equal moles of base initially present, i.e. the equivalence point. **Why the other options are wrong:**

- (B) Maximum buffering occurs on the flat region, not the steep break.
- (C) Half-neutralisation lies earlier, on the gently sloping part.
- (D) Indicator oxidation is not an event represented on an acid–base curve.

Final Answer: E is the equivalence point $\Rightarrow$ A

Answer: (A) Go Back to Q112

Q113.

Solution

Concept — Spectral interference in flame photometry: overlapping emission from another element distorts a reading. **Reasoning:** When a second alkali metal emits light at or near the analyte's measurement wavelength, the photodetector collects both signals and the analyte reading is falsely high. This is a spectral (radiation) interference, corrected by better filters/optics or by matrix matching. **Why the other options are wrong:**

- (A) Path length is an instrument geometry factor, unrelated to this overlap.
- (B) The analyte's molar mass does not change in the flame.
- (D) The error is systematic (from the other element), not random noise.

Final Answer: A spectral (radiation) interference between elements $\Rightarrow$ C

Answer: (C) Go Back to Q113

Q114.

Solution

Concept — GC detector selectivity: the ECD responds strongly to electron-capturing (halogenated) species. **Reasoning:** The electron-capture detector uses a radioactive β-source to create a steady ion current; electronegative analytes such as organochlorines capture electrons and reduce the current. This makes the ECD extremely sensitive and selective for halogenated pesticide residues at trace levels. **Why the other options are wrong:**

- (A) FID responds to almost all organics but is not selective for halogens.
- (B) TCD is universal and far less sensitive for trace halogen work.
- (C) RID is a liquid-chromatography detector, not used in GC.

Final Answer: Electron-capture detector (ECD) $\Rightarrow$ D

Answer: (D) Go Back to Q114

Q115.

Solution

Concept — Releasing agent in AAS: a competing species frees the analyte from a refractory compound. **Reasoning:** Phosphate binds calcium as a heat-stable calcium phosphate that resists atomisation, lowering the signal (chemical interference). Added lanthanum binds the phosphate preferentially, "releasing" calcium so it atomises fully and the absorbance is restored. Lanthanum is therefore a releasing agent. **Why the other options are wrong:**

- (A) Lanthanum does not change the lamp wavelength.
- (C) It is not added to cool the flame or curb ionisation (that is an ionisation suppressor's role).
- (D) It is not used as an internal standard here.

Final Answer: Lanthanum acts as a releasing agent, freeing calcium $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q115

Q116.

Solution

Concept — Salt-gradient elution in ion exchange: added counter-ions displace bound analytes. **Reasoning:** Proteins bind to the charged resin by electrostatic attraction. Raising the salt concentration floods the system with small counter-ions that compete for the fixed charges, displacing the proteins. Weakly bound proteins elute first and strongly bound ones later, giving a separation ordered by binding strength. **Why the other options are wrong:**

- (A) Ion-exchange is run in solution; proteins are not volatilised.
- (B) Salt does not alter the detector path length.
- (D) Salt does not increase the proteins' molecular size.

Final Answer: Counter-ions compete for the sites and displace the proteins $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q116

Q117.

Solution

Concept — Back-titration rationale: used when the analyte reacts slowly or incompletely with the direct titrant. **Reasoning:** Insoluble magnesium hydroxide dissolves and reacts only sluggishly when titrated directly. Adding a measured *excess* of standard HCl drives the reaction to completion; the leftover acid is then back-titrated with standard NaOH, and the analyte is found by difference. **Why the other options are wrong:**

- (B) NaOH does react with HCl; that reaction is the basis of the back-titration.
- (C) Magnesium hydroxide is not a primary standard.
- (D) An indicator (or pH meter) is still required to find the end point.

Final Answer: The solid reacts incompletely directly, so excess acid ensures completion $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q117

Q118.

Solution

Concept — HPLC flow path end: pump $\rightarrow$ injector $\rightarrow$ column $\rightarrow$ detector $\rightarrow$ recorder/data system. **Reasoning:** The unit after the detector that converts the detector's electrical output into the printed or stored chromatogram is the recorder / data-acquisition system. It is the final element in the signal chain. **Why the other options are wrong:**

- (A) The reservoir is at the very start, before the pump.
- (C) The pump is upstream of the injector, not after the detector.
- (D) A guard column sits before the analytical column.

Final Answer: R is the recorder / data-acquisition system $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q118

Q119.

Solution

Concept — Absorbance ratio: a pharmacopoeial identity test compares absorbances at two maxima. **Reasoning:** The ratio is $A_{\lambda_1}/A_{\lambda_2} = 0.80/0.40 = 2.0$.

Because both absorbances are taken on the same solution in the same cell, the path length and concentration cancel, leaving a constant ratio characteristic of the substance. **Why the other options are wrong:**

- (A) 0.50 is the inverse ratio ($0.40/0.80$).
- (B) 1.0 would require equal absorbances at both maxima.
- (C) 0.40 is just the second absorbance, not the ratio.

