NIPER JEE Pharmacy Subjects

Sample Paper – 10

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. Calcium carbonate dissolves as $CaCO_3 \rightleftharpoons Ca^{2+} + CO_3^{2-}$. If its molar solubility in pure water at 25°C is 3.0×10^{-5} mol/L, the solubility product K_{sp} is:

(A) 3.0×10^{-5}

(B) 6.0×10^{-5}

(C) 9.0×10^{-10}

(D) 2.7×10^{-14}

Q2. A drug is shaken with equal volumes of isopropyl myristate and water. At equilibrium 120 mg is recovered from the oil phase and 30 mg from the water phase. The oil/water partition coefficient (P) of the drug is:

(A) 0.25

(B) 4.0

(C) 0.40

(D) 40

Q3. An emulsifier system is blended from 35% Span 60 (HLB $= 4.7$) and 65% Tween 60 (HLB $= 14.9$). The required HLB of the resulting blend is approximately:

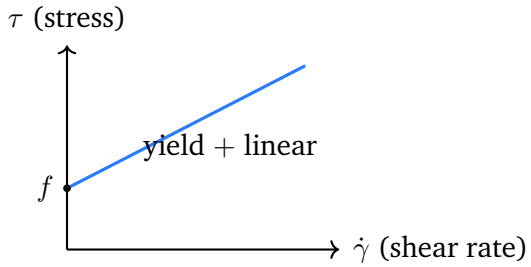
(A) 4.7

(B) 9.8

(C) 14.9

(D) 11.3

Q4. The flow curve below is a straight line that intercepts the shear-stress axis at a positive value f before rising linearly. Which flow behaviour does it represent?



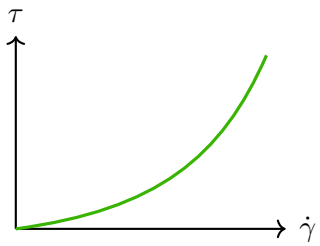
(A) Plastic (Bingham) flow with a yield value

(B) Newtonian flow

(C) Pseudoplastic (shear-thinning) flow

(D) Dilatant (shear-thickening) flow

Q5. The single flow curve below bows toward the shear-rate axis: as the shear rate $\dot{\gamma}$ rises, progressively more stress is needed and the apparent viscosity increases. This behaviour is termed:



(A) Newtonian flow

(B) Pseudoplastic (shear-thinning) flow

(C) Dilatant (shear-thickening) flow

(D) Thixotropic flow

Q6. A liquid has a dynamic viscosity of 1.2 poise and a density of 1.5 g/cm^3. Its kinematic viscosity (in stokes) is:

(A) 1.80

(B) 1.50

(C) 1.25

(D) 0.80

Q7. A granule blend has a bulk density of 0.52 g/mL and a tapped density of 0.65 g/mL. Carr's compressibility index of this powder is:

(A) 25%

(B) 20%

(C) 13%

(D) 35%

Q8. For the same blend (bulk density 0.52 g/mL, tapped density 0.65 g/mL), the Hausner ratio is:

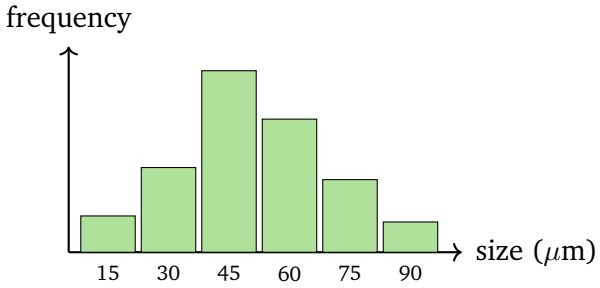
(A) 1.25

(B) 0.80

(C) 1.13

(D) 1.45

Q9. The number-frequency histogram below describes a sieved powder. The modal (most frequent) size class lies in which interval (in μm)?



(A) 15 – 30

(B) 30 – 45

(C) 45 – 60

(D) 75 – 90

Q10. A buffer is prepared from a weak acid ($pK_a = 5.30$) and its sodium salt such that the salt-to-acid concentration ratio is $10 : 1$. The pH of this buffer is:

(A) 4.30

(B) 5.30

(C) 5.40

(D) 6.30

Q11. The sodium-chloride equivalent (E) of a drug is best defined as:

(A) the weight of sodium chloride that produces the same osmotic (colligative) effect as 1 g of the drug

(B) the weight of the drug equivalent to 1 g of sodium chloride by mass

(C) the freezing-point depression caused by a 1% solution of the drug

(D) the molar concentration of the drug equal to isotonic saline

Q12. For two 1:1 drug–ligand complexes, complex M has a stability (formation) constant $K = 5 \times 10^4$ and complex N has $K = 5 \times 10^2$. Which statement is correct?

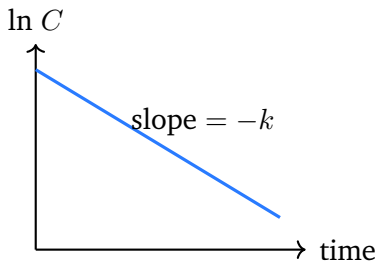
(A) N is the more stable complex because it dissociates faster

(B) M is the more stable complex, being 100 times less dissociated

(C) Both complexes are equally stable

(D) Stability cannot be compared from formation constants

Q13. In the plot below the natural logarithm of the remaining drug concentration ($\ln C$) decreases linearly with time. This indicates that the degradation follows:



(A) zero-order kinetics

(B) second-order kinetics

(C) first-order kinetics

(D) pseudo-zero-order kinetics

Q14. A drug in solution degrades by first-order kinetics with a rate constant $k = 0.0105$ month^{-1}. Its shelf-life t_{90} (time for 10% loss, $t_{90} = 0.105/k$) is approximately:

(A) 5 months

(B) 33 months

(C) 66 months

(D) 10 months

Q15. In an accelerated stability study, the rate of degradation is plotted as $\ln k$ against $1/T$ (the Arrhenius plot). The slope of this straight line equals:

(A) $-E_a/R$

(B) $+E_a/R$

(C) $-R/E_a$

(D) $\ln A$

Q16. The Langmuir adsorption isotherm $\dfrac{x}{m} = \dfrac{abC}{1+bC}$ can be linearised by plotting which of the following?

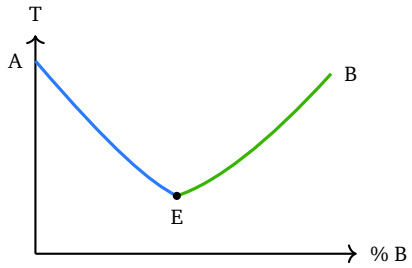
(A) $\log(x/m)$ versus $\log C$

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

(C) x/m versus C

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

Q17. The temperature–composition diagram below for two solids shows two descending liquidus curves meeting at the lowest point E. The point E represents the:



(A) melting point of pure A

(B) melting point of pure B

(C) eutectic point (lowest melting mixture)

(D) point of maximum solubility

Q18. According to the Noyes–Whitney equation $dC/dt = \dfrac{DA}{h}(C_s - C)$, increasing the effective surface area A of a drug powder by micronisation will:

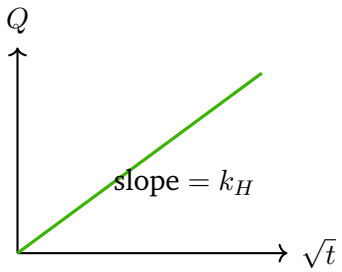
(A) decrease the dissolution rate

(B) have no effect on the dissolution rate

(C) only change the solubility C_s

(D) increase the dissolution rate

Q19. For a matrix tablet, the cumulative amount of drug released Q is plotted against the square root of time and a straight line through the origin is obtained, as shown. This release follows:



(A) zero-order release

(B) Higuchi (diffusion-controlled) release

(C) first-order release

(D) Hixson–Crowell release

Q20. In the Korsmeyer–Peppas model ($M_t/M_\infty = kt^n$), drug release from a swellable cylindrical (matrix) device is governed by anomalous (non-Fickian) transport when the exponent n lies in the range:

(A) $n = 0.45$ (Fickian only)

(B) $n \geq 1.0$ (Case-II only)

(C) $0.45 < n < 0.89$

(D) $n < 0.30$

Q21. A drug shows high aqueous solubility but low intestinal permeability. According to the Biopharmaceutics Classification System (BCS), it belongs to:

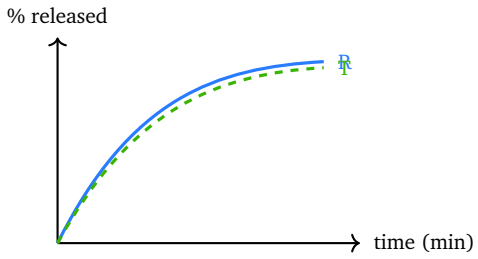
(A) Class I

(B) Class II

(C) Class IV

(D) Class III

Q22. Two dissolution profiles (test T and reference R) are shown below; they nearly overlap throughout. The similarity factor f_2 for such profiles would most likely fall in the range that indicates the profiles are:



(A) similar (f_2 between 50 and 100)

(B) dissimilar ($f_2 < 50$)

(C) identical only if $f_2 = 0$

(D) not comparable by f_2

Q23. Acacia mucilage incorporated into a wet-granulation tablet formulation functions chiefly as a:

(A) disintegrant

(B) binder (adhesive)

(C) glidant

(D) lubricant

Q24. For uncoated tablets of average weight 130 mg, the IP/USP weight-variation tolerance limit is:

(A) $\pm 5\%$

(B) $\pm 15\%$

(C) $\pm 7.5\%$

(D) $\pm 10\%$

Q25. Among the standard hard-gelatin capsule shells used for human oral products, the size with the *smallest* fill capacity is:

(A) size 0

(B) size 1

(C) size 00

(D) size 5

Q26. A drug has a displacement value of 4 in cocoa butter. To prepare 6 suppositories, each containing 0.4 g of drug in a mould of 2 g capacity, the amount of cocoa-butter base required is:

(A) 11.4 g

(B) 12.0 g

(C) 9.6 g

(D) 10.8 g

Q27. An unknown emulsion is tested by placing two electrodes connected to a small lamp into it; the lamp fails to glow (no appreciable current). The emulsion is most likely:

(A) oil-in-water (o/w)

(B) water-in-oil (w/o)

(C) a multiple w/o/w emulsion

(D) a microemulsion

Q28. In a pharmaceutical suspension the final sediment occupies 16 mL of an original total suspension volume of 40 mL. The sedimentation volume (F) is:

(A) 0.16

(B) 0.24

(C) 0.40

(D) 2.50

Q29. Chlorobutanol is most commonly included in a multidose ophthalmic solution to act as a:

(A) tonicity-adjusting agent

(B) viscosity enhancer

(C) buffering agent

(D) antimicrobial preservative

Q30. Hydrophilic petrolatum, which contains wool fat and cholesterol and can take up water to form a w/o emulsion, is best classified as which type of ointment base?

(A) absorption (emulsifiable) base

(B) water-soluble base

(C) oleaginous (hydrocarbon) base

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

Q31. A colloid mill reduces particle size and produces fine dispersions predominantly by the action of:

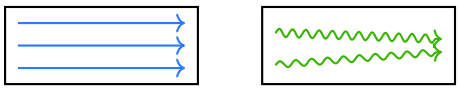
(A) impact and attrition between balls

(B) intense shear in a narrow rotor–stator gap

(C) compression between rollers

(D) cutting by sharp rotating knives

Q32. The two pipe-flow sketches below contrast smooth parallel streamlines (i) with chaotic swirling eddies (ii). Pattern (ii) corresponds to flow for which the Reynolds number is:



(i) laminar (ii) turbulent

(A) below 2000

(B) exactly 2100

(C) above 4000

(D) independent of Reynolds number

Q33. During the hot-air drying of a wet granulation, the period during which the granule surface stays saturated with liquid and the drying rate remains constant is called the:

(A) falling-rate period

(B) equilibrium-moisture period

(C) induction (warm-up) period

(D) constant-rate period

Q34. Niosomes, used as a vesicular drug-delivery carrier and an alternative to liposomes, are best described as vesicles formed from:

(A) non-ionic surfactants (with cholesterol)

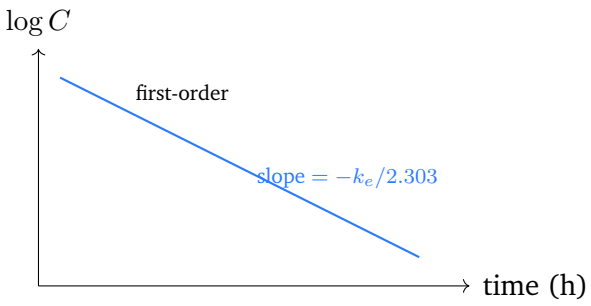
(B) phospholipid bilayers only

(C) biodegradable PLGA polymer shells

(D) cross-linked albumin microspheres

Part B: Pharmacology & Toxicology

Q35. The semi-log plot below shows log plasma concentration falling linearly with time for a drug cleared by a single first-order process. If the slope corresponds to an elimination rate constant $k_e = 0.231\,\text{h}^{-1}$, the elimination half-life is closest to:



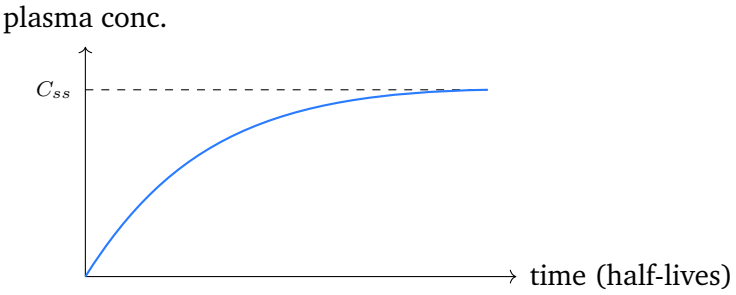
(A) 0.23 h

(B) 1.5 h

(C) 3 h

(D) 7 h

Q36. During a constant-rate IV infusion the plasma concentration rises towards a plateau (steady state) as shown. The fraction of the eventual steady-state concentration attained after a given number of elimination half-lives follows the plateau principle. After how many half-lives is the plasma level approximately 94% of steady state?



(A) 2 half-lives

(B) 4 half-lives

(C) 7 half-lives

(D) 10 half-lives

Q37. A drug with a high hepatic extraction ratio (E $\approx$ 0.9) is given orally. Compared with an IV dose, its oral bioavailability and the main determinant of its hepatic clearance are:

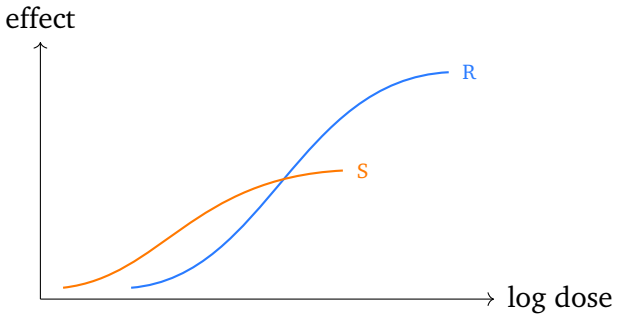
(A) Low oral bioavailability because of extensive first-pass metabolism; its hepatic clearance is flow-limited (depends mainly on hepatic blood flow)

(B) High oral bioavailability; clearance is capacity-limited and independent of blood flow

(C) Bioavailability is unaffected by extraction ratio

(D) Clearance equals the glomerular filtration rate

Q38. On the graded log dose-response curves below, drug R and drug S act at the same receptor. R reaches a higher plateau than S even at saturating doses, while S lies further left. Which property pair is correctly read from the curves?



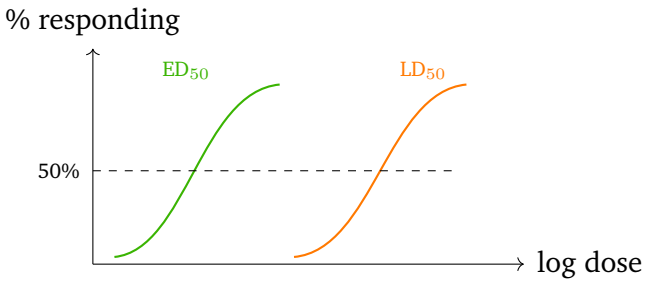
(A) R is more potent and S has greater efficacy

(B) Both have identical efficacy

(C) R is a partial agonist

(D) S is more potent (lower ED_{50}) but R has greater efficacy (higher E_{max}), so S behaves as a partial agonist relative to R

Q39. The quantal curves below give the median effective dose and median lethal dose of a drug. For $ED_{50} = 10$ mg and $LD_{50} = 400$ mg, the therapeutic index (TI) is:



(A) 0.025

(B) 40

(C) 4000

(D) 410

Q40. Which statement about drug metabolism is CORRECT?

(A) Phase I reactions (oxidation, reduction, hydrolysis), often by cytochrome P450, introduce or unmask a functional group, while Phase II reactions conjugate that group to form a more water-soluble product

(B) Phase II conjugation reactions always precede Phase I oxidation

(C) Glucuronidation generally produces a more lipophilic, active metabolite

(D) Cytochrome P450 enzymes catalyse only conjugation reactions

Q41. A patient stabilised on a CYP3A4 substrate is started on a potent enzyme inducer. The expected consequence for the substrate drug is:

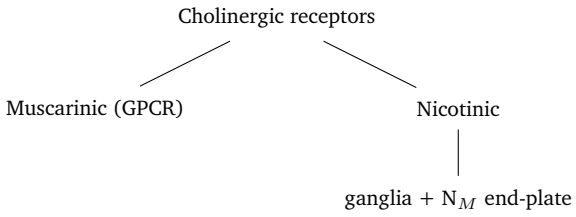
(A) An immediate rise in plasma concentration and toxicity within minutes

(B) No change because induction affects only Phase II

(C) Conversion of the substrate into a prodrug

(D) Increased metabolism, lower plasma levels and reduced therapeutic effect over days to weeks

Q42. In the cholinergic receptor tree shown, the branch ending in “ganglia + skeletal muscle end-plate, ionotropic” identifies which receptor class?



(A) Muscarinic M_2, G_i-coupled

(B) Muscarinic M_3, G_q-coupled

(C) Adrenergic β_2 receptor

(D) Nicotinic receptors, which are ligand-gated cation channels

Q43. Which β_1-selective blocker additionally causes vasodilation through endothelial nitric-oxide release, distinguishing it from older cardioselective agents?

(A) Sotalol

(B) Nebivolol

(C) Pindolol

(D) Carteolol

Q44. For an elderly patient with overactive bladder in whom cognitive side effects must be minimised, which antimuscarinic is relatively M_3-selective and largely peripherally acting?

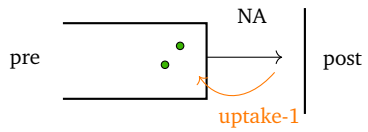
(A) Oxybutynin (immediate-release)

(B) Benztropine

(C) Darifenacin

(D) Dicyclomine

Q45. At the noradrenergic varicosity depicted, cocaine and the tricyclic-type uptake blockers potentiate sympathetic tone mainly by:



(A) Blocking neuronal reuptake (uptake-1) of noradrenaline, raising its synaptic concentration

(B) Inhibiting tyrosine hydroxylase

(C) Acting as direct α_1 agonists on the postsynaptic membrane

(D) Blocking vesicular storage like reserpine

Q46. Which α_1-adrenergic antagonist is highly selective for the α_{1A} subtype found in the prostate, giving prostatic relaxation in benign prostatic hyperplasia with minimal postural hypotension?

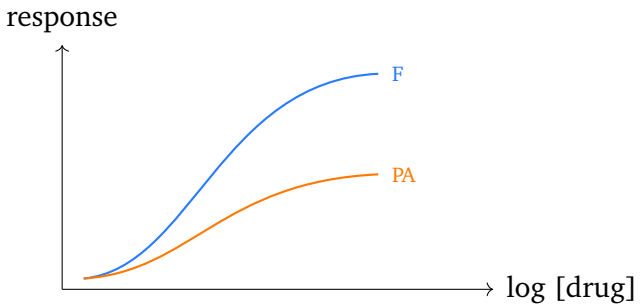
(A) Doxazosin

(B) Phenoxybenzamine

(C) Yohimbine

(D) Silodosin

Q47. On the dose-response curves below, the full agonist F reaches the system maximum while the partial agonist (curve labelled PA) plateaus below it even at saturating dose. Aripiprazole behaves like curve PA at the dopamine D_2 receptor. Aripiprazole is mechanistically distinct from haloperidol because aripiprazole acts as a:



(A) Dopamine D_2 *partial* agonist (a "dopamine system stabiliser"), giving antipsychotic effect with a lower liability for marked hyperprolactinaemia

(B) Pure irreversible D_2 antagonist

(C) Selective serotonin reuptake inhibitor

(D) GABA-A positive allosteric modulator

Q48. Which antidepressant works mainly by inhibiting reuptake of noradrenaline and dopamine (not serotonin), lacks sexual side effects and is also licensed for smoking cessation?

