NIPER JEE Pharmacy Subjects

Sample Paper – 5

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. Silver chromate (Ag_2CrO_4) has a molar solubility of 1.0×10^{-4} mol/L in pure water at 25°C. Using $K_{sp} = 4s^3$ for an A_2B-type salt, its solubility product is:

(A) 1.0×10^{-8}

(B) 1.0×10^{-12}

(C) 2.0×10^{-12}

(D) 4.0×10^{-12}

Q2. A drug has an amyl-acetate/water partition coefficient $P = 4$ (concentration ratio). It is extracted from 50 mL of aqueous solution using 50 mL

of amyl acetate (equal volumes) in a single step. The fraction of drug extracted into the organic layer is:

(A) $1/5$

(B) $1/4$

(C) $4/5$

(D) $1/2$

Q3. An emulsifier system is made of 25% Span 20 (HLB 8.6) and 75% Tween 20 (HLB 16.7). The resultant HLB of the blend (weighted average) is approximately:

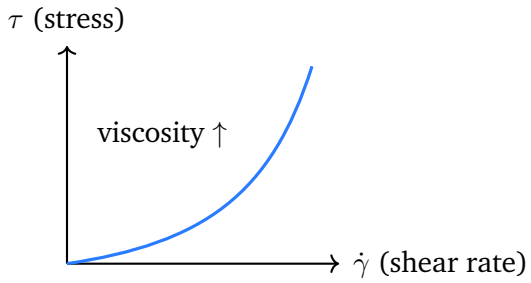
(A) 12.65

(B) 14.68

(C) 16.70

(D) 8.60

Q4. The flow curve below bows toward the shear-rate axis: as the shear rate $\dot{\gamma}$ rises, a disproportionately larger stress is needed (apparent viscosity increases with shear). This behaviour is:



(A) Newtonian flow

(B) pseudoplastic (shear-thinning) flow

(C) dilatant (shear-thickening) flow

(D) Bingham plastic flow

Q5. Kinematic viscosity equals dynamic viscosity divided by density. If a liquid has a dynamic viscosity of 2.0 poise and a density of 0.80 g/cm^3, its kinematic viscosity in stokes is:

(A) 1.6 stokes

(B) 0.40 stokes

(C) 1.0 stokes

(D) 2.5 stokes

Q6. In the capillary-rise method for measuring surface tension, the height h to which a wetting liquid rises in a capillary of radius r is:

(A) inversely proportional to the capillary radius r

(B) directly proportional to the capillary radius r

(C) independent of the surface tension

(D) proportional to the square of the radius

Q7. A microcrystalline-cellulose blend has a bulk density of 0.45 g/mL and a tapped density of 0.54 g/mL. Its Carr's compressibility index (rounded) and the corresponding flow rating are:

(A) 9%, excellent flow

(B) 17%, good flow

(C) 25%, poor flow

(D) 40%, very poor flow

Q8. For the same blend (bulk density 0.45 g/mL, tapped density 0.54 g/mL), the Hausner ratio is:

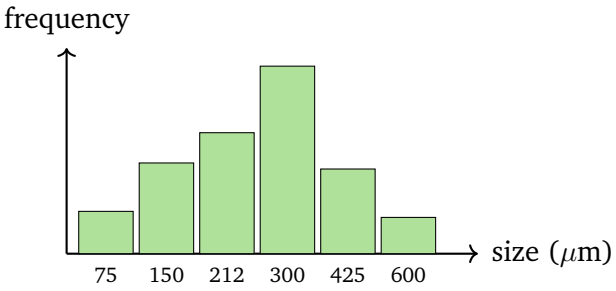
(A) 0.83

(B) 1.09

(C) 1.20

(D) 1.45

Q9. The number-frequency histogram of a milled powder is shown. The modal particle-size interval (in μm) is:



(A) 75 – 150

(B) 150 – 212

(C) 212 – 300

(D) 300 – 425

Q10. A carbonate buffer is prepared with 0.05 M $NaHCO_3$ (acid) and 0.50 M Na_2CO_3 (salt), with pK_{a2} of carbonic acid $= 10.3$ ($\log 10 = 1$). The pH of this buffer is approximately:

(A) 11.3

(B) 10.3

(C) 9.3

(D) 5.0

Q11. The freezing-point depression produced by blood plasma and tears is 0.52°C. A 1% w/v drug solution depresses the freezing point by 0.13°C. The percentage concentration of the drug required to render its solution isotonic is:

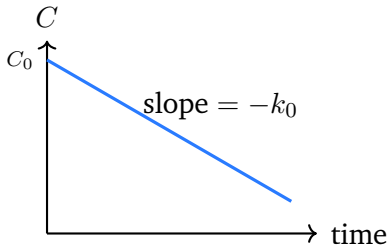
(A) 0.52%

(B) 4.0%

(C) 0.13%

(D) 2.0%

Q12. For a degrading drug suspension, a plot of the remaining drug concentration (C) against time gives a straight line of constant negative slope passing from C_0 downward (figure). The degradation follows:



(A) first-order kinetics

(B) second-order kinetics

(C) zero-order kinetics

(D) half-order kinetics

Q13. A drug in solution degrades by first-order kinetics with a rate constant $k = 3.5 \times 10^{-3}$ month^{-1}. Using $t_{90} = 0.105/k$, its shelf life (time for 10% loss) is approximately:

(A) 3 months

(B) 12 months

(C) 198 months

(D) 30 months

Q14. In accelerated stability testing, the Q_{10} value (the factor by which the degradation rate changes for a 10°C rise in temperature) for many pharmaceutical reactions is commonly taken to be about:

(A) 2 to 4

(B) 0.1 to 0.2

(C) exactly 1

(D) 50 to 100

Q15. The Langmuir adsorption isotherm $\dfrac{x}{m} = \dfrac{y_m bC}{1 + bC}$ is linearised for the determination of y_m and b by plotting:

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

(B) $\dfrac{C}{(x/m)}$ versus C (straight line)

(C) x/m versus C

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

Q16. According to the Stokes–Einstein equation $D = \dfrac{kT}{6\pi\eta r}$, if the hydrodynamic radius r of a spherical drug molecule is doubled (temperature and viscosity unchanged), the diffusion coefficient D will:

(A) double

(B) increase four-fold

(C) fall to half its value

(D) remain unchanged

Q17. In a phase-solubility study, the total solubility of a poorly soluble drug rises linearly with the concentration of a complexing agent and the slope of the line is less than 1. This A_L-type diagram indicates the formation of a:

(A) soluble $1{:}1$ complex that increases apparent solubility

(B) insoluble precipitate at all concentrations

(C) covalent drug-ligand bond

(D) eutectic mixture

Q18. In the Noyes–Whitney equation $dC/dt = \dfrac{DA}{h}(C_s - C)$, reducing the particle size of a drug from 50 μm to 5 μm chiefly accelerates dissolution because it:

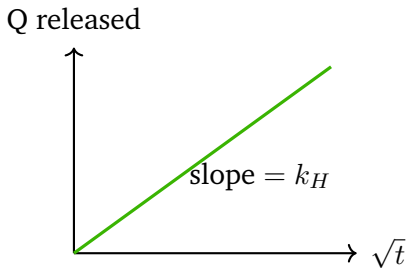
(A) decreases the saturation solubility C_s

(B) thickens the diffusion layer h

(C) lowers the diffusion coefficient D

(D) greatly increases the effective surface area A

Q19. The cumulative amount of drug released from a granular (porous) matrix tablet is plotted against the square root of time and yields a straight line through the origin (figure). The release kinetics obey the:



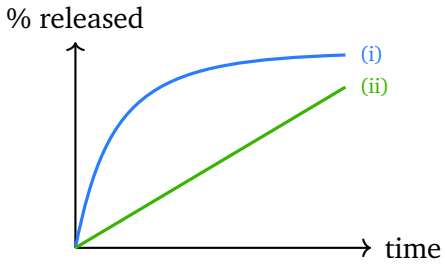
(A) zero-order model

(B) Higuchi (matrix-diffusion) model

(C) first-order model

(D) Hixson–Crowell cube-root model

Q20. The two dissolution profiles below plot cumulative % released against time. Profile (i) rises steeply and plateaus within minutes, while profile (ii) climbs slowly toward a near-linear, prolonged release. Profile (ii) is characteristic of a:



(A) fast-dissolving orodispersible tablet

(B) effervescent tablet

(C) sustained-release (controlled-release) dosage form

(D) intravenous bolus

Q21. An orally administered drug undergoes extensive hepatic first-pass metabolism. Compared with an intravenous dose, the most likely consequence for the same drug given orally is a:

(A) reduced systemic bioavailability (lower AUC)

(B) complete absence of any plasma concentration

(C) higher peak plasma concentration than IV

(D) unchanged bioavailability of 100%

Q22. For dissolution testing of capsules that tend to float, the USP dissolution Apparatus 1, which holds the dosage form in a rotating wire-mesh cage, is the:

(A) paddle apparatus

(B) flow-through cell

(C) reciprocating cylinder

(D) rotating basket apparatus

Q23. Povidone (PVP K-30) added to a wet-granulation tablet formulation functions principally as a:

(A) glidant

(B) binder (granulating agent)

(C) lubricant

(D) disintegrant

Q24. The crushing strength (hardness) of a compressed tablet is measured with a Monsanto or Pfizer hardness tester and is conventionally expressed in:

(A) percent weight loss

(B) minutes to disintegrate

(C) kilograms-force (kg) or newtons

(D) grams per millilitre

Q25. Which of the following is a synthetic *superdisintegrant* that acts mainly by rapid capillary wicking rather than extensive swelling?

(A) Crospovidone (crosslinked povidone)

(B) Liquid paraffin

(C) Magnesium stearate

(D) Sucrose

Q26. Water for Injection (WFI) used to compound parenterals must be free of pyrogens. The pharmacopoeial method of producing WFI that also removes pyrogens is:

(A) simple boiling at 100°C

(B) passage through activated charcoal only

(C) storage over silica gel

(D) distillation (or reverse osmosis to the required standard)

Q27. A suppository mould delivers 2 g of cocoa-butter base per cavity. A drug with a displacement value of 1.5 (1.5 parts of drug displace 1 part of base) is incorporated at 0.45 g per suppository. The mass of base required per suppository is:

(A) 2.0 g

(B) 1.7 g

(C) 1.55 g

(D) 0.45 g

Q28. The slow, *reversible* upward migration of dispersed oil droplets to form a concentrated layer at the top of an o/w emulsion, which can be redispersed by gentle shaking, is termed:

(A) cracking

(B) phase inversion

(C) creaming

(D) Ostwald ripening only

Q29. The sedimentation volume (F) of a pharmaceutical suspension is defined as the ratio of the:

(A) final (ultimate) sediment volume to the original suspension volume

(B) drug mass to the total volume

(C) supernatant volume to the sediment volume

(D) particle radius to the container radius

Q30. A polyethylene-glycol (PEG) ointment base prepared by blending PEG 400 with PEG 4000 is best classified as which type of base?

(A) oleaginous (hydrocarbon) base

(B) water-soluble base

(C) absorption base

(D) water-in-oil emulsion base

Q31. A hammer mill reduces particle size predominantly by the action of:

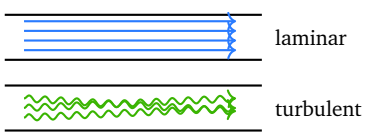
(A) attrition between two grinding plates only

(B) compression between heavy rollers

(C) cutting with sharp knives

(D) high-speed impact of swinging hammers on the feed

Q32. The two pipe-flow sketches below contrast smooth, parallel streamlines (upper) with chaotic, swirling eddies (lower). For Newtonian flow in a circular pipe, the lower pattern (turbulent) is expected when the Reynolds number (Re) exceeds about:



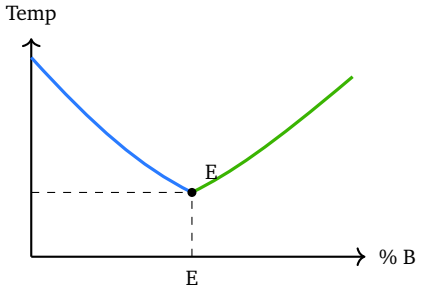
(A) 4000

(B) 2100

(C) 100

(D) 1.0

Q33. The binary temperature–composition diagram below shows two descending liquidus curves that meet at the lowest point E; below the dashed tie-line only solid A and solid B coexist. The point E represents the:



(A) eutectic point (lowest melting composition)

(B) triple point of a single substance

(C) critical solution temperature

(D) glass-transition temperature

Q34. In a *reservoir-type* transdermal therapeutic system, the constant (zero-order) rate of drug delivery to the skin is controlled chiefly by:

(A) the adhesive peel strength alone

(B) the colour of the backing membrane

(C) a rate-controlling polymeric membrane between the drug reservoir and the skin

(D) the patient's body temperature only

Part B: Pharmacology & Toxicology

Q35. A drug is given as a 600 mg IV bolus and the plasma concentration extrapolated to $t = 0$ is 15 mg/L. Assuming a one-compartment model, the apparent volume of distribution (V_d) is:

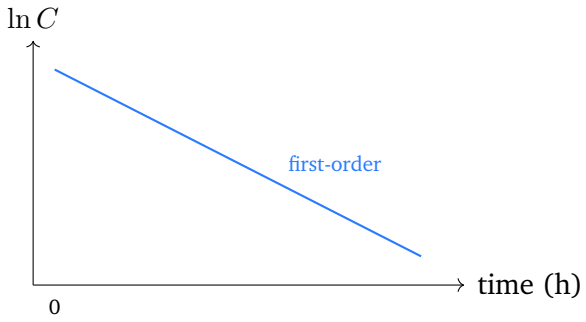
(A) 9 L

(B) 0.025 L

(C) 40 L

(D) 90 L

Q36. The semi-log plot of plasma concentration against time below is a straight line for a drug eliminated by a single first-order process. If the slope of the $\ln C$ vs t line is $-0.231\,\text{h}^{-1}$, the elimination half-life is approximately:



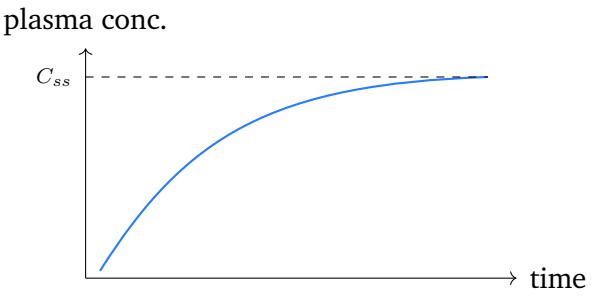
(A) 3 h

(B) 0.231 h

(C) 6.93 h

(D) 23.1 h

Q37. During a continuous IV infusion the plasma concentration climbs to the plateau (C_{ss}) shown below. If the drug has a total body clearance of 8 L/h and the target C_{ss} is 5 mg/L (bioavailability = 1), the required infusion (maintenance) rate is:



(A) 1.6 mg/h

(B) 40 mg/h

(C) 0.625 mg/h

(D) 13 mg/h

Q38. A highly plasma-protein-bound drug (about 99% bound) is displaced from albumin by a second drug, reducing binding to 98%. The most important pharmacological consequence is:

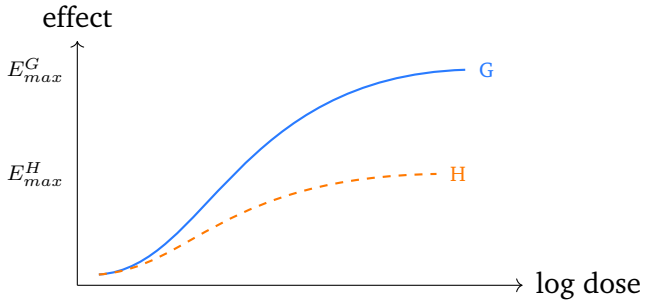
(A) The total plasma concentration immediately doubles

(B) Renal and hepatic clearance of the drug both fall sharply

(C) The drug becomes therapeutically inactive

(D) The free (unbound, pharmacologically active) fraction roughly doubles from 1% to 2%, transiently increasing effect and clearance of free drug

Q39. The two graded log dose-response curves below belong to agonists G and H acting at the same receptor. G reaches the full plateau; H reaches only a lower plateau even at maximal occupancy. The correct interpretation is:



(A) H is a partial agonist (lower intrinsic activity/efficacy) while G is a full agonist

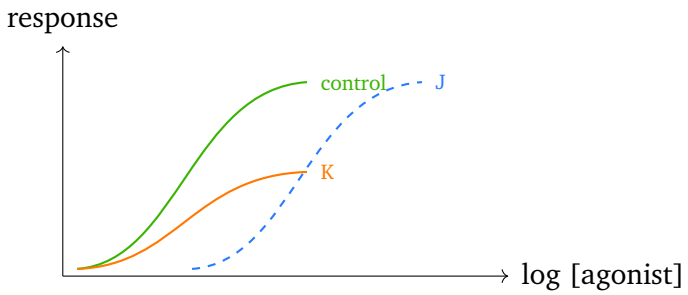
(B) H is more efficacious than G

(C) G and H have identical efficacy

(D) H is a pure competitive antagonist

Q40. Two antagonists are tested against an agonist. Antagonist J shifts the agonist curve rightward in parallel with no change in the maximum; an-

tagonist K lowers the maximum that cannot be restored by more agonist. The curves below identify J and K respectively as:



(A) J = non-competitive; K = competitive

(B) J = reversible competitive (surmountable); K = non-competitive (insurmountable)

(C) Both J and K are competitive antagonists

(D) J = inverse agonist; K = partial agonist

Q41. At a receptor showing constitutive (agonist-independent) activity, an inverse agonist is best defined as a ligand that:

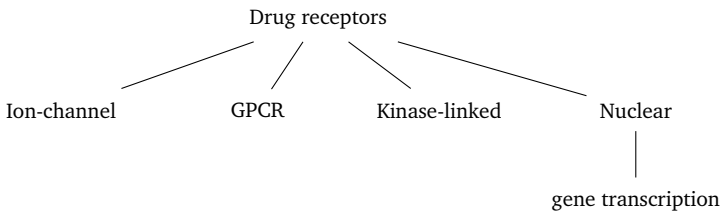
(A) Produces the same maximal effect as a full agonist

(B) Binds without affecting basal activity (a neutral antagonist)

(C) Binds the receptor and reduces its constitutive activity below the basal level (negative intrinsic activity)

(D) Has affinity but no effect on the receptor whatsoever

Q42. In the receptor superfamily tree below, the highlighted branch ends in "cytosolic/nuclear receptor → DNA response element → altered gene transcription" acting over hours. A ligand acting through this branch is:



(A) Acetylcholine at the nicotinic receptor

(B) Noradrenaline at the α_1 receptor

(C) Insulin at the insulin receptor

(D) Thyroid hormone (or a steroid such as testosterone) at its intracellular receptor

Q43. In receptor pharmacology, the term "affinity" of a drug for its receptor is best described as:

(A) The tendency of the drug to bind the receptor, reflected by the dissociation constant K_d (lower K_d = higher affinity)

(B) The maximal biological response the drug can produce

(C) The dose producing 50% of the lethal effect

(D) The rate at which the drug is metabolised

Q44. A patient stabilised on a CYP3A4-metabolised drug is started on a potent enzyme inducer. Over one to two weeks the steady-state concentration of the first drug falls. The most likely inducer among the following is:

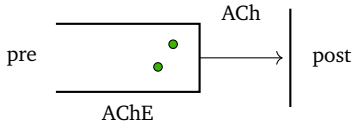
(A) Ketoconazole

(B) Erythromycin

(C) Rifampicin (a classic CYP enzyme inducer)

(D) Grapefruit juice

Q45. At the cholinergic neuromuscular synapse depicted, edrophonium is used in the diagnosis of myasthenia gravis (the Tensilon test) because it is a:



(A) Long-acting irreversible organophosphate cholinesterase inhibitor

(B) Very short-acting reversible cholinesterase inhibitor that transiently raises acetylcholine at the neuromuscular junction

(C) Competitive nicotinic antagonist at the motor end-plate

(D) Selective muscarinic M_3 agonist

Q46. Labetalol is useful in hypertensive emergencies and pregnancy-associated hypertension because, unlike a pure β-blocker, it:

(A) Blocks both α_1- and β-adrenergic receptors, lowering peripheral resistance without marked reflex tachycardia

(B) Is a selective β_2-agonist bronchodilator

(C) Acts only as a central α_2-agonist

(D) Is a calcium-channel blocker

Q47. Scopolamine (hyoscine) is preferred over many other antimuscarinics for motion sickness and as an antisecretory premedicant because it:

(A) Is a quaternary compound that cannot enter the CNS

(B) Is a selective nicotinic antagonist

(C) Selectively stimulates muscarinic receptors in the gut

(D) Is a tertiary-amine antimuscarinic with marked central (anti-emetic, sedative) actions in addition to peripheral antisecretory effects

Q48. Methyldopa lowers blood pressure (and is preferred in pregnancy) by a central mechanism in which it:

(A) Directly blocks peripheral α_1-receptors

(B) Is a non-selective β-blocker

(C) Is converted to α-methylnoradrenaline, a central α_2-agonist that reduces sympathetic outflow

(D) Inhibits angiotensin-converting enzyme

Q49. Adrenaline (epinephrine) is the drug of choice in anaphylactic shock because, acting on multiple adrenoceptors, it simultaneously:

(A) Causes α_1-mediated vasoconstriction (raising blood pressure), β_1 cardiac stimulation, and β_2 bronchodilation

(B) Acts purely as a selective α_2-agonist

(C) Blocks histamine H_1 receptors

(D) Is a pure β_1-antagonist

Q50. Buspirone treats generalised anxiety with little sedation or dependence, distinguishing it from benzodiazepines, because it acts mainly as a:

(A) Positive allosteric modulator of the GABA-A receptor

(B) Partial agonist at serotonin 5-HT_{1A} receptors

(C) Antagonist at dopamine D_2 receptors

(D) Histamine H_1 antagonist

Q51. Amitriptyline, a tricyclic antidepressant, relieves depression but causes anticholinergic and cardiac side effects because it:

(A) Selectively inhibits only the serotonin transporter

(B) Irreversibly inhibits monoamine oxidase

(C) Blocks reuptake of both noradrenaline and serotonin while also antagonising muscarinic, H_1 and α_1 receptors

(D) Is a selective dopamine reuptake inhibitor

Q52. Naltrexone is used orally in the long-term maintenance of detoxified opioid- and alcohol-dependent patients because it is a:

(A) Short-acting partial μ-agonist

(B) Selective κ-agonist analgesic

(C) NMDA receptor antagonist

(D) Long-acting, orally active competitive opioid-receptor antagonist that blocks the euphoric effects of opioids