Final Answer: $0.80/0.40 = 2.0 \Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q119

Q120.

Solution

Concept — TLC visualisation: iodine vapour is a universal, non-destructive locating reagent. **Reasoning:** Many organic compounds adsorb iodine vapour reversibly, appearing as brown spots against the pale plate. The method is non-destructive (spots fade as iodine sublimes off), so the analyte can be recovered for further work. Iodine is acting purely as a visualising/detection reagent. **Why the other options are wrong:**

- (A) Iodine vapour does not re-develop the chromatogram; it only stains spots.
- (B) It is a vapour applied afterwards, not a built-in fluorescent coating.
- (D) It is not a standard for calibrating R_f values.

Final Answer: Iodine vapour is a non-destructive visualising reagent $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q120

Q121.

Solution

Concept — Amperometry: current at a fixed applied potential is proportional to analyte concentration. **Reasoning:** The Clark electrode holds its working electrode at a potential where dissolved O_2 is electrochemically reduced. The resulting diffusion-limited current is proportional to the oxygen concentration, so measuring this current gives the dissolved-oxygen level. **Why the other options are wrong:**

- (B) Mass-to-charge measurement is mass spectrometry.
- (C) The sensor measures current, not a pH change.
- (D) Flame emission is a spectroscopic, not amperometric, method.

Final Answer: Current from the electrochemical reduction of $O_2 \Rightarrow \boxed{A}$

Answer: (A) Go Back to Q121

Q122.

Solution

Concept — OOS handling: a result outside specification triggers a formal documented investigation. **Reasoning:** Under GMP/QA, an out-of-specification value must first be investigated in a documented OOS procedure (laboratory phase, then full investigation if needed) to find whether it is a genuine result or an assignable error. Only after this can any decision on the batch be justified. **Why the other options are wrong:**

- (A) Releasing the batch while ignoring the value violates GMP.
- (C) Averaging a failing result with passing ones to mask it is prohibited.
- (D) Discarding the sample and reporting nothing destroys the evidence and breaches data integrity.

Final Answer: Initiate a documented OOS investigation first $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q122

Q123.

Solution

Concept — Taxonomical classification: Crude drugs may be arranged according to the botanical (taxonomic) position of the source plant, that is by phylum, order, family and genus. **Reasoning:** Grouping all Apocynaceae drugs together and all Solanaceae drugs together is sorting purely by botanical relationship, irrespective of the part used or the constituent, which is the defining feature of taxonomical classification. **Why the other options are wrong:**

- (A) Morphological grouping is by the plant part used.
- (C) Chemical grouping is by constituent class.
- (D) Pharmacological grouping is by therapeutic action.

Final Answer: Grouping by botanical family is taxonomical classification $\Rightarrow$ B

Answer: (B) Go Back to Q123

Q124.

Solution

Concept — Organized drugs: Organized drugs are direct plant organs (fruit, rhizome, wood, leaf, bark, root, seed) possessing a definite cellular structure; unorganized drugs are cell-free exudates and secretions. **Reasoning:** Dill fruit, ginger rhizome and quassia wood are all entire plant organs with cellular tissue, so set (C) is entirely organized. **Why the other options are wrong:**

- (A) Agar, mastic and isabgol husk-mucilage are cell-free products (agar and mastic are unorganized; the set is not organized).
- (B) Tragacanth, kino and catechu are exudates/dried extracts, all unorganized.
- (D) Gum acacia, aloe and opium are exudate, dried juice and latex, all unorganized.

Final Answer: Dill fruit, ginger rhizome and quassia wood are all organized $\Rightarrow$ C

Answer: (C) Go Back to Q124

Q125.

Solution

Concept — Chemotaxonomic classification: This modern approach groups plants (and their drugs) by the distribution of characteristic secondary metabolites, supported by serological and genetic data, as markers of natural relationship. **Reasoning:** Using the pattern of secondary-metabolite chemistry to infer botanical relationship is precisely chemotaxonomy (biochemical/serotaxonomic classification). **Why the other options are wrong:**

- (A) Alphabetical is merely by name order.
- (B) Morphological is by plant part.
- (C) Pharmacological is by therapeutic action.

Final Answer: Grouping by constituent-based natural relationship is chemotaxonomic $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q125

Q126.

Solution

Concept — Purine alkaloids: Caffeine is a methylxanthine (purine) alkaloid found in several beverage plants. **Reasoning:** Commercial caffeine is obtained chiefly from the dried ripe seeds (beans) of *Coffea arabica* (Rubiaceae), and also from tea and as a by-product of decaffeination. **Why the other options are wrong:**

- (B) *Papaver somniferum* yields morphine/codeine.
- (C) *Atropa belladonna* yields atropine/hyoscyamine.
- (D) *Rauwolfia serpentina* yields reserpine.

Final Answer: Source of caffeine is *Coffea arabica* $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q126

Q127.