(A) Mirtazapine

(B) Bupropion

(C) Trazodone

(D) Vortioxetine

Q49. Vigabatrin raises brain GABA levels by:

(A) Blocking voltage-gated sodium channels

(B) Antagonising NMDA receptors

(C) Irreversibly inhibiting GABA-transaminase (GABA-T), the enzyme that degrades GABA

(D) Binding the $\alpha_2\delta$ subunit of calcium channels

Q50. Loperamide controls diarrhoea with negligible central or analgesic action because it:

(A) Is a μ-opioid antagonist

(B) Stimulates intestinal motility through 5-HT_4 receptors

(C) Is an osmotic laxative

(D) Is a μ-opioid agonist that acts on gut myenteric receptors but is a P-glycoprotein substrate poorly entering the CNS at therapeutic doses

Q51. Ketamine produces dissociative anaesthesia and analgesia primarily by:

(A) Potentiating GABA-A chloride currents like propofol

(B) Blocking voltage-gated sodium channels

(C) Activating μ-opioid receptors

(D) Non-competitive blockade of the NMDA glutamate receptor channel

Q52. Suvorexant promotes sleep through a mechanism different from benzodiazepines and Z-drugs, namely:

(A) Agonism at melatonin MT_1/MT_2 receptors

(B) Antagonism of orexin (hypocretin) OX_1/OX_2 receptors, reducing wakefulness drive

(C) Increasing the duration of chloride channel opening

(D) Histamine H_1 agonism

Q53. Rizatriptan aborts an acute migraine attack chiefly by acting as an agonist at:

(A) Dopamine D_2 receptors

(B) μ-opioid receptors

(C) 5-$HT_{1B/1D}$ receptors, causing cranial vasoconstriction and inhibiting neuropeptide release from trigeminal nerves

(D) GABA-B receptors

Q54. Ivabradine lowers heart rate in chronic stable angina and heart failure without negative inotropy because it:

(A) Selectively inhibits the hyperpolarisation-activated "funny" current (I_f) in the sinoatrial node, slowing diastolic depolarisation

(B) Blocks β_1-adrenergic receptors

(C) Blocks L-type calcium channels in cardiac muscle

(D) Inhibits Na^+/K^+-ATPase like digoxin

Q55. Olmesartan lowers blood pressure and, unlike enalapril, does not characteristically cause a dry cough because it:

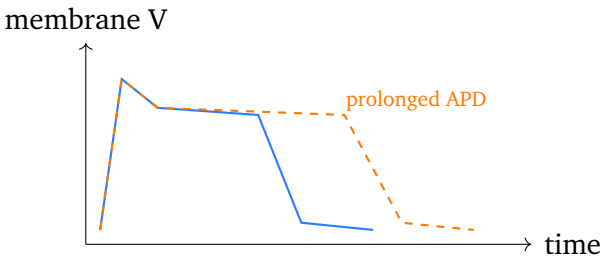
(A) Inhibits angiotensin-converting enzyme

(B) Selectively blocks angiotensin II AT_1 receptors, so bradykinin is not accumulated

(C) Is a direct renin inhibitor

(D) Blocks aldosterone receptors

Q56. The cardiac action potential below shows a control trace (solid) and a Class III drug effect (dashed) that lengthens phase 2/3 and so prolongs the action-potential duration. Dofetilide, used to maintain sinus rhythm in atrial fibrillation, produces this prolongation by:



(A) Blocking fast sodium channels (Class I)

(B) β-adrenergic blockade (Class II)

(C) L-type calcium channel blockade (Class IV)

(D) Selective blockade of the rapid delayed-rectifier potassium current (I_{Kr}), a pure Class III action that lengthens repolarisation

Q57. Eplerenone is preferred over spironolactone in some heart-failure patients because eplerenone:

(A) Is a loop diuretic acting on the thick ascending limb

(B) Blocks epithelial sodium channels directly like amiloride

(C) Is a selective mineralocorticoid (aldosterone) receptor antagonist with far less affinity for androgen/progesterone receptors, so it rarely causes gynaecomastia

(D) Acts on the renin-angiotensin enzyme directly

Q58. Ticagrelor prevents arterial thrombosis after acute coronary syndromes by:

(A) Reversibly inhibiting the platelet $P2Y_{12}$ ADP receptor, blocking ADP-induced platelet activation and aggregation

(B) Irreversibly acetylating cyclooxygenase like aspirin

(C) Inhibiting glycoprotein IIb/IIIa from the plasma

(D) Activating antithrombin

Q59. Fexofenadine causes little sedation compared with chlorpheniramine because fexofenadine:

(A) Blocks histamine H_2 receptors instead of H_1

(B) Is a second-generation H_1 antagonist that is poorly lipophilic and a P-glycoprotein substrate, so it barely crosses the blood-brain barrier

(C) Is a mast-cell stabiliser with no receptor action

(D) Is a potent muscarinic blocker

Q60. Montelukast is used as add-on prophylaxis in asthma because it:

(A) Antagonises the cysteinyl-leukotriene $CysLT_1$ receptor, reducing leukotriene-mediated bronchoconstriction and inflammation

(B) Inhibits 5-lipoxygenase (the zileuton mechanism)

(C) Blocks histamine H_1 receptors

(D) Is an inhaled β_2-agonist

Q61. Etoricoxib spares the gastric mucosa more than naproxen because etoricoxib:

(A) Selectively inhibits COX-2 (induced at inflammation sites) with little effect on constitutive gastric COX-1-derived protective prostaglandins

(B) Selectively inhibits COX-1

(C) Inhibits lipoxygenase only

(D) Is a prostaglandin analogue

Q62. Misoprostol protects against NSAID-induced peptic ulceration because it:

(A) Is a proton-pump inhibitor

(B) Neutralises gastric acid as an antacid

(C) Is a prostaglandin E_1 analogue that restores the prostaglandin-dependent gastric mucosal defence (mucus, bicarbonate, reduced acid)

(D) Blocks H_2 receptors

Q63. Linezolid is active against MRSA and VRE; its antibacterial action is:

(A) Inhibition of cell-wall transpeptidase

(B) Binding the 23S rRNA of the 50S ribosomal subunit and preventing formation of the functional 70S initiation complex

(C) Inhibition of DNA gyrase

(D) Inhibition of dihydropteroate synthase

Q64. Daptomycin is bactericidal against Gram-positive organisms because it:

(A) Inhibits the 30S ribosomal subunit

(B) Inhibits RNA polymerase

(C) Inserts (calcium-dependently) into the bacterial cell membrane, causing rapid depolarisation and loss of membrane potential

(D) Blocks folate synthesis

Q65. Aztreonam is useful in penicillin-allergic patients with Gram-negative infection because this monobactam:

(A) Inhibits cell-wall synthesis (binds PBP3) but acts essentially only on aerobic Gram-negative bacilli and shows little cross-reactive allergenicity with penicillins

(B) Inhibits the 50S ribosome

(C) Is broadly active against Gram-positive cocci and anaerobes

(D) Is a fluoroquinolone

Q66. Oseltamivir shortens the course of influenza by:

(A) Inhibiting viral reverse transcriptase

(B) Blocking the M2 ion channel

(C) Inhibiting viral neuraminidase, preventing release of new virions from infected cells

(D) Inhibiting viral DNA polymerase

Q67. Liraglutide improves glycaemic control in type-2 diabetes and promotes weight loss because it:

(A) Inhibits SGLT2 in the proximal tubule

(B) Is a GLP-1 receptor agonist (incretin mimetic) that enhances glucose-dependent insulin secretion, suppresses glucagon and slows gastric emptying

(C) Is a sulfonylurea closing K_{ATP} channels

(D) Inhibits intestinal α-glucosidase

Q68. In ethylene-glycol or methanol poisoning, fomepizole is given as an antidote because it:

(A) Chelates the parent alcohol directly

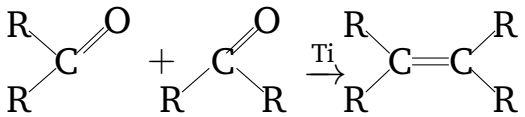
(B) Is a competitive antagonist at GABA-A receptors

(C) Replenishes hepatic glutathione like N-acetylcysteine

(D) Inhibits alcohol dehydrogenase, blocking conversion of the alcohol to its toxic acid metabolites (glycolic/oxalic or formic acid)

Part C: Pharmaceutical & Medicinal Chemistry

Q69. Two molecules of a ketone, on treatment with a low-valent titanium reagent (e.g. $TiCl_3$/Zn), are reductively coupled to give a symmetrical alkene with loss of both oxygen atoms, as shown.



This olefination is the:

(A) Wittig reaction

(B) Tishchenko reaction

(C) McMurry coupling

(D) Reformatsky reaction

Q70. An aldehyde lacking an α-hydrogen, in the presence of an aluminium alkoxide catalyst, undergoes a dimerising disproportionation in which one molecule is oxidised and the other reduced, the two being joined as an ester (e.g. benzaldehyde $\rightarrow$ benzyl benzoate). This is the:

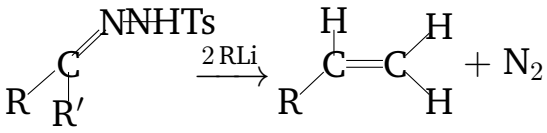
(A) Tishchenko reaction

(B) Cannizzaro reaction

(C) Claisen condensation

(D) Perkin reaction

Q71. A ketone tosylhydrazone, on treatment with two equivalents of a strong organolithium base, eliminates to give the less-substituted alkene with loss of N_2 and the sulfinate, as outlined.



This base-induced olefin synthesis is the:

(A) Bamford–Stevens reaction

(B) Shapiro reaction

(C) Wittig reaction

(D) Horner–Wadsworth–Emmons reaction

Q72. Under thiazolium-salt (NHC) catalysis, an aldehyde is converted into an acyl-anion equivalent that adds in a 1,4-fashion (conjugate addition) to an α,β-unsaturated carbonyl, giving a 1,4-diketone. This umpolung reaction is the:

(A) benzoin condensation

(B) Michael addition

(C) Mannich reaction

(D) Stetter reaction

Q73. In the Corey–Seebach reaction, an aldehyde is first converted to a 1,3-dithiane; deprotonation then gives a nucleophilic carbanion that behaves as a masked acyl anion. After alkylation and hydrolysis of the dithiane, the overall transformation achieves:

(A) umpolung, converting the electrophilic carbonyl carbon into a nucleophile

(B) reduction of the aldehyde to a primary alcohol

(C) oxidation of the aldehyde to a carboxylic acid

(D) a [3,3]-sigmatropic rearrangement

Q74. Treatment of an alkene bearing allylic C–H bonds with *N*-bromosuccinimide (NBS) and a radical initiator, in CCl_4, gives selective substitution at the allylic position rather than addition across the double bond. This radical allylic bromination is the:

(A) Hell–Volhard–Zelinsky reaction

(B) Sandmeyer reaction

(C) Wohl–Ziegler bromination

(D) Appel reaction

Q75. Condensation of a β-arylethylamine with an aldehyde gives an iminium ion that cyclises (intramolecular Mannich-type electrophilic aromatic substitution) onto the ring to form a tetrahydroisoquinoline. This classic alkaloid-forming cyclisation is the:

(A) Bischler–Napieralski reaction

(B) Pictet–Spengler reaction

(C) Skraup synthesis

(D) Combes synthesis

Q76. An elimination that proceeds through a discrete, resonance-stabilised carbanion (conjugate base) intermediate, formed by removal of an acidic β-proton before the leaving group departs, is classified as:

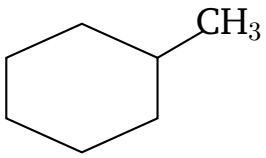
(A) an E2 (concerted) elimination

(B) an S_N1 reaction

(C) an E1 elimination via a carbocation

(D) an E1cb elimination

Q77. The chair conformation of methylcyclohexane drawn below places the methyl group in the position that minimises 1,3-diaxial steric strain.



In this most stable chair, the methyl group occupies which type of position?

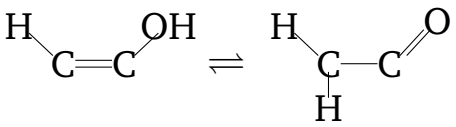
(A) equatorial

(B) axial

(C) bridgehead

(D) anti-periplanar to the ring oxygen

Q78. The two structures below are related by migration of a proton and shift of a double bond, interconverting a carbonyl form and its hydroxyl-alkene form.



This type of constitutional-isomer equilibrium is called:

(A) resonance

(B) conformational isomerism

(C) keto–enol tautomerism

(D) optical isomerism

Q79. In nucleophilic aromatic substitution (S_NAr) of an aryl halide, the rate is greatly accelerated when the ring bears, *ortho* or *para* to the leaving group:

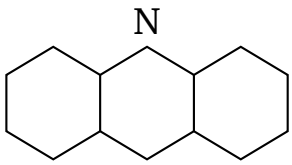
(A) strong electron-donating groups such as –OCH_3

(B) strong electron-withdrawing groups such as –NO_2 that stabilise the Meisenheimer complex

(C) bulky alkyl groups providing steric acceleration

(D) additional halogen atoms acting as activating donors

Q80. The tricyclic, dibenzo-fused, nitrogen-containing aromatic heterocycle drawn below (two benzene rings flanking a central pyridine-type ring) is the parent of several DNA-intercalating dyes and antimalarials.



Identify this heterocyclic system.

(A) carbazole

(B) anthracene

(C) dibenzofuran

(D) acridine

Q81. For the alkene 2-bromo-2-butene, the configuration in which the two higher-CIP-priority groups (the Br on one carbon and the CH_3 on the other) lie on the same side of the double bond is designated:

(A) Z

(B) E

(C) R

(D) meso

Q82. In a Baeyer–Villiger oxidation of an unsymmetrical ketone with a peroxy acid, the oxygen atom is inserted preferentially on the side of the more substituted carbon. The migratory aptitude therefore follows the order:

(A) methyl $>$ primary $>$ secondary $>$ tertiary

(B) all groups migrate equally

(C) tertiary $>$ secondary $>$ primary $>$ methyl

(D) aryl always fails to migrate

Q83. Classical (first-generation) H_1-antihistamines such as diphenhydramine cross the blood–brain barrier and cause sedation chiefly because they are:

(A) highly ionised and water-soluble at physiological pH

(B) relatively lipophilic, uncharged tertiary amines that penetrate the CNS

(C) large peptides actively transported into the brain

(D) irreversible covalent blockers of the H_1 receptor

Q84. The "statin" lipid-lowering drugs (e.g. atorvastatin) act as competitive inhibitors of which biosynthetic enzyme, mimicking its natural substrate?

(A) cyclo-oxygenase (COX)

(B) angiotensin-converting enzyme (ACE)

(C) Na^+/K^+-ATPase

(D) HMG-CoA reductase

Q85. The fluoroquinolone antibacterials (e.g. ciprofloxacin) exert their bactericidal effect primarily by inhibiting:

(A) bacterial DNA gyrase (topoisomerase II) and topoisomerase IV

(B) the 30S ribosomal subunit

(C) dihydropteroate synthase

(D) the β-lactam transpeptidase (PBP)

Q86. Omeprazole and related proton-pump inhibitors are acid-activated prodrugs that, in the acidic parietal-cell canaliculus, rearrange to a reactive sulfenamide which then:

(A) competitively blocks the histamine H_2 receptor

(B) neutralises gastric acid directly as a base

(C) covalently binds cysteine residues of the H^+/K^+-ATPase, irreversibly inhibiting acid secretion

(D) chelates calcium in the gastric lumen

Q87. Trimethoprim is frequently combined with sulfamethoxazole (co-trimoxazole) because the two agents produce a synergistic "sequential blockade" of the folate pathway. Trimethoprim specifically inhibits:

(A) dihydropteroate synthase

(B) bacterial dihydrofolate reductase

(C) thymidylate synthase

(D) DNA polymerase

Q88. According to the pH–partition hypothesis, a weakly acidic drug ($pK_a \approx 4$) is best absorbed across the lipid membrane of the stomach (pH $\approx$ 2) because at that pH it exists predominantly in the:

(A) fully ionised, charged form

(B) zwitterionic form

(C) salt form with chloride

(D) un-ionised (lipid-soluble) form

Q89. Catecholamines such as adrenaline have a very short duration of action when given orally largely because the catechol (3,4-dihydroxyphenyl) moiety is rapidly methylated by:

(A) catechol-*O*-methyltransferase (COMT)

(B) acetylcholinesterase

(C) carbonic anhydrase

(D) xanthine oxidase

Q90. Nitrogen-mustard alkylating agents (e.g. cyclophosphamide) exert their cytotoxic action by forming a reactive aziridinium ion that alkylates and cross-links which biomolecular site?

(A) the catalytic serine of a protease

(B) membrane phospholipids

(C) the N-7 position of guanine bases in DNA

(D) the heme iron of cytochrome P450

Q91. A drug that binds a receptor with high affinity but produces only a sub-maximal response even at full occupancy, and that can antagonise a full agonist, is best described as a:

(A) full agonist

(B) partial agonist

(C) irreversible (non-competitive) antagonist

(D) inverse agonist with full negative efficacy

Q92. Lipinski's "rule of five" predicts poor oral absorption when a candidate molecule violates more than one of the limits. Which of the following is NOT one of the rule-of-five criteria?

(A) molecular weight ≤ 500

(B) $\log P \leq 5$

(C) no more than 5 hydrogen-bond donors

(D) melting point $\leq 200\,^{\circ}\text{C}$

Q93. Captopril inhibits angiotensin-converting enzyme (a zinc metallopeptidase). The functional group on captopril that coordinates the active-site zinc ion is the:

(A) sulfhydryl (–SH) group

(B) aromatic phenol

(C) quaternary ammonium centre

(D) tetrazole ring

Q94. Selective COX-2 inhibitors (coxibs, e.g. celecoxib) were designed to spare gastric COX-1 by exploiting a structural difference in the COX-2 active site, namely:

(A) a much smaller, fully blocked binding channel

(B) the absence of any catalytic tyrosine

(C) a larger side-pocket (Val523 in place of Ile) accommodating a bulky aryl-sulfonamide group

(D) a covalent acetylation site identical to that of aspirin

Q95. A drug exhibiting a very large apparent volume of distribution ($V_d \gg$ total body water) is best interpreted as being:

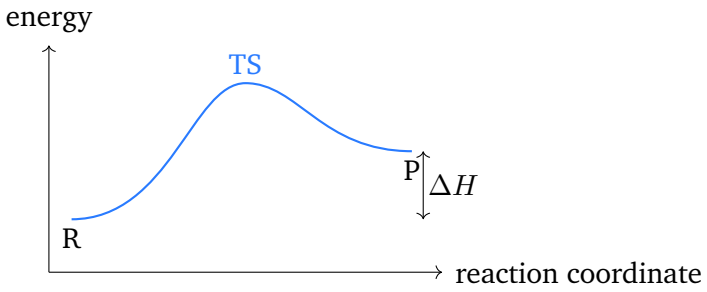
(A) confined entirely to the plasma compartment

(B) extensively bound to, and sequestered in, peripheral tissues

(C) completely un-absorbed from the gut

(D) eliminated solely by glomerular filtration

Q96. The reaction-coordinate diagram below shows a single-step reaction.



Because the products lie at higher energy than the reactants, this reaction is:

(A) endothermic ($\Delta H > 0$)

(B) exothermic ($\Delta H < 0$)

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

(D) barrierless and spontaneous in both directions

Q97. The anticancer drug cisplatin, *cis*-$[Pt(NH_3)_2Cl_2]$, owes its activity to its specific square-planar geometry. The central platinum ion has which d-electron configuration and geometry?

(A) d^{10}, tetrahedral

(B) d^{6}, octahedral

(C) d^{0}, linear

(D) d^{8}, square-planar

Q98. For an acetate buffer the Henderson–Hasselbalch equation is $pH = pK_a + \log([A^-]/[HA])$. If the concentrations of acetate (A^-) and acetic acid (HA) are equal, the pH of the buffer equals:

(A) $pK_a - 1$

(B) the pK_a of acetic acid

(C) $pK_a + 1$

(D) 7.00 regardless of the pK_a

Part D: Pharmaceutical Analysis & Quality Assurance

Q99. In the Kjeldahl method for the estimation of nitrogen in an organic drug, the digestion step converts the organically bound nitrogen into:

(A) Nitrogen gas (N_2)

(B) Ammonium sulphate, $(NH_4)_2SO_4$

(C) Nitric acid (HNO_3)

(D) Nitrous oxide (N_2O)

Q100. A pharmacopoeial cerimetric assay titrates an iron(II) salt with standard ceric sulphate. Which redox indicator, changing from colourless to purple-violet at the end point, is recommended for this titration?

(A) Starch

(B) Eriochrome Black T

(C) Methyl orange

(D) N-phenylanthranilic acid

Q101. In a complexometric assay an excess of standard EDTA is added to an aluminium-containing antacid, and the unreacted EDTA is then determined by back-titration. Which standard solution is most appropriate as the back-titrant for the residual EDTA?

(A) Sodium thiosulphate

(B) Potassium permanganate

(C) Standard zinc sulphate solution

(D) Silver nitrate

Q102. Sodium carbonate (Na_2CO_3) neutralises two equivalents of acid per mole when titrated to the carbonic-acid end point. What is the normality of a solution that is 0.10 molar in Na_2CO_3?

(A) 0.20 N

(B) 0.10 N

(C) 0.050 N

(D) 0.40 N

Q103. The *iodine value* of a fixed oil, determined by the Wijs method, is a measure of the:

(A) Free fatty acid content of the oil

(B) Degree of unsaturation (number of C=C double bonds)

(C) Quantity of ester groups hydrolysed by alkali

(D) Water content of the oil

Q104. In the diazotisation titration of a primary aromatic amine drug with sodium nitrite, the end point is classically detected using starch–iodide paper as an *external* indicator. The end point is signalled when the paper:

(A) Loses its blue colour completely

(B) Turns from blue to colourless then back to blue

(C) Turns blue, indicating the first slight excess of nitrous acid

(D) Turns red on contact with the titrand

Q105. The *saponification value* of a fat or fixed oil is defined as the number of milligrams of potassium hydroxide required to:

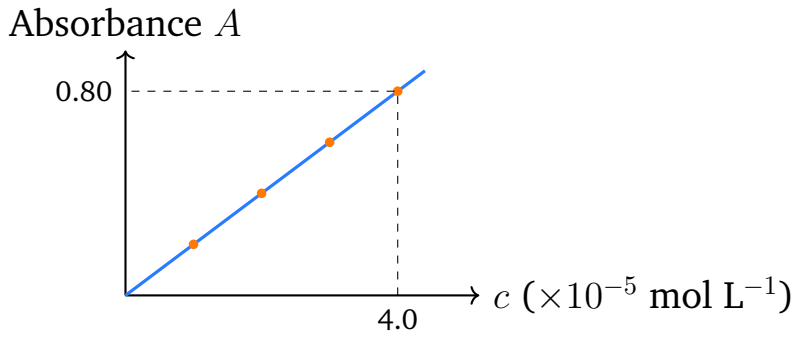
(A) Neutralise the free fatty acids in 1 g of the substance

(B) Add across all the double bonds in 1 g of the substance

(C) Liberate the glycerol from 1 g of the substance

(D) Saponify the esters and neutralise the free acids in 1 g of the substance

Q106. The Beer–Lambert calibration line below was recorded at a drug's λ_{max} in a 1.0 cm cell. From the marked points the absorbance is 0.80 at a concentration of 4.0×10^{-5} mol L^{-1}. The molar absorptivity ε of the drug is closest to:



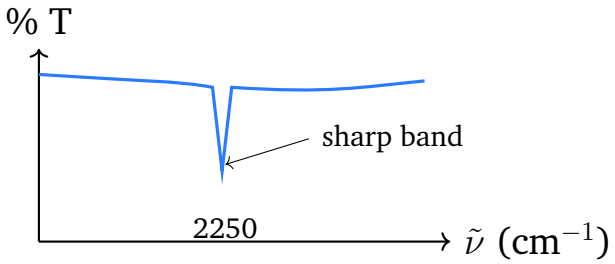
(A) 2.0×10^{4} L mol^{-1} cm^{-1}

(B) 3.2×10^{-5} L mol^{-1} cm^{-1}

(C) 5.0×10^{-5} L mol^{-1} cm^{-1}

(D) 0.80 L mol^{-1} cm^{-1}

Q107. The schematic IR spectrum below shows a sharp band of medium intensity near **2250 cm^{-1}**, in a region where very few other groups absorb. This band is most characteristic of which functional group?