Q53. Lamotrigine is broadly effective in partial and generalised seizures (and as a mood stabiliser) chiefly because it:

(A) Blocks voltage-gated sodium channels (stabilising their inactivated state) and thereby reduces glutamate release

(B) Blocks T-type calcium channels selectively

(C) Is an irreversible GABA-transaminase inhibitor

(D) Binds the SV2A vesicle protein

Q54. Modafinil promotes wakefulness in narcolepsy with a lower abuse potential than classical psychostimulants. Its wake-promoting action is most associated with:

(A) Strong direct agonism at β_2-adrenergic receptors

(B) Weak inhibition of the dopamine transporter (raising synaptic dopamine) together with effects on orexinergic/histaminergic wake pathways

(C) Irreversible monoamine oxidase inhibition

(D) Blockade of GABA-A chloride channels

Q55. Donepezil produces modest cognitive benefit in mild-to-moderate Alzheimer's disease because it is a:

(A) NMDA-receptor antagonist

(B) Muscarinic M_1 antagonist

(C) Reversible, centrally acting acetylcholinesterase inhibitor that raises synaptic acetylcholine in the brain

(D) Dopamine D_2 agonist

Q56. Ivabradine reduces heart rate in chronic stable angina and heart failure without affecting myocardial contractility or blood pressure because it:

(A) Blocks β_1-adrenergic receptors in the SA node

(B) Blocks L-type calcium channels in the myocardium

(C) Inhibits angiotensin-converting enzyme

(D) Selectively inhibits the funny current (I_f) in the sinoatrial node, slowing the rate of spontaneous depolarisation

Q57. Hydrochlorothiazide lowers blood pressure and causes a mild diuresis by inhibiting:

(A) The Na^+/Cl^- symporter in the early distal convoluted tubule

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

(C) Carbonic anhydrase in the proximal tubule

(D) The mineralocorticoid receptor in the collecting duct

Q58. Fenofibrate lowers plasma triglycerides chiefly because it:

(A) Competitively inhibits HMG-CoA reductase

(B) Activates peroxisome-proliferator-activated receptor-α (PPAR-α), increasing lipoprotein-lipase activity and fatty-acid oxidation

(C) Binds bile acids in the gut

(D) Inhibits intestinal cholesterol absorption at NPC1L1

Q59. Alteplase (recombinant tissue plasminogen activator) is used in acute myocardial infarction and ischaemic stroke because it:

(A) Blocks the platelet $P2Y_{12}$ ADP receptor

(B) Directly inhibits factor Xa

(C) Converts plasminogen to plasmin, which lyses fibrin in formed thrombi (a fibrinolytic)

(D) Potentiates antithrombin like heparin

Q60. Paracetamol (acetaminophen) is a good antipyretic and analgesic but a weak anti-inflammatory agent because it:

(A) Inhibits cyclooxygenase mainly in the central nervous system, with little effect on prostaglandin synthesis at peripheral inflammatory sites

(B) Irreversibly acetylates platelet COX-1

(C) Is a selective COX-2 inhibitor

(D) Antagonises histamine H_1 receptors

Q61. Omeprazole suppresses gastric acid more completely than an H_2-blocker because it:

(A) Neutralises acid already present in the stomach

(B) Blocks histamine H_2 receptors on parietal cells

(C) Is a muscarinic antagonist reducing vagal stimulation

(D) Irreversibly inhibits the H^+/K^+-ATPase (proton pump), the final common step of acid secretion in parietal cells

Q62. Aprepitant is added to antiemetic regimens for delayed chemotherapy-induced vomiting because it is a:

(A) 5-HT_3 receptor antagonist

(B) Substance-P / neurokinin-1 (NK_1) receptor antagonist

(C) Dopamine D_2 receptor antagonist

(D) Histamine H_1 receptor antagonist

Q63. Linezolid is active against multidrug-resistant Gram-positive organisms (including MRSA and VRE) because it:

(A) Binds penicillin-binding proteins, inhibiting cell-wall synthesis

(B) Inhibits DNA gyrase

(C) Binds the 50S ribosomal subunit (23S rRNA) and prevents formation of the 70S initiation complex, blocking protein synthesis at an early step

(D) Inhibits dihydrofolate reductase

Q64. Co-trimoxazole (sulfamethoxazole plus trimethoprim) is synergistic because the two drugs:

(A) Block two sequential steps of bacterial folate synthesis: sulfamethoxazole inhibits dihydropteroate synthase and trimethoprim inhibits dihydrofolate reductase

(B) Both inhibit the 30S ribosomal subunit

(C) Both inhibit bacterial cell-wall cross-linking

(D) Both inhibit DNA gyrase

Q65. Isoniazid is bactericidal against actively dividing *Mycobacterium tuberculosis* mainly because, after activation by the mycobacterial catalase-peroxidase (KatG), it:

(A) Inhibits the bacterial 30S ribosomal subunit

(B) Inhibits the synthesis of mycolic acids of the mycobacterial cell wall

(C) Inhibits DNA-dependent RNA polymerase

(D) Chelates magnesium needed for the ribosome

Q66. Chloroquine kills the erythrocytic schizonts of *Plasmodium* chiefly because it:

(A) Inhibits the parasite's dihydrofolate reductase

(B) Inhibits cytochrome electron transport in the parasite mitochondrion

(C) Generates free radicals through an endoperoxide bridge

(D) Concentrates in the food vacuole and inhibits haem polymerisation (haemozoin formation), so toxic free haem accumulates and kills the parasite

Q67. Carbimazole (and its active form methimazole) controls hyperthyroidism because it:

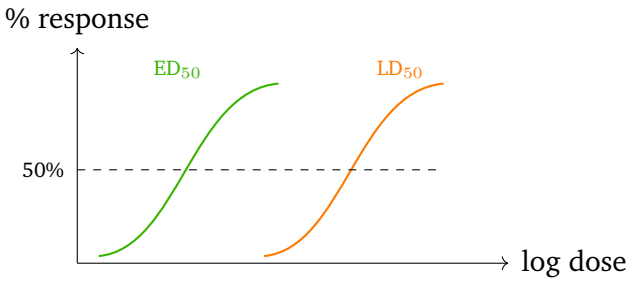
(A) Inhibits thyroid peroxidase, blocking iodination of tyrosine residues and coupling, and so reducing thyroid-hormone synthesis

(B) Blocks peripheral β-adrenergic receptors

(C) Destroys thyroid tissue by emitting beta radiation

(D) Is a thyroid-hormone receptor agonist

Q68. The therapeutic-index diagram below relates the median effective and median lethal doses; toxicology also relies on specific antidotes. Which antidote-poison pairing is INCORRECT?



(A) Digoxin toxicity → digoxin-specific antibody (Fab) fragments

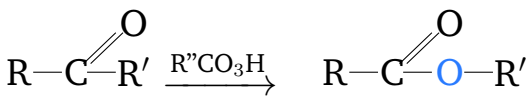
(B) Acute iron poisoning → deferoxamine

(C) Heparin overdose → vitamin K

(D) Warfarin overdose → vitamin K (phytonadione)

Part C: Pharmaceutical & Medicinal Chemistry

Q69. The scheme below shows a ketone being converted to an ester by a peroxy acid (e.g. *m*-CPBA), with insertion of an oxygen atom between the carbonyl carbon and the more substituted (migrating) group.



This oxidative oxygen-insertion that turns a ketone into an ester is the:

(A) pinacol–pinacolone rearrangement

(B) Cannizzaro reaction

(C) Baeyer–Villiger oxidation

(D) Wittig olefination

Q70. A ketone is heated with hydrazine (NH_2NH_2) and a strong base such as KOH in a high-boiling solvent, converting the C=O group completely to a CH_2 group. This basic-conditions deoxygenation of a carbonyl is the:

(A) Wolff–Kishner reduction

(B) Clemmensen reduction

(C) Rosenmund reduction

(D) Meerwein–Ponndorf–Verley reduction

Q71. A primary amide ($RCONH_2$), treated with bromine and aqueous NaOH, loses its carbonyl carbon as carbonate and gives a primary amine with one fewer carbon (RNH_2). This degradation is the:

(A) Gabriel synthesis

(B) Curtius rearrangement

(C) Schmidt reaction

(D) Hofmann bromamide (degradation) reaction

Q72. A stabilised carbanion (e.g. the enolate of diethyl malonate) adds to the β-carbon of an α,β-unsaturated carbonyl compound such as methyl vinyl ketone. This 1,4-conjugate addition of a nucleophile to an enone is the:

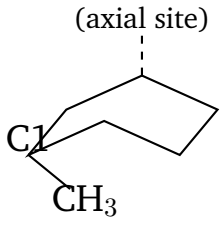
(A) Knoevenagel condensation

(B) Michael addition

(C) Perkin reaction

(D) Stobbe condensation

Q73. Methylcyclohexane exists predominantly in the chair conformation drawn below. The two chairs interconvert by ring-flip, which exchanges axial and equatorial positions.



At equilibrium the methyl group strongly prefers the equatorial orientation. The principal destabilising interaction suffered by an **axial** methyl group is:

(A) 1,3-diaxial (van der Waals) strain with the two axial hydrogens on the same face

(B) ring-opening to a straight chain

(C) loss of aromatic stabilisation

(D) torsional strain identical to that of the equatorial form

Q74. Benzene treated with sodium (or lithium) in liquid ammonia containing an alcohol as proton source is reduced to the non-conjugated 1,4-cyclohexadiene rather than to cyclohexane. This dissolving-metal partial reduction of an aromatic ring is the:

(A) catalytic hydrogenation

(B) Clemmensen reduction

(C) Birch reduction

(D) Rosenmund reduction

Q75. Consider the acidity (in water) of phenol and three chlorophenols. Chlorine is electron-withdrawing by induction, an effect that falls off with distance. Which of the following is the **strongest** acid?

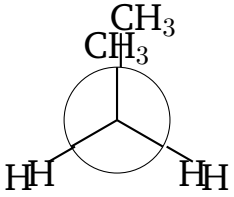
(A) phenol

(B) 4-chlorophenol (*para*)

(C) 3-chlorophenol (*meta*)

(D) 2,4,6-trichlorophenol (three electron-withdrawing chlorines)

Q76. The Newman projection below (sighting along C2–C3 of butane) shows the two methyl groups eclipsing each other (dihedral angle 0°).



With both methyls eclipsed (syn-periplanar), this conformation of butane is the:

(A) anti (lowest-energy) conformation

(B) fully eclipsed (syn) conformation, the highest-energy form

(C) gauche conformation

(D) a staggered conformation

Q77. An aldehyde condenses with a compound bearing an active methylene flanked by two electron-withdrawing groups (e.g. malonic acid or ethyl cyanoacetate), in the presence of a weak amine base, to form an α,β-unsaturated product with loss of water. This reaction is the:

(A) Knoevenagel condensation

(B) Cannizzaro reaction

(C) Wurtz reaction

(D) Kolbe–Schmitt reaction

Q78. In a polar protic solvent (e.g. methanol/water), the halide ions are ranked by nucleophilicity. The order observed is:

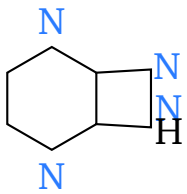
(A) $F^- > Cl^- > Br^- > I^-$

(B) all halides are equally nucleophilic

(C) $I^- > Br^- > Cl^- > F^-$ (the larger, less solvated ion is the better nucleophile)

(D) nucleophilicity is independent of solvation

Q79. The fused bicyclic aromatic system drawn below joins a six-membered pyrimidine ring to a five-membered imidazole ring and contains four nitrogen atoms. It is the core of adenine, guanine, caffeine and many xanthine drugs.



Identify this ring system.

(A) indole

(B) quinoline

(C) pteridine

(D) purine

Q80. Acid-catalysed dehydration of 2-methylbutan-2-ol gives mainly 2-methylbut-2-ene rather than 2-methylbut-1-ene. The major product is the more highly substituted alkene. This regiochemical preference is summarised by:

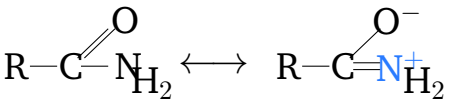
(A) the anti-Markovnikov rule

(B) Zaitsev's (Saytzeff's) rule – formation of the more substituted, more stable alkene

(C) Hofmann's rule – formation of the least substituted alkene

(D) Bredt's rule

Q81. The amide (peptide) bond shows restricted rotation about the C–N bond because of the delocalisation depicted by the two resonance contributors below.



The principal consequence of this resonance for the peptide bond is that:

(A) the C–N bond is a pure single bond and rotates freely

(B) the nitrogen is strongly basic, like an amine

(C) the C–N bond has partial double-bond character, so the amide unit is planar and rotation is restricted

(D) the carbonyl oxygen carries a full positive charge

Q82. A primary alkyl chloride or bromide is converted to the corresponding alkyl iodide by treatment with sodium iodide in acetone; the reaction

is driven by precipitation of insoluble NaCl/NaBr. This halide-exchange reaction is the:

(A) Wurtz reaction

(B) Hunsdiecker reaction

(C) Swarts reaction

(D) Finkelstein reaction

Q83. A pure sample of one enantiomer rotates plane-polarised light by $+52^\circ$ under standard conditions. A second sample of the same compound is found to rotate the light by $+26^\circ$. Assuming identical measurement conditions, the second sample is best described as:

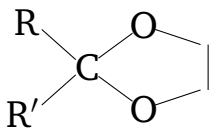
(A) a scalemic mixture with about 50% enantiomeric excess of the (+)-enantiomer

(B) a pure racemate (zero optical rotation)

(C) the pure (–)-enantiomer

(D) a meso compound

Q84. To protect a ketone during a later reaction, it is converted (with a diol and acid catalyst) into the cyclic structure drawn below, which is stable to base and nucleophiles and is later removed by aqueous acid.



This protected form of the carbonyl group is a(n):

(A) hemiacetal

(B) cyclic acetal (1,3-dioxolane)

(C) carboxylic anhydride

(D) Schiff base (imine)

Q85. Losartan, valsartan and irbesartan lower blood pressure by selectively blocking the AT_1 receptor. As a chemical class these "sartans" are:

(A) angiotensin-converting enzyme (ACE) inhibitors

(B) β-adrenergic receptor blockers

(C) thiazide diuretics

(D) angiotensin II receptor blockers (ARBs)

Q86. The oral antidiabetic sulfonylureas (e.g. glibenclamide, glimepiride) lower blood glucose chiefly by:

(A) closing the ATP-sensitive K^+ channel of the pancreatic β-cell, triggering insulin release

(B) inhibiting intestinal α-glucosidase

(C) activating peroxisome-proliferator-activated receptor γ (PPAR-γ)

(D) inhibiting the sodium–glucose cotransporter SGLT2 in the kidney

Q87. Imatinib revolutionised the treatment of chronic myeloid leukaemia. Mechanistically it acts as a:

(A) DNA alkylating agent

(B) topoisomerase poison

(C) small-molecule tyrosine-kinase inhibitor (it blocks the BCR–ABL kinase)

(D) folate antagonist

Q88. In medicinal chemistry, the *pharmacophore* of a drug is best defined as:

(A) the metabolic product responsible for toxicity

(B) the spatial arrangement of steric and electronic features essential for the molecule to interact with its biological target

(C) the counter-ion of the drug salt

(D) the excipient that aids dissolution

Q89. A technique that rapidly synthesises a very large number of structurally diverse compounds (a "library") in parallel for high-throughput screening, often on solid-phase resin beads, is known as:

(A) combinatorial chemistry

(B) fractional distillation

(C) Kjeldahl analysis

(D) gravimetric titration

Q90. During lead optimisation, a chemist systematically lengthens an alkyl side chain by adding successive –CH_2– units (CH_3, C_2H_5, C_3H_7 ...) and measures the effect on potency. This deliberate stepwise chain-lengthening of a series is called:

(A) ring contraction

(B) bioisosterism

(C) racemisation

(D) homologation (varying a homologous series)

Q91. The azole antifungals (e.g. fluconazole, ketoconazole) act by inhibiting a fungal cytochrome-P450 enzyme (lanosterol 14-α-demethylase, CYP51), thereby blocking the synthesis of:

(A) bacterial peptidoglycan

(B) ergosterol, the principal sterol of the fungal cell membrane

(C) viral DNA

(D) human cholesterol exclusively

Q92. Aminoglycoside antibiotics such as gentamicin and streptomycin are bactericidal because they bind irreversibly to which bacterial target, causing misreading of mRNA?

(A) the cell-wall transpeptidase (PBP)

(B) DNA gyrase

(C) the 30S ribosomal subunit

(D) dihydropteroate synthase

Q93. The decomposition of many drugs in solution follows first-order kinetics. A key feature of a **first-order** reaction is that its half-life ($t_{1/2}$):

(A) is independent of the initial concentration and equals $0.693/k$

(B) increases in direct proportion to the initial concentration

(C) depends on the square of the concentration

(D) is always exactly 24 hours

Q94. According to the Arrhenius equation $k = A\,e^{-E_a/RT}$, raising the temperature increases a reaction's rate constant chiefly because:

(A) the activation energy E_a falls to zero

(B) the pre-exponential factor A becomes negative

(C) the enthalpy of reaction changes sign

(D) a larger fraction of molecules acquire energy $\geq E_a$, raising the exponential term

Q95. Which pharmaceutical inorganic compound is administered orally as a **haematinic** to treat iron-deficiency anaemia?

(A) aluminium hydroxide

(B) ferrous sulfate

(C) sodium chloride

(D) zinc oxide

Q96. The silver salt of a carboxylic acid (RCOOAg), on treatment with bromine, is converted to the corresponding alkyl bromide (R–Br) with one fewer carbon atom and loss of CO_2. This decarboxylative halogenation is the:

(A) Hunsdiecker reaction

(B) Finkelstein reaction

(C) Reimer–Tiemann reaction

(D) Sandmeyer reaction

Q97. When D-glucose dissolves in water, the freshly prepared solution slowly changes its optical rotation until it reaches a constant equilibrium value. This phenomenon, due to interconversion of the α- and β-anomers through the open-chain form, is called:

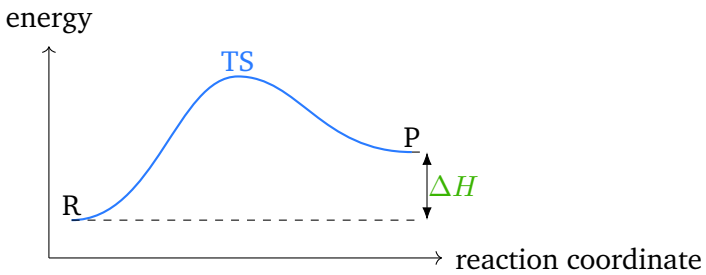
(A) racemisation

(B) tautomerism

(C) mutarotation

(D) epimerisation at every centre

Q98. The single-step reaction-coordinate diagram below has the product energy level lying **above** that of the reactants.



Because the products sit higher in energy than the reactants, the reaction is:

(A) exothermic, with a negative ΔH

(B) thermoneutral ($\Delta H = 0$)

(C) impossible at any temperature

(D) endothermic, with a positive ΔH (the products are less stable than the reactants)

Part D: Pharmaceutical Analysis & Quality Assurance

Q99. In bromometric assays of phenolic and aromatic-amine drugs, a standardised solution that liberates bromine *in situ* when acidified is normally used. This conventional titrant is a mixture of:

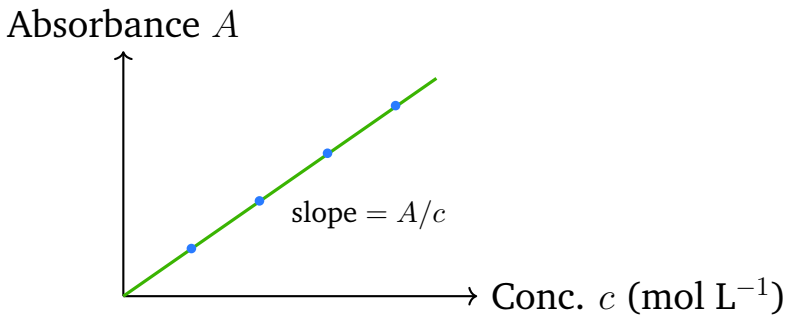
(A) Sodium thiosulphate and starch

(B) Iodine monochloride in acetic acid

(C) Potassium bromate and potassium bromide ($KBrO_3$/KBr)

(D) Ceric ammonium sulphate

Q100. The Beer–Lambert calibration line below was recorded for a drug at its λ_{max} in a 2.0 cm cell. The line passes through the origin with slope 5000 L mol^{-1} cm^{-1} $\times\, l$ shown as A/c. If the measured slope (A/c) is 1.0×10^4 L mol^{-1}, the molar absorptivity ε of the drug is:



(A) 1.0×10^4 L mol^{-1} cm^{-1}

(B) 5.0×10^3 L mol^{-1} cm^{-1}

(C) 2.0×10^4 L mol^{-1} cm^{-1}

(D) 1.0×10^2 L mol^{-1} cm^{-1}

Q101. The diazotization titration, used for the assay of primary aromatic amine drugs such as sulphanilamide, employs which titrant, the end point being detected with a starch-iodide external indicator (or electrometrically)?

(A) Standard potassium permanganate

(B) Standard silver nitrate

(C) Standard perchloric acid

(D) Standard sodium nitrite ($NaNO_2$)

Q102. In HPLC, "gradient elution" differs from "isocratic elution" in that gradient elution:

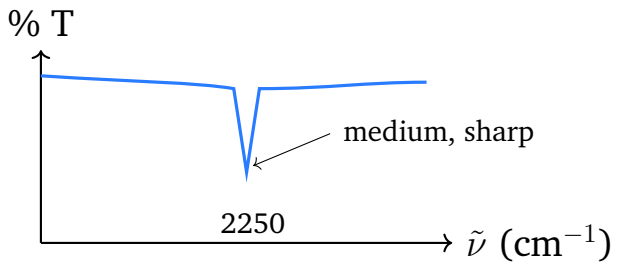
(A) Progressively changes the mobile-phase composition (e.g. % organic modifier) during the run

(B) Keeps the mobile-phase composition constant throughout the run

(C) Changes the stationary phase midway through the run

(D) Uses no pump, relying on gravity flow

Q103. The schematic IR spectrum below shows a single sharp band of medium intensity near **2250 cm^{-1}**, lying in the otherwise empty "triple-bond" region between the C–H and C=O regions. Such a band is most characteristic of which group?