Solution

Concept — Curare alkaloids: Tubocurarine is a bis-benzylisoquinoline alkaloid and the active muscle-relaxant principle of tube curare. **Reasoning:** It is obtained from the dried bark and stem of *Chondrodendron tomentosum* (Menispermaceae), the South American vine used to prepare arrow poison. **Why the other options are wrong:**

- (A) *Cinchona* yields quinine.
- (C) *Ephedra* yields ephedrine.
- (D) *Lobelia* yields lobeline.

Final Answer: Tubocurarine comes from *Chondrodendron tomentosum* $\Rightarrow$ B

Answer: (B) Go Back to Q127

Q128.

Solution

Concept — Pseudo-alkaloids: In pseudo-alkaloids the basic nitrogen is not derived from an amino acid; terpenoid and purine alkaloids fall in this group. **Reasoning:** Aconitine is a diterpenoid alkaloid whose carbon skeleton arises from the terpenoid (mevalonate) pathway, with nitrogen added later and not from an amino-acid precursor, so it is a terpenoid pseudo-alkaloid. **Why the other options are wrong:**

- (A) True alkaloids have amino-acid-derived ring nitrogen.
- (B) Protoalkaloids have amino-acid nitrogen outside a ring.
- (D) Glycoalkaloids are sugar-bearing steroidal alkaloids (e.g. solanine), which aconitine is not.

Final Answer: Aconitine is a terpenoid pseudo-alkaloid $\Rightarrow$ C

Answer: (C) Go Back to Q128

Q129.

Solution

Concept — Alkaloid precipitating reagents: Wagner's reagent is a solution of iodine in potassium iodide (iodine-potassium iodide). **Reasoning:** Alkaloids give a reddish-brown precipitate with Wagner's reagent, a standard positive precipitation test in preliminary phytochemical screening. **Why the other options are wrong:**

- (A) Bornträger's detects anthraquinones.
- (B) Molisch's tests for carbohydrates.
- (C) Legal's detects cardenolides.

Final Answer: Reddish-brown precipitate with iodine-KI is Wagner's test $\Rightarrow$ D

Answer: (D) Go Back to Q129

Q130.

Solution

Concept — Cardiac glycosides: Cardenolides are steroidal glycosides bearing a five-membered butenolide ring; several are found in Apocynaceae leaves. **Reasoning:** Oleandrin is the principal cardenolide glycoside of the dried leaf of *Nerium oleander* (oleander), used historically as a cardiotonic and known for its toxicity. **Why the other options are wrong:**

- (B) *Glycyrrhiza glabra* gives the saponin glycyrrhizin.
- (C) *Cassia angustifolia* gives anthraquinone sennosides.
- (D) *Dioscorea* gives the steroidal saponin diosgenin.

Final Answer: Oleandrin is from *Nerium oleander* $\Rightarrow$ A

Answer: (A) Go Back to Q130

Q131.

Solution

Concept — Bornträger's test: Free anthraquinones liberated and oxidised from anthraquinone glycosides give a pink-to-red colour in the ammoniacal (alkaline) layer. **Reasoning:** Cascarosides of cascara are anthraquinone glycosides; on the (modified) Bornträger's test the freed anthraquinones colour the ammonia layer

pink-red, confirming their presence. **Why the other options are wrong:**

- (A) Keller–Kiliani detects 2-deoxy sugars of cardiac glycosides.
- (C) Baljet detects cardiac-glycoside lactone rings.
- (D) Liebermann–Burchard detects sterols/triterpenes.

Final Answer: Anthraquinones are confirmed by Bornträger's test $\Rightarrow$ B

Answer: (B) Go Back to Q131

Q132.

Solution

Concept — Flavonoid glycosides: A flavonoid glycoside has a flavonoid aglycone (here the flavanone naringenin) joined to a sugar through an O-glycosidic bond. **Reasoning:** In naringin the aglycone naringenin is linked to the disaccharide rutinose; since the aglycone is a flavanone, naringin is classified as a flavonoid (flavanone) glycoside, and the rutinose-7-O linkage accounts for its bitterness. **Why the other options are wrong:**

- (A) The aglycone is a flavanone, not a steroidal cardenolide.
- (B) It bears no anthraquinone nucleus.
- (D) It yields no hydrogen cyanide, so it is not cyanogenetic.

Final Answer: Naringin is a flavonoid (flavanone) glycoside $\Rightarrow$ C

Answer: (C) Go Back to Q132

Q133.

Solution

Concept — Anthraquinone purgatives: The laxative drugs senna, rhubarb, cascara and frangula all act through anthraquinone glycosides that liberate emodin-type aglycones. **Reasoning:** Glucofrangulins of frangula bark hydrolyse to anthraquinone aglycones (frangula-emodin), which stimulate colonic motility, exactly as in senna and rhubarb. **Why the other options are wrong:**

- (A) Cardenolide aglycones are cardioactive, not purgative.
- (B) Saponin aglycones are not the purgative principle here.
- (C) Cyanogenetic aglycones liberate HCN, unrelated to laxation.

Final Answer: Glucofrangulins liberate anthraquinone (emodin) aglycones $\Rightarrow$ D

Answer: (D) Go Back to Q133

Q134.