(A) Carbonyl C=O of an ester

(B) Nitrile C≡N stretch

(C) Aromatic C–H stretch

(D) Hydroxyl O–H stretch

Q108. In ^{1}H NMR spectroscopy, a proton that experiences *greater* deshielding (lower electron density around it) will appear:

(A) Upfield, at a smaller chemical shift (δ)

(B) At $\delta = 0$ ppm, coincident with TMS

(C) Downfield, at a larger chemical shift (δ)

(D) As a broadened singlet only

Q109. An organic compound containing a single chlorine atom shows molecular-ion peaks at M and M+2. Because the natural abundances of ^{35}Cl and ^{37}Cl are about 3:1, the expected intensity ratio of the M to M+2 peaks is approximately:

(A) 1:1

(B) 1:3

(C) 9:1

(D) 3:1

Q110. In fluorimetry, the progressive decrease in fluorescence intensity of an analyte caused by collisions with dissolved molecular oxygen (which de-excites the fluorophore without emission) is an example of:

(A) Collisional (dynamic) quenching

(B) A bathochromic shift

(C) The inner-filter effect at high concentration

(D) Phosphorescence

Q111. When a lithium salt is aspirated into the flame of a flame photometer, the characteristic emission used for its estimation appears as a:

(A) Yellow emission near 589 nm

(B) Crimson-red emission near 670 nm

(C) Violet emission near 405 nm

(D) Green emission near 525 nm

Q112. In the AAS determination of calcium, phosphate suppresses the signal by forming a refractory calcium phosphate in the flame. This chemical interference is overcome by adding an excess of a *releasing agent*, most commonly:

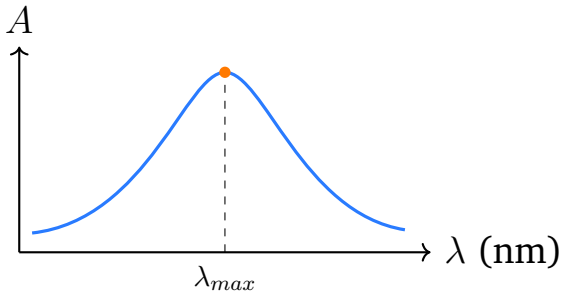
(A) Potassium chloride as an ionisation suppressor

(B) Sodium sulphate

(C) Lanthanum (as lanthanum chloride)

(D) Hydrochloric acid only

Q113. The UV curve below peaks at λ_{max}, where a 0.0020% w/v solution of a drug in a 1.0 cm cell gives an absorbance of 0.60. The specific absorbance $A_{1\,\mathrm{cm}}^{1\%}$ (the absorbance of a 1% w/v solution in a 1 cm cell) is:



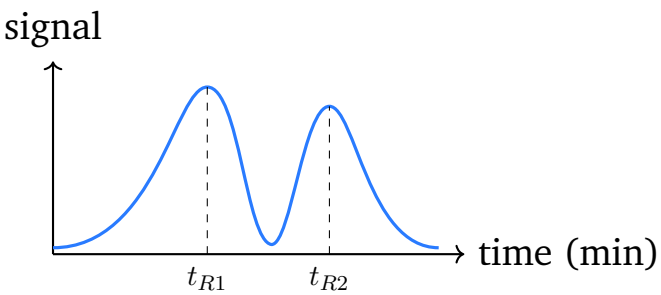
(A) 0.60

(B) 3.0

(C) 1200

(D) 300

Q114. The chromatogram below shows two peaks with retention times $t_{R1} = 4.0$ min and $t_{R2} = 5.2$ min, and equal baseline widths $w_1 = w_2 = 0.80$ min. Using $R_s = 2(t_{R2} - t_{R1})/(w_1 + w_2)$, the resolution between the peaks is:



(A) 1.5

(B) 0.67

(C) 3.0

(D) 0.30

Q115. In HPLC an unretained marker elutes at $t_0 = 1.5$ min and an analyte elutes at $t_R = 7.5$ min. The capacity (retention) factor $k' = (t_R - t_0)/t_0$ for this analyte is:

(A) 5.0

(B) 4.0

(C) 0.20

(D) 9.0

Q116. Which gas-chromatographic detector is a *universal*, non-destructive detector that responds to any analyte whose thermal conductivity differs from that of the carrier gas, making it suitable for permanent gases and water?

(A) Flame ionization detector (FID)

(B) Electron-capture detector (ECD)

(C) Thermal conductivity detector (TCD)

(D) Nitrogen–phosphorus detector (NPD)

Q117. On a strong cation-exchange resin, among ions of the *same* charge the one that is held most strongly (highest selectivity) is generally the one with the:

(A) Largest hydrated radius and lowest polarisability

(B) Lowest atomic number regardless of size

(C) Greatest tendency to remain fully hydrated

(D) Smallest hydrated radius and highest polarisability

Q118. In size-exclusion (gel-filtration) chromatography of a protein mixture, the order in which the molecules leave the column is:

(A) Largest molecules first, smallest molecules last

(B) Smallest molecules first, largest molecules last

(C) Most positively charged first

(D) Most hydrophobic first

Q119. In the van Deemter equation $H = A + B/u + C\,u$, the term that increases with flow velocity u and arises because solute molecules cannot equilibrate instantly between the mobile and stationary phases is the:

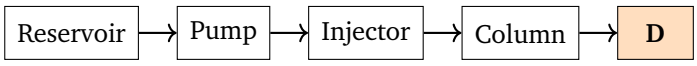
(A) Eddy-diffusion term, A

(B) Mass-transfer (resistance to mass transfer) term, $C\,u$

(C) Longitudinal-diffusion term, B/u

(D) Column-void term

Q120. The block diagram of a liquid chromatograph is shown. Identify the component labelled **D**, which sits immediately *after* the column and generates the electrical signal that is plotted as the chromatogram.



(A) Solvent reservoir

(B) High-pressure pump

(C) Detector

(D) Sample injector

Q121. During the conductometric titration of a *weak* acid (e.g. acetic acid) with a strong base (NaOH), the conductance of the solution *before* the equivalence point behaves as follows:

(A) It falls sharply as highly mobile H^+ ions are removed

(B) It stays exactly constant throughout

(C) It decreases continuously to zero at the end point

(D) It rises gradually as the poorly ionised acid is converted to its more conducting salt

Q122. In analytical method validation, the measure of precision obtained when the *same* method is run in the *same* laboratory but under varied conditions such as different days, analysts, and instruments is termed:

(A) Intermediate precision (ruggedness)

(B) Repeatability

(C) Reproducibility (between laboratories)

(D) Specificity

Part E: Pharmacognosy & Natural Products

Q123. Unorganized crude drugs are cell-free products of plant or animal metabolism, lacking a definite cellular structure. Which of the following sets contains **only unorganized** drugs?

(A) Fennel fruit, Quassia wood, Cinchona bark

(B) Squill bulb, Datura leaf, Ginger rhizome

(C) Gamboge, Beeswax, Catechu

(D) Ergot, Liquorice root, Clove bud

Q124. A monograph groups the drugs *Catharanthus*, *Vinca* and *Strychnos* together solely because their principal constituents are indole-type nitrogenous bases. This basis of arranging crude drugs is:

(A) Chemical classification

(B) Morphological classification

(C) Alphabetical classification

(D) Pharmacological classification

Q125. Arranging the crude drugs *Atropa*, *Hyoscyamus*, *Datura* and *Solanum* under one heading because all belong to the family Solanaceae is an example of which type of classification?

(A) Morphological classification

(B) Taxonomical (botanical) classification

(C) Chemical classification

(D) Therapeutic classification

Q126. Besides reserpine, the roots of *Rauwolfia serpentina* contain a monoterpenoid indole alkaloid used as a peripheral vasodilator in cerebrovascular disorders. This alkaloid, also called raubasine, is:

(A) Vincristine

(B) Quinidine

(C) Strychnine

(D) Ajmalicine

Q127. Among the opium alkaloids, one phthalide-isoquinoline base is essentially non-narcotic and is used clinically as an antitussive. This alkaloid, formerly called narcotine, is:

(A) Noscapine

(B) Thebaine

(C) Papaverine

(D) Apomorphine

Q128. The toxic principle of green/sprouted potato (*Solanum tuberosum*), in which a nitrogen-containing steroidal aglycone (solanidine) is linked to a trisaccharide, is best classified as a:

(A) Purine pseudo-alkaloid

(B) Steroidal glycoalkaloid

(C) Tropane alkaloid

(D) Quinoline alkaloid

Q129. The seeds of *Peganum harmala* yield a β-carboline (indole) alkaloid that is a reversible monoamine-oxidase inhibitor and shows blue fluorescence in ultraviolet light. This alkaloid is:

(A) Colchicine

(B) Pilocarpine

(C) Harmine

(D) Lobeline

Q130. Ouabain (g-strophanthin), a fast-acting, water-soluble cardenolide formerly used by injection, is obtained from the seeds of which plant?

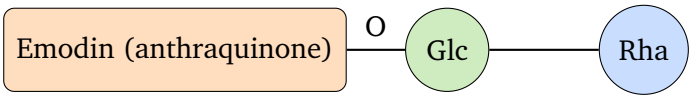
(A) *Digitalis purpurea*

(B) *Nerium oleander*

(C) *Convallaria majalis*

(D) *Strophanthus gratus*

Q131. The schematic below shows glucofrangulin A, the chief purgative glycoside of frangula (*Rhamnus frangula*) bark, in which an emodin-type anthraquinone aglycone carries two sugar units.



O-glycosidic linkages

On the basis of the aglycone, glucofrangulin belongs to which class of glycoside?

(A) Anthraquinone (purgative) glycoside

(B) Cardiac (cardenolide) glycoside

(C) Cyanogenetic glycoside

(D) Steroidal saponin glycoside

Q132. The primary (genuine) cardiac glycoside of *Digitalis lanata* leaves, from which the therapeutic agent digoxin is obtained on partial hydrolysis (loss of glucose and acetyl group), is:

(A) Sennoside A

(B) Lanatoside C

(C) Glycyrrhizin

(D) Sinigrin

Q133. Apigenin, the aglycone of the glycoside apiin found in parsley and chamomile, possesses a 2-phenylchromone (15-carbon C6–C3–C6) skeleton with no 3-hydroxyl group. Apigenin is therefore a:

(A) Flavonol

(B) Anthocyanidin

(C) Flavone

(D) Isoflavone

Q134. Prunasin, found in the leaves of wild cherry (*Prunus serotina*), on enzymatic hydrolysis liberates glucose, benzaldehyde and a toxic gas. The class of this glycoside and the gas evolved are, respectively:

(A) Saponin glycoside; carbon dioxide

(B) Anthraquinone glycoside; hydrogen sulphide

(C) Cardiac glycoside; ammonia

(D) Cyanogenetic glycoside; hydrogen cyanide

Q135. Cassia oil, distilled from the bark and leaves of *Cinnamomum cassia*, differs from true cinnamon bark oil chiefly in being almost devoid of eugenol. Its dominant aldehydic constituent (about 80–90%) is:

(A) Cinnamaldehyde

(B) Menthone

(C) Geraniol

(D) Safrole

Q136. Wintergreen oil, obtained from *Gaultheria procumbens*, is liberated on enzymatic hydrolysis of the glycoside gaultherin and consists almost entirely of a single aromatic ester used as a counter-irritant. This ester is:

(A) Bornyl acetate

(B) Anethole

(C) Methyl salicylate

(D) Linalyl acetate

Q137. Turpentine is the oleoresin obtained by tapping living *Pinus* species. On steam distillation it yields the volatile oil of turpentine (chiefly α- and β-pinene) and a solid residue. This non-volatile residue is:

(A) Benzoin

(B) Colophony (rosin)

(C) Agar

(D) Beeswax

Q138. Ghatti gum (Indian gum), an exudate of *Anogeissus latifolia* used as an emulsifying and suspending agent, is botanically and commercially most similar to which other gum?

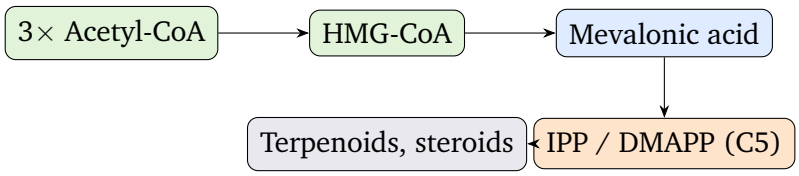
(A) Tragacanth (a swelling gel-former)

(B) Agar (a marine polysaccharide)

(C) Colophony (a resin)

(D) Acacia (a freely water-soluble exudate gum)

Q139. The biosynthetic scheme below depicts the early steps leading to the universal C5 isoprene unit. Identify the pathway shown.



(A) Acetate–mevalonate pathway giving terpenoids and steroids

(B) Shikimic acid pathway giving aromatic amino acids

(C) Acetate–malonate pathway giving fatty acids

(D) Pentose phosphate pathway giving sugars

Q140. In the shikimic acid pathway, the enzyme phenylalanine ammonia-lyase (PAL) catalyses the committed step that channels an aromatic amino acid into the phenylpropanoid pool. The immediate product of PAL acting on phenylalanine is:

(A) Mevalonic acid

(B) *trans*-Cinnamic acid

(C) Geranyl pyrophosphate

(D) Malonyl-CoA

Q141. The official volatile-oil content of a crude drug such as clove or cardamom is determined by hydro/steam distillation and measurement of the oil collected in a graduated trap. The apparatus used for this assay is the:

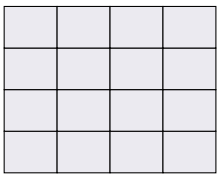
(A) Soxhlet apparatus

(B) Reichert–Meissl flask

(C) Clevenger apparatus

(D) Westphal balance

Q142. The microscopical sketch shows the suberised protective tissue often seen in transverse sections of bark and underground drugs: thin-walled, tabular cells arranged in regular radial rows.



suberised tissue

This tissue, made of dead suberised cells, is:

(A) Cork (phellem)

(B) Xylem vessels

(C) Pith parenchyma

(D) Phloem fibres

Part F: Pharmaceutical Biotechnology & Microbiology

Q143. A type II restriction endonuclease recognises the hexameric palindrome `AGATCT` and cleaves each strand between A and G (A|GATCT), generating four-base 5′ single-stranded extensions. This enzyme is:

(A) *Hae*III, which cuts GG|CC and leaves blunt ends

(B) *Xba*I, which cuts T|CTAGA and recognises TCTAGA

(C) *Bgl*II, which cuts A|GATCT and leaves 5′ GATC overhangs

(D) *Sma*I, which cuts CCC|GGG and leaves blunt ends

Q144. For high-level expression of a glycosylated recombinant protein in cultured **insect cells** (e.g. *Spodoptera frugiperda* Sf9 cells), the foreign gene is most commonly placed under the strong *polyhedrin* promoter of which expression system?

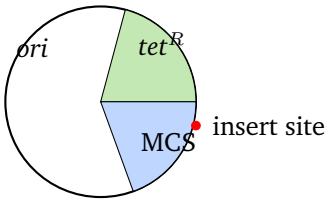
(A) A phagemid rescued by helper phage in *E. coli*

(B) The baculovirus (*Autographa californica* nucleopolyhedrovirus) vector

(C) A pBR322 plasmid driven by the *lac* promoter

(D) A λ replacement vector packaged *in vitro*

Q145. After a ligation is transformed into *E. coli*, the circular vector below carries an *ori*, a tetracycline-resistance marker (tet^R) and a multiple-cloning site (MCS). To rapidly confirm *which* colonies carry an insert of the expected size without first purifying any plasmid, a little of each colony is added directly as template in a PCR using primers flanking the MCS. This screening method is:



(A) Colony PCR, in which lysed colony material is the template

(B) Southern blotting of the intact colony

(C) Rapid amplification of cDNA ends (RACE) on the colony

(D) Sanger sequencing of every colony before any PCR

Q146. Reteplase is a recombinant, non-glycosylated deletion mutant of tissue plasminogen activator (t-PA) that retains only the kringle-2 and protease domains. Its principal therapeutic use is as a:

(A) Recombinant clotting factor for haemophilia B

(B) Colony-stimulating factor for neutropenia

(C) Long-acting insulin analogue for diabetes

(D) Thrombolytic (fibrinolytic) agent given to dissolve clots in acute myocardial infarction

Q147. Abciximab, used to prevent platelet aggregation during coronary angioplasty, is unusual among antibody-derived drugs because the active substance is not a whole IgG but only the:

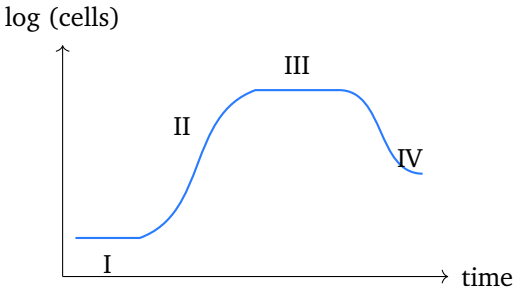
(A) Fc fragment, which blocks complement

(B) Fab fragment of a chimeric antibody directed against the platelet glycoprotein IIb/IIIa receptor

(C) Intact pentameric IgM

(D) Free light chains alone

Q148. The batch growth curve below shows the four phases I–IV of a mould fermentation. **Gluconic acid**, made by submerged aerobic fermentation in which glucose oxidase oxidises glucose, is produced industrially mainly by:



(A) *Aspergillus niger*

(B) *Saccharomyces cerevisiae* under anaerobic conditions

(C) *Lactobacillus delbrueckii*

(D) *Clostridium acetobutylicum*

Q149. **Riboflavin (vitamin B_2)** is manufactured on a commercial scale by microbial overproduction. The organism most widely used for its industrial fermentation is the flavinogenic ascomycete:

(A) *Gluconobacter suboxydans*

(B) *Corynebacterium glutamicum*

(C) *Ashbya gossypii* (*Eremothecium ashbyii*)

(D) *Propionibacterium freudenreichii*

Q150. **Nystatin**, a polyene macrolide antifungal that binds ergosterol in fungal membranes and is used topically and for oral candidiasis, is obtained by fermentation of:

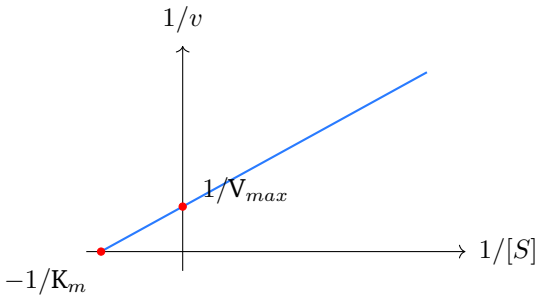
(A) *Penicillium chrysogenum*

(B) *Streptomyces noursei*

(C) *Bacillus licheniformis*

(D) *Micromonospora purpurea*

Q151. The ratio k_{cat}/K_m is widely used to compare an enzyme's catalytic efficiency toward different substrates. The double-reciprocal (Lineweaver–Burk) plot below linearises the kinetics, with intercepts marking $1/V_{max}$ and $-1/K_m$. The quantity k_{cat}/K_m is best described as the:



(A) Substrate concentration giving half-maximal velocity

(B) Total amount of product formed over the whole reaction

(C) Reaction velocity measured at saturating substrate

(D) Specificity constant, the apparent second-order rate constant governing reaction of free enzyme with substrate at low [S]

Q152. In one method of enzyme immobilisation the enzyme molecules are joined directly to one another (with no separate carrier) by a bifunctional reagent such as glutaraldehyde, giving insoluble particles often called cross-linked enzyme aggregates. This support-free method is:

(A) Entrapment within a calcium-alginate gel lattice

(B) Physical adsorption onto an ion-exchange resin

(C) Cross-linking of enzyme molecules with a bifunctional reagent

(D) Covalent attachment to a chemically activated matrix

Q153. Human IgG occurs as four subclasses (IgG1–IgG4) differing in effector function. Which subclass is the **poorest** activator of the classical com-

plement pathway, failing to fix C1q effectively, a property exploited in therapeutic antibodies designed for low effector activity?