(A) C=O stretch of an ester

(B) C≡N stretch of a nitrile

(C) O–H stretch of an alcohol

(D) C–Cl stretch of an alkyl halide

Q104. The oxygen-flask (Schöniger) combustion method is used in pharmaceutical analysis chiefly to:

(A) Determine the optical rotation of a chiral drug

(B) Measure the melting point of an organic solid

(C) Decompose an organic drug so that bound halogen or sulphur can be absorbed and then titrated

(D) Estimate the water content of a hygroscopic powder

Q105. On a TLC plate the solvent front advances 12.0 cm from the origin while a drug spot is centred 4.8 cm from the origin. The R_f value of the drug is:

(A) 0.40

(B) 2.50

(C) 0.48

(D) 0.60

Q106. The specific absorbance, written $A^{1\%}_{1\text{cm}}$ (also $E^{1\%}_{1\text{cm}}$), used in pharmacopoeial UV assays, is defined as the absorbance of a solution containing:

(A) 1 mole per litre in a 1 cm cell

(B) 1 microgram per mL in a 10 cm cell

(C) 1 mole per litre in a 10 cm cell

(D) 1 gram per 100 mL (i.e. 1% w/v) in a 1 cm cell

Q107. Arsenic trioxide (As_2O_3, molar mass 197.8 g mol^{-1}) is a primary standard for cerimetry. Each As(III) is oxidised to As(V), a 2-electron change, and one mole of As_2O_3 supplies 2 atoms of arsenic (4 electrons total). Its equivalent weight is therefore:

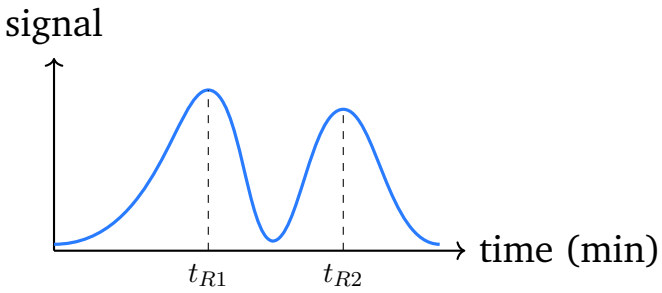
(A) 98.9 g eq^{-1}

(B) 49.45 g eq^{-1}

(C) 197.8 g eq^{-1}

(D) 395.6 g eq^{-1}

Q108. For the two adjacent peaks in the chromatogram, the void time is $t_0 = 1.0$ min and the retention times are $t_{R1} = 3.0$ min and $t_{R2} = 5.0$ min. The selectivity (separation) factor $\alpha = k'_2/k'_1 = (t_{R2} - t_0)/(t_{R1} - t_0)$ is:



(A) 1.67

(B) 1.25

(C) 2.0

(D) 0.5

Q109. Two ^{1}H NMR signals are separated by $\delta = 1.0$ ppm. When the spectrometer field is raised from 300 MHz to 600 MHz, the separation between the two signals expressed *in Hz*:

(A) Stays at the same number of Hz

(B) Falls to half the number of Hz

(C) Becomes zero

(D) Doubles (from 300 Hz to 600 Hz), because Hz separation scales with field while ppm stays constant

Q110. In capillary gas chromatography, "temperature programming" (raising the oven temperature during the run) is used mainly to:

(A) Elute components of widely differing volatility in a reasonable time with good peak shape

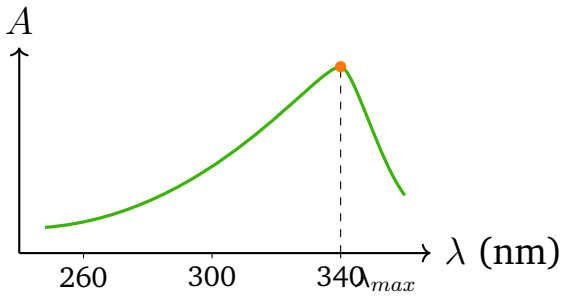
(B) Increase the polarity of the carrier gas

(C) Convert the column from non-polar to polar

(D) Lower the flame temperature of the detector

Q111. The UV absorption curve below is plotted against wavelength with the axis ticked at 260, 300 and 340 nm. For the quantitative assay the analyst

chooses the wavelength of maximum absorbance marked λ_{max}. Read from the figure, that wavelength is approximately:



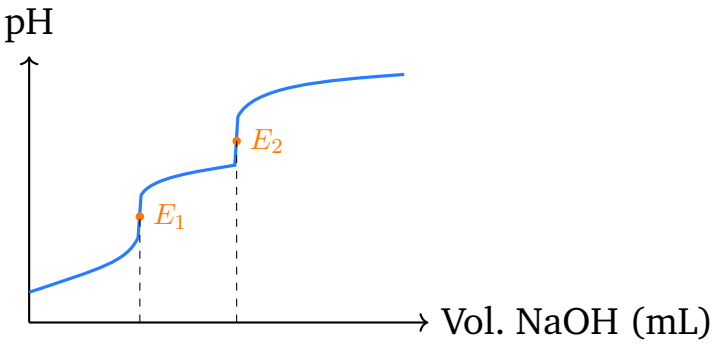
(A) 260 nm

(B) 300 nm

(C) 340 nm

(D) 220 nm

Q112. The curve shows the titration of a **diprotic** acid (such as carbonic or maleic acid) with NaOH. Two distinct steep regions, marked E_1 and E_2, appear on the curve. These two points correspond to:



(A) A single equivalence point measured twice

(B) The neutralisation of the first and second ionisable protons, respectively

(C) The two pK_a values being equal

(D) Decomposition of the indicator at two pH values

Q113. A strong cation-exchange resin used in ion-exchange chromatography carries which fixed functional group bonded to the resin backbone?

(A) Sulphonic acid groups ($-SO_3^-H^+$)

(B) Quaternary ammonium groups ($-NR_3^+$)

(C) Hydroxyl groups only

(D) Neutral C18 alkyl chains

Q114. In a mass spectrum, a fragment-ion peak appearing 15 mass units *below* the molecular-ion peak (i.e. at $m/z = \mathrm{M}-15$) most commonly arises from the loss of:

(A) A water molecule (H_2O)

(B) A carbonyl group (CO)

(C) A hydroxyl radical (OH)

(D) A methyl radical (CH_3, mass 15)

Q115. The peak asymmetry (tailing) factor in HPLC is often calculated at 5% of peak height as $A_s = b/a$, where a is the leading half-width and b the trailing half-width. For a peak with $a = 0.20$ min and $b = 0.30$ min, the asymmetry factor is:

(A) 0.67

(B) 1.5

(C) 0.50

(D) 6.0

Q116. In atomic absorption spectroscopy, a deuterium (D_2) continuum lamp is frequently fitted alongside the hollow-cathode lamp in order to:

(A) Correct for non-specific background absorption (scatter/molecular absorption)

(B) Atomise the sample in place of the flame

(C) Provide the sharp resonance line of the analyte element

(D) Act as the monochromator

Q117. In the mercurimetric titration of chloride, the chloride ion is titrated with a standard solution of mercuric nitrate, $Hg(NO_3)_2$, forming a soluble, slightly dissociated species. That species is:

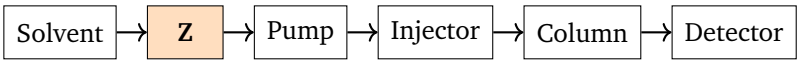
(A) Insoluble Hg_2Cl_2 (calomel) that precipitates

(B) Free chlorine gas

(C) Soluble, poorly dissociated mercuric chloride, $HgCl_2$

(D) Mercuric oxide, HgO

Q118. In the liquid-chromatograph block diagram below, identify the unit labelled **Z**, which is placed *before* the pump and removes dissolved air from the mobile phase to prevent bubble formation in the detector.



(A) The analytical column

(B) The injection valve

(C) The UV detector

(D) The mobile-phase degasser

Q119. Fluorimetry is frequently far more sensitive than ordinary UV absorption spectrophotometry for trace assay chiefly because the fluorescence signal is:

(A) A small difference between two large transmitted-light values

(B) Always at exactly the same wavelength as the absorption

(C) Measured as emitted light against a near-dark background, allowing low-level detection

(D) Independent of the concentration of the analyte

Q120. Oxalic acid ($H_2C_2O_4$) furnishes two replaceable protons per molecule. The normality of a 0.20 M oxalic acid solution (as an acid) is:

(A) 0.10 N

(B) 0.40 N

(C) 0.20 N

(D) 0.80 N

Q121. In classical polarography, the diffusion-controlled limiting current measured at the dropping-mercury electrode is, under fixed conditions, directly proportional to the:

(A) Concentration of the electroactive species (basis of quantitation)

(B) Square of the temperature

(C) Wavelength of the lamp

(D) Refractive index of the solution

Q122. Before running a pharmacopoeial HPLC assay, the analyst injects a standard several times and checks parameters such as resolution, tailing factor, theoretical plates and % RSD of repeated injections. This pre-analysis check is called the:

(A) Limit of detection study

(B) Linearity verification

(C) System suitability test

(D) Forced-degradation study

Part E: Pharmacognosy & Natural Products

Q123. In one well-known system, crude drugs are listed strictly by their Latin or English names in dictionary order, for example Acacia, Benzoin, Cinnamon, Dill. This simple but non-scientific basis of arranging crude drugs is called:

(A) Morphological classification

(B) Alphabetical classification

(C) Serological classification

(D) Chemotaxonomical classification

Q124. A monograph arranges crude drugs strictly according to their botanical position, grouping all members of the family Apocynaceae together and all members of Solanaceae together, irrespective of the plant part or active constituent. This basis of grouping is called:

(A) Taxonomical (botanical) classification

(B) Chemical classification

(C) Pharmacological classification

(D) Alphabetical classification

Q125. Among the following, which crude drug is an **unorganized** drug, being a cell-free oleo-gum-resin exudate rather than an entire plant organ?

(A) Fennel fruit

(B) Cardamom seed

(C) Benzoin (a balsamic resin exudate)

(D) Coriander fruit

Q126. Ergometrine (ergonovine), an oxytocic indole alkaloid of the ergoline group, is obtained from the dried sclerotium of a fungus that parasitises the ovary of rye. The biological source is:

(A) *Aspergillus niger*

(B) *Penicillium notatum*

(C) *Agaricus campestris*

(D) *Claviceps purpurea*

Q127. Piperine, the pungent acid-amide alkaloid responsible for the bite of black pepper and a known bio-enhancer, is obtained from the dried unripe fruits of:

(A) *Piper nigrum*

(B) *Zingiber officinale*

(C) *Elettaria cardamomum*

(D) *Capsicum frutescens*

Q128. Capsaicin, the vanillylamide responsible for the intense pungency of chillies and used in counter-irritant rubefacient preparations, is the chief pungent principle of the dried ripe fruits of:

(A) *Piper longum*

(B) *Capsicum annuum*

(C) *Brassica juncea*

(D) *Myristica fragrans*

Q129. A clarified, slightly acidic plant extract gives a **cream-coloured precipitate** on adding a few drops of potassium mercuric iodide reagent. This positive precipitation reaction for alkaloids is the:

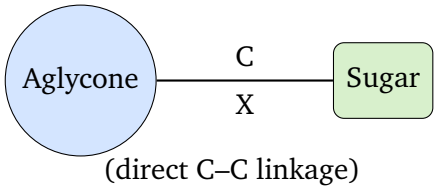
(A) Dragendorff's test (orange-brown)

(B) Wagner's test (reddish-brown)

(C) Mayer's test

(D) Hager's test (yellow)

Q130. The schematic below shows a glycoside in which the sugar is attached directly to a carbon atom of the aglycone instead of through an oxygen bridge.



(direct C–C linkage)

A glycoside in which the sugar is joined to the aglycone by such a direct carbon-to-carbon bond (as in aloin or barbaloin) is classified as a:

(A) O-glycoside

(B) N-glycoside

(C) S-glycoside (thioglycoside)

(D) C-glycoside

Q131. Quillaia bark is a source of triterpenoid saponin glycosides that lower surface tension and are used as foaming and emulsifying agents. The official botanical source of quillaia is:

(A) *Quillaja saponaria*

(B) *Glycyrrhiza glabra*

(C) *Dioscorea deltoidea*

(D) *Centella asiatica*

Q132. Wild cherry bark contains a cyanogenetic glycoside that, on enzymatic hydrolysis by emulsin, liberates benzaldehyde, glucose and hydrocyanic acid, giving it a mild sedative-antitussive action. This cyanogenetic glycoside is:

(A) Sinigrin

(B) Prunasin/amygdalin

(C) Sennoside

(D) Glycyrrhizin

Q133. The dried rinds of unripe *Citrus* fruits are rich in a flavanone-7-rutinoside that is used as a vitamin-P-active capillary protectant. This flavonoid glycoside is:

(A) Diosgenin

(B) Digitoxin

(C) Hesperidin

(D) Sennoside A

Q134. Cardamom, the dried ripe fruit of *Elettaria cardamomum*, yields an aromatic carminative volatile oil whose chief odour-bearing constituents are:

(A) Eugenol and acetyleugenol

(B) Menthol and menthone

(C) Citral and geraniol

(D) 1,8-cineole and α-terpinyl acetate

Q135. Ajowan fruit (*Trachyspermum ammi*) yields a volatile oil from which a crystalline phenol with antiseptic and anthelmintic properties is obtained on cooling and alkali treatment. This phenol, also called "ajwain-ka-phool", is:

(A) Thymol

(B) Eugenol

(C) Anethole

(D) Safrole

Q136. The yellow colouring matter and chief anti-inflammatory principle of turmeric rhizome (*Curcuma longa*) is a diarylheptanoid pigment. This constituent is:

(A) Gingerol

(B) Curcumin

(C) Carvone

(D) Shogaol

Q137. Balsam of Tolu, a pathological balsamic exudate used as an expectorant and flavouring agent, is obtained from the trunk of:

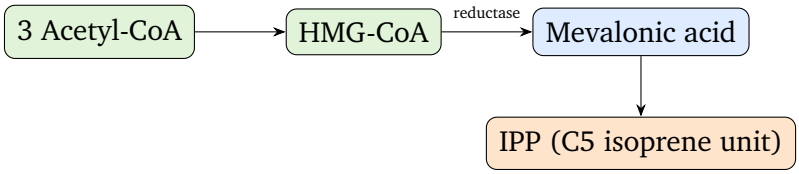
(A) *Liquidambar orientalis*

(B) *Commiphora myrrha*

(C) *Myroxylon balsamum*

(D) *Pinus palustris*

Q138. The biosynthetic scheme below outlines the formation of a key five-carbon precursor of plant terpenoids.



The rate-limiting step converting HMG-CoA to mevalonic acid is catalysed by which enzyme, and what is the immediate product class generated from IPP?

(A) Chorismate synthase; aromatic amino acids

(B) Phenylalanine ammonia lyase; phenylpropanoids

(C) Polyketide synthase; fatty acids

(D) HMG-CoA reductase; terpenoids (via IPP/DMAPP)

Q139. In the phenylpropanoid pathway, the aromatic amino acid phenylalanine (formed via shikimic acid) is deaminated to trans-cinnamic acid as the committed first step toward lignans, coumarins and flavonoids. The enzyme catalysing this deamination is:

(A) Phenylalanine ammonia lyase (PAL)

(B) HMG-CoA reductase

(C) Squalene epoxidase

(D) Tryptophan decarboxylase

Q140. Isabgol (ispaghula) husk is evaluated by a parameter that measures the volume in millilitres occupied by 1 g of the drug, including any adhering mucilage, after it has hydrated and swollen in water for four hours in a graduated cylinder. This parameter is the:

(A) Foaming index

(B) Swelling index

(C) Acid value

(D) Crude fibre content

Q141. In the foaming-index test for saponin-containing drugs, a decoction is serially diluted and the foaming index is taken as 1000 divided by the volume (in mL) of the lowest dilution still producing a 1 cm froth that persists. If a persistent 1 cm froth is just obtained in the tube containing **8 mL** of decoction made up to 10 mL, the foaming index is closest to:

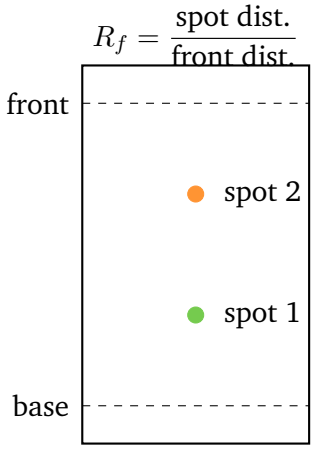
(A) 80

(B) 8

(C) 125

(D) 1000

Q142. Two students standardise a leaf drug. Student 1 reports the *crude fibre* as the residue left after successive boiling with dilute acid and dilute alkali, washing and ignition. Student 2 runs the TLC plate shown to locate marker constituents.



The solvent front travels 4.0 cm from the baseline; spot 1 travels 1.2 cm and spot 2 travels 2.8 cm. Which statement is correct?

(A) Crude fibre is the alcohol-soluble extractive; spot 2 has $R_f = 0.12$

(B) Crude fibre is the total ash; both spots have the same R_f

(C) Crude fibre dissolves fully in acid and alkali; spot 1 has $R_f = 2.8$

(D) Crude fibre is the acid-and-alkali-insoluble organic residue; spot 1 has $R_f = 0.30$ and spot 2 has $R_f = 0.70$

Part F: Pharmaceutical Biotechnology & Microbiology

Q143. The enzymes *Msp*I and *Hpa*II both recognise the same sequence CCGG but differ in their response to cytosine methylation of the internal C. A pair of enzymes that recognise the **same** sequence yet may cut differently (here because of methylation) are termed:

(A) Homing endonucleases

(B) Exonucleases acting from the ends

(C) Nicking enzymes that cut one strand only

(D) Isoschizomers (here a methylation-sensitive isoschizomer pair)

Q144. Insulin glargine is a recombinant long-acting insulin analogue made by site-directed mutagenesis of the human insulin gene (glycine substituted at A21 and two arginines added to the B-chain). The change that gives glargine its prolonged action is that the modified protein:

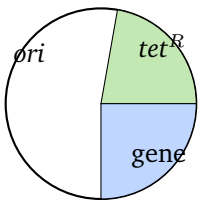
(A) Is soluble at the acidic pH of the formulation but precipitates as microcrystals at physiological pH, dissolving slowly

(B) Binds covalently to serum albumin through a fatty-acid chain

(C) Is digested rapidly by plasma proteases to a faster peak

(D) Cannot bind the insulin receptor and acts as an antagonist

Q145. The recombinant plasmid shown below (an *ori*, a tetracycline-resistance marker, and a cloned gene) is to be introduced into *E. coli*. A brief high-voltage electrical pulse is applied to a suspension of cells and this DNA, transiently making the membrane permeable so the plasmid enters. This physical method of transformation is called:



(A) Transduction by a bacteriophage

(B) Electroporation

(C) Conjugation through a sex pilus

(D) Heat-shock of calcium-chloride-treated cells

Q146. Recombinant *L*-asparaginase is used in the treatment of acute lymphoblastic leukaemia. Its selective anticancer action arises because it:

(A) Cross-links the two strands of tumour-cell DNA

(B) Inhibits dihydrofolate reductase in dividing cells

(C) Hydrolyses circulating asparagine, starving leukaemic cells that cannot synthesise their own

(D) Blocks microtubule assembly during mitosis

Q147. A therapeutic monoclonal antibody requires correct human-type N-linked glycosylation for full effector function and a long serum half-life. For such a product the preferred expression host among the following is a:

(A) Wild-type *E. coli*, because it glycosylates proteins like humans

(B) *Bacillus subtilis* grown anaerobically

(C) Plant chloroplast, because it lacks any glycosylation machinery

(D) Mammalian cell line such as Chinese-hamster-ovary (CHO) cells

Q148. In an antibiotic-production programme, the first step of **primary screening** of soil isolates is carried out to:

(A) Detect, from a large number of isolates, those few that show any antimicrobial activity worth pursuing

(B) Determine the exact chemical structure of the antibiotic

(C) Scale the culture up to a 50,000-litre production fermenter

(D) Carry out clinical trials of the purified drug

Q149. Citric acid is manufactured on the largest scale of any organic acid by submerged fermentation. The microorganism overwhelmingly used in the modern process, grown under iron- and manganese-limited conditions, is:

(A) *Lactobacillus delbrueckii*

(B) *Aspergillus niger*

(C) *Acetobacter aceti*

(D) *Clostridium acetobutylicum*

Q150. Vitamin B_{12} (cyanocobalamin) for pharmaceutical use is produced by fermentation rather than chemical synthesis because of its structural complexity. It is obtained industrially from:

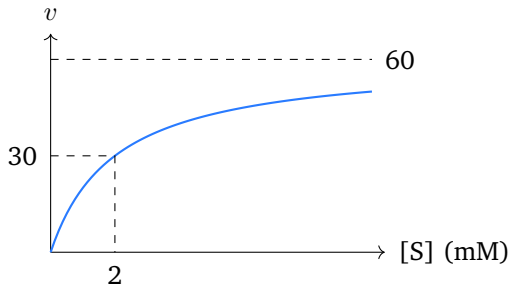
(A) A green plant grown in tissue culture

(B) Pressed yeast extract as a primary metabolite

(C) Bacteria such as *Pseudomonas denitrificans* or *Propionibacterium* species

(D) Chemical reduction of cobalt salts only

Q151. The Michaelis–Menten plot below shows initial velocity v against substrate concentration [S] for an enzyme with V_{max} = 60 μmol min^{-1}. From the half-maximal point marked, the Michaelis constant K_m of this enzyme is:



(A) 2 mM (the [S] giving half of V_{max}, i.e. 30 μmol min^{-1})

(B) 60 mM, equal to V_{max}

(C) 30 mM, half of V_{max}

(D) 4 mM, twice the half-velocity point

Q152. In a branched biosynthetic pathway, the final product binds to a regulatory site (distinct from the active site) on the first committed enzyme and switches it off when product is abundant. This control of the pathway is best described as:

(A) Competitive inhibition at the active site

(B) Allosteric feedback (end-product) inhibition

(C) Irreversible covalent modification by the substrate

(D) Induction of new enzyme synthesis

Q153. The complement system can be activated by three pathways that converge on C3. The **classical** pathway is initiated specifically by:

(A) Spontaneous hydrolysis of C3 in plasma

(B) Mannose-binding lectin attaching to sugars on a microbe

(C) Bacterial lipopolysaccharide directly cleaving C5

(D) C1q binding to antigen–antibody (IgG or IgM) complexes

Q154. Etanercept, used in rheumatoid arthritis, is a recombinant fusion protein in which the soluble TNF-receptor (p75) is joined to the Fc portion of human IgG1. It improves on the bare receptor mainly because the Fc fusion:

(A) Converts the receptor into an agonist that stimulates TNF release

(B) Allows the molecule to cross the blood–brain barrier freely

(C) Forms a dimer and greatly prolongs serum half-life, acting as a decoy that mops up TNF-α

(D) Makes the protein expressible only in *E. coli*

Q155. Many killed and subunit vaccines include an aluminium salt (alum) in the formulation. The chief purpose of this adjuvant is to:

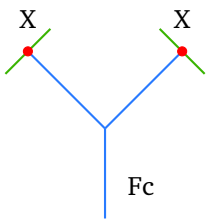
(A) Enhance and prolong the immune response to the antigen, allowing a smaller antigen dose

(B) Act as the antigen itself

(C) Kill any residual live organisms in the vial

(D) Serve only as an isotonicity agent with no immune effect

Q156. Rituximab is a chimeric monoclonal antibody used in B-cell lymphomas. The Y-shaped antibody is drawn below; its two antigen-binding arms (region marked X) recognise a single defined epitope on the surface marker CD20 of B-lymphocytes. Because it binds one and only one epitope, rituximab is described as:



(A) Polyclonal, recognising many epitopes at once

(B) Monoclonal, recognising a single epitope (CD20)

(C) An antitoxin neutralising a bacterial toxin

(D) A complement protein of the lectin pathway

Q157. Bacterial endospores are the most heat- and chemical-resistant living structures and dictate sterilisation requirements. Among the following, the genera that form true heat-resistant endospores are:

(A) *Escherichia* and *Salmonella*

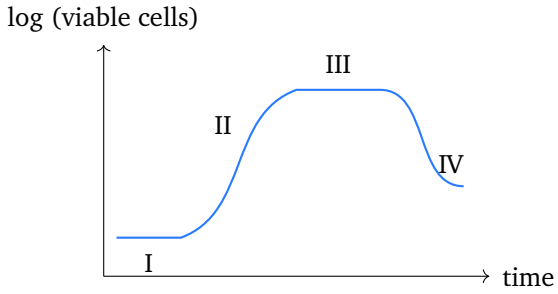
(B) *Staphylococcus* and *Streptococcus*

(C) *Pseudomonas* and *Vibrio*

(D) *Bacillus* and *Clostridium*

Q158. The batch growth curve below plots log(viable cells) against time for a culture in a closed flask, showing four phases I–IV. The phase in which

viable counts fall logarithmically because nutrients are exhausted and toxic products accumulate, so the death rate exceeds any division, is:



(A) Phase IV (death / decline phase)

(B) Phase I (lag phase)

(C) Phase II (log / exponential phase)

(D) Phase III (stationary phase)

Q159. In moist-heat sterilisation validation, the $\mathbf{F}_0$ value of a cycle is defined as the:

(A) Time in hours at 160°C needed for dry-heat sterilisation

(B) Number of viable spores surviving on the biological indicator

(C) Equivalent exposure time in minutes at 121°C delivering the same lethality (with $z = 10$°C)

(D) Filter pore size in micrometres required to remove bacteria

Q160. Pharmacopoeial Water for Injection (WFI) must meet a microbial limit (action level) of not more than **10 colony-forming units (CFU) per 100 mL**. The routine microbiological test used to establish this microbial **bioburden** of the water is:

(A) The phenol-coefficient assay against *Salmonella*

(B) Membrane filtration of a measured volume followed by plate count of total viable aerobic organisms

(C) The Gram stain of a single drop under the microscope

(D) Measurement of conductivity alone, with no incubation

Detailed Solutions

Q1.