Solution

Concept — Dill oil: The volatile oil of dill fruit is rich in the monoterpene ketone carvone together with limonene. **Reasoning:** Carvone gives dill (and caraway) its characteristic odour and contributes to its carminative use; it is the chief odour-bearing constituent. **Why the other options are wrong:**

- (B) Eugenol is the chief constituent of clove oil.
- (C) Cinnamaldehyde is from cinnamon bark.
- (D) Menthol is from peppermint oil.

Final Answer: Chief odour constituent of dill is carvone $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q134

Q135.

Solution

Concept — Ginger pungency: The pungent taste of ginger resides in its non-volatile oleoresin rather than its volatile oil. **Reasoning:** Gingerols are the chief pungent principles of fresh ginger; on drying and heating they partly dehydrate to the even more pungent shogaols. Together these account for the pungency. **Why the other options are wrong:**

- (A) Zingiberene/bisabolene are the volatile aroma sesquiterpenes, not the pungent principles.
- (C) Curcuminoids are the colouring principles of turmeric.
- (D) Piperine and chavicine are the pungent principles of pepper.

Final Answer: The pungent principles are gingerols and shogaols $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q135

Q136.

Solution

Concept — Mastic: Mastic is a pale-yellow oleoresin exudate used as a varnish and dental material. **Reasoning:** It is collected as a pathological exudate from incisions in the stem of *Pistacia lentiscus* (Anacardiaceae), grown chiefly on the island of Chios. **Why the other options are wrong:**

- (A) *Commiphora molmol* yields myrrh.
- (B) *Boswellia serrata* yields olibanum (frankincense).
- (D) *Ferula asafoetida* yields asafoetida.

Final Answer: Mastic is from *Pistacia lentiscus* $\Rightarrow$ C

Answer: (C) Go Back to Q136

Q137.

Solution

Concept — Agar chemistry: Agar is a sulphated galactan extracted from the cell walls of red algae, composed mainly of agarose and agaropectin. **Reasoning:** Its repeating units are galactose residues (D- and L-galactose, partly as 3,6-anhydro-L-galactose, with some sulphate), making agar a galactan polysaccharide that gels firmly and resists bacterial liquefaction. **Why the other options are wrong:**

- (A) Inulin is a fructan, not a galactan.
- (B) Agar is a seaweed wall polysaccharide, not a glucuronic-acid gum exudate.
- (C) Agar is not a tannin/resin.

Final Answer: Agar is a sulphated galactan polysaccharide $\Rightarrow$ D

Answer: (D) Go Back to Q137

Q138.

Solution

Concept — Acetate–mevalonate pathway: Three acetyl-CoA units condense to HMG-CoA, which is reduced to mevalonic acid and decarboxylated to the C5 isoprene units IPP and DMAPP. **Reasoning:** The scheme acetyl-CoA $\rightarrow$ HMG-CoA $\rightarrow$ mevalonate $\rightarrow$ IPP/DMAPP $\rightarrow$ terpenoids/steroids is the classical acetate–

mevalonate (mevalonic acid) pathway producing all terpenoids and steroids. **Why the other options are wrong:**

- (B) Shikimate gives aromatic amino acids, not isoprene units.
- (C) Acetate–malonate gives fatty acids and polyketides.
- (D) The pentose phosphate pathway supplies sugars/NADPH.

Final Answer: It is the acetate–mevalonate pathway ⇒ A

Answer: (A) Go Back to Q138

Q139.

Solution

Concept — Shikimic acid pathway: The shikimate route converts erythrose-4-phosphate and phosphoenolpyruvate through shikimic acid to aromatic compounds, including gallic acid. **Reasoning:** Gallic acid arises from an intermediate (3-dehydroshikimate/3-dehydroshikimic acid) of the shikimic acid pathway, and its galloyl esters form the hydrolysable gallotannins. Hence the aromatic ring is built via shikimate. **Why the other options are wrong:**

- (A) Mevalonate gives terpenoids, not gallic acid.
- (C) Glycolysis yields pyruvate/sugars.
- (D) The citric acid cycle is energy metabolism, not aromatic biosynthesis.

Final Answer: Gallic acid is formed via the shikimic acid pathway ⇒ B

Answer: (B) Go Back to Q139

Q140.

Solution

Concept — Bitterness value: The bitterness value expresses the intensity of bitter taste of a drug as the reciprocal of the highest dilution still recognisably bitter, compared with quinine hydrochloride. **Reasoning:** For bitter tonics such as quassia and gentian this taste-threshold parameter (in units relative to quinine hydrochloride) is the standard measure, so the description matches the bitterness value. **Why the other options are wrong:**

- (A) Swelling index measures volume increase of mucilaginous drugs.
- (B) Foaming index measures saponin-induced froth.

- (D) Acid value measures free fatty acids in oils.

Final Answer: The taste-threshold parameter is the bitterness value $\Rightarrow$ C

Answer: (C) Go Back to Q140

Q141.