(A) IgG4

(B) IgG1

(C) IgG3

(D) IgG2

Q154. When an antibody binds the receptor-binding site of a virus, or the active site of a bacterial exotoxin, so that the pathogen or toxin can no longer attach to or enter host cells, the protective effector function being described is:

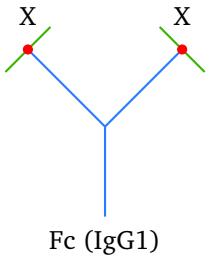
(A) Opsonisation for phagocytosis

(B) Neutralisation

(C) Complement-mediated lysis

(D) Antibody-dependent cell-mediated cytotoxicity

Q155. Etanercept carries the WHO stem `-cept`, marking it as a **receptor–Fc fusion protein** rather than a true antibody. As drawn schematically below, it joins the extracellular ligand-binding portion of the human p75 TNF receptor (region X) to the Fc portion of human IgG1. Its mechanism of action is to:



(A) Replace a deficient clotting factor

(B) Deliver a cytotoxic drug to tumour cells

(C) Provide passive immunity against a virus

(D) Act as a soluble decoy receptor that binds and sequesters TNF-α, blocking it from reaching cell-surface receptors

Q156. In the complement system, all three activation pathways converge on the cleavage of one central component into a small anaphylatoxin fragment and a larger fragment that becomes the major opsonin and seeds the membrane-attack complex. This central component is:

(A) C3, cleaved to C3a and C3b

(B) C9, which polymerises first

(C) C1q, the recognition unit only

(D) Factor H, a soluble regulator

Q157. Thin, tightly coiled spirochaetes such as *Treponema* are too slender to be seen well by ordinary bright-field microscopy and stain poorly by the Gram method. They are therefore observed in fresh specimens as bright, motile helices against a dark background using:

(A) Routine Gram staining under bright-field

(B) Ziehl–Neelsen acid-fast staining

(C) Dark-field (dark-ground) microscopy

(D) Scanning electron microscopy of dried films only

Q158. In the heat sterilisation of microorganisms, the **thermal death point** (TDP) of a culture is defined as the:

(A) Time at a fixed temperature needed to reduce the population by one log cycle

(B) Temperature rise that lowers the D-value to one-tenth

(C) Time needed to kill exactly half of the population

(D) Lowest temperature that kills all the organisms in a fixed-volume suspension within ten minutes

Q159. Glass vials destined for sterile injections must be freed not only of viable microbes but also of bacterial **endotoxin (pyrogen)**. Because endotoxin is far more heat-stable than spores, the depyrogenation step typically uses:

(A) Autoclaving at 121°C for 15 min, which destroys pyrogen

(B) Dry heat at about 250°C for $\geq$30 min, which both sterilises and destroys endotoxin

(C) Filtration through a 0.22 μm membrane, which removes endotoxin

(D) Exposure to 70% ethanol, which inactivates pyrogen

Q160. Microbiological monitoring of a sterile-manufacturing cleanroom includes passive air sampling, in which open Petri dishes of nutrient agar are exposed to the air for a set time and then incubated to count colonies that have settled out. These exposed plates are known as:

(A) Replicate organism detection plates (RODAC contact plates)

(B) Spread plates inoculated with a measured volume

(C) Settle plates (passive air-sampling plates)

(D) Pour plates for total viable count

Detailed Solutions

Q1.

Solution

Concept — Solubility product: For a 1:1 salt $CaCO_3 \rightleftharpoons Ca^{2+} + CO_3^{2-}$, $K_{sp} = [Ca^{2+}][CO_3^{2-}] = s^2$, where s is the molar solubility. **Reasoning:** $s = 3.0 \times 10^{-5}$ mol/L, so $K_{sp} = (3.0 \times 10^{-5})^2 = 9.0 \times 10^{-10}$. **Why the other options are wrong:**

- (A) 3.0×10^{-5} is the solubility s itself, not s^2.
- (B) 6.0×10^{-5} wrongly multiplies s by 2.
- (D) 2.7×10^{-14} corresponds to s^3, valid only for a 1:2 salt such as CaF_2.

Final Answer: $K_{sp} = s^2 = 9.0 \times 10^{-10} \Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept — Partition coefficient: $P = C_{oil}/C_{water}$ at equilibrium; with equal volumes the concentration ratio equals the mass ratio. **Reasoning:** $P = 120/30 = 4.0$. A value above 1 shows the drug favours the lipophilic (oil) phase. **Why the other options are wrong:**

- (A) 0.25 is the inverted (water/oil) ratio.
- (C) 0.40 misplaces the decimal.
- (D) 40 is off by a factor of ten.

Final Answer: $P = 120/30 = 4.0 \Rightarrow$ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept — HLB of a blend: The HLB of a surfactant mixture is the weighted average of the component HLB values by mass fraction. **Reasoning:** HLB $= 0.35(4.7) + 0.65(14.9) = 1.645 + 9.685 = 11.33 \approx 11.3$. **Why the other options are wrong:**

- (A) 4.7 is the Span 60 value alone.

- (B) 9.8 is the simple (unweighted) average, ignoring the proportions.
- (C) 14.9 is the Tween 60 value alone.

Final Answer: $0.35(4.7) + 0.65(14.9) = 11.3 \Rightarrow$ D

Answer: (D) Go Back to Q3

Q4.

Solution

Concept — Plastic (Bingham) flow: A plastic system does not flow until the applied stress exceeds a yield value f; above f the curve is linear. **Reasoning:**

The line meets the stress axis at a positive intercept f and is straight thereafter, the signature of Bingham plastic flow (e.g. concentrated flocculated suspensions, certain ointments). **Why the other options are wrong:**

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

Final Answer: positive yield intercept + linear $\Rightarrow$ plastic flow $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept — Dilatant flow: A shear-thickening system shows rising apparent viscosity as the shear rate increases; the curve bows toward the shear-rate axis. **Reasoning:** More stress is needed at higher $\dot{\gamma}$, so viscosity increases. This is dilatant behaviour, typical of concentrated deflocculated suspensions of small particles. **Why the other options are wrong:**

- (A) Newtonian is a straight line through the origin.
- (B) Pseudoplastic bows toward the stress axis (viscosity falls).
- (D) Thixotropy is a time-dependent hysteresis loop, not a single curve.

Final Answer: curve bows toward shear-rate axis $\Rightarrow$ dilatant $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept — Kinematic viscosity: Kinematic viscosity $\nu = \eta/\rho$; in CGS, poise divided by g/cm^3 gives stokes. **Reasoning:** $\nu = 1.2/1.5 = 0.80$ stokes. **Why the other options are wrong:**

- (A) 1.80 wrongly multiplies η by ρ.
- (B) 1.50 is the density value.
- (C) 1.25 inverts the ratio (ρ/η).

Final Answer: $\nu = 1.2/1.5 = 0.80$ stokes $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept — Carr's index: Carr's % $= \dfrac{(\rho_{tapped} - \rho_{bulk})}{\rho_{tapped}} \times 100$. **Reasoning:** $= \dfrac{0.65 - 0.52}{0.65} \times 100 = \dfrac{0.13}{0.65} \times 100 = 20\%$, indicating fair-to-passable flow. **Why the other options are wrong:**

- (A) 25% over-estimates the density difference.
- (C) 13% mistakenly omits the division by tapped density.
- (D) 35% is far too large.

Final Answer: $(0.13/0.65) \times 100 = 20\% \Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept — Hausner ratio: HR $= \rho_{tapped}/\rho_{bulk}$; ≥ 1 always, with higher values meaning poorer flow. **Reasoning:** HR $= 0.65/0.52 = 1.25$, consistent with the 20% Carr's index of the previous question (passable flow). **Why the other options are wrong:**

- (B) 0.80 is the inverted ratio; HR cannot be below 1.
- (C) 1.13 under-estimates the ratio.
- (D) 1.45 over-estimates it.

Final Answer: HR $= 0.65/0.52 = 1.25 \Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept — Mode of a frequency distribution: The mode is the size class with the tallest bar (highest frequency). **Reasoning:** The tallest bar in the histogram reaches its peak over the 45–60 μm class, which is therefore the modal interval. **Why the other options are wrong:**

- (A) 15–30 is a low-frequency class on the left.
- (B) 30–45 is the second-tallest, not the tallest.
- (D) 75–90 lies in the low-frequency tail.

Final Answer: tallest bar lies in 45–60 μm $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept — Henderson–Hasselbalch: $\text{pH} = \text{pK}_a + \log \dfrac{[\text{salt}]}{[\text{acid}]}$. **Reasoning:** $\log(10/1) = 1$, so pH $= 5.30 + 1 = 6.30$. **Why the other options are wrong:**

- (A) 4.30 subtracts the log term instead of adding.
- (B) 5.30 ignores the salt:acid ratio (would need a 1:1 ratio).
- (C) 5.40 wrongly uses $\log 1.25$ or similar.

Final Answer: pH $= 5.30 + \log 10 = 6.30 \Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept — Sodium-chloride equivalent: The E value relates a drug to its osmotic (tonicity) effect referenced to NaCl. **Reasoning:** By definition, E is the weight of NaCl that gives the same colligative (osmotic) effect as 1 g of the drug,

used in tonicity calculations. **Why the other options are wrong:**

- (B) Equivalence by mass is irrelevant; tonicity depends on number of particles.
- (C) Freezing-point depression of a 1% solution is the ΔT_f value, not E.
- (D) Molar concentration matching saline is not how E is defined.

Final Answer: weight of NaCl giving the same osmotic effect as 1 g drug $\Rightarrow$ A

Answer: (A) Go Back to Q11

Q12.

Solution

Concept — Stability (formation) constant: A larger K means the complexation equilibrium lies further toward the bound complex, i.e. a more stable, less dissociated complex. **Reasoning:** $K_M = 5 \times 10^4$ is 100 times larger than $K_N = 5 \times 10^2$, so complex M is the more stable and less dissociated species. **Why the other options are wrong:**

- (A) Faster dissociation means lower, not higher, stability.
- (C) The constants differ 100-fold, so they are not equally stable.
- (D) Stability is directly read from the formation constants.

Final Answer: larger $K \Rightarrow$ M more stable $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept — First-order kinetics: For first order, $\ln C = \ln C_0 - kt$; a plot of $\ln C$ versus time is linear with slope $-k$. **Reasoning:** The plot shows ln C falling linearly with time, the diagnostic feature of first-order decay. **Why the other options are wrong:**

- (A) Zero order is linear when C (not ln C) is plotted against time.
- (B) Second order is linear when $1/C$ is plotted against time.
- (D) Pseudo-zero-order applies to suspensions, with C versus t linear.

Final Answer: linear ln C vs $t \Rightarrow$ first order $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept — Shelf-life: For first-order degradation, $t_{90} = 0.105/k$ (time for the drug to fall to 90% of its initial content). **Reasoning:** $t_{90} = 0.105/0.0105 = 10$ months. **Why the other options are wrong:**

- (A) 5 months uses an incorrect numerator.
- (B) 33 months is the half-life ($0.693/k = 66$) halved by error.
- (C) 66 months is the half-life $t_{1/2} = 0.693/k$, not t_{90}.

Final Answer: $t_{90} = 0.105/0.0105 = 10$ months $\Rightarrow$ D

Answer: (D) Go Back to Q14

Q15.

Solution

Concept — Arrhenius equation: $\ln k = \ln A - \dfrac{E_a}{R} \cdot \dfrac{1}{T}$; plotting $\ln k$ against $1/T$ gives a straight line. **Reasoning:** Comparing with $y = c + mx$, the slope $m = -E_a/R$, from which the activation energy is obtained. **Why the other options are wrong:**

- (B) The slope is negative, not $+E_a/R$.
- (C) $-R/E_a$ inverts the relationship.
- (D) $\ln A$ is the intercept, not the slope.

Final Answer: slope $= -E_a/R \Rightarrow$ A

Answer: (A) Go Back to Q15

Q16.

Solution

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

- (A) $\log(x/m)$ vs $\log C$ linearises the Freundlich, not Langmuir, isotherm.
- (C) x/m vs C is the curved (non-linear) form.
- (D) $\ln(x/m)$ vs $1/C$ has no theoretical basis here.

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

Answer: (B) Go Back to Q16

Q17.

Solution

Concept — Eutectic point: On a binary phase diagram the two liquidus curves meet at the eutectic, the composition with the lowest melting point of the system. **Reasoning:** Point E is the lowest temperature at which liquid exists; below it both solids crystallise together. It is the eutectic (lowest-melting) mixture. **Why the other options are wrong:**

- (A) and (B) The pure-component melting points are the two upper ends of the curves, not E.
- (D) E is defined by melting behaviour, not maximum solubility.

Final Answer: the lowest meeting point E is the eutectic $\Rightarrow$ C

Answer: (C) Go Back to Q17

Q18.

Solution

Concept — Noyes–Whitney: Dissolution rate dC/dt is directly proportional to the effective surface area A. **Reasoning:** Micronisation increases A, so dC/dt rises and the drug dissolves faster. This is the basis for improving the dissolution of poorly soluble drugs. **Why the other options are wrong:**

- (A) A larger A raises, never lowers, the rate.
- (B) The effect is real and significant.
- (C) C_s (saturation solubility) is essentially unchanged by ordinary micronisation.

Final Answer: larger $A \Rightarrow$ faster dissolution $\Rightarrow$ D

Answer: (D) Go Back to Q18

Q19.

Solution

Concept — Higuchi model: For matrix diffusion, $Q = k_H\sqrt{t}$; a plot of Q against $\sqrt{t}$ is linear through the origin. **Reasoning:** The straight Q vs $\sqrt{t}$ line is the hallmark of Higuchi (diffusion-controlled) release from a matrix. **Why the other options are wrong:**

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

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

Answer: (B) Go Back to Q19

Q20.

Solution

Concept — Korsmeyer–Peppas exponent: The release exponent n classifies the mechanism; the boundary values depend on geometry. **Reasoning:** For a cylinder, $n = 0.45$ is Fickian and $n = 0.89$ is Case-II; values between, $0.45 < n < 0.89$, indicate anomalous (non-Fickian) transport (coupled diffusion and relaxation). **Why the other options are wrong:**

- (A) $n = 0.45$ alone is purely Fickian.
- (B) $n \geq 1.0$ describes super Case-II, beyond the anomalous band.
- (D) $n < 0.30$ is not a defined mechanism boundary.

Final Answer: anomalous transport for $0.45 < n < 0.89 \Rightarrow$ C

Answer: (C) Go Back to Q20

Q21.

Solution

Concept — BCS: Drugs are classed by solubility and permeability: I (high/high), II (low/high), III (high/low), IV (low/low). **Reasoning:** High solubility with low permeability is Class III, where permeation across the gut wall is the rate-limiting step. **Why the other options are wrong:**

- (A) Class I is high solubility and high permeability.

- (B) Class II is low solubility, high permeability.
- (C) Class IV is low solubility and low permeability.

Final Answer: high solubility, low permeability ⇒ Class III ⇒ D

Answer: (D) Go Back to Q21

Q22.

Solution

Concept — Similarity factor f_2: f_2 ranges 0–100; values of 50 or above indicate the two profiles are similar (no more than about 10% average difference). **Reasoning:** The nearly overlapping curves imply a small average difference, so f_2 lies between 50 and 100, indicating similar profiles. **Why the other options are wrong:**

- (B) $f_2 < 50$ would mean dissimilar, contradicting the overlap.
- (C) Identical profiles give $f_2 = 100$, not 0.
- (D) f_2 is precisely the tool used to compare such profiles.

Final Answer: overlapping profiles ⇒ f_2 in 50–100 (similar) ⇒ A

Answer: (A) Go Back to Q22

Q23.

Solution

Concept — Tablet binders: Binders are adhesives added to the granulating fluid to hold powder particles together and give cohesive granules. **Reasoning:** Acacia (gum arabic) mucilage is a classic natural binder used in wet granulation. **Why the other options are wrong:**

- (A) Disintegrants (e.g. starch, croscarmellose) break the tablet apart, the opposite role.
- (C) Glidants (e.g. colloidal silica) improve powder flow.
- (D) Lubricants (e.g. magnesium stearate) reduce die-wall friction.

Final Answer: acacia mucilage is a binder ⇒ B

Answer: (B) Go Back to Q23

Q24.

Solution

Concept — Weight-variation limits: The pharmacopoeial tolerance widens as average tablet weight decreases. **Reasoning:** For average weight in the range 80–250 mg, the IP/USP limit is $\pm 7.5\%$; 130 mg falls in this band. **Why the other options are wrong:**

- (A) $\pm 5\%$ applies to tablets above 250 mg (heaviest band).
- (B) $\pm 15\%$ would only apply to very light/uncommon limits.
- (D) $\pm 10\%$ applies to tablets of 80 mg or less.

Final Answer: 130 mg lies in the 80–250 mg band $\Rightarrow \pm 7.5\% \Rightarrow$ C

Answer: (C) Go Back to Q24

Q25.

Solution

Concept — Capsule sizes: Hard-gelatin shells run from 000 (largest) down to 5 (smallest); the larger the number, the smaller the capsule. **Reasoning:** Among the options, size 5 has the smallest fill capacity. **Why the other options are wrong:**

- (A) Size 0 is much larger than size 5.
- (B) Size 1 is larger than size 5.
- (C) Size 00 is among the largest shells.

Final Answer: size 5 is the smallest $\Rightarrow$ D

Answer: (D) Go Back to Q25

Q26.

Solution

Concept — Displacement value (DV): Base required = (total base for blank moulds) − (drug mass ÷ DV). DV = mass of base displaced by 1 g of drug. **Reasoning:** Blank base for 6 moulds $= 6 \times 2 = 12$ g. Drug $= 6 \times 0.4 = 2.4$ g. Base displaced $= 2.4/4 = 0.6$ g. Base needed $= 12 - 0.6 = 11.4$ g. **Why the other options are wrong:**

- (B) 12.0 g ignores the displacement correction.
- (C) 9.6 g wrongly subtracts the whole drug mass (2.4 g).

- (D) 10.8 g uses an incorrect DV of 2.

Final Answer: $12 - (2.4/4) = 11.4$ g $\Rightarrow$ A

Answer: (A) Go Back to Q26

Q27.

Solution

Concept — Electrical conductivity test: An emulsion conducts well only when its external (continuous) phase is the conducting aqueous phase. **Reasoning:** If the lamp does not glow, the continuous phase is non-conducting oil, so the emulsion is water-in-oil (w/o). **Why the other options are wrong:**

- (A) An o/w emulsion has a conducting water external phase and would light the lamp.
- (C) A w/o/w multiple emulsion has an aqueous outer phase and conducts.
- (D) A typical o/w microemulsion also conducts.

Final Answer: no conduction $\Rightarrow$ oil external phase $\Rightarrow$ w/o $\Rightarrow$ B

Answer: (B) Go Back to Q27

Q28.

Solution

Concept — Sedimentation volume: $F = V_u/V_o$, the ratio of the final sediment volume to the original suspension volume. **Reasoning:** $F = 16/40 = 0.40$. **Why the other options are wrong:**

- (A) 0.16 misreads the original volume.
- (B) 0.24 corresponds to the supernatant fraction (24/40 inverted by error).
- (D) 2.50 is the inverted ratio (V_o/V_u).

Final Answer: $F = 16/40 = 0.40 \Rightarrow$ C

Answer: (C) Go Back to Q28

Q29.

Solution

Concept — Ophthalmic preservatives: Multidose eye drops require an antimicrobial preservative to maintain sterility between uses. **Reasoning:** Chlorobutanol is a classic antimicrobial preservative used in multidose ophthalmic and parenteral products. **Why the other options are wrong:**

- (A) Tonicity is adjusted with NaCl or dextrose, not chlorobutanol.
- (B) Viscosity enhancers are polymers such as methylcellulose.
- (C) Buffering uses borate or phosphate salts.

Final Answer: chlorobutanol is an antimicrobial preservative $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q29

Q30.

Solution

Concept — Ointment base classes: Absorption bases are anhydrous (or contain little water) but can take up water to form w/o emulsions, owing to emulsifiers like wool fat and cholesterol. **Reasoning:** Hydrophilic petrolatum contains wool fat and cholesterol and absorbs water to form a w/o emulsion, so it is an absorption (emulsifiable) base. **Why the other options are wrong:**

- (B) Water-soluble bases (e.g. PEG) contain no oil phase.
- (C) A pure oleaginous base (white petrolatum) cannot absorb appreciable water.
- (D) Water-removable o/w bases are washable creams, not this base.

Final Answer: water-absorbing wool-fat base $\Rightarrow$ absorption base $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q30

Q31.

Solution

Concept — Colloid mill: A colloid mill forces material through a very narrow adjustable gap between a high-speed rotor and a fixed stator, generating intense shear. **Reasoning:** The dominant mechanism is shear in the narrow rotor–stator clearance, ideal for emulsions and fine suspensions. **Why the other options are**

wrong:

- (A) Impact/attrition between balls describes a ball mill.
- (C) Compression between rollers describes a roller mill.
- (D) Cutting by knives describes a cutter (knife) mill.

Final Answer: narrow rotor–stator gap $\Rightarrow$ shear $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q31

Q32.

Solution

Concept — Reynolds number: In pipe flow, $Re < 2000$ is laminar, 2000–4000 is transitional, and $Re > 4000$ is fully turbulent. **Reasoning:** The chaotic swirling eddies of pattern (ii) are turbulent flow, which occurs when Re exceeds about 4000. **Why the other options are wrong:**

- (A) Below 2000 is laminar (pattern i).
- (B) Exactly 2100 lies in the transitional, not fully turbulent, region.
- (D) Flow regime is determined precisely by the Reynolds number.

Final Answer: turbulent eddies $\Rightarrow$ $Re > 4000$ $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept — Drying-rate curve: While the surface stays wetted, evaporation proceeds at a steady maximum rate set by the air conditions: the constant-rate period. **Reasoning:** A saturated surface and unchanging drying rate define the constant-rate period; it ends at the critical moisture content. **Why the other options are wrong:**

- (A) The falling-rate period follows, once the surface dries out.
- (B) Equilibrium moisture is the end-point, not a period of constant rate.
- (C) The induction (warm-up) period precedes the constant-rate stage and is brief.

Final Answer: saturated surface, steady rate $\Rightarrow$ constant-rate period $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q33

Q34.

Solution

Concept — Niosomes: Niosomes are bilayer vesicles built from non-ionic surfactants, usually with cholesterol added to stabilise the bilayer. **Reasoning:** They mimic liposomes but use non-ionic surfactants instead of phospholipids, giving better chemical stability and lower cost. **Why the other options are wrong:**

- (B) Phospholipid bilayers define liposomes, not niosomes.
- (C) PLGA polymer shells describe polymeric nanoparticles/microspheres.
- (D) Cross-linked albumin describes albumin microspheres.

Final Answer: non-ionic surfactant vesicles $\Rightarrow$ A

Answer: (A) Go Back to Q34

Q35.

Solution

Concept — First-order half-life: For first-order elimination $t_{1/2} = 0.693/k_e$. **Reasoning:** With $k_e = 0.231\,\text{h}^{-1}$, $t_{1/2} = 0.693/0.231 = 3$ h. On a semi-log plot the slope is $-k_e/2.303$, confirming a single first-order process. **Why the other options are wrong:**

- (A) 0.23 h confuses the rate constant value with the half-life.
- (B) 1.5 h uses $0.693/(2 \times 0.231)$ in error.
- (D) 7 h would require $k_e \approx 0.10\,\text{h}^{-1}$.

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

Answer: (C) Go Back to Q35

Q36.

Solution

Concept — Plateau principle: During constant-rate infusion the level approaches steady state as $C = C_{ss}(1 - e^{-k_e t})$; the fraction reached depends only on the number of half-lives. **Reasoning:** After n half-lives the fraction of C_{ss} is $1-(1/2)^n$. After 4 half-lives this is $1 - 1/16 = 0.9375 \approx 94\%$. **Why the other options are**

wrong:

- (A) 2 half-lives gives 75%.
- (C) 7 half-lives gives about 99%.
- (D) 10 half-lives gives about 99.9%.