Solution

Concept — Solubility product of an A_2B salt: For $Ag_2CrO_4 \rightleftharpoons 2Ag^+ + CrO_4^{2-}$, $K_{sp} = [Ag^+]^2[CrO_4^{2-}] = (2s)^2(s) = 4s^3$. **Reasoning:** $s = 1.0 \times 10^{-4}$, so $K_{sp} = 4(1.0 \times 10^{-4})^3 = 4 \times 10^{-12}$. **Why the other options are wrong:**

- (A) 1.0×10^{-8} wrongly uses s^2 (a 1:1 salt formula).
- (B) 1.0×10^{-12} uses s^3 but drops the factor 4.
- (C) 2.0×10^{-12} uses a factor 2 instead of 4.

Final Answer: $K_{sp} = 4s^3 = 4 \times 10^{-12} \Rightarrow$ D

Answer: (D) Go Back to Q1

Q2.

Solution

Concept — Single-extraction fraction: With equal volumes the fraction extracted into the organic phase is $\frac{P}{P+1}$. **Reasoning:** $\frac{P}{P+1} = \frac{4}{4+1} = \frac{4}{5}$, i.e. 80% goes into the amyl acetate. **Why the other options are wrong:**

- (A) $1/5$ is the fraction *remaining* in water, not extracted.
- (B) $1/4$ inverts the ratio.
- (D) $1/2$ ignores the value of P.

Final Answer: fraction extracted $= P/(P+1) = 4/5 \Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept — HLB of a blend: The HLB of a surfactant mixture is the weight-fraction average of the component HLB values. **Reasoning:** HLB $= 0.25(8.6) + 0.75(16.7) = 2.15 + 12.525 = 14.675 \approx 14.68$. **Why the other options are wrong:**

- (A) 12.65 uses the wrong weighting.
- (C) 16.70 is the Tween 20 value alone.

- (D) 8.60 is the Span 20 value alone.

Final Answer: $0.25(8.6) + 0.75(16.7) = 14.68 \Rightarrow \boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept — Dilatant flow: In shear-thickening systems the apparent viscosity *increases* with shear rate, so the flow curve bows toward the shear-rate ($\dot{\gamma}$) axis. **Reasoning:** Concentrated deflocculated suspensions of small particles (e.g. high-solids starch/inorganic slurries) dilate and stiffen under high shear; this is dilatant (shear-thickening) flow. **Why the other options are wrong:**

- (A) Newtonian gives a straight line through the origin.
- (B) Pseudoplastic curves bow toward the stress axis (viscosity falls).
- (D) Bingham plastic shows a yield-stress intercept on the stress axis.

Final Answer: curve bowing toward $\dot{\gamma}$ axis $\Rightarrow$ dilatant $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept — Kinematic viscosity: $\nu = \eta/\rho$; in CGS, poise $\div$ (g/cm^3) gives stokes (cm^2/s). **Reasoning:** $\nu = 2.0 \,/\, 0.80 = 2.5$ stokes. **Why the other options are wrong:**

- (A) 1.6 multiplies instead of dividing by density.
- (B) 0.40 inverts the calculation.
- (C) 1.0 ignores the density value.

Final Answer: $\nu = 2.0/0.80 = 2.5$ stokes $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q5

Q6.

Solution

Concept — Capillary rise: $h = \dfrac{2\gamma\cos\theta}{\rho g r}$, so for a given liquid the rise is inversely proportional to the capillary radius r. **Reasoning:** A narrower capillary (smaller r) gives a higher rise; this is the basis of the capillary-rise method for surface tension. **Why the other options are wrong:**

- (B) Rise falls (not rises) with increasing radius.
- (C) Rise is directly proportional to surface tension γ.
- (D) The dependence on radius is $1/r$, not $1/r^2$.

Final Answer: $h \propto 1/r \Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q6

Q7.

Solution

Concept — Carr's index: Carr's % $= \dfrac{\rho_{tapped} - \rho_{bulk}}{\rho_{tapped}} \times 100$. **Reasoning:** $= \dfrac{0.54 - 0.45}{0.54} \times 100 = \dfrac{0.09}{0.54} \times 100 \approx 16.7 \approx 17\%$, which corresponds to good (fair) flow. **Why the other options are wrong:**

- (A) 9% would need a much smaller density difference (excellent flow).
- (C) 25% over-estimates the difference.
- (D) 40% is far too high for these densities.

Final Answer: $(0.09/0.54) \times 100 \approx 17\%$ (good) $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept — Hausner ratio: HR $= \rho_{tapped}/\rho_{bulk}$. **Reasoning:** HR $= 0.54/0.45 = 1.20$, consistent with the $\approx 17\%$ Carr's index (good-to-fair flow). **Why the other options are wrong:**

- (A) 0.83 is the inverted ratio; HR is always ≥ 1.
- (B) 1.09 under-estimates the ratio.

- (D) 1.45 would indicate very poor (cohesive) flow, contradicting the data.

Final Answer: HR $= 0.54/0.45 = 1.20 \Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept — Modal size class: The mode is the size interval whose bar is tallest. **Reasoning:** The tallest bar (height 3.1) is the fourth one, spanning the 300 class, i.e. the 300–425 μm interval. **Why the other options are wrong:**

- (A) 75–150 is a short low-size bar.
- (B) 150–212 is taller but still below the peak.
- (C) 212–300 is the second-tallest, not the peak.

Final Answer: tallest bar lies in 300–425 μm $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept — Henderson-Hasselbalch: $\text{pH} = \text{pK}_a + \log \dfrac{[\text{salt}]}{[\text{acid}]}$. **Reasoning:** $\dfrac{[\text{salt}]}{[\text{acid}]} = \dfrac{0.50}{0.05} = 10$, so $\text{pH} = 10.3 + \log 10 = 10.3 + 1 = 11.3$. **Why the other options are wrong:**

- (B) 10.3 ignores the $10:1$ salt-to-acid ratio.
- (C) 9.3 wrongly subtracts the log term.
- (D) 5.0 is unrelated to the buffer composition.

Final Answer: $\text{pH} = 10.3 + 1 = 11.3 \Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept — Isotonicity by freezing-point depression: A solution is isotonic with plasma when it depresses the freezing point by 0.52°C. **Reasoning:** If 1% gives 0.13°C, the concentration needed is $\dfrac{0.52}{0.13} \times 1\% = 4.0\%$. **Why the other options are wrong:**

- (A) 0.52% confuses the depression value with concentration.
- (C) 0.13% is the depression of the 1% solution, not the target concentration.
- (D) 2.0% uses an incorrect ratio.

Final Answer: $(0.52/0.13) \times 1\% = 4.0\% \Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q11

Q12.

Solution

Concept — Zero-order kinetics: For zero order $C = C_0 - k_0 t$; a plot of C versus t is a straight line of slope $-k_0$. **Reasoning:** A linear concentration-vs-time decline (not ln C, not $1/C$) is the signature of zero-order loss, typical of degradation in suspensions where dissolved drug stays at saturation. **Why the other options are wrong:**

- (A) First-order gives a linear $\ln C$ vs t plot.
- (B) Second-order gives a linear $1/C$ vs t plot.
- (D) Half-order is not indicated by a straight C vs t line.

Final Answer: linear C vs $t \Rightarrow$ zero-order $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q12

Q13.

Solution

Concept — First-order shelf life: $t_{90} = \dfrac{0.105}{k}$ for 10% loss. **Reasoning:** $t_{90} = \dfrac{0.105}{3.5 \times 10^{-3}} = \dfrac{0.105}{0.0035} = 30$ months. **Why the other options are wrong:**

- (A) 3 months misplaces a decimal in k.
- (B) 12 months uses the wrong numerator.

- (C) 198 months would come from the half-life constant 0.693 instead of 0.105.

Final Answer: $t_{90} = 0.105/0.0035 = 30$ months $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept — Q_{10} temperature coefficient: Q_{10} is the factor by which a reaction rate changes for a 10°C rise; for most degradation reactions it lies between about 2 and 4. **Reasoning:** This range underpins accelerated stability testing, where elevated-temperature data are extrapolated to predict room-temperature shelf life via the Arrhenius equation. **Why the other options are wrong:**

- (B) 0.1–0.2 would mean the rate *falls* on heating, which is unphysical for degradation.
- (C) A Q_{10} of exactly 1 means no temperature dependence.
- (D) 50–100 is unrealistically large.

Final Answer: typical $Q_{10} \approx 2$–$4 \Rightarrow$ A

Answer: (A) Go Back to Q14

Q15.

Solution

Concept — Langmuir linearisation: Rearranging $\frac{x}{m} = \frac{y_m bC}{1+bC}$ gives $\frac{C}{(x/m)} = \frac{1}{y_m b} + \frac{C}{y_m}$. **Reasoning:** A plot of $C/(x/m)$ versus C is therefore linear, with slope $1/y_m$ and intercept $1/(y_m b)$. **Why the other options are wrong:**

- (A) $\log(x/m)$ vs $\log C$ is the Freundlich linearisation.
- (C) x/m vs C is curved for Langmuir.
- (D) x/m vs $1/C$ does not linearise the Langmuir form.

Final Answer: $C/(x/m)$ vs C is linear $\Rightarrow$ B

Answer: (B) Go Back to Q15

Q16.

Solution

Concept — Stokes-Einstein: $D = \dfrac{kT}{6\pi\eta r}$, so $D \propto 1/r$ at fixed T and η. **Reasoning:** Doubling the radius halves the diffusion coefficient ($D \to D/2$); larger molecules diffuse more slowly. **Why the other options are wrong:**

- (A) Doubling r does not double D; the relationship is inverse.
- (B) D does not scale with r^2; it scales with $1/r$.
- (D) D depends explicitly on r, so it cannot stay unchanged.

Final Answer: $D \propto 1/r$, so D halves $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q16

Q17.

Solution

Concept — Phase-solubility (A_L) diagram: A linear rise of total drug solubility with ligand concentration, with slope < 1, denotes a soluble first-order (commonly $1:1$) complex. **Reasoning:** The increasing line (A_L type) shows the complex stays in solution, raising apparent solubility; the < 1 slope is consistent with $1:1$ stoichiometry. **Why the other options are wrong:**

- (B) An insoluble complex would give a B-type curve with a plateau/decline.
- (C) Complexation is a reversible association, not a covalent bond.
- (D) A eutectic is a solid-solid phenomenon, unrelated to phase-solubility plots.

Final Answer: rising A_L line $\Rightarrow$ soluble $1:1$ complex $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q17

Q18.

Solution

Concept — Noyes-Whitney: $dC/dt = \dfrac{DA}{h}(C_s - C)$; dissolution rate rises with the effective surface area A. **Reasoning:** Micronizing from 50 to 5 μm hugely increases A per unit mass, so the dissolution rate climbs (a standard route to improve poorly soluble drugs). **Why the other options are wrong:**

- (A) Particle size has little effect on the intrinsic C_s (except for sub-micron Ostwald effects).
- (B) Thickening h would slow, not speed, dissolution.
- (C) D is a property of drug and medium, not changed by milling.

Final Answer: smaller particles $\Rightarrow$ larger A $\Rightarrow$ faster dissolution $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q18

Q19.

Solution

Concept — Higuchi model: $Q = k_H\sqrt{t}$; matrix release is diffusion-controlled, so cumulative amount is linear with $\sqrt{t}$. **Reasoning:** A straight Q vs $\sqrt{t}$ line through the origin (slope k_H) is the signature of the Higuchi (matrix-diffusion) model. **Why the other options are wrong:**

- (A) Zero-order plots Q linearly vs t (not $\sqrt{t}$).
- (C) First-order plots $\log(\%$ remaining$)$ vs t.
- (D) Hixson-Crowell plots the cube root of remaining mass vs t.

Final Answer: Q vs $\sqrt{t}$ linear $\Rightarrow$ Higuchi model $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q19

Q20.

Solution

Concept — Dissolution profile shape: An immediate-release product dissolves fast and plateaus; a sustained-release product releases slowly and steadily over a prolonged period. **Reasoning:** Profile (ii) shows slow, near-linear, prolonged release, the intended behaviour of a sustained-/controlled-release dosage form. **Why the other options are wrong:**

- (A) An orodispersible tablet would release even faster than (i).
- (B) An effervescent tablet also dissolves rapidly.
- (D) An IV bolus bypasses dissolution entirely.

Final Answer: slow prolonged release $\Rightarrow$ sustained-release form $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q20

Q21.

Solution

Concept — First-pass metabolism: Orally absorbed drug passes through the liver (via the portal vein) before reaching systemic circulation, where it may be substantially metabolised. **Reasoning:** This pre-systemic loss lowers the fraction reaching the blood, so oral bioavailability (AUC) is reduced relative to the IV route. **Why the other options are wrong:**

- (B) Some drug usually survives; absorption is reduced, not abolished.
- (C) Oral C_{max} is lower (and later), not higher, than IV.
- (D) Extensive first-pass metabolism precludes 100% bioavailability.

Final Answer: first-pass metabolism lowers oral bioavailability $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q21

Q22.

Solution

Concept — USP dissolution apparatus 1: Apparatus 1 is the rotating basket, which holds the dosage form inside a cylindrical wire-mesh basket attached to a rotating shaft. **Reasoning:** The basket physically retains floating or disintegrating capsules in the medium, making it suitable where such units would otherwise float free of a paddle. **Why the other options are wrong:**

- (A) The paddle is Apparatus 2.
- (B) The flow-through cell is Apparatus 4.
- (C) The reciprocating cylinder is Apparatus 3.

Final Answer: Apparatus 1 is the rotating basket $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept — Tablet binders: Binders impart cohesiveness so granules and tablets hold together; povidone (PVP K-30) is a common solution/dry binder. **Reasoning:** Dissolved in the granulating fluid, PVP forms bridges between particles, producing strong granules and compacts. **Why the other options are wrong:**

- (A) Glidants (e.g. colloidal silica) improve flow, not cohesion.
- (C) Lubricants (e.g. Mg stearate) reduce die-wall friction.
- (D) Disintegrants promote break-up, the opposite of binding.

Final Answer: povidone acts as a binder $\Rightarrow$ B

Answer: (B) Go Back to Q23

Q24.

Solution

Concept — Tablet hardness: Crushing strength is the force needed to fracture a tablet diametrically, measured by Monsanto/Pfizer/Strong-Cobb type testers. **Reasoning:** It is reported in kilograms-force (kg) or, in SI, in newtons (N). **Why the other options are wrong:**

- (A) Percent weight loss is the friability measure.
- (B) Minutes to disintegrate is the disintegration-test result.
- (D) g/mL is a density unit, not hardness.

Final Answer: hardness is expressed in kg-force or N $\Rightarrow$ C

Answer: (C) Go Back to Q24

Q25.

Solution

Concept — Superdisintegrants: These rupture tablets rapidly at low levels; crospovidone is a synthetic crosslinked povidone that works mainly by fast capillary wicking (water uptake) with little swelling. **Reasoning:** Its porous, non-swelling particles draw water in by capillarity, breaking inter-particle bonds and disintegrating the tablet quickly. **Why the other options are wrong:**

- (B) Liquid paraffin is an oily vehicle/lubricant.
- (C) Magnesium stearate is a hydrophobic lubricant.
- (D) Sucrose is a sweetener/diluent.

Final Answer: crospovidone is a wicking superdisintegrant $\Rightarrow$ A

Answer: (A) Go Back to Q25

Q26.

Solution

Concept — Water for Injection: WFI must be sterile and pyrogen-free; pyrogens (endotoxins) are non-volatile, so they do not carry over in vapour. **Reasoning:** Distillation (or reverse osmosis meeting the WFI standard) leaves pyrogens behind in the residue, yielding pyrogen-free water. **Why the other options are wrong:**

- (A) Boiling kills some microbes but does not remove endotoxins.
- (B) Charcoal alone does not guarantee a pyrogen-free, injectable-grade water.
- (C) Silica gel is a desiccant, not a water-purification step.

Final Answer: WFI is produced by distillation/RO, removing pyrogens $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q26

Q27.

Solution

Concept — Displacement value (DV): Mass of base required = (mould capacity) − (mass of drug ÷ DV). **Reasoning:** Drug volume in base terms $= 0.45/1.5 = 0.30$ g; base required $= 2.0 - 0.30 = 1.70$ g per suppository. **Why the other options are wrong:**

- (A) 2.0 g ignores the space taken by the drug.
- (C) 1.55 g comes from wrongly subtracting the full 0.45 g.
- (D) 0.45 g is the drug mass, not the base.

Final Answer: base $= 2.0 - (0.45/1.5) = 1.70$ g $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q27

Q28.

Solution

Concept — Creaming: Creaming is the reversible gravity-driven migration of dispersed droplets (up or down per density) to form a concentrated layer, without coalescence. **Reasoning:** Because the interfacial film is intact, gentle shaking redisperses the droplets, distinguishing creaming from irreversible cracking. **Why the other options are wrong:**

- (A) Cracking is irreversible coalescence into two phases.
- (B) Phase inversion is a change of emulsion type (o/w $\leftrightarrow$ w/o).
- (D) Ostwald ripening is droplet growth by molecular diffusion, not the described upward layering.

Final Answer: reversible droplet layering = creaming $\Rightarrow$ C

Answer: (C) Go Back to Q28

Q29.

Solution

Concept — Sedimentation volume (F): $F = V_u/V_0$, the ratio of the ultimate (equilibrium) sediment volume to the original total volume of the suspension. **Reasoning:** F ranges from near 0 (deflocculated, hard cake) toward 1 (well-flocculated, loose sediment); a high F is desirable. **Why the other options are wrong:**

- (B) Drug mass per volume is concentration, not F.
- (C) F uses original volume, not supernatant/sediment ratio.
- (D) Container geometry is irrelevant to F.

Final Answer: $F = V_u/V_0$ (sediment $\div$ original volume) $\Rightarrow$ A

Answer: (A) Go Back to Q29

Q30.

Solution

Concept — Ointment bases: Water-soluble bases are made wholly of water-soluble components, typically polyethylene glycols, and contain no oils or water. **Reasoning:** A PEG 400 + PEG 4000 blend is anhydrous yet fully water-washable and water-soluble, the prototype water-soluble base. **Why the other options are wrong:**

- (A) Oleaginous bases are hydrocarbons (e.g. petrolatum).
- (C) Absorption bases (e.g. wool fat) take up water to form w/o systems.
- (D) A w/o emulsion base contains an aqueous internal phase, unlike PEG bases.

Final Answer: PEG blend is a water-soluble base $\Rightarrow$ B

Answer: (B) Go Back to Q30

Q31.

Solution

Concept — Hammer mill: A hammer mill carries swinging hammers on a rotor that strike the feed at high speed, shattering particles by impact. **Reasoning:** Material is broken by repeated high-velocity impact until small enough to pass the screen at the discharge; it is versatile for moderate fineness. **Why the other options are wrong:**

- (A) Pure attrition between plates describes a colloid/attrition mill.
- (B) Compression between rollers describes a roller mill.
- (C) Cutting with knives describes a cutter mill.

Final Answer: hammer mill works by high-speed impact $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q31

Q32.

Solution

Concept — Reynolds number: $\text{Re} = \dfrac{\rho v d}{\eta}$ classifies pipe-flow regime: < 2100 laminar, 2100–4000 transitional, > 4000 turbulent. **Reasoning:** The chaotic, swirling lower sketch is turbulent flow, which sets in once Re exceeds about 4000. **Why the other options are wrong:**

- (B) $\text{Re} = 2100$ marks the upper end of laminar/start of transition, not full turbulence.
- (C) $\text{Re} = 100$ is firmly laminar.
- (D) $\text{Re} = 1.0$ is creeping (highly laminar) flow.