Solution

Concept — Sclerenchymatous fibres: Sclerenchyma fibres are elongated, pointed, thick-walled lignified cells with a narrow lumen, common diagnostic elements of barks and woods. **Reasoning:** The pink-red colour with phloroglucinol/hydrochloric acid is the specific reaction for lignin, confirming that the drawn thick-walled, narrow-lumened, tapering cell is a lignified sclerenchymatous fibre. **Why the other options are wrong:**

- (A) Starch granules are rounded inclusions, not fibres.
- (B) Cotton trichomes are non-lignified and do not stain with phloroglucinol.
- (C) Pitted parenchyma cells are thin-walled and isodiametric.

Final Answer: The element is a lignified sclerenchymatous fibre $\Rightarrow$ D

Answer: (D) Go Back to Q141

Q142.

Solution

Concept — Retardation factor (R_f): R_f is the distance travelled by the solute divided by the distance travelled by the solvent front, both measured from the baseline. **Reasoning:** Here $R_f = d/D = 3.2/4.0 = 0.80$. The value is dimensionless and lies between 0 and 1, as required. **Why the other options are wrong:**

- (B) 1.25 is D/d (the inverse) and exceeds 1, impossible.
- (C) 0.32 misplaces the decimal.
- (D) 0.50 ignores the actual distances.

Final Answer: $R_f = 3.2/4.0 = 0.80 \Rightarrow$ A

Answer: (A) Go Back to Q142

Q143.

Solution

Concept — Sticky vs blunt ends: A restriction enzyme that cuts the two strands of its palindrome at staggered (off-centre) positions leaves complementary single-stranded overhangs called cohesive or "sticky" ends. **Reasoning:** The site AAGCTT read $5' \to 3'$ is the same on both strands. Cutting A|AGCTT on the top strand and the symmetric position on the bottom strand leaves a four-base 5′-AGCT overhang on each fragment. These complementary overhangs can re-anneal with any fragment cut by the same enzyme, which is exactly why such ends are used in cloning. **Why the other options are wrong:**

- (A) The cut is staggered, not central, so the ends are not blunt.
- (C) The overhangs are 5′ protruding and retain their phosphates, not 3′ recessed without phosphate.
- (D) Cleavage gives linear fragments with free ends, not single-stranded circles.

Final Answer: A staggered cut at AAGCTT yields 5′ AGCT sticky ends $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q143

Q144.

Solution

Concept — cDNA synthesis: A complementary-DNA (cDNA) copy of a mature, intron-free mRNA is made so that a eukaryotic coding sequence can be expressed in bacteria that cannot splice introns. **Reasoning:** Reverse transcriptase is an RNA-dependent DNA polymerase that uses the mRNA as a template and an oligo-dT (or random) primer to synthesise the first DNA strand. The RNA is then removed and the second strand made to give double-stranded cDNA. Because the template is the spliced mRNA, the cDNA contains only exons. **Why the other options are wrong:**

- (A) Terminal transferase adds nucleotides to 3′ ends without a template (homopolymer tailing).
- (B) Polynucleotide kinase phosphorylates 5′ ends; it is not a polymerase.
- (D) RNase H only degrades the RNA strand of an RNA:DNA hybrid; it does not synthesise DNA.

Final Answer: First-strand cDNA is made by reverse transcriptase $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q144

Q145.

Solution

Concept — Insertional inactivation of a marker: When the unique cloning site lies inside one of two antibiotic-resistance genes, inserting DNA there knocks out that resistance, giving a selectable change. **Reasoning:** Here the *Not*I site is within tet^R. A successful insert disrupts tet^R, so recombinant cells lose tetracycline resistance (become tetracycline-sensitive) but retain the intact kan^R, so they still grow on kanamycin. Replica plating thus identifies recombinants as $kan^R\ tet^S$. **Why the other options are wrong:**

- (B) The kanamycin gene is untouched, so kanamycin resistance is not lost.
- (C) Non-recombinants are resistant to both; recombinants are not.
- (D) The *ori* lies away from the *Not*I site and is not interrupted, so replication is normal.

Final Answer: Recombinants become tetracycline-sensitive but stay kanamycin-resistant $\Rightarrow$ A

Answer: (A) Go Back to Q145

Q146.

Solution

Concept — Vector dephosphorylation: Alkaline phosphatase removes the 5′ terminal phosphate groups from the linearised vector. Ligase needs a 5′ phosphate to form a phosphodiester bond, so a vector lacking these phosphates cannot self-ligate. **Reasoning:** A vector cut once has two compatible ends that ligase could simply re-join, regenerating empty plasmid and lowering cloning efficiency. After phosphatase treatment the vector ends have only 3′-OH groups, so the vector cannot circularise by itself; it can ligate only when the insert (which still carries 5′ phosphates) bridges the two ends. The remaining nicks are sealed in vivo. **Why the other options are wrong:**

- (A) Phosphatase removes phosphates; it does not add them.
- (B) Methylation to block restriction is done by methyltransferases, not phosphatase.
- (C) Phosphatase does not degrade RNA; that is the job of RNases.