Final Answer: $1-(1/2)^4 = 94\%$ at 4 half-lives $\Rightarrow$ B

Answer: (B) Go Back to Q36

Q37.

Solution

Concept — Hepatic extraction ratio: For a high-extraction drug ($E \to 1$) almost all drug entering the liver is removed in one pass. **Reasoning:** Oral bioavailability $F \approx 1 - E$, so a drug with $E \approx 0.9$ has low oral bioavailability owing to extensive first-pass metabolism. Its hepatic clearance approaches hepatic blood flow and is therefore flow-limited. **Why the other options are wrong:**

- (B) High extraction gives low, not high, oral bioavailability; clearance is flow- not capacity-limited.
- (C) Extraction ratio directly governs first-pass loss.
- (D) Hepatic, not renal (GFR), clearance is involved.

Final Answer: Low oral F, flow-limited hepatic clearance $\Rightarrow$ A

Answer: (A) Go Back to Q37

Q38.

Solution

Concept — Potency vs efficacy on graded curves: Left-right position reflects potency (ED_{50}); the height of the plateau reflects efficacy (E_{max}). **Reasoning:** S lies further left, so it is more potent (lower ED_{50}), but R reaches a higher plateau, so R has greater efficacy. Because S cannot match R's maximum at any dose, S behaves as a partial agonist relative to R. **Why the other options are wrong:**

- (A) Reverses the assignment of potency and efficacy.
- (B) The plateaus differ, so efficacy is not identical.
- (C) R is the full agonist (higher E_{max}), not the partial one.

Final Answer: S more potent, R greater efficacy; S is partial agonist $\Rightarrow$ D

Answer: (D) Go Back to Q38

Q39.

Solution

Concept — Therapeutic index: TI $= LD_{50}/ED_{50}$; a larger value indicates a wider margin of safety. **Reasoning:** TI $= 400/10 = 40$. The two quantal curves show the lethal curve far to the right of the effective curve, so the index is well above 1. **Why the other options are wrong:**

- (A) 0.025 inverts the ratio (ED_{50}/LD_{50}).
- (C) 4000 multiplies the doses instead of dividing.
- (D) 410 adds the doses, which is meaningless here.

Final Answer: TI $= 400/10 = 40 \Rightarrow$ B

Answer: (B) Go Back to Q39

Q40.

Solution

Concept — Phase I and Phase II metabolism: Biotransformation is divided into functionalisation (Phase I) and conjugation (Phase II). **Reasoning:** Phase I reactions (oxidation, reduction, hydrolysis), commonly mediated by cytochrome P450, add or expose a reactive group; Phase II then conjugates that group (glucuronide, sulfate, glutathione, acetyl) to give a more polar, usually inactive, readily excreted product. Option (A) states this correctly. **Why the other options are wrong:**

- (B) Phase I usually precedes Phase II, not the reverse.
- (C) Glucuronidation increases water solubility and usually inactivates.
- (D) CYP450 catalyses oxidative Phase I reactions, not conjugation.

Final Answer: Phase I functionalises, Phase II conjugates $\Rightarrow$ A

Answer: (A) Go Back to Q40

Q41.

Solution

Concept — Enzyme induction: Inducers increase the synthesis of metabolising enzymes over time, accelerating clearance of co-administered substrates. **Reasoning:** A potent CYP3A4 inducer (e.g. rifampicin, carbamazepine) raises enzyme levels over days to weeks, increasing metabolism of the substrate so its plasma concentration falls and its therapeutic effect is lost. Option (D) describes this. **Why the other options are wrong:**

- (A) Induction lowers, not raises, substrate levels and is gradual, not within minutes.
- (B) Induction acts mainly on Phase I (CYP) enzymes and does change levels.
- (C) It does not turn a substrate into a prodrug.

Final Answer: Faster metabolism, lower levels, lost effect over days to weeks $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q41

Q42.

Solution

Concept — Cholinergic receptor families: Acetylcholine acts on muscarinic (GPCR) and nicotinic (ionotropic) receptors. **Reasoning:** Nicotinic receptors at autonomic ganglia (N_N) and the skeletal muscle end-plate (N_M) are ligand-gated cation channels that open on ACh binding, giving fast depolarisation. This matches the branch shown. **Why the other options are wrong:**

- (A), (B) Muscarinic M_2/M_3 are GPCRs, not ionotropic channels.
- (C) The β_2 adrenergic receptor is not cholinergic.

Final Answer: Nicotinic receptors are ligand-gated cation channels $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q42

Q43.

Solution

Concept — β_1-selective vasodilating blocker: Some newer cardioselective blockers add a vasodilator action. **Reasoning:** Nebivolol is highly β_1-selective and additionally promotes endothelial nitric-oxide release, producing vasodilation and lowering peripheral resistance, which distinguishes it from older β_1-selective agents. **Why the other options are wrong:**

- (A) Sotalol is a non-selective blocker with Class III action, no NO effect.
- (C) Pindolol is non-selective with intrinsic sympathomimetic activity.
- (D) Carteolol is a non-selective blocker.

Final Answer: Nebivolol (β_1-selective, NO-mediated vasodilation) $\Rightarrow$ B

Answer: (B) Go Back to Q43

Q44.

Solution

Concept — M_3-selective antimuscarinics: Bladder detrusor contraction is mediated mainly by M_3 receptors, so M_3-preferring antagonists relax the bladder. **Reasoning:** Darifenacin is relatively M_3-selective and largely peripheral, reducing detrusor overactivity with less central (M_1) antimuscarinic load, an advantage in older patients at risk of confusion. **Why the other options are wrong:**

- (A) Immediate-release oxybutynin is non-selective and crosses into the CNS, causing more cognitive effects.
- (B) Benztropine is a centrally acting antimuscarinic for parkinsonism.
- (D) Dicyclomine is a non-selective antispasmodic.

Final Answer: Darifenacin is M_3-selective and peripheral $\Rightarrow$ C

Answer: (C) Go Back to Q44

Q45.

Solution

Concept — Termination of noradrenergic transmission: Released noradrenaline is removed mainly by neuronal reuptake (uptake-1) into the nerve terminal. **Reasoning:** Cocaine and tricyclic-type agents block uptake-1, so nora-

drenaline persists in the synaptic cleft and sympathetic effects are potentiated, as the diagram's uptake-1 arrow implies. **Why the other options are wrong:**

- (B) Inhibiting tyrosine hydroxylase (metyrosine) reduces, not potentiates, transmission.
- (C) They are uptake blockers, not direct α_1 agonists.
- (D) Blocking vesicular storage describes reserpine, which depletes transmitter.

Final Answer: Block of neuronal uptake-1 raising synaptic NA $\Rightarrow$ A

Answer: (A) Go Back to Q45

Q46.

Solution

Concept — α_{1A}-selective antagonists in BPH: Prostatic smooth muscle is rich in α_{1A} receptors, so subtype-selective blockers relieve obstruction with little vascular (α_{1B}) effect. **Reasoning:** Silodosin is highly α_{1A}-selective, relaxing prostatic and bladder-neck smooth muscle in benign prostatic hyperplasia while causing little orthostatic hypotension. **Why the other options are wrong:**

- (A) Doxazosin is a non-selective α_1 blocker with greater hypotensive effect.
- (B) Phenoxybenzamine is a non-selective irreversible α blocker.
- (C) Yohimbine is an α_2 antagonist.

Final Answer: Silodosin is α_{1A}-selective $\Rightarrow$ D

Answer: (D) Go Back to Q46

Q47.

Solution

Concept — Dopamine partial agonism: A D_2 partial agonist provides moderate dopaminergic tone, dampening overactivity while preventing excessive blockade. **Reasoning:** Aripiprazole is a D_2 (and 5-HT_{1A}) partial agonist, acting as a "dopamine system stabiliser". This gives antipsychotic efficacy with relatively little extrapyramidal effect and a lower tendency to raise prolactin than full D_2 antagonists. **Why the other options are wrong:**

- (B) Haloperidol, not aripiprazole, is a pure D_2 antagonist.

- (C) It is not an SSRI.
- (D) It does not act on GABA-A receptors.

Final Answer: D_2 partial agonist (dopamine stabiliser) $\Rightarrow$ A

Answer: (A) Go Back to Q47

Q48.

Solution

Concept — NDRI antidepressant: Some antidepressants raise noradrenaline and dopamine rather than serotonin. **Reasoning:** Bupropion inhibits the noradrenaline and dopamine reuptake transporters with little serotonergic action; it lacks the sexual dysfunction common to SSRIs and is also approved as an aid to smoking cessation. **Why the other options are wrong:**

- (A) Mirtazapine is a noradrenergic and specific serotonergic agent acting via α_2 and 5-HT receptor blockade.
- (C) Trazodone is a serotonin antagonist/reuptake inhibitor.
- (D) Vortioxetine is a multimodal serotonergic agent.

Final Answer: Bupropion is the NA/DA reuptake inhibitor $\Rightarrow$ B

Answer: (B) Go Back to Q48

Q49.

Solution

Concept — GABA-transaminase inhibition: Raising brain GABA can be achieved by blocking the enzyme that catabolises it. **Reasoning:** Vigabatrin (vinyl-GABA) is an irreversible inhibitor of GABA-transaminase (GABA-T), so GABA accumulates and inhibitory tone rises, controlling seizures (notably infantile spasms and refractory partial seizures). **Why the other options are wrong:**

- (A) Sodium channel block describes phenytoin or carbamazepine.
- (B) NMDA antagonism is not its action.
- (D) Binding the $\alpha_2\delta$ calcium channel subunit describes gabapentin/pregabalin.

Final Answer: Irreversible GABA-transaminase inhibition $\Rightarrow$ C

Answer: (C) Go Back to Q49

Q50.

Solution

Concept — Peripheral μ-opioid agonism: An opioid that does not reach the brain acts only on gut opioid receptors. **Reasoning:** Loperamide is a μ-agonist that slows intestinal transit by acting on myenteric plexus receptors, but it is a P-glycoprotein substrate that is pumped back out of the CNS, so at therapeutic doses it has negligible central or analgesic effect. **Why the other options are wrong:**

- (A) It is an agonist, not an antagonist.
- (B) It reduces, not stimulates, motility.
- (C) It is not an osmotic laxative.

Final Answer: Peripheral μ-agonist excluded from CNS by P-gp $\Rightarrow$ D

Answer: (D) Go Back to Q50

Q51.

Solution

Concept — NMDA-receptor blockade: Ketamine is a dissociative anaesthetic acting on glutamatergic transmission. **Reasoning:** Ketamine non-competitively blocks the NMDA glutamate receptor ion channel, producing dissociative anaesthesia, analgesia and amnesia while largely preserving airway reflexes and cardiovascular drive. **Why the other options are wrong:**

- (A) GABA-A potentiation is the mechanism of propofol and benzodiazepines.
- (B) It does not act as a sodium channel blocker.
- (C) It is not primarily an opioid agonist.

Final Answer: Non-competitive NMDA-receptor block $\Rightarrow$ D

Answer: (D) Go Back to Q51

Q52.

Solution

Concept — Orexin antagonism: Orexin (hypocretin) neuropeptides drive wakefulness; blocking their receptors promotes sleep. **Reasoning:** Suvorexant is a dual orexin receptor antagonist (OX_1/OX_2) that reduces the wake-promoting signal, a mechanism distinct from the GABAergic action of benzodiazepines and Z-drugs.

Why the other options are wrong:

- (A) Melatonin receptor agonism describes ramelteon.
- (C) Increasing chloride channel-open duration describes barbiturates.
- (D) It does not act as a histamine agonist.

Final Answer: Dual orexin (OX_1/OX_2) receptor antagonism $\Rightarrow$ B

Answer: (B) Go Back to Q52

Q53.

Solution

Concept — Triptan mechanism: Triptans are selective serotonin 5-$HT_{1B/1D}$ receptor agonists. **Reasoning:** Rizatriptan activates 5-HT_{1B} receptors on cranial vessels (causing vasoconstriction) and 5-HT_{1D} receptors on trigeminal nerve endings (inhibiting release of vasoactive neuropeptides such as CGRP), aborting the acute migraine. **Why the other options are wrong:**

- (A) D_2 action relates to anti-nausea drugs, not triptans.
- (B) It is not an opioid.
- (D) GABA-B agonism describes baclofen.

Final Answer: 5-$HT_{1B/1D}$ agonism $\Rightarrow$ C

Answer: (C) Go Back to Q53

Q54.

Solution

Concept — Funny-current (I_f) inhibition: The sinoatrial pacemaker rate depends on the hyperpolarisation-activated I_f current during diastolic depolarisation. **Reasoning:** Ivabradine selectively blocks I_f in the SA node, slowing the rate of spontaneous depolarisation and thus heart rate, without affecting myocardial contractility, blood pressure or conduction below the node. **Why the other options are wrong:**

- (B) It is not a β-blocker.
- (C) It does not block L-type calcium channels.
- (D) Na^+/K^+-ATPase inhibition describes digoxin.

Final Answer: Selective SA-node I_f inhibition $\Rightarrow$ A

Answer: (A) Go Back to Q54

Q55.

Solution

Concept — Angiotensin-receptor blockers: ARBs block the AT_1 receptor downstream of angiotensin II. **Reasoning:** Olmesartan selectively antagonises AT_1 receptors, lowering vasoconstriction and aldosterone without inhibiting ACE; because bradykinin is not accumulated, the dry cough typical of ACE inhibitors is largely avoided. **Why the other options are wrong:**

- (A) ACE inhibition describes enalapril, not an ARB.
- (C) Direct renin inhibition describes aliskiren.
- (D) Aldosterone-receptor blockade describes spironolactone/eplerenone.

Final Answer: Selective AT_1 blockade without bradykinin build-up $\Rightarrow$ B

Answer: (B) Go Back to Q55

Q56.

Solution

Concept — Pure Class III action: Class III antiarrhythmics prolong repolarisation by blocking potassium currents. **Reasoning:** Dofetilide selectively blocks the rapid delayed-rectifier potassium current (I_{Kr}), prolonging the action-potential duration and effective refractory period; this maintains sinus rhythm in atrial fibrillation but carries a risk of QT prolongation. **Why the other options are wrong:**

- (A) Fast Na^+ block is Class I.
- (B) β-blockade is Class II.
- (C) L-type Ca^{2+} block is Class IV.

Final Answer: Selective I_{Kr} block (pure Class III) $\Rightarrow$ D

Answer: (D) Go Back to Q56

Q57.

Solution

Concept — Selective aldosterone antagonism: Mineralocorticoid-receptor blockers reduce sodium retention and have benefit in heart failure. **Reasoning:** Eplerenone is a selective aldosterone (mineralocorticoid) receptor antagonist with much lower affinity for androgen and progesterone receptors than spironolactone, so it rarely causes gynaecomastia or menstrual disturbance. **Why the other options are wrong:**

- (A) It is not a loop diuretic.
- (B) Direct ENaC blockade describes amiloride/triamterene.
- (D) It does not act on the renin-converting enzyme.

Final Answer: Selective aldosterone antagonist, fewer sex-hormone effects $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q57

Q58.

Solution

Concept — P2Y$_{12}$ inhibition: Platelet activation by ADP works through the P2Y$_{12}$ receptor; blocking it impairs aggregation. **Reasoning:** Ticagrelor reversibly inhibits the P2Y$_{12}$ ADP receptor (and, unlike clopidogrel, needs no hepatic activation), reducing ADP-driven platelet activation and the GP IIb/IIIa expression that mediates aggregation, lowering thrombotic risk after acute coronary syndromes. **Why the other options are wrong:**

- (B) Irreversible COX acetylation is the aspirin mechanism.
- (C) GP IIb/IIIa blockade describes abciximab/tirofiban.
- (D) Antithrombin activation describes heparin.

Final Answer: Reversible P2Y$_{12}$ ADP-receptor inhibition $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q58

Q59.

Solution

Concept — Second-generation H_1 antihistamines: Newer H_1 blockers cause little sedation because they barely enter the brain. **Reasoning:** Fexofenadine is a second-generation H_1 antagonist that is relatively hydrophilic and a P-glycoprotein substrate, so it poorly crosses the blood-brain barrier and produces minimal central H_1 blockade (sedation), unlike the lipophilic first-generation chlorpheniramine. **Why the other options are wrong:**

- (A) It blocks H_1, not H_2, receptors.
- (C) It is an H_1 antagonist, not a mast-cell stabiliser.
- (D) It has little antimuscarinic activity.

Final Answer: Poorly CNS-penetrant second-generation H_1 blocker $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q59

Q60.

Solution

Concept — Leukotriene-receptor antagonism: Cysteinyl-leukotrienes (LTC_4, LTD_4, LTE_4) are potent bronchoconstrictors acting at the $CysLT_1$ receptor. **Reasoning:** Montelukast blocks the $CysLT_1$ receptor, preventing leukotriene-induced bronchoconstriction, mucus secretion and inflammation; it is taken orally as add-on asthma prophylaxis and is useful in exercise- and aspirin-induced asthma. **Why the other options are wrong:**

- (B) 5-lipoxygenase inhibition describes zileuton.
- (C) It does not block H_1 receptors.
- (D) It is an oral antagonist, not a β_2-agonist.

Final Answer: $CysLT_1$ leukotriene-receptor antagonism $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q60

Q61.

Solution

Concept — Selective COX-2 inhibition: COX-2 is induced at sites of inflammation, whereas COX-1 maintains protective gastric prostaglandins. **Reasoning:** Etoricoxib selectively inhibits COX-2, providing analgesia and anti-inflammatory effect while largely sparing COX-1-derived gastric mucosal prostaglandins, so gastrointestinal ulceration is less than with non-selective NSAIDs such as naproxen. **Why the other options are wrong:**

- (B) Selective COX-1 inhibition would worsen, not spare, the stomach.
- (C) It inhibits cyclooxygenase, not lipoxygenase.
- (D) It is an enzyme inhibitor, not a prostaglandin analogue.

Final Answer: Selective COX-2 inhibition sparing gastric COX-1 $\Rightarrow$ A

Answer: (A) Go Back to Q61

Q62.

Solution

Concept — Prostaglandin replacement: NSAIDs deplete protective gastric prostaglandins; a prostaglandin analogue restores them. **Reasoning:** Misoprostol is a synthetic prostaglandin E_1 analogue that reinstates the prostaglandin-dependent mucosal defence (increased mucus and bicarbonate, reduced acid secretion), so it prevents NSAID-induced ulcers. **Why the other options are wrong:**

- (A) It is not a proton-pump inhibitor.
- (B) It is not a simple acid-neutralising antacid.
- (D) It does not block H_2 receptors.

Final Answer: PGE_1 analogue restoring mucosal defence $\Rightarrow$ C

Answer: (C) Go Back to Q62

Q63.

Solution

Concept — Oxazolidinone action: Linezolid inhibits an early step of bacterial protein synthesis. **Reasoning:** Linezolid binds the 23S rRNA of the 50S ribosomal subunit and prevents assembly of the functional 70S initiation complex, blocking

protein synthesis. Its novel site explains activity against MRSA and vancomycin-resistant enterococci (VRE). **Why the other options are wrong:**

- (A) Transpeptidase inhibition describes β-lactams.
- (C) DNA gyrase inhibition describes fluoroquinolones.
- (D) Dihydropteroate synthase inhibition describes sulfonamides.

Final Answer: 50S binding blocking the 70S initiation complex $\Rightarrow$ B

Answer: (B) Go Back to Q63

Q64.

Solution

Concept — Lipopeptide membrane disruption: Daptomycin targets the bacterial cell membrane rather than the ribosome or wall. **Reasoning:** In a calcium-dependent manner daptomycin inserts its lipid tail into the Gram-positive cell membrane, forming aggregates that cause rapid potassium efflux and depolarisation, killing the cell without lysis. (It is inactivated by pulmonary surfactant, so it is not used for pneumonia.) **Why the other options are wrong:**

- (A) 30S ribosomal inhibition describes aminoglycosides/tetracyclines.
- (B) RNA polymerase inhibition describes rifampicin.
- (D) Folate-synthesis blockade describes sulfonamides.

Final Answer: Calcium-dependent membrane depolarisation $\Rightarrow$ C

Answer: (C) Go Back to Q64

Q65.

Solution

Concept — Monobactam spectrum: Aztreonam is a monobactam β-lactam with a narrow Gram-negative spectrum. **Reasoning:** Aztreonam binds PBP3 to inhibit cell-wall synthesis, but it is active essentially only against aerobic Gram-negative bacilli (including *Pseudomonas*); its monocyclic structure shows little immunological cross-reactivity with penicillins, so it is useful in penicillin-allergic patients. **Why the other options are wrong:**

- (B) It acts on the cell wall, not the 50S ribosome.
- (C) It is inactive against Gram-positive cocci and anaerobes.

- (D) It is a β-lactam, not a fluoroquinolone.

Final Answer: Gram-negative cell-wall inhibitor, low penicillin cross-allergy $\Rightarrow$ A

Answer: (A) Go Back to Q65

Q66.

Solution

Concept — Neuraminidase inhibition: Influenza neuraminidase cleaves sialic acid to release new virions; blocking it traps virus on the cell. **Reasoning:** Oseltamivir (an oral prodrug activated to oseltamivir carboxylate) inhibits viral neuraminidase, so newly formed virions cannot detach and spread, shortening the illness when started early. **Why the other options are wrong:**

- (A) Reverse-transcriptase inhibition is an antiretroviral action.
- (B) M2 ion-channel block describes amantadine.
- (D) Viral DNA-polymerase inhibition describes aciclovir (a herpesvirus drug).

Final Answer: Inhibition of viral neuraminidase $\Rightarrow$ C

Answer: (C) Go Back to Q66

Q67.

Solution

Concept — Incretin mimetics: GLP-1 receptor agonists reproduce the actions of the gut incretin hormone GLP-1. **Reasoning:** Liraglutide activates the GLP-1 receptor, enhancing glucose-dependent insulin release, suppressing glucagon, slowing gastric emptying and increasing satiety; this lowers blood glucose with a low hypoglycaemia risk and promotes weight loss. **Why the other options are wrong:**

- (A) SGLT2 inhibition describes the gliflozins.
- (C) Closing K_{ATP} channels describes sulfonylureas.
- (D) Intestinal α-glucosidase inhibition describes acarbose.