Final Answer: turbulent flow $\Rightarrow \text{Re} > 4000 \Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q32

Q33.

Solution

Concept — Eutectic point: In a binary system miscible as liquid but immiscible as solid, the two liquidus curves meet at the lowest melting temperature. **Reasoning:**

That intersection (E) is the eutectic point, where a liquid of fixed composition freezes to a fine mixture of both solids at a single temperature. **Why the other options are wrong:**

- (B) The triple point is for one pure substance (solid-liquid-vapour).
- (C) The critical solution temperature applies to partially miscible *liquids*.
- (D) The glass transition is a property of amorphous solids, not a phase-diagram intersection.

Final Answer: lowest-melting intersection = eutectic point $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q33

Q34.

Solution

Concept — Reservoir transdermal system: In a reservoir (membrane-controlled) patch, drug is held in a liquid/gel reservoir and must cross a rate-controlling polymeric membrane before reaching the skin. **Reasoning:** When this membrane is the slowest step, it dictates a constant (zero-order) input rate, the hallmark of reservoir-type transdermal delivery. **Why the other options are wrong:**

- (A) Adhesive strength governs attachment, not release rate.
- (B) Backing colour is irrelevant to delivery rate.
- (D) Body temperature has only a minor, secondary effect on the controlled rate.

Final Answer: a rate-controlling membrane governs delivery $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q34

Q35.

Solution

Concept — Volume of distribution: V_d links the amount of drug in the body to the plasma concentration, $V_d = \text{Dose}/C_0$. **Reasoning:** For an IV bolus in a one-compartment model, C_0 is the concentration extrapolated to $t = 0$. Here $V_d = 600\,\text{mg}/15\,\text{mg/L} = 40$ L. **Why the other options are wrong:**

- (A) 9 L wrongly uses the product divided in error.
- (B) 0.025 L inverts the ratio (C_0/Dose).
- (D) 90 L uses 6.67 mg/L, not the given 15 mg/L.

Final Answer: $V_d = 600/15 = 40$ L $\Rightarrow$ C

Answer: (C) Go Back to Q35

Q36.

Solution

Concept — First-order half-life: The slope of $\ln C$ vs t equals $-k_e$, and $t_{1/2} = 0.693/k_e$. **Reasoning:** The magnitude of the slope is the elimination rate constant, so $k_e = 0.231\,\text{h}^{-1}$. Then $t_{1/2} = 0.693/0.231 = 3$ h. **Why the other options are wrong:**

- (B) 0.231 h confuses k_e with the half-life.
- (C) 6.93 h corresponds to $k_e = 0.10\,\text{h}^{-1}$, not 0.231.
- (D) 23.1 h is ten times too large.

Final Answer: $t_{1/2} = 0.693/0.231 = 3$ h $\Rightarrow$ A

Answer: (A) Go Back to Q36

Q37.

Solution

Concept — Maintenance dosing rate: At steady state the infusion rate equals the rate of elimination, $R_0 = CL \times C_{ss}$. **Reasoning:** $R_0 = 8\,\text{L/h} \times 5\,\text{mg/L} = 40$ mg/h. Clearance and target concentration alone set the maintenance rate; V_d governs the loading dose, not the infusion rate. **Why the other options are wrong:**

- (A) 1.6 mg/h divides instead of multiplying.
- (C) 0.625 mg/h inverts the relationship.

- (D) 13 mg/h has no basis.

Final Answer: $R_0 = CL \times C_{ss} = 40$ mg/h $\Rightarrow$ B

Answer: (B) Go Back to Q37

Q38.

Solution

Concept — Protein binding and free drug: Only the free (unbound) fraction is pharmacologically active and available for distribution, metabolism and excretion. **Reasoning:** Going from 99% to 98% bound means the free fraction rises from 1% to 2%, roughly doubling the active concentration. This transiently increases effect, but the larger free pool is also more rapidly cleared, so total concentration usually falls back toward a new equilibrium. **Why the other options are wrong:**

- (A) Total concentration tends to fall, not double, as free drug is cleared.
- (B) Clearance of free drug increases, it does not fall.
- (C) The drug remains active; indeed its free active form rises.

Final Answer: Free fraction roughly doubles (1% $\rightarrow$ 2%) $\Rightarrow$ D

Answer: (D) Go Back to Q38

Q39.

Solution

Concept — Partial agonist: A partial agonist has affinity but only sub-maximal intrinsic activity, so its plateau lies below that of a full agonist even at full occupancy. **Reasoning:** Curve H reaches a lower maximum than curve G regardless of dose, the signature of a partial agonist (lower efficacy). G, reaching the full plateau, is the full agonist. **Why the other options are wrong:**

- (B) H is less, not more, efficacious.
- (C) Their maxima differ, so efficacy is not identical.
- (D) A pure antagonist would produce no response of its own; H still produces a sub-maximal effect.

Final Answer: H is the partial agonist, G the full agonist $\Rightarrow$ A

Answer: (A) Go Back to Q39

Q40.

Solution

Concept — Competitive vs non-competitive antagonism: A reversible competitive antagonist shifts the curve right in parallel without lowering E_{max} (surmountable); a non-competitive antagonist depresses the maximum (insurmountable). **Reasoning:** Curve J keeps the same plateau but needs more agonist, so J is competitive. Curve K has a reduced maximum that extra agonist cannot overcome, so K is non-competitive (or irreversible). **Why the other options are wrong:**

- (A) Swaps the two interpretations.
- (C) Only J fits competitive behaviour.
- (D) Inverse/partial agonists are not what these antagonist curves show.

Final Answer: J = competitive, K = non-competitive $\Rightarrow$ B

Answer: (B) Go Back to Q40

Q41.

Solution

Concept — Inverse agonist: At a receptor with constitutive activity, ligands span a continuum from full agonist (positive intrinsic activity), through neutral antagonist (zero), to inverse agonist (negative). **Reasoning:** An inverse agonist binds and stabilises the inactive conformation, lowering the basal (constitutive) signalling below the resting level. This requires a receptor with measurable spontaneous activity. **Why the other options are wrong:**

- (A) Producing a full effect describes a full agonist.
- (B) No effect on basal activity describes a neutral antagonist.
- (D) Affinity with no effect at all is a neutral antagonist, not an inverse agonist.

Final Answer: Reduces constitutive activity below basal (negative intrinsic activity) $\Rightarrow$ C

Answer: (C) Go Back to Q41

Q42.

Solution

Concept — Nuclear receptors: Lipid-soluble hormones act on intracellular (cytosolic/nuclear) receptors that bind DNA response elements and change gene transcription, giving slow effects over hours. **Reasoning:** Thyroid hormone (and steroids such as testosterone, cortisol or oestrogen) cross the membrane and bind intracellular receptors, altering transcription. This is the nuclear-receptor branch shown. **Why the other options are wrong:**

- (A) The nicotinic receptor is a ligand-gated ion channel (milliseconds).
- (B) The α_1 receptor is a GPCR (seconds).
- (C) The insulin receptor is a receptor tyrosine kinase (minutes).

Final Answer: Thyroid/steroid hormone at the intracellular receptor $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q42

Q43.

Solution

Concept — Affinity: Affinity is how tightly a drug binds its receptor, measured by the equilibrium dissociation constant K_d. **Reasoning:** K_d is the concentration occupying half the receptors; a lower K_d means tighter binding, i.e. higher affinity. Affinity is distinct from efficacy (the ability to produce a response once bound). **Why the other options are wrong:**

- (B) Maximal response is efficacy, not affinity.
- (C) The 50% lethal dose is the LD_{50}, a toxicity measure.
- (D) Metabolic rate is a pharmacokinetic property, unrelated to receptor binding.

Final Answer: Tendency to bind, reflected by K_d $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q43

Q44.

Solution

Concept — Enzyme induction: Inducers increase synthesis of cytochrome-P450 enzymes over days to weeks, accelerating metabolism of co-administered substrates and lowering their levels. **Reasoning:** Rifampicin is a classic potent CYP (especially CYP3A4) inducer; starting it gradually lowers the steady-state concentration of a CYP3A4-metabolised drug over one to two weeks, the time needed for new enzyme to accumulate. **Why the other options are wrong:**

- (A) Ketoconazole is a strong CYP inhibitor, which would raise the level.
- (B) Erythromycin is a CYP3A4 inhibitor.
- (D) Grapefruit juice inhibits intestinal CYP3A4, raising levels.

Final Answer: Rifampicin is the classic enzyme inducer $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q44

Q45.

Solution

Concept — Edrophonium: It is a reversible anticholinesterase with a very short duration of action (a few minutes). **Reasoning:** By briefly inhibiting acetylcholinesterase it raises acetylcholine at the neuromuscular junction. In the Tensilon test a transient improvement in muscle strength confirms myasthenia gravis; its short action makes it diagnostic rather than therapeutic. **Why the other options are wrong:**

- (A) It is reversible and very short-acting, not an irreversible organophosphate.
- (C) It does not block nicotinic receptors; it raises acetylcholine.
- (D) It is not a direct muscarinic agonist.

Final Answer: Very short-acting reversible AChE inhibitor $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q45

Q46.

Solution

Concept — Labetalol: It is a combined α_1- and β-adrenoceptor antagonist. **Reasoning:** α_1 blockade lowers peripheral vascular resistance while β blockade prevents the reflex tachycardia that a pure vasodilator would cause, giving a smooth fall in blood pressure useful in hypertensive emergencies and pregnancy. **Why the other options are wrong:**

- (B) It is not a β_2-agonist.
- (C) It is not a central α_2-agonist.
- (D) It is not a calcium-channel blocker.

Final Answer: Combined α_1- and β-blockade $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q46

Q47.

Solution

Concept — Scopolamine: It is a tertiary-amine antimuscarinic that readily crosses the blood-brain barrier. **Reasoning:** Its central penetration gives strong anti-emetic (anti-motion-sickness) and sedative effects, while peripherally it reduces secretions, making it a useful antisecretory premedicant. This central action distinguishes it from quaternary antimuscarinics. **Why the other options are wrong:**

- (A) It is a tertiary amine that does enter the CNS.
- (B) It blocks muscarinic, not nicotinic, receptors.
- (C) It is an antagonist, not a stimulant, of muscarinic receptors.

Final Answer: Tertiary-amine antimuscarinic with marked central actions $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q47

Q48.

Solution

Concept — Methyldopa: It is a prodrug acting through a central α_2-agonist metabolite. **Reasoning:** Methyldopa is decarboxylated and hydroxylated to α-methylnoradrenaline, which stimulates central α_2-adrenoceptors in the brainstem,

reducing sympathetic outflow and lowering blood pressure. Its long safety record makes it a preferred antihypertensive in pregnancy. **Why the other options are wrong:**

- (A) It does not directly block peripheral α_1-receptors.
- (B) It is not a β-blocker.
- (D) It does not inhibit ACE.

Final Answer: Central α_2-agonist metabolite reducing sympathetic outflow $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q48

Q49.

Solution

Concept — Adrenaline in anaphylaxis: As a non-selective adrenoceptor agonist it acts on α and β receptors simultaneously. **Reasoning:** α_1-mediated vasoconstriction reverses hypotension and mucosal oedema, β_1 stimulation supports the heart, and β_2 stimulation relieves bronchospasm. This combination is why adrenaline, not an antihistamine alone, is first-line in anaphylaxis. **Why the other options are wrong:**

- (B) It is not a selective α_2-agonist.
- (C) It does not block H_1 receptors.
- (D) It is not a β_1-antagonist; it stimulates β_1.

Final Answer: Combined α_1, β_1 and β_2 actions $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q49

Q50.

Solution

Concept — Buspirone: It is a serotonin 5-HT_{1A} partial agonist anxiolytic. **Reasoning:** By partially agonising 5-HT_{1A} receptors it relieves generalised anxiety over one to two weeks, without acting on the GABA-A receptor. This avoids the sedation, dependence and withdrawal of benzodiazepines. **Why the other options are wrong:**

- (A) GABA-A modulation describes benzodiazepines.
- (C) D_2 antagonism describes antipsychotics.

- (D) H_1 antagonism describes sedating antihistamines.

Final Answer: 5-HT_{1A} partial agonist $\Rightarrow$ B

Answer: (B) Go Back to Q50

Q51.

Solution

Concept — Tricyclic antidepressants: They block reuptake of noradrenaline and serotonin but also antagonise several other receptors. **Reasoning:** Amitriptyline inhibits the NA and 5-HT transporters (the antidepressant action) but additionally blocks muscarinic (dry mouth, constipation), H_1 (sedation) and α_1 (postural hypotension) receptors, and its sodium-channel effects underlie cardiotoxicity in overdose. **Why the other options are wrong:**

- (A) Selective SERT inhibition describes the SSRIs.
- (B) Irreversible MAO inhibition describes MAOIs.
- (D) Selective dopamine reuptake inhibition does not describe TCAs.

Final Answer: Mixed NA/5-HT reuptake block plus muscarinic/H_1/α_1 blockade $\Rightarrow$ C

Answer: (C) Go Back to Q51

Q52.

Solution

Concept — Naltrexone: It is a long-acting, orally active competitive opioid-receptor antagonist. **Reasoning:** Once a patient is detoxified, daily oral naltrexone occupies μ-opioid receptors and blocks the euphoria from any opioid taken, discouraging relapse; it also reduces craving in alcohol dependence. Its long duration suits maintenance, unlike short-acting naloxone used acutely for overdose. **Why the other options are wrong:**

- (A) It is an antagonist, not a partial agonist, and is long-acting.
- (B) It is not a κ-agonist analgesic.
- (C) It does not act at NMDA receptors.

Final Answer: Long-acting oral competitive opioid antagonist $\Rightarrow$ D

Answer: (D) Go Back to Q52

Q53.

Solution

Concept — Lamotrigine: It blocks voltage-gated sodium channels and reduces excitatory glutamate release. **Reasoning:** By stabilising the inactivated state of neuronal sodium channels it limits repetitive firing and curbs the release of glutamate, giving broad efficacy in partial and generalised seizures and a mood-stabilising action in bipolar disorder. **Why the other options are wrong:**

- (B) T-type calcium block describes ethosuximide.
- (C) Irreversible GABA-transaminase inhibition describes vigabatrin.
- (D) SV2A binding describes levetiracetam.

Final Answer: Na^+ channel block reducing glutamate release $\Rightarrow$ A

Answer: (A) Go Back to Q53

Q54.

Solution

Concept — Modafinil: It is a wake-promoting agent acting partly through weak dopamine-transporter inhibition. **Reasoning:** By weakly inhibiting dopamine re-uptake it raises synaptic dopamine, and it engages orexinergic and histaminergic wake-promoting pathways. This profile promotes wakefulness in narcolepsy with less euphoria and abuse potential than amphetamines. **Why the other options are wrong:**

- (A) It is not a direct β_2-agonist.
- (C) It is not an MAO inhibitor.
- (D) It does not block GABA-A channels.

Final Answer: Weak dopamine-transporter inhibition with wake-pathway effects $\Rightarrow$ B

Answer: (B) Go Back to Q54

Q55.

Solution

Concept — Donepezil: It is a reversible, centrally acting acetylcholinesterase inhibitor. **Reasoning:** By inhibiting central acetylcholinesterase it raises synaptic acetylcholine in the brain, partially compensating for the cholinergic deficit of Alzheimer's disease and giving modest symptomatic improvement in cognition. **Why the other options are wrong:**

- (A) NMDA antagonism describes memantine.
- (B) Donepezil does not block M_1 receptors; that would worsen cognition.
- (D) It is not a dopamine agonist.

Final Answer: Reversible central AChE inhibitor $\Rightarrow$ C

Answer: (C) Go Back to Q55

Q56.

Solution

Concept — Ivabradine: It selectively inhibits the funny current (I_f) in the sinoatrial node. **Reasoning:** The I_f pacemaker current drives spontaneous diastolic depolarisation in the SA node. Blocking it slows the heart rate purely by reducing pacemaker firing, with no effect on contractility, conduction or blood pressure, which is useful when β-blockers are not tolerated. **Why the other options are wrong:**

- (A) β_1 blockade describes β-blockers.
- (B) L-type calcium block would reduce contractility.
- (C) ACE inhibition is a separate mechanism.

Final Answer: Selective I_f inhibition in the SA node $\Rightarrow$ D

Answer: (D) Go Back to Q56

Q57.

Solution

Concept — Thiazide diuretics: They act in the early distal convoluted tubule. **Reasoning:** Hydrochlorothiazide blocks the Na^+/Cl^- symporter of the early DCT, producing a mild natriuresis and, over time, vasodilation that lowers blood pres-

sure. It is a mainstay first-line antihypertensive. **Why the other options are wrong:**

- (B) The $Na^+/K^+/2Cl^-$ cotransporter is the loop-diuretic target.
- (C) Carbonic anhydrase is the target of acetazolamide.
- (D) The mineralocorticoid receptor is the target of spironolactone.

Final Answer: Inhibits the DCT Na^+/Cl^- symporter $\Rightarrow$ A

Answer: (A) Go Back to Q57

Q58.

Solution

Concept — Fibrates: They are PPAR-α agonists used mainly to lower triglycerides. **Reasoning:** Fenofibrate activates the nuclear receptor PPAR-α, which increases lipoprotein-lipase expression and fatty-acid oxidation while reducing hepatic VLDL output, markedly lowering plasma triglycerides and modestly raising HDL. **Why the other options are wrong:**

- (A) HMG-CoA reductase inhibition describes statins.
- (C) Bile-acid binding describes resins (cholestyramine).
- (D) NPC1L1 inhibition describes ezetimibe.

Final Answer: PPAR-α activation raising lipoprotein-lipase activity $\Rightarrow$ B

Answer: (B) Go Back to Q58

Q59.

Solution

Concept — Fibrinolytics: Tissue plasminogen activators convert plasminogen to plasmin, which dissolves formed fibrin clots. **Reasoning:** Alteplase is recombinant t-PA; it is relatively fibrin-selective, activating plasminogen bound within the thrombus to generate plasmin that lyses fibrin, restoring vessel patency in acute MI and ischaemic stroke. **Why the other options are wrong:**

- (A) $P2Y_{12}$ blockade describes clopidogrel.
- (B) Direct factor-Xa inhibition describes rivaroxaban.
- (D) Antithrombin potentiation describes heparin.

Final Answer: Converts plasminogen to plasmin to lyse fibrin $\Rightarrow$ C

Answer: (C) Go Back to Q59

Q60.

Solution

Concept — Paracetamol: It inhibits cyclooxygenase predominantly in the central nervous system, with weak peripheral anti-inflammatory action. **Reasoning:** The central COX inhibition explains its good antipyretic and analgesic effects, while the high peroxide tone at peripheral inflammatory sites limits its action there, accounting for its weak anti-inflammatory and minimal anti-platelet activity. **Why the other options are wrong:**

- (B) Irreversible platelet COX-1 acetylation describes aspirin.
- (C) It is not a selective COX-2 inhibitor.
- (D) It does not block H_1 receptors.

Final Answer: Mainly central COX inhibition $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q60

Q61.

Solution

Concept — Proton-pump inhibitors: They irreversibly inhibit the gastric H^+/K^+-ATPase, the final step of acid secretion. **Reasoning:** Omeprazole is a prodrug activated in the acidic parietal-cell canaliculus, where it covalently inhibits the proton pump. Because it blocks the final common pathway (regardless of the stimulus), it suppresses acid more completely and for longer than an H_2-blocker. **Why the other options are wrong:**

- (A) Neutralising acid describes antacids.
- (B) H_2-receptor blockade describes ranitidine/famotidine.
- (C) Muscarinic antagonism is not its mechanism.

Final Answer: Irreversible H^+/K^+-ATPase (proton-pump) inhibition $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q61

Q62.

Solution

Concept — Aprepitant: It is a neurokinin-1 (NK_1) receptor antagonist. **Reasoning:** By blocking the action of substance P at central NK_1 receptors it is especially effective against the delayed phase of chemotherapy-induced nausea and vomiting, complementing 5-HT_3 antagonists and dexamethasone. **Why the other options are wrong:**

- (A) 5-HT_3 antagonism describes ondansetron.
- (C) D_2 antagonism describes metoclopramide/domperidone.
- (D) H_1 antagonism describes antihistamine anti-emetics.

Final Answer: NK_1 (substance-P) receptor antagonist $\Rightarrow$ B

Answer: (B) Go Back to Q62

Q63.

Solution

Concept — Oxazolidinones: Linezolid inhibits bacterial protein synthesis at an early step on the 50S subunit. **Reasoning:** It binds the 23S rRNA of the 50S subunit and prevents formation of the functional 70S initiation complex, blocking the very start of translation. This unique site means little cross-resistance, giving activity against MRSA and vancomycin-resistant enterococci. **Why the other options are wrong:**

- (A) Binding penicillin-binding proteins describes β-lactams.
- (B) DNA-gyrase inhibition describes fluoroquinolones.
- (D) DHFR inhibition describes trimethoprim.

Final Answer: 50S (23S rRNA) binding blocking 70S initiation $\Rightarrow$ C

Answer: (C) Go Back to Q63

Q64.

Solution

Concept — Co-trimoxazole: The combination blocks two sequential steps of bacterial folate synthesis. **Reasoning:** Sulfamethoxazole, a PABA analogue, inhibits dihydropteroate synthase, while trimethoprim inhibits dihydrofolate reductase.

Sequential blockade of the same pathway produces synergistic, often bactericidal, activity and reduces resistance. **Why the other options are wrong:**

- (B) Neither acts on the 30S ribosome.
- (C) Neither inhibits cell-wall cross-linking.
- (D) Neither inhibits DNA gyrase.

Final Answer: Sequential folate-pathway blockade (DHPS + DHFR) $\Rightarrow$ A

Answer: (A) Go Back to Q64

Q65.

Solution

Concept — Isoniazid: After activation by mycobacterial KatG it inhibits mycolic-acid synthesis. **Reasoning:** The activated drug inhibits InhA (the enoyl-ACP reductase), blocking synthesis of mycolic acids that are essential to the mycobacterial cell wall. Loss of these long-chain lipids is lethal to dividing organisms, making isoniazid a key bactericidal first-line antitubercular. **Why the other options are wrong:**

- (A) 30S inhibition describes aminoglycosides.
- (C) RNA-polymerase inhibition describes rifampicin.
- (D) Magnesium chelation is not its mechanism.

Final Answer: Inhibits mycolic-acid synthesis $\Rightarrow$ B

Answer: (B) Go Back to Q65

Q66.