Final Answer: Dephosphorylation stops the vector from self-circularising ⇒ D

Answer: (D) Go Back to Q146

Q147.

Solution

Concept — Inclusion bodies: High-level expression of a recombinant protein in *E. coli* often overwhelms the cell's folding machinery, so the protein precipitates inside the cytoplasm as dense, insoluble, misfolded aggregates known as inclusion bodies. **Reasoning:** For somatropin and similar non-glycosylated polypeptides, the inclusion bodies are first isolated, solubilised with a chaotrope (e.g. urea or guanidine), and then refolded to recover bioactive protein. They are a hallmark of strong intracellular over-expression. **Why the other options are wrong:**

- (B) Plasmids are the DNA vectors, not protein aggregates.
- (C) Endospores are dormant survival structures of certain Gram-positive bacteria, unrelated to over-expression.
- (D) Periplasmic vesicles are not where over-expressed cytoplasmic protein aggregates collect.

Final Answer: The insoluble aggregates are inclusion bodies ⇒ A

Answer: (A) Go Back to Q147

Q148.

Solution

Concept — Fed-batch culture: Substrate is fed gradually into the bioreactor during the run while product accumulates and nothing is removed until harvest. This keeps the substrate concentration low. **Reasoning:** Feeding the carbon source slowly prevents substrate inhibition and the overflow metabolism (Crabtree effect) that would otherwise divert sugar to unwanted by-products such as ethanol or acetate. Fed-batch therefore gives high cell densities and high product yields and is the standard mode for many recombinant-protein and antibiotic processes. **Why the other options are wrong:**

- (A) Pure batch adds all nutrients at the start, with no feeding.
- (C) In continuous (chemostat) culture fresh medium is added and broth removed at the same rate.
- (D) Solid-state surface culture grows organisms on a moist solid substrate,

not a fed liquid.

Final Answer: Incremental feeding with no withdrawal is fed-batch culture $\Rightarrow$ B

Answer: (B) Go Back to Q148

Q149.

Solution

Concept — Microbial origin of vitamin B_{12}: Cyanocobalamin is a cobalt-containing corrinoid synthesised only by certain prokaryotes. Neither plants nor animals can make it, so it must be supplied in the diet or by microbial fermentation. **Reasoning:** Industrial vitamin B_{12} is produced by fermentation with bacteria such as *Pseudomonas denitrificans* or *Propionibacterium freudenreichii*. Because the human requirement cannot be met by endogenous synthesis, B_{12} is an essential dietary vitamin obtained chiefly from animal foods or supplements. **Why the other options are wrong:**

- (A) Plants do not synthesise B_{12}; this is why strict vegetarians may become deficient.
- (B) It is a complex corrinoid coenzyme, not a simple amino acid.
- (D) It is a vitamin coenzyme, not a structural cell-wall carbohydrate.

Final Answer: Only certain microbes make the cobalt corrinoid; animals cannot $\Rightarrow$ C

Answer: (C) Go Back to Q149

Q150.

Solution

Concept — Downstream processing sequence: Recovery of a fermentation product begins with primary separation of biomass from the liquid broth, before any purification or polishing steps. **Reasoning:** For an extracellular product the target is dissolved in the broth, so the first step is to remove cells and insoluble solids by filtration or centrifugation. The clarified liquid then goes on to concentration, primary isolation, purification, and finally polishing/crystallisation. Cell removal must come first because later chromatographic and crystallisation steps require a particle-free feed. **Why the other options are wrong:**

- (A) Lyophilising the whole broth would dry everything, including cells and

salts, not separate them.
- (B) Crystallisation is a final purification/polishing step, not the first.
- (C) Chromatographic polishing comes near the end, after clarification and concentration.

Final Answer: Primary separation by filtration or centrifugation comes first $\Rightarrow$ D

Answer: (D) Go Back to Q150

Q151.

Solution

Concept — EC enzyme classes: The six EC classes are oxidoreductases (1), transferases (2), hydrolases (3), lyases (4), isomerases (5), and ligases (6). An enzyme catalysing electron transfer (oxidation–reduction) is an oxidoreductase. **Reasoning:** Glucose oxidase oxidises β-D-glucose to gluconolactone while reducing molecular oxygen to hydrogen peroxide; this is a redox reaction, so the enzyme is an oxidoreductase (EC 1). The H_2O_2 produced is what biosensors and clinical glucose assays detect. **Why the other options are wrong:**

- (B) Transferases move chemical groups between substrates, not electrons to O_2.
- (C) Hydrolases cleave bonds by adding water.
- (D) Ligases join two molecules using ATP hydrolysis.

Final Answer: An electron-transferring (redox) enzyme is an oxidoreductase $\Rightarrow$ A

Answer: (A) Go Back to Q151

Q152.