Final Answer: GLP-1 receptor agonist (incretin mimetic) $\Rightarrow$ B

Answer: (B) Go Back to Q67

Q68.

Solution

Concept — Toxic-metabolite blockade: Methanol and ethylene glycol are themselves relatively harmless; their toxicity comes from alcohol-dehydrogenase-derived acid metabolites. **Reasoning:** Fomepizole inhibits alcohol dehydrogenase, blocking conversion of methanol to formic acid and of ethylene glycol to glycolic and oxalic acids, so the parent alcohol can be cleared (or dialysed) before toxic acids accumulate. **Why the other options are wrong:**

- (A) It does not chelate the alcohol.
- (B) It is not a GABA-A antagonist.
- (C) Glutathione replenishment describes N-acetylcysteine in paracetamol overdose.

Final Answer: Alcohol-dehydrogenase inhibition preventing toxic acids $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q68

Q69.

Solution

Concept — McMurry coupling: Low-valent titanium reductively couples two carbonyls into an alkene, deoxygenating both. **Reasoning:** $TiCl_3/Zn$ (or similar) reduces the carbonyls to a pinacolate bound on a Ti surface; deoxygenation then expels the oxygens and forms the C=C double bond. Two ketones thus give a tetrasubstituted symmetrical alkene. **Why the other options are wrong:**

- (A) The Wittig uses a phosphorus ylide on one carbonyl, not reductive dimerisation.
- (B) Tishchenko gives an ester, not an alkene.
- (D) Reformatsky uses a zinc enolate and gives a β-hydroxy ester.

Final Answer: McMurry coupling $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q69

Q70.

Solution

Concept — Tishchenko reaction: An aluminium-alkoxide-catalysed disproportionation of an aldehyde lacking α-H to give an ester. **Reasoning:** A hydride is transferred from one aldehyde (oxidised to the acyl) to a second aldehyde (reduced to the alkoxy), and the two combine as an ester. Benzaldehyde thus gives benzyl benzoate. **Why the other options are wrong:**

- (B) Cannizzaro (strong base) gives the separate alcohol + carboxylate salt, not an ester.
- (C) Claisen condensation couples two esters to a β-keto ester.
- (D) Perkin uses an anhydride/base to give a cinnamic acid.

Final Answer: Tishchenko reaction $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q70

Q71.

Solution

Concept — Shapiro reaction: Two equivalents of an organolithium convert a tosylhydrazone into the less-substituted alkene. **Reasoning:** The first base removes the N–H proton, the second removes the α-C–H; loss of the sulfinate gives a vinyl/dianion that expels N_2 to form a vinyllithium, which protonates to the alkene. The regiochemistry favours the less-substituted (Hofmann-type) alkene. **Why the other options are wrong:**

- (A) Bamford–Stevens uses a weaker base/heat and tends to give the more-substituted alkene via a carbene.
- (C) The Wittig needs a phosphorus ylide and a carbonyl.
- (D) HWE uses a stabilised phosphonate carbanion with an aldehyde.

Final Answer: Shapiro reaction $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q71

Q72.

Solution

Concept — Stetter reaction: An NHC (thiazolium) catalyst generates an acyl-anion equivalent that does conjugate (1,4) addition. **Reasoning:** The carbene adds to the aldehyde to form the Breslow intermediate (a d^1 acyl anion), which adds to a Michael acceptor at the β-carbon. Collapse releases the catalyst and gives a 1,4-diketone. This is the umpolung hallmark. **Why the other options are wrong:**

- (A) The benzoin condensation joins two aldehydes (1,2-addition) to an α-hydroxy ketone.
- (B) A simple Michael addition uses a normal (non-umpolung) carbanion.
- (C) Mannich uses an enol/enolate with an iminium ion.

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

Answer: (D) Go Back to Q72

Q73.

Solution

Concept — Corey–Seebach (dithiane) umpolung: A 1,3-dithiane anion is a masked acyl anion. **Reasoning:** Forming the dithiane and deprotonating it makes the originally electrophilic carbonyl carbon nucleophilic. After alkylation and hydrolysis, a new ketone results, so the net effect is reversal of the carbon's polarity (umpolung). **Why the other options are wrong:**

- (B) It is not a reduction to an alcohol.
- (C) It is not an oxidation to an acid.
- (D) No sigmatropic shift is involved.

Final Answer: Umpolung of the carbonyl carbon $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q73

Q74.

Solution

Concept — Wohl–Ziegler bromination: NBS with a radical initiator brominates allylic (or benzylic) C–H bonds. **Reasoning:** NBS maintains a low, steady concentration of Br_2/Br·; the low bromine level favours radical H-abstraction at the allylic position over ionic addition to the alkene, giving the allylic bromide selectively. **Why the other options are wrong:**

- (A) Hell–Volhard–Zelinsky brominates the α-carbon of a carboxylic acid.
- (B) Sandmeyer replaces a diazonium group with a halide.
- (D) Appel converts an alcohol to an alkyl halide (CBr_4/PPh_3).

Final Answer: Wohl–Ziegler allylic bromination $\Rightarrow$ C

Answer: (C) Go Back to Q74

Q75.

Solution

Concept — Pictet–Spengler reaction: A β-arylethylamine plus an aldehyde cyclises to a tetrahydroisoquinoline. **Reasoning:** Condensation gives an iminium ion; the electron-rich aromatic ring attacks it intramolecularly (Mannich-type electrophilic aromatic substitution), closing the new six-membered ring. It is a key step in alkaloid biosynthesis/synthesis. **Why the other options are wrong:**

- (A) Bischler–Napieralski cyclodehydrates an acyl-arylethylamine to a dihydroisoquinoline.
- (C) Skraup builds a quinoline from an aniline and glycerol.
- (D) Combes condenses an aniline with a 1,3-diketone to a quinoline.

Final Answer: Pictet–Spengler reaction $\Rightarrow$ B

Answer: (B) Go Back to Q75

Q76.

Solution

Concept — E1cb mechanism: Elimination via a carbanion (conjugate base) intermediate. **Reasoning:** A base first removes an acidic β-proton (often α to a carbonyl) to give a resonance-stabilised carbanion; the leaving group then departs

in a separate step. This stepwise, base-first pathway defines E1cb. **Why the other options are wrong:**

- (A) E2 is concerted, with no discrete carbanion.
- (B) S_N1 is a substitution via a carbocation.
- (C) E1 goes through a carbocation, the opposite charge.

Final Answer: E1cb elimination $\Rightarrow$ D

Answer: (D) Go Back to Q76

Q77.

Solution

Concept — Chair conformation: Substituents prefer the equatorial position to avoid 1,3-diaxial strain. **Reasoning:** An axial methyl group suffers 1,3-diaxial repulsions with the two axial hydrogens on the same face. Placing it equatorial removes these clashes, so the equatorial chair is the lower-energy, more populated conformer. **Why the other options are wrong:**

- (B) Axial is the strained, higher-energy arrangement.
- (C) Cyclohexane has no bridgehead.
- (D) There is no ring oxygen in methylcyclohexane.

Final Answer: Equatorial $\Rightarrow$ A

Answer: (A) Go Back to Q77

Q78.

Solution

Concept — Keto–enol tautomerism: Two constitutional isomers interconverting by a 1,3-proton shift and double-bond migration. **Reasoning:** The enol (C=C–OH) and keto (O=C–C–H) forms differ in the position of one proton and one π bond, so they are tautomers in dynamic equilibrium, not mere resonance forms. **Why the other options are wrong:**

- (A) Resonance forms differ only in electron position, with fixed atoms.
- (B) Conformers interconvert by bond rotation, not proton shift.
- (D) Optical isomers differ in 3-D handedness, not connectivity.

Final Answer: Keto–enol tautomerism $\Rightarrow$ C

Answer: (C) Go Back to Q78

Q79.

Solution

Concept — S_NAr (addition–elimination): The rate-limiting step forms an anionic Meisenheimer intermediate. **Reasoning:** Electron-withdrawing groups ortho/para to the leaving group stabilise the negative charge of the Meisenheimer complex by resonance, greatly accelerating S_NAr. The $–NO_2$ group is the classic activator. **Why the other options are wrong:**

- (A) Electron donors destabilise the anionic intermediate and slow the reaction.
- (C) Steric bulk does not accelerate S_NAr.
- (D) Halogens are weakly deactivating/withdrawing, not activating donors here.

Final Answer: Strong EWGs (e.g. $–NO_2$) accelerate $S_NAr \Rightarrow$ B

Answer: (B) Go Back to Q79

Q80.

Solution

Concept — Acridine: A dibenzo[b,e]pyridine, i.e. a linear tricyclic aromatic with a central nitrogen-bearing ring. **Reasoning:** The figure shows three linearly fused six-membered rings with one nitrogen in the central ring (the aza-analogue of anthracene). This is acridine, the scaffold of intercalating dyes and antimalarials such as quinacrine/proflavine. **Why the other options are wrong:**

- (A) Carbazole has a central five-membered N-ring (dibenzopyrrole), not a six-membered one.
- (B) Anthracene is the all-carbon analogue (no nitrogen).
- (C) Dibenzofuran has a central oxygen-containing five-membered ring.

Final Answer: Acridine $\Rightarrow$ D

Answer: (D) Go Back to Q80

Q81.

Solution

Concept — E/Z nomenclature: Assign CIP priority on each double-bond carbon; same side = Z, opposite = E. **Reasoning:** On C2 the higher priority is Br ($> CH_3$); on C3 the higher priority is CH_3 ($>$ H). When these two higher-priority groups lie on the same side, the alkene is the Z isomer (German *zusammen* = together). **Why the other options are wrong:**

- (B) E (*entgegen*) means the higher-priority groups are on opposite sides.
- (C) R describes a tetrahedral stereocentre, not a double bond.
- (D) Meso refers to an internally compensated chiral molecule.

Final Answer: Z $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q81

Q82.

Solution

Concept — Baeyer–Villiger migratory aptitude: The group best able to stabilise positive charge migrates to oxygen. **Reasoning:** In the Criegee intermediate the migrating group bears partial positive character in the transition state, so more-substituted (electron-rich) carbons migrate fastest: tertiary $>$ secondary $\approx$ aryl $>$ primary $>$ methyl. Oxygen therefore inserts on the more substituted side. **Why the other options are wrong:**

- (A) Reverses the true order; methyl migrates worst.
- (B) Migration is strongly group-dependent, not equal.
- (D) Aryl groups actually migrate readily.

Final Answer: tertiary $>$ secondary $>$ primary $>$ methyl $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q82

Q83.

Solution

Concept — First-generation H_1-antihistamine SAR: CNS sedation correlates with lipophilicity and BBB penetration. **Reasoning:** Drugs like diphenhydramine are small, lipophilic, largely un-ionised tertiary amines that cross the blood–

brain barrier and block central H_1 receptors, causing sedation. Second-generation agents were made more polar to stay peripheral. **Why the other options are wrong:**

- (A) Highly ionised, water-soluble drugs do not readily cross the BBB.
- (C) They are small molecules, not transported peptides.
- (D) They are reversible competitive antagonists, not covalent blockers.

Final Answer: Lipophilic uncharged amines penetrating the CNS $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q83

Q84.

Solution

Concept — Statin mechanism: Statins competitively inhibit the rate-limiting enzyme of cholesterol synthesis. **Reasoning:** The dihydroxy-acid portion of statins mimics the HMG (mevalonate-precursor) substrate of HMG-CoA reductase, binding the active site with high affinity and blocking mevalonate formation, lowering cholesterol synthesis. **Why the other options are wrong:**

- (A) COX is the target of NSAIDs.
- (B) ACE is inhibited by the "-pril" drugs.
- (C) Na^+/K^+-ATPase is inhibited by cardiac glycosides.

Final Answer: HMG-CoA reductase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q84

Q85.

Solution

Concept — Fluoroquinolone target: They inhibit bacterial type-II topoisomerases. **Reasoning:** Ciprofloxacin and congeners bind the enzyme–DNA complex of DNA gyrase (topoisomerase II) in Gram-negatives and topoisomerase IV in Gram-positives, blocking DNA supercoiling/decatenation and causing lethal double-strand breaks. **Why the other options are wrong:**

- (B) The 30S subunit is the aminoglycoside/tetracycline target.
- (C) Dihydropteroate synthase is the sulfonamide target.
- (D) The transpeptidase (PBP) is the β-lactam target.

Final Answer: DNA gyrase / topoisomerase IV $\Rightarrow$ A

Answer: (A) Go Back to Q85

Q86.

Solution

Concept — Proton-pump inhibitors: Acid-activated prodrugs that covalently inhibit the gastric H^+/K^+-ATPase. **Reasoning:** In the acidic secretory canaliculus, omeprazole rearranges to a reactive sulfenamide that forms a disulfide bond with cysteine residues of the proton pump, irreversibly shutting off acid secretion until new pump protein is synthesised. **Why the other options are wrong:**

- (A) Blocking the H_2 receptor is the mechanism of ranitidine etc.
- (B) PPIs are not direct chemical neutralisers (that is an antacid).
- (D) They do not act by chelating calcium.

Final Answer: Covalent inhibition of the H^+/K^+-ATPase $\Rightarrow$ C

Answer: (C) Go Back to Q86

Q87.

Solution

Concept — Sequential folate blockade: Co-trimoxazole inhibits two consecutive enzymes of folate metabolism. **Reasoning:** Sulfamethoxazole (a PABA analogue) blocks dihydropteroate synthase, while trimethoprim selectively inhibits bacterial dihydrofolate reductase. Hitting two sequential steps gives synergistic, often bactericidal, action. **Why the other options are wrong:**

- (A) Dihydropteroate synthase is the sulfonamide target, not trimethoprim's.
- (C) Thymidylate synthase is inhibited by 5-fluorouracil.
- (D) DNA polymerase is not the target here.

Final Answer: Bacterial dihydrofolate reductase $\Rightarrow$ B

Answer: (B) Go Back to Q87

Q88.

Solution

Concept — pH–partition hypothesis: Only the un-ionised form of a drug crosses lipid membranes readily. **Reasoning:** For a weak acid ($pK_a \approx 4$) at gastric pH ≈ 2, pH $< pK_a$, so the Henderson–Hasselbalch ratio favours the protonated, un-ionised (lipid-soluble) species, which is absorbed best across the gastric mucosa. **Why the other options are wrong:**

- (A) The fully ionised form is poorly lipid-permeable.
- (B) A simple monoprotic weak acid is not zwitterionic.
- (C) Existence as a chloride salt is not what drives passive lipid permeation.

Final Answer: The un-ionised, lipid-soluble form $\Rightarrow$ D

Answer: (D) Go Back to Q88

Q89.

Solution

Concept — Catechol metabolism: The catechol ring is a substrate for COMT. **Reasoning:** Catechol-*O*-methyltransferase methylates one catechol hydroxyl of catecholamines (e.g. adrenaline) using S-adenosylmethionine, rapidly inactivating them; this, with MAO, gives their short duration and poor oral activity. **Why the other options are wrong:**

- (B) Acetylcholinesterase hydrolyses acetylcholine.
- (C) Carbonic anhydrase handles CO_2/bicarbonate, not catechols.
- (D) Xanthine oxidase oxidises purines to uric acid.

Final Answer: Catechol-*O*-methyltransferase (COMT) $\Rightarrow$ A

Answer: (A) Go Back to Q89

Q90.

Solution

Concept — Nitrogen-mustard alkylation: They form an aziridinium electrophile that alkylates DNA. **Reasoning:** The β-chloroethylamine cyclises to a reactive aziridinium ion, which is attacked by the nucleophilic N-7 of guanine. A bifunctional mustard can alkylate two guanines, cross-linking DNA strands and blocking

replication. **Why the other options are wrong:**

- (A) It does not target a protease serine.
- (B) Phospholipid alkylation is not the cytotoxic mechanism.
- (D) It does not bind the P450 heme iron.

Final Answer: N-7 of guanine in DNA $\Rightarrow$ C

Answer: (C) Go Back to Q90

Q91.

Solution

Concept — Partial agonist: High affinity but sub-maximal intrinsic activity (efficacy between 0 and 1). **Reasoning:** A partial agonist occupies the receptor and elicits a response below the system maximum even at full occupancy; in the presence of a full agonist it competes and lowers the overall response, so it can also behave as an antagonist. **Why the other options are wrong:**

- (A) A full agonist gives the maximal response (efficacy $= 1$).
- (C) An irreversible antagonist produces no response and cannot be displaced.
- (D) An inverse agonist reduces constitutive (basal) activity below baseline, a different effect.

Final Answer: Partial agonist $\Rightarrow$ B

Answer: (B) Go Back to Q91

Q92.

Solution

Concept — Lipinski's rule of five: Four physicochemical limits predicting oral absorption. **Reasoning:** The criteria are MW ≤ 500, log$P \leq 5$, H-bond donors ≤ 5, and H-bond acceptors ≤ 10. Melting point is not part of the rule, so it is the odd one out. **Why the other options are wrong:**

- (A) MW ≤ 500 is a genuine rule-of-five limit.
- (B) log$P \leq 5$ is a genuine limit.
- (C) H-bond donors ≤ 5 is a genuine limit.

Final Answer: Melting point is not a rule-of-five criterion $\Rightarrow$ D

Answer: (D) Go Back to Q92

Q93.

Solution

Concept — ACE inhibitor zinc-binding group: Captopril coordinates the catalytic zinc of ACE. **Reasoning:** ACE is a zinc metallopeptidase; captopril's free sulfhydryl (–SH) chelates the active-site Zn^{2+}, giving tight binding. (Later inhibitors such as enalaprilat use a carboxylate instead.) **Why the other options are wrong:**

- (B) Captopril has no phenol coordinating zinc.
- (C) There is no quaternary ammonium zinc ligand.
- (D) The tetrazole is the zinc-unrelated acidic group used in sartans (AT_1 blockers).

Final Answer: The sulfhydryl (–SH) group $\Rightarrow$ A

Answer: (A) Go Back to Q93

Q94.

Solution

Concept — COX-2 selectivity SAR: Coxibs exploit a larger side-pocket unique to COX-2. **Reasoning:** COX-2 has Val523 where COX-1 has the bulkier Ile523, opening an extra side-pocket. The bulky aryl-sulfonamide/sulfone of celecoxib fits this pocket, giving COX-2 selectivity and sparing gastric COX-1. **Why the other options are wrong:**

- (A) The COX-2 channel is larger, not smaller/blocked.
- (B) The catalytic tyrosine is present in both isoforms.
- (D) Coxibs are reversible, not aspirin-like covalent acetylators.

Final Answer: A larger Val523 side-pocket fitting a bulky sulfonamide $\Rightarrow$ C

Answer: (C) Go Back to Q94

Q95.

Solution

Concept — Apparent volume of distribution: V_d relates dose to plasma concentration; a large value implies tissue sequestration. **Reasoning:** When V_d greatly exceeds total body water, only a small fraction of drug remains in plasma; most is bound to or partitioned into peripheral tissues (fat, muscle), giving a low plasma concentration for the dose. **Why the other options are wrong:**

- (A) Plasma confinement gives a small V_d ($\approx$ plasma volume).
- (C) V_d is defined for absorbed drug, not unabsorbed drug.
- (D) Route of elimination does not by itself define V_d.

Final Answer: Extensive tissue binding/sequestration $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q95

Q96.

Solution

Concept — Energy profile and ΔH: The sign of ΔH is set by the relative energies of reactants and products. **Reasoning:** The diagram shows products (P) lying above reactants (R), so heat is absorbed; $\Delta H = E_{\text{products}} - E_{\text{reactants}} > 0$, i.e. the reaction is endothermic. **Why the other options are wrong:**

- (B) Exothermic would put products below reactants.
- (C) Thermoneutral would have R and P at equal energy.
- (D) A finite barrier (TS) is clearly present, so it is not barrierless.

Final Answer: Endothermic ($\Delta H > 0$) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q96

Q97.

Solution

Concept — Cisplatin geometry: Pt(II) is a d^8 ion that adopts square-planar coordination. **Reasoning:** Platinum(II) has the d^8 configuration, which strongly favours square-planar geometry. The *cis* arrangement of the two labile chlorides lets the drug form intrastrand DNA cross-links, which the *trans* isomer cannot. **Why the other options are wrong:**

- (A) d^{10} tetrahedral describes ions like Zn(II), not Pt(II).
- (B) d^{6} octahedral describes ions like Co(III)/Pt(IV).
- (C) d^{0} linear does not match Pt(II).

Final Answer: d^{8}, square-planar $\Rightarrow$ D

Answer: (D) Go Back to Q97

Q98.

Solution

Concept — Henderson–Hasselbalch: At equal acid/conjugate-base concentrations the log term vanishes. **Reasoning:** When $[A^-] = [HA]$, the ratio is 1 and $\log 1 = 0$, so pH = pK_a. This is the centre of the buffer region and the point of maximum buffer capacity. **Why the other options are wrong:**

- (A) $pK_a - 1$ corresponds to a 1:10 base:acid ratio.
- (C) $pK_a + 1$ corresponds to a 10:1 base:acid ratio.
- (D) The pH need not be 7; it equals the pK_a (≈ 4.76 for acetate).

Final Answer: pH = pK_a of acetic acid $\Rightarrow$ B

Answer: (B) Go Back to Q98

Q99.

Solution

Concept — Kjeldahl digestion: hot concentrated H_2SO_4 with a catalyst converts organic nitrogen to ammonium ion. **Reasoning:** During digestion the carbon and hydrogen are oxidised, and the organically bound nitrogen is reduced to ammonia, which is retained in the acidic medium as ammonium sulphate, $(NH_4)_2SO_4$. The ammonia is later liberated by alkali, distilled, and titrated. **Why the other options are wrong:**

- (A) Nitrogen is not evolved as N_2; that would lose the analyte.
- (C) Nitric acid is not formed; nitrogen is reduced, not oxidised, to ammonium.
- (D) Nitrous oxide is not a Kjeldahl digestion product.

Final Answer: Nitrogen is fixed as ammonium sulphate $\Rightarrow$ B

Answer: (B) Go Back to Q99

Q100.

Solution

Concept — Redox indicators for cerimetry: a suitable indicator changes colour over the steep part of the Ce(IV)/Fe(II) titration curve. **Reasoning:** N-phenylanthranilic acid is a classic redox indicator for cerimetric (and dichromate) titrations of iron(II); it is colourless in the reduced form and turns purple-violet on oxidation at the first excess of ceric ion. **Why the other options are wrong:**

- (A) Starch is an indicator for iodine, not for ceric titrations.
- (B) Eriochrome Black T is a metal-ion (complexometric) indicator, not a redox indicator.
- (C) Methyl orange is an acid–base indicator.