Solution

Concept — Chloroquine: It accumulates in the parasite's acidic food vacuole and inhibits haem detoxification. **Reasoning:** As the parasite digests haemoglobin it releases toxic free haem, normally polymerised into inert haemozoin. Chloroquine concentrates in the vacuole and blocks haem polymerisation, so toxic free haem builds up and kills the erythrocytic schizont. **Why the other options are wrong:**

- (A) DHFR inhibition describes pyrimethamine.
- (B) Mitochondrial electron-transport block describes atovaquone.
- (C) The endoperoxide free-radical mechanism describes artemisinins.

Final Answer: Inhibits haem polymerisation, free haem accumulates $\Rightarrow$ D

Answer: (D) Go Back to Q66

Q67.

Solution

Concept — Thionamide antithyroid drugs: Carbimazole/methimazole inhibit thyroid peroxidase. **Reasoning:** By inhibiting thyroid peroxidase they block oxidation and organification of iodide and the coupling of iodotyrosines, reducing synthesis of T_4 and T_3. The effect develops over weeks as stored hormone is depleted. **Why the other options are wrong:**

- (B) β-blockade (e.g. propranolol) only controls symptoms, not synthesis.
- (C) Radioactive destruction describes radioiodine (^{131}I).
- (D) It is not a thyroid-hormone receptor agonist.

Final Answer: Inhibits thyroid peroxidase, reducing hormone synthesis $\Rightarrow$ A

Answer: (A) Go Back to Q67

Q68.

Solution

Concept — Specific antidotes: Each poison has a matched antidote; the therapeutic index (LD_{50}/ED_{50}) frames overdose risk. **Reasoning:** Heparin is reversed by protamine sulphate, NOT by vitamin K. Vitamin K reverses warfarin (which depletes vitamin-K-dependent factors), but it has no effect on heparin's potentiation of antithrombin. So the heparin-vitamin-K pairing in option (C) is incorrect. **Why the other options are wrong:**

- (A) Digoxin toxicity is correctly treated with digoxin-specific Fab fragments.
- (B) Acute iron poisoning is correctly chelated with deferoxamine.
- (D) Warfarin overdose is correctly reversed with vitamin K.

Final Answer: Heparin is reversed by protamine, not vitamin K $\Rightarrow$ C

Answer: (C) Go Back to Q68

Q69.

Solution

Concept — Baeyer–Villiger oxidation: A peroxy acid inserts an oxygen atom adjacent to a ketone carbonyl, converting the ketone into an ester (or a cyclic ketone into a lactone). **Reasoning:** The peroxy acid adds to the carbonyl to give a Criegee intermediate; the more substituted (more electron-rich) group then migrates to oxygen with retention, producing the ester shown. **Why the other options are wrong:**

- (A) Pinacol–pinacolone rearranges a 1,2-diol to a ketone, no O insertion.
- (B) Cannizzaro is a base-induced disproportionation of an aldehyde.
- (D) Wittig converts a carbonyl to an alkene, not an ester.

Final Answer: Baeyer–Villiger oxidation $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q69

Q70.

Solution

Concept — Wolff–Kishner reduction: A carbonyl is reduced to a methylene under strongly basic conditions via a hydrazone. **Reasoning:** The ketone first forms a hydrazone with NH_2NH_2; hot KOH then expels N_2 and delivers the CH_2 group. It is the base-side complement to the acid-side Clemmensen reduction. **Why the other options are wrong:**

- (B) Clemmensen uses Zn(Hg)/HCl (acidic), not hydrazine/base.
- (C) Rosenmund reduces an acyl chloride to an aldehyde.
- (D) Meerwein–Ponndorf–Verley reduces a ketone only to an alcohol.

Final Answer: Wolff–Kishner reduction $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q70

Q71.

Solution

Concept — Hofmann bromamide degradation: A primary amide is converted to a primary amine with loss of one carbon. **Reasoning:** Br_2/NaOH generate an *N*-bromoamide, which rearranges (migration of R to nitrogen) through an iso-

cyanate; hydrolysis releases RNH_2 and carbonate. The amine has one fewer carbon than the amide. **Why the other options are wrong:**

- (A) Gabriel synthesis builds amines from phthalimide, not amides.
- (B) Curtius uses an acyl azide, not Br_2/NaOH.
- (C) Schmidt uses hydrazoic acid (HN_3).

Final Answer: Hofmann bromamide reaction $\Rightarrow$ D

Answer: (D) Go Back to Q71

Q72.

Solution

Concept — Michael addition: Conjugate (1,4) addition of a soft, stabilised nucleophile to an α,β-unsaturated carbonyl. **Reasoning:** The malonate enolate (Michael donor) attacks the β-carbon of methyl vinyl ketone (Michael acceptor); protonation then gives the 1,4-adduct. The driving force is formation of a resonance-stabilised enolate. **Why the other options are wrong:**

- (A) Knoevenagel forms a C=C with loss of water, not a 1,4-adduct.
- (C) Perkin uses an aromatic aldehyde and an anhydride.
- (D) Stobbe is a condensation of succinate esters with carbonyls.

Final Answer: Michael addition $\Rightarrow$ B

Answer: (B) Go Back to Q72

Q73.

Solution

Concept — Conformational analysis of cyclohexane: Axial and equatorial substituents differ in steric strain. **Reasoning:** An axial methyl points directly at the two axial hydrogens on the same face (C3 and C5), creating 1,3-diaxial van der Waals repulsion. The equatorial chair avoids this, so it predominates (A-value of $CH_3 \approx 7.3$ kJ/mol). **Why the other options are wrong:**

- (B) The ring does not open during a flip.
- (C) Cyclohexane is not aromatic, so no such stabilisation exists.
- (D) Axial and equatorial forms are not torsionally identical; the axial is strained.

Final Answer: 1,3-diaxial strain destabilises the axial methyl $\Rightarrow$ A

Answer: (A) Go Back to Q73

Q74.

Solution

Concept — Birch reduction: A dissolving-metal reduction (Na or Li in liquid NH_3 with an alcohol) partially reduces an aromatic ring. **Reasoning:** The solvated electrons add to benzene, and the alcohol protonates the radical-anion intermediates, giving the unconjugated 1,4-cyclohexadiene. The ring is not fully reduced because the diene is less easily reduced than benzene. **Why the other options are wrong:**

- (A) Catalytic hydrogenation would saturate the ring to cyclohexane.
- (B) Clemmensen reduces carbonyls, not aromatic rings.
- (D) Rosenmund reduces acyl chlorides to aldehydes.

Final Answer: Birch reduction $\Rightarrow$ C

Answer: (C) Go Back to Q74

Q75.

Solution

Concept — Inductive effect on acidity: Electron-withdrawing chlorines stabilise the phenoxide conjugate base, raising acidity. **Reasoning:** Each Cl withdraws electron density, and the effect is additive. 2,4,6-Trichlorophenol bears three withdrawing chlorines, so its conjugate base is the most stabilised and it is the strongest acid ($pK_a \approx 6$, far below phenol's ≈ 10). **Why the other options are wrong:**

- (A) Phenol has no electron-withdrawing substituent and is the weakest here.
- (B) and (C) one chlorine gives only a modest increase, less than three.

Final Answer: 2,4,6-Trichlorophenol is the strongest acid $\Rightarrow$ D

Answer: (D) Go Back to Q75

Q76.

Solution

Concept — Butane conformations: Rotation about C2–C3 gives staggered (anti, gauche) and eclipsed forms. **Reasoning:** When the two methyl groups eclipse one another (dihedral 0°), both torsional strain and methyl–methyl steric strain are maximal. This syn (fully eclipsed) conformation is the global energy maximum of butane. **Why the other options are wrong:**

- (A) Anti has the methyls 180° apart, the lowest energy.
- (C) Gauche is staggered with methyls 60° apart.
- (D) Eclipsed forms are not staggered.

Final Answer: Fully eclipsed (syn), highest energy $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q76

Q77.

Solution

Concept — Knoevenagel condensation: An amine-catalysed aldol-type condensation of an active-methylene compound with a carbonyl. **Reasoning:** The doubly activated methylene is deprotonated by the weak amine base; the carbanion adds to the aldehyde and the aldol dehydrates to an α,β-unsaturated product. With malonic acid, decarboxylation follows (Doebner modification). **Why the other options are wrong:**

- (B) Cannizzaro is base-induced disproportionation, no C–C bond formed here.
- (C) Wurtz couples two alkyl halides with sodium.
- (D) Kolbe–Schmitt carboxylates phenoxide with CO_2.

Final Answer: Knoevenagel condensation $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q77

Q78.

Solution

Concept — Nucleophilicity in protic solvents: Solvation by hydrogen bonding masks the smaller, more basic ions. **Reasoning:** In a protic solvent the small F^- is tightly solvated and sluggish, while the large, polarisable, weakly solvated I^- is the best nucleophile. Hence $I^- > Br^- > Cl^- > F^-$ (the reverse of basicity). **Why the other options are wrong:**

- (A) That is the order of basicity, and the order in aprotic solvents, not protic nucleophilicity.
- (B) The halides differ markedly in nucleophilicity.
- (D) Solvation is precisely what governs the trend here.

Final Answer: $I^- > Br^- > Cl^- > F^- \Rightarrow \boxed{C}$

Answer: (C) Go Back to Q78

Q79.

Solution

Concept — Purine ring system: A pyrimidine ring fused to an imidazole ring, containing four nitrogens. **Reasoning:** The figure shows the bicyclic 6–5 fused system with four N atoms, which is the purine nucleus found in adenine, guanine and the xanthine alkaloids (caffeine, theophylline). **Why the other options are wrong:**

- (A) Indole is benzene fused to pyrrole (one N).
- (B) Quinoline is benzene fused to pyridine (one N).
- (C) Pteridine is pyrimidine fused to pyrazine (a different 6–6 system).

Final Answer: It is the purine ring system $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q79

Q80.

Solution

Concept — Zaitsev's rule: Elimination favours the thermodynamically more stable, more substituted alkene. **Reasoning:** Loss of water from the protonated tertiary alcohol gives a tertiary carbocation; loss of the proton that yields the more

substituted (trisubstituted) alkene is preferred, giving 2-methylbut-2-ene as the major product. **Why the other options are wrong:**

- (A) Anti-Markovnikov refers to HX addition with peroxides, not elimination orientation.
- (C) Hofmann (least substituted) applies to bulky quaternary ammonium eliminations.
- (D) Bredt's rule forbids bridgehead alkenes, irrelevant here.

Final Answer: Zaitsev's rule (more substituted alkene) $\Rightarrow$ B

Answer: (B) Go Back to Q80

Q81.

Solution

Concept — Amide resonance: The nitrogen lone pair delocalises into the carbonyl, giving the C–N bond partial double-bond character. **Reasoning:** As the second resonance form ($C{=}N^{+}$, O^{-}) shows, the C–N bond order is greater than one. This restricts rotation (rotation barrier $\approx$ 75–90 kJ/mol), so the six atoms of the peptide unit are coplanar, and the nitrogen is a very weak base. **Why the other options are wrong:**

- (A) It is not a pure single bond; rotation is hindered.
- (B) The lone pair is tied up in resonance, so the amide N is only weakly basic.
- (D) The oxygen bears a partial *negative* charge in the contributing form, not a positive one.

Final Answer: Partial double-bond character makes the amide planar $\Rightarrow$ C

Answer: (C) Go Back to Q81

Q82.

Solution

Concept — Finkelstein reaction: A halide-exchange S_N2 driven by solubility differences. **Reasoning:** NaI is soluble in acetone but NaCl and NaBr are not. As the alkyl iodide forms, the precipitated NaCl/NaBr is removed from equilibrium, pulling the exchange to completion. **Why the other options are wrong:**

- (A) Wurtz couples two alkyl halides with sodium metal.
- (B) Hunsdiecker is a decarboxylative halogenation of silver carboxylates.
- (C) Swarts exchanges Cl/Br for fluorine using metal fluorides.

Final Answer: Finkelstein reaction $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q82

Q83.

Solution

Concept — Optical purity and enantiomeric excess: Observed rotation is proportional to the net excess of one enantiomer. **Reasoning:** Optical purity $= 26/52 = 0.50$, i.e. 50% enantiomeric excess. The sample is a non-racemic (scalemic) mixture that is 75% (+) and 25% (–), giving an excess of 50% of the (+)-form. **Why the other options are wrong:**

- (B) A pure racemate would show zero rotation, not $+26°$.
- (C) The pure (–)-enantiomer would rotate $-52°$.
- (D) A meso compound is achiral and gives zero rotation.

Final Answer: Scalemic mixture, $\sim$50% e.e. of (+) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q83

Q84.

Solution

Concept — Carbonyl protecting groups: A ketone reacts with a 1,2-diol to give a cyclic acetal (1,3-dioxolane). **Reasoning:** The structure shows one carbon bonded to two –OR oxygens of a ring, i.e. an acetal. Acetals are stable to bases, nucleophiles and reducing agents but are cleaved by aqueous acid, which is why they protect carbonyls. **Why the other options are wrong:**

- (A) A hemiacetal has one –OR and one –OH on the same carbon.
- (C) An anhydride has two acyl groups sharing an oxygen.
- (D) A Schiff base contains a C=N, not two C–O bonds.

Final Answer: A cyclic acetal (1,3-dioxolane) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q84

Q85.

Solution

Concept — Renin–angiotensin pharmacology: Angiotensin II raises blood pressure via the AT_1 receptor. **Reasoning:** The "sartans" competitively block the AT_1 receptor, so they are angiotensin II receptor blockers (ARBs). They act *downstream* of, and differently from, ACE inhibitors. **Why the other options are wrong:**

- (A) ACE inhibitors ("-pril") block formation of angiotensin II, not the receptor.
- (B) β-blockers act on adrenoceptors.
- (C) Thiazides are sulfonamide diuretics acting on the renal tubule.

Final Answer: They are angiotensin II receptor blockers $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q85

Q86.

Solution

Concept — Sulfonylurea mechanism: They are insulin secretagogues acting on the pancreatic β-cell. **Reasoning:** Sulfonylureas bind the SUR1 subunit and close the ATP-sensitive K^+ channel; the resulting depolarisation opens voltage-gated Ca^{2+} channels, triggering insulin exocytosis. **Why the other options are wrong:**

- (B) α-glucosidase inhibition is the action of acarbose.
- (C) PPAR-γ activation is the thiazolidinedione (glitazone) mechanism.
- (D) SGLT2 inhibition is the gliflozin mechanism.

Final Answer: They close the β-cell K_{ATP} channel to release insulin $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q86

Q87.

Solution

Concept — Targeted anticancer agents: Imatinib is the prototype small-molecule kinase inhibitor. **Reasoning:** Imatinib occupies the ATP-binding cleft of the constitutively active BCR–ABL tyrosine kinase produced by the Philadelphia chromosome, halting the proliferative signalling that drives chronic myeloid leukaemia. **Why the other options are wrong:**

- (A) Alkylating agents (e.g. cyclophosphamide) cross-link DNA non-selectively.
- (B) Topoisomerase poisons are agents such as etoposide.
- (D) Folate antagonists are antimetabolites such as methotrexate.

Final Answer: A small-molecule tyrosine-kinase (BCR–ABL) inhibitor $\Rightarrow$ C

Answer: (C) Go Back to Q87

Q88.

Solution

Concept — Pharmacophore: The minimal three-dimensional feature set needed for target binding. **Reasoning:** A pharmacophore is the spatial arrangement of steric and electronic features (H-bond donors/acceptors, charged centres, hydrophobic and aromatic groups) that a molecule must present to be recognised by, and to activate or block, its biological target. **Why the other options are wrong:**

- (A) A toxic metabolite is unrelated to the binding definition.
- (C) The salt counter-ion does not define activity.
- (D) An excipient is a formulation aid, not part of the pharmacophore.

Final Answer: The essential 3-D feature arrangement for target binding $\Rightarrow$ B

Answer: (B) Go Back to Q88

Q89.

Solution

Concept — Combinatorial chemistry: Parallel/split-pool synthesis of large compound libraries. **Reasoning:** Combinatorial chemistry generates thousands of diverse analogues simultaneously (often on solid-phase beads using split-and-mix methods) to feed high-throughput screening during early drug discovery. **Why the other options are wrong:**

- (B) Fractional distillation is a physical separation method.
- (C) Kjeldahl analysis estimates nitrogen content.
- (D) Gravimetric titration is a quantitative analytical technique.

Final Answer: Combinatorial chemistry $\Rightarrow$ A

Answer: (A) Go Back to Q89

Q90.

Solution

Concept — Homologation in lead optimisation: Studying a homologous series to map a structure–activity trend. **Reasoning:** Adding successive –CH_2– units generates a homologous series; plotting potency against chain length (or against $\pi/\log P$) reveals the optimal substituent size, a standard SAR exercise. **Why the other options are wrong:**

- (A) Ring contraction changes ring size, not chain length.
- (B) Bioisosterism replaces a group with a similar one, not chain extension.
- (C) Racemisation interconverts enantiomers.

Final Answer: Homologation (a homologous series) $\Rightarrow$ D

Answer: (D) Go Back to Q90

Q91.

Solution

Concept — Azole antifungals: They inhibit fungal sterol biosynthesis. **Reasoning:** The azole ring nitrogen coordinates the heme iron of CYP51 (lanosterol 14-α-demethylase), blocking conversion of lanosterol to ergosterol. Depletion of ergosterol disrupts the fungal membrane. **Why the other options are wrong:**

- (A) Peptidoglycan synthesis is a bacterial-cell-wall target.
- (C) Viral DNA is not the azole target.
- (D) Azoles target the fungal sterol pathway; human CYP inhibition is an unwanted side effect, not the mechanism.

Final Answer: They block ergosterol biosynthesis $\Rightarrow$ B

Answer: (B) Go Back to Q91

Q92.

Solution

Concept — Aminoglycoside mechanism: They are bactericidal protein-synthesis inhibitors. **Reasoning:** Aminoglycosides bind the 30S ribosomal subunit (16S rRNA), causing misreading of the genetic code and premature termination, which produces faulty membrane proteins and bacterial death. **Why the other options**

are wrong:

- (A) The transpeptidase (PBP) is the β-lactam target.
- (B) DNA gyrase is the fluoroquinolone target.
- (D) Dihydropteroate synthase is the sulfonamide target.

Final Answer: The 30S ribosomal subunit $\Rightarrow$ C

Answer: (C) Go Back to Q92

Q93.

Solution

Concept — First-order kinetics: Rate $= k[A]$, and the half-life is concentration-independent. **Reasoning:** For a first-order process $t_{1/2} = \ln 2/k = 0.693/k$, a constant that does not depend on how much drug is present. This is why shelf-life prediction for first-order degradation is straightforward. **Why the other options are wrong:**

- (B) $t_{1/2} \propto [A]_0$ is true of a zero-order reaction, not first-order.
- (C) Concentration-squared dependence belongs to second-order kinetics.
- (D) The half-life depends on k, not a fixed clock time.

Final Answer: $t_{1/2} = 0.693/k$, independent of concentration $\Rightarrow$ A

Answer: (A) Go Back to Q93

Q94.

Solution

Concept — Arrhenius equation: Temperature controls the fraction of molecules exceeding the activation energy. **Reasoning:** Raising T increases the exponential term $e^{-E_a/RT}$, so a greater fraction of collisions have energy $\geq E_a$. The rate constant k rises sharply (roughly doubling per 10 K for many reactions). **Why the other options are wrong:**

- (A) E_a is a property of the pathway and does not fall to zero with temperature.
- (B) The pre-exponential factor A is positive.
- (C) ΔH of reaction is unrelated to the temperature dependence of k.

Final Answer: More molecules reach E_a, raising the exponential term $\Rightarrow$ D

Answer: (D) Go Back to Q94

Q95.

Solution

Concept — Inorganic haematinics: Iron salts replenish body iron stores. **Reasoning:** Ferrous sulfate ($FeSO_4 \cdot 7H_2O$) is the standard oral iron supplement for iron-deficiency anaemia; the ferrous (Fe^{2+}) form is better absorbed than ferric iron. **Why the other options are wrong:**

- (A) Aluminium hydroxide is an antacid.
- (C) Sodium chloride is an electrolyte/tonicity agent.
- (D) Zinc oxide is a topical protective/astringent.

Final Answer: Ferrous sulfate is the haematinic $\Rightarrow$ B

Answer: (B) Go Back to Q95

Q96.

Solution

Concept — Hunsdiecker reaction: Decarboxylative halogenation of a silver carboxylate. **Reasoning:** RCOOAg reacts with Br_2 to give an acyl hypobromite, which undergoes radical decarboxylation to lose CO_2 and form R–Br, a product with one fewer carbon than the acid. **Why the other options are wrong:**

- (B) Finkelstein is a simple halide exchange, no carbon lost.
- (C) Reimer–Tiemann formylates phenols with dichlorocarbene.
- (D) Sandmeyer replaces a diazonium group, not a carboxylate.

Final Answer: Hunsdiecker reaction $\Rightarrow$ A

Answer: (A) Go Back to Q96

Q97.

Solution

Concept — Mutarotation: Anomers of a sugar interconvert in solution, changing the optical rotation until equilibrium. **Reasoning:** Crystalline α-D-glucose ($[\alpha] \approx +112^\circ$) and β-D-glucose ($[\alpha] \approx +19^\circ$) interconvert through the open-chain aldehyde, so any fresh solution drifts to the equilibrium value of about $+52.7^\circ$.

This change is mutarotation. **Why the other options are wrong:**

- (A) Racemisation interconverts enantiomers, but the anomers are diastereomers.
- (B) Tautomerism is a separate keto–enol type equilibrium.
- (D) Only the anomeric (C1) centre changes, not every centre.

Final Answer: It is mutarotation $\Rightarrow$ C

Answer: (C) Go Back to Q97

Q98.

Solution

Concept — Endothermic reactions: If products lie above reactants in energy, heat is absorbed. **Reasoning:** The diagram shows the product level higher than the reactant level, so $\Delta H = H_{\text{products}} - H_{\text{reactants}} > 0$. The reaction absorbs energy and the products are less stable than the reactants. **Why the other options are wrong:**

- (A) Exothermic reactions have products *below* reactants ($\Delta H < 0$).
- (B) Thermoneutral would place reactants and products at the same level.
- (C) Endothermic reactions are entirely feasible, especially when entropy-driven.

Final Answer: Endothermic, positive $\Delta H \Rightarrow$ D

Answer: (D) Go Back to Q98

Q99.

Solution

Concept — Bromometry: bromine is generated *in situ* from a bromate–bromide mixture in acid. **Reasoning:** A standardised $KBrO_3$/KBr solution is the usual bromometric titrant. On acidification, $BrO_3^- + 5Br^- + 6H^+ \rightarrow 3Br_2 + 3H_2O$, and the liberated bromine brominates phenols and aromatic amines quantitatively. **Why the other options are wrong:**

- (A) Thiosulphate and starch belong to iodometric back-titration, not the bromine generator.
- (B) Iodine monochloride is used in iodine-value (Wijs) determinations, not

standard bromometry.
- (D) Ceric ammonium sulphate is the cerimetric oxidant, a different method.