Solution

Concept — Lineweaver–Burk plot: Taking reciprocals of the Michaelis–Menten equation gives $\frac{1}{v} = \frac{K_m}{V_{max}} \cdot \frac{1}{[S]} + \frac{1}{V_{max}}$, a straight line. **Reasoning:** In this linear form the $1/v$-axis intercept (where $1/[S] = 0$) equals $1/V_{max}$, the slope equals K_m/V_{max}, and the intercept on the $1/[S]$-axis equals $-1/K_m$. The question asks for the vertical-axis intercept, which is therefore $1/V_{max}$, exactly as marked in the figure. **Why the other options are wrong:**

- (A) $-1/K_m$ is the intercept on the horizontal ($1/[S]$) axis, not the vertical

one.

- (C) K_m/V_{max} is the slope, not an intercept.
- (D) k_{cat} is a turnover rate constant, not read directly off this plot's y-intercept.

Final Answer: The $1/v$-axis intercept is $1/V_{max} \Rightarrow$ B

Answer: (B) Go Back to Q152

Q153.

Solution

Concept — Phases of bacterial growth: The batch curve runs lag (I) $\rightarrow$ log/exponential (II) $\rightarrow$ stationary (III) $\rightarrow$ death (IV). In the stationary phase, multiplication is offset by cell death, so the viable count plateaus. **Reasoning:** By phase III nutrients are depleted and toxic by-products accumulate, so new cell formation just balances cell death and the net specific growth rate falls to approximately zero, giving the flat top of the curve. Many secondary metabolites (such as antibiotics) are produced predominantly in this phase. **Why the other options are wrong:**

- (A) The lag phase (I) shows adaptation with little division, at the start of the curve.
- (B) The log phase (II) is the steep rising part with maximal, not zero, net growth.
- (D) The death phase (IV) shows a falling viable count, not a balanced plateau.

Final Answer: A plateau where growth balances death is the stationary phase $\Rightarrow$ C

Answer: (C) Go Back to Q153

Q154.

Solution

Concept — Biological indicators for sterilisation: A biological indicator uses standardised spores of an organism far more heat-resistant than ordinary contaminants. If these spores are killed, the cycle is validated. **Reasoning:** For moist-heat (autoclave) cycles the reference indicator is the endospore of *Geobacillus stearothermophilus* (formerly *Bacillus stearothermophilus*), whose spores are ex-

ceptionally resistant to wet heat at 121°C. (Dry-heat and ethylene-oxide cycles instead use *Bacillus atrophaeus* spores.) **Why the other options are wrong:**

- (A) *E. coli* is a non-spore-forming vegetative cell, easily killed, so it cannot validate sterilisation.
- (B) *Staphylococcus aureus* forms no spores and is not heat-resistant enough.
- (C) *Aspergillus niger* is a mould used in other contexts, not the steam-cycle bioindicator.

Final Answer: Steam-cycle validation uses *Geobacillus stearothermophilus* spores $\Rightarrow$ D

Answer: (D) Go Back to Q154

Q155.

Solution

Concept — Immunoglobulin classes and hypersensitivity: IgE binds with high affinity to Fcε receptors on mast cells and basophils. Allergen cross-linking of this surface-bound IgE triggers degranulation and release of histamine, the basis of type I (immediate) hypersensitivity. **Reasoning:** The scenario describes mast-cell degranulation on allergen exposure, the textbook mechanism of IgE-mediated allergy such as seasonal rhinitis. IgE is present in serum only in trace amounts because most of it is tissue-bound to mast cells. **Why the other options are wrong:**

- (B) IgG mediates opsonisation and neutralisation, not classic mast-cell allergy.
- (C) IgM is a pentamer of the early primary response and complement fixation.
- (D) IgA guards mucosal secretions and does not arm mast cells for allergy.

Final Answer: Mast-cell-arming antibody of type I allergy is IgE $\Rightarrow$ A

Answer: (A) Go Back to Q155

Q156.

Solution

Concept — Papain digestion of IgG: Papain cleaves IgG above the hinge disulphides into two identical Fab fragments (each with one antigen-binding site) and one Fc fragment (the constant stem of the two heavy chains). **Reasoning:** The region marked Y is the stem of the Y, i.e. the Fc region formed by the paired C_H2 and C_H3 constant domains of the heavy chains. The Fc carries the effector functions, complement (C1q) binding and Fc-receptor engagement, but it does not bind antigen. Antigen binding is confined to the Fab tips. **Why the other options are wrong:**

- (A) The variable paratope that binds antigen lies in the Fab arms, not the Fc stem.
- (C) The complementarity-determining regions are within the Fab variable domains.
- (D) The light-chain variable domain is part of the Fab, again at the arm tips.

Final Answer: The non-antigen-binding effector stem is the Fc region $\Rightarrow$ B

Answer: (B) Go Back to Q156

Q157.

Solution

Concept — ELISA reporter enzyme: In an enzyme immunoassay the detection antibody is conjugated to an enzyme that converts a substrate into a measurable coloured product, amplifying the signal. **Reasoning:** Horseradish peroxidase (HRP) is one of the two standard ELISA reporter enzymes (the other being alkaline phosphatase). With a chromogenic substrate such as TMB it gives a colour whose intensity is proportional to the bound analyte and is read on a plate reader. The enzyme must catalyse a colour change, which HRP does. **Why the other options are wrong:**

- (A) DNA ligase joins DNA strands and produces no colour.
- (B) *Taq* polymerase is a thermostable DNA polymerase used in PCR, not a colorimetric reporter.
- (D) Reverse transcriptase makes cDNA from RNA; it is not an ELISA reporter.