Final Answer: N-phenylanthranilic acid $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q100

Q101.

Solution

Concept — EDTA back-titration: excess EDTA is added, and the unreacted portion is titrated with a standard metal-ion solution. **Reasoning:** Standard zinc sulphate (or zinc chloride) solution is the usual back-titrant for residual EDTA, with a metallochromic indicator such as Eriochrome Black T marking the point at which all free EDTA has been consumed by zinc. **Why the other options are wrong:**

- (A) Sodium thiosulphate is a redox (iodometric) titrant, not a metal back-titrant.
- (B) Permanganate is a redox titrant and does not titrate EDTA.
- (D) Silver nitrate is an argentometric reagent, not used to titrate excess EDTA.

Final Answer: Standard zinc sulphate solution $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q101

Q102.

Solution

Concept — Normality and equivalents: $N = M\times$ (equivalents per mole). **Reasoning:** Na_2CO_3 neutralises 2 H^+ per mole (to H_2CO_3), so its equivalence factor is 2. Therefore $N = 0.10 \times 2 = 0.20$ N. **Why the other options are wrong:**

- (B) 0.10 N treats it as monoprotic.
- (C) 0.050 N divides instead of multiplies by 2.
- (D) 0.40 N uses an equivalence factor of 4, which is wrong.

Final Answer: $N = 0.10 \times 2 = 0.20$ N $\Rightarrow$ A

Answer: (A) Go Back to Q102

Q103.

Solution

Concept — Iodine value: a halogen reagent adds across carbon–carbon double bonds. **Reasoning:** In the Wijs method, iodine monochloride adds to each C=C double bond; the excess halogen is back-titrated with thiosulphate. The iodine value (g of iodine absorbed per 100 g of oil) therefore measures the degree of unsaturation of the fat. **Why the other options are wrong:**

- (A) Free fatty acid content is given by the acid value, not the iodine value.
- (C) Hydrolysis of esters by alkali is measured by the saponification value.
- (D) Water content is measured by Karl Fischer titration.

Final Answer: Iodine value measures unsaturation $\Rightarrow$ B

Answer: (B) Go Back to Q103

Q104.

Solution

Concept — Diazotisation end point: the first excess of nitrous acid is detected outside the titration vessel. **Reasoning:** Once all the aromatic amine is diazotised, the next drop of sodium nitrite leaves free nitrous acid in solution. A drop placed on starch–iodide paper liberates iodine, which turns the paper blue, signalling the end point. **Why the other options are wrong:**

- (A) The paper does not lose colour; it gains a blue colour at the end point.

- (B) There is no blue-to-colourless-to-blue cycle; a single blue colour marks completion.
- (D) A red colour is not the end-point response of starch–iodide paper.

Final Answer: Paper turns blue at the first excess of nitrous acid $\Rightarrow$ C

Answer: (C) Go Back to Q104

Q105.

Solution

Concept — Saponification value: alkali hydrolyses ester groups and neutralises free acids. **Reasoning:** The saponification value is the mg of KOH needed to saponify the esters *and* neutralise the free fatty acids present in 1 g of the substance. It is inversely related to the average molecular mass of the fatty acids. **Why the other options are wrong:**

- (A) Neutralising only the free acids defines the acid value.
- (B) Adding across double bonds defines the iodine value.
- (C) Liberating glycerol is not how the saponification value is defined.

Final Answer: KOH to saponify esters plus neutralise free acids in 1 g $\Rightarrow$ D

Answer: (D) Go Back to Q105

Q106.

Solution

Concept — Beer–Lambert law: $A = \varepsilon c l$, so $\varepsilon = A/(c\,l)$. **Reasoning:** With $A = 0.80$, $c = 4.0 \times 10^{-5}$ mol L^{-1} and $l = 1.0$ cm, $\varepsilon = 0.80/(4.0 \times 10^{-5} \times 1.0) = 2.0 \times 10^{4}$ L mol^{-1} cm^{-1}. The slope of the line (εl) equals this value since $l = 1$ cm. **Why the other options are wrong:**

- (B) 3.2×10^{-5} multiplies instead of divides, giving the wrong order of magnitude.
- (C) 5.0×10^{-5} inverts the calculation.
- (D) 0.80 is the absorbance reading, not ε.

Final Answer: $\varepsilon = 0.80/(4.0 \times 10^{-5}) = 2.0 \times 10^{4}$ L mol^{-1} cm^{-1} $\Rightarrow$ A

Answer: (A) Go Back to Q106

Q107.

Solution

Concept — IR group frequencies: the C≡N stretch absorbs in the relatively empty 2200–2260 cm^{-1} region. **Reasoning:** A sharp, medium band near 2250 cm^{-1} is the textbook nitrile (C≡N) stretch. Few common groups absorb in this window, so the band is diagnostic. **Why the other options are wrong:**

- (A) Ester C=O appears near 1735–1750 cm^{-1}, much lower.
- (C) Aromatic C–H stretch is just above 3000 cm^{-1}.
- (D) O–H stretch is a broad band near 3200–3600 cm^{-1}.

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

Answer: (B) Go Back to Q107

Q108.

Solution

Concept — Deshielding and chemical shift: loss of electron density moves a proton downfield. **Reasoning:** A deshielded proton feels a stronger effective field, so it resonates at higher frequency, i.e. at a larger δ (downfield, to the left of the spectrum). Electron-withdrawing groups deshield neighbouring protons. **Why the other options are wrong:**

- (A) Upfield (smaller δ) is where shielded protons appear, the opposite case.
- (B) $\delta = 0$ is the TMS reference, not where a deshielded proton lies.
- (D) Deshielding shifts position; it does not by itself broaden the peak into a singlet.

Final Answer: Deshielded protons appear downfield, at larger $\delta \Rightarrow$ C

Answer: (C) Go Back to Q108

Q109.

Solution

Concept — Chlorine isotope pattern: ^{35}Cl and ^{37}Cl occur in roughly a 3:1 ratio. **Reasoning:** For one chlorine atom, the molecular ion (containing ^{35}Cl) and the M+2 ion (containing ^{37}Cl) appear in the same 3:1 intensity ratio as the isotope abundances, i.e. M:M+2 $\approx$ 3:1. **Why the other options are wrong:**

- (A) 1:1 is the bromine pattern (^{79}Br:^{81}Br), not chlorine.
- (B) 1:3 reverses the abundance ratio.
- (C) 9:1 corresponds to two chlorine atoms (M:M+2), not one.

Final Answer: One Cl gives M:M+2 $\approx$ 3:1 $\Rightarrow$ D

Answer: (D) Go Back to Q109

Q110.

Solution

Concept — Quenching of fluorescence: non-radiative loss of excitation energy lowers emission. **Reasoning:** Dissolved oxygen collides with the excited fluorophore and de-excites it without photon emission, reducing fluorescence intensity. Because it depends on diffusional collisions during the excited-state lifetime, this is collisional (dynamic) quenching, often described by the Stern–Volmer relationship. **Why the other options are wrong:**

- (B) A bathochromic shift is a change in wavelength, not a loss of intensity.
- (C) The inner-filter effect arises from light absorption at high concentration, not from oxygen collisions.
- (D) Phosphorescence is a slow emission process, not a quenching mechanism.

Final Answer: Oxygen causes collisional (dynamic) quenching $\Rightarrow$ A

Answer: (A) Go Back to Q110

Q111.

Solution

Concept — Flame emission colours: each alkali metal emits at a characteristic wavelength. **Reasoning:** Lithium gives a crimson-red flame emission near 670 nm, which the flame photometer isolates with the appropriate filter for quantitation. **Why the other options are wrong:**

- (A) The intense yellow line near 589 nm is sodium, not lithium.
- (C) Violet near 405 nm corresponds to potassium, not lithium.
- (D) Green near 525 nm is not the characteristic lithium line.

Final Answer: Lithium emits crimson-red near 670 nm $\Rightarrow$ B

Answer: (B) Go Back to Q111

Q112.

Solution

Concept — Chemical interference in AAS: a releasing agent preferentially binds the interferent. **Reasoning:** Lanthanum (added as $LaCl_3$) combines with phosphate in preference to calcium, freeing the calcium to atomise normally and restoring the signal. Lanthanum is the standard releasing agent for the phosphate interference on calcium. **Why the other options are wrong:**

- (A) Potassium chloride suppresses ionisation; it does not release calcium from phosphate.
- (B) Sodium sulphate adds more interfering anion and does not help.
- (D) Hydrochloric acid alone does not prevent refractory phosphate formation.

Final Answer: Lanthanum acts as the releasing agent $\Rightarrow$ C

Answer: (C) Go Back to Q112

Q113.

Solution

Concept — Specific absorbance scales linearly with concentration: $A^{1\%}_{1\,\mathrm{cm}} = A/(c_{\%}\,l)$. **Reasoning:** Here $c = 0.0020\%$ w/v, $l = 1$ cm and $A = 0.60$, so $A^{1\%}_{1\,\mathrm{cm}} = 0.60/(0.0020 \times 1) = 300$. Equivalently, a 1% solution is 500 times more concentrated than 0.0020%, giving $0.60 \times 500 = 300$. **Why the other options are wrong:**

- (A) 0.60 is the measured absorbance, not the specific absorbance.
- (B) 3.0 wrongly uses 0.20% as the concentration.
- (C) 1200 quadruples the correct value by a concentration error.

Final Answer: $A^{1\%}_{1\,\mathrm{cm}} = 0.60/0.0020 = 300 \Rightarrow$ D

Answer: (D) Go Back to Q113

Q114.

Solution

Concept — Chromatographic resolution: $R_s = 2(t_{R2} - t_{R1})/(w_1 + w_2)$. **Reasoning:** Substituting, $R_s = 2(5.2 - 4.0)/(0.80 + 0.80) = 2(1.2)/1.6 = 2.4/1.6 = 1.5$. An R_s of 1.5 corresponds to baseline resolution. **Why the other options are wrong:**

- (B) 0.67 inverts the ratio.
- (C) 3.0 omits the division by the width sum (or doubles incorrectly).
- (D) 0.30 uses wrong arithmetic on the numerator.

Final Answer: $R_s = 2(1.2)/1.6 = 1.5 \Rightarrow$ A

Answer: (A) Go Back to Q114

Q115.

Solution

Concept — Capacity factor: $k' = (t_R - t_0)/t_0$ measures retention relative to the void time. **Reasoning:** With $t_R = 7.5$ min and $t_0 = 1.5$ min, $k' = (7.5 - 1.5)/1.5 = 6.0/1.5 = 4.0$. A k' of 4 means the analyte spends four times as long in the stationary phase as in the mobile phase. **Why the other options are wrong:**

- (A) 5.0 uses t_R/t_0 instead of $(t_R - t_0)/t_0$.
- (C) 0.20 inverts the ratio.
- (D) 9.0 multiplies rather than divides.

Final Answer: $k' = (7.5 - 1.5)/1.5 = 4.0 \Rightarrow$ B

Answer: (B) Go Back to Q115

Q116.

Solution

Concept — GC detectors: the TCD senses changes in the thermal conductivity of the column effluent. **Reasoning:** The thermal conductivity detector responds to any compound whose thermal conductivity differs from that of the carrier gas, so it is universal and non-destructive, and uniquely able to detect permanent gases and water that an FID cannot. **Why the other options are wrong:**

- (A) FID is destructive and does not respond to water or permanent gases.

- (B) ECD is selective for electronegative (e.g. halogenated) species, not universal.
- (D) NPD responds selectively to nitrogen- and phosphorus-containing compounds.

Final Answer: Thermal conductivity detector (TCD) $\Rightarrow$ C

Answer: (C) Go Back to Q116

Q117.

Solution

Concept — Ion-exchange selectivity: a less hydrated, more polarisable ion binds the resin more tightly. **Reasoning:** Among ions of equal charge, the one with the smallest hydrated radius approaches the fixed charge most closely and is the most polarisable, so it is held most strongly (highest selectivity coefficient). This is why, for example, Cs^+ is retained more strongly than Li^+ on a cation exchanger. **Why the other options are wrong:**

- (A) A large hydrated radius keeps the ion away from the fixed charge, weakening binding.
- (B) Atomic number alone does not govern selectivity.
- (C) Strong hydration shields the charge and lowers affinity.

Final Answer: Smallest hydrated radius, highest polarisability binds strongest $\Rightarrow$ D

Answer: (D) Go Back to Q117

Q118.

Solution

Concept — Size-exclusion separation: large molecules are excluded from the gel pores. **Reasoning:** Molecules too large to enter the pores travel only through the interstitial volume and elute first. Smaller molecules diffuse into the pores, follow a longer path, and elute later. Hence elution is in order of decreasing size: largest first, smallest last. **Why the other options are wrong:**

- (B) Reverses the actual order; smallest elute last, not first.
- (C) SEC separates by size, not by charge.
- (D) SEC separates by size, not by hydrophobicity.

Final Answer: Largest molecules elute first $\Rightarrow$ A

Answer: (A) Go Back to Q118

Q119.

Solution

Concept — van Deemter terms: A (eddy diffusion), B/u (longitudinal diffusion), $C\,u$ (mass-transfer resistance). **Reasoning:** The $C\,u$ term grows linearly with flow velocity because, at high u, solute molecules do not have time to equilibrate between mobile and stationary phases, broadening the band. Minimising C (thin films, small particles) keeps plate height low at higher flow rates. **Why the other options are wrong:**

- (A) The A term (eddy diffusion) is essentially independent of u.
- (C) The B/u term decreases with increasing u, the opposite trend.
- (D) "Column-void term" is not part of the van Deemter equation.

Final Answer: The mass-transfer term $C\,u$ rises with flow velocity $\Rightarrow$ B

Answer: (B) Go Back to Q119

Q120.

Solution

Concept — HPLC flow path: reservoir $\rightarrow$ pump $\rightarrow$ injector $\rightarrow$ column $\rightarrow$ detector. **Reasoning:** The unit placed immediately after the column, which monitors the eluent and converts the analyte response into the electrical signal recorded as the chromatogram, is the detector (e.g. a UV or diode-array detector). **Why the other options are wrong:**

- (A) The reservoir holds the mobile phase at the start of the flow path.
- (B) The pump delivers the mobile phase under pressure, before the injector.
- (D) The injector introduces the sample before the column, not after it.

Final Answer: D is the detector $\Rightarrow$ C

Answer: (C) Go Back to Q120

Q121.

Solution

Concept — Conductometric titration of a weak acid: a poorly ionised acid has low conductance, which rises as its salt forms. **Reasoning:** Acetic acid is only slightly ionised, so the initial conductance is low. As NaOH is added, the weak acid is converted to sodium acetate, a fully dissociated salt, so the concentration of ions rises and the conductance increases gradually up to the equivalence point. **Why the other options are wrong:**

- (A) A sharp fall from removing mobile H^+ is the behaviour for a *strong* acid, where H^+ is fully ionised.
- (B) The conductance is not constant; it changes as salt forms.
- (C) It does not fall to zero; ion concentration increases, not vanishes.

Final Answer: Conductance rises gradually as the salt forms $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q121

Q122.

Solution

Concept — Precision levels (ICH Q2): repeatability, intermediate precision, reproducibility. **Reasoning:** Intermediate precision (historically called ruggedness) expresses within-laboratory variation when the method is run on different days, by different analysts, or on different instruments. It lies between repeatability (same conditions) and reproducibility (between laboratories). **Why the other options are wrong:**

- (B) Repeatability is precision under the *same* operating conditions over a short interval.
- (C) Reproducibility refers to precision *between* different laboratories.
- (D) Specificity is the ability to assess the analyte unequivocally amid other components.

Final Answer: Within-lab varied-condition precision = intermediate precision (ruggedness) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q122

Q123.

Solution

Concept — Organized vs unorganized drugs: Unorganized drugs are cell-free exudates and secretions (gums, resins, waxes, dried juices, extracts) with no cellular tissue, whereas organized drugs are entire plant organs. **Reasoning:** Gamboge is a dried gum-resin, beeswax is an animal secretion, and catechu is a dried aqueous extract. All three are cell-free metabolic products, so the set in option (C) is entirely unorganized. **Why the other options are wrong:**

- (A) Fruit, wood and bark are organized organs.
- (B) Bulb, leaf and rhizome are organized.
- (D) Liquorice root and clove bud are organized; the set is mixed.

Final Answer: Gamboge, beeswax and catechu are all cell-free products $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q123

Q124.

Solution

Concept — Chemical classification: Crude drugs may be grouped by the chemical nature of their chief constituent, regardless of botanical origin or use. **Reasoning:** *Catharanthus*, *Vinca* and *Strychnos* come from different sources but are grouped here only because their actives are indole-type alkaloids, which is grouping by constituent chemistry, i.e. chemical classification. **Why the other options are wrong:**

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

Final Answer: Grouping by constituent class is chemical classification $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q124

Q125.

Solution

Concept — Taxonomical classification: Crude drugs are arranged according to the botanical family, genus and species of the source plant. **Reasoning:** *Atropa*, *Hyoscyamus*, *Datura* and *Solanum* are placed together because all belong to the family Solanaceae, which is a purely taxonomical (botanical) basis. **Why the other options are wrong:**

- (A) Morphological grouping is by the part used.
- (C) Chemical grouping is by constituent class.
- (D) Therapeutic grouping is by pharmacological action.

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

Answer: (B) Go Back to Q125

Q126.

Solution

Concept — *Rauwolfia* alkaloids: The root of *Rauwolfia serpentina* contains many monoterpenoid indole alkaloids besides reserpine, including ajmalicine (raubasine). **Reasoning:** Ajmalicine, also called raubasine, is a peripheral vasodilator used in cerebrovascular insufficiency; it is the alkaloid described. **Why the other options are wrong:**

- (A) Vincristine is from *Catharanthus roseus*.
- (B) Quinidine is from *Cinchona*.
- (C) Strychnine is from *Strychnos nux-vomica*.

Final Answer: The *Rauwolfia* vasodilator is ajmalicine (raubasine) $\Rightarrow$ D

Answer: (D) Go Back to Q126

Q127.

Solution

Concept — Opium alkaloids: Opium contains phenanthrene alkaloids (morphine, codeine, thebaine) and benzylisoquinoline/phthalide-isoquinoline alkaloids (papaverine, noscapine). **Reasoning:** Noscapine (formerly narcotine) is a phthalide-isoquinoline alkaloid that is essentially non-narcotic and is used as an

antitussive, matching the description. **Why the other options are wrong:**

- (B) Thebaine is a convulsant phenanthrene alkaloid, not an antitussive.
- (C) Papaverine is a benzylisoquinoline smooth-muscle relaxant.
- (D) Apomorphine is a semisynthetic emetic/dopaminergic derivative.

Final Answer: The non-narcotic antitussive phthalide-isoquinoline is noscapine $\Rightarrow$ A

Answer: (A) Go Back to Q127

Q128.

Solution

Concept — Glycoalkaloids: In a steroidal glycoalkaloid a nitrogen-containing steroidal aglycone is glycosidically linked to a sugar chain, combining alkaloid and glycoside features. **Reasoning:** Solanine consists of the steroidal aglycone solanidine joined to a trisaccharide (solatriose); it is therefore a steroidal glycoalkaloid of *Solanum tuberosum*. **Why the other options are wrong:**

- (A) Purine pseudo-alkaloids are caffeine-type bases, not steroidal.
- (C) Tropane alkaloids (atropine) have a bicyclic tropane nucleus, not a steroid.
- (D) Quinoline alkaloids (quinine) bear a quinoline ring.

Final Answer: Solanine is a steroidal glycoalkaloid $\Rightarrow$ B

Answer: (B) Go Back to Q128

Q129.

Solution

Concept — β-Carboline alkaloids: *Peganum harmala* seeds yield the indole (β-carboline) alkaloids harmine and harmaline. **Reasoning:** Harmine is a reversible monoamine-oxidase inhibitor that fluoresces blue under UV light, exactly matching the description. **Why the other options are wrong:**

- (A) Colchicine is from *Colchicum autumnale*, an amide alkaloid.
- (B) Pilocarpine is an imidazole alkaloid from *Pilocarpus*.
- (D) Lobeline is a piperidine alkaloid from *Lobelia*.

Final Answer: The *Peganum* β-carboline MAO inhibitor is harmine $\Rightarrow$ C

Answer: (C) Go Back to Q129

Q130.

Solution

Concept — Cardiac glycosides: Ouabain (g-strophanthin) is a polar, fast-acting cardenolide obtained from *Strophanthus gratus* seeds (and *Acokanthera*). **Reasoning:** Its high water solubility and rapid action made it useful by injection; the official seed source is *Strophanthus gratus*. **Why the other options are wrong:**

- (A) *Digitalis purpurea* gives digitoxin from leaves.
- (B) *Nerium oleander* gives oleandrin.
- (C) *Convallaria majalis* gives convallatoxin.

Final Answer: Ouabain is from *Strophanthus gratus* seeds $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q130

Q131.

Solution

Concept — Anthraquinone glycosides: In glucofrangulin an emodin-type anthraquinone aglycone is linked to glucose and rhamnose, giving a purgative anthraquinone glycoside. **Reasoning:** Because the aglycone is an anthraquinone (emodin), glucofrangulin of frangula bark is classed as an anthraquinone (purgative) glycoside. **Why the other options are wrong:**

- (B) Cardenolides have a steroidal aglycone with a butenolide ring.
- (C) Cyanogenetic glycosides release HCN, not anthraquinones.
- (D) Steroidal saponins have a sterol/spirostane aglycone.

Final Answer: The emodin aglycone makes it an anthraquinone glycoside $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q131

Q132.

Solution

Concept — Primary cardiac glycosides: *Digitalis lanata* contains the lanatosides A, B and C as primary (genuine) glycosides carrying an acetyl group and an extra glucose. **Reasoning:** Partial hydrolysis of lanatoside C (loss of glucose and the acetyl group) yields digoxin, the therapeutic agent; lanatoside C is therefore the primary glycoside described. **Why the other options are wrong:**

- (A) Sennoside A is an anthraquinone purgative glycoside.
- (C) Glycyrrhizin is a saponin from liquorice.
- (D) Sinigrin is a glucosinolate from mustard.

Final Answer: The primary glycoside giving digoxin is lanatoside C $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q132

Q133.