Final Answer: Potassium bromate and potassium bromide $\Rightarrow$ C

Answer: (C) Go Back to Q99

Q100.

Solution

Concept — Beer–Lambert law: $A = \varepsilon c l$, so the slope of A versus c equals εl. **Reasoning:** The plotted slope is $A/c = \varepsilon l$. With $l = 2.0$ cm and slope $= 1.0 \times 10^4$ L mol^{-1}, $\varepsilon = (\text{slope})/l = (1.0 \times 10^4)/2.0 = 5.0 \times 10^3$ L mol^{-1} cm^{-1}. **Why the other options are wrong:**

- (A) 1.0×10^4 is the slope itself (εl), not ε; it ignores the 2 cm path.
- (C) 2.0×10^4 multiplies by the path length instead of dividing.
- (D) 1.0×10^2 is off by two orders of magnitude.

Final Answer: $\varepsilon = 1.0 \times 10^4/2.0 = 5.0 \times 10^3$ L mol^{-1} cm^{-1} $\Rightarrow$ B

Answer: (B) Go Back to Q100

Q101.

Solution

Concept — Diazotization titration: primary aromatic amines react with nitrous acid to form diazonium salts. **Reasoning:** The titrant is standard sodium nitrite, which generates nitrous acid in acid medium; this converts the $-NH_2$ group to a diazonium salt in a $1:1$ reaction. The end point is found with a starch–iodide external indicator (blue with the first excess of nitrite) or electrometrically. **Why the other options are wrong:**

- (A) Permanganate is a redox titrant, not used for diazotization.
- (B) Silver nitrate is for argentometric (halide) titrations.
- (C) Perchloric acid is the non-aqueous titrant for weak bases.

Final Answer: Standard sodium nitrite $\Rightarrow$ D

Answer: (D) Go Back to Q101

Q102.

Solution

Concept — Gradient vs isocratic: the difference lies in whether mobile-phase strength changes during the run. **Reasoning:** In gradient elution the mobile-phase composition is changed with time (typically increasing the proportion of organic modifier), which speeds up later-eluting, strongly retained peaks. Isocratic elution holds the composition constant. **Why the other options are wrong:**

- (B) Constant composition is the definition of isocratic, not gradient.
- (C) The stationary phase is not changed during a run.
- (D) HPLC always uses a high-pressure pump; gravity flow describes open-column work.

Final Answer: Mobile-phase composition is progressively changed $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q102

Q103.

Solution

Concept — IR triple-bond region: bands near 2200–2260 cm^{-1} are diagnostic of C≡N and C≡C groups. **Reasoning:** A sharp band of medium intensity at about 2250 cm^{-1}, sitting in the otherwise empty region between C–H ($\sim$2900) and C=O ($\sim$1700) absorptions, is the classic nitrile C≡N stretch. **Why the other options are wrong:**

- (A) An ester C=O appears near 1735 cm^{-1}, far from 2250.
- (C) An alcohol O–H stretch is broad and near 3200–3600 cm^{-1}.
- (D) A C–Cl stretch lies low, around 600–800 cm^{-1}.

Final Answer: 2250 cm^{-1} $\Rightarrow$ nitrile C≡N $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q103

Q104.

Solution

Concept — Oxygen-flask combustion: the drug is burned in oxygen so that heteroatoms become absorbable ions. **Reasoning:** In the Schöniger method the organic sample is ignited in a closed flask filled with oxygen; combustion converts

bound halogen or sulphur into hydrogen halide or sulphate, which is absorbed in an alkaline/peroxide solution and then determined by titration. **Why the other options are wrong:**

- (A) Optical rotation is measured polarimetrically, not by combustion.
- (B) Melting point is a separate physical test.
- (D) Water content is determined by Karl Fischer titration.

Final Answer: Decomposes the drug so halogen/sulphur can be titrated $\Rightarrow$ C

Answer: (C) Go Back to Q104

Q105.

Solution

Concept — R_f in TLC: $R_f = \dfrac{\text{distance moved by solute}}{\text{distance moved by solvent front}}$. **Reasoning:** The solute moved 4.8 cm and the solvent front 12.0 cm, so $R_f = 4.8/12.0 = 0.40$. R_f is dimensionless and lies between 0 and 1. **Why the other options are wrong:**

- (B) 2.50 is the inverse ratio $(12.0/4.8)$; R_f cannot exceed 1.
- (C) 0.48 mistakenly divides by 10.
- (D) 0.60 uses a wrong solute distance.

Final Answer: $R_f = 4.8/12.0 = 0.40 \Rightarrow$ A

Answer: (A) Go Back to Q105

Q106.

Solution

Concept — Specific absorbance: $A_{1\text{cm}}^{1\%}$ is the absorbance of a 1% w/v solution in a 1 cm cell. **Reasoning:** By pharmacopoeial definition, $A_{1\text{cm}}^{1\%}$ is the absorbance of a solution containing 1 g of substance in 100 mL (i.e. 1% w/v) measured over a 1 cm path. It avoids needing the molar mass, which is why pharmacopoeias quote it. **Why the other options are wrong:**

- (A) 1 mol L^{-1} in 1 cm defines molar absorptivity ε, not $A_{1\text{cm}}^{1\%}$.
- (B) A microgram per mL in a 10 cm cell is an arbitrary, incorrect basis.
- (C) 1 mol L^{-1} in 10 cm is neither the molar nor the specific definition.

Final Answer: 1% w/v (1 g/100 mL) in a 1 cm cell $\Rightarrow$ D

Answer: (D) Go Back to Q106

Q107.

Solution

Concept — Equivalent weight: eq. wt $= \dfrac{\text{molar mass}}{\text{number of electrons exchanged per formula unit}}$. **Reasoning:** Each As(III)$\rightarrow$As(V) is a 2-electron change, and one As_2O_3 contains 2 arsenic atoms, so it exchanges 4 electrons per mole. Hence eq. wt $= 197.8/4 = 49.45$ g eq^{-1}. **Why the other options are wrong:**

- (A) $98.9 = 197.8/2$ uses only a 2-electron change.
- (C) 197.8 treats it as a 1-electron change.
- (D) $395.6 = 197.8 \times 2$ multiplies instead of dividing.

Final Answer: $197.8/4 = 49.45$ g eq$^{-1} \Rightarrow$ B

Answer: (B) Go Back to Q107

Q108.

Solution

Concept — Selectivity factor: $\alpha = k_2'/k_1' = (t_{R2} - t_0)/(t_{R1} - t_0)$. **Reasoning:** With $t_0 = 1.0$, $t_{R1} = 3.0$, $t_{R2} = 5.0$: $k_1' = (3.0 - 1.0)/1.0 = 2.0$ and $k_2' = (5.0 - 1.0)/1.0 = 4.0$. So $\alpha = 4.0/2.0 = 2.0$. Equivalently $\alpha = (5.0 - 1.0)/(3.0 - 1.0) = 4.0/2.0 = 2.0$. **Why the other options are wrong:**

- (A) 1.67 comes from forgetting to subtract t_0 $(5.0/3.0)$.
- (B) 1.25 uses wrong arithmetic on the adjusted times.
- (D) 0.5 inverts the ratio.

Final Answer: $\alpha = (5.0 - 1.0)/(3.0 - 1.0) = 2.0 \Rightarrow$ C

Answer: (C) Go Back to Q108

Q109.

Solution

Concept — ppm vs Hz in NMR: chemical shift in ppm is field-independent, but the same shift in Hz scales with the operating frequency. **Reasoning:** Separation in Hz $= \Delta\delta \times \nu_0$. At 300 MHz, 1.0 ppm $= 300$ Hz; at 600 MHz the same 1.0 ppm $= 600$ Hz. So the Hz separation doubles while the ppm value is unchanged. (Higher field therefore spreads peaks out and improves resolution.) **Why the other options are wrong:**

- (A) Hz separation does change with field; only ppm stays fixed.
- (B) It increases, not halves, with higher field.
- (C) The separation does not vanish at high field; it grows.

Final Answer: Hz separation doubles (300→600 Hz) ⇒ $\boxed{\text{D}}$

Answer: (D) Go Back to Q109

Q110.

Solution

Concept — GC temperature programming: raising oven temperature during the run shortens analysis of high-boiling solutes. **Reasoning:** A mixture spanning a wide volatility range gives broad, late, flat peaks if run isothermally. Programming the temperature upward elutes the more volatile components early at low temperature and the heavier ones quickly at high temperature, giving sharp peaks in a reasonable time. **Why the other options are wrong:**

- (B) The carrier gas polarity does not change with temperature.
- (C) The column chemistry (polarity) is fixed; heating does not convert it.
- (D) Detector flame temperature is controlled separately, not by the column program.

Final Answer: Elutes components of differing volatility in good time with sharp peaks ⇒ $\boxed{\text{A}}$

Answer: (A) Go Back to Q110

Q111.

Solution

Concept — Reading λ_{max}: the dashed line drops from the peak top to the wavelength axis. **Reasoning:** The marked maximum (orange dot, dashed line) sits at the right-hand tick, which the axis labels as 340 nm. The peak top corresponds to $\lambda_{max} \approx 340$ nm, the wavelength chosen for the assay. **Why the other options are wrong:**

- (A) 260 nm is on the rising part of the curve, not the maximum.
- (B) 300 nm is below the peak on the ascending slope.
- (D) 220 nm lies off the plotted range and is not the maximum.

Final Answer: $\lambda_{max} \approx 340$ nm $\Rightarrow$ C

Answer: (C) Go Back to Q111

Q112.

Solution

Concept — Diprotic-acid titration: a diprotic acid shows two equivalence points, one per ionisable proton. **Reasoning:** The first steep region E_1 marks complete neutralisation of the first (stronger) proton; the second steep region E_2 marks neutralisation of the second proton. Each step consumes one equivalent of base, so E_2 occurs at roughly twice the volume of E_1. **Why the other options are wrong:**

- (A) They are two genuinely different equivalence points, not one counted twice.
- (C) Two distinct steps appear precisely because the two pK_a values differ.
- (D) Indicators do not decompose; the steps are real neutralisation events.

Final Answer: Neutralisation of the first and second protons $\Rightarrow$ B

Answer: (B) Go Back to Q112

Q113.

Solution

Concept — Ion-exchange resins: cation exchangers bear fixed negative groups that exchange cations. **Reasoning:** A strong cation-exchange resin carries sulphonic acid groups ($-SO_3^-H^+$). These fixed anionic sites exchange their H^+ (or Na^+) counter-ions for cations from the sample solution. **Why the other options are wrong:**

- (B) Quaternary ammonium ($-NR_3^+$) groups define a strong *anion*-exchange resin.
- (C) Hydroxyl groups alone give no ion-exchange capacity.
- (D) Neutral C18 chains are the reverse-phase, not ion-exchange, functionality.

Final Answer: Sulphonic acid groups ($-SO_3^-H^+$) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q113

Q114.

Solution

Concept — Mass-spectral fragment losses: characteristic neutral losses identify lost groups. **Reasoning:** A peak at M-15 corresponds to loss of a fragment of mass 15, which is a methyl radical (CH_3, $12 + 3 = 15$). This is common in compounds bearing a methyl group attached to the charge-bearing skeleton. **Why the other options are wrong:**

- (A) Loss of water (H_2O) gives M-18, not M-15.
- (B) Loss of CO gives M-28.
- (C) Loss of OH gives M-17.

Final Answer: M-15 $\Rightarrow$ loss of a methyl radical (CH_3) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q114

Q115.

Solution

Concept — Peak asymmetry factor: $A_s = b/a$ at 5% of peak height. **Reasoning:** With leading half-width $a = 0.20$ min and trailing half-width $b = 0.30$ min, $A_s = b/a = 0.30/0.20 = 1.5$. A value greater than 1 indicates tailing; 1.0 is an ideal symmetrical peak. **Why the other options are wrong:**

- (A) 0.67 is the reciprocal (a/b), describing fronting, not the defined factor.
- (C) 0.50 takes only the trailing width and divides by total.
- (D) 6.0 misplaces the decimal point.

Final Answer: $A_s = 0.30/0.20 = 1.5 \Rightarrow$ B

Answer: (B) Go Back to Q115

Q116.

Solution

Concept — AAS background correction: a continuum source corrects for non-atomic absorption. **Reasoning:** A deuterium continuum lamp emits broadband light; only broad, non-specific background (molecular absorption and scatter) attenuates it appreciably, whereas the narrow atomic line absorbs a negligible fraction of the continuum. Subtracting the D_2 reading from the hollow-cathode reading corrects the analyte signal for background. **Why the other options are wrong:**

- (B) Atomisation is done by the flame or graphite furnace, not the D_2 lamp.
- (C) The sharp resonance line is supplied by the hollow-cathode lamp.
- (D) The monochromator is a separate dispersing element.

Final Answer: Corrects for non-specific background absorption $\Rightarrow$ A

Answer: (A) Go Back to Q116

Q117.

Solution

Concept — Mercurimetric titration: Hg^{2+} binds chloride as a soluble, weakly dissociated complex. **Reasoning:** Titrating chloride with $Hg(NO_3)_2$ forms soluble mercuric chloride, $HgCl_2$, which is only slightly dissociated, so free Hg^{2+} remains

very low until all chloride is consumed. The end point is shown by an indicator such as diphenylcarbazone reacting with the first excess Hg^{2+}. **Why the other options are wrong:**

- (A) Hg_2Cl_2 (calomel) is mercurous, not the mercuric species formed here.
- (B) No chlorine gas is liberated in the titration.
- (D) HgO is not the titration product.

Final Answer: Soluble, poorly dissociated $HgCl_2 \Rightarrow \boxed{C}$

Answer: (C) Go Back to Q117

Q118.

Solution

Concept — HPLC flow path: solvent $\rightarrow$ degasser $\rightarrow$ pump $\rightarrow$ injector $\rightarrow$ column $\rightarrow$ detector. **Reasoning:** The unit between the solvent reservoir and the pump that removes dissolved air is the mobile-phase degasser. Removing air prevents bubbles that would otherwise disturb the pump and produce spurious detector spikes. **Why the other options are wrong:**

- (A) The column is downstream of the injector, not before the pump.
- (B) The injection valve introduces sample after the pump.
- (C) The UV detector is the last unit, after the column.

Final Answer: Z is the mobile-phase degasser $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q118

Q119.

Solution

Concept — Why fluorimetry is sensitive: emitted light is measured against a dark field. **Reasoning:** In fluorimetry the detector views light emitted (usually at $90°$) against an essentially black background, so even a faint signal stands out. UV absorption, by contrast, measures a small drop in a large transmitted-light value, which limits low-level detection. Hence fluorimetry can be orders of magnitude more sensitive. **Why the other options are wrong:**

- (A) Measuring a small difference between two large signals describes the *less* sensitive absorption method.

- (B) Emission occurs at a longer wavelength than absorption (Stokes shift), not the same.
- (D) Fluorescence intensity is proportional to concentration at low levels, not independent of it.

Final Answer: Emitted light is read against a near-dark background $\Rightarrow$ C

Answer: (C) Go Back to Q119

Q120.

Solution

Concept — Molarity to normality: $N = M\times$ (equivalents per mole). **Reasoning:** Oxalic acid provides 2 replaceable H^+ per molecule, so its acid equivalence factor is 2. Therefore $N = 0.20 \times 2 = 0.40$ N. **Why the other options are wrong:**

- (A) 0.10 N divides by 2 instead of multiplying.
- (C) 0.20 N treats it as monoprotic.
- (D) 0.80 N uses a factor of 4.

Final Answer: $N = 0.20 \times 2 = 0.40$ N $\Rightarrow$ B

Answer: (B) Go Back to Q120

Q121.

Solution

Concept — Polarography (Ilkovič relation): the diffusion-limited current is proportional to analyte concentration. **Reasoning:** At the dropping-mercury electrode, once the applied potential is past the half-wave value, the current is limited by diffusion of the electroactive species to the drop. By the Ilkovič equation the limiting (diffusion) current is directly proportional to bulk concentration, which is the basis of quantitative polarography. **Why the other options are wrong:**

- (B) The current is not proportional to the square of temperature.
- (C) Polarography is an electrochemical, not optical, method; lamp wavelength is irrelevant.
- (D) Refractive index does not govern the diffusion current.

Final Answer: Limiting current $\propto$ concentration of the electroactive species $\Rightarrow$ A

Answer: (A) Go Back to Q121

Q122.

Solution

Concept — System suitability test: a pre-run check that the whole chromatographic system performs adequately. **Reasoning:** Repeated injections of a standard are used to confirm resolution, tailing factor, theoretical plate count and the % RSD of peak responses meet pharmacopoeial limits before the assay proceeds. This verifies the instrument, column and conditions together. **Why the other options are wrong:**

- (A) An LOD study estimates the smallest detectable amount, a validation parameter, not this pre-run check.
- (B) Linearity verification examines response across concentrations, done during validation.
- (D) Forced degradation stresses the drug to test stability-indicating power, not a routine pre-run check.

Final Answer: This pre-analysis check is the system suitability test $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q122

Q123.

Solution

Concept — Alphabetical classification: Crude drugs are arranged in dictionary order of their Latin or common English names. **Reasoning:** Listing drugs as Acacia, Benzoin, Cinnamon, Dill is purely by name order, with no scientific basis. This is alphabetical classification, used in pharmacopoeias and dictionaries for quick reference. **Why the other options are wrong:**

- (A) Morphological grouping is by the plant part used.
- (C) Serological grouping is by immunological/protein relationships.
- (D) Chemotaxonomical grouping combines chemistry with botany.

Final Answer: Ordering by name is alphabetical classification $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q123

Q124.

Solution

Concept — Taxonomical classification: Crude drugs are grouped by their botanical position, that is by family, genus and species. **Reasoning:** Putting all Apocynaceae together and all Solanaceae together, regardless of plant part or constituent, is grouping by botanical relationship, which is taxonomical (botanical) classification. **Why the other options are wrong:**

- (B) Chemical grouping is by constituent class.
- (C) Pharmacological grouping is by therapeutic action.
- (D) Alphabetical grouping is by name order.

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

Answer: (A) Go Back to Q124

Q125.

Solution

Concept — Organized vs unorganized drugs: Unorganized drugs are cell-free products of plant metabolism such as gums, resins, balsams and dried exudates. **Reasoning:** Benzoin is a balsamic resin exudate obtained by incising the bark; it has no cellular tissue and is therefore an unorganized drug. The other three are entire fruits/seeds (organized organs). **Why the other options are wrong:**

- (A) Fennel fruit is an organized organ.
- (B) Cardamom seed is an organized organ.
- (D) Coriander fruit is an organized organ.

Final Answer: Benzoin is a cell-free balsamic exudate $\Rightarrow$ C

Answer: (C) Go Back to Q125

Q126.

Solution

Concept — Ergot alkaloids: Ergot is the dried sclerotium of *Claviceps purpurea* developed on the ovary of rye (*Secale cereale*). **Reasoning:** The ergoline alkaloids, including the oxytocic ergometrine and the vasoconstrictor ergotamine, are produced in this fungal sclerotium, so the source is *Claviceps purpurea*. **Why the**

other options are wrong:

- (A) *Aspergillus niger* is used for citric acid, not ergot alkaloids.
- (B) *Penicillium notatum* gives penicillin.
- (C) *Agaricus campestris* is an edible mushroom.

Final Answer: Ergometrine comes from *Claviceps purpurea* ⇒ D

Answer: (D) Go Back to Q126

Q127.

Solution

Concept — Piperine: Piperine is the pungent acid-amide alkaloid of black pepper, the dried unripe fruit of *Piper nigrum*. **Reasoning:** Piperine accounts for the characteristic bite of pepper and acts as a bio-enhancer by inhibiting drug-metabolising enzymes; its source is *Piper nigrum*. **Why the other options are wrong:**

- (B) *Zingiber officinale* (ginger) yields gingerol.
- (C) *Elettaria cardamomum* yields cardamom volatile oil.
- (D) *Capsicum frutescens* yields capsaicin.

Final Answer: Piperine is from *Piper nigrum* ⇒ A

Answer: (A) Go Back to Q127

Q128.

Solution

Concept — Capsaicin: Capsaicin is the vanillylamide pungent principle of chillies, the dried ripe fruits of *Capsicum annuum* (and related species). **Reasoning:** It is used as a counter-irritant rubefacient and in TRPV1-mediated analgesic preparations; its botanical source among the options is *Capsicum annuum*. **Why the other options are wrong:**

- (A) *Piper longum* yields piperine-type amides.
- (C) *Brassica juncea* yields sinigrin (mustard).
- (D) *Myristica fragrans* yields nutmeg oil.

Final Answer: Capsaicin is from *Capsicum annuum* ⇒ B

Answer: (B) Go Back to Q128

Q129.

Solution

Concept — Alkaloid precipitating reagents: Mayer's reagent is potassium mercuric iodide and gives a cream/white precipitate with alkaloids. **Reasoning:** A cream-coloured precipitate with potassium mercuric iodide is the classic positive Mayer's test for alkaloids. **Why the other options are wrong:**

- (A) Dragendorff's (potassium bismuth iodide) gives an orange-brown precipitate.
- (B) Wagner's (iodine in potassium iodide) gives a reddish-brown precipitate.
- (D) Hager's (picric acid) gives a yellow precipitate.

Final Answer: Cream precipitate with KHgI is Mayer's test $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q129

Q130.

Solution

Concept — Glycoside classification by linkage: Glycosides are named by the atom of the aglycone bound to the sugar: O-, N-, S- or C-glycosides. **Reasoning:** When the sugar is joined to the aglycone by a direct carbon-to-carbon bond, as in aloin/barbaloin, it is a C-glycoside; these resist acid hydrolysis. The schematic shows exactly such a C–C linkage. **Why the other options are wrong:**

- (A) O-glycosides are joined through oxygen.
- (B) N-glycosides are joined through nitrogen.
- (C) S-glycosides (thioglycosides) are joined through sulphur.

Final Answer: A direct C–C sugar link defines a C-glycoside $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q130

Q131.

Solution

Concept — Quillaia saponins: Quillaia (soap bark) yields triterpenoid saponin glycosides that lower surface tension and froth in water. **Reasoning:** The official source of quillaia is *Quillaja saponaria*; its saponins are used as foaming, emulsifying and adjuvant agents. **Why the other options are wrong:**

- (B) *Glycyrrhiza glabra* gives the saponin glycyrrhizin but is liquorice, not quillaia.
- (C) *Dioscorea deltoidea* gives steroidal diosgenin.
- (D) *Centella asiatica* gives triterpene asiaticosides, a different drug.