Final Answer: A common colorimetric ELISA reporter is horseradish peroxidase $\Rightarrow$ C

Answer: (C) Go Back to Q157

Q158.

Solution

Concept — Toxoid vaccines: A toxoid is a bacterial exotoxin chemically inactivated (classically with formaldehyde) so that it can no longer cause disease but still presents its antigenic epitopes to the immune system. **Reasoning:** The diphtheria (and tetanus) components of DPT are formaldehyde-detoxified toxins. They retain immunogenicity, so the body raises neutralising antitoxin antibodies without any risk from the active toxin. This defines a toxoid vaccine. **Why the other options are wrong:**

- (A) A live attenuated vaccine uses weakened but living organisms, not an inactivated toxin.
- (B) A killed whole-cell vaccine uses inactivated whole organisms, not a purified detoxified toxin.
- (C) A conjugate vaccine links a polysaccharide to a carrier protein; that is not what formalin-treated toxin is.

Final Answer: Formalin-detoxified exotoxin is a toxoid vaccine $\Rightarrow$ D

Answer: (D) Go Back to Q158

Q159.

Solution

Concept — HEPA filtration in aseptic areas: A High-Efficiency Particulate Air (HEPA) filter removes at least 99.97% of particles of 0.3 μm, the most-penetrating particle size, and is the standard for delivering sterile, particle-free air in laminar-flow cabinets and clean rooms. **Reasoning:** The unidirectional (laminar) air supplied to the work zone is first passed through a HEPA filter, which traps bacteria, spores, and fine particulates, giving Grade A air for sterile compounding. The defining specification, 99.97% at 0.3 μm, matches the HEPA filter exactly. **Why the other options are wrong:**

- (B) Sintered-glass filters clarify liquids, not air streams to HEPA efficiency.
- (C) A paper pre-filter only removes coarse dust and cannot sterilise air.
- (D) Activated charcoal adsorbs vapours and odours, not microorganisms by size exclusion.

Final Answer: Sterile laminar-flow air is supplied through a HEPA filter $\Rightarrow$ A

Answer: (A) Go Back to Q159

Q160.

Solution

Concept — MIC vs MBC: The minimum inhibitory concentration (MIC) is the lowest antibiotic concentration that prevents visible growth; the minimum bactericidal concentration (MBC) is the lowest that actually kills the organism. Inhibition does not require killing. **Reasoning:** The definition given, the lowest concentration that visibly inhibits growth after standard incubation with no killing requirement, is precisely the MIC. It is read as the first tube or well in a dilution series showing no turbidity. The MBC, determined by subculturing, is usually higher. **Why the other options are wrong:**

- (A) The MBC requires killing of the organism, a stricter end-point than inhibition.
- (C) LD_{50} is a toxicology measure of lethal dose in animals, unrelated to in-vitro susceptibility.
- (D) "Zone of equivalence" refers to optimal antigen–antibody ratios in precipitation, not susceptibility testing.

Final Answer: Lowest concentration that inhibits visible growth is the MIC $\Rightarrow$ B

Answer: (B) Go Back to Q160

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	D	3	C	4	A	5	B
6	C	7	D	8	A	9	C	10	B
11	D	12	A	13	C	14	B	15	D
16	A	17	C	18	D	19	B	20	A
21	C	22	D	23	B	24	C	25	A
26	B	27	C	28	D	29	A	30	B
31	C	32	D	33	A	34	B	35	B
36	D	37	A	38	C	39	D	40	A
41	B	42	C	43	D	44	A	45	B
46	C	47	D	48	A	49	C	50	B
51	D	52	A	53	C	54	B	55	D
56	A	57	C	58	B	59	D	60	A
61	C	62	B	63	D	64	A	65	B
66	C	67	D	68	A	69	C	70	A
71	D	72	B	73	A	74	C	75	D
76	D	77	A	78	B	79	D	80	B
81	C	82	A	83	A	84	B	85	D
86	A	87	C	88	B	89	A	90	D
91	B	92	C	93	D	94	A	95	B
96	C	97	D	98	D	99	D	100	A
101	C	102	B	103	A	104	B	105	D
106	C	107	B	108	A	109	D	110	C
111	B	112	A	113	C	114	D	115	B
116	C	117	A	118	B	119	D	120	C
121	A	122	B	123	B	124	C	125	D
126	A	127	B	128	C	129	D	130	A
131	B	132	C	133	D	134	A	135	B
136	C	137	D	138	A	139	B	140	C
141	D	142	A	143	B	144	C	145	A
146	D	147	A	148	B	149	C	150	D
151	A	152	B	153	C	154	D	155	A
156	B	157	C	158	D	159	A	160	B