Solution

Concept — Flavonoid sub-classes: A 2-phenylchromone (C6–C3–C6) skeleton lacking a 3-hydroxyl group is a flavone; a 3-hydroxyl converts it to a flavonol. **Reasoning:** Apigenin has the flavone nucleus with hydroxyls at 5, 7 and 4′ but no 3-OH, so it is a flavone (the aglycone of apiin). **Why the other options are wrong:**

- (A) A flavonol (e.g. quercetin) bears a 3-hydroxyl.
- (B) An anthocyanidin is a flavylium pigment.
- (D) An isoflavone has the phenyl ring at C-3, not C-2.

Final Answer: Apigenin (no 3-OH) is a flavone $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q133

Q134.

Solution

Concept — Cyanogenetic glycosides: These glycosides release hydrogen cyanide on enzymatic hydrolysis, along with a sugar and a carbonyl compound. **Reasoning:** Prunasin (the monoglucoside of mandelonitrile) of wild cherry hydrolyses to glucose, benzaldehyde and hydrogen cyanide; it is therefore a cyanogenetic

glycoside and the toxic gas is HCN. **Why the other options are wrong:**

- (A) Saponins do not release HCN; the gas is not CO_2.
- (B) No anthraquinone or H_2S is involved.
- (C) It is not a cardiac glycoside and no ammonia is freed.

Final Answer: Prunasin is cyanogenetic and liberates HCN $\Rightarrow$ D

Answer: (D) Go Back to Q134

Q135.

Solution

Concept — Cassia oil: Oil of *Cinnamomum cassia* is dominated by cinnamaldehyde and, unlike true cinnamon bark oil, contains little or no eugenol. **Reasoning:** Cinnamaldehyde forms about 80–90% of cassia oil and accounts for its aroma; this is the dominant aldehydic constituent. **Why the other options are wrong:**

- (B) Menthone is a peppermint constituent.
- (C) Geraniol is from rose/palmarosa-type oils.
- (D) Safrole is from sassafras, not the main cassia constituent.

Final Answer: The chief constituent of cassia oil is cinnamaldehyde $\Rightarrow$ A

Answer: (A) Go Back to Q135

Q136.

Solution

Concept — Wintergreen oil: *Gaultheria procumbens* stores the glycoside gaultherin, which on hydrolysis releases methyl salicylate. **Reasoning:** Wintergreen oil is almost entirely methyl salicylate, an aromatic ester used as a counter-irritant and rubefacient, matching the description. **Why the other options are wrong:**

- (A) Bornyl acetate is a constituent of pine/valerian oils.
- (B) Anethole is from anise/fennel.
- (D) Linalyl acetate is from lavender/bergamot.

Final Answer: Wintergreen oil is essentially methyl salicylate $\Rightarrow$ C

Answer: (C) Go Back to Q136

Q137.

Solution

Concept — Turpentine oleoresin: Turpentine tapped from *Pinus* species is steam-distilled to give oil of turpentine (pinenes) leaving a solid non-volatile residue. **Reasoning:** The non-volatile residue left after distilling off the volatile oil is colophony (rosin), a solid resin used in plasters and varnishes. **Why the other options are wrong:**

- (A) Benzoin is a balsamic resin from *Styrax*.
- (C) Agar is a seaweed polysaccharide.
- (D) Beeswax is an animal wax.

Final Answer: The residue after distilling turpentine oil is colophony (rosin) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q137

Q138.

Solution

Concept — Exudate gums: Acacia and ghatti are freely water-dispersible exudate gums giving viscous sols, whereas tragacanth and karaya largely swell to gels. **Reasoning:** Ghatti gum from *Anogeissus latifolia* behaves like acacia, forming viscous aqueous dispersions, and is used as an acacia-type emulsifier and suspending agent. **Why the other options are wrong:**

- (A) Tragacanth chiefly swells into a gel rather than dispersing freely.
- (B) Agar is a marine polysaccharide that gels on cooling.
- (C) Colophony is a resin, not a gum.

Final Answer: Ghatti gum is most similar to acacia $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q138

Q139.

Solution

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

acetate–mevalonate pathway. **Why the other options are wrong:**

- (B) Shikimate gives aromatic amino acids, not via mevalonic acid.
- (C) Acetate–malonate gives fatty acids/polyketides.
- (D) The pentose phosphate pathway yields sugars and NADPH.

Final Answer: The scheme is the acetate–mevalonate pathway ⇒ A

Answer: (A) Go Back to Q139

Q140.

Solution

Concept — Phenylalanine ammonia-lyase (PAL): PAL deaminates phenylalanine to give *trans*-cinnamic acid, the committed entry step into the phenylpropanoid pathway. **Reasoning:** Loss of ammonia from phenylalanine yields *trans*-cinnamic acid, the C6–C3 precursor of coumarins, lignans and flavonoids. **Why the other options are wrong:**

- (A) Mevalonic acid belongs to the terpenoid pathway.
- (C) Geranyl pyrophosphate is a terpenoid intermediate.
- (D) Malonyl-CoA arises from the acetate–malonate route.

Final Answer: PAL converts phenylalanine to *trans*-cinnamic acid ⇒ B

Answer: (B) Go Back to Q140

Q141.

Solution

Concept — Volatile-oil estimation: The Clevenger apparatus performs hydro/steam distillation and collects the volatile oil in a graduated trap, allowing direct reading of the percentage oil content. **Reasoning:** For drugs such as clove and cardamom the official volatile-oil content is determined in a Clevenger-type assembly, so the Clevenger apparatus is correct. **Why the other options are wrong:**

- (A) The Soxhlet apparatus is for solvent extraction of fixed/total extractives.
- (B) The Reichert–Meissl method assays volatile fatty acids of fats.
- (D) The Westphal balance measures specific gravity, not oil content.

Final Answer: Volatile-oil content is assayed in the Clevenger apparatus ⇒ C

Answer: (C) Go Back to Q141

Q142.

Solution

Concept — Cork (phellem): Cork is the outer secondary protective tissue formed by the phellogen, consisting of dead, suberised, tabular cells in regular radial rows. **Reasoning:** The sketch of thin-walled tabular suberised cells in orderly rows is the classic appearance of cork (phellem), a diagnostic character of barks and underground drugs. **Why the other options are wrong:**

- (B) Xylem vessels are wide lignified conducting tubes with pits/thickenings.
- (C) Pith parenchyma cells are rounded, living and not suberised.
- (D) Phloem fibres are elongated, thick-walled lignified cells.

Final Answer: The suberised radial-row tissue is cork (phellem) $\Rightarrow$ A

Answer: (A) Go Back to Q142

Q143.

Solution

Concept — Restriction enzymes (recognition site and overhang): A staggered cut within a palindrome leaves complementary single-stranded "sticky" ends; a central cut leaves blunt ends. **Reasoning:** *Bgl*II recognises AGATCT and cleaves A|GATCT on each strand, producing four-base 5′-GATC overhangs. These are compatible with other enzymes that leave the same GATC overhang (such as *Bam*HI), which is useful in cloning. **Why the other options are wrong:**

- (A) *Hae*III cuts GG|CC centrally, giving blunt ends.
- (B) *Xba*I recognises TCTAGA, not AGATCT.
- (D) *Sma*I cuts CCC|GGG centrally, blunt ends.

Final Answer: The AGATCT cutter leaving GATC overhangs is *Bgl*II $\Rightarrow$ C

Answer: (C) Go Back to Q143

Q144.

Solution

Concept — Baculovirus expression vector system: Baculoviruses infect insect cells and carry a very strong *polyhedrin* promoter; replacing the dispensable *polyhedrin* gene with a foreign gene gives high-level expression in cells (e.g. Sf9) that perform eukaryotic glycosylation. **Reasoning:** For a glycosylated protein expressed in insect cells under the *polyhedrin* promoter, the appropriate vector is the baculovirus (*Autographa californica* nucleopolyhedrovirus, AcMNPV) system. Insect cells fold and glycosylate proteins better than bacteria. **Why the other options are wrong:**

- (A) Phagemids replicate in *E. coli*, not insect cells, and give no eukaryotic glycosylation.
- (C) pBR322/*lac* is a bacterial system without glycosylation.
- (D) λ replacement vectors are for cloning DNA libraries in *E. coli*.

Final Answer: The *polyhedrin*-driven insect-cell system is the baculovirus vector $\Rightarrow$ B

Answer: (B) Go Back to Q144

Q145.

Solution

Concept — Colony PCR: A heat step lyses a small amount of a transformant colony, releasing template DNA that is amplified directly with primers flanking the cloning site, so an insert of the expected size can be detected without plasmid purification. **Reasoning:** Adding colony material straight into a PCR with MCS-flanking primers gives a product whose size shows whether (and how big) an insert is present. This is the fast routine screen described, namely colony PCR. **Why the other options are wrong:**

- (B) Southern blotting detects DNA by hybridisation, far slower and not a direct colony amplification.
- (C) RACE amplifies unknown cDNA ends from RNA, not a colony-screening method here.
- (D) Sequencing every colony first defeats the purpose of a quick screen.

Final Answer: Direct amplification from lysed colony material is colony PCR $\Rightarrow$ A

Answer: (A) Go Back to Q145

Q146.

Solution

Concept — Recombinant thrombolytics: t-PA-type agents activate plasminogen to plasmin, which lyses fibrin clots. Reteplase is a truncated, non-glycosylated t-PA variant with a longer half-life than alteplase. **Reasoning:** Retaining only the kringle-2 and protease domains, reteplase is a fibrinolytic (thrombolytic) drug used to dissolve coronary thrombi in acute myocardial infarction; its longer half-life allows bolus dosing. **Why the other options are wrong:**

- (A) Clotting-factor replacement (e.g. factor IX) treats haemophilia, the opposite goal.
- (B) Colony-stimulating factors (e.g. filgrastim) treat neutropenia.
- (C) Insulin analogues control blood glucose, unrelated to t-PA.

Final Answer: Reteplase is a recombinant thrombolytic t-PA variant $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q146

Q147.

Solution

Concept — Antibody fragments as drugs: An antibody can be engineered down to its Fab arm, which retains antigen binding but lacks the Fc stem, reducing size and immune effector activity. **Reasoning:** Abciximab is the Fab fragment of a chimeric (mouse/human) antibody against the platelet glycoprotein IIb/IIIa receptor; by blocking this receptor it prevents fibrinogen-mediated platelet aggregation during angioplasty. **Why the other options are wrong:**

- (A) The Fc fragment lacks the antigen-binding site needed to block GPIIb/IIIa.
- (C) It is not an IgM pentamer.
- (D) Free light chains alone do not form a functional antigen-binding site.

Final Answer: Abciximab is the Fab fragment of an anti-GPIIb/IIIa chimeric antibody $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q147

Q148.

Solution

Concept — Organic-acid fermentation: Several organic acids are made by mould fermentation; *Aspergillus niger* is the classic industrial workhorse for both citric and gluconic acid. **Reasoning:** Gluconic acid is produced by submerged aerobic fermentation in which glucose oxidase from *Aspergillus niger* oxidises glucose to gluconic acid. Product accumulates late, around phases III–IV of the curve. **Why the other options are wrong:**

- (B) *Saccharomyces cerevisiae* anaerobically makes ethanol, not gluconic acid.
- (C) *Lactobacillus delbrueckii* makes lactic acid.
- (D) *Clostridium acetobutylicum* runs the ABE (solvent) fermentation.

Final Answer: Gluconic acid is produced by *Aspergillus niger* $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q148

Q149.

Solution

Concept — Vitamin fermentation: Riboflavin (vitamin B_2) is overproduced by certain "flavinogenic" fungi that secrete large amounts of the yellow vitamin into the medium. **Reasoning:** The ascomycete *Ashbya gossypii* (and the related *Eremothecium ashbyii*) is the organism most widely used for industrial riboflavin fermentation, accumulating riboflavin far above its own metabolic need. **Why the other options are wrong:**

- (A) *Gluconobacter suboxydans* oxidises sorbitol (used in vitamin C synthesis), not riboflavin.
- (B) *Corynebacterium glutamicum* makes amino acids such as glutamic acid.
- (D) *Propionibacterium freudenreichii* is used for vitamin B_{12} and Swiss-cheese fermentation.

Final Answer: Riboflavin is produced industrially by *Ashbya gossypii* $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q149

Q150.

Solution

Concept — Polyene antifungals: Polyene macrolides (nystatin, amphotericin B) bind ergosterol in fungal membranes, forming pores; they are products of *Streptomyces* fermentation. **Reasoning:** Nystatin is obtained by fermentation of *Streptomyces noursei*. It is too toxic for systemic use and so is given topically or orally for candidiasis, acting on fungal membrane ergosterol. **Why the other options are wrong:**

- (A) *Penicillium chrysogenum* makes penicillin, a β-lactam antibacterial.
- (C) *Bacillus licheniformis* makes bacitracin, a peptide antibacterial.
- (D) *Micromonospora purpurea* makes gentamicin, an aminoglycoside.

Final Answer: Nystatin comes from *Streptomyces noursei* $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q150

Q151.

Solution

Concept — Specificity constant k_{cat}/K_m: At low [S] the Michaelis–Menten rate approaches $v = (k_{cat}/K_m)\,[E][S]$, so k_{cat}/K_m behaves as an apparent second-order rate constant for the reaction of free enzyme with free substrate, and is the best single measure of catalytic efficiency/substrate preference. **Reasoning:** On the Lineweaver–Burk plot the y-intercept gives $1/V_{max}$ and the x-intercept gives $-1/K_m$; combined with $k_{cat} = V_{max}/[E]_T$, the ratio k_{cat}/K_m is the specificity constant governing turnover when the enzyme is far from saturated. **Why the other options are wrong:**

- (A) The [S] giving half-maximal velocity is K_m itself, not k_{cat}/K_m.
- (B) Total product depends on time and amount, not on this constant.
- (C) Velocity at saturating substrate is V_{max} (or $k_{cat}[E]$), a different quantity.

Final Answer: k_{cat}/K_m is the specificity (second-order) constant at low [S] $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q151

Q152.

Solution

Concept — Immobilisation by cross-linking: Cross-linking uses a bifunctional reagent (commonly glutaraldehyde) to bond enzyme molecules to each other, forming an insoluble, carrier-free network such as a cross-linked enzyme aggregate (CLEA). **Reasoning:** The description, enzyme joined to enzyme by glutaraldehyde with no separate support, is precisely cross-linking. It gives high enzyme density but can lose some activity through chemical modification of the active site. **Why the other options are wrong:**

- (A) Entrapment cages the enzyme in a gel lattice without bonding it.
- (B) Adsorption relies on weak surface forces to a carrier.
- (D) Covalent attachment bonds the enzyme to a separate activated matrix, not enzyme-to-enzyme.

Final Answer: Glutaraldehyde-linked carrier-free aggregates are made by cross-linking $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q152

Q153.

Solution

Concept — IgG subclasses and complement: IgG1 and IgG3 fix C1q strongly and activate the classical pathway well; IgG2 is weak, and IgG4 essentially does not activate complement. **Reasoning:** IgG4 is the poorest classical-pathway activator and binds C1q very poorly, so antibody engineers choose an IgG4 (or mutated) backbone when they want a therapeutic antibody that blocks a target without recruiting complement-mediated killing. **Why the other options are wrong:**

- (B) IgG1 is a strong complement activator.
- (C) IgG3 is the strongest complement activator of the four.
- (D) IgG2 activates weakly but still better than IgG4.

Final Answer: The weakest complement-fixing subclass is IgG4 $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q153

Q154.

Solution

Concept — Neutralisation: An antibody can protect simply by binding a critical site on a virus or toxin (the receptor-binding or active site), physically blocking attachment/entry without needing complement or phagocytes. **Reasoning:** Blocking a virus's receptor-binding site or a toxin's active site so it can no longer engage host cells is neutralisation, the basis of antitoxins and many protective antibodies. **Why the other options are wrong:**

- (A) Opsonisation coats a microbe to enhance phagocytosis, a different function.
- (C) Complement-mediated lysis punches holes via the membrane-attack complex.
- (D) ADCC uses NK cells to kill antibody-coated targets, not simple blocking.

Final Answer: Blocking attachment/entry by binding the key site is neutralisation $\Rightarrow$ B

Answer: (B) Go Back to Q154

Q155.

Solution

Concept — Receptor–Fc fusion proteins (`-cept`): A `-cept` drug fuses the ligand-binding domain of a receptor to an IgG Fc, creating a soluble "decoy" that mops up the ligand before it reaches cell-surface receptors. **Reasoning:** Etanercept links the extracellular p75 TNF-receptor domain (region X) to human IgG1 Fc. It binds and sequesters soluble TNF-α, preventing the cytokine from triggering its membrane receptors, which is why it treats rheumatoid arthritis and other inflammatory disease. **Why the other options are wrong:**

- (A) It is not a clotting factor.
- (B) It carries no cytotoxic payload.
- (C) It is not an antiviral antiserum; it neutralises a self cytokine, not a pathogen.

Final Answer: Etanercept is a soluble TNF decoy receptor that sequesters TNF-α $\Rightarrow$ D

Answer: (D) Go Back to Q155

Q156.

Solution

Concept — Central role of C3 in complement: The classical, lectin and alternative pathways all converge on C3 convertase, which cleaves C3 into C3a (a small anaphylatoxin) and C3b (the major opsonin that also initiates the terminal lytic sequence). **Reasoning:** C3 is the pivotal, most abundant complement component; its cleavage to C3a and C3b is the step common to all three pathways and amplifies the whole cascade, leading on to C5 cleavage and the membrane-attack complex. **Why the other options are wrong:**

- (B) C9 polymerises only at the very end to complete the membrane-attack complex.
- (C) C1q is the recognition unit of just the classical pathway, not the common convergence point.
- (D) Factor H is a regulator that limits the alternative pathway, not the central cleaved component.

Final Answer: The central component cleaved to C3a and C3b is C3 $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q156

Q157.

Solution

Concept — Dark-field microscopy: In dark-field illumination only light scattered by the specimen reaches the objective, so unstained, very thin organisms appear bright against a black field, ideal for live, motile spirochaetes. **Reasoning:** *Treponema* and other slender spirochaetes are below the practical resolution of stained bright-field viewing and Gram-stain poorly, so they are examined live by dark-field microscopy, which shows them as bright, motile helices. **Why the other options are wrong:**

- (A) Gram staining under bright-field does not reveal these thin organisms well.
- (B) Ziehl–Neelsen is for acid-fast mycobacteria, not spirochaetes.
- (D) Electron microscopy needs fixed/dried preparations and cannot show live motility.

Final Answer: Live spirochaetes are seen by dark-field microscopy $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q157

Q158.

Solution

Concept — Thermal death point (TDP): TDP is the lowest temperature that kills all organisms in a standard suspension within a fixed time (conventionally 10 minutes), whereas thermal death time fixes the temperature and measures the time. **Reasoning:** By definition the TDP is the lowest temperature needed to sterilise a fixed-volume suspension in 10 minutes. It is a temperature value, in contrast to time-based measures such as the D-value or thermal death time. **Why the other options are wrong:**

- (A) A one-log reduction time at fixed temperature defines the D-value.
- (B) The temperature change cutting D ten-fold defines the z-value.
- (C) Killing half the population is not how TDP is defined.

Final Answer: TDP is the lowest temperature killing all organisms in $\sim$10 min $\Rightarrow$ D

Answer: (D) Go Back to Q158

Q159.

Solution

Concept — Depyrogenation: Bacterial endotoxin (a lipopolysaccharide pyrogen) is much more heat-resistant than microbial spores, so destroying it requires far harsher dry heat than ordinary sterilisation. **Reasoning:** Glass vials are depyrogenated by dry heat at about 250°C for 30 minutes or more, a cycle that both sterilises and breaks down endotoxin (a $\geq$3-log endotoxin reduction). Steam at 121°C sterilises but does not reliably destroy pyrogen. **Why the other options are wrong:**

- (A) Autoclaving kills microbes but does not reliably destroy endotoxin.
- (C) Filtration removes microbes; dissolved endotoxin can pass a 0.22 μm filter.
- (D) Ethanol disinfects surfaces but does not destroy pyrogen.

Final Answer: Depyrogenation of glass uses dry heat at $\sim$250°C $\Rightarrow$ B

Answer: (B) Go Back to Q159

Q160.

Solution

Concept — Environmental monitoring of cleanrooms: Passive air sampling exposes open agar plates so airborne microbes settle onto them; after incubation the colonies give a measure of viable air contamination. **Reasoning:** These exposed open plates are called settle plates (passive air-sampling plates). They are a standard, simple environmental-monitoring tool alongside active volumetric air samplers and surface contact plates. **Why the other options are wrong:**

- (A) RODAC contact plates sample surfaces, not settling air.
- (B) Spread plates are inoculated with a measured liquid volume, not air settling.
- (D) Pour plates mix sample into molten agar for total counts, not air monitoring.

Final Answer: Open exposed plates for passive air sampling are settle plates $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q160

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	C	2	B	3	D	4	A	5	C
6	D	7	B	8	A	9	C	10	D
11	A	12	B	13	C	14	D	15	A
16	B	17	C	18	D	19	B	20	C
21	D	22	A	23	B	24	C	25	D
26	A	27	B	28	C	29	D	30	A
31	B	32	C	33	D	34	A	35	C
36	B	37	A	38	D	39	B	40	A
41	D	42	D	43	B	44	C	45	A
46	D	47	A	48	B	49	C	50	D
51	D	52	B	53	C	54	A	55	B
56	D	57	C	58	A	59	B	60	A
61	A	62	C	63	B	64	C	65	A
66	C	67	B	68	D	69	C	70	A
71	B	72	D	73	A	74	C	75	B
76	D	77	A	78	C	79	B	80	D
81	A	82	C	83	B	84	D	85	A
86	C	87	B	88	D	89	A	90	C
91	B	92	D	93	A	94	C	95	B
96	A	97	D	98	B	99	B	100	D
101	C	102	A	103	B	104	C	105	D
106	A	107	B	108	C	109	D	110	A
111	B	112	C	113	D	114	A	115	B
116	C	117	D	118	A	119	B	120	C
121	D	122	A	123	C	124	A	125	B
126	D	127	A	128	B	129	C	130	D
131	A	132	B	133	C	134	D	135	A
136	C	137	B	138	D	139	A	140	B
141	C	142	A	143	C	144	B	145	A
146	D	147	B	148	A	149	C	150	B
151	D	152	C	153	A	154	B	155	D
156	A	157	C	158	D	159	B	160	C