Final Answer: Quillaia is *Quillaja saponaria* ⇒ A

Answer: (A) Go Back to Q131

Q132.

Solution

Concept — Cyanogenetic glycosides: These yield hydrocyanic acid on hydrolysis; amygdalin (and its monosaccharide analogue prunasin) occurs in *Prunus* species. **Reasoning:** Wild cherry bark contains prunasin/amygdalin, which on hydrolysis by emulsin gives benzaldehyde, glucose and HCN; the trace HCN accounts for its mild sedative-antitussive use. **Why the other options are wrong:**

- (A) Sinigrin is a glucosinolate (thioglycoside) of mustard.
- (C) Sennoside is an anthraquinone glycoside.
- (D) Glycyrrhizin is a saponin glycoside.

Final Answer: The cyanogenetic glycoside is prunasin/amygdalin ⇒ B

Answer: (B) Go Back to Q132

Q133.

Solution

Concept — Flavonoid glycosides of *Citrus*: The peel of unripe *Citrus* fruits is rich in hesperidin, a flavanone-7-rutinoside. **Reasoning:** Hesperidin is the vitamin-P-active flavonoid glycoside used as a capillary protectant and venotonic; it is recovered from *Citrus* peel. **Why the other options are wrong:**

- (A) Diosgenin is a steroidal sapogenin.
- (B) Digitoxin is a cardiac glycoside.
- (D) Sennoside A is an anthraquinone glycoside.

Final Answer: The *Citrus* flavonoid glycoside is hesperidin ⇒ C

Answer: (C) Go Back to Q133

Q134.

Solution

Concept — Cardamom oil: The volatile oil of cardamom (*Elettaria cardamomum*) is dominated by 1,8-cineole and α-terpinyl acetate. **Reasoning:** These two constituents give cardamom its characteristic aromatic, carminative odour; α-terpinyl acetate provides the sweet note and cineole the camphoraceous note. **Why the other options are wrong:**

- (A) Eugenol/acetyleugenol characterise clove oil.
- (B) Menthol/menthone characterise peppermint oil.
- (C) Citral/geraniol characterise lemongrass oil.

Final Answer: Cardamom oil is rich in 1,8-cineole and α-terpinyl acetate $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q134

Q135.

Solution

Concept — Ajowan / thymol: Ajowan fruit (*Trachyspermum ammi*) yields a volatile oil from which the crystalline phenol thymol separates on cooling. **Reasoning:** Thymol, an isomer of carvacrol, is the antiseptic and anthelmintic phenolic constituent of ajowan oil and is traditionally called "ajwain-ka-phool". **Why the other options are wrong:**

- (B) Eugenol is from clove.
- (C) Anethole is from fennel/anise.
- (D) Safrole is from sassafras.

Final Answer: The ajowan phenol is thymol $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q135

Q136.

Solution

Concept — Turmeric: The yellow diarylheptanoid pigment of turmeric rhizome (*Curcuma longa*) is curcumin. **Reasoning:** Curcumin is the chief colouring and anti-inflammatory/antioxidant principle of turmeric, while the volatile oil contains turmerone. **Why the other options are wrong:**

- (A) Gingerol is the pungent principle of ginger.
- (C) Carvone is a monoterpene of caraway/spearmint.
- (D) Shogaol is a dehydration product of gingerol.

Final Answer: The turmeric pigment is curcumin $\Rightarrow$ B

Answer: (B) Go Back to Q136

Q137.

Solution

Concept — Balsam of Tolu: It is a pathological balsamic exudate rich in benzoic and cinnamic acid esters, used as an expectorant and flavour. **Reasoning:** Balsam of Tolu is obtained from the trunk of *Myroxylon balsamum* (var. *balsamum*); the related balsam of Peru comes from another variety of the same species. **Why the other options are wrong:**

- (A) *Liquidambar orientalis* gives storax.
- (B) *Commiphora myrrha* gives myrrh oleo-gum-resin.
- (D) *Pinus palustris* gives turpentine/colophony.

Final Answer: Balsam of Tolu is from *Myroxylon balsamum* $\Rightarrow$ C

Answer: (C) Go Back to Q137

Q138.

Solution

Concept — Mevalonate pathway: Three acetyl-CoA units condense to HMG-CoA, which is reduced to mevalonic acid and converted to the C5 unit IPP. **Reasoning:** The committed, rate-limiting step (HMG-CoA $\rightarrow$ mevalonate) is catalysed by HMG-CoA reductase. IPP and its isomer DMAPP are the building blocks of all terpenoids and steroids. **Why the other options are wrong:**

- (A) Chorismate synthase belongs to the shikimate pathway.
- (B) PAL acts in the phenylpropanoid pathway.
- (C) Polyketide synthase builds fatty-acid/polyketide chains, not isoprenoids.

Final Answer: HMG-CoA reductase; terpenoids via IPP/DMAPP $\Rightarrow$ D

Answer: (D) Go Back to Q138

Q139.

Solution

Concept — Phenylpropanoid pathway: Phenylalanine, formed via shikimate, is deaminated to trans-cinnamic acid as the entry into C6–C3 metabolites. **Reasoning:** This deamination is catalysed by phenylalanine ammonia lyase (PAL), the committed first step that funnels carbon toward lignans, coumarins, flavonoids and lignin. **Why the other options are wrong:**

- (B) HMG-CoA reductase is in the mevalonate pathway.
- (C) Squalene epoxidase is in triterpene/sterol biosynthesis.
- (D) Tryptophan decarboxylase initiates indole-alkaloid biosynthesis.

Final Answer: The deaminating enzyme is PAL $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q139

Q140.

Solution

Concept — Swelling index: The swelling index is the volume in mL taken up by 1 g of a drug, including adhering mucilage, after swelling in water for four hours. **Reasoning:** Mucilaginous drugs such as isabgol husk are standardised by this parameter, which reflects their water-imbibing (bulk-laxative) capacity. **Why the other options are wrong:**

- (A) Foaming index measures saponin-induced froth.
- (C) Acid value relates to free fatty acids in fixed oils.
- (D) Crude fibre is the acid/alkali-insoluble residue.

Final Answer: The hydration-volume parameter is the swelling index $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q140

Q141.

Solution

Concept — Foaming index: Foaming index $= 1000/a$, where a is the volume in mL of the decoction in the tube that just gives a persistent 1 cm froth. **Reasoning:** Here the lowest dilution still foaming uses $a = 8$ mL of decoction, so foaming index $= 1000/8 = 125$. **Why the other options are wrong:**

- (A) 80 wrongly uses $1000/12.5$ or misreads the volume.
- (B) 8 is the volume a itself, not the index.
- (D) 1000 would require $a = 1$ mL.

Final Answer: Foaming index $= 1000/8 = 125 \Rightarrow$ C

Answer: (C) Go Back to Q141

Q142.

Solution

Concept — Crude fibre and R_f: Crude fibre is the organic residue insoluble in both dilute acid and dilute alkali; R_f = spot distance / solvent-front distance. **Reasoning:** Crude fibre remains after successive acid and alkali boiling, washing and ignition (the inorganic part is corrected by ashing). For the TLC plate, spot 1 gives $R_f = 1.2/4.0 = 0.30$ and spot 2 gives $R_f = 2.8/4.0 = 0.70$, matching option (D). **Why the other options are wrong:**

- (A) Crude fibre is not the alcohol-soluble extractive; R_f of spot 2 is not 0.12.
- (B) Crude fibre is not the total ash, and the two spots have different R_f values.
- (C) Crude fibre does not dissolve in acid/alkali, and R_f cannot exceed 1.

Final Answer: Crude fibre is acid/alkali-insoluble; $R_f = 0.30$ and $0.70 \Rightarrow$ D

Answer: (D) Go Back to Q142

Q143.

Solution

Concept — Isoschizomers: Two restriction enzymes that recognise the identical DNA sequence are isoschizomers; when they cut differently because of methylation they are sometimes called a methylation-sensitive isoschizomer pair. **Reasoning:** *Msp*I and *Hpa*II both read CCGG. *Hpa*II is blocked when the internal cytosine is methylated, whereas *Msp*I still cuts. Sharing one recognition site is the defining feature of isoschizomers, so option (D) is correct. **Why the other options are wrong:**

- (A) Homing endonucleases recognise very long, rare sites.
- (B) Exonucleases chew from DNA ends; these are site-specific endonucleases.

- (C) Nicking enzymes cut only one strand; that is not what defines this pair.

Final Answer: Same-site enzymes are isoschizomers $\Rightarrow$ D

Answer: (D) Go Back to Q143

Q144.

Solution

Concept — Insulin glargine (pI-shift analogue): Adding two arginines to the B-chain raises the isoelectric point of the molecule towards neutral pH, so it is fully soluble in the acidic vial but comes out of solution after injection. **Reasoning:**

Glargine is formulated at pH 4 where it is clear and soluble. On injection into tissue at pH 7.4 it forms fine microprecipitates that slowly redissolve, giving a flat, peakless 24-hour release. The A21-glycine substitution stabilises the molecule at the acidic pH. Hence option (A). **Why the other options are wrong:**

- (B) Albumin binding via a fatty acid is the mechanism of insulin detemir, not glargine.
- (C) Rapid proteolysis would shorten, not prolong, the action.
- (D) Glargine is a full receptor agonist, not an antagonist.

Final Answer: Acid-soluble, precipitates at physiological pH for slow release $\Rightarrow$ A

Answer: (A) Go Back to Q144

Q145.

Solution

Concept — Electroporation: A short high-voltage pulse transiently opens pores in the cell membrane so that plasmid DNA can enter; it is a physical (not biological) transformation method. **Reasoning:** The description, a high-voltage electrical

pulse making the membrane permeable, is exactly electroporation. It gives very high transformation efficiencies and works for many cell types, so option (B) is correct. **Why the other options are wrong:**

- (A) Transduction uses a bacteriophage to carry DNA, no electric pulse.
- (C) Conjugation transfers DNA cell-to-cell through a pilus.
- (D) Heat-shock of $CaCl_2$-treated cells is a chemical method, not an electrical pulse.

Final Answer: A high-voltage pulse driving DNA in is electroporation $\Rightarrow$ B

Answer: (B) Go Back to Q145

Q146.

Solution

Concept — *L*-asparaginase in ALL: Normal cells make their own asparagine, but leukaemic lymphoblasts lack asparagine synthetase and depend on the blood supply. Depleting blood asparagine selectively starves them. **Reasoning:** The recombinant enzyme hydrolyses circulating *L*-asparagine to aspartate and ammonia. Leukaemic cells, unable to synthesise asparagine, cannot make protein and die, while most normal cells are spared. This is option (C). **Why the other options are wrong:**

- (A) DNA cross-linking is the action of alkylating agents.
- (B) Dihydrofolate-reductase inhibition is methotrexate's mechanism.
- (D) Microtubule blockade describes vinca alkaloids/taxanes.

Final Answer: It hydrolyses circulating asparagine, starving the lymphoblasts $\Rightarrow$ C

Answer: (C) Go Back to Q146

Q147.

Solution

Concept — Choosing an expression host: Complex glycoproteins (like antibodies) need human-type N-glycosylation that prokaryotes cannot perform, so mammalian cells are used. **Reasoning:** Chinese-hamster-ovary (CHO) cells fold, assemble and glycosylate antibodies in a near-human pattern, giving the correct effector functions and long half-life. Bacteria do not glycosylate at all. Therefore option (D). **Why the other options are wrong:**

- (A) *E. coli* does not perform human N-glycosylation.
- (B) *Bacillus subtilis* likewise lacks the machinery and the question needs glycosylation.
- (C) A host that lacks glycosylation machinery cannot supply the required glycans.

Final Answer: Glycosylated antibodies need a mammalian (CHO) host $\Rightarrow$ D

Answer: (D) Go Back to Q147

Q148.

Solution

Concept — Primary screening: Primary screening rapidly sifts a very large number of isolates to find the few that show the desired activity (here antimicrobial), without yet quantifying or characterising them. **Reasoning:** The aim at this first stage is simply to pick out promising producers worth carrying forward; structure determination, scale-up and clinical work all come much later. Hence option (A). **Why the other options are wrong:**

- (B) Structure elucidation is a later chemical step.
- (C) Scale-up to a production fermenter is downstream of screening.
- (D) Clinical trials are the final development stage, not screening.

Final Answer: Primary screening detects isolates with any useful activity $\Rightarrow$ A

Answer: (A) Go Back to Q148

Q149.

Solution

Concept — Citric acid fermentation: The dominant industrial process uses the mould *Aspergillus niger*, with iron and manganese kept low to maximise acid accumulation. **Reasoning:** *A. niger* over-produces citric acid when trace metals are limited and pH is low, which suppresses the rest of the TCA cycle. It is by far the most-used organism, so option (B) is correct. **Why the other options are wrong:**

- (A) *Lactobacillus* makes lactic acid.
- (C) *Acetobacter* makes acetic acid (vinegar).
- (D) *Clostridium acetobutylicum* makes acetone/butanol.

Final Answer: Citric acid is made by *Aspergillus niger* $\Rightarrow$ B

Answer: (B) Go Back to Q149

Q150.

Solution

Concept — Vitamin B_{12} production: Cobalamin is too complex to synthesise chemically and is obtained by microbial fermentation using selected bacteria. **Reasoning:** Industrially, *Pseudomonas denitrificans* and *Propionibacterium* species are grown (often with cobalt and a precursor) to accumulate B_{12}, which is then extracted and converted to cyanocobalamin. This is option (C). **Why the other options are wrong:**

- (A) Plants do not make B_{12}.
- (B) Yeast extract is not the commercial source, and B_{12} is not a simple primary metabolite of yeast.
- (D) Chemical reduction of cobalt salts does not build the corrin ring.

Final Answer: B_{12} comes from bacteria such as *Pseudomonas/Propionibacterium* $\Rightarrow$ C

Answer: (C) Go Back to Q150

Q151.

Solution

Concept — Reading K_m from a Michaelis–Menten plot: K_m is the substrate concentration at which v equals one-half of V_{max}. **Reasoning:** Here V_{max} = 60 μmol min^{-1}, so half of V_{max} is 30 μmol min^{-1}. The dashed line shows that v = 30 is reached at [S] = 2 mM. By definition that [S] is K_m, giving K_m = 2 mM, option (A). **Why the other options are wrong:**

- (B) 60 is V_{max} in velocity units, not a concentration.
- (C) 30 is the half-velocity, not the substrate concentration.
- (D) 4 mM is not where the half-maximal velocity is reached.

Final Answer: The [S] at half V_{max} is K_m = 2 mM $\Rightarrow$ A

Answer: (A) Go Back to Q151

Q152.

Solution

Concept — Allosteric feedback inhibition: In many biosynthetic pathways the end product binds a separate regulatory (allosteric) site on the first committed enzyme and lowers its activity, conserving resources when product is plentiful. **Reasoning:** The product binding away from the active site to switch off the first enzyme is the classic definition of end-product (feedback) inhibition acting allosterically, option (B). It is reversible and self-regulating. **Why the other options are wrong:**

- (A) Competitive inhibition acts at the active site, not a separate regulatory site.
- (C) The product does not form an irreversible covalent bond.
- (D) Inducing new enzyme is transcriptional control, not direct inhibition of activity.

Final Answer: End product acting at a regulatory site is allosteric feedback inhibition $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q152

Q153.

Solution

Concept — Classical complement pathway: The classical pathway is antibody-dependent: C1q recognises the Fc of IgG or IgM that is already bound to antigen, triggering the cascade. **Reasoning:** Binding of C1q to antigen–antibody complexes activates C1r and C1s, which cleave C4 and C2 to form the C3 convertase. This antibody requirement distinguishes the classical pathway, so option (D) is correct. **Why the other options are wrong:**

- (A) Spontaneous C3 hydrolysis initiates the alternative pathway.
- (B) Mannose-binding lectin starts the lectin pathway.
- (C) LPS does not directly cleave C5; that is not the classical trigger.

Final Answer: C1q binding antigen–antibody complexes starts the classical pathway $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q153

Q154.

Solution

Concept — Etanercept (TNF-receptor–Fc fusion): Fusing the soluble TNF receptor to an IgG1 Fc creates a dimeric "decoy" that binds and neutralises TNF-α while gaining the long half-life of an antibody. **Reasoning:** The Fc portion makes the molecule dimerise and recycle through the neonatal Fc receptor, greatly extending its plasma half-life, and the receptor arms soak up circulating TNF-α, reducing inflammation in rheumatoid arthritis. This is option (C). **Why the other options are wrong:**

- (A) Etanercept antagonises (mops up) TNF, it is not an agonist.
- (B) Fc fusion does not confer free blood–brain-barrier passage.
- (D) As a glycosylated fusion protein it is made in mammalian cells, not restricted to *E. coli*.

Final Answer: Fc fusion gives a long-lived dimeric TNF decoy $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q154

Q155.

Solution

Concept — Adjuvants: An adjuvant (such as an aluminium salt) is added to boost and prolong the immune response to an antigen without being immunogenic itself. **Reasoning:** Alum forms a depot that releases antigen slowly and activates antigen-presenting cells, producing a stronger, longer-lasting antibody response from a smaller antigen dose. Hence option (A). **Why the other options are wrong:**

- (B) The adjuvant is not the antigen.
- (C) Alum does not sterilise the vial.
- (D) Its role is immunological, not merely tonicity adjustment.

Final Answer: Alum enhances and prolongs the response to antigen $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q155

Q156.

Solution

Concept — Monoclonal antibody: A monoclonal antibody is derived from a single B-cell clone, so every molecule has identical antigen-binding sites recognising one epitope. **Reasoning:** The two Fab arms marked X both bind the same CD20 epitope; because the whole preparation recognises a single epitope, rituximab is monoclonal, option (B). This single specificity gives reproducible, targeted B-cell depletion. **Why the other options are wrong:**

- (A) A polyclonal mixture recognises many epitopes; that is the opposite.
- (C) Rituximab targets a cell-surface marker, not a soluble toxin.
- (D) It is an immunoglobulin, not a complement protein.

Final Answer: One epitope (CD20) means rituximab is monoclonal $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q156

Q157.

Solution

Concept — Endospore-forming genera: Among common bacteria, only the Gram-positive genera *Bacillus* (aerobic) and *Clostridium* (anaerobic) form true heat-resistant endospores. **Reasoning:** These spores survive boiling and harsh chemicals, which is why sterilisation cycles are designed against them. The other listed genera are non-spore-formers, so option (D) is correct. **Why the other options are wrong:**

- (A) *Escherichia* and *Salmonella* are Gram-negative non-spore-formers.
- (B) *Staphylococcus* and *Streptococcus* do not form spores.
- (C) *Pseudomonas* and *Vibrio* are non-spore-forming Gram-negatives.

Final Answer: True endospores are formed by *Bacillus* and *Clostridium* $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q157

Q158.

Solution

Concept — Death/decline phase: In a closed batch culture the four phases are lag (I), log (II), stationary (III) and death/decline (IV). In phase IV viable counts fall, often logarithmically, as nutrients run out and toxins accumulate. **Reasoning:**

The falling part of the curve, where death exceeds division, is the death (decline) phase, marked IV. Microbial death is itself a logarithmic process, the basis of D-values in sterilisation. Hence option (A). **Why the other options are wrong:**

- (B) Phase I (lag) shows adaptation with little change in number.
- (C) Phase II (log) is exponential growth, not death.
- (D) Phase III (stationary) has division balancing death, a plateau.

Final Answer: Logarithmic fall in viable count is the death phase, Phase IV $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q158

Q159.

Solution

Concept — F_0 value: F_0 is the equivalent sterilising time, in minutes, at a reference temperature of 121°C that delivers the same lethality as the actual cycle, calculated with a z-value of 10°C. **Reasoning:** F_0 lets one express the lethal effect of a whole heat-up/hold/cool-down cycle as a single number of minutes at 121°C, so cycles run at different temperatures can be compared. A typical target is $F_0 \geq 8$–12 min. This is option (C). **Why the other options are wrong:**

- (A) 160°C in hours refers to dry-heat, not the moist-heat F_0.
- (B) F_0 is a time, not a surviving-spore count.
- (D) Pore size relates to filtration, not heat lethality.

Final Answer: F_0 is the equivalent minutes at 121°C ($z = 10$°C) $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q159

Q160.

Solution

Concept — Bioburden of WFI: The microbial bioburden of pharmaceutical water is measured by filtering a known volume through a membrane and counting the total viable aerobic colonies that grow on the plate. **Reasoning:** Because WFI has very few organisms, a large measured volume is passed through a 0.45 μm membrane, which is incubated on agar; the colony count gives CFU per 100 mL, checked against the not-more-than-10-CFU/100 mL limit. This is option (B). **Why the other options are wrong:**

- (A) The phenol coefficient tests disinfectants, not water bioburden.
- (C) A single-drop Gram stain cannot count low-level viable organisms.
- (D) Conductivity is a chemical-purity test and detects no microbes.

Final Answer: Membrane filtration with a total viable plate count gives the bioburden $\Rightarrow$ B

Answer: (B) Go Back to Q160

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	D	2	C	3	B	4	C	5	D
6	A	7	B	8	C	9	D	10	A
11	B	12	C	13	D	14	A	15	B
16	C	17	A	18	D	19	B	20	C
21	A	22	D	23	B	24	C	25	A
26	D	27	B	28	C	29	A	30	B
31	D	32	A	33	A	34	C	35	C
36	A	37	B	38	D	39	A	40	B
41	C	42	D	43	A	44	C	45	B
46	A	47	D	48	C	49	A	50	B
51	C	52	D	53	A	54	B	55	C
56	D	57	A	58	B	59	C	60	A
61	D	62	B	63	C	64	A	65	B
66	D	67	A	68	C	69	C	70	A
71	D	72	B	73	A	74	C	75	D
76	B	77	A	78	C	79	D	80	B
81	C	82	D	83	A	84	B	85	D
86	A	87	C	88	B	89	A	90	D
91	B	92	C	93	A	94	D	95	B
96	A	97	C	98	D	99	C	100	B
101	D	102	A	103	B	104	C	105	A
106	D	107	B	108	C	109	D	110	A
111	C	112	B	113	A	114	D	115	B
116	A	117	C	118	D	119	C	120	B
121	A	122	C	123	B	124	A	125	C
126	D	127	A	128	B	129	C	130	D
131	A	132	B	133	C	134	D	135	A
136	B	137	C	138	D	139	A	140	B
141	C	142	D	143	D	144	A	145	B
146	C	147	D	148	A	149	B	150	C
151	A	152	B	153	D	154	C	155	A
156	B	157	D	158	A	159	C	160	B